

US EPA ARCHIVE DOCUMENT

Dichlorvos

TEACH Chemical Summary



U.S. EPA, Toxicity and Exposure Assessment for Children's Health

This TEACH Chemical Summary is a compilation of information derived primarily from U.S. EPA and ATSDR resources, and the TEACH Database. The TEACH Database contains summaries of research studies pertaining to developmental exposure and/or health effects for each chemical or chemical group. TEACH does not perform any evaluation of the validity or quality of these research studies. Research studies that are specific for adults are not included in the TEACH Database, and typically are not described in the TEACH Chemical Summary.

I. INTRODUCTION

Dichlorvos, also known as DDVP, is an organophosphate pesticide that is widely used to treat domestic animals and livestock for internal and external parasites, to control insects commercially and in the home, and to protect crops from insect infestation (1, 2). Dichlorvos occurs as an oily colorless to amber liquid, with an aromatic chemical odor.

The target for health effects of dichlorvos is the nervous system (1, 2). Dichlorvos has been shown to inhibit acetylcholinesterase and cholinesterase activities in red blood cells and in the brain; these enzymes are important for neuron function in the nervous system and other processes (1, 2). Studies of effects of dichlorvos exposure during development have been performed, and some studies have investigated long-term effects of early life exposure (2). Effects on nerve functions following dichlorvos exposure during development have been reported (2-4).

Dichlorvos is found in many household and animal insecticide products, such as flea collars, pest strips (resin strips containing pesticides to hang indoors for killing insects), and pesticide sprays and foggers (1, 2). These residential uses increase the likelihood of children's exposure (1, 2, 5, 6). The U.S. EPA estimated that inhalation exposure from residential uses of dichlorvos in pest strips, professional lawn applications of pesticides, total release foggers, pesticide sprays, and some pet flea collars may reach levels of concern (7). Recent changes in registration have been made by the U.S. EPA and residential exposure should be decreased based on these changes (2). Dichlorvos can also be generated as a metabolic or degradation product of the pesticides naled and trichlorfon in humans and animals; and in food, water, and the environment (2, 7-10).

II. EXPOSURE MEDIA AND POTENTIAL FOR CHILDREN'S EXPOSURE¹

Exposure Media	Relative Potential for Children's Exposure ^{2,3}	Basis ⁴
Indoor Air	Higher	Dichlorvos can be released into the air during indoor and outdoor spraying for pests, and from pest strips. For most children, dichlorvos exposure occurs as a result of use of household and animal products that contain dichlorvos, such as pest strips and flea collars.
Dermal	Medium	Use of pet flea collars containing dichlorvos on pets can result in dermal exposures of children.
Ambient Air	Lower	Dichlorvos readily breaks down and has not been detected in significant levels in ambient air. Concentrations of dichlorvos may be transiently higher in agricultural areas after application to crops.
Groundwater	Lower	Dichlorvos breaks down quickly in water and is not generally found at levels of concern.
Drinking Water	Lower	Dichlorvos breaks down quickly in water and is not generally found at levels of concern.
Diet	Lower	Dichlorvos measured in food has been found at very low or undetectable levels.
Sediment	Unknown	There are little to no data on dichlorvos in this medium.
Soil	Unknown	There are little to no data on dichlorvos in this medium.

¹ For more information about child-specific exposure factors, please refer to the Child-Specific Exposure Factors Handbook (<http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=55145>).

² The Relative Potential for Children's Exposure category reflects a judgment by the TEACH Workgroup, U.S. EPA, that incorporates potential exposure pathways, frequency of exposure, level of exposure, and current state of knowledge. Site-specific conditions may vary and influence the relative potential for exposure. For more information on how these determinations were made, go to http://www.epa.gov/teach/teachprotocols_chemsumm.html.

³ Childhood represents a lifestage rather than a subpopulation, the distinction being that a subpopulation refers to a portion of the population, whereas a lifestage is inclusive of the entire population.

⁴ Information described in this column was derived from several resources (e.g., 1, 2) including studies listed in the TEACH Database (<http://www.epa.gov/teach/>).

Supporting references and summaries are provided in the TEACH database at <http://www.epa.gov/teach/>.

Last revised 6/27/2007: includes research articles and other information through 2006.

