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DOES TDP-43 IMPACT NEURONAL PROTEOSTASIS?

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Proteostasis, the process by which a cell maintains protein synthesis, folding, and clearance is critical for survival; however, the fidelity of this process declines with age. Disturbances in proteostasis contribute to many age-related neurodegenerative diseases, such as amyotrophic lateral sclerosis (ALS), leading to the accumulation of misfolded proteins. The majority of patients with this disease accumulate an aggregated form of the ALS associated protein TDP-43 in neurons, suggesting a disruption in proteostasis. Here, we are using the nematode *C. elegans* to examine whether the protein TDP-43 challenges proteostasis by altering expression of genes that participate in three branches of proteostasis; protein synthesis, protein folding, and protein clearance. *C. elegans* short life cycle, transparency, and conservation of genes with human orthologues provides advantages for experimentation. In this study, upregulated or downregulated genes responsible for proteostasis will be identified in TDP-43 expressing animals. Currently, only four proteostasis related genes, *rpl-11.2, hsp-70, skr-5, and skr-9* have at least a two-fold change in expression in TDP-43 animals compared to wild type. This represents a small fraction of genes within the proteostasis network suggesting that TDP-43 expression has little impact on proteostasis. In future experiments, gene expression changes will be examined in aged animals to enhance imbalances in proteostasis. Investigating genes responsible for proteostasis will further contribute to our understanding of the pathology of neurodegenerative diseases.