6. POTENTIAL FOR HUMAN EXPOSURE

While this document is specifically focused on the primary forms or isomers of DDT, DDE, and DDD (namely *p,p'*-DDT, *p,p'*-DDE, and *p,p'*-DDD), other isomers of these compounds will be discussed when appropriate. It should be noted that DDT, DDE, and DDD are also synonyms for *p,p'*-DDT, *p,p'*-DDE, and *p,p'*-DDD, respectively, and it is usually understood that when DDT, for example, is mentioned *p,p'*-DDT is being referred to and not both *o,p'*- and *p,p'*-DDT. Technical-grade DDT, the grade that was generally used as an insecticide was composed of up to 14 chemical compounds, of which only 65–80% was the active ingredient, *p,p'*-DDT. The other components included 15–21% of the nearly inactive *o,p'*-DDT, up to 4% of *p,p'*-DDD, and up to 1.5% of 1-(*p*-chlorophenyl)-2,2,2-trichloroethanol (Metcalf 1995). In some cases, the term DDT will be used to refer to the collection of all forms of DDT, DDE, and DDD. Should this not be clear from the context, the term ΣDDT (Σ is used to mean sum of) will be used.

6.1 OVERVIEW

DDT and its primary metabolites, DDE and DDD, are manufactured chemicals and are not known to occur naturally in the environment (WHO 1979). Historically, DDT was released to the environment during its production, formulation, and extensive use as a pesticide in agriculture and vector control applications. DDD was also used as a pesticide, but to a far lesser extent than was DDT. Although it was banned for use in the United States after 1972, DDT is still being used in some areas of the world. DDT and its metabolites are very persistent and bioaccumulate in the environment.

DDT gets into the atmosphere as a result of spraying operations in areas of the world where it is still used. DDT and its metabolites also enter the atmosphere through the volatilization of residues in soil and surface water, much of it a result of past use. These chemicals will be deposited on land and in surface water as a result of dry and wet deposition. The process of volatilization and deposition may be repeated many times, and results in what has been referred to as a 'global distillation' from warm source areas to cold polar regions. As a result, DDT and its metabolites are transported to the Arctic and Antarctic regions where they are found in the air, sediment, and snow and accumulate in biota.

When in the atmosphere, about 50% of DDT will be found adsorbed to particulate matter and 50% will exist in the vapor phase (Bidleman 1988). A smaller proportion of DDE and DDD are adsorbed to particulate matter than DDT. Vapor-phase DDT, DDE and DDD react with photochemically-produced hydroxyl radicals in the atmosphere; their estimated half-lives are 37, 17, and 30 hours, respectively.

However, based on the ability of DDT, DDE, and DDD to undergo long range global transport, these estimated half-lives do not adequately reflect the actual lifetimes of these chemicals in the atmosphere.

The dominant fate processes in the aquatic environment are volatilization and adsorption to biota, suspended particulate matter, and sediments. Transformation includes biotransformation and photolysis in surface waters. The fate of DDT in the aquatic environment is illustrated by a microcosm study in which DDT was applied to a pond, and a material balance was performed after 30–40 days. At this time, DDT concentrations in the water column had declined to below the detectable limit (EPA 1979c). It was found that 90% of the initial DDT was not present in the water, sediment, algae, invertebrates, or fish, and was presumed to have volatilized. ΣDDT was present in water mainly as DDT during the first 30 days, as DDT and DDD during the next 30 days, and as DDD in the last 30 days. ΣDDT levels rapidly rose in invertebrates, reaching equilibrium in 5 days and then declining as the ΣDDT content of the water declined. Degradation of DDT is altered by invertebrates, with the conversion of DDT to DDMU. ΣDDT levels in fish rose rapidly and reached a high equilibrium level. In a study of a fresh water lake, DDT was found to accumulate to higher concentrations in fattier fish occupying high trophic levels than in leaner species occupying lower trophic levels (Kidd et al. 2001). Also, accumulation of DDT was significantly higher in the pelagic food web than in the benthic food web.

When deposited on soil, DDT, DDE, and DDD are strongly adsorbed. However, they may also revolatilize into the air, which is more likely to occur from moist soils than dry soils. They may photodegrade on the soil surface and biodegrade. DDT biodegrades primarily to DDE under unflooded conditions (e.g., aerobic) and to DDD under flooded (e.g., anaerobic) conditions. As a result of their strong binding to soil, DDT, DDE, and DDT mostly remain on the surface layers of soil; there is little leaching into the lower soil layers and groundwater. DDT may be taken up by plants that are eaten by animals and accumulate to high levels, primarily in adipose tissue and milk of the animals.

In discussing DDT and other pesticides in soil, agricultural chemists generally speak of persistence and degradation, but it is not always clear what mechanisms are responsible for the loss or dissipation of the chemical. This issue is further complicated in the case of DDT because what is often reported is the disappearance of ΣDDT residues rather than just *p,p'*-DDT. Many studies use first-order kinetics to model the dissipation of DDT in soils because a half-life for the chemical can be defined. The half-life represents the calculated time for loss of the first 50% of the substance, but the time required for the loss of half of that which remains may be substantially longer, and the rate of disappearance may decline further as time progresses. The rate and extent of disappearance may result from transport processes as

well as degradation or transformation processes. Initially, much of the disappearance of DDT is a result of volatilization losses, after which biodegradation becomes more important. When more than one process is responsible for loss, the decrease in the amount of substance remaining will be nonlinear. Recent assessments of long-term monitoring studies have indicated that even DDT biodegradation does not follow first-order kinetics (Alexander 1995, 1997). The reason is that over long periods of time, DDT may become sequestered in soil particles and become less available to microorganisms. The term half-life in this document is used to indicate the estimated time for the initial disappearance of 50% of the compound, and does not necessarily imply that first-order kinetics were observed throughout the experiment unless otherwise noted. The persistence of DDT in soil is highly variable. Dissipation is much greater in tropical than in temperate regions. In tropical and subtropical regions, most of the DDT is lost within a year; the half-life of ΣDDT in 13 countries ranged from 22 to 327 days. The half-life of DDE, the primary degradation product of DDT, ranged from 151 to 271 days. In another country where the soil was extremely acidic, the half-life was >672 days. Comparable half-lives in temperate regions have been reported to range from 837 to 6,087 days. One investigator concluded that the mean lifetime of DDT in temperate U.S. soils was about 5.3 years. In a study of sprayed forest soils in Maine, the half-time for the disappearance of DDT residues was noted to be 20–30 years (Dimond and Owen 1996). Highest residues are found in muck soils and in deeply plowed, unflooded fields (Aigner et al. 1998; Spencer et al. 1996). Significant concentrations of DDT have been found in the atmosphere over agricultural plots. Irrigating the soil dramatically increased the volatilization flux of DDT, which is probably related to the amount of DDT in the soil solution. Volatilization, air transport, and redeposition were found to be the main avenues of contaminating forage eaten by cows.

When deposited in water, DDT will adsorb strongly to particulate matter in the water column and primarily partition into the sediment. Some of the DDT may revolatilize. DDT bioconcentrates in aquatic organisms and bioaccumulates in the food chain. Marine mammals in the Arctic often contain very high levels of DDT and DDE.

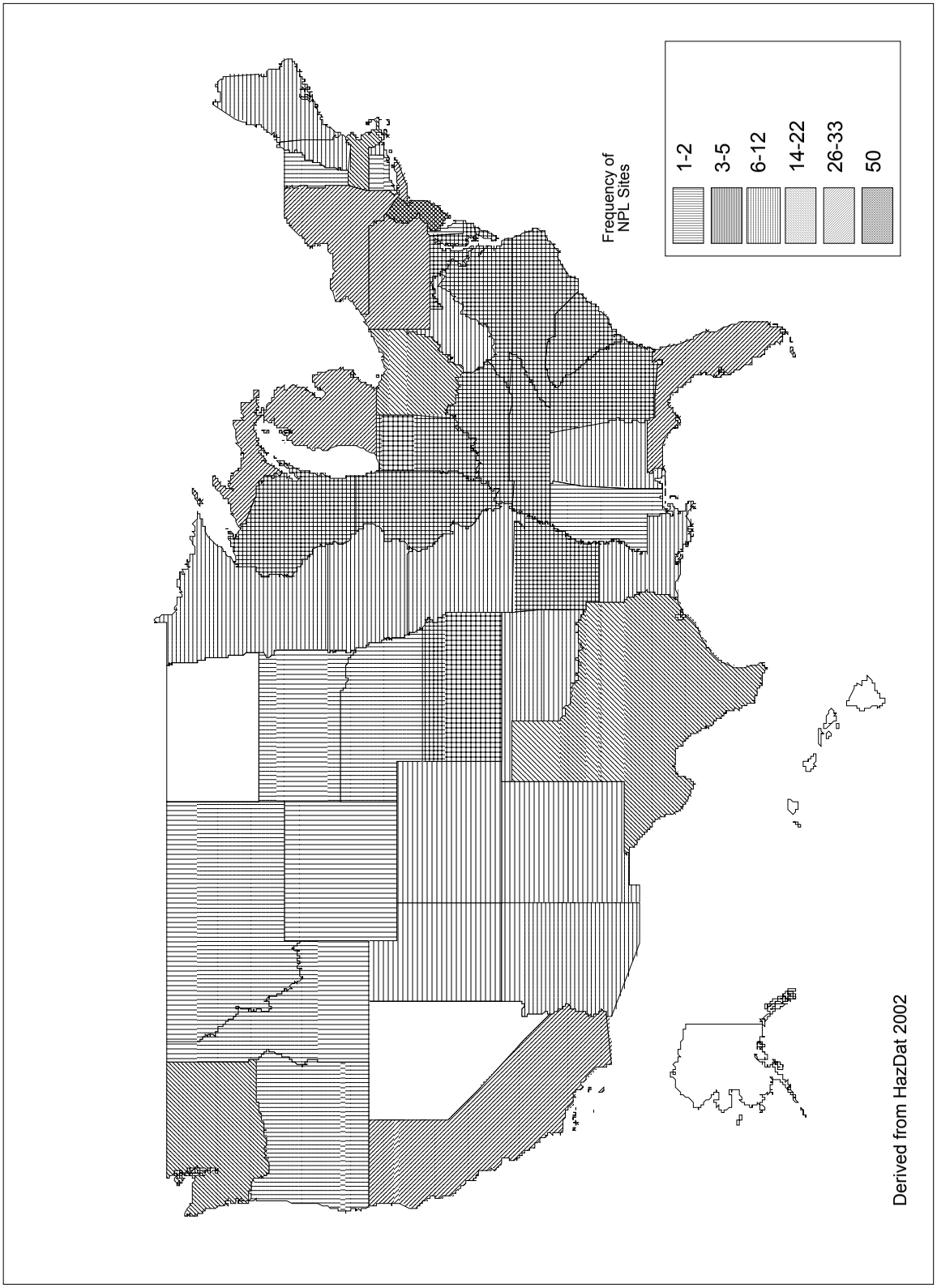
Concentrations of DDT in all media have been declining since DDT was banned in the United States and most of the world (Arthur et al. 1977; Boul et al. 1994; Van Metre and Callender 1997; Van Metre et al. 1997; Ware et al. 1978). For example, the concentration of DDT in lake sediments has decreased by 93% from 1965 to 1994 and declined by 70% in silt loam between 1960 and 1980 (Boul et al. 1994; Van Metre and Callender 1997; Van Metre et al. 1997). GDDT levels in sea lions have decreased by 2 orders of magnitude between 1970 and 1992 (Lieberg-Clark et al. 1995). The Market Basket Surveys have shown a 86% decline in DDT levels measured in all classes of food from 1965 to 1975 (EPA 1980a). However,

because of the extensive past use of DDT worldwide and the persistence of DDT and its metabolites, these chemicals are virtually ubiquitous and are continually being transformed and redistributed in the environment.

Human exposure to DDT is primarily through the diet. Exposure via inhalation at the ambient levels in air (Whitmore et al. 1994) is thought to be insignificant compared with dietary intake. The main source of DDT in food is meat, fish, poultry, and dairy products. DDT residues in food have declined since it was banned. Residues are more likely to occur in food imported from countries where DDT is still used. People eating fish from the Great Lakes were found to consume greater amounts of DDT in their diets (Hanrahan et al. 1999; Laden et al. 1999), but as DDT levels in Great Lakes fish continue to decline, exposure from consuming fish should also decline (Anderson et al. 1998; Hanrahan et al. 1999; Hovinga et al. 1993). The populations having the greatest exposure to DDT are indigenous people in the Arctic who eat traditional foods (e.g., seals, cariboo, narwhal whales, etc.) (Kuhnlein et al. 1995).

DDT, DDE, or DDD have been identified in at least 441 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 DDT, DDE, and DDD is not known. The frequency of these sites can be seen in Figure 6-1. Of these sites, 438 are located in the United States, 1 is located in Guam (not shown), 1 is located in Puerto Rico (not shown), and 1 is located in the U.S. Virgin Islands (not shown). *p,p'*-DDT, *p,p'*-DDD, and *p,p'*-DDE, the only DDT-related compounds targeted for analysis at NPL sites (EPA 1994), were respectively found at 326, 276, and 219 sites. While not targeted, *o,p'*-DDT, *o,p'*-DDD, and *o,p'*-DDE were nevertheless listed as detected at 4, 3, and 4 sites, respectively. Releases of DDT, DDE, or DDD are not required to be reported in the Toxics Release Inventory (TRI) database (EPA 1999).

Figure 6-1. Frequency of NPL Sites with DDT, DDE, and DDD Contamination



6.2 RELEASES TO THE ENVIRONMENT

6.2.1 Air

During the period when DDT was extensively used, a large source of DDT release to air occurred during agricultural or vector control applications. Emissions could also have resulted during production, transport, and disposal. Because use of DDT was banned in the United States after 1972, release of DDT in recent years should be negligible in this country.

Nevertheless, DDT residues in bogs or peat lands across the midlatitudes of North America indicate that DDT was still released, even after it was banned for use in the United States (Rapaport et al. 1985). These areas are unique in that they receive all of their pollutant input from the atmosphere, and therefore, peat cores are important indicators of the atmospheric deposition of a substance and also of its atmospheric levels in the present and the past. An analysis of peat cores, as well as rain and snow samples, indicated that DDT was still present in the atmosphere, although levels were lower compared to those in the 1960s. The implication is that DDT is still being released to the atmosphere either from its current production and use in other countries and transport to the United States or from the volatilization of residues resulting from previous use. The estimated release of DDT into the atmosphere from the Great Lakes in 1994, excluding Lake Huron, was 14.3 kg (Hoff et al. 1996). DDT, DDE, or DDD have been identified in air samples collected from 32 of the 441 NPL hazardous waste sites where it was detected in some in some environmental media (HazDat 2002).

6.2.2 Water

Historically, DDT was released to surface water when it was used for vector control in the vicinity of open waters. This source of release may still be occurring in countries that rely on DDT in insect pest control near open waters. DDT also enters surface water as a result of dry and wet deposition from the atmosphere and direct gas transfer. Atmospheric DDT deposited into tributaries will contribute to the loading in rivers, lakes, and oceans. In 1994, the estimated loading of ΣDDT into the Great Lakes as a result of dry and wet deposition was estimated as 148 kg, down from 278 kg in 1988 (Hoff et al. 1996). Fluvial sources and erosion also contribute to the DDT burden, and they were the predominant source of DDT in many areas in the past. This was clearly shown in a United States Geological Survey (USGS) study of sediment in reservoirs and lakes in Georgia and Texas compared with DDT levels in nearby peat bogs (Van Metre et al. 1997).

Contaminated sediment near an outfall can act as a source of contamination in distant parts of a body of water. This was clearly illustrated in a Norwegian lake that received insecticidal wastes. Nineteen years after closing the outfall, DDT concentrations in pike and perch were 5–10 times those in uncontaminated lakes (Brevik et al. 1996). DDT was disposed to the Joint Water Pollution Control Plant, Los Angeles County by Montrose Chemical Company from about 1950 to 1970, and eventually to the Palos Verdes Shelf via sewer pipes. The distribution of DDT with respect to the outfall diffusers and the fact that the DDT concentration in the overlying water column exponentially decreased with increasing distance from the sea floor indicated that the main source of DDT in the water column was contaminated sediments (Zeng et al. 1999). Studies have shown that a variety of organisms live in sediments at the Palos Verde site to depths of at least 35 cm and disturb the reservoir of contaminants there (Stull et al. 1996). Sediment-bound DDT is being biodiffused up from the subsurface to upper sediments, where they undergo resuspension and redistribution.

DDT, DDE or DDD have been identified in surface water collected at 101 sites and groundwater collected at 247 of the 441 NPL hazardous waste sites where it was detected in some environmental (HazDat 2002).

6.2.3 Soil

In the United States, large amounts of DDT were released to the soil during spraying operations or from direct or indirect releases during manufacturing, formulation, storage, or disposal. Since almost all of the DDT produced was used to control insects damaging crops and trees or responsible for insect-transmitted diseases, we can assume that a large fraction of the DDT produced was released to soil during spraying operations. The largest amounts of DDT released to soil were those used in agriculture which amounted to 27 million pounds in 1966 and 14 million pounds in 1971, shortly before it was banned (Gianessi 1992).

DDT, DDE, or DDD have been identified in soil samples collected from 776 sites and sediment samples at 305 of the 441 NPL hazardous waste sites where it was detected in some environmental media (HazDat 2002).

6.3 ENVIRONMENTAL FATE

A large proportion of the environmental fate studies on pesticides such as *p,p'*-DDT are performed in laboratory or field studies by agricultural chemists interested in the persistence of the active ingredient of the pesticide in the tilled layer of soil. Therefore, studies may not reveal whether the loss of active ingredient is a result of volatilization, leaching, or microbial degradation. Field studies may also report the occurrence of obvious metabolites remaining in surface soil months or years after a pesticide was applied. Clearly, it is not possible to separate these studies into 'Transport and Partitioning' (Section 6.3.1) and 'Transformation and Degradation' (Section 6.3.2). These studies are discussed in Section 6.3.2 with the understanding that 'degradation' may only account for part of the reported loss.

6.3.1 Transport and Partitioning

DDT and its metabolites may be transported from one medium to another by the processes of solubilization, adsorption, remobilization, bioaccumulation, and volatilization. In addition, DDT can be transported within a medium by currents, wind, and diffusion. These processes will be discussed in the following paragraphs.

