

Common Viruses of Florida Honey Bees: Diagnosis and Management¹

James C. Fulton, Kiley J. Epperson, and James D. Ellis²

Introduction

Viruses can have a significant negative economic impact on the international and domestic apiary industries. Globally, more than 70 viruses have been associated with *Apis mellifera*, the western honey bee (hereafter honey bee). Of these viruses, only a small proportion causes noticeable adverse clinical signs (Beaurepaire et al. 2020). It is common for an individual bee to be infected by multiple viruses without obvious consequence, while an entire colony can be affected by even more viruses (Ribière et al. 2008). Often, the impact from these viruses is restricted by individual-level or colony-level defenses (Chen and Siede 2007).

Honey bees demonstrate multi-tiered immune responses, individually and colony-wide, when challenged by viruses. Individually, a bee's physiological and cellular response limits the impact of potential pathogens (Brutscher and Flenniken 2015; Maori et al. 2009). A colony's collective response may include brood-comb fever (Starks et al. 2000), altruistic self-removal of adult bees (Rueppell et al. 2010), culling and removing infected brood via hygienic behavior (Mondet et al. 2016).

Despite these defenses, external stressors such as elevated mite infestation levels (Traynor et al. 2020), inadequate nutrition (DeGrandi-Hoffman and Chen 2015), or human-caused disturbance of the surrounding ecology (McMahon et al. 2018) can allow viruses to cause significant damage. In fact, the significance of viruses to colony health has been demonstrated by studies showing strong correlations between viral infection and colony loss (Cox-Foster et al. 2007; Grozinger and Flenniken 2019). The purpose of this article is to provide beekeepers an overview of bee viruses commonly found in Florida, virus impact on colony health, associated clinical signs, and transmission pathways (Table 1).

Biology

Viruses are invisible to the naked eye. Under an electron microscope, viral particles are found in many shapes, including rods, spheres, and filaments. They are comprised of nucleic acids (single-stranded or double-stranded RNA

or DNA) and a protein coat. Viruses are composed of compact genomes; therefore, they are entirely dependent on their host for replication. They repurpose host cellular machinery to produce their own components. Most described honey bee viruses are RNA viruses; however, a few DNA viruses have also been reported to be associated with honey bees (Amiri et al. 2021).

Clinical Signs

Viruses are often present without causing obvious distress in infected honey bees. In other cases, infections can persist subclinically as chronic disease. When clinical signs are present, they may include physical deformities, paralysis, trembling, shortened lifespans, abrupt mortality, cuticular (body) discoloration, and cognitive impairments. Each virus causes unique disease expression across the various bee life stages (brood, adult, etc.), and the prognosis of an individual bee can vary from no clinical signs to death within a few days.

Diagnosis

For honey bee viruses, detection and diagnosis are confirmed using two main techniques: enzyme-linked immunosorbent assay (ELISA) or reverse-transcriptase (quantitative) polymerase chain reaction [RT-(q)PCR]. ELISA is a protein-based serological technique that uses antibodies to detect the presence or absence of a disease target. RT-(q)PCR is a nucleic-acid-based technique. In this process, RNA is converted into single-stranded complementary DNA (cDNA), which is then used in the PCR. For conventional RT-PCR, the product is visualized on an agarose gel to determine presence or absence of the target gene. For RT-(q)PCR, cDNA amplification is visualized on a computer, and viral loads can be quantified in real time. Because these techniques require trained laboratory technicians and specific equipment, sending suspect samples to one of the honey bee diagnostic laboratories in North America is recommended. ApiaryInspectors.org has [a list of apiary inspectors of America](#).

Epidemiology

Honey bee viruses can spread by various mechanisms, including from queen to egg, orally during trophallaxis (feeding one another), by exposure to fecal material, and by bodily contact. However, one of the most significant factors contributing to the heightened impact of viruses to apiary health is the presence of *Varroa destructor*, a parasitic mite that feeds and reproduces on honey bees. In addition to causing direct damage to honey bee tissue, predation by *V. destructor* also depresses honey bee immunity, impairs detoxification processes, and causes malnutrition as the honey bee physiology responds to constant predation (Morfin et al. 2023). These detrimental effects are aggravated by *V. destructor*'s capacity to vector several honey bee viruses (Damayo et al. 2023). In fact, the spread of *V. destructor* has caused significant increases in the number and diversity of viruses afflicting individuals and colonies (Doublet et al. 2024). The impact of each virus depends largely on whether the virus is vector transmitted.

