Rapid behavioral maturation accelerates failure of stressed honey bee colonies

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Many complex factors have been linked to the recent marked increase in honey bee colony failure, including pests and pathogens, agrochemicals, and nutritional stressors. It remains unclear, however, why colonies frequently react to stressors by losing almost their entire adult bee population in a short time, resulting in a colony population collapse. Here we examine the social dynamics underlying such dramatic colony failure. Bees respond to many stressors by foraging earlier in life. We manipulated the demography of experimental colonies to induce precocious foraging in bees and used radio tag tracking to examine the consequences of precocious foraging for their performance. Precocious foragers completed far fewer foraging trips in their life, and had a higher risk of death in their first flights. We constructed a demographic model to explore how this individual reaction of bees to stress might impact colony performance. In the model, when forager death rates were chronically elevated, an increasingly younger forager force caused a positive feedback that dramatically accelerated terminal population decline in the colony. This resulted in a breakdown in division of labor and loss of the adult population, leaving only brood, food, and few adults in the hive. This study explains the social processes that drive rapid depopulation of a colony, and we explore possible strategies to prevent colony failure. Understanding the process of colony failure helps identify the most effective strategies to improve colony resilience.

behavioral development | temporal polyethism | colony collapse disorder | compartment model | RFID

High honey bee colony failure rates are a continuing concern. In North America, annual colony losses averaged 29.6% between 2006 and 2013 (1). In some cases, colony failure has been so rapid that outwardly healthy colonies lost most of their adult bees in a matter of weeks (2–4). This has been described as colony collapse disorder (3).

Many stressors are contributing to bee colony failure, most with a strong anthropogenic component. These include parasites and pathogens, pesticides, and nutritional stresses (4). The parasitic mite *Varroa destructor* and its associated viruses (4) and the gut parasite *Nosema* have both been linked to high colony failure rates (5). There is increasing concern about the role of pesticides and agrochemicals in honey bee colony losses (6), and nutritional deficits arising from limited floral diversity and abundance have also been linked to elevated rates of colony failure (7).

It is likely that all of these factors are contributing to the elevated rates of colony losses currently experienced across most of Europe and North America (1, 8). None of these issues can be easily resolved within the economic paradigm of intensive agriculture and apiculture. The problem of honey bee colony failure is proving hard to manage because colonies often transition from apparent health to almost complete population loss within a few weeks or less (3, 9), which is often too fast for beekeepers to intervene and attempt to save their colonies.

The failure of a honey bee colony is a breakdown of a society. Bees within a colony interact to maintain the colony as a homeostatic system that has been described as a superorganism (10). Understanding why and how colonies fail therefore requires more than analyzing how individual bees react to stressors. Here we explored via empirical study and modeling how individual stress responses of honey bees interact with social dynamics to cause colony failure.

Honey bees, like many social insects, delay leaving the protected environment of the nest to forage until later in adult life (11, 12). Bees display a predictable pattern of behavioral development. They begin adult life performing in-hive tasks, and, when 2–3 wk old as adults, they become foragers gathering floral resources (13–15). Varied stressors, such as a loss of foragers, starvation (16, 17), or disease (5, 18, 19), can cause bees to accelerate their behavioral development and forage precociously. The transition to foraging is socially regulated such that the presence of an established foraging force in the colony delays the onset of foraging in young bees (20–22). As a consequence of this system, if the colony loses foragers, young bees accelerate their behavioral development to replace them (23, 24). Several diverse stressors cause a similar response in individual bees. These include individual or colony starvation (16, 17), pollen deprivation (25, 26), disease (5, 18), and even wax deprivation (27).

Precocious foragers may not be optimally adapted for the tasks outside the hive (28). There is evidence that precocious foragers are heavier and less efficient fliers than normal-aged foragers (28), and the flight muscle biochemistry of precocious foragers differs from that of typical foragers (29, 30). If young bees reduce the effectiveness of the foraging force, this may have serious consequences for the colony.

Here we used radio frequency tags (RFID) (31–33) to continuously monitor flight activity of focal bees in experimental colonies to determine how foraging performance varied with the age at which bees commenced foraging. Our data revealed that several key aspects of forager performance were strongly age

Significance

Honey bee colony death rates are unsustainably high. While many stressors have been identified that contribute to this problem, we do not know why colonies transition so rapidly from a state of apparent health to failure. It is well known that individual bees react to nutritional and pathogen stresses by foraging precociously: our study explains how colony failure arises from the social responses of individual bees to stress. We used radio tracking to monitor performance of bees and found that workers who begin foraging prematurely perform very poorly. This compounds the stresses on the colony and accelerates failure. We suggest how colonies at risk can be identified early, and the most effective interventions to prevent failure.

