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Abstract

Honey Bee Viruses, Colony Health, and Antiviral Defense †

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Abstract: Honey bee colony losses are influenced by multiple abiotic and biotic factors, including viruses. To investigate the effects of RNA viruses on honey bees, we infected bees with a model virus (Sindbis-GFP) in the presence or absence of double-stranded RNA (dsRNA). In honey bees, dsRNA is the substrate for sequence-specific RNA interference (RNAi)-mediated antiviral defense and is a trigger of sequence-independent\antiviral responses. Transcriptome sequencing identified more than 200 differentially expressed genes, including genes in the RNAi, Toll, Imd, JAK-STAT, and heat shock response pathways, and many uncharacterized genes. To confirm the virus limiting role of two genes (i.e., *dicer* and *mf116383*) in honey bees, we utilized RNAi to reduce their expression in vivo and determined that the virus abundance increased. To evaluate the role of the heat shock stress response in antiviral defense, bees were heat stressed post-virus infection and the virus abundance and gene expression were assessed. Heat-stressed bees had reduced virus levels and a greater expression of several heat shock protein encoding genes (*hsps*) compared to the controls. To determine if these genes are universally associated with antiviral defense, bees were infected with another model virus, Flock House virus (FHV), or deformed wing virus and the gene expression was assessed. The expression of *dicer* was greater in bees infected with either FHV or Sindbis-GFP compared to the mock-infected bees, but not in the deformed wing virus-infected bees. To further investigate honey bee antiviral defense mechanisms and elucidate the function of key genes (*dicer*, *ago-2*, *mf116383*, and *hsps*) at the cellular level, primary honey bee larval hemocytes were transfected with dsRNA or infected with the Lake Sinai virus 2 (LSV2). These studies indicate that *mf116383* and *hsps* mediate dsRNA detection and that MF116383 is involved in limiting LSV2 infection. Together, these results further our understanding of honey bee antiviral defense, particularly dsRNA-mediated antiviral responses, at both the individual bee and cellular levels.

Keywords: insect antiviral defense; dsRNA-mediated antiviral defense; honey bee; RNA viruses; RNAi; heat shock response