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Current Opinion in Insect Science

Death of the bee hive: understanding the failure of an insect society

Andrew B Barron

Since 2007 honey bee colony failure rates overwinter have averaged about 30% across much of North America. In addition, cases of extremely rapid colony failure have been reported, which has been termed colony collapse disorder. Both phenomena result from an increase in the frequency and intensity of chronic diseases and environmental stressors. Colonies are often challenged by multiple stressors, which can interact: for example, pesticides can enhance disease transmission in colonies. Colonies may be particularly vulnerable to sublethal effects of pathogens and pesticides since colony functions are compromised whether a stressor kills workers, or causes them to fail at foraging. Modelling provides a way to understand the processes of colony failure by relating impacts of stressors to colony-level functions.

Address
Department of Biological Sciences, Macquarie University, North Ryde, NSW 2109, Australia

Corresponding author: Barron, Andrew B (Andrew.Barron@mq.edu.au)

Current Opinion in Insect Science 2015, **10**:45–50

This review comes from a themed issue on **Social insects**

Edited by **Christina M Grozinger** and **Jay D Evans**

For a complete overview see the Issue and the Editorial

Available online 28th April 2015

http://dx.doi.org/10.1016/j.cois.2015.04.004

Introduction

Since 2007 the median annual honey bee colony loss rate in North America has been 29.6% (range: 22% in 2012 to 36% in 2008) [1,2]. Such high mortality rates are testing the ability of apiculturalists to maintain their bee stocks [1,3,4].

This period has also seen dramatic reports of mass deaths of bee hives, and cases of rapid colony depopulation with worker bees apparently disappearing from hives leaving just the queen, brood, and some food behind with no obvious cause of such a dramatic population collapse [5]. This phenomenon, termed colony collapse disorder (CCD), has galvanised research into why bee colonies are now failing at such high rates, and what might cause CCD. It is important to recognise that CCD is not the sole cause of the elevated honey bee colony failure rates [3] since in the majority of cases colony failures can be attributed to known stressors. The near global spread of the parasitic mite *Varroa destructor*, and its development of resistance to control measures has certainly driven up colony failure rates [3,6,7], but it is clear that neither CCD nor the general increase in incidences of colony failure can be attributed to any single cause. Both issues are massively multicausal. New research is examining how different stressors interact and synergise to impact bees, and the importance of sublethal effects of stressors that can cause colony failure by compromising individual and or colony function.

Causes

The list of pests, parasites and environmental stressors that have been linked to CCD is enormous [8]. There is now recognition that a stressor does not need to kill individual bees in order to contribute to colony failure. Any factor that compromises bees' abilities to forage effectively or otherwise service their colony can drive a colony into decline [9,10]. This recognition has focussed attention on the social consequences of sublethal effects of stressors on bees.

There is a great deal of concern about the possible impacts of a wide range of pesticides on honey bees at sublethal doses [11••]. Here I pay particular attention to the neonicotinoid insecticides and organophosphate miticides on honey bees: both are in common use in agriculture and apiculture: the former as crop treatments to kill pest insects and the latter as in-hive treatments to control *Varroa* mite. Both target cholinergic neurotransmission in arthropods with potentially very wide-ranging effects on insect physiology and behaviour [12]. Both classes of agrochemical can interfere with signalling in the mushroom bodies of the insect brain at sublethal and field-relevant doses [13] and impair learning and memory in honey bees [14]. If neonicotinoids are damaging learning and memory (and possibly navigation) this may explain why sublethal neonicotinoid exposure reduces successful homing after foraging in bees [9,15–17]. Building on a simple demographic model of a honey bee colony proposed by Khoury *et al.* [18], Henry *et al.* [9] proposed that the forager losses they observed as a consequence of sublethal pesticide exposure could potentially cause colony failure. It now seems clear that sublethal neonicotinoid exposure can compromise colony function and may result in colony failure with symptoms resembling CCD [19].

