Yearly Operational Plan (YOP)

Town of Ashland

2025

Submitted on:
March 20, 2025

Prepared By:
Town of Ashland
Department of Public Works
101 Main Street
Ashland, MA 01721
508-881-0100

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1.0 Program Purpose

The purpose of 333 CMR 11.00, Rights of Way Management, is to promote the implementation of integrated pest management techniques and to establish standards, requirements, and procedures necessary to minimize the risk of unreasonable adverse effects on human health and the environment associated with the use of herbicides to maintain streets, roads, sidewalks and paths. These regulations establish procedures that guarantee ample opportunity for public and municipal agency review and input on the right-of-way maintenance plans.

A yearly operational plan (YOP) must be submitted to the Department of Agricultural Resources (MDAR) every year herbicides are intended for use to maintain rights-of-way. The YOP provides a detailed program for vegetation management for the year. A five-year Vegetation Management Plan (VMP) was approved by the Department and is available for review at the Ashland Department of Public Works, Board of Health, Conservation Commission, and the Board of Selectmen's Office. The VMP was submitted for 2025 through 2029.

Upon receipt of this YOP, the Department publishes a notice in the *Environmental Monitor*. The Town must provide a copy of the proposed YOP and *Environmental Monitor* notice to the Board of Health, Conservation Commission, and the Select Board Chairperson for the Town of Ashland, in which the herbicide treatment is proposed. The Department allows a 45-day comment period on the proposed YOP beginning with the publication of the notice and receipt of the YOP and *Environmental Monitor* notice by the Town.

Public notification of herbicide application to the streets is made in advance of the treatment by a separate notice and in accordance with 333 CMR 11.00. Notice is made to the Department of Agricultural Resources, Board of Selectmen Chairperson, Board of Health, and the Conservation Commission in the Town of Ashland.

Any comments on this YOP should be made to the person designated herein as the person supervising the YOP or the person performing the treatment.

This Yearly Operational Plan, approved by the Massachusetts Department of Agricultural Resources pursuant to Rights-of-Way Management Regulations (333 CMR 11.00), has been adopted by the following roadway vegetation management program in the Town of Ashland. The undersigned hereby acknowledges that the conditions of the Yearly Operational Plan will be adopted and complied with.

Municipality:	Town of Ashland
Name:	Doug Small, Director
Office:	Department of Public Works
Address:	101 Main Street, Ashland, MA 01520
Telephone	Ph: (508) 881-0120
Email:	dsmall@ashlandmass.com
Signature:	_____
Date:	_____
Wetland Determination:	Issued by the Ashland Conservation Commission Date: TBA Valid: TBA

2.0 Individual Supervising YOP

Name and Title:	Evan White, Ashland Senior Engineer
Department:	Department of Public Works
Address:	101 Main Street, Ashland, MA 01721
Telephone:	(508) 881-0120
Signature:	_____

3.0 Municipal Department Performing Herbicide Treatment

Either Town staff that are licensed herbicide applicators or a licensed herbicide applicator under contract to the Town of Ashland Department of Public Works will perform the herbicide treatment. Applicators are certified by the Massachusetts Department of Agricultural Resources in the applicator category with at least one holding a Category 40 License:

Certified Applicator(s) & License Number	To Be Determined
Company or Department:	
Address:	
Telephone Number:	
Email:	

The following information is provided as details of the YOP of the Town of Ashland in accordance with the requirements of 333 CMR 11.06 (2):

4.0 Herbicides Proposed

Active Ingredient Use Restrictions	Product Names (EPA #) Registrant
Aminopyralid	Milestone (62719-519) (Product Review) Opensight (62719-597) (Product Review) Corteva Agriscience LLC’
Fosamine Ammonium Lowest Labeled Rate*	Krenite S (42750-247) Albaugh, Inc.
Glyphosate Lowest Labeled Rate for all Glyphosate products	Ranger Pro Herbicide (524-517) Round Up Pro (524-475) Bayer Cropscience LP Glyphomax Plus (62719-322) Corteva Agriscience LLC Rodeo Corteva Agriscience LLC Aquaneat Aquatic Herbicide (228-365) Nu Farm Americas While Rodeo, and Aquaneat all have aquatic uses, approval for their use as sensitive materials does NOT mean that they can be used for aquatic weed control, or directly applied to water, as part of a rights of way management program. Products are subject to the no-spray and limited spray provisions of 333 CMR 11.04.
Imazapyr 3 pints/acre every 3rd year OR 2 pints/acre every other year for all Imazapyr Products	Arsenal (241-346) Arsenal Powerline (241-431) Polaris AC Complete Herbicide (228-570) (Product Review) Polaris Herbicide (228-534) Nu Farm Americas
Indaziflam	Esplanade 200 SC (432-1516) (Product Review) Bayer Environmental Sciences Esplanade 200 SC (101563-144) (Product Review) Envu, Environmental Sciences, U.S, LLC.
Metsulfuron Methyl Lowest Labeled Rate for all Metsulfuron Methyl Products*	Escort XP (432-1549) Bayer CropScience Escort XP (101563-167) Envu, Environmental Sciences, U.S, LLC. Patriot Selective Herbicide, (228-391) Nu Farm Americas
Metsulfuron Methyl Sulfometuron Methyl Lowest Labeled Rate*	Oust Extra (432-1557) Bayer Environmental Science

Active Ingredient Use Restrictions	Product Names (EPA #) Registrant
	Oust Extra, (101563-173) Envu, Environmental Sciences, U.S, LLC.
Paclobutrazol Lowest Labeled Rate*	Cambistat (74779-3) Rainbow Tree care
	Oust XP (432-1552) Bayer CropScience
Sulfometuron Methyl Lowest Labeled Rate for all Sulfometuron Methyl Products*	**Oust XP (101563-168)** Envu, Environmental Sciences, U.S, LLC.
	Spyder Selective Herbicide (228-408) Nu Farm Americas
Triclopyr, Butoxy Ethyl Ester The lowest of the following rates: 1. Between 10 feet and 50 feet of the resource: Lowest labeled rate* or 0.5 pints per acre 2. Between 50 feet and the boundary of the limited spray zone: Lowest labeled rate* or 3 pints per acre	Garlon 4 (62719-40) Corteva Agriscience LLC Garlon 4 Ultra (62719-527) Corteva Agriscience LLC

Complete information for these products is attached, including links to the Safety Data Sheets (SDS). The herbicide fact sheet for the above listed herbicide is attached to and made part of this YOP.

5.0 Herbicide Application Techniques and Alternative Control Procedures

The herbicide will be applied in accordance with the instructions in the attached manufacturer's information. Alternative control procedures, applicable at the designated "No Spray Zones" will consist of hand cutting, mowing, or selective trimming (mechanical). Other alternative controls will include routine street sweeping along with crack and road repairs.

Foliar treatments will be made using ready to use squirt bottles or hand pump backpacks. High volume foliar application may include a truck-mounted hydraulic sprayer. In both cases, the herbicide solution is applied to lightly wet the target plant/ target area. These techniques have few limitations with the exception being reduced effectiveness on tall, high-density target vegetation and will not be used on vegetation over 12 feet in height.

Cut stump treatments will generally be performed to trees greater than 12' tall and resprout. Cut stump treatments consist of mechanical cutting of target species using chain saws immediately followed by herbicide treatment applied with a squirt bottle, a hand pump sprayer, or painted on the freshly cut surface of the stump. The herbicide is limited to freshly cut surface of the remaining stump.

All equipment used for vegetation management programs must be maintained in good working condition, and should be of adequate design and ability to produce the professional quality of work that the Town requires. Because the Town recognizes the vast variety and performance of herbicide application equipment, dictating how that equipment should be calibrated to deliver precise amounts of herbicide to effectively control a host of vegetation conditions is literally impossible. Therefore, the Town will utilize the most appropriate application equipment, calibrated to effectively and legally control target vegetation.

Town staff will ensure that vegetation management activities are conducted in a professional, safe, efficient manner, with special attention directed towards minimal environmental impact. Town staff holding applicator status are qualified, licensed and certified to apply herbicides. "Qualified" means those personnel who have been trained to recognize and identify target and non-target vegetation and are knowledgeable in the safe and proper use of both mechanical and chemical vegetation management techniques. All personnel applying herbicides in Massachusetts must be licensed in the Commonwealth and must work under the on-site supervision of a certified applicator. All applicator personnel will follow all label instructions regarding Personal Protective Equipment (PPE).

Staff applicators and contractor applicators will comply with all applicable federal and state laws and regulations. These include, but are not limited to, applicable OSHA, FIFRA and DOT regulations, 333 CMR 1-15.00, Rights-of-Way Management, Chapter 132B, Chapter 85 of the Acts of 2000 and 321 CMR 10.00 as managed by NHESP.

Herbicides will only be applied in a safe and judicious manner, in compliance with all-applicable State and Federal pesticide regulations.

Applicators will at all times exercise good judgment and common-sense during herbicide treatment activities, and will immediately cease operations if adverse conditions or other circumstances warrant.

Herbicides will NOT be applied during the following adverse weather conditions:

A. During high wind velocity, per 333 CMR 11.03
B. Foliar applications during periods of dense fog, or moderate to heavy rainfall
C. Foliar applications of volatile herbicides during periods of high temperatures (90 plus degrees Fahrenheit) and low humidity
D. Cut Stump applications when deep snow (i.e. 6” plus or ice frozen on stem or stump) prevents adequate coverage of target plants to facilitate acceptable control

Town staff applicators or a representative of the Town must complete daily vegetation management reports that include:

A. Date, name and address of vegetation management staff
B. Identification of site or work area
C. List of crew members
D. Type of equipment and hours used, both mechanical and chemical
E. Method of application and description of target vegetation
F. Amount, concentration, product name of herbicide(s), adjuvants, and dilutants (EPA registration numbers must be on file)
G. Weather conditions
H. Notation of any unusual conditions or incidents, including public inquiries
I. Recording and/or verification of sensitive areas on ROW maps

A Daily Vegetation Management Form is included in the Appendix.

6.0 Target Vegetation

The target vegetation for this YOP will include hazard, detrimental, nuisance and invasive vegetation.

Vegetation management crews will exercise care to ensure that low-growing desirable vegetation and other non-target organisms are not unreasonably affected by the application of herbicides.

Hazard Vegetation

Hazard vegetation represents vegetation that may: obscure sightlines, obscure signs, obscure vehicular movement, block fire hydrants, windfall hazards, and winter shading (increase in use of deicing). This may include woody vegetation along the edges of roadways.

Detrimental Vegetation

Detrimental vegetation includes grasses and woody plants that are destructive or compromise the function of infrastructure including: cracks along the roadway, pavement/bridge joints, medians/traffic islands, and drainage structures/drainageways.

Nuisance Vegetation

This category includes nuisance vegetation that could cause problems to the general public, employees or contractors. Target vegetation in this category is primarily Poison Ivy and other nuisance vegetation within 10 feet of the edge of pavement, bridge abutments, drainage structures and other areas accessible by the public and/or requiring maintenance.

Invasive Vegetation

Invasive species can colonize a space and virtually eliminate the biodiversity of an area. This can result in changes in wildlife due to habitat change, impede natural hydrologic function and cause an overall change in the natural functions of an area. Target vegetation in this category include Japanese Knotweed.

7.0 Description of Methods Used to Flag or Otherwise Designate Sensitive Areas

Sensitive areas as defined by 333 CMR 11.04 are ‘any areas within Rights-of-Way, including No-Spray and Limited Spray Areas, in which public health, environmental or agricultural concerns warrant special protection to further minimize risks of unreasonable adverse effects.’ The Sensitive Areas Restriction Table at the end of this document defines specific sensitive areas and associated buffer zones and treatment restrictions such as limited-spray and no-spray zones.

The attached map identifies ‘Sensitive Areas Not Readily Identifiable in the Field’. With this map and the assistance of the Conservation Commission Agent, sensitive areas will be identified and marked along the ROW prior to any herbicide application. Field methods may include flagging and/or roadway marking (via paint) of start and stop areas.

8.0 Procedures and Locations for Handling, Mixing and Loading of Herbicide Concentrates

If the herbicide is applied by the DPW staff then it will be mixed in the controlled environment at the Ashland DPW Garage located at 20 Ponderosa Road in Ashland, MA.

Although it is expected that all the mixed herbicide will be used, any remaining will be stored at the DPW Garage in accordance with manufacturer's instructions. The absorbent product "Speedi-Dri" will be available for use at the locations of application. If there is a leak in the hose, the pump will be immediately shutoff. Equipment used will be washed at the DPW Garage.

If a licensed subcontractor will apply the herbicide then all mixing and storing will take place at the subcontractor's offsite facility in a controlled environment.

Herbicides will be handled and applied only in accordance with the label instructions. Applicators will strictly adhere to all mandated safety precautions directed towards the public, the applicator and the environment.

9.0 Remedial Plan to Address Spills and Related Accidents

All mixing and loading of herbicides will be conducted at the central facility where the herbicides are stored. Only the amount of herbicide necessary to carry out the vegetation control, based on monitoring results, will be mixed to ensure that there will be no waste and minimize potential problems. The vehicles carrying out the spray operations will be equipped with a bag of absorbent, activated charcoal, leak-proof containers, a broom and a shovel in case of minor spills. A clipboard log of the herbicides on the vehicle will be kept on the vehicle. Herbicide labels and fact sheets will be carried on-site by the applicator.

As soon as any spill is observed, immediate action will be taken to contain the spill and protect the spill area. The cause of the spill must be identified and secured. Spill containment will be accomplished by covering the spill with absorptive clay or other absorptive material or, for large spills, building clay or soil dikes to impede spill progress. Until completely remediated, the spill area will be protected by the placement of barriers and by the delineation of the spill area by crew members. If a fire is involved, care will be taken to avoid breathing fumes from any burning chemicals.

Minor spills will be remedied by soaking up the spill with adsorption clay or other adsorptive material and placing it in leak proof containers, removed from the site and disposed of properly. Dry herbicides, such as granulars, will be swept up or shoveled up directly in leak proof containers for proper disposal. All contaminated soil will be placed in leak proof containers, removed from the site and disposed of properly. Activated charcoal will be incorporated into the soil at the spill location per label instructions. Any minor spill will be reported to the Pesticide Bureau.

Major spills will be handled in a similar manner as minor spills, except in cases where the spill cannot be contained and/or removed by the crew. In this case the MassDEP Incident Response Unit and the Pesticide Bureau must be contacted.

Emergency first responders (including but not limited to fire and police) will be immediately notified of a major spill and/or any size incident deemed a potential risk to public health, safety and the environment.

MassDEP will be contacted when there is a spill of a regulated quantity, regardless of major or minor spill status and in accordance with 310 CMR 40.0000 Massachusetts Contingency Plan.

10.0 Emergency Contacts

In the event of a spill, information on safety precautions and clean up procedures may be gathered from the following sources:

Table 1. Emergency Resources

Resource	Location/Phone #
Herbicide Label	Approved YOP
Herbicide Safety Data Sheet (SDS)	Approved YOP
Herbicide Manufacturer • Corteva Agriscience (formerly Dow/Dupont) • NuFarm • Bayer	 (800) 992-5994 (877) 325-1840 (866)-99-BAYER
MDAR, Division of Crop & Pest Services Clayton Edwards	(617) 626-1700
Massachusetts Department of Environmental Protection Emergency Response	(888) 304-1133
Department of Public Health Environmental Toxicology Program	(617) 624-5286
Massachusetts Poison Control Center 24-Hour Hotline	(800) 222-1222
Town of Ashland Department of Public Works	(508) 881-0120
Town of Ashland Fire Department	(508) 881-2323 – non-emergency or 911
Town of Ashland Police Department	(508) 881-1212 – non-emergency or 911
Town of Ashland Health Department	(508) 532-7922
Chem-Trec	(800) 262-8200
National Pesticide Information Center	(800) 858-7378
National Animal Poison Control Center	(800) 426-4435

TABLE 2. CONTROL STRATEGIES FOR SENSITIVE AREAS

Sensitive Area Restrictions (333 CMR 11.04)

Sensitive Area	Minimum Buffer Zone (feet)	Control Method	Time Restriction Code
Public Ground Water Supplies	400'	Mechanical Only	None
Primary Recharge Area	Designated buffer zone or 1/2-mile radius	Mechanical, Recommended Herbicides*	1
Public Surface Water Supplies (Class A & Class B)	100'	Mechanical Only	None
	100'-400'	Recommended Herbicides	1
Tributary to Class A Water Source, within 400' upstream of water source	100'	Mechanical Only	None
	100'-400'	Recommended Herbicides	1
Tributary to Class A Water Source, greater than 400' upstream of water source	10'	Mechanical Only	None
	10'-200'	Recommended Herbicides	1
Class B Drinking Water Intake, within 400' upstream of intake	100'	Mechanical Only	None
	100'-200'	Recommended Herbicides	1
Private Drinking Water Supplies	50'	Mechanical Only	None
	50'-100'	Recommended Herbicides	2
Surface Waters	10'	Mechanical Only	None
	10'-100'	Recommended Herbicides	2
Rivers	10' from mean annual high-water line	Mechanical Only	None
	10'-200'	Recommended Herbicides	2
Wetlands	100' (treatment in wetlands permitted up to 10' of standing water)*+	Low-pressure Foliar, CST, Basal Recommended Herbicides	1
Habitated Areas	100' (for high-pressure foliar only)	Recommended Herbicides	2
Agricultural Area (Crops, Fruits, Pastures)	100' (for high-pressure foliar only)	Recommended Herbicides	2
Certified Vernal Pools	10'	Mechanical Only	None
Certified Vernal Pool Habitat	10'-outer boundary of habitat	As recommended by NHESP in their permit process, no treatment without written permission.	
Priority Habitat	As recommended by NHESP in their permit process, no treatment without written permission.		

Restriction Code #1: A minimum of twenty-four months shall elapse between applications.
Restriction Code #2: A minimum of twelve months shall elapse between applications.
*Massachusetts recommended herbicides for sensitive sites.

Appendix

Approved Herbicide Fact Sheets List and SDS Sheets

Please use the provided links to access the fact sheet lists and SDS sheets

Aminopyralid

Fact Sheet: https://www.mass.gov/doc/aminopyralid-2016/download

Milestone (62719-519) SDS

Opensight (62719-597) SDS

Glyphosate

Fact Sheet: https://www.mass.gov/doc/glyphosate-factsheet-2022-updated-may13/download

Ranger Pro Herbicide (524-517) SDS

Round Up Pro (524-475) SDS

Glyphomax Plus (62719-322) SDS

Rodeo SDS

Aquaneat Aquatic Herbicide (228-365) SDS

Indaziflam

https://www.mass.gov/doc/indaziflam-2022/download

Arsenal (241-346) SDS

Imazapyr

https://www.mass.gov/doc/imazapyr-2011pdf/download

Esplanade 200 SC (101563-144)

Export XP (432-1549)

Metsulfuron Methyl

https://www.mass.gov/doc/metsulfuron-methyl-2011pdf/download

Escort XP (101563-167)

Patriot Selective Herbicide, (228-391)

Oust Extra (432-1557)

Sulfometuron Methyl

https://www.mass.gov/doc/sulfometuron-methyl-2011pdf/download

Oust Extra, (101563-173)

Spyder Selective Herbicide (228-408)

Fosamine Ammonium Factsheet

https://www.mass.gov/doc/fosamine-ammonium-2011pdf/download

Triclopyr Factsheet

https://www.mass.gov/doc/triclopyr-2011pdf/download

Paclobutrazol Factsheet

https://www.mass.gov/doc/paclobutrazol-review-jan-2012pdf/download

Cambistat (74779-3)

Oust XP (432-1552)

Summary of Aminopyralid Toxicity and Fate for Application to Sensitive Areas of Rights-of-Way

The following summary addresses use of the herbicide aminopyralid in Sensitive Areas of Rights-of-Way in Massachusetts. The review was jointly conducted by the Massachusetts Department of Environmental Protection (MassDEP) Office of Research and Standards (ORS) and the Massachusetts Department of Agricultural Resources (DAR) in accordance with the cooperative agreement issued between the two agencies in 1987 and updated in 2011 pursuant to the provisions of Section 4(1)(E) of 333 CMR 11.00 Rights-of-Way Management Regulations.

The conclusions summarized in this memo are based upon several sources of information, including a comprehensive review of this herbicide by the USDA Forest Service (Durkin 2007), scientific documents contained in the US Environmental Protection Agency (EPA) docket of information for aminopyralid to support pesticide registration decisions and the results of literature searches for recent pertinent studies on this chemical. As aminopyralid is a relatively new product, very little primary information was found in the literature that was pertinent to the scope of this review and therefore the review was primarily based on information provided by the secondary summary documents described above. The purpose of this review is to ascertain the suitability of this product for use within sensitive areas of rights-of-way, based upon consideration of available information on the potential toxicity of the active ingredient aminopyralid as well as its fate and transport in the environment.

Aminopyralid (2-pyridine carboxylic acid, 4-amino-3,6-dichloro-2-pyridine carboxylic acid) is a pyridine carboxylic acid herbicide manufactured by Dow AgroSciences LLC (DAS) for use in controlling annual and perennial broadleaf weeds. At the time of this active ingredient review, two end-use products containing aminopyralid were requested: Milestone (EPA Reg. No. 62719-519) and OpenSight (EPA Reg. No. 62719-597). Additional details on the evaluation of the products can be found in separate review documents.[1]

Aminopyralid is structurally similar to other pyridine carboxylic acid herbicides that preceded it in development, including clopyralid, picloram and triclopyr. Technical grade picloram and clopyralid contain the carcinogen hexachlorobenzene as well as other carcinogenic chlorinated benzenes as impurities that are byproducts of their synthesis process. According to DAS, the manufacturing process for aminopyralid does not produce these byproducts (John Jachetta, DAS product manager for aminopyralid as cited in Durkin, 2007). EPA has labeled aminopyralid a "reduced risk pesticide" that has a favorable human health toxicity profile when compared to the registered alternatives, because it has a lower application rate, which should alleviate the need for repeat applications and thus result in a lower overall amount used.

Similar to other pyridine carboxylic acids, aminopyralid is a synthetic analogue of an auxin, a plant hormone that regulates development, growth and other plant functions. Though the specific mode of action of these compounds is not fully known, they produce effects on the plant including alterations in

[1] Product review of Milestone Herbicide; Product Review of Opensight Herbicide

cell wall elasticity and gene expression, and non-productive tissue growth that results in leaf curl and disruption of the plant phloem, interfering with transport of nutrients and causing death in days to weeks.

Summary of fate and transport:

Aminopyralid is generally very persistent in the environment. Under favorable light conditions, it can rapidly photodegrade in shallow, clear water (though not in murky deeper water), with a half-life of 0.6 days. It photodegrades slowly in soil, with a half-life of about 72.2 days. It is stable to microbial degradation in sediment and water systems. In aerobic soils, it is metabolized at a moderate rate depending on the type of soil, with a half-life range of about 31.5 days to 193 days in eight soils[2]. It is expected to be stable in anaerobic soils (USEPA, 2014).

Under environmental conditions and pH, 99.9% of aminopyralid will dissociate to its anionic form, which contributes to its high solubility, lack of volatility and very low adsorption to soils. As a result, aminopyralid partitions to water and is expected to have high mobility in most soils. The major route of dissipation of aminopyralid from soil is through runoff and leaching.

Once aminopyralid enters surface water, any residue that is not subject to photolysis will persist and be mobile in aquatic environments. Aquatic field dissipation studies in treated ponds showed half-lives in the range of 10.8 to 14.6 days. Any part of aminopyralid applied to terrestrial vegetation that reaches the soil has a high potential to run off into surface water or leach into the soil profile and groundwater. Once aminopyralid reaches anaerobic depths in soil, degradation will dramatically slow and only its high mobility will determine the rate at which it will contaminate groundwater. Field dissipation in bare ground studies showed dissipation half-lives in the range of 9 to 54 days and leaching depths in the range of 6 to 36 inches. The potential for groundwater contamination with aminopyralid is expected to be higher in areas with shallow groundwater (because there is less depth to travel before reaching groundwater) or when rain occurs soon after application. Additional information on the expected concentrations in surface water and groundwater following the terrestrial applications in rights-of-way is available in the companion document to this review.

[2] Recent assessments by USEPA (2014) and the European Union (EFSA, 2013) provide updated information for aerobic soil metabolism and soil binding parameter values of aminopyralid. USEPA (2014) considered the data from eight soils. The soil half-life values ranged from 31 to 193 days, with an average of 103.7 days. The soil-water partitioning constant (K_D) values ranged from 0.03 to 0.29 mL/g for soils with pH values of 6.1 to 7.8; K_D values of acid soils were in the range of 0.15 to 0.72 mL/g. The K_{OC} values for soils with near-neutral pH values were in the range of 1.05 to 7.54 mL/g and for acidic soils the values were in the range of 19.95 to 24.3 mL/g. In general, K_{OC}-values increase with decreasing pH. USEPA (2014) indicated that these data on soil half-life and soil binding (soil-water distribution coefficient data) are acceptable for use in exposure modeling and risk assessment.
In addition to the USEPA assessment, aquatic exposure modeling conducted as part of a European risk assessment (EFSA, 2013) was reviewed to provide additional data and information. The model input value for soil half-life geometric mean of 54.8 days was lower than the values used in the SERA risk assessment (Durkin, 2007) and the values used by USEPA. The model input value for soil binding parameter (mean $K_{F,OC}$ of 6.64 mL/g) was within the range of values used in the other modeling efforts reviewed above.

The only potentially major degradation products of aminopyralid are formed during aqueous photolysis and include two small amino acid analogs, i.e., malonamic acid and oxamic acid, along with four unidentified acid amides of 2-3 carbons in length. EPA concluded that neither of the two identified compounds would be of concern as they are expected to be readily metabolized following uptake and/or rapidly excreted without any significant biological effects. In addition, none of these compounds are expected to be produced to any great extent as aqueous photolysis only occurs up to the depth that sunlight penetrates a water body. Only carbon dioxide and some non-extractable residues were found in amounts over 10% of the applied study residue in all other laboratory degradation studies of aminopyralid, at maximums of 76.2% in aerobic soil metabolism and 15% in aerobic aquatic metabolism.

Summary of Toxicity and Risk Assessment:

Available toxicity information reviewed by the secondary sources cited above all indicate that aminopyralid at environmentally relevant concentrations has low potential toxicity to humans, as well as terrestrial animals and aquatic organisms. This finding is consistent with its mode of action, which is specific to plant biology. A number of systemic mammalian studies as well as aquatic ecotoxicity studies indicate that exposure concentrations of aminopyralid associated with herbicide applications are well below concentrations of concern for these receptors.

In terms of mammalian effects, the weight of evidence indicates that aminopyralid does not produce significant systemic effects. The effects most often seen following exposure to aminopyralid are on the gastrointestinal tract after oral exposure, with cecal effects in rats and stomach effects in dogs and rabbits. In rats, the typical effect is cecal enlargement. Given that cecal enlargement is typically seen with poorly absorbed osmotically active compounds, this effect is categorized by a number of investigators as an adaptive change and/or not toxicologically significant. The significance of cecal effects to humans, which only have a vestigial trace of this organ, is also unclear. The USDA Forest Service considers the effects on the gastrointestinal system as portal of entry effects. The differences in effects are attributed to differences in species anatomy and methods of exposure (i.e., gavage vs. dietary). Another somewhat notable effect in mammals includes the results of an acute oral toxicity study in rats in which bilateral cloudiness and lacrimation of eyes was seen in all rats after one day but not on subsequent days. Cloudiness of eyes is an unusual effect that has not been seen in any other aminopyralid study. The significance of these findings is unclear. Finally, in one developmental study, incoordination in several adult female rabbits was noted but this effect was rapidly reversible.

EPA developed a chronic Reference Dose (RfD) of 0.5 mg a.e.[3]/kg/day for aminopyralid for the general population derived based on a No Observed Adverse Effect Level (NOAEL) of 50 mg a.e./kg/day from a 24-month feeding study in rats. The endpoint, increase in cecal weights at 500 mg a.e./kg/day, may have very little relevance to potential effects in humans. However, the RfD is based on the most sensitive effect for the most sensitive species from the available database for aminopyralid. EPA also derived a Human Health Benchmark for Pesticide (HHBP) concentration of 3500 ug/L (ppb) from this chronic RfD

[3] Because aminopyralid dissociates from its acid form to its anionic form in the environment, aminopyralid application rates and concentrations are reported as "acid equivalents" (a.e.), instead of "active ingredients" (a.i.) because the acid part of the active ingredient salt is the herbicidally active component.

based upon a 70 kg adult who drinks 2 L/day of water and incorporating a Relative Source Contribution (RSC) factor of 20%.

For short-term/intermediate exposures, EPA developed an acute RfD of 1.0 mg a.e./kg/day derived based on a NOAEL of 104 mg a.e./kg/day from a developmental gavage study in rabbits in which decreased maternal food consumption and body weight as well as spontaneous abortion (in one rabbit) and decreased fetal weights were seen at higher doses.

A comparison of predicted short and long-term exposure to aminopyralid following application indicates that exposures are substantially below the above acute and chronic criteria.

Though the potential for aminopyralid to contaminate groundwater is high due to aminopyralid's high solubility and prolonged half-life in soil, both EPA and the U.S. Forest Service concluded that predicted short and long-term concentrations of aminopyralid in groundwater are substantially below concentrations of health concern for people using groundwater as a source of drinking water.

In terms of ecological effects, it appears that birds are more sensitive to aminopyralid administered through gavage than dietary exposure. A series of ecological benchmark toxicity concentrations were developed by both EPA and the US Forest Service for various terrestrial and aquatic wildlife. Though there were some differences in some of these values between the two agencies, the evaluations conducted by both agencies point to the same conclusion, that there is no indication from the available data that aminopyralid will adversely affect mammals, birds, fish, aquatic and terrestrial invertebrates, terrestrial microorganisms and amphibians.

A couple of ecological data gaps remain in the data submitted by the manufacturer of this compound to the U.S. Environmental Protection Agency (USEPA). These include a cyanobacteria growth study, an early life stage study in fathead minnows and an invertebrate lifecycle study in mysid shrimp. Additional information on data that are needed to address uncertainties in risk assessments is available in documents that were issued with the Registration Review of aminopyralid. The Registration Review of aminopyralid was initiated in 2013 and is scheduled to be completed in 2020. Information and notices related to this review will be available in the docket (USEPA, 2013).

An additional quantitative comparison of modeled concentrations of aminopyralid in surface water and groundwater following land application in rights-of-way areas was done by DAR to available ecological and human health benchmarks. This analysis indicated that projected water concentrations resulting from application of aminopyralid are well below concentrations of concern for ecological receptors in surface water as well as for humans who use these waters as sources of drinking water. For additional details on this evaluation as well as on the modeling conducted, please see the companion document to this review, entitled "Exposure Assessment of Aminopyralid in Surface and Ground Water: Review of Modeling Input Parameter, Refined Modeling and Comparison with Benchmarks."

Plants:

Aminopyralid's auxinic mode of action renders it toxic to all terrestrial (dicot) broadleaf plants. It is generally not toxic to terrestrial (monocot) grasses. While aquatic macrophytes have been shown to be more sensitive to aminopyralid than aquatic organisms, this herbicide is generally not toxic to aquatic macrophytes and algae.

Given that aminopyralid has an auxinic mode of action that can affect all terrestrial broadleaf plants, the potential impact to non-target broadleaf plants, particularly plants that are endangered species, is seen as the greatest concern for this herbicide. In addition, effects on non-target plants that might not be endangered species but which might serve as a food source for endangered animal species would be of concern.

An important consideration with this compound is that aminopyralid ingested by animals in grasses and other vegetation is excreted largely unchanged. As has been found with two of its predecessor compounds, (i.e., clopyralid, and picloram), use of manure from domesticated animals (that have ingested aminopyralid-treated grasses and vegetation) as compost in gardens can have detrimental effects to sensitive broadleaf plants, including plants in the nightshade family such as potatoes, tomatoes, and legumes. The aminopyralid product label warns that manure from animals that have grazed on aminopyralid-treated vegetation within the previous three days should not be used on land used for growing susceptible broadleaf plants. The three-day warning refers to the time it takes for consumed vegetation containing aminopyralid residues to pass through grazing animals. While this warning does not directly apply to application of aminopyralid on rangeland, it should be considered in scenarios where there is the potential for range vegetation to enter the garden compost stream.