Management

Unfortunately, no chemical applications are available for the direct treatment of viral diseases of honey bees. Instead, beekeepers are encouraged to maintain conscientious beekeeper practices to ensure that colonies are successful (Bartlett et al. 2021).

- *Varroa destructor* is responsible for transmitting several honey bee viruses and weakens honey bee populations through parasitism. Consequently, adept use of acaricides and integrated pest management strategies that keep populations of *V. destructor* low/controlled is an essential component of any disease-management program (Locke et al. 2017; Jack and Ellis 2021). See also “[Varroa Management](#),” available at HoneyBeeHealthCoalition.org, for information related to controlling *V. destructor* in honey bee colonies.
- Ensure colonies have access to adequate nutrition (DeGrandi-Hoffman et al. 2010). Access to an abundance of polyfloral pollen will support colony health during viral exposures (Dolezal et al. 2019).
- When choosing queen stock, choose lineages that possess innate resistance or enhanced altruistic behaviors such as self-removal or diseased/dead brood removal, i.e., bees that express hygienic behavior (Wagoner et al. 2019).
- Manage for other pathogens and pests, including *Melissococcus plutonius* (causative agent of European foulbrood) and small hive beetles (*Aethina tumida*), to reduce colony stress, thus likely lessening the impact of viral disease.
- Provide a hygienic environment by sterilizing hive tools, replacing worn and/or degraded hive equipment, and limiting material transfer between apiaries.

- Quarantine and remove or sanitize infectious material (e.g., used frames, dirty extraction equipment, etc. [de Miranda 2012]) to interrupt viral epidemics. For example, if a particular colony develops severe disease pressure, then that colony can be removed from an apiary and the constituent hive material (i.e., frame, foundation, etc.) sanitized and stored until the following season.
- Minimize pesticide exposure, because viral lethality and honey bee susceptibility to infection have been correlated with pesticide exposure (McMenamin et al. 2016).

Common Florida Honey Bee Viruses

Knowledge and understanding of honey bee viruses in Florida are vital for the overall health of these pollinators. Within Florida, 14 viruses are routinely detected, with eight of those posing a particular threat to the industry (Table 1). Common honey bee viruses include acute bee paralysis virus (ABPV), Kashmir bee virus (KBV), Israeli acute paralysis virus (IAPV), black queen cell virus (BQCV), chronic bee paralysis virus (CBPV), deformed wing virus (DWV), Lake Sinai virus (LSV), and sacbrood virus (SBV). Detection and diagnosis allow for proper management techniques as well as stronger colonies.

Acute Bee Paralysis Virus/Kashmir Bee Virus/Israeli Acute Paralysis Virus

Closely related viruses ABPV, KBV, and IAPV share many characteristics that form the acute bee paralysis virus–Kashmir bee virus–Israeli acute paralysis virus complex in the single-stranded RNA Dicistroviridae virus family (de Miranda et al. 2010). Acute bee paralysis virus is often found in honey bee colonies without overt clinical signs and is possibly spread through trophallaxis (Yañez et al. 2020). When *V. destructor* populations become large in a colony, this virus can lead to high bee mortality rates (Bakonyi et al. 2002). Furthermore, ABPV is implicated in colony collapse (Šimenc et al. 2021). Clinical signs may include hair loss and cuticular darkening in adult bees (Amiri et al. 2021) (Figure 1).



Figure 1. Clinical signs representative of *Acute bee paralysis virus*. The adult worker bee is missing most of its hair, a common clinical sign of virus infection, and appears “greasy.”
Credit: UF/IFAS Honey Bee Research and Extension Lab

Kashmir bee virus can infect both brood and adult honey bees. Infected adults often quickly die after inoculation. Low levels of infection are known in the absence of *V. destructor* but can reach epidemic levels when mite populations are high (Chen et al. 2004). Overt signs besides death are not apparent (Riveros et al. 2018).

Similar to ABPV and KBV, IAPV can be spread through trophallaxis and *V. destructor* infestation (Chen et al. 2014). Clinical signs can vary from asymptomatic infection to trembling, paralysis, death (Formato et al. 2011), and behavioral changes (Geffre et al. 2020), ultimately leading to high colony losses (Maori et al. 2009).

