An Overdose of the Arabidopsis Coreceptor BRASSINOSTEROID INSENSITIVE1-ASSOCIATED RECEPTOR KINASE1 or Its Ectodomain Causes Autoimmunity in a SUPPRESSOR OF BIR1-1-Dependent Manner

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The membrane-bound Brassinosteroid insensitive1-associated receptor kinase1 (BAK1) is a common coreceptor in plants and regulates distinct cellular programs ranging from growth and development to defense against pathogens. BAK1 functions through binding to ligand-stimulated transmembrane receptors and activating their kinase domains via transphosphorylation. In the absence of microbes, BAK1 activity may be suppressed by different mechanisms, like interaction with the regulatory BIR (for BAK1-interacting receptor-like kinase) proteins. Here, we demonstrated that BAK1 overexpression in Arabidopsis (Arabidopsis thaliana) could cause detrimental effects on plant development, including growth arrest, leaf necrosis, and reduced seed production. Further analysis using an inducible expression system showed that BAK1 accumulation quickly stimulated immune responses, even under axenic conditions, and led to increased resistance to pathogenic Pseudomonas syringae pv tomato DC3000. Intriguingly, our study also revealed that the plasma membrane-associated BAK1 ectodomain was sufficient to induce autoimmunity, indicating a novel mode of action for BAK1 in immunity control. We postulate that an excess of BAK1 or its ectodomain could trigger immune receptor activation in the absence of microbes through unbalancing regulatory interactions, including those with BIRs. Consistently, mutation of suppressor of BIR1-1, which encodes an emerging positive regulator of transmembrane receptors in plants, suppressed the effects of BAK1 overexpression. In conclusion, our findings unravel a new role for the BAK1 ectodomain in the tight regulation of Arabidopsis immune receptors necessary to avoid inappropriate activation of immunity.

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