CYANIDE IN THE AQUATIC ENVIRONMENT

A Summary

by

Heather Sneddon

Environmental Protection Service

March, 1983

CYANIDE IN THE AQUATIC ENVIRONMENT

Cyanides in the environment may originate from industrial or biological sources. Cyanide containing compounds are widely used and produced industrially, and may be found in the effluents of such industries as mining, metal plating, petroluem refining, plastics manufacturing and chemical processing. Biological sources of cyanide include several fungi, algae and bacteria that produce hydrogen cyanide (HCN) via nitrogen metabolism, as well as certain terrestrial plants containing cyanogenic glycosides, which may potentially release HCN upon decomposition.

Cyanide compounds associated with the gold mining and milling industry are of particular importance regarding their potential impact on aquatic environments. The cyanide compounds of concern with respect to aquatic toxicology are those capable of releasing cyanide ion (CN^-) and hydrocyanic acid (HCN) in aqueous solution.

Terminology

The literature groups the various forms of cyanides pertinent to aquatic systems into categories described by the following terminologies: cyanide ion, molecular hydrogen cyanide, free cyanide, simple cyanide, complex cyanide and total cyanide.

Cyanide ions (CN^-) form complexes with various alkali, alkaline earth and heavy metals, particularly those metals of the transition series (eg. Fe, Co, Ni, Cu, Zn, etc.). The cyanide radical can exist in three oxidative states:

(i) as in HCN (-1), hydrocyanic acid
(ii) as in $(CN)_2$ (0), cyanogen gas
(iii) as in HOCN (+1), cyanic acid

Cyanide ions will hydrolyze readily with water to form hydrogen cyanide or hydrocyanic acid, HCN. Hydrogen cyanide is a very reactive and volatile compound, rarely found in natural environments. HCN is a weak acid, soluble in water, and is the most toxic component of free cyanide.

Cyanide compounds which in aqueous solution are in the form of cyanide ion or molecular HCN are termed "free cyanides". Certain simple and complex cyanides will dissociate to free cyanide, also described as the sum of $[CN^-]$ and $[HCN]$ in solution. The concentrations of cyanide ion and hydrogen cyanide at equillibrium depend on the pH of the aquatic solution.

$$CN^- + H_2O \rightleftarrows HCN + OH^-$$

If the pH is lowered by the addition of an acid, the reaction proceeds to the right and favors an increase in HCN concentration. However, if a base is added and the pH raised, the reaction shifts to the left in favour of increased cyanide ion concentration. The concentrations of cyanide ion and molecular HCN are equal at a pH of 9.36 (at 20°C), and at pH ≤ 8, 96% of the cyanide is in the form of HCN. Therefore, most of the free cyanide present in a natural aquatic system would be in the form of hydrogen cyanide.

Simple cyanide compounds include alkali, alkaline earth and heavy metal cyanide salts (eg. NaCN, CaCN, CuCN, respectively). Alkali salts such as NaCN, KCN and $Ca(CN)_3$ are very soluble in solution and are used as metallurgical reagents in gold recovery operations. Although a wide range of solubilities exists among the simple cyanides, many dissociate to some extent to release cyanide ions and thus may be classified as "free cyanide". The following metal salts, however, are insoluble: $Cu(CN)_2$, CuCN, HgCN, $Pb(CN)_2$ and $Ni(CN)_2$ (IEC, 1979).

Complex cyanides (eg. $K_3Fe(CN)_6$) include compounds which dissolve in water to release metal-cyanide complex ions (eg. $[Fe(CN)_6]^{-3}$), that may in turn dissociate further to liberate cyanide ions. Most metal cyanide ions are very stable and may be described as "fixed cyanides". However, some complexes such as tetracyanozincate (II) $[Zn(CN)_4]^{-2}$ and tetracyanocadminate (II) $[Cd(CN)_4]^{-2}$ dissociate almost completely in solution to form cyanide ions and consequently, HCN. Metallo-cyanide ions are commonly associated with the gold mining industry as by-products of gold recovery. The order of stability of some of these ion-complexes based on degree of dissociation in water to form free cyanide, is as follows:

