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PERSPECTIVE

Acute paralysis viruses of the honey bee

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The alarming decline of honey bee (*Apis mellifera*) colonies in the last decade drove the attention and research to several pathogens of the honey bee including viruses. Viruses challenge the development of healthy and robust colonies since they manage to prevail in an asymptomatic mode and reemerge in acute infections following external stresses, as well as they are able to infect new healthy colonies (de Miranda J R, et al., 2010a; de Miranda J R, et al., 2010b; Di Prisco G, et al., 2013; Nazzi F, et al., 2012; Yang X L, et al., 2005).

The most common viruses involved in acute paralysis of honey bees belong to the Acute bee paralysis virus- Kashmir bee virus- Israeli acute paralysis virus complex (ABPV-KBV-IAPV complex) (de Miranda J R, et al., 2010). ABPV, and IAPV were mostly associated with colony losses worldwide (Berthoud H, et al., 2010; Cox-Foster D L, et al., 2007; de Miranda J R, et al., 2010; de Miranda J R, et al., 2010; Genersch E, et al., 2010). While ABPV was correlated with collapse of overwintering colonies in Europe (Berthoud H, et al., 2010), initial metagenomic studies linked the incidence of IAPV to the syndrome of Colony collapse disorder (CCD), a particular form of collapse of honey bee colonies that was widely observed in California (Cox-Foster D L, et al., 2007). Further studies indicated that stressed CCD-colonies may become susceptible to virus infections (vanEngelsdorp D, et al., 2009).

Many viral surveys in colonies around the world detected a substantial prevalence of IAPV in covert asymptomatic infections (Al-Abbadi A A, et al., 2010; Blanchard P, et al., 2008; Chen Y P, et al., 2014; de Miranda J R, et al., 2010; Formato G, et al., 2011; Granberg F, et al., 2013; Ozkirim A, et al., 2013; Palacios G, et al., 2008; Pohorecka K, et al., 2011; Reynaldi F J, et al., 2011; Vicente-Rubiano M, et al., 2013).

ABPV, IAPV and KBV are dicistroviruses with a monopartite positive-sense stranded RNA genome of 9491–9613 bp. The icosahedral viral particle is about 30 nm diameter (de Miranda J R, et al., 2010). These viruses provoke acute paralysis upon their injection into the hemolymph of adult bees (Bailey L, 1967; Dall D J, 1987; Maori E, et al., 2007). Since IAPV is the most recently discovered virus from this group hereby we will focus on some of the advances in IAPV-research.

Honey bees that were infected experimentally with IAPV exhibited disorientation, shivering wings and crawling that developed to paralysis and subsequent death within or outside the hive (Boncristiani H F, et al., 2013; Maori E, et al., 2007). IAPV-injected pupae die between 48–96 h post infection (Hou C S, et al., 2014; Maori E, et al., 2007). Replicating IAPV was detected in hemolymph, brain, fat body, salivary gland, hypopharyngeal gland, gut, nerve trachea and muscle of virus-infected honey bees, with more prominent abundance in the gut, nerve and hypopharingeal glands (Chen Y P, et al., 2014).

It seems that the oral infectivity of the above dicistroviruses is low, what may contribute to the establishment of asymptomatic infections and relatively large doses are required to provoke infection (Bailey L, 1967; Dall D J, 1987; de Miranda J R, et al., 2010; Maori E, et al., 2009). Recently emerged bees are susceptible to oral infection with IAPV and depending upon the administered dose they show symptoms of increased acute pa-ralysis, trembling, they are unable to fly, and subsequently die between 2 to 7 days post infection (Chejanovsky N, et al., 2014) (Maori E, et al., 2009). The above data indicate that IAPV becomes highly lethal once it reaches the honey bee hemolymph.

IAPV strains exhibit a high degree of diversity (Chen Y P, et al., 2014; Cornman R S, et al., 2012; Palacios G, et al., 2008) but very little is known about molecular determinants of virulence and infectivity (Chen Y P, et al., 2014), mainly due to the lack of appropriate tissue culture systems that could support molecular manipulation of viral clones to

identify those determinants (de Miranda J R, et al., 2010).

It was shown experimentally that *Varroa destructor*, an ectoparasite mite that spread from the oriental bee *Apis cerana* through most colonies of *A. mellifera* in the world (Rosenkranz P, et al., 2010) is able to transmit IAPV (Di Prisco G, et al., 2011).

We studied CCD-colonies that showed high titers of IAPV and found that it was the most prevalent and dominant pathogen in the colonies throughout almost one year of follow up (Hou C S, et al., 2014). A drop in IAPV titers from April to September was observed in these colonies and the number of genomic copies of the virus increased from September through December (Hou C S, et al., 2014). IAPV from infected bees was highly infectious to healthy bees. The continuous infection with IAPV impacted colony health and the size of the adult and brood populations of all the colonies was deeply affected. The structure of the adult vs. brood population of the IAPV-infected colonies was very different from that of the control colonies. Our study indicated that once acquired and induced to replicate, IAPV acts as an infectious factor that affects deeply the health of the colonies and may determine their survival (Hou C S, et al., 2014). A recent epidemiological study supported these conclusions (Chen Y P, et al., 2014).

CCD-colonies were able to mount an initial RNA interference (RNAi) response against honey bee viruses. The colonies were able to recognize the invading virus and dice its replicating genome, suggesting that the virus-specific component of the siRNA pathway mediated by Ago-2 was functional (Chejanovsky N, et al., 2014). Further experimentation and data analysis is required to prove that the whole RNAi pathway in CCD-colonies is fully functional (Chejanovsky N, et al., 2014).

RNAi-mediated silencing of IAPV was shown in laboratory and in field experiments (Hunter W, et al., 2010; Maori E, et al., 2009). Interestingly, silencing of a putative suppressor of RNAi present in the IAPV genome diminished the ability of the virus to amplify its negative-sense RNA strand required for effective replication (Chen Y P, et al., 2014).

Maori and co-workers found partial IAPV genomic sequences in the genome of asymptomatic hosts and suggested that their presence may confer protection to the host from lethal virus infections (Maori E, et al., 2007).

Finally, a recent study found that IAPV up-regulated host genes that control signal transduction and immune responses together with down regulation of genes involved in generating metabolic energy (Chen Y P, et al., 2014).

In conclusion, IAPV replicates in honey bees from weak and CCD-colonies and it persists overtime in honey bee colonies as an infectious agent. The research data indicates that IAPV can be transmitted vertically from the queen through transovarial transmission and from drones to the queen via insemination, and horizontally from workers to the queen, other bees and larvae via food sources and glandular secretions (Chen Y P, et al., 2014; Singh R, et al., 2010).

Biological or chemical stress-factors able to affect the health of the colony could induce awakening of dormant asymptomatic infections with lethal effects to the colony. More detailed studies are required to elucidate precisely the molecular mechanisms involved in IAPV-activation.

Active replication of IAPV can deeply affect the structure of population of the colony and its wealth and lead to significant losses, thus development of treatments to diminish IAPV titers are important to protect colony health.

FOOTNOTES

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