

NORTHWEST COALITION FOR ALTERNATIVES TO PESTICIDES

JOURNAL OF PESTICIDE REFORM

SPRING 2004 • VOLUME 24, NUMBER 1



Solving aphid problems • Coping with mice
Hazards of the herbicide mecoprop

NORTHWEST COALITION FOR ALTERNATIVES TO PESTICIDES

JOURNAL OF PESTICIDE REFORM

SPRING 2004 • VOLUME 24, NUMBER 1

STEENBOCK
MEMORIAL LIBRARY

APR 08 2004

U.W. - MADISON

● TABLE OF CONTENTS

News from NCAP

- 2 Potato Grower Successfully Replaces Soil Fumigation with a Green Manure Crop
- 3 Working Toward Pesticide-Free Parks in Portland
- 4 NCAP Continues Fighting to Protect Salmon

News from Around

- 5 A Better Way to Keep Insects out of Aircraft
- 5 Coming Soon: Roundup Ready Grass on Golf Courses

Alternatives

- 6 A Mouse- and Pesticide-Free House
- 8 Managing Aphid Problems without Pesticides

Herbicide Factsheet

- 10 Mecoprop (MCP)

Research

- 15 Working in Apple Orchards Means More Pesticides for Children and Adults

Letters to the Editor

- 16 From Our Readers



NORTHWEST COALITION FOR ALTERNATIVES TO PESTICIDES

P.O. BOX 1393; EUGENE, OREGON 97440-1393

The *Journal of Pesticide Reform* is published by the Northwest Coalition for Alternatives to Pesticides. NCAP is a nonprofit tax-exempt organization. Donations are tax-deductible.

The NCAP office is located at 1249 Willamette St.; Eugene, OR 97401. Phone: (541) 344-5044. E-mail: info@pesticide.org. Fax: (541) 344-6923. The address of NCAP's web page is <http://www.pesticide.org>.

Permission is granted to reproduce any information found in this publication, except reprinted or copyright articles. Please credit NCAP and the *Journal of Pesticide Reform*.

Some articles in the *Journal of Pesticide Reform* are indexed by AGRICOLA and CAB Abstracts.

JPR is printed on recycled paper.

ISSN: 0893-357X



Editor

Caroline Cox

Contributing Authors

NCAP staff

Graphic Design and Production

Kate Pryka

Cover Artist

Mary Rounds

Contributing Photographers

Bureau of Land Management,
Centers for Disease Control and
Prevention, City of Seattle, Fred
Hutchinson Cancer Research
Center, U.S. Air Force, U.S.
Department of Agriculture

NCAP Staff

Norma Grier, *Executive Director*
Becky Long, *Development Director*
Jody Rolnick, *Grants Coordinator*
Edward Winter, *Financial Manager*
Kay Rumsey, *Librarian*
Aimee Code, *Right to Know, Clean Water*
Pollyanna Lind, *Clean Water*
Megan Kemple, *Public Education*
Jennifer Miller, *Sustainable Agriculture*
Caroline Cox, *Editor, Staff Scientist*

NCAP's Board of Directors

Jim Barngrover
Jean Cameron
Woody Deryckx
Gail Gutsche
Sonya Lunder
Soozie Nichol
John O'Connor
Randy Selig



a member of EarthShare
NEW ORLEANS

● HERBICIDE FACTSHEET

MECOPROP (MCPP)

Mecoprop (MCPP) is a common lawn care herbicide. The U.S. Environmental Protection Agency estimates that 5 million pounds are used every year on U.S. lawns. It is typically sold in products that are combinations of several related herbicides and as "weed and feed" products.

Part of the phenoxy herbicide chemical family, mecoprop kills plants by imitating naturally occurring plant growth hormones.

Symptoms of exposure to mecoprop include burning skin and eyes, nausea, dizziness, and headaches.

In laboratory tests, mecoprop has inhibited the synthesis of DNA (the molecules that contain genetic information), interfered with blood clotting, and inhibited the production of important components of the immune system.

