IMIDACLOPRID
TECHNICAL FACT SHEET

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NATIONAL PESTICIDE INFORMATION CENTER
1.800.858.7378

NPIC Technical Fact Sheets provide information that is complex and intended for individuals with a scientific background and/or familiarity with toxicology and risk assessment. This document is intended to promote informed decision-making. Please refer to the General Fact Sheet for less technical information.

Chemical Class and Type:

- Imidacloprid is a neonicotinoid insecticide in the chloronicotinyl nitroguanidine chemical family.[1,2] The International Union of Pure and Applied Chemistry (IUPAC) name is 1-(6-chloro-3-pyridylmethyl)-N-nitroimidazolidin-2-ylideneamine and the Chemical Abstracts Service (CAS) registry number is 138261-41-3.[2]

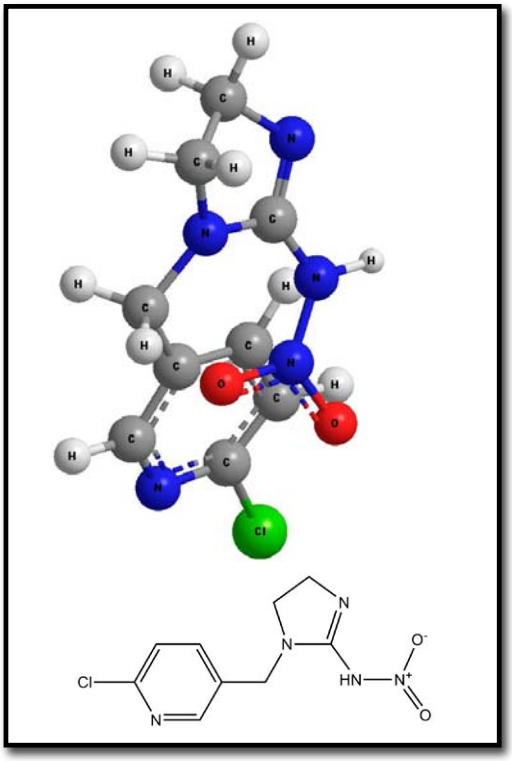
Laboratory Testing: Before pesticides are registered by the U.S. EPA, they must undergo laboratory testing for short-term (acute) and long-term (chronic) health effects. Laboratory animals are purposely given high enough doses to cause toxic effects. These tests help scientists judge how these chemicals might affect humans, domestic animals, and wildlife in cases of overexposure.

- Neonicotinoid insecticides are synthetic derivatives of nicotine, an alkaloid compound found in the leaves of many plants in addition to tobacco.[3,4,5]
- Imidacloprid was first registered for use in the U.S. by the United States Environmental Protection Agency (U.S. EPA) in 1994.[6] See the text box on **Laboratory Testing**.

Physical / Chemical Properties:

- Imidacloprid is made up of colorless crystals with a slight but characteristic odor.[2]
- Vapor pressure[7]: 3 x 10^{-12} mmHg at 20 °C
- Octanol-Water Partition Coefficient (log K_{ow})[2]: 0.57 at 21 °C
- Henry's constant[2]: 1.7 x 10^{-10} Pa·m^3/mol
- Molecular weight[2]: 255.7 g/mol
- Solubility (water)[2]: 0.61 g/L at 20 °C
- Soil Sorption Coefficient (K_{oc})[8,9]: 156-960, mean values 249-336

Molecular Structure - Imidacloprid



Uses:

- Imidacloprid is used to control sucking insects, some chewing insects including termites, soil insects, and fleas on pets. In addition to its topical use on pets, imidacloprid may be applied to structures, crops, soil, and as a seed treatment.[2,10] Uses for individual products containing imidacloprid vary widely. Always read and follow the label when applying pesticide products.
- Signal words for products containing imidacloprid may range from Caution to Danger. The signal word reflects the combined toxicity of the active ingredient and other ingredients in the product. See the pesticide label on the product and refer to the NPIC fact sheets on ***Signal Words*** and ***Inert or "Other" Ingredients***.
- To find a list of products containing imidacloprid which are registered in your state, visit the website http://npic.orst.edu/reg/state_agencies.html and search by "active ingredient."

