ENVIRONMENTAL FATE OF FIPRONIL

Pete Connelly
Environmental Monitoring Branch
Department of Pesticide Regulation
California Environmental Protection Agency
P.O. Box 4015
Sacramento, CA 95812-4015

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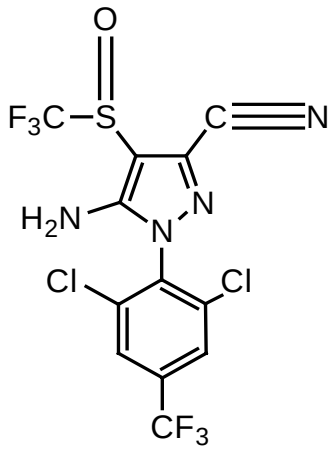
This document reviews the environmental fate and environmental effects of fipronil (5-amino-1-[2,6-dichloro-4-(trifluromethyl)phenyl]-4-[(trifluromethyl)sulfinyl]-1*H*-pyrazole). Fipronil ($C_{12}H_4Cl_2F_6N_4OS$), a phenylpyrazole insecticide, was discovered by Rhone-Poulenc Agro in 1987, introduced in 1993, and registered as a pesticide in the U.S in 1996 (Bobe et al. 1998a; The British Crop Protection Council, 1997; University of Minnesota, 2001). Fipronil can be formulated as roach or ant baits, flea and tick sprays for pets, and in granular turf products to control mole crickets (U.S. EPA, 1996; Kidd and James, 1991; University of Minnesota, 2001). It is also used on a variety of foliar and soil insects including corn rootworm, Colorado potato beetle, and rice water weevil that attack a variety of crops, mostly corn and rice (University of Minnesota, 2001). Fipronil is also effective for locust and termite control, against insects in both larval and adult stages, as well as insects resistant to pyrethroid, organophosphate, and carbamate insecticides (Kidd and James 1991; University of Minnesota 2001; Bobe et al., 1997). Fipronil disrupts normal nerve function by targeting the γ-aminobutyric acid type A (GABA) receptor system of insects (Kidd and James 1991). Fipronil represents the second generation of insecticides that act on the GABA receptor system as a non-competitive blocker. The polychlorocycloalkanes are the first generation of such

insecticides, and these include the pesticides α-endosulfan and lindane (Hainzl et al., 1998). Fipronil's mechanism of action is to block the voltage gated chloride channels of neurons. The effect is to remove normal inhibition, resulting in excessive neuronal activity (Kidd and James, 1991). Cole et al. (1993) determined that fipronil interferes with the passage of chloride ions through the GABA channel. At sufficient doses, fipronil causes excessive neural excitation, severe paralysis and insect death (Bobe et al., 1998a). Fipronil also demonstrates a selective toxicity toward insects by having a tighter binding affinity toward the insect GABA-regulated chloride channels compared to mammalian GABA receptors (Hainzl et al., 1998).



Fipronil

5-amino-1-[2,6-dichloro-4-(trifluromethyl)phenyl]-4-[(trifluromethyl)sulfinyl]-1*H*-pyrazole

Physico/chemical properties: [a]

CAS Number: 120068-37-3
Molecular weight: 437.2
Solubilities [b]
Water (pH = 5): 0.0024 g/l (pH = 9): 0.0022 g/l
Acetone: 545.9 g/l
Hexane: 0.028 g/l
Octanol: 12.2 g/l
Melting Point [ac]: 200-201° C
Melting Point (Technical Grade) [a]: 195.5-203° C
Vapor Pressure [a]: 3.7 x 10^{-4} mPa @ 25° C
Henry's Constant [a]: 3.7 x 10^{-5} (Pa m^3/mol)
log K_{ow} [a] = 4.01
K_{oc} (average value) [g] = 803

Environmental Fate:

