



ABOUT MODELS OF EXPERIMENTAL DEVELOPMENT OF DIABETES 2

Kosimova Dilnoza Sayotovna

Assistant, Department of Pathophysiology, Bukhara State Medical Institute named after A.I. Abu Ali Ibn Sino, Bukhara, Republic of Uzbekistan.

Abstract

The paper analyzes the development models of type 2 diabetes mellitus (DM2). Rodents are best suited for this (mainly mice, including outbred lines), and rats, which have become more widely used recently. As for models of metabolic syndrome and obesity, diet-induced models are also closest in etiology and development mechanisms to DM2 in humans, among which high-fat diets (HFAs) enriched with sucrose or fructose are the most effective.

Keywords: diabetes mellitus, experiment, animals, induction, inbred, outbred.

Currently, DM has been studied to a sufficient extent, there is a large arsenal of drugs and modern treatment regimens for this pathology, but, unfortunately, the disease continues to progress. This is especially noticeable in developed countries [1, 2]. According to WHO, the number of patients diagnosed with diabetes has reached approximately 160 million, by 2025 the number of patients with this diagnosis is predicted to double, and this increase is due to an increase in patients with type 2 diabetes. The main causes of deterioration in the quality of life, disability and mortality in patients with DM are chronic complications caused by hyperglycemia (diabetic nephropathy, diabetic retinopathy, diabetic foot, diabetic neuropathy, atherosclerosis, etc.) [2]. To model DM2, mice and rats are mainly used, less often - guinea pigs, gerbils, etc. In the etiology of DM2 in humans, excess caloric nutrition and obesity are of primary importance. Diet (RD) has a significant impact on the development of DM2. For example, in mice of the outbred C57BL/6 line, the development of obesity and DM2 requires VFA (60% fat), and a faster and more pronounced effect is achieved when a large amount of sucrose is added to it. In Wistar rats, a high fructose diet (35–60% kcal from fructose) was used to induce insulin resistance. But in genetically modified lines of mice of the ApoE^{-/-}, LDLr^{-/-}, LDLr^{-/-}/ApoB100/100 lines, DM2 does not develop at all even on high-calorie RP [3, 4]. In experimental models of DM2, they are used as genetically modified (inbred) animals [(db/db mice, NZO mice (New Zealand Obese), TH mice (TALLYHO/Jng), Zucker Diabetic Fatty (ZDF) rats, etc.)], and outbred (C57BL/6 and BALB/c mice, Wistar





rats, etc.). Inbred mice (MI) have a developmental pathogenesis associated with obesity similar to that of humans. Therefore, they are ideal for studying the manifestations of T2DM in humans. Different lines of MI used to model T2DM differ significantly from each other. So, if the most striking characteristic of NZO mice is pronounced obesity (their body weight reaches 45 g by 3 months, and later can reach 100 g or more), then TH mice weigh 35 g by 3 months, while control lines of wild mice C57BL/6 - 27 g. Almost all researchers note that young mice under the age of 4 weeks, regardless of gender, are tolerant to the introduction of glucose, however, after puberty (earlier in males and a little later in females), they develop impaired tolerance to glucose.) 2020 - 14 . glucose load. Impaired glucose tolerance in animals is accompanied by hypersecretion of insulin (hyperinsulinemia) with subsequent development of insulin resistance: the average level of insulin in the blood plasma (not on an empty stomach) reaches 6 ± 1 ng/ml in females and 8 ± 1 ng/ml in male mice of the TH line , while in C57BL/6 mice it is only 0.4 ± 0.1 ng/ml in females and 0.6 ± 0.2 ng/ml in 6-week-old males [3, 4]. The rapid development of hyperglycemia, insulin resistance and the growth of glycated hemoglobin develops in mice against the background of only VZhR. Summarizing the above, we can conclude that mice, mainly outbred lines, are best suited for creating an experimental model of CD2. For the model of obesity in DM2, diet-induced models are the most effective, among which the most effective are VFAs, which are enriched with polysaccharides. For modeling DM, it is important to use mature animals.

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