Similarly, diseases do not need to kill individual bees to kill a bee hive: if they sufficiently compromise colony function this can cause colony failure. From the perspective of a colony maintaining its resource base and population it makes no difference if a pathogen kills worker bees out right, or simply prevents them successfully returning home from foraging. Both the gut parasite *Nosema ceranae* [20] and the Israeli Acute Paralysis Virus [21,22] reduce efficiency of foraging and increase the numbers of bees that fail to return to the hive from foraging trips. Nosema infections can kill colonies [23] with features similar to that considered diagnostic of CCD [24].

Stressors interact to compromise colony function

In the current apicultural setting a honey bee colony is rarely dealing with a single stressor in isolation, and stressors can interact in complex ways to alter worker physiology and colony function. Treatment with field-relevant sublethal doses of the organophosphate miticide coumaphos and the neonicotinoid pesticide imidacloprid in combination had a greater impact on bees' odour learning and odour discrimination than treatment with either compound alone [14], even though there was no evidence of synergy between the two pesticides in a mortality assay [14]. Pesticides at sublethal doses can interact with complex, and even unpredictable, physiological effects that may not kill bees, but could reduce their performance and survival in a foraging situation. Field exposure of bees to a wide range of pesticides (including fungicides) sprayed on crops can also increase bees' susceptibility to nosema infection, which (as described above) can impair foraging performance [11••,25].

The impacts of pesticides on bees vary with environmental conditions. Low temperatures and low protein diet both increased susceptibility of bees to nicotine poisoning [26,27], which may in part explain why the impacts of pesticides on bee colonies can vary seasonally. Colonies experimentally chronically treated with sublethal doses of the neonicotinoid pesticides imidacloprid and clothianidin progressed normally through summer and autumn, but failed to recommence brood rearing in late winter and hence failed just as control colonies were emerging from successful overwintering [19]. These experimental colonies showed some features of CCD in that no dead adult bees were found in the colony. Dively *et al.* [28], however, reported that effects of chronic imidacloprid exposure via pollen on overwintering survival of colonies were only seen at the higher end of the possible range of expected field contamination.

Bee diseases interact with each other and with season to intensify impacts on colonies [29•]. Heavy infestation during winter of either the varroa mite or deformed wing virus spread by the mite has been shown to be highly predictive of colony failure [30]. Deformed wing virus and other opportunistic infections spread by varroa significantly weaken workers immune systems and energetic reserves, which could seriously impair worker performance [30]. Co-infections may act synergistically to weaken workers and increase transmission of diseases in the colony leading to colony failure with CCD-like symptoms [29•]. In this discussion I have focussed on stressors of workers, but it should be noted that the loss of the queen is also a significant stressor for a colony, and the demographic interruption as colonies replace a lost queen can significantly increase the risk of colony failure [31].

Death of the colony

A honey bee society usually contains within it autoregulatory mechanisms that operate to maintain the functions of the society against external stressors: fully understanding colony failure will require understanding how these social systems have failed. Much of the work in this area has involved modelling of colony demographic processes, and this approach has proved useful for framing and exploring hypotheses of how a colony might react to stress.

Normally a bee hive contains a balanced division of labour. Worker honey bees segregate tasks by age: young adults specialise on brood rearing roles and older adults defend the hive and forage [32–34]. This system enhances colony efficiency by delaying exposing workers to the highest risk tasks until after they have contributed to colony productivity [35]. It is maintained by pheromonally mediated social inhibition whereby old foragers in the hive inhibit younger bees from becoming foragers [36–38] and in this way the colony maintains an appropriate balance of forager and hive bees. If the hive loses its foragers, however, social inhibition is reduced and younger bees are recruited to the foraging force to replace them [36–38]. Precocious foraging by young bees is a common response of individual bees to stressors: individual or colony starvation [39,40], pollen deprivation [41,42], disease [11••,24,35,43–45], and even wax deprivation [46] will all cause young bees to begin foraging precociously. This is an adaptive response to an acute stressor since it rapidly replaces any losses of foragers and shifts the colony to increased resource accumulation, but the reaction of bees to stress by foraging could be problematic in the face of a chronic stressor.

