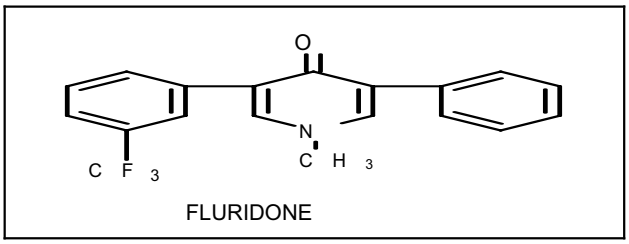
III.5 FLURIDONE



FLURIDONE

SUMMARY

Fluridone (1-methyl-3-phenyl-5-[3-(trifluoromethyl)phenyl]-4(1H)-pyridinone) is a selective systemic aquatic herbicide used to control primarily broad-leaved, submerged aquatic macrophyte species including Eurasian watermilfoil, curly-leaf pondweed as well as native pondweeds (McLaren/Hart, 1995). As opposed to a nonselective contact herbicide which will kill any plant material that it comes in contact with, Sonar is intended for a select group of target species, which are listed on the registered labels (Cockreham, pers. comm.). It is used to treat ponds, lakes, reservoirs, canals and rivers. Fluridone is stable to oxidation and hydrolysis (McCowen *et al.*, 1979 as cited in Aquatic Plant Identification and Herbicide Use Guide, 1988). Volatilization of fluridone is insignificant (Muir and Grift, 1982 as cited in Aquatic Plant Identification Guide, 1988). Breakdown of fluridone in the aquatic environment occurs mostly through photolysis. Other fate processes include plant uptake and adsorption to soil and suspended colloids (Joyce and Ramey, 1986). Some microbial degradation of fluridone has also been reported (Muir and Grift, 1982 as cited in McLaren/Hart, 1995). Fluridone is taken up in fish but studies demonstrate that fish tissue concentrations generally reflect water concentrations and that fish concentrations rapidly clear when fluridone residues are removed from the water (West *et al.*, 1983 and Muir *et al.*, 1982 as cited in McLaren/Hart, 1995). There are no restrictions on the use of fluridone to treat water used for swimming or domestic purposes. Fluridone should not be applied within one-fourth mile of any potable water intake (WSDOE, 1992). However, in making whole lake treatments at a concentration of 10-20 parts per billion for the control of Eurasian watermilfoil and curlyleaf pondweed, Sonar may be applied where functioning potable water intakes are present (Cockreham, pers. comm.).

Fluridone is manufactured by SePRO Corporation under the brand name Sonar. SePRO Corporation purchased all rights to fluridone/Sonar from DowElanco on January 1, 1994. The U.S. Environmental Protection Agency (USEPA) approved the label for Sonar on March 31, 1986 (McLaren/Hart, 1995).

REGISTERED PRODUCTS IN MASSACHUSETTS

There are two products containing Sonar which are registered for use in Massachusetts. These include Sonar A.S. (manufactured as a 41.7% liquid suspension by SePRO) and Sonar SRP (manufactured as a 5% formulation in a slow release pellet by SePRO). The DowElanco registrations in Massachusetts for Sonar A.S. and Sonar SRP were canceled in a notice to the state in June, 1995 (Corte-Real, pers. comm.;
Cockreham, pers. comm.).

FLURIDONE USES AND APPLICATION

Fluridone is used to manage aquatic vegetation in fresh water ponds, lakes, reservoirs, canals and rivers (Cockreham, pers. comm.). It is absorbed from the water by the shoots of submerged plants and from the hydrosoil by the roots of aquatic vascular plants. The effectiveness of fluridone depends on the degree to which the herbicide maintains contact with plants. Rapid water movement or any dilution of this herbicide in water will reduce its effectiveness (DowElanco, 1992; Aquatic Plant Identification and Herbicide Use Guide, 1988; WSDOE, 1992).

Application of fluridone may be made in several ways depending on the formulation used. The liquid suspension may be applied as a spray to the water surface, subsurface or along the bottom of the water body using specialized equipment. The pellet can be spread on the water surface (WSSA, 1983). Water should be used as a carrier during application of the liquid fluridone suspension. No surfactant is specified for use during application.

