

CHAPTER 6. ADEQUACY OF THE DATABASE

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

Data needs are defined as substance-specific informational needs that, if met, would reduce the uncertainties of human health risk assessment. This definition should not be interpreted to mean that all data needs discussed in this section must be filled. In the future, the identified data needs will be evaluated and prioritized, and a substance-specific research agenda will be proposed.

6.1 INFORMATION ON HEALTH EFFECTS

Studies evaluating the health effects of inhalation, oral, and dermal exposure of humans and animals to aldrin or dieldrin that are discussed in Chapter 2 are summarized in Figures 6-1 and 6-2, respectively. The purpose of these figures is to illustrate the information concerning the health effects of aldrin and dieldrin. The number of human and animal studies examining each endpoint is indicated regardless of whether an effect was found and the quality of the study or studies.

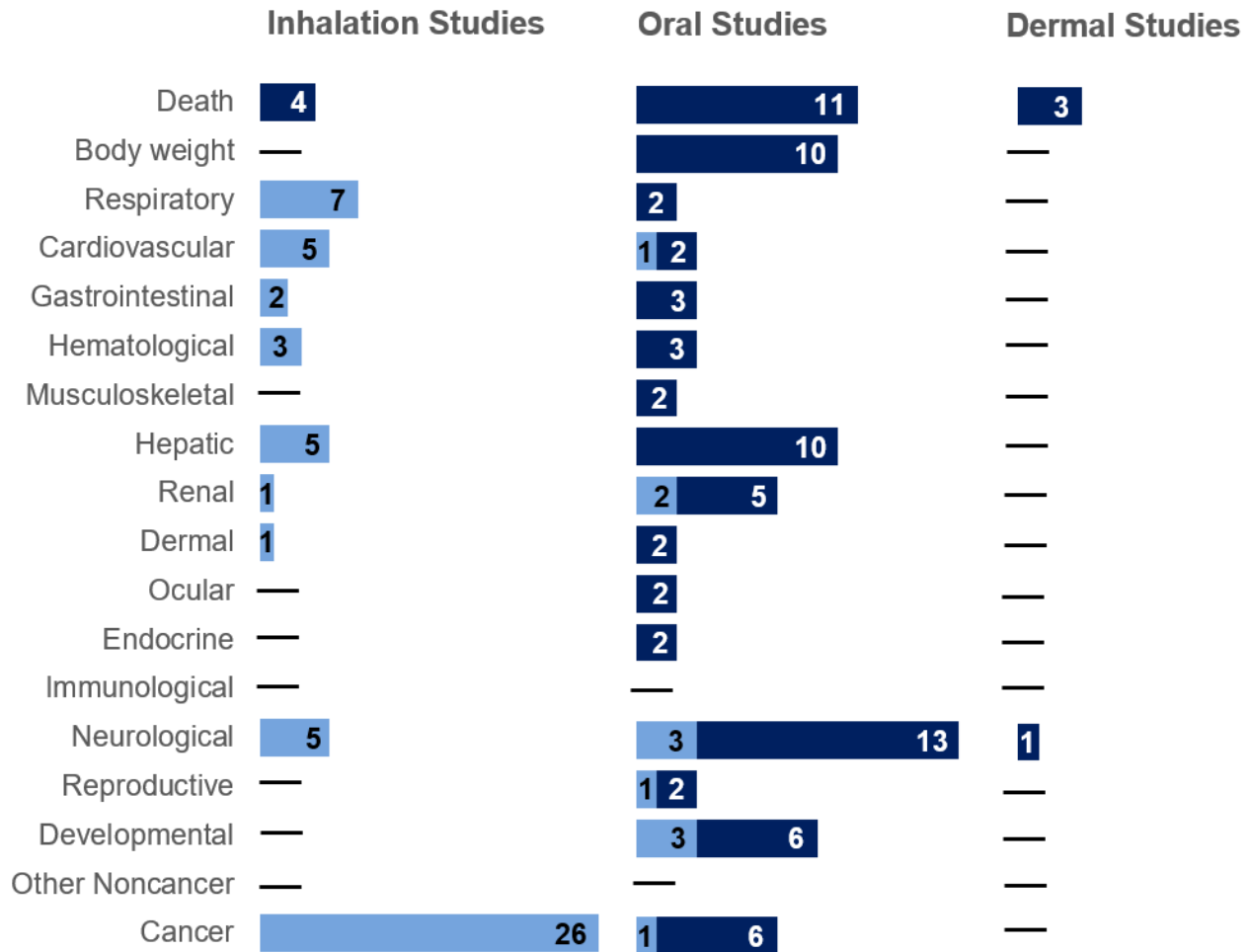
6.2 IDENTIFICATION OF DATA NEEDS

