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Pesticides and Honey Bee Colony Collapse Disorder

By William Quarles

About one-third of all the food we eat requires animal pollination, and most of our fruits, vegetables, and nuts are pollinated by bees. Due to increased development, pesticides, and habitat destruction, native bees that pollinated many of our crops are in decline (Buchmann and Nabhan 1996). Crop production in the U.S. has shifted to large monocultures, pollinated almost entirely by commercially managed colonies of the honey bee, *Apis mellifera*. Large beekeepers transport thousands of bee colonies long distances to provide crop pollination throughout the U.S. (USHR 2007; 2008; NAS 2007).

Large numbers of bees are needed for pollination, and available colonies are overworked. About half the honey bees in the U.S. are needed just to pollinate the 650,000 acre (263,000 ha) California almond crop. Honey bees are trucked like migrant workers into California for the almond crop, then to Washington and Oregon for apples, to Florida for citrus, and into the Northeast for blueberries. In transit, bees are fed poor diets of corn syrup and soy proteins. Pollination of diverse crops at a large number of locations increases exposure to pesticides, mites, and diseases (Delaplane and Mayer 2000; USHR 2007; Cannell 2008; Covina 2007).

Honey bee pollination is big business, and its crop value has been estimated at $14.6 billion. If the value of animals fed on forage crops are added to the estimate, honey bee pollination is worth about $19 billion each year (NAS 2007; Morse and Calderone 2000; Losey and Vaughn 2006).

Photo courtesy of Kathy Keatley Garvey

University of California, Davis researcher and beekeeper Michael "Kim" Fondrk is shown tending bees in the Roy Gill almond orchard, Dixon, California. The boxes are beehives, and more than a million of these colonies are needed to pollinate California almonds.

Decline of the Honey Bee

Despite our dependence on honey bees, we have lost about 45% of them over the past 60 years. According to the USDA, there were 5.9 million colonies in 1947 and about 2.4 million today. However, since 1985, beekeepers with fewer than five colonies have not been counted. This change results in an undercount of about 0.86 million colonies each year. So we have about 3.3 million colonies today, and have lost at least 45% of our bees (NAS 2007).

This 45% reduction in honey bee colonies has been a slow decline marked at points by disaster. Disasters include widespread honey bee deaths due to pesticides, mites, diseases, and recently, colony collapse disorder (see below).

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Update

The first round of major losses was due to the careless use of pesticides, development, loss of forage, and other factors. Honey bee colonies declined from 5.9 million in 1947 to 4.1 million in 1972—a loss of about 30%. This first crisis was mentioned by Rachel Carson in *Silent Spring* (NAS 2007; Carson 1962). In California alone, pesticides killed about one million bee colonies between 1966 and 1979. Large numbers of bees were killed by organochlorine, carbamate, pyrethroid, and organophosphate pesticides. In the 1970s, farmers adjusted application methods to help protect bees. Efforts were made to restrict pesticide applications while crops are in bloom, and during times when honey bees are actively foraging. However, residual pesticides have undoubtedly kept bees under stress (NAS 2007; Atkins 1992; Johansen 1977; Morse 1975).

Mites and Pesticides

Disaster struck again in the 1990s. The weakened bees were attacked starting in 1984 by the trachael mite, *Acarapis woodi*, and by the "vampire mite" *Varroa destructor* in 1987. Tracheal mites interfere with bee respiration. Varroa mites are lethal to a colony because they interfere with reproduction and carry pathogens that infect and destroy the bees. Bee larvae and pupae are parasitized, and those that live to become adults are weakened. They are starved, showing low weight, low serum proteins, severe wing deformations, and reduced longevity. Varroa infestations are fatal to a colony within six months if untreated, and honey bee colony losses of 30-80% were seen in some states in 1995-1996 (NAS 2007; Sammataro et al. 2000; Benjamin and McCallum 2008).

The mites were bad, the reaction also bad. Beekeepers treated mite infestations with pesticides introduced directly into the hive. First, the pyrethroid tau-fluvalinate (Apistan®), then the organophosphate coumaphos (CheckMite®) were used to control the parasitic mites. Exposure to these pesticides depresses bee immune systems and makes them even more susceptible to varroa transmitted pathogens (Desneux et al. 2007; Morse 1975). The mite-pesticide combination has caused a drop from 4.2 million honey bee colonies in 1981 to 2.4 million colonies in 2005. This is a decline of about 43% using raw USDA data. When this figure is corrected for a change in USDA counting methodology in 1985, the estimate of colony losses due to pesticides and mites is 22% (NAS 2007).

Pesticide Resistance

Pesticide treatments were effective for a time, but varroa mites are now resistant to both fluvalinate and coumaphos. Replacement pesticides, including biocontrol fungi have been studied, but have not been widely adopted. Many experts believe that IPM management, including resistant queens may be necessary in the future (Baxter et al. 1998; Delaplane et al. 2006; Oliver 2007; Elzen and Westervelt 2002; NAS 2007).

Other stresses contributing to U.S. honey bee decline include invasion of Africanized bees in 1990, pathogens such as *Nosema* and *Paenibacillus larvae*, and pests such as the small hive beetle, *Aethina tumida*, and the wax moth, *Galleria melonella* (NAS 2007; Quarles 1994).

Massive Losses of Bees

In the last ten years, we have seen large, dramatic losses of honey bees both in the U.S. and elsewhere. Massive honey bee deaths have been seen in France, Germany, Belgium, and the UK. Problems were first noticed in France in 1994. Beekeepers there blamed the sudden deaths on Gaucho®, a new systemic pesticide with the active ingredient imidacloprid. Gaucho was used to protect sunflower seeds against pests. Treated seeds grow into sunflowers containing imidacloprid in flowers (8 ppb), pollen (3 ppb) and nectar (1.9 ppb). Perhaps coincidentally, the French honey bee population

started to crash as the treated sunflowers began to bloom and nectar began to flow. The manufacturer denied that there was any relationship between seed treatments and deaths of the bees (Bonmatin et al. 2005; Schmuck et al. 2001).

