# Effects of *Acarapis woodi* on Overwintered Colonies of Honey Bees (Hymenoptera: Apidae) in New York

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**ABSTRACT** Colonies of honey bees, *Apis mellifera* L., infested with *Acarapis woodi* (Rennie) were studied during the four winters of 1985–1989 in New York state. Samples of bees were obtained from colonies on several dates from fall to spring to determine mite prevalence and mite load scores. Mite infestations were much heavier than those reported elsewhere in North America. Over the two winters for which adequate data were available (1987–1988 and 1988–1989), colonies with heavy mite infestations had significantly greater mortality. Spring brood areas were negatively correlated with mite prevalence and mite load scores. However, the strength of these correlations varied depending on the month and the year. These results indicate that tracheal mites have a substantial negative effect on colonies of honey bees in New York.

**KEY WORDS** Insecta, *Apis mellifera*, tracheal mites, colony mortality

*Acarapis woodi* (Rennie) is a parasite of the trachea of the honey bee, *Apis mellifera* L. The adults of these mites enter the trachea of young bees, where they feed on host haemolymph and reproduce. Among other effects, they have been reported to reduce the longevity of adult bees (Bailey 1958, Bailey & Lee 1959, Giordani 1962, Maki et al. 1986) and to increase colony mortality during the winter (Bailey 1958, 1961; Bailey & Lee 1959; Furgala et al. 1989; Komeili & Ambrose 1990; Royce & Rossignol 1990). Kjer et al. (1989) provided a recent summary of the biology of tracheal mites and their effects on honey bee colonies.

In 1984, tracheal mites were discovered in the United States for the first time. They have subsequently been recorded in >42 states and eight provinces of Canada. There has been disagreement over the potential seriousness of the tracheal mite as a pest of honey bees in North America. This uncertainty arises from conflicting information on the degree of susceptibility of North American bees to the mites (Adam 1962, 1968; Bailey 1965, 1967, 1985; Bailey & Perry 1982) and from a lack of data on the economic effects of tracheal mites in North America. Much of the more recent research on this pest was conducted by Bailey and his associates in England in the 1950s and 1960s, at least 35 yr after *A. woodi* is known to have become established there. Their conclusion that tracheal mites are rarely abundant enough to have much effect on managed bee colonies may not be applicable to the North American setting, where the mite appears to have been recently introduced (Eckert 1961, Shimanuki et al. 1983, Shimanuki & Knox 1989, Furgala et al. 1989), and the bees may not have evolved resistance mechanisms (Adam 1987, Taber 1988). Recent data suggest that tracheal mites are associated with economic losses to beekeepers in North America (Eischen 1987, Eischen et al. 1989, Furgala et al. 1989).

Our objectives were to determine the incidence of mites in infested colonies in commercial beekeeping operations in New York, to quantify the relationship of mite prevalence and mite-load scores to colony mortality and spring brood areas, and to test for association between the incidence of tracheal mites and infection with the protozoan, *Nosema apis* Zander.

## Materials and Methods

Apiaries with honey bee colonies known to be infested with tracheal mites were studied in New York State during four winters. In no case was it known how long the apiaries had been infested with mites. In 1985–1986, we worked in two apiaries with 27 and 12 hives respectively, located near LeRoy, Genesee County, N.Y. The following year, two nearby apiaries, each with 20 colonies and belonging to the same beekeeper, were studied. The colonies were neither equalized in strength nor requeened because we wanted to study colonies that were under routine fall management as performed by the beekeeper. Colonies had two or three hive bodies, with top entrances or holes drilled in the upper hive body and sometimes with empty hive bodies with

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frames on top of the hives above the inner covers. In preparation for overwintering, colonies were fed sugar syrup (1:1 sugar/water) containing sodium sulfathiazole to control American foulbrood disease; they were not fed fumagillin. In both years, several colonies had insufficient food reserves to survive the winter. All live colonies were fed granulated sucrose on the inner hive cover on 17 February 1986 or 10 February 1987, respectively, to prevent starvation.

Samples of adult bees were collected from study colonies on 17 October, 26 November, 2 January, 17 February, 15 March, 28 April, and 6 June 1985–1986 and on 20 June, 16 September, 28 October, 8 December, 29 January, 17 March, and 5 May 1986–1987. Bees were collected from the tops of frames, at top hive entrances, or directly from the outer edge of the bee cluster. (Results by Robinson et al. [1986] indicated that sampling location within the hive does not influence the prevalence of mites in the sample.) Colony mortality was determined during sampling. It was not possible to sample bees without some minor disturbance to the colonies.

