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Cover Page Footnote

Thanks to the SJEL editorial staff for their suggestions and edits.

Banning Neonicotinoids: Ban First, Ask Questions Later

Evan Jensen[†]

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I. INTRODUCTION

Recently, the world's population of bees crashed catastrophically. In North America, approximately one-third of the continent's bee population died each year beginning around 2006. In 2012, peer-reviewed studies concluded that neonicotinoid pesticides are linked to the crash in the bee population. However, pesticide manufacturers and agribusiness companies assert there is insufficient evidence to prove that neonicotinoid pesticides are harmful. To date, the EPA has not suspended or canceled registrations of any pesticides within the neonicotinoid family.

There is a considerable amount of scientific evidence suggesting that neonicotinoids may be causing a mass bee die-off in the form of colony collapse disorder. Because of this evidence, the EPA should temporarily

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suspend the use of neonicotinoid pesticides. The EPA should suspend or cancel any pesticide that has significant evidence suggesting it causes severe environmental harm, even if the harmful nature of the pesticide is scientifically uncertain. The uncertainty of the harm should weigh against the use of the pesticide, not in favor of leaving it in use. The severity and irreversibility of the potential damage caused by pesticides justifies extreme caution. Therefore, if there is a possibility of catastrophic harm, the EPA should act with a heightened level of caution. When presented with strong evidence that suggests a pesticide is or might be causing severe environmental harm, the EPA should suspend the registration of that pesticide until the pesticide manufacturer can prove that the pesticide is safe. By waiting to act until the pesticide is conclusively proven to be harmful, the EPA leaves a potentially damaging pesticide in circulation, and, thus, exposes the environment to an unreasonable level of risk.

This article will explain colony collapse disorder and some of the evidence suggesting that neonicotinoid pesticides may be responsible. Then, it will explain the history of the development of neonicotinoids and their significance in agribusiness. Next, this article will analyze whether the benefits and risks of neonicotinoid pesticides weigh in favor of the EPA either banning the pesticides or doing nothing and continuing to allow their use. Specifically, it will argue that, in all cases when the harm is potentially disastrous, but of an uncertain or unknown likelihood, the EPA should exercise extreme caution and suspend the registration of pesticides when they could be potentially catastrophic. Furthermore, it will argue that the pesticide manufacturer should always bear the burden of proving that its pesticides are safe, and the EPA should not require its opponents to prove the pesticides are harmful before suspending them.

II. WHAT IS COLONY COLLAPSE DISORDER?

In 2006, it became clear that the bee population was already in rapid decline. Millions of bees were vanishing in the United States and Europe every year, and no one could explain why.[1] Approximately one-third of the remaining bee population was dying each year, every year.[2] In the 2012-2013 winter, about half of the remaining bee population died.[3] It didn't take long for beekeepers to start importing bees just to meet the basic

1. *Colony Collapse Disorder Progress Report,* U.S. DEP'T. OF AGRIC. (June 2010), https://www.ars.usda.gov/is/br/ccd/ccdprogressreport2010.pdf.

2. *Id.*

3. Michael Wines, *Mystery Malady Kills More Bees, Heightening Worry on Farms*, N.Y. TIMES, Mar. 28 2013, http://www.nytimes.com/2013/03/29/science/earth/soaring-bee-deaths-in-2012-sound-alarm-on-malady.html?_r=1&.

pollination needs of plant life in the United States.[4] Losses of honey bees and costs to import bees increased the cost to rent migratory beekeepers' pollination services by 20%.[5] This pattern of widespread, sustained, and inexplicable bee death became known as "colony collapse disorder," or CCD.

For years the cause of the mass bee die-off was a complete mystery.[6] Researchers would find intact bee colonies totally abandoned for no apparent reason.[7] All the adult bees seemed to have decided to leave simultaneously, leaving the queen and larvae to starve to death.[8] Puzzlingly, colonies seemed to collapse completely at random.[9] Beekeepers would find some of their colonies wiped out, while the other beehives right next to them continued as normal, completely unaffected.[10] No adult worker bee corpses could be found in the abandoned colonies, and there was no clue to what could be causing the colonies to fail in such shocking numbers.[11]

The massive and ongoing die-off of bees in the United States is a grave situation because bees are integral to the economy, particularly in agriculture. Bees, especially honey bees, are by far the most important pollinators for all crops that require pollination.[12] The economic impact of America's tireless army of worker bees is tremendous.[13] The United States Department of Agriculture estimates that honey bee pollination is worth about $15 billion a year in the US.[14] For comparison, the entire output of all farms in the United States put together constituted $138.7 billion added

4. *Id*.

5. *Id*.

6. *Nature: Silence of the Bees*, PBS.ORG (Oct. 28, 2007), http://www.pbs.org/wnet/nature/episodes/silence-of-the-bees/introduction/38/. *See also* Diana Cox-Foster & Dennis VanEngelsdorp, *Solving the Mystery of the Vanishing Bees*, SCIENTIFIC AMERICAN (2009), *available at* http://www.scientificamerican.com/article/saving-the-honeybee/.

7. *Id*.

8. *Id*.

9. *Id*.

10. *Id*.

11. *Id*.

12. Seth Borenstein, *Honeybee Die-Off Threatens Food Supply*, WASH POST, May 2, 2007, http://www.washingtonpost.com/wp-dyn/content/article/2007/05/02/AR2007050201413.html.

13. John Mburu et al., Economic Valuation of Pollination Services: Review of Methods, Food and Agric. Organization of the United Nations, (June 2006), *available at* http://www.fao.org/fileadmin/templates/agphome/documents/Biodiversity-pollination/econvaluepoll1.pdf.

14. *Bees in crisis*, U.S. DEP'T OF AGRIC., http://www.csrees.usda.gov/newsroom/impact/2008/lgu/144_bees_in_crisis.html (last visited Mar. 15, 2014).

to GDP in 2011.[15] Wild honeybees pollinate crops all over the country for free and make it possible for flowering crops to reproduce. The economic impact on farmers from losing honey bees' free pollination would be enormous.

The consequences of a potential bee extinction are chilling. For starters, honey would become unobtainable. Insect-pollinated crops would become impossible to grow. Other industries that depend on plant products would be seriously affected. Insect-pollinated crops constitute approximately one-third of the human diet worldwide.[16] Without bees, farmers would need to switch crops or manually pollinate the entire crop area, which is almost certainly impossible, or at least economically infeasible. Additionally, many other industries and products depend on pollinators indirectly. For example, beef cattle depend on alfalfa, which is an insect-pollinated crop.[17] Most clothing in the US contains cotton, which also depends on insects for pollination.[18] Without, or even with significantly fewer pollinators, food in general and many other agriculture-dependent products would become very scarce and consequently much more expensive.

Without bees, it would be virtually impossible to grow over 90 different major commercial crops, and insect pollinators are important to over 150 crops.[19] Pollinator-dependent crops are also arguably the best-tasting crops, which are in the highest demand. These include most kinds of nuts, vegetables, and fruits.[20] Many of the world's most popular crops depend on pollinators, including apples, asparagus, blueberries, celery, cherries, cocoa, coffee, peaches, strawberries, soybeans, and all kinds of citrus fruits and melons, to only name a few.[21] Without bees, farmers would be forced to grow almost entirely wind-pollinated crops, leaving the dinner table without fruits and vegetables. Staple crops such as wheat, rice, and corn are wind-pollinated, and could be grown without pollinators. Without bees, the human diet would essentially be reduced to bread and

15. *Ag and Food Sectors and the Economy*, U.S. DEP'T. OF AGRIC. ECON. RESEARCH SERVICE, http://www.ers.usda.gov/data-products/ag-and-food-statistics-charting-the-essentials/ag-and-food-sectors-and-the-economy.aspx#.UyZs4fldWBo (last updated Apr. 8, 2014).

16. Borenstein, *supra* note 12.

17. *Id*.

18. *Facts and Figures*: *The Cotton Trade*, PBS.ORG, http://www.pbs.org/now/shows/310/cotton-trade.html (last visited October 28, 2010).

