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Research article

Single and dual parasitic mite infestations on the honey bee, *Apis mellifera* L.

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Summary. The onset of foraging, proportion of pollen collectors, and weight of pollen loads were compared in individual honey bees (*Apis mellifera*) infested by zero, one (*Acarapis woodi*, the honey bee tracheal mite, or *Varroa jacobsoni,* varroa), or both species of parasitic mites. Phoretic varroa host choice also was compared between bees with and without tracheal mites, and tracheal mite infestation of hosts was compared between bees parasitized or not by varroa during development. The proportion of pollen collectors was not significantly different between treatments, but bees parasitized by both mites had significantly smaller pollen loads than uninfested bees. Mean onset of foraging was earliest for bees parasitized by varroa during development, 15.9 days. Bees with tracheal mites began foraging latest, at 20.5 days, and foraging ages were intermediate in bees with no mites and both, 17.6 and 18.0 days respectively. Phoretic varroa were found equally on bees with and without tracheal mite infestations, but bees parasitized by varroa during development were almost twice as likely to have tracheal mite infestations as bees with no varroa parasitism, 63.9% and 35.5%, respectively. These results indicate that these two parasites can have a biological interaction at the level of individual bees that is detrimental to their host colonies.

Key words: Varroa jacobsoni, Acarapis woodi, colony state, foraging behavior, host selection.

Introduction

Honey bee (*Apis mellifera* L.) colonies are a rich and stable habitat for parasites. Host bees, honey, pollen and wax are present year-round, and bees regulate temperature, humidity and carbon dioxide levels within narrow limits (Winston, 1987). As a consequence, honey bee colonies are exploited by an array of parasites, pathogens and associated organisms (Bailey and Ball, 1991), which may be an important selective force on the evolution of colony structure and function (Gadagkar, 1992; Sherman et al., 1988; Shykoff and Schmid-Hempel, 1991a, b). Over 40 mite species are known to be associated with honey bee colonies (Eickwort, 1988), several of which are parasitic, including two that cause severe bee colony mortality and world-wide losses in the beekeeping industry (Bailey and Ball, 1991). The impact of these two mites, *Acarapis woodi* (Rennie) and *Varroa jacobsoni* Oudemans, on individual honey bees is the focus of this study.

Varroa and tracheal mites feed on honey bee hemolymph, depriving the bee of hemolymph and protein (Bailey and Ball 1991; De Jong et al., 1982; Kovac and Crailsheim, 1988). This effect is usually sub-lethal to the individual bee, but infestation by either mite species can kill a bee colony when not controlled. Although the direct mechanism of death is not known, both mites have been associated with higher levels of bacterial and viral pathogens in bee hemolymph, and it is suspected that mite infestation decreases adult longevity due to these pathogens (Allen and Ball, 1996). Overall, varroa mites are more virulent than tracheal mites, killing a colony within 1–2 years if left unchecked (Bailey and Ball, 1991). Tracheal mite infestations may last for many years without noticeable effects.

The presence of both mites in honey bee colonies is a critical management problem facing beekeepers world-wide, but also provides a fascinating opportunity to explore host-parasite interactions in a social insect. Of particular interest are questions involving how single or dual-species mite infestations might affect individual performance in areas that require integration in colony-level functions, such as foraging. For honey bees, the labor expended to gather nectar and pollen varies at both individual bee and colony levels, due to seasonal, nutritional, heritable, and colony-state factors (Schmid-Hempel et al., 1993). Several stress events, such as wax deprivation (Fergusson and Winston, 1988), depletion of nectar and pollen stores (Rinderer, 1982; Fewell and Winston, 1992) and worker loss (Winston and Fergusson, 1985) induce resource-gathering responses in honey bee colonies,

including increased number of foragers, accelerated task ontogeny (i.e., earlier onset of foraging), or greater effort by individual foragers (foragers carry larger loads or visit more flowers) (Schmid-Hempel et al., 1993). In addition to resource deprivation, stresses that cause precocious foraging include infection with diseases, such as *Nosema apis* (Wang and Moeller, 1970), Sacbrood virus (Bailey and Fernando, 1972), and hemolymph removal (Huang, pers. comm.). Infestation with either varroa or tracheal mites might induce similar responses, and dual parasitism could have additive or even synergistic effects on foraging behavior.

