ALBERTA TECH TRANSFER PROGRAM

The Ultimate Key to Honey Bee Viruses

A detailed guide on how to identify, understand and manage honeybee viruses

ALBERTA BEEKEEPERS COMMISSION

CONTENTS

Introduction

The battle with viral infections is constant within the beekeeping industry, and it is important that beekeepers know the identification, transmission, and management of bee-related viruses. Some of the most common viruses in Alberta include: Deformed Wing Virus (DWV or DWV-A), Varroa Destructor Virus (VDV or DWV-B), Chronic Bee Paralysis Virus (CBPV), Black Queen Cell Virus (BQCV), and Sacbrood Virus (SBV). When present on their own, these viruses have historically been undetectable, causing no major harm to colony health[1]. However, when present in conjunction with other bee-related diseases, viruses can flourish, and cause bigger problems over time. Additionally, when viral levels are higher than usual, other factors such as weather, stress, colony strength, and nutrition can play a role in increasing the level of infection within the colony[2].

One major factor that correlates with viral infections in bees is high Varroa mite infestation within colonies, which can lead to increased viral levels[1]. Varroa mites are a source of transmission for viral diseases, as they can function as a mechanical vector for most viruses, and a biological vector for viruses such as DWV and VDV[1]. A mechanical vector transmits the virus without becoming infected itself, while a biological vector allows for viral replication before transmitting it to a host[3]. Furthermore, mites can cause immunosuppression in bees, making colonies more susceptible to infection when exposed to viral pathogens[1].

The process in which a virus is transmitted is paramount to its persistence, viability, and spread within a host population[4]. The social structure of honey bee colonies creates many opportunities for viral transmission, which includes mainly activities that involve high contact rate between individual bees[4]. For example, bee-bee interactions such as comb building, foraging, cleaning brood cells, rearing offspring, and attending to the queen, all provide opportunities for viral spread[4]. While transmission among colonies and apiaries can happen at shared feeding sites , as well as through drifting and robbing behaviour[5].

Viral transmission can be divided into two major categories: horizontal and vertical transmission. Horizontal transmission, the most common route of infection for bee viruses, is defined as the spread of a virus within the same generation[3]. Specific routes of horizontal transmission include: oral-oral or oral-fecal, topical or bodily contact, venereal, and vector-based transmission[3]. Vertical transmission is defined as the transmission of a virus to the following generation, primarily from queen to offspring[3]. Within honey bee colonies, this form of transmission can either be transovum or transovarial[3].

- **Oral-oral or oral-fecal transmission** is when bees ingest virally contaminated food, feces, or other hive products, and become infected[6].
- **Topical transmission** is caused by increased rubbing, grooming, and general body contact between infected and healthy bees[3]. More specifically, it is the application of viral particles on newly exposed honey bee cuticle; or in other words, when a bees' protective cuticle layer has broken, giving the virus access to the epidermal cytoplasm[3,6].
- **Venereal transmission** is the spread of a virus between two sexes during mating[4]. More clearly, it's the transmission of a virus from drone to queen exclusively during mating flights[3].
- **Vector-based transmission** is a form of indirect transmission, as viruses are harboured within a living vector, such as the *Varroa destructor* mite, and are then passed on to a healthy host during parasitic behaviours[4].
- **Transovum** is the transmission of a virus on the outside of an egg[3].
- **Transovarial** is the transmission of a virus within the egg[3].

