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PARASITIC MITES OF HONEY BEES: Life History, Implications, and Impact

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■ **Abstract** The hive of the honey bee is a suitable habitat for diverse mites (Acari), including nonparasitic, omnivorous, and pollen-feeding species, and parasites. The biology and damage of the three main pest species *Acarapis woodi*, *Varroa jacobsoni*, and *Tropilaelaps clareae* is reviewed, along with detection and control methods. The hypothesis that *Acarapis woodi* is a recently evolved species is rejected. Mite-associated bee pathologies (mostly viral) also cause increasing losses to apiaries. Future studies on bee mites are beset by three main problems: *(a)* The recent discovery of several new honey bee species and new bee-parasitizing mite species (along with the probability that several species are masquerading under the name *Varroa jacobsoni*) may bring about new bee-mite associations and increase damage to beekeeping; *(b)* methods for studying bee pathologies caused by viruses are still largely lacking; *(c)* few bee- and consumer-friendly methods for controlling bee mites in large apiaries are available.

INTRODUCTION

The mites (Acari) that parasitize honey bees have become a global problem. They are threatening the survival of managed and feral honey bees, the beekeeping industry and, due to the role of bees in pollination, the future of many agricultural crops. *Acarapis woodi*, *Varroa jacobsoni*, and *Tropilaelaps clareae* are the main pests, but about 100 mostly harmless mite species are associated with honey bees (66, 167). The significance of bee mites to apiculture is emphasized by the publication of specialized books (35, 131, 135, 206) and by sessions devoted to these pests in apicultural, entomological, and acarological meetings.

Bee mites have greater dispersal potential than most other Acari, first through their hosts and then by humans who move bees primarily for commerce and

pollination. Two species of parasitic mites introduced into the United States in the early 1980s changed beekeeping profoundly by causing epidemic losses, ranging from 25% to 80% of managed colonies between 1995 and 1996 (79). Also, feral bee colonies are virtually gone in regions where both mites occur (115). This void of wild bees has been especially noticed by homeowners and growers who had relied on bees to pollinate their crops. Further consequences of these introduced mites are: *(a)* beekeepers and bee breeders going out of business, *(b)* fewer managed colonies, *(c)* increased demand and cost for leasing hives, *(d)* increased incidence of pathogens in colonies, and *(e)* problems with some commercially reared queens and bees (32).

THE HIVE AS A HABITAT FOR MITES

The biology of parasitic bee mites, and hence control, can best be understood by reference to their hosts. The honey bee, *Apis mellifera*, is a social insect that lives in colonies. Each colony has two female castes: the single queen and the workers who may number 20,000 to 50,000 per colony. Colony life is regulated by an array of pheromones, emitted mainly by the queen (81). The colony also has males, called drones, who serve mainly to inseminate the queen during her mating flight.

Characteristic of social insects, a division of labor exists in the colony. Young workers nurse and groom the queen and brood. Older workers forage for pollen, nectar, propolis, and water, and defend the colony. Nest homeostasis is maintained by the workers, who keep the temperature and humidity nearly constant despite external conditions. An abundance of proteinaceous foods (pollen) and carbohydrates (honey) are usually available. Total development of the queen requires about 16 days, that of workers 21 days, and the drones 24 days. The queen can survive for several years, but workers and drones usually live only a few weeks. During cold periods the workers, who may then survive for several months, cluster around the queen to incubate eggs laid during this time.

Non-bee invaders that circumvent colony defenses will thus benefit from the favorable conditions of the hive environment for their own development and reproduction. Most of the successful invaders are mites, and they make up the largest and most diverse group of honey bee associates. Different honey bee species have varied nesting habitats, and their relationships with acarine parasites are discussed in this review.

Hive mites were placed in four groups (67), namely scavengers, predators of scavengers, phoretics, and parasites. Occasional visitors, found in all four of these groups, will not be discussed. For simplicity, we divide the bee mites into nonparasitic and parasitic groups.

NON-PARASITIC BEE MITES

Three common suborders of mites associated with bees are the Astigmata, Prostigmata, and the Mesostigmata. Many astigmatic mites live on the hive's floor, feeding on bee debris, dead insects and fungi. *Forcellinia faini* (Astigmata), a common, whitish, slow-moving scavenger initially described in Puerto Rico, was abundantly collected in hive debris in northern and southern Thailand (77). A representative of the Prostigmata is the tarsonemid *Pseudacarapis indoapis* (Lindquist), a probable pollen feeder, which is apparently restricted to *Apis cerana* (122a).

Melichares dentriticus (Mesostigmata), a cosmopolitan predator on scavenger mites, is common in stored products (105), while members of *Neocypholaelaps* and *Afrocypholaelaps* live in flowers. They feed on the pollen of subtropical and tropical trees and are phoretic on bees. Several dozen *Afrocypholaelaps africana* were found on individual bees visiting mangrove umbels in Queensland (187). Mites dispersing on *A. mellifera*, mostly as egg-bearing females, board and depart the bees via the tongue. The bees did not appear to be annoyed by these mites, nor were their foraging activities disrupted. Ramanan & Ghai (161) reported that one to 400 *A. africana* occurred on individuals of *A. cerana* in India. As bees return to the hive, mites disembark, roam on the combs and subsist on the pollen.

Melittiphis alvearius has been found in hives and on bees in various parts of the world (57). Serological procedures demonstrated that, contrary to views formerly held, this mite is not a predator, but feeds on stored pollen (90).

PARASITIC MITES

Three species of parasitic bee mites are of economic importance due to their destruction of honey bee colonies worldwide. We will focus on the tracheal, varroa, and tropilaelaps mites, as well as provide some information on lesser known mites on other *Apis* species.