Organic carbon partition coefficients (K_{oc}) of $1.5x10^5$ (Swann et al. 1981), $5.0x10^4$ (Sablejic 1984), and $1.5x10^5$ (Meylan et al. 1992) reported for *p,p'*-DDT, *p,p'*-DDE, and *p,p'*-DDD, respectively, suggest that these compounds adsorb strongly to soil. These chemicals are only slightly soluble in water, with solubilities of 0.025, 0.12, and 0.090 mg/L for *p,p'*-DDT, *p,p'*-DDE, and *p,p'*-DDD, respectively (Howard and Meylan 1997). Therefore, loss of these compounds in runoff is primarily due to transport of particulate matter to which these compounds are bound. For example, DDT and its metabolites have been found to fractionate and concentrate on the organic material that is transported with the clay fraction of the washload in runoff (Masters and Inman 2000). The amount of DDT transported into streams as runoff is dependent on the methods of irrigation used (USGS 1999). In the Western United States, DDT concentrations in streambed sediment increased as the percentage of furrow irrigation, as opposed to sprinkler or drip irrigation, increased. In the San Joaquin River Basin, more DDT was transported during winter runoff than during the irrigation season (Kratzer 1999). Since the compounds are bound strongly to soil, DDT would remain in the surface layers of soil and not leach into groundwater. However, DDT can adsorb to free-moving dissolved organic carbon, a soluble humic material that may occur in the soil solution. This material behaves as a carrier and facilitates transport of DDT into subsurface soil (Ding and Wu 1997). DDT released into water adsorbs to particulate matter in the water column and sediment.

Sediment is the sink for DDT released into water. There it is available for ingestion by organisms, such as bottom feeders. Reich et al. (1986) reported that DDT, DDE, and DDD were still bioavailable to aquatic biota in a northern Alabama river 14 years after 432,000–8,000,000 kg of DDT was discharged into the river. DDT in the water column above the outfall of Los Angeles County's Joint Water Pollution Control Plant's outfall was present both in the dissolved phase and the particulate phase (defined as particles size >0.7 μm) (Zeng et al. 1999). It is interesting to note that more of the DDT was present in the dissolved phase than in the particulate phase despite its high hydrophobicity.

There is evidence that DDT, as well as other molecules, undergoes an aging process in soil whereby the DDT is sequestered in the soil so as to decrease its bioavailability to microorganisms, extractability with solvents, and toxicity to some organisms (Alexander 1995, 1997; Peterson et al. 1971; Robertson and Alexander 1998). At the same time, analytical methods using vigorous extractions do not show significant decreases in the DDT concentration in soil. In one such study, DDT was added to sterile soil at various concentrations and allowed to age (Robertson and Alexander 1998). At intervals, the toxicity of the soil was tested using the house fly, fruit fly, and German cockroach. After 180 days, 84.7% of the insecticide remained in the soil, although more than half of the toxicity had disappeared when the fruit fly was the test species, and 90% had disappeared when the house fly was the test species. The effect with the German cockroach was not as marked. Recently, a study was conducted to determine the bioavailability of DDT, DDE, and DDD to earthworms (Morrison et al. 1999). It was shown that the concentrations of DDT, DDE, DDD, and ΣDDT were consistently lower in earthworms exposed to these compounds that had persisted in soil for 49 years than in earthworms exposed to soil containing freshly added insecticides at the same concentration. The uptake percentages of DDT and its metabolites by earthworms were in the range of 1.30–1.75% for the 49-year-aged soil, but were 4.00–15.2% for the fresh soil (Morrison et al. 1999). Long term monitoring data have also indicated that aged and sequestered DDT are not subject to significant volatilization, leaching, or degradation (Boul et al. 1994). The concentrations of DDT, DDE, and DDD monitored at two sites in a silt loam in New Zealand declined from 1960 to 1980, but very little loss was evident from 1980 to 1989 (Boul et al. 1994). The lack of appreciable biodegradation as DDT ages in soil suggests that the compound is not bioavailable to microorganisms. Aging is thought to be associated with the continuous diffusion of a chemical into micropores within soil particles where it is sequestered or trapped, and is therefore unavailable to microorganisms, plants, and animals (Alexander 1995). In the case of biodegradation, the aging process results in the gradual unavailability of substrate that makes the reaction kinetics appear to be nonlinear.

There is abundant evidence that DDT gets into the atmosphere as a result of emissions or volatilization. The process of volatilization from soil and water may be repeated many times and, consequently, DDT may be transported long distances in the atmosphere by what has been referred to as a 'global distillation' from warm source areas to cold polar regions (Bard 1999; Bidleman et al. 1992; Goldberg 1975; Ottar 1981; Wania and MacKay 1993). As a result, DDT and its metabolites are found in arctic air, sediment, and snow with substantial accumulations in animals, marine mammals, and humans residing in these regions (Anthony et al. 1999; Harner 1997). An analysis of sediment cores from eight remote lakes in Canada indicated that ΣDDT concentrations in surface sediments (0–1.3 cm depth) declined significantly with latitude (Muir et al. 1995). The maximum ΣDDT concentrations in core slices in midcontinent lakes date from the late 1970s to 1980s, which is about 5–10 years later than the maximum for Lake Ontario.

Volatilization of DDT, DDE, and DDD is known to account for considerable losses of these compounds from soil surfaces and water. Their tendency to volatilize from water can be predicted by their respective Henry's law constants, which for the respective *p,p'*- and *o,p'*- isomers are $8.3x10^{-6}$, $2.1x10^{-5}$, $4.0x10^{-6}$, $5.9x10^{-7}$, $1.8x10^{-5}$, and $8.2x10^{-6}$ atm-m^3/mol (Howard and Meylan 1997). The predicted volatilization half-lives from a model river 1 m deep, flowing at 1 m/sec, with a wind of 3 m/sec are 8.2, 3.3, 10.5, 6.3, 3.7, and 8.2 days, respectively. Laboratory studies of the air/water partition coefficient of DDE indicate that it will volatilize from seawater 10–20 times faster than from freshwater (Atlas et al. 1982). The authors suggest that this process may be related to interaction at the bubble-water surface.

Volatilization from moist soil surfaces can be estimated from the Henry's law constant divided by the adsorptivity to soil (Dow Method) (Thomas 1990). The predicted half-life for DDT volatilizing from soil with a K_{oc} of 240,000 is 23 days, compared to an experimental half-life of 42 days. Sleicher and Hopcraft (1984) estimated a volatilization half-life of 110 days for DDT from soil in Kenya based on mass transfer through the boundary layers, and claimed that volatilization of DDT was sufficient to account for its rapid disappearance from soil. However, laboratory experiments in which ^{14}C-*p,p'*-DDT was incubated in an acidic (pH 4.5–4.8), sandy loam soil maintained at 45 EC for 6 hours/day for 6 weeks resulted in neither volatilization of DDT or its metabolites nor mineralization (Andrea et al. 1994). Other studies using a latosol soil (pH 5.7) found that 5.9% of the radioactivity was lost through volatilization during a 6 week incubation at 45 EC (Sjoeib et al. 1994). The volatilization rate of DDT from soil is significantly enhanced by temperature, sunlight, and flooding of the soil (Samuel and Pillai 1990).

Transport of DDT in the atmosphere of central and eastern North America is facilitated by a circulation pattern that brings moisture from the Gulf of Mexico into the Midwest and the airflow patterns across the

eastern seaboard (Rapaport et al. 1985). DDT is removed from the atmosphere by wet and dry deposition and diffusion into bodies of water. The largest amount of DDT is believed to be removed from the atmosphere in precipitation (Woodwell et al. 1971).

DDT, DDE, and DDD are highly lipid soluble, as reflected by their log octanol-water partition coefficients (log K_{ow}) of 6.91, 6.51, and 6.02, respectively for the *p,p'*- isomers and 6.79, 6.00, and 5.87, respectively, for the *o,p'*- isomers (Howard and Meylan 1997). This lipophilic property, combined with an extremely long half-life is responsible for its high bioconcentration in aquatic organisms (i.e., levels in organisms exceed those levels occurring in the surrounding water). Organisms also feed on other animals at lower trophic levels. The result is a progressive biomagnification of DDT in organisms at the top of the food chain. Biomagnification is the cumulative increase in the concentration of a persistent contaminant in successively higher trophic levels of the food chain (i.e., from algae to zooplankton to fish to birds). Ford and Hill (1991) reported increased biomagnification of DDT, DDE, and DDD from soil sediment to mosquito fish, a secondary consumer. No distinct pattern of biomagnification was evident in other secondary consumers such as carp and small mouth buffalo fish. The biomagnification of DDT is exemplified by the increase in DDT concentration in organisms representing four trophic levels sampled from a Long Island estuary. The concentrations in plankton, invertebrates, fish, and fish-eating birds were 0.04, 0.3, 4.1, and 24 mg/kg, whole body basis (Leblanc 1995). Evans et al. (1991) reported that DDE biomagnified 28.7 times in average concentrations from plankton to fish and 21 times from sediment to amphipods in Lake Michigan. In some cases, humans may be the ultimate consumer of these contaminated organisms.

The bioconcentration factor (BCF) is defined as the ratio of the equilibrium concentration of contaminant in tissue compared to the concentration in ambient water, soil, or sediment to which the organism is exposed. There are numerous measurements and estimates of BCF values for DDT in fish. Oliver and Niimi (1985) estimated the steady-state BCF in rainbow trout as 12,000. Other BCF values that have been reported include 51,000–100,000 in fish, 4,550–690,000 in mussels, and 36,000 in snails (Davies and Dobbs 1984; Geyer et al. 1982; Metcalf 1973a; Reish et al. 1978; Veith et al. 1979). DDT bioconcentration studies in aquatic environments with representatives of various trophic levels demonstrate that bioconcentration increases with increasing trophic level (LeBlanc 1995). Trophic level differences in bioconcentration are largely due to increased lipid content and decreased elimination efficiency among higher level organisms. However, biomagnification also contributes to the increased concentration of DDT in higher trophic organisms (LeBlanc 1995).

The BCF values of *p,p'*-DDT in brine shrimp (*Artemia nauplii*) exposed to a mixture containing 0.5 or 1.0 ng/mL of four DDT analogs for 24 hours were significantly higher than for the three other chemicals. The BCF values were 41, 54, 128, and 248 for *p,p'*-DDE, *p,p'*-DDD, *o,p'*-DDT, and *p,p'*-DDT, respectively (Wang and Simpson 1996). The differences in BCF values are due to the different lipid solubility and selectivity of the compounds partitioned in the zooplanktonic organisms. *p,p'*-DDT, which has the greatest polarity of the 4 tested analogs, may have been adsorbed to a greater extent to the surface of the shrimp. In addition to absorbing DDT directly from the water, fish obtain DDT from their diet (Miller 1994; Wang and Simpson 1996). Wang and Simpson (1996) fed brook trout contaminated *A. nauplii* for 24 days followed by depuration for another 24 days during which the trout were fed uncontaminated *A. nauplii*. Although the concentration of *p,p'*-DDE was the lowest of the four analogs in the contaminated brine shrimp, the concentration of this compound in the trout at day 24 was 42.5 ng/g which was roughly 5 times more than the other analogs. The levels of the *p,p'*-isomers initially ranged from 1.0 to 2.7 ng/g, while *o,p'*-DDT was absent. The abnormal accumulation of *p,p'*-DDE in the fish suggests that mixed-function oxidases may have induced the dechlorination of *p,p'*-DDT to *p,p'*-DDE. This may account for the fact that about 70% of ΣDDT in fish is *p,p'*-DDE (Schmitt et al. 1990). After the fish were fed uncontaminated food, *p,p'*-DDE had the lowest percentage depuration. After feeding the trout for 24 days with the more highly contaminated brine shrimp, 14, 62, 17, and 32% depuration were observed for *p,p'*-DDE, *p,p'*-DDD, *o,p'*-DDT, and *p,p'*-DDT, respectively.

Fish move from the Great Lakes or other bodies of water with elevated DDT levels to rivers that feed into these lakes. In doing so, they transport DDT, which may represent a risk to wildlife along the tributaries (Giesy et al. 1994).

Despite being strongly bound to soil, at least a portion of DDT, DDE, and DDD is bioavailable to plants and soil invertebrates. Nash and Beall (1970) studied the DDT residues in soybean plants resulting from the application of [^{14}C]DDT to the surface or subsurface soil. They found that the major source of DDT contamination was due to sorption of volatilized residues from surface-treated soil. This was 6.8 times greater than that obtained through root uptake and translocation after subsurface treatment. In other experiments with oats and peas, root uptake of DDT was low and there was little or no evidence of translocation of the insecticide (Fuhremann and Lichtenstein 1980; Lichtenstein and Schultz 1980). Verma and Pillai (1991a) reported that grain, maize, and rice plants accumulate DDT adsorbed to soil. Most of the residues were found in the roots of the plant, and the lowest concentration of DDT residues was found in the shoots, indicating low translocation of DDT. Earthworms are capable of aiding the

mobilization of soil-bound DDT residues to readily bioavailable forms (Verma and Pillai 1991b). DDT may collect on the leafy part of plants from the deposition of DDT-containing dust.

6.3.2 Transformation and Degradation

6.3.2.1 Air

In the atmosphere, about 50% of DDT is adsorbed to particulate matter and 50% exists in the vapor phase (Bidleman 1988). In the vapor phase, DDT reacts with photochemically produced hydroxyl radicals with an estimated rate constant of 3.44×10^{-12} cm^3/molecule-sec determined from a fragment constant estimation method (Meylan and Howard 1993). Assuming an average hydroxyl radical concentration of 1.5×10^6 per cm^3, its half-life is estimated to be 37 hours. Both DDE and DDD have higher vapor pressures than DDT, and a smaller fraction of these compounds will be adsorbed to particulate matter. The estimated half-lives of vapor-phase DDE and DDD are 17 and 30 hours, respectively. Direct photolysis may also occur in the atmosphere.

It should be noted that the estimated half-lives for vapor-phase DDT, DDE, and DDD do not necessarily reflect the lifetimes of these compounds in air. DDT, DDE, and DDD can be adsorbed on particulate matter, where they are not expected to undergo rapid photooxidation, and therefore, may be subject to long-range transport. Indeed, long-range transport through the atmosphere has been demonstrated for DDT and several of its metabolites (Bard 1999; Bidleman et al. 1992; Goldberg 1975; Ottar 1981; Wania and MacKay 1993). The work of Bidleman (1988) suggests that 50% of DDT in the atmosphere is adsorbed to particulate matter. Further, when atmospheric sampling of pesticides was performed at nine localities in the United States during a time of high DDT usage, DDT was mostly present in the particulate phase (Stanley et al. 1971).

6.3.2.2 Water

DDT, DDE, and DDD present in water may be transformed by both photodegradation and biodegradation. Since the shorter wave radiation does not penetrate far into a body of water, photolysis primarily occurs in surface water and is dependent on the clarity of the water. Direct photolysis of DDT and DDD are very slow in aquatic systems, with estimated half-lives of >150 years (EPA 1979c). Direct photolysis of DDE will vary as a function of photoperiod and brightness, resulting in different half-lives depending on the season and latitude. Over the United States, the direct photolysis of DDE results in a

half-life of about 1 day in summer and 6 days in winter. DDE also undergoes photoisomerization when exposed to sunlight. Photolysis of DDE photoisomers is slower by at least one order of magnitude compared to DDE. Studies with DDT at shorter wavelengths suggest that the initial reaction results in the dissociation of the $Cl_2C–Cl$ bond. Some information exists on the indirect photolysis of DDT; no information on the indirect photolysis of DDE or DDD was located (Coulston 1985; EPA 1979c; Zepp et al. 1977).

Photoinduced 1,2 addition of DDT to a model lipid, methyl oleate, indicates that light-induced additions of DDT to unsaturated fatty acids of plant waxes and cutins may occur on a large scale (Schwack 1988).

DDT undergoes hydrolysis by a base-catalyzed reaction resulting in a half-life of 81 days at pH 9. The product formed in the hydrolysis is DDE. Hydrolysis of DDE and DDD is not a significant fate process (EPA 1979c).

Biodegradation of DDT in water is reported to be a minor mechanism of transformation (Johnsen 1976). Biodegradation of DDE and DDD in the aquatic environment is slower than that of DDT (EPA 1979c).

6.3.2.3 Sediment and Soil

Four mechanisms have been suggested to account for most losses of DDT residues from soils: volatilization, removal by harvest (e.g., plants that have absorbed the residue), water runoff, and chemical transformation (Fishbein 1973). Three of these are transport processes, and the fourth, chemical transformation, may occur by abiotic and biotic processes. Photooxidation of DDT and DDE is known to occur on soil surfaces or when adsorbed to sediment (Baker and Applegate 1970; Lichtenstein and Schultz 1959; Miller and Zepp 1979). The conversion of DDT to DDE in soil was enhanced by exposure to sunlight in a 90-day experiment with 91% of the initial concentration of DDT remaining in the soil for an unexposed dark control and 65% remaining for the sample exposed to light (Racke et al. 1997). However, UV-irradiation of ^{14}C-*p,p'*-DDT on soil for 10 hours mineralized less than 0.1% of the initial amount (Vollner and Klotz 1994). (Mineralization is the complete degradation of a chemical, generally to carbon dioxide and water for an organic chemical containing carbon, hydrogen, and oxygen.) The amount of DDT that may have been converted to DDE was not reported. Biodegradation may occur under both aerobic and anaerobic conditions due to soil microorganisms including bacteria, fungi, and algae (Arisoy 1998; EPA 1979c; Lichtenstein and Schulz 1959; Menzie 1980; Stewart and Chisholm 1971; Verma and Pillai 1991b). Since biodegradation studies generally focus on the loss of the parent

compound rather than complete degradation or mineralization, and since DDT initially biodegrades to DDD or DDE, there still may be dangerous compounds remaining after almost all of the DDT that was originally present has biodegraded.

During biodegradation of DDT, both DDE and DDD are formed in soils. Both metabolites may undergo further transformation but the extent and rate are dependent on soil conditions and, possibly, microbial populations present in soil. The degradation pathways of DDT under aerobic and anaerobic conditions have been reviewed by Zook and Feng (1999) and Aislabie et al. (1997). Ligninolytic or lignin-degrading fungi have been shown to possess the biodegradative capabilities for metabolizing a large variety of persistent compounds, including DDT. Mineralization of DDT and DDE was even observed in laboratory experiments using a member of this group of fungi, *Phanerochaete chrysosporium* (a white rot fungus) (Aislabie et al. 1997; Singh et al. 1999). Other soil microorganisms, such as *Aerobacter aerogenes*, *Pseudomonas fluorescens*, *E. coli*, and *Klebsiella pneumoniae*, have also been shown to have the capability to degrade DDT under both aerobic and anaerobic conditions, forming 4-chlorobenzoic acid and DDE, respectively (Singh et al. 1999). Biodegradation of DDT and its metabolites involves cometabolism, a process in which the microbes derive nutrients for growth and energy from sources other than the compound of concern. DDE, the dominant DDT metabolite found, is often resistant to biodegradation under aerobic and anaerobic conditions (Strompl and Thiele 1997). In laboratory experiments with marine sediments, DDT has been shown to degrade to DDE and DDD under anaerobic and anaerobic conditions, respectively (Kale et al. 1999). In these same experiments, it was shown that extensive degradation of DDT occurred in clams, converting DDT to DDMU. Recent laboratory experiments in marine sediment showed that DDE is dechlorinated to DDMU (1-chloro-2,2-bis[*p*-chloro-phenyl]ethylene) under methanogenic or sulfidogenic conditions (Quensen et al. 1998). The rate of DDE dechlorination to DDMU was found to be dependent on the presence of sulfate and temperature (Quensen et al. 2001). DDD is also converted to DDMU, but at a much slower rate. DDMU degrades further under anaerobic conditions to 2,2-bis(chlorophenyl)acetonitrile (DDNU) and other subsequent degradation species, such as 2,2-bis(chlorophenyl)ethanol (DDOH) and 2,2-bis(chlorophenyl)acetic acid (DDA), through chemical action (Heberer and Dünnbier 1999; Ware et al. 1980). No evidence was found that methylsulfonyl metabolites of DDT are formed as a result of microbial metabolism. The rate at which DDT is converted to DDD in flooded soils is dependent on the organic content of the soil (Racke et al. 1997). In a laboratory study, Hitch and Day (1992) found that soils with a low metal content (e.g., Al, Ba, Cd, Co, Cr, Fe, and K were the major metals examined) degrade DDT to DDE much more slowly than do soils with high metal content.

