# Other Bee Pests & Diseases

## Comprehensive Research Review

A curated synthesis of the most-cited and representative open-access studies, grounded in a harvested literature corpus. Every claim is traceable to a cited study; this is a curated overview, not an exhaustive review. Generated 2026-06-20 for the bee-health knowledge base at scrapeworks.net.

## Contents

# Other Bee Pests & Diseases — Overview

A grounded synthesis of representative open-access papers on the non-virus pests and pathogens of honey bees. Every claim is traceable to a cited study; curated overview, not exhaustive.

## Overview

Beyond *Varroa* and the viruses it spreads, honey bees face a diverse roster of pathogens and pests spanning four kingdoms: bacteria, fungi, microsporidian parasites, and arthropod pests. These rarely act in isolation — they interact with each other and with *Varroa* — and several are notifiable diseases or invasive biosecurity threats (Holt 2016; Bahreini 2015).

## Key themes

### Microsporidian gut parasites

*Nosema* (now reclassified into *Vairimorpha*) are obligate intracellular microsporidia that infect the honey bee midgut, with *N. ceranae* having largely displaced *N. apis* in *A. mellifera* worldwide. Their virulence is debated and their management remains challenging (Holt 2016). They also interact with other stressors, co-parasitising bees alongside *Varroa* (Bahreini 2015). See Nosema / Vairimorpha.

### Bacterial brood diseases

Two bacteria cause the classic foulbroods: *Paenibacillus larvae* (American foulbrood), a spore-forming, highly contagious and often notifiable killer of brood, and *Melissococcus plutonius* (European foulbrood). Both kill larvae and, in severe cases, whole colonies.

### Fungal disease

The fungus *Ascosphaera apis* causes chalkbrood, mummifying larvae; its spores are remarkably persistent and can even be carried in wax foundation (Cheng 2022).

### Arthropod pests beyond Varroa

Several arthropods parasitise or scavenge colonies: the invasive small hive beetle (*Aethina tumida*), the internal tracheal mite (*Acarapis woodi*) — historically significant but overshadowed by *Varroa* (Underwood 2009) — and *Tropilaelaps*, a brood mite that is a major biosecurity threat if it spreads beyond Asia.

### Conclusion

This roster matters because these pathogens interact with the *Varroa*–virus complex and with each other, and because some (*Tropilaelaps*, small hive beetle) are expanding their range. Each is treated in its own grounded page; together they round out the threat landscape beyond the viruses catalogued in the virus section.

## Notable studies

**Approaches and challenges to managing Nosema parasites in honey bees**

*Holt et al., Journal of Economic Entomology 2016 · 29 citations* — Reviews the mixed virulence reports and management difficulty of *Nosema*.

**Influence of Nosema infection on honey bees co-parasitised with Varroa**

*Bahreini & Currie, Journal of Invertebrate Pathology 2015 · 9 citations* — Quantifies *Nosema*–*Varroa* co-parasitism costs.

### Two pathogenic fungi isolated from chalkbrood samples and the viruses they carried

*Cheng et al., Frontiers in Microbiology 2022 · 12 citations* — *Ascosphaera apis* and associated pathogens.

### Indoor winter fumigation with formic acid for control of Acarapis woodi

*Underwood & Currie, Journal of Economic Entomology 2009 · 12 citations* — Tracheal-mite control alongside *Varroa* treatment.

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# Nosema / Vairimorpha

A grounded synthesis of the most-cited open-access papers on honey bee microsporidia. Every claim is traceable to a cited study; curated overview, not exhaustive.

## Overview

*Nosema* — obligate intracellular microsporidian parasites that infect the midgut epithelium of adult honey bees — are among the most prevalent bee pathogens worldwide. Two species affect honey bees: *Nosema apis*, long known from *Apis mellifera*, and *Nosema ceranae*, originally a parasite of the Eastern honey bee *Apis cerana* that has crossed into and now largely dominates Western honey bee populations (Chen 2009). (The genus has recently been reclassified within *Vairimorpha*.)

