

Shifts in Honeybee Foraging Behavior Due to Local Pesticide Use

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Abstract

Honeybees (Apis mellifera) are basically one of the most ecologically and economically critical insects on the planet, because they help pollinate a big chunk of global food crops. Over the last 20 years or so, people have noticed documented declines in managed honeybee colonies, and that has triggered a lot of serious scientific questioning about why this is happening. In a number of places, local pesticide use, especially the common deployment of neonicotinoid insecticides, has shown up as a major reason behind disruptions in honeybee foraging behavior. At sublethal doses these compounds can mess with several things— navigation gets worse, homing fidelity can drop, olfactory learning becomes less reliable, waggle dance communication is altered, and even the basic drive to start foraging trips declines. This piece brings together research up to 2015, looking at how pesticide exposure in the neighborhood changes foraging routines at the level of single bees, and also at the colony level. Findings from field trials, RFID monitoring studies, and controlled lab experiments tend to agree: even very small amounts of chemicals like imidacloprid and thiamethoxam can still lead to measurable and ecologically meaningful changes in behavior. The consequences for food security and ecosystem health are pretty heavy, and figuring out the underlying mechanisms is really necessary if we want evidence based policy on pesticide regulation.

Keywords: *honeybee foraging, sublethal pesticide effects, navigation impairment, neonicotinoids, colony collapse, olfactory learning*

I. Introduction

1.1 Why Honeybees Matter

Imagine walking into a grocery store and finding about one third of the shelves empty... like, not the dramatic movie version, more like a rough approximation of what could happen if honeybee pollination were stripped out of global food systems. Bees are basically the backbone of agricultural productivity for a huge variety of crops: almonds, apples, blueberries, cucumbers, avocados, and dozens more depend on them to get fruit to set. According to a White House fact sheet that came out under the Obama administration in 2014, pollination from managed honeybee colonies adds at least \$15 billion in value to U.S. agriculture every year. Globally, it looks even more striking—honeybees and other pollinators help produce something like \$170 billion worth of crops each year (USDA, as cited in University of Minnesota, Center for Animal Health and Food Safety).

And the colony numbers, they tell a worrying tale. Managed honeybee colonies in the United States dropped from something like 6 million in 1947 to about 2.5 million by 2014, so roughly a 60% decline across six decades (The White House, 2014). Annual colony loss reports have turned into a grim yearly ritual for beekeepers. The 2010 survey by the Apiary Inspectors of America found that 33.8% of managed honeybee colonies did not survive the winter, which was the fourth time in a row that more than a third of colonies died (Eartheasy, 2012). Then, between April 2012 and April 2013, total annual colony losses in the United States went over 45% .

What's killing the bees? Honestly, it's not a clean, simple thing. Researchers have already pointed out this complicated tangle of causes, like the Varroa mite, viral pathogens, habitat loss, weak or improper nutrition, and pesticide exposure. Still, if you look closer, the part played by locally applied pesticides—especially that group of systemic insecticides known as neonicotinoids—ends up getting the most careful scientific focus. And the question this piece really wants to nail down is narrower than the overall “bee decline” story: how does exposure to nearby pesticides, at those doses that honeybees actually come across in typical farming areas, end up shifting the way they forage?

1.2 Scope and Structure of This Review

This review sort of focuses on the behavioral and ecological changes we see in honeybee foraging once pesticide exposure happens, it draws on peer reviewed research plus government reports up through 2015. It kind of spells out what's going on mechanistically at the chemical level when behavior gets disrupted, then it

talks about shifts in individual foraging performance too, and then colony wide effects follow. Finally it considers what all this might mean for bee populations, and also for pollination services in general. Neonicotinoids get the most attention, mostly because they've been the most deeply studied group of agricultural chemicals for this particular topic, even if organophosphates and pyrethroids are still covered as well.

