Department for Environment, Food and Rural Affairs

An assessment of key evidence about Neonicotinoids and bees

March 2013

Contents

Executive summary

Three recent studies in which bees were dosed with neonicotinoids showed sub-lethal effects on bees [1-3]. The results from these studies contrast with a growing body of evidence from field studies that has failed to show an effect of neonicotinoids when bees are allowed to forage naturally in the presence of crops treated with neonicotinoids [4-8]. The evidence suggests the reason for this difference is over-dosing of bees in the dosing studies; in all cases there is evidence that the doses of neonicotinoids presented to bees under laboratory or semi-field conditions were unrealistically high. The dosing studies therefore represented the extreme case in a field situation. In the only study in which dose was measured [1] the dose was much greater than would have ever been experienced in a field situation.

A concentration of 1-5 µg/l of neonicotinoid in nectar appears to be the threshold below which an effect tends not to be observed [9] and most residue measurements in the nectar and pollen of treated crops are normally at or below this level [4,6,7,10,11]. In addition, examination of bee foraging shows that they tend not to feed exclusively on treated crops [7], thus diluting any effects of neonicotinoids. Consequently, the evidence of effects of neonicotinoids on bees come from studies [1-3] in which doses were likely to have been at least 2-10 times above this threshold.

There is a possibilitythat field studies did not have the statistical power to show effects but the accumulated evidence across several independent studies suggests that this is unlikely and, any effects that are present are likely to be small and not biologically significant. Moreover, oilseed rape (OSR) requires insect pollinators to support its productivity [12-18]. The fact that OSR treated with neonicotinoids has been a productive crop for over a decade in the UK is itself evidence that pollinator populations, including bees, are not being reduced by the presence of neonicotinoids.

Conclusion: While this assessment cannot exclude rare effects of neonicotinoids on bees in the field, it suggests that effects on bees do not occur under normal circumstances. This assessment also suggests that laboratory based studies demonstrating sub-lethal effects on bees from neonicotinoids did not replicate realistic conditions, but extreme scenarios. Consequently, it supports the view that the risk to bee populations from neonicotinoids, as they are currently used, is low.

1. Background

1.1. Neonicotinoids are a class of chemical derived from naturally-occurring plant compounds – nicotines - with insecticidal properties. Five neonicotinoid compounds are approved for use as pesticides in the UK and EU. Of these, clothianidin, imidacloprid and thiamethoxam are of similar acute toxicity to bees. Acetamiprid and thiacloprid are far less acutely toxic and are structurally different (being cyano-

substituted neonicotinoids whilst the other three compounds are nitroguanidine-substituted). Concerns about the effects of neonicotinoids on bees have therefore focussed on clothianidin, imidacloprid and thiamethoxam.

1.2. The focus of this assessment is on the use of these three neonicotinoids as seed treatments in plants that are attractive to pollinators, such as oilseed rape (OSR), maize and sunflowers, although the focus here is on OSR. In the field, thiamethoxam is metabolised to clothianidin [19].

1.3. Imidacloprid was the first neonicotinoid to be used in the UK, being introduced in the mid-1990s. Since then, the use of neonicotinoids has steadily increased. In the UK, most oilseed rape and sugar beet crops and a substantial proportion of cereals crops are now grown from seed treated with clothianidin, imidacloprid or thiamethoxam. A number of horticultural crops are also treated with neonicotinoids. In the UK imidacloprid has almost ceased to be used in seed treatments on OSR [7] which has a suspected higher levels of persistence in the environment [20,21].

2. The evidence

Recent studies of greatest relevance are:

2.1. **Henry et al.** [1] dosed honey bees with a single dose of 67 µg/l of thiamethoxam in 20 µl of sucrose solution. This is the equivalent of a full daily dose in a single bolus of 20 µl. They tracked the behaviour of honey bees and found that this dose of thiamethoxam caused a degree of homing failure possibly because of disorientation.

