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INSULIN-PRODUCING SYSTEM AT DIABETES MELLITUS TYPE 2*

Keyworlds: β-cells, insulin+ cells, diabetes mellitus type 2.

Diabetes mellitus (DM) is a chronical disabling disease, leads to the development of complications. The pathogenesis of T2D is based on the peripheral tissue resistance to insulin and reduced insulin secretion. The synthesis of insulin is carried out by β-cells in islets of Langerhans. During development of T2D due to permanent hyperglycemia secondary dysfunction of β-cells take place, and there is a main reason for the decrease of blood insulin level. Currently, proinsulin/insulin+ cells are founded not only in the pancreas, but also in various body tissues, including exocrine part of the pancreas and liver [1, 2].

Materials and methods. Experiment was conducted on animals in accordance with the principles, manifested by the European Parliament and the Council (Directive 2010/63/EU) and was approved by the Ethical Committee of the Institute of Immunology and Physiology of the Ural Branch of the Russian Academy of Science.

20 Wistar males, weighting 303.0±25.3 g, were divided into 2 groups: intact group (10 rats) and T2D group (10 rats). T2D was made by intraperitoneal injections of streptozotocin with preventive injections of nicotinamide [3]. Animals were overdosed by ether after 30 days of experiment. Concentration of glucose, HbA_{1c} and insulin were detected in blood. Morphometrical and immunohistochemical analyses of pancreas and liver was made.

Results. Damage of islets of Langerhans at T2D is manifested in a decrease in the number of islets and β-cells in them, as well as a reduce in functional activity of β-cells. In healthy animals, insulin+ cells are detected in liver and in exocrine part of the pancreas in small quantity. At T2D number of insulin+ cells in pancreas and optical density of their cytoplasm, which may characterize its functional activity, practically do not change. At the same time, significant increase in the number of insulin+ cells in liver is noticed in T2D. Number of PDX1+ cells do not change neither in liver, no in

exocrine part of the pancreas. Revealed lower optical density of cytoplasm in insulin+ cells in liver and exocrine part of the pancreas, compared to β-cells, indicates relatively low content of insulin in these cells and shows its lower functional activity.

Conclusion. In T2D deficiency of insulin can be compensated for by the formation of new cells, producing insulin. Reprogramming of hepatocytes and cells of exocrine part of the pancreas into insulin+ cells is a potentially approach to generate new insulin-producing cells. An increase both the number of insulin+ hepatocytes and quantity in them of a transcription factor PDX1, which regulates expression of insulin gene, is a perspective way in in the development of new methods and approaches in the treatment of diabetes mellitus.

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ISOFLAVONES AS CANCER SENOTHERAPEUTICS: A FUTURE VISION

Keywords: Isoflavones, Cancer senescence, Senolysis.

Isoflavones are a type of polyphenol found in soybeans, chickpeas, fava beans, pistachios, peanuts, nuts, and other fruits [1]. Isoflavones include daidzin, genistin, biochanin A, and formononetin [2]. Isoflavones can inhibit growth of breast, uterine, and prostatic cancers [3–5].