As mentioned earlier, the half-life represents the estimated time for the initial disappearance of 50% of the compound in question and does not necessarily imply that first-order kinetics were observed throughout the experiment unless otherwise noted. In the case of DDT, the disappearance rate slows considerably so that after the initial concentration is reduced by half, the time required for the loss of half of that which remains is substantially longer. This is largely because much of the initial loss of compound is due to volatilization, rather than biodegradation. However, the biodegradation rate also slows in time as discussed in Section 6.3.1. This is because DDT migrates into micropores in soil particles where it becomes sequestered and unavailable to soil microorganisms (Alexander 1995, 1997). In addition, the disappearance of DDT is often reported as the disappearance of ΣDDT residues, and therefore, the reported rate of loss is a summation of the component DDT-related chemicals. DDT breaks down into DDE and DDD in soil, and the parent-to-metabolite ratio (DDT to DDE or DDD) decreases with time. However, this ratio may vary considerably with soil type. In a 1995–1996 study of agricultural soils in the corn belt of the central United States, the ratio of *p,p'*-DDT/*p,p'*-DDE varied from 0.5 to 6.6 with three-quarters of the soils having ratios above 1 (Aigner et al. 1998). In a study of forest soils in Maine, the half-life for the disappearance of DDT residues was noted to be 20–30 years (Dimond and Owen 1996). DDT was much more persistent in muck soils than in dry forest soils. A study of DDT in agricultural soils in British Colombia, Canada reported that over a 19-year period, there was a 70% reduction of DDT in muck soils and a virtual disappearance of DDT from loamy sand soils (Aigner et al. 1998).

Land management practices also affect the persistence of DDT. In 1971, an experiment was conducted in a field containing high amounts of DDT to evaluate the effect of various management tools in the disappearance of the insecticide (Spencer et al. 1996). The site was revisited in 1994 to determine the residual concentrations of DDT and its metabolites and to measure volatilization fluxes. Concentrations of DDT were reduced in all plots and the major residue was *p,p'*-DDE. The highest concentrations of residues were found in deep plowed and unflooded plots. Deep plowing places the DDT deeper into the soil profile, possibly reducing volatilization. As was noted in Section 6.3.1, the volatilization rate of DDT is enhanced by flooding the soil (Samuel and Pillai 1990). Under flooded, reducing conditions, DDD was a more common degradation product of DDT than DDE. Significant concentrations of both *o,p'*- and *p,p'*-DDE and *p,p'*-DDT were detected in the atmosphere over the plots. Irrigating the soil dramatically increased the volatilization flux of all DDT analogs, especially *p,p'*-DDE. This is probably related to the amount of DDT in the soil solution. Volatilization, air transport, and redeposition were found to be the main avenues of contaminating forage eaten by cows. In microcosm experiments, Boul (1996) found that increasing soil water content enhanced DDT loss from generally aerobic soil. His

results suggested that increased biodegradation contributed to these effects. Boule et al. (1994) analyzed DDT residues in pasture soil as they were affected by long-term irrigation and superphosphate fertilizer application. They found that ΣDDT residues in irrigated soil were about 40% that of unirrigated soil. The predominant residue was *p,p'*-DDE, and these residues were much higher in unirrigated than in irrigated soil. *p,p'*-DDE is lost at a lower rate than *p,p'*-DDT. *p,p'*-DDD residues were very low in both irrigated and unirrigated soil indicating that loss of *p,p'*-DDD must occur at a rate at least as great as it is generated from *p,p'*-DDT. Superphosphate treatment, which is known to increase microbial biomass, also resulted in lower levels of *p,p'*-DDT and ΣDDT than in unfertilized controls. The distribution of ΣDDT with depth suggests that irrigation did not cause increased leaching of the insecticide.

A set of experiments was conducted during 1982–1987 and 1989–1993 in 14 countries under the auspices of the International Atomic Energy Agency (IAEA) on the dissipation of ^{14}C-DDT from soil under field conditions in tropical and subtropical areas (Racke et al. 1997). After 12 months, the quantity of DDT and metabolites remaining in soil at tropical sites ranged from 5% of applied in Tanzania to 15% in Indonesia. The half-life of ΣDDT ranged from 22 days in Sudan to 365 days in China. One exception was in an extremely acidic soil (pH 4.5) in Brazil in which the half-life was >672 days. The conclusion of the study was that DDT dissipated much more rapidly under tropical conditions than under temperate conditions. The major mechanisms of dissipation under tropical conditions were volatilization, biological and chemical degradation, and to a lesser extent, adsorption. Comparable half-lives in temperate regions that have been reported range from 837 to 6,087 days (Lichtenstein and Schulz 1959; Racke et al. 1997; Stewart and Chisholm 1971). One investigator concluded that the mean lifetime of DDT in temperate U.S. soils was about 5.3 years (Racke et al. 1997). The primary metabolite detected in tropical soil was DDE. With the exception of highly acidic soil from Brazil, the half-lives for DDE ranged from 151 to 271 days, much less than the >20 years reported for DDE in temperate areas. The increased dissipation of DDT in the tropics compared with that in temperate zones is believed to be largely due to increased volatility under tropical conditions (Racke et al. 1997).

6.4 LEVELS MONITORED OR ESTIMATED IN THE ENVIRONMENT

6.4.1 Air

DDT is transported long distances from source areas to the Arctic and Antarctic. Mean ΣDDT levels in air over a period of 17 weeks at Signy Island, Antarctica in 1992 and over the ocean separating New Zealand and Ross Island, Antarctica between January and March, 1990 were 0.07–0.40 and 0.81 pg/m^3,

respectively (Bidleman et al. 1993; Kallenborn et al. 1998). The concentration declined with increasing latitudes.

Ten samples taken over the Gulf of Mexico in 1977 contained an average of 34 pg/m^3 of DDT, with a range of 10–78 pg/m^3 (Bidleman et al. 1981). Iwata et al. (1993) collected and analyzed 71 samples of air over several oceans (18 sampling locations) from April 1989 to August 1990. The range of mean and maximum concentrations of DDTs were (substance, range of means, maximum concentration): *p,p'*-DDE, 0.3–180 pg/m^3, 180 pg/m^3; *o,p'*-DDT, 0.3–180 pg/m^3, 420 pg/m^3; *p,p'*-DDT, 1.2–220 pg/m^3, 590 pg/m^3; and ΣDDT, 2.4–580 pg/m^3, 1,000 pg/m^3. The highest concentrations of DDT were found at locations near areas where DDT is still used, such as the Arabian Sea off the west coast of India. Other locations with high air concentrations of DDT were the Strait of Malacca, South China Sea, and the Gulf of Mexico. *p,p'*-DDT concentrations obtained from monthly air samples collected from Saginaw Bay, Sault Ste. Marie, and Traverse City, Michigan between November 1990 and October 1991 were below the detection limit during most of the winter months at Saginaw and Traverse City, and were above the detection limit at Sault Ste. Marie only in March, May, July, and August (Monosmith and Hermanson 1996). The highest monthly *p,p'*-DDT concentrations were 35 pg/m^3 in Saginaw (August), 31 pg/m^3 in Sault Ste. Marie (May), and 21 pg/m^3 in Traverse City (July). The corresponding highs for *p,p'*-DDE were 63 pg/m^3 (August), 119 pg/m^3 (May), and 92 pg/m^3 (July). An analysis of the results suggests that higher DDT and DDE levels correlated with air mass movement from the south, perhaps from areas where DDT is still used (i.e., Central America or Mexico). The fact that the ratio of DDT to DDE was <1 in each instance suggests that there is no new DDT use in Michigan. DDT and DDE levels over Green Bay, Wisconsin in 1989 were 8.7 and 15 pg/m^3, and those over the four lower Great Lakes obtained during a cruise were 38 and 59 pg/m^3 (McConnell et al. 1998). An analysis of air masses indicated that the atmospheric sources were not long-range transport, but rather local or regional volatilization.

Stanley et al. (1971) measured atmospheric levels of pesticides in the United States during a time of high DDT usage. Nine localities were sampled representing both urban and agricultural areas. Of 12 pesticides evaluated, only DDT was detected at all localities. Maximum levels of *p,p'*-DDT ranged from 2.7 ng/m^3 in Iowa City, Iowa to 1,560 ng/m^3 in Orlando Florida. Maximum levels of *o,p'*-DDT and *p,p'*-DDE ranged from 2.4 to 131 ng/m^3. The highest levels were found in the agricultural areas of the South. The pesticides were predominantly detected in the particulate phase. Some agricultural areas in which DDT was extensively used have been monitored periodically since usage was halted. Atmospheric conditions in the Mississippi Delta were monitored intermittently from 1972 to 1975 (Arthur et al. 1977). Air samples taken in 1975 from an area with extensive cotton acreage had a mean ΣDDT concentration of

7.5 ng/m^3, compared to 11.9 ng/m^3 in 1974. This represents a 36% decline in ΣDDT levels in 1 year. Between 1972 and 1974, the first 2 years after the use of DDT was banned, the atmospheric ΣDDT levels had declined by 88%. In 3 years, the decrease in ΣDDT air levels was 92%, representing a much more rapid decline than had been expected. In a comparison of the results from a 1995 study of the occurrence and temporal distribution of pesticides in Mississippi with the results obtained in 1967, a decline in the concentration of *p,p'*-DDE in air over agricultural lands was also noted (Coupe et al. 2000). Concentrations of *p,p'*-DDE in air were lower in the 1995 measurements, ranging from 0.13 to 1.1 ng/m^3, as compared to a range of 2.6–7.1 ng/m^3 obtained in 1967. However, these results also attest to the persistence of *p,p'*-DDT degradation products after >2 decades since the ban on DDT use in the United States (Coupe et al. 2000).

p,p'-DDT, *p,p*'-DDE, and *p,p*'-DDD have all been detected in the dissolved and particulate phases of fogwater and air and in rainwater (Millet et al. 1997). Fogwater samples were 1.5–30 times higher in DDT, DDE, and DDD concentration than rainwater samples, and the distribution between dissolved and particulate phase appeared to be governed by the solubility of the chemical. The site of the measurements was a rural area in France between 1991 and 1993. DDT had not been used in the area since the 1970s. Ligocki et al. (1985) conducted concurrent rain and air sampling for rain events in Portland, Oregon, in 1984. In rain samples, no *p,p*'-DDT, *p,p*'-DDE, or *p,p*'-DDD were detected. However, in the gas phase associated with this rainfall, *p,p*'-DDE was detected in five of seven samples. Levels detected in the samples ranged from nondetected to 420 pg/m^3. In another study, Poissant et al. (1997) reported the mean concentration of *p,p'*-DDT in precipitation over a rural site near the St. Lawrence river was 500 pg/L with a 75% frequency of detection. Rapaport et al. (1985) measured DDT residues in rain and snow samples in Minnesota. Samples of snow taken in 1981–1982, 1982–1983, and 1983–1984 contained an average of 0.32, 0.60, and 0.18 ng/L of *p,p'*-DDT, respectively. Two rain samples taken in 1983 contained 0.2 and 0.3 ng/L of *p,p'*-DDT. In rainwater samples taken from a forested region in northeast Bavaria in 1999, *p,p'*-DDT and *p,p'*-DDE were detected in three of six and four of six rainwater samples, respectively, ranging in concentration from not detected to 12.9 ng/L and not detected to 13.3 ng/L, respectively. In the vapour phase, *p,p'*-DDT and *p,p'*-DDE were detected in two of five and three of five air samples, respectively, ranging in concentration from not detected to 0.03 ng/m^3 and from not detected to 0.055 ng/m^3, respectively (Streck and Herrmann 2000).

6.4.2 Water

Although there are numerous reports in the literature of DDT levels in specific bodies of water throughout the United States, there is little information providing evidence of trends in the DDT levels over time. EPA operates STORET (STOrage and RETrieval), a computerized water quality database. Staples et al. (1985) reported limited data on priority pollutants from STORET. Information from data collected from 1980 to 1983 indicated that 3,500–5,700 ambient water samples were analyzed for DDT, DDE, and DDD with approximately 45% of the samples containing one of these compounds. The median level reported for both DDT and DDE was 0.001 µg/L, while the median level reported for DDD was 0.000 µg/L. Approximately 50 samples of industrial effluents were sampled and showed median levels of 0.010 µg/L for all three compounds. DDT was monitored in surface water and sediment as part of the National Surface Water Monitoring Program in 1976–1980. The percent occurrence and maximum concentrations of the reported DDT-related compounds in surface water were: *p,p*'-DDT, 0.5%, 0.70 µg/L (ppb); *o,p*'-DDT, 0.1%, 0.42 µg/L; *p,p*'-DDE, 0.7%, 0.55 µg/L; and *o,p*'-DDE, 0.3%, 0.54 µg/L (Carey and Kutz 1985). The USGS and EPA cooperatively monitored levels of pesticides in water and sediment at Pesticide Monitoring Network stations between 1975 and 1980 (Gilliom 1984). Of the 177 stations (approximately 2,700 samples) monitored, 2.8, 0.6, and 4.0% contained detectible levels of DDT, DDE, and DDD in water, respectively. Fewer than 0.4% of the samples contained detectable DDT-related residues. The levels detected in water were not reported, but the limit of detection was 0.05 µg/L for DDT and DDD, and 0.3 µg/L for DDE. The percentage of sites having detectable levels of DDT-related residues in sediment was much higher (see Section 6.4.3).

Johnson et al. (1988b) reported DDT and metabolite levels in the Yakima River basin in Washington State. Use of DDT was halted in this area when the 1972 ban was initiated; however, considerable residues are present in the river and sediments. Whole unfiltered water samples, collected mainly from the tributaries between May and October 1985, were reported to contain between not detectable to 0.06 µg/L of DDT-related compounds. Concentrations of *p,p*'-DDT in water equaled or exceeded those of *p,p*'-DDE; an unexpected finding in light of what is believed concerning biological half-lives of DDT and its normal environmental degradation (Singh et al. 1999; Wolfe and Seiber 1993). The authors have suggested an unusually long half-life for DDT in Yakima basin soils, which would enter the river through runoff to explain the higher than expected *p,p*'-DDT/*p,p*'-DDE ratios.

In the Malheur watershed, DDT was found to be persistent in the watershed, with estimated concentrations for ΣDDT ranging from 0.13–0.34 ng/L in rural regions of the watershed to 3.0–4.7 ng/L

in urbanized areas of the Malheur River near Ontario, Oregon (Anderson and Johnson 2001). Unlike the relative concentrations of DDT and DDE in the Yakima River, the concentrations of DDE in water samples were higher than those measured for DDT (ranges from 0.03–0.25 and 0.07–0.14 ng/L for DDE and DDT, respectively, in rural areas and 1.9–4.3 and 0.25–0.61 ng/L near Ontario, respectively), indicating that although DDT was still persistent in this watershed, it was undergoing the expected environmental degradation.

A summary of pesticide levels in surface waters of the United States during 1967 and 1968 was reported by Lichtenberg et al. (1970). During these 2 years (which were prior to the ban of DDT use), a total of 224 samples (unfiltered) were analyzed from various sites in all regions of the country. DDT was found in 27 samples at levels ranging from 0.005 to 0.316 µg/L; DDE was found in 3 samples at levels of 0.02–0.05 µg/L; and DDD was found in 6 samples at levels of 0.015–0.840 µg/L.

According to the U.S. Geological Survey's National Water Quality Assessment Plan initiated in 1991, that focuses on the water quality in more than 50 major river basins and aquifer systems, the frequency of detection of DDT and its metabolites in streams and groundwater was very low (USGS 1999). The top 15 pesticides found in water were those with high current use.

Only a few studies report levels of DDT in drinking water. Drinking water in Oahu, Hawaii, was found to contain *p,p'*-DDT at an average level of 0.001 µg/L in 1971 (Bevenue et al. 1972). In a study of Maryland drinking water during the September 1995, *p,p'*-DDE was detected in 22 out of 394 (5.6%) water samples, ranging in concentration from 0.039 to 0.133 µg/L (MacIntosh et al. 1999). Concentrations of *p,p'*-DDT and *p,p'*-DDD could not be measured above their limits of detection of 0.021 and 0.028 ppb, respectively. Keith et al. (1979) reported that DDE was found in 2-month equivalent (the amount of water a person would theoretically consume over a 2 month period) samples collected over 2 days from two of three drinking water plants in New Orleans in 1974; the DDE concentration was 0.05 µg/L in both samples.

Iwata et al. (1993) collected and analyzed 68 samples of surface water from several oceans (18 sampling locations) mainly affected by atmospheric deposition from April 1989 to August 1990. The range of mean and maximum concentrations of DDTs were (substance, range of means, maximum concentration): *p,p'*-DDE, 0.2–3.0 pg/L, 7.9 pg/L; *o,p'*-DDT, <0.1–5.8 pg/L, 14 pg/L; *p,p'*-DDT, 0.1–7.5 pg/L, 19 pg/L; and ΣDDT, 0.3–16 pg/L, 41 pg/L. The highest concentrations of DDT-related compounds were in the

East China Sea. Other seas with high concentrations of DDT were the Bay of Bengal, Arabian Sea, and South China Sea.

Canter and Sabatini (1994) reviewed Records of Decision at 450 Superfund Sites and found 49 cases in which contaminated groundwater threatened local public water supply wells. However, chlorinated organic pesticides were not found to be a major class of contaminants in these cases. In only one of the six sites in which the findings were presented in any detail was a DDT analog found at detectable levels. *p,p'*-DDD was found in monitoring wells from the upper aquifer at Pristine, Inc., an industrial site in Reading, Ohio at 0–0.14 µg/L but not in the lower aquifer or in water supply samples that were taken from the lower aquifer. No *p,p'*-DDT, or *p,p'*-DDE was detected in groundwater samples. Surface water samples contained levels of *p,p'*-DDE, *p,p'*-DDD, and *p,p'*-DDE at ranges of 0–0.86, 0–0.78, and 0–1.82 µg/L, respectively. Even at sites where the surficial soil concentrations of *p,p*'-DDT are extremely high (29–959 mg/kg), the concentration of *p,p*'-DDT and its metabolites, *p,p*'-DDE and *p,p*'-DDD, in groundwater were close to their detection limits (#0.05 µg/L) (Vine et al. 2000). *p,p*'-DDE was detected in monitoring wells (depth range of 20–110 feet) set up around orchards and row crop fields in the Columbia Basin Irrigation Project, but was not detected in shallow domestic wells (depth range of 80–250 feet) that were within 100 feet of these agricultural sites (Jones and Roberts 1999).

