EXCESS DIETARY SUGAR DISRUPTS COMPLEX I ACTIVITY AND MITOCHONDRIAL EFFICIENCY IN *DROSOPHILA MELANOGASTER*

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INTRODUCTION: Although carbohydrates are necessary for the proper functioning of all organisms, an excess of sugar in the diet causes profound metabolic changes. Understanding the mechanisms driven by a sugar overload can provide insights on metabolic disorders such as type 2 diabetes, and the influence of external factors such as diet. OBJECTIVE: Using *Drosophila melanogaster* (*D. melanogaster*) exposed to a diet containing high concentration of sugar, we evaluated the effects of excess sugar on mitochondrial functionality. MATERIALS AND METHODS: *D. melanogaster* flies of the Oregon-R line were fed on standard or high-sugar diet (30% sucrose) for a period of seven days during the adult phase, being kept in an environment with a controlled temperature of 24 degrees Celsius and a 12-h light and dark cycle. Mitochondrial functionality was evaluated by high-resolution respirometry using the Oroboros equipment, where the homogenate of two flies was added to the chamber containing the mitochondrial respiration medium MIR05. The assay followed the substrate-uncoupler-inhibitor titration protocol (SUIT protocol) to measure the different respiration states. All statistical analyses were performed using the GraphPad Prism 8 program, with normality testing followed by the T-test. RESULTS AND CONCLUSION: Excess sugar in the flies' diet caused changes in mitochondrial respiration. Sugar diet caused a significant decrease in the oxidative phosphorylation associated with the mitochondrial complexes 1 and 2, and also a decrease in the electron transfer capacity of complex 1. These events decreased the OXPHOS coupling efficiency and bioenergetic efficiency of mitochondria. Our findings demonstrate that excess dietary sugar causes mitochondrial dysfunction in *D. melanogaster*, primarily through reduced activity of complex I, indicating a potential mechanism by which high sugar intake impairs cellular energy metabolism.

Key-words: mitochondria; metabolic disorders; respirometry; oxidative phosphorylation; bioenergetic efficiency;

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