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Bacterial and Rare Diseases 2019: The effect of G *lucidum* on the lifespan of caenorhabditis *elegans* modelling duchenne muscular dystrophy- Prashanthi Rayapati- Lynbrook High School

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Duchenne muscular dystrophy (DMD) is an X chromosome-linked disease characterized by progressive physical disability, premature death and immobility in affected boys which are Underlying the devastating symptoms of DMD is the loss of dystrophic, a structural protein that connects the extracellular matrix to the cell cytoskeleton and provides protection against contraction-induced damage in muscle cells, leading to chronic peripheral inflammation. Dystrophin is expressed in neurons within specific brain regions, including the hippocampus, a structure associated with learning and memory formation. Linked to this, a subset of boys with DMD exhibit progressing cognitive dysfunction, with deficits in verbal, short-term, and working memory. Genetically comparable dystrophic deficient mouse model of DMD, but not all types of memory and learning are deficient in specific deficits in synaptogenesis and channel clustering at synapses. Majorly it has been given to the cognitive deficits associated with DMD compared with the research conducted into the peripheral effects of Dystrophin deficiency. Therefore, this review focuses on what is known about the role of full-length Dystrophin (Dp427) in the hippocampal neurons. In this experiment, I hypothesized that 100 ug/ml of G. l*ucidum* would extend the lifespan and too much concentration of this herbal medicine would lose its efficacy in treating this disease. A study was conducted through the reactions and lifespan of Caenorhabditis *elegans* exhibiting the lack of Dystrophin to the different concentrations of G. l*ucidum*. As a result, the effect of *G. lucidum* on the *Caenorhabditis elegans* modelling Duchene Muscular Dystrophy was astonishing as 100 ug/ml of *G. lucidum* helped prolong the lifespan of these nematodes by 20%. This data can be reflected onto the lifespan of humans with DMD as the 20% increase in lifespan of these nematodes could mean the prolonged life of 6-8 years for humans. Therefore, too much concentration of *G. lucidum* was shown not to affect the life of the worms. The hypothesized argument was proven correct as the results show the 20% increase of lifespan for the 100 ug/ ml of *G. lucidum* concentration and the effect of too much concentration of this herbal method.

Moreover, the use of herbal medicine like *G. lucidum* could be a new inexpensive and attainable method of treatment for those diagnosed with DMD. The Dystrophin has more importance in learning and memory is assessed, and the potential importance that inflammatory mediators, which are chronically elevated in dystrophinopathies, may have on hippocampal function is also evaluated. Duchenne muscular dystrophy (DMD) is a genetic disorder causes due to loss of the protein Dystrophin. In humans, DMD has early onset, causes developmental delays, muscle necrosis, loss of ambulation, and early death. Currently the animal models have been challenged by their inability to model the early onset and severity of the disease. Thus it remains unresolved if increased sarcoplasmic calcium observed in dystrophic muscles follows or leads the mechanical insults caused by the muscle's disrupted contractile machinery. More important applications for patients and as potential physiotherapeutic treatments it may either help or exacerbate symptoms, depending on dystrophic muscles differ from healthy ones. Recently we showed how burrowing dystrophic (dys-1) C. Here we report dys-1 worms display early pathogenesis, dysregulated However, progress resulted from matching Dystrophin mutations with sensitizing mutations in other related proteins (or through other insults), As animals are more closely recapitulate to the acute motor and muscular decline seen in humans. These approaches are useful, but also cloud interpretation of their results as it becomes challenging to unequivocally assign a phenotype to loss of Dystrophin. Lack of phenotypic severity in these systems may result from compensatory mechanisms (e.g. utrophin upregulation14, 19), and/or the shorter lifespan of these animal models.

Alternatively, their attenuated phenotypes may result from insufficient muscular challenge to animals that are (mostly) kept under low exertion regimens not matching those they would experience in their natural environment. Consistent with this idea, muscle degeneration was directly correlated with strength of muscular contraction in (mdx) mice modelling DMD2 sarcoplasmic calcium, and increased lethality. Sarcoplasmic calcium dysregulation in Dystrophin worms precedes the structural phenotypes (e.g. mitochondrial and contractile machinery damage) and can be mitigated by silencing calmodulin expression. We cultivated animals in environments requiring high amplitude, with high frequency of muscle exertion during locomotion. We find that several muscular parameters sizes improve with their increasing activity. However, longevity in dystrophic animals was negatively associated with muscular exertion, regardless of the duration of the effort. The high degree of phenotypic conservation between dystrophic worms and humans provides a

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unique opportunity to gain insights into the Etiology of the disease, as well as the initial assessment of potential treatment strategies.

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