HEALTH RISKS OF GOLD MINERS: A SYNOPTIC REVIEW

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Abstract. Health problems of gold miners who worked underground include decreased life expectancy; increased frequency of cancer of the trachea, bronchus, lung, stomach, and liver; increased frequency of pulmonary tuberculosis (PTB), silicosis, and pleural diseases; increased frequency of insect-borne diseases, such as malaria and dengue fever; noise-induced hearing loss; increased prevalence of certain bacterial and viral diseases; and diseases of the blood, skin, and musculoskeletal system. These problems are briefly documented in gold miners from Australia, North America, South America, and Africa. In general, HIV infection or excessive alcohol and tobacco consumption tended to exacerbate existing health problems. Miners who used elemental mercury to amalgamate and extract gold were heavily contaminated with mercury. Among individuals exposed occupationally, concentrations of mercury in their air, fish diet, hair, urine, blood, and other tissues significantly exceeded all criteria proposed by various national and international regulatory agencies for protection of human health. However, large-scale epidemiological evidence of severe mercury-associated health problems in this cohort was not demonstrable.

Key words: gold, gold mining, human health, mercury, silicosis

1. Introduction

Gold is almost universally considered to be the symbol of everything precious and of enduring value because of the effort required to extract it from nature and its scarcity relative to other metals (Petralia, 1996; Merchant, 1998). Unfortunately, the human costs of mining, extraction, refining, marketing, and accumulation of gold includes war, slavery, conscripted and convict labour, unhealthy and shortened life span, and degraded living conditions (Anikin, 1983). In this account, I review recent selected literature documenting health problems in gold miners who worked deep underground in a mercury-free environment, and those who worked on the surface using mercury amalgamation as the chief extraction technology. Health risks to gold miners from the use of cyanide in heap leaching and vat leaching gold recovery techniques were comparatively low – unlike effects on wildlife and the landscape (Eisler *et al.*, 1999; Eisler, 2000a) – and were not included in this review. This report is part of a larger work in progress on gold ecotoxicology.

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2. Historical background

Since before recorded time, gold had been mined, collected from alluvial deposits, or separated from the ores of silver, copper, and other metals (Merchant, 1998). Gold is the first metal mentioned in the Old Testament in Genesis 2:11. One gold mine in Saudi Arabia has been mined for more than 3000 years (Kirkemo *et al.*, 2001). Artisans of ancient civilisations used gold lavishly in decorating tombs and temples, and gold objects made more than 5000 years ago have been found in Egypt (Kirkemo *et al.*, 2001). Among the most productive goldfields in ancient times were those in Egypt, where in the deep mines the slave labourers were maltreated, and in Asia Minor near the River Pactolus, the source of Croesus' wealth. The Romans obtained much of their gold from Transylvania (Rose, 1948). Slaves were used to mine gold in Brazil from 1690 to 1850 (Lacerda, 1997b). In 1850–1860, gold production in the United States and Australia were at their peak. In the 1890s, the placers of the Canadian Klondike and Alaska were prominent gold producers. By 1927, the Transvaal (Republic of South Africa (RSA)) had been the richest gold field in the world for many years, although there were important gold fields in every continent and in most countries (Rose, 1948). Major population shifts as a result of gold discoveries are documented for Chile in 1545; in Brazil between 1696 and the 1970s; in Siberia between 1744 and 1866; in the United States in 1799 (North Carolina), 1847 (California), 1858 (Colorado), 1859 (Nevada), 1862 (Idaho), 1864 (Montana), and 1884 (Alaska); in Canada between 1857 and 1896; in Australia between 1850 and 1893; in New Zealand from 1862 to 1865; and in South Africa between 1873 and 1886 (Nriagu and Wong, 1997).

The use of mercury in the mining industry to amalgamate and concentrate precious metals dates from about 2700 BCE when the Phoenicians and Carthaginians used it in Spain. Amalgamation became widespread by the Romans in 50 CE and is similar to that employed today (Lacerda, 1997a). In South America, for example, mercury was used extensively by the Spanish colonisers to extract gold, releasing nearly 200 000 MT of mercury to the environment between 1550 and 1880 as a direct result of this process (Malm, 1998). At the height of the Brazilian gold rush in the 1880s, more than 6 million people were prospecting for gold in the Amazon region alone (Frery *et al.*, 2001). In modern Brazil, where there has been a gold rush since 1980, at least 2000 t of mercury were released, with subsequent mercury contamination of sediments, soils, air, fish, and human tissues; a similar situation exists in Columbia, Venezuela, Peru, and Bolivia (Malm, 1998). Recent estimates of global anthropogenic total mercury emissions range from 2000 to 4000 MT per year of which 460 t are from small scale gold mining (Porcella *et al.*, 1997). Major contributors of mercury to the environment as a result of gold mining activities include Brazil (3000 t since 1979), China (596 t since 1938), Venezuela (360 t since 1989), Bolivia (300 t since 1979), the Philippines (260 t since 1986), Columbia (248 t since 1987), the United States (150 t since 1969), and Indonesia (120 t since 1988; Lacerda, 1997a).

The total number of gold miners in the world using mercury amalgamation to produce gold ranges from 3 to 5 million, including 650 000 from Brazil, 250 000 from Tanzania, 250 000 from Indonesia, and 150 000 from Vietnam (Jernelov and Ramel, 1994). To provide a living – marginal at best – for this large number of miners, gold production and mercury use would come to thousands of tonnes annually; however, official figures account for only 10% of the production level (Jernelov and Ramel, 1994). At least 90% of the gold extracted by individual miners in Brazil is not registered with authorities for a variety of reasons, some financial. Accordingly, official gold production figures reported in Brazil and probably most other areas of the world are grossly underreported (Porvari, 1995). Cases of human mercury contamination have been reported from various sites around the world ever since mercury was introduced as the major mining technique to produce gold and other precious metals in South America hundreds of years ago (de Lacerda and Salomons, 1998). Contamination is reflected by elevated mercury concentrations in air, water, and diet, and in hair, urine, blood, and other tissues. However, only a few studies actually detected symptoms or clinical evidence of mercury poisoning.

