

Environmental Fate of Imidacloprid

Revised by Matthew Fossen, Ph.D.
Environmental Monitoring
Department of Pesticide Regulation
1001 I Street
Sacramento, CA 95812-4015
April 2006

Summary

Imidacloprid is a systemic chloronicotinyl insecticide that enters the target pest via ingestion or direct contact. It acts by disrupting nicotinic acetylcholine receptors in the insect central nervous system. Imidacloprid is used for controlling sucking insects, soil insects, termites, and some chewing insects. It is applied to seeds, soil, crops, and structures, and is used as a topical flea control treatment on domestic pets.

The low vapor pressure of 1.0×10^{-7} mmHg indicates that this insecticide is nonvolatile. In addition, the low Henry's law constant of 6.5×10^{-11} atm m³/mole, indicates that it has low volatility from water. Therefore, it is unlikely to be dispersed in air over a large area from volatilization. There is a possibility for drift when imidacloprid-treated plant materials are dispersed by planting equipment.

The hydrolysis half-life of imidacloprid can range from 33–44 days at pH 7 and 25°C. The aqueous photolysis half-life is less than 3 hours. Imidacloprid has a photolysis half-life of 39 days at the soil surface, with a range of 26.5–229 days when incorporated into the soil. Persistence in soil allows for continual availability for uptake by plant roots. The combination of low K_{oc} between 132–310 and high water solubility of 514 ppm suggests a potential to leach to ground water. Imidacloprid detections have been reported in both ground water and surface water in New York. One surface water detection has been reported in Florida.

Imidacloprid is rapidly moved through plant tissues after applications, and can be present in detectable concentrations in tissues such as leaves, vascular fluids, and pollen. Many non-target beneficial arthropods such as honeybees, parasitic wasps, and predaceous ground beetles are sensitive to imidacloprid. These organisms may be adversely affected by sublethal doses of the insecticide, but the effects vary widely depending on application method and route of intake. There is a potential for stress-related sublethal effects on fish in water contaminated with imidacloprid. Since several imidacloprid metabolites have been shown to be equal or greater in toxicity than the parent compound, their presence in the environment should be studied and thus they should be included in chemical analyses of future environmental studies.

Introduction

This document reviews all routes of environmental fate for imidacloprid under field conditions and potential effects on biota. Imidacloprid is a chloronicotinyl nitroguanidine insecticide, with

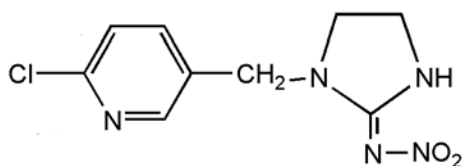
the IUPAC name 1-[(6-chloropyridin-3-yl) methyl]-N-nitro-4, 5-dihydroimidazol-2-amine and a CAS Number of 105827-78-9. The technical product is a colorless, odorless crystal. Imidacloprid is a General Use Pesticide, classified by the EPA as a class II/III agent.

As of March 2006, there were 115 active registered products containing imidacloprid, registered in California under a wide variety of trade names, e.g. Admire, Advantage, Gaucho, Merit, Premise, Provado, and Touchstone (California Department of Pesticide Regulation, 2006). Imidacloprid is a systemic pesticide with physical/chemical properties that allow residues to move into treated plants and then throughout the plant via xylem transport and translaminar (between leaf surfaces) movement (Buchholz and Nauen, 2002). Residues of the insecticide then enter the target pest by ingestion or direct contact, disrupting the insect's nervous system by binding to postsynaptic nicotinic acetylcholine receptors. The disruption of the nervous systems results in modified feeding behavior, paralysis, and subsequent death of the insect (Mullins, 1993).

Imidacloprid controls sucking insects, soil insects, termites, and some chewing insects, and is effective against all feeding stages. It is used to treat seeds, soil, crops and structures, and is a flea control treatment on domestic pets (Meister, 2000). There were 131,394 pounds of imidacloprid active ingredient applied in California in 2004 with the highest statewide commodity use for structural pest control (39,538 pounds). The county reporting the highest total use was Monterey (15,322 pounds; California Department of Pesticide Regulation, 2006b, 2006c).

Chemical Properties

Molecular Structure:



Chemical Formula: C₉H₁₀ClN₅O₂

Table 1. Physical and chemical properties of imidacloprid. All data were submitted in approved studies and obtained from the Pesticide Chemistry Database (California Department of Pesticide Regulation, internal database).

Molecular weight	255.7
Water solubility	514 mg/L (20°C at pH 7)
Vapor pressure	1.00 x 10 ⁻⁷ mmHg (20°C)
Hydrolysis half-life	>30 days (25°C at pH 7)
Aqueous photolysis half-life	<1 hour (24°C at pH 7)
Anaerobic half-life	27.1 days
Aerobic half-life	997 days
Soil photolysis half-life	38.9 days
Field dissipation half-life	26.5 – 229 days
Henry's constant	6.5 x 10 ⁻¹¹ atm m ³ /mole (20°C)
Octanol-water coefficient (K _{ow})	3.7
Soil adsorption coefficient:	
K _d	0.956–4.18
K _{oc}	132–310

Table 2. Imidacloprid toxicity. All toxicity data were submitted in approved studies and obtained from the EXTOXNET database (Oregon State University, 1998).