$$[Mn(CN)_6]^{-3} < [Zn(CN)_4]^{-2} < [Cd(CN)_4]^{-2} < [Ag(CN)_2]^{-1}$$

$$< [Ni(CN)_4]^{-2} < [Cu(CN)_3]^{-2} < [Hg(CN)_4]^{-2} < [Fe(CN)_6]^{-4}$$

$$< [Fe(CN)_6]^{-3} < [Co(CN)_6]^{-3}$$

Total cyanide includes free cyanides as well as the simple and complex cyanides. It essentially refers to all forms of cyanide existing in aqueous solution. ⇒ ?

Thiocyanate
Cyanate

Methodology defined

Fate of Cyanides in the Aquatic Environment

Cyanide compounds do not tend to persist for extended periods of time in the aquatic environment, particularly in rivers and streams. Many physical, chemical and biological interactions may take place which favour the removal of cyanide from the water. The presence of dissolved carbon dioxide in water promotes a reaction with free cyanides to release hydrocyanic acid, HCN, which in turn may volatilize as a gas from the water surface.

$$CN^- + H_2O + CO_2 \rightleftarrows HCO_3^- + HCN$$

Cyanide ions may react with dissolved oxygen or oxygen at the water surface to form cyanate ions (OCN-). This reaction is irreversible and cyanate is usually then hydrolyzed to carbonate and ammonia. Cyanates are also formed when cyanides in waste waters are treated by alkaline chlorination.

$$1/2\ O_2 + CN^- \rightleftarrows OCN^-$$

Free cyanides may be hydrolized, but this reaction requires high activation energy and proceeds slowly.

$$CN^- + 3H_2O \rightleftarrows HCOO^- + NH_4^+ + OH$$

Cyanide ions may be adsorbed on suspended particles or bottom sediment, however, this process is reversible and some of the ions may be re-released slowly. Various fungi, bacteria and other micro-organisms found in aquatic environments are capable of utilizing cyanide as a source of carbon, nitrogen or both. This biological degradation process, which may occur under aerobic or anaerobic conditions, is irreversible and results in permanent removal of the cyanides involved. The bio-degradation process has been used to treat raw sewage wastes containing cyanides. Its potential use by the gold mining industry in the treatment of gold recovery effluents is still under investigation.

The fate of complex cyanides in the aquatic environment depends on the stability of the compound and the rate and extent of its dissociation to form free cyanides. As the free cyanides are removed, more of the complex cyanides will dissociate until equilibrium is reached. This process will continue until the cyanide source is depleted.

Ultraviolet radiation has been found to catalyze the dissociation of such stable complex cyanides as hexacyanoferrate II (ferrocyanide) and hexacyanoferrate III (ferricyanide). This photolysis reaction may occur in natural aquatic environments where exposure to UV radiation via sunlight is usually inevitable. Many factors govern the rate of photolysis in natural systems (eg. time of day, season, geographical location, morphology of water body, etc.) and the dissociation of the iron-cyanide complexes is dependent upon these conditions. Consequently, any free cyanides released would then be subject to removal by volatilization, oxidation and biological degradation.

Very little information exists pertaining to the biomagnification of cyanide compounds. Since low doses of cyanide are readily metabolized and detoxified, and high doses are lethal to most organisms, biomagnification is not expected to occur. A researcher at the University of British Columbia is currently investigating the possibility of thiocyanate biomagnification, however no conclusive information will be available until mid 1983.

Photolysis of Iron Cyanide Complex Ions

Cyanide ions react with iron to form the complex ions hexacyanoferrate II, $[Fe(CN)_6]^{-4}$ (ferrocyanide) and hexacyanoferrate III, $[Fe(CN)_6]^{-3}$ (ferricyanide). Ferrocyanide is the form most commonly found under normal environmental potential levels. Although these compounds are very stable and exhibit low solubilities, they may dissociate under the influence of ultraviolet light. The process, known as photolysis, begins with the oxidation of ferrocyanide to ferricyanide. The resulting ferricyanide is then photochemically oxidized to ferric hydroxide and free cyanide is released.

