The IPM Practitioner
Monitoring the Field of Pest Management
Volume XXXIII, Number 1/2, January/February 2011

Pesticides and Honey Bee Death and Decline

By William Quarles

A large number of overwintering honey bees are dying in the U.S. For the last five years, winter losses of managed honey bee colonies have been around 30% each year (van Engelsdorp et al. 2012). Overwintering honey bees are being killed by pathogens, pests, poor nutrition, and pesticides. Honey bee problems are part of the overall pollinator decline in the U.S. (Spivak et al. 2011; NAS 2007).

Managed honey bees are trucked from state to state and forage over large areas. Most of the crops they encounter have been treated with pesticides, and chemical analysis of overwintering honey bee hives shows extensive pesticide contamination (Mullin et al. 2010).

Pesticides are accumulating in hives, and bees are also being killed while foraging in fields (Krupke et al. 2012). Part of the problem is exposure to systemic insecticides called neonicotinoids. Insecticides are normally applied in ways to mitigate their impact on bees. Mitigation strategies are not possible with systemics because they are always present in the plant. Over 59 million ha (146 million acres) of crops in the U.S. have been treated with systemics. This represents about 45% of the total cropland, and acreage is increasing each year (Mullin et al. 2010; Stokstad 2012; Spivak et al. 2011).

Pesticides can impact bee populations through direct mortality and through sublethal effects on behavior, such as impaired memory, learning and foraging. Impaired foraging can lead to poor nutrition, and pesticides may directly impact bee immune systems, making them more susceptible to disease. In addition, sublethal pesticides interfere with brood development and shorten lifespans of adults (Henry et al. 2012; Pettis et al. 2012; Wu et al. 2012; Desneux et al. 2007).

Photo courtesy of Kathy Keatley Garvey

A honey bee, *Apis mellifera*, is headed toward an almond blossom. Massive losses of these managed honey bees are occurring each year, and pesticide poisoning is part of the problem.

Pesticides may also contribute to Colony Collapse Disorder (CCD). This phenomenon was first observed in the U.S. in 2006. Bees disappear from the hive, leaving food, brood, and even a queen (USHR 2007; Quarles 2008a). Despite intensive research, an exact cause of CCD has not been identified. There may be a number of causes working synergistically. But it has been established that overwintering bee colonies are under stress, and one of those stresses is pesticides (Spivak et al. 2011; USHR 2008; Quarles 2008a). One observation that seems to implicate pesticides is that organic beekeepers do not seem to have CCD (Schacker 2008).

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The IPM Practitioner
Monitoring the Field of Pest Management

The *IPM Practitioner* is published six times per year by the **Bio-Integral Resource Center (BIRC)**, a non-profit corporation undertaking research and education in integrated pest management.

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FEI# 94-2554036.

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 ISSN #0738-968X

Update

Helga Martin Williamson Olkowski
1931-2012
Co-Founder of the Bio-Integral Resource Center

Helga Olkowski passed away peacefully at home on April 27, 2012 from complications from a stroke. Helga was active in many environmental organizations, and she was co-founder of the Farallones Institute, the John Muir Institute, and others. Helga actively promoted organic agriculture, writing for *Organic Gardening* and other magazines. She was coauthor of several influential books, including *The City People's Book of Raising Food*, *The Integral Urban House* and *Common Sense Pest Control*. She was co-founder of the Bio-Integral Resource Center (BIRC) and worked for years as an editor for BIRC, writing articles for the *IPM Practitioner* and *Common Sense Pest Control Quarterly*.

Helga retired from BIRC in 1999, and spent many enjoyable years traveling with her husband, William Olkowski. She suffered a stroke about three years ago from which she never completely recovered. We will miss her.

A more complete biography can be found at her website www.who1615.com

Are Pesticides found in Bee Hives?

Bees can come into contact with pesticides when foraging or when the hive is treated with pesticides to kill mites. Foragers can collect contaminated pollen and nectar and bring it back to the hive. Some of the nectar and pollen is mixed together with enzymes to form bee bread. In the hive bees evaporate water from nectar to produce honey. Any pesticide in the nectar is concentrated at least 4x in the honey, which is stored for later use. So bees can be exposed both in the field and in the hive (Bonmatin et al. 2005; Kievits 2007).

