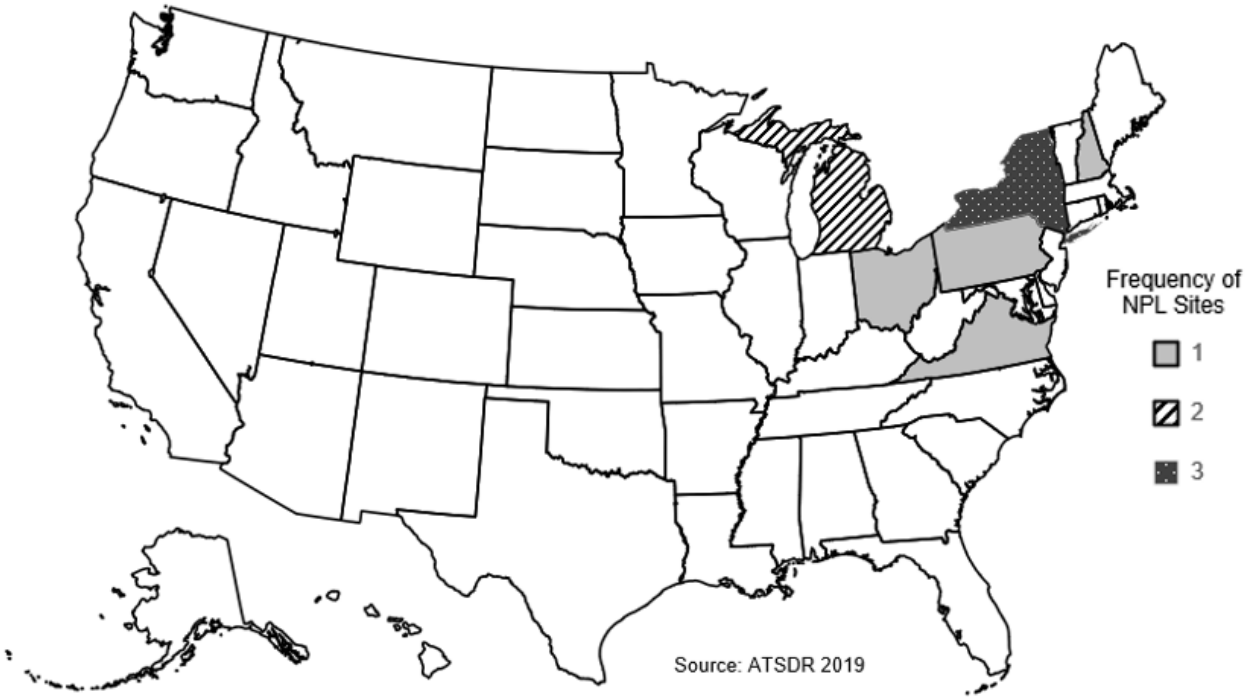
# CHAPTER 5. POTENTIAL FOR HUMAN EXPOSURE

## 5.1 OVERVIEW

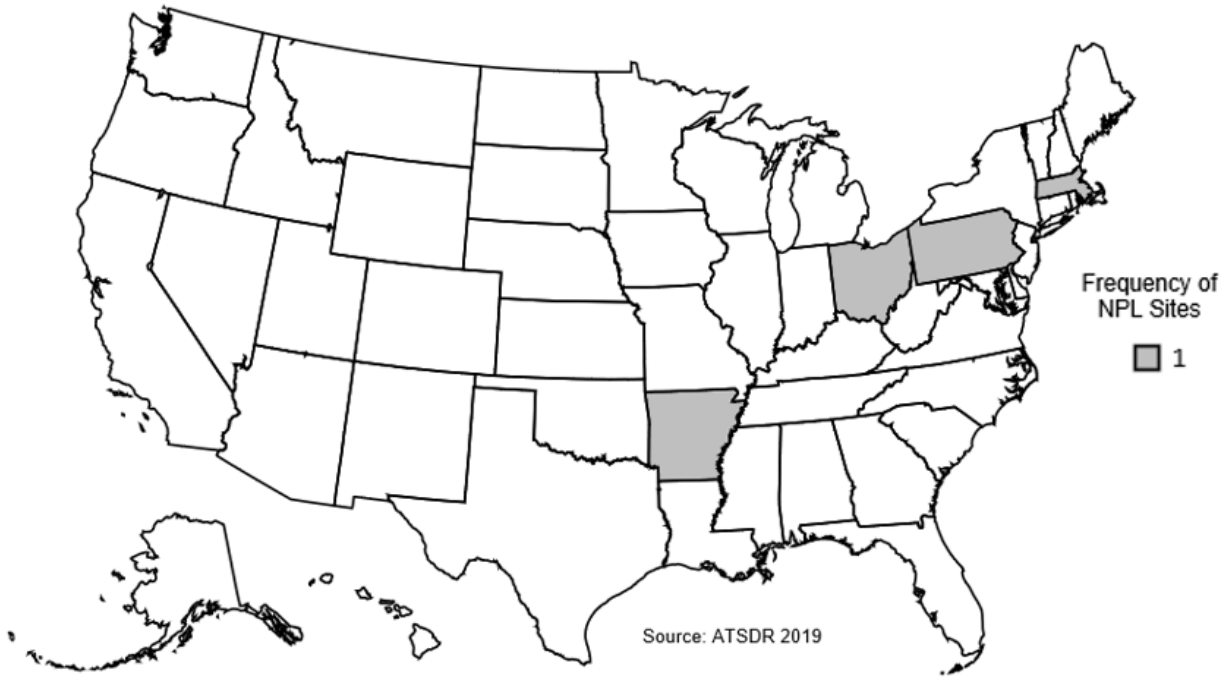
Mirex has been identified in at least 9 of the 1,867 hazardous waste sites that have been proposed for inclusion on the EPA National Priorities List (NPL) (ATSDR 2019). However, the number of sites in which mirex has been evaluated is not known. The number of sites in each state is shown in Figure 5-1.

**Figure 5-1. Number of NPL Sites with Mirex Contamination**




Chlordecone has been identified in at least 4 of the 1,867 hazardous waste sites that have been proposed for inclusion on the EPA National Priorities List (NPL) (ATSDR 2019). However, the number of sites in which chlordecone has been evaluated is not known. The number of sites in each state is shown in Figure 5-2.

**Figure 5-2. Number of NPL Sites with Chlordecone Contamination**




- The most likely source of potential exposure of the general population to mirex or chlordecone is from consumption of contaminated food sources, particularly in the eastern portion of the United States where mirex and chlordecone were most frequently used.
- People who live or work near hazardous waste sites where mirex and/or chlordecone may be stored could most likely be exposed from contaminated sediment or soil.
- Both mirex and chlordecone bind strongly to organic matter in water, sediment, and soil where they may persist for long periods of time.
- Both mirex and chlordecone are lipophilic and bioaccumulate and biomagnify in aquatic and terrestrial food chains.

As a result of human health concerns, production of mirex ceased in 1976, at which time industrial releases of this chemical to surface waters were also curtailed. However, releases from waste disposal sites continue to add mirex to the environment. Virtually all industrial releases of mirex were to surface waters, principally Lake Ontario via contamination of the Niagara and Oswego Rivers. About 75% of the mirex produced was used as a fire retardant additive, while 25% was used as a pesticide. As a pesticide, mirex was widely dispersed throughout the southern United States where it was used in the fire ant eradication program for over 10 years.

Adsorption and volatilization are the more important environmental fate processes for mirex, which strongly binds to organic matter in water, sediment, and soil. When bound to organic-rich soil, mirex is highly immobile; however, when adsorbed to particulate matter in water, it can be transported great

distances before partitioning out to sediment. Atmospheric transport of mirex has been reported based on its detection in remote areas without anthropogenic sources, although this is not a major source of mirex in the environment. Given the lipophilic nature of this compound (high octanol-water partition coefficient), mirex is both bioaccumulated and biomagnified in aquatic and terrestrial food chains.

Mirex is a very persistent compound in the environment and is highly resistant to both chemical and biological degradation. The primary process for the degradation of mirex is photolysis in water or on soil surfaces; photomirex is the major transformation product of photolysis. In soil or sediments, anaerobic biodegradation is also a major removal mechanism whereby mirex is slowly dechlorinated to the 10-monohydro derivative. Aerobic biodegradation in soil is a very slow and minor degradation process. Twelve years after the application of mirex to soil, 50% of the mirex and mirex-related compounds remained on the soil. Between 65 and 73% of the residues recovered were mirex and 3–6% were chlordecone, a transformation product (Carlson et al. 1976).

Mirex has been detected at low concentrations in ambient air (mean 0.35 $pg/m^3$) and rainfall samples (<0.5 ng/L) from polluted areas of the Great Lakes region. In addition, the compound has been detected in drinking water samples from the Great Lakes area of Ontario, Canada. Mirex has also been detected in groundwater samples from agricultural areas of New Jersey and South Carolina.

Mirex has been monitored in surface waters, particularly during the period that it was still being produced. Concentrations of mirex in Lake Ontario, the Niagara River, and the St. Lawrence River were in the ng/L (ppt) range. The highest concentrations of mirex, 1,700 µg/kg (ppb), were found in sediments in Lake Ontario where they accumulated after the deposition of particulate matter to which the mirex was bound. A dynamic mass balance for mirex in Lake Ontario and the Gulf of St. Lawrence estimated that approximately 2,700 kg (6,000 pounds) of mirex have entered Lake Ontario over the past 40 years, of which 550 kg (1,200 pounds) have been removed (exclusive of sedimentation and burial) mainly by transport on sediment particles via outflowing water and migrating biota contaminated with mirex.

The high bioconcentration factor (BCF) values (up to 15,000 for rainbow trout) observed for mirex indicate that this compound will be found in high concentrations in aquatic organisms that inhabit areas where the water and sediments are contaminated with mirex. Fish taken from Lake Ontario, the St. Lawrence River, and the southeastern United States (areas where mirex was manufactured or used as a pesticide) had the highest mirex levels. There were fish consumption advisories in effect in three states (New York, Pennsylvania, and Ohio) that were triggered by mirex contamination in fish. Waterfowl and

game animals have also been found to accumulate mirex in their tissues. Data on mirex residues in foods do not show a consistent trend with regard to contaminant levels or frequency of detection. Mirex was irregularly detected in Food and Drug Administration (FDA) Pesticide Residue Monitoring Studies since 1978, but has not been detected in the most recent FDA survey. Little information on the specific foods in which residues were found or levels detected was located.

General population exposure to mirex has been determined as a result of several monitoring studies (CDC 2019; EPA 1986b; Kutz et al. 1979; Stehr-Green 1989). Levels of mirex in most tissues are very low (at or near the detection limit). Examination of the 1982 National Adipose Tissue Survey failed to detect mirex in the adipose tissues of children <14 years old, although mirex residues were detected in adults. People who live in areas where mirex was manufactured or used have higher levels in their tissues. Women who live in these areas were found to have detectable levels of mirex in their milk that could be passed on to their infants. Since mirex is no longer manufactured, occupational exposure currently is limited to workers at waste disposal sites or those involved in remediation activities involving the clean-up and removal of contaminated soils or sediments.

Production of chlordecone ceased in 1975 as a result of human health concerns; at that time industrial releases of this chemical to surface waters via a municipal sewage system were curtailed. However, releases from waste disposal sites may continue to add chlordecone to the environment. Major releases of chlordecone occurred to the air, surface waters, and soil surrounding a major manufacturing site in Hopewell, Virginia. Releases from this plant ultimately contaminated the water, sediment, and biota of the James River, a tributary to the Chesapeake Bay.

Atmospheric transport of chlordecone particles was reported during production years based on results from high volume air samplers installed at the site and up to 15.6 miles away. Chlordecone is not expected to be subject to direct photodegradation in the atmosphere. Chlordecone is very persistent in the environment. Chlordecone, like mirex, will strongly bind to organic matter in water, sediment, and soil. When bound to organic-rich soil, chlordecone is highly immobile; however, when adsorbed to particulate matter in surface water, chlordecone can be transported great distances before partitioning out to sediment. Sediment in extensive areas of the James River served as a sink or reservoir for this compound. The primary process for the degradation of chlordecone in soil or sediments is anaerobic biodegradation. Based on the lipophilic nature of this compound (high octanol-water partition coefficient), chlordecone has a tendency to both bioaccumulate and biomagnify in aquatic food chains. BCF values >60,000 have been measured in Atlantic silversides, an estuarine fish species.

No information was found on atmospheric concentrations of chlordecone other than historic monitoring data from samples collected in the vicinity of the manufacturing site. Chlordecone has been monitored in surface waters, particularly during the period shortly before and after production was terminated. In 1977, chlordecone was detected in surface water samples from the James River at low concentrations (<10 ng/L [ppt]), although it was not detected in more recent monitoring studies. The highest concentrations of this compound are found in sediments, principally in the James River where it had accumulated after the deposition of particulate matter to which the chlordecone was bound. In 1978, chlordecone was detected in sediments from the James River below its production site at concentrations in the mg/kg (ppm) range.

The high BCF values observed for chlordecone (>60,000) indicate that the compound will be found in high concentrations in aquatic organisms that dwell in waters or sediments contaminated with chlordecone. Chlordecone has been detected in fish and shellfish from the James River, which empties into the Chesapeake Bay, at levels in the μg/g (ppm) range. There was a fish consumption advisory in effect for the lower 113 miles of the James River. Chlordecone residues were detected in foods analyzed in 1978–1982 and 1982–1986 as part of the FDA Pesticide Residue Monitoring Studies. Chlordecone was detected in one of 27,065 food samples analyzed by 10 state laboratories, but was not detected in the FDA Pesticide Residue Monitoring Studies in 1986–1991 or in the most recent (2017) study. No information on the specific foods in which residues were found or levels detected was located.

General population exposure to chlordecone has not been determined because this compound has not been monitored in any national program (CDC 2019; EPA 1986b; Kutz et al. 1979; Phillips and Birchard 1991; Stehr-Green 1989). Levels of chlordecone were detected in 9 of 298 samples of human milk collected from women in the southern United States. Residues were detected only in residents of areas that had been extensively treated with the pesticide mirex for fire ant control. People who lived in the area where chlordecone was manufactured had higher levels in their blood during production years. Women who lived in these areas could pass chlordecone in their milk to their nursing infants. Workers who manufactured chlordecone developed an occupationally-related illness. However, chlordecone is no longer manufactured, so occupational exposure is limited to workers at waste disposal sites or those involved in remediation activities involving the clean-up and removal of contaminated soils or sediments.

## 5.2 PRODUCTION, IMPORT/EXPORT, USE, AND DISPOSAL

### 5.2.1 Production

No information is available in the TRI database on facilities that manufacture or process mirex and chlordecone because these chemicals are not required to be reported under Section 313 of the Emergency Planning and Community Right-to-Know Act (Title III of the Superfund Amendments and Reauthorization Act of 1986) (EPA 2005).

Mirex is not known to occur in the environment as a natural product (IARC 1979; Waters et al. 1977a). Although it was originally synthesized in 1946, mirex was not commercially introduced in the United States until 1959, when it was produced by the Allied Chemical Company under the name GC-1283 for use in pesticide formulations and as an industrial fire retardant under the trade name Dechlorane® (EPA 1978a; IARC 1979; Waters et al. 1977a). Mirex was produced as a result of the dimerization of hexachlorocyclopentadiene in the presence of an aluminum chloride catalyst (IARC 1979; Sittig 1980).

The technical grade of mirex consisted of a white crystalline solid in two particle size ranges, 5–10 and 40–70 microns (IARC 1979). Technical-grade preparations of mirex contained 95.18% mirex, with 2.58 mg/kg chlordecone as a contaminant (EPA 1978b; WHO 1984). Several formulations of mirex have been prepared in the past for various pesticide uses. Some of the more commonly used formulations of mirex used as baits were made from corn cob grit impregnated with vegetable oil and various concentrations of mirex. Insect bait formulations for aerial or ground applications contained 0.3–0.5% mirex, and fire ant formulations contained 0.075–0.3% mirex (IARC 1979).