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dependent. We then constructed a mathematical model of colony demography inspired by Khoury et al. (34, 35) to consider how this new information might change trajectories of colony growth or decline in chronically stressed colonies. We present an explanation for why colonies appear to change so rapidly from conditions of apparent health to colony failure, propose how to identify colonies at risk for failure, and investigate possible strategies to prevent colony failure.

Results

The RFID data showed when a bee was inside the hive and when outside, and therefore the time spent on orientation and foraging activities (Fig. 1). Foraging is typically preceded by nonforaging orientation flights (36, 37). The average cumulative duration of orientation flights has been estimated at 30.89 min (36); therefore, for the purpose of this study, we classed bees that had spent >30 min outside the hive as foragers.

To experimentally induce precocious foraging, we created three "single-cohort colonies" (SCC) in which all bees were 1-d-old adults at the time of establishment (24, 38, 39), and three colonies that had a normal-worker demography (NDC). SCC (24, 40–42) is an established method for creating a colony with a younger age distribution of foragers. It has been argued that, for many social insects, early foraging by individuals might be a response to individual stress resulting in a shift in the demography of the foraging force (18): SCC provides a social manipulation to reduce the modal age of foragers by reducing social inhibition independent of any pathogen pressure. As expected, in SCC, more bees began foraging when less than 14 d old than in NDC (Fig. 1). A proportion of bees in SCC also start much later than normal. This may be due to the shift in colony needs within the hive once it commences brood production and bees are needed

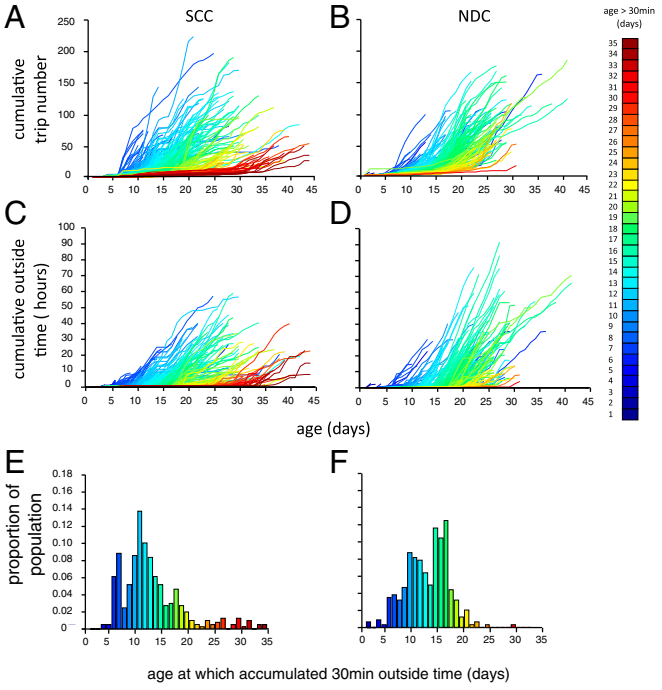

Fig. 1. Cumulative trip number (*A* and *B*) and cumulative time outside the hive (*C* and *D*) and distribution of ages at which each bee commenced foraging (*E* and *F*) for SCC and NDC. In all plots, each bee is color-coded according to the age (in days) at which it exceeded 30 mins cumulatively outside the hive and was classed as a forager [the legend: age > 30 mins (days)] to illustrate how, in both SCC and NDC, performance varied with age of foraging onset. More bees from the SCC (*E*) became foragers when less than 14 d than for the NDC (*F*) (χ^2 = 20.47, df = 1, P = 0.0001).

