Environmental Fate of Imidacloprid

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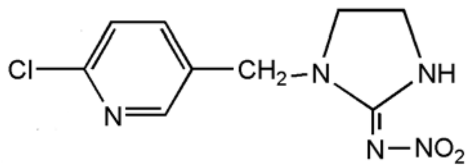
1. Introduction

Imidacloprid is the largest selling insecticide in the world (Simon-Delso et al., 2015). Synthesized by Shinzo Kagabu in 1985, this neonicotinoid insecticide was initially manufactured by Bayer CropScience, but has been off patent since 2006 (Tomizawa and Casida, 2011; Kagabu, 1985). While it is used in both urban and agricultural settings, its largest use is in the agricultural sector. Imidacloprid, like the other neonicotinoids, is a systemic insecticide—it is absorbed by the plant at either the roots or leaves and is translocated throughout the plant. Imidacloprid is also found in veterinary and consumer household products (Simon-Delso et al., 2015). Seed treatment is an especially popular method of imidacloprid application in agriculture since the growing plant is protected from pests by incorporating the insecticide as it grows. The application of neonicotinoids as seed treatments were originally marketed as more environmentally friendly than previous generations of insecticides because of the reduced need for foliar applications (Van Dijk et al., 2013). When piercing and sucking pests like aphids feed on treated plants or treated animals, they ingest the insecticide or are exposed via direct contact following foliar application. Neonicotinoids act by modulating post-synaptic nicotinic acetylcholine receptors (nAChRs), thereby disrupting action potential transmission and ultimately leading to death of the exposed organism (Simon-Delso et al., 2015). Imidacloprid is highly water soluble and is relatively stable in the environment. Imidacloprid and other neonicotinoids have come under scrutiny in the last few years as suspects in pollinator bee colony losses associated with colony collapse disorder (CCD). As such, academia, industry, and regulatory agencies have recently conducted extensive reviews of imidacloprid and neonicotinoids to address the role of these insecticides in CCD (USEPA, 2016; Simon-Delso et al., 2015). In this paper, we update the 2000 and 2006 California Department of Pesticide Regulation reviews and discuss recent findings on imidacloprid's effects on nontarget organisms and its environmental fate (Bacey, 2000; Fossen, 2006).

2. Chemistry

Imidacloprid is a chloronicotinyl nitroguanidine insecticide (Fig. 1). It is a solid at room temperature. Among the neonicotinoids, imidacloprid is grouped with those containing a nitro group (along with clothianidin, nitenpyram, thiamethoxam, and dinotefuran) whereas thiacloprid and acetamiprid are grouped separately as those containing a cyano group (Pisa et al., 2015). Given its low log K_{ow} and high water solubility, imidacloprid is not expected to bind to soils. The physical-chemical properties of imidacloprid are presented in Table 1.

Fig. 1. Molecular Structure:



Chemical Formula: $C_9H_{10}ClN_5O_2$

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 (20ºC @ pH 7)
Vapor pressure	1.00 x 10^{-7} mmHg (20ºC)
Hydrolysis half-life	>30 days (25ºC @ pH 7)
Aqueous photolysis half-life	<2 hours (24ºC @ 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
(20ºC) Octanol-water coefficient (log K_{ow})	3.7
Soil adsorption coefficient:	
K_d	0.956–4.18
K_{oc}	132–310