Mirex is no longer produced commercially in the United States. Hooker Chemical Company (Niagara Falls, New York) manufactured and processed mirex from 1957 to 1976 (Lewis and Makarewicz 1988). An estimated 3.3 million pounds (1.5x106 kg) of mirex were produced by Hooker Chemical Company between 1959 and 1975, with peak production occurring between 1963 and 1968 (EPA 1978b). About 25% of the mirex produced was used as a pesticide and the remaining 75% was used as an industrial fire retardant additive (EPA 1978b). Hooker Chemical Company reported purchasing 1.5 million pounds of mirex (680,400 kg) from Nease Chemical Company during this period. The Nease Chemical Company of State College, Pennsylvania, manufactured mirex from 1966 to 1974 (EPA 1978b). Allied Chemical Company also manufactured technical-grade mirex and mirex bait in Aberdeen, Mississippi (EPA 1978b), but Allied Chemical formally transferred all registrations on mirex, along with the right to manufacture

and sell mirex bait, to the Mississippi Department of Agriculture on May 7, 1976 (IARC 1979; Waters et al. 1977a, 1977b).

Chlordecone is not known to occur in the environment as a natural product (IARC 1979). Chlordecone has been produced by reacting hexachlorocyclopentadiene and sulfur trioxide under heat and pressure in the presence of antimony pentachloride as a catalyst. The reaction product is hydrolyzed with aqueous alkali, neutralized with acid; chlordecone is recovered via centrifugation or filtration and hot air drying (Epstein 1978). Chlordecone was produced in 1951, patented in 1952, and introduced commercially in the United States by Allied Chemical in 1958 under the trade names Kepone® and GC-1189 (Epstein 1978; Huff and Gerstner 1978). The technical grade of chlordecone, which typically contained 94.5% chlordecone, was available in the United States until 1976 (IARC 1979). Chlordecone was also found to be present in technical-grade mirex at concentrations of up to 2.58 mg/kg and in mirex bait formulations at concentrations of up to 0.25 mg/kg (EPA 1978b; IARC 1979). Approximately 55 different commercial formulations of chlordecone have been prepared since its introduction in 1958 (Epstein 1978). The major form of chlordecone, which was used as a pesticide on food products, was a wettable powder (50% chlordecone) (Epstein 1978). Formulations of chlordecone commonly used for nonfood products were in the form of granules and dusts containing 5 or 10% active ingredient (Epstein 1978). Other formulations of chlordecone contained the following percentages of active ingredient: 0.125% (used in the United States in ant and roach traps), 5% (exported for banana and potato dusting), 25% (used in the United States in ant and roach bait), 50% (used to control mole crickets in Florida), and 90% (exported to Europe for conversion to kelevan for use on Colorado potato beetles in eastern European countries) (Epstein 1978).

Chlordecone is no longer produced commercially in the United States. Between 1951 and 1975, approximately 3.6 million pounds (1.6 million kg) of chlordecone were produced in the United States (Epstein 1978). During this period, Allied Chemical Company produced approximately 1.8 million pounds (816,500 kg) of chlordecone at plants in Claymont, Delaware; Marcus Hook, Pennsylvania; and Hopewell, Virginia. In 1974, because of increasing demand for chlordecone and a need to use their facility in Hopewell, Virginia, for other purposes, Allied Chemical transferred its chlordecone manufacturing to Life Sciences Products Company (EPA 1978b). Life Sciences Products produced an estimated 1.7 million pounds (771,000 kg) of chlordecone from November 1974 through July 1975 in Hopewell, Virginia (Epstein 1978). Hooker Chemical Company also produced approximately 49,680 (22,500 kg) pounds of chlordecone in the period from 1965 to 1967 at a plant at Niagara Falls,

New York. Nease Chemical Company produced approximately 65,780 pounds (30,000 kg) of chlordecone between 1959 and 1966 at a plant in State College, Pennsylvania (Epstein 1978).

### 5.2.2 Import/Export

No current data are available regarding import volumes of mirex. Mirex has reportedly been imported to the United States from Brazil, but data on the amounts of mirex imported are not available (DHHS 1991; IARC 1979).

No current data are available regarding import volumes of chlordecone.

Technical mirex and technical chlordecone are not exported since these substances are no longer produced in the United States.

Over 90% of the mirex produced from the 1950s until 1975 was exported to Latin America, Europe, and Africa (Sterret and Boss 1977). No other historic data regarding the export of mirex were located.

Diluted technical-grade chlordecone (80% active ingredient) was exported to Europe, particularly Germany, in great quantities from 1951 to 1975 by the Allied Chemical Company (Epstein 1978) where the diluted technical product was converted to an adduct, kelevan. Approximately 90–99% of the total volume of chlordecone produced during this time was exported to Europe, Asia, Latin America, and Africa (DHHS 1991; EPA 1978a).

### 5.2.3 Use

Because it is nonflammable, mirex was marketed primarily as a flame retardant additive in the United States from 1959 to 1972 under the trade name Dechlorane® for use in various coatings, plastics, rubber, paint, paper, and electrical goods (Budavari et al. 1989; EPA 1978b; IARC 1979; Kutz et al. 1985; Verschueren 1983). Mirex was most commonly used in the 1960s as an insecticide to control the imported fire ants (*Solenopsis invicta* and *S. richteri*) in Alabama, Arkansas, Florida, Georgia, Louisiana, Mississippi, North Carolina, South Carolina, and Texas (Carlson et al. 1976; EPA 1978b; IARC 1979; Waters et al. 1977a, 1977b). From 1962 to 1976, approximately 132 million acres (53.4 million hectares) in nine states were treated with approximately 485,000 pounds (226,000 kg) of mirex at a rate of 4.2 g/hectare (later reduced to 1.16 g/hectare) (IARC 1979). Mirex was chosen for fire ant eradication

programs because of its effectiveness and selectiveness for ants (Carlson et al. 1976; Waters et al. 1977a, 1977b). It was originally applied aerially at concentrations of 0.3–0.5%.

However, aerial application of mirex was replaced by mound application because of suspected toxicity to estuarine species and because the goal of the fire ant program was changed from eradication to selective control. Mirex was also used successfully in controlling populations of leaf cutter ants in South America, harvester termites in South Africa, Western harvester ants in the United States, mealybugs in pineapples in Hawaii, and yellowjacket wasps in the United States (EPA 1978b; IARC 1979; Waters et al. 1977a). All registered products containing mirex were effectively canceled on December 1, 1977 (Sittig 1980). However, selected ground application was allowed until June 30 1978, at which time the product was banned in the United States with the exception of continued use in Hawaii on pineapples until stocks on hand were exhausted (EPA 1976; Holden 1976; Sittig 1980; Waters et al. 1977b).

Until August 1, 1976, chlordecone was registered in the United States for use on banana root borer (in the U.S. territory of Puerto Rico); this was its only registered food use. Additional registered formulations included nonfood use on nonfruit-bearing citrus trees to control rust mites; on tobacco to control tobacco and potato wireworms; and for control of the grass mole cricket, and various slugs, snails, and fire ants in buildings, lawns, and on ornamental shrubs (EPA 1978b; Epstein 1978; IARC 1979). The highest reported concentration of chlordecone in a commercial product was 50%, which was used to control the grass mole cricket in Florida (Epstein 1978). Chlordecone has also been used in household products such as ant and roach traps at concentrations of approximately 0.125% (IARC 1979). The concentration used in ant and roach bait was approximately 25% (Epstein 1978). All registered products containing chlordecone were effectively canceled as of May 1, 1978 (Sittig 1980).

### 5.2.4 Disposal

Since mirex and chlordecone are not flammable and are very stable in the environment, many disposal methods investigated for these chemicals have proven unsuccessful (Sullivan and Krieger 1992; Tabaei et al. 1991; Waters et al. 1977a).

Mirex is unaffected by hydrochloric, sulfuric, and nitric acids, and would be expected to be extremely resistant to oxidation except at the high temperatures of an efficient incinerator (EPA 1978b; Sittig 1980; WHO 1984). A recommended method of disposal for mirex is incineration or long-term storage (Holloman et al. 1975; IARC 1979). Polyethylene glycol or tetraethylene glycol and potassium

hydroxide when used in combination with sodium borohydride or alkoxyborohydrides, produce a powerful reducing media which quantitatively destroys mirex at 70°C. The reduction rate is further increased by using tetrahydrofuran and catalytic quantities of $Bu_3SnH$/AiBN, which produce 100% destruction of mirex to hexahydromirex within 1 hour at 58°C (Tabaei et al. 1991).

Chlordecone is considered an EPA hazardous waste and must be disposed of according to EPA regulations (EPA 1980). Degradation of chlordecone has been evaluated in the presence of molten sodium (Greer and Griwatz 1980). Addition of chlordecone to molten sodium at a temperature of 250°C resulted in significant degradation of chlordecone with small quantities of <12 ppm observed in the reaction products. Microwave plasma has also been investigated as a potential disposal mechanism for chlordecone (DeZearn and Oberacker 1980). An estimated 99% decomposition was observed in a 5-kw microwave plasma system for 80% chlordecone solution, slurry, or solid. Another recommended disposal method for chlordecone is destruction in an incinerator at approximately 850°C followed by off-gas scrubbing to absorb hydrogen chloride (IRPTC 1985).

Activated carbon adsorption has been investigated for the treatment of waste waters contaminated with chlordecone (EPA 1982). The discharge of chlordecone in sewage disposal systems is not recommended, as it may destroy the bacteriological system (IRPTC 1985). Chlordecone as a waste product in water may be dehalogenated by a process involving ultraviolet light and hydrogen as a reductant. The reaction is pH dependent, and degradation is best when the system contains 5% sodium hydroxide. Using this method, 95–99% of chlordecone is removed within 90 minutes. The degradation products are the mono-, di-, tri-, tetra-, and pentahydro derivatives of chlordecone. This degradation method is applicable to chlordecone in hazardous wastes at concentrations in the ppm (mg/L) range and lower (Reimers et al. 1989; Sittig 1980).

## 5.3 RELEASES TO THE ENVIRONMENT

Mirex has been detected in air, surface water, soil and sediment, aquatic organisms, and foodstuffs. Historically, mirex was released to the environment primarily during its production or formulation for use as a fire retardant and as a pesticide. There are no known natural sources of mirex and production of the compound was terminated in 1976. Hazardous waste disposal sites and contaminated sediment sinks in Lake Ontario were the major sources for mirex releases to the environment (Brower and Ramkrishnadas 1982; Comba et al. 1993).

Chlordecone has been detected in the air, surface water, soil and sediment, aquatic organisms and foodstuffs. Historically chlordecone was released to the environment primarily during its production at a manufacturing facility in Hopewell, Virginia. There are no known natural sources of chlordecone and production of the compound was terminated in 1975. Hazardous waste disposal sites and contaminated sediment sinks in the James River were the major sources for chlordecone release to the environment (EPA 1978a; Huggett and Bender 1980; Lunsford et al. 1987).

### 5.3.1 Air

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

Little information on historic releases of mirex to the air was located. Some atmospheric contamination may have occurred due to releases from manufacturing facilities, which were primarily located near Niagara Falls, New York, and State College, Pennsylvania; however, no quantitative sampling data were located (EPA 1978a). Atmospheric releases of mirex could result from airborne dust from the production and processing of mirex or Dechlorane®, combustion of products containing Dechlorane®, or volatilization of mirex applied as a pesticide (WHO 1984). Because mirex was principally dispersed as a pesticide in a bait form associated with corn cob grit particles that settle rapidly, the amount of mirex remaining airborne should have been insignificant. Furthermore, volatilization of mirex after application should also have been insignificant because of the high melting point and low vapor pressure of the bait (EPA 1978a).

Although release of mirex to the atmosphere was probably small in comparison to amounts released to surface water, soil, and sediment, infrequent detections of minute concentrations of mirex in air (mean concentration 0.35 $pg/m^3$) and rainfall (<0.5 ng/L [ppt]) samples have been reported many years after production ceased (Hoff et al. 1992; Strachan 1990; Wania and MacKay 1993). Arimoto (1989) estimated that 5% of the total input of mirex to Lake Ontario was attributed to atmospheric deposition.

Large amounts of chlordecone were released into the air from a chemical manufacturing plant in Hopewell, Virginia, from April 1974 through June 1975. Throughout the manufacturing period, extensive areas of the environment were contaminated with chlordecone because of improper manufacturing and disposal processes (Lewis and Lee 1976). Concentrations of chlordecone in the air surrounding the plant ranged from 0.18 $ng/m^3$ to a maximum of 54.8 $\mu g/m^3$ which was found in a sample

collected 200 m from the plant (Epstein 1978). High-volume air samplers in operation 200 m from the plant were found to contain this chlordecone level, which constituted over 50% of the total particulate loading. Chlordecone concentrations at more distant sites (up to 15.6 miles away) ranged from 1.4 to 20.7 ng/$m^3$ (Epstein 1978). The long-range transport properties of chlordecone indicate that at least a small portion of the chlordecone emissions were of a fine particle size having a relatively long residence time in the atmosphere (Lewis and Lee 1976).

### 5.3.2 Water

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

Mirex has been released to surface waters via waste waters discharged from manufacturing and formulation plants, in activities associated with the disposition of residual pesticides, and as a result of its direct use as a pesticide, particularly in the fire ant eradication program conducted in several southern states.

Releases of mirex in industrial wastes were greatest during the manufacture of this chemical between 1957 and 1976 by the Hooker Chemical and Plastics Corporation in Niagara Falls, New York. Releases to the Niagara River peaked between 1960 and 1962 at 200 kg/year (440 pounds/year), but subsequently declined to 13.3 kg/year (29 pounds/year) in 1979, and 8 kg/year (18 pounds/year) in 1981 (Durham and Oliver 1983; Lewis and Makarewicz 1988). Releases to the Oswego River occurred as a result of discharges from Armstrong World Industries Inc. in Volney, New York (Lum et al. 1987; Mudambi et al. 1992). Since production of mirex was discontinued in 1976 (Kaiser 1978), releases after 1976 were the result of leaching from dump sites adjacent to the Niagara and Oswego Rivers, both of which feed into Lake Ontario (Kaminsky et al. 1983) and releases of mirex from sediment sinks in Lake Ontario. Total loading of mirex to Lake Ontario has been estimated to be 688 kg (1,517 pounds), with half of this incorporated into the sediments (Van Hove Holdrinet et al. 1978; Lewis and Makarewicz 1988). A study by Comba et al. (1993), however, estimated total loading of mirex to Lake Ontario to be 2,700 kg (6,000 pounds) over 40 years, of which 550 kg (1,200 pounds) has been removed mainly by transport via outflowing water into the St. Lawrence River.

In addition to direct releases of mirex to surface waters that occurred at the manufacturing plant in Niagara Falls, New York, an estimated 226,000 kg (498,000 pounds) of mirex were used as a pesticide to

treat 132 million acres (53.4 million hectares) in nine southern states from 1962 to 1976 as part of the fire ant eradication program conducted by the Department of Agriculture (IARC 1979). Mirex insecticide baits were dispersed by aerial applications, and mirex could be released into surface water directly or could reach surface waters via runoff. Because mirex binds tightly to organic-rich soils, leaching is not generally expected to occur. However, mirex residues have been detected (concentration unspecified) in groundwater well samples collected in proximity to agricultural land in New Jersey (Greenburg et al. 1982). In a South Carolina study, mirex was also detected in potable water supplies in two rural counties. Mirex was detected in 12.5% of water samples at a mean concentration of 2 ng/L (ppt) (range from not detectable to 30 ng/L) in Chesterfield County and was detected in 72.7% of the water samples at a mean concentration of 83 ng/L (range of not detectable to 437 ng/L) in rural Hampton County. The authors attributed the higher mirex residues in the potable water of Hampton County to the extensive use of mirex in this county for fire ant control (Sandhu et al. 1978).

