

## 6. POTENTIAL FOR HUMAN EXPOSURE

### 6.1 OVERVIEW

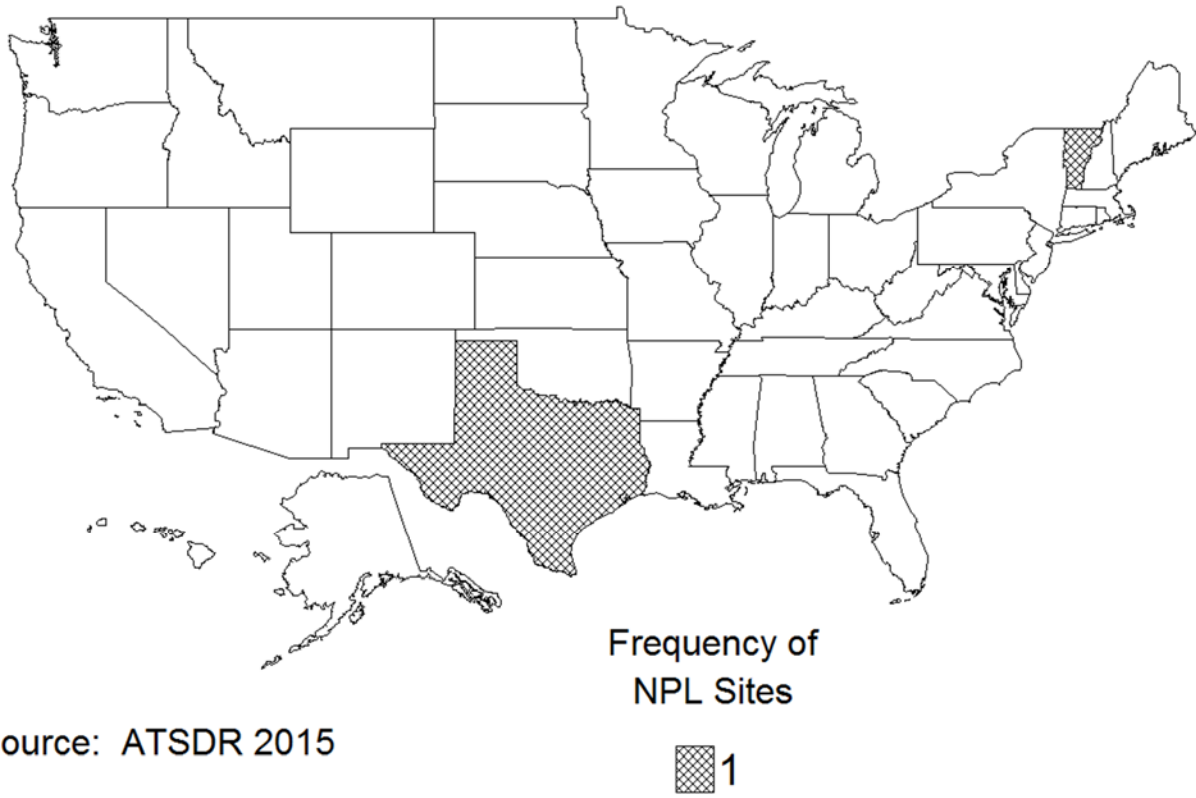
DEET was previously identified in at least 2 of the 1,832 hazardous waste sites across the United States (a refining operation in Friendswood, Texas and a municipal landfill in Bennington, Vermont) that have been proposed for inclusion on the EPA National Priorities List (NPL) (ATSDR 2015). The concentrations of DEET found at these two NPL sites were not considered a health concern (ATSDR 2015). Figure 6-1 shows the states with DEET-contaminated NPL sites, and the frequency or number of occurrences of these sites in each state. However, recent NPL site information indicates that DEET is no longer identified as an existing contaminant at any of the 1,832 hazardous waste sites across the United States that are listed as of that date on the NPL (EPA 2017a, 2017b). The EPA Superfund program is a dynamic system that continually evaluates sites across the United States for inclusion and deletion from the NPL; therefore, the exact number of hazardous waste sites may vary with time. The site in Texas is no longer on the NPL (EPA 2014g) and the site narrative for the Vermont site does not identify DEET as a major contaminant (EPA 2014h). However, the number of sites evaluated for DEET is not known.

DEET enters the environment via direct and indirect sources through its use as a commercial product. DEET is an insect and acarid repellent intended for indoor and outdoor residential human use. Water is the most common environmental medium in which DEET has been detected. DEET has been detected in surface water, groundwater, and drinking water. DEET enters aquatic systems as a result of common human activities, such as showering or bathing and laundering of clothes after products containing DEET have been applied. DEET is expected to be moderately mobile and has the potential to leach into groundwater. It is not expected to undergo hydrolysis in aquatic environments, and biodegradation under anaerobic conditions is negligible. However, DEET is considered inherently and readily biodegradable (Weeks et al. 2012) and is not considered a persistent or bioaccumulative substance.

The most important route of exposure to the general population is through dermal contact via intentional application to the skin of consumer products containing DEET. Dermal application of DEET can result in absorption through the skin. Exposure via inhalation, ocular and oral routes may be possible; however, due to the intended use of end products, these routes are minimal in comparison with dermal exposure.

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**Figure 6-1. Frequency of NPL Sites with DEET Contamination**



Source: ATSDR 2015

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Occupational exposure may occur via dermal contact and inhalation where DEET is manufactured or used. DEET has been monitored in human urine and blood samples.

### 6.2 RELEASES TO THE ENVIRONMENT

The Toxics Release Inventory (TRI) data should be used with caution because only certain types of facilities are required to report (EPA 2005). This is not an exhaustive list. Manufacturing and processing facilities are required to report information to the TRI only if they employ 10 or more full-time employees; if their facility is included in Standard Industrial Classification (SIC) Codes 10 (except 1011, 1081, and 1094), 12 (except 1241), 20–39, 4911 (limited to facilities that combust coal and/or oil for the purpose of generating electricity for distribution in commerce), 4931 (limited to facilities that combust coal and/or oil for the purpose of generating electricity for distribution in commerce), 4939 (limited to facilities that combust coal and/or oil for the purpose of generating electricity for distribution in commerce), 4953 (limited to facilities regulated under RCRA Subtitle C, 42 U.S.C. section 6921 et seq.), 5169, 5171, and 7389 (limited S.C. section 6921 et seq.), 5169, 5171, and 7389 (limited to facilities primarily engaged in solvents recovery services on a contract or fee basis); and if their facility produces, imports, or processes  $\geq 25,000$  pounds of any TRI chemical or otherwise uses  $> 10,000$  pounds of a TRI chemical in a calendar year (EPA 2005).

DEET may be released to the environment directly or indirectly through its use in commercial products. There are no natural sources of DEET known to be environmentally significant.

#### 6.2.1 Air

There is no information on releases of DEET to the atmosphere from manufacturing and processing facilities because these releases are not required to be reported (EPA 2005).

DEET is released into the atmosphere solely by human activities associated with its production and use as an insect and acarid repellent. DEET can enter the air during spray application onto skin or clothing. It has been reported that evaporation from human skin is 9.6% in 1 hour (Spencer et al. 1979). Cheng et al. (2006) surmised that the presence of DEET in air of the Lower Fraser Valley of Canada was primarily due to its widespread use by the Canadian population during the summer. Levels in the urban forest showed a diurnal change ( $3.03 \text{ ng/m}^3$  during the day and  $1.25 \text{ ng/m}^3$  at night), while those near highly visited Golden Ear Provincial Park were higher ( $11.1\text{--}11.4 \text{ ng/m}^3$  in the day and up to  $37.1 \text{ ng/m}^3$  at night when insect density may have been greatest). The lowest levels measured ( $0.53\text{--}0.78 \text{ ng/m}^3$ ) were at a

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remote location and were considered to be the ambient background for the area resulting from spraying livestock in that rural area.

### 6.2.2 Water

There is no information on releases of DEET to the water from manufacturing and processing facilities because these releases are not required to be reported (EPA 2005).

DEET is frequently detected in the aquatic environment (Knepper 2004; Kolpin et al. 2002; Sandstrom et al. 2005). DEET can enter surface waters directly due to recreational activities such as swimming, via swimmers with DEET products on their skin or clothing, or indirectly from overspray during application. Because of its limited absorption through human skin, the majority of applied DEET is washed off the skin (Selim et al. 1995). DEET is released into water systems through routine human activities such as showering and bathing of individuals who have recently applied DEET products. DEET applied to clothing may end up in waste water treatment plants (WWTPs) or may be released with gray water after the clothes are laundered and enter the environment after passing through the WWTPs or domestic septic systems. Additionally, sewage effluent may contain DEET and DEET metabolites due to human absorption and excretion (Aronson et al. 2012; Costanzo et al. 2007). Monitoring data indicate that the highest concentration of DEET in aquatic environments correlates with its increased application during the summer months (Knepper 2004; Sandstrom et al. 2005) and late winter (Sandstrom et al. 2005). DEET can enter groundwater from contaminated surface waters or leachate from landfills (Cordy et al. 2004).

### 6.2.3 Soil

There is no information on releases of DEET to the soil from manufacturing and processing facilities because these releases are not required to be reported (EPA 2005).

DEET may be released to soil as a result of overspray during application as a repellent or irrigation of soils with reclaimed water in which DEET is present. DEET may also be released to soil when it is disposed of in landfills or from accidental spills of products or wastes containing DEET during overland transportation.

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**6.3 ENVIRONMENTAL FATE**

The environmental fate of DEET, which includes the transport, partitioning, and transformation of this substance, is controlled by various physicochemical properties, degradation, and other loss processes. According to European Union (EU) regulatory criteria and the overall data presented below, DEET is unlikely to bioaccumulate and is not expected to be highly persistent in the environment (Aronson 2012; Weeks et al. 2012).

**6.3.1 Transport and Partitioning**

Based on its vapor pressure of  $5.6 \times 10^{-3}$  mm Hg at 20°C, DEET will exist solely as a vapor if released to the atmosphere. Vapor-phase DEET is expected to degrade in the atmosphere via reaction with photochemically-produced hydroxyl radicals, with an estimated half-life of 5 hours (EPIWIN 2012). Therefore, persistence and long-range transport of DEET in air is not expected.

Monitoring data indicate water as the major environmental sink for DEET. If released to water, DEET is not expected to accumulate in aquatic organisms. Experimental bioconcentration factors (BCFs) of 0.8–2.4 L/kg at 0.5 mg/L and <2.4 at 0.05 mg/L measured in carp indicate that the potential for bioconcentration in aquatic organisms is low (CITI 1992; Weeks et al. 2012). Volatilization of DEET from water surfaces is not expected to be an important fate process based on its estimated Henry's Law constant of  $4.5 \times 10^{-8}$  atm·m<sup>3</sup>/mole (EPIWIN 2012).