<i>Daphnia magna</i> LC ₅₀ (48 h)	85 mg/L
Mysid shrimp LC ₅₀ (96 h)	37 mg/L
Rainbow trout LC ₅₀ (96 h)	211 mg/L
Bobwhite quail LD ₅₀	152 mg/kg
Mallard duck LD ₅₀	283 mg/kg
Rat acute oral LD ₅₀	450 mg/kg
Honeybee LD ₅₀ (48 h)	0.008 µg/bee

Water Quality Standards:

U.S. EPA has not established maximum contaminant levels (MCLs) for dietary exposure to imidacloprid through drinking water (U.S. Environmental Protection Agency, 2004). However, in order to conduct an aggregate dietary exposure analysis during registration, the U.S. EPA has developed drinking water levels of comparison (DWLOCs), which determine the theoretical upper limits for a pesticide's concentration in drinking water. DWLOCs are measured in parts-per-billion (ppb).

Table 3. Imidacloprid DWLOCs. Obtained from U.S. Environmental Protection Agency (2005).

Acute exposure	3625 ppb
Chronic exposure	1775 ppb

Environmental Fate

Air: Some formulations of imidacloprid are applied as sprays, allowing for possible offsite movement through drift. The vapor pressure of 1.0 x 10⁻⁷ mmHg indicates that this insecticide is

not volatile. Since imidacloprid has a low soil adsorption coefficient, it has a low potential to be dispersed in air over a large area via air-borne soil particles. The low Henry's law constant of 6.5×10^{-11} atm m³/mole also indicates that it has low potential of volatilizing from water. This combination of physical characteristics makes it unlikely that imidacloprid will be present in the air in detectable amounts following application by any method.

Clark et al. (1998) explored the possibility for imidacloprid to contaminate the air via cigarette smoke. In their study, cigarette smoke made from tobacco that was treated with radiolabelled imidacloprid was analyzed for the presence of residues. The majority of the radioactivity (34.6 percent) was recovered from carbon dioxide; 6.1 percent of the recovered radioactivity was contributed by imidacloprid and imidacloprid urea. It is unlikely that cigarette smoke is a significant source of imidacloprid exposure.

There is evidence that imidacloprid residues can drift off-site on plant debris. Greatti et al. (2003) detected imidacloprid residues on plants growing adjacent to a field sown with seed-treated corn. Plant samples were analyzed using gas chromatography and found to contain imidacloprid and imidacloprid degradates at concentrations ranging from 14–54 ppb. The imidacloprid-treated seeds were sown using a pneumatic corn seed drill, so it is likely that seed debris was lost through the fan exhaust system.

Air monitoring has been conducted in California concurrent with imidacloprid applications for control of glassy-winged sharpshooter (*Homalodisca coagulata*). Air samples collected by the California Department of Pesticide Regulation immediately following imidacloprid foliar spray applications in Butte, Imperial, Santa Clara, and Solano counties did not detect any residues of the insecticide (Walters et al., 2001; Segawa and Walters, 2002; Segawa et al., 2002; Segawa et al., 2004).

Soil: The high water solubility and low K_{oc} for imidacloprid indicates a low tendency for adsorption to soil particles. Field studies have produced a wide range in half-life values ($t^{1/2}$) from 27 to 229 days (Miles, Inc., 1992; Mobay Chemical Corp., 1992). Scholz et al. (1992) found that imidacloprid degradation was more rapid in soils with cover crops than in bare soils, with a $t^{1/2}$ of 48 and 190 days, respectively. Degradation on soil via photolysis has a $t^{1/2}$ of 39 days. The half-life of imidacloprid in the soil tends to increase as soil pH increases (Sarkar et al., 2001). In the absence of light, the longest half-life of imidacloprid was 229 days in field studies and 997 days in laboratory studies (Miles, Inc., 1992; Mobay Chemical Corp., 1992). This persistence in soil in the absence of light makes imidacloprid suitable for seed treatment and incorporated soil application because it allows continual availability for uptake by roots (Mullins, 1993). Thus, imidacloprid can persist in soil depending on soil type, pH, use of organic fertilizers, and presence or absence of ground cover.

The primary imidacloprid breakdown products in soil (as reported by Rouchaud et al., 1996) are as follows:

- imidacloprid urea
- 6-hydroxynicotinic acid
- 6-chloronicotinic acid

CO₂ is then formed from 6-chloronicotinic acid (Scholz, 1992).

