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ЕЖЕМЕСЯЧНЫЙ НАУЧНЫЙ ЖУРНАЛ

Медицинские новости Грузии

საქართველოს სამედიცინო სიახლენი

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თანამშრომლობითა და მისი პატრონაჟით

ЕЖЕМЕСЯЧНЫЙ НАУЧНЫЙ ЖУРНАЛ

ТБИЛИСИ - НЬЮ-ЙОРК

GMN: Georgian Medical News is peer-reviewed, published monthly journal committed to promoting the science and art of medicine and the betterment of public health, published by the GMN Editorial Board and The International Academy of Sciences, Education, Industry and Arts (U.S.A.) since 1994. **GMN** carries original scientific articles on medicine, biology and pharmacy, which are of experimental, theoretical and practical character; publishes original research, reviews, commentaries, editorials, essays, medical news, and correspondence in English and Russian.

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GMN: Медицинские новости Грузии - ежемесячный рецензируемый научный журнал, издаётся Редакционной коллегией и Международной академией наук, образования, искусств и естествознания (IASEIA) США с 1994 года на русском и английском языках в целях поддержки медицинской науки и улучшения здравоохранения. В журнале публикуются оригинальные научные статьи в области медицины, биологии и фармации, статьи обзорного характера, научные сообщения, новости медицины и здравоохранения.

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GMN: Georgian Medical News – საქართველოს სამედიცინო სიახლენი – არის ყოველთვიური სამეცნიერო სამედიცინო რეცენზირებადი ჟურნალი, გამოიცემა 1994 წლიდან, წარმოადგენს სარედაქციო კოლეგიისა და აშშ-ის მეცნიერების, განათლების, ინდუსტრიის, ხელოვნებისა და ბუნებისმეტყველების საერთაშორისო აკადემიის ერთობლივ გამოცემას. GMN-ში რუსულ და ინგლისურ ენებზე ქვეყნდება ექსპერიმენტული, თეორიული და პრაქტიკული ხასიათის ორიგინალური სამეცნიერო სტატიები მედიცინის, ბიოლოგიისა და ფარმაციის სფეროში, მიმოხილვითი ხასიათის სტატიები.

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При представлении в печать научных экспериментальных работ авторы должны указывать вид и количество экспериментальных животных, применявшиеся методы обезболивания и усыпления (в ходе острых опытов).

4. К статье должны быть приложены краткое (на полстраницы) резюме на английском, русском и грузинском языках (включающее следующие разделы: цель исследования, материал и методы, результаты и заключение) и список ключевых слов (key words).

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2. Size of the article, including index and resume in English, Russian and Georgian languages must be at least 10 pages and not exceed the limit of 20 pages of typed or computer-printed text.

3. Submitted material must include a coverage of a topical subject, research methods, results, and review.

Authors of the scientific-research works must indicate the number of experimental biological species drawn in, list the employed methods of anesthetization and soporific means used during acute tests.

4. Articles must have a short (half page) abstract in English, Russian and Georgian (including the following sections: aim of study, material and methods, results and conclusions) and a list of key words.

5. Tables must be presented in an original typed or computer-printed form, instead of a photocopied version. **Numbers, totals, percentile data on the tables must coincide with those in the texts of the articles.** Tables and graphs must be headed.

6. Photographs are required to be contrasted and must be submitted with doubles. Please number each photograph with a pencil on its back, indicate author's name, title of the article (short version), and mark out its top and bottom parts. Drawings must be accurate, drafts and diagrams drawn in Indian ink (or black ink). Photocopies of the X-ray photographs must be presented in a positive image in **tiff format**.

Accurately numbered subtitles for each illustration must be listed on a separate sheet of paper. In the subtitles for the microphotographs please indicate the ocular and objective lens magnification power, method of coloring or impregnation of the microscopic sections (preparations).

7. Please indicate last names, first and middle initials of the native authors, present names and initials of the foreign authors in the transcription of the original language, enclose in parenthesis corresponding number under which the author is listed in the reference materials.

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In GMN style for each work cited in the text, a bibliographic reference is given, and this is located at the end of the article under the title "References". All references cited in the text must be listed. The list of references should be arranged alphabetically and then numbered. References are numbered in the text [numbers in square brackets] and in the reference list and numbers are repeated throughout the text as needed. The bibliographic description is given in the language of publication (citations in Georgian script are followed by Cyrillic and Latin).

