

THE INFLUENCE OF QUERCETIN ON BIOCHEMICAL CHANGES IN RAT LIVER TISSUE ON THE BACKGROUND OF CENTRAL DEPRIVATION OF LUTEINIZING HORMONE SYNTHESIS

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Background and Aim. Testosterone synthesis inhibition can cause various effects on liver tissue, including changes in macrophageal activity and in diversity of their subpopulations. The aim of our study is to find out those changes by investigating arginase and NOsynthases activities.

Material and Methods. The experiment was conducted on 30 adult male rats. Animals were divided into 3 groups: I control (10); II experimental (10) - were injected with triptorelin acetate in the dosage of 0.3 mg/kg of body weight, III experimental (10) - received triptorelin acetate in the same dosage and quercetin 100 mg/kg of body weight 3 times a week. The experiment lasted for 365 days. The nonparametric Mann - Whitney test was used to determine the significant statistical differences between groups. The difference was considered statistically significant at $p < 0.05$.

Results. Arginase, iNOS and cNOS levels were monitored in all groups of animals. Activity of the iNO synthase in the II group increased by 9.1 %, while the activity of the constitutive isoforms did not change significantly. Arginase activity was reduced by 33.8%. Against the background of 365 days of central deprivation of testosterone synthesis the introduction of quercetin led to a decrease in the activity of the iNOS by 61.7 %, while the activity of cNOS decreased by 36.7%. Usage of quercetin increased the activity of arginase by 3.6% in comparison with the experimental group.

Conclusions. INOS activity could be used as a marker of macrophage polarization by the M1 phenotype, while arginase activity was a clear marker of the M2 phenotype. With prolonged central deprivation of luteinizing hormone synthesis by triptorelin, the polarization of liver macrophages was shifted towards the predominance of the M1 phenotype, as the iNOS / ARG ratio increased to 0.91 versus 0.65 in the control group.

Keywords: liver, macrophages, testosterone, triptorelin acetate