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Mode of Action:

Target Organisms

- Imidacloprid is designed to be effective by contact or ingestion.[2] It is a systemic insecticide that translocates rapidly through plant tissues following application.[2,10]
- Imidacloprid acts on several types of post-synaptic nicotinic acetylcholine receptors in the nervous system.[11,12] In insects, these receptors are located only within the central nervous system. Following binding to the nicotinic receptor, nerve impulses are spontaneously discharged at first, followed by failure of the neuron to propagate any signal.[13,14] Sustained activation of the receptor results from the inability of acetylcholinesterases to break down the pesticide.[12] This binding process is irreversible.[5]

Non-Target Organisms

- Imidacloprid's mode of action is similar on target and non-target beneficial insects including honeybees, predatory ground beetles and parasitoid wasps.[10] However, imidacloprid is ineffective against spider mites and nematodes.[2]
- Mammalian nicotinic receptors are made up of a number of subtypes[14] In contrast to insects, these receptors are present at neuromuscular junctions as well as in the central nervous system.[14] However, the binding affinity of imidacloprid at the nicotinic receptors in mammals is much less than that of insect nicotinic receptors.[15] This appears to be true of other vertebrate groups including birds.[16,17]
- The blood-brain barrier in vertebrates blocks access of imidacloprid to the central nervous system, reducing its toxicity.[14]

Acute Toxicity:

LD_{50}/LC_{50}: A common measure of acute toxicity is the lethal dose (LD_{50}) or lethal concentration (LC_{50}) that causes death (resulting from a single or limited exposure) in 50 percent of the treated animals. LD_{50} is generally expressed as the dose in milligrams (mg) of chemical per kilogram (kg) of body weight. LC_{50} is often expressed as mg of chemical per volume (e.g., liter (L)) of medium (i.e., air or water) the organism is exposed to. Chemicals are considered highly toxic when the LD_{50}/LC_{50} is small and practically non-toxic when the value is large. However, the LD_{50}/LC_{50} does not reflect any effects from long-term exposure (i.e., cancer, birth defects or reproductive toxicity) that may occur at levels below those that cause death.

Oral

- Imidacloprid is moderately toxic if ingested.[18] Oral LD_{50} values in rats were estimated to be 450 mg/kg for both sexes in one study and 500 and 380 mg/kg in males and females, respectively in another study.[2,19] In mice, LD_{50} values were estimated at 130 mg/kg for males and 170 mg/kg for females.[19,20] See the text boxes on **Toxicity Classification** and **LD_{50}/LC_{50}**.

Dermal

- Imidacloprid is very low in toxicity via dermal exposure.[18] The dermal LD_{50} in rats was estimated at greater than 5000 mg/kg.[2,19]
- Researchers did not observe eye or skin irritation in rabbits.[19,20] Imidacloprid is not considered a skin sensitizer[20] although reports of hypersensitivity in skin following exposure to imidacloprid have been reported in companion animals.[1]

Inhalation

- Imidacloprid is variable in toxicity if inhaled. The inhalation LC_{50} was estimated to be greater than 5323 mg/m^3 for dust and 69 mg/m^3 for aerosol exposure in rats.[2,20] Imidacloprid dust is considered slightly toxic but the aerosol form is highly toxic[18]

Signs of Toxicity - Animals

- Salivation and vomiting have been reported following oral exposure.[1,6] Very high oral exposures may lead to lethargy, vomiting, diarrhea, salivation, muscle weakness and ataxia, which are all indicative of imidacloprid's action on nicotinic receptors.[1] Other signs of exposure at high doses are uncoordinated gait, tremors, and reduced activity.[20]

TOXICITY CLASSIFICATION - IMIDACLOPRID

	High Toxicity	Moderate Toxicity	Low Toxicity	Very Low Toxicity
Acute Oral LD_{50}	Up to and including 50 mg/kg (≤ 50 mg/kg)	**Greater than 50 through 500 mg/kg (> 50 – 500 mg/kg)**	Greater than 500 through 5000 mg/kg (> 500 – 5000 mg/kg)	Greater than 5000 mg/kg (> 5000 mg/kg)
Inhalation LC_{50}	**Up to and including 0.05 mg/L (≤ 0.05 mg/L) (aerosol)**	Greater than 0.05 through 0.5 mg/L (>0.05 – 0.5 mg/L)	Greater than 0.5 through 2.0 mg/L (> 0.5 – 2.0 mg/L)	**Greater than 2.0 mg/L (> 2.0 mg/L) (dust)**
Dermal LD_{50}	Up to and including 200 mg/kg (≤ 200 mg/kg)	Greater than 200 through 2000 mg/kg (> 200 - 2000 mg/kg)	Greater than 2000 through 5000 mg/kg (>2000 – 5000 mg/kg)	**Greater than 5000 mg/kg (> 5000 mg/kg)**
Primary Eye Irritation	Corrosive (irreversible destruction of ocular tissue) or corneal involvement or irritation persisting for more than 21 days	Corneal involvement or other eye irritation clearing in 8 – 21 days	Corneal involvement or other eye irritation clearing in 7 days or less	**Minimal effects clearing in less than 24 hours**
Primary Skin Irritation	Corrosive (tissue destruction into the dermis and/or scarring)	Severe irritation at 72 hours (severe erythema or edema)	Moderate irritation at 72 hours (moderate erythema)	**Mild or slight irritation at 72 hours (no irritation or erythema)**

