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Reduction of Tracheal Mite Parasitism of Honey Bees by Swarming

L. A. Royce, P. A. Rossignol, D. M. Burgett and B. A. Stringer

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Reduction of tracheal mite parasitism of honey bees by swarming

L. A. ROYCE, P. A. ROSSIGNOL, D. M. BURGETT AND B. A. STRINGER

Department of Entomology, Cordley Hall, Oregon State University, Corvallis, Oregon 97331-2907, U.S.A.

SUMMARY

Based on population dynamics, tracheal mite (*Acarapis woodi*) parasitism of colonies of honey bees (*Apis mellifera*) appears to be, potentially at least, regulatory and stable. Empirical and theoretical considerations suggest, however, that intracolony population dynamics of mite–honey bee worker seem to be unstable in managed situations where honey bee worker population is allowed to grow unchecked.

Experimental studies showed that tracheal mite population levels increased in a managed honey bee colony but were impaired in one in which brood rearing was interrupted by loss of the queen. Mite densities but not prevalence were lowered in experimental swarms kept from rearing brood.

We propose that swarming reduces mite density within a colony, therefore implicating modern techniques of hive management in the sudden historical appearance of the mite on the Isle of Wight.

INTRODUCTION

The tracheal mite parasite *Acarapis woodi* (Rennie) feeds and reproduces in the prothoracic tracheae of the European honey bee, *Apis mellifera* L., where it has a detrimental impact on the individual host and, ultimately, on the colony (Morgenthaler 1931; Giordani 1965, 1977; Eischen 1987; Eischen *et al.* 1989; Royce & Rossignol 1990*a*, *b*), although this claim remains contended (Gary & Page 1989). Early this century, the mite was discovered on the Isle of Wight and implicated to be the etiological agent of a disease that was seriously affecting apiculture there (Rennie 1921); the mite has since appeared in continental Europe and in the Americas.

The duration of its life cycle, from egg to egg, is approximately 20 days (Bailey 1963; Royce *et al.* 1988), which is close to the average honey bee's life expectancy of 30 days during the peak flowering period (Wilson 1971). Newly emerged adult female mites probably mate within the tracheae (Sachs 1958) and then migrate from the honey bee of their birth to a new host honey bee wherein they begin to reproduce. Because of this relatively long life cycle and the observation that infestation rates are highest among recently emerged honey bees (Lee 1963), it appears that young honey bees are crucial for a new infestation, and may in fact be exclusively invaded when available (Gary *et al.* 1989).

We described population dynamics of the parasite and found that a mechanism of mite reduction may be required at the intracolony level. Because mites preferentially invade newly emerged honey bees and because these hosts would be unavailable for a total of several weeks before and after swarming, we determined whether or not a relationship exists between delay of brood appearance and mite density. Epizootiological consequences of historical and management developments are discussed in light of these experiments.

MATERIALS AND METHODS

First, two colonies were maintained in movable frame hive bodies and managed to prevent swarming. Every 12 days from 1 April until 12 October, 1988, the honey bee population was estimated by the method of Burgett & Burikam (1985). After 12 October, cold weather limited examination of every frame within the hives. Colony no. 1 was requeened 1 May. In colony no. 2, requeening failed 5 June and supersedure (natural requeening) was allowed and a new queen started egg laying on 30 June. These events provided two contrasting population structures. Worker bees were sampled for mites weekly throughout spring and summer and frozen for later dissection. Tracheae were excised and placed in dishes where they were opened and all mite stages counted. Once the number of workers was estimated as described above, it was multiplied by the mean number of mites from a sample of 30–200 honey bees to obtain the number of mites in the whole colony.