2.2. **Whitehorn et al.** [2] dosed bumble bees with sugar solution containing 0.7 or 1.4 µg/kg and pollen containing 6 or 12 µg/kg of imidacloprid for 2-4 weeks. The bees were then left to forage freely in the field. The end point of the experiment was the growth in mass of the bee colonies. There was a dose-dependent response in growth of the colonies with those colonies receiving no dose growing fastest and those with the highest dose growing slowest. There was also a reduction in the number of new queens produced by the dosed colonies.

2.3. **Gill et al.** [3] dosed bumble bees with sugar solution containing 10 µg/l of imidacloprid. They observed impairment of foraging that resulted in a reduction of colony productivity shown by a reduced number of worker bees within the colonies.

2.4. **Thompson et al.** [7] (also see Appendix I) placed bumble bee colonies within landscapes known to contain oilseed rape (OSR) treated with neonicotinoids. They measured colony growth rate, production of queens, neonicotinoid residues in nectar and pollen, and the kind of pollen being collected by the bees. They found no relationship between colony growth and neonicotinoid residues within pollen or nectar in the colonies. The bumble bee colonies grew to up to twice the mass of the untreated groups in the **Whitehorn et al.** [2] experiment. Even though the bees were located next to fields of OSR, at all sites the proportion of pollen from OSR

was <26% of the pollen returned by bees showing that bumble bees foraged on many different food sources.

3. Interpretation

3.1. The **Henry et al.** [1], **Whitehorn et al.** [2] and **Gill et al.** [3] studies all used artificial dosing of bees with sucrose solutions. Only in **Henry et al.** [1] was the dose rate controlled. In this case, bees were given an estimated full daily dose in a single bolus of 20 µl. This is unrealistic for two reasons:

3.1.1. Bees would normally metabolise the pesticide as they receive it while foraging across the whole day [9] and so would not be exposed to such a high single dose.

3.1.2. The study assumes that bees only feed on food sources that have been treated with pesticide. An assumption which evidence from **Thompson et al** [7] would suggest is incorrect.

Consequently, the design of the dosing regimen used by **Henry et al.** [1] gives a very high probability of showing deleterious effects of the pesticide. It is most likely to represent an unusually extreme case in a field situation, not the average or normal case.

3.2. Neither the **Whitehorn et al.** [2] nor the **Gill et al.** [3] studies measured the dose received by the bees so it is not possible to assess whether the dose was realistic. However, the following rational suggests that in both studies bees were over-dosed with neonicotinoids. The study designs were biased towards showing a deleterious effect of neonicotinoids because:

3.2.1. Both the **Whitehorn et al.** [2] and **Gill et al.** [3] studies relied upon provision of sugar solution with neonicotinoid concentrations similar to those measured in the nectar of OSR. However, there is a substantial literature showing that both honey bees and bumble bees adjust their foraging rate and their metabolism depending upon the quality and quantity of their food available. When fed syrup in experiments in the lab, bees increase their metabolic rate and their food intake [22-28]. This will have the effect of increasing the dose rate meaning that bees in these experiments probably ate more of the pesticide than they would have in the wild.

3.2.2. Moreover, **Gill et al.** [3] used a concentration of neonicotinoid in sugar solution that was about x6 that found in nectar of treated crops, including OSR [1,4], so the dosing in this study would have been at the high end of the potential spectrum. Even if, as claimed by **Gill et al.** [3], this was likely to be only 50% of the consumption by these bees then the dose was still high. **Gill et al.** [3] admitted that their treatments were “at the high end” of what might be found in the field. In the early stage of the experiment, this would constitute a dose rate of about 10 ng/day/worker bee in the early growth phase of the study. This constitutes about x7 what **Henry et al.** [1] considered to be a reasonable daily dose for honey bees and about x10 what

another lab-based study [9] considered to be the threshold of effect. Finally, **Gill et al.** [3] also used 7 literature sources as justification for their dosing regimen only 3 of which were original sources and in all cases these contained potential sampling bias that would, if used uncritically, tend to magnify the level of exposure of the bees. Consequently, it appears that **Gill et al.** [3] used unrealistically high doses and failed to simulate field conditions.

