chemicalWATCH Factsheet

2,4-D

What is 2,4-D?

2,4-Dichlorophenoxyacetic acid, commonly known as 2,4-D, is a widely used herbicide in the phenoxy class of chemicals. It is the most commonly used pesticide in the non-agricultural sector and the sixth most common in the agricultural sector, with over 40 million pounds being used in the U.S. annually.[1] 2,4-D production supports a world market of over $300 million. While the herbicide is manufactured and marketed by many different companies, Dow AgroSciences is currently the biggest producer.[2]

2,4-D is a general use pesticide, and does not require a license to use or purchase. It is used in a wide variety of locations, including agricultural, residential, and public areas. 2,4-D can be found in many lawn care products, it is used as a treatment for roadsides and rights of way, and to control aquatic weeds. In agriculture it is used as a herbicide for grass-like crops, including wheat and barley.[3] Products containing 2,4-D are often marketed as "weed and feed" and include Aqua-Kleen, Ortho Weed B Gon, Salvo, Spectracide, Scotts Green Sweep, UltraStop, and Weedone.

2,4-D is a selective herbicide, used to kill broadleaf weeds with little harm to grass crops.[3] It is a plant growth regulator, and mimics the natural plant growth hormone, auxin. Unlike auxins, 2,4-D stays at high levels within plant tissues rather than fluctuating. As a result, it causes rapid cell growth and plants die when their transport systems become blocked and destroyed by abnormally fast growth.[4] While 2,4-D is normally applied to a plant's leaves, it can also be absorbed through the roots and stems.[5] The half-life of 2,4-D in soil is about 1-2 weeks and 1-3 weeks in water.[6]

Health effects of 2,4-D are of particular concern due to its widespread distribution. In a 2003 study of indoor air toxins, 2,4-D was found in the dust of 63% of sampled homes.[7] In a 2001 study, levels of 2,4-D in indoor air and on surfaces (floors, tables, windowsills) increased following lawn application of the herbicide. This resulted in exposure levels for children that were ten times higher than pre-application and shows that 2,4-D is easily tracked into homes.[8]

2,4-D has been linked to

- √ **Cancer**
- √ **Endocrine Disruption**
- √ **Reproductive Toxicity**
- √ **Neurotoxicity**
- √ **Kidney/Liver Damage**
- √ **Toxicity to Dogs, Fish, Birds, Earthworms, and Beneficial Insects**

Acute Toxicity

2,4-D is produced in several forms, including acids, salts, amines and esters, and its toxicity varies between the different forms. The EPA toxicity class ranges from I- III (on a I-IV scale with I being the most toxic) depending on the form and method of exposure. The diethylamine salt is the most toxic (class I) by eye exposure. Inhalation generally leads to coughing, burning, dizziness, and loss of muscle coordination.[9] Oral consumption irritates the digestive tract, results in nausea, diarrhea, vomiting, and can lead to kidney and liver damage.[10] 2,4-D is one of few herbicides to cause nervous system damage; both digestion and inhalation affect the central nervous system. Effects to the nervous system include inflamed nerve endings, lack of coordination, stiffness in the arms and legs, inability to walk, fatigue, stupor, coma, and death.[11] 2,4-D is a serious skin and eye irritant. For the acid form, the LD 50 in rats is 375-666 mg/kg orally and approximately 1500 mg/kg dermally.[12]

