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EFFECT OF A COMBINED PREPARATION ON THE ANTIOXIDANT SYSTEM OF RAT LIVER MITOCHONDRIA IN STREPTOZOTOCIN-INDUCED DIABETES

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Abstract. Diabetes mellitus is a chronic metabolic disorder characterized by persistent hyperglycemia and increased oxidative stress, which plays a key role in the development of diabetic complications. Streptozotocin (STZ)-induced diabetes is widely used as an experimental model to investigate metabolic and mitochondrial dysfunctions associated with diabetes. In this study, the effect of a combined preparation on the antioxidant defense system of rat liver mitochondria was evaluated under STZ-induced diabetic conditions. The activity of key antioxidant enzymes, including superoxide dismutase (SOD), catalase, and glutathione peroxidase (GPx), was assessed using spectrophotometric methods.

The results demonstrated a significant decrease in mitochondrial antioxidant enzyme activities in diabetic rats, indicating pronounced oxidative stress and mitochondrial dysfunction. Treatment with the combined preparation significantly improved SOD, catalase, and GPx activities, suggesting restoration of the antioxidant defense system and reduction of oxidative damage. In healthy animals, the combined preparation did not induce significant biochemical changes, indicating its safety under physiological conditions.

These findings suggest that the combined preparation may have a protective effect on mitochondrial function by enhancing antioxidant defense mechanisms in diabetic conditions.

Keywords: diabetes mellitus; streptozotocin; oxidative stress; mitochondria; liver; antioxidant enzymes; superoxide dismutase (SOD); catalase; glutathione peroxidase (GPx); combined preparation.

Diabetes mellitus is one of the most widespread metabolic disorders worldwide, in which oxidative stress and mitochondrial dysfunction play a crucial role in its pathogenesis [1]. Streptozotocin (STZ) exerts a selective toxic effect on pancreatic β-cells, leading to insulin deficiency and persistent hyperglycemia [2]. Under hyperglycemic conditions, excessive production of reactive oxygen species (ROS) occurs, resulting in damage to cellular membranes, proteins, and DNA structures [3]. Mitochondria are one of the main sources of ROS generation, and their impairment leads to disruption of cellular energy metabolism and suppression of the antioxidant defense system [4]. The liver plays a central role in metabolic processes, and its mitochondria are particularly sensitive to oxidative stress.

The aim of this study was to evaluate the effect of a combined preparation on the activity of antioxidant enzymes, namely superoxide dismutase (SOD), catalase, and glutathione peroxidase (GPx), in rat liver mitochondria under streptozotocin-induced diabetic conditions.

The experiment was conducted on white laboratory rats weighing 180–220 g, which were divided into four groups: control group, diabetic group, diabetic group treated with the combined preparation, and healthy group receiving the combined preparation. Experimental diabetes was induced by a single intraperitoneal injection of streptozotocin at a dose of 60 mg/kg [2]. The development of diabetes was confirmed by elevated blood glucose levels.

Liver mitochondria were isolated using differential centrifugation. The activity of antioxidant enzymes in the mitochondrial fraction was determined using spectrophotometric methods. SOD activity was measured based on inhibition of superoxide radicals, catalase activity was assessed by the rate of hydrogen peroxide decomposition, and GPx activity was determined

based on NADPH oxidation rate. Statistical analysis was performed using Student's t-test, and differences were considered significant at $p < 0.05$.

The results showed that in the diabetic group, the antioxidant defense system of liver mitochondria was significantly impaired. Specifically, SOD activity was reduced, leading to decreased neutralization of superoxide radicals. Catalase activity was also decreased, resulting in impaired decomposition of hydrogen peroxide. A reduction in GPx activity contributed to increased lipid peroxidation and further mitochondrial membrane damage, confirming severe oxidative stress in diabetic conditions [1,3].

In the group treated with the combined preparation, a significant improvement in antioxidant enzyme activities was observed. SOD activity increased, enhancing the elimination of superoxide radicals, while catalase activity improved hydrogen peroxide detoxification. GPx activity also increased, reducing lipid peroxidation and improving membrane stability. In healthy animals, the combined preparation did not induce significant changes, indicating its safety under physiological conditions.

These findings suggest that streptozotocin-induced diabetes is associated with mitochondrial dysfunction and oxidative stress due to impaired antioxidant defense mechanisms [4]. The combined preparation enhances mitochondrial antioxidant capacity, reduces ROS production, and improves cellular energy balance. Similar protective effects of biologically active compounds have been reported in experimental models, where combined bioactive agents improved physiological and biochemical parameters, supporting their role in reducing oxidative damage [5].

In conclusion, the combined preparation significantly improves the antioxidant system of rat liver mitochondria in streptozotocin-induced diabetes, reduces oxidative stress, and partially restores mitochondrial function. These results indicate its potential as a therapeutic agent for mitigating oxidative damage associated with diabetes.

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