Experimental & Applied Acarology, 20 (1996) 313–322

Influence of resistant honey bee hosts on the life history of the parasite *Acarapis woodi*

Robert G. Danka* and José D. Villa

US Department of Agriculture, Agricultural Research Service, Honey Bee Breeding, Genetics and Physiology Laboratory, 1157 Ben Hur Road, Baton Rouge, LA 70820, USA

ABSTRACT

Non-infested, young adult honey bees (*Apis mellifera* L.) of two stocks were exposed to tracheal mites (*Acarapis woodi* (Rennie)) in infested colonies to determine how divergent levels of susceptibility in host bees differentially affect components of the mite life history. Test bees were retrieved after exposure and dissected to determine whether resistance is founded on the reduced success of gravid female (foundress) mites to enter the host tracheae, on the suppressed reproduction by foundress mites once established in host tracheae or on both. Cohorts of 30–60 bees from each of ten resistant colonies and eight susceptible colonies were tested in eight trials (three to five colonies per stock per trial) having exposure durations of 4, 9 or 21 days. The principal results were that lower percentages of resistant bees than of susceptible bees routinely became infested by foundress mites, individual infested susceptible bees often had more foundress mites than individual infested resistant bees did and mite fecundity was similar in both host types. The infestation percentage results corresponded well with similar results from a prior field test of these stocks and, thus, suggest that the bioassay is useful for assessing honey bee resistance to *A. woodi*.

Key words: *Apis mellifera*, *Acarapis woodi*, resistance, honey bee, tracheal mite, bioassay.

INTRODUCTION

The honey bee tracheal mite (HBTM), *Acarapis woodi* (Rennie), is a widespread endoparasite that is associated with mortality and reduced productivity of colonies of honey bees, *Apis mellifera* L., in North America (Eischen *et al.*, 1989; Furgala *et al.*, 1989; Otis and Scott-Dupree, 1992). Resistance to the HBTM has recently been found in various stocks of managed honey bees; this may offer beekeepers an effective, economical and environmentally harmless alternative to acaricides for managing HBTM problems. Several characteristics of resistance can be summarized from a variety of studies. Resistant stocks have relatively small percentages of bees become infested (i.e. have a low parasite 'prevalence'; *sensu* Margolis *et al.*, 1982) when small groups are exposed to infested bees for short periods in cages or colonies (Gary and Page, 1987; Clark *et al.*, 1990; Page

* To whom correspondence should be addressed.

0168-8162 © 1996 Chapman & Hall

and Gary, 1990; Szabo *et al.*, 1991; de Guzman, 1994; Lin *et al.*, 1996) or when full size colonies are challenged by mites for longer durations (Milne *et al.*, 1991; de Guzman, 1994; Danka *et al.*, 1995; Lin *et al.*, 1996). Resistant bees also have been reported to have fewer mites per infested bee (i.e. have a lower parasite 'intensity', *sensu* Margolis *et al.*, 1982) (Szabo *et al.*, 1991; de Guzman, 1994; Danka *et al.*, 1995) and fewer mites on a per bee basis (i.e. have a lower parasite 'abundance', *sensu* Margolis *et al.*, 1982) (Lin *et al.*, 1966). There is inconclusive evidence of mite reproduction being reduced in resistant bees (Lin *et al.*, 1996).

These previous studies were conducted mainly to determine whether resistant stock exists; the mechanisms conferring resistance have not been explored. The factors involved can be clarified by considering components of the HBTM life history that regulate mite population dynamics. Mature female HBTMs exit the tracheae of older bees in which they developed and migrate into the tracheae of younger bees where they reproduce and adult female progeny of these 'foundress' female HBTMs start to appear in approximately 10–14 days (Bailey and Ball, 1991; Pettis and Wilson, 1996). Life history events regulating HBTM infestations can thus be separated into components of migration and components of reproduction. Success in migration, in reproduction or in both must differ between mites parasitizing resistant and susceptible bee hosts. The summary of previous research results suggests that migration rather than reproduction is suppressed in mites infesting resistant bee hosts. To test this hypothesis, we used two bee stocks, one resistant and one susceptible to HBTM infestation, to measure the comparative influence of host bee type on HBTM life history components.

