

Apis mellifera

Subjects: Zoology

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The current knowledge about viruses of the Western honey bee, *Apis mellifera*.

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This entry is a summary of the review "[Diversity and Global Distribution of Viruses of the Western Honey Bee, *Apis mellifera*](#)" published in MDPI Insects ([doi:10.3390/insects11040239](#)) from members of the [COLOSS Virus Taskforce](#).

1. What are Viruses of *Apis mellifera*?

Viruses are submicroscopic parasites that can only replicate inside the cells of a living organism: they are essentially a small nucleic acid genome packaged in a protective shell and they cannot reproduce independently. Viruses are a universal feature of all forms of life, and thus also of honey bees (*Apis mellifera*). A growing number of viruses has been documented and several of these contribute significantly to honey bee colony losses. Viruses are both the simplest organisms (essentially a small nucleic acid genome packaged in a protective shell) and the most complex organisms, since many of their key traits are expressed indirectly through their host. The viruses' absolute dependency on a continuous supply of new hosts for its survival requires an infection strategy balancing the needs of the virus with those of its host(s). This balance is dynamic and requires continual, and rapid, adaptability by the virus. This adaptability is mediated by the high variability of the virus genome, and is expressed through physical, physiological and behavioural changes in the host, that frequently affect virus transmission.

Disease or death of the host is not inevitable but rather an adaptive feature: useful only if it furthers transmission and subject to change if needed. Most known bee viruses are therefore asymptomatic, with only a few causing major disease. These viruses can act synergistically with other biotic and abiotic stressors, compromising individual and colony health and functionality. Most viruses infecting honey bees can also infect a wide range of other insect species, mainly other bee species, and are thus part of a more complex disease ecology involving other pollinators, flowering plants and pollination networks. Human industry and globalization have magnified the scale of virus re-distribution across the world, and its risks for local pollinators and pollinator-dependent plants, exemplified by the increasing incidence of emerging infectious diseases (EIDs). In a context of global change, understanding the distribution and spread of diseases is essential to designing appropriate control and containment strategies.

2. Diversity

Viruses of honey bees have historically been discovered through their symptoms, which can be either physical (DWV, CBPV, CWV, AmFV, AIV), developmental (SBV, BQCV), behavioral (DWV, CBPV, ABPV, SBV, SBPV) or demographic (BVX, BVY), and more recently also through high-throughput sequencing (HTS) technologies (Table 1). The overwhelming majority of viruses are asymptomatic, particularly the newly discovered ones, and only a miniscule proportion cause symptoms or disease. Some bee viruses (e.g. DWV, ABPV) are associated with parasitic mites, such as *Varroa destructor* and *Tropilaelaps mercedesae*, that transmit the virus between honeybees. These mites are called 'vectors' of virus transmission. The transmission by vectors can affect the genetic composition of the virus and its virulence, as the virus adapts to a potent new transmission route. The extent of this virological biodiversity by itself poses questions about its origins and functional significance of viruses, either as former/future pathogens or molecular symbionts. The accumulation of honey-bee-derived virus genome sequences needs to be completed by molecular, biological and epidemiological character studies in order to understand their possible functional and ecological roles and thus their current relevance and possible future significance.

3. Transmission

Viral transmission can be divided into horizontal and vertical transmission (Figure 1). Horizontal transmission is the transmission of infectious agents among individuals of the same generation. Horizontal transmission of viruses in honey bees includes transmission to different bee developmental stages via oral (i.e. trophallaxis) and/or body contact. It includes indirect infections through contaminated food such as pollen and larval food (food-borne transmission); and contact with feces. This oral-fecal route is arguably the most common route for bee virus transmission, both within honey bee colonies and between different bee species. There is ample evidence that supports this route of transmission for most viruses found in honey bees. These viruses are mostly shed in copious amounts into the feces, from where they are released into the environment and can be picked up by other bees, through shared floral resources. Horizontal transmission also includes venereal transmission, where virus is transmitted from drones to queens during the nuptial flights or by artificial insemination, and vector-mediated transmission, where transmission is mediated by other organisms (i.e. *V. destructor*, *Tropilaelaps* spp., *Aethina tumida*).

