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The risk of insecticides to pollinating insects

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A key new risk to our pollinators has been identified as exposure to neonicotinoid insecticides. These discoveries have refuelled the debate over whether or not the neonicotinoid insecticides should be banned and conflicting evidence is used in this battle. However, the issue is not black or white, but gray. It is not an issue of whether the neonicotinoids are toxic to insects or not. Clearly, all insecticides were designed and optimized for this attribute. The real question is, or at least should be, which insecticide is the safest for use for a particular need.

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Recent estimates indicate that insect pollinators contribute over $200 billion in pollination services for approximately two-thirds of crop species and most wild flowering plants. This critical food security and ecosystem service is under threat from a number of anthropogenic causes that are contributing to a loss of pollinator abundance and diversity. These involve habitat loss by the intensification of agriculture, leading to potential deficits in pollinator nutrition, a loss of nesting and overwinter sites and geographical isolation. A second major consequence of agricultural intensification is the requirement for increased (quantity or potency) pesticide use.

In addition to the threats experienced by other insect pollinators, the honeybee has suffered from the pressures of globalization, leading to the introduction of the *Varroa destructor* mite and the gut pathogen, *Nosema ceranae* into western honeybees from Asia. However, honeybees are supported by the efforts of beekeepers and actual losses are offset by their careful management and replacement of lost colonies, albeit at the expense of honey production.

The threat to pollinators by pesticides is hotly debated and the answer comes down to the actual field-relevant exposure to a particular compound and the existence of other threats, such as disease, poor nutrition or other pesticides. In the case of the neonicotinoids, exposure is largely via their use as systemic insecticides, where crop seed is pre-coated. Upon germination, the neonicotinoid is translocated throughout the plant, including the nectar and pollen consumed by pollinators. By definition, their use is entirely prophylactic, in the absence of any real or perceived risk from pests but in case they arrive. As commercial oilseed rape is routinely pretreated, it is hard to see how any evidence for a local need is acquired.

Although the actual level of exposure to the neonicotinoids is debated and may be influenced by accumulation in the soil, current estimates suggest that 1–5 ppb is the most likely exposure rate for imidacloprid, clothianidin and thiamethoxam in plant nectar and pollen.[1]

In addition to the influence of the level and duration of exposure,[2] the risk of any pesticide depends on its potency at its intended site of action, as well as any deleterious effects at unexpected/unknown site(s) of action. Thus, a switch to a more potent compound may reduce the amount of pesticide used, but would not alter its risk to the environment. Furthermore, its potency may vary between different species. For example, the neonicotinoids have low potency in humans, as they have a low affinity for human receptors. However,

they are effective in bees where bumblebees appear more sensitive than honeybees.[3] Their potency in other pollinators is unknown, but highly important. Pesticide risk is not limited to its acute toxicity and the level at which it becomes lethal to an individual insect. Chronic, sublethal toxicity may decrease pollinator performance either at the individual or colony level[4] and such risks have been highlighted recently.

Subtle effects of pesticides can have a major impact on insect health and fecundity. For example, the neonicotinoids have been shown to have a negative impact on bumblebee colonies at concentrations close to that found in the nectar and pollen of bee-friendly crops.[1] Recent studies have shown that bumblebee colony growth is decreased when exposed 0.7–6 ppb[5] or 10 ppb.[6] Crucially, in both studies deficits were not evident for at least two weeks, suggesting that toxicity may result from a deficit in bee foraging efficiency causing a limitation of brood production as reported previously, or by developmental defects in the brood. Indeed, poor foraging[6] and queen production[5] was identified in these studies. This evidence of chronic toxicity is in contrast to the current safety-testing regime that is limited to an LD_{50} assessment over a few days.

Therefore, in addition to potential acute and chronic toxicity on adult pollinators, these studies support a developmental consequence of neonicotinoid exposure. Finally, epigenetic changes by pesticide exposure could cause heritable changes in gene expression by mechanisms such as DNA methylation, histone modification or microRNA expression as reported for nicotine exposure in humans.[7,8]

With respect to neonicotinoid toxicity on honeybees, acute exposure to the major learning center of the brain (Kenyon cells in the mushroom body) to 2.5 ppb (imidacloprid or clothianidin) leads to a transient hyper-excitation followed by neuronal inactivation.[9] It is likely that even lower levels would produce brain deficits if present chronically over long periods. The rapid loss of function observed following acute exposure[9] is likely to lead to deficits in honeybee learning and memory and this was confirmed using the proboscis reflex response in conditioned learning experiments on honeybees fed imidacloprid at 2.5 ppb.[10] The importance of these studies is that they identify sublethal effects at field-realistic exposure levels. The consequences of these deficits to whole colonies are yet to be established in controlled experiments where the influences of other major threats can be excluded. However, deficits in honeybee homing have been observed when exposed to 27 ppb.[11] Prolonged exposure to neonicotinoids may lead to cytotoxic effects on honeybee brains,[12,13] malpighian tubules[14] or midgut.[13]

Although these studies do not address the contribution of the neonicotinoids to the observed honeybee losses or pollinator decline generally, they do suggest a major risk to bee health and the importance of further research to establish the risk from neonicotinoids (and other pesticides). Identified risks cannot be extended automatically to all compounds in a class, as risks depend on dose, potency, duration of exposure and longevity within the bee and the environment. Therefore, the risks posed by other neonicotinoids (thiacloprid, acetamiprid and dinotefuran) will need independent testing.

