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 2,4-D 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 2,4-D.

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.

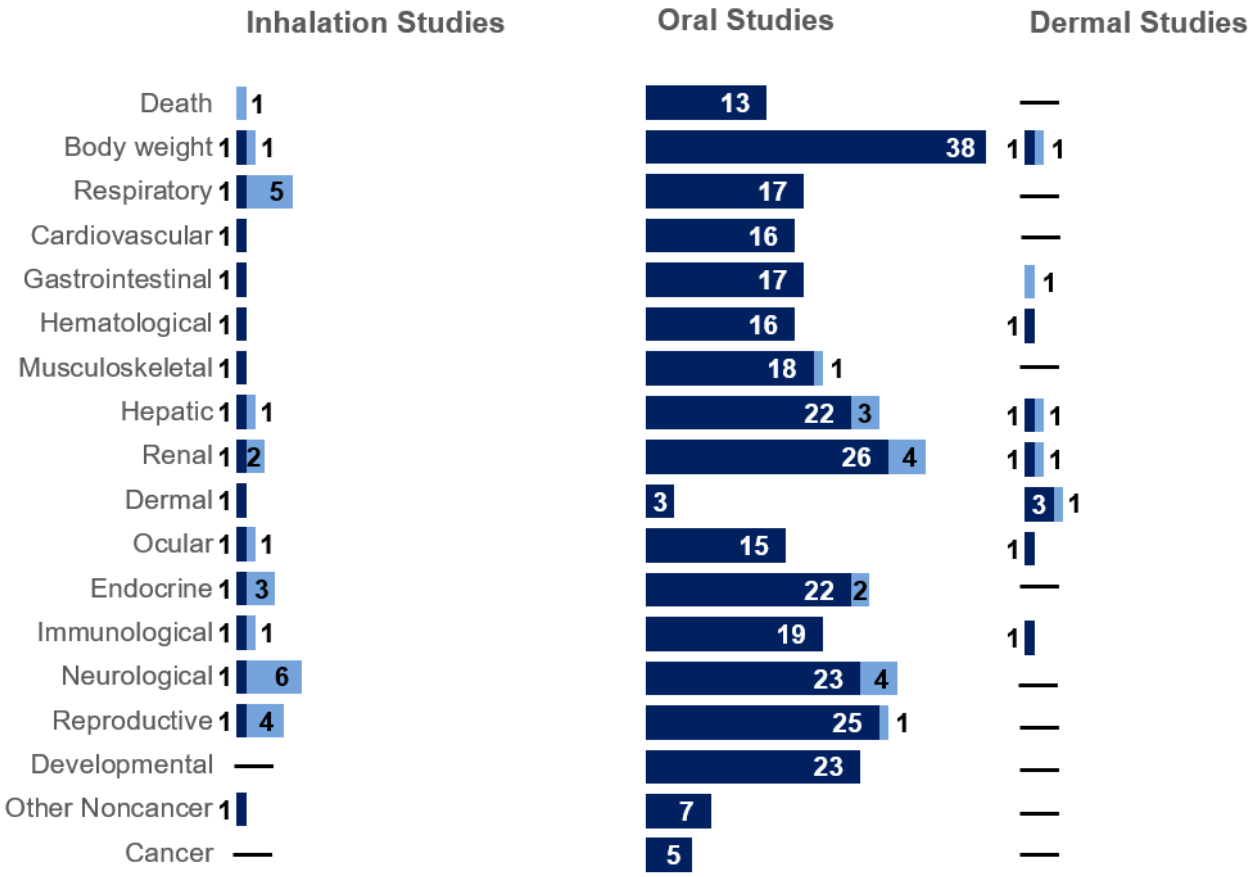
6.1 INFORMATION ON HEALTH EFFECTS

Studies evaluating the health effects of inhalation, oral, and dermal exposure of humans and animals to 2,4-D that are discussed in Chapter 2 are summarized in Figure 6-1. The purpose of this figure is to illustrate the information concerning the health effects of 2,4-D. 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.