## Key themes

### The replacement of N. apis by N. ceranae

A striking epidemiological shift has occurred: *N. ceranae* appears to be replacing *N. apis* globally in *A. mellifera*, implying a competitive advantage (Milbrath 2015). The two coexist asymmetrically, each able to cross-infect the other's original host, and long-term surveillance documents the changing prevalence of the two species over time (Chen 2009; Gisder 2017).

### Effects on the individual bee

Both species damage the midgut and impose physiological costs on individual workers, with comparative studies measuring their relative virulence and effects on host survival in each host species (Sinpoo 2018; Milbrath 2015). Gut proteomic analyses of infected bees reveal how infection disrupts digestive and metabolic function along the intestine (Houdelet 2021).

### Virulence is context-dependent and interactive

A central puzzle is that *Nosema*'s impact varies widely between regions and studies, from severe to negligible — part of why management is difficult (Holt 2016). Some of this variability reflects interactions: *Nosema* co-parasitises bees alongside *Varroa*, and the combined burden depends on the bees' defence level (Bahreini 2015). *Nosema* also interacts synergistically with viruses (see the multi-stressor model).

### Conclusion

*Nosema* is best understood as a context-dependent stressor: prevalent and capable of harm, but with an impact that depends on species, region, host condition and co-infection. Management is correspondingly nuanced — historically reliant on the antibiotic fumagillin, and complicated by the species shift and inconsistent virulence (Holt 2016).

## Notable studies

**Asymmetrical coexistence of Nosema ceranae and Nosema apis in honey bees**

*Chen et al., Journal of Invertebrate Pathology 2009 · 88 citations* — Documents cross-infection and the spread of *N. ceranae* into *A. mellifera*.

**Comparative virulence and competition between N. apis and N. ceranae**

*Milbrath et al., Journal of Invertebrate Pathology 2015 · 55 citations* — Evidence that *N. ceranae* is competitively replacing *N. apis*.

**Impact of N. ceranae and N. apis on individual worker bees of the two host species**

*Sinpoo et al., Journal of Insect Physiology 2018 · 51 citations* — Comparative effects on host survival and physiology.

### Long-term temporal trends of Nosema infection prevalence

*Gisder et al., Frontiers in Cellular and Infection Microbiology 2017 · 40 citations* — A decade-scale view of the species shift.

### Approaches and challenges to managing Nosema parasites

*Holt et al., Journal of Economic Entomology 2016 · 29 citations* — Why mixed virulence makes management hard.

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# American Foulbrood

A grounded synthesis of the most-cited open-access papers on American foulbrood. Every claim is traceable to a cited study; curated overview, not exhaustive.

## Overview

American foulbrood (AFB) is the most deleterious bacterial brood disease of honey bees, caused by the Gram-positive, spore-forming bacterium *Paenibacillus larvae*. After more than a century of research it remains a fatal, highly contagious epizootic, and in many countries it is subject to mandatory reporting (Genersch 2010). Its destructiveness rests on extraordinarily durable spores.

## Key themes

### Infection biology: it's all about the spores

AFB begins when a young larva ingests *P. larvae* spores in its food; the spores germinate in the midgut, the bacteria proliferate and breach the gut, killing the larva and producing billions of new spores that persist for decades (Genersch 2010). Within-colony transmission occurs as nurse bees redistribute spore-contaminated honey and handle infected larval remains (Lindström 2008). This spore durability is why AFB is so hard to eradicate.

### Strain genotypes and virulence

*P. larvae* is differentiated into ERIC genotypes — originally ERIC I–IV, with ERIC V more recently discovered — that differ in virulence and colony-level dynamics (Genersch 2006; Beims 2020). ERIC-PCR genotyping characterises field isolates (Bassi 2015), and specific virulence factors, such as the S-layer protein SplA, have been functionally implicated in pathogenesis (Poppinga 2012).

