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

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

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Cresswell and Thompson have suggested an elegant way to improve honey bee colony simulations when forecasting the fate of colonies exposed to pesticides. Following their recommendations, we rescaled the model on a sound empirical data set. The adjusted forecast is bleaker than their tentative scenario.

Henry *et al*. (*1*) reported that sublethal doses of thiamethoxam, a neonicotinoid pesticide used on some common flowering crops, reduce the ability of exposed foraging honey bees to find the way back to their colony. The daily mortality probability due to homing failure, termed m_{hf}, was estimated to lie somewhere between 0.102 and 0.316 for foragers exposed and released 1 km away from their colony. Honey bee population models (*2*) predict that this abnormal mortality level causes a major deviation from the expected demographic trajectory. Models were run with 1-month exposure durations, with the underlying idea to simulate colonies exposed to treated oilseed rape.

Cresswell and Thompson (*3*) have proposed an adjustment of the population model and found no population change over this duration, at least with the least pessimistic exposure scenario (m_{hf} = 0.102). Specifically, they have suggested an elegant way to assess w, the only model parameter that could not be calculated from empirical data. Parameter w is a negative feedback constant that moderates the production rate of new workers as the colony matures. Cresswell and Thompson cleverly used empirical colony growth data to infer w. They assumed that a colony of 18,000 individuals may grow by 40% in a month during oilseed rape blooming period (*4*), which is reached with w = 16,000. When this analytical solution is transposed to the exposure scenario, no population change is detected [figure 1 in (*3*)]. Cresswell and Thompson noted that our parameterization (w = 27,000) assumed an 11% growth only in the absence of pesticides and therefore predicts an excessive decrease for exposed colonies (–30% with m_{hf} = 0.102).

The technical comment by Cresswell and Thompson is a sound cautionary note about simulation-based risk assessment of nonintentional pesticide effects. However, we would like to rectify an inaccurate statement in their comment. We did not claim that our simulation outcome had predicted colony collapse due to homing failure. Instead, we concluded that the levels of homing failure we measured are high enough to cause "a major deviation from the expected dynamic" (*1*). This conclusion is not ruled out by Cresswell and Thompson's model adjustment, as is merely illustrated by the virtually constant colony size gap (a ~45% difference) between exposed and nonexposed scenarios, regardless of the chosen w value [figure 1 in (*3*)]. Furthermore, we believe that the tentative value of 40% for colony growth on which Cresswell and Thompson have based their reasoning is not robust. It seems that it was obtained from three monitored colonies only, and no indication is given on the use of oilseed rape in the vicinity (*4*). Given the tremendous variability one usually observes among colonies, any attempt to derive model parameters from empirical data deserves stronger support. Here, we followed Cresswell and Thompson's valuable suggestion to solve the calculation of w, using a strong empirical data set.

We reanalyzed the ECOBEE (Ecological Honeybee Colony Monitoring) data set used in our original study to set a range of realistic starting values for colony size (*1*). ECOBEE is managed by our research groups with the objective to provide ecologists with detailed honey bee colony dynamic data under real beekeeping management conditions. Colony monitoring data, including adult population size, are collected biweekly within a network of about 50 colonies per year. Over the 2008 to 2011 beekeeping seasons— i.e., before thiamethoxam was marketed for oilseed rape protection in our study area— a total of 208 colonies have been monitored. They were allocated into 40 apiaries, evenly distributed over the 450-km^2 study area in order to cover a wide range of landscape contexts. As explained in our original study, this territory is an intensive cereal farming system where oilseed rape accounts for 8 to 10% of total land cover.