Information regarding health effects in humans following exposure to 2,4-D comes from case reports of accidental or intentional ingestion of herbicide formulations containing 2,4-D, accidental skin contact with those products by farmers and professional residential applicators, and occupational exposure during manufacture, formulation, or packaging. Information is also available from exposure of the general population. Exposure to 2,4-D during use of products containing this chemical occurred predominantly by dermal contact, but inhalation may have also occurred if a product was sprayed. The general population can be exposed by dermal contact with surfaces treated with products containing 2,4-D, by consumption of contaminated water or food, and also in house dust. No reliable estimates of quantitative exposure could be obtained from case reports, but studies have estimated exposure from measurements of 2,4-D excreted in the urine. There is no evidence suggesting that the toxicity of 2,4-D is route-specific.

Figure 6-1. Summary of Existing Health Effects Studies on 2,4-D By Route and Endpoint*

Potential body weight and kidney 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; many studies examined more than 1 endpoint.

The database in animals is extensive. As seen in Figure 6-1, most studies in animals have been conducted by the oral route of exposure. There is more information regarding the health effects of 2,4-D following intermediate-duration exposure than regarding acute- or chronic-duration exposure.

People living near hazardous waste sites may be exposed to 2,4-D primarily via dermal contact with soil contaminated with 2,4-D, through ingestion of contaminated water, or through contaminated house dust.

6.2 IDENTIFICATION OF DATA NEEDS

Missing information in Figure 6-1 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.

Acute-Duration MRLs. No information was located regarding health effects in humans following inhalation exposure to 2,4-D. No acute-duration inhalation studies in animals were located. Published inhalation studies are needed for all exposure durations. There is information regarding health effects in humans following acute-duration oral exposure to 2,4-D from case reports of intentional or accidental ingestion of herbicide formulations containing 2,4-D. Effects that have been reported following oral exposure to high amounts of 2,4-D include including tachypnea, tachycardia, vomiting, leukocytosis, liver and kidney congestion in fatal cases, metabolic acidosis, and death (Dudley and Thapar 1972; Durakovic et al. 1992; Keller et al. 1994; Nielsen et al. 1965; Smith and Lewis 1987). Because these subjects were exposed to formulations containing 2,4-D along with other ingredients that may have contributed to the effects reported, these studies are inadequate for MRL derivation. Studies in animals provided information on lethality (Drill and Hiratzka 1953; Elo et al. 1988; Gorzinski et al. 1987; Hill and Carlisle 1947) and a wide range of endpoints including systemic effects (Dickow et al. 2000; Mattsson et al. 1997; Steiss et al. 1987), neurological effects (Mattsson et al. 1997; Steiss et al. 1987; Stürtz et al. 2008), reproductive effects (Dinamarca et al. 2007), and developmental effects (Charles et al. 2001; Chernoff et al. 1990; Collins and Williams 1971; Fofana et al. 2002; Kavlock et al. 1987; Schwetz et al. 1971). Long-term oral studies in animals suggest that the kidney is a target for 2,4-D toxicity; however, virtually no data on kidney effects were available in acute-duration studies. Therefore, an acute-duration study that examines the nature of the dose-response for kidney effects in rats or mice would be useful. Two case reports of humans acutely exposed to products containing 2,4-D by skin contact reported long-lasting

neurological alterations (Berkley and Magee 1963; Goldstein et al. 1959). A study in animals with controlled exposure to sublethal doses of 2,4-D would be useful to confirm or refute the reports in humans.