MATERIALS AND METHODS

General methods and analyses

A resistant honey bee stock ('Buckfast') was imported to the USA from the UK in 1990. Colonies representing a mixture of the five imported lines of the stock showed reduced HBTM infestation during a 1 year field test (Danka *et al.*, 1995). We used seven colonies that performed well in the field (all of one maternal line) and three untested colonies derived from three original lines. A susceptible bee stock was represented by eight colonies propagated from Louisiana, USA, colonies that had HBTM prevalences of $>30\%$ after at least 1 year of exposure to HBTMs.

HBTMs were bioassayed by exposing non-infested test bees of each stock to mites in infested inoculation colonies, then retrieving test bees, dissecting the tracheae and counting newly infesting HBTM foundress mites and their immature progeny (Gary and Page, 1987). Brood combs from individual test colonies were held in cages inside incubators (dark, 35°C, 50–80% RH). Thirty to sixty non-infested, newly emerged bees (≤ 6 h old) were identified as to colony source by colour marking with a 1–2 mm dot of gloss enamel paint

(Testors Corp., Rockford, IL, USA) on abdominal tergites V–VI. Bees of each stock were handled separately with latex gloves (to prevent contamination of possible cuticular hydrocarbon cues used by HBTMs in host selection; Phelan *et al.*, 1991) and held separately in plastic cups having rims coated with petrolatum. Test bees of both stocks were placed in the broad nest areas of innoculation colonies in the late afternoon; maximal foundress migration occurs into young adult bees and occurs nocturnally (Pettis *et al.*, 1992). Inoculation colonies were populous field colonies that were unrelated to the test stocks. After a prescribed duration of exposure (4–21 days, depending on the experiment), test bees were retrieved into flasks which contained carbon dioxide and were held on crushed ice to immobilize the bees and mites. Bees were stored frozen until the prothoracic tracheae were dissected and adult female, adult male and immature HBTMs between the spiracle and first tracheal bifurcation were counted at 60× magnification. When infested bees had immatures but no females were seen (1.4% of 962 samples), a count of one female was assigned. The few males seen were classified as progeny. Data were collected from April 1993 to May 1994.

Four parameters were measured to assess the effects of different bee stocks on the HBTM life history: (1) prevalence, defined as the number of infested bees per number of bees in a sample from a test colony (reported here as a percentage), (2) foundress intensity, which is the number of foundress mites per infested bee, (3) mean fecundity, which is the number of eggs and larvae per number of foundress mites in a bee and (4) progeny abundance, which is the total number of HBTM progeny per bee in a sample from a test colony. The progeny abundance gives a relative measure of the future HBTM challenge to a colony coming from the initial generation of HBTMs produced within a cohort of test bees. The effect of bee stock on each of these parameters was assessed by analysis of variance (ANOVA). For the parameters foundress intensity and mean fecundity, measurements from individual bees were averaged to yield the colony level means used in the ANOVA. The responses for all parameters were analysed as least-squares means (SAS Institute, 1989) because of unbalanced cell sizes.

Experiment 1: five inoculation colonies with 9 day exposure

Test bees from five colonies of each stock were used for five trials in different inoculation colonies. The HBTM prevalences in the inoculation colonies at the time of the test bee introduction in the three main trials were 38, 42 and 64%. Two later trials used inoculation colonies with 6 and 66% prevalences and in which all sealed brood was transferred from the colony with 66% prevalence to the colony with 6% prevalence. This manipulation was done to increase further the relative HBTM challenge in the inoculation colony with 66% prevalence by increasing the probability of foundress mites encountering test bees and not newly emerged resident bees. The HBTM challenge was conversely decreased in the inoculation colony with 6% prevalence. Test bees were recovered after 9 days in each trial.

The prevalence, foundress intensity, mean fecundity and progeny abundance

were analysed as a split plot design for the inoculation colonies with 38, 42 and 64% prevalences. The effect of stock was tested as the whole plot (with the term colony within stock used as the error) and the inoculation colony was the subplot (with the interaction term colony within stock × inoculation colony used as the error). Stock means within inoculation colonies were separated using least-significant differences calculated with weighted error terms based on the whole plot and subplot errors (Cochran and Cox, 1957). The stock effects were analysed independently in the inoculation colonies with 6 and 66% prevalences because these colonies were established with exaggerated HBTM challenges.