Tracheal Mites

The honey bee tracheal mites (HBTM) *Acarapis woodi* (Rennie) live inside the tracheae and air sacs of adult bees. Mites in tracheal systems seem to be rare in arthropods and not well studied. Approximately 15 species are known to parasitize members of Hymenoptera, Orthoptera, Lepidoptera, and Hemiptera (176).

HBTM were first observed earlier this century, when bees on the Isle of Wight were dying from an unknown disease. The die-off reached epidemic levels between 1904 and 1919 (36). At first thought to be caused by a bacterium, the disease was identified by Zander (214) as *Nosema apis*, a protozoan parasite of the bee's alimentary tract. As the disease spread throughout Europe (157), a mite living in the bee's tracheal tubes was discovered and named *Tarsonemus woodi*

(165, 166) [later renamed *Acarapis*, from *Acarus*, mite, and *apis*, bee (102)]. The disease was then called Acarine or Isle of Wight disease. The real cause of the loss of colonies during this time is still unknown and may have been several diseases or other factors causing the symptoms (207).

The identification of *A. woodi* in Europe led the United States Congress in 1922 to prohibit importation of all bees (158) and to examine bees for evidence of mites from apiaries in many states. Other species of *Acarapis*, namely *A. externis* Morgenthaler and *A. dorsalis* Morgenthaler (but not *A. woodi*), were found as a consequence of this intensive sampling conducted by the USDA honey bee laboratories (201–203). The reasons for the HBTM's absence in North America remain a mystery: At least eight honey bee subspecies had been imported to the New World since the 1600s (188). HBTM is now thought to be worldwide (wherever European bees have been introduced) except in Sweden, Norway, Denmark, New Zealand, and Australia, and in the state of Hawaii.

South America allowed bee importation and in 1980, *A. woodi* was reported in Colombia (133). The mites moved northward and reached Texas by 1984; they were later reported in seven states that same year. This fast spread was greatly facilitated by the extensive trucking of bee colonies from southern states northward for pollination and for sale as package bees and queens.

The HBTM is responsible for significant colony losses throughout North America. Reports of losses as great as 90% were recorded just two years after initial discovery (176). A heavy HBTM load causes diminished brood area, smaller bee populations, looser winter clusters, increased honey consumption, lower honey yields (10, 68, 140, 147, 170), and, ultimately, colony demise. In temperate regions, mite populations increase during winter, when bees are confined to the hive, and decrease in summer when bee populations are highest (178). In subtropical climates, the cycle is similar (192), even though bees are not so confined. Unfortunately, the introduction of varroa mites has overshadowed the impact HBTM has on bees.

Recent Evolution of HBTM? Eickwort (66) speculated that *Acarapis* might have evolved from saprophagous or predatory mites and their appearance in hives may have been due to the nesting behavior of Apinae, which provided habitats for these mites. The evolutionary history of *Acarapis* suggests that they may have been pre-adapted for this way of life. All members of the tarsonemid subfamily Acarapinae occur and feed on insects; the related Coreitarsonemini are parasitic in the thoractic odoriferous glands of coreid bugs (122a).

Morse & Eickwort (139) hypothesized that the invasion of tracheae by *A. woodi* was a recent evolutionary event, believed to have begun in England around the year 1900. HBTM evolved from one of the closely related external *Acarapis* species, probably *A. dorsalis*. Their argument was based on the (apparent) formerly restricted distribution of HBTM and on the incipient resistance to this pest in North America. Initially, the mite was believed to occur only in England, Switzerland, and Russia, whereas *A. dorsalis* and *A. externis* are worldwide in

distribution. If the pest had been present in England in former times, why was it not noted? And if it was more widely distributed, what prevented it from damaging honey bees in other regions also? Finally, the observation that honey bee populations in North America show a wide range of susceptibilities, as well as resistance, to *A. woodi* could indicate quite recent exposure to the pest (139).

Acceptance of this theory presents many difficulties, such as a rather truncated evolutionary time scale and the differences between this species and *A. dorsalis*, its postulated progenitor. Table 1 lists some of these differences (based on 59, 65, 169). Such an array of changes could hardly have evolved in the brief span of a century. The discovery of HBTM in many regions of mainland Europe, where migratory beekeeping and trade in bees were limited, soon after its initial description (10) also argues against *A. woodi*'s recent evolution.

Why then was the damage attributable to *A. woodi* not noticed earlier? Nowadays beekeeping is much more productive than in the past, and factors that reduce colony yield are more critically observed. Minor diseases and pests, which cause only marginal reductions in bee longevity and yield, were probably masked in the past by more severe mortality factors. The great advances in bee health and management, however, raised the awareness of beekeepers toward pests such as *A. woodi*, whose ravages did not decline through modern control methods. The pest might have been overlooked, even in England, had not the outbreak of the Isle of Wight disease necessitated the intensive examinations of bees. Eickwort (66) suggested a similar scenario in regard to the discovery of *A. woodi* in Europe, where it was noticed only after an epidemic of viral diseases led to bee dissections.

Another explanation for their ''sudden appearance'' concerns beekeeping practices, which have changed significantly in the past century. Straw skep hives were

TABLE 1 Differences between *Acarapis woodi* and *Acarapis dorsalis*.

Character	*Acarapis woodi*	*Acarapis dorsalis*
Site of reproduction	Inside body, in tracheae	Outside bee, in dorsal groove and at wing bases
Adult movement	Remains in one host bee except for questing adults	Moves to other host bees
Maximal attachment to bees	1–6 days	12 days
Season of maximum bee infestation	Autumn to spring	Spring to summer
Length of development	264 hours	216–240 hours
Female anterior median apodeme	Incompletely developed, not joining transverse apodeme	Developed, joining transverse apodeme
Female posterior margin of coxal plate IV	Shallowly notched	Deeply incised
Male solenidion on tibia I	Slightly club shaped	Not club shaped
Number of setae on male femur-genu-tibia of Leg I	4–4–7	3–3–6

used by early beekeepers, and the bees tended to swarm frequently (172) or were killed to harvest honey. Such methods resulted in smaller, more remote apiaries that kept bees apart, admixing neither bee nor mite strains. Modern beekeepers maintain colonies in wooden boxes, in greater numbers, and move hives frequently to take advantage of bee forage or for pollination. Such practices result in large numbers of perennial hives from diverse regions commingling and rapidly infecting all with the mites and diseases.