Chronic Toxicity

Although a mounting body of evidence links 2,4-D to various cancers, particularly non-Hodgkin's lymphoma, EPA has been reluctant to classify it as a carcinogen in the face of industry pressure. EPA lists the herbicide in class D for carcinogenicity. Chemicals in this class are considered to have inadequate evidence for carcinogenicity, or not enough data is available.[13] However, the link between 2,4-D and non-Hodgkin's lymphoma has been demonstrated in the United States, Italy, Canada, Denmark, and Sweden.[14] A 1986 National Cancer Institute (NCI) study found that farmers in Kansas exposed to 2,4-D for 20 or more days per year had a six-fold higher risk of developing non-Hodgkin's lymphoma than non-farmers.[15] The risk of cancer was higher for farmers who mixed or applied the pesticide themselves. Another study done in 1990 found a 50% increase in non-Hodgkin's lymphoma in farmers who handle 2,4-D.[16] A manufacturer's study submitted to EPA in 1986 indicated that the herbicide can cause rare brain tumors in rats.[17] In 1991, an NCI study found that dogs were more likely to contract canine malignant lymphoma if their owners use 2,4-D on their lawns than if owners did not use the herbicide.[18] When 2,4-D was applied four or more times per year, dogs were twice as likely to contract lymphoma. In addition to these epidemiological studies, a laboratory study conducted by the Food and Drug Administration (FDA) found a 4% incidence of lymphoma in rats exposed to 2,4-D and no lymphoma in unexposed rats.[19] Despite these studies, the carcinogenic potential of 2,4-D remains

BEYOND PESTICIDES
701 E Street, S.E., Suite 200, Washington DC 20003
202-543-5450 (v) • 202-543-4791 (f)
info@beyondpesticides.org • www.beyondpesticides.org

controversial. The pesticide industry has criticized some of the studies mentioned here and cites other studies, which support its claim that 2,4-D does not cancer.[20]

Long-term exposure to 2,4-D also results in a wide range of other health problems. Chronic (long-term) oral intake results in lesions of the kidney and liver in both rats and dogs.[21] In humans, two studies showed a connection between hepatitis cases and chronic oral consumption of 2,4-D, by golfers who habitually licked their golf balls.[22]

2,4-D is also an endocrine disruptor, a chemical that can interfere with the body's hormone messaging system and can alter many essential processes.[23] The National Institute of Health Sciences lists 2,4-D as a suspected endocrine disruptor.[24] In studies with rats, 2,4-D has been shown to alter levels of metabolism and sex hormones.[25]

Several studies have demonstrated that 2,4-D can be a mutagen, or a substance that induces genetic mutations. Workers who apply 2,4-D had a higher number of white blood cells with multiple nuclei than people who were not exposed.[26] In rabbits, 2,4-D exposure resulted in unusual numbers of chromosomes in brain cells.[27] Genetic problems like these can have further consequences in terms of cancer and reproductive problems.

Reproductive toxicity has also been observed in relation to 2,4-D. Residues of 2,4-D are detectable in urine and semen samples of men who apply the herbicide.[28] In rats, exposure resulted in fetuses with abnormal cavity bleeding, increased mortality and genetic damage.[29] A 1996 study of private pesticide appliers in Minnesota found a higher rate of birth defects among the children of appliers than the general public.[30] It also found the birth defect rate to be highest in areas where 2,4-D use was the highest. Another study conducted in 2003 examined the wheat producing states of Montana, Minnesota, South Dakota and North Dakota, where more than 85% of the acreage is treated with chlorophenoxy herbicides, including 2,4-D. Children conceived during the time or herbicide production (April- June) were more likely to have birth defects.[31]

Environmental Effects

Due to its relatively short half-life, 2,4-D is said to have low persistence in both soil and water. However, 2,4-D has a high potential to leach from soils, and therefore a potential for contaminating ground water. The herbicide has been detected in ground water in at least five states and Canada. Low concentrations have also been detected in surface water and drinking water in the US.[32]

