

Chemical hazards in the artisanal gold sector

Impacts of mercury, cyanide and silica dust
on human health and environment



Understanding occupational health risks in the artisanal gold sector is an important prerequisite for miners to protect themselves and their communities, and to transition to environmentally sound and safe practices.



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Occupational health risks in the artisanal gold sector

The Artisanal and Small-scale Gold Mining (ASGM) sector typically consists of small groups or individuals extracting gold with semi-mechanized and low-tech methods. Mining sites tend to have poor occupational safety standards and practices exposing miners to various occupational health hazards. Chemical risks include the exposure to toxic substances such as mercury, cyanide and silica dust. Other occupational health risks include painful disorders of the muscles and skeleton (e.g. from heavy lifting), physical trauma (e.g., from slipping, falling, landslides) or exposure to noise, heat and humidity. Furthermore, poor living conditions in artisanal mining communities can increase the susceptibility to communicable diseases such as cholera, malaria or tuberculosis.

This brochure provides a brief overview on the health and environmental impacts of mercury, cyanide and silica dust in the sector. Chemical risks do not only affect miners, but also their communities. Mercury and cyanide are both highly toxic to human health. However, only mercury persists in the environment and can travel long distances in the environment. Exposure to very fine silica dust is a common hazard in hard rock mining, but one that can easily be prevented.

Mercury



Figure 1: Elemental mercury (photo: AGC)

Elemental mercury, a silver liquid (fig. 1), is a highly toxic heavy metal that cannot be destroyed and persists in the environment. It is a deficient but simple and cheap method to extract gold. Miners in artisanal gold mining are exposed to elemental mercury during amalgamation (mixing ore with mercury), squeezing (separating solid amalgam from excess mercury), vaporization (heating of amalgam), smelting (melting sponge gold with residual mercury), and during refining of raw gold doré.

Due to its high volatility, elemental mercury can transform from its liquid state to vapour even at room temperature. The vapour is odorless and colorless, but extremely toxic and very adhesive. Therefore, inhaling elemental mercury vapours during ore processing, and most intensively when burning amalgam, is the most dangerous form of exposure. Lungs retain around 80% of the inhaled vapours, from where the mercury enters the bloodstream. Furthermore, mercury vapour can adsorb to clothes, skin, furniture, work tools and walls. These materials can continue to emit high concentrations of mercury for extended periods of time. Hence, the whole household gets exposed to mercury vapours when mercury is vaporized within the home or if work clothes and work equipment are stored at home.

If liquid elemental mercury is accidentally ingested or comes into contact with unprotected skin, only a very small amount gets absorbed by the body (less than 3%), but the exposure can cause skin and eye irritation.

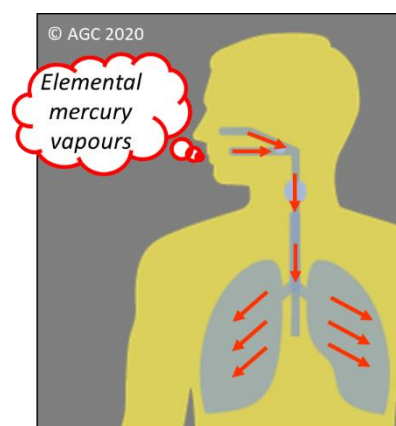


Figure 2: Elemental mercury is mainly absorbed by the lungs

Health data for artisanal gold mining communities is scarce and the prevalence and severity of mercury intoxication differs depending on local practices. According to a global study, one in three active miners and gold shop owners suffers from chronic elemental mercury vapour intoxication.

Mercury in the environment

The ASGM sector is responsible for around 38% of the global mercury emissions to the atmosphere originating from human activity. Mercury in the atmosphere can travel over long distances. For example, an estimated 95% of anthropogenic mercury deposited in Canada comes from sources outside Canada, mainly East Asia. Elemental mercury is not only emitted to the atmosphere during ore processing or from tailings, it is often also directly released into water streams and soils (e.g., from poorly made tailings ponds). Most of the mercury released into the environment will eventually enter aquatic systems (streams, lakes, rivers and oceans) where bacteria can transform it into methylmercury, the most toxic form of mercury (fig. 3, step 2&3). The transformation rate of elemental mercury into methylmercury depends on many different physical, chemical, and biological factors such as presence of specific bacteria, temperature and water flow. Therefore, it is difficult to

