In accordance with the Terms of Reference, which instructed the developer to provide a comprehensive analysis of the key line inquiry, Tyhee should put more emphasis on the questions related to the key line of inquiry. To facilitate this, the information requests in part one contain short descriptions of the information gaps that the Review Board identified in the DAR.

IR Number: 1-1-2

Source:	*Mackenzie Valley Review Board*
To:	*Tyhee*
Issue:	***Cyanide Attenuation***

Background

For cyanide Tyhee appears to rely on natural attenuation through volatilization, leaching, and bacterial activity. In the DAR Tyhee identifies temperature, aeration, UV light availability and bacterial growth as factors determining the rate of attenuation. Yet it does not provide evidence that these factors will allow sufficient attenuation at the Yellowknife Gold Project's sub-arctic location, or an explanation how leaching of cyanide will mitigate environmental effects. In addition, the DAR does not contain an analysis of concentrations of cyanide breakdown compounds.

Request

1. *Please submit studies, or relevant excerpts, that support Tyhee's reliance on natural attenuation of cyanide under the conditions prevailing at the proposed mine site.*
2. *Please provide an explanation how leaching of cyanide will mitigate environmental impacts and a description of where leached cyanide will likely end up and any environmental effects associated with it.*
3. *Please provide an analysis of cyanide breakdown compounds, their toxicity, and their concentrations and distribution.*

Tyhee NWT Corp Response (Revised May 31, 2012)

Request

1. Please submit studies, or relevant excerpts, that support Tyhee's reliance on natural attenuation of cyanide under the conditions prevailing at the proposed mine site.

Tyhee does not plan to rely exclusively on natural attenuation to reduce cyanide in mine effluent water. As stated in Section 4.11.7 of the DAR, a sulfur dioxide (SO_2)-Air cyanide destruction circuit will be used to reduce cyanide concentrations to MMER required concentrations before discharge to the TCA.

Cyanide Destruction Circuit

A sulfur dioxide (SO_2)-Air cyanide destruction circuit (commonly referred to as the INCO process) will be used to reduce cyanide concentrations to MMER required concentrations before discharge to the TCA. The INCO process is based upon conversion of free and WAD cyanides to cyanate using a mixture of SO_2 and air in the presence of a soluble copper

catalyst at a controlled pH. In the INCO process, the forms of cyanide are removed by different processes. One process involves the conversion of free and WAD cyanides to cyanate. Iron complexed cyanides are reduced to the ferrous state and precipitated as insoluble copper-iron-cyanide complexes. Residual metals liberated from the WAD cyanide complexes are precipitated as their hydroxides.

The INCO process has been used at over 80 mining operations worldwide and is the process addressed in this section. A primary application of the sulfur dioxide and air process is in treatment of tailings slurries, but it is also effective for the treatment of solutions for the oxidation of free and WAD cyanides. The process has a track record of being able of reducing total cyanide in leach effluents to less than 1 mg per liter (Mudder et.al, no date). Examples include Lac Mineral's Colosseum (0.4 mg/L), Westmin's Premier Gold (< 0.2 mg/L), and Homestake Chevron's Golden Bear (0.3 mg/L).

Free and weakly complexed metal cyanides (i.e., WAD cyanides) are oxidized to cyanate by sulfur dioxide and air in the presence of a soluble copper catalyst.

$$CN^- + SO_2 + O_2 + H_2O \xrightarrow{Cu\ Catalyst} OCN^- + SO_4^{-2} + 2H^+$$

$$M(CN)_4^{-2} + 4SO_2 + 4O_2 + 4H_2O \xrightarrow{Cu\ Catalyst} 4OCN^- + 8H^+ + 4SO_4^{-2} + M^{+2}$$

The reaction is normally carried out at a pH of about 8.0 to 9.0, and due to the formation of acid in the reactions, lime is added for pH control. Decreases in process performance can occur if the pH fluctuates outside this optimal range. The optimal pH must be determined experimentally, since maximum cyanide and metals removals occur at different pH values.

Temperature has little effect on process performance between 5°C and 60°C. The SO_2 required in the reaction can be supplied either as liquid sulphur dioxide, sodium sulphite (Na_2SO_3) or as sodium metabisulphite ($Na_2S_2O_5$).

Trace metals remaining in solution following oxidation of the weakly complexed metal cyanides are precipitated as their hydroxides according to the following generalized reaction:

$$M^{+2} + 2OH^- = M(OH)_2 \text{ (solid)}$$

Metallurgical work has been conducted to determine the reagent usage in the beneficiation process for Ormsby ore. Initial testing of the cyanide destruction circuit using the INCO process has been completed.

As discussed in Attachment B, the testing program produced residual total cyanide concentrations after the destruction process ranging from <0.05 mg/L for sample DT-6 to 6.43 mg/L for DT-2. The final total CN concentrations for samples DT-3 through DT-6 were all below 1 mg/L. The final total CN concentration for DT-1, the continuous test system, was 2.88 mg/L. Optimization of the process during operation is expected to produce a supernatant with less than 1 mg/L. The leach tailings represent approximately 6 % of the total tailings material.

Natural Attenuation

Natural attenuation within the TCA is expected to further reduce TCA concentrations below those produced after the detoxed leach solution mixed with the supernatant from the flotation tailings. TCA discharge, when it occurs, will mix with meteoric external water, further reducing cyanide concentrations.

The surface effluent testing on flotation tailings (DAR Appendix J) had a pH of 7.99, indicating that the waters in contact with flotation tailings is likely to be approximately pH 8. That result is similar to that obtained after the cyanide destruction process. The process water will, to some extent, mix with local runoff waters. The average pH of Round, Winter, and Narrow Lakes were 7.43, 7.03, and 7.03, respectively based on monitoring from 2004 through 2010 for water license MV2002L2-0017. If pH in the TCA is maintained below a pH of 8, which is achievable through pH adjustment before discharge, more than 90 percent of the cyanide anion (CN^-) present will be present as hydrogen cyanide (HCN) (Logsdon et al., 1999, available online).

Examples of Cyanide Natural Attenuation

Natural attenuation of cyanide in tailings ponds is a function of the environmental conditions and has been shown to be effective. Lupin Mine and Colomac Mine, both in Canada's North, are excellent examples of natural degradation of cyanide in climates similar to Tyhee's Yellowknife Gold Project.

Lupin Mine, NWT (Logsdon et al., 1999); this mine relied solely on natural degradation of cyanide within the TCA, with CN^- entering the TCA at a concentration of 184 mg/L and being discharged from the TCA at a concentration of 0.17 mg/L. This now-closed mine, currently owned by Elgin Mining (formerly MMG, Kinross and Echo Bay Mines) is located approximately 350 kilometers (km) north of the Yellowknife Gold Project.

The Colomac Mine, NWT, mill was not equipped with a cyanide destruction circuit because the TCA was intended to be a zero discharge facility; however; due to faulting and poor construction, the facility was not zero discharge. Historic sampling over the period of 1998 through 2001 logged the degradation of cyanide, thiocyanate, and ammonia. Total cyanide concentrations in the Colomac Mine Tailings Lake decreased naturally without any intervention from 38 mg/L in September 1998 to 1 mg/L in September 2001 (Chapman et al., no date, available online). Enhanced Natural Removal was successfully used through the addition of phosphorus as a limiting nutrient to bring cyanide and its breakdown compounds (i.e., thiocyanate, ammonia) down to concentrations acceptable for release to the environment. This mine is located within 50 km west of the Yellowknife Gold Project.

Request

2. *Please provide an explanation how leaching of cyanide will mitigate environmental impacts and a description of where leached cyanide will likely end up and any environmental effects associated with it.*

As discussed in Section 1.1.1, the revised estimated of expected TCA water quality was based on the data developed during the characterization of the tailings material produced during the testing of the Ormsby ore. The primary source of the solutes in the TCA are from the supernatant from the flotation process which accounts for approximately 94% of the tailings and supernatant produced by the plant, and the detoxified supernatant from

cyanide leaching of the concentrate which account for 6 % of the liquid and solids entering the TCA.

The concentration of the a given solute in the TCA is a function of its concentration in the flotation supernatant, the detoxified leach supernatant, the amount of water reclaimed from the tailings pond, and the amount of makeup water. As no makeup water is used during the first four years of operation, the solute concentrations reach a maximum after the fourth year of operation. To show how the concentrations may evolve during the operation of the facility. Estimates of the concentration of arsenic, copper, cyanide, nickel, lead and zinc in the TCA were prepared for the end of years 1, 4, 8, and 12.

The evaluation indicated the maximum concentrations were present in the TCA in year 4. As shown in the attached report, the estimated concentration of the six parameters in the TCA at the end of year 4 are: arsenic, 59 µg/L; copper, 208 µg/L; total cyanide, 144 µg/L; nickel, 3.3 µg/L; lead, 14 µg/L; and zinc, 2.9 µg/L. These concentrations are all below the MMER guidelines but several exceed the CCME guidance levels.

Impact on Narrow Lake

The impact analysis contained in the DAR was based on the discharge of approximately 900,000 m^3 per year for the life of the project spread over the months of May through October. Approximately two-thirds of this volume was discharged in May and June. Based on the current water balance, no discharge is planned although Thyee expects to need to discharge sometime during the projects term.

As there is no discharge amount required by the water balance, a plausible discharge scenario was needed to evaluate the potential impact of Thyee's expected required future discharge on the receiving water body. The tailings discharge would need to be pumped from the TCA. The available pumps would be the reclaim pumps which have a capacity of approximately 140 m^3/hr. If the discharge lasted for 30 days a total volume of approximately 100,000 m^3 would be discharged. Any discharge would only occur between May and October. The most likely time for a discharge would be in May or June.

An evaluation of the attenuation potential of Narrow Lake for a 30 day discharge at a rate of 140 m^3/hr produced the following expected concentrations in Narrow Lake for a discharge occurring at the end of year 4: arsenic, 4.8 µg/L; copper, 17 µg/L; total cyanide, 11.5 µg/L; nickel; $<$0.5 µg/L; lead, 1.1 µg/L; zinc, $<$0.5 µg/L.

Although no routine discharge from the TCA is necessary or planned, future conditions may result in the need for a temporary, limited, discharge. Based on this analysis, the copper concentration in the TCA is the controlling parameter. With specific reference to copper concentrations within the TCA, these would be monitored during operation as part of the water license SNP and the effects of these concentrations on Narrow Lake, including confirmation of water in Narrow Lake meeting CCME guidelines, would be evaluated prior to discharge.

Request

3. *Please provide an analysis of cyanide breakdown compounds, their toxicity, and their concentrations and distribution."*

Free and Dissociable Cyanide

Free cyanide is the toxic fraction (CCREM 1987; Eisler 1991; USEPA 1985) and from a toxicological perspective the distinction between free cyanide and other forms (generally reported as total cyanide or weak acid dissociable [WAD] cyanide) is critical. Free cyanide is defined and measured as the sum of HCN and the CN^-. Total cyanide is the summation of all of the cyanide species including free cyanide, water-soluble salts (e.g., NaCN, KCN), salts of alkali, alkaline earth, or heavy metals (e.g., $Zn(CN)_2$, $Cd(CN)_2$), and less toxic complex metallocyanides (such as $Cu(CN)_2^-$ and $Fe(CN)_6^{4-}$) (Eisler 1991; Exall et al. 2011). Weak acid dissociable (WAD) cyanide is the fraction of bound cyanide that will release the free cyanide anion (CN^-) following the addition of a weak acid. WAD cyanide is often measured in the environment to account for the fraction of cyanide that may become free and toxic with relatively small changes in environmental conditions (i.e., pH). Many factors can affect the form of cyanide, including pH, temperature, salinity, the concentration of metal ions and complexation materials, dissolved oxygen, and sunlight (USEPA 1985). In addition to the various species of cyanide, there are a number of breakdown and by-products that co-occur in the aquatic environment, including cyanates (-OCN), thiocyantes (-SCN), and ammonia in addition to non-toxic forms of carbon and nitrogen.

Cyanide Breakdown Compounds

The breakdown and by-products of cyanide such as cyanates (-OCN), thiocyanates (-SCN), ferrocyanate complexes (e.g., $Fe(CN)_6^{4-}$), and ammonia (NH_3) are considerably less toxic than cyanide itself. Simple thiocyanates, such as the products of cyanide detoxification, are on the order of 12-times less toxic than cyanide (Eisler 1991) and therefore pose considerably less threat to aquatic life (Lanno et al.1996; Exall et al. 2011). The majority of risk associated with the formation of cyanates, thiocyanates, and metal-cyanide complexes is in their potential to re-release cyanide following decomposition by UV or change in pH (Eisler 1991; Calffe and Little 2003). The production of ammonia (NH_3) from the degradation of cyanide is not considered a risk to the aquatic environment at Tyhee's Yellowknife Gold Project. The CCME water quality guideline for the protection of aquatic life is 0.019 mg/L un-ionized ammonia. At 10°C, a pH of 8, and 1.0 mg/L total ammonia, the percent un-ionized ammonia would be 18.25, or a concentration of 0.018 mg/L ionized ammonia (CCME 2010). At the CWQG of 5 µg/L cyanide, negligible amounts of ammonia will be produced, far below the 1.0 mg/L needed to reach the CWQG.

Cyanide Toxicity

Cyanide(CN^-)- can be toxic as it asphyxiates cells by binding to and deactivating cytochrome c. oxidase inhibiting cellular respiration (Eisler 1991). Cyanide toxicosis is rapid following the absorption of a lethal dose, as it can enter the blood stream and cross cell membranes regardless of route of exposure. At sub-lethal concentrations, cyanide can be detoxified and excreted, and is done so by thiosulfate conjugation by specialized liver enzyme rhodenase to produce thiocyanates, which are considerably less toxic and easily excreted in the urine (Eisler 1991). Owing to efficient cyanide detoxification, it has been suggested that sub-lethal concentrations of cyanide can be tolerated in some animals over extended periods of time (Eisler 1991). In the aquatic environment, fish are considered to be the most sensitive organisms to cyanide (USEPA 1985; Sarkar 1990; Eisler 1991).

The CCME guideline of 5 micrograms per Liter (µg/L) for the protection of aquatic life is based on the U.S. Environmental Protection Agency (USEPA) criterion of 5.2 µg/L for the

protection of aquatic life (USEPA, 1985), as well as a review of the effects on aquatic organisms carried out in 1987 (CCREM 1987). For example, the lowest concentration to which rainbow trout exhibit an acute response (i.e., mortality) was 27 µg/L (Kovacs and Leduc 1982a), while a 50 percent reduction in performance of cold water fish species was observed following the continuous exposure to 10 µg/L free cyanide (Kovacs and Leduc 1982b; CCREM 1987). The USEPA criterion is based on a calculated value, whereby the Species Mean Acute Value (SMAV) for Rainbow trout (Onchorynkus mykiss) of 44.7 µg/L is divided by an acute-chronic ratio to give 8.1 µg/L; a conservative value of 5.2 µg/L is therefore effective in avoiding chronic toxicity.

A sample of the flotation tailings supernatant was used for whole effluent toxicity testing. The results indicate a 100 percent survival rate for both *Daphnia magna* and *Pimephales promelas* (fathead minnow). These data are consistent with testing conducted on the combined Ormsby and Nicholas flotation tailings which reported 100 percent survival for a 48-hour test using *Daphnia magna* and 100 percent survival for a 72-hour test using rainbow trout.

References

To assist the reader, the copies of following selected references are included in Appendix A: CCREM 1987, Calffe and Little 2003, Eisler 1991, Logsdon 1999, Mudder et.al, no date, and USEPA 1985.

Effects of Fire-Retardant Chemical Products on Fathead Minnows in Experimental Streams

Prepared by:

Edward E. Little
and
Robin Calfee

U.S. Geological Survey
Columbia Environmental Research Center
4200 New Haven Rd.
Columbia, MO 65201

Submitted to:

Cecilia Johnson
Project Leader – Fire Chemicals
Wildland Fire Chemical Systems

USDA Forest Service
Missoula Technology and Development Center
5785 West Broadway Street
Missoula, MT 59808

Final Report

CERC Ecology Branch Fire Chemical Report: ECO-04
http://www.cerc.usgs.gov/pubs/center/pdfDocs/ECO-04.PDF

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Acknowledgements

We thank the staff of the Columbia Environmental Research Center for technical consultation and reviews of this report. This research was financially supported by the Forest Service Wildland Fire Chemical Systems Program.

EXECUTIVE SUMMARY

Effects of Fire-Retardant Chemical Products to Fathead Minnows in Experimental Streams

Each year large amounts of fire-retardant chemicals are applied to wildfires across the nation. The assessment of risk posed by the use of these chemicals includes understanding the toxicity of these materials relative to exposure concentrations that are likely to result from applications, the environmental persistence of the material, and surface runoff from treated watersheds. Recent laboratory studies with long-term fire-retardant chemicals indicate a significant photo-enhanced toxicity of products containing sodium ferrocyanide corrosion inhibitors, with up to a 100-fold increase in toxicity to rainbow trout and a 10-fold increase in toxicity to southern leopard frogs in the presence of ultraviolet (UV) light. In contrast, compounds without this corrosion inhibitor were either unaffected in the presence of UV or exhibited a lesser additive toxicity than those containing the corrosion inhibitor. Toxicity data determined in laboratory studies of fire-retardant chemicals may not accurately reflect toxicity in natural habitats because a variety of environmental variables can influence persistence as well as toxicity. Without information on toxicity in natural settings it is difficult to determine the ecological hazards and probability of injury resulting from exposure following field application of the fire-retardant chemicals.

Aside from unintentional spills and overspray, stormwater runoff is a likely pathway of contamination to aquatic systems by fire-retardant chemicals. One might expect such exposures to be transient and of limited duration as the contaminated runoff is diluted and carried downstream by the water flow. In order to assess fire-chemical toxicity resulting from runoff, the fate and effects of two fire-retardant chemicals, Fire-

Trol® GTS-R (GTS-R) and Phos-Chek® D75-R (D75-R) were tested over a 24-hour period in field tests using experimental streams. (Note: The concentrations listed herein refer to milligrams of powdered concentrate mixed in one liter of water.)

This investigation produced eight major results:

GTS-R Exposure

- Under clear sky conditions 100-percent mortality of fish exposed to a concentration of 128 milligrams GTS-R per liter of water (mg GTS-R/liter) with sodium ferrocyanide occurred within 3 hours of exposure. When fish were restocked three hours later at the same concentration, 100-percent mortality again occurred within 3 hours. At the next to the highest concentration (64 mg GTS-R/liter), 59-percent mortality occurred after 6 hours. Survival among controls and the lowest treatment (32 mg GTS-R/liter) was 100 percent during these tests (Figure 1).
- The average weak-acid-dissociable (WAD) cyanide concentration after 6 hours was 153 micrograms per liter (µg/liter) in the 128 mg GTS-R/liter test concentration, and 73 µg/liter in the 64 mg GTS-R/liter test concentration. The highest non-lethal concentration for cyanide at 6 hours was 42 µg/liter in the 32 mg GTS-R/liter test concentration (Figure 2). Surviving fish recovered rapidly as GTS-R was flushed from the streams with fresh well water and no further mortality occurred.
- Although 128 mg GTS-R/liter caused 100-percent mortality under sunny conditions, no mortality occurred when the study was repeated under heavy cloud cover at similar temperatures (Figure 3). This suggests that some minimum solar UV irradiance is necessary to induce toxicity.

- In studies of GTS-R without sodium ferrocyanide, 100-percent survival occurred in the 128-mg GTS-R/liter treatment. This suggests that the toxicity of GTS-R is due to weak acid dissociable cyanide concentrations.
- Results of our study indicate that under sunny conditions, the nonlethal concentration of GTS-R containing sodium ferrocyanide falls between 32 and 64 mg GTS-R/liter. Non-lethal concentrations of GTS-R without the corrosion inhibitor were in excess of 128 mg GTS-R/liter.

D75-R Exposure

- Neither mortality nor signs of behavioral distress were observed among fish exposed to D75-R at any time, even at concentrations as high as 240 mg D75-R/liter. Thus lethal concentrations of D75-R are in excess of 240 mg/liter (Figure 4).
- Total and un-ionized ammonia approached lethal concentrations during tests with 240 mg D75-R/liter. Un-ionized ammonia would have probably exceeded this threshold if the pH of the receiving water were greater than 8.0.
- The site-specific characteristics of the watershed, including the amount of rainfall, extent of run-off, degradation of the chemical, dilution volume, and pH must be considered in determining both toxic concentrations and environmental exposure conditions for D75-R.

Figure 1. Mortality induced by GTS-R

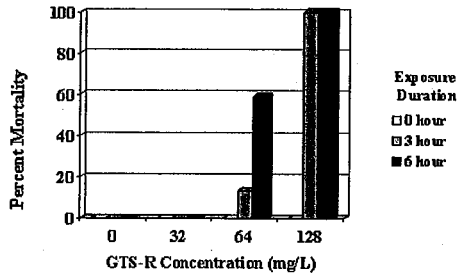

Figure 2. Observed cyanide concentrations during GTS-R Test

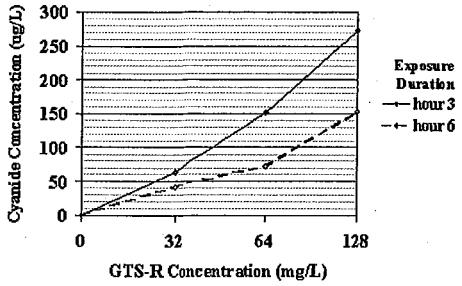

Figure 3. Toxicity of GTS-R under cloudy and sunny conditions

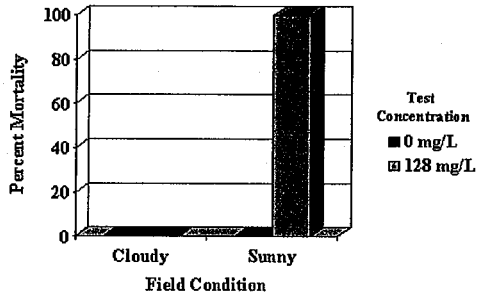

Figure 4. Mortality following exposure to chemicals with sodium ferrocyanide (SF)

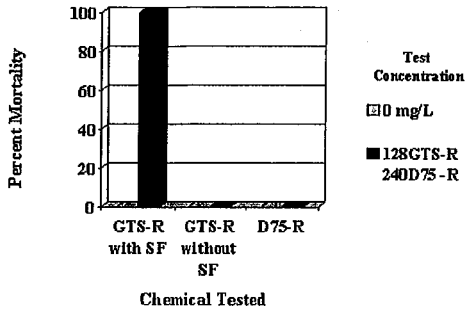

Figure 1. Percent mortality among juvenile fathead minnows exposed to GTS-R for 0 (light gray bar), 3 (dark gray bar), and 6 (black bar) hours. Each histogram represents response of three replicate exposures of 2 groups of fish.

Figure 2. Observed concentrations of weak acid dissociable cyanide (µg/liter) during GTS-R exposure. The lines indicate weak-acid-dissociable (WAD) cyanide concentrations (µg/liter) after 0, 3, and 6 hours of exposure to GTS-R.

Figure 3. Percent mortality among fathead minnows exposed to controls and 120 mg GTS-R/liter under heavy cloud cover and under sunny conditions. Each histogram represents two replicate exposures of two groups of test organisms.

Figure 4. Percent mortality among juvenile fathead minnows exposed to GTS-R with sodium ferrocyanide (w/SF), GTS-R without sodium ferrocyanide (w/o SF), and D75-R, a fire retardant that does not contain sodium ferrocyanide. Each histogram represents two replicate exposures of two groups of 10 fish for the GTS-R tests and three replicate exposures of two groups of 10 fish for the D75-R tests. Note: significant mortality occurred only in the GTS-R treatment with sodium ferrocyanide.

INTRODUCTION

An estimated 70 million liters of fire-retardant chemicals were used during the 2000 fire season to suppress wildland fires primarily in western regions of the United States (USDOI/DA 2000). These chemicals are often applied in relatively pristine and environmentally sensitive areas that are potentially inhabited by endangered, threatened, or sensitive aquatic species.

Most fire-retardant chemicals were thought to have relatively minimal toxicity, but fish kills have been documented when these substances have been accidentally released into surface waters (Dodge 1970; Minshall and Brock 1991). Fire-retardant chemicals are generally of low toxicity to aquatic organisms, however recent laboratory studies determined that the toxicity of certain fire-retardant chemicals was heightened when exposed to UV radiation or sunlight (Little and Calfee 2000). This photo-enhanced toxicity is thought to be the result of the release of free cyanide as a result of photo-activation of sodium ferrocyanide, a corrosion inhibitor used in the Fire-Trol® GTS-R formulation. The objectives of this study were to determine the toxicity of the fire-retardant chemicals Fire-Trol® GTS-R (GTS-R) and Phos-Chek® D75-R (D75-R) during exposure of fish in experimental streams.

METHODS

Test Organisms

Juvenile fathead minnows (*Pimephales promelas*) were obtained from a local fish hatchery (Genesis Randolph County Fish Hatchery, Cairo, MO) and were transferred in oxygen-saturated bags to the USGS Columbia Environmental Research Center (CERC), Columbia, Missouri. Upon arrival at CERC, fish were acclimated to 25° C and kept in

outdoor 1200-liter tanks supplied with flowing, aerated deep well water of the same quality (pH 7.0, hardness 283 mg/L as $CaCO_3$) as to be used in the stream studies. The fathead minnows were tested at approximately 30 to 60 days after yolk absorption.

Chemicals, Receipt, and Handling

Two fire-retardant chemicals, GTS-R and D75-R, were selected for testing. All fire-retardant chemicals were shipped to CERC from the U.S. Forest Service Missoula Technology and Development Center, Wildland Fire Chemical Systems (WFCS) Program (Missoula, Montana), via overnight courier in sealed 19-liter plastic containers. GTS-R formulations used during tests with and without sodium ferrocyanide were received on May 18, 1998. GTS-R and D75-R used for the other stream tests were received on September 1, 2000. Upon receipt, the shipping container was inspected for damage and the security seals were inspected for evidence of tampering. The chemicals were stored in their shipping containers at room temperature according to manufacturers' recommendations in a secured locker at CERC. All concentrations, dilutions, and mixtures are based on the dry concentrate unless otherwise specified. The mixed retardant is prepared by mixing the qualified weight of GTS-R (1.66 pounds) or D75-R (1.20 pounds) with one expressed gallon of water. The mixed retardant is applied from aircraft or ground engines in support of wildland fire activities. The number of gallons of mixed retardant applied to 100 square feet is referred to as GPC. Field applications may range from 1 to 8 GPC.

Experimental Design and Test Conditions

Fire retardant exposure concentrations were selected on the basis of 96-hour LC50 concentrations determined in laboratory studies (Little and Calfee, 2000). The 96-hour LC50 is the concentration lethal to 50 percent of test organisms with 96 hours of exposure. The high concentration of fire-retardants used in our study was approximately 10 times the LC50 concentration for GTS-R under UV exposure (13.6 mg GTS-R/liter) and 2 times the LC50 concentration for D75-R under UV exposure (108 mg D75-R/liter) determined in laboratory tests (Little and Calfee, 2002a). A transient exposure to fire-retardant chemicals in a flowing stream was anticipated as a pulse of stormwater runoff from a fire-retardant-treated watershed. The studies were conducted in experimental stream facilities at CERC as described by Fairchild et al. (1993).

This study followed an incomplete randomized balanced block design (Cochran and Cox, 1957) that paired each of four concentrations of a fire-retardant chemical with each other and provided three replicate tests per concentration. Test sequence and experimental design for the stream study are shown in Table 1. The streams were 50 meters long. Each stream consisted of a V-weir headbox, and a sequence of three riffles each 10-meters long, 1-meter wide, and 10-cm deep, separated by pools that were 10-meters long, 2-meters wide and 30-cm deep. The streams were lined by an inert layer of Hypalon® covered with a 10-cm layer of 1 to 4-cm diameter natural stream gravel. Well water was biologically colonized by passage through a series of earthen holding ponds. Flow rates of each stream were matched and maintained at 5-liters-per-second with the calibrated V-notch weir. Estimated 50 percent volumetric exchange time during rinsing was approximately 30 minutes. The water volumes were 8702 liters for stream 1 and

6671 liters for stream 2. Experimental stream systems used in this study simulate the pool and riffle habitats of first order streams, and had well-established natural communities of periphyton, macrophytes, aquatic invertebrates, mussels, crayfish, and bluegill sunfish (Figure 5).

At least three fire-retardant concentrations and a control were used. These treatments bracketed a range of environmental concentrations anticipated to occur in the field. Each stream was treated with a randomly selected fire-retardant concentration. Juvenile fathead minnows were confined in groups of 10 in three 14-liter containers. The open sides of the test containers were covered with 1.0-mm mesh netting to allow stream water to flow through them. The containers were either covered with 60-percent shade cloth (high UV treatment) or polypropylene filters (low UV treatment) to provide a natural solar exposure within the range of tolerance for the fathead minnow. Solar radiation was measured using an Optronics-754® spectroradiometer with a submersible sphere (Optronic Laboratories, Orlando FL).

Predetermined amounts of dry concentrate (based on stream volumes) were dissolved in 15 liters of well water, and this mixture was released over the length of the stream and allowed to recirculate through the stream for at least 60 minutes. Fish were then added to test containers in the upstream and downstream pool areas. Exposure of fish to the recirculated water continued for 6 hours, then the test waters were flushed from the streams with fresh well water for 10 to 20 hours. During the recirculating exposure time, the fish were observed at selected intervals (3 and 6-hours post dosing) for mortality and behavioral distress. If the exposure was lethal after 3 hours, then dead fish were removed and the exposure container was restocked. The fish were also observed at

1, 3, and 18 to 20 hours of the rinse period to determine if there was delayed toxicity. The pH, temperature, and dissolved oxygen (DO) of the stream water were measured at the onset of the exposure, at the midpoint of the exposure, at the end of the exposure, and at the conclusion of the rinse period. Water samples were also taken at these times for analysis of ammonia and weak-acid-dissociable (WAD) cyanide. The GTS-R tests were conducted from September 14 to 22, 2000 and the D75-R tests were conducted from September 26 to October 12, 2000.

All tests were conducted under clear-sky conditions. Additional tests were conducted on September 20 and October 4, 2000 with GTS-R under fully overcast cloudy conditions to assess the effects of decreased solar irradiance levels on the toxicity of this chemical. UV filters were used during these tests. Limited testing was also conducted with GTS-R with and without sodium ferrocyanide in the formulation on October 6 and October 10, 2000.

UV Measurements

Solar radiation was measured at the water surface and underwater (approximately 10 cm) in the exposure container (with the shade cloth and polypropylene filters) to determine the intensity of UV radiation. Measurements were taken at 2:00 pm (Central Standard Time) under clear sky conditions and under heavy cloud cover (eg., solar disc not visible). A scanning spectroradiometer was used to measure intensity by wavelength at 1 nanometer (nm) intervals over the UVB (290 to 320 nm), UVA (320 to 400 nm), and visible (400 to 700 nm) wavebands. The radiometer was calibrated with a National Institute of Standards and Technology-traceable tungsten lamp and wavelength accuracy

and intensity were checked during the UV measurements. The UV values are reported as cumulative microWatts per square centimeter ($\mu W/cm^2$).

Chemical Analysis

Ammonia was measured as total ammonia (NH_4-N) with a Technicon II Autoanalyzer®, and recorded using the New Analyzer Program® version 2.5 (Labtronics, Guelph, Canada, 1998). The ammonia samples were analyzed using Technicon Industrial Method #329-74W (Technicon Instruments Corporation, Tarrytown, NY). Un-ionized ammonia (NH_3) concentrations in each treatment were calculated using the ammonia equilibrium equation described by Emerson et al. (1975). Nitrate samples were analyzed using standard method 4500-NO_3-F Automated Cadmium Reduction method (APHA, 1989).

Water samples were taken to analyze for WAD cyanide and placed in 250-ml polypropylene bottles and preserved with NaOH. The samples were shipped at 4° C via overnight courier to Severn Trent Laboratories (Arvada, CO) and analyzed using standard colorimetric method 4500-CN-I (APHA, 1989). WAD cyanide is the free ionic form of cyanide that is most likely to be released by the photo-activation of sodium ferrocyanide by UV.

Statistical Analysis

Mortality data were arcsine and square root transformed, then analyzed by Analysis of Variance (ANOVA) to determine if toxicity resulted from chemical

concentration, UV treatment, duration of exposure, or their interactions. A probability level of 0.05 was selected.

RESULTS

The 64 and 128 mg GTS-R /liter exposure concentrations were lethal to the fathead minnows. [Note: all fire-retardant concentrations reported herein refer to dry concentrate per liter of water.] Loss of equilibrium in fish was detected in both UV treatments within an hour of exposure. At 128 mg GTS-R/liter, 100-percent mortality occurred by hour 3 of exposure under clear sky conditions (Table 2). When fish were restocked at hour 3 in streams receiving the 128 mg GTS-R/liter treatments they also died within 1 hour of exposure. No mortality occurred among fish exposed to the 32 mg GTS-R/liter treatment. The average measured WAD cyanide was 273 µg/liter at hour 3 and 153 µg/liter at hour 6 in the 128 mg GTS-R/liter treatments. The average WAD cyanide concentration for the 64 mg GTS-R/liter treatments was 153 µg/liter at hour 3 and 73 µg/liter at hour 6 (Table 2). The WAD cyanide measured in the 32 mg GTS-R/liter treatment was 63 µg/liter at hour 3 and 42 µg/liter at hour 6. Survivors showing loss of equilibrium recovered rapidly during the freshwater flushing period and no further mortality occurred.

When exposures with 128 mg GTS-R/liter were conducted in replicated studies, resident juvenile bluegill showed signs of distress, including jumping out of the streams. Resident crayfish climbed out of the water during the application of 128 mg GTS-R/liter.

When exposures with 128 mg GTS-R/liter were conducted in replicated studies under heavy clouds, no mortality occurred (Table 3). Thus, a minimum solar UV

irradiance was necessary to induce toxicity. The concentration of WAD cyanide in the High UV treatment was 130 µg/liter under sunny conditions, but below detection limits under cloudy conditions.

When replicated studies were conducted with GTS-R formulations with and without sodium ferrocyanide under sunny sky conditions, there was 100-percent survival among fish exposed to the formulation without sodium ferrocyanide, and 100-percent mortality among fish exposed to the formulation with sodium ferrocyanide (Table 4).

Neither mortality nor signs of behavioral distress, such as a loss of equilibrium were observed among fish exposed to D75-R, including concentrations as high as 240 mg D75-R/liter (Table 5).

Cyanide was not detected in the control treatments, in treatments of the GTS-R formulations without sodium ferrocyanide (Table 4), or the D75-R treatments (Table 5). Under sunny conditions, UVB levels of 1.6 and 6.9 $\mu W/cm^2$ (65 and 129 $\mu W/cm^2$ UVA) were recorded in the low and high UV treatments, relative to a surface irradiance of 143 $\mu W/cm^2$ (3233 $\mu W/cm^2$ UVA). Under overcast conditions UVB irradiance was 0.1 and 3.5 $\mu W/cm^2$ (3 and 95 $\mu W/cm^2$ UVA) relative to a surface UVB irradiance of 39.7 $\mu W/cm^2$ (948 $\mu W/cm^2$ UVA).

Total ammonia and un-ionized ammonia concentrations observed during the GTS-R experiments remained within the sublethal range for all concentrations tested (Table 2). Temperature ranged from 19.2 to 25.5° C, and pH ranged from 7.4 to 7.7 during the GTS-R tests (Table 1). Total ammonia and un-ionized ammonia concentrations approached lethal concentrations at the highest concentration of D75-R (Table 5). Temperature ranged from 16.3 to 19° C and pH ranged from 7.6 to 7.8 during the D75-R tests (Table

1). The temperature and pH values were within the range of tolerance for the fathead minnow.

DISCUSSION

Information about environmental concentrations of fire-retardant chemicals is mainly limited to recommended application rates (1 to 8 GPC). Actual applications may exceed recommended amounts depending on the judgment of the Incident Commander or, in the case of prescribed burns, the Fire Management Officer. Aqueous concentrations of GTS-R and D75-R applied in our study (32 to 128 mg GTS-R/liter, or 60 to 240 mg D75-R/liter) were considerably below the minimum concentration of mixed retardants that are typically applied during aerial application (1 GPC). The high concentration of fire-retardants used in our study was approximately 10 times the LC50 concentration of GTS-R to fathead minnows under UV exposure (13.6 mg GTS-R/liter) and 2 times the LC50 concentration for D75-R under UV exposure (108 mg D75-R/liter) determined in laboratory tests (Little and Calfee, 2002a). For GTS-R, this concentration was lethal to all test organisms within 3 hours of exposure under high solar irradiance conditions and was also lethal to organisms restocked after three hours. The observed toxicity of the 64-mg GTS-R/liter treatment was consistent with results from a 96-hour pond enclosure study in which LC50 values ranged from 21.1 to 70.8 mg GTS-R/liter under various UV and sediment treatment conditions (Little and Calfee, 2002b).

Exposures in flowing streams will vary with the rate of flow as the fire-retardant is diluted and removed from the stream segment. Fish exhibiting loss of equilibrium during initial exposure to lethal GTS-R treatments rapidly recovered when placed in

uncontaminated water; thus, recovery occurs when duration of exposure is limited. The 3- and 6-hour exposures in the present study likely reflect worst-case scenarios. In test exposures of the Little Humbolt River (Nevada), Poulton (1997) found that Phos-Chek D75-F, a formulation nearly identical to D75-R, persisted at lethal concentrations for about 45 to 60 minutes.

There are several potentially toxic components in the GTS-R formulation, including total ammonia, un-ionized ammonia, and cyanide. Total ammonia and un-ionized ammonia remained well below lethal concentrations. In contrast, cyanide concentrations (> 73 to 273 µg/liter) measured in the 64- and 128-mg GTS-R/liter treatments were lethal at 3 and 6 hours of exposure. The observed cyanide concentrations are comparable with 96-hour LC50 values of 50 µg/liter WAD cyanide (in 13.6 mg GTS-R/liter) determined for juvenile fathead minnows (Little and Calfee, 2002a).

The role of sodium ferrocyanide in the toxicity of the GTS-R formulation is clearly indicated by the total lack of mortality observed during tests of the formulation that did not contain sodium ferrocyanide. The importance of sunlight in the photoactivation of sodium ferrocyanide is also suggested by the lack of mortality during tests of the full formulation under the UV–limited conditions of heavy cloud cover. The formation of free cyanide from sodium ferrocyanide by ultraviolet radiation is a well-known reaction (Burdick and Lipschuetz, 1950). This photo-transformation takes place in the water column and is a function of UV irradiance levels. In our study, stream irradiance conditions under cloudy conditions of 40 $\mu W/cm^2$ UVB and 948 $\mu W/cm^2$ UVA were likely insufficient for photo-transformation, compared to irradiance levels of 143 $\mu W/cm^2$

UVB and 3233 $\mu W/cm^2$ UVA under sunny conditions. Photo-transformation likely took place throughout the stream, but not in the exposure chambers where UV was limited by filters.

A number of factors will influence photo-enhanced toxicity in natural habitats. Solar angle associated with time of day, season, air pollution, clouds, and surface reflection will influence UV irradiance levels (Little and Fabacher, 1996). Water quality (especially humic acid concentration) will limit the amount of UV penetrating the water column (Scully and Lean, 1994) and may also influence the bioavailability of chemical substances to an organism by binding them (Oris et al., 1990).

Mortality observed during the stream exposure was consistent with previous studies. In pond enclosure studies (Little and Calfee, 2002b), the effects of ultraviolet radiation were clearly apparent as heightened mortality occurred over a concentration range of 25 to 128 mg GTS-R/liter under the high irradiance condition, and by the rapid depletion of cyanide after 24 hours. In contrast, under low UV irradiance conditions only the highest treatment was lethal within 24 hours and cyanide was persistent for at least 4 days (Little and Calfee, 2002b). In laboratory studies of GTS-R, free cyanide concentrations in water ranged from below detections limits under dark control (foil-wrapped chambers) conditions up to 22 µg/liter under UV irradiance conditions (Little and Calfee, 2000). A rapid onset of mortality within 1 to 2 hours of the onset of exposure was also observed during the laboratory tests.

Preliminary results of weathering studies under ambient summer conditions indicate that GTS-R may persist in the environment for at least 21 days and possibly longer (Little and Calfee, 2002a). Runoff from treated watersheds could remain toxic

well after the application of the chemical. Similarly, toxic levels of D75-R remained constant after application on soils (Little and Calfee, 2002a). With both retardant chemicals, soil composition will likely be an important variable in persistence and long term hazards. Poulton (1997) found that chemical half-life of GTS-R and D75-F decreased as clay content of soils increased, with the most rapid degradation occurring in silty clay loam having a 7.5 percent organic content.

D75-R was essentially nontoxic during the stream exposure and toxicity of this retardant was not increased in the presence of sunlight. In the pond enclosure studies mortality was not consistently induced during a 4-day exposure to D75-R (Little and Calfee, 2002b). D75-R does not contain the ferrocyanide corrosion inhibitor in its formulation and photo-enhanced toxicity was not apparent. Mortality in the pond enclosure study was largely associated with heightened concentrations of un-ionized ammonia, particularly as pH increased with algal growth. During the stream tests un-ionized ammonia approached lethal levels in the 240-mg D75-R/liter treatment, but circumneutral pH levels limited un-ionized ammonia to a sublethal range. The pH of receiving waters is likely to be critical to the toxicity of D75-R.

CONCLUSIONS

The GTS-R formulation containing sodium ferrocyanide was toxic under sunny conditions. Toxicity occurred at concentrations of GTS-R similar to those concentrations that were toxic during pond enclosure and laboratory tests. Whether such applications constitute environmental harm depends upon the characteristics of the watershed, particularly the watershed to surface water ratio, as well as the rate of GTS-R

decomposition. Rainwater runoff following applications of this formulation at the recommended rate could result in lethal concentrations in small ponds and in streams receiving limited flow. Larger aquatic systems would likely dilute the formulation to sublethal levels but could have lethal effects at source points. Preliminary results of weathering studies under ambient summer conditions indicate that GTS-R may persist in the environment for at least 45 days, and possibly longer; therefore, delayed effects could occur well after the application of the chemical.

Toxicity of GTS-R and D75-R from un-ionized ammonia may be of concern at the highest concentrations tested if pH is elevated (greater than pH 8) and buffering capacity is limited in the receiving waters.

ENVIRONMENTAL IMPLICATIONS

If 5 cm of rain fell on the area treated with the minimum recommended application rate of 1 GPC, 755 grams of GTS-R would be contained in 474 liters of water. This would result in a dilution of the retardant to a final concentration of 1.6 grams GTS-R/liter of water, an amount 47 times greater than the sublethal concentration of 34 mg GTS-R/liter observed during the stream tests. The rapid recovery of fish when removed from exposure indicates that the duration of exposure and hence the residence time of the chemical in the habitat is a critical variable. The 3- to 6-hour exposures conducted in this study probably reflect worse case situations. Depending on the flow rate of the stream, the duration of exposure may be much shorter than the 3- to 6-hour exposures of the present study, and probably not sufficient to cause harm.

If 5 cm of rain fell on the area treated with the minimum recommended application rate of 1 GPC, 554 grams of D75-R would be contained in 474 liters of water. The resulting concentration would be about 1.2-grams D75-R/ liter, or 10 times the highest D75-R concentration used in this study. Since this concentration did not exhibit toxicity until 14 days of exposure, it is likely that D75-R would be washed downstream before injury occurred.

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Table 1. Test sequence for Fire-Trol® GTS-R and Phos-Chek® D75-R (date of test in parentheses), concentration, average temperature (standard deviation in parentheses), and pH for each test trial.

	GTS-R TESTS					
	Stream 1			**Stream 2**		
	Concentration	**Temp**	**pH**	**Concentration**	**Temp**	**PH**
Test 1 (9/13/00)	64 mg/L	25.0 (4.9)	na	0 mg/L	25.5 (3.1)	na
Test 2 (9/15/00)	32 mg/L	23.5 (3.3)	na	64 mg/L	23.5 (3.3)	na
Test 3 (9/18/00)	128 mg/L	24.0 (4.7)	7.6	64 mg/L	24.0 (4.7)	7.5
Test 4 (9/19/00)	32 mg/L	22.3 (6.0)	7.5	128 mg/L	22.0 (5.9)	7.6
Test 5 (9/21/00)	128 mg/L	19.8 (3.4)	7.4	0 mg/L	19.3 (3.8)	7.7
Test 6 (9/22/00)	0 mg/L	23.0 (3.8)	7.5	32 mg/L	23.0 (3.8)	7.6
	D75-R TESTS					
	Stream 1			**Stream 2**		
	Concentration	**Temp**	**PH**	**Concentration**	**Temp**	**PH**
Test 1 (9/26/00)	240 mg/L	18.8 (3.9)	7.8	120 mg/L	19.0 (4.2)	7.6
Test 2 (9/27/00)	240 mg/L	18.8 (2.1)	7.7	60 mg/L	18.8 (2.1)	7.8
Test 3 (10/02/00)	120 mg/L	16.3 (2.9)	Na	60 mg/L	16.3 (2.90	na
Test 4 (10/03/00)	0 mg/L	16.5 (3.0)	Na	60 mg/L	16.5 (3.0)	na
Test 5 (10/11/00)	240 mg/L	17.5 (1.7)	7.8	0 mg/L	16.3 (2.1)	7.7
Test 6 (10/12/00)	0 mg/L	16.3 (2.9)	7.6	120 mg/L	16.3 (2.9)	7.6

Table 2. Total ammonia concentration, with un-ionized ammonia concentration in parentheses, and WAD cyanide concentration (mean ± standard deviation), and percent mortality of fathead minnows, after 3 and 6 hours of exposure to Fire-Trol GTS-R® with and without YPS in experimental streams (N = 3 tests per treatment; 120 fish total).

GTS-R Concentration (mg/L)	**Total Ammonia Concentration (mg/L)**		**Un-ionized Ammonia Concentration (mg/L)**		**WAD Cyanide Concentration (µg/L)**		**Percent Mortality**	
	Hour 3	**Hour 6**	**Hour 3**	**Hour 6**	**Hour 3**	**Hour 6**	**Hour 3**	**Hour 6**
0	0.1 ± 0.06	0.1 ± 0.06	0	0	ND	ND	0	0
32	1.7 ± 0.26	1.0 ± 0.23	0	0	63 ± 16.9	42 ± 26.7	0	0
64	5.7 ± 0.31	4.6 ± 0.45	0.01 ± 0.001	0.01 ± 0.001	153 ± 25.0	73 ± 30.3	13.3 ± 1.3	59 ± 2.4
128	10.5 ± 0.78	10.5 ± 0.78	0.02 ± 0.008	0.02 ± 0.008	273 ± 20.8	153 ± 20.8	100	100

Table 3. Average surface UV irradiance ± standard deviation, and precent fathead minnow mortality after exposure to Fire-Trol® GTS-R under clear and overcast-sky solar irradiance levels. (N = 2 tests per treatment; 80 fish total).

Sky Condition		**Irradiance Intensity (μW/cm)**		**% Mortality**	
		UVB	UVA	GTS-R Concentration	
	Surface	3.79 ± 5.0	948 ± 95	0 mg/L	128 mg/L
Cloudy	High UV	3.5 ± 0.4	95 ± 5	0	0
	Low UV	0.1 ± 0.02	3 ± 0.3	0	0
Sunny	Surface	143.0 ± 4	3233 ± 200		
	High UV	6.9 ± 0.2	129 ± 1.0	0	100
	Low UV	1.6 ± 0.1	65 ± 5.3	0	100

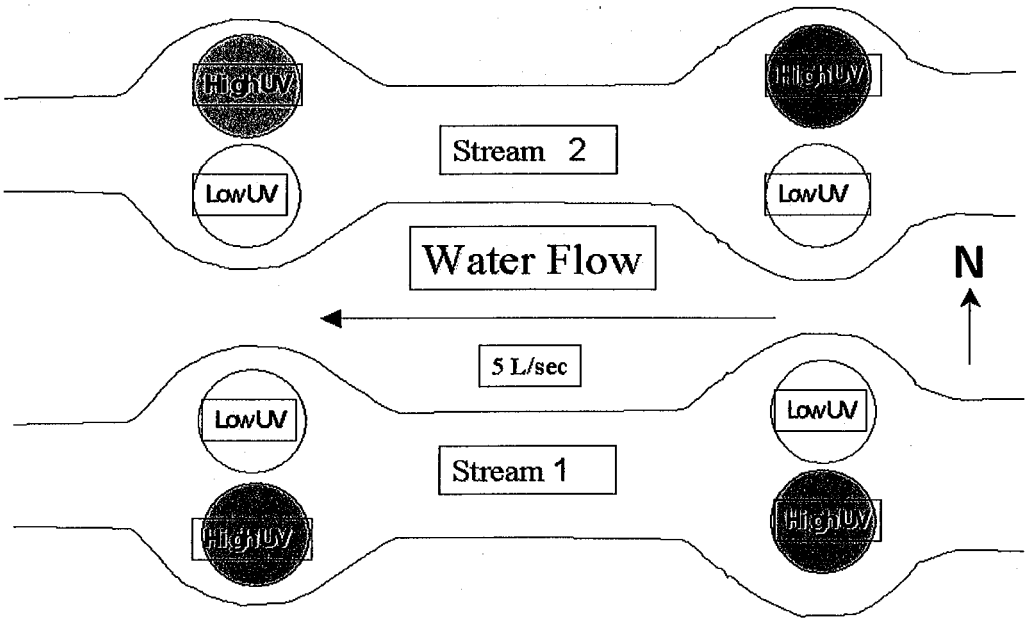
Table 4. Percent fathead minnow mortality and WAD cyanide concentrations (µg/L) observed (standard deviation in parentheses) after 6 hours exposure to 128 mg/L Fire-Trol® GTS-R with and without sodium ferrocyanide in experimental streams. Indicates not detected. (N = 2 tests per treatment; 80 fish total).

Treatment	% Mortality	Cyanide Concentration (µg/L)
GTS-R with sodium ferrocyanide	100	210 ± 57
GTS-R without sodium ferrocyanide	0	ND

Table 5. Total ammonia and un-ionized ammonia concentration (mean ± standard deviation), WAD cyanide concentration and percent mortality of fathead minnows (mean ± standard deviation) after 3 and 6 hours of exposure to Phos-Chek® D75-R in experimental streams (N = 3 tests per treatment; 120 fish total). ND indicates that concentration was below detection limits.

D75-R Concentration (mg/L)	Total Ammonia Concentration (mg/L)		Un-ionized Ammonia Concentration (mg/L)		WAD Cyanide Concentration (µg/L)		Percent Mortality	
	Hour 3	Hour 3	Hour 3	Hour 6	Hour 3	Hour 6	Hour 3	Hour 6
0	0.1 ± 0.01	0.1 ± 0.01	ND	ND	ND	ND	0	0
60	0.1 ± 0.07	0.2 ± 0.05	ND	ND	ND	ND	0	0
120	11.0 ± 0.07	9.4 ± 0.11	0.02 ± 0.004	0.02 ± 0.001	ND	ND	0	0
240	19.9 ± 0.26	17.7 ± 0.44	0.04 ± 0.009	0.04 ± 0.008	ND	ND	0	0

Figure 5. Diagram of experimental streams showing placement of exposure chambers and UV treatment conditions used during tests with GTS-R and D75-R.



Canadian Water Quality Guidelines for the Protection of Aquatic Life

SUMMARY TABLE

Summary of Canadian water quality guidelines for the protection of aquatic life.

Parameter[a]	Freshwater		Marine	
	Concentration (µg·L^{-1})	Date[b]	Concentration (µg·L^{-1})	Date[b]
Acenaphthene [See Polycyclic aromatic hydrocarbons (PAHs)]				
Acridine [See Polycyclic aromatic hydrocarbons (PAHs)]				
Aldicarb	1[c]	1993	0.15[c]	1993
Aldrin + Dieldrin[d]	~~0.004~~ [e, f]	1987		
Aluminum[d]	5–100[g]	1987		
Ammonia (total)[d]	1370–2200[h]	1987		
Aniline	2.2[i]	1993	Insufficient data	1993
Anthracene [See Polycyclic aromatic hydrocarbons (PAHs)]				
Arsenic[j]	5.0[k]	1997	12.5[c]	1997
Atrazine	1.8[i]	1989		
Benz*(a)*anthracene [See Polycyclic aromatic hydrocarbons (PAHs)]				
Benzene[j]	370[c, k]	1999	110[c]	1999
Benzo*(a)*pyrene [See Polycyclic aromatic hydrocarbons (PAHs)]				
2,2-Bis(*p*-chlorophenyl)-1,1,1-trichloroethane [See DDT (total)]				
Bromacil	5.0[c, i]	1997	Insufficient data	1997
Bromoform [See Halogenated methanes, Tribromomethane]				
Bromoxynil	5.0[i]	1993	Insufficient data	1993
Cadmium	0.017[c, l]	1996	0.12[i]	1996
Captan	1.3[c]	1991		
Carbaryl	0.20[i]	1997	0.32[c, i]	1997
Carbofuran	1.8[i]	1989		
Carbon tetrachloride [See Halogenated methanes, Tetrachloromethane]				
Chlordane[d]	~~0.006~~ [e, f]	1987		
Chlorinated benzenes				
Monochlorobenzene	1.3[c, k]	1997	25[c, k]	1997
1,2-Dichlorobenzene	0.70[c, k]	1997	42[c, k]	1997
1,3-Dichlorobenzene	150[c, k]	1997	Insufficient data[k]	1997
1,4-Dichlorobenzene	26[c, k]	1997	Insufficient data[k]	1997
1,2,3-Trichlorobenzene	8.0[c, k]	1997	Insufficient data[k]	1997

Continued.

Continued.

Parameter[a]	Freshwater		Marine	
	Concentration (µg·L^{-1})	Date[b]	Concentration (µg·L^{-1})	Date[b]
Chlorinated benzenes—Continued				
1,2,4-Trichlorobenzene	24[c, k]	1997	5.4[c, k]	1997
1,3,5-Trichlorobenzene[d]	Insufficient data[k]	1997	Insufficient data[k]	1997
1,2,3,4-Tetrachlorobenzene	1.8[c, k]	1997	Insufficient data[k]	1997
1,2,3,5-Tetrachlorobenzene[d]	Insufficient data[k]	1997	Insufficient data[k]	1997
1,2,4,5-Tetrachlorobenzene[d]	Insufficient data[k]	1997	Insufficient data	1997
Pentachlorobenzene	6.0[c, k]	1997	Insufficient data	1997
Hexachlorobenzene[d]	Insufficient data[e, f, k]	1997	Insufficient data	1997
Chlorinated ethanes				
1,2-Dichloroethane	100[c, i]	1991	Insufficient data	1991
1,1,1-Trichloroethane	Insufficient data	1991	Insufficient data	1991
1,1,2,2-Tetrachloroethane	Insufficient data	1991	Insufficient data	1991
Chlorinated ethenes				
1,1,2-Trichloroethene (Tichloroethylene; TCE)	21[c, i]	1991	Insufficient data	1991
1,1,2,2-Tetrachloroethene (Tetrachloroethylene; PCE)	111[c, i]	1993	Insufficient data	1993
Chlorinated methanes [See Halogenated methanes]				
Chlorinated phenols[d]				
Monochlorophenols	7	1987		
Dichlorophenols	0.2	1987		
Trichlorophenols	18	1987		
Tetrachlorophenols	1	1987		
Pentachlorophenol (PCP)	0.5	1987		
Chlorine, reactive [See Reactive chlorine species]				
Chloroform [See Halogenated methanes, Trichloromethane]				
4-Chloro-2-methyl phenoxy acetic acid [See MCPA]				
Chlorothalonil	0.18[c]	1994	0.36[c]	1994
Chlorpyrifos	0.0035	1997	0.002[c]	1997
Chromium				
Trivalent chromium (Cr(III))	8.9[c, k]	1997	56[c, k]	1997
Hexavalent chromium (Cr(VI))	1.0[k]	1997	1.5[k]	1997
Chrysene [See Polycyclic aromatic hydrocarbons (PAHs)]				
Colour	Narrative	1999	Narrative	1999
Copper[d]	2–4[m]	1987		
Cyanazine	2.0[c, i]	1990		
Cyanide[d]	5 (as free CN)	1987		

Continued.

Continued.

Parameter[a]	Freshwater		Marine	
	Concentration (µg·L^{-1})	Date[b]	Concentration (µg·L^{-1})	Date[b]
DDAC (Didecyl dimethyl ammonium chloride)	1.5	1999		
DDT (total)[d] (2,2-Bis(*p*-chlorophenyl)-1,1,1-trichloroethane; dichloro diphenyl trichloroethane)	~~0.001~~ [e, f]	1987		
Debris (litter/settleable matter)			Narrative[c]	1996
Deltamethrin	0.0004	1997	Insufficient data	1997
Deposited bedload sediment [See Total particulate matter]				
Dibromochloromethane [See Halogenated methanes]				
Dicamba	10[c, i]	1993		
Dichlorobenzene [See Chlorinated benzenes]				
Dichlorobromomethane [See Halogenated methanes]				
Dichloro diphenyl trichloroethane [See DDT (total)]				
Dichloroethane [See Chlorinated ethanes]				
Dichloroethylene [See Chlorinated ethanes, 1,2-Dichloroethane]				
Dichloromethane [See Halogenated methanes]				
Dichlorophenols [See Chlorinated phenols]				
1,3-Dichlorophenoxyacetic acid [see Phenoxy herticides]				
Diclofop-methyl	6.1	1993		
Didecyl dimethyl ammonium chloride [See DDAC]				
Diethylene glycol [See Glycols]				
Di(2-ethylhexyl) phthalate [See Phthalate esters]				
Dimethoate	6.2[c]	1993	Insufficient data	1993
Di-*n*-butyl phthalate [See Phthalate esters]				
Di-*n*-octyl phthalate [See Phthalate esters]				
Dinoseb	0.05	1992		
Dissolved gas supersaturation	Narrative	1999	Narrative	1999
Dissolved oxygen	5500–9500[k, n]	1999	>8000 & narrative [c, k]	1996
Endosulfan[d]	0.02	1987		
Endrin[d]	~~0.0023~~ [f, i]	1987		
Ethylbenzene[j]	90[c, k]	1996	25[c, k]	1996
Ethylene glycol [See Glycols]				
Fluoranthene [See Polycyclic aromatic hydrocarbons (PAHs)]				
Fluorene [See Polycyclic aromatic hydrocarbons (PAHs)]				

Continued.

Continued.

Parameter[a]	Freshwater		Marine	
	Concentration ($\mu g \cdot L^{-1}$)	Date[b]	Concentration ($\mu g \cdot L^{-1}$)	Date[b]
Glycols				
Ethylene glycol	192 000[k]	1997	Insufficient data	1997
Diethylene glycol	Insufficient data[k]	1997	Insufficient data	1997
Propylene glycol	500 000[k]	1997	Insufficient data	1997
Glyphosate	65[c]	1989		
Halogenated methanes				
Monochloromethane (Methyl chloride)[d]	Insufficient data	1992	Insufficient data	1992
Dichloromethane (Methylene chloride)	98.1[c, i]	1992	Insufficient data	1992
Trichloromethane (Chloroform)	1.8[c, i]	1992	Insufficient data	1992
Tetrachloromethane (Carbon tetrachloride)	13.3[c, i]	1992	Insufficient data	1992
Monobromomethane (Methyl bromide)[d]	Insufficient data	1992	Insufficient data	1992
Tribromomethane (Bromoform)[d]	Insufficient data	1992	Insufficient data	1992
Dibromochloromethane[d]	Insufficient data	1992	Insufficient data	1992
Dichlorobromomethane[d]	Insufficient data	1992	Insufficient data	1992
HCBD [See Hexachlorobutadiene (HCBD)]				
Heptachlor (Heptochlor epoxide)[d]	~~0.01~~ [e,f]	1987		
Hexachlorobenzene [See Chlorinated benzenes]				
Hexachlorobutadiene (HCBD)	1.3[c, k]	1999		
Hexachlorocyclohexane (Lindane)[d]	0.01	1987		
Hypochlorous acid [See Reactive chlorine species]				
3-Iodo-2-propynyl butyl carbamate [See IPBC]				
IPBC (3-Iodo-2-propynyl butyl carbamate)	1.9	1999		
Iron[d]	300	1987		
Lead[d]	1–7[o]	1987		
Lindane [See Hexachlorocyclohexane]				
Linuron	7.0[c]	1995	Insufficient data	1995
MCPA (4-Chloro-2-methyl phenoxy acetic acid; 2-methyl-4-chloro phenoxy acetic acid)	2.6[c]	1995	4.2[c]	1995
Mercury[d]	0.1	1987		
Methyl bromide [See Halogenated methanes, Monobromomethane]				
Methyl chloride [See Halogenated methanes, Monochloromethane]				
2-Methyl-4-chloro phenoxy acetic acid [See MCPA]				
Methylene chloride [See Halogenated methanes, Dichloromethane]				
Metolachlor	7.8[c]	1991		
Metribuzin	1.0[c]	1990		
Molybdenum[j]	73[c]	1999		
Monobromomethane [See Halogenated methanes]				

Continued.

Continued.

Parameter[a]	Freshwater		Marine	
	Concentration ($\mu g \cdot L^{-1}$)	Date[b]	Concentration ($\mu g \cdot L^{-1}$)	Date[b]
Monochloramine [See Reactive chlorine species]				
Monochlorobenzene [See Chlorinated benzenes]				
Monochloromethane [See Halogenated methanes]				
Monochlorophenols [See Chlorinated phenols]				
Naphthalene [See Polycyclic aromatic hydrocarbons (PAHs)]				
Nickel[d]	25–150[p]	1987		
Nitrate[d]	Concentrations that stimulate weed growth should be avoided.	1987		
Nitrite[d]	60	1987		
Organotins				
Tributyltin	0.008[c]	1992	0.001	1992
Tricyclohexyltin	Insufficient data	1992	Insufficient data	1992
Triphenyltin	0.022[c, i]	1992	Insufficient data	1992
Oxygen, dissolved [See Dissolved oxygen]				
PAHs [See Polycyclic aromatic hydrocarbons (PAHs)]				
PCBs [See Polychlorinated biphenyls (PCBs)(total)]				
PCE [See Chlorinated ethenes, 1,1,2,2-Tetrachloroethene]				
PCP [See Chlorinated phenols, Pentachlorophenol]				
Pentachlorobenzene [See Chlorinated benzenes]				
Pentachlorophenol [See Chlorinated phenols]				
pH	6.5–9[d]	1987	7.0–8.7 & narrative	1996
Phenanthrene [See Polycyclic aromatic hydrocarbons (PAHs)]				
Phenols (mono- & dihydric)	4.0[k]	1999		
Phenoxy herbicides[d, q]	4.0	1987		
Phthalate esters				
Di-*n*-butyl phthalate	19[c]	1993	Insufficient data	1993
Di(2-ethylhexyl) phthalate	16[c]	1993	Insufficient data	1993
Di-*n*-octyl phthalate	Insufficient data	1993	Insufficient data	1993
Picloram	29[c]	1990		
Polychlorinated biphenyls (PCBs) (total)[d]	~~0.001~~[e, f]	1987	~~0.01~~[e, f]	1991

Continued.

Continued.

Parameter[a]	Freshwater		Marine	
	Concentration (µg·L^{-1})	Date[b]	Concentration (µg·L^{-1})	Date[b]
Polycyclic aromatic hydrocarbons (PAHs)				
Acenaphthene	5.8[c]	1999	Insufficient data	1999
Acridine	4.4[c]	1999	Insufficient data	1999
Anthracene	0.012[c]	1999	Insufficient data	1999
Benz*(a)*anthracene	0.018[c]	1999	Insufficient data	1999
Benzo*(a)*pyrene	0.015[c]	1999	Insufficient data	1999
Chrysene	Insufficient data	1999	Insufficient data	1999
Fluoranthene	0.04[c]	1999	Insufficient data	1999
Fluorene	3.0[c]	1999	Insufficient data	1999
Naphthalene	1.1[c]	1999	1.4[c]	1999
Phenanthrene	0.4[c]	1999	Insufficient data	1999
Pyrene	0.025[c]	1999	Insufficient data	1999
Quinoline	3.4[c]	1999	Insufficient data	1999
Propylene glycol [See Glycols]				
Pyrene [See Polycyclic aromatic hydrocarbons (PAHs)]				
Quinoline [See Polycyclic aromatic hydrocarbons (PAHs)]				
Reactive chlorine species (hypochlorous acid and monochloramine)	0.5	1999	0.5	1999
Salinity			<10% fluctuation[c]	1996
Selenium[d]	1.0	1987		
Silver[d]	0.1	1987		
Simazine	10	1991		
Streambed substrate [See Total particulate matter]				
Styrene	72[c]	1999		
Suspended sediments [See Total particulate matter]				
TCE [See Chlorinated ethenes, 1,1,2-Trichloroethene]				
Tebuthiuron	1.6[c]	1995	Insufficient data	1995
Temperature	Narrative[d]	1987	Not to exceed ±1°C[c]	1996
Tetrachlorobenzene [See Chlorinated benzenes]				
Tetrachloroethane [See Chlorinated ethanes]				
Tetrachloroethene [See Chlorinated ethenes]				
Tetrachloroethylene [See Chlorinated ethenes, 1,1,2,2-Tetrachloroethene]				
Tetrachloromethane [See Halogenated methanes]				
Tetrachlorophenols [See Chlorinated phenols]				
Thallium[j]	0.8	1999		
Toluene	2.0[c, j, k]	1996	215[c, k]	1996

Continued.

Continued.

Parameter[a]	Freshwater Concentration (µg·L^{-1})	Freshwater Date[b]	Marine Concentration (µg·L^{-1})	Marine Date[b]
Total particulate matter[r]				
Deposited bedload sediment	Insufficient data	1999	Insufficient data	1999
Streambed substrate	Narrative	1999	Narrative	1999
Suspended sediments	Narrative	1999	Narrative	1999
Turbidity	Narrative	1999	Narrative	1999
Toxaphene[d]	~~0.008~~ [e, f]	1987		
Triallate	0.24[c]	1992		
Tribromomethane [See Halogenated methanes]				
Tributyltin [See Organotins]				
Trichlorobenzene [See Chlorinated benzenes]				
Trichloroethane [See Chlorinated ethanes]				
Trichloroethene [See Chlorinated ethenes]				
Trichloroethylene [See Chlorinated ethenes, 1,1,2-Trichloroethene]				
Trichloromethane [See Halogenated methanes]				
Trichlorophenols [See Chlorinated phenols]				
Tricyclohexyltin [See Organotins]				
Trifluralin	0.20[i]	1993		
Triphenyltin [See Organotins]				
Turbidity [See Total particulate matter]				
Zinc[d]	30	1987		

[a]Unless otherwise indicated, supporting documents are available from the Guidelines and Standards Division, Environment Canada.

[b]The guidelines dated 1987 have been carried over from *Canadian Water Quality Guidelines* (CCREM 1987) and no fact sheet was prepared. The guidelines dated 1989 to 1997 were developed and initially published in CCREM 1987 as appendixes on the date indicated. They are published as fact sheets in this document. Other guidelines dated 1997 and those dated 1999 are published for the first time in this document.

[c]Interim guideline.

[d]No fact sheet created.

[e]This guideline (originally published in *Canadian Water Quality Guidelines* [CCREM 1987 + Appendixes] in 1987 or 1991 [PCBs in marine waters]) is no longer recommended and the value is withdrawn. A water quality guideline is not recommended. Environmental exposure is predominantly via sediment, soil, and/or tissue, therefore, the reader is referred to the respective guidelines for these media.

[f]This substance meets the criteria for Track 1 substances under the national CCME Policy for the Management of Toxic Substances (PMTS) (i.e., persistent, bioaccumulative, primarily the result of human activity, and CEPA-toxic or equivalent), and should be subject to virtual elimination strategies. Guidelines can serve as action levels or interim management objectives towards virtual elimination.

[g]Aluminum guideline = 5 µg·L^{-1} at pH <6.5; [Ca^{2+}] <4 mg·L^{-1}; DOC <2 mg·L^{-1}
= 100 µg·L^{-1} at pH ≥6.5; [Ca^{2+}] ≥4 mg·L^{-1}; DOC ≥2 mg·L^{-1}

[h]Ammonia guideline = 1370 µg·L^{-1} at pH 8.0; 10ºC
= 2200 µg·L^{-1} at pH 6.5; 10ºC

[i]Guideline value slightly modified from CCREM 1987 + Appendixes due to re-evaluation of the significant figures.

[j]The technical document for the guideline is available from the Ontario Ministry of the Environment.

[k]Substance has been re-evaluated since CCREM 1987 + Appendixes. Either a new guideline has been derived or insufficient data existed to derive a new guideline.

[l]Cadmium guideline = $10^{\{0.86[\log(\text{hardness})] - 3.2\}}$.

[m] Copper guideline = 2 $\mu g \cdot L^{-1}$ at $[CaCO_3]$ = 0–120 $mg \cdot L^{-1}$
= 3 $\mu g \cdot L^{-1}$ at $[CaCO_3]$ = 120–180 $mg \cdot L^{-1}$
= 4 $\mu g \cdot L^{-1}$ at $[CaCO_3]$ >180 $mg \cdot L^{-1}$

[n] Dissolved oxygen for warm-water biota: early life stages = 6000 $\mu g \cdot L^{-1}$
other life stages = 5500 $\mu g \cdot L^{-1}$
for cold-water biota: early life stages = 9500 $\mu g \cdot L^{-1}$
other life stages = 6500 $\mu g \cdot L^{-1}$

[o] Lead guideline = 1 $\mu g \cdot L^{-1}$ at $[CaCO_3]$ = 0–60 $mg \cdot L^{-1}$
= 2 $\mu g \cdot L^{-1}$ at $[CaCO_3]$ = 60–120 $mg \cdot L^{-1}$
= 4 $\mu g \cdot L^{-1}$ at $[CaCO_3]$ = 120–180 $mg \cdot L^{-1}$
= 7 $\mu g \cdot L^{-1}$ at $[CaCO_3]$ = >180 $mg \cdot L^{-1}$

[p] Nickel guideline = 25 $\mu g \cdot L^{-1}$ at $[CaCO_3]$ = 0–60 $mg \cdot L^{-1}$
= 65 $\mu g \cdot L^{-1}$ at $[CaCO_3]$ = 60–120 $mg \cdot L^{-1}$
= 110 $\mu g \cdot L^{-1}$ at $[CaCO_3]$ = 120–180 $mg \cdot L^{-1}$
= 150 $\mu g \cdot L^{-1}$ at $[CaCO_3]$ = >180 $mg \cdot L^{-1}$

[q] The guideline of 4.0 $\mu g \cdot L^{-1}$ for phenoxy herbicides is based on data for ester formulations of 2,4-dicholorophenoxyacetic acid.

[r] The technical document for the guideline is available from British Columbia Ministry of Environment, Lands and Parks.

Reference

CCREM (Canadian Council of Resource and Environment Ministers). 1987. Canadian water quality guidelines. Prepared by the Task Force on Water Quality Guidelines.

Reference listing:

Canadian Council of Ministers of the Environment. 1999. Canadian water quality guidelines for the protection of aquatic life: Summary table. In: Canadian environmental quality guidelines, 1999, Canadian Council of Ministers of the Environment, Winnipeg.

For further scientific information, contact:

Environment Canada
Guidelines and Standards Division
351 St. Joseph Blvd.
Hull, QC K1A 0H3
Phone: (819) 953-1550
Facsimile: (819) 953-0461
E-mail: ceqg-rcqe@ec.gc.ca
Internet: http://www.ec.gc.ca

For additional copies, contact:

CCME Documents
c/o Manitoba Statutory Publications
200 Vaughan St.
Winnipeg, MB R3C 1T5
Phone: (204) 945-4664
Facsimile: (204) 945-7172
E-mail: spccme@chc.gov.mb.ca

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Cyanide Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review

by
Ronald Eisler

U.S. Fish and Wildlife Service
Patuxent Wildlife Research Center
Laurel, Maryland 20708

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Tables

Cyanide Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review

by
Ronald Eisler

U.S. Fish and Wildlife Service
Patuxent Wildlife Research Center
Laurel, Maryland 20708

Abstract. Cyanides are used widely and extensively in the manufacture of synthetic fabrics and plastics, in electroplating baths and metal mining operations, as pesticidal agents and intermediates in agricultural chemical production, and in predator control devices. Elevated cyanide levels are normally encountered in more than 1,000 species of food plants and forage crops, and this probably represents the greatest source of cyanide exposure and toxicosis to man and to range animals. Anthropogenic sources of cyanide in the environment include certain industrial processes, laboratories, fumigation operations, cyanogenic drugs, fires, cigarette smoking, and chemical warfare. Although cyanide is ubiquitous in the environment, levels tend to be elevated in the vicinity of metal processing operations, electroplaters, gold-mining facilities, oil refineries, power plants, and solid waste combustion.

Many chemical forms of cyanide are present in the environment, including free cyanide, metallocyanide complexes, and synthetic organocyanides, also known as nitriles. 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.

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. Diagnosis of acute lethal cyanide poisoning is difficult because signs and symptoms are nonspecific, and numerous factors modify its biocidal properties, such as dietary deficiencies in vitamin B_{12}, iodine, and sulfur amino acids. Among the more consistent changes measured in acute cyanide poisoning are inhibition of brain cytochrome oxidase activity, and changes in electrical activity in heart and brain. 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. Antidotes in current use to counteract cyanide poisoning include a combination of sodium nitrite and sodium thiosulphate (United States), cobalt edetate (United Kingdom, Scandinavia, France), or a mixture of 4-dimethylaminophenol and sodium thiosulphate (Germany).

All available evidence suggests that cyanides are neither mutagenic, teratogenic, nor carcinogenic. Moreover, there are no reports of cyanide biomagnification or cycling in living organisms, probably owing to its rapid detoxification. Cyanide seldom persists in surface waters and soils owing to complexation or sedimentation, microbial metabolism, and loss from volatilization. More data are needed on cyanide distribution and transformation in the atmosphere.

Analytical methods for the determination of free and bound cyanides and cyanogenic compounds in biological materials are under constant revision. Further, unless tissue samples are obtained promptly after cyanide exposure and analyzed immediately, erroneous analytical values will result.

Higher plants are adversely affected by cyanide through cytochrome oxidase inhibition; the rate of production and release of cyanide by plants to the environment through death and decomposition is unknown. Nonacclimatized soil bacteria are adversely affected at 0.3 mg HCN/kg; acclimatized populations, however, can degrade wastes containing up to 60 mg total cyanide per kilogram. In some cases, soil bacteria and fungi produce cyanides as secondary metabolites, with adverse effects on certain plants. Several species of arthropods normally contain elevated whole-body cyanide concentrations, and these confer protection against predators and allow consumption of cyanogenic plants.

Fish were the most sensitive aquatic organisms tested. Adverse effects on swimming and reproduction were observed between 5 and 7.2 µg free cyanide per liter; lethal effects usually occurred between 20 and 76 µg/L. Biocidal properties of cyanide in aquatic environments were 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.

Birds that feed predominantly on flesh were more sensitive to cyanide than were herbivores. Free cyanide levels associated with high avian death rates include 0.12 mg/L in air, 2.1-4.6 mg/kg body weight (BW) via acute oral exposure, and 1.3 mg/kg BW administered intravenously. Dietary levels of 135 mg total cyanide per kilogram ration resulted in growth reduction of chicks, but 103 mg total cyanide per kilogram ration had no measurable effect on domestic chickens.

Cyanogenic plants represent a problem for various range animals and wildlife, primarily among species that eat rapidly. Intakes of 4 mg HCN/kg BW are lethal to these species if it is consumed quickly. Cassava (*Manihot esculenta*) is a cyanogenic plant that accounts for up to 70% of human caloric intake in some areas, and this is associated with serious, long-term toxic effects including ataxia, optic nerve lesions, altered thyroid function, demyelination, and increases in tissue thiocyanate levels. Acute oral LD50 values for representative species of mammals ranged between 2 and 3.6 mg HCN/kg BW. Despite the high lethality of large single exposures, repeated sublethal doses--especially in diets--can be tolerated by many species for extended periods, perhaps indefinitely. Mammalian deaths were also recorded at air concentrations of 140 mg HCN/m^3 (exposure for 60 min) and 4,400 mg HCN/m^3 (exposure for 1 min), and at dermal applications between 2.3 mg HCN/kg BW for abraded skin and 100 mg HCN/kg BW for intact skin. Adverse nonlethal effects were noted at drinking water concentrations >150 mg HCN/L and at dietary concentrations >720 mg HCN/kg ration.

Free cyanide criteria currently proposed for natural resource protection include <3 µg/L medium for aquatic life, and <100 mg/kg diet for birds and livestock. For human health protection, free cyanide values are <10 µg/L drinking water, <50 mg/kg diet, and <5 mg/m^3 air.

Key words: Cyanide, toxic effects, wildlife, cyanogenic plants, aquatic organisms, criteria.

The origin of terrestrial life probably depended on the presence and reactivity of hydrogen cyanide and its derivatives; paradoxically, hydrogen cyanide is toxic to the majority of living matter (Marrs and Ballantyne 1987). Cyanide is a general respiratory poison--although uptake can also occur through ingestion or dermal absorption--producing reactions within seconds, and death within minutes (Towill et al. 1978; Environmental Protection Agency [EPA] 1980). The toxic mechanism of cyanide primarily involves the inhibition of cytochrome oxidase, the terminal oxidative enzyme of the mitochondrial electron transport chain, producing blockage of aerobic ATP synthesis (Egekeze and Oehme 1979; Younes and Strubelt 1988). Because of their highly effective lethal potency, cyanides were used for genocidal programs in Germany in World War II, in mass suicides by members of the People's Temple religious sect in Guyana, and in the substitution of medication in Tylenol capsules in drugstores in various cities in the United States. In fact, cyanides are responsible for more human deaths than any other chemicals known, owing to their deliberate use in suicide, murder, chemical warfare, genocide, and judicial execution (Way 1981, 1984; Ballantyne and Marrs 1987a; Gee 1987; Marrs and Ballantyne 1987; Yamamoto 1989). High sublethal doses of cyanide are rapidly detoxified, and accidental acute cyanide poisonings in humans are uncommon (Towill et al. 1978).

Cyanide compounds are useful to society in terms of their key role in synthetic and industrial processes, for certain fumigation and agricultural uses, and for some therapeutic applications (Ballantyne and Marrs 1987a). Cyanides are present in effluents from iron and steel processing plants, petroleum refineries, and metal-plating plants, and constitute a hazard to aquatic ecosystems in certain waste-receiving waters (Smith et al. 1979), and to livestock (EPA 1980;Towill et al. 1978). Cyanide serves no useful purpose in the human body, yet it is present in our food, air, and water (Becker 1985).

Natural sources of cyanide include various species of bacteria, algae, fungi, and higher plants that form and excrete cyanide (Way 1984). The most widely distributed major food crop with a high content of cyanogenic glycosides is cassava (*Manihot esculenta*), also known as manioc. Cassava is a staple food in human diets in over 80 countries, and it is sometimes added to animal feeds as a substitute for more expensive cereal grains (Gomez et al. 1988). In humans, chronic cyanide intoxication caused by consumption of cassava is the main etiological factor in the debilitating tropical ataxic neuropathy (Egekeze and Oehme 1980). Other plants having comparatively elevated cyanide content include fruit pits, sweet potatoes (*Ipomoea batatas*), corn (*Zea mays*), bamboo shoots (*Bambusa* spp.), linseed, (*Linum* sp.), lima beans (*Phaseolus lunatus*), and millet (*Panicum miliaceum*; Way 1984). In higher plants that contain cyanogenic glycosides, at least 20 of these compounds have been identified (EPA 1980). Amygdalin--one of the more intensively studied cyanogenic glycosides--is found in seeds of the cherry (*Prunus* spp.), plum (*Prunus* spp.), peach (*Prunus persica*), apricot (*Prunus armenaica*), apple (*Malus domestica*), pear (*Pyrus communis*), and many parts of the cherry laurel (*Prunus laurocerasus;* EPA 1980). Apricot seeds and peach kernels are food delicacies in Turkey, and have caused at least nine poisonings (two fatal) in children from that country (Gee 1987). Acute cyanide poisoning has occurred in the United States from the ingestion of almond-flavored milkshakes prepared from apricot kernels (Way 1984). Amygdalin is also the chief ingredient in laetrile, a medication prescribed by some physicians to control tumors. Both laetrile and amygdalin-containing fruit pits have been implicated as the causes of acute cyanide poisoning in humans (EPA 1980). Another naturally occurring group of organic cyanides (nitriles) is the highly toxic pseudocyanogenic glycosides, especially cycasin, and these have been implicated in a variety of tropical diseases of the nervous system, and partial or total blindness (EPA 1980). Other nitriles found in plants include the lathyrogenic compounds, glucosinolates, and the cyanopyridine alkaloids (EPA 1980).

