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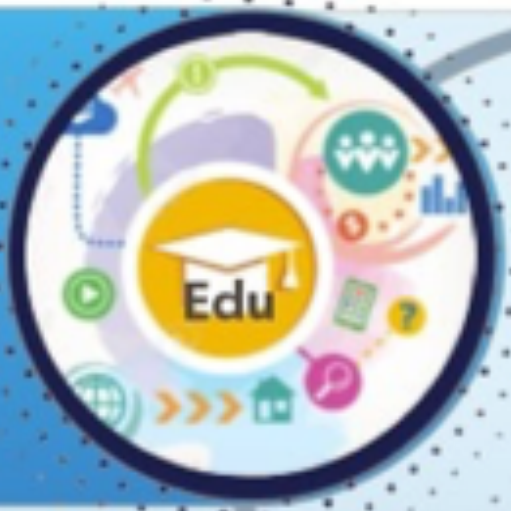

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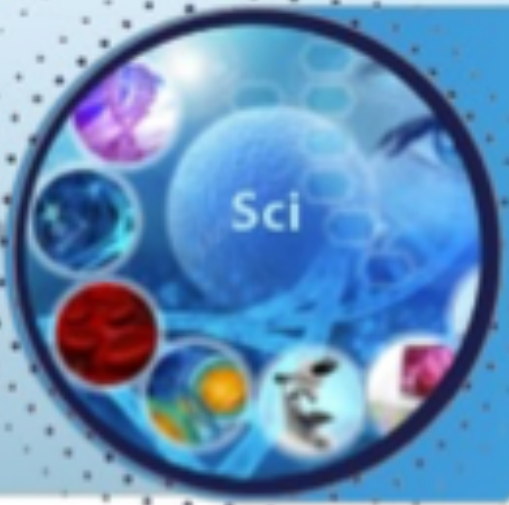

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Review Article

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The Role and Place of Humoral Immunity in Bone Regeneration

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ABSTRACT

The immune system is actively involved in bone regeneration. This reasoning may stem from the peculiarities of immune cell populations, which are known to be diverse and heterogeneous. Such functional and cytological versatility of bone tissue, to a certain extent, may indicate the similarity of their natural relationship. The basis of this connection is regenerative processes, which we decided to describe in this review article.

Keywords: Bone tissue regeneration, role of humoral immunity, callus, bone marrow

Cell-mediated immunity isn't the only system that can respond to bone damage. Traumatic bone injury leads to reactive changes in the humoral link of the immune system as well. In her experimental studies, N.V. Davydova showed how possible it is to inhibit the proliferation of lymphocytes activated in vivo when interacting with fibrillar collagen. He proved that pre-activated lymphocytes could be significantly responsible for altering the regenerative process of bone.

R. Wildburger et al. in their studies revealed a direct correlation between the concentration of specific intrinsic antibodies based on low-density lipoproteins of an oxidative nature and the intensity of bone tissue proliferation. Stimulation and production of such antibodies occur under the influence of bone trauma [32].

When comparing the conditions of the influence of B-lymphocytes on the process of bone tissue healing, M. Richter's studies revealed disorders in the synthesis of immunoglobulins. This process occurred as a result of the action of B-lymphocytes [18]. Data have been obtained that prove an inverse correlation between the production of class G immunoglobulins and the severity of the traumatic agent. Polytrauma, combined injuries, traumatic shock – all of them were accompanied by an intensive decrease in the synthesis of this immunoglobulin. At the same time, in patients with solitary uncomplicated fractures, such changes were not noted. This may indicate a direct link between the synthesis of immunoglobulin G and bone consolidation.

Humoral immunity is characterized by a decrease in the concentration of immunoglobulins of the main classes. Non-specific resistance, primarily oxygen-dependent killing mechanisms (myeloperoxidase) is reduced. Complement activity in traumatic bone injuries is also reduced.

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Bone growth factors, which form the callus over time, differentiate along with collagen production. The sources of this transformation are bone marrow fibroblasts. They are important participants in distraction osteogenesis. With the positive dynamics of such a regenerative process, this whole process contributes to the germination of connective tissue and the formation of calluses [9].