Hydrolysis Half-lives [e]: @22°C: (pH 9.1) = 1,100 days, (pH 7.1) = 1,390 days
@32°C: (pH 9.1) = 11.3 days, (pH 7.1) = 15.6 days
Aqueous Photolysis [d]: 4.1 Hours (pH 5.5)
Aerobic Aquatic Half-Life [f]: 14.5 days
Soil Photolysis [h]: 34 days
Field Dissipation Half-Life [h]: 102-160 days
Aerobic Soil Half-life [h]: 630-693 days
Anaerobic Soil Half-life [h]: 123 days

Toxicity: Technical Grade fipronil

Acute Oral LD_{50} (Rat) [a]: 97 mg/kg
Acute Oral LD_{50} (Mice)[a]: 95 mg/kg
Acute Dermal LD50 (Rabbit)[a]: 354 mg/kg(14)
Acute Percutaneous LD_{50} (Rat)[a]: >2000 mg/kg
Acute Inhalation LC_{50} (Rat)[a]: 0.682 mg/l air-4 hours

Ecological Effects: Technical Grade Fipronil

***Acute toxicity*[b]**

Rainbow Trout 96-hour LC_{50}[I]: 0.248 ppm
Bluegill Sunfish 96-hour LC_{50}[I]: 0.085 ppm
Sheepshead Minnow 96-hour LC_{50}[h]: 0.13 ppm
Mysid Shrimp 96-hour LC_{50} [I]: 0.000140 ppm
Daphnia 48-hour LC_{50}[ac]: 0.19 ppm
Mallard Oral LD_{50}[a]: >2000 mg/kg
Mallard 5-day Dietary LC_{50}[a]: >5,000 mg/kg
Bobwhite Oral LD_{50}[a]: 11.3 mg/kg
Bobwhite 5-day Dietary LC_{50}[a]: 49 mg/kg

Chronic Toxicity[b]:

Invertebrate (Daphnia) Life Cycle NOEC: 0.0098 ppm
Mallard Reproduction NOEC: 1000 ppm
Bobwhite Reproduction NOEC: 10 ppm
Fish (Rainbow Trout) Early Life Stage NOEC: 0.0066 ppm
Fish (Rainbow Trout) Early Life Stage LOEC: 0.015 ppm

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[a]British Crop Protection Council, 1997.
[b]U.S Environmental Protection Agency, 1996.
[c]Kidd and James, 1991.
[d]Bobe, 1998b.
[e]Ramesh et al, 1999.
[f]Feung, 1997.
[g]Mede, 1997.
[h]DPR Pest-Chem Database, 2001.
[I]DPR EcoTox Database, 2001.

Environmental Fate of Fipronil

Air: Fipronil has a relatively low vapor pressure and a low Henry's law constant, so that fipronil does not readily volatilize. Consequently, except for drift that may occur during spray applications, fipronil is not likely to be found in the air (The British Crop Protection Council, 1997; U.S. EPA, 1996).

Soil: *Sorption and Mobility:* The average K_{oc} value for fipronil is 803, indicating low-to-moderate sorption to soil surfaces (Mede, 1997; DPR 2001). It has been suggested by the U.S. EPA that fipronil has a low to slight mobility in soil, this would result in a low potential for ground water contamination (U.S. EPA 1996; Burr, 1997). It is the opinion of the U.S. EPA that the surface degradation of fipronil occurs mainly through slow photolysis and /or soil binding along with microbial mediated processes; however, below the surface fipronil has a tendency to dissipate by soil binding along with gradual microbial breakdown (U.S. EPA, 1996). A comparative study of the adsorption of

fipronil using two Sahelian soils and a Mediterranean soil (Montpellier) found that fipronil sorption is positively correlated with organic carbon content in the soil (Bobe et al., 1997). In a similar study the adsorption/desorption characteristics of one of the major metabolites of fipronil was studied. The objective was to determine the adsorption/desorption characteristics of the sulfide metabolite using four soils and one sediment. The authors suggested that, depending on the soil type, the sulfide metabolite has a low to slight mobility in soil and would not be expected to move to deeper soil layers (Burr et al., 1997).

