Comment on "A Common Pesticide Decreases Foraging Success and Survival in Honey Bees"

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Henry *et al*. (Reports, 20 April, p. 348) used a model to predict that colony collapse in honey bees could be precipitated by pesticide-induced intoxication that disrupts navigation. Here, we show that collapse disappears when the model is recalculated with parameter values appropriate to the season when most pesticide-treated flowering crops bloom.

Systemic neonicotinoids, such as thiamethoxam and imidacloprid, are currently among the most widely used insecticides in crop protection (*1*). Neonicotinoids are applied as foliar sprays or seed dressings, and the chemical pervades the plant systemically to protect it against insect pests (*2*). Honey bees (*Apis mellifera*) ingest residues of these pesticides when they consume nectar and pollen from neonicotinoid-treated flowering crops (*3*), and there is concern that this may contribute to colony collapse because the neonicotinoids are neurotoxic to insects (*4*). Consequently, it is important that pesticide regulators assess the risks that dietary pesticides pose to honey bees.

Laboratory trials have shown that doses of dietary neonicotinoid at trace levels (up to 10 parts per billion) are capable of harming individual honey bees at sublethal levels (*5*). For example, dietary exposure to trace dietary imidacloprid is expected to reduce behavioral performance in adult honey bees by between 6 and 20% (*6*). However, in a social insect like the honey bee, the ecologically relevant entity is the colony, not the individual. Therefore, it is important for risk assessors to establish whether the effects observed on individuals in the laboratory translate into impacts on colonies.

No published field experiment has yet had sufficient statistical power to detect effects on colonies of the magnitude observed on individual bees in the laboratory (*6*). When decisive experiments are unavailable, scientists may instead make forecasts with models and computer simulations. This approach has been used in the case of global climate change (*7*), and the results have the potential to be highly influential among policy-makers and regulators. In their recent paper, Henry *et al*. (*8*) used this approach and predicted impact on honey bee colonies of dietary thiamethoxam. Specifically, Henry *et al*. (*8*) solved a model of colony dynamics (*9*) and concluded that collapse would be precipitated because pesticide-induced intoxication disrupts navigation and foragers fail to return home. Here, we show that the prediction may be inaccurate when more environmentally relevant parameter values are used.

Henry *et al*. populated virtually all parameters in their model with empirically based values except one, namely *w*. Parameter *w* moderates the maximum daily rate of production of new workers, L, so that it has a density-dependent sigmoidal response: $L \times N / (N + w)$, where N is the number of adult bees in the colony. Thus, *w* represents the colony size at which new workers are produced at half the maximum rate. The originators of the model (*9*) appear to have chosen a value of *w* to generate model outputs that fit observations of the average age of onset of foraging by adult bees and their overall life span (*10*).

The model's output is very sensitive to the value of *w* (Fig. 1). Like the model's originators (*9*), Henry *et al*. assumed that $w = 27{,}000$ (*8*), but this is unrealistic because a colony of 18,000 adult bees (*8*) then grows only by 11% in a month in the absence of pesticide (Fig. 1). In spring or early summer, which is when bees in Europe are typically exposed to neonicotinoid-treated mass-flowering crops such as oilseed rape (*Brassica napus*) (*8*, *11*), a colony of this size can increase by >40% over 30 days (*12*, *13*), which is consistent with $w \approx 16{,}000$ (Fig. 1). Indeed, using $w = 16{,}000$ in the model very accurately predicts observations of adult life span on similarly sized colonies in the absence of pesticide (*10*). Specifically, we find a very good correspondence between model and observations (*10*) in both the average age of onset of foraging by bees (model = 17.8 days versus observed = 17.7 to 19.4) and overall adult life span (model = 24.3 days versus observed = 22.3 to 22.8). Thus, our value for *w* is at least as plausible as that used by Henry *et al*. We speculate that Henry *et al*.'s $w = 27{,}000$ may be more appropriate for forecasting pesticide effects on a slow-growing colony, perhaps in autumn.

When we recalculate the model using $w = 16{,}000$ and with pesticide-induced loss of foragers at the rates measured by Henry *et al*., pesticide exposure severely reduces colony size only if the intoxicated workers navigate an unfamiliar landscape (Fig. 1). However, the experimental doses of the neonicotinoid pesticide thiamethoxam administered by Henry *et al*. are daily totals, which would seem to assume that bees forage from a treated crop repeatedly throughout the day. The experimental doses are therefore appropriate only to bees operating in a familiar landscape. From this interpretation, the model predicts that a month of pesticide exposure leaves

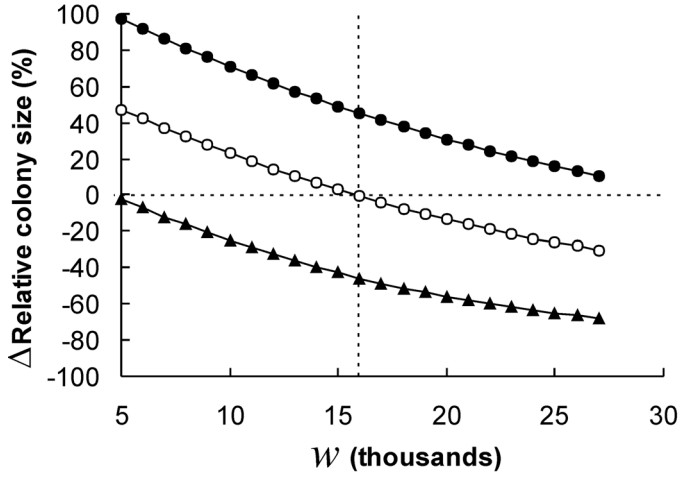

Fig. 1. The change in size of a honey bee colony over 30 days (*y* axis) relative to the parameter that governs the rate of production of new adult workers, *w* (*x* axis), for three scenarios of pesticide exposure. Upper curve (closed circles), background mortality with no additional mortality due to pesticides; middle curve (open circles), background mortality with additional mortality due to pesticide-induced navigation failure in a familiar landscape; lower curve (closed triangles), background mortality with additional mortality due to pesticide-induced navigation failure in an unfamiliar landscape. Horizontal dashed line indicates zero colony growth; vertical dashed line indicates the value of $w = 16{,}000$. In these solutions, the initial size of the colony is $N = 18{,}000$ adults, but $N = 15{,}000$ produces virtually identical results. Other parameter values are as given in Henry *et al*. (*1*).

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colony size virtually unchanged (Fig. 1) and would not precipitate colony collapse.

As Henry *et al*.'s experiments so elegantly demonstrate, there is no question that dietary thiamethoxam harms honey bee colonies by elevating the mortality of adult foragers through navigation failure, at least when the entire daily intake of a forager is consumed in a single dose. However, what is at issue is whether thiamethoxam is capable of causing colony collapse. Our results suggest that dietary thiamethoxam would not precipitate collapse in healthy colonies in spring, but this does not rule out the possibility that colonies will be more vulnerable later in the year when their capacity to replace lost workers has diminished. Based on our analysis, we argue that (i) the forecast impact of thiamethoxam on honey bees is nuanced, being highly contingent on colonies' capacity for producing workers; (ii) pesticide regulators should be cautious in using this model's outcomes when formulating a stance on controlling the future use of thiamethoxam; and (iii) colony-growth models may have a very important role in future risk assessments, but further research is required to ensure that they are fully validated and appropriately configured for the environmentally relevant context in which they are to be applied.

References and Notes

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