ChemicalWatch Factsheet

A Beyond Pesticides Factsheet

IMIDACLOPRID

Imidacloprid (1[(6-chloro-3-pyridinyl)methyl]-N-nitro-2-imidazolidinimine) is a systemic, chloro-nicotinyl insecticide used for the control of sucking insects such as fleas, aphids, whiteflies, termites, turf insects, soil insects, and some beetles. It is used on cotton and vegetable crops as foliar and seed treatments, soil, structures, indoor and outdoor insect control, home gardening and pet products. It was first registered in the U.S. in 1994 as the first chemical in its chemical class by Bayer CropScience. End-use products have pervaded the market place within the last 10 years, with the most popular marketed as Merit®, Admire®, Advantage™, Gaucho™, Provado®, Premise® and Imidicide™. Formulations are available as dustable powder, granular, seed dressing, soluble concentrate, suspension concentrate, and wettable powder. Data collected in 2005 showed that over 600,000 lbs of imidacloprid was used in the U.S. and this number has undoubtly grown. Although the use of imidacloprid has been gaining popularity in agricultural and residential settings, its human and environmental effects have not been fully evaluated, despite its registration over 10 years ago. While many in the industry consider imidacloprid to be a pesticide of relatively low toxicity, it has been found to be extremely toxic to non-target insects like bees, and recently has led to resistance in the Colorado potato beetle.

ChemicalWatch Stats

CAS Registry Number: 105827-78-9

Chemical Class: Chloro-nicotinyl or nicotinoid.

Use: Systemtic insecticide used for seed treatment, soil insects, termites and a host of other agricultural and residential insect pests.

Toxicity rating: Moderately toxic.

Signal Words: Caution, Warning.

Health Effects: It is linked to reproductive and mutagenic effects and is neurotoxic.

Environmental Effects: Highly toxic to bees and other beneficial insects,and is toxic to upland. game birds. It is generally persistent in soils and can leach to groundwater.

Mode of Action

Imidacloprid belongs to the nicotinoid chemical family, a family of chemicals similar to the tobacco chemical, nicotine. It works by interfering with the transmission of stimuli in the insect nervous system causing irreversible blockage of acetylcholine receptors, which are found in a type of neural pathway that is more abundant in insects than in warm-blooded animals. These receptors are rendered incapable of receiving acetylcholine molecules (an important neurotransmitter) and an accumulation of acetylcholine occurs, resulting in the insect's paralysis and eventual death. It is effective on contact and via stomach action.

Acute Toxicity

Imidacloprid is classified by the Environmental Protection Agency (EPA) as both a toxicity class II and class III pesticide (on a scale of I to IV, I being the highest toxicity class), and must be labeled with the signal word "Warning" or "Caution." Symptoms of acute exposure would be similar to nicotinic signs and are expected to be diarrhea, fatigue, twitching, salivation, convulsions, cramps, and muscle weakness including the muscles necessary for breathing. Symptoms can last for five days following exposure. Imidacloprid is quickly and nearly completely absorbed from the gas¬trointestinal tract and eliminated in urine and feces.

The airborne concentration that resulted in mortality to half of the test organisms (LC50) is >69 mg/m3 air in the form of an aerosol, and >5323 mg/m3 in air in the form of dust. It is considered non-irritating to eyes and skin, and non-sensitizing to skin, though some granular formulations may contain clay as an inert ingredi¬ent, which may act as an eye irritant.[3]

Chronic Toxicity

Chronic or long term toxicity of imidacloprid is linked to reproductive and mutagenic effects at relatively high doses. Studies with laboratory rats fed imidacloprid over two years resulted in decreased body weight and increased thyroid lesions. Elevated blood cholesterol levels and stress to the liver were also observed in dogs fed imidacloprid for 1-year. It categorized as a "Group E" carcino¬gen (evidence of non-carcinogenicity for humans) by the EPA.

Reproductive Effects

A three generation reproduction study in rats fed up to 700 ppm imidacloprid resulted in decreased pup body weights. An increase in the frequency of miscarriages and an increase in the number of offspring with abnormal skeletons were observed in pregnant rabbits fed a dose of 72mg/kg/day.

Neurotoxic Effects

Tremors, uncoordinated gait and decreased activity were observed in male rats fed 310 mg/kg imidacloprid. A dose-related decrease in a measure of motor and locomotor activity was observed. Most clinical signs of toxicity were resolved after 1- 5 days of treatment. In EPA studies, neurotoxicity was characterized by decreases in motor or locomotor activity in female rats at 42 mg/kg/day.

