

Neonicotinoid Pesticides and Bees

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Introduction

Recently, the Washington State Department of Agriculture (WSDA) responded to a request from Thurston County Commissioners regarding the use of neonicotinoids in urban areas by unlicensed pesticide applicators. The concern of Thurston County is the impact of over use of neonicotinoids in urban areas and the potential effect they may have on honey bees and other pollinators. The WSDA has opted to not move forward with new rule making on the use of neonicotinoids at this time, primarily due to the lack of data on the urban use of these products and their impact on the pollinators; and the uncertainty about singling out any variable as the definitive cause of the decline of honey bees and native pollinators (See <http://agr.wa.gov/News/2013/13-19.aspx>).

WSDA will focus more attention on this class of chemicals and continue to look at ways of reducing the risk to bees including gathering additional data to address some of the concerns presented by Thurston County. This fact sheet is intended to provide you with information you need to answer questions about the decline of honey bees and other pollinators and concerns regarding the impact of systemic pesticides. Included in this fact sheet is a list of a few of the interesting studies on the impact of neonicotinoids on honey bees and bibliography on various studies related to neonicotinoids, and other variables associated with the decline of honey bees often referred to as colony collapse disorder or CCD.

Industry Use

Neonicotinoids have become an increasingly important class of pesticide for agricultural, landscape and residential use. There are currently more than 465 products containing neonicotinoids (often called neonic's) approved for use, in the State of Washington of which approximately 150 are approved for use in the home or garden. Because of their relative mammalian safety and efficacy they are one of the fastest growing classes of chemicals (Jeschke and Nauen, 2008). One of the main advantages for using neonicotinoid products is that they work systemically within the plant, thus reducing the direct exposure to the applicator and the environment. Ironically it is this systemic action that makes the neonic's a problem for honey bees and other pollinators. The neonicotinoid pesticide carried within the plant can also be expressed in the nectar and pollen of the flowers.

Uptake by Bees

In laboratory experiments researchers have documented several neonicotinoid products that are toxic to bees. Depending on the exposure, the effect on the bees can be lethal or sub-lethal. The sub-lethal effects of neonicotinoids include impaired learning behavior, short and long term memory loss, reduced fecundity, altered foraging behavior, and motor activity of the bees. Researchers have documented similar issues with other pesticides including some products used by beekeepers to control Varroa, a parasitic mite of the honey bee.

European Concerns

The current discussion presented by a growing number of individuals draws a direct link to the decline of native bees and honey bees in the use of neonicotinoids. The European Union has suspended the use of neonicotinoids for two-years as they reassess the impact of this material. Clearly there is justification for taking a closer look at neonicotinoids and placing a precautionary emphasis on their use, but at this point there are insufficient data to suggest that this product is a substantial contributor to the decline of either native bees or honey bees. The value and the benefit of neonicotinoids to agriculture, professional landscapers, and homeowners as a relatively safe and effective product should be considered when making a determination on availability and restrictions for this class of pesticides.

Lab versus Field

Most of the studies on neonicotinoid toxicity to bees have been documented in laboratory studies attempting to emulate field exposure levels to the bees. The handful of field experiments have been conducted in plots where the predominate source of nectar and pollen comes from plants treated with neonicotinoids. Individual honey bees forage on just one source of pollen or nectar at any one time, but the colony as a whole will collect from a variety of sources when available. Most bumble bees and other native bees are generalists and will forage on a diverse array of nectar and pollen sources on any given trip.

Urban Concerns

The suggestion that bees are more likely to be exposed to neonicotinoid pesticides in urban areas where homeowner application of the material may be common is, at this point, undocumented. There are virtually no data showing levels of neonicotinoid use in urban areas being in excess of the levels demonstrated to have either lethal or sub-lethal effects on bees. Further, it is unlikely that all plants grown in urban areas would be treated with the product. Thus, it seems likely that urban exposures would be far less than levels experienced by bees in agricultural monocultures. Prior to enacting restrictions on urban homeowner use of this product, it would be prudent to collect data to quantify urban homeowner use of neonicotinoid pesticides and pollinator exposure from this source.

