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СПЕЦІАЛІЗОВАНИЙ НАУКОВО-ПРАКТИЧНИЙ ЖУРНАЛ

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ЗАСЛАВСКИЙ

Підвищена екскреція іонів натрію супроводжувалася високим кліренсом вказаного катіона і зниженим кліренсом безнатрієвої води практично у всі досліджувані проміжки доби порівняно з контролем, у періоди зростання фільтраційної фракції іонів натрію. На відміну від тварин із гіперфункцією шишкоподібної залози у щурів із пригніченою функцією залози реєстрували компенсаторну активацію проксимальної та пригнічення дистальної реабсорбції даного катіона.

В умовах гіперфункції шишкоподібної залози зазнавали змін параметри іонорегулювальної функції нирок. Ритм екскреції іонів натрію мав інверсний характер щодо контрольних хронограм, а його мезор удвічі перевищував контрольні показники. У всі періоди спостереження показник вірогідно вищий, ніж в інтактних тварин. Незважаючи на низьку фільтраційну фракцію іонів натрію, абсолютна й відносна реабсорбція катіона залишалася зниженою, порушувалися фазові структури ритмів відносно контролю. У результаті концентрація іонів натрію в сечі зростала, а в плазмі крові — знижувалась. Акрофаза проксимальної реабсорбції зміщувалася з 14:00 на 20:00, а дистальної — із 08:00 на 20:00.

Отже, моделювання гіперфункції шишкоподібної залози в умовах темряви призводило до перебудови хроноритмів функцій нирок і викликало дисинхроноз їхніх інтегральних показників більше, ніж за умов гіпофункції шишкоподібної залози, що пов'язано з надмірним освітленням.

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VIRUS INDUCED DIABETES IN ANIMALS (SHORT REVIEW)

Diabetes is a chronic disease, which occurs when the pancreas does not produce enough insulin, or when the body cannot effectively use the insulin it produces. Type 1 diabetes results from the progressive destruction of insulin-producing pancreatic beta cells. Although the etiology of type 1 diabetes is believed to have a major genetic component, studies on the risk of developing type 1 diabetes suggest that environmental factors, such as viruses, may be important etiological determinants (H.S. Jun, J.W. Yoon, 2001).

Main goal of the research. The existence of experimental animal models of this disease helps not only to understand the pathophysiological mechanism of its development, but also to find proper medical drugs for its treatment.

Basic theoretical items of information. More than 10 viruses have been reported to be associated with the development of type 1 diabetes-like syndromes in animals. They are coxsackie B viruses in mice and/or nonhuman primates, encephalomyocarditis (EMC1) virus in mice, mengo virus in mice, foot-and-mouth disease virus in pigs and/or cattle, retrovirus in mice, rubella virus in hamsters and rabbits, bovine viral diarrhea-mucosal disease virus in cattle, reovirus in mice, Kilham rat virus (KRV1) in rats, and cytomegalovirus in the Degu (Young-Hwa Chung, Hee Sook Jun, 2000). Among those viruses, the most clear and unequivocal evidence that a virus induces type 1 diabetes in animals comes from studies on EMC virus in mice. EMC virus is considered to be a primary agent that is selectively injurious to pancreatic beta cells, whereas KRV is considered to be a triggering agent of beta cell-specific autoimmunity without infection of beta cells (Travis R.Wolter, Danny Zipris, 2011).

KRV is a small DNA virus that can induce diabetes by provoking autoimmune responses against the beta cell, rather than by direct beta cell cytolysis, in diabetes-resistant-BioBreeding (DR-BB1) rats. These rats are derived from diabetes-prone progenitors, but they do not normally develop the disease (Zipris et al. 2003). When infected with KRV at 3 weeks of age, approximately 30 % of DR-BB rats develop autoimmune diabetes within 2 to 4 weeks, and a further 30 % show insulitis without diabetes. The incidence of diabetes can be increased to between 80 and 100 % if DR-BB rats are given injections of poly (I : C) along with KRV. It is not clear how KRV causes the destruction of beta cells in DR-BB rats without infection of these cells (Annie J. Kruger, Chaoxing Yang, 2010). Molecular mimicry, such as the existence of a common epitope between a KRV-specific peptide and a beta cell autoantigen, has been suggested as a mechanism for the initiation of beta cell-specific autoimmune diabetes. If molecular mimicry is involved in the initiation of beta cell-specific autoimmunity, then KRV antigen-specific T cells generated by KRV peptides might cross-react with beta cells and attack them, resulting in the development of insulitis and, subsequently, diabetes. To induce KRV antigen-specific T cells, recombinant vaccinia viruses (rVVs1) expressing KRV proteins were used, because previous work showed that rVVs were successful in inducing cell-mediated immune responses against a target protein. In addition, the wild-type strain of vaccinia virus does not induce insulitis or diabetes in DR-BB rats. When DR-BB rats were infected with these rVVs expressing the KRV peptides (VP1, VP2, or nonstructural proteins 1 or 2), it was found that each viral peptide was clearly expressed in the infected DR-BB rats, viral peptide-specific T cells were generated, and antibodies against the KRV peptides were also induced. However, none of the DR-BB rats developed insulitis or diabetes. This result suggests that molecular mimicry between KRV peptides and beta cell-specific autoantigens in DR-BB rats is unlikely to be a mechanism by which KRV induces beta cell-specific autoimmune diabetes (Elizabeth P. Blankenhorn, 2009).

