5. POTENTIAL FOR HUMAN EXPOSURE

5.1 OVERVIEW

HCCPD is an anthropogenic chemical that is released to the atmosphere primarily by emissions from industrial manufacturing and processing facilities and to a lesser extent from the use of products such as chlorinated cyclodiene pesticides that may contain small amounts of HCCPD. Treatment and disposal of HCCPD-containing wastes also contribute to environmental concentrations. HCCPD tends to volatilize readily to the atmosphere from other media and to adsorb strongly to soils and sediments. HCCPD reacts readily with hydroxyl and nitrate radicals and can be rapidly degraded by photolysis (EPA 1984b; Grosjean and Williams 1992). The atmospheric residence time for this chemical is less than one day (EPA 1984b; Grosjean and Williams 1992). Bioconcentration of HCCPD occurs to a very small extent (EPA 1989a; Spehar et al. 1979). Biomagnification through the food chain is unlikely to occur because HCCPD degrades rapidly by photolysis in less than 1 day (Butz et al. 1982; Chou et al. 1987; Podowski and Khan 1996; Wolfe et al. 1982) and by hydrolysis, half-life values of 5 days or less have been reported for aquatic environments (Podowski and Khan 1996; Wolfe et al. 1982). Photolysis and hydrolysis are the most important environmental fate processes, however, biodegradation also occurs in water, soil, and sediment.

Exposure of the general population, including children, to HCCPD is insignificant (EPA 199 1 a). Human exposure to HCCPD occurs primarily in occupational settings (Boogaard et al. 1993; Kominsky et al. 1980; Morse et al. 1979). Individuals in the general population who live in the vicinity of industrial facilities or hazardous waste sites where contamination has been detected may be exposed to potentially higher levels of HCCPD. A unique exposure route that may exist for young children involves hand-mouth activity if they play in or around HCCPD-contaminated soil or sediment in the vicinity of production facilities or hazardous waste sites. However, monitoring data are insufficient to estimate potential exposure levels in children or adults at these sites.

According to the Toxic Chemical Release Inventory, in 1996, a total of 66,678 pounds of HCCPD was released to the environment from 4 large processing facilities (TR196 1998). Of the total environmental releases, 7,966 pounds were released to air, 250 pounds were released via underground injection, 1,580 pounds were released to POTWs, and 56,882 pounds were transferred off-site for disposal.

5. POTENTIAL FOR HUMAN EXPOSURE

HCCPD has been identified in at least 31 of 1,467 hazardous waste sites that have been proposed for inclusion on the EPA National Priorities List (NPL) (HazDat 1998). However, the number of sites evaluated for HCCPD is not known. The frequency of these sites within the United States can be seen in Figure 5-l. Of these sites, all 31 are located in the contiguous United States.

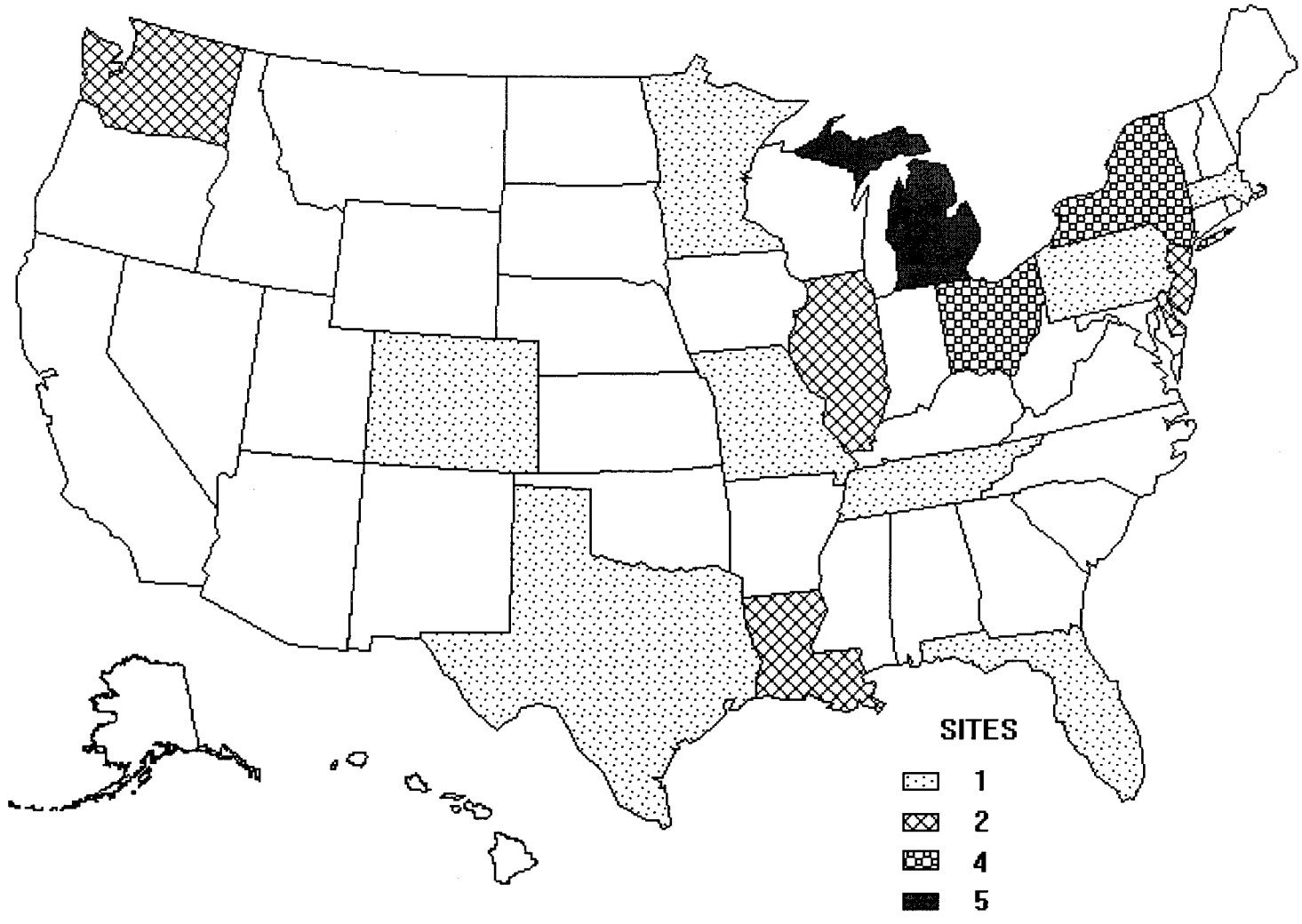
5.2 RELEASES TO THE ENVIRONMENT

According to the Toxic Chemical Release Inventory, in 1996, a total of 66,678 pounds of HCCPD was released to the environment from 4 large processing facilities (TRI96 1998). Table 5- 1 lists amounts released from these facilities. Of the total environmental releases, 7,966 pounds were released to air, 250 pounds were released via underground injection, 1,580 pounds were released to publicly owned treatment works (POTWs), and 56,882 pounds were transferred off-site for disposal. The TRI data should be used with caution because only certain types of facilities are required to report. This is not a exhaustive list. Manufacturing and processing facilities are required to report information to the Toxics Release Inventory only if they employ 10 or more full-time employees; if their facility is classified under Standard Industrial Classification (SIC) codes 20 through 39; and if their facility produces, imports, or processes 25,000 or more pounds of any TRI chemical or otherwise uses more than 10,000 pounds of a TRI chemical in a calendar year (EPA 1997c).