Missing information in Figure 6-1 and/or Figure 6-2 should not be interpreted as a “data need.” A data need, as defined in ATSDR’s *Decision Guide for Identifying Substance-Specific Data Needs Related to Toxicological Profiles* (ATSDR 1989), is substance-specific information necessary to conduct comprehensive public health assessments. Generally, ATSDR defines a data gap more broadly as any substance-specific information missing from the scientific literature.

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Figure 6-1. Summary of Existing Health Effects Studies on Aldrin By Route and Endpoint*

Potential body weight, hepatic, and neurological effects were the most studied endpoints
 The majority of the studies examined oral exposure in **animals** (versus **humans**)

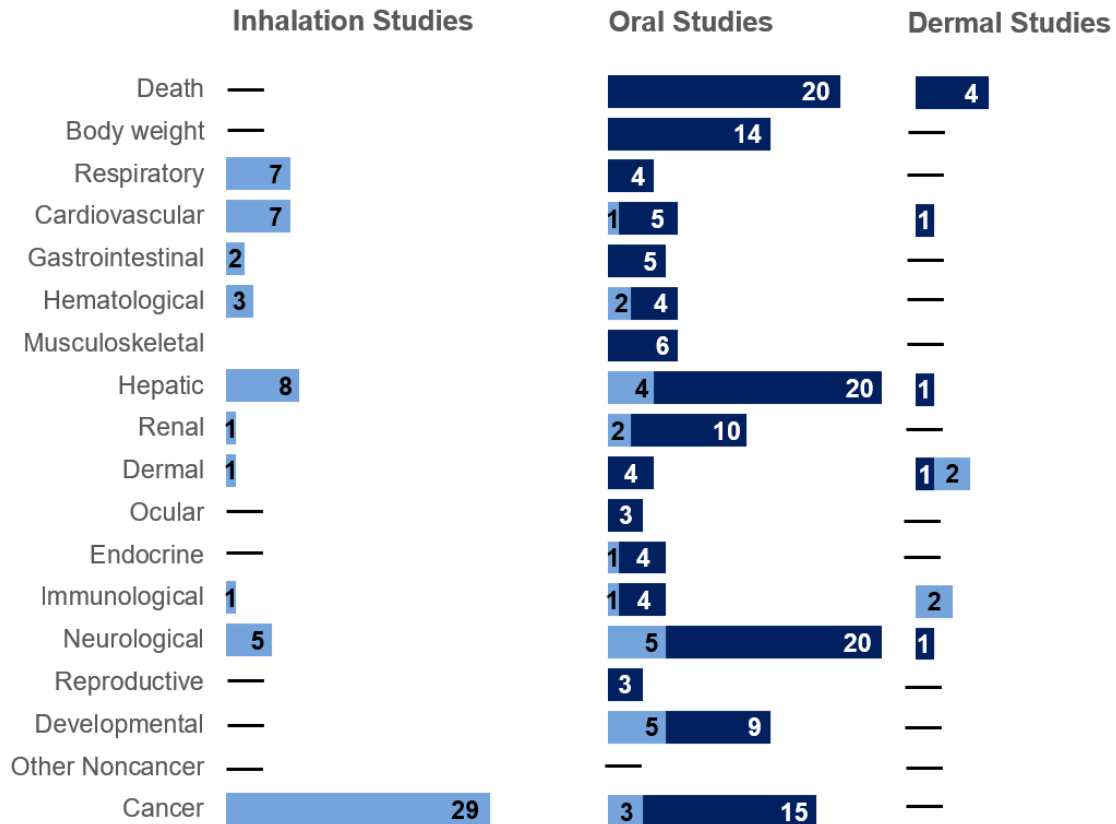


*Includes studies discussed in Chapter 2; the number of studies include those finding no effect; more than one endpoint may have been evaluated in a study. Occupational studies likely involved multiple exposure routes, but are presented as inhalation studies (the most likely major route of exposure).

