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Biochemical and behavioral consequences of ethanol intake in a mouse model of metabolic syndrome

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Abstract

Background: Alcohol abuse is common in people with sedentary lifestyles, unbalanced diets and metabolic syndrome (MS). Both, alcohol abuse and MS have negative effects on the CNS inducing cognitive impairment and impaired brain oxidative status. Considering that a few studies have focused on the combined effects of both conditions in the brain, the aim of this work is to elucidate the effects of alcohol intake in a mouse model of MS, at the behavioral and biochemical level. Methodology: Control (B6.V-Lep ob/+ JRj) and MS (B6.V-Lep ob/obJRj) male mice aging 4 weeks were used in the study, divided in four groups: control (C), ethanol (E), obese (Ob), obese-ethanol (Ob-E). 10% ethanol consumption model was used for 6 weeks. Basal glycemia, insulinemia and a glucose overload test were evaluated at the end of the study. An object recognition test was used to assess short- and long-term memory. The antioxidant enzyme glutathione peroxidase (GPX) activity and the lipid peroxidation product, malondialdehyde (MDA) were analyzed in mice cortex samples.

Results: No significant differences were found among groups in long- and short-term memory. No significant differences between C and E group were found in the basal glycemia and the glucose overload test. However, the Ob group presented a significant increase in both parameters when compared to the C and E groups. These values were significantly decreased in the Ob-E group when compared to Ob group. Insulinemia was increase in both, Ob and Ob-E when compared to C and E groups. The activity of GPX was burst in the E, Ob and Ob-E groups when compared to C animals. No significant differences were observed in MDA concentration.

Conclusion: Four weeks of ethanol administration do not induce significant behavioral or biochemical brain impairments in Ob mice, although it was able to modulate glucose metabolism.

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