DEET is expected to have low adsorption to soils and sediment; therefore, leaching into groundwater is possible and removal by sludge adsorption in sewage treatment plants is incomplete. Experimental soil sorption  $K_{oc}$  factors have been reported as 43.3 L/kg resulting from a high-performance liquid chromatography (HPLC) estimation and 47–126 L/kg from an Organization for Economic Co-operation (OECD) guideline method (Adsorption-Desorption Using a Batch Equilibrium Method) using five soils (ECHA 2010; Weeks et al. 2012). Secondary-treated effluent from a municipal waste treatment plant containing DEET was applied to a 2.4-m soil column packed with Mohall-Laveen sandy loam soil over 23 days in a study assessing the potential for compounds to persist and possibly enter groundwater upon recharge. DEET was detected in the column inflow at the beginning and the end of the experiment at concentrations of 1.4 and 1.6 µg/L, respectively. DEET was finally detected in the drainage samples at the end of the experiment at a concentration of 2.3 µg/L (Cordy et al. 2004).

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**6.3.2 Transformation and Degradation**

DEET can partition into various environmental compartments and is subject to abiotic and biotic degradation processes.

**6.3.2.1 Air**

The major removal process for DEET in the atmosphere is photooxidation via reaction with hydroxyl radicals. The estimated half-life for this reaction is 5 hours, based on an estimated rate constant of  $2.5 \times 10^{-11}$  cm<sup>3</sup>/molecule-second at 25°C (EPIWIN 2012). Direct photolysis in the ambient atmosphere is not expected to be an important fate process because DEET does not absorb light at environmentally relevant wavelengths (EPA 2014m; Weeks et al. 2012).

**6.3.2.2 Water**

DEET is considered to be hydrolytically stable; results from guideline studies however, indicate that DEET will be biodegradable under environmental conditions and should not be persistent in the environment.

Using the Japanese Ministry of International Trade and Industry (MITI) test based on OECD Guideline 301C, DEET, at 100 mg/L, achieved 0% of its theoretical biochemical oxygen demand (BOD) after 4 weeks using a sewage inoculum maintained under aerobic conditions and was not considered readily biodegradable (CITI 1992). DEET was degraded 48.6% after 28 days using the closed bottle (OECD Guideline 301D) test and it was concluded that DEET was probably inherently biodegradable but did not meet the criteria to be classified as readily biodegradable (Weeks et al. 2012). In another guideline study, DEET achieved 83.8% of its theoretical CO<sub>2</sub> evolution after a 28-day incubation period using the modified Sturm (OECD Guideline 301B) test and was considered readily biodegradable (Weeks et al. 2012). The discrepancies in results could be attributed to the toxic effects of DEET on microbial populations at high concentrations, such as those used in OECD Guideline 301C. Testing indicated that DEET only caused minor inhibitory effects on microbial activity and was not typically a concern at environmentally relevant concentrations (ECHA 2010; Weeks et al. 2012).

Hydrolysis in water is not expected to be an important fate process. Results from two studies following OECD Guideline 111, EPA Method 835.2110, and EC C.7 demonstrate that DEET is hydrolytically

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stable at 50°C and pH 4, 7, and 9 (EPA 1998b, 1998c, 2002; Weeks et al. 2012). The UV/visible absorption spectrum for a 10 ppm DEET solution in methanol of 200–225 nm (EPA 2014m) suggests that this chemical does not absorb at wavelengths >290 nm and therefore would not be expected to undergo direct photolysis in sunlight. Direct photolysis in sterile water did not contribute to decomposition in a simulation experiment by Calza et al. (2011) and in a 7-day experiment using distilled water and a xenon arc light (Weeks et al. 2012). Indirect photolysis in river water, however, resulted in degradation. Photocatalytic degradation experiments in river water under illumination and in the dark resulted in half-lives of 5 and 15 days, respectively (Calza et al. 2011). The main transformation products identified were N,N-diethyl-3-hydroxymethyl-benzamide, N,N-diethyl-*m*-benzamide, N-ethyl-*m*-formylbenzamide, and N-ethyl-*m*-toluenamide (Calza et al. 2011). Confirmed DEET microbial degradates that have been reported include 3-methylbenzoate (which further degrades to 3-methylcatechol), N,N-diethyl-*m*-toluamide-N-oxide, and N-ethyl-*m*-toluamide (which further degrades to N-ethyl-*m*-toluamide-N-oxide) (Aronson et al. 2012). Of the degradation products detected in the river water study, it was noted that several resulted from biotic processes, while others were formed from indirect photolysis. Indirect photolysis in sunlit surface waters and biotic degradation under aerobic conditions are the most important removal processes for DEET (Calza et al. 2011). Biotic degradation processes produce products via monohydroxylation (or N-oxidation), N-dealkylation, and demethylation on the benzene ring (Calza et al. 2011; Rivera-Cancel et al. 2007; Seo et al. 2005).

Anaerobic biodegradation of DEET using aquifer slurries obtained from the Norman municipal landfill in Oklahoma was shown to be negligible. Measured DEET concentrations at 0, 1, 8, and 11 months of incubation were 171, 194, 198, and 199 µM, respectively, in aquifer slurries from a sulfate reducing site. DEET did not biodegrade in an aquifer slurry from a methanogenic site; at 0, 1, 8, and 11 months of incubation, concentrations of DEET were 194, 192, 190, and 199 µM, respectively (Kuhn and Sulflita 1989).

### 6.3.2.3 Sediment and Soil

No biodegradation studies in soil samples were located; OECD guideline studies and aquifer studies, however, suggest that DEET is biodegradable under aerobic conditions, but biodegrades slowly under anaerobic conditions (Kuhn and Sulflita 1989; Weeks et al. 2012).

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**6.3.2.4 Other Media**

Results from pure culture studies have demonstrated the ability of *Pseudomonas putida* DTB to metabolize DEET by hydrolyzing the amide bond resulting in two degradation products, 3-methylbenzoate and diethylamine (Rivera-Cancel et al. 2007). 3-Methylbenzoate has been shown to be readily biodegradable, and predictive methods have suggested that other DEET metabolites are not expected to persist in the environment (Aronson et al. 2012). An additional study on the metabolism of DEET by soil fungi (*Cunninghamella elegans*) identified three metabolites: N,N-diethyl-*m*-toluamide-N-oxide, N-ethyl-*m*-toluamide-N-oxide, and N-ethyl-*m*-toluamide (Seo et al. 2005). It should be noted that these studies were not with mixed microbial populations typically found in natural systems and should therefore not be considered definitively representative of the biodegradation of DEET in the environment.

DEET removal from WWTPs varies depending on the specific conditions of each site. Aronson et al. (2012) summarized several studies in which removal from WWTPs ranged from 10 to 99%. Removal from trickling filter treatment plants was generally lower than activated sludge plants. Knepper (2004) observed that elimination rates in WWTPs varied with influent concentration levels of DEET. Elimination rates were negligible in winter and spring months and increased in late summer up to 90% when concentration levels of DEET peak.

The removal of DEET from drinking water and waste water treatment plants located in South Korea was assessed by Kim et al. (2007). Various removal systems including membrane bioreactors, reverse osmosis, nanofiltration, and ultraviolet (UV) irradiation were analyzed. Minimal removal was reported with membrane systems; the other systems, however, removed DEET to concentrations <1 ng/L (initial concentrations averaged 18 ng/L). Utilizing granulated activated carbon was the most efficient removal system for drinking waters.

**6.4 LEVELS MONITORED OR ESTIMATED IN THE ENVIRONMENT**

Reliable evaluation of the potential for human exposure to DEET depends in part on the reliability of supporting analytical data from environmental samples and biological specimens. Concentrations of DEET in unpolluted atmospheres and in pristine surface waters are often so low as to be near the limits of current analytical methods. In reviewing data on DEET levels monitored or estimated in the environment, it should also be noted that the amount of chemical identified analytically is not necessarily



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equivalent to the amount that is bioavailable. The analytical methods available for monitoring DEET in a variety of environmental media are detailed in Chapter 7.

Care should be taken when assessing analytical results for which a limit of detection (LOD) or similar sensitivity value is not provided for the substance of interest, as well as the study reports not having detected that substance; failing to detect a substance does not mean that it is not present.

#### 6.4.1 Air

DEET was detected in an air quality study of  $<2.5 \mu\text{m}$  aerosol samples performed in Canada from August 1 to 30, 2001. Samples were taken both at daytime and nighttime at five locations: Golden Ears Park (in a forested area), Cassiar Tunnel, Slocan Park (low-density urban surrounding), Langley Lochiel (a rural environment), and an elevated Sumas Eagle Ridge (forest/urban area). DEET concentrations ranged from 0.95 to 15.4 ng/m<sup>3</sup> (Cheng et al. 2006).

DEET was detected, but not quantified, in the atmosphere of Rome, Italy in the winter and summer of 2009 (Balducci et al. 2012).

#### 6.4.2 Water

DEET has been detected in streams, surface water and groundwater systems, and sewage treatment plant effluents throughout the United States (Glassmeyer et al. 2005; Kolpin et al. 2002; Sandstrom et al. 2005). A summary of published studies by Brausch and Rand (2011) reported measured concentrations for DEET in 188 surface waters samples throughout the United States ranging from 13 to 660 ng/L (0.013–0.66  $\mu\text{g/L}$ ), with a median value of 55 ng/L (0.055  $\mu\text{g/L}$ ). A review by Costanzo et al. (2007) reported that DEET has been detected and reported in worldwide water samples, such as drinking water, streams, marine waters, groundwater, and treated effluent at concentrations of 40–3,000 ng/L (0.04–3.0  $\mu\text{g/L}$ ) and has also been detected in coastal waterways in Australia at concentrations of 8–1,500 ng/L (0.008–1.5  $\mu\text{g/L}$ ). DEET was detected in 8 of 50 groundwater samples from unconfined ( $<30 \text{ m}$ ) and confined (up to 500 m) aquifers in Tokyo taken between October and November 2007 (Kuroda et al. 2012). The arithmetic mean limit of quantification (LOQ) for the study was reported as 20.8 ng/L (0.0208  $\mu\text{g/L}$ ). The concentrations in these groundwater samples were lower on average, yet were comparable to sewage influent concentrations of DEET (503 ng/L [0.503  $\mu\text{g/L}$ ]) measured in the WWTPs of Tokyo in a previous study (Kuroda et al. 2012). DEET was detected in 83.5% of groundwater samples

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(n=164) obtained from 23 European countries at an average concentration of 9 ng/L (0.009 µg/L) (Loos et al. 2010). Knepper (2004) investigated WWTP samples from June 1998 until October 2002 in Wiesbaden, Germany. During the winter and spring months of 1999, influent and effluent concentrations were comparable, yielding concentrations as high as to 0.6 µg/L. Summer influent concentrations in 1999 increased to 3 µg/L; effluent concentrations increased to 1–1.5 µg/L. Influent concentrations in November 1999 decreased from the summer month concentrations to 0.26–0.49 µg/L (Knepper 2004). Effluents from 90 WWTPs across Europe were sampled in 2010. Out of 156 chemicals targeted for analysis, DEET was one of the highest concentration chemicals found at levels up to 15.8 µg/L, with an average detection of 678 ng/L (0.678 µg/L); LOQ=1 ng/L (0.001 µg/L) (Loos et al. 2013a). DEET was detected in influent samples from three WWTPs serving large metropolitan areas of the United States at levels of 54–500 ng/L (0.054–0.5 µg/L) and in effluent samples at 100–260 ng/L (0.1–0.26 µg/L) (Trenholm et al. 2008).

Guardiola et al. (1989) identified DEET in groundwater samples from wells, which had been closed for several years due to pollution, in the Besos river basin (northeastern Spain) at concentrations up to 34 ng/L (0.034 µg/L). In a United States Geological Survey (USGS) study, samples were taken on September 6, 2000 from five multilevel monitoring wells near the Norman Landfill in Oklahoma, with reported concentrations of DEET ranging from <800 to 1,300 ng/L (<0.8–1.3 µg/L); the detection limit was 40 ng/L (0.04 µg/L). Well depths ranging from 3.26 to 6.29 m and their distances from the landfill were from 1 to 574 m (Barnes et al. 2004). DEET has been detected in surface water samples in numerous studies at concentrations of 2–2,100 ng/L (0.002–2.1 µg/L) (Dougherty et al. 2010). DEET was detected in water samples at 3 of 11 sites sampled in September and April near Liberty Bay, Washington. It was detected in one surface water sample and two groundwater samples at concentrations of 2.3–3.3 ng/L (0.0023–0.0033 µg/L). Two of the sites were also tested with polar organic chemical integrative samplers (POCIS) put in place for 62 days from January to March 2007 and again for 61 days from July to September 2007; DEET was detected at site 1 at 2.1–3.4 ng/POCIS (detection limit 1.0 ng/POCIS) and at site 2 at 3.0–6.3 ng/POCIS (Dougherty et al. 2010).

DEET was detected, but not quantified, in leachate samples of three domestic and industrial waste landfills (Eggen et al. 2010). These sites operated between 1973 and 1989 (this site also accepted separated residual domestic waste from 1985 through 2010 when the paper was written), 1974 and 2006, and 1972 and 2002.

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In a USGS survey during 1999 and 2000, 139 streams from 30 states were sampled. DEET was reported in 74.1% of the samples analyzed at a median concentration of 60 ng/L (0.06 µg/L) and a maximum concentration of 1,100 ng/L (1.1 µg/L) (Kolpin et al. 2002). In the 2000 USGS survey, DEET was detected in 73.2% of 56 stream samples at a median concentration of 0.05 µg/L and a maximum concentration of 1.13 µg/L. The analytical method used methylene chloride liquid-liquid extraction (LLE) of whole water followed by capillary-column gas chromatography/mass spectrometry (GC/MS) operated in selected-ion monitoring mode, and achieved a detection level of 0.02 µg/L, if retention time and ionic abundance criteria were met; otherwise, the reporting limit was 0.08 µg/L (Sandstrom 2005). Site selection focused on urban and agricultural areas, during various seasons, at locations where there was the possibility of waste water contamination, via human, industrial, and agricultural sources, entering the streams. Levels were highest near urban areas and during summer and late winter. An attempt was made to re-analyze these samples for DEET metabolites; however, none of the chemicals could be detected in the samples. Limitations were noted and more accurate methods for their determination need further consideration (Sandstrom et al. 2005). In 2001, Kolpin et al. (2004) detected DEET in water samples collected from 23 stream locations situated upstream and downstream of 10 cities in Iowa. Stream samples were taken during high, normal, and low flow conditions. DEET was detected in the 23 normal-flow samples with a frequency of 4.3% and a maximum concentration of 62 ng/L (0.062 µg/L) and in the 30 low-flow samples with a frequency of 6.7% and a maximum concentration of 130 ng/L (0.13 µg/L). DEET was not detected in any of the 23 high-flow samples. DEET was detected in 43% of samples collected in March, April, and August of 2004 from 18 streams in north-central and northwestern Arkansas. Concentrations in the water samples were below the detection limit (0.5 µg/L) and were estimated as 18–83 ng/L (0.018–0.083 µg/L) (Haggard et al. 2006). Water samples collected from the main-stem Mississippi River during 1987 through 1992 contained DEET at concentrations of 8–110 ng/L (0.008–0.11 µg/L) (Goolsby and Pereira 1996). In 1989, DEET was detected in five of eight surface water samples taken at various locations along the Rhine River in The Netherlands at concentrations of 21–46 ng/L (0.021–0.046 µg/L) (Hendriks et al. 1994). DEET was detected in 12 of 15 sampling sites along the northern River in Germany between June 24 and July 7, 1998 at concentrations of 0.11–1.09 ng/L (0.00011–0.00109 µg/L) (Weigel et al. 2002). Po River water samples collected in July 2008 were analyzed for DEET and its degradation products. Fifteen transformation products were identified in the water samples. DEET was detected in seven of the eight samples at concentrations of 0.6–155.55 ng/L (0.0006–0.156 µg/L). The detection limit was 0.5 ng/L (0.0005 µg/L) (Calza et al. 2011). Freshwater streams were monitored in Hessisches Ried region, Germany from September 2003 to September 2006 (Quednow and Puttmann 2009); 330 samples were collected on 13 different occasions at 26 locations. The mean concentration of DEET detected was 245 ng/L (0.245 µg/L), with the highest

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concentration (1.3 µg/L) occurring in June 2004. Overall, mean concentrations were higher in the summer months than during the other seasons.

Water samples taken from 0.5 m below the water surface of the Zhujiang and Shijing Rivers were collected in July and August 2011 (Yang et al. 2013). Concentrations of DEET were below the LOQ in three samples; however, concentrations ranged from 0.2 to 107 ng/L (0.0002–0.107 µg/L) in all other samples (n=24). The higher levels of DEET at some of the sites were attributed to its use as a pesticide in those areas. Water samples (n=10) taken from 0.5 m below the water surface of the Beijiang River were also collected in July and August 2011; concentrations of DEET were 3–47 ng/L (0.003–0.047 µg/L [ppb]).

Around Norway in 2002, DEET was detected in 12 seawater samples, into which sewage treatment plant effluents and non-treated sewage are discharged, at concentrations of 0.4–13 ng/L (0.0004–0.013 µg/L) (Weigel et al. 2004). Marine samples taken in February, May, and September 2011 and March 2012 from the northern Adriatic Sea approximately 50 cm below the surface contained DEET at concentrations of 0.349, 1.255, 4.995, and 0.460 ng/L, respectively (0.000349, 0.001255, 0.004995, and 0.00046 µg/L); the average LOQ was reported as 0.213 ng/L (0.000213 µg/L [ppb]) (Loos et al. 2013b).

Between November and December 2001, water samples were collected at several sites within a U.S. drinking water treatment facility in a heavily populated, urbanized drainage basin. DEET was detected in 3 of the 12 stream and raw water samples (25% frequency of detection). The highest concentration of DEET in samples of finished water was 0.066 µg/L (ppb) (reporting level 0.5 µg/L) (Stackelberg et al. 2004). DEET was not detected in 15 finished drinking water samples from four water filtration plants in San Diego County, California; the sample dates were between August 2001 and June 2002. DEET was, however, detected in 1 of 13 source water samples for four water filtration plants in San Diego County, California at a mean concentration of 0.131 µg/L (ppb); sample dates were August 2001 to November 2002 (Loraine and Pettigrove 2006). DEET was detected in two of six water samples from a waste water reclamation plant in San Diego County, California at a mean concentration of 1.31 µg/L; sample dates were September 2001 to June 2002 (Loraine and Pettigrove 2006). In samples taken during 2006 and 2007 from drinking water treatment plants across the United States, DEET was detected in the source water at 6 of 19 plants at a maximum concentration of 110 ng/L (0.11 µg/L) and a median concentration of 85 ng/L (0.085 µg/L) and in the finished water at 6 of 18 plants at a maximum concentration of 93 ng/L (0.093 µg/L) and a median concentration of 63 ng/L (0.063 µg/L [ppb]) (Benotti et al. 2009). In New York between May 2003 and January 2005, effluent concentrations, ranging from 0.3 to 15 µg/L

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(ppb), from WWTPs indicated that removal rates were minimal (Phillips et al. 2005). Effluent concentrations in Las Vegas, Nevada in June 2005 and January 2006 were on average between 0.123 and 0.188  $\mu\text{g/L}$  [ppb] (Snyder 2005).

DEET was detected in water samples taken from two locations on Assunpink Creek in Trenton, New Jersey. At the first collection site downstream from a WWTP effluent discharge, DEET was detected at levels of 51–99 ng/L (0.051–0.099  $\mu\text{g/L}$ ). At the second site, 2 miles further downstream, DEET was detected at 45–340 ng/L (0.045–0.34  $\mu\text{g/L}$ ) (Alvarez et al. (2005). DEET was analyzed for in 10 WWTP-influenced sites around the United States. Samples were taken upstream from the plant, from the effluent, and two samples were taken at varying distances downstream. DEET was reported in 70% of the samples, with a median concentration of 0.097  $\mu\text{g/L}$  and a maximum concentration of 2.1  $\mu\text{g/L}$ ; the reporting level was 0.5  $\mu\text{g/L}$  (Glassmeyer et al. 2005). In July 2006, DEET was detected at concentrations of 0.09, 0.02, 0.04, and 0.065  $\mu\text{g/L}$  (median detection level of 0.005  $\mu\text{g/L}$ ) in samples from sites on Wascana Creek, Saskatchewan, Canada. Samples were collected 31.8 km upstream from Regina, a sewage treatment plant, and 9.3, 59.8, and 104.8 km downstream from the sewage treatment plant, respectively. DEET was also detected at the same sampling sites in May 2007 (Waiser et al. 2011).

During October 2006–November 2007, Foster (2007) tested WWTPs in San Marcos, Texas and found that DEET was one of the most frequently detected compounds. The treatment plant uses activated sludge, granular activated carbon filtration, and ultraviolet disinfection. There was no detection of DEET 30 yards upstream from the effluent discharge. DEET was detected at a mean concentration of 1.7  $\mu\text{g/L}$  (ppb) in 100% of the influent samples; DEET was detected at a mean concentration of 0.023  $\mu\text{g/L}$  in 33% of the effluent samples and 0.009  $\mu\text{g/L}$  (ppb) in 33% of the samples 30 yards downstream from the effluent discharge (detection limit=14.5 ng/L).

