Sodium Cyanide Hazards to Fish and Other Wildlife from Gold Mining Operations

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5.1 Introduction

Highly toxic sodium cyanide (NaCN) is used increasingly by the international mining community to extract gold and other precious metals through milling of high grade ores and heap leaching of low grade ores. Of the 98 million kg cyanide (CN) consumed in North America in 1989, about 80% was used in gold mining (Knudson 1990). In Canada, more than 90% of the mined gold is extracted from ores with the cyanidation process. This process consists of leaching gold from the ore as a gold–cyanide complex, and gold being recovered by precipitation (Simovic and Snodgrass 1985). Milling and heap leaching require cycling of millions of liters of alkaline water containing high concentrations of potentially toxic NaCN, free cyanide, and metal cyanide complexes that are frequently accessible to wildlife. Some milling operations result in tailings ponds of 150 ha and larger. Heap leach operations that spray or drip cyanide solution onto the flattened top of the ore heap require solution processing ponds of about 1 ha in surface area. Although not intentional or desired, puddles of various sizes may occur on the top of heaps where the highest concentrations of NaCN are found. Exposed solution recovery channels are usually constructed at the base of leach heaps. All of these cyanide-containing water bodies are hazardous to wildlife if not properly managed (Henny *et al.* 1994). In this account we emphasize hazards of cyanide from mining operations to fish and wildlife species and proposed mitigation to protect them.

5.2 Background

Heap leaching occurs when run-of-mine or crushed ore is stacked on an impermeable plastic pad on the ground surface, with spraying or dripping of a NaCN solution on the flattened top. Large leach heaps may include 272 000 tons of ore and tower 100 m or more (Alberswerth *et al.* 1989). In the milling of gold ores, a NaCN solution is percolated through the crushed ore to dissolve the gold particles (Ripley *et al.* 1996). In both leaching and milling processes, after the gold is chemically precipitated, the solution is adjusted for pH and cyanide concentration, and recycled to precipitate more gold. Eventually, the remaining solution must be treated to recycle the cyanide or to destroy it to prevent escape into the environment.

Alkaline chlorination of wastewaters is one of the most widely used methods of treating cyanide wastes. In this process, cyanogen chloride (CNCl) is formed, which at alkaline pH is hydrolyzed to the cyanate ion (CNO^-). If free chlorine is present, CNO^- can be further oxidized (Way 1981; Leduc *et al.* 1982; Simovic and Snodgrass 1985; Marrs

and Ballantyne 1987). The use of sulfur dioxide in a high dissolved oxygen environment with a copper catalyst reportedly reduces total cyanide in high cyanide rinsewaters from metal plating shops to less than 1 mgl^{-1}; this process may have application in cyanide detoxification of tailings ponds (Robbins 1996). Other methods used in cyanide waste management include lagooning for natural degradation, evaporation, exposure to ultraviolet radiation, aldehyde treatment, ozonization, acidification–volatilization–reneutralization, ion exchange, activated carbon absorption, electrolytic decomposition, catalytic oxidation, and biological treatment with cyanide-metabolizing bacteria (Towill *et al.* 1978; USEPA 1980; Way 1981; Marrs and Ballantyne 1987; Smith and Mudder 1991). Additional cyanide detoxification treatments include the use of $FeSO_4$, $FeSO_4$ plus CO_2, H_2O_2, $Ca(OCl)_2$ (Henny *et al.* 1994), dilution with water, $FeSO_4$ plus H_2O_2, and $(NH_4)HSO_3$ (Wiemeyer, pers. comm.). In Canadian gold-mining operations, the primary treatment for cyanide removal is to retain mill wastewaters in impoundments for several days to months; removal occurs through volatilization, photodegradation, chemical oxidation, and, to a lesser extent, microbial oxidation. Microbial oxidation of cyanide is not significant at this time in mine tailing ponds, which typically have pH >10, a low number of microorganisms, low nutrient levels, large quiescent zones, and cyanide concentrations >10 mg l^{-1} (Simovic and Snodgrass 1985).

Cyanide seldom remains biologically available in soils because it is either complexed by trace metals, metabolized by various microorganisms, or lost through volatilization (Towill *et al.* 1978; Marrs and Ballantyne 1987). Cyanide ions are not strongly adsorbed or retained on soils, and leaching into the surrounding ground water will probably occur. Under aerobic conditions, cyanide salts in the soil are microbially degraded to nitrites or form complexes with trace metals. Under anaerobic conditions, cyanides denitrify to gaseous nitrogen compounds that enter the atmosphere. Mixed microbial communities capable of metabolizing cyanide and not previously exposed to cyanide are adversely affected at 0.3 mg HCN kg^{-1}; however, these communities can become acclimatized to cyanide and can then degrade wastes with higher cyanide concentrations (Towill *et al.* 1978). Acclimatized microbes in activated sewage sludge can often completely convert nitriles to ammonia, sometimes at concentrations as high as 60 mg total CN kg^{-1} (Towill *et al.* 1978).