6.4.3 Sediment and Soil

Gilliom (1984) presented results of pesticide monitoring in sediment at USGS/EPA Pesticide Monitoring Network stations between 1975 and 1980. Of the 171 stations (approximately 900 samples) monitored, 26, 42, and 31 contained detectible levels of DDT, DDE, and DDD, respectively. Fewer than 17% of the samples contained detectable DDT-related residues (limit of detection was 0.5 µg/kg for DDT and DDD, and 3 µg/kg for DDE). The percentage of sites with detectable levels of DDT-related residues in sediment was much higher than in water, reflecting the preferential partitioning of DDT to sediment (see Section 6.4.2). From 1980 to 1983, approximately 1,100 samples of sediments in EPA's STORET database were analyzed for DDT, DDE, and DDD (Staples et al. 1985). The median levels for DDT, DDE, and DDD were 0.1, 0.1, and 0.2 µg/kg dry weight, respectively. In order to investigate circumstances contributing to the high level of DDT in fish and wildlife, soil and sediment samples (n=28) were collected in 1987 from the Upper Steele Bayou Watershed in west-central Mississippi at two depths (2.54–7.62 cm and 25.40–30.48) (Ford and Hill 1991). The results are given below:

Compound	Depth (cm)	Occurrence	Concentration (µg/kg)	
			Mean	Range
p,p'-DDD	2.54–7.62	86%	40	ND–410
p,p'-DDD	25.40–30.48	64%	20	ND–390
p,p'-DDE	2.54–7.62	93%	100	ND–660
p,p'-DDE	25.40–30.48	79%	40	ND–560
p,p'-DDT	2.54–7.62	79%	30	ND–600
p,p'-DDT	25.40–30.48	64%	20	ND–860

ND = not detected

River bed sediment samples collected in 1985 from the Yakima River basin in Washington contained 0.1–234 ug/kg (dry weight) of ΣDDT and its metabolites (Johnson et al. 1988b). Use of DDT was halted in this area in 1972 when the ban was initiated.

The concentrations of DDE, DDD, DDT, and ΣDDT in bed sediment from the San Joaquin River and its tributaries in California (7 sites) in 1992 were 1.4–115, 0.7–14, 0.4–39, and 2.2–170 ng/L, respectively (Pereira et al. 1996). One of the seven sites, Orestimba Creek, had DDT levels far higher than the other sites. Land use along this creek was dominated by orchards and a variety of row crops. Runoff that occurs during winter storms and the irrigation season contributes significant amounts of DDT-ladened sediment into the San Joaquin River and its tributaries (Kratzer 1999). For example, during the 1994 irrigation season, it was estimated that approximately 136 g/day of ΣDDT entered the river and its tributaries from runoff, totaling around 5,190–8,920 g of ΣDDT for the season. Additionally, winter storm runoff can input large amounts of DDT within sediments into these surface waters in short periods of time. For example, a winter storm in January 1995 sent sediment-ladened runoff into the river and its tributaries, carrying upwards of 4,500 g/day of ΣDDT into these surface waters for a total contribution of 1,750–2,620 g of ΣDDT from this one storm alone.

Total DDT in surface sediment collected in eight remote lakes in Canada along a midcontinental transect from 49 EN to 82 EN declined significantly with latitude from 9.7 µg/kg (dry weight) to 0.10 µg/kg (Muir et al. 1995). The pattern of DDT deposition in lake sediment in the continental United States is exemplified by that in White Rock Lake in Dallas. Total DDT concentrations in the lake sediment increased from the mid-1940s to a maximum of 27 µg/kg in about 1965 when DDT usage peaked in the United States and have decreased by 93% to 2 µg/kg in the most recent samples collected in 1994 (Van

Metre and Callender 1997; Van Metre et al. 1997). On the average, DDE accounted for 58% of the total DDT in the lake. DDD levels were about half those of DDE. The mean concentration of ΣDDT in sediment in the Newark Bay Estuary, New Jersey collected between February 1990 and March 1993 ranged from about 100 to 300 µg/kg except for the Arthur Kill, where the mean concentrations exceeded 700 µg/kg (Gillis et al. 1995). These levels may pose a potential threat to aquatic organisms. The maximum concentrations of *p,p'*-DDD, *p,p'*-DDE, and *p,p'*-DDT in sediment from 168 sites sampled along the southeastern coast of the United States as part of the Environmental Monitoring and Trends Program (EMAP) in 1994–1995 were 150.9, 34.2, and 35.0 µg/kg, respectively (Hyland et al. 1998). The median concentrations of these compounds were below the detection limit. DDT was monitored in surface water and sediment as part of the National Surface Water Monitoring Program in 1976–1980. The percent occurrence and maximum concentrations of the reported DDT analogs in sediments were: *p,p*'-DDT, 13.2%, 110.6 µg/kg; *o,p*'-DDT, 2.9%, 7.2 µg/kg; *p,p*'-DDE, 22.7%, 163.0 µg/kg; and *o,p*'-DDE, 0.5%, 1.3 µg/kg (Carey and Kutz 1985). Results were not presented for DDD. In 1983–1984, quarterly samples of bottom sediment were taken from six sites on tributaries of the Tennessee River near Huntsville, Alabama, and were analyzed for ΣDDT (Webber et al. 1989). From 1947 to 1970, DDT was manufactured along the tributary, and DDT-contaminated waste water was discharged into the river. The concentration of ΣDDT in sediment above the discharge point averaged less than 1 mg/kg dry weight. Remaining stations showed a decreasing gradient of ΣDDT with annual means ranging from 2,730 mg/kg at the closed site to the point of discharge to 12 mg/kg where the tributary empties into the Tennessee River 18 km away.

According to the U.S. Geological Survey's National Water Quality Assessment Plan initiated in 1991, which focuses on the water quality in more than 50 major river basins and aquifer systems, the frequency of detection of DDT and its metabolites in bed sediment in the 1990s remains high (USGS 1999). The metabolite with the highest frequency of detection was *p,p'*-DDE which was approximately 60% in urban areas, 48% in agricultural areas, and 46% in mixed land use areas followed by *p,p'*-DDD, *p,p'*-DDT, *o,p'*-DDD, *o,p'*-DDT, and *o,p'*-DDE. The frequency of detection of *o,p'*-DDT and *o,p'*-DDE was below 5%. Sediments can act as repositories for DDT and its metabolites, serving as sources for these compounds for long periods of time, given the long half-lives of these compounds and their resistance to biodegradation (Sanger et al. 1999). Because DDT and its metabolites will fractionate and concentrate in organic material, the sediments of some waterways, such as salt marshes, that receive a large amount of organic content in washloads discharged from sources of water originating from urban and agricultural areas can act as potential DDT repositories (Masters and Inman 2000). Also, the concentrations of DDT and its metabolites are high enough in some sediments to exceed the threshold effects level (TEL),

probable effects level (PEL), and the effects range low and median (ER-L, ER-M) for specific biota in marine and estuarine environments (Carr et al. 2000; Long et al. 1995).

The U.S. National Soils Monitoring Program has provided valuable information on the overall pattern of DDT residues in soil during the years following the DDT ban. Each year since the ban of DDT, approximately 1,500 samples were taken. The results for 1970 are tabulated below (Crockett et al. 1974):

Substance	Occurrence	Concentration (Fg/kg)	
		Mean	Range
p,p'-DDT	20%	180	10–69,300
o,p'-DDT	14%	40	10–11,700
p,p'-DDE	31%	50	10–6,820
o,p'-DDE	3%	<10	ND–510
ΣDDT	23%	300	10–113,090

ND = not detectable

The mean ΣDDT level in five U.S. cities ranged from 120 to 560 µg/kg in 1971 (Carey et al. 1979a). Urban areas generally had higher pesticide levels than did nearby agricultural areas except in some southern cities near which the agricultural use of pesticides was traditionally heavy.

DDT was heavily used in the corn belt in the mid-central United States. In a 1995–1996 sampling of 38 soils in this region, ΣDDT varied from below quantitation to 11,846 µg/kg, with a geometric mean value of 9.63 µg/kg (Aigner et al. 1998). The geometric mean concentrations for *p,p'*-DDE, *p,p'*-DDT, *p,p'*-DDD, and *o,p'*-DDT were 3.75, 4.67, 1.20, and 1.79 µg/kg, respectively. At least one DDT analog was found in 33 of the soils. Nine of the samples contained ΣDDT above 200 µg/kg, while the concentrations in the rest of the samples were below 40 µg/kg. Two garden soils had ΣDDT levels of 30 and 1.07 µg/kg. The soil with the high ΣDDT level was a muck soil with a concentration that was 10 times higher than the sample next highest in concentration and 1,000 times higher than most sample concentrations. *o,p'*-DDD was not found in any of the samples. The DDT/DDE ratio was determined in 21 of the samples and ranged from 0.5 to 6.6. It is interesting to note that the geometric mean *o,p'*-DDT concentration is 38% of the *p,p'*-DDT concentration. Since *o,p'*-DDT comprises between 15 and 21% of technical-grade DDT and 5.5% is comprised of other compounds, it would appear that *o,p'*-DDT degrades more slowly than *p,p'*-DDT. It was shown that the residue level of *p,p'*-DDT decreased about 70% in a silt loam in New Zealand over a 30-year period (1960–1989), while the *o,p'*-DDT level only

decreased by about 50% in the same time frame (Boul et al. 1994). Most of the degradation occurred during the time frame of 1960–1980, with very little loss occurring from 1980–1989. Forest soils in Maine that had been subject to aerial spraying with DDT had ΣDDT levels ranging from 270 to 1,898 µg/kg compared with a maximum concentration of 11 µg/kg in unsprayed locations. A study of DDT in agricultural soils in British Colombia, Canada report that ΣDDT levels ranged from 194 to 763 µg/kg in silt loam soils and from 2,984 to 7,162 µg/kg in muck soils (Aigner et al. 1998). The difference in residue levels reflects DDT's longer persistence in muck soil.

Hitch and Day (1992) reported that three soil samples taken near Dell City, Texas in 1980 contained an average of 4.94 and 0.46 mg/kg (dry weight) of DDT and DDE, respectively. It was suspected that the higher DDT concentrations indicated the possible illegal use of DDT. However, further analysis indicated that the "suspect" soil degraded DDT much slower than most soils and the high levels originally detected in soil were attributed to DDT persistence for many years. DDD was not measured in this study. DDT was extensively used in Arizona for 18 years, after which agricultural residues were closely monitored following a statewide moratorium on DDT use in January 1969. Levels of DDT plus metabolites in green alfalfa fell steadily from an average level of 0.22 mg/kg at the time of the ban to a level of 0.057 mg/kg 18 months later, and a level of 0.027 mg/kg after almost 7 years (Ware et al. 1978). After 3 years, residues in agricultural soils had decreased 23%. Furthermore, the ratio of DDE to DDT was increasing, indicating a transformation of DDT to DDE. Buck et al. (1983) reported similar results from monitoring these same sites over 12 years following the ban on DDT use. After 12 years, residues in green alfalfa averaged 0.020 mg/kg. At the end of the same period, combined DDT and DDE residues in agricultural soils had fallen from 1.2 to 0.39 mg/kg, while those in surrounding desert soil had fallen from 0.40 to 0.09 mg/kg.

In 1985, DDT, DDE, and DDD levels were measured at the Baird and McGuire Superfund Site in Holbrook, Massachusetts. Contamination was due to 60 years of mixing and batching of insecticides. In the highly contaminated areas, the average concentrations of DDT, DDE, and DDD were 61, 10, and 70 mg/kg, respectively. DDT, DDE, and DDD levels in leaf litter and leaf litter invertebrates ranged from 0.2 to 8.4, nondetected to 60, and 0.4 to 25 mg/kg, respectively (Menzie et al. 1992). The high levels of DDT relative to DDE probably indicate that the Superfund Site is largely anaerobic, and that DDT is largely degrading to DDD. In the Palos Verdes Shelf off of Los Angeles where waste from a large DDT manufacturer was discharged via a sewer outfall, sediments contain high levels of DDT isomers and metabolites. The levels of these compounds in surface sediment (0–2 cm) at five sites in the area were (chemical, concentration range): *o,p'*-DDE, 6–45 mg/kg; *p,p'*-DDE, 10–327 mg/kg; *p,p'*-DDD,

1–13 mg/kg; *p,p'*-DDD, 9–25 mg/kg; *o,p'*-DDT, not detectible–2 mg/kg; and *p,p'*-DDT, not detectible–6 mg/kg (Venkatesan et al. 1996).

In summary, DDT, DDE, and DDD have been detected in many soil and sediment surfaces throughout the world. Concentrations are highest in areas with a history of extensive DDT use and are often detected at concentrations close to 1 mg/kg (ppm) or more. Even though concentrations of DDT, DDE, and DDD in soils are declining due to the discontinued production and use of DDT in most countries, detectable levels will probably exist for decades to come because of the long persistence time of these compounds.

6.4.4 Other Environmental Media

From 1980 to 1983, 19, 59, and 14 samples of aquatic biota in EPA's water quality STORET database were analyzed for DDT, DDE, and DDD, respectively (Staples et al. 1985). The median levels for DDT, DDE, and DDD were 14, 26, and 15 µg/kg (wet weight), respectively. All samples tested had detectable levels of these chemicals.

According to the U.S. Geological Survey's National Water Quality Assessment Plan initiated in 1991, which focuses on the water quality in more than 50 major river basins and aquifer systems, DDT and its metabolites were detected in 94% of whole fish samples analyzed in the 1990s even though the total DDT concentration in fish continues to decline (USGS 1999). This is attributed to the presence of DDT in stream beds and continued inputs of DDT to streams as contaminated soils erode. The metabolite with the highest frequency of detection was *p,p'*-DDE followed by *p,p'*-DDD, *p,p'*-DDT, *o,p'*-DDD, *o,p'*-DDE, and *o,p'*-DDT. The frequency of detection of the *o,p'*-isomers was below 15%.

ΣDDT concentrations in fish (8 species, 23 samples) collected in August and September 1990 from 3 rivers in Michigan ranged from 4.71 to 976.92 µmol/kg, wet weight with a median of 82.1 µmol/kg (Giesy et al. 1994). The range of concentrations of DDT and metabolites were (chemical, range in µg/kg wet weight): *p,p'*-DDE, 3.54–627.13; *o,p'*-DDE, 0.15–37.95; *p,p'*-DDD, 0.43–58.82; *o,p'*-DDD, 0.13–81.70; and *p,p'*-DDT, <0.42–89.58. The mean ΣDDT concentrations in samples taken below dams that separated the rivers from the Great Lakes, 0.5–1.6 µmol/kg, were higher than those taken above, 0.05–0.35 µmol/kg. The relative contribution of DDE to ΣDDT was fairly constant in all three rivers both above and below the dams. The ratio of DDE:DDT ranged from 5 to 758, which suggests that the accumulation of DDE resulted from direct exposure to DDE in the diet rather than from recent exposure

to parent DDT. The fact that DDT is still observed in the fish was ascribed to long-range transport and deposition.

From 1986 to 1988, elements of the arctic marine food web near the Canadian Ice Island in the Arctic Ocean were sampled for DDT, DDE, and DDD (Hargrave et al. 1992). The average concentration of ΣDDT in plankton was 11.8 ng/g dry weight (43.5 µg/kg lipid), and the level increased with decreasing size of the plankton. Amphipods collected under pack ice in the open sea, over the Canadian continental shelf (190–315 m depth), and near the bottom of the Alpha Ridge (2,075 m depth), had mean ΣDDT concentrations of <57, 299, and 3,769 µg/kg dry weight (<347, 1,594, and 12,511 µg/kg lipid), respectively. Pelagic fish contained a mean ΣDDT of 200 µg/kg lipid, while abyssal fish (2,075 m) contained 819 µg/kg dry weight (1,465 µg/kg lipid). Similar comparisons have also been conducted on surface and deep-sea fish caught in the North and South Atlantic oceans and northwest Pacific ocean off California, showing higher concentrations of ΣDDT in deep-sea fish (Atlantic 175–1,090 µg/kg lipid; Pacific 2,380–2,420 µg/kg lipid) in comparison to surface fish (Atlantic 59–125 µg/kg lipid; Pacific 1,260–1,875 µg/kg lipid) (Looser et al. 2000). The DDT levels in Arctic plankton are generally lower than those reported elsewhere. It is not clear why the DDT levels are higher in organisms living at greater depths since DDT appears to be evenly distributed in the water column. Since DDT adsorbs to particulate matter that sinks into the sediment, as with detritus from aquatic organisms, fish and other organisms living at the bottom of the sea may accumulate higher levels of DDT than organisms living at the surface because their food chain is associated with benthic feeders. Regional differences in DDT levels in biota may be associated with the productivity of the ocean and greater sedimentation of detritus from aquatic organisms. Arctic mammals feeding on DDT-contaminated fish bioaccumulate the chemical in their fat (Bard 1999). The ringed neck seal (n=19) and polar bear (n=10) had mean ΣDDT concentrations of 1,482 and 266 µg/kg (lipid basis) (Muir et al. 1988). Beluga whales, ringed neck seals, and walruses near Baffin Island in the eastern Arctic had mean ΣDDT levels (wet weight) of 3.16, 0.33, and 1.42 µg/g, respectively (Kuhnlein et al. 1995).

Exposure to DDT could occur to populations that consume fish from DDT-contaminated marine environments. DDT in white croaker and Dover sole of the Southern California Bight, especially the Palos Verdes shelf area, are the highest in the United States. This is due to the fact that this area received 1,000,000 kg of DDT discharged into the Bight from the Montrose Chemical Company and also receives a large amount of sewage outfall from the southern California region (Zeng et al. 1999). Historically, DDT levels in these fish exceeded the Food and Drug Administration (FDA) action level of 5 mg/kg wet

weight of fish tissue, and fish intended for human consumption were confiscated to prevent human exposure to DDT (NOAA 1988).

