Studies on infection mechanisms of oomycete plant pathogens

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Abstract

Plants and pathogens have co-evolved in a defensive–offensive battle for survival. Plants induce basal resistance by sensing broadly conserved pathogen molecules. On the other hand, pathogens have produced effectors that act as a virulence factor and have acquired the ability to infect plants by suppressing their basal resistance. In turn, plants have evolved intracellular receptors called resistance (*R*) genes, which recognize the presence of pathogen effectors directly or indirectly to launch a strong counterattack.

- Recognition avoidance mechanisms in downy mildew -

Hyaloperonospora arabidopsidis (*Hpa*) is an obligate biotrophic oomycete that causes downy mildew in Arabidopsis. Focusing on the Arabidopsis–*Hpa* interaction, we established a system to quantify changes in gene expression in both Arabidopsis and *Hpa* simultaneously during infection. On the basis of transcriptome data in Arabidopsis Col-0 inoculated with the avirulent *Hpa* isolate Emoy2 (recognized by *RPP4*) or the virulent isolate Waco9, we found that *ATR1* (recognized by *RPP1*) is not expressed in *Hpa* Waco9. After resequencing the Waco9 genome, we found that the *ATR1* region is deleted, leading to evasion of recognition by its cognate *R* gene, *RPP1*. Comparative genomics and transcriptomics of different isolates of *Hpa* uncovered the *Hpa* effector *AvrRPP4* recognized by the *R* gene *RPP4* of Arabidopsis. By investigating *Hpa* isolates that evade recognition, we also revealed two mechanisms for evading recognition: suppressing effector gene expression or altering its localization in host cells.

- Mechanisms of plant immune suppression by downy mildew effectors -

By histochemical analysis, we found that *Hpa* suppresses salicylic acid (SA)-inducible *PR1* expression, specifically in host cells with a haustorium into which host-translocated effectors are delivered, but not in adjacent host cells without a haustorium. We also revealed that *Hpa* effectors (HaRxL44, HaRxL62, HaRxL106) were involved in the suppression and identified host targets for HaRxL44 and HaRxL106. Jasmonic acid (JA) is known to act antagonistically with SA in the signaling pathways to regulate the expression of defense genes and resistance to pathogens. HaRxL44 interacts with Med19a, which is a subunit of the mediator complex involved in transcriptional regulation of the host, and promotes the degradation of Med19a, thereby promoting the JA pathway. The activation of JA pathway leads to suppression of SA pathway including *PR1* induction. HaRxL106 suppresses the SA pathway by targeting the transcriptional regulator RADICAL-INDUCED CELL DEATH1 (RCD1) in the host nucleus. These studies revealed that Med19a regulates JA responses and that RCD1 is involved in the SA responses, indicating that the analysis of pathogen effectors can reveal immune mechanisms of plants.

- Conclusion -

Through research on the Arabidopsis–downy mildew interaction, we have identified host target factors for downy mildew effectors and have clarified the mechanisms of recognition avoidance by host *R* genes. We expect that disease control methods based on these results will be developed and applied in the field.