



# Canadian Soil Quality Guidelines for the Protection of Environmental and Human Health

## CYANIDE (FREE) 1997

This fact sheet provides Canadian soil quality guidelines for free cyanide for the protection of environmental and human health (Table 1). Supporting scientific documents are also available (Environment Canada 1996; Health Canada 1996).

### Background Information

Cyanides make up a distinct group of compounds characterized by the presence of the group  $C\equiv N$ . Cyanide compounds may take many forms, including free cyanide, simple cyanides, complex cyanides (metallocyanides), and organic cyanides (nitriles). Free cyanide refers to the sum of molecular HCN and the cyanide anion,  $CN^-$ . Chemical names for HCN include hydrogen cyanide, hydrocyanic acid, and prussic acid. Hydrogen cyanide is a colourless, flammable liquid or gas. Gaseous hydrogen cyanide,

which rarely occurs in nature, is lighter than air and diffuses rapidly. Hydrogen cyanide is a weak acid and remains largely in molecular form in aqueous solutions with a pH lower than 9.2. Above this pH, the molecule dissociates into  $H^+$  and  $CN^-$ . Hydrogen cyanide is completely miscible with water (Towill et al. 1978; Eisler 1991). Impure liquid hydrogen cyanide may polymerize spontaneously and violently, so small quantities of sulphuric or phosphoric acid are generally added as stabilisers (Towill et al. 1978).

Considerable quantities of cyanide compounds are consumed by a variety of Canadian industries, and the demand has been increasing steadily (CPI 1992; Statistics Canada 1992). In Canada, cyanide compounds are primarily used for the extraction/recovery of precious metals (mainly gold) and electroplating; for the production of organic chemicals, plastics, and other

**Table 1. Soil quality guidelines for free cyanide ( $mg \cdot kg^{-1}$ ).**

Guideline	Land use			
	Agricultural	Residential/ parkland	Commercial	Industrial
<b>Guideline</b>	<b>0.9<sup>a</sup></b>	<b>0.9<sup>a</sup></b>	<b>8.0<sup>a</sup></b>	<b>8.0<sup>a</sup></b>
$SQ_{HH}$	29	29	107	420
Limiting pathway for $SQ_{HH}$	Soil ingestion	Soil ingestion	Soil ingestion	Off-site migration
Provisional $SQ_{HH}$	NC <sup>b</sup>	NC <sup>b</sup>	NC <sup>b</sup>	NC <sup>b</sup>
Limiting pathway for provisional $SQ_{HH}$	ND	ND	ND	ND
$SQ_E$	0.9	0.9	8.0	8.0
Limiting pathway for $SQ_E$	Soil contact	Soil contact	Soil contact	Soil contact
Provisional $SQ_E$	NC <sup>c</sup>	NC <sup>c</sup>	NC <sup>c</sup>	NC <sup>c</sup>
Limiting pathway for provisional $SQ_E$	ND	ND	ND	ND
Interim soil quality criterion (CCME 1991)	0.5	10	100	100

**Notes:** NC = not calculated; ND = not determined;  $SQ_E$  = soil quality guideline for environmental health;  $SQ_{HH}$  = soil quality guideline for human health.

<sup>a</sup>Data are sufficient and adequate to calculate an  $SQ_{HH}$  and an  $SQ_E$ . Therefore the soil quality guideline is the lower of the two and represents a fully integrated de novo guideline for this land use, derived in accordance with the soil protocol (CCME 1996a). The corresponding interim soil quality criterion (CCME 1991) is superseded by the soil quality guideline.

<sup>b</sup>Because data are sufficient and adequate to calculate an  $SQ_{HH}$  for this land use, a provisional  $SQ_{HH}$  is not calculated.

<sup>c</sup>Because data are sufficient and adequate to calculate an  $SQ_E$  for this land use, a provisional  $SQ_E$  is not calculated.

The guidelines in this fact sheet are for general guidance only. Site-specific conditions should be considered in the application of these values. The values may be applied differently in various jurisdictions. The reader should consult the appropriate jurisdiction before application of the values.

synthetic materials; and for the synthesis of various inorganic compounds used by the electroplating industry (Montreal Engineering Company 1973; Scott 1989; CPI 1991, 1992; Eisler 1991).

Cyanide and cyanide compounds are present in air, water, soil, and food due to both natural and anthropogenic sources. Plants and other living organisms produce minute quantities of cyanide (Leduc 1984; Knowles 1988; Alström and Burns 1989; Davis 1991; Eisler 1991). Cyanogenic glycosides are widely distributed in more than 1000 species of food plants (notably cassava, peas, beans, and kernels of almonds) (Hulbert and Oehme 1968; Buck et al. 1973; Cade and Rubira 1982; Eisler 1991).

Although cyanide is ubiquitous in the environment, the highest environmental levels are found in the vicinity of combustion sources (automotive exhaust, fires, cigarette smoke, and solid waste incineration); in wastewaters from water treatment facilities, iron and steel plants, and organic chemicals industries; in landfills and associated groundwater; and in areas of road salt applications and runoff (Towill et al. 1978; Fiksel et al. 1981; ATSDR 1991).