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Figure 6-2. Summary of Existing Health Effects Studies on Dieldrin By Route and Endpoint*

Potential hepatic and neurological effects were the most studied endpoints
 The majority of the studies examined oral exposure in **animals** (versus **humans**)



*Includes studies discussed in Chapter 2; the number of studies include those finding no effect; more than one endpoint may have been evaluated in a study. Occupational studies likely involved multiple exposure routes, but are presented as inhalation studies (the most likely major route of exposure).

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Acute-Duration MRLs. No quantitative exposure-response human data are available regarding acute-duration inhalation, oral, or dermal exposure to aldrin or dieldrin.

Available acute-duration inhalation data from experimental animals are limited to findings of death and respiratory effects (mucous membrane irritation) among rats, mice, rabbits, and cats acutely exposed to aldrin vapor generated by sublimating aldrin at 200°C (Treon et al. 1957). Inhalation exposure to aldrin or dieldrin is not likely because both substances were banned for pesticidal usage between 1974 and 1987. Although lack of data for the inhalation exposure route represents a data gap, additional studies regarding the effects of inhaled aldrin or dieldrin do not appear necessary.

The database was considered adequate for derivation of an acute-duration oral MRL for aldrin. Additional animal studies do not appear necessary.

An acute-duration oral MRL was not derived for dieldrin. Additional animal studies providing dose-response data may provide information to derive an acute-duration oral MRL for dieldrin.

Intermediate-Duration MRLs. No quantitative exposure-response human data are available regarding intermediate-duration inhalation, oral, or dermal exposure to aldrin or dieldrin.

No intermediate-duration inhalation data were available for experimental animals. Although lack of data for the inhalation exposure route represents a data gap, additional studies regarding the effects of inhaled aldrin or dieldrin do not appear necessary.

An intermediate-duration oral MRL was not derived for aldrin due to lack of appropriate effect levels. A 3-generation study identified the lowest LOAEL (a serious LOAEL of 0.26 mg/kg/day) for 3.2-fold increased mortality of F1a pups in the absence of an identified NOAEL (Treon et al. 1954a). It is not appropriate to derive an intermediate-duration oral MRL for aldrin because the lowest level tested (0.26 mg/kg/day in the 3-generation study) represents a serious LOAEL in the absence of an identified NOAEL. Given that aldrin is no longer approved for use and is readily converted to dieldrin in the environment, the general public is not likely to be exposed to aldrin by the oral route. It does not appear necessary to conduct additional intermediate-duration oral studies of aldrin in experimental animals.

The intermediate-duration database was considered adequate for derivation of an intermediate-duration oral MRL for dieldrin. Additional animal studies do not appear necessary.

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Chronic-Duration MRLs. No quantitative exposure-response human data are available regarding chronic-duration inhalation, oral, or dermal exposure to aldrin or dieldrin.

No chronic-duration inhalation data were available for experimental animals. Although lack of data for the inhalation exposure route represents a data gap, additional studies regarding the effects of inhaled aldrin or dieldrin do not appear necessary.

A chronic-duration oral MRL has been derived for aldrin. Additional animal studies do not appear necessary.

A chronic-duration oral has been derived for dieldrin. Additional animal studies do not appear necessary.

Health Effects.

Hepatic. Available human data mainly concern evaluation of serum liver enzyme levels in workers exposed to aldrin and/or dieldrin during manufacture and/or use of these pesticides. Slightly increased serum ALT and AST levels were reported in one study of pesticide-exposed workers (Morgan and Lin 1978). However, there was no evidence of exposure-related hepatic effects in other studies (de Jong 1991; Hoogendam et al. 1965; Hunter et al. 1972; Jager 1970; Morgan and Roan 1974; van Sittert and de Jong 1987; Warnick and Carter 1972). No adverse hepatic effects were observed in a study of healthy male subjects who consumed dieldrin at up to 0.0003 mg/kg/day for 18 months in a study designed to evaluate dieldrin kinetics (Hunter and Robinson 1967). Liver dysfunction has been reported in cases of inadvertent or intentional ingestion of relatively large amounts of dieldrin (Black 1974; Garrettson and Curley 1969). Aldrin and dieldrin have not been manufactured in the United States since 1974 and all uses were banned by 1987. In the environment, aldrin is readily converted to dieldrin and dieldrin has persisted and entered some food products. Populations with detectable blood dieldrin levels should be monitored, although it is not likely that the general population would be exposed to dieldrin at levels high enough to cause adverse hepatic effects.