3.2.3. To justify their dosing levels **Whitehorn et al.** [2] referred to two studies [11,29] only one of which quoted any quantitative evidence of the concentration in plants and this referred to crushed sunflowers or sunflower pollen and not OSR. Even then the "low" concentration fed in pollen was x2 the mean concentration measured in the reference study they used to guide the concentration used in the experiment. Consequently, it appears that **Whitehorn et al.** [2] did not know what dose the bees were receiving and the likelihood is that they received a much greater dose than would have been experienced in the field.

3.3. The general conclusion is that all of these studies have presented bees with doses that represent the extreme upper levels to which bees are likely to be exposed. They are, therefore, an examination of an extreme case or they may not be realistic in any circumstances.

3.4. In contrast, while the **Thompson et al.** [7] study had design limitations because of the field conditions under which the study was conducted, it is representative of an increasing number of field-realistic studies [4-8] that have failed to find an effect of neonicotinoids on bees. Why is this?

4. Reasons for the difference between field-realistic and dosing studies

4.1. There are three main lines of evidence to explain the difference:

4.1.1. As explained above, dosing experiments appear not to produce realistic doses as experienced by bees in the field; all of the dosing to date is, at best, close to the upper end of the range that bees are likely to experience and, at worst, it is well beyond that level.

4.1.2. As demonstrated by **Thompson et al.** [7] some bees have very catholic tastes and are unlikely to feed only on crops treated with neonicotinoids. In the case of the **Thompson et al.** [7] study, the crop pollen was a minor part of the food taken by bumble bees even though it was the dominant flowering species in the landscape. This may be why bees and other wild pollinators perform best when there are diverse local flower communities upon which to feed and there is evidence that appropriately managed agro-systems can benefit bee pollinators over natural or semi-natural systems [30-37]. The overall effect of this will be to reduce exposure of pollinators like bees to plants treated with pesticides.

4.1.3. The kind of behavioural changes observed in toxicity dosing studies [1-3] tend only to occur at doses above 1-5 ng/day which was equivalent to feeding at a syrup concentration of 1-5 µg/l [9]. The level of residues found within nectar and pollen in bumble bee colonies by **Thompson et al.** [7], which were similar to those found in free-ranging honey bees given similar exposures [6], were below or at the lower end of this threshold and this is consistent with other evidence of residues measured in relevant materials [4,6,10,11,38] although residue concentrations can be highly variable [39]. This evidence suggests that populations of bees in free-ranging situations do not normally experience the levels of neonicotinoids that result in sub-lethal toxic effects. This, together with the dilution effect of bees not always feeding upon treated crops, is the most likely why field studies do not demonstrate the same effects as studies where bees are given artificial doses of pesticide. An alternative possibility is that field studies [4-8] do not have the statistical power to detect these effects. However, the accumulated evidence across several independent studies suggests that this is unlikely and that, even if effects are present and remain undetected, then these effects are small and unlikely to be biologically significant.

4.2. Insects are significant pollinators of crops like oilseed rape where yields can collapse in the absence of pollinators [12-18]. In the UK, neonicotinoids have been used as seed treatments on OSR for 10 years. This suggests that if pesticide use was reducing pollinator effectiveness then this would also be detrimental to crop productivity. Consequently, the claim that treatment of OSR with neonicotinoids kills pollinators is partly countered by the success of the crops themselves.

