Field evaluation of nine families of honey bees for resistance to tracheal mites

Dennis van Engelsdorp,[1] Gard W Otis[2]

Department of Environmental Biology, University of Guelph, Guelph, Ontario, Canada N1G 2W1

The Canadian Entomologist **133**: 793 – 803 (2001)

Abstract—We evaluated the resistance to tracheal mites, *Acarapis woodi* (Rennie) (Acari: Tarsonemidae), of colonies of honey bees, *Apis mellifera* L. (Hymenoptera: Apidae), headed by daughters of three queens from each of three honey bee stocks: (*i*) British Columbia "mite-resistant" stock, (*ii*) Buckfast "mite-resistant" stock, and (*iii*) Canadian unselected stock. Colonies of all nine families were distributed among four apiaries; half of the colonies in each apiary were treated with formic acid to attempt to control tracheal mites. The study documented significant differences in resistance to tracheal mites among the families of bees, even within each of the three stocks. After the first 4 months of study (by November 1993), differences in mite infestations had developed among the nine families. Formic acid treatments had either short-lived effectiveness (1993) or no effect (1994) on tracheal mite infestations, thereby eliminating the opportunity to evaluate colony performance in the absence of mites. Mite infestations varied significantly among apiary sites. This study highlights the value of evaluating sets of colonies headed by sister queens when identifying mite-resistant stock for breeding purposes.

van Engelsdorp D, Otis GW. 2001. Évaluation de la résistance aux acariens des trachées en nature chez neuf familles d'abeilles. *The Canadian Entomologist* **133** : 793–803.

Résumé—Nous avons évalué la résistance aux acariens des trachées, *Acarapis woodi* (Acari : Tarsonemidae), chez des colonies d'abeilles domestiques, *Apis mellifera* (Hymenoptera : Apidae), dont les reines sont les filles de trois reines provenant de trois stocks connus (*i*) le stock de Colombie-Britannique, « résistant aux acariens » (*ii*) le stock Buckfast, « résistant aux acariens » et (*iii*) un stock canadien non sélectionné. Les colonies des neuf familles étaient réparties dans quatre ruchers: dans chaque rucher, la moitié des colonies a été traitée à l'acide formique dans une tentative de lutte contre aux acariens des trachées. Nous avons enregistré des différences significatives dans la résistance aux acariens des trachées d'une famille d'abeille à une autre, même au sein d'un même stock. Après les 4 premiers mois de l'étude (novembre 1993), il y avait déjà des différences dans l'importance des infestations chez les neuf familles. Les traitements à l'acide formique ont eu des effets de courte durée (1993) ou n'ont pas eu d'effet (1994) sur les infestations d'acariens, éliminant par le fait même la possibilité d'évaluer la performance d'une colonie en l'absence d'acariens. Les infestations variaient significativement d'un rucher à l'autre. Cette étude met en évidence l'intérêt d'évaluer des séries de colonies dont les reines sont soeurs lorsqu'on cherche à identifier les stocks résistants aux acariens pour des fins d'élevage.

[Traduit par la Rédaction]

[1] Present address: Department of Entomology, Comstock Hall, Cornell University, Ithaca, New York, United States 14853.
[2] Author to whom all correspondence should be addressed (E-mail: gotis@evb.uoguelph.ca).

Introduction

The tracheal mite, *Acarapis woodi* (Rennie) (Acari: Tarsonemidae), is an obligate parasite of honey bees, *Apis mellifera* L. (Hymenoptera: Apidae). It can have detrimental effects on honey production (Guzman-Novoa and Zozaya-Rubio 1984; Eischen *et al.* 1989), survival during winter (Bailey 1958, 1961; Bailey and Lee 1959; Eischen 1987; Furgala *et al.* 1989, Komeili and Ambrose 1990; Otis and Scott-Dupree 1992; Tomasko *et al.* 1993), and spring growth of colony populations (Otis and Scott-Dupree 1992). Bailey and Ball (1991), Pettis and Wilson (1996), Sammataro and Needham (1996), and Sammataro *et al.* (2000) have recently summarized the biology of the honey bee tracheal mite.