Bee exposure to pesticides is widespread. Mullin et al. (2010) checked a large number of commercial bee hives for pesticides. Hives from 23 states including Florida, California, Pennsylvania and migratory bees from East Coast colonies were analyzed. Wax, pollen, and bees were highly contaminated with pesticides. There were 121 different pesticides and metabolites in 887 wax, bee, and pollen samples, averaging about 6 pesticides per sample.

This diverse contamination opens the question of synergism. Mixtures of pesticides are known to be more toxic to bees than individual products. Some fungicides, for instance, are known to increase the toxic effects of insecticides (Johansen 1977; Atkins 1992; USHR 2008; Pilling and Jepson 1993; Schmuck et al. 2003; Isawa et al. 2004).

The 350 pollen samples contained about 98 different pesticides and metabolites in concentrations up to 214 ppm. Each pollen sample averaged about 7 different pesticides, up to a maximum of 31.

Pollen was contaminated from miticides and fungicides applied in the hive, and insecticides, herbi-

cides, and fungicides applied in the field. Pyrethroids were the most frequently detected insecticide, and were sometimes found at levels known to disorient foraging bees. Fungicides were the predominant pesticide type found in pollen (Mullin et al. 2010).

Contamination similar to this can lead to delayed development of bees and can shorten life span of adult workers. Premature death of foragers forces nurse bees to forage, with further consequences on colony health (Wu et al. 2011).

Neonicotinoids

Among the pesticides found in bee hives by Mullin et al. (2010) were neonicotinoids. These pesticides are analogs of the neurotoxin nicotine and have similar actions. Neonicotinoids include imidacloprid, clothianidin, thiamethoxam and others. They are applied as seed treatments to a number of crops, including corn, sunflower, cotton, and canola. Foliar sprays, soil drenches, and seed treatments are used. Both crop plants and ornamentals are treated (Elbert et al. 2008; Stokstad 2012; Hopwood et al. 2012).

Mullin et al. (2010) found bee pollen in hives contained imidacloprid at a median concentration of 20 ppb and a maximum concentration of 206 ppb. These levels are known to impact the health of bees. A total of 43 pollen samples (12%) out of 350 contained neonicotinoids or their metabolites. Mullin et al. were analyzing hives foraging on specialty crops such as citrus, apples and others that do not use seed treatments. Where bees forage on crops such as corn, canola, or sunflowers that use neonicotinoid seed treatments, 50% of pollen samples carried by honey bees can be contaminated with these pesticides (Krupke et al. 2012; Lu et al. 2012; Blacquiere et al. 2012).

There is no doubt that these potent new pesticides can kill bees if bees are exposed. Just 3.7 billionths of a gram of imidacloprid will likely kill a bee (oral LD50= 3.7 to 81 ng/bee). The oral LD50 of clothianidin is 2.8 to 3.7 ng/bee,

Photo courtesy of Kathy Keatley Garvey

Commercial hives can be heavily contaminated with pesticides.

and contact toxicity is 22-44 ng/bee. For comparison, the oral LD50 of cypermethrin is 160 ng/bee and for the organophosphate dimethoate 152 ng/bee (Colin et al. 2004; Schmuck et al. 2001; Suchail et al. 2001ab; Krupke et al. 2012).

As we see in Table 1, clothianidin, thiamethoxam, dinotefuran, and imidacloprid are extremely toxic to bees, acetamiprid and thiacloprid less so. We can also see that there can be a wide range of toxicity. Effects can vary depending on genetic variation in bees and other factors (Hopwood et al. 2012; Quarles 2008).

Complicating Factors in the Field

Neonicotinoids are causing concern due to widespread bee exposure, their potency to bees, and their persistence in the field (see Table 1). Sublethal doses can cause impaired learning and foraging. These effects have been measured at very low concentrations in the laboratory, but critics point out that there are mitigating effects in the field. Bees can collect pollen from untreated plants, and dilute pesticide effects. So experiments with neonicotinoids and bees often become a numbers game. If an effect is detected, the first criticism is that doses used were not representative of concentrations found in the field (Stokstad 2012; Hopwood et al. 2012).