Conclusions/Recommendations:

The information contained in the secondary documents from both EPA and the US Forest Service that were reviewed for this evaluation consistently present the same profile and conclusions on the toxicity, fate and transport of this herbicide. No conflicting information was identified in the literature. In addition, supplemental modeling conducted by DAR for this review consistently point to the same conclusions as those reached by EPA, the US Forest Service and others. Modeled concentrations of aminopyralid in environmental media following application as specified in product labels are well below toxicity levels of concern for humans, as well as terrestrial and aquatic wildlife.

Sensitive non-target plant species have been identified as the organisms of concern. Given that herbicides are designed to control plants, this is not surprising. This information, coupled with the fact that aminopyralid is very mobile and persistent in the environment strongly suggests that application of aminopyralid should be targeted as much as possible to avoid impacts on non-target plants. Measures that minimize drift should be used in applying this product. In addition, as with any application, a preliminary field survey should be conducted prior to application to identify any plants on the endangered species list and/or any other plant species that are important to that ecosystem.

Based upon the available database for aminopyralid, use of this herbicide in sensitive areas of rights-of-ways should be acceptable if it is applied in a manner that is consistent with the product label, the above recommendations and the Massachusetts Sensitive Areas of Rights-of-Way Regulations.

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USEPA, 2014. Registration Review; Preliminary problem formulation for environmental fate, ecological risk, endangered species, and human health drinking water exposure assessments for aminopyralid. USEPA, Environmental Fate and Effects Division. February 12, 2014. Accessed at: http://www.regulations.gov/#!documentDetail;D=EPA-HQ-OPP-2013-0749-0011.

Exposure Assessment of Aminopyralid in Surface and Ground Water: Review of Modeling Input Parameter, Refined Modeling and Comparison with Benchmarks

1. Introduction

Aquatic exposure modeling has been used to estimate aminopyralid residue concentrations in surface water and ground water to support human health and ecological risk assessments. The USDA Forest Service document, "Aminopyralid-Human Health and Ecological Risk Assessment-FINAL REPORT", prepared by Syracuse Environmental Research Associates, Inc. (SERA) (Durkin 2007) describes the modeling that was used to estimate the concentrations of aminopyralid that may occur in surface and ground water. The risk assessment also reviews environmental fate input parameters and summarizes results from other modeling efforts conducted by USEPA and DOW AgroSciences (DAS).

The present document reviews these modeling data and also provides the results of additional modeling conducted by DAR, utilizing more recent modeling information and environmental fate input parameters, to complement and refine existing modeling results. All of these modeled concentrations in surface and ground water were assessed by comparing them to benchmark toxicity values for aquatic life and human health established by USEPA.

2. Review of Modeling Data in SERA Risk Assessment

The SERA risk assessment (Durkin 2007) notes that modeling results are sensitive to the input parameter value for soil half-life. The range of input values for aerobic soil metabolism half-life used in the various modeling efforts is related to the limitations and uncertainty in the data that were available for this parameter at the time modeling was conducted. SERA used a slightly higher value for half-life time of 343 days compared to 310.5 days by USEPA. The value used by USEPA was based on a single study result of 103.5 days. USEPA multiplied that half-life value by 3 to account for the uncertainty associated with using only a single study result.

SERA notes that the soil binding parameter (i.e., soil-water partitioning coefficients K_{OC} and K_D) is variable and not closely related to organic carbon content of the soil. Model input values for this parameter used in GLEAMS modeling were refined by using specific values associated with the type of soil. Values used for K_{OC} ranged from 0.87 in clay to 8.91 mL/g in loam; K_D values ranged from 0.39 in sand to 0.63 mL/g in clay. The parameter values used in modeling by USEPA was K_D of 0.03 mL/g and DOW AgroSciences used a K_{OC} value of 0.81mL/g.

The input parameter values used in the modeling described in SERA were considered to be the most conservative and resulted in the highest estimates for concentrations in surface water. The modeling results for selected scenarios that are most representative for Massachusetts are

included in Table 1 for comparison with other modeling results. The SERA report notes that the central estimate for surface water exposure based on GLEAMS modeling is similar to the value estimated by USEPA based on the PRZM/EXAMS modeling. The GLEAMS modeling data were the basis for the concentrations used in the SERA risk assessment.

SERA did not conduct modeling of concentrations in groundwater, but considered groundwater modeling results from USEPA and DAS (see also Table 2). The drinking water exposure assessment described by SERA is based on modeling results for surface water. As noted in SERA, modeling results for concentrations in surface water are higher than modeling results in groundwater.

3. Recent Information Related to Environmental Fate Characteristics and Model Input Values

As noted in the section above, the model input values for soil half-life and soil binding were found to be important parameters in modeling of aquatic exposure. Recent assessments by USEPA (2014A) and the European Union (EFSA, 2013) provide updated information for these properties of aminopyralid.

USEPA (2014A) considered the data from eight soils. The soil half-life values ranged from 31 to 193 days, with an average of 103.7 days. The K_D values ranged from 0.03 to 0.29 mL/g for soils with pH values of 6.1 to 7.8; K_D values of acid soils were in the range of 0.15 to 0.72 mL/g. The K_{OC} values for soils with near-neutral pH values were in the range of 1.05 to 7.54 mL/g and for acidic soils the values were in the range of 19.95 to 24.3 mL/g. In general, K_{OC} values increase with decreasing pH. USEPA (2014A) indicated that these data on soil half-life and soil binding (soil-water distribution coefficient data) are acceptable for use in exposure modeling and risk assessment.

In addition to the USEPA assessment, aquatic exposure modeling conducted as part of a European risk assessment (EFSA, 2013) was reviewed to provide additional data and information. The model input value for soil half-life geometric mean of 54.8 days was lower than the values used in the SERA risk assessment and the values used by USEPA. The model input value for soil binding parameter (mean K_{FOC} of 6.64 mL/g) was within the range of values used in the other modeling efforts reviewed above. The EFSA modeling results are included in Table 1.

Consideration of the data from the recent USEPA and EFSA assessments indicates that the input parameter values used in the GLEAMS modeling described in the SERA risk assessment were conservative values. In the refined modeling described below, DAR considered the recent information with the selection of input parameter values.

4. Additional Aquatic Exposure Modeling

For the purpose of this review, DAR conducted additional modeling using updated input parameter values to complement the existing data with refined exposure modeling results. The modeling conducted by DAR was done with recently released EPA water exposure models (see Appendix 1 and 2.

The model input parameter values for soil half-life and soil binding were based on the environmental fate information and data provided in the recent assessment by USEPA (2014A). The average value for soil half-life of 103.5 day and the lowest value for soil binding parameter K_D of 0.03 mL/g were used for model input. The application rate was the maximum labeled rate of 0.11 lbs of aminopyralid per acre. For surface water modeling, the watershed scenarios modeled were the EPA standard pond, the EPA index reservoir and a custom small pond scenario. Further details on model input can be found in Appendix 1.

The results of DAR modeling are presented below and compared with the modeling data summarized in the SERA risk assessment (Durkin, 2007) and EFSA (2013).

4.1. Surface Water Modeling

Additional modeling of surface water concentrations was conducted to complement the existing modeling data that were generated with EPA standard scenarios using modeling data that are more representative for Massachusetts ROW. The model scenario that was developed for surface water exposure assessment of herbicide components in ROW areas (Wijnja, 2010), was used in the modeling here with the latest version of the EPA surface water exposure model (see Appendix 1). The latest version of the EPA surface water exposure model also allows the modeling of a custom watershed scenario. For the purpose of this assessment, DAR developed a custom small pond scenario. More detailed information on the model input and modeling results can be found in Appendix 1.

The modeled surface water concentrations are summarized and compared with other modeling results in Table 1. To facilitate comparison of modeling results, results from other modeling were scaled, if necessary, to the value representative of an application rate of 0.11 lbs/acre.

The modeling results generated with the MA-specific ROW scenario by DAR show the highest concentrations for the custom small pond scenario. These higher concentrations are attributed to the smaller dimensions of the watershed, including a shallower pond, compared to the EPA standard pond and reservoir.

Comparison of the most conservative refined modeling results (ROW scenario and custom small pond) with the concentrations used in the SERA risk assessment indicate that the results are similar to the central values used in SERA risk assessment.

The results for the MA-specific ROW scenario with standard pond and index reservoir watersheds are lower than the concentration generated by EPA modeling for the same type of watersheds. This is likely the result of difference in the land use scenarios (ROW versus range land or a generic scenario) and weather input data. The results for the ROW scenario and custom small pond watershed resulted in higher concentrations compared to the EPA standard pond and EPA Index Reservoir water bodies.

Table 1. Modeling results for surface water concentrations of aminopyralid. The results are representative of an application rate of 0.11 lbs/acre.

Agency/Org.	Model/Scenario	Concentration (µg/L or ppb)		Source/Notes
		Peak	Longer-term	
DAR	MA ROW scenario with:			
	SWCC, EPA Standard Pond	0.612	0.477	Appendix 1A
	SWCC, EPA Index Reservoir	1.93	1.45	Appendix 1B
	SWCC, Custom Small Pond	12.1	3.32	Appendix 1C
SERA				Durkin, 2007:
	GLEAMS Standard, Pond	3.34 - 14.3	2.21 - 7.76	Table 6; 50 inch rainfall and rate of 0.11 lbs/acre
	GLEAMS-Driver, Pond	8.8 - 34.1	4.4 – 19.8	Table 9, 10; average rainfall and for rate of 0.11 lbs/acre
EPA				Durkin, 2007:
	PRZM/EXAMS, Reservoir	10.01	1.936	Table 11, rate of 0.11 lbs/acre
	GENEEC, EPA Standard Pond	6.38	5.39	″
DOW				Durkin, 2007:
	GENEEC	6.16	3.96	Table 11; rate of 0.11 lbs/acre
				″
SERA	Conc. used for Risk Assess.			Durkin, 2007:
	Central	11	4.4	Table 12, rate of 0.11 lbs/acre
	Lower	0.23	0.11	″
	Upper	66.0	28.6	″
EFSA				EFSA, 2013: Annex A
	FOCUS Step 1	20.4	20.1	Screening-level Assessment
	FOCUS Step 3	0.052	0.049	Late Spring Application, Pond D4 Scenario
	FOCUS Step 3	0.332	0.042	Late Spring Application, Stream Scenario D4

DAR modeling with ROW-scenario also evaluated the sensitivity of the results for the input value of the soil aerobic metabolism half-life. The model results did not change significantly for simulations with a soil aerobic metabolism half-life of 310.5 d compared to 103.5 d (Table 1 in Appendices 1A, 1B and 1C). The 310.5 d value was used in earlier modeling by EPA (see Section 2); the value of 103.5 d was more recently recommended for use in risk assessment (see Section 3).

Modeling data generated by the European EFSA agency show screening-level assessment concentrations that are higher than the DAR custom pond values, but concentrations for specific scenarios are lower than modeling results for all other scenarios included in Table 1.

4.2. Groundwater modeling results

Additional groundwater modeling was conducted with EPA models SCIGROW and PRZM-GW (Water Models | Pesticides | US EPA: http://www.epa.gov/pesticide-science-and-assessing-pesticide-risks/about-water-exposure-models-used-pesticide).

SCI-GROW (Screening Concentration in Groundwater) as a screening-level tool to estimate drinking water exposure concentrations in groundwater resulting from pesticide use. As a screening tool, SCI-GROW provides conservative estimates of pesticides in groundwater. It is a generic model that provides peak estimates of compound concentrations in groundwater based on a given application rate, number of applications, and standard environmental fate parameters of soil aerobic half-life and soil binding constant.

The PRZM-GW (Pesticide Root Zone Model – Ground Water) model has the capability to consider variability in leaching potential of different soils, weather (including rainfall), cumulative yearly applications or depth to aquifer. The conceptual model is based on a rural drinking water well beneath an agricultural field (a high pesticide use area), which draws water from an unconfined, high water-table aquifer. Processes included in the conceptual model that influence pesticide transport through the soil profile include water flow, chemical specific dissipation and transportation parameters (i.e., degradation and sorption), and crop specific factors, including transpiration, pesticide interception and management practices.

Six different scenarios were developed for the PRZM-GW model. The modeling for the review presented here used was based on the Delmarva Sweet Corn - Evesboro Loamy Sand scenario. Delmarva Peninsula sweet corn scenario is one of the six PRZM-GW standard scenarios that fall within regions where groundwater is highly susceptible to nitrate contamination. The six scenarios are expected to provide reasonable upper bound estimates for pesticide concentrations for vulnerable groundwater sources (USEPA, 2015).

The Delmarva Corn scenario most closely represents the Virginia Coastal Plain spatially and characteristically. In the Delmarva Corn scenario, the vadose zone ends and the aquifer begins 9 meters (29.5 feet) below the land surface. It has been reported that 26 of 29 Virginia Coastal Plain counties have at least one domestic well with a depth to the bottom of the well screen of 30 feet or less. Using this example, it follows that modeling with PRZM-GW provides estimated drinking water concentrations (EDWCs) that represent a subset of a broadly distributed population relying on shallow, private drinking water wells.

The scenario characteristics for vegetation were adjusted to be representative of ROW vegetation. Weather input data were representative for Eastern Massachusetts. This model simulation can be considered to be representative of behavior at a vulnerable site given the loamy sand soil profile and the absence of a buffer zone around the well.

Details on the SCI-GROW and PRZM-GW modeling can be found in Appendices 2A and 2B. The modeling results are summarized in Table 2 and compared with the other ground water modeling data.

Table 2. Comparison of groundwater modeling results for concentrations of aminopyralid for maximum application rate of 0.11 lbs/acre.

Agency/Org.	Model/Scenario	Concentration (µg/L or ppb)		Source/Notes
		Peak	Longer-term	
DAR	SCIGROW	5.17		Appendix 2A ; K_{OC}:1.05; soil half-life: 103.5 d
	PRZM-GW	12.6	10.5	Appendix 2B; K_{OC}:1.05; soil half-life: 103.5 d
EPA				Durkin, 2007: Table 11
	SCI-GROW	0.627		Application rate of 0.11 lbs/acre; K_{OC}: 1.05; soil half-life: 38.7 d
DOW				Durkin, 2007:
	SCI-GROW	1.65		Table 11, for application rate of 0.11 lbs/A; K_{OC} of 7.1 and soil half-life of 88.6 d
	SCI-GROW	0.121		Rate: 0.11 lbs/acre; K_{OC} of 7.1; soil half-life of 30 d
				″
EFSA				EFSA, 2013: Annex A
	FOCUS PEARL	0.116		Annual application of 0.053 lbs ai/acre; field dissipation half-life of 14.1 d; K_{foc}: 5.14 mL/g

Modeling results from DAR show the highest concentrations due to the use of conservative values for soil adsorption constant and soil half-life input parameters. These input values are the most recent values that EPA recommends for use in risk assessment (see section 3).

It should be noted that the soil defined in the Delmarva Sweet Corn - Evesboro Loamy Sand scenario represents a sandy soil profile with relatively low organic matter content. Such a soil profile is considered to favor leaching of substances into the profile. In the model scenario, the soil is definedto have low organic matter (highest is 0.52 % organic carbon in top layers and 0.1 – 0.20 % in deeper soil layers). Percentage of sand in the soil layers is greater than 90 % and clay content is between 2 and 5%. These soil particle size distributions are similar to values for sandy soils that occur in southeastern Massachusetts and Cape Cod. For example, the Carver soils are sandy soils with clay content of 1 to 5 % and organic matter content in the ranges of 0.1 – 1.0 %. (Soil Survey for Barnstable County: http://nesoil.com/barnstable/index.htm).

4.3. Groundwater Monitoring Data

The ground water modeling results can further be evaluated by considering results from monitoring studies. At the time of this review, two studies were located that were publicly available (online) that included aminopyralid as a target analyte.

A groundwater monitoring study conducted in Wyoming by the US Geological Survey (USGS) included aminopyralid as a target analyte. Aminopyralid was not detected (Eddy-Miller et al., 2013).

In a monitoring study in the Bitterroot Valley, MT, aminopyralid was detected at a level of 0.1 µg/L in one of 46 samples from 23 wells (Schmidt and Mulder, 2009).

USGS pesticide use data indicate that there was substantial use of this herbicide in both Montana and Wyoming (Fig. 1).

These monitoring study results show low detection frequencies of aminopyralid in areas where this herbicide was used. When detected, the level was much lower than the ground water modeling data presented in section 4.2.

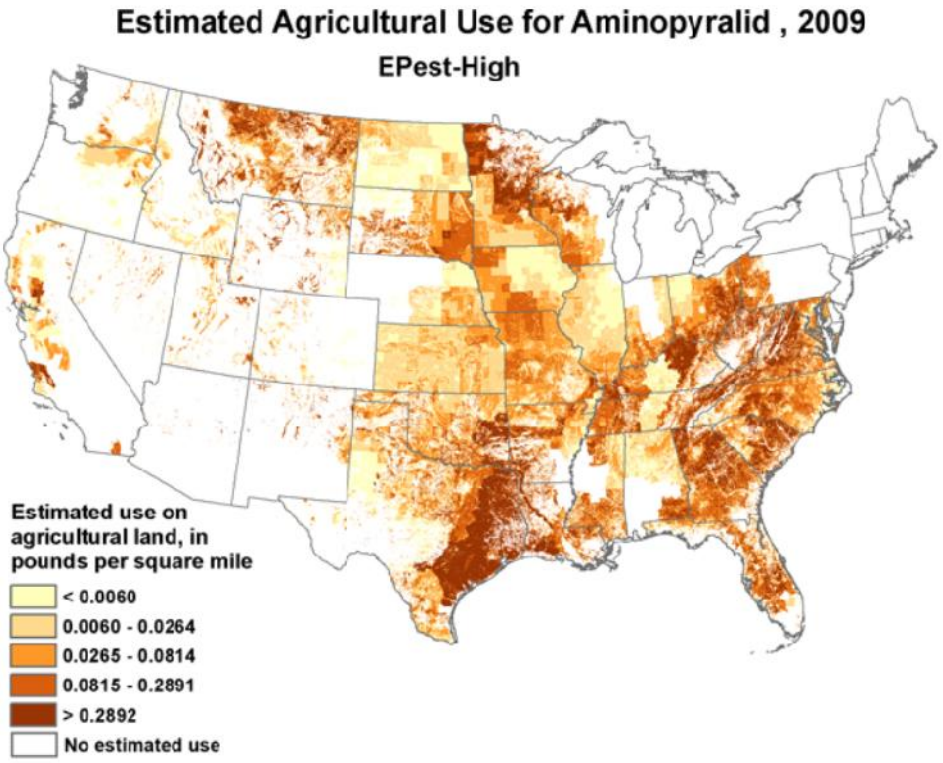

Figure 1 Estimated Agricultural use of Aminopyralid in the US during 2009. Accessed at: USGS NAWQA: The Pesticide National Synthesis Project

5. Comparison of Modeled Concentrations with Aquatic Life and Human Health Benchmarks

EPA developed benchmarks that can assist with the assessment of monitoring and modeling data. Surface water modeling data were compared with aquatic life bench mark to assess the potential for ecological effects in aquatic systems.

Comparison of modeled surface water concentrations with Aquatic Life Benchmarks for Aminopyralid (Table 3) can be helpful to assess risk to aquatic life (USEPA, 2014B). Comparison of the modeled concentrations in Table 1 (DAR data for peak 0.612 – 12.1 µg/L and chronic 0.477 to 3.32 µg/L) with the benchmarks in Table 3 shows levels well below benchmark values. This comparison indicates minimal risk to aquatic life.

Table 3. Aquatic life benchmarks for aminopyralid

Species	Acute (µg/L or ppb)	Chronic (µg/L or ppb)
Fish	>5,000	1360
Invertebrates	>49,300	10200
Non-vascular plants	18,000	
Vascular plants	>88,000	

Comparison of the modeled concentrations with human health benchmark values for aminopyralid can further assist with assessment of potential for human health effects.

The chronic or life-time human health benchmark (HHBM) value for aminopyralid is 3500 ppb (US EPA, 2014C). An acute HHBM value has not been established. The EPA risk assessment notes that aminopyralid is of low acute toxicity and therefore no acute reference dose was identified for any population.

Comparison of the modeled aminopyralid concentrations in groundwater and the HHBM indicates that there is no concern for effects on human health from drinking water containing residues of aminopyralid following application per label specifications..

6. References

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Appendix 1A: Summary of Water Modeling of Aminopyralid and the USEPA Standard Pond

Estimated Environmental Concentrations for aminopyralid are presented in Table 1 for the USEPA standard pond with the RightOfWay_MA_PAX field scenario. A graphical presentation of the year-to-year peaks is presented in Figure 1. These values were generated with the Surface Water Concentration Calculator (SWCC Version 1.106) (Water Models | Pesticides | US EPA)[1]. The SWCC model estimates pesticide concentrations in water bodies that result from pesticide applications to land. The SWCC is designed to simulate the environmental concentration of a pesticide in the water column and sediment and is used for regulatory purposes by the USEPA Office of Pesticide Programs (OPP). The SWCC uses PRZM version 5.0+ (PRZM5) and the Variable Volume Water Body Model (VVWM), replacing the older PE5 shell (last updated November 2006), which used PRZM3 and EXAMS.

Critical input values for the model are summarized in Tables 2 and 3. This model estimates that about 1.1% of aminopyralid applied to the field eventually reaches the water body. The main mechanism of transport from the field to the water body is by runoff (53.3% of the total transport) followed by spray drift (46.7%).

In the water body, pesticide dissipates with an effective water column half-life of 68.2 days. (This value does not include dissipation by transport to the benthic region; it includes only processes that result in removal of pesticide from the complete system.) The main source of dissipation in the water column is photolysis (effective average half-life = 71 days) followed by metabolism (1744.3 days) and volatilization (1.866018E+10 days).

In the benthic region, pesticide dissipation is negligible (1744.3 days). The main source of dissipation in the benthic region is metabolism (effective average half-life = 1744.3 days). The vast majority of the pesticide in the benthic region (92.5%) is in the pore water rather than sorbed to sediment.

Table 1. Estimated Environmental Concentrations (ppb) for aminopyralid.

	Soil half-life 103.5 d	Soil half-life 310.5 d
Peak (1-in-10 yr)	0.610	0.612
4-day Avg (1-in-10 yr)	0.596	0.598
21-day Avg (1-in-10 yr)	0.552	0.553
60-day Avg (1-in-10 yr)	0.476	0.477

[1] USEPA Water Models Pesticides: http://www.epa.gov/pesticide-science-and-assessing-pesticide-risks/about-water-exposure-models-used-pesticide

365-day Avg (1-in-10 yr)	0.145	0.146
Entire Simulation Mean	0.726E-01	0.727E-01

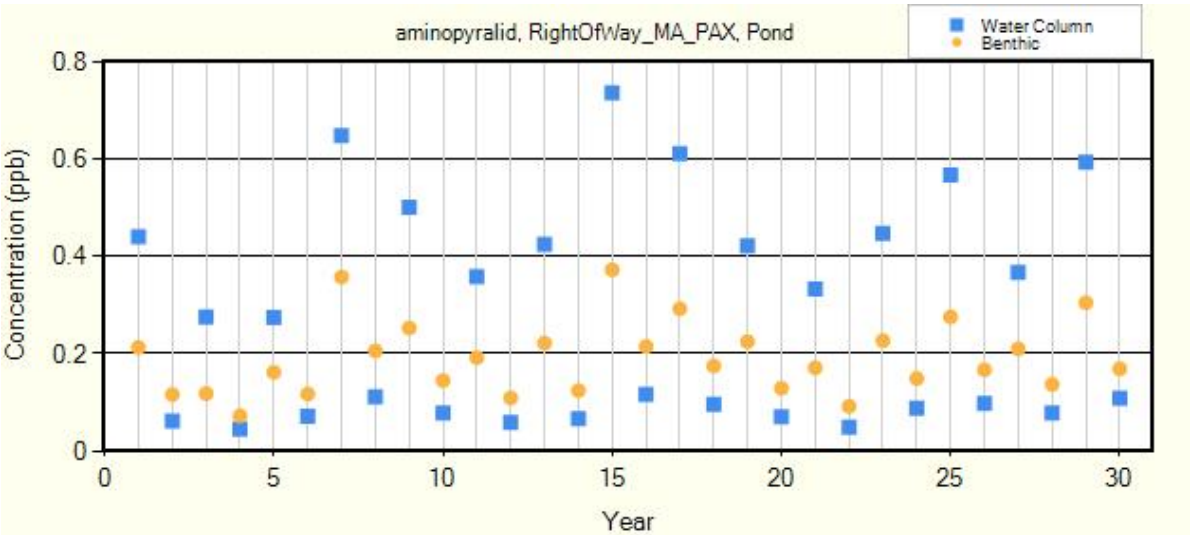
Table 2. Summary of Model Inputs for aminopyralid.

Scenario	RightOfWay_MA_PAX
Cropped Area Fraction	1
K_D (ml/g)	0.03
Water Half-Life (days) @ 20 °C	1073.6
Benthic Half-Life (days) @ 20 °C	1073.6
Photolysis Half-Life (days) @ 42 °Lat	0.6
Hydrolysis Half-Life (days)	0
Soil Half-Life (days) @ 20 °C	103.5
Foliar Half-Life (days)	
Molecular Wt	207
Vapor Pressure (torr)	7.4e-11
Solubility (mg/l)	2480

Table 3. Application Schedule for aminopyralid (every two years)

Date (Mon/Day)	Type	Amount (kg/ha)	Eff.	Drift
07/01	Foliar	0.11	0.95	0.05

Figure 1. Yearly Peak Concentrations


aminopyralid, RightOfWay_MA_PAX, Pond
Water Column
Benthic
Concentration (ppb)
0.8
0.6
0.4
0.2
0
0
5
10
15
20
25
30
Year

Appendix 1B: Summary of Water Modeling of aminopyralid and the USEPA Standard Reservoir

Estimated Environmental Concentrations for aminopyralid are presented in Table 1 for the USEPA standard reservoir with the RightOfWay_MA_PAX field scenario. A graphical presentation of the year-to-year peaks is presented in Figure 1. These values were generated with the Surface Water Concentration Calculator (SWCC Version 1.106). Critical input values for the model are summarized in Tables 2 and 3.

This model estimates that about 0.72% of aminopyralid applied to the field eventually reaches the water body. The main mechanism of transport from the field to the water body is by runoff (78.9% of the total transport) followed by spray drift (21.1%).

In the water body, pesticide dissipates with an effective water column half-life of 53.4 days. (This value does not include dissipation by transport to the benthic region; it includes only processes that result in removal of pesticide from the complete system.) The main source of dissipation in the water column is photolysis (effective average half-life = 97.3 days) followed by washout (126.8 days), metabolism (1744.3 days), and volatilization (2.556444E+10 days).

In the benthic region, pesticide dissipation is negligible (1744.3 days). The main source of dissipation in the benthic region is metabolism (effective average half-life = 1744.3 days). The vast majority of the pesticide in the benthic region (92.5%) is in the pore water rather than adsorbed to sediment.

Table 1. Estimated Environmental Concentrations (ppb) for aminopyralid.

	Soil Half-life 103.5 d	Soil Half-life 310.5 d
Peak (1-in-10 yr)	1.11	1.11
4-day Avg (1-in-10 yr)	1.08	1.08
21-day Avg (1-in-10 yr)	0.985	0.989
60-day Avg (1-in-10 yr)	0.792	0.794
365-day Avg (1-in-10 yr)	0.223	0.224
Entire Simulation Mean	0.938E-01	0.941E-01

Table 2. Summary of Model Inputs for aminopyralid.

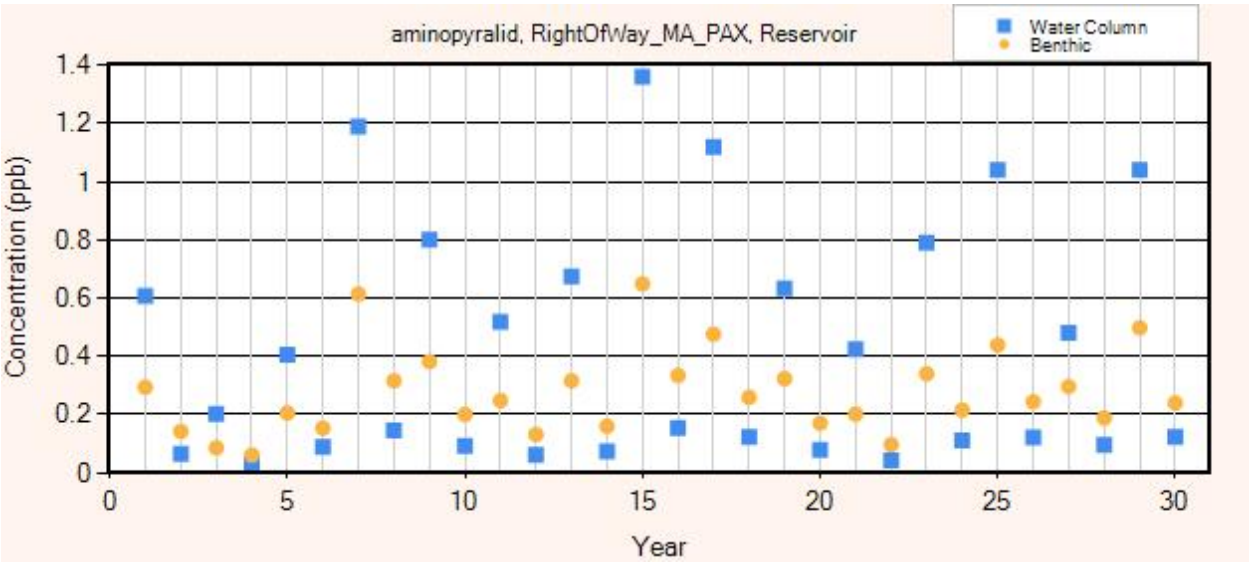
Scenario	RightOfWay_MA_PAX
Cropped Area Fraction	1.0
K_D (ml/g)	0.03

Water Half-Life (days) @ 20 °C	1073.6
Benthic Half-Life (days) @ 20 °C	1073.6
Photolysis Half-Life (days) @ 42 °Lat	0.6
Hydrolysis Half-Life (days)	0
Soil Half-Life (days) @ 20 °C	103.5
Foliar Half-Life (days)	
Molecular Wt	207
Vapor Pressure (torr)	7.4e-11
Solubility (mg/l)	2480

Table 3. Application Schedule for aminopyralid (every two years)

Date (Mon/Day)	Type	Amount (kg/ha)	Eff.	Drift
07/01	Foliar	0.11	0.95	0.05

Figure 1. Yearly Peak Concentrations



Appendix 1C: Summary of Water Modeling of aminopyralid in a Custom Small Pond Scenario

Estimated Environmental Concentrations for aminopyralid are presented in Table 1 for the custom small pond with the RightOfWay_MA_PAX field scenario. A graphical presentation of the year-to-year peaks is presented in Figure 1. These values were generated with the Surface Water Concentration Calculator (SWCC Version 1.106). Critical input values for the model are summarized in Tables 2 and 3.

The custom watershed characteristics were made to be more representative of a ROW scenario by considering a smaller catchment area-to-pond area/volume; it was adapted from the TOXSWA scenario: http://www.pesticidemodels.eu/toxswa/eu-registration . The depth of the pond was chosen to be 0.33 m initial depth and 0.67 m maximum depth. The applications occurred every two years.

This model estimates that about 0.62% of aminopyralid applied to the field eventually reaches the water body. The main mechanism of transport from the field to the water body is by runoff (96.8% of the total transport) followed by spray drift (3.24%).

In the water body, pesticide dissipates with an effective water column half-life of 11.6 days. (This value does not include dissipation by transport to the benthic region; it includes only processes that result in removal of pesticide from the complete system.) The main source of dissipation in the water column is photolysis (effective average half-life = 11.7 days) followed by metabolism (1744.3 days) and volatilization (3.078929E+09 days).