Because of the negative effects of mites on colonies and the costs of chemical treatments to control their populations, research efforts have focused on the development and establishment of mite-resistant stocks of bees. Several strategies have been employed to obtain mite-resistant bee stocks. Loper *et al.* (1992) and Danka *et al.* (1995) reared replacement queens from colonies that exhibited no obvious effects from mites in infested apiaries. Numerous studies have evaluated potentially mite-resistant stock imported from Europe where tracheal mites are known to have existed for decades (British stock: Gary *et al.* 1990; Buckfast stock from England and Denmark: Milne *et al.* 1991, Lin *et al.* 1996, and Nasr *et al.* 2001; and Carniolan stock from Yugoslavia: Guzman *et al.* 1998). Additionally, some researchers have evaluated and selected North American bees that have not undergone any prior selection for mite resistance (Gary and Page 1987; Szabo *et al.* 1991; Huxter and Clark 1991, 1992; Nasr *et al.* 2001).

Despite these studies, only two methods have been used to identify more mite-resistant colonies. Gary and Page (1987) developed a bioassay that involves pooling individually marked young worker bees from many colonies under evaluation in the common environment of another colony that is heavily infested with tracheal mites. Alternatively, one can follow changes in mite populations in hives over several months. Lin *et al.* (1996) and Nasr *et al.* (2001) obtained significant positive correlations between the results of the Gary and Page (1987) bioassay (henceforth referred to as the Gary/Page bioassay) and long-term colony evaluations.

Within Canada, several bee breeding efforts have sought to identify mite-resistant bees. Specifically, the Gary/Page bioassay has been used in British Columbia to enhance tracheal mite resistance. In Ontario, Buckfast bees reported to be resistant (Adam 1987) have been imported by the University of Guelph (Nasr *et al.* 1991) and a breeding program established with three cooperating beekeepers. These ongoing breeding projects presented us with the opportunity to compare temporal changes in mite infestations between these "mite-resistant" stocks and other colonies with no prior history of exposure to tracheal mite infestations. From nine breeder queens (three of each stock), daughter queens were reared and established in colonies in four apiaries. This permitted us to quantify the variability in expression of mite resistance within and among stocks. Furthermore, we attempted to evaluate the performance (*i.e.*, honey production, winter survival) of the nine families both in the presence and absence of mites. To facilitate this goal half the colonies were treated with formic acid. This experimental design also allowed for the assessment of the effects of apiary location on mite populations.

Materials and methods

Nine families of honey bees were selected for study: three families (BC-1, BC-2, BC-3) represented the British Columbia stock selected for mite resistance; three families (BF-4, BF-5, BF-6) represented the Buckfast stock purported to be mite resistant (Adam 1987); and three families (CD-7, CD-8, CD-9) represented the Canadian stock

unselected for tracheal mite resistance. Each family was represented initially by approximately 10 sister queens.

The queens representing the BC-1, BC-2, BC-3, and CD-7 families were reared by Kettle Creek Apiaries, British Colombia (49°03′N, 119°00′W). The British Columbia mite-resistant stock was selected using the Page/Gary bioassay for two generations. Daughter queens of the three selected mite-resistant breeder queens were naturally mated to drones from a group of British Colombia mite-resistant colonies in an isolated mountain valley. The CD-7 queens were open-mated at an apiary with no selected mite-resistant stock nearby.

The queens representing the BF-4, BF-5, BF-6, CD-8, and CD-9 families were reared at the University of Guelph, Ontario (43°31′40′′N, 80°13′39′′W). The Buckfast mite-resistant stock were produced by rearing daughter queens from three Buckfast breeder queens imported in 1992 from Brandstrup Apiaries, Denmark, and naturally mating them to a Buckfast drone line (KB190) in isolation on Thorah Island, Lake Simcoe, Ontario (44°26′N, 79°14′W). The absence of honey bees on blooming apple trees and the failure of five virgin queens to mate during a 3-week period when suitable mating conditions existed confirmed the absence of feral honey bee colonies prior to the establishment of the drone source colonies on Thorah Island. Queens representing the CD-8 and CD-9 unselected families were open-mated at the Eramosa Research Station, University of Guelph (43°38′N, 80°13′W); no mite-resistant stock was maintained within 6 km of the station at that time.