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ავტორთა საყურადღებოდ!

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1. სტატია უნდა წარმოადგინოთ 2 ცალად, რუსულ ან ინგლისურ ენებზე,დაბეჭდილი სტანდარტული ფურცლის 1 გვერდზე, 3 სმ სიგანის მარცხენა ველისა და სტრიქონებს შორის 1,5 ინტერვალის დაცვით. გამოყენებული კომპიუტერული შრიფტი რუსულ და ინგლისურენოვან ტექსტებში - **Times New Roman (Кириллица)**, ხოლო ქართულენოვან ტექსტში საჭიროა გამოვიყენოთ **AcadNusx.** შრიფტის ზომა – 12. სტატიას თან უნდა ახლდეს CD სტატიით.

2. სტატიის მოცულობა არ უნდა შეადგენდეს 10 გვერდზე ნაკლებს და 20 გვერდზე მეტს ლიტერატურის სიის და რეზიუმეების (ინგლისურ, რუსულ და ქართულ ენებზე) ჩათვლით.

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6. ფოტოსურათები უნდა იყოს კონტრასტული; სურათები, ნახაზები, დიაგრამები - დასათაურებული, დანომრილი და სათანადო ადგილას ჩასმული. რენტგენოგრამების ფოტოასლები წარმოადგინეთ პოზიტიური გამოსახულებით **tiff** ფორმატში. მიკროფოტოსურათების წარწერებში საჭიროა მიუთითოთ ოკულარის ან ობიექტივის საშუალებით გადიდების ხარისხი, ანათალების შეღებვის ან იმპრეგნაციის მეთოდი და აღნიშნოთ სურათის ზედა და ქვედა ნაწილები.

7. სამამულო ავტორების გვარები სტატიაში აღინიშნება ინიციალების თანდართვით, უცხოურისა – უცხოური ტრანსკრიპციით.

8. სტატიას თან უნდა ახლდეს ავტორის მიერ გამოყენებული სამამულო და უცხოური შრომების ბიბლიოგრაფიული სია (ბოლო 5-8 წლის სიღრმით). ანბანური წყობით წარმოდგენილ ბიბლიოგრაფიულ სიაში მიუთითეთ ჯერ სამამულო, შემდეგ უცხოელი ავტორები (გვარი, ინიციალები, სტატიის სათაური, ჟურნალის დასახელება, გამოცემის ადგილი, წელი, ჟურნალის №, პირველი და ბოლო გვერდები). მონოგრაფიის შემთხვევაში მიუთითეთ გამოცემის წელი, ადგილი და გვერდების საერთო რაოდენობა. ტექსტში კვადრატულ ფჩხილებში უნდა მიუთითოთ ავტორის შესაბამისი N ლიტერატურის სიის მიხედვით. მიზანშეწონილია, რომ ციტირებული წყაროების უმეტესი ნაწილი იყოს 5-6 წლის სიღრმის.

9. სტატიას თან უნდა ახლდეს: ა) დაწესებულების ან სამეცნიერო ხელმძღვანელის წარდგინება, დამოწმებული ხელმოწერითა და ბეჭდით; ბ) დარგის სპეციალისტის დამოწმებული რეცენზია, რომელშიც მითითებული იქნება საკითხის აქტუალობა, მასალის საკმაობა, მეთოდის სანდოობა, შედეგების სამეცნიერო-პრაქტიკული მნიშვნელობა.

10. სტატიის ბოლოს საჭიროა ყველა ავტორის ხელმოწერა, რომელთა რაოდენობა არ უნდა აღემატებოდეს 5-ს.

11. რედაქცია იტოვებს უფლებას შეასწოროს სტატია. ტექსტზე მუშაობა და შეჯერება ხდება საავტორო ორიგინალის მიხედვით.

12. დაუშვებელია რედაქციაში ისეთი სტატიის წარდგენა, რომელიც დასაბეჭდად წარდგენილი იყო სხვა რედაქციაში ან გამოქვეყნებული იყო სხვა გამოცემებში.

აღნიშნული წესების დარღვევის შემთხვევაში სტატიები არ განიხილება.