The photolytic degradation of hexacyanoferrate II and III is dependent upon such factors as water turbidity, intensity and angle of incidence of the light source. The penetration of light depends on the turbidity, colour and depth of the water body. Light penetration in turbid waters is generally low, however some suggest that photolysis reactions may be enhanced by suspended materials due to forward scattering of light. The photolysis rate in natural water columns decreases exponentially with depth, thus the photochemically active light is expected to penetrate only a short distance. Photodecomposition of iron-cyanides is a direct function of natural light intensity and wavelength at varying weather conditions. The angle of incidence of light varies with the time of day, season, geographical location, extent of cloud cover and ozone layer thickness.

Iron-cyanide photodecomposition may be quite rapid under certain environmental conditions, for example, in clear, shallow waters subjected to significant amounts of sunlight. The loss of HCN due to natural removal mechanism is thought to be considerably slower than HCN formation by photolysis of iron-cyanides at mid-day and near surface conditions (Broderius 1980). However, in deep, turbid or shaded waters where there is little photolytic decomposition, the HCN formed may be removed from the system as quickly as it is released. Ferrocyanides may re-complex to a small extent if photolysis is interrupted at an early stage by a period of darkness. There is no information that indicates whether or not this phenomenon occurs in the natural aquatic enviroment.

Laboratory research on photodecomposition performed by Broderius and Smith in 1977-78 revealed that 85%, by hexacyanoferrate II, and 49%, by hexacyanoferrate III, were the maximum amounts of cyanide that could be released as HCN from solutions prepared with each complex and deionized water. Therefore, for every mole of ferrocyanide and ferricyanide, each containing six moles of CN, five and three moles of CN, respectively, may be released as free cyanide by photolysis under laboratory conditions (Broderius 1980).

A computer program exists which enables prediction of the rate of photolysis and the concentration of free cyanide released by photodecomposition of iron-cyanides in a given water system. Calculations are made as a function of latitude, longitude, time of day and year and depth of water body. The photolysis rate may be predicted as a function of the iron-cyanide concentration and the measure and duration of illumination by sunlight (Zepp and Cline 1977). This program would be very beneficial for predicting the potential extent of free cyanide formation from iron-cyanide containing effluents released into the aquatic receiving environments adjacent gold milling operations.

Toxicity of Free Cyanide

Free cyanides combine in the tissues with enzymes associated with cellular oxidation. Cyanide inhibits cytochrome oxidase, the catalyst of the final oxidation step in cellular respiration. Oxygen becomes unavailable to the tissues and death from asphyxia results. Normal function may be restored upon removal of cyanide provided death has not yet occurred.

Hydrogen cyanide and cyanide ion are both toxic to aquatic organisms, molecular HCN being about 2.3 times more toxic than the cyanide ion (Doudoroff 1976). Complex cyanide ions in most cases exhibit low toxicity. The acute toxicity of simple and complex cyanides to fish is related to the concentration of molecular HCN released from the dissociation of these compounds.

The higher toxicity of molecular hydrogen cyanide in comparison to that of the cyanide ion involves the greater ability of the molecule to penetrate into the blood and tissues. Since the HCN molecule is smaller than the cyanide ion as well as being uncharged, it passes readily across membranes which may be impermeable or less permeable to the larger charged ions. However, once across that barrier, it is the cyanide ion that appears to be the active component in suppression of cellular respiration.

The toxicity of certain metal-cyanide ions generally depends on the stability of the compound and its ability to dissociate in solution to form free cyanides. For example, zinc and cadmium cyanide ions ($Zn(CN)_4]^{-2}$ and $[Cd(CN)_4]^{-2}$) are said to be very toxic in solution where they will dissociate almost completely to form cyanide ions and consequently, HCN molecules. However, silver cyanide ions $[Ag(CN)_2]^-$ and cuprocyanide ions $[Cu(CN)_2]^-$ as such have each been determined to be acutely toxic to fish when studied in alkaline solution with low HCN concentrations and negligible free silver and copper ions. These ions pass through the gills and into the tissues of the fish more readily than do other metal cyanide ions. The copper cyanide ion is more toxic than the silver cyanide ion and its penetration rate has been estimated to be four times that of $[Ag(CN)_2]^-$.
Cuprocyanide and silver cyanide ions both have been shown to produce symptoms in fish resembling those of heavy metal toxicity, such as coagulated mucous on gills and body surfaces (Doudoroff 1976). There appears to be no information pertaining to the chronic or sublethal effects of these compounds.

