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Review Article

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Modern Understanding of Immunological Aspects of Bone Regeneration

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ABSTRACT

The immune system plays an active role in the regeneration of bone tissue. This reasoning can be based on the peculiarity of populations of immune cells, which, as is known, are diverse and heterogeneous. Such functional and cytological versatility of bone tissue may indicate the similarity of their natural relationship. The basis of this connection is regenerative processes, which we decided to describe in this review scientific article. Fundamental information about the bone cell system points to the fact that the monocytic line of bone marrow stem cells is considered a matrix for the formation of macrophages. Along with this, it is this ancestral line of the cell that acts as the basis for the regeneration of the bones of the skeleton – osteoclasts.

Keywords: *Cellular immunity, traumatic injuries of long tubular bones, bone tissue regeneration*

INTRODUCTION

It is known that macrophages, participating in the pathological process, contribute to the destruction of tissues and thereby provoke the development of the following main response stage - repair. Osteoclasts have identical properties in bone tissue regeneration. They, in turn, stimulate the production of osteoblasts, that is, cells whose origin comes from mesenchymal cells. Osteoblasts develop along the osteoclastogenesis pathway, which is expressed by stromal cells. Such an origin of cells is considered natural physiological, as it reflects the entire nature of the hematopoietic process. This is the natural way in which osteoblasts develop [1].

This conclusion is based on a number of experimental and clinical studies that studied various aspects of the hematopoietic cycle and the immune system's influence on bone tissue regeneration [2].

Several studies revealed patterns in the cellular regulation of the regenerative process in bone tissue in the 1990s of the last century. This structural-functional relationship has been designated as a continuous system of mononuclear and bone cells [3].

In the consolidation of destroyed bones, hematopoietic cells, mature mononuclear phagocytes, blood monocytes, and bone marrow fibroblasts play an important role. Subsequently, inducers of both local and systemic nature and modulators of bone formation begin to be

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activated in bone formation. This mechanism is the most acceptable in interpreting the regulation mechanisms of distraction osteogenesis.

The regulation of distraction osteogenesis by lymphocytic and macrophage relationships is determined by both qualitative and quantitative changes in the immunocompetent cytological system. Carrying out such a relationship has the possibility of vector designation, determining the variant of the course of the regenerative process in bone tissue. There are two options for assessing and predicting the regenerative process's outcome: its favourable course and the slowness of bone tissue recovery.

MATERIAL AND METHODS

We analyzed publications over the past 50 years by searching the PubMed catalogue to unify the literature data. Scientific publications were studied according to the keywords "cellular immunity," "traumatic injuries of long bones," and "bone regeneration." A total of 842 publications were received in this area. We have selected the closest in meaning.

RESULTS AND DISCUSSION

Empirical conclusions about the important role of lymphocytes in the regeneration of bone tissue were carried out through banal experimental studies; by assessing the course of the pathological process after the removal of the thymus gland, researchers stated a slowdown in the process of bone regeneration. It was suggested that the deficiency of T-cells leads to a slowdown in regenerative processes in bone tissue. Further studies have shown that all types of lymphocytes do not control the process of bone regeneration in the same way. Thus, according to European researchers, the cytological picture of the bone regeneration zone was characterized by a large spectrum of immune cells of various types. Low values of the number of T-lymphocytes and mast cells were revealed. Against this background, a predominant predominance of mononuclear cells and fibroblasts was noted.

Severe traumatic tissue injuries, polytrauma, and traumatic shock – pronounced disorders in the cellular link of immunity accompany all of them. The suppression of CD2+ receptor expression determined the severity of these processes. Against this background, scientists have identified a decrease in the number of cells with CD4+ and CD8+ phenotypes. Thus, Carl J. Hauser et al. published the results of studies to assess the activity of natural killer cells in patients with injuries [4]. They put forward a hypothesis about the possibility of the participation of natural killer cells in traumatic injuries since, in such conditions, there is a high concentration of cytokines in the blood and, possibly, they regulate the activity of these cells. The study involved patients with fixed bone fragments. For comparison, blood samples from healthy volunteers were examined.

The next stage of the study was to assess the change in natural killer cells and mononuclear cells in the supernatant of the bone fracture zone, which was obtained during surgery. At the same time, bone fracture supernatants and peripheral plasma were collected during the fixation of the open fracture. Voluntary mononuclear cells have been used as effector sources (natural killer cells).

