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ame-miR-34 modulates the larval body weight and immune response of Apis mellifera workers to Ascosphara apis invasion

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Ascosphaera apis exclusively infects bee larvae and causes chalkbrood disease. However, the function and mechanism of miRNAs in the bee larval response to A. apis infection is poorly understood. Here, ame-miR-34, a previously predicted miRNA involved in the larval response to A. apis invasion, was subjected to molecular validation, and overexpression and knockdown were then conducted to explore the regulatory functions of ame-miR-34 in larval body weight and immune response. Stem-loop RT-PCR and Sanger sequencing confirmed the authenticity of amemiR-34 in the larval gut. RT-qPCR results demonstrated that compared with that in the uninfected larval guts, the expression level of ame-miR-34 was significantly downregulated in the guts of A. apis-infected 4-, 5-, and 6-day-old larvae. In comparison with the corresponding negative control (NC) groups, the expression level of ame-miR-34 in the larval guts in the mimic-miR-34 group was significantly upregulated, while that in the inhibitor-miR-34 group was significantly downregulated. Additionally, the body weights of 5- and 6-day-old larvae were significantly increased compared with those in the mimic-NC group; the weights of 5-day-old larvae in the inhibitor-miR-34 group were significantly decreased in comparison with those in the inhibitor-NC group, while the weights of 4- and 6-day-old larvae in the inhibitor-miR-34 group were significantly increased. Furthermore, the expression levels of both hsp and abct in the guts of A. apis-infected 4-, 5-, and 6-day-old larvae were significantly activated after ame-miR-34 overexpression. In contrast, after ame-miR-34 knockdown, the expression levels of the aforementioned two key genes in the A. apis-infected 4-, 5-, and 6-dayold larval guts were significantly downregulated. Together, the results demonstrated that effective overexpression and knockdown of ame-miR-34 in both noninfected and A. apis-infected A. mellifera larval guts could be achieved by the feeding method, and ame-miR-34 exerted a regulatory function in the host body weight and immune response to A. apis invasion through positive regulation of the expression of hsp and abct. Our findings provide a valuable reference for the functional investigation of bee larval miRNAs and a promising molecular target for the treatment of chalkbrood disease.