Laboratory tests using a commercial mecoprop-containing herbicide showed that the fertility of mice who drank water contaminated with low levels of the herbicide was less than that of mice who drank uncontaminated water. Even the lowest dose level tested in this experiment reduced litter size.

A regional study in Canada found that exposure to mecoprop was associated with an increased risk of the cancer non-Hodgkin's lymphoma.

Mecoprop is frequently found in urban streams. One study (done in King County, Washington) found mecoprop in every urban stream sample analyzed.

In both greenhouse and field studies, mecoprop has caused plant diseases to infect more plants or produce more spores.

BY CAROLINE COX

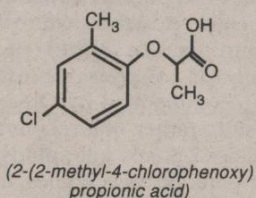
Mecoprop, also known as MCPP (see Figure 1), is one of the most commonly used lawn care herbicides in the U.S. According to the U.S. Environmental Protection Agency (EPA), over 3 million applications,¹ totalling 5 million pounds,² of mecoprop are used in yards and gardens every year. Only two popular yard and garden herbicides (2,4-D and glyphosate) are used more widely.²

As a selective herbicide that targets broadleaf plants, mecoprop typically is used to kill broadleaf plants growing in lawns and turf. Farmers also use it to kill these weeds in cereal crops.³

Mecoprop is often sold in combinations of several related herbicides (including 2,4-D, dicamba, or MCPA).

Caroline Cox is NCAP's staff scientist.

Figure 1
Mecoprop (MCPP)



It is also sold in "weed and feed" products, in which several herbicides are combined with fertilizers. Many major pesticide companies market mecoprop-containing products for home lawns, and there are also products designed for lawn care professionals and turf managers. Mecoprop is sold under a wide array of brand names.⁴

Mecoprop's ability to kill plants was first reported in 1956. It was

registered for use in the U.S. in 1964.⁵

Mode of Action

Mecoprop belongs to the phenoxy herbicide family. Its better-known chemical relatives include 2,4-D and 2,4,5-T. Like all herbicides in this chemical family, mecoprop imitates naturally occurring plant growth hormones called auxins. It causes shoots of broadleaf plants to grow in an elongated and distorted manner. About a week after exposure, the plant collapses, withers, and dies.³

Inert Ingredients

Like most pesticides, commercial mecoprop herbicides contain ingredients in addition to mecoprop which, according to U.S. pesticide law, are called "inert."⁶ In general, they are not identified and not included in most of the testing required in order to register these pesticides.⁷ Hazards of some inerts in commercial mecoprop herbicide products are summarized in

"Inert Ingredients," at right.

Symptoms of Exposure to Mecoprop

Symptoms reported by state health agencies investigating incidents when people were exposed to mecoprop-containing herbicides include red and burning skin, blistered skin, tearing, burning and irritated eyes, blurred vision, nausea,⁸ dizziness, headaches, chest pain, and difficulty breathing.⁹

All these incidents involved herbicides that contained 2,4-D in addition to mecoprop. Most also contained dicamba.^{8,9} These combinations are typical of mecoprop-containing herbicides.

According to the Health and Consumer Protection Directorate-General of the European Commission, mecoprop is irritating to skin and severely irritating to eyes.¹⁰

Ability to Cause Genetic Damage

The National Institute for Occupational Safety and Health labels mecoprop as a "mutagen"¹¹ because it inhibited the synthesis of DNA in a laboratory study of mice.¹¹ DNA is the "molecular basis of heredity,"¹² the molecules that contain genetic information. A single dose of mecoprop reduced DNA production by 60 percent.¹³ (See Figure 2.)

