ARTISANAL GOLD COUNCIL

Chemical hazards in the artisanal gold mining sector

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

Version 2.1, April 2022

Occupational health risks in the artisanal gold mining 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. Millions of people in over 70 low- and middle-income countries depend on artisanal gold mining for their livelihoods, producing around 20% of the global gold production. Depending on the degree of organization and formalization, mining sites tend to have poor occupational safety standards and practices exposing miners to various occupational health hazards.

Chemical risks can include the exposure to toxic substances such as mercury and cyanide which are used to extract gold from the ore, and silica dust as well as other naturally occurring heavy metals or toxic gases during ore extraction and grinding. Biomechanical hazards are caused by physically straining manual work (e.g., painful disorders of muscles and the skeleton from heavy lifting or repetitive movements) and physical trauma (e.g., from slipping, falling, landslides) (WHO, 2016).

Poor hygienic conditions at the mining site and inadequate living conditions in artisanal mining communities can increase the susceptibility to communicable diseases such as malaria, tuberculosis or gastrointestinal diseases, as well as psychosocial hazards (e.g., substance abuse).

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About this brochure

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

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. Exposure to very fine silica dust is a common hazard in hard rock mining, but one that can easily be prevented.

About the AGC

Based in Victoria, Canada, the Artisanal Gold Council (AGC) is a not-for-profit organization dedicated to improving the working conditions, opportunities, environment and health of the millions of people involved in ASGM in the developing world.

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

The AGC provides training on Occupational Health and Safety for artisanal miners, government officials, health professionals and other interested stakeholders along the artisanal gold supply chain.

Acknowledgment

This brochure has been prepared by Mareike Kroll, PhD, Director of Health and Research at the AGC. Version 2.0 has been updated in March 2022.

Mercury

Elemental mercury, a silver liquid (Fig. 1), is a highly toxic heavy metal that cannot be degraded and persists in the environment. The Minamata Convention on Mercury, a global treaty, therefore aims to protect human health and the environment from the adverse effects of mercury (UNE, 2019a).

The artisanal gold mining sector is currently the largest anthropogenic source for mercury releases as it is a deficient but simple and cheap method to extract gold from the ore. It is therefore a priority focus under the Minamata Convention.

Exposure in artisanal gold mining

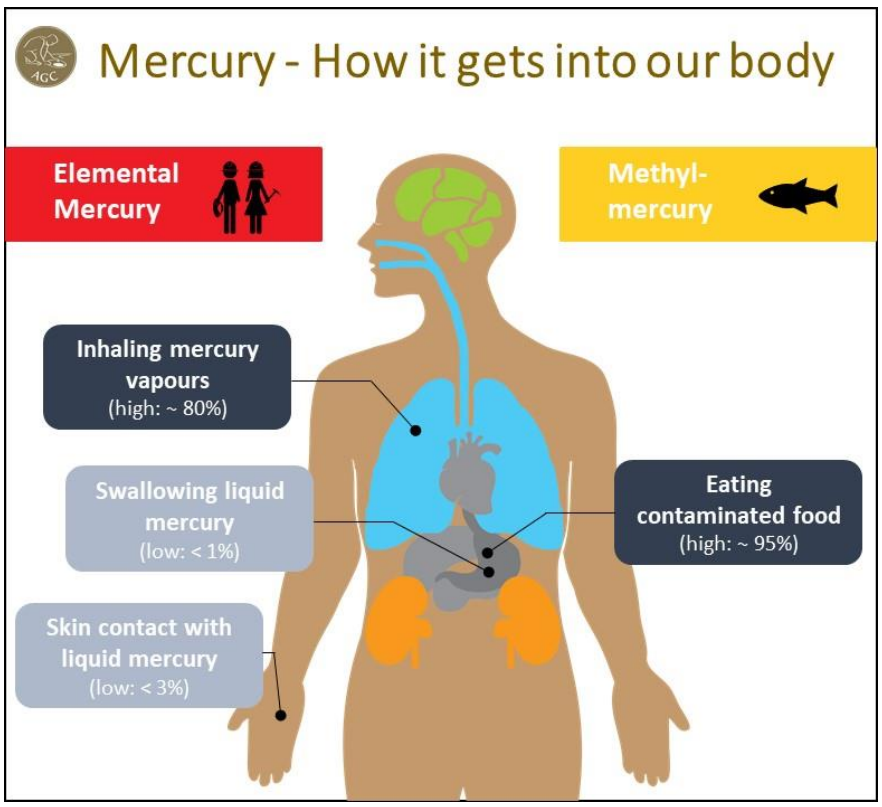
Miners in artisanal gold mining are exposed to elemental mercury during amalgamation (mixing ore with mercury), squeeze (separating solid amalgam from excess mercury), vaporization (heating of amalgam), smelting (melting sponge gold with residual mercury), and refining of raw gold doré.

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 (ATSDR, 2014) (Fig. 2). Furthermore, mercury vapour can adsorb to clothes, skin, furniture, work tools and walls. These materials can continue to emit mercury vapours for extended periods of time. Hence, the whole household gets exposed to mercury vapours when the gold mercury amalgam is burnt within the home or if work clothes and processing equipment are stored at home.

If liquid elemental mercury is accidentally ingested, only a very small amount gets absorbed by the body (less than 0.1%). Absorption is also low during unprotected skin contact (less than 3%), but the exposure can cause skin and eye irritation (NRC, 2000).

