

Study suggests common pesticide clothianidin causes immunity problems in bees

October 22 2013, by Bob Yirka



(Phys.org) —A team of researchers with members from several universities in Italy has found that exposure to the common pesticide clothianidin can cause immunity problems in honeybees, leading to an increased risk of dying from common viral infections. In their paper published in *Proceedings of the National Academy of Sciences*, the researchers found that exposure to clothianidin resulted in an increase in a family of proteins that inhibit the development of other proteins that are involved in the immune process.

Headlines over the past couple of years have made it very clear that

something is causing [honeybees](#) to die in unexplained ways. Whole colonies suddenly die, with no clear explanation. Now known as Colony Collapse Disorder (CCD), the problem has reached the point of panic as honeybees are the chief means for pollination of crops around the world. At this point, scientists suspect that the disorder involves something that is causing the immune system in the [bees](#) to break down, leaving them unable to fight off bacteria and viruses. In this new effort, the research team contends that they've found one of the missing links—a single pesticide that causes bees exposed to it, to develop immunity problems.

In their lab, the researchers started by isolating a family of proteins (called LRR) that are similar to other proteins found in other animals that are known to regulate immune response—specifically, its presence, they found, causes another protein (NF- κ B) directly involved in immune response, to be inhibited. Next, they exposed honeybees to the pesticide clothianidin and subsequently measured gene expression and [protein](#) levels in them. They found an increase in the expression of the gene responsible for LRR levels and lowered levels of NF- κ B, which the researchers claim, suggests a direct link between exposure to the toxin and a damaged [immune system](#). The researchers ran the same tests on bees exposed to another pesticide— chlorpyrifos—and found no ill effects, which they suggest means CCD might be caused by one or a just a few pesticides. Next, the researchers exposed the bees that had been exposed to clothianidin to a pathogen called the deformed wing virus. Normally, healthy bees show resistance to the virus and are not impacted by it. After exposure to clothianidin, however, the researchers found the virus was able to reproduce in the bees, suggesting the bee's [immune response](#) had been compromised.

More information: Neonicotinoid clothianidin adversely affects insect immunity and promotes replication of a viral pathogen in honey bees, *PNAS*, Published online before print October 21, 2013, [DOI: 10.1073/pnas.1314923110](#)

Abstract

Large-scale losses of honey bee colonies represent a poorly understood problem of global importance. Both biotic and abiotic factors are involved in this phenomenon that is often associated with high loads of parasites and pathogens. A stronger impact of pathogens in honey bees exposed to neonicotinoid insecticides has been reported, but the causal link between insecticide exposure and the possible immune alteration of honey bees remains elusive. Here, we demonstrate that the neonicotinoid insecticide clothianidin negatively modulates NF- κ B immune signaling in insects and adversely affects honey bee antiviral defenses controlled by this transcription factor. We have identified in insects a negative modulator of NF- κ B activation, which is a leucine-rich repeat protein. Exposure to clothianidin, by enhancing the transcription of the gene encoding this inhibitor, reduces immune defenses and promotes the replication of the deformed wing virus in honey bees bearing covert infections. This honey bee immunosuppression is similarly induced by a different neonicotinoid, imidacloprid, but not by the organophosphate chlorpyrifos, which does not affect NF- κ B signaling. The occurrence at sublethal doses of this insecticide-induced viral proliferation suggests that the studied neonicotinoids might have a negative effect at the field level. Our experiments uncover a further level of regulation of the immune response in insects and set the stage for studies on neural modulation of immunity in animals. Furthermore, this study has implications for the conservation of bees, as it will contribute to the definition of more appropriate guidelines for testing chronic or sublethal effects of pesticides used in agriculture.

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