In addition, tests conducted for a mecoprop manufacturer as part of the process of registering it as a pesticide, showed other types of genetic damage. These included chromosome damage in bone marrow cells in hamsters and human blood cells, as well as a kind of genetic damage called sister chromatid exchanges in hamster bone marrow cells.¹⁴ (Sister chromatid exchanges are exchanges of DNA within a chromosome as it duplicates.¹⁵)

Liver and Kidney Damage

In 1994, EPA listed mecoprop as a toxic chemical under the Emergency Planning and Community Right-to-Know Act because of its toxicity to the liver and kidneys.¹⁶ In a three month feeding study with rats, mecoprop doses of 9 milligrams per kilogram (mg/kg) of body weight per day

INERT INGREDIENTS

Hazards posed by inert ingredients in household mecoprop-containing herbicides¹ include the following:

Morpholine is a severe eye and skin irritant. It is labeled as a "mutagen" by the National Institute for Occupational Safety and Health because it caused genetic damage in laboratory tests. It also damaged the liver and kidney.²

8-hydroxyquinoline sulfate is labeled as a "mutagen" by the National Institute for Occupational Safety and Health because it caused genetic damage in human blood cells.³

Methyl carbitol reduced fertility in laboratory tests.⁴

Hexylene glycol is a severe eye irritant. It also reduced the functioning of the kidneys and caused muscle weakness in laboratory tests.⁵

Quartz silica is classified as a carcinogen by the International Agency for Research on Cancer. The National Institute for Occupational Safety and Health labels it as a "mutagen" because it caused genetic damage in laboratory tests.⁶

1. U.S. EPA. 2004. Response to Freedom of Information Act request RIN-1178-99. Received by NCAP in February 2004.
2. National Institute for Occupational Safety and Health. 2002. Registry of Toxic Effects of Chemical Substances: Morpholine. www.cdc.gov/niosh/rtecs/qd62ccf8.html.
3. National Institute for Occupational Safety and Health. 1997. Registry of Toxic Effects of Chemical Substances: 8-Quinololinol, sulfate (2:1) (salt). www.cdc.gov/niosh/rtecs/vc7e09a0.html.
4. National Institute for Occupational Safety and Health. 2002. Registry of Toxic Effects of Chemical Substances: Ethanol, 2-(2-methoxyethoxy)-. www.cdc.gov/niosh/rtecs/kl5d75c8.html.
5. National Institute for Occupational Safety and Health. 2002. Registry of Toxic Effects of Chemical Substances: 2,4-pentandiol, 2-methyl-. www.cdc.gov/niosh/rtecs/sac5c10.html.
6. National Institute for Occupational Safety and Health. 2002. Registry of Toxic Effects of Chemical Substances: Silica, crystalline-quartz. www.cdc.gov/niosh/rtecs/vv6fd8d0.html.

caused a change in liver and kidney weights.¹⁷

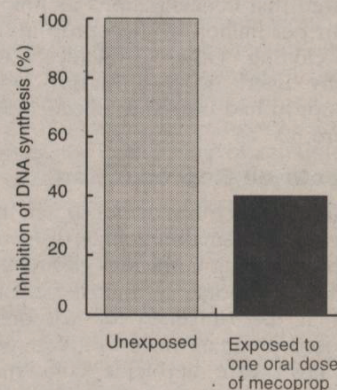
Anemia

Exposure to mecoprop has caused the development of anemia in tests with laboratory animals. As part of mecoprop's registration process, a pesticide manufacturer sponsored two studies with dogs. In one, dogs were fed mecoprop for 12 months; in the other, for three months. In both studies, the amount of hemoglobin in the dogs' blood decreased at doses of about 20 mg/kg.¹⁴ Hemoglobin is the oxygen-carrying molecule in the blood, and deficiencies in hemoglobin result in anemia.¹²

Blood Clotting

Researchers at the University of Kuopio (Finland) noticed that bleeding is a symptom of poisoning by phenoxy herbicides. They then studied the effects of eight of these herbicides including mecoprop on "platelet