Figure 1 (below). Elemental mercury (photo: AGC)

Figure 2 (right). Elemental mercury intake in artisanal gold mining



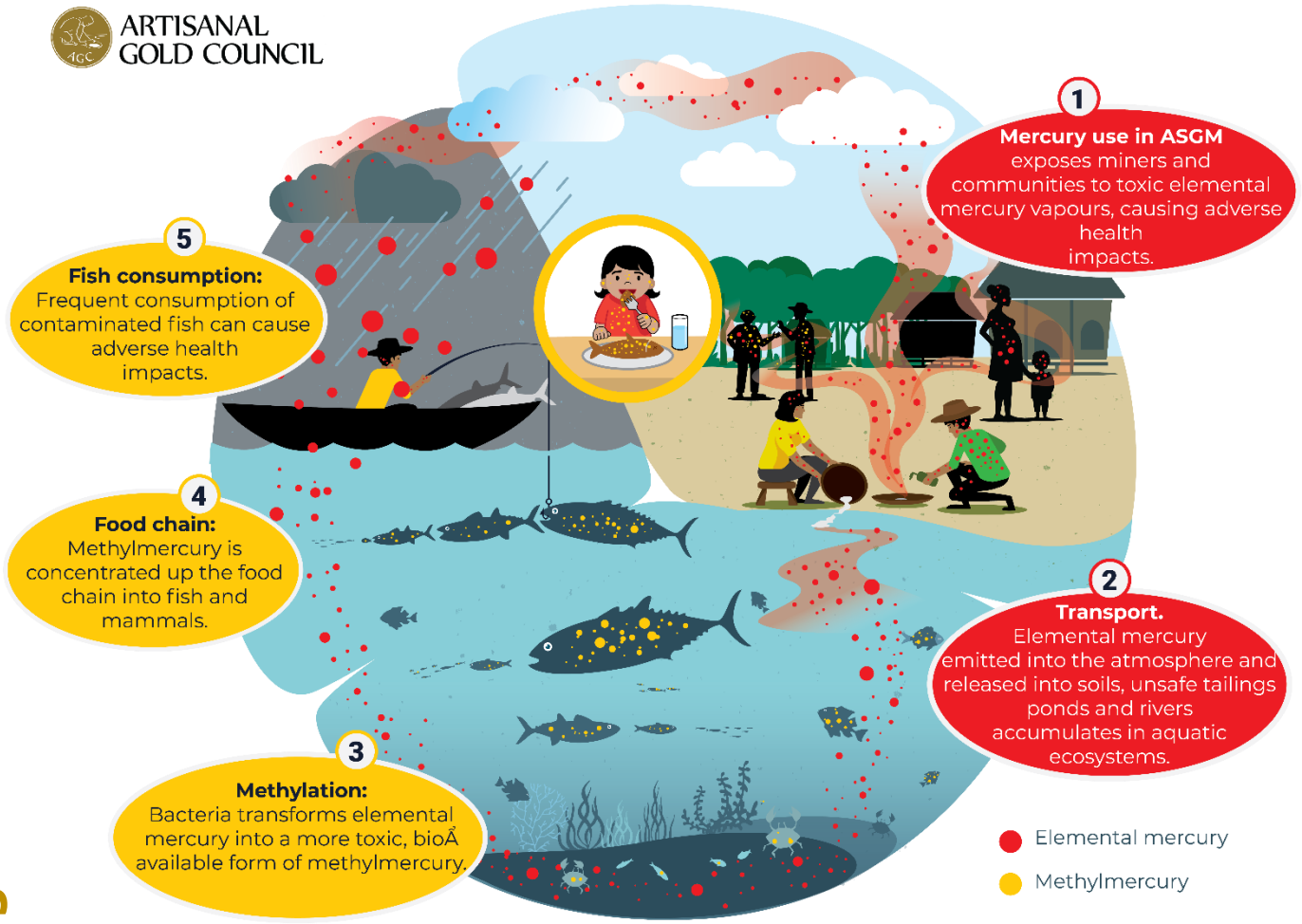
Health data for artisanal gold mining communities are scarce. According to a global study, around one third of active miners suffer from moderate or severe chronic elemental mercury vapour intoxication (Steckling et al., 2017). Though the prevalence differs depending on the local ASGM practices, the figures show that mercury intoxication is a common problem in the sector where mercury is being used and requires immediate action. In addition, the environmental and health impacts of mercury use in the sector go beyond the local mining community.

Figure 3. Elemental mercury emitted to and released in artisanal mining does not only impact the health of miners as mercury persists and travels in the environment.

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 (UNE, 2019b). Elemental mercury is not only emitted to the atmosphere during ore processing or from the tailings, it is often also directly released into water streams and soils (e.g., from poorly made tailings ponds) (Fig. 3, step 1).

Most of the mercury released into the environment will eventually enter aquatic systems (streams, lakes, rivers and ocean) 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 in water bodies and soils depends on their physical, chemical, and biological characteristics, such as presence of specific bacteria, temperature or water flow. Therefore, it is difficult to 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) (Fig. 3, step 4). Frequent consumption of contaminated fish can have adverse impacts on our health (Fig. 3, step 5).