The highlighted boxes relfect the values in the "Acute Toxicity" section of this fact sheet. Modeled after the U.S. Environmental Protection Agency, Office of Pesticide Programs, Label Review Manual, Chapter 7: Precautionary Labeling. http://www.epa.gov/oppfead1/labeling/lrm/chap-07.pdf

- Hypersensitivity reactions in skin have been reported following dermal applications of products containing imidacloprid.[1]
- Onset of signs of toxicity is rapid following acute exposure. In rats, clinical signs of intoxication occurred within 15 minutes of oral exposure.[14,21] Signs of toxicity disappear rapidly, with most resolving within 24 hours of the exposure. Lacrimation and urine staining may persist for up to four days after exposure to some neonicotinoids. Death occurred within 24 hours following administration of lethal doses.[21]
- Neither persistent neurotoxic effects nor effects with a delayed onset have been reported for imidacloprid.[21]

Signs of Toxicity - Humans

- Three case reports of attempted suicides described signs of toxicity including drowsiness, dizziness, vomiting, disorientation, and fever.[22,23,24] In two of these cases, the authors concluded that the other ingredients in the formulated product ingested by the victims were more likely to account for many of the observed signs.[22,23]
- A 69-year-old woman ingested a formulated product containing 9.6% imidacloprid in N-methyl pyrrolide solution. The woman suffered severe cardiac toxicity and death 12 hours after the exposure.[25] Signs of toxicity soon after the ingestion included disorientation, sweating, vomiting, and increased heart and respiratory rates.[25]
- A 24-year-old man who accidentally inhaled a pesticide containing 17.8% imidacloprid while working on his farm was disoriented, agitated, incoherent, sweating and breathless following the exposure.[26]
- Pet owners have reported contact dermatitis following the use of veterinary products containing imidacloprid on their pets.[19]
- Always follow label instructions and take steps to avoid exposure. If any exposures occur, be sure to follow the First Aid instructions on the product label carefully. For additional treatment advice, contact the Poison Control Center at 1-800-222-1222. If you wish to report an incident, please call 1-800-858-7378.

Chronic Toxicity:

Animals

- Rats consumed imidacloprid in their diet for three months at doses of 14, 61, and 300 mg/kg/day for males and 20, 83, and 420 mg/kg/day for females. Researchers noted reductions in body weight gain, liver damage, and reduced blood clotting function and platelet counts at 61 mg/kg/day in males and 420 mg/kg/day in females. Liver damage disappeared after exposure ended, but abnormalities in the blood were not entirely reversible. Researchers estimated the NOAEL at 14 mg/kg/day.[27] See the text box on **NOAEL, NOEL, LOAEL,** and **LOEL**.

NOAEL: No Observable Adverse Effect Level

NOEL: No Observed Effect Level

LOAEL: Lowest Observable Adverse Effect Level

LOEL: Lowest Observed Effect Level

- Imidacloprid dust was administered through the noses of rats for six hours a day, five days a week for four weeks at concentrations of 5.5, 30.0, and 190.0 mg/m^3. Male rats exhibited reduced body weight gain at the two highest doses and at the highest dose, increased liver enzyme activity and increased blood coagulation time was noted. Female rats exhibited increased liver enzyme activity at the two highest doses and at the highest dose, researchers noted enlarged livers and reduced thrombocyte counts. No effects were observed at the lowest dose.[28]

- Researchers applied a paste containing 1000 mg/kg imidacloprid to the shaved flanks and backs of rabbits, exposing the rabbits for 6 hours a day for 15 days. Rabbits showed no effects from the treatment.[29]

- Researchers fed imidacloprid to beagles for one year. Concentrations were 200, 500, or 1250 ppm for the first 16 weeks and 200, 500, and 2500 ppm for the remainder of the trial. Doses were equivalent to 6.1, 15.0, and 41.0 or 72.0 mg/kg/day. Researchers noted reduced food intake in the highest dose group. Females in this group exhibited increased plasma cholesterol concentrations at 13 and 26 weeks. Both males and females in this group exhibited increased cytochrome P450 activity in the liver and increases in liver weights at the end of the study. No adverse effects were observed at the two lowest doses.[30]

Humans

- No studies were found involving human subjects chronically exposed to imidacloprid. See the text box on **Exposure**.

