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Notes and comments

PARAVESTIBULAR HAIRS OF BRITISH BUCKFAST HONEY BEES DO NOT REGULATE GENETIC RESISTANCE TO *ACARAPIS WOODI*

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Some honey bees (*Apis mellifera*) have a genetically based resistance to infestation by the parasitic tracheal mite, *Acarapis woodi*. Resistant bees interrupt the migratory phase of the mite's life cycle (Danka & Villa, 1996), principally because individual workers effectively groom migrating mites off themselves (Danka & Villa, 1998; Pettis & Pankiw, 1998). Another potential resistance mechanism involves the hairs which surround the exoskeletal depressions, or vestibules, in which the prothoracic spiracles are located. Lee (1963) investigated whether the well known resistance of older bees to tracheal mite infestation is attributable to some aspect of these hairs, for example, to their hardening with age. Lee treated bees in three ways: (1), by removing some of the paravestibular hairs (referred to as 'vestibular guard hairs' by Lee); (2), by removing the mesothoracic legs to prevent grooming; and (3), by severing the muscle that closes the spiracle. He concluded that none of these treatments, except possibly leg removal (i.e. presumably a diminished ability to autogroom), resulted in more mites entering the spiracles and vestibules of old bees when mites were placed directly on the bees. Lee did not evaluate bee stocks that differed in susceptibility to mites (genetically based mite resistance had not yet been demonstrated at the time of Lee's research). We revisited the potential role of paravestibular hairs in regulating mite infestation, with an emphasis on possible differences between mite resistant and mite susceptible bee stocks.

On newly emerged (0–6 h old) adult bees, the left or right side was chosen randomly and on that side the hairs on the posterior edge of the pronotal lobe and on the episternum posterior to the lobe were trimmed with a small piece of razor blade to open a gap of c. 0.1–0.3 mm into the vestibule (as shown in Lee, 1963). During the trimming, bees were held in place on the end of a small tube with suction. The other side of each

TABLE 1. Tracheal mite prevalences (%, $\bar{x} \pm$ s.e.) in groups of resistant and susceptible stock bees. The treatment classes within each stock are: control (handled but with paravestibular hairs not removed) or treated (having some hairs on one side trimmed off). For treated bees, prevalences are further partitioned into the mite prevalences in the tracheae on the control side, on the treated side, or on both sides.

Bee stock	Control bees	Treated bees	Treated bees infested on		
			control side	treated side	both sides
Resistant	21.3 ± 6.2	24.2 ± 6.7	9.5 ± 5.0	10.7 ± 4.3	4.0 ± 3.6
Susceptible	43.3 ± 6.2	48.3 ± 6.7	15.5 ± 8.2	20.3 ± 11.0	12.5 ± 10.9

bee served as a control. Other bees without any hairs removed served as a second control group. Both resistant and susceptible stock bees were used. Resistant bees were derived from British Buckfast bees, and susceptible bees were taken mostly from US bees that had not been exposed to tracheal mites (see Danka & Villa, 1996, for more information regarding bee stocks). Treated and control bees were colour coded to colony source with a mark of enamel paint on the abdominal tergites. After treatment, treated and control bees of the two stocks were exposed simultaneously in inoculation colonies with 40% to 56% mite prevalence (i.e. 40–56% of worker bees were infested with tracheal mites). Marked bees were recovered after 4 days and each trachea was examined for mite infestation. Seven trials were conducted using bees from one resistant and one susceptible colony, with two different colonies used in each trial. Mite prevalences for each treatment class were calculated from samples of 23 to 72 bees per trial; in all, 330 ± 62 ($\bar{x} \pm$ s.d.) bees per each of the four treatment classes were examined. Treatment and bee stock effects were evaluated by analysis of variance (PROC MIXED of the SAS system; Littell *et al.* 1996). Two analyses were conducted; one compared infestations in treated and control bees, and the other compared infestations in treated and control sides of individual treated bees.

Treatment effects were insignificant when bees having hairs partially removed from one side were compared to control bees ($F = 1.54$; d.f. = 1,12; $P = 0.238$) (table 1). A bee stock effect was found: mite prevalence was less in resistant bees than in susceptible bees ($F = 11.67$; d.f. = 1,6; $P = 0.014$). Response to treatment was similar in the two bee stocks ($F = 0.11$; d.f. = 1,12; $P = 0.745$ for the stock by treatment interaction).

Within individual treated bees, mite prevalence was greater on the treated side than on the control side ($F = 8.73$; d.f. = 1,12; $P = 0.012$). Mite prevalence was less in treated resistant bees than in treated susceptible bees ($F = 9.26$; d.f. = 1,6; $P = 0.023$) (table 1). The response to treatment was similar in treated bees of the two stocks ($F = 3.06$; d.f. = 1,12; $P = 0.106$ for the stock by treatment interaction).

Contrary to Lee's (1963) finding, we found marginally increased tracheal mite prevalence after removing a portion of the paravestibular hairs from one side of young bees. This suggests that the hairs normally deter some tracheal mites, in addition to other contaminants, from entering the spiracles. However, we found no differential response to treatment in the two stocks we tested. If hairs accounted for the genetic resistance of British Buckfast bees, then removing some hairs should have caused a relatively larger prevalence increase in these bees than in susceptible bees; this was not the case. Thus, differences in the paravestibular hairs do not account for differential susceptibility to tracheal mites in the resistant and susceptible bee stocks we studied. Available evidence points to autogrooming by individual worker bees as the primary mechanism interfering with mite migration (Danka & Villa, 1998; Pettis & Pankiw, 1998).

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