Tissue-Specific Requirement of Autophagy Gene Atg-18 Is Essential for Lipid Metabolism Regulated by Insulin-like Signaling in Caenorhabditis elegans

Jia, Ray (School: Alexander W. Dreyfoos School of the Arts)

The aim of this experiment was to expand upon the relationship established in previous studies between insulin-like signaling regulated autophagy in chemosensory neurons and lipid metabolism in the model organism Caenorhabditis elegans. This year's study focused on broader tissue-specific expression of autophagy. Five new promoters were used: Punc-119, Pges-1, Pdpy-7, Pmyo-3, which is expressed in neuronal cells, intestinal cells, hypodermis, and body wall muscle, respectively. The unc-64 gene was also mutated to determine any role neurotransmitters play in this process. Four strains were used as control: daf-2, daf-2;atg-18, daf-2;atg-18[Patg-18::atg-18], and N2. The test population was comprised of one hundred dauer larvae (L3 larvae for N2 strain). Standard Sudan Black B staining protocol was used. A Zeiss Imager microscope equipped with an AxioCam digital camera was used to photograph the stained worms. The images were quantified using ImageJ/Fiji and Photoshop CC. Using Prims 5, the experimental strains data were compared to the control strains. The data obtained corresponded to previous study data and supported this study's hypotheses. Expression of atg-18 in neurons ($p = 0.3213$) or intestinal cells ($p = 0.3326$) completely restored fat accumulation in daf-2;atg-18. The data supported partial restoration of fat accumulation when atg-18 was expressed in hypodermis ($p = 0.0185$) and body wall muscles ($p = 0.0021$). Mutation of the unc-64 gene showed no statistically significant influence on fat accumulation ($p = 0.1327$). The mutation of both atg-18 and unc-64, however, led to restoration of lipid accumulation ($p = 0.0168$). This suggests that certain neurotransmitters can act downstream of atg-18 to regulate the influence of IGF signaling on fat metabolism.

Awards Won: