6. POTENTIAL FOR HUMAN EXPOSURE

6.1 OVERVIEW

DEHP has been identified in at least 737 of the 1,613 hazardous waste sites that have been proposed for inclusion on the EPA National Priorities List (NPL) (HazDat 2002). However, the number of sites evaluated for DEHP is not known. The frequency of these sites can be seen in Figure 6-1. Of these sites, 730 are located within the United States, 5 are located in the Commonwealth of Puerto Rico, and 1 is located in the Virgin Islands, and 1 is located in Territory of Guam (the Commonwealth of Puerto Rico, the Virgin Islands, and Territory of Guam are not shown in Figure 6-1).

DEHP is a widely used chemical that enters the environment both through disposal of industrial and municipal wastes in landfills and by leaching into consumer products stored in plastics. It tends to sorb strongly to soils and sediments and to bioconcentrate in aquatic organisms. Biodegradation is expected to occur under aerobic conditions. Sorption, bioaccumulation, and biodegradation are likely to be competing processes, with the dominant fate being determined by local environmental conditions.

The principal route of human exposure to DEHP is oral. Much of the monitoring database is old and might not represent current exposures, especially since the uses of DEHP in certain applications has been changing (Mannsville Chemical Products Corporation 1999; Wilkinson and Lamb 1999). Some recent estimates of the average total daily individual ambient exposures to DEHP of 0.210–2.1 mg/day (in a 70-kg adult) have been proposed (Doull et al. 1999; Huber et al. 1996; NTP 2000b; Tickner et al. 2001). Populations residing near hazardous waste disposal sites or municipal landfills might be subject to higher than average levels of DEHP in ambient air and drinking water. Even so, the concentrations of DEHP in these media will be greatly limited by the low volatility and low water solubility of DEHP. Occupational exposures might be significant, but the highest exposures to DEHP result from medical procedures such as blood transfusions (e.g., estimated upper bound limit of 8.5 mg/kg/day) or hemodialysis (e.g., estimated upper bound limit of 0.36 mg/kg/day), during which DEHP might leach from plastic equipment into biological fluids (FDA 2001h). Exposures of neonates to DEHP can be especially high as a result of some medical procedures; total parenteral nutrition (TPN) administration (e.g., estimated upper bound limit of 2.5 mg/kg/day), and extracorporeal membrane oxygenation (ECMO) (e.g., estimated upper bound limit of 14 mg/kg/day) (FDA 2001h).

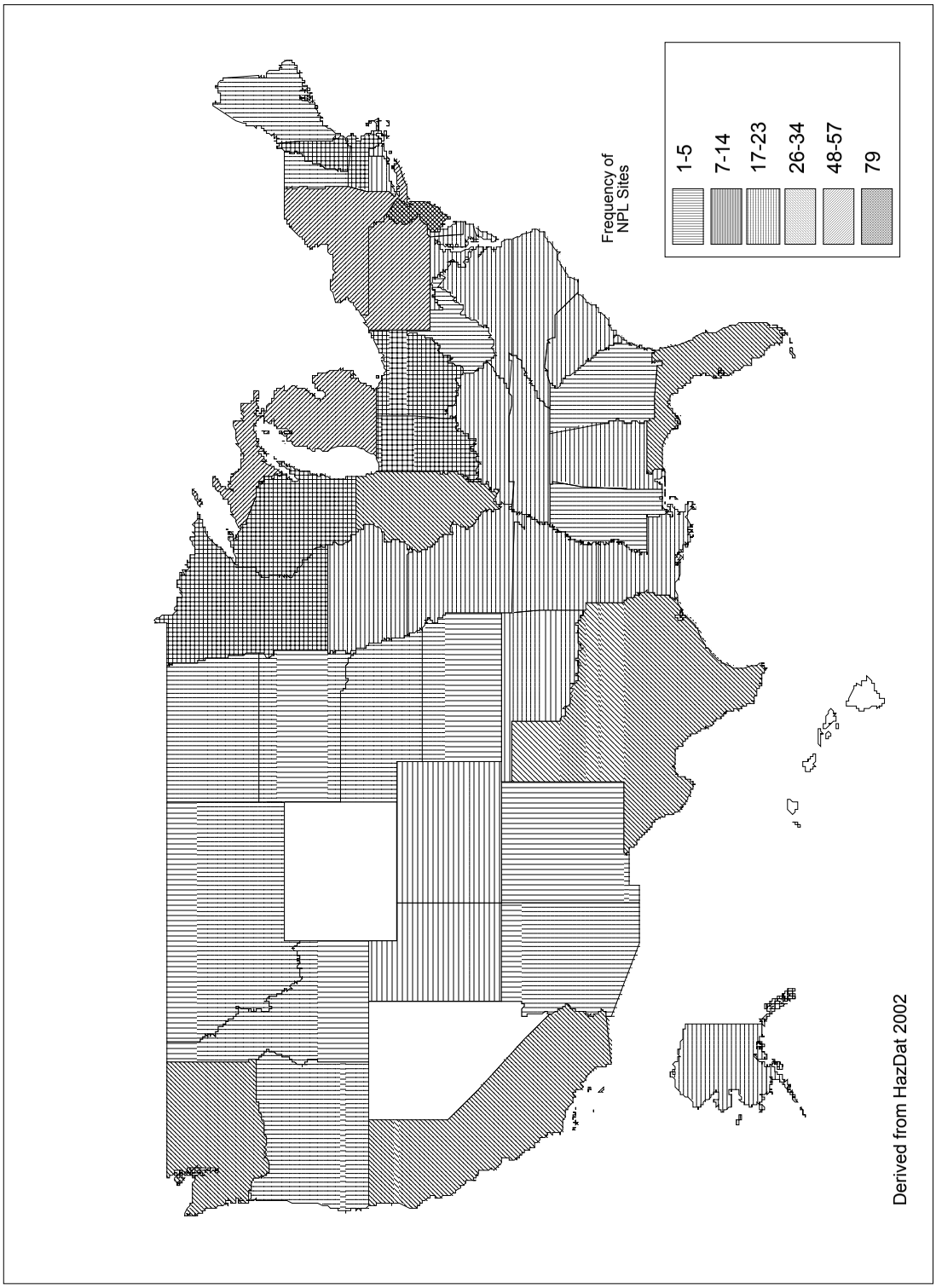

Figure 6-1. Frequency of NPL Sites with DEHP Contamination

When DEHP is present in the environment it is usually at very low levels. It should be noted that it is very difficult to determine these low levels accurately since DEHP is a ubiquitous laboratory contaminant. Laboratory contamination might cause false positives to be reported in the literature. Laboratory contamination often undermines the credibility of the data and, therefore, reported concentrations of DEHP in environmental samples must be carefully reviewed.

6.2 RELEASES TO THE ENVIRONMENT

Industrial manufacturers, processors, and users of DEHP are required to report the quantities of this substance released to environmental media annually (EPA 1988d). The data compiled in the Toxics Release Inventory (TRI99 2001), are for releases in 1999 to air, water, soil, and transfers for offsite disposal. These data are summarized in Table 6-1. Total releases (rounded to three-place accuracy) of DEHP to the environment in 1999 were approximately 264,000 pounds (approximately 120 metric tons) (TRI99 2001), of which approximately 229,000 pounds (104 metric tons), or about 87% of the total, were released to the air. Another 32,000 pounds (14.5 metric tons) or approximately 12% of the total were released to the land, while less than 1.1% (2,880 pounds, 1.3 metric tons) were released to water. The TRI data should be used with caution because only certain types of facilities are required to report. This is not an exhaustive list.

Industrial releases are only a fraction of the total environmental releases of DEHP. Release of DEHP into the environment is thought to originate from diffuse sources, mainly from end-uses of DEHP (e.g., as an additive to plastics). Disposal of plastic products containing DEHP (see Section 5.3) is also a possible source of environmental release (Bauer and Herrmann 1997; EPA 1981). Quantitative information on releases of DEHP to specific environmental media are discussed below.

6.2.1 Air

As presented in Chapter 4, DEHP has a relatively low vapor pressure and Henry's law constant, as well as a relatively high octanol/water partition coefficient and soil sorption coefficient. This combination of properties is consistent with a chemical that is found to only a limited extent in air (see Staples et al. 1997). Nonetheless, DEHP appears to be a common air contaminant that is present globally in the low ng/m^3 concentrations (see Section 6.4), although specific information that quantifies emissions of DEHP to air appears to be insufficient to account for this apparent widespread presence. For example, while

Table 6-1. Releases to the Environment from Facilities that Produce, Process, or Use DEHP

State[b]	Number of facilities	Reported amounts released in pounds per year[a]						
		Air[c]	Water	Underground injection	Land	Total on-site release[d]	Total off-site release[e]	Total on and off-site release
AL	1	10	No data	No data	19,880	19,890	500	20,390
AR	4	7,005	0	No data	No data	7,005	7,544	14,549
CA	16	7,086	5	No data	No data	7,091	27,249	34,340
CO	1	5	No data	No data	No data	5	250	255
CT	3	26,858	3	No data	No data	26,861	2,272	29,133
FL	3	605	No data	No data	No data	605	No data	605
GA	6	1,370	No data	No data	250	1,620	20,316	21,936
IA	3	7	No data	No data	No data	7	1,639	1,646
IL	17	2,773	5	No data	0	2,778	16,774	19,552
IN	7	762	0	No data	No data	762	11,978	12,740
KS	6	290	5	No data	No data	295	28,915	29,210
KY	3	10	No data	No data	0	10	250	260
LA	2	4	0	No data	No data	4	45	49
MA	15	4,269	No data	No data	No data	4,269	24,103	28,372
MD	3	3,044	0	No data	0	3,044	10,000	13,044
MI	5	1,186	No data	No data	7,764	8,950	12,928	21,878
MN	6	174	0	No data	No data	174	4,572	4,746
MO	12	2,797	0	No data	1,685	4,482	139,505	143,987
MS	4	511	5	No data	No data	516	30,417	30,933
NC	14	44,189	250	No data	No data	44,439	114,938	159,377
NE	3	1,653	No data	No data	No data	1,653	41,515	43,168
NH	2	No data	No data	No data	No data	No data	No data	No data
NJ	11	10,418	0	No data	750	11,168	3,630	14,798
NV	2	2,696	No data	No data	No data	2,696	1,500	4,196
NY	6	3,294	No data	No data	No data	3,294	73,613	76,907

Table 6-1. Releases to the Environment from Facilities that Produce, Process, or Use DEHP *(continued)*

State[b]	Number of facilities	Reported amounts released in pounds per year[a]						
		Air[c]	Water	Underground injection	Land	Total on-site release[d]	Total off-site release[e]	Total on and off-site release
OH	24	8,725	9	No data	0	8,734	39,446	48,180
OK	3	60,669	No data	No data	No data	60,669	17,740	78,409
PA	15	7,124	3	0	0	7,127	30,354	37,481
PR	6	572	No data	No data	No data	572	93,992	94,564
RI	3	229	No data	No data	No data	229	6,646	6,875
SC	7	4,186	No data	No data	No data	4,186	24,580	28,766
SD	1	No data	No data	No data	No data	No data	250	250
TN	11	5,863	2,342	No data	No data	8,205	50,933	59,138
TX	15	12,921	3	No data	2,000	14,924	45,513	60,437
UT	2	5	No data	No data	No data	5	No data	5
VA	3	6,427	No data	No data	No data	6,427	No data	6,427
VT	1	No data	No data	No data	No data	No data	No data	No data
WA	4	257	250	No data	5	512	3,150	3,662
WI	6	454	No data	No data	No data	454	56,960	57,414
WV	1	500	No data	No data	No data	500	No data	500
Total	257	228,948	2,880	0	32,334	264,162	944,017	1,208,179

Source: TRI99 2001

[a]Data in TRI are maximum amounts released by each facility.
[b]Post office state abbreviations are used.
[c]The sum of fugitive and stack releases are included in releases to air by a given facility.
[d]The sum of all releases of the chemical to air, land, water, and underground injection wells.
[e]Total amount of chemical transferred off-site, including to publicly owned treatment works (POTW).

monitoring data show that elevated fallout concentrations of DEHP are associated with industrial activity (Thurén and Larsson 1990), elevated fallout concentrations were only seen near a stack, and no elevated concentrations could be seen 2 km away from the stack. In addition, these authors could not correlate DEHP fallout rates with specific sources or transport routes on a nationwide basis in Sweden. They found no "distributional patterns or gradient", which possibly suggests that any local patterns were obscured by DEHP contribution from other sources or that emission sources of roughly equal magnitude are diffuse. By contrast, a pattern associating distance from sources and concentration was seen with DEHP by Ritsema et al. (1989) in Lake Yssel in the Netherlands, while for other lower-molecular-weight phthalate esters, no pattern was evident. The authors suggested that an upstream source is the dominant mechanism by which DEHP enters the lake. The possibility of many diffuse sources of DEHP is potentially supported by some of the uses. For example, some of the products that use DEHP include thin sheets and coatings, such as floor tiles, shower curtains, tablecloths, and furniture upholstery (see Chapter 5). These products characteristically have large surface area-to-volume ratios, which might allow DEHP to volatilize more readily relative to other products with smaller surface area-to-volume ratios. Cadogan et al. (1994) and Cadogan and Howick (1996) reported that an indoor emission rate of $2.3x10^{-4}$ mg/second-m^2 at 25 EC has been calculated for all phthalate plasticizers in products such as wall coverings, flooring, upholstery, and wire insulation. These authors used this emission estimate to calculate overall releases of phthalate esters to air. Cadogan and Howick (1996) also noted that approximately 47% of the phthalate ester used is DEHP. Applying this DEHP use percentage to their emission estimates, total end-use emissions of DEHP to the air from indoor household uses in Western Europe in 1990 is approximately 300 tons per year. Emissions from exterior end uses were estimated to be 2,600 tons per year for DEHP (the authors noted that this estimate was not well defined). These estimates support the conclusion that the major sources of DEHP are from end-uses and that these represent a geographically diffuse source. Finally, Jones et al. (1996) estimated that between 0.001 and 3.6 metric tons of DEHP are emitted per year (depending on assumptions about vapor equilibria and mass transfer used in model calculations) from sewer manholes in a large U.S. city having an average DEHP sewage concentration of 26 µg/L.

Industrial atmospheric emissions (rounded to three-place accuracy) of DEHP reported to the TRI for 1999 totaled about 229,000 pounds (104 metric tons) (TRI99 2001) and comprise about 87% of the total releases. It has been estimated that less than 3% of the total U.S. domestic supply of DEHP is released to air (EPA 1981). Based on an estimated current U.S. supply in 1998 of about 285 million pounds (241 million pounds in 1999) (see Section 5.1), the estimated annual atmospheric emission of DEHP from all sources in the United States is about 6.6 million pounds in 1998 (5.6 million in 1999).

DEHP has been identified in air samples collected from 7 of the 737 NPL hazardous waste sites where it was detected in some environmental media (HazDat 2002).

6.2.2 Water

Total releases of DEHP to water are also estimated to be about 3% of domestic supplies (EPA 1981). Some of these releases are expected to be in industrial effluents from the manufacture and processing of DEHP (EPA 1981). Industrial releases (rounded to three-place accuracy) to surface water reported to the TRI for 1998 were about 2,880 (1.3 metric tons) (TRI99 2001).

DEHP was detected in 13% of 86 samples of urban storm water runoff evaluated for the National Urban Runoff Program, at concentrations ranging from 7 to 39 ppb (Cole et al. 1984). In some locations, storm and sanitary sewers are separated so that storm water runoff in these locations directly enters surface water. Even in locations with combined storm and sanitary sewers, DEHP is still expected to enter the environment, but probably to a lesser extent. For example, Stubin et al. (1996) reported that DEHP was present in 48% of the influent and 12% of the effluent samples taken from New York City sewage treatment plants during 1989–1993. Thus, storm water runoff, even when it goes through a sewage treatment plant, might enter the environment. In addition, DEHP also appears to be present in the treatment plant influent whether or not it receives storm water. DEHP has also been reported in waste water from a petrochemical plant (Castillo et al. 1998), leachate from industrial and municipal landfills (Brown and Donnelly 1988; Castillo et al. 1998; Ghassemi et al. 1984; Roy 1994), and sewage sludge (O'Connor 1996). It is anticipated that water from all these sources enters the environment and might contain DEHP.

DEHP has been identified in ground water samples collected from 650 sites and surface water samples at 215 of the 737 NPL hazardous waste sites where it was detected in some environmental media (HazDat 2002).

6.2.3 Soil

The principal source of DEHP release to land is likely the disposal of industrial and municipal waste to landfills (EPA 1981). Industrial releases (rounded to three-place accuracy) of DEHP to land reported to the TRI for 1998 total about 32,000 pounds (TRI99 2001). In addition, another 944,000 pounds is transferred off-site for treatment (including publically owned treatment works) or disposal and some of

this material might be discharged to soils. Municipal wastes might contain substantial quantities of DEHP-containing plastics, which might significantly increase the total quantity of DEHP released to land. Based on an estimate that 92% of U.S. domestic supplies of DEHP are released to landfills (EPA 1981) and current U.S. domestic supplies in 1998 of approximately 285 million pounds (241 million pounds in 1999) (Section 5.1), about 262 million pounds (222 million pounds in 1999) of DEHP are deposited in landfills annually. Bauer and Herrmann (1997) reported the concentration of DEHP in various fractions of household wastes from the regions of Bayreuth and Straubling in Germany. The wastes included food waste, paper for recycling, unusable paper, cardboard, plastic films, other plastics, textiles, 8–40 mm screened fraction, <8 mm screened fraction, compound packing waste, compound materials, and disposable diapers. DEHP was found in all of the fractions. It is anticipated that household waste from continental Europe is similar to the United States, so that the same profile would be expected in both places. Further information on this study is presented in Section 6.4.4.

Land application of sewage sludge might also release DEHP to soil. The National Sewage Sludge Survey estimated that mean DEHP concentrations in sludge range from 55 to 300 ppm, with a national mean of 75 ppm (EPA 1990d). It is also estimated that about 42% of sewage sludge generated in the United States annually, or 5.1 billion pounds, is applied to land. Another 20% (2.4 billion pounds) is deposited in landfills, and 14% (1.7 billion pounds) are incinerated (EPA 1990d). Using the national mean concentration and a total of 7.5 billion pounds of sludge deposited in soils, sludge accounts for approximately 7,500 pounds of DEHP released to soils annually.

This compound has also been reported in ocean sediments at levels up to 25 ppm at points of urban sewage outfall (Swartz et al. 1985), and in 100% of the sediments in rivers near combined sewer overflows in New Jersey (Ianuzzi et al. 1997). Concentrations of phthalates, including DEHP, are approximately 10 times higher in stream sediments that are influenced by urban activity than in areas under other land-use activities (Lopes and Furlong 2001).

DEHP has been identified in soil samples collected from 582 sites and sediment samples at 248 of 737 NPL hazardous waste sites where it was detected in some environmental media (HazDat 2002).

6.3 ENVIRONMENTAL FATE

6.3.1 Transport and Partitioning

DEHP is ubiquitous in air at low concentrations (e.g., 0.06–5.0 ng/m^3) (Eisenreich et al. 1981; Ligocki et al. 1985a), is in both the vapor phase and associated with particulates, and is subject to both wet (rain and snow) and dry (wind and settling) deposition on the Earth's surfaces. Eisenreich et al. (1981) calculated that wet and dry deposition of DEHP into the five Great Lakes amounted to approximately 47.7 metric tons per year, which corresponds to an average fallout rate of 16.2 µg/m^2 per month. A similar average fallout rate of 23.8 µg/m^2 per month (the range was 5.96–195.5 µg/m^2 per month) was reported by Thurén and Larsson (1990) for DEHP in Sweden. Thurén and Larsson (1990) also estimated the median fallout concentration to be 48 ng/L. The authors noted that the fallout rate for DEHP decreased with increasing distance (but only up to 2 km away) from a stack at a facility that used DEHP (see Section 6.2), but noted that no specific overall concentration or fallout patterns were observed. This is consistent with diffuse sources of DEHP. In addition, DEHP has been found in Antarctic surface and sub-surface snow (up to 3 m deep), and in pack ice (Desideri et al. 1994, 1998), as well as in the atmosphere over the Gulf of Mexico (Giam et al. 1980), suggesting that DEHP can be transported for long distances. Thus, the DEHP measured in one part of the world might have originated elsewhere. This transport is likely particle sorbed DEHP (Atlas and Giam 1981) because vapor phase DEHP reacts rapidly with hydroxyl radicals in the atmosphere (see Section 6.3.2.1), while particle-sorbed DEHP does not react rapidly with hydroxyl radicals. Nearly half the DEHP detected in the atmosphere over the Gulf of Mexico was in the particulate phase (Giam et al. 1980). Atmospheric fallout is negatively correlated with temperature so that less DEHP is subject to fallout in the summer than in the winter (Staples et al. 1997; Thurén and Larsson 1990). This is in keeping with a higher proportion of the atmospheric DEHP in the vapor state in the warm summer and less in the cold winter, and further indicates that the partitioning between particles and vapor are controlled by vapor pressure. DEHP is removed from the atmosphere by both wet (rain and snow) and dry (wind and settling) deposition (Atlas and Giam 1981; Eisenreich et al. 1981; Ligocki et al. 1985a, 1985b).

In water, DEHP is predominantly sorbed to suspended particulates and sediments, but some remains dissolved in the aqueous phase. Volatilization is not a dominant transport process. Volatilization from water and soil is not expected to be important, based on the low Henry's law constant (estimated value 1.71×10^{-5} atm-m^3/mol; Staples et al. 1997). It has been estimated that the evaporative half-life of DEHP

from water would be about 15 years (EPA 1979), and that only about 2% of DEHP loading of lakes and ponds would be volatilized (Wolfe et al. 1980a).

Adsorption onto soils and sediments is a significant sink for DEHP. DEHP released to water adsorbs strongly to suspended particulates and sediments (Al-Omran and Preston 1987; Staples et al. 1997; Sullivan et al. 1982; Wolfe et al. 1980a). Distribution of DEHP between the water column and the sediments was modeled for several types of freshwater aquatic environments (Wolfe et al. 1980a). Between 69 and 99% of DEHP was estimated to partition to the sediments. Adsorption of DEHP to marine sediments might be greater than adsorption to freshwater sediments, due to reduced solubility of DEHP in saltwater (Al-Omran and Preston 1987; Sullivan et al. 1982; Zhou and Liu 2000). Levels of DEHP in a marine environment ranged from 0.1 to 0.7 ppb in the water and from 280 to 640 ppb in the suspended particulates (Preston and Al-Omran 1989). DEHP shows greater adsorption to the smaller size particle fractions of suspended particulates or colloids (Al-Omran and Preston 1987; Zhou and Liu 2000). Complexation of DEHP with fulvic acid, a compound associated with humic substances in water and soil, might increase solubilization and thus increase the mobility of DEHP in aquatic systems (Johnson et al. 1977). Ritsema et al. (1989) noted that DEHP in the River Rhine was mainly associated with suspended particulates, but on some sampling days, dissolved DEHP was at a higher concentration than the sorbed material. By contrast, in Lake Yssel, DEHP concentrations in the suspended material were approximately 100 times higher than the dissolved material. In addition, the authors reported that a distinct concentration gradient was noted across the lake suggesting that DEHP entered the lake from the River Yssel rather than nonpoint sources as was the case with some other phthalates.

Percolation of DEHP through the soil to groundwater might occur during times of rapid infiltration. DEHP concentrations were generally reduced by infiltration through a soil column, but all column effluents contained measurable levels (Hutchins et al. 1983). In hazardous waste sites, the presence of common organic solvents such as alcohols and ketones might increase the solubility of relatively insoluble compounds such as DEHP, thereby increasing the amounts that might leach from the waste site into subsoil and groundwater (Nyssen et al. 1987). This is consistent with the measurement of DEHP in leachate of some landfills at levels in excess of its usual water solubility (see Section 6.2.2).

Bioconcentration of DEHP has been observed in invertebrates, fish, and terrestrial organisms. Mean bioconcentration factors (BCFs) have been reported for algae (3,173±3,149, two species), molluscs (1,469±949, five species), crustacea (1,164±1,182, four species), insects (1,058±772, three species), polychaetes (422, one species), fish (280±230, five species), and amphibians (605, one species) have been

compiled by Staples et al. (1997). Residues of DEHP have been found in the organs of terrestrial animals such as rats, rabbits, dogs, cows, and humans (EPA 1979). However, accumulation of DEHP will be minimized by metabolism, and biomagnification of DEHP in the food chain is not expected to occur (EPA 1979; Johnson et al. 1977; Staples et al. 1997; Wofford et al. 1981). Several metabolites of DEHP might be detected in animal tissues (Johnson et al. 1977). Uptake of DEHP from soil by plants has also been reported (EPA 1986; O'Connor 1996).

6.3.2 Transformation and Degradation

6.3.2.1 Air

Reaction of DEHP vapor with hydroxyl radicals in the atmosphere has been predicted, with an estimated half-life of about 6 hours using the Atmospheric Oxidation Program (Meylan and Howard 1993). The atmospheric half-life, however, is expected to be longer for DEHP adsorbed to atmospheric particulates. Based on the estimated half-life alone, extensive transport of DEHP would not be expected and concentrations in Antarctic snow would not be predicted. Nonetheless, DEHP appears to be present in urban and rural atmospheres (see Section 6.4), and its transport might be mainly in the sorbed state. Data confirming this degradation pathway have not been located. Direct photolysis and photooxidation are not likely to be important (Wams 1987).

6.3.2.2 Water

Biodegradation might be an important fate process for DEHP in water under aerobic, but not anaerobic, conditions (O'Connor et al. 1989; O'Grady et al. 1985; Sugatt et al. 1984; Tabak et al. 1981; Thomas et al. 1986). DEHP was significantly biodegraded (>95%) after gradual acclimation of the microbial population over a period of about 3 weeks under conditions of the static-flask and shake-flask screening tests (Sugatt et al. 1984; Tabak et al. 1981). In the shake flask study using an acclimated inoculum, initial biodegradation was low on days 2 and 3 but increased 5–10-fold by days 6 and 7; degradation to carbon dioxide was 87% at 28 days (Sugatt et al. 1984). The reported half-life of DEHP due to microbial activity in river water is about 1 month (Wams 1987). In freshwater, degradation has been reported to range from 0 to >99%, and is dependent on many variables including temperature (Staples et al. 1997). Reported removal of DEHP from aqueous systems by activated sludge biodegradation under aerobic conditions ranged from 70 to >99%, and from 0 to 90% in waste water depending on the microbial strains present and other variables (Kurane 1986; Nasu et al. 2001; O'Grady et al. 1985; Staples et al. 1997). In spite of

the many reported rapid degradation rates, DEHP has been found in sewage sludge (O'Connor 1996) and in sewage treatment plant effluents (Stubin et al. 1996) indicating that under actual sewage treatment plant conditions (which are more rigorous than environmental waters), DEHP is not always completely degraded, but rather becomes sorbed to sludge solids. Nonetheless, DEHP does not appear to be accumulating in the environment so that biodegradation is removing the apparent constant influx of DEHP. Under anaerobic conditions, biodegradation of DEHP is slower (O'Connor et al. 1989; Staples et al. 1997; Wams 1987).

Chemical hydrolysis of DEHP occurs too slowly to be important (Howard 1989; Staples et al. 1997). The estimated half-life for DEHP hydrolysis in water is 100 years (Wams 1987).

6.3.2.3 Sediment and Soil

Biodegradation of DEHP also occurs in soil, but at a slower rate than in water, since adsorption onto the soil organic matter reduces the availability of DEHP for degradation (Cartwright et al. 2000; Cheng et al. 2000; Wams 1987). According to Cartwright et al. (2000), DEHP is reported to be recalcitrant in soil and, as such, is predicted to account for the majority of phthalate contamination in the environment. Many other environmental factors, in addition to soil organic content, influence the rate of DEHP biodegradation (Cartwright et al. 2000; Gejlsberg et al. 2001). In sediments, optimum degradation of DEHP occurred at high concentration, warm temperatures, and in a nutrient-rich system (Johnson et al. 1984). Anaerobic biodegradation of DEHP in sediments was reported to occur, but more slowly than under aerobic conditions (Johnson et al. 1984).

6.4 LEVELS MONITORED OR ESTIMATED IN THE ENVIRONMENT

One problem that is encountered when reviewing the concentrations of DEHP in environmental water samples is evaluating the accuracy of the reported values of DEHP dissolved in water. Many of the concentrations of DEHP that have been reported for environmental water samples often exceed the solubility of DEHP in distilled or deionized water (Staples et al. 1997). Evaluating the values is complicated by the fact that a true solubility of DEHP in water has been difficult to determine experimentally, with values ranging between 0.0006 and 0.40 mg/L depending on the method of analysis (Staples et al. 1997). In addition, the solubility of DEHP in aqueous environmental media can be greatly affected by the types and concentration of dissolved organics in the sampled water; for example, humic substrates in landfill leachates (Staples et al. 1997). Another complication to determining the

concentration of DEHP in environmental water samples is the possible introduction of DEHP from other sources (Howard et al. 1985). For example, the measurement of DEHP in water can be confounded by a number of sampling problems. Samples can be contaminated by additional amounts of DEHP contained in sampling devices and laboratory containers. Since DEHP is a common laboratory contaminant, many times laboratory and field blanks show concentrations similar to those in the media under study. Sampling of water through the air-water interface can be contaminated by DEHP that is contained in surface films, due to the limited solubility of DEHP in water and a density that is slightly lower than water. Consequently, the reliability of the values that have been reported to represent the concentration of DEHP dissolved in water will have to be judged upon the quality of the sampling and analytical techniques used to measure DEHP in aqueous environmental media.

6.4.1 Air

As presented in Chapter 4, DEHP has a relatively low vapor pressure and Henry's law constant, as well as a relatively high octanol/water partition coefficient and soil sorption coefficient. This combination of properties is consistent with a chemical that is found to only a limited extent in air. Nonetheless, DEHP appears to be ubiquitous in air with urban air having somewhat higher concentrations than air in rural or uninhabited areas. The monitoring studies reported here appear to have taken reasonable efforts to eliminate contamination from their analyses. DEHP has been reported over the Pacific and Atlantic Oceans at mean levels of approximately $1.4x10^{-6}$ mg/m^3 ($3.2x10^{-7}$–$2.6x10^{-6}$ mg/m^3, 0.32–2.68 ng/m^3) (Atlas and Giam 1981; Giam et al. 1980), in outdoor air in Sweden at a median concentration of $2.0x10^{-6}$ mg/m^3 ($2.8x10^{-7}$–$77.0x10^{-6}$) mg/m^3, 0.28–77.0 ng/m^3) (Thurén and Larsson 1990), over Portland, Oregon at a mean level of $3.9x10^{-7}$ mg/m^3 ($6.0x10^{-8}$–$9.4x10^{-7}$ mg/m^3, 0.06–0.94 ng/m^3) (Ligocki et al. 1985a), and over the Great Lakes at a mean concentration of $2.0x10^{-6}$ mg/m^3 ($5.0x10^{-7}$–$5.0x10^{-6}$ mg/m^3, 0.50–5.0 ng/m^3) (Eisenreich et al. 1981). DEHP was not among the four phthalate esters detected in industrialized areas along the Niagara River (Hoff and Chan 1987). DEHP was detected but not quantified in a forest atmosphere in Germany (Helmig et al. 1990). Average atmospheric concentrations reported in the literature appear to be within a relatively narrow range regardless of whether monitored over oceans or in industrial areas. This might suggest that DEHP is mainly emitted from many small sources that are geographically diffuse and that it sorbs to atmospheric particulates that provide a reservoir of DEHP that is not degraded by hydroxyl radicals.

DEHP levels in indoor air might be higher due to slow volatilization from plastic products (EPA 1981; Wams 1987). As noted in Section 6.2.1, Cadogan et al. (1994) reported that an indoor overall emission

rate of $2.3x10^{-4}$ mg/second-m^2 at 25 EC has been calculated for all phthalate plasticizers in products such as wall coverings, flooring, upholstery, and wire insulation. The air from rooms recently covered with polyvinyl chloride (PVC) tiles contained 0.15–0.26 mg/m^3 (150,000–260,000 ng/m^3) phthalate esters (EPA 1981). Indoor air levels in rooms with new flooring could be about 0.2–0.3 mg/m^3 (Wams 1987).

DEHP was detected in samples (one from a commercial site and five from residential sites) of both dust and air obtained from all sites (Rudel et al. 2001). In dust, the concentration of DEHP ranged from 69.4 to 524 µg/g dust, with a mean concentration of 315 µg/g dust. In air, 4/6 sites had DEHP at concentrations above the minimum detection limit (MDL), ranging from 0.02 to 0.114 µg/m^3, with a mean concentration of 0.061 µg/m^3. In another study, indoor measurements of DEHP taken in six homes in the spring of 2000 ranged from 0.04 to 0.23 µg/m^3 (Otake et al. 2001). In one workplace (plastic melting facility), a value of 11.5 µg/m^3 was measured in air (Rudel et al. 2001).

Emission of DEHP from PVC wall coverings (containing 30% phthalic esters) was measured in a test chamber at room temperature, maximum concentration of 0.94 µg/m^3 for DEHP in air over 14-day test period (Udhe et al. 2001). Other citations within this reference noted that DEHP in test chambers was not detectable at room temperature, but maximum concentrations of 5.2 and 2 µg/m^3 were measured at 60 and 40 EC, respectively. Increases in DEHP emissions with increasing ambient temperature are especially important within car interiors, where DEHP concentrations in air have been shown to range from 1 µg/m^3 at room temperature to 34 µg/m^3 at 65 EC (Udhe et al. 2001).

6.4.2 Water

DEHP has been detected frequently in surface water, rainwater, and groundwater in the United States at concentrations generally in the low ppb range. It also has been detected in finished drinking water, but no literature was located reporting positive detections within the past 15 years. Roy (1994) reported a range of 34–7,900 µg/L in U.S. landfill leachate. Concentrations of DEHP have been measured at 0.6–2,400 ppb in surface waters, and at 0.04–420 ppb in groundwater obtained from private wells, offsite from landfills and facilities that use DEHP in manufacturing processes (HazDat 2002). Canter and Sabatini (1994) reported that the Biscayne aquifer in Florida had a maximum DEHP concentration of 8,600 µg/L, but no DEHP was detected in the municipal well fields that draw water from that aquifer. Eckel et al. (1993) also reported the presence of DEHP in the groundwater in Florida. DEHP was detected in petrochemical plant waste waters and industrial landfill leachate at <0.1–30 µg/L (Castillo et al. 1998) and in New York City municipal treatment plant effluents up to 50 µg/L (Stubin et al. 1996).

DEHP was detected in 24% of 901 surface water samples recorded in the STORET database at a median concentration of 10 ppb (Staples et al. 1985) and in water samples from four of the five Great Lakes (IJC 1983). DEHP was also found in water samples from several U.S. rivers (DeLeon et al. 1986; Hites 1973; Sheldon and Hites 1979). Reported concentrations ranged from 0.5 to 1 ppb. Average concentrations of DEHP in seawater ranging from 0.005 to 0.7 ppb have also been reported (Giam et al. 1978a; McFall et al. 1985b). DEHP was detected in drinking water concentrates from several U.S. cities (EPA 1984).

Bauer and Herrmann (1997) reported that DEHP was present in the leachate from various fractions of household wastes from the regions of Bayreuth and Straubling in Germany. The wastes included food waste, paper for recycling, unusable paper, cardboard, plastic films, other plastics, textiles, 8–40 mm screened fraction, <8 mm screened fraction, compound packing waste, compound materials, and disposable diapers. Approximately 50 kg of these wastes were cut into 5–10 cm pieces and placed in laboratory fermenters then flooded with water. Stable methanogenic conditions were obtained in 3 months. Leachate from a mixture of all waste categories except food waste contained a maximum of 147 µg/L of DEHP, while leachate from a mixture of waste categories limited to plastic films, other plastics, textiles, 8–40 mm screened fraction, <8 mm screened fraction, compound materials contained a maximum of 56 µg/kg DEHP. The authors were careful to exclude inadvertent sources of phthalate esters. This report demonstrates that DEHP is present in European household waste and that it leaches from that waste to percolating water. The extent to which this occurs in a landfill is unclear as is whether or not the dissolved DEHP leaches to groundwater after leaving landfills.