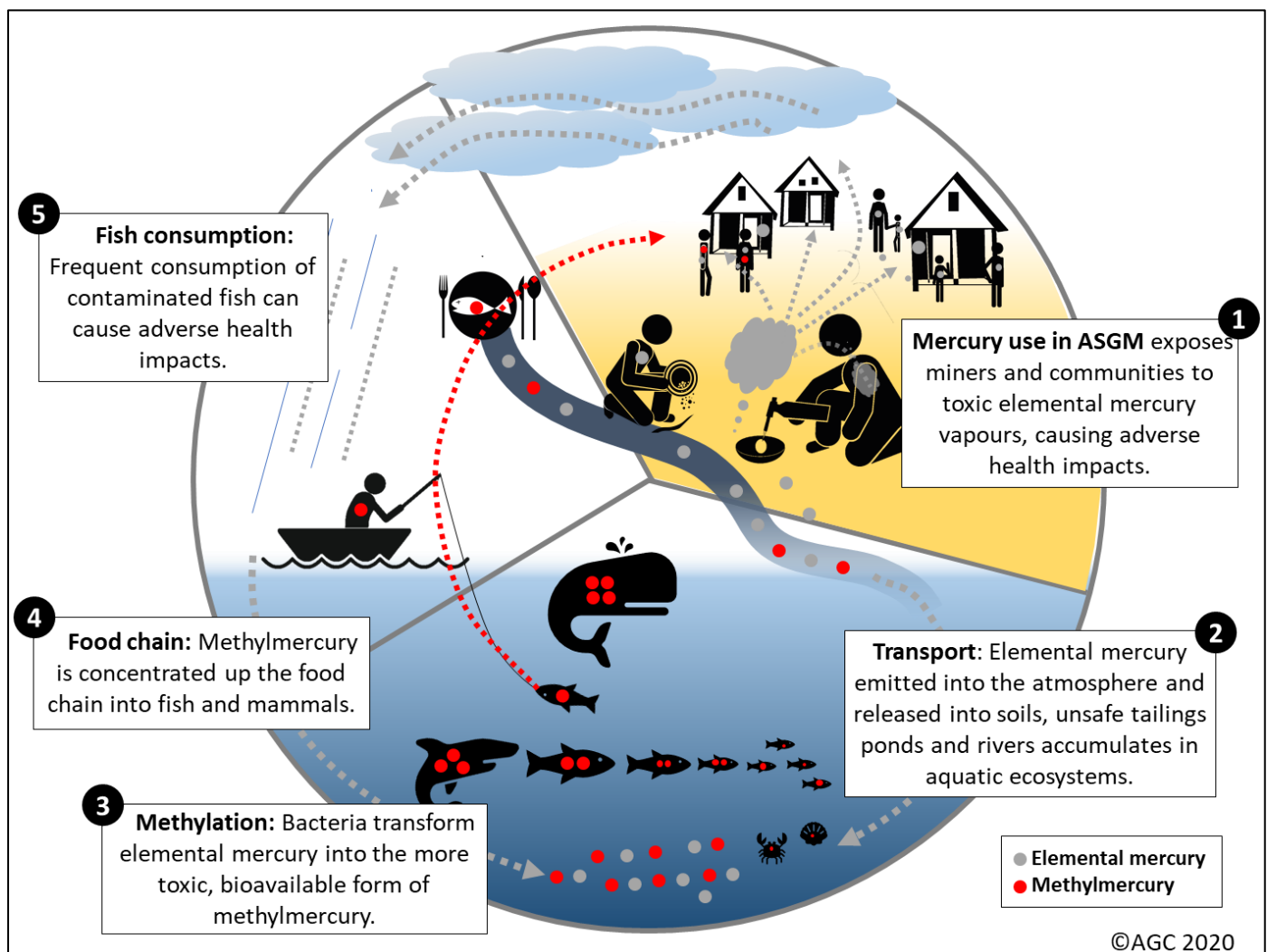


Figure 3: Impacts of mercury use in ASGM on human health and the environment: Mercury persists and travels in the environment, affecting not only miners and their communities locally but also the global environment

predict how much and how fast elemental mercury will be transformed into the more toxic and bioavailable form of methylmercury. Once methylated, the toxins are absorbed by plankton and methylmercury is concentrated up the food chain from plankton to shellfish (e.g., mussel, shrimp, crab), to small fish, to larger predatory fish (e.g., bass, tuna, shark) and mammals (e.g., otters, whales).

Health effects of mercury exposure

Mercury intoxication can appear in three forms: as (1) acute or (2) chronic elemental mercury vapour intoxication in artisanal gold mining, or (3) as chronic methylmercury intoxication.

(1) Acute elemental mercury intoxication in artisanal gold mining

A miner or bystander can suffer from acute elemental mercury intoxication, if the person inhales very high concentrations of elemental mercury vapours during a vaporization event, causing damage primarily to the lungs and the digestive system.

Most symptoms usually develop within a few hours after exposure, starting often with gastrointestinal symptoms (nausea, vomiting). The vapours can irritate the lips and mouth (*stomatitis*), the gums (*gingivitis*) as well as the respiratory tract, causing a burning sensation in the lungs (sore throat), a tightness in the chest, shortness of breath (*dyspnea*) and coughing.

Mercury vapours can also cause an inflammation of lung tissue (*chemical pneumonitis*) or *edema* (swelling caused by excess fluid) with a potentially fatal collapse of the lungs. Additional symptoms reported include excessive salivation, fever, chills, headache, weakness, high protein levels in urine (*proteinuria*), high blood pressure (*hypertension*), irritation of eyes (*uveitis*) and skin as well as vomiting and diarrhea that can lead to shock due to dehydration. Damage to the kidneys that can lead to fatal kidney failure has also been reported.

In survivors, the acute mercury induced lung damage usually resolves completely, but long-term problems such as lung restrictions have been reported. The absorbed mercury can cross the blood-brain barrier which is a highly selective semipermeable border of cells that prevents solutes in the blood from entering the nervous system. Mercury can enter the nervous system and cause symptoms of chronic intoxication (e.g. neurological effects) several days after the initial exposure.