Through 1998 and 1999, DEET was detected in the effluents of 11 out of 19 WWTPs located in Switzerland at concentrations under the detection limit up to 1.3  $\mu\text{g/L}$  (ppb) (Gerecke et al. 2002). Kim et al. (2007) reported a mean concentration of 0.0247  $\mu\text{g/L}$  (ppb) for DEET in seven WWTPs located in South Korea. These plants receive about 85% domestic waste and use mainly activated sludge treatment methods.

In northeastern Kansas, Lee and Rasmussen (2006) detected median levels of DEET at 1.4 and <0.5  $\mu\text{g/L}$  in the effluent of three trickling filter WWTPs and four activated sludge WWTPs (MRL 0.5  $\mu\text{g/L}$ ). In southeastern Miami, an activated sludge WWTP produced median effluent concentrations of

## 6. POTENTIAL FOR HUMAN EXPOSURE

approximately 0.20 µg/L (ppb) (method detection limit=0.14 µg/L) during spring and summer months of March and July 2004 (Lietz and Meyer 2006).

In 1984 and 1991, Eckel et al. (1993) detected, but did not quantify DEET in the leachate from Hipps Road Landfill, Jacksonville, Florida, a site that received waste in 1968 and 1969. In May 1990, DEET was detected, but not quantified, in three municipal landfill leachate samples in Gryta, Vasteras, Sweden (Oman and Hynning 1993).

DEET was detected in marine coastal areas along the Florida Keys following an underwater music festival in which human recreational activities occurred in and around the water. Samples were taken before, during, and after the festival. DEET concentrations ranged from not detected to 17 ng/L (0.017 µg/L) (Chaudhary et al. 2005). DEET was detected in coastal waters of Norway at levels of 4.2–240.8 ng/L (0.0042–0.2408 µg/L) (Langford et al. 2008).

Aronson et al. (2012) reported a study in which DEET produced mean concentrations ranging from 2.6 to 4.3 µg/L (ppb) in confined animal-feeding operation waste waters in Nebraska, while feed lot lagoons in Minnesota had concentrations under the method reporting level of 0.5µg/L (ppb) (Lee et al. 2004). Additionally, these authors compiled concentrations of DEET measured in published studies from 1996 to 2010 found in influent and effluent waste waters, and published studies from 1994 to 2010 of surface waters in and outside of the United States. Kim et al. (2007) studied rivers receiving WWTP effluents and found DEET in seven out of eight samples with a mean concentration of 0.022 µg/L (ppb) (method detection limit=1 ng/L).

DEET was detected in 98% of reclaimed water samples (n=55) collected from sprinkler systems used for daily irrigation in Florida. The water had received primary and secondary treatments not designed to remove micronutrients. One sample reached the maximum concentration of approximately 14,000 ng/L (14 µg/L), while the rest were <1.5 µg/L (Wang and Gardinali 2013).

#### 6.4.3 Sediment and Soil

No data were located on the environmental levels of DEET in sediment or soil.

#### 6.4.4 Other Environmental Media

No data were located on the levels of DEET in other environmental media.

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**6.5 GENERAL POPULATION AND OCCUPATIONAL EXPOSURE**

Exposure of the general population to DEET is expected to be relatively high based on its use as an insect and acarid repellent. Consumer products containing DEET are intended for direct application onto skin and/ or clothing while being worn. Products such as wrist bands or nets may also be impregnated with DEET. The general population is exposed to DEET via dermal contact after direct application of DEET insect repellents.

The Fourth National Report on Human Exposure to Environmental Chemicals (CDC 2009, 2017) includes results from the assessment of DEET levels in the National Health and Nutrition Examination Survey (NHANES) for urine samples from 4,512 members of the U.S. general population surveyed during the years 1999–2000 and 2001–2002. As shown in Table 6-1 and 6-2, the average for DEET was below the detection limit (0.449 µg/L) for the survey years 1999–2000 in each selected percentile. For the survey years 2001–2002, the total geometric mean, and the 50<sup>th</sup> and 75<sup>th</sup> percentiles were also below the detection limit (0.1 µg/L). The 90<sup>th</sup> and 95<sup>th</sup> percentiles were just above the LOD and reported DEET concentrations were 0.11 and 0.18 µg/L (Table 6-1), respectively, and the creatinine corrected values were 0.27 and 0.41 µg/g creatinine, respectively (Table 6-2). For the survey years 2007–2008 and 2009–2010, the average for DEET was again below the detection limit (0.089 µg/L) in each selected percentile. Because DEET undergoes oxidative metabolism in humans, more sensitive biomarkers for assessing DEET exposure are the metabolites DCBA and DHMB (Calafat et al. 2016), which are included in the updated tables, January 2017 of the Fourth National Report on Human Exposure to Environmental Chemicals (CDC 2017). As shown in Tables 6-3 and 6-5, for the survey years 2007–2008, the total geometric mean for DCBA was 3.50 µg/L, while that for DHMB was not determinable, and the respective 90<sup>th</sup> and 95<sup>th</sup> percentiles were 33.9 and 79.2 µg/L for DCBA and 0.229 and 0.780 µg/L for DHMB. For the survey years 2009–2010, the total geometric mean for DCBA was 4.54 µg/L, while that for DHMB was not determinable, and the respective 90<sup>th</sup> and 95<sup>th</sup> percentiles were 51.9 and 165 µg/L for DCBA and 0.455 and 1.34 µg/L for DHMB. In Tables 6-4 and 6-6, for the survey years 2007–2008, the total geometric mean for DCBA was 3.60 µg/g creatinine, while that for DHMB was not determinable, and the respective 90<sup>th</sup> and 95<sup>th</sup> percentiles were 27.3 and 70.8 µg/g creatinine for DCBA and 0.331 and 0.628 µg/g creatinine for DHMB. For the survey years 2009–2010, the total geometric mean for DCBA was 4.74 µg/g creatinine, while that for DHMB was not determinable, and the respective 90<sup>th</sup> and 95<sup>th</sup> percentiles were 44.6 and 131 µg/g creatinine for DCBA and 0.449 and 1.13 µg/g creatinine for DHMB.

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**Table 6-1. Geometric Mean and Selected Percentiles of Urine Concentrations of DEET (in µg/L) for the U.S. Population from the National Health and Nutrition Examination Survey (NHANES) 1999–2002, 2007–2010<sup>a</sup>**

	Survey years	Geometric mean (95% CI)	Selected percentiles (95% CI)				Sample size
			50 <sup>th</sup>	75 <sup>th</sup>	90 <sup>th</sup>	95 <sup>th</sup>	
Total	1999–2000 <sup>b</sup>	*d	<LOD <sup>f</sup>	<LOD	<LOD	<LOD	1,977
	2001–2002 <sup>b</sup>	*d	<LOD	<LOD	0.11 (0.10–0.14)	0.18 (0.14–0.22)	2,535
	2007–2008 <sup>c</sup>	*e	<LOD	<LOD	<LOD	<LOD	2,565
	2009–2010 <sup>c</sup>	*e	<LOD	<LOD	<LOD	<LOD	2,744
Age group							
6–11 years	1999–2000	*	<LOD	<LOD	<LOD	<LOD	480
	2001–2002	*	<LOD	<LOD	0.13 (0.10–0.18)	0.21 (0.12–0.56)	580
	2007–2008	*	<LOD	<LOD	<LOD	<LOD	380
	2009–2010	*	<LOD	<LOD	<LOD	<LOD	386
12–19 years	1999–2000	*	<LOD	<LOD	<LOD	<LOD	672
	2001–2002	*	<LOD	<LOD	0.13 (0.11–0.16)	0.22 (0.13–0.52)	829
	2007–2008	*	<LOD	<LOD	<LOD	<LOD	386
	2009–2010	*	<LOD	<LOD	<LOD	<LOD	400
20–59 years	1999–2000	*	<LOD	<LOD	<LOD	<LOD	825
	2001–2002	*	<LOD	<LOD	0.11 (<LOD–0.13)	0.17 (0.13–0.21)	1,126
	2007–2008	*	<LOD	<LOD	<LOD	<LOD	1,169
	2009–2010	*	<LOD	<LOD	<LOD	<LOD	1,307
≥60 years	2007–2008	*	<LOD	<LOD	<LOD	<LOD	630
	2009–2010	*	<LOD	<LOD	<LOD	<LOD	651
Gender							
Males	1999–2000	*	<LOD	<LOD	<LOD	<LOD	964
	2001–2002	*	<LOD	<LOD	0.11 (0.10–0.15)	0.18 (0.13–0.25)	1,191
	2007–2008	*	<LOD	<LOD	<LOD	<LOD	1,286
	2009–2010	*	<LOD	<LOD	<LOD	<LOD	1,343
Females	1999–2000	*	<LOD	<LOD	<LOD	<LOD	1,013
	2001–2002	*	<LOD	<LOD	0.11 (0.10–0.13)	0.17 (0.13–0.21)	1,344
	2007–2008	*	<LOD	<LOD	<LOD	<LOD	1,279
	2009–2010	*	<LOD	<LOD	<LOD	<LOD	1,401



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**Table 6-1. Geometric Mean and Selected Percentiles of Urine Concentrations of DEET (in µg/L) for the U.S. Population from the National Health and Nutrition Examination Survey (NHANES) 1999–2002, 2007–2010<sup>a</sup>**

	Survey years	Geometric mean (95% CI)	Selected percentiles (95% CI)				Sample size
			50 <sup>th</sup>	75 <sup>th</sup>	90 <sup>th</sup>	95 <sup>th</sup>	
<b>Race/ethnicity</b>							
Mexican Americans	1999–2000	*	<LOD	<LOD	<LOD	<LOD	688
	2001–2002	*	<LOD	<LOD	0.11 (<LOD–0.14)	0.13 (0.11–0.19)	678
	2007–2008	*	<LOD	<LOD	<LOD	<LOD	499
	2009–2010	*	<LOD	<LOD	<LOD	<LOD	600
Non-Hispanic blacks	1999–2000	*	<LOD	<LOD	<LOD	<LOD	518
	2001–2002	*	<LOD	<LOD	0.10 (<LOD–0.14)	0.14 (0.10–0.24)	700
	2007–2008	*	<LOD	<LOD	<LOD	<LOD	570
	2009–2010	*	<LOD	<LOD	<LOD	<LOD	504
Non-Hispanic whites	1999–2000	*	<LOD	<LOD	<LOD	<LOD	598
	2001–2002	*	<LOD	<LOD	0.11 (0.10–0.14)	0.18 (0.13–0.27)	956
	2007–2008	*	<LOD	<LOD	<LOD	<LOD	1,071
	2009–2010	*	<LOD	<LOD	<LOD	<LOD	1,199

<sup>a</sup>Data in this table come from the National Report on Human Exposure to Environmental Chemicals and Update Tables, which is continuously updated with new measurements. The most up-to-date data for environmental chemicals and reference ranges in the U.S. general population are available at the National Report website: <https://www.cdc.gov/exposurereport/>.

