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INFLUENCE OF STREPTOZOTOCIN-INDUCED DIABETES MELLITUS AND SOCIAL STRESS ON THE FUNCTIONAL STATE OF TH17-CELLS IN PERIPHERAL IMMUNE ORGANS OF WISTAR RATS

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Background: T helper type 17 (Th17-cells) are playing an important role in the development of inflammatory and autoimmune processes. These cells differ significantly by genotype, phenotype and physiology of other subpopulations of T helper cells. Th17 have the original receptors, transcription factors, cytokines produced by the set. The major feature of this subpopulation is to generate significant amounts of pro-inflammatory cytokines, therefore appearing to be critically involved in protection against infection caused by extracellular microorganisms, and in the pathogenesis of autoimmune diseases and allergy. The dynamic balance among subsets of T cells is important for the modulation of several steps of the immune response. Disturbances in this balance may cause a shift from normal immunologic physiology to the development of immune-mediated disorders.

Material and methods: The main transcription factor critical for the differentiation of Th17 is ROR γ t. It was spent identification of Th17-cells by using immunofluorescent test with using monoclonal antibodies to transcription factor ROR γ t in serial histological sections of spleen and Peyer's patches (PP) in Wistar rats with streptozotocin-induced diabetes mellitus and exposed to chronic social stress. It was studied the lymphoid follicles, marginal zone and periarterial lymphoid sheath (PALS) of spleen, and lymphoid follicles, subepithelial and interfollicular zone of PP.

Results: It was found that in experimental animals with STZ-induced diabetes mellitus and subjected to chronic social stress changes, which were observed in the number of ROR γ t-immunopositive cells and concentration in these ROR γ t in morphofunctional zones of spleen and PP. Thus, the development of STZ-induced diabetes mellitus duration of 28 days led to an increase in population density ROR γ t⁺-cells in lymphoid follicles, marginal zone and periarterial lymphoid spleen clutches compared with controls, and was accompanied by an increase in the concentration of ROR γ t in T-helper type 17.

Conclusions: The understanding of IL-17 regulatory pathways is necessary for a better comprehension of the pathophysiology of autoimmune diseases and shall contribute to the development of new opportunities for therapeutic intervention.

NEUROBIOLOGICAL ASPECTS OF DYSLEXIA

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Background. Reading and writing are the most important basic skills that a child needs to learn in school. They are closely bound to the knowledge, understanding and use of language and influence social and cultural life. In scientific and professional literature a variety of terms are used to describe this problem: *dyslexia*, *legasthenie*, *reading disorders* and *reading difficulties* that are used as synonyms.

Material and Methods. Theoretical method – study of literature and its analysis. The origins of the studying of reading disorders were in the end of XIX century and the beginning of XX century, when compulsory education was first introduced in Europe. While teaching children to read, teachers and physicians found that there were some students that found it difficult to grasp the content of the taught topic. The most common difficulties were associated with reading and writing disorders. The first to describe such disorders was a physician. English surgeon Pringle-Morgan was the first to coin the term *congenital word blindness* that was later defined by a Budapest-based neurologist P. Ranschburg as reading difficulties or disorders. M. Linder defined reading disorders as a special type of learning disorders found in children with a relatively highly developed intelligence for their age group, that presents difficulties to make use of a written word, including problems with reading and writing it. The aim of this presentation is analyses of primary causes of dyslexia. The 46 scientific literature sources about neurobiological and genetic aspects of dyslexia were analysed.

Results. Genetic investigations approve that reading disabilities can be caused by changes in 1, 2, 6, 15 and 16 chromosome (Grigorenko, 1997, Beckenbach, 2000 et al), by changes in brain function activity in “reading centers” (Galaburda, 1985, Wranke, Hemminger, 2002 et al), by perception disabilities (Mayringer, 1999, Schulte-Körne, 2003 et al) and also by sociopsychological factors.

Conclusions. It is concluded from literature studies and our investigations that dyslexia affect approximately 4 – 10 % school age children, significantly influence their capabilities in reaching academic standards and in future career development, can be caused by genetic reasons, changes in brain function activity, and sociopsychological reasons. Future investigations will be performed.

MOLECULAR PATHOGENESIS OF BREAST CANCER IN POST-GENOMICS ERA OF RESEARCH – THE BASIS FOR SPECIAL INFORMATION SYSTEM

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Background. Interest in post-genomic technologies is due to personalized oncology. In breast cancer (BC) its predictive direction is associated with necessity to find new approaches to personalized determination of propensity to the disease, use of molecular-based therapy via adequate understanding of individual features of the disease pathogenesis and organism properties, development of new targeted drugs. In this sense, functional proteome and metabolome, comparing with genome,