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

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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.

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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].