III. TOXICITY SUMMARY^{5, 6}

Dichlorvos exposure inhibits acetylcholinesterase activity in brain, plasma, and red blood cells; acetylcholinesterase is an enzyme that is important for neurological function (1, 2). Exposure of adults to dichlorvos has resulted in neurotoxicity, and in adverse respiratory and dermatologic irritant effects (1, 2, 11-14). Acute exposure to dichlorvos may result in nausea, anxiousness, restlessness, teary eyes, and heavy sweating (1, 2). Severe poisoning can result in coma, inability to breathe, and death (1, 2). Hematologic effects, including aplastic anemia, were associated with children's exposure to household insecticide products containing mixtures of insecticides that included dichlorvos (15, 16); however, these effects could not be definitively associated with dichlorvos exposure.

Experimental animal studies have demonstrated small but significant decreases in sperm motility in adult males (17). Additional studies reported in the U.S. EPA Interim Reregistration Decision (IREDD) described reproductive and developmental effects (2). Other studies have demonstrated neurodevelopmental toxicity (e.g., decreased brain weight at birth, and altered electrophysiology of nerves) following prenatal and/or lactational exposure to dichlorvos (3, 4, 18, 19). Additional studies listed in the U.S. EPA IREDD described toxicity following maternal exposure to dichlorvos during pregnancy, including reduced maternal weight gain, and reduced numbers of dams bearing a litter (2). Reported effects in offspring following maternal exposure during pregnancy included increased auditory startle reflex in males, and estrous cycle abnormalities in females. Reduced brain, plasma, and red blood cell cholinesterase activities were reported in offspring of exposed dams (2).

Carcinogenicity Weight of Evidence Classification⁷: Dichlorvos is classified by U.S. EPA IRIS as probable human carcinogen by the oral route, based on a finding of forestomach tumors, leukemias, and pancreatic acinar adenomas in adult rodents (1); evidence of mammary gland tumors has also been reported in adult animal studies (www.epa.gov/iris/subst/0151.htm, II.B.2) (20). The U.S. EPA Office of Prevention, Pesticides, and Toxic Substances (OPPTS) in 2006 classified dichlorvos carcinogenicity evidence as "suggestive" because forestomach tumors and mononuclear cell leukemias in experimental animals were not considered relevant to humans, and no evidence of cancer was seen in a two-year inhalation carcinogenicity study (http://www.epa.gov/oppsrrd1/REDDs/ddvp_ired.pdf) (2). The World Health Organization International Agency for Research on Cancer (IARC) classifies dichlorvos as possibly carcinogenic (Group 2B) (<http://monographs.iarc.fr/ENG/Monographs/vol53/volume53.pdf>) (21).

⁵ Please refer to research article summaries listed in the TEACH Database for details about study design considerations (e.g., dose, sample size, exposure measurements).

⁶ This toxicity summary is likely to include information from workplace or other studies of mature (adult) humans or experimental animals if child-specific information is lacking for the chemical of interest. Summaries of articles focusing solely on adults are not listed in the TEACH Database because the TEACH Database contains summaries of articles pertaining to developing organisms.

⁷ For recent information pertaining to carcinogen risk assessment during development, consult "Guidelines for Carcinogen Risk Assessment and Supplemental Guidance on Risks from Early Life Exposure" at <http://www.epa.gov/cancerguidelines>.

IV. EXPOSURE AND TOXICITY STUDIES FROM THE TEACH DATABASE

This section provides a brief description of human and animal studies listed in the TEACH Database. For more details about study design parameters, e.g., doses and exposure information, please refer to article summaries in the TEACH Database. Any consideration should include an understanding that exposure levels in animal studies, in many cases, are greater than exposure levels normally encountered by humans.

