DIBROMOCHLOROPROPANE

($C_3H_5Br_2Cl$) CAS #96-12-8
Synonyms include DBCP, 1-Chloro-2, 3-dibromopropane
3-Chloro-1,2-dibromopropane, Nemagon,
Durham, Nematicode, Fumagon

SOURCE/USE

Dibromochloropropane (DBCP) must be created by chemical synthesis. It is used as a soil fumigant and nematocide. It has been banned from production or application in the United States since 1977, except for restricted use in pineapple horticulture in Hawaii. It is still used in other parts of the world. Dibromochloropropane is expected to be one of the more toxicologically important air contaminants found during remediation projects.

ROUTES OF EXPOSURE

DBCP is absorbed from inhalation, ingestion or skin exposure. All three routes have been considered important in workers exposed to DBCP during manufacture/packaging and during application.

Although the risk of any acute DBCP exposure off-post due to the Rocky Mountain Arsenal remediation activity is very small, 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 remediation at the RMA. DBCP can adsorb onto the surface of dust particles that may be swallowed as well as inhaled. Therefore, gastrointestinal absorption is possible. DBCP is a liquid, colorless when pure but in technical grade is yellow, amber or brown. It has a pungent odor and is highly volatile.

APPLICABLE STANDARDS AND LIMITS	
ATSDR MRL	Not Available
Occupational standards	Not Available
Odor threshold	Not Available
RMA acute fence line criteria	ARC - 0.33 mg/m^3 MARC – 0.98 mg/m^3
RMA chronic fence line criteria	Cancer – 4.0 $\mu g/m^3$ Noncancer – 0.2 $\mu g/m^3$

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 DBCP exposure.

ACUTE HEALTH EFFECTS

High levels of exposure produce CNS depression (rats) which implies that sufficient exposure in man would produce dizziness, confusion and eventually coma and death. CNS depression from DBCP was not observed in humans.

DBCP is a mild irritant of mucous membranes. Ingestion of DBCP may result in pulmonary edema.

DBCP is mildly irritating to skin, especially in repeated contact. DBCP is mildly irritating to the eye. It is not caustic to skin or eye.

Acute ingestion of DBCP may create GI distress. DBCP causes significant liver damage (cloudy swelling) in animal tests, but this was not seen in man.

DBCP causes kidney damage (nephritis) in animal testing and loss of sperm production with sterility in human male workers. This was not reversible in the most severely affected men.

CHRONIC HEALTH EFFECTS

Chronic exposure to DBCP (by any route) may be expected to produce damage to liver, kidney, bone

marrow (pancytopenia or loss of red and white blood cells and platelets) and testicles.

Work exposure has resulted in testicular toxicity with sterility. Similar levels of exposure apparently do not affect ovarian function. Among those males who recovered fertility and fathered offspring, follow-up studies did not detect any excess of birth defects associated with prior DBCP exposure.

DBCP damages chromosomes in a variety of test systems and causes cancer in some animal studies.