5.2.1 Air

The major sources of HCCPD releases to the air are from its production, processing, and use as a chemical intermediate (EPA 1980a, 1984b, 199 la). Releases may also occur from waste water treatment facilities, hazardous waste sites, and from the application and disposal of products such as chlorinated cyclodiene pesticides that may contain small amounts of HCCPD (Clark et al. 1982; Elia et al. 1983; EPA 1984b; Kominsky et al. 1980). In May 1977, HCCPD was detected at 56 ppb in air samples collected from a hazardous waste site in Montague, Michigan (EPA 1980a). An emission rate of 0.26 grams HCCPD per hour was reported at an abandoned hazardous waste site in Michigan (EPA 1984b). At this rate, emissions from this site would amount to about 5 pounds per year. In the United States, releases of HCCPD during 1978 were estimated to be approximately 60,000 pounds: 94% originated from manufacturing and 6% was due to its use (Anderson 1983). This compound has also been identified as a combustion product in emissions from a waste incinerator (Junk and Ford 1980).

Figure 5-1. Frequency of NPL Sites with HCCPD Contamination



Derived from HazDat 1998

Table 5-1. Releases to the Environment from Facilities That Manufacture or Process HCCPD

			Reported amounts released in pounds per year [a]						
STATE [b]	CITY	FACILITY	AIR [c]	WATER	LAND	UNDER-GROUND INJECTION	POTW TRANSFER	OFF-SITE WASTE TRANSFER	TOTAL ENVIRONMENT [d]
NY	NIAGARA FALLS	OCCIDENTAL CHEMICAL CORP.	920	0	0	0	4	5,010	5,934
OH	WEST ALEXANDRIA	MORTON INTL. INC.	63	0	0	0	0	1,130	1,193
TN	MEMPHIS	VELSICOL CHEMICAL CORP.	6,973	0	0	0	1,576	46,207	54,756
TX	BEAUMONT	BASF CORP.	10	0	0	250	0	4,535	4,795
		TOTALS	7,966	0	0	250	1,580	56,882	66,678

Source: TRI96 1998

[a] Data in TRI are maximum amounts released by each facility
[b] Post office state abbreviations used
[c] The sum of fugitive and stack releases are included in releases to air by a given facility
[d] The sum of all releases of the chemical to air, land, and water, and underground injection wells; and transfers off-site by a given facility

POTW = publicly owned treatment works

5. POTENTIAL FOR HUMAN EXPOSURE

Estimated releases of 7,966 pounds (3.6 metric tons) of HCCPD to the atmosphere from 4 domestic manufacturing and processing facilities in 1996 accounted for 12% of the estimated environmental releases (TR196 1998). These releases are summarized in Table 5-1. The TRI data should be used with caution because only certain types of facilities are required to report (EPA 1997c). This is not an exhaustive list. Manufacturing and processing facilities are required to report information to the Toxics Release Inventory only if they employ 10 or more full-time employees; if their facility is classified under Standard Industrial Classification (SIC) codes 20 through 39; and if their facility produces, imports, or processes 25,000 or more pounds of any TRI chemical or otherwise uses more than 10,000 pounds of a TRI chemical in a calendar year (EPA 1997c).

HCCPD has been identified in air samples collected at 2 of the 31 NPL hazardous waste sites where it was detected in some environmental media (HazDat 1998).

5.2.2 Water

Releases of HCCPD to water may occur during production, processing, and disposal of the chemical. In the past, concentrations of HCCPD in waste water from a production plant ranged from 0.156 to 18 mg/L (156-18,000 ppb) (EPA 1980a). HCCPD measurements were taken from the effluent stream of the Memphis North Sewage Treatment Plant from February to July 1982. Monthly average HCCPD concentrations in the effluent stream of this sewage treatment plant located near a production facility (Velsicol Chemical Corporation plant) ranged from 0.15 to 0.61 ppb μg/L), with a maximum concentration of 1.80 ppb (EPA 1984b). More recently, Nubbe et al. (1995) reported that a concentration of HCCPD around 100 mg/L (100,000 ppb) was in the range of concentrations that were typical of an aqueous HCCPD waste stream from a commercial production facility.

HCCPD was detectable in 0.9% of 1,228 effluent samples reported in the Storage and Retrieval (STORET) database maintained by EPA from 1980 to 1982 (Staples et al. 1985). The median concentration for all samples was less than 10 μg/L (ppb). In a recent study of chlorinated organic chemicals associated with industries along the Passaic River in New Jersey, Shear et al. (1996) reported that HCCPD was associated with the following industries: metal finishing, plastics, inorganic chemicals, electroplating, and steam electric power production. However, no quantitative information on the concentrations of HCCPD in the Passaic River or in industrial effluents were presented for this compound.

5. POTENTIAL FOR HUMAN EXPOSURE

In 1996, no HCCPD was reported discharged to water from the four manufacturing and processing facilities in the United States (TR196 1998). However, 1,580 pounds (0.7 metric tons) of HCCPD representing 2.4% of all environmental releases were transferred to POTWs, and a portion of these releases may eventually have been discharged to surface water (TR196 1998). These releases are summarized in Table 5- 1. The TRI data should be used with caution because only certain types of facilities are required to report (EPA 1997~). This is not an exhaustive list. Manufacturing and processing facilities are required to report information to the Toxics Release Inventory only if they employ 10 or more full-time employees; if their facility is classified under Standard Industrial Classification (SIC) codes 20 through 39; and if their facility produces, imports, or processes 25,000 or more pounds of any TRI chemical or otherwise uses more than 10,000 pounds of a TRI chemical in a calendar year (EPA 1997c).

In May 1977, HCCPD was also detected at 17 mg/L (17,000 ppb) in the aqueous discharge collected from a hazardous waste site in Montague, Michigan (EPA 1980a). HCCPD has been identified in surface water and groundwater samples collected at 7 and 15 of the 3 1 NPL hazardous waste sites, respectively, where it was detected in some environmental media (HazDat 1998).

