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Contents of Records Package

EXTOXNET Infobase, accessed November 7, 2005. http://extoxnet.orst.edu/

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Table 1. List of Chemicals included in this records package

Chemical	Chemical Abstract Service (CAS) Registry Number
Alachlor *Syn*: 2-chloro-2';6';-diethyl-N-(methoxymethyl)acetanilide	15972-60-8
Atrazine *Syn*: 2-chloro-2';6';-diethyl-N-(methoxymethyl)acetanilide	1912-24-9
D[2,4-] *Syn*: 2,4-dichlorophenoxyacetic acid	94-75-7
Dalapon *Syn*: 2,2-dichloropropionic acid	75-99-0
DB[2,4-] *Syn*: 4-(2,4-dichlorophenoxy)butanoic acid; 2,4-dichlorophenoxybutyric acid	94-82-6
Dicamba *Syn*: 2-methoxy-3,6-dichlorobenzoic acid	1918-00-9
Dinoseb *Syn*: DNBP; 2,4-dinitro-6-sec-butylphenol (DNBP)	88-85-7
Diquat Syn: 1-1'-ethylene-2,2'-bipyridinium-dibromide; Diquat dibromide	231-36-7 85-00-7 (cation)
Endothall *Syn*: aquathol K; 7-oxabicyclo(2,2,1)heptane-2,3-dicarboxylic acid	145-73-3
Glyphosate *Syn*: N-(phosphonomethyl)glycine; glyphosate acid	1071-83-6
MCPA *Syn*: Chloro-o-tolyloxyacetic[4-] Acid; 2-methyl-4-chlorophenoxyacetic acid	94-74-6
Mecoprop *Syn*: MCPP; 2-(4-chloro-2-methylphenoxy) propionic acid	93-65-2
Picloram *Syn*: 4-amino-3,5,6-trichloropicolinic acid	1918-02-1
Simazine *Syn*: 2-chloro-4,6-bis(ethylamino)-s-triazine	122-34-9

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The EXtension TOXicology NETwork

NEW Check out the EXTOXNET Frequently Asked Questions (FAQs)

NEW Check out the UC Davis Environmental Toxicology Newsletter

You may go directly to the "EXTOXNET Global Search and Browse" page.

So... Are you looking for a source of ***objective, science-based information about pesticides - written for the non-expert?*** The **EXTOXNET InfoBase** may be for you!

The EXTOXNET InfoBase provides a variety of information about pesticides. Access the HOT Pesticide Information Profiles (PIPs) for specific information on pesticides. HOT Toxicology Information Briefs (TIBs) contain a discussion of certain concepts in toxicology and environmental chemistry. Other topic areas include: Toxicology Issues of Concern (TICs), Factsheets, News about Toxicology Issues, Newsletters, Resources for Toxicology Information, and Technical Information.

Information in these topic areas primarily has been developed by toxicologists and chemists within the Extension Service of the land-grant universities listed below. A major goal has been to develop unbiased information in a form understandable by the non-expert, and to make that information fully searchable and selectively retrievable.

Explore EXTOXNET!

- Background information about EXTOXNET.
- Go directly to the "EXTOXNET Global Search and Browse" page.

EXTOXNET is a cooperative effort of University of California-Davis, Oregon State University, Michigan State University, Cornell University, and the University of Idaho.
Primary files are maintained and archived at Oregon State University.

Terry L. Miller
extoxnet@ace.orst.edu

EXTOXNET - EXtension TOXicology NETwork

WHAT IS EXTOXNET?

The EXTension TOXicology NETwork (EXTOXNET) is an effort of University of California, Davis, Oregon State University, Michigan State University, Cornell University, and the University of Idaho. Some of the goals of EXTOXNET are to stimulate dialog on toxicology issues, develop and make available information relevant to extension toxicology, and facilitate the exchange of toxicology-related information in electronic form, accessible to all with access to the Internet. The EXTOXNET InfoBase is accessible via the World Wide Web (WWW).

WHAT KINDS OF INFORMATION ARE PROVIDED BY EXTOXNET?

Various types of pesticide toxicology and environmental chemistry information are available from the WWW server at Oregon State University. These include: discussions of toxicological issues of concern (TICs); toxicology newsletters; other resources for toxicology information; toxicology fact sheets; Pesticide Information Profiles (PIPs); and Toxicology Information Briefs (TIBs). In addition, various closed mailing groups are utilized for EXTOXNET participants to facilitate communication and response to requests for information.

HOW DOES ONE RETRIEVE INFORMATION FROM EXTOXNET?

Information may be retrieved via the WWW by use of a Web browser. The brower - Netscape 2.0 - is recommended, since many of the EXTOXNET web pages have been developed using extensions to html supported by Netscape. The URL is:

URL http://ace.orst.edu/info/extoxnet/

Users without access to a web browser may telnet to:

ace.orst.edu

At the 'login:' prompt, type 'lynx', and enter a return for the 'password' prompt. Then select 'G)o' (i.e., type 'G') and enter the URL above.

The EXTOXNET InfoBase is searchable globally as well as within each topic area, allowing the user to selectively view and retrieve information.

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Welcome to Search and Browse for EXTOXNET...
Pesticide Information Profiles (PIPs)

EXTOXNET is a cooperative effort of University of California-Davis, Oregon State University, Michigan State University, Cornell University, and the University of Idaho. Primary files are maintained and archived at Oregon State University.

This page allows both browsing and searching - select for details.

If you don't have a browser that can supports forms, you can still make a search.

Search...What would you like to search for?

- Search tips...

☑ Case insensitive ☐ Allow partial match to a word

Click here for detailed search options.

Submit search or Clear form

Browse... Browse these PIPs by 'clicking' on the title:

- WHAT ARE PIPs?
- 2,4-D - EXTOXNET PIP
- 2,4-DB - EXTOXNET PIP
- 4-AMINOPYRIDINE - EXTOXNET PIP
- DCPA, CHLORTHAL, CLORTHAL-DIMETHYL - EXTOXNET PIP
- MCPA - EXTOXNET PIP
- ABAMECTIN - EXTOXNET PIP
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- TRIALLATE - EXTOXNET PIP
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- TRICLOPYR - EXTOXNET PIP
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- VALIDAMYCIN - EXTOXNET PIP
- VERNOLATE - EXTOXNET PIP
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- ZIRAM - EXTOXNET PIP

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EXTOXNET

Extension Toxicology Network

Pesticide Information Profiles

A Pesticide Information Project of Cooperative Extension Offices of Cornell University, Oregon State University, the University of Idaho, and the University of California at Davis and the Institute for Environmental Toxicology, Michigan State University. Major support and funding was provided by the USDA/Extension Service/National Agricultural Pesticide Impact Assessment Program.

EXTOXNET primary files maintained and archived at Oregon State University

Revised June 1996

Alachlor

Trade and Other Names: Trade names of commercial herbicides containing alachlor include Alanex, Bronco, Cannon, Crop Star, Lariat, Lasso, and Partner. It mixes well with other herbicides such as Bullet, Freedom, and Rasta, and is found in mixed formulations with atrazine, glyphosate, trifluralin, and imazaquin.

Regulatory Status: Alachlor is a Restricted Use Pesticide (RUP). RUPs may be purchased and used only by certified applicators. The EPA categorizes it as toxicity class III - slightly toxic. However, alachlor products bear the Signal Word DANGER on their labels because of their potential to cause cancer in laboratory animals.

Chemical Class: aniline

Introduction: Alachlor is an aniline herbicide used to control annual grasses and broadleaf weeds in field corn, soybeans, and peanuts. It is a selective systemic herbicide, absorbed by germinating shoots and by roots. It works by interfering with a plant's ability to produce protein and by interfering with root elongation.

This compound is one of the most highly used herbicides in the U.S. Over 50 million pounds were applied annually in 1990. It is available as granules or emulsifiable concentrate.

Formulation: It is available as granules or emulsifiable concentrate.

Toxicological Effects:

- **Acute toxicity:** Alachlor is a slightly toxic herbicide. The LD50 of alachlor in rats is between 930 mg/kg and 1350 mg/kg. In the mouse, the LD50 is between 1910 and 2310 mg/kg [60]. The dermal LD50 in rabbits is 13,300 mg/kg, but some of the formulated materials can be more toxic, with dermal LD50 values ranging from 7800 to 16,000 mg/kg [58]. Skin irritation is slight to moderate. The inhalation LC50 in rats is reportedly greater than 23.4 mg/L for 6 hours of exposure [1,60].
- **Chronic toxicity:** A 90-day study on rats and dogs given diets containing low to moderate amounts of alachlor (1 to 100 mg/kg/day) showed no adverse effects [58]. However, a 6-month dog study showed liver toxicity at all doses above 5 mg/kg/day, and a 1-year study established that above 1 mg/kg/day, alachlor causes effects in the liver, spleen, and kidney. In 2-year rat studies, doses above 2.5 mg/kg/day caused irreversible degeneration of the iris and related eye structures [60].
- **Reproductive effects:** High oral doses (150 or 400 mg/kg/day) fed to rats during gestation resulted in maternal and fetal toxicity, but there was no indication that reproduction was affected [60,61]. Alachlor does not appear to cause reproductive effects.
- **Teratogenic effects:** Doses of up to 150 mg/kg/day fed to rabbits on days 7 through 19 of pregnancy did not result in any birth defects [6]. Similar studies in rats at doses up to 400 mg/kg/day did not result in birth defects, but toxic effects in the mothers

and offspring were seen at the highest dose. These data indicate that alachlor is not likely to cause birth defects.

- **Mutagenic effects:** Alachlor does not appear to be mutagenic. Mutagenicity assays with a variety of microbial strains at numerous concentrations of alachlor were all negative [60,61].
- **Carcinogenic effects:** Rats given high doses of alachlor developed stomach, thyroid, and nasal turbinate tumors [60]. An 18-month mouse study with doses from 26 to 260 mg/kg/day showed an increase of lung tumors at the highest dose for females but not males [60]. Because of inconsistencies in these studies, the oncogenic potential of alachlor is uncertain [62].
- **Organ toxicity:** Long-term studies in rats and dogs showed effects on the liver, spleen, and kidneys. The iris and lung have also been affected.

- **Fate in humans and animals:** Rats given a single low dose of alachlor (14 mg/kg) eliminated most of it within the first 48 hours [60]. Nearly all of the compound was eliminated in 10 days; a third to a half of the alachlor and its breakdown products were excreted in urine and nearly a half in feces. Rats absorbed close to 75% of a single low dose (14 mg/kg) through skin while only 8 to 10% was absorbed through the skin in monkeys [60].

Ecological Effects:

- **Effects on birds:** Alachlor is slightly to practically nontoxic to wildfowl. Alachlor has a 5-day dietary LC50 of greater than 5000 ppm in young mallard ducks and bobwhite quail [58]. The LD50 of alachlor in other mallard ducks was greater than 2000 mg/kg [63]. The LC50 of alachlor in pheasants is greater than 10,000 ppm [8].
- **Effects on aquatic organisms:** Alachlor is moderately toxic to fish. The LC50 (96-hour) for alachlor is 2.4 mg/L in rainbow trout, 4.3 mg/L in bluegill sunfish, 6.5 mg/L in catfish, and 4.6 mg/L in carp [1,8]. It is only slightly toxic to crayfish, with a LC50 (96-hour) of 19.5 mg/L [8,37]. The bioaccumulation factor in the channel catfish is 5.8 times the ambient water concentration, indicating that alachlor is not expected to accumulate appreciably in aquatic organisms [8].
- **Effects on other organisms:** Alachlor is not toxic to bees. It is practically nontoxic to earthworms [61].

Environmental Fate:

- **Breakdown in soil and groundwater:** Alachlor has a low persistence in soil, with a half-life of about 8 days [1,8]. The main means of degradation is by soil microbes [58]. It has moderate mobility in sandy and silty soils, and thus can migrate to groundwater [60]. The largest groundwater testing program for a pesticide, the National Alachlor Well Water Survey, was conducted throughout the last half of the 1980s. Over 6 million private and domestic wells were tested for the presence of alachlor. Less than 1% of all of the wells had detectable levels of alachlor [64]. In the wells where the compound was detected, concentrations ranged from 0.1 to 1.0 ug/L, with the majority having concentrations around 0.2 ug/L [64].
- **Breakdown in water:** Alachlor breaks down rapidly in natural water, primarily due to the action of microorganisms. The breakdown rate is much slower in water with no oxygen [58].
- **Breakdown in vegetation:** Absorption is primarily by germinating shoots and it is readily translocated throughout the plant [37]. Higher concentrations appear in the vegetative parts than in the reproductive parts of the plant. Alachlor is rapidly metabolized to water-soluble products in plants [19]. It is almost completely metabolized within 10 days [8].

Physical Properties:

- **Appearance:** Alachlor is a colorless to yellow crystal compound [1].
- **Chemical Name:** 2-chloro-2',6'-diethyl-N-(methoxymethyl) acetanilide [1]
- **CAS Number:** 15972-60-8
- **Molecular Weight:** 269.77
- **Water Solubility:** 242 mg/L @ 25 C [1]
- **Solubility in Other Solvents:** s. in most organic solvents [1]
- **Melting Point:** 40 C [1]
- **Vapor Pressure:** 2.9 mPa @ 25 C [1]
- **Partition Coefficient:** 2.8998 [58]
- **Adsorption Coefficient:** 170 [11]

Exposure Guidelines:

- **ADI:** 0.0025 mg/kg/day [12]
- **MCL:** 0.002 mg/L [65]
- **RfD:** 0.01 mg/kg day [13]
- **PEL:** Not Available

- **HA**: Not Available
- **TLV**: Not Available

Basic Manufacturer:

Monsanto Company
800 N. Lindbergh Blvd.
St. Louis, MO 63167

- **Phone:** 314-694-6640
- **Emergency:** 314-694-4000

References:

References for the information in this PIP can be found in Reference List Number 10

DISCLAIMER: The information in this profile does not in any way replace or supersede the information on the pesticide product labeling or other regulatory requirements. Please refer to the pesticide product labeling.

EXTOXNET

Extension Toxicology Network

Pesticide Information Profiles

A Pesticide Information Project of Cooperative Extension Offices of Cornell University, Oregon State University, the University of Idaho, and the University of California at Davis and the Institute for Environmental Toxicology, Michigan State University. Major support and funding was provided by the USDA/Extension Service/National Agricultural Pesticide Impact Assessment Program.

EXTOXNET primary files maintained and archived at Oregon State University

Revised June 1996

Atrazine

Trade and Other Names: Trade names include Aatrex, Aktikon, Alazine, Atred, Atranex, Atrataf, Atratol, Azinotox, Crisazina, Farmco Atrazine, G-30027, Gesaprim, Giffex 4L, Malermais, Primatol, Simazat, and Zeapos.

Regulatory Status: Atrazine has been classified as a Restricted Use Pesticide (RUP) due to its potential for groundwater contamination [2]. RUPs may be purchased and used only by certified applicators. Atrazine is toxicity class III - slightly toxic. In November, 1994, the EPA initiated a Special Review which could result in use restrictions or cancellation of atrazine if health data warrant such action. Products containing atrazine must the Signal Word CAUTION.

Chemical Class: triazine

Introduction: Atrazine is a selective triazine herbicide used to control broadleaf and grassy weeds in corn, sorghum, sugarcane, pineapple, christmas trees, and other crops, and in conifer reforestation plantings. It is also used as a nonselective herbicide on non-cropped industrial lands and on fallow lands. Over 64 million acres of cropland were treated with atrazine in the U.S. in 1990. It is available as dry flowable, flowable liquid, liquid, water dispersible granular, and wettable powder formulations.

Formulation: It is available as dry flowable, flowable liquid, liquid, water dispersible granular, and wettable powder formulations.

