Exp Appl Acarol (2009) 47:225–234
DOI 10.1007/s10493-008-9213-3

A qualitative model of mortality in honey bee (*Apis mellifera*) colonies infested with tracheal mites (*Acarapis woodi*)

John B. McMullan · Mark J. F. Brown

Received: 11 August 2008 / Accepted: 29 October 2008 / Published online: 14 November 2008

Abstract The tracheal mite has been associated with colony deaths worldwide since the mite was first discovered in 1919. Yet controversy about its role in honey bee colony mortality has existed since that time. Other pathogens such as bacteria and viruses have been suggested as the cause of colony deaths as well as degenerative changes in individual honey bees. Using data from published work we developed a qualitative mortality model to explain colony mortality due to tracheal mite infestation in the field. Our model suggests that colonies of tracheal-mite infested honey bees, with no other pathogens present, can die out in the late winter/early spring period due to their inability to thermoregulate. An accumulation of factors conspire to cause colony death including reduced brood/bee population, loose winter clusters, reduced flight muscle function and increasing mite infestation. In essence a cascade effect results in the colony losing its cohesion and leading to its ultimate collapse.

Keywords *Apis mellifera* · *Acarapis woodi* · Tracheal mite · Colony mortality · Thermoregulation

Introduction

In 1906, on the Isle of Wight, off the south coast of England, honey bee colonies were dying with no apparent cause (Adam 1968). The condition which later became known as the 'Isle of Wight disease' spread throughout Britain and Ireland, mainland Europe, Asia, Africa, South America and eventually North America (Bailey and Ball 1991). The tracheal mite *Acarapis woodi* (Acari: Tarsonemidae) was first identified in colonies of honey bees *Apis mellifera* (Hymenoptera: Apidae) in 1919 (Rennie et al. 1921) as a result of investigations undertaken to discover the cause of the fatal condition. The tracheal mite has since that time been associated with these colony deaths, yet there has never been any consensus

J. B. McMullan (✉) · M. J. F. Brown
Department of Zoology, School of Natural Sciences, Trinity College Dublin, Dublin 2, Ireland
e-mail: jmcmullan@eircom.net

about the cause of the Isle of Wight disease and doubt about the role of tracheal mites in colony mortality remains to the present day (Bailey 1985, 1999; Adam 1987; Collison 2001).

Recent research into tracheal mite infestation in North America, while generally acknowledging its association with colony mortality, has not established an actual mechanism to explain such mortality. The tracheal mite's appearance as an exotic pathogen in the US in 1984 conformed to the normal pattern of correlating with high initial colony mortality, which eventually settled down at a lower level (Otis and Scott-Dupree 1992; Fries and Camazine 2001). One reason that the exact role of and mechanism relating tracheal mites to colony mortality remains unclear is because they often co-occur with other pathogens. Where this happens a synergistic effect can occur that hastens colony death (Bailey 1958; Downey and Winston 2001). In the recent survey of US honey bees in connection with colony collapse disorder (CCD), tracheal mites were detected in 24% of the samples either on their own or in combination with other pathogens (Cox-Foster et al. 2007).

While the tracheal mite has been with us for a long time and while there is strong evidence that it can cause colony death, there exists no comprehensive explanation as to the mechanisms by which death might occur in the late winter/early spring period. Over time a number of reasons for colony mortality have been suggested. Tracheal mites can have an effect at the individual level as well as at the colony level and it is important that the distinction between these two levels is made in order to understand how mortality occurs in affected colonies. To avoid any confusion caused by this, we review, (1) the effects of tracheal mites on bees, and (2) previously suggested causes of colony death, following which we develop a qualitative model of colony mortality.

Morbidity and mortality of individual infested bees

Bailey (1975, 1985, 1999) suggested that the death of tracheal mite infested honey bees is due to chronic bee paralysis virus (CBPV), with individual bees becoming infested and dying off within a few days. Collison (2001) reviewed previous work and stated that, "it is not known exactly why infested bees die" but suggested that it may be related to degeneration in tracheae, flight muscles, nerve ganglia and hypopharyngeal glands. Komeili and Ambrose (1991) showed that infestation caused degeneration to flight muscles. Tracheal mite infestation has also been shown to cause a reduction in the metabolic rate of individual bees (Nasr et al. 1999; Harrison et al. 2001).

