THE PECULIARITIES OF ARTHROSIS ITSELF

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Annotation: The review article highlights the scientific works of foreign scientists on metabolic, immunological causes and pathological mechanisms of osteoarthritis, which is becoming younger and an urgent problem among arthrolgic diseases in the elderly, leading to early disability and increasing economic problems.

Keywords: Osteoarthritis, metabolic disorders, obesity, immunology, biomarkers, phenotype, leptin, adiponectin, IL-1, IL-6 .

Osteoarthritis (OA) affects 240 million people worldwide [1] and is one of the ten most disabling diseases in developed countries. The most common form of arthritis is osteoarthritis (OA).In the Global Burden of Disease 2010 study, hip and knee OA ranked11th in global disability (Cross et al., 2014). The prevalence of OA will increase in parallel with the increase in the number of people aged 60 years and older and the increase in obesity worldwide. Cohort studies have shown that after aging, obesity and metabolic disorders are the main risk factors for developing OA (Aspden et al., 2001; Felson et al.,1988).Conversely, OA is a risk factor for metabolic syndrome and cardiovascular disease, suggesting that effective treatment of OA may prevent or delay the development of a large number of comorbidities (Haugen et al., 2013; Prior et al.,2014) [2]. There is growing evidence that there are different phenotypes of OA, reflecting different mechanisms of pathology.In 2016, evidence from six potential clinical phenotypes was presented in

theliterature: those occurring with chronic pain; with inflammation;withmetabolic syndrome; bone and cartilagemetabolism;mechanical overload; and minimaljoint involvement[3]. These researchers subsequently examined the prevalence of these phenotypes in the OA Biomarker Consortium (OAI/FNIH) Foundation datasetof the National Institutes of Health [4]]. Another review focused on molecular/mechanicalendotypes, particularly those associated with inflammation (characterized, for example, by c-reactive protein and interleukin-6), bone (e.g., c-terminal collagen telopeptide I [CTX-I]), metabolic syndrome (e.g., adipokines, advanced glycation end products), and aging (markers of aging) [5]]. Recent efforts have been made to standardize the conduct and reporting of phenotypic studiesin OA [6], in which it has been noted that phenotypes are "subtypes of OA that have various underlying pathobiological and pain mechanisms and their structural and functional consequences" [7]. Metabolic syndrome is a term encompassing a complex of hormonal and metabolic disorders, a serious medical and social problem of our time. Due to its prevalence among the adult population, MC is called the "syndrome of the new era" [8]. For the first time in 1988. J.M.Riven formulated the concept of MC as "X-syndrome".Thus, in 1989, abdominal obesity was identified as the most important etiological factor in the formation of insulin resistance, and obesity, especially in the upper body, increased glucose tolerance in hypertension. Thus, this combination is most indicative for increasing mortality from cardiovascular diseases [9].Adipose tissue is a type of connective tissue made up of blood vessels, collagen fibers, fibroblasts, and an extensive network of the immune system surrounded by lipid cells called adipocytes [10]. According to the classification, adipose tissue is divided into two types: white and gray. They are characterized by different anatomical localization, structure and

