The Arabidopsis leucine-rich repeat receptor-like kinases BAK1/SERK3 and BKK1/SERK4 are required for innate immunity to hemibiotrophic and biotrophic pathogens

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Recognition of pathogen-associated molecular patterns (PAMPs) by surface-localized pattern recognition receptors (PRRs) constitutes an important layer of innate immunity in plants. The leucine-rich repeat (LRR) receptor kinases EF-TU RECEPTOR (EFR) and FLAGELLIN SENSING2 (FLS2) are the PRRs for the peptide PAMPs elf18 and flg22, which are derived from bacterial EF-Tu and flagellin, respectively. Using coimmunoprecipitation and mass spectrometry analyses, we demonstrated that EFR and FLS2 undergo ligand-induced heteromerization in planta with several LRR receptor-like kinases that belong to the SOMATIC-EMBRYOGENESIS RECEPTOR-LIKE KINASE (SERK) family, including BRASSINOSTEROID INSENSITIVE1-ASSOCIATED KINASE1/SERK3 (BAK1/SERK3) and BAK1-LIKE1/SERK4 (BKK1/SERK4). Using a novel bak1 allele that does not exhibit pleiotropic defects in brassinosteroid and cell death responses, we determined that BAK1 and BKK1 cooperate genetically to achieve full signaling capability in response to elf18 and flg22 and to the damage-associated molecular pattern AtPep1. Furthermore, we demonstrated that BAK1 and BKK1 contribute to disease resistance against the hemibiotrophic bacterium Pseudomonas syringae and the obligate biotrophic oomycete Hyaloperonospora arabidopsidis. Our work reveals that the establishment of PAMP-triggered immunity (PTI) relies on the rapid ligand-induced recruitment of multiple SERKs within PRR complexes and provides insight into the early PTI signaling events underlying this important layer of plant innate immunity.
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