Dual mite infestations also provide an opportunity to determine whether the presence of one mite influences host choice or survival by the other. Phoretic varroa, those between reproductive bouts on pupae, and host seeking tracheal mites both prefer young adult *A. mellifera* hosts. Tracheal mites are more dependent on their initial host bee, as they spend their life cycle within tracheal tubes and only their offspring exit to choose a new host, in contrast to the highly mobile varroa which easily switch adult hosts. Tracheal mites use surface chemicals to identify young bee hosts (Phelan et al., 1991; Lee, 1963; Gary et al., 1989). Varroa mites probably use aliphatic alcohols and aldehydes found in honey bee cocoons to identify appropriate hosts for reproduction (Donze et al., 1998). Both mite species are known to *A. cerana* and *A. mellifera,* and parasitism may affect a bee's attractiveness as a new host, but it is not known whether parasitism by one species of mite affects host selection by the other species. Infestation by two species of parasitic mites allows examination of the interface of two parasites in a complex multiorganismal system, in addition to its implications for honey bee management. The effect of single species infestations have been studied for several years, but no studies have been conducted to examine the impact of single vs. dual infestations on individual honey bees and honey bee colonies, or the impact of one mite on the other. The main objectives of this project were 1) to compare the impact of tracheal and varroa mites, independently and together, on foraging parameters of individual bees, and 2) to investigate the sensitivity of each mite species to the presence of the other during host selection.

Materials and methods

Onset of foraging, pollen collection and pollen load weight

Experiments were conducted over two summers, 1996 and 1997, in Burnaby, B.C. Canada. Bees that had been fed on by varroa during the bee's pupal stage were obtained in two ways. In June 1996 (Round I) and July 1997 (Round II), a single female varroa mite was inserted into a newly sealed cell containing a larva. Cell cappings were cut open using a disposable scalpel, and varroa were lifted onto a fine paintbrush and allowed to crawl into the cell. The cell capping was tamped back down after insertion. Time of capping was monitored by mapping brood cells 12 h before varroa were to be inserted. Control bees were obtained by performing the same procedure without inserting a varroa mite. Varroa were collected by shaking young bees from a heavily infested colony in icing sugar (Boecking and Ritter, 1993), which causes the mites to drop off host bees. Varroa came from colonies maintained to have very high varroa populations, about 1 km away from the healthy colonies with low varroa infestation from which brood were taken. Cells were seeded in this manner on eight frames, each mapped using a plastic transparency tacked to the frame (Ifantidis, 1983). In August 1997 (Round III) brood frames containing larvae near capping stage were moved from a healthy colony to one heavily infested with varroa, and many cells were naturally infested. Control bees emerged from uninfested cells on the same frames.

Frames from both techniques were placed in a healthy colony with low mite levels to complete bee development. One to two days before emergence, the frames were placed into a hot room (32 °C) and emerging bees were captured and marked with colored and numbered bee tags glued to their thoraces, noting the number of mites that had fed on them during development. Each bee was placed immediately onto a frame of bees taken from the colony it would live its experimental life in, and the frames replaced into the colony every 24 h. This frame rotation ensured immediate bee exposure to tracheal mites.

Each tracheal mite-infested experimental colony had pre-monitored tracheal mite prevalence between 20–60%, determined by dissecting 30 bees collected from the inner cover (or frames nearby) in 70% ethanol and inspecting for tracheal mite presence or absence. All colonies were treated with fluvalinate (Apistan) prior to the experiment, which kills >99% of varroa mites but does not affect tracheal mites. In these colonies, tagged bees (either with or without varroa parasitism during development) were exposed to tracheal mites. Thus each marked bee was in one of four treatment groups: bees with 1) no mites, 2) varroa only, 3) tracheal mites only, or 4) varroa and tracheal mites.