In regard to cyanide use and toxicity in the recovery of precious metals, most authorities currently agree on nine points:

1. Metal mining operations consume most of the current cyanide production.
2. The greatest source of cyanide exposure to humans and range animals is cyanogenic food plants and forage crops–not mining operations.
3. Cyanide is ubiquitous in the environment, with gold-mining facilities only one of many sources of elevated concentrations.
4. Many chemical forms of cyanide are present in the environment, including free cyanide, metallocyanide complexes, and synthetic organocyanides, but only free cyanide (i.e., the sum of molecular hydrogen cyanide, HCN, and the cyanide anion, CN^-) is the primary toxic agent, regardless of origin.
5. Cyanides are readily absorbed through inhalation, ingestion, or skin contact, and are readily distributed throughout the body via blood. Cyanide is a potent and rapid-acting asphyxiant; it induces tissue anoxia through inactivation of cytochrome oxidase, causing cytotoxic hypoxia in the presence of normal hemoglobin oxygenation.

6. At sublethal doses, cyanide reacts with thiosulfate in the presence of rhodanese to produce the comparatively nontoxic thiocyanate, most of which is excreted in the urine. Rapid detoxification enables animals to ingest high sublethal doses of cyanide over extended periods without harm.
7. Cyanides are not mutagenic or carcinogenic.
8. Cyanide does not biomagnify in food webs or cycle extensively in ecosystems, probably because of its rapid breakdown.
9. Cyanide seldom persists in surface waters and soils owing to complexation or sedimentation, microbial metabolism, and loss from volatilization.

(Doudoroff 1976; Towill *et al.* 1978; Smith *et al.* 1979; Egekeze and Oehme 1980; USEPA 1980, 1989; Moore 1981; Vennesland *et al.* 1981; Leduc *et al.* 1982; Biehl 1984; Leduc 1984; Way 1984; Ballantyne and Marrs 1987; Evered and Harnett 1988; Eisler 1991; Smith and Mudder 1991; Hill and Henry 1996; Ripley *et al.* 1996).

5.3 Effects

5.3.1 Aquatic Ecosystems

Accidental spills of cyanide solutions into rivers and streams have produced massive kills of fish, amphibians, aquatic insects, and aquatic vegetation. Precious-metal waste-sources of poisonings have included storage reservoirs of concentrated solutions, overturned rail tank cars, and discharge of substances generating free HCN in the water from hydrolysis or decomposition (Leduc 1984; Albersworth *et al.* 1989). Fish kills from accidental discharges of cyanide gold mining wastes are common (Holden and Marsden 1964; Leduc 1978; Towill *et al.* 1978; USEPA 1980; Albersworth *et al.* 1989; Ripley *et al.* 1996). In one case, mine effluents containing cyanide from a Canadian tailings pond released into a nearby creek killed more than 20 000 steelhead (*Oncorhynchus mykiss*; Leduc *et al.* 1982). In Colorado, overflows of 760 000 l NaCN-contaminated water from storage ponds into natural waterways killed all aquatic life along 28 km of the Alamosa River (Albersworth *et al.* 1989).

Data on the recovery of cyanide-poisoned aquatic ecosystems are scarce. In one case, a large amount of cyanide-containing slag entered a stream from the reservoir of a gold mine in Japan as a result of an earthquake (Yasuno *et al.* 1981). The slag covered the stream bed for about 10 km from the point of rupture, killing all stream biota; cyanide was detected in the water column for 3 days after the spill. Within 1 month flora was established on the silt covering the above-water stones, but there was little underwater growth. After 6–7 months, populations of fish, algae, and invertebrates had recovered, although species composition of algae was altered (Yasuno *et al.* 1981).

Cyanides do not persist in aquatic environments. In small, cold oligotrophic lakes treated with NaCN (1 mg l^{-1}), acute toxicity to aquatic organisms was negligible within 40 days. In warm shallow ponds, no toxicity was evident to aquatic organisms within 4 days after application of the same concentration of NaCN. In rivers and streams, cyanide toxicity fell rapidly on dilution (Leduc 1984). Cyanide was not detectable in water and sediments of Yellowknife Bay, Canada, between 1974 and 1976, despite the continu-

ous input of liquid effluents containing cyanides from an operating gold mine. Nondetection was attributed to rapid oxidation (Moore 1981). Several factors contribute to the rapid disappearance of cyanide from water: bacteria and protozoans may degrade cyanide by converting it to carbon dioxide and ammonia; chlorination of water supplies can result in conversion to cyanate; an alkaline pH favors oxidation by chlorine; and an acidic pH favors volatilization of HCN into the atmosphere (USEPA 1980).

