

ISODRIN
(C₁₂H₈C₁₆) CAS #465-73-6 (Pesticide)
Synonyms include:
1,4;5,8-dimethanonaphthalene,1,2,3,4,10,10-hexachloro-,
4,4a,5,8,8a hexahydro-endo, endo
Compound 711; Experimental Insecticide 711; SD 3418

SOURCE/USE

Isodrin must be synthesized and is no longer manufactured or used commercially in the United States. This solid chlorinated hydrocarbon has melting point of 465 degrees Fahrenheit, but it is unstable and may react with light or acids. In soil it may undergo oxidation by microbes and be converted to endrin. It is not combustible, but can decompose at high temperatures to produce noxious gases (e.g, chlorine, other chlorinated hydrocarbons).

ROUTES OF EXPOSURE

Isodrin can be absorbed by inhalation, ingestion or skin absorption. Although the risk of off-post acute exposure to arsenic as a result of remediation at the Rocky Mountain Arsenal is very small, any such exposure would very 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. Isodrin may adsorb onto the surface of dust particles which may be swallowed as well as inhaled.

APPLICABLE STANDARDS AND LIMITS	
ATSDR acute MRL	Not Available
Occupational standards	Not Available
Odor threshold	Not Available
RMA acute fence line criteria	ARC - 0.053 mg/m ³ MARC - 0.18 mg/m ³
RMA chronic fence line criteria	Cancer - NA Noncancer - 1.1 µg/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 isodrin exposure.

ACUTE HEALTH EFFECTS

The organochlorines, including isodrin, are convulsants causing excitation of the CNS. Seizures may be the first symptom of acute exposure, occurring within minutes to hours of a sufficient dose. Other symptoms of CNS toxicity may include nausea, vomiting, dizziness, headache, tremors, elevated blood pressure, fever, rapid heart rate, and altered behavior. Coma may occur and last beyond the convulsive phase. Respiratory depression occurs with acute organochlorine toxicity. Clonic muscular jerking may occur apart from full blown seizure or convulsive activity.

Respiratory depression is seen with acute CNS toxicity from organochlorines as noted above. Respiratory system irritation of mucous membranes is not a prominent feature of organochlorine exposure, and may be largely due to petroleum distillates in which it was suspended.

Mild irritation of skin and eye may occur with pure organochlorines, presumably including isodrin, and may also be related to petroleum distillates which were used as carriers. Organochlorine exposure can cause skin effects such as acne and erythematobullous dermatitis.

Ingestion produces CNS symptoms, but survivors of any route of exposure may develop liver damage as seen in animal studies.

Animal studies support an expectation of kidney damage in acute (and chronic) exposure when not acutely fatal.

CHRONIC HEALTH EFFECTS

Isodrin is fat soluble and accumulates (as an epoxide) in body fat. Based on other organochlorines, recurrent exposure may produce chronically recurring EEG abnormalities and seizures. This chemical has not been classified as to its carcinogenicity by EPA. Some members of this family of insecticides, notably dieldrin, can produce cancer and birth defects in different animal studies. Liver and kidney damage should be considered possible.