Black Queen Cell Virus

Black queen cell virus is another member of the Dicistroviridae family of RNA viruses. As the name suggests, one of the most visible clinical signs associated with this pathogen is the darkening of dead queens in queen cells (Figure 2). The immature queens developing in these cells die from infection and turn black. Black queen cell virus is one of the most common honey bee viruses and can be found globally (Tentcheva et al. 2004). It is spread through trophallaxis, vertical transmission from queen to eggs, and in worker-produced royal jelly (Chen et al. 2006). In adult bees, this pathogen is found in co-infection with *Nosema apis* and *Malpighamoeba mellificae* (Bailey et al. 1983).



Figure 2. Clinical signs representative of black queen cell virus. The immature honey bee queen is dead and has turned black in the queen cell.
Credit: UF/IFAS Honey Bee Research and Extension Laboratory

Chronic Bee Paralysis Virus

Chronic bee paralysis virus is a global disease of increasing significance (Budge et al. 2020). Two sets of clinical signs exist, Type 1 and Type 2 (Ribi  re et al. 2010). Type 1 infection is categorized by flightless bees with bloated abdomens and trembling wings (Figure 3). Type 2 infection consists of darkened, shiny bees that appear greasy and hairless. Initially these bees can fly, but after a few days they lose the ability to fly. Inoculated bees survive more than 12 days (Bailey et al. 1963). This virus is spread through physical contact with infected bees and contact with contaminated feces (Amiri et al. 2014).



Figure 3. Chronic bee paralysis virus can cause bees like this worker honey bee to appear hairless, nearly entirely black, shiny, and greasy.
Credit: Professor Giles Budge, Newcastle University, used with permission

Deformed Wing Virus

Deformed wing virus is a highly common honey bee virus with four variant strains (A, B, C, and D). It is a positive, single-stranded RNA virus in the *Iflaviridae* family of viruses. One study demonstrated that across 32 countries, over 55% of colonies were affected by at least one strain of DWV (Martin and Brettell 2019). As the name suggests, the most obvious clinical sign of infection includes deformed wings (Figure 4). However, only about 1% of infected bees display this clinical sign (Brettell et al. 2017). Many infected bees will also present bloated abdomens, impaired cognition, erratic behavior, and shorter lifespans (Kevill et al. 2021). However, abnormalities in physiology and/or anatomy usually only occur when viral titers are extremely elevated. The virus is strongly associated with *V. destructor*, with regions without established mite populations having little or no DWV infections (Martin and Brettell 2019). Other forms of DWV spread include reproduction, resource sharing, and trophallaxis (Martin and Brettell 2019).



Figure 4. Worker honey bee with stunted, misshapen wings, clinical signs of deformed wing virus.

Credit: J. D. Ellis and C. M. Zettel Nalen 2022

Lake Sinai Virus

First discovered in 2008 (Runckel et al. 2011), LSV has several strains, with LSV 1 and 2 being the most common. While LSV seems to be a latent virus, producing no apparent signs of infection, there is a correlation between high viral loads and weaker colonies with low populations (Daughenbaugh et al. 2015). There is evidence that LSV is not associated with *V. destructor*. Its mode of infection is not clearly understood (Hou et al. 2023).

Sacbrood Virus

Sacbrood virus, an RNA virus, infects the brood of honey bees. Larvae infected with this virus die before their final molt. Fluid accumulates at the bottom of the larva as viral particles replicate in the larva and prevents it from shedding the endocuticle (Li et al. 2019; Wei et al. 2022) (Figure 5). Subsequently, this causes larval mortality, turning the larva from pale yellow to dark brown

(Grabensteiner et al. 2000). The clinical signs are similar to those associated with American foulbrood (spotty brood pattern, discolored/sunken/perforated brood cell cappings). The key difference is that the dead individuals do not adhere to the cell walls. Sacbrood virus can affect adults with no obvious clinical signs of infection, but the virus can also shorten the lifespan of infected bees (Grabensteiner et al. 2000). SBV infections are spread multiple ways, including possibly through *V. destructor*, reproduction, and trophallaxis (Wei et al. 2022). Sacbrood is most prevalent in the spring due to changing temperatures (Li et al. 2019).



Figure 5. Classic clinical signs of Sacbrood virus include dead immature honey bees (larvae or prepupae), with the cuticle forming a sack and fluid accumulating at the bottom of the sack. At first, the prepupa darkens slightly to brown, with the third of the body with the head darkening to almost black.

Credit: Joachim de Miranda, Swedish University of Agricultural Sciences, used with permission

Conclusion

Bee virology is a challenging field, but our knowledge of viral pathogens, including their effects on the honey bee host and ecological interactions, is constantly improving. Surveillance and monitoring are necessary components of safeguarding the industry. Florida beekeepers are encouraged to use resources available to them, including many available through their Florida Department of Agriculture and Consumer Services Division of Plant Industry. Find a local [apiary inspector](#) and/or a [honey bee](#)

diagnostics laboratory, both available, along with an array of other valuable bee resources, at fdacs.gov.

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Table 1. Common Florida honey bee viruses.

Virus	Genus	Clinical Signs	Potential Impact [€]	Transmission					
				Oral	Fecal	Body contact	Vene-real	Vec-tor	Queen to eggs
Acute bee paralysis virus complex*	<i>Aparavirus</i>	ABPV (Acute bee paralysis virus) <ul style="list-style-type: none"> • Shortened lifespan • Colony decline • Flightless bees • Paralysis • Discoloration and hairlessness on abdomen and thorax • Occasional foulbrood-like signs of infection in affected brood KBV (Kashmir bee virus) <ul style="list-style-type: none"> • Colony decline • Clinical signs inconspicuous IAPV (Israeli acute paralysis virus) <ul style="list-style-type: none"> • Shortened lifespan • Flightless bees • Paralysis • Discoloration and hairlessness of abdomen and thorax • May increase movement between colonies, increasing disease spread 	Medium	+	+	~	~	+Vd	+

Virus	Genus	Clinical Signs	Potential Impact [€]	Transmission					
				Oral	Fecal	Body contact	Vene-real	Vec-tor	Queen to eggs
Black queen cell virus	<i>Triatovirus</i>	<ul style="list-style-type: none"> • Most noticeably affects developing queens • Pupae initially turn yellow and appear as a fluid sack; they eventually turn brown to black as they decay • Darkened cell walls • Affects brood and workers but typically covertly; however, may affect learning and longevity 	Low	+	+	?	~	+Na/c	+
Chronic bee paralysis virus	Unclassified	<ul style="list-style-type: none"> • Flightlessness • Paralysis • Trembling • Blackened and hairless abdomen and thorax 	Medium	+	+	+	-	-/+Vd	+
Deformed wing virus (A, B, C, D)	<i>Iflavirus</i>	<ul style="list-style-type: none"> • Premature pupa death • Deformed wings on adult bees • Smaller abdomens • Cognitive issues • Spotty brood pattern 	High	+	+	-	+	+ Vd, ~At	+
Lake Sinai virus**	<i>Sinivirus</i>	<ul style="list-style-type: none"> • Apparently does not cause clinical signs • Associated with weaker colonies 	?	+	?	?	-	-	+

Virus	Genus	Clinical Signs	Potential Impact [€]	Transmission					
				Oral	Fecal	Body contact	Vene-real	Vec-tor	Queen to eggs
Sacbrood virus	<i>Iflavirus</i>	<ul style="list-style-type: none"> • Premature death of larvae • Yellow, discolored larvae with head region darkening to almost black • Larva appears as fluid-like sac • Larva dries into brown scale. • Adult bees may not present clinical signs but can have a shorter lifespan • Spotty brood pattern, sunken or perforated brood cell capping 	Medium	+	-	-	~	-	+

Table modified from Yañez et al. 2020 and Beaurepaire et al. 2020

*Acute bee paralysis virus complex includes ABPV, KBV, IAPV. These viruses are closely related and share important characteristics such as transmission and disease descriptions. (de Miranda et al. 2010)

**Multiple strains have been described including LSV1, LSV2, LSV3, LSV4, LSV5, LSV6, and LSV7

Vd = *Varroa destructor*

At = *Aethina tumida* (small hive beetle)

Na/c = *Nosema apis/cerana*

Classification of impact potential according to Bartlett 2022

+ indicates an established association

- indicates that there is not an association based on experimental data

~ indicates an unclear association

€ Potential Impact category based on Bartlett, L. J. 2022

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² James C. Fulton, research scientist, Honey Bee Diagnostic Laboratory, FDACS Division of Plant Industry; Kiley J. Epperson, biological scientist, Honey Bee Diagnostics Laboratory, Division of Plant Industry, Florida Department of Agriculture and Consumer Services; James D. Ellis, Gahan endowed professor, apiculture, Honey Bee Research and Extension Laboratory, Department of Entomology and Nematology; UF/IFAS Extension, Gainesville, FL 32611.

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