Life Cycle Like other members of the prostigmatic Heterostigmata, *A. woodi* has a foreshortened life cycle with only three apparent stages, namely egg, larva, and adult. However, the mite has an apdous nymphal instar that remains inside the larval skin (122a). Males complete their development in 11 to 12 days, females in 14 to 15 days. A generation is thus raised in two weeks, explaining the rapid population growth of the HBTM (for detailed biological information see 155 and 207). Mites feed on bee hemolymph, which they obtain by piercing the tracheae with their closed-ended, sharply pointed stylets that move by internal chitinous levers (101). Once the bee trachea is pierced, the mites' mouth, located just below the stylets, is appressed to the wound and the mites suck host hemolymph through the short tube into the pharynx.

All instars live within the tracheae, except during a brief period when adult females disperse to search for new hosts (181, 194). Dispersing female mites (mated) are attracted to air expelled from the prothoracic (first thoracic) spiracle of young bees (101), as well as to specific hydrocarbons from the cuticle of callow (less than four days old) bees (156). Because older bees might not live long enough for the HBTM to complete its cycle, mites are less attracted to older bees.

Once a suitable host is found, preferably a drone (171), the female enters its spiracle to reach the tracheae and lay eggs. Tracheal mites and their eggs also occur at a lower rate in the air sacs of the bees' abdomen and head, and externally on the wing bases (91). In these alternate locations, neither their effect on the host nor their fate is known.

The mite's small size is critical to its survival (females measure 120 to190 μm long by 77 to 80 μm wide and weigh 5.5 $\times$ 10-4 mg; males 125 to 136 μm by 60 to 77 μm and weigh 2.61 $\times$ 10-4 mg; 176). The tiny mites can hide under the flat lobe that covers the bee's first thoracic spiracle (access the main tracheal trunk), which many individuals can thus occupy.

Mites begin to disperse by questing on bee setae when the host bee is more than 13 days old, peaking at 15 to 25 days. *A. woodi* is vulnerable to desiccation and starvation during this time outside the host, and survival depends on the ambient temperature and humidity as well as on its state of nourishment (86, 194). A mite can die after a few hours unless it enters a host (101). Mites are also at risk of being dislodged during bee flight and grooming.

Diagnosis HBTM are not visible to the naked eye, making diagnosis difficult. Consequently, beekeepers often use unreliable bee stress symptoms, which

include dwindling populations, weak bees crawling on the ground with disjointed hind-wings (called K-wings) and abandoned, overwintered hives full of honey.

The only certain way to identify mite infestation is to dissect the tracheae of bees and visualize the parasites. Bees are collected in winter or early spring, when HBTM populations are highest; fewer mites are found in the summer, due to the dilution effect caused by the rapid emergence of many young bees.

Nine methods for diagnosing HBTM are described (190). Most involve some form of dissection, for which a dissecting microscope (at 40X to 60X magnification) and a pair of fine jeweler's forceps are necessary. Because drones are favored by HBTM and are bigger than workers, they should be collected and dissected first. Queens, even those commercially reared, often have HBTM (27, 151). Camazine et al (32) found that infested queens weighed less.

Fresh or frozen specimens are preferred for dissection over alcohol-preserved material, as alcohol darkens the tissues, making visualization of mites difficult. Tomasko et al (198) developed a sequential sampling method to determine how many samples are needed for accurate measurements. A time-consuming method of cutting thoracic discs (190) and staining the tracheae (150) can be used, but this method requires double handling and identification is delayed. Camazine (30) used a kitchen blender to pulverize bee thoraces, allowing the air-filled tracheae to float, then sampled the surface debris for mites.

Serological diagnoses using ELISA techniques have been developed (78, 93, 159, 160). The visualization of guanine, a nitrogenous waste product not excreted by bees, was advocated (144). Removing the head, pulling off the flat lobe covering the first thoracic spiracle and extracting the tracheal tube (193) is faster and provides immediate diagnosis once mastered. If live bees are used, dead versus live mites are counted when testing acaricides (68, 194).

Controlling Tracheal Mites

Chemical The overriding constraints for chemical control are that the chemicals must be effective against the target and harmless to bees, and they must not accumulate in hive products. Because bees and mites are both arthropods, many of their basic physiological processes are similar, narrowing the possibilities for finding suitable toxicants. Bees are extremely sensitive to accidental poisoning by many of the common agrochemicals (8). To control HBTM, the material must reach the bee tracheae via a volatile compound, be inhaled by the bee, and be lethal only to the parasite. A single registered treatment in the United States is pure menthol crystals, originally extracted from the plant *Mentha arvensis*. Each two-story colony requires 1.8 ounces (50 g) or one packet for two weeks. However, in cold conditions menthol sublimation is ineffective because an insufficient amount of vapor is released from the crystals; conversely, at high temperatures the vapors may repel bees from the hive.

An effective pesticide, Miticur (sold as Amitraz) has been withdrawn from the U.S. market but still is used in Israel (40). Formic acid (104), a potentially effec-

tive agent, though caustic to humans, will soon be released in the United States; it is used in Canada.

