THE HISTORY AND BIOLOGY OF A HONEY BEE DISEASE

Chronic Bee Paralysis

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Paralysis has an ancient association with honey bees, with a diverse array of wonderfully descriptive names littering the literature.

These include 'black robbers' and 'little blacks' in Britain; 'Schwarzsucht' (black addiction), 'maladie noire' and 'mal nero' (black disease) in continental Europe; and 'hairless black syndrome' in the United States. It was in the 1960s that one of our personal heroes, Leslie Bailey, first identified the causative organism as a virus and named it chronic bee paralysis virus (CBPV, Figure 1).

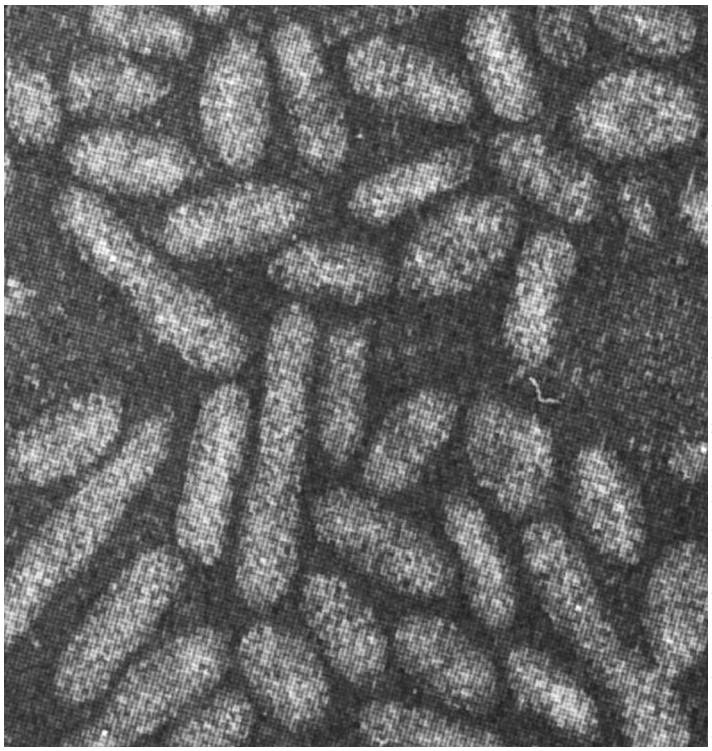

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Figure 1. Electron micrograph of chronic bee paralysis virus (CBPV). Particles vary in length between 30 nm and 60 nm, with a diameter of approximately 20 nm

Symptoms

In a valiant attempt to unite disparate malaises from across the literature, Bailey classified symptoms of individual bees into two 'types'.

Type 1 syndrome was described as the most commonly observed in Britain[1] and included an abnormal trembling motion with paralysis of the wings and bodies. Affected bees are unable to fly and sometimes crawl on the ground. They are often misplaced in the hive, appearing on top bars and lugs, leading some researchers to deem them to be 'stupid'. Abdomens can be bloated and the bees may suffer from dysentery and dislocated wings.

Type 2 syndrome united observations such as hairless bees, appearing almost black, and shiny, greasy bees suffering from nibbling attacks by healthy bees. This gives them the appearance of robber bees (Figure 2, page 9). After a few days, these individuals also suffer from paralysis and die.

Given our modern acceptance that CBPV can cause both types, there seems little benefit in continuing to distinguish between them – symptoms are not mutually exclusive to a honey bee colony and frequently occur in tandem. The symptoms of a severely affected colony can be startling, with dead bees on the floor, inches deep, and thousands piled up just outside the hive entrance, producing a rancid smell as the bees decompose. These colonies can sometimes recover but, more often, the colony will lose so many adult bees that it will enter terminal decline. A recent example of a severe outbreak of chronic bee paralysis led to the loss of 150 out of 400 colonies in a single bee-farm operation.

Diagnosis and Cases of Mistaken Identity

Individually, many of the symptoms can be confused with other afflictions. Nosema can cause dysentery; acarine may cause bees to become flightless; deformed wing virus may cause dislocated wings; and there are at least four other paralysis viruses of the honey bee. However, taken together, the symptoms are most frequently confused with pesticide poisoning.