### Environmental Fate and Behaviour in Soil

The major processes affecting the transport and distribution of cyanide in soils are volatilization and biodegradation. Cyanide ions may also form complexes with heavy metals, particularly iron, and precipitate out of solution (Lagas et al. 1982; Chatwin 1989;). Hydrogen cyanide is not susceptible to photolysis in soils (Cicerone and Zellner 1983), but complex cyanides, such as ferrocyanides and ferricyanides, may rapidly photodissociate and release free cyanide when exposed to sunlight (Callahan et al. 1979; Fiksel et al. 1981; Meeussen et al. 1992). Cyanides may be adsorbed by several materials, including clays and biological solids (Cruz et al. 1974; Raef et al. 1977a, 1977b; Chatwin and Trepanowski 1987; Chatwin 1989). However, existing data indicate that the rate of hydrogen cyanide and metal cyanide adsorption in soils is not significant when compared with rates of volatilization and biodegradation (Raef et al. 1977b; Callahan et al. 1979; ATSDR 1991). Small amounts of cyanide in soil may be oxidized to cyanate (HCNO) (Chatwin 1989). The high volatility of cyanide and the action of soil microbes ensure that high levels of cyanide do not persist or accumulate in soil under natural conditions (Towill et al. 1978; Fuller 1984).

As with surface waters, cyanide must be present as hydrogen cyanide in order to volatilize from soils (Higgs 1992). The rate of volatilization from soils is complex and depends on many factors, including pH, cyanide

solubility, hydrogen cyanide vapour pressure, free cyanide concentration, soil water content, soil sorptive properties, soil porosity, organic matter content, density and clay content, and atmospheric conditions such as barometric pressure, humidity, and temperature (Chatwin and Trepanowski 1987; Chatwin 1989). No quantitative data on the rate of cyanide volatilization from soils was available. Empirical studies on the partitioning of hydrogen cyanide between gas and solution phases in unsaturated soils showed that its migration through soil occurs mainly through gas diffusion. Hydrogen cyanide volatilization from unsaturated soils could account for up to 10% of total cyanide losses (Chatwin 1989). In acidic soils, volatilization becomes a significant removal process and may be the dominant mechanism for cyanide loss from soil surfaces (USEPA 1984; Rouse and Pyrih 1990).

Biodegradation, particularly in aerobic conditions, is expected to be an important cyanide process in soils (Towill et al. 1978). Cyanides may be degraded in the soil environment by a wide variety of microbes, including the fungi *Fusarium solani*, *Stemphylium loti*, and a *Pholiota* sp., and bacteria species such as *Corynebacterium*, *Arthrobacter*, *Bacillus*, *Thiobacillus*, *Pseudomonas*, *Klebsiella*, and *Escherichia* (Towill et al. 1978; Knowles 1988; Silva-Avalos et al. 1990). A strain of *Bacillus pumilus* from clay samples planted with flax was found to degrade a 0.1 mol·L<sup>-1</sup> cyanide solution to carbon dioxide and ammonia (Knowles 1976).

Natural soil microflora have been demonstrated to convert cyanide to carbonate and ammonia (Strobel 1967). Cyanide present at low concentrations will be decomposed to ammonia, carbon dioxide, and nitrogen or nitrate under aerobic conditions, and to the ammonium ion, nitrogen, thiocyanate, and carbon dioxide under anaerobic conditions (Rouse and Pyrih 1990).

The mobility of cyanide compounds in soil depends on stability and dissociation characteristics of the compound, soil type, soil permeability, soil chemistry, and the presence of aerobic and anaerobic microorganisms (Fuller 1984; Higgs 1992). Experimental studies on the mobility of cyanide in saturated anaerobic soils have shown that aqueous simple cyanides and aqueous ferricyanides tend to be very mobile. Cyanides dissolved in leachate were found to move through soils much more slowly than those in aqueous solution as they tended to precipitate out as the relatively immobile compound Prussian Blue (Alesii and Fuller 1976; Fuller 1977, 1984). It should be noted, however, that although Prussian Blue tends to precipitate out in soils with pH >4, some of the compound remains in solution and may result in contamination of groundwater by iron cyanide (Meeussen et al. 1992). Copper, cobalt, zinc, and nickel-cyanide complexes were found to be relatively mobile in soils compared to iron and manganese-

cyanide complexes (Chatwin 1989; Higgs 1992).

Soil conditions increasing the mobility of cyanide include low pH, high negative soil charges, and low clay content. Neutral to alkaline pH, high clay content, high positive soil charges, and the presence of organic matter and iron or other metal oxides appear to increase the attenuation of cyanide in soils (Alesii and Fuller 1976; Fuller 1977, 1984). The presence of aerobic soil microbes is particularly important to the attenuation of cyanide since mobility under aerobic conditions is greatly reduced due to higher rates of biodegradation (Fuller 1984). Thus, cyanide leaching to groundwater is enhanced under anaerobic conditions.

Soils represent the major potential pathway for cyanide contamination of groundwater (Chatwin 1989). High concentrations of cyanide in landfill waste or industrial effluents present a hazard to both soil and groundwater since microbial degradation of the compound may be inhibited (Lagas et al. 1982; ATSDR 1991).

