IN BRIEF

Border Patrol on the Extrahaustorial Membrane: Arabidopsis Resistance Protein RPW8.2 Activates Targeted, Postpenetration Defenses

The spores of powdery mildew fungal pathogens germinate on the leaf surface, attack plants by digesting through the cell wall, and invade the cell by producing a feeding structure, the haustorium (reviewed in Hückelhoven and Panstruga, 2011). The fungus forces changes in the plant cell’s metabolism, cytoskeleton, secretory pathways, and endomembrane system; for example, the fungus reroutes host transport to direct nutrients to the haustorium. Also, the plant cell membrane adjacent to the haustorium, termed the extrahaustorial membrane (EHM), excludes many plasma membrane proteins, but likely contains nutrient transporters that feed the pathogen. The EHM also provides a location for targeted host defenses. For example, the Arabidopsis thaliana resistance protein RPW8.2 is specifically targeted to the EHM, where it can induce postpenetration resistance by activating local plant defenses, including production of reactive oxygen and encasement of the haustorium (Wang et al., 2009). By this strategy, the invaded cell can resist fungal infection, even after the pathogen has broken through other defense barriers such as the cell wall.

To examine the EHM targeting and defense activation by RPW8.2, Wang et al. (pages 4242–4261) constructed more than 100 mutant versions of RPW8.2, guided by naturally occurring rpw8 alleles. They tested whether each version localized to the EHM and whether it could activate defenses. They tested three critical amino acids required for powdery mildew resistance and found that phosphorylation of RPW8.2 may limit activation of cell death. Interactions among mutations further indicated that some natural mutations increased RPW8.2 activity, but others limited its activity, possibly to avoid triggering spontaneous cell death.

The authors also defined a 60-amino acid sequence from RPW8.2, including the putative N-terminal transmembrane domain and two Arg/Lys-rich motifs, as necessary and sufficient for EHM targeting. Most of the RPW8.2 mutants localized to the EHM, but others localized to the nucleus or to a peristromule membrane compartment connecting plastids. Dramatic changes in localization: Does this plasticity indicate that the wild-type RPW8.2 protein may localize to other places? Or do these changes in localization reflect similarities in membrane characteristics or common targeting pathways? Also, the EHM provides a special interface for transport of nutrients into the haustorium: Does the plant exploit the pathogen’s transport pathways to target RPW8.2 to the EHM, thereby activating postpenetration defenses by a last-ditch strategy? Exploration of these possibilities will push the borders of our understanding of plant-pathogen interactions, much as RPW8.2 defends the borders of plant cells against fungal invaders.

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