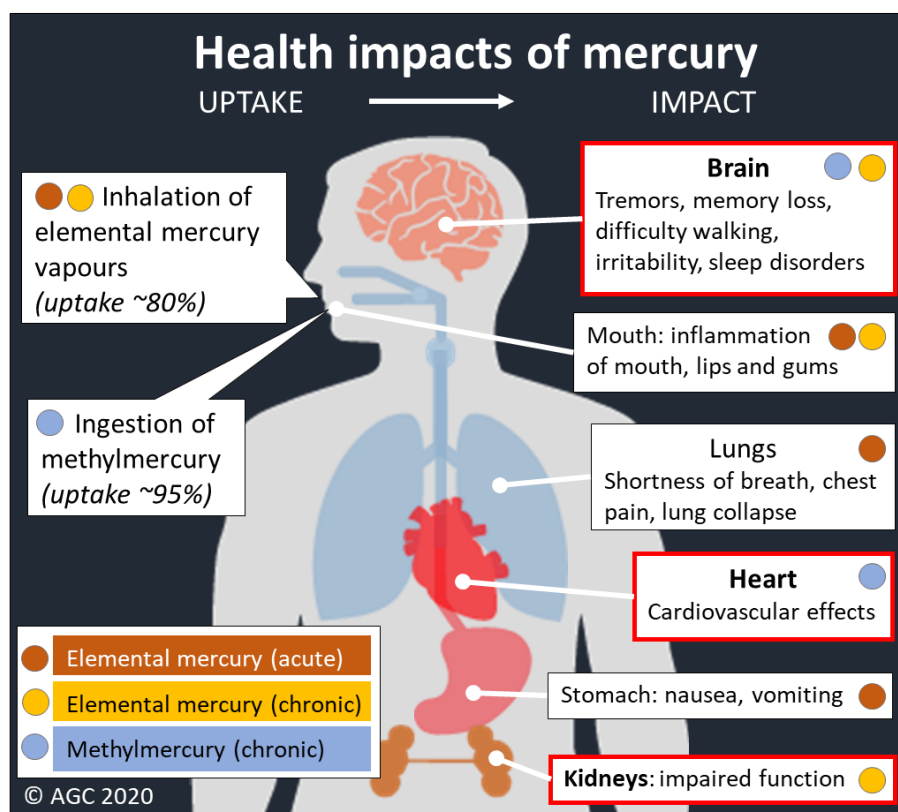


Figure 4: Major health impacts of mercury intoxication

(2) Moderate & severe chronic elemental mercury intoxication

This form is most prevalent in artisanal mining communities and is caused if a miner or bystander is regularly exposed to elemental mercury vapours over an extended length of time. Mercury accumulates in the body and can cause moderate to severe symptoms of chronic elemental mercury intoxication over time (fig. 4). Chronic intoxication mainly affects the brain and the kidneys. Neurological symptoms include tremors in several parts of the body, lack of coordination and movement control, memory impairment, shyness, sleep disorders, blurred vision and sensory disturbances. Damages to kidneys such as proteinuria is more difficult to observe; it can become apparent in symptoms such as swollen hands or feet and blood in the urine. In addition, irritation of the mouth or the gums, loss of appetite and salivation, fatigue and weakened immune systems have also been reported. People with moderate levels of intoxication usually have moderate pain levels and can perform regular activities. However, people with severe intoxication are not able to perform regular activities (e.g., buttoning a shirt because of tremors) and can have severe problems in cognitive functions (e.g. paying attention or memorizing things).

Though some of the symptoms described can occur immediately after the exposure or a few days later, chronic elemental mercury intoxication usually has a slow onset, with symptoms presenting up to 5 to 10 years later. Some organ damage can be wholly or partially reversible, if further exposure to mercury stops. The body excretes elemental mercury mainly through the urine and it takes around 60 to 90 days for the mercury body burden to fall to half (half-life). The risk of permanent organ damage increases with the length and magnitude of elemental mercury exposure.

(3) Health impacts of chronic methylmercury intoxication through fish consumption

When a person consumes contaminated fish or shellfish (> 0.5 mcg MeHg/g fish) on a regular basis over an extended period, methylmercury accumulates in the body and can cause chronic methylmercury intoxication. The uptake and effects of methylmercury in the body are influenced by several factors such as the exposure (frequency and dose of contaminated fish consumption), age (higher vulnerability of fetuses, infants and children) or genetics (causing different sensitivities to methylmercury). In addition, nutritional deficiencies (e.g., iron or folate) or alcohol consumption can increase the uptake, whereas some foods (e.g., omega-3 fatty acids, vitamin E, garlic) seem to attenuate the impact.

Frequent consumption of moderately to highly contaminated fish or shellfish causes methylmercury to accumulate in the brain, causing similar neurological symptoms as chronic elemental mercury exposure (tremors, lack of muscle coordination, behavioral, emotional and memory effects) (fig. 4). Methylmercury also affects the cardiovascular system and can cause high blood pressure, heart attack or coronary artery diseases. Other symptoms include headache, fatigue and reduced fertility in men and women.

It is unclear whether exposure to low doses of methylmercury is toxic in adults. Several countries and international organizations have issued guidelines on fish consumption to advise the general population about safe fish-eating practices. Swordfish, for example, has one of the highest average

concentrations of methylmercury (1 mcg/g fish), whereas tilapia is usually safe to consume (average 0.01 mcg/g fish). According to the Provisional Tolerable Weekly Intake (PTWI) of 1.6 mcg of mercury per kg of body weight, a person weighing 50 kg can safely consume up to 80 grams of swordfish or 8 kg of tilapia per week. This example shows how the contamination of fish depends on the species. Contamination levels of the same species, however, also vary between geographic catchment areas. The provision of public information on which fish to eat and how often in a geographic region is crucial.

Impact of mercury on developing fetuses, infants and children

Elemental and methylmercury have much higher detrimental effects on fetuses, infants, and small children than on adults because their central organs are still developing. Mercury can cross the placental barrier in the mother's womb. Because mercury accumulates in the body, exposure of women even before the beginning of the pregnancy can have negative impacts on the child.