## Behaviour and Effects in Biota

### Soil Microbial Processes

Bacteria exposed to cyanide may exhibit decreased growth, altered cell morphology, decreased motility, mutagenicity, and altered respiration (Towill et al. 1978). Cyanide's toxicity to living cells is a result of three major mechanisms: strong chelation to metals in metallo-enzymes; reaction with keto compounds to form cyanohydrin derivatives of enzyme substrates; and reaction with Schiff-base intermediates during enzymic reactions to form stable nitrile derivatives (Solomonson 1981; Knowles 1988). Cyanide is a major inhibitor of the enzyme cytochrome oxidase as well as hemoproteins and other metal-containing oxidases or oxygenases. At concentrations of about  $10^{-4}$  mol·L<sup>-1</sup> or lower, cyanide is usually highly inhibitory to cytochrome oxidase while other enzymes require  $10^{-4}$  to  $10^{-2}$  mol·L<sup>-1</sup> of cyanide for significant inhibition (Knowles 1976). Unacclimated mixed microbial populations are adversely affected by cyanide at concentrations of 0.3 mg HCN·kg<sup>-1</sup>. Acclimatized populations in activated sewage sludge may be unaffected by concentrations as high as 60 mg total cyanides·kg<sup>-1</sup> Towill et al. 1978).

### Terrestrial Plants

Very few data were available on the uptake of cyanide from soil by plants. Cyanide levels in cyanogenic plants are partially determined by nutrient availability, physical

stressors, and the growth stage of the plant (Buck et al. 1973; Cade and Rubira 1982; Eisler 1991). Consequently, cyanide concentrations in plants are difficult to correlate with levels in surrounding soil (Howe and Noble 1985). Dandelions, a non-cyanogenic plant, harvested from soils containing 11.3–16.2 mg·kg<sup>-1</sup> of cyanide showed cyanide levels of 10.25 to 11.30 mg·kg<sup>-1</sup>, while control plants from soils containing 0.70 mg·kg<sup>-1</sup> showed cyanide levels of 0.50 mg·kg<sup>-1</sup>. Laboratory tests demonstrated that dandelions grown in cyanide solutions and cyanide-containing mine effluents showed cyanide uptakes in proportion to the amount of cyanide in solution (Howe and Noble 1985). A BCF (ratio of cyanide in plant to cyanide in soil) of 0.8 can be calculated from the data on bush beans reported by Wallace et al. (1977).

The effects of cyanide on the seedling emergence of radishes (*Raphanus sativa*) and lettuce (*Lactuca sativa*) grown in an artificial soil were studied by Environment Canada (Environment Canada 1995a, 1995b). The average 3-d NOEC, LOEC, EC<sub>25</sub>, and EC<sub>50</sub> values for radish seedling emergence were 0.9, 1.9, 1.3, and 2.9 mg CN<sup>-</sup>·kg<sup>-1</sup> soil, respectively. The average 5-d NOEC, LOEC, EC<sub>25</sub>, and EC<sub>50</sub> values for lettuce seedling emergence were 5, 10, 7, and 13 mg CN<sup>-</sup>·kg<sup>-1</sup>, respectively.

### Terrestrial Invertebrates

Orchid weevils (*Orchidophilus aterrimus*) were resistant to cyanide at fumigating concentrations up to 4600 mg·L<sup>-1</sup> (Hansen et al. 1991). Southern armyworm (*Spodoptera eridania*) larvae were demonstrated to be extremely resistant to cyanide, with 3-day-old larvae showing an ingested LD<sub>50</sub> of 1492 mg HCN·kg<sup>-1</sup>, while levels of 23 mg CN<sup>-</sup>·kg<sup>-1</sup> soil resulted in significant earthworms mortality (Brattsten et al. 1983).

Soil invertebrate toxicity data, like toxicity data for soil microbes, are nearly nonexistent. In the only soil invertebrate toxicity study available, Environment Canada reported the effects of cyanide (applied as potassium cyanide) on the earthworm *Eisenia foetida* in an artificial soil. The average NOEC, LOEC, LC<sub>25</sub>, and LC<sub>50</sub> values were reported at concentrations of 8, 15, 9, and 12 mg CN<sup>-</sup>·kg<sup>-1</sup> soil, respectively (Environment Canada 1995a, 1995b).

### Livestock and Wildlife

Free cyanide is readily absorbed by terrestrial animals through inhalation, ingestion, and contact with skin and mucous membranes (Egekeze and Oehme 1980). The

most frequent cause of cyanide poisoning in terrestrial animals, particularly livestock, is through ingestion of plants containing cyanogenic glycosides. Free cyanide is released from cyanogenic plants through mastication, digestion, and microbial degradation in the digestive system (Towill et al. 1978). Animals that eat rapidly are at greatest risk (Egekeze and Oehme 1980). Ruminants (e.g., cattle and sheep) tend to be more vulnerable to cyanogenic plants than nonruminants (e.g., horses and pigs), presumably as a result of the greater degradation of plant cells by bacterial enzymes (Cade and Rubira 1982; Reed 1984). Cyanide poisoning through the ingestion of cyanogenic plants is more prevalent under drought conditions since animals are less selective in choice of forage and plant production of cyanogenic glycosides is enhanced under stressful conditions (Buck et al. 1973; Towill et al. 1978; Eisler 1991). In addition to livestock, several bird species have been found dead after ingesting cyanogenic plants (Cameron 1972).

LD<sub>50</sub>s for mammals and birds range from 1.43 to 11.15 mg CN<sup>-</sup>·kg<sup>-1</sup> bw. Detoxification is quite rapid. There is no evidence of cyanide bioaccumulation in any organisms. Low doses of cyanide are rapidly degraded to nontoxic products by most species, while large doses result in death (Towill et al. 1978).

