Imazamox

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Massachusetts Department of Agriculture
Division of Crop and Pest Services

and

Massachusetts Department of Environmental Protection
Office of Research and Standards

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1. Chemical Overview

1.1 Chemical Identity

Imazamox is part of the imidazolinone chemical class. The chemical structure is shown in Figure 1.1:

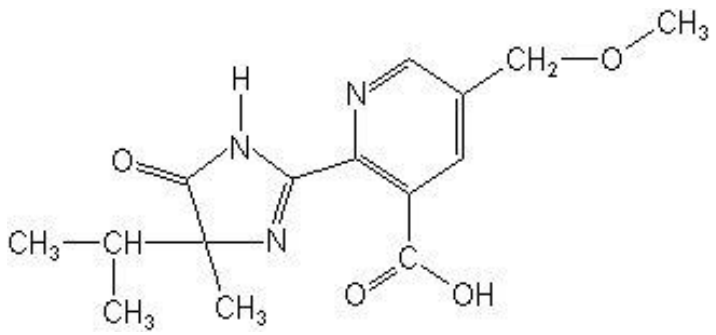

Figure 1.1 Imazamox Chemical Structure

1.2 Registration History

Imazamox was first registered by USEPA in 1997 for the use on soy beans (USEPA, 1997A). It received the designation of reduced risk pesticide (USEPA, 1997B), which allowed an expedited review and approval process. Such a designation is based on having advantages over existing products in terms of low human health impacts, low toxicity to non-target organisms, favorable environmental fate profile, and compatibility with Integrated Pest Management (USEPA, 1998) techniques. Imazamox is exempted from tolerance requirements in all food and feed uses (USEPA, 2003).

1.3. Aquatic Use

In 2008, USEPA approved the use of imazamox for the control of vegetation in and around aquatic sites and terrestrial non-crop sites. Imazamox is herbicidally active on many submerged, emergent, and floating broadleaf and monocot aquatic plants in and around standing and slow-moving water bodies.

1.4 Pesticide Type, Class, and Mode of Action

Imazamox is the common name for (±)-2-[4,5-dihydro-4-methyl-4-(1-methylethyl)-5-oxo-1H-imidazol-2-yl]-5-(methoxymethyl)-3-pyridinecarboxylic acid. Imazamox is a member of the imidazolinone class of herbicides that also includes imazapic, imazapyr, imazethapyr, imazamethabenz, and imazaquin (SERA, 2010). It is used for control of most annual and perennial broadleaf weeds and grasses, woody species, and riparian and emergent aquatic weed species.

Imazamox is formulated both as an acid and as an isopropylamine salt. Uptake of imidazolinone herbicides is primarily through the foliage and roots. The herbicide is then translocated to meristematic tissue (buds or areas of growth) by the xylem and phloem where it inhibits acetohydroxyacid synthase [AHAS; also known as acetolactate synthase (ALS)], an enzyme involved in the synthesis of three essential amino acids (valine, leucine, isoleucine). These amino acids are required for protein synthesis and cell growth. Imazamox thus disrupts protein synthesis and interferes with cell growth and DNA synthesis, causing the plant to slowly die. AHAS is not present in mammals, birds, fish, or invertebrates making it specifically toxic to plants (USEPA, 2008).

Another imidazolinone herbicide, imazapyr, is approved for use in MA lakes and ponds (MDAR, 2012). Unlike imazamox, imazapyr does not demonstrate any in-water herbicidal activity (Hamel, 2012), but its aquatic use is for treatment of floating and emergent vegetation.

1.5 Physical and Chemical and Environmental Fate Properties

Several important physical, chemical, and fate and transport property values for imazamox are listed in Table 1.2 (USEPA, 2008). EPA classified imazamox as moderately persistent. In an aquatic photolysis study, six major degradates of imazamox have been identified: 2,3,5-pyridine tricarboxylic acid (CL-351,543); 2-carbamoyl-5-(ethoxy-methyl) nicotinic acid (CL-359-770); 2-[1-carbamoyl-1,2-dimethylpropyl)carbamoyl]-5-(methosyxmethyl)nicotinic acid (CL-336-554); 2-carbamoyl-3,5-pyradine dicaroxylic acid (CL-359-712); 5-methoxymethy-2-3-pyridine dicoarxylic acid (CL-334-151); and 2-formyl-5-(methoxymethyl) nicotinic acid (AC9960-139A) (NY State, 2009; USEPA, 2008; IUPAC FOOTPRINT Pesticides Properties Database).

Imazamox degrades aerobically in the soil ($t_{½}$ of 27 d) to primarily a non-herbicidal metabolite (CL-354,825 but no chemical name or description available) which is immobile to moderately mobile. Imazamox degraded much more slowly in a soil photodegradation study with a half-life of 65 days. However, degradation in the dark controls confirms the results from the aerobic soil metabolism study. The aerobic soil metabolism half-life was 27 days in sandy loam soil, where the oxidative metabolite AC 312,622 (described as demethylated parent with intact ring structures and two carboxylic acid groups), increased to 40.8-43.6 % of the applied amount by 14-42 days, and then decreased to 2.9 % of the applied amount by 365 days. The other oxidative metabolite AC 354,825 (described as demethylated, decarboxylated parent with intact rings, one carboxylic acid group) increased to 54 % of the applied amount by 365 days. The anaerobic soil metabolism and the anaerobic aquatic metabolism studies both indicate that imazamox does not degrade under anaerobic conditions.

The mobility studies indicate that imazamox is very mobile to mobile with a K_d range of 0.05-2.7 and a K_{oc} range of 5-143 in soils with 0.29-2.59% organic carbon. The metabolite CL-312,622 was very mobile to mobile also with a K_d range of 0.71-2.19. The metabolite CL-354,825 was less mobile with its K_d range of 3.8-26.6. Terrestrial field dissipation studies were conducted at five sites: North Dakota, Georgia, Arkansas, Iowa and California. The respective half-lives were calculated to be 130, 50, 35, 15 and 50 days.

Laboratory studies indicate that imazamox is stable to hydrolysis at pH 5, 7, and 9. Photolysis is the most important route of degradation in water. The laboratory-determined half-life is 0.3 days. Photolysis by UV radiation is faster with a half-life of 0.054 d (Quivet et al., 2006). It degrades rapidly by aqueous photolysis with an average half-life of 6.8 hours at pH 5, 7, and 9. Monitoring data from eleven water bodies that were treated up to three times with imazamox at target application rates of 12 to 200 μg/L indicated a half-life of 20.3 days (upper 90th percentile).

Imazamox will be mobile in many soils, which coupled with its moderate persistence could facilitate its reaching ground water. Information from environmental fate studies indicates that imazamox should not persist in shallow surface waters. However, it should persist in water at greater depths when an anaerobic environment exists and where photolytic degradation is not a factor.

Table 1.2. Environmental fate properties of imazamox determined in standard laboratory tests

Imazamox		
Property	**Value**	**Source**
CAS number	114311-32-9	MRID# 47111801
Molecular weight	305.33	FOOTPRINT*
Molecular formula	$C_{15}H_{19}N_3O_4$	FOOTPRINT
Water solubility (mg/L)	4413	EPA Fact Sheet (1997)
log K_{ow}	0.73	EPA Fact Sheet (1997)
Vapor pressure at 25^{o}C torr	$9.97X\ 10^{-08}$	FOOTPRINT
Henry's Law constant at 25^{o}C (Pa m^3 mol^{-1})	$9.76\ X\ 10^{-07}$	FOOTPRINT
Soil adsorption coefficient K_{OC} (L/kg)	159 (avg)	MRID# 43193242
Hydrolysis half-Life pH = 5 pH = 7 pH = 9	Stable	MRID# 43193240
Photolysis half-life in water (day)	0.3	MRID#43876222
Photolysis half-life in soil (day)	65	MRID 43876223
Aerobic metabolism half-life in soil (day)	27	MRID 43876224
Fish bioconcentration factors	The bioconcentration factor for the inedible tissue was <1X.	MRID 43876231

***: FOOTPRINT* Pesticide Properties Database: http://sitem.herts.ac.uk/aeru/iupac/index.htm**

2.0 Human Health Risk Assessment

2.1 Hazard Characterization

US EPA's (2001) hazard identification assessment report provides a review of the toxicological data for imazamox relative to its use in occupational and residential risk assessments. The herbicidal mode of action of imazamox, (i.e., through inhibition of a biosynthetic pathway not present in animals) is one of the factors contributing to the low toxicity of imazamox to animals. US EPA considers the toxicological database on imazamox to be essentially complete. Imazamox has a low acute toxicity via the oral, dermal and inhalation routes of exposure, and generally falls into toxicity categories III and IV (USEPA uses a ranking system for response ranging from Category I (most severe response) to Category IV (least severe response). It is not a skin irritant or a sensitizer. It is moderately irritating to the eyes of rabbits. No toxicity was seen at doses exceeding the limit-dose in long-term studies in mice (no-observable-adverse-effect-level (NOAEL) = 1053 mg/kg/day), rats (NOAEL= 1068 mg/kg/day) and dogs (NOAEL= 1156 mg/kg/day). No information was provided on the types of toxicity or health endpoints that were evaluated in these studies. No developmental or maternal toxicity was seen at 1000 mg/kg/day in rats and 900 mg/kg/day in rabbits. It is non-mutagenic in various *in vivo* and *in vitro* mutagenic assays. It is not carcinogenic to mice and rats when administered in the diet at limit dose. It has been classified as "not likely to be carcinogenic to humans". It was readily absorbed by male and female rats following intravenous or oral dosing. More than 73% of the administered dose was excreted in the urine within 24 hours of dosing.