Decline in Honey Bee Populations

Sudden disappearance of bees has been reported by beekeepers and researched by scientists for decades and was often called Disappearing Disease (Wilson and Menapace, 1979). In 2006 this phenomenon suddenly became more widespread and has been coined by researchers and the media as Colony Collapse Disorder or CCD. The increase in colony losses corresponded to the increased use of neonicotinoid pesticides (Johnson *et al.*, 2010; Cresswell *et al.*, 2012). This has led many beekeepers to speculate that there is a causative relationship between the increased use of neonicotinoids and this widespread decline in bee populations (Suryanarayanan, 2013). However, to put things in their proper context it is important to look at all the variables associated with CCD.

Reports of dramatic declines in honey bee stock have been widely reported especially in the United States and Europe (Mullin *et al.*, 2010), however, FAO data reveal that globally there has been a ~45% increase in managed colonies since 1960 (Aizen and Lawrence, 2009). The definitive cause for the declines in the United States and Europe has of yet to be fully understood, however, more than 61 variables have been associated with CCD, but none have been clearly identified as the definitive cause of the phenomena (Evans *et al.*, 2009). Some of the major factors associated with the decline in honey bee stocks in the United States include the Varroa mite, pesticides, pathogens, loss of habitat, and nutritional deficiencies. One additional stress placed on honey bees is the intense management strategies needed to ensure strong colonies for almond pollination in California from mid-

February through mid-March. Researchers have ruled out individual stressors such as long distance hauling, of bees on tractor-trailer trucks (Ahn *et al.*, 2012). However, recent studies have placed some additional concerns on the “feed-lot” feeding widely practiced by commercial beekeepers. Beekeeper’s reliance on high-fructose corn-syrup and sucrose in these feed lot situations where tens of thousands of bees are kept prior to their movement into the almond orchards may significantly reduce the bees ability to detoxify pesticides (Mao *et al.*, 2013). Similarly, beekeeper’s reliance on pollen substitute may make adult bees more susceptible to the effect of some pesticides (Jeri Wright, personal communication).

Varroa Mite

Clearly the Varroa mite is playing a major role in the decline of managed honey bee colonies in the United States. Not only the actual impact of the mite, an ectoparasite that impacts adults, pupae, and larvae by feeding on its hemolymph, but also the chemical control measures used by beekeepers to control the Varroa mite. Beekeepers routinely use both registered and unregistered products to control the mite. Without treatment colonies would be dead within two years from exposure to Varroa. Two studies have found the highest levels of pesticides in bees wax and pollen from commercial honey bee colonies are products used by beekeepers in their effort to control the mite (Wu *et al.*, 2011; Mullin *et al.*, 2010). Interestingly, neonicotinoids found in bees wax and pollen was far less common and at much lower concentrations than the miticides or metabolites of these products commonly used to control the Varroa mite (Mullin *et al.*, 2010). Regardless of the levels of the product found in the colonies, sub-lethal effects of many pesticides including some products used for the control of the mite and neonicotinoids have been shown to cause memory impairment of honey bees at field realistic levels. (Williamson and Wright, 2013).

In Summary

Neonicotinoids clearly have a negative effect on honey bees and other insect pollinators including various important species of native bees. However, it is unclear that at field realistic levels if they have a detectable sub-lethal effect on bees. Exposure levels from dust during planting of neonicotinoid treated seed can have a devastating lethal impact, but this mode of exposure can be avoided and more work needs to be done on controlling levels of dust during planting. The real concern is the chronic exposure to neonicotinoids in nectar, pollen, and water (guttation) picked up by bees and returned to the hive. For now the best means of minimizing any adverse effects may be in increasing awareness of the potential through educational forums and via the product label.

As is evident from the References listed below a great deal of research is currently under way, in both Europe and the United States looking very intently at the effects of neonicotinoids on honey bees. Researchers at the University of Minnesota, Washington State University, and Washington State Department of Agriculture are specifically looking at the issue of neonicotinoids in urban areas. Within the next 8 to 12 months WSU Extension will produce one or more factsheets for the general public and for the small beekeeper on the effects of neonicotinoids on honey bees in urban environments.