Exposure: Effects of imidacloprid on human health and the environment depend on how much imidacloprid is present and the length and frequency of exposure. Effects also depend on the health of a person and/or certain environmental factors.

- The chronic dietary reference dose (RfD) has been set at 0.057 mg/kg/day based on chronic toxicity and carcinogenicity studies using rats. The NOAEL was estimated to be 5.7 mg/kg/day and the LOAEL was set at 16.9 mg/kg/day based on increased occurrence of mineralized particles in the thyroid gland of male rats.[31] See the text box on **Reference Dose (RfD)** (page 10).

Endocrine Disruption:

- No data were found evaluating the potential of imidacloprid to disrupt endocrine function.

- Imidacloprid is included in the draft list of initial chemicals for screening under the U.S. EPA Endocrine Disruptor Screening Program (EDSP).[32] The list of chemicals was generated based on exposure potential, not based on whether the pesticide is a known or likely potential endocrine disruptor.

Carcinogenicity:

Animals

- Researchers concluded that Scottish terriers treated with topical flea and tick products, including those containing imidacloprid, did not have a greater risk of developing urinary bladder cancer compared with control dogs.[33] Rats were fed imidacloprid for 18 or 24 months at unspecified concentrations. Although signs of toxicity were noted, researchers concluded that imidacloprid showed no evidence of carcinogenic potential.[20]
- A range of studies using both *in vitro* and *in vivo* techniques concluded that imidacloprid did not damage DNA.[19]

Humans

- The U.S. EPA has classified imidacloprid into Group E, no evidence of carcinogenicity, based on studies with rats and mice.[20,31] See the text box on **Cancer**.

> **Cancer: Government agencies in the United States and abroad have developed programs to evaluate the potential for a chemical to cause cancer. Testing guidelines and classification systems vary. To learn more about the meaning of various cancer classification descriptors listed in this fact sheet, please visit the appropriate reference, or call NPIC.**

- Imidacloprid has not been evaluated for the carcinogenicity by the International Agency for Research on Cancer (IARC), nor the National Toxicology Program (NTP).
- A study of human lymphocytes exposed to greater than 5200 µg/ml of imidacloprid demonstrated a slight increase in chromosome abnormalities *in vitro*, but this result was not found with *in vivo* tests.[19]

Reproductive or Teratogenic Effects:

Animals

- Rats were fed imidacloprid at doses of 10, 30, or 100 mg/kg/day on days 6 to 15 of their pregnancies.[20] On day 21 of the pregnancy, rats at the highest doses showed reduced embryo development and signs of maternal toxicity. In addition, wavy ribs were observed in the fetuses.[20,34]
- Researchers fed rabbits doses of imidacloprid at 8, 24, or 72 mg/kg/day during days 6-18 of pregnancy. On day 28 of pregnancy, researches noted maternal toxicity including death in the highest dose group, and the animals that survived in this group carried embryos with reduced rates of growth and bone ossification. In some of these rabbits, the young were aborted or resorbed.[20,35]
- In a two-generation study of reproductive toxicity, researchers dosed rats with 100, 250, or 700 ppm of imidacloprid in their diet for 87 days until rats mated. This was equivalent to 6.6, 17.0, and 47.0 mg/kg/day. Mother rats exhibited increased O-demethylase activity at doses of 17 mg/kg/day and greater. Reduced body weight gains were noted in pups at doses of 47 mg/kg/day. No effects on reproductive behavior or success were observed.[20,36]

Humans

- No human data were found on the reproductive effects of imidacloprid.

Fate in the Body:

Absorption

- The gastrointestinal tract of rats absorbed 92% of an unspecified dose. Plasma concentrations peaked 2.5 hours after administration.[19]
- Little systemic absorption through the skin occurs following dermal exposure in pets.[1]

- Researchers tested imidacloprid absorption using human intestinal cells. Cells rapidly absorbed imidacloprid at a very high rate of efficiency. Researchers concluded that an active transport system was involved.[37]

Distribution

- Researchers administered a single oral dose of radio-labeled imidacloprid at 20 mg/kg to male rats. One hour after dosing, imidacloprid was detected throughout the bodies with the exception of fatty tissues and the central nervous system.[38]
- No studies were found examining the distribution of imidacloprid in humans.