References

1. Henry M, Beguin M, Requier F, Rollin O, Odoux JF, et al. (2012) A Common Pesticide Decreases Foraging Success and Survival in Honey Bees. Science 336: 348-350.
2. Whitehorn PR, O'Connor S, Wackers FL, Goulson D (2012) Neonicotinoid Pesticide Reduces Bumble Bee Colony Growth and Queen Production. Science 336: 351-352.
3. Gill RJ, Ramos-Rodriguez O, Raine NE (2012) Combined pesticide exposure severely affects individual- and colony-level traits in bees. Nature 491: 105-U119.
4. Blacquiere T, Smagghe G, van Gestel CAM, Mommaerts V (2012) Neonicotinoids in bees: a review on concentrations, side-effects and risk assessment. Ecotoxicology 21: 973-992.
5. Chauzat MP, Carpentier P, Martel AC, Bougeard S, Cougoule N, et al. (2009) Influence of Pesticide Residues on Honey Bee (Hymenoptera: Apidae) Colony Health in France. Environmental Entomology 38: 514-523.
6. Hecht-Rost S (2009) Thiamethoxam (CGA293343): A field study with A9807C treated winter oilseed rape seed, investigating effects on honeybees (*Apis mellifera* L.) over four years in Alsace (France). Germany: Eurofins-GAB GmBH.
7. Thompson H, Harrington P, Wilkins S, Pietravalle S, Sweet D, et al. (2013) Effects of neonicotinoid seed treatments on bumble bee colonies under field conditions. In preparation.

8. Genersch E, von der Ohe W, Kaatz H, Schroeder A, Otten C, et al. (2010) The German bee monitoring project: a long term study to understand periodically high winter losses of honey bee colonies. Apidologie 41: 332-352.
9. Cresswell JE, Page CJ, Uygun MB, Holmbergh M, Li YR, et al. (2012) Differential sensitivity of honey bees and bumble bees to a dietary insecticide (imidacloprid). Zoology 115: 365-371.
10. Bonmatin JM, Marchand PA, Charvet R, Moineau I, Bengsch ER, et al. (2005) Quantification of imidacloprid uptake in maize crops. Journal of Agricultural and Food Chemistry 53: 5336-5341.
11. Bonmatin JM, Moineau I, Charvet R, Fleche C, Colin ME, et al. (2003) A LC/APCI-MS/MS method for analysis of imidacloprid in soils, in plants, and in pollens. Analytical Chemistry 75: 2027-2033.
12. Bommarco R, Marini L, Vaissiere BE (2012) Insect pollination enhances seed yield, quality, and market value in oilseed rape. Oecologia 169: 1025-1032.
13. Jauker F, Bondarenko B, Becker HC, Steffan-Dewenter I (2012) Pollination efficiency of wild bees and hoverflies provided to oilseed rape. Agricultural and Forest Entomology 14: 81-87.
14. Viik E, Mand M, Karise R, Laaniste P, Williams IH, et al. (2012) The impact of foliar fertilization on the number of bees (Apoidea) on spring oilseed rape. Zemdirbyste-Agriculture 99: 41-46.
15. Jauker F, Wolters V (2008) Hover flies are efficient pollinators of oilseed rape. Oecologia 156: 819-823.
16. Abrol DP (2007) Honeybees and Rapeseed: A Pollinator-Plant Interaction. In: Gupta SK, editor. Advances in Botanical Research: Incorporating Advances in Plant Pathology, Vol 45. London: Academic Press Ltd-Elsevier Science Ltd. pp. 337-367.
17. Steffan-Dewenter I (2003) Seed set of male-sterile and male-fertile oilseed rape (Brassica napus) in relation to pollinator density. Apidologie 34: 227-235.
18. Richards AJ (2001) Does low biodiversity resulting from modern agricultural practice affect crop pollination and yield? Annals of Botany 88: 165-172.
19. Nauen R, Ebbinghaus-Kintscher U, Salgado VL, Kaussmann M (2003) Thiamethoxam is a neonicotinoid precursor converted to clothianidin in insects and plants. Pesticide Biochemistry and Physiology 76: 55-69.
20. Castle SJ, Byrne FJ, Bi JL, Toscano NC (2005) Spatial and temporal distribution of imidacloprid and thiamethoxam in citrus and impact on Homalodisca coagulata populations. Pest Management Science 61: 75-84.
21. Byrne FJ, Toscano NC, Urena AA, Morse JG (2007) Toxicity of systemic neonicotinoid insecticides to avocado thrips in nursery avocado trees. Pest Management Science 63: 860-866.
22. Nguyen H, Nieh JC (2012) Colony and Individual Forager Responses to Food Quality in the New World Bumble Bee, Bombus occidentalis. Journal of Insect Behavior 25: 60-69.
23. Gruter C, Moore H, Firmin N, Helantera H, Ratnieks FLW (2011) Flower constancy in honey bee workers (Apis mellifera) depends on ecologically realistic rewards. Journal of Experimental Biology 214: 1397-1402.
24. Nieh JC, Leon A, Cameron S, Vandame R (2006) Hot bumble bees at good food: thoracic temperature of feeding Bombus wilmattae foragers is tuned to sugar concentration. Journal of Experimental Biology 209: 4185-4192.
25. Sadler N, Nieh JC (2011) Honey bee forager thoracic temperature inside the nest is tuned to broad-scale differences in recruitment motivation. Journal of Experimental Biology 214: 469-475.