Chlordecone has been primarily released to surface waters in waste waters from a manufacturing plant in Hopewell, Virginia, and may be released in activities associated with the disposal of residual pesticide stocks, and as a result of the direct use of mirex. Chlordecone has been released directly as a contaminant of mirex and indirectly from the degradation of mirex.

Production of chlordecone at a manufacturing plant in Hopewell, Virginia, from 1966 to 1975, resulted in the release of the compound, primarily through industrial discharge of waste water into the Hopewell municipal sewage system, which discharged into Baileys Creek, and ultimately flowed into the James River. Leaching and erosion of contaminated soils from the plant site and direct discharge of solid wastes also contributed to the chlordecone content in the James River estuary (Colwell et al. 1981; Nichols 1990). Effluent from the manufacturing plant contained 0.1–1.0 mg/L (ppm) chlordecone, and water from the plant's holding ponds contained 2 to 3 mg/L (ppm) chlordecone (Epstein 1978). It has been estimated that 7,500–45,000 kg (16,500–100,000 pounds) of the 1,500,000 kg (3.3 million pounds) of chlordecone produced at the plant entered the estuary in industrial effluent or runoff (Colwell et al. 1981; Nichols 1990).

Another source of chlordecone release to water may result from the application of mirex containing chlordecone as a contaminant and by the degradation of mirex, which was used extensively in several southern states. Carlson et al. (1976) reported that dechlorinated products including chlordecone were formed when mirex bait, or mirex deposited on soil after leaching from the bait, was exposed to sunlight,

other forms of weathering, and microbial degradation over a period of 12 years. Chlordecone residues in the soil could find their way to surface waters via runoff.

### 5.3.3 Soil

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

Mirex is not currently registered for use in the United States, so release of mirex to soil from pesticide applications is no longer of concern. However, use of mirex as a pesticide for fire ant control required the spraying of this chemical on soils of an estimated 132 million acres in the southern United States (IARC 1979). An estimated 226,000 kg (498,000 pounds) of mirex were used in nine states from 1962 to 1976 as part of the fire ant eradication program conducted by the Department of Agriculture (IARC 1979).

Releases of mirex to sediment as a result of industrial waste water discharges were noted in Lake Ontario near the mouth of the Niagara River. Lake Ontario sediment concentrations were correlated with the years of peak production and use, and were found to decrease in the upper sediments as use was restricted in the late 1970s (Durham and Oliver 1983). Total loading of mirex to Lake Ontario has been estimated to be 688 kg (1,517 pounds), with half of this amount incorporated into the sediments (Van Hove Holdrinet et al. 1978; Lewis and Makarewicz 1988). However, a study by Comba et al. (1993) involving development of a mass balance model for mirex in Lake Ontario and the Gulf of St. Lawrence estimated that over 40 years, approximately 2,700 kg (6,000 pounds) of mirex entered Lake Ontario, of which 550 kg (1,200 pounds) has been removed via transport to the St. Lawrence estuary. Removal of mirex from Lake Ontario has resulted primarily by outflowing water containing suspended sediment.

Chlordecone is not currently registered for use in the United States. However, use of chlordecone as a pesticide to control banana borers on bananas, tobacco wireworms on tobacco, mole crickets on turf, and various slugs, snails, and ants in buildings, lawns, and ornamental shrubs, required the application of this chemical to soils (Epstein 1978; IARC 1979). No estimate of the amount of chlordecone released from these uses was found. Chlordecone releases to soils may also occur as a result of the application of mirex containing chlordecone as a contaminant and by the degradation of mirex which was used extensively in a regional fire ant eradication program. As stated in Section 5.2.2, Carlson et al. (1976) reported that dechlorinated products, including chlordecone, were formed when mirex bait, or mirex deposited on soil after leaching from the bait, was exposed to sunlight, other forms of weathering, and microbial

degradation over a period of 12 years. No estimates of the amount of chlordecone released from the application and degradation of mirex are available.

Chlordecone releases to soil occurred at a production facility in Hopewell, Virginia. Soil samples adjacent to the site contained 1–2% chlordecone (l0,000–20,000 mg/kg [ppm]), and surface soils up to 3,000 feet from the site contained concentrations of 2–6 mg/kg (ppm) (Epstein 1978).

The major release of chlordecone to sediments, however, occurred indirectly as a result of waste water discharges, runoff of contaminated soil, and direct disposal of solid wastes at a production facility in Hopewell, Virginia. An estimated l0,000–30,000 kg (22,000–66,100 pounds) of chlordecone are associated with bottom sediment in the James River estuary (Huggett and Bender 1980; Nichols 1990). This sediment serves as a reservoir for future release of chlordecone via resuspension of sediments resulting from storms or dredging activities (Lunsford et al. 1987).

## 5.4 ENVIRONMENTAL FATE

### 5.4.1 Transport and Partitioning

***Mirex.*** Because mirex is a very hydrophobic compound with a low vapor pressure, atmospheric transport is unlikely (Hoff et al. 1992). These authors reported detecting mirex in only 5 of 143 samples at a maximum and mean concentration of 22 $pg/m^3$ and 0.35 $pg/m^3$, respectively. Based on a vapor pressure of $<3x10^{-7}$ mm Hg at 25°C, mirex is expected to exist mainly in the particulate phase with a small proportion existing in the vapor phase in the ambient atmosphere (IARC 1979). A mass balance approach to the movement of mirex within Lake Ontario indicates that 5% of the total input of mirex to the lake can be attributed to atmospheric deposition compared with 72% of benzo(a)pyrene (Arimoto 1989).

Based on a calculated soil sorption coefficient ($K_{oc}$) of 1,200 (5,800 experimental) for mirex, this compound will tightly bind to organic matter in soil and, therefore, will be highly immobile. Thus, mirex is most likely to enter surface waters as a result of soil runoff (Kenaga 1980). In addition, most land applications of mirex to soils containing high organic content would result in very little leaching through soil to groundwater. However, leaching of mirex from some agricultural soils can occur as mirex has been detected in groundwater wells near agricultural areas (Greenburg et al. 1982; Sandhu et al. 1978).

When released to surface waters, mirex will bind primarily (80–90%) to the dissolved organic matter in the water with a small amount (10–20%) remaining in the dissolved fraction, because mirex is a highly

hydrophobic compound (Yin and Hassett 1989). Mean mirex concentrations in sediments, collected at four basins in Lake Ontario between 1982 and 1986, ranged from 30 to 38 μg/kg in three of the basins within the water circulation pattern of the lake. A fourth basin outside the pattern showed much lower concentrations (6.4 μg/kg), indicating that mirex was being transported with the lake water (Oliver et al. 1989). The residence time for mirex in Lake Ontario water was estimated to be 0.3 years. This indicated that mirex was either scavenged by particles or was chemically reactive and, therefore, was rapidly removed from the water column (Arimoto 1989).

Since the only sources of mirex in Lake Ontario are contaminated sediments, mirex in the water column is assumed to have come from resuspended sediments (Oliver et al. 1989). The source of the mirex in Lake Ontario surficial sediments was determined to be suspended sediments from the Niagara River, which were found to contain 8–15 and 55 μg/g (ppm) mirex in the upper and lower river sections, respectively. The surficial sediments contained 3 μg/g in the upper river (above the manufacturing and dump sites), 86 μg/g in the lower river (below the sites), and 10 μg/g in the western basin of Lake Ontario, indicating that mirex-containing sediments were being carried down the river with the current and deposited in Lake Ontario (Mudroch and Williams 1989). Kaminsky et al. (1983), reported a range of 8.2–62 ppb (μg/kg) in sediment from the eastern and central basins of Lake Ontario. Over 94% of the suspended particulate matter entering the lake is eventually deposited in lake sediments (Lum et al. 1987). Mirex concentrations in sediments of Lake Ontario show a strong correlation with peak production years (Durham and Oliver 1983; Eisenreich et al. 1989). Although there was evidence of sediment bioturbation by deposit-feeding worms and burrowing organisms, the sediment profiles for mirex and other chlorinated hydrocarbons were not destroyed (Eisenreich et al. 1989). Between the 1960s when mirex production began, and the early 1980s after production ceased, levels of mirex in bottom sediments increased in Lake Ontario, with the Niagara River being the major source of this compound (Allan and Ball 1990).

Mirex may be removed from Lake Ontario by several mechanisms, including the transport of contaminated suspended particulate material via water outflow into the St. Lawrence River, biomass removal through fishing and migration (e.g., migrating eels contaminated with mirex), volatilization, and photolysis (Comba et al. 1993; Lum et al. 1987). Transport of mirex accumulated in body tissues by eels has been estimated to be 2,270 grams annually or twice the amount of mirex removed by transport of suspended particulates (1,370 grams annually) (Lum et al. 1987).

The transport of mirex out of Lake Ontario, (a known reservoir), to its tributaries is also possible as a result of migrating fish, which move from the lake into the tributary streams to spawn. Fish, such as

Pacific salmon, become contaminated with mirex while in the lake. These fish then swim upstream in the tributaries to their spawning grounds, spawn, and die. A direct transfer of mirex may then occur when resident stream fish feed on the decomposing carcasses and/or eggs, both of which contain mirex residues. Indirect transfer can occur as a result of the release of mirex from the salmon into the water or sediments and subsequent movement up the food chain. Movement of mirex back into Lake Ontario is also possible when the contaminated eggs hatch and surviving juvenile salmon return to the lake (Lewis and Makarewicz 1988).

Algae are known to bioaccumulate mirex, with BCFs in the range of 3,200–7,300, while bacteria have a BCF of 40,000 with an octanol-water partition coefficient of 7.8 million (Baughman and Paris 1981). Based on a water solubility of 0.6 mg/L, a BCF of 820 was calculated for mirex (Kenaga 1980). Bioaccumulation of mirex also occurred in invertebrates exposed to 0.001–2.0 µg/g mirex in water; tissue residues ranged from 1.06 to 92.2 µg/g (de la Cruz and Naqvi 1973). After 28 days of exposure, the BCF values for the amphipod (*Hyallelu azteca*) and crayfish (*Orconectes mississippiensis*) were 2,530 and 1,060 respectively. Fathead minnows exposed to 33 µg/L (ppb) mirex for 56 days accumulated 122 µg/g (ppm) mirex tissue residues (BCF of 3,700), with no other evident metabolic products. Residues decreased to 88.6 µg/g 28 days after mirex was removed from the water (Huckins et al. 1982). The half-life of mirex in rainbow trout was >1,000 days in fish exposed for 96 days to a mean concentration of 4.1 ng/L, although equilibrium was not reached during the test period. A subsequent analysis comparing a laboratory BCF for mirex in rainbow trout (1,200) with an actual BCF found in rainbow trout in Lake Ontario (15,000), indicated that ingestion of contaminated food (as would occur in the lake), rather than absorption across the gills, is the primary exposure route for trout (Oliver and Niimi 1985).

Biomagnification of mirex is supported by a study of various aquatic organisms that comprise an aquatic food chain in Lake Ontario (Oliver and Niimi 1988) (see Table 5-1).

**Table 5-1. Concentrations of Mirex in Aquatic Organisms**

| Sample | Mirex concentration (µg/kg wet weight unless otherwise noted) |
|---|---|
| Water | 31±12 pg/L wet weight |
| Bottom sediment | 3.9±1.9 µg/kg dry weight |
| Suspended sediment | 15±4.4 µg/kg dry weight |
| Plankton | 1.3±0.1 |
| Mysids | 8±2.8 |
| Amphipods | 12±6.7 |
| Oligochaetes | 6.9±2.9 |

**Table 5-1. Concentrations of Mirex in Aquatic Organisms**

| Sample | Mirex concentration (µg/kg wet weight unless otherwise noted) |
|---|---|
| Sculpins | 57 |
| Alewives | 45 |
| Small smelts | 26±3.6 |
| Large smelts | 53 |
| Average fish | 180±150 |

Source: Oliver and Niimi 1988

In these food chains, alewives feed primarily on mysids and to a lesser extent on amphipods; sculpins feed on amphipods, then mysids; smelt feed on mysids. Mysids feed on zooplankton, with amphipods and oligochaetes consuming detrital matter. The alewives and smelt are preyed upon by salmonids, such as trout (Oliver and Niimi 1988). A comparison of concentrations of mirex in lake trout, a predator species, with those in smelt, a prey species, gives a ratio of 1.26, indicating that biomagnification occurs up the food chain (Thomann 1989).

Mirex can also bioaccumulate in terrestrial plants. Azalea leaves, exposed to 0.023 µg/kg of mirex in greenhouse air, had significant uptake of the pesticide resulting in a BCF of $1.18 \times 10^7$ (log BCF=7.07) (Bacci et al. 1990). Mirex residues ranging from 10 to 1,710 µg/kg (ppb) were detected in soybeans, garden beans, sorghum, and wheat seedlings grown on substrates containing 0.3–3.5 mg/kg (ppm) mirex (de la Cruz and Rajanna 1975). Based on these data and known soil concentrations, it has been estimated that plants grown on contaminated soil could contain 0.0002–2 µg/kg (ppb) mirex (EPA 1978a). No information on the uptake of mirex by plants under field conditions was located.

In a 1972 residue study conducted in Mississippi during the time when mirex was being used extensively in fire ant control programs, Naqvi and de la Cruz (1973) reported mirex accumulation in grassland invertebrates (e.g., spiders and grasshoppers) ranging from 100 to 700 µg/kg (ppb) (mean 280 µg/kg). Hebert et al. (1994) studied organochlorine pesticides in a terrestrial food web on the Niagara Peninsula in Ontario, Canada, from 1987 to 1989. These authors reported mirex concentrations in the various food web compartments as follows: soil (not detectable), plants (not detectable), earthworms (not detectable to 0.4 µg/kg), mammals (not detectable to 0.5 µg/kg), starlings (0.9–1.6 µg/kg), robins (4.7–18.9 µg/kg), and kestrels (4.7–22.2 µg/kg), which suggests that biomagnification of mirex is occurring. The earthworm appeared to be a particularly important species for organochlorine transfer from the soil to organisms occupying higher trophic levels. Connell and Markwell (1990) reported transfer of lipophilic compounds

(such as mirex) through a three-phase system involving soil to soil water to earthworm partitioning. The transfer is a passive process and is principally dependent on the lipid content of the worms and the organic content of the soil.

***Chlordecone.*** The fate and transport of chlordecone is very similar to mirex. Based on its low vapor pressure and high $K_{oc}$, chlordecone in the air may be expected to be associated primarily with particulate matter (Kenaga 1980). However, only small amounts of chlordecone may volatilize into the air. Chlordecone volatilizes more slowly from water (0.024% applied amount/mL of evaporated water) than from sand, loam, or humus soil (0.036, 0.035, and 0.032%, respectively) (Kilzer et al. 1979).

Atmospheric transport of chlordecone particles was reported as a result of emissions from a production facility in Virginia. Chlordecone concentrations at up to 15.6 miles away ranged from 1.4 to 20.7 $ng/m^3$ (Epstein 1978). The long-range transport properties of chlordecone indicate that at least a portion of the emissions were of a fine particle size having a relatively long residence time in the atmosphere (Lewis and Lee 1976).

The major industrial release of chlordecone occurred to surface waters of the James River. Chlordecone, because of its relatively low solubility in water and lipophilic nature, is readily absorbed to particulate matter in water and is ultimately deposited in sediments (EPA 1978a; Lunsford et al. 1987). Once adsorbed to sediments, chlordecone remains relatively immobile in the normal range of pH (7–8) and salinity (0.06–19.5 %) encountered in an estuary. While chlordecone is associated mainly with the organic portion of bottom sediments, sediment areas with high percentages of inorganic mineral grains are relatively free of contamination. The greatest mass of chlordecone (an estimated 6,260 pounds [2,840 kg]) was found in a sink where the sedimentation was relatively rapid. Transport is primarily through adsorption of chlordecone to fine organic particles in the water column. Its movement and deposition follow estuarine circulation, which is seaward from the freshwater reaches and upper estuarine water layer, and reflux downward for suspended materials (Nichols 1990).