Intermediate-Duration MRLs. No studies of humans exposed to 2,4-D specifically for intermediate-duration periods (15–354 days) were located. However, it is likely that some subjects from studies mentioned below under Chronic-Duration MRLs were exposed for intermediate durations. An extensive database in animals exposed by the oral route provided information regarding systemic effects (Bortolozzi et al. 1999; Charles et al. 1996a, 1996b, 1996c; EPA 1984, 1985, 1986, 1987b, 1996; Gorzinski et al. 1987; Marty et al. 2013; Mattsson et al. 1997; Mazhar et al. 2014; Ozaki et al. 2001; Saghir et al. 2013a, 2013b; Stürtz et al. 2010; Troudi et al. 2012a, 2012b), neurological effects (Mattsson et al. 1997; Squibb et al. 1983), reproductive effects (Joshi et al. 2012), and developmental effects (Bortolozzi et al. 1999; EPA 1986; Hansen et al. 1971; Marty et al. 2013; Mazhar et al. 2014; Saghir et al. 2013a, 2013b; Stürtz et al. 2010; Troudi et al. 2012a, 2012b). These studies suggested that the kidney is a target for 2,4-D toxicity. Marty et al. (2013) reported the lowest LOAEL for kidney effects (45.3 mg/kg/day for proximal tubule degeneration); the result served as the basis for deriving an intermediate-duration oral MRL for 2,4-D. A single intermediate-duration inhalation study in animals was available for review (EPA 2008). This study examined a comprehensive number of endpoints in rats exposed to 2,4-D dusts for 28 days and established a LOAEL of 50 mg/m^3 2,4-D dusts for respiratory effects in rats; a NOAEL was not established. It would be valuable to conduct a study with lower exposure concentrations to establish a NOAEL for respiratory effects. The single study available was considered an insufficient database for MRL derivation. A report summarizing a 21-day dermal study in rabbits provided information mainly on systemic effects (EPA 1991a). A 13-week dermal study in rats or mice would be useful to examine the dose-response relationship for renal effects.

Chronic-Duration MRLs. There are numerous studies that provided information regarding exposure to 2,4-D and multiple health outcomes in humans (Beard et al. 2013; Beseler et al. 2006; Bloemen et al. 1993; Bond et al. 1988; Burns et al. 2001, 2011; Cantor et al. 1992; De Roos et al. 2003; Dhillon et al. 2008; Faustini et al. 1996; Eriksson et al. 2008; Flower et al. 2004; Fontana et al. 1998; Garry et al. 1996; Hardell and Eriksson 1999; Hardell et al. 1994; Hartge et al. 2005; Hoar et al. 1986; Hoppin et al. 2006a, 2006b, 2008; Kamel et al. 2007; Kluciński et al. 2001; Kogevinas et al. 1995; Lee et al. 2004; Lerda and Rizzi 1991; McDuffie et al. 2001; Miligi et al. 2006; Mills et al. 2005; Slager et al. 2009; Swan et al. 2003; Tanner et al. 2009; Weisenburger 1990; Weselak et al. 2007, 2008; Yang et al. 2014; Zahm et al. 1990). In these studies, exposure occurred predominantly by the dermal and inhalation routes of

exposure. Based on results from these and additional studies, there is no convincing evidence associating exposure to 2,4-D and adverse health effects in humans. As is not uncommon with epidemiological studies, limitations encountered in these studies include unreliable exposure assessment and simultaneous exposures to other chemicals. It seems prudent, however, to continue to monitor populations exposed to 2,4-D, such as pesticide applicators and manufacturers.

A limited number of chronic-duration studies in animals was available for review. These studies provided information on a wide range of endpoints in rats, mice, and dogs exposed orally to 2,4-D and suggested that the kidney is a target for 2,4-D toxicity in mice (Charles et al. 1996a; EPA 1987a, 1996; Hansen et al. 1971). Evidence of 2,4-D treatment-related degenerative/regenerative renal effects from the 2-year study in mice by Charles et al. (1996a) served as the basis for derivation of a chronic-duration oral MRL for 2,4-D. The chronic-duration oral studies also showed no evidence of carcinogenicity for 2,4-D in rats, mice, or dogs. Additional chronic-duration studies with 2,4-D do not seem necessary at this time.

Health Effects.

