Pesticide Action Network UK

Serious shortcomings in assessing risks to pollinators

Bee Declines & Pesticides factsheet 3

This factsheet summarises the main weaknesses in the current risk assessment process for neonicotinoid and other systemic pesticides as carried out in the EU and US. It discusses why the tests required before approving pesticides are inadequate to understand the special risks that systemic pesticides pose to bees and other pollinators. It also highlights the need to consider interactions between different pesticides and with bee diseases and parasites.

Credit: Graham White

An outdated approach to risk assessment

The environmental risk assessment process in use today for evaluating the risks of pesticides to bees (and other non-target organisms) was designed in an era before the widespread use of systemic pesticides. The ecotoxicity tests which pesticide manufacturers must submit before their pesticide is approved and the decision-making procedures by governmental risk assessors were developed for older generation insecticides that were mainly applied by spraying crop foliage. These insecticides tended to degrade in the environment within a few days so the main exposure scenarios assessed for risks to honeybees were acute, contact toxicity during spraying or from contact with recently treated foliage[1].

This approach has come under fire

from various quarters[2,3,4,5,6] for failing to address the specific properties of systemic insecticides, including the neonicotinoids, which hugely increase their risks to pollinators:

- translocation to all parts of the crop plant, including nectar, pollen, guttation drops
- oral exposure via foraging on floral resources containing residues of these compounds
- high persistence in soil, water, plus potential to contaminate untreated crops and wildflowers
- toxic at much lower doses than many older insecticides

Furthermore, risk assessment in Europe and the US continues to rely almost exclusively on short-term acute toxicity testing of adult bees, using the traditional LD50 protocols, to calculate the dose at which 50% of the individual insects are killed, either by contact or by a single dose in food. These classical tests only take into account 'dead or alive' as the toxicological 'endpoint' [7]. Acute toxicity tests on adults are ill-suited for testing systemic insecticides because they don't consider the different routes by which bees may be exposed in reality, particularly through their diet.

The low levels of neonicotinoid residues that occur in treated plants are more likely to affect honeybees by combined acute, chronic and sub-lethal poisoning rather than acute effects alone. Therefore we need a radically different approach for assessing the risks of systemic pesticides, which gives proper consideration to sub-lethal effects and to persistence in soil; residues in pollen and nectar and potential transport by air and water[8,9].

Shortcomings of current tests

Researchers, beekeepers and environmental NGOs have drawn attention to numerous shortcomings in different aspects of current risk assessment testing and decision making. At the initial stage of testing in lab studies (known as Tier 1), current EU guidelines recommend acute toxicity testing on individual adult honeybees and bee larvae ('brood'). Acute toxicity tests to determine the LD50 are the main toxicological consideration here. Other tests aim to identify 'no adverse effect' dose levels but test insects are only exposed over a short time frame, usually 48-96 hours, giving a very incomplete measure of potential harm because only short-term survival of adults is considered[10,11]. They cannot account for chronic toxicity and sub-lethal effects that are now known to be crucial elements of neonicotinoid toxic effects in honeybees and of other systemic compounds like fipronil[12]. Nor does the US EPA require any data on sub-lethal effects for pesticide registration[13].

Credit: Graham White

Moving from lab tests to more realistic field experiments

If no clear conclusions on harmlessness emerge at Tier 1, then Tier 2 semi-field testing (confining small numbers of insects in cages or tunnels) needs to be done to understand what risks might more realistically occur outside lab conditions. Under the EU guidelines (EPPO guidance document 170) the Hazard Quotient is used (= the field application rate of the pesticide/oral or contact LD50) as a decision trigger, requiring further studies if the HQ exceeds a certain threshold value. This threshold value is derived from data only considering spray applications on honey bees and this has been criticised as unsuitable for assessing systemic compounds[14]. Unfortunately, the guidance documents for the long-standing EU pesticide authorisation legislation (directive 91/414) do not provide detailed technical guidance on how to proceed in bee risk assessment for substances with systemic properties and this has yet to be remedied under the new EU authorisation (Regulation 1107/2009, see factsheet 4).

