Materials and methods. The principle of this study execution consists on exploring of different new and suitable methods of treatment and life quality improvement in patients with CKD. Meta-analysis of diverse studies is the key tool for disclosure of optimal strategies for kidney function improvement in the ends stage of diseases.
Results. 1. Regenerative medicine holds the potential to fully heal damaged tissues and organs, offering solutions and hope for people who have conditions that today are beyond repair. 2. A new drug therapy that could potentially control protein leakage from the kidneys. 3. A new approach to prevent the kidney cell irreversible injury: AC1903. 4. Scientists have successfully produced human kidney tissue within a living organism, which is able to produce urine.
Conclusions. The outcome of the study reveals a significant "win" on kidney disease. In Moldova patients reach late stages of chronic renal disease, because a lot them are addressing too late to medical care services. That's why new treatment methods have a great importance to save lives of patients with CKD and to improve their life quality as well.
Key words: Chronic kidney disease (CKD); chronic renal failure; therapeutic methods

241. THE ROLE OF OSTEOMORPHOGENETIC PROTEINS IN POSTTRAUMATIC REGENERATION OF THE BONES

Author: **Iurie Stoica**
Scientific adviser: Lutan Vasile, MD, PhD, Professor, Department of pathophysiology and clinical pathophysiology
Nicolae Testemitanu State University of Medicine and Pharmacy of the Republic of Moldova

Introduction. Certain cells participate in the process of physiological and reparative (posttraumatic) regeneration: osteoprogenic cells - osteogenic stem cell, presteoblasts, osteoblasts. Likewise osteoclasts are involved in the remodeling process of the bones. The activity and interaction of osteogenic cells in the bone regeneration process is controlled by numerous growth factors named as bone morphogenetic proteins (BMP). It has now been demonstrated that these morphogenetic signals organize the structure of all organs, including bones, and the disruption of their functioning leads to different pathologies. Thus, discordance in the activity of these protein-signals may be the cause of tumor growth (e.g., rectal, esophageal cancer). BMPs play a decisive role in the regeneration and organization of the bone.
Aim of the study. Bibliographic analysis of the role of stimulatory factors in osteogenesis.
Materials and methods. Currently PMs obtained by the use of gene engineering method are used in regenerative medicine, including stimulation of post-traumatic bone regeneration. The method consists of inoculating BMP into the bone implant, from where they penetrate to the fracture site during several weeks. Clinical use of the osteomorphogenic protein products is now accepted for the acceleration of fractured bones fragments - Infuse BMP-2 (Medtronic) in dentistry and OP-1 BMP-7 (Stryker Biotech) in long bone fractures. BMPs are used in the recovery of intervertebral cartilage. The broad implementation of BMP is only retained by the expensive costs of the therapy – the standard cure can cost 6000-10,000 $.
Results. Nowadays about 20 osteomorphogenic proteins are known, and the following are directly involved in osteogenesis: BMP1 - metalloprotease - acts on procollagen I, II, II and participates in the cartilage genesis; BMP2 - plays a role in differentiation of osteoblasts and genesis of cartilages and bones; BMP3 - stimulates bone formation; BMP4 - regulates the formation of teeth; BMP5 - has a role in cartilage development; BMP6 - controls homeostasis by regulating hepcidin synthesis; BMP7 - plays a main role in the differentiation of osteoblasts and stimulates SMAD1 formation; BMP8a - participates in the development of the cartilages and bones.

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

Author: **Daniela Botnaru**
Scientific adviser: Stela Todiras, MD, PhD, Associate professor, Department of pathophysiology and clinical pathophysiology
Nicolae Testemitanu State University of Medicine and Pharmacy of the Republic of Moldova

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

Author: **Mihaela Lupascu**
Scientific adviser: Todiras Stela, MD, PhD, Associate professor, Department of Pathophysiology and clinical pathophysiology
Nicolae Testemitanu State University of Medicine and Pharmacy of the Republic of Moldova