

No Longer a **BIG** Mystery

**Recent scientific research confirms
the role of pesticides in pollinator decline**



"The apple trees were coming into bloom but no bees droned among the blossoms, so there was no pollination and there would be no fruit." – Rachel Carson, Silent Spring

The science has become increasingly clear that pesticides, either acting individually or synergistically, play a critical role in the ongoing decline of honey bees and wild pollinators. While studies reveal wide-ranging adverse impacts from a multitude of agents, including poor nutrition, stress, fungicides, and pathogens, the neonicotinoid class of insecticides continues to receive the greatest attention from scientists, beekeepers, and advocacy groups.

Since Beyond Pesticides first started publicizing the role of pesticides in bee decline many years ago, the organization's scientific database has identified new findings demonstrating that pesticides, especially the neonicotinoid class of insecticides, have sub-lethal and chronic impact on bee behavior, immune system, and colony longevity. Neonicotinoids are systemic pesticides, meaning once applied they translocate throughout the entire plant, including stems, and flowers. Pollinators, like honey bees, face unique threats from exposure to these systemic pesticides because they can be exposed through multiple pathways including, foliar appli-

cations, contaminated field dust, as well as through contaminated guttation droplets, pollen, and nectar. Since these pesticides are also very persistent in the environment, exposure becomes continuous, affecting multiple generations.

Some of the studies on pesticides and honey bees played a key role in the decision to invoke a two-year suspension of the neonicotinoids clothianidin, imidacloprid, and thiamethoxam in the European Union in April 2013.¹ The findings of a European Food Safety Authority (EFSA) report show that these chemicals are of "critical concern" to bee health and place honey bees and hives at "high risk."² Within the last few years, the number of studies linking the controversial neonicotinoids to pollinator decline has grown exponentially, reporting harm to bees' reproduction, mobility, and navigation, as well as impairments to feeding, foraging, memory, and learning.

While chemical industry giants, like Bayer CropScience, continue to dismiss pesticides as a concern, and instead choose to point to parasites and beekeeping practices as the cause for the ongoing pollinator crisis, the U.S. Environmental Protection Agency (EPA), as well as the U.S. Department of Agriculture (USDA) acknowledge pesticides as a contributing factor. EPA has requested long-term

field data on larval, queen, and colony health, and the agency is also currently reviewing the registrations of the neonicotinoids. This review will not be completed until 2018, while beekeepers warn that bees and other pollinators do not have that long to wait.

In the meantime, the independent, peer-reviewed, scientific data linking pesticide exposures to bee decline keeps growing. Some of the highlights follow:

Mounting Evidence of Toxicity

The science on bee health is reporting that even small, low-dose (sublethal) neonicotinoid exposures can have detrimental effects on bees. New research published in the journal *Ecotoxicology* in 2014 finds that “near infinitesimal” exposures—levels as low as 0.7ppb—to neonicotinoids causes a reduction in the amount of pollen that bumblebees are able to gather for their colony.³ The authors believe that these low concentrations are field-realistic doses. On the findings, lead author of the study, Hannah Feltham, PhD, remarked, “This work adds another piece to the jigsaw. Even near-infinitesimal doses of these neurotoxins seem to be enough to mess up the ability of bees to gather food. Given the vital importance of bumblebees as pollinators, this is surely a cause for concern.” Adding to the problem, the researchers find that bumblebees continue to underperform even a month after exposure. Another study examining field-realistic doses reports that the growth rate of exposed bumblebee colonies has been significantly reduced, and suffers an 85% reduction in production of new queens.⁴

Similarly, research by Williamson and Wright (2013)⁵ finds that sublethal doses of pesticides significantly impair important behaviors involved in foraging, implying that “pollinator population decline could be the result of a failure of neural function of bees exposed to pesticides.” In this study, the authors observed that bees exposed to imidacloprid are less likely to form long-term memory, and develop impaired olfactory learning ability. To further understand this and similar findings, Palmer, et al., (2013) conducted a study attempting to understand the neurophysiological mechanisms of these effects, and reports that neonicotinoids, specifically clothianidin and imidacloprid, interfere with neuronal signaling and inhibit nicotinic responses in the brain, leading to cognitive impairments.⁶ This study also suggests that exposures

to multiple pesticides with similar modes of action will cause enhanced neurotoxicity.