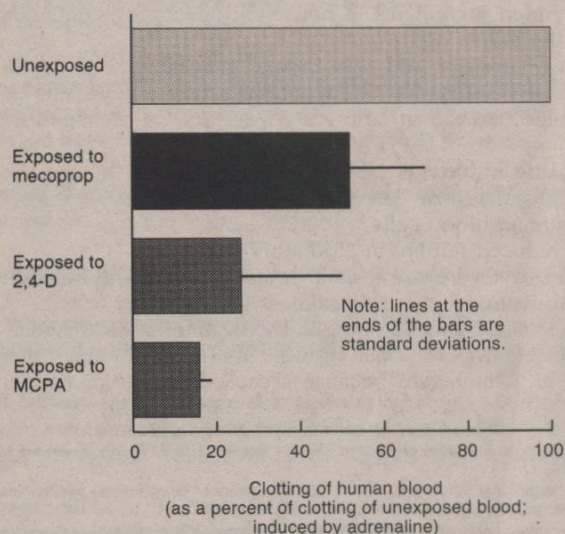
Figure 2
Genetic Damage Caused by Mecoprop



Source: Seiler, J.P. 1979. Phenoxyacids as inhibitors of testicular DNA synthesis in male mice. *Bull. Environ. Contam. Toxicol.* 21:89-92.

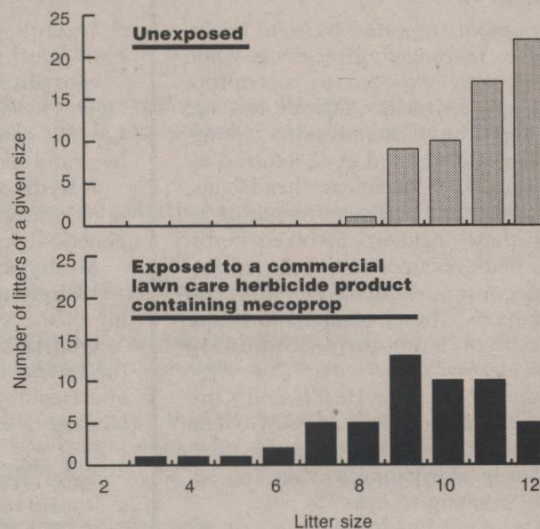
In a study of mice, mecoprop inhibited synthesis of DNA, molecules that carry genetic information.

Figure 3
Effect on Blood Clotting



Source: Elo, H.A., T. Luoma, and P. Ylitalo. 1991. Inhibition of human and rabbit platelet aggregation by chlorophenoxy herbicides. *Arch. Toxicol.* 65:140-144.

Figure 4
Effect on Fertility



Source: Cavieres, M.F., J. Jaeger, and W. Porter. 2002. Developmental toxicity of a commercial herbicide mixture in mice: 1. Effects on embryo implantation and litter size. *Environ. Health Persp.* 110:1081-1085.

Two toxicological problems identified in laboratory tests with mecoprop are disruption of blood clotting and reduced fertility. In samples of human blood, clotting is inhibited when the blood was exposed to mecoprop. In another study, pregnant mice given water with low levels of a lawn care herbicide that contained mecoprop had fewer offspring than mice given uncontaminated water.

aggregation" in human blood. Platelets are a component of blood that assists in clotting. Results of this study showed that concentrations as low as 1 part per million of mecoprop inhibited clotting. Other herbicides commonly used in combination with mecoprop had the same effect.¹⁸ (See Figure 3.)

Effects on Reproduction

New research has demonstrated that exposures to small amounts of mecoprop-containing herbicides can reduce fertility in laboratory animals. Zoologists at the University of Wisconsin exposed pregnant mice to a commercial lawn care herbicide containing mecoprop, 2,4-D, and dicamba in the animals' drinking water. They found that litter size was reduced even at the lowest dose level tested in this experiment, 0.004 mg/kg per day. In unexposed animals, 12 was the most common litter size; in exposed ani-

mals this number dropped to 9 or 10.¹⁹ (See Figure 4.)

