EDS1 Contributes to Nonhost Resistance of Arabidopsis thaliana Against Erwinia amylovora

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Erwinia amylovora causes fire blight in rosaceous plants. In nonhost Arabidopsis thaliana, E. amylovora triggers necrotic symptoms associated with transient bacterial multiplication, suggesting either that A. thaliana lacks a susceptibility factor or that it actively restricts E. amylovora growth. Inhibiting plant protein synthesis at the time of infection led to an increase in necrosis and bacterial multiplication and reduced callose deposition, indicating that A. thaliana requires active protein synthesis to restrict E. amylovora growth. Analysis of the callose synthase-deficient pmr4-1 mutant indicated that lack of callose deposition alone did not lead to increased sensitivity to E. amylovora. Transcriptome analysis revealed that approximately 20% of the genes induced following E. amylovora infection are related to defense and signaling. Analysis of mutants affected in NDR1 and EDS1, two main components of the defense-gene activation observed, revealed that E. amylovora multiplied ten times more in the eds1-2 mutant than in the wild type but not in the ndr1-1 mutant. Analysis of mutants affected in three WRKY transcription factors showing EDS1-dependent activation identified WRKY46 and WRKY54 as positive regulators and WRKY70 as a negative regulator of defense against E. amylovora. Altogether, we show that EDS1 is a positive regulator of nonhost resistance against E. amylovora in A. thaliana and hypothesize that it controls the production of several effective defenses against E. amylovora through the action of WRKY46 and WRKY54, while WRKY70 acts as a negative regulator.

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