Other impacts observed include altered bee feeding and social behavior that can have long-term impacts on the hive. University of California at San Diego biologists found that honey bees treated with a small, single dose of imidacloprid, comparable to what they would receive in nectar, become “picky eaters” preferring to feed only on sweeter nectar and refusing nectars of lower sweetness that they would normally feed on, and which provide important sustenance for the colony.⁷ In addition, waggle dances, which help

bees recruit their nestmates to good food, was not frequently observed in exposed bees.

In conjunction with ongoing investigations into the role of neonicotinoids in bee health, researchers are also beginning to look at the so called “inert” ingredients of many pesticide formulations. A study released by Pennsylvania State University researchers, Ciarlo, et al., (2012), observes that bee learning behavior is impaired by exposure to

low doses of surfactants—other ingredients commonly found in pesticide formulations.⁸ Here, the researchers measured the olfactory learning ability of honey bees treated orally with sublethal doses of the most widely used spray adjuvants on almonds in the Central Valley of California. These ingredients are only now drawing the attention of scientists, and can play a part in bee toxicity.

Beyond Neonicotinoids- Synergistic Mixtures

A mixture of pesticides can have synergistic effects, meaning they become even more toxic in combination than individually. Researchers have been recording the presence of multiple pesticides, most of which have been collected in the foraged pollen. In fact, Mullin, et al., (2010) found 121 different pesticides and metabolites within a number of wax, pollen, bee, and associated hive samples.⁹ A followup study looking at four commonly detected pesticides in pollen and wax—flufenoxuron, coumaphos, chlorothalonil, and chlorpyrifos, found that exposure to these chemicals has serious consequences on bee larvae survival rates: “All pesticides at hive-residue levels triggered a significant increase in larval mortality compared to untreated larvae by over two-fold, with a strong increase after three days of exposure.”¹⁰ Here, combinations of pesticides only served to amplify mortality rates around the four day mark. The researchers found synergistic toxicity with



the mixture of chlorothalonil and fluvalinate, and the mixture of chlorothalonil and coumaphos. Adding to concerns, researchers found that the so-called “inert” ingredient, N-methyl-2-pyrrolidone, caused major damage to honey bee health. The authors stated, “Even for the lowest concentration of [this inert ingredient], the estimated time to cause 50% larval mortality was 4 days.” The study concludes that “pesticide mixtures in pollen be evaluated by adding their toxicities together.”

Increased Susceptibility to Pathogens

The causes of pollinator decline are multiple and complex: pests and diseases, diet and nutrition, genetics, pesticides, and habitat loss are all contributors to pollinator decline, according to the 2012 Congressional Research Service report on the state of bee health.¹¹ Increasingly, however, research shows that exposure to neurotoxic pesticides compromises bee immune system functioning, dramatically raising their susceptibility to pathogens and parasites. Many dead hives have not only been found with high residues of pesticides, but also high levels of disease and para-

sites. For instance, one study by researchers at USDA found an increased probability of infection from the fungal parasite *Nosema* in bees that consumed pollen with a higher fungicide load.¹² This led other researchers to start investigating whether pesticide exposures affect bees’ ability to withstand parasite infection.

Italian researchers, Di Prisco, et al., (2013), set out to find the mechanism through which pesticides can adversely impact the immune system of honey bees. Their study suggests that exposure to neonicotinoids negatively modulates immune signaling in insects, and adversely affects honey bee antiviral defenses.¹³ The authors observed that honey bee exposure to clothianidin enhances this mechanism, reducing immune defenses, and promoting the onset of deformed wing virus in honey bees. Similar results were also observed with imidacloprid.