A. HUMAN EXPOSURE AND EFFECTS

- ▶ One media of exposure to dichlorvos is air. Indoor air concentrations of dichlorvos were found to increase with increasing number of dichlorvos-containing strips hanging in the rooms (22). In a study of pregnant women in New York City, 1 out of 55 personal air samples tested had detectable levels (greater than 0.3 mg/m³) of dichlorvos (23). A study of pesticide exposures of Minnesota children reported that dichlorvos was detected in 17% of personal air samples (24). Children who live on farms may be at increased risk of exposure due to combined domestic and agricultural use of dichlorvos (1, 7, 25). The U.S. EPA estimated that inhalation exposure from residential uses of dichlorvos in pest strips, professional lawn applications of pesticides, total release foggers, pesticide sprays, and some pet flea collars may reach levels of concern (7).
- ▶ Dichlorvos was not detected in analyses of infant and toddler foods (26, 27), or in U.S. FDA and USDA tests of many foods (8). In one study of pesticide exposure of Minnesota children, dichlorvos was not detected in drinking water, and infrequently detected in foods (24). Some dichlorvos exposure can result from uses of naled or trichlorfon (2).
- ▶ Studies of possible correlations between early life exposure to dichlorvos and cancer are sparse. Two studies suggested that dichlorvos may be associated with some immunological effects and hematological cancer in children, including aplastic anemia and acute lymphoblastic leukemia (15, 16). However, in both studies, the insecticides to which the children were exposed contained mixtures of pesticides, of which dichlorvos was one. Thus, further studies are needed to test the association of these effects in children with dichlorvos alone.
- ▶ Most existing studies of adverse health effects following dichlorvos exposure evaluated adults, particularly adult pesticide applicators and industrial workers (1). Of the few available reports on child exposure to dichlorvos, most studies on health effects included small numbers of children (11-14). Case reports of accidental or intentional ingestion (12, 14) or skin application (13) of dichlorvos include children ranging in age from 9 months old to late adolescence.
- ▶ Respiratory irritation following dichlorvos exposure was reported in one study that included children. A strong correlation between acute respiratory symptoms and exposure to dichlorvos during outdoor spraying was observed in both children and adults (11). In this study, the authors could not rule out irritant effects of the solvent used to disperse the dichlorvos (11).

B. EXPERIMENTAL ANIMAL EXPOSURE AND EFFECTS

- ▶ Exposure of adult male rats to dichlorvos via subcutaneous injection resulted in small but significantly reduced motility (movement) of their sperm at doses of dichlorvos that had significantly reduced cholinesterase activity (17).
- ▶ Prenatal exposure to dichlorvos via maternal inhalation had no effect on litter size or gestation length at doses of dichlorvos that decreased maternal plasma cholinesterase levels (28).

Supporting references and summaries are provided in the TEACH database at <http://www.epa.gov/teach/>.

Last revised 6/27/2007: includes research articles and other information through 2006.

- ▶ Animal studies have demonstrated adverse neurological effects of dichlorvos in offspring exposed during pregnancy. In rats, prenatal exposure via maternal ingestion of dichlorvos correlated with changes in the electrophysiology of nerves of the central nervous system in offspring (3, 4). In guinea pig offspring, prenatal exposure to dichlorvos by maternal ingestion (18) or injection (19) resulted in decreased brain weight and hypoplasia (retarded growth or shrinking) of the cerebellum.
- ▶ Lactational exposure of offspring to dichlorvos via maternal ingestion also resulted in measurable changes in the electrophysiology of nerves in the offspring (3, 4). Changes in peripheral nerve conduction velocity and refractory periods were detected at doses of dichlorvos that were lower than doses that caused decreased brain cholinesterase activity (4).

V. CONSIDERATIONS FOR DECISION-MAKERS

This section contains information that may be useful to risk assessors, parents, caregivers, physicians, and other decision-makers who are interested in reducing the exposure and adverse health effects in children for this particular chemical. Information in this section focuses on ways to reduce exposure, assess possible exposure, and, for some chemicals, administer treatment.

- ▶ According to the U.S. EPA, people should stay out of a room or a home for at least 10 hours after fumigating treatment with dichlorvos, during which time the treated space should be ventilated (1).
- ▶ The U.S. EPA provides recommendations for safe storage in the home of insecticide products that contain dichlorvos, and other products containing herbicides, pesticides, and other potential hazards (29). Recommendations include storing products in a locked cabinet, never transferring products to another unlabeled container, and washing toys and home surfaces often.
- ▶ To reduce exposures to children, a program of pest management called Integrated Pest Management (IPM) uses alternatives to pesticides, and is available through the U.S. EPA (30).
- ▶ A treatment guide for clinicians, "Recognition and Management of Pesticide Poisonings," is available online from the U.S. EPA (31). In case of dichlorvos poisoning, emergency rooms have drugs that can stop the harmful effects of dichlorvos (1). One case report suggested that people experiencing dichlorvos poisoning may be more sensitive to succinylcholine, a neuromuscular relaxant which is medically administered in some circumstances (12).
- ▶ Detailed compilations of information pertaining to exposure to and health effects of dichlorvos is available in the ATSDR Toxicological Profile for Dichlorvos (1) and the recent IRED (2). A Hazard Summary for Dichlorvos is also available from the U.S. EPA (32).
- ▶ Dichlorvos is also a metabolite of the pesticides naled and trichlorfon (1, 7). Naled is used mainly for wide-area mosquito abatement applications and for insect control on agricultural crops, but is not registered for use in home products (6). Trichlorfon field crop uses have been canceled by the U.S. EPA, though its use in beef cattle for import into the U.S., on lawn and turf, and in fish ponds continues (7, 10).

