# Potassium cyanide

CAS: 151-50-8
MF: KCN
FW: 65.12
Solubility: dissolves easily in water (1 M at 20°C). Partly soluble in ethanol.

**Major uses**
Cyanide (HCN, hydrogen cyanide) and its salts, such as potassium cyanide (KCN) and sodium cyanide (NaCN) are used in numerous of industrial processes, e.g. electroplating of metals, chemical synthesis, and extraction of gold or silver. KCN is used in photography; in plastic, paper and textile processing; for metal coating, cleaning or polishing etc. KCN can be also used as a fumigant in agriculture, and as a poison against rats and pests [1, 2].

HCN, under the name Zyklon B, was used as a genocidal agent by the Germans in World War II.

**Human toxicity**
Acute poisoning with HCN and KCN is dramatic, acute, and severe, and can produce death within minutes.

In the initial phase of cyanide poisoning, lower exposure doses may produce nausea, vomiting, abdominal pain, confusion, hyperventilation, anxiety, circulatory collapse, tachycardia, hypertension, headache etc. In the later phases, metabolic acidosis, seizures, pulmonary edema, apnea, convulsions, bradycardia, hypotension, coma, and death may occur. Death occurs mainly by cardiac arrest [3].

The fatal dose of KCN is estimated at 200 to 300 mg for an adult; however, there are cases when people survived ingestion of 1 g of KCN [3].

The median lethal dose of cyanide is approximately 2 mg/kg [4].

Whole blood cyanide levels about 3 mg/l and higher have been estimated fatal; coma occurred at the levels of 2.5 mg/l (sub-lethal cyanide level) [5]. In 32 fatal cases (post-mortem observations) a blood cyanide concentration range was from 0.4 to 230 mg/l, with an average of 37 mg/l [6].

Most of the cyanide found in the blood is in red cells, with a red cell:plasma ratio of at least 10:1 [5].

*Carcinogenicity*: not classified as a human carcinogen (IARC, 2004).

**Kinetic data**
*Absorption* is complete; cyanide is rapidly bound to serum albumin and hemoglobin [7, 8]. Human data are restricted because free cyanide ion ($CN^-$) is unstable in blood plasma [7].

*Kinetics* is biphasic [8]

*The volume of distribution ($V_d$)*: 0.41 l/kg (data from a single case of KCN poisoning) [1].

*Elimination kinetics* is biphasic: first phase takes 30 min to 1 h; second phase takes from 6 to 66 h [9].

*Time to peak* (ingestion) is less then 1 h (at the overdose situation) [8].

*Blood protein binding*: about 5% [8].

*Passage of blood-brain barrier*: free [8].

**Metabolism and excretion**

KCN is slowly decomposed by water and rapidly decomposed by acids, releasing highly toxic HCN gas.

In the body, cyanides mixed with water form so called "prussic acid" (hydrocyanic acid, HCN), which can rapidly penetrate mucous and cell membranes.

Cyanide is converted to thiocyanate by reaction with sodium thiosulphate, which occurs in the body in low concentrations [1, 7].

*Excretion*: about 80% of a cyanide dose is detoxified via the liver to thiocyanate, which is excreted in the urine [9].

**Toxicological mechanisms**

Cyanide prevents the cells of the body from getting oxygen, causing hypoxia (very low level of oxygen), or anoxia (lack of oxygen). When this happens, the cells die.

The mechanism of poisoning is as following: the $CN^-$ ion binds to the ferric iron ($Fe^{+++}$) of mitochondrial cytochrome oxidase. This results in inhibition of oxidative phosphorylation, anaerobic metabolism, lactic acid accumulation, and decreased ATP production. Cyanide is more harmful to the heart and brain than to other organs because the heart and brain use a great deal of oxygen [2, 5].

As all cyanides, KCN is a potent poison, inhibiting cytochrome oxidase and thereby the cells' respiration by forming a permanent bind with the iron atom in heme of cytochromes [8].

**Target organs**: CNS (histopathological lesions), heart, vascular system [8].

**References**

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