

Environmental & Health Effects

This document is a general summary of cyanide's effects on human health and the environment, and is not intended to be a complete reference on all the environmental and health effects of cyanide.

Human Health Effects

Cyanide is produced in the human body and exhaled in extremely low concentrations with each breath. It is also produced by over 1,000 plant species including sorghum, bamboo and cassava. Relatively low concentrations of cyanide can be highly toxic to people and wildlife.

Cyanide is acutely toxic to humans. Liquid or gaseous hydrogen cyanide and alkali salts of cyanide can enter the body through inhalation, ingestion or absorption through the eyes and skin. The rate of skin absorption is enhanced when the skin is cut, abraded or moist; inhaled salts of cyanide are readily dissolved and absorbed upon contact with moist mucous membranes.

The toxicity of hydrogen cyanide to humans is dependent on the nature of the exposure. Due to the variability of dose-response effects between individuals, the toxicity of a substance is typically expressed as the concentration or dose that is lethal to 50% of the exposed population (LC50 or LD50). The LC50 for gaseous hydrogen cyanide is 100-300 parts per million. Inhalation of cyanide in this range results in death within 10-60 minutes, with death coming more quickly as the concentration increases. Inhalation of 2,000 parts per million hydrogen cyanide causes death within one minute. The LD50 for ingestion is 50-200 milligrams, or 1-3 milligrams per kilogram of body weight, calculated as hydrogen cyanide. For contact with unabraded skin, the LD50 is 100 milligrams (as hydrogen cyanide) per kilogram of body weight.

Although the time, dose and manner of exposure may differ, the biochemical action of cyanide is the same upon entering the body. Once in the bloodstream, cyanide forms a stable complex with a form of cytochrome oxidase, an enzyme that promotes the transfer of electrons in the mitochondria of cells during the synthesis of ATP. Without proper cytochrome oxidase function, cells cannot utilize the oxygen present in the bloodstream, resulting in cytotoxic hypoxia or cellular asphyxiation. The lack of available oxygen causes a shift from aerobic to anaerobic metabolism, leading to the accumulation of lactate in the blood. The combined effect of the hypoxia and lactate acidosis is depression of the central nervous system that can result in respiratory arrest and death. At higher lethal concentrations, cyanide poisoning also affects other organs and systems in the body, including the heart.

Initial symptoms of cyanide poisoning can occur from exposure to 20 to 40 ppm of gaseous hydrogen cyanide, and may include headache, drowsiness, vertigo, weak and rapid pulse, deep and rapid breathing, a bright-red color in the face, nausea and vomiting. Convulsions, dilated pupils, clammy skin, a weaker and more rapid pulse and slower, shallower breathing can follow these symptoms. Finally, the heartbeat becomes slow and irregular, body temperature falls, the lips, face and extremities take on a blue color, the individual falls into a coma, and death occurs. These symptoms can occur from sublethal exposure to cyanide, but will diminish as the body detoxifies the poison and excretes it primarily as thiocyanate and 2 amino thiazoline 4 carboxylic acid, with other minor metabolites.

The body has several mechanisms to effectively detoxify cyanide. The majority of cyanide reacts with thiosulfate to produce thiocyanate in reactions catalyzed by sulfur transferase enzymes such as rhodanese. The thiocyanate is then excreted in the urine over a period of days. Although thiocyanate is approximately seven times less toxic than cyanide, increased thiocyanate concentrations in the body resulting from chronic cyanide exposure can adversely affect the thyroid. Cyanide has a greater affinity for methemoglobin than for

cytochrome oxidase, and will preferentially form cyanomethemoglobin. If these and other detoxification mechanisms are not overwhelmed by the concentration and duration of cyanide exposure, they can prevent an acute cyanide-poisoning incident from being fatal.

Some of the available antidotes to cyanide poisoning take advantage of these natural detoxifying mechanisms. Sodium thiosulfate, administered intravenously, provides sulfur to enhance the sulfur transferase-mediated transformation of cyanide to thiocyanate. Amyl nitrite, sodium nitrite and dimethyl aminophenol (DMAP) are used to increase the amount of methemoglobin in the blood, which then binds with cyanide to form non-toxic cyanomethemoglobin. Cobalt compounds are also used to form stable, non-toxic cyanide complexes, but as with nitrite and DMAP, cobalt itself is toxic.