A range of semi-field tests can be done for Tier 2 but the problem is that no specific protocols are recommended[15]. Without standardised protocols, each lab uses different approaches, producing results that are difficult to compare[16]. Another issue is that field studies used in risk assessment must demonstrate that bees have been adequately exposed to contaminated resources[17,18].

We do not yet know how strongly many adverse responses measured in the lab (e.g. on learning or memory) actually affect the health of colonies in the field. This unresolved issue is critical for establishing environmental relevance of lab findings. There are disagreements about the minimum size of bee colonies used in cage and tunnel tests[19,20]. In a small caged colony it is not possible to reproduce all the typical behaviour needed by the complexity of a normal, large hive - leading to results which might not reflect real world impacts on reproduction.

Understanding effects at colony level and on other pollinators

To address sub-lethal and chronic lethal effects of pesticides on honeybees, new official guidelines using standardised protocols are urgently needed. These need to reflect the exposure patterns now understood – smaller doses, over a long period of time, through a variety of routes. Chronic feeding tests using whole colonies may provide a better way to quantify the effects of systemics[21]. Currently, it is very difficult to predict impacts in the field of trace level doses in bee diet[22] and much more monitoring of residue levels in different food sources and other exposure scenarios is urged, to feed into the risk assessment process[23,24]. Yet data on pesticide residue presence and levels in various materials used by honeybees are not statutorily requested of pesticide manufacturers[25].

Another important flaw in the wider risk assessment of systemic insecticides is that the honey bee has often served

as a representative test species for all pollinators in the regulatory process, although the toxicity of pesticides to other bees species and different pollinator groups may be very different[26,27]. Many solitary bee species have much smaller foraging ranges than honeybees (a few hundred metres, rather than several km) so there is a proportionately greater risk of dietary contamination for solitary species living close to treated crops.

Flawed studies lead to disputed conclusions

Given the uncertainties in the science, the widely ranging toxicity values obtained in different lab studies and the difficulties in getting unambiguous results from semi-field, let alone field, tests (see factsheet 2), it is hardly surprising that the risk assessment tests chosen and their interpretation are very contentious. Risk conclusions drawn by official assessors from acute toxicity test results have been questioned by other stakeholders. In 2009 PAN Europe and other NGOs challenged the European Commission's approval of imidacloprid, disputing the risk assessment assumptions, process and conclusions on numerous points. Box 1 lists some of these criticisms.

What required tests fail to address at all

Tests on different bee classes (winter adult workers, larvae, nurse bees, drones, queens) at different stages in the hive development and seasonal rhythms are not routinely done when assessing pesticide risks. Research shows that chronic toxicity effects can be very different between young and old bees and they can differ in their sensitivity[28]. Failing to test these differences constitutes a blind spot in the subsequent decision making.

Another is that risk assessment does not consider the effects of contaminated pollen consumption during winter. There

Box 1. Criticisms of the EU imidacloprid risk assessment

Incorrect assumptions on bee feeding rates: The official EC figure for No Observed Effect Level for imidacloprid (46 parts per billion) is based on assuming consumption of 20 microlitres (µl) per bee, the syrup amount given in normal LD50 tests. But in reality a bee can consume much more, with published studies estimating forager bees can consume between 228-989 milligrams over a 7-day period. PAN Europe estimates that the No Observed Effect Level in real conditions should be at least 10 times lower than the EC figure- less than 5 parts per billion.

No consideration of toxic breakdown products: The EC risk assessment looked at only 2 metabolites but ignored another 2 metabolites (olefin and 5-hydroxy- imidacloprid) known to be hazardous for bees and which are detected in pollen and nectar.

Actual exposure levels via food not verified: None of the field and tunnel tests submitted in the official imidacloprid risk assessment report demonstrated that treated pollen had actually been consumed by bees during the tests. Therefore concluding 'absence of effect' from negative results is scientifically unacceptable because the real exposure/consumption levels have not been proven.

are fewer bees in the colony over winter but they live much longer than summer bees and they will feed the brood over a long period, using stores of pollen and honey collected during the previous spring/summer. Without a full year of hive monitoring it is difficult to know the full effects of neonicotinoids on colony health. Individual mortality, foraging activity and colony health should be monitored for 12 months in honeybees, for a full growing season for bumblebees and for the larval development cycle for solitary bees, to observe any delayed effects from feeding on contaminated food stores[29].