Bees in France had definitely been exposed to pesticides. One study used traps to collect pollen samples at 125 widely separated colonies throughout the country. They analyzed for 36 different pesticides and found residues of 19, including fipronil, imidacloprid, carbaryl, aldicarb, cypermethrin, chlorpyrifos and others. Pesticides found in largest concentrations were coumaphos and tau-fluvalinate—the chemicals used to treat varroa mite infestations. The most frequent residues found were imidacloprid (49.4%), imidacloprid metabolites (44.4%), and fipronil (12.4%). Imidacloprid or its metabolites were found in 69% of the samples. Pollen was contaminated with 1-5 pesticides. The contamination was seasonal only with fipronil, with fipronil maxima in March and April (Chauzat et al. 2006).

French Pesticide Ban

Pressure from the French beekeeping industry led to a ban on the use of imidacloprid on sunflowers and corn, but honey bees continued to die. Finally, in 2004 France also banned the pesticide Regent®, which has the active ingredient fipronil. According to Schacker (2008), the bees started to recover in 2005 and even larger numbers were seen in 2006.

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). Another pesticide, fipronil, is also potent with an oral LD50 of 3.7 to 6 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).

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).

Photo courtesy of Kathy Keatley Garvey

A honey bee is headed toward an almond blossom. Honey bee pollination is essential for the California almond crop.

Large Losses in the U.S.

Large losses have also been seen in the U.S. According to a National Academy of Science report (NAS 2007), "During the winter of 1995-1996, northern U.S. beekeepers experienced their largest losses in history; in some states, 30 to 80% of colonies were lost. Similar losses were observed in the winters of 2000-2001 and 2004-2005. Data on colony losses are derived from informal surveys of beekeepers, and the exact causes of colony deaths have not been established." Possible causes were a combination of pesticides, mites, and pathogens (NAS 2007).

Even larger losses have been seen in the last two years. About 33% of the managed honey bee colonies in the U.S. were lost in the winter of 2006-2007. Another 36% were lost during the winter of 2007-2008. Some of the loss was due to overwintering stress, but many colonies that died showed a strange, new behavior. Entomologists studying these large losses called the phenomenon colony collapse disorder (CCD) (USHR 2007; 2008)

Colony Collapse Disorder

Colony collapse disorder (CCD) was first noticed in November of 2006, although it may have started about two years earlier. Normally, a large number of bee colonies, perhaps 15-25%, die due to overwintering stress, including starvation. During the winter of 2006-2007, some beekeeping operations lost 30-90% of their colonies, and average overall losses of about 33% were about 10-15% larger than usual (USHR 2007; Bee 2007; Henderson et al. 2007).

Adult bees were not dying in their hives or near their hives, they simply disappeared. They left and never came back. Beekeepers in Pennsylvania with CCD lost an average of 75% of their colonies in 2006, while those free of CCD lost about 25% (Bee 2007). Losses from CCD also occurred in the winter of 2007-2008, when about 36% of all bee colonies were lost (USHR 2008).

Symptoms of CCD include:

•Foraging adult bees leave the hive and do not return.

•Decline is rapid, and the colony goes from a large, strong colony with no signs of mite infestation to a dead one within a couple of months.

•No dead bees are found in, or near the hive.

•Adult bees left in the hive show signs of a depressed immune system.

•Queen and apparently healthy brood remain in the hive.

•Nearby bees wait weeks before they enter the hive to remove food.

•Predators such as the small hive beetle or the wax moth also delay entry.

•When a dead hive is placed on top of a healthy one, so that the healthy bees are forced to occupy the hive, those bees also disappear (USHR 2007; 2008; USDA 2008; Hayes 2007).

Extent of the Problem

According to an online National Bee Survey through June of 2007, CCD is widespread throughout the country, and has been reported in at least 35 states. Both large commercial beekeepers that transport their bees from state to state and small non-migratory beekeepers are reporting the problem (USHR 2007). Both large and small beekeepers are reporting severe losses, but operations with 1,000-10,000 colonies are reporting extreme losses more often. These are beekeepers that focus on migratory crop pollination. Their bees are exposed to transportation stress, and are more likely to encounter pesticides (Bee 2007).

Is it Real?

Hard data about honey bees is hard to get. The USDA counts the number of honey producing colonies every year. However, this estimate is somewhat unreliable because colonies used only for pollination are not counted. Starting in 1985, beekeepers that maintain fewer than five colonies have not been included in the count. Since colonies are moved from state to state, colonies are sometimes counted more than once. Furthermore, health of honey bee colonies is not monitored, and bees are not systematically checked for exposure to pesticides and pathogens (NAS 2007).

Most of the information about CCD has been obtained from case studies of individual beekeepers, by online surveys completed by beekeepers (625 of them), and by surveys conducted by trade magazines.

Photo courtesy of Kathy Keatley Garvey

Closeup of a hive suffering from colony collapse disorder. Note that brood is present, but that adult bees have disappeared.

This information is not comprehensive and may not reflect the total extent of the problem. Though comprehensive data is not available, it is clear that very large numbers of bees are dying in the U.S. (Bee 2007).

Colony Replacement

If we had not replaced them, we would have lost 55% of our honey bee colonies over the last two years. Fortunately, colonies are replaced in the spring either by ordering "package bees" from suppliers or by splitting existing colonies and adding new queens. All the replacement activity is expensive, and the numbers of U.S. bees are limited. In 2005, U.S. beekeepers were forced to import bees for the first time since 1922. Over 100,000 colonies were imported from Australia. Increased expense has forced many beekeepers out of business, and has driven a steady downward trend in the number of colonies. The overall loss of 45% of our bees since 1947 has occurred despite constant colony replacements (NAS 2007).

At the same time that honey bee numbers are decreasing, planted acreage requiring pollination has increased. Increasing need plus decreased supply has resulted in higher prices for pollination. In 2007, the average rental cost for a bee colony for almond pollination was $150, about double what it was the year before (USHR 2007).

