CHLOROACETIC ACID

($C_2H_3ClO_2$ or $ClCH_2COOH$) CAS #: 79-11-8 (Pesticide)
Synonyms include MCA; monochloroacetic acid;
alpha-Chloroacetic acid; acetic acid;
chloroethanoic acid; monochloroethanoic acid

SOURCE/USE

Chemical manufacturing produces chloroacetic acid. It is used as an herbicide and defoliant, as a bacteriostatic and preservative, as a treatment for plantar warts, as a drying agent for curing hay and as a chemical intermediate in production of several other chemicals. These include carboxymethylcellulose, ethyl chloroacetate, glycine, synthetic caffeine, sarcosine, thioglycolic acid, EDTA, the herbicides 2,4-D and 2,4,5-T and some vitamins.

There is no reported use, production, or disposal of chloroacetic acid at the Rocky Mountain Arsenal (RMA), however, it has been measured in soil and and infrequently in groundwater. Chloroacetic acid at RMA may likely be related to the manufacture of AKTON organophosphate insecticide produced by Shell Chemical Company during 1967. Chloroacetic acid is formed when one of the raw materials used in AKTON production, chloroacetyl chloride, hydrolyzes to chloroacetic acid in water. Chloroacetic acid can also form as a byproduct of mustard (Agent H) degradation products and decontamination materials (e.g., hypochlorite).

ROUTES OF EXPOSURE

Chloroacetic acid ("MCA") can be absorbed from inhalation, ingestion and (intact) skin exposure. Fatal human exposures have been recorded after ingestion (a five year old girl) and massive skin exposure (38 year old man with 25-30% body surface burn from MCA).

The risk of any off-post acute chloroacetic acid exposure due to the Rocky Mountain Arsenal remediation activity is very small, but any such exposure would likely be via inhalation. Also, the concentrations resulting in acute clinical effects discussed in this document reflect occupational exposures or animal studies and are much higher than those likely to be encountered at the fence line during the RMA remediation. Gastrointestinal absorption from swallowed airborne particles with absorbed MCA is a theoretical possibility. When pure, chloroacetic acid is colorless and crystalline, but may often appear as white, or light yellow to light brown crystals. The crystals will absorb moisture from the air and progress to a syrup. MCA has vinegar-like odor, which is detected at fairly low concentrations.

APPLICABLE STANDARDS AND LIMITS	
ATSDR acute MRL	Not Available
Occupational Standards	Not Available
Odor threshold	6 mg/m^3
RMA acute fence line criteria	ARC - X

APPLICABLE STANDARDS AND LIMITS	
	MARC - X
RMA chronic fence line criteria	Cancer - NA Noncancer - 7.0 µg/m^3

X - Acute criteria were not derived for this chemical because it is expected to occur infrequently at very low concentrations and to have low inhalation toxicity.

NA - Not applicable. Cancer criteria were not derived for this chemical because it is not considered a carcinogen or because a cancer slope value is not available.

The goal of the remediation is exposure prevention through remedial design, environmental monitoring, and modeling. Failure of prevention could result in acute and/or chronic exposures. Following is an overview of the types of health effects associated with chloroacetic acid exposure.

ACUTE HEALTH EFFECTS

High exposure to chloroacetic acid can produce central nervous system depression (narcosis) sufficient to produce respiratory depression. Seizures are reported from high exposures and, in fatal cases, cerebral edema is present. Animal data suggest possible neuropathy following exposure.

Inhalation of MCA can produce severe irritation to mucus membranes, including burns, and has produced pulmonary edema in severe exposures.

Chloroacetic acid is irritating to skin and eyes. High doses through dermal contact are corrosive and life threatening. Burns may occur from lesser dermal contact. Eye irritation occurs from vapors.

Ingestion of MCA is severely irritating and can produce burns to mouth, esophagus, stomach and duodenum. Animal experiments show liver damage and human exposure produces elevated liver enzymes in acute poisonings.

Animal and human experience indicates that chloroacetic acid can damage kidney tissue.

Acute poisonings with fatal outcome produced severe hypotension, cardiac dysrhythmias, severe electrolyte disturbances with hypokalemia and hypocalcemia, lysis of muscle cells, severe metabolic acidosis, renal tubular necrosis and fatty liver.

CHRONIC HEALTH EFFECTS

Chloroacetic acid produced tumors in mice in one study but not in others (oral exposure) and is classified as an equivocal tumorigenic agent in mice by RTECS criteria. It shows much more embryotoxicity than maternal toxicity in some animal studies. Cardiovascular malformations in offspring occurred in another study, but only at the highest dose where maternal toxicity was present. Mutation and evidence of chromosomal aberrations due to MCA have occurred in some test systems, but not in others, which indicates some potential for genotoxicity.

Long-term exposure to sufficient vapors may erode the enamel on teeth and possibly lead to perforation of the nasal septum, based on human experience with other acids.