Field Dissipation: Reported values for field dissipation half-lives are 33-75 days for bare soil and 12-15 days in turf. The photodegradation of fipronil in loamy soil is considered slow, with a half-life of 34 days. Under aerobic conditions, organisms present in the soil gradually breakdown fipronil. Aerobic soil metabolism studies reported the half-life of fipronil in sandy loam to be 122 days with the amide and sulfone metabolites accounting for 27-38% and 14-24% of the total applied radioactivity, respectively (U.S. EPA, 1996). In a two-month terrestrial field study in the Niamey region of Niger, 8 g a.i. ha^{-1} was applied to uncultivated soils at Banizoumbou and Saguia. Recovery at 3-days post-treatment was 25% of the original applied dose. In addition to fipronil, the authors detected the formation of four degradates, including a sulfide, which is the product of reduction in soil; an amide, the product of hydrolysis in water or soil; and a sulfone from oxidation in soil (Bobe et al., 1997). The authors also detected a desulfinyl photodegradate. The sulfone degradate appeared rather rapidly in the first 3 days and formation decreased for the rest of the study. The authors did not detect fipronil or

degradates below the 10-cm layer. On days 14 and 28, the 0-10 cm layer was divided into two layers (0-5 and 5-10 cm) fipronil levels were significantly higher in the 0-5 cm layer. Fipronil levels went from 0.002 ppm to below the limit of quantification (0.0001 ppm) at Banizoumbou and from 0.001 to 0.0002 ppm at Saguia. The calculated half-life from the 0-10 cm soil layer at Banizoumbou was approximately 36 hours (Bobe et al., 1997).

Water: The reported solubilities for fipronil are 2.0-2.4 ppm (U.S EPA 1996; Kidd and James 1991; Ayliffe 1998). In Feung and Yennes' (1997) study on aquatic metabolism using radioactively labeled fipronil (^{14}C-fipronil) and a sandy loam with an organic matter content of 8.0% (pH = 5.80), average radioactivity rapidly transferred/adsorbed to the sediment. Aerobic incubations containing sediment and pond water were treated with fipronil to yield an initial concentration of 0.05 ppm, which is based on the total water content of 50 mls in each test container. Two ^{14}C-fipronil incubated test samples were taken at 0, 7 and 14 days, and 1, 2, 3, 6, 9, and 12 months. The average radioactivity decreased in the water from an initial level of 21.06% at day zero to 4.0% at 7 days and remained at 1.6-3.8% for the balance of the study. The extractable radioactive fipronil decreased from 99.46% of the applied dose to 4.07% at 60 days to non-detectable at 12 months. The major metabolite, in the sediment, was identified as the sulfide degradate (reduced methylthio); which rapidly increased from an initial level of 17.67% at 7 days to 78.89% of the applied dose at 60 days, increasing to 80-87% for the remainder of the study. The reported half-life for fipronil under aerobic aquatic conditions was about 14.5 days. The major metabolite (sulfide degradate) represented 74% of the total radioactive residues after 30 days (Feung and Yenne 1997). The remaining minor metabolites were

identified by HPLC as the amide degradate, the carboxamide, and the desulfinyl photodegradate. Each was less than 4% of the applied dose throughout the study. The sulfide degradate was a significant metabolite in anaerobic aquatic conditions whereas under aerobic soil conditions the amide and the sulfone were identified as the major degradation products (Feung and Yenne, 1997).