Effect of the Season

Most of the massive bee kills in the U.S. are occurring during overwintering. This is not too surprising due to the basic biology of the honey bee. Honey bees live in perennial colonies, but the nature of the colony changes with the seasons (see Box A Honey Bee Biology). Queens live 1-3 years, worker bees live about 6 weeks in the summer and 6 months during the fall and winter. 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 more sensitive to the chronic lethal effects of imidacloprid than are summer bees. Cold temperatures also make pyrethroids and organophosphates more toxic to bees (Decourtye et al. 2003; Johansen 1975; Belzunces et al. 2001b).

In Northern states, overwintering bees must cluster for warmth, and eat massive amounts of stored honey. Consumption ranges from 30-80 lbs (13.6-36.3 kg) per hive (Farrar 1952). Clustering bees rarely defecate, so any pesticide residue in the honey could build up in their bodies (Morse 1975; Langstroth 1923). They are exposed all winter to any pesticides in the honeycomb from mite treatments. Unprecedented amounts of fluvalinate, up to 400 ppm, have been found in CCD hives (USHR 2008). Winter colonies are also smaller, typically 10,000 workers versus 40,000 workers found in the summer. Since there are fewer bees in the winter, loss of smaller numbers can lead to colony collapse (Morse 1975; Winston 1987).

Because of the problems of overwintering in cold climates, many beekeepers transport their bees to warmer climates to overwinter. These colonies are allowed to forage and are sometimes fed corn syrup or sucrose solutions. Even these pampered bees are still exposed to any toxins or pathogens present in the hive, and CCD was actually first observed in colonies transported from Pennsylvania to overwinter in Florida (USHR 2007).

According to an online survey of 625 beekeepers, most of them do not feed their overwintering bees. But providing food does not stop CCD. When colonies are fed sucrose syrup, about half these colonies still get CCD. Feeding of this sort does not stop ingestion of stored pollen and exposures to pesticides in wax and honeycomb (see below) (Bee 2007).

What is Causing CCD?

The exact cause of CCD has not yet been determined. A CCD task force has been established, and a number of possibilities are being investigated (USDA 2008). Pathogens are known to kill honey bees, and pathogens are being investigated as a cause of CCD. Many of the colonies are infected with Israeli Acute Paralysis Virus (IAPV). However, this virus is believed to be a marker, not a cause. Also, this pathogen does not fit the specifics of the disorder. Bees dying from pathogens are thrown out of the hive by housekeeping bees, and dead bees accumulate in front of the hive. Certainly mites will kill bees, but again deaths from mites are well characterized. CCD colonies start out healthy and mite free. Surviving bees do not show mite infestations (USHR 2007; Schacker 2008).

Pesticides as a Cause of CCD

The surviving bees are showing signs of stress. Pesticides and other chemicals are known to kill bees and depress their immune systems (Morse 1975; Desneux et al. 2007). Many currently used pesticides such as imidacloprid, clothianidin, fipronil, chlorpyrifos and others are extremely toxic to bees. Pesticides can also exert sublethal effects that include disorientation and loss of short term memory that may prevent bees from returning to the hive. So pesticide exposure and pesticide contamination of the hive could explain most of the symptoms of CCD (USHR 2007; Decourtye et al. 2003; Suchail et al. 2001ab; Chauzat et al. 2006). One observation that seems to implicate pesticides is that organic beekeepers do not seem to have CCD (Schacker 2008).

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).

Pesticides in the Hive

Despite the importance of honey bees, colony health and pesticide exposure has not been systematically monitored, and EPA registration of a pesticide does not require a measurement of sublethal effects on honey bees (NAS 2007). Pesticide contamination of bees is only now being systematically studied. Researchers at Penn State University analyzed bees, pollen, and wax honey combs for pesticide residues in 2008. Some of the samples came from CCD colonies, others came from colonies that showed no symptoms. All of the bees tested were carrying at least one pesticide. A total of 108 pollen samples showed residues of 56 pesticides and metabolites. Each pollen sample averaged 6 pesticides, and one sample showed 31. In 88 wax samples, residues of 22 pesticides and metabolites were found. The miticides coumaphos and fluvalinate were found in all wax samples. 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; PA 2008; USHR 2008; Pilling and Jepson 1993; Schmuck et al. 2003).

According to David Mendes, Vice President of the American Bee-

Box A. Biology of the Honey Bee

A beehive is dynamic, with rapid population turnover, especially in the summer. A summer hive has typically 40-45% of its population as eggs, larvae and pupae. About 55-60% are adult workers that live about 6 weeks, spending their first 3 weeks working in the hive, and then 3 weeks foraging. It takes about 3 weeks for an egg to become an adult, so as the foraging population dies, it is replaced by hive workers, and hive workers are replaced by new adults. Every 3 weeks in the summer there is a new foraging population, and every 6 weeks there is a completely new generation of adults (Wilson 2004).

Numbers of honey bees in colonies vary by the season. A winter colony has about 10,000 workers, a summer colony 40,000. Foragers feed on nectar and collect pollen. The pollen is made into bee bread, which is a combination of pollen, nectar and enzymes. This is used to feed larvae. Up to 100 kg (220 lbs) of nectar is collected by individual foragers, about 50 mg at a time. This is made into about 30 kg (66 lbs) of honey, which is used as food for overwintering (Kievits 2007).

The fate of the colony depends on the workers. Workers in the summer live 15-38 days; spring and fall 30-60 days. Winter bees live an average of 140 days, but as long as a year. Bees drink large amounts of water. They collect water, propolis, which is resinous glue used for construction, nectar, which is used to make honey; and pollen, which is especially used to feed developing larvae. Nectar is 5-80% sugars, mostly sucrose, fructose and glucose. Foragers transfer nectar to nurse bees that add enzymes, reduce the water content to less than 18%, then store it in the honey comb as a food source for the whole colony. Most of the honey bee nutrition comes from pollen, which contains protein, fat, vitamins, minerals and steroids (Winston 1987).