The mononuclear cells were pre-incubated with fractured supernatants and paired peripheral or normal plasma under various conditions.

Fracture supernatants have been shown to suppress the function of natural killer cells faster than peripheral plasma. Supernatants of fractures 1 to 4 days after injury were the most suppressive. Inactivation of complement and reactive oxygen species failed to restore lysis.

Neutralization of antibodies to the cytokines IL-4 and IL-10 further inhibits lysis. Antibodies to transforming growth factor β1 failed to restore lysis. The addition of INF-γ did not restore lysis, but the addition of IL-12 led to the restoration of lysis. In conclusion, it was stated that the supernatant obtained in the fracture zone and the examined blood plasma in patients with fractures suppress natural killer cells. Responsible mediators can be concentrated in soft tissue fractures/injuries. Responses to manipulation of the cytokine environment suggest that fracture cytokines may impair cooperation between natural killer cells and helper cells.

Experimental studies also prove the important role of B lymphocytes in bone tissue formation. Although there may be a close relationship between B lymphocytes and osteoclasts or bone resorbent cells, little is known about the role of B lymphocytes in bone formation. Many researchers have compared novel in vivo bone induction in mice homozygous for gene suppression with B cell deficiencies that lack functional B lymphocytes with bone induction in wild-type control mice.

The immune system, particularly the hematopoietic system, and the bone have a close relationship that can be both functional and anatomical. Osteoclasts, which resorb bone tissue, develop from precursors found in the hematopoietic bone marrow. At the same time, os-

teoblasts, bone-forming cells, can differ from bone stroma. This is an essential requirement for normal hematopoiesis. Many agents produced or acting on immune/hematopoietic cells have potent effects on bones and vice versa.

However, most in vivo data on bone turnover in immunocompromised animals are contradictory. A marked reduction in bone formation and resorption was noted in mice with previously removed thymus glands and lacking T lymphocytes. However, other studies in mice with a similar model or rats have shown that the physiological turnover of bone tissue in these animals is comparable to the physiological turnover of hematopoiesis.

In studies by A. Marusic et al. [5], it was proved that in rats in which the thymus gland had previously been removed, depression of cellular immunity had higher rates of induction of new bone by demineralized bone matrix than control rats.

They also proved that mice with β2-microglobulin gene suppression, which do not have functional class I molecules of the tissue compatibility complex and have an impaired cellular immune response, have physiological bone regeneration.

Other studies have demonstrated that B-lymphocytes can be an important regulator of normal and pathological bone resorption. In particular, estrogen is a powerful regulator of bone mass, which takes an active part in the differentiation of the B-cell hematopoietic line. The increased formation of B-lymphocytes may be involved in the mechanism of stimulated bone resorption, which is observed in estrogen deficiency. In mice with a suppressed nuclear transcription factor gene involved in osteoclast survival, neither B lymphocytes nor mature osteoclasts can be generated.

However, transgenic mice with human T-cell lymphotropic virus type I, commonly associated with T-cell leukaemia, myelopathy, and arthropathy, have an increase in the number of progenitor cells for osteoclasts and B lymphocytes. Provided that IL-7 receptors are suppressed in mice and there is a lack of B cells due to the cessation of B-lymphatic hematopoiesis at the stage of pro-B-cell maturation, the volume of the internal septa of the tubular bones increases.

Although the concurrent changes in osteoclastogenesis and B-lymphopoiesis described in these reports may not be related, recent work on the regulation of B-lymphoid line adherence has provided direct evidence that B-lymphocyte precursor cells can differentiate into osteoclasts in vitro and in vivo.

In contrast to the growing experimental support for a close relationship between B lymphocytes and osteoclasts, the role of B lymphocytes in osteoblast proliferation and differentiation has yet to be investigated. Based on the evidence that osteoclast differentiation is regulated by factors produced or transduced by osteoblasts (stromal cells), it is suggested that B lymphocytes should be involved not only in osteoclast differentiation but also in osteoblasts' regulation of bone formation.

To confirm this hypothesis, a group of scientists led by D. Kitamura studied the formation of new bone tissue in mice with a deficiency of B-lymphocytes caused by a purposeful disruption of the μ-chain that stops the development of B-lymphocytes at the stage before B-cell maturation [6].