functions. Adipose tissue produces many biologically active substances, among which an important role is played by adipokines and adipocytokines [11].More than 50 adipokines are known, including leptin, adiponectin, resistin, visfatin, etc., whichsecrete anti-inflammatory cytokines, such as tumor necrosis factor a (TNFa), interleukin 6 (IL6), inhibitor 1 of the plasminogen activator and others. Adipokines are biologically active substances produced by white adipose tissue and have a pleiotropic effect. They are involved in a wide range of metabolic processes, including not only carbohydrate x and fatmetabolism, but also modulationandimmune and inflammatory responses.Adipokines contribute to the chronic inflammatory process and interact with other cytokines, which together enhance degenerative processes in the joints [12]. Due to synergistic relationships with IL1, adipokines can enhance catabolic processes in cartilage and increase the synthesis of pro-inflammatory mediators in joint tissues [13].Thus, obesity, cardiovascular lesions and other pathogenetic metabolic disorders are associated with OA, which can be viewed as a systemic disease. Adipokine levels in patients with OA were significantly higher than in the control group. In particular, high levels of adiponectin and leptin areassociatedwith female sex and high BMI [14].Odin of the main adipokines involved in metabolic processes in OAis Leptin, itis produced bywhite fat cells. And itscirculation directly depends on the amountof fat in the body. Itis not only the main regulator of body weight,butalso suppresses appetite,stimulates energy expenditure using the receptorof thehypothalamus. As we understand it,obesity is a mild inflammatory process, causingsystemic metabolic dysfunction. In obesity OA, the production of adipokines with pro-inflammatory functions increases, which contributes to the progression of the disease[26]. Recent evidence suggests that adipokines produced by white adipose tissue may provide a link between obesity and

OA, which explains the high prevalence of OA among obese and overweight people (Scotece & Mobasheri, 2015). In their review, Kluzek et al. discussed the role of leptin, resistin, and cystfatin as key mediators of catabolic pathways associated with cartilage degeneration. The paper looks at adipokines as prognostic biomarkers of early post-traumatic osteoarthritis of the knee joint (Kluzek et al., 2015b) [27]. Biomarkers help healthcare professionals diagnose disease at an early stage, observe its course and monitor how well new or existing treatments work. Biomarkers include:•Biomarkers that help diagnose disease•Biomarkers that help predict disease •Biomarkersthat allow you to assess the physical condition of the patient.In 2006, the group Bauer et al., (2006) published a paper presenting the characteristics and classification of OA biomarkers. The diagnosis of OA is usually based on clinical and radiographic changes that occur very late and have low sensitivity for monitoring the progression of the disease (Rousseau & Delmas, 2007, Rousseau & Garnero, 2012)., in addition to biomedical imaging (Rousseau & Garnero, 2012) [28]. To date, there are many barriers to theidentification ofbiomarkers that reflect OA: Gistochemical data are not associated with clinical signs such as pain and function. First of all, it has been shownthat inflammation in OA is not only local, but also systemic, which complicates standard studies. Various factors of systemic inflammation are immeasurable: age, genetics, diet, activity, kidney function, liver function, weight and other comorbidities and manyaboute oe.Adipokines associated with inflammation include classic pro-inflammatory agents (IL-6, TNF-α). IL-6 produced by adipose tissue accounts for about a third of circulating IL-6 and is strongly associated with increased obesity (Proenca et al., 2014) [29].The group of inflammatory cytokines is the most important group of compounds involved in the pathogenesis of OA. They are most responsible for the loss

of metabolic homeostasis of the tissues that form the joints by promoting catabolic and destructive processes. The key role they play in the pathogenesis of OA is as a result of exposure to these compounds on most cells that are found in the joint, and the influence through intracellular signalingpathways on the production of cytokines, as well as other inflammatory compounds and enzymes. Among the many representatives of this group, the greatest importance is attached to the IL-1, IL-6. [30].Interleukin (IL)-1 is a cytokine that plays an important role in inflammatory responses in the contextofinfectionand immune-mediated diseases. IL-1 is present in the synovial tissue and fluids of patients with OA and rheumatoid arthritis. Several in vitro studies have shown that IL-1 stimulates the production of mediators such as prostaglandin E(2), nitric oxide, cytokines, chemokines, andadhesion molecules. which are involved in joint inflammation. In addition, IL-1 stimulates the synthesis and activityof matrixmetalloproteinase and other enzymesinvolved in the destruction of cartilage in OA and RA. The effects of IL-1 are inhibited in vitro and in vivo by natural inhibitors, such as the IL-1 receptor antagonist andsoluble receptors. The IL-1 receptor antagonist inhibits the action of IL-1, blocking its interaction with the surface receptors of cells.

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