Genotoxicity. There are data regarding genetic effects in workers exposed to 2,4-D (i.e., Andreotti et al. 2015; Figgs et al. 2000; Garry et al. 2001; Holland et al. 2002; Hou et al. 2013), animals exposed *in vivo* (Amer and Aly 2001; Charles et al. 1999a, 1999b; Epstein et al. 1972; Kaya et al. 1999; Linnainmaa 1984; Madrigal-Bujaidar et al. 2001; Magnuson et al. 1977; Mustonen et al. 1989; Rasmuson and Svahlin 1978; Schop et al. 1990; Tripathy et al. 1993; Venkov et al. 2000; Vogel and Chandler 1974; Yilmaz and Yuksel 2005; Zettenberg et al. 1977), and *in vitro* exposure of prokaryotic cells (Charles et al. 1999b; Garret et al. 1986; Kubo et al. 2002; Mersch-Sundermann et al. 1994; Styles 1973; Venkat et al. 1995; Venkov et al. 2000; Zetterberg 1978; Zetterberg et al. 1977) and eukaryotic cells (Clausen et al. 1990; Galloway et al. 1987; González et al. 2005; Korte and Jalal 1982; Linnainmaa 1984; Maire et al. 2007; Mikalsen et al. 1990; Mustonen et al. 1986; Soloneski et al. 2007; Turkula and Jalal 1985; Venkov et al. 2000). These studies provided positive and negative results, possibly because of differences in the experimental protocols used by the different studies. Furthermore, unless a population with exposure only to 2,4-D is identified, as in a small group of workers reported by Holland et al. (2002), most studies of farmers or pesticide applicators will provide inconclusive results. However, efforts to design studies to deal with possible confounding should be encouraged.

While there have been studies on the pharmacokinetic profiles for humans (Sauerhoff et al. 1977) and animals (Van Ravenzwaay et al. 2003), it does not appear that much research has been

directed towards the 2,4-D conjugate in urine and the potential for reactive oxygen species or other metabolites that may affect hepatic or renal DNA. Although studies of this nature are important in establishing a link between metabolism, DNA damage, and potential cancer(s), available data do not suggest a genotoxic role for 2,4-D.

Reproductive. Three studies of subjects from agricultural communities did not provide convincing evidence suggesting that exposure to 2,4-D is associated with adverse reproductive effects (Arbuckle et al. 2001; Lerda and Rizzi 1991; Swan et al. 2003). Oral studies in animals provided information on gross and microscopic appearance of reproductive organs from males and females (Charles et al. 1996a, 1996b, 1996c; EPA 1984, 1985, 1986, 1987a; Gorzinski et al. 1987; Hansen et al. 1971) and fertility/reproductive indices (Dinamarca et al. 2007; Hansen et al. 1971; Joshi et al. 2012; Marty et al. 2013; Saghir et al. 2013a). These studies suggest that 2,4-D is not a reproductive toxicant. Additional reproductive toxicity studies in animals do not seem necessary at this time.

Results from *in vitro* and *in vivo* studies did not suggest that 2,4-D is an endocrine disruptor chemical (EPA 2015c, 2015d), although some studies describe behavioral effects (Bortolozzi et al. 1998, 1999, 2003; Evangelista de Duffard et al. 1995).

Developmental. A few studies are available that examined the potential association between 2,4-D and birth defects and respiratory ailments in children from subjects exposed to 2,4-D through farming activities (Garry et al. 1996; Sathyanarayana et al. 2010; Weselak et al. 2007, 2008; Yang et al. 2014). The results did not suggest a role for 2,4-D in the health outcomes examined. Studies in animals provide data on standard developmental endpoints in rodents (Charles et al. 2001; Chernoff et al. 1990; Collins and Williams 1971; EPA 1986; Fofana et al. 2000, 2002; Kavlock et al. 1987; Schwetz et al. 1971; Stürtz et al. 2010), histology of liver and bone from rat pups (Troudi et al. 2012a, 2012b), and neurobehavioral effects in rat pups (Bortolozzi et al. 1999). Some of the studies reported reduced fetal or offspring weight, in many cases accompanied by reduced maternal weight gain during pregnancy or some other maternal effect, and minor soft-tissue and skeletal anomalies, in some studies (Chernoff et al. 1990; Fofana et al. 2000, 2002; Schwetz et al. 1971). 2,4-D did not induce teratogenicity. Animal studies have demonstrated that 2,4-D can enter maternal milk and be transferred to nursing offspring, No adverse health outcomes have been reported in children whose mothers were exposed to 2,4-D

through farming activities; however, no information is available regarding levels of 2,4-D in breast milk or in neonates born to these women.