3. Chemodynamics

3.1 Soil

Imidacloprid is introduced into soil through direct application or diffusion from treated seeds (Mullins, 1993). Degradation in soil is dependent on characteristics such as soil texture, organic matter content, pH, temperature, sunlight exposure, and sunlight intensity for the region. Imidacloprid is not expected to bind to soils given its high water solubility and low adsorption coefficient (K_d). The US EPA modeled 14 turf insecticides and found that imidacloprid had the highest leaching potential among the modeled insecticides (USEPA, 1993). When sorption was studied in Minnesota-sourced soils, Cox et al. (1997) found that sorption increased with organic carbon content in all soils and at all concentrations tested (0.05, 1.5, 25, and 250 μg/L). The predominant factor influencing sorption to soil was found to be soil organic matter (Liu et al., 2006). Thus, leaching of imidacloprid to groundwater through soil may be expected in low organic matter soils. The calculated half-life ($t_{1/2}$) with initial imidacloprid concentration of 50 mg/kg under standard laboratory conditions (25 °C, 60% field moisture capacity and darkness) in red brown earth–Natrixeralf soil (1.2 % organic carbon) collected from suburban Adelaide, Australia ranged from 100 to 1,230 days (Baskaran, 1999). Imidacloprid has a shorter half-life when applied to field with cover crops ($t_{1/2}$=48 days) compared to fields without ($t_{1/2}$ = 190 days) (Scholz et al., 1992). In soil, another study found that imidacloprid could be taken up by plants in tandem with natural degradation processes such that concentrations in soil rapidly decrease over time (Horwood, 2007). Studying degradation rates of various termiticides in soil *in situ,* Horwood (2007) found that "products may degrade more rapidly *in situ* than indicated by laboratory experiments." Taken together, these varying values and ranges suggest that persistence of imidacloprid in soil is highly dependent on field and environmental conditions like soil type, organic matter content, clay content, and emergent vegetation.

3.2 Water

Contamination of surface water can occur during and following many of the methods of application. Dust can settle into surface water following drilling of dressed seeds, spray droplets can drift into nearby water, runoff from treated fields can be contaminated, coated seeds can leach into soil water and ground water, and systemically treated plants can decompose and reintegrate the insecticide back into the soil and soil water (Kreutzweiser et al, 2007). Detections of imidacloprid in surface water (described below) have increased as sales and use have increased. Given the physico-chemical properties of imidacloprid, contamination of groundwater is also possible. Groundwater contamination is likely through similar routes as surface water contamination, yet is a larger concern through seed treatment since the pesticide is placed under the soil surface upon initial treatment. In fact, imidacloprid has a Groundwater Ubiquity Score (GUS) leaching potential index of 3.76, which is classified as high (Bonmatin et al., 2015).

3.3 Air

Imidacloprid has low volatility given its low vapor pressure (1.00×10^{-7} mmHg) and Henry's law constant (6.5×10^{-11} atm m^3/mol). Given the properties of the insecticide, the Air Monitoring Network of CDPR (California Department of Pesticide Regulation) does not monitor for imidacloprid. If imidacloprid is ever present in the air, it will likely be for a brief period following spray application. Another possibility is contaminated, volatilized dust from abrasion and dispersion from mechanical blowers on seed sowing machines during planting of treated seeds (Bonmatin et al., 2015). In this scenario, mechanical abrasion associated with planting coated seeds using a mechanical planter could loosen some of the pesticide coating on treated seeds and the blower on the planter would subsequently disperse the particulate pesticide coating into the air (Greatti et al., 2003), ultimately landing on the soil where it may be incorporated or transported to surface or groundwater.

4. Environmental Degradation

4.1 Biotic

Phugare et al., (2013) reported that imidacloprid degraded up to 78% within 7 days at 30 °C using the bacteria *Klebsiella pneumoniae* strain BCH1. A soil degradation study performed in a laboratory setting (25 °C, 60% field moisture capacity and darkness) found that imidacloprid degraded via first-order kinetics (Baskaran et al., 1999). The 24-month long study found that 37–40% of applied imidacloprid degraded in the red brown earth–Natrixeralf soil. Here, soil moisture content had little to no effect on the rate of imidacloprid degradation. Another study found that in the absence of light, soil degradation half-lives varied between 130 and 160 days (Tisler et al., 2009).