When treating ponds, application should be made to the entire water body. When treating lakes and reservoirs, plots no smaller than ten surface acres should be treated. In addition, areas with a large linear aspect (such as boat lanes and narrow shorelines) should not be treated (Aquatic Plant Identification and Herbicide Use Guide, 1988).

Application of fluridone may be made prior to active growth of aquatic weeds or any time during the spring or summer when weeds are visible (WSSA, 1983; Aquatic Plant Identification and Herbicide Use Guide, 1988).

The application rate of fluridone is dependent on the depth and type of water treated. In ponds averaging less than six feet in depth, a rate of 3.2-5 lbs of Sonar SRP per foot depth or 0.16-0.25 qts of Sonar A.S. per foot depth should be applied per treated surface acre. In lakes and reservoirs, 4-8 lbs of Sonar SRP per foot depth or 0.2-0.4 qts of Sonar A.S. per foot depth should be applied per treated surface acre up to a maximum of 80 lbs or 4 qts per treated acre. The label recommends that for best results in lakes and reservoirs, treatment areas should be a minimum of five acres in size. For control of Eurasian Watermilfoil in whole lake or reservoir treatments where little dilution is expected to occur, Sonar A.S. may be applied early in the growing season at a rate of 0.027-0.05 qts per foot water depth per treated surface acre. The label also specifies that treatment should not be applied within one-fourth mile of any functioning potable water intake (SePRO, 1994).

The SePRO registration label provides a list of vascular aquatic plants controlled by Sonar SRP (SePRO, 1994). This list has been supplemented with several other species which are considered susceptible to Sonar as specified in the New York state-approved labels for Sonar SRP and Sonar A.S. (McLaren/Hart, 1995). The plant selectivity of Sonar is dependent upon dose, application timing and formulation used. In general, the plants listed in Table III.5-1 are controlled by Sonar:

MECHANISM OF ACTION

Fluridone produces its toxic effect in plants by inhibiting synthesis of carotenes (pigments that protect chlorophyll molecules from photodegradation). The absence of carotenes causes degradation or "bleaching" of chlorophyll by sunlight from plants. Plants become whitish-pink or chlorotic at growing points and die slowly. This slow dying-off of plants (i.e., 30-90 days) (Cockreham, pers. comm.) reduces the instantaneous oxygen demand caused by plants dying off and decomposing all at once (Joyce and Ramey, 1986). The herbicidal effects of fluridone usually appear within 7-10 days. Species susceptibility to fluridone may vary depending on time of year, stage of growth and water movement (McLaren/Hart, 1995).

Table III.5-1. List of Aquatic Plants Controlled by Fluridone

American Lotus	*Nelumbo lutea*
Bladderwort	*Ultricularia* spp.
Common Coontail	*Ceratophyllum demersum*
Common Duckweed	*Lemna minor*
Common Elodea	*Elodea canadensis*
Egeria, Brazilian Elodea	*Egeria densa*
Fanwort	*Cabomba caroliniana*
Hydrilla	*Hydrilla verticillata*
Naiad	*Najas* spp.
Pondweed (except Illinois)	*Potamogeton* spp.
Watermilfoil (including Eurasian Watermilfoil)	*Myriophyllum* spp. (including *M. spicatum)*
Spatterdock	*Nuphar luteum*
Waterlily	*Nymphaea* spp.
Waterprimrose (including Waterpurlane)	*Ludwigia* spp. (including *Ludwigia palustris*)
Watershield	*Brasenia schreberi*

(McLaren/Hart, 1995; SePRO, 1994)

ENVIRONMENTAL FATE/TRANSPORT

The major fate process affecting fluridone persistence in aqueous environments is photolysis. Thus any factors which affect sunlight intensity and/or penetration of light into the water column will affect the dissipation rate of fluridone (Joyce and Ramey, 1986). Other factors affecting dissipation include geographic location, date of application, water depth, turbidity, weather and weed cover (West *et al.*, 1983 as cited in McLaren/Hart, 1995). Microbial degradation is also reported to occur in laboratories, but photolysis generally occurs much more quickly (Muir and Grift, 1982 as cited in McLaren/Hart, 1995). Other secondary fate processes include adsorption to soil and suspended colloids and plant uptake (Joyce and Ramey, 1983).