<sup>b</sup>CDC 2009.

<sup>c</sup>CDC 2017.

<sup>d</sup>Not calculated; the proportion of results below limit of detection (LOD) was too high to provide a valid result. The LODs for survey years 1999–2000 and 2001–2002 were 0.449 and 0.1 µg/L, respectively.

<sup>e</sup>Not calculated; the proportion of results below LOD was too high to provide a valid result. The LOD for survey years 2007–2008 and 2009–2010 was 0.089 µg/L.

<sup>f</sup><LOD means less than the limit of detection, which may vary for some chemicals by year and by individual sample.

CI = confidence interval

## 6. POTENTIAL FOR HUMAN EXPOSURE

**Table 6-2. Geometric Mean and Selected Percentiles of Urine Concentrations of DEET (Creatinine Corrected) ( $\mu\text{g/g}$  creatinine) for the U.S. Population from the National Health and Nutrition Examination Survey (NHANES) 1999–2002, 2007–2010<sup>a</sup>**

	Survey years	Geometric mean (95% CI)	Selected percentiles (95% CI)				Sample size
			50 <sup>th</sup>	75 <sup>th</sup>	90 <sup>th</sup>	95 <sup>th</sup>	
Total	1999–2000 <sup>b</sup>	*d	<LOD <sup>f</sup>	<LOD	<LOD	<LOD	1,977
	2001–2002 <sup>b</sup>	*d	<LOD	<LOD	0.27 (0.24–0.30)	0.41 (0.35–0.50)	2,534
	2007–2008 <sup>c</sup>	*e	<LOD	<LOD	<LOD	<LOD	2,563
	2009–2010 <sup>c</sup>	*e	<LOD	<LOD	<LOD	<LOD	2,744
Age group							
6–11 years	1999–2000	*	<LOD	<LOD	<LOD	<LOD	480
	2001–2002	*	<LOD	<LOD	0.33 (0.23–0.63)	0.64 (0.28–1.93)	580
	2007–2008	*	<LOD	<LOD	<LOD	<LOD	380
	2009–2010	*	<LOD	<LOD	<LOD	<LOD	386
12–19 years	1999–2000	*	<LOD	<LOD	<LOD	<LOD	672
	2001–2002	*	<LOD	<LOD	0.19 (0.15–0.24)	0.25 (0.19–0.49)	828
	2007–2008	*	<LOD	<LOD	<LOD	<LOD	384
	2009–2010	*	<LOD	<LOD	<LOD	<LOD	400
20–59 years	1999–2000	*	<LOD	<LOD	<LOD	<LOD	825
	2001–2002	*	<LOD	<LOD	0.27 (<LOD–0.32)	0.41 (0.37–0.50)	1,126
	2007–2008	*	<LOD	<LOD	<LOD	<LOD	1,169
	2009–2010	*	<LOD	<LOD	<LOD	<LOD	1,307
≥60 years	2007–2008	*	<LOD	<LOD	<LOD	<LOD	630
	2009–2010	*	<LOD	<LOD	<LOD	<LOD	651
Gender							
Males	1999–2000	*	<LOD	<LOD	<LOD	<LOD	964
	2001–2002	*	<LOD	<LOD	0.20 (0.17–0.25)	0.32 (0.25–0.44)	1,191
	2007–2008	*	<LOD	<LOD	<LOD	<LOD	1,285
	2009–2010	*	<LOD	<LOD	<LOD	<LOD	1,343
Females	1999–2000	*	<LOD	<LOD	<LOD	<LOD	1,013
	2001–2002	*	<LOD	<LOD	0.33 (0.29–0.37)	0.50 (0.41–0.58)	1,343
	2007–2008	*	<LOD	<LOD	<LOD	<LOD	1,278
	2009–2010	*	<LOD	<LOD	<LOD	<LOD	1,401

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**Table 6-2. Geometric Mean and Selected Percentiles of Urine Concentrations of DEET (Creatinine Corrected) ( $\mu\text{g/g}$  creatinine) for the U.S. Population from the National Health and Nutrition Examination Survey (NHANES) 1999–2002, 2007–2010<sup>a</sup>**

	Survey years	Geometric mean (95% CI)	Selected percentiles (95% CI)				Sample size
			50 <sup>th</sup>	75 <sup>th</sup>	90 <sup>th</sup>	95 <sup>th</sup>	
Race/ethnicity							
Mexican Americans	1999–2000	*	<LOD	<LOD	<LOD	<LOD	688
	2001–2002	*	<LOD	<LOD	0.19 (<LOD–0.23)	0.28 (0.23–0.35)	678
	2007–2008	*	<LOD	<LOD	<LOD	<LOD	498
	2009–2010	*	<LOD	<LOD	<LOD	<LOD	600
Non-Hispanic blacks	1999–2000	*	<LOD	<LOD	<LOD	<LOD	518
	2001–2002	*	<LOD	<LOD	0.13 (<LOD–0.15)	0.19 (0.14–0.27)	699
	2007–2008	*	<LOD	<LOD	<LOD	<LOD	569
	2009–2010	*	<LOD	<LOD	<LOD	<LOD	504
Non-Hispanic whites	1999–2000	*	<LOD	<LOD	<LOD	<LOD	598
	2001–2002	*	<LOD	<LOD	0.30 (0.27–0.35)	0.48 (0.39–0.55)	956
	2007–2008	*	<LOD	<LOD	<LOD	<LOD	1,071
	2009–2010	*	<LOD	<LOD	<LOD	<LOD	1,199

<sup>a</sup>Data in this table come from the National Report on Human Exposure to Environmental Chemicals and Update Tables, which is continuously updated with new measurements. The most up-to-date data for environmental chemicals and reference ranges in the U.S. general population are available at the National Report website: <https://www.cdc.gov/exposurereport/>.

<sup>b</sup>CDC 2009.

<sup>c</sup>CDC 2017.

<sup>d</sup>Not calculated; the proportion of results below limit of detection (LOD) was too high to provide a valid result. The LODs (not corrected for creatinine) for survey years 1999–2000 and 2001–2002 were 0.449 and 0.1  $\mu\text{g/L}$ , respectively.

<sup>e</sup>Not calculated; the proportion of results below LOD was too high to provide a valid result. The LOD (not corrected for creatinine) for survey years 2007–2008 and 2009–2010 was 0.089  $\mu\text{g/L}$ .

<sup>f</sup><LOD means less than the limit of detection for urine samples; not corrected for creatinine.

CI = confidence interval

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**Table 6-3. Geometric Mean and Selected Percentiles of Urine Concentrations of 3-(Diethylcarbamoyl) Benzoic Acid (DCBA) ( $\mu\text{g/L}$ ) for the U.S. Population from the National Health and Nutrition Examination Survey (NHANES) 2007–2010<sup>a</sup>**

	Survey years	Geometric mean (95% CI)	Selected percentiles (95% CI)				Sample size
			50 <sup>th</sup>	75 <sup>th</sup>	90 <sup>th</sup>	95 <sup>th</sup>	
Total	2007–2008	3.50 (2.64–4.64)	2.37 (1.88–3.10)	9.14 (5.61–14.5)	33.9 (20.5–53.1)	79.2 (37.9–145)	2,538
	2009–2010	4.54 (3.35–6.15)	3.40 (2.31–4.95)	13.8 (8.63–20.6)	51.9 (31.1–108)	165 (57.8–464)	2,735
Age group							
6–11 years	2007–2008	4.44 (3.73–5.29)	3.44 (2.70–5.87)	12.7 (9.54–15.9)	42.0 (24.2–70.4)	79.7 (44.9–114)	378
	2009–2010	6.44 (3.72–11.1)	5.35 (2.58–8.86)	18.5 (8.15–37.9)	83.8 (28.4–439)	316 (41.2–3970)	385
12–19 years	2007–2008	5.26 (3.47–7.98)	4.37 (2.68–5.98)	13.1 (6.81–25.8)	35.4 (20.4–71.2)	71.2 (30.7–700)	380
	2009–2010	6.58 (4.49–9.66)	4.63 (2.82–8.64)	18.9 (10.7–33.6)	87.8 (32.9–186)	186 (31.1–1130)	398
20–59 years	2007–2008	3.33 (2.56–4.35)	2.23 (1.83–2.90)	7.95 (5.05–14.5)	30.8 (17.4–53.1)	75.6 (39.3–131)	1,157
	2009–2010	4.39 (3.29–5.86)	3.33 (2.23–4.95)	14.0 (8.36–20.9)	51.4 (32.6–95.8)	138 (52.9–280)	1,300
≥60 years	2007–2008	2.78 (1.75–4.42)	1.64 (.936–3.06)	6.15 (3.08–16.9)	34.7 (16.3–75.4)	103 (32.4–200)	623
	2009–2010	3.42 (2.39–4.91)	2.13 (1.45–4.00)	9.63 (5.33–17.1)	35.4 (19.7–63.8)	103 (43.2–346)	652
Gender							
Males	2007–2008	4.15 (2.88–6.00)	2.90 (2.13–4.34)	11.3 (6.63–19.7)	37.7 (20.7–82.0)	112 (34.7–556)	1,269
	2009–2010	5.58 (3.94–7.90)	4.39 (2.67–6.24)	18.7 (10.8–30.6)	78.3 (37.3–174)	199 (96.2–525)	1,340
Females	2007–2008	2.97 (2.32–3.80)	2.06 (1.64–2.59)	6.84 (4.41–10.8)	30.8 (15.0–40.8)	52.6 (36.4–103)	1,269
	2009–2010	3.73 (2.79–4.98)	2.76 (1.87–4.24)	9.91 (6.35–15.9)	36.2 (22.4–70.4)	94.9 (40.2–278)	1,395

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**Table 6-3. Geometric Mean and Selected Percentiles of Urine Concentrations of 3-(Diethylcarbamoyl) Benzoic Acid (DCBA) ( $\mu\text{g/L}$ ) for the U.S. Population from the National Health and Nutrition Examination Survey (NHANES) 2007–2010<sup>a</sup>**

	Survey years	Geometric mean (95% CI)	Selected percentiles (95% CI)				Sample size
			50 <sup>th</sup>	75 <sup>th</sup>	90 <sup>th</sup>	95 <sup>th</sup>	
Race/ethnicity							
Mexican Americans	2007–2008	3.70 (2.57–5.33)	3.26 (1.87–5.17)	9.63 (5.93–17.6)	28.0 (14.7–69.1)	69.1 (27.5–133)	490
	2009–2010	2.63 (1.61–4.28)	2.03 (.932–4.71)	7.35 (4.22–14.5)	23.1 (12.5–48.2)	48.9 (26.0–94.3)	599
Non-Hispanic blacks	2007–2008	4.36 (3.18–5.96)	3.54 (2.24–6.04)	10.3 (6.78–17.4)	31.9 (19.3–51.6)	62.4 (40.4–103)	562
	2009–2010	3.91 (2.85–5.35)	3.22 (2.24–4.75)	9.53 (5.88–14.4)	23.4 (18.0–33.4)	38.4 (29.1–60.5)	497
Non-Hispanic whites	2007–2008	3.47 (2.35–5.14)	2.22 (1.65–3.19)	9.12 (4.82–17.0)	36.5 (17.7–82.0)	86.9 (32.9–356)	1,064
	2009–2010	5.48 (3.83–7.84)	4.31 (2.64–6.25)	17.7 (10.5–28.4)	67.9 (32.6–195)	200 (63.8–832)	1,199

<sup>a</sup>Data in this table come from the National Report on Human Exposure to Environmental Chemicals and Update Tables, which is continuously updated with new measurements. The most up-to-date data for environmental chemicals and reference ranges in the U.S. general population are available at the National Report website: <https://www.cdc.gov/exposurereport/>.