During the winter of 1987–1988, we worked with colonies belonging to a different beekeeper north of Ithaca, N.Y., between Cayuga and Oneida Lakes. All colonies in four apiaries known to be infested with mites were studied. The apiaries contained 21, 22, 19, and 5 colonies. Colonies were fed sugar syrup (1:1) containing fumagillin from open barrels placed in the apiary in early October. Most were overwintered in two or three brood chambers with substantial stores of honey. Extender patties containing oxytetracycline hydrochloride (Terramycin 25, Pfizer Pharmaceutical Company, Mississauga, Ont.) were applied in April. No other drugs were administered. Bees were sampled from inner covers or outer frames on 9 October, 24 November, and 12 March 1987–1988.

As part of a comparative study of different miticides in a fall treatment program, four additional apiaries containing 110 colonies belonging to one beekeeper were studied in Orleans, Monroe, and Genesee counties in 1988–1989. Samples were taken from inner covers or outer frames on 8 October, 9 November, and 21 April. The miticides had no effect on controlling mite populations (unpublished data); consequently, we report colony mortality as well as initial mite prevalence values.

Samples usually contained >100 bees; few samples contained <80 and none had <60 bees. Bees were placed immediately into 190-ml plastic jars containing 70% ethanol. The presence of mites was determined for ~100 bees in each sample unless the sample contained fewer bees. Thoracic disks were cut such that they contained the main tracheal trunks. Groups of ≥50 disks were incubated in 5% KOH for 24 h in a warm (43°C) oven (Delfinado-Baker 1984). Each disk was inspected individually with a dissecting microscope at 40× or 63× for the presence or absence of mites. If there was any doubt concerning the presence of mites, the tracheae were removed, mounted on a microscope slide, and viewed at 40× or 80× under a compound microscope. In addition, for the first 3 yr, samples taken in March were analyzed for *N. apis* using the technique described by Cantwell (1970).

For all years, we report mite prevalence, which is simply the percentage of mite-infested bees in the sample (Margolis et al. 1982, "prevalence (w)" of Eischen 1987, Eischen et al. 1989). In the second and third winters, approximate mite infestation was ranked for each trachea examined. Rankings were: 0, no mites; 1, <10 mites of all stages; 2, 11–20 mites; 3, 21–30 mites; 4, 31–40 mites; 5, >40 mites. The sum of the ranks for the two tracheae of a bee provided a mite load score. The sum of these scores for all bees in a sample divided by the number of bees in the sample gave the mean mite load score ("parasite load score" of Eischen 1987, Eischen et al. 1989).

Spring brood areas of the surviving colonies were estimated on 28 April 1986, 5 May 1987, and 26 April 1988. A frame divided into six sections of equal area was placed in front of each comb containing brood. Only one side of each comb was measured because brood patterns on the two sides of a comb are usually similar. The percentage of each section containing sealed worker brood was estimated to the nearest 5%. These percentages were converted to areas, doubled to account for the two sides of the combs, and summed to yield total brood area of the colony.

Frequency distributions of mite prevalence values of colonies sampled in October of each year of the study were produced for comparison. A contingency table was constructed to compare mortality of colonies having low mite infestation with those of moderate and high infestation. The mortality data were further analyzed with a logistic regression analysis (SAS general linear models procedure) (SAS Institute 1985). This statistical technique uses the binomial data of colony death or survival over the range of observed mite prevalence values to calculate predicted values of the probability of mortality at any given autumn prevalence of mites. Values of mite prevalence were transformed (arcsine transformation) before correlations were calculated between mite prevalence and spring brood areas. A Spearman rank correlation analysis (Steel & Torrie 1980) was conducted on *Nosema* spore counts and mite prevalence of bees from the same 15 March 1986 samples to test for an association between infestation with these two organisms.

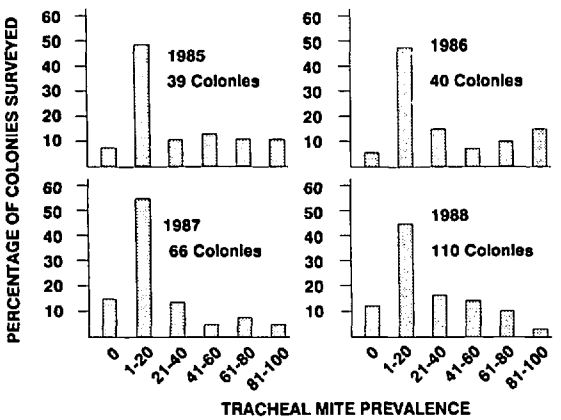


Fig. 1. Frequency distribution of October mite prevalence values (i.e., percentage of mite-infested bees) for 4 yr of study in New York.

