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research highlights

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No sugar for you

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With their immense reserves of carbohydrates, plants are very tempting targets for many microorganisms. Bacteria, fungi and oomycetes have all evolved various strategies to proliferate by tapping into the nutritional reserves of plants. Previous research showed that bacterial pathogens increase the flow of sucrose towards the apoplast, where they grow, by manipulating plant plasma membrane sugar transporters. However, according to new research by Yoshitaka Takano and colleagues from Japan, the plants fight back by reabsorbing the sugars inside the cells, in effect starving the pathogens.

The authors discover that an *Arabidopsis* line mutated in two hexose transporters of the sugar transport protein (STP) family is more susceptible to the bacterial pathogen *Pseudomonas syringae*. They reconstruct the molecular pathway step by step to explain this result. After perception of the microbial signature peptide flg22 (a conserved domain of the flagellin protein) by its receptor FLS2 (FLAGELLIN-SENSING 2), two events occur: co-receptor BAK1 (BRASSINOSTEROID INSENSITIVE 1-ASSOCIATED RECEPTOR KINASE 1) phosphorylates STP13, increasing the activity of the sugar transporter; and STP13 levels become higher in the leaf. So plants react to incoming bacteria both post-translationally and transcriptionally, increasing the reabsorption of hexoses by the cell. This results in less sugar in the apoplast, which restricts proliferation and virulence of the invading bacteria.

This study highlights once again how sugar distribution is an important aspect of plant–pathogen warfare. The high prevalence and conservation of sugar transporters in other plants points towards possible novel resistance strategies in crops.

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