To measure the onset of foraging, experimental bees were divided among six colonies prepared 1–2 weeks before the experiment began. In 1996 (Round I), three colonies were split from each of two large parent colonies. In 1997 (Rounds II and III), the experimental colonies had six different parent colonies. The parent colonies had 10–50% tracheal mite prevalence, which was monitored prior to and after the experiment (sampling method described above). Each five frame colony had 2–3 frames of bees, one frame of brood, one frame with pollen and two frames with honey. The colonies were given open mated sister queens (closest to *A. m. ligustica* in origin). After tagged bees were introduced, colony entrances were closed with a screen twice every day, once between 10:30–12:00, and again between 13:30–16:00, for either 15 min (Rounds I and II) or 30 min (Round III) each time. Any returning tagged bees were assumed to be first time foragers, and were collected and dissected to examine for tracheal mites. Foragers were classified as pollen foragers if they had pollen loads visible to the naked eye, otherwise they were classified as non-pollen foragers. In Rounds II and III, pollen loads were removed with forceps, and weighed (wet weight). Because there is an important genetic component of pollen collection (Page et al., 1995) and foraging onset (Giray and Robinson, 1994), bees from various genetic sources in Round I were assigned randomly to a colony by pooling bees and distributing them to each of the six colonies. In Rounds II and III, bees were mixed and distributed the same way, and the colony source of the bees was recorded to better assess the genetic effect.

Mite host choice

To compare tracheal mite infestation of hosts with and without varroa during development, varroa-parasitized and unparasitized bees were obtained using the second method described above, placing larvae near capping stage in a colony with high varroa levels. Six colonies with one frame of eggs and young larvae, one frame of honey, and a queen were established two weeks prior to the introduction of these bees. Each colony received three cups of a homogenous mixture of bees shaken from colonies with various tracheal mite infestation levels expected to average 40%. Paint-marked bees with and without varroa parasitism during development were introduced; adult varroa were not removed. Seven days later, these bees were collected and dissected to inspect for tracheal mites.

Phoretic varroa host preference was compared among same-age bees with and without tracheal mites. Frames of emerging bees were taken from colonies with low mite levels and placed in a 34 °C incubator

overnight. From these frames, 1200 newly emerged bees were paint-marked and divided randomly and evenly among the six colonies above. These colonies had tracheal mite prevalence between 40–56%. Varroa then were introduced to these colonies. Frames of emerging bees were taken from colonies heavily infested with varroa, placed in a 34 °C hot room, and newly emerged bees were inspected for varroa. Each colony received approximately 400 phoretic varroa, no brood were proper age for the varroa to enter and reproduce. When the marked cohort reached eight days of age, the bees were collected in two groups, with and without phoretic varroa, and dissected to compare tracheal mite infestation levels.

Statistical analysis for onset of foraging, pollen collection, pollen load weight and mite host choice were performed in SAS JMP IN 3.1.5. The onset of foraging was analyzed using a one way ANOVA and Duncan's Multiple Comparison Test. The genetic effect on the onset of foraging was analyzed using a two-way ANOVA. Pollen collection among mite treatments and mite host choice were compared using Chi-Square Tests, and pollen load weights were compared among mite treatments using a General Linear Model with Orthogonal Contrasts.

Results

Onset of foraging, pollen collection and pollen load weight

The mean age when workers began to forage was significantly different between mite treatments ($F = 12.17$, d.f. = 3, $p < 0.005$, $N = 1006$). Bees infested with varroa as pupae foraged significantly earlier (15.9 days of age) than bees with tracheal mites (20.5 days). The foraging age of bees with no mites (18.0 days) was less than bees with tracheal mites but not significantly different from bees with both species (Fig. 1). Foraging age decreased with the severity of parasitism by varroa mites during development but the relationship was very weak (Fig. 2). There also was a genetic effect on the onset of foraging ($F = 5.20$, d.f. = 11, $p = 0.0001$), with means ranging from 13.5–24.0 days with genetic source (Rounds II and III). Foraging began at significantly different ages in Rounds I–III (21.2, 18.6, and 15.1 days, respectively) ($F = 47.06$, d.f. = 2, $p < 0.0001$).