Cyanide concentrations in fish from streams poisoned with cyanide ranged between 10 and 100 μg total CN kg^{-1} whole body FW (Wiley 1984). Gill tissues of salmonids contain from 30 μg kg^{-1} fresh weight (FW) total CN to >7000 μg kg^{-1} FW under widely varying conditions of temperature, nominal water concentrations of free cyanide, and duration of exposure (Holden and Marsden 1964). Unpoisoned fish usually contained <1 μg total CN kg^{-1} FW in gills, although values up to 50 μg kg^{-1} FW occurred occasionally. Lowest cyanide concentrations in gill occurred at elevated (summer) water temperatures; at lower temperatures, survival was greater and residues were higher (Holden and Marsden 1964).

Fish are the most cyanide-sensitive group of aquatic organisms tested (Eisler 1991). Under conditions of continuous exposure, adverse effects on swimming and reproduction usually occurred between 5 and 7.2 μg free CN l^{-1} and on survival between 20 and

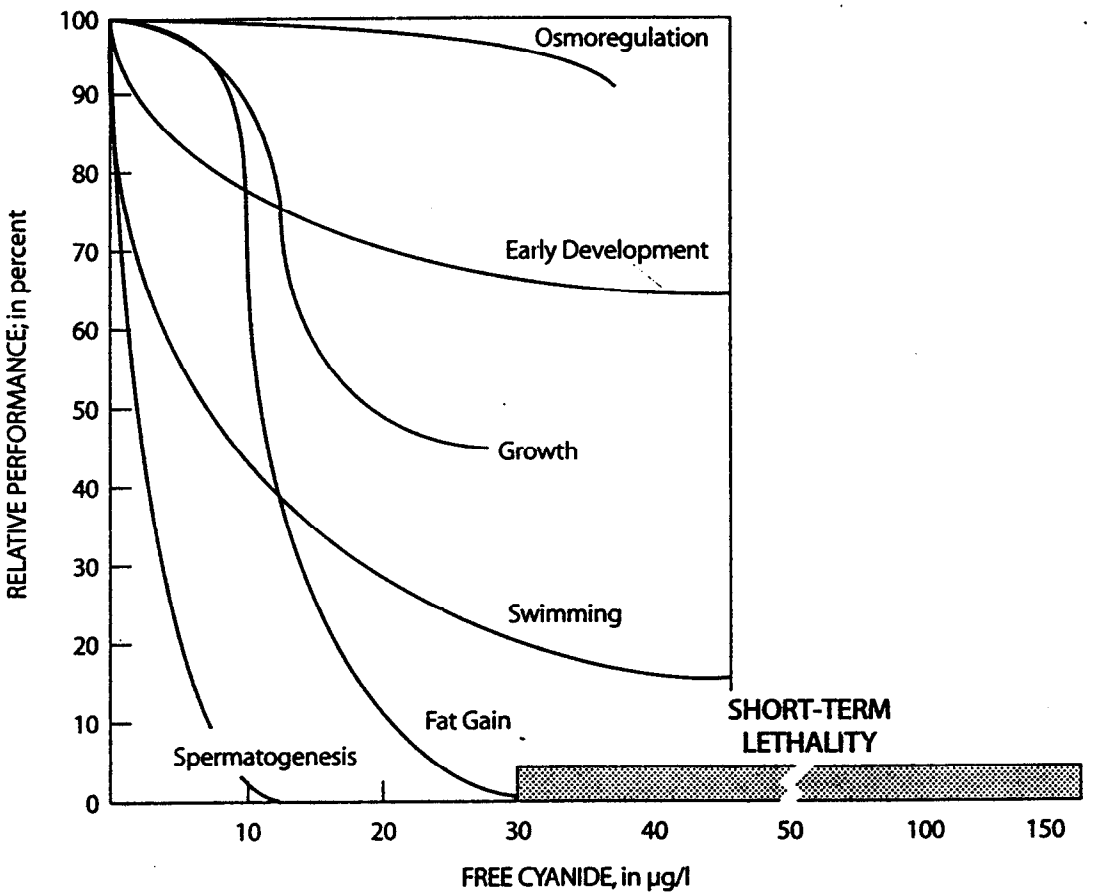

Fig. 5.1. Summary of lethal and sublethal effects of free cyanide on freshwater fish (modified from Leduc *et al.* 1982)