The hepatotoxicity of orally-administered aldrin or dieldrin has been reported in a number of experimental animal species (Ahmed et al. 1986; Fitzhugh et al. 1964; Goel et al. 1988; Harr et al. 1970; Kitselman 1953; Kohli et al. 1977; Shakoory et al. 1982; Thorpe and Walker 1973;

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Treon et al. 1951a, 1955; Walker et al. 1969; Wright et al. 1972, 1978). Additional animal studies do not appear necessary.

Renal. The only available report of aldrin- or dieldrin-related renal effects was clinical chemistry evidence of renal damage in a case of intentional ingestion of an estimated 25.6 mg aldrin/kg (Spiotta 1951). Populations with detectable blood dieldrin levels should be monitored, although it is not likely that the general population would be exposed to dieldrin at levels high enough to cause adverse renal effects.

Adverse renal effects have been reported in studies of rats and dogs administered aldrin or dieldrin orally (Ahmed et al. 1986; Bandyopadhyay et al. 1982b; Deichmann et al. 1967; Fitzhugh et al. 1964; Harr et al. 1970; Kitselman 1953; Reuber 1980; Treon et al. 1955). There were no signs of adverse renal effects in chronic-duration oral studies of rats, mice, or dogs administered aldrin or dieldrin orally at doses in the range of 0.05–4.2 mg/kg/day (NCI 1978a; Treon et al. 1951a; Walker et al. 1969). Additional animal studies do not appear necessary.

Neurological. Excitation of the central nervous system is the principal effect reported in occupational studies of workers employed in either the application or manufacture of aldrin or dieldrin (Avar and Czegledi-Janko 1970; Hoogendam et al. 1965; Jager 1970; Kazantzis et al. 1964; Kazantzis et al. 1964; Patel and Rao 1958). Other central nervous system symptoms reported by workers involved in the manufacture or application of aldrin and/or dieldrin included headaches, dizziness, hyperirritability, general malaise, nausea and vomiting, anorexia, muscle twitching, and myoclonic jerking (Jager 1970; Kazantzis et al. 1964; Patel and Rao 1958). Inadvertent or intentional ingestion of relatively large amounts of aldrin or dieldrin have resulted in a variety of central nervous system signs and symptoms (Black 1974; Garrettson and Curley 1969; Gupta 1975; Spiotta 1951). Populations with detectable blood dieldrin levels should be monitored, although it is not likely that the general population would be exposed to dieldrin at levels high enough to cause adverse neurological effects.

Neurological endpoints have been assessed in multiple experimental animal species following acute- intermediate- or chronic-duration oral exposure of experimental animals to aldrin or dieldrin (Burt 1975; et al. 1989; Carlson and Rosellini 1987; Kitselman 1953; NCI 1978a, 1978b; Smith et al. 1976; Treon et al. 1951b; Wagner and Greene 1978; Walker et al. 1969; Woolley et al. 1985). Additional animal studies do not appear necessary.

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Reproductive. Limited information was located regarding aldrin or dieldrin exposure-related reproductive effects in humans. Populations with detectable blood dieldrin levels should be monitored, although it is not likely that the general population would be exposed to dieldrin at levels high enough to cause adverse reproductive effects.

Several animal studies were designed to evaluate potential aldrin or dieldrin exposure-related reproductive effects (Dean et al. 1975; Deichmann et al. 1971; Epstein et al. 1972; Good and Ware 1969; Harr et al. 1970; Keplinger et al. 1970; Treon et al. 1954a; Virgo and Bellward 1975). Decreased fertility was reported in some studies (Keplinger et al. 1970; Treon et al. 1954a; Virgo and Bellward 1975). Delayed estrus, reduced libido, lack of mammary function and development, and an increased number of stillbirths were reported in dogs orally administered aldrin for 14 months prior to mating (Deichmann et al. 1971). Studies examining the mechanisms of action would be useful in evaluating the human relevance of the reproductive effects observed in laboratory animals.

Developmental. No studies were located regarding aldrin or dieldrin treatment-related developmental effects in humans. Developmental effects such as increased postnatal mortality and external and skeletal malformations/anomalies have been observed in animal studies. Populations with detectable blood dieldrin levels should be monitored, although it is not likely that the general population would be exposed to dieldrin at levels high enough to cause adverse developmental effects.