Toxicological Effects:

- **Acute toxicity:** Atrazine is slightly to moderately toxic to humans and other animals. It can be absorbed orally, dermally, and by inhalation. Symptoms of poisoning include abdominal pain, diarrhea and vomiting, eye irritation, irritation of mucous membranes, and skin reactions [3]. At very high doses, rats show excitation followed by depression, slowed breathing, incoordination, muscle spasms, and hypothermia [3]. After consuming a large oral dose, rats exhibit muscular weakness, hypoactivity, breathing difficulty, prostration, convulsions, and death [16]. Atrazine is a mild skin irritant. Rashes associated with exposure have been reported. The oral LD50 for atrazine is 3090 mg/kg in rats, 1750 mg/kg in mice, 750 mg/kg in rabbits, and 1000 mg/kg in hamsters. The dermal LD50 in rabbits is 7500 mg/kg and greater than 3000 mg/kg in rats [15,16]. The 1-hour inhalation LC50 is greater than 0.7 mg/L in rats. The 4-hour inhalation LC50 is 5.2 mg/L in rats [3,6].
- **Chronic toxicity:** Some 40% of rats receiving oral doses of 20 mg/kg/day for 6 months died with signs of respiratory distress and paralysis of the limbs. Structural and chemical changes in the brain, heart, liver, lungs, kidney, ovaries, and endocrine organs were observed [3,16]. Rats fed 5 or 25 mg/kg/day of atrazine for 6 months exhibited growth retardation. In a 2-year study with dogs, 7.5 mg/kg/day caused decreased food intake and increased heart and liver weights. At 75 mg/kg/day, there were decreases in food intake and body weight gain, increased adrenal weight, lowered blood cell counts, and occasional tremors or stiffness in the rear limbs [3].
- **Reproductive effects:** Dietary doses of atrazine given to rats on days 3, 6 and 9 of gestation up to about 50 mg/kg/day caused

no adverse reproductive effects [3].

- **Teratogenic effects:** Atrazine does not appear to be teratogenic. In mice, atrazine did not cause abnormalities in fetuses whose dams were given doses of 46.4 mg/kg/day during days 6 through 14 of gestation [3].
- **Mutagenic effects:** The weight of evidence from more than 50 studies indicates that atrazine is not mutagenic [3].
- **Carcinogenic effects:** Atrazine did not cause tumors when mice were given oral doses of 21.5 mg/kg/day from age 1 to 4 weeks, followed by dietary doses of 82 mg/kg for an additional 17 months. However, mammary tumors were observed in rats after lifetime administration of high doses of atrazine [3]. Thus, available data regarding atrazine's carcinogenic potential are inconclusive.
- **Organ toxicity:** Lethal doses of atrazine in test animals have caused congestion and/or hemorrhaging to the lungs, kidneys, liver, spleen, brain, and heart [3]. Long-term consumption of high levels of atrazine has caused tremors, changes in organ weights, and damage to the liver and heart [3].

- **Fate in humans and animals:** Atrazine is readily absorbed through the gastrointestinal tract. When a single dose of 0.53 mg atrazine was administered to rats by gavage, 20% of the dose was excreted in the feces within 72 hours. The other 80% was absorbed across the lining of the gastrointestinal tract into the bloodstream. After 72 hours, 65% was eliminated in the urine and 15% was retained in body tissues, mainly in the liver, kidneys, and lungs [3].

Ecological Effects:

- **Effects on birds:** Atrazine is practically nontoxic to birds. The LD50 is greater than 2000 mg/kg in mallard ducks. At dietary doses of 5000 ppm, no effect was observed in bobwhite quail and ring-necked pheasants [15,16].
- **Effects on aquatic organisms:** Atrazine is slightly toxic to fish and other aquatic life. Atrazine has a low level of bioaccumulation in fish. In whitefish, atrazine accumulates in the brain, gall bladder, liver, and gut [16].
- **Effects on other organisms:** Atrazine is not toxic to bees [16].

Environmental Fate:

- **Breakdown in soil and groundwater:** Atrazine is highly persistent in soil. Chemical hydrolysis, followed by degradation by soil microorganisms, accounts for most of the breakdown of atrazine. Hydrolysis is rapid in acidic or basic environments, but is slower at neutral pHs. Addition of organic material increases the rate of hydrolysis. Atrazine can persist for longer than 1 year under dry or cold conditions [21]. Atrazine is moderately to highly mobile in soils with low clay or organic matter content. Because it does not adsorb strongly to soil particles and has a lengthy half-life (60 to >100 days), it has a high potential for groundwater contamination despite its moderate solubility in water [20]. Atrazine is the second most common pesticide found in private wells and in community wells [16]. Trace amounts have been found in drinking water samples and in groundwater samples in a number of states [23,21]. A 5-year survey of drinking water wells detected atrazine in an estimated 1.7% of community water systems and 0.7% of rural domestic wells nationwide. Levels detected in rural domestic wells sometimes exceeded the MCL [23]. The recently completed National Survey of Pesticides in Drinking Water found atrazine in nearly 1% of all of the wells tested [23].
- **Breakdown in water:** Atrazine is moderately soluble in water. Chemical hydrolysis, followed by biodegradation, may be the most important route of disappearance from aquatic environments. Hydrolysis is rapid under acidic or basic conditions, but is slower at neutral pHs. Atrazine is not expected to strongly adsorb to sediments. Bioconcentration and volatilization of atrazine are not environmentally important [21]. Atrazine has been detected in each of 146 water samples collected at 8 locations from the Mississippi, Ohio and Missouri Rivers and their tributaries. For several weeks, 27% of these samples contained atrazine concentrations above the EPA's maximum contaminant level (MCL) [24].
- **Breakdown in vegetation:** Atrazine is absorbed by plants mainly through the roots, but also through the foliage. Once absorbed, it is translocated upward and accumulates in the growing tips and the new leaves of the plant. In susceptible plant species, atrazine inhibits photosynthesis. In tolerant plants, it is metabolized [6]. Most crops can be planted 1 year after application of atrazine. Atrazine increases the uptake of arsenic by treated plants [16].

Physical Properties:

- **Appearance:** Atrazine is a white, crystalline solid [6].
- **Chemical Name:** 2-chloro-4-ethylamine-6-isopropylamino-S-triazine [6]
- **CAS Number:** 1912-24-9
- **Molecular Weight:** 215.69
- **Water Solubility:** 28 mg/L @ 20 C [6]
- **Solubility in Other Solvents:** chloroform v.s.; diethyl ether v.s.; dimethyl sulfoxide v.s. [6]
- **Melting Point:** 176 C [6]

- **Vapor Pressure:** 0.04 mPa @ 20 C [6]
- **Partition Coefficient:** 2.3404 [6]
- **Adsorption Coefficient:** 100 [20]

Exposure Guidelines:

- **ADI:** Not Available
- **MCL:** 0.003 mg/L [25]
- **RfD:** 0.035 mg/kg/day [26]
- **PEL:** Not Available
- **HA:** Not Available
- **TLV:** 5 mg/m3 (8-hour) [16]

Basic Manufacturer:

Ciba-Geigy Corp.
P.O. Box 18300
Greensboro, NC 27419-8300

- **Phone:** 800-334-9481
- **Emergency:** 800-888-8372

References:

References for the information in this PIP can be found in Reference List Number 8

DISCLAIMER: The information in this profile does not in any way replace or supersede the information on the pesticide product labeling or other regulatory requirements. Please refer to the pesticide product labeling.

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Extension Toxicology Network

Pesticide Information Profiles

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EXTOXNET primary files maintained and archived at Oregon State University

Revised June 1996

Diquat dibromide

Trade and Other Names: Trade names include Aquacide, Aquakill, Dextrone, Diquat, Reglone, Reglox, Reward, Tag, Torpedo, Vegetrole, and Weedtrine-D.

Regulatory Status: Diquat dibromide is a moderately toxic compound in EPA toxicity class II [1,2]. It is a General Use Pesticide (GUP). Labels for products containing diquat dibromide must bear the Signal Word WARNING.

Chemical Class: dessicant

Introduction: Diquat dibromide is a nonselective, quick-acting herbicide and plant growth regulator, causing injury only to the parts of the plant to which it is applied. Diquat dibromide is referred to as a desiccant because it causes a leaf or an entire plant to dry out quickly. It is used to desiccate potato vines and seed crops, to control flowering of sugarcane, and for industrial and aquatic weed control. It is not residual; that is, it does not leave any trace of herbicide on or in plants, soil, or water.

Formulation: Not Available

Toxicological Effects:

- **Acute toxicity:** Diquat dibromide is moderately toxic via ingestion, with reported oral LD50 values of 120 mg/kg in rats, 233 mg/kg in mice, 188 mg/kg in rabbits, and 187 mg/kg in guinea pigs and dogs [1,87]. Cows appear to be particularly sensitive to this herbicide, with an oral LD50 of 30 to 56 mg/kg [17]. The acute dermal LD50 for diquat dibromide is approximately 400 to 500 mg/kg in rabbits, indicating moderate toxicity by this route as well [58,87]. A single dose of diquat dibromide was not irritating to the skin of rabbits, but repeated dermal dosing did cause mild redness, thickening, and scabbing [58]. Moderate to severe eye membrane irritation occurred when diquat dibromide was administered to rabbits [88]. Ingestion of sufficient doses may cause severe irritation of the mouth, throat, esophagus, and stomach, followed by nausea, vomiting, diarrhea, severe dehydration, and alterations in body fluid balances, gastrointestinal discomfort, chest pain, diarrhea, kidney failure, and toxic liver damage [87]. Skin absorption of high doses may cause symptoms similar to those that occur following ingestion [89]. Very large doses of the herbicide can result in convulsions and tremors [88]. Test animals (rats, mice, guinea pigs, rabbits, dogs, cows, and hens) given lethal doses of diquat dibromide showed a delayed pattern of illness, with onset approximately 24 hours following dosing, subsequent lethargy, pupil dilation, respiratory distress, weight loss, weakness and finally death over the course of 2 to 14 days after dosing [58,87,89]. There have been reports of workers who have had softening and color changes in one or more fingernails after contact with concentrated diquat dibromide solutions [87]. In some instances, the nail was shed, and did not grow in again [87]. Several cases of severe eye injury in humans have occurred after accidental splashings [87]. In each case, initial irritation was mild, but after several days, serious burns and sometimes scarring of the cornea developed. Direct or excessive inhalation of diquat dibromide spray mist or dust may result in oral or nasal irritation,

nosebleeds, headache, sore throat, coughing, and symptoms similar to those from ingestion of diquat [87].

- **Chronic toxicity:** Chronic effects of diquat dibromide are similar to those of paraquat [87]. Cataracts, a clouding of the eyes which interferes with light entering the eye, occurred in rats and dogs given 2.5 mg/kg/day and 5 mg/kg/day of diquat dibromide, respectively [87]. Cataracts increased in proportion to the dose given in test animals (cats and dogs) [17,88]. Chronic exposure is necessary to produce these effects [87]. Other effects on the eye (hemorrhage, retinal detachment) may occur at higher dosages [87]. Rats fed dietary doses of 2.5 mg/kg/day over 2 years did not exhibit signs of toxicity other than reduced food intake and decreased growth [17]. In another study using rats, oral doses of 4 mg/kg/day over 2 years produced no behavioral or other changes in general condition [87]. At this dose level no evidence of change in the kidneys, liver, or mycocardium (heart muscle) were seen. This dosage (but not 2 mg/kg/day) caused changes in lung tissues [87]. Repeated or prolonged dermal contact may cause inflammation of the skin, and, at high doses, systemic effects in other parts of the body. These may include damage to the the kidneys [58]. Chronic exposure may damage skin, which may increase the permability of the skin to foreign compounds [88].
- **Reproductive effects:** Diquat dibromide generally did not reduce fertility when tested in experimental animals [89]. Rats receiving 1.25 mg/kg/day decreased their food intake and showed slowed growth, but had unchanged reproduction [89]. Fertility was reduced in male mice given diquat dibromide during different stages of sperm formation [87]. Neither fertility nor reproduction was affected in a three-generation study in rats given dietary doses of 12.5 or 25 mg/kg/day of diquat dibromide, although some growth retardation was seen at the 25 mg/kg/day dose [87]. Based on this evidence it is unlikely that diquat dibromide will cause reproductive effects in humans under normal circumstances.
- **Teratogenic effects:** Offspring of pregnant rats given a fatal injected dose of 14 mg/kg of diquat dibromide showed evidence of skeletal defects of the collar bone, as well as little or no ear bone formation upon examination [58,87]. No deformities were found in the unborn offspring of pregnant rats that were injected intraperitoneally with 0.5 mg/kg/day of diquat daily during organogenesis, the stage of fetal development in which organs are formed [26]. Growth retardation was seen in test animals given extremely high doses of diquat. While no actual teratogenesis occurred in rats given single abdominal injections during days 7 to 14 of pregnancy, many rats did not have normal weight gain and bone formation in the unborn was decreased [23]. It is unlikely that diquat dibromide will cause teratogenic effects in humans under normal circumstances.
- **Mutagenic effects:** There is no evidence that diquat dibromide causes permanent changes in genetic material [87]. For example, no mutagenic effects were seen in mice given oral doses of 10 mg/kg/day for 5 days [23].
- **Carcinogenic effects:** An 80-week feeding study showed that dietary doses of 15 mg/kg/day of diquat did not cause tumors in rats [90]. Likewise, dietary levels of 36 mg/kg/day for 2 years did not induce tumors in rats [87]. Based on the evidence, it appears that diquat dibromide is not carcinogenic.
- **Organ toxicity:** In animals, diquat dibromide may affect the gastrointestinal tract, eyes, kidneys or liver, and the lungs.

- **Fate in humans and animals:** Absorption of diquat dibromide from the gut into the bloodstream is low [87]. Oral doses are mainly metabolized within the intestines, with metabolites being excreted in the feces [87,30]. Rat studies showed only a small percentage of the applied oral dose (6%) was absorbed into the bloodstream and then excreted in the urine [87]. Dermal, inhalation, or intravenous exposure results in little processing and rapid elimination in the urine [87]. Following subcutaneous injection in rats, excretion of about 90% of the dose occurred in the urine on the first day and almost all of the remainder on the next day [87]. Complete elimination of the herbicide was seen in urine and feces of rats within 4 days of administration of single oral doses of 5 to 10 mg/kg of diquat dibromide [87].

Ecological Effects:

- **Effects on birds:** Diquat dibromide ranges from slightly to moderately toxic to birds [91]. The reported acute oral LD50 in young male mallards is 564 mg/kg [8]. The oral LD50 for diquat dibromide is 200 to 400 mg/kg in hens [8]. The 5-day dietary LC50 is about 1300 ppm in Japanese quail [36].
- **Effects on aquatic organisms:** Diquat dibromide is moderately to practically nontoxic to fish and aquatic invertebrates. The 8-hour LC50 for diquat dibromide is 12.3 mg/L in rainbow trout and 28.5 mg/L in Chinook salmon [28]. The 96-hour LC50 is 16 mg/L in northern pike, 20.4 mg/L in fingerling trout, 245 mg/L in bluegill, 60 mg/L in yellow perch, and 170 mg/L in black bullhead [37,92]. Research indicates that yellow perch suffer significant respiratory stress when herbicide concentrations in the water are similar to those normally present during aquatic vegetation control programs [93]. There is little or no bioconcentration of diquat dibromide in fish [8].
- **Effects on other organisms:** Diquat dibromide is not toxic to honey bees [1]. Since diquat dibromide is a nonselective herbicide, it may present a danger to non-target plant species [91]. Cows are particularly sensitive to the toxic effects of this material [17].

Environmental Fate:

- **Breakdown in soil and groundwater:** Diquat dibromide is highly persistent, with reported field half-lives of greater than 1000 days [11]. It is very well sorbed by soil organic matter and clay [11]. Although it is water soluble [11], its capacity for

strong adsorption to soil particles suggest that it will not easily leach through the soil, be taken up by plants or soil microbes, or broken down by sunlight (photochemical degradation). Field and laboratory tests show that diquat usually remains in the top inch of soil for long periods of time after it is applied [94].