Aquatic toxicity information has generally been derived from the investigation of individual toxicants. Few studies have dealt with the effects of mixtures of toxicants on fish. Broderius and Smith (1979) demonstrated a less than additive joint toxicity of HCN with hexavalent chromium, and a greater than additive joint toxicity of HCN with zinc and with ammonia. The adverse effects of mixtures of toxicants cannot always be predicted by examining the individual toxicities. More research is required in order to determine the acute as well as sublethal effects of mixtures of cyanides and other compounds commonly found in waste waters.

Toxicity of Other Cyanide Compounds

Cyanogen chloride (CNCl) is a compound which is produced by adding free chlorine to cyanide or thiocyanate solutions. The formulation of CNCl is an intermediate step in the alkaline chlorination treatment of mine wastes. Doudoroff (1976) suggests that CNCl is as toxic if not more toxic than HCN. There is very little information on the toxicity of CNCl to aquatic organisms and what is available is not recent (ie. 1948).

Thiocyanates (SCN-) and cyanates (OCN-) are cyanide containing compounds which are found in the effluents of gold recovery operations. Cyanide combines with sulfur to form thiocyanate, a major by-product of gold ore processing. Cyanates are formed when cyanides in waste waters are subjected to the alkaline-chlorination system. Speyer (1981) performed 96 hour flow-through bioassays with rainbow trout, exposing the fish to solutions of sodium cyanide (NaCN), potassium cyanate (KCNO), and sodium thiocyanate (NaCNS). The resulting LC50's for cyanates and thiocyanates were 228 mg/l and 360 mg/l, respectively, indicating cyanates were slightly more toxic. The sodium cyanide solution was found to be 2000 times more toxic than the cyanate or thiocyanate solutions.

There is minimal information available on cyanate toxicity and what exists suggests that cyanates are toxic, but not to the same extent as hydrogen cyanide. In a 1976 literature review, Doudoroff found effects of thiocyanate toxicity in fish occurred at concentrations ranging from 29 to 5000 mg/l, however the studies sited were performed prior to 1960. Recent work at Montana State University (1982) demonstrated that thiocyanate toxicity is greatly influenced by stress. For example, thiocyanate concentrations that had no lethal effect on resting brook trout produced 100 percent mortality in trout subjected to forced exercise. Stress induced mortalities were almost instant and were found to be related to plasma thiocyanate levels.

Factors Influencing Cyanide Toxicity

Cyanide toxicity in fish has been found to vary with the life history stage. In general, the embryo appears to be the most tolerant, the fry and juvenile stages the least tolerant. Leduc (1978) suggests that a maximum acceptable toxicant concentration of 5 ug/l HCN should not be exceeded in waters bearing embryo and larval salmonids. Smith et al (1979) conclude that all life stages of fish should be able to tolerate concentrations of 5 ug/l HCN even under adverse environmental conditions.

Acute toxicity varies among different fish species. Cardwell et al (1976) exposed five species of fish to sodium cyanide solutions (essentially free cyanide) and found the order of sensitivity to be as follows: fathead minnows > bluegills > adult brook trout > channel catfish > juvenile goldfish.

Environmental factors such as temperature, dissolved oxygen content and pH of the water system may influence cyanide toxicity to fish. The information pertaining to the effect of temperature on cyanide toxicity is quite contradictory among the various researchers. Some studies suggest cyanide toxicity increases at higher temperatures, some suggest lower temperatures increase toxicity and yet others discovered temperature changes had no effect at all on certain fish species. The best conclusion may be that at low cyanide levels (slowly lethal concentrations) fish tend to survive longer at higher temperatures, however, at high cyanide levels (rapidly lethal concentrations) fish may survive longer at lower temperatures. Certain fish species (eg. goldfish) are able to metabolize anaerobically at lower temperatures for prolonged periods of time. Since cyanide poisoning induces anaerobic conditions in the tissues (hypoxia), this may explain why some fish appear to tolerate cyanide concentrations at lower temperatures.