4.2 Abiotic

Hydrolysis

Hydrolysis of imidacloprid is dependent on pH, with increases in alkalinity corresponding to increases in the rate of degradation (Zheng and Liu, 1999). Water with low or neutral pH (pH=3, 5, or 7, respectively) slowly degrades imidacloprid, with one study reporting 1.5% of the pesticide degraded after 3 months (Zheng and Liu, 1999). In pH 9 water, however, original concentrations of imidacloprid decreased by 20% after 3 months. Furthermore, at pH 10.80 and 11.80, the hydrolysis data fit a first-order kinetics equation, with degradation at the higher pH occurring more rapidly. Liu et al., (2006) compared photodegradation and hydrolysis in the dark with intermittent shaking in a 20 mg/L clay-free solution and clay suspension and found that hydrolysis occurred more slowly than photodegradation due to the higher activation energy required by hydrolysis. Zheng and Liu (1999) also reported detection of only one main hydrolysis product, 1-[(6-chloro-3-pyridinyl)methyl]-2-imidazolidone (imidacloprid urea)—a finding also confirmed by Liu et al., (2006).

Photolysis

Imidacloprid degrades via aqueous photolysis following a first-order reaction rate in a matter of hours, with a reported half-life of 43 minutes in HPLC grade water (Wamhoff and Schneider, 1999). Moza et al. (1998) reported that 90% of imidacloprid in aqueous solution (deionized water) degrades after being irradiated (290 nm) for 4 hours - with a half-life of 1.2 hours. More importantly, degradation of the insecticide in this study did not occur when the aqueous solution was kept in the dark. Using GC-MS, Liu et al. (2006) detected similar photoproducts as Moza et al. (1998) (Fig. 2).

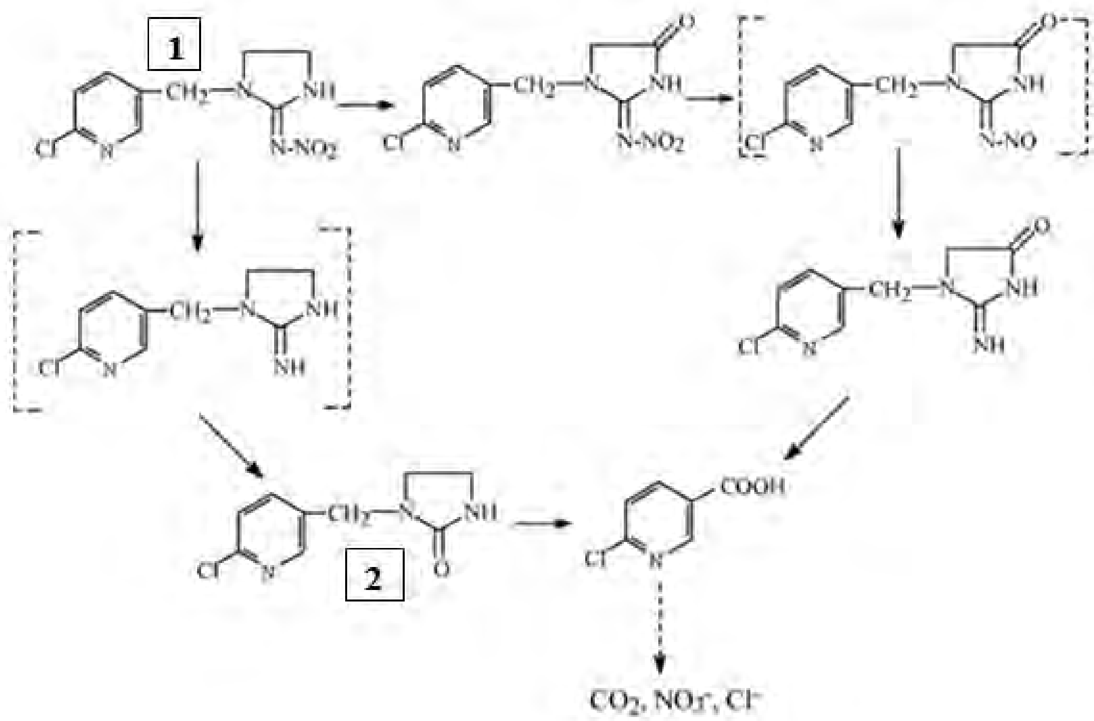


Fig. 2: Proposed pathway for photolysis of imidacloprid in water, adopted from Liu et al. (2006). Dashed brackets represent degradate intermediates. Compound 2, imidacloprid-urea, was the most abundant degradate from the parent imidacloprid, compound 1.

