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DISEASE RESISTANCE AT THE CELL WALL

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Host cell entry represents an Achilles heel during pathogenesis of most biotrophic fungi and marks a lifestyle transition from extracellular to invasive growth. Plant cells respond to fungal invasion attempts by a polar re-organization of the cytoskeleton, directed vesicle transport, and localized cell wall remodelling. Novel molecular genetic data revealed Janus-faced functions of these pathogen-triggered cell polarization events, that is, for fungal pathogenesis and in disease resistance responses at the cell periphery. I will discuss the role of a conserved SNARE protein-dependent and vesicle-associated resistance pathway at the plasma membrane. It appears that a widespread class of biotrophic fungi, powdery mildews, have evolved specific means to sabotage the resistance response by blocking effective SNARE complex assembly at the plasma membrane.

The SNARE protein-dependent resistance mechanism at the cell periphery represents a first line of defence against fungal entry. It is an important component of non-host resistance that limits the host range of pathogen species. Arabidopsis *PEN* loci define components of this common type of resistance, i.e. *pen* mutants fail to terminate entry of inappropriate powdery mildew species at the cell periphery. However, non-host resistance to inappropriate powdery mildews is only partially compromised in the *pen* mutants. I will present evidence that additional mutations in other Arabidopsis genes are required to enable reproduction (sporulation) of the tested powdery mildew species. These data suggest the existence of at least two genetically and spatially separable resistance mechanisms operating in non-host resistance. This might explain why in nature non-host resistance is more durable in comparison to race-specific resistance.