In the benthic region, pesticide dissipation is negligible (1744.3 days). The main source of dissipation in the benthic region is metabolism (effective average half-life = 1744.3 days). The vast majority of the pesticide in the benthic region (92.5%) is in the pore water rather than sorbed to sediment.

Table 1. Estimated Environmental Concentrations (ppb) for aminopyralid.

	Soil Half-life 103.5 d	Soil Half-life 310.5 d
Peak (1-in-10 yr)	12.2	12.3
4-day Avg (1-in-10 yr)	10.6	10.7
21-day Avg (1-in-10 yr)	6.63	6.66
60-day Avg (1-in-10 yr)	3.46	3.47
365-day Avg (1-in-10 yr)	0.598	0.600
Entire Simulation Mean	0.218	0.219

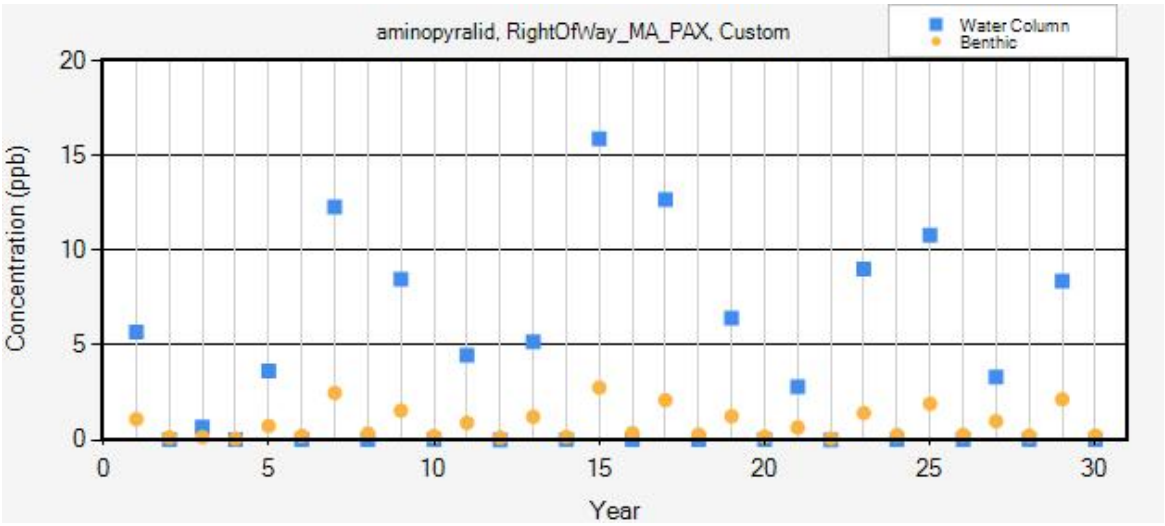
Table 2. Summary of Model Inputs for aminopyralid.

Scenario	RightOfWay_MA_PAX
Cropped Area Fraction	1.0
K_d (ml/g)	0.03
Water Half-Life (days) @ 20 °C	1073.6
Benthic Half-Life (days) @ 20 °C	1073.6
Photolysis Half-Life (days) @ 42 °Lat	0.6
Hydrolysis Half-Life (days)	0
Soil Half-Life (days) @ 20 °C	103.5
Foliar Half-Life (days)	
Molecular Wt	207
Vapor Pressure (torr)	7.4e-11
Solubility (mg/l)	2480

Table 3. Application Schedule for aminopyralid (every two years)

Date (Mon/Day)	Type	Amount (kg/ha)	Eff.	Drift
07/01	Foliar	0.11	0.99	0.01

Figure 1. Yearly Peak Concentrations



Appendix 2A: Groundwater Modeling with SCIGROW

SCI-GROW (Screening Concentration in Groundwater) is a screening-level tool to estimate drinking water exposure concentrations in groundwater resulting from pesticide use. As a screening tool, SCI-GROW provides conservative estimates of pesticides in groundwater. It is a generic model that provides peak estimates of compound concentrations in groundwater based on a given application rate, number of applications, and standard environmental fate parameters of soil aerobic half-life and soil binding constant. SCI-GROW is an empirical model based on a linear best fit through 13 single-application groundwater studies. These studies were typically two to three year studies. SCI-GROW is a screening level risk assessment tool that has been used to evaluate the effect of pesticide use on groundwater. More information on the SCI-GROW model is available at EPA website for water models: Water Models | Pesticides | US EPA [2]

Model input and output is given below.

```
SCIGROW

                    VERSION 2.3
          ENVIRONMENTAL FATE AND EFFECTS DIVISION
               OFFICE OF PESTICIDE PROGRAMS
           U.S. ENVIRONMENTAL PROTECTION AGENCY
                      SCREENING MODEL
              FOR AQUATIC PESTICIDE EXPOSURE


 SciGrow version 2.3
 chemical:Aminopyralid
 time is  2/20/2015  12: 4:28
 ------------------------------------------------------------------------
  Application      Number of       Total Use    Koc      Soil Aerobic
  rate (lb/acre)  applications   (lb/acre/yr)  (ml/g)   metabolism (days)
 ------------------------------------------------------------------------
      0.110           1.0           0.110      1.05E+00      103.5
 ------------------------------------------------------------------------
 groundwater screening cond (ppb) =   5.17E+00
 ************************************************************************
```

[2] USEPA Water Models: http://www.epa.gov/pesticide-science-and-assessing-pesticide-risks/about-water-exposure-models-used-pesticide

Appendix 2B: Groundwater Modeling with PRZM-GW model

Analysis for Aminopyralid and the DELMARVA Sweet Corn - Evesboro Loamy Sand Scenario in the PRZM-GW model system

PRZM-GW (Pesticide Root Zone Model – Ground Water) was developed as the harmonized tool for assessing pesticide concentrations in groundwater. This model has the capability to consider variability in leaching potential of different soils, weather (including rainfall), cumulative yearly applications or depth to aquifer. The conceptual model is based on a rural drinking water well beneath an agricultural field (a high pesticide use area), which draws water from an unconfined, high water-table aquifer. Processes included in the conceptual model that influence pesticide transport through the soil profile include water flow, chemical specific dissipation and transportation parameters (i.e., degradation and sorption), and crop specific factors, including transpiration, pesticide interception and management practices.

Six different scenarios were developed for the PRZM-GW model. The modeling for the review presented here was based on the Delmarva Sweet Corn - Evesboro Loamy Sand scenario. Delmarva Peninsula sweet corn scenario is one of the six PRZM-GW standard scenarios that fall within regions where groundwater is highly susceptible to nitrate contamination. The six scenarios are expected to provide reasonable upper bound estimates for pesticide concentrations for vulnerable groundwater sources (USEPA, 2015)[3].

The Delmarva Corn scenario most closely represents the Virginia Coastal Plain spatially and characteristically. In the Delmarva Corn scenario, the vadose zone ends and the aquifer begins 9 meters (29.5 feet) below the land surface. It has been reported that 26 of 29 Virginia Coastal Plain counties have at least one domestic well with a depth to the bottom of the well screen of 30 feet or less. Using this example, it follows that modeling with PRZM-GW provides estimated drinking water concentrations (EDWCs) that represent a subset of a broadly distributed population relying on shallow, private drinking water wells.

Weather data were representative of Eastern Massachusetts and scenario characteristics for vegetation were adjusted to be representative of ROW vegetation. Vegetation height, root zone depth were set at values that were used in ROW model scenario used of surface water modeling (Wijnja, 2010). Model simulation can be considered to be representative of behavior at a

[3] USEPA, 2015.Implementation of the Pesticide Root Zone Model Groundwater (PRZM-GW) for Use in EPA's Pesticide Exposure Assessments. USEPA, Office of Pesticide Program, Environmental Fate and Effects Division (EFED), September 8, 2015. Accessed at: http://www.epa.gov/sites/production/files/2015-11/documents/attachment_1_-_implementation_report_of_przm-gw_final.pdf

vulnerable site given the loamy sand soil profile and the absence of a buffer zone around the well.

Estimated groundwater concentrations and breakthrough times for aminopyralid are presented in Table 1 for the DELMARVA sweet corn - Evesboro loamy sand groundwater scenario. A graphical presentation of the daily concentrations in the aquifer is presented in Figure 1. These values were generated with the PRZM-GW (Version 1.07). Critical input values for the model are summarized in Tables 2 and 3.

Table 1. Groundwater Results for aminopyralid and the DELMARVA sweet corn - Evesboro loamy sand Scenario for ROW in Massachusetts

	Soil half-life 103.5 d	Soil half-life 310.5 d
Peak Concentration (ppb)	12.6	19.6
Post-Breakthrough Mean Concentration (ppb)	10.5	15.8
Entire Simulation Mean Concentration (ppb)	7.52	11.3
Average Breakthrough Time (days)	3013.025	3013.025
Throughputs	3.63754	3.63754

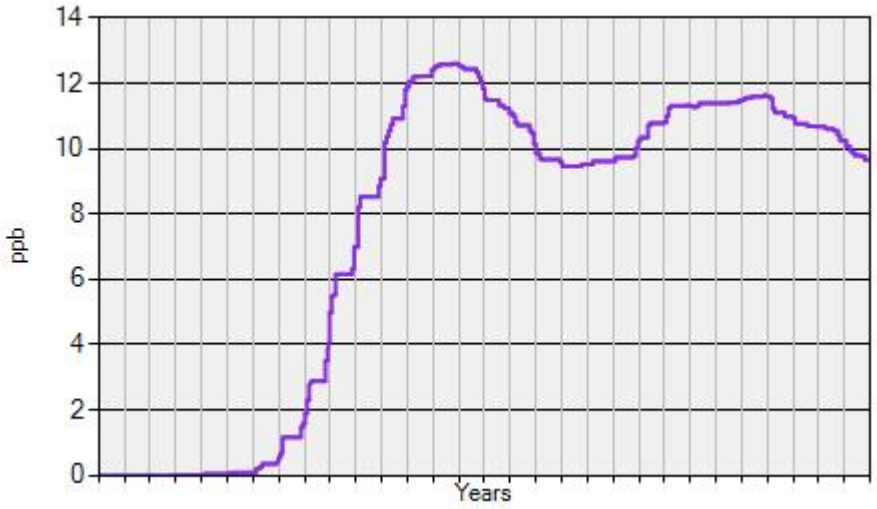
Table 2. Chemical Properties for Groundwater Modeling of aminopyralid.

Koc (ml/g)	1.05
Surface Soil Half Life (days)	103.5 (310.5)
Hydrolysis Half Life (days)	0
Diffusion Coefficint Air (cm2/day)	0.0
Henry's Constant	0.0
Enthalpy (kcal/mol)	0.0

Table 3. Pesticide application scheme used for aminopyralid. This application scheme was applied once every 2 years of the simulation.

Application Date (Month/Day)	Application Method	Application Rate (kg/ha)
07/01	Above canopy application	0.11

Figure 1. Aquifer Breakthrough Curve for aminopyralid and the DELMARVA Sweet Corn - Evesboro Loamy Sand Scenario. Groundwater depth is 10 m and application of 0.11 lbs/acre occur every 2 years. Results shown are for simulation with soil half-life of 103.5 d.



THE COMMONWEALTH OF MASSACHUSETTS
EXECUTIVE OFFICE OF ENERGY AND ENVIRONMENTAL AFFAIRS

Department of Agricultural Resources
251 Causeway Street, Suite 500, Boston, MA 02114
617-626-1700 fax: 617-626-1850 www.mass.gov/agr

MDAR
MASSACHUSETTS DEPARTMENT OF AGRICULTURAL RESOURCES

FOSAMINE-AMMONIUM

In addition to the review that is presented below, comprehensive reviews and assessments are available from U.S. EPA that incorporate more recent studies and data.

Fosamine-Ammonium Registration Review documents are available at www.regulations.gov in docket ID: EPA-HQ-OPP-2010-0215

FOSAMINE AMMONIUM

Common Trade Name: Krenite, Krenite UT

Chemical Name: Ammonium ethyl carbamoylphosphate

CAS No.: 25954—13—6

GENERAL INFORMATION

Fosamine ammonium is usually applied to plants in the late summer and early fall. It is systemically absorbed by buds, stems and foliage. In most plants, effects of herbicide treatment are not evident until the following spring when buds fail to develop, or develop into miniature spindly leaves that do not provide adequate photosynthesis. The plant consequently dies. Although it is translocated within plants, effective treatment requires the complete coverage of all parts of woody plants. In some species of non-deciduous plants, such as pines and bindweed, leaves may turn brown immediately after application.

ENVIRONMENTAL FATE

Mobility

Fosamine ammonium is a low mobility herbicide and is not readily leached from soil. Soil adsorption coefficients (Kd) for Fosamine ammonium are reported as ranging from 0.22 (low organic sandy barns) to 350 (silt barns) (103). The organic matter adsorption coefficients are more variable and range from 20 to 62, with one adsorption coefficient reported at 7400 (103). There does not appear to be a good correlation between the soil adsorption coefficents and organic matter, clay or silt content of the soil.

In a study using soil thin layer plates to assess mobility, the Rf values (ratio of the compound mobility versus the leading edge of the water movement) for Fosamine ammonium ranged from 0.92 to 0.98 on the four soils tested (103). These Rf values indicate a high mobility pesticide, in contrast to the soil adsorption coefficients and leaching studies which indicate low mobility. This information may reflect the solubility of fosamine ammonium and not its mobility characteristics.

Fosamine arnmonium is strongly adsorbed to soil particles and it is not carried away in precipitation, in spite of its high water solubility. In a laboratory study using inclined soil flats (Fallingston sandy loam), Fosamine ammonium

was applied at the rate of 15 lbs a.i/acre followed by simulated rainfall. The Fosamine ammonium remained near the surface of the soil and in the upper part of the flat, thus indicating no appreciable downward or lateral mobility (105). Field studies conducted in Florida, Delaware and Illinois have confirmed the laboratory results and indicate very little or no downward movement in soil of the herbicide or its degradation products (15, 104, 105).

Field studies indicate that Fosamine ammonium has low vertical mobility but, soils with higher adsorption capacities will tend to retard movement more than soil with lower adsorption capacities (15). However, Fosamine ammonium may move with the soil during erosion (14). Due to strong adsorption of fosamine ammonium to soil particles, there is little tendency for ground water contamination or for surface waters to become contaminated without direct application of the material (14, 15).

In the field studies, the Delaware soil (Keyport silt loam) was the most representative soil of Massachusetts conditions. However, the Fallsington sandy loam which was used in the greenhouse studies represents a close approximation to Massachusetts soils. In these studies Fosamine ammonium exhibited slight tendency to leach in both those soils. Consequently, it is expected that fosamine ammonium will exhibit slight leaching in Massachusetts soils.

Persistence

The major route of Fosamine ammonium degradation is metabolism by soil microorganisms. Fosamine ammonium is stable to degradation by hydrolysis at pH values 5, 7, and 9; it is also stable to photodegradation (10, 14, 101, 102).

Fosamine ammonium is not considered a persistent compound in soils. Under field conditions in Florida, Delaware and Illinois, the half-life of Fosamine ammonium in soils was approximately one week following the application of 10 lbs/acre (104).

In the field, the metabolite carbamoylphosphonic acid (CPA) was found several days after initial soil treatment. All Fosamine ammonium and CPA had disappeared completely by 3 to 6 months (14, 15).

Greenhouse soil studies indicate a half-life of about 10 days, which is in close agreement with the field study half—life (15,104). In the field, Fosamine ammonium was metabolized to CPA more quickly in fine sand than in two silt barns (14, 104).

There is little persistence information in the literature for Fosamine ammonium and the only reported field degradation rates are from one study. This might be a cause for concern were it not for the close agreement in soil half-lives reported, not withstanding the varied location and soils used in the field stu-dies. Moreover, the greenhouse degradation study was also in close agreement with the reported field half-life.

It is assumed that the half-lives reported in the previous study have been obtained in spring to summer conditions, since they were not stated. The degradation of fosamine ammonium was investigated for a one year period in the previous study but, because of the short half-life complete degradation had occurred before the winter. It is expected that fosamine ammonium will be applied in summer or fall only since it must be applied to full foliage for control. Consequently, the lack of winter degradation rates is not a major concern.

With most herbicides soil characteristics and local climatic factors have a pronounced effect on soil half—life. This study suggest that degradation of Fosamine ammonium by soil microorganisms is not influenced by soil characteristics or local climate to any appreciable extent.

Due to the similar persistence of Fosamine ammonium in all locations and soils there is no most representative location. In this case, all sites represent expected persistence. Therefore, the half-life of Fosamine ammonium under Massachusetts condition is expected to be approximately one week.

TOXICITY REVIEW

Acute (Mammalian)

The oral LD5Os have been determined for both the formulated product and the formulated product plus surfactant (41.1 to 42% active ingredient (ai) in both cases). The LD5Os in the male rat were 24,400 mg (ai) (formulated product)/kg and 7,295 mg (ai) (formulated product with surfactant)/kg. Female rats had an LD50 of 5,000 (ai) mg (formulated product with surfactant)/kg. The formulated product has an LD50 of 7,380 mg(ai)/kg (formulated product) in male guinea pigs (107).

Fosamine ammonium was tested in an acute dermal study. 10 ml of the formulated product at a dose of 1,683 mg(ai)/kg resulted in no mortalities and no clinical signs of toxicity (107). The formulation plus surfactant was tested in rabbits and was not a primary eye irritant. There was mild transient erythema in tested skin. No sensitization was found in Guinea pigs (107).

The formulation plus surfactant (0.1 ml) produced transient mild corneal opacity and transient conjunctual irritation. The formulation without the surfactant was not an irritant (107).

Metabolism

The metabolism of Fosamine ammonium in the rat is rapid with 86% in feces and 11% in urine after 48 hrs (103,15). Compounds identified in the feces included 14C radiolabelled fosamine ammonium (86%) and 14C Carbamoylphosphonic Acid (CPA) diammonium salt (14%). The compnunds identified in the urine were also fosamine ammonium and CPA (103).

Subchronic and chronic feeding studies have been performed using several species, for various time periods.

The No Observable Effect Level (NOEL) for Fosamine Ammonium in diet studies for rats (90 day), dog (6 month), and sheep (90 day) were: 5,000/10,000 ppm, (286/572 mg/kg); 1,000 ppm (40 mg/kg) and 2,000/2,500 ppm highest dose tested (HDT) respectively (107). In the feeding studies the dose was increased after a certain time point when effects were not observed at the lower dose. These dose groups are written first dose/increased dose. In the six month dog study, the female dogs receiving 5000/7500/10000 ppm had increased stomach weights (107).

Oncogenicity Studies

Long term carcinogenicity studies are not available. These studies have not been required by EPA as there are no food uses proposed for Krenite.

Mutagenicity Studies

Mutagenicity testing has been done using Fosamine Ammonium formulated product. It was negative in 5 strains of the Ames assay, and negative both with and without activation in Chinese Hamster ovary point mutation assay. Chromosome damage was produced in the in vitro cytogenetic assay using Chinese Hamster ovary cells at 1.6% and 3.2 formulation (nonactivated) and 1.4, 2.8 and 5.7% formulation (activated) (107). There were no compound related increases in chromosomal aberrations in an in vivo bone marrow study and no changes in unscheduled DNA synthesis in rat hepatocytes (107).

Developmental Studies

The developmental studies that have been performed using fosamine ammonium include a one generation/two litter rat study and a rat oral teratogenicity study. The doses in the 90 day reproduction study were 0, 200, 1,000 and 5,000/10,000 ppm (0, 11, 57 and 285/570 mg/kg/d). There were no effects observed on reproduction and lactation in the reproduction study (NOEL = 5,000/10,000 ppm HOT). The doses in the teratogenicity study were 0, 200, 1,000 and 5,000/10,000 ppm (0, 11, 57 and 285/570 mg/kg/d). There were no effects observed on teratogenicity and fetoxicity at the 1,000 ppm dose level(107).

(a) In these discussions the assumptions made for conversion of ppm (diet) to mg/kg/D were: Species Body weight (kg) Intake (kg) Rat 0.35 0.020 Mouse 0.03 0.004 Dog 10 0.4

Avian

Unformulated Fosamine ammonium was administered to Mallard ducks and bobwhite quail by intubation in acute toxicity studies. Five birds per species-sex group received doses of 0, 312.5, 625, 1,250, 2,500, and 5,000 mg/kg. The LD50 was greater than 5,000 mg/kg in both the ducks and quail (15, 107).

Ducks and quail were also used in subacute dietary studies at doses of 0, 625, 1,250, 2,500, 5,000 and 10,000 ppm in the diet for 5 days. Basal diet was given for the last three days of the 8 day exposure. The 8 day LC50 in the diet was greater than 10,000 ppm. There was no increase in duck mortality: food consumption was depressed but body weight gain was normal. There was variable quail mortality and food consumption and body weight were decreased as compared with control (15, 107).

Invertebrates:

Fosamine ammonium toxicity has been determined for only a very few microorganisms and invertebrates. The available studies indicate that Fosamine ammonium has a very low acute toxicity to those organisms tested (15):

Fosamine ammonium salt (42% formulation): 48 hr LC5Os range from 1,524 mg/L for Daphnia to 10,000 mg/L for bees sprayed with the herbicide.

Aquatic Species (fish):

Fosamine ammonium has a very low toxicity to those fish species tested.
Fosamine ammonium salt (42% formulation): 96 hr LC5Os range from 670 mg/L for bluegill sunfish to 8,290 mg/L for coho salmon (15).

Except for the LC5O of 670 mg/L for the bluegill sunfish, reported adult fish LC5Os are all in excess of 1000 mg/L. (15) The yolk-sac fry stage in salmonids was the most sensitive to Fosamine ammonium.

Threshold-effect concentrations of Krenite for salmonids in partial life-cycle studies are less than 75 times the maximum theoretical concentration of Krenite that would be found in shallow waters due to direct overhead spray application (15). SUMMARY

Fosamine ammonium is not persistent in the environment and is a low mobility herbicide in soil. Fosamine ammonium has a low potential to leach to groundwater or to reach surface waters from surface runoff. With acute oral LD5Os in rats of greater than 5,000 mg/kg, Fosamine ammonium is considered to be of low acute and subchronic mammalian toxicity. Subchronic exposures to Fosamine ammonium resulted in NOELS of greater than 1,000 ppm in a 6 month dog study. Mutagenicity test were negative in all but one case and there are no carcinogenicity data for this active ingredient. Fosamine ammonium is also considered to have very low aquatic and invertebrate acute toxicity.

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THE COMMONWEALTH OF MASSACHUSETTS
EXECUTIVE OFFICE OF ENERGY AND ENVIRONMENTAL AFFAIRS

Department of Agricultural Resources
251 Causeway Street, Suite 500, Boston, MA 02114
617-626-1700 fax: 617-626-1850 www.mass.gov/agr

MDAR
MASSACHUSETTS DEPARTMENT OF AGRICULTURAL RESOURCES

GLYPHOSATE

In addition to the review that is presented below, comprehensive reviews are available from U.S. EPA and USDA Forest Service that incorporate more recent studies and data. The US Forest Service risk assessment report is available at the U.S. FOREST SERVICE webpage, Pesticide-Use Risk Assessments and Worksheets:

https://www.fs.fed.us/foresthealth/protecting-forest/integrated-pest-management/pesticide-management/pesticide-risk-assessments.shtml

Glyphosate Registration Review documents are available at www.regulations.gov in docket ID: EPA-HQ-OPP-2009-0361

Review conducted by MDAR and MassDEP for use in Sensitive Areas of Rights-of-Way in Massachusetts

Common Trade Name(s): Roundup, Glyphosate VMF Round Up Pro, Rodeo, Accord, Accord Concentrate; Chemical Name: N—(phosphonomethyl)glycine—isopropylamine salt; CAS No.: 1071-83-6

GENERAL INFORMATION Glyphosate, n-phosphonomethyl glycine, is a systemic, broad spectrum herbicide effective against most plant species, including deep rooted perennial species, annual and biennial species of grasses, sedges, and broadleafed weeds. The major pathway for uptake in plants is through the foliage, however, some root uptake may occur. The presence of surfactants and humidity increases the rate of absorption of glyphosate by plants (15).

Foliarly applied glyphosate is readily absorbed and translocated from treated areas to untreated shoot regions. The mechanism of herbicidal action for glyphosate is believed to be inhibition of amino acid biosynthesis resulting in a reduction of protein synthesis and inhibition of growth (10, 15, 101).

Glyphosate is generally formulated as the isopropylamine salt in aqueous solution (122). Of the three products containing glyphosate considered here, Roundup is sold with a surfactant and Rodeo and Accord are mixed with surfactants prior to use (15). Glyphosate has been reviewed by US Forest Service (15), FAO (122), and EPA 00W (51).

ENVIRONMENTAL FATE

Mobility Glyphosate is relatively immobile in most soil environments as a result of its strong adsorption to soil particles. Adsorption to soil particles and organic matter begins almost immediately after application. Binding occurs with particular rapidity to clays and organic matter (l5). Clays and organic matter saturated with iron and aluminum (such as in the Northeast) tend to absorb more glyphosate than those saturated with sodium or calcium. The soil phosphate level is the main determinant of the amount of glyphosate adsorbed to soil particles. Soils which are low in phosphates will adsorb higher levels of glyphosate (14, 15).

Glyphosate is classified as immobile by the Helling and Turner classification system. In soil column leaching studies using aged (1 month) Glyphosate, leaching of glyphosate was said to be insignificant after 0.5 inches of

water per day for 45 days (14).

Persistence It has been reported that glyphosate dissipates relatively rapidly when applied to most soils (14). However, studies indicate that the soil half-life is variable and dependent upon soil factors. The half-life of glyphosate in greenhouse studies when applied to silty clay loam, silt loam, and sandy loam at rates of 4 and 8 ppm was 3, 27 and 130 days respectively, independent of application rate (14). An average half-life of 2 months has been reported in field studies for 11 soils (15).

Glyphosate is mainly degraded biologically by soil micro-organisms and has a minimal effect on soil microflora (15). In the soil environment, glyphosate is resistant to chemical degradation such as hydrolysis and is stable to sunlight (15). The primary metabolite of glyphosate is aminomethyl phosphonic acid (AMPA) which has a slower degradation rate than glyphosate (15). The persistence of AMPA is reported to be longer than glyphosate, possibly due to tighter binding to soil (14). No data are available on the toxicity of this compound.

Glyphosate degradation by microorganisms has been widely tested in a variety of field and laboratory studies. Soil characteristics used in these studies have included organic contents, soil types and pHs similar to those that occur in Massachusetts (117).

Glyphosate degradation rates vary considerably across a wide variety of soil types. The rate of degradation is correlated with microbial activity of the soils and does not appear to be largely dependent on soil pH or organic content (117). While degradation rates are likely temperature dependent, most reviews of studies do not report or discuss the dependence of degradation rate on temperature. Mueller et al. (1981 cited in 117) noted that glyphosate degraded in Finnish agricultural soils (loam and fine silt soils) over the winter months; a fact which indicates that degradation would likely take place in similar soils in the cool Massachusetts climate. Glyphosate halflives for laboratory experiments on sandy loam and loamy sand, which are common in Massachusetts, range up to 175 days (117). The generalizations noted for the body of available results are sufficiently robust to incorporate conditions and results applicable to glyphosate use in Massachusetts.

TOXICITY REVIEW

Acute (Mammalian) Glyphosate has reported oral LD5Os of 4,320 and 5,600 mg/kg in male and female rats (15,4). The oral LD5Os of the two major glyphosate products Rodeo and Roundup are 5,000 and 5,400 mg/kg in the rat (15).

A dermal LD5O of 7,940 mg/kg has been determined in rabbits (15,4). There are reports of mild dermal irritation in rabbits (6), moderate eye irritation in rabbits (7), and possible phototoxicity in humans (9). The product involved in the phototoxicity study was Tumbleweed marketed by Murphys Limited UK (9). Maibach (1986) investigated the irritant and the photo irritant responses in individuals exposed to Roundup (41% glyphosate, water, and surfactant); Pinesol liquid, Johnson Baby Shampoo, and Ivory Liquid dishwashing detergent. The conclusion drawn was that glyphosate has less irritant potential than the Pinesol or the Ivory dishwashing liquid (120).

Metabolism Elimination of glyphosate is rapid and very little of the material is metabolized (6,106). Subchronic/Chronic Studies (Mammalian) In subchronic tests, glyphosate was administered in the diet to dogs and rats at 200, 600, and 2,000 ppm for 90 days. A variety of toxicological endpoints were evaluated with no significant abnormalities reported (15,10).

In other subchronic tests, rats received 0, 1,000, 5,000, or 20,000 ppm (57, 286, 1143 mg/kg) in the diet for 3 months. The no observable adverse effect level (NOAEL) was 20,000 ppm (1,143 mg/kg) (115). In the one year oral dog study, dogs received 20, 100, and 500 mg/kg/day. The no observable effect level (NOEL) was 500 mg/kg (116).

Oncogenicity Studies Several chronic carcinogenicity studies have been reported for glyphosate including an 18 month, mouse study; and a two year rat study. In the rat study, the animals received 0, 30, 100 or 300 ppm in their diet for 2 years. EPA has determined that the doses in the rat study do not reach the maximum tolerated dose (112) and replacement studies are underway with a high dose of 20,000 ppm (123). The mice received 1000, 5000 or 30,000 ppm for 18 months in their diets. These studies were non-positive (112,109). There was a non-statistically significant increase in a rare renal tumor (renal tubular adenoma (benign) in male mice (109). The rat chronic study needs to be redone with a high dose to fill a partial data gap (112). The EPA weight of evidence classification would be D: not classified (51).

Mutagenicity Testing Glyphosate has been tested in many short term mutagenicity tests. These include 7 bacterial (including Salmonella typhimurim and B. subtilis) and 1 yeast strain Sacchomyces cerevisiae as well as a mouse dominant lethal test and sister chromatid exchange. The microbial tests were negative up to 2,000 mg/plate (15), as were the mouse dominant lethal and the Chinese hamster ovary cell tests. EPA considers the mutagenicity requirements for glyphosate to be complete in the Guidance for the Registration of Pesticide Products containing glyphosate (112).

The developmental studies that have been done using glyphosate include teratogenicity studies in the rat and rabbit, three generation reproduction studies in the rat, and a reproduction study in the deer mouse. (15)

Rats were exposed to levels of up to 3,500 mg/kg/d in one rat teratology study. There were no teratogenic effects at 3,500 mg/kg/d and the fetotoxicity NOEL was 1,000 mg/kg/d. In the rabbit study a fetotoxicity NOEL was determined at 175 mg/kg/d and no teratogenic effects were observed at 10 or 30 mg/kg/d in one study and 350 mg/kg/d in the other study (15). No effects were observed in the deer mouse collected from conifer forest sprayed at 2 lbs active ingredient per acre (15).

Tolerances & Guidelines EPA has established tolerances for glyphosate residues in at least 75 agricultural products ranging from 0.1 ppm (most vegetables) to 200 ppm for animal feed commodities such as alfalfa (8).

U.S. EPA Office of Drinking Water has released draft Health Advisories for Glyphosate of 17.50 mg/L (ten day) and 0.70 mg/L (Lifetime)(51).

Avian Two types of avian toxicity studies have been done with glyphosate: ingestion in adults and exposure of the eggs. The species used in the ingestion studies were the mallard duck, bobwhite quail, and the adult hen (chickens). The 8 day feeding LC5Os in the mallard and bobwhite are both greater than 4,640 ppm. In the hen study, 1,250 mg/kg was administered twice daily for 3 days resulting in a total dose of 15,000 mg/kg. No behavioral or microscopic changes were observed (15).

Invertebrates A variety of invertebrates (mostly arthropods) and microorganisms from freshwater, marine, and terrestrial ecosystems have been studied for acute toxic effects of technical glyphosate as well as formulated Roundup. The increased toxicity of Roundup compared with technical glyphosate in some studies indicates that it is the surfactant (MONO 818) in Roundup that is the primary toxic agent (117). Acute toxicity information may be summarized as follows:

Glyphosate (technical): Acute toxicity ranges from a 48 hr EC5O for midge larvae of 55 mg/L to a 96 hr TL5O for the fiddler crab of 934 mg/L (15).

