

Neonicotinoids are a class of insecticide implicated as a possible contributor to Colony Collapse Disorder (CCD). Current research focuses on the chronic toxicity of neonicotinoids at levels below those that are acutely toxic. Both the Environmental Protection Agency (EPA) and the United States Department of Agriculture (USDA) are actively researching CCD. The EPA has sole authority over federal pesticide regulation in the United States. At this time, based upon a lack of conclusive evidence, as cited below, we are not in a position to second guess the EPA's decision to uphold the registration of neonicotinoids for home use.

### **In summary of the current situation**

“The cause of CCD is currently unknown. Scientists are researching several possible causes including pesticides, parasites, disease, poor nutrition and limited or contaminated water supplies.” **3/1/13, [npic.orst.edu/envir/ccd.html](http://npic.orst.edu/envir/ccd.html)**

“A number of factors have been associated with declines in honey bees in North America including disease, pests, poor nutrition, loss of habitat, pesticides and bee management practices; however, none of these factors have been identified as the cause. Researchers have hypothesized that some of these factors may interact and result in losses which are attributed to winter kill and CCD. Although pesticides have not been implicated as the singular cause of insect pollinator declines in general or of declines in honey bees specifically, efforts have been directed at determining the extent to which pesticides may be affecting bees and ways to mitigate potential effects.” **September 2012, EPA**

“While the exact cause(s) of the general decline in pollinator species and the phenomenon characterized as CCD have not been determined, potential contributing factors including diseases, habitat destruction/urbanization, agricultural practices/monocultures, pesticides, nutrition, and bee management practices must be considered. Researchers at the USDA have hypothesized that CCD may be caused by primary stressors (e.g., parasitic varroa mites (*Varroa destructor*), poor bee management, nutrition and/or pesticides) that may in turn cause honey bees to become more susceptible to disease. Surveys of managed migratory bee colonies indicate that a broad range of pesticides have been detected in hive products (e.g., honey, stored pollen, wax). The most frequently detected pesticides and the two that occur in the highest quantity are those used by beekeepers to control varroa mite (coumaphos and fluvalinate). In spite of the presence of these compounds in honey bee colonies, at this time, there has been no correlation between the incidences of CCD or pollinator declines in general, with use of any pesticide or class of pesticides.” **September 2012, EPA**

“Toxicity tests used to evaluate potential effects have been tiered and have first examined effects on individual bees under laboratory conditions while more refined testing is conducted using whole hives under field conditions. These processes are continuing to evolve to reflect the changing science.” **September 2012, EPA**

“Although a number of factors/agents have been hypothesized as potential contributors to CCD, increased overwintering losses, and declines in honey bee health in general, at this time, no factor has been identified as the single cause. Rather, the available science suggests that pollinator declines are a result of multiple factors which may be acting in various combinations. Research is being directed at identifying the individual and combinations of stressors that are most strongly associated with pollinator declines.”

**September 2012, EPA**

“Search for a clear smoking gun has been elusive because of the multitude of parasites, viruses, and fungal diseases the bees must contend with, as well as hive chemicals, pesticides and environmental toxins.”

**2/28/13, Gary Rondeau,**

**oregonsustainablebeekeepers.org**

“Many factors could be involved in the lower level of overall losses in 2011, including the mild winter weather in many parts of the United States.” **June 2012, USDA**

“Although a number of factors continue to be associated with CCD, including parasites and pathogens, poor nutrition, pesticides, bee management practices, habitat fragmentation, and agricultural practices, no single factor or pattern of factors has been proven to be ‘the cause’ of CCD.” **June 2012, USDA**

“Research has shown that weak colonies had overall higher pathogen levels and evidence of pesticide residues, although no pattern of specific pathogens or pesticides was indicated. Colonies in comparatively good health also contained a wide range of pesticide residues.” **June 2012, USDA**

“Previously, viruses and other pathogens and parasites were found to be present at greater levels in colonies affected by CCD than in those not affected by CCD. Studies in 2010 revealed several new viruses and other pathogens affecting honey bees.” **June 2012, USDA**