2,4-D has been shown to have negative impacts on a number of animals. In birds, 2,4-D exposure reduced hatching success and caused birth defects.[33] It also indirectly affects birds by destroying their habitat and food source. The toxicity of 2,4-D to fish is variable. The butoxyethanol ester is very toxic to fish, but other forms are less toxic.[34] 2,4-D also bio-accumulates in fish, meaning that fish tissues will contain a higher concentration of 2,4-D than the water surrounding them, which puts them at even greater risk.[35] 2,4-dichlorophenol, a breakdown product of 2,4-D, is extremely toxic to earthworms, 15 times more toxic than 2,4-D itself.[36] The herbicide also has negative effects on a range of beneficial insects. It reduces offspring numbers in honeybees, kills predatory beetles and ladybug larvae.[37] This reduction in ladybug numbers caused an increase in aphids, a major pest, in oat fields.[38] Consumption of plants treated with 2,4-D has killed cattle and horses and 2,4-D can also indirectly affect many wild mammal species, including moose, gophers, and voles, by damaging or killing plants they rely on for food.[39]

Regulatory Status and History

2,4-D was one of the first herbicides to be commercially marketed. It was first introduced in the United States in the late 1940's.[40] 2,4-D made up a major portion (about 50%) of the herbicide known as Agent Orange, which was used during the Vietnam War. However, it is thought that most of the health problems related to Agent Orange were actually due to dioxin contamination of the other major component, 2,4,5-T.[41] While 2,4,5-T was the main culprit and has now been banned, several forms of dioxin have also been found in 2,4-D, including 2,4,7,8-TCDD.[42]

The history of dioxin contamination further increases the dangers related to 2,4-D, particularly for the amine and ester forms.[42] Dioxins are highly carcinogenic and can cause health problems as severe as weakening of the immune system, decreased fertility, altered sex hormones, miscarriage, birth defects, and cancer. EPA studies in 1994 detected dioxins in a number of 2,4-D products.[43] The Washington Department of Agriculture also detected dioxins in a 2,4-D product in 1998.[44]

2,4-D is currently undergoing EPA's reregistration process. According to EPA the Reregistration Eligibility Decision (RED) is scheduled for May 2005.[45] On June 23, 2004, the EPA released to the public a series of risk assessment documents summarizing current data on the human health and environmental effects of 2,4-D.[46] This began a comment period during which the EPA will accept statements from any interested parties, which will then be considered in the final reregistration decision. As part of the reregistration process, EPA also required over 200 new studies on 2,4-D.[47] A group of major manufacturers of 2,4-D set up the "Industry Task Force II on 2,4-D Research Data" which has now funded 270 of these studies.[48] According to EPA, there are still several data gaps in the current 2,4-D research. The risk assessment indicates that a 28-day inhalation study is needed because there are no data available on the effects of repeated inhalation of 2,4-D. A developmental neurotoxicity study is also needed, as well as a 2-generation reproductive study that addresses endocrine disruptor concerns.[49]

BEYOND PESTICIDES
701 E Street, S.E., Suite 200, Washington DC 20003
202-543-5450 (v) • 202-543-4791 (f)
info@beyondpesticides.org • www.beyondpesticides.org

2,4-D *chemicalWATCH* Factsheet Bibliography

[1] US EPA. 2003. Pesticide Industry Sales and Usage Report: 1998-1999 Market Estimates. Office of Pesticide Programs, Washington DC. <http://www.epa.gov/oppbead1/pestsales/99pestsales/table_of_contents1999.html> (June 21, 2004)

[2] Pesticide Action Network UK. 1997. 2,4-D Factsheet. <http://www.pan-uk.org/pestnews/Actives/24d.htm> (June 21, 2004)

[3] National Pesticide Information Center (NPIC). 2004. Pesticide Fact Sheet for 2,4-D. <http://npic.orst.edu/factsheets/2_4-D.pdf> (June 21, 2004)

[4] Hess, F.D. 1993. Herbicide effects on plant structure, physiology and biochemistry. In *Pesticide interactions in crop production: Beneficial and deleterious effects*, ed. Altman, J. Boca Raton: CRC Press.