Reduction in dissolved oxygen to low but not necessarily lethal concentrations has been demonstrated to decrease the tolerance of fish to cyanide. Doudoroff (1976) states that lethal threshold concentrations of cyanide decrease at low dissolved oxygen concentrations less than 5 mg/l.

The influence of pH on cyanide toxicity is dependent upon the concentrations of hydrogen cyanide and cyanide ions. Theoretically, as pH increases, acute toxicity should decrease due to the decrease in hydrogen cyanide concentration. The more alkaline pH levels favour an increase in cyanide ion concentration. However, Broderius et al (1977) found that when alkaline test pH's were increased from 8.3 to 9.3, an increase in toxicity to fathead minnows was noted. This increase was attributed to the cyanide ions penetrating the gill epithelium, although not to the same extent as the HCN molecules, thus augmenting the toxicity.

Acute and Sublethal Toxicity to Fish: A Summary

Doudoroff, in his 1979 review of the recent literature on cyanide toxicity, concluded that "median lethal threshold concentrations of HCN or of free cyanide as CN for freshwater fishes at different normal temperatures and pH values, and at dissolved oxygen concentrations above 5 mg/l are generally between 0.02 and 0.30 mg/l", that is 20 and 300 ug/l (Doudoroff, 1980, p.i.). Sublethal effects of free cyanide have been found at concentrations as low as 5 ug/l, however, levels below 10 ug/l have not often been examined. Adverse effects observed at the 10 ug/l level included impairment of swimming ability, effects on growth and body composition (eg. reduced growth, lowered fat content and increased water content), reduction in fecundity (eg. impairment of spawning, spermatogenis, egg production and viability), increased occurrence of developmental abnormalities, histopathological effects on liver and gonads, osmoregularity disturbances and changes in respiration rate.

Unfortunately, since most of the cyanide levels determined in natural water systems appear to be measured as total cyanide, it is very difficult to relate the toxicity information (measured as free cyanide or HCN) to the water quality data. Leduc (1981) reported that occasional high levels of cyanide (30-60 ug/l HCN) have been found in Canadian streams by the Prairie Provinces Water Board (1978) and the almost constant low levels of 5 ug/l appeared to exist in the waters surveyed. Additional background water quality data on levels of cyanide as HCN or free cyanide must be provided in order to properly assess the potential for cyanide toxicity in a given water system.

Several other factors must be considered when examining the acute and sublethal effects of cyanide. Most toxicity studies were performed under laboratory conditions and may not be realistic when applied to the natural environment. Toxicity data derived from flow-through bioassay procedures rather than static bioassays should be considered more reliable. Several researchers have noted that the loss of cyanide from static test solutions (ie. no renewal of solution) significantly influences the bioassay results by producing higher tolerance limits. More research is required to determine whether or not fish acclimate to low cyanide levels in waters, thereby increasing their resistance to more toxic concentrations. The effect of intermittent increases of cyanide levels (eg. due to seasonal peaks, sporadic effluent discharges, etc.) on aquatic organisms should be investigated. Additional studies are necessary to determine the effects of cyanide on aquatic invertebrates and plankton. The minimal amount of information in existance seems to indicate that certain invertebrate species are more tolerant than fish are to cyanide.

Guidelines

Health and Welfare Canada (1978) recommends a maximum acceptable concentration of 200 ug/l free cyanide in drinking water, with an objective concentration of less than or equal to 2 ug/l. The United States Environmental Protection Agency (1980) also advocates an ambient water quality criterion of 200 ug/l free cyanide for drinking water. The EPA suggests the criterion to protect freshwater aquatic life should be 3.5 ug/l free cyanide as a 24 hour average and the concentration should not exceed 52 ug/l free cyanide at any time.

