METABOLIC DISORDERS UNDER HIGH FRUCTOSE DIET AND DEXAMETHASONE MODELS OF INSULIN RESISTANCE

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Introduction. Type 2 diabetes is a serious public health problem due to its wide spread, a significant number of macro- and microvascular complications that lead to increased disability and mortality. It is known that the basis of type 2 diabetes is the disturbance of insulin homeostasis: insulin resistance of peripheral tissues and dysfunction of beta cells of the pancreas. To develop antidiabetic drugs, knowledge of the mechanisms of insulin resistance development is necessary. Therefore, the modeling of insulin resistance is important.

Aim. The aim of this work was to investigate some indicators of glucose-insulin homeostasis and prooxidant-antioxidant status in rats blood serum under two models of insulin resistance.

Materials and methods. Insulin resistance was modeled by keeping animals on high fructose diet (HFD) for 6 weeks or intraperitoneal administration of dexamethasone (Dex) for 5 weeks. Glucose and immunoreactive insulin (IRI) content were determined by glucose oxidase and radioimmunoassay using standard kits. Insulin resistance index was calculated using the algorithm HOMA. Determination of 2-thiobarbituric acid-reactive products (TBA-RP) and reduced glutathione (GSH) contents were carried out by spectrophotometric methods. Statistical analysis of the data was performed using STATISTICA software package (StatSoft Inc., USA, version 6.0). The significance of differences between groups was assessed by non-parametric Mann-Whitney test. Significance was assigned at $p<0.05$.

Results and discussion. Both researched models were accompanied by disturbances of glucose-insulin homeostasis and prooxidant-antioxidant balance. It was shown that under HFD the increase in glucose level was 160% compared with the intact group, insulin level − 140%, HOMA index − 170%. Under Dex model glucose content increased 1.9 times in comparison with the intact group, insulin level − 1.5 times, HOMA index – 1.8 times. Regarding the disturbances of prooxidant-antioxidant balance the decline of GSH was the same in both models but the growth of TBA-RP was stronger in dexamethasone model (almost 2 times higher than under HFD).

Conclusions. Thus HFD and Dex model caused approximately the same glucose-insulin homeostasis disturbances, but different changes in prooxidant-antioxidant status due to different mechanisms of fructose excess and dexamethasone action.