Metabolism

- Mammals metabolize imidacloprid in two major pathways discussed below. Metabolism occurs primarily in the liver.[20]
- In the first pathway, imidacloprid may be broken by oxidative cleavage to 6-chloronicotinic acid and imidazolidine. Imidazolidine is excreted in the urine, and 6-chloronicotinic acid undergoes further metabolism via glutathione conjugation to form mercaptonicotinic acid and a hippuric acid.[20,39]
- Imidacloprid may also be metabolized by hydroxylation of the imidazolidine ring in the second major pathway.[20,39] Metabolic products from the second pathway include 5-hydroxy and olefin derivatives.[40]

Excretion

- The metabolic products 5-hydroxy and olefin derivatives resulting from hydroxylation of the imidazolidine ring are excreted in both the feces and urine.[39,41]
- Metabolites found in urine include 6-chloronicotinic acid and its glycine conjugate, and accounted for roughly 20% of the original radio-labeled dose.[42]
- Metabolites in the feces accounted for roughly 80% of the administered dose in rats and included monohydroxylated derivatives in addition to unmetabolized imidacloprid, which made up roughly 15% of the total. Olefin, guanidine, and the glycine conjugate of methylthionicotinic acid were identified as minor metabolites.[2,42]
- Rats excreted 96% of radio-labeled imidacloprid within 48 hours following an unspecified oral dosing, with 90% excreted in the first 24 hours.[40] Radio-labeled imidacloprid was present in low amounts in organs and tissues 24 hours after male rats were orally dosed with 20 mg/kg.[38]
- No information was found on the specific metabolism of imidacloprid in humans.

Medical Tests and Monitoring:

- Researchers have tested for imidacloprid exposure in farm workers by evaluating urine samples with high performance liquid chromatography.[43] The method has not been well studied in humans and the clinical significance of detected residues is unknown.

Environmental Fate:

Soil

- Soil half-life for imidacloprid ranged from 40 days in unamended soil to up to 124 days for soil recently amended with organic fertilizers.[44] See the text box on **Half-life**.
- Researchers incubated three sandy loams and a silt loam in darkness following application of [^{14}C-*methylene*]-imidacloprid for a year. The

The "half-life" is the time required for half of the compound to break down in the environment.

1 half-life	**=**	**50% remaining**
2 half-lives	**=**	**25% remaining**
3 half-lives	**=**	**12% remaining**
4 half-lives	**=**	**6% remaining**
5 half-lives	**=**	**3% remaining**

Half-lives can vary widely based on environmental factors. The amount of chemical remaining after a half-life will always depend on the amount of the chemical originally applied. It should be noted that some chemicals may degrade into compounds of toxicological significance.

degradation time required for imidacloprid to break down to half its initial concentration (DT_{50}) in non-agricultural soil was estimated to be 188-997 days. In cropped soils, the DT_{50} was estimated to be 69 days.[42] Metabolites found in the soil samples included 6-chloronicotinic acid, two cyclic ureas, olefinic cyclic nitroguanidine, a cyclic guanidine, and its nitroso and nitro derivatives. After 100 days, metabolites each accounted for less than 2% of the radiocarbon label.[42]

