Sources of Exposure

General Populations

- The primary route of exposure for the general population is dermal contact from intentional application to skin and clothing of commercial products containing DEET.
- Exposure to DEET can also occur when drinking water or showering with water that contains DEET.
- Levels of DEET in air and water are low.

Occupational Populations

- Workers in industries that manufacture DEET or formulate DEET-containing products are likely to be at higher risk than the general population for DEET exposure.
- Employees who use DEET regularly during their seasonal work, such as those at national parks, can be exposed to higher levels of this substance.

Toxicokinetics and Biomonitoring

Toxicokinetics

- Following oral exposure, DEET is almost completely absorbed through the gastrointestinal tract based on a study of rats.
- Significantly less DEET enters the body when applied onto the skin. Absorption ranges from 4 to 17% of the applied dose. This could be higher or lower with different product formulations.
- Most DEET (up to 80%) is broken down in the body into other chemicals (metabolites) in the liver.
- DEET and its metabolites distribute widely throughout the body but do not accumulate in any one. They have been found in the brain, liver, lung, spleen, kidney, blood, tears and fat.
- DEET has been found in cord blood of pregnant women and animals and at low levels in the fetus.
- Most DEET and metabolites are excreted quickly (within hours) in the urine. A small proportion is excreted in the feces.

NHANES Biomonitoring

In the 2009–2010 NHANES survey, the average urinary DEET level was below the detection limit (0.089 µg/L). The geometric mean for the metabolite DCBA was 4.74 µg/g creatinine, while DHMB was not determinable.

Biomarkers/Environmental Levels

Biomarkers

- DEET in urine may not be a reliable biomarker of exposure because the compound is rapidly metabolized after oral and dermal exposure.
- Urinary metabolites of DEET, 3-(diethylcarbamoyl) benzoic acid (DCBA) and N,N-diethyl-3-(hydroxymethyl) benzamide (DHMB), are more useful biomarkers of exposure.

Environmental Levels

Air

- There are no data regarding levels of DEET in air in the Unites States.

Sediment and Soil

- There are no data regarding levels of DEET in sediment or soil.

Water

- Levels of DEET measured in 188 surface water samples throughout the United States ranged from 0.013 to 0.66 µg/L with a central value of 0.55 µg/L in 2011 DEET has been found in surface and groundwater, treated effluent, and coastal waterways.

Reference

Agency for Toxic Substances and Disease Registry (ATSDR). 2017. Toxicological Profile for DEET (N,N-Diethyl-*meta*-Toluamide). Atlanta, GA: U.S. Department of Health and Human Services, Public Health Services.

ToxGuide™ for

DEET (N,N-Diethyl-*meta*-Toluamide)

$C_{12}H_{17}NO$

CAS# 134-62-3

August 2017

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

ATSDR
AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY

Chemical and Physical Information

DEET

- DEET (N,N-diethyl-*meta*-toluamide) is a nearly clear to amber-color liquid.
- DEET is the active ingredient in many common repellents widely used to repel biting pests such as mosquitos and ticks.
- DEET formulations are typically used as sprays, mists, lotions, and wipes. These can be applied directly onto human skin or onto clothing.
- DEET is currently sold as an ingredient in several common repellent products, including Skeeter Skat, Chigger-wash, as well as various Off!®, Repel, and Old Time Woodsman brand products and various Cutter brand products, such as Cutter All family, Cutter Dry, and Cutter Backwoods.

Routes of Exposure

- Inhalation – Minor route of exposure for the general population.
- Oral – Minor route of exposure the general population through ingestion of contaminated water.
- Dermal – Principal route of exposure for the general population through application of consumer products containing DEET.

DEET in the Environment

- DEET can enter the air during spray applications.
- In air, DEET is removed quickly by reacting with hydroxyl radicals. The estimated half-life for this reaction is 5 hours.
- Long range transport of DEET in the air is not expected.
- DEET enters aquatic systems as a result of human activities such as showering or bathing and laundering of clothes sprayed with insect repellents containing DEET.
- DEET is not expected to volatize from water surfaces. It is degraded in water by aerobic microorganisms.
- DEET is expected to have low adsorption to soil; therefore, leaching to groundwater is possible.
- DEET is not expected to stay in the environment long and is unlikely to bioaccumulate.

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

- No acute- (≤14 days), intermediate- (15–364 days), or chronic- (≥365 days) duration inhalation MRLs were derived for DEET.

Oral

- An intermediate-duration (15–364 days) oral MRL of 1 mg/kg/day was derived for DEET.
- No acute- (≤14 days) or chronic- (≥365 days) duration oral MRLs were derived for DEET.

Health Effects

- There have been occasional reports of adverse health effects after excessive use of repellents containing DEET. These include neurological effects (seizures, uncoordinated movements, agitation and aggressive behavior), low blood pressure, and skin irritation.
- Considering the extensive intentional consumer use of products containing DEET and the few reports of adverse effects, the risk of health effects due to exposure to DEET appears to be quite low.
- No birth defects or abnormalities were seen in offspring of pregnant women or animals exposed to DEET.
- In a multi-generational study, feeding rats DEET throughout mating, pregnancy, and lactation resulted in reduced body weight of male and female pups on during lactation. Some male offspring also had increased incidences of gross and microscopic lesions in the kidney.
- Animals chronically exposed to DEET by either oral or dermal routes did not develop an increased number of tumors. Exposure of some animals resulted in fewer tumors and longer life.
- The U.S. Department of Health and Human Services has not classified DEET as to its carcinogenicity. The U.S. Environmental Protection Agency's (EPA) Office of Pesticide Programs has classified DEET as a Group D chemical, not classifiable as a human carcinogen. The International Agency for Research on Cancer (IARC) has not classified DEET as to its carcinogenicity.

Children's Health

It is not known if children are more sensitive to DEET exposure than adults.