An acute reference dose (aRfD) was not established because an appropriate health endpoint attributable to a single exposure (dose) was not available. In 1997, EPA established a chronic oral reference dose (cRfD) of 3.0 mg/kg/day for imazamox based on a NOEL of 300 mg/kg/day from the developmental toxicity study in rabbits and an uncertainty factor of 100. In 2001, the EPA concluded that the use of 3.0 mg/kg/day was inappropriate because the endpoint of "decreased weight gain" was not biologically significant. EPA suggested that the highest dose tested for these studies should be used as the actual NOAEL (rat = 1,068 mg/kg/day and rabbit = 900 mg/kg/day). No suitable adverse health end point was observed in any of the available oral studies. It was concluded that quantification of risk is not required since no hazard was identified. EPA essentially rescinded the earlier established cRfD.

In a comprehensive human health risk assessment for the use of imazamox in vegetation management by the USDA Forest Service , the chronic RfD of 3.0 mg/kg bw/day originally proposed by USEPA (1997) was maintained as the basis for quantitative risk characterizations—i.e., hazard quotients (HQs) (SERA, 2010). It was noted that a higher RfD of up to about 10 mg/kg bw/day could be justified based on the NOAELs summarized above by USEPA (2001). This argument was not given further consideration because the RfD of 3.0 mg/kg bw/day did not lead to any HQs that exceed the level of concern (HQ=1) (see also the risk characterization below in section 4.3).

2.2 Exposure Assessment

The SERA (2010) review includes a comprehensive exposure and risk characterization for or associated with terrestrial and aquatic applications for workers and the general public. In the

exposure assessment for the general public, the highest non-accidental exposure scenario is associated with the the consumption of contaminated water by a small child, based on expected peak imazamox concentrations in water, resulting in an upper-bound exposure estimate of 0.06 mg/kg bw/day. The dose associated with dermal exposure from swimming in contaminated water is much lower than the oral exposure dose above. The chronic and longer-term exposure levels were much lower than acute exposures. The highest longer-term exposure was about 0.01 mg/kg bw/day associated with consumption of contaminated water and expected longer-term water concentrations of imazamox . The highest accidental exposure dose was for the consumption of contaminated water by a small child, for which the upper-bound dose estimate was 1 mg/kg bw/day.

2.3 Risk Characterization

The SERA (2010) review concluded that imazamox applications will not pose any substantial risks to humans or other species. For humans and mammalian wildlife, confidence in the risk characterization is high. Imazamox has been subject to a standard and relatively extensive series of acute, subacute, and chronic studies in mammals.

The risk was characterized based on the hazard quotient (HQ), which is defined as the exposure divided by the toxicity value. The chronic RfD of 3 mg a.e./kg bw/day was used to characterize both acute and chronic risk. An HQ of 1 was defined as the level of concern. For the general public, the highest risk was associated with the consumption of contaminated water by a child; upper-bound HQ values were 0.02 and 0.8, respectively, falling below the level of concern. The exposure scenarios evaluated included dermal exposure from contaminated vegetation, dermal exposure from swimming in contaminated water, and oral exposure from ingesting contaminated water, vegetation and fish.

2.4 Drinking Water Assessment

Exposure to imazamox from drinking water was considered in the aggregate exposure and risk assessment conducted by US EPA in support of the exemption from requirement of a tolerance in food and feed (USEPA, 2003). Drinking water concentrations were estimated based on the modeling of imazamox concentrations in surface and groundwater from exposure from terrestrial applications of imazamox at maximum label rates. The acute exposures were estimated to be 5.7 μg/L for surface water and 1.0 μg/L for groundwater; chronic exposure estimates were 0.61 μg/L and 1.0 μg/L, respectively. These concentrations were used in the drinking water assessment. There is no information that indicates that EPA conducted a drinking water assessment specifically for exposure associated with aquatic applications of imazamox.

SERA (2010) estimated a chronic dose of 0.01 mg/kg for an adult drinking water from an imazamox- treated water body. This dose was based on an exposure to a concentration of 360 μg/L in the treated water, which in turn was based on an initial peak level of 500 μg/L and degradation with a half-life of 90 days. Estimated concentrations in surface water from exposures associated with terrestrial applications were much lower (5 μg/L; 2-10.4 μg/L). The risk characterization indicated that these concentrations were far below the level of concern.

For the review presented here, an additional drinking water risk assessment was done by considering the health-based screening level for imazamox. Health-Based Screening Levels (HBSLs)[1] are benchmark concentrations of contaminants in water that may be of potential concern for human health, if exceeded. HBSLs are non-enforceable benchmarks that were developed by the USGS in collaboration with USEPA and others using USEPA methodologies for establishing drinking-water guidelines and the most current, USEPA peer-reviewed, publicly available human-health toxicity information (Toccalino et al., 2008).

For noncarcinogens, the HBSL represents the contaminant concentration in drinking water that is not expected to cause any adverse effects over a lifetime of exposure. HBSL calculations adopt USEPA assumptions for establishing drinking-water guidelines, namely, lifetime ingestion of 2 liters of water per day by a 70-kilogram adult. It also is assumed that 20 percent of the total contaminant exposure comes from drinking water sources and that 80 percent comes from other sources (for example, food and air). If data are available to quantify the percentage of contaminant exposure that comes from water, then a data-derived percentage is used instead of the default of 20 percent.

An HBSL has not been established for imazamox. For the purpose of the review presented here, a chronic drinking water guideline value of 21,000 µg/L was calculated following the USGS methodology for deriving an HBSL (USGS, n.d.) using the cRfD of 3 mg/kg/day (USEPA, 1997).

Even the most conservative screening-level scenarios with the maximum concentration allowed in treated surface water of 500 µg/L with no attenuation, or the recharge of groundwater with treated surface water with no attenuation, resulting in a groundwater concentration of 500 µg/L, are well below the guideline value. This comparison of expected levels in water bodies to the guideline indicates that there is no concern for effects on human health from drinking water containing residues of imazamox from aquatic applications or terrestrial applications.

2.5 Deficiencies and Data Gaps

The hazard identification assessment report (USEPA, 2001) states that there were no data gaps identified with the review of the mammalian toxicological data for imazamox.

[1] For more information on HBSLs see: USGS Health-Based Screening Levels

3 Ecological Risk Assessment

USEPA conducted an ecological risk assessment as part of the evaluation associated with the registration for the use of imazamox to control vegetation in aquatic and non-crop sites (USEPA, 2008). The SERA review for USDA Forest Service (SERA, 2010) also provides a comprehensive ecological risk assessment that was consulted for this special review presented here.

3.1 Ecological Hazard Characterization

The risk assessment document by USEPA (2008) describes the effects characterization and is summarized below.

Fish: Available acute toxicity data for aquatic species indicate that imazamox acid is practically non-toxic to fish, both freshwater and estuarine. LC_{50} and EC_{50} values for fish were not determined from aquatic toxicity tests because there were no adverse effects observed at the highest concentrations tested. The acute No Observed Adverse Effect Concentrations (NOAEC) ranged from 94.2 mg/L for sheepshead minnow to 122 mg/L for rainbow trout. Chronic toxicity studies were not submitted to USEPA. As noted in the SERA review, it is not clear why these studies were not submitted to EPA since the studies were submitted to the European Commission (2002). The review by the European Commission provides two longer-term studies in rainbow trout showing a 28-d NOEC of 122 mg/L and a 96-day NOEC of 11.8 mg/L.

Aquatic Invertebrates: Similar to the data for fish, imazamox is practically non-toxic to aquatic invertebrates, both fresh water and estuarine. Lorenzo et al., (2013) determined LC_{50} values for hypogean (i.e., in groundwater) and epigean (i.e., above the ground surface) copepods of 199.23 mg/L and 232.44 mg/L respectively. LC_{50} and EC_{50} values for other invertebrates were not determined in acute toxicity tests. The NOEC was 115 mg/L for *Daphnia magna* and 89.3 mg/L for mysid shrimp. Chronic toxicity studies were not submitted to the USEPA, but the review by the European Commission provides a 21-d NOEC of 137 mg/L for *Daphnia magna*. In addition, chronic exposure levels for hypogean and epigean copepods of 29.52 mg/L and 67.67 mg/L respectively were modeled by Lorenzo et al. (2014) using the Acute/Chronic Estimation Method (ACE V3.0) (Mayer et al.,1999 as cited in Lorenzo et al., 2014).