Interesting data are presented in several literature sources regarding the role of lactoferrin in bone consolidation. Such studies have been conducted in a variety of formats and environments. This conclusion indicates that lactoferrin, which usually indicates the generalization of the inflammatory process, due to immunological competence through the humoral pathway, exerts its influence on the course of bone tissue regeneration [1, 2].

Lactoferrin is known to be found in the secretory granules of neutrophils. Its secretion is determined by the intensity of the inflammatory process [23].

To date, the mediating effect of lactoferrin has been proven by a large amount of scientific research [14].

It has been proven that lactoferrin under normal conditions of generalization of the inflammatory process can affect the production of chemokines [20].

Lactoferrin may affect the production of pro-inflammatory cytokines that increase the intensity of the inflammatory process. Among them are the pro-inflammatory cytokines TNF-α and IL-lα. These cytokines are osteolytic and can reduce the intensity of bone regeneration [11].

Due to such properties as stabilization of binding complexes, lactoferrin has a direct effect on the development of bone cells – osteocytes [39].

Lactoferrin is known to have a stimulating effect on the intensive growth of cartilage cells. This has been proven in studies where the stimulating effect of lactoferrin on the growth of osteoblasts has been revealed. Moreover, lactoferrin has the function of protecting osteoblasts, creating conditions for bone tissue regeneration in both the diaphyseal and epiphyseal zones. By preventing apoptosis of bone cells, lactoferrin can enhance bone cell function by creating conditions for maintaining normal bone structure [10].

More in-depth studies on the potential effects of lactoferrin on osteoclasts were conducted in the laboratory on the bone marrow of mice. Its superiority has been noted over other cell growth factors. Among them, calcitonin, C-terminal peptides, several pro-inflammatory cytokines (IL-1α, TNF-α, IL-18), adrenomodulin, amylin, insulin and others were used. All of them were lower in their effects than lactoferrin [3].

A group of scientists led by G. Mundy, based on the results of the studies described above, began to apply lactoferrin topically in mice with a bone fracture and without a pathological process under experimental conditions. Lactoferrin has been shown to have a potent anabolic effect on osteoblasts. In animals with bone fractures, topical application of lactoferrin showed an increase in regeneration. At the same time, local administration of lactoferrin to animals with whole bones was observed to suppress bone resorption [36].

Preosteoclasts are target cells for lactoferrin. It is their action that leads to the formation of a large number of mature cells of this genesis. However, several researchers, creating isolation conditions with mature osteoclasts, under the use of lactoferrin, could not reveal any resorption effect on bone tissue [21].

K.P. Palmano and D.F. Elgar discovered and quantified lactoferrin in bovine serum samples by reversible-phase high-performance liquid chromatography on polystyrene-divinylbenzene. In this work, the resorption properties of lactoferrin on bone tissue were demonstrated. Such an impact was reflected through the activation of the nuclear factor system [31].

In their studies, S. He et al. proved the possibility of inhibiting mast cell activation by neutrophil lactoferrin [40]. In particular, mast cell uptake and interaction with tryptase, chimaze and cathepsin G under the action of lactoferrin have been proven. Under such conditions, lactoferrin begins to act on bone cells, stimulating differential growth, which creates conditions for the development of research in this direction.

Low-density lipoproteins belong to the family of receptor proteins that affect the primary culture of the osteoblastic line [6, 7, 8].

Along with this, osteoclastic lines of such lipoproteins also lead to an increase in osteogenesis under the influence of the same lactoferrin. This potential reflects their ability to bind to ligands [22].

Scientists have come to a consensus on low-density lipoproteins in the role of lactoferrin, which can interact with receptors in bone cells. It is osteoblasts that can identify receptors that promote bone healing during the development of an immunological reaction [41].

These studies have a certain practical value in regulating the possibility of using various methods of treatment not only for bone fractures but also for osteoporosis. Such effects are possible in both physiological and

pharmacological effects. Moreover, it is possible to use other substances in bone tissue. Lactoferrin receptors have the effect of substances on bone tissue [12].

Similar studies of lipoprotein receptors, i.e. those associated with low-density lipoproteins, suggest their role in regulating bone regeneration [5].