New data has shown that precocious foragers are markedly less effective than bees that begin foraging at the typical age of more than two weeks old [10]. Precocious foragers survived less long as foragers, completed fewer foraging trips and were less far more likely to die during their first few flights outside the hive than bees that commenced foraging at a typical age [10].

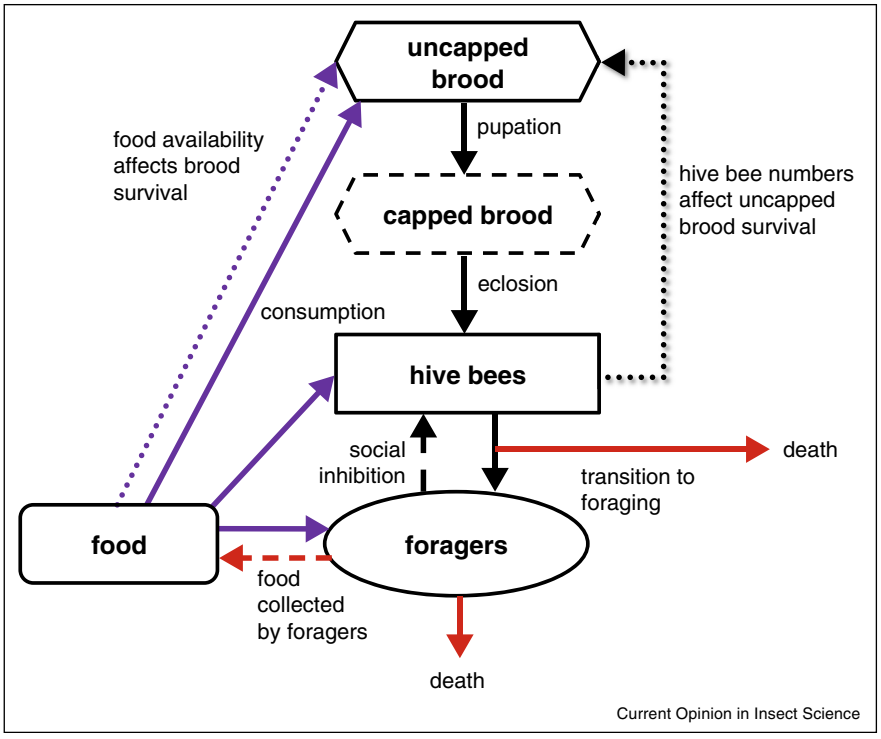
Perry *et al*. [10] constructed a simple compartment model of honey bee colony demography as a tool to explore how the reactions of individual bees to stressors (precocious foraging and poor foraging performance) might alter colony function (Figure 1) [18,47]. The model assumed that the age at which bees commenced foraging was regulated by social inhibition, and that foraging performance (in terms of food collection) and forager survival were both age-dependent and declined as bees began foraging at younger ages (Figure 1). The model was then used to examine the consequence of a colony suffering a chronic high rate of forager death, as might occur if foragers were impacted by disease or pesticide and becoming lost while foraging (Figure 2a–d).

In simulations if forager death rates were chronically raised above a threshold adult population decline was both rapid and markedly non-linear (Figure 2). Initially colonies buffered the consequences: populations stabilised for a period and brood rearing continued (Figure 2d). During this period, however, the foraging force became progressively younger and less effective until it could no longer sustain food levels in the colony, which triggered a very rapid terminal decline in the adult population. In the model the terminal phase saw a complete breakdown of division of labour with most bees becoming foragers and dying soon after, leaving just the queen, a few adult bees and abandoned brood in the colony.

