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Potyviral effector targets and viral co-opting of the untranslated protein response in soybean

Aardra Kachroo, Department of Plant Pathology, University of Kentucky, Kentucky, USA

The seed-borne and aphid-transmitted *Potyvirus,* Soybean mosaic virus (SMV) causes mosaic and severe necrosis in soybean, affecting both seed yield and quality. Analysis of SMV-infected soybean plants shows ultrastructural changes associated with ER membrane reorganization ultimately resulting in the untranslated protein response (UPR). UPR, which involves the accumulation of unfolded proteins at the ER, often occurs during the ER stress response, and is an important coping mechanism for plants undergoing biotic or abiotic stresses. Depending on the severity and length of stress conditions, UPR can either induce autophagy as a mechanism of cell survival or result in cell death. The mechanisms underlying ER stress activation or viral perception by ER stress sensors in the plant are not known. Our recent work suggests that physical interactions between soybean eukaryotic elongation factor alpha (eEF1A) and the ER-localized SMV P3 protein might mediate viral perception leading to UPR. Chemical induction of UPR promotes SMV infection in soybean. Conversely, knockdown of *eEF1A* expression inhibits the ability of soybean plants to induce UPR, and enhances soybean resistance to SMV. Thus, plants lacking eEF1A or the associated eEF1B show reduced UPR in response to SMV infection and better resist viral accumulation. P3-responsive changes in the subcellular localization of eEF1A and the involvement of eEF1A in the soybean cell death response are reminiscent of the effects of the Human immunodeficiency virus 1 Nef protein on mammalian eEF1A and programmed cell death. This raises the possibility that P3 affects eEF1A function by altering its subcellular localization in infected cells. The comparable virulence-related roles of SMV P3 and HIV-1 Nef in promoting viral virulence further suggest possible parallel mechanistic functions for these viral proteins.