19. *What Is Pollination?*, ECOLOGICAL SOCIETY OF AMERICA, http://www.esa.org/ecoservices/poll/body.poll.scie.ispo.html (last visited Mar. 15, 2014); Borenstein, *supra* note 12.

20. Borenstein, *supra* note 12.

21. *Id*.

water. If left unchecked, colony collapse disorder could destroy agriculture as we know it.

Early studies to identify the cause of colony collapse disorder found that the process was actually quite complicated. Not all bee colonies collapsed for the same reason, and the simultaneous spike in different causes for the same strange phenomenon was puzzling.[22] Studies found bees were dying from all sorts of common bee pests, including several types of viruses such as *Varroa* mites,[23] the parasite *Nosema*,[24] and other bee pathogens. However, no single pathogen could be isolated as the root cause of the massive die-off. In general, bees were stressed and malnourished, with no hint as to why.[25] Many explanations were proposed, ranging from the prevalence of high-fructose corn syrup,[26] to shrinking agricultural biodiversity,[27] to the possibility that electromagnetic radiation[28] may be damaging the bees somehow. None could be demonstrated to be more than conjecture. For years the bee population was in free fall, and there was nothing anyone could do to stop the looming extinction of human agriculture's most important species.

III. WHY ARE THE BEES DYING?

Five years after the bees began rapidly disappearing, scientists began to unravel the mystery of colony collapse disorder. In April of 2012, two separate studies published in the same volume of *Science* found a connection between neonicotinoids and colony collapse disorder. The first of the two studies, the *Whitehorn* study, found that even very low level exposure to neonicotinoids, nonlethal to any individual bee, had a significant negative effect on colony growth and queen production.[29] The

22. Dennis van Engelsdorp et al., Fall Dwindle Disease: A Preliminary Report, Mid-Atlantic Apiculture Research and Extension Consortium (MAAREC) – CCD Working Group (Jan. 5, 2006), *available at* http://www.beekeeping.com/articles/us/ccd.pdf.

23. Jennifer Welsh, *Mites and Virus Team Up to Wipe Out Beehives*, LIVESCIENCE.COM (June 7, 2012 02:00 PM), http://www.livescience.com/20815-honeybee-collapse-mite-virus.html.

24. Mariano Higes et al., *Nosema ceranae, A New Microsporidian Parasite In Honeybees In Europe*, 92 JOURNAL OF INVERTEBRATE PATHOLOGY 59, 93-95 (2006).

25. *Engelsdorp*, *supra* note 22, at 1.

26. Petra Steinberger, *Das spurlose Sterben*, SÜDDEUTSCHE.DE, May 19, 2010, http://www.sueddeutsche.de/wissen/raetselhafter-exitus-der-bienen-das-spurlose-sterben-1.911906.

27. Kate Pickett, *Postcard from Hughson*, TIME (Mar. 12, 2009), http://content.time.com/time/magazine/article/0,9171,1884835,00.html

28. Geoffrey Lean & Harriet Shawcross, *Are mobile phones wiping out our bees?*, THE INDEP., Apr. 15, 2007,

http://www.independent.co.uk/environment/nature/are-mobile-phones-wiping-out-our-bees-444768.html.

29. Penelope R. Whitehorn et al., *Neonicotinoid Pesticide Reduces Bumble Bee Colony Growth and Queen Production*, 336 SCIENCE no. 6079, at 351 (Mar. 29, 2012), *available at*

Henry study, conducted independently and published in the same volume, found that neonicotinoid exposure decreases honey bee foraging ability substantially and causes bees to become disoriented and unable to navigate effectively.[30] These concurring studies gave a significant amount of credibility to the speculation that perhaps neonicotinoid pesticides were responsible for colony collapse disorder.[31]

The two initial studies published in April led to a flurry of dozens of other studies and reports confirming the results. Additional research found a wide variety of other debilitating, but nonlethal, symptoms of exposure to low levels of neonicotinoids in bees.[32] Neonicotinoids impair bees' olfactory abilities, memory, learning, and ability to navigate.[33] And, most worryingly, several studies found that clothianidin, a specific type of neonicotinoid, seriously compromises the bee's immune system.[34] Exposure to clothianidin leaves the bee's immune system critically weakened and easy prey for bacteria, viruses, and parasites. Worse still, if an entire colony's immune system is compromised by clothianidin, a single bee might carry a pathogen into the hive where it will run rampant and wipe out the entire colony. This phenomenon explains the difficulty of

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http://www.sciencemag.org/content/336/6079/351.full?sid=da27b0a3-4e10-4269-8cb1-a218db43561d.

30. Mickael Henry Et Al., *A Common Pesticide Decreases Foraging Success and Survival in Honey Bees*, 336 SCIENCE no. 6079, at 348 (Mar. 29, 2012), *available at* http://www.sciencemag.org/content/336/6079/348.full?sid=da27b0a3-4e10-4269-8cb1-a218db43561d.

31. Erik Stokstad, *Field Research on Bees Raises Concern About Low-Dose Pesticides*, 335 SCIENCE no. 6079, at 1555 (Mar. 30, 2012), *available at* http://www.sciencemag.org/content/335/6076/1555.full?sid=f85409d2-dc06-4180-be5e-e30065285db9.

32. Richard J. Gill et al., *Combined pesticide exposure severely affects individual- and colony-level traits in bees*, 491 NATURE no. 7422, at 105 (Nov. 1, 2012), *available at* http://www.nature.com/nature/journal/v491/n7422/full/nature11585.html?WT.ec_id=NATURE-20121101; Lu Chensheng et al., *Sub-lethal exposure to neonicotinoids impaired honeybee winterization before proceeding to colony collapse disorder*, 67 BULL. OF INSECTOLOGY no. 1, at 125 (2014), http://www.bulletinofinsectology.org/pdfarticles/vol67-2014-125-130lu.pdf; Christian Krupke et al., *Multiple Routes of Pesticide Exposure for Honey Bees Living Near Agricultural Fields*, 7 PUB. LIBR. OF SCI. no. 1, at 1 (Jan. 3, 2012), http://www.plosone.org/article/fetchObject.action?uri=info%3Adoi%2F10.1371%2Fjournal.pone.0029268&representation=PDF; Gennaro Di Prisco et al., *Neonicotinoid clothianidin adversely affects insect immunity and promotes replication of viral pathogen in honey bees*, 110 PROCEEDINGS OF THE NAT'L ACAD. OF SCI. OF THE U.S. no. 46, at 18465 (Nov. 12, 2013), http://www.pnas.org/content/110/46/18466.full; Tapparo et al., *Assessment of the Environmental Exposure of Honeybees to Particulate Matter Containing Neonicotinoid Insecticides Coming from Corn Coated Seeds*, 46 ENVTL. SCI. & TECH. no. 5, at 2592 (Jan. 31, 2012).

33. Sally M. Williamson et al., *Exposure to multiple cholinergic pesticides impairs olfactory learning and memory in honeybees*, 216 THE J. OF EXPERIMENTAL BIOLOGY 1799 (Mar. 15, 2013), http://jeb.biologists.org/content/216/10/1799.full.pdf+html.

34. Gennaro Di Prisco et. al, *supra* note 32.

isolating the cause of colony collapse disorder, and explains how so many different illnesses and parasites were implicated. In short, impairment to all of a colony's workers eventually causes the colony to fail, but the hive failure can be proximately caused by a variety of different circumstances.