It is true that dilution from untreated plants can occur in the field. Nguyen et al. (2009) found that imidacloprid treated corn fields in Belgium had no effect on mortality of honey bee hives found within 3 km (1.8 mi) of the fields. However, only 13.2% of the corn acreage within foraging range had been treated, and these treated fields represented a maximum 2.48% of the foraging area. So effects on bees from treated acreage can be diluted in the field by access to other food sources. But as more and more acreage is planted with systemics, then bees will have problems finding untreated plants (Hopwood et al. 2012).

Experiments have been conducted where hives are placed near treated fields and monitored for effects. Unfortunately, these colonies are often monitored over a relatively short period of time. Honey bee colonies have at least two generations a year. So it is not enough to measure the effects on one generation. Chronic sublethal doses in one generation can reduce the number

Table 1.
Toxicity of Neonicotinoids to the Honey Bee, *Apis mellifera

Neonicotinoid	Oral LD50 (ng/bee)	Contact LD50 (ng/bee)	Soil Half Life (days)
Clothianidin	2.8-3.79	22-44	148-1,155
Imidacloprid	3.7-81	17.9-243	40-997
Thiamethoxam	5	24-29	25-100
Dinotefuran	7.6-23	24-61	138
Thiacloprid	8,510-17,300	14,600-38,830	1-27
Acetamiprid	8,850-14,520	7,100-8,091	1-8

*from Hopwood et al. 2012, Laurino et al. 2011. One nanogram (ng) is one-billionth of a gram.

Photo courtesy of Kathy Keatley Garvey

Honey bees can be killed by acute exposure to aerially dispersed seed coatings containing neonicotinoids. Chronic exposure can cause foragers to lose their way home.

of bees in the next generation (Lu et al. 2012).

Sublethal Doses in Hives

Krupke et al. (2012) found sick hives had pollen concentrations up to 10.7 ppb clothianidin or 20.4 ppb of thiamethoxam. Pollen is fed to larvae by nurse bees. A nurse bee will consume 65 mg of pollen in 10 days. If the pollen contains 20 ppb clothianidin, 65 mg will contain 1.3 ng, about 50% of the LD50 of 2.8 ng/bee. Sublethal doses of 1.3 ng are high enough to disorient foragers and cause field losses of bees (Henry et al. 2012).

Sublethal concentrations of neonicotinoids and other pesticides in brood comb can delay development of adult bees. Delayed development can make the bees more susceptible to mites. Pesticides in the brood comb also shorten life span of adult bees (Wu et al. 2011).

Bee colonies have even been killed by feeding them neonicotinoids at chronic sublethal concentrations of 20 ppb, which is close to what they could encounter in the field. Lethal effects were not seen for months (Lu et al. 2012).

Can Field Concentrations of Neonicotinoids Kill Bees?

Neonicotinoids can kill bees foraging in fields. Most of the 35.7 million ha (88.2 million acres) of corn in the U.S. are treated. Application rates are 0.25 to 1.25 mg/kernel, and the pesticide on one seed is enough to kill 80,000 bees. Fortunately, most of the pesticide is buried with the seed (Hopwood et al. 2012; Krupke et al. 2012).

But flying bees can be directly exposed to aerially dispersed seed coatings and talc from planting machines. Talc can contain 3,400 to 15,043 ppb clothianidin, which is many times the lethal dose for a bee. Exposures of this kind have led to honey bee deaths in the field. Mortality increases with humidity (Krupke et al. 2012; Marzaro et al. 2011; Tapparo et al. 2012; Girolami et al. 2012).

In May of 2008, about 50% of honey bees in the German state of Baden-Wurttemberg were killed. The problem was traced to the application of the systemic pesticides clothianidin and imidacloprid to seeds. According to the manufacturer, farmers applied these pesticides without using the adhesives recommended to keep the pesticides localized to seeds. Germany banned the use of these pesticides for seed treatment after this incident (ENS 2008; EPA 2008). Bee deaths during planting season have been seen in other European countries (Mazaro et al. 2011).