On 13 April 1993, approximately 130 colonies in two nearby apiaries near Vineland, Niagara Peninsula, Ontario (43°08′N, 79°23′W), were sampled to estimate tracheal mite infestations (25 bees/hive). We selected the 100 colonies with the highest mite infestations for further study. Frames of sealed brood were exchanged among colonies to equalize colony populations. Locations of lightly and heavily infested hives were exchanged to reduce differences in mite infestation among the colonies. The colonies were randomly assigned and moved to four apiaries 3–5 km from each other, south of Smithville, Ontario (43°06′N, 79°33′W), on 8 May 1993. Equalization of worker populations continued every 2 weeks until mid-July. Prestudy mite prevalence (percentage of bees in a sample infested by mites) was determined (Shimanuki and Cantwell 1978; Delfinado-Baker 1984) from 50 bees/colony collected from the inner covers of each hive on 12 July 1993.

On 14 August 1993, the queens of the study colonies were removed and the marked experimental queens were introduced. Approximately 2 weeks later, the colonies in which the queens were not accepted (*e.g.*, no eggs or larvae present) were removed from the study. In early September, colonies headed by queens that lacked identifying markings were also removed. In total, 30 colonies were eliminated from the study, leaving 4–8 colonies/family. On 24 September 1993, one half (n = 35) of the remaining colonies were treated with 40 mL of 65% formic acid, which was applied as a liquid to the bottom boards. This was done to assess the performance of the nine families of bees with and without the influence of tracheal mites. Bee mortality (0.2–0.5 kg/colony) was observed shortly after the formic acid treatment, thus subsequent formic acid applications (21 and 28 October 1993) consisted of only 30 mL of 65% formic acid applied to paper towels on the frames of the upper brood chamber. No bee mortality was observed following these latter two treatments. Colonies were wrapped with tarpaper in mid-November 1993.

On 22 April 1994, the tarpaper was removed and the presence of marked queens was reassessed. Colonies with unmarked queens (n = 14) and dead or nearly dead colonies (n = 12) were removed from the study. Each apiary still contained 10–15 colonies at this time. Throughout the summer colonies were managed for honey production and were evaluated for numerous other traits of economic significance to beekeepers (van

Engelsdorp and Otis 2000). Colonies treated with formic acid the previous year were retreated three times with 30 mL of 65% formic acid on paper towels on 12, 21, and 27 September 1994; no bee mortality was observed following these treatments.

Samples of worker bees were collected for tracheal mite analysis from the inner covers or the edge of the cluster on 11 November 1993, and 24 April, 4 June, 25 August, and 4 November 1994. Analyses consisted of either 100 bees/hive (November-1993 sample) or 50 bees/hive (all other samples). Mite prevalence (percentage of bees infested) was determined for all samples. In addition, mite abundance (number of mites of all life stages/number of bees in the sample) was determined for the November-1993, June-1994, and November-1994 samples (terminology *sensu* Margolis *et al.* 1982).

Samples of bees used for mite infestation analysis were taken and stored in 70% ethanol prior to analysis. Both prevalence and abundance levels were determined by cutting a transverse section of the thorax. The resulting discs were cleared of muscle tissue by placing them at 37°C in a 5% solution of potassium hydroxide for 24 h (Shimanuki and Cantwell 1978; Delfinado-Baker 1984). The number of mites of each developmental stage was ascertained by examining the revealed thoracic tracheal tubes under a dissecting microscope (40×).

All data were analyzed for normality and heterogeneity of variance (SAS Institute Inc 1985). Data sets for mite prevalence and abundance were log-transformed to maintain homogeneity of variance. Correlation analyses were performed (CORRELATION procedure; SAS Institute Inc 1985) between the variables on the same and different sample dates. A three-way ANOVA was conducted (GLM procedure; SAS Institute Inc 1985), where bee family ($n = 9$), formic acid treatment ($n = 2$), apiary site ($n = 4$), and interactions between these variables were examined simultaneously. Resulting pairwise comparisons of populations and sites, using Fisher's least significant differences test, were examined. Survival of queens reared in Ontario or British Colombia and queens treated or not treated with formic acid were compared using χ^2 tests (FREQ procedure; SAS Institute Inc 1985).

Results

November-1993 mite-prevalence values were correlated with April-1994 mite-prevalence values, but were not correlated with any other estimate of tracheal mite infestation taken in 1994 (Table 1). All mite-prevalence and -abundance values determined during and after June 1994 were significantly correlated. The magnitude of the correlation decreased with increasing time interval between the sample dates. Throughout the study, mite prevalence was consistently highly correlated with mite abundance on the same date. Consequently, we only present detailed results from the analysis of mite-prevalence data.

