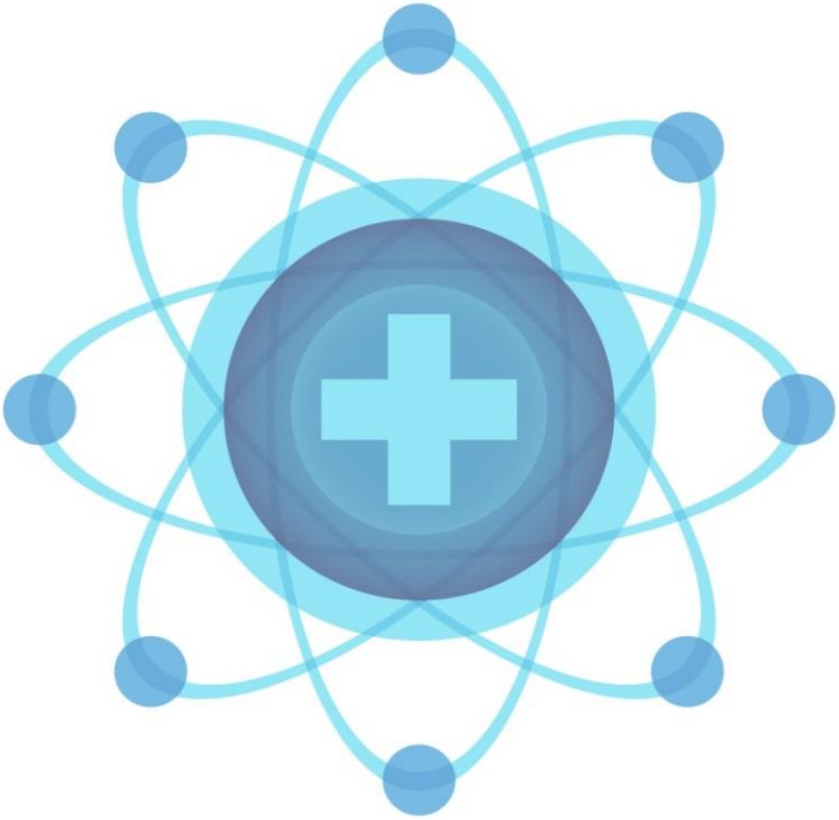

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EFFECTS OF THE METABOLIC SYNDROME ON THE LEVEL OF PRO-INFLAMMATORY CYTOKINES (IL-1β AND IL-6) IN PATIENTS WITH OSTEOARTHRITIS

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Abstract: This article summarizes the current understanding of the role of cytokines in the pathogenesis of osteoarthritis (OA). An imbalance between pro-inflammatory and anti-inflammatory cytokines in the joint tissues contributes to the development of inflammation and cartilage damage, which leads to progressive joint degeneration. This research studied the role of pro-inflammatory cytokines in OA and the impact on the course of disease against the background of metabolic syndrome.

Keywords: osteoarthritis, pro-inflammatory cytokines, interleukin-1β, interleukin-6, metabolic syndrome.

Introduction. Osteoarthritis (OA) is a heterogeneous morphological disease with various etiological factors through similar biological, morphological, and clinical presentations and outcomes. Simultaneously, the developing process damages cartilage and all joint tissues: synovial membrane, subchondral bone, bone capsule, ligamentous and periarticular muscles. Pro-inflammatory mediators express the synovial membrane, cartilage, and subchondral bone, even without classic signs of inflammation. [1,2].

Primarily OA manifests with inflammation, pain (the inflammatory process is one of the main mechanisms of pain formation), and a significant decrease in the quality of life (QoL) [3].

In this disease, cytokines play an essential role in determining the inflammatory process [4]. Last scientific data, OA has been named an autoimmune chronic inflammatory disease with the progressive development of synovial disorders, and cytokines imbalance is the basis of pathogenesis [5].

Cytokines are specific proteins, and the immune system uses them to exchange information among various immune cells and coordinate actions [6]. Interleukin-1β (IL-1 β) and interleukin-6 (IL-6) are the most significant importance among the many representatives of this group. The study of the cytokines levels that regulate the development, physiological functions and protective reactions of the body provides information on the functional activity of cells, the stage of the inflammatory process and its severity [7].

IL-1 includes two different cytokines called IL-1alpha and IL-1beta, derived from two genes. Different immune cell types produce IL-1alpha and IL-1beta after being stimulated by bacterial products, cytokines and immune complexes. Monocytes/macrophages are the primary sources of IL-1beta. IL-1 is present in the synovial tissue and fluids of OA patients. Several in vitro studies have shown that IL-1 stimulates the production of mediators such as prostaglandin E(2), nitric oxide, cytokines, chemokines, and adhesion molecules involved in joint inflammation. In addition, IL-1 stimulates the synthesis and activity of matrix metalloproteinase and other enzymes involved in cartilage destruction in OA. It is multifunctional, stimulates,