

**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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