GEORGIAN MEDICAL NEWS
No 5 (326) 2022

Содержание:

Ataman Y.A., Brizhataia I.A., Zharkova A. V., Moiseenko I.O., Ovechkin D.
LONG-TERM BLOOD PRESSURE VARIABILITY IN STRENGTH AND ENDURANCE PROFESSIONAL ATHLETES WITH OFFICE PREHYPERTENSION OVER ANNUAL TRAINING MACROCYCLE …….....7

Pivtorak K., Monastyrskiy V., Kuleshov O., Fedzhaga I., Pivtorak N.
RELATIONSHIP BETWEEN SARCOPENIA AND OSTEOPOROSIS IN NON-ALCOHOLIC FATTY LIVER DISEASE... ……12

Borys B. Samura, Mariia O. Panasenko.
SST2 AS A PREDICTOR OF STATIN TREATMENT EFFICACY IN PATIENTS WITH MULTIPLE MYELOMA….....18

Stanislav Mashyn., Sergey Borodanov., Oksana Klymenko., Igor Lev., Katerina Shipova.
THE ROLE OF LACTOBACILLI IN THE HUMAN MICROBIOME AND METHODS OF THEIR CULTIVATION AND PRESERVATION.....23

Abdrakhmanova M.G1, Kassenova A.S[1], Omarova Sh[2], Shinalieva K.A[1], Baltabaeva A.S[1], Bakirova K.T[1].
THE USE OF THROMBOLYSIS THERAPY IN ACUTE STROKE IN THE REPUBLIC OF KAZAKHSTAN, THE COUNTRIES OF NEAR AND FAR ABROAD.…….....36

Gargin V[1,2]., Volokhova H[3]., Koshelnyk O[3]., Gulbs O[4]., Kachailo I[1]., Lytvynenko M
THE INFLUENCE OF IMMUNODEFICIENCY ON THE LEVEL OF CD34-POSITIVE CELLS IN THE CERVIX…….....40

GEORGIAN MEDICAL NEWS
No 5 (326) 2022

RELATIONSHIP BETWEEN SARCOPENIA AND OSTEOPOROSIS IN NON-ALCOHOLIC FATTY LIVER DISEASE

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The relationship between bone, muscle and adipose tissue is increasingly evident in the prevalence of sarcopenia and osteoporosis in chronic diseases such as diabetes, metabolic syndrome and obesity. Recent studies have shown [1] that skeletal muscles and bones act as endocrine organs. The coexistence of osteoporosis and sarcopenia has recently been considered a syndrome called "osteosarcopenia". The term osteoporosis reflects low bone mass and deterioration of bone microarchitecture, while sarcopenia is a loss of muscle mass, strength and function [2]. Osteopenia is a condition in which there is a decrease in bone mineral density and bone mass, but the changes are not yet so pronounced as to be considered osteoporosis.

In 2018, the European Working Group on Sarcopenia (EWGSOP) revised the definition of sarcopenia. In the revised version, low muscle strength (the most reliable indicator of muscle function) replaced low muscle mass as the main parameter of sarcopenia [3]. Sarcopenia, osteoporosis, osteosarcopenia, and vertebral fractures were significantly common and closely related in patients with liver cirrhosis. In particular, patients with osteosarcopenia had the highest risk of vertebral fractures [4].

The aim of the work. is to analyze the scientific literature on the mechanisms of interaction of bones, muscles, and adipose tissue in non-alcoholic fatty liver disease (NAFLD).

Mechanisms of sarcopenia, osteopenia and osteoporosis. Obesity is thought to affect bone health through a variety of mechanisms, including body weight, fat volume, bone formation and resorption, and proinflammatory cytokines along with the bone marrow microenvironment [5]. Human bones consist of cellular units - osteoblasts, osteoclasts and osteocytes, as well as collagen, proteins and minerals. At the same time, two processes invariably take place in the bones - destruction and remodeling of "free" components.