That certain plants, such as bitter almonds *(Prunus dulcis),* cherry laurel leaves, and cassava, are poisonous if consumed in sufficient quantities has been known for at least 2,000 years. But it was not until the 1700's that cyanide was recognized as the basis for their lethal toxicity. The first account of an experimental administration of extract of bitter almonds and other poisons to dogs (*Canis familiaris*) dates from 1679, as reviewed by Sykes (1981) and Ballantyne (1987a). In 1731, two fatal cases of human poisoning in Ireland were caused by drinking cherry laurel water, in this instance used as a flavoring agent in cooking and to dilute brandy. In that same year it was shown that cherry laurel water administered to dogs by various routes proved rapidly fatal. By 1781, it was well established that mammals, birds, reptiles, amphibians, fish, and insects could all be killed with small doses of laurel water, and that death was more rapid than that produced by other poisons tested. It was also at this time that cyanide was first implicated as a homicidal agent in England. In 1782, hydrocyanic acid was isolated from Prussian blue (a dye) by the Swedish chemist Scheele. In 1786, Scheele accidentally broke a vial of the material and died from vapor poisoning. In 1787, it was determined that hydrocyanic acid contained hydrogen, carbon, and nitrogen, but did not contain oxygen, formerly believed to be an essential component of all acids. Between 1802 and 1815, hydrocyanic acid was found to be lethal in small quantities to birds and dogs, and to act rapidly when given orally, intravenously, or applied to the eye surface. By 1803, it was known that cyanide occurred naturally and could be extracted from apricots or almonds. In 1815, hydrocyanic acid was prepared in a semipure form. Between 1817 and 1948, cyanide, in appropriate doses, was used therapeutically in England for the treatment of pulmonary diseases and tuberculosis, and as a sedative. By 1830, cyanogenic glycosides containing HCN were isolated from cassava; today, more than 800 species of cyanogenic plants have been identified. In 1876, it was first demonstrated that cyanide inhibited tissue oxidation. In 1894, cobalt compounds were suggested as antidotes due to their marked cyanide-binding capacity. Studies on cyanide detoxification conducted between 1877 and 1894 showed that thiosulphate administration caused the formation of thiocyanate--a relatively harmless metabolite. By the late 1800's, cyanide was regarded as a common plant metabolite rather than as an unusual poison. In 1929, it was conclusively demonstrated that cyanide combines with the trivalent iron atom in cytochrome oxidase, a respiratory enzyme that links the tricarboxylic acid cycle and formation of metabolic water. Many reviews have been published on cyanide in the environment; particularly useful are those by Doudoroff (1976), Towill et al. (1978), Smith et al. (1979), Egekeze and Oehme (1980), EPA (1980, 1989), Vennesland et al. (1981a), Leduc et al. (1982), Leduc (1984), Way (1984), Ballantyne and Marrs (1987a), and Evered and Harnett (1988).

Cyanide hazards to fish, wildlife, and livestock are well documented. Massive kills of freshwater fish by accidental discharges of cyanide wastes are fairly common (Holden and Marsden 1964; Leduc 1978; Towill et al. 1978; EPA 1980). In one case, cyanide-containing mine effluents from a Canadian tailings pond released into a nearby creek killed more than 20,000 steelhead (Oncor*hynchus mykis;* Leduc et al. 1982). Many species

of birds were found dead near burrows of the blacktailed prairie dog (*Cynomys ludovicianus*) after the burrows had been treated with calcium cyanide to control prairie dog populations; dead birds included the burrowing owl (*Athene cunicularia*), the bald eagle (*Haliaeetus leucocephalus*), and the golden eagle (*Aquila chrysaetos;* Wiemeyer et al. 1986). An endangered California condor (*Gymnogyps californianus*) found dead in Kern County, California, in November 1983 had particles of a yellow fluorescent tracer in its mouth; these particles were similar to those mixed with sodium cyanide in M-44 spring-loaded ejector mechanism devices used in a U.S. Fish and Wildlife Service Animal Damage Control Program in that vicinity, suggesting that cyanide was a possible cause of death (Krynitsky et al. 1986). M-44 devices are known to have caused the death of magpies (*Pica* sp.), ravens and crows *(Corvus* spp.), wild turkeys *(Meleagris gallopavo),* and various unidentified species of hawks and vultures (Wiemeyer et al. 1986). Between 1980 and 1989, 519 mammals--mostly rodents (35%) and bats (34%)--were found dead at cyanide-extraction, gold-mine leach ponds in California, Nevada, and Arizona; the list included coyote *(Canis latrans),* foxes, skunks, badger *(Taxidea taxus),* weasels, rabbits, deer, and beavers (Clark and Hothem 1991). Also found dead at these same leach ponds were 38 reptiles, 55 amphibians, and 6,997 birds (Clark and Hothem 1991), including many species of waterfowl and songbirds (Allen 1990). The influence of cyanide-extraction gold-mining operations on wildlife is currently under investigation by scientists at the Patuxent Wildlife Research Center.

The major threat of cyanide poisoning to livestock and terrestrial mammalian wildlife is through ingestion of plants containing high levels of cyanogenic glycosides (Towill et al. 1978; Marrs and Ballantyne 1987). Plants implicated in cyanide poisoning of animals include the sorghums (Johnson grass, *Sorghum halepense;* Sudan grass, *Sorghum sudanense*), arrowgrass (*Triglochin* spp.), elderberry (*Sambucus* spp.), wild cherry (*Prunus* spp.), and the pits of several common fruits, such as apple, peach, and apricot; these plants and fruit pits have the potential of releasing cyanide upon ingestion (Egekeze and Oehme 1980). Domestic goats (*Capra* spp.) died of cyanide poisoning after eating leaves and fruit of the crab apple *(Malus sylvestris);* the crab apple contains cyanogenic glycosides in its leaves and fruit (Shaw 1986). Cyanide poisoning of cattle (*Bos* spp.) by forage sorghums and various hybrid cultivars has been reported in India (Bapat and Abhyankar 1984) and elsewhere (Cade and Rubira 1982; Biehl 1984). Cattle appear to be more vulnerable to cyanide poisoning than are sheep (*Ovis aries*), horses (*Equus caballus*), and pigs (*Sus* spp.; Cade and Rubira 1982). *Equine sorghum cystitis ataxia* is a condition observed in horses grazing on Sorghum or hybrid Sudan grass pastures; it is characterized by urinary incontinence, posterior incoordination, and degenerative central nervous system lesions (Egekeze and Oehme 1980). Grazing cyanogenic plants can induce sulfur deficiency in sheep, presumably because sulfur detoxifies the released cyanide (Towill et al. 1978). The increasing use of cassava and other cyanogenic plants in animal feeding portends a greater exposure to dietary cyanides (Davis 1981).

This report briefly reviews the technical literature on ecological and toxicological aspects of cyanide, with emphasis on fishery and wildlife resources, and provides recommendations for the protection of sensitive species of concern to the U.S. Fish and Wildlife Service. This account is part of a continuing series of synoptic reviews prepared in response to informational requests from Service environmental specialists.

Chemical Properties

The chemical speciation of cyanides varies according to their source. Specific terms used to describe cyanide include free cyanide, cyanide ion, simple cyanides, complex cyanides, nitriles, cyanogens, and total cyanide. The most common forms of cyanide in the environment are free cyanide, metallocyanide complexes, and synthetic nitriles. A brief description of each cyanide species follows (Smith et al. 1978, 1979; Towill et al. 1978; Egekeze and Oehme 1980; EPA 1980, 1989; Davis 1981; Leduc 1981, 1984; Leduc et al. 1982; Simovic and Snodgrass 1985; Ballantyne 1987a; Homan 1987; Marrs and Ballantyne 1987).

Free cyanide is the primary toxic agent in the aquatic environment. Free cyanide refers to the sum of molecular HCN and the cyanide anion (CN^-), regardless of origin. In aqueous solution with pH 9.2 and lower, the majority of the free cyanide is in the form of molecular HCN. The chemical names for HCN include hydrogen cyanide, hydrocyanic acid, cyanohydric acid, and prussic acid. Hydrogen cyanide (Table 1) is a colorless, flammable liquid or gas that boils at 25.7° C and freezes at -13.2° C. The gas rarely occurs in nature, is lighter than air, and diffuses rapidly; it is usually prepared commercially from ammonia and methane at elevated temperatures with a platinum catalyst. It is miscible with water and alcohol, but is only slightly soluble in ether. In water, HCN is a weak acid with the ratio of HCN to CN^- about 100 at pH 7.2, 10 at pH 8.2, and 1 at

pH 9.2. HCN can dissociate into H and CN^-. Cyanide ion, or free cyanide ion, refers to the anion CN^- derived from hydrocyanic acid in solution, in equilibrium with simple or complexed cyanide molecules. Cyanide ions resemble halide ions in several ways and are sometimes referred to as "pseudohaline" ions. For example, silver cyanide is almost insoluble in water, as are silver halides. Cyanide ions also form stable complexes with many metals.

Simple cyanides typically refer to alkali water-soluble salts, such as NaCN, KCN, $Ca(CN)_2$, and $Hg(CN)_2$, but also include several cyanide salts of alkali, alkaline earth, or heavy metals, that is, $Zn(CN)_2$, $Cd(CN)_2$, $Ni(CN)_2$, and AgCN, of varying degrees of solubility. In water, NaCN and KCN will completely dissociate to give free cyanide. All simple cyanides ionize in water to release cyanide ion which, depending on pH, will form hydrocyanic acid. For sodium cyanide, the reaction proceeds as follows:

(1) $NaCN \quad Na^+ + CN^-$

(2) $CN^- + HOH \quad HCN + OH^-$

Increased pH will maintain a larger fraction of the cyanide as CN^-, and acidification will cause the reverse. At pH 7, about 99% of the free cyanide is in the form of HCN, whereas at pH 9.3 HCN composes 50%. Since HCN is extremely water soluble and is also one of the most toxic cyanide species, it is noteworthy that the toxicity of simple cyanides will not be affected measurably below pH 8.3. Acidification of dilute (milligrams per liter) cyanide solutions will not initiate any greater release of HCN, but acidification of concentrated (grams per liter) solutions promotes HCN formation and release.

Table 1. Some properties of potassium cyanide, hydrogen cyanide, and sodium cyanide (from EPA 1989).

Property	Potassium cyanide	Hydrogen cyanide	Sodium cyanide
CAS number	151-50-8	74-90-8	143-33-9
Chemical formula	KCN	HCN	NaCN
Molecular weight	65.12	27.03	49.01
Physical state	Solid	Gas or liquid	Solid
Boiling point (º C)	--	25.7	1,496
Melting point (º C)	634.5	–13.21	563.7
Specific gravity	1.5	0.7 (liquid)	1.6
Solubility in water (g/L)	716 at 20º C	Miscible	480 at 10º C

Complex cyanides are compounds in which the cyanide anion is incorporated into a complex or complexes; these compounds are different in chemical and toxicologic properties from simple cyanides. In solution, the stability of the cyanide complex varies with the type of cation and the complex that it forms. Some of these are dissociable in weak acids to give free cyanide and a cation, while other complexes require much stronger acidic conditions for dissociation. The least-stable complex metallocyanides include $Zn(CN)_4^{2-}$, $Cd(CN)_3^-$, and $Cd(CN)_4^{2-}$; moderately stable complexes include $Cu(CN)_2^-$, $Cu(CN)_3^{2-}$, $Ni(CN)_4^{2-}$, and $Ag(CN)^{2-}$; and the most stable complexes include $Fe(CN)_6^{4-}$and $Co(CN)_6^{4-}$**.** The toxicity of complex cyanides is usually related to their ability to release cyanide ions in solution, which then enter into an equilibrium with HCN; relatively small fluctuations in pH significantly affect their biocidal properties.

Cyanogen [$(CN)_2$] is the simplest compound containing the cyanide group. Cyanogen is an extremely toxic, flammable gas that reacts slowly with water to form HCN, cyanic acid, and other compounds; it is rapidly

degraded in the environment. Cyanogen and its halide derivations are comparable in toxicity to hydrogen cyanide.

Nitriles are defined as organic compounds (RCN) containing the cyanide group. Cyanide bound to carbon as nitriles (other than as cyanogenic glycosides) are comparatively innocuous in the environment, and are low in chemical reactivity and are biodegradable. For simple mononitriles there is a clear progression, with more cyanide being released as chain length increases. A similar pattern exists in dinitriles, but corresponding compounds require a longer carbon chain than mononitriles before free cyanide is produced. Based on studies with chicken liver homogenates (Davis 1981), mononitriles were more toxic than dinitriles, and within each group the order of toxicity was $CH_3 > C_2H_5 > C_3H_7 > C_4H_9 > C_5H_{11} > C_7H_{15}$. Cyanohydrins [$R_2C(OH)CN$] and cyanogenic glycosides [$R_1R_2C(OR_3)CN$] are special classes of nitriles, in that under appropriate conditions they will decompose to HCN and cyanide ions. Cyanogens (not to be confused with cyanogen), such as acrylonitrile, propionitrile, and succinonitrile, are nitrile-containing materials of varying complexity and lability, and can liberate free and toxicologically available amounts of cyanide. But the nonnitrile portion of the cyanogen molecule may exert an independent or interactive toxicity, causing a complex response.

Cyanates contain the OCN group. Inorganic cyanates that are formed industrially by the oxidation of cyanide salts hydrolyze in water to form ammonia and bicarbonate ion. Alkyl cyanates are insoluble in water and form cyanurates. Alkyl isocyanates contain the OCN radical, are formed from cyanates, and, like cyanates, are readily hydrolyzed. Thiocyanates (SCN group) are formed from cyanides and sulfur-containing materials and are relatively stable.

Total cyanides refers to all cyanide-containing compounds, including simple and complex cyanides, cyanoglycosides, and free cyanide. Total cyanides is a chemical measurement of free cyanide present in solution or released by acidification or digestion. Only free cyanide is considered to be a biologically meaningful expression of cyanide toxicity. Under most circumstances, the concentration of total cyanide will exceed that of HCN. In some waters, however, the total cyanide concentration may consist almost entirely of free cyanide, or it may contain cyanides that readily photodecompose or dissociate to yield HCN. The relation between total cyanide and free cyanide in natural waters varies with receiving-water conditions, type of cyanide compounds present, degree of exposure to daylight, and presence of other chemical compounds.

Hydrogen cyanide has frequently been associated with the odor of bitter almonds (Ballantyne 1983; Gee 1987). The threshold odor for olfactory detection of atmospheric HCN is 1 mg/L, but the odor may not be detected for various reasons, including the presence of other odors and the fact that only 20% to 40% of those tested could detect a cyanide odor.

Analytical methods for determining free and bound cyanide and cyanogenic compounds in biological materials are under revision. Current methods include chromatography; enzymic postcolumn cleavage; electrochemical detection; and ultraviolet, infrared, proton, and carbon-13 nuclear magnetic resonance spectroscopies (Brimer 1988). Proposed newer analytical methodologies include chemiluminescence (Wu et al. 1989); deproteinization techniques (Krynitsky et al. 1986); thin film dissociation coupled with preferential ultraviolet irradiation (Kelada 1989); differential pulse polarography (Westley 1988); and modified spectrophotometric (Blago 1989; Ohno 1989), colorometric (Lundquist and Sorbo 1989), and ion chromatographic determinations (Nonomura and Hobo 1989). Analysis of cyanide and cytochrome oxidase is usually conducted with samples of whole blood, serum, plasma, brain, or ventricular myocardium tissues. Samples should be obtained as soon as possible after cyanide exposure and analyzed immediately, otherwise erroneous analytical values will result (Towill et al. 1978; Ballantyne 1983). Brain and liver are recommended for cyanide analysis if removed and analyzed within a week (Ballantyne et al. 1974). Cyanide measurements are further confounded by the presence of various antidotal agents (Ballantyne 1983); by various tissue preservatives, such as formaldoxime (Knocke 1981) and sodium fluoride (Curry et al. 1967); and by the spontaneous postmortem production of cyanide in various tissues (e.g., sterile blood, brain, liver, kidney, uterus, intestines) over time in cases of noncyanide death (Curry et al. 1967; Ballantyne et al. 1974).

Mode of Action

Cyanide is a potent and rapid-acting asphyxiant. At lethal doses, inhalation or ingestion of cyanide produces reactions within seconds and death within minutes. Cyanide's toxic effect is due to its affinity for the ferric heme form of cytochrome a_3, also known as cytochrome c oxidase, the terminal oxidase of the mitochondrial respiratory chain (Towill et al. 1978; Egekeze and Oehme 1980; Solomonson 1981; Way 1981, 1984; Leduc et al. 1982; Biehl 1984; Ballantyne 1987a; Marrs and Ballantyne 1987; Yamamoto 1989). Inhibition of the enzyme cytochrome c oxidase is thought to involve a two-step reaction--initial penetration of cyanide into a protein crevice followed by binding to heme iron. Formation of a stable cytochrome c oxidase-CN complex in the mitochondria produces a blockage of electron transfer from cytochrome oxidase to molecular oxygen and cessation of cellular respiration, causing cytotoxic hypoxia in the presence of normal hemoglobin oxygenation. Tissue anoxia induced by the activation of cytochrome oxidase causes a shift from aerobic to anaerobic metabolism, resulting in the depletion of energy-rich compounds such as glycogen, phosphocreatine, and adenosine triphosphate, and the accumulation of lactate with decreased blood pH. The combination of cytotoxic hypoxia with lactate acidosis depresses the central nervous system--the most sensitive site of anoxia--resulting in respiratory arrest and death. If the absorption rate is significantly greater than the detoxification rate, there will be a rapid accumulation of free cyanide in tissues and body fluids, resulting in the prompt onset of signs of acute cyanide poisoning. Acute cyanide poisoning is frequently encountered as a relatively massive overdose, where the amount of cyanide greatly exceeds the minimal concentration necessary to inhibit cytochrome c oxidase. In such cases, many enzymes and biological systems are disrupted, including various metalloenzymes, nitrate reductase, nitrite reductase, myoglobin, various peroxidases, catalase, and ribulose diphosphate carboxylase, resulting in severe signs of toxicity and rapid death.

The great majority of the absorbed cyanide reacts with thiosulfate in the presence of enzymes to produce thiocyanate, which is excreted in the urine over a period of several days. Owing to this rapid detoxification, animals can ingest high sublethal doses of cyanide over extended periods without harm (Towill et al. 1978; Egekeze and Oehme 1980; EPA 1980; Davis 1981; Solomonson 1981; Leduc 1984; Ballantyne 1987a; Oh et al. 1987; Marrs and Ballantyne 1987; Westley 1988; Mengel et al. 1989). Authorities are also in general agreement on several points: thiosulfate is usually low in the body, and higher levels can significantly protect against cyanide toxicity; species vary considerably in both the extent to which thiocyanate is formed and the rate at which it is eliminated from the body; thiocyanate metabolites resulting from the transulfuration process are about 120 times less toxic than the parent cyanide compound; thiocyanate may accumulate in tissues and has been associated with developmental abnormalities and other adverse effects; the two enzyme systems responsible for the transulfuration process are thiosulfate-cyanide sulfurtransferase--also known as rhodanese--and beta-mercaptopyruvate cyanide sulfurtransferase. Rhodanese is widely distributed in the body, but activity levels in mammals are highest in the mitochondrial fraction of liver. Rhodanese activity levels in catalyzing the transformation of thiosulfate to thiocyanate are limited by the availability of sulfur.

Minor detoxification pathways for cyanide include exhalation in breath as HCN and as $C0_2$ from oxidative metabolism of formic acid; conjugation with cystine to form 2-iminothiazolidene- 4-carboxylic acid or 2-aminothiazoline-4-carboxylic acid; combining with hydroxocobalamin (B_{12}) to form cyanocobalamin, which is excreted in urine and bile; and binding by methemoglobin in the blood (Towill et al. 1978; EPA 1980; Ballantyne 1987a; Marrs and Ballantyne 1987).

Absorption of hydrogen cyanide liquid or gas readily occurs through inhalation, ingestion, or skin contact (Towill et al. 1978; Egekeze and Oehme 1980; EPA 1980; Homan 1987). Inhalation and skin absorption are the primary hazardous routes in cyanide toxicity in occupational exposure. Skin absorption is most rapid when the skin is cut, abraded, or moist. Inhalation of cyanide salts is also potentially hazardous because the cyanide dissolves on contact with moist mucous membranes. Regardless of route of exposure, cyanide is readily absorbed into the bloodstream and distributed throughout the body. Cyanide concentrates in erythrocytes through binding to methemoglobin (Towill et al. 1978; EPA 1980), and free cyanide concentrations in plasma are now considered one of the better indicators of cytotoxicity (Ballantyne 1987a). Because of the affinity of cyanide for the mammalian erythrocyte, the spleen may contain elevated cyanide concentrations when compared to blood; accordingly, spleen should always be taken for analysis in cases of suspected cyanide poisoning (Ballantyne 1975). Cyanide also accumulates in various body cells through binding to metalloproteins or enzymes such as catalase and cytochrome c oxidase (EPA 1980). The brain is probably the major target organ

of cytotoxic hypoxia, and brain cytochrome oxidase may be the most active site of lethal cyanide action, as judged by distribution of cyanide, thiosulfate, and rhodanese (Solomonson 1981; Ballantyne 1987a). Significant positive correlations exist between cyanide concentrations in plasma, cerebrospinal fluid, and brain (Ballantyne 1987a); these correlations need further exploration.

Hydrogen cyanide formation may contribute to the toxicity of snake venom, owing to the high levels of L-amino acid oxidase in some snake venoms (Vennesland et al. 1981b). This enzyme is harmless on injection, but the tissue destruction caused by other venom components probably provides the required substrate and cofactor for HCN production.

Cyanide inhibits ion transport mechanisms in amphibian skin, gall bladder, and proximal renal tubules (Bello-Reuss et al. 1981). Measurable changes in cell membrane potentials of isolated gall bladder epithelium cells, for example, were induced by NaCN in a salamander (Bello-Reuss et al. 1981). Cyanide-induced hyperpolarization was caused primarily by an increase in permeability of the cell membrane to potassium, which, in turn, was mediated by an elevation of intracellular calcium ion activity, attributable to release from mitochondrial sources.

The binding rate of CN to hemeproteins, specifically hemoglobin components Ill and IV, is 370 times to 2,300 times slower in a marine polychaete annelid (*Glycera dibranchiata*), when compared to guinea pig (*Cavia* spp.), soybean (*Glycine* max), and sperm whale (*Physeter macrocephalus*); the significance of this observation is unclear but warrants further exploration (Mintorovitch et al. 1989).

Clinical Features

Accidental exposure to cyanides or cyanogens through inhalation, skin exposure, and swallowing occurs in agricultural fumigation, laboratories, industrial operations, domestic abuse, and products of combustion (Ballantyne and Marrs 1987b). Intentional exposure is reported from homicides, suicides (usually uncommon), judicial executions, chemical warfare, and covert activities (Ballantyne and Marrs 1987b).

Diagnosis of lethal cyanide poisoning is difficult because of the absence of gross pathology or histology, nonspecific congestion of viscera, and cerebral or pulmonary edema. Sometimes the blood is bright red, and sometimes the odor of bitter almonds is detected, but neither is sufficiently consistent for diagnostic purposes (Ballantyne and Marrs 1987b).

At low lethal doses of cyanide, the effects are principally on cytochrome oxidase in the central nervous system. At higher doses, cardiovascular signs and changes in electrical activity of the brain are among the most consistent changes measured (Way 1981, 1984). Acute and subacute toxic effects of poisoning with cyanide can vary from convulsions, screaming, vomiting, and bloody frothing to less dramatic events, such as a slow, quiet onset to coma and subsequent death (Way 1981). In the first stage of cyanide poisoning, victims exhibit headache, vertigo, weak and rapid pulse, nausea, and vomiting. In the second stage, there are convulsions, falling, dilated pupils, clammy skin, and a weaker and more rapid pulse. In the final stage, heartbeat becomes irregular and slow; body temperature falls; there is cyanosis of lips, face, and extremities, coma, frothy bloody saliva flow from mouth, and death (Way 1981). If acute exposure is to a sublethal dose of cyanide, this may lead to signs of toxicity, but as detoxification proceeds these signs will become less obvious and eventually vanish, and cyanide will be excreted as thiocyanate without accumulating (Ballantyne 1987a).

Chronic cyanide poisoning may develop in individuals who ingest significant quantities of cyanide or cyanide precursors in their diets; effects are exacerbated by dietary deficiencies in vitamin B_{12}, iodine, and sulfur amino acids, as well as by low levels and insufficient distribution of detoxifying enzymes such as rhodanese (Solomonson 1981). Cyanide toxicity of dietary origin has been implicated in acute animal deaths and as a major etiologic factor in toxic ataxic neuropathy in humans, and as a cause of blindness in humans suffering from tobacco amblyopia and Leber's hereditary optic atrophy (Egekeze and Oehme 1980). An increase in blood plasma cyanide is observed in healthy individuals who smoke cigarettes (Cailleux et al. 1988). An increase in blood plasma thiocyanate is also seen in smokers and in hemodialysis patients just before dialysis (Cailleux et al. 1988). Continuous intake of cyanide causes high levels of plasma thiocyanate and goiters in mammals; the antithyroid action (goiters) results from cyanide interference with iodine transport and thyroxine synthesis (Solomonson 1981; Leduc 1981, 1984). Signs of chronic cyanide poisoning include demyelination, lesions of the optic nerve, decrease in sulfur-containing amino acids, increase in thiocyanate, goiter, ataxia, hypertonia,

and depressed thyroid function (Solomonson 1981). These effects are common in areas that depend on cyanogenic plants--such as cassava--as a major dietary component (Solomonson 1981).

Biochemically, cyanide affects the citric acid cycle; strongly inhibits catalases and proteinases; induces glycolysis in protozoans, fish, and mammals; produces vitamin B_{12} deficiency; and modifies the phosphorylation mechanism of respiratory mitochondrial enzymes, causing arrested respiration due to inability to use oxygen (Leduc 1984).

Cyanide biomagnification or cycling has not been reported, probably because of cyanide's high chemical reactivity and rapid biotransformation (Towill et al. 1978; Marrs and Ballantyne 1987).

There is no evidence that chronic exposure to cyanide results in teratogenic, mutagenic, or carcinogenic effects (EPA 1980). Cyanide possibly has antineoplastic activity, as judged by a low therapeutic success against rat sarcomas (EPA 1980), but this requires additional documentation.

Confirmatory evidence of cyanide poisoning includes elevated blood thiocyanate levels--except, perhaps, when death was rapid--and reduced cytochrome oxidase activity in brain and myocardium, provided that all tissues were taken within a day or so of death, frozen quickly, and analyzed shortly thereafter (Biehl 1984; Marrs and Ballantyne 1987). Evaluation of cyanide poisoning and metabolism includes signs of toxicity, LD50 values, measurement of cyanide and thiocyanate concentrations, cytochrome c oxidase activity, metabolic modification of in vivo cyanogenesis, rate of cyanide liberation in vitro, and influence of modifying factors such as the animal species, dose, rate and frequency of administration, route of exposure, differential distribution of cyanide, detoxification rates, circadian rhythm interactions, age of the organism, and presence of antidotes (Ballantyne 1987a). For example, the concentration of cyanide measured in body fluids and tissues in humans and other animals following lethal administration of cyanide depends on several factors: route of exposure, with oral route yielding highest residues and inhalation route the lowest; amount and duration of exposure; nature of the material, with HCN and CN^- being most toxic; time to death; antidotes used; time to autopsy, with marked loss documented from simple evaporation, thiocyanate formation, hydrolysis, and polymerization; and time from autopsy to sample analysis, wherein cyanide concentrations may increase due to microbial action (Ballantyne and Marrs 1987b).

Antidotes

The antagonism of cyanide intoxication has been under investigation for at least 150 years. In 1840, cyanide lethality was reported to be antagonized by artificial respiration. In 1888, amyl nitrite was reported effective in antagonizing lethal effects of cyanide in dogs. In 1894, cobalt was shown to form a stable metal complex with cyanide and was used to antagonize cyanide. In 1933, the use of sodium thiosulfate as the sulfur donor was described (Way 1984). Many compounds are used today as cyanide antidotes including cobalt salts, rhodanese, sulfur donors, methemoglobin producers, carbohydrates, drugs used to treat acidosis, oxygen, methylene blue, 4-dimethylaminophenol, various aromatic amino- and nitro-compounds (such as aniline, p-aminopropiophenone, nitrobenzene), carbonyl compounds, and sodium pyruvate (Egekeze and Oehme 1980; EPA 1980; Solomonson 1981; Way 1981, 1984; Biehl 1984; Becker 1985; Ballantyne 1987b; Marrs 1987; Marrs and Ballantyne 1987; Way et al. 1988). Different antidotes are preferred in different countries: in the United States, a mixture of sodium nitrite and sodium thiosulfate; in France and the United Kingdom, cobalt edetate, also known as Kelocyanor; and in Germany, a mixture of 4-dimethylaminophenol and sodium thiosulfate.

The classic nitrite-thiosulfate treatment of cyanide poisoning, developed almost 60 years ago, is one of the antidotal combinations still employed (Way 1981). Excess oxygen improves this antidotal combination by potentiating the effectiveness of the nitrite-thiosulfate combination, as confirmed by studies in sheep and rats (Way 1984), even though, theoretically, oxygen should serve no useful purpose (Way et al. 1988). This therapeutic regimen protected rats against 20 LD50 doses of cyanide (Towill et al. 1978). Nitrite converts hemoglobin to methemoglobin, which has a high affinity for cyanide. The methemoglobin-HCN complex then slowly releases cyanide, which is converted to thiocyanate by way of rhodanese (Solomonson 1981). Sodium nitrite, administered intravenously, is now considered one of the more rapid therapeutic methods (Way 1984). The injection of sodium thiosulfate provides sulfur for the enzyme rhodanese to mediate the biotransformation of cyanide to the much less toxic thiocyanate (Egekeze and Oehme 1980). Multiple injections of sodium thiosulfate protected mice against death by organic cyanides and were more effective than sodium nitrite

(Willhite and Smith 1981). The nitrite-thiosulfate antidotal combination is one of the most effective treatments of cyanide poisoning, even though the specific mechanism of action of these two compounds is now being questioned, and concerns have been raised because of the toxicity of nitrite (Way 1981, 1984). One accepted therapy is an intravenous combination of sodium nitrite (1 mL of 20% solution) and sodium thiosulfate (3 mL of 20% solution), giving 4 mL of this mixture per 45 kg of body weight (Egekeze and Oehme 1980). For maximal effectiveness in treating cyanide intoxication in sheep, large doses of sodium thiosulfate (660 mg/kg BW) are given in combination with conventional doses of sodium nitrite (6.6 mg/kg BW; Egekeze and Oehme 1980). Livestock treatment in cases of suspected cyanide intoxication consists of intravenous administration of sodium nitrite at 10-20 mg/kg BW followed by sodium thiosulfate at 30-40 mg/kg BW; however, a sodium thiosulfate dose of 500 mg/kg BW, or more, may be more efficacious (Biehl 1984). Once clinical signs have abated, 1 g of activated charcoal per kilogram BW may be administered as a drench by way of a stomach tube (Biehl 1984). A 30-kg female goat *(Capra* sp.) was successfully treated after eating the leaves and fruit of the crab apple *(Malus sylvestris),* a plant that contains high levels of cyanogenic glycosides in leaves and fruits (Shaw 1986). Treatment consisted of four hourly treatments of 100 g of animal charcoal and bismuth subnitrate in water as a drench, followed by 300 mg sodium nitrite as a 1% aqueous solution, then 25 g of sodium thiosulfate. Another goat died despite identical treatment (Shaw 1986).

Cobalt compounds, such as hydroxocobalamin and its derivatives (i.e., cobalt histidine, cobalt chloride, dicobalt ethylenediamine tetracetic acid) have been used to treat cyanide poisoning for more than 100 years. Their efficacy was confirmed in pigeons (*Columba* sp.) and rabbits (*Oryctolagus sp*.), but cobalt compounds did not receive wide support as cyanide antagonists because of the inherent toxicity of cobalt ion (Way 1981, 1984). Nevertheless, proponents of the use of cobalt compounds (i.e., the United Kingdom, Scandinavia, much of Europe) stress the rapidity of action in forming a stable metal complex with cyanide, thereby preventing its toxic effect (Towill et al. 1978; Way 1984). One of the more frequently used cobalt compounds in cyanide treatment is hydroxocobalamin, which reverses cyanide toxicity by combining with cyanide to form cyanocobalamin (EPA 1980; Solomonson 1981). Hydroxocobalamin has been used in guinea pigs and baboons *(Papio anubis)* to lower blood cyanide levels, and in humans after inhalation or ingestion of cyanide compounds (Egekeze and Oehme 1980).

Dimethylaminophenol (DMAP) forms methemoglobin by setting up a catalytic cycle inside the erythrocyte, in which oxygen oxidizes the DMAP to N-N-dimethylquinoneimine, the latter oxidizing the hemoglobin to methemoglobin (Marrs 1987). Dogs poisoned with KCN and given DMAP intravenously had restored respiration and decreased plasma cyanide levels. The 4-dimethylamino-phenol induced ferrihemoglobin production, which combined with the cyanide in the red cells to form ferrihemoglobin cyanide (Christel et al. 1977).

No usable cyanide prophylactic therapy now exists for humans, although sodium thiosulfate, hydroxocobalamin, and other compounds have been used to protect against cyanide toxicity in laboratory animals (Mengel et al. 1989). For example, pyridoxal 5-phosphate, the active form of vitamin B_6, readily forms complexes with cyanides, and was effective in providing significant protection to rats (Keniston et al. 1987). Fructose fed prior to insult lessens cyanide-induced hepatotoxicity in rats (Younes and Strubelt 1988). L-ascorbic acid and dehydroascorbic acid probably act as protectants against cyanide toxicity by way of nontoxic cyanohydrin formation (Sprince et al. 1982). Carbon tetrachloride pretreatment was effective in protecting mice against death from most nitriles (Willhite and Smith 1981), and pretreatment with p-aminopropiophenone serves to protect against cyanide toxicity (D'Mello 1987).

Sources and Uses

Production of cyanides in the United States increased from about 136 million kg in 1963 to 318 million kg in 1976 (Towill et al. 1978; Way 1981; Marrs and Ballantyne 1987). Cyanide consumption in North America was 64 million kg in 1988 and 98 million kg in 1989; about 80% of these amounts was used in gold mining (Knudson 1990).

About 84% of domestic HCN production is used to produce organic cyanides, also known as nitriles, including acrylonitriles, methyl methacrylate, and adiponitrile (Towill et al. 1978). Nitriles tend to polymerize, which is the basis for their use in the manufacture of synthetic fibers, resins, plastics, dyestuffs, vitamins, solvents, elastomers, agricultural insecticides, and high pressure lubricants (Willhite and Smith 1981). The widespread usefulness of HCN is related to its strong tendency and that of its inorganic salts to form complexes

with metals. For example, sodium cyanide is used in metallurgy for the extraction of gold and silver from ores and in electroplating baths because it forms stable soluble complexes. Similar behavior makes alkali cyanide solutions excellent for cleaning silverware and other precious metals and is responsible for their general use in industry as metal cleaners (Towill et al. 1978). In Canada, more than 90% of the gold mined 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 precipitated with the addition of zinc dust. A variety of cyanide compounds are produced during gold cyanidation (Simovic and Snodgrass 1985). In addition to their primary use in the metals and electroplating industries, and in the manufacture of synthetic fibers and plastics, various cyanide compounds have been used directly or as an intermediate to produce synthetic rubber, fumigants, rodenticides, insecticides, predator control agents, rocket fuels, paints and paint finishes, paper, nylon, pharmaceuticals, photographic chemicals, mirrors, cement, perfume, bleaches, soaps and detergents, riot control agents, fertilizers, and herbicides (Towill et al. 1978; Way 1981; Willhite and Smith 1981; Leduc 1984; Homan 1987).

Hydrogen cyanide vapor, because of its high and rapid acute lethal toxicity and ready diffusion, has been used widely to fumigate buildings, ships, and warehouses; to exterminate rabbits, rodents, and large predators; and in horticultural practice, to control insect pests that have developed resistance to other pesticides (Homan 1987; Ballantyne 1988). Typically, fumigation powders containing either calcium cyanide, $Ca(CN)_2$, or sodium cyanide, NaCN, are blown into burrows or scattered over the floor in greenhouses. On coming into contact with water, such powders liberate HCN vapor (Ballantyne 1988). Hydrogen cyanide released from $Ca(CN)_2$ is registered for use on almonds, dried beans, citrus, cocoa beans, grains, nuts, and spices (Towill et al. 1978). Cyanide-containing compounds are used for a variety of agricultural and pesticidal agents. These compounds include cyanogen (NCCN), as an intermediate in the production of some commercial fertilizers; cyanogen chloride (CNCl), in the manufacture of triazine herbicides; cyanogen bromide (CNBr), as a pesticidal fumigant; hydrogen cyanide, in the synthesis of methionine for animal feeds; ammonium thiocyanate (NH_4SCN), as a cotton defoliant; sodium thiocyanate (NaSCN), as a weedkiller; and calcium cyanamide (CaNCN), as a plant fertilizer, herbicide, pesticide, and defoliant of cotton and tomatoes (Homan 1987). Cyanide compounds have also been used as preservatives for raw vegetables (Towill et al. 1978).

Sodium cyanide has been used for about 50 years by the U.S. Fish and Wildlife Service against coyote in attempts to protect livestock, especially sheep. The Service has made extensive use of two NaCN ejector devices: "the coyote getter," from the late 1930's to 1970; and the M-44, from about 1968 to the present, except for the period 1972-74, when all uses of NaCN for predator control were canceled (EPA 1976a; Connolly and Simmons 1984). Although both ejectors dispense toxicant when pulled, they differ in the way ejection is achieved. In the coyote getter, the toxicant is in a 0.38-caliber cartridge case and is expelled by the explosive force of the primer plus a small powder charge. The M-44 uses a spring-driven plunger to push out its toxic contents. M-44 capsules weigh about 0.94 g, and consist of about 89% NaCN, 6% Celatom MP-78 (mostly diatomaceous silica), 5% potassium chloride, and 0.25% FP Tracerite yellow--used as a fluorescent marker (Connolly and Simmons 1984). Coyote getters and M-44's are set into the ground with only their tops protruding. Fetid scent or lure stimulates a coyote to bite and pull, whereupon a lethal dose of NaCN is ejected into its mouth; coma and death follow in 30 to 60 s. Although coyote getters were about 99% effective against coyotes, compared with 73% for M-44's, the Service decided that spring-driven plungers were less hazardous to operators than were explosive-driven plungers (Connolly and Simmons 1984). The coyote getter was generally much more selective than the trap for the capture of coyotes. It was less destructive than traps to small mammals, birds of prey, ground-nesting birds, deer, antelope, and domestic sheep, but more destructive to dogs, bears, and cattle (Robinson 1943). In a 1-year test period (1940-41) in Colorado, Wyoming, and New Mexico, the following numbers of animals were killed by the coyote getter: 1,107 coyotes, 2 bobcats (*Lynx rufus*), 24 dogs, 14 black-billed magpies (*Pica pica*), 7 foxes (*Vulpes* sp.), 8 unidentified skunks, 2 badgers, 2 unidentified eagles, 2 bears (*Ursus* sp.), and 1 each of hawk (unidentified), pika (*Ochotona* sp.), and cow (Robinson 1943).

Cyanide compounds have been used to collect various species of freshwater fish. In England and Scotland, cyanides are used legally to control rabbits, and illegally to obtain Atlantic salmon (*Salmo salar*) and brown trout (*Salmo trutta*) from rivers, leaving no visible evidence of damage to the fish (Holden and Marsden 1964). Sodium cyanide has been applied to streams in Wyoming and Utah to collect fish through anesthesia; mountain whitefish (*Prosopium williamsoni*) were sensitive to cyanide and died at concentrations that were tolerable to salmon and trout (Wiley 1984). Sodium cyanide was also used as a fish control agent in Illinois, Nebraska,

South Dakota, Missouri, and in the lower Mississippi River valley, but was never registered for this use because of human safety concerns (Lennon et al. 1970).

Cyanide compounds have been prescribed by physicians for treatment of hypertension and cancer (Sprince et al. 1982). Sodium nitroprusside ($Na_2Fe(CN)_5NO\text{-}2H_2O$) was widely used for more than 30 years to treat severe hypertension and to minimize bleeding during surgery (Solomonson 1981; Vesey 1987). Laetrile, an extract of ground apricot kernels, has been used for cancer chemotherapy and, in deliberate high intakes, as an attempted suicide vehicle (Gee 1987).

Road salt in some areas may contribute to elevated cyanide levels in adjacent surface waters (Ohno 1989). In climates with significant snowfall, road salt is applied as a deicing agent. Road salts are commonly treated with anticaking agents to ensure uniform spreading. One anticaking agent, sodium hexacyanoferrate, decomposes in sunlight to yield the highly toxic free cyanide that contaminates surface waters by runoff (Ohno 1989). Another anticaking agent, yellow prussiate of soda (sodium ferrocyanide), has been implicated in fish kills when inadvertently used by fish culturists (Barney 1989).

The military uses of HCN were first realized by Napoleon III, but it was not until World War I (WW I) that this application received widespread consideration. About 3.6 million kg of hydrogen cyanide were manufactured by France as a chemical weapon and used in WWI in various mixtures called Manganite and Bincennite, although its use was not highly successful because of limitations in projectile size and other factors. During WW II, the Japanese were armed with 50-kg HCN bombs, and the United States had 500-kg bombs. More than 500,000 kg of HCN chemical weapons were produced during WWII by Japan, the United States, and the Soviet Union, but it is not known to what extent these weapons were used in that conflict (Way 1981).

Cyanides are widely distributed among common plants in the form of cyanogenic glycosides (Egekeze and Oehme 1980; Solomonson 1981; Way 1981; Biehl 1984; Homan 1987; Marrs and Ballantyne 1987). Their toxicity following ingestion is primarily related to the hydrolytic release of HCN. Ingestion of cyanogenic plants probably has accounted for most instances of cyanide exposure and toxicosis in man and range animals. Of chief agricultural importance among plants that accumulate large quantities of cyanogenic glycosides are the sorghums, Johnson grass, Sudan grass, corn, lima beans, flax, pits of stone fruits (cherry, apricot, peach), vetch, linseed, sweet potatoes, bamboo shoots, southern mock orange, millet, almonds, and cassava. Factors favoring cyanide build-up in cyanogenic plants include high nitrogen and low phosphorus in soils (Biehl 1984); the potential for high glycoside levels is greatest in immature and rapidly growing plants (Egekeze and Oehme 1980). At present, more than 28 different cyanoglycosides have been measured in about 1,000 species of higher plants (Leduc 1984). In cassava, for example, more than 90% of the cyanide is present as linamurin, a cyanogenic glycoside, and the remainder occurs as free (nonglycoside) cyanide (Gomez et al. 1983). Laetrile, a preparation made from apricot kernels, contains high levels of amygdalin, a cyanogenic glycoside that can be degraded in the gut to cyanide and benzaldehyde. Several cases of cyanide poisoning in humans have been reported from intake of laetrile, either orally or anally (Solomonson 1981; Homan 1987). Cyanide formation in higher plants and microorganisms can also occur with compounds other than cyanogenic glycosides, such as glycine, glyoxylate plus hydroxylamine, or histidine (Solomonson 1981; Vennesland et al. 1981b). In some cases, plants may contain cyanide residues resulting from fumigation with HCN (Way 1981).

Many species of plants, including some fungi, bacteria, algae, and higher plants, produce cyanide as a metabolic product (Leduc et al. 1982; Leduc 1984). Some species of soil bacteria suppress plant diseases caused by soilborne pathogens by producing metabolites with antibiotic activity. Certain strains of *Pseudomonas fluorescens,* a soil bacterium, suppress black root rot of tobacco caused by the fungus *Thielaviopsis basicola* by excreting several metabolites, including HCN (Voisard et al. 1989). A wide variety of bacteria and fungi can degrade cyanide compounds, and may be useful in the treatment of cyanide wastes (Towill et al. 1978). For example, several species of fungi known to be pathogens of cyanogenic plants can degrade cyanide by hydration to formamide; dried mycelia of these species are now sold commercially to detoxify cyanide in industrial wastes (Knowles 1988).

Anthropogenic sources of cyanide in the environment include industrial processes, laboratories, fumigation operations, cyanogenic drugs, fires, cigarette smoking, and chemical warfare operations (Marrs and Ballantyne 1987). Cyanides are present in many industrial wastewaters, especially those of electroplaters; manufacturers of paint, aluminum, and plastics; metal finishers; metallurgists; coal gasification processes; certain mine

operations; and petroleum refiners (Towill et al. 1978; Egekeze and Oehme 1980; Way 1981, 1984). Electroplaters are a major source. In the United States alone, electroplaters discharge about 9.7 million kg of cyanide wastes annually into the environment from 2,600 electroplating plants (Marrs and Ballantyne 1987). Paint residues annually contribute an additional 141,300 kg of cyanide wastes into the environment, and paint sludges 20,400 kg (Way 1981; Marrs and Ballantyne 1987). Cyanide can also originate from natural processes, such as cyanide production by bacteria, algae, and fungi, and from many terrestrial plants that release free HCN when their cellular structure is disrupted (Leduc 1981). Hospital wastewaters usually contain no detectable cyanide, but concentrations up to 64 µg CN^-/L have been measured after alkali chlorination treatment (Tatsumoto and Hattori 1988). It seems that various compounds common in hospital wastewaters will produce 15-25 µg CN^-/L after alkali chlorination; these compounds include hydantoin (an antiepilepsy agent) and related nitrogenous compounds, such as hydantonic acid, 5,5-diphenyl hydantoin, imidazole, and 2-imidazolidinone (Tatsumoto and Hattori 1988).

Free hydrogen cyanide occurs only rarely in nature because of its high reactivity. The gas is sometimes found in the atmosphere, however, as a result of emissions from the petrochemical industry, malfunctioning catalytic converters on automobiles, fumigation of ships and warehouses, incomplete combustion of nitrogen-containing materials, and from tobacco smoke (Towill et al. 1978; Way 1981, 1984). Hydrogen cyanide is known to be produced in fires involving nitrogen-containing polymers and is probably the most important narcotic fire product other than carbon monoxide (Purser et al. 1984). Cyanide-related fire deaths and injuries, as judged by elevated blood cyanide and thiocyanate concentrations, have been documented in airplanes, jails, and high-rises (Becker 1985; Ballantyne 1987b; Lundquist and Sorbo 1989). In a study of fire victims in Scotland, elevated blood cyanide levels were found in 78% of fatalities, and 31% had blood levels considered to be toxic (Purser et al. 1984). Major factors that influence HCN release include the chemical nature of the material, temperature, oxygen availability, and burning time (Ballantyne 1987b). Substantial quantities of free HCN and organic cyanides are known to be produced in fire settings involving horsehair, tobacco, wool, silk, and many synthetic polymers, such as polyurethane and polyacrylonitriles (Egekeze and Oehme 1980; Purser et al. 1984; Becker 1985; Ballantyne 1987b). Polyacrylonitrile, for example, is used in fabrics, upholstery covers, paddings, and clothing; about 50% of the mass of the polymer is theoretically available as HCN under thermal decomposition (Purser et al. 1984; Homan 1987).

Background Concentrations

The reactivity of HCN, and its ability to condense with itself and other compounds, was probably responsible for the prebiotic formation of the majority of biochemical compounds required for life (Marrs and Ballantyne 1987). Cyanide is now known to be present in a number of foodstuff and forage plants, as a metabolite of certain drugs, and in various industrial pollutants; it also may be formed by the combustion of cyanide-releasing substances, such as plastics in airplane fires and tobacco in smoking (Robinson et al. 1985). Hydrogen cyanide production may occur in hepatopancreas of mussels, *Mytilus edulis* (Vennesland et al. 1981b), in rat liver (Solomonson 1981), and in green and blue-green algae during nitrate metabolism (Leduc et al. 1982). Except for certain naturally occurring organic cyanide compounds in plants, it is uncommon to find cyanide in foods consumed in the United States (EPA 1980).

The cyanide anion is found in a variety of naturally occurring plant compounds as cyanogenic glycosides, glycosides, lathyrogenic compounds, indoleacetonitrile, and cyanopyridine alkaloids. Plants that contain cyanogenic glycosides are potentially poisonous because bruising or incomplete cooking can result in glycoside hydrolysis and release of HCN (Towill et al. 1978). Cyanide concentrations in cyanogenic plants are usually highest in leaves of young plants; levels drop rapidly after pollination (Biehl 1984). There are about 20 major cyanogenic glycosides, of which usually only one or two occur in any plant. They are synthesized from amino acids and sugars and are found in many economically important plants, such as sorghum, flax, lima bean, cassava, and many of the stone fruits (Table 2; Towill et al. 1978; Shaw 1986). Cassava contains linamurin and lotaustralin, whereas the main cyanogenic glycoside in cereals is dhurrin; consumption of foods containing toxic cyanogens (primarily cassava) has been associated with death or morbidity--on an acute basis--or goiter and tropical ataxic neuropathy on a chronic consumption basis (Okolie and Ugochukwu 1989). Cassava is a perennial shrub, native to the neotropics, grown for its tuberous starchy roots, and a traditional dietary staple of many indigenous populations in Amazonia, especially the Tukanoan Indians in northwestern Amazonia (Dufour 1988). Cassava is one of the few food plants in which the cyanide content may create toxic problems. All

varieties of cassava contain cyanogenic glycosides capable of liberating HCN, but amounts vary greatly depending on variety and environmental conditions. Bitter cultivars of cassava provide over 70% of the Tukanoan's food energy, appearing in the diet as bread, meal, a starch drink, and boiled cassava juice. The greatly elevated total cyanide content in bitter varieties (Table 2) may contain 5.1-13.4% of the total as the toxic free cyanide (Dufour 1988).

The production of HCN by animals is almost exclusively restricted to various arthropods: 7 of about 3,000 species of centipedes; 46 of 2,500 species of polydesmid millipedes; and 10 of 750,000 species of insects, including 3 species of beetles, 4 moths, and 3 butterflies (Duffey 1981). Millipedes--which are eaten frequently by toads and starlings--secrete cyanide for defensive purposes in repelling predators; in zygaenid moths, cyanide seems to be localized in eggs (Table 2; Duffey 1981).

Cyanide concentrations in fish from streams that were deliberately poisoned with cyanide ranged between 10 and 100 µg total cyanide per kilogram whole body fresh weight (FW; Wiley 1984). Total cyanide concentrations in gill tissues of salmonids under widely varying conditions of temperature, nominal water concentrations, and duration of exposure ranged from about 30 µg/kg FW to >7,000 µg/kg (Holden and Marsden 1964). Unpoisoned fish usually contained < 1 µg/kg FW in gills, although values up to 50 µg/kg occurred occasionally. Lowest cyanide concentrations in gills occurred at elevated (summer) water temperatures; at lower temperatures, survival was greater and residues were higher (Holden and Marsden 1964). Fish retrieved from cyanide-poisoned environments, dead or alive, can probably be consumed by humans because muscle cyanide residues were considered to be low (i.e., <1,000 mg/kg FW; Leduc 1984).

Cyanide pollution is likely to occur in many places, ranging from industrialized urban areas to gold mines in the western United States and Northwest Territories of Canada (Table 2). Cyanides are ubiquitous in industrial effluents, and their increasing generation from power plants and from the combustion of solid wastes is expected to result in elevated cyanide levels in air and water (Leduc 1984). However, data are scarce on background concentrations of cyanides in various nonbiological materials. In soils, for example, high concentrations are unusual and are nearly always the result of improper waste disposal (Towill et al. 1978). Cyanides in soils are not absorbed or retained; under aerobic conditions, microbial metabolism rapidly degrades cyanides to carbon dioxide and ammonia; under anaerobic conditions, cyanides are converted by bacteria to gaseous nitrogen compounds that escape to the atmosphere (Towill et al. 1978). Heat treatment wastes from metal processing operations may contain up to 200 g CN/kg, mostly as NaCN, and are frequently hauled to landfills for disposal (Lagas et al. 1982). The presence of cyanide in landfill waste is potentially hazardous because of the possibility that cyanide may leach to soil and groundwater, release HCN, and disturb natural microbiological degradation of organic materials. Measurements at landfills in England and the Netherlands showed total cyanide levels up to 560 g/kg in soil and 12 µg/L in groundwater (Lagas et al. 1982). However, 7-month-long experimental studies of cyanide in heat treatment wastes in landfills showed that between 72 and 82% of the cyanide was converted, mostly to ammonium and organic nitrogen compounds; between 4 and 22% of the cyanide leached as free or complex cyanide; and up to 11% remained in the landfill (Lagas et al. 1982).

Table 2. Background concentrations of cyanide in selected living resources and nonbiological materials. Values are in milligrams total cyanide per kilogram fresh weight or milligrams per liter.

Environmental compartment	Concentration[a] (mg/kg or mg/L)	Reference[b]
Biological		
Cyanogenic plants		
Bamboo (*Bambusa, Arundinaria, Dendrocalamus*)		
Tip	Max. 8,000	1
Stem	Max. 3,000	1
Stargrass, *Cynodon plectostachyus*, whole	180	1
Rose family, *Malus* spp., *Pyrus* spp.	Max. 200	2
Cassava, *Manihot esculenta*		
Bitter varieties		
Leaves	347–1,000	3, 4

Roots	327–550	1, 4
Dried roots	95–2,450	1, 3, 4
Stem	1,130	1
Mash	162	5
Bark		
Total cyanide	1,351	6
Free cyanide	102	6
Peel		
Total cyanide	1,390	6
Free cyanide	255	6
Pulp		
Total cyanide	810	6
Free cyanide	53	6
Sweet varieties		
Leaves	377–500	3, 4
Roots	138	4
Dried roots	46–<100	3, 4
Mash	81	5
Lima beans, *Phaseolus lunatus*		
United States	100–170	1
Burma	2,100	1
Puerto Rico	3,000	1
Java	3,120	1
Almond, *Prunus amygdalus*, nut		
Bitter	(280–2,500)	1
Spicy	(86–98)	1
Sweet	(22–54)	1
Seeds, 4 species, Nigeria, whole, frequently consumed by humans		
Phaseolus sp.	(381–1,093)	7
Vigna sp.	(285–1,223)	7
Cajanus sp.	(208–953)	7
Canavalia sp.	(285–953)	7
Sorghum, *Sorghum spp.*, young plant, whole	Max. 2,500	1
Cyanogenic arthropods		
Millipede, *Apheloria corrugata*, whole	428	8
Millipede, *Apheloria kleinpeteri*, whole	18	8
Zygaenid moth, *Zygaena filipendulae*, whole	668	8
Mammals		
Humans, *Homo sapiens*		
Blood		
Normal	<0.2	9
Afflicted with Leber's optic atrophy	1.4	9
Plasma		
Nonsmokers	0.05; Max. 0.11	10
Smokers	0.075; Max. 0.3	10
Nonbiological		
Air		
Automobile exhaust		
Adverse conditions	Max. 10.0	1
Equipped with catalytic convertor	1.1	1
Sewage sludge		

From publicly owned treatment works, United States	749[c]	18
Water, uncontaminated		
Rural watersheds	0.003	11,12
Industrial areas	0.02	11, 12
Small watersheds, covered with grasslands and forest, uninhabited by humans	0.0007–0.002; Max. 0.005	12
Western and central Canada, 11 rivers, 1974–77	Max. 0.006	12
U.S. water supplies, 2,595 samples nationwide	0.0009; Max. 0.008	1, 13
U.K. water supplies	<0.05; Max. 0.1	1
Wastewaters/runoff		
Electroplaters		
Total cyanide	0.2; Max. 3.0	14, 15
Dissociable cyanide	0.07	15
Complex cyanide	0.2	15
Thiocyanate	0.02	15
Plating wastewater		
Before treatment with alkaline chlorination	0.18	16
After treatment	0.005	16
Road salt dock		
Total cyanide	25.6	15
Dissociable cyanide	2.9	15
Complex cyanide	23.1	15
Thiocyanate	0.0	15
Steel industry		
Plating baths	72 (9–115)	1, 14
Coke oven liquor	6 (0–8)	1
Oil refineries		
Total cyanide	0.01; Max. 4.0	14, 15
Dissociable cyanide	0.0	15
Complex cyanide	0.01	15
Thiocyanate	2.2	15
Coking operations		
Total cyanide	2.1	15
Dissociable cyanide	0.3	15
Complex cyanide	0.8	15
Thiocyanate	23.6	15
Hospital wastewaters		
Before alkaline chlorination	ND	17
After treatment	0.06	17
Gold mills, Canada	0.3–26.5	14
Gold mine cyanide extraction leach ponds, California, Nevada, and Arizona	Usually 200–300, frequently 700, occasionally 9,000	19
Wastewater treatment plants,		
Chicago		
Treated effluent		
Total cyanide	0.005–0.03	15
Dissociable cyanide	0.003–0.007	15
Complex cyanid	0.002–0.02	15
Thiocyanate	0.006–0.03	15
Untreated wastewater		
Total cyanide	0.02–0.06	15

Dissociable cyanide	0.004–0.05	15
Complex cyanide	0.02–0.08	15
Thiocyanate	0.03–0.27	15
Sludge		
Total cyanide	0.49–3.79	15
Dissociable cyanide	0.06–0.44	15
Complex cyanide	0.43–5.4	15
Thiocyanate	0.2–0.9	15

[a]Concentrations are shown as means, range (in parentheses), and maximum (Max.).

[b]1, Towill et al. 1978; 2, Shaw 1986; 3, Gomez et al. 1983; 4, Casadei et al. 1984; 5, Ukhun and Dibie 1989; 6, Dufour 1988; 7, Okolie and Ugochukwu 1989; 8, Duffey 1981; 9, Berninger et al. 1989; 10, Egekeze and Oehme 1980; 11, Leduc 1981; 12, Leduc 1984; 13, EPA 190; 14, Leduc et al. 1982; 15, Kelada 1989; 16, Nonomura and Hobo 1989; 17, Vennesland et al. 1981a; 18, Beyer 1990; 19, Clark and Hothem 1991.

[c]Concentration is in milligrams per kilogram dry weight.

Hydrogen cyanide (HCN) is a common industrial pollutant and frequently occurs in water at concentrations between 0.1 and several milligrams per liter of free HCN (Leduc 1978; Leduc et al. 1982). Total cyanides is the most often cited measurement in aqueous solutions, owing to limitations in analytical methodologies (Leduc et al. 1982). Cyanides have been identified in fresh waters of rural and wilderness areas in Canada and Germany. Concentrations ranging between 30 and 60 µg total cyanides per liter seem related to runoff, with cyanide peaks more frequent in fall and winter during periods of minimal runoff (Leduc et al. 1982). In larger rivers, cyanide was low in winter owing to dilution by high runoff, but peaked in summer because of cyanide production by plants (Leduc 1984). Cyanides do not seem to persist in aquatic environments. In small, cold oligotrophic lakes treated with 1 mg NaCN/L, acute toxicity was negligible within 40 days. In warm shallow ponds, toxicity disappeared within 4 days after application of 1 mg NaCN/L. In rivers and streams, toxicity rapidly disappeared on dilution (Leduc 1984). Cyanide was not detectable in water and sediments of Yellowknife Bay, Canada, between 1974 and 1976, although the bay receives 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 (EPA 1980). An alkaline pH favors oxidation by chlorine, and an acidic pH favors volatilization of HCN into the atmosphere (EPA 1980).

Persistence in Water, Soil, and Air

In water, cyanides occur as free hydrocyanic acid, simple cyanides, easily degradable complex cyanides such as $Zn(CN)_2$, and sparingly decomposable complex cyanides of iron and cobalt; complex nickel and copper cyanides are intermediate between the easily decomposable and sparingly degradable compounds (Towill et al. 1978). Cyanide has relatively low persistence in surface waters under normal conditions but may persist for extended periods in groundwater (Way 1981). Volatilization is the dominant mechanism for removal of free cyanide from concentrated solutions and is most effective under conditions of high temperatures, high dissolved oxygen levels, and at increased concentrations of atmospheric carbon dioxide (Leduc et al. 1982; Simovic and Snodgrass 1985). Loss of simple cyanides from the water column is primarily through sedimentation, microbial degradation, and volatilization (Leduc et al. 1982; Marrs and Ballantyne 1987). Water-soluble strong complexes, such as ferricyanides and ferrocyanides, do not release free cyanide unless exposed to ultraviolet light. Thus, sunlight may lead to cyanide formation in wastes containing iron-cyanide complexes (Towill et al. 1978; Leduc et al. 1982; Simovic and Snodgrass 1985; Marrs and Ballantyne 1987).

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). 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; EPA 1980; Way 1981; Marrs and Ballantyne 1987). In the case of Canadian gold-mining operations, the primary treatment for cyanide removal is to retain gold 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 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 (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.

Volatile cyanides occur only occasionally in the atmosphere, due largely to emissions from plating plants, fumigation, and other special operations (Towill et al. 1978). Under normal conditions cyanide has relatively low persistence in air, usually between 30 days and 1 year (Way 1981), although some atmospheric HCN may persist for up to 11 years (Marrs and Ballantyne 1987). Data are lacking on the distribution and transformation of cyanide in the atmosphere (Towill et al. 1978) and should be acquired.

Lethal and Sublethal Effects

Terrestrial Flora and Invertebrates

Bacteria exposed to cyanide may exhibit decreased growth, altered cell morphology, decreased motility, mutagenicity, and altered respiration (Towill et al. 1978). Mixed microbial populations capable of metabolizing cyanide and not previously exposed to cyanide were adversely affected at 0.3 mg HCN/kg; however, these populations can become acclimatized to cyanide and can then degrade wastes with higher cyanide concentrations (Towill et al. 1978). Acclimatized populations in activated sewage sludge can often completely convert nitriles to ammonia, sometimes at concentrations as high as 60 mg total cyanides per kilogram (Towill et al. 1978). Cyanide can be degraded by various pathways to yield a variety of products, including carbon dioxide, ammonia, beta-cyanoalanine, and formamide (Knowles 1988). Several species of fungi can accumulate and metabolize cyanide, but the products of cyanide metabolism vary. For example, carbon dioxide and ammonia are formed as end products by *Fusarium solani*, whereas alpha-amino butyronitrile is a major cyanide metabolite of *Rhizoctonia solani*(Towill et al. 1978). Significant amounts of cyanide are formed as secondary metabolites by many species of fungi and some bacteria by decarboxylation of glycine (Knowles 1988). Rhizobacteria may suppress plant growth in soil through cyanide production. In one case volatile metabolites--including cyanide--from fluorescent pseudomonad soil bacteria prevented root growth in seedlings of lettuce, *Lactuca sativa* (Alstrom and Burns 1989). Not all cyanogenic isolates inhibited plant growth. Some strains promoted growth in lettuce and beans by 41-64% in 4 weeks versus 49-53% growth reduction by inhibitory strains (Alstrom and Burns 1989).

In higher plants, elevated cyanide concentrations inhibited respiration (through iron complexation in cytochrome oxidase) and ATP production and other processes dependent on ATP, such as ion uptake and phloem translocation, eventually leading to death (Towill et al. 1978). Cyanide produces chromosomal aberrations in some plants, but the mode of action is unknown (Towill et al. 1978). At lower concentrations, effects include inhibition of germination and growth, but cyanide sometimes enhances seed germination by stimulating the pentose phosphate pathway and inhibiting catalase (Towill et al. 1978; Solomonson 1981). The detoxification mechanism of cyanide is mediated by rhodanese. This enzyme is widely distributed in plants (Solomonson 1981; Leduc 1984). The rate of production and release of cyanide by plants to the environment through death and decomposition is unknown (Towill et al. 1978).

Free cyanide is not found in intact plant cells. Many species of plants, such as cassava, sorghum, flax, cherries, almonds, and beans, contain cyanogenic glycosides that release HCN when hydrolyzed (Towill et al. 1978). Cyanide poisoning of livestock by forage sorghums, such as Sudan grass and various hybrid cultivars, is well known (Cade and Rubira 1982) and has led to the development of several variations of sorghums that have a reduced capability of producing cyanide poisoning (Egekeze and Oehme 1980). Cyanogenesis has an

important role in plant defense against predatory herbivores. This herbivore-plant interaction is not simple; the degree of selectivity by herbivores varies among individuals and by differences in hunger and previous diet (Jones 1988).

Cyanide metabolism in higher plants involves amino acids, N-hydroxyamino acids, aldoximes, nitriles, and cyanohydrins (Halkier et al. 1988). Cyanide is a coproduct of ethylene synthesis in higher plants. The increase in ethylene production that occurs during the senescence of certain flowers and the ripening of fruits is accompanied by a rise in beta-cyanoalanine activity; activity of this enzyme correlates closely with that of ACC (1-aminocyclopropane-l-carboxylic acid) oxidase, the last enzyme in the ethylene pathway. Manning (1988) suggested that ACC oxidase reacts with various amino acids to liberate cyanide. Cyanide added to isolated castorbean (*Ricinus communis*) mitochondria significantly enhanced the rate and amount of protein synthesis. Cyanide stimulated mitochondrial protein synthesis in a dose-dependent manner, with an optimal stimulation of over 2 times at 26 µg/L, but at this concentration mitochondrial respiration was inhibited by 90% (Kaderbhai et al. 1989). Cyanide is a weak competitive inhibitor of green bean *(Phaseolus vul*garis) lipoxygenase, an enzyme that catalyzes the formation of hydroperoxides from polyunsaturated fatty acids (Adams 1989). Because degradation of hydroperoxides causes unacceptable changes in bean flavor and color, compounds that inhibit lipoxygenase may enjoy wide application in the frozen vegetable industry (Adams 1989). Corn seedlings from cold-resistant cultivars were more resistant to 65 mg KCN/L at low temperatures (13°C) than were seedlings from cold-susceptible cultivars (25°C), as judged by respiratory activity of mitochondria (Van De Venter 1985). Results suggest that cyanide-resistant respiration may play a role in cold resistance in maize seedlings, although more evidence is needed to demonstrate that cold-resistant plants actually use their greater potential for alternative respiration at low temperatures (Van De Venter 1985).

The cyanogenic system comprising cyanogenic glycosides, cyanohydrins, betaglucosidases, and nitrile lyases is widespread in plants, but also occurs in several species of arthropods, including the tiger beetle (*Megacephala virginica*), leaf beetle (*Paropsis atomaria*), zygaenid moths, and certain butterflies (Nahrstedt 1988). In *Zygaena trifolii*, cyanide compounds seem to function as protection against predators (Nahrstedt 1988). Defensive secretions of cyanide have also been reported in polydesmid millipedes, and these organisms seem to be more tolerant than other species when placed in killing jars containing HCN (Towill et al. 1978). In a millipede (*Apheloria* sp.), cyanide is generated in a two-compartment organ by hydrolysis of mandelonitrile; cyanide generation occurs outside the gland when the components of the two compartments are mixed during ejection (Towill et al. 1978).

Highly toxic substances, such as cyanides, are sometimes feeding cues and stimulants for specialized insects. For example, instar larvae of the southern armyworm (*Spodoptera eridania*) strongly prefer cyanogenic foods, such as foliage of the lima bean, a plant with comparatively elevated cyanide content--up to 31 mg/kg in some varieties--in the form of linamurin (Brattsten et al. 1983). Feeding was stimulated in southern armyworms at dietary levels up to 508 mg KCN/kg (208 mg HCN/kg) for first to fourth instar larval stages, and between 1,000 and 10,000 mg KCN/kg diet for fifth and sixth instar larvae (Brattsten et al. 1983). Sixth instar larvae preexposed to diets containing 5,000 mg KCN/kg showed no adverse affects at dietary levels of 10,000 mg KCN/kg; however, previously unexposed larvae showed reversible signs of poisoning at 10,000 mg/kg diet, including complete inhibition of oviposition and 83% reduction in adult emergence (Brattsten et al. 1983). Experimental studies with southern armyworm larvae and thiocyanate--one of the in vivo cyanide metabolites--showed that 5,000 mg thiocyanate per kilogram diet reduced pupation by 77%, completely inhibited oviposition, and reduced adult emergence by 80% (Brattsten et al. 1983), strongly suggesting that thiocyanate poisoning is the primary effect of high dietary cyanide levels in southern armyworms.

Resistant species, such as southern armyworms, require injected doses up to 800 mg KCN/kg BW (332 mg HCN/kg BW) or diets of 3,600 mg KCN/kg for 50% mortality (Brattsten et al. 1983), but data are scarce for other terrestrial invertebrates. Exposure to 8 mg HCN/L air inhibits respiration in the granary weevil (*Sitophilus granarius*) within 15 min and kills 50% in 4 h; some weevils recover after cessation of 4-h exposure (Towill et al. 1978).

Aquatic Organisms

Numerous accidental spills of sodium cyanide or potassium cyanide into rivers and streams have resulted in massive kills of fishes, amphibians, aquatic insects, and aquatic vegetation; sources of poisonings were storage reservoirs of concentrated solutions, overturned rail tank cars, or discharge of substances generating free HCN

in the water from hydrolysis or decomposition (Leduc 1984). Data on the recovery of poisoned ecosystems are scarce. In one case, a large amount of cyanide-containing slag entered a stream from the reservoir of a Japanese gold mine as a result of an earthquake (Yasuno et al. 1981). The slag covered the streambed for about 10 km from the point of rupture, killing all stream biota; cyanide was detected in the water column for only 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).

Fish were the most sensitive aquatic organisms tested under controlled conditions. Significant adverse nonlethal effects, including reduced swimming performance and inhibited reproduction, were observed in the range of 5.0-7.2 µg free cyanide per liter; deaths were recorded for most species between 20 and 76 µg/L (Table 3). Among invertebrates, adverse nonlethal effects were documented between 18 and 43 µg/L, and lethal effects between 30 and 100 µg/L--although some deaths were recorded in the range 3-7 µg/L for the amphipod *Gammarus pulex* (Table 3). Algae and macrophytes were comparatively tolerant; adverse effects were reported at >160 µg free cyanide per liter (Table 3).

Table 3. Cyanide effects on selected species of aquatic organisms. All concentrations are shown as micrograms of hydrogen cyanide per liter (ppb) of medium at start unless indicated otherwise.

Species, concentration, and other variables	Effects	Reference[a]
Algae and macrophytes		
Alga, *Chlorella* sp.		
7,300	Inhibition of photosynthesis	3
30,000	Enzyme inhibition	2
Water hyacinth, *Eichhornia crassipes*		
300,000	Nonphytotoxic in 72 h; plants contained total cyanide of 6.7 g/kg dry weight (DW), equivalent to bioconcentration factor (BCF) of x22	5
Freshwater aquatic plants, nine species, 65,000, 30-min exposure	No effect on respiratory oxygen uptake in six species of angiosperms (*Myriophyllum* sp., *Potamogeton* spp., *Elodea* sp., *Ruppia* sp., *Cabomba* sp.); some effect on two species of bryophytes (*Rhynchostegium riparioides*, *Fontinalis antipyretica*) and one species of alga (*Cladophora glomerata*)	4
Alga, *Microcystis aeruginosa*		
7,990	90% kill	2
Alga, *Prototheca zopfi*		
3,000	Inhibition of respiration	2
Alga, *Scenedesmus quadricauda*		
160, as CN^-	Toxic	1
Invertebrates		
Copepod, *Acartia clausi*		
30	LC50 (96 h)	2
Isopod, *Asellus communis*		
29–40	MATC[b]	2, 8
1,834	LC50 (11 days)	
Oyster, *Crassostrea* sp.		
150	Motor activity suppressed after 10 min	2

Daphnid, *Daphnia magna*		
160	LC50 (96 h)	10
Daphnid, *Daphnia pulex*		
83	LC50 (96 h)	2
Amphipod, *Gammarus pseudolimnaeus*		
16–21	MATC[b]	8
58	LC50 (96 h) at 20º C	8
184	LC50 (96 h) at 5.2º C	8
Amphipod, *Gammarus pulex*		
3	LC50 (15 h); 50% dead in 14 days after exposure for 5 h	6
7.5	LC50 (9h); exposure for 66 min results in 50% mortality 14 days after exposure	6
15	LC50 (6 h); exposure for 45 min causes 50% mortality 14 days after exposure	6
75	LC50 (3 h); exposure for 18 min results in 50% kill 14 days after exposure	6
Mussel, *Mytilus edulis*		
18	After exposure for 14 days growth was reduced and uptake of glycine was inhibited	9
100	LC20 (14 days)	9
Mysid shrimp, *Mysidopsis bahia*		
11, 20, 43, or 70	Life-cycle (29 days) exposure produced adverse effects on survival at 70 µg/L, and on reproduction at 43 µg/L; no measurable effects at lower doses of 11 and 20 µg/L	7
93–113	LC50 (96 h)	2, 7
Snail, *Physa heterostropha*		
432	LC50 (96 h)	3, 10
Fiddler crab, *Uca tangeri*		
Isolated perfused gills subjected to 26,000 CN^-/L, as KCN	Inhibited sodium chloride absorption across gill epithelium; effect reversible if exposure <5 min and nonreversible if >30 min. Salt absorption effect regulated by $(Na^+ + K^+)$ ATPase	11
Fish		
Brown bullhead, *Ictalurus nebulosus*		
Subjected to steadily increasing concentration of waterborne cyanide over a 9-h period: 200 at 1 h, 600 at 3 h, 1,000 at 5 h, and 1,800 at 9 h	Increased heart beat rate at lower concentrations and decreased rate at higher concentrations; hyperventilation in first 3 h followed by decrease in ventilation rate; oxygen consumption paralleled changes in heart and ventilatory rates. Death in 9 h	21
Longnose gar, *Lepisosteus osseus*		
12 µg CN^-/kg BW, as sodium cyanide, equivalent to 10.7 µg CN or 20 µg NaCN, single injection	Hypoxic response and bradycardia; effects appear earlier when administered into the ventral aorta or conus than into the dorsal aorta	27
Bluegill, *Lepomis macrochirus*		
5.0	Inhibited spawning following chronic exposure	22
5.2	Complete inhibition of spawning after exposure for 57–289 days	2, 8
9.3–19.8	MATC[b]	2

19.4	Reduced survival of fry in 57-day exposure which began with eggs	8
50	Tolerated concentration at higher temperatures, but no reproduction	8
56–227	LC50 (96 h) for juveniles	8, 22
109–218	LC50 (96 h) for fry	8
232–365	LC50 (96 h) for eggs	22
535–690	LC50 (96 h) for eggs at hatching	8
Largemouth bass, *Micropterus salmoides*		
101	LC50 (96 h) for juveniles	8
Cutthroat trout, *Oncorhynchus clarki*		
1,000 for 20 min	All fish recovered within 12 min and fed and grew normally during the next 6 months	31
Coho salmon, *Oncorhynchus kisutch*		
7.0	Reduction of 50% in swimming performance during 8-day exposure	13
10	Swimming speed reduced after exposure for 2 h	2
Rainbow trout, *Oncorhynchus mykiss*		
0.1 or 1.0	No effect on sperm motility or on fertilization rate at lower dose; some effect on sperm motility at higher dose	12
5.0	Reduction of 50% in swimming performance in 20-day exposure	13
10	No effect on growth during 20-day exposure at 6º C	13
10	Increased respiration rate in 4 days, growth reduction and liver damage in 9 days, abnormal oocyte development and reduced spermatogonia production in 18–20 days	2
10, 20, or 30 for 7 days, sexually mature females	Exposure to 10 or 20 µg/L caused a reduction in serum calcium to levels insufficient for the production of exogenous yolk; this was not observed in the 30 µg/L group	14
10, 20, or 30 for 18 days, juveniles	Degenerative necrosis of liver hepatocytes at all concentrations in a dose-dependent pattern. Severe initial growth repression at all concentrations followed by a significant increase, but growth remained depressed 40% and 95% in the 20 and 30 µg/L groups, respectively, at 18 days	15
10 or 20, exposure for 20 days during midsummer, sexually maturing females	Both concentrations resulted in abnormal oocytes, delayed development, and significantly reduced the number of eggs for spawning	16
15	No effect on growth during 20-day exposure at 12º C	13
18	No deaths in 96 h at 6º C	13
20	65% reduction in weight gain after 21 days	2
28	LC50 (96 h) at 6º C	10, 13, 17
30	No effect on growth during 20-day exposure at 18º C	13
32	No deaths in 96 h at 12º C	13
42	LC50 (96 h) at 12º C	13
43	LC50 (96 h) for nonexercised juveniles	18

	during winter	
46–75	LC50 (96 h) for juveniles	8, 19
52	LC50 (96 h) for exercised juveniles during winter	18
60	No deaths in 96 h at 18º C	13
68	LC50 (96 h) at 18º C	10, 13, 17
96	LC50 (144 h)	20
Subjected to steadily increasing concentrations of waterborne cyanide: 0 at start, 200 at 1 h, 600 at 3 h, 1,000 at 5 h, and 1,800 at 9 h	Reduction in heart rate, hyperventilation, increased oxygen consumption, death in 9 h	21
Chinook salmon, *Oncorhynchus tshawytscha*		
10	After 64 days, increased growth rate and production when compared to controls	13
20	Growth reduced 27% after exposure for 64 days	2
Yellow perch, *Perca flavescens*		
76–108	LC50 (96 h) for juveniles	8, 22
288–>389	LC50 (96 h) for eggs and fry	8, 22
Fathead minnow, *Pimephales promelas*		
12.9–19.6	MATC[b]	8, 22
18–58	Reduction in RNA content in larva in 96 h at 18–36 µg HCN/L, and in DNA and protein at 18–58 µg/L	28
19	Egg reduction of 59% after exposure for 265 days at 25º C	13
35	Reduction in growth rate during chronic exposure	5
44	Hatching reduced 83% after chronic exposure	13
47	Growth reduction in 30 days	28
58	Toxicosis occurred in yolk-sac larvae within 24 h as judged by significant reductions in content of RNA and protein; however, effects were not measurable in 96 h suggesting development of partial tolerance	29
>61	Adverse effects on growth and survival during lifetime exposure	13
82–113	LC50 (96 h) for fry at 25º C	8
83–137	LC50 (96 h) for juveniles	8, 22
99	LC50 (96 h) for fry at 20º C	8
107	Reduced survival in 96 h	28
121–202	LC50 (96 h) for eggs at 25º C	8
121–352	LC50 (96 h) for eggs; more toxic at low dissolved oxygen	22
122	LC50 (96 h) for fry at 15º C	8
Mixture of NaCN plus $CdSO_4$, equivalent to 170 µg CN/L	LC50 (96 h) for adults	30
Mixture of NaCN plus $ZnSO_4$, equivalent to 180 µg CN/L	LC50 (96 h) for adults	30
230, as NaCN	LC50 (96 h) for adults	30
273	LC50 (96 h) for eggs at 20º C	8
352	LC50 (96 h) for eggs at 15º C	8
Mixture of NaCN plus $NiSO_4$, equivalent to 650 µg CN/L	LC50 (96 h) for adults	8
Black crappie, *Pomoxis nigromaculatus*		
60	Some deaths in <24 h	3

101	LC50 (96 h) for juveniles	8
Plainfin midshipman, *Porichthys notatus*		
Isolated photophores exposed to 2,600, as KCN	Maximal luminescence induced by KCN; effect inhibited by d-glucose, d-glyceraldehyde 3-phosphate, and 3-phosphoglycerate	32
Atlantic salmon, *Salmo salar*		
5.0 for 12 days, adult females	Decline in plasma and gonad vitellogenin levels	23
10	Abnormal embryonic development after 58-day exposure	2
10, 80, or 100; newly fertilized eggs continually exposed for 5 months to end of sac-fry stage	Hatching delayed 6–9 days at 80 and 100 µg/L. Hatching success reduced 15% to 40% at all test concentrations, but no measurable effects on growth or survival after hatching. Abnormalities (mostly defects of eye, mouth, vertebral column) were 6% at 10 µg/L, and 19% at 100 µg/L	24
24	LC50 (24 h) at dissolved oxygen of 3.5 mg/L	25
73	LC50 (24 h) at dissolved oxygen of 10 mg/L	25
5,000, 10,000, 25,000, 50,000, or 125,000 for 30 min	Total cyanide residues in gills ranged from 1.0 to 6.6 mg/kg fresh weight (FW) in a dose dependent manner	26
50,000 for 10, 15, 20, 25, or 30 min	Residues in gills, in mg total CN/kg FW, ranged from 1.3 (10 and 15 min) to 1.9 (15 and 20 min) to 4.5 (30 min)	26
Brown trout, *Salmo trutta*		
90	LC50 (96 h)	10
5,000, 10,000, 25,000, 50,000, 75,000, or 100,000, as CN- for 30 min	Residues in gills ranged in a dose-dependent manner from 0.6 mg CN/kg FW in the 5 mg/L group to 3.4 mg/kg FW in the 100 mg/L group	26
50,000 for 10, 15, 20, or 25 min	Residues in tissues, in mg/kg FW, ranged from 0.7 to 1.8 in gill, 0.6 to 2.3 in brain, and 1.3 to 2.5 in liver; concentrations were directly related to length of exposure	26
Brook trout, *Salvelinus fontinalis*		
5.0	Reduction of 50% in swimming performance in 29-day exposure	13
5.7–11.2	MATC[b]	8, 22
10	75% reduction in swimming endurance after exposure for 26 min	2
10–50	Swimming ability reduced 98% after exposure for 29 days	20
11	Continuous exposure of mature females for 144 days before spawning resulted in 50% reduction in number of eggs produced and 15% reduction in egg viability; however, 90 days after hatch trout were 18% heavier and 10% longer than controls	13
25	Inhibited oxygen intake after 5 h	2
33	Adverse effects on juvenile growth rate during exposure for 90 days	2, 8
56–112	LC50 (96 h) range for swimup fry and juveniles	8, 22
108–518	LC50 (96 h) for sac-fry	8, 22
>212	LC50 (96 h) for eggs	8, 22

[a]1, Towill et al. 1978; 2, EPA 1980; 3, EPA 1973; 4, Azcon-Bieto et al. 1987; 5, Low and Lee 1981; 6, Abel and Garner 1986; 7, Lussier et al. 1985; 8, Smith et al. 1979; 9, Thompson 1984; 10, Leduc et al. 1982; 11, Drews and Graszynski 1987; 12, Billard and Roubaud 1985; 13, Leduc 1984; 14, Da Costa and Ruby 1984; 15, Dixon and Leduc 1981; 16, Lesniak and Ruby 1982; 17, Kovacs and Leduc 1982b; 18, McGeachy and Leduc 1988; 19, Marking et al. 1984; 20, Ballantyne 1987a; 21, Sawyer and Heath 1988; 22, Smith et al. 1978; 23, Ruby et al. 1987; 24, Leduc 1978; 25, Alabaster et al. 1983; 26, Holden and Marsden 1964; 27, Smatresk et al. 1986; 28, Barron and Adelman 1984; 29, Barron and Adelman 1985; 30, Doudoroff 1956; 31, Wiley 1984; 32, Rees and Baguet 1989.