Mecoprop's ability to reduce fertility in laboratory animals had also been demonstrated in a study done in the 1980s in Germany.¹¹

Carcinogenicity (Ability to Cause Cancer)

The link between exposure to phenoxy herbicides, including mecoprop, and cancer has been controversial for decades. In 1987, based on a series of studies of people who had been occupationally exposed to these herbicides, the International Agency for Research on Cancer (IARC) classified phenoxy herbicides as "possibly carcinogenic to humans" (IARC's Group 2B). IARC has not updated its classification since then.²¹

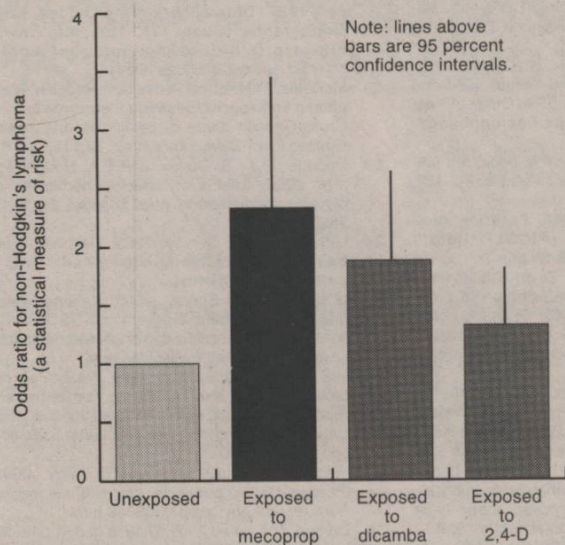
Recent research supports concerns about the carcinogenicity of mecoprop. In 2001, scientists at the University of Saskatchewan and several Canadian

cancer institutes compared pesticide use by hundreds of Canadian men diagnosed with the cancer non-Hodgkin's lymphoma (NHL) with pesticide use by a comparison group of men who didn't have cancer. The men lived in six Canadian provinces and were exposed to mecoprop either at work or at home. They found that "the risk of NHL was statistically significantly increased"²² by exposure to three phenoxy herbicides: mecoprop, 2,4-D, and dicamba. The odds ratio (a statistical measure of the increased cancer risk) was over 25 percent larger for mecoprop than for the other two phenoxy herbicides.²² (See Figure 5.)

Effects on Immune System Function

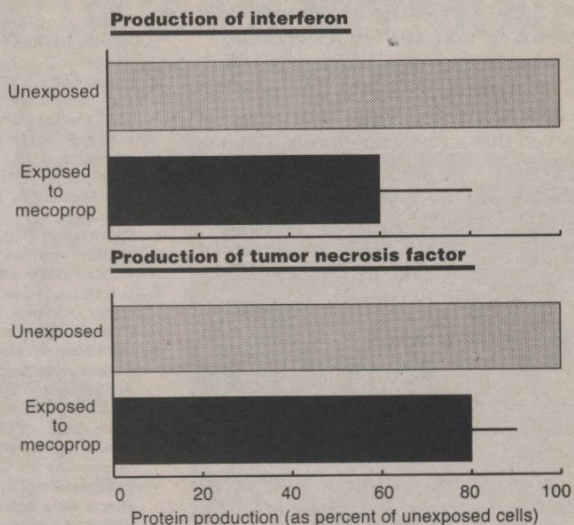
One "sensitive indicator for monitoring perturbation [disturbance] of the immune system" is the production of proteins that are used by the immune system to protect us from disease.

Figure 5
Effect on Cancer Risk



Source: McDuffie, H.H. et al. 2001. Non-Hodgkin's lymphoma and specific pesticide exposures in men: Cross-Canada study of pesticides and health. *Cancer Epid. Biomarkers Prev.* 10:1155-1163.

Figure 6
Effect on Immune System Function



Source: Hooghe, R.J., S. Devos, and E.L. Hooghe-Peters. 2000. Effects of selected herbicides on cytokine production in vitro. *Life Sci.* 66: 2519-2525.