The proportion of bees collecting pollen was not significantly different among treatment groups: no mites 36.8%, tracheal mite 36.3%, varroa mite 32.3%, and both mites

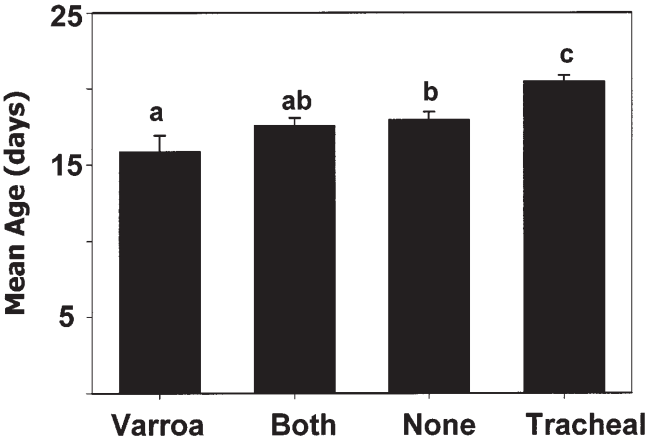

Figure 1. Effect of mite parasitism on mean age (± SE) of foraging onset. Letters indicate means that are significantly different ($p < 0.005$) using Duncan's Multiple Range test

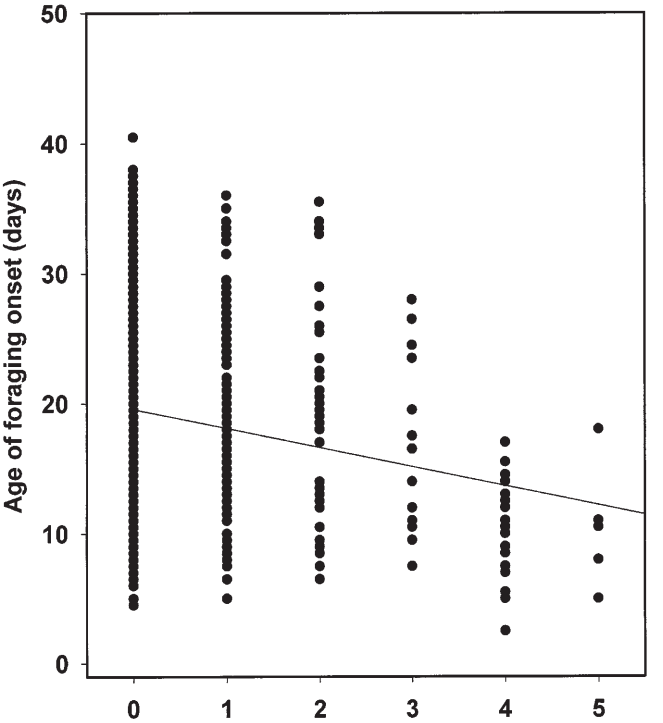

Figure 2. Relationship between age at foraging onset and number of varroa mites in the bee's cell during development. (Linear regression: $y = 19.55x - 1.47$, $p < 0.0001$, $r^2 = 0.0329$, $n = 991$)

30.7% ($X^2 = 2.793$, $p = 0.425$, d.f. = 3, $N = 1006$). Pollen collection did vary with the genetic source of the bees: range = 21.2 – 43.8% pollen foragers ($X^2 = 17.46$, $p = 0.0146$, d.f. = 7, $N = 8$). The percentage of returning marked bees with pollen loads was highest in June 1996 (38%, $N = 439$) lower in July 1997 (36%, $N = 335$) and was lowest in August 1997 (28%, $N = 247$) ($X^2 = 7.88$, $p = 0.0195$, d.f. = 2, $N = 3$).