The limits of detection for survey years 2007–2008 and 2009–2010 were 0.93 and 0.475  $\mu\text{g/L}$ , respectively.

CI = confidence interval

Source: CDC 2017

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**Table 6-4. Geometric Mean and Selected Percentiles of Urine Concentrations of 3-(Diethylcarbamoyl) Benzoic Acid (DCBA) (Creatinine Corrected) ( $\mu\text{g/g}$  creatinine) for the U.S. Population from the National Health and Nutrition Examination Survey (NHANES) 2007–2010<sup>a</sup>**

	Survey years	Geometric mean (95% CI)	Selected percentiles (95% CI)				Sample size
			50 <sup>th</sup>	75 <sup>th</sup>	90 <sup>th</sup>	95 <sup>th</sup>	
Total	2007–2008	3.60 (2.79–4.65)	2.79 (2.14–3.55)	8.55 (5.49–13.2)	27.3 (17.8–47.9)	70.8 (34.1–170)	2,537
	2009–2010	4.74 (3.48–6.46)	3.35 (2.30–5.26)	12.9 (8.53–20.6)	44.6 (28.3–86.3)	131 (47.0–405)	2,735
Age group							
6–11 years	2007–2008	5.64 (4.72–6.75)	4.84 (3.65–5.78)	14.2 (10.7–19.8)	47.7 (34.2–55.1)	88.6 (47.9–182)	378
	2009–2010	8.72 (5.03–15.1)	6.42 (4.02–11.6)	23.5 (13.2–36.7)	75.4 (28.6–673)	365 (46.2–4,980)	385
12–19 years	2007–2008	4.08 (2.81–5.93)	2.96 (2.14–5.36)	11.0 (6.38–16.0)	24.3 (14.8–53.4)	53.4 (19.3–345)	379
	2009–2010	5.65 (3.76–8.50)	3.76 (2.56–6.22)	16.5 (7.29–31.7)	68.6 (25.3–182)	154 (25.3–1,270)	398
20–59 years	2007–2008	3.34 (2.61–4.27)	2.73 (1.99–3.46)	7.57 (4.92–12.1)	24.8 (14.9–44.7)	57.8 (30.9–117)	1,157
	2009–2010	4.41 (3.25–5.97)	2.98 (2.11–5.32)	11.7 (7.71–19.6)	39.1 (24.7–82.6)	112 (51.3–228)	1,300
≥60 years	2007–2008	3.42 (2.33–5.02)	2.47 (1.45–3.64)	7.33 (4.25–16.8)	33.8 (15.9–86.0)	93.3 (34.2–244)	623
	2009–2010	4.06 (2.95–5.59)	2.68 (2.10–3.96)	10.8 (6.79–15.9)	37.7 (22.2–51.1)	108 (42.7–393)	652
Gender							
Males	2007–2008	3.46 (2.46–4.87)	2.72 (1.76–3.73)	8.68 (5.34–14.4)	27.8 (16.9–69.4)	87.0 (27.8–403)	1,269
	2009–2010	4.97 (3.49–7.08)	3.29 (2.14–5.94)	14.7 (9.13–24.0)	60.0 (28.5–134)	185 (74.8–433)	1,340
Females	2007–2008	3.74 (2.99–4.68)	2.88 (2.27–3.63)	8.55 (5.36–13.2)	27.2 (16.2–46.3)	54.8 (34.2–117)	1,268
	2009–2010	4.54 (3.40–6.05)	3.35 (2.39–4.67)	11.8 (7.55–18.4)	35.3 (24.2–53.4)	77.6 (36.7–252)	1,395

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**Table 6-4. Geometric Mean and Selected Percentiles of Urine Concentrations of 3-(Diethylcarbamoyl) Benzoic Acid (DCBA) (Creatinine Corrected) ( $\mu\text{g/g}$  creatinine) for the U.S. Population from the National Health and Nutrition Examination Survey (NHANES) 2007–2010<sup>a</sup>**

	Survey years	Geometric mean (95% CI)	Selected percentiles (95% CI)				Sample size
			50 <sup>th</sup>	75 <sup>th</sup>	90 <sup>th</sup>	95 <sup>th</sup>	
Race/ethnicity							
Mexican Americans	2007–2008	3.79 (2.57–5.58)	3.60 (1.99–5.96)	9.91 (6.55–16.9)	27.8 (15.0–60.6)	60.6 (24.4–107)	490
	2009–2010	2.74 (1.75–4.28)	2.03 (1.12–5.18)	7.57 (3.92–14.0)	23.3 (13.6–31.8)	37.4 (23.6–90.5)	599
Non-Hispanic blacks	2007–2008	3.33 (2.46–4.49)	2.74 (1.91–3.76)	7.07 (4.88–11.9)	22.5 (12.6–45.1)	53.9 (28.7–103)	561
	2009–2010	2.98 (2.26–3.94)	2.41 (1.70–3.37)	6.73 (4.34–9.46)	16.5 (13.0–20.4)	30.6 (19.4–51.1)	497
Non-Hispanic whites	2007–2008	3.78 (2.69–5.32)	2.82 (2.05–4.02)	8.70 (5.23–14.9)	30.7 (16.7–57.2)	76.9 (26.7–432)	1,064
	2009–2010	5.97 (4.13–8.64)	4.41 (2.61–7.43)	17.4 (10.7–26.7)	61.1 (29.0–189)	189 (56.4–849)	1,199

<sup>a</sup>Data in this table come from the National Report on Human Exposure to Environmental Chemicals and Update Tables, which is continuously updated with new measurements. The most up-to-date data for environmental chemicals and reference ranges in the U.S. general population are available at the National Report website: <https://www.cdc.gov/exposurereport/>.

The limit of detection (not corrected for creatinine) for survey years 2007–2008 and 2009–2010 were 0.93 and 0.475  $\mu\text{g/L}$ , respectively.

CI = confidence interval

Source: CDC 2017

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**Table 6-5. Geometric Mean and Selected Percentiles of Urine Concentrations of N,N-Diethyl-3-(Hydroxymethyl) Benzamide (DHMB) ( $\mu\text{g/L}$ ) for the U.S. Population from the National Health and Nutrition Examination Survey (NHANES) 2007–2010<sup>a</sup>**

	Survey years	Geometric mean (95% CI)	Selected percentiles (95% CI)				Sample size
			50 <sup>th</sup>	75 <sup>th</sup>	90 <sup>th</sup>	95 <sup>th</sup>	
Total	2007–2008	*b	<LOD <sup>c</sup>	<LOD	0.229 (<LOD–0.525)	0.780 (0.326–1.51)	2,562
	2009–2010	*b	<LOD	<LOD	0.455 (0.162–0.956)	1.34 (0.644–3.10)	2,736
Age group							
6–11 years	2007–2008	*	<LOD	<LOD	0.275 (0.168–0.433)	0.640 (0.264–2.64)	380
	2009–2010	*	<LOD	<LOD	0.655 (<LOD–2.93)	2.82 (0.205–24.6)	385
12–19 years	2007–2008	*	<LOD	<LOD	0.356 (<LOD–0.879)	0.665 (0.165–8.14)	386
	2009–2010	*	<LOD	<LOD	0.472 (<LOD–1.59)	1.20 (0.201–4.11)	398
20–59 years	2007–2008	*	<LOD	<LOD	0.188 (<LOD–0.413)	0.767 (0.335–1.30)	1,167
	2009–2010	*	<LOD	<LOD	0.498 (0.172–0.956)	1.34 (0.729–2.29)	1,304
≥60 years	2007–2008	*	<LOD	<LOD	0.256 (<LOD–0.787)	0.787 (0.194–1.81)	629
	2009–2010	*	<LOD	<LOD	0.257 (0.106–0.512)	0.840 (0.521–2.46)	649
Gender							
Males	2007–2008	*	<LOD	<LOD	0.325 (0.091–0.909)	1.05 (0.249–4.86)	1,283
	2009–2010	*	<LOD	<LOD	0.744 (0.323–1.43)	1.81 (0.946–3.94)	1,339
Females	2007–2008	*	<LOD	<LOD	0.165 (<LOD–0.326)	0.512 (0.256–0.968)	1,279
	2009–2010	*	<LOD	<LOD	0.220 (<LOD–0.521)	0.796 (0.329–2.05)	1,397



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**Table 6-5. Geometric Mean and Selected Percentiles of Urine Concentrations of N,N-Diethyl-3-(Hydroxymethyl) Benzamide (DHMB) ( $\mu\text{g/L}$ ) for the U.S. Population from the National Health and Nutrition Examination Survey (NHANES) 2007–2010<sup>a</sup>**

Race/ethnicity	Survey years	Geometric mean (95% CI)	Selected percentiles (95% CI)				Sample size
			50 <sup>th</sup>	75 <sup>th</sup>	90 <sup>th</sup>	95 <sup>th</sup>	
Mexican Americans	2007–2008	*	<LOD	<LOD	0.216 (0.092–0.509)	0.509 (0.207–0.989)	499
	2009–2010	*	<LOD	<LOD	0.228 (<LOD–0.504)	0.507 (0.223–0.866)	598
Non-Hispanic blacks	2007–2008	*	<LOD	<LOD	0.310 (0.091–0.470)	0.640 (0.378–1.29)	567
	2009–2010	*	<LOD	<LOD	0.135 (<LOD–0.292)	0.449 (0.212–0.884)	503
Non-Hispanic whites	2007–2008	*	<LOD	<LOD	0.255 (<LOD–0.861)	0.884 (0.225–4.84)	1,071
	2009–2010	*	<LOD	<LOD	0.644 (0.182–1.34)	1.89 (0.770–5.34)	1,195

<sup>a</sup>Data in this table come from the National Report on Human Exposure to Environmental Chemicals and Update Tables, which is continuously updated with new measurements. The most up-to-date data for environmental chemicals and reference ranges in the U.S. general population are available at the National Report website: <https://www.cdc.gov/exposurereport/>.