Epidemiology and Human Dosimetry Studies. A variety of human studies are available. Several investigators reported cases of accidental or intentional poisonings (Black 1974; Garrettson and Curley 1969; Hoogendam et al. 1965; Kazantzis et al. 1964; Patel and Rao 1958; Spiotta 1951). Two cohorts of workers in the manufacture of aldrin and/or dieldrin have been followed (Amoateng-Adjepong et al. 1995; Brown 1992; de Jong 1991; Ditraglia et al. 1981; Swaen et al. 2002; van Amelsvoort et al. 2009). Other studies evaluated health effects in workers involved with application of aldrin and/or dieldrin (Hoogendam et al. 1965; Jager 1970; Morgan and Lin 1978; Morgan et al. 1980; Sandifer et al. 1981; van Raalte 1977; van Sittert and de Jong 1987; Versteeg and Jager 1973; Warnick and Carter 1972). A number of studies evaluated possible associations between self-reported use of aldrin and/or dieldrin and selected health outcomes (Alavanja et al. 2014; Bonner et al. 2017; Brown et al. 1990; Cantor et al. 1992; Clary and Ritz 2003; Dennis et al. 2010; De Roos et al. 2003; Engel et al. 2005; Flower et al. 2004;

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Koutros et al. 2013a, 2013b; Lee et al. 2004a, 2004b; Louis et al. 2017; McDuffie et al. 2001; Pahwa et al. 2011; Schroeder et al. 2001).

Exposures in the case reports are virtually all oral, whereas exposures in the epidemiological studies are mainly inhalation and dermal, with very slight potential for accidental oral intake. Additional follow-up of cohorts from previously conducted epidemiological studies would be the best approach for obtaining additional human data. Locating new populations for future epidemiological studies is likely to be difficult because aldrin and dieldrin have not been manufactured in the United States since 1974 and all uses were banned by 1987.

Biomarkers of Exposure and Effect. Exposure to aldrin and dieldrin is currently measured almost exclusively by determining the level of dieldrin in the blood (Jager 1970). This measure is specific for both aldrin and dieldrin. However, because aldrin is rapidly converted to dieldrin in the body (Wong and Terriere 1965), it is impossible to determine which of the two substances caused the blood levels of dieldrin to rise. Because dieldrin has a long half-life of elimination in humans (Hunter and Robinson 1967; Hunter et al. 1969; Jager 1970), measurement of dieldrin levels in the blood does not give any information about whether an acute-, intermediate-, or chronic-term exposure has occurred, whether such exposures have occurred recently, or whether a substantial period of time has elapsed since exposure occurred. The sensitivity of this biomarker of exposure appears to be sufficient to measure background levels in the population; thus, no new biomarkers of exposure appear to be needed at this time.

The central nervous system excitation resulting from aldrin or dieldrin exposure can be monitored, to a great extent, by monitoring EEG changes (Hoogendam et al. 1962, 1965; Jager 1970). Characteristic changes include bilateral synchronous spikes, spike and wave complexes, and slow theta and delta waves (Avar and Czeglédi-Janko 1970; Garrettson and Curley 1969; Hoogendam et al. 1962, 1965; Jager 1970; Kazantzis et al. 1964; Spiotta 1951). However, similar changes may be recorded in cases of central nervous system excitation caused by other agents. Thus, this measure is not specific for aldrin- or dieldrin-induced neurotoxicity. Blood levels of dieldrin have been correlated with adverse neurological effects caused by aldrin and dieldrin (Brown et al. 1964; Jager 1970). Such a measurement may also be used to monitor for adverse neurotoxic effects caused by these agents. Also, as understanding of the fundamental mechanism by which aldrin and dieldrin cause central nervous system excitation evolves, tests may be developed to specifically monitor underlying neurological changes caused by aldrin and dieldrin. No tests specific for aldrin- or dieldrin-induced toxic effects on the liver or kidney exist; however, standard liver and kidney function tests should be able to identify the hepatic or renal toxicity

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that is produced. Microsomal enzyme induction may be measured by determining parameters such as urinary levels of D-glucaric acid and the ratio of urinary 6- β -hydroxycortisol to 17-hydroxycorticosteroids. However, these tests are not specific for aldrin or dieldrin. Immune suppression of the type produced by aldrin or dieldrin may be detected by challenge with a T lymphocyte-dependent antigen; however, this test also is not specific for aldrin or dieldrin.