Research at USDA’s Bee Research Laboratory (2012), led by Jeffery Pettis, PhD, finds that *Nosema* infections increased significantly in bees exposed to pesticides in their hives, demonstrating an indi-

Table 1. Partial list of key studies demonstrating the impacts of neonicotinoids and other pesticides on pollinators

Species	Authors/Date	Pesticides	Significance
Honey bee	Zhu, et al., 2014	Fluvalinate, coumaphos, chlorothalonil, chlorpyrifos	Combination of the four most common pesticides found in pollen/wax synergize, increase bee larvae mortality.
	Doublet, et al., 2014	Thiacloprid	Sublethal doses of a neonicotinoid pesticide interact with parasite <i>Nosema ceranae</i> and black queen cell virus to elevate honey bee mortality.
	Carrillo, et al., 2014	Fipronil, imidacloprid	Learning, as evaluated through proboscis (e.g. mouthparts used for feeding) extension, is diminished.
	Di Prisco, et al., 2013	Clothianidin	Altered immune response allowed replication of viral pathogens in exposed bees.
	Williamson & Wright 2013	Clothianidin, coumaphos	Long term memory, short-term memory, and odor differentiation all decrease.
	Palmer, et al., 2013	Imidacloprid, clothianidin, organophosphate miticides	Cognitive damage from exposure causes “epileptic type” hyperactivity with implications for survival.
	Matsumoto 2013	Clothianidin, dinotefuran, etofenprox, fenitrothion	Demonstrates behavioral changes and declines in homing success.
	Derecka, et al., 2013	Imidacloprid	Metabolizing genes for honeybee larvae reduce at low levels of exposure.
	Hatjina, et al., 2013	Imidacloprid	Of the few physiological studies, this finds sublethal doses decrease phyopharyngeal glands and respiratory rhythm.
Africanized honey bees	Sandrock, et al., 2013	Thiamethoxam, clothianidin	Sublethal exposure to neonicotinoids is expressed in complex fitness related ways, including a 50% reduction in offspring.
Solitary bee	Bryden, et al., 2013	Imidacloprid	Chronic sublethal stress causes bee colony failure according to models.
Bumblebee	Gill, et al., 2012	Imidacloprid, lambda-cyhalothrin	Combination of two pesticides impairs foraging, increases worker mortality, and reduces colony success.
	Whitehorn, et al., 2012	Imidacloprid	Field realistic levels drastically reduce queen production and growth rates.
Wildlife	Goulson 2013	Clothianidin, thiamethoxam, imidacloprid	Reviews the environmental risks of these chemicals to bees, birds, and beneficials.

rect effect of pesticides on pathogen growth in honey bees.¹⁴ Similarly, a French study (2011) reports that exposure to sublethal doses of pesticide results in higher mortality *Nosema*-infected honey bees than in uninfected ones.¹⁵ Alaux, et al., (2010) also report that the combination of both imidacloprid and *Nosema* caused the highest individual mortality rates and energetic stress, suggesting a synergistic interaction between these agents and, in the long-term, a higher susceptibility of the colony to pathogens.¹⁶ Also of note is the impact of pesticide exposure during the developmental stages of bees. Wu, et al., (2012) finds that a higher proportion of bees reared from high levels of pesticide contaminated brood comb became infected with *Nosema* at a younger age, compared to those reared in low residue brood combs.¹⁷

Recently, Furst, et al., (2014) state that deformed wing virus and *Nosema* could be spreading from honey bees to bumblebees, dramatically shortening the lifespan of wild bumblebees. It suggests that managed, highly-dense populations of honey bees are breeding grounds for pathogens, which may then be transmitted to wild bumblebee populations. Infected bumblebees, however, are much more affected by the disease, with their lives shortened by six full days.¹⁸

