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Characterization of AICD-Grb2 vesicle trafficking

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The ascertainment of elevated levels of Amyloid Precursor Protein Intracellular domain (AICD) in Alzheimer's Disease (AD) L brains and the fact that it contributes to AD like pathology has geared the search towards a new paradigm1. It is reported that hippocampal and select cortical neurons in AD exhibit phenotypic changes indicative of neurons re-entering the cell division cycle2,3. Although in normal brain AICD is known to colocalize with its interactor Growth factor receptor-bound protein 2 (Grb2) throughout the cell body and dendrites, in the AD brain the localization is reported to be confined to the neuronal cell body4. Of late we have established from our group that Grb2 co-localized to compartments along with APP and/or AICD affecting their turnover by inhibiting the exosomal exit route5. The vesicles formed in a clathrin and dynamin independent manner and matured into autophagosomes. Merging of these autophagosomes with lysosome relieved the cells of AICD overload and established, for the first time, the role of Grb2 in autophagy and in handling protein overload

Biography

Kasturi Roy is a 3rd year Graduate student at Structural Genomics Division, Saha Institute of Nuclear Physics, Kolkata, India. Using cell biological techniques she is currently trying to understand the molecular events behind autophagy in Alzheimer's disease scenario. Debashis Mukhopadhyay, an Associate Professor with an initial training in Structural Biology, has co-authored about thirty papers to his credit. Currently his group is involved in correlating adult neurodegeneration and neurogenesis

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