Amphibians: For ecological risk assessment of amphibians, USEPA generally uses fish toxicity data as a surrogate for aquatic-phase amphibians (USEPA, 2013). While EPA does not make reference to specific data in support of this practice, several reviews of toxicity data in the literature indicate that this approach is justified in most cases.

Mayer and Ellersieck (1986) developed a database with 410 chemicals (mainly pesticides) and 66 species of aquatic animals. Their analysis of acute toxicity data indicated the following order of toxicity: insects and crustaceans>fish> amphibians .

Kerby et al. (2002) analyzed the acute toxicity data (LC_{50}) from almost 24,000 studies retrieved from US EPA's ECOTOX database and concluded that amphibians are of low to moderate

sensitivity to metals, inorganic chemicals and pesticides when compared with 13 other classes of organisms, including fishes. The exceptions were three phenolic compounds that are highly toxic to amphibians.

Weltje et al. (2013) analyzed acute and chronic data obtained from the ECOTOX database and data from the scientific and regulatory literature. Acute toxicity comparisons of amphibian and fish sensitivity were made for 55 chemicals, including 32 pesticides. Chronic toxicity comparisons were made for 52 chemicals, including 20 pesticides. The overall outcome was that fish and amphibian toxicity data are highly correlated and that fish are generally more sensitive (both acute and chronic) than amphibians. Four of 55 chemicals were 10-and 100-fold more acutely toxic to amphibians than fish: (aluminum chloride [25-fold], 2,4-D herbicide [12-fold], malathion (insecticide) [34 fold] and pentachlorophenol-sodium salt [12-fold]. Two were more than 100-fold more acutely toxic to amphibians than fish: (p-nonylphenol (2,111-fold) and dimethoate (7,300-fold). More detailed examination of the few cases showing higher sensitivity for amphibians indicated similar acute sensitivity of amphibians and fish.

For chronic toxicity, Weltje et al. (2013) also identified dexamethasone (an anti-inflammatory and immunosuppressant) which was 11-fold more toxic to amphibians than fish. This compound interferes with amphibian metamorphosis. If fish data were used as a surrogate for amphibians to screen for potential toxicity, this compound might not be identified. However, several other compounds known to influence amphibian metamorphosis were included in the analysis, and these did not affect amphibians disproportionately.

Bioconcentration of Aquatic Organisms
Laboratory bioconcentration studies with bluegill sunfish, eastern oyster, and grass shrimp indicate that parent imazamox, even though long-lived in the [terrestrial] environment, is not subject to bioconcentration. Imazamox did not significantly accumulate in bluegill sunfish. Concentrations in whole fish and edible tissue were less than the minimum quantifiable limit while the bioconcentration factor for inedible tissue was <1X.
Therefore, food-chain exposures are not expected to be significant in aquatic systems.

<u>*Aquatic Plants*</u>: A screening-level study with unicellular plant species (diatom and algal species) showed no adverse effects of imazamox at concentrations up to 0.040 mg/L.

A 14-day laboratory study by Netherland et al. (2009) with three planktonic blue-green algae and three beneficial planktonic green algae species exposed to imazamox and two other ALS inhibiting herbicides indicated that it does not have significant activity at levels up to 0.500 mg/L. This was measured by chlorophyll-*a* content at concentrations of 0.100, 0.200 and 0.500 mg a.i. /L in a 14-day study. The results showed that imazamox does not have significant activity against these unicellular plant species. It was noted that further testing with different target and beneficial algal species is needed to determine direct impacts of imazamox and the herbicides on different algal classes (e.g., diatoms and filamentous algae).

It is also useful to consider information from toxicity prediction models. The Ecological Structure Activity Relationships (ECOSAR) Class Program is a computerized predictive system that

estimates aquatic toxicity (USEPA, 2013B). For imazamox, ECOSAR predicts a chronic toxicity value (geometric mean of NOEC and LOEC) of 5.1 mg/L for green algae.

In contrast to microscopic algae, imazamox appears to be toxic to aquatic macrophytes, albeit this is based on limited information for duckweed (*Lemna gibba*). The 14-day EC_{50} is 0.011 mg a.i. /L with an NOEC of 0.0045 mg a.i./L. The SERA review notes data for another duckweed species (*Lemna minor*). Those 4- and 7-day exposure studies showed EC_{50} values of 0.055 and 0.029 mg/L, respectively.

Hamel (2012) reviewed a study by Nissen, et al. (2007) who found that in small tank studies, Eurasian water milfoil (*Myriophyllum spicatum*) was sensitive to 0.200 mg/L imazamox, although sago pondweed (*Stuckenia pectinatus*) was not susceptible to even up to 0.800 mg/L imazamox.

Avian Toxicity: Available acute and chronic toxicity data indicate that imazamox acid is slightly to practically non-toxic to upland game birds and waterfowl (USEPA, 2008; SERA, 2010). For instance, a gavage dose of 1846 mg/kg (the highest dose tested) was not associated with mortality or signs of toxicity in quail. Acute oral toxicity to mallard was >1950 mg/kg bw;
An acute 5-day dietary study showed no mortality or signs of toxicity at mean measured dietary concentrations of 5572 mg/kg (LC_{50} >5000 mg a.i./kg diet) for both bobwhite quail and mallard ducks). Chronic NOAECs/LOAECs for bobwhite quail and mallard duck were >2000 mg a.i./kg diet determined in reproduction studies.

Mammalian Toxicity. Acute and chronic toxicity data also indicate that imazamox is practically non-toxic to mammals. The acute LD_{50} value for rats was 2313 mg a.i./kg bw for male and 2121 mg a.i./kg for female. The rat reproduction study indicated a NOAEL of >20,000 ppm equivalent to 1,284 mg/kg/day (no effects observed at the highest dose).

Terrestrial Reptiles. For terrestrial reptiles, EPA uses avian toxicity data as a surrogate (USEPA, 2013). The Agency has models available that are specifically designed for the estimation of exposure to terrestrial amphibians and reptiles. EPA did not conduct a risk assessment for terrestrial reptiles as part of the assessment of imazamox for aquatic use.

Terrestrial Invertebrates. Acute contact studies indicate that imazamox acid is practically non-toxic to honey bees (LD_{50} >0.025 mg a.i./bee).

Terrestrial Plants. Terrestrial plant toxicity studies with monocots and dicots indicate that seedling emergence and vegetative vigor are severely impacted by exposure to imazamox. Most sensitive in seedling emergence in monocots was oat with an EC_{25} of 0.0018 lb a.i./acre (0.224 mg a.i./m^2) and in dicots was cabbage with an EC_{25} of 0.0018 lb ae/acre (0.202 mg a.i./m^2). Most sensitive in vegetative vigor in monocots was oat an EC_{25} of 0.0016 lb a.i./acre (0.180 mg a.i./m^2) and in dicots it was tomato with an EC_{25} of 0.0010 lbs a.i./acre (0.1120 mg a.i./m^2).

3.2 Aquatic Exposure Assessment for Direct Applications to Water

USEPA (2008) derived exposure concentrations resulting from *direct application to water bodies* from monitoring data. Eleven water bodies were treated up to three times with imazamox to achieve target concentrations of 0.012 to 0.200 mg/L The results from this monitoring study indicated a half-life of 20.3 days (upper 90th percentile). Using this half-life value, the concentrations following at various times after an application at the maximum rate of 0.500 mg/L were calculated. These values are shown in Table 3.1.

Table 3.1. Estimated Environmental Concentrations (EEC) (μg/L) of imazamox in surface water based on monitoring data (USEPA, 2008)

Peak	4-day	21-day	60-day	90-day
500	436	244	60.2	24

The exposure assessment described in the SERA review (2010) used the maximum application concentration of 500 μg/L for acute exposure and a 90-day time-averaged concentration of 361 μg/L for chronic exposure (based on half-life of 90 days). This dissipation half-life of imazamox was used for both human health risk assessment and aquatic life risk assessment. The use of the 90-day half-life value was admitted to be arbitrary and can be regarded as a conservative assumption compared to the monitoring study-derived value used by EPA However, it was pointed out that the assumptions concerning the dissipation half-life did not impact the final risk assessment conclusions.

Modeling with the AQUATOX model

An additional simulated exposure was conducted for this review using the AQUATOX model (Appendix 1). AQUATOX is an ecosystem simulation model that predicts the fate of various pollutants, such as excess nutrients and organic chemicals, and their effects on aquatic ecosystems. It has the capability to estimate the concentration of an applied herbicide in a water body after *direct application*. The fate portion of the model was used here to characterize the dissipation of imazamox following an application to a standard pond. EECs[2] for aquatic uses were calculated for cases of direct application to the surface of standing water bodies of 1.0 ft (0.30 m), 3.0 ft (0.91 m), 3.94 ft (1.2 m) and 6.6 ft (2.0 m) depths (Appendix 1). The 6.6 ft (2.0 m) depth is representative of the water depth in the standard pond scenario used by the Environmental Fate and Effects Division (EFED) of EPA for most ecological effects assessments. The 1 and 3 foot water depths are typical of use conditions in irrigation and drainage ditches, and for edge of pond depths, where problematic aquatic weeds are typically found. The 3.94 ft depth was included since it is the depth in the model scenario used in AQUATOX (a Missouri farm pond).