While much of the chlordecone that was present in contaminated sediments in 1976 is still in the sediment, it is continuously being buried under several centimeters of new sediment each year (Huggett and Bender 1980). Storm activities and dredging are of concern because they would result in reenrichment of the surface sediments in areas with chlordecone contaminated sediment previously buried by natural ongoing sedimentation processes in the estuary (Huggett and Bender 1980; Lunsford et al. 1987).

Chlordecone has been found to have a very high bioaccumulation potential in fish and other aquatic organisms. Atlantic menhaden (*Brevoortia lyrunnus*) and Atlantic silver-sides (*Menidia menidiu*) had 28-day BCFs of 2,300–9,750 and 21,700–60,200, respectively (Roberts and Fisher 1985). Based on a water solubility of 3 mg/L, a BCF of 333 was estimated for chlordecone. However, the measured value was 8,400 (Kenaga 1980). Using a log octanol-water partition coefficient for chlordecone of 6.08, a BCF of 6,918 was estimated for the oyster (Hawker and Connell 1986). However, an oyster BCF of 10,000 has been reported with tissue concentrations at equilibrium within 8–17 days (Bahner et al. 1977). For estuarine organisms such as mysids, grass shrimp, sheepshead minnows, and spot, BCFs were measured to be 13,000, 11,000, 7,000, and 3,000, respectively (Bahner et al. 1977). Shad roe taken from the James River contained chlordecone levels that were 140% higher than muscle tissue residues, indicating a partitioning of the chemical into the lipid-rich eggs (Bender and Huggett 1984).

The accumulation of chlordecone was studied in a terrestrial/aquatic laboratory model ecosystem by Francis and Metcalf (1984). Radiolabeled chlordecone was applied to sorghum seedlings grown on the terrestrial portion of the aquarium. The treated seedlings were eaten by salt marsh caterpillars. In the aquatic portion, chlordecone was transferred through several species-an algae, snail, water flea mosquito larvae, and mosquito fish. After 33 days, the BCFs were 0.35 for the algae, 637.4 for the snails, 506.9 for the mosquito larvae, and 117.9 for the mosquito fish. A BCF for chlordecone of approximately 2.1 was determined for a water-algae-oyster food chain; however, a biomagnification factor >10.5 was measured for a water-brine shrimp-mysid-spot food chain with a water concentration of 0.1 mg/L (ppm) chlordecone (Bahner et al. 1977).

Plant uptake of chlordecone from the soil via the roots, and volatilization of chlordecone from soil with plant uptake via the leaves were found to be negligible in a closed laboratory system using barley seedlings. This indicates that bioaccumulation of chlordecone by plants (lowest on the terrestrial food chain) is very unlikely based on its log soil adsorption coefficient of almost 4.0 (Topp et al. 1986). No information on the uptake of chlordecone by plants under field conditions was located.

### 5.4.2 Transformation and Degradation

#### Air

***Mirex.*** Little information was found on the degradation of mirex in the atmosphere. Mirex is expected to be stable against photogenerated hydroxyl radicals in the atmosphere (Eisenreich et al. 1981).

***Chlordecone.*** Photolysis of chlordecone in the atmosphere does not appear to be an important degradation pathway for this compound. While nonvolatile products of photolysis were not monitored, only 1.8% of the chlordecone adsorbed on silica gel and exposed to ultraviolet light (wavelength >290 nm) was photolyzed to carbon dioxide or other volatile compounds (Freitag et al. 1985).

#### Water

***Mirex.*** The degradation of mirex in water occurs primarily by photolysis. During the photo-decomposition of mirex, the chlorine atoms are replaced by hydrogen atoms. The primary photoreduction product of mirex in water is photomirex (Andrade et al. 1975); the rate of this reaction can be increased by the presence of dissolved organic matter (such as humic acids) and was greatest at 265 nm in Lake Ontario water (Mudambi and Hassett 1988). In Lake Ontario, Mudambi et al. (1992) reported that the ratio of photomirex to mirex (P/M) increased in the stratified surface layer of the lake from spring until autumn and in water from Oswego Harbor. P/M ratios in the mirex source sediments (the Niagara and Oswego Rivers) were very low (<0.07), whereas higher P/M ratios were seen in the lake bottom sediments (>0.10) and surface waters (>0.30). These findings suggest that photomirex in Lake Ontario is produced by photolysis of mirex present in the surface waters and it is then partitioned between water, sediment, and biota.

***Chlordecone.*** Degradation of chlordecone to an unidentified compound was studied in water in a terrestrial/aquatic laboratory model ecosystem. Degradation occurred to some extent during the 33-day exposure period, and unidentified metabolites were detected in all organisms in the system-algae, snail, mosquito, and mosquito fish (Francis and Metcalf 1984). An earlier laboratory study in which fathead minnows were exposed to chlordecone in a flow-through diluter system for 56 days found that chlordecone was bioconcentrated 16,600 times by the minnows; however, only 1–5% of these residues were chlordecone (Huckins et al. 1982). Several observations suggested that some of the chlordecone residues present in the minnows were chemically bound to biogenic compounds.

*Pseudomonas aeruginosa* strain K03 and a mixed aerobic enrichment culture isolated from sewage sludge lagoon water were found to aerobically transform chlordecone to monohydrochlordecone in 8 weeks. Monohydrochlordecone constituted 14.2 and 14.5% of the chlordecone transformation products for the *P. aeruginosa* and mixed aerobic enrichment culture, respectively. The *P. aeruginosa* K03 strain and the mixed culture also produced 15.6 and 4.2% dihydrochlordecone, respectively (Orndorff and Colwell 1980). None of the bacterial strains were able to use chlordecone as a sole carbon source; therefore, co-metabolism appeared to be the only degradation process. Complete mineralization of chlordecone by bacteria is unlikely (Orndorff and Colwell 1980). Degradation of chlordecone can occur via microbial action, but the rate and extent of transformation are such that microbial action will not cause rapid removal of chlordecone from the environment except under highly enriched and selected conditions. Aerobic degradation of chlordecone by activated sludge from a municipal sewage plant showed that <0.1% of the applied chlordecone was degraded in 5 days, and the sludge showed a bioaccumulation factor of 9,900 compared with the concentration in the water (Freitag et al. 1985).

### Sediment and Soil

***Mirex***. Degradation of mirex in soil may occur by photolysis or anaerobic biodegradation, both of which are very slow removal processes. Mirex is highly resistant to aerobic biodegradation and, as such, is extremely persistent in soils (estimated half-life of 10 years) (Carlson et al. 1976; Lal and Saxena 1982). Mirex appears to have no adverse effect on resident microbial communities (Jones and Hodges 1974). Upon exposure to ultraviolet light, mirex is known to degrade to chlordecone, photomirex, and/or dihydromirex (Francis and Metcalf 1984). Detectable levels of mirex photodegradation products (monohydro derivative and chlordecone hydrate) occur within 3 days after exposure of mirex to sunlight, although after 28 days of exposure, approximately 90% of the mirex was unchanged (Ivie et al. 1974b). Anaerobic degradation relies on iron(II) porphyrin as the reductant for the dehalogenation reaction (Kuhn and Suflita 1989).

Under anaerobic conditions, mirex was slowly dechlorinated to the 10-monohydro derivative by incubation with sewage sludge bacteria for 2 months (Andrade and Wheeler 1974; Andrade et al. 1975; Williams 1977). The primary removal mechanism for mirex was anaerobic degradation as demonstrated by the 6-month stability of the compound in nine aerobic soils and lake sediments (Jones and Hodges 1974).

Aerobic degradation of mirex is a very slow and minor degradation process. Twelve years after the application of mirex to soil at 1 pound/acre, 50% of the mirex and mirex-related organochlorine compounds remained in the soil; 65–73% of the residues consisted of mirex and 3–6% consisted of chlordecone. Although concentrations were slightly higher, similar ratios of mirex (76–81%) and chlordecone (1–6%) residues were seen 5 years after an accidental spill of mirex bait on soil. Mirex underwent photolysis to form four dechlorination products: two monohydro and two dihydro compounds (Carlson et al. 1976). Two soil microbes, *Bacillus sphaericus* and *Streptomyces albus*, isolated from a field previously treated with mirex, were able to utilize 1% mirex as a sole carbon source. However, the rate of degradation, as demonstrated by carbon dioxide evolution, was slow and only about 10–20% greater than the controls after 20 hours (Aslanzadeh and Hedrick 1985).

No evidence of microbial degradation was detected for mirex exposed to hydrosoils (soils formed under conditions of saturation, flooding, or ponding long enough during the growing season to develop anaerobic conditions in the upper part) from a reservoir (not previously contaminated with chlordecone) and from chlordecone-contaminated hydrosoils from the James River area of Virginia under either anaerobic or aerobic conditions for 56 days (Huckins et al. 1982). The concentrations of chlordecone in the anaerobic and aerobic hydrosoils averaged 0.38 and 0.54 µg/g, respectively. Some photodegradation of mirex to photomirex was seen in an artificial salt marsh ecosystem; the photomirex was subsequently photodegraded to the 2,8- or 3,8-dihydro derivative. Most mirex loss occurred during the first 7 days after application (from 2.65 to 2.13 mg/g) with a steady accumulation of photomirex (610 ppb/day [µg/kg/day]) through day 21, accumulation of 17 µg/kg/day of 2,8- or 3,8-dihydro derivative through day 35, and an accumulation rate of 206 µg/kg/day for the 10-monohydro photoproduct that is formed in the presence of amines. The 8-monohydro derivative (photomirex) was found to accumulate in the salt marsh organisms and sediment (Cripe and Livingston 1977).

Application of radiolabeled mirex to plants grown in a terrestrial/aquatic laboratory model ecosystem indicated that when the plant leaves were eaten by caterpillars, the aquatic system became contaminated. Mirex was detected in all segments of two aquatic food chains (alga > snail and plankton > daphnia > mosquito > fish) within 33 days. Undegraded mirex contributed to over 98.6, 99.4, 99.6, and 97.9% of the radiolabel in fish, snails, mosquitoes, and algae, respectively. No metabolites of mirex were found in any of the organisms (Francis and Metcalf 1984; Metcalf et al. 1973).

***Chlordecone.*** Chlordecone is similar to mirex in structure and is also highly persistent in soils and sediments (half-life expected to be analogous to 10 years duration for mirex) because of its resistance to

biodegradation, although some microbial metabolism of chlordecone has been reported (Lal and Saxena 1982; Orndorff and Colwell 1980). No evidence of microbial degradation was detected for chlordecone exposed to hydrosoils from a reservoir (not previously contaminated with chlordecone) and from Bailey Creek (contaminated with chlordecone) under either anaerobic or aerobic conditions for 56 days (Huckins et al. 1982).

Three *Pseudomonas* species extracted from soil samples to which chlordecone was added (1 mg/mL) were found to utilize chlordecone, as a sole carbon source, with quantifiable degradation (67–84%) in 14 days. Among the degradation products of chlordecone, only hydrochlordecone and dihydrochlordecone were identified (George and Claxton 1988; George et al. 1986). Sewage sludge bacteria and sediment bacteria, primarily *P. aeruginosa* strain KO3, were able to aerobically degrade chlordecone by 10–14% to monohydrochlordecone and, to a lesser extent, dihydrochlordecone in 8 weeks. None of the bacterial strains was able to use chlordecone as a sole carbon source; therefore, co-metabolism appeared to be the only degradation process. Complete mineralization of chlordecone by bacteria is unlikely (Orndorff and Colwell 1980). Concentrations of chlordecone >0.2 mg/L are likely to inhibit microbial activity, whereas concentrations <0.01 mg/L had no effects on cell count or uptake of amino acids. Bacteria in James River sediment did not produce significant concentrations of chlordecone metabolites (Colwell et al. 1981).

Degradation of chlordecone in a terrestrial ecosystem was studied by applying the compound to soil, growing plants on the soil; and then determining the amount of chlordecone in each compartment after 1 week. During this time, only 0.1% of the applied chlordecone (2 mg/kg) was decomposed to carbon dioxide from the soil, and 0.3 mg/kg (approximately 15% of the applied concentration) was accumulated by the barley plants. Less than 10% of the applied chlordecone was degraded in the soil or converted by the barley plants, and there was no volatilization of the compound from the soil to the air (Kloskowski et al. 1981). A laboratory soil-plant system showed that degradation of chlordecone, as determined by soil residues remaining after volatilization and mineralization, was 1–3% after 1 week; this compared favorably with the residues remaining in soil in the field after one growing season (Scheunert et al. 1983). Analysis of soil contaminated with chlordecone collected in the vicinity of the chlordecone production facility showed some photolytic degradation of the compound with the production of small amounts of monohydro isomers of chlordecone (Borsetti and Roach 1978).

## 5.5 LEVELS IN THE ENVIRONMENT

Reliable evaluation of the potential for human exposure to mirex and chlordecone depends, in part, on the reliability of supporting analytical data from environmental samples and biological specimens. Concentrations of mirex and chlordecone 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 mirex and chlordecone levels monitored or estimated 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 lowest limit of detections that are achieved by analytical analysis in environmental media are summarized in Table 5-2 for mirex and Table 5-3 for chlordecone.

**Table 5-2. Lowest Limit of Detection for Mirex Based on Standards[a]**

| Media | Detection limit | Reference |
|---|---|---|
| Air | 0.1 ng/m$^3$ | Lewis et al. 1977 |
| Drinking water | 10 ng/L | Sandhu et al. 1978 |
| Surface water and groundwater | 10 ng/L | Sandhu et al. 1978 |
| Soil | 1 ppb | Seidel and Lindner 1993 |
| Sediment | 0.002 ppb | Sergeant et al. 1993 |
| Whole blood | 0.04 ng/g | Mes 1992 |

[a]Detection limits based on using appropriate preparation and analytics. These limits may not be possible in all situations.

**Table 5-3. Lowest Limit of Detection for Chlordecone Based on Standards[a]**

| Media | Detection limit | Reference |
|---|---|---|
| Air | 10 ng/sample | NIOSH 1984 |
| Water | 20 ng/L | Saleh and Lee 1978 |
| Soil | 10–20 ppb | Saleh and Lee 1978 |
| Sediment | 10–20 ppb | Saleh and Lee 1978 |
| Whole blood | 10 µg/L | Caplan et al. 1979 |

[a]Detection limits based on using appropriate preparation and analytics. These limits may not be possible in all situations.

No data are available on levels of mirex or chlordecone in air, water, and soil at NPL sites (ATSDR 2019).

### 5.5.1 Air

***Mirex.*** Mirex has been detected in wet precipitation over rural areas at concentrations <1 ng/L (ppt) (EPA 1981). Rain fall samples collected at several sites in 1985–1986 as part of the Great Lakes Organics Rain Sampling Network contained from >0.2 to <0.5 ng/L (ppt) of mirex. Mirex was not detected consistently at many stations throughout the sampling period; therefore, quantitative results for mirex were not presented (Strachan 1990). Air samples taken over southern Ontario in 1988 showed mirex in 5 of 143 samples, at an annual mean concentration of 0.35 pg/$m^3$ (range, 0.1–22 pg/$m^3$), with all of the positive samples detected in polluted environments (Hoff et al. 1992).

***Chlordecone.*** Information on atmospheric concentrations of chlordecone is limited to air sampling results obtained at the Life Sciences Products Company production site in Hopewell, Virginia. High volume air filter samples collected 200 m from the plant in March 1974 prior to initiation of production at the site contained only 0.18 to 0.35 ng/$m^3$ of chlordecone. Subsequent air sampling after production was initiated ranged from 3 to 55 μg/$m^3$. During production years 1974 and 1975, air concentrations at more distant sites up to 15.6 miles from Hopewell, Virginia, ranged from 1.4 to 20.7 ng/$m^3$ (Epstein 1978).

### 5.5.2 Water

***Mirex.*** Mirex was detected in rural drinking water samples at concentrations ranging from not detectable to 437 ng/L (ppt) (Sandhu et al. 1978). In a survey in 1987, mirex was detected in only 5 of 1,147 drinking water samples from Ontario, Canada (maximum concentration of 5 ng/L [ppt]) (Environment Canada 1992).