Life Stages

Life stages include eggs, larvae, pupae and adults. Adults include males (drones), female workers, and a queen. The queen starts laying eggs in December. Over a period of 3 days the egg membrane slowly dissolves, revealing the larva. Larvae are fed by nurse bees a combination of glandular secretions and honey over the first four days, and larvae molt daily. Food is often just dumped into the cell and larvae are exposed to the food by contact and ingestion. Larvae are fed hundreds of times by nurse bees. Pollen is fed starting on the 3rd day. By the 6th day of the larval stage and 9 days after egglaying, the cell is capped. Over a period of 3-5 days after the cell is capped, larvae stand upright in the cell, defecate and spin a cocoon for the pupal stage. The pupal stage lasts about 8-9 days, then the pupae molts, becoming an adult. The process for development of a new worker takes about 21 days (Winston 1987).

Adults eat honey and pollen. Honey is obtained from storage or by begging and trophallaxis. Lack of pollen and protein during the first 10 days of adult life reduces life span. Some pollens are more nutritious than others, but bees must obtain 10 essential amino acids to continue living (Winston 1987).

Hives and Swarms

Wild honey bees swarm with a queen looking for a nest. When they find an appropriate space, they start building the waxy honey comb. Hexagonal cells for worker brood and honey are the same size, drone cells are larger; queens are raised in thimble sized appendages to the comb. Comb construction takes about 45 days. Several combs are constructed, spaced about 0.95 cm (3/8 in) apart. Combs are glued to the nest site with bee glue or propolis, which is also used to cement cracks, and insulate the nesting cavity (Winston 1987).

Domestic beehives are based on the Langstroth principle. Wooden frames containing beeswax or plastic hexagonal cell templates are suspended in a wooden box about 0.95 cm (3/8 in) apart. These boxes are called supers, and they can be stacked vertically. Supers are placed on a wooden foundation, and the top super has one or two wooden lids. This simple arrangement makes it easy to move the hives around. Honey is produced in the top supers, brood in the lower ones. Honey laden frames can be removed, uncapped with a knife, and honey is removed with a centrifuge. Frames are reused for years (Langstroth 1923; Winston 1987).

Age and Work

Jobs inside the hive are done by young adults, older adults act as guards and foragers. Hive jobs include first cleaning, then acting as nurses, food processing and comb building. These jobs are age related because they rely on glandular development. Nurses are about 7-13 days old. Adults start foraging when they are about 23 days old. Variations are seen in these ages according to complex factors and the needs of the colony. When there are large colony losses, workers start foraging at younger ages (Winston 1987).

Bees spend very roughly 10-15 days collecting nectar; 15-20 days collecting pollen; 30 days eating pollen; about 30 days processing and storing nectar; and about 10 days feeding larvae. So adults are exposed to nectar for 45 days, to pollen about 50 days of their adult lives. Finally, there is extensive trophallaxis so that food collected by a few bees is distributed throughout a colony within 24 hours (Winston 1987)

Foraging Behavior

Honey bees specialize in their foraging. They tend to collect either nectar or pollen from a single species of flowers until the food source is depleted. For instance, one study showed 58% of foragers collected only nectar, 25% only pollen, and 17% both nectar and pollen. Good nectar producers are maple, milkweed, phacelia, sage, thyme, acacia and figwort. Colonies need 15-30 kg (33-66 lbs) of pollen every year, but may collect up to 55 kg (121 lbs). Colonies need 60-80 kg (132-176 lbs) of honey each year. Workers must make one to four million trips a year to manage this supply. Relatively few resources are used for scouting. A few scouts find a rich resource, and communicate its location through a dance that recruits large numbers of foragers (Winston 1987)

keeping Federation, 18 of his hives were used in the above study. Samples of pollen from Florida citrus showed high levels of imidacloprid and aldicarb. In Massachusetts cranberries, fungicide levels as high as 7000 ppb were found in pollen. Only four hives were still alive 10 months later (USHR 2008).

Are Systemic Pesticides Causing CCD?

Though many pesticides can stress bees, some U.S. beekeepers believe that CCD is caused by bee exposures to new pesticides, such as imidacloprid (IMD), clothianidin, fipronil, and others (USHR 2007). For instance, imidacloprid is used extensively in the U.S. on crops such as blueberries, citrus, cranberries, strawberries, pecans, stone fruits, cotton, corn, melons, vegetables, forests, ornamentals, and turf. Some of these are crops commercially pollinated by bees. According to David Hackenberg, the beekeeper who discovered CCD, "beekeepers that have been most affected so far have been close to corn, cotton, soybeans, canola, sunflowers, apples, vine crops, and pumpkins. So what is it about these crops that are killing the bees?"(USHR 2007).

Hackenberg's idea is that foragers "may bring pollen and nectar back to the hive and store it in their comb to use later. It is usually several months later when natural sources of pollen and nectar slow down in the field that the bees would use this store of pollen and nectar to raise brood that the symptoms appear....What may finally kill the hive are two things: first, the loss of most of the adult bees because when sick bees leave the hive to collect food they do not return (disappearing disease) and second the remaining young bees in the hive may have such a weakened immune system that normal pathogens found in the hive such as fungus easily overwhelm them" (USHR 2007).

The best way to test this hypothesis is with nationwide monitoring of bee colonies. A number of migratory colonies should be monitored through a season, and levels of pesticides checked on a regular basis. If the colony starts to collapse, and significant pesticide residues are found in bee bread and honey, then the hypothesis is confirmed. As mentioned above, these kinds of experiments are now underway (USDA 2008; USHR 2008).

Photo courtesy of Kathy Keatley Garvey

Susan Cobey, University of California bee breeder and geneticist, is holding a "frame" of healthy bees. Each beehive contains several of these frames.

We already know that pesticide contamination of bee food can occur. Bayer researchers fed honey bees sugar syrup containing 10 ppb IMD, and the resulting honey contained 5-8 ppb (Schmuck et al. 2001). Wallner et al. (1999) and Wallner (2001) exposed honey bees to *Phacelia tanacetifolia* that had been treated with IMD. Concentration in collected nectar ranged from 3-10 ppb, and similar concentrations were found in bee bread.