Fluridone will adhere to sediment particles/organics in the sediment. Eventually, the fluridone will desorb and photodegrade into the water column from the hydrosoil (Elanco, 1981 as cited in McLaren/Hart, 1995). The pH of the water can affect this rate (with the lower the pH, the higher the adsorption rate (Malik and Drennan, 1990 as cited in McLaren/Hart, 1995).

Fluridone is taken up in fish tissue. Fluridone fish concentrations generally reflect the concentrations of fluridone in the water (McLaren/Hart, 1995). When fluridone residues are removed from the water column, the fluridone concentration from fish tissue clears (West *et al.*, 1983; Muir *et al.*, 1983 as cited in McLaren/Hart). Based on a low bioaccumulation rate in fish and high levels of fluridone necessary to produce toxic responses in mammals and birds, it is not expected that fish-eating animals would be affected by fluridone used at recommended (registered) application rates (McLaren/Hart, 1995).

The primary metabolite of fluridone degradation in fish was identified as 1-methyl-3-(4-hydroxyphenol)-5-[3-trifluoromethyl)phenyl]-4[1H]-pyridone (West *et al.*, 1983 as cited in McLaren/Hart, 1995). This compound was also identified as a minor metabolite in water and hydrosoil (Muir and Grift, 1982 as cited in McLaren/Hart, 1995). 1,4-dihydro-1-methyl-4-oxo-5-[3-(trifluoromethyl)phenyl]-3-pyridone was also identified as the major hydrosoil metabolite in hydrosoil studies conducted in the laboratory; however, this compound has not been identified in the hydrosoil of small ponds under natural conditions (West *et al.*, 1983 as cited in McLaren/Hart, 1995). A number of other metabolites including benzaldehyde, 3-(trifluoromethyl)-benzaldehyde, benzoic acid and 3-(trifluoromethyl)-benzoic acid were produced as photolytic breakdown products in one laboratory study (Saunders and Mosier, 1983, as cited in McLaren/Hart, 1995). N-methylformamide (NMF) was produced in another study. However, NMF has not been identified as a breakdown product under natural conditions (Saunders and Mosier, 1983 as cited in McLaren/Hart, 1995).

The half-life of fluridone in water of small, artificial ponds ranged from 1-7 days. In hydrosoils, the compound persisted for 8 weeks to one year (Joyce and Ramey, 1986; WSDOE, 1992). Fluridone has a water solubility of 12 mg/l and an octanol-water partition coefficient (K_{ow}) of 74.1 (Elanco Products Company, 1985 as cited in Aquatic Plant Identification and Herbicide Use Guide, 1988). Fluridone is stable to oxidation and hydrolysis (McCowen *et al.*, 1979). Volatilization of fluridone is not expected to be a significant process, (Muir and Grift, 1982 as cited in Aquatic Plant Identification and Herbicide Use Guide, 1988).

PHARMACOKINETICS

Metabolism and distribution studies have shown that fluridone is absorbed and excreted in the feces within 72 hours of oral administration to rats (McLaren/Hart, 1995). No bioaccumulation of fluridone was noted. 90% of the absorbed fluridone was cleared in 96 hours (USEPA, 1988).

HEALTH EFFECTS

Avian:

Fluridone has very low toxicity to birds. A number of acute toxicity studies were conducted in various bird species. An oral LD50 value of >2,000 mg/kg was obtained for bobwhite quail. The EPA considers this value to represent slight

toxicity (USEPA, 1986). An LD50 of >2,000 was identified for mallard ducks (WSDOE, 1992). Oral LC50 values of > 5,000 ppm were identified for bobwhite quail and mallard duck (USEPA, 1986). No impairment on reproduction for the above species was noted up to a dietary exposure concentration of 1,000 ppm (USEPA, 1986). In other studies, an LC50 value of about 10,000 ppm was identified for bobwhite quail and an LC50 value of >20,000 ppm was identified for mallard duck (WSDOE, 1992).