The European Commission asked the European Food Safety Authority (EFSA) to perform a study on the three most common neonicotinoid pesticides: imidacloprid, clothiandin, and thiamethoxam. The EFSA study was published in January of 2013, and contained strong evidence that bees are exposed to the pesticide through multiple previously unknown vectors, including pollen, nectar, and dust.[35] The report went on to say that neonicotinoids present in pollen and nectar of plants treated with the pesticides were sufficient to cause significant impairment of the bees' cognitive and motor functions.[36] The report was conclusive and damning, and determined that neonicotinoids, especially clothianidin, pose a "high acute risk" to bees.[37] The EFSA report was the basis for the European Commission to move ahead with banning neonicotinoids, and in March of 2013, the European Commission banned all three of the neonicotinoids. On December 1st, 2013, a two-year moratorium on the three most popular neonicotinoids went into effect in the European Union.[38] In response, the manufacturers of the three pesticides, Bayer and Syngenta, sued in an attempt to get the ban lifted.[39]

The EFSA report on neonicotinoids' effects on bees shows that they are dangerous to bees even in exceptionally low concentrations.[40] Neonicotinoids completely suffuse plants when used as seed or water treatments. Every fiber of each treated plant is infused with pesticide as it grows. Even the nectar and pollen of treated plants contain pesticides, which the bee consumes and carries on its body. The EFSA report reviewed existing data submissions to the EU and also to Member States, and, despite its previous approval, the report determined there were critical

35. Press Release, EUROPEAN FOOD SAFETY AUTHORITY, EFSA identifies risks to bees from neonicotinoids (Jan. 16, 2013), *available at* http://www.efsa.europa.eu/en/press/news/130116.htm?utm_source=homepage&utm_medium=infocus&utm_campaign=beehealth.

36. Conclusion on the peer review of the pesticide risk assessment for bees for the active substance clothianidin, EUROPEAN FOOD SAFETY AUTHORITY, 2 (Mar. 14, 2013), http://www.efsa.europa.eu/en/efsajournal/doc/3066.pdf.

37. *Id*. at 14.

38. European Comm'n, *Bees & Pesticides: Commission goes ahead with plan to better protect bees*, EUROPEAN COMMISSION, http://ec.europa.eu/food/animal/liveanimals/bees/neonicotinoids_en.htm (last updated May 30, 2013).

39. Tania Rabesandratana, *Pesticidemakers Challenge EU Neonicotinoid Ban in Court*, SCIENCEINSIDER (28 August 2013 12:30 PM), http://news.sciencemag.org/europe/2013/08/pesticidemakers-challenge-e.u.-neonicotinoid-ban-court.

40. EUROPEAN FOOD SAFETY AUTHORITY, *supra* note 36.

data gaps regarding exposure to pollinators; therefore the pesticides were potentially dangerous.[41] The EFSA determined the pesticide concentrations in nectar, pollen, and even airborne dust were sufficient to substantially impair bees.[42] Neonicotinoids can be suspended in water and soil, spreading and leaching throughout any permeable ground. Furthermore, because neonicotinoids endure in soil and water for extended periods of time, they can be harmful to bees long after their application. Neonicotinoids' potency and stability make them highly dangerous in low concentrations for extended periods of time.

Neonicotinoids were tested for their impact on non-target organisms before they were registered, including honey bees. However, the EPA approved neonicotinoids for widespread use, presumably because the EPA did not detect the subtle, slow, long-term debilitation to bees described in the studies connecting neonicotinoids to colony collapse disorder. The EPA cannot be expected to administer an IQ test to every honeybee that meandered through a test field, much less months after the fact. Such nonlethal impairment would have been undetectable, even though it indirectly resulted in a bee population collapse of unprecedented scope and speed.

Even at miniscule doses, bees' cognitive and nervous system functions are seriously impaired by neonicotinoids. However, at sub-lethal doses the effects of the neonicotinoids are difficult to detect. But the bees are sufficiently debilitated by chronic exposure to the pesticides, such that they become weaker, less effective foragers, and more vulnerable to common bee pathogens. Despite the sub-lethal effects of chronic low-level exposure of neonicotinoids on bees, neonicotinoids have caused a widespread epidemic of collapsing bee colonies and a sharp decline in the bee population.

IV. WHAT ARE NEONICOTINOIDS?

Neonicotinoids are a relatively new family of pesticides that have rapidly become the most widely used pesticides in the world. As the name suggests, neonicotinoids use the same mode of action as the chemical nicotine, which is commonly found in tobacco. Nicotine is highly toxic to insects and has been infrequently used as a pesticide for over 200 years.[43]

41. *Id.* at 7.

42. *Id.* at 14.

43. István Ujváry, *Nicotine and Other Insecticidal Alkaloids*, *in* NICOTINOID INSECTICIDES AND THE NICOTINIC ACETYLCHOLINE RECEPTOR 29-69 (Izuru Yamamoto & John Casida eds., 1999), *available at* http://books.google.com/books?id=_kbFQ9-RUyUC&printsec=frontcover&source=gbs_ge_summary_r&cad=0#v=onepage&q&f=false.

However, there are two critical problems with nicotine as a pesticide. First, nicotine is toxic to a wide variety of organisms and is not selective enough to safely apply on a large scale. Nicotine is so toxic it is actually banned for use as a pesticide in the United States.[44] Second, nicotine degrades quickly in the environment, making it both laborious and expensive to frequently reapply to a large crop area. Because of the problems with older, highly toxic pesticides, large agriculture businesses needed to find a pesticide that was selectively targeted to only eliminate pests, and that would persist in the field long enough to eliminate the need for repeated and expensive reapplications.

The first research into neonicotinoids began in the 1980s.[45] The concept was simple. Nicotine, the well-known toxin found in tobacco, has a substantially different toxic response in mammals than it does in insects. Specifically, nicotine is highly toxic to mammals and less toxic to insects. Scientists at Bayer inferred that the same toxic substance must cause different chemical responses in mammal physiology and insect physiology. Bayer, a pharmaceutical corporation famous for inventing aspirin and other well-known drugs, had hit upon something huge for the future of agriculture. Theoretically, Bayer reasoned, it should be possible to design a chemical that has the opposite toxicity profile to nicotine - a super-pesticide that which would be violently toxic to insects, yet harmless to mammals, including humans.

Fast forward to 1986 when Bayer filed for a patent on a chemical called imidacloprid, the first commercially viable neonicotinoid.[46] Imidacloprid is a brilliant invention by any standard. Imidacloprid works by targeting specific acetylcholine receptors only found in the insect central nervous system.[47] As a result, imidacloprid is highly effective at killing insect pests, and it is non-toxic to mammals because mammals lack that particular kind of receptor.[48] Therefore, there is no risk of poisoning humans, pets, livestock, or a variety of other creatures, like earlier and less sophisticated pesticides would. Imidacloprid clearly kills insects, and after

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44.7 C.F.R. § 205.602.

45. Willy D. Kollmeyer Et. Al., *Discovery of the Nitromethylene Heterocycle Insecticides*, *in* NICOTINOID INSECTICIDES AND THE NICOTINIC ACETYLCHOLINE RECEPTOR, 71, 71-89 (Izuru Yamamoto & John Casida eds., 1999).

46. Izuru Yamamoto, *Nicotine to Nicotinoids: 1962 to 1997*, *in* NICOTINOID INSECTICIDES AND THE NICOTINIC ACETYLCHOLINE RECEPTOR 3-27 (Izuru Yamamoto & John Casida eds., 1999), *available at* http://books.google.com/books?id=_kbFQ9-RUyUC&printsec=frontcover&source =gbs_ge_summary_r&cad=0#v=onepage&q&f=false.

47. J.A. Gervais Et.Al., *Imidacloprid Technical Fact Sheet*, NAT'L PESTICIDE INFO. CENTER, http://npic.orst.edu/factsheets/imidacloprid.html (last visited Feb. 15, 2014).

48. *Id.*

thorough examination it has virtually no effect on mammals, except in extreme doses.[49] The Patent & Trademark Office granted the patent to Bayer in 1988, Bayer applied to register the chemical as a pesticide in 1992, and the EPA approved its pesticide registration in 1994.[50]

In the mid 1990s, the time was ripe for the next generation of pesticides to hit the market. Previous agriculture techniques were labor and planning-intensive, including crop rotation, careful maintenance of an ecosystem of natural predators of undesirable insects, and the use of highly toxic chemical controls as a last resort only.[51] This complex ecosystem, called "integrated pest management," or "bio-IPM," had previously worked for decades. Wasteful use of pesticides was uneconomical for farmers, due to both the high cost of repeated applications and the toxicity of the chemicals damaging the crops, which could possibly harm someone that consumed them later. Regulatory pressures to stop using highly toxic pesticides were building, and pressures from patented seed giants and an evolving pest ecosystem made it more challenging to use the complex, labor-intensive methods of IPM. When imidacloprid hit the market in the mid 1990s, it revolutionized pesticides because it was a non-toxic, low-cost pesticide that could cheaply inoculate an entire field. In only a few years, large agribusiness shifted away from integrated pest management completely and began widespread, heavy, and sustained preemptive use of chemical pesticides across massive farms.

