Acetaldehyde

CAS: 75-07-0
MF: CH_3CHO
MW: 44.05
Acetaldehyde is colorless, volatile liquid; it is flammable and highly reactive.
Solubility: miscible in water and most common solvents.

Major use
Acetaldehyde is used in the manufacture of a number of synthetic chemical products, including certain plastics, synthetic rubber, perfumes, aniline dyes, and polyester resins [1, 2].

Acetaldehyde can be produced by most hydrocarbon oxidation. It is present in tobacco smoke, where it is produced by oxidation of ethylene. It has also been detected in diesel and gasoline exhaust

See also the short descriptions for **paraldehyde** ($C_6H_{12}O_3$), which is acetaldehyde trimer, and for **ethanol** (C_2H_5OH).

Human toxicity
Acetaldehyde may cause toxicity if inhaled, ingested, or absorbed through the skin. It is a skin and mucous membrane irritant which causes a burning sensation of the nose, throat, and eyes.

Fatalities, following inhalation, are due to anesthesia when prompt and pulmonary edema when delayed. Very large exposure may cause death due to respiratory paralysis.

At poisoning, the following clinical effects have been observed: a) cardiovascular: tachycardia, and hypertension; b) respiratory: bronchitis, pulmonary edema, and respiratory paralysis; c) neurological: CNS depression, and narcosis; d) gastrointestinal: nausea, vomiting; d) hepatic: acetaldehyde can impair mitochondrial respiration in the liver, similar to effects seen with ethanol [2].

The minimum lethal human dose for acetaldehyde has not been delineated [2].

An acute oral dose of ethanol to volunteers produces blood acetaldehyde concentrations of 0.9-1.3 mg/l, whereas in chronic alcoholics, these levels may range from 1.7-2.5 mg/l. Following co-administration of ethanol and an inhibitor of acetaldehyde metabolism, such as disulfiram or calcium carbamide, acetaldehyde blood concentrations may increase 5-10 fold over normal level (reviewed in [1]).

The threshold limit value (TLV) is 25 ppm (45 mg/m^3) [2].

Carcinogenicity: EPA classification: rating B2 (probable human carcinogen; based on sufficient evidence of carcinogenicity in animals) {reviewed in [2]}.

Kinetic data
A little amount of data is available.

Absorption: acetaldehyde is rapidly absorbed and metabolized in humans.

Distribution: systemic poison; does not accumulate in the body.

Volume of distribution: unknown.

Passage of blood brain barrier: free (as a metabolite of ethanol).

Plasma protein binding: unknown.

Metabolism and excretion
The major portion of a dose of acetaldehyde is metabolized to acetic acid and then to carbon dioxide [1].

Acetaldehyde is a metabolic intermediate in humans. The main source of exposure to acetaldehyde in the general population is through metabolism of ethanol. Ethanol is primarily metabolized in liver by alcohol dehydrogenase to acetaldehyde and then to acetic acid [3].

Acetaldehyde is converted to acetyl coenzyme A, which is then oxidized through the citric acid cycle or utilized in various anabolic reactions involved in synthesis of cholesterol, fatty acids and other tissue constituents [4].

Excretion: via urine; certain part is excreted unchanged in the expired breath [1].

Toxicological mechanisms
The mechanisms are not well understood, but some hypothetical mechanisms have been proposed (reviewed in [Poisindex]):
1) Acetaldehyde causes sympathomimetic effects (stimulation of sympathetic nervous system) possibly due to release of norepinephrine from adrenergic nerve endings. Main signs of these effects are tachycardia, hypertension, and increased respiratory ventilation.
2) Acetaldehyde may inhibit mitochondrial oxygen consumption and energy production in rat liver *in vitro*.
3) Acetaldehyde can also deplete cellular stores of glutathione, which leads to lipid peroxidation.

Target organs: unspecified; systemic poison.

References
1. Baselt, R.C. & Cravey, R.H. (1995) *Disposition of Toxic Drugs and Chemicals in Man*. 4th ed., pp.1-2. Chemical Toxicology Institute, Foster City, California.
2. Poisindex, Thomson Micromedex (2006).
3. Hazardous Substances Data Base, HDSB (2006). Via Internet.
4. *Goodman and Gilman's The Pharmacological Basis of Therapeutics* (1990). Gilman, A.G., Rall, T.W., Nies, A.S., Taylor, P., eds., 8th ed., p. 379. New York, NY. Pergamon Press.

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