Field Levels of Imidacloprid

Imidacloprid can be applied as a seed treatment, a soil drench, or as a foliar spray. According to the California EPA, where IMD is being used, models suggest expected concentrations in surface water of 17 ppb, and 2 ppb is expected in groundwater. When IMD treated seeds are planted, seed residue can be blown offsite by planting equipment. Residues on plants near a crop site can be 14-54 ppb (Fossen 2006). 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. Leaves of sugar beets can have 15.2 ppm (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.

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).

Imidacloprid is used extensively in California, and was the 6th most commonly used insecticide (162,254 lbs) according to acreage (788,402 acres) in 2005. It is used on citrus, almonds, and other crops pollinated by bees. Large numbers of CCD colonies have been found in CA (DPR 2006; Bee 2007; Henderson et al. 2007).

According to Schacker (2008), colony collapse disorder is not seen in states such as Nevada where imi-

Box B. Toxicity of Imidacloprid

Exposure to a lethal dose of imidacloprid causes immediate excitation, including trembling and tumbling. After several hours, workers slow down and become inactive. Death occurs 4 to 96 hours after poisoning (Suchail et al. 2001ab; 2003). There is a 100 fold variation in acute toxicity according to the kind of bee and the season. The LD50 has been measured at 3.7 ng/bee in bees from the UK, and 40.9 ng/bee for German bees. The acute oral LD50 is somewhere between 3.7 and >80 ng/bee. The LD50 by contact is 81 ng/bee. This large variation in sensitivity could explain why some colonies die from CCD and others do not (Decourtye et al. 2003; Suchail et al. 2001ab; Schmuck et al. 2001).

Imidacloprid is metabolized by bees into toxic metabolites. Bees metabolize 50 µg/kg (5 ng/bee) acute doses of IMD quickly, the half life is about 4.5 hrs, and it is completely gone after 24 hours. The toxic metabolites peak about 4 hours after oral ingestion of IMD. One of these metabolites, an olefin derivative, is twice as toxic to bees as IMD. Since death occurs 4-96 hours after exposure, mortality may be due mostly to metabolites (Suchail et al. 2003; 2004).

Chronic Toxicity

Toxicity accumulates, so repeated feeding of doses lower than the LD50 is also lethal. Large variations have been seen in chronic toxicity (Suchail et al. 2001ab). Decourtye et al. (2004) found mortality occurred at imidacloprid feeding concentrations in the range 24-48 ppb after 11 days. Bayer researchers found no effect on bee mortality at feeding concentrations of 20 ppb (Schmuck et al. 2001). Suchail et al. (2001ab) found very high chronic toxicity for imidacloprid. Half of bees fed sugar solutions of 0.1 to 10 ppb imidacloprid over a 10 day period died. Concentration levels of imidacloprid similar to this are found in nectar and pollen of plants receiving IMD seed treatment. The discrepancy between the work of Suchail and others may be due to the "large variability in effects induced by imidacloprid." Suchail also used a feeding solution that contained dimethylsulfoxide (DMSO) that might have acted as a synergist. There is known synergism between imidacloprid and fungicides, and maybe DMSO is also a synergist.

Sublethal Effects

Concerns have also been raised about sublethal effects. Various experiments have shown that learning in bees can be confounded by pesticides. Homing flight durations, food intake, odor learning, and bee to bee communication can be affected (Pham-Delegue et al. 2002; Colin et al. 2001; Bortolotti et al. 2003; Medrzycki et al. 2003; Colin et al. 2004; Desneux et al. 2007). Concentrations as low as 6 ppb of IMD and 2 ppb of fipronil have caused observed sublethal effects (Colin et al. 2004).

For instance, doses of deltamethrin 27x lower than the LD50 caused 80% of foragers to lose their way back to the hive (Colin et al. 2001a). 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).

Curé et al. (2001) found that the no effect level of imidacloprid in sugar syrup was 20 ppb. Kirchner (1999) found that a dose above 20 ppb, "causes not only a reduction in the foraging activity of treated bees, but also induces trembling dances that discourage other worker bees from foraging. The waggle dance that communicates foraging direction becomes less precise."

Decourtye et al. (2004) found that 24 ppb IMD reduced honey bee numbers foraging at a feeder, and reduced syrup intake by a factor of three. This concentration also reduced olfactory learning, which correlates with finding food supplies.

Sublethal doses of 6 ppb may cause bees to eat less. Colin et al. (2004) found that 6 ppb concentrations of IMD did not reduce the number of foragers at a sugar feeder, but fewer foragers ingested the contaminated syrup. If honey bees have an aversion to eating their food stores, resulting starvation and forced foraging in wintertime could manifest as CCD.

Synergism

Toxic, chronic and sublethal thresholds can be lowered by synergism. Toxic effects of pyrethroids, neonicotinoids such as IMD, and fungicides are synergistic (Brobyn 2001; Schmuck et al. 2003; USHR 2007; Iwasa et al. 2004).

dacloprid is not used. This observation, however, does not prove that imidacloprid causes CCD, because those states also have few crops to pollinate, and few migratory bees.

Can Field Levels of IMD Harm Bees?

Some bees are extremely sensitive to IMD, and acute toxicity levels can vary by a factor of 100 (Schmuck et al. 2001; Suchail et al. 2001ab) (see Box B). This fact could explain why some colonies get CCD, while others in the same area do not (Hayes 2007). Reasonable estimates of bee IMD exposure through pollen and nectar in treated fields are 3 to 10 ppb (Bonmatin et al. 2005; Wallner et al. 1999). Toxicity is cumulative, and one experiment showed 50% mortality when bees were fed 0.1 to 10 ppb IMD for 10 days (Suchail et al. 2001ab). (See Box B.)

Other experiments have shown that mortality starts at feeding levels between 24-48 ppb (Decourtye et al. 2004). Bees probably do not receive this level of exposure in treated fields. However, other pesticides and especially fungicides have been shown to lower toxic thresholds through synergism (Johansen 1977; Pilling and Jepson 1993; USHR 2007; Schmuck et al. 2003).