Soil adsorption and half-life estimates have been shown to be dependent on soil properties. Field and laboratory studies have determined that imidacloprid adsorption to soil particles increases as the concentration of the insecticide decreases (Cox et al., 1998; Oi, 1999; Kamble and Saran, 2005). The sorption level of imidacloprid is also affected by soil properties such as organic carbon and minerals. As the organic carbon levels and laminar silicate clay content in the soil increase, the potential for imidacloprid to leach would decrease (Cox et al., 1997, 1998b, 1998c). Organic fertilizers, such as chicken and cow manure, increased the pesticide adsorption to the organic matter and increased its half-life. Half-lives ranged from 40 days when no organic fertilizers were used to 124 days when cow manure was used. However, residual insecticide soil concentrations were low at the time of harvest, similar to those not treated with organic fertilizers; no correlation was found between K_{oc} and the soil carbon content (Rouchaud et al., 1996b). In contrast, imidacloprid adsorption in a calcereous soil was found to decrease with the addition of organic carbon (obtained from peat and tannic acid) (Flores-Céspedes et al., 2002); this could increase the mobility and leaching potential of imidacloprid. The presence of conflicting studies seems to indicate that the fate of imidacloprid in the soil is highly sensitive to soil composition and sources of organic carbon.

Surface Water: Imidacloprid breakdown in surface waters is primarily due to aqueous photolysis. The primary degradation products resulting from aqueous photolysis (as reported by Moza et al., 1998) are as follows:

- imidacloprid urea
- 6-chloronicotinic aldehyde
- 6-chloro-N-methylnicotinacidamide
- 6-chloro-3-pyridyl-methylethylenediamine

Due to the short half-life of imidacloprid in water when exposed to sunlight (less than three hours) (Moza et al., 1998; Wamhoff et al., 1999), it is unlikely that the parent compound will be detected in surface waters. There have not been any detections of imidacloprid in California surface waters as of January 2004 (California Department of Pesticide Regulation, 2004). However, imidacloprid has been detected in surface water surveys conducted in Florida (Pfeuffer and Matson, 2001) and New York (Phillips and Bode, 2002).

Table 4. Summary of imidacloprid detections in surface water. Obtained from Pfeuffer and Matson (2001) and Phillips and Bode (2002).

	Florida	New York
Number of detections	1	2
Total number of sites	38	47
Maximum concentration	1.0 ppb	0.2 ppb
Minimum concentration		0.07 ppb

In the absence of large numbers of imidacloprid detections, the U.S. EPA has used the FQPA Index Reservoir Screening Tool (FIRST) and Screening Concentration In Ground Water (SCI-GROW) models to calculate estimated environmental concentrations (EECs). These values are compared with the DWLOCs to determine exposure risks. For surface water, the EECs are as follows:

Table 5. Estimated environmental concentrations of imidacloprid in surface waters. Obtained from U.S. Environmental Protection Agency (2004).

Acute surface water exposure	36.04 ppb
Chronic surface water exposure	17.24 ppb

Ground Water: The hydrolytic half-life of this pesticide is greater than 30 days at pH 7 and 25°C. Sarkar et al. (1999) reported that the hydrolytic half-life varies from 33–44 days at the same pH and temperature, while Zheng et al. (1999) observed less than 1.5 percent hydrolysis in neutral water after three months. Imidacloprid was found to hydrolyze more rapidly in alkaline water, with a half-life of 20.0 days at pH 10.8 and 2.85 days at pH 11.8 (Zheng et al., 1999). In wettable powder formulations, hydrolysis half-lives increased by three to six days compared to liquid formulations (Sarkar et al., 1999). Gupta et al. (2002) found that imidacloprid leaching in soil column studies varied considerably with formulation whereby wettable powder formulations were the worst leachers, followed by soluble concentrates and suspension concentrates. The primary hydrolytic degradate of imidacloprid is imidacloprid urea (Miles, Inc., 1993; Zheng et al., 1999).

The leaching potential of imidacloprid when applied via chemigation was explored by Felsot et al. (1998). In this study, imidacloprid was applied to a fine sandy loam soil through a subsurface drip system installed in an experimental hops field. After seven days of irrigation applied at a depth of 0.38 cm of water per day, the insecticide was detected at the maximum sampled soil depth of 105 cm at concentrations as high as 120 ppb. Although the amount of irrigation water applied was not matched to local evapotranspiration and may have resulted in saturated soil, the study demonstrated the potential for imidacloprid residues to move downward through the soil with percolating water. Júnior et al. (2004) discovered that preferential transport through macropores might facilitate downward imidacloprid movement through heavy clay soils that are not normally considered conducive to leaching.