Immunotoxicity. Two studies of workers exposed to herbicides (2,4-D among them) found no evidence that 2,4-D played a role in minor immunological alterations reported in some workers (Faustini et al. 1996; Kluciński et al. 2001). An epidemiological study did find that male offspring were more prone to allergies (Weselak et al. 2007); however, the pathway for this result has not been studied. De Roos et al. (2005) found no association between rheumatoid arthritis and exposure to 2,4-D among female spouses of participants in the AHS. For the most part, studies in animals have only provided information regarding weight and gross and microscopic appearance of lymphoreticular organs and tissues from rats, mice, and dogs; no significant effects have been reported (Charles et al. 1996b, 1996c; EPA 1984, 1985, 1987a; Gorzinski et al. 1987; Hansen et al. 1971; Marty et al. 2013; Steiss et al. 1987). Only one study monitored parameters of immunocompetence in rats and reported negative results (Marty et al. 2013). 2,4-D was a respiratory allergen in mice sensitized with 2,4-D dermally and challenged with 2,4-D intratracheally (Fukuyama et al. 2009). Conduction of a Tier I screen immunology battery in B6C3F1 mice exposed to 2,4-D would be reassuring.

Neurotoxicity. There is limited information regarding neurological effects from cases of oral or dermal intoxication with commercial products containing 2,4-D (Berkley and Magee 1963; Berwick 1970; Durakovic et al. 1992; Dudley and Thapar 1972; Goldstein et al. 1959). Several studies also examined the potential association between exposure to 2,4-D and Parkinson's disease (Dhillon et al. 2008; Hancock et al. 2008; Kamel et al. 2007; Tanner et al. 2009). Only Tanner et al. (2009) reported an association between 2,4-D and Parkinson's disease. Two studies did not find an association between 2,4-D and depression among female spouses from pesticide applicators in the AHS (Beard et al. 2013; Beseler et al. 2006). Oral studies in animals did not find gross or microscopic alterations in tissues of the nervous system following exposure to 2,4-D (Charles et al. 1996b, 1996c; EPA 1984, 1987a; Gorzinski et al. 1987; Hansen et al. 1971; Marty et al. 2013; Mattsson et al. 1997; Squibb et al. 1983; Steiss et al. 1987). The available chronic-duration oral animal studies did not conduct neurobehavioral tests. However, based on available information, 2,4-D does not appear to present a particular neurotoxicity concern to humans at environmentally-relevant exposure levels.

Epidemiological and Human Dosimetry Studies. Many epidemiological studies provided information regarding exposure to 2,4-D and a wide range of health outcomes (see Chronic-Duration MRL above for references). Although some studies found that exposure to 2,4-D was positively associated with adverse outcomes, others did not. As previously noted, being significantly associated does not imply causality, although it suggests that exposure to the chemical plays some role in the health outcome assessed and that biological plausibility exists. Conduction of studies in areas where exposures to 2,4-D and other chemicals in the workplace can be adequately characterized would provide valuable information.

Biomarkers of Exposure and Effect.

Exposure. Further refinements to the methodology for estimating exposure levels from urinary levels of 2,4-D, including awareness of factors that can determine the extent of exposure, such as type of application method, glove use, repairing equipment, size of the area treated, and personal hygiene practices, would be valuable. Examining how urine collection timing in relation to exposure can affect the estimates of exposure levels also would be valuable.

Effect. There are no 2,4-D-specific effects following exposure to this substance. Effects that have been associated with acute exposure to high amounts of 2,4-D can also be induced by exposure to other chemicals or can even be caused by conditions unrelated to chemical exposures. Any research aimed at identifying a specific biomarker of effect for 2,4-D would be valuable.

Absorption, Distribution, Metabolism, and Excretion. Information is available regarding absorption, distribution, metabolism, and excretion of 2,4-D in humans and animals following oral and dermal exposure to 2,4-D (Feldmann and Maibach 1974; Griffin et al. 1997a; Harris and Solomon 1992; Khanna and Fang 1966; Kohli et al. 1974; Moody et al. 1990, 1994; Sauerhoff et al. 1977; van Ravenzwaay et al. 2003; Wester et al. 1996). These and additional studies have shown that 2,4-D is almost completely absorbed from the gastrointestinal tract, but dermal absorption is relatively low. 2,4-D distributes widely in tissues following oral exposure, does not accumulate in tissues, is subject to limited metabolism, and is eliminated via the kidneys by a mechanism that involves a saturable carrier protein. The available studies have provided a fairly good characterization of the toxicokinetics of 2,4-D and further studies do not seem necessary at this time.