Health effects of mercury exposure

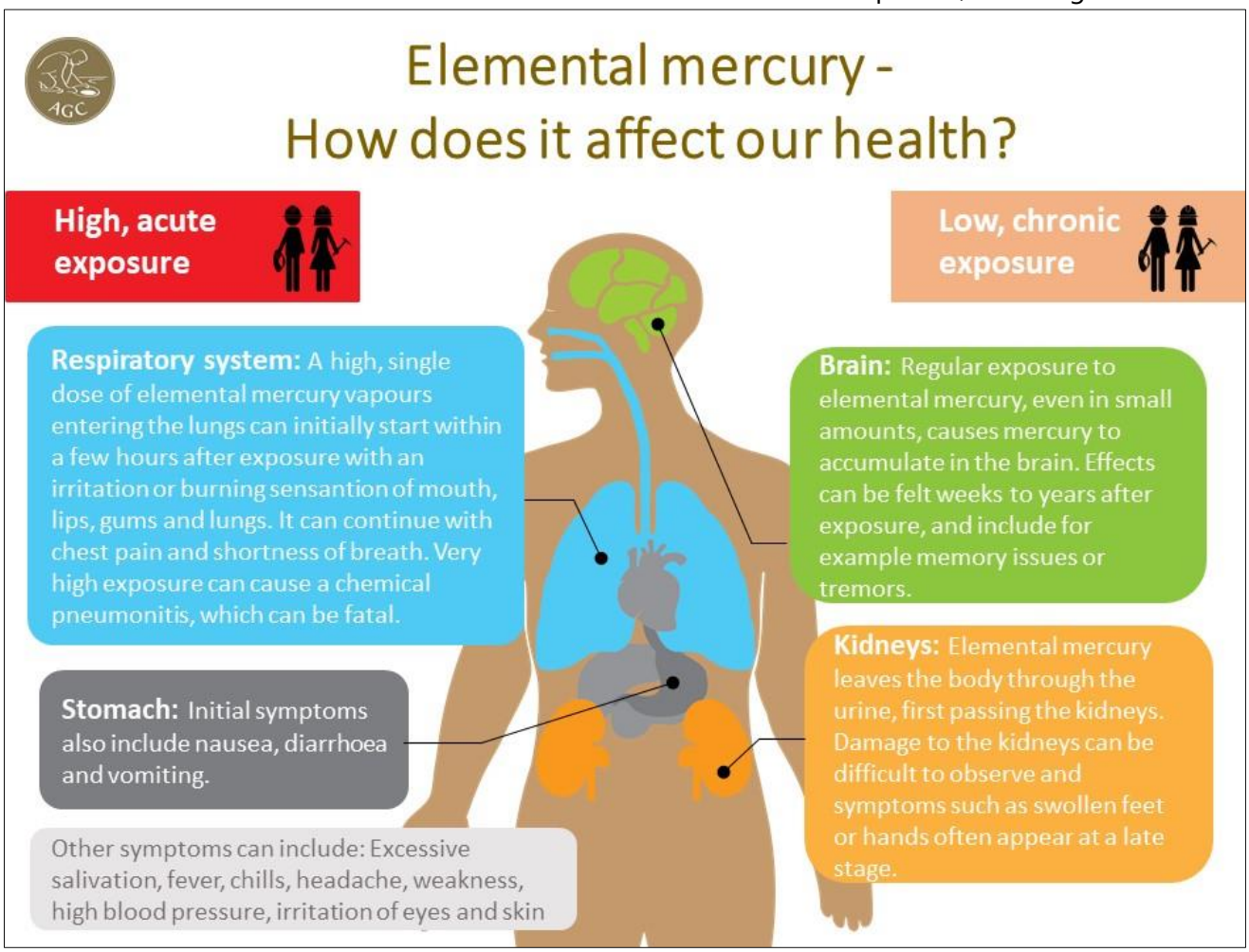
Mercury intoxication can appear in three forms: as (1) acute or (2) chronic elemental mercury vapour intoxication, esp. in artisanal gold mining but also other occupational settings (ILO, 2022), or (3) as chronic methylmercury intoxication caused by the consumption of contaminated food.

(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 (>1000 mcg/m^3) during the burning of amalgam (one-time exposure), causing damage primarily to the lungs and the digestive system.

Most symptoms usually develop within a few hours after exposure, starting often with

Figure 4. Health impacts of acute and chronic exposure to elemental mercury



gastrointestinal symptoms (nausea, vomiting) (Fig. 4). 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. 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 chronic 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 many solutes in the blood from entering the central nervous system. If mercury reaches the brain, it can cause symptoms of chronic intoxication (e.g. neurological effects) several days after the initial exposure.

(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. Chronic intoxication mainly affects the brain and the kidneys (Fig. 4). 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 (Fig. 6).

Damage to kidneys such as proteinuria is more difficult to observe; in later stages, 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) (Fig. 5).

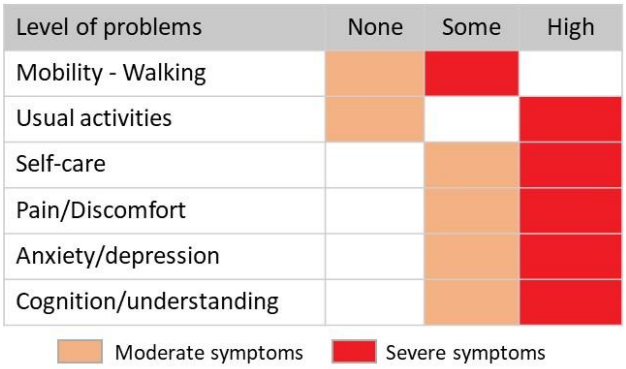


Figure 5: Impact of a moderate and severe chronic mercury intoxication on daily activities (adapted from: Steckling et al., 2015)

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 fully 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 damage increases with the length and magnitude of elemental mercury exposure. Therefore, artisanal miners who are using elemental mercury over many years during ore processing