Mutagenic Effects

Imidacloprid may be weakly mutagenic. It has been determined that imidacloprid increased the frequency of genetic damage by chemically binding to DNA. It also tested positive for causing changes in human lymphocyte chro¬mosomes and for genotoxicity in Chinese hamster ovary cells.[4]

Metabolites

There are several break-down products or metabolites of imidacloprid, many with toxic effects. The main urinary metabolites are 6-chloronicotinic acid and two monohydroxylated metabolites (5-hydroxyimidacloprid) and (4-hydroxyimidacloprid), among others. The nitroso metaboloite (1-(6-chloro-3-pyridylmethyl)-N-nitroso(imidazolidin-2-ylideneamine) when fed to rats in drinking water led to higher lymphocyte counts and lower numbers of polymorphonuclear cells (a category of white blood cells).[4]

One metabolite found in imidacloprid-treated plants, called the olefin metabolite (1-(6-Chloro-3-pyridylmethyl)-N-nitro(4-imidazolin-2-ylidene)amine), is more toxic to insects than imidacloprid itself. The guanidine metabolite however, does not possess insecticidal properties, but has a higher mammalian toxicity than the parent compound.

Ecological Effects

Imidacloprid is toxic to upland game and birds especially Japanese quail, house sparrow, canary and pigeons. So toxic is imidacloprid to birds that the EPA concluded that the 'levels of concern' for secondary exposures were exceeded for both non-endangered and endangered songbirds.[2] Imidacloprid causes abnormal behavior, such as lack of coordination, lack of responsiveness and an inability to fly, in birds for which it is not considered highly toxic, such as mallards. Other adverse effects observed include eggshell thinning (at exposures of 61mg/kg), decreased weight (at exposures of 150 ppm) in food) and reduced egg production and hatching success. Imidacloprid also appears to repel birds when used as a seed treatment.

It is of moderate to low toxicity to fish and extremely toxic to some species of freshwater and estuarine crustaceans. Earthworms exposed to imidacloprid experience reproductive and mutagenic effects, even at low concentrations. Despite being an insecticide, imidacloprid can be toxic to plants should drift and runoff occur. Cases documenting damage to greenhouse crops exposed to imidacloprid have been reported. Imidacloprid can also reduce blue-green algal communities and diatoms at moderate concentrations.[2]

Beneficial Insects

Honeybees: Imidacloprid is highly toxic to bees when used in foliar applications, and most recently has been identified, along with other pesticides in its chemical class, as one of the pesticides that may be responsible for the decline in honeybee populations in the U.S. and abroad. The rapid disappearance of the honeybees, also dubbed "Colony Collapse Disorder" or CCD, has been observed in the U.S. since 2006. This prompted researchers to investigate the cause of this phenomenon and regulators have found studies to be inconclusive. Despite this, imidacloprid has been linked to sublethal effects in honeybees, which include disruptions in mobility, navigation, and feeding behavior. Lethal and sublethal exposures to imidacloprid have been shown to decrease foraging activity, along with olfactory learning performance and decrease hive activity. Bees are exposed when they pollinate flowering crops treated with imidacloprid, or pesticide drift (via wind) from surrounding areas. Honeybees intercept, and are contaminated by, particles on crops and suspended in the air, and retain them in their hair and/or accumulate them in their bodies and hives, leading to loses of entire colonies. The effects of CCD can be especially devastating since honeybees are essential pollinators of crops that constitute over one third of the U.S. food supply or $15 billion worth of food.

Other Insects: Spiny soldier bugs, whiteflies, ladybirds, lacewings and mired bugs- all beneficial predators- have also been adversely affected when exposed to imidacloprid. This can lead to a resurgence in pests they would normally prey on, which results in an increase in crop damage.

Pet Products

Imidacloprid pet products, such as Advantage™, are used to control fleas on cats and dogs, and carry the signal word "Warning" on the product label.8 Imidacloprid, when applied, spreads over the body with the body oils, and collects in the hair follicles and then is released over time (up to one month) from the hair follicles onto the skin and hair. Skin irritation has been reported, and in one severe case, an already sick cat developed a severe rash at the application spot, which led to intestinal problems, heart failure and death.2 It is suspected that exposure to imidacloprid worsened the cat's condition. Even though imidacloprid is more toxic to insects than mammals, its guanidine metabolite has a

higher toxicity to mammals. Despite this, exposures to pets and humans have not been fully researched.