Cultural An alternate, environmentally safe control is to apply a vegetable shortening and sugar patty at peak mite populations. A quarter-pound (113 g) patty, placed on the top bars at the center of the broodnest where it comes in contact with the most bees, will protect young bees (which are most at risk) from becoming infested. The oil appears to disrupt the questing female mite searching for a new host (181). Because young bees emerge continuously, the patty must be present for an extended period. The optimal application season is in the fall and early spring, when mite levels are rising.

Resistant Bees Several lines of bees resistant to tracheal mites have been developed, starting with Brother Adam's Buckfast bees (23). Some of this stock is commercially reared and sold to beekeepers. Resistance to HBTM seems to be accomplished by the increased grooming behavior of bees (42, 43, 122, 145).

Varroa Mites

The varroa mite, *Varroa jacobsoni (Oudemans)*, is currently considered the major pest of honey bees in most parts of the world. Only Australia, New Zealand and the state of Hawaii remain free of this pest. The pathology it causes is commonly called varroasis (also seen as varroatosis or varrosis). Initially discovered in Java (148), varroa was originally confined to Southeast Asia where it parasitizes the Asian honey bee, *Apis cerana*. This bee has probably coevolved with the parasite, which adapted to keep the mite under control (163, 182).

A post–World War II increase in international travel and commerce has facilitated the worldwide dispersal of varroa (39). Once established, the mite spreads on drifting, robbing, and feral bees, on swarms, and are even reported on wasps (87) although this may an artifact of wasps robbing infected bee colonies. De Jong et al (53) documented the history of the spread of varroa (see 95 for the first reliable map of varroa worldwide distribution). An animated map of the spread of varroa is found on the Worldwide Web at http: //www.csfnet.org/image/animap2.gif. Varroa became an economic concern in Japan and China in the 1950s and 1960s, in Europe in the late 1960s and 1970s, and in Israel and North America in the 1980s.

Life Cycle Adult females measure 1.1 mm long $\times$ 1.6 mm wide, weigh approximately 0.14 mg (176) and are a reddish-brown color. The ovoid males are much smaller, about 500 μm wide, and are light in color.

Several key morphological features help make varroa a successful ectoparasite. It can survive off the host for 18 to 70 hours, depending on the substrate (46). The female's chelicerae are structurally modified, the fixed digit is lacking, and the moveable digit is a saw-like blade capable of piercing and tearing the host's

integument (56). The mite's body is dorsoventrally compressed, allowing the mite to fit beneath the bee's abdominal sclerites, thus lessening water loss from transpiration (213). Its hiding there reduces varroa's vulnerability to grooming and to dislodgment during host activity.

Female varroa are often found on adult bees, which provide for dispersal (phoresy) and serve as short-term hosts. Varroa prefers young ''house'' bees to older workers, probably because of the lower titer of the Nasonov gland pheromone geraniol, which strongly repels the mite (103). The mite pierces the soft intersegmental tissues of the bee's abdomen or behind the bee's head, and feeds on the hemolymph. When in an actively reproducing bee colony, the mite disembarks and seeks brood cells containing third-stage bee larvae. Varroa, which prefers drone larvae but also invades workers' cells, is attracted to fatty acid esters, which are found in higher quantities on immature drones than on workers (119). Other known attractants are the aliphatic alcohols and aldehydes from bee cocoons (64) and perhaps the larger volume of drone cells.

Varroa enters the prepupal cells one to two days prior to capping and hides from the nurse bees by submerging in the remaining liquid brood food, lying upside down. The mite's modified peritremes protrude snorkel-like out of the fluid surface, enabling them to respire (63). The female remains concealed until the brood cell is capped. To keep from becoming trapped, the female attaches herself to the bee larva as it spins its cocoon. Once the prepupa is formed, the mite begins to feed at a site located on the prepupa's fifth abdominal segment.

Varroa produces its first egg 60 hours after the cell is sealed (110). The first egg is usually a haploid male, and the subsequent female eggs are laid at 30-hour intervals. Mites go through the following instars: pharate larvae, mobile protonymph, pharate deutonymph, mobile deutonymph, pharate adult, and adult (50, 63). Mobile nymphs actively feed and grow while pharate instars are quiescent—all active postembryonic instars are eight legged. The foundress mite keeps the feeding site open to allow her offspring to eat and will even push away the prepupa's posterior legs to keep the site exposed (62).

Ideal temperatures for optimal varroa development correspond to the ideal temperatures of drone brood (118, 120). Young females mature in 6.5 to 6.9 days (50), emerge with the callow bee, and may be phoretic for a time (four to five days on average) on other bees before invading new brood to repeat the cycle. Each varroa female may undergo two to three reproductive cycles (128). If mites invade drone cells, each foundress produces on average 2.6 new female offspring. In worker cells, the mean is 1.3 (186), although the numbers may vary considerably depending on the number of foundress mites in the cell, their fertility level, and/or the race of bees.

Fecal pellets (mostly guanine) are deposited on the cell wall and act as an aggregation site for immatures and a meeting place for mating (62). Males mature in 5.5 to 6.3 days, then mate frequently with each emerging sister mite, in succession, using specially modified chelicerae to inject sperm into the pair of induction pores between the bases of legs III and IV in females (62). Unmated females

produce only male offspring. The total life cycle of the male is completed in the brood cell, after which it dies.

Recent molecular techniques have demonstrated that *V. jacobsoni* most likely is a complex of species represented by at least five sibling species (6a), only one of which has spread from *A. cerana* to *A. mellifera*, causing the enormous bee losses. Morphological differences have been recorded between these species and the species appear to be reproductively isolated from one another. Hence the varroa on *A. mellifera* could soon be renamed. The name *Varroa destructor* has been proposed (D Anderson, personal communication) and if several species and strains are masquerading under the name *V. jacobsoni*, and they possess different levels of virulence, the survival of untreated bee colonies despite varroa presence in South America may be explained (6, 48, 49, 52). Martin & Ball (127) reported differences in mite virulence and virus associations on honey bee survival in the United Kingdom. Other factors contributing to bee resistance may be climatic differences (51), the behavior and life cycle of the Africanized honey bee (31) *A. m. scutellata* (e.g. shorter post-capping period of bee brood), or other as yet undiscovered factors or combinations of factors.