Sarcopenia is based on several pathological processes: reduction in the number of muscle fibers, reduction of their size, violation of the innervation of myofibrils, as well as fatty infiltration of muscles (myosteatosis) [6]. One of the features of aging-associated sarcopenia is the relative mismatch between muscle mass and muscle function, which may be due in part to myosteatosis [6]. Fatty muscle infiltration is also considered as part of the aging process, and it does not always depend on the presence of systemic obesity. At the heart of myosteatosis may be a decrease in physical activity, and fatty infiltration of muscles, in turn, leads to an additional weakening of their contractile activity, reducing muscle strength and functional abilities of the elderly. Adipokines (mainly leptin), sex hormones, glucocorticoids and impaired glucose metabolism also play a role in the development of myosteatosis. Several studies have shown that fatty infiltration of muscles not only leads to loss of muscle mass and strength, but also contributes to insulin resistance, NAFLD and type 2 diabetes [6].

Mature muscle fibers consist of multinucleated cells that are unable to divide, so muscle growth and regeneration occur due to the proliferation of satellite cells. In sarcopenia, first of all, the number of satellite cells and type II fibers decreases, which leads to the inability of the patient to make rapid movements [7].

There are important relationships between bone, fat and muscle tissue. For example, direct relationships between adipose tissue and bone mineral density are well known [8]. This includes primarily the positive effect of mechanical stress (especially overweight), which stimulates the formation of bone tissue by reducing apoptosis and increasing the proliferation and differentiation of osteoblasts and osteocytes [9]. In addition, the relationship between adipose and bone tissue may be mediated by various biologically active substances, including leptin and estrogen, which are synthesized by adipocytes, as well as sclerostin and osteocalcin, which are synthesized by osteoblasts and stimulate the secretion of adipokines and insulin [10]. Sclerostin expression leads to suppression of osteoblast-induced bone formation, and therefore to a decrease in bone mass [11].

Osteoporosis occurs due to any imbalance between the activity of osteoblasts and osteoclasts, which leads to excessive degradation of bone tissue [12]. During remodeling, bone can coordinate the activities of osteoblasts, osteocytes, and osteoclasts, thereby maintaining a dynamic balance of bone metabolism, in which osteoblasts (bone formation) and osteoclasts (bone resorption) play a crucial role in bone metabolism [13].

All cellular elements responsible for bone metabolism, including osteoblasts, osteocytes, osteoclasts, chondroblasts and chondrocytes, act under the influence of muscles, which emphasizes the key role of this tissue in bone formation [14]. Moreover, several scientific studies emphasize that both tissues perform an important endocrine function, the products of which, represented by osteokines for bone tissue and myokines for muscle tissue, realize biochemical connections between bones and muscles [15].

Vitamin D is known to play an important role in the development and maintenance of bones and muscles in the body due to its ability to regulate the absorption of calcium and phosphorus. Recent studies have shown that the level of serum vitamin D in obese people is lower than in people with normal weight, which is negatively correlated with body weight, BMI and fat mass [16].

The interdependence between bone and muscle tissue can be explained by several factors. First of all, with increasing muscle mass, the load on the bone increases, which helps to strengthen it [17]. The increase in muscle mass leads to lengthening of

collagen fibers and hypertrophy of the periosteum at the site of muscle attachment, which stimulates bone growth in this area. The blood flow to the extremities increases in proportion to the increase in muscle mass, and the increase in blood flow to the bone, obviously, increases bone strength. Muscles also perform an endocrine function, synthesizing biologically active molecules (myokines) that can affect the regulation of bone tissue [18]. The reduction of bone and muscle tissue as we age may be the same pathogenetic factors: subclinical inflammation, deficiency of hormones and nutrients, as well as reduced physical activity [18].

In chronic inflammation, proinflammatory cytokines, especially IL-6, IL-1 and TNF-α, promote osteoclast activation and subsequent bone resorption [19]. IL-6 and IL-1 directly modulate osteoclastogenesis, enhancing osteoclast function [20]. Because IL-6 levels increase during liver damage to stimulate liver regeneration, existing elevated IL-6 levels may affect bone remodeling in various types of liver disease [21].

In particular, TNF-α enhances CSF-1 receptor gene expression in the early stages of osteoclastogenesis and subsequently stimulates osteoblast precursors, leading to increased osteoclast formation regardless of the kappa nuclear factor receptor (RANKL) pathway. These proinflammatory cytokines affect the development of osteoporosis in viral hepatitis and nonalcoholic steatohepatitis (NASH). Serum soluble TNF receptor p55 levels have been shown to be significantly higher in patients with cirrhosis and osteoporosis than in patients without osteoporosis and positively associated with bone resorption [22]. In addition, because obesity is considered a chronic inflammatory condition, these proinflammatory cytokines contribute to the development of osteoporosis in NAFLD [23].