### **Human and Experimental Animal Health Effects**

The toxicity of cyanide will vary according to the route of exposure. Inhalation is the most rapid route of entry and results in the rapid onset of toxic effects. Ingestion of soluble salts results in lower absorption via the gut and a faster detoxification. The chemical form of cyanide will also affect toxicity. Hydrogen cyanide is the most toxic cyanide form, whereas a complex cyanide compound such as acetonitrile requires metabolism to release free cyanide, and thus the toxic effects may be delayed by as much as 12 h (Ballantyne 1984).

The rate of absorption of cyanide across the gastrointestinal mucosa is dependent of the pH, pK<sub>a</sub>, and the liposolubility of the cyanide compound. The rapid lethal effects observed following oral intakes of cyanide indicates that cyanides are readily absorbed from the gastrointestinal tract (Gosselin et al. 1976).

Because of their liposolubility, cyanides can rapidly penetrate the epidermis. Corrosive properties (to the skin) of some cyanides (such as KCN) can increase the rate of absorption (NIOSH 1976). In one instance, a worker wearing a respirator suffered severe cyanide poisoning

within 5 min after spilling liquid HCN on his hands (Potter 1950).

Cyanides are rapidly absorbed following inhalation. In humans, a concentration of ≥2000 ppm is fatal in <1 min (Reiders 1971). Humans retain 58–77% of HCN in the lungs following inhalation (Landahl and Herrmann 1950).

Cyanides are distributed throughout the body following administration, and the detoxification process for cyanides occurs in various organs and tissues including nasal cavities, liver, and muscle (Dahl and Waruszewski 1989).

Gonzales and Sabatini (1989) described the mode of action of cyanide. It inhibits the final step of oxidative-phosphorylation of the cytochrome oxidase reaction by binding to cytochrome a-a<sub>3</sub> complex. Cyanide inhibits enzymes containing ferric iron and to a lesser extent enzymes containing ferrous iron. Iron is found in hemoglobin, and cyanide will react with hemoglobin to form cyanohemoglobin in small amounts. Death results from inhibition of cellular respiration.

Cyanide is extensively metabolized in the liver. The major pathway for detoxification of cyanide is via a mitochondrial enzyme, rhodanese. Cyanide is detoxified to form thiocyanate, which is excreted in the urine. The reaction forming thiocyanate is essentially irreversible (Way 1984). Estimates of the detoxification rates in humans vary from 0.5 μg·kg<sup>-1</sup> per minute to 170 μg·kg<sup>-1</sup> per minute (Bright and Marrs 1988).

Free cyanide is extremely toxic in acute doses via all routes of exposure. Most victims of acute cyanide poisoning (e.g., suicide attempts and accidental poisonings) die almost immediately. Fatal oral doses of cyanide compounds range from 0.5 to 3.5 mg CN<sup>-</sup>·kg<sup>-1</sup> bw, with most of the values between 0.5 and 1.0 mg·kg<sup>-1</sup> (USEPA 1992). A fatal dermal dose for HCN was estimated at 100 mg·kg<sup>-1</sup>. Inhalation of gaseous cyanide is expected to be immediately fatal at concentrations of 270 ppm HCN (300 μg·L<sup>-1</sup>) and fatal after 30 min exposure to concentrations of 135 ppm (150 μg·L<sup>-1</sup>) (Hartung 1981).

Symptoms of acute cyanide poisoning include vomiting, unconsciousness, coma, respiratory failure, and metabolic acidosis. The central nervous system is the most sensitive endpoint of cyanide toxicity, partly because of its high metabolic demands. Several diseases have been associated with the chronic ingestion of small doses of cyanide, including tobacco amblyopia, retrobulbar neuritis with pernicious anaemia, Leber's optic atrophy, Nigerian nutritional ataxic, neuropathy, and sterility in women who are heavy smokers.

No cancer studies in animals and no epidemiological studies with carcinogenicity in humans have been reported. Cyanides have tested negative for mutagenicity and effects on DNA synthesis in vitro, except in a study where a marginally mutagenic response for HCN in *Salmonella typhimurium* strain TA100 without metabolic activation was reported.

The general Canadian population is exposed to free cyanide in ambient air, drinking water, soil, and food. Because of lack of adequate data, it was not possible to directly characterize Canadian exposure to free cyanide via the diet. Estimates of total daily intake of free cyanide via air, water and soil (but excluding food) ranged from  $\geq 0.07 \mu\text{g}\cdot\text{kg}^{-1}$  bw per day in adults to  $\geq 0.11 \mu\text{g}\cdot\text{kg}^{-1}$  bw per day in infants. Air appeared to be a significant source of free cyanide exposure. Cyanide exposure in smokers is likely to be significantly higher (500×) than that of the general population.

The U.S. Environmental Protection Agency (IRIS 1994) has recommended a chronic oral reference dose of  $0.02 \text{ mg}\cdot\text{kg}^{-1}$  for CN<sup>-</sup> and HCN, based on the Howard and Hanzal (1955) study and using an uncertainty factor of 100 (10 for intraspecies variation, 10 for interspecies variation) and a modifying factor of 5 (for the apparent difference of tolerance to cyanide depending on the mode of ingestion [gavage, drinking water, or in food]). This reference dose is adopted provisionally as a TDI by the Bureau of Chemical Hazards of Health Canada for the derivation of health soil quality guidelines for free cyanide at contaminated sites in Canada.