When imidacloprid entered the market in the 1990s, it was clearly superior to the available pesticide choices. The best available pesticides were highly toxic, including organophosphates, organochlorine compounds (e.g., DDT), and methyl carbamates.[52] Organophosphates were the most common type of pesticide, comprising approximately 43% of the world's pesticide market share.[53] While undeniably effective at killing insects, organophosphates, organochlorine compounds, and other highly toxic pesticides also tend to kill everything else, and, thus, must be used very carefully. These chemicals are so toxic they have also been weaponized for intentional use against humans. For example,

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49. *Id*.

50. U.S. Patent No. 4,742,060 (filed Jan. 21, 1986).

51. *Integrated Pest Management (IPM) Principles*, EPA, http://www.epa.gov/pesticides /factsheets/ipm.htm (last updated Aug. 5, 2014).

52. *Types of Pesticides*, EPA, http://www.epa.gov/pesticides/about/types.htm (last updated Aug. 5, 2014).

53. Peter Jeschke, et al., *Overview of the Status and Global Strategy for Neonicotinoids*, 59 JOURNALS OF AGRIC. AND FOOD CHEMISTRY 2897, 2897 (2011), *available at* http://www.moraybeedinosaurs.co.uk/neonicotinoid/global.pdf.

organophosphates were used in World War II as nerve agents.[54] Farmers were literally using chemical weapons on human food in the war against pests. Pesticides like neonicotinoids that are actually safe for humans to ingest made highly toxic pesticides obsolete. The EPA made the quite reasonable move of using the newly developed neonicotinoid pesticides to phase out highly toxic and dangerous pesticides.

Apart from its selectivity, Imidacloprid has two key properties that make it an extremely effective and efficient pesticide for large-scale agriculture. First, imidacloprid is incredibly potent. While its application rate varies by preparation and intended crop, it is generally effective against insects at doses in the parts-per-billion range, or around 0.1 pounds of active ingredient per acre of crops.[55] Second, imidacloprid is water-soluble and stable in the environment for months.[56] These properties enable a wide variety of delivery methods, such as seed treatments and additives to water used to irrigate crops.[57] As a result of these properties, plant seeds or roots can be treated to cause the plant to absorb the pesticide.[58] Seed treatments are the most popular application of imidacloprid.[59] If an insect, such as an aphid, consumes some of a treated plant, it will die. The plant itself is completely suffused with micro-dosage levels of pesticide that make it poisonous to insects, but still perfectly edible for humans.[60] Pesticides that are fully absorbed into the plant are called systemic pesticides. Imidacloprid is the most popular systemic pesticide, and currently is the most widely used pesticide in the world.[61]

Since it entered the market in the 1990s, imidacloprid has been extremely successful, and equally lucrative. Imidacloprid is still in heavy use today, and remains the most popular neonicotinoid pesticide with over $1 billion in revenue in 2009.[62] Imidacloprid's potency, efficiency, and unprecedented highly desirable selectivity made it an easy sell. Its tiny volume and low cost for large crop areas made it extremely efficient for

54. EPA, *supra* note 52.

55. BAYER INC., CONFIDOR® 200 SC, BAYER CROPSCIENCE, *available at* http://www.bayercropscience.com.au/resources/uploads/label/file9728.pdf?2014217618 (pesticide application instructions).

56. Jeschke Et Al., *Overview of the Status and Global Strategy for Neonicotinoids*, 59 JOURNALS OF AGRIC. AND FOOD CHEMISTRY 2897, 2900 (2011).

57. *Id*.

58. Jeschke et al., *supra* note 53, at 2900-2901.

59. *Id*. at 2901.

60. *Id*. at 2900.

61. Izuru Yamamoto & John Casida, Nicotinoid Insecticides And The Nicotinic Acetylcholine Receptor 3–27 (Izuru Yamamoto & John Casida eds., 1999).

62. Jeschke et al., *supra* note 53, at 2900.

large agriculture businesses to apply to tremendous tracts of land.[63] Marketed as a safe pesticide, sales of imidacloprid ballooned over the years since its development.[64] Imidacloprid also branched out into different formulations, and today imidacloprid is used in a tremendous variety of products from seed preparations to pet flea treatments.[65] The invention of imidacloprid sparked a revolution in pesticides because they made it safe and affordable to preemptively carpet an entire field with pesticides.[66] Imidacloprid was the first and is still the most successful of the neonicotinoids, pesticides engineered to eliminate pests and cause minimal harm to other organisms.

Imidacloprid was the first neonicotinoid pesticide, but since its invention in 1986 other variations of neonicotinoids have been developed. There are significant differences between the different neonicotinoid compounds, but the other neonicotinoids share imidacloprid's basic properties of selectivity, potency, water-solubility, and stability. Different neonicotinoids are used in different ways and in different formulations, on different crops, and by different companies. Other neonicotinoids include clothianidin, thiamethoxam, and acetaminiprid, among others.[67] Their patents and registrations vary, but they share a common chemistry that governs their effects regardless of their documentation.

V. HOW ARE PESTICIDES REGULATED?

All pesticides in the United States are tightly regulated. Under the Federal Insecticide Fungicide and Rodenticide Act (FIFRA), all pesticides are scrutinized by the Environmental Protection Agency before they can be registered and subsequently used.[68]

The EPA will only register pesticides that will not cause unreasonable harm to the environment, and the registration process requires a significant amount of data before the pesticide can be registered.[69] The registrant, typically the manufacturer of the pesticide, must provide certain data to the EPA.[70] In addition to basic information about the pesticide, the registrant must provide data about the pesticide's "environmental fate," that contains studies assessing environmental effects

63. Id.
64. *Id*.
65. *Id*.
66. *Id.* at 2898.
67. *Id*. at 2901.
68. 7 U.S.C. § 136a (a) (2007).
69. *Id*.
70. 7 U.S.C. § 136a (c) (2007).

and including nontarget organisms.[71] A particular pesticide registration will also specify the crops and sites on which it may be used, labeling requirements, and other limitations on the pesticide's use. Each permitted use must be supported by research data on the pesticide's effects.[72] The EPA will periodically review every pesticide once every fifteen years.[73] The EPA can also give new pesticides conditional registrations while the EPA obtains the data needed to make a thorough analysis of the pesticide.[74] Furthermore, the registrant must notify the EPA of any newly uncovered facts concerning adverse environmental effects.[75] Pesticides have the potential to cause immense environmental harm; therefore the government tightly controls their use.

The critical component of FIFRA regarding environmental harm concerns the EPA's required findings of the risks and benefits of the pesticide. The EPA must determine that the pesticide will perform its intended function "without unreasonably adverse effects on the environment."[76] An unreasonable adverse effect on the environment is defined as either (1) any unreasonable risk to man or the environment, taking into account the economic, social, and environmental costs and benefits of the use of the pesticide, or (2) a human dietary risk from residues that result from a use of a pesticide in or on any food.[77] In its assessment, The EPA can also consider economic or environmental harms as well as other possible effects. Any pesticide that creates an unreasonable risk must be denied registration.[78]

The EPA's authority over pesticide registrations does not end when registration is granted. The EPA can cancel or suspend any registration at any time at its discretion.[79] Furthermore, if the EPA believes a pesticide poses an "imminent hazard," it can issue an emergency order to suspend the use of the pesticide, which immediately freezes the sale, distribution, and use of the pesticide.[80] A pesticide registration is a tightly controlled limited permit that is revocable at any time. Under FIFRA, the EPA decides whether to register a pesticide in part based upon the economic and environmental risks of the pesticide balanced against the benefits of

71. 40 C.F.R. § 158 (2014).
72. 40 C.F.R. § § 155-158; 40 C.F.R. § 171 (2014).
73. 7 U.S.C. § 136a (g)(1)(A) (2007).
74. 7 U.S.C. § 136a (c)(7) (2007).
75. 7 U.S.C. § 136a (g)(1)(A) (2007); 40 C.F.R. § 159 (2014).
76. 7 U.S.C. § 136a (c)(5)(C) (2014)
77. 21 U.S.C. § 346a (2014)
78. 7 U.S.C. § 136a (2014)
79. 7 U.S.C. § 136d (b); 7 U.S.C. § 136d (c) (2014).
80. 7 U.S.C. § 136d(c)(3) (2014).

using the pesticide. The environmental fate of the pesticide, including its effects on nontarget organisms, is a critical component of the pesticide's data requirements.