An extensive investigation of the impact of tracheal mites on the respiration and thermoregulation of honey bees was undertaken in Canada (Skinner 2000). This research work demonstrated that individual bees with a high prevalence of mites had a significantly lower oxygen consumption than uninfested bees even at the relatively high temperature of 15°C (8.93 ± 0.07 vs. 9.82 ± 0.07 ml O_2 bee^{-1} h^{-1}). Both prothoracic tracheae were infested in 75% of the test bees. When both prothoracic spiracles were sealed with wax, simulating 100% bilateral infestation, there was a further significant reduction in oxygen consumption to 8.33 ± 0.09 ml O_2 bee^{-1} h^{-1}.

Other studies indicate that the mortality of infested individual bees varies depending on the season. As individuals, honey bees have a limited ability to heat themselves. There is evidence that individual summer bees infested with the mites have a similar life span to uninfested ones (Bailey and Lee 1959; Gary and Page 1989). In relation to winter conditions Bailey (1958, 1961) showed that infested bees die sooner than uninfested individuals and the difference becomes significant in late winter. We recently observed tracheal-mite-infested

bees that had left the hive in early spring and appeared to be dying on the grass in late evening; when collected and put into an incubator at 32°C they lived for several weeks (JBMcM, unpublished data). Also, recent work reported as an abstract by Villa (2006) showed that individual-infested bees in unheated cages die significantly sooner than uninfested-individual bees but the difference is not significant when the bees are kept in an incubator (hoarding cages). He concluded that "the effect of tracheal mites on worker longevity may be variable and even absent under some circumstances". Nevertheless, together these data suggest that individual mortality due to tracheal mites is temperature-dependent.

Explanations for colony mortality

The initial report on widespread devastation of honey bee colonies on the Isle of Wight described the 'paralysis' of the bees. Cooper's report (1906) is worth quoting:

> During a bright, sunny day, recently, the bees in coming out of the hive dropped to the grass by the dozen, and seemed quite powerless, so far as using their wings…. The abdomen of the bee was not distended in every case, while the wings were often twisted back, having the appearance of being dislocated…. They kept crawling up the blades of grass and on to the alighting board only to tumble off again, and when the sun went down, and the day became colder, the bees collected in little bunches of a dozen or so in each, and soon perished. Another hive since becoming affected by paralysis has developed dysentery, and the bees are rapidly dwindling.

This account was typical of the terminal behaviour of colonies that showed the symptoms of the 'Isle of Wight disease' and also in colonies infested by the mite after 1919 when the mite was first identified (Rennie et al. 1921; Adam 1968). However, there have been different interpretations of these symptoms over the years. Adam (1987) considered that colonies can die due solely to the presence of high infestations of tracheal mites and only express clinical symptoms for a short period prior to the death of the colony. However, the eventual symptoms of infested bees in affected colonies have been claimed by Bailey (1975) to be due to a paralysis virus. Bailey maintained that CBPV causes the rapid death of affected colonies and also that tracheal mites are not a vector of CBPV (Bailey 1975, 1985, 1999). How individual paralysis and mortality translated into colony mortality was left unspecified. However, this claim of death being due to CBPV contrasts with the findings from more recent research that has taken place in the US and Canada. The mite was first discovered in the US in 1984 and since then widespread colony mortality has occurred (Mussen 2001). There is evidence that the mite had moved from south and central America into the US and that concerted sampling in the Southern States prior to 1984 had failed to find any mites (Mussen 2001). This adds to the direct relationship between the presence of tracheal mites and colony mortality. There have also been no claims of the presence of CBPV in these colonies, which is one of the few viruses that have clearly defined clinical symptoms. Studies that we have undertaken (unpublished data) show that colonies can die with only tracheal mite infestation and in particular with no presence of CBPV detected either by clinical symptoms or molecular analysis.