Symptoms

Varroasis symptoms can be confused with other disorders, and even with pesticide poisoning. Here are the most notable symptoms: *(a)* Pale or dark reddish-brown mites are seen on otherwise white pupae. *(b)* Colonies are weak with a spotty brood pattern and other brood disease symptoms are evident. *(c)* The drone or worker brood has punctured cappings. *(d)* Disfigured, stunted adults with deformed legs and wings are found crawling on the combs or on the ground outside (50). Additionally, bees are seen discarding larvae and pupae and there is a general colony malaise, with multiple disease symptoms. Because mite populations increase in proportion to the available bee larvae, varroa can quickly overrun a colony and often colonies are dead by the fall.

Interrupted brood rearing during the winter slows the population increase of varroa in temperate regions, but in warmer climates, colonies can be destroyed within months (88). Treated apiaries can still perish if the beekeeper is not diligent; reinfestation occurs due to the robbing of varroa-infested and weakly defended colonies.

Detecting Varroa

Observation of Brood Mites can be detected by pulling up capped brood cells using a cappings scratcher (with fork-like tines); varroa appears as brown or whitish spots on the white pupae. Guanine, the fecal material of varroa, can be seen as white spots on the walls of brood frames in highly infested colonies (76a).

Ether Roll About 100 to 200 bees are collected in a clear glass jar and sprayed briefly with engine starter fluid; the jar is shaken to dislodge mites and then rolled

so mites adhere to the sides. Adding alcohol or soapy water to the jar and re-agitating the contents will displace the remaining mites. Pouring the liquid through coarse mesh will strain out the bees, after which the mites can be fine-filtered and counted.

Sticky Board A white paper or plastic sheet covered with petroleum jelly or another sticky agent is placed on the bottom board of a colony and the hive is smoked with pipe tobacco in a smoker. After closing the hive for 10 to 20 minutes, the board is removed and the mites counted. Alternately, a sticky board by itself can be left in place for one to three days. This last method is more efficient than sampling brood or bees and is widely used as a research tool to monitor mite levels (82). Special sticky boards (145a) have been developed to ease the task of counting mites.

Economic injury levels and economic damage have not been reliably calculated for varroa. Delaplane & Hood (54) found late-season drops on a sticky board of more than 100 mites per day sufficient to justify treatment in Georgia. However, other researchers find this number too high (D Caron, personal communication). Defining a reliable ratio of critical infestation of mites to bees is problematic. If varroa is present in high numbers early in the season, treatment should be immediate. Finding mites during a honeyflow precludes chemical applications for treatment (see next section).

Control of Varroa

Chemical While long-range, non-chemical controls are vigorously being sought, beekeepers need immediate relief from existing mite infestations. Fluvalinate (Apistan®), a pyrethroid, is currently the only U.S.-registered pesticide for varroa control. Coumaphos (Bayer Bee Strips or CheckMite), an organophosphate, received Section 18 registration in Florida in 1999; it is granted this status in many other states as well. Section 18 allows a limited, short-term, single-use application. Coumaphos is the only product known to control the small hive beetle, *Aethina tumida*, introduced from South Africa and identified in the United States in 1998. These new strips will also be used on fluvalinate-resistant varroa populations. Both chemicals are applied as pesticide-impregnated plastic strips, which are hung between frames of bees in a hive. Treatment time is in the spring and again in the fall as needed, and only when there is no honeyflow. Applied in this manner, fluvalinate is released slowly and dispersed by adult bees (26).

These chemical options for varroa pose a serious problem because repeated exposure to the same pesticides select for resistant mites (89). Recent reports of fluvalinate-resistant mites have surfaced in Italy (124, 125, 134), France (38) and Israel (R Mozes-Koch, A Dag, Y Slabezki, H Efrat, H Kaleb and BA Yakobson, unpublished data) and some U.S. states (70, 71, 74, 152, 153). Coumaphos resistance is also reported in Italy (204) and the United States and recent studies (73)

found low levels of resistance to Amitraz (another chemical previously used for mite control) in one beekeeping operation that exhibited fluvalinate resistance. Cross- or multiple-resistance studies need to be done. This resistance crisis is being compounded by contamination of hive products (205). In addition, drone survival is found to be lower in both varroa-infested colonies and colonies treated with fluvalinate (166a), which may also affect their mating ability. Poor quality or low numbers of drones result in poorly mated queens.

Organic Acids Formic acid kills varroa and HBTM (104), but is temperature dependent and dangerous to humans. When used with absorbent paper over the top bars, the evaporating fumes kill HBTM and varroa (82). In 1998, the USDA-ARS licensed a U.S. bee supplier to produce formic acid gel packs, which should be available soon. Other organic acids, such as oxalic and lactic, are used in Europe (22a) and are applied in sugar syrup trickled on bees. These acids require broodless bees and may cause bee mortality (22a).

Essential Oils Another approach is the use of volatile plant essential oils to control bee mites (29, 37, 84, 111) and other bee diseases (34, 80, 132). Many beekeepers are already experimenting with such ''natural'' products (179), but plant oils are complex compounds that may have unwanted side effects on bees and beekeepers (183), and could contaminate hive products.

Biological/Cultural Controls

Other methods have been used to control mites, but most are too labor intensive and impractical in large apiaries. Used in combination with or in an integrated pest management (IPM) project, they may be helpful.