- Sorption of imidacloprid to soil generally increases with soil organic matter content.[45,46] However, researchers have demonstrated that sorption tendency also depends on imidacloprid concentration in the soil. Sorption is decreased at high soil concentrations of imidacloprid. As imidacloprid moves away from the area of high concentration, sorption again increases, limiting further movement.[46]
- Imidacloprid's binding to soil also decreases in the presence of dissolved organic carbon in calcareous soil. The mechanism may be through either competition between the dissolved organic carbon and the imidacloprid for sorption sites in the soil or from interactions between imidacloprid and the organic carbon in solution. Such interactions suggest that the potential for imidacloprid to leach into ground water would increase in the presence of dissolved organic carbon.[47]
- Researchers found no imidacloprid residue in soil 10-20 cm under or around sugar beets grown from treated seeds, and concluded that no leaching had occurred.[48]
- Metabolites found in agricultural soils used for growing sugar beets from imidacloprid-treated seed included 6-hydroxynicotinic acid, (1-[(6-chloro-3-pyridinyl)methul]-2-imidazolidone), 6-chloronicotinic acid, with lesser amounts of a fourth compound, 2-imidazolidone.[48]
- In another laboratory study of soil and imidacloprid, researchers determined that half lives varied by both product formulation and soil type. Metabolites were first detected 15 days after imidacloprid was applied.[49]
- Imidacloprid residues became increasingly bound to soil with time, and by the end of the one year test period, up to 40% of the radio-label could not be extracted from the soil samples.[42]
- In a water-sediment system, imidacloprid was degraded by microbes to a guanidine compound. The time to disappearance of one-half of the residues (DT_{50}) was 30-162 days.[42]
- Photodegradation at the surface of a sandy loam soil was rapid at first in a laboratory test, with a measured DT_{50} of 4.7 days, but the rate slowed after that time. Metabolites included 5-hydroxy-imidacloprid, which was the major product, and lesser amounts of an olefin, nitroso derivative, a cyclic urea, and 6-chloronicotinic acid in addition to two unidentified products.[42]

Water

- Imidacloprid is broken down in water by photolysis.[45] Imidacloprid is stable to hydrolysis in acidic or neutral conditions, but hydrolysis increases with increasing alkaline pH and temperature.[50]
- Researchers determined that hydrolysis of imidacloprid produced the metabolite 1-[(6-chloro-3-pridinyl)methyl]-2-imidazolidone.[50] This may be further broken down via oxidative cleavage of the N-C bond between the pyridine and imidazolidine rings, and the resulting compounds may be broken down into $C0_2$, $N0_3^-$, and Cl^-.[45]
- When imidacloprid was added to water at pH 7 and irradiated with a xenon lamp, half of the imidacloprid was photolyzed within 57 minutes.[42] Nine metabolites were identified in the water, of which five were most prominent. These included a cyclic guanidine derivative, a cyclic urea, an olefinic cyclic guanidine, and two fused ring products. These metabolites accounted for 48% of the radio carbon label following two hours of radiation, and the parent compound accounted for 23% of the label.[42]
- Although hydrolysis and photodegradation proceeded along different metabolic pathways in aqueous solution, the main metabolite was imidacloprid-urea in both cases.[45]

- At pH 7, only 1.5% of the initial concentration of 20 mg/L of imidacloprid was lost due to hydrolysis in three months, whereas at pH 9, 20% had been hydrolyzed in samples that were kept in darkness for the same time period.[50]
- The presence of dissolved organic carbon in calcareous soil may decrease the sorption potential of imidacloprid to soil, and thus increase the potential for imidacloprid to leach and contaminate groundwater.[47]
- A total of 28.7% of imidacloprid applied to a 25 cm soil column in the laboratory was recovered in leachate. Formulated products showed greater rates of leaching likely due to the effects of carriers and surfactants. Under natural conditions, soil compaction and rainfall amount may also affect leaching potential.[51]
- Imidacloprid is not expected to volatilize from water.[7]

Air

- Volatilization potential is low due to imidacloprid's low vapor pressure.[7]
- Imidacloprid is metabolized by photodegradation from soil surfaces and water.[42]

Plants

- Imidacloprid applied to soil is taken up by plant roots and translocated throughout the plant tissues.[2] Freshly cut sugar beet leaves contained 1 mg/kg imidacloprid residues up to 80 days following sowing of treated seed although residues were undetectable at harvest 113 days after sowing.[44] In a similar study, sugar beet leaves harvested 21 days after the sowing of treated seeds contained an average of 15.2 µg/g imidacloprid.[52]
- Researchers grew tomato plants in soil treated with 0.333 mg active ingredient per test pot, and monitored the plants and fruits for 75 days. Plants absorbed a total of 7.9% of the imidacloprid over the course of the experiment, although absorption of imidacloprid declined with time since application.[53]
- More than 85% of the imidacloprid taken up by the tomato plants was translocated to the shoots, and only small quantities were found in the roots. Shoot concentrations declined towards the top of the plant. These patterns were also seen in sugar beets grown from treated seed.[52] The tomato fruits also contained imidacloprid, although tissue concentrations were not related to the position of the fruits on the plant.[53]
- Although tomato fruits contained primarily unmetabolized imidacloprid, the plants' leaves also included small quantities of the guanidine metabolite, a tentatively identified olefin metabolite, and an unidentified polar metabolite in addition to the parent compound.[53] However, sugar beets grown from treated seed appeared to rapidly metabolize imidacloprid in the leaves. On day 97 after sowing, the majority of the radio-label was associated with metabolites, not the parent compound.[52]
- Researchers sprayed imidacloprid on eggplant, cabbage, and mustard crops at rates of 20 and 40 g/ha when the crops were at 50% fruit formation, curd formation, and pod formation, respectively.[54] The researchers calculated foliar half-lives of 3 to 5 days based on the measured residues.[54]
- Metabolites detected in the eggplant, cabbage, and mustard plants included the urea derivative [1-(6-chloropyridin-3-ylmethil)imidazolidin-2-one] and 6-chloronicotinic acid 10 days after foliar application. Residues of 2.15-3.34 µg/g were detected in the eggplant fruit.[54]
- Three plant metabolites of imidacloprid, the imidazolidine derivative, the olefin metabolite and the nitroso-derivative, were more toxic to aphids than imidacloprid itself.[55]