It is notable that in this model colonies failed displaying several of the features of CCD [10]: an abrupt change from apparent health with successful brood rearing to total loss of the adult population, a decline in the average age of workers and in the ratio of nurse bees to brood in the colony, and colony failure leaving few adult bees but a queen and brood in the colony (Figure 2). If this model captures the demographics of a stressed bee colony then it may explain why colonies sometimes depopulate so rapidly and die.

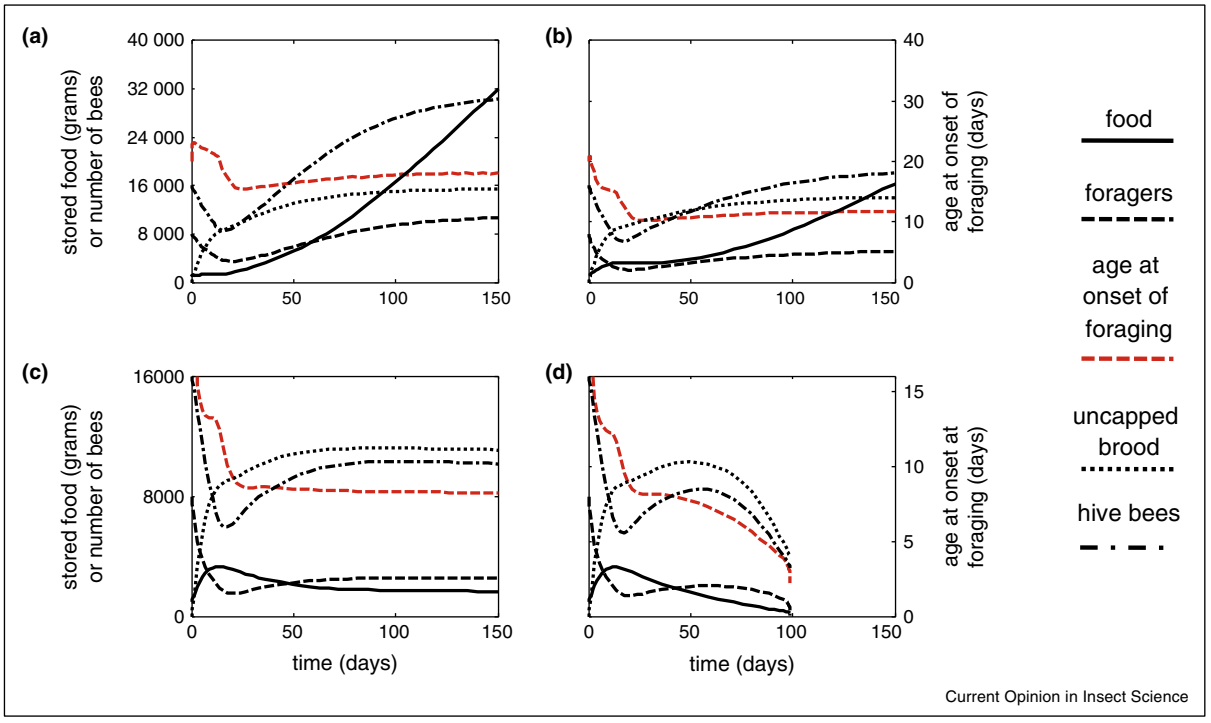
The models of Perry *et al*. [10] and Khoury *et al*. [18,47] considered how a colony might respond to chronically elevated forager losses. Betti *et al*. [48••] built on the Khoury model framework [18,47] to consider how a contagious bee disease might interact with the colony's demographic processes to damage colony function. In their model, highly transmittable diseases had a greater impact on colony function than diseases that were more lethal,

Figure 1



Schematic representation of key demographic processes operating in a honey bee colony considered as a compartment model by Perry *et al*. [10]. Workers develop as larvae, pupate as capped brood, emerge as adult hive bees and mature to foragers. Existing foragers inhibit hive bees from becoming foragers by social inhibition [36,56]. Blue lines represent consumption of food. Food also influences brood numbers indirectly since in periods of low food influx workers will cannibalise larvae (blue dotted line) [57]. 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) varied with the age at onset of foraging.*Source:* Figure adapted from Perry *et al*. [10] with permission.