There are no data in humans or experimental animals upon which to base a conclusion regarding the carcinogenic potential of free cyanide. It is therefore “unclassifiable with respect to carcinogenicity in humans” (Group VI.B) according to the classification scheme employed at the Bureau of Hazardous Chemicals of Health Canada in 1994. Free cyanide is treated as a substance for which the critical effect is believed to have a threshold of exposure for setting human health soil quality guidelines.

## **Guideline Derivation**

Canadian soil quality guidelines were derived for different land uses following the process outlined in CCME (1996a) using different receptors and exposure scenarios for each land use category. Detailed derivations for cyanide soil quality guidelines are provided in Environment Canada (1996) and Health Canada (1996).

## *Soil Quality Guidelines for Environmental Health*

Environmental soil quality guidelines (SQ<sub>ES</sub>) are based on soil contact using data from toxicity studies on plants and invertebrates. In the case of agricultural land use, soil and food ingestion toxicity data for mammalian and avian species are included. To provide a broader scope of protection, a nutrient and energy cycling check is calculated. For industrial land use, an off-site migration check is also calculated.

For all land uses, the preliminary soil contact value (also called threshold effects concentration [TEC] or effects concentration low [ECL], depending on the land use) is compared to the nutrient and energy cycling check. If the nutrient and energy cycling check is lower, the geometric mean of the preliminary soil contact value and nutrient energy cycling check is calculated as the soil quality guideline for soil contact. If the nutrient and energy cycling check is greater than the preliminary soil contact value, the preliminary soil contact value becomes the soil quality guideline for soil contact.

For agricultural land use, the lower of the soil quality guideline for soil contact and the soil and food ingestion guideline is recommended as the SQ<sub>ES</sub>.

For residential/parkland and commercial land uses, the soil quality guideline for soil contact is recommended as the SQ<sub>ES</sub>.

For industrial land use, the lower of the soil quality guideline for soil contact and the off-site migration check is recommended as the SQ<sub>ES</sub>.

In the case of cyanide, there are insufficient data to calculate the nutrient and energy cycling check. Therefore, the soil contact guidelines are recommended as the SQ<sub>ES</sub> for all land uses (Table 2).

## *Soil Quality Guidelines for Human Health*

The free cyanide soil concentration, based on direct exposure from soil ingestion, has been approved by the Standards and Guidelines Rulings Committee of the Bureau of Chemical Hazards of Health Canada as a preliminary human health soil quality guideline (SQ<sub>HH</sub>). However, the CCME recommends the application of various check mechanisms, when relevant, in order to provide a broader scope of protection. The lower of the soil ingestion guideline and any of the calculated checks is recommended as the SQ<sub>HH</sub>.

**Table 2. Soil quality guidelines and check values for free cyanide (mg·kg<sup>-1</sup>).**

Guideline	Land use			
	Agricultural	Residential/ parkland	Commercial	Industrial
	<b>0.9<sup>a</sup></b>	<b>0.9<sup>a</sup></b>	<b>8.0<sup>a</sup></b>	<b>8.0<sup>a</sup></b>
Human health guidelines/check values				
SQG <sub>HH</sub>	29 <sup>b</sup>	29 <sup>b</sup>	110 <sup>b</sup>	420 <sup>b</sup>
Soil ingestion guideline	29	29	110	2300
Inhalation of indoor air check	NC <sup>c</sup>	NC <sup>c</sup>	NC <sup>c</sup>	NC <sup>c</sup>
Off-site migration check	—	—	—	420
Groundwater check (drinking water)	NC <sup>d</sup>	NC <sup>d</sup>	NC <sup>d</sup>	NC <sup>d</sup>
Produce, meat, and milk check	NC <sup>e</sup>	NC <sup>e</sup>	—	—
Provisional SQG <sub>HH</sub>	NC <sup>f</sup>	NC <sup>f</sup>	NC <sup>f</sup>	NC <sup>f</sup>
Limiting pathway for provisional SQG <sub>HH</sub>	ND	ND	ND	ND
Environmental health guidelines/check values				
SQG <sub>E</sub>	0.9 <sup>g</sup>	0.9 <sup>g</sup>	8.0 <sup>g</sup>	8.0 <sup>g</sup>
Soil contact guideline	0.9	0.9	8.0	8.0
Soil and food ingestion guideline	11	—	—	—
Nutrient and energy cycling check	NC <sup>h</sup>	NC <sup>h</sup>	NC <sup>h</sup>	NC <sup>h</sup>
Off-site migration check	—	—	—	14
Groundwater check (aquatic life)	NC <sup>d</sup>	NC <sup>d</sup>	NC <sup>d</sup>	NC <sup>d</sup>
Provisional SQG <sub>E</sub>	NC <sup>i</sup>	NC <sup>i</sup>	NC <sup>i</sup>	NC <sup>i</sup>
Limiting pathway for provisional SQG <sub>E</sub>	ND	ND	ND	ND
Interim soil quality criterion (CCME 1991)	0.5	10	100	100

**Notes:** NC = not calculated; ND = not determined; SQG<sub>E</sub> = soil quality guideline for environmental health; SQG<sub>HH</sub> = soil quality guideline for human health. The dash indicates a guideline/check value that is not part of the exposure scenario for this land use and therefore is not calculated.

<sup>a</sup>Data are sufficient and adequate to calculate an SQG<sub>HH</sub> and an SQG<sub>E</sub>. Therefore the soil quality guideline is the lower of the two and represents a fully integrated de novo guideline for this land use, derived in accordance with the soil protocol (CCME 1996a). The corresponding interim soil quality criterion (CCME 1991) is superseded by the soil quality guideline.