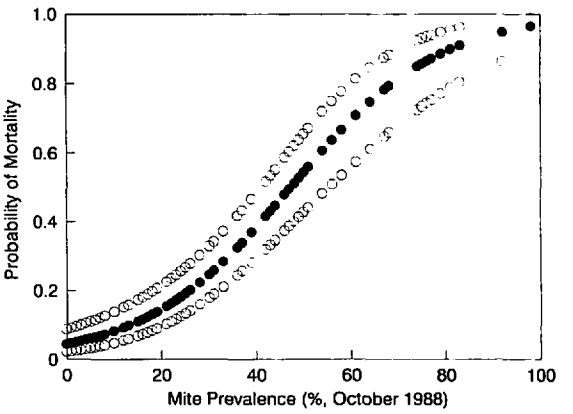


Fig. 2. Results of logistic regression analysis on 1988–1989 data of colony mortality and mite prevalence values. Solid dots represent the predicted probability of mortality as calculated from the regression equation; open circles indicate 95% confidence limits calculated for each predicted mortality value.

## Results

Frequency distributions of mite prevalence values of colonies sampled in October of each year of study are presented in Fig. 1. The percentage of colonies with >20% mite prevalence was 43.6% ($n$ = 39) in 1985, 47.5% ($n$ = 40) in 1986, 30.3% ($n$ = 66) in 1987, and 43.6% ($n$ = 110) in 1988; the overall average was 40.8% ($n$ = 255). This compares with an average of 10.4% (range in yearly average, 0.7–21.5%) reported by Bailey (1961) from 1955 to 1960 in England. Almost all colonies sampled in New York were infested with mites; only three colonies in 1987–1988 and five colonies in 1988–1989 had no mites detected in three 100-bee samples.

Colony mortality was related to mite infestations. In 1987–1988, lightly infested colonies (0–20% mite prevalence, $n$ = 46) in October experienced 9.1% mortality during the subsequent winter. Among moderately infested colonies (21–60% mite prevalence, $n$ = 12) there was 16.7% mortality, and among heavily infested colonies (>60% mite prevalence, $n$ = 8), 75% died. Mortality among more heavily infested colonies was significantly greater ($G$ test with Yates correction, $G$ = 5.36, $P < 0.025$). In 1988–1989, the mortality of moderately infested (33.3%, $n$ = 33) and heavily infested (86.6%, $n$ = 15) colonies was even greater than in the preceding year. The combined mortality among moderately and heavily infested colonies was significantly greater ($G$ test with Yates correction, $G$ = 23.24, $P < 0.001$) than the mortality among lightly infested colonies (8.1%, $n$ = 62). For comparison, Bailey (1961) recorded values similar to ours for mortality of colonies with light infestation (9.7%, $n$ = 893), moderate infestations (32.0%, $n$ = 78), and heavy infestations (82.9%, $n$ = 35).

The mortality data for 1988–1989 have been further analyzed with a logistic regression analysis to calculate predicted values of the probability of mortality at any given autumn prevalence of mites (Fig. 2). The regression equation, $\ln(p/[1-p]) = -3.094 + 0.6478x$, where $p$ is the probability of mortality and $x$ is mite prevalence, is highly significant ($P < 0.001$). Inclusion of an $x^2$ term failed to improve the fit of the regression line to the data ($P$ = 1.0).

In the first 2 yr of the study, the more heavily infested colonies experienced greater mortality (in 1985–1986, 10/19 lightly infested colonies and 13/17 moderately and heavily infested colonies died; in 1986–1987, 12/20 lightly infested colonies and 17/19 moderately and heavily infested colonies died). However, these differences were not significant because of the high overall mortality (presumably the result of poor colony management) and the relatively small numbers of colonies studied.

There was a negative correlation between mite infestation level and spring brood area. For each of the first 3 yr of the study, mean prevalence was negatively correlated with log-transformed spring brood area at some time during fall or winter (Table 1). These data suggest that those colonies with high mite infestations that did survive usually had reduced brood areas. However, the pattern from year to year was variable. In 1985–1986, the highest correlation occurred in March, whereas in 1986–1987, the highest correlation occurred in late October. In the final year, 1987–1988, the correlations for fall and early spring were almost identical. Data from late November–early December samples are depicted in Fig. 3. The additional information obtained from mite load scores did not substantially improve the values of the correlation coefficients.