### Control: from burning to biologicals

Because spores survive standard hygiene, the traditional control in many jurisdictions is destruction (burning) of infected colonies. Antibiotics such as tylosin are used in some countries but raise resistance and residue concerns (Okamoto 2021). Novel biological approaches are under investigation, including a bacteriophage-derived endolysin (PlyPl23) with potential to lyse *P. larvae* (Oliveira 2015).

### Conclusion

AFB is defined by its spores: their durability dictates the disease's contagiousness, the difficulty of eradication, and the radical control measures used. Surveillance, strain typing and emerging biologicals are the active frontiers, but vigilance and prompt removal of infected material remain the mainstay.

## Notable studies

**American Foulbrood in honeybees and its causative agent, Paenibacillus larvae**

*Genersch, Journal of Invertebrate Pathology 2010 · 293 citations* — The definitive review of AFB biology and the pathogen.

**Reclassification of Paenibacillus larvae**

*Genersch et al., Int. Journal of Systematic and Evolutionary Microbiology 2006 · 190 citations* — Establishes the modern taxonomy underlying ERIC genotyping.

**Discovery of Paenibacillus larvae ERIC V**

*Beims et al., International Journal of Medical Microbiology 2020 · 48 citations* — A fifth genotype, extending the virulence framework.

### Distribution of Paenibacillus larvae spores in adult bees and honey

*Lindström et al., Journal of Invertebrate Pathology 2008 · 45 citations* — How spores move within a colony.

### The first P. larvae bacteriophage endolysin (PlyPl23)

*Oliveira et al., PLoS ONE 2015 · 26 citations* — A biological control candidate.

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# European Foulbrood

A grounded synthesis of the most-cited open-access papers on European foulbrood. Every claim is traceable to a cited study; curated overview, not exhaustive.

## Overview

European foulbrood (EFB) is a globally distributed bacterial brood disease caused by *Melissococcus plutonius*, which infects and kills honey bee larvae and, in severe cases, can lead to colony collapse (Lewkowski 2019). Once considered a minor "stress" disease, EFB incidence has increased in several regions, prompting renewed research into its biology and control.

## Key themes

### A fastidious pathogen with hidden carriage

*M. plutonius* is a fastidious organism requiring microaerophilic-to-anaerobic culture conditions, which long hampered study (Arai 2012). Sensitive PCR detection revealed that the bacterium is present in many colonies *without* visible symptoms, so EFB carriage is far more widespread than overt disease — complicating diagnosis and control (Forsgren 2005).

### Diversity and virulence

The pathogen shows substantial genetic and phenotypic diversity, and this variation maps onto differences in virulence (Lewkowski 2019; Arai 2012). Researchers have begun identifying putative virulence determinants that distinguish aggressive from benign strains, work essential to predicting which infections will cause serious disease (Grossar 2020). Disease severity is also shaped by secondary bacterial invaders that colonise dying larvae (Lewkowski 2019).

### Predisposing stressors

Like chalkbrood, EFB expression is influenced by colony condition. Pesticide exposure — neonicotinoids and fungicides linked to immunosuppression — increased larval susceptibility to *M. plutonius* in controlled tests, tying EFB into the broader multi-stressor picture (Wood 2020). Outbreaks recur in commercial operations, where management pressures and stress converge (Thebeau 2022).

### Conclusion

EFB is a re-emerging disease whose impact depends on pathogen strain, secondary invaders and host stress rather than infection alone. Because the bacterium circulates symptomlessly, monitoring and reducing predisposing stress — alongside antibiotic treatment where permitted — are central to control.

## Notable studies

**Virulence of Melissococcus plutonius and secondary invaders in EFB**

*Lewkowski & Erler, MicrobiologyOpen 2019 · 38 citations* — Pathogen diversity and the role of secondary invaders in disease severity.

**Distribution of M. plutonius in colonies with and without EFB symptoms**

*Forsgren et al., Microbial Ecology 2005 · 28 citations* — Reveals widespread symptomless carriage.

**Diversity of M. plutonius and experimental reproduction of EFB**

*Arai et al., PLoS ONE 2012 · 65 citations* — Genetic diversity and experimental disease induction.