26. Whitney HM, Dyer A, Chittka L, Rands SA, Glover BJ (2008) The interaction of temperature and sucrose concentration on foraging preferences in bumblebees. Naturwissenschaften 95: 845-850.
27. Farina WM, Wainselboim AJ (2001) Changes in the thoracic temperature of honeybees while receiving nectar from foragers collecting at different reward rates. Journal of Experimental Biology 204: 1653-1658.
28. Moffatt L (2001) Metabolic rate and thermal stability during honeybee foraging at different reward rates. Journal of Experimental Biology 204: 759-766.
29. Brittain C, Potts SG (2011) The potential impacts of insecticides on the life-history traits of bees and the consequences for pollination. Basic and Applied Ecology 12: 321-331.
30. da Silva CI, Bordon NG, da Rocha Filho LC, Garofalo CA (2012) The importance of plant diversity in maintaining the pollinator bee, Eulaema nigrita (Hymenoptera: Apidae) in sweet passion fruit fields. Revista de biologia tropical 60: 1553-1565.
31. Jha S, Kremen C (2013) Resource diversity and landscape-level homogeneity drive native bee foraging. Proceedings of the National Academy of Sciences of the United States of America 110: 555-558.
32. Mayer C, Michez D, Chyzy A, Bredat E, Jacquemart AL (2012) The Abundance and Pollen Foraging Behaviour of Bumble Bees in Relation to Population Size of Whortleberry (Vaccinium uliginosum). Plos One 7.
33. Murray TE, Fitzpatrick U, Byrne A, Fealy R, Brown MJF, et al. (2012) Local-scale factors structure wild bee communities in protected areas. Journal of Applied Ecology 49: 998-1008.
34. Goulson D, Lepais O, O'Connor S, Osborne JL, Sanderson RA, et al. (2010) Effects of land use at a landscape scale on bumblebee nest density and survival. Journal of Applied Ecology 47: 1207-1215.
35. Carvell C, Meek WR, Pywell RF, Goulson D, Nowakowski M (2007) Comparing the efficacy of agri-environment schemes to enhance bumble bee abundance and diversity on arable field margins. Journal of Applied Ecology 44: 29-40.
36. Hoehn P, Steffan-Dewenter I, Tscharntke T (2010) Relative contribution of agroforestry, rainforest and openland to local and regional bee diversity. Biodiversity and Conservation 19: 2189-2200.
37. Klein AM, Brittain C, Hendrix SD, Thorp R, Williams N, et al. (2012) Wild pollination services to California almond rely on semi-natural habitat. Journal of Applied Ecology 49: 723-732.
38. Krupke CH, Hunt GJ, Eitzer BD, Andino G, Given K (2012) Multiple Routes of Pesticide Exposure for Honey Bees Living Near Agricultural Fields. Plos One 7.
39. Laurent FM, Rathahao E (2003) Distribution of C-14 imidacloprid in sunflowers (Helianthus annuus L.) following seed treatment. Journal of Agricultural and Food Chemistry 51: 8005-8010.

This document/publication is also available on our website at:

http://www.defra.gov.uk/environment/quality/chemicals/pesticides/insecticides-bees/

PB 13937