

A Study of the Impact of Aging on Autophagic Vacuole Maturity in the Synapse

Fathima Syed, Hilary Grosso Jasutkar

Rutgers University, Institute for Neurological Therapeutics, Robert Wood Johnson Medical School

Macroautophagy, herein referred to as autophagy, is the process by which cells break down old or damaged cytosolic proteins and organelles and plays an important role in the homeostasis and normal function of the synapse. Autophagy in neurons begins with the formation of an immature phagophore in the synapse, which travels via retrograde transport to the soma, simultaneously progressing into a mature autophagic vacuole (AV). This process is implicated in having an important role in neuronal processes such as synaptic plasticity and neurotransmitter release. The importance of autophagy increases as a person ages, while disrupted autophagy has been associated with Alzheimer's disease. The purpose of this study is to examine how the retrograde transport of autophagic vacuoles changes with age. Immunofluorescent imaging was used to assess the density and maturity of AVs in the axons of mice at different ages. Brain sections will be labeled with different markers for the corresponding stages of AV maturity and a marker to visualize the axon. Brain sections from mice expressing GFP-tagged LC3, a protein found on all AVs, were labeled with an axonal marker and markers of AV maturity and then visualized using a Leica microscope. We hypothesize that there is decreased retrograde transport, and therefore maturation, of AVs as the mice age. We therefore expect that more immature AVs will accumulate at the distal axon and that there will be an overall lower number of AVs at the soma in older mice. Supported by NJ ACTS NIH R25TR004777 CREST Program.