Several studies have provided evidence that imidacloprid does not leach through the soil profile into ground water (Miles, Inc., 1992; Rouchaud et al., 1994). The field sites used by Rouchaud et al. had silty loam and silt soils with high organic matter content (2.16% and 2.4–4.3%, respectively), which may have reduced the mobility of the pesticide; no residues were found beyond a depth of 10 cm. The Miles, Inc. field site was a California sandy loam soil with low organic matter content (0.3–1.0%) in which the applied imidacloprid did not move beyond a soil depth of 12 inches. The field sites in both studies received enough irrigation/precipitation to allow percolation through the soil: 60 inches over one year in the 1992 study and 10 inches over three months in the 1994 study. The Department of Pesticide Regulation placed imidacloprid on the Ground Water Protection List (Title 3, California Code of Regulations, section 6800(b)) due to its potential to contaminate ground water. A sampling of 33 wells in high-use areas in California in 2003–2004 did not detect imidacloprid or imidacloprid degradates (guanidine, urea, and guanidine-olefin) (Schuette et al., 2004), which helps validate the findings reported by Miles, Inc.

Despite these findings, there have been numerous detections of imidacloprid in ground water. A ground water monitoring project conducted by Bayer Corporation (1998) on Long Island, NY,

detected imidacloprid in a single agricultural well at concentrations ranging from less than 0.1 ppb to 1.0 ppb over a five month sampling period; the well was located in a sandy loam soil with a water table at 18 feet. Ground water monitoring data submitted to U.S. EPA by the New York State Department of Environmental Conservation, Division of Solid and Hazardous Materials (U.S. Environmental Protection Agency, 2003) contained several imidacloprid detections in agricultural, homeowner, and golf course wells located in highly vulnerable soils.

Table 6. Summary of imidacloprid detections in ground water in New York. Obtained from U.S. Environmental Protection Agency (2003).

Number of detections	≈20
Total number of sites	≈2000
Maximum concentration	6.69 ppb
Minimum concentration	0.1 ppb

Ground water EECs are presented in Table 7. It should be noted that although the maximum detected concentration of imidacloprid in ground water exceeds both the acute and chronic EECs, it is far below the relevant DWLOC and therefore presents little risk of dietary exposure.

Table 7. Estimated environmental concentrations of imidacloprid in ground water. Obtained from U.S. Environmental Protection Agency (2004).

Acute ground water exposure	2.09 ppb
Chronic ground water exposure	2.09 ppb

Biota: Imidacloprid is readily translocated through plant tissues following direct contact. When used as a seed treatment, the insecticide is absorbed by the seedling from the disintegrating seed coat. In a French study, sunflower plants that were seed-treated at a rate of 1.0 mg/seed produced pollen that contained imidacloprid at a concentration of 13.0 ppb (Laurent and Rathahao, 2003). Detections in corn plants that were seed-treated at a rate of 0.7 mg/seed ranged from an average of 2.1 ppb in pollen to 6.6 ppb in the flowers (Bonmatin et al., 2005). A study conducted by Westwood et al. (1998) found that the leaves of sugar beet seedlings contained an average of 15.2 ppm three weeks after treatment at a rate of 0.9 mg/seed; similar findings were reported by Rouchaud et al. (1994).

The main breakdown products of imidacloprid in plants are (Miles, Inc., 1993b):

- a monohydroxy metabolite
- imidacloprid guanidine
- imidacloprid olefin
- a monoglucoside of 6-chloropicolyl alcohol

Imidacloprid is highly toxic to honeybees (*Apis mellifera*), with a reported LD₅₀ of 8 ng/bee. Actual toxicity varies widely depending on honeybee subspecies and type of exposure. Acute oral toxicity LD₅₀ values for both *Apis mellifera mellifera* and *Apis mellifera caucasica* are approximately 5 ng/bee, while contact LD₅₀ values are 14 ng/bee for *A. m. caucasica* and 24 ng/bee for *A. m. mellifera* (Suchail et al., 1999). Imidacloprid is toxic in smaller doses when ingested over an extended period: chronic LD₅₀ values range from 0.01–1.0 ng/bee (Suchail et al., 2001). Low doses of imidacloprid and imidacloprid metabolites have been found to

negatively affect honeybee foraging and learning behavior (Decourtye et al., 2003, 2004). Bumblebees (*Bombus terrestris*) showed no adverse effects from foraging on sunflowers that were seed-treated at a standard rate of 0.7 mg/seed where imidacloprid residues were detected in the nectar of treated plants at concentrations less than 10 ppb (Tasei et al., 2001).

Significant adverse effects of imidacloprid have been reported on non-target beneficial invertebrates. Kunkel et al. (2001) found that the carabid beetle *Harpalus pennsylvanicus* exhibited sublethal intoxication when exposed to imidacloprid-treated turfgrass, resulting in increased mortality due to ant predation. These effects were lessened by post-treatment irrigation. Carabid beetles feeding on corn seedlings that were seed-treated with imidacloprid at a rate of 0.16 mg/seed suffered nearly 100 percent mortality (Mullin et al., 2005). Predatory minute pirate bug (*Orius* spp.) populations were significantly reduced in field sown with corn that was seed-treated with imidacloprid at a rate of 4.9 g/kg seed (Albajes et al., 2003). Imidacloprid is highly toxic to the convergent lady beetle, *Hippodamia convergens* (Kaakeh et al., 1996; Stark et al., 1995). In mortality studies conducted on the aphid parasitoid *Aphidius gifuensis*, imidacloprid applied at field rates to female adults and pupae caused 71 and 67 percent mortality, respectively (Kobori and Amano, 2004).