Smoke and Dropping Mites Partial control in lightly infested apiaries can be obtained with tobacco smoke or smoke from other plant materials that cause mite knockdown (69, 72). Smoke dislodges mites and can be used periodically to remove those that subsequently emerge from brood cells. A sticky board used in conjunction with smoke traps mites dislodged by the smoke. Pettis & Shimanuki (154) found varroa that dropped to the bottom board of a hive were more likely to remain there unless a bee passed within seven mm of it. Using a screen to separate fallen varroa from bees may help keep mite levels lower.

Traps Because varroa prefer drones, combs of drone brood can be used to attract, trap, and remove mites by cutting out drone brood (184, 185). Worker brood can also be removed (83). Drone brood can also be cut out of frames (83, 184, 185). Also, caging the queen of *A. cerana* for 35 to 40 days and separating the brood frames helped interrupt the brood/mite cycle (75) in *A. cerana*.

Heat Another method employs heat: The mite succumbs at or around 111° F (44° C), whereas sealed brood survives. Using a combination of these methods, along with a lactic acid treatment, mite numbers were reduced in Denmark (22).

Resistant Bees Attributes that enhance honey bee tolerance to varroa are reviewed (24). Some beekeepers let all susceptible colonies die and then rear queens from the survivors to head new colonies. Untreated Africanized colonies were maintained in Arizona for several years with few tracheal and varroa mites; resistant mechanisms were not discussed (76).

Hygienic Behavior and Grooming Bees will open capped brood cells and remove dead or dying brood. Such hygienic activity (19, 20, 195) reduces the mite levels in untreated colonies, which require less chemical treatment to manage varroa. Bee grooming (both autogrooming and allogrooming) has been observed in bees infested with mites (25). Defensive behaviors against varroa in races of *A. cerana* were studied (163, 182) and grooming is an important component in mite reduction. However, grooming is highly variable in *A. mellifera* (24). Bees will remove mites from each other and some even kill them using their mandibles (149). Unfortunately, this trait (mite biting) may not be heritable in some European bee stock (96, 97).

Length of Post-Capping Stage The pupal period influences the number of mites completing development. Shortening this time results in fewer varroa reaching maturity; if the capped cell stage is reduced by only six hours, fewer immature mites will become adults. Two African bee races have a heritable (worker) post-capping period of only 10 days (138), whereas European races require 11 to 12 days. Some researchers (51, 136) suggest climate plays a more important role, as this trait is difficult to maintain in *A. mellifera* colonies in northern regions.

Brood Attractiveness The larvae of European bees are highly attractive to varroa; the ARS-Y-C-1 strain (*A. m. carnica*) was less attractive than other bee stocks (47). Differences in chemical components or levels in the brood may be the reason, but these possibilities were not tested.

Low Mite Fecundity At times, varroa mites do not reproduce (31), die without producing offspring, or get caught in the cocoons of bee pupae and die. Some of these traits are genetic (96, 97). Harris & Harbo (99) have further classified mite fecundity: live mites that do not lay eggs, live mites delayed in laying eggs, and mites that die before oviposition. Mites with lower or no fertility were found to have fewer (or no) spermatozoa in their seminal receptacle.

Queens selected for suppression of mite reproduction trait (SMRT) had reduced mite fecundity even after these queens were placed in susceptible colonies (J Harbo & J Harris, unpublished data). This trait, used conjointly with

hygienic behavior and other IPM methods, may help solve the bee/varroa problem in the next decade (180).

Tropilaelaps clareae

This mite belongs in the mesostigmatic family Laelapidae, which has members that are mammalian parasites. *Tropilaelaps clareae*, originally obtained from a field rat (55) from the Philippines, normally occurs on *A. dorsata*; this mite also parasitizes *A. mellifera* (117). Currently, *T. clareae* is restricted to Asia, from Iran in the northwest to Papua New Guinea in the southeast (129). A single, alarming report of this mite in Kenya (116, 130) has not been repeated.

Life Cycle The life cycle is similar to that of varroa, but is shorter. Females are medium sized (1030 μm long × 550 μm wide), elongated and light reddish-brown; males are similar but less sclerotized. The foundress mites place three to four eggs on mature bee larvae shortly before capping and the progeny, usually a (first) male and several females feed only on bee brood. Development requires about one week and the adults, including the foundress mite, emerge with the adult bee and search for new hosts. The shortened life cycle, as well as a very brief stay on adult bees, explains how *T. clareae* populations can grow faster than those of varroa. *T. clareae* also out-competes the latter when both infest the same colony of *A. mellifera* (191). Nevertheless, populations of both mites can survive in the same apiary for 12 months, probably because their niches are not completely congruent (164).

Like varroa, the female mites are dispersed by bees, but phoretic survival is of short duration because the unspecialized chelicerae of tropilaelaps cannot pierce the integument of adult bees. Gravid female mites die within two days unless they deposit their mature eggs (208, 210).

The mouthparts are stubby, with an apically bidentate fixed upper digit, and a longer, unidentate and pointed moveable digit. This piercing-grasping structure is more suitable to piercing soft brood tissue, rather than the tearing-sawing type of varroa. This implies that tropilaelaps can feed only on soft tissues, such as honey bee brood (94).

Symptoms Irregular brood pattern, dead or malformed wingless bees at the hive's entrance, and the presence of fast-running, brownish mites on the combs, are diagnostic for *T. clareae* (50). The faster development rate make *T. clareae* more dangerous to European honey bees than varroa (209) where they cohabit.

Treatment Fluvalinate controls *T. clareae* (126), as do monthly dustings with sulfur (9) and treatment with formic acid (85) or with chlorobenzilate (208). The inability of this mite to feed on adult bees, or to survive outside sealed brood for more than a few days, is being used as a nonchemical control method (209, 211).