[b]Maximum acceptable toxicant concentration. Lower value in each pair indicates highest concentration tested producing no measurable effect on growth, survival, reproduction, or metabolism during chronic exposure; higher value indicates lowest concentration tested producing a measurable effect.

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 (EPA 1980). Water hyacinth *(Eichhornia crassipes)* can survive for at least 72 h in nutrient solution containing up to 300 mg CN/L and can accumulate up to 6.7 g/kg dry weight (DW) plant material. On this basis, 1 ha of water hyacinths has the potential to absorb 56.8 kg of cyanide in 72 h, and this property may be useful in reducing the level of CN in untreated wastewater from various electroplating factories, where concentrations generally exceed 200 mg CN/L (Low and Lee 1981). Cyanide may also affect plant community structure. Some algae, for example, metabolized CN at water concentrations <1 mg/L, but at concentrations of 1-10 mg/L, algal activity was inhibited, leaving a biota dominated by *Actinomycetes*--a filamentous bacterium (Knocke 1981).

Cyanide adversely affects fish reproduction by reducing 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 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 to as little as 10 µg HCN/L for 12 days during the onset of the reproductive cycle caused a reduction in plasma vitellogenin levels and a reduction in ovary weight. The loss of vitellogenin in the plasma would remove a major source of yolk (Ruby et al. 1986). Reproductive impairment in adult bluegills (*Lepomis macrochirus*) has been reported following exposure to 5.2 µg CN/L for 289 days (EPA 1980). Fertilized fish eggs are usually resistant to cyanide prior to blastula formation, but delayed effects occur at 60 to 100 µg HCN/L, including birth defects and reduced survival of embryos and newly hatched larvae (Leduc et al. 1982). Concentrations as low as 10 µg HCN/L caused developmental abnormalities in embryos of Atlantic salmon 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).

Other adverse effects of cyanide on fish include delayed mortality, pathology, impaired swimming ability and relative performance, susceptibility to predation, disrupted respiration, osmoregulatory disturbances, and altered growth patterns. Free cyanide concentrations between 50 and 200 µg/L were fatal to the more-sensitive fish species over time, and concentrations >200 µg/L were rapidly lethal to most species of fish (EPA 1980). Cyanide- induced pathology in fish includes subcutaneous hemorrhaging, liver necrosis, and hepatic damage. Exposure of fish for 9 days to 10 µg HCN/L 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 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 can rapidly and irreversibly impair the swimming ability of salmoneds in well-aerated water (Doudoroff 1976). Osmoregulatory disturbances recorded at 10 µg HCN/L may affect migratory patterns, feeding, and predator avoidance (Leduc et al. 1982; Leduc 1984). In general, fish experience a significant reduction in relative performance (based on osmoregulation, growth, swimming, and spermatogenesis) at 10 µg HCN/L, and although fish can survive indefinitely at 30 µg HCN/L in the laboratory, the different physiological requirements necessary to survive in nature could not be met (Leduc 1978, 1981; Leduc et al. 1982; Figure). Increased predation by green sunfish (*Lepomis cyanellus*) on fathead minnows *(Pimephales promelas)* was noted at sublethal concentrations of HCN, but it was uncertain if fatheads became easier prey or if green sunfish had greater appetites (Smith et al. 1979).

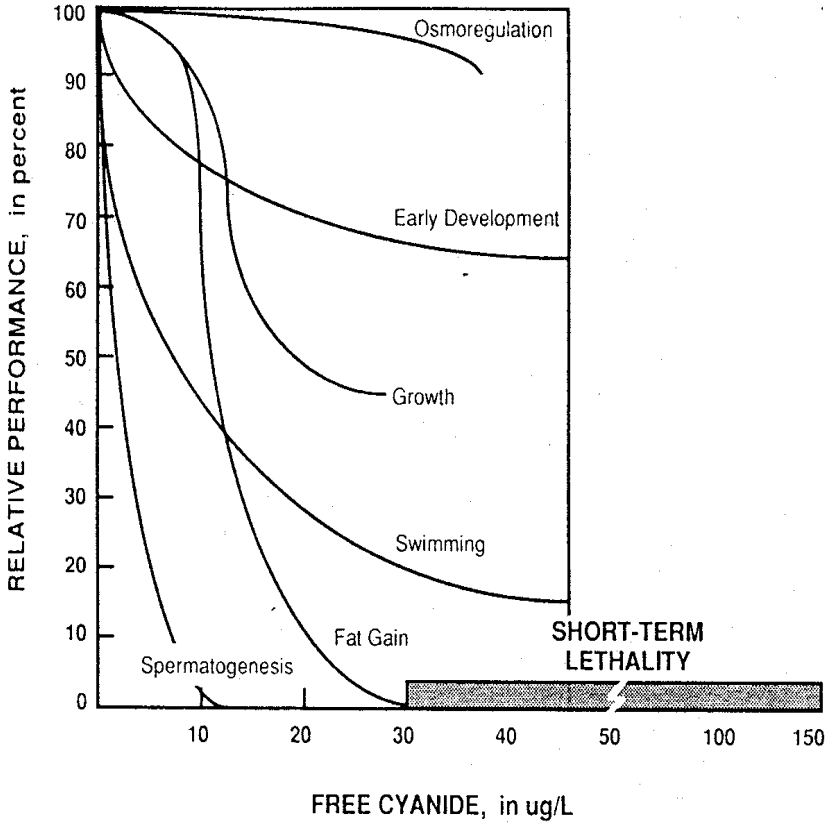

Figure. Summary of lethal and sublethal effects of free cyanide on freshwater fish. Modified from Leduc et al. (1982).

Sodium cyanide has stimulatory effects on oxygen-sensitive receptors in lungfish, amphibians, reptiles, birds, and mammals (Smatresk 1986). Facultative and aquatic air breathers appear to rely on air breathing when external chemoreceptors are stimulated, whereas obligate air-breathing fish are more responsive to internal stimuli (Smatresk 1986). Gill ventilation frequency of longnose gar (*Lepisosteus osseus*), for example, was little affected by external cyanide application, but responded strongly when cyanide was administered internally by injection (Smatresk 1986). Cyanide, like many other chemicals, can stimulate growth of fish during exposure to low sublethal levels. This phenomenon, referred to as hormesis, is little understood and warrants additional research (Leduc 1984).

The observed toxicity to aquatic life of simple and complex cyanides was attributed almost entirely to molecular (undissociated) HCN derived from ionization, dissociation, and photodecomposition of cyanide-containing compounds. The toxicity of the cyanide ion, CN^-, which is a minor component of free cyanide (HCN + CN^-) in waters that are not exceptionally alkaline is of little importance (Doudoroff 1976; Towill et al. 1978; Smith et al. 1979; EPA 1980). The acute toxicity of stable silver cyanide and cuprocyanide complex anions is much less than that of molecular HCN, but is nevertheless important; these ions can be the principal toxicants, even in some very dilute solutions. The much lower toxicities of the ferrocyanide and ferricyanide complexions--which are of high stability but subject to extensive and rapid photolysis, yielding free cyanide on direct exposure to sunlight--and the nickelocyanide ion complex are not likely to be of practical importance (Doudoroff 1976). Toxicity to aquatic organisms of organic cyanide compounds, such as lactonitrile, is similar to that of inorganic cyanides because they usually undergo rapid hydrolysis in water to free cyanide (Towill et al. 1978). There is general agreement that total cyanide concentrations in water in most cases will overestimate the actual cyanide toxicity to aquatic organisms, and that the analytically determined HCN concentration in cyanide-polluted waters

is considered to be the most reliable index of toxicity (Doudoroff 1976; Smith et al. 1979; EPA 1980; Abel and Garner 1986).

Cyanide acts rapidly in aquatic environments, does not persist for extended periods, and is highly species selective; organisms usually recover quickly on removal to clean water. The critical sites for cyanide toxicity in freshwater organisms include the gills, egg capsules, and other sites where gaseous exchange and osmoregulatory processes occur. On passing through a semipermeable membrane, the HCN molecules are usually distributed by way of the circulatory system to various receptor sites where toxic action or detoxification occurs (Leduc 1984). Once in the general circulation, cyanide rapidly inhibits the electron transport chain of vital organs. Signs of distress include increased ventilation, gulping for air at the surface, erratic swimming movements, muscular incoordination, convulsions, tremors, sinking to the bottom, and death with widely extended gill covers (Leduc 1981, 1984). The acute mode of action of HCN is limited to binding those porphyrins that contain Fe^{+3}, such as cytochrome oxidase, hydroperoxidases, and methemoglobin. At lethal levels, cyanide is primarily a respiratory poison and one of the most rapidly effective toxicants known (Leduc et al. 1982). The detoxification mechanism of cyanide is mediated by thiosulfate sulfur transferase, also known as rhodanese. This enzyme is widely distributed in animals, including fish liver, gills, and kidney. Rhodanese plays a key role in sulfur metabolism, and catalyzes the transfer of a sulfane-sulfur group to a thiophilic group (Leduc 1984). Thiosulfate administered in the water with cyanide reduced the toxicity of cyanide to fish, presumably by increasing the detoxification rate of cyanide to thiocyanate (Towill et al. 1978).

Additive or more-than-additive toxicity of free cyanide to aquatic fauna has been reported 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 nickelocyanide complex markedly reduces the toxicity of both cyanide and nickel at high concentrations in alkaline pH. At lower concentrations and acidic pH, solutions increase in toxicity by more than 1,000 times, owing to dissociation of the metallocyanide complex to form hydrogen cyanide (Towill et al. 1978). Mixtures of cyanide and ammonia may interfere 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 is influenced by water alkalinity and pH. Toxicity decreased with increasing alkalinity and pH from 0.42 mg CN/L at 5 mg $CaCO_3$/L and pH 6.5; to 1.4 mg CN/L at 70 mg $CaCO_3$/L and pH 7.5; to 730 mg CN/L at 192 mg $CaCO_3$/L and pH 8.0 (Doudoroff 1956).

Numerous biological and abiotic factors are known to modify the biocidal properties of free cyanide, including water pH, temperature, and oxygen content; life stage, condition, and species assayed; previous exposure to cyanide compounds; presence of other chemicals; and initial dose tested. There is general agreement that cyanide is more toxic to freshwater fish under conditions of low dissolved oxygen (Doudoroff 1976; Towill et al. 1978; Smith et al. 1979; EPA 1980; Leduc 1984); that pH levels within the range 6.8-8.3 had little effect on cyanide toxicity but enhanced toxicity at acidic pH (Smith et al. 1979; EPA 1980; Leduc et al. 1982; Leduc 1984); that juveniles and adults were the most sensitive life stages tested and embryos and sac fry the most resistant (Smith et al. 1978, 1979; EPA 1980; Leduc 1984); and that substantial interspecies variability exists in sensitivity to free cyanide (Smith et al. 1979; EPA 1980). Initial dose and water temperature both modify the biocidal properties of HCN to freshwater teleosts. At slowly lethal concentrations (i.e., < 10 µg HCN/L), cyanide was more toxic at lower temperatures; at high, rapidly lethal HCN concentrations, cyanide was more toxic at elevated temperatures (Kovacs and Leduc 1982a, 1982b; Leduc et al. 1982; Leduc 1984). By contrast, aquatic invertebrates were 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 resistance to cyanide correlated with higher activity induced by exercise and higher temperatures, suggesting a faster detoxification rate or higher oxidative and anaerobic metabolisms. Low levels of cyanide that were 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 low sublethal levels of cyanide through continuous exposure might 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 resistance only slightly to lethal concentrations (Alabaster et al. 1983). Juvenile rainbow trout previously exposed to low 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). Experimental evidence is lacking on exposure to lethal concentrations after prior exposure to high sublethal concentrations; some investigators predict decreased resistance (Leduc 1984), and others increased survival (Towill et al. 1978).

Birds

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-resistant species, such as domestic chickens, 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 15-30 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 was not related to body size but seemed to be associated with diet (Wiemeyer et al. 1986). Birds that feed predominantly on flesh, such as vultures, kestrels, and owls, were more sensitive to cyanide than were species that feed mainly on plant material--with the possible exception of mallard (Anas *platyrhynchos*)--as judged by acute oral LD50 values (Table 4).

Table 4. Cyanide effects on selected species of birds.

Species, dose, and other variables	Effects	Reference[a]
Mallard, *Anas platyrhynchos*		
Single oral dose of NaCN		
0.53 mg CN/kg body weight (BW), equivalent to 1 mg NaCN/kg BW	No deaths	7
1.1 mg CN/kg BW (2.0 mg NaCN/kg BW)	About 6% dead	7
1.27 mg CN/kg BW (2.4 mg NaCN/kg BW)	About 33% dead	7
1.43 mg mg CN/kg BW (2.7 mg NaCN/kg BW)	LD50; 95% confidence interval (C.I.) of 2.2 and 3.2 mg NaCN/kg bW	7
Turkey vulture, *Cathartes aura*		
Single oral dose of 19.1 mg CN/kg BW, equivalent to 36 mg NaCN/kg BW	Up to 80% of the cyanide in blood was present as free cyanide and the remainder as bound cyanide	1
Single oral dose of 19.1 mg CN/kg BW, equivalent to 36 mg NaCN/kg BW	Average time to death was about 19 min and ranged between 8 and 41 min; cyanide residues postmortem, in mg CN/kg fresh weight (FW), were 6.7 in blood (Max. 21) and 0.6 in liver (Max. 2.8)	2
Rock dove, *Columba livia*		
0.12 mg CN/L air, as HCN	All dead in 10 min	2
1.6 mg CN/kg BW, equivalent to 4.0 mg KCN/kg BW	Minimum lethal dose when administered intravenously or intramuscularly	2
Black vulture, *Coragyps atratus*		
Single oral dose, as NaCN		
1.6 mg CN/kg BW	No deaths in 60 min. Mean and maximum blood CN concentrations, in mg/kg FW, were 0.7 and 0.9, respectively	2
2.4 lmg CN/kg BW	Some deaths within 30 min. Mean blood CN residues in	2

	mg/kg FW, were 0.7 in dead birds vs. 1.2 in those surviving 60 min	
2.54 mg CN/kg BW	Acute oral LD50; 95% C.I. of 2.3 and 2.8 mg CN/kg BW (4.4–5.3 mg NaCN/kg BW)	2
3.7 and 19.1 mg CN/kg BW	All dead within 16 min; maximum blood CN levels postmortem were 2.1 mg/kg FW in the low dose group and 4.2 in the high dose group	2
Japanese quail, *Coturnix japonica*		
Single oral dose, as NaCN		
4.5 mg CN/kg BW	Acute oral LD50 for adult females; 95% C.I. of 3.1 and 6.5 mg CN/kg BW	2
5.5 mg CN/kg BW	Acute oral LD50 for adult males; 95% C.I. of 4.0 and 7.5 mg CN/kg BW	2
American kestrel, *Falco sparverius*		
2.12 mg CN/kg BW, as NaCN	Acute oral LD50; 95% C.I. of 1.6 and 2.8 mg CN/kg BW	2
Domestic chicken, *Gallus domesticus*		
Intravenous route		
0.01 µg/kg BW	Most of dose recovered in urine as thiocyanate in 6 h; excretion limited by availability of transferable sulfur	3
0.6 mg CN/kg BW, equivalent to 1.5 mg KCN/kg BW	Lethal	2
0.78 mg CN/kg BW, as KCN	Sublethal; thiocyanate excretion increased 10 times after 10 min and returned to normal levels after 3.5 h; the total thiocyanate collected was equivalent to 85% of the administered dose	4
1.3 mg CN/kg BW, as KCN	Lethal	4
Inhalation route		
0.12 mg HCN/L air	All survived for at least 60 min	2
Single oral dose, as NaCN		
3.2 mg CN/kg BW, equivalent to 6.0 mg NaCN/kg BW	No deaths in 30 min; maximum CN levels, in mg/kg FW, were 1.1 in blood and 0.06 in liver	2
6.4 mg CN/kg BW	Some deaths in 30 min; maximum CN levels, in mg/kg FW were 1.6 in blood and 0.12 in liver	2
11.1 mg CN/kg BW	Acute oral LD50; 95% C.I. of 6.4 and 19.1 mg CN/kg BW	2
25.4 mg CN/kg BW	Advanced signs of acute poisoning; death probable within 30 min; maximum CN levels, in mg/kg FW, were 1.5 in blood and 0.6 in liver	2
Dietary route		
Fed cassava diets containing 4, 37, 70,	At all dietary levels, there	5

or 103 mg total cyanide per kilogram ration to day-old chicks for 8 weeks	was no significant effect on survival, growth, histology, hemoglobin, hematocrit, or lymphocyte number; however, serum thiocyanate levels increased in a dose-dependent manner	
Fed diets containing 135 mg HCN/kg Chicks, 20-day exposure	Growth and food intake significantly depressed; plasma thiocyanate concentration increased	6
Adults, 14-day exposure	Urinary excretion of thiocyanate increased 5 times in laying hens	6
California condor, *Gymnogyps californianus*		
Juvenile (8.4 kg), found dead, presumably of cyanide poisoning	No evidence of injuries or disease; yellow fluorescent particles found in mouth appeared like those placed in NaCN ejector mechanisms used in predator control. However, blood cyanide concentration was similar to that found in nonexposed vultures, including two captive California condors	2
Eastern screech-owl, *Otus asio*		
4.6 mg CN/kg BW, equivalent to 8.6 mg NaCN/kg BW	Acute oral LD50; 95% C.I. of 3.8 and 5.4 mg CN/kg BW	2
Canary, *Serinus canarius*		
0.12 mg HCN/L air	All dead in 3 min	2
European starling, *Sturnus vulgaris*		
9.0 mg CN/kg BW, as NaCN	Acute oral LD50; 95% C.I. of 4.8 and 17 mg CN/kg BW	2
Andean condor, *Vultur gryphus*		
Single oral dose of 19.1 mg CN/kg BW (36 mg NaCN/kg BW)	Blood sampled immediately after death contained 1.2 mg free CN per liter and 0.5 mg bound CN per liter	1

[a]1, Krynitsky et al. 1986; 2, Wiemeyer et al. 1986; 3, Oh et al. 1987; 4, Davis 1981; 5, Gomez et al. 1988; 6, Elzubeir and Davis 1988b; 7, Personal communication, E. F. Hill, Patuxent Wildlife Research Center.

Many species of migratory birds 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 (>200 mg total cyanide per liter) waters (Clark and Hothem 1991). Migratory bird mortality from cyanide toxicosis may be eliminated at these facilities by screening birds from toxic solutions (Hallock 1990) or lowering the cyanide concentrations with hydrogen peroxide to <50 mg total cyanide per liter (Allen 1990), although the latter procedure requires verification (Clark and Hothem 1991).

Some birds may not die immediately after drinking lethal cyanide solutions. In Arizona, a 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.

Elevated cyanide concentrations were found in blood of chickens that died of cyanide poisoning; however, these concentrations overlapped those in survivors. Despite this variability, blood is considered more reliable than liver as an indicator of cyanide residues in exposed birds (Wiemeyer et al. 1986). No gross pathological changes in birds related to cyanide dosing were observed at necropsy (Wiemeyer et al. 1986), similar to other taxonomic groups tested.

Cyanide-nutrient interactions are reported for alanine, which appears to exacerbate cyanide toxicity, and for cystine, which seems to alleviate toxicity (Davis et al. 1988). Dietary cyanide--at levels that do not cause growth depression--alleviates selenium toxicity in chickens, but not the reverse (Davis et al. 1988; Elzubeir and Davis 1988a). For example, dietary selenium, as selenite, at 10 mg/kg for 24 days, reduced growth, food intake, and food utilization efficiency, and produced increased liver size and elevated selenium residues; the addition of 45 mg CN/kg diet (100 mg sodium nitroprusside per kilogram) eliminated all effects except elevated selenium residues in liver. The mechanism of alleviation is unknown and may involve a reduction of tissue selenium through selenocyanate formation, or increased elimination of excess selenium by increasing the amount of dimethyl selenide exhaled (Elzubeir and Davis 1988a). At dietary levels of 135 mg CN/kg plus 10 mg selenium per kilogram, chick growth was significantly decreased (Elzubeir and Davis 1988a). This interaction can be lost if there is a deficiency of certain micronutrients or an excess of vitamin K (Davis et al. 1988).

Mammals

Much of the toxicological interest in cyanide relating to mammals has focused on its rapid lethal action; however, its most widely distributed toxicologic problems are due to its toxicity from dietary, industrial, and environmental factors (Way 1981, 1984; Gee 1987; Marrs and Ballantyne 1987). Chronic exposure to cyanide is correlated with specific human diseases: Nigerian nutritional neuropathy, Leber's optical atrophy, retrobulbar neuritis, pernicious anemia, tobacco amblyopia, cretinism, and ataxic tropical neuropathy (Towill et al. 1978; Way 1981; Sprince et al. 1982; Berninger et al. 1989; Ukhun and Dibie 1989). The effects of chronic cyanide intoxication are confounded by various nutritional factors, such as dietary deficiencies of sulfur-containing amino acids, proteins, and water-soluble vitamins (Way 1981).

Most authorities now agree on five points: (1) cyanide has low persistence in the environment and is not accumulated or stored in any mammal studied; (2) cyanide biomagnification in food webs has not been reported, possibly due to rapid detoxification of sublethal doses by most species, and death at higher doses; (3) cyanide has an unusually low chronic toxicity, but chronic intoxication exists and, in some cases, can be incapacitating; (4) despite the high lethality of large single doses or acute respiratory exposures to high vapor concentrations of cyanide, repeated sublethal doses seldom result in cumulative adverse effects; and (5) cyanide, in substantial but sublethal intermittent doses can be tolerated by many species for long periods, perhaps indefinitely (Towill et al. 1978; EPA 1980; Way 1984; Ballantyne and Marrs 1987a; Table 5).

The toxicity of cyanogenic plants is a problem for both domestic and wild ungulates. Poisoning of herbivorous ungulates is more prevalent under drought conditions, when these mammals become less selective in their choice of forage; dry growing conditions also enhance cyanogenic glycoside accumulations in certain plants (Towill et al. 1978). Animals that eat rapidly are at greatest risk, and intakes of 4 mg HCN/kg BW can be lethal if consumed quickly (Egekeze and Oehme 1980). In general, cattle are most vulnerable to cyanogenic plants; sheep, horses, and pigs--in that order--are more resistant than cattle (Cade and Rubira 1982). Deer *(Odocoileus* sp.) and elk *(Cervus* sp.) have been observed to graze on forages that contain a high content of cyanogenic glycosides; however, cyanide poisoning has not been reported in these species (Towill et al. 1978).

Ruminant and nonruminant ungulate mammals that consume forage with high cyanogenic glycoside content, such as sorghums, Sudan grasses, and corn, may experience toxic signs due to microbes in the gut that hydrolyze the glycosides, releasing free hydrogen cyanide (Towill et al. 1978). 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 can occur quickly, depending on the dose administered (Towill et al. 1978; Cade and Rubira 1982). Thyroid dysfunction has been reported in sheep grazing on stargrass *(Cynodon plectostachyus),* a plant with high cyanogenic glycoside and low iodine content. Sheep developed enlarged thyroids and gave birth to

lambs that were stillborn or died shortly after birth (Towill et al. 1978). Cyanogenic foods can exacerbate selenium deficiency, as judged by the increased incidence of nutritional myopathy in lambs on low-selenium diets (Elzubeir and Davis 1988a). A secondary effect from ingesting cyanogenic glycosides from forage is sulfur deficiency as a result of sulfur mobilization to detoxify the cyanide to thiocyanate (Towill et al. 1978).

Cyanide poisonings of livestock by forage sorghums and other cyanogenic plants are well documented (Cade and Rubira 1982). Horses in the southwestern United States grazing on Sudan grass and sorghums developed posterior muscle incoordination, urinary incontinence, and spinal cord histopathology; offspring of mares that had eaten Sudan grass during early pregnancy developed musculoskeletal deformities (Towill et al. 1978). Salt licks containing sulfur (8.5%) have been used to treat sheep after they failed to gain weight when grazing on sorghum with high HCN content (Towill et al. 1978). Sugar gum (*Eucalyptus cladocalyx*) and manna gum (*Eucalyptus viminalis*) contain high levels of cyanogenic glycosides, and both have been implicated as the source of fatal HCN poisoning in domestic sheep and goats that had eaten leaves from branches felled for drought feeding, or after grazing sucker shoots on lopped stumps (Webber et al. 1984). In one case, 10 goats died and 10 others were in distress within 2 h after eating leaves from a felled sugar gum. Dead goats had bright red blood that failed to clot and subepicardial petechial hemorrhages. Rumens of dead goats contained leaves of *Eucalyptus* spp. and smelled of bitter almonds. The 10 survivors were treated intravenously with 3 mL of a 1-L solution made to contain 20 g of sodium nitrite and 50 g of sodium thiosulphate; four recovered and six died. Of 50 afflicted goats, 24 died within 24 h and the remainder recovered (Webber et al. 1984). In rare instances HCN poisoning occurs when animals are exposed to chemicals used for fumigation or as a fertilizer (Webber et al. 1984), but there is general agreement that ingestion of plants containing high levels of cyanogenic glycosides is the most frequent cause of cyanide poisoning in livestock.

Cassava, also known as maniac, tapioca, yuca, or guacamate, is one of the very few--and, by far, the most important--food crops in which the cyanide content creates toxic problems (Cooke and Coursey 1981). Cassava is a major energy source for people and livestock in many parts of the world; it accounts for an average of 40% of the human caloric intake in Africa (Casadei et al. 1984), to more than 70% in some African diets (Way 1984). In comparison to other tropical crops it produces the highest yield per hectare (Okeke et al. 1985). Cassava is native to tropical America from southern Mexico to northern Argentina and probably has been under cultivation there for 4,000-5,000 years. It has been introduced to east Africa, Indian Ocean islands, southern India, and the Far East (Cooke and Coursey 1981). The global production of cassava roots was estimated at 50 million tons in 1950, and 100 million tons in 1980; about 44.2 million tons are grown annually in Africa, 32.7 million tons in tropical America, and 32.9 million tons in Asia (Cooke and Coursey 1981). Linamurin is the principal cyanogenic glycoside in cassava; its toxicity is due to hydrolysis by intestinal microflora releasing free cyanide (Padmaja and Panikkar 1989). Rabbits *(Oryctolagus cuniculus)* fed 1.43 mg linamurin per kilogram BW daily (10 mg/kg BW weekly) for 24 weeks showed effects similar to those of rabbits fed 0.3 mg KCN/kg BW weekly. Specific effects produced by linamurin and KCN included elevated lactic acid in heart, brain, and liver; reduced glycogen in liver and brain; and marked depletion in brain phospholipids (Padmaja and Panikkar 1989).

The use of cassava in animal feed presents two major problems: the presence of cyanogenic glycosides in the tuber, and the remarkably low protein levels in fresh and dried cassava. Pigs fed low-protein cassava diets for 8 weeks had reduced food consumption and lowered liver weight; addition of protein supplement to the diet reversed these trends (Tewe 1982b). Removal of cyanogenic glycosides from cassava tubers, mash, peels, and root meal is accomplished with several techniques. Usually, the cassava root is dried in the sun for several weeks, and this process removes most of the cyanogenic glycosides; however, under conditions of famine or food shortage, this process cannot be carried out properly (Cliff et al. 1984). Long fermentation periods, especially under conditions of high moisture content, may be effective in substantial detoxification of cassava mash (Ukhun and Dibie 1989). Cassava peels containing as much as 1,061 mg HCN/kg FW can be rendered suitable for feeding to livestock (4-625 mg/kg) by boiling for 7 min, roasting for 30 min, soaking for 15 h, or drying in the sun for 7.6 days (Okeke et al. 1985). Cassava root meal (up to 40% of cassava meal) is satisfactory as a diet supplement for domestic pigs, provided cyanide content is <100 mg/kg ration (Gomez et al. 1983).

Neuropathies associated with cassava ingestion (i.e., cyanide intoxication) can develop into a syndrome in humans and domestic animals, characterized by nerve deafness, optic atrophy, and an involvement of the sensory spinal nerve that produces ataxia. Other symptoms include stomatitis, glossitis, and scrotal dermatitis (Way 1981). Potentially more serious are long-term effects such as ataxic neuropathy, goiter, and cretinism,

which have been attributed to high cassava content in diets. Thiocyanate--one of the detoxification products--inhibits iodine absorption and promotes goiter, a common ailment in tropical countries (Cooke and Coursey 1981). At high dietary cyanide intakes there is an association with diabetes and cancer (Cliff et al. 1984), but this requires verification. The first case of cassava toxicity occurred almost 400 years ago (Cooke and Coursey 1981). The toxic principle was later identified as a cyanogenic glycoside, shown to be identical with flax linamurin (2-(beta-D-glucopyranosyloxy)-isobutyronitrile). All parts of the plant, except possibly the seeds, contain the glycoside together with the enzyme linamarase. This enzyme effects hydrolysis of the nitrile to free HCN when the tissue cellular structure is damaged (Cooke and Coursey 1981). Mantakassa disease is related to chronic cyanide intoxication associated with a diet consisting almost exclusively of cassava; in times of famine and sulfur-poor diets, Mantakassa effects were more pronounced (Casadei et al. 1984). Symptoms of Mantakassa disease include the sudden onset of difficulty in walking, increased knee and ankle reflexes, elevated serum thiocyanate levels, fever, pain, headache, slurred speech, dizziness, and vomiting. Women of reproductive age and children were the most seriously affected. Symptoms persisted for up to 4 months after treatment with hydroxycobalamin, vitamin supplements, and a high protein, energy-rich diet (Cliff et al. 1984). Mantakassa was reported in 1,102 victims in Mozambique in 1981 from a drought-stricken cassava staple area; from Zaire in 1928, 1932, 1937, and again in 1978-81; in Nigeria; and in the United Republic of Tanzania. The mean serum thiocyanate level in patients with Mantakassa is 2.6 times higher than in non-Mantakassa patients in Nigeria, and 3.5 times higher than in Tanzanian patients. Pesticides, infection, viruses, and consumption of food other than cassava were eliminated as possible causative agents in Mantakassa disease. Still unresolved is whether the disease is triggered when a threshold level of thiocyanate is reached, or when a critical combination of cyanide intoxication plus nutritional deficiency occurs (Cliff et al. 1984).

Routes of administration other than dietary ingestion should not be discounted. Livestock found dead near a cyanide disposal site had been drinking surface water runoff from the area that contained up to 365 mg HCN/L (EPA 1980). The use of cyanide fumigant powder formulations may be hazardous by contact of the powder with moist or abraded skin, contact with the eye, swallowing, and inhalation of evolved HCN (Ballantyne 1988). In rabbits, lethal systemic toxicity was produced by contamination of the eye, moist skin, or abraded skin (but not dry skin) with cyanide powder formulations (40% NaCN plus 60% kaolin) administered at 1-5 g powder per cubic meter (Ballantyne 1988). Hydrogen cyanide in the liquid state can readily penetrate the skin, and 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 (EPA 1980).

Use of poisons in livestock collars is both specific and selective for animals causing depredations, as is the case for cyanide collars to protect sheep against coyotes (Sterner 1979; Table 5). These collars contain a 33% NaCN solution and are usually effective against coyotes. However, field results indicate that some coyotes kill by means other than neck attack, and some exhibit great wariness in attacking collared sheep (Savarie and Sterner 1979).

Calcium cyanide in flake form was used in the 1920's to kill black-tailed prairie dogs and pocket gophers *(Geomys bursarius)* in Kansas, and various other species of rodents in Nova Scotia (Wade 1924). For prairie dog control, the usual practice was to place 43-56 g of calcium cyanide 0.3-0.7 m below the rim of the burrow and to close the entrances. The moisture in the air liberated HCN gas, which remained in the burrow for several hours, producing 100% kill. A lower dose of 28 g per burrow was about 90% effective (Wade 1924). Control of prairie dogs with cyanide sometimes resulted in the death of burrowing owls that lived in the prairie dog burrows (Wade 1924).

Clinical signs of acute cyanide poisoning in mammals last only a few minutes after ingestion and include rapid and labored breathing, ataxia, cardiac irregularities, dilated pupils, convulsions, coma, respiratory failure, and rapid death (Egekeze and Oehme 1980; Ballantyne 1983). Cyanide poisoning causes cardiovascular changes as well as its better known effects on cellular respiration. Cyanide increases cerebral blood flow in rabbits and cats, and disrupts systemic arterial pressure in dogs (Robinson et al. 1985). Cyanide affects mammalian behavior, mostly motor functions, although these effects have not been quantified. Cyanide-induced motor alterations observed in rats and guinea pigs include muscular incoordination, increased whole-body locomotion, disrupted swimming performance, and altered conditioned avoidance responses (D'Mello 1987). As a consequence of the cytotoxic hypoxia in acute cyanide poisoning, there is a shift from aerobic to

anaerobic metabolism, and the development of lactate acidosis. A combination of rapid breathing, convulsions, and lactate acidosis is strongly suggestive of acute cyanide poisoning (Ballantyne 1983). As with other chemical asphyxiants, the critical organs that are most sensitive to oxygen depletion are the brain and heart (Egekeze and Oehme 1980). The only consistent postmortem changes found in animals poisoned by cyanide are those relating to oxygenation of the blood. Because oxygen cannot be utilized, venous blood has a bright-red color and is slow to clot (Egekeze and Oehme 1980). Bright-red venous blood is not a reliable indicator of cause of death, however, because it is also associated with chemicals other than cyanide (Ballantyne 1983).

Cyanide poisoning is associated with changes in various physiological and biochemical parameters. The earliest effect of cyanide intoxication in mice seems to be inhibition of hepatic rhodanese activity, due to either blockage by excess binding to the active site or to depletion of the sulfane-sulfur pool. These changes do not seem to occur in blood, where rhodanese functions at its maximal rate, thus preventing cyanide from reaching the target tissues and causing death (Buzaleh et al. 1989). Cyanide causes dose- and species-dependent responses on vascular smooth muscle; studies with isolated aortic strips indicate that rabbits are 80 times more sensitive than dogs or ferrets (*Mustela putorius*; Robinson et al. 1985). Rabbits killed with HCN had higher concentrations of cyanide in blood and other tissues and lower tissue cytochrome oxidase activities than did those killed with KCN (Ballantyne et al. 1972). Cyanide promotes dose- and calcium-dependent release of dopamine tissues in the domestic cat, and reductions in adenosine triphosphate (ATP) content of the carotid body (Obeso et al. 1989). Cyanide-induced hypoxia is believed to decrease ATP content of Type I glomus cells. The decrease in the phosphate transfer potential is a crucial step in the overall transduction process, that is, the activation of the transmitter release from Type I cells, with resultant release and activation of sensory nerve endings (Obeso et al. 1989). Studies with isolated heart of the domestic ferret demonstrate that cyanide affects intracellular ionic exchange of H^+, Na^+, and calcium (Fry et al. 1987); inhibits cardiac action potential (Elliott et al. 1989); and inhibits oxidative phosphorylation accompanied by an intracellular acidosis, a decrease in phosphocreatinine, and a rise in inorganic phosphate (Eisner et al. 1987). When oxidative phosphorylation is inhibited in cardiac muscle, there is a rapid decrease of developed force or pressure; most of the decrease of developed pressure produced by cyanide in ferret heart is not produced by intracellular acidosis, and may result from increased inorganic phosphate (Eisner et al. 1987). Observed changes in rat cerebral oxidative responses to cyanide may be due to redistribution of intracellular oxygen supply to mitochondria respiring in an oxygen-dependent manner or by branching effects within brain mitochondria (Lee et al. 1988). Hyperammonemia and the increase of neutral and aromatic amino acids may also be important in loss of consciousness induced by cyanide (Yamamoto 1989).

Table 5. Cyanide effects on selected species of mammals.

Species, dose, and other variables	Effects	Reference[a]
Cattle, *Bos* sp.		
Fed hybrid sorghum Sudan grass cross 988 at 15–20 kg per animal daily for 3–8 days	Of 180 cows, 21 were affected and 13 died; toxic cyanide levels were measured in fodder and in liver and ruminal contents of dead cows	44
Dog, *Canis familiaris*		
Administered doses up to 2 mg NaCN/kg body weight (BW), once or twice daily for 15 months	Acute toxic signs evident after each administration, but complete recovery within 30 min; no measurable adverse effects after 15 months	1
5.4 mg NaCN/kg BW, single subcutaneous injection	LD50	2
24 mg CN/kg BW, single oral or slow intravenous injection	Lethal; at time of respiratory arrest, blood plasma	3

route	concentration was 1 mg total CN per liter or about 0.4 mg free cyanide per liter	
Fed diets containing 150 mg NaCN/kg for 30 days	No measurable effect on food consumption, blood chemistry, behavior, or organ histology	1
Coyote, *Canis latrans*		
Single forced oral dose of NaCN, in mg/kg BW		
4	Immobilized in 13 min, but all survived for at least 30 days; some sacrificed after 30 min: NaCN residues in mg/kg fresh weight (FW) were 0.03 in blood and 0.9 in stomach	2
4.1 (2.1–8.3)	LD50	2
8	Immobilization in 9 min, death within 41 min	2
16, 32, or 64	All immobilized in less than 1 min and all died in less than 8 min. Maximum NaCN residues were 0.14 mg/L in blood and 13.0 mg/L FW in stomach	2
"Toxic" collars attached to neck of sheep and camouflaged with wool; each collar contained 50 mL of a 33% NaCN solution; toxic action commences when coyote attacks sheep and punctures collar; all coyotes tested were known to attack sheep in laboratory pens	Of three coyotes tested, one was immobilized in 1 min and died within 18 min; the other two coyotes recovered; the dead coyote had mouthed the collar for about 2 s; residues in mg NaCN/kg, were 0.26 in blood and <0.1 in stomach; the other two coyotes had mouthed the collar for 3–15 s and had NaCN levels, in mg/kg FW, of 0.014 and 0.029 in blood, and 0.6 and <0.1 in stomach	2
Toxic collar, as above; each coyote tested was known to have fatally attacked at least three domestic sheep within a 30-day period	Of the 12 coyotes that attacked the neck region of the sheep and punctured the collar, nine received lethal doses and became immobilized in 1–3 min and died 3–25 min later; the mean time to death was 11.6 min; one of the three sublethally dosed coyotes survived at last three successful attacks in which the collar was punctured, and two survived two attacks; in all cases, contact with NaCN	4

	produced shaking of the head, pawing at the mouth, rubbing the snout on the ground, and ataxia	
African giant rat, *Cricetomys gambianus*		
Weanlings fed diets for 16 weeks containing 0 mg HCN/kg (maize), 110 mg HCN/kg (cassava pulp), 150 mg HCN/kg (cassava tuber), or 597 mg HCN/kg (cassava peel)	Food consumption was similar in all diets; no pathology was observed in any organ of animals on all treatments; rats on maize and cassava pulp diets had significantly increased growth rate, feed efficiency, and protein efficiency; rats on cassava peel and tuber diets had significantly increased thiocyanate levels in serum, organs, and urine	5
Juveniles, age 10–14 weeks, fed cassava peel diets for 2 weeks containing 720 mg HCN/kg	Adverse effects on growth when cassava peel exceeds 7.8% of the ration	6
Weanlings fed 1,000 mg CN/kg diet, as KCN, for 12 weeks	Reduction in feed intake, reduced body weight, elevated thiocyante concentrations in serum (37.4 mg/L vs. 12.6), urine (341 mg/L vs. 25), liver (1.7 g/kg FW vs. 0.4), kidney (2.4 g/kg FW vs. 0.4), and spleen (2.1 g/kg FW vs. 0.3)	7
Humans, *Homo sapiens*		
Intentional oral ingestion of unknown amount of NaCN or KCN, three cases	Death between 5 and 30 min; stomach cyanide concentrations ranged between 100 and 164 mg; tissue residues postmortem in mg/kg FW, were 0.3–1.1 in blood, 0.3–1.0 in liver, and 0.2–0.3 in brain	8
Found dead, four cases, time to death unknown	Maximum cyanide concentration in stomach was 230 mg; maximum tissue residues, in mg/kg FW were 3.5 in blood, 6.3 in liver, and 0.5 in brain	8
Attempted suicide by 39-year-old-male, unknown amount of NaCN	Severe tremors and progressive loss of muscle tone--representing the first case of cyanide intoxication with delayed onset of symptoms	9
Inhalation of HCN gas, in mg/m^3, for various time intervals		

140 for 60 min	Calculated LC50	10
220 for 30 min	Calculated LC50	10
504 for 10 min	Calculated LC50	10
680 for 5 min	Calculated LC50	10
1,500 for 3 min	Calculated LC50	10
4,400 for 1 min	Calculated LC50	10
Inhalation of 2,000 mg HCN/L	First breath results in deep, rapid breathing, with collapse, convulsions, and death within 1 min	11
Inhalation of cyanogen chloride, in mg/L, for various time intervals		
1, 10 min	Irritant	1
48, 30 min	Fatal	1
159, 10 min	Fatal	1
Inhalation of cyanogen bromide, in mg/L, for various time intervals		
1.4, no time given	Irritant to eyes and nose	1
92, 10 min	Fatal	1
Single oral dose		
0.5–3.5 mg HCN/kg BW	Lethal	12, 41
0.7–3.5 mg KCN/kg BW, equivalent to 50 to 250 mg KCN/adult	Fatal	10
2 mg HCN/kg BW, or total of about 150 mg HCN	Acute LD50 for adults	13
1–5 g of NaCN or KCN, equivalent to 0.2 g/adult or 3 mg/kg BW	Minimum lethal dose	14
Tissue residues		
Whole blood, 1–2 mg free cyanide per liter	Usually lethal	42
Whole blood, 2.6–3.1 mg total CN per liter	Minimum cyanide concentration associated with death in an otherwise healthy individual	13
Whole blood, 2.6–3.1 mg total CN per liter	Minimum cyanide concentration associated with death in an otherwise healthy individual	13
Whole blood, 4–45 mg total CN per liter	Levels measured in known suicides	13
Whole body, 7 mg HCN/kg BW	Residue associated with minimum lethal dose	11
Daily dietary intake of 15–31.5 mg hydrogen cyanide from cassava	Mantakassa disease--see text for discussion	15
100 mg HCN/kg BW applied to skin surface	LD50	11
Clothing inundated with 10% NaCN solution, pH 11.4	Clinical signs of toxicity within 25 min and death in about 60 min	13
Livestock		
>200 mg HCN/kg plant materials in diet	Potentially dangerous	13

Cynomolgus monkeys, *Macaca* spp.		
Given multiple sublethal doses of KCN (5–18 mg) for 23 days	Brain histoapathology	3
Exposed to HCN gas produced from combustion of polyacrylonitrile materials at various temperatures		
300º C, 87–170 mg HCN/L air	Incapacitated in 16–30 min; blood cyanide of 4.3 mg/L	16
600º C, 120–174 mg HCN/L air	Incapacitated between 6 and 24 min, blood cyanide of 2.96 mg/L	16
900º C, 166–196 mg HCN/L air	Incapacitated between 2 and 13 min; blood cyanide concentration of 3.1 mg/L	16
Exposed to HCN gas at air concentrations of 60, 80, or 150 mg HCN/L for 30 min	At 60 mg/L, HCN had only a slight depressive effect on the central nervous system; at 80 and 150 mg/L, severe CNS depression and incapacitation occurred	17
Exposed to HCN gas at air concentrations of 100, 1092, 123, 147, or 156 mg HCN/L air	Incapacitated in 8 min at higher doses to 19 min at lowest dose tested; blood cyanide after 30 min exposure ranged between 1.7 mg/L at 100 mg HCN/L and 3.2 mg/L at 156 mg HCN/L; after recovery for 60 min, blood CN ranged between 2.0 and 2.9 mg/L	16
Domestic mouse, *Mus* spp.		
Single intraperitoneal injection		
HCN, 2.8 mg/kg BW	LD50	10
NaCN, 4.6–5.9 mg/kg BW	LD50	10
KCN, 5.3–6.7 mg/kg BW	LD50	10
Acetone cyanohydrin, $(CH_3)_2C(OH)CN$, 8.7 mg/kg BW	LD50 (7 days); first death in 5 min	18
Malonitrile, $NCCH_2CN$, 18 mg/kg BW	LD50 (7 days); first death in 4.8 h	18
Propionitrile, CH_3CH_2CN, 28 mg/kg BW	LD50 (7 days); first death in 21 h	18
N-butyronitrile, 38 mg/kg BW	LD50 (7 days); first death in 2.2 h	18
Acrylonitrile, CH_2CHCN, 46 mg/kg BW	LD50 (7 days); first death in 2.3 h	18
Succinonitrile, $NCCH_2CH_2CN$, 62 mg/kg BW	LD50 (7 days); first death in 5.1 h	18
Acetonitrile, CH_3CN, 175 mg/kg BW	LD50 (7 days); first death in 7.1 h	18
Single subcutaneous injection		
HCN, 7.8–12.0 mg/kg BW	LD50	10
KCN, 10 mg/kg BW	Loss of consciousness in 100%; blood ammonia levels increased 2.5 times; brain amino acid levels (i.e., leucine,	19

	isoleucine, tyrosine, phenylalanine) increased by 1.5–3.0 times; alpha ketoglutarate, at 500 mg/kg BW by intraperitoneal injection, completely blocked the development of cyanide-induced loss of consciousness and hyperammonemia	
Single oral dose		
8.5 mg KCN/kg BW, equivalent to 3.4 mg CN^-/kg BW	LD50	10, 20
Drinking water, 1,000 mg KCN/L, exposure for 40 days	Marked inhibition of cytochrome oxidase activity in liver, brain, and blood; increased cyanide concentrations in all tissues; inhibition of rhodanese activity; diminished labile sulfur tissue levels	43
Rabbit, *Oryctolagus* spp.		
Isolated aorta strips, 0.00014 µg NaCN/L–140 µg/L	Small contractions measured at lowest dose tested, ED50 at 70 µg/L, and maximum response at 140 µg/L; higher doses up to 14 mg/L produced relaxation	21
Single intramuscular injection, in mg/kg BW		
0.5–1.5	LD50 for HCN	10
1.6	LD50 for NaCN	10
3.1–3.3	LD50 for KCN	10
8.0		
Killed with KCN	Cyanide concentrations, in mg/kg FW, were 1.6 in serum, 5.3 in blood, and <0.4 in other tissues sampled	22
Killed with HCN	Cyanide concentrations, in mg/kg FW, were 9.3 in blood, 2.1 in brain, 2.0 in serum, 0.5 in myocardium, and <0.4 in other tissues	22
Single intravenous injection, in mg/kg BW		
0.6	LD50 for HCN	10
1.2	LD50 for NaCN	10
1.9	LD50 for KCN	10
Single dose administered to eye surface, in mg/kg BW		
1.0	LD50 for HCN	10
4.5–5.1	LD50 for NaCN	10
7.9	LD50 for KCN	10
11.2	Signs of NaCN poisoning in 3 min, death in 7 min	23
Single intraperitoneal injection, in mg/kg BW		
1.7–2.0	LD50 for HCN	10
2.8–2.9	LD50 for NaCN	10

3.6–4.0	LD50 for KCN	10
Administered as solution to skin, in mg/kg BW		
2.3	LD50 for HCN and abraded skin	10
6.9	LD50 for HCN and intact skin	10
14.3	LD50 for KCN and abraded skin	10
19.3	Abraded skin; signs of NaCN poisoning evident in 25 min, death in 41 min	23
22.3	LD50 for KCN and intact skin	10
29.5	Moist skin; signs of NaCN poisoning evident in 79 min, death in 117 min	23
>110	Dry skin; no signs of NaCN poisoning, no deaths	23
Single oral dose, in mg/kg BW		
2.5	LD50 for HCN	10
5.1	LD50 for NaCN	10
5.8	LD50 for KCN	10
12.8	Signs of NaCN poisoning in 4 min, death in 22 min	23
Single oral dose, NaCN, 10–15 mg/kg BW	All dead in 14–30 min; blood cyanide ranged between 3.7 and 5.4 mg/L	24
Inhalation of HCN from combustion of 20 g of polyacrylonitrile	All dead in 12–16 min; blood cyanide ranged between 1.6 and 3.1 mg/L	24
Interval between death and removal of tissues for analysis in rabbits killed by KCN		
Brain	Concentrations dropped from 1.6 mg/kg FW immediately after death to 1.2 in 1 day, 0.92 in 3 days, and 0.04 in 7 days	25
Blood	Residues, in mg/kg FW were 5.7 immediately after death and 2.3 after 21 days	25
Lung	Cyanide concentrations dropped from 2 mg/kg FW just after death, to 0.8 in 7 days	25
Domestic sheep, *Ovis aries*		
Intravenous or intraarterial injection, fetal lambs 80% through gestation (120 days), NaCN, 50–400 µg	Slowing of fetal heart rate, disruption of respiratory movements, significant but inconsistent changes in arterial blood pressure	26
Single intramuscular injection of 10 mg KCN/kg BW	All dead within 17 min; cyanide concentrations postmortem, in mg/kg FW, were 3.3 in blood, 1.5 in plasma, 1.6 in serum, 1.4 in cerebrospinal fluid, 0.9 in brain grey matter, and 1.0 in brain white matter	3, 10, 27
Laboratory white rat, *Rattus* spp.		
Single intraperitoneal injection		

0.1–10 mg CN/kg BW	LD50	28
5 mg NaCN or KCN/kg BW	50% decrease in brain cytochrome oxidase activity within 5–10 min	14
5 mg KCN/kg BW	Reversible intracellular metabolic changes including acidosis and increased lactate levels--typical of cellular anoxia	29
Intravenous injection, constant infusion of 0.15–0.20 mg CN/kg BW per min	LD50 in about 20 min. Rapid progressive reduction in cerebrocortical cytochrome oxidase (cytochrome aa_3) concomitant with increases up to 200% in cerebral blood flow	30
Single intracartoid artery injection of KCN		
1–2 mg/kg BW	Modest acute clinical dysfunction and incomplete suppression of brain electroencephalographic (EEG) activity	31
2.5 mg/kg BW	Some deaths; survivors showed rapid abolition of brain EEG activity, 52% reduction in brain cytochrome oxidase activity, 600% increase in lactate, 85% decrease in glycogen, 32% reduction in ATP, and 73% increase in ADP; all values returned to normal in 6–24 h, and remained normal for balance of 7-day observation period	31
3.5–5 mg/kg BW	High incidence of cardiovascular collapse and death within minutes	31
Tissue residues 2.6–2.9 mg HCN/L	Minimum lethal concentrations in rats poisoned orally with KCN	13
Inhalation exposure route, HCN vapor, in mg/m^3, for various periods		
3,778 for 10 s	LC50	10
1,128 for 1 min	LC50	10
493 for 5 min	LC50	10
151–173 for 30–60 min	LC50	10
Single oral dose		
3.4 mg KCN/kg BW	LD25	32
3.6–4.2 mg HCN/kg BW	LD50	10
5.1–5.7 mg NaCN/kg BW	LD50	10
5.7 mg KCN/kg BW	LD50	32
6, 10, or 14 mg KCN/kg BW	Some deaths in all groups; all dead at higher doses within 60 min; those killed 10 min postadministration had	13

	higer blood CN concentrations than those killed near death or at survival at 60 min	
6.4 mg NaCN/kg BW	LD50	13
7.5–10 mg KCN/kg BW	LD50	10, 13
8.6 mg KCN/kg BW	LD98	32
10 mg KCN/kg BW, equivalent to 4 mg HCN/kg BW	LD50	20
13.2 mg NaCN/kg BW or 7 mg HCN/kg BW	Dead in 10.3 min; tissue cyanide levels, in mg/kg FW, were 8.9 in liver, 5.9 in lung, 4.9 in blood, 2.1 in spleen, and 1.5 in brain	33
40 mg NaCN/kg BW, equivalent to 21 mg HCN/kg BW	Dead in 3.3 min	33
Drinking water exposure		
Equivalent to 8 mg CN/kg BW daily for 21 days	Liver normal	20
Equivalent to 21 mg CN/kg BW daily for 21 days	Significantly increased liver weight	20
200 mg CN/L for 4 weeks	Reduced growth	34
Drinking water of adults contained 150 mg CN/L, as KCN, for 2 weeks, followed by injection with radioselenium-75 and observed for 15 days	Cyanide-treated rats excreted significantly more radioselenium in urine than did controls; half-time persistence of radioselenium in treated group was 28 days vs. 38 days in controls	35
Drinking water of weanling males contained 150 mg CN/L for 9 weeks	Significant reduction in glutathion activity, and in selenium concentrations in blood kidney, liver, and muscle	35
Dietary exposure		
Fed 12 mg CN/kg BW daily for 2 years, equivalent to 300 mg HCN/kg ration	No measurable adverse effects on blood chemistry, growth, survival, or histology; elevated thiocyanate levels in liver and kidneys	1
Fed 500 mg HCN/kg ration to pregnant rats through gestation and lactation	No effect on reproduction	20
Weanlings fed diets of raw lima beans containing 727 mg CN/kg for 3 weeks, or 727 mg CN/kg diet as KCN for 3 weeks	Lima bean diet alone increased hepatic glutamate dehydrogenase (GLDH) and decreased isocitrate dehydrogenase (ICDH) activities; KCN diet had no effect on GLDH and increased ICDH activity, emphasizing the importance of dietary components when evaluating CN-containing diets	36
750 mg CN/kg diet (1,875 mg KCN/kg diet) for 8 weeks,	No measurable effect on food consumption or growth rate;	37

adequate protein	significantly increased serum and urinary thiocyanate concentrations	
As above, protein deficient diet	Reduction in body weight gain, reduction in serum thiocyanate concentration	37
Weanling males fed diets containing 1,500 mg KCN/kg, or 2,240 mg potassium thiocyanate (KSCN)/kg for 50 weeks	No deaths or clinical signs of toxicity; both groups had decreased thyroid gland activity; cyanide, but not thiocyanate, caused reduction in growth rate	38
Isolated liver segments from starved rats exposed to 100 mg KCN/L	Oxygen consumption reduced 80%, and evidence of hepatotoxicity as judged by enzyme release, glutathione depletion, and calcium accumulation in liver; hepatotoxicity prevented by feeding rats fructose	39
Domestic pig, *Sus* spp.		
Fed diet containing 96 mg CN/kg ration, as cassava peel for 72 days	No effect on food consumption or protein metabolism	40

[a]1, EPA 1980; 2, Sterner 1979; 3, Christel et al. 1977; 4, Savarie and Sterner 1979; 5, Tewe 1984; 6, Tewe 1988; 7, Tewe 1982a; 8, Curry 1963; 9, Grandas et al. 1989; 10, Ballantyne 198a; 11, Towill et al. 1978; 12, Ukhun and Dibie 1989; 13, Egekeze and Oehme 1980; 14, Way 1981; 15, Casadei et al. 1984; 16, Purser et al. 1984; 17, Purser 1984; 18, Willhite and Smith 1981; 19, Yamamoto 1989; 20, EPA 1989; 21, Robinson et al. 1985; 22, Ballantyne et al. 1972; 23, Ballantyne 1988; 24, Yamamoto et al. 1979; 25, Ballantyne et al. 1974; 26, Itskovitz and Rudolph 198; 27, Ballantyne 1975; 28, Brattsten et al. 1983; 29, Lotito et al. 1989; 30, Lee et al. 1988; 31, MacMillan 1989; 32, Keniston et al. 1987; 33, Yamamoto et al. 1982; 34, Palmer and Olson 1981; 35, Beilstein and Whanger 1984; 36, Aletor and Fetuga 1988; 37, Tewe and Maner 1985; 38, Philbrick et al. 1979; 39, Younes and Strubelt 1988; 40, Tewe and Pessu 1982; 41, Way 1984; 42, Marrs and Ballantyne 1987; 43, Buzaleh et al. 1989; 44, Bapat and Abhyankar 1984.

Organic cyanide compounds, or nitriles, have been implicated in numerous human fatalities and signs of poisoning—specially acetonitrile, acrylonitrile, acetone cyanohydrin, malonitrile, and succinonitrile. Nitriles hydrolyze to carboxylic acid and ammonia in either basic or acidic solutions. Mice *(Mus* sp.) given lethal doses of various nitriles had elevated cyanide concentrations in liver and brain; the major acute toxicity of nitriles is CN release by liver processes (Willhite and Smith 1981). In general, alkylnitriles release CN much less readily than aryl alkylnitriles, and this may account for their comparatively low toxicity (Davis 1981).

No human cases of illness or death due to cyanide in water supplies are known (EPA 1980). Accidental acute cyanide poisonings in humans are uncommon (Towill et al. 1978); however, a man accidentally splashed with molten sodium cyanide died about 10 h later (Curry 1963). Human cyanide deaths usually involve suicides, where relatively large amounts of sodium cyanide or potassium cyanide are ingested and the victims die rapidly in obvious circumstances. Recovery after oral ingestion is rare. In one case, a spouse emptied capsules containing medicine and repacked them with 40% solid NaCN. The victim took one capsule and ingested about 0.05 g, but vomited and recovered completely (Curry 1963). Human deaths are increasing from gas or smoke inhalation from urban fires, possibly owing to the increased toxicity of fire atmospheres caused by the use of organocyanide plastics in modern construction and furnishings (Egekeze and Oehme 1980). Hydrogen cyanide may be important in some fires in producing rapid incapacitation, causing the victims to remain in the fire and die from carbon monoxide or other factors, although HCN concentrations of 60 mg/L air and lower had minimal effects (Purser 1984). Exposure to the mixture of HCN and carbon monoxide, with accompanying changes in cerebral blood flow during attempts to escape from fires, may be a cause of collapse and subsequent death

(Purser 1984). For example, cynomolgus monkeys (*Macaca* spp.) exposed to pyrolysis products of polyacrylonitrile (PAN) and to low-level HCN gas had similar physiological effects in both atmospheres, specifically: hyperventilation, followed by loss of consciousness after 1-5 min; and brachycardia, with arrhythmias and T-wave abnormalities. Recovery was rapid following cessation of exposure (Purser et al. 1984). Because HCN is the major toxic product formed by the pyrolysis of PAN, Purser et al. (1984) suggested that HCN may produce rapid incapacitation at low blood levels of cyanide in fires, while death may occur later due to carbon monoxide poisoning or other factors.

Finally, cyanide does not appear to be mutagenic, teratogenic, or carcinogenic in mammals (EPA 1980; Ballantyne 1987a). In fact, there has been a long-standing hypothesis for an anticancer effect of the cyanogenic glycoside amygdalin (also called laetrile). The hypothesis is based on amygdalin's selective hydrolysis by a beta glucosidase, liberating cyanide and benzaldehyde at the neoplastic site. The cyanide then selectively attacks the cancer cell, which is presumed to be low in rhodanese, whereas normal cells are assumed to possess sufficient rhodanese and sulfur to detoxify the cyanide (Way 1981). However, many tumors are neither selectively enriched in beta glucosidase nor low in rhodanese (Way 1981).

Recommendations

Proposed free cyanide criteria suggest that sensitive species of aquatic organisms are protected at <3 µg/L, birds and livestock at <100 mg/ kg diet, and human health at concentrations of <10 µg/L drinking water, <50 mg/kg diet, and <5 mg/m^3 air (Table 6).

Analytical methodologies need to be developed that differentiate between free cyanide (HCN and CN^-) and other forms of cyanide, and that are simple, sensitive (i.e., in the µg/L range), and accurate (Smith et al. 1979; Leduc et al. 1982). Procedures need to be standardized that ensure prompt refrigeration and analysis of all samples for cyanide determination because some stored samples generate cyanide while others show decreases (Gee 1987).

Periodic monitoring of cyanide in waterways is unsatisfactory for assessing potential hazards because of cyanide's rapid action, high toxicity, and low environmental persistence. A similar case is made for cyanide in the atmosphere. Development of a continuous monitoring system of cyanides in waterways and air is recommended, with emphasis on point source dischargers, such as industrial and municipal facilities (Towill et al. 1978; Egekeze and Oehme 1980; Leduc et al. 1982). Information is needed on the fate of cyanide compounds in natural waters, relative contributions of natural and anthropogenic sources, and critical exposure routes for aquatic organisms (Leduc et al. 1982). Additional research is needed on the origin of cyanide in wilderness and rural watershed areas, specifically the roles of organic wastes and their associated bacterial flora, aquatic vegetation induced by nutrient enrichment, and terrestrial plant cover in the watershed (Leduc 1984).

Table 6. Proposed free cyanide criteria for the protection of living resources and human health.

Resource criterion, and other variables	Concentration	Reference[a]
Freshwater organisms		
Effect levels, in µg/L medium		
Minimal impairment, most species of fish	3–5	1, 2, 3, 4, 5, 6
Reduced survival, amphipods	>3–34	1, 7
Safe, most fish species	3.5 (24-h average, not to exceed 52 at any time)	7
Significant impairment, most species of fish	8–16, exposure for at least 20 days	6, 7
Hazardous		
Most fish species	>11	1, 4
Microorganisms	>300	8

Reduced survival, chronic exposure		
Bivalve molluscs, larvae	>14	1
Fish, many species	30–150	1, 5
Impaired reproduction, sensitive species of fish	>25	2
Impaired swimming ability, growth, development, and behavior	>100	3, 6
Lethal to rapidly lethal, acute exposure	300–1,000	5
Marine organisms		
Effect levels, in µg/L seawater		
Adverse effects, chronic exposure	>2	7
Minimal risk	<5	1
Hazardous	>10	1
Lethal	>30	7
Sediments, Great Lakes		
Effect level, in mg total cyanide/kg dry weight (DW)		
Nonpolluted	<0.10	20
Moderately polluted	0.1–0.25	20
Heavily polluted	>0.25	20
Birds		
Domestic chickens		
Diet, safe level, in mg total cyanide/kg ration fresh weight (FW)	90–<100	9, 10
Waterfowl		
Drinking water, safe	<50	21, 22
Livestock		
level in mg/L total cyanide		
Diet, safe level, in mg/kg FW		
Free cyanide	<100	9
Total cyanide	<625	11
Forage, hazardous level, in mg/kg FW	>200	8
Laboratory white rat		
Diet, safe level, in mg/kg ration FW	<1,000	19
Blood, in mg/L		
Normal	0.25–0.45	12
Minimum lethal concentration	2.6–2.9	12
Liver		
Minimum lethal concentration, in mg/kg FW	0.5–6.1	12
Human health		
Drinking water, in µg/L		
Recommended	<5–<10	1, 6, 8, 13
United States nationwide survey	Max. 8	7
Safe	<10	1
Goal, United States	<10	7, 14
Maximum allowable limit, United States	10	13
Goal, Canada	<20	7
Lifetime health advisory, United States and Canada	<154	14

Acceptable	<200	7
Mandatory limit	200	13
Rejected	>200	1, 8
10-day health advisory		
Child	<220	14
Adult	<770	14
Diet		
Acceptable daily intake		
Water	1.5 mg, equivalent to 0.02 mg/kg body weight (BW) daily for 70-kg adult	15
Food, in mg/kg BW	8.4	7
Food, in mg/kg FW	<50	15
Food, in mg total cyanide/kg FW	<415	11
Cassava, *Manihot esculenta*, roots, total cyanide, in mg/kg FW		
Safe	<50	16
Moderately toxic	50–100	16
Very poisonous	>100	16
Food items, in mg/kg		
Cocoa	<20 DW	13
Beans, nuts	<25 DW	1
Cereals, grains	<25 DW	13
Citrus fruits	<50 FW	1
Uncooked pork	<50 FW	13
Grains	<75 FW	1
Cereals flours	<125 DW	13
Spices	<250 FW	1, 13
Frozen meat	<950 FW	1, 13
Bakery products, yeast	<1,500 DW	13
Egg white solids	<1,000 DW	13
Tissue residues		
Blood and spleen, in µg/L or µg/kg FW		
Normal	77	17
Suspected poisoning	>1,000	17
Whole blood, in µg/L		
Usually fatal	1,000–2,000	15
Whole body, in mg/kg BW		
Fatal	4, if taken rapidly	18
Air, in mg/m^3		
Recommended safe levels		
Soviet Union, Romania, Hungary, Bulgaria, Czechoslovakia	<0.3	1
United States	<5	14
Most countries	<11	1, 15
Occupational exposure		
Proposed safe level, United States	<3	15
Safe ceiling concentration	<5	1
Hazardous levels	4.2–12.4	1
Soils in mg/kg DW		
Free cyanide		
Background	1	20
Moderate contamination	10	20
Requires cleanup	100	20
Complex cyanide		
Background	5	20

Moderate contamination	50	20
Requires cleanup	100	20

[a]1, Towill et al. 1978; 2, Smith et al. 1979; 3, Doudoroff 1976; 4, Leduc 1981; 5, Leduc 1984; 6, Leduc et al. 1982; 7, EPA 1980; 8, Egekeze and Oehme 1980; 9, Gomez et al. 1983; 10, Gomez et al. 1988; 11, Okeke et al. 1985; 12, Egekeze and Oehme 1979; 13, EPA 1973; 14, EPA 1989; 15, Marrs and Ballantyne 1987; 16, Dufour 1988; 17, Gee 1987; 18, Shaw 1986; 19, Tewe 1982; 20, Beyer 1990; 21, Allen 1990; 22, Clark and Hothem 1991.

In aquatic systems research is needed in several areas: (1) long-term effects of cyanide on life cycles, growth, survival, metabolism, and behavior of a variety of aquatic organisms and microorganisms in addition to fish (Towill et al. 1978; Leduc et al. 1982); (2) effects of seasonal pulses of cyanide on aquatic organisms in rural and wilderness areas (Leduc 1984); (3) influence of various environmental parameters (e.g., oxygen, pH, temperature), if any, on adaptive resistance to cyanide (Leduc 1981, 1984); and (4) usefulness of various biochemical indicators of cyanide poisoning, such as cytochrome oxidase inhibition (Gee 1987) and vitellogenin levels in fish plasma (*gairdneri*) (Ruby et al. 1986).

The use of M-44 sodium cyanide capsules for predator control was suspended and cancelled by the U.S. Environmental Protection Agency on 9 March 1972. M-44 use was again permitted by the U.S. Environmental Protection Agency beginning on 4 February 1976, provided that "each authorized or licensed applicator shall carry an antidote kit on his person when placing or inspecting M-44 devices. The kit shall contain at least 6 pearls of amylnitrite and instructions on their use. Each authorized or licensed applicator shall also carry on his person instructions for obtaining medical assistance in the event of accidental exposure to sodium cyanide" (EPA 1976a, 1976b).

Farmers need to be aware of factors that influence the cyanogenic potential of forage crops and to conduct regular inspections of grazing fields for cyanogenic plants. Moreover, hay and silage should be properly cured in order to minimize cyanide content before feeding to livestock (Egekeze and Oehme 1980). Selective breeding of plants with low cyanide content will help reduce livestock poisoning, but the most advisable prevention method at present is to prohibit grazing on fields where cyanogenic plants are present (Egekeze and Oehme 1980). More research seems needed on (1) effects of drought and other factors that may increase the concentration of cyanogenic glycosides in livestock forage plants, (2) mechanisms of cyanide liberation by plants, and (3) effects of cyanide on wildlife and range animals that graze on foliage with high cyanogenic glycoside content (Towill et al. 1978).

Research is needed on low-level, long-term cyanide intoxication in mammals by oral and inhalation routes in the vicinities of high cyanide concentrations, especially on the incidence of skin dermatitis, nasal lesions, and thyroid dysfunction, and on urinary thiocyanate concentrations. These types of studies may provide a more valid rationale in establishing standards and threshold limit values for HCN and inorganic cyanide (Towill et al. 1978; Egekeze and Oehme 1980).

Data are scarce on the carcinogenic, teratogenic, and mutagenic properties of cyanide, and on the distribution and transformation of cyanides in air, land, or water. Additional analysis of available information and more research in these areas is recommended. Finally, more research is needed on cyanide toxicokinetics because cyanide is a very reactive nucleophile that distributes widely through the body, is permeable to cell membranes, and may accumulate in the fetus (Towill et al. 1978).

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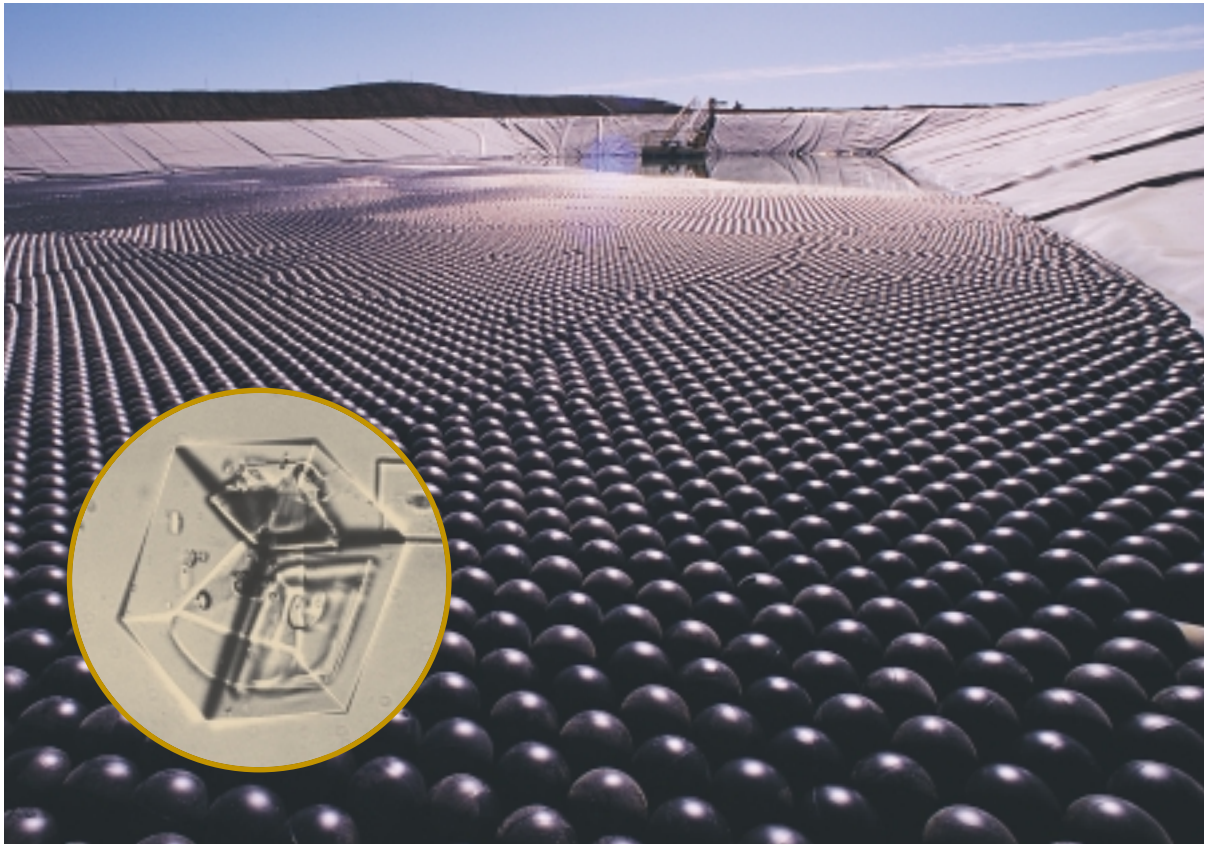
The Management of Cyanide in Gold Extraction

by

Mark J. Logsdon, MSc

Karen Hagelstein, PhD, CIH

Terry I. Mudder, PhD



International Council on
Metals and the Environment

ICME

The International Council on Metals and the Environment (ICME) has published this document as part of its ongoing efforts to provide information on environmental and related health matters affecting the mining and metals sector. The contents of ICME publications range from general and technical information to discussions of policy and regulatory issues. The topics examined may be of interest not only to industry, but also to others, including policy makers, regulators, educators and the public at large. It is hoped that ICME publications provide insight into what are sometimes difficult and complex issues.

Although the views expressed are those of the authors, ICME welcomes questions and comments on the perspectives and information contained in its documents. ICME also appreciates suggestions regarding other issues of public importance for possible future publications.

Founded in 1991, ICME is a non-governmental organization that promotes the development and implementation of sound environmental and health policies and practices in the production, use, recycling and disposal of non-ferrous and precious metals. In addition to its publications, ICME's extensive information program includes a Web site and a quarterly newsletter with a worldwide distribution.

International Council on Metals and the Environment
294 Albert Street, Suite 506
Ottawa, Ontario
CANADA K1P 6E6
Tel: (613) 235-4263
Fax: (613) 235-2865
e-mail: info@icme.com
http://www.icme.com

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Mark J. Logsdon, MSc
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Terry I. Mudder, PhD

ICME

INTERNATIONAL COUNCIL ON
METALS AND THE ENVIRONMENT

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Foreword

The mining industry, and in particular the gold mining industry, has been using cyanide in its production processes for many decades. While cyanide is commonly perceived as being a deadly substance, it is in fact a widely used chemical that is essential to the modern world. The key to its safe use is the implementation of sound management practices.

While public concern about cyanide is valid and indeed understandable, much of the recent media attention and public reaction regarding the use of cyanide in mining operations has arisen due to a lack of understanding of the nature of cyanide and its effects on health and the environment. While there is considerable technical information available to those who produce, transport and use cyanide, easy-to-understand information has not heretofore been provided for a less technical audience. In an attempt to remedy this situation and to address public concern about the use of cyanide in gold extraction, the International Council on Metals and the Environment has commissioned the present document.

The Management of Cyanide in Gold Extraction gives an overview of the chemical's uses and risks, with special emphasis on its use in the recovery of gold. The publication begins by describing the properties of cyanide and its general uses in industry, then moves on to address more specifically the life cycle of cyanide in the mining environment—its production, use in mineral extraction, and general and environmental chemistry. After presenting this information, the publication explains how the principles of risk assessment, risk management and risk communication contribute to the safe use of cyanide in gold recovery.

This work has been prepared by recognized experts and should be a useful reference for anyone involved in decision making related to the presence of cyanide in mining operations, whether from a local or global perspective. It is hoped that international regulators, policy makers, community leaders and all other interested readers, including those engaged in the mining and metals industry, will find the work to be both balanced and informative, and thereby gain a better understanding of the characteristics of cyanide and its unique role in gold recovery.

Gary Nash
Secretary General
ICME

Executive Summary

Cyanide is the chemical of choice for gold recovery.

Cyanide is one of only a few chemical reagents that will dissolve gold in water. It is a common industrial chemical that is readily available at a reasonably low cost. For both technical and economic reasons, cyanide is the chemical of choice for the recovery of gold from ores. Cyanide has been used in metal extraction since 1887 and is now safely used and managed in gold recovery around the world. Gold mining operations use very dilute solutions of sodium cyanide, typically in the range of 0.01% and 0.05% cyanide (100 to 500 parts per million).

Most of the cyanide produced is used as a basic building block for the chemical industry.

Cyanide is produced in large amounts (about 1.4 million tonnes each year) as one of a few basic compounds used chiefly to synthesize a wide range of industrial organic chemicals such as nylon and acrylics. Gold recovery accounts for approximately 18% of total world cyanide production.

Cyanide is produced naturally in a number of microorganisms, insects and plants.

Cyanide is a naturally occurring molecule of carbon and nitrogen. It existed on Earth before life began and was one of the fundamental building blocks in the evolution of life. Low concentrations of cyanide are present in nature, for example in many insects and plants, including a wide range of vegetables, fruits and nuts, where it provides protection against predators. In addition, cyanide is present in much of the everyday environment to which we are exposed, for example in road salt and automobile exhaust and as a stabilizer in table salt.

Cyanide is not persistent.

One of the major health and environmental concerns with some synthetic chemicals is that they do not decompose readily and can thereby accumulate in the food chain. Cyanide, however, is transformed by natural physical, chemical and biological processes into other, less toxic chemicals. Since cyanide oxidizes when exposed to air or other oxidants, it decomposes and does not persist. While it is a deadly poison when ingested in a sufficiently high dose, it does not give rise to chronic health or environmental problems when present in low concentrations.

Cyanide is attenuated through natural processes.

Over time, natural processes such as exposure to sunlight can reduce the concentration of toxic forms of cyanide in solutions to very low values.

The risks of cyanide production, use and disposal can be well managed.

Responsible companies in both the chemical industry and the mining industry employ stringent risk management systems to prevent injury or damage from the use of cyanide. Cyanide in mining solutions is collected, either to be recycled or destroyed, after gold is removed. Managing risks associated with the use of cyanide involves sound engineering, careful monitoring and good management practices in order to prevent and mitigate potential releases of cyanide to the environment.

Communicating information about the risks of cyanide to employees and the public is essential to sound management practices.

The environmental fate of cyanide has been well studied. Cyanide is highly regulated and its risk management is well documented. Risk communication provides information about cyanide both within the operating plant and externally, to the public. Communication of information to the internal staff is the first step in communicating the nature and extent of risk to the general public. Effective communication and emergency planning programs should also be coordinated with the proper local authorities.

SECTION 1

What Is Cyanide?

Cyanide is a general term for a group of chemicals containing carbon and nitrogen. Cyanide compounds include both naturally occurring and human-made (anthropogenic) chemicals. There are more than 2,000 natural sources of cyanide, including various species of arthropods, insects, bacteria, algae, fungi and higher plants. The principal human-made cyanide forms are gaseous hydrogen cyanide and solid sodium and potassium cyanide. Because of its unique properties, cyanide is used in the manufacture of metal parts and numerous common organic products such as plastics, synthetic fabrics, fertilizers, herbicides, dyes and pharmaceuticals.

There is justifiable public concern about the use of cyanide in industrial settings. Cyanide is a toxic substance and can be lethal if ingested or inhaled in sufficient amounts. This is also true for many other chemicals such as gasoline and common household cleaning supplies. As is the case for the thousands of other chemicals used in our modern industrial processes, knowledge, proper handling procedures and a responsible attitude are critical to the safe and beneficial use of cyanide.

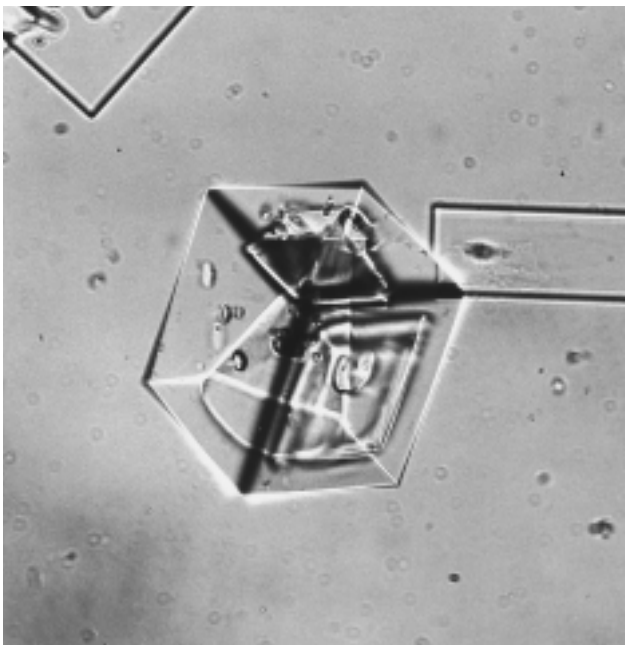


Photo courtesy of DuPont

Microscopic view of sodium cyanide crystals.

