

INFLUENCE OF MELATONIN SUPPLEMENTATION ON THE MUTAGENICITY OF MICE FED A CAFETERIA DIET

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INTRODUCTION: Obesity and associated metabolic comorbidities are a worldwide public health problem. It is related to development of chronic low grade systemic inflammation in adipose tissue associated with an increase in reactive oxygen species and a decrease in antioxidant defenses. Due to its alterations, obesity leads to damage to our macromolecules, such as DNA. Melatonin supplementation (MEL) has been studied as a therapeutic approach in this context, due to its antioxidant and anti-inflammatory properties. Animal research uses the cafeteria diet (CAF) as a model for inducing obesity, as it is capable of generating metabolic syndrome in rats, leading to an increase in visceral adipose tissue. **OBJECTIVE:** This study was to evaluate the mutagenic effect of CAF and antimutagenic effect of MEL through Micronucleus Test (MN). **MATERIALS AND METHODS:** Was carried out for 21 weeks with 48 male Swiss mice, divided into 6 groups (n=8): standard diet (SD) fed with standard chow (SC); SD+MEL fed with SC and supplemented with MEL; CAF fed with CAF; CAF+MEL fed with CAF and supplemented with MEL; CAF/CAF+MEL fed with CAF in the first 17 weeks and in the last 4 weeks they started the MEL consumption; CAF+MEL/CAF fed with CAF and supplemented with MEL in the first 17 weeks and in the last 4 weeks they stopped the consumption of MEL. The melatonin solution was available in light-protected water bottles (2 mg/L). CAF was composed of foods such as pound cake, sausage, among others. At 21st week the animals were euthanized and the bone marrow was removed to perform the MN. **RESULTS:** The results have demonstrated mutagenicity in the CAF group in polychromatic erythrocytes (PCE), when compared to the SD group. The CAF+MEL and CAF/CAF+MEL groups showed a reduction in micronucleated PCE when compared to the CAF group. No significant differences were observed in the PCE/NCE ratio. **CONCLUSION:** MEL proved to be effective in attenuating DNA damage caused by obesity, and its antioxidant effects are more satisfactory when supplemented concomitantly with metabolic, molecular and biochemical changes.

Keywords: Obesity; Antimutagenicity; Micronucleus test.

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