VI. WHAT SHOULD THE EPA DO?

Before granting registration, the EPA requires research data to show a pesticide is safe.[81] In order to determine if a pesticide is safe, the EPA must determine whether a pesticide represents an unreasonable risk.[82] The EPA can also cancel or suspend the registration on neonicotinoids, or even on particular compounds or uses.[83]

However, after a registration has been granted, the EPA adopts the stance that it must find the pesticide is harmful before it will cancel the pesticide's registration. The EPA is very aware of the possibility that neonicotinoids are harmful, and has decided to leave them registered while it conducts further research.[84] In the EPA's own words from its response to a public petition requesting a ban on a specific neonicotinoid: "This extensive review will determine if any restrictions are necessary to protect people, the environment, or pollinators."[85] The EPA went on to say:

> [P]etitioners failed to provide evidence to show that there is a substantial likelihood that the alleged threatened harm will occur . . . The EPA agrees with the scientific community that additional research is necessary to address CCD. However, the existence of uncertainty as to these questions is not sufficient to satisfy the high probability standard necessary to support a finding of imminent hazard.[86]

Simply put, the EPA will not suspend first and then conduct research to prove the pesticide's safety. Instead, the EPA adopts the stance of leaving it on the market while researching its safety.

The decision on whether to suspend or cancel the registrations of neonicotinoid pesticides requires weighing the benefits of using them

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81. 7 U.S.C. § 136a(c)(2) (2014).

82. " Explanation of Statutory Framework for Risk-Benefit Balancing for Public Health Pesticides, EPA, http://epa.gov/pesticides/health/risk-benefit.htm (last updated May 9, 2012)

83. 7 U.S.C. 136a.

84. *Clothianidin - Registration Status and Related Information*, EPA, http://www.epa.gov/opp00001/about/intheworks/clothianidin-registration-status.html (last updated July 11, 2013).

85. *Id*.

86. STEVEN P. BRADBURY, EPA, No. EPA-HQ-OPP-2012-0334-0006, CLOTHIANIDIN EMERGENCY CITIZEN PETITION DATED MARCH 20, 2012, (July 17, 2012), *available at* http://www.regulations.gov/#!docketDetail;dct=FR%252BPR%252BN%252BO%252BSR;rpp=25;po=0;D=EPA-HQ-OPP-2011-0865.

against the risks. Farmers and agribusinesses have realized huge gains from using neonicotinoids, which are strong considerations in favor of permitting the pesticides to be used. Leaving neonicotinoids on the market is risky because recent studies suggest the pesticides are causing a rapid collapse in the bee population. Ideally, scientific certainty would be available to enable the EPA to make a decision with clarity and certainty. However, the EPA must make a decision regarding whether to suspend the registration of neonicotinoids, and must decide without scientific certainty. Unfortunately, the EPA is hesitant to suspend the registration of neonicotinoids because the theory that colony collapse disorder is caused by the pesticides has not been established with scientific certainty. The issue is in dealing with the uncertainty of causation of a potentially catastrophic harm.

Canceling or suspending the use of neonicotinoids has large costs. Neonicotinoids are beneficial to the efficiency and productivity of agriculture and significantly increase the yield and profitability of farms. They are also safer for humans and other mammals than more toxic pesticides. Suspending or banning the pesticides would result in profits lost that could have been realized by farmers and pesticide manufacturers. Agriculture companies that use neonicotinoids extensively will have to take other steps, which may reduce their yield or profitability. Pesticide manufacturers will lose billions in revenue. Farmers may be forced to use less sophisticated, highly toxic pesticides. Taking steps to mitigate risks is burdensome.

Biotech giants and agribusiness companies have a huge vested interest in continuing to sell and use neonicotinoids. Pesticides like neonicotinoids undoubtedly have tremendous advantages. They are safer, less toxic, and more efficient than previous pesticides. Arguably, it makes no sense to force farmers to use older, clearly inferior pesticides just on the mere suspicion that an otherwise clearly superior pesticide *might* have a problem. Banning them would be a pointlessly painful burden, a regressive restraint on the progress of science and technology out of fear. From the business perspective, it almost seems like a preposterous idea that the EPA would suspend the use of such a marvelous chemical that is already so widely used with such success.

However, the case that neonicotinoids are causing colony collapse disorder is strong enough to warrant serious consideration. Multiple concurring scientific studies suggest a link between neonicotinoids and colony collapse disorder. Those studies explain the symptoms and the nature of the collapse of hives due to a variety of other factors, including pathogens because of compromised immune systems, reduced foraging

capability due to cognitive impairment, and other symptoms. The theory that neonicotinoid pesticides are causing colony collapse disorder perfectly explains the mysterious characteristics of the phenomenon. Given the best available information and mounting evidence, it seems probable that neonicotinoids are causing colony collapse disorder.

The sheer severity of the consequences of continuing to use neonicotinoids, if they are indeed the cause, must also weigh heavily in the decision. Suppose for the sake of argument that neonicotinoids are in fact causing a rapid extinction of bees. Continuing to use neonicotinoids would cause one of the largest biological disasters in history. Without bees, many flowering crops would be unable to reproduce. Hundreds of types of crops and other plants would become extremely difficult, if not totally infeasible, to grow. Billions of dollars in sales of flowering crops would be lost with untold damage to other industries dependent on plant products. Perhaps even worse than the economic harm would be the dietary impoverishment that would follow when all crops, except staples like wheat and rice, would be astronomically expensive. Fruits, vegetables, and flowers would all require extraordinary efforts to pollinate them by some other means, such as manually pollinating each flower by hand. If neonicotinoids are causing colony collapse disorder, the consequences of leaving them registered and on the market will be catastrophic.

However, the theory that neonicotinoids are causing colony collapse disorder is still unproven and could possibly later turn out to be incorrect. The pesticide manufacturers claim that this lack of proof is sufficient justification not to suspend the registration of neonicotinoids. Other factors may be causing colony collapse disorder; therefore suspending the registration of neonicotinoids may be futile and unnecessary. Further, proponents of neonicotinoids claim that suspending the use of the best pesticides would just make farmers use worse pesticides. This was a compelling argument in the 1990s, and the EPA responsibly granted registration to imidacloprid based on the best information available at the time. At that time, the best available data about neonicotinoids showed they were much safer than existing pesticides, In particular, they were safer for humans and other mammals, which were the main concern. But today the primary concern has shifted to pollinators, and the best information available now suggests that neonicotinoids may be unsafe for bees, despite their safety for humans.

The decision to ban neonicotinoids is muddied by uncertainty. If it were already conclusively proven that neonicotinoids were causing a mass extinction in the bee population, the decision would be easy.

Neonicotinoids would need to be banned because the harm of bee extinction would obviously outweigh the benefits. But if neonicotinoids are actually unrelated to colony collapse disorder, then banning them would be a huge mistake. The direct economic harm from banning them would be totally unnecessary and devoid of any benefit. The connection between neonicotinoids and colony collapse disorder is not conclusively established, and even if it eventually is proven, the process will take time.