PBPK models for 2,4-D in rabbits, rats, and humans have been reported (Durkin et al. 2004; Kim et al. 1994, 1995, 1996, 2001). The Kim et al. (1994, 1995, 1996, 2001) and Durkin et al. (2004) models have very different structures, although they appear to yield similar predictions of plasma elimination kinetics when optimized to the same intravenous dosing studies in rats. A particular feature of the Durkin et al. (2004) model is reversible suppression of glomerular filtration and renal blood flow at high 2,4-D concentrations, which results in dose-dependent suppression of urinary excretion. Experimental verification of reversibility of suppression of renal blood flow by 2,4-D would be useful for further validation of this model and its application to human exposures that result in high 2,4-D concentrations.

Comparative Toxicokinetics. Studies in animals have shown the existence of sex and species differences in the toxicokinetics of 2,4-D (Griffin et al. 1997a; Timchalk 2004; van Ravenzwaay et al. 2003). Differences are due principally to the species-dependent activity of the OAT1 carrier protein responsible for the secretion of 2,4-D into the urine. Species with lower capacity to excrete 2,4-D exhibit higher plasma half-life and increased susceptibility to 2,4-D toxicity, as is the case for dogs. Studies of possible genetic determinants of the OAT1 activity carrier in humans could help identify human populations with potentially increased sensitivity to 2,4-D. Studies of OAT1 activity by age, sex, health, and other conditions would be of value to help characterize acceptable exposures for susceptible populations.

Children's Susceptibility. Data needs relating to both prenatal and childhood exposures, and developmental effects expressed either prenatally or during childhood, are discussed in detail in the Developmental Toxicity subsection above.

The limited information available regarding effects of 2,4-D in children comes from epidemiological studies of farming communities where 2,4-D has been used and have included monitoring of children. These studies have not provided conclusive evidence of associations between 2,4-D and adverse health outcomes in children (Flower et al. 2004; Garry et al. 1996; Metayer et al. 2013; Weselak et al. 2007, 2008; Yang et al. 2014). Continuous monitoring of children exposed to 2,4-D in farming communities is indicated to generate more data.

Animal studies have shown that 2,4-D can be transferred to the offspring through the placenta and via the mother's milk and that it distributes widely in fetal or neonatal tissues (Elo and Ylitalo 1979; Lindquist and Ullberg 1971; Marty et al. 2013; Saghir et al. 2013a, 2013b; Sandberg et al. 1996; Stürtz et al. 2000, 2006). No adverse health outcomes have been reported in children whose mothers were exposed to 2,4-D

through farming activities. Although no information is available regarding levels of 2,4-D in breast milk or in neonates born to these women, their 2,4-D exposure levels were likely many times lower than those employed in animal studies. Therefore, 2,4-D does not appear to present a particular toxicity concern to breastfeeding mothers. Monitoring of women with the highest exposures in farming communities would not likely provide valuable information.

As summarized in Section 2.17, Developmental Effects, studies in rodents have shown that, for the most part, adverse developmental effects (i.e., mainly reduced body weight in the offspring) occur at maternal dose levels that induced maternal toxicity, mainly reduced maternal weight during pregnancy. Reduced offspring weight was reported in a study in rats administered a relatively low postpartum dose of 2.5 mg 2,4-D/kg/day (Stürtz et al. 2010). Because no such effects have been reported in other studies that exposed dams to considerably higher doses, it would be useful to try to replicate those findings.

Physical and Chemical Properties. The physical-chemical properties of 2,4-D are provided in Chapter 4. Important properties such as melting point, boiling point, vapor pressure, solubility, log K_{ow} and Henry's Law constant are available. No data needs are identified.

Production, Import/Export, Use, Release, and Disposal. According to the Emergency Planning and Community Right-to-Know Act of 1986, 42 U.S.C. Section 11023, industries are required to submit substance release and off-site transfer information to the EPA. The TRI, which contains this information for 2016, became available in 2018. This database is updated yearly and should provide a list of industrial production facilities and emissions.