Deformed Wing Virus (DWV)

DESCRIPTION

Deformed Wing Virus is one of the most prevalent viral pathogens of honey bees, targeting all bee life stages: egg, larva, pupa, and adult[2]. It's common for DWV to be present at low levels in a healthy bee population[7]. However, as viral levels increase, colony health decreases and clinical signs of disease can become inherently more visible[7]. The main visual sign associated with DWV infection is malformed wings, from which the virus gets its name. Bees are born with poorly developed wings, which appear to be shrunken and crumpled as though they have been chewed off[1]. Additional signs of infection include pupa death, shrunken abdomen (small bees), and discoloration in adult bees, causing infected bees to appear lighter and 'washed-out'[7]. One of the main effects of this virus on worker bees is a shortened lifespan and decreased contribution to hive activities during their lifetime[2]. At the colony level, studies have shown that DWV-infected colonies have a lower brood and adult bee population, likely due to the effect of this virus on individual bees (i.e., premature death and decreased performance in hive activities)[2]. Due to these impairments on colony population and health, infected colonies may have decreased honey production and increased overwinter mortality[2].

KEY IDENTIFIERS

Figure 1. Bees with deformed wings.

TRANSMISSION

The ability of DWV to cause a serious threat upon infection is minimal, as bees infected with DWV do not instantly die. Instead, replication of the virus within the bee happens slowly, but constantly, and the bee maintains the infection during its lifespan[1]. As a result, DWV can persist within a colony at low levels without causing major health issues. The main source of DWV transmission is **vector-based** transmission via the *Varroa destructor* mite[2]. It has been found that DWV can replicate within the mite before spreading to honey bees, increasing the quantity of virus particles transmitted[3]. Therefore, varroa mites act as both mechanical and biological vectors for DWV[3]. Other less frequent modes of transmission include oral-fecal, venereal, and vertical. Viral levels of DWV were found to be highest in the gut, indicating that **oral-fecal** transmission pathways (i.e., trophallaxis activities, cell/hive cleaning, cannibalism) are potential routes of DWV infection[3,4,6]. Deformed wing virus has also been detected in the queen spermatheca (queen's internal organ responsible for storing semen), indicating that this virus can be **venereally** transmitted from drone to queen[3]. Vertical transmission of DWV has also been confirmed, as the virus has been detected in queen ovaries and/or eggs[3]. In this case, DWV is transmitted from the queen to her offspring via **transovarial** transmission, when viruses in ovarian tissue spread to developing eggs before being released[4].

Figure 2. Small bees, indicated by the white arrows.

Varroa Destructor Virus (VDV)

DESCRIPTION

Varroa Destructor Virus (aka DWV-B or VDV-1), is also one of the most prevalent viruses of honey bees, following DWV[8]. VDV is a close genetic relative of DWV and presents similar clinical signs of infection. These include: wing deformities in newly emerged bees, pupal mortality, shortened lifespan, small bees, and increased overwinter mortality[6]. However, many bees infected with VDV appear to be asymptomatic[9]. While signs of VDV infection can be similar to DWV, the former has shown to be more virulent, meaning that disease infection can progress more quickly and cause more harm to the colony[10]. Over the past 10 years, VDV levels have increased significantly and have become more widespread[8]. Researchers predict that VDV will become the dominant DWV variant and one of the main predictors of overwinter colony losses[8,11].

KEY IDENTIFIERS

Figure 1. Bee with deformed wings.

Figure 2. Small bees, indicated by the white.

TRANSMISSION

Similar to DWV, the main route of VDV transmission is **vector-based** transmission via the *Varroa destructor* mite[6]. This virus is strongly associated with mite infestation as Varroa mites act as both mechanical and biological vectors, causing rapid increases in viral load within infected colonies[3]. It has been found that VDV replicates at a much faster rate, compared to DWV, in both the Varroa mite and the honey bee, making VDV the more virulent DWV variant[10]. Transmission can also happen via the same routes as DWV: **oral-oral/oral-fecal**, **venereal**, and **vertical** routes. The presence of VDV in pollen and honey samples suggests that the consumption of these contaminated food products can introduce viruses into the bee digestive tract, and ultimately infect healthy bees via the oral-oral/oral-fecal route[3,4].

Figure 3. Bees with parasitic Varroa mites.