Indigenous peoples of the Amazon living near gold mining activities have elevated levels of mercury in hair and blood. Other indigenous groups are also at risk from mercury contamination as well as from malaria and tuberculosis (Greer, 1993). The miners, mostly former farmers, are also victims of hard times and limited opportunities. Small scale gold mining offers an income, and an opportunity for upward mobility (Greer, 1993). Throughout the Brazilian Amazon, about 650 000 small scale miners are responsible for about 90% of Brazil's gold production and for the discharge of 90–120 t of mercury to the environment every year. About 33% of the miners had elevated concentrations in tissues over the tolerable limit set by the World Health Organisation (WHO) (Greer, 1993). In Brazil, it is alleged that health authorities are unable to detect conclusive evidence of mercury intoxication due to difficult logistics and the poor health conditions of the mining population which may mask evidence of mercury poisoning. There is a strong belief that a silent outbreak of mercury poisoning has the potential for regional disaster (de Lacerda and Salomons, 1998).

3. Health risks: underground miners

Health problems of gold miners from selected locations in Australia, North America, South America, and Africa are briefly documented.

3.1. AUSTRALIA

Australian gold miners are vulnerable to dengue fever (a mosquito-borne acute infectious viral disease characterised by headache, severe joint pain, and rash), silicosis (massive fibrosis of the lungs marked by shortness of breath and caused

by inhalation of silica dusts, usually SiO_2), and phthisis (a historical term used to describe a wasting condition, possibly pulmonary tuberculosis (PTB)).

Gold miners were the first recorded victims of dengue fever in 1885 in tropical north-eastern Queensland (Russell *et al.*, 1996). In the dengue epidemic of 1993, 2% of the population was infected despite source reduction of surface mosquito breeding grounds. In 1994, larvae and pupae of the dengue vector mosquito *Aedes aegypti* were found in flooded unused shafts of gold mines more than 45 m below ground. Copepods, *Mesocyclops aspericornis*, were also found in some flooded shafts and were found to be effective predators of mosquito larvae in the laboratory. Copepods ($N = 50$) were added to about half the mosquito-infested wells and the rest were untreated controls. After 9 months, all copepod-inoculated shafts were free of mosquitoes and all untreated wells contained *A. aegypti* larvae. The use of *M. aspericornis* is recommended as an effective control agent of *A. aegypti*, especially in comparatively inaccessible breeding sites, such as flooded gold mine shafts (Russell *et al.*, 1996).

Gold miners from Bendigo suffered – in epidemic proportions for 100 years, from the 1860s to the 1960s – a wasting disease, possibly silicosis or PTB. Eventually, it was treated as an occupational sickness, with social, economic, and political implications that resulted in marked improvements in working conditions, better medical treatment, and improved productivity (Kippen, 1995). In Western Australia, three retired gold miners were diagnosed with asbestos-related pleural disease after working in gold mines for 5–17 years (Lee *et al.*, 1999). They had no other significant known asbestos exposure except for possible asbestos contamination of gold mine dust. Although air from these mines contained measurable concentrations of asbestos fibers, this is the first report of asbestos-related diseases among gold miners. In view of the large number of potentially exposed workers, additional assessment is recommended on the relation between dust exposure from gold mining and asbestos-related lung disease (Lee *et al.*, 1999).

In Western Australia 2297 gold miners were examined in 1961, 1974, 1985, and 1993 for lung cancer and silicosis (de Klerk and Musk, 1998). The incidence of silicosis was clearly related to exposure to silica and the onset of silicosis conferred a significant increase in risk for subsequent lung cancer. But there was no evidence that exposure to silica caused lung cancer in the absence of silicosis. Silica has recently been reclassified as carcinogenic to humans based largely on the observed increase in rates of lung cancer in patients with silicosis. The International Agency for Research on Cancer has reclassified crystalline silica inhaled in the form of quartz or cristobalite from occupational sources as carcinogenic to humans (Class 1). Previously, silica was in Class 2A, that is, carcinogenic to animals and probably carcinogenic to humans (de Klerk and Musk, 1998).

3.2. NORTH AMERICA

Canadian gold miners had an increased risk of cancer of the trachea, bronchus, lung, and stomach. In the United States, gold miners had significantly higher rates

of lung cancer, silicosis, and tuberculosis when compared to the general population, and elevated risks for several debilitating diseases including diseases of the blood, skin, and musculoskeletal system.

A significant excess of mortality from carcinoma of the stomach was demonstrated in gold miners from Ontario, Canada, when compared to other miners (Kusiak *et al.*, 1993). The increased frequency of stomach cancer appeared 5–19 years after they began gold mining in Ontario. Twenty or more years after the gold miners started work, stomach cancer cases were significantly greater in miners born outside North America when compared to a reference population, but not in those native born. This late increase is similar to the excess of gastric carcinoma evident in residents of Ontario born in Europe. Possible explanations to account for the excess of stomach cancer in Canadian gold miners include exposures to arsenic, chromium, mineral fibre, diesel emissions, and aluminium powder. Diesel emissions and aluminium powder were rejected because gold miners and uranium miners were exposed to both agents but excess stomach cancer was noted only in gold miners. Exposure to dust was significant and the time-weighted duration of exposure to dust in gold mines was found in miners under age 60. A statistically significant time-weighted correlation for chromium – but not arsenic or mineral fibre – occurred, especially among gold miners under age 60. Exposure to chromium is associated with the development of the intestinal, rather than the diffuse, type of gastric cancer (Kusiak *et al.*, 1993). Gold miners in Ontario with 5 or more years of gold mining experience before 1945 had a significantly increased risk of primary cancer of the trachea, bronchus, or lung (Kabir and Bilgi, 1993). A minimum of 15 years latency was recorded between first employment in a dusty gold mining occupation and diagnosis of primary lung cancer. For purposes of occupational exposure assessment in establishing work-relatedness, authors concluded that primary lung cancer in Ontario gold miners was related to exposure to silica, arsenic, and radon decay products and were consistent with miner's age at first exposure, length of exposure to dust, and latency (Kabir and Bilgi, 1993).

