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# Effect of Neo-Nicotinoids on Beneficial Organisms

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## SUMMARY

Neonicotinoids are now widely of insecticides used group of insecticides in the world, and their use has been used steadily in increasing in the world. Neonicotinoids have been replacing organophosphate and carbamate compounds, use of which are increasingly being restricted due to concerns about pest resistance and effects on human and environment health. Since initial registration in the mid- 1990s, neonicotinoids have been promoted as low-risk chemicals that have low impact on human health, low toxicity to non target organisms, lower application rates, and compatibility with Integrated Pest Management.

## INTRODUCTION

Neonicotinoids are a class of neuro-active insecticides chemically similar to nicotine. The development of this class of insecticides began with work in the 1980s by Shell and the 1990s by Bayer (Kollmeyer, et al., 1999). The neonicotinoids were developed in large part because they show reduced toxicity compared to previously used OP and carbamate insecticides. Neonicotinoids are the first new class of insecticides introduced in the last 50 years, and the neonicotinoid. Imidacloprid is currently the most widely used insecticide in the world. The use of some members of this class has been restricted in some countries due to some evidence of a connection to honey-bee colony collapse disorder. In January 2013, the European Food Safety Authority stated that neonicotinoids pose an unacceptably high risk to bees, and that the industry sponsored science upon which regulatory agencies' claims of safety have relied may be flawed. Unfortunately, the many studies completed since uses of these compounds were approved have not born out the validity of these assumptions. Although neonicotinoids are less acutely toxic than older insecticides to mammals and some other, vertebrates, they may be more toxic and targeted to nonpest invertebrates than older chemistries. Numerous studies demonstrate the negative impact of these insecticides on honey bees and native bees such as bumble bees (Goulson, 2013). Studies also show that neonicotinoids are detrimental to aquatic organisms (Mineau and Palmer, 2013).

"Pesticides are applied without scouting fields to see if they are needed, violating a bedrock principle of integrated pest management. The result is a biological diversity desert in many corn and soybean fields in the agricultural Midwest, and signs that the surviving insects are becoming resistant to several key bug-fighting tools now available to farmers." Diana Yates, University of Illinois, USA." Illinois, USA.

There are 7 different active ingredients: acetamiprid, clothianidin, dinotefuran, imidacloprid, nitenpyram, thiacloprid, and thiamethoxam. In 2006, these were being marketed in 530 products in 123 countries. The most common of these are imidacloprid, clothianidin, and thiamethoxam. Thiamethoxam breaks down into clothianidin. Imidacloprid was the first neonicotinoid to be introduced. It is used on more than 140 crops in more than 120 countries, and is one of the fastest growing insecticides in terms of sales. It is highly toxic to bees, as are most of the neonicotinoids, and is implicated in honeybee Colony Collapse Disorder, and in potential ecological collapse. Several of the neonicotinoids also pose human health concerns.

### History

Nicotine acts as an insecticide but is also toxic to mammals, with a lower lethal dose for rats than flies. This spurred a scientific search for compounds that retain the insecticidal properties of nicotine but have selectively less effect on mammals, but initial investigation of nicotine related compounds (nicotinoids) as insecticides was not successful. The precursor to nithiazine was first synthesized by a chemist at Purdue University (Kollmeyer, *et al*., 1999). Shell researchers found in screening that this precursor showed insecticide potential and refined it to develop nithiazine. Nithiazine was later found to be a postsynaptic acetylcholine receptor agonist, meaning it has the same mode of action as nicotine. Nithiazine does not act as an acetylcholinesterase inhibitor, (Schroeder and Flattum, 1984). In contrast to the organophosphate and carbamate insecticides. While nithiazine has the desired specificity (i.e. low mammalian toxicity), it is not photostable (it breaks down in sunlight), so it was not commercially viable. Neonicotinoids were developed after the lack of commercial success of nithiazine. The first commercial neonicotinoid, imidacloprid, was developed by Bayer (Kollmeyer, *et al*., 1999).

Most neonicotinoids are water-soluble and break down slowly in the environment, so they can be taken up by the plant and provide protection from insects as the plant grows. During the late 1990s this class of pesticides, primarily imidacloprid, became widely used. Beginning in the early 2000s, two other neonicotinoids, clothianidin and thiamethoxam were in use as well. Currently, virtually all corn that is planted in the Midwestern United States is treated with one of these two insecticides and various fungicides. In addition, most soybean seeds are also treated with a neonicotinoid insecticide, usually thiamethoxam.