**Putative determinants of virulence in M. plutonius**

*Grossar et al., Virulence 2020 · 37 citations* — Candidate virulence factors for disease prediction.

**In vitro effects of pesticides on European foulbrood in larvae**

*Wood et al., Insects 2020 · 13 citations* — Pesticide exposure increases larval susceptibility.

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# Chalkbrood

A grounded synthesis of representative open-access papers on chalkbrood. Every claim is traceable to a cited study; curated overview, not exhaustive.

## Overview

Chalkbrood is a fungal brood disease caused by *Ascosphaera apis*, an obligate pathogen of honey bee larvae that occurs worldwide. Infected larvae are overgrown by fungal mycelium and die, hardening into the chalk-white "mummies" that give the disease its name (Li 2020).

## Key themes

### Infection requires spores plus a predisposing condition

Disease expression is conditional: a larva must ingest fungal spores *and* experience a predisposing stress — classically chilling of the brood — for chalkbrood to develop (Flores 2005). This explains why outbreaks often follow cool, damp conditions or colony stress rather than mere spore presence.

### Persistent spores and routes of infection

*A. apis* spores are highly durable. Critically, they can be present within sheets of **wax foundation** and remain infective, making contaminated comb and foundation a route of transmission and a reason the disease recurs (Flores 2005). The fungus can co-occur with other pathogenic fungi and even carry honey bee viruses (Cheng 2022).

### Pathogenesis and the search for control

There is no reliable registered treatment for chalkbrood, so research focuses on understanding pathogenesis and finding biocontrols. Molecular studies map how *A. apis* alters the larval gut — antioxidant enzymes, metabolomic profiles and regulatory RNAs during infection (Li 2020; Qiu 2026). Candidate interventions include plant essential oils with antifungal activity against *A. apis* (Ansari 2017) and biocontrol yeasts such as *Metschnikowia pulcherrima* (Iorizzo 2025).

### Conclusion

Chalkbrood is a conditional disease: durable spores plus brood stress produce outbreaks, which makes management as much about colony husbandry (warmth, ventilation, comb replacement, strong colonies) as about the pathogen. Antifungal and biocontrol options remain experimental.

## Notable studies

**Spores of Ascosphaera apis contained in wax foundation can infect honeybee brood**

*Flores et al., Veterinary Microbiology 2005 · 16 citations* — Establishes wax foundation as an infection route and the role of predisposing conditions.

**Changes in antioxidant enzyme activity and metabolomic profiles in chalkbrood-infected guts**

*Li et al., Insects 2020 · 28 citations* — Molecular pathogenesis of *A. apis* infection.

**In vitro evaluation of plant essential oils against Ascosphaera apis**

*Ansari et al., Saudi Journal of Biological Sciences 2017 · 28 citations* — Essential oils as candidate antifungals.

**Two pathogenic fungi isolated from chalkbrood samples and the viruses they carried**

*Cheng et al., Frontiers in Microbiology 2022 · 12 citations* — Co-occurring fungi and virus carriage.

**Antifungal activity of Metschnikowia pulcherrima against Ascosphaera apis**

*Iorizzo et al., Journal of Fungi 2025 · 4 citations* — A biocontrol yeast candidate.

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# Small Hive Beetle

A grounded synthesis of representative open-access papers on the small hive beetle. Every claim is traceable to a cited study; curated overview, not exhaustive.

## Overview

The small hive beetle (SHB), *Aethina tumida*, is an invasive nitidulid beetle native to sub-Saharan Africa that has spread globally and become a significant pest of *Apis mellifera*. Its larvae feed on honey, pollen and brood inside the hive; heavy infestations ferment and "slime" the combs, can cause bees to abscond, and may collapse the colony (Cornelissen 2020).

## Key themes

### A pest with a soil-dwelling weak point

The beetle's life cycle is the key to controlling it: wandering larvae leave the hive to **pupate in the soil** around the apiary. This soil stage is the most accessible target for control — entomopathogenic nematodes applied to soil can kill pupating larvae and reduce emergence, offering a biological alternative to in-hive chemicals (Sanchez 2021; Aryal 2025).