The recent acute toxicity data seems to indicate a median lethal threshold concentration range of 20 to 300 ug/l (as HCN or free cyanide) for freshwater fishes under normal environmental conditions. Sublethal effects have been observed in various freshwater fish species at levels of 5 to 10 ug/l free cyanide and above. A concentration of 5 ug/l free cyanide is believed to ensure protection of the most sensitive fish life history stage, even under adverse environmental conditions. Therefore, a criterion of 3.5 ug/l free cyanide for freshwater aquatic life would appear to be a reasonable estimate. Aquatic invertebrates and plants, which tend to be more tolerant to cyanide than are freshwater fish, would also be protected.

Analytical Techniques

The Inland Waters Directorate - Water Quality Laboratory analyses water and wastewater samples for complex and simple cyanides using the Automated Pyridine - Barbituric Acid Method. Both complex and simple cyanides may be analyzed simultaneously. The complex cyanides are irradiated with ultraviolet light and converted to HCN. Analysis for complex cyanide essentially measures total cyanide. This value includes free and simple cyanide as well as complex cyanides such as thiocyanates and iron and cobalt cyanides. The simple cyanides are those which may be distilled from phosphoric acid solution without ultraviolet radiation. Simple cyanides may also be termed "weak acid dissociable" cyanides. The range of determination for both complex and simple cyanides is 0.50 to 50 ug/l (ppb) CN; the upper range may be extended by sample dilution. The detection limit is 0.50 ug/l (ppb).

The Environmental Protection Service Chemistry Laboratory analyses for "weak acid dissociable" cyanides (simple cyanides) and "total" cyanides using a manual procedure. Total cyanide analysis is able to measure the cyanide from metal cyanide complexes, but not from thiocyanate. The detection limit for both total and weak acid dissociable cyanide is 30 ug/l (ppb) CN.

According to the Can Test Laboratory in Vancouver (Maynard 1982), analysis for free cyanide (HCN + CN^-) in mine wastes or waters containing such, is virtually impossible. Analysis for free cyanide involves a simple titration with silver nitrate. This technique was originally developed to analyse metal plating wastes where complex cyanides are negligible. Mine wastes may include many substances which interfere with cyanide analysis (eg. thiocyanates) and the resulting levels may be unrealistically high. Can Test suggests analysing for "weak acid dissociable" cyanide (WAD) by a method which uses an acidic buffer in the digestion and thus does not break down all the cyanide complexes. The WAD cyanide values will be slightly higher than the actual free cyanide yet still provide a fairly accurate estimate of the toxic component.

The level of cyanide as free cyanide or molecular HCN in an aquatic receiving environment must be determined in order to assess the potential toxicity of the total cyanide input. Water quality data expressed as total cyanide is not toxicologically meaningful as it does not define the amount of free cyanide in the system. When free cyanide cannot be measured, water samples should be analysed for simple or "weak acid dissociable" cyanides.

Summary of Cyanide Levels

EPS Chemistry Lab detection limit	L30.00 ug/l CN
IWD Chemistry Lab detection limit	L0.50 ug/l CN
EPA Criterion for freshwater aquatic life	3.5 ug/l (free cyanide)
Level not to be exceeded	52 ug/l (free cyanide)
Sublethal effects in various freshwater fishes	$\geq$ 5-10 ug/l (free cyanide)
Medial lethal threshold concentrations for various freshwater fishes	20-300 ug/l (free cyanide)
EPA, Health and Welfare Canada water quality criterion for drinking water	200 ug/l (free cyanide)
Health and Welfare Canada objective for drinking water	$\leq$ 2 ug/l (free cyanide)

Bibliography

1. Broderius S.J. et al, "Relative Toxicity of Free Cyanide and Dissolved Sulfide Forms to the Fathead Minnow (Pimephales promelas)", J. Fish Res. Board Can., 34, 2323 (1977).

2. Broderius, S.J. and L. L. Smith Jr., "Lethal and Sublethal Effects of Binary Mixtures of Cyanide and Hexavalent Chromium, Zinc, or Ammonia to the Fathead Minnow (Pimphales promelas) and Rainbow Trout (Salmo gairdneri)", J. Fish Res. Board Can., 36, 164 (1979).