Detailed information on the model input and results can be found in Appendix 1. Figure 3.1 shows the modeled imazamox concentration and dissipation in a 3-ft deep pond. Similar trends in concentration and dissipation were found with the other pond depths (Appendix 1, Fig. A2-1 through Fig. A2-4). The model results indicate that dissolved imazamox dissipates in large part

[2] EEC – estimated environmental concentration

within two months following the application and that dissipation of imazamox is primarily the result of photolysis (Fig. 3.1). The modeling was done with a photolysis half-life of 10 d. This is a value that was selected based on the laboratory half-life value of 0.3 d and the half-life of 20.3 d used by EPA. Hamel (2012) states that typical field half-life values are expected to be in the 5 to 10 day range. The model simulation results are reflecting a situation of intermediate persistence of imazamox. The EPA exposure assessment is more conservative and reflects a situation with higher persistence of imazamox such as under lower light conditions.

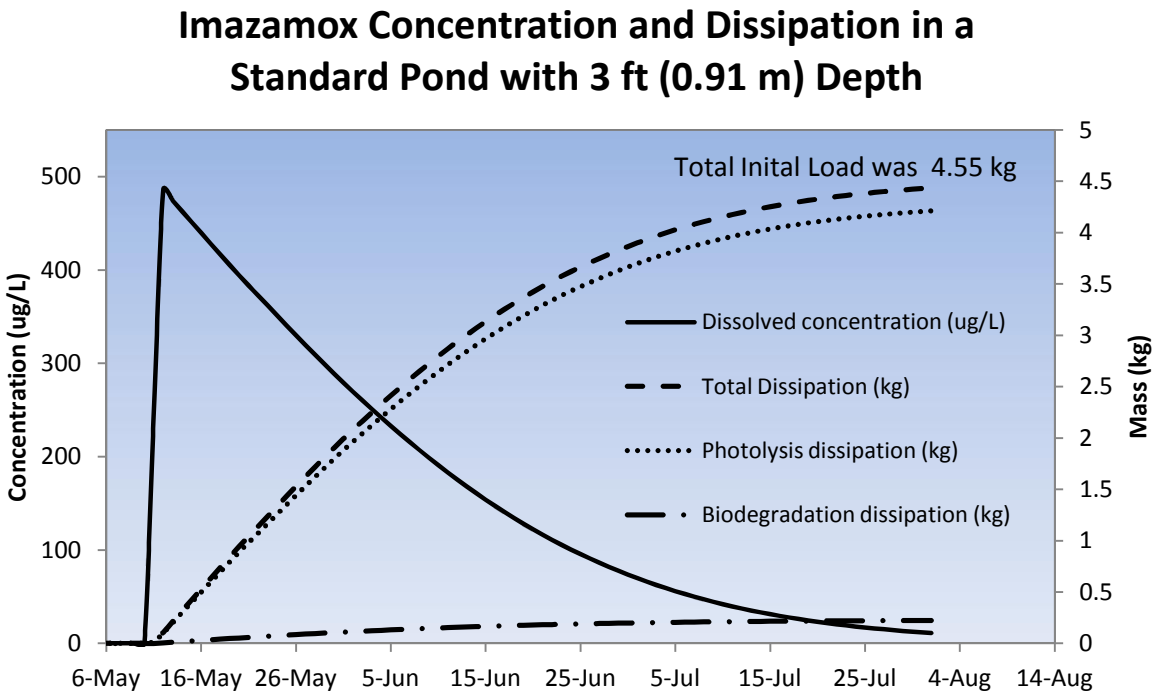

Figure 5.1. AQUATOX model-predicted imazamox concentration development and dissipation in standard pond with 3 ft depth. Application of imazamox to achieve an initial concentration of 500 µg/L was simulated to take place on May 10th. See Appendix 1 for modeling details and additional modeling results.

Table 3.3. Summary of the AQUATOX model estimated concentrations of imazamox in the standard pond with the depths as described above and in Appendix 1.

Depth		Concentration (µg/L)		
ft	m	Peak	1 Month	2 Months
6.6	2.0	490	250	84
3.9	1.2	489	213	54
3.0	0.91	485	190	42
1.0	0.30	482	124	16

The model results indicate that the pond depth affects the overall dissipation rate of imazamox in the pond water. Imazamox dissipates faster in a shallow pond. This may be attributed to a higher

photolysis rate which is associated with more intense light penetration in a shallow pond. This is also illustrated in Fig. A1-6 in Appendix 1. The concentrations after 1 and 2 months show the same trend with lower residue levels in shallower ponds.

It is also noted that the modeling scenario using the maximum application rate resulted in a concentration of 500 μg/L. Typical applications use lower rates as specified on the label that result in concentrations of 50 to 200 μg/L. The photolysis rate based on a half-life of 10 days is also reasonable , actual field dissipation may be faster based on or given that the laboratory photolysis half-life is 0.5 day.

3.3 Risk Characterization

Ecological risk characterization integrates the exposure and ecotoxicity data to evaluate the likelihood of adverse ecological effects. USEPA typically uses a deterministic approach to evaluate the likelihood of adverse ecological effects to non-target species. In this approach, risk quotients (RQs) are calculated by dividing EECs by ecotoxicity values for non-target species, both acute and chronic. RQs are then compared to established levels of concern (LOCs). These LOCs are criteria used by US EPA to indicate potential risk to non-target organisms and the need to consider regulatory action. The RQs were calculated based on the most conservative exposure assessment. In the HERA risk assessment, hazard quotients (HQ) are used as risk indices. HQ values are calculated in the same way as RQ values.

Aquatic Animals

As discussed in Section 5.1, imazamox is practically non-toxic to freshwater and estuarine fish and invertebrates on an acute exposure basis. No mortality was observed at the highest concentration tested and LC_{50} values could not be determined. Therefore, no RQ calculation was done. The NOECs are in the range of 94.2 to 122 mg/L for fish and 89.3 to 115 mg/L for invertebrates. The peak EEC estimation is 0.5 mg/L. A comparison of the NOECs with the peak EEC in surface water indicates a 179 to 244-fold difference between the highest estimated EEC and the concentrations which produced no effects. Therefore, it is concluded that the acute risk to fish and aquatic invertebrates is expected to be very low.

USEPA did not estimate chronic risk. Chronic data for aquatic animals were not available to the agency, but were available to the European Commission. The lack of adequately documented chronic toxicity data precluded the development of chronic risk quotients. However, comparison of the high-end EECs of less than 0.3 mg/L (Section 3.2) with NOECs for fish (28-d NOEC of 122 mg/L and a 96-d NOEC of 11.8 mg/L and a 21-d NOEC of 137 mg/L for *Daphnia magna*) indicates low potential for chronic harm.

SERA (2010) concludes that based on the risk characterization for fish and other groups of aquatic and terrestrial animals for which there are data available, it is unlikely that exposures to imazamox will cause significant risk to aquatic-phase amphibians.

Aquatic Plants

Aquatic single–celled plants are not adversely affected by imazamox with the EC_{50} being greater than the highest concentration tested (40 μg/L) which was based on 0.048 lb ai/A applied to 6 inch water column. USEPA (2008) concluded that these aquatic plant species have not been adequately tested for phytotoxic effects. The maximum concentration of 40 μg/L that the unicellular species were exposed to is well short of the maximum EEC 500 μg/L that the label states for aquatic weed control. The study by Netherland et al. (2009) provides data on planktonic blue-green algae and planktonic green algae at concentrations up to 500 μg/L. These data indicate that it is unlikely that imazamox would cause wide spread detrimental effects to these unicellular species at concentrations up to 500 μg/L. However, the study also points out that further testing with different target and beneficial algal species is needed to determine direct impacts of imazamox and the herbicides on different algal classes (e.g., diatoms and filamentous algae).

Table 3.4 shows the risk quotients for non-target vascular aquatic plants from imazamox application for aquatic weed control (USEPA, 2008). Imazamox application for aquatic weed control would exceed the Agency's LOC for non-target aquatic vascular plants.

Table 3.4. Risk Quotient Values for Non-Target Aquatic Plants (USEPA, 2008)

Plant Type	Species	Toxicity value used	Listed species RQ	RQ
Aquatic Weed Control				
Vascular plant	Duckweed (*Lemna gibba*)	EC50 = 11 μg/L	---	45.5
Vascular plant	Duckweed (*Lemna gibba*)	NOEC = 4.5 μg/L	111	---

*If RQ > 1.0, the LOC is exceeded, resulting in potential for risk to that plant group.

USEPA assessment results predicted that aquatic weed control will adversely impact nearby aquatic vascular plants. The RQs for non-target aquatic vascular plants that may be near the sites of aquatic weed control are 45.5 and 111 for non-listed and listed species, respectively, well above the LOC of 1.0.

