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Chemical control of receptor kinase signaling by rapamycin-induced dimerization

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摘要

Membrane-localized leucine-rich repeat receptor kinases (LRR-RKs) sense diverse extracellular signals, and coordinate and specify cellular functions in plants. However, functional understanding and identification of the cellular signaling of most LRR-RKs remain a major challenge owing to their genetic redundancy, lack of ligand information, and subtle phenotypes of LRR-RK overexpression. Here, we report an engineered rapamycin-inducible dimerization (RiD) receptor system that triggers a receptor-specific LRR-RK signaling independent of their cognate ligands or endogenous receptors. Using the RiD-receptors, we demonstrated that the rapamycin-mediated association of chimeric cytosolic kinase domains from the BRI1/BAK1 receptor/co-receptor, but not the BRI1/BRI1 or BAK1/BAK1 homodimer, is sufficient to activate downstream brassinosteroid signaling and physiological responses and that the engineered RiD-FLS2/BAK1 activates flagellin-22-mediated immune signaling and responses. We also identified the potential function of an unknown orphan receptor in immune signaling and revealed the differential activities of SERK co-receptors of LRR-RKs. Our results demonstrated that the RiD method can serve as a synthetic biology tool for precise temporal manipulation of LRR-RK signaling and for understanding LRR-RK biology.

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