The health of 3328 gold miners who worked underground in a South Dakota gold mine for at least 1 year (average was 9 years) between 1940 and 1965 was analysed through 1990, with emphasis on exposures to silica and non-asbestiform minerals, by death certificates and radiographic surveys (Steenland and Brown, 1995a,b). Miners had been exposed to a median silicon level of $0.05\,mg\,m^3$ after 1930 and $0.15\,mg\,m^3$ for those hired before 1930. The risk of silicosis was less than 1% with a cumulative exposure under $0.5\,mg\,m^3$ years, increasing to 68–84% for the highest cumulative exposure category of more than $4\,mg\,m^3$ years. Cumulative exposure was the best predictor of silicosis, followed by duration of exposure and average exposure. After adjustment for competing causes of death, a 45-year exposure under the current US Occupational Safety and Health Administration (OSHA) standard of $0.09\,mg\,Si\,m^3$ would lead to a lifetime risk of silicosis of 35–47%, suggesting that the current OSHA silicon

exposure level is unacceptably high (Steenland and Brown, 1995b). The lung cancer rate of these miners was 13% higher than the general USA population, 25% higher when the county was the referral group, and 27% higher 30 years post-exposure. Miners had significantly higher frequencies of tuberculosis and silicosis with clear exposure-response trends. Renal disease associated with silica exposure was elevated for those hired as young men, and also showed a positive correlation with length of exposure. This group also had significant excesses of arthritis, musculoskeletal diseases, skin diseases, diseases of autoimmune origin, and diseases of the blood and haematopoietic organs (Steenland and Brown, 1995a).

3.3. SOUTH AMERICA

Death from mining accidents in Columbia, increased prevalence of malaria in Brazil, and increased frequency of attacks by rabid vampire bats (*Desmodus rotundus*) in Venezuela are documented.

In Columbia, at least 28 gold miners were killed by landslides and dozens reported missing while digging at a condemned strip mine. The victims were said to be poor people who had ignored government warnings that erosion had made the mine unsafe (Toro, 2001). This incident was documented in a newspaper, and also, perhaps, in official mining records that were difficult to obtain. It is reasonable to conclude that gold mining fatalities are probably grossly underreported. The prevalence of malaria in Brazil has increased dramatically since the 1980s, particularly in Amazonian gold mining areas where increased colonisation and deforestation is recorded (de Andrade *et al.*, 1995). About 600 000 cases of malaria are reported annually in Brazil. The Amazon River Basin accounts for 99% of the cases in Brazil and for about 50% of all cases in the Americas. Infections by *Plasmodium vivax* protozoans represent about 58% of the cases, followed by *Plasmodium falciparum* (41%) and *Plasmodium malariae* (1.0%). Many of the infected miners have no obvious symptoms of malaria and often do not take prescribed antimalarial agents. Malarial control programs rely on early detection and treatment; however, special problems are associated with limited access to gold mining areas, the high mobility of the mining population, and the steady increase in drug-resistant *Plasmodium* species. These alluvial gold-mining sites are important reservoirs of drug-resistant *P. falciparum* and other parasites, and non-miners (Indians, farmers, loggers) who live there are at increased risk of malaria (de Andrade *et al.*, 1995). An outbreak of attacks by rabid vampire bats (154 cases in 4 months in a population of about 1500) was documented for the gold mining village of Payapal in southeastern Venezuela (Caraballo, 1996). Cattle and horses were bitten by vampire bats in the 2-month period preceding the human attacks. The outbreak may be due to loss of normal prey habitat of bats from mining, deforestation, and housing construction, and with human blood providing an alternative food source.

3.4. AFRICA

Gold miners in Africa show increased prevalence of various bacterial and viral diseases (Gabon), noise-induced hearing loss (Ghana), lung cancer (Zimbabwe), carbon monoxide poisoning (Kenya) and, in the RSA – the largest producer of gold in the world – almost the entire spectrum of mining-related health problems, especially lung diseases and cancer.

Residents of five gold-panning villages in northeastern Gabon were analysed for seroprevalence of leptospirosis and Ebola virus, both of which can cause lethal haemorrhagic fever (Bertherat *et al.*, 1999). The villages surveyed were remote, isolated communities and their economy was entirely dependent on gold. The seroprevalence was 15.7% for leptospirosis (14.7% of gold miners, 0% of fishermen) and 10.2% for Ebola virus (11.3% of miners, 25.0% of fishermen), demonstrating the persistence of this infection among the endemic population and the need to consider it a potential cause of haemorrhagic fever in Gabon. In another survey, residents from these same villages had elevated (up to 8.5%) blood serum titres for spotted fever and typhus group *Rickettsia* bacteria (Bertherat *et al.*, 1998). The influence of *Rickettsia* on public health in Africa remains unknown, but victims sometimes die as a result of infection by louse and flea vectors (Bertherat *et al.*, 1998).

Noise pollution laws are usually not enforced in developing countries. This was the case at a large gold mining company in central Ghana where 20% of all workers experienced significant noise-induced hearing loss, with frequency rates of 34% for miners, 20% for machine operators, and 0% for office workers (Amedofu *et al.*, 1996). In general, hearing loss increased with increasing age and noise exposure. The authors conclude that mining companies need to implement hearing conservation programs to protect workers exposed to hazardous noise levels. Lung cancers were reported in gold miners from Zimbabwe, with silica dust and arsenic considered relevant exposures (Boffetta *et al.*, 1994). In Kenya, carbon monoxide is responsible for many deaths underground; in 1980, for example, seven miners died underground due to suffocation from carbon monoxide released from a faulty water pump (Ogola *et al.*, 2002).