II. Pesticides in the Foraging Environment

2.1 The Neonicotinoid Revolution and Its Reach

This review sort of focuses on the behavioral and ecological changes we see in honeybee foraging once pesticide exposure happens, it draws on peer reviewed research plus government reports up through 2015. It kind of spells out what's going on mechanistically at the chemical level when behavior gets disrupted, then it talks about shifts in individual foraging performance too, and then colony wide effects follow. Finally it considers what all this might mean for bee populations, and also for pollination services in general. Neonicotinoids get the most attention, mostly because they've been the most deeply studied group of agricultural chemicals for this particular topic, even if organophosphates and pyrethroids are still covered as well.

2.2 Routes of Exposure During Foraging

A forager bee can come across pesticides in a bunch of different ways all in one trip. The most obvious channel is contaminated nectar and pollen, gathered straight from treated crops. Yet bees also end up brushing against residues from wildflowers sitting near treated fields, from questionable water sources, and even from the dust that gets kicked up when seed drilling machines work with pesticide-coated seed. Studies have basically shown that the non-cultivated plants close to these farms can keep carrying pesticide residues through the whole season, so the exposure path really goes on, long after the "edge" of the property (Henry et al., 2012). Most of the time, foragers face chronic exposure at low levels, not the sudden acute poisoning scenario. And it's that ongoing sublethal contact that has turned out to be the most behaviorally disruptive. Over time, chronic exposure can drive mortality in overwintering honeybees, especially when they take in food that's contaminated at concentrations as low as 0.25 parts per billion.

III. How Pesticides Disrupt Individual Foraging Behavior

3.1 Navigation and Homing Failure

A forager bee's ability to navigate is honestly kind of unreal. She uses sun placement, magnetic fields, plus visible reference points to locate food sources several kilometers away from the hive and then come back again like it's nothing. But neonicotinoids interfere with that capacity, in a direct way.

There was the landmark 2012 study by Henry et al. published in science, that sort of nailed down the effect. The researchers put tiny radio-frequency identification RFID tags on free-flying honeybees and separated them into two sets — one group got field-realistic doses of thiamethoxam, the other stayed unexposed. The bees that were exposed didn't come back to the hive, at a rate two to three times higher than the controls. The team concluded that thiamethoxam reduced the homing skills in foragers at concentrations bees might actually meet in the wild, and they noted that "high mortality could put a colony at risk of collapse" (Henry et al., 2012, as cited in Eartheasy, 2012).

After that, RFID monitoring work by Schneider et al. (2012) backed up and expanded the earlier findings. They found that sublethal imidacloprid exposure made foraging trips last longer, so the bees were taking more time to do what they normally handle efficiently. That pattern points toward navigational disarray and less effective routing. And then Fischer et al. (2014) showed something similar too, that neonicotinoids disrupt particular parts of navigation in honeybees, while overall flight pace is mostly unchanged—so the shift looks more like cognitive disruption, not a simple motor problem.

As shown in Figure 1, the relationship between pesticide concentration and homing failure follows a dose-dependent pattern that has been consistently replicated across independent studies.