- ▶ A dietary risk assessment for dichlorvos that included children is available from the U.S. EPA; risks from dietary exposure were estimated to be low (8). This risk assessment included analysis of contributions of naled and trichlorfon to dichlorvos exposure from foods, and found detectable concentrations that were below U.S. EPA levels of concern based in part on monitoring by the U.S. FDA and USDA (8). Analysis of infant formulas and toddler foods in New Zealand (26), as well as foods in the diets of preschool children in Washington State (27), found little or no dichlorvos in the foods, suggesting that diet may not be a major source of exposure to dichlorvos.
- ▶ A risk assessment for exposures of children to dichlorvos via inhalation has been performed (25); major sources of domestic and agricultural exposure include insecticide use in the home, as well as the use of pesticides on farms. Recent U.S. EPA exposure estimates based on registration changes calculated that inhalation exposure from residential uses of dichlorvos do not reach levels of concern (2).
- ▶ A cumulative risk assessment for organophosphates, a group of closely-related pesticides, includes an assessment of dichlorvos (33). The organophosphate pesticides have a common mechanism of action in their impact on the nervous system, and cumulative (or combined) exposure to pesticides within this group was assessed together.
- ▶ A revised risk assessment that includes an assessment of health risks from dichlorvos exposure during development is also available (2). A meta-analysis of human and animal studies comparing dichlorvos sensitivity between species also included developmental studies (34).
- ▶ Dichlorvos is listed as number 243 out of 275 chemicals on the 2005 Priority List of Hazardous Substances for the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) section 104 (i), as amended by the Superfund Amendments and Reauthorization Act (SARA). This is a list of substances of concern, in the order of priority, that are most commonly found at sites listed on the National Priorities list (NPL) (35).
- ▶ Consult the U.S. EPA “Child-Specific Exposure Factors Handbook” (EPA-600-P-00-002B) for factors to assess children’s dermal absorption, ingestion, and inhalation rates (36). An updated External Draft of the 2006 version of this handbook is available (37).

VI. TOXICITY REFERENCE VALUES

A. Oral/Ingestion

U.S. EPA OPP Acute Dietary Reference Dose (RfD) (for general population including infants and children): 0.0008 mg/kg/day, based on acute oral cholinesterase studies in rats, measuring red blood cell and brain cholinesterase depression; $UF_A=10X$, $UF_H=10X$, and $FQPA\ SF=1X$ (http://www.epa.gov/oppsrrd1/REDS/ddvp_ired.pdf, p. 138) (2). Last revised 7/31/06.

U.S. EPA OPP Chronic Dietary Reference Dose (RfD) (for all populations): 0.0005 mg/kg/day, based on a 1-year dog study, measuring plasma and red blood cell cholinesterase depression; $UF_A=10X$, $UF_H=10X$, and $FQPA\ SF=1X$ (http://www.epa.gov/oppsrrd1/REDS/ddvp_ired.pdf, p. 138) (2). Last revised 7/31/06.

U.S. EPA OPP Short-Term Incidental Oral (1-30 days): Lowest Observed Adverse Effect Level (LOAEL) = 0.1 mg/kg/day, based on human 21-day oral study, measuring red blood cell cholinesterase depression; $UF_H=10X$, $FQPA\ SF=3X$ (UF_L) (http://www.epa.gov/oppsrrd1/REDS/ddvp_ired.pdf, p. 138) (2). Last revised 7/31/06.

U.S. EPA OPP Acute Dermal and Acute Incidental Oral: $BMDL_{10} = 0.8$ mg/kg/day, based on acute oral cholinesterase studies in rats, measuring red blood cell and brain cholinesterase depression; $UF_A=10X$, $UF_H=10X$, and $FQPA\ SF=1X$ (http://www.epa.gov/oppsrrd1/REDS/ddvp_ired.pdf, p. 138) (2). Last revised 7/31/06.