Thus, lactoferrin, providing bone growth, can fulfil the therapeutic goal of local action on the preservation of bone integrity and regeneration of destroyed bone integrity [24].

Systemic disorders in bone fractures on the part of the humoral link of the immune system are manifested not only by cytokines and immunoglobulins, but also by such components of the general humoral system as coloniostimulating factors, interferons, growth factors, prostaglandins, and even some lipoproteins. This makes it possible to coordinate the connection of the pathological process zone with other body systems: endocrine, nervous, and cardiovascular.

Local and general manifestations of pathological processes determine the physiological and pathophysiological manifestations of cytokine actions. At the same time, the basis of their manifestations is the relationship between the immune system, endocrine and nervous systems of the body in response to bone damage.

The immune system is closely related to the process associated with traumatic bone injury and bone regeneration. Such a connection in the cellular immune system was directly related. However, in the humoral immune system, the connection with the skeletal system occurs indirectly, along with other humoral factors. These include various representatives of pro-inflammatory and anti-inflammatory cytokines [34], ligands and cytological receptors [42, 43], coloniostimulating factors [35], and various signalling molecules. The latter is necessary for the normal functioning of the immune system and bone repair.

Cytokines such as TNF-α, TNF-β, IL-6 and IL-17 contribute to bone regeneration. They stimulate the formation of osteoclasts and regulate resorptive activity, contributing to the destruction of bone tissue [17]. In contrast, cytokines such as IL-4, IL-10, IL-13 and several others inhibit the formation of osteoclasts, both new and already existing, and thereby stimulate bone formation [13].

One of the main cytokines that can influence the process of bone formation is IL-1, which determines the number and functional activity of osteoclasts, their interaction with osteoblasts and stimulates osteoclastic resorption under physiological conditions and during pathological processes.

The effect of IL-1 on osteoclasts is mediated through the primary interaction with osteoblasts, as a result of which the activity of alkaline phosphatase is inhibited, and the synthesis of plasminogen and collagenase is activated, which leads to increased destruction of collagen.

In this way, osteoclasts gain access to the mineral component of the bone. It is assumed that the resorptive effect of IL-1 is partially realized by stimulating osteoblasts to produce prostaglandin-E2, which in turn affects the maturation of osteoclast precursors.

The stimulating effect of IL-1 on macrophage monocytes with an increase in bone formation in the demineralized bone matrix has been established. Comparative analysis showed that IL-1 stimulates the release of Ca+ from bone 13 times more strongly than IL-1α and 1000 times more strongly than TNF-α and TNF-β. With the combined effect of IL-1α, TNF-α and TNF-β there is a synergy in their action. In addition, IL-1α contributes to the potentiating effect of TNF-α.

Thus, IL-1α is an initiating factor of bone resorption, preparing the basis for the synergistic action of subsequent mediators of bone destruction. Along with activating the excretion of calcium ions from the bone, IL-1α promotes the release of proteoglycans from the bone.

IL-1 affects the proliferation and functional state of mature osteoblasts, as well as their differentiation into osteocytes, it can activate the proliferation and synthesis of collagen in osteoblasts, as well as the production of progesterone-E2 in them [25, 26, 27, 27, 29].

Other cytokines that play an important role in the regulation of osteogenesis include IL-2, which is considered one of the active inducers of T-lymphocytes. As a mitogen, it stimulates the release of collagen by fibroblasts and causes hypercalcemia, which indirectly indicates the activation of bone resorption. According to several authors, IL-2, acting on specific receptors, inhibits the growth of colony-forming units of fibroblasts, preventing their entry into phase 8, which inhibits osteoblastic processes [4].

It has been shown that a bone fracture is accompanied by a significant change in the spectrum of cytokines secreted by T-cells: the concentration of IL-2 (type 1 T-helper cytokines) and IL-10 is significantly reduced.

IL-3, which stimulates the proliferative activity of almost all hematopoietic sprouts, activates the proliferation and differentiation of osteoclasts and increases the expression of calcitonin receptors. It does not have a significant effect on the activity of osteoclastic bone resorp-

tion, however, IL-3 can modulate the proliferative activity of osteogenic progenitor cells.

