**Acute iron exposure increases ROS levels in *C. elegans***

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**INTRODUCTION:** Iron (Fe) exists in abundance in the environment and we are constantly exposed to Fe mainly via food intake. Normally, Fe absorption is regulated physiologically to avoid iron toxicity. It has been is a central factor in Fenton reaction and, consequently, in OH⁻ production, which is expected to damage biomolecules and to contribute to oxidative stress leading to neurodegeneration. The *Caenorhabditis elegans*, a round worm that lives in the soil, has several advantages as handling and maintenance in the laboratory. In this study we evaluated the ROS levels in *C. elegans* exposed to Fe.

**MATERIALS AND METHODS:** L1 worms were exposed to Fe concentrations of 0.05mM; 0.1mM; 0.5mM; 1mM; 1.5mM e 2mM. The acute exposure lasted 30 minutes in the absence of food. After Fe exposure, L1 worms (10.000 per tube) were frozen and thawed twice and homogenized by sonication and then centrifuged. The 2’7’ dichlorodihydrofluorescein diacetate (DCF-DA) was added and their fluorescence levels were measured (excitation: 485 nm; emission: 535 nm) in a fluorimeter. Measurements were repeated 3 times, each condition was performed in duplicate.

**RESULTS AND DISCUSSION:** The lower Fe concentrations showed to cause increase in ROS levels in comparison to control group. On the other hand, the higher concentrations showed a decrease in ROS levels, probably due to dead animals that did not absorb the DCF-DA probe. Reactive oxygen species (ROS) play various important roles in living systems and their excess has been implicated in numerous diseases and aging.

**CONCLUSIONS:** In conclusion, our data suggest that the Fe toxicity is able to increase ROS levels even at low concentrations and can be associated to changes in behavior and others endpoints. More experiments have to be made to comprove and understand the consequences of this oxidative stress in worms, especially in the neurons system.

Keywords: Iron, Oxidative stress, Reactive oxygen species.

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