The majority of toxicity studies have focused on the parent compound, imidacloprid. It should be noted that two imidacloprid derivatives (olefin and nitrosimine) occur as metabolites in treated plants and have greater insecticidal activity than the parent compound (Nauen et al., 1998). The guanidine metabolite of imidacloprid does not possess insecticidal properties, but has a higher mammalian toxicity than the parent compound (Tomizawa and Casida, 1999).

Due to a high LD₅₀ for rainbow trout (211 mg/L), imidacloprid is not considered acutely toxic to fish. However, there exists a possibility for detrimental sublethal effects at lower doses. Japanese medaka fish (*Oryzias latipes*) in an imidacloprid-treated rice paddy were highly stressed relative to control treatments and suffered severe ectoparasite (*Cychochaeta domerguei*) infestations (Sánchez-Bayo and Goka, 2005).

References

- Albajes, R., C. López, and X. Pons. 2003. Predatory fauna in cornfields and response to imidacloprid seed treatment. *J. Econ. Entomol.* 96(6): 1805–1813.
- Bayer Corp. 1998. Imidacloprid memo to U.S. EPA. June 5, 1998.
- Bonmatin, J.M., P.A. Marchand, R. Charvet, I. Moineua, E.R. Bengsch, and M.E. Colin. 2005. Quantification of imidacloprid uptake in maize crops. *J. Agric. Food Chem.* 53: 5336–5341.
- Buchholz, A. and R. Nauen. 2002. Translocation and translaminar bioavailability of two neonicotinoid insecticides after foliar application to cabbage and cotton. *Pest Manag. Sci.* 58(1): 10–16.
- California Department of Pesticide Regulation. 2004. Surface Water Database. [Online]. Available at <http://www.cdpr.ca.gov/docs/sw/surfddata.htm>. (Verified 09 January 2006). CDPR, Sacramento, CA.

- California Department of Pesticide Regulation. 2006. Pesticide Use Reporting (PUR). [Online]. Available at <http://www.cdpr.ca.gov/docs/pur/purmain.htm>. (Verified 09 January 2006). CDPR, Sacramento, CA.
- California Department of Pesticide Regulation. 2006b. California Product/Label Database Queries & Lists. [Online]. Available at <http://www.cdpr.ca.gov/docs/label/labelque.htm>. (Verified 24 January 2006). CDPR, Sacramento, CA.
- California Department of Pesticide Regulation. 2006c. Summary of pesticide use report data 2004 indexed by chemical. [Online]. Available at <http://www.cdpr.ca.gov/docs/pur/pur04rep/chmrpt04.pdf>. (Verified 25 January 2006). CDPR, Sacramento, CA.
- Clark, T., E. Kaußmann, E. Römer, and G. Schepers. 1998. The fate of imidacloprid in tobacco smoke of cigarettes made from imidacloprid-treated tobacco. *Pestic. Sci.* 52: 119–125.
- Cox, L., W. Koskinen, and P. Yen. 1997. Sorption-desorption of imidacloprid and its metabolites in soils. *J. Agric. Food Chem.* 45(4): 1468–1472.
- Cox, L., W. Koskinen, and P. Yen. 1998. Changes in sorption of imidacloprid with incubation time. *Soil Sci. Soc. Am. J.* 62(2): 342–347.
- Cox, L., W. Koskinen, R. Celis, P. Yen, M. Hermosin, and J. Cornejo. 1998b. Sorption of imidacloprid on soil clay mineral and organic components. *Soil Sci. Soc. Am. J.* 62(4): 911–915.
- Cox, L., W. Koskinen, and P. Yen. 1998c. Influence of soil properties on sorption-desorption of imidacloprid. *J. Environ. Sci. Health.* B33(2): 123–134.
- Decourtye, A., E. Lacassie, and M.H. Pham-Delègue. 2003. Learning performances of honeybees (*Apis mellifera* L.) are differentially affected by imidacloprid according to the season. *Pest Manag. Sci.* 59: 269–278.
- Decourtye, A., J. Devillers, S. Cluzeau, M. Charreton, and M.H. Pham-Delègue. 2004. Effects of imidacloprid and deltamethrin on associative learning in honeybees under semi-field and laboratory conditions. *Ecotoxicol. Environ. Safety* 57: 410–419.
- Extension Toxicology Network (EXTOXNET). 1998. Imidacloprid Pesticide Information Profile. [Online]. Available at <http://extoxnet.orst.edu/pips/imidaclo.htm>. (Verified 09 January 2006). Oregon State University, Corvallis, OR.
- Felsot, A.S., W. Cone, J. Yu, and J.R. Ruppert. 1998. Distribution of imidacloprid in soil following subsurface drip chemigation. *Bull. Environ. Contam. Toxicol.* 60: 363–370.
- Flores-Céspedes, F., E. González-Pradas, M. Fernández-Pérez, M. Villafranca-Sánchez, M. Socías-Viciano, and M.D. Ureña-Amate. 2002. Effects of dissolved organic carbon on sorption and mobility of imidacloprid in soil. *J. Environ. Qual.* 31: 880–888.
- Gupta, S., V.T. Gajbhiye, and N.P. Agnihotri. 2002. Leaching behavior of imidacloprid formulations in soil. *Bull. Environ. Contam. Toxicol.* 68: 502–508.
- Greatti, M., A.G. Sabatini, R. Barbattini, S. Rossi, and A. Stravisi. 2003. Risk of environmental contamination by the active ingredient imidacloprid used for corn seed dressing: Preliminary results. *Bull. Insectol.* 56(1): 69–72.