The gold mining industry in the RSA began around 1886 when gold was discovered on the Witwatersrand (Butchart, 1996). By 1920, about 200 000 migrant African labourers were employed in the RSA gold mines; in 1961, this number was 427 000, and in 1988 just over 500 000. Most worked underground at depths up to 3500 m. Until the mid-1970s, when recruiting patterns began to shift towards domestic sources of migrant labour, most workers were recruited from Mozambique and Malawi, with smaller numbers coming from Angola, Botswana, Zambia, and Zimbabwe. In the 1970s, critical studies appeared on the conditions of extreme social and physical deprivation governed by monetary interests and racist policies. These conditions reportedly rendered the labour force excessively prone to tuberculosis and pneumonia, parasitic infections, and traumatic injury or death as a result

of poor safety procedures in the mines and the culture of violence from housing in ethnically segregated single sex hostels. During this period mining medicine improved to sustain productivity, although it was widely perceived by black miners as yet another means to repress the African persona (Butchart, 1996).

Black miners in RSA comprise approximately 85% of all gold miners (Murray *et al.*, 1996). Between 1975 and 1991, and based on 16 454 case histories, the prevalence of tuberculosis (TB) increased from 0.9% in 1975 to 3.9% in 1991; for silicosis, these values were 9.3% in 1975 and 12.8% in 1991. The frequency of both diseases increased with age and duration of service. Silicosis was the most significant predictor of TB. Lowering of dust levels in the mines was recommended to prevent an increased disease burden (Murray *et al.*, 1996). In a 7-year study, it was shown that miners with chronic simple silicosis had a nearly three-fold greater risk of developing TB than did their fellow workers of similar age who did not show radiographic evidence of silicosis at the start of the study; about 25% of the miners with silicosis will have developed TB by age 60 years (Cowie, 1994). Death rates of black RSA gold miners from pulmonary TB and silicosis were higher than those from their white counterparts, possibly because of greater severity of silicosis and a high rate of HIV infection (Hnizdo and Murray, 1998). By 1996, the death rate from tuberculosis among black migrant miners had risen to 2476 per 100 000, accounting for the largest single cause of death among this group, apart from trauma in the workplace (Churchyard *et al.*, 1999). Concomitantly, HIV prevalence in RSA miners with TB increased from 15% in 1993 to 45% in 1996; HIV is known to interfere with the accuracy of radiological TB screening programs. TB is likely to remain the most important health hazard in RSA mines during the new millennium, necessitating greater commitment to TB control and reduction of risk factors, such as silicosis and HIV infection (Churchyard *et al.*, 1999). The role of HIV, a retrovirus that infects human T cells and causes acquired immune deficiency syndrome (AIDS) – a condition of deficiency of certain leucocytes resulting in infections and cancer – is discussed later.

During the periods 1980–1989 and 1990–1994, cancer deaths of black male gold miners in the early period were primarily due to liver cancer followed by oesophageal and lung cancers (Boffetta *et al.*, 1994; McGlashan and Harington, 2000). Primary liver cancer during this period was the fourth leading cause of death in the RSA, but first among black gold miners who worked underground (McGlashan and Harington, 2000). In the period 1990–1994, oesophageal cancer had overtaken liver cancer in number of deaths. New cases of oesophageal cancer had doubled. New cases of respiratory cancer had also doubled. The reasons for these trends are uncertain but may be associated with repatriation of transient workers to their homelands outside RSA where health care was not as extensive (McGlashan and Harington, 2000). In another study, pulmonary dysfunction was measured in black South African gold miners with reactive airways (Cowie, 1989). Reactive airways were found in 12% of 1197 older miners, and were not related to extent of exposure to the underground environment. However, those so afflicted

were more susceptible to bronchial tree problems after correction for age, tobacco smoking, and presence of silicosis.

White South African miners who had spent at least 85% of their working life in gold mines and had worked underground at least 15% of their shifts, had a 30% chance of dying sooner than the general population due to higher frequencies of lung cancer (140%), heart disease (124%), pulmonary disease (189%), and cirrhosis of the liver (155%). However, very little of this increase could be attributed to gold mining and was instead associated with their unhealthy life style when compared to other South African white males, particularly in smoking and excessive alcohol consumption (Reid and Sluis-Cremer, 1996). There is, however, growing evidence that white RSA gold miners – like their black counterparts – were also vulnerable to silicosis (Hnizdo and Sluis-Cremer, 1993), emphysema (Hnizdo *et al.*, 2000), lung cancer (Hnizdo *et al.*, 1997), asthma (Cowie and Mabena, 1996), and PTB (Hnizdo and Murray, 1998).