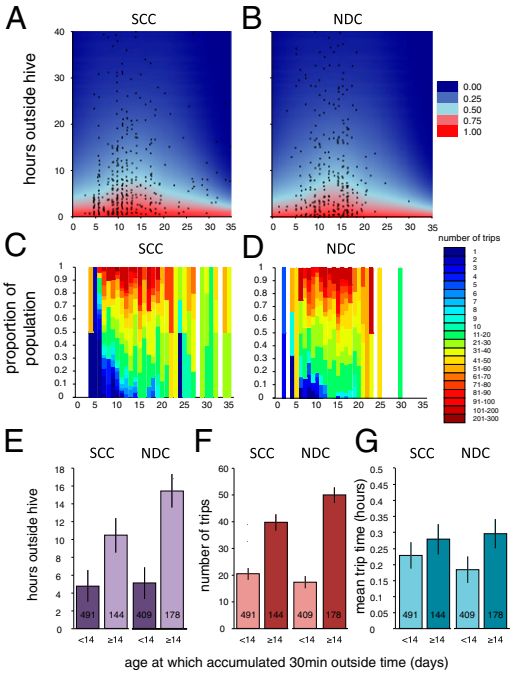

Fig. 2. (*A* and *B*) Cumulative time outside the hive during the lifespan of all bees overlaid on predicted survival probabilities (from polynomial survival regression) dependent upon the age at which bees began foraging in SCC (*A*) and NDC (*B*). (*C* and *D*) Number of completed foraging trips in lifetime plotted against age at which bees commenced foraging in both SCC (*C*) and NDC (*D*). (*E*–*G*) Comparisons of total cumulative time outside the hive (*E*), total lifetime trip number (*F*), and mean trip duration (*G*) for bees that commenced foraging when <14 d old and ≥14 d (mean ± SE) in both SCC and NDC colonies. Numbers in bars are sample sizes. Generalized linear modeling analyses of effects of age of foraging onset and colony type on each of these parameters in Table S1.

for nursing roles until the first new generation of bees has emerged (Fig. 1).

In both SCC and NDC, foraging performance varied with the age at which bees began foraging. Bees' survival per hour of time spent outside the hive (Fig. 2 *A* and *B*) and number of completed trips per bee (Fig. 2 *C* and *D*) were greatest for bees that began foraging at about 14 d old. Polynomial survival regression (Fig. 2 *A* and *B*) accounting for colony type and replicate (colony type was treated as a fixed factor, whereas replicate was included as a random factor) showed a significant relationship between age at onset of foraging and total time spent outside the hive (χ^2 = 34.04, P < 0.001, n = 749).

The consequences of precocious foraging were severe. Bees that began foraging when <14 d old spent less time outside the hive, completed fewer flights, and performed longer foraging trips than bees that began foraging at ≥14 d (Fig. 2 *E*–*G* and Table S1). In both SCC and NDC, the likelihood of surviving past 30 min of flight activity (and becoming a forager) increased as age of first flight increased (Fig. 3 and Table S2).

Modeling Consequences of Precocious Foraging for Colony Function. Precocious foraging is a reaction to various acute stressors (5, 16–18, 23, 24), which may be adaptive, as it rapidly replaces any losses to the foraging force to enable more food gathering. There is a risk, however, that chronic stress could lead to a progressively younger and less effective foraging force. To examine the consequences of chronic stress on the colony, we developed a compartment model of honey bee population dynamics, implemented as a set of delay differential equations, where survival and food collection per forager varied with age (Fig. 4).

Modeling forager performance and survival as age dependent caused a dramatic change in trajectories of colony growth and

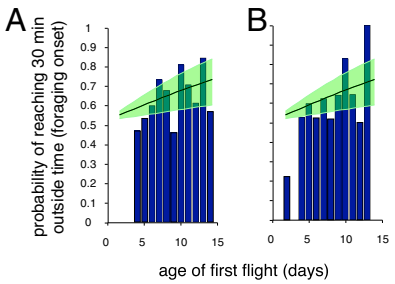

Fig. 3. Proportion of bees that successfully accumulate more than 30 min of time outside the hive plotted against age of first flight for SCC (*A*) and NDC (*B*). Summary of logistic regression analysis of these data in Table S2. Overlaid line is fit from logistic regression analysis of mortality during the first 30 min outside colony and age of first flight, green area is SE of fit (Table S2).

failure (Figs. 5 *A*–*D* and 6) compared with earlier models in which performance was age independent (34). Model colonies buffered small changes in forager mortality to maintain a stable population (Fig. 5 *A*–*C*), but if forager mortality was chronically elevated above a threshold, then colony population was unsustainable. In conditions of chronic high mortality, the population stabilized for a period and brood rearing continued (Fig. 5*D*) but the foraging force became progressively younger and less effective. Ultimately, this triggered a very rapid terminal decline once the forager force became unable to sustain food levels. Food limitation prompted accelerated recruitment to the foraging force: Most bees became foragers and died soon after, leaving abandoned brood in the colony.

In model colonies, the time to failure was sensitive to the initial amounts of brood and food (Fig. 7). Increasing stored food at the start of the simulation extended the time to collapse (Fig. 7*B*). Adding some brood had a similar effect to a point, but, above a threshold, adding more brood accelerated colony collapse as the additional brood depleted food stores more quickly (Fig. 7*A*). Progressive supplemental feeding also extends colony life (Fig. 8*B*). Inhibiting precocious foraging, however, did not prevent colony collapse (Fig. 8 *C* and *D*). Although inhibiting the transition of hive bees to foragers kept bees safely in the hive, it also reduced the size of the foraging force and inhibited the collection of food. This accelerated the point at which food limited brood production and accelerated colony population decline.