Even if adhesives have been properly applied, bees can still be killed by careless operation of planting machines. Krupke et al. (2012) investigated the cause of dead bees in apiaries in Indiana. They found that dead bees and pollen from their hives contained the neonicotinoids thiamethoxam and clothianidin. Some of the pollen samples had clothianidin levels higher than the LD50. Returning foragers from hives near fields had pollen concentrations up to 88 ppb of clothianidin.

Aerial seed waste also contaminates soil, surface water, and wild plants found near field margins. Some of these, such as dandelions, are attractive to bees. Concentrations of 6 ppb clothianidin were found in soil after treated seeds were planted. Dandelion plants near corn fields had residues of up to 9.4 ppb. The neonicotinoids are persistent, and some have soil half lives of more than a year. This means that material from one year can appear in the next year's planting. Soil contamination can also put soil nesting bees at risk (Hopwood et al. 2012).

According to the California EPA, where imidacloprid is being used, models suggest expected concentrations in surface water of 17 ppb, and 2 ppb is expected in groundwater. Residues on plants near a crop site can be 14-54 ppb (Fossen 2006).

Some of these problems can be mitigated by filtering air from planting machines to prevent dispersal of contaminated talc and seed coatings (Mazaro et al. 2011). Other

researchers question the need for seed treatments in corn, citing effective IPM practices, and the problem of insect resistance with systemic pesticides (Maini et al. 2010).

Neonicotinoids in Guttation Drops

Bees can also be exposed through guttation water from plants. Corn excretes droplets of water along leaf margins called guttation drops. For about 3 weeks after emergence, droplets from treated corn contain large concentrations of neonicotinoids: 47 to 83 mg/liter imidacloprid, 23 mg/liter clothianidin, about 12 mg/liter for thiamethoxam (Girolami et al. 2009). Therefore, the levels in guttation fluid can be 254 times the LD50 for imidacloprid, 280 times the LD50 for clothianidin and 48 times the LD50 for thiamethoxam. Guttation droplets fed to bees in the laboratory will kill them (Thompson 2010). Lethal guttation drops can also be produced by melon crops with neonicotinoid soil treatments (Hoffman and Castle 2012).

Critics say that bee behavior must be taken into account. Droplets may appear in the morning before bees start foraging. Bees use water to cool their hives. Hives may not need cooling in the morning. Guttation water may not be a common source of water, since bees need large amounts of water and are fond of irrigation water and large sources (Hopwood et al. 2012; Girolami et al. 2009).

Neonicotinoids in Pollen and Nectar

Bees can also be exposed to contaminated nectar and pollen produced by treated plants. Though concentration in nectar and pollen may be low, chronic doses can accumulate because bee metabolism and elimination of neonicotinoids such as imidacloprid (IMD) are slow. Metabolism is complex and thiamethoxam is actually converted by metabolism into clothianidin (Hopwood et al. 2012; Krupke et al. 2012; Suchail et al. 2001ab).

Imidacloprid (IMD) is often applied as a seed treatment. Sunflower seed treatments can lead to concentrations of 13 ppb in sunflower pollen (Laurent and Rathahao 2003). Other experiments show 3.9 ppb in sunflower pollen, 8 ppb in flowers, and 1.9 ppb in nectar. Rape has 4.4 to 7.6 ppb in pollen. Corn can have average concentrations of 2.1 ppb in pollen and 6.6 ppb in flowers. Some corn plants show concentrations of 18 ppb in pollen (Fossen 2006; Bonmatin et al. 2005). Bees could ingest IMD in pollen, nectar, and water. They could be exposed by contact on flowers and leaves of treated plants (Blacquiere et al. 2012).

From Nixon 1954

Soil contamination puts groundnesting bumble bees, *Bombus* spp., at risk.

Treated plants metabolize IMD to toxic metabolites, and one of them is twice as toxic to bees as IMD. Chauzat et al. (2006) found IMD metabolites in 44% of pollen samples collected in France. Bayer researchers found that about 15% of IMD in sunflower pollen had metabolized (Sur and Stork 2003).

