IN BRIEF

Linking Multivesicular Bodies to Resistance against Fungal Invasion

Powdery mildew fungi infect a wide variety of monocots and dicots, forming feeding structures called haustoria in epidermal cells of their hosts (reviewed in Eichmann and Hückelhoven, 2008). In response to these fungi, plants reinforce their cell walls with callose-rich appositions, called papillae, at the site of attack. Proteins involved in two pathways of penetration resistance have been identified in Arabidopsis thaliana: PENETRATION2 (PEN2) and PEN3 appear to function in the same pathway for secretion of secondary metabolites, probably antifungal chemicals. The other pathway involves PEN1, a syntaxin most likely involved in vesicle transport (reviewed in Robatzek, 2007).

New work from Böhlenius et al. (pages 3831–3844) explores the role of host cell vesicle transport in barley (Hordeum vulgare) resistance against the powdery mildew fungus Blumeria graminis f. sp. hordei (Bgh). The authors searched for ADP-ribosylation factor (ARF) GTPases, which are involved in vesicle budding (see Hwang and Robinson, 2009), and identified six that were expressed in the epidermis. Interestingly, two of these genes (ARFA1b and ARFA1c) encode identical proteins (together referred to as ARFA1b/1c), and when these two genes were silenced using single cell transient-induced gene silencing in barley leaves, resistance against Bgh penetration was compromised. Böhlenius and coworkers confirmed ARFA1b/1c’s involvement in penetration resistance by bombarding barley leaves with constructs expressing dominant-negative mutant versions of the protein, which also allowed increased Bgh penetration.

The authors tested whether ARF1b/1c’s role in penetration resistance was linked to that of REQUIRED FOR MLO-SPECIFIED RESISTANCE2 (ROR2; the barely ortholog of PEN1), since absence of either of these two components results in increased Bgh haustoria formation and both are involved in vesicle trafficking. When mutant ARF1b/1c protein was expressed in cells of ror2 mutant leaves, there was no additive effect on penetration frequency, suggesting that the two proteins act in the same pathway. ROR2 accumulates in papillae, and the authors tested whether ARFA1b/1c is involved in getting it there. Indeed, they found that ROR2 was mislocalized during Bgh attack in the presence of mutant ARFA1b/1c. Furthermore, they found that whereas callose deposition in papillae was delayed in ror2 mutants, callose was nearly absent from papillae in cells expressing the mutant version of ARFA1b/1c.

Böhlenius et al. went on to show that ARFA1b/1c is located in mobile organelles that accumulate at Bgh penetration sites before callose deposition begins. They found that the ARFA1b/1c organelles were endocytic in origin and likely to be multivesicular bodies (see figure). Together, the findings in this report describe ARFA1b/1c as a new component of the ROR2 pathway for penetration resistance and suggest the intriguing possibility that multivesicular bodies accumulate near penetration sites to deliver ROR2 and callose to papillae.

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REFERENCES


Barley ARFA1b/1c is present in multivesicular bodies. Onion epidermal cells coexpressing ARFA1b/1c fused to green fluorescent protein fusion (left) and red fluorescent protein fused to ARA7 (a multivesicular body marker; middle) are shown. The right panel shows the merged image. Bars = 10 μM. (Image from Böhlenius et al. [2010].)