Mecoprop exposure has also been linked with cancer and immune system problems. In a Canadian study, exposure to mecoprop (and other herbicides with which it is commonly used) increased the risk of the cancer non-Hodgkin's lymphoma. In a second study, toxicologists working with human blood cells found that production of two important components of the immune system is inhibited by mecoprop.

According to toxicologists from the Flemish Institute for Technological Research and the Free University of Brussels (Belgium), mecoprop has this kind of effect.²³ In human white blood cells, these toxicologists showed that mecoprop inhibited production of interferon,²³ an antiviral protein,¹² and tumor necrosis factor,²³ a protein that causes destruction of some tumor cells and activates white blood cells.¹² (See Figure 6.)

Water Contamination

During the 1990s, the U.S. Geological Survey began a monitoring program, the first of its kind, looking for pesticides in rivers and streams across the U.S.²⁴ Because mecoprop was not one of the pesticides analyzed in this program,²⁵ there are no national data about the extent of mecoprop contamination of rivers or streams. However, local monitoring efforts indicate that contamination of streams and rivers with mecoprop may be startlingly

common. For example, in urban streams in King County, Washington, a collaboration between the county, the state Department of Ecology, and USGS found mecoprop in every sample taken during spring rainstorms.²⁶ In Bellingham, Washington, the Department of Ecology found that mecoprop was the third most frequently detected pesticide (out of 19 total) in an urban stream.²⁷ An Environment Canada study of wetlands in Saskatchewan, Canada, found that mecoprop was one of the most commonly detected herbicides.²⁸ A second Canadian study, done at the Lethbridge Research Centre, found that mecoprop contaminated rainfall, particularly in urban areas.²⁹

One of the reasons that mecoprop often contaminates water is that its chemical characteristics make it very mobile in soil. According to the Oregon State University Extension Service, mecoprop's "pesticide movement ranking" is high.³⁰

While there are likely to be many sources of the mecoprop that contaminates water in a particular community, golf course mecoprop use is one identified source of contamination. Three different types of studies all showed that mecoprop contaminates the water leaving treated golf courses: intensive monitoring of a single golf course, extensive monitoring of multiple golf courses within a community, and measurements conducted on simulated golf courses. The studies were conducted by scientists at the University of Georgia, the U.S. Department of Agriculture, the Tokyo Metropolitan Research Laboratory of Public Health, and the Tokyo College of Pharmacy.^{31,32}

Effects on Aquatic Ecosystems

Several important components of aquatic ecosystems are harmed by mecoprop. Biologists at the University of Hull (United Kingdom) showed that

mecoprop is toxic to several species of freshwater bacteria that can play key roles in purifying water.³³ Diatoms, plankton that are abundant in freshwater and marine ecosystems,³⁴ are also sensitive to mecoprop. Studies submitted to EPA as part of mecoprop's registration as a pesticide showed that a concentration of 17 parts per billion kills diatoms.³⁵ Diatoms are ecologically significant: they account for about a quarter of all photosynthesis, are a major food resource for aquatic animals, and are a major source of atmospheric oxygen.³⁴

Effects on Birds

Use of mecoprop can impact birds when the herbicide kills plants that provide habitat for insects used as food by birds. A study by The Game Conservancy Trust (United Kingdom) showed that insects used as food by juvenile birds were less than half as abundant in areas treated with mecoprop (combined with two other broadleaf herbicides) than they were in untreated areas.³⁶

Effects on Plant Diseases

Both field and greenhouse experiments have demonstrated that mecoprop can promote plant disease.