RSA gold miners have among the highest rates of TB in the world. This is attributed, in part, to the high endemic rate of TB in rural regions from which miners are recruited, crowding, silica dust exposure, increasing age of the workforce, and HIV infection. Rates are rising, despite cure rates that meet WHO targets in patients with new TB (Godfrey-Faussett *et al.*, 2000). The incidence of PTB in RSA gold miners increased from 686 per 100 000 workers in 1989 to more than 1800 per 100 000 in 1995 (Sonnenberg *et al.*, 2000). Changes were associated with longer service and a rise in the average age of the work force. Miners with pulmonary mycobacterial disease were more likely to have non-tuberculosis mycobacteria (NTM) than *Mycobacterium tuberculosis* (TB) if they worked longer underground, had silicosis, or were treated previously for TB. Attempts to reduce the incidence of all pulmonary mycobacterial disease among gold miners should include early diagnosis and treatment (Sonnenberg *et al.*, 2000). Despite a control program that cures 86% of new cases, most TB in this mining community is due to ongoing transmission from persistently infectious individuals who have previously failed treatment and may be responsible for as many as one-third of TB cases (Godfrey-Faussett *et al.*, 2000). There is a low incidence of NTM isolates and diseases in developed countries; however, this incidence is 27% in RSA miners (Corbett *et al.*, 1999a), and is largely attributable to chronic chest disease from silicon dust inhalation and prior tuberculosis (Corbett *et al.*, 1999c). Previous studies have shown that isolates of the most common NTM species, *M. kansasii* and *M. scrofulaceum*, occur with high incidence and are often associated with NTM risk factors such as silicosis and lung diseases than either patients with TB or control patients (Corbett *et al.*, 1999a). NTM were isolated from 118 patients during the study period of whom 40 (34%) were HIV positive (Corbett *et al.*, 1999a). HIV infection has recently become an additional risk factor for mycobacterial disease in miners and is likely to become increasingly important as the HIV epidemic progresses (Corbett *et al.*, 1999c).

Silicosis reflects a failure in adequate control of occupational dust exposure (Cowie, 1998). The risk of silicosis in a cohort of 2235 white RSA gold miners, with an average of 24 years of mining experience between 1940 and 1970, was followed up to 1991 for radiological signs of onset of silicosis (Hnizdo and Sluis-Cremer, 1993). About 14% of the miners developed silicosis at an average age of 56 years, with radiological signs appearing, on average, 7.4 years after mining exposure ceased. The risk of silicosis was strongly dose-dependent, although the latency period was variable. Silicosis risk increased exponentially with the cumulative dust dose, the accelerated increase occurring after 7 mg m^3 years. At the highest exposure level of 15 mg m^3 years – equivalent to about 37 years of gold mining exposed to an average respirable dust concentration of 0.4 mg m^3 – the cumulative risk for silicosis reached 77% (Hnizdo and Sluis-Cremer, 1993). There is also a positive association between exposure to silica dust and risk of lung cancer (Boffetta *et al.*, 1994); risks were higher among those exposed to higher dust exposures and also diagnosed with silicosis (Hnizdo *et al.*, 1997). Miners who had withdrawn from dusty occupations showed declines in lung function similar to those who continued to work underground for 5 years (Cowie, 1998). RSA gold miners with chronic obstructive airway disease from working in a dusty atmosphere in scheduled mines or works were entitled to workmen's compensation, as judged by lung function tests for airflow obstruction (Hnizdo *et al.*, 2000).

The association between silicosis and PTB is well established (Hnizdo and Murray, 1998). Epidemiological and case studies show that workers exposed to silica dust have increased morbidity and mortality from PTB. In one study, a cohort of 2255 white RSA gold miners were evaluated for increased risk of PTB from 1968 to 1971 when they were 45–55 years of age to December 1995. During the follow-up, 1592 (71%) of this cohort died. Of these, 1296 (81%) were necropsied to determine the presence of silicosis and PTB. It was concluded that exposure to silica dust is a risk factor for the development of PTB in the absence of silicosis, even after exposure to silica dust ends; the risk of PTB increases with the presence of silicosis, and in miners without silicosis with increasing exposure to dust; and severity of silicosis was associated with increasing risk of PTB (Hnizdo and Murray, 1998). In addition to silicosis, TB, and obstructive airways disease, RSA gold miners show a high prevalence of previously undiagnosed and untreated pneumoconiosis, a lung disease caused by habitual inhalation of irritant mineral or metallic particles (Trapido *et al.*, 1998).

South Africa currently harbours one of the fastest-growing HIV epidemics in the world (Bredell *et al.*, 1998). The prevalence of HIV-1 in pregnant women has increased from 0.76% in 1990 to 14.1% in 1996, with more than 2.5 million South Africans infected. Migrant workers employed as RSA gold miners were found infected with HIV-1 (Bredell *et al.*, 1998). HIV infection and silicosis are powerful risk factors for TB and are associated with an increased risk of death among RSA gold miners. The incidence of TB was almost five times greater in HIV-positive than HIV-negative miners (Corbett *et al.*, 2000). Among RSA gold miners

with TB, the prevalence of HIV infection increased rapidly to about 50% of all cases between 1993 and 1997 (Churchyard *et al.*, 2000). NTM disease incidence, morbidity, and mortality are likely to increase further among miners as the HIV epidemic progresses (Corbett *et al.*, 1999b).

RSA gold miners have a high prevalence of HIV infection (Campbell, 1997). Most are migrants from rural areas within South Africa and others are from surrounding countries such as Lesotho, Botswana, and Mozambique. The vast majority of these workers are housed in single sex hostels close to their workplace. Despite extensive education from mine operators on the consequences of unprotected sex, this group perceives condom use as a diminishment of their masculinity and continue to practice risky behaviours with sex workers, and the incidence of sexually transmitted diseases in these men is extremely high (Corbett *et al.*, 1999c). Many workers commented that the risk of HIV/AIDS appear minimal compared to the risks of death or injury underground and that this was the reason why many mine workers did not bother with condoms. It remains unclear how best to communicate risks of HIV and prevent transmission by altering risky behaviours in African populations (Campbell, 1997).

4. Health risks: surface miners who use mercury

In general, mercury concentrations in drinking water, soils, sediments, tailings, or edible fish near gold mining operations exceeded national and international limits in the Philippines (Appleton *et al.*, 1999; Akagi *et al.*, 2000), Kenya (Ogola *et al.*, 2002) and Brazil (Malm *et al.*, 1990, 1995b), but not in fish diets of farmers and miners in Tanzania (van Straaten, 2000), and Columbia (Olivero *et al.*, 1997). Selected case histories of mercury intoxication associated with gold mining activities are shown for Australia, the Philippines, Brazil, and Venezuela. Measured mercury concentrations in hair, urine, blood and other human tissues in the vicinity of gold amalgamation extraction and refining activities are shown together with similar data for ambient air and fish diets.

