ACUTE ADMINISTRATION OF GALACTOSE INDUCES OXIDATIVE STRESS IN BRAIN AND LIVER OF RATS


Introduction and objectives: Galactosemia is an inborn error of galactose metabolism, leading to the accumulation of this carbohydrate in tissues and fluids of patients. Galactosemic patients present mainly brain and liver damage. The main of this study was available oxidative stress parameters in brain structures and liver of young rats. Materials and methods: For this purpose, 30-day-old males Wistar rats were divided into two groups: galactose group, that received a single injection of this carbohydrate (5 μmol/g), and control group, that received saline 0.9% in the same conditions. One, twelve or twenty-four hours after the administration, animals were euthanized by decapitation and cerebral cortex, cerebellum, and liver were cleaned and isolated. It was evaluated TBA-RS levels, nitrates/nitrites concentration, carbonyl content and superoxide dismutase (SOD) and catalase activities in these tissues. Results and conclusions: It was observed increased TBA-RS levels, carbonyl content, and nitrates/nitrites concentrations in cerebral cortex one hour after the administration. Twelve hours after galactose administration, TBA-RS levels were found to be increased in cerebral cortex, cerebellum and liver, carbonyl content was increased in cerebellum, nitrate/nitrite concentrations were also increased in cerebral cortex and cerebellum. Furthermore, catalase and SOD activity were decreased in all structures. In addition, TBA-RS levels were increased in cerebral cortex, cerebellum and striatum of animals that received galactose, as well as nitrate/nitrite concentrations were increased in cerebral cortex and liver and carbonyl content was increased in cerebellum and liver twenty-four hour after the administrations. Moreover, SOD and catalase activities were inhibited in cerebral cortex and liver. Taken together, the present results suggest that acute galactose administration induced oxidative stress and this effect increased with time of exposition.

Key Words: Galactose, oxidative stress, rats.

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