Before the introduction of experimental queens (12 July 1993), the percentage of bees infested by mites did not differ among the colonies intended for the introduction of queens representing the nine families of bees (Table 2; $P = 0.224$, prevalence range 0–20%). By November 1993, mite prevalence differed among the various bee families (three-way ANOVA, $F_{8,70} = 7.60$, $P = 0.007$). No differences were detected in April, June, or August 1994 ($P = 0.153$, $P = 0.107$, and $P = 0.132$, respectively), but by November 1994 significant differences had developed again among the families ($F_{8,16} = 2.82$, $P = 0.033$). The BC-2, BC-3, and BF-6 selected families and CD-8 unselected family had low mite-prevalence values throughout the study. In contrast, the unselected CD-9 family and the BC-1 and BF-4 families selected for mite resistance had consistently higher mite infestations.

TABLE 1. Relationship between *Acarapis woodi* prevalence (P) and abundance (A) over time.

		Sample date							
		Nov. 1993		Apr. 1994	June 1994		Aug. 1994	Nov. 1994	
Sample date		P	A	P	P	A	P	P	A
Nov. 1993		—	0.756	0.822	ns	ns	ns	ns	ns
			—	ns	ns	ns	ns	ns	ns
Apr. 1994				—	ns	ns	ns	ns	ns
June 1994					—	0.923	0.637	0.523	0.415
	A					—	0.507	0.655	0.586
Aug. 1994	P						—	0.726	0.795
Nov. 1994	P							—	0.935
	A								

NOTE: Correlation coefficient (r) is reported when significant (CORRELATION procedure, $P < 0.05$; SAS Institute Inc 1985). ns, not significant.

Percentages of bees infested with mites were lower in colonies treated with formic acid than in untreated colonies only on the first sample date (November 1993) following treatment (three-way ANOVA, $F_{1,70} = 45.5$, $P = 0.0001$). The difference in mite prevalence between treated and untreated colonies diminished over time (1994: April, $P = 0.116$; June, $P = 0.185$; August, $P = 0.767$). The autumn-1994 formic acid treatments had no apparent effect on tracheal mite prevalence (November, $P = 0.534$). Trends in the number of mites per bee (*i.e.*, mite abundance) paralleled mite-prevalence data. In November 1993, mean mite abundance differed greatly between bees from treated and untreated colonies ($F_{1,70} = 54.2$, $P = 0.001$), but on subsequent sampling dates no differences in mite abundance were detected between the two groups (1994: June, $P = 0.18$; November, $P = 0.69$).

There were initially no differences in mite prevalence among apiaries (Table 3; $P = 0.091$). Strong differences in mite prevalence among apiaries developed by November 1993 (three-way ANOVA, $F_{3,70} = 5.56$, $P = 0.001$), disappeared by April 1994 ($P = 0.890$), and gradually developed again over the summer and fall of 1994 (June, $P = 0.130$; August, $F_{3,22} = 2.72$, $P = 0.05$; November, $F_{3,16} = 3.84$, $P = 0.03$). The Cosby apiary had high mite infestations in November of both years. In contrast, the Travis apiary, which had the highest mite infestations in November 1993, had the lowest mite infestations by November 1994. None of the interaction effects (family × treatment, family × apiary, apiary × treatment, and family × treatment × apiary) were significant ($P > 0.05$) on any of the sample dates.

No differences were detected in the acceptance of queens reared in Ontario compared with those raised in British Columbia ($\chi^2_1 = 1.323$, $P = 0.250$), nor were there differences in the acceptance of queens of different lines ($\chi^2_8 = 9.321$, $P = 0.316$). Similarly, no differences were detected in colonies removed as a result of late fall supersedure or loss of queen markings between formic acid treated and untreated groups ($\chi^2_1 = 0.073$, $P = 0.787$) or bee families ($\chi^2_8 = 10.632$, $P = 0.223$). There were no differences between treated and untreated groups in the number of colonies that died between September 1993 and April 1994 or were removed due to the small colony size in April 1994 ($\chi^2_1 = 0.073$, $P = 0.787$). Over the course of the study the BC-3 line experienced the highest mortality.

