



## Examples of Cyanide Compounds

cyanamide	cyanic acid, sodium salt	cyanogen
cyanates	cyanidol	cyanogen halides
cyanatotributylstannane	2-cyanoacetamide	cyanoacetic acid
	cyanotrimeprazine	

## SOURCES AND EMISSIONS

### A. Sources

Cyanide is used in pesticides and rodenticides, silver and metal polishes, photographic and electroplating solutions, metallurgical processes and fumigating products (Sax, 1989). Cyanide is also used in the manufacture of acrylonitrile, methyl methacrylate, adiponitrile, cyanide salts, cyanuric chloride, dyes, chelates, and hexamethylenediamine. It is also used in the production of synthetic fibers or plastics and cyanide salts for extracting metals, electroplating, and hardening of metals (HSDB, 1991).

The primary stationary sources that have reported emissions of cyanide compounds in California are manufacturers of screw machinery, nuts and bolts, mining of gold and silver ores, and manufacturers of guided missiles and space vehicles (ARB, 1997b).

Copper thiocyanate (cuprous thiocyanate) is registered for use in antifoulant paints for application to ships and boats for the control of aquatic organisms. Sodium cyanide is registered as an insecticide. It is used for the control of stored product insects. It is also registered for rangeland applications for the control of vertebrate predators (DPR, 1996).

The licensing and regulation of pesticides for sale and use in California are the responsibility of the Department of Pesticide Regulation (DPR). Information presented in this fact sheet regarding the permitted pesticidal uses of copper thiocyanate and sodium cyanide has been collected from pesticide labels registered for use in California and from DPR's pesticide databases. This information reflects pesticide use and permitted uses in California as of October 15, 1996. For further information regarding the pesticidal uses of these compounds, please contact the Pesticide Registration Branch of DPR (DPR, 1996).

### B. Emissions

The total emissions of cyanide compounds from stationary sources in California are estimated to be at least 1,100,000 pounds per year, based on data reported under the Air Toxics "Hot Spots" Program (AB 2588) (ARB, 1997b).

### C. Natural Occurrence

Some plants synthesize cyanoglucosides which upon decomposition form free cyanide. One example, amygdalin, is found in apricots, peaches, cherries, apples, pears, and similar fruit pits and in sweet almonds. Cyanide is released by the enzyme beta-glucosidase. Although these enzymes are not found in mammalian tissues, the human intestinal microflora possess enzymes capable of releasing cyanide resulting in human poisoning. Photosynthetic microorganisms among the blue-green algae can produce free cyanide in the process of nitrate metabolism (HSDB, 1991).

### **AMBIENT CONCENTRATIONS**

No Air Resources Board data exist for ambient measurements of cyanide compounds.

### **INDOOR SOURCES AND CONCENTRATIONS**

No information about the indoor sources and concentrations of cyanide was found in the readily-available literature.

### **ATMOSPHERIC PERSISTENCE**

Most cyanide in the atmosphere is expected to exist almost entirely as hydrogen cyanide gas, although small amounts of metal cyanides may be present as particulate matter in air. The calculated half-life and lifetime of hydrogen cyanide due to reaction with the hydroxyl (OH) radical are 0.9 years and 1.3 years, respectively (Atkinson, 1995). Hydrogen cyanide is expected to be resistant to direct photolysis. The relatively slow rate of degradation of hydrogen cyanide suggests that this compound has the potential to be transported over long distances before being removed by physical or chemical processes. Since hydrogen cyanide is miscible with water, wet deposition may also be an important removal process. Metal cyanide particles are expected to be removed from air by both wet and dry deposition (HSDB, 1991). The average half-life and lifetime for particles in the troposphere is estimated to be about 3.5 to 10 days and 5 to 15 days (Balkanski et al., 1993; Atkinson, 1995).

### **AB 2588 RISK ASSESSMENT INFORMATION**

The Office of Environmental Health Hazard Assessment reviews risk assessments submitted under the Air Toxics "Hot Spots" Program (AB 2588). Of the risk assessments reviewed as of December 1996, for non-cancer health effects, hydrogen cyanide contributed to a total chronic hazard index greater than 1 in 1 of the approximately 89 risk assessments (OEHHA, 1996b).

### **HEALTH EFFECTS**

Probable routes of human exposure to cyanide compounds are inhalation, ingestion, and dermal contact (HSDB, 1991).

Non-Cancer: The cyanide ion inhibits cytochrome oxidase impairing cellular respiration. Cyanide is very toxic to humans and inhalation exposure can be rapidly lethal. Long-term inhalation exposure may cause central nervous system effects (U.S. EPA, 1994a).

An acute non-cancer Reference Exposure Level (REL) of  $3.3 \times 10^3$  micrograms per cubic meter ( $\mu\text{g}/\text{m}^3$ ) and a chronic REL of  $70 \mu\text{g}/\text{m}^3$  are listed for hydrogen cyanide in the California Air Pollution Control Officers Association Air Toxics "Hot Spots" Program, Revised 1992 Risk Assessment Guidelines. The toxicological endpoints considered for chronic toxicity are the central or peripheral nervous systems, and the central nervous system for acute toxicity (CAPCOA, 1993).

The United States Environmental Protection Agency (U.S. EPA) has established a Reference Concentration (RfC) of  $3.0 \mu\text{g}/\text{m}^3$  for hydrogen cyanide based on central nervous system symptoms and thyroid effects in humans. The U.S. EPA estimates that consumption of this dose or less, over a lifetime, would not result in the occurrence of chronic, non-cancer effects. The U.S. EPA has established an oral Reference Dose (RfD) of 0.02 milligrams per kilogram per day for hydrogen cyanide based on weight loss, thyroid effects, and myelin degeneration in rats. The U.S. EPA estimates that consumption of this dose or less, over a lifetime, would not likely result in the occurrence of chronic non-cancer effects (U.S. EPA, 1994a).

No studies were found on adverse reproductive or developmental effects of cyanide in humans from inhalation exposure. Hypothyroidism in newborn babies related to the mother's ingestion of a cyanide-containing vegetable and iodine deficiency, has been indicated in results from human studies carried out in Africa. It has been suggested, based on results of animal studies, that oral exposure to cyanide may be associated with malformations in the fetus and low fetal body weights (U.S. EPA, 1994a).

Cancer: No information is available on the carcinogenic effects of cyanide in humans or animals (U.S. EPA, 1994a). The International Agency for Research on Cancer and the U.S. EPA have not classified cyanide with respect to potential carcinogenicity (IARC, 1994; U.S. EPA, 1994a).