Mining is one industrial activity that uses a significant amount of cyanide—about 20% of total production. Since 1887, cyanide solutions have been used primarily to extract gold and silver from ores that otherwise could not be mined effectively. In addition, cyanide is used in low concentrations as a flotation reagent to aid in the recovery of base metals such as lead, copper and zinc.

SECTION 2

Natural Occurrences of Cyanide

Carbon and nitrogen, the two elements that make up cyanide, are present all around us. Together they make up almost 80% of the air we breathe, and both are present in the organic molecules that are the basis of all life forms. Hydrogen cyanide was formed in the earliest stages of the development of our planet as a precursor to amino acids, from which life on Earth evolved. Cyanide is formed naturally. It is produced and used by plants and animals as a protective mechanism that makes them an unattractive food source. Many organisms may either adapt to the presence of cyanide or detoxify it.

A natural source of hydrogen cyanide (HCN) is a sugar-like compound called amygdalin, which exists in many fruits, vegetables, seeds and nuts, including apricots, bean sprouts, cashews, cherries, chestnuts, corn, kidney beans, lentils, nectarines, peaches, peanuts, pecans, pistachios, potatoes, soybeans and walnuts. In the kernel of bitter almond, there is about 1 mg of HCN as amygdalin. Table 1 presents data on the amount of cyanide present in a variety of other foodstuffs.

TABLE 1. Cyanide Concentrations in Selected Plants

Plant Species	**Concentration ($mg.kg^{-1}$)**
Cassava (sweet varieties)	
leaves	377–500
roots	138
dried roots	46–<100
mash	81
Bamboo tip	Max. 8,000
Lima bean (Burma)	2,100
Almond (Bitter)	280–2,500
Sorghum (young plant, whole)	Max. 2,500

Source: Excerpted from Eisler, 1991

Cyanide compounds are produced in thousands of plant species and in other life forms. In some plants, cyanide occurs in concentrations that would be judged “hazardous” if they were associated with manufactured sources. Plants such as alfalfa, sorghum and cassava are known sources of cyanide poisoning to livestock and humans.

In addition to these naturally occurring forms of cyanide, cyanide compounds are also present in such everyday anthropogenic sources as automobile exhaust, cigarette smoke, and even road and table salt.

SECTION 3

Industrial Uses of Cyanide

Cyanide is one of the main building blocks for the chemical industry because of its composition of carbon and nitrogen—both common elements—and the ease with which it reacts with other substances.

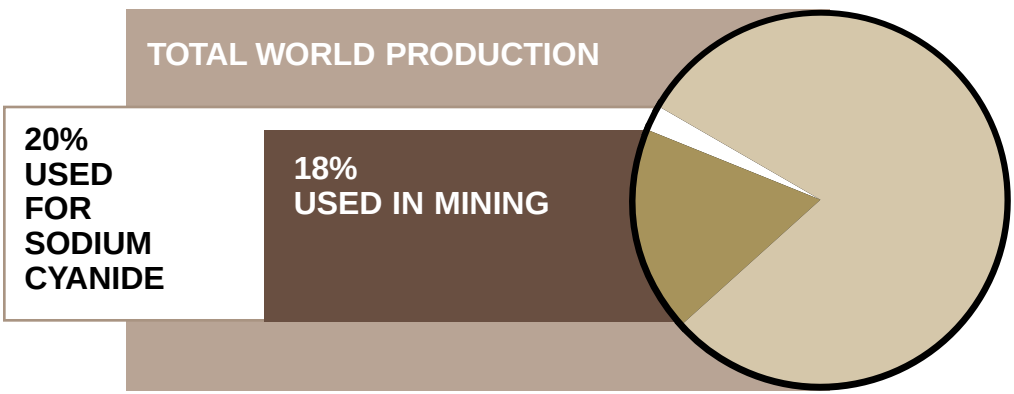
Over one million tonnes of cyanide, representing about 80% of total production, are used annually in the production of organic chemicals such as nitrile, nylon and acrylic plastics. Other industrial applications include electroplating, metal processing, steel hardening, photographic applications and synthetic rubber production.

Iron cyanides are often used in road salt as an anti-caking additive. Hydrogen cyanide vapour has been widely used to exterminate rodents and large predators, and in horticultural practice to control insect pests that have developed resistance to other pesticides.

In addition, cyanide is used in pharmaceuticals such as the anticancer substance laetrile and the blood pressure–reducing drug nitroprusside. Cyanide compounds are also used in surgical dressings that promote healing and reduce scarring.

The remaining 20% of cyanide production is used to manufacture sodium cyanide, a solid form of cyanide that is relatively easy and safe to handle. Of this, 90% (i.e. 18% of total production) is used in mining around the world, mostly for gold recovery.

FIGURE 1. Portion of World Cyanide Production Used in Mining



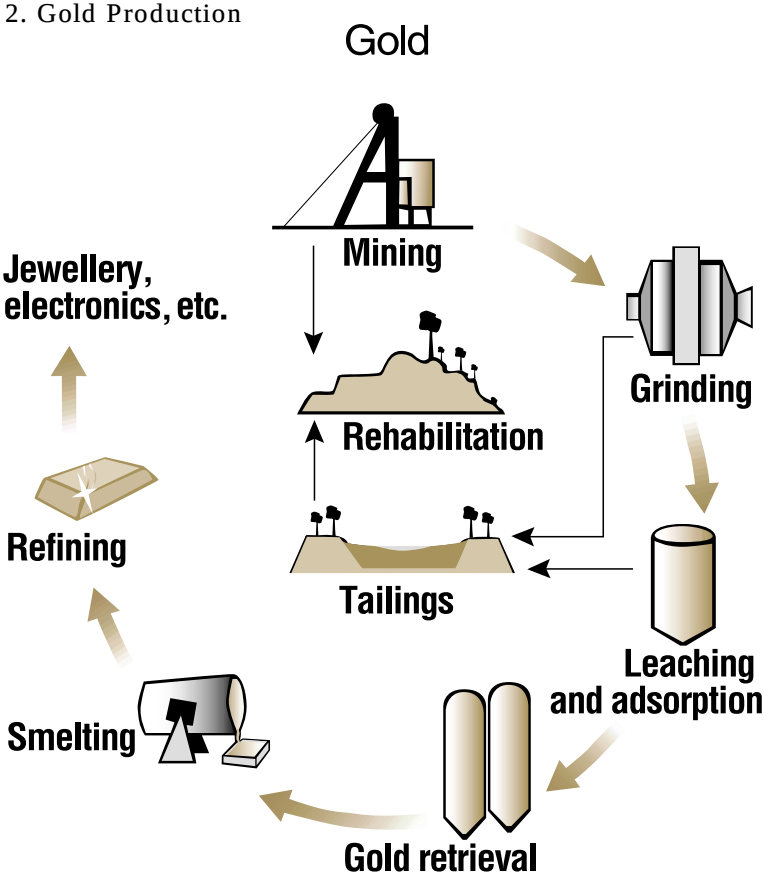
SECTION 4

Cyanide Use in Gold Production

One of the reasons for the high value placed on gold is its resistance to attack by most chemicals. One exception is cyanide, or more specifically, a cyanide-containing solution, which dissolves the precious metal.

Cyanide is used in mining to extract gold (and silver) from ores, particularly low-grade ores and ores that cannot be readily treated through simple physical processes such as crushing and gravity separation.

FIGURE 2. Gold Production



Courtesy of WMC Limited

The Process

The use of water-based solutions to extract and recover metals such as gold is called "hydrometallurgy." Gold mining operations use very dilute solutions of sodium cyanide (NaCN), typically in the range of 0.01% and 0.05% cyanide (100 to 500 parts per million). The process of metal dissolution is called leaching. The sodium cyanide dissolves in water where, under mildly oxidizing conditions, it dissolves the gold contained in the ore. The resultant gold-bearing solution is called "pregnant solution." Either zinc metal or activated carbon is then added to the pregnant solution to recover the gold by removing it from the solution. The residual or "barren" solution (i.e. barren of gold) may be re-circulated to extract more gold or routed to a waste treatment facility. Approaches to treating this waste solution of cyanide are discussed in Section 7.

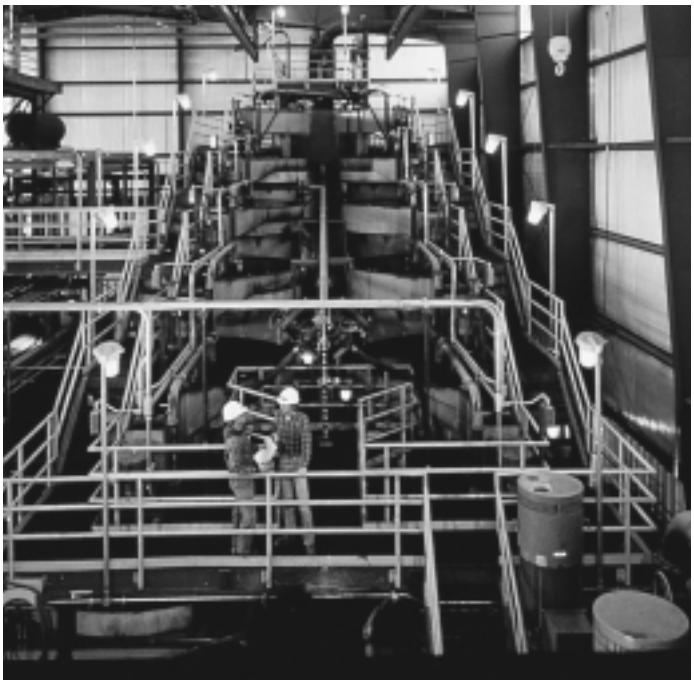


Photo courtesy of Minorco

Gold recovery from cyanide solution using activated carbon (charcoal).

There are two general approaches to leaching gold from mined ore using cyanide: tank leaching and heap leaching.

Tank leaching is the conventional method, in which gold ore is crushed and ground to a size of less than one millimetre in diameter. In some cases, a portion of the gold can be recovered from this finely ground material as discrete particles of gold using gravity-separation techniques. In most cases, the finely ground ore is directly leached in tanks to dissolve the gold in a cyanide solution. When gold is recovered in a conventional plant with leaching in tanks, the barren solution will be collected along with the solid wastes (tailings) in a tailings impoundment system. There, part of the solution will remain within the pores of the settled tailings and part will decant and collect in a pond on top of the tailings, from which it is recycled back to the plant. In most plants, because impurities

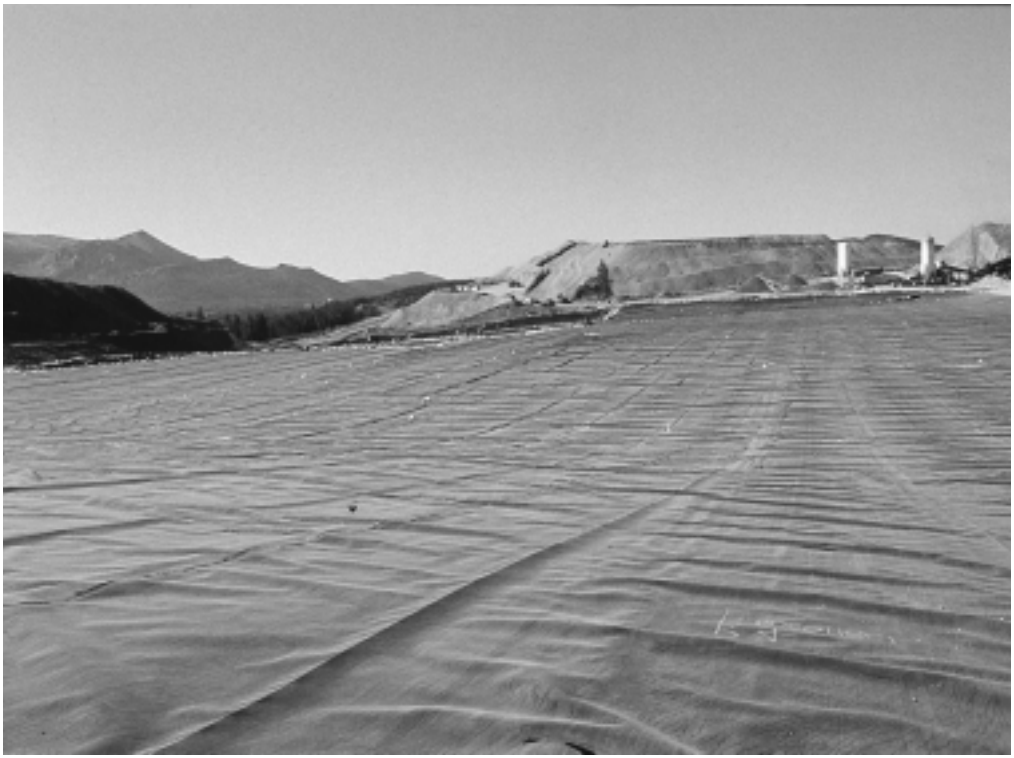


Photo courtesy of Minorco

Construction of a leach pad at Pikes Peak, Colorado, USA.

build up in these solutions, some of the cyanide-bearing solutions must be pumped to a treatment system for disposal (see Section 7).

Recent technical advances enable the heap-leaching of some gold ores. With this method, the ore is crushed to less than a few centimetres in diameter and placed in large piles or heaps. A solution of cyanide is trickled through these heaps to dissolve the gold. When heap-leaching technology is used to extract gold, the barren solution is collected in a pond, from which it is commonly recharged with cyanide and recycled back into the leaching system.

The modern gold industry uses cyanide almost exclusively as the leaching agent for gold. Other complexing agents such as thiourea, chlorides and other halides have been used to extract gold from ores, but these are not generally cost-effective and present their own environmental and health concerns. Cyanide complexes are more stable and effective, and do not require additional aggressive chemicals to effect gold recovery. Cyanide has

been used in mining for over a century *(see box)*. An older technique for gold recovery, which is no longer used in modern gold plants, is amalgamation with liquid mercury. In some developing countries, artisanal miners still use liquid mercury as a means of complexing gold from small mine workings. This practice is discouraged, however, as poor management of both liquid mercury and the vapour arising from volatilizing mercury contributes to serious health problems among artisanal miners.

Box 1. History of Cyanide Use in Mining

While environmental concerns over the use of cyanide in mining have become more public only in the last few years, there actually is a very long history of cyanide use in metallurgical and related processes all around the world. Dippel and Diesbach discovered "Prussian blue" (iron ferrocyanide) in 1704. The earliest well-documented work was Scheele's studies of solubility of gold in cyanide solutions dating from 1783 in Sweden. Gold-cyanide chemistry was studied actively in the mid-19th century in England (Faraday), Germany (Elsner), and Russia (Elkington and Bagration). By 1840, Elkington held a patent for the use of potassium cyanide solutions for electroplating gold and silver. Elsner led the evaluation of the role of oxygen in gold dissolution using cyanide solutions, and "Elsner's Equation" describing the extraction of gold from ores by cyanide was known by 1846.

Patents formalized by McArthur and the Forrest brothers in 1887 and 1888 effectively established the current cyanidation process, the use of cyanide dissolution and precipitation using zinc. However, there were still earlier patents in the USA for cyanide leaching (Rae in 1869) and recovery from chlorinated solutions using charcoal (Davis in 1880). The first commercial-scale cyanidation plant began operating at the Crown Mine in New Zealand in 1889, and by 1904 cyanidation processes were also in place in South Africa, Australia, United States, Mexico and France. Therefore, by the turn of the century, the use of cyanide to extract gold from low-grade ores was a fully established metallurgical technology.

SECTION 5

Production and Handling of Cyanide

Cyanide is produced industrially in one of two ways: as a by-product of the manufacture of acrylic fibres and certain plastics, or by combining natural gas and ammonia at high temperatures and pressures to produce hydrogen cyanide (HCN) gas. Subsequently, hydrogen cyanide gas can be combined with sodium hydroxide (NaOH) to produce sodium cyanide (NaCN) and water (H_2O). The water is then removed by drying and filtering, and the sodium cyanide is formed into solid, white briquettes that are about 10 centimetres square.

The solid sodium cyanide briquettes are maintained under controlled temperature and moisture. At the manufacturing location, the briquettes are packaged in labelled, sealed containers to protect the briquettes from both crushing and moisture. The containers may be disposable plywood boxes with non-returnable liners, non-returnable steel drums, or re-useable steel bins. In some circumstances, the briquettes are dissolved and the cyanide solution is transported as a liquid in specially designed tanker trucks.

All shipments of sodium cyanide are accompanied by Material Safety Data Sheets (MSDSs) that provide the chemistry and toxicity of sodium cyanide, instructions in case of accidents, emergency telephone numbers for assistance and additional information from the manufacturer. All shipments are inventoried as material leaves the producer, and the inventory is checked against delivery records to ensure proper surveillance at all times.

There are three primary producers of solid, liquid and gaseous cyanide in the world: Dupont, in the United States, ICI, in England, and Degussa Corporation, in Germany. Annual worldwide production is approximately 1.4 million tonnes of HCN.[1] As mentioned earlier, 20% of the total HCN production is used to produce sodium cyanide (NaCN) and the remaining 80% is used in numerous other industrial activities such as the production of chemicals. Sodium cyanide is also produced in the USA by FMC Corporation.

The three primary producers are major international chemical manufacturers that understand their responsibility for their products. For example, formal corporate policies

1 1996 amounts. Usage in mining has remained essentially constant for the last decade.

Photo courtesy of DuPont

Storage of drums containing sodium cyanide.

ensure that cyanide is sold only to companies that have the ability and commitment to protect workers, the public and the environment. The manufacturers contract only with selected carriers that have records of transportation safety consistent with the manufacturers' internal standards. The manufacturers maintain a staff of safety and transportation specialists to work with purchasers and others in the areas of training, facility design and related safety measures.

Mining companies store sodium cyanide in secure areas that are kept dry, cool, dark and ventilated. In the storage area, cyanide packages are placed on pallets in their original containers above watertight floors, usually made of concrete, with proper containment in the unlikely event of spillage. Regardless of the container type, empty containers are washed and the rinse water is used in the site's gold recovery plant (to take advantage of

the small amounts of cyanide that could be present) or is processed through the wastewater treatment system prior to being discharged under controlled and permitted conditions.

Mining companies hold special training programs for all employees who work with or around cyanide. They also have materials handling and safety plans prepared by qualified industrial hygienists and supervised by project safety officers. These health and safety plans assign employee responsibilities and control the handling and use of sodium cyanide from its arrival at the mine site through to the metallurgical process. Area gas monitors, proper protective clothing, self-contained breathing apparatus and first-aid stations equipped with eyewash and shower facilities are utilized by cyanide-handling operations at mines. Companies' industrial hygiene programs include annual training, access to all MSDSs and air monitoring to ensure worker safety, as well as procedures for documenting all health and safety information and incidents at mine sites.

Photo courtesy of Degussa Corporation

On-site assistance and safety training are provided to gold mines by cyanide producers.

Modern industrial hygiene programs at gold mining operations have been effective at minimizing accidental cyanide poisoning at mine sites. Indeed, a search of industrial accident records in Australia, Canada, New Zealand and the United States has revealed only three accidental deaths in which cyanide was implicated at gold mine sites in the past 100 years. The first was not directly related to gold recovery, the second involved entry into an enclosed space—a fatal mistake, and the third was not conclusively attributed to cyanide.[2]

2 Both incidents were found in the 107-year fatality database of the Ontario Minister of Labour. In 1952, a blacksmith at the MacLeod-Cockshutt Gold Mines died due to cyanide poisoning following an explosion of molten cyanide; he had been preparing a bath of melted sodium cyanide to case-harden a wrench. In 1961, a worker at the Hallnor Mines Mill died of poisoning from hydrocyanic gas after climbing into an agitator tank to retrieve flake cyanide he had inadvertently thrown into the tank. In 1982, a labourer at an Arizona gold recovery operation collapsed at work and died five days later. Cyanide was suspected, but the evidence as to how the worker became exposed to cyanide was inconclusive.

SECTION 6

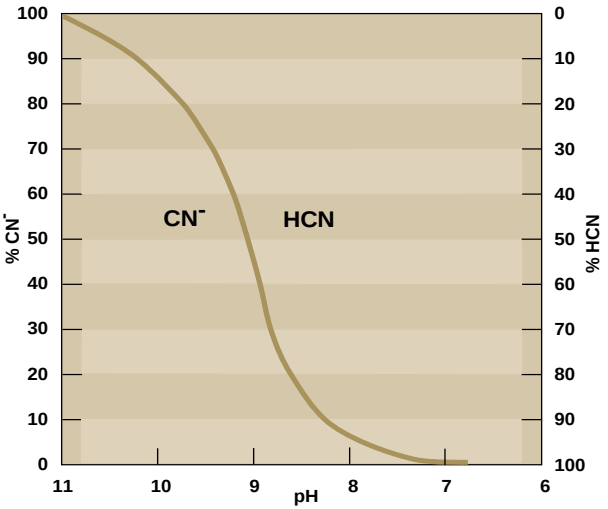
Cyanide in Solutions

After gold is extracted via the hydrometallurgical processes, three principal types of cyanide compounds may be present in wastewater or process solutions: free cyanide, weakly complexed cyanide and strongly complexed cyanide. Together, the three cyanide compounds constitute "total cyanide." An understanding of the chemistry of these three types of cyanide provides insights into their behaviour with respect to safety and the environment.

Free Cyanide

"Free cyanide" is the term used to describe both the cyanide ion (CN^-) that is dissolved in the process water and any hydrogen cyanide (HCN) that is formed in solution. The solid sodium cyanide briquettes dissolve in water to form sodium ion and the cyanide anion (CN^-). The cyanide anion then combines with hydrogen ion to form molecular HCN. The concentration of hydrogen ion in the process water is expressed by the familiar parameter pH.[3] Nearly all free cyanide is present as HCN when there is ample hydrogen ion present, (i.e. at a pH value of 8 or less). This HCN can then volatilize and be dispersed into the air. When the pH is greater than 10.5, there is little hydrogen ion present and nearly all of the free cyanide is present as CN^-. Under normal conditions of temperature and pressure, the concentrations of HCN and CN^- are equal at a pH value of approximately 9.4.

FIGURE 3. CN^-/HCN Equilibrium with pH



Source: Scott and Ingles, 1981.

3 When the pH of a solution is 7, the solution is said to be neutral. Solutions with pH less than 7 are said to be acidic, whereas those with pH greater than 7 are said to be alkaline.

These forms of free cyanide are important because they are considered to be the most toxic cyanides. However, they also happen to be the forms that are readily removed from solutions through both engineered treatment processes and natural attenuation mechanisms. The biological, chemical and physical processes that affect cyanide concentrations in water, soil and air have been extensively studied during the last two decades, so that their behaviour in the environment is well understood.

One of the most important reactions affecting free cyanide concentration is the volatilization of HCN, which, like most gases, will separate from water and escape into the air. Free cyanide is not persistent in most surface waters because the pH of such waters is usually about 8, so that HCN volatilizes and disperses. Hydrogen cyanide's volatility and subsequent transformation to benign compounds in air are important because they act as a natural mechanism for controlling free cyanide concentrations in waste and process waters at mines.

Natural processes alone can reduce the free cyanide concentration from solutions in areas open to the atmosphere in the gold production facilities, such as process ponds and tailings impoundments, to very low values—often to levels below regulatory concern or even the limits of detection.

In the gold plant, however, operators maintain the solution pH at values near 10.5 in order to prevent volatilization. This preserves cyanide in the gold extraction system where it is needed and at the same time limits the risk of worker inhalation exposure to high concentrations of HCN gas in a confined space.

Barrick Gold Corporation

Control centre for gold recovery plant (cyanidation).

Cyanide Complexes

While cyanide-bearing solutions are used in mining because they react with gold, they also react with other metals. Gold ores almost always contain other metals, including iron, copper, zinc, nickel and silver as well as other elements such as arsenic. In most ore bodies, the concentrations of other metals typically exceed the concentration of gold by several orders of magnitude. For example, a low-grade gold ore suitable for cyanide leaching might contain 0.5 to 1 gram of gold per tonne (0.5 to 1 part per million [ppm] gold); in contrast, the iron concentration of average crustal rocks is about 3.5% (35,000 ppm). Metals such as copper, zinc and nickel may be present in concentrations ranging

TABLE 2. Analyses of Barren Solutions[4]

	CONCENTRATION RANGE milligrams per litre[5] ($mg.L^{-1}$)
Total Cyanide	50–2000
Arsenic	0–115
Copper	0.1–300
Iron	0.1–100
Lead	0–0.1
Molybdenum	0–4.7
Nickel	0.3–35
Zinc	13–740

from tens to thousands of parts per million. Table 2 shows that significant amounts of other metals may be dissolved when ores containing them are leached with cyanide solutions.

Chemical analyses of process solutions and wastewater derived from the processing indicate that most of the cyanide in solution is chemically linked with metals other than the small amounts of gold or silver. When chemical elements combine in solution to form soluble species, chemists refer to them as "complexes." There is a wide range of chemical and physical interactions between the components of complexes. Some complexes are very stable, whereas others are easily destroyed. Analytical chemists are able to define the relative stability of cyanide complexes of different metals with great precision. The evaluation of the quantity and types of cyanide is important to all aspects of cyanide use. It is particularly important to be able to distinguish both accurately and precisely between the various cyanide compounds to ensure the selection of an effective detoxification methodology.

4 Scott, J. S., *Status of Gold Mill Waste Effluent Treatment*, Report to CANMET, Natural Resources Canada, March 1993.

5 In environmental studies, concentrations of cyanide and other solutes in solutions are ordinarily presented in terms of mass per unit volume, or sometimes as the dimensionless unit "part per million" (ppm). Concentrations in milligrams per litre ($mg.L^{-1}$) are the same as concentrations in grams per cubic metre ($g.m^{-3}$), and both of these are essentially identical to concentrations in ppm (because the density of solutions is usually very close to 1 kilogram per litre [$kg.L^{-1}$]).

Weak and Strong Cyanide Complexes

Conventionally, cyanide chemists distinguish “weak” from “strong” cyanide complexes. The weak cyanide complexes, often referred to as “weak acid dissociable” or WAD cyanide, can dissociate in solution to produce environmentally significant concentrations of free cyanide. The weak complexes include cyanide complexes of cadmium, copper, nickel, silver and zinc. The degree to which these complexes dissociate is dependent largely on the pH of the solution.

Strong cyanide complexes, on the other hand, degrade much more slowly than WAD cyanide under normal chemical and physical conditions. Complexes of cyanide with gold, cobalt and iron are strong and stable in solution. This stability of the gold–cyanide complex is a key factor in the use of cyanide for the extraction of gold from ores. Once gold enters into solution tied to the cyanide, it remains complexed with the cyanide until process conditions are changed in order to remove it from solution. Cobalt is present only in trace amounts but iron is virtually ubiquitous in geological materials. For most mining situations, the strong complexes of cyanide are predominantly iron cyanides.

The rate at which complexes dissociate and release free cyanide into solution depends on several other factors, including the initial concentration of the cyanide complex, the temperature, the pH of the solution, and the intensity of light, especially ultraviolet radiation.

Analysing and Monitoring Cyanide

Cyanide is generally measured by one of two analytical methods: total cyanide analysis or WAD cyanide analysis. The first is used to determine total cyanide in solutions, including free cyanide and metal-bound cyanides, such as the more stable, non-toxic iron cyanides. The analytical procedure for determining WAD cyanide is used for free and complexed forms of cyanide, except iron cyanide. An older but still used alternative method to that of WAD cyanide analysis is called “cyanide amenable to chlorination.”

Cyanide analyses are needed for operational control, regulatory compliance and toxicity evaluation, as well as for public information about the handling of hazardous materials. Monitoring cyanide both during and after the gold recovery process is essential to good operating practice and the protection of both health and the environment. Rigorous sampling protocols and analytical procedures are required to ensure the quality of information available for decision making. This requires excellent planning and performance from trained personnel working with well-designed and well-managed systems.

SECTION 7

Attenuation of Cyanide Concentrations in the Environment

As explained in Section 4, once gold has been recovered, the solution becomes barren of gold but still contains cyanide. The processes that decrease the concentration of cyanide in solution, whether in the natural environment or in engineered facilities, are called "attenuation." Volatilization of HCN, which reduces the concentration of free cyanide in solution, is the prominent natural attenuation process. Figure 4 provides a schematic representation of the relationships between forms of cyanide and the processes controlling them.

Over the past two decades, the chemical and mining industries have made major advances in handling waste cyanide solutions so that they will not harm public health or the environment. Two technologies are used, often in combination: treatment and recycling.

Cyanide Solution Treatment and Re-use

Treatment: Four general forms of cyanide solution treatment are in use:

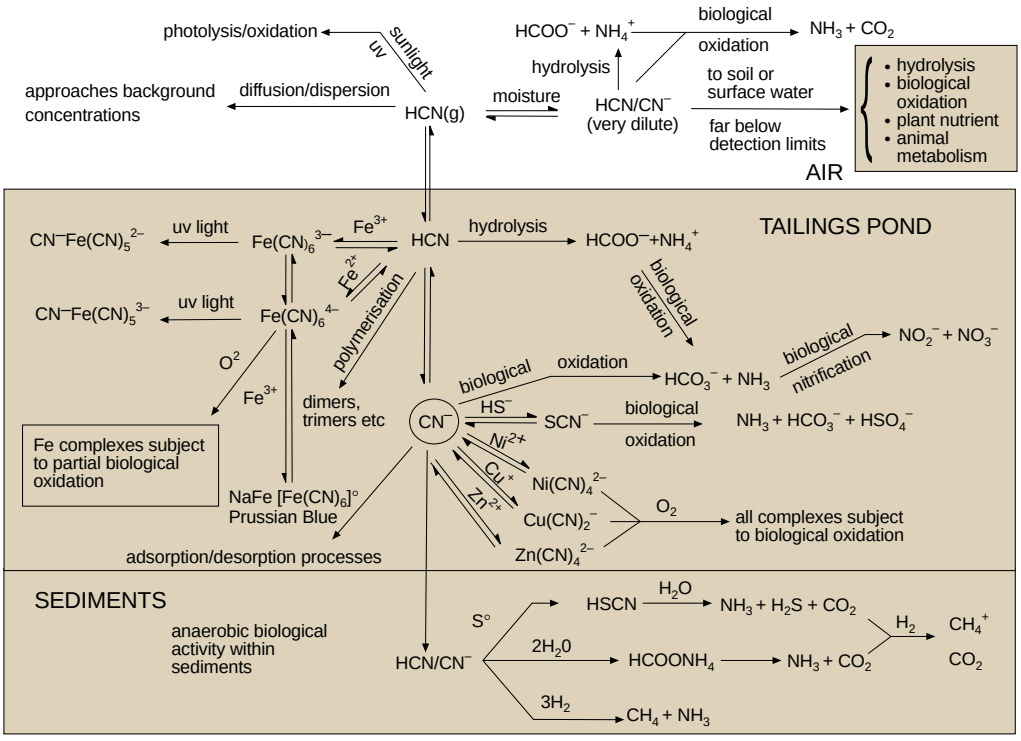
- Natural degradation
- Chemical oxidation
- Precipitation
- Biodegradation

In addition, several technologies enable the re-use of cyanide through recycling.

Natural degradation: The principal natural degradation mechanism is volatilization with subsequent atmospheric transformations to less toxic chemical substances. Other factors such as biological oxidation, precipitation and the effects of sunlight also contribute to cyanide degradation.

Cyanide species may be adsorbed on the surfaces of minerals or organic carbon debris in the soils of a pond embankment, in a clay liner, or along a groundwater flow path. In soils, bacteria assimilate the cyanide through a variety of aerobic and anaerobic reactions. In some instances, the combination of these processes of natural degradation are sufficient to meet regulatory requirements for discharge of cyanide-containing solutions.

FIGURE 4. The Cyanide Cycle



Source: Smith and Mudder, 1991. *Courtesy of Environment Australia*

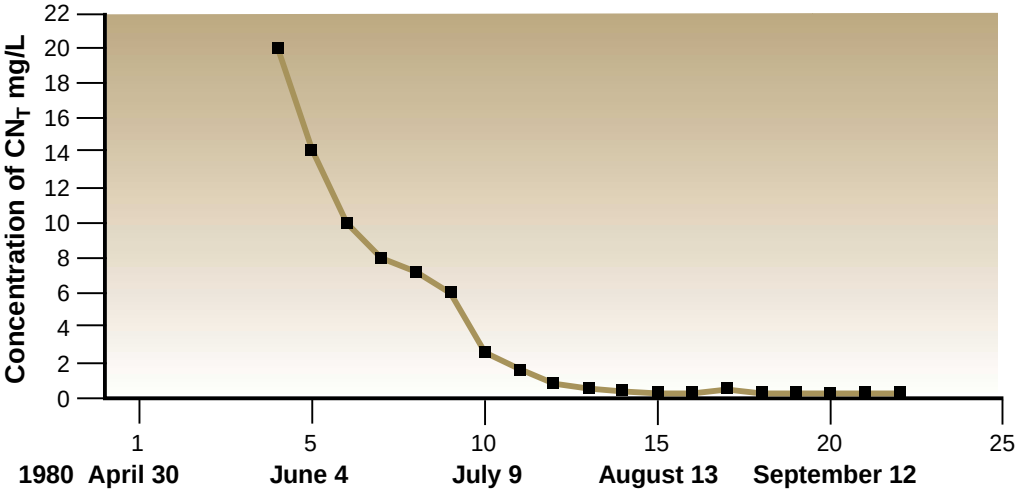
In tailings impoundments, the large surface area enables decomposition of WAD cyanide. Figure 5 illustrates a typical situation in which half of the total cyanide (CN_T) degraded naturally in less than three weeks from the initial concentration of 20 milligrams per litre. The CN_T disappeared almost completely within about 100 days.

Actual degradation rates need to be determined through test work on a site-specific basis using conditions that mimic, as closely as possible, the types of solution and the natural processes that are likely to occur at that location.

Table 3 compiles data from natural degradation systems at a number of gold mines around the world. The values in this table demonstrate the ability of natural degradation to reduce the cyanide concentration of solutions.

Chemical oxidation processes for cyanide treatment include the SO_2/Air process (developed by the Canadian mining company INCO) and the H_2O_2 (hydrogen peroxide)

FIGURE 5. Example of Cyanide Degradation in a Shallow Pond



Source: adapted from Schmidt et al., 1981.

treatment process (pioneered by Degussa). An older chemical oxidation alternative, the Alkaline Chlorination Process, is rarely used in the mining industry today.

In the SO_2/Air process, free and WAD cyanide are oxidized, and iron cyanide is precipitated as an insoluble solid. The process can be applied to either solutions or slurries, and reaction is rapid. Potential limitations are the need to obtain a licence to use the process,

TABLE 3. Natural Degradation of Cyanide in Tailings Impoundments

MINE	**CN entering the tailings system ($mg.L^{-1}$)**	**CN discharging from the tailings system ($mg.L^{-1}$)**
Lupin, NWT, Canada[a]	184	0.17
Holt McDermott, Ontario, Canada[a]	74.8	0.02
Cannon, Washington, USA[b]	284	<0.05
Ridgeway, South Carolina, USA[c]	480	0.09
Golden Cross, New Zealand[d]	6.8 (WAD CN)	0.33 (WAD CN)

Sources: a) Scott, 1993; b) Smith et al., 1985; c) Smith, 1987; d) Smith, 1994

the cost of building a processing plant, the need for empirical testing to optimize the system, and the inability of the process to oxidize intermediate by-products of cyanide.

Hydrogen peroxide, a strong oxidant, oxidizes free and WAD cyanide to ammonium and carbonate. Iron cyanides are not oxidized by peroxide, but precipitate as insoluble and stable solids. Sometimes it is necessary to add chemicals to control the copper concentration of solutions to meet environmental regulations. The peroxide system is not as well suited to the treatment of slurries because of irregular hydrogen peroxide requirements when solids are present.

Both methods of chemical oxidation are capable of producing residual concentrations of cyanide that can meet stringent discharge standards. Both processes require testing on representative samples of site-specific materials prior to the final plant design. Caro's acid, which combines sulphuric acid with hydrogen peroxide to form H_2SO_5, is also used as an oxidation agent to decompose cyanide in solution.

Precipitation of stable cyanides can be achieved by the deliberate addition of complexing agents such as iron. This reduces the free cyanide concentration and is also effective in controlling elevated levels of other metals that may be present. Iron cyanides may react with other chemicals in solution and produce solid precipitates, which may contain a dozen insoluble cyanide salts, thereby removing cyanide from solution. Some of the cyanide in process solutions will react with other chemical components within the system to form much less toxic concentrations of compounds such as ammonia, nitrate and carbon dioxide.

Biodegradation of cyanide is the basis for industrial wastewater treatment systems such as those used by Homestake Mining Company in the United States and ICI Bioproducts in the United Kingdom. A biological process has been used to treat cyanide to meet environmental discharge criteria for more than a decade at the Homestake Mine in Lead, South Dakota. Aerobic conditions are much more favourable to cyanide degradation than are anaerobic conditions, although anaerobic organisms can be effective in treating cyanide at concentrations of up to several milligrams per litre. Both active and passive biological treatment systems have been built—these systems remove cyanide using either aerobic or anaerobic micro-organisms.

At Homestake, the gold-mill barren solution is channelled through reaction vessels containing bacteria. These use oxygen from air to decompose cyanide compounds into nitrates, bicarbonates and sulfates. This microbial process is capable of oxidizing metal cyanide complexes, the metal ions from the WAD cyanide species and intermediate by-products of cyanide oxidation.

Advantages of the biological treatment process are its simple design and operational process control, low chemical costs and capacity of treating all forms of cyanide and its by-products. Potential limitations of biological treatment systems include reduced performance at cold temperatures and at very high cyanide concentrations.

Recycling: While the technologies for cyanide management have centred on cyanide destruction in single-pass systems, it is possible to recover and re-use cyanide, thus minimizing the total amount of cyanide used and reducing operational costs for some mines. Recycling lowers cyanide concentrations in waste solutions and decreases the cost of cyanide destruction.

Cyanide recovery and recycling has been used since the 1930s, notably at Flin Flon (Manitoba, Canada), Pachuca (Hidalgo, Mexico) and Golcanda Minerals (Tasmania, Australia). The basic process involves three steps: pH control, volatilization under highly controlled conditions, and capture of the cyanide that has been released. Recent engineering advances have made it a much more attractive prospect than was the case formerly, and cyanide recovery has been adapted in the last decade to treatment of slurries in a patented, commercial process called Cyanisorb. The process is being applied at the Golden Cross Mine (Waikato, New Zealand) and at the Delamar Silver Mine (Idaho, USA). Two additional Cyanisorb plants have recently been started up in Brazil and Argentina.

Research into cyanide recovery continues, including the testing of a treatment approach that separates cyanide complexes from solutions and absorbs them onto polystyrene-resin beads called Vitrokele (the Cyanosave process). Modifications of this process can be applied to either solutions or slurries, and both cyanide and metals can be recovered. The recovered cyanide is then recycled for use in the gold plant. While there have been successful tests of the process at mines in Canada, Australia and the USA, no commercial plant yet exists, and development continues.

SECTION 8

Evaluating and Managing Risks of Cyanide

The comprehensive approach to treating risk is made up of three key activities which occasionally overlap: risk assessment, risk management and risk communication. All three activities will be described in this and the following sections, beginning with risk assessment.

As stated already, it is well known that sodium cyanide and some of its derivatives are poisons and that cyanide compounds are classified as hazardous. Indeed, modern society safely utilizes many substances that are potentially hazardous, thanks to the ability to assess and manage the associated risks. Since the 1970s, it has become common practice to evaluate the risks associated with hazardous processes and materials through a systematic "risk assessment" process. Many of the concepts of risk assessment arose from more general methods developed by the insurance industry. These have their theoretical basis in probability and mathematical statistics. One of the key concepts that has carried over into environmental risk assessment is the fundamental definition of risk as the probability of a defined consequence.

Risk assessment consists of four parts:

1. Hazard identification is defined as the determination of the adverse effects which chemical, physical and biological agents have an inherent capacity or potential to cause to humans and the environment. Physical hazards include combustion, explosivity, flammability and corrosivity. Health hazards are categorized as acute (e.g. skin and eye irritation, lethal effects, asphyxiation) or chronic (e.g. carcinogenicity, sensitization, effects on reproductive system, effects on nervous system, effects on organs). Ecological hazards include mortality (acute) and reduced growth and reproduction (chronic) in representative species.

Hazard identification is only the first step in risk assessment. It is not an appropriate basis upon which to make a risk management decision. However, hazard identification is a critical step commonly carried out before chemicals and products are introduced to the market. In the case of human health and the environment, results of toxicity/ecotoxicity testing and epidemiology data are used to determine hazard.

2. Dose-response evaluation is the determination of the relationship between the magnitude of an administered, applied or internal dose and a specific biological response. The dose is the total amount of a substance administered to, ingested, inhaled or absorbed by an organism under standardized laboratory conditions used for toxicology testing. The end points of toxicity (or dose response) can be expressed as the measured or observed incidence, the percent response in groups of subjects (or population), or the probability of occurrence of a response in a population.

3. Exposure assessment is the evaluation of the pathways by which the hazard may contact a sensitive receiver. The receiver may be a single person, a real or hypothetical population, or a set of ecological recipients such as fish or wildfowl. The exposure assessment determines how and under what circumstances the receiver may be exposed to the hazard. It may also determine the quantities of the hazardous substance and the length of exposure.

4. Risk characterization summarizes the information from hazard identification, dose-response evaluation and exposure assessment into an overall conclusion on risk in a form that is useful to decision makers, legislators, the media and members of the public. Risk characterization provides a quantitative or qualitative description of the potential hazards of a particular exposure. Quantitative risk characterization conveys a numerical estimate of the magnitude of the risk that a substance poses to humans or to the environment. This risk may be expressed as individual risk or population risk. Qualitative risk characterization describes in narrative form the adverse effect or effects associated with exposure to an agent and provides some measure of the evidence for the association.[6]

Health and Environmental Impacts of Cyanide

Complete risk assessments require detailed specifications of the site-specific conditions. In the case of cyanide, its use varies so much that risk can be meaningfully evaluated only if the specific operating procedures at a particular site are considered. Nevertheless, it is possible to describe the hazards posed by cyanide and the potential exposures.

Toxicity and Epidemiology of Cyanide in Humans

Cyanide is a very fast-acting poison that is capable of killing a person within minutes if he or she is exposed to a sufficiently high dose. Humans may be exposed to cyanide by inhalation, ingestion or absorption through the skin. Cyanide prevents oxygen from being used by the cells, causing tissue hypoxia and “cyanosis” (a bluish discolouration of

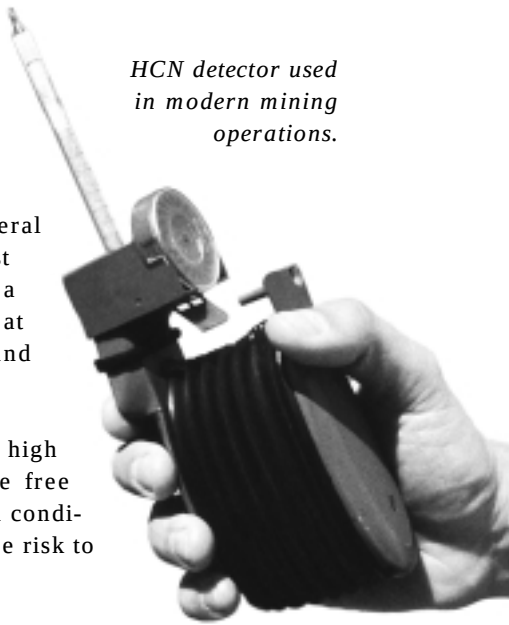
6 From George M. Gray, Jeffery, W. G. and Marchant. G. E., *Risk Assessment and Risk Management of Non-Ferrous Metals: Realizing the Benefits and Managing the Risks*, International Council on Metals and the Environment, 1997.

the skin). The respiratory system fails to nourish the cells with oxygen, a condition which, if untreated, causes rapid, deep breathing followed by convulsions, loss of consciousness and suffocation. The most common antidote is amyl nitrite, which may be taken orally or by injection.

Although there are many everyday sources of exposure to cyanide (automobile exhaust, tobacco smoke, fires), cyanide does not accumulate in tissues because the body transforms such small amounts into a less toxic compound called thiocyanate, which is then excreted. Cyanide is not known to cause cancer or birth defects or adversely affect reproduction.

The most toxic form of cyanide is HCN gas. The American Conference of Governmental Industrial Hygienists (ACGIH) lists the ceiling threshold limit of HCN at 4.7 ppm.[7] At concentrations of 20 to 40 ppm of HCN in air, some respiratory distress may be observed after several hours. Death occurs in minutes at HCN concentrations above approximately 250 ppm in air.

For free cyanide, the lethal dosage to humans by ingestion or inhalation ranges from 50 to 200 mg (1 to 3 mg of free cyanide per kg body mass). The lethal dosage for dermal absorption is considerably higher, at about 100 mg per kg of body weight.



HCN detector used in modern mining operations.

Photo courtesy of DuPont

Worker Exposure

Risk assessments address not only the impacts on the general population, but also the impacts on those who are most likely to be exposed to the hazard, such as the workers at a specific site. The potential for worker contact with cyanide at mines occurs during the receiving, unloading, handling and storage of solid sodium cyanide briquettes.

Provided that the cyanidation process is maintained at a high level of alkalinity (pH of 10.5 or above), almost all the free cyanide is present as CN^- in process solutions. Under such conditions, the volatility of HCN from solutions is low, so that the risk to workers through inhalation is manageable.[8]

7 1998 TLVs and BEIs—Threshold Limit Values for Chemical Substances and Physical Agents, published by the ACGIH.

8 Ingestion of process solution by workers (all of whom are trained and briefed on safety issues) is not considered a credible exposure pathway, because of the unlikelihood of anyone drinking such a solution.

Workers are required to wear respiratory protection against potential airborne hazards. Training in the fitting, use and testing of such equipment is incorporated into the company health and safety procedures. Most modern mining operations have HCN gas detectors or monitors that sound alarms in confined areas where HCN gas may be present. Most humans can detect the odour of hydrogen cyanide gas (a bitter almond smell) at concentrations below those that are hazardous to their health.

Environmental Toxicology and Impacts

Hazardous materials affect not only humans, but also ecological receptors. For mining environments, three groups of ecological or environmental receptors are of concern: mammals, reptiles and amphibians; birds (especially migratory wildfowl); and fish and other aquatic life.

There are few reports of major adverse impacts to animals from cyanide at mining sites. Sodium cyanide and cyanide-bearing solutions are handled in restricted areas of mining sites. Access by animals that walk or crawl is limited by walls, concrete pads, berms and fences, while the presence of humans around the mining facilities also deters animals from approaching. Government evaluations in the USA showed that standard containment designs and good engineering control have effectively mitigated threats to mammals, reptiles and amphibians.[9]

The principal concern for wildfowl has always been exposure to cyanide in open ponds, especially for migratory wildfowl passing through relatively arid regions such as the western USA, where use of cyanide in mining has become quite common. It should be noted, however, that the mortality of birds in Nevada due to exposure to cyanide solutions has been reduced dramatically from about 1,300 in 1990 to 220 in 1995, a decrease of 83%. This improvement is largely due to limiting the WAD cyanide concentration of uncovered ponds to less than 50 ppm. This concentration of WAD cyanide is not acutely toxic to ducks, which are shown to be very sensitive to cyanide as compared with other wildfowl and wildlife.

As a result of effective regulations and good management practice in mining, specific steps have been taken to further limit cyanide concentrations and exposure to wildfowl in open ponds. Netting has been useful in covering small process ponds, but netting of full-scale tailing impoundments is limited due to the weight of the nets, especially with accumulated snow or ice in cold climates, and due to the accidental trapping of wildlife in the nets. However, netting is still practised today for covering ponds in which the cyanide concentrations must be maintained at full strength for metallurgical purposes.

9 General Accounting Office (GAO), 1991.

Other methods of keeping birds away from cyanide solutions in ponds include the use of air cannons, noisemakers, plastic balls or other floating devices increasingly being used to cover the entire surface of small process ponds. This last method also aids in minimizing the loss of free cyanide due to volatilization.

Young, cold-water fish such as salmonids appear to be one of the aquatic species most sensitive to cyanide. Aquatic insects such as stoneflies, caddisflies, mayflies and beetles are generally less sensitive to the substance. It is the weak acid dissociable forms of cyanide that are considered the most "toxicologically significant." Laboratory and field studies have demonstrated that even sensitive aquatic species, such as trout, can tolerate low levels of WAD cyanide. Many discharge permits and regulatory standards are based upon WAD cyanide. In addition, site-specific standards for WAD cyanide have been promulgated for mining operations in such jurisdictions as the United States and New Zealand.

FPhoto courtesy of Placer Dome

Floating "bird balls" cover the surface of a solution containment pond at the Cortez gold mine, a Placer Dome–Kennecott joint venture in Nevada, USA.

SECTION 9

Risk Management for Cyanide in the Mining Industry

There are four major risk scenarios that need to be addressed through site-specific plans:

- Exposure of humans or ecological receptors to cyanide spilled during a transportation accident.
- Exposure of workers, particularly to HCN gas in enclosed areas.
- Exposure of humans through releases of cyanide in solution to surface or ground water that may be ingested.
- Exposure of ecological receptors, such as birds or fish, to cyanide-bearing solutions.

Transportation regulations and diligent safety programs limit the risks associated with the first scenario. As to the second, while adverse impacts from releases of process solutions have occurred in the past, scientific and engineering procedures exist to allow the safe and reliable operation of cyanidation processes. When site-specific standards relating to the third and fourth scenarios are set within the water-quality regulatory framework, protection of human health and the environment can be effectively realized.

Management Systems and Research and Development

Risk management in all of its aspects—from health and safety to prudent financial operations—is understood by today's mining industry to be an integral part of corporate management and a critical factor for the success of an industrial/commercial enterprise. Modern mining companies apply the generalized concept of "management systems" to their programs involving cyanide. Increasingly, this methodology is seen as part of good stewardship in mining, as in other industrial activities. Effective management systems involve four formal steps:

1. Plan: Written plans are prepared to detail the proper handling procedures and the accident response with respect to cyanide transportation and receiving, storage, solution

preparation, metallurgical processes and waste management. Such plans include spill and containment procedures at mining operations as well as health and safety procedures for protecting employees from the potential hazards of cyanide.

2. Execute: For a program to be effective, there must be a commitment to executing the written plans routinely and continuously at every operation. Additionally, each individual employee's responsibilities for executing and documenting the actions required by the plans must be spelled out in detail and clearly defined.

3. Review and document: Part of management's responsibility is to audit performance on a routine basis. The responsibility for reviewing and documenting performance is typically given to persons who are not part of the line operation and who report to a corporate level of authority. This ensures an independent evaluation of the performance of the system. It also ensures that the appropriate level of management in the company is informed about operational performance. The corporate authority may then review and effectively manage the potential risks by implementing policies and programs applicable to multiple sites.

4. Take corrective action, if necessary: Risk management programs may have deficiencies which subsequently become evident in the daily operations and processes. When these are identified in the review process, priority must be given to taking appropriate corrective actions, and the effects of those actions must be reviewed and documented in subsequent audits.

Product Stewardship

The most important aspect of a well-managed system is the understanding that the people in contact with cyanide must take responsibility for its safe use.

Cyanide producers audit purchasers and transportation systems. They also design special packaging for the transport of cyanide. The three primary producers of industrial cyanide, Degussa, Dupont and ICI, have all committed themselves to the principles of Responsible Care®.[10] Truck, rail and barge transporters screen their employees,

10 Responsible Care®, begun in 1985 by the Canadian Chemical Producers' Association (CCPA), is a new ethic for the safe and environmentally sound management of chemicals over their life cycle which has spread to over 40 countries around the world. Under this approach, the CEO or most senior executive of every member of CCPA and of most chemical associations throughout the world must commit to implement the guiding principles and codes of practice of Responsible Care® within three years of joining the association and must agree to submit to public verification. The expectations of members and partners in Responsible Care® go beyond the required implementation of the 151 management practices called for in three codes of practice to include CEO networking via leadership groups, public input through a national advisory panel, mutual assistance through sharing best practices, peer pressure under a conformance process and the public communication of performance improvement measurements.

carefully inventory packages, and establish and maintain systems for loading and unloading. The products are handled and transported according to protocols set by the respective industries and in compliance with national and international regulations.

Mining companies establish inventory control systems, maintain worker training and industrial hygiene programs, as well as build and maintain process-solution and waste-management systems that are specifically designed to mitigate and prevent exposure to cyanide. On a project-specific basis, all risk management components of good product stewardship must be integrated to achieve success.

Conservation and Recycling

Another component of good stewardship of cyanide products is the general concept of waste minimization. By reducing the amount of cyanide physically present at a mining site, the potential exposure pathways are inevitably reduced, and therefore, so is the total risk. Costs as well as risks are reduced when the amount of cyanide used in an operation is kept to the minimum level needed to achieve production goals. This objective requires approaches, such as value engineering, that help to

Photo courtesy of Degussa Corporation

Cyanide producers provide training to ensure safe transportation and handling of sodium cyanide.

Photo courtesy of FMC Corporation

An essential aspect of a well-managed system is that the people in contact with cyanide must take responsibility for its safe use.

conserve the total amount of cyanide used and consumed in a mining process. The advent of cyanide recycling processes provides mining projects with alternatives for conserving the total amount of cyanide required.

Regulations and Voluntary Programs Addressing Worker Safety and Public Health

Regulations, imposed most often by governments, attempt to enforce the management of risks. Examples of regulations in the cyanide life cycle include: (a) establishing packaging and transportation standards; (b) setting industrial hygiene standards for cyanide concentrations in the air and worker safety; and (c) establishing limitations on effluent discharge to surface and ground waters. Governments have used results from research and development and a public-policy process to establish procedures and standards that are protective of worker safety, public health and the environment.

Some examples of regulatory standards for cyanide to protect human health and the environment were given in Section 6. For example, the most toxic form of cyanide, hydrogen cyanide gas, is regulated by industrial hygiene standards such as the ACGIH standards of 4.7 ppm in air.

On a worldwide basis, the total cyanide limit for protection of human health generally is set near the United States Environmental Protection Agency proposed drinking water standard of 0.2 $mg.L^{-1}$. Also, there is an emerging international consensus, based on technical data, that WAD cyanide concentrations in open ponds should be maintained at concentrations of less than 50 $mg.L^{-1}$ to protect migratory birds and other waterfowl against adverse impact.

But the management of risks and its enforcement are not imposed by governments alone, nor need they be. Voluntary programs can have the same effect as regulations without the onus of legal coercion. For example, the major producers of cyanide compounds have made internal decisions to deal only with end users and transportation companies that have proven records of safe performance. While the methods used by each producer may differ, all have the same result of using market mechanisms requiring specific performance criteria to protect the general public from the hazards of cyanide.

SECTION 10

Risk Communication

Risk communication is a key component in any comprehensive program for properly addressing risks related to cyanide in the mining environment. Communication is required both within the operating plant and externally with the public. Internal company education and training of the managers and workers at a site is critical. Employees at a mine or any other industrial facility are also members of the public who live near the site. They and their families, friends and neighbours have many of the same concerns about the safe use of cyanide and about protection of the environment as anyone else living nearby. The proper communication of all cyanide information to the internal staff is therefore the first step in communicating the nature and extent of risk to the general public.

Photo courtesy of Placer Dome

Placer Dome's Sigma Mine, located in Val d'Or, Quebec, Canada.

Beyond complying with formal, regulatory requirements, effective risk communication involves public information and participation. In addition to coordinating emergency planning programs with the proper local authorities, it means giving access to data about the types and quantities of cyanide compounds in the mine's operational processes and inventory, as well as monitoring data. Effective public communication is also bi-directional, encouraging public concerns to be voiced and addressed.

Mine management practices with respect to cyanide should be made public and be implemented through programs which are explained to members of the local communities by company representatives who are effective communicators. Furthermore, positive community relations programs can provide substance as well as form, and serve to show the general population that cyanide and other hazards are being handled safely in the community. Today, a growing number of mining companies around the world have embraced this approach, opening the lines of communication with local communities to the greater benefit of all concerned.

SECTION 11
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About the Authors

Mark J. Logsdon, AB, MSc

Mark J. Logsdon is currently President and Principal Geochemist of Geochimica, Inc., a consulting firm located in Ojai, California, USA. He holds an AB Honors in Geology from Princeton University as well as an MS in Geology from the University of New Mexico. Mr. Logsdon specializes in hydrogeochemistry and environmental geochemistry, particularly related to waste management. In addition to consulting experience, Mr. Logsdon's 30-year career has encompassed research and academic instruction as well as governmental experience, notably with the USA's Nuclear Regulatory Commission and the New Mexico Bureau of Mines and Mineral Resources. Particular skills and experience include designing and implementing multidisciplinary earth science investigations involving waste characterization, geochemistry and hydrology. Geochimica projects include hydrogeology and hydrogeochemistry studies of acid mine drainage, cyanide hydrogeochemistry at mining sites and water-quality site investigations.

Karen Hagelstein, BS, MS, PhD, CIH

Karen Hagelstein is a Partner and Senior Environment Scientist at Times Limited, an environmental science and engineering firm located in Bozeman, Montana, USA, specializing in water and waste-water engineering, aquatic toxicology and human health evaluations. Dr. Hagelstein is a Certified Industrial Hygienist and received her BS from the University of South Dakota, her MS in Physiology and Biophysics from the University of Iowa, and her PhD in Civil and Environmental Engineering from the University of Iowa. During her career of over 15 years, Dr. Hagelstein has held positions as Senior Environmental Scientist, Environmental Engineer, and Health and Safety Officer with a number of consulting firms, and also spent six years as Associate Professor at the South Dakota School of Mines and Technology. Mining-related projects undertaken at Times Limited include reviewing and summarizing human toxicity data and risks with respect to environmental hazard exposures, air-dispersion modelling for estimating pollutant concentrations, and summarizing biological and chemical water quality monitoring data.

Terry I. Mudder, BS, MS, PhD

Terry I. Mudder is co-owner of Times Limited. He has a BS and MS in organic and analytical chemistry, and a PhD in environmental science and engineering. Dr. Mudder has 20 years' experience in the investigation of the chemistry, analysis, fate, aquatic toxicity and disposal of cyanide-bearing wastes. He has served as adjunct professor, thesis advisor and guest lecturer at universities throughout the world. He has worked on over 100 precious metal and non-ferrous mining-related projects on six continents and has written over three dozen technical papers. He has given numerous lectures and been involved with short courses and workshops on cyanide. He has co-authored several pamphlets and books, including *The Chemistry and Treatment of Cyanidation Wastes* and *The Cyanide Monograph*, both published by Mining Journal Books. He has been instrumental in the development and application of many of the chemical, physical and biological treatment processes for cyanide and metals, for which he has received both national and international awards, and obtained worldwide patents. He has provided technical assistance to the BC Ministry of Environment, Environment Australia, the Peruvian Environmental Protection Service, the US EPA, US State regulatory agencies, as well as to several industry-based organizations.

Chemistry and Treatment of Cyanidation Wastes

SECOND EDITION

Terry I. Mudder, Ph.D.
Michael M. Botz, M.S., P.E.
and
Adrian Smith, Ph.D.

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6.12 SULPHUR DIOXIDE AND AIR

6.12.1 Introduction

There are two patented versions of the sulphur dioxide cyanide destruction process. The first patented process and most widely applied is marketed by INCO Ltd. The INCO process is based upon conversion of WAD cyanides to cyanate using a mixture of SO_2 and air in the presence of a soluble copper catalyst at a controlled pH. In the INCO process, the forms of cyanide are removed by different processes. One process involves the conversion of WAD cyanides to cyanate. Iron complexed cyanides are reduced to the ferrous state and precipitated as insoluble copper-iron-cyanide complexes. Residual metals liberated from the WAD cyanide complexes are precipitated as their hydroxides.

The second sulphur dioxide process was developed at Heath Steel Mines Ltd. and the patent assigned to Noranda Incorporated (Ferguson and Walker, 1985). In the Noranda process, pure sulphur dioxide is fed into a solution or slurry to lower the pH into the range of 7.0 to 9.0. A copper sulphate solution is then added at such a rate to yield an effluent containing the desired cyanide concentration.

The INCO process has been used at over 80 mining operations worldwide and is the process addressed in this section. A primary application of the sulfur dioxide and air process is in treatment of tailings slurries, but it is also effective for the treatment of solutions for the oxidation of free and WAD cyanides.

6.12.2 Process Chemistry

Free and weakly complexed metal cyanides (i.e., WAD cyanides) are oxidized to cyanate by sulfur dioxide and air in the presence of a soluble copper catalyst.

(6.32) $$CN^- + SO_2 + O_2 + H_2O \xrightarrow{Cu^{+2}\ Catalyst} OCN^- + SO_4^{-2} + 2H^+$$

(6.33) $$M(CN)_4^{-2} + 4SO_2 + 4O_2 + 4H_2O \xrightarrow{Cu^{+2}\ Catalyst} 4OCN^- + 8H^+ + 4SO_4^{-2} + M^{+2}$$

The reaction is normally carried out at a pH of about 8.0 to 9.0, and due to the formation of acid in the reactions, lime is normally required for pH control. Decreases in process performance can occur if the pH fluctuates outside this optimal range. The optimal pH must be determined experimentally, since maximum cyanide and metals removals occur at different pH values. Temperature has little effect on process performance between 5°C and 60°C.

The theoretical usage of SO_2 in the process is 2.46 grams SO_2 per gram of WAD cyanide oxidized, but in practice the actual usage ranges from about 3.0 to 5.0 grams SO_2 per gram of WAD cyanide oxidized. The SO_2 required in the reaction can be supplied either as liquid sulphur dioxide, sodium sulphite (Na_2SO_3) or as sodium metabisulphite ($Na_2S_2O_5$). Solutions of ammonium bisulphite (NH_4HSO_3) have also been used in the process, but this requires a careful examination regarding the impact ammonia addition will have on the treated effluent. The choice of one reagent versus another is primarily associated with cost and availability.

The approximate lime requirement can be calculated from the above reactions according to the anticipated acid production resulting from SO_2 addition. Oxygen (O_2) is also required in the reaction and this is generally supplied by sparging atmospheric air into the reaction vessels. Required reaction times vary from about 30 minutes to 2 hours.

The soluble copper catalyst is normally added as a solution of copper sulphate pentahydrate ($CuSO_4$-$5H_2O$) to a level of about 10% to 20% of the initial WAD cyanide level. However, in cases where dissolved copper is already present in the tailings solution or slurry, the need for copper sulphate addition may be eliminated.

Iron cyanide removal is initiated by reduction of iron from the ferric to the ferrous state according to the following reaction:

(6.34) $2Fe(CN)_6^{-3} + SO_2 + 2H_2O \rightarrow 2Fe(CN)_6^{-4} + 4H^+ + SO_4^{-2}$

The ferrous cyanide complex is then removed through precipitation with either copper, nickel or zinc according to the following generalized reaction:

(6.35) $2M^{+2} + Fe(CN)_6^{-4} \rightarrow M_2Fe(CN)_6$ (solid)

Trace metals remaining in solution following oxidation of the weakly complexed metal cyanides are precipitated as their hydroxides according to the following generalized reaction:

(6.36) $M^{+2} + 2OH^- \rightarrow M(OH)_2$ (solid)

The oxidation of thiocyanate, which is usually limited to 10% to 20% in the process, and the hydrolysis of cyanate occur according to the following reactions:

(6.37) $SCN^- + 4SO_2 + 4O_2 + 5H_2O \rightarrow OCN^- + 10H^+ + 5SO_4^{-2}$

(6.38) $OCN^- + H^+ + 2H_2O \rightarrow HCO_3^- + NH_4^+$

Generally, the best application of this process is with slurries containing low to moderately high initial levels of cyanide when treated cyanide levels of less than about 5 mg/L are required. In some cases, solutions treated with this process may be of suitable quality to permit their discharge.

With regard to oxidant supply, sodium sulphite (Na_2SO_3) or sodium meta-bisulphite ($Na_2S_2O_5$) are supplied as powders and must be dissolved in concentrated form prior to use. As a result, a dissolution tank and a chemical storage and feed system are necessary. In the case sulphur dioxide is used, it is usually delivered as a bulk liquid or supplied in one-ton cylinders. Sulphur dioxide gas can also be generated on-site by burning pure sulphur and collecting the combustion products into an acidic solution using a scrubber tower. Exhaust gases can also be directly injected into the slurry or solution without intermediate scrubbing.

In a typical two-stage process configuration, the sulphur dioxide, lime and copper sulphate are introduced into the first stage to complete the oxidation of cyanide. Additional lime or other chemicals (e.g., ferric chloride) are added to the second reactor to maximize metals precipitation. This approach is needed in some instances since the complete oxidation of WAD cyanide requires a lower pH than does the precipitation of metals from the solution or slurry. The flowsheet for a typical two-stage process is shown on Figure 6.21.

The primary process variables include retention time, air feed rate, copper dosage, pH and sulphur dioxide feed rate. The quantity of sulphur dioxide or other reagent used is adjusted based on the WAD cyanide concentration in treated solution, and determined through laboratory and/or pilot plant evaluations. The copper requirement for the process is also determined experimentally. Generally, the copper dosage can be held to <50 mg/L, unless elevated iron concentrations are present which demand additional copper for iron cyanide precipitation. Laboratory evaluations of the process are generally conducted in one or two reaction vessels placed in series. Sulphur dioxide or another source of oxidant is added, either pre-mixed in air or separately as a sulphite solution. The tests are completed at various reagent dosages and pH values to determine the optimal reaction conditions and achievable level of treatment.

6.12.3 Performance

The performance of the INCO process at varying levels of copper addition and pH values is shown on Figure 6.22. As indicated, the process was found to be most effective with a copper concentration above about 10 mg/L and at a pH in the range of about 6.0 to 10.0. Summaries of the cyanide destruction performance and reagent usages achieved for the treatment of several tailings slurries using the INCO process are presented in Table 6.30. A summary of the cyanide destruction performance and reagent usages for the treatment of several barren solutions and decant waters are presented in Table 6.31, and process performances for treatment of three plating solutions are presented in Table 6.32.

Capital costs for the process depend upon whether a slurry or solution is being treated, the level of WAD cyanide, iron cyanide and the concentration of copper in the untreated material. The primary capital items include the reactor(s), agitator(s), an air compressor and piping, a feed system for the sulphur dioxide source (i.e., for either sulphur dioxide, sodium sulphite, or sodium metabisulphite), a copper sulphate storage and feed system, and a slaked lime preparation, storage and feed system.

Operating costs include labour, reagents, electrical power and maintenance. Lower treatment costs are associated with treatment of solutions containing low cyanide levels, while the higher costs are associated with treatment of tailings slurries and higher levels of cyanide. The INCO process is patented technology and does require a license and user fee.

The various advantages and disadvantages of the INCO sulphur dioxide process are presented in Table 6.33.

FIGURE 6.21 Flowsheet for a Two-Stage INCO Cyanide Destruction Process

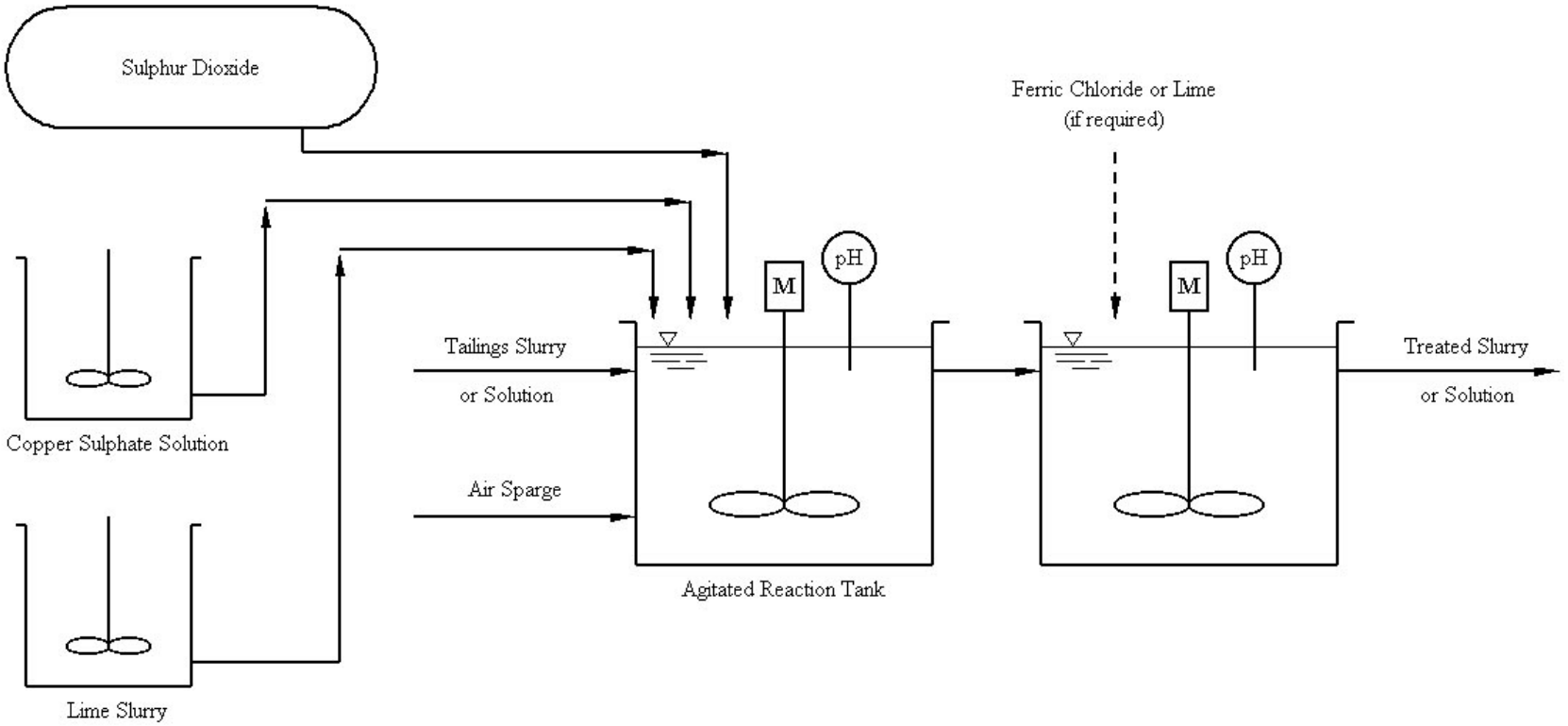
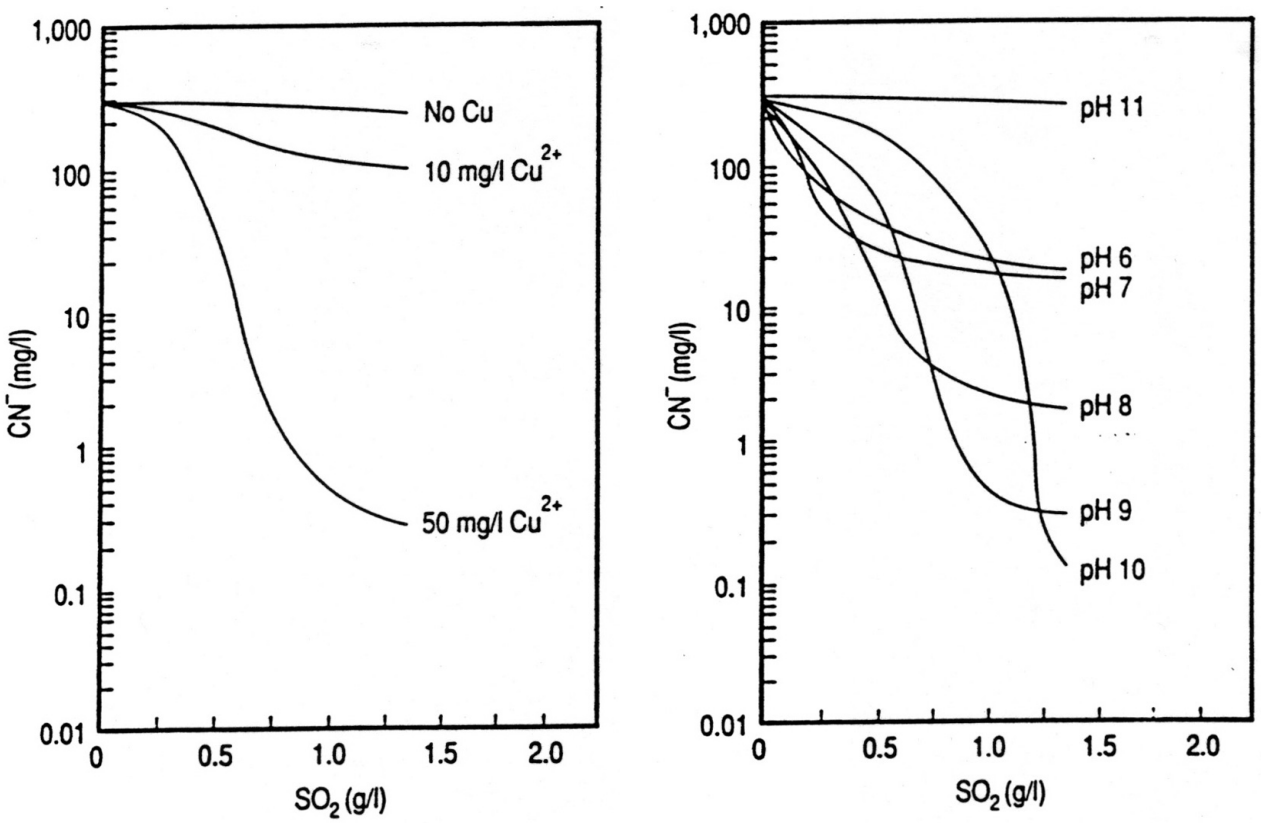


FIGURE 6.22 The Effects of Copper Concentration and pH on the Performance of the INCO Cyanide Destruction Process



Source: Robbins, 1996

TABLE 6.30 Oxidation of Cyanide in Tailings Slurry Using the INCO SO_2/Air Process

	CN_{TOT} Assay (mg/l)		Reagent Usage (g/g CH_{TOT})		
Mine	**Before**	**After**	**SO_2**	**Lime**	**Cu^{+2}**
Colosseum	364	0.4	4.6	0.12	0.04
Ketza River	150	5.0	6.0	0	0.30
Equity	175	2.3	3.4	0	0.03
Casa Berardi	150	1.0	4.5	--	0.10
Westmin Premier	150	<0.2	5.8	--	0.12
Golden Bear	205	0.3	2.8	--	--

Source: Devuyst et al., 1989a, 1989b and 1991

TABLE 6.31 Oxidation of Cyanide in Solutions Using the INCO SO_2/Air Process

	CN_{TOT} Assay (mg/l)		Reagent Usage (g/g CH_{TOT})		
Mine	**Before**	**After**	**SO_2**	**Lime**	**Cu^{+2}**
McBean (barren)	370	0.2	4.0	4.0	0
Lynngold (pond)	106	0.6	7.0	9.0	0.12
Mineral Hill (barren)	350	0.5	6.0	9.0	0
Lac Short (pond)	10	0.5	5.0	--	0
Citadel (barren)	350	5.0	4.0	--	0
St. Andrew (pond)	15	1	5.0	--	0.10

Source: Devuyst et al., 1989a, 1989b and 1991

TABLE 6.32 Oxidation of Cyanide in Electroplating Wastes Using the INCO SO_2/Air Process

	CN_{TOT} Assay (mg/l)		Reagent Usage (g/g CH_{TOT})		
Mine	**Before**	**After**	**SO_2**	**NaOH**	**Cu^{+2}**
Kuntz	150	0.2	6.0	--	--
Precious Plate	30,300	60	2.7	0.6	0
Superfinish	640	1.3	3.4	--	0.02

Source: Devuyst et al., 1989a, 1989b and 1991

TABLE 6.33 Advantages and Disadvantages of the INCO SO_2/Air Process

Advantages	
1	The process has been proven in numerous full-scale applications to yield low effluent cyanide and metals concentrations.
2	The process is effective in treating slurries as well as solutions.
3	The process is suitable for batch or continuous treatment.
4	All forms of cyanide are removed from solution, including the stable iron cyanide complexes.
5	Capital and operating costs are comparable with other chemical treatment processes.
Disadvantages	
1	If treating high levels of cyanide, the costs for reagents and electrical power can be high.
2	Cyanide is not recovered.
3	Undesirable levels of sulphate in the treated solution can result.
4	Additional treatment may be necessary for the removal of iron cyanide, thiocyanate, cyanate, ammonia, nitrate and/or metals for solutions to be discharged to the environment.

United States
Environmental Protection
Agency

Office of Water
Regulations and Standards
Criteria and Standards Division
Washington, DC 20460

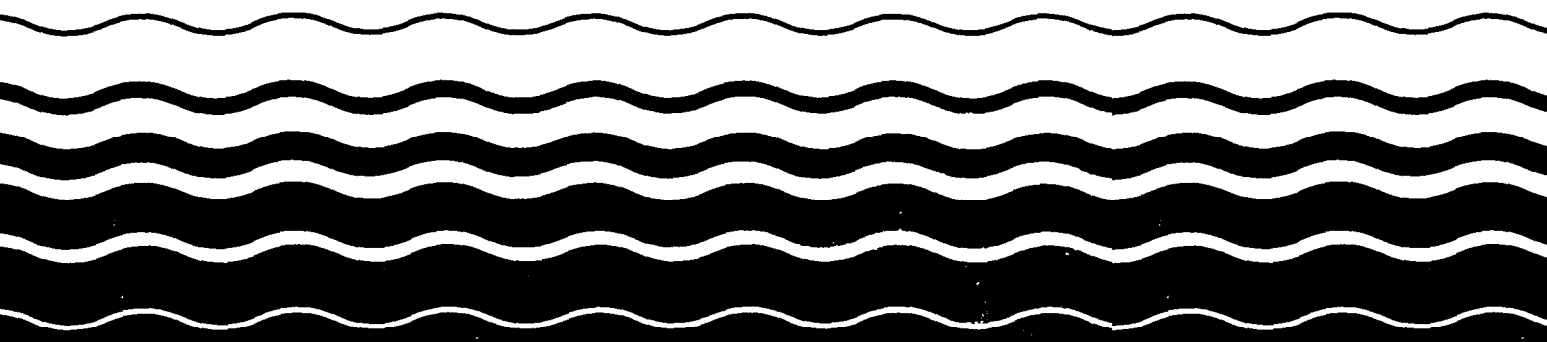
EPA 440/5-84-028
January 1985

Water

EPA

Ambient Water Quality Criteria for

Cyanide - 1984



AMBIENT AQUATIC LIFE WATER QUALITY CRITERIA FOR

CYANIDE

U.S. ENVIRONMENTAL PROTECTION AGENCY
OFFICE OF RESEARCH AND DEVELOPMENT
ENVIRONMENTAL RESEARCH LABORATORIES
DULUTH, MINNESOTA
NARRAGANSETT, RHODE ISLAND

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FOREWORD

Section 304(a)(1) of the Clean Water Act of 1977 (P.L. 95-217) requires the Administrator of the Environmental Protection Agency to publish criteria for water quality accurately reflecting the latest scientific knowledge on the kind and extent of all identifiable effects on health and welfare which may be expected from the presence of pollutants in any body of water, including ground water. This document is a revision of proposed criteria based upon a consideration of comments received from other Federal agencies, State agencies, special interest groups, and individual scientists. The criteria contained in this document replace any previously published EPA aquatic life criteria.

The term "water quality criteria" is used in two sections of the Clean Water Act, section 304(a)(1) and section 303(c)(2). The term has a different program impact in each section. In section 304, the term represents a non-regulatory, scientific assessment of ecological effects. The criteria presented in this publication are such scientific assessments. Such water quality criteria associated with specific stream uses when adopted as State water quality standards under section 303 become enforceable maximum acceptable levels of a pollutant in ambient waters. The water quality criteria adopted in the State water quality standards could have the same numerical limits as the criteria developed under section 304. However, in many situations States may want to adjust water quality criteria developed under section 304 to reflect local environmental conditions and human exposure patterns before incorporation into water quality standards. It is not until their adoption as part of the State water quality standards that the criteria become regulatory.

Guidelines to assist the States in the modification of criteria presented in this document, in the development of water quality standards, and in other water-related programs of this Agency, have been developed by EPA.

Edwin L. Johnson
Director
Office of Water Regulations and Standards

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Steven J. Broderius
(freshwater author)
Environmental Research Laboratory
Duluth, Minnesota

John H. Gentile
(saltwater author)
Environmental Research Laboratory
Narragansett, Rhode Island

Charles E. Stephan
(document coordinator)
Environmental Research Laboratory
Duluth, Minnesota

David J. Hansen
(saltwater coordinator)
Environmental Research Laboratory
Narragansett, Rhode Island

Clerical Support: Terry L. Highland

CONTENTS

TABLES

Introduction*

Compounds containing the cyanide group (CN) are used and readily formed in many industrial processes and can be found in a variety of effluents, such as those from the steel, petroleum, plastics, synthetic fibers, metal plating, mining, and chemical industries. Cyanide occurs in water as hydrocyanic acid (HCN), the cyanide ion (CN^-), simple cyanides, metallocyanide complexes, and as simple chain and complex ring organic compounds (Callahan, et al. 1979). "Free cyanide" is defined as the sum of the cyanide present as HCN and as CN^-, and the relative concentrations of these two forms depend mainly on pH and temperature. When pH is below 8 and temperature is below 25 C, at least 94 percent of the free cyanide exists as HCN. When pH or temperature or both are higher, a greater percentage of free cyanide exists as CN^-. For example, when pH is 9 and temperature is 30 C, about 55 percent of the free cyanide exists as HCN.

