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Opinion Article

Short Note on the Role of RNA in Stress Granules

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DESCRIPTION

Hormones and neurotransmitters activate the Gαq/phospholipase Cβ1 (PLCβ1) signaling system eliciting cellular calcium responses. Beside the plasma layer, PLCβ1 has a cytosolic population that binds stress granule related proteins preventing their collection, and activation of Gαq shifts PLCβ1 to the membrane releasing bound proteins and advancing the development of stress granules. The cellular impact of stress granules shaped upon routine Gαq protein signaling is obscure. Here, we have characterized Ago_2 stress granules formed in response to neurotransmitter activation in cultured PC_{12} cells. We observe these stress granules have a distinct protein composition, and unlike stress granules formed upon heat shock, contain just two mRNA transcripts, chromogranin B, which is engaged with secretory capacity, and ATP synthase 5f1b, which is expected for ATP synthesis. Our investigations show a surprising pathway where Gαq/PLCβ manages the interpretation of specific proteins.

Binding of extracellular ligands like acetylcholine, serotonin and histamine, to their particular G protein coupled receptor will actuate Gαq, one of the four significant G proteins pathways. Gαq, thusly, actuates phospholipase Cβ, which catalyzes the hydrolysis of the signaling lipid phosphoinositide 4,5 bisphosphate leading an expansion in intracellular calcium. Alongside this significant layer work, PLCβ1 has been found to have an abnormal cytosolic population that ties to the advertiser of RNA-induced silencing, C_3PO, as well as a several proteins involved with pressure granules formation. Stress granules are ended ribosomal edifices that safeguard mRNAs under stress conditions, for example, arsenite treatment, heat/cold shock and osmotic stress. Proteins that bind spot PLCβ1 include $_e$FI5A, Polyadenylate Binding Protein (PABPC1) and Ago_2. Ago_2 is moreover the nuclease component of the RNA-induced silencing complex (RISC) that degrades mRNA with the assistance of C3PO. Whenever Ago2-bound mRNA matches impeccably with a bound miR, Ago_2 transitions to an active conformation to hydrolyze the mRNA. In any case, assuming matching is defective, it will frame a slowed down complex resulting in stress granules.

Our new study showed that reducing the cytosolic PLCβ1 population increases the number and size of particles containing Ago2 along with the pressure granule makers Polyadenylate-binding protein 1(PABPC1) and G3BP1. Restricting among PLCβ1 and stress granule proteins assists keep them with scattering, while initiation of Gαq promotes relocalization of cytosolic PLCβ1 to the plasma film, advancing arrival of bound proteins and the development of stress granules. This system proposes that Gαq might be associated with protein translation through cytosolic PLCβ1.

We have characterized the composition of Ago_2 stress granules formed in response to neurotransmitter activation in differentiated PC_{12}, and contrasted these with traditional stress responses. PC_{12} cells have a large endogenous articulation of Gαq and PLCβ1, and although not neuronal in origin, when treated with nerve growth factor, the cells take on neuronal morphology and secrete particles mimicking synaptic vesicles. We find that Gαq activation produces pressure granules that have an unmistakable protein composition when contrasted with different stress. Likewise, not at all like heat shock that contain different mRNA and miRs, Gαq stress granules contain just two major mRNA transcripts. Our studies show a connection between physiological G protein activation and protein translation.

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