The pollution of the Niagara River from chemical manufacturing effluents and leachates from chemical manufacturing waste dumps has been well documented. Between 1975 and 1982, mirex was detected in the aqueous phase of 6 of 22 samples in the Niagara River at levels between 0.0005 and 0.0075 ng/L (ppt) (Allan and Ball 1990). Twelve percent of 104 whole water samples, collected from the Niagara River between 1981 and 1983, had mirex concentrations that ranged from below the detection limit (0.06 ng/L[ppt]) to 2.6 ng/L, with a median concentration of 0.06 ng/L (Oliver and Nicol 1984). Mirex was detected in the suspended particulate phase of 42 Niagara River water samples taken at the mouth of the river in 1986–1987; 17% of the samples had a mean mirex concentration of 0.022 ng/L (ppt) (Allan and Ball 1990).

In 1982, Mudambi et al. (1992) reported the mean mirex concentrations in the Lake Ontario system ranging from 1.85 to 30 pg/L. An intralake comparison of chemicals found in the Great Lakes during the 1986 spring turnover did not detect mirex in any of the lakes (Stevens and Neilson 1989), nor in the dissolved or particulate fractions of water from the St. Lawrence River between 1981 and 1987 (Germain and Langlois 1988). In 1986, low levels of mirex were found in 8 of 14 water samples taken at various locations along the St. Lawrence River (Kaiser et al. 1990). The highest concentration observed was 0.013 ng/L (ppt). Sergeant et al. (1993) reported mirex concentrations in Lake Ontario water samples declined from 0.0015 μg/L (1.5 ng/L) in 1986 to <0.0004 μg/L (0.4 ng/L) in 1988.

Mirex was detected in water samples taken in 1972 from areas in Mississippi that had been aerially treated with mirex to control the imported red fire ant (Spence and Markin 1974). Water samples taken from the bottom of a pond showed residue values that remained higher and more constant than those taken from the surface of the pond. Water showed the highest residues immediately after treatment (bottom, 0.53 μg/L [ppb]; surface, 0.02 μg/L [ppb]), and detectable levels were still present as long as 3 months after treatment (bottom, 0.005 μg/L [ppb]; surface, 0.003 μg/L [ppb]) (Spence and Markin 1974).

***Chlordecone.*** The solubility of chlordecone in water is low (1–3 mg/L) and as with mirex, contamination is more likely to be associated with the particulate matter in the water rather than the water itself. Chlordecone was detected primarily in water samples collected in and around the production facility site in Hopewell, Virginia, and in adjacent waters of the James River estuary. Effluent from the Life Sciences Products Company facility contained 0.1–1.0 mg/L (ppm) chlordecone, while water in holding ponds at the site contained 2–3 mg/L (ppm) chlordecone (Epstein 1978). Levels of chlordecone in river water in August 1975 ranged from not detectable (<50 ng/L [ppt]) in the York River and Swift Creek areas, to levels of 1–4 μg/L (ppb) in Baileys Creek which received direct effluent discharges from the Hopewell Sewage Treatment Plant. Water concentrations of up to 0.3 μg/L (ppb) were detected in the James River at the mouth of Bailey Creek and in the Appomattox River (upstream from Hopewell) at 0.1 μg/L (ppb) (Epstein 1978). Hopewell drinking water drawn from the James River contained no detectable chlordecone levels (EPA 1978a; Epstein 1978). In 1977, 12 years after production of chlordecone began and 2 years after production ceased, average concentrations of chlordecone in estuarine water (dissolved) were <10 ng/L (ppt) (Nichols 1990). In October 1981, 6 years after production at the plant ceased, chlordecone water concentrations ranged from not detectable to 0.02 μg/L (ppb) (Lunsford et al. 1987).

### 5.5.3 Sediment and Soil

***Mirex.*** Mirex was identified in sediment samples collected in 1979 from Bloody Run Creek, which is a drainage ditch for the Hyde Park landfill in Niagara Falls, New York. Mirex levels in the sediment ranged from 0.5 to 2 mg/kg (ppm) (detection limit, 0.5 mg/kg [ppm]) (Elder et al. 1981).

Between 1979 and 1981, mean mirex concentrations in suspended sediments of the Niagara River declined from 12 to 1 ng/L (ppt); concentrations in bottom sediments were generally low, ranging from <1 μg/kg (ppb) to a maximum value of 890 μg/kg (ppb), at a site believed to be the source of mirex to the river (Allan and Ball 1990). In 1981, mirex was detected in sediments of Lake Ontario near the mouth of the Niagara River at increasing concentrations to a maximum of 1,700 μg/kg (ppb) at a sediment depth of 9 cm. Concentrations decreased between 9 and 13 cm and were not detected in sediments below a depth of 13 cm. Concentrations were chronologically correlated with mirex production and peak sales periods and were reduced when its use was restricted (Durham and Oliver 1983). In 1982, mirex was detected in settling particulates from sediment traps in the Niagara River (average, 7 μg/kg [ppb]; range, 3.9–18 μg/kg [ppb]), resuspended bottom sediments from the Niagara Basin of Lake Ontario (average, 9.45 μg/kg [ppb], range 5.2−16 μg/kg [ppb]), and bottom sediments from Lake Ontario (average, 48 μg/kg [ppb]) (Oliver and Charlton 1984).

An analysis of urban runoff and sediment runoff collected between 1979 and 1983 from 12 urban areas in the Canadian Great Lakes Basin showed that mirex was not detected in any runoff waters, although it was found in 10% of 129 runoff sediment samples at a mean concentration of 1.3 μg/kg (ppb) (Marsalek and Schroeter 1988). Sediment samples collected from the St. Lawrence River between 1979 and 1981 contained low concentrations of mirex (median, <0.1 μg/kg; range, <0.1–3.3 μg/kg), indicating that Lake Ontario is the source of the contamination to the river (Sloterdijk 1991). Low levels of mirex were found in bottom sediment core samples taken from the riverine lakes in the St. Lawrence River in October 1985; the average concentration of mirex was 0.43 μg/kg (range, <0.01–0.95 μg/kg) (Kaiser et al. 1990). In 1987, mirex was detected in suspended sediments throughout the St. Lawrence River. At the St. Lawrence River stations near Kingston, the mirex concentration was approximately 5 μg/kg (ppb), but declined to about 1 μg/kg (ppb) near Quebec City (Kaiser et al. 1990).

In 1971 and 1972, mirex was detected in soil and sediment samples taken from areas in Louisiana and Mississippi that had been aerially treated with mirex to control the imported red fire ant (Spence and Markin 1974). In Louisiana, samples were collected throughout the first year after spraying. Soil and

sediment residues in the Louisiana study peaked after 1 month (soil, 2.5 μg/kg [ppb]; sediment, 0.7 μg/kg [ppb]) and gradually declined over the remainder of the year. In Mississippi, samples were collected for 4 months following spraying. Sediment residues in Mississippi also peaked about 1 month after spraying (1.1 μg/kg [ppb]) and gradually declined over the next couple of months. The residue levels found in soil in Mississippi were much more variable and showed no distinctive pattern (Spence and Markin 1974).

Less than 10% of the sediment samples taken from the San Joaquin River and its tributaries in California (an area of heavy organochloride pesticide use) in 1985 contained mirex residues; all samples contained <0.1 μg/kg (ppb) (Gilliom and Clifton 1990).

Studies of sediment from seven sampling stations in the Upper Rockaway River, New Jersey, showed that sediment quality corresponded to the land-use data for the area (Smith et al. 1987). The two upstream stations, which drain primarily forested areas of the Upper Rockaway Basin, had low mirex concentrations in the sediments (<0.1 μg/kg). The remaining stations, which drained an area consisting of residential, commercial, and industrial land including six EPA Superfund sites, had mirex concentrations ranging from 8.2 to 80 μg/kg (ppb) (Smith et al. 1987).

Sediment samples taken from 51 sampling locations in the Gulf of Mexico for the National Oceanic and Atmospheric Administration (NOAA) Status and Trends Mussel Watch Program were analyzed for mirex contamination (Sericano et al. 1990; Wade et al. 1988). Average mirex concentrations of 0.07 μg/kg (ppb) (range, <0.01–0.67) and 0.18 μg/kg (ppb) (range, <0.02–3.58) were found in sediments in 1986 and 1987, respectively. The sampling sites represent the contaminant loading for the Gulf of Mexico estuaries removed from known point-sources of contamination (Sericano et al. 1990; Wade et al. 1988).

***Chlordecone.*** With the exception of the James River area of Virginia, very little information is available on chlordecone residues in soil and sediment. Chlordecone was detected in soil immediately surrounding the Life Sciences Products Company in Hopewell, Virginia, at levels of 1–2% (10,000–20,000 mg/kg) and contamination extended to 1,000 m at concentrations of 2–6 mg/kg (ppm) (Huggett and Bender 1980).

Assessment of sediment cores taken from the James River below Hopewell, Virginia, indicated that chlordecone concentrations were greatest nearest the release site. Sediment concentrations of chlordecone in Baileys Creek, the waterbody into which effluent from the Hopewell municipal sewage treatment facility was discharged, were 2.2 mg/kg (ppm) (Orndorff and Colwell 1980). Chlordecone

concentrations of 0.44–0.74 mg/kg were found at sediment depths of 55–58 cm in the main channel of the James River. This area had the highest sedimentation rate (>19 cm/year). Further downriver, (80 km from Hopewell) in the James River estuary, chlordecone concentrations decreased and maximum concentrations were found closer to the sediment surface. The highest chlordecone concentration of 0.18 mg/kg (ppm) was from a sediment depth of 46–48 cm in an area with a sedimentation rate of 10 cm/year (Cutshall et al. 1981).

### 5.5.4 Other Media

***Mirex.*** In general, because releases of mirex from its production and use as a pesticide were terminated in the late 1970s, mirex residues in various biological organisms are much lower than those reported during or shortly after its peak years of production and use. This trend is supported by both regional and national studies.

In areas where mirex was historically used for fire ant control, it has been detected in fish and other aquatic biota from contaminated rivers. An analysis of mirex residues in primary, secondary, and tertiary consumers in oxbow lakes in Louisiana in 1980 indicated that although mirex was not detected in any water or sediment samples, or in the tissues of primary consumers (some fish), it was detected in the tissues of secondary consumers (fish and birds that consume invertebrates and insects), and in all tertiary consumers (fish-eating fish, birds, and snakes). The highest mean mirex concentrations were found in cottonmouth snakes (0.11 mg/kg [ppm]) (Niethammer et al. 1984). Fish taken from the lower Savannah River during 1985 had mirex residues in their tissues that ranged from nondetectable to 1 mg/kg (ppm) wet weight, although most residues were near 0.02 mg/kg (Winger et al. 1990).

Of all the Coho salmon collected from all of the Great Lakes in 1980, only fish taken from Lake Ontario contained detectable mirex residues at an average concentration of 0.14 μg/g (ppm) (Clark et al. 1984). The mean concentration of mirex residues in rainbow trout taken from Lake Ontario was 0.11 μg/g (ppm), while the mean water concentration in the lake was 0.008 ng/L (ppt) (Oliver and Niimi 1985). Borgmann and Whittle (1991) studied the contaminant concentration trends in Lake Ontario lake trout from 1977 to 1988. Mirex concentrations generally declined from 0.38 μg/g (ppm) in 1977 to 0.17 μg/g (ppm) in 1988, although there was considerable variability in the mirex residue data. The concentrations of mirex also showed a distinct east-west gradient across the lake. The highest mirex residues were detected in fish collected at the western side of the basin and were 70% above those detected in fish collected at the eastern portion of the basin. Suns et al. (1993) conducted a similar study of spatial and temporal trends of

organochlorine contaminants in spottail shiners from selected sites in the Great Lakes. These authors reported that mirex was only detected in fish from the Niagara River, the Credit River in western Lake Ontario, and in the St. Lawrence River at Cornwall. Mirex concentrations in spottail shiners collected during the late 1980s were generally lower than mirex residues found in spottail shiner samples collected during the 1970s. Considerable fluctuation in mirex residues in spottail shiners was observed, which precluded proper trend assessment. Based on the fish data, mirex inputs to Lake Ontario appeared to be continuing on an intermittent basis. Newsome and Andrews (1993) analyzed mirex in fillet samples of 11 commercial fish species from the Great Lakes. The highest mirex concentrations were found in carp from a closed fishery area (120 μg/kg [ppb]), eel (56.8 μg/kg), carp from an open fishery area (5.24 μg/kg), bullhead (3.63 μg/kg), and trout (2.38 μg/kg).

Burbot, a bottom-feeding fish, taken from remote lakes in Canada in 1985–1986, contained liver concentrations of mirex ranging between 3.7 and 17.4 μg/kg (ppb) lipid weight (detection limit, 0.5 μg/kg), while photomirex was not detected. The lowest mirex values were seen in fish from the most remote locations, suggesting that atmospheric transport of this compound was occurring (Muir et al. 1990).

Ninety percent of the mussels collected in 1985 at various points along the St. Lawrence River contained mirex at levels up to 1.6 μg/kg (ppb). The only source of mirex was contaminated particles entering the river from Lake Ontario; mussels collected from the Ottawa River, which does not receive its water from Lake Ontario, did not contain any mirex. The mirex concentrations in the mussels decreased with distance from the lake (Metcalfe and Charlton 1990).

Mirex concentrations were measured in 78 snapping turtles collected from 16 sites in southern Ontario, Canada, during 1988–1989 to evaluate the risk to human health (Hebert et al. 1993). Mean concentrations of mirex in the muscle tissue were below fish consumption guidelines for mirex (100 μg/kg [ppb]) and ranged from not detectable to 3.95 μg/kg (ppb). However, mirex concentrations in older turtles from some sites were as high as 9.3 μg/kg (ppb).

Freshwater fish sampled (as part of the U.S. Fish and Wildlife Service National Contaminant Biomonitoring Program) between 1980 and 1984 contained detectable concentrations of mirex. Mirex was detected in 18% of the 1980 samples (maximum concentration, 210 μg/kg [ppb]; mean concentration, 0.01 μg/g) and in 13% of the 1984 samples (maximum concentration, 440 μg/kg [ppb]; mean concentration, 10 μg/kg). The highest mirex concentrations were detected in whole fish taken from Lake

Ontario, the St. Lawrence River, and the southeastern United States, all areas where mirex had been manufactured or used (Schmitt et al. 1990). In the EPA National Study of Chemical Contaminants in Fish, mirex was detected at 38% of 362 sites sampled. The mean mirex concentration was 3.86 μg/kg (ppb) and the maximum concentration was 225 μg/kg (ppb). The highest concentrations of mirex were detected in fish collected in the Lake Ontario area of New York State (EPA 1992). In the EPA National Study of Chemical Contaminants in Lake Fish (EPA 2009a), mirex was detected in 2% of the 486 fish sampling locations for predator fish, with a maximum concentration of 9 ppb, and in 4.8% of the 395 sample locations for bottom dwelling fish, with a maximum concentration of 29 ppb.

Of oysters (*Crassostrea virginica*) sampled throughout the United States between 1965 and 1972 for the National Pesticide Monitoring Program, only those from South Carolina locations had detectable mirex residues (maximum concentration, 540 μg/kg [ppb]) with most residues being <38 μg/kg (ppb) (Butler 1973). Oysters taken from 49 sampling locations in the Gulf of Mexico for the NOAA Status and Trends Mussel Watch Program 1986–1987 were analyzed for mirex contamination (Sericano et al. 1990; Wade et al. 1988). Average mirex concentrations of 1.40 μg/kg (ppb) (range, <0.25–15.8 μg/kg) and 1.38 μg/kg (ppb) (range, <0.25–16.1) were found in oysters in 1986 and 1987, respectively (Sericano et al. 1990). The sampling sites represent the contaminant loading for the Gulf of Mexico estuaries removed from known point-sources of contamination (Wade et al. 1988).

