**Batten disease** is an uncurbable juvenile neurodegenerative disorder, where symptoms don’t appear until the age of four and progress throughout one’s lifespan [1]. Believed to be caused by a knockout mutation in CLN3 gene, this disease creates vision loss, degradation in cognitive activity, as well as diminishes nearly all motor function [1]. Cln3 proteins are transmembrane proteins that reside near the neuronal contractile vacuole, and are believed to be involved in osmoregulation [1,2]. *Cln3 protein function is not entirely known, however, there is buildup of substances such as water (from a lack of osmoregulation) and gangliosides (glycosphingolipids with one or more sialic acids found on the surface of neuronal cells) that are suggestive of its function*[2]*.* Osmoregulation is essential for cell life, and the buildup of gangliosides inhibits the passing of neurotransmitters through the synapse, meaning it is essential to understand the role of Cln3, to prevent this disease from occurring.

**My long-term goal** of this research is to understand the function of the CLN3 protein, and what exact mutation in the protein sequence causes this disease. **My primary goal** is to better understand the role the Cln3 protein has with trafficking gangliosides in vesicles, between neurons. **My hypothesis** is that the Cln3 protein transports gangliosides into the neuronal lysosomes, which when “full” is expelled from a presynaptic cell via vesicles. My model organism will be the *Dictyostelium,* whose genome contains homologs of 11 of the 13 NCL genes [3]. Also, *Dictyostelium* has been widely studied for other neurodegenerative diseases for its single-celled and multi-cellular processes [3].

References:

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