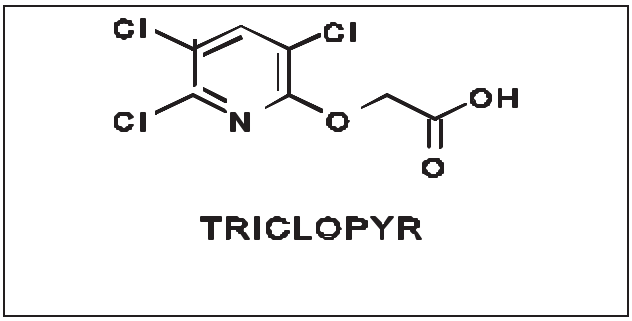
III.8 TRICLOPYR



SUMMARY

Triclopyr [(3,5,6-trichloro-2-pyridynyl) oxy] acetic acid is a synthetic herbicide that is used to control a wide variety of woody plants as a foliar spray or as a basal spray when applied to cut surfaces. There are three formulations of triclopyr commonly used to control nuisance vegetation: triclopyr acid (CASRN 55335063 (CASRN 057213691), the triethylamine salt (TEA) (CASRN 057213691); and the butoxyethyl ester of triclopyr (TBEE) (CASRN 008008206). Several authors have evaluated the use of triclopyr for control of Eurasian watermilfoil (Getsinger and Westerdahl, 1984; Green *et al.*, 1989; Netherland and Getsinger, 1992), but currently there are no formulations of triclopyr that are registered for aquatic uses in Massachusetts.

The U.S. EPA approved a Reregistration Eligibility Decision (RED) on September 30, 1997 for triclopyr which includes the triclopyr acid, as well as the TEA and TBEE formulations.

Photodegradation is the major pathway for the transformation of triclopyr in aquatic systems. The triethylamine salt and the butoxyethyl ester formulations are rapidly converted to the acid form in water (McCall and Gavit, 1986 and Solomon *et al.*, 1988; as cited in DFA/DEP Adhoc Committee, 1990).

REGISTERED PRODUCTS IN MASSACHUSETTS

As of the printing of this GEIR, there are currently no formulations of triclopyr registered for aquatic use in Massachusetts. However, this chemical is currently undergoing review by the Massachusetts Division of Agricultural Resources (DAR). The review of the environmental fate information has been completed and the DAR is still waiting to receive the results of many of the recent toxicity studies (Kennedy, pers. comm., 2004). The current list of aquatic herbicides that are registered in Massachusetts can be accessed at http://www.state.ma.us/dfa/pesticides/water/aquatic/ aquatic/profile.htm on the DAR Aquatic Pesticide Website. The DAR updates this list regularly with changes. The status of triclopyr can be followed by consulting this website. In addition, the DAR can be contacted directly at (617) 626-1700 for more specific questions regarding updates for this product.

TRICLOPYR USES AND APPLICATION

Triclopyr and its formulations, TBEE and the TEA salt, are often used to control unwanted woody plants and annual and perennial broadleaf weeds in rangeland, permanent pastures, forests and on non-crop areas including rights of way such as electric power lines, communication lines, pipelines, roadsides and railroads. There are also formulations of the TEA salt being marketed for aquatic weed control. The salt formulation is used to control nuisance aquatic vegetation.

For specific information on recommended application rates for a particular product, the product label should be consulted. The USEPA Office of Pesticide Programs (OPP) has a link to a database of product pesticide labels at http://www.epa.gov/pesticides/pestlabels/. Manufacturers' product labels should also be consulted for recommended product application rates and use restrictions.

MECHANISM OF ACTION

Triclopyr is an auxin type herbicide that is absorbed by the roots and leaves, is translocated through the plant and accumulates in the meristematic tissues of the root and shoot. The auxin-type response in plants associated with triclopyr interferes with normal growth processes, therefore, maximal effect occurs when applications are made soon after leaves are fully developed and when there is sufficient moisture for plant growth.