Experiment 2: one inoculation colony with 4 and 9 day exposures
The number of foundress mites in the prothoracic tracheae starts to decline when adult bees are 4 days old (Gary *et al.*, 1989). Interpretation of HBTM reproduction patterns would be problematic if the magnitude of decline differed in bees of different stocks. We tested for the possibility of such a stock effect. Test bees from three resistant and four susceptible colonies were introduced on 16 and 21 June 1993 and recovered on 25 June from a single inoculation colony having HBTM prevalences measured at 44 and 41% on the first and second days of introduction, respectively. The four HBTM life history parameters were evaluated as a slit plot design using an approach similar to that described for experiment 1. The stock was the whole plot and the time of exposure (4 or 9 days) was the subplot.

Experiment 3: two inoculation colonies with 3 week exposure
This experiment tested whether the factors responsible for resistance to HBTM infestation manifested at 9 days are demonstrated over a longer period. We examined the HBTM life history parameters 3 weeks after the introduction of test bees because the HBTM intensity is maximal at approximately this time (Pettis and Wilson, 1996). In two trials, test bees from five colonies of each bee type were exposed in inoculation colonies having 42% (in spring 1993) and 70% (in autumn 1993) prevalences. Test bees were recovered after 21 days. One to two generations of mites and their offspring existed and the emigration of females from infested bees probably occurred. The prevalence and total HBTM abundance (i.e. foundress mites and all subsequent progeny per bee in the sample) were analysed separately for each trial. The foundress intensity and mean fecundity were not able to be estimated owing to the overlap of generations of mites.

Experiment 4: effect of colour marks
A control experiment tested whether the colour used for marking affected any of the HBTM life history parameters. On three occasions, emerging brood was obtained from inoculation colonies (28, 42 and 82% prevalences), ten subsets of bees each were marked with one of the ten different colours and the test bees were returned to their natal colony for 9 days. For each of the four HBTM life

history parameters, the effect of the colour mark was tested by ANOVA; inoculation colonies (which were the sources of the test bees) were considered as blocks in the analyses.

RESULTS

Experiment 1

The prevalences were lower in resistant bees than in susceptible bees when the test bees were exposed in inoculation colonies having prevalences of 38–64% (Tables 1 and 2). Resistant bees had mean prevalences less than those of the inoculation colonies and susceptible bees had mean prevalences greater than those of the inoculation colonies in these three trials. The mean prevalences in the test bees increased when the inoculation colony prevalences were greater. Resistant bees had lower foundress intensities than susceptible bees did, but this trend was not consistently strong. The mean foundress intensities increased when the inoculation colony prevalences were greater. The mean fecundity was not influenced by the bee stock or by the prevalence in the inoculation colony; on average, each foundress mite produced four to five progeny during the 9 day test. The progeny abundances were approximately two to three times greater in susceptible bees than in resistant bees.

The trends observed in the inoculation colonies with 38–64% prevalence were not found in the inoculation colonies with 6 and 66% prevalences and with artificially created high and low, respectively, ratios of non-infested young bees to infested bees. Under these more extreme conditions, the HBTM life history parameters were similar in resistant and susceptible bees.

Experiment 2

The principal finding was that the means of each HBTM life history parameter changed between days 4 and 9 similarly for mites in the test bees of both stocks; there were no statistical interactions of the effects of stock and day for any parameter (Tables 1 and 2). Both the prevalences and foundress intensities decreased after day 4, thus corroborating earlier findings by Gary *et al.* (1989). The bee stock effects included resistant bees overall having significantly reduced prevalences and progeny abundances and numerically lower foundress intensities. As in experiment 1, the mean fecundity did not differ between the stocks.

Experiment 3

The bee stock effects on HBTM life history during the two 3 week exposure tests were less distinct than those seen in the shorter tests. In an autumn trial using an inoculation colony with 70% prevalence, the total HBTM abundances were less in resistant bees than in susceptible bees and the stock effects on the HBTM prevalences approached significance ($P = 0.064$) (Tables 1 and 2). The bee stock

TABLE 1

Life history parameters (least-squares means ± standard errors) for honey bee tracheal mites infesting bee hosts of two stocks that differed in susceptibility to infestation

