# Overview of Recent Publications on Neonicotinoids and Pollinators

Several recently-published studies have reported on evaluations of the impact of neonicotinoid insecticides on pollinators,, often accompanied by considerable media attention claiming singular importance in explaining overall pollinator health status. These studies included evaluations of potential effects of sublethal doses of neonicotinoids on honey bees, the potential effects of exposure to neonicotinoid-contaminated diet on bumble bee colony development, and the potential exposure of pollinators to neonicotinoids resulting from planting of treated seeds. The results of these studies are summarized below and discussed in light of the results of many years of research on the risks of neonicotinoids to bees, including a summary of recent review articles interpreting the extensive literature on neonicotinoids.

## Sublethal Exposure of Honey Bees

Three studies [Henry, et al. (2012), Schneider et al. (2012), and Pettis et al. (2012)] evaluated the potential effects of sublethal exposure of bees to neonicotinoid insecticides. Henry, et al. (2012) and Schneider (2012) employed the use of radio frequency identification (RFID) tags on honey bees to study whether exposure to a neonicotinoid insecticide impairs the ability of forager bees to return to the hive and if so, whether this is likely to have consequences for the long-term survival of the colony. The first research team used thiamethoxam while the second team used imidacloprid and clothianidin in their experiment. However, in the experiments of Henry, et al., bees were tested at a single dose that was over twenty times greater than a worst-case estimate of the acute oral dose that is field relevant, while Schneider et al., tested a range of different exposure concentration which included concentrations corresponding to field-relevant exposure scenarios. As might be expected, Henry, et al., concluded that exposure to thiamethoxam residues in pollen and nectar could lead to adverse effects in pollinating bees while Schneider, et al., concluded that “at field-relevant doses for nectar and pollen no adverse effect was observed for either substance”. It is highly likely that Henry et al. (2012) would have come to the same conclusion for their test substance thiamethoxam, if they would have followed Schneider et al.’s scientifically sound approach to test a range of concentrations that also included field-relevant dose rates.

The conclusions of Schneider et al. (2012) are confirmed by more than 30 field studies conducted with neonicotinoids where no effect on foraging and homing behavior of honeybees exposed to treated crops has been observed (see for instance Maus et al. 2003, Schmuck et al. 2005, Schmuck & Keppler 2003). In fact, there is no field evidence linking hive depopulations to sublethal exposures to neonicotinoids.

In the recently issued publication of Pettis et al. (2012), interactions between chronic, sublethal exposure of honeybee colonies to imidacloprid, and the infection of individual honeybees with the fungal gut parasite *Nosema* were investigated. This study found that individual honey bee vulnerability to *Nosema* infection was enhanced by the presence of the neonicotinoid imidacloprid when the *Nosema* infection and exposure part of the study was conducted under artificial laboratory conditions with individual worker bees out of the context of the colony and its complex interactions and compensation mechanisms. In such an artificial laboratory

environment, bees may react quite different from a situation under realistic field conditions. The impact of the artificial laboratory conditions is demonstrated by the fact that the parent colonies in the Pettis, et al., study, which were treated with imidacloprid but left in the field, failed to develop increased *Nosema* levels related to the exposure. In fact, the lowest spore counts were found in the colonies exposed to the highest pesticide concentration.

From the reported findings, the authors conclude an interaction between sub-lethal exposure to imidacloprid at the colony level and *Nosema* spore production in individual bees, and postulate that sublethal exposure of honeybee colonies to pesticides may cause adverse effects by making the bees more susceptible to pathogens. They noted that this risk has so far been overlooked in the pesticide risk assessment process. However, the study clearly shows that under realistic field conditions exposure to imidacloprid did not increase the *Nosema* spore count and once more underlines the fact that field conditions are fundamentally different from what can be tested in the laboratory, and that laboratory data cannot be directly extrapolated to the field.

## Bumble Bee Colony Development

The effects on bumble bee colony development resulting from exposure to imidacloprid-contaminated diet were evaluated in a study conducted by Whitehorn, et al. (2012). After the initial exposure period the control colonies gained more cumulative weight and produced significantly more queens than the treatment colonies. However, the test design applied by Whitehorn et al. (2012) was not validated and the reported lower colony weight of treatment colonies compared to control colonies is basically just a difference between different experimental groups. The results presented in this study originate from a study conducted under artificial conditions and are in conflict with the results of Tasei et al. (2001) and Gels & Potter (2002). These authors reported no effects on bumble bees when imidacloprid was applied as a sunflower seed treatment and to lawns containing clover, respectively, when the compound was applied according to label directions.

## Exposure of Bees to Neonicotinoids from Use of Treated Seeds

Two recent studies evaluated the potential exposure of honey bees to pesticides resulting from the use of neonicotinoid treated seeds. Krupke et al. (2012) report the findings of a study on potential routes of exposures for honeybees to pesticides, especially to neonicotinoids, conducted in a corn-growing region in USA in 2010. The study was initiated in response to reports of bee kills at Indiana apiaries in spring of 2010 which coincided with the corn planting period in the area and which were believed to be related to neonicotinoid seed treatment products.

This study does not provide any fundamentally new evidence about honeybee exposure to neonicotinoid seed treatment products. The exposure levels reported in soil, pollen and nectar are generally consistent with previous research and were not high enough to represent a significant risk for honey bees. Higher concentrations found in waste talc collected from inside pneumatic equipment post-planting represent an intrinsic hazard to honey bees; however actual exposure of bees to this material was not demonstrated and would appear to be preventable. In their field experiment, low exposure levels and no adverse effects were observed for bee colonies placed “in harm’s way” around the perimeter of a field as it was planted with treated corn seeds. Overall, the

publication represents an interesting case study, but it does not provide any significant new insights into exposure of honeybees to neonicotinoid insecticides.

