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EFFECTS OF BIO-NORMALIZER ON BIOCHEMICAL AND STRUCTURAL PARAMETERS IN RATS WITH STREPTOZOTOCIN-INDUCED DIABETES

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This study was designed to further evaluate the cellular and molecular mechanisms of beneficial clinical effects of Bio-Normalizer on diabetes mellitus. For the purpose, the experimental model of streptozotocin-induced diabetes in rats was used. The male Wister rats were injected with 45 μ g of streptozotocin (STZ) intravenously. Starting from the third day after injection, the experimental group of animals (n=15) was given BN (100 mg/kg of weight) for one week, and the control group (n=12) was given the pure glucose (100 mg/kg weight for one week). The animals of both groups were sacrificed under phenobarbital narcosis at the third and tenth days of experimental procedure. The following parameters were analyzed: glucose level in the blood; nitric oxide and superoxide production by peritoneal macrophages and circulating granulocytes; lipid peroxidation and glutathione content in red blood cells (RBC); lipid peroxidation and structural abnormalities in pancreas and brain. We found a three-fold increased glucose level in STZ-treated rats at the third day after STZ injection. The microscopical examination revealed that STZ injection led to the development of acute inflammatory reaction in endocrine part of pancreas, reversible damage of islet β-cells, and increased contraction of islet and brain capillaries. There were increased release of NO from macrophages (2- and 1.5-fold increase at the third and tenth days, respectively) and highly suppressed NO production by circulating leukocytes. The peritoneal macrophages of diabetic rats produced slightly higher than normal levels of superoxide spontaneously as well as being activated by phorbol ester (PMA) as was revealed by cytochrome c reduction. Activation by PMA resulted in the increased production of O_2^- by diabetic leukocytes. Significantly suppressed lipid peroxidation in both liver and pancreas and increased lipid peroxidation in RBC accompanied the development of diabetes. There was a simultaneous GSH depletion in RBC. BN but not glucose administration decreased substantially the glucose level in the blood of diabetic rats, prevented vascular abnormalities in islets and brain, restored the normal NO and superoxide production by peritoneal macrophages and blood granulocytes, and normalized the GSH level in red blood cells. At the same time, BN affected the intensity of lipid peroxidation neither in liver nor in pancreas nor in red blood cells.