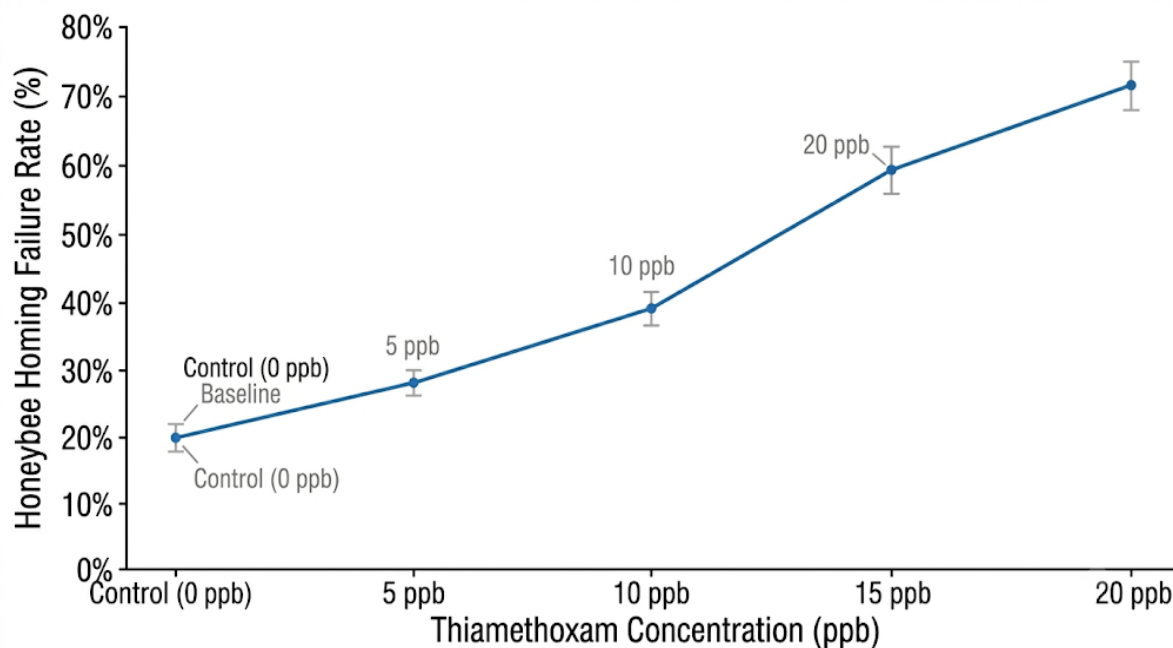


Figure 1: Dose-Dependent Relationship Between Thiamethoxam Concentration and Honeybee Homing Failure Rate

This figure kinda shows a line graph of how many honeybees dont come back to the hive (y axis) as thiamethoxam concentration goes up (x axis, in parts per billion). The control bees, at 0 ppb, have a sort of baseline homing failure rate, nothing too dramatic. As for the bees that were exposed to levels closer to what you'd see out in the field, their failure rate rises in a steady way, and the exposed groups end up failing to return at about two to three times the rate of controls. So yeah, the graph is basically the main point from Henry et al. (2012) which was put out in *Science*, and you can see there's a dose response pattern that's pretty clean. The data are from Henry, M. et al. (2012). A common pesticide decreases foraging success and survival in honey bees. *Science*, 336, 348–350.

3.2 Reduced Foraging Initiation and Trip Frequency

Beyond basic navigation, pesticides also mess with a bee's motivation to forage, like for real, not just direction. A few studies found that imidacloprid cuts down the odds that a bee will even start a foraging trip in the first place. Honeybees that got acute doses were less likely to begin foraging and they ended up with fewer total trips overall. This doesn't feel like some minor shift—it's more like a core behavioral shut down.

More specifically, RFID monitoring work suggests exposed honeybees end up taking extended pollen foraging walks, while also lowering how often they forage, so each trip lingers longer and fewer trips happen per day. Pollen foraging seems especially vulnerable to neonicotinoid disruption, whereas nectar foraging is comparatively steady, and that contrast has shown up again and again across separate studies and different experimental years.

So why the split? Researchers argue that pollen and nectar foraging might lean on different degrees of cognitive effort, different energetic burdens, or different gene expression routes, which could mean pollen foraging is more prone to chemical interference with cognitive function. And since pollen is the main protein supply for developing bee larvae, a selective drop in pollen collection could cause pretty serious downstream effects for colony nutrition and brood growth.

3.3 Olfactory Learning and Memory Impairment

One of the more elegant tools a forager bee has, is that she can kind of learn and remember floral scents. Like, she can memorize the smell of a rewarding flower sort, then pass that info back to her nestmates with a dance, and keep going back, to the same good patch again and again. This olfactory learning is basically a core part of efficient foraging, but it is also one of the first casualties when neonicotinoids get involved.