It is extremely worrying that risk assessment does not currently insist on tests on bee behaviour effects even though disruption of normal behaviour, such as reduced foraging, can lead to decreased pollination, lower reproduction and finally in colony mortality due to lack of food[30]. Testing must be expanded to include foraging behaviour bioassays and confined and field tests which continue long enough to evaluate side effects on over-wintering colonies and at second year spring[31].

Nor is there any assessment of exposure through tiny particles of treated seed coat and contaminated dust released during sowing- situations known to have caused mass poisoning incidents in several countries[32]. There is a similar blind spot in pollinator exposure (and therefore likely risk levels to be considered) in surface water, wild flowers or in guttation drops on treated crops[33,34].

Current testing regimes continue a narrow focus on individual adult workers. This approach is totally inadequate to make a genuine evaluation of the impacts on complex social insects which depend on the colony as a whole for health, reproduction and survival. A more appropriate approach has been proposed for the 'super-organism' that is the bee colony and should be the unit to be protected, rather than individual bees, since colony survival is based on complex interactions between different groups of honeybee[35]. Risk assessment frameworks that aim to protect ecosystems, rather than species, are already used in other aspects of environmental protection. For bee-toxic pesticides, a colony-level protection approach would use data for different categories of bees in relation to amounts of contaminated pollen and nectar each bee class consumes, including from food stores.

Pesticide cocktail effects and links to disease susceptibility ignored

Evidence has emerged that the effects of low-level exposures to neonicotinoids can be worsened when bees are also exposed to certain other pesticides. Some pesticides can increase the toxicity of others, making the combined effect far more toxic than just the effect of each individual pesticide added together. These kind of interactions are known as 'synergy'. Studies show that honeybee colonies are routinely exposed to a huge range of pesticides in their environment[36,37]. Not surprisingly, residues of the miticides deliberately introduced into hives by beekeepers to control varroa mite parasites are often present in hive food and materials, but also other insecticides and fungicides sprayed onto crops.

Some of the commonly used fungicides in field crops, such as propiconazole, are now known to synergise the toxicity of some neonicotinoids to honeybees in lab studies, sometimes increasing the neonicotinoid toxicity by over a thousand-fold[38].

Pyrethroid insecticides, another group which is toxic to bees, are also widely used and can synergise with fungicides. The finding of commonly used fungicides turning up in pollen[39] raises the question of the effect on individual insects and hive health of regular joint exposure to these mixtures (the 'cocktail effect'). In reality pollinators may be exposed to dozens of different pesticides on a daily basis, yet in risk assessment the effects of pesticides on non-target organisms are considered in isolation, one pesticide at a time. Not only does the hive accumulate pesticide residues from the workers' wide foraging range but field applications commonly use product mixes, for example, fungicides often sprayed in combination with insecticides. The risk assessment process is blind to possible cocktail effects but these could help explain why hives are suffering poor health at trace levels of neonicotinoid exposure. More research is called for on mixture toxicity and the accumulation of neonicotinoid residues in the environment as use increases[40].

Interactions between exposure to neonicotinoids and the fungal bee disease *Nosema*, which has become more common and more virulent in recent years, is another example of synergism. Three recent studies have demonstrated that hives exposed to these compounds, even at incredibly low doses, are significantly more susceptible to infection by the fungus[41,42,43]. The Vidau et al. study found that exposure to sub-lethal doses of fipronil and thiacloprid highly increased mortality of honey bees already infected by *Nosema* disease. The Pettis et al. study proved that fungus + neonicotinoid interactions are possible in the real world, not just in a lab setting. Bees and other social insects display complex hygiene behaviour and keeping the colony protected from disease-causing microbes and parasites forms part of their group 'immune response'. As with foraging behaviour, sub-lethal doses of neonicotinoids and other pesticides could be harming pollinator immune functions and resistance to diseases in ways we do not understand and which could compromise a colony's ability to survive.

