Mitophagy upregulation as a therapeutic strategy for Alzheimers disease

https://neurodegenerationresearch.eu/survey/mitophagy-upregulation-as-a-therapeutic-strategy-for-alzheimers-disease/

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USA

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Mitophagy upregulation as a therapeutic strategy for Alzheimers disease

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1

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Research Abstract

We seek to explore the relationship between mitochondrial dysfunction and accumulation of damaged mitochondria in AD. We have established C. elegans models to investigate whether mitophagy contributes to AD pathology. We have detected mitochondrial quality in wild type (N2) and hu-Tau or Abeta-overexpressed AD C. elegans models (RB809/ptl-1 (ok621); BR5270 (byIs161); CL2120 (dvIs14); CL2355) using confocal microscopy, electron microscopy, and Seahorse respirometry. All the AD worms showed accumulation of damaged mitochondria,

impaired mitochondrial network, and decreased basal oxygen consumption. We propose that defective mitophagy contributes to accumulation of damaged mitochondria in AD. Mitophagy will be evaluated using multiple techniques such as immunoblotting for protein expression, a mitophagy reporter worm strain, mitophagy dyes, and the mt-mKeima reporter. Our preliminary data indicate defective mitophagy in Tau and Abeta overexpressing cells. In the future, we apply therapeutic strategies to modulate mitophagy and offset AD mitochondrial phenotypes. We will investigate whether mitophagy induction can improve healthspan endpoints (swimming movement; pharyngeal pumping; maximum velocity), and extend lifespan of AD worms models.

Further information available at:

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United States of America

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