5.2.3 Soil

HCCPD can be released to soil and sediment directly from manufacturing and processing facilities. HCCPD has been identified in the soil and river sediments downstream from a Virginia manufacturing plant, even after pesticide production at the plant was discontinued (EPA 1980a). Historically, HCCPD may have been released during application of several of the chlorinated cyclodiene pesticides. For example, chlordane has been reported to contain up to 1% HCCPD in the technical grade (Dorough and Ranieri 1984; Lu et al. 1975). Since the use of most of these chlorinated cyclodiene pesticides containing HCCPD as an impurity has been banned or restricted in the United States by the EPA, direct deposition on soil currently should be minimal with the possible exception of disposal at waste sites, accidental spills, and disposal by illegal methods (EPA 1984b). Deposition of volatilized HCCPD from air to soil is also not expected to be significant (see Section 5.3.1).

As shown in Table 5-1, in 1996, no HCCPD was reported discharged to soil from the four manufacturing and processing facilities in the United States (TR196 1998). However, 250 pounds (0.1 metric tons) accounting for 0.37% of the total environmental releases were released to soil via underground injection. This is not an exhaustive list. The TRI data should be used with caution because only certain types of

facilities are required to report (EPA 1997c). Manufacturing and processing facilities are required to report information to the Toxics Release Inventory only if they employ 10 or more full-time employees; if their facility is classified under Standard Industrial Classification (SIC) codes 20 through 39; and if their facility produces, imports, or processes 25,000 or more pounds of any TRI chemical or otherwise uses more than 10,000 pounds of a TRI chemical in a calendar year (EPA 1997c).

HCCPD has been identified in soil and sediment samples collected at 16 and 8 of the 31 NPL hazardous waste sites, respectively, where it was detected in some environmental media (HazDat 1998).

5.3 ENVIRONMENTAL FATE

In general, HCCPD is not persistent in air, water, or soil. Volatilization, photolysis, hydrolysis and biodegradation are key processes influencing the environmental fate and transformation of HCCPD (EPA 1984b).

5.3.1 Transport and Partitioning

Because HCCPD has a vapor pressure of 0.063 mm Hg at 25 °C (see Table 3-2), when released to the atmosphere, it will exist almost entirely in the vapor phase (Eisenreich et al. 1981). Detection of HCCPD in ambient air downwind (distance not reported) of a hazardous waste site indicates that atmospheric transport of HCCPD may occur (EPA 1984b). However, transported distance will be limited by the high reactivity of the chemical in the atmosphere (see Section 5.3.2.1). No data were located on deposition of HCCPD from air to water or soil, but deposition would probably be limited by the chemical's high reactivity. The relatively low water solubility of HCCPD suggests that there is little potential for washout in precipitation.

HCCPD released to water or soil may volatilize into the air or adsorb onto soil and sediment particles. Volatilization is most likely to occur from moving water bodies, with estimated removal of about 15% of the HCCPD in a turbid river compared with less than 5% removal from a lake or pond (Callahan et al. 1979). The volatilization rate from aquatic systems depends on specific conditions, including adsorption to sediments, pH of the medium, and air flow rate (Kilzer et al. 1979). In a laboratory study, 5.87% of ^{14}C-HCCPD per mL of evaporated water volatilized during the first hour (Kilzer et al. 1979). Data from the same study indicated that volatilization is much lower from soils. The volatilization rate of HCCPD

was expressed as the percentage of applied radioactivity per mL of evaporated water; for sand, loam, and humus soils, rates were 0.83, 0.33, and 0.14%, respectively, for the first hour and 0.23, 0.11, and 0.05%, respectively, for the second hour. Volatilization was highest from the sand and lowest from the humus. Volatilization was greater in soils with low organic content (Kilzer et al. 1979). In another study, the rate of volatilization of HCCPD from Maury silt loam soils was measured. Following the application of 100 mg of 14C-HCCPD to soil, the cumulative evaporation of HCCPD and its nonpolar metabolites (pentaand tetrachlorocyclopentadiene) on days 1, 2, 3, 5, 7, and 14 were 9.3, 10.2, 10.6, 10.8, 11.0 and 11.2%, respectively. The results indicate that HCCPD evaporation to air occurred mainly during the first day following application and was probably associated with the soil surface only (EPA 1984b).

HCCPD is readily adsorbed by soils and sediments (Chou and Griffin 1983; Chou et al. 198 1; Wolfe et al. 1982). Results of adsorption studies with nine different soil types indicate that adsorption capacity for HCCPD increases with increases in the total organic carbon (TOC) content of the soil (Chou and Griffin 1983; Chou et al. 1981). HCCPD was significantly more mobile in low TOC soils. Under the study conditions, HCCPD was fairly immobile in all soils when leached with tap water, landfill leachate, and caustic brine solution, but was highly mobile when the leaching agent was an organic solvent, such as acetone, methanol, or dioxane. Only 0.0005% of the HCCPD spiked into loamy sand was leached with tap water. Thus, the authors cautioned that to decrease the risk of migration of HCCPD from landfills into groundwater, wastes containing this chemical should not be disposed of in the same landfill location as organic solvents. The mechanisms of attenuation in soil materials were not reported. In another study, an average of about 68% of an applied dose of HCCPD was adsorbed to a flooded soil (EPA 1984b).

In aquatic environments, partitioning of HCCPD to sediments is likely. The soil adsorption properties of compounds such as HCCPD can be predicted from their soil organic carbon-water partition coefficients (K_{OC}). Kenaga (1980) examined the adsorption properties of 100 chemicals and concluded that compounds with K_{OC} values >l000 are tightly bound to soil components and are immobile in soils. Accordingly, the K_{OC} value is useful as an indicator of potential soil leachability of the chemical. Because K_{OC} values for HCCPD ranged from 4,786 to 12,023 (Mabey et al. 1982; Wolfe et al. 1982), the compound will tend to be tightly bound to soil and sediment particles. In a computer simulation of the fate of HCCPD in four different aquatic systems, the major portion of the chemical (86-99%) was predicted to be distributed to the sediments (Wolfe et al. 1982). The transport and partitioning data presented were consistent with the measured log K_{OW} of 5.04 (28 °C), and the experimental Henry's law constant of $2.7x10^{-2}$ atm-m^3/mol (Mabey et al. 1982; Wolfe et al. 1982).