Despite the lack of evidence for the CBPV explanation, establishing the relationship between the presence of tracheal mites and colony mortality has undoubtedly been confounded by the presence of other pathogens. It is likely that many diseases such as nosema, paralysis, American foulbrood and chalk brood, which were common for much of the last century (Fries and Camazine 2001), would have made it difficult to isolate causes.

Despite this, several different lines of evidence suggest that environmental temperature may be related to mite-driven colony mortality, just as it is with individual worker mortality. Bailey (1961) found that when over 30% of the individual bees in a colony were infested the increase in colony mortality in winter was significant. More recently, Otis and Scott-Dupree (1992) suggested that a combination of the longer winters endured by bees in cold regions, the direct effect of the parasitic mites on individual bees and the indirect effect of pathogens introduced by feeding mites to parasitized bees cause colony death before brood production can produce enough young bees in spring. However, even if this suggestion is correct, the mechanism linking these various factors together remains unspecified.

Jeffree (1955, 1959), based on the experience at that time of tracheal mite infestation, suggested that mite infestation would only occur globally within particular temperature ranges. He suggested that tracheal mites would be absent outside of the regions enclosed by January isotherms of −3 and 12°C and July isotherms of 11 and 19°C. His predictions were largely based on reported colony infestations which, particularly up to the mid twentieth century, would have closely corresponded with colony mortality. There was little random testing for the presence of the tracheal mite at that time and the presence of infestation would therefore have been largely identified by post mortem subsequent to colony deaths. It has since been shown that tracheal-mite infestation of honey bees was much more widely distributed. (Matheson 1993). However, if Jeffree had stated his theory as predicting colony mortality he would, in our opinion, have been largely correct. In many sub-tropical locations tracheal mite infestation can be high and also widespread but with colony deaths remaining at a low level. This is the case in locations such as the Southern States of America, while further north colony deaths are commonplace (Eischen 1987; Otis and Scott-Dupree 1992; Gard Otis and Diane Sammataro, personal communication). This is further evidence that a relationship exists between the prevailing climatic conditions and colony mortality.

Skinner (2000) demonstrated that clusters of infested bees at 5°C had significantly lower oxygen consumption than uninfested bees (36.18 ± 0.14 vs. 40.31 ± 0.7 ml O_2 g^{-1} h^{-1}). This lower oxygen consumption occurred even when over 60% of bees were only infested in one prothoracic trachea. The oxygen demands of clusters at 5°C were three times greater than at 24°C demonstrating the dramatic effect of reduced temperature on a colony. It was further shown that during early spring periods cluster temperatures were significantly lower in high-prevalence (>40%) compared to low-prevalence (<10%) colonies. Mean cluster temperatures in (1) an indoor apiary were 29.04 ± 0.53 versus 35.65 ± 0.35°C and in (2) outdoor unwrapped colonies 15.12 ± 0.88 versus 25.46 ± 1.12°C for high and low-prevalence colonies, respectively. The winter mortality in high-prevalence colonies over a number of wintering systems (indoor, wrapped, etc.) averaged over three times higher than in the low-prevalence colonies. Also, during early spring the weight of dead bees collected outside the hives in the higher infested colonies was significantly higher than in the lower infested colonies. This corroborated previous work in Canada and reported in an abstract by Nasr et al. (1999) that winter mortality in colonies with high mite infestation (>50%) was significantly higher than in uninfested colonies when poor winter wrapping was used.

Consequently, we suggest that a key factor driving the relationship between climate, tracheal mites and colony mortality is the requirement on honey bee colonies to thermoregulate.

Colony thermoregulation

Individual bees have a limited ability to heat themselves due to rapid heat loss from their small bodies which have a disproportionately large surface area (Southwick 1991). To

overcome this thermal disadvantage, individual honey bees in a colony form a cluster and hence use scale to keep the colony warm (Southwick 1991). This occurs as the surface area of an individual bee is about 2 cm^2, while within a typical winter cluster the surface area per bee reduces to 0.07 cm^2 (Seeley 1985).