Chronic Bee Paralysis Virus (CBPV)

DESCRIPTION

Chronic Bee Paralysis Virus is an emerging viral pathogen of honey bees within Canada[5]. Over the past decade, cases of CBPV in commercial honey bee colonies have risen exponentially in various parts of the world (i.e., North America, Asia, Europe and the U.K)[5,12]. Clinical signs of disease associated with this virus are:

- Abnormal trembling in adult bees[5]
- Bloated abdomens[1]
- Adult bees exhibiting abnormally positioned wings that look disjointed ("K-wing"), limiting flight[1]
- Increasing crawling behaviour due to partial paralysis[1]
- Bees that have no hair and appear black and shiny[1,5]

Although CBPV has been detected in all life stages of bees, paralysis has only been observed in infected adult bees[6]. A common indication that a colony has high levels of CBPV is an increased number of bees crawling and shaking in front of the hive entrance[1]. It takes approximately 6 days for infected adult bees to develop clinical signs, with symptomatic bees usually dying within a week[5,6]. This can potentially lead to large mounds of dead bees present in front of the hive entrance and possible colony collapse[5]. Colonies that are heavily infected can become very weak, reducing the hive's ability to pollinate and produce honey[5].

KEY IDENTIFIERS

Figure 1. Hairless bee appearing black and shiny.

Figure 2. Bee with wings that look disjpointed ("K-wing").

TRANSMISSION

Levels of CBPV can increase during periods of overcrowding caused by events that reduce foraging activity, such as poor weather, limited nectar availability, and a high number of colonies per unit area[6]. **Topical** transmission happens during these events as there is an increase in the number of bees present in the hive for extended periods of time; therefore, increasing the rate of rubbing and contact between healthy and unhealthy bees[13]. This form of transmission requires a significant number of viral particles to be present and has only been linked to CBPV[14]. It is also known that CBPV can be transmitted via the **oral-fecal** route, as it has been detected in honey samples and feces of infected adult bees, leading to increased bee contact with viral particles[3,4]. Contact with CBPV-contaminated feces can trigger an overt infection (i.e., exhibiting clear signs of infection) in healthy adult bees[3]. Varroa mites act as mechanical **vectors** for CBPV, as viral particles have been detected within varroa mites and therefore can be transmitted to host adult bees[3]. Finally, CBPV has been detected in queen ovaries and/or eggs, indicating that **transovarial vertical** transmission is possible[3,4].

Figure 3. Mounds of dead bees in front of colony entrance.

Black Queen Cell Virus (BQCV)

DESCRIPTION

Black Queen Cell Virus primarily targets capped queen pupae during the spring and early summer[1,15]. The onset of BQCV disease causes the pupae to turn pale yellow and eventually black with a tough sac-like skin[6]. Progression of the disease leads to pupae death, followed by the walls of the queen cell becoming dark brown/black in colour[6]. Black Queen Cell Virus can remain present in a colony through infected asymptomatic worker bees and brood[6]. While this virus is mainly a disease of developing queen pupae and larvae, studies have shown the negative effects of BQCV infection in adult honey bees co-infected with *Nosema* spp.[1]. This positive relationship is likely due to adult bees with a previous *Nosema* spp. infection presenting a compromised midgut cell lining, allowing for easy viral infection and replication[6]. Adult honey bees co-infected with BQCV and *Nosema* spp. are found to have a shorter life span than bees infected with *Nosema* spp. alone[6]. Due to this link between BQCV and *Nosema* spp., an increase in viral levels is often observed in the spring and early summer, historically when *Nosema* spp. levels are high[1]. At the colony level, reduced adult bee population due to a shortened lifespan and queen pupae death can have negative effects on colony strength and productivity throughout the season[1].

KEY IDENTIFIERS

Figure 1. Black queen pupae with a tough sac-like skin.