The EPA can decide either to suspend the pesticide registration, or do nothing and, thereby, continue to allow the use of neonicotinoids. The EPA must make a decision before knowing for certain whether neonicotinoid pesticides actually cause colony collapse disorder. Acting to suspend the registration incurs an immediate cost, but the uncertainty of the possible disastrous consequences of inaction should loom large in the decision. When it comes to the issue of suspending a pesticide's registration, which will *definitely* be harmful to agribusiness, an easy answer does not exist.

So, the real issue is this: what constitutes an unreasonable risk? Does leaving an already-registered pesticide on the market become an unreasonable risk with the mere *possibility* that it may be harmful, provided that the possible harm is catastrophic?

VII. WHAT CONSTITUTES AN UNREASONABLE RISK?

A risk consists of both a negative possible outcome, and a probability that the undesirable outcome will actually manifest. Dealing with uncertainty means dealing with risk instead of with cold, absolute fact. A risk is unreasonable when the severity and likelihood of the harm considered together are greater than the cost of mitigating the risk. Harm that is more likely and/or more serious constitute greater risks.

Often, mitigating risks comes with real costs that may turn out to be "wasted" when it turns out that the harmful outcome did not occur, or never would have happened anyway. However, this does not mean that the steps taken to mitigate the risk were futile. The probability of the harm must be taken into account to determine whether the risk is unreasonable. Smart risk management will often result in costs to remove risks that would not manifest because it is impossible to know the outcome in advance.

There is some unknown probability that the environment will be very seriously affected by allowing neonicotinoids to remain in use. Furthermore, mitigating the risk that neonicotinoids may be contributing to colony collapse disorder carries a smaller, but more direct cost in the form of reduced profitability, decreased yield, and other diminished gains

for farmers and agribusiness. An accurate assessment of the costs, benefits, risks, and probabilities involved would be extremely useful, but will not be available for some time. So, the EPA must assess whether the balance of the risks makes the risk unreasonable. By doing nothing, the EPA is throwing the dice on that risk rather than take the conservative approach of accepting a flat cost to eliminate the risk.

A flat analysis of the facts of a pesticide based on the current knowledge of its chemistry, toxicity, and so on, will always overlook a vital consideration in the use of pesticides: the risk of the unknown. An analysis of the facts naturally assumes that the current best available information is complete and reliable, and that the information is useless in the face of known uncertainty. With this type of analysis, the *fact* of whether the pesticide causes harm is considered to be more important than the *possibility* that the pesticide might cause harm. Under such a regime, it makes sense to do nothing until the best information available can confirm whether or not a pesticide is harmful. And that is exactly what the EPA does.

However, a rational analysis of the risks should lead the impartial analyst to a quite different conclusion. The EPA has no choice but to make the decision in an environment of uncertainty. Deferring the decision until certainty is reached is also a decision: inaction. And the decision to do nothing has the effect of allowing a potentially harmful pesticide to remain in widespread use. The decision should not be deferred until the EPA has access to scientific certainty. The decision must be made as soon as the uncertainty can be identified, knowing full well the decision is made with limited information. The analyst's knowledge that the available information is insufficient to conclusively identify the correct action should prompt a rational analyst to exercise extreme caution.

The EPA should suspend any pesticide's registration as soon as there is evidence that it could be seriously harmful. The costs of suspending the registration are small compared to the potentially catastrophic unknowns of inaction. Even large profits do not in any way justify the risk of leaving such a destructive pesticide in widespread use. Paying marginally more for food until scientific certainty can be reached on the issue is a manageable cost for a limited time. The suspension could even be temporary. After the pesticide is conclusively proven to be safe, the EPA could allow its use again. The prudent course is to immediately suspend the use of neonicotinoids, or any pesticide, as soon as there is reasonable doubt about its safety, such as evidence suggesting they may cause catastrophic harm.

By contrast, gambling that the pesticide is safe is not a wager that anyone can afford to lose. The unknown probability of such a catastrophic amount of economic and environmental harm makes leaving neonicotinoids in use a highly unreasonable risk. Leaving a pesticide registered in the face of uncertain harm is a high-stakes gamble. The potential consequences are so severe that inaction is comparable to russian roulette. Even if the EPA is very confident it will win its gamble, the wager is unbalanced. Just a single loss could wipe out thousands of farmers, deal terrible and irreparable damage to the environment as well as to the economy, and make many flowering crops virtually unavailable. Leaving a potentially catastrophic pesticide in widespread, heavy agricultural use could rapidly have disastrous adverse effects, even during the few years needed to conclusively prove that the pesticide is the cause. The potential consequences are so severe that the only sensible course is to take extra precautions to first confirm that using neonicotinoids will not result in ecological and economic disaster before allowing their use.

Ignoring a potentially catastrophic risk with unknowns in the equation is an enormous mistake. Because of the unknowns, the EPA should adopt the cautious approach of suspending the pesticide. At the very least, the EPA should temporarily suspend the registration of neonicotinoids to wait until the scientific community has established with certainty whether neonicotinoids are safe. The burden to prove that the pesticide is safe must be placed with the pesticide manufacturer, instead of allowing the pesticide to remain in use until the scientists that oppose its registration conclusively prove that it was destroying the environment from the vantage of several years after the fact.

VIII. WHAT IS THE PRECAUTIONARY PRINCIPLE?

The *precautionary principle* is the theory that steps should be taken to mitigate even a potentially beneficial risk when it makes sense to do so. Applied to regulation or public policy, the precautionary principle essentially means that the burden of proof lies with the proponent of a potentially risky policy or action to prove that it is safe, and not on the opponent to prove that it is harmful. The precautionary principle is simply a formalization of common sense risk mitigation. Every person engages this type of risk mitigation every day by wearing a seatbelt and any number of other common precautions. Harms that are irreversible or irremediable warrant a special degree of caution. In simplest terms, the proponent of the risky action must prove that it is safe as a default stance, rather than perform the risky action, unless its opponent can prove it is dangerous.

To argue that a pesticide's registration should not be canceled or suspended, pesticide manufacturers claim that the connection between the pesticide and the claimed harm is unclear. Moreover, because of the EPA's permissive stance towards previously registered pesticides, muddying the waters and creating uncertainty effectively serves their interest of keeping the pesticide on the market. The pesticide manufacturers can easily assert, "You can't *prove* that X causes Y" and, until there is certainty that the pesticide does in fact cause the harm claimed, the EPA will not cancel or suspend the pesticide's registration. Manufactured controversy has the effect of keeping the pesticide on the market; for every year the pesticide in question is on the market, the manufacturer collects billions of dollars in revenue from the sale of the pesticide.

Apportioning the risks of pesticide use in this way defies all common sense. In every other walk of life, the person proposing the risky action must prove that it is a safe, reasonable risk, as opposed to requiring others to prove that it is unsafe.

Imagine if this same argument were applied to pharmaceutical drugs; "You can't *prove* that this drug kills people, so we should be allowed to sell it." Expecting the opponents of the risk to establish conclusive causation of harm imposes a high evidentiary and research burden that will take time to meet; in the meantime the drug remains on the market. Establishing causation with certainty is very difficult, and findings that suggest causation are easily muddied and disputed by additional findings linking other related factors. The only common sense approach is to require the drug manufacturer to prove the drug is safe before it can be sold to consumers.

Pesticides have strong parallels with pharmaceuticals for medical applications. Pesticides are, in every sense that counts, drugs. In fact, many of the same companies manufacture pesticides and pharmaceuticals, including Bayer, who invented aspirin and many other pharmaceuticals in addition to imidacloprid and many other pesticides. However, the possible consequences for using pesticides, whose safety is uncertain, are far more severe than giving a patient drugs of unproven safety. A drug can cause side effects in one human patient, at worst, killing the person. Pesticides can cause side effects spread across the environment, potentially resulting in irreversible harm on the national, or even global scale. Unlike pharmaceuticals, there is only one patient. And death or serious side effects in the patient are absolutely not acceptable due to the scope and irreversibility of the harm. Therefore, the EPA should exercise extreme caution with pesticides.