TABLE 2. Mean ± SE percentage of *Apis mellifera* infested with *Acarapis woodi* among selected and unselected families.

	Prevalence											
	July 1993*		Nov. 1993†		Apr. 1994‡		June 1994†		Aug. 1994*		Nov. 1994†	
Family§	*n*	Mean ± SE	*n*	Mean ± SE	*n*	Mean ± SE	*n*	Mean ± SE	*n*	Mean ± SE	*n*	Mean ± SE
BC-1 (s)	6	4.8±2.0	6	9.6±2.5*a*	5	21.9±4.6	5	5.1±5.1	3	30.2±17.9	3	18.0±20.3*ab*
BC-2 (s)	6	3.8±2.2	6	2.7±2.8*ac*	5	0.9±5.0	5	0.0±5.2	4	8.0±19.0	3	0.2±16.8*c*
BC-3 (s)	7	3.5±2.0	7	0.0±2.6*c*	5	1.4±5.6	2	6.0±8.5	2	11.0±19.9	1	na
BF-4 (s)	9	1.9±2.0	8	3.2±2.6*ab*	8	1.5±4.2	7	13.1±4.4	5	36.6±15.0	3	48.4±16.2*a*
BF-5 (s)	7	1.9±1.8	7	4.4±2.4*ab*	6	1.6±4.6	6	1.8±4.8	3	20.2±18.8	3	20.9±16.8*abc*
BF-6 (s)	6	0.7±2.2	7	0.2±2.8*c*	6	1.2±4.6	6	1.3±4.6	5	13.8±14.3	5	19.4±13.3*abc*
CD-7 (u)	8	3.8±1.9	8	5.4±2.6*a*	8	4.6±4.0	8	6.3±4.0	6	27.2±12.9	6	30.2±11.7*bc*
CD-8 (u)	4	0.9±2.8	4	1.8±3.6*c*	4	3.0±5.6	3	2.6±6.8	3	0.0±19.1	0	na
CD-9 (u)	7	1.7±2.2	7	12.3±2.5*a*	6	6.3±4.5	6	1.6±4.7	4	21.0±15.9	4	38.2±14.1*ab*

NOTE: Different letters indicate significant differences (two-way ANOVA, $P < 0.05$) in tracheal mite prevalence on designated dates. na, not applicable.

* Queens either not accepted or assumed not accepted as a result of paint loss are not included in the mean calculation.

† Means confounded with formic acid treatment and apiary effect.

‡ Means confounded with formic acid treatment effect.

§ (s), selected and (u), unselected.

TABLE 3. Mean ± SE percentage of *Apis mellifera* infested with *Acarapis woodi* in colonies from different apiaries.

	Prevalence											
	July 1993		Nov. 1993*		Apr. 1994		June 1994†		Aug. 1994		Nov. 1994*	
Yard	*n*	Mean ± SE	*n*	Mean ± SE	*n*	Mean ± SE	*n*	Mean ± SE		Mean ± SE	*n*	Mean ± SE
Cosby	13	5.8±1.6	13	6.2±2.0*b*	10	11.3±4.0	9	5.3±3.7	6	42.8±14.2*a*	5	62.3±11.6*a*
Fisher	15	2.7±1.6	15	1.3±1.8*c*	13	3.7±3.4	12	5.1±3.0	12	14.1±9.4*a*	9	34.0±8.7*ab*
Travis	17	0.9±1.4	15	8.0±1.8*a*	15	5.7±3.2	12	1.4±3.2	8	6.3±1.2*b*	6	5.0±10.6*b*
Krick	15	1.3±1.4	15	2.2±1.7*bc*	15	3.4±2.9	14	6.2±2.9	9	6.9±1.0*b*	9	11.6±8.7*ab*

NOTE: Different letters indicate significant differences (three-way ANOVA, $P < 0.05$) in tracheal mite prevalence on designated dates among apiaries.

* Means confounded with family effect.

