Regulatory Mechanisms of Pathogen-Mediated Cellular Stress Signaling in Arabidopsis

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Abiotic and biotic stresses can severely perturb endoplasmic reticulum (ER) function. The unfolded protein response (UPR) is a three-pronged signaling axis dedicated to preserving ER homeostasis. If these mechanisms of adaptation and survival are insufficient to recover the ER homeostasis, cells will initiate apoptosis.

My laboratory focuses on two main branches of UPR. GCN2 (general control nonderepressible 2) is a serine/threonine-protein kinase that acts as a global eukaryotic translational regulator. GCN2 mitigates cellular stresses by directly binding with uncharged tRNAs and phosphorylating its target, eukaryotic initiation factor 2 alpha (eIF2α). We demonstrate that GCN2 is involved in ABA signaling and stomatal immunity by affecting the pathogen-triggered stomatal closure and coronatine-mediated stomatal reopening.

Another UPR sensor, IRE1 (Inositol-requiring enzyme 1) directly cleaves bZIP60 (basic leucine zipper 60) mRNA in response to various environmental stresses, leading to the production of an active transcription factor that promotes the expression of multiple ER stress-responsive genes. We identified IRE1 signaling components that activate cellular pro-survival pathways, but under acute or chronic conditions can induce pro-death responses leading to apoptosis.