Negative health outcomes from prenatal exposure to mercury depend on the exposure level and can range from high blood pressure, memory problems and deficits in fine motor skills to impaired movement and coordination problems (*cerebral palsy*), convulsions (*seizures*), severe intellectual disability, distortion of limbs, and death. Effects of prenatal exposure to mercury may also appear later on during child development, for example in the form of learning disabilities during school age. Most of the damage caused by mercury during prenatal exposure is permanent.



Figure 5: A child gets exposed to elemental mercury vapours during vaporization (photo: AGC)

The effects of mercury on infants and children are less known. Since children have less body mass and a proportionally higher lung volume, exposure to elemental mercury vapours or contaminated fish leads to a higher body burden compared to adults. If present during amalgamation (fig. 5), children can also be exposed to higher levels of mercury since they are closer to the ground and therefore often closer to flames and vapours, compared to adults. Children are also at a higher risk of playing with mercury or mercury contaminated items and can accidentally ingest or inhale it. Lastly, mercury can also be transferred from the mother to the child via breastfeeding. However, breast milk is important for the child's development and mothers should not be discouraged from breastfeeding.

The risk of permanent organ damage increases the earlier and the more infants and children are exposed to mercury. Furthermore, acrodynia ('pink disease') has been reported among small children. Acrodynia is characterized by body rash, swelling of extremities (*edema*), irritation of palms and soles, skin peeling (*desquamation*) of hands and feet, abnormal dermal sensation such as prickling (*paresthesia*), irritability, pain or discomfort due to the exposure of eyes to sunlight (*photophobia*), fever, insomnia and profuse sweating.

Treatment of mercury intoxication

The medical treatment options for the different forms of mercury intoxication are limited. The most important intervention is to stop any further mercury exposure at the mining site and/or through the consumption of high-risk fish. If required, supportive care can alleviate symptoms, for example the provision of supplemental oxygen for respiratory distress or physiotherapy for muscle control problems. In addition, clinical tests might be necessary to assess any organ damage (e.g. renal function test, electrocardiography (ECG)) and necessary treatments.

Chelation therapy is only advised to treat severe cases of acute elemental mercury intoxication. Chelating agents bind to mercury in the bloodstream and remove it from the body through the kidneys in the urine. However, the use of chelation therapy is controversial, and it is not advised for chronic intoxication with elemental mercury or methylmercury. Once mercury has accumulated in the organs, especially the brain, it is usually resistant to treatment with chelating agents.

Prevention at mining sites

Several interventions at work sites can protect miners and surrounding communities from mercury exposure or at least reduce the risks for human health and environmental contamination (fig. 6). These interventions reach from exposure prevention of most vulnerable groups (women at childbearing ages, infants and children), to exposure reduction, to eventually the elimination of mercury use through mercury free processing systems.