"Interactions between pesticides and pathogens could be a major contributor to increased mortality of honey bee colonies, including colony collapse disorder, and other pollinator declines worldwide"

Jeff Pettis, USDA Bee Research Lab & University of Maryland colleagues, 2012

However, neither the EU nor the US pesticide regulators require any studies about potential synergies between neonicotinoids and disease-causing pathogens that can undermine bee health[44], nor even a consideration of this issue. This failure constitutes another yawning chasm in the risk assessment, especially given the significance of new scientific understanding.

Improving the risk assessment process

A growing community of researchers and beekeepers has been criticising current risk assessment for some years and calling for change (see factsheet 8). The European Beekeeping Coordination (www.beelife.eu) provides an accessible and comprehensive critique, along with short and longer term

recommendations for improving the risk assessment tests and decision making framework[45]. These include testing and taking fully into account:

- possible synergistic interactions between pesticides (not just insecticides but also frequently used fungicides)
- possible interactions between insect disease pathogens and pesticides
- sub-lethal and chronic effects from long-term exposure
- effects on the immune capacity system of bees
- chronic lethal toxicity, as well as acute toxicity

Many of the inadequacies of the current procedures in relation to neonicotinoid and other systemic pesticides have finally been recognised by regulators and risk assessment professionals but progress in updating or improving the evaluation procedures is slow and limited (see factsheet 4).

Key points

- Current tests required of pesticide manufacturers and the way governments assess toxicity data are totally inadequate to assess the risks that systemic insecticides pose to pollinators.
- Tests focus on short term, acute toxicity to adult worker bees and mainly ignore chronic toxicity and sub-lethal effects on bee behaviour, on larvae and on hive overwintering.
- Pollinator exposure via contaminated nectar and pollen or other routes now known to pose risks, such as seed drilling dust, are not properly considered in the risk assessment process.
- Interactions between neonicotinoids and other pesticides, notably commonly sprayed fungicides, can increase the toxicity by several hundredfold or more, but this 'cocktail effect' is not assessed.
- Hives exposed to neonicotinoids are much more susceptible to infection by major bee diseases and possibly to parasites, but these types of interaction are also ignored.

In this series

If you would like to find out more about the relationship between pesticides and pollinator declines, all of these leaflets and other info are available via PAN UK's bee webpages at: **http://bees.pan-uk.org**

Bee Declines and the Link with Pesticides. Summary leaflet.

Fact sheets:

1. Different routes of pesticide exposure
2. Sub-lethal and chronic effects of neonicotinoids on bees and other pollinators
3. **Serious shortcomings in assessing risks to pollinators**
4. Different regulatory positions on neonicotinoids across Europe
5. Can restrictions on systemic insecticides help restore bee health?
6. What could farmers do to rely less on neonicotinoids?
7. Opportunities for improving and expanding pollinator habitats
8. Action on neonicotinoid and other bee-toxic pesticides

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PAN UK's vital work in the UK and in developing countries

Pesticide Action Network UK is a registered charity dedicated to:-

- Eliminating the most hazardous pesticides,
- Reducing dependence on chemical pesticides,
- Promoting sustainable and equitable food systems and increasing the use of alternatives to chemical pest control in agriculture, urban areas, public health and homes and gardens

In the UK, we campaign for tighter regulatory controls on pesticides and encourage retailers to tackle pesticide problems in their supply chains. We provide advice on alternative ways to control pests and work with local communities to reduce public exposure to pesticides. In the developing world, we raise awareness about pesticide hazards and train farmers in organic and low input agricultural techniques to help them to make a decent living without putting their own health, their families or their environment at risk.

Populations of bees and other insect pollinators have fallen dramatically in recent years. The reasons for these declines are complex and wide ranging, but there is little doubt that pesticides are playing a key part. PAN UK has prepared these fact sheets to cut through the confusion and provide an up-to date and balanced explanation of the role of pesticides in pollinator declines. To find out more and what you can do, please visit http:\\bees.pan-uk.org

Published by Pesticide Action Network UK. September 2012

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