ENVIRONMENTAL FATE AND TRANSPORT

The triclopyr acid is short-lived in the aquatic environment with reported half-lives from 2.1 hours at the water's surface in the summer at 40° N latitude to 14 hr at 1 meter water depth in winter (McCall and Gavitt, 1986). In a study by Dow Elanco, half-lives for triclopyr were determined at 20, 30, 40 and 50° N latitude in summer and in winter (Batzer, 1994). In summer, from 20 to 50° N latitude, triclopyr had calculated half-lives of less than 1.2 days in both pH 7 buffered and natural waters.

Triclopyr acid is stable to hydrolysis in buffered solutions for periods of up to 9 months at pH 5, 7 and 8 at 15, 25 and 35°C (Hamaker, 1977). Therefore, hydrolysis is not expected to be a major route of degradation. Photolysis is the major degradation pathway for triclopyr acid in water. The rate of triclopyr photodegradation in water is rapid in both natural sunlight and in the laboratory. A photolysis half-life of 10 hours is reported by Hamaker (1977). In winter, the calculated photolysis half-life of triclopyr varied in latitude from 0.54 to 10 days in pH 7 buffered solutions and from 1.5 to 29 days in natural water. A half-life of 142 days has been reported for the metabolism of triclopyr conducted under dark conditions in a 30 day aquatic metabolism study (Woodburn and Cranor, 1987).

The principle decay product of the acid is the 3,5,6-trichloro-2-pyrindol (TCP), a transient metabolite in water with half-lives ranging from minutes to one day (Dilling *et al.*, 1984). Woodburn demonstrated that the triethylamine salt of triclopyr experimentally applied to a lake in Florida also provides useful comparative data on the persistence of triclopyr degradation products. TCP rapidly degrades into non-halogenated, low molecular weight organic acids (Woodburn, 1993), with phototransformation playing a larger role than hydrolysis in this process.

The fate of the butoxyethyl ester of triclopyr (TBEE) in water is summarized in Table III.8-1. This table shows the major degradation pathways for the ester in water but does not include the processes such as sediment and particulate adsorption. The fate of the ester in water has also been simulated with a modeling technique by McCall *et al.* (1988). The degradation pathway is believed to be TBEE to triclopyr acid to 3,5,6-trichloro-2-pyrindol (TCP) to non-halogenated organic acids.

TBEE degrades quite rapidly in water to triclopyr acid. Laboratory studies indicate that photolysis is the principal degradation pathway, with hydrolysis also contributing. Several studies indicate that the half-life of the ester in water can range from 1.5 to 6.6 days as a result of photolysis (McCall and Gavit, 1986; Solomon *et al.*, 1988; Havens and Shepler, 1993). Hydrolysis half-lives are dependent upon pH and temperature and range from 0.06 days to 208 days in natural waters. They decrease with increasing temperature and increasing pH. Acidic conditions increase the persistence of the ester substantially. The 208 day half-life was observed in natural waters at pH 5 at 15°C. Waters with this pH level occur in Massachusetts. One laboratory study produced contradictory results where the ester was stable to hydrolysis and little photodegradation of the ester occurred over 9 months (Hameker, 1977). This study

was performed with buffered, sterile water. Modeling results for the dissipation of the ester indicate that the decay should be fairly rapid with a half-life of 12 to 18 hours (McCall *et al.*, 1988).

A half-life of 3.8 to 4.3 days at 16 to 17°C was calculated for the degradation of the ester to TCP in an Ontario Lake (Solomon *et al.*, 1988). Woodburn (1993) added Triclopyr salt to a Florida lake and determined a half-life of 0.5 to 3.6 days at 30°C for the breakdown of the ester (or salt) to organic acids. With the exceptions of the Hameker (1977) study and slow breakdown at pH 5, most studies indicate that TBEE in water is degraded relatively rapidly.