Scientists at the ARC Weed Research Organization showed that treatment of winter wheat with mecoprop increased the incidence of take-all disease by 66 percent.³⁷

More recently, an Iowa State University horticulturist showed that a leaf spot fungus produced more spores on greenhouse-grown Kentucky bluegrass when the soil was treated with mecoprop than when the soil was untreated.³⁸

Effects on Mycorrhizal Fungi

Mecoprop can also damage mycorrhizal fungi, beneficial fungi that promote growth of many plant species and also help them resist stress. Researchers from the Swiss Federal Research Station for Fruit-Growing showed that mecoprop, applied at typical application rates, reduced from 80 percent to 35 percent the proportion of corn plants with viable mycorrhizal fungi.³⁹ ✱

References

- Whitmore, R.W., J.E. Kelly, and P.L. Reading. 1992. National home and garden pesticide use survey. Final report, vol. 1: Executive summary, results, and recommendations. Table G.1. Research Triangle Park NC: Research Triangle Institute.
- Donaldson, D., T. Kiely, and A. Grube. 2002. Pesticides industry sales and usage: 1998 and 1999 market estimates. U.S. EPA. Office of Pesticide Programs. www.epa.gov/oppbead1/pestsales. p 15.
- Ware, G.W. 2000. *The pesticide book*. 5th edition. Fresno CA: Thomson Publications. Pp. 122, 190, 345.
- Washington State Univ. 2003. Pesticide information center online (PICOL). <http://picol.cahe.wsu.edu/labels/labels.php>.
- U.S. EPA. Pesticides and Toxic Substances. 1988. Guidance for the reregistration of products containing mecoprop (MCP) as the active ingredient. Washington, D.C. p.6.
- Federal Insecticide, Fungicide and Rodenticide Act § 2(a) and 2(m).
- 40 Code of Federal Regulations § 158.340.
- Calif. Dept. of Pesticide Regulation. Worker Health and Safety Branch. 2004. Case reports received by the California Pesticide Illness Surveillance Program, 1999-2001, in which health effects were definitely, probably, or possibly attributed to exposure to mecoprop, alone or in combination. (Unpublished database printout.)
- Texas Dept. of Health. 2002. Possible pesticide exposure of employees at a government agency. Disease Prevention News 62. www.tdh.state.tx.us/phpep.
- European Commission, Health and Consumer Protection Directorate-General. 2003. Mecoprop: Commission working document. http://europa.eu.int/comm/food/plant/protection/evaluation/exist_subs_rep_en.htm.
- National Institute for Occupational Safety and Health. 1997. Registry of Toxic Effects of Chemical Substances: Propionic acid, 2-[(4-chloro-o-tolyl)oxyl]-. www.cdc.gov/niosh/rtecs/ue94c5f0.html.
- National Library of Medicine. 2003. MEDLINEplus health information Merriam-Webster medical dictionary. www.nlm.nih.gov/medlineplus/medlineplusdictionary.html.
- Seiler, J.P. 1979. Phenoxyacids as inhibitors of testicular DNA synthesis in male mice. *Bull. Environ. Contam. Toxicol.* 21:89-92.
- Calif. EPA. Dept. of Pesticide Regulation. Medical Toxicology Branch. 1999. Summary of toxicology data: MCP. www.cdpr.ca.gov/docs/toxsums/toxsumlist.htm.
- U.S. EPA. Prevention, Pesticides and Toxic Substances. 1998. Health effects test guidelines. OPPTS 870.5915. In vivo sister chromatid exchange assay. www.epa.gov/pesticides.
- U.S. EPA. 1994. Addition of certain chemicals; Toxic chemical release reporting; Community right-to-know; Final rule. *Fed. Reg.* 59: 61437.
- U.S. EPA. 1994. Addition of certain chemicals; Toxic chemical release reporting; Community right-to-know; Proposed rule. *Fed. Reg.* 59: 1788-1859.