Honey bees use their wing muscles to generate heat by 'shivering' to enable colony thermoregulation and to raise their own thorax temperature to initiate flight (Heinrich 1996). The degeneration in the flight muscles of tracheal-mite infested bees (Komeili and Ambrose 1991) would reduce their ability to use their wing muscles to generate heat. The greater demands of colony thermoregulation in winter can explain the reduced life span of winter infested bees while summer bees are largely unaffected. Similarly, in the case of individual infested bees in an incubator, thermoregulation is provided and little difference in longevity between infested and uninfested bees would be expected. This is also evidenced by infested honey bee queens living for many years (Fyg 1964). Furthermore, as mite prevalence in a colony increases during the winter period, there will be fewer healthy (uninfested bees) to undertake the task of heating the colony. This would put increased demands on individual infested bees to assist in the task of heating the colony and to cause further wing function deterioration through increased use of their wing muscles. Such colonies would experience reduced thermoregulation, bee flight ability and bee population and, in a cool climate, may reach the stage where colony death will occur in the late winter/early spring period.

Here we suggest, using a qualitative model based on published papers, that such colony mortality is not simply an extrapolation of individual bee mortality, but is in fact driven by a failure in colony-level thermoregulation. The mechanism causing death is the cumulative effect of many factors, each of which is related to tracheal mite infestation of individual bees.

Mortality model

The honey bee colony during late autumn to early spring is in its dormant period when stored honey (chemical energy) is largely converted to heat energy to maintain the colony temperature (by replacing the cluster heat loss) but also to feed any honey bee brood present. It is essentially a closed energy system that conforms to the principles of physics. We developed a model for colony mortality (Fig. 1) which is illustrated as a spiral demonstrating the continuous, progressive and cumulative effects of the different influences on a colony infested with tracheal mites. The proposed model is qualitative and the rate of deterioration of the colony will depend on prevailing conditions. Notwithstanding the widely varying conditions that can be present, the influences outlined in the model will apply. Ten nodes are highlighted in the model. The spiral contains an inner loop and an outer loop between Nodes 1 and 7. The outer loop (Nodes 2–4) identifies the demand on the colony to increase its heat energy output per bee while the inner loop (Nodes 5–6) identifies the reduced ability of the colony to increase its heat energy output per bee. These elements culminate in temperature stress on the colony at Node 7. Increasing mite prevalence and reducing flying ability of the bees give rise to a reduced colony population that can lead to colony death (Nodes 8–10).

Node 1: Infested colony in winter. This is the starting position for an infested colony of tracheal-mite susceptible bees in the late autumn/early winter period.