OTHER PARASITIC BEE MITES

Most parasitic bee mites (with the exception of *Acarapis*) are in the tribe Varroini (or Group V) in the family Laelapidae (see Table 2; 34a).

Euvarroa

Euvarroa sinhai is a parasite of *A. florea*, occurring in Asia from Iran through India to Sri Lanka. The mite develops naturally on the capped drone brood (142) but has been reared in the laboratory on *A. mellifera* worker brood (141). Development requires less than one week, and each female produces four to five offspring. Drones as well as workers are used for dispersal. The female mite overwinters in the colony, probably feeding on the clustering bees. Colony infestation by *E. sinhai* is somehow hindered by the construction of queen cells (1) and its population growth is inhibited in the presence of *T. clareae* and of *V.*

TABLE 2 Mesostigmatic mites parasitizing bees, arranged according to host bee species (113). Asterisk (*) denotes mites believed to be originally associated with the particular bee species.

***Apis* species**	**Mites**	**Source**
andreniformis	*Euvarroa sinhai*	60
	*Euvarroa wongsirii**	121
cerana	*Tropilaelaps clareae* (?)	60
	*Varroa jacobsoni**	
	*Varroa underwoodi**	60
dorsata	*Tropilaelaps clareae**	61
	*Tropilaelaps koenigerum**	60
florea	*Euvarroa sinhai**	
	Tropilaelaps clareae	60
koschevnikovi	*Varroa rindereri**	45
	Varroa jacobsoni	60
laboriosa	*Tropilaelaps clareae*	
	Tropilaelaps koenigerum	60
mellifera	*Euvarroa sinhai*	114
	Tropilaelaps clareae	60
	Varroa jacobsoni	60
nigrocincta	*Varroa underwoodi*	7
nuluensis	*Varroa jacobsoni*	60
	Varroa near *underwoodi*	44

jacobsoni (191). Transfer experiments (114) confirmed that *E. sinhai* may survive on *A. mellifera* and *A. cerana*, emphasizing their ability to cross-infest exotic hosts.

Euvarroa wongsirii parasitizes drone brood of *A. andreniformis* in Thailand and Malaysia. Its biology appears similar to *E. sinhai* and it can live for at least 50 days on worker bees outside the nest (137).

PARASITIC MITES ON OTHER SPECIES OF APIS

The systematics of *Apis* is still unsettled. Only four species, namely *cerana, dorsata, florea,* and *mellifera,* were recognized until recently (174), with other entities being treated as races or subspecies. Wu & Kuang (212) distinguished *andreniformis* from *florea*, whereas the separate status of *koschevnikovi* was confirmed in 1989 (175). Koeniger (113) added *laboriosa, nigrocincta* and *nuluensis*, the latter discovered from Sabah (northern Borneo) (197). Most *Apis* species occur in south Asia and more are likely to be distinguished there (146, 194a). As these species come to be recognized, along with their unique acarine associates, a more complete pattern of the host relationships should emerge, based on phylogenetic analyses of both bees and mites.

Another untapped source for additional parasitic Acari could be the associates of the African ''races'' of *A. mellifera* (100, 173), about which very little is known (18). Most parasitic mites are described from the four ''classic'' bees. However, several new mites were recently discovered parasitizing the classic as well as the newly-recognized honey bee species (see Table 2).

Delfinado et al (60) and Koeniger (112) noted the relationship between bee nesting habitat and the genus or family of associated parasitic mites. The small bush-nesting species with single combs (*andreniformis* and *florea*) are attacked by *Euvarroa* spp. (121). The bees that build multi-comb nests in caves and trees (*cerana, koschevnikovi,* and *mellifera*) are parasitized by *Varroa* spp. (45), and the tree bees with single combs (*dorsata* and *laboriosa*) by *Tropilaelaps* spp. (61). This pattern also seems to hold for the more recently described *V. rindereri*, but it could change due to the mites' facility for cross infestation (e.g. *T. clareae* occurring on *cerana* and *mellifera*).

These data, along with the tentative suggestion that a bee from Luzon in the Philippines may be distinct from *A. cerana* and from *A. nigrocincta* (41), indicate that more and perhaps unexpected associations between honey bees and parasitic mites remain to be discovered. Signs of things to come are the significant differences found between *E. sinhai* from India and from Thailand (137), and the collection of *Varroa* sp. near *underwoodi* on *A. nuluensis* in Borneo (44). More species of varroids and *Tropilaelaps* are likely to be found in southeast Asia; if these mites find their way to European or the Western Hemisphere beehives, significant problems could result.

MITE-ASSOCIATED BEE PATHOLOGIES

Several mite-related pathologies have been reported (177), but their etiology and precise relation to bees are obscure. Even the Isle of Wight disease, which precipitated the discovery of *A. woodi*, is now considered to have been ''of unknown origin'' (10).

Bee Parasitic Mite Syndrome (BPMS)

First reported when bee colonies were stressed by varroa mites, the name BPMS or PMS was given (189) to explain why colonies infested with both HBTM and varroa were not thriving. BPMS may be related to both mites vectoring a virus, such as acute paralysis virus (106, 108). The symptoms, which can be present any time of the year, include the presence of mites, the presence of various brood diseases with symptoms similar to that of the foulbroods and sacbrood but without any predominant pathogen, American foulbrood-like symptoms, spotty brood pattern, increased supersedure of queens, bees crawling on the ground, and a lowered adult bee population. Although BPMS remains an enigma, feeding colonies Terramycin (antibiotic) syrup or patties and pollen supplements and using resistant bee stock have shown promise in keeping bees alive.