“A survey of bees, honey, and comb for the presence of 170 pesticides or pesticide residues performed in 2010 did not find any pattern of exposure that correlated with CCD incidents, which would be expected if pesticides were a major factor in causing CCD. The pesticides detected with the greatest frequency and in the largest quantities were those used by beekeepers to control *Varroa* mites. Pesticide effects on bees continue to be a subject area of intense research.” **June 2012, USDA**

“Analysis of a broad set of healthy and CCD-affected hives did not reveal a sole causative agent but rather a host of viral, bacterial, and fungal pathogens that occurred at higher levels in CCD-affected hives.” **June 2012, USDA**

“Recent efforts by Cornman and colleagues did not identify a single unique pathogen that could be a CCD causative agent, but the data did lend support to earlier studies suggesting that whereas a complex set of pathogens may be involved in the cause of colony losses, no single pathogen can be solely linked to CCD.” **June 2012, USDA**

“Research efforts jointly supported by ARS and NIFA continue to investigate numerous factors alone or in combination that may play a role in causing CCD. These include parasites and pathogens, pesticides, poor nutrition, bee management practices, and to a lesser extent, other pests such as the small hive beetle (*Aethina tumida*). The *Varroa destructor* mite remains one of the primary threats to honey bee health.” **June 2012, USDA**

“It is difficult to point to direct effects of insecticides *per se* because the direct cause of the colony decline may be a combination of viruses and other pathogens. Locally, we have not seen the same level of losses reported in some of the more agricultural sections of the country.” **12/4/12, personal communication from Philp Smith, Oregon Sustainable Beekeepers**

“To date, we’re aware of no data demonstrating that an EPA-registered pesticide used according to the label instructions has caused CCD.” **May 2012, EPA**

“Exposure to dust drift from sowing treated seeds was identified as a relevant exposure route.” **May 2012, EFSA**

“Two other studies have measured multiple factors associated with CCD and non-CCD colonies across the US to see what risk factors were predictive of CCD. The first study looked at one factor at a time among 61 variables as potential causes of CCD and found that no one factor could account for CCD.” **August 2011, Frazier et al. (Penn State University)**

“Our residue results based on 2210 samples which include Mullin et al. (2010) and subsequently more than 230 additional samples do not support sufficient amounts and frequency of imidacloprid in pollen to broadly impact bees.” **August 2011, Frazier et al. (Penn State University)**

### **There is a well established need for additional research**

“The beekeepers feel that the very sub-acute doses of neonicotinoids are harming their bees. That has not been demonstrated with any short- or long-term studies that have been published, to my knowledge. If plants are treated with neonicotinoids and they flower, the nectar and pollen will contain measurable levels of the chemicals. Whether or not that is important to native and honey bee health still is unresolved.” **2/26/13, personal communication from Eric Mussen, UC, Davis**

“EFSA’s intention to continue exploring the subject is thus a necessary next step...” **December, 2012, European Parliament**

“EFSA concluded that before drawing definite conclusions on the behavioural effects of sublethal exposure of foragers exposed to actual doses of the tested neonicotinoids (thiamethoxam, clothianidin and imidacloprid) – and the consequences for the bee colonies –, it would be necessary to repeat the tests with different exposure levels and/or in different situations. According to Tennekes (2010) “time-to-effect approaches” (which provide information on the doses and exposure times) would be needed to have a better idea about toxic effects of neonicotinoids on beneficial organisms. They would be necessary because the consideration of toxic effects at fixed exposure times does not allow extrapolation from measured endpoints to effect that may occur at other times of exposure.” **December, 2012, European Parliament.**

Field tests with similar concentrations of neonicotinoids in bee feed have not duplicated the effects of laboratory tests. “Apparently there is a buffering action of some sort in the hives.” **2/26/13, personal communication from Eric Mussen, UC, Davis**