- **Breakdown in water:** Studies on the erosion of diquat-treated soils near bodies of water indicate that diquat dibromide stays bound to soil particles, remaining biologically inactive in surface waters, such as lakes, rivers, and ponds [95]. When diquat dibromide is applied to open water, it disappears rapidly because it binds to suspended particles in the water [95]. Diquat dibromide's half-life is less than 48 hours in the water column, and may be on the order of 160 days in sediments due to its low bioavailability [94,95]. Microbial degradation and sunlight play roles in the breakdown of the compound [95]. At 22 days after a weed infested artificial lake was treated, only 1% of the applied diquat dibromide remained in the water and 19% was adsorbed to sediments [9].
- **Breakdown in vegetation:** Diquat dibromide is rapidly absorbed into the leaves of plants, but usually kills the plant tissues necessary for translocation too quickly to allow movement to other parts of the plant. The herbicide interferes with cell respiration, the process by which plants produce energy. Diquat dibromide is broken down on the plant surface by photochemical degradation [58]. It is rapidly absorbed by aquatic weeds from the surrounding water and concentrated in the plant tissue [8]. Thus, even low concentrations of the herbicide can control aquatic weeds [8].

Physical Properties:

- **Appearance:** Technical diquat dibromide, which is greater than 95% pure, forms white to yellow crystals [1].
- **Chemical Name:** 1,1'-ethylene-2,2'-bipyridyldiylium dibromide salt [1]
- **CAS Number:** 85-00-7
- **Molecular Weight:** 344.06
- **Water Solubility:** 700,000 mg/L @ 20 C; v.s. [1]
- **Solubility in Other Solvents:** i.s. in nonpolar solvents such as chloroform, diethyl ether, and petroleum ether [1]; s.s in alcohol and hydroxylic solvents [1]
- **Melting Point:** Decomposes above 300 C [1]
- **Vapor Pressure:** Negligible @ 20 C [1]
- **Partition Coefficient:** -4.6021 [1]
- **Adsorption Coefficient:** 1,000,000 (estimated) [11]

Exposure Guidelines:

- **ADI:** 0.002 mg/kg/day [12]
- **MCL:** 0.02 mg/L [65]
- **RfD:** 0.0022 mg/kg/day [13]
- **PEL:** Not Available
- **HA:** Not Available
- **TLV:** 0.1 mg/m3 (8-hour) (respirable fraction) [17]

Basic Manufacturer:

Zeneca Ag Products
1800 Concord Pike
Wilmington, DE 19897

- **Phone:** 800-759-4500
- **Emergency:** 800-759-2500

References:

References for the information in this PIP can be found in Reference List Number 10

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MCPA

Trade and Other Names: Trade or other names for MCPA or products containing it include: Agritox, Agroxone, Agrozone, Agsco MXL, Banlene, Blesal MC, Bordermaster, Cambilene, Cheyenne, Chimac Oxy, Chiptox, Class MCPA, Cornox Plus, Dakota, Ded-Weed, Empal, Envoy, Gordon's Amine, Kilsem, Legumex, Malerbane, Mayclene, MCP, Mephanac, Midox, Phenoxylene, Rhomene, Rhonox, Sanaphen-M, Shamrox, Selectyl, Tiller, U 46 M-Fluid, Vacate, Weed-Rhap, and Zhelan.

Regulatory Status: MCPA is a slightly toxic compound in EPA toxicity class III, and is a General Use Pesticide (GUP). Labels for products containing MCPA must carry the Signal Word DANGER due to its potential to cause severe eye irritation.

Chemical Class: phenoxy compound

Introduction: MCPA is a systemic postemergence phenoxy herbicide used to control annual and perennial weeds (including thistle and dock) in cereals, flax, rice, vines, peas, potatoes, grasslands, forestry applications, and on rights-of-way. This herbicide is very compatible with many other compounds and may be used in formulation with many other products, including bentazone, bromoxynil, 2,4-D, dicamba, fenoxaprop, MCPB, mecoprop, thifensulfuron, and tribenuron.

NOTE: As with some of the other phenoxy herbicides, MCPA is an acid, but is often formulated as a salt (e.g. dimethylamine salt) or an ester (e.g. isooctyl ester). Unless otherwise indicated, this document will refer to the acid form.

Formulation: This herbicide is very compatible with many other compounds, and may be used in formulation with many other products, including bentazone, bromoxynil, 2,4-D, dicamba, fenoxaprop, MCPB, mecoprop, thifensulfuron, and tribenuron.

Toxicological Effects:

- **Acute toxicity:** MCPA acid is slightly toxic via ingestion, with reported oral LD50 values for the technical product in rats ranging from 700 mg/kg to 1160 mg/kg [5,6] and ranging in mice from 550 to 800 mg/kg [5,6]. It is slightly toxic via the dermal route as well, with reported dermal LD50 values ranging from greater than 1000 mg/kg in rats to greater than 4000 mg/kg in rabbits [5,6]. Symptoms in humans from very high acute exposure could include slurred speech, twitching, jerking and spasms, drooling, low blood pressure, and unconsciousness [1].
- **Chronic toxicity:** Dietary levels of approximately 50 mg/kg/day and 125 mg/kg/day over 7 months caused reduced feeding rates and retarded growth rates in rats [1]. White blood cell counts and ratios were not affected, but some reductions in red blood cell counts and hemoglobin did appear to be associated with exposure to MCPA at oral dose levels of approximately 20 mg/kg/day. In the same study, oral doses of approximately 5 mg/kg/day caused increased relative kidney weights, and oral doses of approximately 20 mg/kg/day caused increased relative liver weights [1]. Another study in rats showed no effects on kidney or liver weights over an unspecified period at oral doses of 60 mg/kg/day, but oral doses of 150 mg/kg/day did cause

reversible increases in these weights over a course of 3 months [1]. Very high dermal doses of 500 mg/kg/day caused reduced body weight, and even higher dermal doses of 1000 and 2000 mg/kg/day resulted in increased mortality and observable changes in liver, kidney, spleen, and thymus tissue [1].

- **Reproductive effects:** A two-generation rat study at doses of up to 15 mg/kg/day affected reproductive function. Even smaller amounts of the compound were toxic to the fetuses. Dogs receiving relatively small amounts of MCPA (8 and 16 mg/kg) for 13 weeks showed adverse sperm and testes changes [8]. It is unlikely that humans will experience these effects under normal exposure conditions.
- **Teratogenic effects:** Offspring of pregnant rats fed low to moderate doses of MCPA (20 to 125 mg/kg) on days 6 to 15 of gestation, had no birth defects. However, when the ethyl ester form of MCPA was fed to pregnant rats (2 to 100 mg/kg/day on days 8 to 15 of gestation), cleft palate, heart defect, and kidney anomalies were observed in the offspring [7]. Mice fed 5 to 100 mg/kg/day of MCPA on days 6 to 15 showed significantly reduced fetal weight and delayed bone development at the highest dose [24]. Teratogenic effects in humans are unlikely at expected exposure levels.
- **Mutagenic effects:** MCPA is reportedly weakly mutagenic to bone marrow and ovarian cells of hamsters, but negative results were reported for other mutagenic tests [38]. It was negative in a bacterial test system (both with and without metabolic activation), negative in spot tests, and negative in host-mediated tests [1]. It produced no detectable increase in chromosomal aberrations in house flies [4]. Some irregularities occurred in gene transfer during cell division in brewers yeast, although at levels which caused massive cell death [1]. It appears that the compound poses little or no mutagenic risk.
- **Carcinogenic effects:** All of the available evidence on MCPA indicates that the compound does not cause cancer [1]. Forestry and agricultural workers occupationally exposed to MCPA in Sweden did not show increased cancer incidence [39].
- **Organ toxicity:** Target organs identified in animal studies include the liver, kidneys, spleen. and thymus. Farm worker exposure has resulted in reversible anemia, muscular weakness, digestive problems, and slight liver damage [1].

- **Fate in humans and animals:** MCPA is rapidly absorbed and eliminated from mammalian systems [1]. Rats eliminated nearly all of a single oral dose within 24 hours, mostly though urine with little or no metabolism [1,6]. In another rat study, three quarters of the dose was eliminated within 2 days. All was gone by the 8 days [1]. Humans excreted about half of a 5 mg dose in the urine within a few days. No residues were found after day 5 [1]. Cattle and sheep fed low to moderate doses of MCPA in the diet for 2 weeks showed no residues from levels less than about 18 mg/kg [1]. The major metabolite of MCPA is 2-methyl-4-chlorophenol in the free and conjugated form, which is formed in the liver [38].

Ecological Effects:

- **Effects on birds:** MCPA is moderately toxic to wildfowl; the LD50 of MCPA in bobwhite quail is 377 mg/kg [5,6].
- **Effects on aquatic organisms:** MCPA is only slightly toxic to freshwater fish, with reported LC50 values ranging from 117 [5] to 232 mg/L in rainbow trout [6]. MCPA is practically nontoxic to freshwater invertebrates, and estuarine and marine organisms.
- **Effects on other organisms:** It is nontoxic to bees, with a reported oral LD50 of 104 ug/bee [5,6].

Environmental Fate:

- **Breakdown in soil and groundwater:** MCPA and its formulations are rapidly degraded by soil microorganisms and it has low persistence, with a reported field half-life of 14 days to 1 month, depending on soil moisture and soil organic matter [21]. Decreased soil moisture and microbial activity, as well as increased soil organic matter, will prolong the field half-life for MCPA [12]. With less than 10% organic matter in soil, the compound is degraded in 1 day and, with greater than 10% levels in soil, it takes 3 to 9 days to degrade. The half-life is 5 to 6 days in slightly acidic to slightly alkaline soils [12]. MCPA readily leaches in most soils, but its mobility decreases with increasing organic matter [12]. MCPA and its formulations show little affinity for soil.
- **Breakdown in water:** It is relatively stable to light breakdown [5], but can be rapidly browken down by microorganisms. In sterilized water, it takes about 5 weeks for half of the compound to degrade due to the action of sunlight. In rice paddy water, however, MCPA is almost totally degraded by aquatic microorganisms in under 2 weeks [12].
- **Breakdown in vegetation:** MCPA is readily absorbed and translocated in most plants [5]. It works by concentrating in the actively growing regions of a plant (meristematic tissue), where it interferes with protein synthesis, cell division, and ultimately the growth of non-resistant plants [7]. It is actively broken down in plants, the major metabolite being 2-methyl-4-chlorophenol [5].

Physical Properties:

- **Appearance:** Pure MCPA occurs as colorless crystals [6].
- **Chemical Name:** (4-chloro-2-methylphenoxy)acetic acid [6]

- **CAS Number:** 94-74-6
- **Molecular Weight:** 200.62
- **Water Solubility:** 825 mg/L @ 25 C (acid) [5]
- **Solubility in Other Solvents:** v.s. in ether, ethanol, toluene, xylene; s. in methanol [6]
- **Melting Point:** 118-119 C [6]
- **Vapor Pressure:** 0.2 mPa @ 20 C [6]
- **Partition Coefficient:** Not Available
- **Adsorption Coefficient:** MPCA acid, 100; MCPA salts, 20 (estimated); MCPA ester, 1000 (estimated) [21]

Exposure Guidelines:

- **ADI:** Not Available
- **MCL:** Not Available
- **RfD:** 0.0005 mg/kg/day [31]
- **PEL:** Not Available
- **HA:** 0.01 mg/L (lifetime) [38]
- **TLV:** Not Available

Basic Manufacturer:

Gilmore, Inc.
5501 Murray Road
Memphis, TN 38119-3703

- **Phone:** 901-761-5870
- **Emergency:** Not Available

References:

References for the information in this PIP can be found in Reference List Number 7

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2,4-D

Trade and Other Names: 2,4-D is used in many commercial products. Commercial names for products containing 2,4-D include Aqua-Kleen, Barrage, Lawn-Keep, Malerbane, Planotox, Plantgard, Savage, Salvo, Weedone, and Weedtrine-II.

Regulatory Status: 2,4-D is a General Use Pesticide (GUP) in the U.S. The diethylamine salt is toxicity class III- slightly toxic orally, but toxicity class I- highly toxic by eye exposure. It bears the Signal Word DANGER - POISON because 2,4-D has produced serious eye and skin irritation among agricultural workers.

Chemical Class: phenoxy compound

Introduction: There are many forms or derivatives of 2,4-D including esters, amines, and salts. Unless otherwise specified, this document will refer to the acid form of 2,4-D. 2,4-D, a chlorinated phenoxy compound, functions as a systemic herbicide and is used to control many types of broadleaf weeds. It is used in cultivated agriculture, in pasture and rangeland applications, forest management, home, garden, and to control aquatic vegetation. It may be found in emulsion form, in aqueous solutions (salts), and as a dry compound.

The product Agent Orange, used extensively throughout Vietnam, was about 50% 2,4-D. However, the controversies associated with the use of Agent Orange were associated with a contaminant (dioxin) in the 2,4,5-T component of the defoliant.

Formulation: It may be found in emulsion form, in aqueous solutions (salts), and as a dry compound.

Toxicological Effects:

- **Acute toxicity:** The acid form is of slight to moderate toxicity. The oral LD50 of 2,4-D ranges from 375 to 666 mg/kg in the rat, 370 mg/kg in mice, and from less than 320 to 1000 mg/kg in guinea pigs. The dermal LD50 values are 1500 mg/kg in rats and 1400 mg/kg in rabbits, respectively [1,5,7]. In humans, prolonged breathing of 2,4-D causes coughing, burning, dizziness, and temporary loss of muscle coordination [1]. Other symptoms of poisoning can be fatigue and weakness with possible nausea. On rare occasions following high levels of exposure, there can be inflammation of the nerve endings with muscular effects [25].
- **Chronic toxicity:** Rats given high amounts, 50 mg/kg/day, of 2,4-D in the diet for 2 years showed no adverse effects. Dogs fed lower amounts in their food for 2 years died, probably because dogs do not excrete organic acids efficiently. A human given a total of 16.3 g in 32 days therapeutically, lapsed into a stupor and showed signs of incoordination, weak reflexes, and loss of bladder control [1,5,7].
- **Reproductive effects:** High levels of 2,4-D (about 50 mg/kg/day) administered orally to pregnant rats did not cause any adverse effects on birth weights or litter size. Higher doses (188 mg/kg/day) resulted in fetuses with abdominal cavity bleeding

and increased mortality [1,5,7]. DNA synthesis in the testes was significantly inhibited when mice were fed large amounts (200 mg/kg/day) of 2,4-D [7]. The evidence suggests that if 2,4-D causes reproductive effects in animals, this only occurs at very high doses. Thus reproductive problems associated with 2,4-D are unlikely in humans under normal circumstances.