In an aquatic field dissipation study (Mede et al., 1997) using rice fields in Mississippi, Texas, and California, rice seed and soil plots treated with fipronil were used to monitor the extent of dissipation and mobility of fipronil, its primary metabolites, and photodegradate under actual field conditions. There were four analytes studied, including the sulfone, sulfide, amide, and desulfinyl photodegradate. For both trials flowable suspensions of fipronil (700g ai/L) were used to treat seeds or soil. For the soil trials fipronil was used to treat the soil before planting, called pre-plant incorporation (PPI). For each analyte the limit of quantification (LOQ) for soil was 0.005 ppm and for the water 0.001 ppm. For the soil trials, two samples were taken, one at 0.3 inches and one at 0.6 inches. Water samples were taken at flood up and at predetermined intervals thereafter. The only quantifiable residue in the water was found in California. The authors postulated that this is likely due to the different seeding technique employed in California. The rice is water seeded in California and dry seeded in Mississippi and Texas. With water seeding the field was flooded then seeded with fipronil treated seeds, so the field floodwater comes into contact with the fipronil treated seed immediately. In the dry seeding method the plot is flooded 4-6 weeks after being seeded, allowing photodegradation to occur as well as giving the analytes time to bind to the soil.

According to the authors, data from paddy water in California indicated considerable binding of the residues to soil. The combined residue (fipronil plus the combined analytes) in water data was <0.02 ppm and reportedly dissipated. The authors reported soil and water residue data that include the dissipation half-lives of the combined residue, the parent compound (fipronil), as well as the individual degradates. In California rice water, the estimated half-life for the combined residue is, 7.3 days (seed) and 5.6 days (PPI). The water for the locations that were dry seeded (Texas and Mississippi) yielded no residues (Mede et al., 1997). For the parent compound, the estimated water half-life (California) from the seed treatment and PPI was 1.7 and 2.9 days, respectively. The half-life of the photo-degradate was 17.4 days for the seed treatment and 18.8 days for the soil PPI. The water half-lives for the other three major metabolites, the sulfide, sulfone, and the amide were below the limit of quantification (LOQ). The reported soil half-lives for the seed and the soil treatments (PPI) for the combined residues were: Texas, 99 and 347; Mississippi, 35 and 97.6; California 210 and 257 days, respectively. For the parent compound the soil half-lives for seed and soil treatments (PPI) were: Texas, 12.6 and 18.8; Mississippi, 14.6 and 17.4; California 11.9 and 119 days, respectively. According to the authors, the combined metabolites show a slower degradation rate than fipronil, indicating a continuing degradation of these analytes. The average K_{oc} value for fipronil is 803. The K_{oc} for the metabolites are 2719 for the sulfide, 4209 for the sulfone, 1290 for the photo-degradate, and 166 for the amide (Mede et al., 1997). Larger K_{oc}s indicate a tendency to become more strongly adsorbed to soil particles, decreasing mobility in soil. Based on the K_{oc} data, the photodegradate is expected to display a low to moderate mobility, the sulfide and sulfone a low mobility, and the amide a high mobility.

Hydrolysis of fipronil is pH dependent and follows pseudo-first-order kinetics. Ramesh (1999) reported no observable change in the hydrolysis of fipronil at pH of 4-9 at 5°C. At 5-22° C fipronil is stable to hydrolysis in both acidic and neutral buffer solutions. The half-life of fipronil in aqueous buffered solutions at 22° was 1,660 days, 1,390 days, and 1,100 days at pH 4.1, 7.1, and 9.1, respectively. In contrast at 32°, the initial fipronil concentration was reduced by 80% in acidic buffer, 92% in neutral buffer and 98% in basic buffer in 16 days (Ramesh et al., 1999). At 50°C fipronil was completely degraded by the twentieth day in both acidic and neutral conditions and by the fifteenth day in basic buffer (U.S. EPA). In comparison at 22°C, Bobe et al. (1998b) showed that fipronil was stable in acid (pH 5.5) and neutral (pH 7.0) aqueous solutions, in both cases 80% remaining unchanged after 100 days. Under alkaline conditions at 22°C (pH 9.0-12.0), fipronil degradation was 300 times faster at pH 12 than at pH 9.0. The hydrolysis $t_{1/2}$ values were 0.1 and 32 days, respectively (Bobe et al., 1998b). In summary, the hydrolysis data suggest that hydrolysis will not be a major degradative pathway for fipronil at typical environmental pH's.