4.3 Use and Detections

Imidacloprid monitoring data, including detections, in California surface water are available beginning in 2000 in the CDPR Surface Water Database (SURF). Unfortunately, there is no data on imidacloprid in the CDPR SURF database for 2006–2009. In 2005, there were 9 detections of imidacloprid (52.9% of the 17 samples analyzed) in California surface water (according to the CDPR SURF Database), but none of the detections exceeded the US EPA chronic invertebrate aquatic life benchmark of 1.05 µg/L (US EPA 2015). However, in 2010, 32 detections (37.2% of the 86 samples analyzed) were recorded with one US EPA benchmark exceedance. By 2014, there were 82 detections of imidacloprid (71.3% of the 115 analyzed samples) in surface water by studies cited in the CDPR SURF database (CDPR, 2016). The newest data for 2015 contain 113 analyzed samples with 78 detections (69.0% detection frequency) and 16 benchmark exceedances. Of the 841 samples stored in the SURF database since records for imidacloprid monitoring became available in 2000, 65 were above the US EPA benchmark (CDPR SURF Database).

Reported use in agricultural settings in California derived from the Pesticide Use Reporting (PUR) database, which does not include seed treatments, in 2014 (the year for which the most-current data is available) totaled 374,061 pounds (CDPR, 2015). The top three sites that were treated with imidacloprid were wine grapes, structural pest control, and grapes (Table 2). Reported imidacloprid agricultural use more than tripled from 2003 to 2013 (Fig. 3). This trend comes as no surprise given the previously reported sales and use figures for imidacloprid (Simon-Delso et al., 2015). Linear regressions were performed between existing benchmark exceedance frequency and imidacloprid use data from PUR for the same year and one year prior. Analysis with PUR of one year prior (i.e., use one year prior chosen to capture all runoff into surface water from previous applications) can give insight into exceedances of the current year and their correlation to product applications from the previous year. The results suggest that benchmark exceedance is correlated with PUR (correlation coefficient=0.708 and 0.859 for PUR of the same year and one year prior, respectively) (Fig. 4, Fig. 5).

Table 2. Top ten use sites for imidacloprid in California in 2014, according to PUR

Site	Pounds imidacloprid
Grape, Wine	56,254
Structural Pest Control	44,093
Grape	36,939
Tomato, Processing	35,344
Orange	22,160
Broccoli	15,970
Landscape Maintenance	15,084
Tangerine	14,244
Pistachio	12,643
Lettuce, Head	12,471

A monitoring study focusing on three agricultural regions in California in 2010 identified the potential for imidacloprid to move off-site and contaminate surface water (Starner and Goh, 2012). This study reported that 14 water samples (19% of total samples) exceeded the US EPA chronic invertebrate aquatic life benchmark. Pursuant to section 13145(d) of the California Food and Agricultural Code, imidacloprid is on the CDPR Groundwater Protection List—a list of pesticides identified by CDPR that have the potential to pollute groundwater. However, a 2009 study by CDPR that monitored for imidacloprid in groundwater did not detect it in any of the 34 wells sampled (Bergin and Nordmark, 2009).

In a study focused on urban surface water monitoring in Southern California, imidacloprid was detected in 73% of the 40 samples analyzed during the July 1, 2014–June 30, 2015 sampling period (Budd, 2016). The Northern California branch of the same monitoring program detected imidacloprid during the same sampling period in 6 of the 36 samples analyzed (Ensminger, 2016).