Pollen load weight was significantly lower in dually infested bees than in uninfested bees (Fig. 3) ($F = 6.077$, d.f. = 1, $p = 0.0147$). Genetic source had a significant impact on pollen load weight ($F = 2.15$, d.f. = 8, $p = 0.041$), with the range of pollen load weights (mean ± SE) 4.6 ± 1.0 to 8.6 ± 0.9 mg.

Mite host choice

Tracheal mites infested bees with and without phoretic varroa at statistically indistinguishable levels (33.3%, $N = 144$ and 28.9%, $N = 204$, respectively) (Fig. 4A). Tracheal mites were found almost twice as often in bees that had been parasitized by varroa during development ($N = 108$, 63.9% with tracheal mites) than in bees without varroa during development ($N = 197$, 35.5% with tracheal mites) (Fig. 4B). In the foraging trials, bees with varroa parasitism were 76.9% infested with tracheal mites, significantly higher than in bees without varroa (64.2%) ($X^2 = 15.361$, $p < 0.001$, d.f. = 1, $N = 1006$), despite the fact that both groups of bees in Round I were 90% infested with tracheal mites. Excluding data for

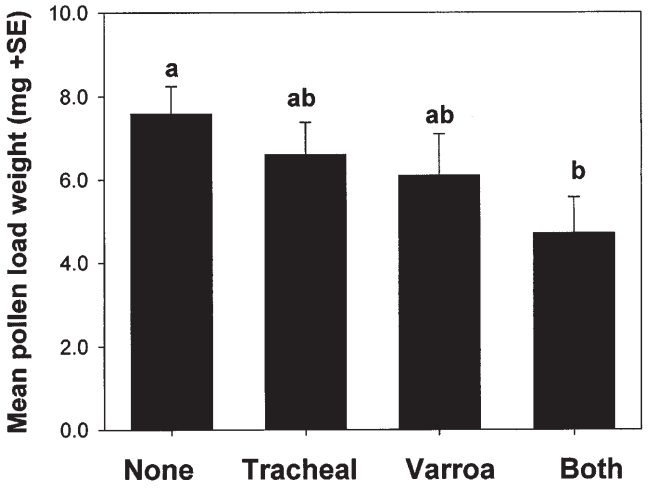

Figure 3. Weight of pollen loads (mean ± SE) with tracheal and varroa mites. Means with different letters are significantly different (GLM with orthogonal contrasts, $p < 0.05$)

Round I gave 64.8% tracheal mite infestation in bees parasitized by varroa and 46.8% tracheal mite infestation in bees with no varroa parasitism ($X^2 = 14.029$, $p < 0.001$, d.f. = 1, N = 570). Tracheal mite infestation did not vary significantly with genetic source of the bees ($X^2 = 10.88$, $p = 0.1438$, d.f. = 7, n = 556).

Discussion

The most surprising result of this study was that the two mite species alone had *opposite* effects on the ages at which bees began foraging, while bees infested with both mites showed intermediate foraging ages, equal to unparasitized bees. The decreased foraging age of bees infested by varroa as pupae was the more predictable result, since a generalized honey bee response to stress is to increase foraging effort and accelerate temporal caste ontogeny (Schmid-Hempel et al., 1993). These stress responses maximize immediate resource collection, but bees that forage sooner will die younger due to mortality factors such as predation, inclement weather, and physiological aging, so that the cost of accelerated ontogeny is higher than that of increased effort by individuals already foraging (Fergusson and Winston, 1988).

Varroa induces precocious foraging (Schneider, 1986) (Fig. 1), and this effect is amplified by mite reproduction and multiple infestation, since foraging begins even earlier (Fig. 2). Fergusson and Winston (1988) document a graded honey bee stress response, whereby lesser stresses cause foragers to increase their efforts, and greater stress causes precocious foraging. In the present study, varroa parasitism did cause accelerated ontogeny but bees parasitized by varroa were not more likely to collect pollen, nor did they have bigger pollen loads.