Terrestrial Organisms

The RQ values calculated by USEPA (2008) for terrestrial animal exposure are <0.1 for acute risk and <0.52 for chronic risk. No acute or chronic LOC is exceeded for birds or mammals.
In the HERA risk assessment, the highest hazard quotients (HQ) value was 0.01 for small mammals consuming contaminated water following an accidental spill. This HQ value is below the LOC (HQ = 0.5) (USEPA, 2012B)) by a factor 50. For birds, the highest HQ value of 0.003, which was associated with an accidental spill in a water body, was 167 times lower than the LOC.

Federally Threathened and Endangered Species

USEPA (2008) discussed the risk to federally threatened and endangered species. It was concluded that the use of imazamox on non-cropland and for aquatic weed control is not anticipated to exceed the Agency's acute LOC for avian, mammalian, fish or aquatic invertebrate listed species. As noted above, USEPA was not able to estimate the chronic risks to listed aquatic

animals with the exception of birds for which the chronic risk LOC is not exceeded. Use of Imazamox for aquatic weed control may likely impact listed aquatic vascular plants.

USEPA also discussed the implications of indirect effects. The exceedance of LOCs for vascular plants indicates that there is potential for adverse effect on wildlife through effects on food and habitat. Limited data indicate that imazamox has no significant toxicity to several planktonic green algal species though there is no toxicity information for other algal classes. In certain situations, there may be improvements in food and habitat as a result of removal of invasive aquatic plants (Hamel, 2012). Indirect effects may be beneficial to some species and adversely affect other species depending on specific situations.

3.4 Uncertainties, Assumptions, Limitations and Data Gaps

USEPA (2008) indicated that data were missing on the ecotoxicity of imazamox. As indicated in Section 3.1 EPA was unable to estimate the chronic risk to aquatic animals due to the lack of chronic data for fish and aquatic invertebrates. Although USEPA (2008) indicated that there is minimal acute risk to aquatic animals, USEPA states that this would not rule out the potential for chronic toxicity to aquatic animals. However, the chronic data that were submitted to the European Commission indicate low potential for chronic harm (see Section 3.1).

SERA (2010) concludes that the confidence in the risk characterization for humans and mammalian wildlife is high. Data on birds are less extensive than mammals, but available data do not identify any potential hazards to birds. Toxicity data for other groups of animals, including amphibians, terrestrial invertebrates, fish and aquatic invertebrates are more limited or nonexistent. While available data for these groups do not indicate any hazards, the confidence in the risk characterization is less. Adverse effects on vascular plants can be expected and applications of imazamox will likely affect the vegetation in the treatment area, which may result in secondary effects on wildlife due to effects on food and habitat. As discussed earlier, these secondary effects vary greatly depending on the species and site characteristics.

4.0 References

European Commission, 2002. Review report for the active substance imazamox. Final. Accessed at: http://ec.europa.eu/food/plant/protection/evaluation/newactive/imazamox.pdf

Hamel, K. 2012. Environmental Impact Statement for Penoxsulam, Imazamox, Bispyribac-sodium, Flumioxazin, & Carfentrazone-ethyl. Addendum to the Final Supplemental Environmental Impact Statement for Freshwater Aquatic Plant Management. Washington State Department of Ecology, Olympia, Washington. Accessed at: https://fortress.wa.gov/ecy/publications/summarypages/0010040Addendum1.html

IUPAC FOOTPRINT Pesticides Properties Database, n.d. Accessed at: http://sitem.herts.ac.uk/aeru/iupac/

Kerby JL, Richards-Hrdlicka KL, Storfer A, Skelly DK. 2009. An examination of amphibian sensitivity to environmental contaminants: Are amphibians poor canaries? Ecol. Lett.12:1–8.

Lorenzo, T., W. D. Marzio, et al. (2014). "Sensitivity of hypogean and epigean freshwater copepods to agricultural pollutants." Environmental Science and Pollution Research Int. Mar;21(6):4643-55

Mattson, M.D., P.J. Godfrey, R.A. Barletta and A. Aiello, 2004. Eutrophication and Aquatic Plant Management in Massachusetts. Final Generic Environmental Impact Report. Edited by Kenneth J. Wagner. Department of Environmental Protection and Department of Conservation and recreation, Executive Office of Environmental Affairs, Commonwealth of Massachusetts. Available at: http://www.mass.gov/dcr/watersupply/lakepond/techassist.htm

Mayer FL Jr. and Ellersieck MR, 1986. Manual of acute toxicity: interpretation and data base for 410 chemicals and 66 species of freshwater animals, Resource Publication No.160, U.S.Department of the Interior, Fish and Wildlife Service, Washington, DC, 505 p.

MDAR, 2012. Imazapyr: Review for Use in Lakes & Ponds in Massachusetts. Available at: http://www.mass.gov/agr/pesticides/aquatic/docs/imazapyr.pdf.

N.Y. State, 2009. Review of Clearcast herbicide in support of registration in N.Y. State. N.Y State Dept. of Environmental Conservation. Accessed at: http://pmep.cce.cornell.edu/profiles/herb-growthreg/fatty-alcohol-monuron/imazamox/clrcast_mcl_0209.pdf

Netherland, M.D., C.A. Lembi, and L.M. Gromski. 2009. Potential for Selective Activity of the ALS Inhibitors Penoxsulam, Bispyribac-sodium, and Imazamox on Algae Responsible for Harmful Blooms. J. Aquat. Plant Manage. 47: 147-150. Accessed at: http://www.apms.org/japm/vol47/v47p147_2009.pdf`

Nissen, S. J., J. D. Vassios, and G. Brunk. 2007. Eurasian Water milfoil and Sago Pondweed Response to Imazamox. Abstract from the 26th Annual Western Aquatic Plant Management Society Annual Conference. Coeur d'Alene, Idaho.

Quivet, E., R Faure , J. Georges, J. Olivier Païssé, B. Herbreteau, and P. Lantéri. 2006. Photochemical Degradation of Imazamox in Aqueous Solution: Influence of Metal Ions and Anionic Species on the Ultraviolet Photolysis. J. Agric. Food Chem. 54: 3641–3645.

SEPRO Corp., 2012. Clearcast Herbicide product label and MSDS. Accessed on May 14, 2012 at: http://www.sepro.com/canals/Labels-MSDS.aspx

SERA, 2010. Imazamox: Human Health and Ecological Risk Assessment, Final Report. Syracuse Environmental Research Associates, Inc. (SERA). Submitted by Patrick R. Durkin to USDA Forest Service. Accessed at: http://www.fs.fed.us/foresthealth/pesticide/pdfs/052-24-02a_Imazamox.pdf

Toccalino, P.L., Norman, J.E., Booth, N.L, and Zogorski, J.S., 2008, Health-based screening levels: A tool for evaluating what water-quality data may mean to human health: U.S. Geological Survey, National Water-Quality Assessment Program, accessed July 28, 2011, at http://water.usgs.gov/nawqa/HBSL/

U.S. Geological Survey (USGS). n.d. Health-Based Screening Levels: benchmarks for evaluating water-quality data. Accessed December 26, 2012. Web site: http://infotrek.er.usgs.gov/apex/f?p=169:1:0::NO:::

USEPA, 1997. Imazamox Pesticide Fact Sheet. Available at: http://www.epa.gov/opprd001/factsheets/imazamox.pdf.

USEPA, 1997B. Pesticide Registration (PR) Notice 97-3: Guidelines for Expedited Review of Conventional Pesticides under the Reduced-Risk Initiative and for Biological Pesticides. Accessed at: http://www.epa.gov/PR_Notices/pr97-3.html

USEPA, 1998. General Overview: Reduced-Risk Pesticide Program. Accessed on June 13, 2012 at: http://www.epa.gov/oppfead1/trac/safero.htm.

USEPA, 2001. Imazamox: Report of the Hazard Identification Assessment review committee. Memorandum by P.V. Shah from HED to William Donovan, HED. July 11, 2001.

USEPA, 2003. Imazamox; Exemption from the requirement for a tolerance. https://federalregister.gov/a/03-3699

USEPA, 2008. Ecological risk assessment evaluating imazamox for the proposed new use for the control of vegetation in and around aquatic and non-cropland sites. Memorandum by Ibrahim Abdel-Saheb and Michael Davy from EFED to James Tompkins, Herbicide Branch. Courtesy of EFED.