Environmental Fate

In soil, imidacloprid has the ability to readily leach due to its high water solubility and its inability to adhere to soil particles. Several soil half-lives have been reported for imidacloprid under various soil conditions ranging from 27-229 days.[6] The soil half life of imidacloprid tends to increase with soil pH, as well as with the absence of light. It is this persistence that makes imidacloprid suitable for seed treatment and for other soil application. It is also more persistent in bare soils than in soil with plant cover. The fate of imidacloprid in the soil is also highly sensitive to soil composition and sources of organic carbon.[6]

Imidacloprid has a short half life in surface water and is rapidly broken down in water by sunlight. However, it is more persistent in groundwater. Groundwater tests in the state of New York have detected imidacloprid at concentrations ranging from less than 0.1 ppb to 1.0 ppb. The State of California has placed imidacloprid on the Ground Water Protection List due to its potential to contaminate groundwater.[6] Despite the ability of imidacloprid to contaminate groundwater and its registered uses for residential and agricultural settings, the EPA has not listed it as a Restricted Use pesticide, citing economic reasons.[2]

Imidacloprid has low volatility, with a vapor pressure of 1.0 x 10-7 mmHg. It also it has a low potential to be dispersed in air over a large area via air borne soil particles, since it does not readily adsorb to particles. However, imidacloprid still has the ability to drift as a result of the dispersal of tiny seed debris from imidacloprid-treated seeds that have been lost by way of the mechanism of seed drills used to plant fields. When used as a seed treatment, imidacloprid is readily translocated though seedling/plant tissues, including leaves, flowers and pollen.

Resistance

Insect resistance to pesticides is a phenomenon that undermines pest control and carries a heavy economic burden. Imidacloprid resistance in the Colorado potato beetle appeared after only two years of imidacloprid use on potatoes in Michigan. Resistance was also reported from several locations in New York, Delaware and southern Maine.

Regulatory Status

Imidacloprid is registered as a general use pesticide, classified by the EPA as both a toxicity class II and class III chemical. Based on its risk assessments, the agency concluded that the risk to the U.S. population from aggregate exposure to imidacloprid residues is acceptable.[3] There are food tolerances for residues of imidacloprid and its metabolites on food/feed additives ranging from 0.02 ppm in eggs to 3.0 ppm in hops.[2]

The use of imidacloprid (Gaucho™) was severely restricted in France after it was suspected to be responsible for the decline of honeybee populations. In 1999, the French Ministry of Agriculture took the precautionary approach and suspended the use of imidacloprid on sunflowers in an attempt to limit the risks of exposing bees to the potentially detrimental effects of Gaucho, much to the dismay of Bayer, which claims that no relationship could be found between the application of Gaucho and reduction in the bee population. Since then the restrictions on the use of imidacloprid have been extended several times and include several other crops. Similar concerns about imidacloprid have also been raised in Canada where high rates of bee colony loses have also been reported. In June 2008, Germany suspended eight toxic insecticides, including products containing imidacloprid, believed to be associated with the bee decline.

Citations

[1] USDA Forest Service. 2005. Imidacloprid- Human Health and Ecological Risk Assessment Final Report. Arlington VA

[2] Cox, C. 2001. Factsheet: Imidacloprid. J. of Pesticide Reform Vol.21(1)

[3] Extension Toxicology Network (EXTOXNET). 1995. Pesticide Information Profile: Imidacloprid. http://pmep.cce.cornell.edu/profiles/extoxnet/haloxyfop-methylparathion/imidacloprid-ext.html

[4] INCHEM. 2001. Toxicological Evaluations: Imidacloprid. International Program on Chemical Safety(IPCS) http://www.inchem.org/documents/jmpr/jmpmono/2001pr07.htm#2.2.3

[5] U.S. EPA. 1998. Imidacloprid; Pesticide Tolerances. Federal Registrar Sept 18, 1998 Volume 63, Number 181

[6] Fossen, M. 2006. Environmental Fate of Imidacloprid. California Dept of Pesticide Regulation http://www.cdpr.ca.gov/docs/emon/pubs/fatememo/Imidclprdfate2.pdf

[7] Pesticide Action Network UK. 2003. Imidacloprid. http://www.pan-uk.org/pestnews/Actives/imidaclo.htm

[8] National Pesticide Telecommunication network (NPTN). 1998. Factsheet: Imidacloprid. Oregon State University. http://npic.orst.edu/factsheets/imidacloprid.pdf

[9] Desneaux, N. et al., 2007. Sublethal Effects of Pesticides on Benefical Anthropods. Annual Review of Entomology, 52:81-106

[10] Decourtye, A. et al., 2004. Effects of imidacloprid and deltamethrin on associative learning in honeybees under semi-field and laboratory conditions. Ecotoxicology and Environmental Safety.57: 410-419

[11] U.S. EPA. 2008. Pesticide issues in the works: honey bee colony collapse disorder. http://www.epa.gov/pesticides/about/intheworks/honeybee.htm

[12] Alyokin, A. et al. 2006. Resistance and cross-resistance to imidacloprid and thiamethoxam in the Colorado potato beetle Leptinotarsa decemlineata. Pest Management Science, 63(1):32-41