Discussion

Individual honey bees react to a range of social, pathogen, and nutritional stressors by accelerating their behavioral development and foraging precociously (5, 16–18, 23, 24), but this is not without significant cost to the individual bee, or potentially to its colony. Precocious foragers had a greatly reduced effective foraging life compared to normal-aged foragers (Figs. 2 and 3). We used modeling to explore the possible impacts of age-dependent foraging performance on a colony experiencing chronic stress. Initially, precocious foraging enabled colonies to buffer the impacts of stress. Colony populations stabilized for a period, and brood rearing continued (Fig. 5*D*), but, in effect, this concealed the true extent of the chronic stresses on the colony until it entered a final population decline as its buffering capacity was exhausted. The foraging force became progressively younger and less effective until it could no longer sustain food levels in the colony. This triggered a very rapid terminal decline as food limitation prompted accelerated recruitment of hive bees to the foraging force. Most bees that became foragers died soon after, leaving abandoned brood in the colony.

These model trajectories of population decline are strikingly similar to field reports of honey bee colony collapse (3) where colonies that appeared healthy were found later to be almost devoid of adult bees and with abandoned brood (2, 3). The results from this model shed light on why honey bee colonies often appear to undergo a very rapid state change from a condition of apparent health and growth to a crash in the adult population.

Several earlier models of honey bee colony population dynamics have been developed (34, 35, 43–46). Of these, the most relevant studies are those of Schmickl and Crailsheim (46), Makela et al. (45), and Khoury et al. (35), which have all modeled how changes in food and mortality can influence colony growth. However, to our knowledge, our approach is the first to recognize (based on our new data) how bee foraging performance varies with age. The decline in foraging performance as bees aged meant that stressed colonies with a younger foraging force became less efficient at gathering food. This interaction dramatically altered the trajectories of colony population collapse compared with earlier similar models that predicted a slow decay in adult bee populations rather than a sudden decline (35). Our model is the first, to our knowledge, to display dynamics of colony population collapse that are similar to field reports of colony collapse disorder.

In model colonies, a dramatic population collapse (similar to some reports of CCD) was seen only when colonies began with quite a low base of food stores and experienced chronically sustained forager death rates of more than twice that experienced by a healthy hive (Fig. 5*D*). This would be in the order of 30% of foragers dying each day. These are rather extreme conditions; hence, modeling suggests dramatic population collapses can be expected to be rare events. Such high rates of forager mortality could be caused by chronic diseases such as Varroensis or Nosema (4, 5, 19), or perhaps agrochemicals (6), which may explain why incidences of colony collapse have seemed to occur in sporadic clusters (3, 4). In model colonies, the time to failure was sensitive to the initial amounts of brood and low food stores (Fig. 7). This suggests that colonies may be at their most vulnerable when internal food stores and brood production are low. It may explain why failure is most common in spring as colonies attempt growth with food stores that have been depleted over winter (1). Our model proposes that food limitation imposed by inefficiency of a young foraging force would accelerate colony terminal decline. We note however that there are reports of CCD in which colonies died leaving some honey stores (3). In our model, we combined pollen (protein) and nectar/

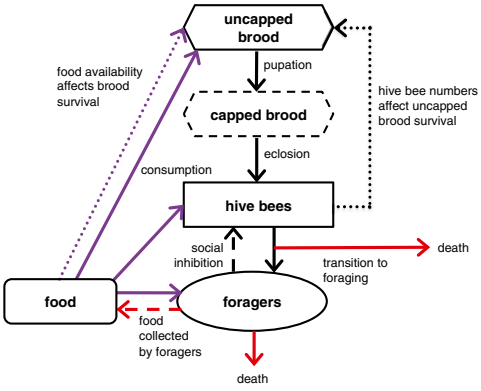

Fig. 4. Schematic representation of the model. Workers develop as brood, emerge as adult hive bees, and mature to foragers. Existing foragers inhibit hive bees from becoming foragers by social inhibition (20, 49). Purple lines represent consumption of food. Food also influences brood numbers indirectly because, in periods of low food influx, workers will cannibalize larvae (purple dotted line). Death rates during the transition from hive bee to forager, forager death rate, and the rate of food collection by foragers (indicated by red arrows) were functions of the age at onset of foraging. "Capped brood" appears in a box with a dashed border because it is not modeled explicitly, but the period that brood spends as capped pupae is modeled by a delay in the model between the time a larva enters pupation and emergence as an adult.