[5] See Ref 3

[6] Extension Toxicology Network (ETN). 1996. Pesticide Information Profile for 2,4-D. <http://extoxnet.orst.edu/pips/24-D.htm> (June 21, 2004)

[7] Rudel, Ruthann, et al. "Phthalates, Alkylphenols, Pesticides, Polybrominated Diphenyl Ethers, and Other Endocrine-Disrupting Compounds in Indoor Air and Dust." *Environmental Science and Technology* 37(20): 4543-4553.

[8] Nishioka, M., et al. "Distribution of 2,4-D in Air and on Surfaces inside Residences after Lawn Applications: Comparing Exposure Estimates from Various Media for Young Children." Environmental Health Perspectives 109(11): 1185-1191.

[9] See Ref 6

[10] US EPA. 2003. 2,4-D (2,4-Dichlorophenoxyacetic Acid). Technology Transfer Network- Air Toxics Website. <http://www.epa.gov/ttn/atw/hlthef/dioxyac.html> (June 21, 2004)

[11] See Ref 6 and Ref 10

[12] See Ref 6

[13] US EPA. 2000. List of chemicals evaluated for carcinogenic potential. Office of Pesticide Programs. Washington DC.

[14] Cox, Caroline. 1999. Herbicide Factsheet: 2,4-D: Toxicology, Part 2. *Journal of Pesticide Reform* 19(2): 14-19.

[15] S.K. Hoar, et al. 1986. Agricultural Herbicide Use and a Risk of Lymphoma and Soft-Tissue Sarcoma. *Journal of the American Medical Association* 256(9): 1141-1147.

[16] Zahm, S.H. 1990. A case control study of non-Hodgkin's lymphoma and the herbicide 2,4-D in Eastern Nebraska. *Epidemiology* 1(5): 349-356.

[17] Pesticide and Toxic Chemical News. 1986. 2,4-D produces rare brain tumor in male rats, EPA officials note. June 25, 1986.

[18] Hayes, H.M. et al. 1991. Case-control study of canine malignant lymphoma: positive association with dog owner's use of 2,4-D herbicides. *Journal of the National Cancer Institute* 83(17): 1226-1231.

[19] Hansen, W.H. et al. 1971. Chronic toxicity of 2,4-D in rats and dogs. *Toxicology and Applied Pharmacology* 20: 122-129.

[20] Page, D.L. 1996. 2,4-D Passes EPA Re-registration Muster. *Wheat Life* 29(9).

[21] Cox, Caroline. 1999. Herbicide Factsheet, 2,4-D: Toxicology Part 1. *Journal of Pesticide Reform* 19 (1): 14-19.

[22] Leonard, C. et al. 1997. "Golf Ball Liver": Agent orange hepatitis. *Gut* 40: 687-688; Johnston, J., et al. 1998. "Golf Ball Liver": A cause of chronic hepatitis? *Gut* 42: 143-146.

[23] Colborn, T., Saal, F.S. and Soto, A.M. 1993. Developmental Effects of Endocrine-Disrupting Chemicals in Wildlife and Humans, *Environmental Health Perspectives* 101(5): 378-384.

[24] National Institute of Health Sciences. 1999. List of endocrine disrupting pesticides. <http://www.nihs.go.jp/hse/endocrine-e/paradigm/pesticide/pest-list-e.html> (June 21, 2004)

[25] Florsheim, W.H. and S.M. Velcoff. 1962. Some effects of 2,4-dichlorophenoxyacetic acid on thyroid function in the rat: Effects on iodine accumulation. *Endocrinology* 72: 327-333; Liu, R.C. et al. 1996. The direct effect of hepatic peroxisome proliferators on rat Leydig cell function in vitro. *Fundamental Applied Toxicology* 30: 102-108.

[26] Figgs, L. et al. 1998. Occupational exposure to the herbicide 2,4-dichlorophenoxyacetic acid is associated with increased lymphocyte replicative index. *Proceedings of the American Association for Cancer Research* 39: 337-338.

[27] Atanassov, K. 1992. Effect of the herbicide Schpritshormit (Na salt of 2,4-D) on the karotype of the domesticated rabbit. *Animal science* 29: 54-61.