<sup>b</sup>The SQG<sub>HH</sub> is the lowest of the human health guidelines and check values.

<sup>c</sup>Applies only to volatile organic compounds and is not calculated for free cyanide.

<sup>d</sup>Applies to organic compounds and is not calculated for free cyanide. Concerns about cyanide should be addressed on a site-specific basis.

<sup>e</sup>Applies to nonpolar organic compounds and is not calculated for free cyanide. Concerns about cyanide should be addressed on a site-specific basis.

<sup>f</sup>Because data are sufficient and adequate to calculate an SQG<sub>HH</sub> for this land use, a provisional SQG<sub>HH</sub> is not calculated.

<sup>g</sup>The SQG<sub>E</sub> is based on the soil contact guideline.

<sup>h</sup>Data are insufficient/inadequate to calculate the environmental nutrient and energy cycling check.

<sup>i</sup>Because data are sufficient and adequate to calculate an SQG<sub>E</sub> for this land use, a provisional SQG<sub>E</sub> is not calculated.

Therefore, the  $SQ_{G_{HH}}$ s for agricultural, residential/parkland, and commercial land uses are based on the soil ingestion guidelines. For industrial land use, the  $SQ_{G_{HH}}$  is based on the off-site migration check (Table 2).

## Soil Quality Guidelines for Free Cyanide

For each land use category, the soil quality guideline is the lower of the  $SQ_{G_{HH}}$  and  $SQ_{G_E}$ . For all land uses, the soil quality guideline is the soil concentration calculated for the  $SQ_{G_E}$ , which is based on the soil contact guideline (Table 1).

Because there are sufficient data to calculate an  $SQ_{G_{HH}}$  and an  $SQ_{G_E}$ , the soil quality guideline represents a fully integrated de novo guideline for each land use, derived according to the soil protocol (CCME 1996a). The interim soil quality criteria (CCME 1991) for cyanide are superseded by the soil quality guidelines.

CCME (1996b) provides guidance on potential modifications to the final recommended soil quality guidelines when setting site-specific objectives.

## References

- Alesii, B.A., and W.H. Fuller. 1976. The mobility of three cyanide forms in soils. In: Residual management by land disposal, Proceedings of the Hazardous Waste Research Symposium, February 2–4, 1976, Tucson, Arizona, W.H. Fuller, ed. EPA-600/9-76-015. U.S. Environmental Protection Agency, Cincinnati, OH.
- Alström, S., and R.G. Burns. 1989. Cyanide production by rhizobacteria as a possible mechanism of plant growth inhibition. *Biol. Fertil. Soils* 7:232–238.
- ATSDR (Agency for Toxic Substances and Disease Registry). 1991. Toxicological profile for cyanide. Draft for public comment. U.S. Department of Health and Human Services, Public Health Service.
- Ballantyne, B. 1984. Comparative toxicity of hydrogen cyanide and its salts. In: Proceedings of the Fourth Annual Chemical Defense Bioscience Review, 30 May–1 June, R.E. Linstrom, ed. U.S. Army Medical Research Institute of Chemical Defense, Aberdeen Proving Ground, Maryland.
- Brattsten, L.B., J.H. Samuelian, K.Y. Long, S.A. Kincaid, and C.K. Evans. 1983. Cyanide as a feeding stimulant for the southern armyworm, *Spodoptera eridania*. *Ecol. Entomol.* 8:125–132.
- Bright, J.E., and T.C. Marrs. 1988. Pharmacokinetics of intravenous potassium cyanide. *Hum. Toxicol.* 7(2):183–186.
- Buck, W.B., G.D. Osweiler, and G.A. Van Gelder. 1973. Cyanide. In: Clinical and diagnostic veterinary toxicology, G.A. Van Gelder, ed. Kendall/Hunt Publishing Company, Dubuque, IA.
- Cade, J.W., and R.J. Rubira. 1982. Cyanide poisoning of livestock by forage sorghums. *Agnote*, Government of Victoria, Department of Agriculture, Australia.
- Callahan, M.A., M.W. Slimak, N.W. Gabel, I.P. May, and C.F. Fowler. 1979. Water-related environmental fate of 129 priority pollutants. Vol. I. U.S. Environmental Protection Agency, Office of Water Planning and Standards, Office of Water and Waste Management, Washington, DC.