4.1. CASE HISTORIES

Most countries recognise mercury hazards to health, although occupational health problems and direct inorganic mercury toxicosis rarely appear (Porcella *et al.*, 1997). The following case histories provide anecdotal evidence for mercury poisoning associated with the amalgamation process, although large-scale epidemiological evidence for the association does not exist.

A 19-year old male in Queensland, Australia, developed hand tremors and fatigue after starting work at a placer gold mine where he was exposed to Au–Hg amalgam (Donoghue, 1998). His mercury urine level of 143 $\mu g\,l^{-1}$ exceeded the recommended no-adverse-effect-level of 50 $\mu g\,l^{-1}$. Seven weeks after removal from the work environment, his mercury urine level had fallen to 32 $\mu g\,l^{-1}$ and the

tremors had almost disappeared. Contaminated air was the source of the exposure. Smelting of retorted gold with previously unrecognised mercury resulted in peak air levels of 0.53 Hg m^3 (*v* a recommended threshold level of 0.05 mg Hg m^3). Engineering and procedural controls were instituted to prevent further occurrences (Donaghue, 1998).

In 1987, 11 Filipinos became ill and one died after 8 h spent blowtorching about 2 kg of a Au–Hg amalgam indoors (Greer, 1993; de Lacerda and Salomons, 1998), strongly indicating the need to burn amalgam and melt gold in closed vessels (Drasch *et al.*, 2001). It was alleged that Filipinos who lived within 500 m of a similar mercury source for 30 months had a 75% probability of exhibiting symptoms of clinical mercury poisoning (Greer, 1993). Latent effects on health of mercury exposure in Philippine gold miners were attributed to occupational exposure during the 1960s, 30 years earlier (Akagi *et al.*, 2000). The United Nations Environment Program recommends the banning of elemental mercury in amalgamation processes (Greer, 1993). But in the Philippines and elsewhere, little appears to have been accomplished by attempts to educate miners about mercury's hazards or by regulations concerning its use as long as the price of gold makes extraction and processing economically viable (Greer, 1993).

In another case from the Philippines, school children from an area of intense mercury amalgamation activities were frequently under height, under weight, and presented with gum discolourations and skin abnormalities (Akagi *et al.*, 2000). Blood mercury concentrations were elevated (up to 57 μg total Hg l^{-1}; 47 μg methylmercury l^{-1}), as were mercury concentrations in hair (up to 20 mg total Hg kg^{-1} dry weight (DW); up to 18 mg methylmercury kg^{-1} DW). Symptoms were attributed to ingestion of mercury contaminated fish containing up to 0.44 mg total Hg kg^{-1} fresh weight (FW) muscle, or 0.38 mg methylmercury kg^{-1} FW (Akagi *et al.*, 2000).

In Brazil, armed force by the military has proven ineffective to stop the illegal use of mercury by more than an estimated 1 million gold miners (Veiga *et al.*, 1995). It is postulated that mercury pollution is sufficiently severe to cause adverse effects on brain development, as evidenced by elevated (>10 mg kg^{-1}) mercury concentrations in hair of Brazilian children ages 7–12 (Grandjean *et al.*, 1999). About 80% of the children examined had >10 mg Hg kg^{-1} hair. Decreasing test scores of children subjected to neuropsychological tests of motor function, attention span, and visuospatial performance were associated with increasing hair mercury levels (Grandjean *et al.*, 1999). Mercury discharges from informal gold mining activities in the Amazon region is attributed to a lack of concern of the miners for the environment as well as poor knowledge about efficient gold extraction techniques. Moreover, individuals in contact with these miners, including priests, physicians, hygienists, social workers, nurses, mining inspectors, union personnel, and others, also lack information about mercury transformations in the environment. Education on the toxicological properties and its transformation products is strongly recommended as a viable solution to reducing emissions and

for creating opportunities for illegal miners to form their own legal companies (Veiga and Meech, 1995; Veiga *et al.*, 1995). Current approaches to protect human health in Brazil against mercury intoxication include prohibition of elemental mercury in gold mining activities and the temporary suspension of gold mining operations (Malm *et al.*, 1995a). But these solutions are not realistic for economic and political reasons. Minimising mercury emissions and limiting consumption of larger carnivorous fish meals seem reasonable recommendations (Malm *et al.*, 1995a).

Venezuelan gold miners ($N = 40$) exposed to elevated air concentrations of mercury exceeded hair and urine mercury occupational exposure guidelines (Rojas *et al.*, 2001). Overall, mercury guidelines for air were exceeded in 18% of the measurements, in urine 48% of the cases, and in hair 24% of the individuals. Despite substantial occupational exposure to mercury for some individuals, few adverse health effects were observed that were clearly related to mercury exposure (Rojas *et al.*, 2001).

4.2. MERCURY IN TISSUES

Data from gold miners and gold mining communities show conclusively that mercury concentrations in urine, blood, hair, and breast milk exceed by a wide margin the most conservative criteria of mercury contamination proposed by various national and international regulatory agencies. However, the large variability in concentrations of mercury in blood, urine, and hair among individuals exposed to mercury vapour, inorganic mercury, and methylmercury, do not seem to adequately monitor mercury burdens of brain and other target tissues (Drasch *et al.*, 2001).