The differences we observed in foraging ages suggest that parasitism by varroa could be used to explore the interaction between physiological effects of stress on individuals and colony-level responses, especially involving temporal caste

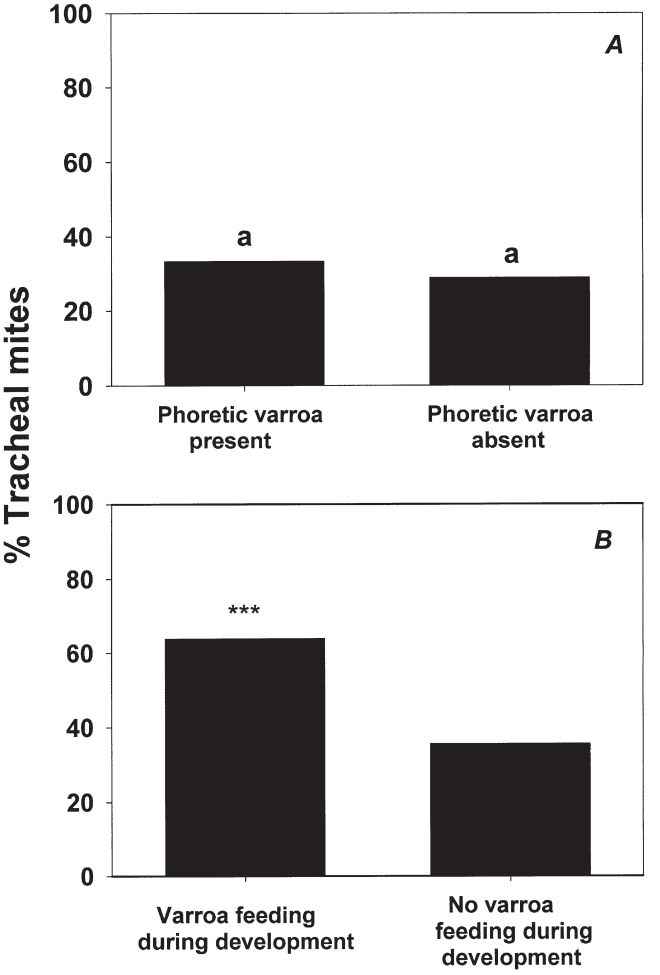

Figure 4. **A** Tracheal mite prevalence (%) in bees with and without phoretic varroa. Treatments are not significantly different ($X^2 = 0.772$, $p > 0.05$, d.f. = 1). **B** Tracheal mite prevalence (%) in bees with and without varroa parasitism during development. Treatments are significantly different ($X^2 = 22.613$, $p < 0.001$, d.f. = 1)

structure. Bees fed on by varroa during development emerge underweight (De Jong et al., 1982), and hemolymph volume and protein content are both substantially (15–50%) depleted (De Jong, 1990). Although adult bees can supplement protein levels by pollen feeding, protein deprivation during larval development could affect gland development, making these bees less suitable for in-hive tasks such as nursing. The effect we observed of earlier onset of foraging by bees that had been fed upon by varroa mites is consistent with the hypothesis that feeding by varroa during pupal development causes a more rapid increase in juvenile hormone (JH) titres in the adult bee. The relationship between JH titres and foraging has been well documented (Robinson, 1992). The effect of varroa on JH is presently unknown and should be investigated.