The major issue currently is whether imidacloprid, clothianidin and thiamethoxam should have a moratorium placed on them. Scientifically, it is not yet possible to provide a definitive answer. Certainly, if these neonicotinoids can be removed from use and not replaced, then a precautionary principle is a wise strategy to engage. On April 29, 2013, the EU member states voted on the question of placing a two-year moratorium on the use of imidacloprid, clothianidin and thiamethoxam for seed or folia treatment on bee-attractive plants and cereals. Although a majority of EU states supported this proposal, the qualified majority necessary was not reached. Therefore, the European Commission exercised their right to decide to push forward the moratorium, which will apply from December 1, 2013, until new information on the safety of these products is available (http://europa.eu/rapid/press-release_IP-13-379_en.htm).

In support of this decision, the banning of neonicotinoids in France has not resulted in a decreased crop yield for corn or sunflowers.[15,16] Perhaps the imagined risk from skulking pests, and the need for prophylactic treatment, no longer exists? Alternatively, perhaps the neonicotinoids remain in the soil for substantial periods and continue to suppress the pests. Indeed, some reports on a lack of increase of yield when neonicotinoids are applied[15,16] suggest that the threats may not exist.

It is not clear whether a two-year ban will be sufficient for the intended purpose of gaining more information. First, this is insufficient time for new grant proposals to be generated, reviewed, funded, new data produced and peer reviewed before publishing. Moreover, is a new funding scheme to be announced that will provide for such an urgent push on research? In terms of there being a reversal in the losses of honeybees (or more importantly, pollinators generally), is two years sufficient to clear the soil of residual neonicotinoids? If not, the issue of their need and effectiveness will remain controversial. Moreover, longevity in the soil is not the only barrier to pollinator recovery as the use of neonicotinoids on crops not attractive to bees will continue in Europe. For this strategy to be valid, a careful control of crop rotation will be required to ensure that bee friendly crops do not encounter previously applied neonicotinoids. Similarly, wildflower field margins may also translocate soil neonicotinoids into their nectar and pollen and deliver these to pollinators.

Where evidence does exist to indicate that the neonicotinoids must be replaced by other insecticides due to a consequential inability to maintain food security, then the question is an empirical one of whether the replacement is more, or less, toxic than the neonicotionoid being withdrawn. When we remember that all pesticides have passed the same safety tests and that only after further investigations into the sublethal effects of the neonicotinoids have new risks been identified. Therefore, when these same standards are applied to all pesticides, we must expect the failure of other pesticides. Therefore, no replacements should be employed until they have passed the same strict criteria by which the neonicotinoids have been called into question. Otherwise, all the political and scientific efforts have been in vain. Everyone will agree that we do not wish to replace the neonicotinoids with even more toxic insecticides. By the empirical testing of

specific alternatives, side by side, at the pharmacological, physiological, individual bee and colony level, it will be possible to step forward, one step at a time, toward progressively less toxic methods of controlling insect pests. Otherwise, we may replace the neonicotinoids with more toxic compounds, which would be a step backward. Field studies on the risks of pesticides are prohibitively difficult due to the complexities of the environment, including multiple threats to our pollinators. Such experiments cannot be controlled, all threats cannot be monitored and all levels of toxicity cannot be investigated.

Clearly, the long-term future is the use of integrated pest management, using good agricultural practices such as crop rotation and native predators. The use of pesticides or biological alternatives seems unavoidable, to ensure our food security, as long as pest insects exist and we depend on large monocultures, with little natural insect forage. Therefore, a risk/benefit balance will need to be sought according to environmental damage caused and our need for the crop resource/economic gain.

In the case of the recreational use of pesticides for gardens and for amenity uses (parks, golf courses and road verges) such risks are harder to justify. A limitation of the risks to pollinators from pesticide use and of compromised food security by lost pollinators or a failure to control pest species, can be achieved by a restriction of all pesticides to uses that are essential to our needs. Moreover, the remaining landscape could then be managed to help mitigate against the risks by the provision of improved forage and nesting/overwinter sites.

Disclosure of Potential Conflicts of Interest

No potential conflicts of interest were disclosed.

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