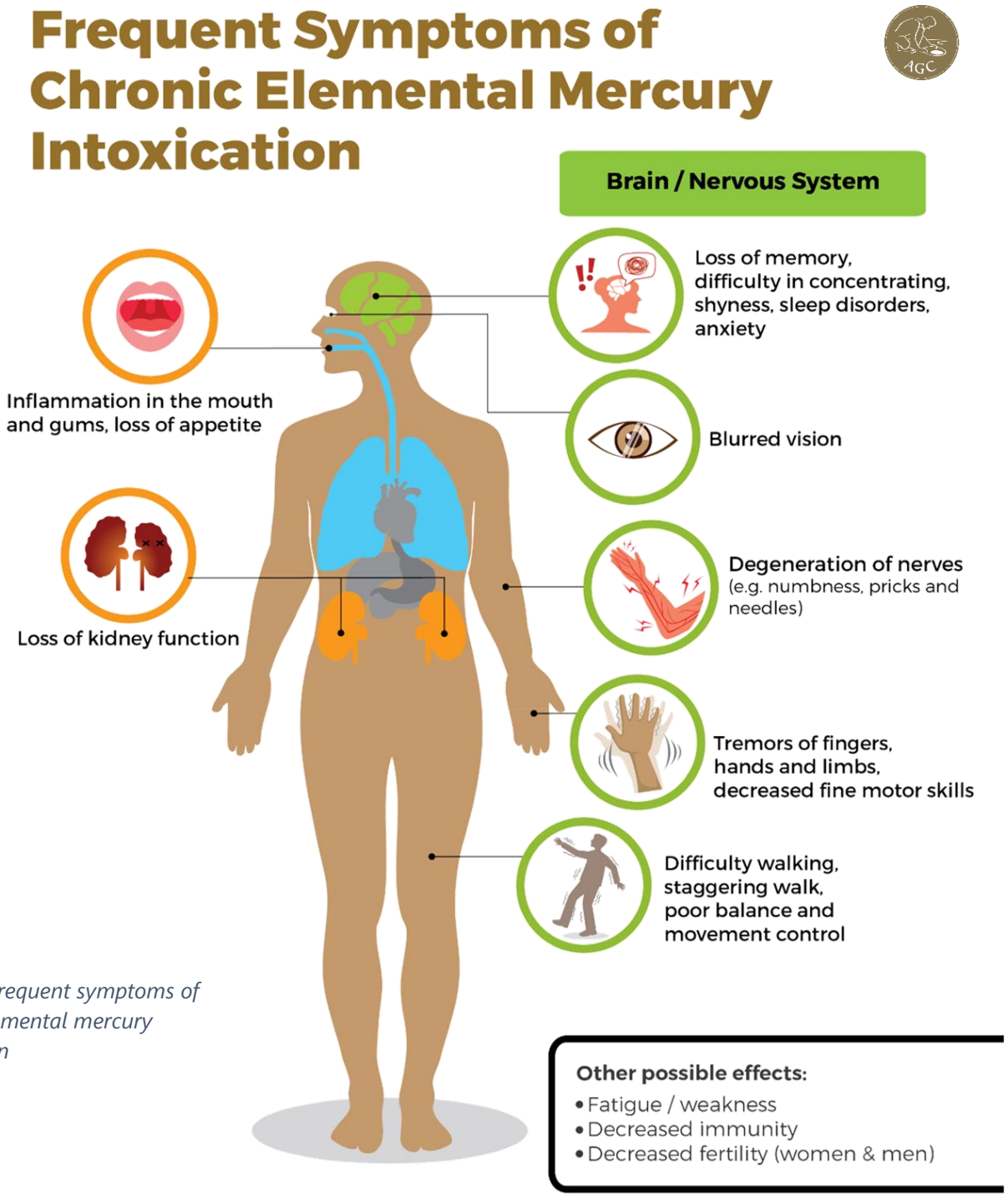


Figure 6. Frequent symptoms of chronic elemental mercury intoxication

are especially susceptible. But even a contaminated retort stored at home can put family members, especially children, at risk.

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

Exposure through fish consumption

When a person consumes contaminated fish or shellfish (> 0.5 mcg/g) on a regular basis over an extended period, methylmercury accumulates in the body. 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 frequent alcohol consumption can increase the

uptake, where some foods (e.g., omega-3 fatty acids, vitamin E, garlic) seem to attenuate the impact.

It is unclear whether exposure to low doses of methylmercury is toxic in adults. Though, the negative impact on developing fetuses through maternal exposure has been proven. Several countries and International Organizations have issued guidelines on fish consumption to advise the general population, and especially pregnant women, about safe fish-eating practices.

Swordfish, for example, has one of the highest average concentrations of methylmercury (average 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 for all populations (WHO/UNEP, 2008), 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 for specific geographic regions on which fish to eat and how often is crucial.

Health implications

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. These include, for example, tremors, lack of muscle coordination, behavioral, emotional and memory effects (Fig. 7). Methylmercury also affects the cardiovascular system and can cause high blood pressure, heart attack or coronary artery diseases in persons with high intake of methylmercury. Other symptoms include headache, fatigue and reduced fertility in men and women.

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 level of exposure and can range from development delays, impaired movement and coordination problems (*cerebral palsy*), convulsions (*seizures*), hearing and vision impairments, heart malformations, to the point of distortion of limbs, severe retardation and death.

Figure 7. A child gets exposed to elemental mercury vapours during vaporization (photo: AGC)

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 damages caused by mercury during prenatal exposure is permanent.

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 is leading to a higher body burden compared to adults. If present during amalgamation (Fig. 7), 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 very important for the child's development and mothers should not be discouraged from breastfeeding.

The risk of permanent organ damage increases the earlier infants and children are exposed to mercury (with the highest risk in utero). 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 very limited. In chronic cases, 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 (acute case) or physiotherapy for muscle control problems (chronic case). 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 (Beauchamp et al., 2019). Chelating agents bind 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 the work sites can protect miners and surrounding communities from mercury exposure or at least reduce the risks for human health and environmental contamination (Fig. 8). Artisanal miners do not always have access to different forms of capital that are required in order to reduce and eliminate the risk, for example knowledge on mercury toxicity and OHS safety standards, technical know-how, or financial means to buy personal protective equipment or a retort.