Systemic Contamination Making Broader Impact

In a comprehensive review released in 2013, Dave Goulson, PhD, provides the first overview of the widespread issue of neonicotinoid contamination, persistence, and impacts on wildlife.¹⁹ Taking data from chemical manufacturer Bayer, Dr. Goulson analyzes the persistence of neonicotinoids in soil and water. The data reveals that the soil half-life of the most commonly used neonicotinoid seed treatments can range from 200-1000 days. Clothianidin, in particular, has a half-life of 148-6,931 days (Note: Other estimates of half-life range from 148 to 1,555 days²⁰). According to Dr. Goulson, once in soil, neonicotinoids have a high propensity to leach into groundwater, streams, and ponds. For instance, one California study (2012) reports 89% of water samples taken from rivers, creeks, and drains in the state contain imidacloprid, with 19% of those samples at levels above EPA guidelines.²¹

After neonicotinoids are applied to farmland, their persistence in soil and water can cause broad and far-reaching impacts on ecosystem health, much of which is not fully studied. Dr. Goulson explains, "Any pesticide that can persist for many years, build up in soil, and leach into waterways is likely to have effects far beyond

the pest insects it intends to target. This is particularly so when the pesticide is highly toxic to non-target organisms. For example, less than one part per billion of the neonicotinoid imidacloprid in streams is enough to kill mayflies."

Krupke, et al., (2012) have also reported detecting neonicotinoids in the soil of planted fields and unplanted fields.²² Neighboring

plants, such as dandelions, which bees visit, were

also found to contain neonicotinoids. This

study finds high levels of clothianidin and thiamethoxam in planter exhaust material (field dust) produced during the planting of treated seed.

These contaminated dust plumes (also referred to as fugitive dust) can travel for miles, depositing residues far off-site. Work by both Drs. Krupke and Goulson support strong evidence that the concentration of neonicotinoids found in agricultural fields have the potential to cause catastrophic acute and sublethal impacts on honey bees and on colony level success for honey bees and bumblebees.

In addition to bees, Pierre Mineau, PhD suggests that pesticide toxicity to birds is also an important factor in grassland bird declines.

In a report released by the American Bird Conservancy (2013), Dr. Mineau finds that it takes a single corn kernel to kill a song bird and about 1/10th of a corn seed per day to impact avian reproduction.²³ This report also identifies aquatic systems as under threat from neonicotinoid contamination. According to the report, contamination levels in both surface and ground water are already beyond the threshold found to kill many aquatic invertebrates, leading to long-term impacts on aquatic food chains.

What You Can Do

As pollinator declines continue at an unprecedented rate, the time for action is now. The risk that neonicotinoids and other bee-killing pesticides pose to the stability of the global food system and the natural world warrants their permanent prohibition. Beyond Pesticides and others want the EPA to take neonicotinoid-treated seeds off the market. See www.BEEprotective.org to find out how you can help this effort and how to get your community, schools, and local government to take action to protect pollinators.

For more information, please go to www.BEEprotective.org. Drew Toher, Xoco Shinbrot, and Nichelle Harriott contributed to this piece.