In urine, mercury concentrations from different gold mining sites and groups occupationally exposed to mercury emissions frequently exceeded the WHO levels of $50\,\mu g\,l^{-1}$, the maximum acceptable concentration, and $100\,\mu g\,l^{-1}$, the minimum concentration before developing symptoms of mercury poisoning (de Kom *et al.*, 1998; de Lacerda and Salomons, 1998; Drake *et al.*, 2001; Drasch *et al.*, 2001). Workers in gold trader shops at three Brazilian locations had maximum levels of 79, 160, and $1168\,\mu g\,Hg\,l^{-1}$ urine, respectively (Malm *et al.*, 1995b; de Lacerda and Salomons, 1998). About 22% of all Brazilian gold shop workers exposed to mercury vapours had urinary levels in excess of $50\,\mu g\,Hg\,l^{-1}$ (Santa Rosa *et al.*, 2000). In Tanzania, where it is estimated that 250 000 people are involved in small scale gold mining using mercury amalgamation, mercury levels in urine of miners reached $241\,\mu g\,l^{-1}$ *v* $3\,\mu g\,l^{-1}$ in a control group (Ikingura and Akagi, 1996); about 36% of Tanzanian gold miners working with amalgam exceeded the WHO guideline for mercury in urine (van Straaten, 2000). Elevated mercury levels in urine were positively correlated with increasing fish consumption, alcohol consumption on a yearly basis, number of hours worked daily, and number of dental amalgam fillings (Santa Rosa *et al.*, 2000). In China, mean mercury concentrations in urine of villagers from a gold mining community ranged between 38 and $87\,\mu g\,l^{-1}$, reaching maximum concentrations of $540\,\mu g\,l^{-1}$ in boys under 16,

418 $\mu g\,l^{-1}$ in adult women, 290 $\mu g\,l^{-1}$ in adult men, and 195 $\mu g\,l^{-1}$ in girls under 16; for the entire population, 91% exceeded 50 $\mu g\,Hg\,l^{-1}$ urine (Lin *et al.*, 1997).

In hair, mercury concentrations, as methylmercury, should not exceed 4–7 $mg\,kg^{-1}$; higher concentrations of 10–20 $mg\,kg^{-1}$ are associated with abnormal infant development, and 50–100 $mg\,kg^{-1}$ with paraesthesia (de Lacerda and Salomons, 1998). In Brazilian gold mining populations that consume more than 100 g of fish daily, levels of methylmercury in hair of women of child-bearing age should be monitored because 83% exceeded 10 mg/kg DW and 8.3% exceeded 30 mg methylmercury/kg hair (Kehrig *et al.*, 1997). Elevated concentrations of total mercury (25–37 $mg\,kg^{-1}$) were observed in hair of inhabitants from fishing villages near gold mining areas but not in gold miners (4.1 $mg\,kg^{-1}$); more than 90% of the total mercury in hair from both groups was in the form of methylmercury (Akagi *et al.*, 1995a). To reach 50 mg total $Hg\,kg^{-1}$ hair – the recommended maximum acceptable limit – it is necessary to consume 330 g of fish muscle daily containing 1 $mg\,Hg\,kg^{-1}$ FW (Aula *et al.*, 1994). Total mercury levels in hair, up to 71 $mg\,kg^{-1}$, are recorded in Brazilian gold mining areas, and up to 176 $mg\,kg^{-1}$ in fishing villages from consumption of mercury-contaminated fish (Malm *et al.*, 1995a, 1997; Akagi *et al.*, 1995b; Kehrig *et al.*, 1997; Lacher and Goldstein, 1997). In one case, a hair mercury level of 240 $mg\,kg^{-1}$ was measured in a fisherman who consumed 14 fish meals weekly from fish captured in an artificial reservoir that received gold mining wastes *v* 8.5 $mg\,kg^{-1}$ in a nearby population that consumed, on average, only two fish meals weekly (Leino and Lodenius, 1995). Elevated ($>10\,mg\,kg^{-1}$) hair mercury concentrations attributed to gold mining activities, especially to consumption of mercury-contaminated fish, are reported in Bolivia (Maurice-Bourgoin *et al.*, 2000), Tanzania (Harada *et al.*, 1999), the Philippines (Appleton *et al.*, 1999; Akagi *et al.*, 2000; Drasch *et al.*, 2001), French Guiana (Frery *et al.*, 2001), and Columbia (Olivero *et al.*, 1998). In French Guiana, for example, 57% of the native Amerindians had hair mercury concentrations $>10\,mg\,kg^{-1}$, the WHO recommended limit (Frery *et al.*, 2001). Incidentally, some Tanzanian subjects who showed a high total mercury level in hair made habitual use of toilet soap containing about 2% mercuric iodide (Harada *et al.*, 1998; van Straaten, 2000), and this needs to be considered in evaluating mercury risk assessment in gold mining areas. To reduce mercury loadings in the hair of gold miners, fisherman, and their families, a change in diet is recommended from predatory to herbivorous fishes, and a reduction in mercury emissions from current gold mining practices (Leino and Lodenius, 1995).

Blood mercury concentrations in gold miners are primarily related to exposure to metallic mercury vapour in the air and to consumption of mercury-contaminated fish diets (Barbosa, 1997). Blood mercury levels greater than 10 $\mu g\,l^{-1}$ (the recommended maximum level) were exceeded by 33% of gold prospectors in the Brazilian Amazon (de Lacerda and Salomons, 1998). Gold miners and refiners contained 25 $\mu g\,Hg\,l^{-1}$ (8–159) blood when exposure to mercury was within the past 2 days; blood mercury levels were 7.6 $\mu g\,l^{-1}$ (2.2–19.4) when this group

had been exposed 2–60 days previously; and 5.6 $\mu g\,l^{-1}$ (3–14) when exposed to mercury vapours more than 60 days earlier (Aks *et al.*, 1995). Blood mercury concentrations up to 108 $\mu g\,l^{-1}$ were measured in ball mill workers from gold mining areas of Mindanao, Philippines (Drasch *et al.*, 2001), and up to 57 $\mu g\,l^{-1}$ (47 $\mu g\,l^{-1}$ as methylmercury) in school children age 5–17 years studying near 11 Philippine gold mills and processing plants (Akagi *et al.*, 2000).