USEPA, 2012A. EPA Pesticide Chemical Search. Available at: http://iaspub.epa.gov/apex/pesticides/f?p=chemicalsearch:1

USEPA, 2012B. Ecological Risk Assessment: Technical Overview. Accessed on June 13, 2012 at: http://www.epa.gov/oppefed1/ecorisk_ders/index.htm

USEPA, 2013A. Technical Overview of Ecological Risk Assessment Analysis Phase: Ecological Effects Characterization. Accessed on February 13, 2013 at: http://www.epa.gov/oppefed1/ecorisk_ders/toera_analysis_eco.htm

USEPA, 2013B. Ecological Structure Activity Reselationships (ECOSAR). Accessed on January 15, 2013 at: http://www.epa.gov/oppt/newchems/tools/21ecosar.htm

VASSIOS, J.D, S. J. NISSEN, AND G. R. BRUNK. Imazamox absorption, desorption, and metabolism by Eurasian watermilfoil. J. Aquat. Plant Manage.49: 44-49

Weltje L, Simpson P, Gross M, Crane M, Wheeler JR., 2013. Comparative acute and chronic sensitivity of fish and amphibians: A critical review of data. Environ.Toxicol. Chem. Feb 4. (Accepted) doi: 10.1002/etc.2149.

APPENDIX 1

Estimated Environmental Concentrations (EECs) and Dissipation Behavior of Imazamox following Direct Application to Water using the AQUATOX model

Modeling of Concentration and Dissipation of Imazamox in Standard Pond

AQUATOX is a model that is available from USEPA and has the capability to estimate the concentration in a water body from *direct application* of pesticides to a water body. AQUATOX is an ecosystem simulation model that predicts the fate of various pollutants, such as excess nutrients and organic chemicals, and their effects on aquatic ecosystems. An overview of the model is given below. For the purpose of the aquatic exposure assessment for the review presented here, the AQUATOX model was used to estimate the concentration and dissipation characteristics of imazamox in a standard pond. The fate portion of the model was used to here to characterize the dissipation of imazamox following an application to a standard pond.

Brief overview of the AQUATOX Model

AQUATOX is an ecosystem simulation model that predicts the fate of various pollutants, such as excess nutrients and organic chemicals, and their effects on aquatic ecosystems, including fish, invertebrates, and aquatic plants. AQUATOX is a valuable tool for ecologists, biologists, water quality modelers, and anyone who performs ecological risk assessments for aquatic ecosystems.

AQUATOX simulates the transfer of biomass, energy and chemicals from one compartment of the ecosystem to another. It does this by simultaneously computing each of the most important chemical or biological processes for each day of the simulation period; therefore it is known as a process-based or mechanistic model. AQUATOX can predict not only the environmental fate of chemicals in aquatic ecosystems, but also their direct and indirect effects on the resident organisms. Therefore it has the potential to establish causal links between chemical water quality and biological response and aquatic life uses.

AQUATOX is the only general ecological risk model that represents the combined environmental fate and effects of conventional pollutants, such as nutrients and sediments, and toxic chemicals in aquatic ecosystems. It considers several trophic levels, including attached and planktonic algae and submerged aquatic vegetation, invertebrates, and forage, bottom-feeding, and game fish; it also represents associated organic toxicants. It has been implemented for streams, ponds, lakes, estuaries, reservoirs, and experimental enclosures.

The fate portion of the model, which is applicable especially to organic toxicants, includes: partitioning among organisms, suspended and sedimented detritus, suspended and sedimented inorganic sediments, and water; volatilization; hydrolysis; photolysis; ionization; and microbial degradation. The effects portion of the model includes: acute toxicity to the various organisms modeled; and indirect effects such as release of grazing and predation pressure, increase in detritus and recycling of nutrients from killed organisms, dissolved oxygen sag due to increased decomposition, and loss of food base for animals.

AQUATOX is the latest in a long series of models, starting with the aquatic ecosystem model CLEAN (Park et al., 1974) and subsequently improved in consultation with numerous researchers

at various European hydrobiological laboratories, resulting in the CLEANER series (Park et al., 1975, 1979, 1980; Park, 1978; Scavia and Park, 1976) and LAKETRACE (Collins and Park, 1989). The MACROPHYTE model, developed for the U.S. Army Corps of Engineers (Collins et al., 1985), provided additional capability for representing submersed aquatic vegetation. Another series started with the toxic fate model PEST, developed to complement CLEANER (Park et al., 1980, 1982), and continued with the TOXTRACE model (Park, 1984) and the spreadsheet equilibrium fugacity PART model. AQUATOX combined algorithms from these models with an ecotoxicological construct borrowed from the FGETS model (Suárez and Barber, 1992); and additional code was written as required for a truly integrative fate and effects model (Park, 1990, 1993). In the late 1990s, AQUATOX was restructured and linked to Microsoft Windows interfaces to provide even greater flexibility, capacity for additional compartments, and user friendliness.

- AQUATOX Release 1 was produced in 2002 and was the first EPA release to run under Windows.
- AQUATOX Release 2 was completed in 2003 and included more state variables and multi-age-class fish along with a refined user-interface.
- AQUATOX Release 2.1 was completed in 2005 and included additional chemical modeling options and variable stoichiometry among numerous other refinements.
- AQUATOX Release 2.2 was completed in 2006 and included updated simulations and parameter databases along with minor interface enhancements.
- AQUATOX Release 3 was completed in 2009 and includes linked segments, simulations of estuaries, dramatically improved output capabilities, and many other model improvements.

In 2009, EPA released an enhanced version of AQUATOX, Release 3, which includes the capability to represent estuaries and to more realistically model nutrients. More information on the model and its applications, including references to peer-reviewed publications, is available at http://www.epa.gov/waterscience/models/aquatox/.

Model Input

The model package contains a number of scenarios. The model guidance suggests to select a scenario from the model package, and modify it as needed to make it more representative for a specific situation and conditions. The model results are suggested to be used for screening-level assessments. For the purpose of the review presented here, the model was used to simulate the fate of imazamox in the default Missouri farm pond. The study on esfenvalerate in a Missouri Farm Pond was selected as a starting scenario. The state variable defined for this model scenario and their initial values are listed in the attached Table A1.1. The site characteristics and chemical parameters are shown in **attachment 1 and 2**. (will be included in final version!!!!!!!!!)

The site characteristic for the latitude was adjusted to 42 degrees in order to make it more representative for the light situation in MA. The dimensions of the pond were modified to represent the dimensions of the EPA standard pond: Surface area of 1 ha (10000 m^2), a depth of 2

m (6.56 ft), and a resulting volume of 20,000 m^3. Subsequently, depths of 1.2 m (3.94 ft), 0.91 m (3 ft) and 0.304 m (1ft) (with associated volumes) of this pond were also simulated.
Chemical Properties and Fate Data were adjusted to be representative of imazamox. The following parameter values were used (see also Section 2.4 in main document):
Molecular weight: 305; Henry's Law constant: 9/.76E-7 Pa m^3 mol^{-1}; Octanol-water partitioning constant (log): 0.73; Water partitioning coefficient: 50 L/kg; rate of anaerobic microbial degradation: 0.00198 d^{-1}(calculated using the half life value of 350 d and k = ln(20/half life); Maximum rate of aerobic microbial degradation: 0.00198 d^{-1} (see above); and photolysis rate: 0.069 d^{-1} (based on half-life of 10 d).

The herbicide application was programmed to occur on May 10th considering a scenario with the highest aquatic exposure level of an application to submerged vegetation with a maximum concentration of 500 ppb. The amount of imazamox for model input was determined based on the concentration of 500 ppb in the volume of the water body modeled, which varied with the depths that were considered. The following amounts of imazamox were used: 1524 g for a pond with a depth of 0.304 m (1ft); 4557 g with a depth of 0.91 m (3 ft); 6,000 g with a depth of 1.2 m (3.94 ft); and 10,000 g with a depth of 2 m (6.6 ft). The model simulation was run from May 1st through August 31st.

Results

From the model output, the following parameters were selected: dissolved imazamox concentration, imazamox photolyzed, imazamox biodegraded and total loss of imazamox. The results are presented in graphs that are shown below. Note the differences in the concentration scale in the graphs of ponds with different depths.
The depth of the pond appears to be an important factor in the dissipation rate of imazamox in the pond water. Dissipation rate is faster in a shallow pond. This may be attributed to a higher photolysis rate which is associated with more intense light penetration in a shallow pond. This is also illustrated in Fig. A1-6. The concentration trend after 1 and 2 months indicate the higher dissipation rates in shallower ponds.