	Bee stock	Prevalence (%)	Foundress intensity	Mean fecundity	Progeny abundance
Experiment 1 (9 days)					
Inoculation colony at 6% prevalence	R	5 ± 2	1.28 ± 0.46	5.08 ± 0.82	0.30 ± 0.14
	S	6 ± 2	1.50 ± 0.40	3.13 ± 0.71	0.26 ± 0.14
Inoculation colony at 38% prevalence	R	16 ± 4**[a]	1.14 ± 0.12	4.24 ± 0.36	0.81 ± 0.50*
	S	41 ± 4	1.31 ± 0.12	4.84 ± 0.36	2.71 ± 0.50
Inoculation colony at 42% prevalence	R	34 ± 4**	1.13 ± 0.12*	4.24 ± 0.36	1.70 ± 0.50**
	S	55 ± 4	1.54 ± 0.12	4.81 ± 0.36	4.07 ± 0.50
Inoculation colony at 64% prevalence	R	55 ± 4*	1.76 ± 0.12**	4.71 ± 0.36	4.55 ± 0.50***
	S	73 ± 4	2.30 ± 0.12	4.48 ± 0.36	8.23 ± 0.50
Inoculation colony at 66% prevalence	R	75 ± 3	2.69 ± 0.15	4.45 ± 0.20	9.42 ± 0.87
	S	85 ± 3	2.66 ± 0.15	3.87 ± 0.20	9.35 ± 0.87
Experiment 2					
4 days	R	22 ± 6	1.19 ± 0.30	1.74 ± 0.41	0.44 ± 0.23
	S	32 ± 5	1.49 ± 0.21	1.38 ± 0.29	0.68 ± 0.20
9 days	R	4 ± 6	1.00 ± 0.30	4.17 ± 0.41	0.17 ± 0.23
	S	19 ± 5	1.28 ± 0.21	4.53 ± 0.29	0.97 ± 0.20
Experiment 3 (3 weeks)					
Inoculation colony at 42% prevalence	R	33 ± 6	NA	NA	9.14 ± 2.48[b]
	S	42 ± 6			9.65 ± 2.48
Inoculation colony at 70% prevalence	R	35 ± 5			3.77 ± 0.88*
	S	52 ± 5	NA	NA	7.79 ± 0.88

R, resistant bee hosts; S, susceptible bee hosts; NA, the parameter is not applicable for this experiment.

[a] Differences between resistant and susceptible bees are shown only in cells of experiments in which the effect of host bee type was significant at $p \leq 0.05$ according to analysis of variance. Magnitudes of differences (determined by pairwise t-tests) between two means in a cell are indicated as $***P \leq 0.001$, $**0.001 < P \leq 0.01$ and $*0.01 < P \leq 0.05$.

[b] For experiment 3, this parameter includes all mites (foundress mites and all subsequent progeny of all generations) per bee sampled.

effects were weaker during a late spring trial using an inoculation colony with 42% prevalence.

Experiment 4

None of the four HBTM life history parameters was affected ($P \geq 0.378$) by the colour used in marking the test bees (Table 2).

DISCUSSION

Resistance to the HBTM in selected honey bees was based solely on the comparatively reduced migration of adult female HBTMs into the tracheae of

TABLE 2

Results of analysis of variance of effects influencing parameters of honey bee tracheal mite infestation in two honey bee stocks

	Variance source	Prevalence *F*, df, *P*	Foundress intensity *F*, df, *P*	Mean fecundity *F*, df, *P*	Progeny abundance *F*, df, *P*
Experiment 1					
Inoculation colonies at 38, 42 and 64% prevalences	Stock	21.32, 1,8, 0.002	8.92, 1,8, 0.017	0.84, 1,8, 0.386	30.70, 1,8, <0.001
	Inoculation colony	44.90, 2,16, <0.001	26.15, 2,16, <0.001	0.02, 2,16, 0.977	45.78, 2,16, <0.001
	Stock × inoculation colony	0.40, 2,16, 0.678	1.26, 2,16, 0.310	0.88, 2,16, 0.432	1.66, 2,16, 0.220
Inoculation colony at 6% prevalence	Stock	0.07, 1,8, 0.803	0.14, 1,5, 0.728	3.29, 1,5, 0.130	0.04, 1,8, 0.840
Inoculation colony at 66% prevalence	Stock	3.89, 1,8, 0.084	0.01, 1,8, 0.915	4.00, 1,8, 0.081	<0.01, 1,8, 0.960
Experiment 2	Stock	10.20, 1,5, 0.024	2.92, 1,4, 0.163	<0.01, 1,4, 0.998	12.63, 1,5, 0.016
	Day	7.74, 1,5, 0.039	0.59, 1,4, 0.486	60.57, 1,4 0.002	<0.01, 1,5, 0.986
	Stock × day	0.21, 1,5, 0.663	<0.01, 1,4, 0.962	1.04, 1,4, 0.366	1.76, 1,5, 0.242
Experiment 3					
Inoculation colony at 42% prevalence	Stock	1.32, 1,8, 0.284	NA	NA	0.02, 1,8 0.889
Inoculation colony at 70% prevalence	Stock	5.14, 1,6, 0.064	NA	NA	10.43, 1,6, 0.018
Experiment 4	Colour	1.15, 9,18, 0.378	0.68, 9,17, 0.714	0.69, 9,17, 0.713	0.39, 9,18, 0.925

