Smells Like a Solution Autophagy in Chemosensory Neurons Is Essential for Lipid Metabolism Regulated by Insulin-Like Signaling in Caenorhabditis elegans

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The aim of this experiment was to examine how rescuing autophagy, a lysosomal degradation pathway, in the chemosensory neurons of the model organism Caenorhabditis elegans affects lipid accumulation. By rescuing autophagy in C. elegans chemosensory neurons, including olfactory neurons, there is a possibility that we can discover a new pathway by which lipid accumulation can be regulated. Four gene promoters were used to express autophagy gene atg-18 in specific chemosensory neurons; Punc-42, Pgpa-3, Podr-2, and Pdaf-11, and three strains were used as controls: daf-2, daf-2;atg-18, and N2. One hundred dauer larvae (L3 larvae for N2) were picked as a test population. A standard Sudan Black B protocol was used to stain the worms. Stained worms were mounted on slides and images of the worms were captured using a Zeiss Imager microscope equipped with an AxioCam digital camera. To quantify the images, ImageJ/Fiji and Photoshop CS3 were used. The experimental strains data were compared with the control strains. Lipid accumulations showed upwards of a 40% increase in the predicted experimental worms. A t test was performed, and the statistical analysis showed significant difference (p-value less than 0.05 in all comparisons). It can be concluded that increased autophagy in the eleven chemosensory neurons of C. elegans leads to increased lipid accumulation when IGF-1 signaling is suppressed. These data is very significant. Future research can be conducted on pharmaceutical regulation of autophagy in olfactory neurons to suppress lipid accumulation and treat obesity, diabetes, and even cancer.

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