Sublethal effects such as reduced feeding have been found at levels of

6 ppb. Reduced feeding could lead to poor nutrition, which could contribute to CCD. Longterm feeding of 4-40 ppb impairs olfactory learning, which is associated with finding food (Decourtye et al. 2001; 2004). Other sublethal effects on foraging start at 24 ppb (Kirchner 1999). Again, pesticide synergism could lower the toxic thresholds.

Because bees can mix pollen from treated plants with that from untreated plants, exposure depends on percent of food obtained from treated crops. But bee recruitment tends to concentrate foragers from the same hive on the same plants (see Box A). If migratory bees are pollinating a treated crop, much of the collected pollen should be contaminated (Bonmatin et al. 2005). The situation is complicated because bee exposure varies according to life stage and job tasks. Despite the complications, French researchers believe that concentrations of IMD found in the field are large enough to put bees at risk (Halm et al. 2006; Rortais et al. 2005; Bonmatin et al. 2005).

Conclusion

Colony collapse disorder is probably caused by stress, and pesticides are definitely one of those stresses. Preliminary data suggest that CCD hives are contaminated with a number of pesticides, including imidacloprid. Imidacloprid has profound effects on honey bee mortality and behavior. Toxic thresholds are low, and can be lowered further through synergism with other pesticides. Bees show a wide range of sensitivity, and this fact could explain why some colonies collapse, others do not. Though the evidence is suggestive, it will take a nationwide monitoring program to confirm or deny the role of pesticides in colony collapse disorder.

If colony collapse disorder has taught us anything, it is to take better care of our bees. The EPA should require sublethal toxicity tests. Bee colonies should be systematically monitored on a regular basis for diseases and pesticide exposures. Beekeepers should avoid toxic miticides and switch to IPM methods to control mites. These methods are more labor intensive, but pesticide resistance can be minimized.

If we do not take better care of our bees, there could be a significant impact on crop production. Some foods could become scarce and expensive. We should also treat our bees better because they are our friends, they enrich our planet, and it is the right thing to do.

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ESA 2007 Annual Meeting Highlights—Part 5

By Joel Grossman

These Conference Highlights are from the Dec. 9-12, 2007, Entomological Society of America (ESA) annual meeting in San Diego, California. ESA's next annual meeting is November 16-19, 2008, in Reno, Nevada. For more information contact the ESA (10001 Derekwood Lane, Suite 100, Lanham, MD 20706; 301/731-4535; http://www.entsoc.org).

Poinsettia IPM Ecology

"Competition among herbivores can impact pest management practices, thus it is important to understand these relationships for successful integration of management tactics," said Claudia Kuniyoshi (Ohio State Univ, 1680 Madison Ave, Wooster, OH 44691; kuniyoshi.1@osu.edu). In poinsettias there is an interaction between aboveground pests such as silverleaf whitefly, *Bemisia argentifolii*,and belowground pests such as darkwinged fungus gnats, *Bradysia impatiens*. There are also interactions from high nitrogen fertilizer levels.

At high nitrogen levels, whitefly survival is higher than at low nitrogen levels. High nitrogen levels also encourage fungus gnat populations, and whitefly egg laying increases. Eventually, whitefly populations suppress belowground fungus gnat populations. These ecological effects may in part be caused by pest modification of plant quality.

Pumpkin IPM

Jim Jasinski (Ohio State Univ, 1512 S US Hwy 68, Ste B100, Urbana, OH 43078; jasinski.4@osu.edu) talked about a survey of pumpkin growers in Indiana, Illinois, Ohio, Michigan, Minnesota, Wisconsin and Ontario, Canada. About 52% called disease control the number one management problem; 18% said weeds; and 14% insects. Among the IPM practices adopted: 73% scout for plant diseases; 76% cultivate for weed control until the vines close the row; 65% monitor for squash bugs; and 84% of growers apply pesticide sprays in early morning or evening to reduce impacts on honey bees.

Pastoral Beneficials

"Pastured dairy and beef cattle provide a critical link in the food supply system by utilizing marginal land," said Phillip Kaufman (Univ of Florida, PO Box 110620, Gainesville, FL 32611; pkaufman@ifas.ufl.edu). Pastured systems can provide positive environmental impacts to watersheds and nutrient recycling. Dung burying beetles play a valuable role in pasture ecosystem sustainability by reducing pasture fouling, nitrogen volatilization, parasitism and pest flies, with averted losses estimated at $0.38 billion.

"Current active IPM efforts for pastured cattle include monitoring animals for populations of harmful fly pests, and preemptive use of cultural and chemical controls," said Kaufman. "Augmentative biological control of pasture pests is in its infancy."

In a baited pitfall trap survey in Florida, the major dung beetle species stabilizing pasture ecosystems, accounting for 26% of the beetles collected, included brown dung beetle, *Onthophagus gazella* ;bull-headed dung beetle, *O. taurus;* and *Euoniticellus intermedius*.

Camp Ant IPM

In South Carolina state parks, where the Argentine ant, *Linepithema humile*, is invading campsites, tents, public facilities and recreational vehicles, "approximately 70% of the campers used their own pesticides to treat for *L. humile*," said Brittany Russ (Clemson Univ, 114 Long Hall, Clemson, SC 29634; brittar@clemson.edu). "Campers were typically found to be over-treating their camping areas or were proactively treating areas against potential ant infestations." Despite the *ad hoc* insecticide spray and dust onslaught, the number of ant trails remained constant in state parks, indicating "a need for better control strategies."

"Establishing baiting areas by park

Everett "Deke" Dietrick

Everett "Deke" Dietrick, entomologist and pioneer in the field of biological pest control, died at his home in Ventura on December 23. His scientific training in entomology and his boundless interest in insect ecology on farms led him to collaborate in founding Rincon-Vitova Insectaries. Through his encouragement and advice many hundreds of farmer clients rejected conventional chemical control and transitioned to biological control methods.

After returning from the war in 1947, he eagerly took an opening with the UC Statewide Department of Biological Control, led by one of his most cherished mentors, Professor Harry Smith. Twelve years of university field research observing insect ecology, pesticide resistance, and the phenomenal success of classical biocontrol projects led him to believe that chemical pesticides usually cause more problems than they solve.