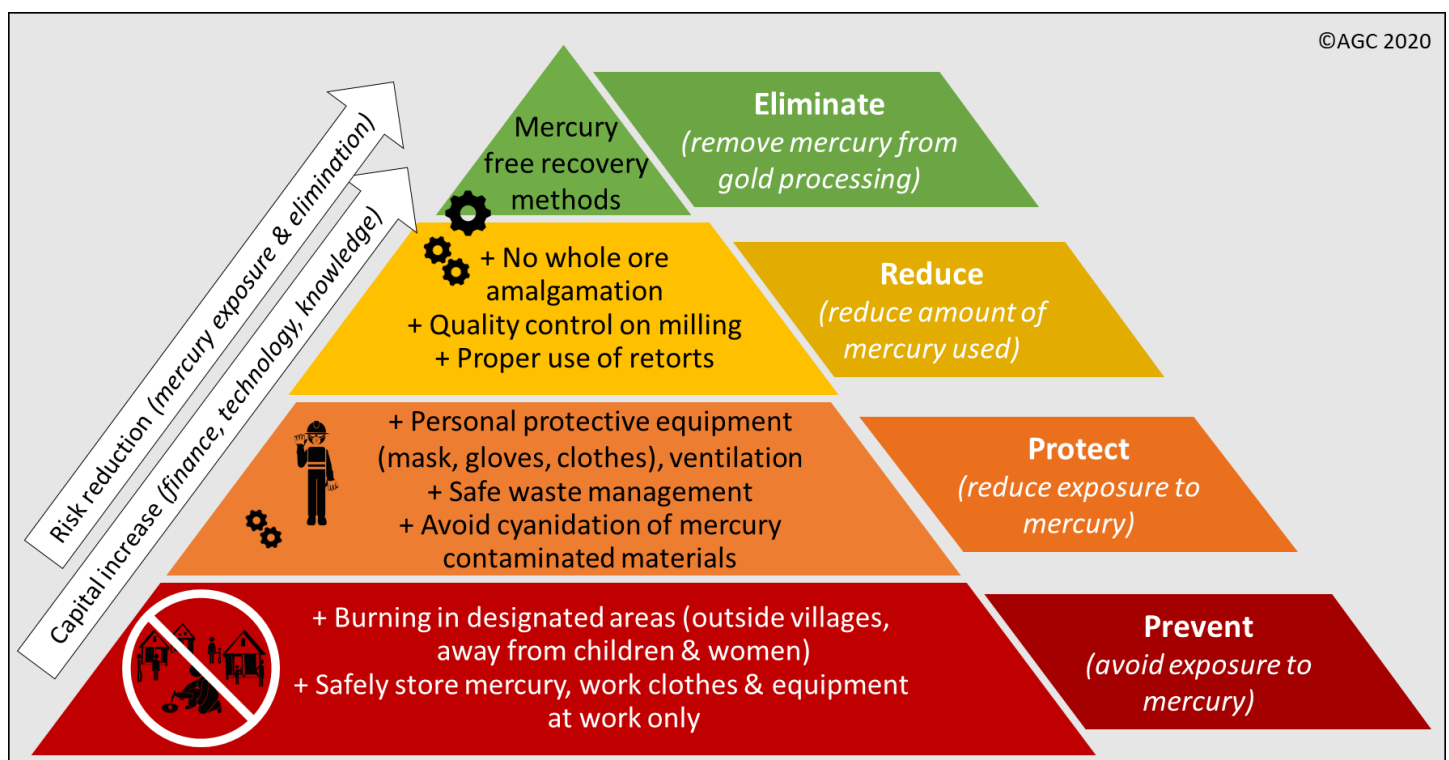


Figure 6: Risk pyramid of mercury use in artisanal gold mining

Cyanide

Cyanide is a rapidly acting, potentially deadly chemical that can exist in various forms: a colorless gas (hydrogen cyanide), a colorless liquid, or in form of crystals, powder or pellets (cyanide salts such as sodium cyanide). Cyanide is sometimes described as having a bitter almond smell, but it does not always give an odor, nor can everybody detect it. Cyanide is a naturally occurring chemical compound and is found in several plants (e.g. cassava roots, almonds) and in cigarette smoke.

Cyanide exposure in artisanal gold mining

Cyanide is used for gold extraction in many large-scale mining operations worldwide. In artisanal mining, cyanide leaching techniques have been used in Asia and Latin America for several decades and have more recently begun in Sub-Saharan Africa. With a gold recovery rate of around 60 to 90%, its performance is superior to mercury with a recovery rate of 20 to 50%, depending on the processing system. Sometimes, cyanide is used to capture residual gold by leaching ore and tailings to which mercury has previously been added – i.e. mercury contaminated materials. This is a worst practice under the Minamata Convention since cyanide increases the environmental mobility of mercury.

If cyanide is adequately handled, risks for human health and the environment can be minimized. However, lack of safety precautions and inadequate tailings management are common in artisanal gold mining and can cause negative impacts on miners, their communities and local aquatic systems. Human health hazards include the inhalation of hydrogen cyanide gas, especially when the pH of the cyanide solution is not maintained. This is most dangerous in enclosed, non-ventilated spaces. Miners may also encounter skin contact if they are handling cyanide without protective gear, for example when working with bare feet in cyanidation ponds (fig. 11, page 14). Furthermore, mining communities can be exposed to contaminated water and food. Children can accidentally play with containers containing cyanide pellets or crystals and poison themselves or can be exposed during the cyanidation process. While the lungs and the gastrointestinal system can absorb high amounts of cyanide, the absorption level through skin exposure is moderate (fig. 7).

If cyanide enters water bodies, fish and other aquatic biota are particularly sensitive to cyanide exposure, causing death even at low concentrations.

Effects of cyanide on health

Cyanide is highly toxic. A small dose of ingested cyanide salts may be deadly within a short time unless the person receives antidote therapy immediately after exposure. The inhalation of 120 to 150 mg/m³ can cause life-threatening injuries and may lead to death after 30 to 60 minutes; exposure to over 300 mg/m³ is immediately fatal.

Similar to mercury, individual factors such as age, nutritional status or pre-existing medical conditions impact cyanide absorption rate. For example, absorption is higher in people with impaired renal function (e.g., caused by chronic mercury intoxication).

Once absorbed by the lungs, the stomach or the skin, cyanide is quickly distributed through the body via the blood stream. It prevents the cells from absorbing oxygen, causing cell death. Since the heart and the brain use a lot of oxygen, the impacts on these organs are the most severe (fig. 7). The symptoms vary depending on the dose and frequency of exposure.