Second, three colonies of honey bees (labelled A, B and C) with 67, 67 and 70% levels of mite infestation were used for making artificial 'queenright' packages or 'swarms'. A package is an apicultural term for a large number of workers taken from a colony, essentially a man-made swarm, and put in a cage or package. Two swarms were shaken from each of the colonies on 3 July, 1989 and ranged in weight from 0.7 to 1.2 kg. New queens were kept in queen cages within the packages. After 2 days, a package from each of the colonies was 'hived' (placed in and allowed to colonize) in a 'nuclear' colony containing three frames,

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each frame with a 7 cm starter strip of beeswax foundation and one frame with honey. A nuclear colony is an apicultural term for a four frame hive body, half the size of a standard hive of 45 l. The other swarms were set up in nuclear colonies 11 days after splitting in sets of three (one from each colony) in the same manner. Swarms were fed 50% high fructose corn syrup before being hived. The nuclear colonies were placed with hive entrances facing in different directions to reduce drift of workers between colonies.

The first sample of 30 honey bees from the nuclear colonies was taken three days from being established and then weekly thereafter for four weeks. The population of eggs, larvae, pupae and adult honey bees in each nucleus was monitored (Burgett & Burikam 1985). All colonies were maintained in Corvallis, Oregon.

Swarms were also shaken from the same colonies and kept for 5 and 8 days after splitting before being used to start a nuclear colony. Several of these nuclear colonies lost their queen before completion of the experiments and were not included in the experiment.

Table 1. *List of symbols*

X	population of uninfested colonies
Y	population of infested colonies
α	death rate of colonies due to tracheal mites
β	transmission coefficient (drifting)
ρ	modified growth characteristics of infested colonies
a	rate of swarming
b	death rate due to natural causes
f	proportion of swarms from infested colonies which will be infested
x	rate of worker bee production by queen
r_c	intrinsic rate of natural increase of honey bee colony
r_m	intrinsic rate of natural increase of mite
N	total population of colonies
N^*	equilibrium population of colonies
N_h	threshold population of colonies
N_{b_t}	population of individual honey bees in a colony at time t
N_{m_t}	population of mites in a colony at time t
t	time in terms of periods possibly intermitted by swarming
e	base of natural logarithm

MODELLING ANALYSIS

(*a*) *Colony to colony transmission*

Our aim is to describe as parsimoniously as possible the complex relationship that exists between the two organisms and determine whether or not stability is possible and not to contribute to parasitism theory as such. Transmission of mites between colonies occurs either through drifting, which is the accidental entry of a honey bee into the wrong hive and is equivalent to horizontal transmission, or through swarming, which is equivalent to vertical transmission (see figure 5*a*) (Royce & Rossignol 1990*b*); no other means of intercolony transmission are suspected. If colonies are considered as the units of reproduction, then the equations for the change in populations of infested colonies and populations of uninfested colonies would be (see table 1 for list of symbols):

$$\frac{dX}{dt} = a(X+Y) - bX - \beta XY - afY, \tag{1}$$

$$\frac{dY}{dt} = \beta XY - (\alpha + b - af)\,Y, \tag{2}$$

the sum of which is:

$$\frac{dN}{dt} = rN - \alpha Y. \tag{3}$$

The modified growth characteristic, ρ, of the population, assuming no immunity, will be:

$$\rho = r_c - \alpha, \tag{4}$$

and there will be a threshold, or critical density, of colonies for successful introduction of mites, given by:

$$N_h = \frac{\alpha + b - fa}{\beta}, \tag{5}$$

for both vertical (through swarming) and horizontal (through drifting) transmission. Now, if $\alpha > r_c$, then the host population will be regulated by the parasite at:

$$N^* = \frac{\alpha(\alpha + b - fa)}{\beta(\alpha - r_c)} \tag{6}$$

and if $\alpha < r_c$, then the population of honey bee colonies will grow exponentially at a rate given by $r_c - \alpha$.

The analysis is close to and based on that of Anderson & May (1981) and as such suggests that a stable equilibrium is achievable if we consider the host unit to be the colony rather than the individual honey bee. In the above equations, no density-dependent regulatory effect other than the disease is considered and density within colonies is not tracked.