[28] Health Canada. 1999. Herbicide Residues Found in Semen. *Farm Family Health Newsletter*. Population and Public Health Branch. Vol. 7, No. 2. <http://www.hc-sc.gc.ca/pphb-dgspsp/publicat/ffh-sfa/vol7-2/ff7-2h_e.html> (June 21, 2004)

[29] See Ref. 6

[30] Garry, V.F. 1996. Pesticide appliers, biocides, and birth defects in rural Minnesota. *Environmental Health Perspectives* 104: 394-399.

[31] Schreinemachers, D. M. 2003. Birth malformations and other adverse perinatal outcomes in four US wheat-producing states. *Environmental Health Perspectives* 111(9): 1259-1264.

[32] See Ref. 2 and Ref. 6

[33] Duffard, R., L. Traini, and A. Evangelista de Duffard. 1981. Embryotoxic and teratogenic effects of phenoxy herbicides. *Acta Physiol. Latinoam.* 31: 39-42; Lutz, H. and Y. Lutz-Ostertag. 1972. The action of different pesticides on the development of bird embryos. *Adv. Exp. Med. Biol.* 27: 127-150.

[34] World Health Organization. 1989. 2,4-Dichlorophenoxyacetic acid (2,4-D)- Environmental aspects. Geneva, Switzerland.

[35] Wang, Y., C. Jaw, and Y. Chen. 1994. Accumulation of 2,4-D and glyphosate in fish and water. *Water Air Soil Pollut*. 74: 397-403.

[36] Roberts, B.L. and H.W. Dorough. 1984. Relative toxicity of chemicals to the earthworm. *Environmental Toxicology and Chemistry* 3:67-78.

[37] Cox, Caroline. 1999. Herbicide Factsheet: 2,4-D: Ecological Effects. *Journal of Pesticide Reform* 19(3): 14-19.

[38] Adams, J.B. and M.E. Drew. 1965. Grain aphids in New Brunswick III, Aphid populations in herbicide treated oat fields. *Canadian Journal of Zoology* 43: 789-794.

[39] See Ref. 37

[40] See Ref. 21

[41] See Ref. 6

[42] See Ref. 6 and Ref. 14

[43] US Dept of Health and Human Services. 1998. Toxicological profile for chlorinated dibenzo-p-dioxins. Public Health Service, Agency for Toxic Substances and Disease Registry; US EPA. 1994. Estimating exposure to dioxin-like compounds, Vol. II: Properties, sources, occurrence and background exposures. Office of Research and Development. Review draft. Washington DC, June.

[44] Washington Depts. of Ecology, Agriculture, and Health. 1998. Screening survey for metals and dioxins in fertilizers, soil amendments, and soils in Washington state. Olympia WA.

[45] US EPA. 2004. Pesticide Reregistration Status. Office of Pesticide Programs. <http://cfpub.epa.gov/oppref/rereg/status.cfm> (June 21, 2004)

[46] US EPA. 2004. 2,4-Dichlorophenoxyacetic Acid, Availability of Risk Assessment. *Federal Register*. Wednesday, June 23, 2004. 69(120): 35019-35021.

[47] Hammond, L. 1996. New Perspectives on an Essential Product: 2,4-D. *Down to Earth* 50(2).

[48] See Ref. 20

[49] US EPA. Office of Pesticide Programs. Overview of 2,4-D Risk Assessments. June 18, 2004. EPA Docket Number: OPP-2004-0167-0002; Toxicology Chapter for the Reregistration Eligibility Decision Document. Prepared by Linda L. Taylor. EPA Docket Number: OPP-0167-0019.

Updated July 2004

BEYOND PESTICIDES
701 E Street, S.E., Suite 200, Washington DC 20003
202-543-5450 (v) • 202-543-4791 (f)
info@beyondpesticides.org • www.beyondpesticides.org