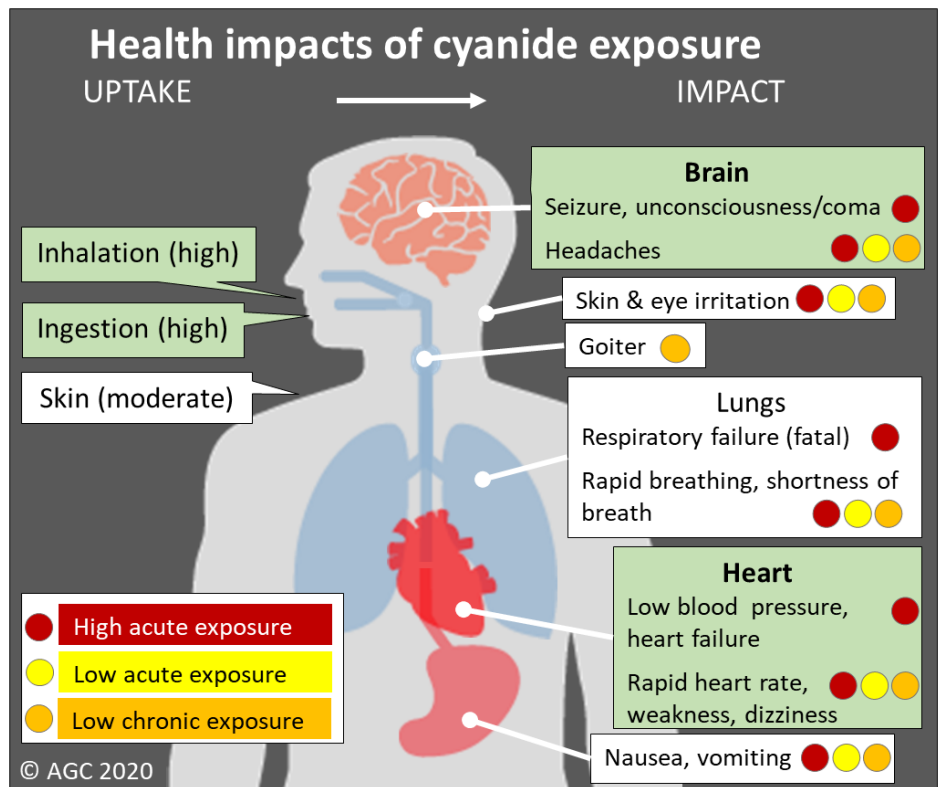


Figure 7: Major health impacts of cyanide

Low acute exposure: Symptoms of low, acute exposure include dizziness, headache, nausea and vomiting, rapid breathing, rapid heart rate, restlessness, weakness, hypoxia (decrease of oxygen supply to the brain) and skin and eye irritation. At low levels of exposure, most cyanide leaves the body within 24 hours after exposure without any long-term health implications.

High acute exposure: In cases of high acute exposure, the following symptoms can occur in addition to the low exposure effects described above: Convulsions (*seizures*), loss of consciousness, low blood pressure, lung injury to the point of respiratory failure or coma (both fatal). Death can occur within minutes and up to an hour after a lethal dose of cyanide gas and within minutes after ingesting a lethal dose of cyanide salt.

Low chronic exposure: The effects of regular cyanide exposure over a longer period are poorly understood. Yet, cyanide can damage the central nervous system (causing e.g., headaches, dizziness, numbness, tremors, loss of visual acuity), the cardiovascular system (e.g., rapid heart rate) and the respiratory system (e.g., breathing difficulties, chest pain). Furthermore, goiter (enlargement of the thyroid gland) has also been observed. In a study among artisanal gold miners in Burkina Faso, vomiting, abdominal pain, anxiety, changes in taste and bizarre behavior were reported in addition to chest pain and headaches.

Children exposed to cyanide show similar symptoms as adults. However, smaller amounts of cyanide are more harmful and can be fatal due to the lower body mass. Since cyanide can pass the placental barrier of pregnant mothers, it also impacts the developing fetus. Children have been born with thyroid disease due to prenatal exposure to cyanide.

Treatment of cyanide intoxication

Acute cyanide intoxication can be treated with antidotes and supportive medical care (esp. for respiratory and cardiovascular symptoms). Antidotes are more effective if given immediately after exposure, therefore seeking immediate medical care is important. If cyanide is ingested, activated carbon can help to adsorb it in the gut and prevent the uptake in the blood stream. The carbon should be used within the first hour after exposure; a single dose of 50g of activated charcoal in adults and 1 g/kg, up to a maximum of 50g, in children. Since adequate medical care is often not available in remote artisanal mining sites, risk mitigation becomes even more important. All operations using cyanide should have an approved cyanide antidote kit on hand and a trained person to administer the kit (in most cases intravenously) to the patient.

Cyanide in the environment

Cyanide is a serious risk to the environment when it contaminates soil and water, particularly to aquatic life. For example, a free cyanide concentration of 20 mcg/L (about one small drop of cyanide per liter) is fatal to fish. Therefore, safe handling of cyanide and adequate tailings management is crucial!

Cyanide does not accumulate in plant life or animals over time like mercury, and it can be safely used and destroyed if handled professionally. Unlike mercury, cyanide is a degradable chemical compound which can be destroyed, either through cyanide removal (*AVR process: acidification–volatilization–reneutralization*) or through natural oxidation to less toxic cyanide species. The natural degradation process reduces the toxicity of cyanide species over time through different mechanisms (fig. 8), mainly volatilization from surface water to hydrogen cyanide gas (concentrations in air are usually low and not harmful to humans), atmospheric oxidation, absorption by other minerals, photo-degradation (destruction through sunlight) and biodegradation (biological oxidation) through microorganisms such as bacteria and plants. Cyanide ultimately degrades into nontoxic carbon dioxide and nitrates.

In soil, cyanide can evaporate, be transformed by microorganisms or it can seep into ground water where it is less likely to be degraded due to lack of sunlight and oxygen. It has been detected in groundwaters in contaminated