Cyanide does not accumulate or biomagnify, so chronic exposure to sublethal concentrations of cyanide does not appear to result in acute toxicity. However, chronic cyanide poisoning has been observed in individuals whose diet includes significant amounts of cyanogenic plants such as cassava. Chronic cyanide exposure is linked to demyelination, lesions of the optic nerve, ataxia, hypertonia, Leber's optic atrophy, goiters and depressed thyroid function.

There is no evidence that chronic cyanide exposure has teratogenic, mutagenic or carcinogenic effects.

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Cyanide in the Environment

Cyanide is produced naturally in the environment by various bacteria, algae, fungi and numerous species of plants including beans (chickpeas and lima), fruits (seeds and pits of apple, cherry, pear, apricot, peach and plum), almond and cashew nuts, vegetables of the cabbage family, grains (alfalfa and sorghum), roots (cassava, potato, radish and turnip), white clover and young bamboo shoots. Incomplete combustion during forest fires is believed to be a major environmental source of cyanide, and incomplete combustion of articles containing nylon produces cyanide through depolymerization.

Once released in the environment, the reactivity of cyanide provides numerous pathways for its degradation and attenuation:

Complexation: Cyanide forms ionic complexes of varying stability with many metals. Most cyanide complexes are much less toxic than cyanide, but weak acid dissociable complexes such as those of copper and zinc are relatively unstable and will release cyanide back to the environment. Iron cyanide complexes are of particular importance due to the abundance of iron typically available in soils and the extreme stability of this complex under most environmental conditions. However, iron cyanides are subject to photochemical decomposition and will release cyanide if exposed to ultraviolet light. Metal cyanide complexes are also subject to other reactions that reduce cyanide concentrations in the environment, as described below.

Precipitation: Iron cyanide complexes form insoluble precipitates with iron, copper, nickel, manganese, lead, zinc, cadmium, tin and silver. Iron cyanide forms precipitates with iron, copper, magnesium, cadmium and zinc over a pH range of 2-11.

Adsorption: Cyanide and cyanide-metal complexes are adsorbed on organic and inorganic constituents in soil, including oxides of aluminum, iron and manganese, certain types of clays, feldspars and organic carbon. Although the strength of cyanide retention on inorganic materials is unclear, cyanide is strongly bound to organic matter.

Cyanate: Oxidation of cyanide to less toxic cyanate normally requires a strong oxidizing agent such as ozone, hydrogen peroxide or hypochlorite. However, adsorption of cyanide on both organic and inorganic materials in the soil appears to promote its oxidation under natural conditions.

Thiocyanate: Cyanide reacts with some sulfur species to form less toxic thiocyanate. Potential sulfur sources include free sulfur and sulfide minerals such as chalcopyrite (CuFeS₂), chalcocite (Cu₂S) and pyrrhotite (FeS), as well as their oxidation products, such as polysulfides and thiosulfate.

Volatilization: At the pH typical of environmental systems, free cyanide will be predominately in the form of hydrogen cyanide, with gaseous hydrogen cyanide evolving slowly over time. The amount of cyanide lost through this pathway increases with decreasing pH, increased aeration of solution and with increasing temperature. Cyanide is also lost through volatilization from soil surfaces.

Biodegradation: Under aerobic conditions, microbial activity can degrade cyanide to ammonia, which then oxidizes to nitrate. This process has been shown effective with cyanide concentrations of up to 200 parts per million. Although biological degradation also occurs under anaerobic conditions, cyanide concentrations greater than 2 parts per million are toxic to these microorganisms.

Hydrolysis: Hydrogen cyanide can be hydrolyzed to formic acid or ammonium formate. Although this reaction is not rapid, it may be of significance in ground water where anaerobic conditions exist.

Effects on Wildlife: Although cyanide reacts readily in the environment and degrades or forms complexes and salts of varying stabilities, it is toxic to many living organisms at very low concentrations.