76 μg l^{-1} (Fig. 5.1). Other adverse effects on fish of chronic cyanide exposure included susceptibility to predation, disrupted respiration, osmoregulatory disturbances, and altered growth patterns. Free cyanide concentrations between 50 and 200 μg l^{-1} were fatal to the more-sensitive fish species over time, and concentrations >200 μg l^{-1} were rapidly lethal to most species of fish (USEPA 1980). Cyanide-induced pathology in fish included subcutaneous hemorrhaging, liver necrosis, and hepatic damage. Exposure of fish to 10 μg HCN l^{-1} for 9 days was sufficient to induce extensive necrosis in the liver, although gill tissue showed no damage. Intensification of liver histopathology was evident at dosages of 20 and 30 μg HCN l^{-1} and exposure periods up to 18 days (Leduc 1984). Cyanide has a strong, immediate, and long-lasting inhibitory effect on the swimming ability of fish (Leduc 1984). Free cyanide concentrations as low as 10 μg l^{-1} can rapidly and irreversibly impair the swimming ability of salmonids in well-aerated water (Doudoroff 1976). Osmoregulatory disturbances recorded at 10 μg HCN l^{-1} may affect migratory patterns, feeding, and predator avoidance (Leduc *et al.* 1982; Leduc 1984). Figure 5.1 shows that under laboratory conditions, fish experienced a significant reduction in relative performance (based on osmoregulation, growth, swimming, and spermatogenesis) at 10 μg HCN l^{-1} and survived indefinitely at 30 μg HCN l^{-1} (Leduc 1978, 1981; Leduc *et al.* 1982). Laboratory findings, however, may not be applicable to cyanide-stressed natural environments that are subject to wide fluctuations in physicochemical regimes. Cyanide, like many other chemicals, stimulates growth of fish during exposure to low sublethal levels. This phenomenon, known as hormesis, is little understood and warrants additional research (Leduc 1984).

Cyanide adversely affects fish reproduction by reducing both the number of eggs spawned and the viability of the eggs by delaying the process of secondary yolk deposition in the ovary (Lesniak and Ruby 1982; Ruby *et al.* 1986). Vitellogenin, a glycolipophosphoprotein present in plasma of fish during the process of yolk formation, is synthesized in the liver under stimulation of estrogen and subsequently sequestered in the ovary; it is essential for normal egg development. Exposure of naturally reproducing female rainbow trout (*Oncorhynchus mykiss*) to as little as 10 μg HCN l^{-1} for 12 days during the onset of the reproductive cycle produced a reduction in plasma vitellogenin levels and a reduction in ovary weight. The loss of vitellogenin in the plasma removed a major source of yolk (Ruby *et al.* 1986). Reproductive impairment in adult bluegills (*Lepomis macrochirus*) occurred following exposure to 5.2 μg CN l^{-1} for 289 days (USEPA 1980). Newly fertilized fish eggs were usually resistant to cyanide prior to blastula formation, but delayed effects occurred at 60 to 100 μg HCN l^{-1}, including birth defects and reduced survival of embryos and newly hatched larvae (Leduc *et al.* 1982). Concentrations as low as 10 μg HCN l^{-1} caused developmental abnormalities in embryos of Atlantic salmon (*Salmo salar*) after extended exposure (Leduc 1978). These abnormalities, which were absent in controls, included yolk sac dropsy and malformations of eyes, mouth, and vertebral column (Leduc 1984).

Among aquatic invertebrates, adverse nonlethal effects occurred between 18 and 43 μg l^{-1}, and lethal effects between 30 and 100 μg l^{-1} – although some deaths occurred between 3 and 7 μg l^{-1} for the amphipod *Gammarus pulex* (Eisler 1991). Aquatic plants are comparatively tolerant to cyanide; adverse effects occurred at >160 μg free CN l^{-1} (Eisler 1991). Adverse effects of cyanide on aquatic plants are unlikely at concentrations that cause acute effects to most species of freshwater and marine fishes and invertebrates (USEPA 1980).