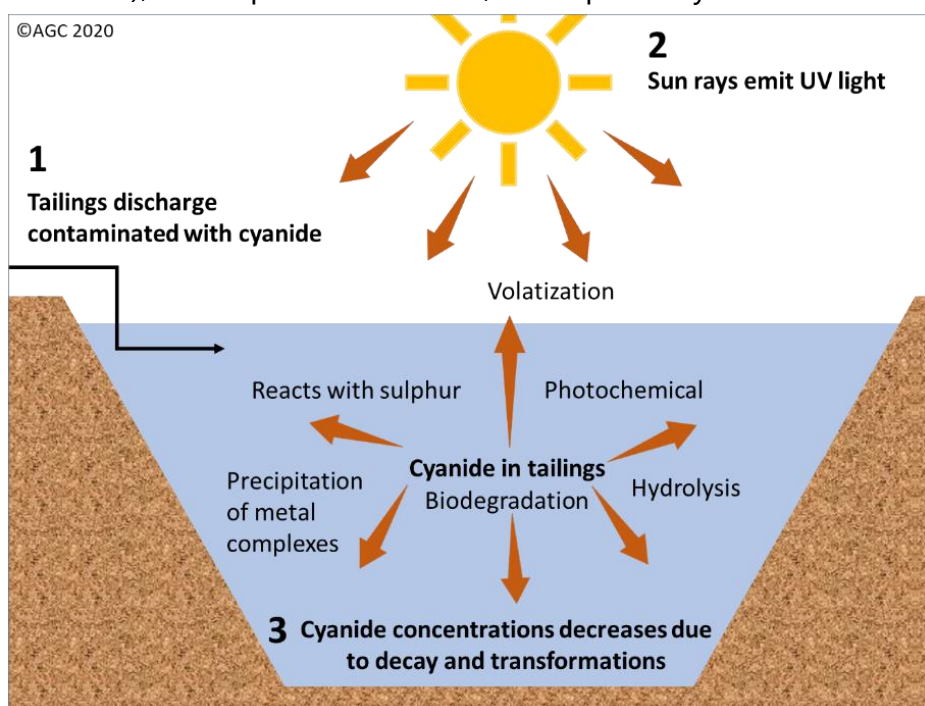


Figure 8: Natural degradation processes of cyanide in tailings ponds

settings such as landfill leachates where it can sterilize microorganisms within the soil, consequently preventing its degradation, causing the contaminated plume of groundwater to spread further.

The natural degradation rate for different cyanide species differs, depending on the factors influencing the degradation processes, e.g., temperature, pH, intensity of light, surface area to depth ratio of water bodies, solution agitation, and type of soil. Accordingly, the natural detoxification process tends to be faster in tropical and subtropical climates where temperatures and light intensity are higher. However, natural destruction rates are difficult to predict, and proper use of cyanide requires effluent testing to ensure discharged waters are not toxic to fish. There is currently very little formal data on the extent and environmental impact of cyanide use in artisanal gold mining, but field observations show that its use is widespread and growing, and its environmental management remains very poor to non-existent.

An additional management and environmental problem in artisanal gold mining is the use of cyanide to leach mercury-contaminated tailings to capture residual gold. This is highly problematic because cyanide dissolves not only gold but also mercury. This increases environmental mobility of mercury, causing it to be more readily emitted to the atmosphere and increasing its bioavailability. Therefore, this practice is considered a worst practice under the Minamata Convention. Unlike for mercury, there is currently no international regulatory framework for cyanide use in artisanal mining. The International Cyanide Management Code (ICMC) is a voluntary initiative of large-scale gold and silver mining industries to standardize safe practices.

Preventive measures at artisanal mining sites

Health and environmental risks associated with cyanide use can be controlled through various preventive measures:

Sodium cyanide decomposes on contact with acids, water and carbon dioxide, producing highly toxic, flammable hydrogen cyanide gas; therefore, it should be stored in a dry, secure and well-ventilated area in a closed container with a label signaling dangerous contents.

Workers should be alerted when cyanide is present and in use; smoking, eating and drinking should not be allowed during this time. When handling cyanide, workers should wear personal protective equipment (gloves, rubber boots, goggles, respirator); for example, wearing gloves while preparing leach solutions to avoid skin contact.

pH is key! - One important safety aspect during ore processing with cyanide is the elevation of the pH level through the addition of a base (calcium oxide (CaO) or lime). If the pH is not elevated to above 10, toxic hydrogen cyanide gas forms and volatilizes. Mixing of solutions should also be conducted in a ventilated area, in case there is a pH shock and hydrogen cyanide is off gassed.

Tailings containing cyanide should be managed to enhance the natural destruction of cyanide and reduce environmental contamination. All effluents should be regularly tested; a fish test where live fish is placed into the effluent outlet is a simple and an effective testing approach.

Silicosis

Silicosis is one of the most prevalent occupational diseases in the world, affecting millions of workers in dusty industries such as mining, agriculture and construction. Silicosis is the most common form of pneumoconiosis, the general term for dust borne interstitial lung diseases (examples of other forms of dust are coal, cotton or asbestos). Crystalline silica minerals such as quartz and chalcedony are basic components of soils, sands, and rocks like granites. Silica minerals form small particles within dust.

Silica exposure in artisanal gold mining

The prolonged inhalation of dust during drilling, mineral extraction, ore crushing and milling in the mining sector causes fine silica to be deposited in lungs. These deposits cause irritation of the lungs (*pneumonitis*) and development of scar tissues (*lesions, nodules and fibroids*), which in turn cause obstruction of the airways (fig. 9). Silica particles can penetrate different parts of the lungs depending on their size; smaller particles are therefore most dangerous because they can travel deeper into the respiratory system and cause greater damage than larger particles.

The exposure to silica usually goes unnoticed because its inhalation does not cause any immediate health effects such as lung irritation. Therefore, silicosis – one of the oldest occupational diseases – is still considered a hidden epidemic in many countries.

There are no international guidelines on occupational exposure limits to silica dust and national occupational exposure thresholds vary between 0.025 and 3 mg/m³. The US National Institute for Occupational Safety and Health (NIOSH) recommends an exposure limit (REL) of 0.05 mg/m³ as a time-weighted average (TWA) for up to 10 hours/day during a 40-hour workweek. Some studies have shown that this level does not provide sufficient protection and that only zero exposure can be considered safe. According to OSHA (2016), the exposure to 0.05 mg/m³ over a period of 45 years causes silicosis in 5 out of 100 people, the exposure to twice as much silica dust (to 0.1 mg/m³) increases the risk to 30 in 100 people. Data on silica exposure in the artisanal mining sector are very limited but field observations suggests it is extensive, especially in hard rock mining. A study in Tanzania found average concentrations of 0.19 mg/m³ in aboveground operations, exceeding the REL by four times. There are no reported data for below ground concentrations. Wet alluvial processing usually poses a much lower exposure to silica dust compared to dry hard rock processing and dry alluvial processing.