Mammalian:

Acute:

Most of the available information on the toxic effects of fluridone comes from the manufacturer, SePRO. Generally, the acute toxicity of fluridone is low. The LD_{50} for both rats and mice exposed through ingestion to technical grade fluridone is greater than 10,000 mg/kg. The oral LD_{50}s for cats and dogs exposed to technical grade fluridone are 250 mg/kg and 500 mg/kg, respectively. The LD_{50} for rabbits exposed through the skin to technical grade fluridone is greater than 2,000 mg/kg (Elanco, 1981 as cited in McLaren/Hart, 1995).

Fluridone was found to produce eye irritation in rabbits with effects including redness, corneal dullness and conjunctivitis. Fluridone was found to be neither irritating nor a sensitizer to rabbit skin at 2,000 mg/kg (USEPA, 1988).

Subchronic/Chronic:

In a three-week study in which fluridone was applied to rabbit skin daily at doses ranging from 192-768 mg/kg/day, dose-dependent skin irritation was produced at all doses. No systemic effects were noted at any dose. An increase in organ weight was noted at 384 mg/kg/day (USEPA, 1988).

In a three-month subchronic feeding study with fluridone, no treatment-related effects were noted in rats administered doses of 62 mg/kg or in mice administered doses of 330 mg/kg (Elanco, 1981 as cited in McLaren/Hart, 1995). A dietary level of fluridone of 16.5 mg/kg/day administered to mice for three months resulted in a partial enlargement of livers. A dietary level of 166 mg/kg administered to rats for three months resulted in an increase in liver weights. A No Observed Effect Level (NOEL) of 30 mg/kg/day was identified in rats administered fluridone in the diet for three months (EPA, 1988). A concentration of 0.033% of fluridone fed to mice for three months produced morphologic changes in the liver and an increase in absolute liver weights in male mice (USEPA, 1988). In a study conducted with dogs, daily dietary fluridone levels up to 200 mg/kg/day did not result in any treatment-related effects (Elanco, 1978 as cited in USEPA, 1990).

In a one-year chronic study in which dogs were administered fluridone by capsule in food, a number of effects including weight loss, an increase in liver weight and an increase in liver enzymes were noted at a dose level of 150 mg/kg/day. A NOEL of 75 mg/kg/day was identified (USEPA, 1988). In a two-year feeding study in which mice were administered fluridone concentrations in the diet of up to 330 ppm fluridone, there was an increase in liver enzymes in males exposed at 330 ppm. No other toxic effects or lesions were noted at any of the doses (USEPA, 1988). In another two-year study, rats were exposed to doses of 0, 8, 25 and 81 mg/kg/day. At 25 mg/kg/day, rats experienced inflammation in the kidney, atrophy of the testes, inflammation of the cornea, weight loss and decreased organ weights (USEPA, 1988; USEPA, 1990).

Developmental/Reproductive:

In a study in which rats were exposed to up to 200 mg/kg/day of fluridone, neither maternal nor fetotoxic effects were noted (USEPA, 1988). In a three-generation study conducted in rats exposed to fluridone at a dose of 100 mg/kg/day, no teratogenic or maternal effects were noted. However, a dose of 100 mg/kg/day was found to be toxic to rat pups (USEPA, 1988; USEPA 1990). In a teratology study in which rabbits were exposed to fluridone doses of up to 750 mg/kg/day, a level of 300 mg/kg resulted in maternal effects including a decrease in body weight gain and abortion. Fetal effects, also noted at this level, included resorptions (USEPA, 1988). No teratogenic effects were noted (USEPA,

1990). In a pilot study in which rabbits were exposed to fluridone at doses of 0, 250, 500, 750 and 1,000 ppm, a maternal NOEL of 500 mg/kg was identified. A level of 750 mg/kg produced a maternal loss in body weight. A NOEL of 250 mg/kg/day was identified for fetal effects. At 500 mg/kg/day, fetal resorptions occurred (USEPA 1988). In another study, rats were administered doses by oral gavage of 0, 100, 300 and 1,000 mg/kg/day. A maternal NOEL of 100 mg/kg/day was identified. At 300 mg/kg/day, there was a decrease in maternal body weight. The NOEL for developmental effects was identified as 300 mg/kg/day. At 1,000 mg/kg/day, fetal effects included a decrease in fetal weight and delayed ossification. The NOEL for teratogenic effects was greater than 1,000 mg/kg/day (USEPA 1988).