TRANSMISSION

Black Queen Cell Virus is known to replicate in the larval stage of the honey bees, but it can also multiply rapidly in adult honey bees that have been co-infected with *Nosema* spp.[15]. As a result, **oral-oral** transmission generally happens when infected nurse bees, caring for the queen larvae, feed glandular secretions containing viral particles[1]. There has been no definitive evidence that Varroa mites can act as a transmission vector for BQCV. However, it has been observed that BQCV infections are frequently associated with high Varroa levels[4]. **Vertical** transmission can also occur, resulting in approximately a quarter of an infected queens' offspring having detectable levels of BQCV[6]. The potential for venereal transmission of BQCV was first identified when the virus was detected in the semen of infected drones, suggesting that **venereal** transmission can occur via seminal fluid[3].

Black Queen Cell Virus poses a higher threat to queen breeders[5]. Newly grafted queens can become infected with BQCV and the virus can easily spread among queen cells, potentially resulting in a poor queen hatch[8]. Additionally, Nosema levels tend to be high in the spring when breeders are extensively raising queens for the upcoming season[5]. This puts breeders at an even higher risk during this time of the year, due to the positive relationship between Nosema and BQCV-infection in adult honey bees.

Figure 2. Feces on frames as a result of *Nosema* spp. infection.

Sacbrood Virus (SBV)

DESCRIPTION

Sacbrood Virus is a common viral infection of honey bees, found nearly worldwide[16]. It is not considered to be a major threat to colony health and, historically, occurs mainly during colony expansion in the spring and early summer[6]. This virus attacks bees in both the larval and adult stages of life, with larvae that are approximately 2 days old being the most susceptible[6]. Upon infection, the larvae will turn greyish-yellow in colour and develop a tough skin-like outer layer, holding the larvae in a sac that can be removed whole from the cell[15]. Following infection, the larvae will continue to develop until the capped cell phase, when infected cells are then uncapped by worker bees[6]. Infected, greyish-yellow larvae will fail to pupate and will generally be found lying straight on the side of the cell with the head pointing upwards[15]. Adult honey bees can also be infected, but will usually appear asymptomatic. However, SBV-infected adult honey bees may show a decreased pollen consumption, shorter life span, and an early onset of foraging behaviour with a strong preference for nectar[6]. As a result, nutritional deficiencies within the colony can arise due to lack of pollen, reducing healthy worker life expectancy[16]. Highly infected colonies will have several uncapped brood cells scattered amongst the capped brood, causing the brood pattern to be 'spotty'[16].

KEY IDENTIFIERS

Figure 1. Yellow/grey SBV infected larvae laying straight on the side of the cell.

TRANSMISSION

Sacbrood Virus is transmitted within the colony via infected nurse and forager bees. Certain activities, such as cannibalizing eggs and young larvae during periods of protein deprival, removal of diseased brood containing virus particles, and cell/hive cleaning (i.e., removal of feces and dead bees from the bottom), are all opportunities of **oral-oral/oral-fecal** transmission[4,6]. Activities involving trophallaxis, the mouth-to-mouth sharing of food between colony members, can be another significant oral-oral route of SBV transmission[3]. **Venereal** transmission of SBV has also been observed, as SBV has been detected in the queens spermatheca following insemination, indicating that viral particles can be transferred via the drone's seminal fluid[3]. **Vertical transovarial** transmission is also a possible route for SBV, as viral particles have been recorded in queen ovaries and/or eggs, passing along the virus from queen to offspring[3].

Figure 2. Larvae pulled out in a tough sac-like package.

Figure 3. Spotty brood pattern.

Management

Unfortunately, there are currently no chemical treatments available to help combat viral infections. Therefore, a chemical-free management must be the primary method of virus control, applying the principles of Integrated Pest Management (IPM). There are five main components of IPM: cultural practices, monitoring, physical control, biological control, and chemical control[15]. Due to the lack of available/effective physical, biological, and chemical controls, there is a strong emphasis on utilizing **cultural practices** and **colony monitoring** as the main methods of management for viral infections.