Roundup: Acute toxicity ranges from a 48 hr EC5O for Daphnia of 3 mg/L to a 95 hr LC5O for crayfish of 1000 mg/L (15).

Among the insects tested, the LD50 for honeybees was 100 mg/bee 48 hours after either ingestion, or topical application of technical glyphosate and Roundup. This level of experimental exposure is considerably in excess of exposure levels that would occur during normal field applications (15).

Aquatic Species (Fish) Technical glyphosate and the formulation Roundup have been tested on various fish species. Roundup is more toxic than glyphosate, and it is the surfactant that is considered to be the primary toxic agent in Roundup:

Glyphosate (technical): Acute 96 hr LC5Os range from 24 mg/L for bluegill (Dynamic test) to 168 mg/L for the harlequin fish (15).

Roundup: Acute lethal toxicity values range from a 96 hr LC5O for the fathead minnow of 2.3 mg/L to a 96 hr TL5O for rainbow trout of 48 mg/L (15).

Tests with Roundup show that the egg stage is the least sensitive fish life stage. The toxicity increases as the fish enter the sac fry and early swim up stages.

Higher test temperatures increased the toxicity of Roundup to fish, as did higher pH (up to pH 7.5). Above pH 7.5, no change in toxicity is observed.

Glyphosate alone is considered to be only slightly acutely toxic to fish species (LC5Os greater than 10 mg/L), whereas Roundup is considered to be toxic to some species of fish, having LC5Os generally lower than 10 mg/L (15,118).

SUMMARY

Glyphosate when used as recommended by the manufacturer, is unlikely to enter watercourses through run-off or leaching following terrestrial application (117). Toxic levels are therefore unlikely to occur in water bodies with normal application rates and practices (118).

Glyphosate has oral LD5Os of 4,320 and 5,600 in male and female rats respectively. The elimination is rapid and very little of it is metabolized. The NOAEL in rats was 20,000 ppm and 500 mg/kg/d in dogs. No teratogenic effect was observed at doses up to 3,500 mg/kg/d and the fetotoxicity NOELS were 1,000 mg/kg/d in the rat and 175 mg/kg/d in the rabbit.

The evidence of oncogenicity in animals is judged as insufficient at this time to permit classification of the carcinogenic potential of glyphosate. The compound is not mutagenic.

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THE COMMONWEALTH OF MASSACHUSETTS
EXECUTIVE OFFICE OF ENERGY AND ENVIRONMENTAL AFFAIRS

Department of Agricultural Resources
251 Causeway Street, Suite 500, Boston, MA 02114
617-626-1700 fax: 617-626-1850 www.mass.gov/agr

MDAR
MASSACHUSETTS DEPARTMENT OF AGRICULTURAL RESOURCES

IMAZAPYR

In addition to the review that is presented below, comprehensive reviews are available from U.S. EPA and USDA Forest Service that incorporate more recent studies and data. The US Forest Service risk assessment report is available at the U.S. FOREST SERVICE webpage, Pesticide-Use Risk Assessments and Worksheets:

https://www.fs.fed.us/foresthealth/protecting-forest/integrated-pest-management/pesticide-management/pesticide-risk-assessments.shtml

Imazapyr Registration Review documents are available at www.regulations.gov in docket ID: EPA-HQ-OPP-2014-0200

Review conducted by MDAR and MassDEP for use in Sensitive Areas of Rights-of-Way in Massachusetts

Common Trade Name(s): Arsenal

Chemical Name: Imazapyr: 2-(4-isopropyl-4-methyl--5-oxy-2-imidazolin-2-yl) nicotinic acid with isopropyl amine (2) CAS No.: 81510-83-0

GENERAL INFORMATION

Imazapyr is effective against and provides residual control of a wide variety of annual and perennial weeds, deciduous trees, vines and brambles in non—cropland situations. It also provides residual control and may be applied either pre or postemergence. Postemergence is the preferred method especially for the control of perennial species. Imazapyr is readily absorbed by the foliage and from soil by the root systems. Imazapyr kills plants by inhibiting the production of an enzyme, required in the biosynthesis of certain amino acids, which is unique to plants (10, 100).

ENVIRONMENTAL FATE

Mobility

There are few studies which have investigated the mobility of Imazapyr in soil, but available reports indicate that Imazapyr does not leach and is strongly absorbed to soil (100). Imazapyr has a high water solubility (1 — 1.5%) which could generally indicate a high leaching potential, but as with other organic acids Imazapyr is much less mobile than would normally be expected (100). No soil partition coefficients have been reported, but they may be expected to be quite high (100).

One field study investigated Imazapyr mobility in a sandy loam soil (0.9% organic matter, 8.0% clay; 38.8% silt). Imazapyr did not leach below the 18—21 inch layer after 634 days and 49.6 inches of rain. The levels found below the 12 inch layer were just above the 5 ppb detection limit. In addition, this study investigated the off—target mobility of Imazapyr and found no residues further than 3 inches from the sprayed area after 1 year (102).

Although low levels of Imazapyr did move to the 18 to 21 inch layer this was only after nearly 2 years and fifty inches of rain. This indicates that imazapyr is relatively non-mobile and does not leach through the soil profile. Imazapyr remains near the soil surface and heavy precipitation may cause some off target movement from surface erosion of treated soils.

Persistence

The main route of Imazapyr degradation is photolysis. In a study of photodegradation in water, the half— life of Imazapyr was calculated as 3.7, 5.3 and 2.5 days in distilled water, pH 5 and pH 9 buffers respectively (101). A soil photolysis study for Arsenal on sandy loam calculated a half—life of 149 days (101).

Studies have investigated the persistence of Imazapyr in soil under aerobic and anaerobic conditions. The half-life of Imazapyr in soil has been reported as varying from 3 months to 2 years (100). A laboratory study found the half-life to be 17 months (101). Detectable residues were found in a field study in all soil layers to 21 inches at 634 days (102). Vegetation was sprayed with radio-labelled Imazapyr at a rate of 1 lb. a.i./acre. The soil was a sandy loam (0.9% organic matter) which received 49.6 inches of rain during 634 days. The highest level of radioactivity (0.234 ppm Imazapyr) was found in the top 3 inches of soil at 231 days after application and there were detectable levels in the 9-12 inch layer. The concentrations in the top layer increased steadily from day 4 to 231 when they reached their maximum (0.234 ppm) and then declined. At day 634 the level in the top layer (0-3 inch) was 0.104 ppm (102). These data indicate that Imazapyr is persistent in soil and, most importantly, that Imazapyr is translocated within plants from the plant shoots back to the roots and released back into soil. Very little of the Imazapyr actually reached the soil during application. The soil residues may be due to the decay of plant material containing Imazapyr in the soil (102).

TOXICITY REVIEW

Acute (Mammalian)

The acute oral LD5O in both male and female rats was greater than 5000 mg/kg using technical Imazapyr. The acute dermal LD5O in male and female rabbits was greater than 2000 mg/kg. The compound was irritating to the rabbit eye but recovery was noted 7 days after application of 100 mg of the test substance. It was classified as mildly irritating to the rabbit skin following application of 0.5 grams of the material on abraded or intact skin (103).

Arsenal product formulation was tested in a similar battery of tests. The rat oral LD5O value was greater than 5000 mg/kg and the rabbit dermal LD5O was greater than 2148 mg/kg. The irritation was observed following installation of 0.5 ml of the test substance in the skin study and 0.1 ml in the eye study (104).

Technical Imazapyr was administered to rats as an aerosol for four hours at a concentration of 5.1 mg/L. There were ten rats per sex and the animals were observed for 14 days after treatment before they were sacrificed. Slight nasal discharge was seen in all rats on day one but disappeared on day two (105).

The inhalation LC5O is greater than 5.0 mg/L for both the formulation and the technical product (105,106). Technical Imazapyr was applied dermally at the following dosages: 0, 100, 200 and 400 mg/kg/day (109). Arsenal was used at 0, 25, 50 and 100% of the formulated solution in sterile saline. Each dose group consisted of 10 male and 10 female rabbits and the test substance was applied to either intact or abraded skin and occluded for 6 hours each day.

The result of the dermal studies with Imazapyr as well as Arsenal were non remarkable with regard to body weights, food consumption, hematology, serum chemistry, clinical observations, necropsy observations and histopathology. It was noted that Arsenal, undiluted, was locally irritating (109).

Subchronic and Chronic Studies (Mammalian) In the subchronic tests a NOEL for systemic toxicity with dermal administration in rabbits was 400 mg/kg/d (2,109). After dietary administration for 13 weeks in the rat, there was no effect at 10,000 ppm

(571. mg/kg/d) which was the highest dose tested (141).

A bioassay is currently underway to evaluate the potential oncogenicity of technical Imazapyr. Groups of 65 rats per sex per dose group have received 0, 1000, 5000 or 10,000 ppm in the diet. Hematology, clinical chemistry and urinalysis tests were conducted at 3, 6 and 12 months and will also be done at 18 months and at study termination. At the 12 month sacrifice the only effect noted was a slight increase in mean food consumption in all treated female groups. Most of the increases were statistically significant, but they did not always exhibit a dose response. The oncogenicity test is due to be submitted to the EPA in the spring of 1989 (115).

Oncogenicity Studies

Chronic bioassays as discussed in the subchronic/chronic section are underway.

Mutagenicity Testing

Five different bacterial strains of Salmonella typhimurium (TA1535, TA98, TAlOO, TA1537, and TA1538) and one of Escherichia coli (WP-2 uvrA-) were used to evaluate the mutagenicity of Imazapyr. It is unclear whether the compound used was technical or formulated Imazapyr. Dose levels up to 5000 micrograms/plate were used and each strain was evaluated both in the presence or absence of PCB— induced rat liver 5—9 microsomes. Negative results were noted in all assays. The six tester strains were designed to detect either base-pair substitutions or frameshift mutations (113).

Developmental Studies (Mammalian)

Two teratology studies have been done and both of these studies evaluated technical Imazapyr. One study used rats as the test species and the other utilized rabbits (111,112).

Pregnant rats received dosages of 0, 100, 300 or 1000 mg/kg/d of Imazapyr during days 6—15 of gestation. There were 22 rats in the control group and 24, 23 and 22 in the low, mid and high dose groups. All doses were administered orally by gavage. Salivation was noted only during the dosing period in 6 of the 22 females in the highest dose group (1000 mg/kg). No other adverse observations were noted in the treated dams (111). Fetal body weight and crown-rump length data for the treated groups were comparable to controls. Fetal development (external, skeletal and visceral) “revealed no aberrant structural changes which appeared to be the result of the exposure to Imazapyr” (111). The NOEL for maternal toxicity was 300 mg/kg and the NOEL for teratogenicity and fetoxicity was 1000 mg/kg (116).

Four groups of 18 pregnant rabbits were exposed on days 6-18 of gestation to doses of 0, 25, 100, 400 mg/kg/d Imazapyr. There was no statistically significant difference between control and treated groups at any dose (112).

Avian

Acute oral LD5Os of Imazapyr in bobwhite quail and mallard duck were 2150 mg/kg. The 8 day dietary LC5O in the bobwhite quail and mallard duck were greater than 5000 ppm (101).

Invertebrates

The dermal honey bee LD5O for Imazapyr is greater than 100 mg/bee (101). The LD5O (48 hr) was greater than 100 mg/L for the water flea (100).

Aquatic The LC50s of Imazapyr in the rainbow trout, bluegill sunfish and channel catfish were greater than 100 mg/L (101).

SUMMARY Imazapyr is a relatively immobile herbicide in the soil profile even when used in sandy and low organic content soils. It is also persistent in soils. The low mobility and persistence may result in off-target movement of Imazapyr from surface erosion of treated soils.

The atypical soil—plant flux characteristics of Imazapyr and delayed maximum soil concentrations indicate that repeated annual applications may result in build—up of Imazapyr in soil. Consequently, an interval is required to allow for the degradation of soil residues before a repeated application is made.

The oral LD5O of Imazapyr in rats is greater than 5000 mg/kg and the derrnal LD5O is greater than 2000 mg/kg in rabbits. The oncogenicity bioassay is currently underway and the only effect reported in the interim study was an increase in food consumption in the treated females. No mutagenic effects were observed.

The acute oral LD5Os of Imazapyr and the Arsenal formulation are greater than 5000 mg/kg. In the subchronic 13 week rat study there was no effect observed at the highest dose tested 10,000 ppm. The oncogenicity study is currently underway.

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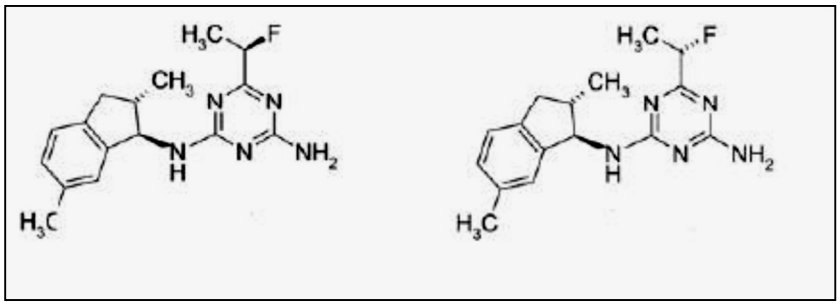
Review of Indaziflam for Application to Sensitive Areas of Rights-of-Way

This document summarizes the environmental fate and transport, as well as toxicological and ecological effects of the herbicide indaziflam. The information summarized in this review was considered in the evaluation of indaziflam for use in Sensitive Areas of Rights-of-Ways in Massachusetts. This review was jointly conducted by the Massachusetts Department of Environmental Protection (MassDEP) Office of Research and Standards (ORS) and the Massachusetts Department of Agricultural Resources (MDAR) in accordance with the cooperative agreement issued between the two agencies in 1987 and updated in 2011 pursuant to the provisions of Section 4(1)(E) of 333 CMR 11.00 Rights-of-Way Management Regulations.

Much of the information used to conduct this review is from the US Environmental Protection Agency (US EPA), including the Pesticide Fact Sheet for Indaziflam (US EPA 2010a), as well as information from several supporting documents available in the US EPA docket no. EPA-HQ-OPP-2009-0636. This information was supplemented by additional, more recent information on ecological risk from Bayer CropScience, reviews of indaziflam conducted by the New York State Department of Environmental Conservation, Health Canada and the Australian Pesticides and Veterinary Medicines Authority, as well as fate and transport studies obtained from the literature.

Indaziflam (N-[(1R, 2S)-2,3-dihydro-2,6-dimethyl-1H-inden-1-yl]-6-[(1RS)-1-fluoroethyl]-1,3,5-triazine-2,4-diamine) is an alkylazine herbicide manufactured by Bayer used for preemergent control of annual grasses and broadleaf weeds. It is an active ingredient contained in several herbicide products manufactured by Bayer. The active ingredient indaziflam was initially registered by the US EPA in 2010 for non-crop use and then in 2011 for food crop (such as citrus, stone and pome fruit, and grapes) uses. Technical grade indaziflam is a mixture of two isomers, including 95-100% of isomer A and 0-5% of isomer B (NYSDEC, 2012)).

Figure 1. Indaziflam Isomer Structures



Isomer A Isomer B

At the time of this active ingredient review by MDAR and MassDEP, Esplanade 200 SC (EPA Reg. No. 432-1516), an end-use product manufactured by Bayer Environmental Science, was submitted for review. Additional details on the evaluation of this product can be found in a separate review document.[1]

Herbicidal Mode of Action:

Indaziflam is a cellulose biosynthesis inhibitor. It prevents the deposition of cellulose into the plant cell wall, thus severely affecting cell wall formation, cell division and cell elongation. It interferes with synthesis of the cell wall in actively growing parts of the plant, where cellulose synthesis is occurring, such as in actively growing meristematic tissues, dividing cells, expanding cells and growing roots. It targets seed growth prior to germination and during root development. Indaziflam has little to no effect on fully developed leaves and plant tissues in which cellulose synthesis is not taking place. Thus, its main use is in targeting pre-emergent weeds (US EPA, 2010a,b; APVMA, 2015; HC, 2011).

Indaziflam Fate and Transport:

Indaziflam applied to soil is moderately mobile, with reported K_{oc} values ranging from 396 to 789 L/kg (APVMA, 2015, HC, 2011). It is moderately persistent in aerobic soils, with reported half-lives of greater than 150 days, and persistent (stable) in anaerobic soils and sediments. Photolysis is not a major degradation pathway of indaziflam in soil. Indaziflam dissipates mainly through biotic degradation and leaching.

In water, indaziflam is a weak acid and has low solubility. In clear, shallow waters, it degrades fairly rapidly by photolysis, with a half-life of about 3.7 days though is stable to hydrolysis. It readily partitions to sediment in 0-3 days, where it is persistent.

The major environmental metabolites of indaziflam (see Figure 2.) include triazine-indanone, indaziflam carboxylic acid, indaziflam-hydroxyethyl, indaziflam-olefin, diaminotriazine and dihydrotriazine (APVMA, 2015). The degradates of indaziflam are more mobile than the parent indaziflam and were detected at the deepest depths sampled (i.e., up to 120 cm). Of the three major metabolites identified in soil (i.e., triazine-indanone, indaziflam carboxylic acid and diaminotriazine), diaminotriazine is also more persistent as well as being mobile to highly mobile and thus has the potential to leach to groundwater (APVMA, 2015, USEPA, 2010a).

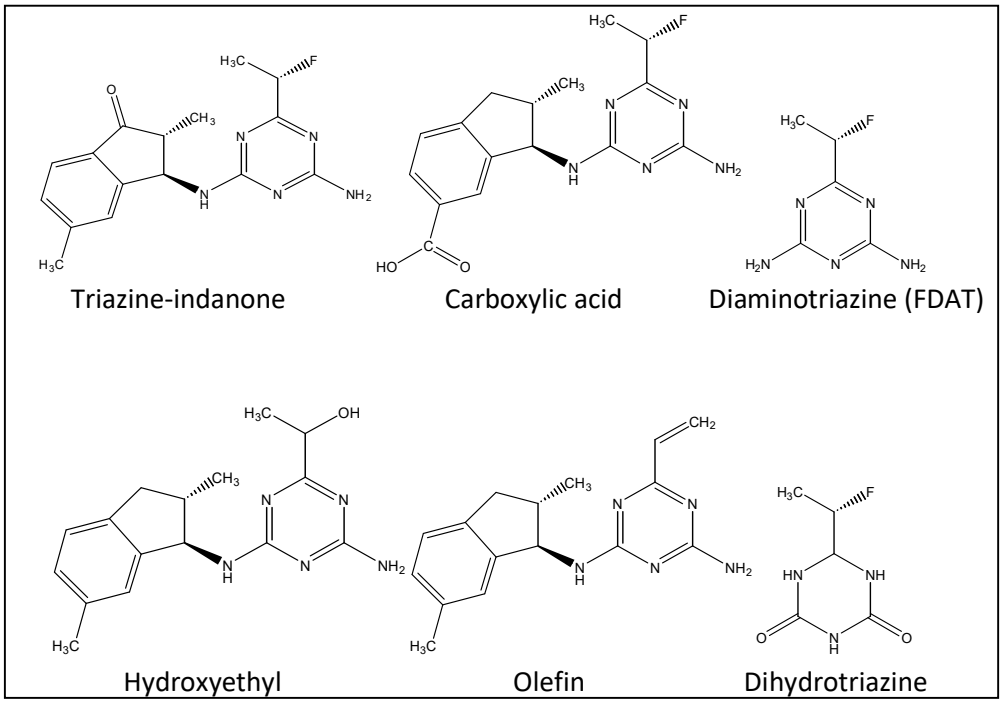
Environmental modeling conducted by several of the secondary sources cited above and confirmed by MDAR however, indicate that predicted concentrations of indaziflam in groundwater are low.

Based on the GUS or Groundwater Ubiquity Score[2], indaziflam has moderate potential to move toward

[1] Product Review of Esplanade Herbicide For Addition to the Sensitive Area Materials List in Massachusetts
[2] Groundwater Ubiquity Score (GUS) (orst.edu)

groundwater and ranks lower in such potential compared to several other herbicides on the Sensitive Area Materials List.[3]

Figure 2. Indaziflam Major Environmental Metabolites



(HC, 2015)

<u>Human Toxicity</u>:

Once ingested, indaziflam is rapidly and completely absorbed. In animal studies, it was also metabolized and excreted rapidly mainly in the feces and urine, with elimination of the administered dose complete by 48 hours. Thus, the potential for indaziflam to bioaccumulate is low. About 40% of the parent indaziflam was excreted unchanged. The major metabolite is an oxidized carboxylic acid form of indaziflam. Dermal absorption of indaziflam is low.

Technical indaziflam has low acute toxicity in rats by the dermal, inhalation and ingestion exposure routes. It was non-irritating to the eyes and skin of rabbits and not a skin sensitizer in guinea pigs. In subchronic and chronic studies in rats and dogs, the nervous system is the major target organ. There are species differences in toxicity, with the dog being the most sensitive, greater than ten times more sensitive than the rat. Other organs affected by indaziflam in rodent studies include the kidney, liver, thyroid, stomach, seminal vesicles, and ovaries.

[3] Sensitive Area Materials List: Rights of Way Sensitive Area Materials List | Mass.gov; GUS values for individual herbicides can be found in the Pesticide Properties Database (https://sitem.herts.ac.uk/aeru/ppdb/)

There is no evidence of carcinogenicity in long-term studies with mice and rats. Neither indaziflam, nor two of its metabolites (i.e., diaminotriazine and indaziflam carboxylic acid) were found to be mutagenic in a battery of genotoxicity tests. Based on the results of these tests, the US EPA classified indaziflam as, "not likely to be carcinogenic to humans".

Indaziflam caused some developmental effects in the offspring of rats, but not rabbits, at doses that also caused maternal toxicity. The US EPA concluded that there is evidence of increased quantitative susceptibility to rat fetuses exposed *in utero* to indaziflam.
Because indaziflam and its metabolite, (fluoroethyl) diaminotriazine (FDAT), both contain a triazine ring (i.e., a six-membered benzene-like ring that includes three nitrogens), the possibility that this structure is associated with toxicity endpoints similar to several other triazine herbicides (i.e., atrazine, simazine, propazine) and their metabolites (desethyl-s-atrazine (DEA), deisopropyl-s-atrazine (DIA), and diaminochlorotriazine (DACT)) has been considered by US EPA and others. These other analogous compounds have been designated as a group by US EPA, known as the "triazine common mechanism group" (TCMG). The TCMG chemicals have a common mechanism of toxicity on the endocrine system, producing effects on the reproductive system in female rats, including a decrease in the luteinizing hormone surge, altered pregnancy outcome and delayed preputial separation, in addition to an increase in the incidence of mammary gland tumors. However, US EPA concluded that, despite the structural similarities, indaziflam and its metabolite did not meet the criteria for inclusion in the TCMG group based on both structural and toxicological reasons. Indaziflam and FDAT contain a fluoroethyl group in their triazine rings whereas the TCMG chemicals contain a chlorine. In addition, the same types of toxicological responses noted above were not seen in an Indaziflam reproduction and fertility study in rats, other than delayed sexual maturation at the highest dose, but at a much higher dose as compared to DACT. Therefore, US EPA does not assume that indaziflam and its metabolite have a common method of toxicity and thus does not include them in a cumulative risk approach as it does for the TCMG chemicals (US EPA, 2010a).

Due to the structural similarity of indaziflam to its metabolites, US EPA assumes that all of the metabolites of indaziflam have comparable toxicity to the parent compound. Diaminotriazine, a single-ring metabolite, is not expected to be more toxic than indaziflam based on its non-neurotoxic mode of action.

The US EPA developed a chronic Population Adjusted Dose (cPAD) for indaziflam of 0.02 mg/kg/day based on the most sensitive effect in the most sensitive species in the indaziflam database. This value, which is similar to a US EPA Reference Dose (RfD) was identified as the No Observed Adverse Effect Level (NOAEL) of 2.0 mg/kg/day from a chronic toxicity study in dogs, to which was applied an uncertainty factor of 100. In this study, degeneration of nerve fibers occurred in the brain, spinal cord and sciatic nerve at the Low Observed Adverse Effect Level (LOAEL) of 6 mg/kg/day and 7 mg/kg/day in males and females, respectively (US EPA, 2010a).

For short- and intermediate-term incidental oral, dermal and inhalation exposure, the US EPA developed a short-term acute Population Adjusted Dose (aPAD) level of 0.075 mg/kg/day based on a NOAEL of 7.5 mg/kg body weight/day from a subchronic toxicity study in dogs, to which an uncertainty factor of 100 was applied. The same effect (i.e., degeneration of nerve fibers in the brain, spinal cord and sciatic nerve) was seen at the NOAEL of 7.5 as in the chronic study. This short-term value is also adopted as relevant for acute exposure. Though an acute exposure study in rats was available and was the basis of a previous short-term level developed by US EPA in 2010, the US EPA observed that the dog is much more sensitive (i.e., greater than the ten-fold factor for inter-species differences that is part of the 100-fold uncertainty factor used to derive this value) than the rat, and though generally, use of a subchronic endpoint as the basis of an acute value is conservative, given the severity of observed neurotoxic effects in the dog as compared to the rat and the absence of a neurotoxicity study in dogs, US EPA concluded that this conservative approach was prudent (US EPA, 2010a,b; APVMA, 2015; HC, 2011).

In its review of the active ingredient, indaziflam, US EPA calculated aggregate exposure estimates for indaziflam from food, water and residential exposures, including via ingestion, inhalation and dermal exposure, compared these to the appropriate points of departure (i.e., the aPAD and cPAD) to determine whether acute and chronic dietary pesticide exposures are safe, and concluded that there is a reasonable certainty that no harm will result to the general population or to infants and children from these exposures. Though agriculturally related or residential exposures are not relevant to exposures expected in the ROW application scenario, ingestion of surface water and/or groundwater is identified as a relevant dietary pathway for the general public.

Since the potential for several metabolites of indaziflam to contaminate groundwater is high, due to its high mobility and propensity to leach, the US EPA used water exposure models that estimate, based on their physical, chemical and fate/transport characteristics, surface water and groundwater concentrations of indaziflam and its metabolites following indaziflam application at label rates. Total toxic residue concentrations in water were conservatively calculated for indaziflam, the four major indaziflam metabolites that maintain the dual ring structure of the parent indaziflam, and the two single-ring metabolites. As discussed above, all metabolites are assumed to be of comparable toxicity to the parent.

US EPA compared these modeled concentrations to acute one-day (500 ug/L) and chronic (100 ug/L) drinking water benchmark concentrations, known as US EPA Human Health Benchmarks for Pesticides (HHBP) (derived by the US EPA based on the aPAD and cPAD information discussed above) and demonstrated that the predicted concentrations of these compounds are well below the HHBP values and thus have low, potential toxicity to humans.

EPA also examined potential ingestion of indaziflam in surface water by conducting a similar conservative evaluation considering the potential for both indaziflam and its metabolites to enter surface water and demonstrated that expected concentrations of indaziflam and its metabolites following indaziflam application in accordance with label instructions, would be well below the HHBP benchmark concentrations. Given that ROW areas in Massachusetts must observe setbacks from

streams and waterbodies, the concern that high concentrations of Indaziflam will enter surface waters is even less likely.

The groundwater and surface water conclusions reached by the US EPA and others were also confirmed in a modeling evaluation conducted by MDAR (2020). MDAR modeled a very conservative scenario, in which indaziflam was applied annually for thirty years at the maximum concentration to a watershed with sandy soils (to simulate soils in areas such as southeastern Massachusetts and Cape Cod) at the maximum label rate use. The model results assume application to 100% of the area whereas in a ROW area, only fractions of a given area receive pesticide applications, plus there is a 100 foot setback requirement from surface drinking water supplies. Peak, modeled concentrations for this worst-case scenario were well below both acute and chronic HHBP levels. See MDAR (2020) for additional details (USEPA, 2010c; MDAR, 2020).

Ecotoxicity

Indaziflam has low toxicity to wild mammals, upon both acute as well as chronic exposure. Toxicity to most birds was also low, though there was an outstanding question regarding potential reproductive effects in mallards. At the request of the US EPA, the manufacturer conducted an additional mallard reproductive study, in which several female birds were found with regressed ovaries. However, no statistically significant differences were found in adult body weight effects, or mortality, egg or embryo reproductive effects, or hatchling effects and body weight. US EPA identified a LOAEL of 720 ppm in this study, which corresponds to a daily dietary dose of 89 mg/kg/day of the active ingredient. EPA protocol for evaluating toxicity to reptiles uses data for birds as a surrogate and, as such, toxicity to reptiles is assumed by EPA to be low.

Indaziflam has low toxicity to honeybees, non-target arthropods, and earthworms. It is not toxic to freshwater and sediment-dwelling invertebrates. It is acutely highly toxic to fish, both freshwater and marine/estuarine, moderately to highly toxic to estuarine invertebrates, and slightly toxic to moderately toxic to freshwater invertebrates. Toxicity to amphibians was evaluated using data on the most sensitive fish species as a surrogate. Thus, indaziflam is assumed to be toxic to amphibians as well.

Almost all of the fish and aquatic toxicity tests were classified by the US EPA as supplemental because test solutions were not centrifuged to accurately determine how much of the indaziflam was actually in solution (NYSDEC, 2012). However, according to Bayer, their procedure is to evaluate the solubility of the test material in water prior to testing with aquatic animals to determine the limits of solubility in the test system—and they only centrifuge or filter the test solutions prior to chemical analysis if they observe precipitate in the test solutions. They state that they are confident that the analytical measurements are valid and adequately reflect the dissolved concentrations—and that the fact that US EPA did not request them to repeat the aquatic studies indicates that the information is of sufficient quality to be used in a risk assessment (US EPA, 2010a).

Despite the high toxicity of indaziflam to aquatic organisms, application rates of indaziflam are low—and thus environmental concentrations of indaziflam in ROWs predicted using modeling are low. This is confirmed by the results of surface water exposure modeling for ecological risk assessment conducted by the US EPA and repeated by DAR (using conservative assumptions as well as land, soil and weather modeling data that are more representative of Massachusetts ROW areas).

This modeling conservatively does not account for the fact that in Massachusetts ROW areas, application of herbicides must observe setbacks from streams and waterbodies, which would likely further decrease predicted concentrations of indaziflam in surface water to negligible concentrations. The EPA acknowledges that toxicity to aquatic organisms is high and requires label language to help mitigate these risks and keep the herbicide on the intended treatment area. Thus, concern that high concentrations of Indaziflam will enter surface waters is low if indaziflam applications are made as specified in the product label.

According to the US EPA, based on the most sensitive ecological taxa tested, indaziflam-olefin and indaziflam-hydroxyethyl, are similar in toxicity to the parent compound, while the rest of the environmental degradates demonstrate a toxicity about 2-7 times less than the parent compound. Thus, none of the degradates are any more toxic than the parent compound (US EPA, 2010).

Plant Toxicity

Given indaziflam's mode of action, which is specific to plant cell wall biology, it is not surprising that non-target nonvascular and vascular aquatic plants, as well as both monocot and dicot terrestrial plants, are sensitive to it. These non-target sensitive plants include a number of plants listed under the Endangered Species Act. In addition, effects on non-target plants that might not be endangered species but which might serve as a food source for endangered animal species would be of concern (US EPA 2010a).

Similar to the strategy used for aquatic life, the US EPA mitigates potential risks to plants by requiring label language intended to keep the herbicide on the intended treatment area (US EPA, 2010a).

Conclusions

Review of secondary documents from both US EPA and other agencies consistently present the same profile and conclusions on the toxicity, fate and transport of this herbicide. This information, supplemented by additional MDAR predictive modeling of indaziflam concentrations in groundwater and surface water following its application as per label instructions in ROW area in Massachusetts, indicate that exposures to indaziflam residues by human and ecological receptors should not be of toxicological concern.

While indaziflam and its metabolites do have the potential to leach to groundwater especially in looser soils, predicted concentrations of these compounds in groundwater used as drinking water following indaziflam application at label rates are well below toxicity benchmarks for humans.

