PLANT IMMUNITY TO ROOT-KNOT NEMATODES: PATTERN-TRIGGERED IMMUNITY AND *MI-1*-MEDIATED RESISTANCE

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Pattern-triggered immunity (PTI) and effector-triggered immunity (ETI) are two major forms of plant defense engaging plasma membrane and cytoplasmic localized receptors, respectively. Root-knot nematodes (RKNs; *Meloidogyne* spp.) are plant parasites with a broad host range causing great losses worldwide. To parasitize their hosts, RKNs establish feeding sites in roots known as giant cells. The majority of work studying plant-RKN interactions in susceptible hosts deal with the establishment of the giant cells and not with early defense responses. Here we show that similar to microbial pathogens, early defense or PTI also exists against RKN. To investigate the role of PTI against RKN, we infected Arabidopsis (*Arabidopsis thaliana*) Col-0 and *bak1-5* mutant with RKN and evaluated nematode attraction, penetration and root galling. Although nematodes were equally attracted to roots of both genotypes, nematode penetration and root galling were significantly higher in *bak1-5* roots. Expression of PTI marker genes, *WRKY11*, *MYB51* and *CYP71A12,* were induced in wild-type roots after infection with RKN. Although induction of the transcription factors *WRKY11* and *MYB51* were abolished in *bak1-5* mutant, expression of *CYP71A12*, a cytochrome P450 involved in camalexin biosynthesis, was only attenuated after RKN infection. In addition, the *pad3* mutant, impaired in camalexin production, showed enhanced susceptibility to RKN similar to *bak1-5*. Furthermore, mutants of *BIK1 and RbohD/F,* components of PTI recognition complex, were also more susceptible to RKN. Combined, our results indicate the presence of *BAK1*-dependent and independent PTI against RKN in Arabidopsis.

The tomato (*Solanum lycopersicum; Sl*) gene *Mi-1* mediates ETI against three species of RKN and three phloem feeding insects including potato aphids (*Macrosiphum euphorbiae*). It is not clear how Mi-1 is able to recognize avirulence effectors from these diverse groups of pests and whether the detection of nematode and insect pests involve similar recognition complexes. *Mi-1* encodes a nucleotide-binding leucine-rich repeat immune receptor with no subcellular localization signal. Surprisingly, using confocal microscopy and biochemical fractionation, we found that Mi-1 is localized to three subcellular pools including the plasma membrane, cytoplasm and the nucleus. Using forward genetics, we identified *Somatic Embryogenesis Receptor Kinase 1* (*SERK1*) to be required for *Mi-1*-mediated aphid resistance but not for RKN resistance. SERK1 is a transmembrane protein localized to the plasma membrane. Co-immunoprecipitation experiments in both *Nicotiana benthamiana,* transiently expressing Mi-1 and *Sl*-SERK1, and in 35S-*Sl-SERK1*-HA resistant tomato cultivar Motelle showed that

Mi-1 and *Sl*-SERK1 are present in a complex in the microsomal fractions. Using reverse genetics, we have identified among others, members of WRKY transcription factors, known regulators of plant immunity inducible transcriptional network. *Sl-WRKY72a* and *Sl-WRKY72b* are upregulated by both RKN and aphid feeding and required for resistance to both pests. Interestingly, *Arabidopsis thaliana* WRKY72 seem to regulate a network of genes independent of the known defense hormone salicylic acid. Our work suggests similar *Mi-1*-mediated defense responses to RKN and aphids but distinct Mi-1 recognition complexes to these pests.