Absorption, Distribution, Metabolism, and Excretion. Human and animal data demonstrate that aldrin and dieldrin are absorbed after inhalation, oral, or dermal exposure (Feldmann and Maibach 1974; Graham et al. 1987; Hayes 1974; Heath and Vandekar 1964; Hunter and Robinson 1967; Hunter et al. 1969; Mehendale and El-Bassiouni 1975; Stacey and Tatum 1985). Quantitative data on the absorption of aldrin and dieldrin in humans and animals following exposure via all routes are limited. Animal studies indicate that aldrin and dieldrin are absorbed rather quickly and that the amount absorbed is proportional to the dose applied for the oral and dermal routes (Graham et al. 1987; Heath and Vandekar 1964; Iatropoulos et al. 1975). Because of the limited number of absorption studies for all three routes in general, it would be helpful to have additional quantitative data in animals that might serve as a basis for estimates of absorption in humans.

Data exist regarding distribution after oral administration of aldrin or dieldrin (Adeshina and Todd 1990; Ahmad et al. 1988; Deichmann et al. 1968; de Vlieger et al. 1968; Hayes 1974; Holt et al. 1986; Hunter and Robinson 1967, 1968; Hunter et al. 1969; Iatropoulos et al. 1975). These studies indicate that dieldrin is distributed in the blood to adipose tissue, brain, and liver tissues, and is then redistributed primarily to fat. Concentrations of dieldrin have been shown to increase in a dose-related manner in blood and adipose tissues of humans and eventually reach a steady state (Hunter and Robinson 1967; Hunter et al. 1969). Kinetic studies in rats and dogs support these findings and provide further information on steady-state kinetics following repeated dosing (Baron and Walton 1971; Davison 1973; Ludwig et al. 1964; Walker et al. 1969). Because data are sufficient regarding distribution following oral exposure to aldrin or dieldrin, no more studies are needed.

Comparative Toxicokinetics. Numerous studies using a variety of animal species indicate that the kinetics of aldrin and dieldrin differ across species (Baldwin et al. 1972; Hutson 1976; Klein et al. 1968; Klevay 1970; Ludwig et al. 1964; Matthews et al. 1971; Müller et al. 1975). The differences are primarily quantitative. Although the kinetic data alone do not allow for the identification of target organs common to humans and animals, the distribution data, coupled with toxicity data, appear to suggest that target organs are similar. Interspecies differences and sex-related differences in rats and mice have been

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observed for the metabolism and excretion of aldrin and dieldrin. These interspecies differences, coupled with a lack of data across different routes, indicate that it may be difficult to compare the kinetics of aldrin or dieldrin in animals with that in humans. Further studies across several species and via all three exposure routes would be useful in determining similarities and differences between humans and animals.

Children's Susceptibility. Limited reports of adverse effects in aldrin- or dieldrin-exposed children (Garrettson and Curley 1969; Gupta 1975) indicate similar signs and symptoms to those in adults. Limited animal data indicate that young animals may respond to aldrin or dieldrin differently than adult animals (Buck and Van Note 1968; Lu et al. 1965), but there is no conclusive evidence to suggest that young animals are more susceptible than older ones. Further studies that evaluate a number of different endpoints in young as well as older organisms would provide valuable information.

No information was located concerning whether the developmental process is altered in humans exposed to aldrin or dieldrin either prenatally or postnatally. Studies in animals have provided conflicting evidence regarding developmental malformations and anomalies (Chernoff et al. 1975; Dix et al. 1977; Ottolenghi et al. 1974), and further well-conducted research would be helpful to clarify this issue. Although animal studies suggest that aldrin and dieldrin may be disruptive of reproductive hormone levels in males and weakly estrogenic in females, additional well-designed studies are needed to clarify the developmental significance of these findings.

No data were located concerning whether pharmacokinetics of aldrin or dieldrin in children are different from adults. Although dieldrin has been detected in human placenta, amniotic fluid, fetal blood, and breast milk (Polishuk et al. 1977a; Schechter et al. 1989a), additional quantitative studies in animals would provide valuable information. There are no PBPK models for aldrin or dieldrin in either adults or children. There is no information to evaluate whether absorption, distribution, metabolism, or excretion of aldrin or dieldrin in children might be different than in adults.

