Title: Analyzing plant stress granules in response to plant viruses

Abstract:

Plant viruses have the ability to redirect host machineries and processes to establish a productive infection. Virus-host interactions lead to the reprogramming of the plant cell cycle and transcriptional controls, inhibition of cell death pathways, interference with cell signaling and protein turnover, and suppression defense pathways. Stress granules (SGs) are structures within cells that regulate gene expression during stress response, e.g. viral infection. In mammalian cells assembly of SGs is dependent on the Ras-GAP SH3-domain–binding protein (G3BP). The C-terminal domain of the viral nonstructural protein 3 (nsP3) of Semliki Forest virus (SFV) forms a complex with mammalian G3BP and sequesters it into viral RNA replication complexes in a manner that inhibits the formation of SGs. The binding domain of nsP3 to HsG3BP was mapped to two tandem 'FGDF' repeat motifs close to the C-terminus of the viral proteins. It was speculated that plant viruses employ a similar strategy to inhibit SG function.
We are currently characterizing the G3BP-like protein family of Arabidopsis thaliana. We can show that all 7 members of this family form cytoplasmic-localized granules upon heat shock and co-localize with known SG marker proteins. Moreover, the nuclear shuttle protein (NSP) of the begomovirus Abutilon mosaic virus (AbMV), which harbors a 'FVSF'-motif at its C-terminal end, interacts with one the AtG3BP-like proteins, as does the 'FNGSF'-motif containing NSP of Pea necrotic yellow dwarf virus (PNYDV), a member of the Nanoviridae family. P1 of Turnip mosaic virus (P1-TuMV) harbors an FGSF-motif and FGSL-motif at its N-terminal end. These motifs are also predicted to be a binding motif for the host protein G3BP. This suggests that also P1-TuMV interacts with G3BP to control and regulate plant SGs to optimize cellular conditions for the production of viral proteins. P1-TuMV co-localized with AtG3BP under stress conditions and interaction was shown by co-immunoprecipitation assays, bimolecular fluorescence complementation (BiFC) experiments and fluorescence resonance energy transfer/fluorescence-lifetime imaging microscopy (FLIM-FRET). Alanine substitution mutants reveal that both motifs are necessary to target P1-TuMV into stress granules or G3BP interaction, respectively. In the mammalian system, virus interference with G3BP is often a strategy of the virus to inhibit SG formation. We therefore propose that SG formation upon stress is conserved between mammalian and plant cells and that plant viruses may follow a similar strategy to inhibit plant SG function as it has been shown for their mammalian counterparts.

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