(*b*) *Honey bee to honey bee transmission*

An important characteristic of colony growth is that, within a colony of honey bees, there is but a single reproductive female, the queen, and, although she is tremendously fecund, the population of worker bees within the hive increases linearly:

$$N_{b_{t+1}} = N_{b_t} + xt. \tag{7}$$

The mite population, however, has many reproductive females and its population increase would be exponential:

$$N_{m_{t+1}} = N_{m_t} e^{rm}. \tag{8}$$

A stable equilibrium is not excluded by a host linear growth rate; classical experiments in infectious disease epidemiology artificially provided such conditions and achieved regulation (Greenwood & Topley 1925; Fenner 1948). The agent involved in those studies was a microparasite, which means that parasite density was not a factor in morbidity and mortality; hosts were

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either infected or not. We do not know of such experiments with a macroparasite, such as the tracheal mite, nor do we know if such regulation is theoretically possible or not. Whatever the case may be, honey bee population within a colony is unlike most animal populations because none of the infected individuals are reproductive except for a single one, the population grows linearly and because honey bees have a built-in carrying capacity determined largely by the cavity size. Standard models do not take such conditions into account.

We suggest that in keeping with equations (1)–(6), the colony as a whole be regarded as the ecologically meaningful individual. If an infested colony is to have a probability of recovery $(1-f)$, however, it most likely, although not necessarily, possesses some form of 'resistance' or defences; other honey bee–mite relationships exhibit behavioural checks on parasitism (Peng *et al.* 1987; Burgett *et al.* 1990). Since the intracolony relationship appears to have some potentially strong destabilizing qualities, namely, time-delayed morbidity and reproduction within the reproductive host (the colony) (Royce & Rossignol 1990), we determined whether or not mites can self-regulate in the absence of swarming and, if not, whether or not swarming might regulate parasite density within a colony by interrupting the chain of transmission for a few weeks, allowing for a form of host-dependent cyclical intracolony stability.

EXPERIMENTAL RESULTS

Preliminary observations were made to establish the presence or absence of a relationship between mite and honey bee population levels within non-swarming colonies. Populations were monitored in two colonies, both of which were requeened in spring, but which had different success in rearing brood.

Colony no. 1 was requeened in May and the honey bee population increased linearly (figure 1) while the mite population in this colony showed an expected exponential increase. Colony no. 2 also was requeened in May but rejected the new queen after which natural queen replacement (supersedure) was allowed; the fluctuating honey bee population in this colony reflected the difficulty of requeening (figure 2). The

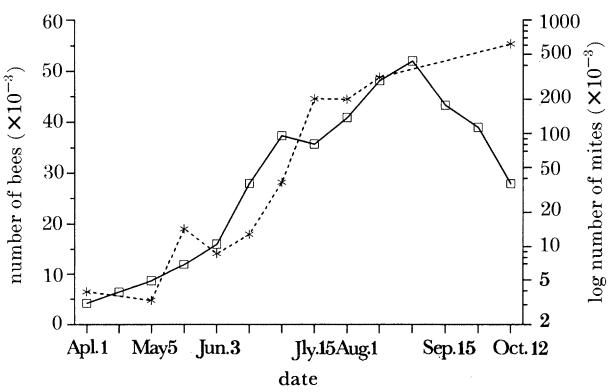

Figure 1. Population of honey bees (solid line) and of tracheal mites (dashed line) in normal colony (no. 1), as a function of time after founding.