Although simple cyanides such as sodium cyanide and potassium cyanide readily dissociate and hydrolyze to form CN^- and HCN, the metallocyanide complex anions have a wide range of stabilities. Zinc and cadmium cyanide complexes dissociate rapidly and nearly completely in dilute solutions, whereas the stability of the copper and nickel metallocyanide anions are pH-dependent. Cyanide complexes of iron dissociate very little, but they are subject to photolysis by natural light. Release of cyanide ion by photodecomposition might be important in relatively clear receiving waters.

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*An understanding of the "Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and Their Uses" (Stephan, et al. 1985), hereafter referred to as the Guidelines, is necessary in order to understand the following text, tables, and calculations.

The apparent toxicity to aquatic organisms of most simple cyanides and metallocyanide complexes is due mainly to the presence of HCN derived from dissociation, photodecomposition, and hydrolysis (Doudoroff, et al. 1966; Smith, et al. 1979), although CN^- is apparently also toxic (Broderius, et al. 1977). Most metallocyanide complexes are not very toxic. The available literature on the toxicity of cyanides and related compounds to fish was critically reviewed by Doudoroff (1976, 1980). Additional reviews on the environmental effects of cyanides have been prepared by Leduc (1984), Leduc, et al. (1982), and Towill, et al. (1978).

Because (a) both HCN and CN^- are toxic to aquatic life, (b) the vast majority of free cyanide usually exists as the more toxic HCN, and (c) CN^- can be readily converted to HCN at pH values that commonly exist in surface waters, cyanide criteria will be stated in terms of free cyanide expressed as CN. Free cyanide is a much more reliable index of toxicity to aquatic life than total cyanide because total cyanide can include nitriles (organic cyanides) and relatively stable metallocyanide complexes. In highly alkaline waters a criterion that takes into account the relative toxicities of HCN and CN^- may be appropriate due to the dependence of the form of free cyanide on pH.

If performed often enough over a wide enough geographical area, measurement of free cyanide (ASTM, 1984; Broderius, 1981) should be adequate for monitoring cyanide in a body of water. However, because dissociation of several metallocyanide complexes is very dependent on pH in the range that commonly occurs in many water bodies, a measurement such as (a) free cyanide at the lowest pH occurring in the receiving water or (b) cyanide amenable to chlorination or total cyanide (U.S. EPA, 1983a) is probably more appropriate

if only a few measurements are made on a water body and whenever measurements are made on an effluent. Dilution of an effluent with receiving water before measuring cyanide should demonstrate whether the receiving water can decrease the cyanide of concern because of sorption or complexation. Some measurements of total cyanide in the receiving water or effluent or both are desirable because if total cyanide is much higher than free cyanide or cyanide amenable to chlorination, the importance of release of cyanide from metallocyanide complexes by photolysis should receive consideration.

All cyanide concentrations reported herein are in terms of free cyanide expressed as CN. Thus, data reported in the original literature in terms of free cyanide expressed as CN did not have to be adjusted. However, when free cyanide was expressed as HCN, KCN, etc., the results were adjusted using the molecular weights of the compound and CN. When data were reported in the original literature in terms of HCN, rather than in terms of free cyanide, the data were converted from molecular HCN to free cyanide as CN as follows:

$$(\mu g \text{ of free cyanide as CN/L}) = (\mu g \text{ of HCN/L}) \left(1 + 10^{pH-pK_{HCN}}\right) \times \frac{\text{mol. wt. CN}}{\text{mol. wt. HCN}}$$

where $pK_{HCN} = 1.3440 + \frac{2347.2}{T + 273.16}$ (Izatt, et al. 1962)

and T = degrees Celsius. The criteria presented herein supersede previous aquatic life water quality criteria for cyanide (U.S. EPA, 1976, 1980) because these new criteria were derived using improved procedures and additional information. Whenever adequately justified, a national criterion may be replaced by a site-specific criterion (U.S. EPA, 1983b), which may include not only site-specific criterion concentrations (U.S. EPA, 1983c), but also site-specific durations of averaging periods and site-specific frequencies of allowed exceedences (U.S. EPA, 1985). The latest literature

search for information for this document was conducted in May, 1984; some newer information was also used.

Acute Toxicity to Aquatic Animals

Most of the invertebrate species tested were considerably more resistant than fishes, but Daphnia sp. and Gammarus pseudolimnaeus were comparable to fishes in sensitivity. On the other hand, about half of the tests with invertebrate species were static and the test concentrations were not measured, whereas many of the tests with fish were flow-through tests in which free cyanide concentrations were measured (Table 1).

Certain life stages and species of fish appear to be more sensitive to cyanide than others. Embryos, sac fry, and warmwater species tended to be the most resistant. Free cyanide concentrations from about 50 to 200 µg/L eventually were fatal to juveniles of most of the more sensitive fish species, with concentrations much above 200 µg/L being rapidly fatal to most juvenile fish. Thus, there is a relatively narrow range of species sensitivity for fish. A comparison of acute toxicity values for fishes (Table 1) supports the conclusion (Doudoroff, 1976) that results of static toxicity tests tend to be somewhat higher than results of renewal or flowthrough tests of equal, fairly prolonged duration.

The toxicity of cyanide increases with reduction in dissolved oxygen below the saturation level (Doudoroff, 1976; Smith, et al. 1978) and the resistances of fishes to cyanide solutions that are rapidly lethal decreases with an increase in temperature. Long-term lethality tests, however, have demonstrated that juvenile fishes are more sensitive to cyanide with a reduction in temperature (Doudoroff, 1980; Leduc, et al. 1982; Smith, et al.

1978). No pronounced relationship has been observed between the acute toxicity of cyanide to fishes and alkalinity, hardness, or pH below about 8.3.

Genus Mean Acute Values (Table 3) were calculated as the geometric means of the available Species Mean Acute Values (Table 1). Data are available for more than one species in two genera and the Species Mean Acute Values in each are within a factor of 2. Of the 15 genera the most sensitive, Salmo, is 39 times more sensitive than the most resistant, Tanytarsus (Table 3). A freshwater Final Acute Value of 62.68 µg/L was calculated from the Genus Mean Acute Values using the calculation procedure described in the Guidelines. However, the Species Mean Acute Value for the important rainbow trout is 44.73 µg/L. Because this value is based on the results of flow-through tests in which the concentrations were measured, it replaces the calculated freshwater Final Acute Value (Table 3). At low temperatures acute effects on rainbow trout have been observed (Kovacs, 1979; Kovacs and Leduc, 1982b) at concentrations below the Final Acute Value (Table 1).

Data are available on the acute toxicity of cyanide to saltwater species in three fish genera and five invertebrate genera (Tables 1 and 3). Species Mean Acute Values for invertebrates ranged from 4.893 µg/L for larvae of the rock crab, Cancer irroratus, to over 10,000 µg/L for larvae of the common Atlantic slippershell, Crepidula fornicata. C. irroratus is six times more sensitive to cyanide than the next most sensitive species, the calanoid copepod, Acartia tonsa. Acute values for fishes only ranged from 59 µg/L to 372 µg/L. Only the genus Mysidopsis contained more than one species and the Species Mean Acute Values were within a factor of 1.1. The saltwater Final Acute Value calculated from the Genus Mean Acute Values in Table 3 is 2.030 µg/L, which is approximately one-half the Species Mean Acute Value of the most sensitive of the nine species for which acute values are available.

Chronic Toxicity to Aquatic Animals

The long-term survival and growth of various freshwater fish species were observed to be substantially reduced at free cyanide concentrations of about 20 to 50 µg/L (Tables 2 and 5). Based on reduced long-term survival in an early life-stage test with the bluegill, and reduced reproduction by the brook trout and fathead minnow in a partial life-cycle and life-cycle test, the chronic values were 13.57, 7.849, and 16.39 µg/L, respectively. Life-cycle tests (Table 2) have been conducted with two freshwater invertebrates. The chronic values were 34.06 µg/L for the isopod, Asellus communis, and 18.33 µg/L for the amphipod, Gammarus pseudolimnaeus.

Four of the freshwater acute-chronic ratios are between 7 and 11, whereas the one for the resistant isopod is 68.29 (Tables 2 and 3). It seems reasonable to use the geometric mean of the four as the freshwater Final Acute-Chronic Ratio. Division of the Final Acute Value by the Final Acute-Chronic Ratio results in a freshwater Final Chronic Value of 5.221 µg/L (Table 3).

Data are available on the chronic toxicity of cyanide to the saltwater fish, Cyprinodon variegatus, and the mysid, Mysidopsis bahia (Table 2). The early life-stage test with the sheepshead minnow, C. variegatus, showed that growth was not significantly reduced at a cyanide concentration of 462 µg/L. Survival, however, was significantly reduced at cyanide concentrations $\geq$45 µg/L but not at $\leq$29 µg/L. Thus, the chronic value for sheepshead minnow is 36.12 µg/L. A life-cycle test with the mysid, M. bahia, showed that growth and survival were not affected at cyanide concentrations $\leq$43 µg/L. Acute toxicity, however, occurred at 113 µg/L. The chronic limits for this species were defined, therefore, as 43 and 113 µg/L. The geometric mean of these limits results in a chronic value of 69.71 µg/L.

The two acute-chronic ratios available from tests with saltwater species are 8.306 and 1.621 (Table 3), but both of these species are relatively resistant to cyanide and the acute values in those ratios were obtained with juveniles of the fish and mysid. On the other hand, the acute value for the sensitive rock crab was obtained using larvae of that species. Thus, this acute value for the rock crab is probably a better indication of the chronic sensitivity of this species than would be obtained by dividing this acute value by an acute-chronic ratio. Therefore, it seems reasonable to set the saltwater Final Chronic Value equal to the Criterion Maximum Concentration of 1.015 µg/L (Table 3). Division of the geometric mean of the two saltwater acute-chronic ratios into the Species Mean Acute Values of all saltwater species except the rock crab results in values that are at least 1.6 times greater than this Final Chronic Value.

Toxicity to Aquatic Plants

Data on the toxicity of free cyanide to freshwater and saltwater plant species are presented in Table 4. Both freshwater and saltwater plants show a wide range of sensitivities to cyanide, and the saltwater red macroalga, Champia parvula, is extremely sensitive to cyanide poisoning with growth and reproductive effects occurring at 11 to 25 µg/L. Adverse effects of cyanide on plants are unlikely, however, at concentrations which do not cause chronic effects on most freshwater and saltwater animal species.

Bioaccumulation

No studies have been reported showing a biomagnification of cyanide in the food chain (Towill, et al. 1978). Pennington, et al. (1982) found no detectable levels of cyanide in four species of fish from a Mississippi lake.

Murachi, et al. (1978) and Holden and Marsden (1964) measured the concentration of cyanide in various tissues of fish exposed to very rapidly lethal cyanide levels. It is obvious from such experiments that cyanide does penetrate aquatic organisms but bioaccumulation cannot be demonstrated because it is readily metabolized.

Other Data

Embryos of the fathead minnow are possibly slightly less sensitive to cyanide than fry and juveniles, whereas embryos of yellow perch are about as sensitive as fry, but less sensitive than juveniles (Tables 1 and 5) (Broderius, et al. 1977; Smith, et al. 1978). Several authors (Broderius, 1970; Dixon and Leduc, 1981; Kovacs, 1979; Kovacs and Leduc, 1982a; Leduc, 1977, 1978; Leduc and Chan, 1975; Lesniak, 1977; McCracken and Leduc, 1980; Neil, 1957; Oseid and Smith, 1979; Ruby, et al. 1979) reported adverse effects due to cyanide concentrations as low as 10 µg/L. In another study, Kimball, et al. (1978) reported that no reproduction occurred among adult bluegills when exposed for 289 days to the lowest concentration tested (5.2 µg of HCN/L = 5.4 µg of free cyanide as CN/L). During this period, however, only a total of 13 spawnings occurred in two controls and no concentration-effect relationship was observed. Because of reservations regarding the spawning data, the chronic value for the bluegill was based on long-term fry survival. On the other hand, the most sensitive adverse effect of cyanide on both the fathead minnow and brook trout was reduced reproduction.

Unused Data

Some data on the effects of cyanide on aquatic organisms were not used because the studies were conducted with species that are not resident in

North America (Abram, 1964; Brockway, 1963; Costa, 1966; Lomte and Jadhav, 1982; Woker and Wuhrmann, 1950). Data were not used if cyanide was a component of a complex cyanide (Doudoroff, 1976) or an effluent (Lloyd and Jordan, 1964; Shelford, 1917).

Some data were not used because the results were only presented graphically (Downing, 1954; Renn, 1955; Smith and Heath, 1979). Studies conducted using inadequate dilution water (Jones, 1941) or without controls (Bridges, 1958; Costa, 1965a,b,c) were also not used. Bringmann and Kuhn (1982) cultured Daphnia magna in one water but conducted tests in another water. Data in some papers were not used because either the test conditions were not clearly stated (Burdick and Lipschuetz, 1950; Ishio, 1965; Lewis and Tarrant, 1960; Whittingham, 1952) or the test procedures were considered inadequate (Lund, 1918; Moore and Kin, 1968; Summerfelt and Lewis, 1967; Washburn, 1948). The 96-hr values reported by Buikema, et al. (1977) were subject to error because of possible reproductive interactions.

Summary

Data on the acute toxicity of free cyanide (the sum of cyanide present as HCN and CN^-, expressed as CN) are available for a wide variety of freshwater species that are involved in diverse community functions. The acute sensitivities ranged from 44.73 µg/L to 2,490 µg/L, but all of the species with acute sensitivities above 400 µg/L were invertebrates. A long-term survival, and a partial and life-cycle test with fish gave chronic values of 13.57, 7.849, and 16.39 µg/L, respectively. Chronic values for two freshwater invertebrate species were 18.33 and 34.06 µg/L. Freshwater plants were affected at cyanide concentrations ranging from 30 µg/L to 26,000 µg/L.

The acute toxicity of free cyanide to saltwater species ranged from 4.893 µg/L to >10,000 µg/L and invertebrates were both the most and least sensitive species. Long-term survival in an early life-stage test with the sheepshead minnow gave a chronic value of 36.12 µg/L. Long-term survival in a mysid life-cycle test resulted in a chronic value of 69.71 µg/L. Tests with the red macroalga, Champia parvula, showed cyanide toxicity at 11 to 25 µg/L, but other species were affected at concentrations up to 3,000 µg/L.

National Criteria

The procedures described in the "Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and Their Uses" indicate that, except possibly where a locally important species is very sensitive, freshwater aquatic organisms and their uses should not be affected unacceptably if the four-day average concentration of cyanide does not exceed 5.2 µg/L more than once every three years on the average and if the one-hour average concentration does not exceed 22 µg/L more than once every three years on the average.

The procedures described in the "Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and Their Uses" indicate that, except possibly where a locally important species is very sensitive, saltwater aquatic organisms and their uses should not be affected unacceptably if the one-hour average concentration of cyanide does not exceed 1.0 µg/L more than once every three years on the average.

EPA believes that a measurement such as free cyanide would provide a more scientifically correct basis upon which to establish criteria for cyanide. The criteria were developed on this basis. However, at this time, no EPA approved methods for such a measurement are available to implement the

criteria through the regulatory programs of the Agency and the States. The Agency is considering development and approval of methods for a measurement such as free cyanide. Until available, however, EPA recommends applying the criteria using the total cyanide method. These criteria may be overly protective when based on the total cyanide method.

The recommended exceedence frequency of three years is the Agency's best scientific judgment of the average amount of time it will take an unstressed system to recover from a pollution event in which exposure to cyanide exceeds the criterion. Stressed systems, for example, one in which several outfalls occur in a limited area, would be expected to require more time for recovery. The resilience of ecosystems and their ability to recover differ greatly, however, and site-specific criteria may be established if adequate justification is provided.

The use of criteria in designing waste treatment facilities requires the selection of an appropriate wasteload allocation model. Dynamic models are preferred for the application of these criteria. Limited data or other factors may make their use impractical, in which case one should rely on a steady-state model. The Agency recommends the interim use of 1Q5 or 1Q10 for Criterion Maximum Concentration (CMC) design flow and 7Q5 or 7Q10 for the Criterion Continuous Concentration (CCC) design flow in steady-state models for unstressed and stressed systems respectively. These matters are discussed in more detail in the Technical Support Document for Water Quality-Based Toxics Control (U.S. EPA, 1985).

Table 1. Acute Toxicity of Cyanide to Aquatic Animals

Species	Method*	LC50 or EC50 (µg/L)**	Species Mean Acute Value (µg/L)**	Reference
		FRESHWATER SPECIES		
Snail, *Physa heterostropha*	S, U	432	432	Cairns & Scheier, 1958; Patrick, et al. 1968
Cladoceran, *Daphnia magna*	S, U	<1,800	-	Anderson, 1946
Cladoceran, *Daphnia magna*	S, U	160	160	Dowden & Bennett, 1965
Cladoceran, *Daphnia pulex*	S, U	83	-	Lee, 1976
Cladoceran, *Daphnia pulex*	S, M	110	95.55	Cairns, et al. 1978
Isopod, *Asellus communis*	FT, M	2,326	2,326	Oseid & Smith, 1979
Amphipod, *Gammarus pseudolimnaeus*	FT, M	167	167	Oseid & Smith, 1979
Stonefly, *Pteronarcys dorsata*	FT, M	426	426	Call & Brooke, 1982
Midge, *Tanytarsus dissimilis*	S, M	2,490	2,490	Call, et al. 1983
Rainbow trout (fry), *Salmo gairdneri*	S, U	90	-	Bills, et al. 1977
Rainbow trout (juvenile), *Salmo gairdneri*	S, U	97	-	Skibba, 1981
Rainbow trout (juvenile), *Salmo gairdneri*	S, U	46.3	-	Marking, et al. 1984
Rainbow trout (juvenile), *Salmo gairdneri*	S, U	52.1	-	Marking, et al. 1984
Rainbow trout (juvenile), *Salmo gairdneri*	S, U	54.1	-	Marking, et al. 1984

Table 1. (Continued)

Species	Method*	LC50 or EC50 (µg/L)**	Species Mean Acute Value (µg/L)**	Reference
Rainbow trout (juvenile), Salmo gairdneri	S, U	62.1	-	Marking, et al. 1984
Rainbow trout (juvenile), Salmo gairdneri	S, U	74.8	-	Marking, et al. 1984
Rainbow trout (juvenile), Salmo gairdneri	FT, M	57	-	Smith, et al. 1978; Broderius & Smith, 1979
Rainbow trout (juvenile), Salmo gairdneri	FT, M	27	-	Kovacs, 1979; Kovacs & Leduc, 1982b
Rainbow trout (juvenile), Salmo gairdneri	FT, M	40	-	Kovacs, 1979; Kovacs & Leduc, 1982b
Rainbow trout (juvenile), Salmo gairdneri	FT, M	65	44.73	Kovacs, 1979; Kovacs & Leduc, 1982b
Atlantic salmon (juvenile), Salmo salar	R, M	90	90	Tryland and Grande, 1983
Brook trout (sac fry), Salvelinus fontinalis	FT, M	105***	-	Smith, et al. 1978
Brook trout (sac fry), Salvelinus fontinalis	FT, M	342***	-	Smith, et al. 1978
Brook trout (sac fry), Salvelinus fontinalis	FT, M	507***	-	Smith, et al. 1978
Brook trout (sac fry), Salvelinus fontinalis	FT, M	252***	-	Smith, et al. 1978
Brook trout (swim-up fry), Salvelinus fontinalis	FT, M	84	-	Smith, et al. 1978
Brook trout (swim-up fry), Salvelinus fontinalis	FT, M	54.4	-	Smith, et al. 1978
Brook trout (swim-up fry), Salvelinus fontinalis	FT, M	86.5	-	Smith, et al. 1978

Table 1. (Continued)

Species	Method*	LC50 or EC50 (µg/L)**	Species Mean Acute Value (µg/L)**	Reference
Brook trout (swim-up fry), Salvelinus fontinalis	FT, M	104	-	Smith, et al. 1978
Brook trout (swim-up fry), Salvelinus fontinalis	FT, M	90.3	-	Smith, et al. 1978
Brook trout (juvenile), Salvelinus fontinalis	FT, M	73.5	-	Smith, et al. 1978
Brook trout (juvenile), Salvelinus fontinalis	FT, M	83	-	Smith, et al. 1978
Brook trout (juvenile), Salvelinus fontinalis	FT, M	75	-	Smith, et al. 1978
Brook trout (juvenile), Salvelinus fontinalis	FT, M	86.4	-	Smith, et al. 1978
Brook trout (juvenile), Salvelinus fontinalis	FT, M	91.9	-	Smith, et al. 1978
Brook trout (juvenile), Salvelinus fontinalis	FT, M	99	-	Smith, et al. 1978
Brook trout (juvenile), Salvelinus fontinalis	FT, M	96.7	-	Smith, et al. 1978
Brook trout (juvenile), Salvelinus fontinalis	FT, M	112	-	Smith, et al. 1978
Brook trout (juvenile), Salvelinus fontinalis	FT, M	52	-	Smith, et al. 1978
Brook trout (juvenile), Salvelinus fontinalis	FT, M	60.2	-	Smith, et al. 1978
Brook trout (juvenile), Salvelinus fontinalis	FT, M	66.8	-	Smith, et al. 1978
Brook trout (juvenile), Salvelinus fontinalis	FT, M	71.4	-	Smith, et al. 1978

Table 1. (Continued)

Species	Method*	LC50 or EC50 (µg/L)**	Species Mean Acute Value (µg/L)**	Reference
Brook trout (juvenile), Salvelinus fontinalis	FT, M	97	-	Smith, et al. 1978
Brook trout (juvenile), Salvelinus fontinalis	FT, M	143	-	Smith, et al. 1978
Brook trout (adult), Salvelinus fontinalis	FT, M	156	85.80	Cardwell, et al. 1976
Goldfish (juvenile), Carassius auratus	FT, M	318	318	Cardwell, et al. 1976
Fathead minnow (juvenile), Pimephales promelas	S, U	230	-	Doudoroff, 1956
Fathead minnow, Pimephales promelas	S, M	350	-	Henderson, et al. 1961
Fathead minnow, Pimephales promelas	S, M	230	-	Henderson, et al. 1961
Fathead minnow (fry), Pimephales promelas	FT, M	120	-	Smith, et al. 1978
Fathead minnow (fry), Pimephales promelas	FT, M	98.7	-	Smith, et al. 1978
Fathead minnow (fry), Pimephales promelas	FT, M	81.8	-	Smith, et al. 1978
Fathead minnow (fry), Pimephales promelas	FT, M	110	-	Smith, et al. 1978
Fathead minnow (fry), Pimephales promelas	FT, M	116	-	Smith, et al. 1978
Fathead minnow (juvenile), Pimephales promelas	FT, M	119	-	Smith, et al. 1978
Fathead minnow (juvenile), Pimephales promelas	FT, M	126	-	Smith, et al. 1978

Table 1. (Continued)

Species	Method*	LC50 or EC50 (µg/L)**	Species Mean Acute Value (µg/L)**	Reference
Fathead minnow (juvenile), Pimephales promelas	FT, M	81.5	-	Smith, et al. 1978
Fathead minnow (juvenile), Pimephales promelas	FT, M	124	-	Smith, et al. 1978; Broderius & Smith, 1979
Fathead minnow (juvenile), Pimephales promelas	FT, M	137	-	Smith, et al. 1978
Fathead minnow (juvenile), Pimephales promelas	FT, M	131	-	Smith, et al. 1978
Fathead minnow (juvenile), Pimephales promelas	FT, M	105	-	Smith, et al. 1978
Fathead minnow (juvenile), Pimephales promelas	FT, M	119	-	Smith, et al. 1978
Fathead minnow (juvenile), Pimephales promelas	FT, M	131	-	Smith, et al. 1978
Fathead minnow (juvenile), Pimephales promelas	FT, M	122	-	Smith, et al. 1978
Fathead minnow (juvenile), Pimephales promelas	FT, M	161	-	Smith, et al. 1978
Fathead minnow (juvenile), Pimephales promelas	FT, M	188	-	Smith, et al. 1978
Fathead minnow (juvenile), Pimephales promelas	FT, M	175	-	Smith, et al. 1978
Fathead minnow (juvenile), Pimephales promelas	FT, M	163	-	Smith, et al. 1978
Fathead minnow (juvenile), Pimephales promelas	FT, M	169	-	Smith, et al. 1978
Fathead minnow (juvenile), Pimephales promelas	FT, M	120	-	Broderius, et al. 1977

Table 1. (Continued)

Species	Method*	LC50 or EC50 (µg/L)**	Species Mean Acute Value (µg/L)**	Reference
Fathead minnow (juvenile), Pimephales promelas	FT, M	113	-	Broderius, et al. 1977
Fathead minnow (juvenile), Pimephales promelas	FT, M	128	-	Broderius, et al. 1977
Fathead minnow (juvenile), Pimephales promelas	FT, M	128	125.1	Broderius, et al. 1977
Guppy (adult), Poecilia reticulata	FT, M	147	147	Anderson & Weber, 1975
Bluegill (juvenile), Lepomis macrochirus	S, U	180	-	Cairns & Scheier, 1958, 1968; Patrick, et al. 1968
Bluegill, Lepomis macrochirus	S, M	220	-	Cairns & Scheier, 1959
Bluegill, Lepomis macrochirus	S, M	180	-	Cairns & Scheier, 1959
Bluegill, Lepomis macrochirus	S, M	230	-	Cairns & Scheier, 1959
Bluegill (juvenile), Lepomis macrochirus	S, M	150	-	Henderson, et al. 1961
Bluegill (juvenile), Lepomis macrochirus	S, M	160	-	Cairns & Scheier, 1963
Bluegill (fry), Lepomis macrochirus	FT, M	364***	-	Smith, et al. 1978
Bluegill (fry), Lepomis macrochirus	FT, M	232***	-	Smith, et al. 1978
Bluegill (fry), Lepomis macrochirus	FT, M	279***	-	Smith, et al. 1978
Bluegill (fry), Lepomis macrochirus	FT, M	273***	-	Smith, et al. 1978

Table 1. (Continued)

Species	Method*	LC50 or EC50 (µg/L)**	Species Mean Acute Value (µg/L)**	Reference
Bluegill (juvenile), Lepomis macrochirus	FT, M	81	-	Smith, et al. 1978
Bluegill (juvenile), Lepomis macrochirus	FT, M	85.7	-	Smith, et al. 1978
Bluegill (juvenile), Lepomis macrochirus	FT, M	74	-	Smith, et al. 1978
Bluegill (juvenile), Lepomis macrochirus	FT, M	100	-	Smith, et al. 1978
Bluegill (juvenile), Lepomis macrochirus	FT, M	107	-	Smith, et al. 1978
Bluegill (juvenile), Lepomis macrochirus	FT, M	99	-	Smith, et al. 1978
Bluegill (juvenile), Lepomis macrochirus	FT, M	113	-	Smith, et al. 1978
Bluegill (juvenile), Lepomis macrochirus	FT, M	121	-	Smith, et al. 1978
Bluegill (juvenile), Lepomis macrochirus	FT, M	126	99.28	Smith, et al. 1978
Largemouth bass (juvenile), Micropterus salmoides	FT, M	102	102	Smith, et al. 1979
Black crappie, Pomoxis nigromaculatus	FT, M	102	102	Smith, et al. 1979
Yellow perch (embryo), Perca flavescens	FT, M	281***	-	Smith, et al. 1978
Yellow perch (fry), Perca flavescens	FT, M	288***	-	Smith, et al. 1978
Yellow perch (fry), Perca flavescens	FT, M	350***	-	Smith, et al. 1978

Table 1. (Continued)

Species	Method*	LC50 or EC50 (µg/L)**	Species Mean Acute Value (µg/L)**	Reference
Yellow perch (juvenile), Perca flavescens	FT, M	88.9	-	Smith, et al. 1978
Yellow perch (juvenile), Perca flavescens	FT, M	93	-	Smith, et al. 1978
Yellow perch (juvenile), Perca flavescens	FT, M	74.7	-	Smith, et al. 1978
Yellow perch (juvenile), Perca flavescens	FT, M	94.7	-	Smith, et al. 1978
Yellow perch (juvenile), Perca flavescens	FT, M	101	-	Smith, et al. 1978
Yellow perch (juvenile), Perca flavescens	FT, M	107	92.64	Smith, et al. 1978
		SALTWATER SPECIES		
Common Atlantic slippershell, Crepidula fornicata	S, U	>10,000	>10,000	Gardner & Nelson, 1981
Copepod, Acartia clausi	S, U	30	30	Gentile, 1980
Mysid, Mysidopsis bahia	S, U	93	-	Gentile, 1980
Mysid, Mysidopsis bahia	FT, M	113	113	Lussier, et al. Manuscript
Mysid, Mysidopsis bigelowi	S, U	124	124	Gentile, 1980
Amphipod, Ampelisca abdita	S, U	1,220	-	Scott, et al. Manuscript
Amphipod, Ampelisca abdita	S, U	1,150	-	Scott, et al. Manuscript

Table 1. (Continued)

Species	Method*	LC50 or EC50 (µg/L)**	Species Mean Acute Value (µg/L)**	Reference
Amphipod, *Ampelisca abdita*	S, U	704	995.9	Scott, et al. Manuscript
Rock crab (larva), *Cancer irroratus*	FT, M	4.2	-	Johns & Gentile, 1981
Rock crab (larva), *Cancer irroratus*	FT, M	5.7	4.893	Johns & Gentile, 1981
Sheepshead minnow, *Cyprinodon variegatus*	FT, M	300	300	Schimmel, et al. 1981
Atlantic silverside, *Menidia menidia*	FT, M	59	59	Gardner & Berry, 1981
Winter flounder, *Pseudopleuronectes americanus*	S, U	372	372	Cardin, 1980

* S = static, R = renewal, FT = flow-through, U = unmeasured, M = measured.

** Results are expressed as free cyanide as CN.

***Not used in calculations because data are available for a more sensitive life stage.

Table 2. Chronic Toxicity of Cyanide to Aquatic Animals

Species	Test*	Limits (µg/L)**	Chronic Value (µg/L)**	Reference
		FRESHWATER SPECIES		
Isopod, Asellus communis	LC	29-40	34.06	Oseid & Smith, 1979
Amphipod, Gammarus pseudolimnaeus	LC	16-21	18.33	Oseid & Smith, 1979
Brook trout, Salvelinus fontinalis	LC	5.6-11.0	7.849	Koenst, et al. 1977
Fathead minnow, Pimephales promelas	LC	13.3-20.2	16.39	Lind, et al. 1977
Bluegill, Lepomis macrochirus	ELS	9.3-19.8	13.57	Kimball, et al. 1978
		SALTWATER SPECIES		
Mysid, Mysidopsis bahia	LC	43-113	69.71	Lussier, et al. Manuscript
Sheepshead minnow, Cyprinodon variegatus	ELS	29-45	36.12	Schimmel, et al. 1981

* LC = life cycle or partial life cycle; ELS = early life stage.

**Results are expressed as free cyanide as CN.

Acute-Chronic Ratio

Species	Acute Value (µg/L)	Chronic Value (µg/L)	Ratio
Isopod, Asellus communis	2,326	34.06	68.29
Amphipod, Gammarus pseudolimnaeus	167	18.33	9.111

Table 2. (Continued)

Acute-Chronic Ratio

Species	Acute Value (µg/L)	Chronic Value (µg/L)	Ratio
Brook trout, *Salvelinus fontinalis*	83.14***	7.849	10.59
Fathead minnow, *Pimephales promelas*	125.1****	16.39	7.633
Bluegill, *Lepomis macrochirus*	99.28*****	13.57	7.316
Mysid, *Mysidopsis bahia*	113	69.71	1.621
Sheepshead minnow, *Cyprinodon variegatus*	300	36.12	8.306

*** Geometric mean of 19 values from Smith, et al. (1978) in Table 1.

**** Geometric mean of 24 values from Smith, et al. (1978) and Broderius, et al. (1977) in Table 1.

***** Geometric mean of 9 values from Smith, et al. (1978) in Table 1.

Table 3. Ranked Genus Mean Acute Values with Species Mean Acute-Chronic Ratios

Rank*	Genus Mean Acute Value (µg/L)	Species	Species Mean Acute Value (µg/L)	Species Mean Acute-Chronic Ratio
		FRESHWATER SPECIES		
15	2,490	Midge, *Tanytarsus dissimilis*	2,490	-
14	2,326	Isopod, *Asellus communis*	2,326	68.29
13	432	Snail, *Physa heterostropha*	432	-
12	426	Stonefly, *Pteronarcys dorsata*	426	-
11	318	Goldfish, *Carassius auratus*	318	-
10	167	Amphipod, *Gammarus pseudolimnaeus*	167	9.111
9	147	Guppy, *Poecilia reticulata*	147	-
8	125.1	Fathead minnow, *Pimephales promelas*	125.1	7.633
7	123.6	Cladoceran, *Daphnia magna*	160	-
		Cladoceran, *Daphnia pulex*	95.55	-
6	102	Largemouth bass, *Micropterus salmoides*	102	-
5	102	Black crappie, *Pomoxis nigromaculatus*	102	-
4	99.28	Bluegill, *Lepomis macrochirus*	99.28	7.316

Table 3. (Continued)

Rank[a]	Genus Mean Acute Value (µg/L)	Species	Species Mean Acute Value (µg/L)	Species Mean Acute-Chronic Ratio
3	92.64	Yellow perch, *Perca flavescens*	92.64	-
2	85.80	Brook trout, *Salvelinus fontinalis*	85.80	10.59
1	63.45	Rainbow trout, *Salmo gairdneri*	44.73	-
		Atlantic salmon, *Salmo salar*	90.00	-
		SALTWATER SPECIES		
8	>10,000	Common Atlantic slippershell, *Crepidula fornicata*	>10,000	-
7	995.9	Amphipod, *Ampelisca abdita*	995.9	-
6	372	Winter flounder, *Pseudopleuronectes americanus*	372	-
5	300	Sheepshead minnow, *Cyprinodon variegatus*	300	8.306
4	118.4	Mysid, *Mysidopsis bahia*	113	1.621
		Mysid, *Mysidopsis bigelowi*	124	-
3	59	Atlantic silverside, *Menidia menidia*	59	-
2	30	Copepod, *Acartia clausi*	30	-
1	4.893	Rock crab, *Cancer irroratus*	4.893	-

Table 3. (Continued)

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* Ranked from most resistant to most sensitive based on Genus Mean Acute Value.

Fresh water

Final Acute Value = 62.68 µg/L (calculated from Genus Mean Acute Values)

Final Acute Value = 44.73 µg/L (lowered to protect rainbow trout - see text)

Criterion Maximum Concentration = (44.73 µg/L) / 2 = 22.36 µg/L

Final Acute-Chronic Ratio = 8.568 (see text)

Final Chronic Value = (44.73 µg/L) / 8.568 = 5.221 µg/L

Salt water

Final Acute Value = 2.030 µg/L

Criterion Maximum Concentration = (2.030 µg/L) / 2 = 1.015 µg/L

Final Chronic Value = 1.015 µg/L (see text)

Table 4. Toxicity of Cyanide to Aquatic Plants

Species	Effect	Result (µg/L)*	Reference
	FRESHWATER SPECIES		
Blue-green alga, Microcystis aeruginosa	90% kill	8,000	Fitzgerald, et al. 1952
Blue alga, Microcystis aeruginosa	Incipient inhibition	75	Bringmann, 1975; Bringmann & Kuhn, 1976, 1978a,b
Green alga, Scenedesmus quadricauda	Incipient inhibition	30	Bringmann & Kuhn, 1977a, 1978a,b, 1979, 1980b
Diatom, Navicula seminulum	50% reduction in division	277-491	Academy of Natural Sciences, 1960
Volvocales, Chlamydomones sp.	No effect on mean or maximum growth rate	10-100	Cairns, et al. 1978
Duckweed, Lemna gibba G3	Decreased potassium uptake	26,000	Kondo & Tsudzuki, 1980
Eurasian watermilfoil, Myriophyllum spicatum	32-day EC50 (root weight)	22,400	Stanley, 1974
	SALTWATER SPECIES		
Green alga, Prototheca zopfi	Respiration inhibition	3,000	Webster & Hackett, 1965
Green alga, Chlorella sp.	Enzyme inhibition	30,000	Nelson & Tolbert, 1970
Red alga, Champia parvula	Reduced tetrasporophyte growth	16	Steele & Thursby, 1983
Red alga, Champia parvula	Reduced tetrasporangia production	25	Steele & Thursby, 1983
Red alga, Champia parvula	Reduced female growth	11	Steele & Thursby, 1983

Table 4. (Continued)

Species	Effect	Result (µg/L)*	Reference
Red alga, *Champia parvula*	Stopped sexual reproduction	11	Steele & Thursby, 1983

* Results are expressed as free cyanide as CN.

Table 5. Other Data on Effects of Cyanide on Aquatic Organisms

Species	Duration	Effect	Result (µg/L)*	Reference
		FRESHWATER SPECIES		
Green alga, Scenedesmus quadricauda	96 hr	Incipient inhibition	160**	Bringmann & Kuhn, 1959a,b
Bacteria, Escherichia coli	-	Incipient inhibition	400-800	Bringmann & Kuhn, 1959a
Bacteria, Pseudomonas putida	16 hrs	Incipient inhibition	1	Bringmann & Kuhn, 1976, 1977a, 1979, 1980b
Protozoan, Entosiphon sulcatum	72 hrs	Incipient inhibition	1,800	Bringmann, 1978; Bringmann & Kuhn, 1979, 1980b, 1981
Protozoan, Microregma heterostoma	28 hrs	Incipient inhibition	40	Bringmann & Kuhn, 1959b
Protozoan, Chilomonas paramecium	48 hrs	Incipient inhibition	1,200	Bringmann, et al. 1980, 1981
Protozoan, Uronema parduezi	20 hrs	Incipient inhibition	270	Bringmann & Kuhn, 1980a, 1981
Rotifer, Philodina acuticornis	48 hrs	LC50	20,000-145,000	Cairns, et al. 1978
Worm, Aeolosoma headleyi	48 hrs	LC50 (5 C) (10 C) (15 C) (20 C) (25 C)	10,000 9,000 120,000 160,000 160,000	Cairns, et al. 1978
Snail, Goniobasis livescens	48 hrs	LC50	760,000	Cairns, et al. 1976
Snail, Nitocris sp.	48 hrs	LC50 (5 C) (10 C) (15 C) (20 C) (25 C)	13,600 12,800 10,000 8,000 7,000	Cairns, et al. 1978

Table 5. (Continued)

Species	Duration	Effect	Result (µg/L)*	Reference
Snail, *Lymnaea emarginata*	48 hrs	LC50	3,300	Cairns, et al. 1976
Snail (embryo), *Lymnaea* sp.	96 hrs	LC50	52,000	Dowden & Bennett, 1965
Snail, *Physa heterostropha*	96 hrs	LC50 (periodic low D.O.)	190	Cairns & Scheler, 1958
Snail, *Physa integra*	48 hrs	LC50	1,350	Cairns, et al. 1976
Cladoceran, *Daphnia magna*	48 hrs	EC50	800**	Bringmann & Kuhn, 1959a,b
Cladoceran, *Daphnia magna*	24 hrs	LC50	530	Bringmann & Kuhn, 1977b
Cladoceran, *Daphnia pulex*	48 hrs	LC50 (5 C) (10 C) (15 C) (25 C)	330 330 180 1	Cairns, et al. 1978
Amphipod, *Gammarus pseudolimnaeus*	98 days	Competition with *Asellus* affects HCN toxicity	9	Oseid & Smith, 1979
Mayfly, *Stenonema rubrum*	48 hrs	LC50	500	Roback, 1965
Caddisfly (larva), *Hydropsyche* sp.	48 hrs	LC50	2,000	Roback, 1965
Midge, *Tanytarsus dissimilis*	48 hrs	EC50	<880	Call, et al. 1979
Coho salmon, *Oncorhynchus kisutch*	2 hrs	Swimming speed reduced	10	Broderius, 1970
Coho salmon (juvenile), *Oncorhynchus kisutch*	36 days	Reduction in growth	77	Leduc, 1966

Table 5. (Continued)

Species	Duration	Effect	Result (µg/L)*	Reference
Chinook salmon (juvenile), Oncorhynchus tshawytscha	64 days	27% reduction in biomass	20	Neqilski, 1973
Rainbow trout (juvenile), Salmo gairdneri	250 min	Approximate median survival time	200	Dep. Sci. Ind. Res., 1956
Rainbow trout (adult), Salmo gairdneri	2 min	Mean survival time	2,000	Herbert & Merkens, 1952
Rainbow trout (adult), Salmo gairdneri	8 min	Mean survival time	300	Herbert & Merkens, 1952
Rainbow trout (adult), Salmo gairdneri	12 min	Mean survival time	250	Herbert & Merkens, 1952
Rainbow trout (adult), Salmo gairdneri	12 min	Mean survival time	200	Herbert & Merkens, 1952
Rainbow trout (adult), Salmo gairdneri	24 min	Mean survival time	180	Herbert & Merkens, 1952
Rainbow trout (adult), Salmo gairdneri	72 min	Mean survival time	160	Herbert & Merkens, 1952
Rainbow trout (adult), Salmo gairdneri	90 min	Mean survival time	140	Herbert & Merkens, 1952
Rainbow trout (adult), Salmo gairdneri	2,525 min	Mean survival time	100	Herbert & Merkens, 1952
Rainbow trout (adult), Salmo gairdneri	1,617 min	Mean survival time	90	Herbert & Merkens, 1952
Rainbow trout (adult), Salmo gairdneri	3,600 min	Mean survival time	80	Herbert & Merkens, 1952
Rainbow trout (adult), Salmo gairdneri	4,441 min	Mean survival time	70	Herbert & Merkens, 1952
Rainbow trout, Salmo gairdneri	48 hrs	LC50	68	Brown, 1968

Table 5. (Continued)

Species	Duration	Effect	Result (µg/L)*	Reference
Rainbow trout (juvenile), Salmo gairdneri	18 days	Weight gain reduced	9.6	Dixon & Leduc, 1981
Rainbow trout (juvenile), Salmo gairdneri	4 days	Increased respiration rate	9.6	Dixon & Leduc, 1981
Rainbow trout (juvenile), Salmo gairdneri	18 days	Liver damage (necrobiosis)	9.6	Dixon & Leduc, 1981
Rainbow trout (juvenile), Salmo gairdneri	18 days	Reduction in fat content	19	Dixon & Leduc, 1981
Rainbow trout (juvenile), Salmo gairdneri	18 days	Higher relative body water content	9.6	Dixon & Leduc, 1981
Rainbow trout (yearling), Salmo gairdneri	21 days	65% reduction in weight gain	19	Speyer, 1975
Rainbow trout (yearling), Salmo gairdneri	21 days	75% reduction in swimming ability	19	Speyer, 1975
Rainbow trout (yearling), Salmo gairdneri	21 days	Higher relative body water content	19	Speyer, 1975
Rainbow trout (juvenile), Salmo gairdneri	28 days	Altered blood chloride and osmolarity	9.6	Leduc & Chan, 1975
Rainbow trout (yearling), Salmo gairdneri	20 days	Abnormal oocyte development	9.6	Lesniak, 1977; Lesniak & Ruby, 1982
Rainbow trout (juvenile), Salmo gairdneri	18 days	Production of dividing spermatogonia reduced by 13%	9.6	Ruby, et al. 1979
Rainbow trout (juvenile), Salmo gairdneri	18 days	Production of dividing spermatogonia reduced by 50%	29	Ruby, et al. 1979
Rainbow trout (yearling), Salmo gairdneri	7 days	Serum calcium reduced; hepatosomatic indices declined	9.6 19	Costa & Ruby, 1984

Table 5. (Continued)

Species	Duration	Effect	Result (µg/L)*	Reference
Rainbow trout (juvenile), Salmo gairdneri	24 hrs	LC50 (5 C) (12 C) (18 C)	90 98 92	Cairns, et al. 1978
Rainbow trout (juvenile), Salmo gairdneri	21 days	No effect on dry weight gain	33	Dixon & Sprague, 1981
Rainbow trout (juvenile), Salmo gairdneri	21 days	Kidney damage	33	Dixon & Sprague, 1981
Rainbow trout (juvenile), Salmo gairdneri	144 hrs	LC50	93	Dixon & Sprague, 1981
Rainbow trout (juvenile), Salmo gairdneri	20 days	Reduction in swimming ability (6-18 C)	4.8-43	Kovacs, 1979; Kovacs & Leduc, 1982a
Rainbow trout (juvenile), Salmo gairdneri	20 days	Threshold concentration (6-18 C) for reduction of relative: wet weight gain dry weight gain fat gain	 9.6-29 <4.8-29 <4.8-24	Kovacs, 1979; Kovacs & Leduc, 1982a
Rainbow trout (juvenile), Salmo gairdneri	20 days	Increase in relative water content (6-18 C)	4.8-43	Kovacs, 1979; Kovacs & Leduc, 1982a
Rainbow trout (juvenile), Salmo gairdneri	20 days	No effect on wet or dry weight relative growth rate or fat weight change for 8 g fish forced to swim at 12 cm/sec and 10 C	9.6	McCracken & Leduc, 1980
Rainbow trout (juvenile), Salmo gairdneri	20 days	Increased food maintenance requirements, decreased wet and dry weight relative growth rate and fat weight change for 18 g fish forced to swim at 12 cm/sec and 10 C	13	McCracken & Leduc, 1980

Table 5. (Continued)

Species	Duration	Effect	Result (µg/L)*	Reference
Rainbow trout (juvenile), *Salmo gairdneri*	20 days	Decreased wet weight gain for 27 g fish forced to swim at 12 cm/sec and 10 C	9.6	McCracken & Leduc, 1980
Atlantic salmon (larva), *Salmo salar*	58 days	Abnormal embryo and larval development	9.6	Leduc, 1978
Atlantic salmon (smolt), *Salmo salar*	24 hrs	LC50 (10 mg D.O./L) (3.5 mg D.O./L)	70 23	Alabaster, et al. 1983
Brown trout (fry), *Salmo trutta*	8.2 min	Death	8,030	Karsten, 1934
Brown trout (fry), *Salmo trutta*	8.9 min	Death	4,140	Karsten, 1934
Brown trout (fry), *Salmo trutta*	8.2 min	Death	2,070	Karsten, 1934
Brown trout (fry), *Salmo trutta*	140 min	Death	217	Karsten, 1934
Brown trout (juvenile), *Salmo trutta*	6.58 min	Geometric mean time to death	1,006	Burdick, et al. 1958
Brown trout (juvenile), *Salmo trutta*	15 min	Geometric mean time to death	510	Burdick, et al. 1958
Brown trout (juvenile), *Salmo trutta*	30.1 min	Geometric mean time to death	320	Burdick, et al. 1958
Brown trout (juvenile), *Salmo trutta*	5 hrs	Oxygen uptake inhibited	25	Carter, 1962
Brook trout (fry), *Salvelinus fontinalis*	15.2 min	Death	8,640	Karsten, 1934
Brook trout (fry), *Salvelinus fontinalis*	10.8 min	Death	4,290	Karsten, 1934
Brook trout (fry), *Salvelinus fontinalis*	11.7 min	Death	2,130	Karsten, 1934

Table 5. (Continued)

Species	Duration	Effect	Result (µg/L)[a]	Reference
Brook trout (fry), Salvelinus fontinalis	26 min	Death	853	Karsten, 1934
Brook trout (fry), Salvelinus fontinalis	58 min	Death	392	Karsten, 1934
Brook trout (fry), Salvelinus fontinalis	210 min	Death	217	Karsten, 1934
Brook trout (fry), Salvelinus fontinalis	130 hrs	Death	50	Karsten, 1934
Brook trout (fry), Salvelinus fontinalis	27 days	100% survival	20	Karsten, 1934
Brook trout (juvenile), Salvelinus fontinalis	3.6 days	Death	80	Nell, 1957
Brook trout (juvenile), Salvelinus fontinalis	40 days	No death	50	Nell, 1957
Brook trout (juvenile), Salvelinus fontinalis	25.5 min	75% reduction in swimming endurance	10	Nell, 1957
Brook trout (juvenile), Salvelinus fontinalis	90 days	Reduced growth	33	Koenst, et al. 1977
Goldfish (juvenile), Carassius auratus	336 hrs	LC50	261	Cardwell, et al. 1976
Goldfish (juvenile), Carassius auratus	24 hrs	LC50 (5 C) (15 C) (30 C)	3,250 440 280	Cairns, et al. 1978
Golden shiner (juvenile), Notemigonus crysoleucas	24 hrs	LC50 (5 C) (15 C) (30 C)	540 310 300	Cairns, et al. 1978
Fathead minnow, Pimephales promelas	48 hrs	LC50	240	Black, et al. 1957
Fathead minnow (juvenile), Pimephales promelas	5 days	LC50	120	Cardwell, et al. 1976

Table 5. (Continued)

Species	Duration	Effect	Result (µg/L)*	Reference
Fathead minnow (juvenile), Pimephales promelas	10 days	LC50	114	Cardwell, et al. 1976
Fathead minnow (juvenile), Pimephales promelas	28 days	Reduced increase in length	35	Lind, et al. 1977
Fathead minnow (juvenile), Pimephales promelas	56 days	Reduced increase in length and weight	62	Lind, et al. 1977
Fathead minnow (embryo), Pimephales promelas	96 hrs	LC50	347	Smith, et al. 1978
Fathead minnow (embryo), Pimephales promelas	96 hrs	LC50	272	Smith, et al. 1978
Fathead minnow (embryo), Pimephales promelas	96 hrs	LC50	201	Smith, et al. 1978
Fathead minnow (embryo), Pimephales promelas	96 hrs	LC50	123	Smith, et al. 1978
Fathead minnow (embryo), Pimephales promelas	96 hrs	LC50	186	Smith, et al. 1978
Fathead minnow (embryo), Pimephales promelas	96 hrs	LC50	200	Smith, et al. 1978
Fathead minnow (embryo), Pimephales promelas	96 hrs	LC50	206	Smith, et al. 1978
Blacknose dace, Rhinichthys atratulus	24 hrs	LC50	220	Lipschuetz & Cooper, 1955
Channel catfish (juvenile), Ictalurus punctatus	26 hrs	LC50	161	Cardwell, et al. 1976
Channel catfish (juvenile), Ictalurus punctatus	24 hrs	LC50 (5 C) (15 C) (30 C)	200 310 230	Cairns, et al. 1978
Flagfish, Jordanella floridae	10 days exposure	Reduced fecundity and hatching	63	Cheng & Ruby, 1981

Table 5. (Continued)

Species	Duration	Effect	Result (µg/L)*	Reference
Mosquitofish, *Gambusia affinis*	96 hrs	LC50 (high turbidity)	640	Wallen, et al. 1957
Guppy (juvenile), *Poecilia reticulata*	120 hrs	Threshold concentration	236	Chen & Selleck, 1969
Threespine stickleback, *Gasterosteus aculeatus*	90 min	Depressed respiration rate to 32% of normal	1,040	Jones, 1947
Threespine stickleback (adult), *Gasterosteus aculeatus*	824 min	Median survival time	134	Broderius, 1973
Threespine stickleback (adult), *Gasterosteus aculeatus*	642 min	Median survival time	170	Broderius, 1973
Threespine stickleback (adult), *Gasterosteus aculeatus*	412 min	Median survival time	237	Broderius, 1973
Bluegill (juvenile), *Lepomis macrochirus*	202 min	Median survival time	198	Broderius, 1973
Bluegill (juvenile), *Lepomis macrochirus*	260 min	Median survival time	194	Broderius, 1973
Bluegill (juvenile), *Lepomis macrochirus*	351 min	Median survival time	165	Broderius, 1973
Bluegill (juvenile), *Lepomis macrochirus*	258 min	Median survival time	165	Broderius, 1973
Bluegill (juvenile), *Lepomis macrochirus*	352 min	Median survival time	144	Broderius, 1973
Bluegill (juvenile), *Lepomis macrochirus*	655 min	Median survival time	127	Broderius, 1973
Bluegill (juvenile), *Lepomis macrochirus*	48 hrs	LC50	134	Cardwell, et al. 1976

Table 5. (Continued)

Species	Duration	Effect	Result (µg/L)*	Reference
Bluegill (juvenile), Lepomis macrochirus	48 hrs	LC50	280	Turnbull, et al. 1954
Bluegill (juvenile), Lepomis macrochirus	50 min	Median resistance time	960	Doudoroff, et al. 1966
Bluegill (juvenile), Lepomis macrochirus	91 min	Median resistance time	720	Doudoroff, et al. 1966
Bluegill (juvenile), Lepomis macrochirus	129 min	Median resistance time	540	Doudoroff, et al. 1966
Bluegill (juvenile), Lepomis macrochirus	700 min	Median resistance time	170	Doudoroff, et al. 1966
Bluegill (juvenile), Lepomis macrochirus	72 hrs	LC50	154	Doudoroff, et al. 1966
Bluegill (juvenile), Lepomis macrochirus	24 hrs	LC50 (5 C) (15 C) (30 C)	240 160 190	Cairns, et al. 1978
Bluegill (juvenile), Lepomis macrochirus	96 hrs	LC50 (periodic low D.O.)	48	Cairns & Scheler, 1958
Bluegill (adult), Lepomis macrochirus	48 hrs	LC50	160	Cairns, et al. 1965
Bluegill (adult), Lepomis macrochirus	289 days	Survival reduced	67.8	Kimball, et al. 1978
Bluegill (adult), Lepomis macrochirus	289 days	No reproduction	5.4	Kimball, et al. 1978
Smallmouth bass (juvenile), Micropterus dolomieui	7.8 min	Geometric mean time to death	1,900	Burdick, et al. 1958
Smallmouth bass (juvenile), Micropterus dolomieui	12.4 min	Geometric mean time to death	1,430	Burdick, et al. 1958

Table 5. (Continued)

Species	Duration	Effect	Result (µg/L)[a]	Reference
Smallmouth bass (juvenile), Micropterus dolomieui	15.4 min	Geometric mean time to death	978	Burdick, et al. 1958
Smallmouth bass (juvenile), Micropterus dolomieui	30.6 min	Geometric mean time to death	755	Burdick, et al. 1958
Smallmouth bass (juvenile), Micropterus dolomieui	42.8 min	Geometric mean time to death	478	Burdick, et al. 1958
Smallmouth bass (juvenile), Micropterus dolomieui	80.5 min	Geometric mean time to death	338	Burdick, et al. 1958
Smallmouth bass (juvenile), Micropterus dolomieui	122 min	Geometric mean time to death	243	Burdick, et al. 1958
Smallmouth bass (juvenile), Micropterus dolomieui	290 min	Geometric mean time to death	175	Burdick, et al. 1958
Largemouth bass (juvenile), Micropterus salmoides	2 days	Significant increases in opercular rate	40	Morgan & Kuhn, 1974
Largemouth bass (juvenile), Micropterus salmoides	24 hrs	Affected opercular rhythm	10	Morgan, 1979
		SALTWATER SPECIES		
Oyster, Crassostrea sp.	10 min	Suppressed ciliary activity	150	Usuki, 1956
Oyster, Crassostrea sp.	3 hrs	Inhibited ciliary activity	30,000	Usuki, 1956

Table 5. (Continued)

Species	Duration	Effect	Result (µg/L)*	Reference
Atlantic salmon, *Salmo salar*	24 hrs	LC50	20-75	Alabaster, et al. 1983
Pinfish, *Lagodon rhomboides*	24 hrs	LC50	69	Daugherty & Garrett, 1951

* Results are expressed as free cyanide as CN.

**in river water.

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Canadian Water Quality Guidelines for the Protection of Aquatic Life

SUMMARY TABLE

Summary of Canadian water quality guidelines for the protection of aquatic life.

Parameter[a]	Freshwater		Marine	
	Concentration (µg·L^{-1})	Date[b]	Concentration (µg·L^{-1})	Date[b]
Acenaphthene [See Polycyclic aromatic hydrocarbons (PAHs)]				
Acridine [See Polycyclic aromatic hydrocarbons (PAHs)]				
Aldicarb	1[c]	1993	0.15[c]	1993
Aldrin + Dieldrin[d]	~~0.004~~ [e, f]	1987		
Aluminum[d]	5–100[g]	1987		
Ammonia (total)[d]	1370–2200[h]	1987		
Aniline	2.2[i]	1993	Insufficient data	1993
Anthracene [See Polycyclic aromatic hydrocarbons (PAHs)]				
Arsenic[j]	5.0[k]	1997	12.5[c]	1997
Atrazine	1.8[i]	1989		
Benz*(a)*anthracene [See Polycyclic aromatic hydrocarbons (PAHs)]				
Benzene[j]	370[c, k]	1999	110[c]	1999
Benzo*(a)*pyrene [See Polycyclic aromatic hydrocarbons (PAHs)]				
2,2-Bis(*p*-chlorophenyl)-1,1,1-trichloroethane [See DDT (total)]				
Bromacil	5.0[c, i]	1997	Insufficient data	1997
Bromoform [See Halogenated methanes, Tribromomethane]				
Bromoxynil	5.0[i]	1993	Insufficient data	1993
Cadmium	0.017[c, l]	1996	0.12[i]	1996
Captan	1.3[c]	1991		
Carbaryl	0.20[i]	1997	0.32[c, i]	1997
Carbofuran	1.8[i]	1989		
Carbon tetrachloride [See Halogenated methanes, Tetrachloromethane]				
Chlordane[d]	~~0.006~~ [e, f]	1987		
Chlorinated benzenes				
Monochlorobenzene	1.3[c, k]	1997	25[c, k]	1997
1,2-Dichlorobenzene	0.70[c, k]	1997	42[c, k]	1997
1,3-Dichlorobenzene	150[c, k]	1997	Insufficient data[k]	1997
1,4-Dichlorobenzene	26[c, k]	1997	Insufficient data[k]	1997
1,2,3-Trichlorobenzene	8.0[c, k]	1997	Insufficient data[k]	1997

Continued.

Continued.

Parameter[a]	Freshwater		Marine	
	Concentration (µg·L^{-1})	Date[b]	Concentration (µg·L^{-1})	Date[b]
Chlorinated benzenes—Continued				
1,2,4-Trichlorobenzene	24[c, k]	1997	5.4[c, k]	1997
1,3,5-Trichlorobenzene[d]	Insufficient data[k]	1997	Insufficient data[k]	1997
1,2,3,4-Tetrachlorobenzene	1.8[c, k]	1997	Insufficient data[k]	1997
1,2,3,5-Tetrachlorobenzene[d]	Insufficient data[k]	1997	Insufficient data[k]	1997
1,2,4,5-Tetrachlorobenzene[d]	Insufficient data[k]	1997	Insufficient data	1997
Pentachlorobenzene	6.0[c, k]	1997	Insufficient data	1997
Hexachlorobenzene[d]	Insufficient data[e, f, k]	1997	Insufficient data	1997
Chlorinated ethanes				
1,2-Dichloroethane	100[c, i]	1991	Insufficient data	1991
1,1,1-Trichloroethane	Insufficient data	1991	Insufficient data	1991
1,1,2,2-Tetrachloroethane	Insufficient data	1991	Insufficient data	1991
Chlorinated ethenes				
1,1,2-Trichloroethene (Tichloroethylene; TCE)	21[c, i]	1991	Insufficient data	1991
1,1,2,2-Tetrachloroethene (Tetrachloroethylene; PCE)	111[c, i]	1993	Insufficient data	1993
Chlorinated methanes [See Halogenated methanes]				
Chlorinated phenols[d]				
Monochlorophenols	7	1987		
Dichlorophenols	0.2	1987		
Trichlorophenols	18	1987		
Tetrachlorophenols	1	1987		
Pentachlorophenol (PCP)	0.5	1987		
Chlorine, reactive [See Reactive chlorine species]				
Chloroform [See Halogenated methanes, Trichloromethane]				
4-Chloro-2-methyl phenoxy acetic acid [See MCPA]				
Chlorothalonil	0.18[c]	1994	0.36[c]	1994
Chlorpyrifos	0.0035	1997	0.002[c]	1997
Chromium				
Trivalent chromium (Cr(III))	8.9[c, k]	1997	56[c, k]	1997
Hexavalent chromium (Cr(VI))	1.0[k]	1997	1.5[k]	1997
Chrysene [See Polycyclic aromatic hydrocarbons (PAHs)]				
Colour	Narrative	1999	Narrative	1999
Copper[d]	2–4[m]	1987		
Cyanazine	2.0[c, i]	1990		
Cyanide[d]	5 (as free CN)	1987		

Continued.

Continued.

Parameter[a]	Freshwater		Marine	
	Concentration (µg·L^{-1})	Date[b]	Concentration (µg·L^{-1})	Date[b]
DDAC (Didecyl dimethyl ammonium chloride)	1.5	1999		
DDT (total)[d] (2,2-Bis(*p*-chlorophenyl)-1,1,1-trichloroethane; dichloro diphenyl trichloroethane)	~~0.001~~ [e, f]	1987		
Debris (litter/settleable matter)			Narrative[c]	1996
Deltamethrin	0.0004	1997	Insufficient data	1997
Deposited bedload sediment [See Total particulate matter]				
Dibromochloromethane [See Halogenated methanes]				
Dicamba	10[c, i]	1993		
Dichlorobenzene [See Chlorinated benzenes]				
Dichlorobromomethane [See Halogenated methanes]				
Dichloro diphenyl trichloroethane [See DDT (total)]				
Dichloroethane [See Chlorinated ethanes]				
Dichloroethylene [See Chlorinated ethanes, 1,2-Dichloroethane]				
Dichloromethane [See Halogenated methanes]				
Dichlorophenols [See Chlorinated phenols]				
1,3-Dichlorophenoxyacetic acid [see Phenoxy herticides]				
Diclofop-methyl	6.1	1993		
Didecyl dimethyl ammonium chloride [See DDAC]				
Diethylene glycol [See Glycols]				
Di(2-ethylhexyl) phthalate [See Phthalate esters]				
Dimethoate	6.2[c]	1993	Insufficient data	1993
Di-*n*-butyl phthalate [See Phthalate esters]				
Di-*n*-octyl phthalate [See Phthalate esters]				
Dinoseb	0.05	1992		
Dissolved gas supersaturation	Narrative	1999	Narrative	1999
Dissolved oxygen	5500–9500[k, n]	1999	>8000 & narrative [c, k]	1996
Endosulfan[d]	0.02	1987		
Endrin[d]	~~0.0023~~ [f, i]	1987		
Ethylbenzene[j]	90[c, k]	1996	25[c, k]	1996
Ethylene glycol [See Glycols]				
Fluoranthene [See Polycyclic aromatic hydrocarbons (PAHs)]				
Fluorene [See Polycyclic aromatic hydrocarbons (PAHs)]				

Continued.

Continued.

Parameter[a]	Freshwater		Marine	
	Concentration ($\mu g \cdot L^{-1}$)	Date[b]	Concentration ($\mu g \cdot L^{-1}$)	Date[b]
Glycols				
Ethylene glycol	192 000[k]	1997	Insufficient data	1997
Diethylene glycol	Insufficient data[k]	1997	Insufficient data	1997
Propylene glycol	500 000[k]	1997	Insufficient data	1997
Glyphosate	65[c]	1989		
Halogenated methanes				
Monochloromethane (Methyl chloride)[d]	Insufficient data	1992	Insufficient data	1992
Dichloromethane (Methylene chloride)	98.1[c, i]	1992	Insufficient data	1992
Trichloromethane (Chloroform)	1.8[c, i]	1992	Insufficient data	1992
Tetrachloromethane (Carbon tetrachloride)	13.3[c, i]	1992	Insufficient data	1992
Monobromomethane (Methyl bromide)[d]	Insufficient data	1992	Insufficient data	1992
Tribromomethane (Bromoform)[d]	Insufficient data	1992	Insufficient data	1992
Dibromochloromethane[d]	Insufficient data	1992	Insufficient data	1992
Dichlorobromomethane[d]	Insufficient data	1992	Insufficient data	1992
HCBD [See Hexachlorobutadiene (HCBD)]				
Heptachlor (Heptochlor epoxide)[d]	~~0.01~~ [e,f]	1987		
Hexachlorobenzene [See Chlorinated benzenes]				
Hexachlorobutadiene (HCBD)	1.3[c, k]	1999		
Hexachlorocyclohexane (Lindane)[d]	0.01	1987		
Hypochlorous acid [See Reactive chlorine species]				
3-Iodo-2-propynyl butyl carbamate [See IPBC]				
IPBC (3-Iodo-2-propynyl butyl carbamate)	1.9	1999		
Iron[d]	300	1987		
Lead[d]	1–7[o]	1987		
Lindane [See Hexachlorocyclohexane]				
Linuron	7.0[c]	1995	Insufficient data	1995
MCPA (4-Chloro-2-methyl phenoxy acetic acid; 2-methyl-4-chloro phenoxy acetic acid)	2.6[c]	1995	4.2[c]	1995
Mercury[d]	0.1	1987		
Methyl bromide [See Halogenated methanes, Monobromomethane]				
Methyl chloride [See Halogenated methanes, Monochloromethane]				
2-Methyl-4-chloro phenoxy acetic acid [See MCPA]				
Methylene chloride [See Halogenated methanes, Dichloromethane]				
Metolachlor	7.8[c]	1991		
Metribuzin	1.0[c]	1990		
Molybdenum[j]	73[c]	1999		
Monobromomethane [See Halogenated methanes]				

Continued.

Continued.

Parameter[a]	Freshwater		Marine	
	Concentration ($\mu g \cdot L^{-1}$)	Date[b]	Concentration ($\mu g \cdot L^{-1}$)	Date[b]
Monochloramine [See Reactive chlorine species]				
Monochlorobenzene [See Chlorinated benzenes]				
Monochloromethane [See Halogenated methanes]				
Monochlorophenols [See Chlorinated phenols]				
Naphthalene [See Polycyclic aromatic hydrocarbons (PAHs)]				
Nickel[d]	25–150[p]	1987		
Nitrate[d]	Concentrations that stimulate weed growth should be avoided.	1987		
Nitrite[d]	60	1987		
Organotins				
Tributyltin	0.008[c]	1992	0.001	1992
Tricyclohexyltin	Insufficient data	1992	Insufficient data	1992
Triphenyltin	0.022[c, i]	1992	Insufficient data	1992
Oxygen, dissolved [See Dissolved oxygen]				
PAHs [See Polycyclic aromatic hydrocarbons (PAHs)]				
PCBs [See Polychlorinated biphenyls (PCBs)(total)]				
PCE [See Chlorinated ethenes, 1,1,2,2-Tetrachloroethene]				
PCP [See Chlorinated phenols, Pentachlorophenol]				
Pentachlorobenzene [See Chlorinated benzenes]				
Pentachlorophenol [See Chlorinated phenols]				
pH	6.5–9[d]	1987	7.0–8.7 & narrative	1996
Phenanthrene [See Polycyclic aromatic hydrocarbons (PAHs)]				
Phenols (mono- & dihydric)	4.0[k]	1999		
Phenoxy herbicides[d, q]	4.0	1987		
Phthalate esters				
Di-*n*-butyl phthalate	19[c]	1993	Insufficient data	1993
Di(2-ethylhexyl) phthalate	16[c]	1993	Insufficient data	1993
Di-*n*-octyl phthalate	Insufficient data	1993	Insufficient data	1993
Picloram	29[c]	1990		
Polychlorinated biphenyls (PCBs) (total)[d]	~~0.001~~[e, f]	1987	~~0.01~~[e, f]	1991

Continued.

Continued.

Parameter[a]	Freshwater		Marine	
	Concentration (µg·L^{-1})	Date[b]	Concentration (µg·L^{-1})	Date[b]
Polycyclic aromatic hydrocarbons (PAHs)				
Acenaphthene	5.8[c]	1999	Insufficient data	1999
Acridine	4.4[c]	1999	Insufficient data	1999
Anthracene	0.012[c]	1999	Insufficient data	1999
Benz*(a)*anthracene	0.018[c]	1999	Insufficient data	1999
Benzo*(a)*pyrene	0.015[c]	1999	Insufficient data	1999
Chrysene	Insufficient data	1999	Insufficient data	1999
Fluoranthene	0.04[c]	1999	Insufficient data	1999
Fluorene	3.0[c]	1999	Insufficient data	1999
Naphthalene	1.1[c]	1999	1.4[c]	1999
Phenanthrene	0.4[c]	1999	Insufficient data	1999
Pyrene	0.025[c]	1999	Insufficient data	1999
Quinoline	3.4[c]	1999	Insufficient data	1999
Propylene glycol [See Glycols]				
Pyrene [See Polycyclic aromatic hydrocarbons (PAHs)]				
Quinoline [See Polycyclic aromatic hydrocarbons (PAHs)]				
Reactive chlorine species (hypochlorous acid and monochloramine)	0.5	1999	0.5	1999
Salinity			<10% fluctuation[c]	1996
Selenium[d]	1.0	1987		
Silver[d]	0.1	1987		
Simazine	10	1991		
Streambed substrate [See Total particulate matter]				
Styrene	72[c]	1999		
Suspended sediments [See Total particulate matter]				
TCE [See Chlorinated ethenes, 1,1,2-Trichloroethene]				
Tebuthiuron	1.6[c]	1995	Insufficient data	1995
Temperature	Narrative[d]	1987	Not to exceed ±1°C[c]	1996
Tetrachlorobenzene [See Chlorinated benzenes]				
Tetrachloroethane [See Chlorinated ethanes]				
Tetrachloroethene [See Chlorinated ethenes]				
Tetrachloroethylene [See Chlorinated ethenes, 1,1,2,2-Tetrachloroethene]				
Tetrachloromethane [See Halogenated methanes]				
Tetrachlorophenols [See Chlorinated phenols]				
Thallium[j]	0.8	1999		
Toluene	2.0[c, j, k]	1996	215[c, k]	1996

Continued.

Continued.

Parameter[a]	Freshwater Concentration (µg·L^{-1})	Freshwater Date[b]	Marine Concentration (µg·L^{-1})	Marine Date[b]
Total particulate matter[r]				
Deposited bedload sediment	Insufficient data	1999	Insufficient data	1999
Streambed substrate	Narrative	1999	Narrative	1999
Suspended sediments	Narrative	1999	Narrative	1999
Turbidity	Narrative	1999	Narrative	1999
Toxaphene[d]	~~0.008~~ [e, f]	1987		
Triallate	0.24[c]	1992		
Tribromomethane [See Halogenated methanes]				
Tributyltin [See Organotins]				
Trichlorobenzene [See Chlorinated benzenes]				
Trichloroethane [See Chlorinated ethanes]				
Trichloroethene [See Chlorinated ethenes]				
Trichloroethylene [See Chlorinated ethenes, 1,1,2-Trichloroethene]				
Trichloromethane [See Halogenated methanes]				
Trichlorophenols [See Chlorinated phenols]				
Tricyclohexyltin [See Organotins]				
Trifluralin	0.20[i]	1993		
Triphenyltin [See Organotins]				
Turbidity [See Total particulate matter]				
Zinc[d]	30	1987		

[a]Unless otherwise indicated, supporting documents are available from the Guidelines and Standards Division, Environment Canada.

[b]The guidelines dated 1987 have been carried over from *Canadian Water Quality Guidelines* (CCREM 1987) and no fact sheet was prepared. The guidelines dated 1989 to 1997 were developed and initially published in CCREM 1987 as appendixes on the date indicated. They are published as fact sheets in this document. Other guidelines dated 1997 and those dated 1999 are published for the first time in this document.

[c]Interim guideline.

[d]No fact sheet created.

[e]This guideline (originally published in *Canadian Water Quality Guidelines* [CCREM 1987 + Appendixes] in 1987 or 1991 [PCBs in marine waters]) is no longer recommended and the value is withdrawn. A water quality guideline is not recommended. Environmental exposure is predominantly via sediment, soil, and/or tissue, therefore, the reader is referred to the respective guidelines for these media.

[f]This substance meets the criteria for Track 1 substances under the national CCME Policy for the Management of Toxic Substances (PMTS) (i.e., persistent, bioaccumulative, primarily the result of human activity, and CEPA-toxic or equivalent), and should be subject to virtual elimination strategies. Guidelines can serve as action levels or interim management objectives towards virtual elimination.

[g]Aluminum guideline = 5 µg·L^{-1} at pH <6.5; [Ca^{2+}] <4 mg·L^{-1}; DOC <2 mg·L^{-1}
= 100 µg·L^{-1} at pH ≥6.5; [Ca^{2+}] ≥4 mg·L^{-1}; DOC ≥2 mg·L^{-1}

[h]Ammonia guideline = 1370 µg·L^{-1} at pH 8.0; 10ºC
= 2200 µg·L^{-1} at pH 6.5; 10ºC

[i]Guideline value slightly modified from CCREM 1987 + Appendixes due to re-evaluation of the significant figures.

[j]The technical document for the guideline is available from the Ontario Ministry of the Environment.

[k]Substance has been re-evaluated since CCREM 1987 + Appendixes. Either a new guideline has been derived or insufficient data existed to derive a new guideline.

[l]Cadmium guideline = $10^{\{0.86[\log(\text{hardness})] - 3.2\}}$.

[m] Copper guideline = 2 $\mu g \cdot L^{-1}$ at $[CaCO_3]$ = 0–120 $mg \cdot L^{-1}$
= 3 $\mu g \cdot L^{-1}$ at $[CaCO_3]$ = 120–180 $mg \cdot L^{-1}$
= 4 $\mu g \cdot L^{-1}$ at $[CaCO_3]$ >180 $mg \cdot L^{-1}$

[n] Dissolved oxygen for warm-water biota: early life stages = 6000 $\mu g \cdot L^{-1}$
other life stages = 5500 $\mu g \cdot L^{-1}$
for cold-water biota: early life stages = 9500 $\mu g \cdot L^{-1}$
other life stages = 6500 $\mu g \cdot L^{-1}$

[o] Lead guideline = 1 $\mu g \cdot L^{-1}$ at $[CaCO_3]$ = 0–60 $mg \cdot L^{-1}$
= 2 $\mu g \cdot L^{-1}$ at $[CaCO_3]$ = 60–120 $mg \cdot L^{-1}$
= 4 $\mu g \cdot L^{-1}$ at $[CaCO_3]$ = 120–180 $mg \cdot L^{-1}$
= 7 $\mu g \cdot L^{-1}$ at $[CaCO_3]$ = >180 $mg \cdot L^{-1}$

[p] Nickel guideline = 25 $\mu g \cdot L^{-1}$ at $[CaCO_3]$ = 0–60 $mg \cdot L^{-1}$
= 65 $\mu g \cdot L^{-1}$ at $[CaCO_3]$ = 60–120 $mg \cdot L^{-1}$
= 110 $\mu g \cdot L^{-1}$ at $[CaCO_3]$ = 120–180 $mg \cdot L^{-1}$
= 150 $\mu g \cdot L^{-1}$ at $[CaCO_3]$ = >180 $mg \cdot L^{-1}$

[q] The guideline of 4.0 $\mu g \cdot L^{-1}$ for phenoxy herbicides is based on data for ester formulations of 2,4-dicholorophenoxyacetic acid.

[r] The technical document for the guideline is available from British Columbia Ministry of Environment, Lands and Parks.

Reference

CCREM (Canadian Council of Resource and Environment Ministers). 1987. Canadian water quality guidelines. Prepared by the Task Force on Water Quality Guidelines.

Reference listing:

Canadian Council of Ministers of the Environment. 1999. Canadian water quality guidelines for the protection of aquatic life: Summary table. In: Canadian environmental quality guidelines, 1999, Canadian Council of Ministers of the Environment, Winnipeg.

For further scientific information, contact:

Environment Canada
Guidelines and Standards Division
351 St. Joseph Blvd.
Hull, QC K1A 0H3
Phone: (819) 953-1550
Facsimile: (819) 953-0461
E-mail: ceqg-rcqe@ec.gc.ca
Internet: http://www.ec.gc.ca

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Phone: (204) 945-4664
Facsimile: (204) 945-7172
E-mail: spccme@chc.gov.mb.ca

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Cyanide Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review

by
Ronald Eisler

U.S. Fish and Wildlife Service
Patuxent Wildlife Research Center
Laurel, Maryland 20708

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Tables

Cyanide Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review

by
Ronald Eisler

U.S. Fish and Wildlife Service
Patuxent Wildlife Research Center
Laurel, Maryland 20708

Abstract. Cyanides are used widely and extensively in the manufacture of synthetic fabrics and plastics, in electroplating baths and metal mining operations, as pesticidal agents and intermediates in agricultural chemical production, and in predator control devices. Elevated cyanide levels are normally encountered in more than 1,000 species of food plants and forage crops, and this probably represents the greatest source of cyanide exposure and toxicosis to man and to range animals. Anthropogenic sources of cyanide in the environment include certain industrial processes, laboratories, fumigation operations, cyanogenic drugs, fires, cigarette smoking, and chemical warfare. Although cyanide is ubiquitous in the environment, levels tend to be elevated in the vicinity of metal processing operations, electroplaters, gold-mining facilities, oil refineries, power plants, and solid waste combustion.

Many chemical forms of cyanide are present in the environment, including free cyanide, metallocyanide complexes, and synthetic organocyanides, also known as nitriles. 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.

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. Diagnosis of acute lethal cyanide poisoning is difficult because signs and symptoms are nonspecific, and numerous factors modify its biocidal properties, such as dietary deficiencies in vitamin B_{12}, iodine, and sulfur amino acids. Among the more consistent changes measured in acute cyanide poisoning are inhibition of brain cytochrome oxidase activity, and changes in electrical activity in heart and brain. 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. Antidotes in current use to counteract cyanide poisoning include a combination of sodium nitrite and sodium thiosulphate (United States), cobalt edetate (United Kingdom, Scandinavia, France), or a mixture of 4-dimethylaminophenol and sodium thiosulphate (Germany).

All available evidence suggests that cyanides are neither mutagenic, teratogenic, nor carcinogenic. Moreover, there are no reports of cyanide biomagnification or cycling in living organisms, probably owing to its rapid detoxification. Cyanide seldom persists in surface waters and soils owing to complexation or sedimentation, microbial metabolism, and loss from volatilization. More data are needed on cyanide distribution and transformation in the atmosphere.

Analytical methods for the determination of free and bound cyanides and cyanogenic compounds in biological materials are under constant revision. Further, unless tissue samples are obtained promptly after cyanide exposure and analyzed immediately, erroneous analytical values will result.

Higher plants are adversely affected by cyanide through cytochrome oxidase inhibition; the rate of production and release of cyanide by plants to the environment through death and decomposition is unknown. Nonacclimatized soil bacteria are adversely affected at 0.3 mg HCN/kg; acclimatized populations, however, can degrade wastes containing up to 60 mg total cyanide per kilogram. In some cases, soil bacteria and fungi produce cyanides as secondary metabolites, with adverse effects on certain plants. Several species of arthropods normally contain elevated whole-body cyanide concentrations, and these confer protection against predators and allow consumption of cyanogenic plants.

Fish were the most sensitive aquatic organisms tested. Adverse effects on swimming and reproduction were observed between 5 and 7.2 µg free cyanide per liter; lethal effects usually occurred between 20 and 76 µg/L. Biocidal properties of cyanide in aquatic environments were 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.

Birds that feed predominantly on flesh were more sensitive to cyanide than were herbivores. Free cyanide levels associated with high avian death rates include 0.12 mg/L in air, 2.1-4.6 mg/kg body weight (BW) via acute oral exposure, and 1.3 mg/kg BW administered intravenously. Dietary levels of 135 mg total cyanide per kilogram ration resulted in growth reduction of chicks, but 103 mg total cyanide per kilogram ration had no measurable effect on domestic chickens.

Cyanogenic plants represent a problem for various range animals and wildlife, primarily among species that eat rapidly. Intakes of 4 mg HCN/kg BW are lethal to these species if it is consumed quickly. Cassava (*Manihot esculenta*) is a cyanogenic plant that accounts for up to 70% of human caloric intake in some areas, and this is associated with serious, long-term toxic effects including ataxia, optic nerve lesions, altered thyroid function, demyelination, and increases in tissue thiocyanate levels. Acute oral LD50 values for representative species of mammals ranged between 2 and 3.6 mg HCN/kg BW. Despite the high lethality of large single exposures, repeated sublethal doses--especially in diets--can be tolerated by many species for extended periods, perhaps indefinitely. Mammalian deaths were also recorded at air concentrations of 140 mg HCN/m^3 (exposure for 60 min) and 4,400 mg HCN/m^3 (exposure for 1 min), and at dermal applications between 2.3 mg HCN/kg BW for abraded skin and 100 mg HCN/kg BW for intact skin. Adverse nonlethal effects were noted at drinking water concentrations >150 mg HCN/L and at dietary concentrations >720 mg HCN/kg ration.