Mirex was also detected in the muscle and liver tissues of seven species of aquatic and terrestrial mammals collected in areas of Alabama and Georgia that had been repeatedly treated with mirex to suppress fire ant populations from March 1973 through July 1976. At 6 months post-treatment, skunk and opossum muscle tissue contained the highest mean mirex concentrations of 3.50 and 1.5 1 μg/g (ppm), respectively (Hill and Dent 1985). Two years post-treatment, muscle residues declined in all species except the mink, which increased from 0.14 μg/g at 6 months post-treatment to a mean muscle residue of 0.28 μg/g at 1 year post-treatment and 0.53 μg/g at 2 years post-treatment.

Mirex was detected in the subcutaneous fat and breast muscle of 55 waterfowl collected in New York State during 1981 and 1982. Average mirex levels were 280 μg/kg (ppb) in fat and 2.0 μg/kg in breast muscle (Kim et al. 1985). Mirex was detected at a concentration of >500 μg/kg (ppb) in 24 of 164 samples of subcutaneous fat of six species of waterfowl (mallard, black duck, scaup, wood duck, bufflehead, and Canada goose) harvested by hunters in 1983–1984 (Foley 1992). Mirex was detected in fat samples from 5 of 26 goldeneyes shot by hunters in December 1988 in New York State; however, no quantitative information on mirex residues was provided (Swift et al. 1995). Gebauer and Weseloh

(1993) used farm-raised mallards as sentinels for accumulation of pollutants at three sites in southern Ontario, Canada. The sites included the Hamilton Harbor Confined Disposal Facility designated as an "Area of Concern" because of high pollutant concentrations of sediment; the Winona Sewage Lagoons, which contained high concentrations of metals; and Big Creek Marsh, which served as a reference area. The geometric mean concentrations of mirex detected in muscle tissue at each site were 7.1 μg/kg (ppb) at the Hamilton Harbor site after 115 days; 0.07 μg/kg at the sewage lagoon site after 112 days; and 0.14 μg/kg at the reference site after 30 days.

Mirex residues were detected in food samples analyzed as part of the FDA Pesticide Residue Monitoring Studies conducted from 1978 to 1982 of 49,877 food samples and from 1982 to 1986 of 49,055 food samples; however, the frequency of detection was unspecified but was <1 and 2% respectively (Yess 1988; Yess et al. 1991). Mirex was not detected in 27,065 samples of food collected in 10 state food laboratories from 1988 and 1989 (Minyard and Roberts 1991). Mirex was also not detected in domestically produced or imported foods sampled as part of the FDA Pesticide Residue Monitoring Study during 1989 (FDA 1990), was detected (at <1% occurrence) in foods sampled in 1990 (FDA 1991), and was not detected in foods sampled in 1991 (FDA 1992) and 1992 (FDA 1993) or in the most recent (2017) survey (FDA 2019).

***Chlordecone.*** Because releases of chlordecone from its production and use ceased in the late 1970s, current chlordecone residues in various biological organisms are generally lower than those reported during its peak production years (1974–1975). Releases of chlordecone from the manufacturing plant in Hopewell, Virginia, severely contaminated the James River estuary in Virginia from 1966 through 1975. In 1977, 12 years after production of chlordecone began and 2 years after it ceased, average chlordecone concentrations in various biological organisms in the estuary were as follows (Nichols 1990): phytoplankton, 1.30 μg/g; zooplankton, 4.80 μg/g; freshwater fish, 2.50 μg/g; migratory fish, 0.40 μg/g; and benthic fauna (molluscs), 1.50 μg/g. Considerable variations in chlordecone concentrations detected in fish species in the James River were in part associated with different life histories and residence times of each species in the estuary (Huggett and Bender 1980). Freshwater species that were permanent residents in the upper estuary exhibited the highest range in tissue residues varying from <0.1 μg/g (ppm) for channel catfish to >2 μg/g for largemouth bass. Residues in marine fish increased with length of exposure time in the James River. American shad that inhabited the estuary only briefly showed average chlordecone residues of <0.1 μg/g. Longer-term residents that spent 6–9 months in the estuary, such as spot and croaker, contained 1 μg/g. Concentrations in resident estuarine species ranged from 0.7 μg/g for the bay anchovy to 2.7 μg/g for white perch.

Dredging of the James River in Virginia increased the chlordecone levels in resident clams (*Rangia cuneata*). The river has contaminated sediments containing up to 3.5 μg/g (ppm) chlordecone. Prior to the 2-week dredging period, chlordecone concentrations in the water column ranged from nondetectable to 0.02 μg/L (ppb); background concentrations in the clams ranged from 0.06 to 0.14 μg/g. During the dredging, body burdens of chlordecone in clams increased by 0.01–0.04 μg/g (ppm). Two weeks after the dredging was completed, residues in the clams had not returned to predredging levels (Lunsford et al. 1987).

In addition to the James River area, chlordecone residues of 0.025 and 0.23 mg/kg (ppm) were detected in trout and suckers, respectively, collected from Spring Creek 18 miles downstream of the Nease Chemical Plant in Pennsylvania (EPA 1978a). This plant produced small quantities of chlordecone from 1966 to 1974 (Epstein 1978).

Because chlordecone contamination of the James River in Virginia and Spring Creek in Pennsylvania represented relatively isolated incidents resulting from industrial negligence and because the compound was not used extensively on agricultural crops in the United States, monitoring for this compound has not been included as part of the U.S. Fish and Wildlife Service National Contaminant Biomonitoring Program (Schmitt et al. 1990), the EPA National Study of Chemical Residues in Fish (EPA 1992), EPA National Study of Chemical Contaminants in Lake Fish (EPA 2009a).

Chlordecone residues were detected in the FDA Pesticide Residue Monitoring Studies of 49,877 food samples from 1978 to 1982 and of 49,055 food samples from 1982 to 1986; however, the frequency of detection was unspecified but was less than 1 and 2%, respectively (Yess 1988; Yess et al. 1991). Chlordecone was also detected in 1 of 27,065 samples of food collected from 10 state laboratories during 1988 and 1989 (Minyard and Roberts 1991). Chlordecone was not detected in any domestically produced or imported foods analyzed as part of the FDA Pesticide Residue Monitoring Studies during 1988–1989, 1989–1990, 1990–1991, and 1991–1992 (FDA 1990, 1991, 1992, 1993) or in the most recent (2017) survey of imported foods (FDA 2019).

## 5.6 GENERAL POPULATION EXPOSURE

***Mirex.*** Mirex has not been produced since 1976 and has not been used in the United States since 1977, when all registered uses of the product were canceled. The potential for exposure of the general

population, therefore, is relatively small and should continue to diminish over time. Members of the general population may be exposed to low concentrations of mirex primarily through consumption of contaminated food stuffs, in particular contaminated fish and shellfish from Lake Ontario, the St. Lawrence River, and Spring Creek in Pennsylvania, which were all contaminated by industrial discharges, and areas of the southern United States that were extensively treated with mirex for fire ant control. No dietary intake estimates are available (FDA 1990, 1991, 1992) since mirex has been so infrequently found in foodstuffs in recent years. Mirex exposure from drinking water has not been found to constitute significant human exposure since mirex is relatively insoluble in water and rapidly adsorbs to sediment (EPA 1978a).

Mirex has been detected in the general U.S. population. The National Human Monitoring Program for Pesticides detected mirex at low frequencies in human adipose tissue collected nationwide. In 1972, mirex was detected in 0.05% of all samples and in 1973, mirex was detected in 0.09% of all samples; however, by 1974, the percentage of positive samples had increased to 0.11% (Kutz et al. 1979). Mirex was detected in 13% of samples collected as part of the 1982 National Adipose Tissue Survey (EPA 1986b). Concentrations of mirex ranged from 0.008 to 0.39 μg/g (ppm) (mean concentration 0.025 μg/g). Further analysis of adipose tissue samples collected as part of the 1982 National Adipose Tissue Survey failed to detect mirex in any tissues from children (newborn infants to 14-year-olds); however, tissue samples from adults 15–44 and ≥45 years old were found to contain mirex residues. The greatest concentrations (values not provided) for 15–44-year-old adults were found in the Northeast and South Atlantic States, while the greatest concentrations for >45-year-old adults were found in the West South Central States and Northeast States (Phillips and Birchard 1991).

In a survey of human adipose tissue from residents of southwestern Ontario between 1976 and 1979, mirex was detected in 32.8% of the samples at mean concentrations of <0.01 mg/kg (ppm). In 1980–1981, it was detected in more samples (64.8%) at greater concentrations (mean concentration, 0.04 mg/kg); however, in 1983–1984, it was detected in only 6.2% of the samples at an average concentration of 0.06 mg/kg. Adipose tissue collected from 13 infants during this time contained <0.01 mg/kg mirex, except for one sample that contained 0.02 mg/kg. Mirex was not detected in any blood or human milk samples collected for this survey (Frank et al. 1988). A 1985 nationwide study of chlorinated hydrocarbons in the adipose tissue of Canadians found mirex to be present in all 108 samples collected nationwide at a mean concentration of 7 ng/g (ppb) (maximum concentration, 72 ng/g). The high rate of detection was a result of improved analytical procedures and lower limits of detection than those used in earlier studies. Residues were evenly distributed throughout the country and did not differ

significantly between the sexes or by age (Mes et al. 1990). In a 1990–1991 survey of human adipose tissue from residents of British Columbia, Canada, mirex was detected at a minimum, mean, and maximum concentration of 1.15, 6.10, and 33.3 ng/g (ppb) lipid, respectively (Teschke et al. 1993).

Mirex residues in human blood serum were measured as part of the National Report on Human Exposure to Environmental Chemicals. In the Second National Health and Nutrition Examination Survey (NHANES II), conducted between 1976 and 1980. Of the 4,038 samples analyzed, mirex concentrations ranged from not detectable to detected but below quantifiable levels (10 µg/L [ppb]) (Stehr-Green 1989). In the Fourth National Report on Human Exposures to Environmental Chemicals (CDC 2019), mirex levels in serum (lipid adjusted) were reported according to various age groups, gender, and race/ethnicity. The results are presented in Tables 5-4, 5-5, 5-6, and 5-7.

Mirex was detected (mean detection limit 3 pg/g [ppt]) in 62% of 412 breast milk samples collected from women in all Canadian provinces (Mes et al. 1993). The mean, median, and maximum mirex concentrations were 0.14, 0.08, and 6.56 ng/g (ppb), respectively, in whole milk and 4.2, 2.3, and 124.5 ng/g, respectively, in milk fat. In previous studies, mirex residues were not detected. None of the 1,436 human milk samples collected in the United States in the late 1970s as part of the National Human Milk Study contained identifiable levels of mirex (Savage et al. 1981). A similar national study of nursing mothers in Canada (Mes et al. 1986) also failed to detect mirex in any human milk samples. The high rate of detection in the Mes et al. (1993) study was a result of improved analytical procedures and lower limits of detection.

An analysis of potential human exposure to contaminants in drinking water and foods was conducted in Ontario, Canada, in 1980. Mirex was detected only in edible fish taken from Toronto Harbor on Lake Ontario. The average mirex concentrations were 0.001 mg/kg (ppm) wet weight for white sucker, 0.01 mg/kg wet weight for rainbow trout, and 0.033 mg/kg wet weight for northern pike. Estimated human exposure levels, based on an average fish consumption of 0.53 kg/year for each fish species, were 0.0005 for white sucker, 0.005 for rainbow trout, and 0.017 mg/year for northern pike, respectively (Davies 1990).

Mirex is no longer manufactured, formulated, or used in the United States. Therefore, there is currently no occupational exposure to this chemical associated with its production or application as a pesticide. Current occupational exposure is most likely to occur for workers employed at waste disposal sites or those engaged in remediation activities including removal of soils and sediments contaminated with

**Table 5-4. Geometric Mean and Selected Percentiles of Mirex (Lipid Adjusted) Serum Concentrations (in ng/g of Lipid or Parts per Billion on a Lipid-Weight Basis) for the U.S. Population from the National Health and Nutrition Examination Survey (NHANES) 1999–2004**

| | Survey years[a] | Geometric mean (95% CI) | Selected percentiles (95% CI) | | | | Sample size |
|---|---|---|---|---|---|---|---|
| | | | 50th | 75th | 90th | 95th | |
| Total | 1999–2000 | *[b] | <LOD | <LOD | <LOD | <LOD | 1,853 |
| | 2001–2002 | * | <LOD | <LOD | 15.8 (<LOD–73.7) | 57.1 (13.2–230) | 2,257 |
| | 2003–2004 | * | <LOD | <LOD | 8.40 (<LOD–13.0) | 13.2 (7.90–29.6) | 1,951 |
| Age group | | | | | | | |
| 12–19 years | 1999–2000 | * | <LOD | <LOD | <LOD | <LOD | 659 |
| | 2001–2002 | * | <LOD | <LOD | <LOD | <LOD | 728 |
| | 2003–2004 | * | <LOD | <LOD | <LOD | <LOD | 592 |
| ≥20 years | 1999–2000 | * | <LOD | <LOD | <LOD | <LOD | 1,194 |
| | 2001–2002 | * | <LOD | <LOD | 19.6 (<LOD–108) | 71.0 (14.6–305) | 1,529 |
| | 2003–2004 | * | <LOD | <LOD | 9.10 (<LOD–15.6) | 15.4 (8.10–37.1) | 1,359 |
| Gender | | | | | | | |
| Males | 1999–2000 | * | <LOD | <LOD | <LOD | <LOD | 887 |
| | 2001–2002 | * | <LOD | <LOD | 16.1 (<LOD–65.6) | 50.8 (12.3–225) | 1,052 |
| | 2003–2004 | * | <LOD | <LOD | 9.70 (<LOD–15.4) | 15.5 (9.70–24.4) | 949 |
| Females | 1999–2000 | * | <LOD | <LOD | <LOD | <LOD | 966 |
| | 2001–2002 | * | <LOD | <LOD | 15.0 (<LOD–108) | 63.0 (12.0–374) | 1,205 |
| | 2003–2004 | * | <LOD | <LOD | <LOD | 11.6 (<LOD–31.3) | 1,002 |
| Race/ethnicity | | | | | | | |
| Mexican Americans | 1999–2000 | * | <LOD | <LOD | <LOD | <LOD | 617 |
| | 2001–2002 | * | <LOD | <LOD | <LOD | <LOD | 548 |
| | 2003–2004 | * | <LOD | <LOD | <LOD | <LOD | 459 |
| Non-Hispanic blacks | 1999–2000 | * | <LOD | <LOD | 15.5 (<LOD-42.2) | 39.5 (<LOD–115) | 398 |
| | 2001–2002 | * | <LOD | 13.7 (<LOD–47.3) | 51.3 (15.4–230) | 153 (30.5–425) | 500 |
| | 2003–2004 | * | <LOD | <LOD | 18.1 (8.70–40.8) | 40.3 (15.5–82.7) | 484 |

**Table 5-4. Geometric Mean and Selected Percentiles of Mirex (Lipid Adjusted) Serum Concentrations (in ng/g of Lipid or Parts per Billion on a Lipid-Weight Basis) for the U.S. Population from the National Health and Nutrition Examination Survey (NHANES) 1999–2004**

| | Survey years[a] | Geometric mean (95% CI) | Selected percentiles (95% CI) | | | | Sample size |
|---|---|---|---|---|---|---|---|
| | | | 50th | 75th | 90th | 95th | |
| Non-Hispanic whites | 1999–2000 | * | <LOD | <LOD | <LOD | <LOD | 688 |
| | 2001–2002 | * | <LOD | <LOD | 15.1 (<LOD–104) | 66.7 (12.5–291) | 1,049 |
| | 2003–2004 | * | <LOD | <LOD | <LOD | 11.6 (<LOD–23.4) | 884 |

[a]The limit of detection for survey years 1999–2000, 2001–2002, and 2003–2004 were 14.6, 10.5, and 7.8 ng/g, respectively.
[b]Not calculated: proportion of results below limit of detection was too high to provide a valid result.