5. POTENTIAL FOR HUMAN EXPOSURE

The reported log K_{OW} of 5.04 (Wolfe et al. 1982) indicates that bioconcentration of HCCPD could be substantial. However, data indicate that HCCPD does not bioconcentrate, bioaccumulate, or biomagnify in the food chain to a substantial degree (Lu et al. 1975; Podowski and Khan 1984; Podowski et al. 1991; Spehar et al. 1979; Wolfe et al. 1982), primarily because it is rapidly degraded in air, water, and soil and because it is metabolized in aquatic organisms. Podowski and Khan (1984) studied elimination, metabolism, and tissue distribution of HCCPD injected intraperitoneally into goldfish and concluded that the goldfish eliminate HCCPD both rapidly and linearly. Fish were injected with 39.6 μg of ^{14}C-HCCPD and analyzed 3 days later. Of the 97% of the labeled dose recovered, 19% was eliminated by the fish, 47% was extractable in an organic solvent (little of the labeled material could be identified as HCCPD, which indicated that biotransformation had occurred); 11% was water soluble metabolites; and 20% was unextractable. None of the metabolites were identified. A biphasic elimination of HCCPD was observed; rapid at first, followed by a slower phase. Based on a study of goldfish injected with ^{14}C -HCCPD, the elimination of HCCPD occurs in multiple stages, with a reported half-life in the organism of 7 days and predicted clearance of 90 and 95% of the chemical after 162 and 211 days, respectively (Podowski et al. 1991).

In a model ecosystem, a moderate potential for bioaccumulation of HCCPD was reported (Lu et al. 1975). The model ecosystem consisted of 50 sorghum (*Sorghum vulgare*) plants (3-4 inches tall) in the terrestrial portion; 10 snails (*Physa sp.*), 30 water fleas (*Daphnia magna*), filamentous green algae *(Oedogonium cardiacum)* and a plankton culture were added to the aquatic portion. The sorghum plants were treated topically with 5 mg of ^{14}C-HCCPD in acetone to simulate a terrestrial application of 1 lb/acre (1.1 kg/ha). Ten early-fifth-instar caterpillar larvae *(Estigmene acrea*) were placed on the plants. The insects consumed most of the treated plant surface within 3-4 days. The feces, leaf grass, and the larvae themselves contaminated the moist sand, permitting distribution of the labeled metabolites by water throughout the ecosystem. After 26 days, 300 mosquito larvae *(Culex pipiens quinquefasciatus)* were added to the ecosystem, and on day 30, three mosquitofish *(Gambusia affinis)* were added. The experiment was terminated after 33 days, and the various parameters were analyzed. The ecological magnification (EM) values reported were 340 for algae, 929 for snails, 1,634 for mosquitoes, and 448 for fish. However, biomagnification of HCCPD from algae to snails was 4.8, and from mosquito larvae to fish was 0.48, both of which were not substantial.

A measured bioconcentration factor (BCF) of less than 11 was reported in fathead minnows (Spehar et al. 1979). After adjustment of the BCF for lipid content, the weighted average BCF for the edible portion of

freshwater and estuarine aquatic organisms was calculated and found to be 4.34 (EPA 1980a). The available data on bioconcentration are currently under review by EPA, but this value will be used until the review is completed (EPA 1989a).

5.3.2 Transformation and Degradation

HCCPD is not persistent in air, water or soil. Photolysis, hydrolysis, and biodegradation are key processes influencing the environmental transformation and degradation of HCCPD (EPA 1984b).

5.3.2.1 Air

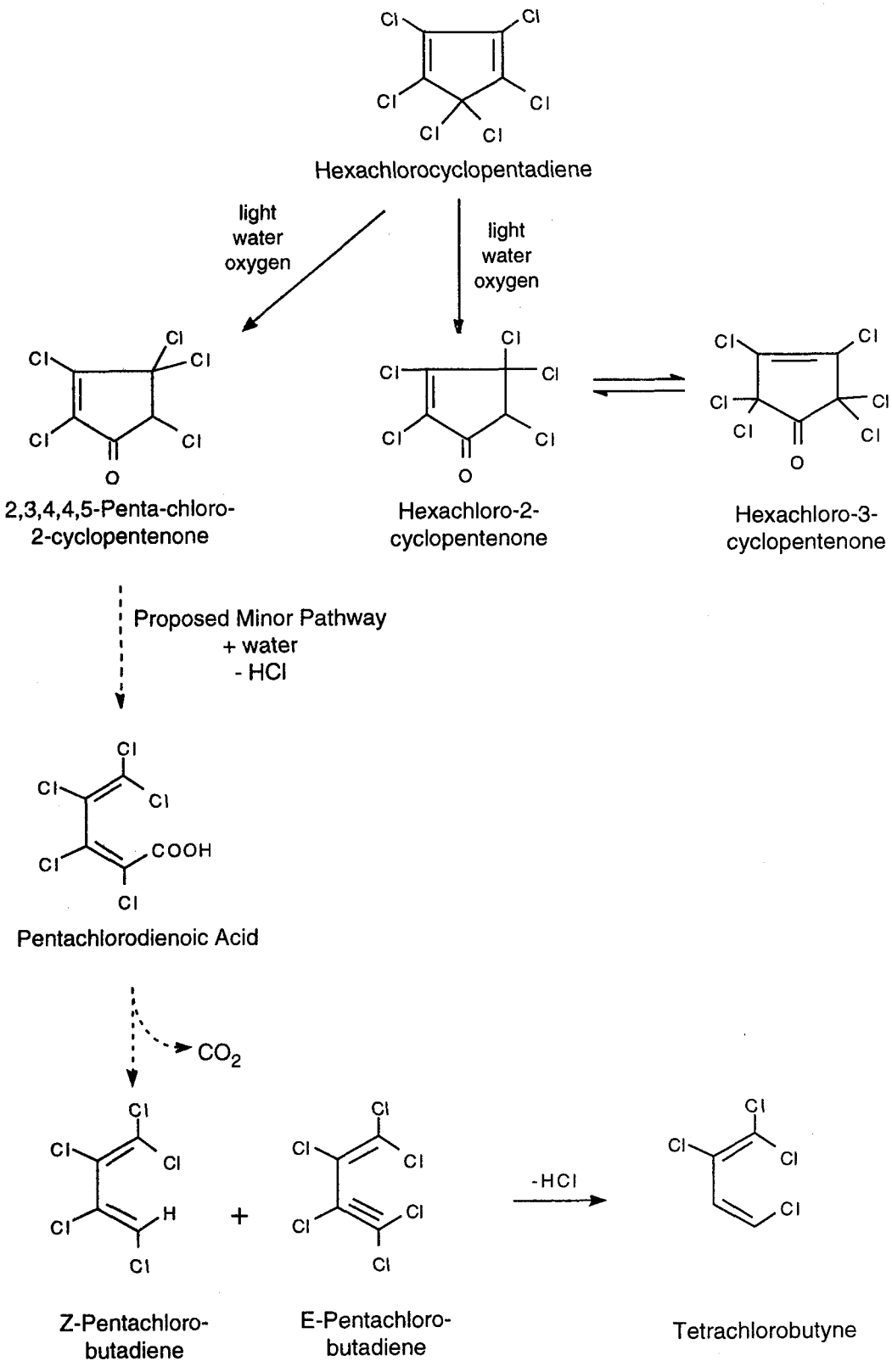
Although no measured values for HCCPD reactions in air were located, the chemical is expected to react rapidly with hydroxyl and nitrate radicals and ozone, and to be degraded rapidly by photolysis. Estimates of reaction rates with hydroxyl and nitrate radicals and ozone are available. Based on estimated reaction rates of the chemical with hydroxyl radicals and ozone of $59x10^{-12}$ and $8x10^{-18}$ cm^3 $molecule^{-1}$ sec^{-1}, respectively, the tropospheric residence time was estimated to be 5 hours (Cuppitt 1980). Recent estimates of the reaction rates with hydroxyl and nitrate radicals are $9.0x10^{-11}$ cm^3 $molecule^{-1}$ sec^{-1} and $2.14x10^{-12}$, cm^3 $molecule^{-1}$ sec^{-1}, respectively, based on structure-activity relationships (Grosjean and Williams 1992). Based on the estimated reaction rate and an atmospheric concentration of 10^6 OH molecule/cm3, the halflife of HCCPD in air is estimated to be less than 1 day (Grosjean and Williams 1992).