Free cyanide criteria currently proposed for natural resource protection include <3 µg/L medium for aquatic life, and <100 mg/kg diet for birds and livestock. For human health protection, free cyanide values are <10 µg/L drinking water, <50 mg/kg diet, and <5 mg/m^3 air.

Key words: Cyanide, toxic effects, wildlife, cyanogenic plants, aquatic organisms, criteria.

The origin of terrestrial life probably depended on the presence and reactivity of hydrogen cyanide and its derivatives; paradoxically, hydrogen cyanide is toxic to the majority of living matter (Marrs and Ballantyne 1987). Cyanide is a general respiratory poison--although uptake can also occur through ingestion or dermal absorption--producing reactions within seconds, and death within minutes (Towill et al. 1978; Environmental Protection Agency [EPA] 1980). The toxic mechanism of cyanide primarily involves the inhibition of cytochrome oxidase, the terminal oxidative enzyme of the mitochondrial electron transport chain, producing blockage of aerobic ATP synthesis (Egekeze and Oehme 1979; Younes and Strubelt 1988). Because of their highly effective lethal potency, cyanides were used for genocidal programs in Germany in World War II, in mass suicides by members of the People's Temple religious sect in Guyana, and in the substitution of medication in Tylenol capsules in drugstores in various cities in the United States. In fact, cyanides are responsible for more human deaths than any other chemicals known, owing to their deliberate use in suicide, murder, chemical warfare, genocide, and judicial execution (Way 1981, 1984; Ballantyne and Marrs 1987a; Gee 1987; Marrs and Ballantyne 1987; Yamamoto 1989). High sublethal doses of cyanide are rapidly detoxified, and accidental acute cyanide poisonings in humans are uncommon (Towill et al. 1978).

Cyanide compounds are useful to society in terms of their key role in synthetic and industrial processes, for certain fumigation and agricultural uses, and for some therapeutic applications (Ballantyne and Marrs 1987a). Cyanides are present in effluents from iron and steel processing plants, petroleum refineries, and metal-plating plants, and constitute a hazard to aquatic ecosystems in certain waste-receiving waters (Smith et al. 1979), and to livestock (EPA 1980;Towill et al. 1978). Cyanide serves no useful purpose in the human body, yet it is present in our food, air, and water (Becker 1985).

Natural sources of cyanide include various species of bacteria, algae, fungi, and higher plants that form and excrete cyanide (Way 1984). The most widely distributed major food crop with a high content of cyanogenic glycosides is cassava (*Manihot esculenta*), also known as manioc. Cassava is a staple food in human diets in over 80 countries, and it is sometimes added to animal feeds as a substitute for more expensive cereal grains (Gomez et al. 1988). In humans, chronic cyanide intoxication caused by consumption of cassava is the main etiological factor in the debilitating tropical ataxic neuropathy (Egekeze and Oehme 1980). Other plants having comparatively elevated cyanide content include fruit pits, sweet potatoes (*Ipomoea batatas*), corn (*Zea mays*), bamboo shoots (*Bambusa* spp.), linseed, (*Linum* sp.), lima beans (*Phaseolus lunatus*), and millet (*Panicum miliaceum*; Way 1984). In higher plants that contain cyanogenic glycosides, at least 20 of these compounds have been identified (EPA 1980). Amygdalin--one of the more intensively studied cyanogenic glycosides--is found in seeds of the cherry (*Prunus* spp.), plum (*Prunus* spp.), peach (*Prunus persica*), apricot (*Prunus armenaica*), apple (*Malus domestica*), pear (*Pyrus communis*), and many parts of the cherry laurel (*Prunus laurocerasus;* EPA 1980). Apricot seeds and peach kernels are food delicacies in Turkey, and have caused at least nine poisonings (two fatal) in children from that country (Gee 1987). Acute cyanide poisoning has occurred in the United States from the ingestion of almond-flavored milkshakes prepared from apricot kernels (Way 1984). Amygdalin is also the chief ingredient in laetrile, a medication prescribed by some physicians to control tumors. Both laetrile and amygdalin-containing fruit pits have been implicated as the causes of acute cyanide poisoning in humans (EPA 1980). Another naturally occurring group of organic cyanides (nitriles) is the highly toxic pseudocyanogenic glycosides, especially cycasin, and these have been implicated in a variety of tropical diseases of the nervous system, and partial or total blindness (EPA 1980). Other nitriles found in plants include the lathyrogenic compounds, glucosinolates, and the cyanopyridine alkaloids (EPA 1980).

That certain plants, such as bitter almonds *(Prunus dulcis),* cherry laurel leaves, and cassava, are poisonous if consumed in sufficient quantities has been known for at least 2,000 years. But it was not until the 1700's that cyanide was recognized as the basis for their lethal toxicity. The first account of an experimental administration of extract of bitter almonds and other poisons to dogs (*Canis familiaris*) dates from 1679, as reviewed by Sykes (1981) and Ballantyne (1987a). In 1731, two fatal cases of human poisoning in Ireland were caused by drinking cherry laurel water, in this instance used as a flavoring agent in cooking and to dilute brandy. In that same year it was shown that cherry laurel water administered to dogs by various routes proved rapidly fatal. By 1781, it was well established that mammals, birds, reptiles, amphibians, fish, and insects could all be killed with small doses of laurel water, and that death was more rapid than that produced by other poisons tested. It was also at this time that cyanide was first implicated as a homicidal agent in England. In 1782, hydrocyanic acid was isolated from Prussian blue (a dye) by the Swedish chemist Scheele. In 1786, Scheele accidentally broke a vial of the material and died from vapor poisoning. In 1787, it was determined that hydrocyanic acid contained hydrogen, carbon, and nitrogen, but did not contain oxygen, formerly believed to be an essential component of all acids. Between 1802 and 1815, hydrocyanic acid was found to be lethal in small quantities to birds and dogs, and to act rapidly when given orally, intravenously, or applied to the eye surface. By 1803, it was known that cyanide occurred naturally and could be extracted from apricots or almonds. In 1815, hydrocyanic acid was prepared in a semipure form. Between 1817 and 1948, cyanide, in appropriate doses, was used therapeutically in England for the treatment of pulmonary diseases and tuberculosis, and as a sedative. By 1830, cyanogenic glycosides containing HCN were isolated from cassava; today, more than 800 species of cyanogenic plants have been identified. In 1876, it was first demonstrated that cyanide inhibited tissue oxidation. In 1894, cobalt compounds were suggested as antidotes due to their marked cyanide-binding capacity. Studies on cyanide detoxification conducted between 1877 and 1894 showed that thiosulphate administration caused the formation of thiocyanate--a relatively harmless metabolite. By the late 1800's, cyanide was regarded as a common plant metabolite rather than as an unusual poison. In 1929, it was conclusively demonstrated that cyanide combines with the trivalent iron atom in cytochrome oxidase, a respiratory enzyme that links the tricarboxylic acid cycle and formation of metabolic water. Many reviews have been published on cyanide in the environment; particularly useful are those by Doudoroff (1976), Towill et al. (1978), Smith et al. (1979), Egekeze and Oehme (1980), EPA (1980, 1989), Vennesland et al. (1981a), Leduc et al. (1982), Leduc (1984), Way (1984), Ballantyne and Marrs (1987a), and Evered and Harnett (1988).

Cyanide hazards to fish, wildlife, and livestock are well documented. Massive kills of freshwater fish by accidental discharges of cyanide wastes are fairly common (Holden and Marsden 1964; Leduc 1978; Towill et al. 1978; EPA 1980). In one case, cyanide-containing mine effluents from a Canadian tailings pond released into a nearby creek killed more than 20,000 steelhead (Oncor*hynchus mykis;* Leduc et al. 1982). Many species

of birds were found dead near burrows of the blacktailed prairie dog (*Cynomys ludovicianus*) after the burrows had been treated with calcium cyanide to control prairie dog populations; dead birds included the burrowing owl (*Athene cunicularia*), the bald eagle (*Haliaeetus leucocephalus*), and the golden eagle (*Aquila chrysaetos;* Wiemeyer et al. 1986). An endangered California condor (*Gymnogyps californianus*) found dead in Kern County, California, in November 1983 had particles of a yellow fluorescent tracer in its mouth; these particles were similar to those mixed with sodium cyanide in M-44 spring-loaded ejector mechanism devices used in a U.S. Fish and Wildlife Service Animal Damage Control Program in that vicinity, suggesting that cyanide was a possible cause of death (Krynitsky et al. 1986). M-44 devices are known to have caused the death of magpies (*Pica* sp.), ravens and crows *(Corvus* spp.), wild turkeys *(Meleagris gallopavo),* and various unidentified species of hawks and vultures (Wiemeyer et al. 1986). Between 1980 and 1989, 519 mammals--mostly rodents (35%) and bats (34%)--were found dead at cyanide-extraction, gold-mine leach ponds in California, Nevada, and Arizona; the list included coyote *(Canis latrans),* foxes, skunks, badger *(Taxidea taxus),* weasels, rabbits, deer, and beavers (Clark and Hothem 1991). Also found dead at these same leach ponds were 38 reptiles, 55 amphibians, and 6,997 birds (Clark and Hothem 1991), including many species of waterfowl and songbirds (Allen 1990). The influence of cyanide-extraction gold-mining operations on wildlife is currently under investigation by scientists at the Patuxent Wildlife Research Center.

The major threat of cyanide poisoning to livestock and terrestrial mammalian wildlife is through ingestion of plants containing high levels of cyanogenic glycosides (Towill et al. 1978; Marrs and Ballantyne 1987). Plants implicated in cyanide poisoning of animals include the sorghums (Johnson grass, *Sorghum halepense;* Sudan grass, *Sorghum sudanense*), arrowgrass (*Triglochin* spp.), elderberry (*Sambucus* spp.), wild cherry (*Prunus* spp.), and the pits of several common fruits, such as apple, peach, and apricot; these plants and fruit pits have the potential of releasing cyanide upon ingestion (Egekeze and Oehme 1980). Domestic goats (*Capra* spp.) died of cyanide poisoning after eating leaves and fruit of the crab apple *(Malus sylvestris);* the crab apple contains cyanogenic glycosides in its leaves and fruit (Shaw 1986). Cyanide poisoning of cattle (*Bos* spp.) by forage sorghums and various hybrid cultivars has been reported in India (Bapat and Abhyankar 1984) and elsewhere (Cade and Rubira 1982; Biehl 1984). Cattle appear to be more vulnerable to cyanide poisoning than are sheep (*Ovis aries*), horses (*Equus caballus*), and pigs (*Sus* spp.; Cade and Rubira 1982). *Equine sorghum cystitis ataxia* is a condition observed in horses grazing on Sorghum or hybrid Sudan grass pastures; it is characterized by urinary incontinence, posterior incoordination, and degenerative central nervous system lesions (Egekeze and Oehme 1980). Grazing cyanogenic plants can induce sulfur deficiency in sheep, presumably because sulfur detoxifies the released cyanide (Towill et al. 1978). The increasing use of cassava and other cyanogenic plants in animal feeding portends a greater exposure to dietary cyanides (Davis 1981).

This report briefly reviews the technical literature on ecological and toxicological aspects of cyanide, with emphasis on fishery and wildlife resources, and provides recommendations for the protection of sensitive species of concern to the U.S. Fish and Wildlife Service. This account is part of a continuing series of synoptic reviews prepared in response to informational requests from Service environmental specialists.

Chemical Properties

The chemical speciation of cyanides varies according to their source. Specific terms used to describe cyanide include free cyanide, cyanide ion, simple cyanides, complex cyanides, nitriles, cyanogens, and total cyanide. The most common forms of cyanide in the environment are free cyanide, metallocyanide complexes, and synthetic nitriles. A brief description of each cyanide species follows (Smith et al. 1978, 1979; Towill et al. 1978; Egekeze and Oehme 1980; EPA 1980, 1989; Davis 1981; Leduc 1981, 1984; Leduc et al. 1982; Simovic and Snodgrass 1985; Ballantyne 1987a; Homan 1987; Marrs and Ballantyne 1987).

Free cyanide is the primary toxic agent in the aquatic environment. Free cyanide refers to the sum of molecular HCN and the cyanide anion (CN^-), regardless of origin. In aqueous solution with pH 9.2 and lower, the majority of the free cyanide is in the form of molecular HCN. The chemical names for HCN include hydrogen cyanide, hydrocyanic acid, cyanohydric acid, and prussic acid. Hydrogen cyanide (Table 1) is a colorless, flammable liquid or gas that boils at 25.7° C and freezes at -13.2° C. The gas rarely occurs in nature, is lighter than air, and diffuses rapidly; it is usually prepared commercially from ammonia and methane at elevated temperatures with a platinum catalyst. It is miscible with water and alcohol, but is only slightly soluble in ether. In water, HCN is a weak acid with the ratio of HCN to CN^- about 100 at pH 7.2, 10 at pH 8.2, and 1 at

pH 9.2. HCN can dissociate into H and CN^-. Cyanide ion, or free cyanide ion, refers to the anion CN^- derived from hydrocyanic acid in solution, in equilibrium with simple or complexed cyanide molecules. Cyanide ions resemble halide ions in several ways and are sometimes referred to as "pseudohaline" ions. For example, silver cyanide is almost insoluble in water, as are silver halides. Cyanide ions also form stable complexes with many metals.

Simple cyanides typically refer to alkali water-soluble salts, such as NaCN, KCN, $Ca(CN)_2$, and $Hg(CN)_2$, but also include several cyanide salts of alkali, alkaline earth, or heavy metals, that is, $Zn(CN)_2$, $Cd(CN)_2$, $Ni(CN)_2$, and AgCN, of varying degrees of solubility. In water, NaCN and KCN will completely dissociate to give free cyanide. All simple cyanides ionize in water to release cyanide ion which, depending on pH, will form hydrocyanic acid. For sodium cyanide, the reaction proceeds as follows:

(1) $NaCN \quad Na^+ + CN^-$

(2) $CN^- + HOH \quad HCN + OH^-$

Increased pH will maintain a larger fraction of the cyanide as CN^-, and acidification will cause the reverse. At pH 7, about 99% of the free cyanide is in the form of HCN, whereas at pH 9.3 HCN composes 50%. Since HCN is extremely water soluble and is also one of the most toxic cyanide species, it is noteworthy that the toxicity of simple cyanides will not be affected measurably below pH 8.3. Acidification of dilute (milligrams per liter) cyanide solutions will not initiate any greater release of HCN, but acidification of concentrated (grams per liter) solutions promotes HCN formation and release.

Table 1. Some properties of potassium cyanide, hydrogen cyanide, and sodium cyanide (from EPA 1989).

Property	Potassium cyanide	Hydrogen cyanide	Sodium cyanide
CAS number	151-50-8	74-90-8	143-33-9
Chemical formula	KCN	HCN	NaCN
Molecular weight	65.12	27.03	49.01
Physical state	Solid	Gas or liquid	Solid
Boiling point (º C)	--	25.7	1,496
Melting point (º C)	634.5	–13.21	563.7
Specific gravity	1.5	0.7 (liquid)	1.6
Solubility in water (g/L)	716 at 20º C	Miscible	480 at 10º C

Complex cyanides are compounds in which the cyanide anion is incorporated into a complex or complexes; these compounds are different in chemical and toxicologic properties from simple cyanides. In solution, the stability of the cyanide complex varies with the type of cation and the complex that it forms. Some of these are dissociable in weak acids to give free cyanide and a cation, while other complexes require much stronger acidic conditions for dissociation. The least-stable complex metallocyanides include $Zn(CN)_4^{2-}$, $Cd(CN)_3^-$, and $Cd(CN)_4^{2-}$; moderately stable complexes include $Cu(CN)_2^-$, $Cu(CN)_3^{2-}$, $Ni(CN)_4^{2-}$, and $Ag(CN)^{2-}$; and the most stable complexes include $Fe(CN)_6^{4-}$and $Co(CN)_6^{4-}$**.** The toxicity of complex cyanides is usually related to their ability to release cyanide ions in solution, which then enter into an equilibrium with HCN; relatively small fluctuations in pH significantly affect their biocidal properties.

Cyanogen [$(CN)_2$] is the simplest compound containing the cyanide group. Cyanogen is an extremely toxic, flammable gas that reacts slowly with water to form HCN, cyanic acid, and other compounds; it is rapidly

degraded in the environment. Cyanogen and its halide derivations are comparable in toxicity to hydrogen cyanide.

Nitriles are defined as organic compounds (RCN) containing the cyanide group. Cyanide bound to carbon as nitriles (other than as cyanogenic glycosides) are comparatively innocuous in the environment, and are low in chemical reactivity and are biodegradable. For simple mononitriles there is a clear progression, with more cyanide being released as chain length increases. A similar pattern exists in dinitriles, but corresponding compounds require a longer carbon chain than mononitriles before free cyanide is produced. Based on studies with chicken liver homogenates (Davis 1981), mononitriles were more toxic than dinitriles, and within each group the order of toxicity was $CH_3 > C_2H_5 > C_3H_7 > C_4H_9 > C_5H_{11} > C_7H_{15}$. Cyanohydrins [$R_2C(OH)CN$] and cyanogenic glycosides [$R_1R_2C(OR_3)CN$] are special classes of nitriles, in that under appropriate conditions they will decompose to HCN and cyanide ions. Cyanogens (not to be confused with cyanogen), such as acrylonitrile, propionitrile, and succinonitrile, are nitrile-containing materials of varying complexity and lability, and can liberate free and toxicologically available amounts of cyanide. But the nonnitrile portion of the cyanogen molecule may exert an independent or interactive toxicity, causing a complex response.

Cyanates contain the OCN group. Inorganic cyanates that are formed industrially by the oxidation of cyanide salts hydrolyze in water to form ammonia and bicarbonate ion. Alkyl cyanates are insoluble in water and form cyanurates. Alkyl isocyanates contain the OCN radical, are formed from cyanates, and, like cyanates, are readily hydrolyzed. Thiocyanates (SCN group) are formed from cyanides and sulfur-containing materials and are relatively stable.

Total cyanides refers to all cyanide-containing compounds, including simple and complex cyanides, cyanoglycosides, and free cyanide. Total cyanides is a chemical measurement of free cyanide present in solution or released by acidification or digestion. Only free cyanide is considered to be a biologically meaningful expression of cyanide toxicity. Under most circumstances, the concentration of total cyanide will exceed that of HCN. In some waters, however, the total cyanide concentration may consist almost entirely of free cyanide, or it may contain cyanides that readily photodecompose or dissociate to yield HCN. The relation between total cyanide and free cyanide in natural waters varies with receiving-water conditions, type of cyanide compounds present, degree of exposure to daylight, and presence of other chemical compounds.

Hydrogen cyanide has frequently been associated with the odor of bitter almonds (Ballantyne 1983; Gee 1987). The threshold odor for olfactory detection of atmospheric HCN is 1 mg/L, but the odor may not be detected for various reasons, including the presence of other odors and the fact that only 20% to 40% of those tested could detect a cyanide odor.

Analytical methods for determining free and bound cyanide and cyanogenic compounds in biological materials are under revision. Current methods include chromatography; enzymic postcolumn cleavage; electrochemical detection; and ultraviolet, infrared, proton, and carbon-13 nuclear magnetic resonance spectroscopies (Brimer 1988). Proposed newer analytical methodologies include chemiluminescence (Wu et al. 1989); deproteinization techniques (Krynitsky et al. 1986); thin film dissociation coupled with preferential ultraviolet irradiation (Kelada 1989); differential pulse polarography (Westley 1988); and modified spectrophotometric (Blago 1989; Ohno 1989), colorometric (Lundquist and Sorbo 1989), and ion chromatographic determinations (Nonomura and Hobo 1989). Analysis of cyanide and cytochrome oxidase is usually conducted with samples of whole blood, serum, plasma, brain, or ventricular myocardium tissues. Samples should be obtained as soon as possible after cyanide exposure and analyzed immediately, otherwise erroneous analytical values will result (Towill et al. 1978; Ballantyne 1983). Brain and liver are recommended for cyanide analysis if removed and analyzed within a week (Ballantyne et al. 1974). Cyanide measurements are further confounded by the presence of various antidotal agents (Ballantyne 1983); by various tissue preservatives, such as formaldoxime (Knocke 1981) and sodium fluoride (Curry et al. 1967); and by the spontaneous postmortem production of cyanide in various tissues (e.g., sterile blood, brain, liver, kidney, uterus, intestines) over time in cases of noncyanide death (Curry et al. 1967; Ballantyne et al. 1974).

Mode of Action

Cyanide is a potent and rapid-acting asphyxiant. At lethal doses, inhalation or ingestion of cyanide produces reactions within seconds and death within minutes. Cyanide's toxic effect is due to its affinity for the ferric heme form of cytochrome a_3, also known as cytochrome c oxidase, the terminal oxidase of the mitochondrial respiratory chain (Towill et al. 1978; Egekeze and Oehme 1980; Solomonson 1981; Way 1981, 1984; Leduc et al. 1982; Biehl 1984; Ballantyne 1987a; Marrs and Ballantyne 1987; Yamamoto 1989). Inhibition of the enzyme cytochrome c oxidase is thought to involve a two-step reaction--initial penetration of cyanide into a protein crevice followed by binding to heme iron. Formation of a stable cytochrome c oxidase-CN complex in the mitochondria produces a blockage of electron transfer from cytochrome oxidase to molecular oxygen and cessation of cellular respiration, causing cytotoxic hypoxia in the presence of normal hemoglobin oxygenation. Tissue anoxia induced by the activation of cytochrome oxidase causes a shift from aerobic to anaerobic metabolism, resulting in the depletion of energy-rich compounds such as glycogen, phosphocreatine, and adenosine triphosphate, and the accumulation of lactate with decreased blood pH. The combination of cytotoxic hypoxia with lactate acidosis depresses the central nervous system--the most sensitive site of anoxia--resulting in respiratory arrest and death. If the absorption rate is significantly greater than the detoxification rate, there will be a rapid accumulation of free cyanide in tissues and body fluids, resulting in the prompt onset of signs of acute cyanide poisoning. Acute cyanide poisoning is frequently encountered as a relatively massive overdose, where the amount of cyanide greatly exceeds the minimal concentration necessary to inhibit cytochrome c oxidase. In such cases, many enzymes and biological systems are disrupted, including various metalloenzymes, nitrate reductase, nitrite reductase, myoglobin, various peroxidases, catalase, and ribulose diphosphate carboxylase, resulting in severe signs of toxicity and rapid death.

The great majority of the absorbed cyanide reacts with thiosulfate in the presence of enzymes to produce thiocyanate, which is excreted in the urine over a period of several days. Owing to this rapid detoxification, animals can ingest high sublethal doses of cyanide over extended periods without harm (Towill et al. 1978; Egekeze and Oehme 1980; EPA 1980; Davis 1981; Solomonson 1981; Leduc 1984; Ballantyne 1987a; Oh et al. 1987; Marrs and Ballantyne 1987; Westley 1988; Mengel et al. 1989). Authorities are also in general agreement on several points: thiosulfate is usually low in the body, and higher levels can significantly protect against cyanide toxicity; species vary considerably in both the extent to which thiocyanate is formed and the rate at which it is eliminated from the body; thiocyanate metabolites resulting from the transulfuration process are about 120 times less toxic than the parent cyanide compound; thiocyanate may accumulate in tissues and has been associated with developmental abnormalities and other adverse effects; the two enzyme systems responsible for the transulfuration process are thiosulfate-cyanide sulfurtransferase--also known as rhodanese--and beta-mercaptopyruvate cyanide sulfurtransferase. Rhodanese is widely distributed in the body, but activity levels in mammals are highest in the mitochondrial fraction of liver. Rhodanese activity levels in catalyzing the transformation of thiosulfate to thiocyanate are limited by the availability of sulfur.

Minor detoxification pathways for cyanide include exhalation in breath as HCN and as $C0_2$ from oxidative metabolism of formic acid; conjugation with cystine to form 2-iminothiazolidene- 4-carboxylic acid or 2-aminothiazoline-4-carboxylic acid; combining with hydroxocobalamin (B_{12}) to form cyanocobalamin, which is excreted in urine and bile; and binding by methemoglobin in the blood (Towill et al. 1978; EPA 1980; Ballantyne 1987a; Marrs and Ballantyne 1987).

Absorption of hydrogen cyanide liquid or gas readily occurs through inhalation, ingestion, or skin contact (Towill et al. 1978; Egekeze and Oehme 1980; EPA 1980; Homan 1987). Inhalation and skin absorption are the primary hazardous routes in cyanide toxicity in occupational exposure. Skin absorption is most rapid when the skin is cut, abraded, or moist. Inhalation of cyanide salts is also potentially hazardous because the cyanide dissolves on contact with moist mucous membranes. Regardless of route of exposure, cyanide is readily absorbed into the bloodstream and distributed throughout the body. Cyanide concentrates in erythrocytes through binding to methemoglobin (Towill et al. 1978; EPA 1980), and free cyanide concentrations in plasma are now considered one of the better indicators of cytotoxicity (Ballantyne 1987a). Because of the affinity of cyanide for the mammalian erythrocyte, the spleen may contain elevated cyanide concentrations when compared to blood; accordingly, spleen should always be taken for analysis in cases of suspected cyanide poisoning (Ballantyne 1975). Cyanide also accumulates in various body cells through binding to metalloproteins or enzymes such as catalase and cytochrome c oxidase (EPA 1980). The brain is probably the major target organ

of cytotoxic hypoxia, and brain cytochrome oxidase may be the most active site of lethal cyanide action, as judged by distribution of cyanide, thiosulfate, and rhodanese (Solomonson 1981; Ballantyne 1987a). Significant positive correlations exist between cyanide concentrations in plasma, cerebrospinal fluid, and brain (Ballantyne 1987a); these correlations need further exploration.

Hydrogen cyanide formation may contribute to the toxicity of snake venom, owing to the high levels of L-amino acid oxidase in some snake venoms (Vennesland et al. 1981b). This enzyme is harmless on injection, but the tissue destruction caused by other venom components probably provides the required substrate and cofactor for HCN production.

Cyanide inhibits ion transport mechanisms in amphibian skin, gall bladder, and proximal renal tubules (Bello-Reuss et al. 1981). Measurable changes in cell membrane potentials of isolated gall bladder epithelium cells, for example, were induced by NaCN in a salamander (Bello-Reuss et al. 1981). Cyanide-induced hyperpolarization was caused primarily by an increase in permeability of the cell membrane to potassium, which, in turn, was mediated by an elevation of intracellular calcium ion activity, attributable to release from mitochondrial sources.

The binding rate of CN to hemeproteins, specifically hemoglobin components Ill and IV, is 370 times to 2,300 times slower in a marine polychaete annelid (*Glycera dibranchiata*), when compared to guinea pig (*Cavia* spp.), soybean (*Glycine* max), and sperm whale (*Physeter macrocephalus*); the significance of this observation is unclear but warrants further exploration (Mintorovitch et al. 1989).

Clinical Features

Accidental exposure to cyanides or cyanogens through inhalation, skin exposure, and swallowing occurs in agricultural fumigation, laboratories, industrial operations, domestic abuse, and products of combustion (Ballantyne and Marrs 1987b). Intentional exposure is reported from homicides, suicides (usually uncommon), judicial executions, chemical warfare, and covert activities (Ballantyne and Marrs 1987b).

Diagnosis of lethal cyanide poisoning is difficult because of the absence of gross pathology or histology, nonspecific congestion of viscera, and cerebral or pulmonary edema. Sometimes the blood is bright red, and sometimes the odor of bitter almonds is detected, but neither is sufficiently consistent for diagnostic purposes (Ballantyne and Marrs 1987b).

At low lethal doses of cyanide, the effects are principally on cytochrome oxidase in the central nervous system. At higher doses, cardiovascular signs and changes in electrical activity of the brain are among the most consistent changes measured (Way 1981, 1984). Acute and subacute toxic effects of poisoning with cyanide can vary from convulsions, screaming, vomiting, and bloody frothing to less dramatic events, such as a slow, quiet onset to coma and subsequent death (Way 1981). In the first stage of cyanide poisoning, victims exhibit headache, vertigo, weak and rapid pulse, nausea, and vomiting. In the second stage, there are convulsions, falling, dilated pupils, clammy skin, and a weaker and more rapid pulse. In the final stage, heartbeat becomes irregular and slow; body temperature falls; there is cyanosis of lips, face, and extremities, coma, frothy bloody saliva flow from mouth, and death (Way 1981). If acute exposure is to a sublethal dose of cyanide, this may lead to signs of toxicity, but as detoxification proceeds these signs will become less obvious and eventually vanish, and cyanide will be excreted as thiocyanate without accumulating (Ballantyne 1987a).

Chronic cyanide poisoning may develop in individuals who ingest significant quantities of cyanide or cyanide precursors in their diets; effects are exacerbated by dietary deficiencies in vitamin B_{12}, iodine, and sulfur amino acids, as well as by low levels and insufficient distribution of detoxifying enzymes such as rhodanese (Solomonson 1981). Cyanide toxicity of dietary origin has been implicated in acute animal deaths and as a major etiologic factor in toxic ataxic neuropathy in humans, and as a cause of blindness in humans suffering from tobacco amblyopia and Leber's hereditary optic atrophy (Egekeze and Oehme 1980). An increase in blood plasma cyanide is observed in healthy individuals who smoke cigarettes (Cailleux et al. 1988). An increase in blood plasma thiocyanate is also seen in smokers and in hemodialysis patients just before dialysis (Cailleux et al. 1988). Continuous intake of cyanide causes high levels of plasma thiocyanate and goiters in mammals; the antithyroid action (goiters) results from cyanide interference with iodine transport and thyroxine synthesis (Solomonson 1981; Leduc 1981, 1984). Signs of chronic cyanide poisoning include demyelination, lesions of the optic nerve, decrease in sulfur-containing amino acids, increase in thiocyanate, goiter, ataxia, hypertonia,

and depressed thyroid function (Solomonson 1981). These effects are common in areas that depend on cyanogenic plants--such as cassava--as a major dietary component (Solomonson 1981).

Biochemically, cyanide affects the citric acid cycle; strongly inhibits catalases and proteinases; induces glycolysis in protozoans, fish, and mammals; produces vitamin B_{12} deficiency; and modifies the phosphorylation mechanism of respiratory mitochondrial enzymes, causing arrested respiration due to inability to use oxygen (Leduc 1984).

Cyanide biomagnification or cycling has not been reported, probably because of cyanide's high chemical reactivity and rapid biotransformation (Towill et al. 1978; Marrs and Ballantyne 1987).

There is no evidence that chronic exposure to cyanide results in teratogenic, mutagenic, or carcinogenic effects (EPA 1980). Cyanide possibly has antineoplastic activity, as judged by a low therapeutic success against rat sarcomas (EPA 1980), but this requires additional documentation.

Confirmatory evidence of cyanide poisoning includes elevated blood thiocyanate levels--except, perhaps, when death was rapid--and reduced cytochrome oxidase activity in brain and myocardium, provided that all tissues were taken within a day or so of death, frozen quickly, and analyzed shortly thereafter (Biehl 1984; Marrs and Ballantyne 1987). Evaluation of cyanide poisoning and metabolism includes signs of toxicity, LD50 values, measurement of cyanide and thiocyanate concentrations, cytochrome c oxidase activity, metabolic modification of in vivo cyanogenesis, rate of cyanide liberation in vitro, and influence of modifying factors such as the animal species, dose, rate and frequency of administration, route of exposure, differential distribution of cyanide, detoxification rates, circadian rhythm interactions, age of the organism, and presence of antidotes (Ballantyne 1987a). For example, the concentration of cyanide measured in body fluids and tissues in humans and other animals following lethal administration of cyanide depends on several factors: route of exposure, with oral route yielding highest residues and inhalation route the lowest; amount and duration of exposure; nature of the material, with HCN and CN^- being most toxic; time to death; antidotes used; time to autopsy, with marked loss documented from simple evaporation, thiocyanate formation, hydrolysis, and polymerization; and time from autopsy to sample analysis, wherein cyanide concentrations may increase due to microbial action (Ballantyne and Marrs 1987b).

Antidotes

The antagonism of cyanide intoxication has been under investigation for at least 150 years. In 1840, cyanide lethality was reported to be antagonized by artificial respiration. In 1888, amyl nitrite was reported effective in antagonizing lethal effects of cyanide in dogs. In 1894, cobalt was shown to form a stable metal complex with cyanide and was used to antagonize cyanide. In 1933, the use of sodium thiosulfate as the sulfur donor was described (Way 1984). Many compounds are used today as cyanide antidotes including cobalt salts, rhodanese, sulfur donors, methemoglobin producers, carbohydrates, drugs used to treat acidosis, oxygen, methylene blue, 4-dimethylaminophenol, various aromatic amino- and nitro-compounds (such as aniline, p-aminopropiophenone, nitrobenzene), carbonyl compounds, and sodium pyruvate (Egekeze and Oehme 1980; EPA 1980; Solomonson 1981; Way 1981, 1984; Biehl 1984; Becker 1985; Ballantyne 1987b; Marrs 1987; Marrs and Ballantyne 1987; Way et al. 1988). Different antidotes are preferred in different countries: in the United States, a mixture of sodium nitrite and sodium thiosulfate; in France and the United Kingdom, cobalt edetate, also known as Kelocyanor; and in Germany, a mixture of 4-dimethylaminophenol and sodium thiosulfate.

The classic nitrite-thiosulfate treatment of cyanide poisoning, developed almost 60 years ago, is one of the antidotal combinations still employed (Way 1981). Excess oxygen improves this antidotal combination by potentiating the effectiveness of the nitrite-thiosulfate combination, as confirmed by studies in sheep and rats (Way 1984), even though, theoretically, oxygen should serve no useful purpose (Way et al. 1988). This therapeutic regimen protected rats against 20 LD50 doses of cyanide (Towill et al. 1978). Nitrite converts hemoglobin to methemoglobin, which has a high affinity for cyanide. The methemoglobin-HCN complex then slowly releases cyanide, which is converted to thiocyanate by way of rhodanese (Solomonson 1981). Sodium nitrite, administered intravenously, is now considered one of the more rapid therapeutic methods (Way 1984). The injection of sodium thiosulfate provides sulfur for the enzyme rhodanese to mediate the biotransformation of cyanide to the much less toxic thiocyanate (Egekeze and Oehme 1980). Multiple injections of sodium thiosulfate protected mice against death by organic cyanides and were more effective than sodium nitrite

(Willhite and Smith 1981). The nitrite-thiosulfate antidotal combination is one of the most effective treatments of cyanide poisoning, even though the specific mechanism of action of these two compounds is now being questioned, and concerns have been raised because of the toxicity of nitrite (Way 1981, 1984). One accepted therapy is an intravenous combination of sodium nitrite (1 mL of 20% solution) and sodium thiosulfate (3 mL of 20% solution), giving 4 mL of this mixture per 45 kg of body weight (Egekeze and Oehme 1980). For maximal effectiveness in treating cyanide intoxication in sheep, large doses of sodium thiosulfate (660 mg/kg BW) are given in combination with conventional doses of sodium nitrite (6.6 mg/kg BW; Egekeze and Oehme 1980). Livestock treatment in cases of suspected cyanide intoxication consists of intravenous administration of sodium nitrite at 10-20 mg/kg BW followed by sodium thiosulfate at 30-40 mg/kg BW; however, a sodium thiosulfate dose of 500 mg/kg BW, or more, may be more efficacious (Biehl 1984). Once clinical signs have abated, 1 g of activated charcoal per kilogram BW may be administered as a drench by way of a stomach tube (Biehl 1984). A 30-kg female goat *(Capra* sp.) was successfully treated after eating the leaves and fruit of the crab apple *(Malus sylvestris),* a plant that contains high levels of cyanogenic glycosides in leaves and fruits (Shaw 1986). Treatment consisted of four hourly treatments of 100 g of animal charcoal and bismuth subnitrate in water as a drench, followed by 300 mg sodium nitrite as a 1% aqueous solution, then 25 g of sodium thiosulfate. Another goat died despite identical treatment (Shaw 1986).

Cobalt compounds, such as hydroxocobalamin and its derivatives (i.e., cobalt histidine, cobalt chloride, dicobalt ethylenediamine tetracetic acid) have been used to treat cyanide poisoning for more than 100 years. Their efficacy was confirmed in pigeons (*Columba* sp.) and rabbits (*Oryctolagus sp*.), but cobalt compounds did not receive wide support as cyanide antagonists because of the inherent toxicity of cobalt ion (Way 1981, 1984). Nevertheless, proponents of the use of cobalt compounds (i.e., the United Kingdom, Scandinavia, much of Europe) stress the rapidity of action in forming a stable metal complex with cyanide, thereby preventing its toxic effect (Towill et al. 1978; Way 1984). One of the more frequently used cobalt compounds in cyanide treatment is hydroxocobalamin, which reverses cyanide toxicity by combining with cyanide to form cyanocobalamin (EPA 1980; Solomonson 1981). Hydroxocobalamin has been used in guinea pigs and baboons *(Papio anubis)* to lower blood cyanide levels, and in humans after inhalation or ingestion of cyanide compounds (Egekeze and Oehme 1980).

Dimethylaminophenol (DMAP) forms methemoglobin by setting up a catalytic cycle inside the erythrocyte, in which oxygen oxidizes the DMAP to N-N-dimethylquinoneimine, the latter oxidizing the hemoglobin to methemoglobin (Marrs 1987). Dogs poisoned with KCN and given DMAP intravenously had restored respiration and decreased plasma cyanide levels. The 4-dimethylamino-phenol induced ferrihemoglobin production, which combined with the cyanide in the red cells to form ferrihemoglobin cyanide (Christel et al. 1977).

No usable cyanide prophylactic therapy now exists for humans, although sodium thiosulfate, hydroxocobalamin, and other compounds have been used to protect against cyanide toxicity in laboratory animals (Mengel et al. 1989). For example, pyridoxal 5-phosphate, the active form of vitamin B_6, readily forms complexes with cyanides, and was effective in providing significant protection to rats (Keniston et al. 1987). Fructose fed prior to insult lessens cyanide-induced hepatotoxicity in rats (Younes and Strubelt 1988). L-ascorbic acid and dehydroascorbic acid probably act as protectants against cyanide toxicity by way of nontoxic cyanohydrin formation (Sprince et al. 1982). Carbon tetrachloride pretreatment was effective in protecting mice against death from most nitriles (Willhite and Smith 1981), and pretreatment with p-aminopropiophenone serves to protect against cyanide toxicity (D'Mello 1987).

Sources and Uses

Production of cyanides in the United States increased from about 136 million kg in 1963 to 318 million kg in 1976 (Towill et al. 1978; Way 1981; Marrs and Ballantyne 1987). Cyanide consumption in North America was 64 million kg in 1988 and 98 million kg in 1989; about 80% of these amounts was used in gold mining (Knudson 1990).

About 84% of domestic HCN production is used to produce organic cyanides, also known as nitriles, including acrylonitriles, methyl methacrylate, and adiponitrile (Towill et al. 1978). Nitriles tend to polymerize, which is the basis for their use in the manufacture of synthetic fibers, resins, plastics, dyestuffs, vitamins, solvents, elastomers, agricultural insecticides, and high pressure lubricants (Willhite and Smith 1981). The widespread usefulness of HCN is related to its strong tendency and that of its inorganic salts to form complexes

with metals. For example, sodium cyanide is used in metallurgy for the extraction of gold and silver from ores and in electroplating baths because it forms stable soluble complexes. Similar behavior makes alkali cyanide solutions excellent for cleaning silverware and other precious metals and is responsible for their general use in industry as metal cleaners (Towill et al. 1978). In Canada, more than 90% of the gold mined 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 precipitated with the addition of zinc dust. A variety of cyanide compounds are produced during gold cyanidation (Simovic and Snodgrass 1985). In addition to their primary use in the metals and electroplating industries, and in the manufacture of synthetic fibers and plastics, various cyanide compounds have been used directly or as an intermediate to produce synthetic rubber, fumigants, rodenticides, insecticides, predator control agents, rocket fuels, paints and paint finishes, paper, nylon, pharmaceuticals, photographic chemicals, mirrors, cement, perfume, bleaches, soaps and detergents, riot control agents, fertilizers, and herbicides (Towill et al. 1978; Way 1981; Willhite and Smith 1981; Leduc 1984; Homan 1987).

Hydrogen cyanide vapor, because of its high and rapid acute lethal toxicity and ready diffusion, has been used widely to fumigate buildings, ships, and warehouses; to exterminate rabbits, rodents, and large predators; and in horticultural practice, to control insect pests that have developed resistance to other pesticides (Homan 1987; Ballantyne 1988). Typically, fumigation powders containing either calcium cyanide, $Ca(CN)_2$, or sodium cyanide, NaCN, are blown into burrows or scattered over the floor in greenhouses. On coming into contact with water, such powders liberate HCN vapor (Ballantyne 1988). Hydrogen cyanide released from $Ca(CN)_2$ is registered for use on almonds, dried beans, citrus, cocoa beans, grains, nuts, and spices (Towill et al. 1978). Cyanide-containing compounds are used for a variety of agricultural and pesticidal agents. These compounds include cyanogen (NCCN), as an intermediate in the production of some commercial fertilizers; cyanogen chloride (CNCl), in the manufacture of triazine herbicides; cyanogen bromide (CNBr), as a pesticidal fumigant; hydrogen cyanide, in the synthesis of methionine for animal feeds; ammonium thiocyanate (NH_4SCN), as a cotton defoliant; sodium thiocyanate (NaSCN), as a weedkiller; and calcium cyanamide (CaNCN), as a plant fertilizer, herbicide, pesticide, and defoliant of cotton and tomatoes (Homan 1987). Cyanide compounds have also been used as preservatives for raw vegetables (Towill et al. 1978).

Sodium cyanide has been used for about 50 years by the U.S. Fish and Wildlife Service against coyote in attempts to protect livestock, especially sheep. The Service has made extensive use of two NaCN ejector devices: "the coyote getter," from the late 1930's to 1970; and the M-44, from about 1968 to the present, except for the period 1972-74, when all uses of NaCN for predator control were canceled (EPA 1976a; Connolly and Simmons 1984). Although both ejectors dispense toxicant when pulled, they differ in the way ejection is achieved. In the coyote getter, the toxicant is in a 0.38-caliber cartridge case and is expelled by the explosive force of the primer plus a small powder charge. The M-44 uses a spring-driven plunger to push out its toxic contents. M-44 capsules weigh about 0.94 g, and consist of about 89% NaCN, 6% Celatom MP-78 (mostly diatomaceous silica), 5% potassium chloride, and 0.25% FP Tracerite yellow--used as a fluorescent marker (Connolly and Simmons 1984). Coyote getters and M-44's are set into the ground with only their tops protruding. Fetid scent or lure stimulates a coyote to bite and pull, whereupon a lethal dose of NaCN is ejected into its mouth; coma and death follow in 30 to 60 s. Although coyote getters were about 99% effective against coyotes, compared with 73% for M-44's, the Service decided that spring-driven plungers were less hazardous to operators than were explosive-driven plungers (Connolly and Simmons 1984). The coyote getter was generally much more selective than the trap for the capture of coyotes. It was less destructive than traps to small mammals, birds of prey, ground-nesting birds, deer, antelope, and domestic sheep, but more destructive to dogs, bears, and cattle (Robinson 1943). In a 1-year test period (1940-41) in Colorado, Wyoming, and New Mexico, the following numbers of animals were killed by the coyote getter: 1,107 coyotes, 2 bobcats (*Lynx rufus*), 24 dogs, 14 black-billed magpies (*Pica pica*), 7 foxes (*Vulpes* sp.), 8 unidentified skunks, 2 badgers, 2 unidentified eagles, 2 bears (*Ursus* sp.), and 1 each of hawk (unidentified), pika (*Ochotona* sp.), and cow (Robinson 1943).

Cyanide compounds have been used to collect various species of freshwater fish. In England and Scotland, cyanides are used legally to control rabbits, and illegally to obtain Atlantic salmon (*Salmo salar*) and brown trout (*Salmo trutta*) from rivers, leaving no visible evidence of damage to the fish (Holden and Marsden 1964). Sodium cyanide has been applied to streams in Wyoming and Utah to collect fish through anesthesia; mountain whitefish (*Prosopium williamsoni*) were sensitive to cyanide and died at concentrations that were tolerable to salmon and trout (Wiley 1984). Sodium cyanide was also used as a fish control agent in Illinois, Nebraska,

South Dakota, Missouri, and in the lower Mississippi River valley, but was never registered for this use because of human safety concerns (Lennon et al. 1970).

Cyanide compounds have been prescribed by physicians for treatment of hypertension and cancer (Sprince et al. 1982). Sodium nitroprusside ($Na_2Fe(CN)_5NO\text{-}2H_2O$) was widely used for more than 30 years to treat severe hypertension and to minimize bleeding during surgery (Solomonson 1981; Vesey 1987). Laetrile, an extract of ground apricot kernels, has been used for cancer chemotherapy and, in deliberate high intakes, as an attempted suicide vehicle (Gee 1987).

Road salt in some areas may contribute to elevated cyanide levels in adjacent surface waters (Ohno 1989). In climates with significant snowfall, road salt is applied as a deicing agent. Road salts are commonly treated with anticaking agents to ensure uniform spreading. One anticaking agent, sodium hexacyanoferrate, decomposes in sunlight to yield the highly toxic free cyanide that contaminates surface waters by runoff (Ohno 1989). Another anticaking agent, yellow prussiate of soda (sodium ferrocyanide), has been implicated in fish kills when inadvertently used by fish culturists (Barney 1989).

The military uses of HCN were first realized by Napoleon III, but it was not until World War I (WW I) that this application received widespread consideration. About 3.6 million kg of hydrogen cyanide were manufactured by France as a chemical weapon and used in WWI in various mixtures called Manganite and Bincennite, although its use was not highly successful because of limitations in projectile size and other factors. During WW II, the Japanese were armed with 50-kg HCN bombs, and the United States had 500-kg bombs. More than 500,000 kg of HCN chemical weapons were produced during WWII by Japan, the United States, and the Soviet Union, but it is not known to what extent these weapons were used in that conflict (Way 1981).

Cyanides are widely distributed among common plants in the form of cyanogenic glycosides (Egekeze and Oehme 1980; Solomonson 1981; Way 1981; Biehl 1984; Homan 1987; Marrs and Ballantyne 1987). Their toxicity following ingestion is primarily related to the hydrolytic release of HCN. Ingestion of cyanogenic plants probably has accounted for most instances of cyanide exposure and toxicosis in man and range animals. Of chief agricultural importance among plants that accumulate large quantities of cyanogenic glycosides are the sorghums, Johnson grass, Sudan grass, corn, lima beans, flax, pits of stone fruits (cherry, apricot, peach), vetch, linseed, sweet potatoes, bamboo shoots, southern mock orange, millet, almonds, and cassava. Factors favoring cyanide build-up in cyanogenic plants include high nitrogen and low phosphorus in soils (Biehl 1984); the potential for high glycoside levels is greatest in immature and rapidly growing plants (Egekeze and Oehme 1980). At present, more than 28 different cyanoglycosides have been measured in about 1,000 species of higher plants (Leduc 1984). In cassava, for example, more than 90% of the cyanide is present as linamurin, a cyanogenic glycoside, and the remainder occurs as free (nonglycoside) cyanide (Gomez et al. 1983). Laetrile, a preparation made from apricot kernels, contains high levels of amygdalin, a cyanogenic glycoside that can be degraded in the gut to cyanide and benzaldehyde. Several cases of cyanide poisoning in humans have been reported from intake of laetrile, either orally or anally (Solomonson 1981; Homan 1987). Cyanide formation in higher plants and microorganisms can also occur with compounds other than cyanogenic glycosides, such as glycine, glyoxylate plus hydroxylamine, or histidine (Solomonson 1981; Vennesland et al. 1981b). In some cases, plants may contain cyanide residues resulting from fumigation with HCN (Way 1981).

Many species of plants, including some fungi, bacteria, algae, and higher plants, produce cyanide as a metabolic product (Leduc et al. 1982; Leduc 1984). Some species of soil bacteria suppress plant diseases caused by soilborne pathogens by producing metabolites with antibiotic activity. Certain strains of *Pseudomonas fluorescens,* a soil bacterium, suppress black root rot of tobacco caused by the fungus *Thielaviopsis basicola* by excreting several metabolites, including HCN (Voisard et al. 1989). A wide variety of bacteria and fungi can degrade cyanide compounds, and may be useful in the treatment of cyanide wastes (Towill et al. 1978). For example, several species of fungi known to be pathogens of cyanogenic plants can degrade cyanide by hydration to formamide; dried mycelia of these species are now sold commercially to detoxify cyanide in industrial wastes (Knowles 1988).

Anthropogenic sources of cyanide in the environment include industrial processes, laboratories, fumigation operations, cyanogenic drugs, fires, cigarette smoking, and chemical warfare operations (Marrs and Ballantyne 1987). Cyanides are present in many industrial wastewaters, especially those of electroplaters; manufacturers of paint, aluminum, and plastics; metal finishers; metallurgists; coal gasification processes; certain mine

operations; and petroleum refiners (Towill et al. 1978; Egekeze and Oehme 1980; Way 1981, 1984). Electroplaters are a major source. In the United States alone, electroplaters discharge about 9.7 million kg of cyanide wastes annually into the environment from 2,600 electroplating plants (Marrs and Ballantyne 1987). Paint residues annually contribute an additional 141,300 kg of cyanide wastes into the environment, and paint sludges 20,400 kg (Way 1981; Marrs and Ballantyne 1987). Cyanide can also originate from natural processes, such as cyanide production by bacteria, algae, and fungi, and from many terrestrial plants that release free HCN when their cellular structure is disrupted (Leduc 1981). Hospital wastewaters usually contain no detectable cyanide, but concentrations up to 64 µg CN^-/L have been measured after alkali chlorination treatment (Tatsumoto and Hattori 1988). It seems that various compounds common in hospital wastewaters will produce 15-25 µg CN^-/L after alkali chlorination; these compounds include hydantoin (an antiepilepsy agent) and related nitrogenous compounds, such as hydantonic acid, 5,5-diphenyl hydantoin, imidazole, and 2-imidazolidinone (Tatsumoto and Hattori 1988).

Free hydrogen cyanide occurs only rarely in nature because of its high reactivity. The gas is sometimes found in the atmosphere, however, as a result of emissions from the petrochemical industry, malfunctioning catalytic converters on automobiles, fumigation of ships and warehouses, incomplete combustion of nitrogen-containing materials, and from tobacco smoke (Towill et al. 1978; Way 1981, 1984). Hydrogen cyanide is known to be produced in fires involving nitrogen-containing polymers and is probably the most important narcotic fire product other than carbon monoxide (Purser et al. 1984). Cyanide-related fire deaths and injuries, as judged by elevated blood cyanide and thiocyanate concentrations, have been documented in airplanes, jails, and high-rises (Becker 1985; Ballantyne 1987b; Lundquist and Sorbo 1989). In a study of fire victims in Scotland, elevated blood cyanide levels were found in 78% of fatalities, and 31% had blood levels considered to be toxic (Purser et al. 1984). Major factors that influence HCN release include the chemical nature of the material, temperature, oxygen availability, and burning time (Ballantyne 1987b). Substantial quantities of free HCN and organic cyanides are known to be produced in fire settings involving horsehair, tobacco, wool, silk, and many synthetic polymers, such as polyurethane and polyacrylonitriles (Egekeze and Oehme 1980; Purser et al. 1984; Becker 1985; Ballantyne 1987b). Polyacrylonitrile, for example, is used in fabrics, upholstery covers, paddings, and clothing; about 50% of the mass of the polymer is theoretically available as HCN under thermal decomposition (Purser et al. 1984; Homan 1987).

Background Concentrations

The reactivity of HCN, and its ability to condense with itself and other compounds, was probably responsible for the prebiotic formation of the majority of biochemical compounds required for life (Marrs and Ballantyne 1987). Cyanide is now known to be present in a number of foodstuff and forage plants, as a metabolite of certain drugs, and in various industrial pollutants; it also may be formed by the combustion of cyanide-releasing substances, such as plastics in airplane fires and tobacco in smoking (Robinson et al. 1985). Hydrogen cyanide production may occur in hepatopancreas of mussels, *Mytilus edulis* (Vennesland et al. 1981b), in rat liver (Solomonson 1981), and in green and blue-green algae during nitrate metabolism (Leduc et al. 1982). Except for certain naturally occurring organic cyanide compounds in plants, it is uncommon to find cyanide in foods consumed in the United States (EPA 1980).

The cyanide anion is found in a variety of naturally occurring plant compounds as cyanogenic glycosides, glycosides, lathyrogenic compounds, indoleacetonitrile, and cyanopyridine alkaloids. Plants that contain cyanogenic glycosides are potentially poisonous because bruising or incomplete cooking can result in glycoside hydrolysis and release of HCN (Towill et al. 1978). Cyanide concentrations in cyanogenic plants are usually highest in leaves of young plants; levels drop rapidly after pollination (Biehl 1984). There are about 20 major cyanogenic glycosides, of which usually only one or two occur in any plant. They are synthesized from amino acids and sugars and are found in many economically important plants, such as sorghum, flax, lima bean, cassava, and many of the stone fruits (Table 2; Towill et al. 1978; Shaw 1986). Cassava contains linamurin and lotaustralin, whereas the main cyanogenic glycoside in cereals is dhurrin; consumption of foods containing toxic cyanogens (primarily cassava) has been associated with death or morbidity--on an acute basis--or goiter and tropical ataxic neuropathy on a chronic consumption basis (Okolie and Ugochukwu 1989). Cassava is a perennial shrub, native to the neotropics, grown for its tuberous starchy roots, and a traditional dietary staple of many indigenous populations in Amazonia, especially the Tukanoan Indians in northwestern Amazonia (Dufour 1988). Cassava is one of the few food plants in which the cyanide content may create toxic problems. All

varieties of cassava contain cyanogenic glycosides capable of liberating HCN, but amounts vary greatly depending on variety and environmental conditions. Bitter cultivars of cassava provide over 70% of the Tukanoan's food energy, appearing in the diet as bread, meal, a starch drink, and boiled cassava juice. The greatly elevated total cyanide content in bitter varieties (Table 2) may contain 5.1-13.4% of the total as the toxic free cyanide (Dufour 1988).

The production of HCN by animals is almost exclusively restricted to various arthropods: 7 of about 3,000 species of centipedes; 46 of 2,500 species of polydesmid millipedes; and 10 of 750,000 species of insects, including 3 species of beetles, 4 moths, and 3 butterflies (Duffey 1981). Millipedes--which are eaten frequently by toads and starlings--secrete cyanide for defensive purposes in repelling predators; in zygaenid moths, cyanide seems to be localized in eggs (Table 2; Duffey 1981).

Cyanide concentrations in fish from streams that were deliberately poisoned with cyanide ranged between 10 and 100 µg total cyanide per kilogram whole body fresh weight (FW; Wiley 1984). Total cyanide concentrations in gill tissues of salmonids under widely varying conditions of temperature, nominal water concentrations, and duration of exposure ranged from about 30 µg/kg FW to >7,000 µg/kg (Holden and Marsden 1964). Unpoisoned fish usually contained < 1 µg/kg FW in gills, although values up to 50 µg/kg occurred occasionally. Lowest cyanide concentrations in gills occurred at elevated (summer) water temperatures; at lower temperatures, survival was greater and residues were higher (Holden and Marsden 1964). Fish retrieved from cyanide-poisoned environments, dead or alive, can probably be consumed by humans because muscle cyanide residues were considered to be low (i.e., <1,000 mg/kg FW; Leduc 1984).

Cyanide pollution is likely to occur in many places, ranging from industrialized urban areas to gold mines in the western United States and Northwest Territories of Canada (Table 2). Cyanides are ubiquitous in industrial effluents, and their increasing generation from power plants and from the combustion of solid wastes is expected to result in elevated cyanide levels in air and water (Leduc 1984). However, data are scarce on background concentrations of cyanides in various nonbiological materials. In soils, for example, high concentrations are unusual and are nearly always the result of improper waste disposal (Towill et al. 1978). Cyanides in soils are not absorbed or retained; under aerobic conditions, microbial metabolism rapidly degrades cyanides to carbon dioxide and ammonia; under anaerobic conditions, cyanides are converted by bacteria to gaseous nitrogen compounds that escape to the atmosphere (Towill et al. 1978). Heat treatment wastes from metal processing operations may contain up to 200 g CN/kg, mostly as NaCN, and are frequently hauled to landfills for disposal (Lagas et al. 1982). The presence of cyanide in landfill waste is potentially hazardous because of the possibility that cyanide may leach to soil and groundwater, release HCN, and disturb natural microbiological degradation of organic materials. Measurements at landfills in England and the Netherlands showed total cyanide levels up to 560 g/kg in soil and 12 µg/L in groundwater (Lagas et al. 1982). However, 7-month-long experimental studies of cyanide in heat treatment wastes in landfills showed that between 72 and 82% of the cyanide was converted, mostly to ammonium and organic nitrogen compounds; between 4 and 22% of the cyanide leached as free or complex cyanide; and up to 11% remained in the landfill (Lagas et al. 1982).

Table 2. Background concentrations of cyanide in selected living resources and nonbiological materials. Values are in milligrams total cyanide per kilogram fresh weight or milligrams per liter.

Environmental compartment	Concentration[a] (mg/kg or mg/L)	Reference[b]
Biological		
Cyanogenic plants		
Bamboo (*Bambusa, Arundinaria, Dendrocalamus*)		
Tip	Max. 8,000	1
Stem	Max. 3,000	1
Stargrass, *Cynodon plectostachyus*, whole	180	1
Rose family, *Malus* spp., *Pyrus* spp.	Max. 200	2
Cassava, *Manihot esculenta*		
Bitter varieties		
Leaves	347–1,000	3, 4

Roots	327–550	1, 4
Dried roots	95–2,450	1, 3, 4
Stem	1,130	1
Mash	162	5
Bark		
Total cyanide	1,351	6
Free cyanide	102	6
Peel		
Total cyanide	1,390	6
Free cyanide	255	6
Pulp		
Total cyanide	810	6
Free cyanide	53	6
Sweet varieties		
Leaves	377–500	3, 4
Roots	138	4
Dried roots	46–<100	3, 4
Mash	81	5
Lima beans, *Phaseolus lunatus*		
United States	100–170	1
Burma	2,100	1
Puerto Rico	3,000	1
Java	3,120	1
Almond, *Prunus amygdalus*, nut		
Bitter	(280–2,500)	1
Spicy	(86–98)	1
Sweet	(22–54)	1
Seeds, 4 species, Nigeria, whole, frequently consumed by humans		
Phaseolus sp.	(381–1,093)	7
Vigna sp.	(285–1,223)	7
Cajanus sp.	(208–953)	7
Canavalia sp.	(285–953)	7
Sorghum, *Sorghum spp.*, young plant, whole	Max. 2,500	1
Cyanogenic arthropods		
Millipede, *Apheloria corrugata*, whole	428	8
Millipede, *Apheloria kleinpeteri*, whole	18	8
Zygaenid moth, *Zygaena filipendulae*, whole	668	8
Mammals		
Humans, *Homo sapiens*		
Blood		
Normal	<0.2	9
Afflicted with Leber's optic atrophy	1.4	9
Plasma		
Nonsmokers	0.05; Max. 0.11	10
Smokers	0.075; Max. 0.3	10
Nonbiological		
Air		
Automobile exhaust		
Adverse conditions	Max. 10.0	1
Equipped with catalytic convertor	1.1	1
Sewage sludge		

From publicly owned treatment works, United States	749[c]	18
Water, uncontaminated		
Rural watersheds	0.003	11,12
Industrial areas	0.02	11, 12
Small watersheds, covered with grasslands and forest, uninhabited by humans	0.0007–0.002; Max. 0.005	12
Western and central Canada, 11 rivers, 1974–77	Max. 0.006	12
U.S. water supplies, 2,595 samples nationwide	0.0009; Max. 0.008	1, 13
U.K. water supplies	<0.05; Max. 0.1	1
Wastewaters/runoff		
Electroplaters		
Total cyanide	0.2; Max. 3.0	14, 15
Dissociable cyanide	0.07	15
Complex cyanide	0.2	15
Thiocyanate	0.02	15
Plating wastewater		
Before treatment with alkaline chlorination	0.18	16
After treatment	0.005	16
Road salt dock		
Total cyanide	25.6	15
Dissociable cyanide	2.9	15
Complex cyanide	23.1	15
Thiocyanate	0.0	15
Steel industry		
Plating baths	72 (9–115)	1, 14
Coke oven liquor	6 (0–8)	1
Oil refineries		
Total cyanide	0.01; Max. 4.0	14, 15
Dissociable cyanide	0.0	15
Complex cyanide	0.01	15
Thiocyanate	2.2	15
Coking operations		
Total cyanide	2.1	15
Dissociable cyanide	0.3	15
Complex cyanide	0.8	15
Thiocyanate	23.6	15
Hospital wastewaters		
Before alkaline chlorination	ND	17
After treatment	0.06	17
Gold mills, Canada	0.3–26.5	14
Gold mine cyanide extraction leach ponds, California, Nevada, and Arizona	Usually 200–300, frequently 700, occasionally 9,000	19
Wastewater treatment plants,		
Chicago		
Treated effluent		
Total cyanide	0.005–0.03	15
Dissociable cyanide	0.003–0.007	15
Complex cyanid	0.002–0.02	15
Thiocyanate	0.006–0.03	15
Untreated wastewater		
Total cyanide	0.02–0.06	15

Dissociable cyanide	0.004–0.05	15
Complex cyanide	0.02–0.08	15
Thiocyanate	0.03–0.27	15
Sludge		
Total cyanide	0.49–3.79	15
Dissociable cyanide	0.06–0.44	15
Complex cyanide	0.43–5.4	15
Thiocyanate	0.2–0.9	15

[a]Concentrations are shown as means, range (in parentheses), and maximum (Max.).

[b]1, Towill et al. 1978; 2, Shaw 1986; 3, Gomez et al. 1983; 4, Casadei et al. 1984; 5, Ukhun and Dibie 1989; 6, Dufour 1988; 7, Okolie and Ugochukwu 1989; 8, Duffey 1981; 9, Berninger et al. 1989; 10, Egekeze and Oehme 1980; 11, Leduc 1981; 12, Leduc 1984; 13, EPA 190; 14, Leduc et al. 1982; 15, Kelada 1989; 16, Nonomura and Hobo 1989; 17, Vennesland et al. 1981a; 18, Beyer 1990; 19, Clark and Hothem 1991.

[c]Concentration is in milligrams per kilogram dry weight.

Hydrogen cyanide (HCN) is a common industrial pollutant and frequently occurs in water at concentrations between 0.1 and several milligrams per liter of free HCN (Leduc 1978; Leduc et al. 1982). Total cyanides is the most often cited measurement in aqueous solutions, owing to limitations in analytical methodologies (Leduc et al. 1982). Cyanides have been identified in fresh waters of rural and wilderness areas in Canada and Germany. Concentrations ranging between 30 and 60 µg total cyanides per liter seem related to runoff, with cyanide peaks more frequent in fall and winter during periods of minimal runoff (Leduc et al. 1982). In larger rivers, cyanide was low in winter owing to dilution by high runoff, but peaked in summer because of cyanide production by plants (Leduc 1984). Cyanides do not seem to persist in aquatic environments. In small, cold oligotrophic lakes treated with 1 mg NaCN/L, acute toxicity was negligible within 40 days. In warm shallow ponds, toxicity disappeared within 4 days after application of 1 mg NaCN/L. In rivers and streams, toxicity rapidly disappeared on dilution (Leduc 1984). Cyanide was not detectable in water and sediments of Yellowknife Bay, Canada, between 1974 and 1976, although the bay receives 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 (EPA 1980). An alkaline pH favors oxidation by chlorine, and an acidic pH favors volatilization of HCN into the atmosphere (EPA 1980).

Persistence in Water, Soil, and Air

In water, cyanides occur as free hydrocyanic acid, simple cyanides, easily degradable complex cyanides such as $Zn(CN)_2$, and sparingly decomposable complex cyanides of iron and cobalt; complex nickel and copper cyanides are intermediate between the easily decomposable and sparingly degradable compounds (Towill et al. 1978). Cyanide has relatively low persistence in surface waters under normal conditions but may persist for extended periods in groundwater (Way 1981). Volatilization is the dominant mechanism for removal of free cyanide from concentrated solutions and is most effective under conditions of high temperatures, high dissolved oxygen levels, and at increased concentrations of atmospheric carbon dioxide (Leduc et al. 1982; Simovic and Snodgrass 1985). Loss of simple cyanides from the water column is primarily through sedimentation, microbial degradation, and volatilization (Leduc et al. 1982; Marrs and Ballantyne 1987). Water-soluble strong complexes, such as ferricyanides and ferrocyanides, do not release free cyanide unless exposed to ultraviolet light. Thus, sunlight may lead to cyanide formation in wastes containing iron-cyanide complexes (Towill et al. 1978; Leduc et al. 1982; Simovic and Snodgrass 1985; Marrs and Ballantyne 1987).

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). 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; EPA 1980; Way 1981; Marrs and Ballantyne 1987). In the case of Canadian gold-mining operations, the primary treatment for cyanide removal is to retain gold 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 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 (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.

Volatile cyanides occur only occasionally in the atmosphere, due largely to emissions from plating plants, fumigation, and other special operations (Towill et al. 1978). Under normal conditions cyanide has relatively low persistence in air, usually between 30 days and 1 year (Way 1981), although some atmospheric HCN may persist for up to 11 years (Marrs and Ballantyne 1987). Data are lacking on the distribution and transformation of cyanide in the atmosphere (Towill et al. 1978) and should be acquired.

Lethal and Sublethal Effects

Terrestrial Flora and Invertebrates

Bacteria exposed to cyanide may exhibit decreased growth, altered cell morphology, decreased motility, mutagenicity, and altered respiration (Towill et al. 1978). Mixed microbial populations capable of metabolizing cyanide and not previously exposed to cyanide were adversely affected at 0.3 mg HCN/kg; however, these populations can become acclimatized to cyanide and can then degrade wastes with higher cyanide concentrations (Towill et al. 1978). Acclimatized populations in activated sewage sludge can often completely convert nitriles to ammonia, sometimes at concentrations as high as 60 mg total cyanides per kilogram (Towill et al. 1978). Cyanide can be degraded by various pathways to yield a variety of products, including carbon dioxide, ammonia, beta-cyanoalanine, and formamide (Knowles 1988). Several species of fungi can accumulate and metabolize cyanide, but the products of cyanide metabolism vary. For example, carbon dioxide and ammonia are formed as end products by *Fusarium solani*, whereas alpha-amino butyronitrile is a major cyanide metabolite of *Rhizoctonia solani*(Towill et al. 1978). Significant amounts of cyanide are formed as secondary metabolites by many species of fungi and some bacteria by decarboxylation of glycine (Knowles 1988). Rhizobacteria may suppress plant growth in soil through cyanide production. In one case volatile metabolites--including cyanide--from fluorescent pseudomonad soil bacteria prevented root growth in seedlings of lettuce, *Lactuca sativa* (Alstrom and Burns 1989). Not all cyanogenic isolates inhibited plant growth. Some strains promoted growth in lettuce and beans by 41-64% in 4 weeks versus 49-53% growth reduction by inhibitory strains (Alstrom and Burns 1989).

In higher plants, elevated cyanide concentrations inhibited respiration (through iron complexation in cytochrome oxidase) and ATP production and other processes dependent on ATP, such as ion uptake and phloem translocation, eventually leading to death (Towill et al. 1978). Cyanide produces chromosomal aberrations in some plants, but the mode of action is unknown (Towill et al. 1978). At lower concentrations, effects include inhibition of germination and growth, but cyanide sometimes enhances seed germination by stimulating the pentose phosphate pathway and inhibiting catalase (Towill et al. 1978; Solomonson 1981). The detoxification mechanism of cyanide is mediated by rhodanese. This enzyme is widely distributed in plants (Solomonson 1981; Leduc 1984). The rate of production and release of cyanide by plants to the environment through death and decomposition is unknown (Towill et al. 1978).

Free cyanide is not found in intact plant cells. Many species of plants, such as cassava, sorghum, flax, cherries, almonds, and beans, contain cyanogenic glycosides that release HCN when hydrolyzed (Towill et al. 1978). Cyanide poisoning of livestock by forage sorghums, such as Sudan grass and various hybrid cultivars, is well known (Cade and Rubira 1982) and has led to the development of several variations of sorghums that have a reduced capability of producing cyanide poisoning (Egekeze and Oehme 1980). Cyanogenesis has an

important role in plant defense against predatory herbivores. This herbivore-plant interaction is not simple; the degree of selectivity by herbivores varies among individuals and by differences in hunger and previous diet (Jones 1988).

Cyanide metabolism in higher plants involves amino acids, N-hydroxyamino acids, aldoximes, nitriles, and cyanohydrins (Halkier et al. 1988). Cyanide is a coproduct of ethylene synthesis in higher plants. The increase in ethylene production that occurs during the senescence of certain flowers and the ripening of fruits is accompanied by a rise in beta-cyanoalanine activity; activity of this enzyme correlates closely with that of ACC (1-aminocyclopropane-l-carboxylic acid) oxidase, the last enzyme in the ethylene pathway. Manning (1988) suggested that ACC oxidase reacts with various amino acids to liberate cyanide. Cyanide added to isolated castorbean (*Ricinus communis*) mitochondria significantly enhanced the rate and amount of protein synthesis. Cyanide stimulated mitochondrial protein synthesis in a dose-dependent manner, with an optimal stimulation of over 2 times at 26 µg/L, but at this concentration mitochondrial respiration was inhibited by 90% (Kaderbhai et al. 1989). Cyanide is a weak competitive inhibitor of green bean *(Phaseolus vul*garis) lipoxygenase, an enzyme that catalyzes the formation of hydroperoxides from polyunsaturated fatty acids (Adams 1989). Because degradation of hydroperoxides causes unacceptable changes in bean flavor and color, compounds that inhibit lipoxygenase may enjoy wide application in the frozen vegetable industry (Adams 1989). Corn seedlings from cold-resistant cultivars were more resistant to 65 mg KCN/L at low temperatures (13°C) than were seedlings from cold-susceptible cultivars (25°C), as judged by respiratory activity of mitochondria (Van De Venter 1985). Results suggest that cyanide-resistant respiration may play a role in cold resistance in maize seedlings, although more evidence is needed to demonstrate that cold-resistant plants actually use their greater potential for alternative respiration at low temperatures (Van De Venter 1985).

The cyanogenic system comprising cyanogenic glycosides, cyanohydrins, betaglucosidases, and nitrile lyases is widespread in plants, but also occurs in several species of arthropods, including the tiger beetle (*Megacephala virginica*), leaf beetle (*Paropsis atomaria*), zygaenid moths, and certain butterflies (Nahrstedt 1988). In *Zygaena trifolii*, cyanide compounds seem to function as protection against predators (Nahrstedt 1988). Defensive secretions of cyanide have also been reported in polydesmid millipedes, and these organisms seem to be more tolerant than other species when placed in killing jars containing HCN (Towill et al. 1978). In a millipede (*Apheloria* sp.), cyanide is generated in a two-compartment organ by hydrolysis of mandelonitrile; cyanide generation occurs outside the gland when the components of the two compartments are mixed during ejection (Towill et al. 1978).

Highly toxic substances, such as cyanides, are sometimes feeding cues and stimulants for specialized insects. For example, instar larvae of the southern armyworm (*Spodoptera eridania*) strongly prefer cyanogenic foods, such as foliage of the lima bean, a plant with comparatively elevated cyanide content--up to 31 mg/kg in some varieties--in the form of linamurin (Brattsten et al. 1983). Feeding was stimulated in southern armyworms at dietary levels up to 508 mg KCN/kg (208 mg HCN/kg) for first to fourth instar larval stages, and between 1,000 and 10,000 mg KCN/kg diet for fifth and sixth instar larvae (Brattsten et al. 1983). Sixth instar larvae preexposed to diets containing 5,000 mg KCN/kg showed no adverse affects at dietary levels of 10,000 mg KCN/kg; however, previously unexposed larvae showed reversible signs of poisoning at 10,000 mg/kg diet, including complete inhibition of oviposition and 83% reduction in adult emergence (Brattsten et al. 1983). Experimental studies with southern armyworm larvae and thiocyanate--one of the in vivo cyanide metabolites--showed that 5,000 mg thiocyanate per kilogram diet reduced pupation by 77%, completely inhibited oviposition, and reduced adult emergence by 80% (Brattsten et al. 1983), strongly suggesting that thiocyanate poisoning is the primary effect of high dietary cyanide levels in southern armyworms.

Resistant species, such as southern armyworms, require injected doses up to 800 mg KCN/kg BW (332 mg HCN/kg BW) or diets of 3,600 mg KCN/kg for 50% mortality (Brattsten et al. 1983), but data are scarce for other terrestrial invertebrates. Exposure to 8 mg HCN/L air inhibits respiration in the granary weevil (*Sitophilus granarius*) within 15 min and kills 50% in 4 h; some weevils recover after cessation of 4-h exposure (Towill et al. 1978).

Aquatic Organisms

Numerous accidental spills of sodium cyanide or potassium cyanide into rivers and streams have resulted in massive kills of fishes, amphibians, aquatic insects, and aquatic vegetation; sources of poisonings were storage reservoirs of concentrated solutions, overturned rail tank cars, or discharge of substances generating free HCN

in the water from hydrolysis or decomposition (Leduc 1984). Data on the recovery of poisoned ecosystems are scarce. In one case, a large amount of cyanide-containing slag entered a stream from the reservoir of a Japanese gold mine as a result of an earthquake (Yasuno et al. 1981). The slag covered the streambed for about 10 km from the point of rupture, killing all stream biota; cyanide was detected in the water column for only 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).

Fish were the most sensitive aquatic organisms tested under controlled conditions. Significant adverse nonlethal effects, including reduced swimming performance and inhibited reproduction, were observed in the range of 5.0-7.2 µg free cyanide per liter; deaths were recorded for most species between 20 and 76 µg/L (Table 3). Among invertebrates, adverse nonlethal effects were documented between 18 and 43 µg/L, and lethal effects between 30 and 100 µg/L--although some deaths were recorded in the range 3-7 µg/L for the amphipod *Gammarus pulex* (Table 3). Algae and macrophytes were comparatively tolerant; adverse effects were reported at >160 µg free cyanide per liter (Table 3).

Table 3. Cyanide effects on selected species of aquatic organisms. All concentrations are shown as micrograms of hydrogen cyanide per liter (ppb) of medium at start unless indicated otherwise.

Species, concentration, and other variables	Effects	Reference[a]
Algae and macrophytes		
Alga, *Chlorella* sp.		
7,300	Inhibition of photosynthesis	3
30,000	Enzyme inhibition	2
Water hyacinth, *Eichhornia crassipes*		
300,000	Nonphytotoxic in 72 h; plants contained total cyanide of 6.7 g/kg dry weight (DW), equivalent to bioconcentration factor (BCF) of x22	5
Freshwater aquatic plants, nine species, 65,000, 30-min exposure	No effect on respiratory oxygen uptake in six species of angiosperms (*Myriophyllum* sp., *Potamogeton* spp., *Elodea* sp., *Ruppia* sp., *Cabomba* sp.); some effect on two species of bryophytes (*Rhynchostegium riparioides*, *Fontinalis antipyretica*) and one species of alga (*Cladophora glomerata*)	4
Alga, *Microcystis aeruginosa*		
7,990	90% kill	2
Alga, *Prototheca zopfi*		
3,000	Inhibition of respiration	2
Alga, *Scenedesmus quadricauda*		
160, as CN^-	Toxic	1
Invertebrates		
Copepod, *Acartia clausi*		
30	LC50 (96 h)	2
Isopod, *Asellus communis*		
29–40	MATC[b]	2, 8
1,834	LC50 (11 days)	
Oyster, *Crassostrea* sp.		
150	Motor activity suppressed after 10 min	2

Daphnid, *Daphnia magna*		
160	LC50 (96 h)	10
Daphnid, *Daphnia pulex*		
83	LC50 (96 h)	2
Amphipod, *Gammarus pseudolimnaeus*		
16–21	MATC[b]	8
58	LC50 (96 h) at 20º C	8
184	LC50 (96 h) at 5.2º C	8
Amphipod, *Gammarus pulex*		
3	LC50 (15 h); 50% dead in 14 days after exposure for 5 h	6
7.5	LC50 (9h); exposure for 66 min results in 50% mortality 14 days after exposure	6
15	LC50 (6 h); exposure for 45 min causes 50% mortality 14 days after exposure	6
75	LC50 (3 h); exposure for 18 min results in 50% kill 14 days after exposure	6
Mussel, *Mytilus edulis*		
18	After exposure for 14 days growth was reduced and uptake of glycine was inhibited	9
100	LC20 (14 days)	9
Mysid shrimp, *Mysidopsis bahia*		
11, 20, 43, or 70	Life-cycle (29 days) exposure produced adverse effects on survival at 70 µg/L, and on reproduction at 43 µg/L; no measurable effects at lower doses of 11 and 20 µg/L	7
93–113	LC50 (96 h)	2, 7
Snail, *Physa heterostropha*		
432	LC50 (96 h)	3, 10
Fiddler crab, *Uca tangeri*		
Isolated perfused gills subjected to 26,000 CN^-/L, as KCN	Inhibited sodium chloride absorption across gill epithelium; effect reversible if exposure <5 min and nonreversible if >30 min. Salt absorption effect regulated by $(Na^+ + K^+)$ ATPase	11
Fish		
Brown bullhead, *Ictalurus nebulosus*		
Subjected to steadily increasing concentration of waterborne cyanide over a 9-h period: 200 at 1 h, 600 at 3 h, 1,000 at 5 h, and 1,800 at 9 h	Increased heart beat rate at lower concentrations and decreased rate at higher concentrations; hyperventilation in first 3 h followed by decrease in ventilation rate; oxygen consumption paralleled changes in heart and ventilatory rates. Death in 9 h	21
Longnose gar, *Lepisosteus osseus*		
12 µg CN^-/kg BW, as sodium cyanide, equivalent to 10.7 µg CN or 20 µg NaCN, single injection	Hypoxic response and bradycardia; effects appear earlier when administered into the ventral aorta or conus than into the dorsal aorta	27
Bluegill, *Lepomis macrochirus*		
5.0	Inhibited spawning following chronic exposure	22
5.2	Complete inhibition of spawning after exposure for 57–289 days	2, 8
9.3–19.8	MATC[b]	2

19.4	Reduced survival of fry in 57-day exposure which began with eggs	8
50	Tolerated concentration at higher temperatures, but no reproduction	8
56–227	LC50 (96 h) for juveniles	8, 22
109–218	LC50 (96 h) for fry	8
232–365	LC50 (96 h) for eggs	22
535–690	LC50 (96 h) for eggs at hatching	8
Largemouth bass, *Micropterus salmoides*		
101	LC50 (96 h) for juveniles	8
Cutthroat trout, *Oncorhynchus clarki*		
1,000 for 20 min	All fish recovered within 12 min and fed and grew normally during the next 6 months	31
Coho salmon, *Oncorhynchus kisutch*		
7.0	Reduction of 50% in swimming performance during 8-day exposure	13
10	Swimming speed reduced after exposure for 2 h	2
Rainbow trout, *Oncorhynchus mykiss*		
0.1 or 1.0	No effect on sperm motility or on fertilization rate at lower dose; some effect on sperm motility at higher dose	12
5.0	Reduction of 50% in swimming performance in 20-day exposure	13
10	No effect on growth during 20-day exposure at 6º C	13
10	Increased respiration rate in 4 days, growth reduction and liver damage in 9 days, abnormal oocyte development and reduced spermatogonia production in 18–20 days	2
10, 20, or 30 for 7 days, sexually mature females	Exposure to 10 or 20 µg/L caused a reduction in serum calcium to levels insufficient for the production of exogenous yolk; this was not observed in the 30 µg/L group	14
10, 20, or 30 for 18 days, juveniles	Degenerative necrosis of liver hepatocytes at all concentrations in a dose-dependent pattern. Severe initial growth repression at all concentrations followed by a significant increase, but growth remained depressed 40% and 95% in the 20 and 30 µg/L groups, respectively, at 18 days	15
10 or 20, exposure for 20 days during midsummer, sexually maturing females	Both concentrations resulted in abnormal oocytes, delayed development, and significantly reduced the number of eggs for spawning	16
15	No effect on growth during 20-day exposure at 12º C	13
18	No deaths in 96 h at 6º C	13
20	65% reduction in weight gain after 21 days	2
28	LC50 (96 h) at 6º C	10, 13, 17
30	No effect on growth during 20-day exposure at 18º C	13
32	No deaths in 96 h at 12º C	13
42	LC50 (96 h) at 12º C	13
43	LC50 (96 h) for nonexercised juveniles	18

	during winter	
46–75	LC50 (96 h) for juveniles	8, 19
52	LC50 (96 h) for exercised juveniles during winter	18
60	No deaths in 96 h at 18º C	13
68	LC50 (96 h) at 18º C	10, 13, 17
96	LC50 (144 h)	20
Subjected to steadily increasing concentrations of waterborne cyanide: 0 at start, 200 at 1 h, 600 at 3 h, 1,000 at 5 h, and 1,800 at 9 h	Reduction in heart rate, hyperventilation, increased oxygen consumption, death in 9 h	21
Chinook salmon, *Oncorhynchus tshawytscha*		
10	After 64 days, increased growth rate and production when compared to controls	13
20	Growth reduced 27% after exposure for 64 days	2
Yellow perch, *Perca flavescens*		
76–108	LC50 (96 h) for juveniles	8, 22
288–>389	LC50 (96 h) for eggs and fry	8, 22
Fathead minnow, *Pimephales promelas*		
12.9–19.6	MATC[b]	8, 22
18–58	Reduction in RNA content in larva in 96 h at 18–36 µg HCN/L, and in DNA and protein at 18–58 µg/L	28
19	Egg reduction of 59% after exposure for 265 days at 25º C	13
35	Reduction in growth rate during chronic exposure	5
44	Hatching reduced 83% after chronic exposure	13
47	Growth reduction in 30 days	28
58	Toxicosis occurred in yolk-sac larvae within 24 h as judged by significant reductions in content of RNA and protein; however, effects were not measurable in 96 h suggesting development of partial tolerance	29
>61	Adverse effects on growth and survival during lifetime exposure	13
82–113	LC50 (96 h) for fry at 25º C	8
83–137	LC50 (96 h) for juveniles	8, 22
99	LC50 (96 h) for fry at 20º C	8
107	Reduced survival in 96 h	28
121–202	LC50 (96 h) for eggs at 25º C	8
121–352	LC50 (96 h) for eggs; more toxic at low dissolved oxygen	22
122	LC50 (96 h) for fry at 15º C	8
Mixture of NaCN plus $CdSO_4$, equivalent to 170 µg CN/L	LC50 (96 h) for adults	30
Mixture of NaCN plus $ZnSO_4$, equivalent to 180 µg CN/L	LC50 (96 h) for adults	30
230, as NaCN	LC50 (96 h) for adults	30
273	LC50 (96 h) for eggs at 20º C	8
352	LC50 (96 h) for eggs at 15º C	8
Mixture of NaCN plus $NiSO_4$, equivalent to 650 µg CN/L	LC50 (96 h) for adults	8
Black crappie, *Pomoxis nigromaculatus*		
60	Some deaths in <24 h	3

101	LC50 (96 h) for juveniles	8
Plainfin midshipman, *Porichthys notatus*		
Isolated photophores exposed to 2,600, as KCN	Maximal luminescence induced by KCN; effect inhibited by d-glucose, d-glyceraldehyde 3-phosphate, and 3-phosphoglycerate	32
Atlantic salmon, *Salmo salar*		
5.0 for 12 days, adult females	Decline in plasma and gonad vitellogenin levels	23
10	Abnormal embryonic development after 58-day exposure	2
10, 80, or 100; newly fertilized eggs continually exposed for 5 months to end of sac-fry stage	Hatching delayed 6–9 days at 80 and 100 µg/L. Hatching success reduced 15% to 40% at all test concentrations, but no measurable effects on growth or survival after hatching. Abnormalities (mostly defects of eye, mouth, vertebral column) were 6% at 10 µg/L, and 19% at 100 µg/L	24
24	LC50 (24 h) at dissolved oxygen of 3.5 mg/L	25
73	LC50 (24 h) at dissolved oxygen of 10 mg/L	25
5,000, 10,000, 25,000, 50,000, or 125,000 for 30 min	Total cyanide residues in gills ranged from 1.0 to 6.6 mg/kg fresh weight (FW) in a dose dependent manner	26
50,000 for 10, 15, 20, 25, or 30 min	Residues in gills, in mg total CN/kg FW, ranged from 1.3 (10 and 15 min) to 1.9 (15 and 20 min) to 4.5 (30 min)	26
Brown trout, *Salmo trutta*		
90	LC50 (96 h)	10
5,000, 10,000, 25,000, 50,000, 75,000, or 100,000, as CN- for 30 min	Residues in gills ranged in a dose-dependent manner from 0.6 mg CN/kg FW in the 5 mg/L group to 3.4 mg/kg FW in the 100 mg/L group	26
50,000 for 10, 15, 20, or 25 min	Residues in tissues, in mg/kg FW, ranged from 0.7 to 1.8 in gill, 0.6 to 2.3 in brain, and 1.3 to 2.5 in liver; concentrations were directly related to length of exposure	26
Brook trout, *Salvelinus fontinalis*		
5.0	Reduction of 50% in swimming performance in 29-day exposure	13
5.7–11.2	MATC[b]	8, 22
10	75% reduction in swimming endurance after exposure for 26 min	2
10–50	Swimming ability reduced 98% after exposure for 29 days	20
11	Continuous exposure of mature females for 144 days before spawning resulted in 50% reduction in number of eggs produced and 15% reduction in egg viability; however, 90 days after hatch trout were 18% heavier and 10% longer than controls	13
25	Inhibited oxygen intake after 5 h	2
33	Adverse effects on juvenile growth rate during exposure for 90 days	2, 8
56–112	LC50 (96 h) range for swimup fry and juveniles	8, 22
108–518	LC50 (96 h) for sac-fry	8, 22
>212	LC50 (96 h) for eggs	8, 22

[a]1, Towill et al. 1978; 2, EPA 1980; 3, EPA 1973; 4, Azcon-Bieto et al. 1987; 5, Low and Lee 1981; 6, Abel and Garner 1986; 7, Lussier et al. 1985; 8, Smith et al. 1979; 9, Thompson 1984; 10, Leduc et al. 1982; 11, Drews and Graszynski 1987; 12, Billard and Roubaud 1985; 13, Leduc 1984; 14, Da Costa and Ruby 1984; 15, Dixon and Leduc 1981; 16, Lesniak and Ruby 1982; 17, Kovacs and Leduc 1982b; 18, McGeachy and Leduc 1988; 19, Marking et al. 1984; 20, Ballantyne 1987a; 21, Sawyer and Heath 1988; 22, Smith et al. 1978; 23, Ruby et al. 1987; 24, Leduc 1978; 25, Alabaster et al. 1983; 26, Holden and Marsden 1964; 27, Smatresk et al. 1986; 28, Barron and Adelman 1984; 29, Barron and Adelman 1985; 30, Doudoroff 1956; 31, Wiley 1984; 32, Rees and Baguet 1989.