CI = confidence interval; LOD = limit of detection

Source: CDC 2019

**Table 5-5. Weighted Arithmetic Mean and Unadjusted Standard Error of Mirex (Lipid Adjusted) Pooled Serum Concentrations (in ng/g of Lipid or Parts per Billion on a Lipid-Weight Basis) for the U.S. Population from the National Health and Nutrition Examination Survey (NHANES) 2005–2010**

| Category | Age (years) | Survey years[a] | Weighted arithmetic mean[b] | Unadjusted standard error[c] | Number of pools[d] |
|---|---|---|---|---|---|
| Non-Hispanic white male | 12–19 | 2005–2006 | *[e] | * | 9 |
| | | 2007–2008 | * | * | 6 |
| | | 2009–2010 | * | * | 10 |
| | 20–39 | 2005–2006 | 3.88[f] | 2.18 | 12 |
| | | 2007–2008 | * | * | 15 |
| | | 2009–2010 | * | * | 17 |
| | 40–59 | 2005–2006 | 6.39[f] | 2.15 | 12 |
| | | 2007–2008 | 4.25 | 0.31 | 16 |
| | | 2009–2010 | 5.25 | 1.32 | 17 |
| | ≥60 | 2005–2006 | 5.32 | 0.61 | 15 |
| | | 2007–2008 | 6.36 | 1.34 | 23 |
| | | 2009–2010 | 4.89 | 0.44 | 21 |
| Non-Hispanic white female | 12–19 | 2005–2006 | * | * | 10 |
| | | 2007–2008 | * | * | 7 |
| | | 2009–2010 | * | * | 8 |
| | 20–39 | 2005–2006 | * | * | 16 |
| | | 2007–2008 | * | * | 13 |
| | | 2009–2010 | * | * | 19 |
| | 40–59 | 2005–2006 | 2.42 | 0.14 | 13 |
| | | 2007–2008 | 2.05 | 0.28 | 17 |
| | | 2009–2010 | 3.32 | 0.33 | 17 |
| | ≥60 | 2005–2006 | 3.51 | 0.24 | 17 |
| | | 2007–2008 | 3.90 | 0.39 | 21 |
| | | 2009–2010 | 4.42 | 0.4 | 22 |
| Non-Hispanic black male | 12–19 | 2005–2006 | * | * | 13 |
| | | 2007–2008 | * | * | 6 |
| | | 2009–2010 | * | * | 6 |
| | 20–39 | 2005–2006 | 2.68 | 0.59 | 6 |
| | | 2007–2008 | * | * | 6 |
| | | 2009–2010 | * | * | 7 |
| | 40–59 | 2005–2006 | 5.90 | 0.49 | 5 |
| | | 2007–2008 | 16.8[f] | 6.1 | 6 |
| | | 2009–2010 | 6.44 | 1.04 | 7 |
| | ≥60 | 2005–2006 | 27.2[f] | 10.1 | 5 |
| | | 2007–2008 | 13.9 | 2.1 | 8 |
| | | 2009–2010 | 14.2 | 4.1 | 9 |

**Table 5-5. Weighted Arithmetic Mean and Unadjusted Standard Error of Mirex (Lipid Adjusted) Pooled Serum Concentrations (in ng/g of Lipid or Parts per Billion on a Lipid-Weight Basis) for the U.S. Population from the National Health and Nutrition Examination Survey (NHANES) 2005–2010**

| Category | Age (years) | Survey years[a] | Weighted arithmetic mean[b] | Unadjusted standard error[c] | Number of pools[d] |
|---|---|---|---|---|---|
| Non-Hispanic black female | 12–19 | 2005–2006 | * | * | 14 |
| | | 2007–2008 | * | * | 5 |
| | | 2009–2010 | * | * | 6 |
| | 20–39 | 2005–2006 | 1.62 | 0.32 | 7 |
| | | 2007–2008 | * | * | 8 |
| | | 2009–2010 | * | * | 7 |
| | 40–59 | 2005–2006 | 5.92 | 0.65 | 7 |
| | | 2007–2008 | 5.42 | 1.21 | 8 |
| | | 2009–2010 | 5.03 | 0.84 | 7 |
| | ≥60 | 2005–2006 | 10.3 | 2.7 | 5 |
| | | 2007–2008 | 24.0[f] | 9.3 | 7 |
| | | 2009–2010 | 7.49 | 1.68 | 7 |
| Mexican American male | 12–19 | 2005–2006 | * | * | 11 |
| | | 2007–2008 | * | * | 6 |
| | | 2009–2010 | * | * | 8 |
| | 20–39 | 2005–2006 | * | * | 9 |
| | | 2007–2008 | * | * | 9 |
| | | 2009–2010 | * | * | 8 |
| | 40–59 | 2005–2006 | 2.66 | 0.74 | 4 |
| | | 2007–2008 | 4.37[f] | 1.38 | 6 |
| | | 2009–2010 | 3.08 | 0.83 | 8 |
| | ≥60 | 2005–2006 | 2.89 | 0.78 | 4 |
| | | 2007–2008 | 11.0[f] | 8.0 | 5 |
| | | 2009–2010 | 5.1 | 1.27 | 5 |

**Table 5-5. Weighted Arithmetic Mean and Unadjusted Standard Error of Mirex (Lipid Adjusted) Pooled Serum Concentrations (in ng/g of Lipid or Parts per Billion on a Lipid-Weight Basis) for the U.S. Population from the National Health and Nutrition Examination Survey (NHANES) 2005–2010**

| Category | Age (years) | Survey years[a] | Weighted arithmetic mean[b] | Unadjusted standard error[c] | Number of pools[d] |
|---|---|---|---|---|---|
| Mexican American female | 12–19 | 2005–2006 | * | * | 16 |
| | | 2007–2008 | * | * | 5 |
| | | 2009–2010 | * | * | 7 |
| | 20–39 | 2005–2006 | * | * | 9 |
| | | 2007–2008 | * | * | 8 |
| | | 2009–2010 | * | * | 10 |
| | 40–59 | 2005–2006 | 1.84 | 0.34 | 6 |
| | | 2007–2008 | 3.76[f] | 1.3 | 6 |
| | | 2009–2010 | * | * | 9 |
| | ≥60 | 2005–2006 | 2.84 | 0.37 | 3 |
| | | 2007–2008 | 2.59 | 0.49 | 5 |
| | | 2009–2010 | 4.04 | 0.97 | 6 |
| All Hispanic male | 12–19 | 2009–2010 | * | * | 11 |
| | 20–39 | 2009–2010 | * | * | 13 |
| | 40–59 | 2009–2010 | 4.58 | 1.27 | 13 |
| | ≥60 | 2009–2010 | 5.18 | 0.82 | 8 |
| All Hispanic female | 12–19 | 2009–2010 | * | * | 10 |
| | 20–39 | 2009–2010 | * | * | 14 |
| | 40–59 | 2009–2010 | * | * | 14 |
| | ≥60 | 2009–2010 | 4.13 | 0.55 | 11 |

[a]The limits of detection for survey years 2005–2006, 2007–2008, and 2009–2010 were 1.46, 1.4, and 2.19 ng/g, respectively.
[b]Weighted arithmetic means are not comparable to weighted geometric means.
[c]Unadjusted standard errors do not incorporate survey design effects.
[d]Each pool was composed of serum from eight persons.
[e]Not calculated: proportion of results below limit of detection was too high to provide a valid result.
[f]Standard error of the mean estimate is >30%.

CI = confidence interval; LOD = limit of detection

Source: CDC 2019

**Table 5-6. Geometric Mean and Selected Percentiles of Mirex (Whole Weight) Serum Concentrations (in ng/g of Serum or Parts per Billion) for the U.S. Population from the National Health and Nutrition Examination Survey (NHANES) 1999–2004**

| | Survey years[a] | Geometric mean (95% CI) | Selected percentiles (95% CI) | | | | Sample size |
|---|---|---|---|---|---|---|---|
| | | | 50th | 75th | 90th | 95th | |
| Total | 1999–2000 | *[b] | <LOD | <LOD | <LOD | <LOD | 1,853 |
| | 2001–2002 | * | <LOD | <LOD | 0.100 (<LOD–0.470) | 0.410 (0.080–1.73) | 2,257 |
| | 2003–2004 | * | <LOD | <LOD | 0.54 (<LOD–0.084) | 0.093 (0.052–0.170) | 1,951 |
| Age group | | | | | | | |
| 12–19 years | 1999–2000 | * | <LOD | <LOD | <LOD | <LOD | 659 |
| | 2001–2002 | * | <LOD | <LOD | <LOD | <LOD | 728 |
| | 2003–2004 | * | <LOD | <LOD | <LOD | <LOD | 592 |
| ≥20 years | 1999–2000 | * | <LOD | <LOD | <LOD | <LOD | 1,194 |
| | 2001–2002 | * | <LOD | <LOD | 0.140 (<LOD–0.690) | 0.470 (0.090–1.92) | 1,529 |
| | 2003–2004 | * | <LOD | <LOD | 0.059 (<LOD–0.102) | 0.106 (0.053–0.215) | 1,359 |
| Gender | | | | | | | |
| Males | 1999–2000 | * | <LOD | <LOD | <LOD | <LOD | 887 |
| | 2001–2002 | * | <LOD | <LOD | 0.110 (<LOD–0.470) | 0.370 (0.090–1.37) | 1,052 |
| | 2003–2004 | * | <LOD | <LOD | 0.064 (<LOD–0.106) | 0.108 (0.062–0.170) | 949 |
| Females | 1999–2000 | * | <LOD | <LOD | <LOD | <LOD | 966 |
| | 2001–2002 | * | <LOD | <LOD | 0.090 (<LOD–0.510) | 0.430 (0.070–1.79) | 1,205 |
| | 2003–2004 | * | <LOD | <LOD | <LOD | 0.077 (<LOD–0.170) | 1,002 |
| Race/ethnicity | | | | | | | |
| Mexican Americans | 1999–2000 | * | <LOD | <LOD | <LOD | <LOD | 617 |
| | 2001–2002 | * | <LOD | <LOD | <LOD | <LOD | 548 |
| | 2003–2004 | * | <LOD | <LOD | <LOD | <LOD | 459 |
| Non-Hispanic blacks | 1999–2000 | * | <LOD | <LOD | 0.090 (<LOD–0.220) | 0.220 (<LOD–0.450) | 398 |
| | 2001–2002 | * | <LOD | 0.090 (<LOD–0.240) | 0.310 (0.090–1.41) | 1.08 (0.170–3.02) | 500 |
| | 2003–2004 | * | <LOD | <LOD | 0.112 (0.055–0.268) | 0.256 (0.089–0.635) | 484 |

**Table 5-6. Geometric Mean and Selected Percentiles of Mirex (Whole Weight) Serum Concentrations (in ng/g of Serum or Parts per Billion) for the U.S. Population from the National Health and Nutrition Examination Survey (NHANES) 1999–2004**

| | Survey years[a] | Geometric mean (95% CI) | Selected percentiles (95% CI) | | | | Sample size |
|---|---|---|---|---|---|---|---|
| | | | 50th | 75th | 90th | 95th | |
| Non-Hispanic whites | 1999–2000 | * | <LOD | <LOD | <LOD | <LOD | 688 |
| | 2001–2002 | * | <LOD | <LOD | 0.100 (<LOD–0.610) | 0.450 (0.080–1.79) | 1,049 |
| | 2003–2004 | * | <LOD | <LOD | <LOD | 0.079 (<LOD–0.174) | 884 |

[a]The limit of detection for survey years 1999–2000, 2001–2002, and 2003–2004 were 14.6, 10.5, and 7.8 ng/g, respectively.
[b]Not calculated: proportion of results below limit of detection was too high to provide a valid result.

CI = confidence interval; LOD = limit of detection

Source: CDC 2019

**Table 5-7. Weighted Arithmetic Mean and Unadjusted Standard Error of Mirex (Whole Weight) Pooled Serum Concentrations (in ng/g of Serum or Parts per Billion) for the U.S. Population from the National Health and Nutrition Examination Survey (NHANES) 2005–2010**

| Category | Age (years) | Survey years[a] | Weighted arithmetic mean[b] | Unadjusted standard error[c] | Number of pools[d] |
|---|---|---|---|---|---|
| Non-Hispanic white male | 12–19 | 2005–2006 | *[e] | * | 9 |
| | | 2007–2008 | * | * | 6 |
| | | 2009–2010 | * | * | 10 |
| | 20–39 | 2005–2006 | 0.027[f] | 0.014 | 12 |
| | | 2007–2008 | * | * | 15 |
| | | 2009–2010 | * | * | 17 |
| | 40–59 | 2005–2006 | 0.048[f] | 0.016 | 12 |
| | | 2007–2008 | 0.031 | 0.003 | 16 |
| | | 2009–2010 | 0.034 | 0.008 | 17 |
| | ≥60 | 2005–2006 | 0.036 | 0.004 | 15 |
| | | 2007–2008 | 0.040 | 0.008 | 23 |
| | | 2009–2010 | 0.030 | 0.003 | 21 |
| Non-Hispanic white female | 12–19 | 2005–2006 | * | * | 10 |
| | | 2007–2008 | * | * | 7 |
| | | 2009–2010 | * | * | 8 |
| | 20–39 | 2005–2006 | * | * | 16 |
| | | 2007–2008 | * | * | 13 |
| | | 2009–2010 | * | * | 19 |
| | 40–59 | 2005–2006 | 0.018 | 0.002 | 13 |
| | | 2007–2008 | 0.014 | 0.002 | 17 |
| | | 2009–2010 | 0.021 | 0.002 | 17 |
| | ≥60 | 2005–2006 | 0.026 | 0.002 | 17 |
| | | 2007–2008 | 0.026 | 0.003 | 21 |
| | | 2009–2010 | 0.027 | 0.002 | 22 |
| Non-Hispanic black male | 12–19 | 2005–2006 | * | * | 13 |
| | | 2007–2008 | * | * | 6 |
| | | 2009–2010 | * | * | 6 |
| | 20–39 | 2005–2006 | 0.016 | 0.004 | 6 |
| | | 2007–2008 | * | * | 6 |
| | | 2009–2010 | * | * | 7 |
| | 40–59 | 2005–2006 | 0.038 | 0.003 | 5 |
| | | 2007–2008 | 0.109[f] | 0.04 | 6 |
| | | 2009–2010 | 0.041 | 0.008 | 7 |
| | ≥60 | 2005–2006 | 0.168[f] | 0.062 | 5 |
| | | 2007–2008 | 0.084 | 0.012 | 8 |
| | | 2009–2010 | 0.076 | 0.023 | 9 |