Mutagenicity:

Fluridone was not found to be mutagenic in several test assays. Fluridone produced negative results in the Ames assay and did not induce sister chromatid exchange in Chinese hamster bone marrow cells. In addition, fluridone did not promote unscheduled DNA synthesis in rat hepatocytes (USEPA, 1988).

Carcinogenicity:

Based on negative cancer findings in the two chronic toxicity studies discussed above, there is no evidence indicating that fluridone is carcinogenic. The EPA Health Effects Division has designated fluridone as a Group E carcinogen (i.e., having evidence of noncarcinogenicity for humans) (USEPA, 1995).

AVAILABLE TOXICITY CRITERIA

The EPA Carcinogen Risk Assessment Verification Endeavor (CRAVE) (RfD/RfC) workgroup has developed an oral Reference Dose (RfD) of 0.08 mg/kg/day for fluridone based on one of the two-year rat feeding studies conducted by Elanco cited earlier (USEPA, 1990). The EPA Office of Pesticide Programs (OPP) has calculated the same RfD value based on the same study (USEPA, 1995). The RfD is an estimate, (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime (USEPA, 1990).

The EPA has designated an acceptable residue level for fluridone in potable water of 0.15 ppm. This level is based on the maximum application rate for fluridone as registered under FIFRA (Federal Insecticide, Fungicide and Rodenticide Act) (USEPA, 1986 as cited in McLaren/Hart, 1995). The EPA has also established a tolerance of 0.5 ppm for residues of fluridone and its primary metabolites in fish and crayfish. In addition, EPA has established tolerances for crops irrigated with water containing fluridone residue concentrations at 0.15 ppm as well as for a number of raw, agricultural commodities (USEPA, 1986 as cited in McLaren/Hart, 1995).

ECOLOGICAL TOXICITY

Aquatic Organisms:

A number of studies have been conducted with fluridone to determine the LD50 or LC50 values for a variety of organisms. The LD50 (or LC50) is the dose (or concentration) to which a particular species is exposed, which results in the death of 50% of the test population. The EPA has cited the results of a number of these studies. EPA considers these studies to demonstrate moderate toxicity. These studies are listed in the following table:

Table III.5-2. Acute Toxicity Tests

SPECIES	TEST TYPE	VALUE
Daphnia magna	48-hr LC50	6.3 mg/l
Bluegill	96-hr LC50	12 mg/l
Rainbow trout	96-hr LC50	11.7 mg/l
Sheepshead minnow	96-hr LC50	10.91 mg/l
Oyster embryo larvae	48-hr LC50	16.51 mg/l

(USEPA, 1986)

In addition, a Maximum acceptable theoretical concentration (MATC) value for fathead minnow (second generation fry) was calculated to be between 0.48 mg/l and 0.96 mg/l, meaning no treatment-related effects were noted at or below 0.48 mg/l. Total length of 3-day old fry was reduced at 2 mg/l fluridone (USEPA, 1986).

No adverse effects were noted on crayfish, bass, bluegill, catfish, long-neck soft-shelled turtles, frogs, water snakes and waterfowl from the use of 0.1 to 1.0 ppm fluridone during field experiments (Arnold, 1979, McCowen, 1979 as cited in WSDOE, 1992). Application of 1.0 ppm fluridone to zooplankton caused a reduction in population, but the population quickly recovered. Application of 0.3 ppm did not cause a change in the total number of benthic organisms whereas application of 1.0 ppm did cause a change (Parka *et al.*, 1978 as cited in WSDOE, 1992). An aqueous solution of fluridone caused a reduction in population of the amphipod *Hyalella azteca* when applied at a rate of 1.0 ppm but not when applied at a rate of 0.3 ppm (Arnold, 1979 as cited in McLaren/Hart, 1995). Fish abundance and community structure remained unchanged in ponds exposed to a fluridone concentration level of 0.125 ppm (Struve *et al.* 1991 as cited in McLaren/Hart, 1995). LC50 values for a variety of microscopic crustaceans including *Diaptomus*, sp., *Eucyclops* sp, *Alonella* sp., and *Cypria* sp., ranged from 8.0 - 13.0 ppm (Naqvi and Hawkins, 1989 as cited in McLaren/Hart, 1995).