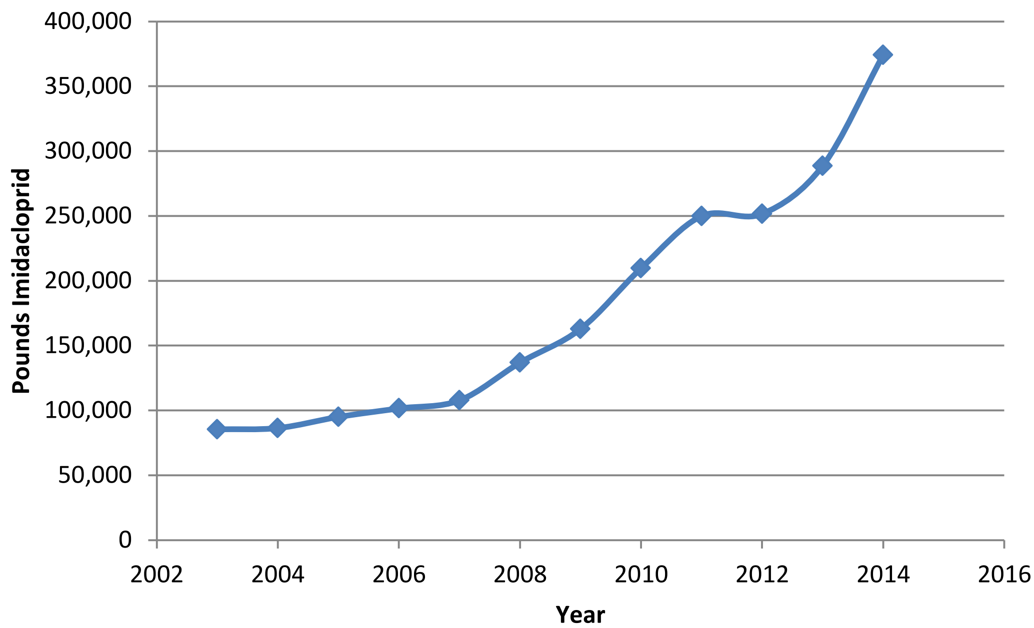


Fig. 3. Imidacloprid pesticide use, California, 2003–2014.

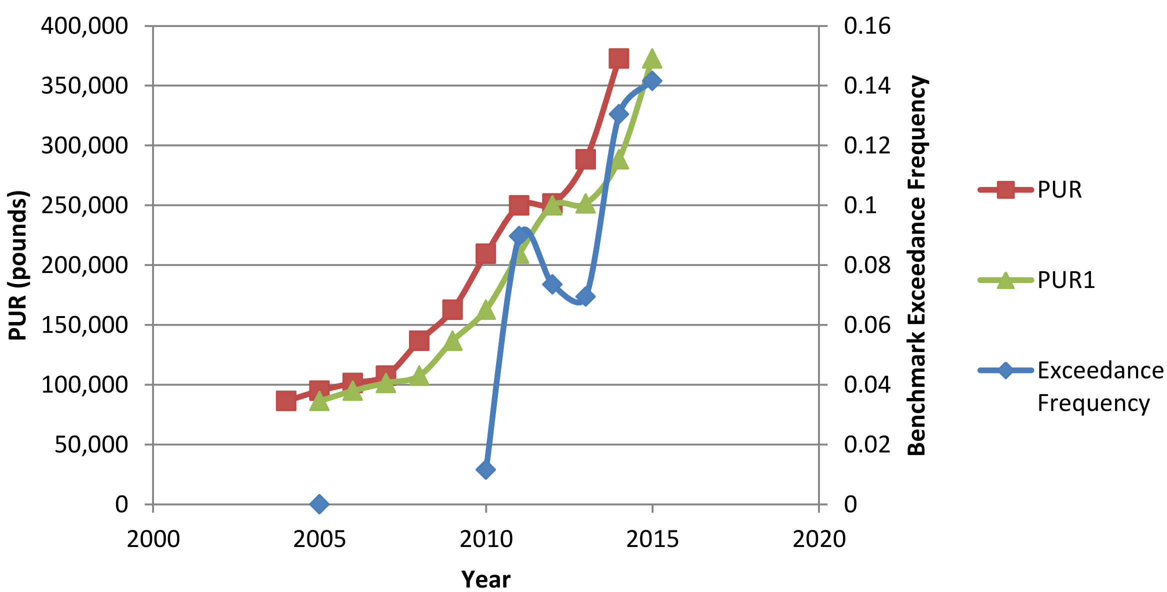


Fig. 4. Imidacloprid pesticide use (PUR) and use one year prior (PUR1) versus chronic aquatic life benchmark exceedance frequency.