Levels of ΣDDT have declined markedly since the early 1970s in fish, shellfish, and aquatic mammals (Addison and Stobo 2001; Bard 1999; Lauenstein 1995; Lieberg-Clark et al. 1995; Odsjo et al. 1997; Schmitt et al. 1990). Levels of DDT in fish were determined at 112 locations across the United States by the National Contaminant Biomonitoring Program in 1976 and 1984 (Schmitt et al. 1990). The mean concentrations of *p,p′*-DDT, *p,p′*-DDE, *p,p′*-DDD, and ΣDDT decreased from 50, 260, 80, and 370 µg/kg, respectively, in 1976 to 30, 190, 60, and 260 µg/kg, respectively, in 1984. A follow-up study of DDT in California sea lions reported a decrease in ΣDDT and DDE of over two orders of magnitude between 1970 and 1992 (Lieberg-Clark et al. 1995). ΣDDT concentrations in maternal grey seals decreased from 12 µg/g lipid in 1974 to 0.5 µg/g lipid in 1994; ΣDDT concentrations in seal pups were lower (60% of maternal concentrations) and decreased at similar rates over the same 20-year period (Addison and Stobo 2001). ΣDDT for mussels and oysters analyzed as part of the National Oceanic and Atmospheric Administration's National Status and Trends Mussel Watch Project in 1992 reported a geometric mean ΣDDT concentration for mussels and oysters at 51 sites of 20 µg/kg dry weight, down from a high of 53 µg/kg in 1977 (Lauenstein 1995). Over 90% of the ΣDDT present was as metabolites rather then the parent compounds (*p,p'*- and *o,p'*-DDT).

Among the metabolites of DDT are two methylsulfonyl metabolites of DDE, 2-methylsulfonyl-DDE (2-$MeSO_2$-DDE) and 3-methylsulfonyl-DDE (3-$MeSO_2$-DDE). These DDE metabolites are known to be persistent and have been measured in several species of mammals, including humans (for example, Bergman et al. 1994). The methylsulfonyl derivatives of DDE are formed through the action of phase I and II enzymes in the liver and the mercapturic acid pathway, as shown in Figure 3-4 of Chapter 3 (Letcher et al. 1998; Weistrand and Norén 1997). DDE is converted to an arene oxide through the action of the phase I cytochrome (CYP) P450 2B-type enzymes, followed by the conjugation of the arene oxide with glutathione as part of the phase II reactions. As part of the mercapturic acid pathway, the glutathione function is converted to a cysteine residue, which is then cleaved by C-S lyase to form the thiol-substituted intermediates, 2-SH-DDE or 3-SH-DDE. These thiolated DDE derivatives are methylated by adenosyl-methionine and then oxidized to the methylsufonyl derivatives of DDE. The ratio of 2-$MeSO_2$-DDE to 3-$MeSO_2$-DDE varies between species and tissue site (Bergman et al. 1994).

The two sulfonyl DDE metabolites have been measured in fat and various tissues of arctic mammals and in humans (Bergman et al. 1994; Haraguchi et al. 1989; Letcher et al. 1998; Norén et al. 1996;

Weinstrand and Norén 1997). In pooled adipose tissue of polar bears from 12 arctic regions, the concentration of 3-$MeSO_2$-DDE ranged from 0.60 to 11 µg/kg lipids, and the ratio of methylsulfone to DDE ranged from 0.009 to 0.056 with a mean of 0.033 (Letcher et al. 1995). These ratios may be the result of both biotransformation of DDE to methylsulfonyl-DDE in the animal and bioaccumulation (Letcher et al. 1998). In the polar bear food chain, lipid adjusted concentrations of 3-$MeSO_2$-DDE in arctic cod (<0.01 ng/g, in whole body pools), ringed seal (0.4 ng/g, in blubber) and polar bear (2.0 ng/g, in fat tissue) were measured, showing an increase in the concentration of 3-$MeSO_2$-DDE as a function of the trophic level (Letcher et al. 1998). In humans, methylsulfonyl-DDE has been measured in liver, lung, and adipose tissue at respective concentrations of 1.1, 0.3, and 6.8 ppb, wet weight (Haraguchi et al. 1989). In plasma, the concentration of 3-$MeSO_2$-DDE (0.1–2 ng/g lipid) was 2–3 orders of magnitude lower than the concentration of DDE (0.11–0.88 µg/g lipid) (Norén et al.1999). In a comparison of the concentrations of the two methylsulfonyl-DDE isomers in paired human liver and adipose tissues, 3-$MeSO_2$-DDE is the most abundant of the two isomers in these tissues (Weistrand and Norén 1997). In human breast milk, the concentration of 3-$MeSO_2$-DDE has been found to range between 0.4 and 5 ng/g lipid (Norén et al. 1996).

From 1979 to 1983, a study was conducted on the presence of DDT and metabolites in wildlife, predominantly birds, in orchards in central Washington State (Blus et al. 1987). Technical DDT was applied at very high rates to orchards in Washington between 1946 and 1970 with some areas probably receiving more than 1,000 kg/ha over this period. High levels of DDE, DDT, and DDD were found in the wildlife. Ninety-six percent of the wildlife samples (n=552) contained >0.01 µg/g of DDE, and 70% contained levels > 0.1 µg/g. In addition, many samples contained unusually low (#10:1) DDE:DDT ratios. The study attempted to identify whether the residues resulted from past legal use of DDT, ongoing illegal use, use of dicofol and related compounds, or foreign sources. While this matter wasn't completely resolved, it was suspected that residues were from several sources. However, residues in certain samples, particularly resident wildlife, apparently originated from past legal use of the insecticide. High concentrations have been noted in animals from areas of historically high DDT use. Mean GDDT concentrations were 1,362 µg/kg in spring peeper frogs living in southern Ontario, Canada (Russell et al. 1995). These concentrations exceed the suggested maximum concentration of 1,000 µg/kg proposed by the Great Lakes Water Quality Agreement of 1978. DDT was also applied at very high rates in the Delta region of Mississippi. The geometric mean concentration of *p,p'*-DDE residues in resident wood ducks decreased from 0.75 mg/kg in 1984 to 0.21 mg/kg in 1988 (Ford and Hill 1990). This decrease also corresponded with the reduction of residue levels in wood duck eggshells. Recent studies reporting concentrations of DDT and its metabolites in various biota is shown in Table 6-1.

Even with the reduction in the levels of DDT in the environment, there are still areas of concern where heavy applications of DDT during past legal uses of the pesticide have resulted in high concentrations of residual DDT and its metabolites that can, in turn, have potentially adverse effects on wildlife. For example, the transfer of DDT, DDE, and DDD from fruit orchard soils to American robins had been investigated in Okanagan, British Columbia, and Ontario, Canada, showing increasing concentrations of these compounds in soil6earthworm6robin eggs (Harris et al. 2000). In Okanagan, high average concentrations (mg/kg, dry weight) of DDE and DDT in soil (5.5 and 9.2), earthworms (52 and 21) and robin eggs (484 and 73) were consistent with the recorded contamination of this area. These concentrations are comparable to those where mortality or reproductive effects have been observed to occur in field studies. These results also illustrate one way in which DDT and its metabolites in soil can

Table 6-1. Concentrations of DDT and Metabolites in Biota

Species	Location	Year	Concentration	Type	Reference
Marine Mammals					
Pilot whale (n=7)	North Atlantic	Since 1987	3,847 (942–7,118) ng/g (f.w.) [DDE] 7,748 (1,708–13,035) ng/g (f.w.) [ΣDDT]	mean (range)	Becker et al. 1997[a]
Harbor Porpoise (n=5)	North Atlantic	Since 1987	3,260 (1,880–4,900) ng/g (f.w.) [DDE] 7,280 (4,690–11,200) ng/g (f.w.) [ΣDDT]	mean (range)	Becker et al. 1997[a]
Beluga whale (n=12)	Arctic	Since 1987	1,415 (142–2,230) ng/g (f.w.) [DDE] 2,492 (332–3,820) ng/g (f.w.) [ΣDDT]	mean (range)	Becker et al. 1997[a]
Beluga whale (n=12)	Cook Inlet	Since 1987	624 (65.9–1,630) ng/g (f.w.) [DDE] 1,050 (133–2,350) ng/g (f.w.) [ΣDDT]	mean (range)	Becker et al. 1997[a]
Northern fur seal (n=2)	North Pacific	Since 1987	1,190 (1,050–1,330) ng/g (f.w.) [DDE] 1,280 (1,090–1,480) ng/g (f.w.) [ΣDDT]	mean (range)	Becker et al. 1997[a]
Ringed seal (n=4)	Arctic	Since 1987	198 (27–350) ng/g (f.w.) [DDE] 543 (35–1,430) ng/g (f.w.) [ΣDDT]	mean (range)	Becker et al. 1997[a]
Harbour seals (n=18)	Northern Sea	1987	3,161 (355–6,598) µg/kg (f.w.) [ΣDDT]	mean (range)	Vetter et al. 1996
Harbour seals (n=32)	Northern Sea	1988	3,903 (1,501–11,475) µg/kg (f.w.) [ΣDDT]	mean (range)	Vetter et al. 1996
Beluga whale	Canadian Arctic	1988	3.16 µg/g (w.w.) [ΣDDT]	mean	Kuhnlein et al. 1995
Narwhal whale (n=unspecified)	Canadian Arctic	1988	2.73 µg/g (w.w.) [ΣDDT]	mean	Kuhnlein et al. 1995
Walrus (n=unspecified)	Canadian Arctic	1988	1.42 µg/g (w.w.) [ΣDDT]	mean	Kuhnlein et al. 1995
Ringed seal (n=unspecified)	Canadian Arctic	1988	0.33 µg/g (w.w.) [ΣDDT]	mean	Kuhnlein et al. 1995

Table 6-1. Concentrations of DDT and Metabolites in Biota *(continued)*

Species	Location	Year	Concentration	Type	Reference
Beluga whale (neonate) (n=1)	St. Lawrence estuary near Quebec	1991	702 ng/g (brain); 2,332 ng/g (kidney); 3,467 ng/g (liver); 2,230 ng/g (fat) [ΣDDT] 689 ng/g (brain); 2,289 ng/g (kidney); 3,370 ng/g (liver); 2,106 ng/g (fat) [DDE] ND (brain); ND (kidney); 15 ng/g (liver); 17 ng/g (fat) [DDD]		Gauthier et al. 1998
Terrestrial mammals					
Polar bear (n=320)	Arctic (16 regions)	1989–1993	219 µg/kg (f.w.) [DDE] 52–560 µg/kg (f.w.) [DDE]	median range of geomeans	Norstrom et al. 1998
Arctic ground squirrel (n=13)	Elusive Lake	1991–1993	6.13 (0.34–34.08) µg/kg (w.w.) (liver) [ΣDDT] 1.51 (0.33–5.57) µg/kg (w.w.) (liver) [DDE]	mean (range)	Allen-Gil et al. 1997
Arctic ground squirrel (n=6)	Feniak Lake	1991–1992	1.43 (0.19–5.16) µg/kg (w.w.) (liver) [ΣDDT] 0.86 (0.19–3.10) µg/kg (w.w.) (liver) [DDE]	mean (range)	Allen-Gil et al. 1997
Arctic ground squirrel (n=17)	Schrader Lake	1992–1993	12.25 (0.12–39.76) µg/kg (w.w.) (liver) [ΣDDT] 4.47 (0.12–13.63) µg/kg (w.w.) (liver) [DDE]	mean (range)	Allen-Gil et al. 1997
Birds					
Bald eagle chicks (n=51)	Great Lakes region	1990–1996	ND–0.0171 mg/kg (plasma) [DDT] 0.0036–0.1484 mg/kg (plasma) [DDE]	range	Donaldson et al. 1999
Bald eagle eggs (n=6)	Lake Erie	1974–1980	24.4 (13.8–35.8) mg/kg [DDE]	mean (range)	Donaldson et al. 1999
Bald eagle eggs (n=6)	Lake Erie	1989–1994	10.8 (2.7–22.2) mg/kg [DDE]	mean (range)	Donaldson et al. 1999
Bald eagle eggs (n=7)	Lake of the Woods, Canada	1993–1996	3.3 (0.9–12.6) mg/kg [DDE]	mean (range)	Donaldson et al. 1999

Table 6-1. Concentrations of DDT and Metabolites in Biota *(continued)*

Species	Location	Year	Concentration	Type	Reference
Blue heron eggs (n=10)	Southern Lake Michigan	1993	0.02 (ND–0.12) (µg/g) (w.w.) [DDT] 1.58 (0.23–13.00) (µg/g) (w.w.) [DDE] 0.03 (ND–0.12) (µg/g) (w.w.) [DDD]	mean (range)	Custer et al. 1998
Fish and shellfish					
Mussels and oysters	United States (51 sites)	1992	0.51–1,400 ng/g (d.w.) [ΣDDT] 20 ng/g (d.w.) [ΣDDT]	range of sites geomean	Lauenstein 1995
Clams	San Joaquin River (Orestimba Creek)	1992	4,350 ng/g (w.w.) [ΣDDT] 3,300 ng/g (w.w.) [DDE] 390 ng/g (w.w.) [DDD]	mean	Pereira et al. 1996
Clams	San Joaquin River (Dry Creek)	1992	29 ng/g (w.w.) [ΣDDT] 25 ng/g (w.w.) [DDE] 0.5 ng/g (w.w.) [DDD]	mean	Pereira et al. 1996
Clams	San Joaquin River (Mokelumne River)	1992	15 ng/g (w.w.) [ΣDDT] 13 ng/g (w.w.) [DDE] 0.5 ng/g (w.w.) [DDD]	mean	Pereira et al. 1996
Clams	San Joaquin River (Stanislaus River)	1992	24 ng/g (w.w.)[ΣDDT] 22 ng/g (w.w.) [DDE] <0.5 ng/g (w.w.)[DDD]	mean	Pereira et al. 1996
Mountain whitefish (10 composites from 7 sites)	Yakima River Basin, Washington	1989–1991	0.10–1.7 mg/kg (w.w.) (whole fish) [ΣDDT]	range of composites	Marien and Laflamme 1995

Table 6-1. Concentrations of DDT and Metabolites in Biota *(continued)*

Species	Location	Year	Concentration	Type	Reference
Largescale sucker (18 composites from 13 sites)	Yakima River Basin, Washington	1989–1991	0.05–4.37 mg/kg (w.w.) (whole fish) [ΣDDT]	range of composites	Marien and Laflamme 1995
Perch (n=5)	Lake Ørsjøen, Norway Mid-lake	1994	1.15 ng/g (w.w.), 1,643 ng/g (f.w.) [ΣDDT] 0.53 ng/g (w.w.), 757 ng/g (f.w.) [DDE] 0.26 ng/g (w.w.), 371 ng/g (f.w.) [DDD] 0.28 ng/g (w.w.), 400 ng/g (f.w.) [DDT]	mean	Brevik et al. 1996
Perch (n=5)	Lake Ørsjøen, Norway.	1994	5.59 ng/g (w.w.), 11,180 ng/g (f.w.) [ΣDDT] 2.56 ng/g (w.w.), 5,120 ng/g (f.w.) [DDE] 1.48 ng/g (w.w.), 2,960 ng/g (f.w.) [DDD] 1.15 ng/g (w.w.), 2,300 ng/g (f.w.) [DDT]	mean	Brevik et al. 1996
Pike (n=5)	Lake Ørsjøen, Norway Mid-lake	1994	7.3 ng/g (w.w.), 8,111 ng/g (f.w.) [ΣDDT] 3.5 ng/g (w.w.), 3,888 ng/g (f.w.) [DDE] 1.5 ng/g (w.w.), 1,667 ng/g (f.w.) [DDD] 1.8 ng/g (w.w.), 2,000 ng/g (f.w.) [DDT]	mean	Brevik et al. 1996
Lake trout (n=59)	Lake Ontario	1992	1.159 µg/g (w.w.) [DDE]	mean	Kiriluk et al. 1995
Rainbow smelt (n=8)	Lake Ontario	1992	0.256 µg/g (w.w.) [DDE]	mean	Kiriluk et al. 1995

[a]U.S. National Biomonitoring Specimen Bank

d.w. = dry weight; f.w. = fat weight basis; n = number; ND = not detected; geomean = geometric mean; w.w.= wet weight

be mobilized and bioaccumulated by soil organisms which, in turn, are further accumulated in higher trophic levels.

Market Basket Surveys indicated that there were decreases in the overall residue levels on a lipid basis of DDT and DDE in all classes of food tested from 1965 to 1975 (EPA 1980a). Between 1970 and 1973, DDE residues decreased only 27% compared to decreases of 86 and 89% for DDT and DDD, respectively (EPA 1980a). A study by Duggan et al. (1983) reported the following average residues of *p,p'*-DDT and *o,p'*-DDT in grocery items from 1969 to 1976: domestic cheese, 3 ppb; ready-to-eat meat, fish, and poultry, 5 ppb; eggs, 4 ppb; domestic fruits, 13 ppb; domestic leaf and stem vegetables, 24 ppb; domestic grains, 7 ppb; corn and corn products, 0.7 ppb; and peanuts and peanut products, 11 ppb.

Mean DDT residues by food group have been reported by Gartrell et al. (1985, 1986a, 1986b) as part of the FDA Total Diet Studies for October 1979–September 1980 and October 1980–March 1982. The average DDE and DDT residues for 12 food groups and the daily intake for each of these groups obtained from the Total Diet Studies are shown in Table 6-2. The highest intake of DDE is shown to come from meat, fish, and poultry. More recent Total Diet Studies have only reported the number of occurrences of a pesticide and not the concentration levels. In the survey for 1984–1986, there were 433 findings of DDE out of 1,872 samples analyzed (Gunderson 1995b). In the Total Diet Study for 1993–1994, *p,p'*-DDE was found in 115 out of 783 (15%) items analyzed (FDA 1995). In the 1999 FDA Total Diet Study, DDT was found in 255 out of 1,040 (22%) items analyzed (FDA 1999). In a recent FDA study, the mean concentrations of *p,p*-DDT and *o,p*-DDT ranged from 0.0002 to 0.005 ppm. The mean concentrations of *p,p*-DDE ranged from 0.0001 to 0.0257 ppm, with the highest values found in dairy, fish and vegetable products (FDA 2001). Analyses of samples from 10 states taken during fiscal years (FY) 1988 (n=13,980) and 1989 (n=13,085) resulted in a frequency of detection of 0.028 and 0.12%, respectively, for *p,p'*-DDT. DDE (any isomer) was detected in 1.5 and 0.99% of samples and *p,p'*-DDE in 0.18 and 0.25% of samples in 1988 and 1989, respectively (Minyard and Roberts 1991). Overall, these surveys indicate that DDT and DDE levels are very low in food commodities. However, with continued use of DDT in other countries, imported foods may continue to contribute small amounts of DDT and DDE to the daily diet of consumers. From 1981 to 1986, the FDA analyzed 13,283 imported agricultural commodities for pesticide residues. No commodities exceeded the EPA tolerance levels for DDT or DDE; however, 3.1% of the samples had detectable levels of DDT or DDE (Hundley et al. 1988). A pesticide residue survey of produce from 1989 to 1991 found that 41 out of 6,970 samples analyzed contained *p,p'*-DDE (Schattenberg and Hsu 1992).