- Cameron, J.F. 1972. Natural substances suspected of killing birds in British Columbia. *Biol. Conserv.* 4(3):223.
- CCME (Canadian Council of Ministers of the Environment). 1991. Interim Canadian environmental quality criteria for contaminated sites. CCME, Winnipeg.
- . 1996a. A protocol for the derivation of environmental and human health soil quality guidelines. CCME, Winnipeg. [A summary of the protocol appears in Canadian environmental quality guidelines, Chapter 7, Canadian Council of Ministers of the Environment, 1999, Winnipeg.]
- . 1996b. Guidance manual for developing site-specific soil quality remediation objectives for contaminated sites in Canada. CCME, Winnipeg. [Reprinted in Canadian environmental quality guidelines, Chapter 7, Canadian Council of Ministers of the Environment, 1999, Winnipeg.]
- Chatwin, T.D. 1989. Cyanide attenuation/degradation in soil. Resource Recovery and Conservation Company ( $R^2C^2$ ), Salt Lake City, UT.
- Chatwin, T.D., and J.J. Trepanowski. 1987. Utilization of soils to mitigate cyanide releases. In: Proceedings of the 3rd Western Regional Conference on Precious Metals, Coal and the Environment, K.N. Han and C.A. Kliche, eds. Black Hills Section, A.I.M.E., Rapid City, SD.
- Cicerone, R.J., and R. Zellner. 1983. The atmospheric chemistry of hydrogen cyanide (HCN). *J. Geophys. Res.* 88(C15):10 689–10 696.
- CPI (Canadian Process Industries). 1991. Product profiles: Sodium cyanide. Corpus Information Services, Don Mills, ON.
- . 1992. Product profiles: Acrylonitrile. Corpus Information Services, Don Mills, ON.
- Cruz, M., A. Kaiser, P.G. Rouxhet, and J.J. Fripiat. 1974. Adsorption and transformation of HCN on the surface of copper and calcium montmorillonite. *Clays and Clay Minerals* 22:417–425.
- Dahl, A.R., and B.A. Waruszewski. 1989. Metabolism of organonitriles to cyanide by rat nasal tissue enzymes. *Xenobiotica* 19(11):1201–5.
- Davis, R.H. 1991. Cyanogens. In: Toxic substances in food plants, J.P. Felix D'Mello, C.M. Duffus, and J.H. Duffus, eds. The Royal Society of Chemistry, London.
- Egekeze, J.O., and F.W. Oehme. 1980. Cyanides and their toxicity: A literature review. *Vet. Q.* 2(2):104–114.
- Eisler, R. 1991. Cyanide hazards to fish, wildlife, and invertebrates: A synoptic review. Biological Report 85(1.23). Contaminant Hazard Reviews Report 23. U.S. Department of the Interior, Fish and Wildlife Service, Washington, DC.
- Environment Canada. 1995a. Toxicity testing of National Contaminated Sites Remediation Program priority substances for the development of soil quality criteria for contaminated sites. Environmental Conservation Service, Evaluation and Interpretation Branch, Guidelines Division, Ottawa. Unpub.
- . 1995b. Toxicity testing of National Contaminated Sites Remediation Program priority substances for the development of soil quality criteria for contaminated sites: Retesting volatile substances. Environmental Conservation Service, Evaluation and Interpretation Branch, Guidelines Division, Ottawa. Unpub.
- . 1996. Canadian soil quality guidelines for free cyanide: Environmental. Supporting document — Final draft. December 1996. Science Policy and Environmental Quality Branch, Guidelines Division, Ottawa.
- Fiksel, J., C. Cooper, A. Eschenroeder, M. Goyer, J. Perwak, K. Scow, R. Thomas, W. Tucker, and M. Wood. 1981. An exposure and risk assessment for cyanide EPA 440/4-85-008. U.S. Environmental Protection Agency, Office of Water Regulations and Standards, Monitoring and Data Support Division, Washington, DC.
- Fuller W.H. 1977. Movement of selected metals, asbestos, and cyanide in soil: Applications to waste disposal problems. EPA-600/2-77-020. U.S. Environmental Protection Agency, Office of Research and