<sup>b</sup>Not calculated; the proportion of results below limit of detection (LOD) was too high to provide a valid result. The LOD for survey years 2007–2008 and 2009–2010 was 0.083  $\mu\text{g/L}$ .

<sup>c</sup><LOD means less than the limit of detection, which may vary for some chemicals by year and by individual sample.

CI = confidence interval

Source: CDC 2017

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**Table 6-6. Geometric Mean and Selected Percentiles of Urine Concentrations of N,N-Diethyl-3-(Hydroxymethyl) Benzamide (DHMB) (Creatinine Corrected) ( $\mu\text{g/g}$  creatinine) for the U.S. Population from the National Health and Nutrition Examination Survey (NHANES) 2007–2010<sup>a</sup>**

	Survey years	Geometric mean (95% CI)	Selected percentiles (95% CI)				Sample size
			50 <sup>th</sup>	75 <sup>th</sup>	90 <sup>th</sup>	95 <sup>th</sup>	
Total	2007–2008	*b	<LOD <sup>c</sup>	<LOD	0.331 (<LOD–0.452)	0.628 (0.393–1.32)	2,560
	2009–2010	*b	<LOD	<LOD	0.449 (0.300–0.720)	1.13 (0.548–2.41)	2,736
Age group							
6–11 years	2007–2008	*	<LOD	<LOD	0.370 (0.289–0.524)	0.831 (0.347–1.37)	380
	2009–2010	*	<LOD	<LOD	0.572 (<LOD–3.40)	3.12 (0.370–18.4)	385
12–19 years	2007–2008	*	<LOD	<LOD	0.253 (<LOD–0.555)	0.544 (0.191–1.76)	384
	2009–2010	*	<LOD	<LOD	0.436 (<LOD–0.869)	0.869 (0.246–8.42)	398
20–59 years	2007–2008	*	<LOD	<LOD	0.331 (<LOD–0.441)	0.582 (0.441–0.866)	1,167
	2009–2010	*	<LOD	<LOD	0.468 (0.300–0.702)	1.10 (0.572–1.79)	1,304
≥60 years	2007–2008	*	<LOD	<LOD	0.394 (<LOD–0.701)	1.01 (0.389–2.48)	629
	2009–2010	*	<LOD	<LOD	0.395 (0.315–0.489)	0.875 (0.548–2.41)	649
Gender							
Males	2007–2008	*	<LOD	<LOD	0.300 (0.176–0.826)	0.866 (0.304–3.33)	1,282
	2009–2010	*	<LOD	<LOD	0.524 (0.280–1.39)	1.45 (0.718–3.16)	1,339
Females	2007–2008	*	<LOD	<LOD	0.341 (<LOD–0.393)	0.572 (0.419–0.734)	1,278
	2009–2010	*	<LOD	<LOD	0.419 (<LOD–0.488)	0.723 (0.458–1.85)	1,397

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**Table 6-6. Geometric Mean and Selected Percentiles of Urine Concentrations of N,N-Diethyl-3-(Hydroxymethyl) Benzamide (DHMB) (Creatinine Corrected) ( $\mu\text{g/g}$  creatinine) for the U.S. Population from the National Health and Nutrition Examination Survey (NHANES) 2007–2010<sup>a</sup>**

	Survey years	Geometric mean (95% CI)	Selected percentiles (95% CI)				Sample size
			50 <sup>th</sup>	75 <sup>th</sup>	90 <sup>th</sup>	95 <sup>th</sup>	
Race/ethnicity							
Mexican Americans	2007–2008	*	<LOD	<LOD	0.315 (0.203–0.446)	0.467 (0.337–0.720)	498
	2009–2010	*	<LOD	<LOD	0.297 (<LOD–0.401)	0.415 (0.299–0.718)	598
Non-Hispanic blacks	2007–2008	*	<LOD	<LOD	0.234 (0.175–0.411)	0.487 (0.315–1.05)	566
	2009–2010	*	<LOD	<LOD	0.221 (<LOD–0.262)	0.362 (0.246–0.648)	503
Non-Hispanic whites	2007–2008	*	<LOD	<LOD	0.343 (<LOD–0.583)	0.826 (0.349–2.99)	1,071
	2009–2010	*	<LOD	<LOD	0.531 (0.305–1.36)	1.59 (0.524–5.83)	1,195

<sup>a</sup>Data in this table come from the National Report on Human Exposure to Environmental Chemicals and Update Tables, which is continuously updated with new measurements. The most up-to-date data for environmental chemicals and reference ranges in the U.S. general population are available at the National Report website: <https://www.cdc.gov/exposurereport/>.

<sup>b</sup>Not calculated; the proportion of results below limit of detection (LOD) was too high to provide a valid result. The LOD (not corrected for creatinine) for survey years 2007–2008 and 2009–2010 was 0.083  $\mu\text{g/L}$ .

<sup>c</sup><LOD means less than the limit of detection, which may vary for some chemicals by year and by individual sample.

CI = confidence interval

Source: CDC 2017

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Records of human exposure to DEET and/or DEET and other products were compiled from centers reporting to the Toxic Exposure Surveillance System from 1993 to 1997 (Bell et al. 2002). There were 20,764 exposure cases identified. Cases involving infants, children, and teenagers accounted for 18.6, 64.5, and 3.1% respectively. Of all the exposures, 89.2% occurred at the subject's home due to misuse of the product. There were 20,346 cases that involved products intended for human use, while 418 of the cases involved DEET-containing veterinary products. Ingestion accounted for 51.8% of the exposure incidents. Ocular, dermal, and multiple route exposures accounted for 21.3, 10.5, and 13.4% respectively. Of all the cases, 6,267 involved products containing <11% DEET, 9,003 involved products containing between 11 and 50% DEET, and 2,111 involved products containing >50% DEET; 3,293 of the cases reported unknown concentrations of DEET. A similar study conducted between 1985 and 1989 evaluated 9,086 human exposures of any product containing DEET reported to Poison Control Centers (Veltri et al. 1994). Most of the exposures occurred between May and September when DEET use is at its highest. Close to two-thirds of the incidents resulted in minor symptoms or did not have any adverse effects. Forty-nine percent of the exposures were due to ingestion, 32% resulted from ocular exposure, 12% were reported from multiple exposure routes, 4.2% from dermal exposure, and 2% via inhalation. More than 65% of exposure cases involved children 2–5 years of age. In a more recent report (from the 2012 Annual Report of the American Association of Poison Control Centers' National Poison Data System [NPDS] 30th Annual Report) (AAPCC 2013), it is stated that there were 4,158 cases in which DEET was mentioned and 4,075 cases that involved solely DEET exposure. There were 3,759 cases reported as unintentional. The majority of the cases (2,316 cases) involved children  $\leq 5$  years old. The outcome of all the exposure incidents were typically minor (1,176 cases) or none at all (576 cases). Moderate (83 cases), major (3 cases), and deadly (2 cases) outcomes were rarely observed. Of the exposures reported, 88% did not produce symptoms that required treatment in a health care facility (Veltri et al. 1994).

Wu et al. (1979) found DEET in the urine sample of a 30-year-old male who applied a commercial product containing DEET 18 hours after exposure. Eight hours after application, the DEET concentration in the blood was reported at 0.3 mg%. It was concluded that DEET was absorbed through the skin and about 10–14% was excreted unchanged. Urinary metabolites such as N-ethyl-*m*-toluamide and *m*-carboxyl-N,N-diethylbenzoylamide were identified, but not quantified, in the study. In 1991, average exposure estimates were derived for DEET based on one application/day to typical amounts used per application (see Table 6-7). Daily exposure values determined were 12.10, 9.68, 21.05, and 37.63 mg/kg/day for adult males, adult females, children ages 13–17 years old, and children  $\leq 12$  years old, respectively. These values may underestimate actual exposure levels in some users as it is possible that some users may apply the product more than once per day (EPA 1998b, 1998c). Exposure

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assessments considering scenarios of individual adults who applied either a spray or aerosol product have been done following Standard Operating Procedures for Residential Pesticide Assessment developed by EPA's Health Effects Division (EPA 2012c). Individuals weighing 80 kg and applying spray or aerosol products with 98% active ingredient (a.i.) were reported to be treating themselves with 9,453 and 16,771 mg a.i./day, respectively (Table 6-7).

A survey by the DEET Joint Venture reported on the use of products containing DEET as an active ingredient: 37% of the U.S. population is expected to use insect repellents and 60% of this usage occurs in June and July. During these 2 months, repellents were used on an average of 7.5 and 5.6 days by adults and children, respectively (EPA 2002). The yearly averages for numbers of days in which insect repellents were used by the general population and children were 12.5 and 9.3 days, respectively (EPA 2002). It was estimated that either 5.9 g (aerosol), 1.0 g (lotion), or 2.3 g (pump spray) are applied as a single application either directly to skin or clothing (EPA 2002).

DEET was detected in urine samples from eight national park employees who applied approximately 1 g of lotion containing 71% DEET daily to their skin and clothes for 1 week. The DEET concentration in the urine collected mid-week ranged from <180 to 5,960 µg/L. In a laboratory study, two of nine male volunteers ages 18–34 years, who applied a DEET-containing lotion, had quantifiable levels in their urine. Levels for the subject with higher readings were 2,020, 900, and 1,050 µg/L respectively at 4, 12.5, and 22.0 hours after application. The urine concentration of the second subject with quantifiable concentrations at the last time point reported was 3-fold less at 310 µg/L. The remaining seven volunteers had levels <90 µg/L (LOD=90 µg/L); below the limit of quantification (LOQ=180 µg/L) of DEET in their urine. The highest concentration quantified was 2,020 µg/L at 4 hours after application and the lowest concentration was 310 µg/L at 22 hours after application. Blood samples from the nine volunteers had concentrations of DEET <LOQ to 1.17 µg/g (the LOQ for serum samples was 0.18 µg/g) (Smallwood et al. 1992).

Although exposure from contaminated drinking water is minimal compared to that of exposure via dermal application, DEET has been found at trace levels in water intended for human consumption (Benotti et al. 2009; Calza et al. 2011; Kim et al. 2007).