In breast milk of nursing mothers, ingestion of mercury-contaminated diets as a result of gold mining activities in Amazonia is alleged to be the cause of elevated mercury concentrations (Barbosa and Dorea, 1998). Concentration of total mercury in breast milk from nursing mothers in the Amazon Basin, Brazil, ranged from 0.0 to 24.8 $\mu g\,kg^{-1}$ with a mean of 5.8 $\mu g\,kg^{-1}$ (*v* 0.9 $\mu g\,kg^{-1}$ in USA). Infants fed milk from these mothers may receive more than 0.5 $\mu g\,Hg\,kg^{-1}$ body weight (the tolerable daily mercury intake recommended for adults by the WHO) in 53% of the infants measured. The mercury concentration in breast milk was not significantly correlated with mercury content in hair of the mother or infant (Barbosa and Dorea, 1998).

4.3. MERCURY IN AIR AND IN FISH DIET

Air and fish diet – the main routes of mercury entry into gold miners – are typically grossly contaminated with mercury. In Brazil, for example, more than 130 t of mercury are discharged into the biosphere each year as a result of gold mining activities; 55% is discharged into the atmosphere and 45% into aquatic ecosystems (Frery *et al.*, 2001). Within the biogeochemical cycle of the metal, Hg^0 can be oxidised to inorganic Hg^{2+} and then methylated by biotic (bacteria) and abiotic (humic acids as methyl group donors) processes (Eisler, 2000b; Frery *et al.*, 2001).

Inhalation of metallic mercury vapour is the main route of human occupational exposure, and gold shop workers are at the greatest risk (Malm *et al.*, 1995b; Hacon *et al.*, 1997). Concentrations of mercury in ambient air of gold dealer shops and workplaces in gold mining areas exceeded the generally recommended exposure level of 50 $\mu g\,Hg\,m^3$ in the Philippines (1664 $\mu g\,m^3$), Brazil (292 $\mu g\,m^3$), and elsewhere (de Lacerda and Salomons, 1998). In Venezuela, where ambient air concentrations of occupational gold workers reached a maximum of 6315 $\mu g\,m^3$ and averaged 183 $\mu g\,m^3$, about 20% of the exposures exceeded 50 $\mu g\,m^3$ – the recommended exposure limit of the US National Institute of Occupational Safety and Health – and 26% exceeded 25 $\mu g\,m^3$ – the threshold limit value recommended by the American Conference of Governmental Industrial Hygienists (Drake *et al.*, 2001). About 72% of Philippine workers who refined amalgam through burning were classified as mercury-intoxicated (Drasch *et al.*, 2001). In Dexing County, Jiangxi Province, China, about 200 small scale gold mines were in operation between 1990 and 1995 using mercury amalgamation to extract gold (Lin *et al.*, 1997). Gold firing was usually conducted in private residences with excessive

mercury contamination of the air in workrooms (up to 2600 $\mu g\, m^3$) and workshops (up to 1000 $\mu g\, m^3$). Since September 1996, most small scale gold mining activities were prohibited through China's national environmental legislation (Lin *et al.*, 1997).

In the Mato Grosso's Pantanal, the world's largest wetlands, fish muscle contained mercury concentrations up to 24 times higher than the level considered safe (<0.5 mg total $Hg\, kg^{-1}$ FW muscle) for human consumption by the WHO (Greer, 1993). Mercury-contaminated fish were detected up to 590 km downstream from some gold mining areas (Greer, 1993). In the Amazonian region of Bolivia, regular consumption of fish contaminated by mercury from gold mining activities is considered to be a major threat to public health (Bidone *et al.*, 1997; Maurice-Bourgoin *et al.*, 2000). Mercury concentrations in muscle of edible fish collected near various mercury-amalgamation gold mining sites were routinely elevated in Columbia (Olivero *et al.*, 1998), French Guiana (Frery *et al.*, 2001), the Philippines (Appleton *et al.*, 1999), and Brazil (Malm *et al.*, 1990, 1995a, 1997; Akagi *et al.*, 1995a,b; Porvari, 1995; Bidone *et al.*, 1997). Mercury concentrations in muscle of Brazilian fishes sampled in 1997–1998 were highest in carnivorous species, lowest in herbivores, and intermediate in omnivores; concentrations were lower with increasing distance from gold mining areas (Aula *et al.*, 1994; Lima *et al.*, 2000). In some areas of the Amazonian region of Brazil, the estimated intake of mercury from fish consumption is 114 μg daily, or about 3–4 times higher than the daily tolerable intake recommended by the WHO; the estimated exposure level of 1.6 $\mu g\, Hg\, kg^{-1}$ body weight for adult consumers was nearly 5 times higher than the recommended consumption limit of 0.3 $\mu g\, Hg\, kg^{-1}$ BW daily (Bidone *et al.*, 1997). Variations in mercury content of muscle from some Amazon fish species could not be related to nearby gold mining activities (Jernelov and Ramel, 1994).

5. Recommendations

To protect the health of underground workers, authorities recommend continued intensive monitoring of atmospheric dust levels in order to conform to recognised safe occupational levels, implementation of more frequent medical examinations with emphasis on early detection and treatment of disease states, and continuation of educational programs on hazards of risky behaviours outside of the mine environment; these recommendations can be implemented satisfactorily through mine management. Miners who use elemental mercury to extract gold need to control mercury emissions in confined environments, limit consumption of larger carnivorous fishes, and be made cognisant of mercury hazards through educational programs administered by informed nurses, social workers, clergy, union personnel, and others. Intensive monitoring by physicians and toxicologists of populations at high risk from mercury poisoning is strongly recommended in order to provide

evidence of adherence to existing criteria, as is re-examination of current mercury criteria to protect human health.

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