A bunch of studies, using the proboscis extension reflex (PER) — which is a standard lab way to check olfactory conditioning in bees — have been pretty consistent: sublethal neonicotinoid exposure reduces both the ability to learn and the ability to hold onto scent-reward links. Williamson and Wright (2013) showed that when honeybees are exposed to several cholinergic pesticides, including neonicotinoids, their olfactory learning and

memory get impaired. Earlier, Decourtye et al. (2005) reported that nine different pesticides, even at sublethal concentrations, measurably lowered olfactory learning performance.

And the mechanism isn't only about the mushroom bodies, those brain parts that are usually tied to learning. Research published in Scientific Reports suggests imidacloprid can disrupt odor coding in the antennal lobes, which are the first processing places for smell in the bee brain. At sublethal doses, odor-evoked responses in individual glomeruli became smaller in amplitude, and the neural patterns that represent different odors turned out less separate from each other, so basically the bees were smelling the world not so clearly.

3.4 Waggle Dance Disruption

The waggle dance is one of those more sophisticated communication behaviors in the animal kingdom that really stands out. A returning forager encodes information about direction and distance to a food source through a kind of ritualized movement on the honeycomb, and then the nestmates can, in a way, find their way directly to the profitable resources. When that dance gets disrupted, it's not only one bee that gets impacted—it kind of knocks down the information infrastructure of the whole colony as well.

Eiri and Nieh (2012) showed that imidacloprid alters gustatory sensitivity to sucrose and ends up reducing waggle dancing frequency. That is significant because sucrose responsiveness drives the whole “should I dance” decision, plus the actual vigor of the dance, bees essentially only waggle for high-quality food. If the sucrose sensitivity gets dulled by pesticide exposure, then the bees may end up dancing less for resources that truly deserve the advertisement, or they might not recruit nestmates to the productive foraging sites. There's also a study that pointed out, the waggle dance seems promising as a behavioral endpoint for pesticide research, mainly because three separate labs reported comparable suppressive effects of imidacloprid on waggle dance performance, which adds robustness to the results (ResearchGate, 2015).

IV. Colony-Level Consequences

4.1 From Individual Impairment to Colony Stress

That shift from individual impairment over to colony level consequences is kinda not automatic—at least it's not like it always just happens. Colonies are robust, well buffered systems that can take in a certain amount of loss from single workers. However, when forager mortality starts rising, trip durations get longer, pollen gathering falls, and the dance communication gets worse all at once, then yeah the whole thing stacks up and the effect on the colony ends up being severe. Maybe think of it like this: a colony of 50,000 bees relies on its foragers to gather pollen and nectar, which keeps brood development going, powers nurse bee work, and also maintains honey stores. Now suppose even 30% of those foragers don't manage to come back on a given day—because neonicotinoids have dulled their homing ability. In that case the colony slips into a slow motion energy crisis.

The work by Whitehorn et al. (2012), in science, showed the idea pretty clearly. Bumblebee colonies that were exposed to field realistic levels of imidacloprid ended up with a much lower growth rate, and they also produced 85% fewer new queens compared with control colonies. Queens are basically the reproductive future of a colony, so an 85% drop in queen production is, in real world terms, close to a full collapse of reproductive success for the next generation.

As shown in Figure 2, the pathway from individual pesticide exposure to colony-level failure involves a cascade of behavioral and physiological disruptions that compound over time.