Photolysis: Ten-milliliter samples of 2.5 ppm aqueous solutions (2.5% methanol in water, at pH 5.5) of fipronil were irradiated using simulated sunlight for periods of 0, 1, 2, 3, 4, 6, 14, 18, and 24 hours. The aqueous photo degradation (photolysis) half-life of fipronil was determined to be 4.1 hours at the hydrolytically stable pH of 5.5 (Bobe et al., 1998b). These data indicate that fipronil photolysis is more important than hydrolysis for degradation of aqueous fipronil at environmental pHs.

Biota: In a photochemical reaction on the surface of plants, fipronil was transformed yielding the desulfinyl photodegradate. This photoproduct has high neuroactivity, effectively blocking the GABA chloride channels. This neurotoxic photoproduct is not a metabolite in mammals; however, it does have high affinity towards the insect GABA system, contributing to fipronil's selective toxicity toward insects by binding irreversibly to the insect GABA system (Casida et al., 1996).

Fipronil is toxic to mammals via ingestion. The oral LD_{50} for rats is 97mg/kg. It is slightly toxic to nontoxic via the dermal route, with a reported dermal LD_{50} of greater than 2000 mg/kg in rats (U.S. EPA, 1996). Fipronil is not a skin sensitizer in guinea pigs, but has shown slight dermal irritation in rabbits (U.S. EPA 1996). Fipronil has moderate inhalation toxicity with an acute LC_{50} of 0.682 mg/l in rats (U.S. EPA, 1994-1996).
On an acute and sub-chronic level fipronil is practically non-toxic to waterfowl with an acute oral LC_{50} of >2,000 mg/kg and a 5-day dietary LC_{50} of >5,000 mg/kg for Mallard ducks. However, according to the ecological effects data on upland game birds, fipronil is highly toxic on an acute oral basis and very highly toxic on a sub-acute dietary basis. The oral LC_{50} for Bobwhite quail is 11.3 mg/kg, and the LC_{50} for 5-day dietary is 49 mg/kg (U.S EPA, 1996). Using an endpoint of reproductive success, chronic toxicity studies indicate no effect at the highest levels tested in mallards (NOEC = 1000ppm) and quail (NOEC = 10ppm). The sulfone metabolite is more toxic than the parent compound to certain bird species. This metabolite has shown a very high toxicity toward upland game

birds and moderate toxicity toward waterfowl on an acute oral basis (U.S. EPA 1996, Bobe et al., 1997).

Fipronil is considered highly toxic to rainbow trout and very highly toxic to bluegill sunfish with an LC_{50} of 0.246 ppm and 0.083 ppm, respectively. In early life-stage studies on rainbow trout fipronil affected larval growth with a NOEC of 0.0066 ppm and a LOEC (Lowest Observable Effect Concentration) of 0.015 ppm. The sulfone metabolite is 6.3 times more toxic to rainbow trout and 3.3 times more toxic than the parent compound to bluegill sunfish. Fipronil demonstrates a high toxicity toward freshwater aquatic invertebrates as well. In acute daphnia life cycle studies, fipronil affected growth: daphnid length was decreased at concentrations greater then 9.8 ppb. The sulfone metabolite is 6.6 times more toxic and the desulfinyl photodegradate 1.9 times more toxic on an acute basis to freshwater invertebrates than the parent compound (U.S. EPA 1996).

Conclusions

Fipronil (5-amino-1-[2,6-dichloro-4-(trifluromethyl)phenyl]-4-[(trifluromethyl)sulfinyl]-1*H*-pyrazole), a phenylpyrazole insecticide, exhibits neurotoxic activity by blocking the GABA- regulated chloride channels of neurons. It is useful for the control of roaches, ants, termites, fleas and ticks. It is often formulated as insect bait, sprays for pets, and as a granular turf product to control mole crickets.