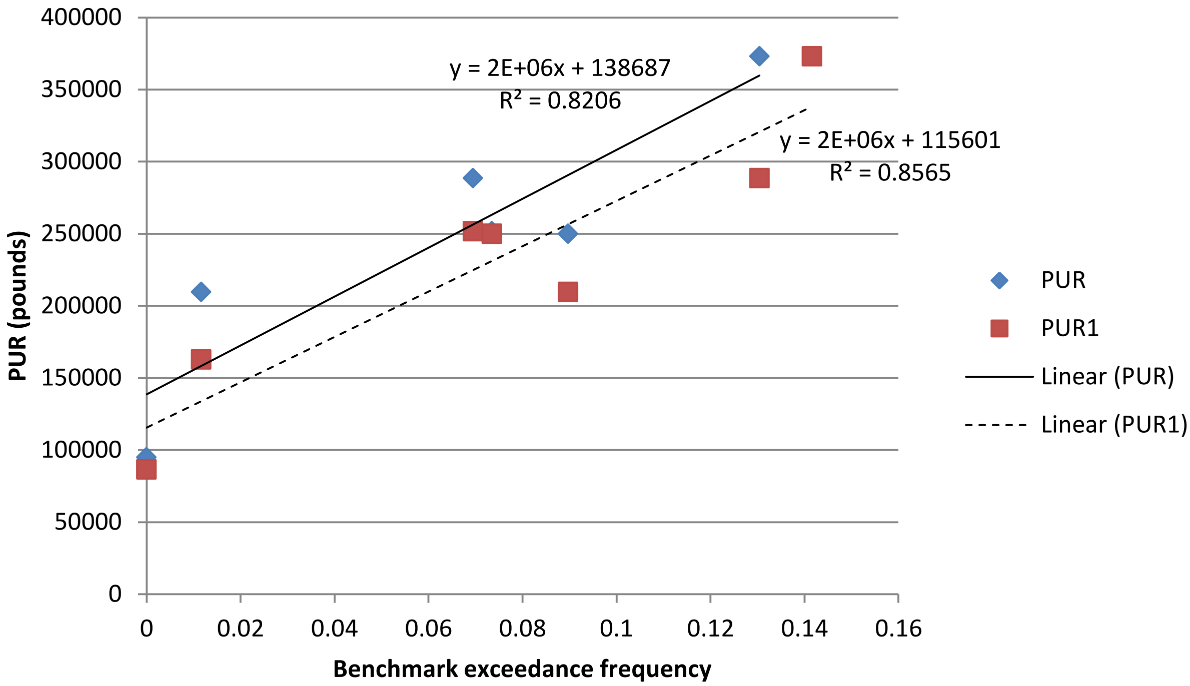


Fig. 5. Linear regressions of pesticide use (PUR) and use one year prior (PUR1) vs benchmark exceedance frequency.

5. Toxicology

5.1 Mode of Action

Imidacloprid acts at the insect nicotinic acetylcholine receptor (nAChR; Liu and Casida, 1993). The insecticide mimics the activity of neurotransmitters by agonistically binding and sending unwarranted neural transmissions. Ultimately, receptors and cells involved in neural transmission become exhausted and fail to function, which results in paralysis (Nishiwaki et al., 2003). Nicotinic receptors with affinity for imidacloprid and other neonicotinoids occur in lower numbers in vertebrates than invertebrates. Thus neonicotinoid toxicity, including imidacloprid, is generally higher in invertebrates than vertebrates (Simon-Delso et al., 2015).