3. Broderius, S.J. and L.L. Smith Jr., "Direct Photolysis of Hexacyanoferrate Complexes. Proposed Applications to the Aquatic Environment", Ecol. Res. Ser., EPS - 600 13-80-003, United States Environmental Protection Agency, Duluth, Minnesota, ix + 49 pp (1980).

4. Brown, V.M., "The Calculation of the Acute Toxicity of Mixtures of Poisons to Rainbow Trout", Water Research, 2, 723 (1968).

5. Burdick, G.E. and M. Lipschuetz, "Toxicity of Ferro and Ferricyanide Solutions to Fish, and Determination of the Cause of Mortality", Trans. Amer. Fish. Soc., 78, 192 (1950).

6. Cairns, J. et al, "The Effects of Temperature Upon the Toxicity of Chemicals to Aquatic Organisms" Hydrobiologia, 47 (1), 135 (1975)

7. Cardwell, R.D. et al, "Acute Toxicity of Selected Toxicants to Six Species of Fish", Ecol. Res. Ser., EPA-600/3-76-008, United States Environmental Protection Agency, Duluth, Minnesota, vii + 117 pp. (1976).

8. Conn, K., "Cyanide Analysis in Mine Effluents", presented at the Cyanide and Gold Mining Industry Seminar, Environment Canada Technical Seminar, Ottawa, Ontario, January, 1981.

9. Doudoroff, P. et al, "Acute Toxicity to Fish of Solutions Containing Complex Metal Cyanides, in Relation to Concentrations of Molecular Hydrocyanic Acid", Trans. Amer. Fish. Soc., 95, 6 (1966).

10. Doudoroff, P., "Toxicity to Fish of Cyanides and Related Compounds. A Review", Ecol. Res. Ser. EPA-600/3-76-083, United States Environmental Protection Agency, Duluth, Minnesota, vii + 155 pp. (1976).

11. Doudoroff, P., "A Critical Review of Recent Literature on the Toxicity of Cyanides to Fish", American Petroleum Institute, Washington, D.C., iv + 71pp. (1980).

12. Dixon, D.G. and G. Leduc, "Chronic Cyanide Poisoning of Rainbow Trout and Its Effects on Growth, Respiration, and Liver, Histopathology", Arch. Environm. Contm. Toxicol., 10, 117 (1981).

13. Ecological Analysts, Inc., "Cyanide: An Overview and Analysis of the Literature on Chemistry, Fate, Toxicity, and Detection in Surface Waters", prepared for the Inter-Industry Cyanide Group, (1979).

14. Haliburton, D., "Discussion Paper on Water Pollution Control in the Primary Gold Mining and Milling Industry", Water Pollution Control Directorate, Environmental Protection Service, First Draft, 42 pp. (1980).

15. Health and Welfare Canada, "Guidelines for Canadian Drinking Water Quality 1978", Prepared by the Federal-Provincial Working Group on Drinking Water, 79 pp. (1978).

16. IEC International Environmental Consultants Ltd., "Factors Affecting Natural Degradation of Free and Metal-Complexed Cyanides From Gold Milling Effluents", Prepared for Fisheries and Environment Canada, Burlington, Ontario, Second Draft, 45 pp. (1979).

17. Ingles, J., "Iron Cyanide Chemistry in Relation to Mine - Mill Effluent Treatment", Water Pollution Control Directorate Environmental Protection Service, Draft Report, 47 pp. (1981).

18. Inland Waters Directorate, "Cyanide - Complex and Simple (Automated Pyridine-Barbituric Acid)", Unpublished Method of Analysis (1980).

19. Kimball, G.L. et al, "Chronic Toxicity of Hydrogen Cyanide to the Bluegill", Trans. Amer. Fish. Soc., 107(2), 341 (1978).

20. Koenst, W.M. et al, "Effect of Chronic Exposure of Brook Trout to Sublethal Concentrations of Hydrogen Cyanide", Environ. Sci. Tech., 11(9) 883 (1977).

21. Kovacs, T.G. and G. Leduc, "Acute Toxicity of Cyanide to Rainbow Trout (Salmo gairdneri) Acclimated at Different Temperatures", Can. J. Fish. Aquat. Sci., 39, 1426 (1982).