Since HCCPD is known to photolyze rapidly (half-life ~10 minutes) in water (Atallah et al. 1981; Butz et al. 1982; Wolfe et al. 1982), atmospheric photolysis is also expected (EPA 1984b). In addition, HCCPD absorbs light in the solar spectral region (EPA 1984b). However, no estimate of the reaction rate for atmospheric photolysis was located.

5.3.2.2 Water

Degradative processes for removal of HCCPD from water include photolysis, hydrolysis, and biodegradation. In shallow or flowing waters, photolysis is the predominant fate process; in deeper waters hydrolysis and biodegradation may be more important environmental fate processes (EPA 1984b).
The major pathways for transformation of HCCPD in water are shown in Figure 5-2.

Figure 5-2. Transformation of HCCPD



5. POTENTIAL FOR HUMAN EXPOSURE

HCCPD photolyses rapidly in water when exposed to sunlight or a mercury-vapor light source (Butz et al. 1982; Chou et al. 1987; Wolfe et al. 1982). The half-life values ranged from less than 2 minutes (Wolfe et al. 1982) to 4 minutes in natural sunlight (Chou et al. 1987) and less than 1.03 minutes when irradiated with a mercury-vapor light source (Butz et al. 1982). More recently, Podowski and Khan (1996) reported that HCCPD was photolyzed rapidly as a solution in acetone (half-life, <1 day) to apolar, polar, and hydrophilic products. The 15-day photolysis mixture contained no HCCPD. The reported photodegradation products included three primary products (2,3,4,4,5-pentachloro-2-cyclopentenone, hexachloro-2-cyclopentenone, and hexachloro-3-cyclopentenone) and three secondary products (pentachloro-cis-2,4-pentadienoic acid, Z- and E-pentachlorobutadiene, and tetrachlorobutyne (Chou et al. 1987). Dimerization of 2,3,4,4,5-pentachloro-2-cyclopentenone to form higher molecular weight compounds such as hexachloro-indenone may be a minor degradation pathway. This indicates that degradation of HCCPD in water does not always produce lower molecular weight, less toxic products (Butz et al. 1982; Chou et al. 1987). Pentachlorocyclopentenone has been reported to be the primary photolysis product by Butz et al. (1982) and the dimerization has been proposed as an artifact. However, more recent work by Podowski and Khan (1996) reject the idea that the dimer is an artifact as they could not produce the dimer compound by injecting pentachlorocyclopentenone into the GC at 190 °C. The authors also reported that the dimer, with a proposed molecular formula of C_9 C_{18} 0, was not mirex or chlordecone as judged by its GC behavior, indicating that HCCPD may not form at least the latter stable chemicals.

Hydrolysis of HCCPD in water occurs much more slowly than photolysis. In a study of the transformation pathways of HCCPD in aquatic systems, the reported average hydrolysis rate constant over a pH range of 3-10 was 1.5×10^{-6} sec^{-1} at 30 °C (Wolfe et al. 1982), which corresponds to a half-life of 5.35 days (EPA 1984b). More recently, Podowski and Khan (1996) reported that the time it took for HCCPD to reach 50, 10, and 5% of its initial concentration in water (7 ppb) was 4, 27, and 40 days, respectively.

Wolfe et al. (1982) found hydrolysis of HCCPD to be independent of pH over a range of pH 3-10. The rate of hydrolysis at neutral pH and 30 “C corresponded to a half-life (first order kinetics) of 5.45 days. The rate of hydrolysis was temperature dependent, with the half-life estimated to be 3.3, 1.7, and 0.6 days at 30, 40, and 50 °C, respectively. The addition of 0.5 molar NaCl did not affect the hydrolysis rate, suggesting that the rate constant was also applicable to marine environments. Addition of natural sediments sufficient to adsorb up to 92% of the HCCPD caused the rate constant to vary by less than a

factor of 2. Wolfe et al. (1982) therefore concluded that sorption to sediments would not significantly affect the rate of hydrolysis.

Some changes in hydrolysis rate did occur with changes in pH (EPA 1984b). The stability of ^{14}C-HCCPD in water at pH 3, 6, 9, and 12 at 25 and 45 °C was studied under dark conditions. HCCPD was relatively unstable at alkaline pH. At 25°C, the half-lives were 11.4, 11.4, 6 and 0.1 days at pH 3, 6, 9, and 12, respectively. At 45 °C, the half-lives were 9.2, 10.6 and 4.4 days at pH 3, 6, and 9, respectively. The above data indicate that at neutral pH, the hydrolysis half-life is from 3-l 1 days compared with a much more rapid photolytic half-life of < 10 minutes.

HCCPD also reacts with ozone in ozonated waters (Yao and Haag 1991). The measured rate constant for the consumption of the compound was 90 moles^{-1} second-‘, indicating that HCCPD will react significantly (greater than 10% conversion) with ozone at typical treatment conditions of 1 ppm ozone for 10 minutes (Yao and Haag 1991).