We tested for an association between infestation with tracheal mites and *N. apis* infection. A

**Table 1. Correlations of mite prevalence and spring brood area**

| Year | Date[a] | Mite prevalence, $r^b$ | Mite load score, $r^c$ |
|---|---|---|---|
| 1985–1986 | 17 Oct. | −0.168ns | — |
| | 26 Nov. | −0.50ns | — |
| | 2 Jan. | −0.564* | — |
| | 17 Feb. | −0.605* | — |
| | 15 Mar. | −0.723* | — |
| | 28 Apr. | −0.594* | — |
| 1986–1987 | 16 Sept. | −0.587ns | −0.519ns |
| | 28 Oct. | −0.718* | −0.684* |
| | 8 Dec. | −0.707* | −0.648* |
| | 29 Jan. | −0.614ns | −0.627ns |
| | 17 Mar. | −0.632* | −0.517ns |
| | 5 May | −0.427ns | −0.444ns |
| 1987–1988 | 9 Oct. | −0.578*** | −0.662*** |
| | 24 Nov. | −0.552*** | −0.668*** |
| | 12 Mar. | −0.572*** | −0.627*** |

Spring brood areas were measured on 28 April 1986, 5 May 1987, and 26 April 1988, and were log-transformed before correlations were calculated. ns, not significant; *, $P < 0.05$; ***, $P < 0.001$.

[a]Date samples were taken for mite analyses. The data for italicized dates are presented graphically in Fig. 3.

[b]Linear correlations of mite prevalence (arcsine square root of percent bees infested) and spring brood areas.

[c]Linear correlations of mite load scores (based on rankings of number of mites per bee) and spring brood areas.

Spearman rank correlation analysis (Steel & Torrie 1980) was conducted on *Nosema* spore counts and mite prevalence of bees from the same 15 March 1986 samples. The correlation coefficient was negative and not significant ($r_s = -0.321$; $0.10 > P > 0.05$). *Nosema* was found to be negligible in 1987 and 1988. Spore counts were not taken in 1989.

## Discussion

In this study we have documented relatively high levels of mite infestation and a negative relationship between mite prevalence and both winter survival of colonies and the strength of surviving colonies in spring. Mite prevalence was much higher than reported by Bailey (1961) in England during 1955–1960. In comparison with Bailey's (1961) findings, we consistently found that three to four times more colonies in New York had mite prevalence values >20% regardless of location, management system, and queen stocks. Other researchers have reported high mite prevalence in Mexico (Eischen 1987), Minnesota (Furgala et al. 1989), Arizona (Waller & Hines 1990), and elsewhere in the United States. In contrast, tracheal mites are currently present at low levels throughout most of Europe and only occasionally become common enough to affect colony performance (Otis 1990). The most likely explanation for the striking difference between mite prevalence values in North America and Europe is that North American bees lack resistance to tracheal mites. This has also been suggested by Adam (1962, 1968) and Taber (1988). Studies that have been conducted to address this question have suggested that there was no difference in susceptibility between British and North American stocks of bees (Bailey 1965, 1967; Gary et al. 1990). However, a recent reevaluation of Bailey's experiments reached the opposite conclusion (Kjer et al. 1989). Additional studies comparing European and North American stocks of bees are needed to settle these contradictory conclusions.

Other explanations for the difference in mite prevalence values are possible. It has long been known that climate and foraging conditions influence the incidence of tracheal mites (Bailey 1985). However, this does not appear to be the cause of the differences that have been observed. According to Bailey (1985), the generally stronger honey flows in North America should result in a lower incidence of tracheal mites, whereas we have observed a higher mite incidence. Another possibility is that the mites in North America are different than those in Europe. There are no data to address this question at present.

In all 4 yr of our study, heavily infested colonies experienced greater winter mortality. In the latter 2 yr in which colony management was not