Indaziflam in surface water quickly partitions to sediment, and it dissipates quickly via photolysis in shallow water. The probability that high concentrations of indaziflam will enter surface water is very low, especially since herbicide application in Massachusetts ROW's must observe a 100-foot setback from surface water bodies used as a source of public water. Modeling conducted by the US EPA and MDAR confirm this. Thus, concentrations of indaziflam in surface water used as drinking water following application of indaziflam as per label instructions are also expected to be well below toxicity benchmarks for human exposure.

Indaziflam is absorbed completely and metabolized fairly quickly so the potential for it to bioaccumulate in ecological receptors is low. While it is toxic to mammals, especially dogs, at doses administered in laboratory studies, exposure concentrations of indaziflam associated with herbicide applications are well below concentrations of concern for these receptors.

Although Indaziflam is highly toxic to freshwater and marine/estuarine fish, moderately toxic to freshwater invertebrates, highly toxic to marine/estuarine invertebrates and assumed to be toxic to amphibians, application rates of indaziflam are low and modeling based on these applications predict low exposures to ecological receptors. However, impacts to amphibians and reptiles are based on surrogate toxicity information for fish and birds respectively, and as such have additional uncertainty. Therefore, additional precautions should be taken as warranted to identify potentially significant amphibian and reptilian habitat prior to application.

Sensitive non-target plant species have been identified as organisms of concern. Given that herbicides are designed to control plants, this is not surprising. This information, coupled with the fact that indaziflam is moderately mobile and some of its metabolites are highly mobile strongly indicates that application of indaziflam should be targeted as much as possible to avoid impacts on non-target plants. Measures that minimize drift should be used in applying this product. In addition, as with any application, a preliminary field survey should be conducted prior to application to identify any plants on the endangered species list and/or any other plant species that are important to that ecosystem.

Based upon the available database for indaziflam, use of this herbicide in sensitive areas of rights-of-ways should be acceptable if it is applied in a manner that is consistent with the product label, the above recommendations and the Massachusetts Sensitive Areas of Rights-of-Way Regulations.

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THE COMMONWEALTH OF MASSACHUSETTS
EXECUTIVE OFFICE OF ENERGY AND ENVIRONMENTAL AFFAIRS

Department of Agricultural Resources
251 Causeway Street, Suite 500, Boston, MA 02114
617-626-1700 fax: 617-626-1850 www.mass.gov/agr

MDAR
MASSACHUSETTS DEPARTMENT OF AGRICULTURAL RESOURCES

METSULFURON METHYL

In addition to the review that is presented below, comprehensive reviews are available from U.S. EPA and USDA Forest Service that incorporate more recent studies and data. The US Forest Service risk assessment report is available at the U.S. FOREST SERVICE webpage, Pesticide-Use Risk Assessments and Worksheets:

https://www.fs.fed.us/foresthealth/protecting-forest/integrated-pest-management/pesticide-management/pesticide-risk-assessments.shtml

Metsulfuron-methyl Registration Review documents are available at www.regulations.gov in docket ID: EPA-HQ-OPP-2011-0375

Review conducted by MDAR and MassDEP for use in Sensitive Areas of Rights-of-Way in Massachusetts

Common Trade Names: Escort, Escort XP (2)

Chemical Name: Methyl 2 E[C[(4-Methoxy—6-methyl-l,3,5-Triazifl—
2-yl) aminolcarbonyl] amino] sulfonyl.]benzoate] (9)

CAS NO.: 74223-64-6

GENERAL INFORMATION

Metsulfuron methyl is a sulfonyl urea herbicide initially registered by E.I. DuPont in 1986. It is a foliar herbicide registered for use on wheat and barley and non-cropland sites such as Right of Way (9).

ENVIRONMENTAL FATE

Mobility Metsulfuron methyl is a relatively new herbicide. The studies reviewed here have been provided by the registrant, EI DuPont.

The soil water partition coefficients (Kd) of Metsulfuron Methyl have been determined in four different soils: Cecil sand, Flanagan silt loam, Fallsington silt loam, and keyport silt loam. The Kd values range from 0.36 for Cecil sand to 1.40 for Flanagan silt loam, and Kom values ranged from 29 for Fallsington silt loam to 120 for Cecil sand (100). The values for Kd and Kom indicate that metsulfuron methyl is not adsorbed well to soil and that the organic content of the soil is not the only adsorption component. The silt and clay contents appear to influence adsorption, but there are probably other factors also involved.

The previous study also determined the Rf values for soil. Thin layer chromatography was performed on four soils

for metsulfuron methyl. The Rf values ranged from 0.64 to 1.00; only one value was less than 0.90 (100). This result confirms the validity of the Kd values, indicating that metsulfuron methyl is mobiie and that the organic matter content of the Soil is a significant component of adsorption.

Metsulfuron methyl was applied to tops of 12 inch columns [containing four different soils], and eluted with 20 inches of water in 20 hours. Following the percolation of the total volume of water, 106% of the metsulfuron methyl was eluted from the Fallsington sandy loam, 96% from the Flanagan silt loam, 81% for Keyport silt loam and 93% for Myakka sand (100). The breakthrough volumes for the Fallsington, Flangan, Keyport and Myakka soils were 6.5, 4.5, 6.9 and 5.8 inches of water respectively (101).

Metsulfuron methyl is relatively mobile in most soils, but will be retained longer in soils with higher percentages of organic matter. Persistence There are two studies which have reviewed the persistence of metsulfuron methyl in the soil. One study was conducted in the southern United States and the second was in the northern United States and Canada. The results of the studies indicate a somewhat contradictory picture of the persistence of metsulfuron methyl.

The soil half-lives in Delaware, North Carolina, Mississippi and Florida were 1 week, 4 weeks, 3 weeks and 1 week respectively following an application in mid to late summer (102). The results are varied and indicate that either climatic or soil factors determine the persistence. The climate is sufficiently similar to be able to discount that as a factor. However, both of the locations where the shortest half-lives were observed had the highest organic matter content in the soils. Furthermore, the half—lives correspond with the organic matter content.

The half—lives following spring applications were 4 and 56 weeks for two sites in Colorado, 6 weeks in North Dakota and 28 weeks in Idaho (103). In contrast to the southern United States study there does not appear to be any correlation with climatic or soil characteristics. There appears to be a slightly shorter half—life in acidic soils in the same location.

Metsulfuron methyl was also applied in the fall and the half-lives determined in two sites in Colorado, North Dakota and Idaho. These half—lives were 8 weeks, 12 weeks, 42 weeks and 28 weeks respectively. As was expected there were longer half—lives following fall applications in North Dakota (6 weeks vs. 42 weeks) however, in Idaho there was no change at all, which is unexpected.

In Canada following spring applications the reported half-lives were 10 weeks, 4 weeks, 4 weeks and 6 weeks for Alberta, 2 locations in Saskatchewan and Manitoba (103). One would expect longer half lives in Northern locations due to the effects of temperature on degradation rates. The results from Canada are generally shorter than those in the U.S. locations, which is unexpected.

Therefore, the half-life of Metsulfuron methyl in the soil is variable and dependent on the location. It is shorter when applied in the spring but appears independent of other environmental factors in most locations.

TOXICITY REVIEW

Acute (Mammalian) The toxicology database for Metsulfuron methyl has been reviewed and accepted by the EPA (9). DuPont supplied excerpts from their monograph on Ally herbicide (112). Summaries of studies were supplied by DuPont for subchronic, chronic and reproductive studies. Technical metsulfuron methyl has been tested in two acute oral LD50 studies in Crl:CD Rats. In the first study the LD5O was greater than 5,000 mg/kg and in the second it was greater than 25,000 mg/kg (the maximum feasible dose) (112). Clinical signs included salivation, chromodacryorrhea, stained face, stained perineal area and weight loss (112).

In a 10—dose subacute study using male rats, a single repeated dose of 3,400 mg/kg/day for 10 days over a 2 week period was administered. This was followed by a two week recovery period. No deaths occurred and slight weight loss was the only clinical sign observed. In addition, no gross or microscopic changes were observed (112). The dermal LD50 is greater than 2,000 mg/kg in male and female rabbits (112). Technical metsulfuron methyl caused mild erythema as a 40% solution in guinea pigs. There was no reaction observed at the 4% concentration. No response occurred when treated animals were challenged (112).

In rabbits, moderate areas of slight corneal clouding and severe to moderate conjunctivitis were observed in both washed and unwashed eyes following treatment with technical metsulfuron methyl. The unwashed eyes were normal in 3 days and the washed eyes in 14 days (112).

Metabolism Elimination of metsulfuron methyl in the rat is rapid, with 91% of a radioactive dose excreted over 96 hours (9). The routes of elimination were not specified within the report.

Subchronic/Chronic (Mammalian) Ninety day feeding studies have been done with metsulfuron methyl in rats and mice. The rat study was done in conjunction with a one generation reproduction study (see Developmental Study Section). In this study rats received 0, 100, 1000, or 7500 ppm (0, 5.7, 57, 428 mg/kg/d) (a) in their diets. Effects observed at the high dose were: a decrease in body weight and an increase in total serum protein in the females, and a decrease in liver weight and a decrease in cytoplasmic clearing of hepatocytes in the males the NOEL in this study was 1000 ppm (104).

The 90 day mouse study was done in conjunction with the 18 month mouse study. Groups of 90 mice per sex per dose received 0, 5, 25, 500, 2500 or 5000 ppm (0, 0.66, 3.3, 66.6, 333.3, 666.6 mg/kg/d) in their diets. Clinical evaluations were made at 1, 2, 3, 6, 12 and 18 months. Ten animals per group were sacrificed at the 90 day time point for pathological evaluation. The 2500 ppm group was sacrificed at 12 months. Sporadic effects were observed on the body weight, food consumption, and organ weights. These were not dose related, resulting in a NOEL of 5000 ppm in diet for mice (111).

In the twenty-one day dermal rabbit study, the intact skin of male and female New Zealand White Rabbits received doses of 0, 125, 500 and 2,000 mg/kg for 6 hrs/day for 21 days. Clinical signs observed were sporadic weight loss and diarrhea in a few rabbits. These effects were not dose related. Non dose related histological effects were observed in male rabbits. This effect was characterized as mild testicular atrophy occurring sporadically at all doses (112, 108).

Feeding studies in dogs have been done with purebred beagles. The animals received metsulfuron methyl in diets at dose levels of 0, 50, 500 and 5000 ppm (0, 0.2, 2, 20 mg/kg/d) for one year. There was a decrease in food consumption in the high dose males. There was a decrease in serum lactate dehydrogenase in all groups of both sexes at two or more doses these values were within the historical controls. The NOEL was 500 ppm in the males and 5000 ppm in females (112).

In a chronic feeding study in rats, the animals received metsulfuron methyl at doses of 0, 5, 25, 500, 2500 or 5000 ppm (0, 0.28, 1.4, 28.6, 143 or 286 mg/kg/d. Interim sacrifices were done at 13 and 52 weeks (105).

At the 13 week sacrifice there was a decrease in body weight in the 2500 and 5000 ppm groups; there was a decrease in absolute liver weight at 2500 and 5000 ppm males. There was a decrease in the relative liver weights in the 2500 and 5000 ppm females.

(a) In these discussions the assumptions made for estimated conversion of ppm (diet) to mg/kg/D were: Species Body weight (kg) Intake (kg) Rat 0.35 0.020 Mouse 0.03 0.004 Dog 10 0.4 When data were presented as ppm, the dose was estimated in mg/kg and is presented in parenthesis.

Findings at the 52 week sacrifice included increase in kidney weight (2500 ppm males) and increased absolute brain weights (at doses of 25, 500, 2500 and 5000 ppm) in males and at doses of 2,500 and 5000 ppm in females. There was an increase in absolute heart weight at 2500 ppm in males and at 2500 and 5000 ppm in females. The absolute organ weights were back to normal at termination. Relative brain weights of the 2500 and 5000 ppm groups were increased (105)

Oncogenicity Studies There were no gross or histopathological changes observed in mice receiving up to 5000 ppm metsulfuron methyl in their diets (112. 111). Similar results were obtained in the 104 week rat study; there were no histopathological changes observed which were attributable to metsulfuron methyl (105, 112). EPA concludes that there were no oncogenic effects in rats or mice at the highest dose tested; 5000 ppm in both cases (9).

Mutagenicity Testing

Metsulfuron methyl was negative in the unscheduled DNA synthesis assay; in vivo bone marrow cytogenic assay in rats (doses were 500, 1,000, and 5,000 mglkg bw); CHO/HGPRT Assay; Salmonella typhimurium reverse mutation assay four strains with and without S9 metabolic activation; and also in the in vivo mouse micronucleus assay at doses of 166, 500, 1666, 3000 and 5000 mg/kg (112). 'T¶e only positive mutagenicity assay was in the in vitro assay for chromosome aberrations in Chinese Hamster Ovary at high doses (greater than 2.63 mM, 1.0 mg/mL)). In this assay no increases in structural aberrations were observed at 0.13 or 1.32 mM(0.05 or 0.5 mg/mL) (112).

Developmental Studies Several studies have been done to investigate the effects of Metsulfuron methyl on reproduction and development in rats and rabbits.

Pregnant Cr1: COBS CD(SD) BR rats received metsulfuron methyl at doses of 0, 40, 250 or 1000 mg/kg by the oral route on days 5 to 14 of gestation. There were 25 rats per group. Maternal toxicity was observed at doses of 250 and 1000 mg/kg/d. The maternal toxicity NOEL was 40 mg/kg/d. There was no evidence of "teratogenic" response or embryo fetal toxicity (112).

In the rabbit study, New Zealand white rabbits received 0, 25, 100, 300 or 700 mg/kg/d on days 6 to 18 gestation. There was a dose related increase in maternal deaths; 1, 2 and 12 deaths at doses of 100, 300 and 700 mg/kg respectively. The maternal toxicity NOEL was 25 mg/kg/d and there was no evidence of teratogenic or embryolethal effects observed in this study (112).

Several multigenerational studies have been done with Metsulfuron methyl. A four litter reproduction study was done concurrently with the chronic bioassay. Rats from each treatment were separated from the main study and bred. The doses were 0, 5, 25, 500, 2500, and 5000 ppm (0, 0.28, 1.4, 28.6, 143 and 286 mg/kg/d). There was a dose dependent decrease in body weight in the parental (P1) generation at doses of 25 ppm and greater in males and females. This effect was not present in dams during gestation or lactation (106).

Overall fertility in the P1 and filial (Fl) matings was low in both control and treated groups with no apparent cause. There was a decrease in pup size in the Fla but not the Flb, F2a, or F2b litters. The gestation index was 100% for all groups in both filial generations with the exception of F2a when it was 90%. On the basis of the lower body weights and lower growth rates, the NOEL was 25 ppm for this study (106).

In a 90 day, 2 generation 4 litter protocol, rats received 0, 25, 500 or 5000 ppm (0, 1.4, 28.6, 286 mg/kg/d) Metsulfuron methyl in their diets for 90 days prior to mating. In this protocol the parental generation was bred twice first to produce the Fla and then the FiB. The FiB rats were then fed the appropridte diet for 90 days (after weaning). There was a decrease in litter size in the 5000 ppm group in the F2a generation, but not in any other generation. The NOEL for this study was 500 ppm (107).

In a 90 day feeding, one generation rat study, 16 male and 16 female rats received 0, 100, 1000 or 7500 ppm in their diet prior to mating. There were no differences observed in reproduction and lactation performance or litter survival among groups. There was an overall low fertility in the control and treated groups. This result made the effects of metsulfuron methyl on fertility difficult to assess from this study (104).

Tolerances and Guidelines Tolerances have been set for metsulfuron methyl in barley wheat (from 0.05 to 20 ppm, depending on the commodity) and in meat and meat byproducts (0.1 ppm). The tolerance in milk is 0.05 ppm (8, 9). The acceptable daily intake is 0.0125 mg/kg/d based on a one year dog NOEL of 1.25 mg/kg/d using a safety factor of 100 (9).

Avian Metsulfuron methyl has been tested in two species of birds, the mallard duck and the bobwhite quail. The acute oral LD5O is greater than 2150 mg/kg in the duck. Two, 8 day dietary studies have been done. The 8 day LC5O is greater than 5620 ppm in both the duck and the quail (9).

Invertebrates

The 48 hour LC5O for Daphnia is greater than 150 ppm and the acute toxicity in the honeybee is greater than 25 mg/bee (9). Aquatic Metsulfuron methyl has acute LC5O of greater than 150 ppm in both the rainbow trout and the bluegill sunfish (9).

Summary Metsulfuron methyl has a moderate to high mobility in the soil profile and is relatively persistent in the environment, especially when applied in the fall. These factors would be of concern under most circumstances. However, metsulfuron methyl is applied at very low rates (3-4 ozs./A) and therefore the amounts which reach the soil are quite low. Consequently, Metsulfuron methyl should not impact groundwater as a result of leaching or migrate from the target area. Metsulfuron methyl has low toxicity (EPA Toxicity Category III) for acute dermal exposure and primary eye irritation and is category IV for all other acute exposures. The chronic studies indicate no oncogenicity response and the systemic NOEL's are 500 ppm in rats and 5000 ppm in mice. There was no evidence of teratological effects in the rat or the rabbit at the highest dose tested in both species. While there was evidence of maternal toxicity at 40 mg/kg/d in the rat and 100 mg/kg/d in the rabbits.

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2. DuPont Adsorption of 14C DPX-T6376 on Soil (AI'IR-66-82).

3. DuPont Field Soil Dissipation Study of DPX-T6376 in Delaware, North Carolina, Florida, and Mississippi (AMR 66—82).

4. DuPont Field Soil Dissipation of [Phenyl (U) -14C] Metsulfuron Methyl on United States and Canadian Soils (AMR 476-86).

5. DuPont HL 180-82; 90 day feeding one generation Reproduction Study in Rats.

6. DuPont HLO-61-85; Chronic Feeding Study with Concurrent Two Generation Reproduction Study in Rats - Chronic.

7. DuPont HLO-65-85 Chronic Feeding Reproduction Phase.

8. DuPont HLR-524-84 Two generation, Four Litter Reproductive Study in Rats.

9. DuPont HLR 137-83 Subchronic Dermal Study (21 Days) in Rabbits.

10. DuPont HLR 463-84 Ninety-Day and Long Term Feeding Study in Mice.

11. Ally Herbicide Product Monograph

THE COMMONWEALTH OF MASSACHUSETTS
EXECUTIVE OFFICE OF ENERGY AND ENVIRONMENTAL AFFAIRS

Department of Agricultural Resources
251 Causeway Street, Suite 500, Boston, MA 02114
617-626-1700 fax: 617-626-1850 www.mass.gov/agr

Paclobutrazol

Review Conducted by MDAR and MassDEP for Use in Sensitive Areas of Rights-of-Way in Massachusetts

January 2012

Massachusetts Department of Environmental Protection

Massachusetts Department of Agricultural Resources

Active Ingredient Paclobutrazol:

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1. INTRODUCTION

The review presented here was initiated by the request for the addition of Cambistat® (EPA Reg. No. 74779-3), containing the active ingredient paclobutrazol, to the Massachusetts Rights-of-Way Sensitive Area Materials List. Paclobutrazol is a tree growth regulator that provides a tool for utility arborists to limit the size and growth of trees and shrubs in power line and utility rights-of-way corridors. Tree growth regulator products such as Cambistat® are regularly applied in high visibility locations such as parks, historic downtowns, residential areas and other areas where trees have a cultural value (Paul Sellers, NSTAR, pers. comm.). The utility industry is seeking approval of Cambistat® for use in sensitive areas in order to have the ability to use this product in the same locations that happen to be located within areas of rights-of-way that are regulated by 333 CMR 11.00.

The regulations specified in 333 CMR 11.00 provide standards, requirements and procedures for the use of herbicides in vegetation management in areas of rights-of-way, while minimizing the potential impacts to human health and the environment. Specific restrictions exist for sensitive areas within rights-of-way, including the list of herbicides that have been specified as acceptable for use in these sensitive areas. The herbicides included on the Sensitive Area Materials List have been evaluated to further scrutinize potential risks to sensitive receptors in these areas. The review presented here is the evaluation of the active ingredient paclobutrazol and products for use in sensitive areas of rights-of-way.

Paclobutrazol (PBZ) was first registered by U.S. EPA in 1985. At the time of preparation of this review in 2011, PBZ was undergoing registration review by U.S. EPA to determine whether it continues to meet the FIFRA standard for registration (U.S. EPA, 2007A). As part of the registration review process, a summary document was issued (U.S. EPA, 2007B). This document includes a factsheet describing the use of this active ingredient, the status of human health and ecological risk assessments, and the problem formulation and scope of work necessary to support the registration review at U.S. EPA.

Additional information was obtained from documents issued by the European Food Safety Authority (EFSA) that evaluated PBZ for use as a plant growth regulator on winter oilseed rape. The evaluation data package of the EFSA assessment included various documents describing data summaries, scientific evaluations, risk assessments, and conclusions of the peer review. The documents consulted for the review presented here included the Draft Assessment Report (DAR) (EFSA, 2006), the Additional Report to the DAR (EFSA, 2010A) and the Conclusion of the Peer Review (EFSA, 2010B).

The secondary review documents generated by the regulatory agencies U.S. EPA and EFSA are primarily based on the consideration of registrant-submitted studies in support of product registration. These studies are generally classified as Confidential Business Information (CBI) and therefore not available for review outside of these agencies. Additional information from scientific publications and other government documents was also considered, when available and as needed, for the assessment described in this review.

This document describes a review of the chemical and physical properties, product use characteristics, environmental fate characteristics and toxicity data. Environmental concentrations of PBZ were estimated using screening-level simulation models and calculation

methods. The risks to classes of organisms that are most likely to be exposed, including aquatic organisms and soil invertebrates, were characterized. The exposure to groundwater resources was also assessed.

The review described herein was conducted according to the established procedures and criteria for review of herbicide products for use within sensitive areas of Rights-of-Way (ROW) (MDAR, 2011). These review procedures and criteria address both the herbicide active ingredients as well as the “inert” or “other” ingredients, more specifically the surfactants.

2. CHEMICAL AND PRODUCT IDENTITY AND PROPERTIES

2.1. Chemical Identity and Properties

- Common Chemical Name: Paclobutrazol (***PBZ*** *acronym will be used*)
- IUPAC name: 2*RS*,3*RS*-1-(4-chlorophenyl)-4,4-dimethyl-2-(1*H*-1,2,4-triazol-1-yl) pentan-3-ol
- CAS No.: 76738-62-0

Paclobutrazol (PBZ) is a plant growth regulator belonging to the triazole chemical class (U.S. EPA, 2007B). The nomenclature is summarized in Table A1.1 in Appendix 1. PBZ is a racemic mixture of the (2R, 3R) and (2S, 3S) enantiomers. Chemical and physical properties are listed in Table A1.2 in Appendix 1.

2.2. Formulated Product

The product considered in this review, Cambistat®, is a suspension concentrate containing 22.3% PBZ. The MSDS document (Rainbow Treecare, 2011) for this product indicates that the formulation also contains propylene glycol at an unspecified concentration. No other ingredients were specified in the MSDS document (Rainbow Treecare, 2011).

Propylene glycol (PG) is a colorless, odorless liquid which is generally recognized as safe (GRAS) by the U.S. Food and Drug Administration (FDA) in 21 CFR § 184.1666 for use as a direct food additive under the conditions prescribed. It is approved by the U.S. FDA for certain indirect food additive uses. PG has a wide range of practical applications such as antifreezes, coolants and aircraft deicing fluids; solvents; food; flavors and fragrances; cosmetics and personal care products; pharmaceuticals; chemical intermediates; plasticizers; and thermoset plastic formulations (DOW, 2006). PG is not acutely toxic (single dose, high exposure). It is essentially non-irritating to the skin and mildly irritating to the eyes. Available data indicate that propylene glycol is not a skin sensitizer or a carcinogen. PG is not volatile and is miscible with water. It is not expected to bioaccumulate and it is not acutely toxic to water organisms except at very high concentrations (OECD/SIDS, 2001). Given the characteristics and regulatory status of this ingredient, propylene glycol was not further evaluated for this review.

Proprietary information on the other formulation ingredients was obtained. Two of the proprietary ingredients can be classified as surfactants. One of the surfactants belongs to a class of surfactants that has been approved for use in sensitive areas of rights-of-way in Massachusetts (MDAR, 2010A and B). Consequently, this ingredient did not have to undergo additional review and passed the surfactant policy portion of the review process for the sensitive area materials list. Nevertheless, both surfactants were included in the evaluation of proprietary ingredients.

The proprietary ingredients were evaluated as part of the review process for addition to the Sensitive Area Materials List, but cannot be disclosed here for proprietary reasons. In most cases, a quantitative or semi-quantitative evaluation was conducted based on available toxicity

endpoints and estimates for maximum soil, surface water and ground water concentrations. In some cases, only a qualitative evaluation was possible. Based on these evaluations, it was concluded that these compounds are of a nature and/or present at levels in the product such that use of it as directed would not cause unreasonable adverse effects to human health and the environment.

2.3. Mode of Action

PBZ is a cell elongation and internode extension inhibitor that retards plant growth by inhibition of gibberellins biosynthesis. Gibberellins stimulate cell elongation. When gibberellin production is inhibited, cell division still occurs, but the new cells do not elongate. The result is shoots with the same numbers of leaves and internodes compressed into a shorter length. Reduced growth in the diameter of the trunk and branches has also been observed. Another response of trees to treatment with PBZ is increased production of the hormone abscisic acid and the chlorophyll component phytol, both beneficial to tree growth and health. PBZ may also induce morphological modifications of leaves, such as smaller stomatal pores, thicker leaves, and increased number and size of surface appendages, and increased root density that may provide improved environmental stress tolerance and disease resistance (Chaney, 2005). PBZ also has some fungicidal activity due to its capacity as a triazole to inhibit sterol biosynthesis (Chaney, 2005; U.S. EPA, 2007B; BCPC, 2000).

3. USE PATTERN AND APPLICATION CHARACTERISTICS

3.1. Use as Tree Growth Regulator

The use pattern of PBZ considered in this review is as a tree growth regulator, more specifically as a tree growth retardant (TGR). PBZ was one of the three active ingredients that were used by utility arborists in the 1980s. The products were applied by trunk injection as a formulation containing alcohol solvents. Due to problems associated with trunk injection of these products (e.g., tree injury and wood discoloration) there was a decline of the use of TGRs. In 2005, PBZ was the only remaining TGR for use on trees. Modifications in formulations and application methods, satisfactory performance as a TGR and benefits to overall tree health resulted in a rebound in the use of PBZ. Current formulations of PBZ TGRs such as Cambistat® for TGR use, such as Cambistat®, are applied as a water suspension by soil injection or basal drench (Chaney, 2005).

PBZ is also registered for use on ornamental plants grown in containers in nurseries, greenhouses and interior landscapes. It is also used on turf to control annual grasses and broadleaf weeds, to reduce the mowing frequency and to increase turf density.

3.2. Application Methods and Rates

PBZ formulated as Cambistat® is applied by soil injection or application as a basal drench. The species-specific dose rate is determined by measuring the tree diameter at breast height (DBH). Based on the dose rate information on the product label, it can be calculated that the dose rate of active ingredient is in the range of 4.1 g (0.009 lbs) to 202.5 g (0.446 lbs) PBZ per individual tree. Dose rates may be reduced by 25 to 30% based on consideration of canopy size and structure, stressed or declining tree status, or the presence of a confined or compromised root system. Given the use pattern of treating individual trees, the application rate expressed in mass use per acre has not been established. The water suspension of PBZ can be injected approximately 2-6 inches deep at 50 to 200 psi as close to the tree trunk as possible. Alternatively, the water suspension can be poured into a shallow trench around the tree. Retreatment may be done every 3 years or until the effects from the previous application subside (Rainbow Treecare, 2011).

4. ENVIRONMENTAL FATE OF PACLOBUTRAZOL

4.1. Environmental Fate Parameter Summary

The environmental fate properties of PBZ are summarized in Table A2.1 in Appendix 2. The mobility and persistence characteristics are described in more detail in the following two sections.

4.2. Mobility

PBZ has been characterized as a compound with a moderate potential for mobility in soil and water environments (U.S. EPA, 2007B). The summary document for registration review prepared by U.S. EPA (2007B) documents that laboratory batch equilibrium studies indicated that PBZ has the capacity to be mobile under certain conditions. Studies with nine US soils ranging in texture from sand to silt loam indicated values for the soil adsorption coefficient K_D in the range from 1.3 to 23.0 ml/g. Adsorption appeared to increase with an increase in soil organic matter content and a decrease in soil pH. In the draft assessment report prepared by the United Kingdom (EFSA, 2006) adsorption data for 13 soils are summarized that show K_D values in the range of 0.8 – 21.3 ml/g with a geometric mean of 4.3 ml/g. The ketone metabolite showed on average a slightly higher affinity for adsorption to soil with K_D values in the range of 2.1 – 13.5 with a mean of 8.0 across 6 soils.

Results from laboratory soil column leaching experiments summarized in U.S. EPA (2007B) indicated low mobility in the experiments using methine-labeled PBZ in soils ranging in texture from sand to clay-loam. The experiments using triazole-labeled PBZ showed low mobility in columns of sand and sandy loam soils, and mobility in loamy sand and clay loam soils. In all cases, the majority (58.6 – 90.7%) of applied PBZ aged residue did not leach out of the upper 10 cm of the treated soil columns.

An issue noted in the draft assessment report (EFSA, 2006) was the identification in a column leaching study of the degradate hydroxyl triazole at a concentration of 12 μg/L in the leachate. Even though this degradate was not detected in the soil metabolism experiments, the observation in the column leaching experiment raised concerns for risks to groundwater and a data gap was identified. This data gap was addressed in the additional report to the DAR (EFSA, 2010A). Groundwater exposure modeling using additional soil degradation and adsorption data for the degradate hydroxyl triazole showed a maximum concentration of the degradate in groundwater (80^{th} percentile annual average concentration in leachate leaving the top 1 m soil layer) did not exceed 0.1 μg/L except in one of the six scenarios, where it was modeled at a concentration of 0.1192 μg/L. The modeling study concluded that the potential for the degradate hydroxyl triazole to reach groundwater at high concentrations is low.

PBZ is unlikely to volatilize to any significant extent owing to a low estimated vapor pressure. The octanol-water partitioning coefficient (log K_{OW}) of 3.2 indicates a potential for this chemical to bioaccumulate in fish. A fish bioaccumulation study, which was only conducted for 14 days, showed BCF factors of 20x for edible tissues (day 3), 248x for non edible tissues (day 3), and 44x for whole fish (day 10) (U.S. EPA, 2007B).

Although characterized as moderately mobile in laboratory studies, no significant movement of PBZ was detected in field studies in agricultural soils. In the orchard studies, PBZ residues (parent plus degradate) were detected at 10% or less of total applied in soils with depths of 48 inches in the California study, 24 inches in West Virginia study, and 48 inches in the Florida study. These depths are the maximum depths sampled at each study. No information was provided on the nature or type of soils in the summary document. The PBZ ketone metabolite was predominately detected in the subsurface soil layers, also at insignificant levels (U.S. EPA, 2007B).

A scientific publication by Baris et al. (2010) provided information regarding the potential of PBZ to impact groundwater from its use on turf areas. PBZ was included in a comprehensive evaluation of water quality monitoring data and assessment. This evaluation considered water quality data for a large number of turf-related pesticides from 44 studies involving 80 golf courses in the US over a 20-year period. PBZ was found in 3/440 groundwater samples, with the highest detection at 4.2 μg/L.

4.3. Persistence

PBZ has been characterized as an environmentally stable compound in soil and water environments (U.S. EPA, 2007B). Laboratory studies with US loam and silt-loam soils indicated that PBZ degraded with a half-life of more than 1 year under both aerobic and anaerobic conditions.

Summaries of laboratory half-lives, normalized to 20 °C with moisture content at field capacity, show values in the range of 43 to 618 d with a mean of 183 d (6 soils) (EFSA, 2006). Data from field studies in the UK and Italy indicated dissipation half-lives of 58 to 389 d with a mean of 114 d. Field accumulation studies conducted for a period of 4 to 8 years with annual applications of PBZ showed no apparent build up of PBZ residues except in one of the 7 sites.