Biocidal properties of cyanide in aquatic environments may be significantly modified by water pH, temperature, and oxygen content; life stage, condition, and species assayed; previous exposure to cyanides; presence of other chemicals; and initial dose tested. There is general agreement that cyanide is more toxic to freshwater fishes under conditions of low dissolved oxygen (Doudoroff 1976; Towill *et al.* 1978; Smith *et al.* 1979; USEPA 1980; Leduc 1984); that pH levels within the range 6.8 to 8.3 have little effect on cyanide toxicity but enhance toxicity at more acidic pH (Smith *et al.* 1979; USEPA 1980; Leduc *et al.* 1982; Leduc 1984); that juveniles and adults are the most sensitive life stages and embryos and sac fry the most resistant (Smith *et al.* 1978, 1979; USEPA 1980; Leduc 1984); and that substantial interspecies variability exists in sensitivity to free cyanide (Smith *et al.* 1979; USEPA 1980). Initial dose and water temperature both modify the biocidal properties of HCN to freshwater teleosts. At low lethal concentrations (i.e., near 10 μg HCN l^{-1}), cyanide is more toxic at lower temperatures; at high, rapidly lethal HCN concentrations, cyanide is more toxic at elevated temperatures (Kovacs and Leduc 1982a, b; Leduc *et al.* 1982; Leduc 1984). By contrast, aquatic invertebrates are most sensitive to HCN at elevated water temperatures, regardless of dose (Smith *et al.* 1979). Season and exercise modify the lethality of HCN to juvenile rainbow trout (McGeachy and Leduc 1988); higher tolerance to cyanide correlates with higher activity induced by exercise and higher temperatures, suggesting a faster detoxification rate or higher oxidative and anaerobic metabolism. Low levels of cyanide that are harmful when applied constantly may be harmless under seasonal or other variations that allow the organism to recover and detoxify (Leduc 1981).

Acclimatization by fish to sublethal levels of cyanide through continuous exposure was theorized to enhance their resistance to potentially lethal concentrations (Leduc 1981, 1984), but studies with Atlantic salmon and rainbow trout indicate otherwise. Prior acclimatization of Atlantic salmon smolts to cyanide increased their tolerance only slightly to potentially lethal concentrations (Alabaster *et al.* 1983). Juvenile rainbow trout previously exposed to sublethal concentrations showed a marked reduction in fat synthesis and swimming performance when challenged with higher cyanide doses; effects were most pronounced at low water temperatures (Kovacs and Leduc 1982a).

Knowledge of cyanide interactions with other chemicals is essential to the understanding of cyanide toxicokinetics and in evaluating risk to living resources. Additive or synergistic toxicity of free cyanide to aquatic fauna may occur in combination with ammonia (Smith *et al.* 1979; Leduc *et al.* 1982; Alabaster *et al.* 1983; Leduc 1984) or arsenic (Leduc 1984). However, conflicting reports on the toxicity of mixtures of HCN with zinc or chromium (Towill *et al.* 1978; Smith *et al.* 1979; Leduc *et al.* 1982; Leduc 1984) require clarification. Formation of the nickel–cyanide complex markedly reduced the toxicity of both cyanide and nickel at high concentrations in alkaline pH. At lower concentrations and acidic pH, nickel–cyanide solutions increased in toxicity by more than 1000 times, owing to dissociation of the metallocyanide complex to form hydrogen cyanide (Towill *et al.* 1978). Mixtures of cyanide and ammonia may have interfered with seaward migration of Atlantic salmon smolts under conditions of low dissolved oxygen (Alabaster *et al.* 1983). The 96-h toxicity of mixtures of sodium cyanide and nickel sulfate to fathead minnows (*Pimephales promelas*) was influenced by water alkalinity and pH. Toxicity, as measured by LC50 values, decreased with increasing alkalinity and pH from 0.42 mg CN l^{-1} at 5 mg $CaCO_3$ l^{-1} and pH 6.5, to 1.4 mg CN l^{-1} at 70 mg $CaCO_3$ l^{-1} and pH 7.5, to 730 mg CN l^{-1} at 192 mg $CaCO_3$ l^{-1} and pH 8.0 (Doudoroff 1956).

5.3.2 Birds

Many species of migratory birds – including waterfowl, shorebirds, passerines, and raptors – were found dead in the immediate vicinity of gold-mine heap-leach extraction facilities and tailings ponds, presumably as a result of drinking the cyanide-contaminated waters (Clark and Hothem 1991; Henny *et al.* 1994; Hill and Henry 1996). About 7000 dead birds – mostly waterfowl and songbirds – were recovered from cyanide-extraction, gold-mine leach ponds in the western United States between 1980 and 1989; no gross pathological changes related to cyanide were observed in these birds at necropsy (Allen 1990; Clark and Hothem 1991). No gross pathology was evident in cyanide-dosed captive birds (Wiemeyer *et al.* 1986), and this is similar to the findings of laboratory studies with cyanide and other animal orders that were tested and examined (Eisler 1991).