We computed an empirical colony size model to derive real colony growth data and to recompute exposure simulations accordingly. Colony size was modeled using generalized additive mixed models (GAMM). This modeling technique allows adjusting a temporal spline based on maximum likelihood, while giving the possibility to account for repeated measurements on the same colonies within a given year. The temporal axis was scaled on the Julian date since the beginning of oilseed rape blooming. Blooming dates are available from local long-term phenological surveys (*5*). The temporal spline predicts a steep population growth encompassing the blooming period, and a gradual decline thereafter, as colonies expend less effort into reproduction and

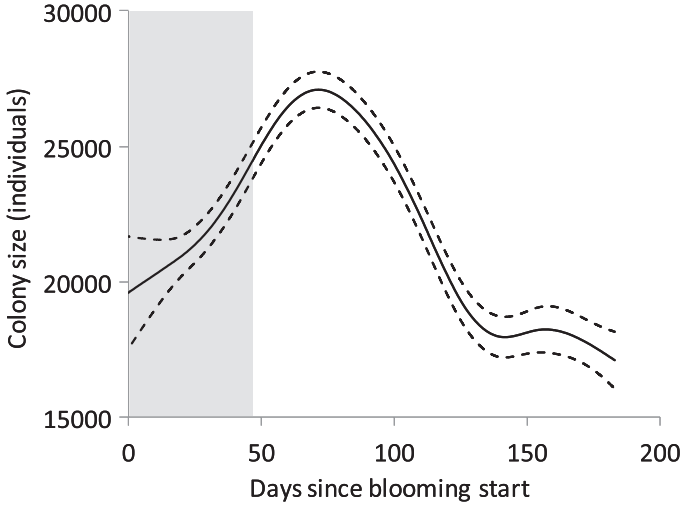

Fig. 1. Empirical honey bee colony size data (±1 SE) used to recalculate population models. Colony size is measured biweekly by weighting all hive elements (comb, frames, and supers) with and without bees, as a part of the ECOBEE monitoring facility. Seasonal changes in colony size are modeled by a temporal spline (GAMM). The shaded area denotes oilseed rape blooming period.

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more into food storage (Fig. 1). Although the oilseed rape blooming period hardly lasts more than 1 month in a particular field, there is a substantial temporal lag among fields and phenotypes, so that blooming period covered on average 48 days at the territory scale during the 2009 to 2011 ECOBEE monitoring program. To pinpoint the most plausible range of values for w, we sought for the analytical solutions that matched the average colony size values observed during different 30-day periods: (i) the initial blooming period (days 0 to 30 on the temporal axis, 11.7% growth), (ii) the full blooming period (days 10 to 40, 15.0%), and (iii) the late blooming period encompassing the steepest population growth (days 18 to 48, 18.7%). Analytical solutions for w were 24,932, 22,880, and 20,886, respectively, and also returned a close correspondence between observed and theoretical average age of onset of foraging (observed = 17.7 to 19.4 days, model = 18.2 days) and overall adult life span (observed = 22.3 to 22.8, model = 24.6). When homing failure was set to $m_{hf} = 0.102$, predicted population changes were –28.6%, –25.5%, and –22.1%, respectively.

These empirical-based scenarios are more pessimistic than the steady colony state predicted by Cresswell and Thompson. However, we agree that substantial improvement is needed before one could use honey bee colony modeling in its current form for risk assessment. We initially used modeling as a tool to get estimates of what observations made at the individual level would imply for the colony as a whole. Sound model adjustments have been proposed (*3*), but further issues remain to be documented to gain accuracy. Among others, homing failure should be re-evaluated with regard to (i) doses matching in-hive exposures of conspecifics and larvae by contaminated pollen and honey (*6*) and (ii) acute versus chronic exposure regimes at the foragers' life scale. The latter aspect, in particular, is still an unsolved debate. It is currently unclear whether acute experimental exposures overestimate sublethal effects compared with chronic regimes (*7*). Likewise, population modelers should consider trying different values of the egg-laying rate, which appears to follow a sharp decline after oilseed rape blooming, as well as the post-exposure homing distance foraging honey bees need to cover. Those two parameters are expected to be largely influential in the procedure.

References and Notes

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