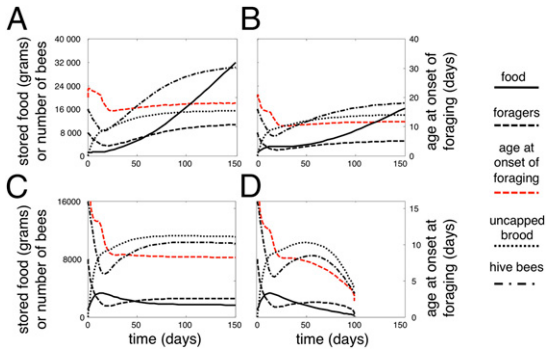

Fig. 5. The effects of stress on the model colony shown as plots of populations of uncapped brood (dotted line), hive bees (dash-dot black line), and foragers (dashed line); food (solid line) and age at onset of foraging (dashed red line) against time for different values of the underlying death rate. Death rate is expressed as the ratio between death rate of the simulated hive and a healthy hive (m_r). All hives start with 1 kg of food, 16,000 hive bees, 8,000 foragers, and no brood. (*A*) Plot for a healthy hive ($m_r = 1$); (*B*) $m_r = 1.6$; (*C*) $m_r = 1.9$. At this value, the hive will collapse eventually but not when $t < 150$ days; (*D*) $m_r = 2.0$. For this death rate, the forager population reached zero at about $t = 99$ d. At about 3 wk before collapse, the colony in *D* had 8,250 uncapped brood items, 6,780 hive bees, and 1,780 foragers. The mathematics are such that modeling could not continue beyond the point of zero foragers. Note that *A* and *B* are on a different vertical scale to *C* and *D*.

honey (carbohydrate) into a single category of food because too few field data exist on the fluxes of these in hives to consider them separately. A colony with abundant residual honey stores could still be nutrient-limited and fail to produce brood if it had no pollen. Better field data on the consequences of protein and carbohydrate limitation on colonies would certainly further improve our understanding of how nutritional factors contribute to colony growth and failure.

Effective interventions to stop colony failure hinge on early identification of vulnerable colonies. Beekeepers usually rely on snapshot assessments of brood production or colony honey stores to assess colony health, but our modeling suggests these parameters would be slow to react to a change in forager mortality (Fig. 5 *A*–*D*). Rather, our modeling suggests that forager mortality rate and age at onset of foraging may provide the best information on colony condition (Fig. 5 *A*–*D*).

Understanding the social dynamics that drive colony population decline is essential if effective preventative measures are to be developed. Our model framework allows exploration of the impacts of possible treatments. Here we focused on three (Figs. 7 and 8): supplemental feeding (either in the initial condition or progressive food supplementation throughout the simulation), adding brood, and blocking the precocious foraging response to stress. The latter might be achieved by supplementing colonies with pheromones that inhibit foraging in young bees (20). Of these possible treatments, progressive supplemental feeding was clearly the most effective in delaying or preventing colony population collapse (Fig. 8*B*). Adding brood also had a positive effect when a hive was marginal, but too much brood accelerated colony failure, because demands of the brood increased nutritional stress on the colony (Fig. 7*A*). Slowing worker behavioral development to prevent precocious foraging (20) was not at all effective, because this strategy also compromised food collection (Fig. 8 *C* and *D*).

Our modeling framework clarifies why honey bee colonies might collapse and refines hypotheses for how it might be prevented, but experimental studies are urgently needed to test the validity of these hypotheses. We recognize that there are many different stressors impacting bee colonies, and each has specific modes of action and symptoms. We do not try to capture this detail in our simple modeling approach, but our objective is to provide an overview of how colonies' internal demographic processes might generally react to stressors.

Given the difficulties beekeepers have in identifying stressed colonies while there is still time to intervene, better strategies to identify vulnerable colonies and reduce colony losses are urgently needed. Our findings propose an explanation for the enigmatic phenomenon of rapid colony population collapse. In situations of chronically elevated forager loss, the social mechanisms that normally stabilize colony function instead accelerate colony failure by causing a destructive positive feedback response that increases forager mortality and decreases forager age, resulting in colony population collapse. Monitoring rate of forager mortality may give a more immediate assessment of colony health than assessments of brood and stored food.

Materials and Methods

Experiments were carried out between February 2012 and May 2013. Honey bees (*Apis mellifera*) were obtained from research apiaries maintained at Macquarie University (Sydney, Australia). Bees were housed in four-frame nucleus hives each containing ~3,000 bees located inside a laboratory building but connected to the outside via a custom-designed entrance that separated entering and exiting bees into different channels, each with their own entrance. Single SCC and NDC colonies were constructed simultaneously and observed for 40 d. The experiment was replicated three times. Bees and brood to establish SCC and NDC colonies were sourced from the same seven research colonies. Each paired SCC and NDC was provided with new young sister queens. For NDC, ~3,000 bees (estimated by weight) were shaken off brood and honey combs from donor colonies into the nucleus hive box, which was then sealed with mesh and placed in a dark room at 24 °C for 2 d before connecting to the entrance tunnel and adding a new queen. SCCs were constructed from ~3,000 bees that were within 24 h of emergence from brood frames. To obtain newly emerged bees, brood combs were removed from research colonies and placed in an incubator maintained at 32 °C and 37% humidity overnight. Newly emerged bees were brushed from frames the following morning.