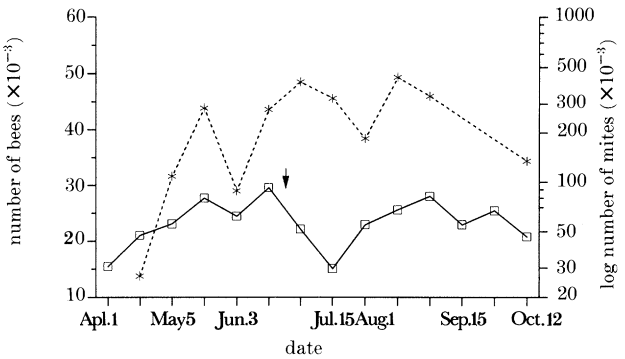

Figure 2. Population changes of honey bees (solid line) and of tracheal mites (dashed line) in colony (no. 2) that lost queen (at arrow), as a function of time after founding.

honey bee population never exceeded 30000 individuals and the mite population fluctuated more than in colony no. 1 and failed to reach as high a level (figure 2). Supersedure was accompanied by an absence of brood production, and mite levels decline following such an episode. Colony no. 1 died in the following winter, while colony no. 2 died the next spring. Mite densities therefore appeared to fluctuate with brood production and no apparent parasite self-regulation appears to occur when brood production is constant.

To determine if mite density was regulated by the absence of honey bee brood, brood production was manipulated experimentally by simulating swarming and delaying brood rearing for different periods of time. After a month, the number of adult and juvenile mites (juvenile being a resident stage and adult female being the invasive stage) were lower in the colonies that experienced the longer delay between swarming and brood rearing, although not necessarily lower than in the source colony (table 2). During this period, the ratio of juvenile mite density between the 2 and 11 day treatments was always less than unity (figure 3). The pattern for adult mites was similar. Mite density thus appears to be regulated by a delay in brood rearing.

A similar analysis of prevalence was done to assess the reliability of this criterion to assess mite-induced

Table 2. *Mite density in the original colonies (0) compared to that in swarms prevented from initiating a colony for either 2 or 11 days. Samples were taken 33 days after colony initiation. (* indicates sample taken after 19 days; queen was subsequently lost.)*

		No. of mites per 30 honey bees	
colony	delay (days)	female mites	total mites
A	0	178	622
	2	173	713
	11	65	487
B	0	227	466
	2	210	965
	11	110	414
C	0	112	386
	2*	89	322
	11	64	355

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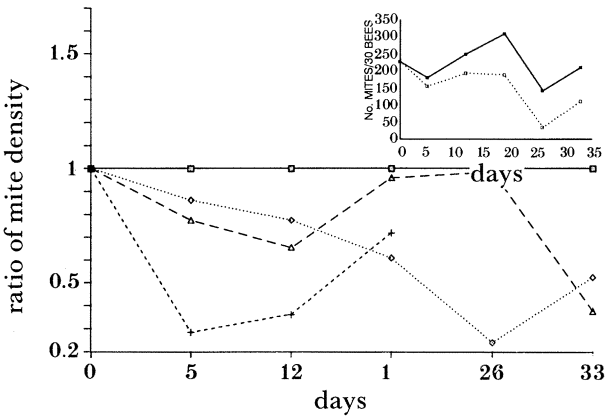

Figure 3. Mite density ratios of nuclear colonies prevented from brood rearing for 11 days to nuclear colonies from respective original colonies (A, long dashes; B, dots; C, short dashes) prevented from brood rearing for 2 days (solid line). Inset graph shows the actual conditions for the two respective nuclear colonies from parent colony B.

Table 3. *Mite prevalence (%) in the original colonies (0) compared to that in swarms prevented from initiating a colony for either 2 or 11 days. Samples were taken 33 days after colony initiation. (* indicates sample taken after 19 days; queen was subsequently lost.)*

colony	delay (days)	infested honey bees (%)
A	0	67
	2	83
	11	83
B	0	70
	2	97
	11	93
C	0	67
	2*	81
	11	80