In M.R. Urist's studies, new bone formation was evaluated using two well-defined in vivo models: ectopic bone induction by morphogenetic bone proteins, which recapitulates cellular events in endochondral bone formation and bone regeneration after fracture repair, and bone regeneration after bone marrow injury in the C model. synchronous bone formation in a well-defined anatomical place [7].

The studies of T. Shimizu et al. [8], L.J. Suva et al. [9], and Tanaka et al. [10] have also proven the popularity of such experimental models for the study of bone regeneration.

Strong evidence was presented that mice lacking functional B lymphocytes responded to the osteoinductive stimulus by producing a larger volume of new bone morphologically similar to the response of naturally occurring control groups. Changes in the expression of bone-related markers and inflammatory/immunomodulatory cytokines accompanied these changes.

Because bone can develop according to two differentiation patterns, the researchers presented an analysis of bone formation in vivo in two models: osteogenesis via cartilage bridges and osteoblasts that differed directly from mesenchymal progenitors.

The cell cascade of the epiphyseal part of the bone, which was induced by a recombinant human morphogenetic protein in a blood clot as a carrier in natural-type mice, was similar to that described in several scientific papers using recombinant morphogenetic bone proteins or bone matrix gelatin. Chemotaxis and proliferation of mesenchymal cells, precursors to cartilage, led to cartilage differentiation and hypertrophy, angiogenesis, and invasion of bone marrow cells. In remodelling, osteoblasts' differentiation and the new bone's final formation were represented by filling it with bone marrow.

Researchers typically select two times in a given cell cascade for histological analysis of newly formed tissues: at the beginning of cell proliferation and differentiation and when the ossicle is fully organized. The first moment was noted 7 days after implantation of recombinant human bone morphogenetic protein when a blood clot as a source of recombinant human bone morphogenetic protein was surrounded by a large mass of proliferating mesenchyme with very little newly induced cartilage or bone. The implants' relative volumes of blood clots, mesenchyme, cartilage, bone, or bone marrow were similar in wild-type mice.

It is important to emphasize that histomorphometric analysis is always performed on successive implant sites, which allows researchers to conclude. The volume and weight of whole implants or newly induced tissue were more significant in the B-lymphocyte-suppressed mice than in intact mice.

In the bone marrow injury model, there is an orderly process of bone marrow regeneration after bone marrow ablation, which includes capillary invasion of the bone marrow cavity, the appearance of mesenchymal cells, the proliferation of osteoblasts, the formation of cancellous bone, the reappearance of hematopoietic tissue, and osteoclastic resorption, which leads to the final regeneration of normal bone marrow.

B lymphocytes may play a role in regulating the induction and regeneration of new bone in vivo. In a model of epiphyseal osteogenesis induced by recombinant human bone morphogenetic protein, newly formed bone cells were significantly larger in mice without functional B lymphocytes than in control groups of animals.

The similarity between the relative volumes of newly induced cartilage and bone in the two groups of mice indicates that the absence of B lymphocytes did not affect the differentiation potential of osteoprogenitor cells in the newly induced tissue but rather the recruitment and proliferation of chondrogenic and osteogenic progenitors in response to the implanted recombinant human bone morphogenetic protein.

Models of novel bone induction have an important immunological component. In a model of ectopic chondro-osteogenesis induced by recombinant human bone morphogenetic protein or demineralized bone matrix, a certain degree of immune response is required to initiate an osteoinductive cell cascade following implantation of a bone matrix or bone morphogenetic protein.

Macrophages accumulating near the demineralized bone matrix early after implantation in vivo can induce directed monocyte migration at femtomolar concentrations in vitro. This suggests that monocyte chemotaxis is a key step in bone induction.

Studies by N.D. Cunningham et al. [11] have shown that morphogenetic cells also stimulate the expression of TGF-β in monocytes, which in turn stimulates bone formation and additional expression of the cells themselves. Similar results were obtained by J.M. Wozney and V. Rosen [12].

In the bone marrow ablation model, mechanical bone marrow removal is followed by blood clots, monocyte and lymphocyte infiltration, and expression of the corresponding receptors. From the study's perspective, the absence of B-lymphocytes may lead to a change in the immunological environment of the local osteoinductive sequence, which stimulates the initial accumulation and proliferation of mesenchymal progenitor cells. Increased expression of bone sialoprotein, an early bone marker, and mice resorbed large blood clots, forming more and more trabeculae of bone after bone marrow ablation. All this also confirms their early influence on osteogenesis.