5.2 Aquatic organisms

A large body of published literature exists that addresses the effects of imidacloprid on aquatic macrofauna and other nontarget organisms (Table 3). These studies include lab toxicity tests to stream mesocosm studies to field studies. Fish are less sensitive than invertebrates to the toxic effects of imidacloprid. The LC_{50} values of fish species tested, according to Gibbons et al. (2015), range from 1.2 mg/L for rainbow trout fry to 241 mg/L for zebrafish. These fish sensitivities are orders of magnitude higher than ambient concentrations detected by CDPR. Thus, it is unlikely that mortality from direct exposure to imidacloprid will affect fish species at current ambient concentrations. Investigating effects to more sensitive invertebrates, Stoughton et al. (2008) conducted a 28-day chronic exposure using the aquatic invertebrates *Chironomus tentans* and *Hyalella azteca*. Growth and survival as measured by the Lowest Observed Effect Concentration (LOEC) were inhibited in *C. tentans* at concentrations >1.14 μg/L. Likewise, *H. azteca* had a 28-d LOEC of 11.46 μg/L. The reported 28-day LC_{50} for *C. tentans* in this same study was 0.91 μg/L. Sanchez-Bayo et al. (2006) reports that ostracods, a class of crustaceans, (48-hour LC_{50}=301–715 μg/L) are orders of magnitude more sensitive to acute imidacloprid exposure than cladocerans, an order of crustaceans (48-hour LC_{50}=65–133 mg/L). Chen et al. (2010) reported a 48-hour LC_{50} of imidacloprid to *Ceriodaphnia dubia* as 2.1 μg/L. The same study found that 19% of the exposed population survived (relative to the control) following chronic exposure at a concentration of 0.3 μg/L. The US EPA chronic invertebrate aquatic life benchmark for imidacloprid is 1.05 μg/L (US EPA, 2015). However, this benchmark was developed in 2008 and there are recent calls for the benchmark value to be lowered drastically in an effort to reflect newer data (Morrissey et al., 2015; Smit et al., 2015). Morrissey et al., (2015) and Smit et al., (2015) agree that the acute threshold should be 0.2 μg/L in order to avoid chronic effects on the most sensitive invertebrate species, but each realizes a different chronic threshold—0.035 μg/L and 0.0083 μg/L, respectively. Nevertheless, concentrations of imidacloprid, especially in agricultural areas of California, are reported in the SURF database (CDPR, 2016) at levels capable of causing short- and long-term impacts on aquatic invertebrate species.

Table 3. Range of LC_{50} values for different taxa

Taxon	96-hr LC_{50} range	Reference
Mammal	131–475 mg/kg	SERA, 2005
Bird	13.9–283 mg/kg	SERA, 2005; Fossen, 2006; Anon 2012
Fish	1.2–241mg/L	SERA, 2005; Cox, 2001
Amphibia	82–366 mg/L	Feng et al., 2004; Nian 2009
Coccinellid	17.25–364 mg/kg	Khani et al., 2012; Youn et al., 2003
Hemiptera	0.3–5,180 mg/kg (residual contact)	Delbeke et al., 1997; Prabhaker et al., 2011
Branchiopoda	.0021–10.4 mg/L	Song et al., 1997; Chen et al., 2010

5.3 Mammals and Birds

Much of the focus in toxicology research has been on invertebrates, especially pollinators (discussed below). Nevertheless, a number of studies have focused on effects to birds and mammals. Imidacloprid can affect birds and mammals directly through toxicity or indirectly through effects to the food chain (Gibbons et al., 2015; Mineau and Palmer, 2013). While imidacloprid is more toxic at lower concentrations to invertebrates than vertebrates, the latter still experiences toxicity from imidacloprid (Gibbons et al., 2015). The 96-hour LC_{50} for different vertebrate taxa varies greatly (Table 2). The LD_{50} for the range of bird species tested spans from 13.9 mg/kg bodyweight for the gray partridge to 283 mg/kg bodyweight for the mallard (Gibbons et al., 2015). While direct exposure is a concern, the indirect effects like growth, development, and reproduction on vertebrate wildlife pose unique challenges as well. One hypothesized indirect effect is the relationship between sensitive invertebrate prey and the vertebrate wildlife that depend on them as a food source. The evidence is not clear as to whether there is a link between pesticide use resulting in decreased invertebrate prey and a decline in vertebrate wildlife populations (Gibbons et al., 2015). Given that indirect effect endpoints like growth and development are difficult to assess, more research is needed to characterize the potential role of imidacloprid to cause sublethal effects.