Aquatic Organisms: Fish and aquatic invertebrates are particularly sensitive to cyanide exposure. Concentrations of free cyanide in the aquatic environment ranging from 5.0 to 7.2 micrograms per liter reduce swimming performance and inhibit reproduction in many species of fish. Other adverse effects include delayed mortality, pathology, susceptibility to predation, disrupted respiration, osmoregulatory disturbances and altered growth patterns. Concentrations of 20 to 76 micrograms per liter free cyanide cause the death of many species, and concentrations in excess of 200 micrograms per liter are rapidly toxic to most species of fish. Invertebrates experience adverse nonlethal effects at 18 to 43 micrograms per liter free cyanide, and lethal effects at 30 to 100 micrograms per liter (although concentrations in the range of 3 to 7 micrograms per liter caused death in the amphipod *Gammarus pulex*).

Algae and macrophytes can tolerate much higher environmental concentrations of free cyanide than fish and invertebrates, and do not exhibit adverse effects at 160 micrograms per liter or more. Aquatic plants are unaffected by cyanide at concentrations that are lethal to most species of freshwater and marine fish and invertebrates. However, differing sensitivities to cyanide can result in changes to plant community structure, with cyanide exposures leaving a plant community dominated by less sensitive species.

The toxicity of cyanide to aquatic life is probably caused by hydrogen cyanide that has ionized, dissociated or photochemically decomposed from compounds containing cyanide. Toxic effects of the cyanide ion itself on aquatic organisms are not believed to be significant, nor are the effects of photolysis of ferro- and ferricyanides. It is therefore the hydrogen cyanide concentration of water that is of greatest significance in determining toxicity to aquatic life rather than the total cyanide concentration.

The sensitivity of aquatic organisms to cyanide is highly species specific, and is also affected by water pH, temperature and oxygen content, as well as the life stage and condition of the organism.

Birds: Reported oral LD₅₀ for birds range from 0.8 milligrams per kilogram of body weight (American racing pigeon) to 11.1 milligrams per kilogram of body weight (domestic chickens). Symptoms including panting, eye blinking, salivation and lethargy appear within one-half to five minutes after ingestion in more sensitive species, and up to ten minutes after ingestion by more resistant species. Exposures to high doses resulted in deep, labored breathing followed by gasping and shallow intermittent breathing in all species. Mortality typically occurred in 15 to 30 minutes; however birds that survived for one hour frequently recovered, possibly due to the rapid metabolism of cyanide to thiocyanate and its subsequent excretion.

Ingestion of WAD cyanide solutions by birds may cause delayed mortality. It appears that birds may drink water containing WAD cyanide that is not immediately fatal, but which breaks down in the acidic conditions in the stomach and produces sufficiently high cyanide concentrations to be toxic.

Sublethal effects of cyanide exposure to birds, such as an increase in their susceptibility to predators, have not been fully investigated and reported.

Mammals: Cyanide toxicity to mammals is relatively common due to the large number of cyanogenic forage plants such as sorghum, sudan grasses and corn. Concentrations of cyanide in these plants are typically highest in the spring during blooming. Dry growing conditions enhance the accumulation of cyanogenic glycosides in certain plants as well as increase the use of these plants as forage.

Reported oral LD50 for mammals range from 2.1 milligrams per kilogram of body weight (coyote) to 6.0-10.0 milligrams per kilogram of body weight (laboratory white rats). Symptoms of acute poisoning usually occur within ten minutes of ingestion, including: initial excitability with muscle tremors; salivation; lacrimation; defecation; urination; labored breathing; followed by muscular incoordination, gasping and convulsions. In general, cyanide sensitivity for common livestock decreases from cattle to sheep to horses to pigs; deer and elk appear to be relatively resistant.

Although present in the environment and available in many plant species, cyanide toxicity is not widespread due to a number of significant factors:

Cyanide has low persistence in the environment and is not accumulated or stored in any mammal studied.

There is no reported biomagnification of cyanide in the food chain.

Although chronic cyanide intoxication exists, cyanide has a low chronic toxicity. Repeated sublethal doses of cyanide seldom result in cumulative adverse effects. Many species can tolerate cyanide in substantial yet sublethal intermittent doses for long periods of time.

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