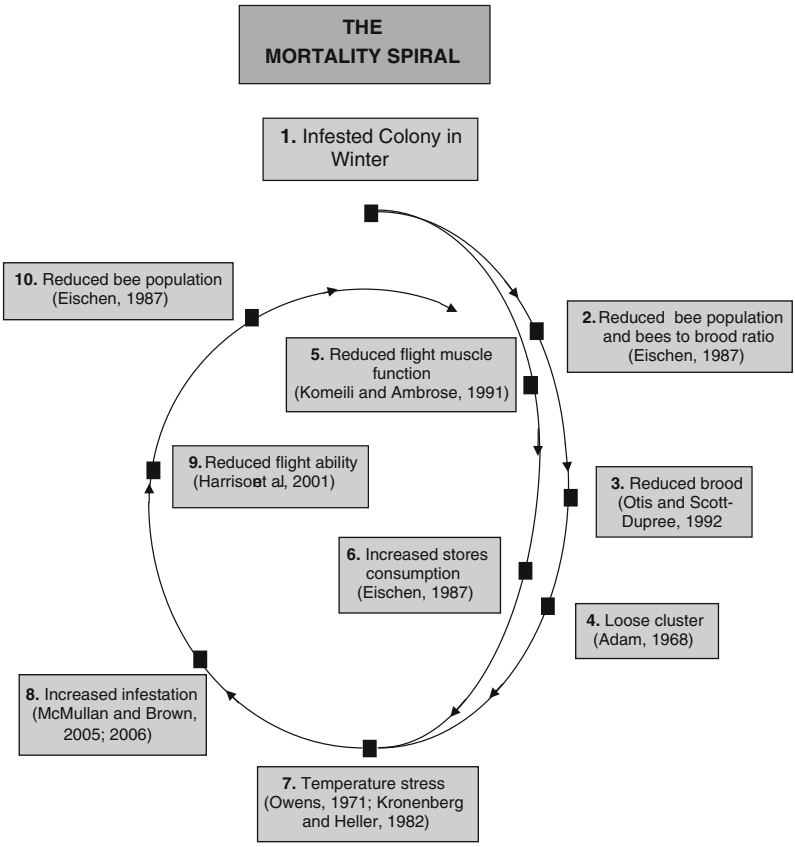

Fig. 1 Mortality spiral showing the dynamic influences on a moderately infested colony during the winter/early spring period

Outer loop Nodes 2–4

Node 2: Reduced bee population and bees to brood ratio. Increased mite prevalence in colonies results in reduced bee populations at the beginning of winter and at the beginning of spring (Eischen 1987). In addition, analysing the data in Eischen's paper shows that the ratio of bees to brood in infested colonies is lower; in November and February for three stages of colony infestation (uninfested, moderately infested and heavily infested) the bees to brood ratio was 4.4, 3.4, 3.6 and 3.7, 2.6, 2.2, respectively. Reduced bees to brood ratio will mean that there are fewer bees per unit of brood area to maintain the brood at the required temperature, normally 34–35°C. This reduced bee population and bees to brood ratio with increased infestation in the late autumn and late winter period will require individual bees in infested colonies to increase their thermal output.

Node 3: Reduced brood. An infested colony has reduced brood rearing in the late winter/early spring period (Otis and Scott-Dupree 1992). The reduced brood and corresponding reduced emergence of callow bees will increase the infestation pressure on the existing callow bees and drive up the mite prevalence of the colony. The reduced bee population and hence the smaller brood-nest cluster will result in an increase in cluster surface area to

volume of bees. This will cause the colony to lose economy of scale and require greater heat generation by individual bees.

Node 4: Loose cluster. Infested colonies have looser and fragmented winter clusters (Adam 1968; Shah 1987; Otis and Scott-Dupree 1992; Wilson et al. 1997). The effect of loose clusters is to reduce the level of cluster insulation and, combined with the continuous bee movement, to increase the cluster heat loss and hence increase the energy needs of the bees.

Inner loop Nodes 5–6

Node 5: Reduced 'shivering' function. Honey bees use their thoracic muscles to generate heat in the winter cluster by 'shivering' (Heinrich 1996). Tracheal mite infested bees have degeneration in their flight muscles and hence reduced ability to generate heat (Komeili and Ambrose 1991). Harrison et al. (2001) have also identified reduced flight metabolic rate in bees with mite infestation and this was particularly so in hypoxic air conditions. In 10% oxygen atmospheres the flight metabolic rate was reduced by 20% for moderate mite infestation and 40% for severe mite infestation. Van Nerum and Buelens (1997) showed that the normal winter cluster maintains a low oxygen level (~15%) with the lowest oxygen level in the centre of the cluster (typically 6% below) and increasing towards the outside of the mantle, with the rise in oxygen level slowest towards the back of the hive This would further reduce the bees ability to metabolise the honey stores and put additional pressure on the colony's ability to maintain the cluster temperature.