Tables III.8-3a, III.8-3b and III.8-3c at the end of this triclopyr summary list the physical and chemical properties for the parent compound, triclopyr, the triethylamine salt and the butoxyethyl ester. The data indicate that triclopyr is relatively mobile and is not expected to bioaccumulate in aquatic organisms.

Table III.8-1. Aquatic Fate of Triclopyr

Pathway	Finding	Authors
Hydrolysis	insignificant route of degradation	Cleveland and Holbrook, 1991
Photodegradation	most significant route of degradation	Woodburn *et al*., 1990
Aerobic Aquatic Degradation	slow metabolism, half-life est. @ 4.7 months under dark conditions	Woodburn and Cranor, 1987
Anaerobic Aquatic Degradation	slow degradation, est. 3.5 years	Laskowski and Bidlack, 1984

Tables III.8-3a, III.8-3b and III.8-3c at the end of this triclopyr summary list the physical and chemical properties for the parent compound, triclopyr, the triethylamine salt and the butoxyethyl ester. The data indicate that triclopyr is relatively mobile and is not expected to bioaccumulate in aquatic organisms.

PHARMACOKINETICS

Both the triclopyr acid and the TBEE salt are readily absorbed from the gastrointestinal tract (Dryzga *et al.*, 1994). This same study indicated that both compounds are distributed similarly, have a similar plasma clearance (0.92 and 0.95 ml/min-kg^{-1}) and are both eliminated in 4.5 hours. Triclopyr is excreted primarily in the urine (Dow Elanco, 1992).

Triclopyr acid and triclopyr-BEE have been shown to be bioequivalent following a single low oral dose and a single high oral dose in rats (Dryzga *et al.*, 1994). There were no differences in plasma levels or pharmacokinetics between the triclopyr free acid and the TBEE under the conditions of the testing regime. Results of the high dose administration demonstrate that the tissue distribution was similar for both forms of triclopyr. The USEPA considers the triclopyr acid, the triclopyr butoxyethyl ester and the triclopyr triethylamine salt as bioequivalent (McMaster, 1995).

HEALTH EFFECTS

Avian:

The toxic effects of triclopyr on birds have been investigated in studies conducted by Dow Elanco. For mallard ducks, acute oral LD50 values are reported at 1648 mg/kg for unformulated triclopyr, 3176 mg/kg for Garlon 3A, and 4640 mg/kg for Garlon 4 (Pesticide Background Statement, USEPA, 1984). Eight day subchronic oral LC50 values are reported in Table III.8-2 below for the various triclopyr formulations:

Table III.8-2. Eight-day LC50 Values

Triclopyr	mallard duck bobwhite quail Japanese quail	LC50 = 5,000 ppm[1] LC50 = 2,935 ppm[1] LC50 = 3,278 ppm[1]
a formulation of the triethylamine salt (TEA)	mallard duck bobwhite quail	LC50 = 10,000 ppm[2] LC50 = 11,622 ppm[2]
a formulation of the butoxyethyl ester (TBEE)	mallard duck bobwhite quail bobwhite quail bobwhite quail	LC50 = 10,000 ppm[2] LC50 = 9,026 ppm[2] LD50 = 735 mg/kg[3] LC50 > 5401 ppm[4]

(1) WSSA, 1983
(2) Mc Call *et al.*,1988
(3) Campbell and Lynn, 1991
(4) Lynn *et al.*, 1991

The data summarized above indicate low acute and subchronic toxicity to bird species tested. No field studies on the toxic effects of triclopyr or its formulations in birds were available.

Mammalian

Acute:

The oral LD50 for triclopyr in rats is 729 mg/kg in males and 630 mg/kg in females (Pesticide Background Statements, 1984; Dow tech. data sheet). The rat LD50 for combined sexes has been reported as 713 mg/kg (WSSA, 1983; GEIR, 1985). Rabbits and guinea pigs are more sensitive via oral administration with LD50 of 550 and 310 mg/kg respectively. Both the TEA and TBEE formulations have oral LD50s of greater than 2,000 mg/kg.