References

1. European Commission. 2013. Bees & Pesticides: Commission to proceed with plan to better protect bees. European Commission - IP/13/379 Brussels. Available at http://europa.eu/rapid/press-release_IP-13-379_en.htm?locale=en
2. EFSA. 2013. Press Release: EFSA identifies risks to bees from neonicotinoids. January 16, 2013. Available at <http://www.efsa.europa.eu/en/press/news/130116.htm>
3. Feltham, H, Park, K, and Goulson, D. 2014. Field realistic doses of pesticide imidacloprid reduce bumblebee pollen foraging efficiency. *Ecotoxicology* 10 (1007).
4. Whitehorn PR, O'Connor S, Wackers FL, Goulson D. 2012. Neonicotinoid pesticide reduces bumble bee colony growth and queen production. *Science*. 336(6079):351-2.
5. Williamson, SM & GA Wright. 2013. Exposure to multiple cholinergic pesticides impairs olfactory learning and memory in honeybees. *The Journal of Experimental Biology* 217 (6).
6. Palmer, MJ, Moffat, C, Saranzewa, N, Harvey, J, Wrights, G and C Connolly. 2013. Cholinergic pesticides cause mushroom body neuronal inactivation in honeybees. *Nature Communications* 4 (1643).
7. Eiri, D.M. and Nieh, J. C. 2012. A nicotinic acetylcholine receptor agonist affects honey bee sucrose responsiveness and decreases waggle dancing. *J Exp Biol* 215:2022-2029.
8. Ciarlo TJ, Mullin CA, Frazier JL, Schmehl DR. 2012. Learning impairment in honey bees caused by agricultural spray adjuvants. *PLoS One*. 7(7):e40848.
9. Mullin CA, Frazier M, Frazier JL, et al., 2010. High levels of miticides and agrochemicals in North American apiaries: implications for honey bee health. *PLoS One*. 5(3):e9754.
10. Zhu, W, Schmehl, D, Mullin, C, and J Frazier. 2014. Four common pesticides, their mixtures and a formulation solvent in the environment have a high oral toxicity to honey bee larvae. *PLoS ONE* 9(1).
11. Schierow, L and R Johnson. 2012. Bee health: the role of pesticides. Congressional Research Service 7-5700.
12. Pettis JS, Lichtenberg EM, Andree M et al., 2013. Crop pollination exposes honey bees to pesticides which alters their susceptibility to the gut pathogen *Nosema ceranae*. *PLoS One*. 8(7):e70182.
13. Di Prisco, G, Cavaliere, V, Annoscia, D, Varricchio, P, Caprio, E, Nazzi, F, Gargiulo, G and F Pennacchio. 2013. Neonicotinoid clothianidin adversely affects insect immunity and promotes replication of a viral pathogen in honey bees. *Proceedings of the National Academy of Sciences* 110(46):18466-71.
14. Pettis JS, vanEngelsdorp D, Johnson J, Dively G. 2012. Pesticide exposure in honey bees results in increased levels of the gut pathogen *Nosema*. *Naturwissenschaften*. 99(2):153-8.
15. Vidau C, Diogon M, Aufauvre J, et al., 2011. Exposure to sublethal doses of fipronil and thiacloprid highly increases mortality of honeybees previously infected by *Nosema ceranae*. *PLoS One*. 6(6):e21550.
16. Alaux, C. et al., 2010. Interactions between *Nosema* microspores and a neonicotinoid weaken honeybees (*Apis mellifera*). *Environmental Microbiology*. 12(3):774-782.
17. Wu JY, Smart MD, et al., 2012. Honey bees (*Apis mellifera*) reared in brood combs containing high levels of pesticide residues exhibit increased susceptibility to *Nosema* (Microsporidia) infection. *J Invertebr Pathol*. 109(3):326-9.
18. Furst, MA, McMahon, D, et al., 2014. Disease associations between honeybees and bumblebees as a threat to wild pollinators. *Nature*. 506,364–366.
19. Goulson, D. 2013. Review: An overview of the environmental risks posed by neonicotinoid pesticides. *J Applied Ecology* 50(4): 977-987.
20. USEPA. 2010. Clothianidin Registration of Prosper T400 Seed Treatment on Mustard Seed (oilseed and Condiment) and Poncho/Votivo Seed Treatment on Cotton. Office of Pesticide Programs. Washington DC.
21. Starner, K. & Goh, K.S. 2012. Detection of the neonicotinoid insecticide imidacloprid in surface waters of three agricultural regions of California, USA, 2010–2011. *Bulletin of Environmental Contamination and Toxicology*. 88, 316–321.
22. Krupke CH, Hunt GJ, Eitzer BD et al., 2012. Multiple routes of pesticide exposure for honey bees living near agricultural fields. *PLoS One*. 7(1):e29268.
23. Mineau P, Whiteside M. 2013. Pesticide Acute Toxicity Is a Better Correlate of U.S. Grassland Bird Declines than Agricultural Intensification. *PLoS ONE* 8(2): e57457.