Node 6: Increased stores consumption. Infested colonies consume more honey stores per bee than healthy colonies during winter (Eischen 1987). This would be directly related to the increased need for more heat energy per bee as a result of the changed colony conditions (Nodes 2 and 3) and the increased heat loss due to loose clusters (Node 4). This increased honey consumption would also be explained by the infested bees having a low efficiency in metabolising the honey stores and converting it to heat energy since Komeili and Ambrose (1991) have identified that infested bees have flight muscle degeneration. N. B. Where tracheal-mite-infested colonies eventually die, typically in the early spring period, these colonies have normally more stored honey than healthy colonies. This can be explained by infested colonies having rapidly reduced bee populations and brood in late winter/early spring.

Nodes 7–10

Node 7: Temperature stress. While healthy colonies of *A. mellifera* have good regulation of brood temperature, it is also known that in adverse conditions the brood temperature held by the bees can be well below typical levels; as low as 29.5°C within the brood area (Kronenberg and Heller 1982; Owens 1971). Conversely, in normal conditions bees that are not healthy may have difficulty regulating brood temperature. If the heat energy produced per bee by shivering is less than the heat energy lost per bee, the temperature of the cluster will drop to maintain an energy balance (Winston 1987). In the longer term, as worker populations decline, the temperature will continue to decline and eventually drop below the minimum core cluster temperature. Hence, the compound effect of the influences in the outer loop (Nodes 2–4) requiring increased heat output by the bees, and the inner loop (Nodes 5–6) resulting in reduced heat output by the bees, may reach a stage where the core cluster temperature will have to fall.

Node 8: Increased infestation. As the winter progresses, a lower cluster temperature will be experienced. This reduced temperature will increase the susceptibility of the young emerging bees to tracheal mite infestation (McMullan and Brown 2005, 2006). The effect will be to increase further the infestation level in an already increasingly-infested colony.

Node 9: Reduced flight ability. Honey bees with moderate mite infestation have reduced flight metabolic rate in severe atmospheric conditions and this reduces further with increased mite infestation (Harrison et al. 2001). Harrison et al. (2001) also demonstrated that in cool weather, bees that were unable to return to the hive during late winter flights had significantly higher levels of mite infestation than those that returned safely. Thus infested flying bees in cold weather conditions will have difficulty in returning to the hive. Some infested bees may not be able to fly, whether due to severe oxygen delivery restriction or damage to their thoracic wing muscles, and can only crawl out of the hive.

Node 10: Further reduced bee population. The infested bees in highly infested colonies leaving the hive in this period (most likely to defecate by attempting to fly or crawl out of the hive) will increasingly not return resulting in a dramatic reduction in the colony population (Eischen 1987). The increased honey consumption per bee (Node 6) would also increase the need for winter cleansing flights and hence increase the risk of the bees being lost. Most tracheal-mite infested bees die outside the hive (Adam 1987).

The spiral of decline proposed in this model will have a cumulative and progressive effect on the colony driving it downwards and may eventually result in colony collapse.

Discussion

The model describes the interaction of many variables and whether or not the infested colony actually dies out will primarily depend on factors influencing the tracheal mite infestation and also the prevailing thermal conditions. The model demonstrates how the cohesion of a superorganism (honey bee colony) that depends on scale for its existence will break down as its population declines under adverse conditions causing a cascade effect leading to ultimate death. A colony with a high level of tracheal mite infestation in the autumn can die out in the late winter/early spring period with no other significant pathogen present. The cause of the decline is the inability of the honey bee colony to thermoregulate. Of course, honey bee colonies infested with tracheal mites may also occasionally be afflicted with other diseases. Downey and Winston (2001) demonstrated that where tracheal and varroa mites are present a synergistic effect occurs giving rise to rapid colony deterioration. Hence knowledge of the mite status of colonies should be of particular concern to beekeepers as relatively low levels of tracheal mite infestation in conjunction with other diseases can endanger colony winter survival.

This model used evidence from the accumulated body of research to date to explain why colonies with tracheal mites die. While a lot of research has been undertaken particularly in the last two decades since the tracheal mite reached central and North America, to our knowledge this paper is the first attempt to explain the interplay between the many factors influencing colony mortality. Understanding the dynamics of mortality in tracheal-mite infested colonies is important as a contribution to our knowledge of honey bee-parasite behaviour as well as its practical use in beekeeping colony management. In the latter case the model would suggest that more widespread use of winter wrapping could reduce colony deaths.