- **Teratogenic effects:** 2,4-D may cause birth defects at high doses. Rats fed 150 mg/kg/day on days 6 to 15 of pregnancy had offspring with increased skeletal abnormalities, such as delayed bone development and wavy ribs [7]. This suggests that 2,4-D exposure is unlikely to be teratogenic in humans at expected exposure levels.
- **Mutagenic effects:** 2,4-D has been very extensively tested and was found to be nonmutagenic in most systems. 2,4-D did not damage DNA in human lung cells. However, in one study, significant effects occurred in chromosomes in cultured human cells at low exposure levels [26]. The data suggest that 2,4-D is not mutagenic or has low mutagenic potential.
- **Carcinogenic effects:** 2,4-D fed to rats for 2 years caused an increase in malignant tumors [7]. Female mice given a single injection of 2,4-D developed cancer (reticulum-cell sarcomas) [7]. Another study in rodents shows a low incidence of brain tumors at moderate exposure levels (45 mg/kg/day) over a lifetime [1,7]. However, a number of questions have been raised about the validity of this evidence and thus about the carcinogenic potential of 2,4-D. In humans, a variety of studies give conflicting results. Several studies suggest an association of 2,4-D exposure with cancer. An increased occurrence of non-Hodgkin's lymphoma was found among a Kansas and Nebraska farm population associated with the spraying of 2,4-D [25,27]. Other studies done in New Zealand, Washington, New York, Australia, and on Vietnam veterans from the U.S. were all negative. There remains considerable controversy about the methods used in the various studies and their results [28]. Thus, the carcinogenic status of 2,4-D is not clear.
- **Organ toxicity:** Most symptoms of 2,4-D exposure disappear within a few days, but there is a report of liver dysfunction from long-term exposure [1,25].
- **Fate in humans and animals:** The absorption of 2,4-D is almost complete in mammals after ingestion and nearly all of the dose is excreted in the urine. The compound is readily absorbed through the skin and lungs. Men given 5 mg/kg excreted about 82% of the dose as unchanged 2,4-D. The half-life is between 10 and 20 hours in living organisms. There is no evidence that 2,4-D accumulates to significant level in mammals or in other organisms [20]. Between 6 and 8 hours after doses of 1 mg/kg, peak concentrations of 2,4-D were found in the blood, liver, kidney, lungs, and spleen of rats. There were lower levels in muscle and brain. After 24 hours, there were no detectable tissue residues. Only traces of the compound have been found in the milk of lactating animals for 6 days following exposure. 2,4-D passes through the placenta in pigs and rats. In rats, about 20% was detected in the uterus, placenta, fetus, and amniotic fluid [27]. Chickens given moderate amounts of 2,4-D in drinking water from birth to maturity had very low levels of the compound in eggs [7].

Ecological Effects:

- **Effects on birds:** 2,4-D is slightly toxic to wildfowl and slightly to moderately toxic to birds. The LD50 is 1000 mg/kg in mallards, 272 mg/kg in pheasants, and 668 mg/kg in quail and pigeons [5-7].
- **Effects on aquatic organisms:** Some formulations of 2,4-D are highly toxic to fish while others are less so. For example, the LC50 ranges between 1.0 and 100 mg/L in cutthroat trout, depending on the formulation used. Channel catfish had less than 10% mortality when exposed to 10 mg/L for 48 hours [1,9]. Green sunfish, when exposed to 110 mg/L for 41 hours, showed no effect on swimming response. Limited studies indicate a half-life of less than 2 days in fish and oysters [24]. Concentrations of 10 mg/L for 85 days did not adversely affect the survival of adult dungeness crabs. For immature crabs, the 96-hour LC50 is greater than 10 mg/L, indicating that 2,4-D is only slightly toxic. Brown shrimp showed a small increase in mortality at exposures of 2 mg/L for 48 hours [7,20].
- **Effects on other organisms:** Moderate doses of 2,4-D severely impaired honeybees brood production. At lower levels of exposure, exposed bees lived significantly longer than the controls. The honeybee LD50 is 0.0115 mg/bee [6,7].

Environmental Fate:

- **Breakdown in soil and groundwater:** 2,4-D has low soil persistence. The half-life in soil is less than 7 days [21]. Soil microbes are primarily responsible for its disappearance [20]. Despite its short half-life in soil and in aquatic environments, the compound has been detected in groundwater supplies in at least five States and in Canada [20]. Very low concentrations have also been detected in surface waters throughout the U.S. [23].
- **Breakdown in water:** In aquatic environments, microorganisms readily degrade 2,4-D. Rates of breakdown increase with increased nutrients, sediment load, and dissolved organic carbon. Under oxygenated conditions the half-life is 1 week to several weeks [20].
- **Breakdown in vegetation:** 2,4-D interferes with normal plant growth processes. Uptake of the compound is through leaves, stems, and roots. Breakdown in plants is by a variety of biological and chemical pathways [10]. 2,4-D is toxic to most broad leaf crops, especially cotton, tomatoes, beets, and fruit trees [7].

Physical Properties:

- **Appearance:** 2,4-D is a white powder [6].
- **Chemical Name:** (2,4-dichlorophenoxy)acetic acid [6]
- **CAS Number:** 94-75-7
- **Molecular Weight:** 221.04
- **Water Solubility:** 900 mg/L @ 25 C (acid) [5]
- **Solubility in Other Solvents:** ethanol v.s.; diethyl ether v.s.; toluene s.; xylene s. [6]
- **Melting Point:** 140.5 C [6]
- **Vapor Pressure:** 0.02 mPa @ 25 C (acid) [5]
- **Partition Coefficient:** 2.81 [20]
- **Adsorption Coefficient:** 20 (acid) [21]

Exposure Guidelines:

- **ADI:** 0.3 mg/kg/day [29]
- **MCL**: 0.07 mg/L [30]
- **RfD:** 0.01 mg/kg/day [31]
- **PEL:** 10 mg/m3 (8-hour) [32]
- **HA**: Not Available
- **TLV**: Not Available

Basic Manufacturer:

Rhone-Poulenc Ag. Co.
P.O. Box 12014
2 T.W. Alexander Dr.
Research Triangle Park, NC 27709

- **Phone:** 919-549-2000
- **Emergency:** 800-334-7577

References:

References for the information in this PIP can be found in Reference List Number 7

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Revised June 1996

Dalapon

Trade and Other Names: Trade names include Alatex, Basinex P, Dalacide, Dalapon-Na (Dalapon-Sodium), Devipon, Ded-Weed, Dowpon, DPA, Gramevin, Kenapon, Liropon, Radapon, Revenge, and Unipon. Dalapon is also called sodium dalapon or magnesium dalapon.

Regulatory Status: Dalapon is classified by the U.S. Environmental Protection Agency (EPA) as a General Use Pesticide (GUP). Dalapon is in toxicity class II - moderately toxic. Products containing the herbicide bear labels with the Signal Word WARNING.

Chemical Class: organochlorine

Introduction: Dalapon is an organochlorine herbicide and plant growth regulator used to control specific annual and perennial grasses, such as quackgrass, Bermuda grass, Johnson grass, as well as cattails and rushes. The major food crop use of dalapon is on sugarcane and sugar beets. It is also used on various fruits, potatoes, carrots, asparagus, alfalfa, and flax, as well as in forestry, home gardening, and in or near water to control reed and sedge growth. Dalapon is applied both before the target plant comes up and after the plant emerges. Commercial products consist of the sodium salt or mixed sodium and magnesium salts of dalapon.

Formulation: Commercial products consist of the sodium salt or mixed sodium and magnesium salts of dalapon.

Toxicological Effects:

- **Acute toxicity:** Dalapon is moderately toxic to humans. Skin and inhalation exposure could be of significance to dalapon production workers, pesticide applicators, and some agricultural workers [39]. Symptoms of high acute exposure include loss of appetite, slowed heartbeat, skin irritation, eye irritation such as conjunctivitis or corneal damage, gastrointestinal disturbances such as vomiting or diarrhea, tiredness, pain, and irritation of the respiratory tract [40]. Dalapon is an acid which may cause corrosive injury to body tissues [17]. Eye exposure to this material can cause permanent eye damage. Skin burns may occur from dermal exposure to dalapon, especially when skin is moist. Oral LD50 values range from 9330 mg/kg in male rats to 7570 mg/kg in female rats [4]. The oral LD50 is 3860 mg/kg in female rabbits and greater than 4600 mg/kg in female guinea pigs. Dalapon is moderately irritating to skin and eyes [41,17]. The application of the sodium salt of dalapon (in a dry powder formulation) to rabbit eyes produced pain and irritation, followed by severe conjunctivitis and corneal injury, which healed after several days [17].
- **Chronic toxicity:** Long-term dalapon feeding studies in dogs and rats did show increased kidney weights in animals fed very high daily doses [17,41]. Rats fed 50 mg/kg/day for 2 years showed a slight average increase in kidney weight. No adverse effects were seen in this study in rats fed 15 mg/kg/day. In a 1-year feeding study with dogs fed 100 mg/kg/day, there was a slight average increase in kidney weight. No adverse effects were seen at 50 mg/kg/day [17,41]. These mild effects on the

kidneys are consistent with data that show that ingested dalapon is rapidly excreted in the urine.

- **Reproductive effects:** Tests indicate that dalaopn does not produce adverse effects on fertility or reproduction, except at extremely high doses [41].
- **Teratogenic effects:** Sodium dalapon was not teratogenic in the rat at doses as high as 2000 mg/kg/day [42].
- **Mutagenic effects:** Dalapon was not mutagenic when tested in several organisms [42].
- **Carcinogenic effects:** No carcinogenic effects were seen in rats fed the sodium salt of dalapon at 5, 15, or 50 mg/kg/day for 2 years [41,42].
- **Organ toxicity:** Dalapon dust and vapor may be irritating to the respiratory tract [17]. Repeated or prolonged exposure to dalapon may cause irritation to the mucous membrane linings of the mouth, nose, throat, lungs, and to the eyes [17]. Chronic skin contact can lead to moderate irritation or even mild burns, although occasional contact is not likely to produce irritation. Dalapon is not absorbed through the skin in toxic amounts [41].

- **Fate in humans and animals:** The half-life of dalapon in human blood is 1.5-3 days [39]. Dalapon's half-life in the blood system of dogs is about 12 hours [39,41]. Dalapon and all of its known breakdown products dissolve easily in water. They are readily washed from cells and tissues. Because dalapon is insoluble in organic solvents and lipids, it does not build up in animal tissues. A nonmetabolized form of dalapon was excreted in the urine of animals fed the herbicide. Less than 1% of the ingested dose appeared as residues in the milk of dairy cows that were fed dalapon [17,39].

Ecological Effects:

- **Effects on birds:** Dalapon is practically nontoxic to birds. When dalapon was fed to 2-week old birds for 5 days, followed by untreated feed for 3 days, the LC50 of dalapon was more than 5000 ppm in mallards, ring-necked pheasants, and Japanese quail [17,43]. The acute oral LD50 of dalapon is 5660 mg/kg for chickens. While dalapon is practically nontoxic to birds, reproduction rates of birds are decreased at very high doses [17]. Reproduction was depressed in mallard ducks that were fed one fourth the dose of dalapon that caused death [43].
- **Effects on aquatic organisms:** Dalapon is practically nontoxic to fish [43]. While there were no deaths reported in goldfish after a 24-hour exposure to 100 mg/L of dalapon, all fish died after a similar exposure to 500 mg/L or above [17]. The 1-to-21-day LC50 values for dalapon in fish are all on the order of 100 mg/L for several species tested [9,17]. The LC50 for dalapon in bluegills is 105 mg/L [9,17]. Its toxicity to aquatic invertebrates varies, depending on the species. Values can be as low as the 48-hour effective concentration (EC) of 1 mg/L in brown shrimp, or as high as the 96-hour LC50 of 4800 mg/L in other crustaceans. Aquatic crustaceans and insects are the most dalapon-sensitive of the aquatic invertebrates. Dalapon is only slightly toxic to mollusks [9,17].
- **Effects on other organisms:** Dalapon is relatively nontoxic to honeybees and other insects and has low toxicity to soil microorganisms [9,17].

Environmental Fate:

- **Breakdown in soil and groundwater:** Dalapon has a low to moderate persistence in soil. It remains in the soil for 2 to 8 weeks [14]. Dalapon has residual activity in soil for 3 to 4 months when it is applied at high rates (22 kg/hectare) [9]. Dalapon does not readily bind to soil particles. In clay and clay loam soils, there may be no adsorption. Since it does not adsorb to soil particles, dalapon has a high degree of mobility in all soil types and leaching does occur. However, dalapon movement in soil is usually limited by rapid and complete breakdown of the herbicide into naturally-occurring compounds by soil microorganisms [12,14]. Dalapon is not found below the first 6-inch soil layer. Higher temperatures and increased soil moisture speed up degradation. At higher temperatures, dalapon can also be degraded by ultraviolet light from the sun [39]. In a national groundwater survey, dalapon was not found in groundwater [17].
- **Breakdown in water:** In ponds and streams, dalapon disappears via microbial degradation, hydrolysis, and photolysis [12]. Microbial degradation tends to be the most active form of its breakdown in water. In the absence of microbial degradation, the half-life of dalapon, by chemical hydrolysis, is several months at temperatures less than 25 C. Hydrolysis is accelerated with increasing temperature and pH [39,42].
- **Breakdown in vegetation:** Dalapon is absorbed by plant roots and leaves and moved (or translocated) within plants [9]. It tends to build up in the areas of greatest plant metabolic activity, such as developing seeds and in the tips of roots, shoots, and leaves. At high rates of application, dalapon precipitates out of solution as an acid, and has immediate and local acute effects on foliage [17]. It is easily washed off foliage. In addition to herbicidal activity, dalapon is a plant-growth inhibitor. Conditions of increased light and high temperature may cause nutrient solutions or soil applications of dalapon to build up in the tops of plants, via transpiration [17].

Physical Properties:

- **Appearance:** Dalapon is a type of acid that is usually formulated with sodium and magnesium salts [44]. The acid itself is not used directly. Commercial products usually contain 85% sodium salt or mixed sodium and magnesium salts of dalapon [17]. In its pure acid form, dalapon is a colorless liquid with an acrid odor. As sodium-magnesium salts, it is a white to off-white powder [9,39].
- **Chemical Name:** 2,2-dichloropropionic acid [9]
- **CAS Number:** 127-20-8 (sodium salt); 75-99-0 (acid)
- **Molecular Weight:** 164.95
- **Water Solubility:** 900,000 mg/L @ 25 C (sodium salt) [9]
- **Solubility in Other Solvents:** alkali solvents, v.s.; ethanol v.s.; acetone, benzene, and methanol s. [9]
- **Melting Point:** (with decomposition) 166.5 C (sodium salt) [9]
- **Vapor Pressure:** 0.01 mPa @ 20 C (sodium salt) [9]
- **Partition Coefficient:** 0.778 [17]
- **Adsorption Coefficient:** 1 (sodium salt) [14]

Exposure Guidelines:

- **ADI:** Not Available
- **MCL:** 0.2 mg/L [8]
- **RfD:** 0.03 mg/kg/day [8]
- **PEL:** Not Available
- **HA:** Not Available
- **TLV:** Not Available

Basic Manufacturer:

BASF Corp.
Agricultural Products Group
P.O. Box 13528
Research Triangle Park, NC 27709-3528

- **Phone:** 800-669-2273
- **Emergency:** 800-832-4357

References:

References for the information in this PIP can be found in Reference List Number 6

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2,4-DB

Trade and Other Names: 2,4-DB is 4-(2,4-dichlorophenoxy)butyric acid. Trade names for products containing 2,4-DB include Butoxone, Butyrac, Butirex, Embutone, Embutox, and Venceweed. 2,4-DB may also be found in formulations with other herbicides such as cyanazine, MCPA, benazolin, linuron, and mecoprop.

Regulatory Status: 2,4-DB is a General Use Pesticide (GUP). The U.S. Environmental Protection Agency has classified it as toxicity class III - slightly toxic. Products containing it bear the Signal Word CAUTION.

Chemical Class: phenoxy compound

Introduction: 2,4-DB is a selective systemic herbicide in the phenoxy family. It is used for the control of many annual and perennial broad-leaved weeds in alfalfa, peanuts, soybeans, and other crops. In the plant, the compound changes to 2,4-D and inhibits growth at the tips of stems and roots.

This compound is not to be confused with another phenoxy compound, 2,4-D, and its derivatives, or with the derivatives of 2,4-DB, such as the sodium salt, the isooctyl ester, or the butyl ester. Each of these are slightly different compounds and thus have different toxicities and environmental characteristics. It is found in formulations as a soluble concentrate and as an emulsified concentrate. All of the material in this profile refers to the acid form of 2,4-DB unless specifically stated otherwise. Well over 1 million pounds of 2,4-DB were used in the U.S. in 1990.