Table 6-2. Average Residues in Food Groups and Average Daily Intake from U.S. FDA Total Diet Studies

	October 1979 – September 1980[a]				October 1980 – March 1982[b]			
	DDE		DDT		DDE		DDT	
Food Group	Residue (ppb)	Intake (µg/day)	Residue (ppb)	Intake (µg/day)	Residue (ppb)	Intake (µg/day)	Residue (ppb)	Intake (µg/day)
Dairy products	0.9	0.626	0	0	1.5	1.05	0	0
Meat, fish, and poultry	4.8	1.28	0.8	0.219	3.0	0.777	0	0
Grains and cereal	0	0	0	0	0	0	0	0
Potatoes	0.5	0.0847	<0.1	0.0079	0.5	0.0864	0	0
Leafy vegetables	1.7	0.0954	0.2	0.0137	2.4	0.132	0	0.0195
Legumes	0	0	0	0	<0.1	0.0014	0.4	0
Root vegetables	1.0	0.0309	0	0	4.6	0.146	0	0.0192
Garden vegetables	0.2	0.0185	0	0	0.1	0.0095	0.6	0
Fruits	0	0	0	0	<0.1	0.0081	0	0
Oils and fats	<0.1	0.0028	0	0	<0.1	0.0018	0	0
Sugar	<0.1	0.0042	0	0	<0.1	0.0018	0	0
Beverages	0	0	0	0	0	0	0	0

[a]Adapted from Gartrell et al. 1985
[b]Adapted from Gartrell et al. 1986b

Baking, frying, broiling, smoking, and microwaving all effectively reduce the total DDT concentration in fish and meat tissue (Bayarri et al. 1994; Khanna et al. 1997; Wilson et al. 1998). The average reduction in fish ranged from 16 to 82% and in lamb from 37 to 56% depending on cooking method. It is not clear whether residues are lost as a result of volatilization or decomposition or carried away in fat runoff. *p,p'*-DDT (but not *p,p'*-DDE or *p,p'*-DDD) decomposes on heating (see Table 4-2). Concentrations of *p,p*'-DDT in tomatoes could be reduced by between 11.5 and 33.7% by washing the fruit with acetic acid and sodium chloride solutions; the concentrations of *p,p*'-DDT, *o,p*'-DDE, *p,p*'-DDD, and *o,p*'-DDD residues in tomatoes could be reduced by up to approximately 80% from the fruit by simply removing the peel (Abou-Arab 1999). Production of tomato paste through home-canning methods reduced *p,p*'-DDT concentrations by 30.7%.

Djordjevik et al. (1995) assessed the chlorinated pesticide residues in U.S. and foreign cigarettes manufactured from the 1960s to the 1990s. Since 1970, the concentration of DDT analogs decreased by >98%. Concentration ranges of DDT-related compounds in samples of cigarettes manufactured between 1961 and 1979 and between 1983 and 1994 were (chemical, 1961–1979 levels, 1983–1994 levels): *p,p'*-DDD, 1,540–30,100 ng/g, 12.6–99.7 ng/g; *o,p'*-DDD, 396–7,150 ng/g, ND-19.0 ng/g; *p,p'*-DDT, 720–13,390 ng/g, 19.7–145 ng/g; *o,p'*-DDT, 105–1,940 ng/g; ND-88 ng/g; *p,p'*-DDE, 58–959 ng/g, 6.6–15.8 ng/g; and *p,p'*-DDMU (1-chloro-2,2-bis(*p*-chlorophenyl)ethylene), 92.7–2,110 ng/g, ND-27.5 ng/g. The transfer rate from tobacco into mainstream smoke amounts to 22% for DDD, 19% for DDT, and 27% for DDE.

Monitoring in older homes reveal that carpeting in these homes may have high levels of DDT, DDE, and DDD (Lewis et al. 1994). In one house built in 1930, the carpeting, which was believed to be at least 25 years old, contained up to 10.8 μg/m^2 or 5.7 μg/g of ΣDDT (*p,p'*-DDT, DDD, and DDE).

Organochlorine pesticides have been detected and quantified in composting feedstocks and finished compost (Büyüksönmez et al. 2000). Although banned for several decades, DDT and its metabolites have been detected in lawn trimmings and municipal waste compost as recently as 1990–1996, with concentrations of DDT, DDE, and DDD at 0.01–0.21, 0.01–0.11, and 0.007–0.13 ppm, respectively. The concentrations of the DDT, DDE, and DDD have been found to typically decrease as composting feedstocks are converted to finished compost. For example, DDT and DDE concentrations in lawn trimmings were found to decrease from 0.0466 and 0.0143 ppm to 0.0159 and 0.0108 ppm, respectively, after 90 days of composting. However, under some compositing conditions, DDE concentrations have been observed to increase in the finished compost (mean concentration of 0.0807 ppm, maximum value of

0.483 ppm) compared to the initial feedstock (mean concentration of 0.0516 ppm, maximum value of 0.201 ppm) (Strom 2000).

6.5 GENERAL POPULATION AND OCCUPATIONAL EXPOSURE

The general population is currently exposed to DDT and its metabolites primarily in food. As indicated in the previous section, although residue levels in food continue to slowly decline, there are measurable quantities in many commodities. A 1989 pesticide screening program of produce delivered to supermarkets in Texas, for example, found *p,p'*-DDE residues in 41 of the 6,970 produce samples tested (Schattenberg and Hsu 1992). An FDA study of residues in infant foods and adult food eaten by infants and children in which over 10,000 samples of domestic and imported foods were analyzed during 1985–1991 was published (Yess et al. 1993). ΣDDT was detected in 2 of 2,464 apples at a maximum concentration of 0.08 ppm; 312 of 2,464 plain milk samples at a maximum concentration of 0.92 ppm; 8 of 180 vitamin D fortified milk samples at a maximum concentration of 0.10 ppm; and 1 of 735 imported apple juice samples at 0.18 ppm (Yess et al. 1993). A similar 1992–1994 Canadian survey found DDE or DDT residues in 1 of 380 domestic heads of lettuce; 1 of 769 domestic potatoes; 36 of 612 imported carrots; 4 of 721 imported cucumbers; 1 of 702 imported heads of lettuce; 14 of 121 imported green onions; 7 of 17 imported parsnips; 1 of 933 imported peppers; 5 of 264 imported spinach; 1 of 155 imported tomato pastes; and 1 of 1,153 imported tomatoes (Neidert and Saschenbrecker 1996). In a U.S. Market Basket study of ready to eat foods, *o,p'*-DDE was detected 8 times in 4 different food items at an average concentration of 0.0025 µg/g; *p,p'*-DDE was detected 1,700 times in 142 different food items at an average concentration of 0.0026 µg/g; *o,p'*-DDT was detected 5 times in 4 different food items at an average concentration of 0.0053 µg/g; *p,p'*-DDT was detected 98 times in 31 different food items at an average concentration of 0.0045 µg/g (KAN-DO Office and Pesticide Team 1995).

Because of the extreme persistence of DDT and DDE, it is anticipated that low levels of residues will be present in commodities for decades. In fact, depending on use and export patterns in other countries, levels in the diet may even increase (Coulston 1985). Even in domestic commodities, the potential for low levels of dietary exposure of consumers may result from residues bioaccumulated in some food items, including fish.

The estimated dietary intake of DDT and metabolites in the United States was 62 µg/person/day in 1965, 240 µg/person/day in 1970, and 8 µg/person/day in 1974 (Coulston 1985). The FDA Adult Total Diet

Study for October 1979–September 1980 (FY 1980) found that the intakes of ΣDDT, DDE, DDT, and DDD were 0.034, 0.003, 0.031, and <0.001 µg/kg body weight/day, respectively, down from highs of 0.093, 0.004, 0.087, and 0.002, respectively, in FY 1979 (Gartrell et al. 1986a). The adult intake was assumed to be the diet of a 16- to 19-year-old male. Analogous studies for infants and toddlers for FY 1980 reported daily intakes of the respective DDTs as 0.034, 0.034, ND (not determined), and ND µg/kg body weight/day for infants and 0.049, 0.045, 0.002, and 0.002 µg/kg body weight/day for toddlers (Gartrell et al. 1986b). Estimated dietary intakes of DDT determined from the FDA Total Diet Studies for June 1984–April 1986 and July 1986–April 1991 for eight population groups appear in Table 6-3 (Gunderson 1995a, 1995b). To facilitate comparisons of DDT intakes from Gunderson (1995a, 1995b) with those of earlier estimates (Coulston 1985), the daily intake of ΣDDT for a 70 kg 16-year-old male as reported by Gunderson (1995a, 1995b) would have been 6.51, 2.38, 1.49, and 0.97 µg/day for 1978–1979, 1979–1980, 1984–1986, and 1986–1991, respectively. The acceptable daily intake of DDT established by WHO/FAO is 20 µg/kg/day (WHO 1991).

Exposure to DDT, DDE, and DDD in imported foods is minimized due to FDA enforcement programs. FDA randomly collects and analyzes a wide variety of imported commodities (e.g., coffee, tropical fruits) to determine if pesticide residues are above EPA tolerances. Pesticide tolerances established by EPA apply equally to domestic and imported food (Wessel and Yess 1991).

Arctic indigenous people ingest high levels of DDT from traditional foods. A study covering three age groups in communities in the eastern and western Canadian Arctic found the average daily ΣDDT intake of 24.2 to 27.8 µg/day for the eastern Arctic community and 0.51 to 1.0 µg/day for the western Arctic communities (Kuhnlein et al. 1995). The foods with the highest ΣDDT concentrations were raw Beluga whale blubber (316 µg/g wet weight) and aged Narwhal whale blubber (273 µg/g wet weight) in the eastern Arctic, and baked Loch (species of fish) liver (1.85 µg/g wet weight) and smoked Canada goose meat (1.47 µg/g wet weight) in the western Arctic.

In 1986–1988, EPA collected data at two sites, Jacksonville, Florida and Springfield/Chicopee, Massachusetts, to assess the nonoccupational exposure to pesticides (NOPES) for residents of these cities (Whitmore et al. 1994). Indoor *p,p'*-DDE and *p,p'*-DDT levels in air were higher than outdoor levels in these communities, and the highest number of indoor air samples with detectable DDT was observed in

Table 6-3. Mean Daily Intake of DDT Per Unit Body Weight (µg/kg body weight/day) for Various Age Groups in the United States

Analyte	6–11 mo	2 yr	14–16 yr F	14–16 yr M	25–30 yr F	25–30 yr M	60–65 yr F	60–65 yr M
1984–1986								
ΣDDT	0.0485	0.0499	0.0154	0.0213	0.0128	0.0155	0.0111	0.0124
o,p'-DDE	0.0002	0.0001	<0.0001	<0.0001	<0.0001	<0.0001	0.0001	<0.0001
p,p'-DDE	0.0468	0.0484	0.0149	0.0207	0.0123	0.0150	0.0105	0.0119
p,p'-DDT	0.0004	0.0010	0.0003	0.0004	0.0003	0.0003	0.0003	0.0003
p,p'-DDD	0.0011	0.0004	0.0002	0.0002	0.0002	0.0002	0.0002	0.0002
1986–1991								
ΣDDT[1]	0.0448	0.0438	0.0138	0.0139	0.0106	0.0127	0.0090	0.0104
o,p'-DDE	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001
p,p'-DDE	0.0441	0.0420	0.0130	0.0151	0.0099	0.0119	0.0082	0.0096
o,p'-DDT	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001
p,p'-DDT	0.0004	0.0011	0.0005	0.0005	0.0005	0.0005	0.0006	0.0006
p,p'-DDD	0.0003	0.0007	0.0003	0.0003	0.0002	0.0003	0.0002	0.0002

Source: Gunderson 1995a, 1995b

[1]The average daily ΣDDT intake of 0.8 Fg/day for an adult used in Section 1.3 was derived from the average intakes for 25–30 year old males and females assuming a body weight of 70 kg. The data presented in the table were derived from the June 1984 through April 1991 FDA Total Diet Studies.

F = female; M = male; mo = month; yr = year

the spring in Jacksonville (14%) and in the winter in Springfield/Chicopee (20%), with estimated mean air DDE and DDT concentrations of #1.0 ng/m^3. Mean ΣDDT air exposures were estimated as 22 ng/day in Jacksonville and 94 ng/day in Springfield/Chicopee. For comparison, dietary exposures in these two communities for 1982–1984 were estimated to be around 1,900 ng/day. Nine of 11 carpets tested in Jacksonville contained ΣDDT with median and mean levels of 0.7 and 1.2 µg/g, respectively.

Until 1970, tobacco smoke contributed significantly to the intake of DDT by people, but since then, the amount of DDT in tobacco has dropped markedly and today, cigarette smoke is a minor source of human exposure (Djordjevic et al. 1995).

Because of the extremely low solubility of DDT and DDE in water and the efficiency of standard water treatment methods in eliminating DDT-type chemical residues, intake of these compounds via drinking water is believed to be negligible. The criterion cited in the EPA Ambient Water Quality Criteria document is 0.059 ng/L, based on ingestion of 2L of drinking water per day plus 6.5 g of fish and shellfish per person (EPA 1999a). This criterion corresponds to an estimated increased cancer risk level of $1x10^{-7}$ or 1 in 10 million.

Data indicate that, even with relatively high doses, there is minimal absorption of DDT through skin (Gaines 1969; Wester et al. 1990; Wolfe and Armstrong 1971). Therefore, exposure via dermal absorption was considered to be negligible. However, in reviewing the literature and using a dermal absorption factor of 15% measured in their laboratory, Moody and Chu (1995) calculated that in the worse-case scenario where a swimmer was in contact with 1 ppm of DDT from a water slick or sediment for 1 hour, a swimmer would absorb 200 µg of DDT, equivalent to a dose from a meal of contaminated fish.

DDT and its metabolites are ubiquitous in the atmosphere but are present in such low concentrations that exposure via inhalation is negligible. Potential inhalation of relatively high levels of DDT should be possible only in areas of production or formulation. Wolfe and Armstrong (1971) estimated a respiratory exposure potential of 14.1 mg/person/hour for formulating plant workers; however, no current data were located on exposure of workers utilizing modern technology in the production and formulation of these compounds.

DDT and DDE elimination from the body is not an efficient process; therefore, tissue levels will increase with repeated exposure if the absorbed dose is high enough. For this reason, body burdens of DDT and

DDE tend to correspond with exposure levels, as indicated in long-term studies. From July 1969 to 1975, residues of DDT and its metabolites were measured in human adipose tissue collected through an annual, national survey — the National Human Monitoring Program for Pesticides (Kutz et al. 1977). During that time, levels of DDT and DDE in tissue samples declined. However, the frequency of occurrence in lipid samples did not decline, indicating both a long biological half-life and the ubiquitous occurrence of these compounds in the population. For FY 1970–1974, all samples were positive for DDT and metabolites (a total of 1,412 samples). Using all age groups sampled, the geometric mean lipid DDT and metabolite (combined) levels reported for each year from 1970–1974 were 7.88, 7.95, 6.88, 5.89, and 5.02 ppm, respectively. Notable trends reported in Kutz et al. (1977) included increasing body burden with increasing age as well as a significant increase in residues in blacks when compared to whites. Results published for 1975 showed little change compared to 1974 (Kutz et al. 1979). Exposure to DDT in nonoccupationally exposed individuals, as manifested by their plasma DDE concentrations, was most reliably predicted by age and serum cholesterol concentration (Laden et al. 1999). Kutz et al. (1991) contains a listing of studies on DDT, DDE, and DDD levels in human adipose tissue in the general population of various countries from the 1950s to the mid 1980s.

The Second National Health and Nutrition Examination Survey (NHANES II) has served as a continuation of the National Human Monitoring Program; however, published results have been few. Murphy and Harvey (1985) published selected results from the NHANES II survey for 1976–1980 based on data from the Northeast, Midwest, and South. These results are based, not on adipose samples, but on serum samples. For the years covered, 3,300 serum specimens were analyzed for DDT and DDE. In 31% of those samples *p,p*'-DDT was detected, with a median quantifiable level of 3.3 ppb (0.0033 ppm). However, *p,p*'-DDE was detected in 99% of samples tested, with a median quantifiable level of 11.8 ppb (0.0118 ppm). The limits of detectability was 2 ppb for *p,p*'-DDT and 1 ppb for *p,p*'-DDE. These results offered further proof of the extensive biological half-life of DDE as compared to DDT. Again, for both compounds, serum levels increased with increasing age. These data were not reported for each year, but a decreasing trend could be expected based on the data of Murphy and Harvey (1985). A more recent report on NHANES II for the period of 1976–1980 confirmed the above results on serum samples from 5,994 persons. *p,p′*-DDE was detected in the serum of 99.5% of persons with a median level of 12.6 ppb (range: 0–379 ppb) whereas *p,p′*-DDT was quantifiable (>2 ppb) in only 10% of serum samples (Stehr-Green 1989). Levels of *p,p′*-DDE increased with age and were higher in farm residents and in the South and West.

Results of EPA's 1986 National Human Adipose Tissue Survey (NHATS) in which 671 adipose tissue specimens were pooled into composite samples according to age, census region, sex, and race showed significant differences in *p,p'*-DDT and *p,p'*-DDE levels depending on age and census region (Lordo et al. 1996). The concentration of both compounds increased with age group, and while levels of *p,p'*-DDT were highest in the Northeast and lowest in the South, those of *p,p'*-DDE were highest in the West and lowest in the North Central region. Levels of both compounds had significantly increased from the 1984 NHATS. The estimated national mean with relative standard error (%) *p,p'*-DDT concentrations for the 1982, 1984, and 1986 NHATS were 189 (31%), 123 (11%), and 177 (20%) ng/g, respectively. Those for *p,p'*-DDE were 1,840 (350%), 1,150 (90%), and 2,340 (270%) ng/g, respectively. A 1985 survey of 108 Canadian autopsy samples resulted in respective mean and maximum levels of *p,p'*-DDE at 811 and 6,070 ng/g and *p,p'*-DDT at 48 and 250 ng/g (Mes et al. 1990). Adeshina and Todd (1990) analyzed DDT isomer and metabolite levels in 35 human adipose tissue samples of North Texas residents who were not occupationally exposed to DDT. The samples were obtained during autopsy in 1987 and 1988. The geometric mean concentrations were (substance, ng/g lipid): *o,p'*-DDE, 8 ng/g; *p,p'*-DDE, 679 ng/g; *o,p'*-DDT, 14 ng/g; *p,p'*-DDT, 294 ng/g; and ΣDDT, 1,031 ng/g. The ΣDDT levels can be compared with those from the human adipose tissue survey which were 7,950 ng/g lipid in 1970, 5,150 ng/g lipid in 1974, and 1,670 ng/g lipid in 1983 (Adeshina and Todd 1990).