One group of investigators conducted extensive acute toxicity tests on a variety of aquatic invertebrates including amphipods, midges, daphnids, crayfish, blue crabs, eastern oysters and pink shrimp. The average 48-hour or 96-hour LC50 or EC50 (concentration at which 50% of the organisms exhibit an effect) was calculated as 4.3 ± 3.7 ppm (Hamelink *et al.*, 1986 as cited in McLaren/Hart, 1995). The same investigators also conducted studies with a variety of fish including rainbow trout, fathead minnows. channel catfish, bluegills and sheepshead minnows. A 96-hour LC50 value of 10.4 ± 3.9 was calculated (Hamelink *et al.*, 1986 as cited in McLaren/Hart, 1995).

Daphnids, amphipods and midge larvae exposed chronically to fluridone concentrations of 0.2, 0.6 and 0.6 ppm as well as catfish fry exposed to fluridone concentrations of 0.5 ppm showed no treatment-related significant effects. Exposure to concentrations of 1 ppm produced a decreased growth rate of catfish fry and concentrations of 0.95 and 1.9 ppm produced a decreased survival rate of fathead minnows within 30 days after hatching (Hamelink *et al.*, 1986 as cited in McLaren/Hart, 1995).

Plants:

Fluridone selectively controls a number of broad-leaved submerged and floating aquatic macrophyte species as specified by its EPA label. In addition, the literature contains reports of fluridone's variable efficacy in controlling other species. The efficacy of fluridone is very dependent on contact time with plants. Thus, fluridone should be applied during periods of minimum water movement. Factors related to fluridone's variable efficacy include temperature, pH and light levels (Wells *et al.* 1986 as cited in WSDOE, 1992). In addition, one investigator found that in *Hydrilla*

exposed to fluridone at various concentrations for 1, 3 and 5 weeks, plant recovery was directly related to the concentration of active iron (Fe^{2+}) in the plant at the time of treatment (Spencer and Ksander, 1989 as cited in WSDOE, 1992).

Fluridone did not appear to adversely affect desirable phytoplankton but some reduction in population of the less desirable species given as *Anabaena* and *Anacystis* occurred upon application of fluridone at levels of 0.3 and 0.1 ppm (Parka et al, 1978 as cited in WSDOE, 1992). A drastic reduction in phytoplankton population in Greek ponds including the disappearance within two months of a population of Cyanophyceae (Cyanobacteria) occurred after fluridone application. Diatom populations, a more desirable species, increased significantly, especially epiphytic and benthic species (Kamarianos *et al.*, 1989 as cited in WSDOE, 1992). No sufficient reduction in phytoplankton densities was noted when they were consistently exposed to a fluridone concentration of 0.125 ppm (Struve *et al.*, 1991 as cited in McLaren/Hart, 1995).

An aqueous solution of fluridone applied at a concentration of 1.0 ppm produced a significant reduction in a zooplankton population whereas a concentration of 0.3 ppm had no effect. The 1.0 ppm population returned to pretreatment levels within 43 days (Arnold, 1979 as cited in McLaren/Hart, 1995). As specified by its EPA label, 0.15 ppm is the maximum registered rate for any use site (Cockreham, pers. comm.).

Table III.5-3. Properties of Fluridone

CAS #:	59756-60-4
Synonyms:	1-methyl-3-phenyl-5-[3-(trifluoromethyl)phenyl-4(1H)-pyridinone; EL 171; Sonar
Molecular formula	$C_{19}H_{14}F_3NO$
Molecular weight	329.3
Physical properties	white, crystalline solid
Melting point	154-155^{o}C
Vapor pressure	< 1 x 10^{-7} mm Hg at 25^{o}C
Photolysis half-life	1-6 days
Hydrolysis half-life	stable
Biodegradation half-life	2-60 days (based on overall half-life)
K_{ow}	74.1 at 20^{o} C
K_{oc}	~350-2460 ml/g
BCF	0.9-15.5
Water solubility	12 mg/l at 25^{o} C and pH 7

(Reinert and Rodgers, 1987; WSSA, 1983; Aquatic Plant Identification and Herbicide Use Guide, 1988; WSSA, 1994)

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