The dermal LD50s are greater than 2,000 mg/kg in rabbits and greater than 3980 mg/kg in rabbits for the TBEE and TEA formulations respectively (Dow tech. data sheet; Dow MSDS for Garlon 3A; Dow MSDS for Garlon 4).

Triclopyr affects the eyes of rabbits and these effects are dependent on the chemical derivative involved: the TBEE formulation is essentially nonirritating (GEIR, 1985; Pesticide Background Statements, 1984; Dow MSDS for Garlon 4). The TEA formulation is not only an irritant but can cause serious injury (GEIR, 1985; Dow MSDS for Garlon 3A). These eye injuries include conjunctival irritation, moderate internal redness and moderate to severe corneal damage which may be permanent.

An inhalation study showed that 100% of the test rats survived a 1 hour exposure to 3 to 20 dilutions of the TEA formulation in air. Transitory nasal irritation to rats was noted after a 4 hour exposure to an aerosol formulation of the TBEE. (GEIR, 1985)

Subchronic/Chronic:

In subchronic studies, the 90 day dietary No Observed Effect Levels (NOELs) were 30 mg/kg/d and 20 mg/kg/d for rats and mice respectively. Dogs were more sensitive to dietary administration of triclopyr, demonstrating a decreased urinary excretion at 2.5 mg/kg/d (GEIR, 1985; Dow Tox profile for Garlon). In a one year study, dogs received doses of 0, 0.5, 2.5 or 5 mg/kg/d. Minimal kidney effects were observed at doses of 2.5 and 5.0 mg/kg/d. These were considered non-adverse effects by Dow making the No Observed Adverse Effect Level (NOAEL) 5.0 mg/kg/d and the NOEL 0.5 mg/kg/d (Quast., 1988).

Two studies in monkeys were conducted to investigate kidney effects in primates. In one of the studies the monkeys received 0, 10, 20 or 30 mg/kg/d in their diets for 28 days; no effects were reported (DOW, nd). In a second study, 4 monkeys received triclopyr at 5 mg/kg/d for 28 days. The dose was then increased to 20 mg/kg/d for 102 days (DFA/DEP Adhoc Comm., 1990). The effects reported from this study were stool softening and diarrhea.

Long-term bioasssays have been done with triclopyr in rats (Eisenbrandt *et al.*, 1987) and mice (Tsuda., 1987). Fischer 344 rats received 5, 20, 50 or 250 mg/kg/d in a preliminary 13 week study. There was a decrease in body weight gain at 50 and 250 mg/kg/d and kidney effects were seen in both sexes at doses of 20 mg/kg or greater. In the full two year study, the doses were 0, 3, 12 and 36 mg/kg/d. The dose related effects included increased body weight at 12 and 36 mg/kg/d in the males and increased pigmentation of the proximal tubules at 3, 12 and 36 mg/kg/d in females. Neither the weight increase in males or the hyperpigmentation in females was accompanied by morphological, histological or functional changes. The NOAEL for males and females was reported to be 3 mg/kg/d (Eisenbrandt *et al.*, 1987).

In the mouse bioassays, ICR-mice received triclopyr in their diets for 22 months. The doses were 0, 50, 250 and 1250 ppm (0, 5, 55, 28.6 and 143 mg/kg/d in males and 0, 5.09, 26.5 and 135 mg/kg/d in females). The range finding study included doses of 0, 200, 400, 800, 1600 or 3200 ppm. At the high dose there were decreases in body weight, anemia, changes in urine, increased cholesterol levels and multiple changes in liver function. Some of the liver changes were also noted at the 1600 and 800 ppm levels. There were decreases in body weights, changes in the kidney and urine and liver effects at the 1250 ppm dose. At 250 ppm there were mild kidney effects and the NOEL was reported as 10 ppm (5.55 mg/kg/d in males and 5.09 mg/kg/d for females) (Tsuda., 1987.)