Biodegradation of HCCPD occurs in water under laboratory conditions. In a static laboratory culture, 100% of HCCPD was lost within 7 days from both 5 mg/L (ppm) and 10 mg/L (ppm) solutions (Tabak et al. 198 1). Volatilization was not reported to occur under the test conditions. However, in an evaluation of the potential for biodegradation as a spill-cleanup technique, HCCPD was reported not to be directly accessible to microorganisms in aquatic media (Thuma et al. 1983). The reported degradation of HCCPD ranged from 16 to 40% after 7 days, and from 35 to 60% after 14 days. Addition of methanol as a solubilizer increased the rate of biodegradation in 3 of 7 test cultures, with degradation up to 76%. Atallah et al. (1981) conducted an aqueous aerobic biodegradation study to determine whether and at what rate, HCCPD can be degraded to CO_2. The inoculum was a mixed acclimated culture containing secondary municipal waste effluent and several strains of *Pseudomonas putida.* HCCPD labeled with ^{14}C was the sole source of carbon in the study with the exception of trace levels of vitamins. Total removal of ^{14}C, primarily as volatile organics, was >80% in the first day in both uninoculated and inoculated media, although removal was slightly higher in inoculated media. $^{14}C0_2$ was released from both media, indicating that CO_2 was a product of hydrolysis as well as biodegradation. These studies show that HCCPD can be degraded in aquatic media under laboratory conditions. However, another study of the fate of HCCPD found biodegradation to be a relatively unimportant process in aquatic systems, based on the observation that there was no detectable difference in hydrolysis rates between sterile and nonsterile studies and measured numbers of microorganisms (Wolfe et al. 1982).

5.3.2.3 Sediment and Soil

Degradation of HCCPD occurs via both abiotic and biotic degradation processes in both sterile and nonsterile soil systems. Limited information shows that degradation rates are faster under nonsterile aerobic and anaerobic conditions, indicating that biodegradation has a role in the degradation process (EPA 1984b; Tabak et al. 1981). Potential exists for significant losses via photolysis of HCCPD on soil surfaces, while in moist soils hydrolysis may also occur. The relative importance of each process is difficult to assess and is dependent on site specific physical, chemical, and biological conditions (EPA 1984b).

The metabolism of HCCPD by soil microbial populations is an important process in its environmental degradation. Soil degradation is rapid under nonsterile aerobic and anaerobic conditions. In one study, several types of treatments and soil pHs were used to determine if the biodegradation of HCCPD in Maury loam soil was biologically and/or chemically mediated (EPA 1984b). Soils were incubated in glass flasks covered with perforated aluminum foil and maintained in the laboratory, presumably exposed to ambient lighting. When ^{14}C-HCCPD was applied to nonsterile soil at 1 ppm, only 6% was recovered as nonpolar material (either HCCPD or nonpolar degradation products) 7 days after treatment, and 72% was polar and unextractable material. By comparison, in autoclaved soil (control), 36% of the applied dose was recovered as nonpolar material and only 33% was recovered as polar and unextractable material. The degradation of HCCPD under anaerobic (flooded) conditions occurred at a slightly faster rate than under aerobic conditions (EPA 1984b). However, no single, flooded control was used to determine the effects of hydrolysis which could have accounted for the observed difference in this treatment.

A study was undertaken to examine the feasibility of using selected pure cultures of organisms (strains not identified) to biodegrade spills of hazardous chemical including HCCPD. Twenty-three of the test strains were found to remove 2 to 76% of the HCCPD from the aqueous culture medium within 14 days and 7 of the 23 strains degraded more than 33% of the HCCPD in 14 days (EPA 1984b).

5. POTENTIAL FOR HUMAN EXPOSURE

5.4 LEVELS MONITORED OR ESTIMATED IN THE ENVIRONMENT

Reliable evaluation of human exposure to HCCPD depends in part on the reliability of supporting analytical data from environmental samples and biological specimens. Concentrations of HCCPD in unpolluted atmospheres and in pristine surface waters are often so low as to be near the limits of current analytical methods. In reviewing data on HCCPD levels monitored in the environment, it should also be noted that the amount of chemical identified analytically is not necessarily equivalent to the amount that is bioavailable. The analytical methods available for monitoring HCCPD in a variety of environmental media are detailed in Chapter 6 (Analytical Methods).

5.4.1 Air

Because HCCPD is readily degraded in the atmosphere (see Section 5.3.2. l), it is not expected to be detected frequently in ambient outdoor air, and data from few monitoring studies were located. HCCPD was detected in indoor air at levels ranging from 0.06 to 0.10 pg/m3 (0.0053-0.0089 ppb) in 3 of 5 Tennessee homes where a contaminated groundwater supply was used as a source of potable water. The groundwater supply was contaminated with leachate from a pesticide waste dump operated by the Velsicol Corporation near Memphis, Tennessee (Clark et al. 1982).

HCCPD was also detected in air samples collected in June 1978 at the Memphis North Treatment Plant, the POTW that handled wastewater from the pesticide manufacturer (Clark et al. 1982; Elia et al. 1983). Concentrations of HCCPD ranged from 0.03 to 39 $\mu g/m^3$ (Elia et al. 1983). HCCPD also was detected in ambient outdoor air near a hazardous waste site at concentrations ranging from 0.032 to 0.053 ppb (0.36-0.59 $\mu g/m^3$) (EPA 1984b).

In May 1977, HCCPD was detected at 56 ppb in air samples collected from a hazardous waste site in Montague, Michigan (EPA 1980a). An emission rate of 0.26 grams HCCPD per hour was reported at an abandoned hazardous waste site in Michigan (EPA 1984b). HCCPD has been identified in air samples collected at 2 of the 3 1 NPL hazardous waste sites where it was detected in some environmental media (HazDat 1998).

5. POTENTIAL FOR HUMAN EXPOSURE

5.4.2 Water

HCCPD is rarely found in drinking water, surface water, or groundwater. HCCPD was tentatively identified in 1 of 16 samples of Philadelphia drinking water samplgd during 1976 (Suffet et al. 1980). HCCPD was also detected at a median concentration of 0.05 μg/L (ppb) (range, trace to 2.2 ug/L) in private wells used for drinking water by residents in the Toone-Teague area of Hardeman County, Tennessee (Clark et al. 1982). The source of the well water contamination was leachate from a pesticide waste dump site. HCCPD was detected in treated drinking water samples in Ottawa, Canada, at concentrations ranging from 57 to 110 ng/L (0.057-0.11 ppb), but was not detected (<50 ng/L [<050 ppb]) in raw water (Benoit and Williams 1981). Chlorination of the drinking water may be the source of the HCCPD, since the chemical reportedly formed during chlorination of a humic acid solution (Meier et al. 1985).

Data reported in the STORET database indicate that the chemical was detectable in only 0.1% of 854 surface water samples (Staples et al. 1985). The median concentration for all samples was less than 10 μg/L (10 ppb). HCCPD was also detected in Lake Ontario water, but not in water samples from Lakes Erie, Michigan, or Superior (Great Lakes Water Quality Board 1983).

