

Iron Effects on a Model of Huntington Disease

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Introduction: Neurodegenerative diseases have become more prevalent and of great epidemiological importance. These diseases are characterized by progressive accumulation of proteins aggregates and disruption of the proteostasis. Iron (Fe) is an important metal to the organism homeostasis, which abundantly exist in the environment and has also been linked to the etiology of neurodegenerative diseases such as Huntington's disease (HD). HD is a fatal and late-onset neurodegenerative disorder with an expansion of a glutamine as PolyQ aggregates in the huntingtin protein, which leads to neuronal degeneration and death. Notably, *Caenorhabditis elegans* models of protein aggregation have provided some key insights into mechanisms that promote aggregation. Several studies show *C. elegans* as a potential model for toxicity testing and also as an alternative method to replace animal models. **Objectives:** the aim of this study was to evaluate the effects of Fe in life and healthspan using transgenic worms for HD. **Material and Methods:** Nematodes used in this study were wild-type N2 and HA659(rtl11 [osm-10p::Htn-Q150, osm-10p::OSM-10::GFP]), obtained from Caenorhabditis Genetics Center, Minnesota, USA. First larval stage (L1) worms were exposed to Fe concentrations of 0.5mM and 1mM. The acute exposure lasted 30 minutes in the absence of food. 48h after the exposure, the lifespan and behavior assays were performed. **Results and Discussion:** Our study showed that Fe exposure decreases *C. elegans* life and healthspan which includes decreased locomotor activity and decreased mechanosensitivity, demonstrating that Fe may accelerate protein aggregation that leads to neurodegeneration. **Conclusions:** These results suggest a dysfunction of the nervous system which can be associated with misfolded protein accumulation. More studies should be done about the role of Fe in protein aggregation and its toxicity mechanisms in neurodegeneration.

Key-words: Neurodegeneration, Healthspan, Locomotor behavior