Indoor

- No information regarding indoor half-life or residues was found for imidacloprid.

- Researchers measured residue transfer of a commercial spot-on product containing imidacloprid on dogs' fur to people. Gloves worn to pat the dogs contained an average of 254 ppm of imidacloprid 24 hours following application of the product. Residues from the fur declined to an average of 4.96 ppm by the end of the first week.[56]

Food Residue

- The United States Department of Agriculture (USDA) Pesticide Data Program monitored imidacloprid residues in food and published their findings in 2006. Imidacloprid was detected in a range of fresh and processed fruits and vegetables. It was detected in over 80% of all bananas tested, 76% of cauliflower, and 72% of spinach samples. In all cases, however, the levels detected were below the U.S. EPA's tolerance levels. Imidacloprid was also found in 17.5 % of applesauce and 0.9% raisin samples, although percentage of detections were greater in the fresh unprocessed fruit (26.6% of apples sampled, and 18.1% of grapes sampled).[57]

- Imidacloprid was not one of the compounds sampled for the 2006 Food and Drug Administration (FDA) Center for Food Safety and Applied Nutrition's Pesticide Residue Monitoring Program.[57]

Ecotoxicity Studies:

Birds

- The acute LD_{50} for birds varies by species; it was determined to be 31 mg/kg in Japanese quail but 152 mg/kg in bobwhite quail. However, dietary LC_{50} values for a five-day interval were 2225 mg/kg/day for bobwhite quail and in excess of 5000 mg/kg for mallard ducks.[2]

Fish and Aquatic Life

- LC_{50} values for a 96-hour exposure were 237 mg/L for golden orfe (*Leuciscus idus*) and 21 mg/L for rainbow trout (*Oncorhyncus mykiss*).[2]

- Researchers determined LC_{50} values of 85 mg/L for *Daphnia* with a 48-hour exposure. A concentration of greater than 100 mg/L for 72 hours was required to reduce the growth rate of the alga *Pseudokirchneriella subcapitata* by 50%.[2]

- The EC_{50} of imidacloprid for *Daphnia magna* was 96.65 mg/L. However, the EC_{50} declined to 90.68 mg/L when predator cues were added to the water as an additional stress. Sublethal exposures reduced feeding and increased respiration rates in *Daphnia*. Exposed *Daphnia* did not respond to predator cues as quickly as did control animals, and failed to mature as quickly or produce as many young. These changes led to reduced population growth rate following exposure.[58] See the text box on **EC_{50}**.

EC_{50}: The median effective concentration (EC_{50}) may be reported for sublethal or ambiguously lethal effects. This measure is used in tests involving species such as aquatic invertebrates where death may be difficult to determine. This term is also used if sublethal events are being monitored.

Newman, M.C.; Unger, M.A. *Fundamentals of Ecotoxicology*; CRC Press, LLC.: Boca Raton, FL, 2003; p 178.

Terrestrial Invertebrates

- Oral LD_{50} values for bees range from 3.7 to 40.9 ng per bee, and contact toxicity values ranged from 59.7 to 242.6 ng per bee.[59] Based on these values, imidacloprid is considered to be highly toxic to bees.[18] Colonies of bees (*Apis mellifera*) appeared to vary in their sensitivity to imidacloprid, perhaps due to differences in oxidative metabolism among colonies. The 5-hydroxyimidacloprid and olefin metabolites were more toxic to honeybees than the parent compound.[60]