In a recent study of chlorinated organic chemicals associated with industries along the Passaic River in New Jersey, Shear et al. (1996) reported that HCCPD was associated with metal finishing, plastics, inorganic chemicals, electroplating, and steam electric power production facilities. However, no quantitative information and concentrations of HCCPD in the Passaic River were presented for this compound.

HCCPD was detected in leachate from a hazardous waste site (Hauser and Bromberg 1982). HCCPD also has been identified in surface water and groundwater samples collected at 7 and 15 of the 31 NPL hazardous waste sites, respectively, where it was detected in some environmental media (HazDat 1998).

5.4.3 Sediment and Soil

No data were located documenting HCCPD concentrations in soil or sediments. HCCPD was not detectable in any of 344 sediment analyses reported in the STORET database (Staples et al. 1985). The median detection limit was 500 μg/kg (ppb).

5. POTENTIAL FOR HUMAN EXPOSURE

HCCPD has been identified in soil and sediment samples collected at 16 and 8 of the 3 1 NPL hazardous waste sites, respectively, where it was detected in some environmental media (HazDat 1998).

5.4.4 Other Environmental Media

HCCPD was qualitatively detected in fish samples collected from waters near one industrial source (a pesticide manufacturing plant in Michigan (Spehar et al. 1977). However, the chemical was not detected in any of the 116 fish samples reported in the STORET database (Staples et al. 1985), nor was it detected in fish samples from waters near other industrial sources (EPA 1984b) or from 14 Lake Michigan tributaries (Camanzo et al. 1987). HCCPD was qualitatively identified in fish collected from the Great Lakes and major watersheds to the Great Lakes during 1979 in 28 whole fish composite samples (7% positive detections; however, no detection limits were reported) (Kuehl and Leonard 1983). No recent information was located on detection of HCCPD in any commercially available fish or shellfish species, and no information was located on detection of HCCPD in raw or prepared foods.

In a study of the semiconductor industry, Bauer et al. (1995) reported detecting HCCPD at a concentration of 1.26 mg/kg (ppm) in waste oil samples collected from vacuum pump oils contaminated from aluminum plasma etching processes.

5.5 GENERAL POPULATION AND OCCUPATIONAL EXPOSURE

Exposure of the general population to HCCPD is extremely low (EPA 1991a). The chemical has not been frequently detected in any environmental medium. However, this may be due to analytical difficulties (see Section 6.2). Ambient air is the most likely source of HCCPD for exposed individuals in the general population. Nevertheless, due to the high reactivity of HCCPD, it is unlikely to remain in air or in other environmental media for extended periods.

Occupational exposure to HCCPD is mainly by inhalation, but dermal exposure may also occur. NIOSH estimated that 1,427 workers were potentially exposed to HCCPD in 1980, and the Velsicol Chemical Company of Memphis, Tennessee, estimated that about 157 employees are potentially exposed to HCCPD at their facility (EPA 1991a). Monitoring data indicate that workplace air concentrations of HCCPD ranged from 0.001 to 2.0337 ppm (0.0113-22.98 mg/m^3 check) in 1982 at various locations in industrial facilities producing or using HCCPD (EPA 1984b). The &hour time-weighted average (TWA)

concentrations ranged from 0.0003 to 0.035 ppm (0.0034-0.396 mg/m^3 check). In those areas of the facility where exposure to HCCPD is possible, respirators are required and are worn (EPA 1984b).

Exposure to HCCPD has been determined for operators employed in a chemical plant producing chlorinated hydrocarbons, including HCCPD, for an average of 8.2 years (range, 0.5-23 years) (Boogaard et al. 1993). Exposures, calculated as mean airborne concentrations over an &hour time-weighted average, occasionally exceeded 0.11 mg/m^3 and were higher during maintenance stops than during routine operation of the plant.

The Occupational Safety and Health Administration (OSHA) has not set a Permissible Exposure Limit (PEL) for HCCPD in the workplace (OSHA 1974). The American Conference of Governmental Industrial Hygienists (ACGIH) recommends a threshold limit value/TWA (TLV/TWA) of 0.1 mg/m^3 (0.01 ppm) for occupational exposures (ACGIH 1998). The recommended exposure limit for occupational exposure (TWA) set by the National Institute for Occupational Safety and Health (NIOSH) is 0.1 mg/m^3 (0.01 ppm) based on a 10-hour average workday (NIOSH 1997).

5.6 EXPOSURES OF CHILDREN

This section focuses on exposures from conception to maturity at 18 years in humans and briefly considers potential pre-conception exposure to germ cells. Differences from adults in susceptibility to hazardous substances are discussed in Section 2.6, 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, and 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; they put things in their mouths; they may ingest inappropriate things such as dirt or paint chips; they spend more time outdoors. Children also are closer to the ground, and they do not have the judgement of adults in avoiding hazards (NRC 1993).

From the preceding sections of the profile, it is apparent that information on the exposure of children to HCCPD is non-existent.

5. POTENTIAL FOR HUMAN EXPOSURE

No measurements of HCCPD or its metabolite levels in amniotic fluid, meconium, cord blood, or neonatal blood that would indicate prenatal exposure have been made. HCCPD is a lipophilic compound, so there should be no significant barrier to its crossing the placenta. In addition, no information was located on measurements of HCCPD in breast milk that might result in post-natal exposure of an infant. Because HCCPD is a lipophilic molecule, its passage into breast milk is also possible and could result in exposure of breast-fed infants.

HCCPD is currently found as only a small component of two pesticides (endosulfan and Pentac), and neither of these pesticides is used in the home. There is no information on HCCPD concentrations in infant and toddler foods, baby formula, or prepared foods for infants and children.

Children may receive higher doses of pesticides such as HCCPD from dermal exposures if they play on soil in contaminated areas such as a production facility or hazardous waste sites (Youngren et al. 1991). In addition, children may receive potentially higher oral doses from intentionally ingesting dirt containing HCCPD, from accidentally ingesting dirt by putting their hands in their mouths while playing in contaminated areas, or from putting contaminated toys or other objects in their mouths. Because HCCPD is relatively immobile in soil and soil is a temporary reservoir for HCCPD in the environment, these behaviors could be a source of HCCPD exposure for children. Although the bioavailability of HCCPD from the ingested soil particles via intestinal absorption in humans is not known, the gastrointestinal absorption of even free HCCPD is limited (see Section 2.3.1 for further details).

Occupational exposures appears to constitute the only documented source of human exposure to HCCPD (Boogaard et al. 1993; Kominsky et al. 1980; Morse et al. 1979). Occupational exposure to HCCPD is mainly by inhalation of contaminated air (Boogaard et al. 1993; EPA 1984b, 1991 a), so it is unlikely that workers would inadvertantly bring home HCCPD on their hair or clothing.