Developmental/Reproductive:

The teratology of triclopyr was investigated using the rabbit as a model of human exposure. Doses in the range finding study were 0, 25, 50, 100 and 200 mg/kg. There was 50% and 71% mortality in the 100 and 200 mg/kg group respectively. The doses used in the full study were 0, 10, 25 and 75 mg/kg/d for days 6 to 18 of gestation. There were 16 rabbits per dose group. One dam in the 25 mg/kg/d dose group aborted and one dam in the 75 mg/kg/d dose group died. In the 25 mg/kg/d dose group, one fetus had hyperplasia of the aortic arch with pulmonary arterial semilunar valve stenosis. Another fetus had a missing gallbladder. There was a statistically significant but non-dose related increase in resorptions at 10 mg/kg/d. This increase is within historical control variability. The development of the NOEL was reported at 75 mg/kg/d with a slight increase in maternal mortality (WSSA, 1983).

Mutagenicity:

Triclopyr has been tested for mutagenicity in a variety of test systems and found to be weakly positive in one, the dominant lethal study in rats. Triclopyr was non-mutagenic in bacterial system assays, cytogenic assays and mouse dominant lethal studies (Pesticide Background Statement, USEPA, 1984).

Carcinogenicity:

There have been two chronic bioassays done for triclopyr. Rats received 0, 3, 12 or 36 mg/kg/d and mice received 0, 50, 250 or 1250 ppm (5.55, 28.6, 143 mg/kg/d for males and 5.09, 26.5, and 135 mg/kg/d for females). The only positive result was an increase in the combined incidence of mammary adenomas and adenocarcinomas in the female rats at the high dose. There was no evidence of multiple tumors and the effects were not dose related (Tsuda., 1987; Eisenbrandt *et al.*, 1987). The most recent information available at the time of publication of this GEIR indicates that the U.S.EPA Office of Pesticide Programs (OPP) has designated triclopyr as a Group D carcinogen. Under the new U.S.EPA cancer classification system using descriptors, a Group D carcinogen corresponds to the descriptor, "Data are inadequate for an assessment of human carcinogenic potential".

Available Toxicity Criteria:

The Environmental Protection Agency (EPA) Office of Pesticide Programs (OPP) has developed an oral Reference Dose (RfD) of 0.005 mg/kg/d for triclopyr based on a one-year feeding study in beagle dogs (Federal Register, 1995; USEPA, 1995).

ECOLOGICAL TOXICITY

Invertebrates:

Little data are available on the toxicity of triclopyr acid to invertebrates and microorganisms. Data are available for the TEA and triclopyr BEE formulations. Data for TEA indicate low acute lethal toxicity to organisms tested, with a 96h LC50 of between 326 and 895 ppm in grass shrimp (Ward *et al.*, 1992; DFA/DEP Adhoc Comm., 1990), 96h LC50 greater than 1,000 ppm in crabs and 48h LC50 ranging between 58 and 87 ppm in oysters (Boeri, 1993). The 48h LC50 in *Daphnia* is reported as 1,170 ppm (Pesticide Background Statement, 1984). After 72h of incubation with 500 ppm of triclopyr, no apparent effects on growth were observed in six soil microorganisms when compared with controls.

In crayfish (*Procambarus clarki*), a 96h LC50 of greater than 326 mg/l was determined for the TEA (Barron *et al.*,1989). Exposure of grass shrimp (*Palaemonetes pugio*) resulted in a 96 hour LC50 of 326 mg/l (Ward *et al.*, 1992). A NOEL of 132 mg/l was derived from the same study.

The BEE of triclopyr was tested under flow-through conditions for acute toxicity to grass shrimp (*Palaemonetes pugio*) and a 96-hour LC50 of 2.4 mg/l was determined (Ward and Boeri, 1991a). In oysters (*Crassostrea virginica*), a 96-h EC50 of 0.66 mg/l was reported for the triclopyr-BEE (Ward and Boeri, 1991b). Tidewater silversides that were exposed to the BEE of triclopyr in a 96-hour assay showed an LC50 value of 0.45 mg/l (Ward and Boeri, 1991c).