DDT in milk is discussed in Section 6.6. Levels of DDE and DDT in human milk, blood, and tissue appear in Table 6-4. Correlations in the concentrations of DDT and its metabolites in human milk, adipose tissue, and blood serum have been observed. The lipid adjusted mean concentrations of *p,p*'-DDT, *p,p*'-DDE, and *p,p*'-DDD in human milk (0.65, 4.00, and 0.01 mg/kg, respectively) have been shown to correlate well (r^2=0.95, 0.89, and 0.75, respectively) with concentrations in adipose tissue (1.22, 4.36, and 0.02 mg.kg, respectively) (Waliszewski et al. 2001). The lipid adjusted serum concentrations of *p,p*'-DDE were observed to correlate with concentrations of this compound in breast adipose tissue (Dorea et al. 2001). The concentrations of *p,p'*-DDT and *p,p'*-DDE in blood serum taken from mothers in Veracruz, Mexico (1.848 and 4.378 mg/kg fat, respectively) have also been observed to correlate (r^2=0.854 and 0.779, respectively) with concentrations in umbilical cord blood (2.800 and 4.676 mg/kg fat, respectively) (Waliszewski et al. 2000).

Table 6-4. Levels of DDT Compounds in Human Milk, Blood, and Tissues — Recent Studies

DDT compound	Population	Tissue	Mean[a] concentration	Units as reported	Reference
Milk					
p,p'-DDT	Maternity patients in Mexico City, Cuernavaca and rural Morelos	Milk fat	0.71, 1.69, 4.84[b]	mg/kg	Elvia et al. 2000
p,p'-DDT	Mothers in Sweden	Milk fat	14	µg/g	Norén and Meironyté 2000
p,p'-DDT	Maternity patients in Mexico City	Milk fat	0.162	mg/kg	Torres-Arreola et al. 1999
p,p'-DDT	Women in Germany	Milk fat	0.7 (estimated from graph)	mg/kg	Scheele et al. 1995
o,p'-DDT	Maternity patients in Mexico City	Milk fat	0.138	mg/kg	Torres-Arreola et al. 1999
p,p'-DDE	Maternity patients in Mexico City, Cuernavaca and rural Morelos	Milk fat	3.85, 6.51, 16.52[b]	mg/kg	Elvia et al. 2000
p,p'-DDE	Mothers in Sweden	Milk fat	129	µg/g	Norén and Meironyté 2000
p,p'-DDE	Inuit women in Canada	Milk fat	962	µg/kg	Dewailly et al. 2000
p,p'-DDE	Quebec women between 1989 and 1990 (n=536)	Milk fat	0.34	mg/kg	Dewailly et al. 1996
p,p'-DDE	Maternity patients in Mexico City	Milk fat	0.594	mg/kg	Torres-Arreola et al. 1999
p,p'-DDE	Maternity patients in Veracruz, Mexico	Milk fat	5.302	mg/kg	Pardio et al. 1998

Table 6-4. Levels of DDT Compounds in Human Milk, Blood, and Tissues — Recent Studies *(continued)*

DDT compound	Population	Tissue	Mean[a] concentration	Units as reported	Reference
p,p'-DDE	Mothers of hospitalized children in Zagreb, Croatia	Milk fat	0.318	mg/kg	Krauthaker et al. 1998
ΣDDT	Canadian women - 1986 (n=412)	Milk fat	0.385	mg/kg	Smith 1999
ΣDDT	Arkansas women - 1986 (n=536)	Milk fat	0.99	mg/kg	Smith 1999
Blood					
p,p'-DDT	Workers in Sao Paulo, Brazil	Serum	13.5 (DDT appliers) 1.5 (unexposed)	µg/L	Minelli and Ribeiro 1996
o,p'-DDT	Workers in Sao Paulo, Brazil	Serum	<0.7–4.7 (range; DDT appliers)	µg/L	Minelli and Ribeiro 1996
p,p'-DDE	Workers in Sao Paulo, Brazil	Serum	64.3 (DDT appliers) 14.3 (unexposed)	µg/L	Minelli and Ribeiro 1996
p,p'-DDT	Men in southeast Sweden	Blood plasma	0.11 (lipid adjusted)	ng/g	Asplund et al. 1994
p,p'-DDE	Woman hospital patients in New Haven, Connecticut	Serum (lipid-adjusted)	967 (median), <1.0–2261.5 (range) (n=36)	ng/g	Archibeque-Engle et al. 1997
p,p'-DDE	Iowa and North Carolina farmers and spouses	Serum	0.39–6.51 (range)	µg/L	Brock et al. 1998
o,p'-DDE	Iowa and North Carolina farmers and spouses	Serum	0.71–2.31 (range)	µg/L	Brock et al. 1998

Table 6-4. Levels of DDT Compounds in Human Milk, Blood, and Tissues — Recent Studies *(continued)*

DDT compound	Population	Tissue	Mean[a] concentration	Units as reported	Reference
p,p'-DDE	Women without breast cancer in Long Island	Serum	4.7	µg/L	Stellman et al. 1998
p,p'-DDE	New York University Women's Health Study (1985 to 1991)	Serum	11.0±9.1 in cancer patients (n=58) 7.7±6.8 controls (n=171)	µg/L	Wolff et al. 1993
p,p'-DDE	Female hospital patients in New York City	Plasma	6.93–7.29 (range of mean values)	µg/L	Gammon et al. 1997
p,p'-DDE	Female hospital patients in New York City	Plasma (lipid-adjusted)	0.963–0.997 (range of mean values)	µg/mL	Gammon et al. 1997
p,p'-DDE	Mothers in Veracruz, Mexico	Serum	14.5	ng/mL	Waliszewski et al. 2001
p,p'-DDE	Men in southeast Sweden	Plasma	2.4–14 (range of mean values among groups of men with different levels of fish consumption)	ng/g	Asplund et al. 1994
p,p'-DDE	Men in southeast Sweden	Plasma (lipid-adjusted)	750–4500 (range of mean values among groups of men with different levels of fish consumption)	ng/g	Asplund et al. 1994
DDE	Controls in a case-control study nested within the Nurses Health Study (n=240)	Plasma	7.09	ppb[c]	Laden et al. 1999
p,p'-DDE	Four groups of refugees from Asia, 'USSR', Africa, 'Yugoslavia' (n=103); Controls from Germany (n=34)	Plasma	2.30–16.90 (range of median values) 12.20–93.00 (range of maximum values) (refugees) 1.14 (median), 4.97 (maximum) (controls)	µg/L	Schmid et al. 1997

Table 6-4. Levels of DDT Compounds in Human Milk, Blood, and Tissues — Recent Studies *(continued)*

DDT compound	Population	Tissue	Mean[a] concentration	Units as reported	Reference
p,p'-DDT	Residents of Nainital, India	Serum	4.46 (mean), range (0.78–14.29)	mg/L	Dua et al. 2001
p,p'-DDE	Residents of Nainital, India	Serum	1.55 (mean), range (0.14–4.10)	mg/L	Dua et al. 2001
p,p'-DDD	Residents of Nainital, India	Serum	0.91 (mean), range (ND–2.82)	mg/L	Dua et al. 2001
p,p'-DDE	New Bedford area infants	Cord blood	0.493	ng/g serum	Korrick et al. 2000a
p,p'-DDE	Maternity patients in Veracruz, Mexico	Cord blood	6.0	ng/mL	Waliszewski et al. 2001
p,p'-DDE	Maternity patients in Nicaragua	Venous blood (lipid-adjusted)	7.12 (mean), range (0–35.23) (n=52)	ng/g	Dorea et al. 2001
p,p'-DDE	Maternity patients in Nicaragua	Cord blood (lipid-adjusted)	6.39 (mean), range (0–9.35) (n=52)	ng/g	Dorea et al. 2001
p,p'-DDT	Great Lakes fishermen (n=30); Controls (n=180)	Serum	0.3 (median), 0.05–0.8 (range) ND (controls)	ppb[c]	Anderson et al. 1998
o,p'-DDT	Great Lakes fishermen (n=30); Controls (n=180)	Serum	0.06 (median), 0.03–0.3 (range) ND (controls)	ppb[c]	Anderson et al. 1998
p,p'-DDE	Great Lakes fishermen (n=30); Controls (n=180)	Serum	5.2 (median), 0.6–23.9 (range) 2.8 (median), ND–38.5 (range) (controls)	ppb[c]	Anderson et al. 1998

Table 6-4. Levels of DDT Compounds in Human Milk, Blood, and Tissues — Recent Studies *(continued)*

DDT compound	Population	Tissue	Mean[a] concentration	Units as reported	Reference
DDE	Frequent GLSCF (Lake Michigan) males (n=98); females (n=83); male controls (n=23); female controls (n=22)	Serum	6.9 (males), 2.9 (females), 2.6 (controls, males), 1.4 (controls, females) (geometric means)	ppb[c]	Hanrahan et al. 1999
DDE	Frequent GLSCF (Lake Huron) males (n=65); females (n=37); male controls (n=3); female controls (n=3)	Serum	3.5 (males), 2.3 (females), 2.6 (controls, males), 0.6 (controls, females) (geometric means)	ppb[c]	Hanrahan et al. 1999
DDE	Frequent GLSCF (Lake Erie) males (n=89); females (n=67); males controls (n=31); female controls (n=17)	Serum	3.8 (males), 2.0 (females), 2.0 (controls, males), 1.7 (controls, females) (geometric means)	ppb[c]	Hanrahan et al. 1999
*Σ*DDT	1982 Great Lakes fish eaters (n=572); controls (n=419)	Serum	28.8 10.6 (controls)	ppb[c]	Hovinga et al. 1992
*Σ*DDT	1982 Southern Great Lakes fish eaters (n=115); controls (n=95)	Serum	25.8 9.6 (controls)	ppb[c]	Hovinga et al. 1992
*Σ*DDT	1989 Southern Great Lakes fish eaters[d] (n=115); controls (n=95)	Serum	15.6 6.8 (controls)	ppb[c]	Hovinga et al. 1992
Adipose and other tissue					
p,p'-DDT	Children in Germany	Adipose	0.6 (estimated from graph)	mg/kg	Scheele et al. 1995

Table 6-4. Levels of DDT Compounds in Human Milk, Blood, and Tissues — Recent Studies *(continued)*

DDT compound	Population	Tissue	Mean[a] concentration	Units as reported	Reference
p,p'-DDT	Children in Germany	Bone marrow (lipid-adjusted)	1.75 (estimated from graph)	mg/kg	Scheele et al. 1995
p,p'-DDT	Woman hospital patients in New Haven, Connecticut	Adipose, breast (lipid-adjusted)	132.2 (median), 54.0–418.2 (range) (n=36)	ng/g	Archibeque-Engle et al. 1997
p,p'-DDT	Maternity patients in Veracruz, Mexico	Adipose, abdomenal (lipid-adjusted)	1.22 (mean), 0.01–9.03 (range) (n=60)	mg/kg	Waliszewski et al. 2001
p,p'-DDE	Maternity patients in Veracruz, Mexico	Adipose, abdomenal (lipid-adjusted)	4.36 (mean), 0.31–16.04 (range) (n=60)	mg/kg	Waliszewski et al. 2001
p,p'-DDD	Maternity patients in Veracruz, Mexico	Adipose, abdomenal (lipid-adjusted)	0.02 (mean), ND–0.25 (range) (n=60)	mg/kg	Waliszewski et al. 2001
p,p'-DDE	Woman hospital patients in New Haven, Connecticut	Adipose, breast (lipid-adjusted)	970 (median), 240.0–2,644.1 (range) (n=36)	ng/g	Archibeque-Engle et al. 1997
p,p'-DDT	Adults in Germany	Bone marrow (dry lipid-adjusted)	0.364	ppm[c]	Scheele 1998
p,p'-DDE	Adults in Germany	Bone marrow (dry lipid-adjusted)	1.689	ppm[c]	Scheele 1998
p,p'-DDE	Women without breast cancer in Long Island	Adipose	546.7	ng/g	Stellman et al. 1998
p,p'-DDE	Adults in Sweden who suffered sudden death	Liver (lipid-adjusted)	836	ng/g	Weistrand and Norén 1998

Table 6-4. Levels of DDT Compounds in Human Milk, Blood, and Tissues — Recent Studies *(continued)*

DDT compound	Population	Tissue	Mean[a] concentration	Units as reported	Reference
p,p'-DDE	Adults in Sweden who suffered sudden death	Adipose, abdominal	788	ng/g	Weistrand and Norén 1998
p,p'-DDE	FY1986 National Adipose Tissue Survey Composite samples (n=50, from 671 specimens)	Adipose	2,340 (SE 12) (nation) 1,710 (SE 22%) (0–14 years) 2,150 (SE 17%) (15–44 years) 3,080 (SE 13%) (45+ years)	ng/g	Lordo et al. 1996
p,p'-DDT	FY1986 National Adipose Tissue Survey Composite samples (n=50, from 671 specimens)	Adipose	177 (SE 11%) (nation) 73.0 (SE 36%) (0–14 years) 177 (SE 16%) (15–44 years) 252 (SE 13%) (45+ years)	ng/g	Lordo et al. 1996
p,p'-DDE	Women patients at Hartford Hospital, Hartford, Connecticut	Adipose, breast (lipid basis)	2,200±1,470 cancer patients (n=20) 1,487±842 controls (n=20)	ng/g	Falck et al. 1992
p,p'-DDT	Women patients at Hartford Hospital, Hartford, Connecticut	Adipose, breast (lipid-adjusted)	216±174 cancer patients (n=20) 148±75 controls (n=20)	ng/g	Falck et al. 1992

[a]Arithmetic mean concentrations are reported unless otherwise specified.
[b]Geometric mean concentrations
[c]ppm=µg/g; ppb=ng/g
[d]Same Southern Great Lakes fish eaters that participated in the 1982 study.

FY = fiscal year; ND = not detected; SE = standard error

Comparison of the concentrations of DDT and its metabolites in breast tissues and serum have been conducted in women with breast cancer in comparison to control subjects. The mean concentrations (unadjusted for age) of *p,p*'-DDT and *p,p*'-DDD in breast tissues of U.S. women with breast cancer (261.6 and 9.8 ng/g lipid, respectively) did not statistically differ (p=0.23 and 0.79) from those measured in control subjects (267.3 and 24.0 ng/g lipid); however, the concentration of *p,p*'-DDE was statistically higher (p=0.006) in breast cancer patients (800.0 ng/g lipid) than in control subjects (709.1 ng/g lipid) (Bagga et al. 2000). When the mean concentrations for *p,p'*-DDT, *p,p'*-DDE, and *p,p'*-DDD were adjusted for age, there was no statistical difference (p#0.001) between cases and controls for all three compounds. Statistically higher concentrations ($p<0.05$) of *p,p*'-DDE in serum were observed in both premenopausal (2.40 µg/g lipid) and postmenopausal (5.10 µg/g lipid) breast cancer patients from Mexico City, Mexico, in comparison to controls (1.93 and 3.12 µg/g lipid, respectively) (Romieu et al. 2000). The increased concentrations of *p,p*'-DDE in breast tissues of cancer patients has been attributed to increased exposures of these women to *p,p*'-DDE rather than to differences in the metabolism of *p,p*'-DDE by cancer cells (Romieu et al. 2000). However, in a study of breast cancer patients and control subjects in the New York University Women's Health Study, there was no statistical difference between the geometric means of the *p,p'*-DDE concentrations in serum (1,097 ng/g lipid in patients versus 977 ng/g lipid in controls) obtained from these two groups of women (Wolff et al. 2000b). A number of other studies have also found no statistical differences in the mean concentrations of DDT and/or its metabolites in serum or adipose tissue between cancer cases and controls (Demers et al. 2000; Dorgan et al. 1999; Helzlsouer et al. 1999; Krieger et al. 1994; Laden et al. 2001a; Liljegren et al. 1998; Lopez-Carrillo et al. 1997; Mendonca et al. 1999; Moysich et al. 1998; Schecter et al. 1997; Unger et al. 1984; van't Veer et al. 1997; Ward et al. 2000; Wolff et al. 2000; Zheng et al. 1999, 2000).

Methyl sulfonyl metabolites of *p,p'*-DDE, primarily the 3-methylsulfone isomer, have been found in seven surveys of human milk in Sweden between 1972 and 1992 (Norén et al. 1996). In that time, levels declined from 5.05 to 0.46 ng/g lipids and the ratio of the 3-methylsulfone metabolite to *p,p'*-DDE remained constant at 0.002.

Fish from areas like the Great Lakes and Baltic Sea appear to be an important source of exposure to DDT and DDE, and human blood levels of these compounds have been found to correlate with the consumption of fish containing high levels of DDT and DDE (Anderson et al. 1998; Asplund et al. 1994; Hovinga et al. 1992). A Swedish study found that the mean plasma lipid concentrations of *p,p'*-DDE were 750, 1,200, and 4,500 ng/g in groups of men eating no fish, moderate quantities of fish, and large quantities of fish, respectively, from the Baltic Sea (Asplund et al. 1994). The respective lipid plasma

concentrations of *p,p'*-DDT in these groups of men were 20, 45, and 130 ng/g. The mean serum DDT level in individuals eating more than 20 pounds of sport-caught Great Lakes fish dropped from 25.8 to 15.6 ppb (65% decrease) during the period from 1982 to 1989. Mean serum DDT levels in the controls dropped from 9.6 to 6.8 ppb (41% decrease). It was concluded that the decrease in serum DDT concentrations was due to lower levels of DDT in the fish and in the environment, rather than to a decrease in fish consumption (Hovinga et al. 1992).

A study of residents in Triana, Alabama, living downstream from a former DDT manufacturing facility revealed mean serum levels of total DDT of 76.2 ppb (Kreiss et al. 1981). This was several times higher than other reported levels. Kreiss et al. (1981) also found that serum DDT levels increased with increasing age. Residents living near a pesticide dump site in Aberdeen, North Carolina, known to contain high concentrations of DDT, have been shown to have age-adjusted mean levels of DDE in their blood of 4.05 ppb, which is higher than the mean value of 2.95 ppb obtained from residents of neighboring communities (Vine et al. 2000).

Mean levels of total equivalent of DDT, DDE, and DDD in maternal blood in pregnant women in India (20 samples) were found to be 25.3 ppb compared to levels in placental tissue of 22.2 ppb (Saxena et al. 1987). Similar levels (30.8 ppb) were seen in maternal blood in Brazilian women (Procianoy and Schvartsman 1981). Saxena et al. (1981, 1983) presented data on a limited number of samples of blood and placental tissues of women that aborted or delivered prematurely, which suggested that *p,p'*-DDE concentrations were elevated compared to control groups.

Adipose tissue from a subgroup of 40 workers engaged in spraying DDT for malaria control in Mexico contained the following median and maximum levels of DDT metabolites (µg/g): ΣDDT, 114.60, 665.56; *p,p'*-DDT, 46.96, 344.98; *o,p'*-DDT, 2.96, 29.74; *p,p'*-DDE, 64.96, 298.42; and *p,p'*-DDD, 0.62, 3.51 (Rivero-Rodriguez et al. 1997). Based on these measurements and a survey of the work habits of other workers, a geometric mean *p,p'*-DDE concentration of 67.41 µg/g was predicted for the population of 331 workers, 80% of whom were employed in the sanitation campaign for 20 years. Mean ΣDDT serum level in a group of 26 malaria control sprayers in Brazil was 76.9 µg/L and ranged from 7.5 to 473.5 µg/L, whereas 16 unexposed workers had mean serum levels of 16.1 µg/L (range: 5.1–32.9 µg/L) (Minelli and Ribeiro 1996). *p,p'*-DDT and *p,p'*-DDE serum levels in the exposed workers ranged from 1.6 to 62.9 and 5.9 to 405.9 µg/L, respectively.