Neonicotinoids are also used as foliar sprays, as soil drenches, and for treating landscape ornamentals as well as crop plants. Amounts used on ornamentals lead to residues 12-16x greater than found on crop plants (Hopwood et al. 2012).

Cresswell (2011) combined a number of studies on imidacloprid into a meta-analysis and concluded that "trace dietary imidacloprid at field realistic levels in nectar will have no lethal effects, but will reduce expected performance in honey bees by between 6 and 20%."

Cresswell, however, included no studies on ornamentals and tossed out studies (Suchail et al. 2001ab) showing mortality from small chronic doses. Also excluded were studies showing lethal field levels of imidacloprid due to guttation droplets and airborne seed residues (Krupke et al. 2012; Girolami et al. 2009).

Can Field Concentrations of Pesticides Lead to Impaired Foraging?

Yang et al. (2008) found that concentrations of imidacloprid of 40-50 ppb in sugar water were enough to cause impaired foraging of honey bees in the field. Nectar concentrations from seed treatments are lower than this, but even if nectar concentrations are low, fairly large chronic doses can be delivered. A bee ingests 20-30 µl of nectar each time, and the half life of IMD is about 4.5 hrs, making chronic accumulation possible. Imidacloprid is also metabolized by bees into toxic metabolites that can also accumulate (Suchail et al. 2003; 2004).

Although nectar from seed treatments do not regularly reach 40-50 ppb in the field, these concentrations occur with some other crops. Thiamethoxam soil drenches to pumpkins at label rates with half applied to transplants and half applied during flowering led to nectar concentrations of 54.8-90.4 ppb (Hopwood et al. 2012). Bees exposed to this concentration could receive the doses used by Yang et al. (2008).

Cresswell (2011) estimates that a honeybee ingests an average nectar load of 40 mg. If nectar contained 50 ppb (ng/gram), then 2.0 ng of toxin would be ingested with each load. Faucon et al. (2005) estimate that foraging bees have an 11.5 mg/hour nutrient need from pollen and nectar. If the pollen or nectar contained 50 ppb, about 1.8 ng of toxin would be accumulated in 3 hours.

Neonicotinoids are known to impair bee foraging efficiency in the laboratory. An experimental challenge is measuring these effects in

a field situation. One way is to identify treated bees with a microchip. Henry et al. (2012) equipped 653 honey bees with a 3 mg microchip. An individual adult bee weighs 80-100 mg, so this roughly represents a weight handicap of about 3%. Bees were treated with a sublethal dose of 1.34 ng of thiamethoxam, which is about 27% of the LD50 of 5 ng/bee. It was administered in a sugar solution containing thiamethoxam (1.34 ng in 20 µl).

Losses Higher in Unfamiliar Terrain

Henry et al. (2012) released treated bees, along with equal numbers of untreated bees up to 1 km (0.6 mi) away from the hive. Hives were equipped with microchip (RFID)

Foraging of bumble bees, *Bombus* spp., can be impaired by neonicotinoids.

Photo courtesy of Gary McDonald

monitoring equipment. Some of the treated foragers were released in a familiar field of *Phacelia*, others were released in unfamiliar surroundings. About 10% of treated bees released in familiar surroundings failed to make it back to the hive. About 32% of treated bees released in unfamiliar surroundings failed to return. Most commercial honey bee hives are trucked from place to place and released in an unfamiliar environment, maximizing pesticide effects on foraging.

Schneider et al. (2012) found similar results with microchip experiments. When bees were treated with 1 ng of orally ingested clothianidin, about 26% did not return to hive. With 2 ng, 79% did not return. Impairment was noticed at 1.5 ng imidacloprid or 0.5 ng clothianidin.

Calculations by Henry et al. (2012) showed that if 90% of a colony was exposed to nectar of a treated oilseed crop each day, and these levels of foraging mortality occurred, "populations would follow a marked decline during the blooming period, and would hardly recover afterwards."