Free cyanide levels associated with high avian death rates have included 0.12 mg l^{-1} in air, 2.1–4.6 mg kg^{-1} body weight (BW) via acute oral exposure, and 1.3 mg kg^{-1} BW administered intravenously. In cyanide-tolerant species, such as the domestic chicken (*Gallus domesticus*), dietary levels of 135 mg total CN kg^{-1} ration resulted in growth reduction of chicks, but 103 mg total CN kg^{-1} ration had no measurable effect on these chicks (Eisler 1991; Hill and Henry 1996). First signs of cyanide toxicosis in sensitive birds appeared between 0.5 and 5 min after exposure, and included panting, eye blinking, salivation, and lethargy (Wiemeyer *et al.* 1986). In more tolerant species, signs of toxicosis began 10 min after exposure. At higher doses, breathing in all species tested became increasingly deep and labored, followed by gasping and shallow intermittent breathing. Death usually followed in 15 to 30 min, although birds alive at 60 min frequently recovered (Wiemeyer *et al.* 1986). The rapid recovery of some birds exposed to cyanide may be due to the rapid metabolism of cyanide to thiocyanate and its subsequent excretion. Species sensitivity to cyanide is not related to body size but seems to be associated with diet (Wiemeyer *et al.* 1986). Birds that feed predominantly on flesh, such as black vultures (*Coragyps atratus*), American kestrels (*Falco sparverius*), and eastern screech-owls (*Otus asio*), were more sensitive to NaCN than were species that feed mainly on plant material – with the possible exception of mallards (*Anas platyrhynchos*) – as judged by acute oral LD50 values (Table 5.1).

Some birds may not die immediately after drinking lethal cyanide solutions. Sodium cyanide rapidly forms free cyanide in the avian digestive tract (pH 1.3–6.5), whereas formation of free cyanide from metal cyanide complexes is comparatively slow (Huiatt *et al.* 1983). A high rate of cyanide absorption is critical to acute toxicity, and absorption may be retarded by the lower dissociation rates of metal-cyanide complexes (Henny *et al.* 1994). In Arizona, a single red-breasted merganser (*Mergus serrator*) was found dead 20 km from the nearest known source of cyanide, and its pectoral muscle tissue tested positive for cyanide (Clark and Hothem 1991). A proposed mechanism to account for this phenomenon involves weak-acid dissociable (WAD) cyanide compounds. Cyanide bound to certain metals, usually copper, is dissociable in weak acids such as stomach acids. Clark and Hothem (1991) suggested that drinking of lethal cyanide solutions by animals may not result in immediate death if the cyanide level is sufficiently low; these animals may die later when additional cyanide is liberated by stomach acid. More research is needed on WAD cyanide compounds and delayed mortality.

Table 5.1. Single dose toxicity of sodium cyanide (in mg NaCN kg^{-1} body weight) fatal to 50% of selected birds and mammals (listed from most sensitive to most tolerant)

Species	Oral LD50 (95% confidence limits)	Reference[a]
Mallard (*Anas platyrhynchos*)	2.7 (2.2–3.2)	1
Human (*Homo sapiens*)	3.0 estimated	2
American kestrel (*Falco sparverius*)	4.0 (3.0–5.3)	3
Coyote (*Canis latrans*)	4.1 (2.1–8.3)	4
Black vulture (*Coragyps atratus*)	4.8 (4.4–5.3)	3
Laboratory rat (*Rattus norvegicus*)	5.1–6.4	5, 6
Little brown bat (*Myotis lucifugus*)	8.4 (5.9–11.9)	7, 8
Eastern screech-owl (*Otus asio*)	8.6 (7.2–10.2)	3
House mouse (*Mus musculus*)	8.7 (8.2–9.3)	7, 8
Japanese quail (*Coturnix japonica*)	9.4 (7.7–11.4)	3
European starling (*Sturnus vulgaris*)	17 (14–22)	3
Domestic chicken (*Gallus domesticus*)	21 (12–36)	3
White-footed mouse (*Peromyscus leucopus*)	28 (18–43)	7, 8

[a] *1*, Henny *et al.* (1994); *2*, Way (1981); *3*, Wiemeyer *et al.* (1986); *4*, Sterner (1979); *5*, Ballantyne (1987); *6*, Egekeze and Oehme (1980); *7*, Clark *et al.* (1991); *8*, D.R. Clark Jr. (unpubl.).