While the elimination of mercury through mercury-free processing is the desired outcome, miners are often confronted by various barriers to achieve this goal, from formalization to access to finance and technology.

Therefore, the risk pyramid also shows measures that can be implemented by miners without or with limited access to technical equipment and know-how.

The simplest level of intervention can prevent the exposure of vulnerable groups (women at

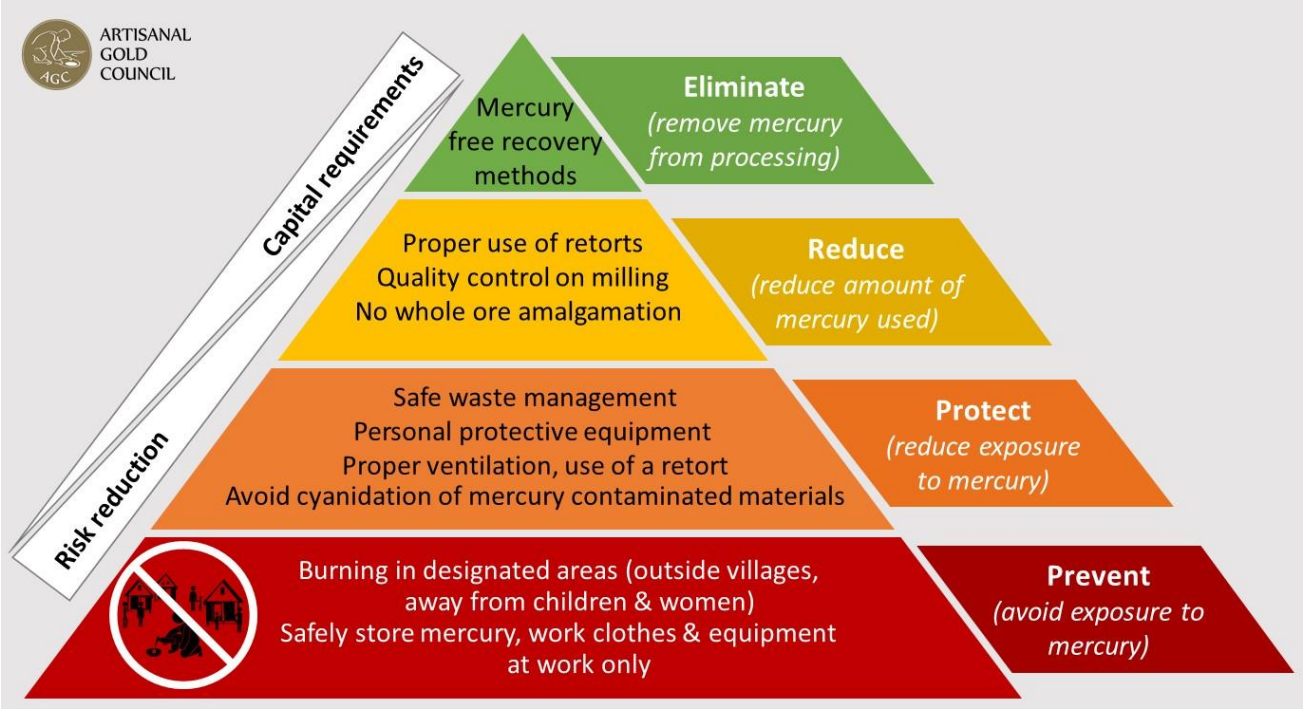
childbearing ages, infants and children) to mercury by burning the mercury-gold amalgam outside settlements, away from women and children, and by storing mercury, work cloths and equipment securely away from home.

The second level of interventions protect workers by reducing their exposure to mercury, for example by using personal protective equipment (e.g., respirator with charcoal filter), by using a retort or by burning the amalgam in a properly ventilated place. Avoiding the cyanidation of mercury contaminated tailings, a worst practice identified under the Minamata Convention (see Cyanide), prevents the increased mobility and bioavailability of mercury compounds in the environment. Safe waste management of contaminated tailings can also reduce environmental contamination.

The third level of intervention aims at reducing the amount of mercury being used during processing, for example by using a retort and reusing the captured mercury, or by adding the mercury only to the concentrate, after grinding and quality control of milling. The avoidance of whole ore amalgamation can reduce the amount of mercury being used considerably.

The transition towards mercury-free processing is a challenging but necessary task for the artisanal and small-scale gold mining sector, which will require support from various stakeholders along the gold supply chain and the international community.

Figure 8. Risk pyramid of mercury use in artisanal gold mining



Cyanide

Cyanide is a toxic chemical that can have a dangerous impact on human health and the environment. It exists in various forms, mainly as 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, apricot pits) and in cigarette smoke.

Compared to mercury, cyanide is rapidly acting and potentially fatal within minutes, but it is not a persistent pollutant and can be destroyed in the environment.

Cyanide use and 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%, cyanide is superior to mercury in recovering gold from the ore, depending on the processing system.

However, cyanide is used in artisanal mining often after the use of mercury in order to capture residual gold by leaching mercury contaminated tailings. This is a worst practice under the Minamata Convention since it generates mercury-cyanide complexes that are highly mobile in the environment, causing it to be more readily emitted to the atmosphere and increasing its bioavailability (i.e., methylation).

If cyanide is adequately handled, risks on human health and the environment can be minimized. 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 (Stapper et al., 2021). Lack of safety precautions are common and can cause serious injuries and death. Poor environmental management can lead to the contamination of soils and water bodies; fish and other aquatic biota are particularly sensitive to cyanide exposure, causing death already at low concentrations.

Miners can be exposed to hydrogen cyanide gas during processing, 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 bare feet in cyanidation ponds (Fig. 11).