“Increased levels of refinement focus on areas where specific risks may exist and are intended to be increasingly representative of actual use/exposure conditions.”  
**September 2012, EPA**

“The unusually warm 2011-2012 winter could be one contributing factor to the drop in colony losses, although no direct scientific research has been performed to discern whether a connection exists between weather and CCD.” **June 2012, USDA**

“*Nosema ceranae*, a microsporidial pathogen that was recently introduced into the United States, was the dominant species of *Nosema* detected. *N. ceranae* has been tentatively linked in some studies as contributing to CCD in the United States, although no conclusive evidence has been found.” **June 2012, USDA**

“Some recent scientific evidence appears to indicate that some pesticides may have sublethal effects on honey bees; however, the relevancy of some of these effects to estimating overall risks to honey bees is uncertain.” **June 2012, USDA**

“Many potentially associated factors have been identified throughout the course of research, a number of which appear to have a high correlation with the pattern of CCD incidents. However, the strength of these associations has varied considerably, and it has become increasingly clear that no single factor is responsible for the syndrome. In addition, research has not been able to determine whether all cases of CCD are caused by the same set of factors or the same factors in a particular combination.” **June 2012, USDA**

“Other data indicate that some pesticides at high concentrations interact with other pesticides, honey bee parasites, or pathogens in ways that significantly increase individual bee mortality rates. Further studies are needed to ascertain whether these synergistic effects occur at environmentally relevant concentrations or whether managed honey bee colonies are commonly exposed to these levels of pesticides.” **June 2012, USDA**

“Researchers have not yet determined how honey bees and other pollinators react when exposed to environmentally relevant levels and whether a significant number of bees would likely be present in or around fields that are planted or seeded.” **June 2012, USDA**

“In study results published in 2012, exposure of individual, immature honey bees to sublethal levels of neonicotinoids resulted in increased susceptibility to the gut pathogen *Nosema*, although the response was not concentration dependent. The colony’s overall health, population, ability to gather nectar, and pollinate were not affected by these sublethal effects on individual bees, but this study does demonstrate that there are complex interactions among various factors that could contribute to weakening individual bees and making them more susceptible to additional perturbations.” **June 2012, USDA**

“What is clear is that researchers must look beyond simple one-factor causes of bee decline and losses. This research is multifactoral and more challenging.” **June 2012, USDA**

“There are no adequate studies of long-term sub-lethal effects of these very toxic chemicals that focus on whole-colony dynamics with honey bees.” **2/9/13, Gary Rondeau, oregonsustainablebeekeepers.org**

“The overview of the available studies on such sub-lethal doses and long-term effects of pesticides on bees, mostly available from laboratory studies, highlighted gaps in knowledge and future research needs.” **May 2012, EFSA**

“Field testing currently follows the EPPO 170 (4). A scientific assessment of these guidelines showed that it has several major weaknesses (e.g., the small size of the colonies, the very small distance between the hives and the treated field, the very low surface of the test field), leading to uncertainties concerning the real exposure of the honey bees.” **May 2012, EFSA**

“There is no direct link demonstrated between neonicotinoids and the honey bee syndrome known as CCD. However, recent research suggests that neonicotinoids may make honey bees more susceptible to parasite and pathogens, including the intestinal parasite *Nosema*, which has been implicated as one causative factor in CCD.” **2012, Xerces Society**

“At the end of 10 weeks, eight of 30 [colonies] tested positive for *Nosema* but there was surprisingly no relationship between *Nosema* infection and imidacloprid treatment which would have been predicted by the lab study.” **December 2011, Pettis et al.**

“Individual bees in our study showed a marked increase in *Nosema* spore production in the laboratory but the parent colonies failed to show increased *Nosema* levels over time.” **December 2011, Pettis et al.**

“To our knowledge no studies have examined chronic effects of dietary exposure to imidacloprid in functional colonies over multiple brood cycles and potential synergistic effects of pesticide and disease interactions.” **December 2011, Pettis et al.**