Environmental Fate. The environmental fate and transport of 2,4-D is understood and no data needs are identified. The mobility of 2,4-D in soils is expected to be high based on measured K_{oc} values; however, detection of 2,4-D in groundwater is infrequent since it degrades rapidly in soil. Volatilization is generally considered low. Hydrolysis in acidic soils and photolysis may result in some degradation of 2,4-D. Biodegradation primarily accounts for the removal of 2,4-D from the environment.

Bioavailability from Environmental Media. 2,4-D has been detected in aquatic and terrestrial organisms (Schultz and Whitney 1974) and is therefore bioavailable to some extent from environmental media; however, elimination from the organisms was rapid. Aerobic biodegradation reduces its bioavailability. No data needs are identified.

Food Chain Bioaccumulation. Measured BCFs of 2,4-D in fish suggest that bioaccumulation in aquatic organisms is not high. No data needs are identified.

Exposure Levels in Environmental Media. Reliable monitoring data for the levels of 2,4-D in contaminated media at hazardous waste sites are needed so that the information obtained on levels of 2,4-D in the environment can be used in combination with the known body burden of 2,4-D to assess the potential risk of adverse health effects in populations living in the vicinity of hazardous waste sites.

Exposure Levels in Humans. Humans are exposed to 2,4-D mainly by dermal exposure during application as an herbicide. Populations may also be exposed by transport of 2,4-D into residential homes from agricultural spray drift, volatilization, soil or dust resuspension, track-in on shoes, and on clothing. Adequate biomonitoring data are available to assess 2,4-D exposure to the general population of the United States. Continued monitoring of the general U.S. population through the NHANES program can provide information on the trend of exposure to 2,4-D and identify subsets in the population with the highest levels of exposure.

Exposures of Children. Children are exposed to 2,4-D mainly by dermal exposure to residue transported into homes from applicators and from direct contact with treated residential lawns. Adequate biomonitoring data are available to assess 2,4-D exposure to children of the United States. Continued monitoring through the NHANES program is needed in order to understand future exposures. Additional research on exposures of neonates and young children of workers who handle 2,4-D is needed and justifiable. No human data were located regarding 2,4-D in breast milk and this is a data need.

6.3 ONGOING STUDIES

The following ongoing research pertaining to 2,4-D (Table 6-1) was identified in the National Institutes of Health (NIH) Research Portfolio Online Reporting Tools (RePORTER 2018):

Table 6-1. Ongoing Studies on 2,4-D

Investigator	Affiliation	Research description	Sponsor
Laura Beane Freeman	Division of Cancer Epidemiology and Genetics of NCI	Investigation of potential associations between exposure to pesticides (2,4-D among them) and a wide range of health endpoints in participants in the AHS	NCI

Table 6-1. Ongoing Studies on 2,4-D

Investigator	Affiliation	Research description	Sponsor
Jian Feng	VA Western New York Healthcare System	The interaction of parkin and environmental toxins (2,4-D among them) in Parkinson's disease	VA
Melissa Friesen	Division of Cancer Epidemiology and Genetics of NCI	Assessment of occupational exposure to a variety of substances including 2,4-D	NCI
Jane Hoppin	Biology, Schools of Arts and Sciences, North Carolina State University Raleigh	Environmental pesticide exposure (includes 2,4-D) and respiratory outcomes in women and children	NIEHS
Timothy D. Howard	Obstetrics & Gynecology, Schools of Medicine, Wake Forest University Health Sciences	Human pesticide exposure (includes 2,4-D) and epigenetic changes in sperm DNA	NIEHS
Lee S. Newman	Public Health & Preventive Medicine, Schools of Public Health, University of Colorado Denver	Etiologic and mechanistic factors underlying chronic kidney disease in Guatemalan sugarcane workers	NIEHS
Steven D. Stellman	Eunice Kennedy Shriver NICHD, Foundation for Worker/Veteran Environmental Health	Agent orange (2,4-D is a component) and adverse birth outcomes: a re-examination	NICHD

2,4-D = 2,4-dichlorophenoxyacetic acid; AHS = Agricultural Health Study; DNA = deoxyribonucleic acid; NCI = National Institutes of Health; NICHD = National Institute of Child Health and Human Development; NIEHS = National Institute of Environmental Health Sciences; VA = Veteran's Affairs

Source: RePORTER 2018