Biomedical studies show that some myokines (insulin-like growth factor 1, fibroblast growth factor, follistatin, osteonectin, osteoglycine, irisin, and interleukin-15) have an anabolic effect on bone, while other myokines (myostatin and interleukin-causing tissue) [24].

Although the mechanisms that cause bone and muscle loss are still unclear, it is now accepted that decreased muscle function causes a decrease in bone load, leading to bone loss [25]. However, a decrease in bone mass fully explains the onset of sarcopenia, and muscle atrophy does not explain the development of osteoporosis.

Pathogenetic link between sarcopenia, osteoporosis and NAFLD.

Clinical studies describe a strong association between NAFLD and sarcopenia, although the final causal relationship has yet to be determined [26]. Four large meta-analyzes showed that patients with sarcopenia had a 1.3 to 1.5-fold increased risk of NAFLD compared with those without sarcopenia [27]. Among patients with rheumatoid arthritis with concomitant NASH diagnosed with osteopenia and osteoporosis in 87.93% of cases. The European Association for the Study of the Liver (EASL), the European Association for the Study of Diabetes (EASD) and the European Association for the Study of Obesity (EASO) have linked NAFLD to colorectal cancer, metabolic bone diseases (vitamin D deficiency, osteoporosis) and rare metabolic diseases. [28].

Sarcopenia was observed in almost half of patients with cirrhosis of the liver and adversely affected mortality and prognosis of liver disease [29]. Multivariate analysis has shown that osteosarcopenic obesity, which included myosteatosis, sarcopenia, and osteopenia, has a significant association with NAFLD in women over 50 years of age, even with normal BMI [30].

Skeletal muscle plays a crucial role in the transmission of insulin signals as the primary tissue responsible for insulin-mediated glucose removal. Decreased skeletal muscle mass can cause insulin resistance and dysglycemia, which will eventually lead to NAFLD and its characteristic features [5]. Oxidative stress and chronic inflammation cause muscle atrophy and lead to stress responses in hepatocytes, leading to the progression of NASH-associated liver fibrosis, one of the stages of NAFLD development [31]. The role of an aggravating factor - hyperammonemia has been clarified. Ammonia is converted to glutamate with subsequent conversion to glutamine with the formation of leucine, which occurs in skeletal muscle. As a result of this reaction, the function of mitochondria is impaired and the level of adenosine triphosphate is reduced, which leads to a deterioration of contractile function and a decrease in muscle mass.

Skeletal muscle is an endocrine organ that secretes peptides (myokins), which have a protective effect against the development of NAFLD. Irisin is one of the myokines that plays an important role in the β-oxidation of fatty acids in the liver. Therefore, it is likely that a decrease in skeletal muscle mass may be the cause of NAFLD due to decreased secretion of various myokines [32].

According to the mechanism of sarcopenia is divided into primary (due only to the aging process) and secondary, which occurs in the presence of any pathology that can affect the condition of muscle tissue (eg, systemic inflammatory diseases). To date, many studies have been conducted to study this problem in the elderly and in patients with a number of chronic diseases. However, there are not many scientific studies on the composition of the body in the elderly and long-lived, and their results are quite contradictory [33].

Our anthropometric studies of men and women with NAFLD showed that according to step-by-step discriminant analysis, the most informative index for the diagnosis of NAFLD was muscle mass according to Matejko [34]. According to Matejko's formulas, body fat in patients with NAFLD was statistically significantly higher, and muscle and bone mass were statistically significantly less than in healthy men and women of the same sex. The inverse of medium strength ($r = 0.52$, $p < 0.001$) correlations were found between Matejko's bone mass and the body mass index in men and women with NAFLD [35]. In Ukraine, a method has been developed to calculate the probability of osteoporosis risk in patients with NAFLD using statistical methods of multidimensional factor analysis and logistic regression [36].