† Means confounded with formic acid treatment and family effect

Discussion

This study demonstrated extensive variation in tracheal mite infestation within the families of bees under investigation. Because mite prevalence did not differ among bee families at the beginning of the study, we are confident that real differences in mite resistance underlie our results. Nearly all North American studies comparing bees of different ancestries have detected differences in relative resistance to tracheal mites (Gary and Page 1987; Gary *et al.* 1990; Milne *et al.* 1991; Szabo *et al.* 1991; Danka *et al.* 1995; Lin *et al.* 1996; Guzman *et al.* 1998). Successful breeding efforts (Page and Gary 1990; Danka and Villa 2000; Nasr *et al.* 2001) have confirmed that tracheal mite resistance has a genetic basis.

Our study differs from others because it examined differences in mite infestations among families within three stocks. The results are particularly relevant to people involved in bee breeding programmes. By November 1993, significant differences in mite prevalence were evident among the three families representing each of the three stocks (BC-2 and BC-3 < BC-1; BF-6 < BF-4 and BF-5; CD-8 < CD-7 and CD-9). This suggests that comparisons of mite infestations among bee stocks, without consideration of family variation within a stock, could mask real differences in mite resistance. Conversely, comparison of individual colonies may identify colonies that appear to be highly mite resistant, but without the ability to replicate performance, this identification cannot be confirmed statistically. Evaluation of a family of bees (*e.g.*, several daughter queens reared from a potential breeder queen) as done in our study solves these problems. More accurate evaluation of breeder queens can be expected if, as in our study, all daughter queens are mated to drones from a common source so that differences among sister colonies derive predominantly from the breeder queen under evaluation. Future breeder queens can be chosen by selecting individual colonies within each family that exhibit the lowest mite infestation, thereby maintaining if not improving mite resistance.

In the case of the British Columbia lines, the variability in mite infestations may reflect the relatively few generations of selection for tracheal mite resistance. The variable performance of the Buckfast lines is inconsistent with previous results (Milne *et al.* 1991; Danka *et al.* 1995; Lin *et al.* 1996; Nasr *et al.* 2001) that have consistently shown that Buckfast bees are more resistant to tracheal mites than unselected North American bees. All of these studies, however, like ours have found variation within the Buckfast bees under evaluation, pointing out the need to confirm mite resistance of a colony prior to rearing daughter queens from it.

Both mite prevalence and mite abundance were quantified on three separate dates. The two measures quantified from the same set of bees were highly correlated, as has been generally true in all the studies done on tracheal mites at the University of Guelph. In this study, mite prevalence proved to be more sensitive in detecting differences among the families of bees (*e.g.*, probability values were generally smaller) than mite abundance. This finding contradicts that of Lin *et al.* (1996) who reported that abundance is the better variable. The range of mite prevalence among colonies was greater in the present study (1–48%) than in the Lin *et al.* (1996) study (10–23%), which may have improved the ability of this measure of mite infestation to detect differences. Prevalence is considerably easier, faster, and cheaper to quantify than abundance; however, abundance should be the preferred variable to quantify when mite populations are small or the anticipated range in prevalence values is low.

Beekeepers would welcome the ability to predict the need to control tracheal mites. The correlations between prevalence and abundance values at different sample dates in this study demonstrated a weak ability to predict future tracheal mite infestations. For example, the overwintering mite populations in the second year (November

1994) were poorly predicted in June and only moderately well predicted in August. By August it is almost too late for beekeepers in north temperate climates to collect samples, have them analyzed, and apply appropriate treatments before winter. Dawicke *et al.* (1989) reached a similar conclusion.

We attempted to control mites in half of the colonies to evaluate honey production both with and without the influence of mites. This objective could not be realized because the differences in mite infestations between treated and untreated hives created with the September-1993 formic acid treatment had disappeared by the spring of 1994.

Our experimental design and statistical analysis permitted us to treat apiary as a fixed effect, and to detect highly significant differences in mite infestations among the four apiaries (Table 3). These differences suggest that apiary locations influence mite populations. Because the apiaries were relatively close (<6 km) to each other and there were no apparent differences in resource availability (as indicated by nonsignificant differences in colony mass gain among apiaries; van Engelsdorp 1995; van Engelsdorp and Otis 2000), we speculate that the apiary effect probably reflects micro-environmental factors. The apiaries differed in physical features (*e.g.*, tree cover, proximity to standing water) that probably influenced temperature, humidity, and wind speed. Climatic conditions are known to affect mite infestations. For example, Harbo (1993) demonstrated that elevated colony temperatures can kill tracheal mites. Local differences in tracheal mite infestations have been reported by Czerwiński *et al.* (1963) and Niemczuk (1970) who suggested that the local differences in tracheal mite infestations were related to differences in temperature and humidity. The effects of microclimatic conditions on tracheal mite infestations are very poorly understood and warrant further investigation.

