The Plant Cell, Vol. 21: 2543, September 2009, www.plantcell.org

IN BRIEF

The *Arabidopsis* RPW8 Resistance Protein Is Recruited to the Extrahaustorial Membrane of Biotrophic Powdery Mildew Fungi

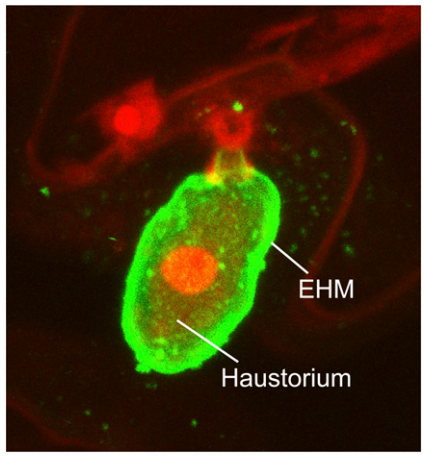
Biotrophic fungal and oomycete pathogens, which include the rusts and powdery mildews, are important pathogens of many crops, causing significant annual yield losses around the globe. These pathogens depend on living plant tissue for their growth and reproduction. To achieve this, they penetrate the cell wall and produce specialized feeding structures called haustoria via invagination of the host cell plasma membrane (PM). The haustorium is encased in an extrahaustorial membrane (EHM), which is the focal point for host–pathogen interactions and trafficking of nutrients from the host to the pathogen. However, the origin and constitution of the EHM remain enigmatic.

Wang et al. (pages 2898–2913) report that *Arabidopsis* RPW8.2, which is associated with broad-spectrum resistance to powdery mildew, is targeted specifically to the EHM of invading powdery mildew fungi. RPW8 proteins are atypical resistance (R) proteins that contain an N-terminal transmembrane domain and one to two coiled-coil domains, whereas most characterized plant R proteins are nucleotide binding-site, leucine-rich-repeat (NB-LRR) proteins. However, they elicit broad-spectrum resistance to powdery mildew in a similar manner to NB-LRR proteins that function in race-specific resistance, via a conserved signaling pathway dependent on salicylic acid and the defense signaling protein EDS1 (Xiao et al., 2005).

To investigate the function of RPW8, Wang et al. constructed transgenic plants expressing RPW8.2 fused to yellow fluorescent protein (YFP) and used confocal laser scanning microscopy to follow localization after inoculation with powdery mildew-causing *Golovinomyces*. Time-lapse images show RPW8.2-YFP induced after inoculation, and small RPW8.2-YFP–positive vesicles moving toward and fusing with the EHM of developing haustoria (see figure). Localization to the EHM was associated with development of a callose-rich layer encasing the haustorium and with H_2O_2 accumulation in the haustorial complex, leading the authors to speculate on a possible role for RPW8.2 in constraining development of the haustorium and reducing oxidative damage to the host cell. Wang et al. provide evidence that both EHM localization and defense activation are required for induction of broad-spectrum resistance against powdery mildew. For example, *RPW8.2* alleles from *Arabidopsis* accessions lacking resistance to powdery mildew were found to encode proteins that were either unable to activate defense at the EHM or were less efficient in EHM localization.

It has been shown that numerous host PM proteins are absent from the EHM of powdery mildew–infected *Arabidopsis* (Koh et al., 2005), suggesting that the EHM originates from de novo assembly or from the PM with a mechanism for excluding host proteins. Meyer et al. (2009) recently showed that *Arabidopsis* PEN1 syntaxin, involved in protein trafficking and disease resistance at the plant cell wall, is localized to callose-rich haustorial encasements of invading powdery mildew fungi. Preliminary analysis by Wang et al. showed that RPW8.2 targeting to the EHM is not dependent on PEN1, suggesting the existence of another defense-related trafficking pathway to the EHM. Therefore, RPW8.2 might be used as a reporter protein to investigate EHM-specific protein trafficking.

The work of Wang et al. suggests that RPW8.2 functions in broad-spectrum mildew resistance by targeting the haustorial complex of the invading pathogen. In addition, the results support the hypothesis that the EHM is a highly specialized interfacial membrane that may be modified to varying degrees by both pathogen and host to support pathogenesis and defense, respectively.



RPW8.2-YFP (green) is targeted to the EHM of powdery mildew–causing *Golovinomyces* in an *Arabidopsis* epidermal cell. (See also supplemental movie online associated with Wang et al., 2009.)

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www.plantcell.org/cgi/doi/10.1105/tpc.109.210911