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Partial list of selected journal articles

Authors/Date	Pesticide(s)	Species	Setting	Application	Exposure Range	Effects Tested	Significance	Comments
Creswell et al. 2012 Pest. Man. Sci.	neonics	A. mellifera	n/a	oral	n/a	causal to decline	neonics not implic. in decline	A review using epidem. criteria
Elston et al 2013 Apidol.	thia., prop.	B. terrestris	lab	oral	thia. (1, 10ug/kg) prop. (23, 230 mg/kg)	colony initiation. food consump.	thia. - @ 10ug/kg = no larvae prop. - no pop. effect	Both reduced food consumption
Henry et al 2012	thia.	A. mellifera	field	oral	1.34 ng/bee	Homing success (foraging)	Differences in post exposure homing failure between treated and control	Auth. note this field realistic rate doubles the prob. of forager death on a given day.
Krupke et al 2012	var. neonics. and fung. clo., thia., metochlor, azo., tri.	A. mellifera	field/lab	n/a	0 to 52 ppb soil, 4 to 15,030 ppm in talc, 0-4 ppb in field pollen, 0-88 ppb returning forager pollen, etc	n/a	Showed poss. routes of exposure from planting and plant expression, found higher levels in dead and dying bees	Indicated poss. side effect of seed treatments
Laycock et al 2012	Imid.	B. terrestris	lab/nest box	oral	imid. var. 0 to 125ug/L	Ovary develop., fecundity	Dose-dependent decline in fecundity, 1ug/L reduced fecundity by 1/3, no effect of ovary dev. except highest dos.	Fecundity reduction at "environmentally realistic" dosages
Matsumoto 2013 J. Insect.	clo., dino., eto., fen.	A. mellifera	lab/field	topical	var. (0.5 to .025 LD50)	Behavioral/homing success	clo./dino. @ 0.1 LD50 and > eto. @ 0.25 LD50, fen. = n.s.	2 neos, 1 pyreth., 1 OP
Schneider et al 2012 Plos One	imid., clo.	A. mellifera	field	oral	imid. - .15 to 6 ng/bee clo. - .05 to 2 ng/bee	foraging	Not at "field relevant" doses. imid.>.5ng/, clo. > 1.5 ng/ reduc. foraging and longer flight times	Sub-lethal foraging effects
Tapparo et al. 2012	Var. neonics imid., clo., thia., fip	A. mellifera	field/lab	n/a	Varied. = planter exhaust dust, caged bees, etc. foraging bees over field/ planting showed mean clo. 78-1240 ng/bee	(cont.) thia. 128-302 ng/bee	Sim. To Krupke study - high exposure of bees possible during planting	Seed treatment effects. Fip. banned in France following evidence of bee kill during planting.
Whitehorn et al. 2012	imid.	B. terrestris	Lab/field	Oral pollen and sugar water	Pollen 6 ug/kg, 12 mg/kg (low and high) Sugar water 7ug/kg, 14 ug/kg (low and high)	Growth rate, queen prod.	Diff in number of queens produced Control. = 13.7, low 2.0 and high 1.4 queens	Authors" trace levels of neonics can have string neg. consequences for queen production)
Williamson et al. 2013	Imid., coum	A. mellifera	Lab	Oral	Sucrose 10 ⁻¹ , 100 ⁻¹ and 1 ⁻¹ μmol	Short and long term memory	Imidacloprid, coumaphos and a combination of the two compounds impaired the bees' ability to differentiate odor during the memory test.	exposure to sublethal doses significantly impairs foraging and pollinator population decline
Cresswell et al. 2013	Imid	A. mellifera B. terrestris	Lab	Oral	125 μg L-1 imidacloprid, 98 μg kg-1	Feeding and locomotion	imidacloprid did not affect the behaviour of honey bees but it reduced feeding and locomotory activity in bumble bees.	The authors attribute the differential behavioral resilience of the two species to the observed differential in bodily residues.

azo. = azoxystrobin (fungicide); clo. = clothianadin (neonicotinoid); dino. = dinotefuran (neonicotinoid); etof. = etofenprox (pyrethroid); fen. = fenitrothion (organophosphate); fip. = fipronil (GABA blocker); imid. = imidacloprid (neonicotinoid); thia. = thiomethoxam (neonicotinoid); tri. = trifloxystrobin (fungicide); prop. = propiconazole (fungicide); coum. = coumaphos (organothiophosphate)