5.4 Pollinators

Honeybees (*Apis mellifera*) have been widely studied and discussed in recent years since pollinators responsible for a large portion of food crop pollination have seen steady population declines associated with CCD (Pisa et al., 2015). Given the high toxicity of imidacloprid and other neonicotinoids to bees and non-target invertebrates, studies have recently focused on the relationship between neonicotinoid use, CCD, and the health of the global bee population. Mullin (2010) reported an average bee LD_{50} of 280 ng/g bee despite other values ranging from 4 to 104 ng/honeybee (Johnson and Pettis, 2014). Bonmatin et al. (2005) reported that imidacloprid has an acute LD_{50} to bees of 3.7 ng/bee. To put this in perspective, the LD_{50} for DDT is 27,000 ng/bee. Other reported values for the LD_{50} of imidacloprid are higher. Risk assessments focusing on bees reported the LD_{50} to be 490 ng/bee (DEFRA, 2007; 2009). This large discrepancy in reported values may be explained by the differences between oral and contact toxicity, with oral ingestion serving as the more sensitive route of exposure (Pisa et al., 2015).

Sublethal effects of imidacloprid on bees have also been studied. Blanken et al. (2015) studied the relationship between imidacloprid and the parasitic mite *Varroa destructor* with respect to flight capacity of forager bees. Previous studies found that imidacloprid and neonicotinoids could reduce homing of forager bees by altering orientation abilities (Henry et al., 2012). Blanken et al. (2015) found that exposure to *V. destructor* reduced flight distance but the effect increased when bee colonies were exposed to both *V. destructor* and imidacloprid. Despite the increased focus of research efforts on neonicotinoids and honeybees, as Pisa et al., (2015) point out, “No single cause for high losses has been identified, and high losses are associated with multiple factors including pesticides, habitat loss, pathogens, parasites, and environmental factors.”

An extensive risk assessment was released in January 2016 by the US EPA that analyzed the risk imidacloprid poses to bees on different crops (US EPA 2016). This assessment found that imidacloprid sprayed on citrus and cotton posed a risk to bee colony health. A no-observable adverse effect concentration (NOAEC) was set to 25 μg/L for nectar with a lowest-observable adverse effect concentration (LOAEC) at 50 μg/L. Citrus and cotton were identified as risks in the study given the pollen and nectar exposure routes for bees. In these two crops, nectar and pollen may contain imidacloprid above the NOAEC. Other studied crops like corn, which do not contain nectar, are not serious risks to bees for imidacloprid exposure.

6. Summary

Imidacloprid, the predominant neonicotinoid and largest selling insecticide in the world, was initially synthesized in 1985. It is a systemic insecticide applied predominantly in agriculture as a seed treatment to protect against crop damage from biting-sucking pests. Following ingestion, imidacloprid disrupts action potential transmission in the pest by agonistically binding to post-synaptic nAChR receptors. The predominant environmental route for breakdown of imidacloprid is through aqueous photolysis, which has a half-life of <2 hours. The insecticide is highly water soluble (514 mg/L) with a Henry’s Law constant of 6.5×10^{-11} atm m^3/mole. Thus, volatilization is not a major dissipation pathway. While not a concern in air, imidacloprid remains a threat to sensitive species in surface water—prompting calls for a reduced chronic aquatic life benchmark. Imidacloprid is on the CDPR Groundwater Protection List, but CDPR studies monitoring for imidacloprid have not detected it in the state.

The science behind the effect of imidacloprid on honey bees and other pollinators, especially with respect to CCD, is still not settled. The recently published US EPA risk assessment on imidacloprid identified cotton and citrus as the only two crops which, when treated with imidacloprid, could introduce bees to toxic concentrations. It is important to note that other stressors like the *V. destructor* mite, habitat loss, and nutrition quality are factors in the reported decline of pollinators nationwide. More research and analysis of existing data is needed in order to decisively identify the relationships between pollinator stressors and CCD.

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