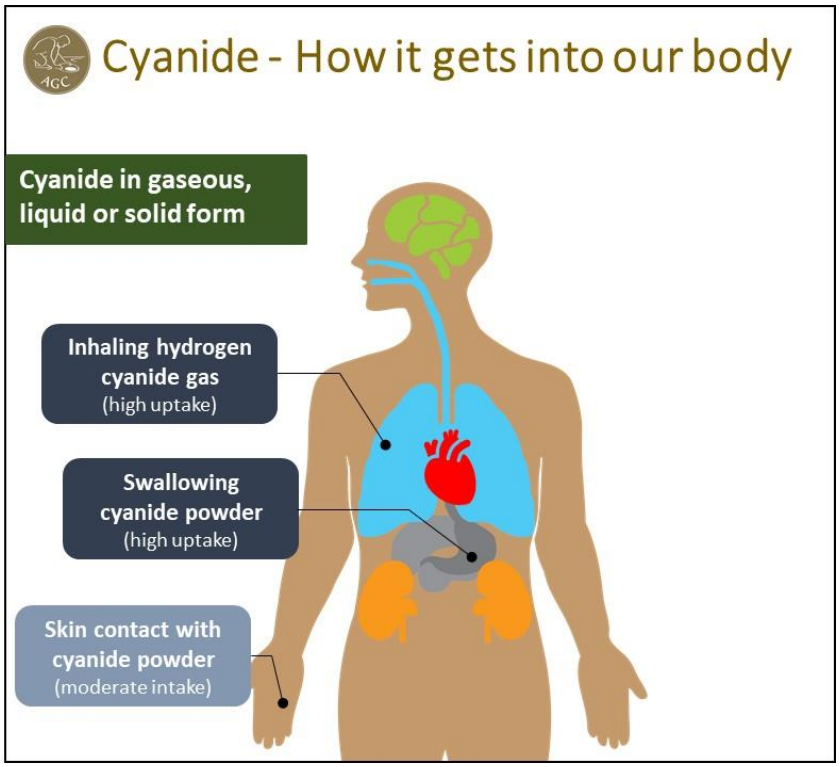


Figure 9. Exposure routes to cyanide

Furthermore, improper storage, handling and disposal can contaminate food and drinking water, or children can accidentally play with containers containing cyanide pellets or crystals and poison themselves.

Effects of cyanide on human health

Cyanide is highly toxic. Our body absorbs high amounts of mercury if we inhale cyanide gas or swallow cyanide in its solid form, or contaminated food or water. 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 120-150 mg/m^3 can cause life-threatening injuries and may lead to death after 30 to 60 minutes, exposure to over 300 mg/m^3 is immediately fatal (WHO, 2004). Cyanide can also be absorbed through the skin, though the uptake is considered as moderate, with slower onset of symptoms (Fig. 9).

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., through 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. 10). The symptoms vary depending on the dose and frequency of exposure but are mostly similar for oral and respiratory intake (ATSDR, 2006a/b; WHO, 2004).

Low acute exposure

One of the first symptoms of one-time exposure to a low dose of cyanide, which can appear within few minutes, is rapid breathing and shortness of breath (*dyspnea*), caused by lack of oxygen (*hypoxia*), which may be followed by lung irritation, chest pain and cough. Effects on the central nervous system (brain) cause the following symptoms within minutes: dizziness, headache, restlessness and weakness. Lack of oxygen can also cause a rapid heart rate. In addition, skin and eye irritation has been reported. Nausea and vomiting can also occur, particularly if cyanide has been ingested.

At low levels of one-time exposure, most cyanide leaves the body within 24 hours after exposure without any long-term health implications.

Low chronic exposure

The effects of regular exposure to low doses of cyanide over a longer period are poorly understood. Symptoms reported from one-time exposure might become chronic, and damage to the central nervous system (brain) may also cause tremors, numbness, muscular weakness

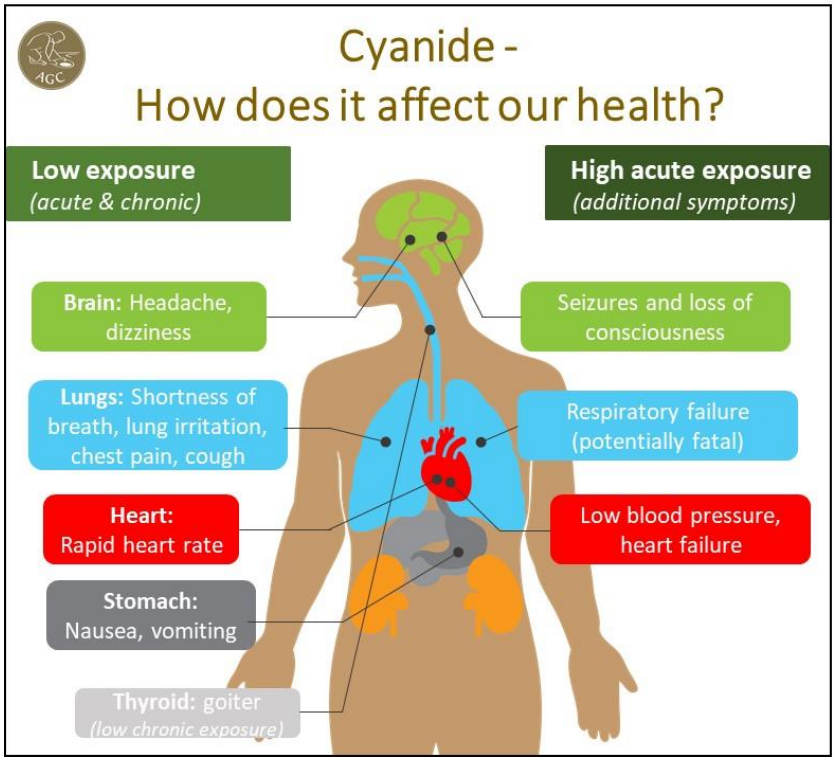


Figure 10. Major health impacts of low and high cyanide exposure

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

and poor vision. 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 (Knoblauch et al., 2020).

