THE METFORMIN ROLE IN CONTROL OF OXIDATIVE STRESS IN DIABETIC RATS MUSCLE

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Introduction and Objectives: Metformin can acts in muscle inhibiting the complex I of the electron transport chain and activating a signaling cascade for glut-4 translocation responsible for increasing glucose uptake. Our hypothesis is that this inhibition can decreases mitochondrial reactive oxigen species (ROS), minimizing cell damage caused by oxidative stress. Thus, the aim of this study was to evaluate the activity and expression of oxidative stress enzymes, total antioxidant status and lipid peroxidation in gastrocnemius muscle of rat with induced diabetic Mellitus (DM) and treated with metformin. Materials and Methods: The DM was induced in Wistar rats by intraperitoneal injection of streptozotocin (45 mg/kg). After 10 days, through the blood glycemic level the diabetized animals were divided into four groups: untreated, treated with insulin, insulin plus metformin, and metformin. A non-diabetic group was used as control. After glycaemia evaluation in the seventh day of treatment, animals were euthanized by sodium thiopental overdose and the gastrocnemius muscle collected and stored at -80C. Muscles homogenates were tested for the following parameters: superoxide dismutase (SOD) and catalase (activity and immunoblotting), glutathione peroxidase, glutathione reductase and glucose-6-phosphate dehydrogenase (activity), reduced glutathione, lipid peroxidation and total antioxidant capacity were measured. Results and conclusions: The groups treated with metformin showed a decrease in activity and expression of SOD and catalase enzymes, as well as a decrease in total antioxidant capacity and activity of G6PDH, when compared to the untreated group. This decrease appears to be associated with the complex I inhibition and reduction of ROS production, resulting in a lower lipid peroxidation. Therefore, this study suggest that metformin may is involved in the regulation of the oxidative stress in muscle cells decreasing lipid peroxidation which could improve glucose uptake.

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Key words: Hyperglycemia, lipid peroxidation, reactive oxygen species.