Tapparo et al. (2012) report on results of field experiments that measured emissions of particulate matter containing neonicotinoid insecticides from the sowing of dressed maize seeds and resulting potential exposure levels for honey bees. Various types of treated corn seeds were sown into a test field using two different types of pneumatic planters and the amount of total particulate matter and active ingredient emitted into the air and deposited at various distances away from the planter or downwind edge of the field were determined. The experiments were run with and without downward deflectors mounted on planter's air exhaust outlet. Two different types of experiments were performed: "mobile sowing" and "static sowing". As part of the static sowing experiments, sugar syrup feeders and honey bee hives were placed so that bees would fly directly through the air exhaust of the planter, and become "powdered" with any emitted dust.

The authors conclude that particulate matter released by drilling machines during sowing of maize seeds coated with neonicotinoid insecticides represents a significant mechanism of environmental diffusion of these insecticides. Bees flying over the sowing field and approaching the emission cloud of the drilling machine can efficiently intercept the suspended particles being directly contaminated with an elevated dose of insecticide, significantly higher than contact LD50 values. These exposures, according to the author, therefore represent a concern for both apiculture and crop production based on bee pollination.

These results demonstrate that honey bees that fly through the air exhaust of pneumatic corn planters can become contaminated with abraded dust from insecticide-treated maize seeds and this can sometimes result in the death of individual bees. However, their research results and the available records of field incidents suggest that the problem of toxic exposure of bees to corn seed dust is limited in scope, and continues to be minimized with improved seed coatings/lubricants, planter modifications and product stewardship measures. This phenomenon has not been scientifically linked to, and is not suspected by mainstream scientists to be the cause of colony collapse disorder or widespread honey bee colony losses.

In their publication "In situ replication of honey bee colony collapse disorder", Lu et al. (2012) describe a trial in which they claim they have replicated inducing symptoms of Colony Collapse Disorder (CCD) by chronically exposing bee colonies to high-fructose corn syrup (HFCS) contaminated with imidacloprid, and bring forward a new hypothesis about the origin of CCD. The authors hypothesize that CCD is caused by imidacloprid residues originating from seed treatment in corn in HFCS which is used to feed honeybee colonies. However, the study was conducted according to a faulty design that was based on numerous incorrect and unsupportable assumptions which are totally inconsistent with a sound scientific approach. Not least amongst the assumptions is the fact that high levels of Imidacloprid in HFCS were tested in the study despite the fact that Imidacloprid or other neonicotinoids have not been detected in HFCS nor would be expected based on the highly purified nature of HFCS. The results are accordingly insignificant for any risk assessment; moreover the authors interpret them in a very questionable way. The study provides no evidence at all that the exposure of honeybee colonies to neonicotinoids under realistic conditions might have any adverse effect.

## Neonicotinoids in Bees: Review and Risk Assessment

Blacquiere, et al., summarized fifteen years of research on the risks of neonicotinoids to bees by looking at neonicotinoid residue levels in plants, bees and bee products as well as reported side-effects with special attention to sublethal effects. They then looked at the potential of using an existing risk assessment scheme designed for systemic compounds to evaluate neonicotinoids. They point out that "it is now accepted that the abundance of pollinators in the environment is influenced by multiple factors, including biotic ones like pathogens, parasites, availability of resources due to habitat fragmentation and loss; and abiotic ones like climate change and pollutants." They further note that although "the extensive use of chemical pesticides against pest insects for crop protection may have contributed to the loss of pollinators", "to feed the fast growing global population, chemical insecticides are important to crop productivity … preserv(ing) one-fifth of the crop yield." The authors conclude that the reported levels of neonicotinoids in nectar and pollen are below acute and chronic toxicity levels and the levels in bee-collected pollen, bees and bee products are low. They encourage collection of additional residue data before drawing final conclusions. Blacquiere, et al., note that laboratory studies have shown many lethal and sublethal effects of neonicotinoids, but no effects have been observed in the field studies with field-realistic dosages.

Cresswell, et al., used Hill's epidemiological causality criteria to examine the evidence that the agricultural use of neonicotinoids is a cause of the recently observed decline in honey bees. They note that "the question of whether neonicotinoids cause bee population declines would be settled beyond reasonable doubt if realistically dosed honey bee colonies showed sufficient harm under field conditions. Based on their assessment of the available data they "conclude that dietary neonicotinoids cannot be implicated in honey bee declines." This conclusion is supported by the field studies that have shown no adverse effects from neonicotinoids applied according to label directions. As with Blacquiere, et al., Cresswell, et al., recommend further investigation to resolve remaining uncertainty.

## Conclusions

A scientific evaluation of all of the available data demonstrates that neonicotinoids are toxic to honey bees when exposed to sufficient concentration under artificial laboratory conditions or when exposed to abraded dust from poorly treated seed. However, exposure of bees to residues in pollen and nectar in plants grown from seed properly treated with neonicotinoids does not contribute to bee decline. Specifically, an evaluation of the above studies shows that at field-relevant doses for nectar and pollen no adverse effects (lethal or sublethal) are observed for neonicotinoids. Similarly, no adverse effects were observed when bumble bees are exposed to neonicotinoids used according to label directions. Finally, the available records of field incidents suggest that the problem of toxic exposure of bees to dust from treated seed is limited in scope, and can be minimized with improved seed coatings/lubricants, planter modifications and product stewardship measures. Therefore, as supported by most researchers and government agencies, the available data continue to demonstrate that the decline of bee health, particularly in pollinator honey bees, is the result of many factors including pathogens, parasites, pesticides, habitat, bee management and others (e.g.USDA, 2011; Delaplane, 2012).

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