Conclusions. Osteomorphogenic proteins present important factors in the cartilage and bone genesis and open a real clinical perspective regarding the acceleration of post-traumatic bone regeneration.
Key words: BMP; cytokines; osteoblast

242. MOLECULAR ASPECTS IN PATHOGENESIS OF CANCEROGENESIS: REVIEW

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Introduction. According to the WHO, it is estimated that the annual number of cancer's cases will increase by about 70% in the next two years.
Aim of the study. To evaluate and systematize pathogenetic factors that contribute to cancerogenesis. Cancerogenesis is defined as the static process by which a normal cell acquires properties that allow the development of malignant phenotype (uncontrolled proliferation, local invasion and metastasis), or a cascade of events that lead to the transformation of a normal cell, often a clonogenic cell (stem cell) into cancer. Cancerogenesis is the multistage process in which mutations lead to the development of malignant phenotype, which is the result of multiple interactions between various exogenous and endogenous factors. Cancerogenesis proceeds through the accumulation of genetic and epigenetic changes that allow cells to break free from the tight network of controls that regulate the homeostatic balance between cell proliferation and cell death.
Conclusions. 1. In recent years, the development of genome-wide analytic methods has opened the possibility of identifying simultaneously multiple changes in gene expression as well as in genetic or epigenetic alterations affecting the genome of cancer cells. 2. The Mutator Phenotype can be caused by a number of mechanisms, such as defects in cell-cycle regulation, apoptosis, specific DNA repair pathways, or error-prone DNA polymerase, and it can have its source in inherited genetic defects that make subjects prone to specific cancers. 3. Mutations in cancer cells cover a wide range of structural alterations in DNA, including changes in chromosomes copy numbers or chromosomal alterations encompassing millions of base-pairs such as translocations, deletions or amplifications, as well as smaller changes in nucleotide sequences such as point mutations affecting a single nucleotide at a critical position of a cancer-related gene (Sugimura et al., 1992). These different kinds of alterations often co-exist within a single tumour. 4. TP53 mutations in plasma DNA have been reported in patients with cancers of the colon, pancreas, lung, and liver. 5. EGFR and HER2 are often altered in diverse human cancers, by amplification, point mutation, or both. Amplifications of EGFR have been detected in brain cancers and in a small proportion of a number of epithelial cancers such as squamous oral or esophageal cancer. Amplification and overexpression of HER2 are a frequent event in breast and ovarian cancer (Harari and Yarden, 2000).
Key words: cancerogenesis, review

243. PATHOGENETIC FACTORS INVOLVED IN THE PRODUCTION OF LATE COMPLICATIONS OF DIABETES

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Introduction. Diabet Melitus is a complex and heterogeneous sindrom caused by an innate or acquired disorder of insulin secretion or resistance of peripheral tissues to the insulin produced, it produces profound disturbances in carbohydrate, protein and mineral metabolism. All these leads to the appearance of cronic complications it has become one of the most common metabolic disease .In the world there are 449,3 mln people with diabetes. In the Republic of Moldova there are 90.000 people with diabetes. Studying the role of the: hyperglicaemia final glycation products, inflammation, oxidative stress, the rennin angiotensin aldosterone system, could serve as information markers involved in the production of late complications of diabet mellitus.
Aim of the study. In these review, I will describe the pathogenetic factors involved in the production of late complications of diabet mellitus.
Materials and methods. The material was searched using the PubMed engine along with the psycarticles database. The following keywords joined the search for titles/ abstracts via PubMed: Pathogenesis of late complications of diabet mellitus.
Results. Hyperglycaemia is the basis for chronic lesions in diabet mellitus. In hyperglycemia the body is trying to metabolize glucose in an accelerated way, to decrease the amount of glucose in the same time is formed a series of intermediate toxic products which lead to training advanced glycation end products(AGE). The most important pathological effect of AGE is that many cells have surface receptors for AGE called (RAGE), by binding AGE to receptors(RAGE) are stimulated the inflammation and oxidative stress. The inflammation is involved, the source which produce the inflammation is the adipos tissue trough adipocytes and macrophages which releases pro-inflamatory mediators. TNF alfa, IL-6,IL-1,IL-8, gamma interferon increase the inflammation and aggravate insulin resistance also induces the apoptosis and disfunction of beta pancreatic cells. The oxidative stress means excessive formation of free radicals: reactive oxygen molecules(ROS) and reactive nitrogen molecules(RNS) they alter the structure of proteins, lipids and nucleic acids all leading to vascular damage. ANG II it's an vasoconstrictor factor involved in vascular remodeling and atherosclerosis. Aldosterone has the effect of stimulating proliferation of fibroblasts and stimulating the inflammation.
Conclusions. The control of pathogenetic factors will allow development of pathogenetic therapy of Diabetes and only then we will be able to stopped the occurrence of late complications of diabetes.
Key words: late complications, inflammation

244. PATHOGENETIC FACTORS INVOLVED IN METABOLIC INFLAMMATION INDUCED BY OBESITY

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Introduction. Obesity is a complex problem, multifactorial disorder, characterized by the increase in body weight due to adipose tissue. In recent decades, it has become one of the most common nutritional diseases in the world, being considered the 21st century disease. Its increasing incidence requires attention due to the associated mortality and morbidity potential. Obesity is the most important risk factor for atherosclerosis, hypertension, dyslipidemia, diabetes, being a constituent part of the metabolic syndrome. Studying the role of ghrelin, leptin, adiponectin, TNF could serve as information markers of obesity and other metabolic disorders, organ and obesity-related disorders.
Aim of the study. In this review, I will evaluate and systematize the pathogenetic factors originating from the adipose tissue involved in the metabolic disorder.