Uncoupling of sustained MAMP receptor signaling from early outputs in an Arabidopsis endoplasmic reticulum glucosidase II allele

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Recognition of microbe-associated molecular patterns (MAMPs), conserved structures typical of a microbial class, triggers immune responses in eukaryotes. This is accompanied by a diverse set of physiological responses that are thought to enhance defense activity in plants. However, the extent and mechanisms by which MAMP-induced events contribute to host immunity are poorly understood. Here we reveal Arabidopsis priority in sweet life4 (psl4) and psl5 mutants that are insensitive to the bacterial elongation factor (EF)-Tu epitope elf18 but responsive to flagellin epitope flg22. PSL4 and PSL5, respectively, identify beta- and alpha-subunits of endoplasmic reticulum-resident glucosidase II, which is essential for stable accumulation and quality control of the elf18 receptor EFR but not the flg22 receptor FLS2. We notice that EFR signaling is partially and differentially impaired without a significant decrease of the receptor steady-state levels in 2 weakly dysfunctional gII alpha alleles, designated psl5-1 and rsw3. Remarkably, rsw3 plants exhibit marked supersusceptibility against a virulent bacterial phytopathogen despite nearly intact coactivation of MAPKs, reactive oxygen species, ethylene biosynthesis, and callose deposition in response to elf18, demonstrating that these signaling outputs alone are insufficient to mount effective immunity. However, rsw3 plants fail to maintain high transcript levels of defense-promoting WRKY, PR1, and PR2 genes at late time points (4 to 24 h) after elf18 elicitation. This points to an unexpected separation between initial and sustained activation of EFR-mediated signaling in the absence of proper glucosidase II-mediated endoplasmic reticulum quality control. Our findings strongly suggest the importance of sustained MAMP receptor signaling as a key step in the establishment of robust immunity.

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