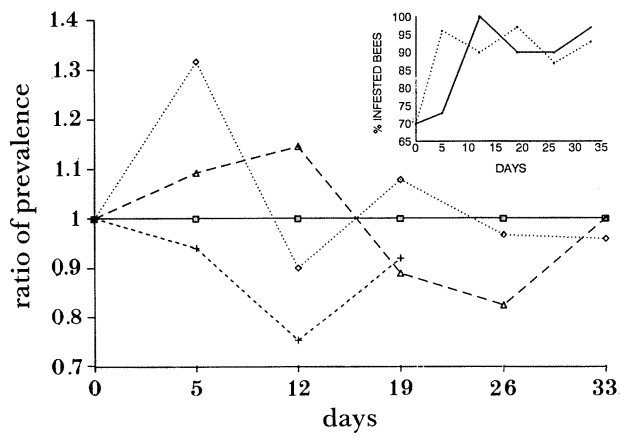

Figure 4. Prevalence ratios of nuclear colonies prevented from brood rearing for 11 days to nuclear colonies from respective original colonies (A, long dashes; B, dots; C, short dashes) prevented from brood rearing for 2 days (solid line). Inset graph shows the non-converted prevalence for the two respective nuclear colonies from parent colony B.

pathology. Overall, prevalence increased in all treatments (table 3) and no clear pattern developed between the two treatments (figure 4).

DISCUSSION

We theoretically show that intercolony transmission dynamics of the tracheal mite does not differ from a model of host–parasite dynamics with horizontal and vertical transmission (Anderson & May 1981). In the case of tracheal mites, horizontal transmission occurs through drifting of workers between colonies, vertical transmission occurs at the time of swarming and the 'individual' host is the colony, not the worker bee. We have assumed no recovery of non-swarming colonies because specific anti-mite defensive behaviour has not been documented for this mite, although such a parameter could easily be integrated. The rate of vertical transmission in infested colonies, fa, will have a major effect on the critical colony density needed to introduce successfully the parasite. In feral conditions, it is likely that drifting is rarer (β smaller) than in domesticated conditions, because of the great distance between colonies. Efficiency of vertical transmission, f, would then have to be high for the parasite to maintain itself.

The tracheal mite–honey bee relationship introduces a difficult problem common to eusocial insects, that is, the relevant host in the transmission model is the colony although it is the individual honey bee that is infested. The inherent malthusian characteristics of mite population growth could theoretically lead at some point to a high density within a colony, since the honey bee population within a colony grows linearly. Although local extinction undoubtedly occurs, it would appear that a colony infestation is fatal unless mite growth is controlled. The model also deals with the mite as a microparasite of colonies; although the mite is obviously a macroparasite on the individual honey bee worker (Royce & Rossignol 1990*a*), it does appear parsimonious, pending further work, to regard it as a microparasite of the colony as it reproduces at a high rate within the host colony and has a short life expectancy compared to the colony itself, both characteristics of microparasites.

We suggest therefore that swarming regulates mite density within a colony because it inserts a period during which young honey bees are not available. The experiments confirm that mite density indeed depends on the availability of emerging brood as a source of callow adult honey bee hosts and that an interruption in egg laying limits mite density and minimizes honey bee mortality. The loss of a queen or the splitting off and founding of a new colony, similar to swarming, may cause such an interruption. Reduction in overall density of mites from the initial conditions was observed and intensified proportionately with delay in brood rearing.

Splitting packages of honey bees from infested parent colonies is not identical to swarming since it was imposed on a colony rather than occurring naturally. The natural history of swarming (see Winston 1987), however, would suggest that mite limitation also might

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occur in both swarm and mother colony. Indeed, in preparing for swarming, honey bees prevent the old queen from laying eggs for about a week before swarming. The old queen then leaves with a swarm to start a new colony, and a new queen emerges soon after in the mother colony. The new queen then must mature, take nuptial flights and does not start laying eggs for at least another week. Therefore, swarming interrupts brood production for long periods in both the swarm and mother colony. Egg laying is probably interrupted for about two weeks, a longer period than in any of the simulated swarms, and one would expect mite density to be even more affected.