Acknowledgments

We could not have performed this study without the assistance of our technicians, Paul Kelly and Tillie Welsh, and of beekeeper Bill Minnick. M Damus, J McCarthy, C Morin, and A Meinen provided additional field assistance. C Scott-Dupree and T Szabo provided feedback on versions of the manuscript, as did A Aldous, N Calderone, and R Magee. We thank the three anonymous reviewers of the manuscript for their helpful and insightful comments. Funding was provided by Agriculture Canada FSAM-II monies administered by the Canadian Honey Council, the Ontario Ministry of Agriculture, Food and Rural Affairs Bees and Pollination program, and several fellowships to DvE from the University of Guelph.

References

Adam Br. 1987. *Beekeeping at Buckfast Abbey*. Hebden Bridge, United Kingdom: Northern Bee Books

Bailey L. 1958. The epidemiology of the infestation of the honeybee, *Apis mellifera* L., by the mite *Acarapis woodi* Rennie and the mortality of infested bees. *Parasitology Research* **48**: 493–506

Bailey L. 1961. The natural incidence of *Acarapis woodi* (Rennie) and the winter mortality of honeybee colonies. *Bee World* **42**: 96–100

Bailey L, Ball BV. 1991. *Honey Bee Pathology*. 2nd edition. London, United Kingdom: Academic Press

Bailey L, Lee DC. 1959. The effect of infestation with *Acarapis woodi* (Rennie) on the mortality of honey bees. *Journal of Insect Pathology* **1**: 15–24

Czerwiński M, Januszewski J, Niemczuk R. 1963. Analiza i ocena wpływu środowiska geograficznego dolnego ślaska na rozprzestrzenianie sie choroby roztoczowej pszczół. *Zycie weteryn* **3**: 19–24 [Cited in *Apicultural Abstracts* No. 352/65]

Danka RG, Villa JD. 2000. Inheritance of resistance to *Acarapis woodi* (Acari: Tarsonemidae) in first-generation crosses of honey bees (Hymenoptera: Apidae). *Journal of Economic Entomology* **93**: 1602–5

Danka RG, Villa JD, Rinderer TE, Delatte GR. 1995. Field test of resistance to *Acarapis woodi* (Acari: Tarsonemidae) and of colony production by four stocks of honey bees (Hymenoptera: Apidae). *Journal of Economic Entomology* **88**: 584–91

Dawicke BL, Otis GW, Scott-Dupree CD. 1989. Predicting tracheal mite infestations and effects on colonies. *American Bee Journal* **129**: 814

Delfinado-Baker M. 1984. *Acarapis woodi* in the United States. *American Bee Journal* **124**: 805–6

Eischen FA. 1987. Overwintering performance of honey bee colonies heavily infested with *Acarapis woodi* (Rennie). *Apidologie* **18**: 293–304

Eischen FA, Cardoso-Tamez D, Wilson WT, 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, 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. *American Bee Journal* **129**: 195–7

Gary NE, Page RE Jr. 1987. Phenotypic variation in susceptibility of honey bees, *Apis mellifera*, to infestation by tracheal mites, *Acarapis woodi*. *Experimental and Applied Acarology* 3: 291–305

Gary NE, Page RE Jr, Morse RA, Henderson CE, Nasr ME, Lorenzen K. 1990. Comparative resistance of honey bees (*Apis mellifera* L.) from Great Britain and United States to infestation by tracheal mites (*Acarapis woodi*). *American Bee Journal* **130**: 667–9

Guzman-Novoa E, Zozaya-Rubio A. 1984. The effects of chemotherapy on the level of infestation and production of honey in colonies of honey bees with acariosis. *American Bee Journal* **124**: 669–72

Guzman LI de, Rinderer TE, Delatte GT. 1998. Comparative resistance of four honey bee (Hymenoptera: Apidae) stocks to infestation by *Acarapis woodi* (Acari: Tarsonemidae). *Journal of Economic Entomology* **91**: 1078–83

Harbo JR. 1993. Field and laboratory tests that associate heat with mortality of tracheal mites. *Journal of Apicultural Research* **32**: 159–65