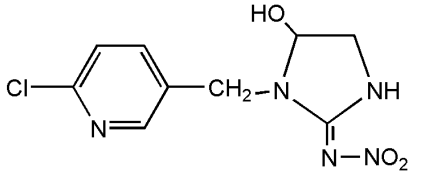
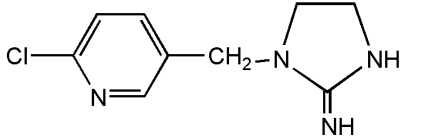
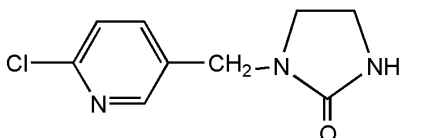
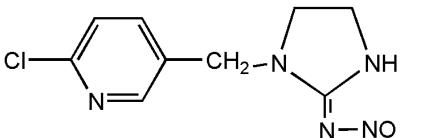
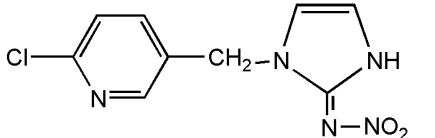
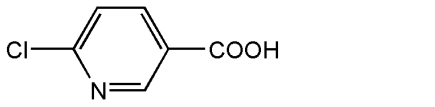
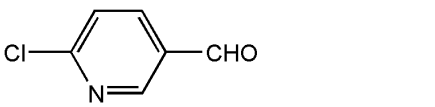
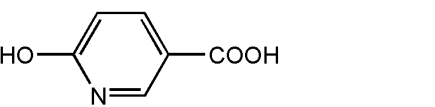
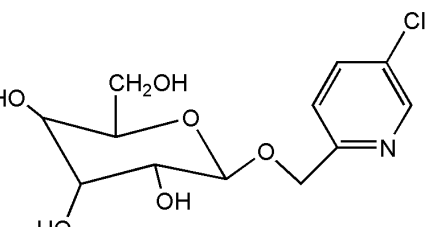
- Júnior, R.P.S., J.H. Smelt, J.J.T.I. Boesten, R.F.A. Hendriks, and S.E.A.T.M. van der Zee. 2004. Preferential flow of bromide, bentazon, and imidacloprid in a dutch clay soil. *J. Environ. Qual.* 33: 1473–1486.
- Kaakeh, N., W. Kaakeh, and G.W. Bennett. 1996. Topical toxicity of imidacloprid, fipronil, and seven conventional insecticides to the adult convergent lady beetle (Coleoptera: Coccinellidae). *J. Entomol. Sci.* 31: 315–322.
- Kamble, S.T. and R.K. Saran. 2005. Effect of concentration on adsorption of three termiticides in soil. *Bull. Environ. Contam. Toxicol.* 75: 1077–1085.
- Kobori, Y. and H. Amano. 2004. Effects of agrochemicals on life-history parameters of *Aphidius gifuensis* Ashmead (Hymenoptera: Braconidae). *Appl. Entomol. Zool.* 39(2): 255–261.
- Koester J. 1992. Comparative metabolism of (pyridinyl-¹⁴C-methyl)-imidacloprid in plant cell suspension cultures. *Proc. Brighton Crop Protection Conference – Pests and Dis.* 2: 901–906
- Krohn, J. and E. Hellpointner. 2002. Environmental fate of imidacloprid. *Pflanzenschutz-Nachrichten Bayer* 55: 1–26.
- Laurent, F.M. and E. Rathahao. 2003. Distribution of [¹⁴C]imidacloprid in sunflowers (*Helianthus annuus* L.) following seed treatment. *J. Agric. Food Chem.* 51: 8005–8010.
- Meister, R.T. 2000. *Farm Chemical Handbook 86*. Meister Publishing Company, Willoughby, OH.
- Miles, Inc. 1992. Premise termiticide - Environmental fate: Terrestrial field dissipation for California site. Volume No. 51950–0032. Department of Pesticide Regulation, Sacramento, CA.
- Miles, Inc. 1993. NTN 33893 (imidacloprid) - Environmental fate summary. Volume No. 51950–0113. Department of Pesticide Regulation, Sacramento, CA.
- Miles, Inc. 1993b. Imidacloprid (syn. PREMISE, NTN 33893) - Comparative metabolism in plant cell suspension cultures. Volume No. 51950–0078. Department of Pesticide Regulation, Sacramento, CA.
- Mobay Chemical Corp. 1992. Premise termiticide - Environmental fate: Hydrolysis; Aqueous and soil photolysis. Volume No. 51950–0027. Department of Pesticide Regulation, Sacramento, CA.
- Moza, P.N., K. Hustert, E. Feicht, and A. Kettrup. 1998. Photolysis of imidacloprid in aqueous solution. *Chemosphere.* 36(3): 497–502.
- Mullin, C.A., M.C. Saunders II, T.W. Leslie, D.J. Biddinger, and S.J. Fleischer. 2005. Toxic and behavioral effects to Carabidae of seed treatments used on Cry3Bb1- and Cry1Ab/c-protected corn. *Environ. Entomol.* 34(6): 1626–1636.
- Mullins, J. W. 1993. Imidacloprid: A new nitroguanidine insecticide. *Am. Chem. Soc. Symposium Series* 5240097–6156.
- Nauen, R., K. Tietjen, K. Wagner, and A. Elbert. 1998. Efficacy of plant metabolites of imidacloprid against *Myzus persicae* and *Aphis gossypii* (Homoptera: Aphididae). *Pestic. Sci.* 52: 53–57.