NA, this variance source is not applicable for this parameter.

new bee hosts. The HBTM reproduction was similar in resistant and susceptible bees and, thus, did not contribute to the differential susceptibility to the parasite. When exposed for 9 days to moderate HBTM challenges (i.e. of approximately 40–60% HBTM prevalence in unmanipulated inoculation colonies), cohorts of resistant bees had lesser prevalences and susceptible bees had greater prevalences than the prevalences of the bees to which they were exposed. Measures of progeny abundance or total mite abundance usually indicated a 2–3-fold greater imminent future threat to susceptible stock colonies than to resistant stock colonies. These simple infestation trends could advance differential infestation in these two bee stocks over longer periods; such differences occurred during a 1 year field test of these stocks using standard colonies (Danka *et al.*, 1995). The lack of consistently strong differences in the 3-week trials reported here suggests that additional unmeasured factors (possibly including seasonal effects) affect HBTM populations in normal colonies.

The HBTM reproduction was not reduced in resistant host bees. The findings of Lin *et al.* (1996), who similarly tested resistant Buckfast bees from Great Britain, generally agreed with this result but included an observation of reduced mite reproduction in one of three trials. The slightly different results of these studies may stem from variance in the bee stocks, notably from different susceptible stocks being used.

The comparative prevalence findings from the short-term bioassay corresponded well with those obtained in an earlier field test of these stocks (Danka *et al.*, 1995). This congruency suggests that the bioassay is useful as a tool for screening for resistance in bee stocks. Lin *et al.* (1996) concluded likewise when the mite 'abundance' (number of mites per total number of bees in a sample) was measured. Note, however, that neither the differences in the susceptibilities of the bee stocks to HBTM infestation nor the ability of the short-term test to demonstrate those differences is invariable. Several cautions about this bioassay are thus warranted. First, the resistance identified in this bioassay is relative, not absolute, so the outcome of any particular comparison should be considered valid only for the specific bees studied. Evaluations of HBTM life history responses in other resistant and susceptible bee stocks are clearly warranted because a variety of resistance mechanisms may exist. Second, the prevalence differences between the stocks did not occur in this or other studies by us (unpublished) when the HBTM prevalence in the inoculation colonies was too high or too low; a great many or very few test bees of both stocks became infested under these respective conditions. Inoculation colonies having HBTM prevalences near 50% give the most consistent results. Third, the population sizes and demographics of both the resident bees and the HBTMs in the inoculation colonies vary, are difficult to standardize and probably affect the bioassay results even when the HBTM prevalences are in the recommended range. Trials therefore should be replicated before inferences about resistance are drawn. Finally, the bioassay cannot detect potential mechanisms of resistance which are expressed only at the colony level (e.g. allogrooming or reduced contact between infested bees and young bees).

In conclusion, resistance to HBTM infestation in the stock of honey bees we studied has its basis in interfering with the successful migration of foundress mites from natal host bees into the tracheae of new young host bees. This information should serve to guide future investigations into the specific mechanisms governing HBTM resistance in these bees. The results suggest that new studies should focus on areas such as bee anatomy, behaviour and cuticular chemistry that potentially control interhost movement of the parasite.

ACKNOWLEDGEMENTS

We thank Brad Hildago for assistance in processing samples and Steven Buco for guidance on statistical analyses. This research was completed in cooperation with the Louisiana State Agricultural Experiment Station.

REFERENCES

Bailey, L. and Ball, B.V. 1991. Honey Bee Pathology, 2nd edn. Academic Press, London.

Clark, K.J., Huxter, E., Gates, N.J. and Szabo, T.I. 1990. Screening breeder honey bee stock for resistance to tracheal mites. Am. Bee J. 130: 800.

Cochran, W.G. and Cox, G.M. 1957. Experimental Designs, 2nd edn. John Wiley and Sons, New York.

Danka, R.G., Villa, J.D., Rinderer, T.E. and Delatte, G.T. 1995. Field test of resistance to *Acarapis woodi* (Acari: Tarsonemidae) infestation and of colony production by four stocks of honey bees (Hymenoptera: Apidae). J. Econ. Entomol. 88: 584–591.

de Guzman, L.I. 1994. Tolerance potential and defense mechanisms of honey bees (*Apis mellifera* L.) to *Varroa jacobsoni* Oud. (Acari: Varroidae) and *Acarapis* species (Acari: Tarsonemidae). PhD dissertation, Louisiana State University, USA.

Eischen, F.A., Cardoso-Tamez, D., Wilson, W.T. and Dietz, A. 1989. Honey production of honey bee colonies infested with *Acarapis woodi* (Rennie). Apidologie 20: 1–8.

Furgala, B., Duff, S., Aboulfaraj, S., Ragsdale, D. and Hyser, R. 1989. Some effects of the honey bee tracheal mite (*Acarapis woodi* Rennie) on non-migratory, wintering honey bee (*Apis mellifera* L.) colonies in east central Minnesota. Am. Bee J. 129: 195–197.

Gary, N.E. and Page, R.E., Jr 1987. Phenotypic variation in susceptibility of honey bees, *Apis mellifera*, to infestation by tracheal mites, *Acarapis woodi*. Exp. Appl. Acarol. 3: 291–305.

Gary, N.E., Page, R.E., Jr and Lorenzen, K. 1989. Effect of age of worker honey bees (*Apis mellifera*) on tracheal mite (*Acarapis woodi*) infestation. Exp. Appl. Acarol. 7: 153–160.

Lin, H., Otis, G.W. and Scott-Dupree, C. 1996. Comparative resistance in Buckfast and Canadian stocks of honey bees (*Apis mellifera* L.) to infestation by honey bee tracheal mites (*Acarapis woodi* (Rennie)). Exp. Appl. Acarol. 20: 87–101.

Margolis, L., Esch, G.W., Holmes, J.C., Kuris, A.M. and Schad, G.A. 1982. The use of ecological terms in parasitology (Report of an *ad hoc* committee of the American Society of Parasitologists). J. Parasitol. 68: 131–133.

Milne, C.P., Otis, G.W., Eischen, F.A. and Dormaier, J.M. 1991. A comparison of tracheal mite resistance in two commercially available stocks of honey bees. Am. Bee J. 131: 713–718.

Otis, G.W. and Scott-Dupree, C.D. 1992. Effects of *Acarapis woodi* on overwintered colonies of honey bees (Hymenoptera: Apidae) in New York. J. Econ. Entomol. 85: 40–46.

Page, R.E., Jr and Gary, N.E. 1990. Genotypic variation in susceptibility of honey bees (*Apis mellifera*) to infestation by tracheal mites (*Acarapis woodi*). Exp. Appl. Aracol. 8: 275–283.

Pettis, J.S. and Wilson, W.T. 1996. Life history of the honey bee tracheal mite (Acari: Tarsonemidae). Ann. Entomol. Soc. Am. in press.

Pettis, J.S., Wilson, W.T. and Eischen, F.A. 1992. Nocturnal dispersal by female *Acarapis woodi* in honey bee (*Apis mellifera*) colonies. Exp. Appl. Acarol. 15: 99–108.

Phelan, P.L., Smith, A.W. and Needham, G.R. 1991. Mediation of host selection by cuticular hydrocarbons in the honeybee tracheal mite *Acarapis woodi* (Rennie). J. Chem. Ecol. 17: 463–473.

SAS Institute. 1989. SAS/STAT User's Guide, Version 6, 4th edn. Vol. 2. SAS Institute, Cary, NC.

Szabo, T.I., Lefkovitch, L.P. and Clark, K.J. 1991. Comparative resistance of honey bees from a closed population to infestation by tracheal mites. Am. Bee J. 131: 643–645.