Figure 2



Outputs of the model of honey bee demography shown in schematic in Figure 1 and described in full in [10]. Responses of a colony to chronic stress 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 increasing rates of forager bee mortality. In these plots death rate is expressed as the ratio between death rate of the simulated hive and a healthy hive (m_r). **(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)** $m_r = 2.0$ (twice the death rate of a healthy hive). For this death rate, the forager population reached zero at about $t = 99$ days. At about 3 weeks before collapse the colony in (d) had 8250 uncapped brood items, 6780 hive bees and 1780 foragers, suggesting a colony could decline from an apparently healthy size and strong brood production to zero foragers in less than three weeks. The mathematics are such that modelling could not continue beyond the point of zero foragers. Note that (a) and (b) are on a different vertical scale to (c) and (d). In the model chronically elevated forager death rates caused a shift in the proportions of forager bees, hive bees and brood in the colony and resulted in a younger forager population. When death rates were chronically maintained at more than twice that of a healthy hive the colony rapidly collapsed.
Source: Figure adapted from Perry *et al*. [10] with permission.

since the former persisted in colonies for longer and infected bees when younger [48••]. In their model colonies were most at risk of failure if infected before winter since the infected colonies were unable to effectively resume brood production in spring. It is often observed that the winter/spring transition is when colony failure is most likely to occur [2,5,49], and this may be a point of particular vulnerability for colonies since they are attempting rapid growth having all but exhausted their food reserves [50]. Much of the challenge in understanding colony failure is that the interactions of stressors on individual bees with colony dynamics and environmental factors such as season and resource availability are complex, dynamic and layered. It is not easy to frame simple hypotheses for how best to intervene to improve colony health, but here the new modelling approach of Becher *et al*. [51] which considers colony performance and impact of disease in a landscape context could be a very powerful tool.

Models of honey bee colonies are important because they help clarify thinking of the demographic processes we imagine to operate within colonies, and they propose hypotheses of how and why colonies might fail [51]. Testing these hypotheses requires long-term studies of the changes in brood, food, adult population and foraging performance as colonies develop and decline. Such studies are now possible thanks to new sensor technologies such as continuous weight monitoring [52•] and RFID tracking of bees [10,53–55]. A very promising future direction will be combing the capacity of these new technologies to generate high-quality field data with new approaches to modelling honey bee hive performance [51]. Modelling can help frame and explore hypotheses for how to improve colony performance and prevent colony failure. New experiments then enable testing of these hypotheses and the provision of new field data to improve models.

Conclusions

Honey bee colony failure rates have increased, and in some circumstances colonies collapse rapidly and completely. The ultimate explanation of both of these phenomena may simply be that far too many honey bee stressors (environmental, agrochemical, parasitic and pathogenic) have increased in frequency and intensity, and honey bee colonies are too often dealing with too many chronic stressors.

A honey bee colony many be particularly vulnerable to sublethal effects of diseases and pathogens because colony functions are damaged to the same degree whether a stressor kills a worker outright, or simply causes her to fail in her foraging effort. Honey bee colonies have internal demographic processes to buffer against forager losses by recruiting young bees to the foraging force. This may be an effective response to restore colony function in the face of acute stressors, but modelling suggests it may accelerate colony failure and population collapse in the face of chronic stressors. Experimental analyses of the demographic processes of the bee hive combined with modelling have shed some light on what may happen when colonies rapidly fail. Field studies to test predictions of these models are now urgently needed to identify the best interventions to stop the process.

Acknowledgements

This work was funded by a Hermon Slade Foundation research grant (no 11/7) to Andrew Barron.

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