Viruses

Honey bees are subject to many viruses (4, 17), five of which are associated with varroa and one with HBTM. Virus particles are probably always present in latent or inapparent form in or on bees, or in the hive environment. Some viruses may be induced or activated by puncturing healthy bees (109), similar to the wounds inflicted by mites. Such etiology could explain why these diseases appear mostly in infested colonies and when colonies are heavily parasitized by mites that the mortality is so acute (14, 16). Challenges to controlling bee viruses include early diagnosis and identification, inconsistent association with mites, or methodical problems in establishing the relationship. In addition, many viral strains from different countries may be related. Molecular and serological techniques are expected to clarify these issues. Most of these viruses were unknown prior to the introduction of varroa.

Acute Paralysis Virus (APV) This virus kills both adult bees and brood, especially in varroa-infested colonies in Europe and the U.S. (5, 15, 107, 109). The virus, activated by an unknown mechanism, multiplies when mites feed on infected bees. Nurse bees parasitized by mites infected with this activated virus can transmit it to larvae in the brood food, or to other adult bees. In this manner the activated virus can spread quickly and, once systemic, overwhelm the colony.

Kashmir Bee Virus (KBV) This disease was originally reported from *A. cerana* in Kashmir (13). Similar in size to several picornavirus-like agents, it is the most virulent and widespread disease, found everywhere bees are present. Like APV, KBV may be activated in the presence of varroa, multiplying to lethal levels, but KBV was held responsible for bee deaths in Australia (12) where varroa is not present. Strains of KBV from Canada and Spain resemble APV in serological tests (3). The pathology of KBV is still being studied and new molecular techniques may help to identify it (196).

Deformed Wing Virus (DWV) First reported from Poland (199), where young bees had malformed wings and were stunted, DWV is now found wherever varroa is found, even on *A. cerana* in China (17, 21). Originally attributed to varroa feeding on bee pupae, DWV is transmitted to healthy brood by varroa (17).

Other viral associations include slow paralysis virus (SPV), found in the United Kingdom (17), and cloudy wing virus (CWV), reported in the United States, Greece, United Kingdom, and Australasia. Pathologies are far from clear. CWV may be carried by air via the tracheal tubes. A recent report from Pennsylvania (33) verified the presence of an unknown iridovirus in varroa; the significance of this is not yet understood.

HBTM and Virus

Chronic Paralysis Virus (CPV) First reported in 1933, this is the only specific tracheal mite-associated virus (11, 28). CPV may have caused the Isle of Wight disease: The symptoms are very similar to HBTM infestation. CPV comes in two syndromes. Type I is distinguished by bees trembling, unable to fly, with K-wings and distended abdomens. Type II, called the hairless black syndrome, is recognized by hairless, black shiny bees crawling at the hive entrance. The virus is prevalent in colonies where bees are confined for long periods, and the circumstances of the disease are the same as those that aggravate mite infestations. While CPV can cause occasional outbreaks in the absence of mites, in mite-infested colonies the virus was markedly increased (15). Susceptibility to CPV may also be inherited.

Bacteria Varroa may transmit *Serratia marcescens*, a bacterium that causes septicemia in bees (92). About 20% of healthy brood become diseased if infected mites are allowed to feed on bee larvae. Varroa may also transmit other bacteria, such as *Hafnia alvei* (15). Although an increase in European foulbrood (EFB) was reported from infested colonies (200), the transmission of the bacterium by varroa was not confirmed. Because EFB is a stress-related disorder, colonies that are heavily infested with varroa are susceptible to EFB. American foulbrood

(AFB) spores have been photographed on the surface of varroa, but the mite has not been implicated in transmitting the disease (2).

Fungi Fungi are ubiquitous organisms found in all bee colonies, but varroa-stressed colonies appear to have an increased incidence of chalkbrood disease, *Ascosphera apis* (123). Such outbreaks may be attributable to inadequate brood care by mite-stressed workers and depleted bee populations. Chilled brood is more prone to succumb to the fungus than brood adequately incubated by nurse bees. Spores of *Aspergillus* spp. (the cause of stonebrood) have also been found on varroa (123).

CONCLUSIONS

The sudden global emergence of bee mites during the past decade prompted increased research on detecting, monitoring, and controlling them. Meantime, basic mite studies have been lagging. Several areas of research must be addressed if bees are to remain a viable segment of agriculture. For example, it is not clear how particular bee mites actually damage their hosts, and what the role various disease organisms play in the ensuing colony decline. The presence of viruses is a special challenge and controlling these mites would almost certainly diminish viral effects. Next, the emerging recognition of new bee species and their mite parasites makes continued research a high priority. We need to determine whether any of these ''new'' mites will also cross-infest the dominant domesticated honey bee, *A. mellifera*, should they ever be introduced. More parasitic mites would be disastrous for the bee industry.

That *V. jacobsoni* may actually consist of several genotypes (or species) attests to large gaps in our knowledge. Further investigation of this matter must be pursued vigorously. Then, the development of chemicals or other modes of controlling bee mites would be facilitated by rearing them apart from bees. Much more work on in vitro culture of all parasitic bee mites is needed, although laboratory methods for culturing varroa and *T. clareae* were developed by Rath (162).

Finally, the most pressing problem remains control, preferably without synthetic chemicals. Thus we conclude by advocating the vigorous testing of various active ingredients, such as ''botanicals,'' the breeding of bees for resistance to both mites, and exploring other biological or cultural techniques. The challenges ahead include attracting mites away from bees, killing mites without contaminating hive products or injuring bees, and keeping the controls economic, effortless, and easy for both the hobbyist and commercial beekeeper. Another problem is to determine an economic injury level of mite infestation and a threshold to indicate treatment times; doing so would take the guesswork out of when to medicate. The long-term solution to parasitic bee mites is in developing an integrated pest management program to manage mites by multiple means, not relying on any one or two chemical treatments.

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