6.4.3 Sediment and Soil

Monitoring data for DEHP in soil were not located. DEHP was detected in both marine and freshwater sediments at average levels ranging from 6.6 to 1,500 ppb. Maximum values are usually observed near industrial effluent discharge points (Fallon and Horvath 1985; Murray et al. 1981; Ray et al. 1983). The highest value reported in the United States was 7,800 ppb in a marine sediment from Portland, Maine (Ray et al. 1983), and a value of 1,480 ppm was reported in a sediment sample from a river in Sweden (Thuren 1986). In the New York Bight (a sector of the Middle Atlantic Ridge adjoining the New York and New Jersey shorelines), which is an area containing disposal sites for dredging mud, sewage sludge, and industrial acid waste, DEHP has been measured in sediments at concentrations ranging from 0.1 to 10.1 ppm (Friedman et al. 2000). Iannuzzi et al. (1997) reported that DEHP was present in every sediment sample taken adjacent to combined sewer overflows to the Passaic River in New Jersey at concentrations between 960 and 27,000 µg/kg (a total of 40 samples). Of the 431 stream bed sediments

collected from throughout the United States, 39.2% showed DEHP concentrations, with a median concentration of 180 µg/kg (the high concentration was 17,000 µg/kg) (Lopes et al. 1997). DEHP was reported in 40% of 367 sediment samples recorded on the STORET database at a median concentration of 1,000 ppb (Staples et al. 1985) and in sediments near a hazardous waste site (Hauser and Bromberg 1982).

6.4.4 Other Environmental Media

DEHP has been found in several kinds of food. Fish and other seafood have been reported to be contaminated with concentrations ranging from 2 to 32,000 ppb (DeVault 1985; Giam and Wong 1987; Giam et al. 1975; McFall et al. 1985a; Ray et al. 1983; Stalling et al. 1973; Williams 1973). DEHP was detected in 33% of 139 biota samples (not necessarily edible) recorded on the STORET database at a median concentration of 3,000 ppb (Staples et al. 1985). DEHP has also been reported in processed canned and frozen fish in Canada at concentrations up to 160 ppb (Williams 1973).

DEHP can become an indirect additive in packaged foods due to its use in plastic wraps, heat seal coatings for metal foils, closure seals for containers, and printing inks for food wrappers and containers (Ministry of Agriculture Fisheries and Foods 1990); however, DEHP has not been used in food film wrap products for over 20 years (Mannsville Chemical Products Corporation 1999). Monitoring data indicate that DEHP residues are generally low in U.S. foods, but the available data are in excess of 10 years old and might not be representative of current conditions. DEHP has been detected in such foods as milk, cheese, meat, margarine, eggs, cereal products, baby food, infant formula, in addition to fish (Cerbulis and Byler 1986; EPA 1981; Petersen and Breindahl 2000). Most samples contained less than 1 ppm DEHP, but fatty foods had higher levels. Chocolate bars contained DEHP at levels up to 2.4 ppm (Castle et al. 1989). Although one study found that levels of DEHP in fatty foods such as milk, cheese, and meat did not differ significantly from background levels (CMA 1986), high levels of DEHP in "blank" samples and other analytical problems indicate that laboratory contamination might have confounded the results.

DEHP has been detected in indoor dust samples. Øie et al. (1997) reported that sedimented dust samples from 38 dwellings in Oslo, Norway contained an average of 640 µg/mg sedimented dust (100–1,610 µg/g), while suspended particulate matter from six dwellings contained an average of 600 µg/g (24–94 µg/g).

Blood products available for transfusions might be contaminated with DEHP due to leaching from the plastic equipment used to collect and store the blood. Reported concentrations of DEHP in blood products stored in PVC bags are: whole blood (2–620 ppm); platelet concentrates (23.4–267 ppm); red cell concentrates (4.3–152 ppm); and plasma (4.3–1,230 ppm) (Ching et al. 1981b; Cole et al. 1981; Contreras et al. 1991; Dine et al. 1991; FDA 2001h; Jaeger and Rubin 1972; Loff et al. 2000; NTP 2000b; Rock et al.1978; Shintani 2000; Sjoberg et al. 1985c; Vessman and Rietz 1974). DEHP was also detected in intravenous fluids, such as saline and glucose, used for parenteral therapy of hospitalized patients, at levels ranging from 9 to 13 ppb (Ching et al. 1981b). Karle et al. (1997) reported that DEHP concentrations at the end of the blood prime in extracorporeal membrane oxygenation circuits in an *in vitro* study had mean values of 18.3, 21.8, and 19.3 µg/mL for different circuits and was dependent on the surface area of each circuit. After 3 days, DEHP concentrations in infants averaged 4.9±4.0 µg/mL. Shneider et al. (1991) reported that serum DEHP concentrations varied depending on the nature of the treatment. They reported that for an infant cardiopulmonary bypass, pediatric hemodialysis, exchange transfusion, and extracorporeal membrane oxygenation, serum DEHP concentrations ranges were 1.1–5.1, 0.4–4.2, 5.4–21.5, and 18–98 µg/mL, respectively.

Soft plastic products available for use by infants might contain low levels of DEHP. DEHP was detected in four commercial pacifiers at concentrations of 31–42% by weight (Lay and Miller 1987). However, current levels of DEHP in these products have been greatly reduced, since manufacturers of toys and pacifiers voluntarily agreed to reduce the use of DEHP in their products (Wilkinson and Lamb 1999), although some PVC toys manufactured in a small number of foreign countries have been reported to contain up to 11–19% DEHP (Stringer et al. 2000). DEHP was the most common plasticizer in soft PVC products intended for children until the early 1980s. Manufacturers of PVC pacifiers and teethers set a limit of 3% by weight DEHP as a voluntary standard (Wilkinson and Lamb 1999). This limit should effectively eliminate the intentional addition of DEHP as a plasticizer in those products manufactured under the voluntary guidelines for DEHP content. In fact, the U.S. Consumer Product Safety Commission has reported that DEHP could not be found in pacifiers or bottle nipples currently on the market (Consumer Product Safety Commission 1998, 1999d). Except for one manufacture whose pacifier and bottle nipple products were found to contain diisooctylphthalate, all other known manufacturers of pacifiers and bottle nipples used latex or silicone instead of PVC in their products.

As presented in Section 6.4.2 above, Bauer and Herrmann (1997) reported that mixed household waste contained DEHP. Table 6-2 summarizes the concentration of DEHP detected in various categories of waste. The authors also calculated that 177.5–1,469.5 mg/kg DEHP was present in the waste on a dry-

weight basis and constituted the most commonly found phthalate ester, constituting 91.9–93.3% of the total phthalates found in the waste.

6.5 GENERAL POPULATION AND OCCUPATIONAL EXPOSURE

The general population is exposed to DEHP via oral, dermal, and inhalation routes of exposure. DEHP is present in environmental media and in numerous consumer articles that are used world-wide (see Chapter 4). Estimates of the average total daily individual ambient exposure to DEHP in the United States have ranged from 0.21 to 2.1 mg/day (David 2000; Doull et al. 1999; Huber et al. 1996; Kohn et al. 2000; Tickner et al. 2001). These estimates do not include workplace air exposures or exposures to DEHP offgassing from building materials. DEHP exposures in the Canadian population were estimated in 1994 to be 8.9–9.1, 19, 14, 8.2, and 5.8 µg/kg body weight/day for age groups 0–0.5, 0.5–4, 5–11, 12–19, and 20–70 years, respectively (NTP 2000b). Some of the information presented is based on old data and might not represent current exposures, especially since there have been recent changes in the use patterns for DEHP (see Section 6.6 for specific examples). However, there are current estimates, based on the measurement of the DEHP metabolite, MEHP, in human urine obtained by Blount et al. (2000a), which suggest that the average total daily ambient exposure of individuals in the United States to DEHP is likely to be <3.6 µg/kg body weight/day (David 2000; Kohn et al. 2000). Larger study populations will be required beyond the 289 individuals examined in the Blount et al. (2000a) work to

Table 6-2. Concentration of DEHP in Categories of Household Waste

	Concentration of DEHP (mg/kg)[a,b]		
Waste fraction	Minimum	Maximum	Mean
Food waste	64.3	4.8	334.7
8–40 mm Fraction	1,259.1	584.9	2,253.5
<8 mm Fraction	95.5	76.1	132.5
Paper for recycling	29.7	10.0	60.3
Unusable paper	71.1	41.4	106.4
Cardboard	47.4	10.1	70.5
Plastic films	444.9	169.0	907.9
Other plastics	1,027.6	373.8	2,035.3
Textiles	205.7	14.9	686.1
Compound packing waste	151.9	57.7	393.7
Compound materials	16,820.6	7,862.4	26,352.0
Disposable diapers[c]	74.1	14.2	322.2

Source: Bauer and Herrmann (1997)

[a]Results are from six extractions except “compound material” for which the results are for nine extractions.
[b]The precision of the values presented in this table is the same as the original paper.
[c]Described as “nappies” in the original paper

gain a representative sampling of the exposure of the U.S. population to DEHP, including possible demographic variations in exposure and/or metabolism that was noted by Blount and coworkers.