Formulation: It is found in formulations as a soluble concentrate and as an emulsified concentrate.

Toxicological Effects:

- **Acute toxicity:** 2,4-DB is a slightly toxic compound through oral exposure. It has an oral LD50 ranging between 370 mg/kg and 700 mg/kg in rats. The sodium salt has a substantially lower toxicity with an LD50 of 1500 mg/kg in rats and 400 mg/kg in mice [6,7]. In rabbits 2,4-DB is only slightly toxic when exposure is through the skin. The acute dermal LD50 of 2,4-DB is greater than 2000 mg/kg [6].
- **Chronic toxicity:** At 25 and 80 mg/kg/day, dogs experienced changes in body weight and tissue effects [33]. In a longer-term chronic feeding study with rats, 30 mg/kg effects noted included decreases in body weight, a decrease in heart weight, and changes in blood chemistry [33].
- **Reproductive effects:** Female rats fed high doses of 75 mg/kg/day of 2,4-DB, experienced a number of chronic effects, including lower ovarian weights and fewer offspring born [33]. These data suggest that reproductive effects are not likely in humans at expected exposure levels.
- **Teratogenic effects:** No data are currently available.

- **Mutagenic effects:** Chromosome changes occurred due to exposure to 2,4-DB in Chinese hamster cells; however, the exposure was relatively high (just below a threshold of significant cell toxicity) and for relatively prolonged periods of time (17 or more hours). Tests over 2 hours produced no chromosome changes [33]. The Ames test produced no chromosomal abnormalities in bacterial cells. The data, though minimal, indicate that 2,4-DB is weakly or nonmutagenic.
- **Carcinogenic effects:** Preliminary evidence suggests that 2,4-DB can cause liver cancer in mice (hepatocellular carcinomas) [33]. However, in another study with rats fed lower doses (to 30 mg/kg/day), no cancer-related changes occurred [33]. Thus the carcinogenic status of 2,4-DB is not clear.
- **Organ toxicity:** Target organs identified in long-term animal studies include the heart and liver.
- **Fate in humans and animals:** No data are currently available.

Ecological Effects:

- **Effects on birds:** Several dietary feeding tests indicate that 2,4-DB is practically nontoxic (LC50 values in excess of 5000 ppm) to waterfowl and upland game birds [33].
- **Effects on aquatic organisms:** 2,4-DB is slightly to moderately toxic to fish. The LC50 value for the compound is 18 mg/L in the fathead minnow, 7.5 to 17 mg/L in the bluegill sunfish, and 2 to 14 mg/L in the rainbow trout [33,34]. No studies were available on the toxicity of the compound to freshwater or marine invertebrates.
- **Effects on other organisms:** 2,4-DB is nontoxic to bees [7].

Environmental Fate:

- **Breakdown in soil and groundwater:** In soil, 2,4-DB is broken down by the action of soil microbes to the product 2,4-D. The half-life for 2,4-DB is about 7 days (21). Because of the risk of groundwater contamination from the whole family of 2,4-D related compounds, special advisories are required on the labels of end-use products related to 2,4-D including 2,4-DB. 2,4-DB has been detected in only 1 well in Texas (out of 928 tested) [23]. It had a concentration of 0.002 mg/L of 2,4-DB.
- **Breakdown in water:** No data are currently available.
- **Breakdown in vegetation:** The compound is taken up by the roots and moved throughout the plant. In plants, the compound 2,4-DB is degraded to 2,4-D, which is then broken down further to less toxic materials. In plants tolerant to the herbicide, the breakdown from 2,4-DB to 2,4-D is very slow [6].

Physical Properties:

- **Appearance:** 2,4-DB is a colorless to white crystal with a slightly phenolic odor. It is slightly corrosive to iron [6].
- **Chemical Name:** 4-(2,4-dichlorophenoxy)butyric acid [6]
- **CAS Number:** 94-82-6
- **Molecular Weight:** 249.10
- **Water Solubility:** 46 mg/L @ 25 C [6]
- **Solubility in Other Solvents:** acetone v.s.; ethanol v.s.; diethyl ether v.s.; benzene s.s.; toluene s.s.; and kerosene s.s. [6]
- **Melting Point:** 117-119 C [6]
- **Vapor Pressure:** Negligible (acid and salts) [6]
- **Partition Coefficient:** Not Available
- **Adsorption Coefficient:** 20 (estimated) (salt) [21]

Exposure Guidelines:

- **ADI:** Not Available
- **MCL**: Not Available
- **RfD:** 8 x 10-3 mg/kg/day [31]
- **PEL:** Not Available
- **HA**: Not Available
- **TLV**: Not Available

Basic Manufacturer:

Rhone-Poulenc Ag. Co.
P.O. Box 12014
2 T.W. Alexander Drive

Research Triangle Park, NC 27709

- **Phone:** 919-549-2000
- **Emergency:** 800-334-7577

References:

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Dicamba

Trade and Other Names: Trade names include Banfel, Banvel, Banvel CST, Banvel D, Banvel XG, Dianat, Dicazin, Fallowmaster, Mediben, Metambane, Tracker, and Trooper.

Regulatory Status: The EPA has classified this General Use Pesticide (GUP) as toxicity class III - slightly toxic. Products containing dicamba bear the Signal Word WARNING. This is because of its irritating, corrosive effect on skin and eyes.

Chemical Class: benzoic acid compound

Introduction: Dicamba is a benzoic acid herbicide. It can be applied to the leaves or to the soil. Dicamba controls annual and perennial broadleaf weeds in grain crops and grasslands, and it is used to control brush and bracken in pastures. It will kill broadleaf weeds before and after they sprout. Legumes will be killed by dicamba. In combination with a phenoxyalkanoic acid or other herbicide, dicamba is used in pastures, range land, and non-crop areas such as fence-rows and roadways to control weeds.

Formulation: Not Available

Toxicological Effects:

- **Acute toxicity:** Dicamba is slightly toxic by ingestion and slightly toxic by inhalation or dermal exposure [7]. The oral LD50 for dicamba is 757 to 1707 mg/kg in rats, 1190 mg/kg in mice, 2000 mg/kg in rabbits, and 566 to 3000 mg/kg in guinea pigs [1,6]. The dermal LD50 in rabbits is greater than 2000 mg/kg (7). The inhalation LC50 for dicamba in rats is greater than 200 mg/L [7]. Symptoms of poisoning with dicamba include loss of appetite (anorexia), vomiting, muscle weakness, slowed heart rate, shortness of breath, central nervous system effects (victim may become excited or depressed), benzoic acid in the urine, incontinence, cyanosis (bluing of the skin and gums), and exhaustion following repeated muscle spasms [1,5]. In addition to these symptoms, inhalation can cause irritation of the linings of the nasal passages and the lungs, and loss of voice [7]. Most individuals who have survived severe poisoning from dicamba have recovered within 2 to 3 days with no permanent effects [7]. Dicamba is very irritating and corrosive, and can cause severe and permanent damage to the eyes [7]. The eyelids may swell and the cornea may be cloudy for a week after dicamba is splashed in the eyes [1]. In some individuals, dicamba is a skin sensitizer and may cause skin burns [7]. There is no evidence that dicamba is absorbed into the body through the skin [1].
- **Chronic toxicity:** Doses of 25 mg/kg/day in the diet administered to rats for 2 years produced no observable effects on survival, body weight, food consumption, organ weight, blood chemistry, or tissue structure [1,5,11]. Consumption of dicamba at high levels over a long period of time has been shown to cause changes in the liver and a decrease in body weight in rats [1,5,11]. In mice, some enlargement of liver cells has occurred [1].
- **Reproductive effects:** In a three-generation study, dicamba did not affect the reproductive capacity of rats [1]. When rabbits were given doses of 0.5, 1, 3, 10, or 20 mg/kg/day of technical dicamba from days 6 through 18 of pregnancy, toxic effects on

the mothers, slightly reduced fetal body weights, and increased loss of fetuses occurred at the 10 mg/kg dose [5,10]. These data suggest that dicamba is unlikely to cause reproductive effects in humans at expected exposure levels.

- **Teratogenic effects:** No teratogenic effects have been shown in lab animals such as rabbits and rats exposed to dicamba [36].
- **Mutagenic effects:** Dicamba has not been shown to be a mutagen [5,10].
- **Carcinogenic effects:** Rats fed up to 25 mg dicamba/kg/day for 2 years showed no increased incidence of tumors [5,10]. This evidence suggests that dicamba is not carcinogenic.
- **Organ toxicity:** Chronic exposure can lead to the development of the same symptoms as described for acute exposure.

- **Fate in humans and animals:** Dicamba was excreted rapidly by rats, mainly in the urine, when administered orally or subcutaneously; 1 to 4% was excreted in the feces [1]. Mice, rats, rabbits, and dogs excreted 85% of an oral dose as unmetabolized dicamba in the urine within 48 hours of dosing. Eventually, between 90 and 99% of the dose was excreted unmetabolized in the urine. This indicates that dicamba is rapidly absorbed into the bloodstream from the gastrointestinal tract [10]. When dicamba was ingested daily in the feed, the concentrations in different organs reached a steady state within 2 weeks. When daily intake stopped, storage in the organs declined rapidly [1]. It is therefore concluded that dicamba does not bioaccumulate in mammalian tissues.

Ecological Effects:

- **Effects on birds:** Dicamba is practically nontoxic to birds. The LD50 for technical dicamba in mallard ducks is 2009 mg/kg. The 8-day dietary LC50 in mallards and in bobwhite quail is greater than 10,000 ppm [5,7].

- Effects on aquatic organisms: Dicamba is of low toxicity to fish [5,7]. The LC50 (96-hour) for technical dicamba is 135 mg/L in rainbow trout and bluegill sunfish, greater than 100 mg/L in grass shrimp, and greater than 180 mg/L in fiddler crab and sheepshead minnow [5]. The LC50 (48-hour) for dicamba is 35 mg/L in rainbow trout, 40 mg/L in bluegill, 465 mg/L in carp, and 110 mg/L in Daphnia magna, a small freshwater crustacean [5,7].
- **Effects on other organisms:** Dicamba poses little threat to wildlife. Dicamba is not toxic to bees [7].

Environmental Fate:

- **Breakdown in soil and groundwater:** Dicamba is moderately persistent in soil. The half-life of dicamba in soil is typically 1 to 4 weeks [21]. Under conditions suitable for rapid metabolism, the half-life is less than 2 weeks [20]. Metabolism by soil microorganisms is the major pathway of loss under most soil conditions. The rate of biodegradation increases with temperature and increasing soil moisture, and tends to be faster when soil is slightly acidic. When soil moisture increases above 50%, the rate of biodegradation declines [5]. Dicamba slowly breaks down in sunlight [5]. Volatilization from soil surfaces is probably not significant, but some volatilization may occur from plant surfaces [20]. It is stable to water and other chemicals in the soil [6]. Dicamba does not bind to soil particles and is highly soluble in water. It is therefore highly mobile in the soil and may contaminate groundwater [21]. In humid areas, dicamba will be leached from the soil in 3-12 weeks [20].
- **Breakdown in water:** In water, microbial degradation is the main route of dicamba disappearance. Photolysis may also occur. Aquatic hydrolysis, volatilization, adsorption to sediments, and bioconcentration are not expected to be significant [20].
- **Breakdown in vegetation:** Dicamba is rapidly taken up by the leaves and roots of plants, and it is readily translocated to other plant parts. It some plant species, dicamba accumulates in the tips of mature leaves [5]. Desirable broadleaf plants such as fruit trees and tomatoes may be harmed during their growth and development stages [5]. Residues of dicamba on treated plants can disappear through exudation from the roots into the surrounding soil, metabolism within the plant, or by loss from leaf surfaces [5].

Physical Properties:

- **Appearance:** Pure dicamba is an odorless, white crystalline solid. The technical acid is a pale buff crystalline solid [6].
- **Chemical Name:** 3,6-dichloro-O-anisic acid [6]
- **CAS Number:** 1918-00-9
- **Molecular Weight:** 221.04
- **Water Solubility:** 6500 mg/L @ 25 C [6]
- **Solubility in Other Solvents:** acetone s.; dichloromethane s.; dioxane v.s.; ethanol s.; toluene s.; xylene s. [6]
- **Melting Point:** 114-116 C [6]
- **Vapor Pressure:** 4.5 mPa @ 25 C [6]
- **Partition Coefficient:** -0.5376 [5]
- **Adsorption Coefficient:** 2 (salt) [21]

Exposure Guidelines:

- **ADI:** Not Available
- **MCL**: Not Available
- **RfD:** 0.03 mg/kg/day [31]
- **PEL:** Not Available
- **HA**: 0.2 mg/L [10]
- **TLV**: Not Available

Basic Manufacturer:

Sanex, Inc.
5300 Harvester Road
Burlington, Ontario
L7L 5N5 Canada

- **Phone:** 905-639-7535
- **Emergency:** Not Available

References:

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Revised June 1996

Dinoseb

Trade and Other Names: Product names for pesticides containing dinoseb include Basanite, Caldon, Chemox, Chemsect DNBP, Dinitro, Dynamyte, Elgetol, Gebutox, Hel-Fire, Kiloseb, Nitropone, Premerge, Sinox General, Subitex, and Vertac Weed Killer.

Regulatory Status: The use of dinoseb was cancelled in the U.S. in 1986. This action was based on the potential risk of birth defects and other adverse health effects for applicators and other persons with substantial dinoseb exposure. This pesticide is not commercially available in the U.S.

Dinoseb is a highly toxic compound. Prior to its ban, it was a Restricted Use Pesticide (RUP), and products containing dinoseb were required to be labeled with a DANGER Signal Word.

Chemical Class: phenolic compound

Introduction: Dinoseb is a phenolic herbicide used in soybeans, vegetables, fruits and nuts, citrus, and other field crops for the selective control of grass and broadleaf weeds (e.g., in corn). It is also used as an insecticide in grapes, and as a seed crop drying agent. It is produced in emuslifiable concentrates or as water soluble ammonium or amine salts. The information presented here pertains to the technical grade of dinoseb unless otherwise specified.

Formulation: It is produced in emuslifiable concentrates or as water soluble ammonium or amine salts.

Toxicological Effects:

- **Acute toxicity:** Dinoseb is highly toxic by ingestion, with reported oral LD50 values of 25 to 58 mg/kg in rats and 25 mg/kg in guinea pigs [1,29]. It is highly toxic by skin exposure, with a reported dermal LD50 of 80 to 200 mg/kg in rabbits and 200 to 300 mg/kg in guinea pigs [1,29]. Dinoseb did not cause skin irritation in rabbits [29]. Inhalation of dusts and sprays may be irritating to the lungs and eyes, and may cause serious illness; direct skin contact may cause irritation, yellow stains, burns, and dermatitis, and more serious effects in humans [29]. In one fatal incident, a farm worker was using a backpack hand-held sprayer that leaked dinoseb onto his body and penetrated his skin [29]. Symptoms in persons receiving accidental exposure include fatigue, thirst, sweating, insomnia, weight loss, headache, flushing of the face, nausea, abdominal pain, and occasional diarrhea [29]. In one case, some of these persisted for several months following exposure [29].
- **Chronic toxicity:** Dinoseb interferes with cellular conversion of food molecules (such as glucose) into useable energy for the body [83]. Specifically, it disturbs the production of adenosine triphosphate (ATP) in the mitochondria of the cells, ATP being the molecule that provides energy for all cellular activities [83]. This may account for many of the toxic effects caused by dinoseb. Dietary levels of about 25 mg/kg/day caused marked food refusal and some deaths after five or more doses [29].