Two prenatal urine samples, the first at ~13 weeks of gestation and the second at ~26 weeks of gestation, were collected from 538 pregnant women (≥18 years of age) living in the Salinas Valley of California

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**Table 6-7. Estimated Daily DEET Exposures by Consumers Using Insect Repellents**

Category of exposure	Amount of DEET per application (mg)	Body weight (kg)	Daily exposure (mg/kg/day)
Adult male	952.25	78.70	12.10
Adult female	649.31	67.10	9.68
Child, 13–17 years old	1,065.24	50.60	21.05
Child, ≤12 years old	940.83	25.00	37.63

Sources: EPA 1998b, 1998c

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**Table 6-8. Estimating DEET Exposures by Spray Treatment**

Repellent <sup>a</sup>	Amount of DEET in product (mg a.i. <sup>b</sup> /mg product)	Formulation application rate (mg product/cm <sup>2</sup> skin)	Fraction of body exposed	Surface area to body weight ratio (cm <sup>2</sup> /kg)	Exposure time (hours/day)	Application frequency (number/hour)	Exposure (mg a.i./kg/day) <sup>c</sup>	Exposure (mg a.i./individual/day) <sup>d</sup>
Pump spray, adult human	0.98	0.62	0.75	280	3.7	0.25	118	9,453
Aerosol spray, adult human	0.98	1.10	0.75	280	3.7	0.25	210	16,771

<sup>a</sup>Without sunscreen.

<sup>b</sup>Active ingredient.

<sup>c</sup>Exposure estimated by multiplying the values in first six columns.

<sup>d</sup>Individual exposure estimated by multiplying estimated body weight (e.g., 80 kg) by exposure (mg a.i./kg/day).

Source: EPA 2012c

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during 1999–2000. The LOD of the analytical method was reported as 0.1 µg/L. For the first and second samples, the maximum levels detected were 1.9 and 0.3 µg/L, the 95<sup>th</sup> percentile levels were 0.1 µg/L and <LOD, and detection frequencies were 5.6 and 1.9%, respectively (Castorina et al. 2010). The maximum concentrations were higher than the 95<sup>th</sup> percentile values reported during the latest monitoring period (2009–2010) by CDC (2017) in Table 6-1.

Cheng et al. (2006) reported finding DEET in air of the Lower Fraser Valley of Canada due to its widespread use during summer. The lowest levels measured (0.53–0.78 mg/m<sup>3</sup>) at a remote location and were considered to be the ambient background for the area resulting from spraying livestock in that rural area. Higher levels in the urban forest were 3.03 ng/m<sup>3</sup> during the day and 1.25 ng/m<sup>3</sup> at night. The highest levels were in nearby Golden Ear Provincial Park, measuring 11.1–11.4 ng/m<sup>3</sup> in the day and up to 37.1 ng/m<sup>3</sup> at night when insect density and DEET use may have been greatest.

### 6.6 EXPOSURES OF CHILDREN

This section focuses on exposures from conception to maturity at 18 years in humans. Differences from adults in susceptibility to hazardous substances are discussed in Section 3.7, Children's Susceptibility.

Children are not small adults. A child's exposure may differ from an adult's exposure in many ways. Children drink more fluids, eat more food, breathe more air per kilogram of body weight, and have a larger skin surface in proportion to their body volume than adults. A child's diet often differs from that of adults. The developing human's source of nutrition changes with age: from placental nourishment to breast milk or formula to the diet of older children who eat more of certain types of foods than adults. A child's behavior and lifestyle also influence exposure. Children crawl on the floor, put things in their mouths, sometimes eat inappropriate things (such as dirt or paint chips), and may spend more time outdoors. Children also are generally closer to the ground and have not yet developed the adult capacity to judge and take actions to avoid hazards (NRC 1993).

Data regarding the exposure of children to DEET indicate that dermal exposure from direct application of consumer products containing DEET is the most likely route. Inhalation is possible during aerosol product application, albeit a minor concern for exposure; additionally, hand-to-mouth behavior may result in oral exposure. Application of sunscreens containing DEET may result in unintentional overexposure to children if the sunscreen is applied repeatedly throughout the day as many consumer sunscreen products suggest. In 2012, AAPCC (2013) reported that 57%, or 2,316 case reports, of exposure to DEET was in



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children  $\leq 5$  years of age. This may indicate a propensity for parents to apply DEET more liberally to protect their young children from insect bites, rather than a differential susceptibility. A recent interim review of DEET by the EPA, under the Registration Review Program, states that DEET is approved for use on children with no age restriction or percentage of DEET in the product; however, DEET should not be applied by children under 10 and application should follow the guidelines stated on specific product labels (EPA 2014i). In addition, the AAP recommends that repellents used on children should not contain more than 30% DEET and that no repellents should be used on infants below the age of 2 months (AAP 2015). The CDC concurs with this use profile as adjusted by AAP (CDC 2015).

Daily exposure estimates for DEET, assuming one application per day and standard body weights, were calculated as 21.05 mg/kg/day for children 13–17 years old, and 37.63 mg/kg/day for children  $\leq 12$  years old, in comparison to estimates of 93.68 mg/kg/day for adult females, and 12.10 mg/kg/day for adult males. These values may underestimate actual exposure due to individual consumer use patterns and do not include exposure via inhalation or oral routes, although these are judged to be minor (EPA 1998b, 1998c).

Menon and Brown (2005) documented patterns of children's exposure to DEET products as a result of their direct use as insect repellents. Between 31 and 65% of the subjects did not follow recommended procedures described in Chapter 1 of this document for the proper use of the products with respect to children, resulting in conditions that could lead to unnecessary overexposure. For example, when applying DEET to the facial area, first apply to your hands and then rub the product onto your face. Avoid direct spraying to the face as this could cause the product to get into your eyes, mouth, or lungs. And, be sure to take off DEET products before going to bed (by showering or using a wash cloth) to avoid overexposure. Do not apply to children's hands, and do not allow children to handle products containing DEET since this can increase internal exposure through hand-to-mouth activities typical of some children.

DEET exposure may occur during pregnancy. Schaefer and Peters (1992) reported a case in which a pregnant woman living in Africa applied a lotion with 25% DEET to her arms and legs once or twice a day during pregnancy. Bradman et al. (2003) did not detect DEET in amniotic fluid samples (15–18 weeks of gestation) from 100 women in California (LOD = 0.4  $\mu\text{g/L}$ ). However, a study conducted from July 2003 to May 2004 of 150 women detected DEET in maternal serum samples at 1.82 to 18.84 ng/g and in corresponding cord serum at 2.06–13.07 ng/g (Barr et al. 2010). DEET was the most frequently detected (degree of frequency = 100%) pesticide in both maternal and cord serum samples of

## 6. POTENTIAL FOR HUMAN EXPOSURE

150 women in New Jersey at concentrations of 1.819–18.844 and 2.060–13.671 pg/mL, respectively (LOD=0.01 pg/mL) (Yan et al. 2009).

In 2004, Arcury et al. (2007) evaluated urine samples from 60 farm children (1–6 years old) in eastern North Carolina. Ten percent of the children had detectable levels of the metabolite for DEET (LOD=0.1 ng/mL) in their urine.

### 6.7 POPULATIONS WITH POTENTIALLY HIGH EXPOSURES

Workers in industries that manufacture and formulate DEET and DEET-containing products are likely to be at higher risk than the general population for DEET exposure. People who work or recreate outdoors (e.g., park rangers, hikers, hunters, campers) are more likely to be exposed to higher levels of DEET through the use of products containing this substance as opposed to people who work and recreate indoors (i.e., city dwellers) (Smallwood et al. 1992). Consumers who use commercial products containing DEET regularly, as a preventative measure for warding off insect bites, are exposed to higher levels of DEET than the general population who do not directly use DEET products. Children have the potential to be overexposed through misuse of the product (Bell et al. 2002).

### 6.8 ADEQUACY OF THE DATABASE

Section 104(i)(5) of CERCLA, as amended, directs the Administrator of ATSDR (in consultation with the Administrator of EPA and agencies and programs of the Public Health Service) to assess whether adequate information on the health effects of DEET is available. Where adequate information is not available, ATSDR, in conjunction with NTP, is required to assure the initiation of a program of research designed to determine the health effects (and techniques for developing methods to determine such health effects) of DEET.

The following categories of possible data needs have been identified by a joint team of scientists from ATSDR, NTP, and EPA. They are defined as substance-specific informational needs that if met would reduce the uncertainties of human health assessment. This definition should not be interpreted to mean that all data needs discussed in this section must be filled. In the future, the identified data needs will be evaluated and prioritized, and a substance-specific research agenda will be proposed.

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**6.8.1 Identification of Data Needs**

**Physical and Chemical Properties.** The physical chemical properties of DEET are summarized in Chapter 4 (HSDB 2001; O'Neil et al. 2013; Weeks et al. 2012). No data needs are identified.

**Production, Import/Export, Use, Release, and Disposal.** No information is available in the TRI database on facilities that manufacture or process 2-hexanone because this chemical is not required to be reported under Section 313 of the Emergency Planning and Community Right-to-Know Act (Title III of the Superfund Amendments and Reauthorization Act of 1986) (EPA 2005).

**Environmental Fate.** Transport, partitioning, and bioconcentration data are available for DEET. The fate of DEET in WWTPs has been summarized (ECHA 2010; Weeks et al. 2012). Biodegradation in aquifer slurries and standard tests are available; however, no studies were located that assess biodegradation in soils.

**Bioavailability from Environmental Media.** No data were identified that assess the bioavailability of DEET from environmental media such as soil and foods.

**Food Chain Bioaccumulation.** Studies are available that indicate that DEET does not bioconcentrate in aquatic organisms and is not expected to bioaccumulate in the food chain (CITI 1992). No data needs are identified.

**Exposure Levels in Environmental Media.** Reliable monitoring data for the levels of DEET in contaminated media at hazardous waste sites are needed so that the information obtained on levels of DEET in the environment can be used in combination with the known body burden of DEET to assess the potential risk of adverse health effects in populations living in the vicinity of hazardous waste sites.

Monitoring data are available for DEET in air (Balducci 2012; Cheng et al. 2006) and water (Brausch and Rand 2011; Glassmeyer et al. 2005; Kolpin et al. 2002; Sandstrom et al. 2005). No monitoring data were located for DEET in soil and sediment.

**Exposure Levels in Humans.** Exposure levels of DEET in human biological samples are available (CDC 2009; Wu et al. 1979). Continued biological monitoring of human serum and urine samples is useful since DEET is contained and used in many consumer products used by a high percentage of the

## 6. POTENTIAL FOR HUMAN EXPOSURE

population. This information is necessary for assessing the need to conduct health studies on these populations.

**Exposures of Children.** Children are exposed to DEET by the same routes that affect adults (primarily dermal exposure). Continued monitoring of children's exposure to DEET is considered a data need. Child health data needs relating to susceptibility are discussed in Section 3.12.2, Identification of Data Needs: Children's Susceptibility.

**Exposure Registries.** The information amassed in the National Exposure Registry facilitates the epidemiological research needed to assess adverse health outcomes that may be related to exposure to this substance; however, no exposure registries for DEET were located. DEET is not currently one of the compounds for which a sub-registry has been established in the National Exposure Registry. DEET will be considered in the future when chemical selection is made for sub-registries to be established.

### 6.8.2 Ongoing Studies

No ongoing environmental fate studies for DEET were identified using the NIH RePORTER version 6.1.0 or the Defense Technical Information Center (DTIC) online database.