He left the university to pioneer the new profession of pest control advisor. In partnership with Ernest "Stubby" Green, and then later Jack Blehm, he established insectaries in Ventura and Riverside to grow various insect predators and parasites for commercial use against crop pests. The two companies merged in 1971 to form Rincon-Vitova Insectaries, Inc. now owned and managed by daughter Jan Dietrick.

Deke was a Board Certified Entomologist and an Emeritus Member of the Entomological Society of America. In 1972 he served as an expert witness at Congressional hearings in Washington, DC that led to the banning of DDT in the United States.

For over 40 years Deke mentored scores of individuals who wanted to be part of his work. He labored to teach people in letters, articles, talks, and papers (some of which are posted at www.rinconvitova. com/dietrick_papers.htm). Interviews of Deke between 1994 and 1997 can be found at www.rinconvitova.com/ dietrick_interviews.htm. He was increasingly hopeful in the last several years, seeing the rise in environmental awareness and the growth of the organic industry.

Expressions of remembrance and support for Deke and his work may be directed in the form of tax-deductible donations to the Dietrick Institute for Applied Insect Ecology, www.dietrick.org, a non-profit organization offering training in ecologically based pest management, PO Box 2506, Ventura, CA 93002.

Calendar

December 4-6, 2008. Acres USA Eco-Farm Conference. St. Louis, MO. Contact: 800/355-5313; www.acresusa.com

December 5-6, 2008. Sustainable Agriculture Pest Management Conference. San Luis Obispo, CA. Contact: CCOF, 831/423-2263; www.ccof.org

December 8-11, 2008. Annual Meeting, North Central Weed Science Soc. Indianapolis, IN. Contact: 217/352-4212; www.ncwss.org

January 19-23, 2009. 21st Annual Advanced Landscape IPM Short Course. College Park, MD. Contact: U. Maryland, 301/405-3913 Ex 3911; www.raupplab.umd.edu/conferences/adv-landscape

January 21-24, 2009. Annual Ecological Farming Conference: United We Grow. Asilomar, CA. Contact: www.eco-farm.org

January 24, 2009. 32nd Annual Bay Area Environmental Education Resource Fair. San Rafael, CA. Contact: K. Hanley, 510/657-4847; www.baeerfair.org

January 28, 2009. 11th Annual San Francisco IPM Conference. Presidio, San Francisco. Contact: SF Dept. of Environment, 11 Grove St., San Francisco, CA 94102; 415/355-3776; jessian.choy@sfgov.org

February 10-12, 2009. Pacific Northwest Sustainable Agriculture Conference. Richland, WA. Contact: KCCD, 6-7 E. Mountain View Avenue, Ellensburg, WA 98962; 509/525-3389.

February 24, 2009. 10th Annual Organic Turf Trade Show. Farmingdale, NY. Contact: Neighborhood Network, 7180 Republic Airport, East Farmingdale, NY 11735; 631/963-5454, www.neighborhood-network.org

February 26-28, 2009. 20th Annual Organic Farming Conference. La Crosse, WI. Contact: MOSES, PO Box 339, Spring Valley, WI 54767; www.mosesorganic.org

March 1-3, 2009. California Small Farm Conference. Sacramento, CA. Contact: www.californiafarmconference.com

March 24-26, 2009. Sixth International IPM Symposium. Portland, OR. Contact: Tom Green, IPM Institute, 608/232-1410; www.ipm-centers.org/IPMsymposium09/

December 13-17, 2009. Entomological Society of America Annual Meeting. Indianapolis, IN. Contact: ESA, 9301 Annapolis Road, Lanham, MD 20706; Fax 301/731-4538; www.entsoc.org

Conference Notes

personnel for foraging *L. humile* early in the camping season may be beneficial for quelling larger infestations from occurring as the camping season progresses," said Russ. "We expect the improved control measures to decrease the number of complaints by campers about *L. humile* and reduce the misuse or overuse of cleaning powders, insecticide powders and broadspectrum insecticide sprays used by visitors and park personnel."

Ozone Hive Fumigation

"Ozone (O3) is a powerful oxidant that has the ability to disinfect, eliminate odors, taste, and color, and remove pesticides," said Rosalind James (USDA-ARS, 5310 Old Main Hill, Logan, UT 84322; rosalind.james@ars.usda.gov). "Ozone can potentially be used to treat old comb and bee equipment. It is already used as an agricultural fumigant for stored products. O3 also breaks down quickly to H20 (water) and O2 (molecular oxygen), so it will not persist in wax comb, wood, or plastic."

Ozone fumigation can potentially eliminate pests of old honeycomb such as greater wax moth, *Galleria mellonella*, which creates a large amount of webbing as it feeds on old comb, plus pesticides such as coumaphos (Checkmite™) and fluvalinate (Apistan™) that persist in hives after varroa mite treatment. The prototype ozone generator (O3Zone Company, ID) fed 500-1,000 ppm ozone in one side of a gas incubator and out the other side, creating 215-430 ppm O3 inside the test chamber.

At 77-95°F (25-35°C), ozone fumigation degraded coumaphos faster than fluvalinate. Though pesticide degradation products need to be tested, decontamination of low pesticide levels should take no more than 13 hours. Small wax moth larvae were killed in 1-2 hours by the lowest O3 concentrations. Wax moth adults were killed within 6 hours. Eggs were most resistant, requiring 48 hours at 33.7°C (93°F) for complete kill. "One method to avoid the long exposure times required to kill all the eggs may be to treat twice," said James. "Once to kill adults and any larvae or pupae that may be present, and then a second short treatment 5-6 days later to kill the newly emerged larvae that hatched from any eggs that survived the first treatment."

Woody Plant Termite Reservoirs

Formosan subterranean termites, *Coptotermes formosanus*, have caused an estimated $300 million in damages in the city of New Orleans from 1966 to 1996, and $500 million in damage in the state of Louisiana. An areawide IPM program using bait stations and non-repellent termiticides was begun in New Orleans' French Quarter in 1998. "Trees and woody plants may be reservoirs for Formosan subterranean termites to infest structures," said Dennis Ring (Louisana State Univ, 404 Life Sci Bldg, Baton Rouge, LA 70803; dring@agctr.lsu.edu). Thus, trees and woody plants were inspected visually and using an acoustical probe.