**Table 5-7. Weighted Arithmetic Mean and Unadjusted Standard Error of Mirex (Whole Weight) Pooled Serum Concentrations (in ng/g of Serum or Parts per Billion) for the U.S. Population from the National Health and Nutrition Examination Survey (NHANES) 2005–2010**

| Category | Age (years) | Survey years[a] | Weighted arithmetic mean[b] | Unadjusted standard error[c] | Number of pools[d] |
|---|---|---|---|---|---|
| Non-Hispanic black female | 12–19 | 2005–2006 | * | * | 14 |
| | | 2007–2008 | * | * | 5 |
| | | 2009–2010 | * | * | 6 |
| | 20–39 | 2005–2006 | 0.009 | 0.002 | 7 |
| | | 2007–2008 | * | * | 8 |
| | | 2009–2010 | * | * | 7 |
| | 40–59 | 2005–2006 | 0.038 | 0.004 | 7 |
| | | 2007–2008 | 0.032 | 0.008 | 8 |
| | | 2009–2010 | 0.028 | 0.005 | 7 |
| | ≥60 | 2005–2006 | 0.067 | 0.016 | 5 |
| | | 2007–2008 | 0.146[f] | 0.057 | 7 |
| | | 2009–2010 | 0.043 | 0.01 | 7 |
| Mexican American male | 12–19 | 2005–2006 | * | * | 11 |
| | | 2007–2008 | * | * | 6 |
| | | 2009–2010 | * | * | 8 |
| | 20–39 | 2005–2006 | * | * | 9 |
| | | 2007–2008 | * | * | 9 |
| | | 2009–2010 | * | * | 8 |
| | 40–59 | 2005–2006 | 0.022[f] | 0.007 | 4 |
| | | 2007–2008 | 0.031[f] | 0.01 | 6 |
| | | 2009–2010 | 0.020 | 0.005 | 8 |
| | ≥60 | 2005–2006 | 0.022[f] | 0.008 | 4 |
| | | 2007–2008 | 0.074[f] | 0.052 | 5 |
| | | 2009–2010 | 0.031 | 0.008 | 5 |
| Mexican American female | 12–19 | 2005–2006 | * | * | 16 |
| | | 2007–2008 | * | * | 5 |
| | | 2009–2010 | * | * | 7 |
| | 20–39 | 2005–2006 | * | * | 9 |
| | | 2007–2008 | * | * | 8 |
| | | 2009–2010 | * | * | 10 |
| | 40–59 | 2005–2006 | 0.014 | 0.003 | 6 |
| | | 2007–2008 | 0.024[f] | 0.008 | 6 |
| | | 2009–2010 | * | * | 9 |
| | ≥60 | 2005–2006 | 0.022 | 0.005 | 3 |
| | | 2007–2008 | 0.018 | 0.003 | 5 |
| | | 2009–2010 | 0.023 | 0.005 | 6 |
| All Hispanic male | 12–19 | 2009–2010 | * | * | 11 |
| | 20–39 | 2009–2010 | * | * | 13 |
| | 40–59 | 2009–2010 | 0.031 | 0.008 | 13 |
| | ≥60 | 2009–2010 | 0.032 | 0.005 | 8 |

**Table 5-7. Weighted Arithmetic Mean and Unadjusted Standard Error of Mirex (Whole Weight) Pooled Serum Concentrations (in ng/g of Serum or Parts per Billion) for the U.S. Population from the National Health and Nutrition Examination Survey (NHANES) 2005–2010**

| Category | Age (years) | Survey years[a] | Weighted arithmetic mean[b] | Unadjusted standard error[c] | Number of pools[d] |
|---|---|---|---|---|---|
| All | 12–19 | 2009–2010 | * | * | 10 |
| Hispanic | 20–39 | 2009–2010 | * | * | 14 |
| female | 40–59 | 2009–2010 | * | * | 14 |
| | ≥60 | 2009–2010 | 0.027 | 0.003 | 11 |

[a]The limits of detection for survey years 2005–2006 and 2007–2008 were 1.46 and 1.4 ng/g, respectively.
[b]Weighted arithmetic means are not comparable to weighted geometric means.
[c]Unadjusted standard errors do not incorporate survey design effects.
[d]Each pool was composed of serum from eight persons.
[e]Not calculated: proportion of results below limit of detection was too high to provide a valid result.
[f]Standard error of the mean estimate is >30%.

CI = confidence interval; LOD = limit of detection

Source: CDC 2019

mirex. There is a slight possibility of exposure for workers involved in dredging activities (e.g., sediment remediation work performed by the Corps of Engineers).

***Chlordecone.*** Chlordecone has not been produced since 1975 or used in the United States since 1978 when all registered uses of the product were canceled. The potential for exposure of the general population, therefore, is relatively small and should continue to diminish over time. Members of the general population may be exposed to low concentrations of chlordecone primarily through consumption of contaminated foodstuffs, in particular contaminated fish and shellfish from the James River in Virginia. No dietary intake estimates are available (FDA 1990, 1991, 1992) since chlordecone has been so infrequently found in foodstuffs in recent years. Chlordecone exposure from drinking water has not been found to constitute significant human exposure since chlordecone is relatively insoluble in water and rapidly adsorbs to sediment (EPA 1978a).

No information was located for the general population on chlordecone concentrations in human adipose tissue or blood as this compound was not included in any major national study (e.g., National Human Adipose Study). Chlordecone was detected in 9 of 298 samples of human milk collected in the southern United States; however, the detection limit was relatively high (1 μg/kg) (EPA 1978a).

With regard to occupational exposures, chlordecone was detected in blood samples from workers at the Life Sciences Products Company in Hopewell, Virginia. Chlordecone levels in the blood of 32 workers at the manufacturing plant ranged from 0.165 to 26.0 µg/mL (ppm) (Epstein 1978). The mean blood level of workers exhibiting symptoms of nervousness and tremors was 8.48 µg/mL, compared to a mean of 1.57 µg/mL in workers exhibiting no symptoms (Epstein 1978). In another occupational study, Cannon et al. (1978) reported maximum chlordecone blood levels in workers at the Hopewell facility of 11.8 µg/mL. Chlordecone blood levels of workers who reported illness averaged 2.53 µg/mL, while blood levels for workers reporting no illness averaged 0.6 µg/mL.

In 1975, when chlordecone was still being produced, over half of the workers at a manufacturing plant developed clinical illness characterized by nervousness, tremor, weight loss, opsoclonus, pleuritic and joint pain, and oligospermia (Cannon et al. 1978). During the years of production, chlordecone was also detected in family members of the plant workers at the Life Sciences Products Company in Hopewell, Virginia. Although half of the workers at the plant had clinical signs of chlordecone poisoning, such signs were detected in only two family members who washed contaminated clothes (Cannon et al. 1978). Another study also found higher chlordecone levels in members of chlordecone workers' families compared with families of workers at other local industries or other community residents (Taylor et al. 1978). Such illness could have been mitigated by appropriate occupational health measures that would prevent the transport of contaminated materials from the workplace, such as not bringing work clothes home (Knishkowy and Baker 1986).

Current occupational exposure is most likely to occur for workers employed at waste disposal sites or those engaged in remediation activities associated with the clean-up or removal of soils or sediments that are contaminated with chlordecone.

## 5.7 POPULATIONS WITH POTENTIALLY HIGH EXPOSURES

A susceptible population will exhibit a different or enhanced response to mirex and chlordecone than will most persons exposed to the same level of mirex or chlordecone in the environment. Reasons include genetic make-up, developmental stage, age, health and nutritional status (including dietary habits that may increase susceptibility, such as inconsistent diets or nutritional deficiencies), and substance exposure history (including smoking). These parameters result in decreased function of the detoxification and excretory processes (mainly hepatic, renal, and respiratory) or the pre-existing compromised function of target organs (including effects or clearance rates and any resulting endproduct metabolites). For these

reasons, the elderly with declining organ function and the youngest of the population with immature and developing organs are generally expected be more vulnerable to toxic substances than healthy adults.

Review of the literature regarding toxic effects of mirex and chlordecone did not reveal any human populations that are known to be unusually sensitive to mirex or chlordecone. However, based on knowledge of the toxicities of mirex and chlordecone, some populations can be identified that may demonstrate unusual sensitivity to these chemicals. Those with potentially high sensitivity to mirex include the very young. Those with potentially high sensitivity to chlordecone include juvenile and elderly person and persons being treated with some classes of antidepressants that affect serotonin or the anticonvulsant, diphenylhydantoin.

In experimental animals, mirex administered within the week after birth causes a high incidence of cataracts and other lesions of the lens (Chernoff et al. 1979a; Gaines and Kimbrough 1970; Rogers and Grabowski 1984; Scotti et al. 1981). These effects were observed whether the neonatal animals received mirex through the milk of lactating dams or directly by gavage. Although it is unclear whether the lens of humans also undergoes a similar period of susceptibility, the possibility exists that newborn children may also develop cataracts if exposed to mirex shortly after birth.

Studies in rats have demonstrated that certain treatments exacerbate the tremors associated with chlordecone exposure. These include pretreatment with the anticonvulsant, diphenylhydantoin (Hong et al. 1986; Tilson et al. 1985, 1986), and treatment with the nonselective serotonergic receptor agonist, quipazine (Gerhart et al. 1983). Therefore, persons being treated with diphenylhydantoin for epilepsy or quipazine for depression may be likely to experience more severe tremors upon exposure to high levels of chlordecone. Extrapolating from the effects seen in animals with quipazine, it might be likely that persons taking the prescription drug Prozac®, a SSRI used to treat depression, will also experience more severe tremors. Furthermore, the elderly may be a susceptible population because serotonin metabolism is increased during aging (Walker and Fishman 1991).

Studies in animals have also shown that juvenile animals experience a higher death rate than adults following exposure to chlordecone at equivalent mg/kg doses (Huber 1965). No explanation was given for these findings, but similar sensitivities may exist in children. Furthermore, although inhibition of Na+-K+ATPase, Mg2+ATPase, and Ca2+ATPase activities have not been definitively shown to be the mechanism underlying chlordecone toxicity, sufficient evidence exists to suggest that their inhibition may be involved in a number of adverse effects. Neonatal rats have shown a greater inhibition of these

enzymes than adult rats (Jinna et al. 1989). This provides additional support for the suggestion that infants and young children may represent a susceptible population to the toxic effects of chlordecone.

In contrast, a study of developing postnatal rats has shown that the young may be less susceptible to at least one of the toxic effects of chlordecone. Young and adolescent rats show less potentiation of carbon tetrachloride toxicity than adult rats (Cai and Mehendale 1993). This may be due to a combination of incomplete development of the microsomal enzyme systems and a higher level of hepatic regenerating activity in the very young rats. In adolescent rats (35 and 45 days old), the microsomal enzyme activity is comparable to adult levels, but the level of damage is still less than in adult rats (60 days old). This may be due to that fact that hepatic regenerating activity remained higher in the adolescents than in the adults.

In studies performed by Sobel and coworkers (Sobel et al. 2005, 2006; Wang et al. 2008), chronic exposure of systemic lupus erythematosus-prone female (NZB x NZW) F1 mice to chlordecone via subcutaneously-implanted pellets significantly shortened the time to onset of elevated autoantibody titers and renal disease in a dose-related manner. These effects were not seen in nonlupus-prone BALB/c mice. These results indicate that humans with lupus may be particularly sensitive to chlordecone toxicity.

Members of the general population who currently have potentially high exposures to mirex include recreational and subsistence fishers who may consume large quantities of fish and shellfish from waterbodies with mirex contamination, hunters who consume game species that may be contaminated with mirex, populations living near sites where mirex was manufactured or waste disposal sites contaminated with mirex, or populations living in areas where mirex was used extensively for fire ant control.

Mirex contamination has triggered the issuance of several human health advisories nationwide. In 1993, mirex was identified as the causative pollutant in eight fish consumption advisories in three different states (Ohio, New York, and Pennsylvania) (EPA 1993). In 2019, New York still had mirex fish advisories in six waterbodies (Lake Ontario, Niagara River downstream of Niagara Falls, Irondequoit Bay, Oswego River, Salmon River, and St. Lawrence River) (NYS 2019).

Persons living in areas where mirex has been used for fire ant control or near where it was manufactured may be at increased risk of exposure. Human tissue samples (unspecified) taken from 186 people at sites treated with mirex over the previous 10 years had mirex residues in the range of <1–1.32 μg/g (ppm) (mean concentration, 0.38 μg/g) (EPA 1980). A 1975–1976 survey of 624 human adipose tissue samples

from subjects living in eight southern states where mirex had been used for fire ant control indicated that 10.2% of the population in the area had detectable levels of mirex at a geometric mean concentration of 0.286 μg/g (ppm). Populations living in two states, Texas and North Carolina, had no detectable mirex residues in their tissues, whereas 51.1% of the samples from populations in Mississippi had detectable levels (mean concentration, 0.290 μg/g) (Kutz et al. 1985). Mirex was detected in human adipose tissue samples from residents of northeast Louisiana during the late 1970s (Greer et al. 1980). Concentrations of mirex in adipose tissue collected during surgery and during postmortem examinations ranged from 0.01 to 0.60 μg/g (ppm) with a mean mirex concentration of 0.14 μg/g. Human adipose tissue samples from northeastern Louisiana, an agricultural area, contained detectable amounts of mirex in 20 of 22 samples in 1977 at a mean concentration of approximately 0.15 μg/g (ppm), 10 of 10 samples in 1980 at a mean concentration of 0.25 μg/g, and only 2 of 10 samples in 1984 at a mean concentration of 0.15 μg/g (Holt et al. 1986).

A comparison of mirex residues in adipose tissue samples collected between 1979 and 1981 from residents of Kingston, Ontario (a city located on Lake Ontario), and residents of Ottawa, Ontario, indicated that persons living in Kingston had significantly higher mirex and photomirex residues than those in Ottawa (27 and 9 ng/g [ppb], respectively, in Kingston versus 11 and 6 ng/g, respectively, in Ottawa). Males from Kingston had significantly higher levels of mirex (38 ng/g) than females from the area (12 ng/g); this gender difference was not explained or seen in the Ottawa samples (Williams et al. 1984). A subsequent 1984 study examined mirex levels in six additional cities on the Canadian portion of Lake Ontario. The overall mean mirex residue in human adipose tissue was 11±13 ng/g (ppb) (males, 12±15 ng/g; females, 9.6±10 ng/g) (Williams et al. 1988).

Mirex levels in the blood of pregnant women in Jackson, Mississippi, and the Mississippi Delta area where mirex was extensively used were correlated with the health of the infants they bore. The mean mirex level in maternal blood was 0.54 μg/L (ppb) for 106 samples; however, mirex levels in the blood of the infants were not correlated with differences in gestation times, Apgar score, or other problems at birth. Only three children with neurological problems had mothers with pesticide levels, including mirex, above the mean levels (Lloyd et al. 1974).

In 1977, mirex was detected in human milk and colostrum samples of women living in upstate New York. Milk from women in Oswego and Rochester, areas adjacent to Lake Ontario (known to be contaminated with mirex), was compared with milk from women in Albany (considered to be free from mirex contamination). Mean mirex concentrations from women in each area were as follows: 0.057 ng/g in

colostrum (n=24) and 0.07 ng/g in milk (n=6), Albany; 0.51 ng/g in colostrum (n=18) and 0.120 ng/g in milk (n=16), Oswego; and 0.035 ng/g in colostrum (n=4) and 0.162 ng/g in milk (n=6), Rochester. Only 2 of the 28 milk samples (both from Oswego) were below the detection limit of 0.01 ng/g (ppb), while 16 of 24 colostrum samples in Albany, 10 of 18 colostrum samples from Oswego, and 2 of 4 colostrum samples from Rochester were below the detection limit. None of the women reported eating freshwater fish, a possible source of the mirex contamination (Bush et al. 1983).

Members of the general population currently having potentially higher exposure to chlordecone include recreational and subsistence fishers who may consume large quantities of fish and shellfish from waterbodies with chlordecone contamination, populations living near sites where chlordecone was manufactured, or waste disposal sites contaminated with chlordecone.

Chlordecone contamination has triggered the issuance of one human health advisory. As of September 1993, chlordecone was identified as the causative pollutant in an advisory issued by the State of Virginia for the 113 miles of the James River Estuary. The advisory extends from Richmond, Virginia, downstream to the Hampton-Norfolk Bridge Tunnel including all tributaries to the James River (EPA 1993).

The only data on chlordecone residues in populations living near a production site are historic and were collected several decades ago. The EPA initiated a community survey in August 1975 shortly after production of chlordecone was halted to determine chlordecone levels in blood of persons living in the vicinity of the Hopewell manufacturing plant. Two hundred nine community residents, none of whom had ever been employed at the Allied Chemical plant or Life Sciences Products Company (LSPC) were surveyed. Chlordecone blood levels were <5 ppb in 39% of residents living 0.25 miles south of the LSPC plant, in 7.7% of residents living 0.25 miles north of the LSPC plant, in 5.9% of residents living 0.5 miles from the site, in 2.6% of residents living 0.75 miles from the site, and in 3.3% of residents living 1 mile from the site. Chlordecone blood levels were approximately linear as a function of proximity to the LSPC site (Epstein 1978). No additional information was located on current chlordecone levels in residents of the Hopewell, Virginia, area.