RFID System. Between 500 and 1,000 Radio Frequency Identification (RFID; Invengo Technology) tagged bees were introduced to each colony. RFID were glued to the dorsal thoraxes of newly emerged bees within 12 h of emergence. Each nucleus colony was equipped with two RFID antennae placed within a modified entrance to monitor each of the entering and exiting channels.

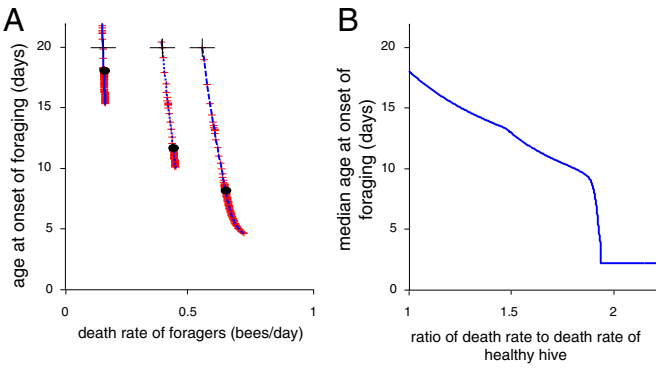

Fig. 6. (*A*) Plot of the age at onset of foraging as a function of the death rate of foragers as a function of time for different values of m_r (that is, the ratio between death rate of the simulated hive and a healthy hive). Each hive started with 1 kg of food, 16,000 hive bees, 8,000 foragers, and no brood, and the simulation was run until $t = 300$ d or the hive collapsed, whichever occurred sooner. The same values of m_r were used as in Fig. 5 *A*–*C*: solid line, $m_r = 1$; dotted line, $m_r = 1.6$; dashed line, $m_r = 1.9$. Each red cross is a time point at an integer value of *t*. The black cross is the initial value, and the black dot is the median value over all integer values of *t*. (*B*) Plot of the median age at onset of foraging (illustrated for three different values in *A*) as a function of the ratio of death rate of the simulated colony to the death rate of the healthy hive (m_r). The median age of onset of foraging drops rapidly for values of m_r that produce colonies that are close to collapse. When the colony collapses, then the median age of onset of foraging is 2 d, which is the minimum allowed in the model.

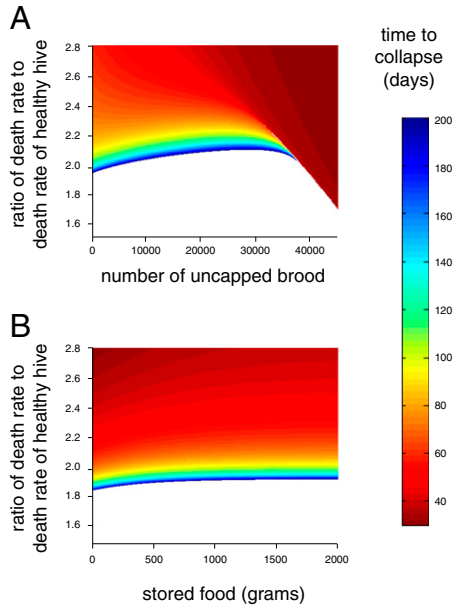

Fig. 7. Contour plots that show time to collapse as a function of m_r (the ratio of the death rate to the death rate of a healthy hive) and (*A*) initial number of uncapped brood and (*B*) initial mass of stored food. The white regions in the plots represent colonies that did not collapse within 200 d. In *A*, the hive had initially 1 kg of stored food, and in *B*, the hive had no uncapped brood at $t = 0$. In *A*, the modeling also assumed that there was an equivalent number of capped brood items so that adults emerged continuously from the start of the simulation. The time to collapse increased as the brood numbers increased until it reached a maximum at about 25,000–30,000, depending on m_r. If the initial brood numbers were increased by about 10,000 beyond the value where this maximum occurred, then the hive collapsed very rapidly, which suggests that there is a maximum number of brood which can be comfortably fed without putting a colony under extreme food stress. The time to collapse was not particularly sensitive to the mass of stored food (Fig. 7*B*); indeed, increasing the mass of stored food present at $t = 0$ beyond 2 kg made a negligible difference to the results. Increasing the initial levels of stored food from zero to 2 kg increased the time to collapse by about 50 d and, for m_r between 1.8 and 1.95, could prevent collapse altogether.