Andy Wattam, the national bee inspector, kindly highlighted two important differences between pesticide poisonings and CBPV infections. First, adult bees afflicted with chronic bee paralysis virus never do the 'dying fly' act – where individuals spin in circles on their backs seemingly unable to fly. This symptom is found exclusively with pesticide poisonings. Second, chronic bee paralysis is associated with piles of dead bees directly outside the front of the hives, whereas pesticide poisonings sometimes show a carpet of dead bees more evenly spread across the apiary, perhaps caused by bees not quite making it back to the colony.

It is worth noting that there is help at hand if you suspect a pesticide poisoning. Simply contact your local National Bee Unit (NBU) inspector and, if they also suspect pesticides, your samples could be entered into a national monitoring scheme known as the Wildlife Incident Investigation Scheme (for more details see www.nationalbeeunit.com/index.cfm?sectionid=33).

Diagnosis in the field requires several of the above mentioned symptoms to be observed together. However, given the opportunity for misidentification, the only definitive diagnosis

Figure 2. Image of a black hairless 'robber' bee with nibbled wings (left) and a greasy looking bee with dislocated wings (right). Both are classic symptoms of chronic bee paralysis

comes from confirming the presence of the virus using sensitive molecular tests.

Prevalence and Transmission

Chronic bee paralysis is found on every continent where honey bees are kept. A large-scale survey completed by the NBU in 2011 suggested that CBPV was not commonly detected when random apiaries were tested – only seven in 1000 apiaries tested positive. Interestingly, the prevalence was found to be far higher when apiaries showing poor health were targeted – in this case 160 in 1000 apiaries tested positive[2].

All stages of the honey bee life cycle may be infected with CBPV but it is most concentrated in adult bees.

When an adult honey bee is infected, its nervous system and brain are hijacked by the virus for the purposes of viral replication, causing the characteristic trembling and paralysis. A single individual showing paralysis may harbour up to 10 trillion virus particles. The infected bee secretes the viruses from its epidermis and in its faeces, turning it into a mobilised virus factory. The hive mates of the infected bees can be infected by simply brushing up against the infected bees or through contact with faeces from contaminated individuals[1].

CBPV can spread rampantly in crowded conditions and so can be associated with large populous colonies and exacerbated by prolonged periods of confinement due to bad weather[3]. CBPV is not transmitted by varroa.

As is often the case with honey bee viruses, CBPV is able to infect and replicate in other hymenopteran hosts, in this case ants[4]. For a wonderfully complete review of virus biology, please see the article presented by Magali Ribière[1].

Control

Historically, chronic bee paralysis has been a very difficult disease to study because onset of symptoms can be sudden and outbreak location unpredictable. As such, there are no scientific papers that provide robust evidence to demonstrate successful treatments. Queen replacement has been suggested, but given that all races of bee appear to be susceptible, any success by this method could be attributed to a drop in adult bee population due to the brood break associated with queen replacement.

A recent promising management method appears to be a modification of the shook swarm described by Chris Neel[5].

The method is as follows:

1 the queen is caged
2 the colony is moved at least 50 m and the bees emptied into the air (rather than onto the ground)
3 the colony is replaced in its original location and only the healthy bees return.

This method probably breaks the bee-to-bee transmission route and could need to be repeated again six days later to remove previous symptomless carriers of the virus. It is early days, but some (if not all) have found this method to be helpful.

Summary

To summarise, chronic bee paralysis can be a very severe disease that is seemingly increasing in prevalence in recent years. The reasons for this increase are currently unknown, but they could be driven by more changeable weather causing confinement in populous colonies.

There appears to be a clear need to extend our knowledge of chronic bee paralysis and provide more useful tools for beekeepers to manage this damaging viral disease. ⌘

References

1 Ribière, M, Olivier, V and Blanchard, P (2010). Chronic bee paralysis: a disease and a virus like no other? *Journal of Invertebrate Pathology,* **103**, Suppl 1, S120–131.

2 Budge, GE, et al (2015). Pathogens as Predictors of Honey Bee Colony Strength in England and Wales. *Plos One*, **10**(7), e0133228.

3 Bailey, L, et al (1983). Honey bee paralysis: Its natural spread and diminished incidence in England and Wales. *Journal of Apicultural Research*, **22**(3), 191–195.

4 Celle, O, et al (2008). Detection of Chronic bee paralysis virus (CBPV) genome and its replicative RNA form in various hosts and possible ways of spread. *Virus Research*, **133**, 280–284.

5 Neel, C (2016). Chronic bee paralysis virus. *Bee Farmer*, **2**(2), 11.

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