## Classification

Neonicotinoids are classified in three group these are as follows

A) Nitroguanidine neonicotinoid insecticides Clothianidin Dinotefuran Imidaclothiz Thiamethoxam

B) Nitromethylene neonicotinoid insecticides Nitenpyram Nithiazine

C) Pyridylmethylamine neonicotinoid insecticides Acetamiprid Imidacloprid Nitenpyram Thiacloprid

## Mode of Action

Neonicotinoids, like nicotine, bind to nicotinic acetylcholine receptors of a cell and triggers a response by that cell. In mammals, nicotinic acetylcholine receptors are located in cells of both the central and peripheral nervous systems. In insects these receptors are limited to cells of the CNS. While low to moderate activation of these receptors causes nervous stimulation, high levels overstimulate and block the receptors (Yamamoto, 1999). This receptor blockage causes paralysis and death. Nicotinic acetylcholine receptors are normally activated by the neurotransmitter acetylcholine. Normally, acetylcholine is broken down by acetylcholinesterase to terminate signals from these receptors. However, acetylcholinesterase cannot break down neonicotinoids, and the binding is irreversible. Because most neonicotinoids bind much more strongly to insect neuron receptors than to mammal neuron receptors, these insecticides are selectively more toxic to insects than mammals. (Tomizawa, 2004).

## Present Status of Neonicotinoids

Neonicotinoids are registered in more than 120 countries. With a turnover of €1.5 billion, they represented 24% of the global market for insecticides in 2008. Neonicotinoids are even more important in the market for seed treatments. After the introduction of the first neonicotinoids in the 1990s, this market has grown from €155 million in 1990 to €957 million in 2008. Neonicotinoids made up 80% of all seed treatment sales in 2008. Jeschke, *et al*., 2011.

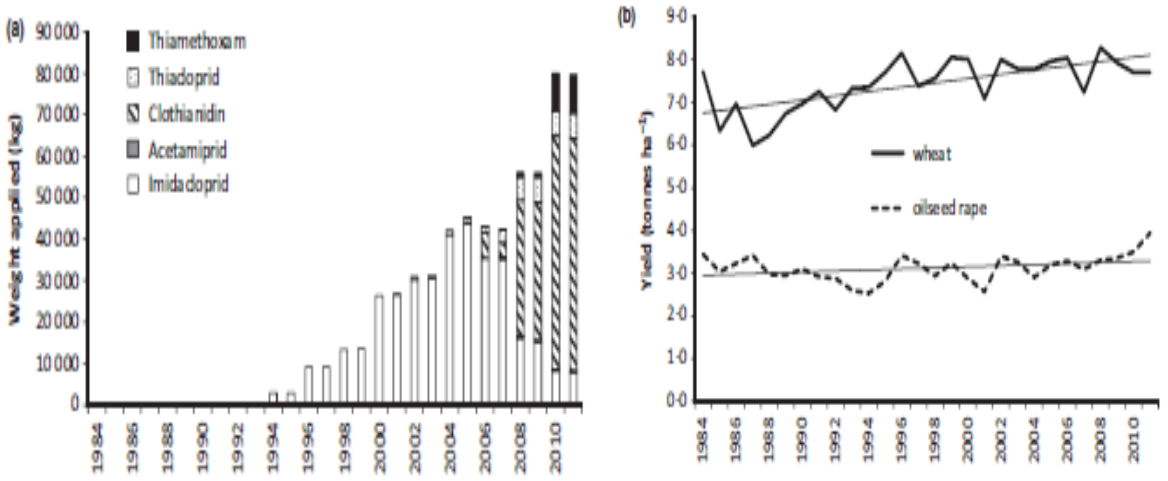


Fig.1. (a) Annual usage (kg) of neonicotinoids in agriculture and horticulture in the UK, one of few countries from which detailed records are available (Defra 2012a).
Note: Figures do not include garden or amenity use, or use for treatment of pets. In 2011, the area of land treated was approximately 13 million ha.

(b) UK yields of two crops that are now widely treated with neonicotinoids as a seed dressing (Defra 2012b). There has been no significant rise in oilseed rape yield since its introduction, while winter wheat yields have risen slightly (linear regressions, $F_{1,26} = 4.01$, ns and $F_{1,26} = 21.1$, $P < 0.001$, respectively).