- Elo, H.A., T. Luoma, and P. Ylitalo. 1991. Inhibition of human and rabbit platelet aggregation by chlorophenoxy herbicides. *Arch. Toxicol.* 65:140-144.
- Cavieses, M.F., J. Jaeger, and W. Porter. 2002. Developmental toxicity of a commercial herbicide mixture in mice: 1. Effects on embryo implantation and litter size. *Environ. Health Persp.* 110:1081-1085.
- Roll, R. and G. Matthiaschek. 1983. Comparative studies on the embryotoxicity of 2-methyl-4-chlorophenoxyacetic acid, mecoprop and dichloroprop in MNRI mice. *Arzneimittelforschung* 33:1479-1483. (Abstract.)
- International Agency for Research on Cancer. 1987. Chlorophenoxy herbicides. IARC Monographs (Suppl. 7): 156. <http://www-cie.iarc.fr/htdocs/monographs/suppl7/chlorophenoxyherbicides.html>.
- McDuffie, H.H. et al. 2001. Non-Hodgkin's lymphoma and specific pesticide exposures in men: Cross-Canada study of pesticides and health. *Cancer Epidemiol. Biomarkers Prev.* 10:1155-1163.
- Hooghe, R.J., S. Devos, and E.L. Hooghe-Peters. 2000. Effects of selected herbicides on cytokine production in vitro. *Life Sci.* 66: 2519-2525.
- U.S. Geological Survey. 2003. National water-quality assessment program. <http://water.usgs.gov/nawqa/>.
- U.S. Geological Survey. 2003. National water-quality assessment program: USGS NAWQA constituents - pesticides. <http://water.usgs.gov/nawqa/constituents/pesticides.html>.
- U.S. Geological Survey. 1999. Pesticides detected in urban streams during rainstorms and relations to retail sales of pesticides in King County, Washington. <http://wa.water.usgs.gov/pubs/fs/097-99/>.
- Washington State Dept. of Ecology. 2003. Padden Creek pesticide study: Final report. www.ecy.wa.gov/biblio/0303048.html.
- Donald, D.A. et al. 2001. Diffuse geographic distribution of herbicides in northern prairie wetlands. *Environ. Toxicol. Chem.* 20:273-279.
- Hill, B.D. et al. 2002. Herbicides in Alberta rainfall as affected by location, use and season: 1999-2000. *Water Qual. Res. J. Canada* 37:515-542.
- Oregon State Univ. Extension. 1994. OSU Extension pesticide properties database. <http://npic.orst.edu/ppdmove.htm>.
- Suzuki, T. et al. 1998. Estimation of leachability and persistence of pesticides from point-source monitoring and model to predict pesticide leaching to groundwater. *Environ. Sci. Technol.* 32:920-929.
- Ma, O. et al. 1999. Water runoff and pesticide transport from a golf course fairway: Observations vs. Opus model simulations. *J. Environ. Qual.* 28:1463-1473.
- Milner, C.R. and Goulder, R. 1986. Comparative toxicity of chlorophenols, nitrophenols, and phenoxyalkanoic acids to freshwater bacteria. *Bull. Environ. Contam. Toxicol.* 37:714-718.
- Univ. of California Museum of Paleontology. Undated. Diatoms: Life history and ecology. <http://www.ucmp.berkeley.edu/chromista/diatoms/diatomh.html>.
- U.S. EPA. Office of Pesticide Programs. 2003. Pesticide ecotoxicity database. Unpublished database. Received from EPA on May 13.
- Chiverton, P.A. and N.W. Sotherton. 1991. The effects on beneficial arthropods of the exclusion of herbicides from cereal crop edges. *J. Appl. Ecol.* 28:1027-1039.
- Tottman, C.R. and W. Thompson. 1978. The influence of herbicides on the incidence of take-all disease (*Gaeumannomyces graminis*) on the roots of winter wheat. *Proc. 1978 Brit. Crop Protec. - Weeds*: 609-615.
- Hodges, C.F. 1992. Vegetative growth and sporulation of *Bipolaris sorokiniana* on infected leaves of *Poa pratensis* exposed to postemergence herbicides. *Can. J. Bot.* 70:568-570.
- Dehn, B., M. Bodmer, and H. Schüepp. 1990. Influence of Herbicides on VA mycorrhizal propagation in soil. *Symbiosis* 9:223-227.