Lower doses (5 to 20 mg/kg/day) caused statistically significant decreases in growth [29]. Some formulations of dinoseb may cause anemia-like effects, jaundice, and increased excretion of hemoglobin-related compounds (coproporphyrinuria) [29].

- **Reproductive effects:** Dinoseb is reported to adversely affect reproduction in rats and mice at levels that are commonly found among occupational workers. Decreased sperm count and abnormal sperm shape were observed in male rats and mice after 3 weeks at low exposure levels (about 10 mg/kg/day) for 30 days [84]. In a separate study, rats were exposed to relatively small quantities of dinoseb through their diet for a total of 22 weeks. Effects such as decreased fertility, slow weight gain, and poor survival of newborns appeared to be related to this pesticide [85]. Because of the adverse effects observed in laboratory animals at low chronic exposure levels, it is believed that dinoseb may cause decreased fertility or sterility in humans [83].
- **Teratogenic effects:** Low levels of dinoseb fed to rats and rabbits caused birth defects in the fetuses of exposed females [8]. When dinoseb was administered to pregnant mice, its breakdown products were found in the embryos. However, no teratogenic effects were noted. Various tests of mice and rats fed or injected with small amounts of dinoseb (around 10 mg/kg/day) have shown maternal toxicity, decreased fetal body weights and changes in fetal development [29]. In some studies of mice, oral doses to pregnant mothers caused an increased death rate in the exposed animals but caused no fetal damage [8]. Other studies indicate that dinoseb is a stronger teratogen when injected than when ingested. At low feeding levels the compound was responsible for skeletal deformities and neurological problems in newborn rats [83]. Based on the data, dinoseb may produce teratogenic effects in humans.
- **Mutagenic effects:** Dinoseb was not mutagenic or genotoxic in laboratory studies performed using eukaryotic cells (the type found in mammals and other higher order species) [29,83]. This evidence suggests that mutagenic effects in humans due to dinoseb exposure are unlikely.
- **Carcinogenic effects:** Dinoseb didn't cause significant increases in tumors when administered to two strains of mice at the maximum tolerated dose over a period of 18 months [29]. While not carcinogenic to male mice, it was found to be carcinogenic to female mice in another study [83]. The compound caused liver cancer in these animals at moderate to high doses [83]. The evidence regarding the carcinogenic potential of dinoseb is currently inconclusive.
- **Organ toxicity:** Dinoseb has the potential to damage the eye. It may also affect the immune system, liver, kidneys, and spleen [29].

- **Fate in humans and animals:** Dinoseb is readily absorbed through the skin, gastrointestinal tract, and lung surface [29]. Esters of dinoseb are rapidly transformed into dinoseb, which is the active toxicant [29]. The chemical is excreted in the urine and feces and is metabolized in the liver. Breakdown products are found in the liver, kidneys, spleen, blood, and urine [29]. Dinoseb can also pass through the placenta into the fetus of experimental animals.

Ecological Effects:

- **Effects on birds:** The compound is very highly toxic to birds, with reported acute oral LD50 values between 7 and 9 mg/kg [8]; its reported 5- to 8-day dietary LC50 ranges from 409 ppm in quail to 515 ppm in pheasants [8]. It thus has the potential to negatively impact local pheasant and songbird populations.
- **Effects on aquatic organisms:** Dinoseb is highly toxic to fish, with reported 96-hour LC50 values ranging from 44 ug/L in lake trout to 118 ug/L in catfish [8]. Other 96-hour LC50 values are 100 ug/L in coho salmon and 67 ug/L in cutthroat trout [8,37]. It is more toxic to fish in acidic water than in neutral or alkaline water [37]. Dinoseb has caused fish kills in small Scottish streams when washed from fields by rain [88]. The bioconcentration factor is 135 (86). Dinoseb is rapidly taken up by fish, but is is rapidly eliminated from exposed fish if placed in clean water. Dinoseb, thus, does not pose a significant risk for bioaccumulation [84].
- **Effects on other organisms:** Dinoseb is toxic to bees [1].

Environmental Fate:

- **Breakdown in soil and groundwater:** Dinoseb is of low persistence regardless of the form (phenolic or salt). Reported field half-lives for both types of dinoseb range from 5 to 31 days [53]. An overall representative value is estimated to be 20 tp 30 days in most circumstances, although persistence may be much longer in the vadose zone [9]. Photodegradation and microbial breakdown may play roles in the breakdown of dinoseb in the soil environment, but volatilization should not be a significant route of loss [9]. The phenolic form of dinoseb is slightly soluble in water and moderately sorbed by most soils [53]. Studies have shown soil sorption capacity to be much greater at lower pH values [9]. It thus should present only a moderate risk to groundwater. On the other hand, the ammonium and amine salt forms of dinoseb are much more water-soluble and much less strongly bound to soils [8]. These may pose a significant risk to groundwater. Over a 10-year period, dinoseb was found to be one of three particularly persistent contaminants in Ontario wells [8]. Entry to the wells was due to spills of concentrated and dilute herbicide, drift during spraying, and from storm runoff. Well water concentrations ranged from 0.05 to 5000 ug/L in these wells, and removal of dinoseb proved to be very difficult [9].
- **Breakdown in water:** Photodegradation may occur in surface waters, but hydrolysis is essentially negligible [9]. Dinoseb has been found in streams at about 5 ug/L [8].

- **Breakdown in vegetation:** Dinoseb persists on treated crop soils for 2 to 4 weeks, under average conditions of use [8].

Physical Properties:

- **Appearance:** Dinoseb is a dark reddish-brown solid or dark orange viscous liquid, depending on the temperature [1].
- **Chemical Name:** 2-(sec-butyl)-4,6-dinitrophenol [1]
- **CAS Number:** 88-85-7
- **Molecular Weight:** 240.22
- **Water Solubility:** 52 mg/L @ 20 C [1]
- **Solubility in Other Solvents:** v.s. in alcohol, ethanol, and heptane; s. in spray oil and in most organic solvents and oils [1]
- **Melting Point:** 32-42 C [1]
- **Vapor Pressure:** 6.7 mPa @ 25 C [53]
- **Partition Coefficient:** Not Available
- **Adsorption Coefficient:** 30 (estimated) [53]

Exposure Guidelines:

- **ADI:** Not Available
- **MCL:** Not Available
- **RfD:** 0.001 mg/kg/day [13]
- **PEL:** Not Available
- **HA:** Not Available
- **TLV:** Not Available

Basic Manufacturer:

DowElanco
9330 Zionsville Road
Indianapolis, IN 46268-1054

- **Phone:** 317-337-7344
- **Emergency:** 800-258-3033

References:

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ENDOTHALL

TRADE OR OTHER NAMES: Endothall is endothal in Great Britain. Trade names for the acid form of endothall (technical endothall) include Aquathol, Hydrothal-47 and Hydrothal-191. Trade names for the disodium salt of endothall (disodium endothall) include Accelerate, Des-I-Cate, Tri-endothal, Ripenthol, Hydrothol, and Niagrathol (1, 211, 226). The amine salt of endothall is also called Hydrothol (22).

REGULATORY STATUS: Endothall is a general use pesticide (GUP). When used as an aquatic herbicide, some water use restrictions may apply (307).

INTRODUCTION: Endothall is a member of the dicarboxylic acid chemical class (308,207). It is a selective contact herbicide. The potassium and amine salts of endothall are used as aquatic herbicides to control a variety of plants including plankton, pondweed, niad, coontail, milfoil, elodea, and algaes in water bodies and rice fields. Endothall is also used to control annual grass and broadleaf weeds in sugar beets, spinach and turf. It reduces sucker branch growth in hops. Endothall is a desiccant to aid the harvest of alfalfa, potatoes, clover, and cotton (1). The EPA has classified endothall as Toxicity Class II - moderately toxic. Products containing endothall bear the SIGNAL WORD: WARNING (223).

FORMULATION: It is available as granules or as a soluble concentrate (1).

TOXICOLOGICAL EFFECTS

- **Acute Toxicity:** Endothall is moderately toxic. The LD50 is the dose which kills half of the test animals treated. The oral LD50 for disodium endothall is 51 mg/kg for rats and 250 mg/kg for guinea pigs (31, 211). The LD50 is 750 mg/kg for rats and 100 mg/kg for rabbits whose skin is exposed to disodium endothall (31, 227, 309). In humans, ingestion of 7 to 8g of disodium endothall causes repeated vomiting, hemorrhages, swelling in the lungs, and bleeding in the gastrointestinal tract (310). The LD50 for the amine salt of endothall is 206 mg/kg for rats and 143 mg/kg for rabbits whose skin is exposed to it (31, 1). The oral LD50 for technical endothall is 38 mg/kg for rats. Endothall is very irritating to the eyes, skin, and mucous membranes (31, 1, 308).
- **Chronic Toxicity:** Information follows.
- **Reproductive Effects:** A three generation study was conducted by feeding male and female rats disodium endothall until they were 100 days old and then mating them. Three successive generations were maintained on the test diet for 100 days and then bred to produce the next generation. When examined at 21 days, rat pups in all three generations whose parents were given 15 mg/kg/day of disodium endothall had decreased body weights. No adverse reproductive effects were observed (NOEL) at 5 mg/kg/day (310).There were no observable signs of developmental toxicity at dose levels that were fatal to the females (310).
- **Teratogenic Effects:** Technical endothall was not teratogenic at the highest dose tested, 30 mg/kg/day (310).
- **Mutagenic Effects:** Studies show that technical endothall is not mutagenic in Salmonella bacteria nor in mouse cells. Aquathol K, a formulation of dipotassium endothall, is not mutagenic in fruit flies, mold, or human white blood cells. However, "commercial endothall," with no further description, was mutagenic in fruit flies (310).
- **Carcinogenic Effects:** No statistically significant numbers or types of tumors were observed in rats fed as much as 125 mg/kg/day of disodium endothall for two years. Thus, available evidence suggests that endothall does not cause cancer (310).
- **Organ Toxicity:** In male dogs, high doses of 20 mg/kg/day of disodium endothall for 6 weeks caused vomiting, diarrhea,

damaged intestinal walls, and hemorrhages in the stomach. In rats, very high doses of 50 mg/kg/day of disodium endothall for four weeks caused liver and kidney damage (310).

- **Fate in Humans and Animals:** In rats dosed with technical endothall, over 95% of the dose was excreted within 48 hours. Within 72 hours after dosing, 99% of the dose was excreted. Approximately 90% of a dose of technical endothall is excreted in the feces and 7% in urine(310).

ECOLOGICAL EFFECTS

- **Effects on Birds:** No information is currently available.
- **Effects on Aquatic Organisms:** Endothall is toxic to some species of fish (1). Inorganic salts of endothall in aquatic formulations are safe to fish in 100-500 ppm concentrations. However, amine salts of endothall are more toxic to fish than the dipotassium endothall (312). Endothall has a low toxicity to crustaceans and a medium toxicity to aquatic insects (226). Long-term ingestion may cause severe damage to the digestive tract, liver and testes in fish (30).
- **Effects on Other Animals (Nontarget species):** Endothall is not toxic to bees (1).

ENVIRONMENTAL FATE

- **Breakdown of Chemical in Soil and Groundwater:** Endothall is highly mobile in soil, however rapid degradation limits the extent of leaching. Endothall disappears from soil in 7-21 days (1). The half-life, the amount of time needed for the concentration to be reduced by half, of endothall in soil is 4-5 days in clay soils and 9 days in soils with high organic content (9).
- **Breakdown of Chemical in Surface Water:** Endothall is rapidly degraded in water (1, 65). Its half-life is 4 to 7 days for dipotassium endothall and about 7 days for technical endothall in surface water (312). It biodegrades more slowly when air is not present (9).
- **Breakdown of Chemical in Vegetation:** No information is currently available.

PHYSICAL PROPERTIES AND GUIDELINES

Physical Properties: All properties are for technical endothall unless otherwise noted.

- **Appearance:** Technical endothall is a colorless or white crystal which is stable to light, weak acidic media and weak alkaline media.
- **Chemical Name:** 3,6-endoxohexahydrophthalic acid (technical endothall),Disodium-3,6-endoxohexahydrophthalate (disodium endothall), 3,6-endoxohexahydrophthalic acid amine salt (amine salt of endothall)
- **CAS Number:** 145-73-3 (technical endothall), 129-67-9 (disodium salt of endothall), 6385-60-0 (amine salt of endothall)
- **Molecular Weight:** 186.2
- **Water Solubility:** 100 g/kg at 20 degrees C (1, 9)
- **Solubility in Other Solvents:** Soluble in benzene, isopropanol, acetone, dioxane, and methanol (disodium salt of endothall and technical endothall) (1)
- **Melting Point:** 144 degrees C (1)
- **Vapor Pressure:** 2.09 x 10-5 mPa (24.3 degrees C)
- **Partition Coefficient:** Not Available
- **Adsorption Coefficient:** -0.87 (9)

Exposure Guidelines: All guidelines are for technical endothall unless otherwise noted.

- **ADI:** 0.02 mg/kg (310)
- **MCL:**0.1 mg/l (65)
- **RfD:** 0.02 mg/kg/day (310)
- **PEL:** Not Available
- **HA:** 0.8 mg/l (310)
- **TLV:** Not Available

BASIC MANUFACTURER:

ELF Atochem, North America
Three Parkway, Room 619
Philadelphia, PA 19102

- **Telephone:** 215-587-7885

REFERENCES

References for the information in this PIP can be found in Reference List Number 10

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Glyphosate

Trade and Other Names: Trade names for products containing glyphosate include Gallup, Landmaster, Pondmaster, Ranger, Roundup, Rodeo, and Touchdown. It may be used in formulations with other herbicides.

Regulatory Status: Glyphosate acid and its salts are moderately toxic compounds in EPA toxicity class II. Labels for products containing these compounds must bear the Signal Word WARNING. Glyphosate is a General Use Pesticide (GUP).

Chemical Class: Not Available

Introduction: Glyphosate is a broad-spectrum, nonselective systemic herbicide used for control of annual and perennial plants including grasses, sedges, broad-leaved weeds, and woody plants. It can be used on non-cropland as well as on a great variety of crops. Glyphosate itself is an acid, but it is commonly used in salt form, most commonly the isopropylamine salt. It may also be available in acidic or trimethylsulfonium salt forms. It is generally distributed as water-soluble concentrates and powders. The information presented here refers to the technical grade of the acid form of glyphosate, unless otherwise noted.

Formulation: Glyphosate itself is an acid, but it is commonly used in salt form, most commonly the isopropylamine salt. It may also be available in acidic or trimethylsulfonium salt forms. It is generally distributed as water-soluble concentrates and powders.

Toxicological Effects:

- **Acute toxicity:** Glyphosate is practically nontoxic by ingestion, with a reported acute oral LD50 of 5600 mg/kg in the rat. The toxicities of the technical acid (glyphosate) and the formulated product (Roundup) are nearly the same [58,96]. The oral LD50 for the trimethylsulfonium salt is reported to be approximately 750 mg/kg in rats, which indicates moderate toxicity [58]. Formulations may show moderate toxicity as well (LD50 values between 1000 mg/kg and 5000 mg/kg) [58]. Oral LD50 values for glyphosate are greater than 10,000 mg/kg in mice, rabbits, and goats [8,96]. It is practically nontoxic by skin exposure, with reported dermal LD50 values of greater than 5000 mg/kg for the acid and isopropylamine salt. The trimethylsulfonium salt has a reported dermal LD50 of greater than 2000 mg/kg. It is reportedly not irritating to the skin of rabbits, and does not induce skin sensitization in guinea pigs [58]. It does cause eye irritation in rabbits [58]. Some formulations may cause much more extreme irritation of the skin or eyes [58]. In a number of human volunteers, patch tests produced no visible skin changes or sensitization [58]. The reported 4-hour rat inhalation LC50 values for the technical acid and salts were 5 to 12 mg/L [58], indicating moderate toxicity via this route. Some formulations may show high acute inhalation toxicity [58]. While it does contain a phosphatyl functional group, it is not structually similar to organophosphate pesticides which contain organophosphate esters, and it does not significantly inhibit cholinesterase activity [1,58].
- **Chronic toxicity:** Studies of glyphosate lasting up to 2 years, have been conducted with rats, dogs, mice, and rabbits, and with few exceptions no effects were observed [96]. For example, in a chronic feeding study with rats, no toxic effects were

observed in rats given doses as high as 400 mg/kg/day [58]. Also, no toxic effects were observed in a chronic feeding study with dogs fed up to 500 mg/kg/day, the highest dose tested [58,97].

