Nucleo-Cytoplasmic Basal Resistance Regulator EDS1 Connects Pathogen Effector Recognition to Cell Compartment–Specific Immune Responses

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Bacterial type III effectors are recognized by plant intracellular nucleotide binding–leucine-rich repeat (NB-LRR) receptors. So far, processes linking activation of these receptors to downstream defense responses remain elusive.

Lipase-like EDS1 (ENHANCED DISEASE SUSCEPTIBILITY1) is a nucleo-cytoplasmic basal resistance regulator that is indispensible for immunity mediated by TIR (Toll–interleukin-1 receptor)–NB-LRR receptors. Here, our data demosntrate that Arabidopsis EDS1 molecularly connects TIR-NB-LRR disease resistance protein RPS4 recognition of bacterial effector AvrRps4 to defense pathways.

We were able to detect RPS4-EDS1 and AvrRps4-EDS1 complexes inside nuclei of living N. benthamiana cells after transient coexpression and in Arabidopsis soluble leaf extracts after resistance activation. By using various chimeric forms of AvrRps4 fused to domains forcing this effector to be localized to the host cytoplasm or nucleus, we were able to reveal cell compartment–specific RPS4-EDS1 defense branches.

Although nuclear processes are involved in the restriction of bacterial growth,programmed cell death and transcriptional reprogramming associated to resistance necessitate nucleo-cytoplasmic coordination.

This work reveals that EDS1 is a host component targeted by bacterial effector that constitutes an activated TIR-NB-LRR signal transducer for defense responses across cell compartments.

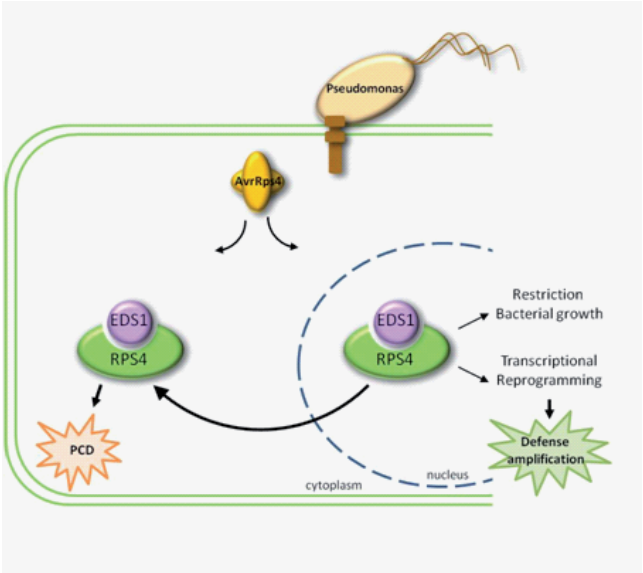

Figure Legend

Model for different subcellular immune outputs triggered by RPS4 with EDS1 in response to Pseudomonas effector AvrRps4.

Bacterial AvrRps4 triggers distinct subcellular defense branches through an RPS4-EDS1 receptor signaling complex that can accumulate in the cytoplasm and nucleus.
RPS4 receptor engages EDS1 to intercept AvrRps4 and transduce receptor activation to downstream defenses.

Arabidopsis EDS[1] connects pathogen effector recognition to cell compartment-specific immune responses. Science 2011; 334:1401-4.