The degradation pathway of PBZ, described in EFSA (2006), occurs via the ketone analog, (2RS)-1-(4-chlorophenyl)-4,4-dimethyl-2-(1,2,4-triazol-1-yl)-pentan-3-one, which was detected in the aerobic soil metabolism study at approximately 18% of total applied and at less than 10% in other soil studies. The ketone analog is degraded via separation of the 1-H-1,2,4-triazole moiety. The 1,2,3-triazole moiety was only observed at a maximum of 3%. Degradation of the 1,2,4-triazole proceeds via triazole acetic acid and hydroxyl triazole. Hydroxy triazole was identified in a soil column leaching study but was not observed in any of the soil metabolism studies (EFSA, 2006).

The major ketone-metabolite is less persistent than the PBZ parent with half-lives of 23 – 90 d (mean of 54 d) in an aerobic degradation study with 3 soils. A minor metabolite 1,2,4-triazole is even less persistent as indicated by its half-life of 6.3 – 12.3 d (mean 9.5 d) in aerobic soil degradation studies.

Field dissipation studies from the US showed PBZ residues that were persistent and relatively mobile. Half-lives of PBZ residues ranged from 450-950 days for orchard soils in California,

West Virginia, Florida and 25 weeks to 36 weeks in agricultural soils in Mississippi, North Carolina, and Illinois.

Laboratory studies indicated that PBZ is relatively stable to degradation by hydrolysis. More than 94 percent of PBZ was still present after 30 d in pH 4, 7 and 9 solutions, respectively (U.S. EPA, 2007B). PBZ did not undergo appreciable photolysis in water when exposed to light in pH 7 buffer. More than 96 percent of PBZ was still present after 10 d of exposure (U.S. EPA, 2007B). In the presence of light, degradation of PBZ in soil was slightly accelerated with a calculated half-life of 188 d. It was concluded that soil photolysis is unlikely to be a significant route of dissipation (EFSA, 2006).

Degradation in a water-sediment system was reported in EFSA (2006). The data indicate a low degradation rate in both the water and the whole system. The half-life determined for the whole system was 164 d, with most of the PBZ remaining in the water phase.

5. MAMMALIAN TOXICITY

With regard to the existing toxicological data of PBZ, the work plan for registration review by U.S. EPA (2007B) makes reference to RfD/Peer Review reports from 1986 and 1994 among the primary resources for the status update. A more recent review and evaluation of toxicological information was organized by the European Food Safety Authority (EFSA) as part of the peer review of the pesticide risk assessment of PBZ in European Community. The more up-to-date information available in the EFSA-organized peer review documents was the primary source of information for review presented here. The EFSA-organized review was initiated in 2006 (EFSA, 2006), subsequently withdrawn, and then resubmitted along with additional toxicological information, and was completed in 2010 (EFSA, 2010A and B). Information on the mammalian toxicology from registrant-submitted studies considered in these review documents is summarized below.

Acute toxicity, irritation and sensitization

PBZ exhibits moderate acute toxicity by the oral route in the species tested. The LD_{50} is 1954 mg/kg in male rats and 1336 mg/kg in female rats; 490 mg/kg and 1219 mg/kg in male/female mice, respectively; 542 mg/kg and 400-640 mg/kg in male/female guinea pigs, respectively; and 835 mg/kg and 937 mg/kg in male/female rabbits, respectively. New data for rats indicated an oral LC_{50} > 2000 mg/kg.

Acute dermal LC_{50} values are greater than 2000 mg/kg in rats and greater than 1000 mg/kg in rabbits. Overall, PBZ is of low acute toxicity by the dermal route.

Acute inhalation studies showed a 4h-LC_{50} value of greater than 2 mg/L particulate to rat indicating moderate toxicity by inhalation.

Skin irritation studies with rats (5 repeated applications) and with rabbits (single application) indicated that PBZ is slightly irritating to skin. Eye irritancy studies with rabbits indicated mild irritancy to the eye. PBZ is not a skin sensitizer based on the results of studies with guinea pigs.

Overall, the acute toxicity data indicate that PBZ is of moderate acute toxicity by the oral and inhalation routes and of low acute toxicity by the dermal route. PBZ is slightly irritating to skin and eye and is not a skin sensitizer.

Toxicokinetics

In the rat, absorption was rapid and extensive (88-95%) and did not show saturation at a high dose. Absorbed material was readily oxidized to PBZ diol, which was subject either to excretion or to further oxidation to the carboxylic acid. Biotransformation was limited to the tertiary butyl moiety, with no metabolism detected in either the triazole or chlorinated phenyl rings. Male rats oxidized a greater proportion of PBZ to the carboxylic acid than did female rats.

A small proportion of radioactivity equilibrated into the tissues and was subsequently eliminated. The highest concentrations of radioactivity were seen in the liver after a high or low dose. There was no evidence of bioaccumulation.

Excretion at a low dose was relatively rapid with more than 70% of radioactivity excreted within 48 hours. The delay in excretion in the high dose animals (>70% excretion not achieved until 72 hours after dosing) and the significant amount of radioactivity in faeces (well beyond normal transit time) were due to significant enterohepatic recirculation. In cannulated rats, biliary excretion at a low dose represented >50% and 70% of the administered dose in females and males, respectively. In cannulated rats, 5% was excreted as unchanged parent.

In the dog, following a single oral low dose, radioactivity was rarely absorbed reaching peak concentrations in plasma and blood within 1 hour and declining below the limits of detection by 72 hours. Most of the radioactivity was associated with plasma. Elimination was faster than for rats with >75% of radioactivity eliminated in urine and faeces within 24 hours. At 168 hours after dosing, there was almost a complete absence of radioactivity in all tissues examined (with the exception of the liver in one animal). There was no evidence of bioretention of PBZ or its metabolites in dogs.

Short-term toxicity

The short-term toxicity of PBZ was investigated by the oral route in rats (90 days) and dogs (90 days and 1 year), and by the dermal route in rabbits (21 days).

The liver is the target organ of PBZ oral toxicity in the rat. Signs of liver toxicity (clinical chemistry changes, increased weight and marginal increases in hydropic and fatty changes) were observed in males and females at 1250 ppm (93 and 107 mg/kg/day in males and females, respectively). These effects were accompanied by decreases in food consumption and body weight gain. There were no effects at 250 ppm (20 mg/kg/day). An overall short-term NOAEL of 20 mg/kg/day was identified for the rat from this subchronic study.

Similar findings were observed in the dog. Liver toxicity (clinical chemistry changes, increased weight, enzyme induction and ballooned hepatocytes), accompanied by decreases in food consumption and body weight gain, was observed from a dose of 75 mg/kg/day (in the 1-year study). There were no effects at 15 mg/kg/day (1-year study). Therefore, an overall short-term NOAEL of 15 mg/kg/day was identified for the dog from the chronic study.

A repeat dose dermal toxicity study in rabbits showed no signs of systemic toxicity up to 100 mg/kg bw/day.

No short-term studies in the mouse were available; however, results from the mouse carcinogenicity study do not indicate that the mouse was more sensitive to PBZ than rats or dogs.

Genotoxicity

The mutagenic, clastogenic, and aneugenic potential of PBZ was studied in several *in vitro* test systems using bacteria and mammalian cells and *in vivo* test systems in rats and mice. PBZ was negative in an *in vitro* bacterial reverse mutation assay and an *in vitro* gene mutation test in mouse lymphoma cells. No clastogenic effects were seen in an *in vitro* human lymphocyte cytogenetics test, two *in vivo* rat cytogenetics tests and two *in vivo* mouse micronucleus tests. No evidence of DNA damage or repair was noted in an *in vivo* UDS assay. PBZ had no effect on

either fertility or dominant lethality in mice in a dominant lethality test. Based on these *in vitro* and *in vivo* mutagenicity tests, it was concluded that PBZ is not genotoxic.

Long-term toxicity and carcinogenicity

The chronic toxicity and carcinogenicity of PBZ was investigated in two standard dietary studies in rats and mice.

The liver is the target organ of PBZ oral chronic toxicity in the rat. Signs of liver toxicity (decreases in plasma triglycerides in females and increases in plasma BUN levels in females, increased liver weights in males and females and increased incidence of hepatocyte steatosis/hypertrophy in males and females) were seen at the top dose of 1250 ppm. These were accompanied by decreases in body weight gain and food consumption in females. At 250 ppm, body weight gains were still significantly reduced in females and liver steatosis was still significantly increased in males. There were no toxicologically significant effects at 50 ppm (2.2 and 2.8 mg/kg bw/day in males and females, respectively).

In mice, the target organ of PBZ oral chronic toxicity was also the liver (and related fat metabolism), as indicated by increased liver weights, increased severity of steatosis in males and reduced serum cholesterol in males and triglyceride levels in females at the top dose level of 750 ppm. There were no toxicologically significant effects at 125 ppm (14 and 16 mg/kg bw/day in males and females, respectively).

There was no evidence of carcinogenic effect of PBZ in rats or mice.

Reproductive and developmental toxicity

The reproductive toxicity of PBZ has been investigated in a 2-generation study in the rat and in pre-natal developmental toxicity studies in rats and rabbits.

In the 2-generation study, dietary administration of PBZ caused general toxicity in the parental animals at the top dose of 1250 ppm, observed as increased incidence of chromocryorrhea and thickened eyelids and increases in liver weights and associated histopatology (centrilobular fatty changes). PBZ also caused adverse effects in the young F_1 and F_2 offspring at the top dose of 1250 ppm, observed as a reduction in pup bodyweight gains, increased incidence of chromodacryorrhea, thickened eyelids, dental malocclusion and twisted snout and increases in liver weights and associated histopatology (centrilobular fatty changes). However, fertility mating performance, litter size and pup survival were not affected by treatment. Accordingly, on the basis of this study, it can be concluded that PBZ is not a specific hazard to fertility and reproductive performance, as no effects were seen up to the top dose of 1250 ppm (117 mg/kg/day in males and 124 mg/kg/d in females). Classification for effects on fertility was not required. However, a NOAEL of 250 ppm (23 mg/kg/day in males and 25 mg/kg/day in females) was identified for general parental toxicity and for effects on the offspring.

New information confirmed the increased incidence of dental malocclusion and twisted snout observed in the F1 and F2 offspring is unlikely to be a developmental effect of PBZ. As the same finding was detected in the treated adult animals of the F_O generation with a similar incidence, it

was considered that, at most, it represents a generalized, unspecific toxic effect of PBZ to pups and adult animals.

Two developmental toxicity studies in the rat are available. In the first study, a NOAEL for maternal toxicity of 100 mg/kg bw/day was identified on the basis of reduced food consumption and deaths at the next dose level of 250 mg/kg bw/day (top dose). Developmental toxicity was limited to delayed ossification of a number of bones. A no-effect level for developmental effects could not be established because a statistically significant, dose-related increase in partially ossified 7th transverse process was apparent at all dose levels (from 40 mg/kg bw/day = LOAEL). There was also an increased incidence of cleft palate (1.28% vs 0% in concurrent and historical controls) at the highest dose which may have been the consequence of maternal toxicity (including lethality); however a direct teratogenic effect could not be ruled out.

In a second study, conducted to determine a no-effect level for developmental toxicity, there were no effects on the dams up to the top dose tested (100 mg/kg bw/day = NOAEL for maternal toxicity). Developmental toxicity was limited to an increased incidence of partial ossification of the transverse processes of the 7th cervical vertebra and extra 14th rib at 40 and 100 mg/kg bw/day. There were no developmental effects at 10 mg/kg bw/day (NOAEL for developmental toxicity).

In two separate developmental toxicity studies in the rabbit, there was no evidence of developmental effects up to the top dose tested of 125 mg/kg bw/day at which maternal toxicity (reduced body weight gain and food consumption) was observed. Additional information confirmed that the reported skeletal variants are chance findings unrelated to treatment and that PBZ is not a developmental toxicant in the rabbit up to maternally toxic dose levels.

Overall, therefore, PBZ causes developmental toxicity in rats, manifested as a low incidence of cleft palate (1.28% affected foetuses vs 0% in concurrent and historical controls), seen in a preliminary study at 240 mg/kg bw/day and in one of the two definitive studies at the top dose of 250 mg/kg bw/day. The lack of the observation in the second definitive study is consistent with the findings of the other studies as the highest dose tested in the second study was only 100 mg/kg bw/day. Although the cleft palate occurred in the presence of severe maternal toxicity (including lethality), there is no evidence that the finding is a secondary non-specific consequence of maternal toxicity. PBZ also causes small changes in the incidences of common skeletal variants in the rat (partial ossification of the transverse processes of the 7th cervical vertebra and extra 14th rib). Although these occurred both in the absence of observable maternal toxicity and in the presence of maternal toxicity, they were observed in isolation, did not show a consistent pattern and were not accompanied by any effects on other foetal parameters, such as body weight. Nevertheless, as cleft palate toxicity is very rare in the rat and is not considered to be a secondary non-specific consequence of maternal toxicity, classification for developmental toxicity in a category representing substances with possible risk of harm to the unborn child was considered to be appropriate.

Tolerances and other guidelines

Since there are no food uses of PBZ, no maximum residue levels for PBZ have been established for agricultural commodities in the US (U.S. EPA, 2007A). A drinking water standard is also not

established in the US. The derivation of a maximum allowable concentration in drinking water of 66 μg/L is described in EFSA (2010A). This value is based on an allowable daily intake of 0.022 mg/kg/day.

In the context of the evaluation water quality data and assessment of pesticide impacts, Baris et al. (2010) calculated a lifetime health advisory level following procedures used by U.S. EPA and reported a value of 460 μg/L for PBZ.

6. ECOTOXICITY

Data on the ecotoxicity of PBZ were available in EPA's summary document for registration review (U.S. EPA, 2007B), in the draft assessment report (EFSA, 2006), and in the additional report to DAR (EFSA, 2010A). The toxicity data considered in these regulatory reviews were primarily obtained from registrant-submitted data. Summaries of these studies are available in review documents generated by EFSA (2006 and 2010A). The ecotoxicity information is described below. A data summary table is included in Appendix 3.

6.1. Acute and Chronic Toxicity of Paclobutrazol

Avian

PBZ is slightly toxic to practically non-toxic to avian species based on acute oral toxicity data (see Appendix 3) ranging from >2100 to >7913 mg/kg b.w. and the ecotoxicity categories as defined by U.S. EPA (2011A). The sub-acute dietary toxicity data indicate that PBZ is slightly toxic to mallard and bobwhite quail. The no-observed-effect-concentration (NOEC) corresponded to a daily dose of 3106 mg/kg/d for mallard and 101 mg/kg/d for bobwhite quail, respectively. A reproductive toxicity effect study with mallard ducks indicated a NOEC that corresponded to a daily dose of 38.8 mg/kg bw/d.

Aquatic Species

The acute toxicity data for bluegill sunfish, rainbow trout, mirror carp and sheepshead minnow listed in Appendix 3 show a range of LC_{50} values from 23.6 to 27.8 mg/L. These data indicate that PBZ is slightly acutely toxic to fish. Aquatic-phase amphibian toxicity data were available from a study with toad tadpoles that indicated a slight toxicity of PBZ with a LC_{50} value of 11 mg/L.

Chronic toxicity data for rainbow trout indicated a NOEC of 3.3 mg/L. The endocrine activity was studied in zebra fish (*Danio rerio*). No activity was found at levels up to and including the mean measured concentration of 3.2 mg/L. No NOEC could be established. However, statistically significant reductions in vitellogenin levels were observed at all test concentrations in male fish, while non-significant decreases were observed in top dose levels in female fish. Fish gonadal screening assays for endocrine activity in zebra fish showed no histopathological treatment-related effect on the gonads, liver, and kidneys.

Bioaccumulation

Bioaccumulation factors in bluegill sunfish were approximately 44 in whole fish, 20 in muscle, and 248 in viscera. During the depuration period the accumulated residues were rapidly eliminated, with ^{14}C-residue concentrations returning to background levels within 7 days.

Aquatic invertebrates

The toxicity data for aquatic invertebrates, including water fleas (*Daphnia magna*), mysid shrimp (*M. bahia*), and Pacific oyster larvae (*C. gigas*), indicate that PBZ is slightly toxic to this class of organisms with LC_{50} data in the range of >9 to 35 mg/L. Chronic toxicity data for water fleas (*D. magna*) indicated a 22-d NOEC value of 0.32 mg/L based on effect on *D. magna* length.

Aquatic plants

For non-vascular aquatic plants, the toxicity of PBZ to green algae (*Selenastrum capricornutum*) the 96-hr E_bC_{50} and E_rC_{50} [1] for PBZ were 7.2 mg/L and >15.2 mg/L, respectively. For blue-green algae (*Anabaena flos-aquae*) these values were estimated to be greater than 23.2 mg/L. PBZ is more toxic to vascular aquatic plants. The data for duckweed (*Lemma gibba*) 7-d E_bC_{50} and E_rC_{50} for PBZ were 8.2 μg/L (0.0082 mg/L) and 28.3 μg/L (0.0283 mg/L), respectively.

Terrestrial Vertebrates

Mammalian toxicity was presented in Section 5. The reader is referred to that section for information relative to the ecotoxicity for terrestrial invertebrates.

Bees

Honey bees (*Apis hellifera*) exposed to PBZ by contact with doses in the range of 2 to 40 μg per bee and orally by dosing at 2 μg per bee indicated contact and oral LD_{50} values that were determined to be >40 μg/bee and >2 μg/bee, respectively.

Earthworms

Clitelate adult earthworms (*Eisenia foetida*) were exposed at a single test concentration of 1000 mg/kg soil for 14 days. The 14 d LC_{50} value was >1000 mg/soil. No deaths, abnormalities in behavior or external condition were observed at the test concentration. There was a statistically significant 20% reduction in body weight. The 14 d LC_{50} value for the ketone degradate was also determined to be >1000 mg/soil.

6.2 Acute and Chronic Toxicity of Metabolites

Metabolites that are considered relevant for ecotoxicological risk assessment are the ketone analog of PBZ, 1,2,4,-triazole and hydroxyl triazole (EFSA, 2006 and 2010). The available toxicity data for these metabolites are listed in Table 6.1. The data for PBZ are included for comparison.

[1] The EbC50 value is the concentration at which 50% reduction of biomass is observed; the ErC50 is the concentration at which a 50% inhibition of growth rate is observed (Bergtold and Dohmen, 2011).

Table 6.1. Comparison of acute (LC_{50}/EC_{50}) and chronic (NOEC) ecotoxicity data of paclobutrazol and its metabolites ketone, 1,2,4-triazole, and hydroxy-triazole (EFSA, 2006 and 2010).

Species	Paclobutrazol (mg/L)	Ketone (mg/L)	1,2,4-triazole (mg/L)	Hydroxy-triazole (mg/L)
ACUTE				
Fish (*O. mykiss*, 96-h LC_{50})	23.6	-	498	-
Invertebrates (*D. magna*, 48-h EC_{50})	27.8	-	>100	-
Algae (*P. subcaptitata*, 72-h EC_{50})	7.2	-	12	-
Aquatic plants (*L. gibba*, 7-d EC_{50})	0.0283	0.57		>100
CHRONIC				
Fish (*O. mykiss*, NOEC)	3.3		100	

The data in Table 6.1 show that the metabolites are less toxic than the parent compound PBZ. In the case of the ketone metabolite, only aquatic plants have been tested. Such an approach was considered acceptable in the review by EFSA (2006) as this group of organisms is considered more sensitive to the parent compound than the other aquatic organism groups tested and the ketone is closer in structure to the parent and is formed higher up in the metabolic pathway.

7. EXPOSURE ASSESSMENT

In order to perform an ecological risk assessment, the exposure assessment is needed to estimate the environmental concentrations associated with the application of PBZ. Given the application method of PBZ as tree growth regulator by soil injection around the base of a tree, the exposure assessment was done for the environmental compartments surface water, ground water, and the soil in and immediately adjacent to the injection area. Potential off-site migration routes that are likely to be relevant for the applied product include runoff and leaching through the soil toward surface water and groundwater. Off-target migration through spray drift is not considered given that the application method is by soil injection.

7.1 Surface Water Exposure

The exposure to surface water was estimated using a Tier I screening-level exposure model that is used by the Environmental Fate and Effects Division of U.S. EPA's Office of Pesticide Programs (EFED-OPP) to assess the risk of a pesticide product to the environment. This Tier I model is designed as a coarse screen and estimates expected concentrations from several basic chemical and environmental fate parameters, and application information. This GENeric Expected Environmental Concentration Program (GENEEC) uses a candidate chemical's soil/water partition coefficient and degradation half-life values to estimate runoff from a ten hectare field into a one hectare by two meter deep pond. GENEEC is a program to calculate both acute and chronic generic expected environmental concentration values. It considers reduction in dissolved pesticide concentration due to adsorption of pesticide to soil or sediment, incorporation into the soil, degradation in soil before wash-off to a water body, direct deposition of spray drift into the water body, and degradation of the pesticide within the water body. It is designed to mimic the more sophisticated PRZM-EXAMS model simulation (Tier II model in EFED-OPP) (U.S. EPA, 2011B).

The model requires input values for parameters associated with application and the characteristics of the active ingredient. An application rate for Cambistat expressed in amount of product or active ingredient per acre has not been established because of its use pattern of treating individual trees. The application rate for the model input was set at 3 lbs per acre for a single application. This application rate was based on the annual maximum rate as for applications on turf (4 application per year of 0.75 lbs PBZ per acre = 3 lbs PBZ per acre) as was used with the exposure modeling described in U.S. EPA (2007B). This rate can be considered a reasonable high-end estimate of a per-acre rate considering the use pattern of treating individual trees. Since the product is injected into the soil, the option of granular application was selected in order to not simulate aerial spray drift. The incorporation depth of 6.0 inches was selected to be representative of the recommended injection depth used with the application of this product.

The values of the chemical and environmental fate properties were a K_D of 2.7 (lowest non-sand value in EFSA (2006), soil half-life of 437 days (according to GENEEC manual instructions for selecting conservative parameter value), aquatic half-life of 164 d, and photolysis half-life of 365 d (stable). The GENEEC input and output for this scenario are included in Appendix 4.

The model output shows that the simulated peak generic environmental concentration was 19.98 µg/L (0.01998 mg/L), the maximum concentration was 19.34 µg/L at 21 d and 17.35µg/L at 90

days. It is important to note that the GENEEC model simulates conservative pesticide concentrations for aquatic ecological exposure assessments.

7.2. Groundwater Exposure Assessment

The exposure of herbicides to groundwater was evaluated by using the SCI-GROW model simulations. SCI-GROW (***S***creening ***C***oncentration ***I***n ***GRO***und ***W***ater) is a screening model which the Office of Pesticide Programs (OPP) in EPA frequently uses to estimate pesticide concentrations in vulnerable ground water (U.S. EPA, 2011C). The model provides an exposure value which is used to determine the potential risk to the environment and to human health from drinking water contaminated with the pesticide. The SCI-GROW estimate is based on environmental fate properties of the pesticide (aerobic soil degradation half-life and linear adsorption coefficient normalized for soil organic carbon content), the maximum application rate, and existing data from small-scale prospective ground-water monitoring studies at sites with sandy soils, low organic matter content (on average <1%) and shallow ground water (on average 14 ft).

Pesticide concentrations estimated by SCI-GROW represent conservative or high-end exposure values because the model is based on ground-water monitoring studies which were conducted by applying pesticides at maximum allowed rates and frequency to vulnerable sites (i.e., shallow aquifers, sandy, permeable soils, and substantial rainfall and/or irrigation to maximize leaching). In most cases, a large majority of the use areas will have ground water that is less vulnerable to contamination than the areas used to derive the SCI-GROW estimate.

The input parameters for SCI-GROW include the application rate, soil degradation (soil half-life value) and a soil mobility parameter (soil organic matter-water partitioning coefficient (K_{OC}). Following the instructions for input value selection, the annual application rate used was 3 lbs PBZ per acre (as described with surface water assessment), the soil half-life was 285 days (see surface water assessment), and the K_{OC} was 106 mL/g (determined from the lowest non-sand K_D value used above with surface water and the corresponding organic carbon content of 2.5%: K_{OC} = K_D/ fraction OC).

The SCI-GROW simulated screening-level groundwater concentration using the selected input values as described above was 14.3 µg/L(see also Appendix 5).

7.3. Soil Exposure at the Application Site

The exposure of PBZ in the soil following the injection of the product in a band around the trunk base of a tree was estimated by considering the amount of product applied according to label instruction to a tree with an assumed trunk diameter and assumed dimensions of a soil band around the trunk base of the tree that would received the initial application of the product. Details on the calculation of the PBZ concentration in the soil of the treated area around a tree are shown in Appendix 6. The initial peak concentration of PBZ in the treated soil band was calculated to be 150 mg/kg dry soil.

8. RISK CHARACTERIZATION

8.1 Ecological Risk Assessment

Ecological risk characterization integrates the results of the exposure and ecotoxicity data to evaluate the likelihood of adverse ecological effects. For most ecological risk assessments, U.S. EPA uses a deterministic approach or the quotient method to compare toxicity to environmental exposure. In the deterministic approach, a risk quotient (RQ) is calculated by dividing exposure estimates by ecotoxicity values, both acute and chronic. RQ values are then compared to established levels of concern (LOCs). The LOCs are criteria used by U.S. EPA to indicate potential risk to non-target organisms. The RQ ratio is a screening-level method that identifies high- or low-risk situations (U.S. EPA, 2011D).

As pointed out earlier, the environmental compartments that are most likely to be exposed to the products or residues thereof are the soil in and adjacent to the treatment area, and surface and ground water. The ecological risk assessment will therefore consider the risk to aquatic organisms and earthworms. Based on the localized application of product in the soil of tree rooting area it can be expected that the exposure to terrestrial vertebrates and birds is going to be minimal. The groundwater is not considered as a relevant environmental compartment for ecological risk, but will be addressed separately for a drinking water assessment.

The RQ values for the groups of organisms considered in this ecological risk assessment are listed in Table 8.1 along with the corresponding toxicity endpoint and EEC data. The RQ are compared with the established LOCs (U.S. EPA, 2011D).

Table 8.1. Ecological risk assessment data for paclobutrazol.

Species	Toxicity Endpoint	Endpoint Value	EEC	RQ	LOC[1]
AQUATIC INVERTEBRATES		(mg/L)	mg/L	EEC/ Endpoint	
Daphnia magna	Acute 96-h LC_{50}	35	0.01998	0.0006	0.5
Mysid Shrimp	Acute 96-h LC_{50}	>9	0.01998	>0.0022	0.5
Pacific oyster larvae	Acute 48-h EC_{50}	>10	0.01998	>0.0020	0.5
Daphnia magna	Chronic NOEC	0.32	0.0173	0.0541	1
FISH					
Bluegill sunfish	Acute 96-h LC_{50}	23.6	0.01998	0.0008	0.5
Rainbow trout	Acute 96-h LC_{50}	27.8	0.01998	0.0007	0.5
Mirror Carp	Acute 96-h LC_{50}	26.0	0.01998	0.0008	0.5
Sheepshead minnow	Acute 96-h LC_{50}	24.3	0.01998	0.0008	0.5
Rainbow trout	Chronic 22-d NOEC	3.3	0.01735	0.0053	1

Species	Toxicity Endpoint	Endpoint Value	EEC	RQ	LOC[1]
AMPHIBIAN (aquatic phase)					
Bufo bufo (toad)	Acute 72-h LC_{50}	11	0.01998	0.0018	0.5
AQUATIC PLANTS					
Green algae	Growth E_bC50	7.2	0.01988	0.0028	1
	Growth E_rC50	15.2	0.01988	0.0013	1
Blue-green algae	Growth E_bC50	>23.2	0.01988	>0.0009	1
	Growth E_rC50	>23.2	0.01988	>0.0009	1
Duck weed	Growth E_bC50	0.0082	0.01988	**2.4244**	1
	Growth E_rC50	0.0283	0.01988	0.7025	1
EARTHWORMS		mg/kg soil	mg/kg soil		
Eisenia foetida	Acute 14-d LC_{50}	>1000	150	0.15	0.5

[1] LOC values established by U.S. EPA, 2011D.

Comparison of the RQ values with the established LOCs indicates that all are well below the established LOCs, except for duckweed. The low RQ values indicate low potential for adverse effects on most aquatic organisms. The RQ value for growth effects on duckweed biomass indicates that there is some potential for adverse effects for vascular aquatic plants. This can be expected from exposure of plants to a growth retardant compound. Given the slight exceedance of the LOC and that the effect is on growth, it is not expected that the impact would be detrimental for this group of organisms. In addition, the estimated surface water concentration is a screening-level assessment that is based on conservative assumptions. The screening-level concentration can be considered to be representative of a high-end exposure and will not occur in most situations.

Earthworms are organisms that could be exposed to PBZ following a soil injection application around the perimeter of a tree trunk. However, the level of exposure associated with such an application would not exceed the LOC for this group of organisms. PBZ soil concentration and associated exposure by earthworms would also decrease over time as the PBZ is gradually taken up by the tree.

Acute and chronic risk to mammals from potential exposure to PBZ residues in food was assessed in the review by EFSA (2006). The exposure assessment was based on the application rate of 0.0557 lbs PBZ per acre as proposed for use on an oil seed crop. The food intake rate considered was for a medium-sized herbivorous mammal and residue characteristics were

representative for application to a leafy crop. The estimated theoretical exposure was 2.18 mg PBZ/kg bw/d (acute) and 0.51 mg PBZ/kg bw/d (chronic). The toxicological endpoints used in this risk assessment were the LD_{50} for male mouse (490 mg PBZ/kg bw) and developmental toxicity NOAEL of 10 mg/kg bw in rat. A developmental end-point was used as this was the lowest longer-term end-point and therefore considered to represent the worst-case scenario. Using this information, EFSA calculated a toxicity exposure ratio (TER) of 224.8 for acute risk and 19.6 for chronic risk. Based on comparison with the levels of concern (TER values of greater than 10 for acute risk and greater than 5 for chronic risk are not of concern), EFSA concluded that the acute and chronic risks to mammals were not a concern.

It should be pointed out that the developmental endpoint is toxicologically not considered a long-term or chronic endpoint. Developmental exposure is typically viewed as being of intermediate exposure. The evaluation of chronic toxicity using a toxicity value based on intermediate exposure is not protective.

Alternative long-term toxicological end-points for mammalian species identified by EFSA were the NOAEL of 23.2 mg/kg bw/d for parental toxicity and 108 mg/kg bw/d for reproductive toxicity. Evaluation of chronic risk based on these endpoints results in TER values of 45 (parental) and 212 (reproductive) which can be considered protective. Given that there was no estimated theoretical exposure of medium duration generated in the EFSA evaluation, it is not possible to properly evaluate the developmental endpoint, (i.e., the most sensitive endpoint) based on the available information. It is likely that if an exposure estimate of intermediate exposure were to be generated, that it would indicate that developmental effects would not be of concern—however, such a conclusion cannot be drawn based on the current information.

The risk to earthworm-eating mammals was assessed by considering the residue estimates in earthworms that were based on estimated bioconcentration factors and concentrations of PBZ in soil. The residue estimates were converted to a daily dose that had a value of 0.18 mg PBZ/kg bw/d. Compared to the long-term NOAEL of 10 mg/kg bw/d, the toxicity exposure ratio was 55.6. This value exceeds the trigger value (level of concern) of 5 (a TER value greater than 5 for chronic risk is not of concern) and therefore it was concluded that the risk to earthworm-eating mammals was not a concern.

The risk assessments described above were done assuming an application scenario representative for the use of PBZ on oilseed crops, which includes broadcast foliar applications resulting in residues that mostly occur on above ground plant material. The use scenario for tree treatments, in contrast, is by soil injection around the tree trunk perimeter, which results in a much more localized application of the material in the soil. It is likely that tree trunk application results in higher concentrations of PBZ occur in soil compared to soil concentrations associated with broadcast foliar applications. However, it is unlikely that small mammals would feed exclusively and permanently in a treated tree trunk area. It is therefore unlikely that the exposure of mammals to PBZ in a tree trunk treatment scenario would exceed the exposure levels as described above in the broadcast oil seed crop scenario. The risks to mammals from PBZ exposure associated with tree trunk applications is not expected to be significant.

