

 MICROBIAL ECOLOGY

A bacterial decoy skews plant defences

“ bacterial symbionts of CPB larvae specifically suppress anti-herbivore defences. ”

Herbivorous insects possess a diverse range of microbial symbionts that can modulate plant–insect interactions; however, the potential role of symbionts in manipulating plant defences is poorly understood. A study now shows that the larvae of the Colorado potato beetle (CPB; *Leptinotarsa decemlineata*) secrete bacterial symbionts to suppress an anti-herbivore defence pathway in tomato plants, thus promoting larval growth.

Plants have evolved sophisticated defence strategies against different biotic attackers. The jasmonic acid-regulated pathway is crucial to defend against most herbivorous insects and microbial pathogens that feed on dead plant material, whereas the salicylic acid-regulated pathway responds to microbial pathogens that thrive on living hosts. Crosstalk between these pathways

has previously been shown to be important for fine-tuning defence responses. As a counter-strategy, several herbivores produce effectors to disarm such defences, which often involves negative crosstalk between these two signalling pathways.

CPB larvae deposit oral secretions on tomato plant leaves during feeding. Chung *et al.* observed that, unlike those of antibiotic-treated larvae, the oral secretions deposited on plant leaves by untreated larvae contained large amounts of bacteria, which correlated with enhanced larval growth. In addition, plants exposed to untreated larvae showed lower polyphenol oxidase (PPO; an indicator of jasmonic acid signalling) activity than those exposed to antibiotic-treated larvae, indicating that CPB bacterial symbionts reduce jasmonic acid-mediated signalling.

To investigate the effects of symbionts in more detail, the authors measured the expression of genes encoding components specific for each pathway. They found that untreated larvae decreased the expression of jasmonic acid-responsive genes, whereas expression of a salicylic acid-responsive gene was increased. Furthermore, in plants deficient in salicylic acid signalling, the suppression of jasmonic acid-mediated defences was blocked, indicating that negative crosstalk between these two signalling pathways is required for symbiont-induced suppression of anti-herbivore defences.

To identify the symbionts involved, bacteria were isolated from the oral secretions of untreated larvae,

cultured *in vitro* and subsequently applied to mechanically wounded plant leaves. From the 22 bacterial isolates tested, three isolates significantly reduced PPO activity, and 16S rRNA sequencing revealed that they belonged to the genera *Stenotrophomonas*, *Pseudomonas* and *Enterobacter*. Re-inoculation of antibiotic-treated larvae with each individual bacterium restored jasmonic acid-mediated signalling, which confirmed that these symbionts are capable of manipulating plant defences. Finally, flagellin purified from the *Pseudomonas* sp. isolate was shown to reduce PPO activity in wounded plant leaves, suggesting that this is a specific effector protein involved in defence suppression. Previous work in *Arabidopsis thaliana* has shown that flagellin elicits salicylic acid signalling, so this effector is probably involved in mediating negative crosstalk, thereby down-regulating the herbivore-specific jasmonic acid defence response.

Together, these data show that bacterial symbionts of CPB larvae specifically suppress anti-herbivore defences. The clever strategy deployed by CPB larvae relies on using the bacterial symbionts as a decoy, which — owing to the distinct defence pathway that they elicit in plants — results in the insect being perceived as a microbial rather than a herbivorous threat.

Christina Tobin Kährström

ORIGINAL RESEARCH PAPER Chung, S. H. *et al.* Herbivore exploits orally secreted bacteria to suppress plant defenses. *Proc. Natl Acad. Sci. USA* <http://dx.doi.org/10.1073/pnas.1308867110> (2013)



Bacteria deposited on a tomato plant leaf by antibiotic-untreated CPB larvae. Image courtesy of G. W. Felton, Pennsylvania State University, Pennsylvania, USA.