Cyanide is a respiratory poison because of its affinity for the cytochrome oxidase complex of the mitochondrial respiratory chain (Keilin 1929; Nicholls *et al.* 1972). High dosages of cyanide are lethal through inhibition of cytochrome oxidase via cessation of mitochondrial respiration and depletion of ATP (Jones *et al.* 1984). Mallards given single oral doses of KCN (1.0 mg KCN kg^{-1} BW) at cyanide concentrations and amounts similar to those found at gold-mining tailings ponds (40 mg $CN^{-}l^{-1}$) – although it is NaCN that is used almost exclusively in mining – had elevated concentrations of creatine kinase in serum, suggesting tissue damage (Pritsos and Ma 1997). At 0.5 mg KCN kg^{-1} BW, mitochondrial function (an indicator of oxygen consumption) and ATP concentrations were significantly depressed in heart, liver, and brain (Ma and Pritsos 1997). Rhodanese and 3-mercaptopyruvate sulfurtransferase – two enzymes associated with cyanide detoxification – were induced in brain but not in liver or heart of KCN-dosed mallards. Although cyanide concentrations as high as 2.0 mg KCN kg^{-1} BW (at 80 mg $CN^{-}l^{-1}$) were not acutely toxic to mallards, the long term effects of such exposures were not determined and may have serious consequences for migratory birds exposed sublethally to cyanide at gold mine tailings ponds.

5.3.3 Mammals

Gold and silver mining are probably the most widespread sources of anthropogenic cyanides in critical wildlife habitat, such as deserts in the western United States (Hill

and Henry 1996). Between 1980 and 1989, 519 mammals – mostly rodents (35%) and bats (34%) – were reported as found dead at cyanide-extraction, gold-mine mill tailings and heap leach ponds in California, Nevada, and Arizona (Clark and Hothem 1991). The list also included such species as coyote (*Canis latrans*), badger (*Taxidea taxus*), beaver (*Castor canadensis*), mule deer (*Odocoileus hemionus*), blacktail jackrabbit (*Lepus californicus*), and kit fox (*Vulpes macrotis*), as well as skunks, chipmunks, squirrels, and domestic dogs, cats, and cattle. Also found dead at these same ponds were 38 reptiles, 55 amphibians, and 6997 birds. At the time of this study (1980–1989) there were about 160 cyanide-extraction gold mines operating in California, Nevada, and Arizona and these mines were operating within the geographic ranges of 10 endangered, threatened, or otherwise protected species of mammals. Bats comprised 6 of the 10 listed species. Because bats were not identified to species, members of these 6 protected species could have been among the 174 reported dead bats (Clark and Hothem 1991). A population of Townsend's big-eared bats (*Plecotus townsendii*), one of the 10 protected species, may have been extirpated by cyanide at a nearby mine in California (Dr. P. Brown, pers. comm.). Badgers were another of the 10 protected species; 6 were counted among the 519 mammals reported dead. A vat-leach gold mine in South Carolina with a large tailings pond reported 271 dead vertebrates found in the immediate vicinity between December 1988 and the end of 1990; 86% were birds, 13% mammals (29 of 35 were bats), and the rest reptiles and amphibians (Clark 1991).

Signs of acute cyanide poisoning in livestock usually occur within 10 min and include initial excitability with muscle tremors, salivation, lacrimation, defecation, urination, and labored breathing, followed by muscular incoordination, gasping, and convulsions; death may occur quickly, depending on the dose administered (Towill *et al.* 1978; Cade and Rubira 1982). Acute oral LD50 values for representative species of mammals ranged between 4.1 and 28.0 mg HCN kg^{-1} BW and overlapped those of birds (Table 5.1). Despite the high lethality of large single exposures, repeated sublethal doses – especially in diets – are tolerated by many species for extended periods, perhaps indefinitely (Eisler 1991). Livestock found dead near a cyanide disposal site had been drinking surface water runoff that contained up to 365 mg HCN l^{-1} (USEPA 1980). Rats exposed for 30 days to 100 or 500 mg KCN l^{-1} drinking water had mitochondrial dysfunction, depressed ATP concentrations in liver and heart, and a depressed growth rate; little effect was observed at 50 mg KCN l^{-1} (Pritsos 1996). The adverse effect on growth is consistent with the biochemical indicators of energy depletion. However, the concentrations should be viewed with caution as cyanide may have volatilized from the water solutions prior to ingestion by the rats, due to presumed neutral pH.

Hydrogen cyanide in the liquid state can readily penetrate the skin (Homan 1987). Skin ulceration has been reported from splash contact with cyanides among workers in the electroplating and gold extraction industries, although effects in those instances were more likely due to the alkalinity of the aqueous solutions (Homan 1987). In one case, liquid HCN ran over the bare hand of a worker wearing a fresh air respirator; he collapsed into unconsciousness in 5 min, but ultimately recovered (USEPA 1980). No human cases of illness or death due to cyanide in water supplies are known (USEPA 1980). Accidental acute cyanide poisonings in humans are rare (Towill *et al.* 1978); however, a male accidentally splashed with molten NaCN died about 10 h later (Curry 1963).