Observed increase in prevalence may be explained by an initial invasion of uninfested young honey bees taken along in the swarm and kept in contact with older infested honey bees. There thus appears to be no association between length of delay in brood rearing and prevalence, an important point since prevalence is often used to evaluate pathology. A paradox thus arises between a decreased mite density and increased prevalence. Parasite density has been the basis of estimating parasite pathology in host–parasite systems (Crofton 1971) and specifically in the tracheal mite–honey bee system (Royce & Rossignol 1990*a*). Although in most instances prevalence is associated with density and thus indirectly with pathology, this is an example where it is not. Estimates of parasite pathology based solely on prevalence (as in Gary & Page 1989) should be evaluated further.

An explanation for the sudden historical appearance of tracheal mites and the role of apicultural practices in this event can be tentatively proposed. When honey bees were first exploited systematically, the primitive techniques used would have had little effect on swarming (*a*). Because feral hives are widely dispersed (Bailey 1958; Free 1958; Jay & Warr 1984), horizontal transmission (β) was relatively rare although required if $f < 1$. This leaves vertical transmission (f) as the major pathway for parasite transmission (figure 5*a*). Overall, density of mites may have been low both per colony and per honey bee.

As human interaction grew beyond honey hunting, feral colonies were collected and kept close together in beeyards. The previously minor role of horizontal transmission gained importance since drifting was facilitated by close proximity and hive similarity (figure 5*b*). In Europe, for example, upright hives existed in apiaries before A.D. 1000 (Crane 1983), and in Britain, from 1300–1900, colonies were kept in woven 'skeps', with numerous such hives recessed into specially constructed walls. This new level of interaction may have brought about an increase in the proportion of infested colonies but not in mite density within an