8.2 Comparison of Estimated Groundwater Concentration with Drinking Water Standards

The screening-level groundwater concentration of 14.3 ppb is below the maximum allowable concentration in drinking water of 66 µg/L reported in EFSA (2010A). This screening-level concentration is also below the lifetime health advisory level of 460 µg/L calculated by Baris et al. (2010).

With the consideration of the risk to groundwater it is important to consider that the screening-level concentrations generated by the SCI-GROW model represent conservative or high-end exposure. In most cases, the use areas will have ground water that is less vulnerable to contamination than the areas used to derive the SCI-GROW estimate. In addition, the model does not consider buffer zones around a drinking water well as is required by ROW regulations.

9. RISK MITIGATION AND USE PRECAUTIONS

The product label (Rainbow Treecare, 2011) offers a number of precautionary practices that may be taken to mitigate potential risks to non-target organisms. Given that the product is a plant growth inhibitor, non-target plants have the highest potential to be affected by PBZ exposure through off-site movement of applied product. This potential risk to non-target plants is addressed by warning and precautionary language on the label:

Localized stunting or injury of turfgrass or other non-target plants immediately adjacent to the treatment site may occur if the product flows off of the application site.

Avoid basal drench applications on inclines and other areas where treated soil is likely to be washed away from the base of the tree by rainfall or irrigation.

Shrubs and/or herbaceous ornamentals next to treated trees may be affected if their roots extend into the treatment zone.

The risk to aquatic organisms is addressed by language that states that the product should not be applied directly to water, to areas where surface water is present or to intertidal areas below the mean high water mark.

Other label language addresses the treatment of trees that produce products for human consumption such as maple trees, and fruit and nut trees.

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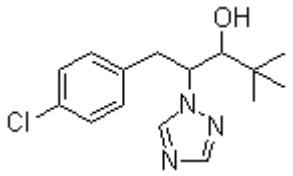
Appendix 1

Table A1.1. Paclobutrazol structure and nomenclature

Paclobutrazol	
Structure	
Molecular Formula	$C_{15}H_{20}ClN_3O$
IUPAC Name	(2*RS*,3*RS*)-1-(4-chlorophenyl)-4,4-dimethyl-2-(1*H*-1,2,4-triazol-1-yl)pentan-3-ol
CAS name	(a*R*,ß*R*)-*rel*-ß-[(4-chlorophenyl)methyl]-a-(1,1-dimethylethyl)-1*H*-1,2,4-triazole-1-ethanol
CAS Number	76738-62-0
PC Code	125601

Source: U.S. EPA, 2007B

Table A1.2. Physical and chemical properties of paclobutrazol

Parameter	Value	Source
Molecular Mass	293.8	EFSA, 2006[1)]
Melting/Boiling point	164 °C/ 384 °C	EFSA, 2006
Density	1.23 g/cm^3 (20 °C)	EFSA, 2006
Vapor Pressure	1.9 × 10^{-6} Pa (very slightly volatile)	EFSA, 2006
Volatility from water (Henry's constant	2.39 × 10^{-5} Pa m^3 mol^{-1}	EFSA, 2006
Solubility in water	26 mg/L (20 °C)	BCPC, 2000[2)]
Octanol-water partitioning constant (Log P)	3.2	BCPC, 2000

1) EFSA, 2006, Section B.2.1; [2)] British Crop Protection Council, 2000 (The Pesticide Manual).

Appendix 2

Table A2.1. Environmental fate properties for mobility and persistence of paclobutrazol

Parameter	Value	Source
Hydrolysis	Stable: <6% degradation after 30 d at pH 4,7, and 9	U.S. EPA, 2007B
Photolysis in water	Stable: < 5% degradation after 10 d at pH 7	U.S. EPA, 2007B
Aerobic soil metabolism (half-life)	> 1 yr 43 – 618 d (mean 183 d)	U.S. EPA, 2007B EFSA, 2006 [1)]
Anaerobic soil metabolism (half-life)	> 1 yr	U.S. EPA, 2007B
Field dissipation (half-life)	450-950 d in orchard US soils 175 – 252 d in agricultural US soils	U.S. EPA, 2007B EFSA, 2006 [1)]
Aquatic metabolism (half-life)	164 d	EFSA, 2007B
Soil Adsorption Coefficient (K_D) mL/g	1.3 – 23.0 0.8 – 21.3 (mean of 4.3)	U.S. EPA, 2007B EFSA, 2006 [1)]

[1)] EFSA, 2006: Volume 3, Annex B, Section 8.

Appendix 3

Table A3.1. Summary of ecotoxicity data for paclobutrazol. Data were obtained from U.S. EPA (2007B), EFSA (2006) and EFSA (2010).

Species	Toxicity	Endpoint	Values
AVIAN			(mg/kg b.w.)
Mallard	Acute Oral[1]	LD50	>7913
Japanese Quail	Acute Oral	LD50	>2100
Mallard	Sub-acute dietary[2]	LD50	>3106
		NOEC	3106
Bobwhite Quail	Sub-acute dietary	LD50	>2791
		NOEC	101
Mallard	Long-term/ Reproductive[3]	NOEC	38.8
AQUATIC INVERTEBRATES			mg/L
Daphnia magna (flea)	Acute	48 hr EC50 static	35
Mysid Shrimp	Acute	96 hr EC50 semi- static	>9
Pacific oyster larvae	Acute	48 hr EC50 static	>10
Daphnia magna	Chronic	22-d NOEC semi-static	0.32
FISH			mg/L
Bluegill sunfish	Acute	96 hr EC50 semi- static	23.6
Rainbow trout	Acute	96 hr EC50 semi- static	27.8
Mirror Carp	Acute	96 hr EC50 semi- static	26.0
Sheepshead minnow	Acute	96 hr EC50 static	24.3
Rainbow trout	Chronic	28-d NOEC	3.3
AMPHIBIAN (aquatic phase)			mg/L
Bufo bufo (toad)	Acute	24-h LC50	11
VERTEBRATES (terrestrial)			mg/kg
Rat	Acute Oral[1]	LD50	1954 (male) 1336 (female)
Mouse	Acute Oral	LD50	490 (male) 1219 (female)
Guinea Pig	Acute Oral	LD50	542 (male) 400-640 (female)

Species	Toxicity	Endpoint	Values
Rabbit	Acute Oral	LD50	835 (male) 937 (female)
BEES			µg/bee
Honey bee *(Apis mellifera)*	Acute	48-hr LD50	>40 (contact) >2 (oral)
EARTHWORMS			mg/kg soil
Eisenia foetida	Acute	14-d LC50	>1000
AQUATIC PLANTS			mg/L
Green algae	Growth	96-h E_bC50 96-h E_rC50	7.2 15.2
Blue-green algae	Growth	96-h E_bC50 96-h E_rC50	>23.2 >23.2
Duck weed	Growth	7-d E_bC50 7-d E_rC50	0.0082 0.0283

[1] Exposed by a single oral dose

[2] Exposed by diets containing PBZ for 5 d

[3] Exposed by diets containing PBZ for 21 wks

Appendix 4

GENEEC Surface Water Model Input and Output:

```
RUN No.**** FOR Paclobutrazol  ON  Trees     * INPUT VALUES *
  ---------------------------------------------------------------------
  RATE (#/AC)  No.APPS &  SOIL SOLUBIL  APPL TYPE NO-SPRAY INCORP
  ONE(MULT)  INTERVAL   Kd  (PPM )  (%DRIFT)  ZONE(FT) (IN)
  ---------------------------------------------------------------------
 3.000( 3.000)  1  1    2.7  26.0  GRANUL(  .0)  .0  6.0


  FIELD AND STANDARD POND HALFLIFE VALUES (DAYS)
  ---------------------------------------------------------------------
  METABOLIC DAYS UNTIL HYDROLYSIS  PHOTOLYSIS  METABOLIC COMBINED
  (FIELD)  RAIN/RUNOFF  (POND)   (POND-EFF)  (POND)   (POND)
  ---------------------------------------------------------------------
  437.00    2     N/A  365.00-45260.00  164.00  163.41


  GENERIC EECs (IN MICROGRAMS/LITER (PPB))   Version 2.0 Aug 1, 2001
  ---------------------------------------------------------------------
    PEAK   MAX 4 DAY   MAX 21 DAY  MAX 60 DAY  MAX 90 DAY
    GEEC   AVG GEEC    AVG GEEC   AVG GEEC   AVG GEEC
  ---------------------------------------------------------------------
    19.98    19.88     19.34     18.17     17.35
  ---------------------------------------------------------------------
```