## Factor Affecting Honey Bee Reduction

Various factor affecting the honey bee population. They are categorised into two groups are as follows. Biotic. The living organisms which causes disease in the bees and various living organisms feed externally or internally *i.e*. ecto or endoparasites and in Abiotic pesticides electromagnetic radiation

## Effect of Neonicotinoids on Honey Bees

The behavioral functions, induced mortality affected in the honey bees. Thiamethoxam by contact induced either a significant decrease of olfactory memory of honey bees. Limited effects on the motor and sensory functions of the honeybee (Aliouane, *et al*., 2009).

Bees contribute around 80% of insect pollination, so it is imperative we understand and mitigate the causes of current declines. Bees are under pressure from disease and habitat loss, but another threat has come to the fore recently. Since about 2006 there has been a world-wide dramatic rise in the number of hive losses and a reduction of wild bees. (Copping, 2007).

## Colony Collapse Disorder (CCD)

The concern of persistent loss of honey bee (*Apis mellifera* L.) colonies worldwide since 2006, a phenomenon referred to as colony collapse disorder (CCD), has led us to investigate the role of imidacloprid, one of the neonicotinoid insecticides, in the emergence of CCD. CCD is commonly characterized by the sudden disappearance of honey bees (specifically worker bees) from hives containing adequate food and various stages of brood in abandoned colonies that are not occupied by honey bees from other colonies. Honey bees were able to replicate CCD with sub-lethal doses of imidacloprid. The imidacloprid-treated hives were nearly empty, consistent with CCD, and the authors exclude Varroa or Nosema as contributing causes. (Lu *et al.*, 2012).

### Effect of Neonicotinoids on Natural Enemies

Indirectly exposed when they consume prey or plant materials that are contaminated with an insecticides. The below table shows the impact of neonicotinoids on the natural enemies.
The treatments with the insecticides shows the effect on predatory insect species living on cucumber and tomato palnt in autumn planttation. Population of *Orius* sp. was the most reduced compared *to Crysoperla caneae, Paederus alfierii* and *Coccinella undecempunctata* species by using imidacloprid followed by thiamethoxam and acetamiprid, AL-Kherb, 2011.
Imidacloprid spray treatment to pest eggs only slightly reduced emergence of *Trichogramma cacoeciae*, a parasitic wasp, but direct expose to spray caused high mortality of the adult parasitoid wasps (Saber, 2011).

### Effect of Neonicotinoids on Non Target Organisms

Organisms present in soil such as earthworms, ants and mound-building termites are considered to be "ecosystem engineers" for their ability to influence natural functions on a landscape level. Soil invertebrates enhance microbial activity, speed up decomposition and influence movement of water, nutrients, oxygen, $CO_2$, salt and pollutant within the soil.
Earthworms and other invertebrates that dwell in soil or leaf litter can be exposed to neonicotinoids applied as soil drenches.
Imidacloprid, clothianidin, thicloprid and acetamiprid are more toxic to earthworm than other modern synthetic insecticides, including carbamates, OP and pyrethroids (Wang *et al*., 2012).

### Health Effects on Birds, Aquatic Invertebrates, and Other Wildlife

In March 2013, the American Bird Conservancy published a review of 200 studies on neonicotinoids including industry research obtained through the US Freedom of Information Act, calling for a ban on neonicotinoid use as seed treatments because of their toxicity to birds, aquatic invertebrates, and other wildlife (Mineau and Palmer, 2013).

### Neonicotinoid pesticide banned in Germany May 21, 2008.

The German Federal Office of Consumer Protection and Food Safety (BVL) suspended registration of seed treatment products used in oilseed rape and sweetcorn.The suspended insecticides are Antarc (imidacloprid; Bayer), Chinook (imidacloprid; Bayer), Cruiser (thiamethoxam; Syngenta), Elado (clothianidin; Bayer), Faibel (imidacloprid; Bayer), Mesurol (methiocarb; Bayer) and Poncho (clothianidin; Bayer).

## CONCLUSION

Neonicotinoids are harmful to non-target organisms to varying degree. The adoption of prophylactic use of neonicotinoids as has led to the abandonment of the long established principles of IPM. Avoids neonicotinoids wherever possible and avoids use of pesticides that persist in the environment. Any indirect effects must be evaluated to determine if pesticides are compatible with natural enemies so as not to compromise long-term success of biological control programs.

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