[b]Maximum acceptable toxicant concentration. Lower value in each pair indicates highest concentration tested producing no measurable effect on growth, survival, reproduction, or metabolism during chronic exposure; higher value indicates lowest concentration tested producing a measurable effect.

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 (EPA 1980). Water hyacinth *(Eichhornia crassipes)* can survive for at least 72 h in nutrient solution containing up to 300 mg CN/L and can accumulate up to 6.7 g/kg dry weight (DW) plant material. On this basis, 1 ha of water hyacinths has the potential to absorb 56.8 kg of cyanide in 72 h, and this property may be useful in reducing the level of CN in untreated wastewater from various electroplating factories, where concentrations generally exceed 200 mg CN/L (Low and Lee 1981). Cyanide may also affect plant community structure. Some algae, for example, metabolized CN at water concentrations <1 mg/L, but at concentrations of 1-10 mg/L, algal activity was inhibited, leaving a biota dominated by *Actinomycetes*--a filamentous bacterium (Knocke 1981).

Cyanide adversely affects fish reproduction by reducing 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 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 to as little as 10 µg HCN/L for 12 days during the onset of the reproductive cycle caused a reduction in plasma vitellogenin levels and a reduction in ovary weight. The loss of vitellogenin in the plasma would remove a major source of yolk (Ruby et al. 1986). Reproductive impairment in adult bluegills (*Lepomis macrochirus*) has been reported following exposure to 5.2 µg CN/L for 289 days (EPA 1980). Fertilized fish eggs are usually resistant to cyanide prior to blastula formation, but delayed effects occur at 60 to 100 µg HCN/L, including birth defects and reduced survival of embryos and newly hatched larvae (Leduc et al. 1982). Concentrations as low as 10 µg HCN/L caused developmental abnormalities in embryos of Atlantic salmon 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).

Other adverse effects of cyanide on fish include delayed mortality, pathology, impaired swimming ability and relative performance, susceptibility to predation, disrupted respiration, osmoregulatory disturbances, and altered growth patterns. Free cyanide concentrations between 50 and 200 µg/L were fatal to the more-sensitive fish species over time, and concentrations >200 µg/L were rapidly lethal to most species of fish (EPA 1980). Cyanide- induced pathology in fish includes subcutaneous hemorrhaging, liver necrosis, and hepatic damage. Exposure of fish for 9 days to 10 µg HCN/L 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 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 can rapidly and irreversibly impair the swimming ability of salmoneds in well-aerated water (Doudoroff 1976). Osmoregulatory disturbances recorded at 10 µg HCN/L may affect migratory patterns, feeding, and predator avoidance (Leduc et al. 1982; Leduc 1984). In general, fish experience a significant reduction in relative performance (based on osmoregulation, growth, swimming, and spermatogenesis) at 10 µg HCN/L, and although fish can survive indefinitely at 30 µg HCN/L in the laboratory, the different physiological requirements necessary to survive in nature could not be met (Leduc 1978, 1981; Leduc et al. 1982; Figure). Increased predation by green sunfish (*Lepomis cyanellus*) on fathead minnows *(Pimephales promelas)* was noted at sublethal concentrations of HCN, but it was uncertain if fatheads became easier prey or if green sunfish had greater appetites (Smith et al. 1979).

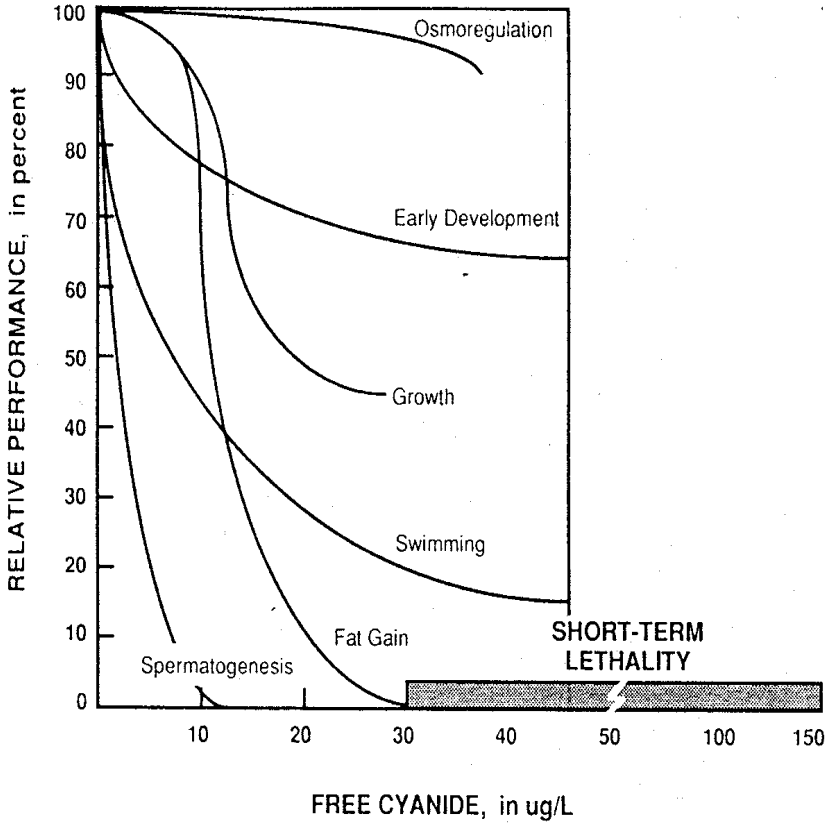

Figure. Summary of lethal and sublethal effects of free cyanide on freshwater fish. Modified from Leduc et al. (1982).

Sodium cyanide has stimulatory effects on oxygen-sensitive receptors in lungfish, amphibians, reptiles, birds, and mammals (Smatresk 1986). Facultative and aquatic air breathers appear to rely on air breathing when external chemoreceptors are stimulated, whereas obligate air-breathing fish are more responsive to internal stimuli (Smatresk 1986). Gill ventilation frequency of longnose gar (*Lepisosteus osseus*), for example, was little affected by external cyanide application, but responded strongly when cyanide was administered internally by injection (Smatresk 1986). Cyanide, like many other chemicals, can stimulate growth of fish during exposure to low sublethal levels. This phenomenon, referred to as hormesis, is little understood and warrants additional research (Leduc 1984).

The observed toxicity to aquatic life of simple and complex cyanides was attributed almost entirely to molecular (undissociated) HCN derived from ionization, dissociation, and photodecomposition of cyanide-containing compounds. The toxicity of the cyanide ion, CN^-, which is a minor component of free cyanide (HCN + CN^-) in waters that are not exceptionally alkaline is of little importance (Doudoroff 1976; Towill et al. 1978; Smith et al. 1979; EPA 1980). The acute toxicity of stable silver cyanide and cuprocyanide complex anions is much less than that of molecular HCN, but is nevertheless important; these ions can be the principal toxicants, even in some very dilute solutions. The much lower toxicities of the ferrocyanide and ferricyanide complexions--which are of high stability but subject to extensive and rapid photolysis, yielding free cyanide on direct exposure to sunlight--and the nickelocyanide ion complex are not likely to be of practical importance (Doudoroff 1976). Toxicity to aquatic organisms of organic cyanide compounds, such as lactonitrile, is similar to that of inorganic cyanides because they usually undergo rapid hydrolysis in water to free cyanide (Towill et al. 1978). There is general agreement that total cyanide concentrations in water in most cases will overestimate the actual cyanide toxicity to aquatic organisms, and that the analytically determined HCN concentration in cyanide-polluted waters

is considered to be the most reliable index of toxicity (Doudoroff 1976; Smith et al. 1979; EPA 1980; Abel and Garner 1986).

Cyanide acts rapidly in aquatic environments, does not persist for extended periods, and is highly species selective; organisms usually recover quickly on removal to clean water. The critical sites for cyanide toxicity in freshwater organisms include the gills, egg capsules, and other sites where gaseous exchange and osmoregulatory processes occur. On passing through a semipermeable membrane, the HCN molecules are usually distributed by way of the circulatory system to various receptor sites where toxic action or detoxification occurs (Leduc 1984). Once in the general circulation, cyanide rapidly inhibits the electron transport chain of vital organs. Signs of distress include increased ventilation, gulping for air at the surface, erratic swimming movements, muscular incoordination, convulsions, tremors, sinking to the bottom, and death with widely extended gill covers (Leduc 1981, 1984). The acute mode of action of HCN is limited to binding those porphyrins that contain Fe^{+3}, such as cytochrome oxidase, hydroperoxidases, and methemoglobin. At lethal levels, cyanide is primarily a respiratory poison and one of the most rapidly effective toxicants known (Leduc et al. 1982). The detoxification mechanism of cyanide is mediated by thiosulfate sulfur transferase, also known as rhodanese. This enzyme is widely distributed in animals, including fish liver, gills, and kidney. Rhodanese plays a key role in sulfur metabolism, and catalyzes the transfer of a sulfane-sulfur group to a thiophilic group (Leduc 1984). Thiosulfate administered in the water with cyanide reduced the toxicity of cyanide to fish, presumably by increasing the detoxification rate of cyanide to thiocyanate (Towill et al. 1978).

Additive or more-than-additive toxicity of free cyanide to aquatic fauna has been reported 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 nickelocyanide complex markedly reduces the toxicity of both cyanide and nickel at high concentrations in alkaline pH. At lower concentrations and acidic pH, solutions increase in toxicity by more than 1,000 times, owing to dissociation of the metallocyanide complex to form hydrogen cyanide (Towill et al. 1978). Mixtures of cyanide and ammonia may interfere 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 is influenced by water alkalinity and pH. Toxicity decreased with increasing alkalinity and pH from 0.42 mg CN/L at 5 mg $CaCO_3$/L and pH 6.5; to 1.4 mg CN/L at 70 mg $CaCO_3$/L and pH 7.5; to 730 mg CN/L at 192 mg $CaCO_3$/L and pH 8.0 (Doudoroff 1956).

Numerous biological and abiotic factors are known to modify the biocidal properties of free cyanide, including water pH, temperature, and oxygen content; life stage, condition, and species assayed; previous exposure to cyanide compounds; presence of other chemicals; and initial dose tested. There is general agreement that cyanide is more toxic to freshwater fish under conditions of low dissolved oxygen (Doudoroff 1976; Towill et al. 1978; Smith et al. 1979; EPA 1980; Leduc 1984); that pH levels within the range 6.8-8.3 had little effect on cyanide toxicity but enhanced toxicity at acidic pH (Smith et al. 1979; EPA 1980; Leduc et al. 1982; Leduc 1984); that juveniles and adults were the most sensitive life stages tested and embryos and sac fry the most resistant (Smith et al. 1978, 1979; EPA 1980; Leduc 1984); and that substantial interspecies variability exists in sensitivity to free cyanide (Smith et al. 1979; EPA 1980). Initial dose and water temperature both modify the biocidal properties of HCN to freshwater teleosts. At slowly lethal concentrations (i.e., < 10 µg HCN/L), cyanide was more toxic at lower temperatures; at high, rapidly lethal HCN concentrations, cyanide was more toxic at elevated temperatures (Kovacs and Leduc 1982a, 1982b; Leduc et al. 1982; Leduc 1984). By contrast, aquatic invertebrates were 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 resistance to cyanide correlated with higher activity induced by exercise and higher temperatures, suggesting a faster detoxification rate or higher oxidative and anaerobic metabolisms. Low levels of cyanide that were 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 low sublethal levels of cyanide through continuous exposure might 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 resistance only slightly to lethal concentrations (Alabaster et al. 1983). Juvenile rainbow trout previously exposed to low 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). Experimental evidence is lacking on exposure to lethal concentrations after prior exposure to high sublethal concentrations; some investigators predict decreased resistance (Leduc 1984), and others increased survival (Towill et al. 1978).

Birds

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-resistant species, such as domestic chickens, 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 15-30 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 was not related to body size but seemed to be associated with diet (Wiemeyer et al. 1986). Birds that feed predominantly on flesh, such as vultures, kestrels, and owls, were more sensitive to cyanide than were species that feed mainly on plant material--with the possible exception of mallard (Anas *platyrhynchos*)--as judged by acute oral LD50 values (Table 4).

Table 4. Cyanide effects on selected species of birds.

Species, dose, and other variables	Effects	Reference[a]
Mallard, *Anas platyrhynchos*		
Single oral dose of NaCN		
0.53 mg CN/kg body weight (BW), equivalent to 1 mg NaCN/kg BW	No deaths	7
1.1 mg CN/kg BW (2.0 mg NaCN/kg BW)	About 6% dead	7
1.27 mg CN/kg BW (2.4 mg NaCN/kg BW)	About 33% dead	7
1.43 mg mg CN/kg BW (2.7 mg NaCN/kg BW)	LD50; 95% confidence interval (C.I.) of 2.2 and 3.2 mg NaCN/kg bW	7
Turkey vulture, *Cathartes aura*		
Single oral dose of 19.1 mg CN/kg BW, equivalent to 36 mg NaCN/kg BW	Up to 80% of the cyanide in blood was present as free cyanide and the remainder as bound cyanide	1
Single oral dose of 19.1 mg CN/kg BW, equivalent to 36 mg NaCN/kg BW	Average time to death was about 19 min and ranged between 8 and 41 min; cyanide residues postmortem, in mg CN/kg fresh weight (FW), were 6.7 in blood (Max. 21) and 0.6 in liver (Max. 2.8)	2
Rock dove, *Columba livia*		
0.12 mg CN/L air, as HCN	All dead in 10 min	2
1.6 mg CN/kg BW, equivalent to 4.0 mg KCN/kg BW	Minimum lethal dose when administered intravenously or intramuscularly	2
Black vulture, *Coragyps atratus*		
Single oral dose, as NaCN		
1.6 mg CN/kg BW	No deaths in 60 min. Mean and maximum blood CN concentrations, in mg/kg FW, were 0.7 and 0.9, respectively	2
2.4 lmg CN/kg BW	Some deaths within 30 min. Mean blood CN residues in	2

	mg/kg FW, were 0.7 in dead birds vs. 1.2 in those surviving 60 min	
2.54 mg CN/kg BW	Acute oral LD50; 95% C.I. of 2.3 and 2.8 mg CN/kg BW (4.4–5.3 mg NaCN/kg BW)	2
3.7 and 19.1 mg CN/kg BW	All dead within 16 min; maximum blood CN levels postmortem were 2.1 mg/kg FW in the low dose group and 4.2 in the high dose group	2
Japanese quail, *Coturnix japonica*		
Single oral dose, as NaCN		
4.5 mg CN/kg BW	Acute oral LD50 for adult females; 95% C.I. of 3.1 and 6.5 mg CN/kg BW	2
5.5 mg CN/kg BW	Acute oral LD50 for adult males; 95% C.I. of 4.0 and 7.5 mg CN/kg BW	2
American kestrel, *Falco sparverius*		
2.12 mg CN/kg BW, as NaCN	Acute oral LD50; 95% C.I. of 1.6 and 2.8 mg CN/kg BW	2
Domestic chicken, *Gallus domesticus*		
Intravenous route		
0.01 µg/kg BW	Most of dose recovered in urine as thiocyanate in 6 h; excretion limited by availability of transferable sulfur	3
0.6 mg CN/kg BW, equivalent to 1.5 mg KCN/kg BW	Lethal	2
0.78 mg CN/kg BW, as KCN	Sublethal; thiocyanate excretion increased 10 times after 10 min and returned to normal levels after 3.5 h; the total thiocyanate collected was equivalent to 85% of the administered dose	4
1.3 mg CN/kg BW, as KCN	Lethal	4
Inhalation route		
0.12 mg HCN/L air	All survived for at least 60 min	2
Single oral dose, as NaCN		
3.2 mg CN/kg BW, equivalent to 6.0 mg NaCN/kg BW	No deaths in 30 min; maximum CN levels, in mg/kg FW, were 1.1 in blood and 0.06 in liver	2
6.4 mg CN/kg BW	Some deaths in 30 min; maximum CN levels, in mg/kg FW were 1.6 in blood and 0.12 in liver	2
11.1 mg CN/kg BW	Acute oral LD50; 95% C.I. of 6.4 and 19.1 mg CN/kg BW	2
25.4 mg CN/kg BW	Advanced signs of acute poisoning; death probable within 30 min; maximum CN levels, in mg/kg FW, were 1.5 in blood and 0.6 in liver	2
Dietary route		
Fed cassava diets containing 4, 37, 70,	At all dietary levels, there	5

or 103 mg total cyanide per kilogram ration to day-old chicks for 8 weeks	was no significant effect on survival, growth, histology, hemoglobin, hematocrit, or lymphocyte number; however, serum thiocyanate levels increased in a dose-dependent manner	
Fed diets containing 135 mg HCN/kg Chicks, 20-day exposure	Growth and food intake significantly depressed; plasma thiocyanate concentration increased	6
Adults, 14-day exposure	Urinary excretion of thiocyanate increased 5 times in laying hens	6
California condor, *Gymnogyps californianus*		
Juvenile (8.4 kg), found dead, presumably of cyanide poisoning	No evidence of injuries or disease; yellow fluorescent particles found in mouth appeared like those placed in NaCN ejector mechanisms used in predator control. However, blood cyanide concentration was similar to that found in nonexposed vultures, including two captive California condors	2
Eastern screech-owl, *Otus asio*		
4.6 mg CN/kg BW, equivalent to 8.6 mg NaCN/kg BW	Acute oral LD50; 95% C.I. of 3.8 and 5.4 mg CN/kg BW	2
Canary, *Serinus canarius*		
0.12 mg HCN/L air	All dead in 3 min	2
European starling, *Sturnus vulgaris*		
9.0 mg CN/kg BW, as NaCN	Acute oral LD50; 95% C.I. of 4.8 and 17 mg CN/kg BW	2
Andean condor, *Vultur gryphus*		
Single oral dose of 19.1 mg CN/kg BW (36 mg NaCN/kg BW)	Blood sampled immediately after death contained 1.2 mg free CN per liter and 0.5 mg bound CN per liter	1

[a]1, Krynitsky et al. 1986; 2, Wiemeyer et al. 1986; 3, Oh et al. 1987; 4, Davis 1981; 5, Gomez et al. 1988; 6, Elzubeir and Davis 1988b; 7, Personal communication, E. F. Hill, Patuxent Wildlife Research Center.

Many species of migratory birds 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 (>200 mg total cyanide per liter) waters (Clark and Hothem 1991). Migratory bird mortality from cyanide toxicosis may be eliminated at these facilities by screening birds from toxic solutions (Hallock 1990) or lowering the cyanide concentrations with hydrogen peroxide to <50 mg total cyanide per liter (Allen 1990), although the latter procedure requires verification (Clark and Hothem 1991).

Some birds may not die immediately after drinking lethal cyanide solutions. In Arizona, a 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.

Elevated cyanide concentrations were found in blood of chickens that died of cyanide poisoning; however, these concentrations overlapped those in survivors. Despite this variability, blood is considered more reliable than liver as an indicator of cyanide residues in exposed birds (Wiemeyer et al. 1986). No gross pathological changes in birds related to cyanide dosing were observed at necropsy (Wiemeyer et al. 1986), similar to other taxonomic groups tested.

Cyanide-nutrient interactions are reported for alanine, which appears to exacerbate cyanide toxicity, and for cystine, which seems to alleviate toxicity (Davis et al. 1988). Dietary cyanide--at levels that do not cause growth depression--alleviates selenium toxicity in chickens, but not the reverse (Davis et al. 1988; Elzubeir and Davis 1988a). For example, dietary selenium, as selenite, at 10 mg/kg for 24 days, reduced growth, food intake, and food utilization efficiency, and produced increased liver size and elevated selenium residues; the addition of 45 mg CN/kg diet (100 mg sodium nitroprusside per kilogram) eliminated all effects except elevated selenium residues in liver. The mechanism of alleviation is unknown and may involve a reduction of tissue selenium through selenocyanate formation, or increased elimination of excess selenium by increasing the amount of dimethyl selenide exhaled (Elzubeir and Davis 1988a). At dietary levels of 135 mg CN/kg plus 10 mg selenium per kilogram, chick growth was significantly decreased (Elzubeir and Davis 1988a). This interaction can be lost if there is a deficiency of certain micronutrients or an excess of vitamin K (Davis et al. 1988).

Mammals

Much of the toxicological interest in cyanide relating to mammals has focused on its rapid lethal action; however, its most widely distributed toxicologic problems are due to its toxicity from dietary, industrial, and environmental factors (Way 1981, 1984; Gee 1987; Marrs and Ballantyne 1987). Chronic exposure to cyanide is correlated with specific human diseases: Nigerian nutritional neuropathy, Leber's optical atrophy, retrobulbar neuritis, pernicious anemia, tobacco amblyopia, cretinism, and ataxic tropical neuropathy (Towill et al. 1978; Way 1981; Sprince et al. 1982; Berninger et al. 1989; Ukhun and Dibie 1989). The effects of chronic cyanide intoxication are confounded by various nutritional factors, such as dietary deficiencies of sulfur-containing amino acids, proteins, and water-soluble vitamins (Way 1981).

Most authorities now agree on five points: (1) cyanide has low persistence in the environment and is not accumulated or stored in any mammal studied; (2) cyanide biomagnification in food webs has not been reported, possibly due to rapid detoxification of sublethal doses by most species, and death at higher doses; (3) cyanide has an unusually low chronic toxicity, but chronic intoxication exists and, in some cases, can be incapacitating; (4) despite the high lethality of large single doses or acute respiratory exposures to high vapor concentrations of cyanide, repeated sublethal doses seldom result in cumulative adverse effects; and (5) cyanide, in substantial but sublethal intermittent doses can be tolerated by many species for long periods, perhaps indefinitely (Towill et al. 1978; EPA 1980; Way 1984; Ballantyne and Marrs 1987a; Table 5).

The toxicity of cyanogenic plants is a problem for both domestic and wild ungulates. Poisoning of herbivorous ungulates is more prevalent under drought conditions, when these mammals become less selective in their choice of forage; dry growing conditions also enhance cyanogenic glycoside accumulations in certain plants (Towill et al. 1978). Animals that eat rapidly are at greatest risk, and intakes of 4 mg HCN/kg BW can be lethal if consumed quickly (Egekeze and Oehme 1980). In general, cattle are most vulnerable to cyanogenic plants; sheep, horses, and pigs--in that order--are more resistant than cattle (Cade and Rubira 1982). Deer *(Odocoileus* sp.) and elk *(Cervus* sp.) have been observed to graze on forages that contain a high content of cyanogenic glycosides; however, cyanide poisoning has not been reported in these species (Towill et al. 1978).

Ruminant and nonruminant ungulate mammals that consume forage with high cyanogenic glycoside content, such as sorghums, Sudan grasses, and corn, may experience toxic signs due to microbes in the gut that hydrolyze the glycosides, releasing free hydrogen cyanide (Towill et al. 1978). 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 can occur quickly, depending on the dose administered (Towill et al. 1978; Cade and Rubira 1982). Thyroid dysfunction has been reported in sheep grazing on stargrass *(Cynodon plectostachyus),* a plant with high cyanogenic glycoside and low iodine content. Sheep developed enlarged thyroids and gave birth to

lambs that were stillborn or died shortly after birth (Towill et al. 1978). Cyanogenic foods can exacerbate selenium deficiency, as judged by the increased incidence of nutritional myopathy in lambs on low-selenium diets (Elzubeir and Davis 1988a). A secondary effect from ingesting cyanogenic glycosides from forage is sulfur deficiency as a result of sulfur mobilization to detoxify the cyanide to thiocyanate (Towill et al. 1978).

Cyanide poisonings of livestock by forage sorghums and other cyanogenic plants are well documented (Cade and Rubira 1982). Horses in the southwestern United States grazing on Sudan grass and sorghums developed posterior muscle incoordination, urinary incontinence, and spinal cord histopathology; offspring of mares that had eaten Sudan grass during early pregnancy developed musculoskeletal deformities (Towill et al. 1978). Salt licks containing sulfur (8.5%) have been used to treat sheep after they failed to gain weight when grazing on sorghum with high HCN content (Towill et al. 1978). Sugar gum (*Eucalyptus cladocalyx*) and manna gum (*Eucalyptus viminalis*) contain high levels of cyanogenic glycosides, and both have been implicated as the source of fatal HCN poisoning in domestic sheep and goats that had eaten leaves from branches felled for drought feeding, or after grazing sucker shoots on lopped stumps (Webber et al. 1984). In one case, 10 goats died and 10 others were in distress within 2 h after eating leaves from a felled sugar gum. Dead goats had bright red blood that failed to clot and subepicardial petechial hemorrhages. Rumens of dead goats contained leaves of *Eucalyptus* spp. and smelled of bitter almonds. The 10 survivors were treated intravenously with 3 mL of a 1-L solution made to contain 20 g of sodium nitrite and 50 g of sodium thiosulphate; four recovered and six died. Of 50 afflicted goats, 24 died within 24 h and the remainder recovered (Webber et al. 1984). In rare instances HCN poisoning occurs when animals are exposed to chemicals used for fumigation or as a fertilizer (Webber et al. 1984), but there is general agreement that ingestion of plants containing high levels of cyanogenic glycosides is the most frequent cause of cyanide poisoning in livestock.

Cassava, also known as maniac, tapioca, yuca, or guacamate, is one of the very few--and, by far, the most important--food crops in which the cyanide content creates toxic problems (Cooke and Coursey 1981). Cassava is a major energy source for people and livestock in many parts of the world; it accounts for an average of 40% of the human caloric intake in Africa (Casadei et al. 1984), to more than 70% in some African diets (Way 1984). In comparison to other tropical crops it produces the highest yield per hectare (Okeke et al. 1985). Cassava is native to tropical America from southern Mexico to northern Argentina and probably has been under cultivation there for 4,000-5,000 years. It has been introduced to east Africa, Indian Ocean islands, southern India, and the Far East (Cooke and Coursey 1981). The global production of cassava roots was estimated at 50 million tons in 1950, and 100 million tons in 1980; about 44.2 million tons are grown annually in Africa, 32.7 million tons in tropical America, and 32.9 million tons in Asia (Cooke and Coursey 1981). Linamurin is the principal cyanogenic glycoside in cassava; its toxicity is due to hydrolysis by intestinal microflora releasing free cyanide (Padmaja and Panikkar 1989). Rabbits *(Oryctolagus cuniculus)* fed 1.43 mg linamurin per kilogram BW daily (10 mg/kg BW weekly) for 24 weeks showed effects similar to those of rabbits fed 0.3 mg KCN/kg BW weekly. Specific effects produced by linamurin and KCN included elevated lactic acid in heart, brain, and liver; reduced glycogen in liver and brain; and marked depletion in brain phospholipids (Padmaja and Panikkar 1989).

The use of cassava in animal feed presents two major problems: the presence of cyanogenic glycosides in the tuber, and the remarkably low protein levels in fresh and dried cassava. Pigs fed low-protein cassava diets for 8 weeks had reduced food consumption and lowered liver weight; addition of protein supplement to the diet reversed these trends (Tewe 1982b). Removal of cyanogenic glycosides from cassava tubers, mash, peels, and root meal is accomplished with several techniques. Usually, the cassava root is dried in the sun for several weeks, and this process removes most of the cyanogenic glycosides; however, under conditions of famine or food shortage, this process cannot be carried out properly (Cliff et al. 1984). Long fermentation periods, especially under conditions of high moisture content, may be effective in substantial detoxification of cassava mash (Ukhun and Dibie 1989). Cassava peels containing as much as 1,061 mg HCN/kg FW can be rendered suitable for feeding to livestock (4-625 mg/kg) by boiling for 7 min, roasting for 30 min, soaking for 15 h, or drying in the sun for 7.6 days (Okeke et al. 1985). Cassava root meal (up to 40% of cassava meal) is satisfactory as a diet supplement for domestic pigs, provided cyanide content is <100 mg/kg ration (Gomez et al. 1983).

Neuropathies associated with cassava ingestion (i.e., cyanide intoxication) can develop into a syndrome in humans and domestic animals, characterized by nerve deafness, optic atrophy, and an involvement of the sensory spinal nerve that produces ataxia. Other symptoms include stomatitis, glossitis, and scrotal dermatitis (Way 1981). Potentially more serious are long-term effects such as ataxic neuropathy, goiter, and cretinism,

which have been attributed to high cassava content in diets. Thiocyanate--one of the detoxification products--inhibits iodine absorption and promotes goiter, a common ailment in tropical countries (Cooke and Coursey 1981). At high dietary cyanide intakes there is an association with diabetes and cancer (Cliff et al. 1984), but this requires verification. The first case of cassava toxicity occurred almost 400 years ago (Cooke and Coursey 1981). The toxic principle was later identified as a cyanogenic glycoside, shown to be identical with flax linamurin (2-(beta-D-glucopyranosyloxy)-isobutyronitrile). All parts of the plant, except possibly the seeds, contain the glycoside together with the enzyme linamarase. This enzyme effects hydrolysis of the nitrile to free HCN when the tissue cellular structure is damaged (Cooke and Coursey 1981). Mantakassa disease is related to chronic cyanide intoxication associated with a diet consisting almost exclusively of cassava; in times of famine and sulfur-poor diets, Mantakassa effects were more pronounced (Casadei et al. 1984). Symptoms of Mantakassa disease include the sudden onset of difficulty in walking, increased knee and ankle reflexes, elevated serum thiocyanate levels, fever, pain, headache, slurred speech, dizziness, and vomiting. Women of reproductive age and children were the most seriously affected. Symptoms persisted for up to 4 months after treatment with hydroxycobalamin, vitamin supplements, and a high protein, energy-rich diet (Cliff et al. 1984). Mantakassa was reported in 1,102 victims in Mozambique in 1981 from a drought-stricken cassava staple area; from Zaire in 1928, 1932, 1937, and again in 1978-81; in Nigeria; and in the United Republic of Tanzania. The mean serum thiocyanate level in patients with Mantakassa is 2.6 times higher than in non-Mantakassa patients in Nigeria, and 3.5 times higher than in Tanzanian patients. Pesticides, infection, viruses, and consumption of food other than cassava were eliminated as possible causative agents in Mantakassa disease. Still unresolved is whether the disease is triggered when a threshold level of thiocyanate is reached, or when a critical combination of cyanide intoxication plus nutritional deficiency occurs (Cliff et al. 1984).

Routes of administration other than dietary ingestion should not be discounted. Livestock found dead near a cyanide disposal site had been drinking surface water runoff from the area that contained up to 365 mg HCN/L (EPA 1980). The use of cyanide fumigant powder formulations may be hazardous by contact of the powder with moist or abraded skin, contact with the eye, swallowing, and inhalation of evolved HCN (Ballantyne 1988). In rabbits, lethal systemic toxicity was produced by contamination of the eye, moist skin, or abraded skin (but not dry skin) with cyanide powder formulations (40% NaCN plus 60% kaolin) administered at 1-5 g powder per cubic meter (Ballantyne 1988). Hydrogen cyanide in the liquid state can readily penetrate the skin, and 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 (EPA 1980).

Use of poisons in livestock collars is both specific and selective for animals causing depredations, as is the case for cyanide collars to protect sheep against coyotes (Sterner 1979; Table 5). These collars contain a 33% NaCN solution and are usually effective against coyotes. However, field results indicate that some coyotes kill by means other than neck attack, and some exhibit great wariness in attacking collared sheep (Savarie and Sterner 1979).

Calcium cyanide in flake form was used in the 1920's to kill black-tailed prairie dogs and pocket gophers *(Geomys bursarius)* in Kansas, and various other species of rodents in Nova Scotia (Wade 1924). For prairie dog control, the usual practice was to place 43-56 g of calcium cyanide 0.3-0.7 m below the rim of the burrow and to close the entrances. The moisture in the air liberated HCN gas, which remained in the burrow for several hours, producing 100% kill. A lower dose of 28 g per burrow was about 90% effective (Wade 1924). Control of prairie dogs with cyanide sometimes resulted in the death of burrowing owls that lived in the prairie dog burrows (Wade 1924).

Clinical signs of acute cyanide poisoning in mammals last only a few minutes after ingestion and include rapid and labored breathing, ataxia, cardiac irregularities, dilated pupils, convulsions, coma, respiratory failure, and rapid death (Egekeze and Oehme 1980; Ballantyne 1983). Cyanide poisoning causes cardiovascular changes as well as its better known effects on cellular respiration. Cyanide increases cerebral blood flow in rabbits and cats, and disrupts systemic arterial pressure in dogs (Robinson et al. 1985). Cyanide affects mammalian behavior, mostly motor functions, although these effects have not been quantified. Cyanide-induced motor alterations observed in rats and guinea pigs include muscular incoordination, increased whole-body locomotion, disrupted swimming performance, and altered conditioned avoidance responses (D'Mello 1987). As a consequence of the cytotoxic hypoxia in acute cyanide poisoning, there is a shift from aerobic to

anaerobic metabolism, and the development of lactate acidosis. A combination of rapid breathing, convulsions, and lactate acidosis is strongly suggestive of acute cyanide poisoning (Ballantyne 1983). As with other chemical asphyxiants, the critical organs that are most sensitive to oxygen depletion are the brain and heart (Egekeze and Oehme 1980). The only consistent postmortem changes found in animals poisoned by cyanide are those relating to oxygenation of the blood. Because oxygen cannot be utilized, venous blood has a bright-red color and is slow to clot (Egekeze and Oehme 1980). Bright-red venous blood is not a reliable indicator of cause of death, however, because it is also associated with chemicals other than cyanide (Ballantyne 1983).

Cyanide poisoning is associated with changes in various physiological and biochemical parameters. The earliest effect of cyanide intoxication in mice seems to be inhibition of hepatic rhodanese activity, due to either blockage by excess binding to the active site or to depletion of the sulfane-sulfur pool. These changes do not seem to occur in blood, where rhodanese functions at its maximal rate, thus preventing cyanide from reaching the target tissues and causing death (Buzaleh et al. 1989). Cyanide causes dose- and species-dependent responses on vascular smooth muscle; studies with isolated aortic strips indicate that rabbits are 80 times more sensitive than dogs or ferrets (*Mustela putorius*; Robinson et al. 1985). Rabbits killed with HCN had higher concentrations of cyanide in blood and other tissues and lower tissue cytochrome oxidase activities than did those killed with KCN (Ballantyne et al. 1972). Cyanide promotes dose- and calcium-dependent release of dopamine tissues in the domestic cat, and reductions in adenosine triphosphate (ATP) content of the carotid body (Obeso et al. 1989). Cyanide-induced hypoxia is believed to decrease ATP content of Type I glomus cells. The decrease in the phosphate transfer potential is a crucial step in the overall transduction process, that is, the activation of the transmitter release from Type I cells, with resultant release and activation of sensory nerve endings (Obeso et al. 1989). Studies with isolated heart of the domestic ferret demonstrate that cyanide affects intracellular ionic exchange of H^+, Na^+, and calcium (Fry et al. 1987); inhibits cardiac action potential (Elliott et al. 1989); and inhibits oxidative phosphorylation accompanied by an intracellular acidosis, a decrease in phosphocreatinine, and a rise in inorganic phosphate (Eisner et al. 1987). When oxidative phosphorylation is inhibited in cardiac muscle, there is a rapid decrease of developed force or pressure; most of the decrease of developed pressure produced by cyanide in ferret heart is not produced by intracellular acidosis, and may result from increased inorganic phosphate (Eisner et al. 1987). Observed changes in rat cerebral oxidative responses to cyanide may be due to redistribution of intracellular oxygen supply to mitochondria respiring in an oxygen-dependent manner or by branching effects within brain mitochondria (Lee et al. 1988). Hyperammonemia and the increase of neutral and aromatic amino acids may also be important in loss of consciousness induced by cyanide (Yamamoto 1989).

Table 5. Cyanide effects on selected species of mammals.

Species, dose, and other variables	Effects	Reference[a]
Cattle, *Bos* sp.		
Fed hybrid sorghum Sudan grass cross 988 at 15–20 kg per animal daily for 3–8 days	Of 180 cows, 21 were affected and 13 died; toxic cyanide levels were measured in fodder and in liver and ruminal contents of dead cows	44
Dog, *Canis familiaris*		
Administered doses up to 2 mg NaCN/kg body weight (BW), once or twice daily for 15 months	Acute toxic signs evident after each administration, but complete recovery within 30 min; no measurable adverse effects after 15 months	1
5.4 mg NaCN/kg BW, single subcutaneous injection	LD50	2
24 mg CN/kg BW, single oral or slow intravenous injection	Lethal; at time of respiratory arrest, blood plasma	3

route	concentration was 1 mg total CN per liter or about 0.4 mg free cyanide per liter	
Fed diets containing 150 mg NaCN/kg for 30 days	No measurable effect on food consumption, blood chemistry, behavior, or organ histology	1
Coyote, *Canis latrans*		
Single forced oral dose of NaCN, in mg/kg BW		
4	Immobilized in 13 min, but all survived for at least 30 days; some sacrificed after 30 min: NaCN residues in mg/kg fresh weight (FW) were 0.03 in blood and 0.9 in stomach	2
4.1 (2.1–8.3)	LD50	2
8	Immobilization in 9 min, death within 41 min	2
16, 32, or 64	All immobilized in less than 1 min and all died in less than 8 min. Maximum NaCN residues were 0.14 mg/L in blood and 13.0 mg/L FW in stomach	2
"Toxic" collars attached to neck of sheep and camouflaged with wool; each collar contained 50 mL of a 33% NaCN solution; toxic action commences when coyote attacks sheep and punctures collar; all coyotes tested were known to attack sheep in laboratory pens	Of three coyotes tested, one was immobilized in 1 min and died within 18 min; the other two coyotes recovered; the dead coyote had mouthed the collar for about 2 s; residues in mg NaCN/kg, were 0.26 in blood and <0.1 in stomach; the other two coyotes had mouthed the collar for 3–15 s and had NaCN levels, in mg/kg FW, of 0.014 and 0.029 in blood, and 0.6 and <0.1 in stomach	2
Toxic collar, as above; each coyote tested was known to have fatally attacked at least three domestic sheep within a 30-day period	Of the 12 coyotes that attacked the neck region of the sheep and punctured the collar, nine received lethal doses and became immobilized in 1–3 min and died 3–25 min later; the mean time to death was 11.6 min; one of the three sublethally dosed coyotes survived at last three successful attacks in which the collar was punctured, and two survived two attacks; in all cases, contact with NaCN	4

	produced shaking of the head, pawing at the mouth, rubbing the snout on the ground, and ataxia	
African giant rat, *Cricetomys gambianus*		
Weanlings fed diets for 16 weeks containing 0 mg HCN/kg (maize), 110 mg HCN/kg (cassava pulp), 150 mg HCN/kg (cassava tuber), or 597 mg HCN/kg (cassava peel)	Food consumption was similar in all diets; no pathology was observed in any organ of animals on all treatments; rats on maize and cassava pulp diets had significantly increased growth rate, feed efficiency, and protein efficiency; rats on cassava peel and tuber diets had significantly increased thiocyanate levels in serum, organs, and urine	5
Juveniles, age 10–14 weeks, fed cassava peel diets for 2 weeks containing 720 mg HCN/kg	Adverse effects on growth when cassava peel exceeds 7.8% of the ration	6
Weanlings fed 1,000 mg CN/kg diet, as KCN, for 12 weeks	Reduction in feed intake, reduced body weight, elevated thiocyante concentrations in serum (37.4 mg/L vs. 12.6), urine (341 mg/L vs. 25), liver (1.7 g/kg FW vs. 0.4), kidney (2.4 g/kg FW vs. 0.4), and spleen (2.1 g/kg FW vs. 0.3)	7
Humans, *Homo sapiens*		
Intentional oral ingestion of unknown amount of NaCN or KCN, three cases	Death between 5 and 30 min; stomach cyanide concentrations ranged between 100 and 164 mg; tissue residues postmortem in mg/kg FW, were 0.3–1.1 in blood, 0.3–1.0 in liver, and 0.2–0.3 in brain	8
Found dead, four cases, time to death unknown	Maximum cyanide concentration in stomach was 230 mg; maximum tissue residues, in mg/kg FW were 3.5 in blood, 6.3 in liver, and 0.5 in brain	8
Attempted suicide by 39-year-old-male, unknown amount of NaCN	Severe tremors and progressive loss of muscle tone--representing the first case of cyanide intoxication with delayed onset of symptoms	9
Inhalation of HCN gas, in mg/m^3, for various time intervals		

140 for 60 min	Calculated LC50	10
220 for 30 min	Calculated LC50	10
504 for 10 min	Calculated LC50	10
680 for 5 min	Calculated LC50	10
1,500 for 3 min	Calculated LC50	10
4,400 for 1 min	Calculated LC50	10
Inhalation of 2,000 mg HCN/L	First breath results in deep, rapid breathing, with collapse, convulsions, and death within 1 min	11
Inhalation of cyanogen chloride, in mg/L, for various time intervals		
1, 10 min	Irritant	1
48, 30 min	Fatal	1
159, 10 min	Fatal	1
Inhalation of cyanogen bromide, in mg/L, for various time intervals		
1.4, no time given	Irritant to eyes and nose	1
92, 10 min	Fatal	1
Single oral dose		
0.5–3.5 mg HCN/kg BW	Lethal	12, 41
0.7–3.5 mg KCN/kg BW, equivalent to 50 to 250 mg KCN/adult	Fatal	10
2 mg HCN/kg BW, or total of about 150 mg HCN	Acute LD50 for adults	13
1–5 g of NaCN or KCN, equivalent to 0.2 g/adult or 3 mg/kg BW	Minimum lethal dose	14
Tissue residues		
Whole blood, 1–2 mg free cyanide per liter	Usually lethal	42
Whole blood, 2.6–3.1 mg total CN per liter	Minimum cyanide concentration associated with death in an otherwise healthy individual	13
Whole blood, 2.6–3.1 mg total CN per liter	Minimum cyanide concentration associated with death in an otherwise healthy individual	13
Whole blood, 4–45 mg total CN per liter	Levels measured in known suicides	13
Whole body, 7 mg HCN/kg BW	Residue associated with minimum lethal dose	11
Daily dietary intake of 15–31.5 mg hydrogen cyanide from cassava	Mantakassa disease--see text for discussion	15
100 mg HCN/kg BW applied to skin surface	LD50	11
Clothing inundated with 10% NaCN solution, pH 11.4	Clinical signs of toxicity within 25 min and death in about 60 min	13
Livestock		
>200 mg HCN/kg plant materials in diet	Potentially dangerous	13

Cynomolgus monkeys, *Macaca* spp.		
Given multiple sublethal doses of KCN (5–18 mg) for 23 days	Brain histoapathology	3
Exposed to HCN gas produced from combustion of polyacrylonitrile materials at various temperatures		
300º C, 87–170 mg HCN/L air	Incapacitated in 16–30 min; blood cyanide of 4.3 mg/L	16
600º C, 120–174 mg HCN/L air	Incapacitated between 6 and 24 min, blood cyanide of 2.96 mg/L	16
900º C, 166–196 mg HCN/L air	Incapacitated between 2 and 13 min; blood cyanide concentration of 3.1 mg/L	16
Exposed to HCN gas at air concentrations of 60, 80, or 150 mg HCN/L for 30 min	At 60 mg/L, HCN had only a slight depressive effect on the central nervous system; at 80 and 150 mg/L, severe CNS depression and incapacitation occurred	17
Exposed to HCN gas at air concentrations of 100, 1092, 123, 147, or 156 mg HCN/L air	Incapacitated in 8 min at higher doses to 19 min at lowest dose tested; blood cyanide after 30 min exposure ranged between 1.7 mg/L at 100 mg HCN/L and 3.2 mg/L at 156 mg HCN/L; after recovery for 60 min, blood CN ranged between 2.0 and 2.9 mg/L	16
Domestic mouse, *Mus* spp.		
Single intraperitoneal injection		
HCN, 2.8 mg/kg BW	LD50	10
NaCN, 4.6–5.9 mg/kg BW	LD50	10
KCN, 5.3–6.7 mg/kg BW	LD50	10
Acetone cyanohydrin, $(CH_3)_2C(OH)CN$, 8.7 mg/kg BW	LD50 (7 days); first death in 5 min	18
Malonitrile, $NCCH_2CN$, 18 mg/kg BW	LD50 (7 days); first death in 4.8 h	18
Propionitrile, CH_3CH_2CN, 28 mg/kg BW	LD50 (7 days); first death in 21 h	18
N-butyronitrile, 38 mg/kg BW	LD50 (7 days); first death in 2.2 h	18
Acrylonitrile, CH_2CHCN, 46 mg/kg BW	LD50 (7 days); first death in 2.3 h	18
Succinonitrile, $NCCH_2CH_2CN$, 62 mg/kg BW	LD50 (7 days); first death in 5.1 h	18
Acetonitrile, CH_3CN, 175 mg/kg BW	LD50 (7 days); first death in 7.1 h	18
Single subcutaneous injection		
HCN, 7.8–12.0 mg/kg BW	LD50	10
KCN, 10 mg/kg BW	Loss of consciousness in 100%; blood ammonia levels increased 2.5 times; brain amino acid levels (i.e., leucine,	19

	isoleucine, tyrosine, phenylalanine) increased by 1.5–3.0 times; alpha ketoglutarate, at 500 mg/kg BW by intraperitoneal injection, completely blocked the development of cyanide-induced loss of consciousness and hyperammonemia	
Single oral dose		
8.5 mg KCN/kg BW, equivalent to 3.4 mg CN^-/kg BW	LD50	10, 20
Drinking water, 1,000 mg KCN/L, exposure for 40 days	Marked inhibition of cytochrome oxidase activity in liver, brain, and blood; increased cyanide concentrations in all tissues; inhibition of rhodanese activity; diminished labile sulfur tissue levels	43
Rabbit, *Oryctolagus* spp.		
Isolated aorta strips, 0.00014 µg NaCN/L–140 µg/L	Small contractions measured at lowest dose tested, ED50 at 70 µg/L, and maximum response at 140 µg/L; higher doses up to 14 mg/L produced relaxation	21
Single intramuscular injection, in mg/kg BW		
0.5–1.5	LD50 for HCN	10
1.6	LD50 for NaCN	10
3.1–3.3	LD50 for KCN	10
8.0		
Killed with KCN	Cyanide concentrations, in mg/kg FW, were 1.6 in serum, 5.3 in blood, and <0.4 in other tissues sampled	22
Killed with HCN	Cyanide concentrations, in mg/kg FW, were 9.3 in blood, 2.1 in brain, 2.0 in serum, 0.5 in myocardium, and <0.4 in other tissues	22
Single intravenous injection, in mg/kg BW		
0.6	LD50 for HCN	10
1.2	LD50 for NaCN	10
1.9	LD50 for KCN	10
Single dose administered to eye surface, in mg/kg BW		
1.0	LD50 for HCN	10
4.5–5.1	LD50 for NaCN	10
7.9	LD50 for KCN	10
11.2	Signs of NaCN poisoning in 3 min, death in 7 min	23
Single intraperitoneal injection, in mg/kg BW		
1.7–2.0	LD50 for HCN	10
2.8–2.9	LD50 for NaCN	10

3.6–4.0	LD50 for KCN	10
Administered as solution to skin, in mg/kg BW		
2.3	LD50 for HCN and abraded skin	10
6.9	LD50 for HCN and intact skin	10
14.3	LD50 for KCN and abraded skin	10
19.3	Abraded skin; signs of NaCN poisoning evident in 25 min, death in 41 min	23
22.3	LD50 for KCN and intact skin	10
29.5	Moist skin; signs of NaCN poisoning evident in 79 min, death in 117 min	23
>110	Dry skin; no signs of NaCN poisoning, no deaths	23
Single oral dose, in mg/kg BW		
2.5	LD50 for HCN	10
5.1	LD50 for NaCN	10
5.8	LD50 for KCN	10
12.8	Signs of NaCN poisoning in 4 min, death in 22 min	23
Single oral dose, NaCN, 10–15 mg/kg BW	All dead in 14–30 min; blood cyanide ranged between 3.7 and 5.4 mg/L	24
Inhalation of HCN from combustion of 20 g of polyacrylonitrile	All dead in 12–16 min; blood cyanide ranged between 1.6 and 3.1 mg/L	24
Interval between death and removal of tissues for analysis in rabbits killed by KCN		
Brain	Concentrations dropped from 1.6 mg/kg FW immediately after death to 1.2 in 1 day, 0.92 in 3 days, and 0.04 in 7 days	25
Blood	Residues, in mg/kg FW were 5.7 immediately after death and 2.3 after 21 days	25
Lung	Cyanide concentrations dropped from 2 mg/kg FW just after death, to 0.8 in 7 days	25
Domestic sheep, *Ovis aries*		
Intravenous or intraarterial injection, fetal lambs 80% through gestation (120 days), NaCN, 50–400 µg	Slowing of fetal heart rate, disruption of respiratory movements, significant but inconsistent changes in arterial blood pressure	26
Single intramuscular injection of 10 mg KCN/kg BW	All dead within 17 min; cyanide concentrations postmortem, in mg/kg FW, were 3.3 in blood, 1.5 in plasma, 1.6 in serum, 1.4 in cerebrospinal fluid, 0.9 in brain grey matter, and 1.0 in brain white matter	3, 10, 27
Laboratory white rat, *Rattus* spp.		
Single intraperitoneal injection		

0.1–10 mg CN/kg BW	LD50	28
5 mg NaCN or KCN/kg BW	50% decrease in brain cytochrome oxidase activity within 5–10 min	14
5 mg KCN/kg BW	Reversible intracellular metabolic changes including acidosis and increased lactate levels--typical of cellular anoxia	29
Intravenous injection, constant infusion of 0.15–0.20 mg CN/kg BW per min	LD50 in about 20 min. Rapid progressive reduction in cerebrocortical cytochrome oxidase (cytochrome aa_3) concomitant with increases up to 200% in cerebral blood flow	30
Single intracartoid artery injection of KCN		
1–2 mg/kg BW	Modest acute clinical dysfunction and incomplete suppression of brain electroencephalographic (EEG) activity	31
2.5 mg/kg BW	Some deaths; survivors showed rapid abolition of brain EEG activity, 52% reduction in brain cytochrome oxidase activity, 600% increase in lactate, 85% decrease in glycogen, 32% reduction in ATP, and 73% increase in ADP; all values returned to normal in 6–24 h, and remained normal for balance of 7-day observation period	31
3.5–5 mg/kg BW	High incidence of cardiovascular collapse and death within minutes	31
Tissue residues 2.6–2.9 mg HCN/L	Minimum lethal concentrations in rats poisoned orally with KCN	13
Inhalation exposure route, HCN vapor, in mg/m^3, for various periods		
3,778 for 10 s	LC50	10
1,128 for 1 min	LC50	10
493 for 5 min	LC50	10
151–173 for 30–60 min	LC50	10
Single oral dose		
3.4 mg KCN/kg BW	LD25	32
3.6–4.2 mg HCN/kg BW	LD50	10
5.1–5.7 mg NaCN/kg BW	LD50	10
5.7 mg KCN/kg BW	LD50	32
6, 10, or 14 mg KCN/kg BW	Some deaths in all groups; all dead at higher doses within 60 min; those killed 10 min postadministration had	13

	higer blood CN concentrations than those killed near death or at survival at 60 min	
6.4 mg NaCN/kg BW	LD50	13
7.5–10 mg KCN/kg BW	LD50	10, 13
8.6 mg KCN/kg BW	LD98	32
10 mg KCN/kg BW, equivalent to 4 mg HCN/kg BW	LD50	20
13.2 mg NaCN/kg BW or 7 mg HCN/kg BW	Dead in 10.3 min; tissue cyanide levels, in mg/kg FW, were 8.9 in liver, 5.9 in lung, 4.9 in blood, 2.1 in spleen, and 1.5 in brain	33
40 mg NaCN/kg BW, equivalent to 21 mg HCN/kg BW	Dead in 3.3 min	33
Drinking water exposure		
Equivalent to 8 mg CN/kg BW daily for 21 days	Liver normal	20
Equivalent to 21 mg CN/kg BW daily for 21 days	Significantly increased liver weight	20
200 mg CN/L for 4 weeks	Reduced growth	34
Drinking water of adults contained 150 mg CN/L, as KCN, for 2 weeks, followed by injection with radioselenium-75 and observed for 15 days	Cyanide-treated rats excreted significantly more radioselenium in urine than did controls; half-time persistence of radioselenium in treated group was 28 days vs. 38 days in controls	35
Drinking water of weanling males contained 150 mg CN/L for 9 weeks	Significant reduction in glutathion activity, and in selenium concentrations in blood kidney, liver, and muscle	35
Dietary exposure		
Fed 12 mg CN/kg BW daily for 2 years, equivalent to 300 mg HCN/kg ration	No measurable adverse effects on blood chemistry, growth, survival, or histology; elevated thiocyanate levels in liver and kidneys	1
Fed 500 mg HCN/kg ration to pregnant rats through gestation and lactation	No effect on reproduction	20
Weanlings fed diets of raw lima beans containing 727 mg CN/kg for 3 weeks, or 727 mg CN/kg diet as KCN for 3 weeks	Lima bean diet alone increased hepatic glutamate dehydrogenase (GLDH) and decreased isocitrate dehydrogenase (ICDH) activities; KCN diet had no effect on GLDH and increased ICDH activity, emphasizing the importance of dietary components when evaluating CN-containing diets	36
750 mg CN/kg diet (1,875 mg KCN/kg diet) for 8 weeks,	No measurable effect on food consumption or growth rate;	37

adequate protein	significantly increased serum and urinary thiocyanate concentrations	
As above, protein deficient diet	Reduction in body weight gain, reduction in serum thiocyanate concentration	37
Weanling males fed diets containing 1,500 mg KCN/kg, or 2,240 mg potassium thiocyanate (KSCN)/kg for 50 weeks	No deaths or clinical signs of toxicity; both groups had decreased thyroid gland activity; cyanide, but not thiocyanate, caused reduction in growth rate	38
Isolated liver segments from starved rats exposed to 100 mg KCN/L	Oxygen consumption reduced 80%, and evidence of hepatotoxicity as judged by enzyme release, glutathione depletion, and calcium accumulation in liver; hepatotoxicity prevented by feeding rats fructose	39
Domestic pig, *Sus* spp.		
Fed diet containing 96 mg CN/kg ration, as cassava peel for 72 days	No effect on food consumption or protein metabolism	40

[a]1, EPA 1980; 2, Sterner 1979; 3, Christel et al. 1977; 4, Savarie and Sterner 1979; 5, Tewe 1984; 6, Tewe 1988; 7, Tewe 1982a; 8, Curry 1963; 9, Grandas et al. 1989; 10, Ballantyne 198a; 11, Towill et al. 1978; 12, Ukhun and Dibie 1989; 13, Egekeze and Oehme 1980; 14, Way 1981; 15, Casadei et al. 1984; 16, Purser et al. 1984; 17, Purser 1984; 18, Willhite and Smith 1981; 19, Yamamoto 1989; 20, EPA 1989; 21, Robinson et al. 1985; 22, Ballantyne et al. 1972; 23, Ballantyne 1988; 24, Yamamoto et al. 1979; 25, Ballantyne et al. 1974; 26, Itskovitz and Rudolph 198; 27, Ballantyne 1975; 28, Brattsten et al. 1983; 29, Lotito et al. 1989; 30, Lee et al. 1988; 31, MacMillan 1989; 32, Keniston et al. 1987; 33, Yamamoto et al. 1982; 34, Palmer and Olson 1981; 35, Beilstein and Whanger 1984; 36, Aletor and Fetuga 1988; 37, Tewe and Maner 1985; 38, Philbrick et al. 1979; 39, Younes and Strubelt 1988; 40, Tewe and Pessu 1982; 41, Way 1984; 42, Marrs and Ballantyne 1987; 43, Buzaleh et al. 1989; 44, Bapat and Abhyankar 1984.

Organic cyanide compounds, or nitriles, have been implicated in numerous human fatalities and signs of poisoning—specially acetonitrile, acrylonitrile, acetone cyanohydrin, malonitrile, and succinonitrile. Nitriles hydrolyze to carboxylic acid and ammonia in either basic or acidic solutions. Mice *(Mus* sp.) given lethal doses of various nitriles had elevated cyanide concentrations in liver and brain; the major acute toxicity of nitriles is CN release by liver processes (Willhite and Smith 1981). In general, alkylnitriles release CN much less readily than aryl alkylnitriles, and this may account for their comparatively low toxicity (Davis 1981).

No human cases of illness or death due to cyanide in water supplies are known (EPA 1980). Accidental acute cyanide poisonings in humans are uncommon (Towill et al. 1978); however, a man accidentally splashed with molten sodium cyanide died about 10 h later (Curry 1963). Human cyanide deaths usually involve suicides, where relatively large amounts of sodium cyanide or potassium cyanide are ingested and the victims die rapidly in obvious circumstances. Recovery after oral ingestion is rare. In one case, a spouse emptied capsules containing medicine and repacked them with 40% solid NaCN. The victim took one capsule and ingested about 0.05 g, but vomited and recovered completely (Curry 1963). Human deaths are increasing from gas or smoke inhalation from urban fires, possibly owing to the increased toxicity of fire atmospheres caused by the use of organocyanide plastics in modern construction and furnishings (Egekeze and Oehme 1980). Hydrogen cyanide may be important in some fires in producing rapid incapacitation, causing the victims to remain in the fire and die from carbon monoxide or other factors, although HCN concentrations of 60 mg/L air and lower had minimal effects (Purser 1984). Exposure to the mixture of HCN and carbon monoxide, with accompanying changes in cerebral blood flow during attempts to escape from fires, may be a cause of collapse and subsequent death

(Purser 1984). For example, cynomolgus monkeys (*Macaca* spp.) exposed to pyrolysis products of polyacrylonitrile (PAN) and to low-level HCN gas had similar physiological effects in both atmospheres, specifically: hyperventilation, followed by loss of consciousness after 1-5 min; and brachycardia, with arrhythmias and T-wave abnormalities. Recovery was rapid following cessation of exposure (Purser et al. 1984). Because HCN is the major toxic product formed by the pyrolysis of PAN, Purser et al. (1984) suggested that HCN may produce rapid incapacitation at low blood levels of cyanide in fires, while death may occur later due to carbon monoxide poisoning or other factors.

Finally, cyanide does not appear to be mutagenic, teratogenic, or carcinogenic in mammals (EPA 1980; Ballantyne 1987a). In fact, there has been a long-standing hypothesis for an anticancer effect of the cyanogenic glycoside amygdalin (also called laetrile). The hypothesis is based on amygdalin's selective hydrolysis by a beta glucosidase, liberating cyanide and benzaldehyde at the neoplastic site. The cyanide then selectively attacks the cancer cell, which is presumed to be low in rhodanese, whereas normal cells are assumed to possess sufficient rhodanese and sulfur to detoxify the cyanide (Way 1981). However, many tumors are neither selectively enriched in beta glucosidase nor low in rhodanese (Way 1981).

Recommendations

Proposed free cyanide criteria suggest that sensitive species of aquatic organisms are protected at <3 µg/L, birds and livestock at <100 mg/ kg diet, and human health at concentrations of <10 µg/L drinking water, <50 mg/kg diet, and <5 mg/m^3 air (Table 6).

Analytical methodologies need to be developed that differentiate between free cyanide (HCN and CN^-) and other forms of cyanide, and that are simple, sensitive (i.e., in the µg/L range), and accurate (Smith et al. 1979; Leduc et al. 1982). Procedures need to be standardized that ensure prompt refrigeration and analysis of all samples for cyanide determination because some stored samples generate cyanide while others show decreases (Gee 1987).

Periodic monitoring of cyanide in waterways is unsatisfactory for assessing potential hazards because of cyanide's rapid action, high toxicity, and low environmental persistence. A similar case is made for cyanide in the atmosphere. Development of a continuous monitoring system of cyanides in waterways and air is recommended, with emphasis on point source dischargers, such as industrial and municipal facilities (Towill et al. 1978; Egekeze and Oehme 1980; Leduc et al. 1982). Information is needed on the fate of cyanide compounds in natural waters, relative contributions of natural and anthropogenic sources, and critical exposure routes for aquatic organisms (Leduc et al. 1982). Additional research is needed on the origin of cyanide in wilderness and rural watershed areas, specifically the roles of organic wastes and their associated bacterial flora, aquatic vegetation induced by nutrient enrichment, and terrestrial plant cover in the watershed (Leduc 1984).

Table 6. Proposed free cyanide criteria for the protection of living resources and human health.

Resource criterion, and other variables	Concentration	Reference[a]
Freshwater organisms		
Effect levels, in µg/L medium		
Minimal impairment, most species of fish	3–5	1, 2, 3, 4, 5, 6
Reduced survival, amphipods	>3–34	1, 7
Safe, most fish species	3.5 (24-h average, not to exceed 52 at any time)	7
Significant impairment, most species of fish	8–16, exposure for at least 20 days	6, 7
Hazardous		
Most fish species	>11	1, 4
Microorganisms	>300	8

Reduced survival, chronic exposure		
Bivalve molluscs, larvae	>14	1
Fish, many species	30–150	1, 5
Impaired reproduction, sensitive species of fish	>25	2
Impaired swimming ability, growth, development, and behavior	>100	3, 6
Lethal to rapidly lethal, acute exposure	300–1,000	5
Marine organisms		
Effect levels, in µg/L seawater		
Adverse effects, chronic exposure	>2	7
Minimal risk	<5	1
Hazardous	>10	1
Lethal	>30	7
Sediments, Great Lakes		
Effect level, in mg total cyanide/kg dry weight (DW)		
Nonpolluted	<0.10	20
Moderately polluted	0.1–0.25	20
Heavily polluted	>0.25	20
Birds		
Domestic chickens		
Diet, safe level, in mg total cyanide/kg ration fresh weight (FW)	90–<100	9, 10
Waterfowl		
Drinking water, safe	<50	21, 22
Livestock		
level in mg/L total cyanide		
Diet, safe level, in mg/kg FW		
Free cyanide	<100	9
Total cyanide	<625	11
Forage, hazardous level, in mg/kg FW	>200	8
Laboratory white rat		
Diet, safe level, in mg/kg ration FW	<1,000	19
Blood, in mg/L		
Normal	0.25–0.45	12
Minimum lethal concentration	2.6–2.9	12
Liver		
Minimum lethal concentration, in mg/kg FW	0.5–6.1	12
Human health		
Drinking water, in µg/L		
Recommended	<5–<10	1, 6, 8, 13
United States nationwide survey	Max. 8	7
Safe	<10	1
Goal, United States	<10	7, 14
Maximum allowable limit, United States	10	13
Goal, Canada	<20	7
Lifetime health advisory, United States and Canada	<154	14

Acceptable	<200	7
Mandatory limit	200	13
Rejected	>200	1, 8
10-day health advisory		
Child	<220	14
Adult	<770	14
Diet		
Acceptable daily intake		
Water	1.5 mg, equivalent to 0.02 mg/kg body weight (BW) daily for 70-kg adult	15
Food, in mg/kg BW	8.4	7
Food, in mg/kg FW	<50	15
Food, in mg total cyanide/kg FW	<415	11
Cassava, *Manihot esculenta*, roots, total cyanide, in mg/kg FW		
Safe	<50	16
Moderately toxic	50–100	16
Very poisonous	>100	16
Food items, in mg/kg		
Cocoa	<20 DW	13
Beans, nuts	<25 DW	1
Cereals, grains	<25 DW	13
Citrus fruits	<50 FW	1
Uncooked pork	<50 FW	13
Grains	<75 FW	1
Cereals flours	<125 DW	13
Spices	<250 FW	1, 13
Frozen meat	<950 FW	1, 13
Bakery products, yeast	<1,500 DW	13
Egg white solids	<1,000 DW	13
Tissue residues		
Blood and spleen, in µg/L or µg/kg FW		
Normal	77	17
Suspected poisoning	>1,000	17
Whole blood, in µg/L		
Usually fatal	1,000–2,000	15
Whole body, in mg/kg BW		
Fatal	4, if taken rapidly	18
Air, in mg/m^3		
Recommended safe levels		
Soviet Union, Romania, Hungary, Bulgaria, Czechoslovakia	<0.3	1
United States	<5	14
Most countries	<11	1, 15
Occupational exposure		
Proposed safe level, United States	<3	15
Safe ceiling concentration	<5	1
Hazardous levels	4.2–12.4	1
Soils in mg/kg DW		
Free cyanide		
Background	1	20
Moderate contamination	10	20
Requires cleanup	100	20
Complex cyanide		
Background	5	20

Moderate contamination	50	20
Requires cleanup	100	20

[a]1, Towill et al. 1978; 2, Smith et al. 1979; 3, Doudoroff 1976; 4, Leduc 1981; 5, Leduc 1984; 6, Leduc et al. 1982; 7, EPA 1980; 8, Egekeze and Oehme 1980; 9, Gomez et al. 1983; 10, Gomez et al. 1988; 11, Okeke et al. 1985; 12, Egekeze and Oehme 1979; 13, EPA 1973; 14, EPA 1989; 15, Marrs and Ballantyne 1987; 16, Dufour 1988; 17, Gee 1987; 18, Shaw 1986; 19, Tewe 1982; 20, Beyer 1990; 21, Allen 1990; 22, Clark and Hothem 1991.

In aquatic systems research is needed in several areas: (1) long-term effects of cyanide on life cycles, growth, survival, metabolism, and behavior of a variety of aquatic organisms and microorganisms in addition to fish (Towill et al. 1978; Leduc et al. 1982); (2) effects of seasonal pulses of cyanide on aquatic organisms in rural and wilderness areas (Leduc 1984); (3) influence of various environmental parameters (e.g., oxygen, pH, temperature), if any, on adaptive resistance to cyanide (Leduc 1981, 1984); and (4) usefulness of various biochemical indicators of cyanide poisoning, such as cytochrome oxidase inhibition (Gee 1987) and vitellogenin levels in fish plasma (*gairdneri*) (Ruby et al. 1986).

The use of M-44 sodium cyanide capsules for predator control was suspended and cancelled by the U.S. Environmental Protection Agency on 9 March 1972. M-44 use was again permitted by the U.S. Environmental Protection Agency beginning on 4 February 1976, provided that "each authorized or licensed applicator shall carry an antidote kit on his person when placing or inspecting M-44 devices. The kit shall contain at least 6 pearls of amylnitrite and instructions on their use. Each authorized or licensed applicator shall also carry on his person instructions for obtaining medical assistance in the event of accidental exposure to sodium cyanide" (EPA 1976a, 1976b).

Farmers need to be aware of factors that influence the cyanogenic potential of forage crops and to conduct regular inspections of grazing fields for cyanogenic plants. Moreover, hay and silage should be properly cured in order to minimize cyanide content before feeding to livestock (Egekeze and Oehme 1980). Selective breeding of plants with low cyanide content will help reduce livestock poisoning, but the most advisable prevention method at present is to prohibit grazing on fields where cyanogenic plants are present (Egekeze and Oehme 1980). More research seems needed on (1) effects of drought and other factors that may increase the concentration of cyanogenic glycosides in livestock forage plants, (2) mechanisms of cyanide liberation by plants, and (3) effects of cyanide on wildlife and range animals that graze on foliage with high cyanogenic glycoside content (Towill et al. 1978).

Research is needed on low-level, long-term cyanide intoxication in mammals by oral and inhalation routes in the vicinities of high cyanide concentrations, especially on the incidence of skin dermatitis, nasal lesions, and thyroid dysfunction, and on urinary thiocyanate concentrations. These types of studies may provide a more valid rationale in establishing standards and threshold limit values for HCN and inorganic cyanide (Towill et al. 1978; Egekeze and Oehme 1980).

Data are scarce on the carcinogenic, teratogenic, and mutagenic properties of cyanide, and on the distribution and transformation of cyanides in air, land, or water. Additional analysis of available information and more research in these areas is recommended. Finally, more research is needed on cyanide toxicokinetics because cyanide is a very reactive nucleophile that distributes widely through the body, is permeable to cell membranes, and may accumulate in the fetus (Towill et al. 1978).

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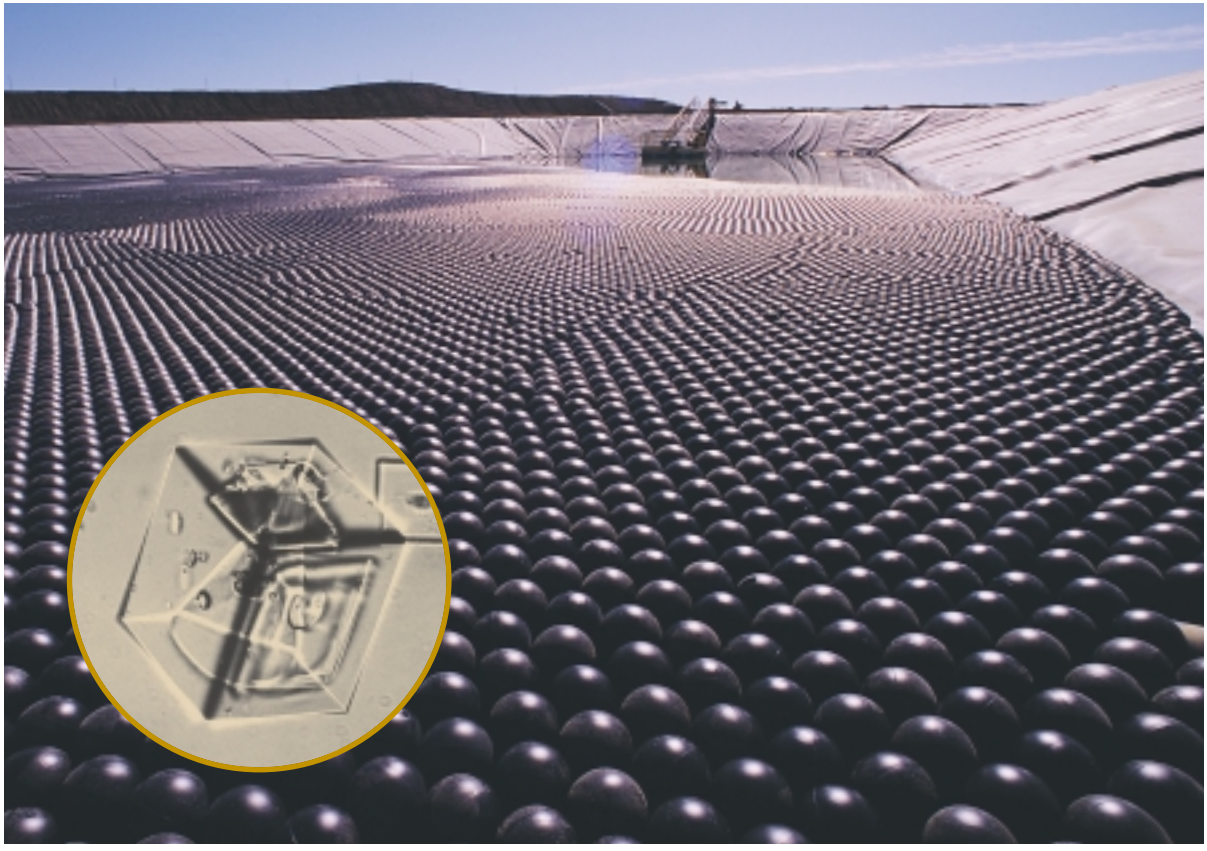
The Management of Cyanide in Gold Extraction

by

Mark J. Logsdon, MSc

Karen Hagelstein, PhD, CIH

Terry I. Mudder, PhD



International Council on
Metals and the Environment

ICME

The International Council on Metals and the Environment (ICME) has published this document as part of its ongoing efforts to provide information on environmental and related health matters affecting the mining and metals sector. The contents of ICME publications range from general and technical information to discussions of policy and regulatory issues. The topics examined may be of interest not only to industry, but also to others, including policy makers, regulators, educators and the public at large. It is hoped that ICME publications provide insight into what are sometimes difficult and complex issues.

Although the views expressed are those of the authors, ICME welcomes questions and comments on the perspectives and information contained in its documents. ICME also appreciates suggestions regarding other issues of public importance for possible future publications.

Founded in 1991, ICME is a non-governmental organization that promotes the development and implementation of sound environmental and health policies and practices in the production, use, recycling and disposal of non-ferrous and precious metals. In addition to its publications, ICME's extensive information program includes a Web site and a quarterly newsletter with a worldwide distribution.

International Council on Metals and the Environment
294 Albert Street, Suite 506
Ottawa, Ontario
CANADA K1P 6E6
Tel: (613) 235-4263
Fax: (613) 235-2865
e-mail: info@icme.com
http://www.icme.com

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Mark J. Logsdon, MSc
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ICME

INTERNATIONAL COUNCIL ON
METALS AND THE ENVIRONMENT

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Foreword

The mining industry, and in particular the gold mining industry, has been using cyanide in its production processes for many decades. While cyanide is commonly perceived as being a deadly substance, it is in fact a widely used chemical that is essential to the modern world. The key to its safe use is the implementation of sound management practices.

While public concern about cyanide is valid and indeed understandable, much of the recent media attention and public reaction regarding the use of cyanide in mining operations has arisen due to a lack of understanding of the nature of cyanide and its effects on health and the environment. While there is considerable technical information available to those who produce, transport and use cyanide, easy-to-understand information has not heretofore been provided for a less technical audience. In an attempt to remedy this situation and to address public concern about the use of cyanide in gold extraction, the International Council on Metals and the Environment has commissioned the present document.

The Management of Cyanide in Gold Extraction gives an overview of the chemical's uses and risks, with special emphasis on its use in the recovery of gold. The publication begins by describing the properties of cyanide and its general uses in industry, then moves on to address more specifically the life cycle of cyanide in the mining environment—its production, use in mineral extraction, and general and environmental chemistry. After presenting this information, the publication explains how the principles of risk assessment, risk management and risk communication contribute to the safe use of cyanide in gold recovery.

This work has been prepared by recognized experts and should be a useful reference for anyone involved in decision making related to the presence of cyanide in mining operations, whether from a local or global perspective. It is hoped that international regulators, policy makers, community leaders and all other interested readers, including those engaged in the mining and metals industry, will find the work to be both balanced and informative, and thereby gain a better understanding of the characteristics of cyanide and its unique role in gold recovery.

Gary Nash
Secretary General
ICME

Executive Summary

Cyanide is the chemical of choice for gold recovery.

Cyanide is one of only a few chemical reagents that will dissolve gold in water. It is a common industrial chemical that is readily available at a reasonably low cost. For both technical and economic reasons, cyanide is the chemical of choice for the recovery of gold from ores. Cyanide has been used in metal extraction since 1887 and is now safely used and managed in gold recovery around the world. Gold mining operations use very dilute solutions of sodium cyanide, typically in the range of 0.01% and 0.05% cyanide (100 to 500 parts per million).

Most of the cyanide produced is used as a basic building block for the chemical industry.