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 3.8 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).

Children are exposed to DDT through their diet. Since the greatest dietary intake of DDT is from meat, fish, poultry, and dairy products, infants and young children for whom a substantial part of their food is milk may be exposed to DDT. According to the FDA study of 1986–1991, the mean daily intake of DDT and its metabolites is 0.0448, and 0.0438 µg/kg body weight/day for a 6–11-month-old infant and 2-year-old child, respectively (Gunderson 1995b). This is roughly four times the intake per unit body weight for an adult (see Table 6-3).

DDT and DDE selectively partition into fatty tissue and into human breast milk, which has a higher fat content than cow's milk. The concentration of DDT, or other hydrophobic pollutants, in milk is often expressed on a lipid basis (i.e., µg/g lipid rather than µg/mL milk) as it is a more accurate measure of DDT content due to the fluctuating fat content of the milk. Generally, these compounds are found in human breast milk in concentrations higher than in cow's milk or other infant foods. As a result, breast-fed infants may receive higher dietary exposure than those who are not breast-fed. If a woman has been exposed to high levels of DDT in the past, her milk may contain high levels of DDT, which would be transferred to her child. Women exposed to high levels of DDT would include Eskimos and Indian women in Arctic regions who eat traditional foods as well as women who eat large quantities of fish from lakes and rivers known to have high concentrations of DDT in fish, such as the Great Lakes and the Yakima River, Washington (Bard 1999; Kuhnlein et al. 1995; Marien and Laflamme 1995). Methods have been proposed for estimating breast milk lipid concentrations of DDT from a mother's daily intake (Marien and Laflamme 1995). Mean levels of *p,p'*-DDT in human breast milk in pooled milk from the

Mothers' Milk Center in Stockholm steadily declined from 0.71 µg/g lipid in 1972 to 0.36, 0.18, and 0.061 µg/g lipid in 1976, 1980, and 1984–1985, respectively (Norén 1988). Mean levels of *p,p'*-DDE for these years were 2.42, 1.53, 0.99, and 0.50 µg/g lipid, respectively. Between 1967 and 1985, the levels of *p,p'*-DDE and *p,p'*-DDT in human milk in Sweden declined by 75 and 95% (Norén 1993). In another study conducted between 1972 and 1992, this same group of investigators noted a similar decline in *p,p'*-DDE and *p,p'*-DDT concentrations in human milk in Sweden; the rates at which the concentration of these two compounds have been declining has also been progressively decreasing with time during this same period (Norén et al. 1996). The use of DDT was banned in Sweden in 1970. Mean (maximum) *p,p'*-DDT concentrations in 54 samples of mothers' milk from Hawaii (1979–1980) were 0.16 (0.52) µg/g lipid compared with 0.19 (1.7) µg/g lipid in 102 samples from the U.S. mainland (Takei et al. 1983). Mean (maximum) *p,p'*-DDE levels in Hawaiian and mainland samples were 2.0 (5.7) and 1.9 (11.0) µg/g lipid. A 1982 Canadian survey that included 210 samples of breast milk from across the country resulted in mean levels of *p,p'*-DDE, *p,p'*-DDT, *p,p'*-DDD, and *o,p'*-DDT in ng/g milk (ng/g milkfat) of 34 (911), 3 (80), 1 (27), and trace (12), respectively, down from 103, 33, 4, and 5 ng/g milk, respectively, obtained in a 1967 survey (Mes et al. 1986). The maximum *p,p'*-DDE, *p,p'*-DDT, *p,p'*-DDD, and *o,p'*-DDT levels in the 1982 survey were 5,500, 450, 113, and 58 ng/g milkfat. Levels of DDT in breast milk have shown a downward trend starting in about 1970. In 28 studies from the United States and Canada, average DDT levels in breast milk were about 4,000–5,000 ng/g lipid in the early 1970s, and then steadily declined by 1975. For 13 studies from 1975 on, there was an 11–21% reduction in mean ΣDDT levels per year. Another way of viewing this is that the mean breast milk level in the population is being reduced by one-half in 4.2–5.6 years. Similar reductions have been observed in Western European countries. While exposure of humans by eating fish from the Great Lakes has been a source of concern, in one study, Mes and Malcolm (1992) found that levels of DDE and DDT in breast milk were lower in women in the Great Lake's Basin than in women in the rest of Canada. Levels of DDE in cow's milk have similarly declined. The mean level of DDE in milk supplies in Southern Ontario, Canada declined from 96 ng/g lipid in 1970–1971 to 16 ng/g lipid in 1985–1986, indicating that the levels are being reduced by one-half in 5.8 years (Frank and Braun 1989). Since levels of DDT in food have been declining, exposure of children to DDT through their diet would be much less than in the past.

Children may be exposed to DDT by ingesting contaminated soil or dust, from dermal contact with the soil, or by inhaling in the dust and then swallowing it after mucociliary transport up out of the lungs. DDT is extremely persistent in soil and there are soils that still contain high levels of the insecticide. No reports have been found, however, concerning childhood exposures to DDT by ingesting dirt. DDT is strongly adsorbed to soil, especially when the organic content of the soil is high. No studies were found

as to how bioavailable DDT-adsorbed soil is when ingested. In addition, no information was found on the absorption of ingested DDT in any form in children. Children may also be exposed to DDT improperly stored at waste sites. A recent study indicated that old carpeting may contain high levels of DDT (Lewis et al. 1994). The DDT may have contaminated the carpet material or may have been tracked in from outside. Children may be exposed to this DDT while crawling around or playing on contaminated carpeting.

Since DDT partitions into lipids and is not readily metabolized, levels of DDT in adipose tissue increase with age. Levels of DDT and DDE in children aged 0–14 as reported in EPA's FY 1986 National Adipose Tissue Survey appear in Table 6-4 (Lordo et al. 1996).

6.7 POPULATIONS WITH POTENTIALLY HIGH EXPOSURES

Because of the ban on DDT use after 1972, fewer persons in the United States should be exposed to high levels of these compounds today than in the past. Only fish and marine mammal consumption in the Arctic appear to be significant dietary contributors to human exposure to DDT in the general population (Laden et al. 1999). A 1982 study by the Michigan Department of Public Health found that people eating large quantities of Great Lakes fish had significantly higher serum DDT levels compared to non-fish-eating controls. Furthermore, fish consumption was a major predictor of exposure. A follow-up study in 1989 found that serum DDT levels were primarily a reflection of historic exposures and previously established body burden rather than recent exposure (Hovinga et al. 1993). Other studies confirm these findings (Anderson et al. 1998; Hanrahan et al. 1999). The best predictors of serum DDE levels in frequent Great Lakes sport fish consumers were found to be age, years of eating sport caught fish, male gender, and body mass index (BMI), which respectively accounted for 20, 10, 9, and 9% of the variance (Hanrahan et al. 1998). In general, DDT-contaminated fish are caught by sport or subsistence fisherman and not purchased at the market (Laden et al. 1999). As the levels of DDT in Great Lakes fish decline, fish consumption is less likely to be a source of potentially high exposure. Because of the partitioning of DDT and DDE into fatty tissue and fluids, breast-fed infants are likely to receive doses in excess of those occurring from ingestion of cow's milk or other infant foods. Monitoring exposure of infants via breast milk has been extensive and provides evidence of the persistence of DDT and DDE in fatty tissues. The finding that old carpeting may contain high levels of DDT indicates that this may be an important, but unevaluated source of exposure, especially in small children crawling on the carpeting (Lewis et al. 1994). More details about children's exposures can be found in Section 6.6 Exposures of Children.

Workers involved with formulation, packaging, and application of DDT in the past would be expected to have been exposed to levels higher than those encountered in the environment. Persons who live near NPL sites containing DDT, DDE, or DDD might be exposed to higher levels than the general population since DDT has been detected in 441 of the 1,613 hazardous waste sites that have been proposed for inclusion on the EPA NPL (HazDat 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 DDT, DDE, and DDD 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 DDT, DDE, and DDD.

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 *p,p'*-DDT, DDE, and DDD are well described in the literature although there are some gaps in data for the *o,p'*-isomers (see Table D-1). The *p,p'*-isomers are those of primary environmental concerns and the data available are sufficient to allow estimation of the environmental fate of DDT, DDE, and DDD.

Production, Import/Export, Use, Release, and Disposal. Since the banning of DDT in the early 1970s in the United States, there has been little information published on the production of DDT. DDT is no long produced in the United States or in most countries in the world. The most recent information indicates that it is produced in at least two countries, and is used in some underdeveloped countries for vector control. However, data would be useful on the production and use of DDT world wide. This type of information is important for estimating the potential for environmental releases from

various uses, as well as estimating the potential environmental burden. In turn, this would provide a basis for estimating potential exposure and public health risk.

Disposal information is equally important for determining environmental burden and areas where environmental exposure may be high. Although disposal methods for DDT and its metabolites are reported to a limited extent, no current information on disposal sites and quantity disposed was located. Information on how the current users (e.g., hazardous waste clean-up crews) wash DDT equipment and dispose of the remaining waste would be helpful for estimating potential environmental and human exposure.

Environmental Fate. DDT, DDE, and DDD released to the environment may be transported from one medium to another by the processes of solubilization, adsorption, bioaccumulation, or volatilization. The transport of DDT, DDE, and DDD between environmental compartments has been predicted mostly from their physical and chemical properties. Volatilization and adsorption account for loss of DDT and its metabolites from surface water and soil. Monitoring studies indicate that DDT and its isomers and metabolites are extremely persistent in soil (EPA 1986a) and substantiate their predicted environmental fate. DDT, DDE, and DDD are highly lipid soluble. This, combined with their extremely long persistence, contributes to bioaccumulation of DDT and its metabolites in freshwater and marine life. Limited data were located on the soil degradation rates of DDT and its metabolites. Data are available for disappearance rates including losses due to transport processes. While adequate data are available on the time for the disappearance of 50% of the DDT initially applied to a variety of soils, there is abundant evidence that subsequent declines in DDT in soil occur at a much slower rate largely due to an aging process. More data on the biodegradation rates of DDT and its metabolites as well as how soil properties and aging affect these rates would be useful. Experimental information characterizing the environmental fate of DDT, DDE, and DDD, particularly on those properties that govern transport to air, would be helpful to further confirm their predicted environmental behavior and potential human exposure.

Bioavailability from Environmental Media. Limited information was located regarding the bioavailability of DDT, DDE, and DDD from environmental media. It has been shown that the bioavailability of DDT in soil declines with time (Alexander 1995, 1997; Robertson and Alexander 1998) and soil properties that influence the bioavailability of DDT and its toxicity to certain organisms have been studied (Peterson et al. 1971). More information regarding the aging process of DDT in soil and its affect on bioavailability would be helpful in identifying potential routes of human exposure. It is known that fish and some plants bioaccumulate these compounds and that those who consume these fish and

plants will incur some exposure to these compounds. However, because of universal body burdens of these compounds, the relative contribution of any particular medium, especially soil and sediment, is not clearly understood. Even if DDT, DDE, and DDD concentrations in various media are known, the difference between the exposure level and the absorbed dose is still unknown.

Food Chain Bioaccumulation. Information was located regarding food chain biomagnification of total DDT in the arctic marine food web (Hargrave et al. 1992). Fairly extensive monitoring of fish populations has been performed and a bioconcentration factor in fish is available. The steady-state BCF in rainbow trout was reported as 12,000, suggesting that bioconcentration in aquatic organisms is very high (Oliver and Niimi 1985). Although DDT has been detected in plants and vegetables, root uptake of DDT is considered low (Fuhremann and Lichtenstein 1980; Lichtenstein and Schultz 1980). A clearer understanding of the potential for bioaccumulation would aid in determining how levels in the environment affect the food chain and potentially impact human exposure levels. This type of information could be obtained by studying accumulation of these compounds in organisms from several trophic levels.

Exposure Levels in Environmental Media. Information on environmental levels of DDT, DDE, and DDD are abundant for the 1970s and 1980s (Blus et al. 1987; Carey et al. 1979b; Crockett et al. 1974; Ford and Hill 1990; Hargrave et al. 1992; Lichtenberg et al. 1970; Stanley et al. 1971). More recent information has been more limited in scope (Aigner et al. 1998; McConnell et al. 1998; Monosmith and Hermanson 1996). Continuation of data collection on environmental levels would contribute to the understanding of current worldwide concentrations and trends, especially in regions where DDT is currently used in vector control for malaria and as an agricultural pesticide.

Reliable monitoring data for the levels of DDT, DDE, and DDD in contaminated media at hazardous waste sites are needed so that the information obtained on levels of DDT, DDE, and DDD in the environment can be used in combination with the known body burden of DDT, DDE, and DDD to assess the potential risk of adverse health effects in populations living in the vicinity of hazardous waste sites.

Estimates of human intake have been limited to dietary intakes based on current market basket surveys (EPA 1980a; Gartrell et al. 1985, 1986a, 1986b; Gunderson 1995a). Additional information is needed relating to the levels in environmental media to which the general population is exposed, particularly at or near hazardous waste sites, and the subsequent development of health effects.

Exposure Levels in Humans. Data are available on levels of DDT and its metabolites in adipose tissue, blood, and milk (Hovinga et al. 1992; Lordo et al. 1996; Smith 1999). Recent monitoring data appear in Table 6-4.

Exposures of Children. More data are needed on the concentrations of DDT in breast milk of exposed women and on the DDT intake of breast-fed infants. In addition, the oral availability of DDT from soil and dust is lacking. Such data would allow for the estimation of the exposure of children to DDT from eating soil and dust.

Child health data needs relating to susceptibility are discussed in Section 3.13.2, Identification of Data Needs: Children's Susceptibility.

Exposure Registries. No exposure registries for DDT, DDE, or DDD were located. The ATSDR Division of Health Studies will consider these chemicals when primary chemical selection is made for future subregistries for the National Exposure Registry. The information that is amassed in the National Exposure Registry facilitates the epidemiological research needed to assess adverse health outcomes that may be related to exposure to these substances.

6.8.2 Ongoing Studies

The bioavailability of DDT, DDE, and DDD in soils to earthworms and the estimation of bioavailability by chemical extraction methods is being investigated by Professor Martin Alexander and coworkers at Cornell University. These studies will aid in the understanding of how the aging process of chemicals affects bioavailability.

The remediation of soil and sediments contaminated with select organic contaminants, like DDT and its metabolites, is being investigated by several groups. A study of the bioremediation of soil through the action of coryneform bacteria in metabolizing chlorinated hydrocarbon insecticides, as compared to faster-growing pseudomonad bacteria is being investigated by D.D. Focht and colleagues at the University of California at Riverside. This group is examining the substrate specificity of a biphenyl-A gene (bphA) in the metabolism of chlorinated hydrocarbon insecticides and are also looking at the role of surfactants in uptake and catabolism of chlorinated hydrocarbon pesticides. Laboratory investigations into the feasibility of remediation of sediments contaminated with select organic contaminants, like DDT,

are currently being undertaken by W.L. Mauck and colleagues at the Columbia Environmental Research Center in Columbia, Missouri.

An investigation of the phytoremediation of soils to remove persistent organics like *p,p*'-DDE is being conducted by J.C. White and colleagues at the Connecticutt Agriculture Experiment Station in New Haven, Connecticutt. In this research, the investigators plan to identify plants that are able to intercept and actively remove *p,p*'-DDE and other compounds from soils through the use of such techniques as chelating agents to open up soil and the addition of surfactants to mobilize recalcitrant compounds in soil. These investigators also propose to identify and test organisms in rhizosphere and nonrhizosphere zones in soil that effectively degrade pesticide-contaminants and then innoculate the most tolerant and effective degraders into rhizosphere of plants to stimulate the effectiveness of phytoremediation.

Exposures of persistent organochlorine compounds, like DDT, to wildlife is in the deep sea, in remote, pristine environments and in the Columbia River system are ongoing. D.C. Kadkoto and R.G. Zika at the University of Miami, and C.R. Smith at the University of Hawaii, are investigating the exposure of deep-sea whale-fall communities in the Santa Catalina Basin to persistent organic compounds, like DDT, through the measurement of these compounds in the skeletal remains of whales. J.E. Estes and colleagues at the Western Ecological Research Center in Santa Cruz, California, are monitoring endocrine-disrupting compounds in Adak Island, Alaska, measuring environmental concentrations of select compounds, like DDT, in species (e.g., bald eagles, fish, mussels, seabirds, otters) and aboitic media, gaining both temporal and spatial assessments of contamination. Monitoring of persistent compounds (dioxins, furans, and PCBs) in Columbia River system and the relationship of the presence of these compounds to the nesting success of Osprey is being investigated by C.J. Henny and colleagues at the Forest and Rangeland Ecosystem Science Center in Corvallis, Oregon.

A study of dietary human exposure to select contaminants, such as *p,p*'-DDE, is being conducted by D.L. Macintosh and colleagues in the Department of Environmental Health Sciences, University of Georgia, Athens, Georgia. Associations will be drawn between contaminant levels in biological fluids, such as blood, and urine, drawn from study participants to concentrations of these contaminants in environmental samples such as food products, air, water, and soil, with the purpose of identifying various exposure pathways with regard to total exposure. These investigators will also investigate temporal (e.g., time of year), spatial (e.g., region of country), and demographic (e.g., socioeconomic status) factors on the magnitude and sources of exposure.

An analytical methodology has been developed to quantitate pesticides in carpet and surface dust and is now being used to assess exposure of agricultural workers, and their families, or families in agricultural regions, to pesticides, with an emphasis on children's exposures to select pesticides, like DDT. A.T. Lemley, S.M. Snedeker and colleagues at Cornell University will use this information, in conjunction with information available in the literature, to assess cancer risk and to identify risk reduction procedures.

A number of research groups are investigating the levels of DDT and its metabolites in humans and the association of these levels with specific human health end points. R. Bhatia and colleagues at the Public Health Institute, Berkley, California, are examining DDT concentrations in the cord blood of a subset of 50 infants from the Children's Health and Development Studies cohort to determine if maternal levels of DDT have an effect on the incidence of male genital anomalies. Another group headed by S. Korrick at the School of Public Health, Harvard University, is measuring DDE and other compounds in cord serum and/or breast milk in children/women living near a PCB-contaminated harbor (Superfund site) in Massachusetts to ascertain the effect that these compounds may have on childhood cognitive function. In the Oswego Newborn and Infant Development Project, T. Darvill and colleagues are tracking newborns of women who did or did not consume Great Lakes fish during pregnancy, analyzing cord blood and placental tissues to determine the effect persistent compounds, such as DDT, have on cognitive function in children. Breast cancer risk as a function of exposure to DDT and other xenoestrogens, as measured in blood samples, collected from women in Wisconsin is being investigated by P.L. Remington at the University of Wisconsin at Madison.