THE EFFECTS OF A VIRAL SILENCING SUPPRESSOR PROTEIN ON PLANT-APHID INTERACTIONS

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Infection with cucumber mosaic virus strain Fny (Fny-CMV) induced resistance to aphids (*Myzus persicae*) in *Arabidopsis thaliana* ecotype Col-0. Electrical penetration graph analysis of aphid feeding behavior and microbalance measurements showed that aphids fed less and grew poorly on infected plants, leading to the production of smaller colonies. Experiments with the Fny-CMV∆2b deletion mutant and *Fny2b*-transgenic plants showed that the CMV 2b RNA silencing suppressor protein induces aphid resistance. Consistent with this, glucosinolate accumulation increased in plants infected with Fny-CMV but not Fny-CMV∆2b. Fny-CMV infection caused dramatic changes in the transcriptome typically associated with salicylic acid (SA)-dependent defense responses (most likely due to induction of SA accumulation by the virus) while most jasmonic acid-regulated genes were unaffected. Nevertheless, experiments with defensive signaling mutants showed that neither hormone is critical for aphid resistance induction by Fny-CMV. In Arabidopsis Col-0, the Fny2b protein, but not the LS-CMV strain 2b protein, strongly inhibits microRNA-mediated turnover of host mRNA. Aphid resistance was not triggered in LS-CMV-infected or *LS2b*-transgenic plants, which suggests an important role for the microRNA pathway in regulating aphid resistance. The results in Arabidopsis contrasted with those from tobacco (*Nicotiana tabacum*) where Fny-CMV induced susceptibility to aphids and Fny-CMV∆2b induced a strong nicotine-independent resistance that caused increased mortality among these insects. The potential epidemiological implications of host-specific and CMV strain-specific effects on plant-aphid interactions will be discussed.