High acute exposure

Death can occur within few minutes up to an hour after a lethal dose of cyanide gas and within minutes after ingesting a lethal dose of cyanide salt. In cases of high acute exposure, convulsions (*seizures*) and lack of consciousness follow shortness of breath. Severe lung injury can cause fatal respiratory failure. Low blood pressure (*hypotension*) can also cause fatal cardiovascular failure. In survivors, sequalae can experience long-term damage to the central nervous system and cardiovascular system.

Exposure in children

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. For example, children who ate accidentally certain fruit pits, such as apricot kernels, have experienced symptoms and even death. Since cyanide can pass the placental barrier of pregnant mothers, it also impacts the developing fetus. Children, who experienced prenatal exposure to cyanide, have been born with thyroid disease (ATSDR, 2016b).

Treatment of cyanide intoxication

Acute cyanide intoxication can be treated with antidotes and supportive medical care (e.g., supplementary oxygen and ventilation to treat respiratory distress). Antidotes such as Hydroxocobalmin (Cyanokit) are more effective if given immediately after exposure; therefore, seeking immediate medical care can save lives!

If cyanide is ingested and medical care is not immediately available, 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 (Zellner et al., 2019).

Since adequate medical care is often not available in remote artisanal mining sites and immediate medical response is necessary in the case of a severe, acute intoxication, risk mitigation through adequate safety measures becomes even more important. All operations using cyanide should have a cyanide emergency response plan and a trained person who can provide first aid and administer an approved cyanide antidote kit (in most cases intravenously) to the patient.

Cyanide in the environment

Cyanide is a serious risk to the environment due to its high toxicity particularly to aquatic life. For example, a free cyanide concentration as low as 20 to 50 mcg/L (about one small drop of cyanide per liter) can be fatal to fish. Therefore, safe storage of cyanide, handling during processing and adequate tailings management are 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.

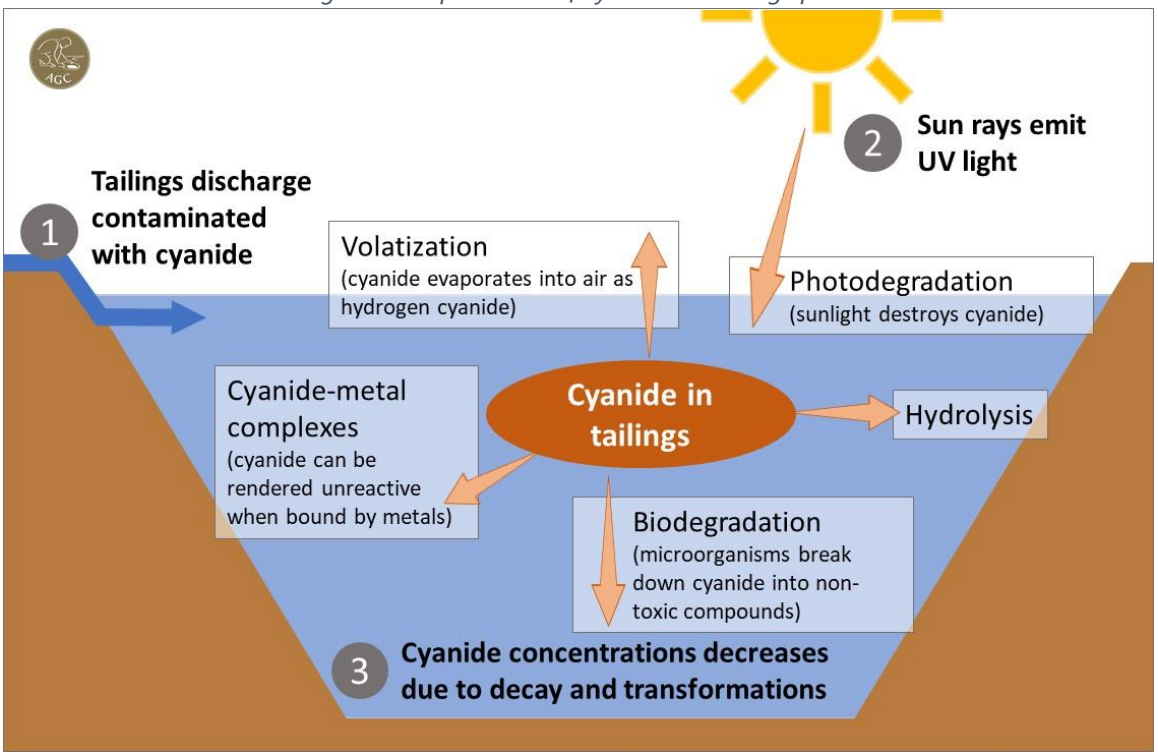
The natural degradation process reduces the toxicity of cyanide species over time through various mechanisms (Fig. 12): In surface water, UV light from sun rays can destroy cyanide (photodegradation) or it evaporates into air as hydrogen cyanide (volatization; concentrations in air are usually low and not harmful to humans). In addition, cyanide can be rendered unreactive or broken down to less harmful substances when binding to other metals (building a cyanide-metal complex) or when being absorbed by minerals. In addition, microorganisms such as bacteria and plants can break down cyanide into non-toxic compounds (biodegradation). Through these processes, cyanide ultimately degrades into nontoxic carbon dioxide and nitrates.