Releasing treated bees in a familiar area only 70 meters (230 ft) from the hive still led to excess forager mortality—about 6% of them did not return. Field impact studies often put hives immediately adjacent to treated fields to assess effects. This study shows that this method would tend to underestimate pesticide induced foraging impairment (Henry et al. 2012).

Bumble Bees also Affected

Although the latest research does not definitely establish a unique link between neonicotinoids and colony collapse disorder, it does show that neonicotinoids can have detrimental effects on bees at realistic field concentrations. Bumble bees, native bees, and honey bees are all at risk (Hopwood et al. 2012; Quarles 2008b). Several studies have shown that field concentrations in pollen and nectar from seed treatments on average are in the range 0.7-10 ppb. Concentrations as high as 88 ppb have been found in corn pollen (Krupke et al. 2012).

Sublethal doses of IMD have been shown to affect bumble bee foraging. After 9 days of foraging in sunflowers treated with IMD, about 10% more bumble bees were lost in the field compared to bumble bee foragers in untreated fields (Taséi et al. 2001).

Whitehorn et al. (2012) fed 25 bumble bee colonies in the laboratory for 14 days on pollen containing 6 ppb imidacloprid and sugar water containing 0.7 ppb. Exposures of this sort would be obtained if bees foraged mostly on treated fields, and rarely sought alternate food sources. Another 25 colonies received food containing twice this concentration and another 25 were fed untreated food. Colonies were then left to forage in the field for six weeks.

After six weeks, treated colonies weighed 8-12% less than untreated controls. This amount represents a combined drop in weight of food stores, wax, immature and adult bees. The weight drop was likely due to pesticide induced impairment of food gathering efficiency.

Treated colonies also had on average about 85% fewer queens (13.72 vs 1.7), probably because bumble bee queen production is dependent on colony size. According to Whitehorn et al. "our results suggest that trace levels of neonicotinoid pesticides can have strong negative consequence for queen production by bumble bee colonies under realistic field conditions, and this is likely to have a substantial population level impact."

Summer Bees, Winter Bees

There are two kinds of adult honey bees—summer bees that have a relatively short lifetime (40 days), and adult winter bees that live for 6 months or more. Summer bees gather food and feed the larvae that will develop into adult winter bees. Winter bees emerge September through November, and are responsible for colony overwintering, sometimes in very cold situations (see Quarles 2008a).

Most of the massive bee kills in the U.S. are occurring during overwintering. Large numbers of foragers collect nectar and pollen during the summer. Foraging kills a lot of them, and colony numbers drop in the fall. A smaller colony overwinters, then queens start laying eggs in late December, and the colony starts to expand in January (Winston 1987; Langstroth 1923; Morse 1975).

Adult winter bees are old bees, and are physiologically different from summer bees. Because of their relatively long lifetime, winter bees have had more time to be exposed to pesticides and pathogens. Winter bees are often more susceptible to pesticides. This may be because they have greater fat deposits, allowing pesticides to accumulate. For instance, winter bees are 4x