- **Reproductive effects:** Laboratory studies show that glyphosate produces reproductive changes in test animals very rarely and then only at very high doses (over 150 mg/kg/day) [58,96]. It is unlikely that the compound would produce reproductive effects in humans.
- **Teratogenic effects:** In a teratology study with rabbits, no developmental toxicity was observed in the fetuses at the highest dose tested (350 mg/kg/day) [97]. Rats given doses up to 175 mg/kg/day on days 6 to 19 of pregnancy had offspring with no teratogenic effects, but other toxic effects were observed in both the mothers and the fetuses. No toxic effects to the fetuses occurred at 50 mg/kg/day [97]. Glyphosate does not appear to be teratogenic.
- **Mutagenic effects:** Glyphosate mutagenicity and genotoxicity assays have been negative [58]. These included the Ames test, other bacterial assays, and the Chinese Hamster Ovary (CHO) cell culture, rat bone marrow cell culture, and mouse dominant lethal assays [58]. It appears that glyphosate is not mutagenic.
- **Carcinogenic effects:** Rats given oral doses of up to 400 mg/kg/day did not show any signs of cancer, nor did dogs given oral doses of up to 500 mg/kg/day or mice fed glyphosate at doses of up to 4500 mg/kg/day [58]. It appears that glyphosate is not carcinogenic [97].
- **Organ toxicity:** Some microscopic liver and kidney changes, but no observable differences in function or toxic effects, have been seen after lifetime administration of glyphosate to test animals [97].

- **Fate in humans and animals:** Glyphosate is poorly absorbed from the digestive tract and is largely excreted unchanged by mammals. At 10 days after treatment, there were only minute amounts in the tissues of rats fed glyphosate for 3 weeks [98]. Cows, chickens, and pigs fed small amounts of glyphosate had undetectable levels (less than 0.05 ppm) in muscle tissue and fat. Levels in milk and eggs were also undetectable (less than 0.025 ppm). Glyphosate has no significant potential to accumulate in animal tissue [99].

Ecological Effects:

- **Effects on birds:** Glyphosate is slightly toxic to wild birds. The dietary LC50 in both mallards and bobwhite quail is greater than 4500 ppm [1].
- **Effects on aquatic organisms:** Technical glyphosate acid is practically nontoxic to fish and may be slightly toxic to aquatic invertebrates. The 96-hour LC50 is 120 mg/L in bluegill sunfish, 168 mg/L in harlequin, and 86 mg/L in rainbow trout [58]. The reported 96-hour LC50 values for other aquatic species include greater than 10 mg/L in Atlantic oysters, 934 mg/L in fiddler crab, and 281 mg/L in shrimp [58]. The 48-hour LC50 for glyphosate in Daphnia (water flea), an important food source for freshwater fish, is 780 mg/L [58]. Some formulations may be more toxic to fish and aquatic species due to differences in toxicity between the salts and the parent acid or to surfactants used in the formulation [58,96]. There is a very low potential for the compound to build up in the tissues of aquatic invertebrates or other aquatic organisms [96].
- **Effects on other organisms:** Glyphosate is nontoxic to honeybees [1,58]. Its oral and dermal LD50 is greater than 0.1 mg/ bee [98]. The reported contact LC50 values for earthworms in soil are greater than 5000 ppm for both the glyphosate trimethylsulfonium salt and Roundup [58].

Environmental Fate:

- **Breakdown in soil and groundwater:** Glyphosate is moderately persistent in soil, with an estimated average half-life of 47 days [58,11]. Reported field half-lives range from 1 to 174 days [11]. It is strongly adsorbed to most soils, even those with lower organic and clay content [11,58]. Thus, even though it is highly soluble in water, field and laboratory studies show it does not leach appreciably, and has low potential for runoff (except as adsorbed to colloidal matter) [3,11]. One estimate indicated that less than 2% of the applied chemical is lost to runoff [99]. Microbes are primarily responsible for the breakdown of the product, and volatilization or photodegradation losses will be negligible [58].
- **Breakdown in water:** In water, glyphosate is strongly adsorbed to suspended organic and mineral matter and is broken down primarily by microorganisms [6]. Its half-life in pond water ranges from 12 days to 10 weeks [97].
- **Breakdown in vegetation:** Glyphosate may be translocated throughout the plant, including to the roots. It is extensively metabolized by some plants, while remaining intact in others [1].

Physical Properties:

- **Appearance:** Glyphosate is a colorless crystal at room temperature [1].
- **Chemical Name:** N-(phosphonomethyl) glycine [1]
- **CAS Number:** 1071-83-6
- **Molecular Weight:** 169.08

- **Water Solubility:** 12,000 mg/L @ 25 C [1]
- **Solubility in Other Solvents:** i.s. in common organics (e.g., acetone, ethanol, and xylene) [1]
- **Melting Point:** 200 C [1]
- **Vapor Pressure:** negligible [1]
- **Partition Coefficient:** -3.2218 - -2.7696 [58]
- **Adsorption Coefficient:** 24,000 (estimated) [11]

Exposure Guidelines:

- **ADI:** 0.3 mg/kg/day [12]
- **MCL**: Not Available
- **RfD:** 0.1 mg/kg/day [13]
- **PEL:** Not Available
- **HA**: 0.7 mg/L (lifetime) [98]
- **TLV**: Not Available

Basic Manufacturer:

Monsanto Company
800 N. Lindbergh Blvd.
St. Louis, MO 63167

- **Phone:** 314-694-6640
- **Emergency:** 314-694-4000

References:

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MECOPROP

TRADE OR OTHER NAMES: Mecoprop is commonly called MCPP. Trade names include Kilprop, Mecopar,Triester-II, Mecomin-D, Triamine-II (with MCPA and 2.4-DP), Triplet (with 2,4-D and dicamba), TriPower (with MCPA and dicamba), Trimec (with 2,4-D and dicamba), Trimec-Encore (with MCPA and dicamba), and U46 KV Fluid (41, 43).

REGULATORY STATUS: Mecoprop is a general use pesticide (GUP).

INTRODUCTION: Mecoprop is a selective, hormone-type phenoxy herbicide. It is applied postemergence and is used on ornamentals and sports turf, for forest site preparation, and on drainage ditch banks for selective control of surface creeping broadleaf weeds such as clovers, chickweed, lambsquarters, ivy, plantain and others. It is also used on wheat, barley, and oats (41, 43). Mecoprop is absorbed by plant leaves and translocated to the roots. It affects enzyme activity and plant growth (6). It acts relatively slowly requiring three to four weeks for control (43). The U.S. EPA has classified mecoprop as toxicity class III- slightly toxic. Products containing mecoprop bear the Signal Word "Caution" (41). It is available as a liquid concentrate, granules, and is sprayed on fertilizer pellets to produce weed and feed products (45).

TOXICOLOGICAL EFFECTS

- **Acute Toxicity:** Mecoprop has a low acute toxicity to test animals. The LD50, the oral dose that kills half of the test animals, is 930 -1210 mg/kg for rats and 650 mg/kg for mice (41, 43, 44). The LD50 for rats exposed dermally is greater than 4000 mg/kg (41, 40). Mecoprop is irritating to skin and eyes. It causes redness and swelling and can cause cloudy vision (46). The concentration in air which kills half of the test animals, the LC50 (4 hours) for rats, is greater than 12.5 mg/l air (6).
- **Chronic Toxicity:** No information is currently available.
- **Reproductive Effects:** No information is currently available.
- **Teratogenic Effects:** Mecoprop is a teratogen in rats at moderate to high doses. Oral doses of 125 mg/kg/day of MCPP in pregnant rats from days 6 to 15 of gestation caused increased intra-uterine deaths, decreased body lengths, and an increased incidence of delayed or absent bone formation in offspring. Mecoprop is not teratogenic in rabbits (45).
- **Mutagenic Effects:** Studies show that mecoprop may be mutagenic at very high doses. Tests of mecoprop on four strains of salmonella and on s. colelicolor showed no mutagenic effects (43). However, MCPP caused an increase in sister chromatid exchange after single oral doses of 470 and 3,800 mg/kg in Chinese hamsters (45).
- **Carcinogenic Effects:** A study of people employed in the manufacture of phenoxy herbicides including mecoprop showed an association between these herbicides and cancer of soft tissues and non-Hodgkin's lymphoma (47). However, other data do not support this conclusion (49). Thus, it is not clear if occupational exposures to phenoxy herbicides can cause cancer.
- **Organ Toxicity:** Oral doses of 9 mg/kg/day to female rats and 27 mg/kg/day to male rats cause kidney damage (45).
- **Fate in Humans and Animals:** Mecoprop is eliminated unchanged in the urine of mammals(6).

ECOLOGICAL EFFECTS

- **Effects on Birds:** Mecoprop is practically non-toxic to birds. The LC50 is greater than 5,620 ppm for mallard ducks and 5,000 ppm for bobwhite quail (45). The LD50 (oral) is 740 mg/kg for Japanese quail and 700 mg/kg for bobwhite quail (6).
- **Effects on Aquatic Organisms:** Mecoprop is virtually non-toxic to fish. Available data indicate a low potential of mecoprop

to bioaccumulate in fish (45). The LC50 (96 hours) is 124 ppm for rainbow trout and greater than 100 ppm for bluegill sunfish (6, 45).

- **Effects on Other Animals (Nontarget species):** Mecoprop is not toxic to bees (41).

ENVIRONMENTAL FATE

- **Breakdown of Chemical in Soil and Groundwater:** The duration of mecoprop's residual activity in soil is about two months. Adsorption of mecoprop increases with an increase in organic matter in the soil. Unaged MCPP and its salt forms are very mobile in a variety of soils (6). Because of this high mobility, it may potentially leach into groundwater (45). However, in general, phenoxy herbicides such as MCPP are not sufficiently persistent to reach groundwater (45).
- **Breakdown of Chemical in Surface Water:** No information is currently available.
- **Breakdown of Chemical in Vegetation:** No information is currently available.

PHYSICAL PROPERTIES AND GUIDELINES

It exists as a mixture of two optically active isomers of which one, the R+ form, mecoprop-p is herbicidally active (6). It is stable to heat, hydrolysis, reduction, and oxidation. It is acidic. Solutions of the salts of mecoprop are stable for several years under normal storage conditions. In cooler temperatures, the salt may crystallize out of solution but will re-dissolve on warming (41, 45).

Physical Properties: Properties are of the acid form unless otherwise noted.

- **Appearance:**Mecoprop is an odorless, white to light brown crystalline solid.
- **Chemical Name:** 2-(4-chloro-2-methyl phenoxy) propionic acid
- **CAS Number:** 7085-19-0 (acid) 1929-86-8 (potassium salt) 1432-14-0 (diethanolamine salt) 28473-03-2 (isooctyl ester)
- **Molecular Weight:** 214.6
- **Water Solubility:** all forms are very soluble in water at 20 degrees C (6)Soluble in acetone, alcohol, benzene, diethyl ether, and ethyl acetate (6)
- **Solubility in Other Solvents:** In acetone, diethyl ether, ethanol > 1000
- **Melting Point:** 94-95 degrees C (6)
- **Vapor Pressure:** 0.31 mPa (20 degrees C)
- **Partition Coefficient:** 1.26 at pH 7 (6)
- **Adsorption Coefficient:** Not Available

Exposure Guidelines: Guidelines are for the acid form unless otherwise noted.

- **ADI:** Not Available
- **MCL:** Not Available
- **RfD:** 1 x 10 to the minus 3 mg/kg/day (48)
- **PEL:** Not Available
- **HA:** Not Available
- **TLV:** Not Available

BASIC MANUFACTURER:

PBI/Gordon
P.O. Box 4090
1217 W. 12th Street
Kansas City, MO 64101

- **Telephone:** 816-421-4070

REFERENCES

References for the information in this PIP can be found in Reference List Number 7

DISCLAIMER: The information in this profile does not in any way replace or supersede the information on the pesticide product

label/ing or other regulatory requirements. Please refer to the pesticide product label/ing.

EXTOXNET

Extension Toxicology Network

Pesticide Information Profiles

A Pesticide Information Project of Cooperative Extension Offices of Cornell University, Oregon State University, the University of Idaho, and the University of California at Davis and the Institute for Environmental Toxicology, Michigan State University. Major support and funding was provided by the USDA/Extension Service/National Agricultural Pesticide Impact Assessment Program.

EXTOXNET primary files maintained and archived at Oregon State University

Revised June 1996

Picloram

Trade and Other Names: Commercial products containing the compound include Access, Grazon, Pathway, and Tordon. It may be used in formulations with other herbicides such as bromoxynil, diuron, 2,4-D, MCPA, triclorpyr, and atrazine. It is also compatible with fertilizers.

Regulatory Status: Picloram is a slightly toxic compound in EPA toxicity class III. Products containing it must bear the Signal Word CAUTION on the label. All products except for Tordon RTU and Pathway are Restricted Use Pesticides (RUPs). RUPs may be purchased and used only by certified applicators.

Chemical Class: pyridine compound

Introduction: Picloram, in the pyridine family of compounds, is a systemic herbicide used for control of woody plants and a wide range of broad-leaved weeds. Most grasses are resistant to picloram, so it is used in range management programs. Picloram is formulated either as an acid (technical product), a potassium or triisopropanolamine salt, or an isooctyl ester, and is available as either soluble concentrates, pellets, or granular formulations. The materials in this document refer to the technical acid form unless otherwise indicated.

Formulation: Picloram is formulated either as an acid (technical product), a potassium or triisopropanolamine salt, or an isooctyl ester, and is available as either soluble concentrates, pellets, or granular formulations.

Toxicological Effects:

- **Acute toxicity:** Picloram is slightly to practically nontoxic via ingestion, with reported oral LD50 values of greater than 5000 mg/kg to 8200 mg/kg in rats, 2000 to 4000 mg/kg in mice, and approximately 2000 mg/kg in rabbits [1,58]. The reported dermal LD50 in rabbits is greater than 4000 mg/kg, a level which produced no mortality or toxic signs [58,6]. This indicates slight toxicity via the dermal route as well. Technical picloram is reported to cause no skin and moderate eye irritation in the rabbit, and to cause no skin sensitization in the guinea pig [1,58]. Some formulations have caused mild or slight skin irritation and skin sensitization in test animals [58]. The technical grade is moderately toxic by inhalation, with a reported 4-hour inhalation LC50 of greater than 0.35 mg/L [3]. Formulated products may show a lesser toxicity via this route [58]. There is no documented history of human intoxication by picloram, so symptoms of acute exposure are difficult to characterize.
- **Chronic toxicity:** Male mice receiving picloram at dietary doses of 1000 to 2000 mg/kg/day over 32 days showed no clinical signs of toxicity nor changes in blood chemistry, but females did show decreased body weight and increased liver weights [6,8]. Liver effects were also seen in rats at very high doses of 3000 mg/kg/day over an exposure period of 90 days, and above 225 mg/kg/day for 90 days [58]. Dogs, sheep, and beef cattle fed low levels of picloram for a month experienced no toxic effects. The ester and triisopropanolamine salt showed low toxicity in animal tests [58]. Picloram may show additive effects if

mixed with other herbicides such as 2,4-D [118].