- Oi, M. 1999. Time-dependent sorption of imidacloprid in two different soils. *J. Agric. Food Chem.* 47: 327–332.
- Pfeuffer, R.J. and F. Matson. 2001. Pesticide surface water quality report, March 2001 sampling event. [Online]. Available at <http://www.sfwmd.gov/curre/pest/P0103rpt.pdf>. (Verified 17 January 2006). South Florida Water Management District, West Palm Beach, FL.
- Phillips, P.J. and R. W. Bode. 2002. Concentrations of pesticides and pesticide degradates in the Croton River Watershed in Southeastern New York, July-September 2000. [Online]. Available at <http://ny.water.usgs.gov/pubs/wri/wri024063/wrir02-4063.pdf>. (Verified 12 January 2006). USGS-NY, Troy, NY.
- Rouchaud, J., F. Gustin, and A. Wauters. 1994. Soil biodegradation and leaf transfer of insecticide imidacloprid applied in seed dressing in sugar beet crops. *Bull. Environ. Contam. Toxicol.* 53(3): 344–350.
- Rouchaud, J., F. Gustin, and A. Wauters. 1996. Imidacloprid insecticide soil metabolism in sugar beet field crops. *Bull. Environ. Contam. Toxicol.* 56: 29–36.
- Rouchaud, J., A. Thirion, A. Wauters, F. Van de Steene, F. Benoit, N. Ceustermans, J. Gillet, S. Marchand, and L. Vanparys. 1996b. Effects of fertilizer on insecticide adsorption and biodegradation in crop soils. *Arch. Environ. Contam. Toxicol.* 31(1): 98–106.
- Sánchez-Bayo, F. and K. Goka. 2005. Unexpected effects of zinc pyrethrin and imidacloprid on Japanese medaka fish (*Oryzias latipes*). *Aquatic Toxicol.* 74: 285–293.
- Sarkar, M., P. Biswas, S. Roy, R. Kole, and A. Chowdhury. 1999. Effect of pH and type of formulation on the persistence of imidacloprid in water. *Bull. Environ. Contam. Toxicol.* 63(5): 604–609.
- Sarkar, M., S. Roy, R. Kole, and A. Chowdhury. 2001. Persistence and metabolism of imidacloprid in different soils of West Bengal. *Pest Manag. Sci.* 57: 598–602.
- Scholz, K. and M. Spiteller. 1992. Influence of groundcover on the degradation of ¹⁴C-imidacloprid in soil. *Proc. Brighton Crop Protection Conference – Pests and Dis.* 883–888.
- Schuette, J., J. Troiano, M. Pepple, and J. Dias. 2004. 2004 Update of the Well Inventory Database. [Online]. Available at <http://www.cdpr.ca.gov/docs/emppm/pubs/ehapreps/eh0404.pdf>. (Verified 12 January 2006). CDPR, Sacramento, CA.
- Segawa, R. and J. Walters. 2002. Preliminary Monitoring Results of Imidacloprid Applications for Glassy-winged Sharpshooter Control in Residential Areas of Santa Clara County. [Online]. Available at <http://www.cdpr.ca.gov/docs/gwss/r101602.pdf>. (Verified 12 January 2006). CDPR, Sacramento, CA.