There are no biomarkers of exposure or effect that have been validated in children. There are no data on interactions of aldrin or dieldrin with other chemicals in children, and extremely limited data in adults which are inadequate to determine whether the same effects will be observed in children. There are no pediatric-specific methods to reduce peak absorption of aldrin or dieldrin following exposure, or to reduce body burden, or to interfere with mechanisms of action for aldrin or dieldrin.

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Physical and Chemical Properties. The physical and chemical properties of aldrin and dieldrin are sufficiently well defined to allow assessments of their environmental fate (Budavari et al. 2001; Clayton and Clayton 1994; Guerin and Kennedy 1992; Hayes 1982; NLM 2020a, 2020b; NIOSH 1997; Verschueren 2001). No additional information is needed.

Production, Import/Export, Use, Release, and Disposal. The risk for exposure of the general population to substantial levels of aldrin or dieldrin is quite low. Aldrin and dieldrin have not been produced in the United States since 1974, nor is there any indication that U.S. production of either of these two chemicals will resume (EPA 1990a). Aldrin has not been imported into the United States since 1985 (EPA 1986a). No information was available regarding exports of aldrin or dieldrin, nor was information available regarding the amount of these insecticides currently stockpiled in the United States. Information regarding stockpile levels of aldrin and dieldrin would prove useful.

Currently, all uses of aldrin and dieldrin have been canceled (EPA 1990a). However, due to the persistence of dieldrin in the environment, the likelihood of its bioconcentration, and the former widespread use of both aldrin and dieldrin, these agents are still found at low levels in foods such as root crops and meat and dairy products. Concentrations of dieldrin are significantly higher than aldrin residues due to the high rate of conversion of aldrin to dieldrin in the environment and dieldrin's relative stability in environmental matrices.

The soil around dwellings that have been treated with termiticides containing aldrin and dieldrin is the environmental media most likely to be contaminated with significant quantities of aldrin and dieldrin. The air within treated homes may also contain elevated levels of these agents.

According to the Emergency Planning and Community Right-to-Know Act of 1986, 42 U.S.C. Section 11023, industries are required to submit chemical release and off-site transfer information to the EPA. TRI, which contains this information for 2018, became available in 2019. This database will be updated yearly and should provide a list of industrial production facilities and emissions.

Incineration and activated-carbon adsorption have >99% efficiencies as methods for disposing of aldrin or dieldrin (EPA 1981). However, no information is available regarding the amounts of aldrin or dieldrin disposed of by each method. Additional information on current disposal patterns would prove useful.

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Environmental Fate. Aldrin released to surface and shallow subsurface soils partitions to the atmosphere where it is transported (Caro and Taylor 1971; Elgar 1975; McLean et al. 1988). In deeper subsurface soils, aldrin generally is sorbed to soil particulates (McLean et al. 1988); under most environmental conditions, aldrin should not leach to groundwater (McLean et al. 1988). Aldrin is biotransformed to dieldrin in aerobic soils (Gannon and Bigger 1958; Gupta and Kavdia 1979). Additional information is needed on the transformations of aldrin in anaerobic soils and sediments.

Dieldrin sorbs to soils and sediments (Briggs 1981; Cliath and Spencer 1971). The compound also partitions to biota and slowly volatilizes from soils to the atmosphere (Nash 1983). Dieldrin is transported in the particulate phase in surface water runoff (Caro and Taylor 1971; Eye 1968; Hardee et al. 1964) and in the atmosphere (Baldwin et al. 1977). In deep subsurface soils, dieldrin is sorbed to particulates and does not leach to groundwater (Dobbs et al. 1989). The compound is persistent in environmental media, being resistant to biodegradation and abiotic transformation (Gannon and Bigger 1958; Jagnow and Haider 1972). Based on dieldrin's vapor pressure, it will exist in both the vapor and particulate phase in the atmosphere (Grayson and Fosbraey 1982). Vapor-phase dieldrin is expected to react with hydroxyl radicals, while particulate phase dieldrin will be removed from the atmosphere by wet and dry deposition. Information concerning the relative percentage of dieldrin that will exist in the particulate and vapor-phase in the environment would prove useful in predicting its atmospheric fate.

Bioavailability from Environmental Media. Limited available pharmacokinetic data indicate that the compounds are absorbed by humans following inhalation of contaminated air (Stacey and Tatum 1985). Absorption also occurs following oral and dermal exposures (Feldmann and Maibach 1974; Heath and Vandekar 1964; Hunter and Robinson 1967; Hunter et al. 1969; Iatropoulos et al. 1975). Additional information is needed on the absorption of the compounds following ingestion of contaminated drinking water and soils. This information would be useful in evaluating the importance of various routes of exposure to populations living in the vicinity of hazardous waste sites.