Finally, in this qualitative model we have shown that the mechanism linking the various relationships that contribute to mortality is the thermoregulation of the colony. A further stage would be to build a quantitative model which predicts the outcome (mortality) for a tracheal mite infested colony. The factors determining outcome from our qualitative model are those that influence the degree of tracheal mite infestation in a colony and also those that influence the thermal conditions of the colony. These include severity of the original

infestation (Bailey 1961), strain of bee (Adam 1968; Danka and Villa 1996), hive insulation (Nasr et al. 1999) and winter climate (Eischen 1987; Otis and Scott-Dupree 1992; McMullan and Brown 2005). Otis and Scott-Dupree (1992) developed a relationship between the probability of colony mortality in New York State and the mite prevalence values. Using a model for the honey bee in a particular geographical area, the mortality can be predicted using two factors: the level of initial mite infestation and the degree of heat loss. Data from past research supplemented by that from new experiments would enable a quantitative overwintering-mortality model to be developed to estimate the probability of colony death in any climatic area.

Acknowledgments The authors would like to thank the editor and two anonymous reviewers for their considerable contribution, which has improved both the content and clarity of the text. Also John and Dorothy Stapleton for access to honey bee colonies.

References

Adam B (1968) "Isle of Wight" or acarine disease: its historical and practical aspects. Bee World 49:6–18
Adam B (1987) The honey-bee tracheal mite-fact and fiction. Am Bee J 127:36–38
Bailey L (1958) The epidemiology of the infestation of the honeybee. *Apis mellifera* L., by the mite *Acarapis woodi* Rennie and the mortality of infested bees. Parasitology 48:493–506
Bailey L (1961) The natural incidence of *Acarapis woodi* (Rennie) and the winter mortality of honeybee colonies. Bee World 4:96–100
Bailey L (1975) Honey bee pathology: the end of the beginning. The Cent Assoc of Beekeepers, Ilford
Bailey L (1985) *Acarapis woodi*: a modern appraisal. Bee World 66:99–104
Bailey L (1999) The century of *Acarapis woodi*. Am Bee J 139:541–542
Bailey L, Ball BV (1991) Honey bee pathology. Academic Press, London
Bailey L, Lee DC (1959) The effect of infestation with *Acarapis woodi* (Rennie) on the mortality of honey bees. J Insect Pathol 1:15–24
Collison CH (2001) The pathological effects of the tracheal mite on its host. In: Webster TC, Delaplane KS (eds) Mites of the honey bee. Dadant and Sons, Hamilton, pp 57–71
Cooper HM (1906) Bee paralysis: is the cause known? Br Bee J 34:56–57
Cox-Foster DL, Conlan S, Holmes EC et al (2007) A metagenomic survey of microbes in honey bee colony collapse disorder. Science 5848:283–287
Danka RG, Villa JD (1996) Influence of resistant honey bee hosts on the life history of the parasite *Acarapis woodi*. Exp App Acarol 20:313–322. doi:10.1007/BF00052961
Downey DL, Winston ML (2001) Honey bee colony mortality and productivity with single and dual infestations of parasitic mite species. Apidologie (Celle) 32:567–575. doi:10.1051/apido:2001144
Eischen FA (1987) Overwintering performance of honeybee colonies heavily infest with *Acarapis woodi* (Rennie). Apidologie (Celle) 18:293–304. doi:10.1051/apido:19870401
Fries I, Camazine S (2001) Implications of horizontal and vertical pathogen transmission for honey bee epidemiology. Apidologie (Celle) 32:199–214. doi:10.1051/apido:2001122
Fyg W (1964) Anomolies and diseases of the queen honey bee. Annual Review of Entomology 9:207–224. doi:10.1146/annurev.en.09.010164.001231
Gary NE, Page RE (1989) Tracheal mite (Acari: Tarsonemidae) infestation effects on foraging and survivorship of honey bees (Hymenoptera: Apidae). J Econ Entomol 82:734–739
Harrison JF, Camazine S, Marden JH et al (2001) Mite not make it home: tracheal mites reduce the safety margin for oxygen delivery of flying honeybees. J Exp Biol 204:805–814
Heinrich B (1996) How the honey bee regulates its body temperature. Bee World 77:130–137
Jeffree EP (1955) Acarine disease of the honeybee and temperature. Nature 175:91. doi:10.1038/175091a0
Jeffree EP (1959) The world distribution of acarine disease of honeybees and its probable dependence on meteorological factors. Bee World 40:4–15
Komeili AB, Ambrose JT (1991) Electron microscope studies of the tracheae and flight muscles of noninfested, *Acarapis woodi* infested and crawling honey bees *Apis mellifera*). Am Bee J 131:253–257
Kronenberg F, Heller C (1982) Colonial thermoregulation in honey bees (*Apis mellifera*). Journal of Comparative Physiology 148:65–76
Matheson A (1993) World bee health report. Bee World 74:176–212