22. Kovacs, T.G. and G. Leduc, "Sublethal Toxicity of Cyanide to Rainbow Trout (Salmo gairdneri) at Different Temperatures", Can. J. Fish Aquat. Sci., 39, 1389 (1982).

23. Leduc, G. and K.S. Chan, "The Effects of Chronic Cyanide Poisoning on the Tolerance of Rainbow Trout to Varying Salinity", in Proc. 10th Canadian Symp. 1975: Water Pollution Research Canada, pp. 118-125, (1975).

24. Leduc, G., "Deleterious Effects of Cyanide on Early Life Stages of Atlantic Salmon (Salmo salar)", J. Fish. Res. Board. Can., 35, 166 (1978).

25. Leduc, G., "Ecotoxicology of Cyanides in Freshwater", in Cyanide in Biology, B. Vennesland et al, Eds., Academic Press, London, England, pp. 487-494, (1981).

26. Leduc, G. et al, "The Effects of Cyanides on Aquatic Organisms With Emphasis Upon Freshwater Fishes", National Research Council of Canada, Publication No. NRCC 19246, 139 pp. (1982).

27. Lind, D.L. et al, "Chronic Effects of Hydrogen Cyanide on the Fathead Minnow", J. Water Poll. Control Fed., 49(2), 262 (1977).

28. McCracken, T.R. and G. Leduc, "Allometric Growth Response of Exercised Rainbow Trout to Cyanide Poisoning", in Aquatic Toxicology J.G. Eaton et al, Eds., American Society for Testing and Materials, ASTM STP 707, Philadelphia, Pa., pp. 303-320 (1980).

29. Maynard, A.W., Can Test Ltd., Vancouver, B.C., in letter to Scottie Gold Mines Ltd., Vancouver, B.C., July, 1982.

30. Montana State University, Bozeman, "Metabolism and Toxicity of Thiocyanate in the Trout, Salmo gairdneri", Fisheries Bioassay Laboratory, Unpublished manuscript, (1982).

31. Oseid, D.M. et al, "The Effects of Hydrogen Cyanide on Asellus communis and Gammarus pseudolimnaeus and Changes in Their Competitive Response When Exposed Simultaneously", Bull. Environ. Contam. Toxicol., 21, 439 (1979).

32. Ruby, S.M. et al, "Inhibition of Spermatogenesis in Rainbow Trout During Chronic Cyanide Poisoning", Arch. Environm. Contam. Toxicol., 8, 533 (1979).

33. Sax, N.J., "Dangerous Properties of Industrial Materials", Fifth Ed., Van Nostrand Reinhold Co., New York, N.Y., p. 728 (1979).

34. Smith, L.L. et al, "Acute Toxicity of Hydrogen Cyanide to Freshwater Fishes", Arch. Environm. Contam. Toxicol., 7, 325 (1978).

35. Smith, L.L. et al, "Acute and Chronic Toxicity of HCN to Fish and Invertebrates", Ecol. Res. Ser., EPA-600/3-79-009, United States Environmental Protection Agency, Duluth, Minnesota, xiv + 115 pp. (1979).

36. Smith, M.J. and A.G. Heath, "Acute Toxicity of Copper, Chromate, Zinc and Cyanide to Freshwater Fish: Effect of Different Temperatures", Bull. Environm. Contam. Toxicol., 22, 113 (1979).

37. Speyer, M.R., "Toxicity of Cyanide Compounds", Noranda Research Center, Process Technology Department, Pointe-Claire, Quebec, Unpublished data.

38. Tupper, D., Environmental Protection Service, Laboratory Services, Personal communication.

39. United States Environmental Protection Agency, "Ambient Water Quality Criteria for Cyanides", EPA-440/5-80-037, Office of Water Regulations and Standards, U.S. Environmental Protection Agency, Washington, D.C., vi + 36 pp. (1980).

40. Zepp, R.G. and D.M. Cline, "Rates of Direct Photolysis in Aquatic Environment", Environ. Sci. Tech., 11(4), 359 (1977).