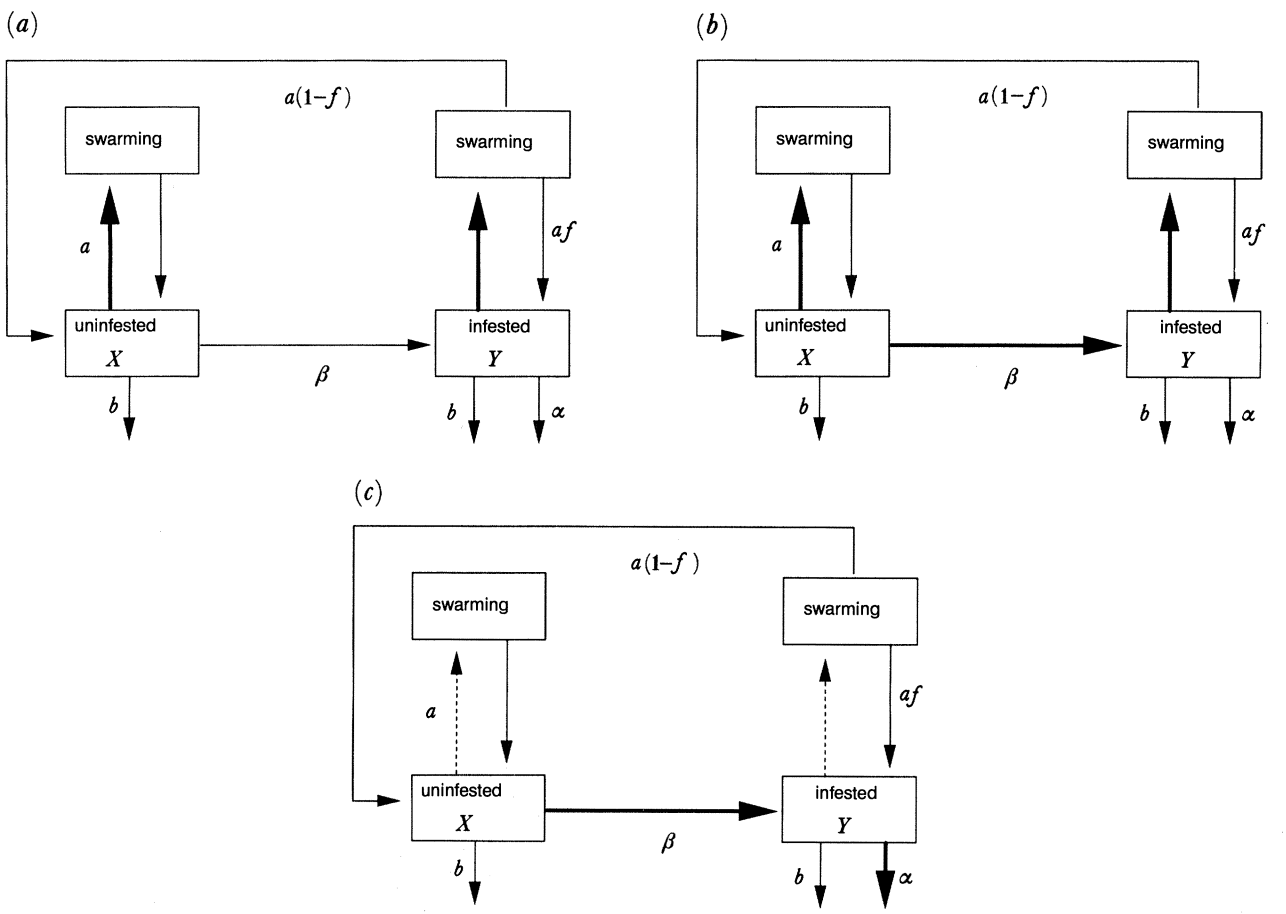

Figure 5. Flow charts of population dynamics of tracheal mites and honey bee colonies illustrating possible changes in the relative importance of parameters following human interference (symbols defined in table 1). The lines represent the flow of honey bee colonies between compartments while the parameters represent rates of flow. (*a*) No management, such as harvesting of feral colonies; (*b*) rudimentary management, such as skeps and beeyards; (*c*) modern management, such as Langstroth moveable frame hives.

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infested colony because swarming was not interrupted. Pathology (α), being a function of mite density in a colony (Royce & Rossignol 1990*a*), was probably still low; in any event, colonies were destroyed at the time of honey harvest (Crane 1983).

In the late 1800s, a new management technique was developed, namely, the Langstroth movable frame hive (see Naile 1976), which is now the standard technique of beekeeping in much of the world. Colonies in Langstroth hives could be prevented from swarming and be maintained from year to year. As a consequence, mortality due to parasitism may have increased because, as the honey bee population grew within a colony, mite density grew exponentially and may have reached a critical level of pathology not normally reached under previous conditions (figure 5*c*).

The sudden appearance of the Isle of Wight disease (Rennie 1921) may be explained as the interruption of swarming due to management techniques inherent to movable frame modular hives, introduced by Langstroth. Bailey (1964) had noted the coincidence between emergence of the disease and new management techniques on the Isle of Wight, and suggested that these innovations rather than the mite parasite were directly responsible for colony losses. We reach the same ultimate conclusion but propose a different mechanism, namely, that the technical progress of apiculture towards optimizing honey yield eventually broke down two crucial regulatory mechanisms of parasitism, in this case dispersion (β) and swarming (a), and led to devastating mite epizootics. In further support, it should be noted that the apicultural practice of colony splitting, reported from some areas, has been thought to reduce mite density (Eischen *et al.* 1988).

Based on our studies, we suggest that a standard population dynamics model may be applicable to the tracheal mite–honey bee relationship for intercolony transmission but that another mechanism, namely swarming, regulates intracolony densities. The experimental work confirms that swarming, because it causes brood unavailability, reduces mite density. By using the model's parameters, we propose a link between the sudden appearance of the mite early this century and the introduction of the Langstroth hive technique that results in reduced swarming.

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