Vertebrates:

Triclopyr Acid:

The available information on triclopyr toxicity to fish indicate a wide response of fish to two formulations of triclopyr and to unformulated triclopyr. In fish, 96-hour LC50 values of 117 ppm and 148 ppm have been reported in rainbow trout and bluegill sunfish, respectively (WSSA, 1983).

Triethylamine salt:

The TEA salt is "slightly toxic" to fish with 96h LC50 values of 552 and 891 ppm for rainbow trout and bluegill sunfish respectively. The corresponding values for the unformulated triclopyr are 117 ppm for rainbow trout and 148 for bluegill sunfish. Both species were less sensitive to the TEA salt than to the active ingredient (DFA/DEP Adhoc Committee, 1990).

Butoxyethyl ester:

The BEE of triclopyr is "highly toxic to fish" with the 96h LC50 values for rainbow trout and bluegill sunfish of 0.74 and 0.87 ppm respectively (McCall *et al.*, 1988). The corresponding value for juvenile Coho salmon is 1.3 ppm (Mayes *et al.*, 1986). In 1993, Woodburn *et al*. reported LC50 values in blue gill sunfish (*Lepomis macrochirus*) of 0.63 mg/l (24-h), 0.44 mg/l(48-h), 0.40 mg/l (72-h) and 0.36 mg/l (96-h). Based on the categorization scheme used by USEPA, triclopyr-BEE is "highly toxic" to bluegill sunfish. No fish toxicity data are available for the 3,5,6-trichloro-2-pyrindol (TCP), the intermediate breakdown product of the triclopyr acid to the non-halogenated organic acid end product.

The persistence data described earlier and the simulation results of McCall et al (1988), provide a description of the probable fate of triclopyr in toxicity tank tests. The majority of the fish mortalities during the toxicity tests with bluegill sunfish and rainbow trout exposed to the ester occurred during the first 24 hours of the test, a pattern consistent with the change of the toxic ester form to a less toxic breakdown product during this period (McCarty *et al.* n.d.).

Plants:

The triclopyr-BEE has been tested for toxicity to non-target vegetation. Milazzo *et al*. (1993), reported an EC50 (i.e., a concentration at which 50% of the test organisms manifest effects in cell growth) of 2.2-3.7 mg/l in duckweed (*Lemna gibba*), 0.193 mg/l in the diatom *Navicula pelliculosa* (Hughes and Alexander, 1993) and an EC50 of 0.193 mg/l in the blue-green alga *Anabaena flos-aquae.*

Table III.8-3a. Properties of Triclopyr Acid

Molecular formula	$C_7H_4Cl_3NO_3$
Molecular weight (g/mol)	256.48
Vapor pressure (mm Hg @ 20^o C)	1.26 x10^{-6}
Water solubility (g/mol @ 24.5^o C)	440
Octanol-water partition coefficient (at pH 7)	0.36
pKa[1]	2.93
K_{oc}[2]	59 ml/g
BCF (Bluegill sunfish)	0.03
BCF (Catfish)	0.04

1. Martin, E.J., 1988.
2. Woodburn *et al.*, 1988

Table III.8-3b. Properties of the Triethylamine (TEA) Salt of Triclopyr)

Molecular formula	$C_{13}H_{18}O_3N_2Cl_3$
Molecular weight	355
Vapor pressure (mm Hg)	< 1 x 10^{-8}
Water solubility (g/l)	0.412
Octanol-water Partition coefficient (at pH 7)	0.196

(WSSA, 1994)

Table III.8-3c. Properties of the Triclopyr-Butoxyethyl Ester (TBEE)

Molecular formula	$C_{13}H_{16}Cl_3NO_4$
Molecular weight (g/mol)	356.69
Vapor pressure (mm Hg @ 33^0 C)	1 x 10^{-5}
Water solubility (mg/l)	5.75
Octanol-water partition coefficient	4.1 x 10^{-4}

(WSSA, 1994)

Triclopyr References

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