IL-4 inhibits the formation and functional activity of osteoclasts through oxygen-dependent processes in cells and also inhibits bone resorption stimulated by IL-1. IL-4 can induce bone marrow mineralization.

IL-6 increases when bone resorption increases. Its action on bone cells is carried out by activating the production of other interleukins, for example, IL-l, as well as by altering the synthesis of acute-phase proteins. The involvement of IL-6 in bone remodelling processes has only recently been described, but IL-6 is the main cytokine that regulates bone formation.

The involvement of IL-8 in the regulation of osteogenesis, unlike other cytokines, is under study. An increase in basal and stimulated bone production has been shown in various pathological conditions of bone tissue [15].

IL-10 is a regulator of macrophage cell differentiation and is involved in the regulation of bone formation through the activation of hematopoiesis mechanisms. As a stimulator of T-lymphocyte proliferation, it can influence osteoclastic processes through macrophage lines [30].

IL-11 is produced by fibroblasts, and trophoblast cells and is a polypoteptic hematopoietic factor, synergistically with other hematopoietic factors, stimulates the proliferation of cell precursors. To a lesser extent, IL-11 stimulates the migration of osteoclasts. An embryonic mouse fibroblast model has shown that IL-11 enhances bone resorption and this effect is mediated by prostaglandin-E2 [37].

IL-12, along with IL-18, leads to a significant reduction in the formation of osteoclasts in cell cultures containing osteoblasts and spleen cells [33]. Experimental data suggest that the action of both cytokines is manifested only in the presence of T-lymphocytes, but the exact molecular mediator of this effect is unknown [16].

IL-18, produced by osteoblasts and stromal cells, initiates the production of IFN-γ T lymphocytes. Most studies have shown that these substances inhibit osteoclastogenesis [38], although some studies indicate that their action depends on experimental conditions.

The mechanism of bone resorption under the action of TNF-α and TNF-β is due to both the ability to cause catabolism of bone tissue and to prevent its recovery. The main target for TNF-α and TNF-β are osteoblasts, which reduce their activity under their influence. This is proven by reducing the content of alkaline phosphatase in them to 85%. TNF-α and TNF-β block the synthesis of collagen and non-collagen proteins by osteocytes.

Compared to TNF-β and TNF-α a more powerful inducer of osteoclastic processes. It activates the proliferation of fibroblasts and stimulates their production of progesterone-E2, while at the same time, it can block the gene responsible for collagen synthesis. Together with these properties, it promotes the attachment of lymphocytes to the vascular endothelium, which is important for the redistribution of cells from circulating blood to tissues during inflammation and regeneration.

The main component of the final manifestation of the activity of the kallikrein-kinin system is prostaglandins, which are synthesized through this system. This conclusion has been proven in experimental studies both in vitro and in vivo. Moreover, thrombin leads to the development of bone resorption in two ways: prostaglandin-dependent and prostaglandin-independent. Increased bone resorption also occurs as a result of the organization of a complex of bradykinins and thrombin with pro-inflammatory cytokines, in particular with IL-1.

Vascular endothelial growth factor is considered to be another factor that is involved in the regulation of bone formation. The difference in cell differentiation in the area of bone fracture can determine the timing of bone regeneration processes.

Several cytokines can affect the process of cartilage tissue repair, namely the differentiation of chondrocytes. This nature of the regenerative process is characteristic of traumatic injuries of the epiphyseal part of the bone. At the same time, the balance of the correlation of cytokine action is determined by the possibility of endothelial growth factor secretion. For example, in the case of IL-lβ and TNF-α, there is an inhibition of vascular endothelial growth factor secretion. However, IL-17 has the opposite direction of action and restores the level of secretion of vascular endothelial growth factor.

The relationship to the production of vascular endothelial factors by cartilage tissue is twofold. The first variant is manifested by an increase in the production of vascular endothelial factor by cartilage tissue under the influence of a released number of cytokines, such as IL-17, IL-lβ and TNF-α. The second variant is manifested by inhibition of the production of vascular endothelial factor as a result of traumatic damage to cartilage tissue. Accordingly, two antagonistic variants of the manifestation of the functional activity of chondrocytes can be in a balanced state in traumatic injuries of the epiphyseal part of the bone.