5.7 POPULATIONS WITH POTENTIALLY HIGH EXPOSURES

In addition to those individuals who are occupationally exposed to HCCPD during its production and processing (see Section 5.5), several other groups within the general population may receive potentially higher inhalation exposures. These groups include individuals working in waste water treatment plants or living or working near manufacturing and processing facilities or near hazardous waste sites where HCCPD has been detected in some environmental media. Air concentrations in the treatment areas of a waste water treatment plant were reported to range from 270 to 970 ppb (3,050-10,960 $\mu g/m^3$) (Morse et

al. 1979). However, these concentrations resulted from an incident in which large quantities of HCCPD were dumped into a municipal sewage system in Kentucky.

Individuals living in the vicinity of manufacturing or processing facilities or hazardous waste sites are most likely to be exposed to higher concentrations of HCCPD in the air; however, for workers at disposal sites, dermal contact with HCCPD may also occur during remediation activities. Individuals who consume groundwater from HCCPD-contaminated wells as their primary drinking water supply may also be exposed to higher concentrations of HCCPD than the general population.

5.8 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 HCCPD is available. Where adequate information is not available, ATSDR, in conjunction with the 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 HCCPD.

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.

5.8.1 Identification of Data Needs

Physical and Chemical Properties. The physical and chemical properties of HCCPD are sufficiently well characterized for most properties to allow estimation of its environmental fate (Amoore and Hautala 1983; EPA 1991a; Mabey et al. 1982; Ruth 1986; Verschueren 1983; Weast 1989; Wolfe et al. 1982). However, information on the autoignition temperature, flashpoint, flammability limits, and explosive limits would be helpful.

5. POTENTIAL FOR HUMAN EXPOSURE

Production, Import/Export, Use, Release, and Disposal. HCCPD is manufactured by one facility (SRI International 1997; USITC 1991). However, recent production volume and import/export information are not available. More current information on import and export volumes of the chemical ant on uses of this compound would be helpful, as would additional information on the current registered uses of the chlorinated cyclodiene pesticides in which HCCPD is present as an impurity. Current comprehensive information on disposal volumes and methods are also needed. This information would be useful in assessing current exposure of workers and the general population to HCCPD.

According to the Emergency Planning and Community Right-to-Know Act of 1986,42 U.S.C. Section 11023, industries are required to submit chemical release and off-site transfer information to the EPA. The Toxics Release Inventory (TRI), which contains this information for 1996, became available in May of 1998. This database will be updated yearly and should provide a list of industrial production facilities and emissions.

Environmental Fate. The environmental fate of HCCPD has been well described (Chou and Griffin 1983; Chou et al. 1981; EPA 1984b; Wolfe et al. 1982). The chemical is highly reactive and degrades readily in environmental media. However, further research on the metabolic, degradation, and reactive products would help in assessing the impact of HCCPD on the environment and humans. It is not likely that exposure of the general public is of concern. Nevertheless, because it appears to migrate at a higher rate in soil in the presence of other organic chemicals (Chou and Griffin 1983; Chou et al. 1981), additional studies might be useful to assess the potential for leaching of this chemical from soils at hazardous waste c sites into groundwater.

Bioavailability from Environmental Media. The occurrence of toxic effects in animals following inhalation, oral, and dermal exposures to HCCPD demonstrates that it is bioavailable from the media used in these studies (air, corn oil, peanut oil, and Ultrasene) (Abdo et al. 1984; Treon et al. 1955). Bioavailability from the gastrointestinal tract appears to be limited, presumably due to binding of HCCPD to intestinal contents (El Dareer et al. 1983). No data were available concerning bioavailability of HCCPD from soil or sediments. Research on the nature of the HCCPD interactions with soils, sediments, and food materials would help in evaluating the risk posed by contamination at hazardous waste sites.

5. POTENTIAL FOR HUMAN EXPOSURE

Food Chain Bioaccumulation. Bioaccumulation and biomagnification of HCCPD are not expected to be substantial in the food chain, since the chemical is rapidly metabolized in aquatic organisms (Lu et al. 1975; Podowski and Khan 1984; Podowski et al. 1991; Spehar et al. 1979; Wolfe et al. 1982). HCCPD has only rarely been detected in edible aquatic species (Camanzo et al. 1987; EPA 1984b). On this basis, it does not appear that exposure of humans by this route is of concern, and further research in this area does not seem to be a high priority.

Exposure Levels in Environmental Media. HCCPD is infrequently detected in environmental media (EPA 1984b, 1991a; Staples et al. 1985). This may be due to difficulties in analysis (see Section 6.2) and/or its rapid degradation in air, water, and soil. Insufficient data are available to estimate exposure levels in drinking water, food, or air (EPA 199Oc), but exposure of the general population to HCCPD from these sources is not expected to be significant. However, exposure in the workplace and in the vicinity of hazardous waste sites may occur at levels that could be of concern. Additional monitoring data for various inhalation exposure situations would be useful for determining occupational exposure and exposures of individuals in the general population living near manufacturing and processing facilities or near hazardous waste sites where HCCPD has been detected.

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

Exposure Levels in Humans. Detection of HCCPD in urine of waste water treatment plant workers indicates occupational exposures to this chemical (Elia et al. 1983). However, since HCCPD is rapidly metabolized *in vivo*, it was not possible to directly associate urine levels with environmental concentrations. Additional information on exposure levels in humans would be helpful. This information is necessary for assessing the need to conduct health studies on these populations.

Exposures of Children. A unique exposure route that potentially exists for children involves intentional ingestion and hand-mouth activity associated with young children. Exposure may arise if these behaviors occur in children who play in or around contaminated soil or sediment (e.g., hazardous waste sites). HCCPD has been detected in both soil and sediment collected at some NPL hazardous waste sites (HazDat 1998). As soil is at least a short-term reservoir of HCCPD in the environment, additional studies

of the transfer of HCCPD via both oral and dermal routes of exposure in children are warranted as are studies of the bioavailability of the chemical from soil and sediment. Quantitative information is also needed to determine the concentrations of HCCPD in air associated with hazardous waste sites.

Current information on whether children are different in their weight-adjusted intake of HCCPD via inhalation, oral, or dermal exposures was not located. A study should be conducted to determine if there are any HCCPD residues in breast milk, especially in women residing in areas in proximity to production facilities and hazardous waste sites. This information would be useful in determining which exposure pathway is most important for children. Since exposure to HCCPD is likely to be negligible in members of the general population, exposure and body burden studies for children would be helpful especially in those children living in proximity to production facilities or hazardous waste sites.

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

5.8.2 Ongoing Studies

No additional information was located on current studies that would fill existing data needs for HCCPD (FEDRIP 1998).