- **Reproductive effects:** In multi-generational studies, pregnant rats exposed during critical periods of gestation to doses of about 180 mg/kg/day of picloram showed no changes in fertility [58]. The fertility of pregnant mice fed 15 mg/kg/day for 4 days before and 14 days after mating was not adversely affected [8]. Other studies showed no effects on fertility or fecundity in rats at doses as high as 1000 mg/kg/day [58]. Picloram does not appear to cause reproductive toxicity.
- **Teratogenic effects:** No teratogenic effects were seen in the offspring of pregnant rats exposed during gestation to 400 mg/kg/day of the acid or potassium salt, or to 1000 mg/kg/day of the ester or other salt [58]. At 2000 mg/kg/day, maternal toxicity was noted but did not induce malformation in the pups [8]. It appears that picloram is not teratogenic.
- **Mutagenic effects:** One test has shown that picloram is mutagenic (to the bacterium Saccharomyces cerevisiae) and another test has shown that it is not mutagenic (Ames test) [118]. In tests for unscheduled DNA synthesis and structural chromosome aberrations, the results were also negative [58]. These data suggest that picloram is either nonmutagenic or weakly mutagenic.
- **Carcinogenic effects:** Mice fed average doses of 18 mg/kg/day or 30 mg/kg/day for 80 weeks and observed for another 10 weeks did not display any carcinogenic effects [8,118]. Male rats fed 17.5 or about 40 mg/kg/day for 80 weeks and observed for 33 weeks showed no carcinogenicity, but females developed benign liver tumor nodules [58]. Other tests have indicated an increased incidence of cancer among animals treated with picloram, but these data are difficult to interpret due to possible interference of hexachlorobenzene contaminants [8,118]. These data suggest that picloram is noncarcinogenic or weakly carcinogenic.
- **Organ toxicity:** Animal studies show the target organs for picloram to be the liver and kidneys.
- **Fate in humans and animals:** Picoloram was rapidly absorbed through the gastrointestinal tract in studies using human volunteers, and was excreted unchanged in the urine [119]. Half of the product was excreted within a day or so. Skin absorption is minimal [119]. Rats showed similar results, with administered doses excreted virtually unchanged in urine and feces within 48 hours [119]. Picloram does not accumulate in fat [119]. No measurable residues were found in milk from cows fed small amounts of the herbicide in their diets [8]. At higher levels of exposure, milk levels of picloram were low (0.05 to 0.29 ppm) and declined rapidly upon withdrawal of picloram from the diet [8].

Ecological Effects:

- **Effects on birds:** Picloram is slightly to practically nontoxic to birds; the acute oral LD50 is greater than 2000 to 5000 mg/kg in ducks, pheasants and quail, with no mortality seen at even the highest levels [6].
- **Effects on aquatic organisms:** Picloram is slightly to moderately toxic to fish and aquatic invertebrates. The reported 96-hour LC50 values for picloram are 19.3 mg/L in rainbow trout, 14.5 mg/L in bluegill sunfish, and 55 mg/L in fathead minnow [58]. The 48-hour LC50 in Daphnia is 50 mg/L, indicating moderate toxicity [58]. Most salts are of similar or lesser toxicity, but the isooctyl ester may be highly toxic. The reported 96-hour LC50 for the isooctyl ester in rainbow trout is 4 mg/L, and in channel catfish is 1.4 mg/L [118]. Other LC50 values in aquatic invertebrates ranged from 10 to 68 mg/L [8]. Picloram is not expected to accumulate appreciably in aquatic organisms; the measured bioconcentration factor in bluegill sunfish was less than 0.54 [9].
- **Effects on other organisms:** The compound is nontoxic to bees [1].

Environmental Fate:

- **Breakdown in soil and groundwater:** Picloram is moderately to highly persistent in the soil environment, with reported field half-lives from 20 to 300 days and an estimated average of 90 days [11]. Photodegradation is significant only on the soil surface and volatilization is practically nil [58]. Degradation by microorganisms is mainly aerobic, and dependent upon application rates [58]. Increasing soil organic matter increases the sorption of picloram and increases the soil residence time [58]. Picloram is poorly bound to soils, although it is bound better by soils with higher proportions of soil organic matter [11]. It is soluble in water, and therefore may be mobile [1]. These properties, combined with its persistence, mean it may pose a risk of groundwater contamination. Picloram has been detected in the groundwater of eleven states at concentrations ranging from 0.01 ug/L to 49 ug/L [9].
- **Breakdown in water:** In laboratory studies, sunlight readily broke down picloram in water, with a half-life of 2.6 days [9,58]. Herbicide levels in farm ponds were 1 mg/L directly following spraying, and decreased to 0.01 mg/L within 100 days, primarily due to dilution and the action of sunlight [6].
- **Breakdown in vegetation:** Picloram is readily absorbed by plant roots, less so by the foliage, and is readily translocated throughout plants. It remains stable and intact in plants [58].

Physical Properties:

- **Appearance:** Picloram is a colorless crystal [1].
- **Chemical Name:** 4-amino-3,5,6-trichloropyridine-2-carboxylic acid [1]
- **CAS Number:** 1918-02-1

- **Molecular Weight:** 241.48
- **Water Solubility:** 430 mg/L @ 25 C [1]
- **Solubility in Other Solvents:** v.s. in acetone, ethanol, benzene, and dichloromethane [1]
- **Melting Point:** Decomposes @ 215 C [1]
- **Vapor Pressure:** 0.082 mPa @ 35 C [1]
- **Partition Coefficient:** 0.1461 [58]
- **Adsorption Coefficient:** 16 [11]

Exposure Guidelines:

- **ADI:** Not Available
- **MCL**: Not Available
- **RfD:** 0.07 mg/kg/day [13]
- **PEL:** 5 mg/m3 (8-hour) (respirable fraction) [14]
- **HA**: 0.5 mg/L (lifetime) [120]
- **TLV**: Not Available

Basic Manufacturer:

DowElanco
9330 Zionsville Road
Indianapolis, IN 46268-1054

- **Phone:** 317-337-7344
- **Emergency:** 800-258-3033

References:

References for the information in this PIP can be found in Reference List Number 10

DISCLAIMER: The information in this profile does not in any way replace or supersede the information on the pesticide product labeling or other regulatory requirements. Please refer to the pesticide product labeling.

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Revised June 1996

Simazine

Trade and Other Names: Trade names include Aquazine, Caliber, Cekusan, Cekusima, Framed, Gesatop, Primatol S, Princep, Simadex, Simanex, Sim-Trol, Tanzine and Totazine. This compound may also be found in formulations with other herbicides such as amitrole, paraquat dichloride, metolachlor, and atrazine.

Regulatory Status: Simazine is a General Use Pesticide (GUP). It is in EPA toxicity class IV - practically nontoxic. Products containing simazine bear the Signal Word CAUTION. In November 1994, the U.S. EPA began a Special Review of simazine which could result in use restrictions or even cancellation if data warrant such action.

Chemical Class: Triazine

Introduction: Simazine is a selective triazine herbicide. It is used to control broad-leaved weeds and annual grasses in field, berry fruit, nuts, vegetable and ornamental crops, turfgrass, orchards, and vineyards. At higher rates, it is used for nonselective weed control in industrial areas. Before 1992, simazine was used to control submerged weeds and algae in large aquariums, farm ponds, fish hatcheries, swimming pools, ornamental ponds, and cooling towers. Simazine is available in wettable powder, water dispersible granule, liquid, and granular formulations. It may be soil-applied.

Formulation: Simazine is available in wettable powder, water dispersible granule, liquid and granular formulations. It may be soil-applied.

Toxicological Effects:

- **Acute toxicity:** Simazine is slightly to practically nontoxic. The reported oral LD50 for technical simazine in rats and mice is >5000 mg/kg [6,15]; its dermal LD50 is 3100 mg/kg in rats and > 10,000 mg/kg in rabbits [6,15]. The 4-hour inhalation LC50 in rats is greater than 2 mg/L (6). The formulated products, in most cases, are less toxic via all routes [15]. Simazine is nonirritating to the skin and eyes of rabbits except at high doses [3]. Patch tests on humans have shown that simazine is not a skin irritant, fatiguing agent, or sensitizer [3]. However, rashes and dermatitis from occupational exposure to simazine have occurred [3]. The triazine herbicides disturb energy metabolism (thiamin and riboflavin functions). Symptoms include difficulty in walking, tremor, convulsions, paralysis, cyanosis, slowed respiration, miosis (pinpoint pupils), gut pain, diarrhea, and impaired adrenal function [3]. No cases of poisoning in humans have been reported from ingestion of simazine [3]. Rats given an oral dose of 5000 mg/kg exhibited drowsiness and irregular breathing. In another study, a single oral dose of 4200 mg/kg produced anorexia, weight loss, and some deaths in rats within 4 to 10 days [26]. For unknown reasons, sheep and cattle are especially susceptible to poisoning by simazine. Doses of 500 mg/kg were fatal in sheep with death delayed for 5 to 16 days. Symptoms exhibited by poisoned sheep included lower food intake, higher water intake, incoordination, tremors, and weakness, especially in the hindquarters [3].

- **Chronic toxicity:** Some 90-day feeding studies showed reduced body weight at 67 to 100 mg/kg/day [10]. This same effect and kidney toxicity were seen in rats at doses of 150 mg/kg/day [10]. In 2-year chronic oral feeding studies in which rats were given daily dosages of 5 mg/kg/day of simazine in the diet, no gross or microscopic signs of toxicity were seen [3]. When rats were given repeated doses of 15 mg/kg/day, some liver cells degenerated during the first 3 days, but the condition did not progress. Instead, the liver adapted and the compound was metabolized [3]. Other effects observed in test animals include tremors, damage to the testes, kidneys, liver, and thyroid, disturbances in sperm production, and gene mutations [10].
- **Reproductive effects:** No adverse effects on reproductive capacity or development were observed in a three-generation study of rats fed 5 mg/kg/day simazine [10]. High rates of fetotoxicity and decreased birth weight were noted in the fetuses of pregnant rabbits fed 75 mg/kg/day [26]. Reproductive effects are not likely in humans under normal circumstances.
- **Teratogenic effects:** No dose-related teratogenic effects were observed when rabbits were given daily doses of 5, 75, or 200 mg/kg for days 7 through 19 of pregnancy [26]. Chronic inhalation of a cumulative dose of 0.3 mg/L for 8 days in pregnant rats resulted in no treatment-related developmental abnormalities [10]. Simazine does not appear to be teratogenic.
- **Mutagenic effects:** Simazine has shown negative results in a variety of mutagenicity tests on bacterial cultures [10]. Tests on human lung cell cultures have produced both positive and negative results [10]. When injected into adult male fruitflies, simazine increased the frequency of sex-linked lethal mutations, but failed to do so when fed to larvae. Other tests for mutagenicity in fruitflies were negative [3]. It is likely that simazine is either nonmutagenic or weakly mutagenic.
- **Carcinogenic effects:** Simazine was not tumorigenic in mice at the maximum tolerated dose of 215 mg/kg/day over an 18-month period [10]. In other studies, doses as low as 5 mg/kg/day produced excess tumors (thyroid and mammary) in female rats [3,10]. Because of inconsistencies in the data, it is not possible to determine simazine's carcinogenic status.
- **Organ toxicity:** Damage to the testes, kidneys, liver, and thyroid has been observed in test animals [3,10].

- **Fate in humans and animals:** Studies in rats, goats, and sheep reveal that 60 to 70% of the ingested dose may be absorbed into the system [10], with approximately 5 to 10% distributed systemically to tissues. The remainder is eliminated via urine within 24 hours [6]. Distribution led to detectable levels in red blood cells (highest), liver, kidney, fat, bone, and plasma [10]. When a cow was fed 5 ppm for 3 days, no simazine was found in the cow's milk during the next 3 days. It has been reported that simazine residues were present in the urine of sheep for up to 12 days after administration of a single oral dose. The maximum concentration in the urine occurred from 2 to 6 days after administration [16].

Ecological Effects:

- **Effects on birds:** Simazine is practically nontoxic to birds [6,16]. The reported LD50 values in mallard and Japanese quail are >4600 mg/kg and 1785 mg/kg, respectively [6]. The acute dietary LD50 values in hens and pigeons are both greater than 5000 ppm [2]. The 8-day dietary LC50 in bobwhite quail is >5260 ppm and in mallard ducks is >10,000 ppm [6,15].
- **Effects on aquatic organisms:** Simazine is slightly to practically nontoxic to aquatic species [6,15]. The 96-hour LC50 for simazine is >100 mg/L [46] in rainbow trout, 100 mg/L (wettable powder) in bluegill sunfish, 0.100 mg/L in fathead minnows [46], as well as carp [2]. It may be more toxic to Daphia and stoneflies [46]. A 96-hour LC50 of >3.7 mg/L is reported in oysters [15].
- **Effects on other organisms:** While many mammals may be insensitive to simazine [16], sheep and cattle are especially sensitive [3]. Simazine is nontoxic to bees [6,16]. A soil LC50 in earthworms of >1000 mg/kg has been reported [16].

Environmental Fate:

- **Breakdown in soil and groundwater:** Simazine is moderately persistent with an average field half-life of 60 days [20]. Soil half-lives of 28-149 days have been reported [20]. Residual activity may remain for a year after application (2 to 4 kg/ha) in high pH soils. Simazine is moderately to poorly bound to soils [20]. It does, however, adsorb to clays and mucks. Its low water solubility, however, makes it less mobile, limiting its leaching potential [15]. Simazine has little, if any, lateral movement in soil, but can be washed along with soil particles in runoff. Simazine is subject to decomposition by ultraviolet radiation, but this effect is small under normal field conditions. Loss from volatilization is also insignificant. In soils, microbial activity probably accounts for decomposition of a significant amount of simazine in high pH soils. In lower pH soils, hydrolysis will occur [15]. Simazine residues have been detected in groundwater in at least 16 states. The range was from 0.00002 mg/L to 0.0034 mg/L [23].
- **Breakdown in water:** The average half-life of simazine in ponds where it has been applied is 30 days, with the actual half-life dependent on the level of algae present, the degree of weed infestation, and other factors [15]. Simazine may undergo hydrolysis at lower pH. It does not readily undergo hydrolysis in water at pH = 7 [15].
- **Breakdown in vegetation:** Plants absorb simazine mainly through the roots, with little or no foliar penetration. From the roots, it is translocated upward to the stems, leaves, and growing shoots of the plant [6,15]. It acts to inhibit photosynthesis [6,15]. Resistant plants readily metabolize simazine. Plants that are sensitive to simazine accumulate it unchanged [6]. It is possible that livestock or wildlife grazing on these plants could be poisoned.

Physical Properties:

- **Appearance:** Simazine is a white or colorless crystalline solid [6].
- **Chemical Name:** 6-chloro-N2,N4-diethyl-1,3,5-triazine-2,4-diamine [6]
- **CAS Number:** 122-34-9
- **Molecular Weight:** 201.70
- **Water Solubility:** 5 mg/L @ 20 C [6]
- **Solubility in Other Solvents:** s. in methanol, chloroform, and diethyl ether [6]; s.s. in pentane [6]
- **Melting Point:** 225-227 C [6]
- **Vapor Pressure:** 0.000810 mPa @ 20 C [6]
- **Partition Coefficient:** 1.9600 [6]
- **Adsorption Coefficient:** 130 [20]

Exposure Guidelines:

- **ADI:** Not Available
- **MCL:** 0.004 mg/L [25]
- **RfD:** 0.005 mg/kg/day [26]
- **PEL:** Not Available
- **HA:** Not Available
- **TLV:** Not Available

Basic Manufacturer:

Ciba-Geigy Corporation
P.O. Box 18300
Greensboro, NC 27419-8300

- **Phone:** 800-334-9481
- **Emergency:** 800-888-8372

References:

References for the information in this PIP can be found in Reference List Number 8

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