Increased neovascularization also occurs as a result of the production of vascular endothelial growth factor by pro-inflammatory cytokines. In this case, cartilage cells – chondrocytes – can serve as a source of vascular endothelial factors. The development of such a pathogenetic mechanism is possible in the conditions of traumatic injury of the epiphyseal zone of the bone.

To summarize this section of the chapter, cytokines can form a complete immunological response that can affect the regeneration of damaged bone tissue. This reaction involves the stimulation of fibroblast migration and the synthesis of C-reactive protein, which in turn stimulates collagenesis and the scarring process.

At the same time, the cytokine reaction, uniting into a single network of all the leading components of the immune system, participates in the regulation of osteogenesis. The importance of their role can also be considered as a starting point for the involvement of neuroendocrine, vascular and metabolic mechanisms of regeneration and remodelling in the process of bone tissue repair.

The cytokine network integrates not only with the components of the immune system that are involved in the regulation of osteogenesis, but also mobilizes the neuroendocrine, vascular, and metabolic response to create a single functional space for the repair of damaged bone tissue.

CONCLUSION

Despite a significant amount of research in the field of immunology of bone tissue damage, there is currently no complete picture of the interaction of cellular and humoral factors. Existing data require in-depth analysis to understand the mechanisms of mutual influence of bone tissue and the immune system.

An interesting direction is the study of the production and cooperation of interleukins, the synthesis of which changes in the course of damage and repair reactions since the regulatory effect of interleukins on osteogenesis has not been sufficiently studied in the clinic. Analysis of the literature data showed that osteoclasts with local production of cytokines and pro-inflammatory mediators, such as IL-1, IL-3, IL-4, IL-6, IL-11, TNF-α, TNF-β, coloniostimulating factors, leukaemia-inhibitory factor, INF-γ, TGF-β, are involved in bone metabolism and inflammatory-induced bone resorption.

T-lymphocytes and B-lymphocytes can actively influence the process of bone cell formation. A regulated function can be controlled with a proper understanding of the mechanisms of their interaction.

There is a lack of information about the importance of the influence of various methods of treatment for a fracture of long tubular bones and the possibility of immunological control of the course of regeneration processes. This would make it possible, to a certain extent, to predict the possible development of negative treatment outcomes, thereby improving their immediate and long-term outcomes, and reducing disability and economic costs.

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SUYAK TO'QIMALARINING REGENERATSIYASIDA GUMORAL IMMUNITETNING O'RNI

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[1]Respublika ixtisoslashtirilgan travmatologiya va ortopediya ilmiy-amaliy tibbiyot markazi Samarqand filiali
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ABSTRAKT

Immun tizimi suyuk regeneratsiyasida faol ishtirok etadi. Ushbu mulohaza turli xil va heterogen ekanligi ma'lum bo'lgan immun hujayra populyatsiyalarining o'ziga xos xususiyatlaridan kelib chiqishi mumkin. Suyak to'qimalarining bunday funktsional va sitologik ko'p qirraliligi, ma'lum darajada, ularning tabiiy munosabatlarining o'xshashligini ko'rsatishi mumkin. Ushbu ulanishning asosi regenerativ jarayonlar bo'lib, biz ushbu maqolada tasvirlashga qaror qildik.

Tayanch iboralar: Suyak to'qimalarining regeneratsiyasi, hujayraviy immunitetning roli, kalla, suyak iligi

РОЛЬ И МЕСТО ГУМОРАЛЬНОГО ИММУНИТЕТА В РЕГЕНЕРАЦИИ КОСТНОЙ ТКАНИ

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АБСТРАКТ

Иммунная система принимает активное участие в регенерации костной ткани. Данное рассуждение может исходить из особенность популяций иммунных клеток, которые, как известно, многообразны и гетерогенны. Подобная функциональная и цитологическая многогранность костной ткани, в определенной степени, может свидетельствует о схожести их природной взаимосвязи. Основу данной связи составляют регенераторные процессы, о которых мы решили изложить в данной обзорной статье.

Ключевые слова: Регенерация костной ткани, роль гуморального иммунитета, костная мозоль, костный мозг