In the rat, $CD4^+$ T cells can be divided into Th1-like $CD45RC^+CD4^+$ T cells, which express IL-2 and IFN-γ and play an important role in cell-mediated immune responses, and Th2-like $CD45RC^-CD4^+$ T cells, which express IL-4 and IL-10 and play an important part in humoral immune responses. It has been suggested that the immune balance between Th1- and Th2-type cells plays an important role in the maintenance of peripheral tolerance. The dominance of Th1 cells over Th2 cells is associated with the development of autoimmune type 1 diabetes, whereas the dominance of Th2 cells over Th1 cells is associated with the prevention of type 1 diabetes. It was previously found that KRV infection in DR-BB rats increased the expression of Th1-type cytokines in the splenocytes and pancreatic infiltrates. Therefore, it is possible that the proportions of Th1 and Th2 cells are altered during KRV infection in DR-BB rats (Todd J.A., Walker N.M., 2007).

Conclusions. Different experimental models of diabetes give an opportunity to find the new approaches for this disease treatment as well as to prevent complications connected with it.

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PROMOTING OLIGODENDROGENESIS AND MYELIN REPAIR USING THE MULTIPLE SCLEROSIS MEDICATION GLATIRAMER ACETATE

The formation of oligodendrocytes (oligodendrogenesis) and myelin is regulated by several neurotrophic factors. Strategies to increase the level of these trophic molecules may facilitate repair in demyelinating conditions, such as multiple sclerosis (MS). Because leukocytes are a source of neurotrophic factors, and as glatiramer acetate (GA) generates T helper 2 (Th2) lymphocytes that are not known to be harmful, we tested the hypothesis that GA regulates oligodendrogenesis and myelin formation. First, we generated GA-reactive Th2 cells and determined that they produced transcripts for neurotrophic factors, including insulin-like growth factor-1 (IGF-1). The conditioned medium from GA-reactive T cells elevated IGF-1 protein and promoted the formation of oligodendrocyte precursor cells (OPCs) from embryonic brain-derived forebrain cells in culture. We next subjected mice to lysolecithin-induced demyelination of the spinal cord. At 7 days after the insult, the number of OPCs in the demyelinated dorsal column was higher than that in uninjured controls, and was further increased by the daily s.c. injection with GA. Increased OPC generation by GA was associated temporally with the elevation of IGF-1 and brain-derived neurotrophic factor (BDNF) in the spinal cord. Einally, the resultant remyelination at 28 days was higher in mice treated with GA during the first 7 days of injury compared with vehicle controls. These results indicate that GA promotes oligodendrogenesis and remyelination through mechanisms that involve the elevation of growth factors conducive for repair.

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REPRODUCTIVE HEALTH OF ADOLESCENT GIRLS WITH ENDOCRINE DISORDERS

Pubertal uterine bleedings are a topical problem of modern adolescent gynecology. As a rule, in case of disturbances of the menstrual cycle in girls in the form of pubertal menorrhagias the hormonal background changes, the synthesis and secretion of the gonadotropic hormones is disturbed, the secretion of estradiol and progesterone changes. Thyroid hormones exert an immediate effect on the organs of the reproductive system, inhibiting the follicle-stimulating function and enhancing the luteinizing function of the hypophysis and in its turn, the sensitivity of the ovaries to the gonadotropic hormones and the endometrium to estrogens increases.

Aim and object of the research. We have studied the concentration of the sex and thyroid hormones of the serum in the blood of teen-age girls, suffering from pubertal menorrhagias against a background of the thyroid gland pathology.

Material and methods of the research. We have examined 70 adolescent girls with pubertal menorrhagias who were treated at the gynecological unit of Municipal Clinical Maternity Hospital № 1 (MCMH № 1) of Chernivtsy City and subdivided into two groups: group I (basic) — 30 teen-age girls with the diagnosis of pubertal menorrhagias with underlying concomitant pathology of the thyroid gland, group II (of comparison) — 40 teen-age girls with the diagnosis of pubertal menorrhagias, 27 apparently healthy teen-age girls (the control group).

All the subjects underwent a multimodality hormonal examination with an evaluation of the concentration of hormones in the blood serum by means of the immune enzyme analysis (IEA) method, using the immunoenzymatic microplate semiautomatic analyzer — Expert Plus Asus, (Biochrom Ltd, England) and anassay kit VECTOR Best (Russia). Statistical data processing was carried out by means of a package of computer programs Statistica 6,0. Just this very investigation of sex hormones as estradiol (E2), progesterone (P), follicle-stimulating hormone (FSH), luteinizing hormone (LH) and thyroid hormones — thyroxine (T_4), triiodothyronine (T_3) and thyrotropin–releasing hormone (TRH) was assumed as the basis for the purpose of studying their concentration in the blood serum in teen-age girls with pubertal menorrhagias with concomitant pathology of the thyroid gland.