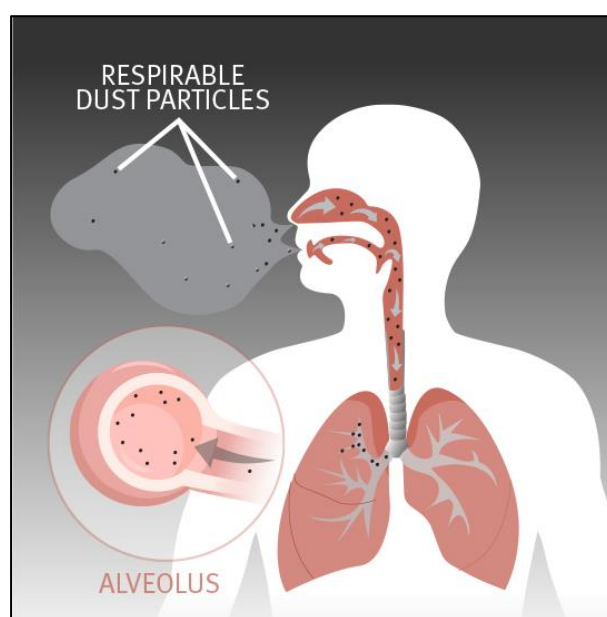


Figure 9: Inflammation and lung obstruction caused by silica dust (© The State of Queensland)

Types of silicosis and symptoms

Miners can develop three different forms of silicosis, depending on the level of silica concentration they are exposed to and the duration:

- (1) **Chronic silicosis** (most common form) usually occurs after long-term exposure (over 10 to 20 years) to low concentrations of silica dust; the disease shows a slow progression.
- (2) **Accelerated silicosis** occurs after exposure to larger amounts of silica over a shorter period of time (5-15 years). The onset of symptoms is faster and evident between 5 to 10 years after exposure.
- (3) **Acute silicosis** (rare, unusual reaction) occurs after short-term and very intense exposure to high amounts of silica that causes severe lung inflammation; it appears few weeks to months after very intense exposure.

Common respiratory symptoms of silicosis are shortness of breath, cough and phlegm production. An acute form of silicosis can also be accompanied by fever and chest pain. The severity of symptoms depends on the dose and duration of exposure and individual factors such as smoking habits and history of lung diseases that have weakened the lungs. This increases a person's susceptibility to silicosis. Genetic factors may also affect the clearance and defense mechanisms of the lungs.

Silicosis is irreversible and progressive (worsening with time), incurable and potentially fatal; it can substantially shorten the life span of an affected miner. Since the onset of symptoms is usually slow (several years), silicosis remains often undetected and undiagnosed. Furthermore, silicosis weakens the respiratory system and increases the risk of comorbidities.

Tuberculosis, a communicable, opportunistic disease called silicotuberculosis in silicosis patients, is a common comorbidity. Other health problems include lung cancer, chronic bronchitis (arising from irritation of tubes that carry air to the lungs) and chronic obstructive lung disease (COPD). It is also suspected to increase the risk of autoimmune diseases (body's natural defense system fails to differentiate its own cells from foreign cells, causing the body to mistakenly attack normal cells), e.g. lupus erythematosus and rheumatoid arthritis.

Medical interventions

Silicosis cannot be treated or cured. It is only possible to ease symptoms for example through supplemental oxygen or steroids to relax tubes, and cough medicine. If a person is diagnosed with silicosis, the person should avoid any further exposure to silica dust and cigarette smoke. From a public health perspective, the primary goal is therefore to detect workers exposed to silica as early as possible through routine screening (e.g. regular occupational health check-ups with lung functioning tests). Since artisanal mining sites often operate informally and/or without adequate occupational and health protocols (e.g. no regular health check-ups at work site), medical practitioners should ask patients working in the mining sector about any respiratory problems and – if possible – test the lung functionality.

In order to prevent silicosis at the mining site, the two most important interventions are the use of adequate respiratory masks and the use of water and wet processing techniques to suppress dust.

Mercury

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Figure 10: A miner preparing the mercury gold amalgam for vaporization (photo: AGC)

Cyanide

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Figure 11: A miner stands on a batch of ore in a heap leaching pit, which uses cyanide as leaching agent (Photo: AGC)

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Figure 12: Dust exposure in artisanal hard rock mining is a common threat (Photo: AGC)

Our Activities



Capacity development

- Gender-sensitive training for miners and communities on occupational and safety hazards in artisanal mining and community health problems
- Capacity building for health care professionals to detect, diagnose and treat ASGM related health hazards including mercury intoxication, and to provide information on risk prevention



Research & monitoring

- Research on health hazards and their relation to livelihoods in artisanal mining communities
- Institutional capacity assessments of the health care sector
- Developing strategies for creating national health data infrastructure to monitor the disease burden in ASGM communities and evaluate health programs



Public health policies

- Policy consultation for governments to develop Public Health Strategies for the ASGM sector as part of the National Action Plan to meet the goals of the Minamata Convention on Mercury



Health as cross-cutting issue

- AGC considers health as cross-cutting issue in all other interventions such as process engineering, geology & geochemistry, environmental management, and access to finance and markets.

About the AGC

The Artisanal Gold Council (AGC) is a not-for-profit organization dedicated to improving the economic opportunities and livelihoods of the millions of people involved in the artisanal and small-scale gold mining sector (ASGM) in 80+ countries.

Our integrated approach seeks to build an environmentally sound, socially responsible, and formalized ASGM sector effective at transferring wealth from rich to poor.



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