IN VITRO STIMULATION OF OXIDATIVE STRESS BY CLASSICAL GALACTOSEMIA IN CEREBRUM OF RATS


INTRODUCTION AND OBJECTIVES: Classical galactosemia is an autosomal recessive inborn error of metabolism caused by deficiency of galactose-1-phosphate uridylytransferase activity and subsequent accumulation of galactose-1-phosphate and galactose. Affected patients present central nervous system dysfunction, including cognitive difficulties, psychiatric symptoms, and speech and motor problems, whose pathophysiology is still obscure. We investigated the in vitro effect of different concentrations of galactose on thiobarbituric acid-reactive substances (TBA-RS), total sulfhydryl content, protein carbonyl content and on the activities of antioxidant enzymes [catalase (CAT), glutathione peroxidase (GSH-Px) and superoxide dismutase (SOD)] in the cerebral cortex and hippocampus of 30-day-old male Wistar rats.

MATERIALS AND METHODS: For in vitro experiments, galactose was added to the assay at 0.1, 3.0, 5.0 and 10.0 mM final concentrations. TBA-RS, total sulfhydryl content and protein carbonyl content were determined by the method of Esterbauer and Cheeseman (1990), Aksenov and Markesbery (2001) and Stadtman and Levine (2003), respectively. The activities of CAT, SOD and GSH-Px by the method of Aebi (1984), Wendel (1981) and Marklund (1985), respectively. Data were analyzed by ANOVA, followed by the Duncan multiple range test, when the F-test was significant.

RESULTS AND CONCLUSION: Results showed that galactose increased TBA-RS (3mM:18%; 5mM:40%; 10mM:46%, p<0.001), protein carbonyl content (5mM:20%; 10mM:31%, p<0.001), and CAT activity (10mM:17%, p<0.05), but did not alter total sulfhydryl content, GSH-Px and SOD activities in the cerebral cortex. Galactose also increased TBA-RS (3mM:99%; 5mM:198%; 10mM:360%, p<0.001) and CAT activity (5mM:23%; 10mM:31%, p<0.01), decreased GSH-Px activity (10mM:28.8%, p<0.001), but did not alter SOD activity, protein carbonyl content and total sulfhydryl content in the hippocampus.
of rats. Findings suggest that classical galactosemia alters antioxidant defenses, causes damage to proteins and induces lipid peroxidation in the cerebrum of rats.

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**Key Words:** classical galactosemia, oxidative estress, cerebrum.