McMullan JB, Brown MJF (2005) Brood pupation temperature affects the susceptibility of honeybees (*Apis mellifera*) to infestation by tracheal mites (*Acarapis woodi*). Apidologie (Celle) 36:97–105. doi:10.1051/apido:2004073

McMullan JB, Brown MJF (2006) The role of autogrooming in the differential susceptibility to tracheal mite (*Acarapis woodi*) infestation of honeybees (*Apis mellifera*) held at both normal and reduced temperatures duration pupation. Apidologie (Celle) 37:471–479. doi:10.1051/apido:2006017

Mussen EC (2001) Introduction, spread, and economic impact of tracheal mites in North America. In: Webster TC, Delaplane KS (eds) Mites of the honey bee. Dadant and Sons, Hamilton, pp 43–56

Nasr ME, Skinner AJ, Kevan PG (1999) Oxygen consumption, thermoregulation and wintering of honey bees infested with tracheal mites (*Acarapis woodi*). Am Bee J 139:313 Abstract from the 1999 American bee research conference, Baton Rouge, Louisiana

Otis GW, Scott-Dupree CD (1992) Effects of *Acarapis woodi* on overwintered colonies of honey bees (Hymenoptera: Apidae) in New York. Journal of Economic Entomology 85:40–46

Owens CD (1971) The thermology of wintering honey bee colonies. US Department of Agriculture. Tech Bull 1429:1–32

Rennie J, White PB, Harvey EJ (1921) Isle-of-Wight disease in hive bees. The etiology of the disease. Trans R Soc Edinb 52:737–755

Seeley TD (1985) Honeybee ecology. Princeton University Press, Princeton

Shah FA (1987) Twenty years of acarine mite in India. Gleanings bee Cult 115:517

Skinner AJ (2000) Impacts of tracheal mites (*Acarapis woodi* (Rennie)) on the respiration and thermoregulation of overwintering honey bees in a temperate climate. M.Sc. thesis. The University of Guelph, Guelph, Ontario

Southwick EE (1991) The colony as a thermoregulating superorganism. In: Goodman LJ, Fisher RC (eds) The behaviour and physiology of bees. Redwood, Melksham

Van Nerum K, Buelens H (1997) Hypoxia-controlled winter metabolism in honeybees (*Apis mellifera*). Comparative Biochemistry and Physiology 117A:447–455

Villa JD (2006) Do tracheal mites reduce the longevity of workers? Am Bee J 146:450–451 Abstract from the 2006 American bee research conference, Baton Rouge, Louisiana

Wilson WT, Pettis JS, Henderson CE et al (1997) Tracheal mites. In: Morse RA, Fluttum K (eds) Honey bee pests, predators and diseases. AI Root Company, Medina, Ohio, pp 253–277

Winston ML (1987) The biology of the honey bee. Harvard University Press, Massachusetts