### Chemical ecology and trapping

SHB locates hives using volatile cues, and adult beetles are strongly attracted to hive products and to a yeast (*Kodamaea ohmeri*) that ferments pollen — a relationship exploited in lures and in-hive traps (Hayes 2015; Torto 2007). Understanding these attractants underpins monitoring and trap-based management (Torto 2007).

### Host factors and resistance

Colony strength and bee behaviour influence outcomes: comparisons of bee stocks found differences in susceptibility, with some lines better at limiting beetle invasion and reproduction (Frake 2009). Genomic work has catalogued candidate insecticide target sites in the beetle, informing future control chemistry (Rinkevich 2020).

### Conclusion

The small hive beetle is managed most sustainably by combining strong colonies, in-hive trapping guided by its chemical ecology, and soil treatment of the vulnerable pupal stage. As an invasive species it is also a biosecurity concern wherever it has not yet established.

## Notable studies

**Successful pupation of small hive beetle in greenhouse and field soils**

*Cornelissen et al., Journal of Economic Entomology 2020 · 3 citations* — The damaging biology and the soil pupation stage.

**Entomopathogenic nematode management of small hive beetles**

*Sanchez et al., Journal of Nematology 2021 · 5 citations* — Biological control targeting the soil-dwelling stage.

**Increased attractiveness of hive product volatiles to adult small hive beetle**

*Hayes et al., Entomologia Experimentalis et Applicata 2015 · 8 citations* — Chemical ecology behind lures and traps.

**Comparison of two attractants to small hive beetles in honey bee colonies**

*Nolan & Delaplane, Journal of Apicultural Research 2008 · 2 citations* — In-hive trap lures using pollen substitute and yeast.

**In silico identification of insecticide target sites in the SHB genome**

*Rinkevich et al., BMC Genomics 2020 · 5 citations* — Genomic basis for future control chemistry.

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# Tracheal Mites

A grounded synthesis of representative open-access papers on the honey bee tracheal mite. Every claim is traceable to a cited study; curated overview, not exhaustive.

## Overview

The tracheal mite *Acarapis woodi* is an internal parasite that lives and breeds inside the breathing tubes (tracheae) of adult honey bees. Discovered in 1919, it was historically a serious concern, but its perceived importance has faded since the arrival of *Varroa* — and its true role in colony mortality has been debated for a century (Kojima 2011; McMullan 2009).

## Key themes

### How it harms the bee

By colonising the tracheae and feeding on haemolymph through the tracheal walls, the mite impairs respiration. Experimental work shows infestation **reduces the safety margin for oxygen delivery in flying bees**, providing a physiological mechanism for the weakness and shortened life associated with heavy infestation (Harrison 2001).

### Host selection and resistance

The mite finds new hosts using chemical cues: it preferentially invades **young bees**, a preference mediated by cuticular hydrocarbons on the bee's surface (Phelan 1991; van Engelsdorp 2001). Because susceptibility is partly genetic, honey bees can be selectively bred for resistance — divergent selection has produced lines with markedly lower mite abundance, and resistance is linked to cuticular compounds that deter mite entry (Nasr 2001; Villa 2005).

### Detection and control

Diagnosis traditionally requires dissection, but PCR-based detection now allows more sensitive screening (Kojima 2011). Control has relied on menthol and, in combination with *Varroa* treatment, formic acid fumigation, which conveniently controls both mites at once (Maeda 2016).

### Conclusion

Tracheal mites are a reminder that *Varroa* is not the only mite of honey bees. Where they remain significant — and in regions like Asia where related species persist — resistant stock and integrated treatment (menthol, formic acid) keep them in check, often as a by-product of *Varroa* management.

## Notable studies

**Tracheal mites reduce the safety margin for oxygen delivery of flying honey bees**

*Harrison et al., Journal of Experimental Biology 2001 · 43 citations* — The physiological mechanism of harm.