A total of 1,026 properties on 35 city blocks with 1,873 trees and woody plants were inspected. First, "aerial photographs of each block of the French Quarter in the test area were obtained," said Ring. "Then visual inspections were made to locate trees by walking blocks. Property owners were contacted and appointments were made to inspect trees. Inspections consisted of visually inspecting around the trunks of trees for termites, mud tubes or packs of soil. An acoustical probe was used to detect termites in trees. The soil around trees was probed with the distance between probes no more than 12 in (30 cm) apart because 12 in is the upper limit to the effectiveness of the acoustical probe."

The termite infestation rate for trees was about 2% (17 properties; 39 trees). Pest management professionals treated 13 of the infested properties with bait stations and 4 with liquid termiticides. Only one tree was reinfested and retreated. "Trees and woody plants infested by termites will be re-inspected periodically and infested trees will be retreated," said Ring. "Continued inspection of trees and woody plants in other blocks included in the program are ongoing."

Predatory Mite Sugar Supplements

"Sugary supplements (M-30; 30% solution of 1:1 fructose & glucose) enhance *Phytoseiulus persimilis* survival and fecundity even in the presence of prey," said Guadalupe Rojas (USDA-ARS, 59 Lee Rd, Stoneville, MS 38776; grojas@msa-stoneville.ars.usda.gov). "The presence or absence of extra floral nectaries may significantly impact the performance of *P. persimilis* in the field. Mass rearing of the predator may be enhanced by providing sugary supplements in addition to prey."

Globalization of Woodboring Beetles

"Wood and wood products used to support, brace or package commodities

Conference Notes

during shipment provide the major pathway for global transport of woodboring beetles, a well-known group that causes significant ecological and economic impacts to forests," said Charles Bellamy (CDFA, 3294 Meadowview Rd, Sacramento, CA 95832; cbellamy@cdfa.ca.gov). "Annual losses to forest-derived products attributed to introductions of non-native insect species into the U.S. are about $2.1 billion per year," and "as globalization continues we are likely to see an increase in introductions of non-native organisms."

"Approximately 93% of all insects intercepted on wood articles at U.S. ports of entry between 1985 and 1998 were beetles, making an urgent case for developing diagnostic tools associated with this large group of insects," said Bellamy. "Because of the potential catastrophic damage that could be caused by the introduction and spread of non-native invasive wood-boring beetle taxa, it is of no surprise that an identification resource is the most frequently requested tool by Plant Pest Quarantine."

Lucid3 (CBIT, Univ of Queensland, Australia; www.lucidcentral.org), a Java-based program running on varied operating systems (e.g. Windows, Macintosh, LINUX), is being used to build a series of woodboring beetle identification keys for scientists, the general public and quarantine and plant protection personnel.

Biocontrol Aromas

"Herbivore-induced plant volatiles have been shown to attract natural enemies," said Christina Harris (Penn State Univ, 121 Chem Ecol Lab, Orchard Rd, University Park, PA 16802; cmh347@psu.edu). On DPL90 cotton, feeding of cabbage looper, *Trichoplusia ni,* but not beet armyworm, *Spodoptera exigua*, induces cotton plants to release beta-farnesene.

Beet armyworm (BAW) feeding, but not cabbage looper (CL) feeding, induces the release of the sesquiterpene volatiles alpha-humulene and gamma-bisabolene. "Terpene emission was quantitatively higher for BAW-infested relative to CL-damaged and intact plants," said Harris. "These findings may explain field observations that CL is more heavily parasitized by the generalist parasitoid wasp *Cotesia marginiventris* in the presence of BAW."

Hotel Bed Bugs

"Generally commercial clients, hotels in particular, have a great understanding of the liability issues associated with bed bugs," and their cooperation with pest control efforts is above average, said Judith Black (Steritech Group, Inc, 5742 W 114th Pl, Broomfield, CO 80020; judy.black@steritech.com).

"The process we go through is an extremely thorough inspection and treatment" using basic detection tools like bright flashlights and putty knives, said Black. "We are ripping the room apart, taking off outlet covers, taking pictures off the walls, taking down curtain rods, all that kind of thing. We are basically dismantling the room. We are having them quarantine linens, and then get them laundered separately. We do ask that the mattress and box spring be disposed of." Encasement of old mattresses and box springs could potentially harbor live bed bugs and present a liability issue. But encasements are recommended when installing new beds, to avoid future replacement.

Within hotels, the number of treated rooms ranged from none to 23%; the average was 2.5%. The total number of rooms treated for bed bugs went from 7 in 2003 to over 700 rooms in 293 hotels in 2007. Over the five year period, over 60% of the 700 mid-range hotels were treated for bed bugs, though over 99% of the 75,000 rooms did not have bed bug problems in any given year. About half the time, a hotel with a problem called back only once or twice, indicating bed bugs were not being reintroduced. However, 8% called more than 10 times.

About 20% of the time, the "secondary rooms" which are above, below and beside the infested "primary" room also were infested; the former secondary room becomes a primary room if an infestation is found, and then a new set of secondary rooms are inspected. About 70% of the time the infested secondary room is next door to the primary room; 10% of the time it is above, and 18% of the time it is below.

"Bed bugs have legs" and they use wall voids to move within a facility. When moving from the primary room, bed bugs typically move into the adjacent secondary room sharing the wall with the bed headboard. In 2006 retreatments went up dramatically, and there were many field reports of pyrethroid resistance. So, in 2007 a switch was made from synthetic pyrethroid-based liquid residuals and dusts to non-synthetic pyrethroid products and encasements were introduced; and the number of retreatments decreased.

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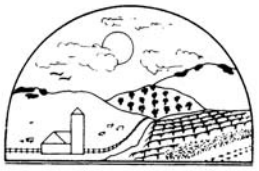

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