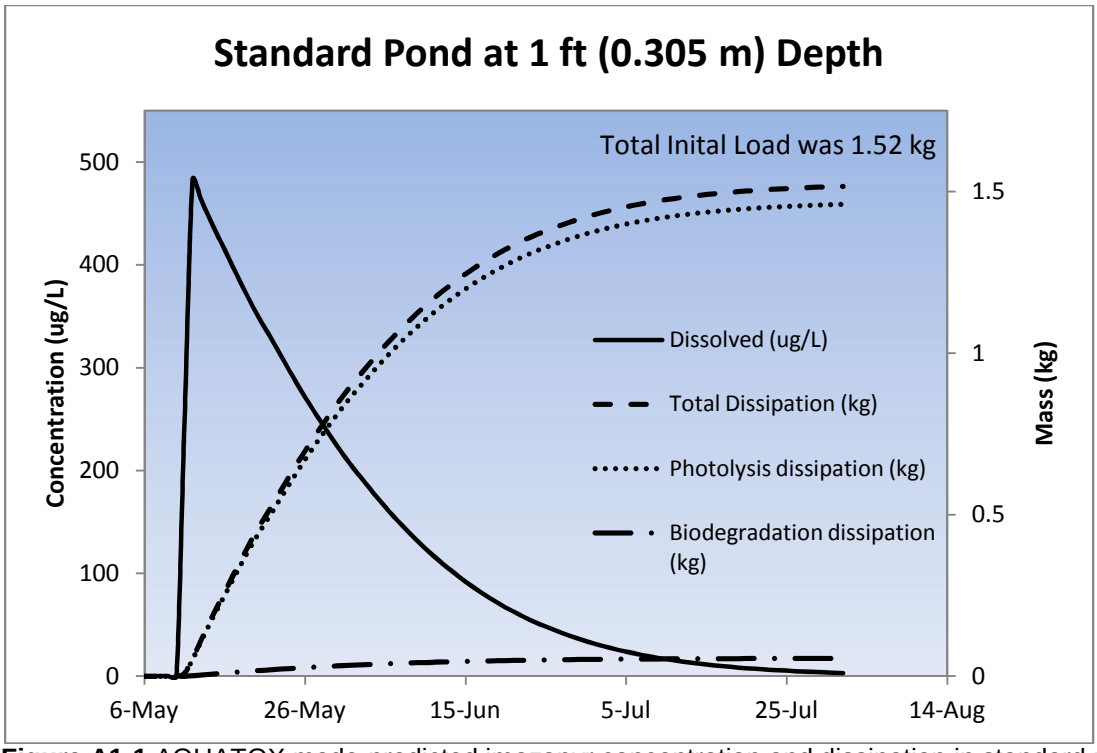

Figure A1-1 AQUATOX mode-predicted imazapyr concentration and dissipation in standard pond with 1 ft depth

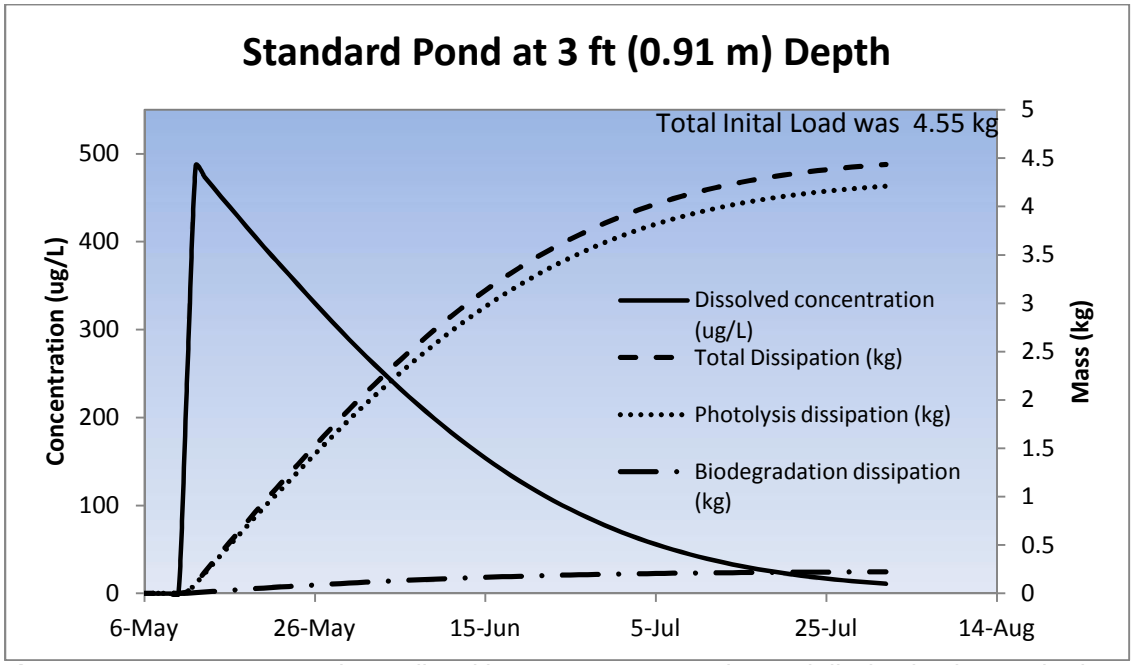

Figure A1-2 AQUATOX mode-predicted imazapyr concentration and dissipation in standard pond with 3 ft depth

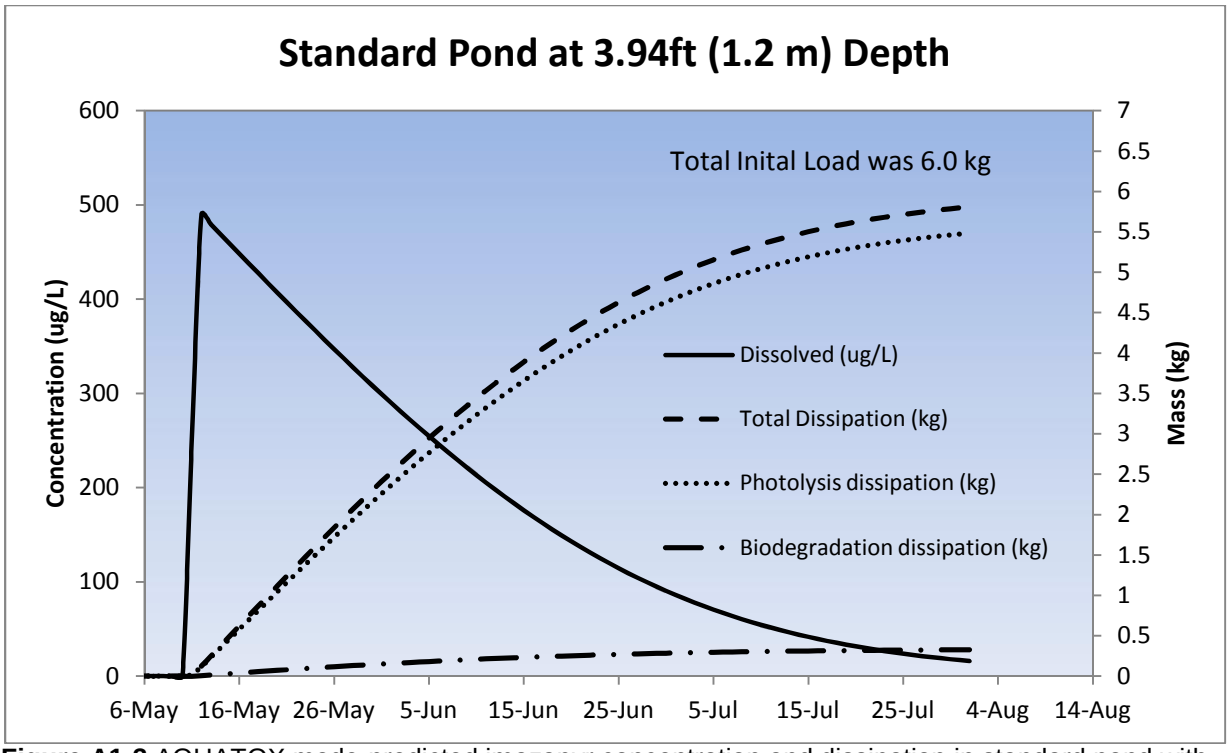

Figure A1-3 AQUATOX mode-predicted imazapyr concentration and dissipation in standard pond with 3,94 ft depth