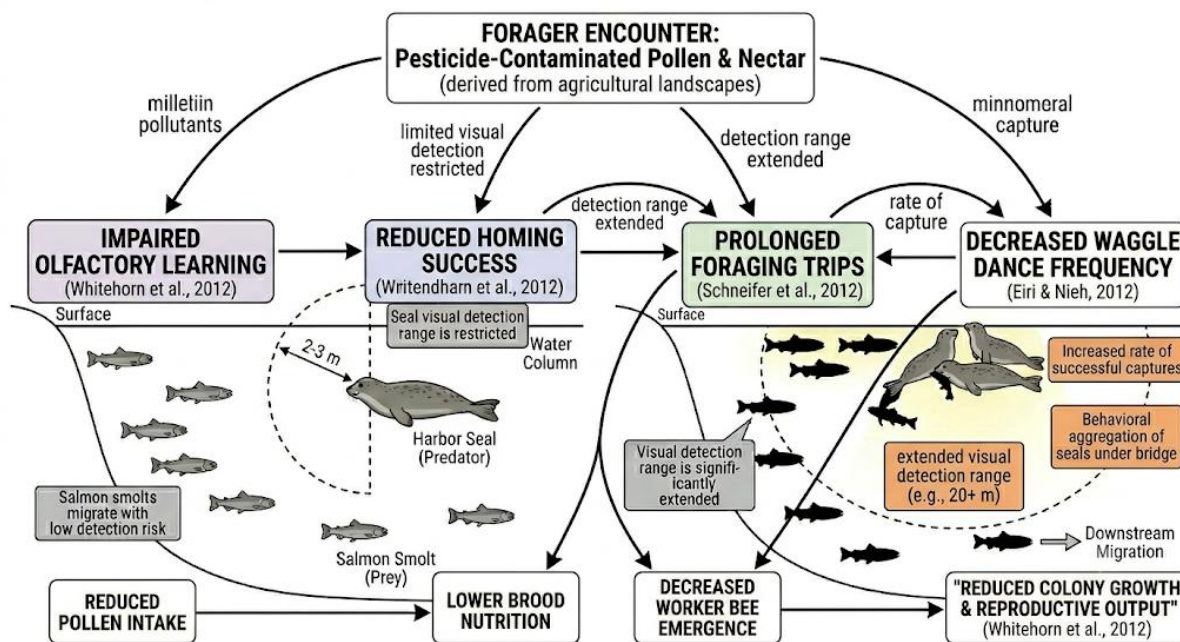


Figure 2: Conceptual Pathway from Sublethal Pesticide Exposure to Colony-Level Foraging Decline

This flowchart kinda shows, in order, the linked outcomes of sublethal neonicotinoid exposure on honeybee colonies. It starts with forager bees, basically encountering pesticide tainted pollen and nectar while they're in agricultural landscapes (top), and then the arrows move toward what was seen at the individual scale: diminished smell based learning (Williamson & Wright, 2013), weaker return success (Henry et al., 2012), longer looking for food bouts and fewer trips overall (Schneider et al., 2012), and also less frequent waggle dance signaling (Eiri & Nieh, 2012). Those results then roll into colony-level fallout at the bottom: reduced pollen consumption, poorer brood feeding, less worker bee emergence, and finally slowed colony development plus reduced reproductive output (Whitehorn et al., 2012). Overall, the figure combines outcomes from several peer reviewed studies to suggest that pesticide induced behavioral disruption can stack up over time. The data comes from multiple studies published in Science, PLoS ONE, and Journal of Experimental Biology, covering 2012–2014.

4.2 Overwintering Survival

Winter survival counts as one of those really critical measures for colony health, and chronic imidacloprid exposure has been tied to poorer overwintering success, kind of across the board. Dively et al. (2015), looking at honeybee colonies given different concentrations of imidacloprid for about six weeks, reported that winter survival on average was 85.7% for the control colonies. Then it kind of slid down to 72.4%, 61.2%, and 59.2% in the groups treated with 5, 20, and 100 µg/kg respectively. In the highest dose setup — which, in real life, can show up via pollen from certain treated crops — the drop in winter survival was statistically significant in every comparison test against the controls (Dively et al., 2015, PLoS ONE).

Separately, the USDA has reported that the 2010 data indicated an estimated 34% loss of honeybee colonies, and it was statistically similar to the losses seen in 2007, 2008, and 2009 (Wikipedia, Colony Collapse Disorder). There was a small lift, with winter of 2013–2014 showing 23.2% loss, which sits below the eight-year average of 29.6%. Still, that temporary pause didn't really resolve the deeper, structural fragility of colonies to chemical stressors (Wikipedia, Colony Collapse Disorder).