RFID tags contained a unique 12-byte hexadecimal identifier that allowed us to individually track the life history of each bee. Data were collected on a PC (Windows) into a .csv file containing data for each successful trip for each bee, including the date and time the bee left the hive, the date and time the bee returned to the hive, and the RFID number for that bee. The RFID entrance tunnel was customized to detect >99% of all tagged bee entries and exits from the colony from adult emergence to the time of last detection. Trips that were filtered from the data included trips that lasted over 8 h (some bees spent one or more nights outside the hive, and these data skewed the comparisons) and those that lasted less than 10 s (trips less than 10 s long were considered misreads by the hardware and occurred infrequently and usually in addition to successfully logged trips). MATLAB (version 8.0) was used to analyze data and create figures.

Model Formulation. The population of uncapped brood is denoted by *B*, hive bees by *H*, foragers by *F*, and the weight of stored food by *f*.

The rate of change of stored food is given by the rate that food is collected by foragers minus the rate that it is consumed by adult bees and brood,

$$\frac{df}{dt} = c_T N(a) F - \gamma_A (F + H) - \gamma_B B,$$

where c_T is the quantity of food collected on a single trip (expressed in grams of food) and $N(a)$ is the number of trips that a forager makes each day as a function of age of onset of foraging, a; γ_A and γ_B are the average weight of food consumed per day by an individual adult bee or brood item, respectively.

The rate of change of the population of uncapped brood is given by

$$\frac{dB}{dt} = L \frac{f^2}{f^2 + b^2} \frac{H}{H + v} - \phi B.$$

The first term on the right-hand side models the production and survival of uncapped brood, which depends on the queen's laying rate L and the amount of stored food and the number of hive bees. Here b and ν are constants. Brood leaves the uncapped brood class at a rate of ϕ when it pupates.

The rate of change of the hive bee population is given by

$$\frac{dH}{dt} = \phi B(t - \tau) - R(H, F, f) H.$$

The first term models the emergence of adult bees that entered pupation τ days earlier. The second term models the recruitment of hive bees to foraging where

$$R(H, F, f) = \alpha_{\min} + \alpha_{\max} \left(\frac{b^2}{b^2 + f^2} \right) - \sigma \left(\frac{F}{F + H} \right).$$

The first two terms in the function $R(H,F,f)$ represent the intrinsic rate of transition from hive bee to forager and the enhanced transition rate when stored food is scarce. The last term represents social inhibition.

The rate of change of the forager population is modeled as

$$\frac{dF}{dt} = T(a) R(H, F, f) H - m_r M(a) F$$

where $T(a)$ is the transition survival, represented as the proportion of bees that leave the hive bee class that successfully become foragers. $M(a)$ is the death rate of foragers in a healthy hive as a function of age at onset of foraging a, and m_r is the ratio of the death rate in a hive under stress to the death rate of a healthy hive. The age of onset of foraging is given by $a = 1/R(H,F,f)$, and the foraging span is equal to $1/(m_r M(a))$.

The expressions for the functions $N(a)$, $T(a)$ and $M(a)$ that depend on the age at onset of foraging are

$$N(a) = \begin{cases} -0.02(a-10)^2 + 3.5 & \text{for } a \le 10 \\ 0.015(a-10)^2 + 3.5 & \text{for } a > 10 \end{cases}$$

$$T(a) = \begin{cases} -0.06(a-5) + 0.5 & \text{for } a \le 13.3 \\ 1 & \text{for } a > 13.3 \end{cases}$$

$$M(a) = \frac{(a - a_0)^4 + 3}{(4.94 + 0.8a)(a - a_0)^4}$$

where $a_0 = 2$ is the minimum age at which hive bees can become foragers in this model.

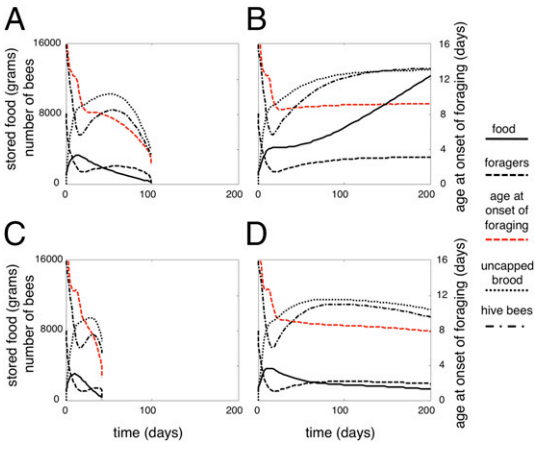

Fig. 8. The effect of in-hive additional feeding and treatment to delay onset of foraging in young bees. All these plots are for $m_r = 2$. (*A*) Colony with no treatment. (This is the same plot as Fig. 5*D*.) (*B*) Colony with in-hive feeding only with $C_F = 60$ grams per day. (*C*) Colony with pheromonal treatment only with $Q = 500$. (*D*) Colony with both in-hive feeding ($C_F = 60$) and pheromonal treatment ($Q = 500$). The line styles are the same as Fig. 5 *A*–*D*. The modeling suggests that in-hive feeding is generally helpful in preventing vulnerable colonies from collapse. Inhibiting precocious foraging, however, hastens colony collapse.