Fipronil affects target species by blocking the chloride channels of neurons; therefore the resting membrane potential cannot be re-established, the result is excessive neuronal activity. At sufficient doses fipronil causes paralysis and insect death.

Due to its very low vapor pressure and Henry's Law constant, fipronil is not likely to be found in the air. Fipronil is readily transformed into its desulfinyl photodegradate when exposed to sunlight. This photoproduct has a high affinity for insect GABA regulated chloride channels. Consequently, the photoproduct is neurotoxic toward insects.

Laboratory data indicate that fipronil is much more susceptible to breakdown through photolysis rather than hydrolysis in water. Under environmental pH's fipronil is stable to hydrolysis with a half-life of 1390 days at pH 7.1 (22° C). The laboratory photolytic half-life was 4.1 hours; suggesting that photolysis is a more important pathway for the degradation of aqueous fipronil. Hydrolysis of fipronil is only important at a very basic pH. The hydrolysis half-lives for pH of 12 and 9 in aqueous solutions were 0.1 and 32 days, respectively. In soil, fipronil tends to dissipate by soil binding along with gradual microbial breakdown; however, on the soil surface photolysis may also be important.

A reported field half-life of fipronil under aerobic aquatic conditions was 14.5 days. The major metabolite was the sulfide degradate. In an aerobic metabolism study fipronil readily partitioned from the aqueous layer into the sediment, with most of the fipronil reaching the sediment layer within seven days after application. The extractable radioactive fipronil decreased from 99.46% of the applied dose to 4.07% at 60 days of incubation to non-detectable at 12 months. The major metabolite in anaerobic aquatic

conditions was the sulfide degradate while both the amide and sulfone were products of aerobic soil conditions.

The degradation products of fipronil are high to highly acutely toxic to rainbow trout, bluegill sunfish, and freshwater invertebrates. The sulfone degradate is 6.3 times more toxic to rainbow trout, 3.3 times more toxic to bluegill sunfish, and 6.6 times more toxic to freshwater invertebrates. The sulfide degradate is 1.9 times more toxic to freshwater invertebrates. The sulfone degradate is very highly toxic to upland game birds and moderately toxic to waterfowl on an acute oral basis.