- Segawa, R., J. Walters, S. Fan, S. Matsumoto, and H. Casjens. 2002. Preliminary Monitoring Results of Imidacloprid and Carbaryl Applications for Glassy-Winged Sharpshooter Control in Residential Areas of Imperial County. [Online]. Available at <http://www.cdpr.ca.gov/docs/gwss/r100902.pdf>. (Verified 12 January 2006). CDPR, Sacramento, CA.
- Segawa, R., J. Walters, and S. Fan. 2004. Preliminary Monitoring Results of Imidacloprid and Cyfluthrin Applications for Glassy-Winged Sharpshooter Control in a Residential Area of Solano County. [Online]. Available at <http://www.cdpr.ca.gov/docs/gwss/gwss091704.pdf>. (Verified 12 January 2006). CDPR, Sacramento, CA.
- Stark, J.D., P.C. Jepson, and D.F. Mayer. 1995. Limitations to use of topical toxicity data for predictions of pesticide side effects in the field. *J. Econ. Entomol.* 88: 1081–1088.
- Suchail, S., D. Guez, and L.P. Belzunces. 1999. Characteristics of imidacloprid toxicity in two *Apis mellifera* subspecies. *Environ. Toxicol. Chem.* 19(7): 1901–1905.
- Suchail, S., D. Guez, and L.P. Belzunces. 2001. Discrepancy between acute and chronic toxicity induced by imidacloprid and its metabolites in *Apis mellifera*. *Environ. Toxicol. Chem.* 20(11): 2482–2486.
- Tasei, J.N., G. Ripault, and E. Rivault. 2001. Hazards of Imidacloprid Seed Coating to *Bombus terrestris* (Hymenoptera: Apidae) When Applied to Sunflower. *J. Econ. Entomol.* 94(3): 623–627.
- Tomizawa, M. and J. E. Casida. 1999. Minor structural changes in nicotinoid insecticides confer differential subtype selectivity for mammalian nicotinic acetylcholine receptors. *Brit. J. Pharmacol.* 127: 115–122.
- U.S. Environmental Protection Agency. 2003. Imidacloprid; Pesticide Tolerances. *Fed. Regist.* 68: 35303–35315. [Online]. Available at <http://www.epa.gov/fedrgstr/EPA-PEST/2003/June/Day-13/p14880.htm>. (Verified 09 January 2006). U.S. EPA, Washington, DC.
- U.S. Environmental Protection Agency. 2004. Imidacloprid; Order Denying Objections to Issuance of Tolerance. *Fed. Regist.* 69: 30041–30076. [Online]. Available at <http://www.epa.gov/fedrgstr/EPA-PEST/2004/May/Day-26/p11779.htm>. (Verified 11 January 2006). U.S. EPA, Washington, DC.
- U.S. Environmental Protection Agency. 2005. Imidacloprid; Pesticide Tolerances for Emergency Exemptions. *Fed. Regist.* 70: 3634–3642. [Online]. Available at <http://www.epa.gov/fedrgstr/EPA-PEST/2005/January/Day-26/p1438.htm>. (Verified 17-March-2006). U.S. EPA, Washington, DC.
- Walters, J., H. Casjens, and K. Goh. 2001. Preliminary Monitoring Results of Imidacloprid and Cyfluthrin Applications for Glassy-Winged Sharpshooter Control in Commercial Areas of Butte County. [Online]. Available at http://www.cdpr.ca.gov/docs/gwss/r1018_01.pdf. (Verified 12 January 2006). CDPR, Sacramento, CA.
- Wamhoff, H. and V. Schneider. 1999. Photodegradation of imidacloprid. *J. Agric. Food Chem.* 47(4): 1730–1734.

- Westwood, F., K. Bean, A. Dewar, R. Bromilow, and K. Chamberlain. 1998. Movement and persistence of [¹⁴C] imidacloprid in sugar-beet plants following application to pelleted sugar-beet seed. *Pestic. Sci.* 52(2): 97–103.
- Zheng, W. and W. Liu. 1999. Kinetics and mechanism of the hydrolysis of imidacloprid. *Pestic. Sci.* 55(4): 482–485.

Figure 1: Imidacloprid Breakdown Products

	<p>1 monohydroxy-imidacloprid</p>
	<p>2 Imidacloprid guanidine: 1-(6-chloropyridin-3-ylmethyl)imidazolidin-2-ylideneamine</p>
	<p>3 Imidacloprid urea: 1-(6-chloro-3-pyridimethyl)-imidazolin-2-one</p>
	<p>4 Imidacloprid nitrosimine</p>
	<p>5 Imidacloprid olefin: 1-(6-chloropyridin-3-ylmethyl)-N-nitroimidazolin-2-ylideneamine</p>
	<p>6 6-chloronicotinic acid</p>
	<p>7 6-chloronicotinic aldehyde</p>
	<p>8 6-hydroxynicotinic acid</p>
	<p>9 monoglucoside of 6-chloropicolyl alcohol</p>