Food Chain Bioaccumulation. Aldrin and dieldrin are bioconcentrated by plants, animals, and aquatic organisms and biomagnified in aquatic and terrestrial food chains (Bhatnagar et al. 1988; Cole et al. 1976; Connell 1989; Donaldson et al. 1999; Metcalf et al. 1973; Sanborn and Yu 1973; Shannon 1977; Travis and Arms 1988). Food chain bioaccumulation appears to be a more important fate process for dieldrin, which is very persistent in nature, than for aldrin, which is rapidly converted to dieldrin (EPA 1980; Metcalf et al. 1973). No additional information is necessary.

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Exposure Levels in Environmental Media. Aldrin and dieldrin have historically been detected in ambient air (Hoff et al. 1996), surface water (EPA 1980; Stubin et al. 1996), drinking water (EPA 1980, 2001), soils (ATSDR 2007; Eisenreich et al. 1989; Kutz et al. 1976), sediments (Bergersen 1987; Staples et al. 1985), and foods (EPA 1985; FDA 2006; Hundley et al. 1988). Studies suggest that the concentrations of both aldrin and dieldrin in environmental matrices are decreasing (CalEPA 1995; MacIntosh et al. 1999; Miller et al. 1992). Aldrin and dieldrin have been identified at several of the hazardous waste sites that have been proposed for inclusion on the EPA NPL (ATSDR 2019). Estimates of dietary intake, which is believed to be the most important source of exposure for most members of the general population, are also available (FDA 1991, 1995). More recent monitoring data would be useful in more accurately predicting human exposure.

Exposure Levels in Humans. The presence of dieldrin in human blood and adipose tissue has been used as an indicator of exposure to aldrin and dieldrin (Brock et al. 1998; CDC 2019). The compounds have also been widely detected in human breast milk (Davies and Mes 1987; Quinsey et al. 1996; Savage et al. 1981; Takei et al. 1983). Additional information on the concentration of these compounds in the biological tissue and fluids of populations living in the vicinity of NPL sites would be helpful in assessing the extent to which these populations have been exposed to these compounds.

Exposures of Children. With the detection of dieldrin in drinking water (EPA 2001; Kolpin et al. 1997), studies that detail the exposure of infants fed formula prepared from tap water would prove helpful. More data are needed to properly assess aldrin and dieldrin exposure to children who live, play, or attend school near NPL sites and farmlands that have been treated with these pesticides. Information regarding the number of houses in the United States that have been treated with aldrin and dieldrin formulations in the past would be useful in determining the number of children that would be potentially exposed today. The stability of these compounds, especially dieldrin, suggests the possibility that they may be brought home by farm workers who work on farmlands previously treated with these compounds. More exposure studies that monitor aldrin and dieldrin exposure to children of farm workers would be useful for evaluating potential exposure.

6.3 ONGOING STUDIES

No ongoing research pertaining to aldrin was identified. The following ongoing research pertaining to dieldrin (Table 6-1) was identified in the National Institutes of Health (NIH) Research Portfolio Online Reporting Tools (RePORTER 2019).

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Table 6-1. Ongoing Studies on Dieldrin

Investigator	Affiliation	Research description	Sponsor
Alison Bernstein	Neurosciences, Schools of Medicine, Michigan State University	Dieldrin exposure and synucleinopathy	NIEHS
Alison Bernstein	Neurosciences, Schools of Medicine, Michigan State University	Epigenetic effects of adult and developmental exposure to Parkinsonian toxicants	NIEHS
Jonathan A. Doorn	Pharmacology, Schools of Pharmacy, University of Iowa	Pesticide-mediated generation of a toxic neurotransmitter metabolite	NIEHS
Anumantha Gounder Kanthasamy	Veterinary Sciences, Schools of Veterinary Medicine, Iowa State University	Novel mechanisms of pesticide-induced neurotoxicity	NIEHS
Mark N. Wu	Neurology, School of Medicine, Johns Hopkins University	Molecular and cellular mechanisms underlying the circadian timing of sleep (dieldrin included in the list of project terms)	NINDS

NIEHS = National Institute of Environmental Health Sciences; NINDS = National Institute of Neurological Disorders and Stroke

Source: RePORTER 2019