Numerous studies convincingly suggest that abdominal obesity, hypertension, dyslipidemia and dysglycemia, which are considered components of the metabolic syndrome, are closely related to osteoporosis [37]. Patients with NAFLD with

liver fibrosis were found to be significantly more likely to have severe osteoporosis and hip fractures in Korean men over the age of 50, and this positive association was more pronounced in patients with sarcopenia [38].

The pathophysiology of osteoporosis associated with chronic liver disease is complex. Increased bone resorption is an important pathological feature of osteoporosis, especially in patients with liver disease such as viral hepatitis and NAFLD. Molecular mechanisms underlying changes in bone structure in chronic liver disease include nuclear factor Kappa receptor activator (RANK), osteoprotegerin (OPG), activation of proinflammatory cytokines such as interleukin-1 (IL-1), IL-6 and factor tumor necrosis alpha (TNF-α), as well as low testosterone [39]. Bone remodeling is strictly regulated by osteocytes and osteoblasts by various cytokines and hormones that modulate activation and resorption [40].

Interactions between muscles and bones involve the interaction of osteokines released from bones, muscle myokines, and cytokines of dual origin, which trigger common signaling pathways leading to fibrosis, inflammation, or protein synthesis or degradation [41]. That is, if bone cells secrete less "osteokines" and skeletal muscles secrete less "myokins", fat metabolism may be impaired, as well as renal function, and even testosterone levels may change, affecting many organ functions and are usually interpreted. as "consequences of aging". This new view forces us to reconsider the pathogenesis of NAFLD. Deterioration of bone and muscle mass density, which leads to osteoporosis and sarcopenia, respectively, is an age-related process. The prevalence of NAFLD also increases with age. In addition to physiological aging, these three conditions have common underlying mechanisms, and their explanation may be critical to developing more effective NAFLD treatment strategies.

Prevention and treatment of sarcopenia and osteoperosis.

The primary task of treating patients with NAFLD with overweight and obesity is to reduce the body weight of patients. There are currently no tools for the pathogenetic therapy of NAFLD that have a convincing evidence base. The pathogenetic treatment of NAFLD is to reduce insulin resistance, oxidative and cytokine-mediated stress, reduce the level of free fatty acids and stop fibrogenesis [42].

Despite the high prevalence and clinical significance of osteoporosis and sarcopenia in patients with liver disease, the treatment of these musculoskeletal disorders is often not considered in the clinical practice of treatment of these patients. Currently, there is no specific treatment for sarcopenia, the primary prevention is. Exercise, especially those where the load gradually increases, is a stimulus for the synthesis of muscle protein. In a meta-analysis of 37 studies, including 34 randomized clinical trials, the authors assessed the effects of exercise on muscle mass in the elderly, when almost 80% of cases increased muscle mass through exercise [43].

Like other diseases, osteosarcopenia is a serious global health problem, so the focus should be on, among other things, preventing it [44]. Bone mass can also be affected by mechanical stress due to the involvement of cellular mechanisms that regulate bone remodeling. Therefore, physical therapy, which includes increasing muscle strength, is an effective way to improve bone health. Several scientific studies have confirmed the existence of a correlation between mechanical stimuli, muscle mass and protein synthesis. Increased mechanical stress causes changes in muscle mass and increases protein synthesis, which causes hypertrophy [45].

It should be emphasized that although increased protein intake effectively increases muscle mass [46], the effects on sarcopenia-related indicators such as strength and function are less amenable to correction [47], although the effects of dosed muscle load have a significant impact [48]. The results of a study in rats with glucocorticoid-induced osteoporosis showed that training on a treadmill and vibrating platform significantly reduced RANKL expression and increased OPG levels, improving osteoporosis [49]. Thus, the exercises can modulate the transmission of RANK, RANKL, OPG signals, having a beneficial effect on the condition of bone tissue.

The best modern protection against osteosarcopenia is prevention. In fact, it is well known that bone is a dynamic tissue that responds to a variety of physical stimuli, including movement, traction, and vibration, that provide movement and are fundamental to bone and muscle remodeling. The most appropriate type, intensity, duration and frequency of exercises for a positive effect on osteosarcopenia are not yet known [50].