Hematopoietic cells carry specific receptors and thus can participate in bone morphogenesis. Evidence of the importance of lymphocytes in the early phases of bone regeneration is also described in studies of progressive fibrous dysplasia. In one study, the disabling of ectopic osteogenesis was associated with overexpression of lymphocytes, and in the other, the earliest lesions were characterized by acute infiltration of both B lymphocytes and T lymphocytes.

The source and nature of the signals by which B-lymphocytes can influence the proliferation of mesenchymal cartilages and bone progenitor cells are currently only the subject of a hypothesis.

Many in vitro studies have documented the importance of individual cytokines and local growth factors for osteoclast and osteoblast functions. IL-1 and TNF-α are potent stimulators of bone resorption and are also involved in regulating bone formation. IL-6 acts as a downstream cytokine induced by IL-1 and TNF-α, stimulating osteoclastogenesis and osteoblastogenesis.

In the in vivo situation, there are probably very complex interactions between immunological and bone cells, and it is difficult to understand the contribution of individual factors. For this reason, many researchers' analytical comparisons differ in their opinions about the role of immunological indicators in bone tissue regeneration.

At present, researchers do not usually analyze the expression of individual cytokines but rather monitor the

profile of cytokines and bone-related factors expressed during bone fragment regeneration.

The pattern of cytokine expression during both types of osteogenesis changes in the absence of B lymphocytes. During epiphyseal osteogenesis induced by recombinant human bone morphogenetic protein, IL-1α and IL-1β, TNF-α and IL-6 are increased with bone cell differentiation in intact mice but decreased in immunosuppressed mice.

Because these cytokines may play a role in both initial cellular chemotaxis during osteogenesis and osteoclastic resorption that finally remodels bone tissue, the differences observed in the cytokine expression pattern may be due to a change in one or both of these processes in mice with immune cell suppression.

The altered pattern of cytokine expression in mice with immune cell suppression during the early phases of bone induction may reflect an altered immunological environment that promotes the early attraction and proliferation of cartilage and/or bone cells. The role of these cytokines in the differentiation and activation of osteoclasts in the later stages of the osteoinduction sequence requires further study.

The change in the cytokine expression model during osteogenic regeneration after bone marrow ablation in immunosuppressed mice is in the opposite direction from that in the epiphyseal osteogenesis model. This is not surprising because the local immunological, and in this case, the hematopoietic environment in the bone marrow is different from the environment of subcutaneous tissue, where epiphyseal osteogenesis is induced by a recombinant morphogenetic protein of human bone.

Cell-suppressed mice showed a consistent increase in IL-1α and IL-1β expression and a slight change in TNF-α expression during the first 10 days after bone marrow ablation compared to unchanged or decreasing expression of these cytokines in wild-type controls.

Studies by C.A. Dinarello have shown that since the IL-1 and TNF-α isoforms have been implicated in normal hematopoiesis [13], the changes observed in cell-suppressed mice may reflect altered bone marrow regeneration. This regeneration repopulates the resorbic trabecular reticulum in the diaphysis, compensating for the absence of B-lymphopoiesis.

M. Bhatia et al. found that the differences between non-laboratory and cell-suppressed mice in early expression were similar to those in cytokine expression, and may reflect their involvement in hematopoiesis [14].

The change in cytokine expression pattern in cell-suppressed mice was also possible with a change in osteoclast differentiation and activation during bone marrow regeneration. Most of the studies focused on bone formation because the amount of bone at 10 days after bone marrow ablation, the last time point that followed in the studies, stayed the same from earlier trials, and extensive resorption still needed to be evident.

However, the change from thicker and less separated trabeculae at day 6 post-ablation to more numerous but thinner trabeculae at day 10 after ablation, as well as a decrease in the expression of bone-specific markers at later time points in cell-suppressed mice compared to control animals, suggests that these mice may also differ in resorption and trabecular remodelling.

The results of the study by L.J. Suva et al. [15] and H. Tanaka [16] showed that such studies should assess the number of osteoclasts and their resorption activity at later points in time. All this occurs when the space of the diaphyseal bone marrow is restored by complete resorption of the trabecular meshwork.

The induction of new bone formation in the adult body in vivo has an important immunological aspect related to B lymphocytes. The stimulation of the initial steps in the cellular sequence of osteoinduction observed in cell-suppressed mice can be explained by a general imbalance of the immunological system in the absence of B lymphocytes because it is well known that B lymphocyte depletion reduces humoral (Th2) immunity and shifts the immune response towards an inflammatory and cell-mediated (Th1) response.