The EPA evaluates pesticides on the basis of *unreasonable risk*. This means the EPA should cancel or suspend the registration of any pesticide that is an unreasonable risk, incorporating the balance of probabilities of harm into the analysis. When an unknown enters the equation, the EPA should assume the worst because the unknown harm, or unknown likelihood of harm, is a risk. Furthermore, by assuming the worst, the EPA places the burden of proof that the pesticide is not an unreasonable risk with the pesticide manufacturer. The EPA already places this burden on the manufacturer during the initial registration process. This burden should remain with the manufacturer during the registration. After this change, in order to keep the pesticide on the market, the pesticide manufacturer must provide enough information to the EPA to remove any unknowns and establish that the risks are reasonable.

Requiring pesticide manufacturers to provide additional information about the pesticide under these circumstances is the most expedient option. The pesticide manufacturer has the most knowledge and information about the pesticide, and the most interest in the market for the pesticide. Thus, when new evidence arises suggesting that a pesticide is unsafe, the EPA should suspend the pesticide's registration until the pesticide manufacturer can prove the pesticide is safe.

IX. SHOULD THE EPA BAN NEONICOTINOIDS?

In the wake of studies suggesting a link between neonicotinoids and colony collapse disorder, the use of neonicotinoids has become controversial. Activists and petitions calling to ban neonicotinoids have appeared. Pesticide manufacturers and agribusiness groups have replied that there are many factors contributing to colony collapse disorder, and that it has not been conclusively proven that neonicotinoids are the cause.[87] This puts the EPA in a difficult position of weighing the benefits derived from neonicotinoids against a specter of harm, which may or may not be conclusively established later.

Governments in Europe have taken the crisis of bee population collapse far more seriously than the United States. The European Commission requested studies on neonicotinoids and based on the clearly negative results of those studies, imposed a two-year moratorium on the pesticides. If during that two-year period the scientific community reaches the consensus that neonicotinoids are safe, the moratorium will presumably expire instead of being extended or made permanent. The

87. *Beekeepers call for immediate ban on CCD-linked pesticide*, PESTICIDE ACTION NETWORK (Dec. 9, 2010), http://www.panna.org/blog/beekeepers-call-immediate-ban-ccd-linked-pesticide.

European Commission has made a prudent decision by temporarily banning the pesticides, given the evidence currently available.

The EPA has been much less active in its investigation and regulation of neonicotinoids. In response to the EFSA report, the EPA has stated that "the EPA's scientific conclusions are similar to those expressed in the EFSA report with regard to the potential for acute effects and uncertainty about chronic risk."[88] The EPA went on to say, "The neonicotinoid pesticides are currently being re-evaluated through registration review, the EPA's periodic re-evaluation of registered pesticides to ensure they meet current health and safety standards."[89] This refers to the EPA's evaluation of each registered pesticide once every 15 years. However, the EPA's review schedule is overloaded and functions slowly. In fiscal year 2012, the EPA opened 744 pesticide cases comprising 1,165 active ingredients.[90] By law, the EPA must complete its fifteen year cycle by October 1, 2022.[91] As of yet, the EPA has done nothing to impede the use of any neonicotinoid pesticide. In the words of the EPA, "The EPA is not currently banning or severely restricting the use of neonicotinoid pesticides."[92]

Evidence has been mounting since the initial *Science* studies in 2012 that neonicotinoids are not safe for pollinators, and that certain neonicotinoids are in fact extremely dangerous. There is particularly compelling evidence that suggests that Clothianidin is highly hazardous to bees.[93] Clothianidin is the newcomer to the neonicotinoid party; the EPA granted conditional registration for clothianidin in 2003.[94] In its initial conditional registration, the EPA identified numerous data gaps in the submitted data, including a field test for ecological effects on pollinators.[95] Along with the conditional registration, the EPA requested that Bayer CropScience submit several additional studies, including a study on chronic exposure to honeybees through nectar and pollen. The approval process for clothianidin specifically was highly irregular, and likely

88. *Colony Collapse Disorder: European Bans on Neonicotinoid Pesticides*, EPA, http://www.epa.gov/pesticides/about/intheworks/ccd-european-ban.html.

89. *Id*.

90. *Pesticides: Registration Review Program Highlights*, EPA (Sept. 30, 2013), http://www.epa.gov/oppsrrd1/registration_review/highlights.htm.

91. *Id*.

92. EPA, *supra* note 88.

93. *See, e.g.* EUROPEAN FOOD SAFETY AUTHORITY, *supra* note 36; Erik Stokstad, *Pesticides Under Fire for Risks to Pollinators*, 340 SCIENCE no. 6133, at 674 (May 10, 2013), *available at* http://www.sciencemag.org/content/340/6133/674.full ; Gennaro Di Prisco et al., *supra* note 32.

94. *Clothianidin Registration Fact Sheet*, EPA (May 30, 2003), http://www.epa.gov/opp00001/chem_search/reg_actions/registration/fs_PC-044309_30-May-03.pdf.

95. *Id*. at 18.

resulted in a registration that should have been denied. The EPA identified critical data gaps in Bayer's submitted data for clothianidin's registration, and, at Bayer's request, granted conditional registration on the condition that Bayer conduct field studies to test the pesticide's impact on bees.[96] The study was delayed multiple times at Bayer's request, and when it was eventually conducted, it was insufficiently rigorous. Specifically, the field test was conducted on the wrong crop, during the wrong time of year, over an insufficient time period, and with inadequate controls.[97] Nonetheless, a pesticide can be commercially used under a conditional registration, and clothianidin has been in widespread commercial use since 2003.

Clothianidin is essentially a super-neonicotinoid. It is more toxic to insects, more stable in the environment, and easier to absorb into plants through water.[98] Like imidacloprid, clothianidin can be sprayed or used in seed treatments, and it can also be mixed with water used for irrigation. However, it is more potent, having an application rate of between 0.01 to 0.024 pounds per acre.[99] In other words, even in microscopic concentrations, clothianidin is extremely toxic - its application rate calls for only about four grams, spread over an entire acre of land. Clothianidin is also more stable, with a half-life in soil of between 277 and 1,386 days.[100] After being dispersed, clothianidin can remain in the environment for many years, possibly decades. If applied repeatedly over a period of many years, clothianidin levels will gradually accumulate to highly dangerous levels in both soil and water. Worse still, clothianidin is highly mobile and can leach through soil and water; it can leach between bodies of water, including groundwater, and even be transported through the air in dust.[101] The European Food Safety Authority report states that "A high acute risk to honey bees was identified from exposure via dust drift for the seed treatment. A high acute risk was also identified from exposure via residues in nectar and pollen."[102] Once released into the environment,

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96. *Clothianidin Conditional Registration Timeline*, PESTICIDE ACTION NETWORK, http://www.panna.org/sites/default/files/Clothianidin-Condl-Reg-Timeline.pdf (last visited Oct. 29, 2014); *Clothianidin Registration of Prosper T400 Seed Treatment on Mustard Seed and Poncho/Votivo Seed Treatment on Cotton*, EPA (Nov. 2, 2010), http://www.panna.org/sites/default/files/Memo_Nov2010_Clothianidin.pdf.

97. *Beekeepers Ask EPA to Remove Pesticide Linked to Colony Collapse Disorder, Citing Leaked Agency Memo*, PESTICIDE ACTION NETWORK (Dec. 8, 2010), http://www.panna.org/media-center/press-release/beekeepers-ask-epa-remove-pesticide-linked-colony-collapse-disorder-citin.

98. *Clothianidin Fact Sheet*, EPA, 15 (May 15, 2003), http://www.epa.gov/opp00001/chem_search/reg_actions/registration/fs_PC-044309_30-May-03.pdf.

99. *Id*.

100. *Id*. at 15.

101. *See supra* note 37.

102. *Id*.

clothianidin gets *everywhere*, and it takes a very long time to degrade. Using such enduring pesticides repeatedly on such a huge, industrial scale has the potential to suffuse the entire environment with microscopic, but still highly dangerous concentrations of pesticides.