Vertical transmission consists of the transmission of viruses to the next generation, which for honey bees is primarily from queens to their eggs. This transmission can be defined as either transovum or transovarial transmission, depending on whether viruses are transmitted on the egg surface or within the egg, respectively. A second form of vertical transmission is transspermal transmission, if the virus is present inside the sperm, which would be the drone equivalent of transovarial transmission. However, this has not been detected in honey bees so far.

Figure 1 - overview of the transmission routes of viruses of bees

A comprehensive review on transmission of viruses of *A. mellifera* can be found in [Bee viruses: routes of infection in Hymenoptera](#).

4. Symptoms

The pathogenicity of viruses of honey bees is the consequence of their replication within the cells of their hosts. Some of the viruses have tropism towards many organs, while other viruses are restricted to specific organs (see Table 1). Thus, if symptoms appear in an individual bee, they are a direct outcome from the virus interrupting the function of one or more organs/systems in the bee's body. However, clear symptoms usually only appear at very high virus titers, and many persistent or asymptomatic infections that may cause long-term damage to the colony can remain undetected.

Many bee viruses do not produce clear physical or behavioral symptoms in honey bees, while for others symptoms may exist but have not yet been identified. Many viruses are present in seemingly healthy colonies as asymptomatic, covert infections. In most cases some promoting factor, like varroosis (i.e. infestation of the colony by varroa mites) are necessary to turn these pathogens into a symptomatic infection. The honey bee viral diseases that have been most extensively studied usually possess one or more distinctive symptoms that can help make diagnoses. Virus pathology, symptom development and virulence depend on several factors (mostly amount of virus produced, where in the body it is produced and its transmission route). For instance, the occurrence of symptoms of deformed wing virus infections strongly depends on the transmission route, namely, it appears that infections through vertical, venereal or oral transmission are effectively symptomless, even if the virus titers are as high as those achieved by Varroa-mediated transmission.

Table 1. Symptoms of viral infections in *A. mellifera*. Tissue tropism refers to the organs in which the virus was found. Symptoms report the physical and physiological effect of viruses as observed in honey bees. Only viruses with known symptoms are reported here.

Virus	Tropism	Symptoms
<i>Acute bee paralysis virus complex</i>	Nervous system, cytoplasm of fat body cells, brain, and hypopharyngeal glands	Trembling, inability to fly, gradual darkening and loss of hair from the thorax and abdomen, crawling on the ground, and upward on grass, rapid death for highly infected bees
<i>Apis iridovirus</i>	NA	Iridescence of most internal organs
<i>Apis mellifera filamentous virus</i>	NA	Milky-white haemolymph
<i>Bee virus X</i>	NA	Shortened lifespan of adult bees
<i>Bee virus Y</i>	NA	Shortened lifespan of adult bees
<i>Black queen cell virus</i>	Gut tissue	Yellowish queen larvae with sac- appearance that resembles SBV and with time evolves to dark brown, infected pupae turn brown and die, dark brown to black coloured walls in queen cells , Significantly shortened life span in adult bees
<i>Chronic bee paralysis virus</i>	Nervous system, alimentary tract, mandibular, and hypopharyngeal glands	Syndrome 1: trembling of the wings and bodies, bloated abdomen, inability to fly, crawling on the ground, and upward on grass, gather in groups in the warmest areas of the nest, death within few days Syndrome 2 ('black robbers'): hairless (thus appearing smaller), darker, greasy in appearance, shiny suffer nibbling attacks by the healthy bees, death within few days
<i>Cloudy wing virus</i>	Tracheal tissue and thoracic muscles	opaque wings, shortened lifespan of adult bees
<i>Deformed wing virus</i>	Whole body, including the queen ovaries, queen fat body, spermatheca, and drone seminal vesicles, tissues of wings, head, thorax, legs, hemolymph, and gut	crumpled or aborted wings, shortened abdomens, paralysis, severely shortened adult life span for emerging bees, modified responsiveness to sucrose, impaired learning , impaired foraging behavior
<i>Invertebrate iridescent virus Type 6</i>	NA	flightless clustering bees

<i>Sacbrood virus</i>	Hypopharyngeal glands of worker bees, cytoplasm of fat, muscle, and tracheal-end cells of larvae	Pupation failure, 'sac' phenotype: swollen larvae filled of ecdysial fluid full of viral particles, precocious foraging, reduction of adult life span and metabolic activities, impaired foraging activity
<i>Slow bee paralysis virus</i>	Nervous system	Paralysis of the two anterior legs a day or two before death

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