Cyanide is produced in large amounts (about 1.4 million tonnes each year) as one of a few basic compounds used chiefly to synthesize a wide range of industrial organic chemicals such as nylon and acrylics. Gold recovery accounts for approximately 18% of total world cyanide production.

Cyanide is produced naturally in a number of microorganisms, insects and plants.

Cyanide is a naturally occurring molecule of carbon and nitrogen. It existed on Earth before life began and was one of the fundamental building blocks in the evolution of life. Low concentrations of cyanide are present in nature, for example in many insects and plants, including a wide range of vegetables, fruits and nuts, where it provides protection against predators. In addition, cyanide is present in much of the everyday environment to which we are exposed, for example in road salt and automobile exhaust and as a stabilizer in table salt.

Cyanide is not persistent.

One of the major health and environmental concerns with some synthetic chemicals is that they do not decompose readily and can thereby accumulate in the food chain. Cyanide, however, is transformed by natural physical, chemical and biological processes into other, less toxic chemicals. Since cyanide oxidizes when exposed to air or other oxidants, it decomposes and does not persist. While it is a deadly poison when ingested in a sufficiently high dose, it does not give rise to chronic health or environmental problems when present in low concentrations.

Cyanide is attenuated through natural processes.

Over time, natural processes such as exposure to sunlight can reduce the concentration of toxic forms of cyanide in solutions to very low values.

The risks of cyanide production, use and disposal can be well managed.

Responsible companies in both the chemical industry and the mining industry employ stringent risk management systems to prevent injury or damage from the use of cyanide. Cyanide in mining solutions is collected, either to be recycled or destroyed, after gold is removed. Managing risks associated with the use of cyanide involves sound engineering, careful monitoring and good management practices in order to prevent and mitigate potential releases of cyanide to the environment.

Communicating information about the risks of cyanide to employees and the public is essential to sound management practices.

The environmental fate of cyanide has been well studied. Cyanide is highly regulated and its risk management is well documented. Risk communication provides information about cyanide both within the operating plant and externally, to the public. Communication of information to the internal staff is the first step in communicating the nature and extent of risk to the general public. Effective communication and emergency planning programs should also be coordinated with the proper local authorities.

SECTION 1

What Is Cyanide?

Cyanide is a general term for a group of chemicals containing carbon and nitrogen. Cyanide compounds include both naturally occurring and human-made (anthropogenic) chemicals. There are more than 2,000 natural sources of cyanide, including various species of arthropods, insects, bacteria, algae, fungi and higher plants. The principal human-made cyanide forms are gaseous hydrogen cyanide and solid sodium and potassium cyanide. Because of its unique properties, cyanide is used in the manufacture of metal parts and numerous common organic products such as plastics, synthetic fabrics, fertilizers, herbicides, dyes and pharmaceuticals.

There is justifiable public concern about the use of cyanide in industrial settings. Cyanide is a toxic substance and can be lethal if ingested or inhaled in sufficient amounts. This is also true for many other chemicals such as gasoline and common household cleaning supplies. As is the case for the thousands of other chemicals used in our modern industrial processes, knowledge, proper handling procedures and a responsible attitude are critical to the safe and beneficial use of cyanide.

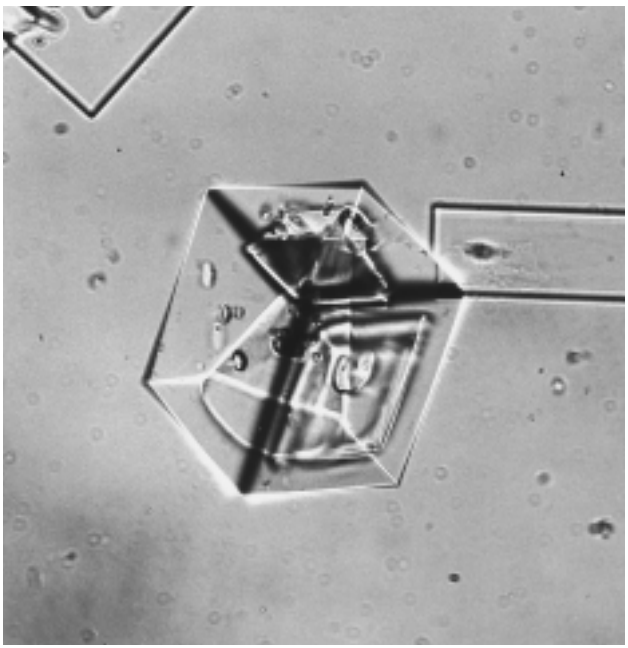


Photo courtesy of DuPont

Microscopic view of sodium cyanide crystals.

Mining is one industrial activity that uses a significant amount of cyanide—about 20% of total production. Since 1887, cyanide solutions have been used primarily to extract gold and silver from ores that otherwise could not be mined effectively. In addition, cyanide is used in low concentrations as a flotation reagent to aid in the recovery of base metals such as lead, copper and zinc.

SECTION 2

Natural Occurrences of Cyanide

Carbon and nitrogen, the two elements that make up cyanide, are present all around us. Together they make up almost 80% of the air we breathe, and both are present in the organic molecules that are the basis of all life forms. Hydrogen cyanide was formed in the earliest stages of the development of our planet as a precursor to amino acids, from which life on Earth evolved. Cyanide is formed naturally. It is produced and used by plants and animals as a protective mechanism that makes them an unattractive food source. Many organisms may either adapt to the presence of cyanide or detoxify it.

A natural source of hydrogen cyanide (HCN) is a sugar-like compound called amygdalin, which exists in many fruits, vegetables, seeds and nuts, including apricots, bean sprouts, cashews, cherries, chestnuts, corn, kidney beans, lentils, nectarines, peaches, peanuts, pecans, pistachios, potatoes, soybeans and walnuts. In the kernel of bitter almond, there is about 1 mg of HCN as amygdalin. Table 1 presents data on the amount of cyanide present in a variety of other foodstuffs.

TABLE 1. Cyanide Concentrations in Selected Plants

Plant Species	**Concentration ($mg.kg^{-1}$)**
Cassava (sweet varieties)	
leaves	377–500
roots	138
dried roots	46–<100
mash	81
Bamboo tip	Max. 8,000
Lima bean (Burma)	2,100
Almond (Bitter)	280–2,500
Sorghum (young plant, whole)	Max. 2,500

Source: Excerpted from Eisler, 1991

Cyanide compounds are produced in thousands of plant species and in other life forms. In some plants, cyanide occurs in concentrations that would be judged “hazardous” if they were associated with manufactured sources. Plants such as alfalfa, sorghum and cassava are known sources of cyanide poisoning to livestock and humans.

In addition to these naturally occurring forms of cyanide, cyanide compounds are also present in such everyday anthropogenic sources as automobile exhaust, cigarette smoke, and even road and table salt.

SECTION 3

Industrial Uses of Cyanide

Cyanide is one of the main building blocks for the chemical industry because of its composition of carbon and nitrogen—both common elements—and the ease with which it reacts with other substances.

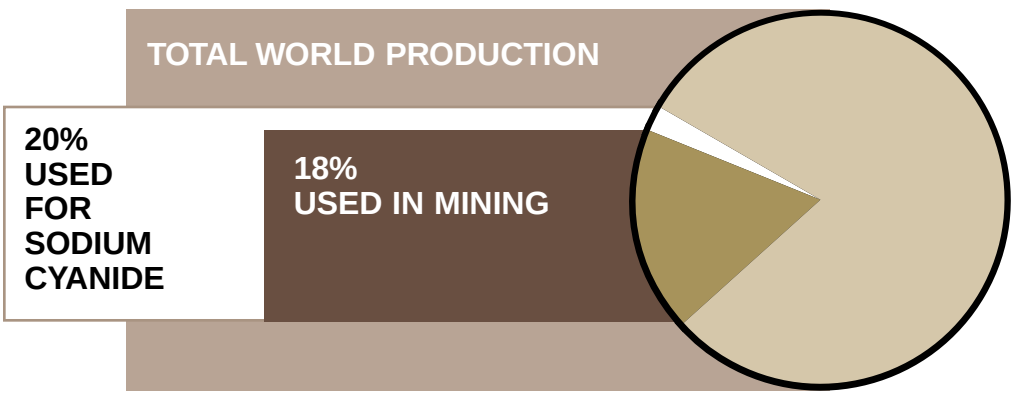
Over one million tonnes of cyanide, representing about 80% of total production, are used annually in the production of organic chemicals such as nitrile, nylon and acrylic plastics. Other industrial applications include electroplating, metal processing, steel hardening, photographic applications and synthetic rubber production.

Iron cyanides are often used in road salt as an anti-caking additive. Hydrogen cyanide vapour has been widely used to exterminate rodents and large predators, and in horticultural practice to control insect pests that have developed resistance to other pesticides.

In addition, cyanide is used in pharmaceuticals such as the anticancer substance laetrile and the blood pressure–reducing drug nitroprusside. Cyanide compounds are also used in surgical dressings that promote healing and reduce scarring.

The remaining 20% of cyanide production is used to manufacture sodium cyanide, a solid form of cyanide that is relatively easy and safe to handle. Of this, 90% (i.e. 18% of total production) is used in mining around the world, mostly for gold recovery.

FIGURE 1. Portion of World Cyanide Production Used in Mining



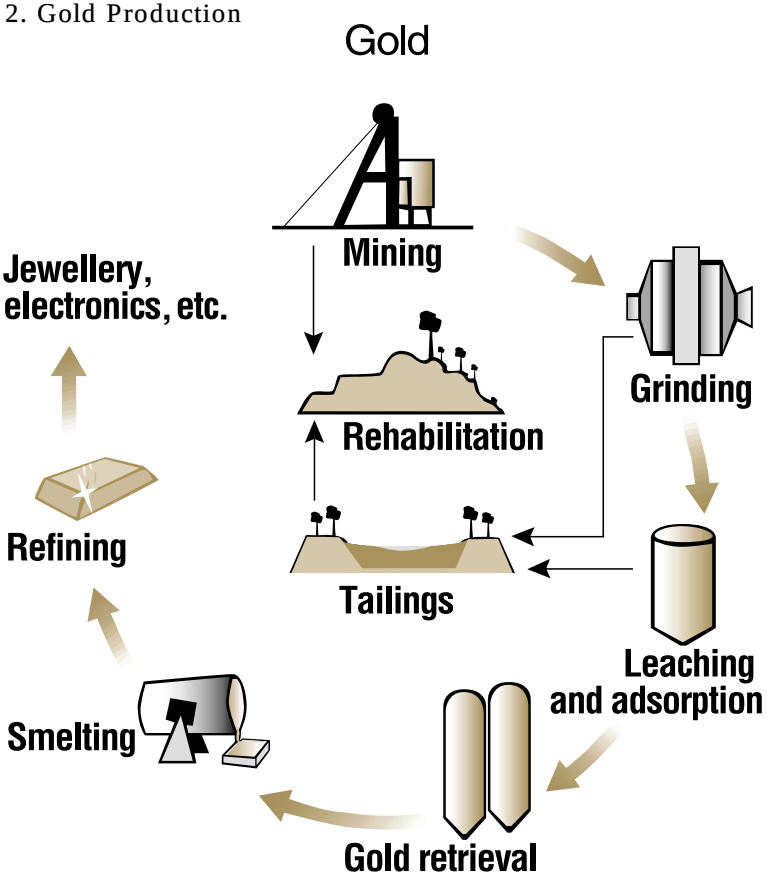
SECTION 4

Cyanide Use in Gold Production

One of the reasons for the high value placed on gold is its resistance to attack by most chemicals. One exception is cyanide, or more specifically, a cyanide-containing solution, which dissolves the precious metal.

Cyanide is used in mining to extract gold (and silver) from ores, particularly low-grade ores and ores that cannot be readily treated through simple physical processes such as crushing and gravity separation.

FIGURE 2. Gold Production



Courtesy of WMC Limited

The Process

The use of water-based solutions to extract and recover metals such as gold is called "hydrometallurgy." Gold mining operations use very dilute solutions of sodium cyanide (NaCN), typically in the range of 0.01% and 0.05% cyanide (100 to 500 parts per million). The process of metal dissolution is called leaching. The sodium cyanide dissolves in water where, under mildly oxidizing conditions, it dissolves the gold contained in the ore. The resultant gold-bearing solution is called "pregnant solution." Either zinc metal or activated carbon is then added to the pregnant solution to recover the gold by removing it from the solution. The residual or "barren" solution (i.e. barren of gold) may be re-circulated to extract more gold or routed to a waste treatment facility. Approaches to treating this waste solution of cyanide are discussed in Section 7.

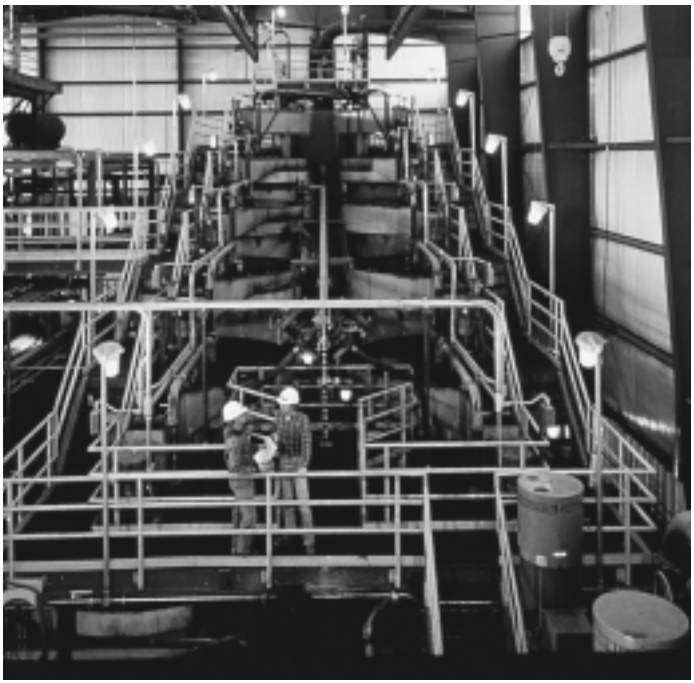


Photo courtesy of Minorco

Gold recovery from cyanide solution using activated carbon (charcoal).

There are two general approaches to leaching gold from mined ore using cyanide: tank leaching and heap leaching.

Tank leaching is the conventional method, in which gold ore is crushed and ground to a size of less than one millimetre in diameter. In some cases, a portion of the gold can be recovered from this finely ground material as discrete particles of gold using gravity-separation techniques. In most cases, the finely ground ore is directly leached in tanks to dissolve the gold in a cyanide solution. When gold is recovered in a conventional plant with leaching in tanks, the barren solution will be collected along with the solid wastes (tailings) in a tailings impoundment system. There, part of the solution will remain within the pores of the settled tailings and part will decant and collect in a pond on top of the tailings, from which it is recycled back to the plant. In most plants, because impurities

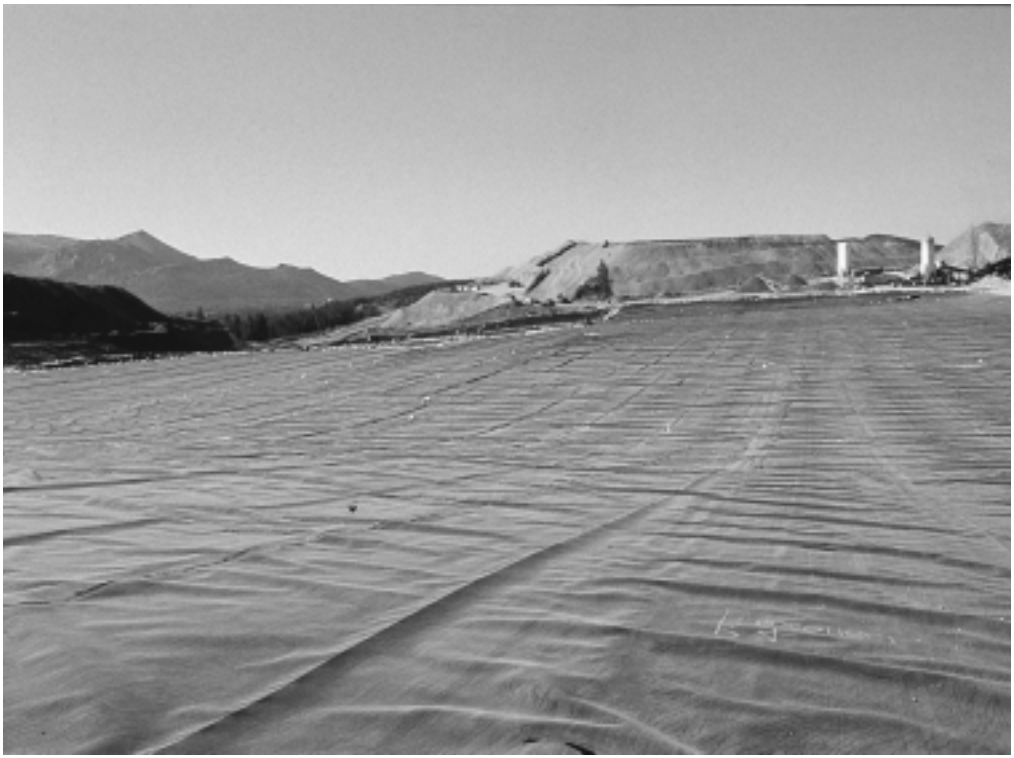


Photo courtesy of Minorco

Construction of a leach pad at Pikes Peak, Colorado, USA.

build up in these solutions, some of the cyanide-bearing solutions must be pumped to a treatment system for disposal (see Section 7).

Recent technical advances enable the heap-leaching of some gold ores. With this method, the ore is crushed to less than a few centimetres in diameter and placed in large piles or heaps. A solution of cyanide is trickled through these heaps to dissolve the gold. When heap-leaching technology is used to extract gold, the barren solution is collected in a pond, from which it is commonly recharged with cyanide and recycled back into the leaching system.

The modern gold industry uses cyanide almost exclusively as the leaching agent for gold. Other complexing agents such as thiourea, chlorides and other halides have been used to extract gold from ores, but these are not generally cost-effective and present their own environmental and health concerns. Cyanide complexes are more stable and effective, and do not require additional aggressive chemicals to effect gold recovery. Cyanide has

been used in mining for over a century *(see box)*. An older technique for gold recovery, which is no longer used in modern gold plants, is amalgamation with liquid mercury. In some developing countries, artisanal miners still use liquid mercury as a means of complexing gold from small mine workings. This practice is discouraged, however, as poor management of both liquid mercury and the vapour arising from volatilizing mercury contributes to serious health problems among artisanal miners.

Box 1. History of Cyanide Use in Mining

While environmental concerns over the use of cyanide in mining have become more public only in the last few years, there actually is a very long history of cyanide use in metallurgical and related processes all around the world. Dippel and Diesbach discovered "Prussian blue" (iron ferrocyanide) in 1704. The earliest well-documented work was Scheele's studies of solubility of gold in cyanide solutions dating from 1783 in Sweden. Gold-cyanide chemistry was studied actively in the mid-19th century in England (Faraday), Germany (Elsner), and Russia (Elkington and Bagration). By 1840, Elkington held a patent for the use of potassium cyanide solutions for electroplating gold and silver. Elsner led the evaluation of the role of oxygen in gold dissolution using cyanide solutions, and "Elsner's Equation" describing the extraction of gold from ores by cyanide was known by 1846.

Patents formalized by McArthur and the Forrest brothers in 1887 and 1888 effectively established the current cyanidation process, the use of cyanide dissolution and precipitation using zinc. However, there were still earlier patents in the USA for cyanide leaching (Rae in 1869) and recovery from chlorinated solutions using charcoal (Davis in 1880). The first commercial-scale cyanidation plant began operating at the Crown Mine in New Zealand in 1889, and by 1904 cyanidation processes were also in place in South Africa, Australia, United States, Mexico and France. Therefore, by the turn of the century, the use of cyanide to extract gold from low-grade ores was a fully established metallurgical technology.

SECTION 5

Production and Handling of Cyanide

Cyanide is produced industrially in one of two ways: as a by-product of the manufacture of acrylic fibres and certain plastics, or by combining natural gas and ammonia at high temperatures and pressures to produce hydrogen cyanide (HCN) gas. Subsequently, hydrogen cyanide gas can be combined with sodium hydroxide (NaOH) to produce sodium cyanide (NaCN) and water (H_2O). The water is then removed by drying and filtering, and the sodium cyanide is formed into solid, white briquettes that are about 10 centimetres square.

The solid sodium cyanide briquettes are maintained under controlled temperature and moisture. At the manufacturing location, the briquettes are packaged in labelled, sealed containers to protect the briquettes from both crushing and moisture. The containers may be disposable plywood boxes with non-returnable liners, non-returnable steel drums, or re-useable steel bins. In some circumstances, the briquettes are dissolved and the cyanide solution is transported as a liquid in specially designed tanker trucks.

All shipments of sodium cyanide are accompanied by Material Safety Data Sheets (MSDSs) that provide the chemistry and toxicity of sodium cyanide, instructions in case of accidents, emergency telephone numbers for assistance and additional information from the manufacturer. All shipments are inventoried as material leaves the producer, and the inventory is checked against delivery records to ensure proper surveillance at all times.

There are three primary producers of solid, liquid and gaseous cyanide in the world: Dupont, in the United States, ICI, in England, and Degussa Corporation, in Germany. Annual worldwide production is approximately 1.4 million tonnes of HCN.[1] As mentioned earlier, 20% of the total HCN production is used to produce sodium cyanide (NaCN) and the remaining 80% is used in numerous other industrial activities such as the production of chemicals. Sodium cyanide is also produced in the USA by FMC Corporation.

The three primary producers are major international chemical manufacturers that understand their responsibility for their products. For example, formal corporate policies

1 1996 amounts. Usage in mining has remained essentially constant for the last decade.

Photo courtesy of DuPont

Storage of drums containing sodium cyanide.

ensure that cyanide is sold only to companies that have the ability and commitment to protect workers, the public and the environment. The manufacturers contract only with selected carriers that have records of transportation safety consistent with the manufacturers' internal standards. The manufacturers maintain a staff of safety and transportation specialists to work with purchasers and others in the areas of training, facility design and related safety measures.

Mining companies store sodium cyanide in secure areas that are kept dry, cool, dark and ventilated. In the storage area, cyanide packages are placed on pallets in their original containers above watertight floors, usually made of concrete, with proper containment in the unlikely event of spillage. Regardless of the container type, empty containers are washed and the rinse water is used in the site's gold recovery plant (to take advantage of

the small amounts of cyanide that could be present) or is processed through the wastewater treatment system prior to being discharged under controlled and permitted conditions.

Mining companies hold special training programs for all employees who work with or around cyanide. They also have materials handling and safety plans prepared by qualified industrial hygienists and supervised by project safety officers. These health and safety plans assign employee responsibilities and control the handling and use of sodium cyanide from its arrival at the mine site through to the metallurgical process. Area gas monitors, proper protective clothing, self-contained breathing apparatus and first-aid stations equipped with eyewash and shower facilities are utilized by cyanide-handling operations at mines. Companies' industrial hygiene programs include annual training, access to all MSDSs and air monitoring to ensure worker safety, as well as procedures for documenting all health and safety information and incidents at mine sites.

Photo courtesy of Degussa Corporation

On-site assistance and safety training are provided to gold mines by cyanide producers.

Modern industrial hygiene programs at gold mining operations have been effective at minimizing accidental cyanide poisoning at mine sites. Indeed, a search of industrial accident records in Australia, Canada, New Zealand and the United States has revealed only three accidental deaths in which cyanide was implicated at gold mine sites in the past 100 years. The first was not directly related to gold recovery, the second involved entry into an enclosed space—a fatal mistake, and the third was not conclusively attributed to cyanide.[2]

2 Both incidents were found in the 107-year fatality database of the Ontario Minister of Labour. In 1952, a blacksmith at the MacLeod-Cockshutt Gold Mines died due to cyanide poisoning following an explosion of molten cyanide; he had been preparing a bath of melted sodium cyanide to case-harden a wrench. In 1961, a worker at the Hallnor Mines Mill died of poisoning from hydrocyanic gas after climbing into an agitator tank to retrieve flake cyanide he had inadvertently thrown into the tank. In 1982, a labourer at an Arizona gold recovery operation collapsed at work and died five days later. Cyanide was suspected, but the evidence as to how the worker became exposed to cyanide was inconclusive.

SECTION 6

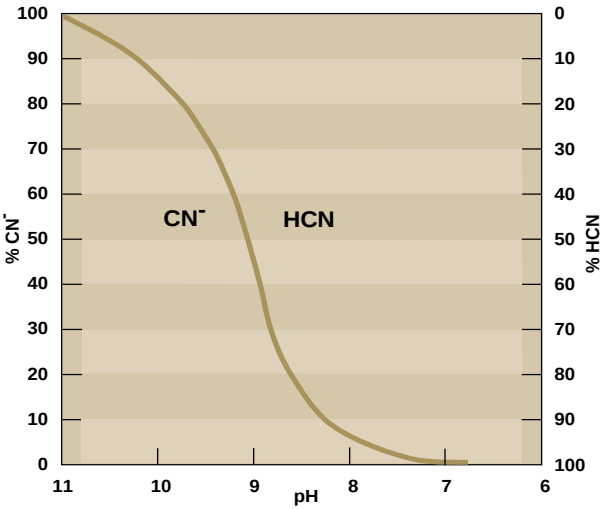
Cyanide in Solutions

After gold is extracted via the hydrometallurgical processes, three principal types of cyanide compounds may be present in wastewater or process solutions: free cyanide, weakly complexed cyanide and strongly complexed cyanide. Together, the three cyanide compounds constitute "total cyanide." An understanding of the chemistry of these three types of cyanide provides insights into their behaviour with respect to safety and the environment.

Free Cyanide

"Free cyanide" is the term used to describe both the cyanide ion (CN^-) that is dissolved in the process water and any hydrogen cyanide (HCN) that is formed in solution. The solid sodium cyanide briquettes dissolve in water to form sodium ion and the cyanide anion (CN^-). The cyanide anion then combines with hydrogen ion to form molecular HCN. The concentration of hydrogen ion in the process water is expressed by the familiar parameter pH.[3] Nearly all free cyanide is present as HCN when there is ample hydrogen ion present, (i.e. at a pH value of 8 or less). This HCN can then volatilize and be dispersed into the air. When the pH is greater than 10.5, there is little hydrogen ion present and nearly all of the free cyanide is present as CN^-. Under normal conditions of temperature and pressure, the concentrations of HCN and CN^- are equal at a pH value of approximately 9.4.

FIGURE 3. CN^-/HCN Equilibrium with pH



Source: Scott and Ingles, 1981.

3 When the pH of a solution is 7, the solution is said to be neutral. Solutions with pH less than 7 are said to be acidic, whereas those with pH greater than 7 are said to be alkaline.

These forms of free cyanide are important because they are considered to be the most toxic cyanides. However, they also happen to be the forms that are readily removed from solutions through both engineered treatment processes and natural attenuation mechanisms. The biological, chemical and physical processes that affect cyanide concentrations in water, soil and air have been extensively studied during the last two decades, so that their behaviour in the environment is well understood.

One of the most important reactions affecting free cyanide concentration is the volatilization of HCN, which, like most gases, will separate from water and escape into the air. Free cyanide is not persistent in most surface waters because the pH of such waters is usually about 8, so that HCN volatilizes and disperses. Hydrogen cyanide's volatility and subsequent transformation to benign compounds in air are important because they act as a natural mechanism for controlling free cyanide concentrations in waste and process waters at mines.

Natural processes alone can reduce the free cyanide concentration from solutions in areas open to the atmosphere in the gold production facilities, such as process ponds and tailings impoundments, to very low values—often to levels below regulatory concern or even the limits of detection.

In the gold plant, however, operators maintain the solution pH at values near 10.5 in order to prevent volatilization. This preserves cyanide in the gold extraction system where it is needed and at the same time limits the risk of worker inhalation exposure to high concentrations of HCN gas in a confined space.

Barrick Gold Corporation

Control centre for gold recovery plant (cyanidation).

Cyanide Complexes

While cyanide-bearing solutions are used in mining because they react with gold, they also react with other metals. Gold ores almost always contain other metals, including iron, copper, zinc, nickel and silver as well as other elements such as arsenic. In most ore bodies, the concentrations of other metals typically exceed the concentration of gold by several orders of magnitude. For example, a low-grade gold ore suitable for cyanide leaching might contain 0.5 to 1 gram of gold per tonne (0.5 to 1 part per million [ppm] gold); in contrast, the iron concentration of average crustal rocks is about 3.5% (35,000 ppm). Metals such as copper, zinc and nickel may be present in concentrations ranging

TABLE 2. Analyses of Barren Solutions[4]

	CONCENTRATION RANGE milligrams per litre[5] ($mg.L^{-1}$)
Total Cyanide	50–2000
Arsenic	0–115
Copper	0.1–300
Iron	0.1–100
Lead	0–0.1
Molybdenum	0–4.7
Nickel	0.3–35
Zinc	13–740

from tens to thousands of parts per million. Table 2 shows that significant amounts of other metals may be dissolved when ores containing them are leached with cyanide solutions.

Chemical analyses of process solutions and wastewater derived from the processing indicate that most of the cyanide in solution is chemically linked with metals other than the small amounts of gold or silver. When chemical elements combine in solution to form soluble species, chemists refer to them as "complexes." There is a wide range of chemical and physical interactions between the components of complexes. Some complexes are very stable, whereas others are easily destroyed. Analytical chemists are able to define the relative stability of cyanide complexes of different metals with great precision. The evaluation of the quantity and types of cyanide is important to all aspects of cyanide use. It is particularly important to be able to distinguish both accurately and precisely between the various cyanide compounds to ensure the selection of an effective detoxification methodology.

4 Scott, J. S., *Status of Gold Mill Waste Effluent Treatment*, Report to CANMET, Natural Resources Canada, March 1993.

5 In environmental studies, concentrations of cyanide and other solutes in solutions are ordinarily presented in terms of mass per unit volume, or sometimes as the dimensionless unit "part per million" (ppm). Concentrations in milligrams per litre ($mg.L^{-1}$) are the same as concentrations in grams per cubic metre ($g.m^{-3}$), and both of these are essentially identical to concentrations in ppm (because the density of solutions is usually very close to 1 kilogram per litre [$kg.L^{-1}$]).

Weak and Strong Cyanide Complexes

Conventionally, cyanide chemists distinguish “weak” from “strong” cyanide complexes. The weak cyanide complexes, often referred to as “weak acid dissociable” or WAD cyanide, can dissociate in solution to produce environmentally significant concentrations of free cyanide. The weak complexes include cyanide complexes of cadmium, copper, nickel, silver and zinc. The degree to which these complexes dissociate is dependent largely on the pH of the solution.

Strong cyanide complexes, on the other hand, degrade much more slowly than WAD cyanide under normal chemical and physical conditions. Complexes of cyanide with gold, cobalt and iron are strong and stable in solution. This stability of the gold–cyanide complex is a key factor in the use of cyanide for the extraction of gold from ores. Once gold enters into solution tied to the cyanide, it remains complexed with the cyanide until process conditions are changed in order to remove it from solution. Cobalt is present only in trace amounts but iron is virtually ubiquitous in geological materials. For most mining situations, the strong complexes of cyanide are predominantly iron cyanides.

The rate at which complexes dissociate and release free cyanide into solution depends on several other factors, including the initial concentration of the cyanide complex, the temperature, the pH of the solution, and the intensity of light, especially ultraviolet radiation.

Analysing and Monitoring Cyanide

Cyanide is generally measured by one of two analytical methods: total cyanide analysis or WAD cyanide analysis. The first is used to determine total cyanide in solutions, including free cyanide and metal-bound cyanides, such as the more stable, non-toxic iron cyanides. The analytical procedure for determining WAD cyanide is used for free and complexed forms of cyanide, except iron cyanide. An older but still used alternative method to that of WAD cyanide analysis is called “cyanide amenable to chlorination.”

Cyanide analyses are needed for operational control, regulatory compliance and toxicity evaluation, as well as for public information about the handling of hazardous materials. Monitoring cyanide both during and after the gold recovery process is essential to good operating practice and the protection of both health and the environment. Rigorous sampling protocols and analytical procedures are required to ensure the quality of information available for decision making. This requires excellent planning and performance from trained personnel working with well-designed and well-managed systems.

SECTION 7

Attenuation of Cyanide Concentrations in the Environment

As explained in Section 4, once gold has been recovered, the solution becomes barren of gold but still contains cyanide. The processes that decrease the concentration of cyanide in solution, whether in the natural environment or in engineered facilities, are called "attenuation." Volatilization of HCN, which reduces the concentration of free cyanide in solution, is the prominent natural attenuation process. Figure 4 provides a schematic representation of the relationships between forms of cyanide and the processes controlling them.

Over the past two decades, the chemical and mining industries have made major advances in handling waste cyanide solutions so that they will not harm public health or the environment. Two technologies are used, often in combination: treatment and recycling.

Cyanide Solution Treatment and Re-use

Treatment: Four general forms of cyanide solution treatment are in use:

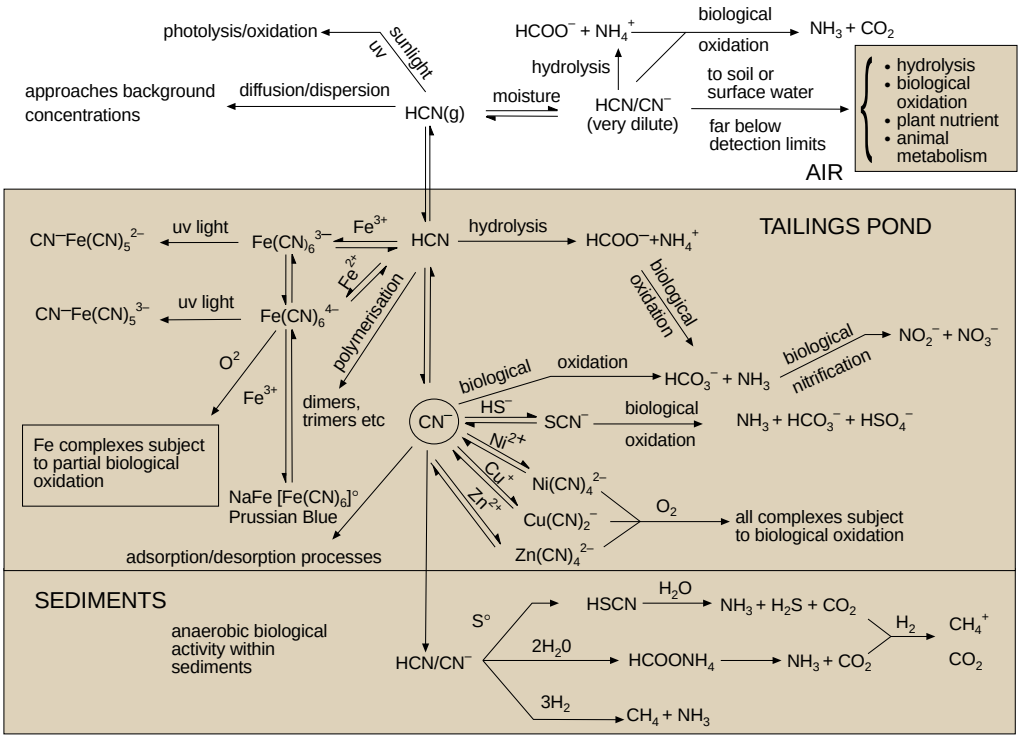
- Natural degradation
- Chemical oxidation
- Precipitation
- Biodegradation

In addition, several technologies enable the re-use of cyanide through recycling.

Natural degradation: The principal natural degradation mechanism is volatilization with subsequent atmospheric transformations to less toxic chemical substances. Other factors such as biological oxidation, precipitation and the effects of sunlight also contribute to cyanide degradation.

Cyanide species may be adsorbed on the surfaces of minerals or organic carbon debris in the soils of a pond embankment, in a clay liner, or along a groundwater flow path. In soils, bacteria assimilate the cyanide through a variety of aerobic and anaerobic reactions. In some instances, the combination of these processes of natural degradation are sufficient to meet regulatory requirements for discharge of cyanide-containing solutions.

FIGURE 4. The Cyanide Cycle



Source: Smith and Mudder, 1991. *Courtesy of Environment Australia*

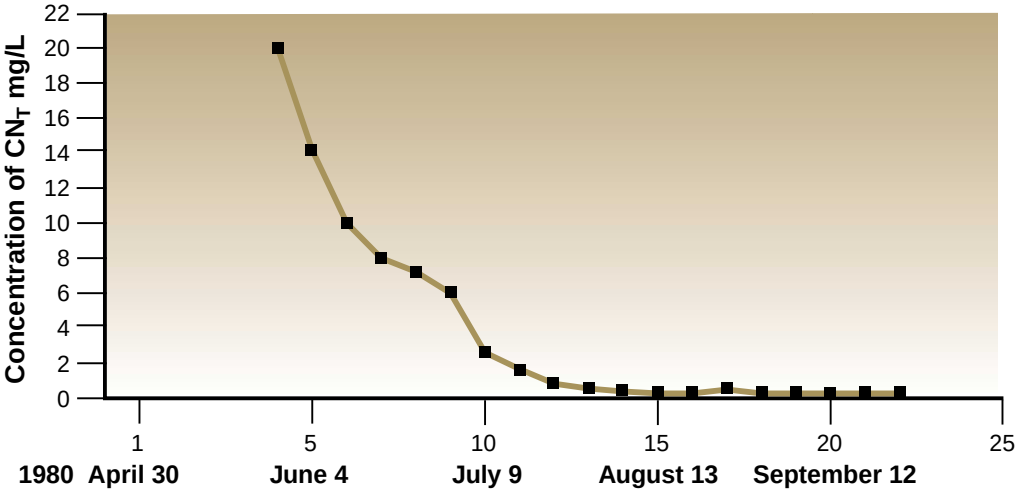
In tailings impoundments, the large surface area enables decomposition of WAD cyanide. Figure 5 illustrates a typical situation in which half of the total cyanide (CN_T) degraded naturally in less than three weeks from the initial concentration of 20 milligrams per litre. The CN_T disappeared almost completely within about 100 days.

Actual degradation rates need to be determined through test work on a site-specific basis using conditions that mimic, as closely as possible, the types of solution and the natural processes that are likely to occur at that location.

Table 3 compiles data from natural degradation systems at a number of gold mines around the world. The values in this table demonstrate the ability of natural degradation to reduce the cyanide concentration of solutions.

Chemical oxidation processes for cyanide treatment include the SO_2/Air process (developed by the Canadian mining company INCO) and the H_2O_2 (hydrogen peroxide)

FIGURE 5. Example of Cyanide Degradation in a Shallow Pond



Source: adapted from Schmidt et al., 1981.

treatment process (pioneered by Degussa). An older chemical oxidation alternative, the Alkaline Chlorination Process, is rarely used in the mining industry today.

In the SO_2/Air process, free and WAD cyanide are oxidized, and iron cyanide is precipitated as an insoluble solid. The process can be applied to either solutions or slurries, and reaction is rapid. Potential limitations are the need to obtain a licence to use the process,

TABLE 3. Natural Degradation of Cyanide in Tailings Impoundments

MINE	**CN entering the tailings system ($mg.L^{-1}$)**	**CN discharging from the tailings system ($mg.L^{-1}$)**
Lupin, NWT, Canada[a]	184	0.17
Holt McDermott, Ontario, Canada[a]	74.8	0.02
Cannon, Washington, USA[b]	284	<0.05
Ridgeway, South Carolina, USA[c]	480	0.09
Golden Cross, New Zealand[d]	6.8 (WAD CN)	0.33 (WAD CN)

Sources: a) Scott, 1993; b) Smith et al., 1985; c) Smith, 1987; d) Smith, 1994

the cost of building a processing plant, the need for empirical testing to optimize the system, and the inability of the process to oxidize intermediate by-products of cyanide.

Hydrogen peroxide, a strong oxidant, oxidizes free and WAD cyanide to ammonium and carbonate. Iron cyanides are not oxidized by peroxide, but precipitate as insoluble and stable solids. Sometimes it is necessary to add chemicals to control the copper concentration of solutions to meet environmental regulations. The peroxide system is not as well suited to the treatment of slurries because of irregular hydrogen peroxide requirements when solids are present.

Both methods of chemical oxidation are capable of producing residual concentrations of cyanide that can meet stringent discharge standards. Both processes require testing on representative samples of site-specific materials prior to the final plant design. Caro's acid, which combines sulphuric acid with hydrogen peroxide to form H_2SO_5, is also used as an oxidation agent to decompose cyanide in solution.

Precipitation of stable cyanides can be achieved by the deliberate addition of complexing agents such as iron. This reduces the free cyanide concentration and is also effective in controlling elevated levels of other metals that may be present. Iron cyanides may react with other chemicals in solution and produce solid precipitates, which may contain a dozen insoluble cyanide salts, thereby removing cyanide from solution. Some of the cyanide in process solutions will react with other chemical components within the system to form much less toxic concentrations of compounds such as ammonia, nitrate and carbon dioxide.

Biodegradation of cyanide is the basis for industrial wastewater treatment systems such as those used by Homestake Mining Company in the United States and ICI Bioproducts in the United Kingdom. A biological process has been used to treat cyanide to meet environmental discharge criteria for more than a decade at the Homestake Mine in Lead, South Dakota. Aerobic conditions are much more favourable to cyanide degradation than are anaerobic conditions, although anaerobic organisms can be effective in treating cyanide at concentrations of up to several milligrams per litre. Both active and passive biological treatment systems have been built—these systems remove cyanide using either aerobic or anaerobic micro-organisms.

At Homestake, the gold-mill barren solution is channelled through reaction vessels containing bacteria. These use oxygen from air to decompose cyanide compounds into nitrates, bicarbonates and sulfates. This microbial process is capable of oxidizing metal cyanide complexes, the metal ions from the WAD cyanide species and intermediate by-products of cyanide oxidation.

Advantages of the biological treatment process are its simple design and operational process control, low chemical costs and capacity of treating all forms of cyanide and its by-products. Potential limitations of biological treatment systems include reduced performance at cold temperatures and at very high cyanide concentrations.

Recycling: While the technologies for cyanide management have centred on cyanide destruction in single-pass systems, it is possible to recover and re-use cyanide, thus minimizing the total amount of cyanide used and reducing operational costs for some mines. Recycling lowers cyanide concentrations in waste solutions and decreases the cost of cyanide destruction.

Cyanide recovery and recycling has been used since the 1930s, notably at Flin Flon (Manitoba, Canada), Pachuca (Hidalgo, Mexico) and Golcanda Minerals (Tasmania, Australia). The basic process involves three steps: pH control, volatilization under highly controlled conditions, and capture of the cyanide that has been released. Recent engineering advances have made it a much more attractive prospect than was the case formerly, and cyanide recovery has been adapted in the last decade to treatment of slurries in a patented, commercial process called Cyanisorb. The process is being applied at the Golden Cross Mine (Waikato, New Zealand) and at the Delamar Silver Mine (Idaho, USA). Two additional Cyanisorb plants have recently been started up in Brazil and Argentina.

Research into cyanide recovery continues, including the testing of a treatment approach that separates cyanide complexes from solutions and absorbs them onto polystyrene-resin beads called Vitrokele (the Cyanosave process). Modifications of this process can be applied to either solutions or slurries, and both cyanide and metals can be recovered. The recovered cyanide is then recycled for use in the gold plant. While there have been successful tests of the process at mines in Canada, Australia and the USA, no commercial plant yet exists, and development continues.

SECTION 8

Evaluating and Managing Risks of Cyanide

The comprehensive approach to treating risk is made up of three key activities which occasionally overlap: risk assessment, risk management and risk communication. All three activities will be described in this and the following sections, beginning with risk assessment.

As stated already, it is well known that sodium cyanide and some of its derivatives are poisons and that cyanide compounds are classified as hazardous. Indeed, modern society safely utilizes many substances that are potentially hazardous, thanks to the ability to assess and manage the associated risks. Since the 1970s, it has become common practice to evaluate the risks associated with hazardous processes and materials through a systematic "risk assessment" process. Many of the concepts of risk assessment arose from more general methods developed by the insurance industry. These have their theoretical basis in probability and mathematical statistics. One of the key concepts that has carried over into environmental risk assessment is the fundamental definition of risk as the probability of a defined consequence.

Risk assessment consists of four parts:

1. Hazard identification is defined as the determination of the adverse effects which chemical, physical and biological agents have an inherent capacity or potential to cause to humans and the environment. Physical hazards include combustion, explosivity, flammability and corrosivity. Health hazards are categorized as acute (e.g. skin and eye irritation, lethal effects, asphyxiation) or chronic (e.g. carcinogenicity, sensitization, effects on reproductive system, effects on nervous system, effects on organs). Ecological hazards include mortality (acute) and reduced growth and reproduction (chronic) in representative species.

Hazard identification is only the first step in risk assessment. It is not an appropriate basis upon which to make a risk management decision. However, hazard identification is a critical step commonly carried out before chemicals and products are introduced to the market. In the case of human health and the environment, results of toxicity/ecotoxicity testing and epidemiology data are used to determine hazard.

2. Dose-response evaluation is the determination of the relationship between the magnitude of an administered, applied or internal dose and a specific biological response. The dose is the total amount of a substance administered to, ingested, inhaled or absorbed by an organism under standardized laboratory conditions used for toxicology testing. The end points of toxicity (or dose response) can be expressed as the measured or observed incidence, the percent response in groups of subjects (or population), or the probability of occurrence of a response in a population.

3. Exposure assessment is the evaluation of the pathways by which the hazard may contact a sensitive receiver. The receiver may be a single person, a real or hypothetical population, or a set of ecological recipients such as fish or wildfowl. The exposure assessment determines how and under what circumstances the receiver may be exposed to the hazard. It may also determine the quantities of the hazardous substance and the length of exposure.

4. Risk characterization summarizes the information from hazard identification, dose-response evaluation and exposure assessment into an overall conclusion on risk in a form that is useful to decision makers, legislators, the media and members of the public. Risk characterization provides a quantitative or qualitative description of the potential hazards of a particular exposure. Quantitative risk characterization conveys a numerical estimate of the magnitude of the risk that a substance poses to humans or to the environment. This risk may be expressed as individual risk or population risk. Qualitative risk characterization describes in narrative form the adverse effect or effects associated with exposure to an agent and provides some measure of the evidence for the association.[6]

Health and Environmental Impacts of Cyanide

Complete risk assessments require detailed specifications of the site-specific conditions. In the case of cyanide, its use varies so much that risk can be meaningfully evaluated only if the specific operating procedures at a particular site are considered. Nevertheless, it is possible to describe the hazards posed by cyanide and the potential exposures.

Toxicity and Epidemiology of Cyanide in Humans

Cyanide is a very fast-acting poison that is capable of killing a person within minutes if he or she is exposed to a sufficiently high dose. Humans may be exposed to cyanide by inhalation, ingestion or absorption through the skin. Cyanide prevents oxygen from being used by the cells, causing tissue hypoxia and “cyanosis” (a bluish discolouration of

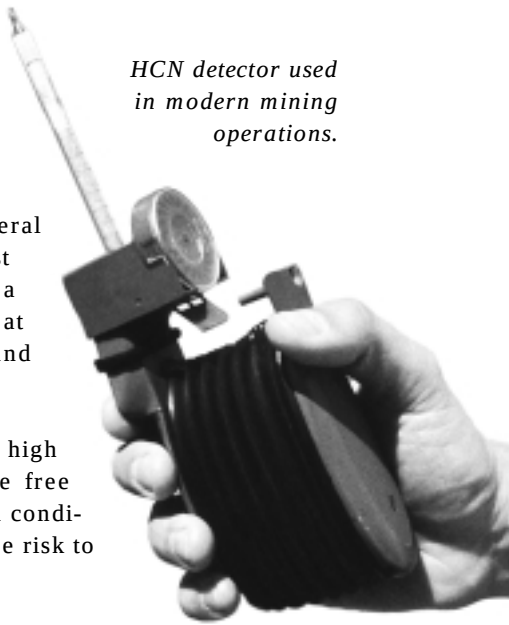
6 From George M. Gray, Jeffery, W. G. and Marchant. G. E., *Risk Assessment and Risk Management of Non-Ferrous Metals: Realizing the Benefits and Managing the Risks*, International Council on Metals and the Environment, 1997.

the skin). The respiratory system fails to nourish the cells with oxygen, a condition which, if untreated, causes rapid, deep breathing followed by convulsions, loss of consciousness and suffocation. The most common antidote is amyl nitrite, which may be taken orally or by injection.

Although there are many everyday sources of exposure to cyanide (automobile exhaust, tobacco smoke, fires), cyanide does not accumulate in tissues because the body transforms such small amounts into a less toxic compound called thiocyanate, which is then excreted. Cyanide is not known to cause cancer or birth defects or adversely affect reproduction.

The most toxic form of cyanide is HCN gas. The American Conference of Governmental Industrial Hygienists (ACGIH) lists the ceiling threshold limit of HCN at 4.7 ppm.[7] At concentrations of 20 to 40 ppm of HCN in air, some respiratory distress may be observed after several hours. Death occurs in minutes at HCN concentrations above approximately 250 ppm in air.

For free cyanide, the lethal dosage to humans by ingestion or inhalation ranges from 50 to 200 mg (1 to 3 mg of free cyanide per kg body mass). The lethal dosage for dermal absorption is considerably higher, at about 100 mg per kg of body weight.



HCN detector used in modern mining operations.

Photo courtesy of DuPont

Worker Exposure

Risk assessments address not only the impacts on the general population, but also the impacts on those who are most likely to be exposed to the hazard, such as the workers at a specific site. The potential for worker contact with cyanide at mines occurs during the receiving, unloading, handling and storage of solid sodium cyanide briquettes.

Provided that the cyanidation process is maintained at a high level of alkalinity (pH of 10.5 or above), almost all the free cyanide is present as CN^- in process solutions. Under such conditions, the volatility of HCN from solutions is low, so that the risk to workers through inhalation is manageable.[8]

7 1998 TLVs and BEIs—Threshold Limit Values for Chemical Substances and Physical Agents, published by the ACGIH.

8 Ingestion of process solution by workers (all of whom are trained and briefed on safety issues) is not considered a credible exposure pathway, because of the unlikelihood of anyone drinking such a solution.

Workers are required to wear respiratory protection against potential airborne hazards. Training in the fitting, use and testing of such equipment is incorporated into the company health and safety procedures. Most modern mining operations have HCN gas detectors or monitors that sound alarms in confined areas where HCN gas may be present. Most humans can detect the odour of hydrogen cyanide gas (a bitter almond smell) at concentrations below those that are hazardous to their health.

Environmental Toxicology and Impacts

Hazardous materials affect not only humans, but also ecological receptors. For mining environments, three groups of ecological or environmental receptors are of concern: mammals, reptiles and amphibians; birds (especially migratory wildfowl); and fish and other aquatic life.

There are few reports of major adverse impacts to animals from cyanide at mining sites. Sodium cyanide and cyanide-bearing solutions are handled in restricted areas of mining sites. Access by animals that walk or crawl is limited by walls, concrete pads, berms and fences, while the presence of humans around the mining facilities also deters animals from approaching. Government evaluations in the USA showed that standard containment designs and good engineering control have effectively mitigated threats to mammals, reptiles and amphibians.[9]

The principal concern for wildfowl has always been exposure to cyanide in open ponds, especially for migratory wildfowl passing through relatively arid regions such as the western USA, where use of cyanide in mining has become quite common. It should be noted, however, that the mortality of birds in Nevada due to exposure to cyanide solutions has been reduced dramatically from about 1,300 in 1990 to 220 in 1995, a decrease of 83%. This improvement is largely due to limiting the WAD cyanide concentration of uncovered ponds to less than 50 ppm. This concentration of WAD cyanide is not acutely toxic to ducks, which are shown to be very sensitive to cyanide as compared with other wildfowl and wildlife.

As a result of effective regulations and good management practice in mining, specific steps have been taken to further limit cyanide concentrations and exposure to wildfowl in open ponds. Netting has been useful in covering small process ponds, but netting of full-scale tailing impoundments is limited due to the weight of the nets, especially with accumulated snow or ice in cold climates, and due to the accidental trapping of wildlife in the nets. However, netting is still practised today for covering ponds in which the cyanide concentrations must be maintained at full strength for metallurgical purposes.

9 General Accounting Office (GAO), 1991.

Other methods of keeping birds away from cyanide solutions in ponds include the use of air cannons, noisemakers, plastic balls or other floating devices increasingly being used to cover the entire surface of small process ponds. This last method also aids in minimizing the loss of free cyanide due to volatilization.

Young, cold-water fish such as salmonids appear to be one of the aquatic species most sensitive to cyanide. Aquatic insects such as stoneflies, caddisflies, mayflies and beetles are generally less sensitive to the substance. It is the weak acid dissociable forms of cyanide that are considered the most "toxicologically significant." Laboratory and field studies have demonstrated that even sensitive aquatic species, such as trout, can tolerate low levels of WAD cyanide. Many discharge permits and regulatory standards are based upon WAD cyanide. In addition, site-specific standards for WAD cyanide have been promulgated for mining operations in such jurisdictions as the United States and New Zealand.

FPhoto courtesy of Placer Dome

Floating "bird balls" cover the surface of a solution containment pond at the Cortez gold mine, a Placer Dome–Kennecott joint venture in Nevada, USA.

SECTION 9

Risk Management for Cyanide in the Mining Industry

There are four major risk scenarios that need to be addressed through site-specific plans:

- Exposure of humans or ecological receptors to cyanide spilled during a transportation accident.
- Exposure of workers, particularly to HCN gas in enclosed areas.
- Exposure of humans through releases of cyanide in solution to surface or ground water that may be ingested.
- Exposure of ecological receptors, such as birds or fish, to cyanide-bearing solutions.

Transportation regulations and diligent safety programs limit the risks associated with the first scenario. As to the second, while adverse impacts from releases of process solutions have occurred in the past, scientific and engineering procedures exist to allow the safe and reliable operation of cyanidation processes. When site-specific standards relating to the third and fourth scenarios are set within the water-quality regulatory framework, protection of human health and the environment can be effectively realized.

Management Systems and Research and Development

Risk management in all of its aspects—from health and safety to prudent financial operations—is understood by today's mining industry to be an integral part of corporate management and a critical factor for the success of an industrial/commercial enterprise. Modern mining companies apply the generalized concept of "management systems" to their programs involving cyanide. Increasingly, this methodology is seen as part of good stewardship in mining, as in other industrial activities. Effective management systems involve four formal steps:

1. Plan: Written plans are prepared to detail the proper handling procedures and the accident response with respect to cyanide transportation and receiving, storage, solution

preparation, metallurgical processes and waste management. Such plans include spill and containment procedures at mining operations as well as health and safety procedures for protecting employees from the potential hazards of cyanide.

2. Execute: For a program to be effective, there must be a commitment to executing the written plans routinely and continuously at every operation. Additionally, each individual employee's responsibilities for executing and documenting the actions required by the plans must be spelled out in detail and clearly defined.

3. Review and document: Part of management's responsibility is to audit performance on a routine basis. The responsibility for reviewing and documenting performance is typically given to persons who are not part of the line operation and who report to a corporate level of authority. This ensures an independent evaluation of the performance of the system. It also ensures that the appropriate level of management in the company is informed about operational performance. The corporate authority may then review and effectively manage the potential risks by implementing policies and programs applicable to multiple sites.

4. Take corrective action, if necessary: Risk management programs may have deficiencies which subsequently become evident in the daily operations and processes. When these are identified in the review process, priority must be given to taking appropriate corrective actions, and the effects of those actions must be reviewed and documented in subsequent audits.

Product Stewardship

The most important aspect of a well-managed system is the understanding that the people in contact with cyanide must take responsibility for its safe use.

Cyanide producers audit purchasers and transportation systems. They also design special packaging for the transport of cyanide. The three primary producers of industrial cyanide, Degussa, Dupont and ICI, have all committed themselves to the principles of Responsible Care®.[10] Truck, rail and barge transporters screen their employees,

10 Responsible Care®, begun in 1985 by the Canadian Chemical Producers' Association (CCPA), is a new ethic for the safe and environmentally sound management of chemicals over their life cycle which has spread to over 40 countries around the world. Under this approach, the CEO or most senior executive of every member of CCPA and of most chemical associations throughout the world must commit to implement the guiding principles and codes of practice of Responsible Care® within three years of joining the association and must agree to submit to public verification. The expectations of members and partners in Responsible Care® go beyond the required implementation of the 151 management practices called for in three codes of practice to include CEO networking via leadership groups, public input through a national advisory panel, mutual assistance through sharing best practices, peer pressure under a conformance process and the public communication of performance improvement measurements.

carefully inventory packages, and establish and maintain systems for loading and unloading. The products are handled and transported according to protocols set by the respective industries and in compliance with national and international regulations.

Mining companies establish inventory control systems, maintain worker training and industrial hygiene programs, as well as build and maintain process-solution and waste-management systems that are specifically designed to mitigate and prevent exposure to cyanide. On a project-specific basis, all risk management components of good product stewardship must be integrated to achieve success.

Conservation and Recycling

Another component of good stewardship of cyanide products is the general concept of waste minimization. By reducing the amount of cyanide physically present at a mining site, the potential exposure pathways are inevitably reduced, and therefore, so is the total risk. Costs as well as risks are reduced when the amount of cyanide used in an operation is kept to the minimum level needed to achieve production goals. This objective requires approaches, such as value engineering, that help to

Photo courtesy of Degussa Corporation

Cyanide producers provide training to ensure safe transportation and handling of sodium cyanide.

Photo courtesy of FMC Corporation

An essential aspect of a well-managed system is that the people in contact with cyanide must take responsibility for its safe use.

conserve the total amount of cyanide used and consumed in a mining process. The advent of cyanide recycling processes provides mining projects with alternatives for conserving the total amount of cyanide required.

Regulations and Voluntary Programs Addressing Worker Safety and Public Health

Regulations, imposed most often by governments, attempt to enforce the management of risks. Examples of regulations in the cyanide life cycle include: (a) establishing packaging and transportation standards; (b) setting industrial hygiene standards for cyanide concentrations in the air and worker safety; and (c) establishing limitations on effluent discharge to surface and ground waters. Governments have used results from research and development and a public-policy process to establish procedures and standards that are protective of worker safety, public health and the environment.

Some examples of regulatory standards for cyanide to protect human health and the environment were given in Section 6. For example, the most toxic form of cyanide, hydrogen cyanide gas, is regulated by industrial hygiene standards such as the ACGIH standards of 4.7 ppm in air.

On a worldwide basis, the total cyanide limit for protection of human health generally is set near the United States Environmental Protection Agency proposed drinking water standard of 0.2 $mg.L^{-1}$. Also, there is an emerging international consensus, based on technical data, that WAD cyanide concentrations in open ponds should be maintained at concentrations of less than 50 $mg.L^{-1}$ to protect migratory birds and other waterfowl against adverse impact.

But the management of risks and its enforcement are not imposed by governments alone, nor need they be. Voluntary programs can have the same effect as regulations without the onus of legal coercion. For example, the major producers of cyanide compounds have made internal decisions to deal only with end users and transportation companies that have proven records of safe performance. While the methods used by each producer may differ, all have the same result of using market mechanisms requiring specific performance criteria to protect the general public from the hazards of cyanide.

SECTION 10

Risk Communication

Risk communication is a key component in any comprehensive program for properly addressing risks related to cyanide in the mining environment. Communication is required both within the operating plant and externally with the public. Internal company education and training of the managers and workers at a site is critical. Employees at a mine or any other industrial facility are also members of the public who live near the site. They and their families, friends and neighbours have many of the same concerns about the safe use of cyanide and about protection of the environment as anyone else living nearby. The proper communication of all cyanide information to the internal staff is therefore the first step in communicating the nature and extent of risk to the general public.

Photo courtesy of Placer Dome

Placer Dome's Sigma Mine, located in Val d'Or, Quebec, Canada.

Beyond complying with formal, regulatory requirements, effective risk communication involves public information and participation. In addition to coordinating emergency planning programs with the proper local authorities, it means giving access to data about the types and quantities of cyanide compounds in the mine's operational processes and inventory, as well as monitoring data. Effective public communication is also bi-directional, encouraging public concerns to be voiced and addressed.

Mine management practices with respect to cyanide should be made public and be implemented through programs which are explained to members of the local communities by company representatives who are effective communicators. Furthermore, positive community relations programs can provide substance as well as form, and serve to show the general population that cyanide and other hazards are being handled safely in the community. Today, a growing number of mining companies around the world have embraced this approach, opening the lines of communication with local communities to the greater benefit of all concerned.

SECTION 11
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About the Authors

Mark J. Logsdon, AB, MSc

Mark J. Logsdon is currently President and Principal Geochemist of Geochimica, Inc., a consulting firm located in Ojai, California, USA. He holds an AB Honors in Geology from Princeton University as well as an MS in Geology from the University of New Mexico. Mr. Logsdon specializes in hydrogeochemistry and environmental geochemistry, particularly related to waste management. In addition to consulting experience, Mr. Logsdon's 30-year career has encompassed research and academic instruction as well as governmental experience, notably with the USA's Nuclear Regulatory Commission and the New Mexico Bureau of Mines and Mineral Resources. Particular skills and experience include designing and implementing multidisciplinary earth science investigations involving waste characterization, geochemistry and hydrology. Geochimica projects include hydrogeology and hydrogeochemistry studies of acid mine drainage, cyanide hydrogeochemistry at mining sites and water-quality site investigations.

Karen Hagelstein, BS, MS, PhD, CIH

Karen Hagelstein is a Partner and Senior Environment Scientist at Times Limited, an environmental science and engineering firm located in Bozeman, Montana, USA, specializing in water and waste-water engineering, aquatic toxicology and human health evaluations. Dr. Hagelstein is a Certified Industrial Hygienist and received her BS from the University of South Dakota, her MS in Physiology and Biophysics from the University of Iowa, and her PhD in Civil and Environmental Engineering from the University of Iowa. During her career of over 15 years, Dr. Hagelstein has held positions as Senior Environmental Scientist, Environmental Engineer, and Health and Safety Officer with a number of consulting firms, and also spent six years as Associate Professor at the South Dakota School of Mines and Technology. Mining-related projects undertaken at Times Limited include reviewing and summarizing human toxicity data and risks with respect to environmental hazard exposures, air-dispersion modelling for estimating pollutant concentrations, and summarizing biological and chemical water quality monitoring data.

Terry I. Mudder, BS, MS, PhD

Terry I. Mudder is co-owner of Times Limited. He has a BS and MS in organic and analytical chemistry, and a PhD in environmental science and engineering. Dr. Mudder has 20 years' experience in the investigation of the chemistry, analysis, fate, aquatic toxicity and disposal of cyanide-bearing wastes. He has served as adjunct professor, thesis advisor and guest lecturer at universities throughout the world. He has worked on over 100 precious metal and non-ferrous mining-related projects on six continents and has written over three dozen technical papers. He has given numerous lectures and been involved with short courses and workshops on cyanide. He has co-authored several pamphlets and books, including *The Chemistry and Treatment of Cyanidation Wastes* and *The Cyanide Monograph*, both published by Mining Journal Books. He has been instrumental in the development and application of many of the chemical, physical and biological treatment processes for cyanide and metals, for which he has received both national and international awards, and obtained worldwide patents. He has provided technical assistance to the BC Ministry of Environment, Environment Australia, the Peruvian Environmental Protection Service, the US EPA, US State regulatory agencies, as well as to several industry-based organizations.

Chemistry and Treatment of Cyanidation Wastes

SECOND EDITION

Terry I. Mudder, Ph.D.
Michael M. Botz, M.S., P.E.
and
Adrian Smith, Ph.D.

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6.12 SULPHUR DIOXIDE AND AIR

6.12.1 Introduction

There are two patented versions of the sulphur dioxide cyanide destruction process. The first patented process and most widely applied is marketed by INCO Ltd. The INCO process is based upon conversion of WAD cyanides to cyanate using a mixture of SO_2 and air in the presence of a soluble copper catalyst at a controlled pH. In the INCO process, the forms of cyanide are removed by different processes. One process involves the conversion of WAD cyanides to cyanate. Iron complexed cyanides are reduced to the ferrous state and precipitated as insoluble copper-iron-cyanide complexes. Residual metals liberated from the WAD cyanide complexes are precipitated as their hydroxides.

The second sulphur dioxide process was developed at Heath Steel Mines Ltd. and the patent assigned to Noranda Incorporated (Ferguson and Walker, 1985). In the Noranda process, pure sulphur dioxide is fed into a solution or slurry to lower the pH into the range of 7.0 to 9.0. A copper sulphate solution is then added at such a rate to yield an effluent containing the desired cyanide concentration.

The INCO process has been used at over 80 mining operations worldwide and is the process addressed in this section. A primary application of the sulfur dioxide and air process is in treatment of tailings slurries, but it is also effective for the treatment of solutions for the oxidation of free and WAD cyanides.

6.12.2 Process Chemistry

Free and weakly complexed metal cyanides (i.e., WAD cyanides) are oxidized to cyanate by sulfur dioxide and air in the presence of a soluble copper catalyst.

(6.32) $$CN^- + SO_2 + O_2 + H_2O \xrightarrow{Cu^{+2}\ Catalyst} OCN^- + SO_4^{-2} + 2H^+$$

(6.33) $$M(CN)_4^{-2} + 4SO_2 + 4O_2 + 4H_2O \xrightarrow{Cu^{+2}\ Catalyst} 4OCN^- + 8H^+ + 4SO_4^{-2} + M^{+2}$$

The reaction is normally carried out at a pH of about 8.0 to 9.0, and due to the formation of acid in the reactions, lime is normally required for pH control. Decreases in process performance can occur if the pH fluctuates outside this optimal range. The optimal pH must be determined experimentally, since maximum cyanide and metals removals occur at different pH values. Temperature has little effect on process performance between 5°C and 60°C.

The theoretical usage of SO_2 in the process is 2.46 grams SO_2 per gram of WAD cyanide oxidized, but in practice the actual usage ranges from about 3.0 to 5.0 grams SO_2 per gram of WAD cyanide oxidized. The SO_2 required in the reaction can be supplied either as liquid sulphur dioxide, sodium sulphite (Na_2SO_3) or as sodium metabisulphite ($Na_2S_2O_5$). Solutions of ammonium bisulphite (NH_4HSO_3) have also been used in the process, but this requires a careful examination regarding the impact ammonia addition will have on the treated effluent. The choice of one reagent versus another is primarily associated with cost and availability.

The approximate lime requirement can be calculated from the above reactions according to the anticipated acid production resulting from SO_2 addition. Oxygen (O_2) is also required in the reaction and this is generally supplied by sparging atmospheric air into the reaction vessels. Required reaction times vary from about 30 minutes to 2 hours.

The soluble copper catalyst is normally added as a solution of copper sulphate pentahydrate ($CuSO_4$-$5H_2O$) to a level of about 10% to 20% of the initial WAD cyanide level. However, in cases where dissolved copper is already present in the tailings solution or slurry, the need for copper sulphate addition may be eliminated.

Iron cyanide removal is initiated by reduction of iron from the ferric to the ferrous state according to the following reaction:

(6.34) $2Fe(CN)_6^{-3} + SO_2 + 2H_2O \rightarrow 2Fe(CN)_6^{-4} + 4H^+ + SO_4^{-2}$

The ferrous cyanide complex is then removed through precipitation with either copper, nickel or zinc according to the following generalized reaction:

(6.35) $2M^{+2} + Fe(CN)_6^{-4} \rightarrow M_2Fe(CN)_6$ (solid)

Trace metals remaining in solution following oxidation of the weakly complexed metal cyanides are precipitated as their hydroxides according to the following generalized reaction:

(6.36) $M^{+2} + 2OH^- \rightarrow M(OH)_2$ (solid)

The oxidation of thiocyanate, which is usually limited to 10% to 20% in the process, and the hydrolysis of cyanate occur according to the following reactions:

(6.37) $SCN^- + 4SO_2 + 4O_2 + 5H_2O \rightarrow OCN^- + 10H^+ + 5SO_4^{-2}$

(6.38) $OCN^- + H^+ + 2H_2O \rightarrow HCO_3^- + NH_4^+$

Generally, the best application of this process is with slurries containing low to moderately high initial levels of cyanide when treated cyanide levels of less than about 5 mg/L are required. In some cases, solutions treated with this process may be of suitable quality to permit their discharge.

With regard to oxidant supply, sodium sulphite (Na_2SO_3) or sodium meta-bisulphite ($Na_2S_2O_5$) are supplied as powders and must be dissolved in concentrated form prior to use. As a result, a dissolution tank and a chemical storage and feed system are necessary. In the case sulphur dioxide is used, it is usually delivered as a bulk liquid or supplied in one-ton cylinders. Sulphur dioxide gas can also be generated on-site by burning pure sulphur and collecting the combustion products into an acidic solution using a scrubber tower. Exhaust gases can also be directly injected into the slurry or solution without intermediate scrubbing.

In a typical two-stage process configuration, the sulphur dioxide, lime and copper sulphate are introduced into the first stage to complete the oxidation of cyanide. Additional lime or other chemicals (e.g., ferric chloride) are added to the second reactor to maximize metals precipitation. This approach is needed in some instances since the complete oxidation of WAD cyanide requires a lower pH than does the precipitation of metals from the solution or slurry. The flowsheet for a typical two-stage process is shown on Figure 6.21.

The primary process variables include retention time, air feed rate, copper dosage, pH and sulphur dioxide feed rate. The quantity of sulphur dioxide or other reagent used is adjusted based on the WAD cyanide concentration in treated solution, and determined through laboratory and/or pilot plant evaluations. The copper requirement for the process is also determined experimentally. Generally, the copper dosage can be held to <50 mg/L, unless elevated iron concentrations are present which demand additional copper for iron cyanide precipitation. Laboratory evaluations of the process are generally conducted in one or two reaction vessels placed in series. Sulphur dioxide or another source of oxidant is added, either pre-mixed in air or separately as a sulphite solution. The tests are completed at various reagent dosages and pH values to determine the optimal reaction conditions and achievable level of treatment.

6.12.3 Performance

The performance of the INCO process at varying levels of copper addition and pH values is shown on Figure 6.22. As indicated, the process was found to be most effective with a copper concentration above about 10 mg/L and at a pH in the range of about 6.0 to 10.0. Summaries of the cyanide destruction performance and reagent usages achieved for the treatment of several tailings slurries using the INCO process are presented in Table 6.30. A summary of the cyanide destruction performance and reagent usages for the treatment of several barren solutions and decant waters are presented in Table 6.31, and process performances for treatment of three plating solutions are presented in Table 6.32.

Capital costs for the process depend upon whether a slurry or solution is being treated, the level of WAD cyanide, iron cyanide and the concentration of copper in the untreated material. The primary capital items include the reactor(s), agitator(s), an air compressor and piping, a feed system for the sulphur dioxide source (i.e., for either sulphur dioxide, sodium sulphite, or sodium metabisulphite), a copper sulphate storage and feed system, and a slaked lime preparation, storage and feed system.

Operating costs include labour, reagents, electrical power and maintenance. Lower treatment costs are associated with treatment of solutions containing low cyanide levels, while the higher costs are associated with treatment of tailings slurries and higher levels of cyanide. The INCO process is patented technology and does require a license and user fee.

The various advantages and disadvantages of the INCO sulphur dioxide process are presented in Table 6.33.

FIGURE 6.21 Flowsheet for a Two-Stage INCO Cyanide Destruction Process

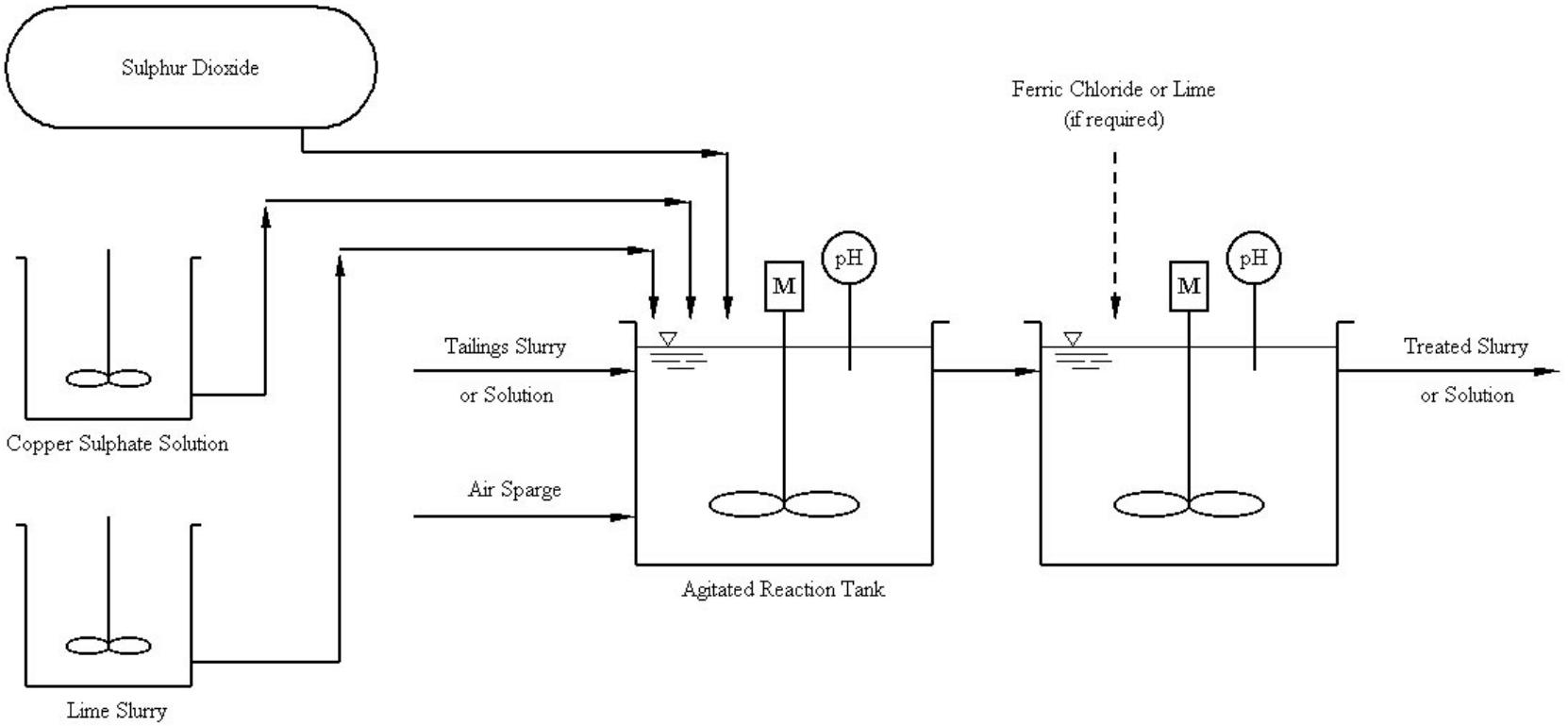
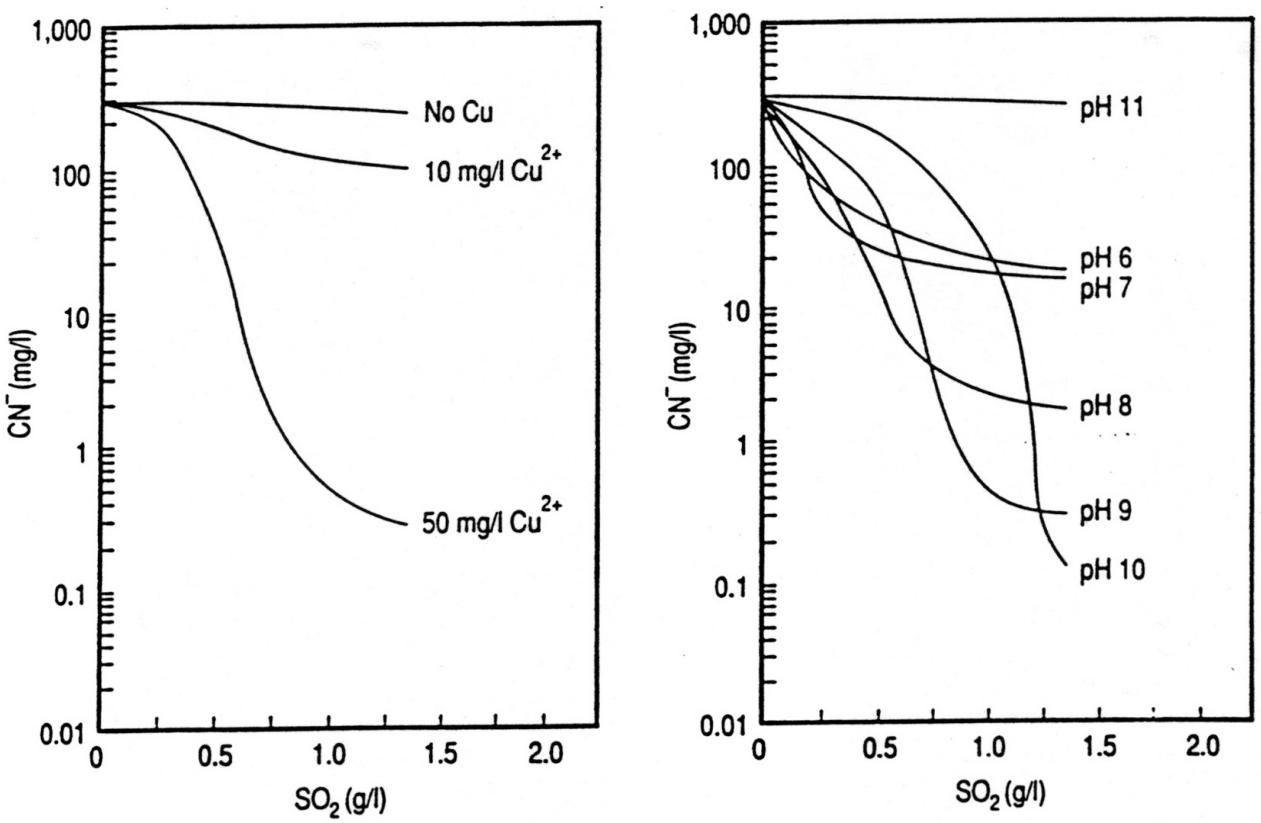


FIGURE 6.22 The Effects of Copper Concentration and pH on the Performance of the INCO Cyanide Destruction Process



Source: Robbins, 1996

TABLE 6.30 Oxidation of Cyanide in Tailings Slurry Using the INCO SO_2/Air Process

	CN_{TOT} Assay (mg/l)		Reagent Usage (g/g CH_{TOT})		
Mine	**Before**	**After**	**SO_2**	**Lime**	**Cu^{+2}**
Colosseum	364	0.4	4.6	0.12	0.04
Ketza River	150	5.0	6.0	0	0.30
Equity	175	2.3	3.4	0	0.03
Casa Berardi	150	1.0	4.5	--	0.10
Westmin Premier	150	<0.2	5.8	--	0.12
Golden Bear	205	0.3	2.8	--	--

Source: Devuyst et al., 1989a, 1989b and 1991

TABLE 6.31 Oxidation of Cyanide in Solutions Using the INCO SO_2/Air Process

	CN_{TOT} Assay (mg/l)		Reagent Usage (g/g CH_{TOT})		
Mine	**Before**	**After**	**SO_2**	**Lime**	**Cu^{+2}**
McBean (barren)	370	0.2	4.0	4.0	0
Lynngold (pond)	106	0.6	7.0	9.0	0.12
Mineral Hill (barren)	350	0.5	6.0	9.0	0
Lac Short (pond)	10	0.5	5.0	--	0
Citadel (barren)	350	5.0	4.0	--	0
St. Andrew (pond)	15	1	5.0	--	0.10

Source: Devuyst et al., 1989a, 1989b and 1991

TABLE 6.32 Oxidation of Cyanide in Electroplating Wastes Using the INCO SO_2/Air Process

	CN_{TOT} Assay (mg/l)		Reagent Usage (g/g CH_{TOT})		
Mine	**Before**	**After**	**SO_2**	**NaOH**	**Cu^{+2}**
Kuntz	150	0.2	6.0	--	--
Precious Plate	30,300	60	2.7	0.6	0
Superfinish	640	1.3	3.4	--	0.02

Source: Devuyst et al., 1989a, 1989b and 1991

TABLE 6.33 Advantages and Disadvantages of the INCO SO_2/Air Process

Advantages	
1	The process has been proven in numerous full-scale applications to yield low effluent cyanide and metals concentrations.
2	The process is effective in treating slurries as well as solutions.
3	The process is suitable for batch or continuous treatment.
4	All forms of cyanide are removed from solution, including the stable iron cyanide complexes.
5	Capital and operating costs are comparable with other chemical treatment processes.
Disadvantages	
1	If treating high levels of cyanide, the costs for reagents and electrical power can be high.
2	Cyanide is not recovered.
3	Undesirable levels of sulphate in the treated solution can result.
4	Additional treatment may be necessary for the removal of iron cyanide, thiocyanate, cyanate, ammonia, nitrate and/or metals for solutions to be discharged to the environment.