One of the main inducers of viral infection is the onset of colony stressors. Some examples of these stressors are: Varroa mite infestation, co-infection with other pathogens, and lack of proper nutrition[4]. Therefore, the management and prevention of viral infections tends to revolve around reducing colony stressors in order to keep bees vigorous. Viral management is also comprised of both minimizing transmission and reducing viral levels[6].

Figure 1. Hive tool sterilization using heat.

CULTURAL PRACTICES

Cultural management practices are currently one of the most important aspects of IPM when working to control viral diseases. Viruses can remain present in healthy colonies at low levels, but when the colony is put under stress, these unapparent infections can turn into overt infections (i.e., exhibiting clear signs of infection)[4]. Due to the close linkage between Varroa mite infestation and viral infection, there is a strong emphasis on the use of effective mite management techniques to reduce vector-based transmission of Varroa-related viruses[17]. However, even after successful Varroa control, the threat of virus infection isn't completely diminished as viral levels do not drop to zero[18]. Viral infection has the potential to become cyclical as Varroa infestation repeatedly occurs[18]. Therefore, constant, and effective control and monitoring of Varroa mites are important to reduce vector-mediated transmission of viruses[18]. Additionally, selecting a genetic stock that has been bred for virus or Varroa resistance (e.g., hygienic behaviour, Varroa sensitive hygiene) will also reduce viral levels[15,19,20]. Colonies with these genetics have consistently shown lower Varroa levels, reduced viral titers, and increased colony survival[20].

Some additional cultural practices that can be implemented to manage viral infections include:

- Ensuring colonies have access to adequate nutritional resources[21]. Access to a diverse source of pollen and lipids is especially important in viral defense[22].
- Placing apiaries a minimum of 1 km apart and adjusting colony density based on surrounding floral density.
- Implementing good biosecurity practices between yards such as the frequent sterilization of beekeeping equipment and limiting exchange of bee products (i.e., honey and brood frames)[21].
- Separating sick colonies from healthy colonies[6].
- Placing bee pallets well apart and entrances in different orientations to reduce drifting[6].
- Supplementing colonies with additional nutritional resources (i.e., pollen patties/protein substitute and sugar syrup) during times of dearth to reduce nutritional stress[6].
- Introducing a young healthy queen to stop the vertical transmission of viruses[16].
- Rotating out 10-20% of equipment (i.e., frames and brood boxes) each year, specifically older equipment.

Figure 2. Colony health inspection.

MONITORING

Frequently monitoring colony health is a very important aspect of any disease management plan. However, monitoring for viruses can prove to be difficult as they can persist within a colony as unapparent infections, and lab diagnostics can become costly[1]. Overt virus infections tend to go hand-in-hand with other pathogens, such as Varroa mites and Nosema[6]. Therefore, monitoring for bee pathogens is extremely important for viral management, as colonies suffering from other diseases are likely to be suffering from viral diseases as well[4]. Visual inspections for viral diseases are difficult and not always accurate. An alternative is to take samples of suspected diseased colonies and send them to a diagnostic laboratory for confirmation[1]. Having exact data regarding viral presence and levels creates a record of colony health. Producers can then look at viral levels throughout their operation and use critical thresholds as a guide, and to determine the success of pathogen control practices[18]. This can be used in combination with other IPM practices, such as separating sick colonies from healthy colonies[18].