It is difficult to determine the dominant source of DEHP exposure by the oral route. While in the past, it is likely that food represented the major source of DEHP for at least the general population and possibly many children (Doull et al. 1999; Huber et al. 1996; NTP 2000b), much of the literature supporting this is 10–25 years old (see Huber et al. 1996). Some attempts have been made to estimate exposures of DEHP to the general population (3–30 µg/kg body weight/day) through ingestion that is based on current use patterns for DEHP (NTP 2000b), but more information is still needed. The National Health and Nutrition Examination Survey (NHANES) has obtained measurements of DEHP metabolites (mono-2-ethylhexyl phthalate) in urine as a method to assess exposures of the general population (6 years of age and older) to DEHP (Blount et al. 2000b; CDC 2001); however, no correlation of these measurements with actual DEHP intake has yet been determined. In addition, much of the current literature on DEHP contamination of foodstuffs is foreign or not typically associated with consumer exposures, and it is uncertain how applicable this information is to U.S. exposures (e.g., Fayad et al. 1997, migration of DEHP into bottled water, Saudi Arabia; Gramiccioni et al. 1990, migration of DEHP from caps into foods, Italy; Cohen et al. 1991, migration of DEHP from a plastic bag containing contaminated corn in a laboratory [the corn was not intended for consumer use], Canada/France; Tsumura et al. 2001, migration of DEHP from PVC gloves to prepared food, Japan; Bluthgen 2000, post-secretory migration of DEHP during milk processing and storage, Germany; and Morelli-Cardoso et al. 1999, migration of DEHP into food simulants, Brazil). Finally, while the Food and Drug Administration (FDA) allows the use of DEHP in food contact applications (e.g., can coatings FDA 1999g; adhesives FDA 1999a; defoaming agent in paper manufacture FDA 1999e; as a flow promoter at no more than 3% in acrylic and modified acrylic single and repeated use containers FDA 1999c; in cellophane used for food packaging at a concentration not to exceed 5% FDA 1999b; and as a surface lubricant in the processing of metal foil at a concentration not to exceed 0.015 mg/in^2 of metal surface FDA 1999d), it is not clear if industry currently uses DEHP in these applications (e.g., Mannsville Chemical Products Corporation [1999] reported that DEHP has not been used in food film wrap for over 20 years; they also included no food applications in their list of uses, see Chapter 5). Thus, the uncertainty associated with current concentrations in food as outlined above, makes quantifying intakes speculative. This might be especially true given the recent activity (as noted in Section 6.6) in eliminating phthalates from some consumer products.

Oral exposure from drinking water is not expected to be a significant route of exposure (Doull et al. 1999; Huber et al. 1996; NTP 2000b) based on estimates of <30 ppb for DEHP in water (Huber et al. 1996).

Stubin et al. (1996) noted that DEHP was commonly present (48% of the samples) in municipal sewage treatment plant influent, suggesting that DEHP is present in domestic waste water. DEHP in domestic waste water can come from either the source tap water or from activities within the household such as washing floors that contain DEHP, showering using a shower curtain containing DEHP, or washing other DEHP-containing materials.

Dermal exposure to DEHP can occur when items containing DEHP as a plasticizer are handled. Schwope and Reid (1988) noted that DEHP migrated into dry materials in contact with PVC containing DEHP. From the data available in this study, however, it is not clear how much DEHP will be transferred. A study of the migration of DEHP from PVC film to rat skin found that the mean dermal uptake of DEHP was small, only 0.24 µg/cm^2-hour (Deisinger et al. 1998), a rate that is likely to be 2–4 times faster than is expected for human skin (Barber et al. 1992; Scott et al. 1987). A report to the Consumer Product Safety Commission (2001) provided a range of time-weighted average daily dermal exposure estimates for another phthalate diester, diisononyl phthalate (DINP), of between 0.45 and 11 µg/kg/day in adults (Consumer Product Safety Commission 2001). However, it is noted in the same report that these current estimates of potential dermal exposure remain speculative. Because many common household products contain DEHP, including furniture upholstery, shower curtains, tablecloths, wall coverings, floor tiles, garden hoses, swimming pool liners, rainwear, baby pants, toys, shoes, and automobile upholstery (see Chapter 5), there are opportunities for exposure, but how much DEHP is transferred to, and absorbed by, an individual is still indeterminate.

Inhalation exposure can occur from breathing ambient air and indoor air, and is not considered to be a primary or significant route of exposure to DEHP. Huber et al. (1996) and Doull et al. (1999) have suggested, based on monitoring studies from the 1970s and 1980s, that inhalation exposures from breathing ambient air are low. No recent ambient air studies were found in the available literature; however, available studies report concentrations that span a relatively narrow range, even in industrialized areas (see Section 6.4.1); although industrial areas appear to have higher concentrations in some cases. Thurén and Larsson (1990) reported that higher concentrations of DEHP were seen adjacent to a facility using DEHP, but these concentrations fell off rapidly. Thus, it is anticipated that people living near DEHP use and disposal areas might be exposed to elevated levels, but it is unclear how much higher these concentrations might be. It is further anticipated that use facilities where DEHP is actively used, such as DEHP production or PVC manufacturing facilities, will emit more DEHP to the offsite environment (e.g., through air-borne particulates or water) than storage or disposal facilities because of the tendency of DEHP to sorb to organic matter in the soil or sediment (see Section 6.3.1).

Occupational exposure to DEHP might be important during the manufacture and processing of this compound. Workers might be exposed to relatively high concentrations of DEHP during the compounding of this plasticizer with resins and the manufacture of PVC plastic products. The National Institute for Occupational Safety and Health (NIOSH) estimated that about 340,000 workers (of which approximately 106,900 were female) were potentially exposed to DEHP in the early 1980s (NOES 1990). Workplace air levels of DEHP ranging from 0.02 to 4.1 mg/m^3 were reported at facilities using or manufacturing the compound (Hill et al. 2001; IARC 1982; Liss et al. 1985). These levels are below the current OSHA Permissible Exposure Limit (PEL) for DEHP for an 8-hour work-day of 5 mg/m^3 (OSHA 1989). Exposures of phthalate and PVC production workers to DEHP are estimated to be typically less than 143 and 286 µg/kg body weight/workday, respectively (NTP 2000b). The types of industries using DEHP-containing products (see Table 6-3) can be inferred from the types of industries that reported DEHP emissions to the TRI (TRI99 2001).

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. 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 spend more time outdoors. Children also are closer to the ground, and they do not use the judgment of adults to avoid hazards (NRC 1993).

Table 6-3. Types of Industries Using DEHP-containing Products

abrasive products	mechanical rubber goods
adhesives and sealants	medicinals and botanicals
agricultural chemicals	minerals (ground or treated)
asbestos products	motor vehicle parts and accessories
boat building and repairing	motor vehicles and car bodies
cement	noncurrent-carrying wiring devices
chemical preparations	nonferrous wire drawing and insulating
chemicals and allied products	nonmetallic mineral products
coated fabrics (not rubberized)	packing and sealing devices
crowns and closures	paints and allied products
current-carrying wiring devices	paper (coated and laminated)
custom compound purchased resins	pharmaceutical preparations
electrical industrial apparatus	photographic equipment and supplies
electromedical equipment	plastics foam products
electronic capacitors	plastics materials and resins
electronic components	plastics pipe
fabric dress and work gloves	plastics products
fabricated metal products	plating and polishing
fabricated rubber products	refuse systems
gaskets	rubber and plastic footwear
gray and ductile iron foundries	rubber and plastic hose and belting
hand and edge tools	sporting and athletic goods
hard surface floor coverings	surface active agents
household laundry equipment	surgical and medical instruments
hydraulic	tires and inner tubes
industrial inorganic chemicals	unsupported plastics film and sheet
industrial organic chemicals	unsupported plastics profile shapes
manufacturing industries	wood household furniture
mattresses and bedsprings	wood products
meat packing plants	

Source: TRI (TRI99 2001)

Children are exposed to DEHP orally from mouthing toys and other soft PVC products and possibly food, by inhalation from ambient and indoor air, and dermally from handling materials containing DEHP. In addition, children are potentially exposed from medical devices via the inhalation, dermal, oral, and intravenous routes. Exposures from medical devices will be treated separately in this section. It should be noted that assessing exposures to DEHP, and especially children's exposures, is difficult because the uses of DEHP, while constant for many years, have changed over the last 20 years (Consumer Product Safety Commission 1999d; Wilkinson and Lamb 1999). For example, manufacturers including Chicco, Little Tikes, Disney, Mattel (Fisher-Price ARCOTOYS, Tyco Preschool), Evenflo, Safety 1st, The First Years, Sassy, Gerber, Shelcore Toys, and Hasbro (including Playskool) stopped using phthalates in teethers and rattles in early 1999 (Consumer Product Safety Commission 1999d). In addition, a number of retail stores, including Toys-R-Us, Walmart, Sears, Target, K-Mart, ShopKo Stores, Inc., and Warner Brothers Studio Stores, have removed phthalate-containing teethers, rattles, pacifiers, and bottle nipples from their product lines. This change, and others that might be made in the near future, makes an assessment of a child's exposure to DEHP more difficult than would otherwise be the case.

It is difficult to determine the dominant source of DEHP exposure by the oral route for children, just as is the case with the general population. While in the past, food represented the dominant source of oral exposure to DEHP, as noted above in Section 6.5, the literature base supporting this is old and might not represent current exposures. A recent Danish study published by Petersen and Breindahl (2000) estimated the dietary intake of DEHP in infants (based on measurements of DEHP in baby food and formula) to be between 0.005 and 0.010 mg/kg body weight. However, without more recent information, quantifying either the amount or the extent of food exposures would be speculative. Drinking water is not anticipated to be a significant source of DEHP exposure; however, the database supporting this is old (see Section 6.5). DEHP concentrations in human breast milk of 70–160 µg/kg milk (mean concentration of 93±37.5 µg/kg milk) and 0–110 µg/kg milk (mean concentration of 0.034±0.043 µg/kg milk) have been reported (FDA 2001h). However, no information is available relating the concentration of DEHP in human breast milk obtained from women with high occupational exposures to DEHP or exposures that result from medical treatments (e.g., hemodialysis).