U.S. EPA Reference Dose (RfD) for Chronic Oral Exposure: $5E-4$ (or 0.0005) mg/kg-day, based on plasma and red blood cell cholinesterase inhibition in adult animal study, includes consideration of developmental studies in rats (www.epa.gov/iris/subst/0151.htm, I.A.1) (20). Last Agency Verification Date 5/27/92.

U.S. EPA Cancer Oral Slope Factor: $2.9E-1$ (or 0.29) per (mg/kg)/day, protective of forestomach tumors, pancreas tumors, and leukemia in adult studies (www.epa.gov/iris/subst/0151.htm, II.B.1) (20). Last Agency Verification Date 9/22/88.

U.S. EPA Cancer Drinking Water Unit Risk: $8.3E-6$ (or 0.0000083) per $\mu\text{g/L}$, derived using extrapolation method with the linearized multistage procedure, extra risk (www.epa.gov/iris/subst/0151.htm, II.B.1) (20). Last Agency Verification Date 9/22/88.

U.S. EPA Drinking Water Concentrations at Specified Risk Levels for Cancer: $1E-4$ (or 1 in 10,000), $1E+1$ (or 10.0) $\mu\text{g/L}$; $1E-5$ (or 1 in 100,000), $1E+0$ (or 1.0) $\mu\text{g/L}$; $1E-6$ (or 1 in 1,000,000), $1E-1$ (or 0.1) $\mu\text{g/L}$ (www.epa.gov/iris/subst/0151.htm, II.B.1) (20). Last Agency Verification Date 9/22/88.

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B. Inhalation

U.S. EPA OPP Acute Inhalation (1 Day): BMDL₁₀ = 0.8 mg/kg/day (inhalation absorption rate = 100%), air concentration equivalent = 0.8 mg/m³, based on rat acute oral cholinesterase studies; UFA=10X, UF_H=10X or 3X, and FQPA SF=1X

(http://www.epa.gov/oppsrrd1/REDS/ddvp_ired.pdf, p. 138) (2). Last revised 7/31/06.

U.S. EPA OPP Short- and Intermediate-Term Inhalation of Vapors: Oral study LOAEL = 0.1 mg/kg/day, uncertainty factor = 30, air concentration equivalent = 0.35 mg/m³, based on human 21-day oral study, measuring red blood cell cholinesterase depression; UF_H=10X, FQPA SF=3X (UFL) (http://www.epa.gov/oppsrrd1/REDS/ddvp_ired.pdf, p. 138) (2). Last revised 7/31/06.

U.S. EPA OPP Short- and Intermediate-Term Inhalation During Application: LOAEL = 0.1 mg/kg/day, uncertainty factor = 30, air concentration equivalent = 0.35 mg/m³, based on human 21-day oral study, measuring red blood cell cholinesterase depression; UF_H=10X, FQPA SF=3X (UFL) (http://www.epa.gov/oppsrrd1/REDS/ddvp_ired.pdf, p. 138) (2). Last revised 7/31/06.

U.S. EPA OPP Long-Term Inhalation of Vapors: BMDL₁₀ = 0.07 mg/m³, based on a 2-year inhalation study in rats, measuring red blood cell cholinesterase depression; UFA=10X, UF_H=3X, and FQPA SF=1X (http://www.epa.gov/oppsrrd1/REDS/ddvp_ired.pdf, p. 138) (2). Last revised 7/31/06.

U.S. EPA Reference Concentration (RfC) for Chronic Inhalation Exposure: 5E-4 (or 0.0005) mg/m³, based on decreased brain cholinesterase activity in adult rats (www.epa.gov/iris/subst/0151.htm, I.B.1) (20). Last Agency Verification Date 11/5/92.

U.S. ATSDR Minimal Risk Level (MRL): 0.002 ppm (acute inhalation); 0.0003 ppm (intermediate inhalation); 0.00006 ppm (chronic inhalation); 0.004 mg/kg/day (acute oral); 0.003 mg/kg/day (intermediate oral); 0.0005 mg/kg/day (chronic oral) [1ppm = 9.04 mg/m³ - vapor phase]; all protective of neurological effects in adult studies (www.atsdr.cdc.gov/mrls.html) (38). Last revised 9/97.

C. Dermal

U.S. EPA OPP Short, Intermediate, and Long-Term Dermal: Oral study LOAEL = 0.1 mg/kg/day with dermal absorption = 11%, based on a human 21-day oral study measuring red blood cell cholinesterase inhibition; UF_H=10X, FQPA SF=3X (UFL)

(http://www.epa.gov/oppsrrd1/REDS/ddvp_ired.pdf, p. 138) (2). Last revised 7/31/06.