Appendix 5

SCI_GROW model input and output for Paclobutrazol:

```
             SCIGROW
            VERSION 2.3
     ENVIRONMENTAL FATE AND EFFECTS DIVISION
        OFFICE OF PESTICIDE PROGRAMS
      U.S. ENVIRONMENTAL PROTECTION AGENCY
           SCREENING MODEL
       FOR AQUATIC PESTICIDE EXPOSURE

SciGrow version 2.3
chemical:Paclobutrazol
time is 6/13/2011 16:34:39
-------------------------------------------------------------------------
Application   Number of    Total Use  Koc   Soil Aerobic
rate (lb/acre) applications  (lb/acre/yr) (ml/g)  metabolism (days)
-------------------------------------------------------------------------
  3.000      1.0      3.000   1.06E+02   285.0
-------------------------------------------------------------------------
groundwater screening cond (ppb) =  1.43E+01
*************************************************************************
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Appendix 6

Estimation of Paclobutrazol concentration in soil band around tree trunk:

Assumptions:

- Diameter of trunk at breast height of 50 inches
- Mass of applied PBZ is 202.5 g (calculated from information on Cambistat Label)
  (833 ml product x 1.09 g/ml x 22.3 % PBZ = 202.5 g PBZ)
- Diameter trunk at ground level is 60 inches
- Soil band treated begins 2 inches from trunk resulting in an inside diameter of soil band of 64 inches
- A 1-foot wide band will initially be exposed to product: Outside diameter of band is 76 inches
- Treatment reaches initially a depth of 1 ft
- Dry bulk density of soil to be 1.3 g/ml

Conversions:	Inside diameter:	64 inches =	162.56	cm
	Outside diameter:	76 inches =	193.04	cm
	Depth	12 inches =	30.48	cm

Calculations:

Area of treated soil band: Calculated by subtracting the areas of the circles with outside and inside diameters:

	Outside diameter:	Inside diameter:		
Circle areas (cm^2):				
(πR^2)	117069.7	83018.95		
Difference between circle areas is band area:			34050.74	cm^2
Volume of treated soil band: (area x depth):			1037867	cm^3
Mass of dry soil is volume x bulk density:			1349227	g
			1349.227	kg
Mass of applied PBZ in band area of soil:			202.5	g
Concentration of PBZ in soil (mg/kg or ppm)			150.086	ppm

THE COMMONWEALTH OF MASSACHUSETTS
EXECUTIVE OFFICE OF ENERGY AND ENVIRONMENTAL AFFAIRS

Department of Agricultural Resources
251 Causeway Street, Suite 500, Boston, MA 02114
617-626-1700 fax: 617-626-1850 www.mass.gov/agr

MDAR
MASSACHUSETTS DEPARTMENT OF AGRICULTURAL RESOURCES

SULFOMETURON METHYL

In addition to the review that is presented below, comprehensive reviews are available from U.S. EPA and USDA Forest Service that incorporate more recent studies and data. The US Forest Service risk assessment report is available at the U.S. FOREST SERVICE webpage, Pesticide-Use Risk Assessments and Worksheets:

https://www.fs.fed.us/foresthealth/protecting-forest/integrated-pest-management/pesticide-management/pesticide-risk-assessments.shtml

Sulfometuron-methyl Registration Review documents are available at www.regulations.gov in docket ID: EPA-HQ-OPP-2012-0433

Review conducted by MDAR and MassDEP for use in Sensitive Areas of Rights-of-Way in Massachusetts

COMMON TRADE NAME(S): Oust

CHEMICAL NAME: N-[4,6-dimethylpyrimidin-2-yI) amino-carbonyl -2 methoxycarbonylbenzenesulfonaflhide

CAS NO: 74222-97-2

GENERAL INFORMATION

Sulfometuron methyl, the active ingredient in the herbicide Oust, is a member of the group of sulfonylurea herbicides. Sulfometuron Methyl is a broad-spectrum selective weed control agent used in non-crop areas. Oust is applied pre- or post-emergence which provides control against many broad-leaf weeds and grasses through contact and residual activity. (15)

ENVIRONMENTAL FATE

Mobility

The mobility of sulfometuron methyl has been reported in literature and the database available is complete. Sulfometuron methyl is a weak acid (pKa 5.2) and consequently, adsorption coefficients were calculated for various soils at pH values of 5, 6, and 7. In a low organic matter I soil (1%) the adsorption coefficients were 2.0, 0.8 and 0.3 at the respective pH values. This study indicates that sulfometuron methyl is more strongly adsorbed to soil as the pH decreased, and as organic matter increases. (15)

Soil thin layer chromatography and adsorption coefficients were performed and calculated for four standard soils. Kd values ranged from 0.71 to 2.85 and Rf values ranged from 0.33 to 0.85 indicated a moderate mobility. In

addition, soil column studies using the same four soils indicate a moderate to moderately high mobility pesticide. Koc values calculated from the soil Kd values range from 61 to 122 which is lower than the EPA guideline of 400. (101) In a field mobility study, sulfometuron methyl was applied to soil tubes in five locations (Delaware, North Carolina, Oregon, Colorado, and Saskatchewan, Canada) at a rate of 1 lb a.i./Acre. There was no report of rainfall at these sites. Each application was made at a different time making it difficult to compare results. Samples were taken for a minimum of a year and at some for two years, and at 8 cm (3 in) intervals to 32 cm (12 inches). Results indicate that sulfometuron methyl is moderately mobile under most conditions. One surprising fact is that immediately after application, all locations had detectable residues in a layer below the top layer of soil, and in two locations (Colorado and Oregon) in the deepest layer sampled. All locations except Delaware also had detectable residues at the 24-32 cm layer at other times during the study. There are also indications that sulfometuron methyl would leach further than the deepest soil layer which was sampled. (102)

Persistence

Sulfometuron methyl is degraded by microbial action, photo-decomposition and by hydrolysis at acidic pH's. The photolysis half-life on soil is between 1 to 2 weeks and in distilled water, approximately 160 hours. The hydrolysis half-life at pH 2 and 5 is 100 and 475 hours respectively. At neutral or basic pH's, sulfometuron methyl is stable to hydrolysis. (15,100, 101)

Reports indicate that the overall rate of sulfometuron methyl degradation in soil depends on pH and soil moisture content. Half-lives of one week were reported under laboratory conditions, but field studies at neutral pH revealed greater persistence. Increased soil moisture content resulted in increased degradation rates, but only approximately 10%. (15, 101)

The soil half-life is reported as four weeks with longer times in colder conditions. A review of available studies, however reveals that the shortest half-life was six weeks in Delaware. In the same study the half-life ranged from six weeks to one year in Oregon. (15, 102)

The reported half-life of four weeks is relatively short and would not be cause for concern. However, it seems evident that in most circumstances it may be significantly longer. In all cases reported in this study, the half-life was six weeks or longer and a more realistic estimate may be closer to two months. Another point discussed in the literature is the lack of any significant degradation during the cold periods of the year. Applications in the late fall could lead to longer half-lives and thereby more potential for increased leaching.

The field study discusses the faster degradation rates of sulfometuron methyl in the east as possibly attributable to the more acidic and moister soils in the east. This is certainly true and may in fact have contributed to shorter half-lives, but a point which is not discussed was the timing of the applications. The two western sites were treated in early to mid-July, whereas the western sites were treated in the fall. Saskatchewan was treated in late July, but the climate at that location is cooler and becomes much colder.

TOXICITY REVIEW

Five animals per sex per group were gavaged with sulfometuron methyl suspended in corn oil at a dosage of 5,000 mg/kg. Gross pathological examination revealed slight weight increase in the lungs that were pale red with grey foci in males and similar lung effects in one female. In addition, four females had a pink thymus and one had a slight liver weight. The oral LD50 in male and female ChR-CD rats was determined to be greater than 5,000 mg/kg. (110)

The inhalation LC5O was tested in groups of five male and five female Crl:CD rats. Rats were exposed to control air or test concentrations of either 6.4 or 11 mg/L. There were no clinical or pathological differences between controls or test groups. The inhalation LC5O was greater than 5.0 mg/L (111) while sulfometuron methyl was tested at 6.4 and 11 mg/L. The EPA cutoff for LC5O concentration is 5 mg/L.

Acute skin absorption LD5O tests were performed on five male and five female New Zealand white rabbits. Doses of 2,000 mg/kg of pesticide were applied to abraded skin on the back of the rabbit. Clinical signs in males were

sporadic weight loss, slight erythema 1 to 2 days after treatment and diarrhea at 11 days. Gross pathological examination showed no changes due to the test material. The dermal LD5O in rabbits was greater than 2,000 mg/kg. (112)

In a separate acute dermal LD5O test, four groups of five adult male and one group of five adult female New Zealand rabbits were used. Groups of males were dosed at the following levels: 1,500 mg/kg, 2,000 mg/kg, and 8,000 mg/kg and the females were dosed at 2,000 mg/kg. Clinical signs in all the groups of males were moderate to mild redness and sporadic weight loss. The animals in the two highest dose experienced mild swelling, the 2,000 mg/kg group showed moderate swelling while the 1,500 mg/kg group had slight swelling. Clinical signs in the females were severe to mild redness, severe to slight swelling and sporadic weight loss. There were no compound related pathological observations. There was one death in the male 2,000 kg/mg group, but it was not believed to be related to the compound. The LD5O for the acute skin absorption in rabbits was greater than 2,000 mg/kg. (116)

Eye irritation studies were performed by placing 10 mg of solid test material in the conjunctival ac of each of two albino rabbits. There were no corneal or iritic effect. However, there was redness (1 hour to 1 day; not washed eyes and mild for 1 hour unwashed eyes); swelling (1 to 4 hours unwashed eyes) and no discharge was observed. Both washed and unwashed eyes were normal within 1 to 2 days. (113)

In guinea pigs, both primary skin irritation and sensitization tests were run. Ten animals per group were exposed to 0.05 ml of either a 50% or a 5% suspension of sulfometuron methyl. The 50% suspension showed mild to no skin irritation response in 24 hours and no irritation at 48 hours. The 5% suspension reproduced no skin irritation. There was no sensitization response. (114)

The oral LD5O test was conducted with the formulation using young male and female adult Crl:CD rats, five rats per group. 5,000 mg/kg was administered by gavage in a 25% suspension in corn oil. The only clinical finding was alopecia in males. Gross pathological examination showed in both males and females slightly heavy lungs that were pale to pale red with red to dark red foci and white mottling in 1 to 3 animals. The LD5O is greater than 5,000 mg/kg. Additionally in a range finding study, no mortalities were seen in doses from up to 7,500 mg/kg. (115)

Nine male albino rabbits were tested for eye irritation studies. The right eyes were treated with 0.1 ml (61.8 mg) of test material. The left eyes served as untreated controls. Results indicated a transient localized area of slight corneal cloudiness in 2 of the 6 unwashed eyes. The eyes returned to normal in 2 to 3 days. Two of the three eyes treated and washed showed a transient localized area slight corneal cloudiness and mild conjunctivitis with no iritic effects. The washed eyes returned to normal within 3 to 4 days. This compound was considered a slight to mild irritant.
(117)

Skin irritation tests were conducted on six male albino rabbits. Doses of 0.5 g of solid pesticide (moistened with saline) were applied to two intact and two abraded skin areas on each rabbit. Each rabbit serves as its own control; treated areas were compared to adjacent untreated areas. Observations and scoring were done by the method of Draize (118) and at 24 and 72 hours after exposure. The compound was not found to be a primary irritant on either intact or abraded skin of rabbits. (119)

Primary skin irritation tests were performed on ten guinea pigs. The procedure was the same as used in testing the technical sulfometuron methyl. Doses of 0.05 ml of a 50% suspension of the pesticide in dimethyl phthalate were used. The 50% suspension caused mild to no irritation in five of the animals. No irritation was caused by the 5% suspension. No sensitization response was observed. (120)

Subchronic and Chronic Studies (Mammalian)

Male and female CD-i mice were fed diets to which had been added 0, 100, 1,000, or 7,500 ppm (0, 13.3, 133, or 997 mg/kg) (a) sulfometuron methyl for 90 days. Hematological evaluations were conducted on all mice (tail cut bleeding at approximately 1, 2 and 3 months after study initiation. All mice were sacrificed and necropsied at 90 days. Organs were weighed and examined histologically. Male mice fed the diet containing 7,500 ppm pesticide showed reduced

mean body weights and weight gains. Growth of the 100 and 1,000 ppm groups of males and all treated females was the same as that in the control group. No mortalities occurred. (121)

Hemolytic effects were seen as a result of dietary exposure to sulfometuron methyl in all groups. Significant increases in leukocyte count were found in the 7,500 ppm (997 mg/kg) males. There were statistically significant changes in other blood parameters that were not dose related. Mean absolute and relative liver weights were elevated in all male treatment groups. Histological examination revealed bile stasis in five of ten males in the 7,500 ppm group. In the females, a slight increase in relative liver weight and increased hepatocellular cytoplasmic granularity was observed. Decreases in both mean and relative thymus weights were observed in all treated male groups. Thymic cortical atrophy occurred in three males in the 7,500 ppm group and one male in the 100 ppm group. Because of low frequency of occurrence 7,500 and 100 ppm and absence in the 1,000 ppm group, the thymic cortical atrophy is not considered to be related to the decreased thymus wrights. Based on the observed hemolytic effect, there was no NOEL from this study.

In a second mouse study, five groups of 80 males and 80 female Crl:CD-1 (1 CR)BR mice were fed diets containing one of the following concentrations of sulfometuron methyl: 0. 5, 20, 100, or 1,000 ppm (0, 0.66, 2.66, 13.3,133 mg/kg) for 18 months. Food consumption was monitored throughout the study, mice were weighted and hematological evaluations were performed at regular intervals. At 18 months, mice were sacrificed and necropsied. Mean body weights and mean body weight gains in all treatment groups except for the 1,000 ppm female group were comparable to control groups. Sporadic changes in weight gain were observed in that group.

(a) In these discussions the assumptions made for conversion of ppm (diet) to mg/kg/D were:

SPECIES BODYWEIGHT (kg) INTAKE ((kg)
Rat 0.35 0.020 Mouse 0.03 0.004 Dog 10 0.4
(133) When data was presented as ppm the does was estimated in mg/kg and is presented in parenthesis.

Mild anemia was observed in the female 1,000 ppm group as evidenced by statistically significant decreases in erythrocyte count, hemoglobin concentration and hematocrit. There was also a significant increase in mean corpuscular volume and platelet count. While the hematological results appear to differ from those in the 90 day mouse study, the data indicate that there were several statistically significant changes in some blood parameters at the three month (90 day) sampling time which were not apparent at other sampling times. However, although reticulocyte smears were made, they were not evaluated and it cannot be ascertained that a response to a hemolytic effect actually occurred. If it did, a NOEL in this strain of mice for a hemolytic effect at 90 days in the 18 month study would be 5 ppm. There was a non-dose related but, statistically significant increase in the incidence of amyloidosis in the female 1,000 ppm groups, but no specific target organ was identified. The overall NOEL for dietary intake of sulfometuron methyl for male and female mice was 1,000 ppm (133 mg/kg) and 100 ppm (13.3 mg/kg) respectively under the conditions of this study based on body weight, body weight gain, clinical pathology and pathological findings. (124)

Groups of 16 male and 16 female CD rats were fed diets containing 0, 100, 1,000, 5,000 ppm (0, 5.7 57, 285 mg/kg) sulfometuron methyl. At 1, 2 and 3 months after the study initiation, hematological, urological and clinical chemistry evaluations were performed. At the end of the study, ten rats from each group were sacrificed and evaluated pathologically. There were no differences between treatments and controls in body weight, weight gain, food consumption and food efficiency. There were no mortalities. The only clinical sign observed was alopecia in three males in the 100 ppm group. The male 5,000 ppm treatment group showed slightly elevated mean leukocyte counts, increased mean relative number of lymphocytes and decreased mean relative number of neutrophils. Due to the effects of white blood cells in male 5,000 ppm group, the NOEL dietary concentration i this study was 1,000 ppm (56 mg/kg/D).
(122)

Four groups of five male and five female New Zealand white rabbits were dermally exposed to either 1, 125, 500, or 2,000 mg/kg, six hours per day for 21 consecutive days. After the exposure period, three male and three female rabbits per group were sacrificed for pathological evaluation. The remaining two males and two females from each group were sacrificed and evaluated pathologically following a two week recovery period. Clinical signs observed in rabbits

from all test groups including controls were sporadic weight loss and diarrhea. Histopathological and clinical pathological examination showed no compound-related effects. One rabbit did after the eighth dose from causes not related to the test substance. (123)

Groups of 80 male and 80 female Crl:CD (SD) BR rates were fed diets containing 0, 50, 500 or 5,000 ppm (0, .8, 28.5, or 285 mg/kg) sulfometuron methyl for approximately two years. Hematological, clinical chemistry and urological testing was conducted a 3, 6, 9,12,18, and 24 months. After 12 months, ten male and ten female rats per group were randomly selected, sacrificed and pathologically examined. At 24 months, all surviving rats were sacrificed, necropsied, and examined pathologically.

In the female 5,000 ppm group, food consumption throughout the study was slightly depressed and overall mean weight gain during the first year and mean body weights during the second year were significantly depressed. There were no abnormalities in appearance or behavior observed during the study.

Decreased erythrocyte count and hematocrit in the male 500 and 5,000 ppm groups were observed at the 24 month clinical evaluation suggesting a minimal dose-related hemolytic effect. There were no other compound related hematological, clinical chemistry or urological abnormalities observed. Mean absolute brain weights were significantly lower in the male 5,000 ppm group at both one and two sacrifice times. However, no abnormal gross or histological observation were noted. Mean relative and absolute thymus weight of the 500 and 5,000 ppm males was decreased compared to controls at terminal sacrifice. Mean testes weights of rats in the 5600 and 5,000 ppm groups were less than controls.

Histological examinations revealed dose-dependent increases in the incidence of bile duct hyperplasia and fibrosis in the female 500 and 5,000 ppm groups at the two year sacrifice. Severity of the lesions were minimal to mild, suggesting a slightly toxic effect of sulfometuron methyl on the livers of these female rats.

The NOEL in this strain of rat under these study conditions was 50 ppm (2.8 mg/kg/D). (125)

Oncogenicity Studies

Oncogenic endpoints were evaluated in the chronic mouse and rat studies for sulfometuron methyl. Cr1: CD-i (1 CR) BR mice received 0, 5, 20, 100, or 1,000 ppm sulfometuron in the diet of 18 months. There were no compound related increases in tumor incidence (124). CRL:CD (SD) BR rats received 0, 50, 500, or 5,000 ppm sulfometuron in the diet for two years. There was no increase in frequency of occurrence of tumors in these rats (125). Sulfometuron methyl is not carcinogenic in rats and mice under these conditions.

Mutagenicity Testing

The Ames Salmonella/microsome assay tested the ability of Sulfometuron methyl to revert four strains of Salmonella typhimurium from histidine dependence to histidine independence. The assay was performed both with and without a rat liver homogenate (S-9) activation system. The test substance was found not to be mutagenic for these strains of bacteria under the test conditions at doses from 2.5 to 1,000 mg/plate. (129)

Frequency of chromosome aberrations was tested in CHO cells both with and without metabolic activation (S-9). The doses tested ranged from 300 ug/mI to 10 ng/ml in a half log series. No increase in chromosome aberrations was observed in culture exposed under the test conditions to these concentrations of the test material. (130)

The CHO cell line was used to test mutations in the gene coding for the enzyme hypoxanthineguanine phosphoribosyl transferase (HGPRT) both in the presence and absence of an activation (S-9) system. Concentration of the test material ranged from 0 to .1 mM. No mutagenic activity was detected. (131)

The ability of sulfometuron methyl to induce unscheduled DNA (UDS) synthesis in freshly isolated rat hepatocytes was tested. Concentrations of test material ranged for 1 X 10 -5 to 1.0 mM in half log increments. Under these test conditions, no induction of UDS was detected. (132)

Developmental Studies

Groups of 17 female artificially inseminated rabbits wee gavaged with test material on days 6 to 18 of gestation. Dosage levels were 0, 30, 100, and 300 mg/kg suspended in 0.5% methylcellulose in water. Animals were sacrificed on day 29 of gestation and fetuses wee removed by cesarean section. No treatment-related effects were observed in the maternal clinical observations or gross pathology. There were no statistically significant differences between control and treatment groups in any of the other parameters measured (maternal body weight changes, clinical observations, survival, gross pathology pregnancy rates, numbers and percentages of corpora lutea, implantations, resorptions in each maternal animal, fetal sex, viability and development). Under the conditions of this study, sulfometuron methyl was not considered to be teratogenic in New Zealand white rabbits. (127)
A teratology study was conducted using female Crl:CD (SR) BR rats which were fed a diet containing sulfometuron methyl. Concentrations of 0, 50, 1,000, and 5,000 ppm were used. Thirty-five rats were used as controls, 25 rats were assigned to the 50 and 1,000 ppm group and 15 rats were assigned to the 5,000 ppm group. Rats were fed the test diet on days 6 to 15 of gestation and sacrificed on day 21 of gestation for gross and histological examination. (128)

Rats on the highest dose level gained significantly less weight and ate significantly less feed than controls. The fetuses of this exposure group weighted significantly less than those of the control dams. No other adverse effects were noted in the lower exposure groups. No teratogenicity was demonstrated in this study. The minimum effect level of maternal toxicity and embryofetal toxicity was 5,000 ppm (286 mg/kg) and the NOEL under these study conditions was 1,000 ppm (57 mg/kg). (128) Reproductive studies were performed in conjunction with the 90 day feeding study in rats and the two year feeding study in rats.

In the 90 day feeding study (122), six male and six female rats which had been fed diets obtaining 0,100,1,000, and 5,000 ppm of sulfometuron methyl (for 90 days) were mated and delivered litters. No adverse effects were observed as indicated by fertility, gestation, viability and lactation indies. In addition, there wee no differences between treatment and controls in the mean body weights and survival of weaning pups.

In the two year feeding study (125), 20 rats per group were used in a two generation, four litter reproduction study, initiated 90 days after the start of the long-term feeding study. Fo rats were mated. Females were allowed to give birth and Fla pups wee followed until weaning (21 days) at which time they were sacrificed. Fo females wee again mated, but to different Fo males. Fib pups were delivered and observed. At weaning, 20 males and 20 females were selected from each dietary level (0, 50, 500, and 5,000 ppm) and continued on the treatment for 90 days. Fib rats were bred twice within their respective group, producing F2a and F2b litters. Ten males and ten females from the F2b litters were sacrificed and examined histologically. (125)

During the 90 day feeding period for Fl b rats, body weight and diet consumption were decreased in the female 5,000 ppm group. The number of pus born and the number of pups born alive to the 5,000 ppm groups was consistently lower in both the Fl and F2 generations and was statistically significant for F2b litters. Decreased pup counts may reflect the general health status of the mother as evidenced by decreased body weight and diet consumption of the Fl b 5,000 ppm group. No gross or histopathological changes or effects on organ weights were observed in the weaned F2b rats. The NOEL established, based on this sub-study was 500 ppm (28 mg/kg). (125)

Avian Toxicity

Sulfometuron methyl has been tested in the bobwhite quail and the mallard duck. The **8** day dietary LC5O's were greater than 5,620 and 5,000 ppm respectively. The acute oral LD5O in the mallard duck was greater than 5,000 mg/kg. (101)

Invertebrate Toxicity

The aquatic invertebrate, Daphnia magna was tested and the 48 hour LOSO was greater than 12.5 ppm sulfometuron methyl. (15)

Aquatic Toxicity

Species tested on the aquatic toxicity studies include bluegill sunfish (96 hour) and rainbow trout (96 hour). In both cases the LC5O was greater than 12.5 ppm.
A life stage study was done using the fathead minnow. There were no effects observed on embryo hatch, larval survival or growth at concentrations of 1.2 mg/L or less. (15)

SUMMARY

Sulfometuron methyl is a material both moderately mobile and moderately persistent. A closer look at the material however, reveals that the Oust is applied at the average rate of five ounces of product (3.75 oz a.i.)/acre or 106 grams per acre. These studies were conducted with applications of 1 lb a.i./acre. The lower application rates both minimize the persistence of sulfometuron methyl in soil and thereby diminish the amount of material which is available to leach through the soil. Therefore, sulfometuron may be used if the application rates are kept sufficiently low. This is because the soil organic material and soil microorganisms are able to absorb and degrade lower rates of pesticides.

The oral LD50 in rats for sulfometuron methyl is greater than 5,000 mg/kg and the dermal LD5O is greater than 2,000 mg/kg in rabbits.

The sub-chronic and chronic NOELS are 50 ppm (2.8 mg/kg/D) in rates; 200 ppm (i mg/kg/D) in dogs; and 5 ppm (0.66 mg/kg/D) at 90 days for the reversible hemolytic effect and 100 ppm (13.3 mg/kg/D) at two years in the mouse. This makes the mouse at 90 days the most sensitive species with a transient hemolytic effect, to sulfometuron methyl exposure.

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THE COMMONWEALTH OF MASSACHUSETTS
EXECUTIVE OFFICE OF ENERGY AND ENVIRONMENTAL AFFAIRS

Department of Agricultural Resources
251 Causeway Street, Suite 500, Boston, MA 02114
617-626-1700 fax: 617-626-1850 www.mass.gov/agr

MDAR
MASSACHUSETTS DEPARTMENT OF AGRICULTURAL RESOURCES

TRICLOPYR

In addition to the review that is presented below, comprehensive reviews are available from U.S. EPA and USDA Forest Service that incorporate more recent studies and data. The US Forest Service risk assessment report is available at the U.S. FOREST SERVICE webpage, Pesticide-Use Risk Assessments and Worksheets:

https://www.fs.fed.us/foresthealth/protecting-forest/integrated-pest-management/pesticide-management/pesticide-risk-assessments.shtml

Triclopyr Registration Review documents are available at www.regulations.gov in docket ID: EPA-HQ-OPP-2014-0576

Review conducted by MDAR and MassDEP for use in Sensitive Areas of Rights-of-Way in Massachusetts

Common Trade Name(s): Garlon 3A, Garlon 4

Chemical Name: Triclopyr [(3 ,5,6-Trichloro-2-pyridinyl) oxy] acetic acid

CAS No: 55335—06—3

GENERAL INFORMATION

Triclopyr is a picolinic acid derivative and is marketed as Garlon 3A the triethylamine (TEA) salt (CAS #057213-69-1) and Garlon 4 the butoxyethyl ester ***(CAS#*** 008008-20-6).

Triclopyr is effective against a wide variety of woody plants as a foliar spray, basal spray and when applied to cut surfaces. Triclopyr is absorbed by both plant leaves and roots and is readily translocated throughout the plant. It produces an auxin-type response in growing plants in that it appears to interfere with normal growth processes. Thus, maximal plant response occurs when applications are made soon after full leaf development and when there is sufficient soil moisture for plant growth.

ENVIRONMENTAL FATE

Mobility

Most laboratory and field studies indicate that Triclopyr is a relatively mobile herbicide under most conditions. Soil organic carbon partition coefficients K(oc) were determined for the TEA salt in 12 soils which ranged from 0.081% to 21.7% organic carbon. The K(oc) values range from 12 to 78 (14), indicating that Triclopyr should be mobile in most soils. In the same study the K(oc) values of trichloropyridinol, the major metabolite, were reported to range from 114 to 156 in three soils which were not identified. This indicates that trichloropyridinol is less mobile than Triclopyr and should have moderate mobility in soil(14).

In a laboratory study using sandy loam soil with a low organic matter content (0.62%), 75-80% of the applied Triclopyr leached through a 12 inch soil column between days 11 and 15. Water was applied at the rate of 0.5 inches/day for 45 days. The major degradation product, tricloropyridinol required 13 inches of applied water to elute, nearly twice as much (7.5 inches) as Triclopyr(14).

In a field study, Garlon 3A was applied at the rate of 3 gallons/ acre (9 lbs/acre) to six soils ranging from clays to loamy sands in six states. Rainfall was reported to be normal, but not given. Small amounts of Triclopyr and its metabolites were found in the 6—12 inch and 12-18 inch layers of soil 28 to 56 days after application (14,15). Although an application rate of 9 lbs per acre is rather high, the presence of Triclopyr at those depths should be noted especially since there is a correlation with the previous laboratory studies. In other studies, Triclopyr exhibited significantly lower mobility than had been previously reported. In a field study conducted in Massachusetts, Triclopyr was applied to sandy loam soil at a rate of 0.6 lb/acre. Rainfall was reported as normal, but not given. Triclopyr was never detected below the top ten inch layer of soil at any time during the three month study (100). As part of the same study, Triclopyr was applied to soil columns containing the same soil as in the field study at the rate of 0.6 and 6.0 lbs/acre. Simulated rainfall was applied to the soil columns at a rate of 1 inch per week for a total of 5 inches. Triclopyr was not detected below the top 4 inch layer of soil (100). These results indicate lower mobility than previously reported, but they may reflect the short persistence of Triclopyr in soil rather than its mobility through the soil profile.

Persistence

Soil

Microbial degradation is the primary mechanism by which Triclopyr is degraded in soils to two metabolites (15). Degradation under anaerobic conditions (i.e. saturated soils) is reported to be 5 to 8 times slower than under aerobic conditions (14). Triclopyr in soils is not thought to be degraded to any appreciable extent by chemical hydrolysis and, due to its low volatility, is not thought to volatilize from soil to any great extent (15).

A review by TRW states that Triclopyr "is not considered to be a persistent compound in soils" (95). Studies indicate that under certain conditions the half-life of Triclopyr can be relatively short. The Dow Chemical Company has reported a half-life of 10 days in silty clay loam (96). In a small West Virginia watershed the half-life was estimated as between 14 and 16 days (15). Triclopyr was applied aerially at the rate of 10 lbs/acre, but much of the Triclopyr was intercepted by foliage. Average Triclopyr residues in soil from the treated area of this study, measured on the day of the treatment, were non—detectable in densely wooded areas, 4.4 ppm in lightly wooded areas, and 18 ppm in open areas (15). In a Massachusetts field study, the half—life of Triclopyr was reported as 10 days after the applications of 0.6 and 6.0 lbs/acre Triclopyr to non-target vegetation (100).

Most other studies suggest a much longer persistence for Triclopyr in soil. In a laboratory study, Dow reported a half-life of 46 days for Triclopyr in loam. The loam was maintained in the laboratory at **95 deg** F with moisture at field capacity for the duration of the study (96). A **95 deg** soil temperature and moisture at field capacity are both quite high and indicate that the persistence at less than ideal conditions would be longer. Dow also reports the average half-life of Triclopyr in soil to be 30 days (101). An average half-life of 46 days is reported in the Herbicide Handbook (10) and by Ghassemi et al. (95). In addition, other investigators have reported a half—life in soil of "less than 50 days" at temperatures between 25-35 deg C, and between 79 and 156 days at 15deg C (14). In a field study conducted in Sweden, Garlon 3A was applied at the rate of 2 lbs (a.i.)/acre to eight different forest soils. Residues of Triclopyr persisted for 1 to 2 years, and in some cases in excess of 2 years, at levels approximately 10 percent or less of initial soil residue levels (15). It must be noted that soil temperature levels never exceeded 14deg C (57 deg F) and these temperatures are not favorable to microbial degradation (15). These low maximum temperatures are not typical of year round Massachusetts temperatures, but indicate the increased persistence that may occur when applications are made in the fall and are followed by cold weather.

The variable half-lives reported for Triclopyr indicate that soil half-life may be dependent on the soil and climatic conditions. As in most situations of microbial degradation; cold and, dry or saturated soils decrease the decomposition rate, while warm moist soils increase it.

Aquatic

The fate of the butoxyethyl ester of Triclopyr (TBEE) in water is summarized in Figure 1. This diagram shows the major degradation pathways for the ester in water, but does not include processes such as sediment and particulate adsorption. The fate of the ester in water has also been simulated with a modelling technique by McCall et al., 1988 (115). A recent study by Woodburn (116) with the triethylamine salt of Triclopyr experimentally applied to a lake in Florida also provides useful comparative data on the persistence of Triclopyr degradation products. The degradation path is believed to be TBEE to Triclopyr acid to 3,5,6—trichloro-2-pyridinol (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 (117, 118). Several studies indicate that the half-life of the ester in water can range from 1.5—2 days as a result of photolysis (117, 119). Hydrolysis half—lives are dependent upon water pH and temperature and range from 0.06 d to 208 d in natural waters. They decrease with increasing temperature and increasing pH. Acidic conditions increase the persistence of the ester substantially. The 208 d half—life was observed in natural unbuffered water at pH 5 and 15 °C. Waters with this pH level occur in Massachusetts. One laboratory study has produced contradictory results where the ester was stable to hydrolysis, and little photodegradation of the ester occurred over 9 months (120). This study however was performed with buffered, sterile water. Modelling results for the dissipation of the ester indicate that decay should be fairly rapid with a half-life of 12-18 hours (115).

The acid is short-lived in the aquatic environment with reported half—lives of from 2.1 hours at the water's surface in summer at **40deg** N latitude to 14 hr at 1m water depth in winter (117). The principal decay product of the acid is 3,5,6-trichloro-2-pyridinol (TCP), a transient metabolite in water with half— lives ranging from minutes to one day (121). TCP rapidly degrades into nonhalagenated, low molecular weight organic acids (116,121), with phototransformation playing a larger role than hydrolysis in this process.

Salomon et al. (118) demonstrated a half—life of 3.8-4.3 days at l6-17 deg C for the ester to TCP step in an Ontario Lake. Woodburn (116) added Triclopyr salt to a Florida lake and determined a half—life of 0.5—3.6 d at 300 C for the salt to organic acid step. The time scales of both of these studies are in general agreement with the other data on the time course of breakdown for the ester (or salt) to organic acids. With the exceptions of the Hamaker (120) study and a slow breakdown at pH 5, most studies indicate that TBEE in water is degraded relatively rapidly.

TOXICITY REVIEW

Acute (Mammalian)

The Triclopyr toxicity database has been reviewed in several places including the GEIR on the Control of Vegetation on Utility and Railroad Rights-of-Way in Massachusetts (14), Herbicide Handbook Weed Science Society of America (10), and by the U.S. Forest Service (15). Several Dow Publications review the Triclopyr information (101) and Garlon products (102 and 103).

The oral LD5O for Triclopyr in rats is 729 mg/kg in males and 630 mg/kg in females (15, 101). The rat oral LD5O for combined sexes has been reported as 713 mg/kg (10, 14). Rabbits and guinea pigs are more susceptible to oral administration of Triclopyr with LDSOs of 550 and 310 mg/kg respectively (14, 15, 10). The Garlon products have oral LD5Os of greater than 2000 mg/kg (10, 14, 15, 101, 103, 103).

The dermal LD5Os are greater than 2000 mg/kg in rabbits (Triclopyr), and greater than 3980 mg/kg in rabbits for Garlon 4 and Garlon 3A (101, 102, 103)

The effects of Triclopyr on the eye are dependent on the chemical derivative involved: the butoxyethyl ester found in Garlon 4 is essentially non—irritating (102, 15, 14, and 101), while the triethylamine salt is not only an irritant but can cause serious injury (101, 14, 15). These eye injuries include conjunctival irritation, moderate internal redness and moderate to severe corneal damage which may be permanent (14). An inhalation study showed that 100% of the test rats survived a 1 hour exposure to 3 to 20 dilutions of Garlon 3A in air. Transitory nasal irritation to rats was noted after a 4 hour exposure to Garlon 4 aerosol (14).

Metabolism

Two studies, one dermal and one oral have been done in humans to determine pharmacokinetic and metabolic profiles. Five mg/kg acid equivalent (ae) was applied to the forearm of 5 volunteers in the dermal study. One point five eight percent to 1.11% of the applied dose was absorbed and the percutaneous absorption half -life was 16.8 hours (108). In the oral study, 6 volunteers received 0.1 or
0.5 mg/kg Triclopyr (acid equivalent) in apple juice. The excretion half—life is 5 hours and 80% of the dose is recovered as unchanged Triclopyr in the urine (109). The 20% which was unaccounted for could be attributed to one of several explanations including incomplete collections of urine, incomplete absorption of material or metabolism to an unknown metabolite.

Subchronic/Chronic Studies (Mammalian)

Long—term bioassays have been done using Triclopyr in rats (107) and mice (106). Summaries of these studies, provided by Dow Chemical Company have been reviewed for this discussion.

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 observed in both sexes at doses of 20 mg/kg or greater (107). In the full two year study, the doses were 0, 3, 12 and 36 mg/kg/d. The dose related effects in the males were increased body weight at 12 and 36 mg/kg/d, and in females there was an increase in pigmentation in the proximal tubules at 3, 12 and 36 mg/kg/d. Neither the weight increase in the males nor the increased pigmentation in the females were accompanied by morphological, histological or functional changes. The NOAEL for males and females was reported to be 3 mg/kg/d (107).
In the mouse bioassay, ICR mice received Triclopyr in their diets for twenty-two months. The doses were 0, 50, 250, 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, increase in cholesterol levels and multiple changes in liver functions. Some of the liver changes were also observed in the 1600 and 800 ppm groups. There were decreases in body weights, changes in kidney and urine (at various doses and points in time) and liver effects at the 1250 ppm dose. At 250 ppm there were mild kidney effects and the NOEL was reported as 50 ppm (5.55 and 5.09 mg/kg/d for males and females respectively) (106).

In subchronic studies, the 90 day dietary 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, with kidney effects (decrease in excretion) at 2.5 mg/kg/d (14, 101). Dogs refused to eat food that would result in doses of 30 and 100 mg/kg (104). In a one year study, dogs received doses of 0. 0.5, 2.5 or 5.0 mg/kg/d. Minimal kidney effects were observed at 2.5 and 5.0 mg/kg/d. These findings were considered non—adverse by Dow making the NOAEL 5.0 mg/kg/d and the NOEL 0.5 mg/kg/d (105).

Two monkey studies were done to investigate kidney effects in primates. In one study, the monkeys received 0, 10, 20 or 30 mg/kg/d in diet for 28 days. There was no effect on urinary excretion or other responses observed (101, 104). 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. The effects observed in this study were stool softening and diarrhea (104).

Oncocrenicitv Studies 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 (0, 5.55, 28.6, 143 mg/kg/d for males and 0, 5.09, 26.5 and 135 mg/kg/d for females). The only positive result was an increase in combined incidence of mammary adenomas and adenocarcinomas in the female rats at the high dose. There was no evidence of multiple tumors and the effect was not dose related (107, 106).

Mutagenicitv Testing

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 assay systems, cytogenic assays, and mouse dominant lethal studies (15).

Developmental Studies

The teratology of Triclopyr was investigated using the rabbit model. 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 groups 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 group aborted and one dam in the 75 mg/kg/d group died. In the 25 mg/kg group one fetus had hyperplasia of the aortic arch with pulmonary arterial semilunar valve stenosis. Another fetus had a missing gall bladder. There was a statistically significant but non-dose related increase in resorptions at 10 mg/kg/d. This increase was within historical control variability. The developmental NOEL was reported as 75 mg/kg/d with a slight increase in maternal mortality
(110) Tolerances and Other Guidelines

Tolerances are set for Triclopyr on 5 raw agricultural commodities: grasses, forage (500 ppm); grasses, forage, hay (500 ppm); milk (0.01 ppm); meat, fat and meat by products (except liver and kidney) of cattle, goats, hogs, horses, and sheep (0.05 ppm); and liver and kidney of cattle, goats, hogs, horses, and sheep (0.5) ppm (8).

The Dow internal guideline for inhalation exposure to Triclopyr is 10 milligrams/cubic meter (102, 103).

Avian

The toxic effects of Triclopyr on birds have been investigated in a small number of studies conducted by the Dow Chemical Company. For mallard ducks, acute oral LCSOs are reported at 1,698 mg/kg for unformulated Triclopyr, 3,176 mg/kg for Garlon 3A, and 4,640 mg/kg for Garlon 4. Eight day subchronic oral LC5Os are reported as follows for the various triclopyr formulations:

Triclopyr

mallard duck LC50 = 5,000 ppm bobwhite quail LC50 = 2,935 ppm Japanese quail LC50 = 3,278 ppm

Garlon 3A mallard duck LC50=10,000 ppm bobwhite quail LC50=11,622 ppm *Garlon 4* mallar d duck LC50=l0,000 ppm bobwhite quail LC50=9,026 ppm

Source: (15) The data summarized above indicate low acute and subchronic toxicity to the bird species tested. No field studies on the toxic effects of Triclopyr or its formulations in birds have been reported (15).

Invertebrates

Very little data were available on the invertebrate and microorganism toxicity of Triclopyr. The data reported are primarily for the triethylamine salt (Garlon 3A) and were generated by the Dow Chemical Company.

The data indicate low acute lethal toxicity* to organisms tested, with a 96 hr LC5O of 895 ppm in shrimp, 96 hr LC5O greater than 1000 ppm in crabs, and 48 hr LC5Os ranging between 56 and 87 ppm in oysters (15). The 48 hr LC5O for Daphnia is reported as 1,170 ppm (15). After 72 hours of incubation with 500 ppm of Triclopyr, no apparent effects on growth were observed in six soil microorganisms when compared to a control (15).

No information was obtained on the invertebrate toxicity of Garlon 4, the butoxyethyl ester of Triclopyr.

Aquatic The available information on Triclopyr toxicity to fish indicate a wide response of fish to the two formulations of Triclopyr and to unformulated Triclopyr. The butoxyethyl ester of Triclopyr (Garlon 4) is "highly toxic to fish", based upon the Clarke et al. criteria. The 96 hour LC5O values for rainbow trout and bluegill sunfish are 0.74 and 0.87 ppm respectively (15). The corresponding value for juvenile Coho salmon is 1.3 ppm (122).

The triethylamine salt formulation (Garlon 3A) is "slightly toxic" to fish with 96 hour LC5Os of 552 and 891 ppm for rainbow trout and bluegills respectively. The corresponding values for unformulated Triclopyr are 117 ppm for rainbow trout and 148 ppm for bluegill. Both fish species were less sensitive to Garlon 3A than to the active ingredient (15).

No fish toxicity data are available for 3,5,6—trichloro—2—pyridinol (TCP), the intermediate breakdown product from the Triclopyr acid to the non—halogenated organic acid end product.

Dow Chemical Company reports that in natural soil and aquatic environments, both amine and ester formulations rapidly convert (photodegrade) to Triclopyr acid, which in turn is neutralized to a salt at normal environment pH (5.5-6.5)(15). No information is provided with any of the fish toxicity data on the actual form of Triclopyr present in the test water. The persistence data summarized in a previous section and the simulation results of McCall et al. (115), however provide a description of the probable fate of Triclopyr in the toxicity test tanks. 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 less toxic breakdown products during this period (124).

EXPOSURE ASSESSMENT

For the exposure assessment, we have chosen to analyze the fate of the butoxyethyl ester form of Triclopyr (Garlon 4) in water because of its reported high aquatic toxicity in laboratory studies. Garlon 4 would be applied basally at an average application rate of 0.5 pints per acre for the proposed utility program.

In aquatic organisms, LC5Os greater than 10 ppm are considered to be indicative of only slight toxicity and LC5Os less than 1 ppm are considered to reflect high acute toxicity (Clarke et al., 1970 as referenced in [15]).

Since Garlon 4 contains 61.6% of the active ingredient, this application could distribute 37 mg Triclopyr BEE/m^2. The requested maximum application rate is 2 pints per acre.

Two aquatic exposure scenarios have been constructed to evaluate the potential contamination of non-target surface waters with Garlon 4 from a typical land application. The first, most extreme, and very unlikely scenario is for the case of a static stream traversing a treated acre with a percentage of all of the herbicide applied to the acre running into the water. The second represents a more shallow, static stream or standing water body of much less volume with runoff from a portion of the bordering land.

SCENARIO (1) ASSUMPTIONS: Application rate = 0.5 pint/acre
0.47 L/pint 61.6% active ingredient 20% of herbicide applied to acre runs off density of applied herbicide = 1.0 g/ml

RUNOFF:
0.20 x 0.5 pt/acre x 0.47 L/pt x 0.616 = 0.03 L/acre
RECEIVING WATER: Static stream crossing a treated acre

Dimension: 0.3 x 1.22 x 64 m = 23.4 **in** (volume) DILUTION:

0.03L into 23.4 m = 1.3 mL/m^3

1.3 mL/m x 1 m /10 L = 1.3 x 10 mL/L
1.3 x l0 mL/L x 1 g/ml x l0 mg/g = 1.3 mg TBEE/L

SCENARIO (2)

ASSUMPTIONS: Application Rate = 0.5 pt/acre
0.47 L/pt 61.6% active ingredient **2**

20% of herbicide applied to 3m runs off density of applied herbicide = 1.0 g/ml

RUNOFF:
0.2 x 0.5 pt/acre x 0.47 L/pt x 0.616 x 2.47
x 10 acre/m x 10 mL/L x 3 m = 0.02 mL

RECEIVING WATER: Static stream,

Dimensions: 0.15 x 1 x 5 m = 0.75 m (volume)

DILUTION:

0.02 mL into 0.75 m3 = 0.03 mL/m

0.03 mL/ m x 10 m/L x 10 mg/g x 1 g/ml = <u>0.03 mg/L</u>

The calculations presented above illustrate that the probable immediate post—runoff concentrations of TBEE in static water bodies will be in the sub-parts per million range. At maximum application rates (2 pts/acre), these concentrations would range from about 0.1 to 5.2 mg/L. The concentrations for the worst exposure scenario (#1) are greater than (7x) the 96 hour LC5O concentrations for freshwater fish; those for the other scenario are almost an order of magnitude less. The no effect level for TBEE with juvenile Coho salmon is <u>≤</u>1.0 mg/L (122). Therefore, under the worst exposure scenario with the maximum application rate of herbicide, the 96 hour LC5O could be exceeded. Under other, less extreme conditions at average application rates, predicted concentrations of the active ingredient would be substantially less than the reported no effect level in Coho salmon. The persistence characteristics of TBEE are such that the ester form of Triclopyr would not likely persist in surface waters for longer than a couple of days, except in those waters in Massachusetts which are acidic where the ester may persist for up to several months. It is also very unlikely that rainbow trout would be impacted at application rates of 0.5 pts/acre based on the reasonable scenario (#2) which predicts water concentrations of Garlon 4 less than toxic

concentrations.

The following factors would also tend to reduce the exposure concentrations that fish would experience: flowing waters would provide greater dilution than assumed for static conditions; the Massachusetts Right-of-Way Management Act mandates an application setback of 10 feet from standing or flowing waters or from wetlands (33 CMR 1l.04:(l) and (4) (a)); and actual runoff of the applied herbicide would probably be less than used for these sample calculations. Scenario 1 represents an extremely unlikely event where 20% of all the herbicide applied to an acre runs off into a small water course. The conditions which would foster this type of runoff across setbacks (i.e. heavy rains) would tend to turn static stream systems into flowing water courses and hence increase dilution.

The application rate used in the previous non—target species assessment (June 23, 1990) was 0.5 pints per acre applied basally. The utilities involved in managing rights-of-way and the manufacturer of Garlon 4 have since indicated that the required application rate may range as high as 2-3 quarts of Garlon 4 per acre for effective control of vegetation. The following addition to the exposure assessment examines the resultant changes in the predicted exposure concentrations that might occur in freshwater fish habitats when Garlon 4 is applied at the 2-3 quarts /acre rate.

The change in the application rate will result in the following differences in predicted exposure concentrations from those originally predicted for 0.5 pts/acre:
2 at/acre x 2pt/ qt = x 8 0.5 pt/acre

3at/acre x 2pt/qt = x 12 0.5 pt/acre

Application rates will therefore be 8-12 times greater than for the 0.5 pts/acre case. The probable concentrations in water after runoff as previously predicted were 1.3 (Scenario 1) and 0.03 mg/L (Scenario 2) ing butoxyethyl ester of Triclopyr / L. These concentrations would therefore range from 0.24
— 15.6 ing/L for application rates between two and six quarts.

These predicted concentrations encompass and substantially exceed the reported LCSO concentrations for fish (in range of 0.7 - 1.3 mg/L and the NOEL of 1 mg/L for juvenile Coho salmon. The more realistic exposure scenario (#2) predicts exposure concentrations of the same order of magnitude as the LC5O values.

Given that the higher application rates required for vegetation control in some areas have the potential to produce potentially lethal concentrations of the butoxyethyl ester of Triclopyr to fish in water as a result of runoff, a setback greater than the mandated 10 feet from standing or flowing waters (333 CMR 11.04: (1) and (4) (a)) will provide an additional level of protection when application rates exceed 0.5 pts/acre.

SUMMARY

Triclopyr exhibits moderate mobility in most of the soils tested. Soils with higher organic carbon content would be expected to retard the mobility of Triclopyr. Trichloropyridinol, the major breakdown product, is less mobile than Triclopyr.

Microbial degradation is the primary mechanism by which Triclopyr is degraded in soils. Degradation rates are variable and appear to be dependent on the soil and climatic conditions. In Massachusetts conditions, Triclopyr can be expected to have moderate persistence when applied in warm weather (late spring — early fall), and slightly longer persistence in colder weather.713 mg/kg. Rabbits and guinea pigs have oral LDSOs of 550 and 310 mg/kg respectively. The target organ for Triclopyr is in the liver. The only positive result in the oncogenicity studies was an increase in the combined incidence of mammary adenomas and adenocarcinoinas in the female rats at the high dose. Mutagenicity tests were negative. The developmental NOEL was reported as 75 mg/kg/d with a slight increase in maternal mortality. Using EPA's carcinogen classification scheme, Triclopyr may be considered a group C carcinogen (possible human carcinogen: limited animal evidence).

RECOMMENDATION

The herbicide Garlon 4, containing the butoxyethyl ester of Triclopyr (EPA Reg. No. 464-554), is recommended for use in sensitive areas only at application rates of 0.5 pt/acre pursuant to 333 CMR 11.00. Applications at rates up to three quarts per acre are permitted with a setback of 50 feet from standing or flowing waters suitable for fish habitat. The set back restriction may be waived upon demonstration to both the Departments of Food and Agriculture and Environmental Protection that runoff concentrations from applications of Garlon 4 with setbacks less than 50 feet do not pose a threat to fish.

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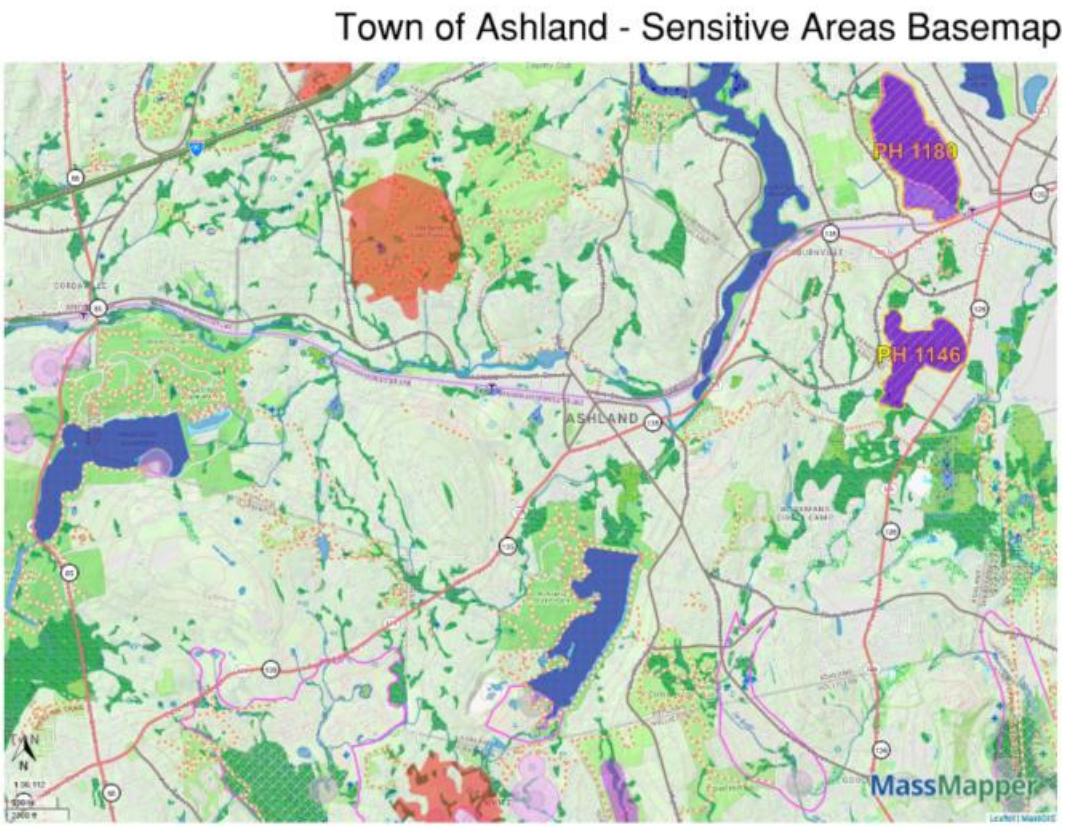
Draft Sensitive Areas Basemap:



BioMap Core Habitat Components: Rare Species Core

BioMap Core Habitat Components: Vernal Pool Core

NHESP Priority Habitats of Rare Species

Major MassGIS-MassDOT Roads by Road
- Interstate Shields
- Limited Access Highway
- U.S. Route Shields
- Multi-lane Hwy, Not Limited Access
- State Route Shields
- Other Numbered Highway
- Major Road, Collector
- Ramp
- Tunnel
- Tunnel (Limited Access Hwy)
- Tunnel (Multi-lane Hwy)
- Tunnel (Other Numbered Hwy)

Hiking and Wilderness Trails
- Hiking and Wilderness Trails

Long Distance Trails
- Appalachian Trail
- Bay Circuit Trail
- Mohican Mohawk Trail
- Midstate Trail
- New England Trail
- Taconic Crest Trail
- Warner Trail

Tracks and Trails MHD
- Track
- Trail

Zone Is Dissolved

Zone IIs Dissolved

IWPAs Dissolved

USGS Rivers and Streams 25k
- Stream
- INTERMITTENT STREAM
- SHORELINE
- INTERMITTENT SHORELINE
- MANMADE SHORELINE
- DITCH/CANAL
- AQUEDUCT
- DAM
- CHANNEL IN WATER

DEP Wetlands Hydrologic Connections

NWI Wetlands
- Estuarine and Marine Deepwater
- Estuarine and Marine Wetland
- Freshwater Emergent Wetland
- Freshwater Forested/Shrub Wetland
- Freshwater Pond
- Lake
- Riverine
- Other

DEP Wetlands Detailed With Outlines
- Barrier Beach System
- Barrier Beach-Deep Marsh
- Barrier Beach-Wooded Swamp Mixed Trees
- Barrier Beach-Coastal Beach
- Barrier Beach-Coastal Dune
- Barrier Beach-Marsh
- Barrier Beach-Salt Marsh
- Barrier Beach-Shrub Swamp
- Barrier Beach-Wooded Swamp Coniferous
- Barrier Beach-Wooded Swamp Deciduous
- Bog
- Coastal Bank Bluff or Sea Cliff
- Coastal Beach
- Coastal Dune
- Cranberry Bog
- Deep Marsh
- Barrier Beach-Open Water
- Open Water
- Rocky Intertidal Shore
- Salt Marsh
- Shallow Marsh Meadow or Fen
- Shrub Swamp
- Tidal Flat
- Wooded Swamp Coniferous
- Wooded Swamp Deciduous
- Wooded Swamp Mixed Trees

Potential Vernal Pools

NHESP Certified Vernal Pools

Property Tax Parcels