A Novel G-Protein Activation Mechanism Mediates a TCP14-JAZ3 Circuit in Immune Responses by Repressing JA Signaling

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The plant hormones salicylic acid (SA) and jasmonic acid (JA) act in mutual negative-feedback regulation to balance the plant growth-defense trade-off. Heterotrimeric Gα-Gβ-Gγ proteins are hubs that regulate SA and JA defense signaling. In Arabidopsis, the Gα (GPA1) and Gβ (AGB1) subunits are required for defense against biotrophic and necrotrophic pathogens. We found that G proteins mainly are negative regulators of JA signaling in response to pathogen attack. Both TCP14 and JAZs are transcriptional regulators operating in the JA pathways. Mechanistically, GPA1 interacts with TCP14 within nuclear foci and AGB1 interacts with TCP14 and most of the JAZs regulators including JAZ3. GPA1 slows the proteasomal degradation of the G protein-TCP14-JAZ3 complex, a process is normally promoted by JA and the bacterial virulence effector HopBB1, thus boosting SA-based defense. GPA1 activity is regulated by JA-induced phosphorylation of a conserved residue located near the nucleotide-binding pocket and other residues within the N-terminal α-helix. These phosphomimic mutants, do not affect GTP binding or hydrolysis, but enhance GPA1 interaction with TCP14 and JAZ3, thereby preventing their degradation. This newly discovered phosphorylation-dependent mechanism of de-sequestering G protein partners to modulate transcriptional regulation may extend to both yeast and human cells.