In soil, cyanide concentrations and toxicity can also decrease through volatization and biodegradation. If cyanide seeps into ground water, it is less likely to be degraded due to lack of sunlight and oxygen. In highly contaminated settings, cyanide can also sterilize microorganisms within the soil, consequently preventing its degradation. Hence, cyanide has been detected in groundwater close to landfill sites where leachates entered the groundwater (ATSDR, 2006a).

The natural degradation rate for different cyanide species differs, depending on the factors influencing the degradation processes. The main factors are 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.

Figure 12. Dominant natural degradation processes of cyanide in tailings ponds



Preventive measures for reducing cyanide risks at artisanal mining sites

Cyanide use in artisanal mining can pose a major health and environmental hazard and it should never be used to recover gold from mercury-contaminated tailings. Cyanide use therefore needs to be carefully controlled and should only be considered in settings where it is used by organized and trained miners in compliance with chemical management protocols (see Stapper, et al., 2021 on best management practices of cyanide use in ASGM; UNEP, 2021).

For example, all operations using cyanide should have a cyanide emergency response plan and a trained person on site to provide first aid in case of a cyanide exposure. Health and environmental risks associated with cyanide use can be controlled through various preventive measures. These include, for example, the safe and secure storage of cyanide in dry and well-ventilated places in a closed container with a label signaling dangerous content. Sodium cyanide decomposes on contact with acids, water and carbon dioxide, producing highly toxic, flammable hydrogen cyanide gas.

Safe operating standards should include for example spill prevention and response, the use of personal protective equipment of workers, as well as the maintenance of an appropriate pH 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

Unlike for mercury, there is currently no international regulatory framework for cyanide use in artisanal mining. The Cyanide Code is a voluntary initiative of large-scale gold and silver mining industries to standardize and monitor safe practices (UNEP, 2021). While such a Code could be an important social and environmental safeguard in ASGM, lack of organization, safety standards, costs and prevailing use of mercury are current barriers.

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 for 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. Silica particles can penetrate different parts of the lungs depending on their size (Fig. 13); smaller particles are therefore most dangerous because they can travel deeper into the respiratory system and cause greater damage than larger particles.

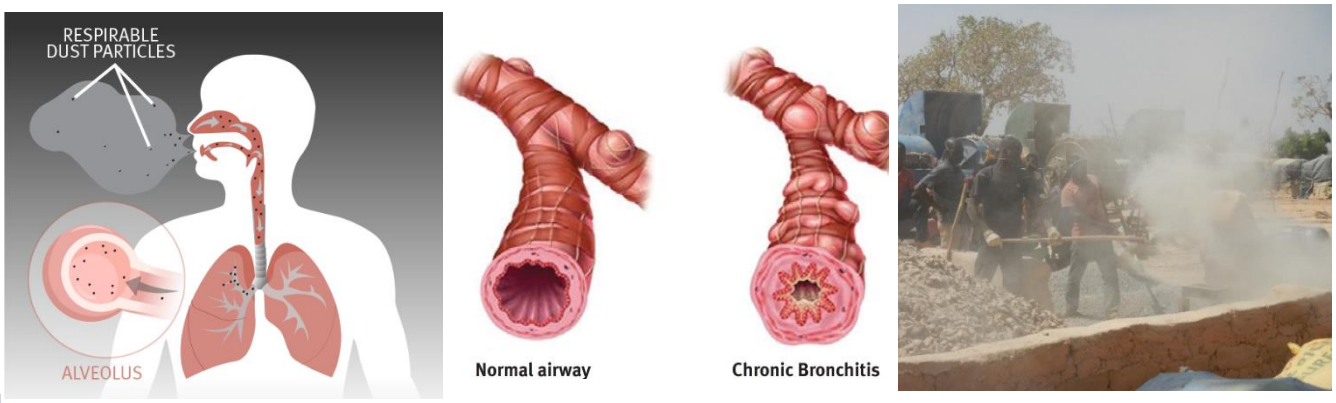
The exposure to silica usually goes unnoticed because its inhalation does not cause any immediate health effects such as lung irritation. Therefore, silicosis – one of the oldest occupational diseases – is still considered a hidden epidemic in many countries.

There are no international guidelines on occupational exposure limits to silica dust and national occupational exposure thresholds vary between 0.025 and 3 mg/m^3. The US National Institute for Occupational Safety and Health (NIOSH) recommends an exposure limit (REL) of 0.05 mg/m^3 as a time-weighted average (TWA) for up to 10 hours/day during a 40-hour workweek (CDC, 2014).

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/m^3 over a period of 45 years causes silicosis in 5 out of 100 people, the exposure to twice as much silica dust (0.1 mg/m^3) increases the risk to 30 in 100 people.

Data on silica exposure in the ASGM sector are limited but field observations suggests it is extensive, especially in hard rock mining (Fig. 15). A study in Tanzania found average concentrations of 0.19 mg/m^3 in aboveground operations, exceeding the recommended exposure limit defined by NIOSH by four times

Figure 13 (left) & 14 (center). Dust particles are so small that they are able to travel deep into our lungs; scar tissue causes an obstruction of the airways (© Resources Safety & Health Queensland, 2020)
Figure 15 (right). Dust exposure in artisanal hard rock mining is a common threat (Photo: AGC)



(Gottesfeld et al., 2015). 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.

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 stop exposing him or herself 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.

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ARTISANAL GOLD COUNCIL

AGC's health-related activities in the artisanal gold mining sector

Capacity development

- Awareness building on mercury intoxication for various stakeholder groups along the gold supply chain
- 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

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

Research & monitoring

- Research on health hazards and their relation to livelihoods in artisanal mining communities
- Institutional capacity assessments of the health care sector to respond to occupational diseases in artisanal mining
- 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