Sources of Exposure

General Populations

- Significant exposure of the general population to disulfoton is not likely because the U.S. Environmental Protection Agency (EPA) cancelled production of pesticides containing disulfoton in the United States in 2009. Leftover stock was permitted for sale until 2011, and pesticides containing disulfoton were used as recently as 2016.
- Inhalation and dermal exposure of the general population to disulfoton is low, and exposure in drinking water is likely negligible.
- People who live near manufacturing or processing sites, or hazardous waste sites containing disulfoton may be exposed to higher levels than the general population.
- Exposure to small amounts of disulfoton may occur from some foods, especially ones produced outside of the United States. These levels are expected to be low.

Occupational Populations

- Occupational exposure is not likely since disulfoton is no longer produced.
- Previously, occupational exposure was most likely to occur through inhalation of or dermal contact with pesticides containing disulfoton.
- Workers who manufacture, handle, or apply disulfoton or who are involved in disposing of disulfoton are at higher risk of exposure than the general population.

Toxicokinetics and Biomonitoring

Toxicokinetics

- Disulfoton is well-absorbed through the gastrointestinal tract.
- Absorbed disulfoton is primarily distributed to the liver. It is also distributed to the kidneys, whole blood, red blood cells, plasma, fat, skin, muscles, brain, small intestine, pancreas, and bile.
- Disulfoton undergoes metabolism through oxidation reactions and hydrolysis. The urinary metabolites of disulfoton are diethyl phosphate (DEP), diethyl thiophosphate (DETP), diethyl dithiophosphate (DEDPT), and diethyl phosphorothiolate (DEPTh).
- The major route of excretion of disulfoton and its metabolites is through urine, with smaller amounts being excreted in feces and expired breath. Excretion through breastmilk is unknown.

NHANES Biomonitoring

- No information on blood or urine levels of disulfoton in the U.S. population were identified. Metabolites, not exclusive to disulfoton exposure, have been detected in urine samples among the U.S. population.

Biomarkers/Environmental Levels

Biomarkers

Disulfoton and its metabolites can be measured in blood and urine to indicate exposure. However, the metabolites are not specific to disulfoton and can result from exposure to other organophosphates. Red blood cell cholinesterase can also be measured and is an indicator of an effect from exposure; however, this is not specific to disulfoton.

Environmental Levels

These levels are from domestic samples taken between 1974 to 2007 prior to the U.S. cancellation of disulfoton in 2009. Levels in environmental media today are expected to be much lower.

Air

- Outdoor air range: less than the limit of detection–4.7 ng/m^3

Water

- Groundwater range: 0.39–6 µg/L
- Surface water range <0.002–3,300 ng/L

Soil and Sediment

- Range: <0.2–227.8 µg/kg

Food

- Crops range: 0.05–1.0 mg/kg
- Fish tissue range: 0–80.3 ng/g

Reference

Agency for Toxic Substances and Disease Registry (ATSDR). 2022. Toxicological Profile for Disulfoton. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Services.

ToxGuide™ for Disulfoton

$C_8H_{19}O_2PS_3$

CAS# 298-04-4

August 2022

U.S. Department of Health and Human Services
Public Health Service
Agency for Toxic Substances and Disease Registry
www.atsdr.cdc.gov

ATSDR
AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY

Chemical and Physical Information

Disulfoton is a Colorless Oil

- Disulfoton, also known as Di-Syston, is a systemic organophosphate insecticide and acaricide.
- Disulfoton is a manmade, non-naturally occurring oily liquid with low volatility and low water solubility.
- It has a sulfur-like odor, is colorless to yellow, and is expected to be short-lived in the atmosphere.

Routes of Exposure

- Inhalation – Inhalation is a primary route of exposure for workers who apply disulfoton as a pesticide. Exposure via inhalation is otherwise not likely to occur due to the short half-life in air (approximately 3 hours) and low volatility.
- Oral – A minor route of exposure may occur through ingestion of foods contaminated with disulfoton.
- Dermal – Dermal contact is a potential route of exposure for workers. Children may also be exposed if playing in contaminated soils.

Disulfoton in the Environment

- Disulfoton is not expected to be found in the environment at high levels due to its cancellation by the EPA in 2009.
- Previously, disulfoton was found in the atmosphere. Despite the short estimated half-life, detection of disulfoton in regions where it is not used indicates that vapor-phase disulfoton may travel long distances.
- In water, disulfoton adsorbs to suspended soils and sediments. Volatilization from water is slow to negligible.
- Disulfoton was primarily measured in soils where mobility was slight to moderate. Small amounts could leach into groundwater or be absorbed up plant root systems.
- In the environment, disulfoton degraded or transformed with hydrolysis in air and water; photooxidation, biodegradation, or biotic processes in water, soil, or sediment.
- Disulfoton does not bioaccumulate in fish or other aquatic species.

Relevance to Public Health (Health Effects)

Health effects are determined by the dose (how much), the duration (how long), and the route of exposure.

Minimal Risk Levels (MRLs)

Inhalation

- An intermediate-duration (15–364 days) inhalation MRL of 0.0006 mg/m^3 (0.6 µg/m^3) was derived for disulfoton; this was adopted for the acute-duration (≤14 days) inhalation MRL.
- No chronic-duration (≥365 days) inhalation MRL was derived for disulfoton.

Oral

- An acute-duration (≤14 days) oral MRL of 0.0003 mg/kg/day (0.3 µg/kg/day) was derived for disulfoton.
- An intermediate-duration (15–364 days) oral MRL of 0.00009 mg/kg/day (0.09 µg/kg/day) was derived for disulfoton.
- A chronic-duration oral MRL of 0.00006 mg/kg/day (0.06 µg/kg/day) was derived for disulfoton.

Health Effects

- Neurotoxicity is the most sensitive endpoint in human and animals exposed to disulfoton following inhalation, oral, or dermal exposure. Neurotoxicity is primarily characterized by depressed acetylcholinesterase activity with signs of

Health Effects

- In workers and cases of high oral exposure, neurotoxic signs include headaches, nausea, weakness, fatigue, confusion, and muscle spasms.
- In animal studies, depressed cholinesterase was observed in the offspring of exposed animals, along with signs of developmental delay.
- One man had edema of lungs and intra-alveolar bleeding following ingestion. Studies in rats have shown respiratory effects such as discolored, mottled lungs, nasal turbinate inflammation, and breathing difficulties.
- Mice and rat studies showed that inhalation and oral exposure resulted in reproductive effects in males and females such as lesions on reproductive organs, reduced mating, and increased stillbirths.
- Neither the Department of Health and Human Services (HHS) nor the International Agency for Research on Cancer (IARC) have classified disulfoton regarding its carcinogenicity. The EPA has classified disulfoton in Group E, indicating evidence of noncarcinogenicity for humans.

Children's Health

- There are no data regarding developmental effects in humans. Impaired offspring growth in animal studies suggests that disulfoton may affect human development; however, doses used in animal studies were generally much higher than current environmental levels.