In contrast, the delayed onset of foraging in bees parasitized by tracheal mites is unusual because no stress on honey bee colonies is known to retard task ontogeny. Gary and Page (1989) found that tracheal mite infestation caused no significant differences in number of foraging trips, frequency of

foraging trips, round trip times, frequency of pollen collection, time between foraging trips, nectar loads and summer survival, although this study may not have captured the impact of tracheal mites due to low infestation levels. Foraging puts a bee at greater risk than in-hive tasks, and delayed foraging might provide a safer environment for the many tracheal mites living in each bee. Delayed foraging could reflect host manipulation, and there are many other examples of host manipulation in social insects (Schmid-Hempel, 1995). Tracheal mites also could alter individual fitness; impaired respiration or flight ability could make infested bees less suitable for foraging. The increased foraging age observed in tracheal mite-infested bees is also compatible with a possible effect of mite infestation on JH synthesis. Offsetting effects of infestation by the two mite species on JH production could explain the intermediate age at foraging onset for bees infested with both mites.

Their opposite impact on the onset of foraging may reflect differences in tracheal and varroa mite life history with regard to transmission. Varroa parasitism increases forager drift (Sakofski, 1990), mites can move quickly to a new host even if a drifting bee is not accepted in a foreign hive. In contrast, long-lived bees host large populations of tracheal mites (Pettis and Wilson, 1996) and this whole population of tracheal mites is jeopardized when that bee leaves the hive. Retarded ontogeny maximizes safety and longevity of tracheal mite host bees, and transmission is primarily in-hive, at night, between calm bees (Pettis et al., 1992). The opposite impact of these mites on the onset of foraging could reflect host manipulation.

Colony state variables can have an important role in individual foraging (Schmid-Hempel et al., 1993), and parent colony was a significant factor in Round I, and also in the experimental colonies. There is a genetic component to precocious foraging (Winston and Katz, 1982; Giray and Robinson, 1994) and pollen collection (Page et al., 1995), consistent with our results indicating that the genetic source of the bee was a significant factor in the onset of foraging, propensity to collect pollen and pollen load weight.

Pollen loads were smaller in dually infested bees than in uninfested bees, but this was not coupled with greater proportions of foragers due to infestation. Thus, dual infestation may hinder a colony's ability to collect pollen. Other studies suggest that varroa parasitism also reduces colony efficiency at using pollen to rear brood (Janmaat, 1998), an adverse impact that would not be observed as foraging behavior but could have a profound impact on colony productivity and success.

Host selection by phoretic varroa was not affected by tracheal mite presence, however tracheal mites were found almost twice as often in bees that had been fed on by varroa during development. This implies that varroa parasitism may alter a bee in a way that makes it a more frequent tracheal mite target, or in ways that would enhance tracheal mite survival once they locate a host bee. Varroa parasitism during the pupal stage might change the external chemical profile of an adult bee, alter the bee's internal chemistry, or change its ability to autogroom, an activity associated with lower tracheal mite levels (Pettis and Pankiw, 1994; Danka and Villa, 1998). Tracheal mites disperse nocturnally, likely because bees are in colonies and less active at night (Pettis et al., 1992). If varroa parasitism causes bees to further decrease activity, they could be more vulnerable to tracheal mite infestation. More research is needed to understand tracheal mite host selection, acceptance, and successful infestation to elucidate the mechanism underlying this result.

Although effect varies, each of these mites can be devastating to a honey bee colony. This study focused on individual bees to observe the interactive aspects of these parasites sharing a host. Colony-level studies measuring productivity and mortality with regard to dual infestation also indicate a colony-level synergistic effect in addition to the individual effects, with significantly higher colony mortality with dual compared to single species infestations. (Downey, in prep.).

Our results have important implications for understanding colony resiliency to parasitic mite stress and host choice by parasites. In general, parasite and pathogen pressure on social insects can affect the evolution of colony structure and maintenance of social organization. Examining the effects of parasitic mites on colony functions such as foraging can expose the underlying integrative response mechanisms of parasitised individuals *and* the resulting colony condition. As varroa and tracheal mites continue to share host bees and colonies, honey bees can be used to answer many interesting questions regarding parasite interactions and host sharing. The complexity of social insect behavior and the lack of information regarding these parasites leaves many opportunities for further study.

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