A lawsuit is currently pending - a coalition of environmental groups, beekeepers' associations, and other groups have sued the EPA, claiming that the EPA granted registration to the pesticide clothianidin after virtually no review whatsoever.[103] The EPA granted clothianidin conditional registration in 2003, despite identifying serious "data gaps" in the submitted studies, and requested an additional study about the effects of clothianidin on pollinators.[104] Bayer delayed for a year, requested an extension, and delayed again for two more years. Bayer also requested permission to perform the pollinator study on canola fields in Canada instead of on corn fields in the United States, which the EPA granted. The study of clothianidin's effects on pollinators was finally conducted in 2007. At the time, the EPA classified the study as "acceptable."[105] But in 2010, the EPA issued a memorandum that stated that the study Bayer submitted regarding the effects of clothianidin on pollinators was inadequate.[106]

Still, the clearest evidence of clothianidin's effects on honeybees actually comes directly from the EPA. In 2010, Bayer petitioned the EPA to allow it to sell clothianidin for use on cotton and mustard seed, in addition to its other crop treatments, including corn, canola, soy, and other crops.[107] A leaked internal EPA memo in connection with the request from Bayer clearly identified that clothianidin posed an acute risk to honey bees:

> Clothianidin's major risk concern is to nontarget insects (that is, honey bees). Clothianidin is a neonicotinoid insecticide that is both persistent and systemic. Acute toxicity studies to honey bees show

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103. Avery Fellow, *Beekeepers Sue EPA Over Pesticide Approvals*, BLOOMBERG (March 22, 2013 10:55 AM), http://www.bloomberg.com/news/2013-03-22/beekeepers-sue-epa-over-pesticide-approvals.html.

104. EPA, EPA MEMO: CLOTHIANIDIN REGISTRATION OF PROSPER T400 SEED TREATMENT ON MUSTARD SEED AND PONCHO/VOTIVO SEED TREATMENT ON COTTON 53 (Nov. 2, 2010), *available at* http://www.epa.gov/pesticides/chem_search/cleared_reviews/csr_PC-044309_2-Nov-10_b.pdf.

105. ANITA PEASE, EPA, PC Code 044309, RECLASSIFICATION OF MRID 46907807/46907802 DATA PACKAGE 336888 FOR CLOTHIANIDIN, (Dec. 22, 2010), *available at* http://www.epa.gov/pesticides/chemical/foia/cleared-reviews/reviews/044309/044309-2010-12-22a.pdf.

106. *Id*.

107. EPA Memo: Clothianidin Registration of Prosper T400 Seed Treatment on Mustard Seed and Poncho/Votivo Seed Treatment on Cotton, EPA, 1 (Nov. 2, 2010), http://www.epa.gov/pesticides/chem_search/cleared_reviews/csr_PC-044309_2-Nov-10_b.pdf.

> that clothianidin is highly toxic on both a contact and an oral basis. Although EFED does not conduct RQ based risk assessments on non-target insects, information from standard tests and field studies, as well as incident reports involving other neonicotinoids insecticides (e.g., imidacloprid) suggest the potential for long term toxic risk to honey bees and other beneficial insects.[108]

Nonetheless, clothianidin was conditionally registered and remains registered today, despite Bayer's failure to comply with the EPA's requests for information about its ecological impact, and despite the EPA's knowledge of clothianidin's toxicity to bees. Using the conditional registration process and a protracted campaign of delay and reluctant compliance, Bayer has de facto avoided the EPA's scrutiny, and has successfully sold clothianidin since 2003.

The EPA should at least suspend the use of clothianidin immediately. Preferably, because of the magnitude of the potential harm involved, the EPA should declare that clothianidin poses an "imminent hazard" to the environment and immediately freeze all sale and use of the pesticide. Clothianidin was never adequately scrutinized by the EPA to ensure that it is safe before it was granted conditional registration. Multiple studies[109] have linked clothianidin to colony collapse disorder. Leaving clothianidin on the market poses an unreasonable risk to the economy and the environment, and the EPA should immediately suspend its registration. If the pesticide is later proven to be safe, the EPA can register it again. However, until clothianidin is proven to be safe, using it is an unreasonable risk.

Despite the shortcomings of the analysis of clothianidin and the clearer connection between clothianidin and colony collapse disorder, other neonicotinoids are still suspect. While clothianidin should be suspended or canceled until the EPA at least conducts a review, other neonicotinoid products also deserve heightened scrutiny. The EPA should strongly consider a temporary suspension until additional research data can conclusively prove that neonicotinoids are safe.

Going a step further, the EPA should change its stance of permitting registered pesticides to remain in use until proven harmful. Instead, the EPA should suspend pesticides from use as soon as evidence arises that suggests they are unsafe, until the pesticide manufacturer submits sufficient research data to prove the pesticide is safe.

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108. *Id*. at 2.

109. *See supra* note 22.

X. CONCLUSION

In the specific case of neonicotinoids, the EPA should immediately suspend the registration of all neonicotinoid pesticides. Leaving neonicotinoids on the market despite the possibility that they are causing a massive die-off of bees is an incredibly high-stakes gamble. Leaving neonicotinoids in widespread use is an egregiously unreasonable risk to both the economy and the environment.

In the case of neonicotinoids, the consequences would certainly be very severe, but with an unknown probability. Nonetheless, the EPA has presently decided to allow the use of neonicotinoids until scientific certainty can be established. The EPA should not wait until neonicotinoids are proven to be harmful to suspend its registration. Instead, the EPA should suspend any pesticide as soon as compelling evidence creates doubt about its safety. Then, once the pesticide is later proven to be safe it can once again be registered for widespread agricultural use.

However, neonicotinoids are not the final word about environmental precaution because a similar problem will inevitably arise sometime in the future. Neonicotinoids are a cautionary tale about a fundamental mistake in environmental risk analysis; a tendency to gamble by failing to give potentially massive unknowns their due respect. Cavalierly ignoring the worst-case scenario is a foolhardy gamble. Regardless of whether the gamble succeeds or fails for neonicotinoids, it is a dangerous error that must not be repeated.

The EPA must determine whether a pesticide should be registered for use, or whether it constitutes an unreasonable risk. In order to make that decision, the EPA must consider the benefits of the pesticide and the potential consequences of allowing it to be used. Such an analysis is straightforward if all the facts are available about the consequences of the decision. But the EPA is sometimes forced to make decisions in the face of scientific uncertainty. The EPA must weigh costs and consequences when it may be difficult to estimate the magnitude or probability of those consequences and inaction could be disastrous.

Under the EPA's current policy, the EPA will leave a pesticide on the market until it is scientifically proven to be harmful. If the scientific community reaches a consensus that neonicotinoids are harmful, the EPA will most likely ban them at that time. But even if neonicotinoids eventually turn out not to be the cause of colony collapse disorder, leaving them on the market while uncertain of that fact is still a potentially catastrophic mistake.

The EPA's decision should be remembered because this exact analysis from a position of uncertainty will arise again, with a different

pesticide or perhaps some other technology. Evidence will arise about a new pesticide or perhaps some other technology currently in use suggesting it might have potentially disastrous consequences, but no scientific certainty (yet) that it definitely causes those dire consequences. In all such cases the EPA should temporarily suspend, cancel, or otherwise prohibit the use of that pesticide. The substantial costs and lost profits from temporary and reversible suspension are dwarfed by potentially catastrophic and irreversible consequences from leaving the pesticide in use with so many unknowns.

The story of the adoption of neonicotinoids should serve as a cautionary tale about the burden of proof, the misallocation of risk, and the failure to respond to large uncertainties. The proponent of the potentially risky action or activity must prove that the activity is safe before it should be accepted. In the case of pesticides, it is wrong that others must first prove a pesticide is dangerous before the EPA will cancel its registration. Instead, the EPA must always require the pesticide manufacturer to submit research data to prove the pesticide's safety. When new evidence creates doubt about an already-registered pesticide's safety, the EPA should suspend its registration until the manufacturer can prove the pesticide is safe. Leaving a pesticide in commercial use when we don't know if it causes catastrophic harm is a categorically unreasonable risk to both the environment and the economy.