Other Bee Virus

Some other common viruses to be aware of in honey bee populations are: Acute Bee Paralysis Virus (ABPV), Kashmir Bee Virus (KBV), and Israeli Acute Paralysis Virus (IAPV). Varroa mites act as effective vectors for all three of these viruses, making the control of Varroa mites a crucial part of reducing viral diseases[1]. ABPV is known to be similar to CBPV, however the former is highly virulent as it rapidly kills adult honey bees within 3-5 days following infection[15]. KBV is also considered to be a highly virulent virus that persists in honey bee colonies as an unapparent infection and has undefined disease characteristics[1]. High KBV levels are not only associated with parasitic mites but also with other pathogens, such as *Nosema* spp. and European Foulbrood (EFB)[15]. IAPV has similar clinical signs to CBPV, including bee paralysis, shaking wings, and the presence of black hairless bees, leading to severe disease and possible colony death[1]. All three of these viruses are commonly present in colonies at low levels as asymptomatic infections , making visual detection difficult[1]. Similar to other honey bee viruses, colony stressors, such as pathogen pressure (i.e., varroa infestation) and nutritional deficiency, can prompt apparent infections[1]. Therefore, the best management plan to limit apparent viral infections is to implement management practices that work to reduce colony stressors and pathogen pressure.

References

1. Tantillo, G., Bottaro, M., Di Pinto, A., Martella, V., Di Pinto, P., & Terio, V. (2015) Virus infections of honeybees Apis mellifera. Italian Journal of Food Safety, 4(3). doi:10.4081/ijfs.2015.5364

2. Martin, S. J., & Brettell, L. E. (2019). Deformed wing virus in honeybees and other insects. Annual Reviews of Virology, 6(1), 49-69. https://doi.org/10.1146/annurev-virology-092818-015700

3. Yañez, O., Piot, N., Dalmon, A., De Miranda, J. R., Chantawannakul, P., Panziera, D., Amiri, E., Smagghe, G., Schroeder, D., & Chejanovsky, N. (2020). Bee viruses: Routes of infection in Hymenoptera. Frontiers in Microbiology, 11, 943. https://doi.org/10.3389/fmicb.2020.00943

4. Chen, Y., Evans, J., & Feldlaufer, M. (2006). Horizontal and vertical transmission of viruses in the honey bee, Apis mellifera. Journal of Invertebrate Pathology, 92(3), 152-159. https://doi.org/10.1016/j.jip.2006.03.010

5. Budge, G. E., Simcock, N. K., Holder, P. J., Shirley, M. D., Brown, M. A., Van Weymers, P. S., Evans, D. J., & Rushton, S. P. (2020). Chronic bee paralysis as a serious emerging threat to honey bees. Nature Communications, 11(1), 1-9. https://doi.org/10.1038/s41467-020-15919-0

6. de Miranda, J. R., Gauthier, L., Ribière, M., & Chen, Y. P. (2011). Honey bee viruses and their effect on bee and colony health. In D. Sammataro & J. A. Yoder (Eds.), Honey Bee Colony Health (pp. 71-102). CRC press. https://doi.org/10.1201/b11318

7. de Miranda, J. R., & Genersch, E. (2010). Deformed wing virus. Journal of Invertebrate Pathology, 103, S48-S61. https://doi.org/10.1016/j.jip.2009.06.012

8. Ryabov, E. V., Childers, A. K., Chen, Y., Madella, S., Nessa, A., vanEngelsdorp, D., & Evans, J. D. (2017). Recent spread of Varroa destructor virus-1, a honey bee pathogen, in the United States. Scientific Reports, 7(1), 1-10. https://doi.org/10.1038/s41598-017-17802-3

9. Zioni, N., Soroker, V., & Chejanovsky, N. (2011). Replication of Varroa destructor virus 1 (VDV-1) and a Varroa destructor virus 1–deformed wing virus recombinant (VDV-1–DWV) in the head of the honey bee. Virology, 417(1), 106-112. https://doi.org/10.1016/j.virol.2011.05.009

10. Tehel, A., Vu, Q., Bigot, D., Gogol-Döring, A., Koch, P., Jenkins, C., Doublet, V., Theodorou, P., & Paxton, R. (2019). The two prevalent genotypes of an emerging infectious disease, deformed wing virus, cause equally low pupal mortality and equally high wing deformities in host honey bees. Viruses, 11(2), 114. https://doi.org/10.3390/v11020114

