OFFICIAL ABSTRACT and CERTIFICATION

GFP Tagged Mitochondrial IMG1 and BSCI Proteins Disrupt Normal Huntingtin Inclusion Body Formation in Saccharomyces cerevisiae

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A known characteristic of Huntington's disease is the unfolding of the Huntingtin protein and aggregation of this unfolded protein known as inclusion bodies (IBs). A clone collection of proteins that were hypothesized to interact with huntingtin IB were created and strains were created in which the protein being studied was tagged with gfp and mHtt was tagged with mCherry using a Saccharomyces cerevisiae model. Since tagging proteins is known to disrupt their function, any change in the IBs can be accredited to the tagging of one specific protein which supports the idea that the specific protein plays a role in IB formation and regulation. Two proteins were found to have a noticeable impact on IBs, creating one large IB and smaller aggregates in one cell which is very different than the parental strain which only has one normal sized aggregate per cell. These proteins were BSCI and IMG1, both mitochondrial proteins that play an important role in respiration and are vital to cell growth. Although the exact process is unknown, it is hypothesized that tagging these proteins altered their function and affected the IBs. There has been an established correlation between mitochondrial degeneration and other neurodegenerative diseases such as Parkinson's disease and Alzheimer's disease. Based on previously published papers and the correlations between mitochondrial proteins and IB formation found in this study, new avenues of study can come about and scientists can find different ways to alter the pathogenesis of Huntington's disease.

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