Huxter E, Clark K. 1991. Identification and development of Canadian honey bee stocks resistant to tracheal mites. CAFDI (Canadian Agri-Food Development Initiative) Project No. 4075/90-91/E902-162. Interim report, March 1991

Huxter E, Clark K. 1992. Identification and development of Canadian honey bee stocks resistant to tracheal mites. CAFDI Project No. 4075/90-91/E902-162. Final report, January 1992

Komeili AB, Ambrose T. 1990. Biology, ecology and damage of tracheal mites on honey bees (*Apis mellifera*). *American Bee Journal* **130**: 193–9

Lin H, Otis GW, 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)). *Experimental and Applied Acarology* **20**: 87–101

Loper GM, Waller GD, Steffens D, Roselle RM. 1992. Selection and controlled natural mating: a solution to the honey bee tracheal mite problem. *American Bee Journal* **132**: 603–6

Margolis L, Esch GW, Holmes JC, Kuris AM, Schad GA. 1982. The use of ecological terms in parasitology. (Report of an *ad hoc* committee of the American Society of Parasitologists.) *Journal of Parasitology* **68**: 131–3

Milne CP Jr, Otis GW, Eischen FA, Dormaier JM. 1991. A comparison of tracheal mite resistance in two commercially available stocks of honey bees. *American Bee Journal* **131**: 713–8

Nasr ME, Otis GW, Scott-Dupree CD. 1991. Genetic breeding for tracheal mite resistance in Ontario, 1990. *Bee Science* **1**: 122–3

Nasr ME, Otis GW, Scott-Dupree CD. 2001. Tracheal mite resistance in honey bees (Hymenoptera: Apidae): divergent selection and evaluation. *Journal of Economic Entomology* **94**: 332–8

Niemczuk R. 1970. Climatic treatment in acarapidosis apium. *Acta Parasitologica Polonica* **18**: 551–4 [Cited in *Apicultural Abstracts* No. 760/72]

Otis GW, Scott-Dupree C. 1992. Effects of *Acarapis woodi* on overwintered colonies of honey bees (Hymenoptera: Apidae) in New York. *Journal of Economic Entomology* **85**: 40–6

Page RE, Gary NE. 1990. Genotypic variation in susceptibility of honey bees (*Apis mellifera*) to infestation by tracheal mites (*Acarapis woodi*). *Experimental and Applied Acarology* **8**: 275–83

Pettis JS, Wilson WT. 1996. Life history of the honey bee tracheal mite *Acarapis woodi* (Acari: Tarsonemidae). *Annals of the Entomological Society of Amercia* **89**: 368–74

Sammataro D, Needham GR. 1996. Host-seeking behaviour of tracheal mites (Acari: Tarsonemidae) on honey bees (Hymenoptera: Apidae). *Experimental and Applied Acarology* **20**: 121–36

Sammataro D, Gerson U, Needham G. 2000. Parasitic mites of honey bees: life history, implications, and impact. *Annual Review of Entomology* **45**: 519–48

SAS Institute Inc. 1985. *SAS/STAT user's guide, version* 6.04. Cary, North Carolina: SAS Institute Inc

Shimanuki H, Cantwell GE. 1978. *Diagnosis of honey bee diseases, parasites, and pests.* USDA Publication ARS-NE-87

Szabo TI, Lefkovitch LP, Clark KJ. 1991. Comparative resistance of honey bees from a closed population to infestation by tracheal mites. *American Bee Journal* **128**: 740–1

Tomasko M, Finley J, Harkness W, Rajotte E. 1993. A sequential sampling scheme for detecting the presence of tracheal mite (*Acarapis woodi*) infestations in honey bee (*Apis mellifera* L.) colonies. Bulletin No. 871, Pennsylvania State Agriculture Experiment Station, University Park

van Engelsdorp D. 1995. A field evaluation of nine genetic lines of honey bees for tracheal mite resistance and other economically important traits. MSc thesis, University of Guelph, Guelph, Ontario, Canada

van Engelsdorp D, Otis GW. 2000. Application of a modified selection index for honey bees (Hymenoptera: Apidae). *Journal of Economic Entomology* **93**: 1606–12

(Received: 30 January 2001; accepted: 16 August 2001)