

Salicylic Acid Engages Central Metabolic Regulators SnRK1 and TOR to Govern Immunity by Differential Phosphorylation of NPR1

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Immunity requires a delicate balance between combating infection and preserving essential metabolic functions. However, the mechanisms by which immune responses are coordinated with cellular metabolism remain largely unknown. Here, we show that NONEXPRESSER OF PR GENES 1 (NPR1), the central plant immune regulator of salicylic acid (SA)-mediated defense responses, is controlled by a cascade of posttranslational modifications (PTMs) involving two master nutrient-sensing kinases. In the absence of pathogens, TARGET OF RAPAMYCIN (TOR) inhibits NPR1 through phosphorylation at Ser 55/59. During defense responses, elevated SA reduces sugar phosphate levels and enhances SNF1-RELATED KINASE 1 (SnRK1) activity, which in turn inhibits TOR signaling and phosphorylates NPR1 at Ser-557, a modification required for subsequent PTMs and NPR1 activation. Together, our findings establish SA as a critical coordinator of immunity and growth in response to pathogen challenge by modulating the central metabolic regulators SnRK1 and TOR, which antagonistically control NPR1 activity through phosphorylation.