Most parameter values are taken from ref. 34, where they replicate the field data of Harbo (47); that is, $L = 2{,}000$, $f = 1/9$, $\nu = 5{,}000$, $\sigma = 1.3$, $\alpha_{\min} = 0.25$, $\alpha_{\max} = 0.25$, $b = 500$, $\gamma_A = 0.007$, and $\gamma_B = 0.018$. We also use $\tau = 12$ as worker bees pupate for 12 d, and $c_T = 0.033$. Estimating c_T is not easy from available data. Harbo (47) does not measure this directly, but from his data, it can be inferred that foragers returned 0.1 g food each day to the colony (47, 48). From our flight data, the mean number of trips per day for all bees (including those that did not complete any foraging trips) was approximately three. Therefore, for our model, we set $c_T = 0.033$.

In all of the simulations shown in Fig. 5, the age of onset of foraging changed over time and was a function of the actual death rate of foragers, m_r $M(a)$. We tracked these changes for $m_r = 1$, 1.6, and 1.9 and recorded the age of onset of foraging for each day, that is, for each integer value of t. Although the age of onset of foraging did change with time, there were long periods when it changed very little and so we took the median value of age of onset of foraging over the simulation as a measure of the typical age of onset of foraging for each value of m_r, which is the ratio of the death rate in the simulated hive to the death rate in a healthy hive. Fig. 6 shows three simulations, each corresponding to a different value of m_r, and a plot of median age of onset of foraging vs. m_r. The age of onset of foraging drops steadily as m_r increases and, as m_r approaches the critical value where collapse occurs before $t = 300$, the median age of onset of foraging plummets. Once collapse occurs within 300 d, the median age of onset is reduced to 2 d, which is the youngest age that bees are able to commence foraging in this model. The small kink in the curve of Fig. 6*B* at median age 13.3 is caused by the slope discontinuity of the function $T(a)$ when $a = 13.3$.

We included the effect of extra, in-hive feeding in the model by adding an extra constant term C_F to the equation for the rate of change of food so that the equation became

$$\frac{df}{dt} = c_T N(a) F - \gamma_A (F + H) - \gamma_B B + C_F.$$

The effect of pheromone treatment to mimic the presence of extra foragers and thus inhibit hive bee to forager transition was modeled by adding a constant Q in the numerator of the ratio of foragers to total adult bee in the transition function so that

$$R(H, F, f) = \alpha_{\min} + \alpha_{\max}\left(\frac{b^2}{b^2 + f^2}\right) - \sigma\left(\frac{Q + F}{F + H}\right).$$

Here Q effectively represents that number of foragers that the hive bees perceive to have been added due to the increased concentration of the social inhibition pheromone (Fig. 8 *C* and *D*).

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Supporting Information

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Table S1. Summary of linear mixed models examining life-time foraging parameters (Fig. 2 EFG) in relation to age of foraging onset

Dependent variable	Fixed factors	df	Estimate	SE	*F*	*P*
Total hours	**>14**	1	5.81	1.06	128.62	2.2e-16
	colony type	1	0.33	2.56	0.399	0.5619
	>14*colony type	1	4.75	1.47	10.49	0.0012
Total trips	**>14**	1	19.15	3.00	158.48	2.0e-16
	colony type	1	−3.09	3.10	0.024	0.8847
	>14*colony type	1	13.27	4.14	10.18	0.0014
Mean trip time	**>14**	1	0.05	0.04	14.43	0.0002
	colony type	1	−0.04	0.07	0.24	0.6526
	>14*colony type	1	0.06	0.04	1.95	0.1625

The dependent variables were total hours outside of hive, total trips taken, and mean time per trip. Whether a bee started foraging before reaching 14 d of age and the colony type (SCC or NDC) as well as the interaction term between the two was included as fixed factors, colony replicate was included as a random effect. The significant terms are highlighted in bold.

Table S2. Summary of a generalized linear mixed model of the probability that a bee would accumulate more than 30 min of outside time before dying as a consequence of onset of flight and colony type

Fixed term	df	χ^2	*P*
Colony type	1	0.1841	0.6679
Flight age	1	4.9906	0.0255
Colony type * flight age	1	0.1779	0.6732

The dependent variable, >30 min, was the probability that a bee would accumulate more than 30 min of time outside the hive before being lost. The age at which a bee started flying, the colony type (SCC or NDC), and the interaction term between the two were included as fixed factors; colony replicate was included as a random effect. Significant terms are highlighted in bold.