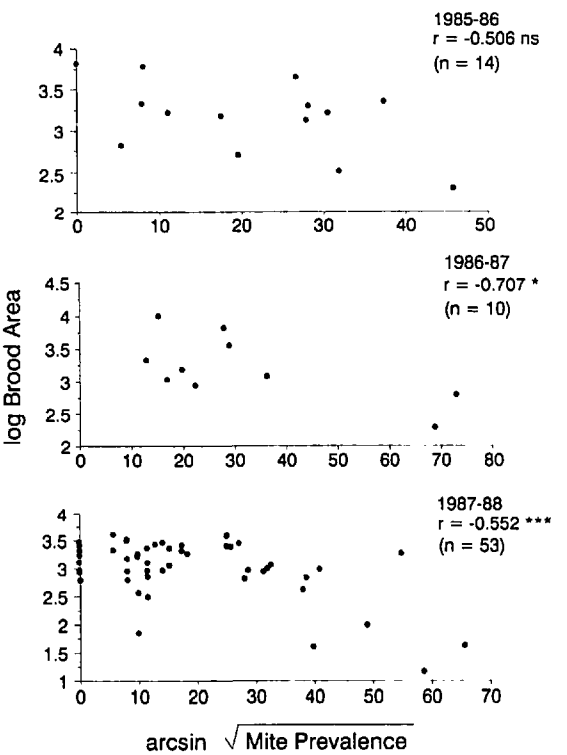


Fig. 3. Relationship between early winter mite prevalence and spring brood areas. Mite prevalence was based on samples taken on 26 November 1985, 8 December 1986, or 24 November 1987. Correlations of spring brood areas and mite prevalence values from all months are presented in Table 1.

a major factor affecting mortality, the probability of colony death was significantly related to mite prevalence. Interestingly, our data on the relationship between mite prevalence and percentage mortality are very similar to those of Bailey (1961). In contrast, in warmer climates, tracheal mites infrequently cause death of colonies (Eischen 1987; H. Cromroy, personal communication) and have little effect on individual bees (Gary & Page 1989). Where winters are longer and more severe than in New York (e.g., Minnesota), mortality at any given mite prevalence is greater than we recorded (Furgala et al. 1989). This trend of greater effect of tracheal mites in colder climates is probably related to the longer broodless period and the continued mite reproduction in their host bees throughout most of the winter (Otis et al. 1988). Winter bees in New York must live at least 5 mo, and the direct effect of the parasitic mites over an extended period of time and perhaps the indirect effect of pathogens that may be introduced into the bees by feeding mites apparently cause the death of the colony before brood production can produce enough young bees in the spring. It is through this effect on wintering colonies that tracheal mites are likely to have their most significant effect on North American beekeeping.

The physical symptoms of mite-infested colonies deserve mention. There were no obvious signs of mite infestation in the bees themselves. Generally the colonies appeared normal as well, but we had the impression that the winter cluster was not as tight in heavily infested colonies. Moreover, on warm, late-winter days, large numbers of bees could sometimes be seen crawling away from heavily infested hives and not returning. Similar behavior has been observed by Killion & Lindenfelser (1988) and Thoenes & Buchmann (1990). By early spring, these colonies frequently had ample honey and pollen stores but only a handful of (or no) bees alive in the hive. The often rapid reduction in numbers of bees led to chilled brood and small clusters of dead bees on the combs, very different from the tight cluster of bees between frames and in cells when colonies starve in late winter. These are symptoms that beekeepers now associate with tracheal mite infestations.

Mite infestations are highly variable over time. Within a colony, mite prevalence can increase or decrease dramatically over a period of a few weeks (at the same time that neighboring colonies are undergoing changes in prevalence in the opposite direction) (Otis et al. 1988). Rapid declines in mite infestation typically occur in late spring as the number of newly emerged bees is rapidly increasing and old infested bees are dying (see summary in Otis et al. 1988), but other changes in prevalence during fall and winter remain unexplained. This unexplained variation confounds detailed analysis of the changes in mite populations. It may also have contributed to the relatively weak correlations of mite prevalence and mite load scores with spring brood areas.

A recent report by Hyser (1986) suggested that there was a positive correlation between *Nosema* infection and tracheal mite prevalence. In our study, there was a weak negative correlation between these two variables. Lozano de Haces et al. (1989) detected no statistical association between the presence of *Nosema* and *A. woodi* in northeastern Mexico. Wille (1966) found very little *Nosema* in colonies infested with tracheal mites, although other indications of pathogenic organisms such as bacterial septicaemia and amoeba cysts were present. From the information that is available, it seems that *Nosema* is not consistently associated with tracheal mites, if at all. If further research indicates a significant relationship between mites and *N. apis*, it might be usefully expanded by investigating correlations with other variables (e.g., microclimatic differences between apiary sites).

Several studies have concluded that *A. woodi* are not significant pests of honey bees in Europe (Bailey 1961, 1964; Otis 1990; Wille et al. 1987). These results strongly contrast with recent data on the effects of tracheal mites on overwintering honey bee colonies in North America (Eischen 1987; Eischen et al. 1989; Furgala et al. 1989; Waller & Hines 1990, present study). The major difference appears to be due to the widespread infestation of virtually all colonies, often at high levels of mite prevalence, in fall and winter in North America. Research directed at understanding the host–parasite interaction, especially mite and bee population dynamics and mechanisms of resistance to mites, are needed to enhance the effectiveness of control strategies.

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