

Wayne State University

Medical Student Research Symposium

School of Medicine

March 2024

Activating Neuroprotective Effects of Glial Cells in Vps13Dassociated Ataxia

Abrielle Fretz hn3127@wayne.edu

Follow this and additional works at: https://digitalcommons.wayne.edu/som_srs

Part of the Medicine and Health Sciences Commons

Recommended Citation

Fretz, Abrielle, "Activating Neuroprotective Effects of Glial Cells in Vps13D-associated Ataxia" (2024). *Medical Student Research Symposium*. 352. https://digitalcommons.wayne.edu/som_srs/352

This Research Abstract is brought to you for free and open access by the School of Medicine at DigitalCommons@WayneState. It has been accepted for inclusion in Medical Student Research Symposium by an authorized administrator of DigitalCommons@WayneState.

Activating Neuroprotective Effects of Glial Cells in Vps13D-associated Ataxia

Ataxia is a common symptom among many neurological conditions. Due to its high prevalence and quality of life impact, there is significant interest in understanding the underlying cause, as well as potential therapeutic approaches to mitigate its severity. Our lab uses a model of Vps13D-associated ataxia in the fruit fly, Drosophila Melanogaster, which recapitulates aspects of the human disease including age-dependent motor deficits.

The anti-oxidant compounds gastrodin and 4-HBA were found to have neuroprotective effects in *Drosophila* and mouse models of Parkinson's disease. Previous work has demonstrated that these compounds can activate the Nrf2-pathway, a master regulator of anti-oxidant defenses. These compounds are believed to act through supportive glial cells, upregulating their anti-oxidant associated assistance of degenerating neurons.

We supplemented Drosophila diet with low-dose (0.1mM) and high-dose (1mM) concentrations of gastrodin and 4-HBA to determine the impact of these compounds on ataxia in our animal model. Flies' motor ability was tested through negative geotaxis assays at one and two weeks of age. Supplemented animals were compared to untreated controls.

We found that low-dose gastrodin may improve motor function in later life. In addition, low-dose 4-HBA supplementation may improve motor function early in the disease course. Gastrodin is a glycoside precursor of 4-HBA, so these data are interesting in their differing temporal effects. The lack of similarity may indicate a complex metabolic pathway or extensive interactions within glial cells. These data lay the foundation for further investigation into the possibility of glial cell antioxidant activation in the treatment of neurodegenerative ataxia.