A source of DEHP exposure for young children by the oral route might be plastic toys. The exposure will be dependent on the time that a child spends mouthing a toy and the DEHP content of the toy. Information on children's mouthing behavior is available and indicates that the behavior is dependent on the age of the child and the items mouthed (Consumer Product Safety Commission 2001; Juberg et al. 2001). Juberg et al. (2001) found that children spend an average of 23 minutes/day (children between the

ages of 0 and 18 months), and 5 minutes/day (children between the ages of 19 and 36 months) mouthing toys and teethers; these times are shorter than the estimated mouthing times (e.g., 1–3 hours) found elsewhere (Health Canada 1998). These average mouthing times provided by Juberg et al. included children who did not exhibit mouthing behavior. If the averages included only children exhibiting mouthing behavior, then the time spent by these children mouthing teethers and toys increases to 48 minutes/day (children between the ages of 0 and 18 months), and 41 minutes/day (children between the ages of 19 and 36 months). Juberg et al. (2001) also reported pacifier use to average 108±187 (mean±1 standard deviation) minutes/day for children ages 0–18 months, and 126±246 minutes/day for children ages 19–36 months. However, manufacturers for some children's toys have discontinued the use of DEHP and other phthalates in pacifiers, teethers, rattles, and toys designed for very young children (see above). Therefore, the mouthing of pacifiers, teethers, and toys is not expected to be a significant route of exposure of young children to DEHP. Yet, there is no information about a similar action with other toys or toys for older children (toys, and specifically dolls, were identified by Mannsville Chemical Products Corporation (1999) as products that used DEHP as a plasticizer). In addition, families might hand down toys containing phthalates from older children rather than buy new toys that contain no phthalates. While some of these toys might have less than 3% DEHP (Wilkinson and Lamb 1999), this percentage likely cannot be applied to all toys that young children might play with. At the present time, however, sufficient information is not available to quantify these exposures.

Some research has been conducted to examine the migration of DEHP and other plasticizers from PVC into saliva. Steiner et al. (1998) reported that migration of DEHP from PVC into a saliva simulant was dependent on the contact time and agitation of the test matrix. *In vivo* studies of the migration of DEHP into human saliva from four adult volunteers chewing PVC balls (185 mg DEHP/g) showed a migration rate of 44.4 µg/10 cm^2/hour (Niino et al. 2001). However, no other studies, especially in children, are available evaluating DEHP migration rates in toys.

Other potential sources of oral exposure for young children as well as dermal exposure to all children include general household items made from PVC including dolls, plastic baby pants, furniture upholstery, floor tiles, shower curtains, and tablecloths (all of which are available for mouthing by children in addition to touching) (see also Chapter 5). In addition, young children might be exposed to DEHP when wearing such items as rainwear and shoes made from PVC. Dermal uptake of DEHP from PVC film to rat skin was found to be low, only 0.24 µg/cm^2-hour (Deisinger et al. 1998), but is expected to be 2–4 times lower for human skin (Barber et al. 1992; Scott et al.1987). A report to the Comsumer Product

Safety Commission (2001) provided a range of time-weighted average daily dermal exposure estimates for another phthalate diester, diisononyl phthalate (DINP), of between 3.2 and 79 µg/kg/day in children ages 19–36 months (Consumer Product Safety Commission 2001). However, it is noted in the same report that these current estimates of potential dermal exposure remain speculative. Oral exposure also might occur when children handle PVC items containing DEHP, and then the children's hands are mouthed. However, no specific reference to DEHP transfer from items to skin was found in the available literature. Therefore, sufficient information is not available to assess this route of exposure to DEHP.

All children might have inhalation exposures from both vapor and particle bound DEHP as well as oral exposure to DEHP from inhalation of large particles containing DEHP followed by deposition in the upper airways and swallowing (Hill et al. 2001). Øie et al. (1997) reported that sedimented dust samples from 38 dwellings in Oslo, Norway (including samples taken from sheets in a child's bed and floor in a child's bedroom) contained an average of 640 µg/g sedimented dust (100–1,610 µg/g), while suspended particulate matter from six dwellings contained an average of 600 µg/g (24–94 µg/g). The authors noted that exposure to particle bound DEHP is 0.4–1.2 µg/day for adults, but suggested that children, and especially small children, are "subject to the highest exposure risk" because they usually have small rooms that have higher surface to volume ratios and few doors or windows. Children's exposures to DEHP from inhalation of outdoor air is likely small because of the relatively low ambient concentrations (Doull et al. 1999; Huber et al. 1996). While the database of outdoor concentrations is dated (1970s through the 1980s), the concentrations appear to be very consistent both spatially and temporally.

A possible exception to the anticipated low exposure from inhalation to outdoor air might be in the vicinity of hazardous waste sites containing large concentrations of DEHP or use facilities. DEHP has a low volatility and is not expected to enter the air extensively; nonetheless, Thurén and Larsson (1990) noted higher concentrations of DEHP near a facility that used it, indicating that somewhat higher concentrations might be anticipated near use or storage facilities. Those industries that emitted more than 10,000 pounds into the air in 1998 were crowns and closures, fabricated rubber products, motor vehicle parts and accessories, plastics foam products, unsupported plastics film and sheet (TRI99 2001). Children living near the vicinity of one of these facilities might be exposed to somewhat elevated concentrations of DEHP, although exact concentrations are not known.

Children's exposures to DEHP during medical procedures have been reported (Hill et al. 2001; Karle et al. 1997; Latini and Avery 1999; NTP 2000b; Plonait et al. 1993; Shneider et al. 1991). Shneider et al. (1991) reported that serum DEHP concentrations varied depending on the nature of the treatment. They

reported that for an infant cardiopulmonary bypass, pediatric hemodialysis, exchange transfusion, and extracorporeal membrane oxygenation (ECMO), serum DEHP concentrations ranges were 1.1–5.1, 0.4–4.2, 5.4–21.5, and 18–98 µg/mL, respectively. Karle et al. (1997) confirmed this study, but reported lower concentrations. The authors reported that blood DEHP concentrations at the end of the blood prime during *in vitro* studies in extracorporeal membrane oxygenation circuits had mean values of 18.3, 21.8, and 19.3 µg/mL for different circuits and was dependent on the surface area of each circuit. The authors noted that the extraction rates were 0.32±0.12 and 0.57±0.14 µg/mL-hour for circuits A and B, respectively, but the extraction rates were identical when corrected for surface area. In contrast to the other two, the concentration of DEHP in circuit C decreased at a rate of 0.2±0.04 µg/mL-hour. The authors suggested that this was due to the heparin coating on the surfaces and noted that heparin might offer protection from DEHP leaching. After 3 days, DEHP concentrations in 18 infants averaged 4.9 µg/mL; the highest level seen was 8.3±5.7 µg/mL. Karle et al. (1997) calculated DEHP exposures during ECMO therapy averaged 1.2 mg/kg (2.0 mg/kg maximum) for a 3-day exposure, based on an average patient weight of 3.3 kg and an average blood volume of 800 mL for the 18 infants studied. These authors also reported that DEHP concentrations were below the detection limit in all patients after decannulation. They also noted that patients treated for longer periods did not have higher DEHP concentrations.

Latini and Avery (1999) reported that 60–120 mg of DEHP/g of tube was removed from endotracheal tubes during use (range of 44 samples). The authors also noted that zinc was lost from the tubes as well. Plonait et al. (1993) studied 16 newborn infants receiving blood exchange transfusions. The authors calculated exposures of 1.2–22.6 mg/kg-body weight, based on the volume of blood transfused and the mean DEHP concentration in the plasma of the blood units. They also noted that for three infants, 12.5, 22.9, and 26.5% of the DEHP introduced into the infants was eliminated in the waste (exchanged) blood. The authors reported that no correlation was found between the volume of blood transfused and the serum DEHP concentration immediately after the transfusion. There was also no correlation between the concentration of DEHP in the plasma and the storage time of the red cell bag. The authors reported that serum DEHP concentrations decreased rapidly after the transfusion was complete. Plonait et al. (1993) also reported that ethylhexanoic acid concentrations in the urine of infants undergoing transfusion therapy was below the detection limit (45 ng/mL) before or during the transfusion, but ranged from 50 to 416 ng/mL (median 130 ng/mL) in six infants 6 hours after the transfusion. Peak levels occurred within the first 18 hours, and then declined to close to the detection limit where they remained for 96 hours. Finally, these authors noted that for two infants, DEHP concentrations appeared to accumulate resulting

in higher concentrations in the post exchange serum than the average DEHP concentration in the blood received by the patients.

6.7 POPULATIONS WITH POTENTIALLY HIGH EXPOSURES

Several population subgroups might have above average exposure to DEHP. These include hemophiliacs and others who require frequent blood transfusions, dialysis patients who might be exposed to DEHP leached from the dialysis tubing (see Section 6.4.4), and preterm infants (Doull et al. 1999; FDA 2001h; Huber et al. 1996; Latini 2000; NTP 2000b; Tickner et al. 2001). Estimates of exposure levels indicate that hemophiliacs might be exposed up to 1–2 mg/day and dialysis patients might receive average doses of 40 mg/day (Pollack et al. 1985a; Wams 1987). Faouzi et al. (1999) estimated that dialysis patients received an average of 75 mg of DEHP per treatment and an average of almost 12 g of DEHP over a 1-year period (assuming dialysis treatments 3 times a week). Adult exposures to DEHP from hemodialysis have been estimated at <5–155 mg/day or <0.1–3.1 mg/kg/day and can vary considerably between patients (Dine et al. 2000; FDA 2001h; Huber et al. 1996; NTP 2000b). Infants receiving exchange transfusions might be exposed to >4 mg/kg/day (FDA 2001h; Sjoberg et al. 1985c), based on a worst-case scenerio. Plonait et al. (1993) reported higher plasma concentrations than those in the Sjoberg et al. (1985c) study, but the blood units used had a lower initial DEHP concentration. Plonait et al. (1993) suggest that this can be explained by pauses during the exchange transfusion during the Sjoberg et al. (1985c) study, which resulted in a lowering of the DEHP concentration. Faouzi et al. (1994) reported that administration of teniposide is sometimes associated with a nonionic surfactant polyoxyethylated castor oil. The presence of this surfactant increases the concentration of DEHP that is leached from the PVC bags into the administered solution. The authors reported that 52 mg was extracted at 48-hour room temperature storage. Preterm infants can be exposed to DEHP at levels estimated to be as high as 10–20 mg/day during the course of their care (Loff et al. 2000). Measured concentrations of DEHP in total parental nutrition (TPN) solutions (423±47 µg/mL), blood products (platelet-rich plasma, 13.9±2.5 µg/mL; fresh frozen plasma, 24.9±17 µg/mL) and selected drugs (propofol, 655±96 µg/mL) have been obtained in these solutions/products as a consequence of contact with PVC bags and tubing. Exposures to DEHP can be especially high for infants receiving TPN solutions (contains approximately 20% lipid emulsions), where a 24-hour infusion can deliver up to an estimated 10 mg of DEHP (Loff et al. 2000).

Workers in industries manufacturing or using DEHP plasticizer might be frequently exposed to above average levels of this compound (see Section 6.5). Those living near industrial facilities or hazardous

waste sites with higher than average levels of DEHP in water might also have potential above average exposure (see Section 6.4).

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 DEHP is available. Where adequate information is not available, ATSDR, in conjunction with the National Toxicology Program (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 DEHP.

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.

6.8.1 Identification of Data Needs

Physical and Chemical Properties. The physical and chemical properties of DEHP are sufficiently well characterized to allow estimation of its environmental fate and transport profile. On this basis, it does not appear that further research in this area is required.

Production, Import/Export, Use, Release, and Disposal. Data on the production and uses of DEHP in the United States are available (ChemExpo 1999; HSDB 1990; Mannsville Chemical Products Corporation 1999; TRI99 2001). Production has been fairly constant over the past 5 years and is expected to remain so during the next few years due to limited growth in PVC markets. Disposal of DEHP is mainly to landfills and the recently promulgated land disposal restrictions should ensure reduction of the disposal of untreated DEHP wastes. Available information appears to be sufficient for assessing the potential for release of, and exposure to, DEHP.

According to the Emergency Planning and Community Right-to-Know Act of 1986, 42 U.S.C. Section 11023, industries are required to submit chemical release and off-site transfer information to the EPA. The Toxics Release Inventory (TRI) contains this information from 1988 to 1999.

While information on uses is available (Mannsville Chemical Products Corporation 1999), specific information on uses in certain potentially high exposure applications is either changing or lacking. Specifically, information on the use of DEHP in food contact applications such as coatings used in cans, bottle caps, and films would allow a better estimation of potential exposures from food. Currently, the only information available is that these applications are allowed by FDA rules, but it is unclear if DEHP is used.

Environmental Fate. The environmental fate of DEHP has been fairly well characterized. Its transport in the atmosphere, sorption to sediments, bioconcentration in aquatic organisms, and biodegradation by water and soil microorganisms seem to be well understood (Al-Omran and Preston 1987; Atlas and Giam 1981; Barrows et al. 1980; Eisenreich et al. 1981; EPA 1980a; Kenaga 1980; Ligocki et al. 1985a, 1985b; Suggatt et al. 1984; Sullivan et al. 1982; Thurén and Larsson 1990; Wams 1987; Wolfe et al. 1980a). Sorption and biodegradation are competing processes for DEHP removal from water (Ritsema et al. 1989; Wams 1987). Reaction of atmospheric DEHP is rapid (Meylan and Howard 1993), but sorption might play an important role in slowing the oxidation process. Additional data on the rates of these reactions under various environmental conditions would be useful for more accurately predicting the fate of DEHP in all environmental media. Of particular interest would be additional information on the fate of DEHP leached into groundwater in order to document further that it is of minor concern in subsurface environments. Additionally, information on the atmospheric oxidation rate of DEHP sorbed to particulates would be of great interest. In designing such studies, it is critical to address the issue of laboratory contamination by DEHP.

Bioavailability from Environmental Media. On the basis of data from available toxicokinetics studies, DEHP will be absorbed following ingestion of contaminated drinking water and foodstuffs and inhalation of contaminated ambient air. Absorption following dermal exposure to soils is expected to be limited because of the strong sorption of DEHP to soils and because, in the absence of solvents, DEHP does not penetrate skin well. However, additional information would be useful to determine whether DEHP would be absorbed following dermal exposure to contaminated water and soils and ingestion of contaminated soils. This information will be helpful in assessing the relative importance of these pathways for exposed humans.

Food Chain Bioaccumulation. Bioconcentration of DEHP in aquatic organisms has been documented for several aquatic species (Barrows et al. 1980; EPA 1980a; Kenaga 1980; Staples et al. 1997). Based on the relatively high K_{ow}, it appears that accumulation can occur. However, rapid metabolism of DEHP in higher organisms seems to prevent biomagnification in the food chain (EPA 1979; Johnson et al. 1977; Staples et al. 1997; Wofford et al. 1981).

Exposure Levels in Environmental Media. Several studies are available documenting levels of DEHP in air, water, sediments, and biota in rural and urban areas. DEHP has been detected in surface water, groundwater, and soil samples taken in the environs of hazardous waste sites during recent monitoring surveys (Canter and Sabatini 1994; Eckel et al. 1993; Hauser and Bromberg 1982; Plumb 1987). Concentrations in ambient air at hazardous waste sites are available at only four sites. Ambient levels of DEHP are generally low in all environmental media. Since DEHP is a ubiquitous laboratory contaminant (see Chapter 7), it is very difficult to accurately determine these low levels. Often, laboratory contamination has undermined the credibility of the data generated. Additional monitoring data for DEHP in all environmental media, using recently suggested techniques for reducing laboratory contamination (see Section 7.2), would be useful to better assess the potential for human exposure to this compound.

Exposure Levels in Humans. Detection of DEHP in blood, urine, and adipose tissue might be indicators of human exposure. However, since DEHP is rapidly metabolized *in vivo*, levels in these biological materials have not been directly associated with environmental levels. Additional data correlating levels in environmental media with human tissue levels of DEHP or its metabolites, particularly for populations living in the vicinity of hazardous waste sites containing DEHP, would be helpful in establishing levels of DEHP to which humans have been exposed.

Exposures of Children. Little is known about exposures of children for DEHP. DEHP is widely used in many applications that can result in exposures. Toys were once considered an important route of exposure for children, especially in children under 36 months of age. However, toy manufactures have voluntarily reduced DEHP content below 3% by weight since 1986 and, more recently, have been phasing out the use of DEHP in most toys or using non-PVC materials, especially in pacifiers and teethers. However, there is only limited information on exposure of children to DEHP in items commonly encountered within the household and elsewhere (e.g., automobile interiors, daycare centers, etc.). The need for this information is highlighted by the fact that children below the age of 36 months were found to mouth many household items, in addition to those items currently under investigation, such

as pacifiers, teethers, and toys that are manufactured for use by young children (Juberg et al. 2001). In addition, while information is available on dust exposure in Norway, no information is available for the United States. This type of information along with indoor vapor measurements would allow a more precise estimation of indoor exposures where children, and especially young children, spend significant amounts of time.

Child health data needs relating to susceptibility are discussed in Section 3.12.2 Identification of Data Needs: Children's Susceptibility.

Exposure Registries. No exposure registries for DEHP were located. This compound is not currently one of the compounds for which a subregistry has been established in the National Exposure Registry. The compound will be considered in the future when chemical selection is made for subregistries to be established. The information that is amassed in the National Exposure Registry facilitates the epidemiological research needed to assess adverse health outcomes that might be related to the exposure to this compound.

6.8.2 Ongoing Studies

The Federal Research in Progress (FEDRIP 2001) database provides additional information obtainable from ongoing studies that might fill in some of the data needs identified in Section 6.8.1. No ongoing projects were identified in FEDRIP (2001).

The Consumer Product Safety Commission (1999c) has proposed a study to examine the mouthing behavior of children between 36 and 72 months old. The results of this study will help to refine the understanding of children's exposures. However, the status of this research could not be determined. Recent work from Juberg et al. (2001) has provided data that will aide in understanding the mouthing behavior children between the ages of 0 and 36 months.

Remedial investigations and feasibility studies at NPL sites that contain DEHP will provide further information on environmental concentrations and human exposure levels near waste sites.