DICYCLOPENTADIENE

($C_{10}H_{12}$) CAS #: 77-73-6 (Volatile Organic Compound)
Synonyms include DCPD; 1,3-CPD; bicyclopentadiene;
Cyclopentadiene dimer; alpha-dicyclopentadiene (endo isomer);
4,7-methano-1H-indene, 3a,4,7,7a-tetrahydro-; tricyclo(5,2,1,0)-3,8-decadiene

SOURCE/USE

Dicyclopentadiene (DCPD) must be created by chemical synthesis. It is not known to occur in nature. It is used as a chemical intermediate in the production of chlorinated hydrocarbon insecticides. It is also used as a flame retardant for plastics, as a hardener in linseed oil and in soybean oil, as a repellent for animals and in the production of plastics, elastomers, resins, varnishes and paints. Dicyclopentadiene is expected to be one of the more toxicologically important air contaminants found during remediation projects.

ROUTES OF EXPOSURE

Dicyclopentadiene can be absorbed through inhalation, ingestion or through the skin. The risk of acute DCPD exposure off-post due to the RMA remediation activity is very small, but in the event of any such exposure, inhalation would be the most likely route. 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 remediation at the RMA. DCPD can adsorb onto the surface of dust particles that may be swallowed as well as inhaled. Therefore, gastrointestinal absorption is possible.

DCPD is colorless and crystalline at room temperature and is a liquid above 90 degrees F. It possesses an odor that is described as sweet, sharp, disagreeable, and camphor-like. It is highly volatile at relevant ambient temperatures and is expected to volatilize when released into the environment. It has very low solubility in water. It may adsorb onto soil and suspended particulates that may slow its rate of volatilization.

APPLICABLE STANDARDS AND LIMITS	
ATSDR MRL	Not Available
Occupational standards	Not Available
Odor threshold	Not Available
RMA acute fence line criteria	ARC – 1.8 mg/m^3 MARC – 5.5 mg/m^3
RMA chronic fence line criteria	Cancer - NA Noncancer - 0.21 $\mu g/m^3$

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

ACUTE HEALTH EFFECTS

High levels of experimental exposure to DCPD in a variety of animals produces CNS depression with loss of coordination, convulsions and death which implies that sufficient exposure in man would produce similar toxicity. This was not reported in humans.

DCPD irritates mucous membranes and produces lung hemorrhage in experimental animals.

DCPD is an irritant to skin and eyes in experimental animals and causes mild eye irritation to eyes of human volunteers at an exposure of 1 ppm in air.

Ingestion of DCPD by experimental animals produces hemorrhage in stomach and intestine, and would be expected to cause GI irritation in humans if ingestion occurred. Liver toxicity was not reported.

DCPD causes hemorrhage in bladder and kidney of experimentally exposed animals.

DCPD causes hemorrhage in the thymus gland of acutely exposed experimental animals.

CHRONIC HEALTH EFFECTS

Chronic exposure to DCPD in experimental animals produced damage to kidneys, and caused damage to the bronchioles (bronchiectasis) as well as chronic pneumonia.

No data was found on carcinogenicity. DCPD is toxic to offspring in animal reproductive studies but no teratogenic effects were found. That is, toxicity in offspring with reduction in litter size and altered organ weights occurs at doses which maternal toxicity also occurred.