Calmodulin-binding transcription activator AtSR1/CAMTA3 fine-tunes plant immune response by transcriptional regulation of the salicylate receptor NPR1

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Abstract

Calcium signaling regulates salicylic acid (SA)-mediated immune response through calmodulin-meditated transcriptional activators, AtSRs/CAMTAs, but its mechanism is not fully understood. Here, we report an AtSR1/CAMTA3-mediated regulatory mechanism involving the expression of the SA receptor, NPR1. Transcriptional expression of NPR1 increased in knockout mutant, atsr1, independently of SA biosynthesis. AtSR1 directly bound to a CGCG box in the NPR1 promotor. The atsr1 mutant exhibited resistance to the virulent strain of Pseudomonas syringae pv. tomato (Pst), however it was susceptible to an avirulent Pst strain carrying avrRpt2, due to the failure of the induction of hypersensitive responses. These resistant/susceptible phenotypes in the atsr1 mutant were reversed in the npr1 mutant background, suggesting that AtSR1 regulates NPR1 as a downstream target during plant immune response. The virulent Pst strain triggered a transient elevation in intracellular Ca2+ concentration, whereas the avirulent Pst strain triggered a prolonged change. The distinct Ca2+ signatures were decoded into the regulation of NPR1 expression through AtSR1's IQ motif binding to calcium-free-CaM2, while AtSR1's calmodulin-binding domain binding to calcium-bound-CaM2. These observations reveal a role for AtSR1 as a Ca2+-mediated transcription regulator for controlling the NPR1-mediated plant immune response.

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