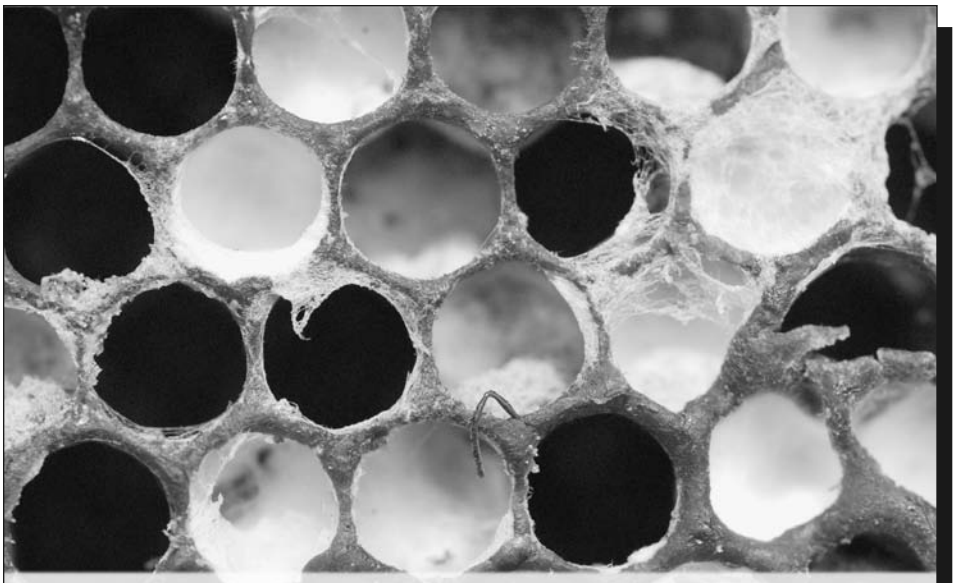

Photo courtesy of Kathy Keatley Garvey

Closeup of a hive killed by Colony Collapse Disorder. Note that brood is present, but all adult bees have disappeared.

more sensitive to the chronic lethal effects of imidacloprid than are summer bees. Cold temperatures also make pesticides more toxic to bees (Decourtye et al. 2003; Johansen 1975; Belzunces et al. 2001b).

Summer Bees Poisoned, Winter Bees Die

Most of the bee toxicity experiments are done either on individual bees or on hives monitored for a limited amount of time. Lu et al. (2012) chronically dosed summer bees with imidacloprid, then stopped. Mortality was delayed for several months. Bees were fed imidacloprid in high fructose corn syrup for about three months (13 weeks), starting July 1. Very low concentrations were used for one month, then amounts likely to cause damage were fed for two months. High fructose corn syrup containing 20, 40, 200, and 400 ppb imidacloprid were fed to the bees. Treatment was applied from July 1 to September 30.

After treatment, bees were allowed unhindered foraging until mid December, when overwintering colonies were given supplemental food. All colonies were still alive 12 weeks after the last dose was given (December 22), but hives receiving the largest dose were showing some toxic effects. However, 23 weeks (March 10, 2011) after the last dose of imidacloprid, 15 of 16 of the treated hives were dead.

Dead hives had no bees, but still had food. Summer bees were fed imidacloprid, and the winter bees died. This kind of delayed mortality mimics some of the manifestations of Colony Collapse Disorder. The lowest feeding dose was 20 ppb. Earlier experiments had shown no effect on overwintering bees when summer bees were fed 5 ppb of imidacloprid in sugar syrup. There were four untreated control hives, and three of four survived (Faucon et al. 2005; Lu et al. 2012).

Since this experiment mimics some of the manifestations of Colony Collapse Disorder, Lu et al. (2012) hypothesize that bee keepers may have produced CCD by feeding overwintering bees with corn syrup laced with imidacloprid. Imidacloprid is used extensively on corn, and relatively high tolerances (50 ppm) are permitted. However, the researchers provide no evidence that high fructose corn syrup is contaminated with IMD. Further research is needed to confirm these results and to check field samples of corn syrup for pesticide contamination.

Conclusion

Honey bees receive widespread exposure to pesticides. Large numbers of different pesticides accumulate in stored pollen and in wax combs. Large numbers increase the likelihood of synergism. Sublethal concentrations known to affect bee health and behavior have been found in many bee hives.

Pesticide exposure is a likely contributing factor to colony collapse disorder. Pesticides can depress the bees' immune system, interfere with normal brood development, and lead to poor nutrition through impaired foraging. Sublethal doses can shorten lifespan, and make bees more susceptible to mites and pathogens. Effects can be subtle, as bees poisoned in one generation may not show effects until the next generation appears.

Though bees are being impacted by a large number of pesticides, neonicotinoids are receiving increased attention. Widespread use of seed treatments, foliar sprays, and soil drenches are exposing bees to these potent pesticides over a large area. Careless use of planting machines is contaminating water, soil, and wild plants near treated fields, and exposing bees to lethal airborne seed waste and talc.

William Quarles, Ph.D., is an IPM Specialist, Executive Director of the Bio-Integral Resource Center (BIRC), and Managing Editor of the IPM Practitioner. He can be reached by email, birc@igc.org.

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