Such a change in local populations of lymphocytes and other immune cells and their activity can promote the migration and proliferation of mesenchymal precursors in response to an osteoinductive stimulus. Further in vivo molecular studies will be important to elucidate the possible roles and interactions of B lymphocytes with other cell populations in bone regulation.

Understanding these processes will be important in identifying molecular defects in many pathological conditions associated with the hematopoietic and skeletal systems, such as hematologic malignancies, immunodeficiency disorders, and osteoporosis.

Physiological metabolism in bone tissue can also occur in the absence of mature T-lymphocytes. Meanwhile, as the studies indicate, when the pathological process is modelled in the form of a bone fracture or surgical interventions are performed on bone tissue, the deficiency of mature T-lymphocytes is accompanied by a violation of the consolidation of bone fragments with the formation of an inferior callus.

Several scientific studies have proven the leading roles of changes in the absolute number of T-lymphocytes and B-lymphocytes and their populations in other pathological conditions that have the basis of hormonal dysfunction. These research results were similar to each other. Thus, a group of scientists led by C.J. Rosen proved a significant decrease in the absolute number of CD4+ and CD8+ lymphocytes in women with osteoporosis against the background of menopause with menopausal syndrome.

B. Abrahamsen et al. noted a significant increase in the CD4+/CD8+ ratio in this pathological condition [17], and the results of S. Epstein's studies showed an increase in the absolute number of CD3+ and CD56+ T-lymphocytes in women with this manifestation of the pathological process [18].

In a study by E.M. Shevach et al. [19] and D.P. Huston [20] on the effect of immunological control on osteoclastogenesis, it was found that classes of T-lymphocytes, such as CD4+ and CD8+, act as the main levers for triggering this process.

All of them indicate that T-lymphocytes can suppress the formation of bone cells and the resorption of their minerals. At the same time, the studies of H. Takayanagi [21] and N. Udagawa [22] showed that the suppression of osteoclastogenesis is possible under the condition of the production of interferon γ. Stimulation of the formation of bone cells, in particular osteoclasts, was achieved in experimental studies outside a living organism by a group of scientists led by V. John [23]. In these studies, CD8+ T lymphocytes were removed from cell culture. All of them contained osteoclast cells and bone pulp cells. As a result, there is an increased formation of mature osteoclasts.

D. Greevise et al. proved that these changes occur by a mechanism with the active participation of prostaglandins [24]. P. Sallusto proved that not only varieties of T-lymphocyte populations play a certain role in bone regeneration but also the variant of their influence in combination with local secretion of cytokines [25]. A corresponding vicious circle is formed, in which the destruction of bone tissue is compensated by its increased osteosclerosis. In this process, cytokines, which T-lymphocytes produce, take the starting position. Bone metabolism increases with increased bone tissue formation. Lymphocytes, especially T-lymphocytes, can also affect the regeneration of cartilage tissue. Such results of experimental studies were obtained by a group of scientists led by A. Marusise [26].

Thus, the discoveries made about the role of T-lymphocytes in the regeneration of bone and cartilage tissue indicate the presence of a relationship between the immune and skeletal systems of the body. However, as the researchers themselves point out, several other studies have yet to be carried out that could reveal the whole picture of the ongoing processes; all these studies are at the stage of continuation and to date they have also made it possible to determine the role of B-lymphocytes in the regeneration of bone tissue.

Early B lymphocytes are located in close contact with the endosteal surface of the bone and determine their role in traumatic bone injury. As they mature, B lymphocytes move to the central zone of the bone marrow, where they exert their influence through the increased production of immature cells and, accordingly, have a stimulating effect on regenerative processes in bone tissue.

A mechanism was described in which immature B-lymphocytes adhere to the vascular wall in the bone marrow in a similar way to intercellular adhesion molecules. These molecules are expressed on the surface of stromal cells, directly binding to immature forms of B-lymphocytes. A team of scientists led by P.A. Koni described this phenomenon in detail in the Journal of Experimental Medicine in 2021 [27]. According to the homing phenomenon, such molecules play a leading role in the migration of lymphocytes to the bone marrow.

