

INVESTIGATION OF HYPERLEPTINEMIA CORRECTION MECHANISMS AFFECTED BY HYDROXYCITRIC ACID UNDER HIGH CALORIE DIET IN RATS

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Introduction. It is well known that the metabolic syndrome (MS) pathogenesis is closely associated with obesity, insulin resistance (IR) and hyperphagia caused by an imbalance of the eating behavior humoral regulators, in particular leptin. Leptin is the peptide hormone possessing anorexigenic activity. It is synthesized in adipocytes in proportion to the adipose tissue value, which leads to the hyperleptinemia development under the obesity conditions.

Aim. The aim of our study was to investigate the impact of hydroxycitric acid on leptin level changes which are developed under hypercaloric diet in rats.

Materials and methods. IR was modeled in Wistar rats weighing 160-200 g, by keeping on high-fructose diet and long-lasting dexamethasone injections. The test substance – hydroxycitric acid was injected intragastrically in a dose of 0.1g per 100 g of body weight. The animals were tested for glucose, immunoreactive insulin (IRI) and leptin levels in blood serum. For statistical data processing used program STATISTICA.

Result and discussion. Keeping rats on a high-fructose diet with dexamethasone injections led to the glucose and immunoreactive insulin (IRI) levels significant increase in the rats' serum (1.20 and 1.11 times, respectively), which indicates the IR progression. The leptin concentration in experimental animal's blood was also significantly increased by 1.04 times, but the expected depression of appetite did not occur, probably due to the leptin resistance development. According to the literature, insulin and leptin have a crossover mechanism of intracellular biosignaling, the disruption of which can be caused by the same factor. Under IR conditions, there is an intense release of free fatty acids, which are lipotoxic and this is a significant factor in the development of resistance to leptin.

Conclusions. Intragastric administration of hydroxycitric acid resulted in lower levels of glucose and IRI, which did not significantly differ from those of intact control. The blood concentration of leptin was within physiological norms. Thus, it can be assumed that analyte increases the cells sensitivity to the insulin and leptin action, probably through it's ability to reduce the intensity of free radical oxidation within cells and enhance signal transduction of these hormones' receptors, which, however, requires further research.