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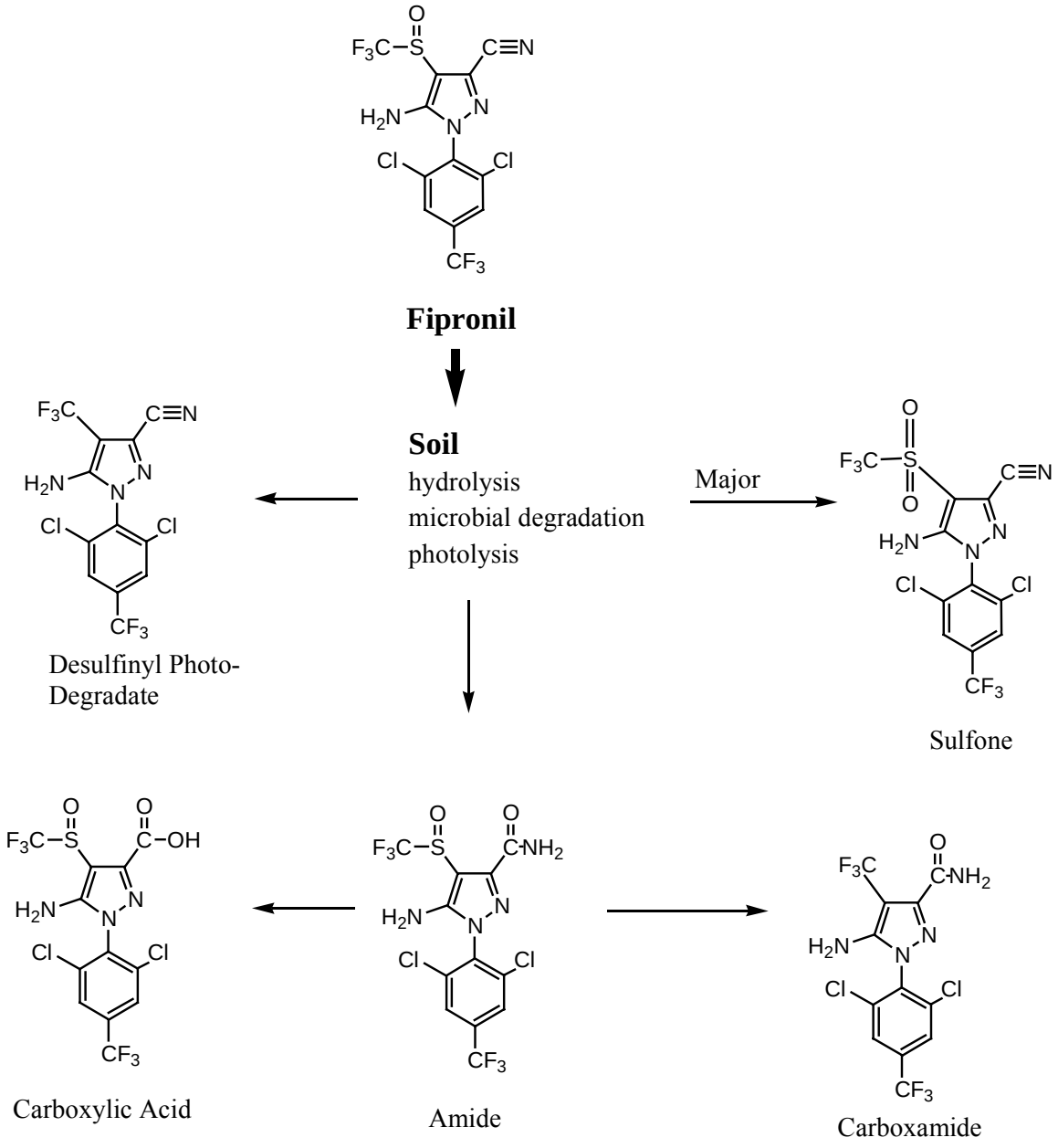
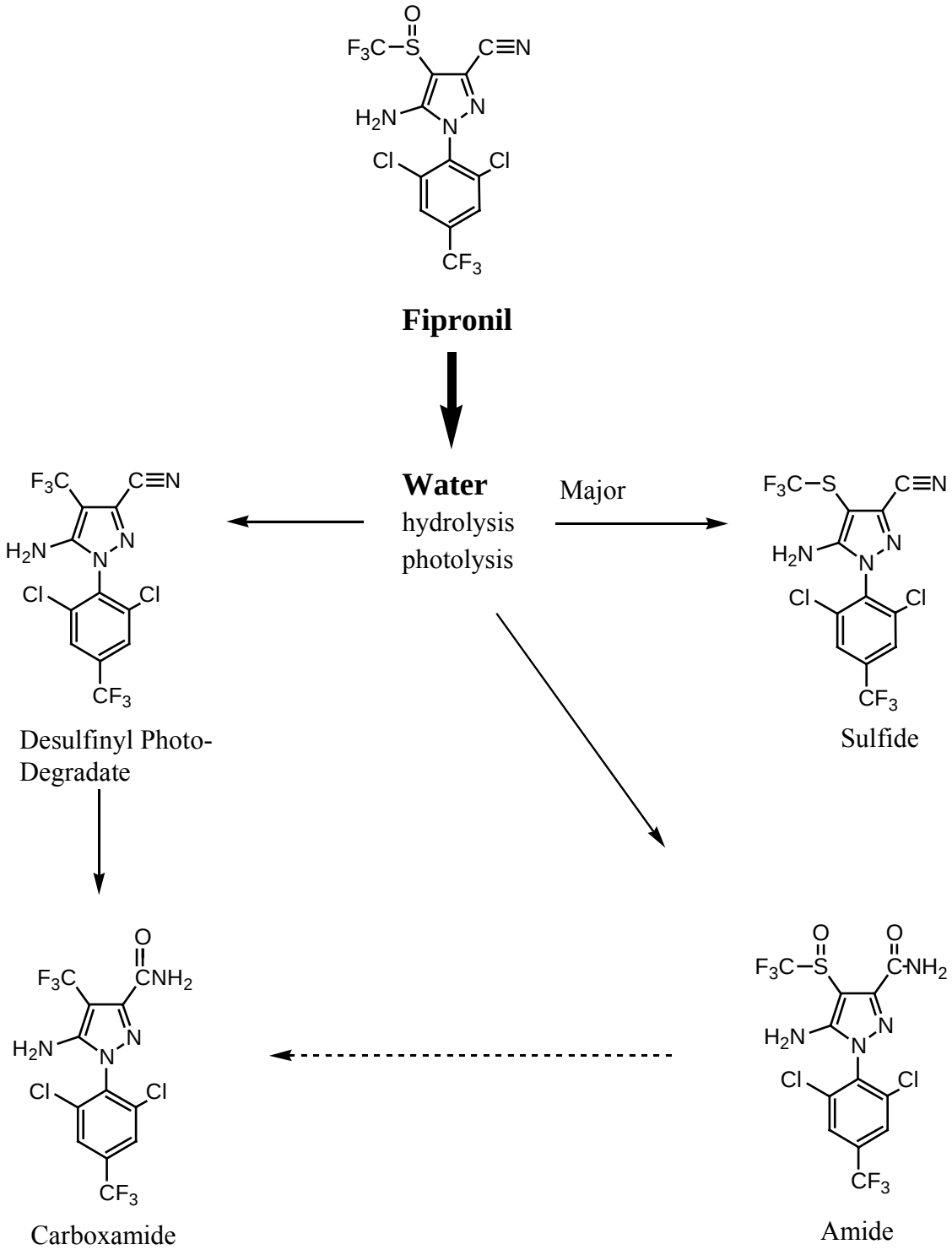
Degradation Pathway for Fipronil