VII. U.S. FEDERAL REGULATORY INFORMATION

- ▶ Dichlorvos is one of 188 hazardous air pollutants (HAPs) listed under section 112(b) of the 1990 Clean Air Act Amendments and is regulated for use from more than 170 industrial source categories (39).
- ▶ Dichlorvos is a restricted use pesticide (RUP) meaning that some dichlorvos labels require professionally-trained applicators, and specific instructions for labeling are stated in the Interim Reregistration Eligibility Decision (IREDD) (2).
- ▶ The U.S. EPA issued a document in 1995 describing voluntary cancellations of use of dichlorvos by the sole registrant of dichlorvos production, Amvac Chemical Corporation (40). The deleted uses included many, but not all, domestic uses, all aerial applications, and other uses. Additional voluntary cancellations were implemented in 2006 (2).
- ▶ The U.S. EPA requires reporting of quantities of certain chemicals that exceed a defined reportable quantity, and that quantity varies for different chemicals (41). Under the Emergency Planning and Community Right-to-Know Act (EPCRA) Section 313 "Toxic Chemicals," quantities of dichlorvos greater than 25,000 pounds manufactured or processed, or greater than 10,000 pounds otherwise used, is required (41). Dichlorvos is also listed as an Extremely Hazardous Substance (EHS) with reporting requirements of 1,000 pounds under Section 302, and 10 pounds under Section 304 of EPCRA (41). Dichlorvos is listed under the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA), and reporting releases of any quantity exceeding 10 pounds is required (41).

VII. BACKGROUND ON CHEMICAL

A. CAS Number: 62-73-7

B. Physicochemical Properties: The chemical formula for dichlorvos is $C_4H_7Cl_2O_4P$. Dichlorvos occurs as an oily colorless to amber liquid, with an aromatic chemical odor. Search for dichlorvos at <http://chem.sis.nlm.nih.gov/chemidplus>.

C. Production: Dichlorvos is an organophosphate insecticide produced in many countries, including the United States. The World Health Organization estimated worldwide production in 1989 at 4,000 tons per year (42). Reported total disposals and releases in the U.S. in 2005 totaled 265 pounds, though this value should be considered a minimum estimate, as reporting of disposals and releases of dichlorvos is not required for all facilities that use it (1, 43).

D. Uses: The registered uses of dichlorvos include: insect control in agricultural, commercial, industrial, and residential sites (4). Dichlorvos is used in sprays for indoor and outdoor uses, resin pest strips, crack and crevice treatment, and pet flea collars. Dichlorvos is used in mushroom houses and food storage areas, non-perishable raw and processed agricultural commodities, food manufacturing and processing plants, and parasite treatment of cattle and poultry. Dichlorvos is also used in some veterinarian treatments including use on swine (1).

Supporting references and summaries are provided in the TEACH database at <http://www.epa.gov/teach/>.
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E. Environmental Fate: Dichlorvos enters the environment during production and use, and through accidental spills. It volatilizes readily into air and breaks down quickly; higher temperature and humidity facilitate rapid breakdown (1). Dichlorvos is also a metabolic product of dieldrin and trichlorfon, generated by the metabolism of plants, animals, and humans (7). The half-life of dichlorvos in water has been calculated to be between 1.5 and 57 days and depends on temperature and pH (1). Dichlorvos does not bioaccumulate in animals or plants, and 90% of applied dichlorvos in greenhouses and food storage areas is broken down in 3-6 hours. Dichlorvos does not bind to soil but can remain in dry acidic soil for days (1).

F. Synonyms and Trade Names: 2,2-dichlorovinyl dimethyl phosphate, Atgard, DDVP, Dichlorvos, Dichlorphos, Deriban, Astrobot, Fly-die, Mafu, No-Pest Strip, Nuvan, Szklarniak, Task, Vapona

Additional information on dichlorvos is available in the TEACH Database for Dichlorvos and at the following Web sites:

www.epa.gov/oppsrrd1/op/ddvp.htm

www.regulations.gov (search for dichlorvos)

www.atsdr.cdc.gov/toxprofiles/tp88.html

www.atsdr.cdc.gov/tfacts88.html

www.epa.gov/iris/subst/0151.htm

www.health.state.mn.us/divs/eh/children/healthrisks.html

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