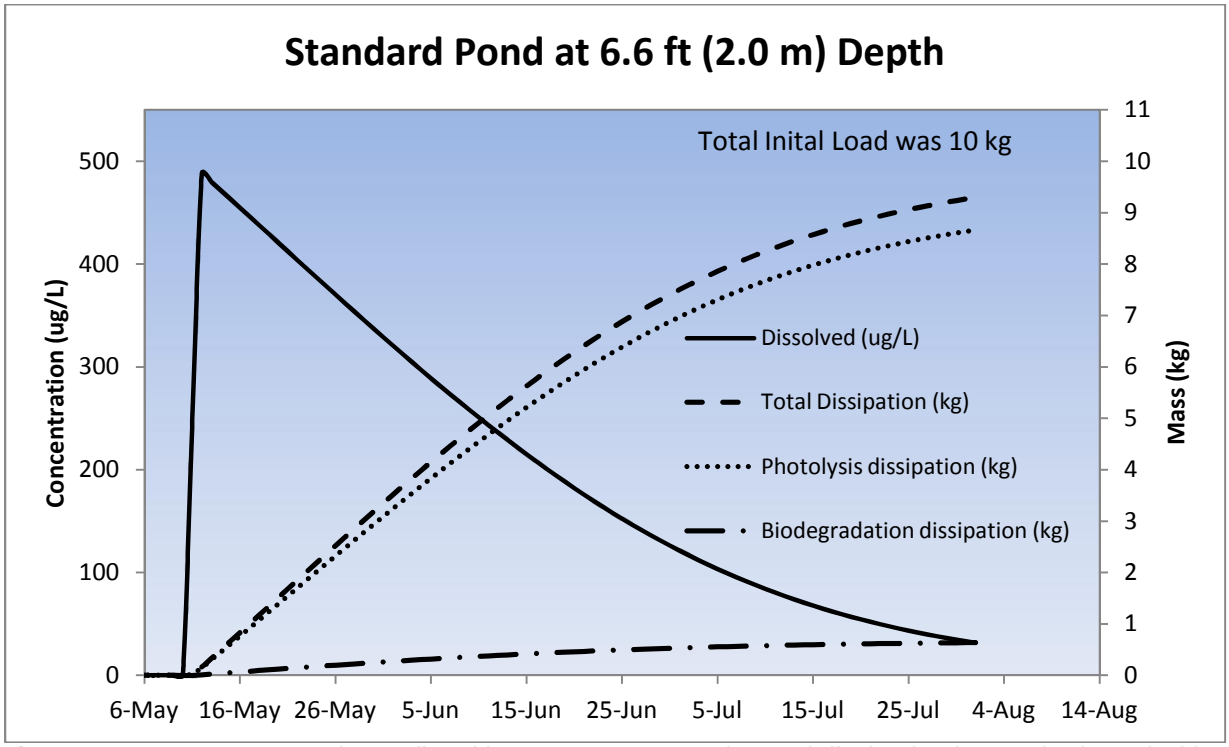

Figure A1-4 AQUATOX mode-predicted imazapyr concentration and dissipation in standard pond with 6.6 ft depth

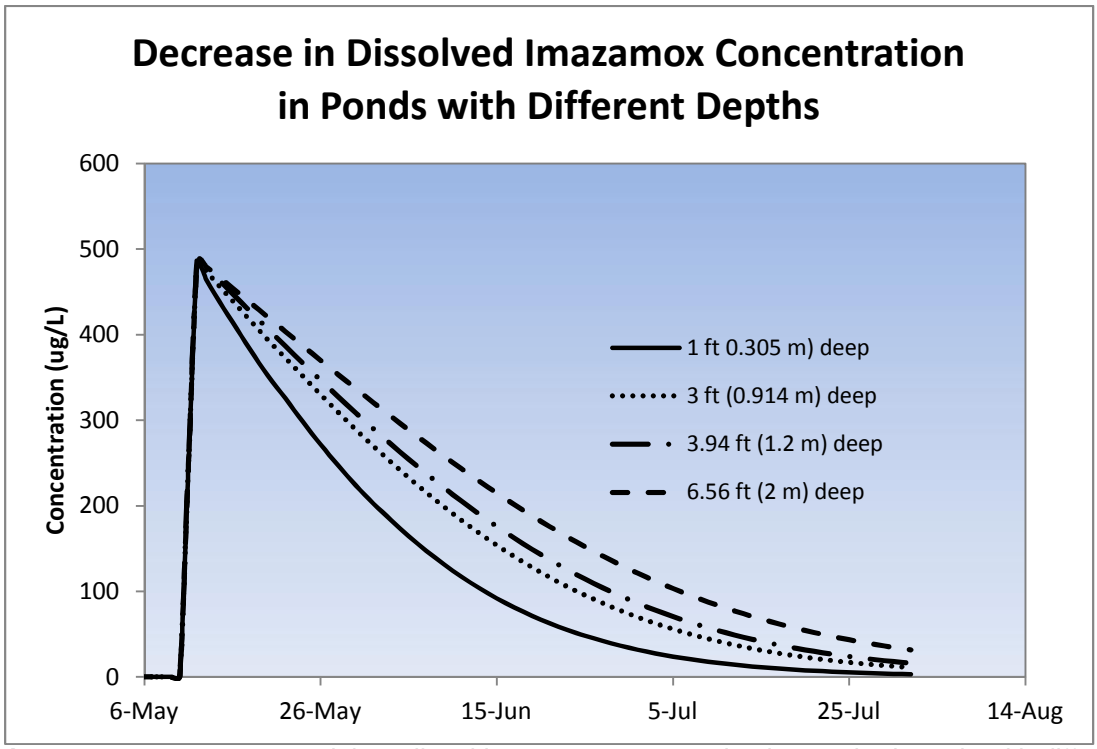

Figure A1-5 AQUATOX model-predicted imazapyr concentration in standard ponds with different depths

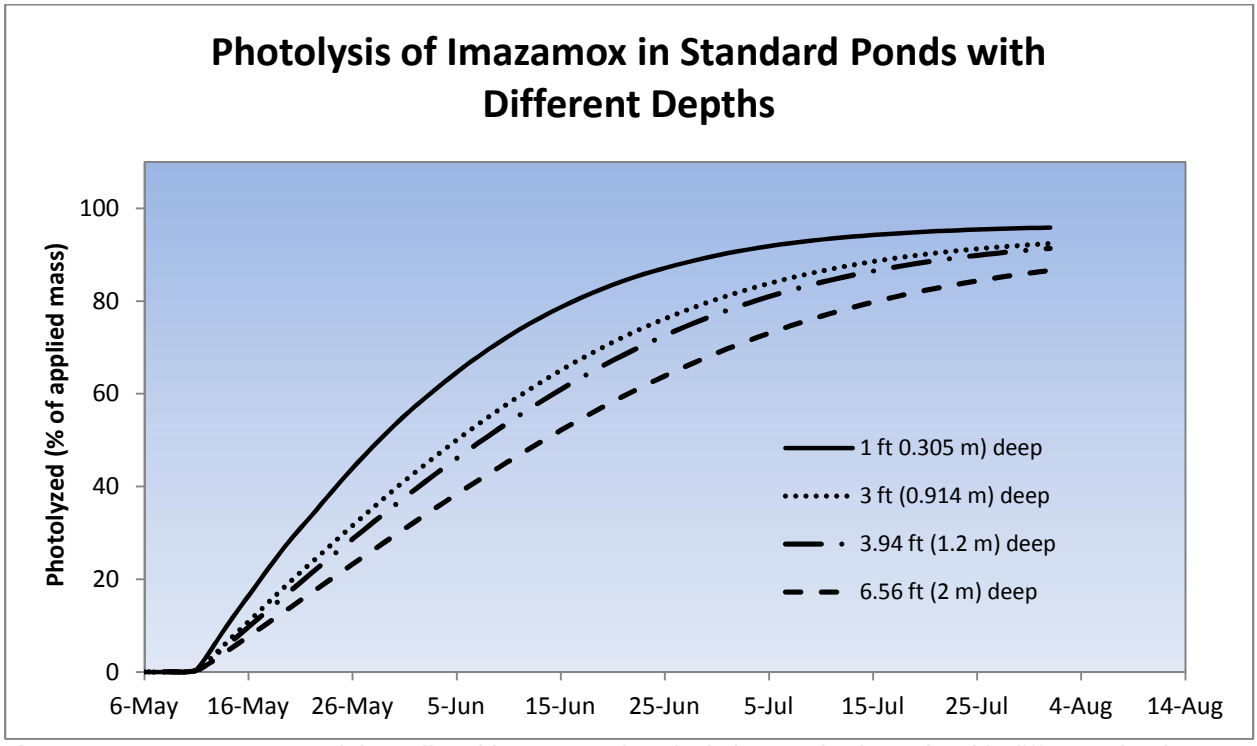

Figure A1-6 AQUATOX model-predicted imazapyr photolysis in standard ponds with different depths

Table A1.1 State Variables for Missouri Farm Pond and their initial values

State Variable Name	Init. Cond.	Units
NH3 & NH4+	0.08	mg/L
NO3	0.05	mg/L
Tot. Sol. P	0.05	mg/L
CO2	1.5	mg/L
Oxygen	12	mg/L
R detr sed	3	g/m2 dry
L detr sed	3	g/m2 dry
R detr diss	0.72	mg/L dry
L detr diss	0.18	mg/L dry
R detr part	0.08	mg/L dry
L detr part	0.02	mg/L dry
BuryRDetr	2	g/m2
BuryLDetr	2	g/m2
Peri High-Nut Diatom	36.86	g/m2 dry
Phyt High-Nut Diatom	0.00	mg/L dry
Peri, Green	0.01	g/m2 dry
Phyto, Green	0.00	mg/L dry
Phyt, Blue-Greens	0.00	mg/L dry
Cryptomonas	0.07	mg/L dry
Myriophyllum	36.67	g/m2 dry
Chironomid	2.29	g/m2 dry
Daphnia	0.05	mg/L dry
Copepod	0.32	mg/L dry
Sphaerid	2.46	g/m2 dry
Mayfly (Baetis)	0.24	g/m2 dry
Rotifer, Keratella	0.07	mg/L dry
Gastropod	3.68	g/m2 dry
Shiner	4.02	g/m2 dry
Largemouth Bass, YOY	0.21	g/m2 dry
Largemouth Bass, Lg	4.43	g/m2 dry
Water Vol	2004	cu.m
Temp	16	deg. C
Wind	0	m/s
Light	333	Ly/d
pH	6.8	pH