Fipronil
Water
hydrolysis
photolysis
Major
Desulfinyl Photo-
Degradate
Sulfide
Carboxamide
Amide

CAS Names for chemical structures in degradation pathways:

Desulfinyl Photodegradate = 1-H-Pyrazole-3-Carbonnitrile, 5-amino-1-[2,6-dichloro-4-(trifluoromethyl)phenyl]-4-trifluoromethyl

Sulfone = 1-H-Pyrazole-3-Carbonnitrile, 5-amino-1-[2,6-dichloro-4-[(trifluoromethyl)phenyl]-4-[(trifluoromethyl)sulfonyl]

Sulfide = 1-H-Pyrazole-3-carboxylic acid, 5-amino-1-[2,6-dichloro-4-[(trifluoromethyl)phenyl]-4-[(trifluoromethyl)thio]

Carboxylic Acid = 1-H-Pyrazole-3-carboxylic acid, 5-amino-1-[2,6-dichloro-4-[(trifluoromethyl)phenyl]-4-[(trifluoromethyl)sulfonyl]

Amide = 1-H-Pyrazole-3-carboxylic acid, 5-amino-1-[2,6-dichloro-4-[(trifluoromethyl)phenyl]-4-[(trifluoromethyl)sulfinyl]

Carboxamide = 1-H-Pyrazole-3-carboxylic acid, 5-amino-1-[2,6-dichloro-4-[(trifluoromethyl)phenyl]-4-[trifluoromethyl]