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Non-host resistance describes the immunity of an entire plant species against non-adapted pathogens. Although this form of resistance is widespread in nature, it is still poorly understood at the molecular level. We have begun to dissect the molecular basis of non-host resistance by searching for *Arabidopsis* mutants that exhibit altered infection phenotypes to two ascomycete powdery mildew fungi that in nature colonize grass or pea species. The non-adapted fungi almost always fail to enter leaf epidermal cells of wild-type *Arabidopsis*. The underlying resistance mechanisms involve specific isoforms of a plasma membrane resident syntaxin (PEN1) and ABC transporter (PEN3) as well as a peroxisomal glycosyl hydrolase (PEN2). PEN genes act as components of at least two separate and inducible resistance mechanisms. Each of the three PEN proteins become recruited to fungal entry sites. Evidence is provided suggesting functions of PEN proteins in secretory processes at the cell periphery that terminate fungal pathogenesis. Interestingly, adapted host powdery mildews of *Arabidopsis* appear to have evolved mechanisms to subvert PEN-dependent immune responses against fungal entry. While the basic machinery of the PEN secretory machinery evolved before the divergence of dicots and monocots, some components appear to be recent innovations of Arabidopsis/dicotyledonous plants.