- Bees were offered sugar solution spiked with imidacloprid at nominal concentrations of 1.5, 3.0, 6.0, 12.0, 24.0, 48.0, or 96.0 µg/kg for 14 days. The experiment was repeated with bees that matured in July (summer bees) and between December and February (winter bees). Summer bees died at greater rates than controls in the 96 µg/kg treatment, whereas winter bees demonstrated increased mortality at 48 µg/kg. Reflex responses of summer bees decreased at 48 µg/kg, whereas the reflex responses of winter bees were unaffected. Learning responses in summer bees were decreased following exposures of 12 µg/kg imidacloprid, and winter bees demonstrated reduced learning responses at doses of 48 µg/kg.[61]

- Surveys of pollen collected by bees from five locations in France revealed detectable residues of imidacloprid or its metabolite 6-chloronicotinic acid in 69% of the samples. Maximum detected concentrations were 5.7 µg/kg and 9.3 µg/kg for imidacloprid and the metabolite, respectively.[62]
- Researchers performed 10-day chronic exposure tests on honeybees and found that mortality increased over controls at doses as low as 0.1 µg/L of imidacloprid and six metabolites.[60]
- Researchers fed bumblebees (*Bombus terrestris*) nectar and pollen spiked with either 10 µg/kg or 25 µg/kg imidacloprid in syrup and 6 µg/kg or 16 µg/kg in pollen. Worker survival rates declined by 10% in both treatment groups and brood production was reduced in the low-dose group.[63]
- Researchers grew sunflowers from seeds treated with 0.7 mg imidacloprid per seed and found imidacloprid residue in nectar (1.9 ±1 ppt) and pollen (3.3 ± 1 ppt). No metabolites were found in nectar or pollen. They also grew sunflowers from untreated seeds in soil with imidacloprid residues at concentrations up to 15.7 ppt. In that test, neither imidacloprid nor its metabolites were found in nectar or pollen.[59]
- Researchers have found that bees avoided feeding on a sugar solution spiked with imidacloprid at 24 µg/kg concentrations, and that this avoidance appeared to be due to a repellent or antifeedant effect.[64]
- The predatory insect *Hippodamia undecimnotata* experienced reduced survival, delayed and reduced egg production, reduced longevity, and reduced population growth rate following exposure to aphids raised on potted bean plants which had been treated 10 days earlier with imidacloprid applied at 0.0206 mg active ingredient per pot or 1/14 the label rate.[65]
- Adult green lacewings (*Chrysoperla carnea*) exhibited reduced survival rates after feeding on the nectar of greenhouse plants that had been treated with granules of a commercial product containing 1% imidacloprid. Treatments were done with imidacloprid-containing products mixed at label rates and at twice the label rate three weeks prior to the experiment. Insects fed on the treated plants even when untreated plants were present.[66]
- The LC_{50} for the earthworm *Eisenia foetida* was determined to be 10.7 mg/kg in dry soil.[2] In a separate study, two earthworm species (*Aporrectodea nocturna* and *Allolobophoria icterica*) were placed in soil cores treated with 0.1 or 0.5 mg/kg imidacloprid. At the highest dose, both species of worms produced shorter burrows. *A. nocturna* also produced fewer surface casts at the highest dose, and gas diffusion through the soil cores was reduced by approximately 40% compared to controls.[67]

Regulatory Guidelines:

- The reference dose (RfD) is 0.057 mg/kg/day.[31] See the text box on **Reference Dose (RfD)**.
- The U.S. EPA has classified imidacloprid into Group E, no evidence of carcinogenicity, based on studies with rats and mice.[20,31] See the text box on **Cancer** (page 5).
- The acute Population Adjusted Dose (aPAD) is 0.14 mg/kg.[31]
- The chronic Population Adjusted Dose (cPAD) is 0.019 mg/kg/day.[31]

Reference Dose (RfD): The RfD is an estimate of the quantity of chemical that a person could be exposed to every day for the rest of their life with no appreciable risk of adverse health effects. The reference dose is typically measured in milligrams (mg) of chemical per kilogram (kg) of body weight per day.

U.S. Environmental Protection Agency. Office of Water. 2002 Edition of the Drinking Water Standards and Health Advisories. EPA 822-R-02-038. http://www.epa.gov/ost/drinking/standards/dwstandards.pdf

Date Reviewed: April 2010

Please cite as: Gervais, J. A.; Luukinen, B.; Buhl, K.; Stone, D. 2010. *Imidacloprid Technical Fact Sheet*; National Pesticide Information Center, Oregon State University Extension Services. http://npic.orst.edu/factsheets/imidacloprid.pdf.

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