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RESEARCH AREA

One of the serious health and economic problems of our time is the increasing ageing of the population in developed societies. Increased longevity is associated with the risk of many diseases of old age. We must invest more and more energy and resources in the symptomatic treatment of neurodegenerative diseases, which are still incurable. The primary goal of our research group is to identify new regulatory mechanisms that enhance the quality of life in old age and prevent the onset of various neurodegenerative diseases. For ageing, the survival of neurons is essential, as these cells cannot divide and regenerate themselves. Autophagy is a cell-protective degradative process in which cells eliminate their harmful components (toxic molecules that are involved in neurodegenerative diseases). Currently available autophagy activators are not specific enough and can cause numerous adverse side effects. We expect our findings to identify new regulatory points that can be used to activate autophagy to promote cell survival specifically. These new regulatory points could be used to design drug candidates to prevent the progression of dementia or neurodegenerative diseases in later life.

TECHNIQUES AVAILABLE IN THE LAB

Drosophila melanogaster is one of the best-known model organisms. The *Drosophila* genome contains the orthologs of more than 70% of the genes that cause human. *Drosophila* is also an excellent candidate for research on ageing and neurodegeneration, due to its short lifespan and the fact that its nervous system is composed of neurons and glia cells similar to humans. In our experiments, our research group uses a combination of genetic, cell imaging, and molecular tools. Our research involves the investigation of several neurodegenerative models from a pharmacological and genetic point of view.

SELECTED PUBLICATIONS

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Sturm, Á., Saskői, É., Hotzi, B., Tarnóci, A., Barna, J., Bodnár, F., Sharma, H., **Kovács, T.**, Ari, E., Weinhardt, N., Kerepesi, C., Perczel, A., Ivics, Z., & Vellai, T. (2023). Downregulation of transposable elements extends lifespan in *Caenorhabditis elegans*. *Nat Commun* **14**(1): 5278.

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