11. Kevill, J. L., de Souza, F. S., Sharples, C., Oliver, R., Schroeder, D. C., & Martin, S. J. (2019). DWV-A lethal to honey bees (Apis mellifera): A colony level survey of DWV variants (A, B, and C) in England, Wales, and 32 states across the US. Viruses, 11(5), 426.doi:10.3390/v11050426

12. Traynor, K. S., Mondet, F., de Miranda, J. R., Techer, M., Kowallik, V., Oddie, M. A., Chantawannakul, P., & McAfee, A. (2020). Varroa destructor: A complex parasite, crippling honey bees worldwide. Trends in Parasitology, 36(7), 592-606. https://doi.org/10.1016/j.pt.2020.04.004

13. Dubois, E., Reis, C., Schurr, F., Cougoule, N., & Ribière-Chabert, M. (2018). Effect of pollen traps on the relapse of chronic bee paralysis virus in honeybee (Apis mellifera) colonies. Apidologie, 49(2), 235-242. https://doi.org/10.1007/s13592-017-0547-x

14. Ribière, M., Olivier, V., & Blanchard, P. (2010). Chronic bee paralysis: a disease and a virus like no other?. Journal of Invertebrate Pathology, 103, S120-S131. https://doi.org/10.1016/j.jip.2009.06.013

15. Pernal, S. F., & Clay, H. (Eds.). (2013). Honey Bee Diseases and Pests, 3rd edition. Canadian Association of Professional Apiculturists, Beaverlodge, AB, Canada.

16. Wei, R., Cao, L., Feng, Y., Chen, Y., Chen, G., & Zheng, H. (2022). Sacbrood virus: A growing threat to honeybees and wild pollinators. Viruses, 14(9), 1871. https://doi.org/10.3390/v14091871

17. Woodford, L., Christie, C. R., Campbell, E. M., Budge, G. E., Bowman, A. S., & Evans, D. J. (2022). Quantitative and qualitative changes in the deformed wing virus population in honey bees associated with the introduction or removal of Varroa destructor. Viruses, 14(8), 1597. https://doi.org/10.3390/v14081597

18. Amiri, E., Meixner, M., Nielsen, S. L., & Kryger, P. (2015). Four categories of viral infection describe the health status of honey bee colonies. PLoS One, 10(10), e0140272. https://doi.org/10.1371/journal.pone.0140272

19. de Guzman, L. I., Simone-Finstrom, M., Frake, A. M., & Tokarz, P. (2019). Comb irradiation has limited, interactive effects on colony performance or pathogens in bees, Varroa destructor and wax based on two honey bee stocks. Insects, 10(1), 15. https://doi.org/10.3390/insects10010015

20. O'Shea-Wheller, T. A., Rinkevich, F. D., Danka, R. G., Simone-Finstrom, M., Tokarz, P. G., & Healy, K. B. (2022). A derived honey bee stock confers resistance to Varroa destructor and associated viral transmission. Scientific Reports, 12(1), 1-19. https://doi.org/10.1038/s41598-022-08643-w

21. Nagaraja, N., & Rajagopal, D. (2019). Viral diseases. In, Honey Bees: Diseases, parasites, pests, predators, and their management (pp. 25-46). MJP publishers.

22. Dolezal, A. G., Carrillo-Tripp, J., Judd, T. M., Allen Miller, W., Bonning, B. C., & Toth, A. L. (2019). Interacting stressors matter: diet quality and virus infection in honeybee health. Royal Society Open Science, 6(2), 181803. https://doi.org/10.1098/rsos.181803


ALBERTA
BEEKEEPERS
COMMISSION


ALBERTA TECH TRANSFER
PROGRAM


Alberta


Canada


CANADIAN
AGRICULTURAL
PARTNERSHIP
Innovate. Grow. Prosper.


COSTCO
WHOLESALE


ProjectApis m