- Development, Municipal Environmental Research Laboratory, Solid and Hazardous Waste Research Division, Cincinnati, OH.
- Fuller, W.H. 1984. Cyanides in the environment with particular attention to the soil. In: *Cyanide and the environment, Proceedings of a Conference, Tucson, Arizona, vol. 1:19–44.* Geotechnical Engineering Program, Colorado State University, Fort Collins, CO.
- Gonzales, J., and S. Sabatini. 1989. Cyanide poisoning: Pathophysiology and current approaches to therapy. *Int. J. Artif. Organs* 12(6):347–355.
- Gosselin, R.E., H.C. Hodge, R.P. Smith, and M.N. Gleason. 1976. *Clinical toxicology of commercial products.* 4th ed. Williams and Wilkins Co., Baltimore. (Cited in ATSDR 1989.)
- Hansen, J.D., A.H. Hara, H.T. Chan, Jr., and V.L. Tenbrink. 1991. Efficacy of hydrogen cyanide fumigation as a treatment for pests of Hawaiian cut flowers and foliage after harvest. *J. Econ. Entomol.* 84(2):532–536.
- Hartung, R. 1981. Cyanides and nitriles. In: *Patty's industrial hygiene and toxicology.* Vol. 10. John Wiley and Sons, New York.
- Health Canada. 1996. *Canadian soil quality guidelines for free cyanide: Human health. Draft.*
- Higgs, T.W. 1992. Technical guide for the environmental management of cyanide in mining. British Columbia Technical and Research Committee on Reclamation Cyanide Sub-Committee.
- Howard, J.W., and R.F. Hanzal. 1955. Chronic toxicity of rats of food treated with hydrogen cyanide. *J. Agric. Food Chem.* 3(4):325–329. (Cited in ATSDR 1989.)
- Howe, M., and D. Noble. 1985. Effect of cyanide residue on vegetation bordering a black hills stream. *Proc. S. D. Acad. Sci.* 64:112–122.
- Hulbert, L.D., and F.W. Oehme. 1968. *Plants poisonous to livestock: Selected plants of the United States and Canada of importance to veterinarians.* 3d ed. Kansas State University, Manhattan, KS.
- IRIS (Integrated Risk Information System). 1994. On-line data base. Vol. 23. Expires January 31, 1995.
- Knowles, C.J. 1976. Microorganisms and cyanide. *Bacteriol. Rev.* 40(3):652–680.
- . 1988. Cyanide utilization and degradation by microorganisms. In: *Cyanide compounds in biology,* D. Evered and S. Harnett, eds. CIBA Foundation Symposium 140. John Wiley, Chichester, England.
- Lagas, P., J.P.G. Loch, and K. Harmsen. 1982. The behaviour of cyanide in a landfill and the soil beneath it. In: *Effects of Waste Disposal on Groundwater and Surface Water,* R. Perry, ed. International Association of Hydrological Sciences, Publication 139:169–178.
- Landahl, H.D., and R.G. Herrmann. 1950. Retention of vapours and gases in the human nose and lung. *Arch. Ind. Hyg. Occup. Med.* 1:36–45. (Cited in ATSDR 1989.)
- Leduc, G. 1984. Cyanides in water: toxicological significance. In: *Aquatic toxicology,* Vol. 2, L.J. Weber, ed. Raven Press, New York.
- Meeussen, J.C.L., M.G. Keizer, W.H. van Riemsdijk, and F.A.M. de Haan. 1992. Dissolution behaviour of iron cyanide (Prussian Blue) in contaminated soils. *Environ. Sci. Technol.* 26(9):1832–1838.
- Montreal Engineering Company. 1973. *A preliminary survey of cyanide bearing liquid wastes in Canada.* Department of the Environment, Environmental Protection Service.
- NIOSH (National Institute for Occupational Safety and Health). 1976. *Occupational exposure to hydrogen cyanide and cyanide salts.* Department of Health, Education and Welfare, Public Health Service, Center for Disease Control, Rockville, MD. (Cited in ATSDR 1989.)
- Potter, A.L. 1950. The successful treatment of two recent cases of cyanide poisoning. *Br. J. Ind. Med.* 7:125–130. (Cited in ATSDR 1989.)
- Raef, S.F., W.G. Characklis, M.A. Kessick, and C.H. Ward. 1977a. Fate of cyanide and related compounds in aerobic microbial systems—I. Chemical reaction with substrate and physical removal. *Water Res.* 11:477–483.
- . 1977b. Fate of cyanide and related compounds in aerobic microbial systems—II. Microbial degradation. *Water Res.* 11:485–492.
- Reed, R.E. 1984. Cyanide compounds in plants and their effects on animals. In: *Cyanide and the environment, Proceedings of a Conference, Tucson, Arizona, vol. 1: 47–50.* Geotechnical Engineering Program, Colorado State University, Fort Collins, CO.
- Reiders, F. 1971. Noxious gases and vapours: I, Carbon monoxide, cyanides, methaemoglobin and sulfhemoglobin. In: *Drill's pharmacology in medicine,* 4th ed., J.R. DiPalma, ed. McGraw-Hill, New York.
- Rouse, J.V., and R.Z. Pyrih. 1990. Geochemical attenuation and natural biodegradation of cyanide compounds in the subsurface. In: *Symposium on environmental management for the 1990s,* D.J. Lootens, W.M. Greenslade, J.M. Barker, eds. Northwest Mining Association and Colorado Mining Association, Littleton, CO.
- Scott, J.S. 1989. An overview of gold mill effluent treatment. In: *Proceedings of the Gold Mining Effluent Treatment Seminars,* February 15–16, 1989 (Vancouver, British Columbia), and March 22–23, 1989 (Mississauga, Ontario).
- Silva-Avalos, J., M.G. Richmond, O. Nagappan, and D.A. Kunz. 1990. Degradation of the metal-cyano complex tetracyanonickelate(II) by cyanide-utilizing bacterial isolates. *Appl. Environ. Microbiol.* 56(12):3664–3670.
- Solomonson, L.P. 1981. Cyanide as a metabolic inhibitor. In: *Cyanide in biology,* B. Vennesland, E.E. Conn, C.J. Knowles, J. Westley, and F. Wissing, eds. Academic Press, New York.
- Statistics Canada. 1992. *Imports by commodity.* International Trade Division, Statistics Canada.
- Strobel, G.A. 1967. Cyanide utilization in soil. *Soil Sci.* 103(4):299–302.
- Towill, L.E., J.S. Drury, B.L. Whitfield, E.B. Lewis, E.L. Galyan, and A.S. Hammons. 1978. *Reviews of the environmental effects of pollutants: Part V, Cyanide.* EPA 600/1-78-027. U.S. Environmental Protection Agency, Office of Research and Development, Health Effects Research Laboratory, Cincinnati, OH.
- USEPA (U.S. Environmental Protection Agency). 1984. *Health effects assessment for cyanide.* EPA/540/1-86-011. U.S. Environmental Protection Agency, Office of Research and Development, Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH.
- . 1992. *Drinking water criteria document for cyanide.* Prepared by the Office of Health and Environmental Assessment, Cincinnati, OH.
- Wallace, A., J.W. Cha, and R.T. Mueller. 1977. Cyanide effects on transport of trace metals in plants. *Commun. Soil Sci. Plant Anal.* 8(9):709–712.
- Way, J.L. 1984. Cyanide intoxication and its mechanism of antagonism. *Ann. Rev. Pharmacol. Toxicol.* 24:451–481.

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Canadian Council of Ministers of the Environment. 1999. Canadian soil quality guidelines for the protection of environmental and human health: Cyanide (free) (1997). In: Canadian environmental quality guidelines, 1999, Canadian Council of Ministers of the Environment, Winnipeg.

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