5.4
Proposed Mitigation

Aquatic birds are naturally attracted to large open ponds, and efforts to deter or chemically repel them have been generally ineffective (Hill and Henry 1996). However, some chemical repellents when added to dump leachate pond water showed promise at reducing consumption of leachate water when tested on European starlings (*Sturnus vulgaris*), especially o-aminoacetophenone and 4-ketobenztriazine (Clark and Shah 1993). Exclusion from cyanide solutions or reductions of cyanide concentrations to nontoxic levels are the only certain methods of protecting vertebrate wildlife from mine water poisoning (Henny *et al.* 1994). Mortality of migratory birds from cyanide toxicosis may be curtailed at small ponds associated with leach heaps by screening birds from toxic solutions (Hallock 1990). Fencing and covering of small solution ponds with polypropylene netting have proved effective for excluding most birds, bats, and larger mammals, provided that the fencing and netting are properly maintained (Henny *et al.* 1994). A few mines in Nevada are now covering surfaces of small ponds with approximately 10.2 cm-diameter high density polyethylene balls (Wiemeyer, pers. observations); birds are no longer attracted to these ponds as water sources. Although initial costs of the balls are higher than installation of netting, there are no maintenance expenses for the balls, whereas netting needs continual maintenance. Gold mine operators in southern California and Nevada used plastic sheeting to cover the cyanide leach pond, resulting in a cessation of wildlife mortality. The comparatively high cost of this process was soon recouped through reduced evaporation of water and cyanide (Wiemeyer, pers. observations; Clark and Hothem, pers. observations).

Cyanide concentrations in the water column of mill tailings ponds were reduced at one Nevada site using naturally detoxified recycled tailings water (Henny *et al.* 1994). Lowering the cyanide concentrations in tailings ponds with hydrogen peroxide has been successful at a few mines in Nevada (Allen 1990), but this procedure is still preliminary (Clark and Hothem 1991). To reduce the potential for puddling on ore heaps, ores should be less compacted; this can be accomplished by reducing the clay content of the ores and stacking ores using conveyer belts rather than trucks (Henny *et al.* 1994). Puddling can also be reduced by careful monitoring of solution application rates and maintenance of solution distribution systems. Wildlife have been excluded from leaching solution on the heaps by substituting drip lines for sprinklers and covering the drip lines with a layer of gravel (Henny *et al.* 1994; Hill and Henry 1996). Some mines use small net panels over areas of puddling on heaps and in collection channels to exclude birds and mammals (Henny *et al.* 1994).

Water hyacinth (*Eichornia crassipes*) has been proposed as the basis of a cyanide removal technology. Hyacinths can survive for at least 72 h in a nutrient solution containing as much as 300 mg CN l^{-1} and can accumulate up to 6.7 g CN kg^{-1} DW plant material. On this basis, 1 ha of hyacinths has the potential to absorb 56.8 kg cyanide in 72 h, and this property may be useful in reducing the level of total cyanide in untreated wastewaters where concentrations generally exceed 200 mg CN l^{-1} (Low and Lee 1981). Large scale use of water hyacinths for this purpose has not yet been implemented, possibly due to disagreement over appropriate disposal mechanisms.

Free cyanide criteria currently proposed for the protection of natural resources include <3 μg l^{-1} medium for aquatic life, and <100 mg kg^{-1} diet for birds and livestock

(Eisler 1991). For human health protection, free cyanide values are <10 $\mu g\ l^{-1}$ drinking water, <50 $mg\ kg^{-1}$ diet, and <5 $mg\ m^{-3}$ air (Eisler 1991). Additional research seems needed to establish legally enforceable standards and threshold limit values for potentially toxic cyanides in various forms, including HCN and inorganic cyanide. This includes research on low-level, long-term cyanide intoxication in birds and mammals by oral and inhalation routes in the vicinities of high cyanide concentrations, especially on the incidence of nasal lesions, thyroid dysfunction, and urinary thiocyanate concentrations (Towill *et al.* 1978; Egekeze and Oehme 1980). Research is also needed on threshold limits in water where birds and mammals may be exposed, including the role of CN–metal complexes, and on sublethal effects of free cyanide on vertebrate wildlife. In aquatic systems, research is needed on: (1) long-term effects of low concentrations of cyanide on growth, survival, metabolism, and behavior of a variety of aquatic organisms (Towill *et al.* 1978; Leduc *et al.* 1982; Eisler 1991); (2) adaptive resistance to cyanide and the influence, if any, of oxygen, pH, temperature and other environmental variables (Leduc 1981, 1984); and (3) usefulness of various biochemical indicators of cyanide poisoning, such as cytochrome oxidase inhibition (Gee 1987) and vitellogenin levels in fish plasma (Ruby *et al.* 1986).

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