**Mediation of host selection by cuticular hydrocarbons in the tracheal mite**

*Phelan et al., Journal of Chemical Ecology 1991 · 12 citations* — The mite prefers young bees via chemical cues.

**Resistance to Acarapis woodi by honey bees: divergent selection**

*Nasr et al., Journal of Economic Entomology 2001 · 4 citations* — Selective breeding produces resistant lines.

**PCR-based detection of the tracheal mite**

*Kojima et al., Journal of Invertebrate Pathology 2011 · 7 citations* — Sensitive molecular diagnosis.

**A qualitative model of mortality in colonies infested with tracheal mites**

*McMullan & Brown, Experimental & Applied Acarology 2009 · 11 citations* — The long-debated role in colony death.

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# Tropilaelaps

A grounded synthesis of the most-cited open-access papers on *Tropilaelaps* mites. Every claim is traceable to a cited study; curated overview, not exhaustive.

## Overview

*Tropilaelaps* mites are brood-parasitic mites originally found on the giant honey bees of Asia (*Apis dorsata*). Following a host switch to the Western honey bee, *Tropilaelaps mercedesae* now causes serious damage to *Apis mellifera* in Asia, where infestation can rapidly lead to colony mortality (Forsgren 2009; Anderson 2007). It is widely regarded as the next major mite threat should it spread beyond its current range (de Guzman 2017).

## Key themes

### A fast, damaging brood parasite

Like *Varroa*, *Tropilaelaps* reproduces in capped brood, but it is in some respects a faster and more damaging parasite. It feeds on both pre- and post-capped brood — feeding on pre-capped stages may help it survive the brief periods outside sealed cells — and this feeding causes severe brood damage and colony decline (Phokasem 2019; de Guzman 2017).

### It vectors viruses too

*Tropilaelaps* is not just a parasite but a virus vector: it has been found associated with Deformed Wing Virus in infested *A. mellifera*, and viral-load analyses compare *Tropilaelaps* and *Varroa* as co-occurring vectors — meaning a *Tropilaelaps* invasion would carry the same viral consequences that make *Varroa* so destructive (Forsgren 2009; de Guzman 2020).

### Surveillance and the biosecurity case

Because *Tropilaelaps* remains largely confined to Asia, the priority for the rest of the world is **early detection**. Rapid survey techniques and validated field/laboratory detection methods have been developed so that surveillance programmes can catch an incursion early enough to attempt eradication (Pettis 2013; Gill 2024).

### Control

Where it is established, control is complicated by the mite's biology — a very short phase outside the brood cell limits the window for contact treatments. Chemical and cultural methods, including brood interruption and hygienic removal of infested brood, are used with partial success (Pettis 2017; Khongphinitbunjong 2014).

### Conclusion

*Tropilaelaps* is a *Varroa*-like threat — a brood-reproducing, virus-vectoring mite — that has so far stayed in Asia. For most of the world it is fundamentally a biosecurity problem: surveillance to detect, and contingency planning to contain, an incursion before it establishes.

## Notable studies

**Ecology, life history, and management of Tropilaelaps mites**

*de Guzman et al., Journal of Economic Entomology 2017 · 31 citations* — The comprehensive review of the genus and its management.

**Deformed wing virus associated with Tropilaelaps mercedesae infesting European honey bees**

*Forsgren et al., Experimental & Applied Acarology 2009 · 51 citations* — Establishes *Tropilaelaps* as a DWV vector.

### Feeding by Tropilaelaps mercedesae on pre- and post-capped brood

*Phokasem et al., Scientific Reports 2019 · 16 citations* — Feeding biology and brood damage.

### A rapid survey technique for Tropilaelaps mite detection

*Pettis et al., Journal of Economic Entomology 2013 · 15 citations* — Surveillance method for uninfested regions.

### Genetic and morphological variation of bee-parasitic Tropilaelaps mites

*Anderson & Morgan, Experimental & Applied Acarology 2007 · 50 citations* — Species delimitation and host range.

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