The regenerative properties of B-lymphocytes during bone healing are also determined by B-cell-specific activators. This assumption was made in the works of S.L. Nutt et al. [28] and M. Sigvardsson [29]. Its presence contributes to the maturation of B-lymphocytes and vice versa; in the lack of a B-cell-specific activator, this process is suppressed or does not occur at all. Along with this, G. Smithson et al. demonstrated that immature B lymphocytes under such conditions can easily pass into the sources of other cells (macrophages or osteoclasts), which naturally originate from the bone marrow pool.

The intraoral separating part of the bone increases in volume, which can also reliably indicate the suppression of B-lymphocyte maturation processes in the bone marrow.

In 2000, a group of scientists led by J.A. Lorenzo published an article in the Journal of Bone and Mineral Research in which researchers conducted the same experiments but in the process of ontogenesis. The results of these experiments also showed that (we quote) "under conditions of ontogenesis, the lack of mature B-lymphocytes practically does not affect the cellular balance and metabolism in bone tissue" [30].

CONCLUSION

A local inflammatory reaction develops under the influence of a traumatic agent. Such a reaction can be exudative, destructive, or purulent-infectious overnight. As the severe form of the inflammatory process develops, the role of neutrophils becomes more pronounced. The outcome of any inflammatory process depends on them, in particular on their reactivity.

However, their direct role in bone tissue repair has yet to be revealed. The data available in the literature describe neutrophils' effector effect in the subsequent phases of the ongoing inflammatory process. It should be noted here that the severity of the inflammatory process determines the course of collagenosis through the production of factors that activate fibroblasts and a number of regenerative enzymes, such as collagenase, gelatinase, and stromelysin. They, in turn, play an important and direct role in remodelling the pericellular matrix.

Their functional inferiority determines the main manifestation of neutrophil disorders in traumatic injury. Such changes on the part of neutrophils occur in any type of injury and do not depend on the severity of the injury or the traumatic agent. Accordingly, the development of traumatic shock cannot indicate specific changes in this cellular immune response system. However, the literature also describes the results of scientific studies that demonstrate an increase in the migration ability of polymorphonuclear neutrophils, especially in traumatic bone injuries.

The mechanisms by which neutrophil migration ability is impaired in the event of traumatic injuries are still unclear. Researchers only state a decrease in this ability of neutrophils, but the causal effect has yet to be identified. Research in this area will be very productive for clinical practice.

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SUYAK REGENERATSIYASINING IMMUNOLOGIK JIHATLARI BO'YICHA ZAMONAVIY TUSHUNCHA

Oxunov A.O.
Toshkent tibbiyot akademiyasi

ANNOTATSIYA

Immun tizimi suyak to'qimasining qayta tiklanishida faol ishtirok etadi. Ushbu fikrlash, ma'lum bo'lganidek, turli xil va heterojen bo'lgan immun hujayralari populyatsiyalarining o'ziga xos xususiyatiga asoslanishi mumkin. Suyak to'qimasining bunday funktsional va sitologik ko'p tomonlamaligi, ma'lum darajada ularning tabiiy munosabatlarining o'xshashligini ko'rsatishi mumkin. Ushbu aloqaning asosi rejenerativ jarayonlardir, biz ushbu ko'rib chiqilgan ilmiy maqolada tasvirlashga qaror qildik. Suyak hujayralari tizimi haqidagi asosiy ma'lumotlar suyak iligi ildiz hujayralarining monositik chizig'i makrofaglarning shakllanishi uchun matritsa hisoblanadi. Shu bilan birga, hujayraning bu ajdodlar chizig'i skelet suyaklari - osteoklastlarning qayta tiklanishi uchun asos bo'lib xizmat qiladi.

Kalit so'zlar: hujayrali immunitet, uzun tubular suyaklarning shikastlanishi, suyak to'qimalarining regeneratsiyasi

СОВРЕМЕННОЕ ПРЕДСТАВЛЕНИЕ О ИММУНОЛОГИЧЕСКИХ АСПЕКТАХ РЕГЕНЕРАЦИИ КОСТИ

Охунов А.О.
Ташкентская медицинская академия

АННОТАЦИЯ

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 научной статье. Фундаментальные сведения относительно клеточной системы костной ткани указывают на тот факт, что моноцитарная линия стволовых клеток костного мозга считается матрицей для образования макрофагов. Наравне с этим, именно данная родоначальная линии клетки выступает в качестве основы регенерации костей скелета – остеокластов.

Ключевые слова: клеточный иммунитет, травматические повреждения длинных трубчатых костей, регенерация костной ткани