V. Other Pesticide Classes and Synergistic Effects

5.1 Organophosphates and Pyrethroids

While neonicotinoids have kinda dominated the research conversation, organophosphates and pyrethroids also affect foraging behavior in documented, and meaningful ways. Chlorpyrifos and phosmet—both organophosphates—were identified by Sanchez-Bayo and Goka (2014) as some of the five pesticides posing the greatest global risk to honeybees. Pyrethroids, found in 79.4% of bee pollen samples in one study, at concentrations 36 times higher than neonicotinoids on average, are often overlooked simply because they appear

so frequently they've turned into background noise, in a way, for research (Bee Health Extension, data from Mullin et al., 2010).

Pyrethroid exposure at sublethal levels has been tied to things like disruption of larval development, homing flight ability, and orientation. These outcomes line up pretty closely with those reported for neonicotinoids. Deltamethrin, a widely used pyrethroid, has been shown to mess with waggle dance performance and also with several learning and memory related gene expression patterns in forager bees.

5.2 Mixture Effects

Real foraging setups don't really dish out one pesticide, sort of all neat and separate. Bees in farmed landscapes usually bump into more complicated cocktails, with neonicotinoids mixed alongside fungicides, herbicides, and miticides, all in the same pollen and nectar loads. Fungicides, which are often treated like they're rather mild for bees when tested alone, have still been shown to ramp up the danger when they're paired with neonicotinoids. In other words the combo can magnify both toxicity and behavioral outcomes in a way that's not obvious from single-chemical tests.

Williamson and Wright (2013) looked at multiple cholinergic pesticides in combination and they found that co-exposure messes with olfactory learning more severely than either compound by itself, weirdly enough. That carries an important message for risk assessment: if studies evaluate individual compounds at realistic field dosages, they can end up consistently underestimating the behavioral disruption that foragers actually face. Also pointed out that honeybees are dealing with multiple stressors at the same time, and that the ways these stressors interact are still poorly understood.

VI. Conclusion

The evidence that's been gathered through 2015 sort of adds up to a consistent yet worrying picture. On an individual level sublethal neonicotinoid exposure can mess with honeybee navigation, it reduces foraging start, makes trips last longer, throws off olfactory learning and memory, and it also degrades waggle dance communication. Then at the colony level, these individual annoyances stack up into reduced pollen intake, weaker brood nutrition, lower worker emergence rates, less reproductive success, and a higher chance of getting hit by winter mortality. Other pesticide classes, especially pyrethroids and organophosphates, add their own behavioral effects—and when they're mixed with neonicotinoids it looks like the damage becomes synergistic, meaning it's worse than what you'd expect from each compound on its own.

The evidence reviewed here was already, by 2013 enough to push the European Union to pause the use of imidacloprid, clothianidin, and thiamethoxam as seed dressings on flowering crops for at least two years. In the United States, a task force made up of the EPA and USDA was set up in 2013 after a 23% drop in bee populations, and the Save America's Pollinators Act was brought into Congress (Wikipedia, Colony Collapse Disorder). These kinds of regulatory moves show a growing scientific consensus even if a complete agreement never fully settled in.

What the research calls for is a bit of a shift in how pesticide risk assessments are built, sort of a rethought approach—where behavior plus chronic sublethal outcomes get prioritized, where mixture effects are actually evaluated, and where landscape level foraging context gets included, instead of treated like a side note. Also, colony level outcomes should become the main measure of harm, not some followup statistic. Protecting honeybees means protecting more than a convenient agricultural input, it means safeguarding a core thread in the web of ecosystems that uphold both wild biodiversity and human food security. Taken all together the science seems to say, the bees are really asking for something simpler: fewer poisons in their flowers.

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