Exercise is known to significantly reduce age-related bone and muscle loss and benefit the whole body [51]. In particular, it has been suggested that dynamic resistance supplements, supported by adequate dietary supplements, may be the most promising strategy for improving the clinical condition of elderly patients with osteosarcopenia with beneficial metabolic effects and positive effects on the nervous and cardiovascular systems. Based on these data, Kemmler et al. showed the effect of high-intensity exercise with dynamic loads in combination with the introduction of milk protein, calcium and vitamin D in elderly men with osteosarcopenia [52].

Restoring the relative skeletal muscle mass can help prevent the onset or progression of NAFLD in both non-pathologists and patients with sarcopenia [32].

Conclusion.

• Analysis of recent research has shown that a number of factors in NAFLD (insulin resistance, lipolysis, oxidative stress, decreased sex hormone levels, chronic systemic inflammation, decreased insulin-like growth factor, hyperammonemia) are the basis of the pathogenetic mechanism of sarcopenia and osteopathy.

• Decreased insulin-like growth factor and sex hormones through the intracellular signaling pathway, whose central components are the enzymes phosphoinositide-3-kinase (PI3K), AKT and mTOR kinases, have been shown to accelerate the vicious circle of decreased muscle protein synthesis and enhanced muscle growth. ulcers.

• The molecular mechanisms underlying bone resorption in NAFLD include the triad: cytokine receptor-cytokine-receptor-trap (RANKL – RANK – OPG), activation of proinflammatory cytokines, and low testosterone levels.

• Osteocyte-secreted sclerostin inhibits osteoblast differentiation and reduces bone formation.

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RELATIONSHIP BETWEEN SARCOPENIA AND OSTEOPOROSIS IN NON-ALCOHOLIC FATTY LIVER DISEASE

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Abstract. The article analyzes the scientific literature on the mechanisms of interaction of bones, muscles and adipose tissue in non-alcoholic fatty liver disease.

Modern views on the mechanisms of sarcopenia, osteopenia and osteoporosis are highlighted. Interactions between muscles and bones involve the interaction of osteokines released from bones, muscle myokines, and cytokines, which trigger common signaling pathways leading to fibrosis, inflammation, or protein synthesis or degradation. The pathogenetic link between sarcopenia, osteoporosis and non-alcoholic fatty liver disease has been established. Numerous studies convincingly

suggest that abdominal obesity, hypertension, dyslipidemia and dysglycemia are considered components of the metabolic syndrome and are closely related to osteoporosis. Methods of prevention and treatment of sarcopenia and osteoperosis have been clarified. It has been suggested that dynamic resistance exercises, supported by adequate nutritional therapy, may be the most promising strategy for improving the clinical condition of elderly patients with osteosarcopenia with beneficial metabolic effects and positive effects on the nervous and cardiovascular systems. Thus, direct relationships have been established between adipose and muscle tissue and bone mineral density. Fatty infiltration of muscles not only leads to loss of muscle mass and strength, but also contributes to insulin resistance, non-alcoholic fatty liver disease. Oxidative stress and chronic inflammation cause muscle atrophy and lead to stress responses in hepatocytes, leading to the progression of liver fibrosis. Exercise with exercise significantly reduces the loss of bone and muscle mass.

Keywords. Non-alcoholic fatty liver disease, bone, skeletal muscle, osteoporosis, sarcopenia.

РЕЗЮМЕ

ВЗАИМОСВЯЗЬ МЕЖДУ САРКОПЕНИЕЙ И ОСТЕОПОРОЗОМ ПРИ НЕАЛКОГОЛЬНОЙ ЖИРОВОЙ БОЛЕЗНИ ПЕЧЕНИ

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В статье проведен анализ научной литературы относительно механизма взаимодействия костей, мышц и жировой ткани при неалкогольной жировой болезни печени. Освещены современные взгляды на механизмы развития саркопении, остеопении и остеопороза. Взаимодействие между мышцами и костями включает взаимодействие остеокинов, выделяемых из костей, мышечных миокинов и цитокинов, которые запускают совместные сигнальные пути, ведущие к фиброзу, воспалению, синтезу или деградации белка. Установлена патогенетическая связь между саркопенией, остеопорозом и НАЖБП. Многочисленные исследования убедительно свидетельствуют о том, что абдоминальное ожирение, гипертензия, дислипидемия и дисгликемия, которые считаются компонентами метаболического синдрома, тесно связаны с остеопорозом. Выяснены методы профилактики и лечения саркопении и остеопеороза. Высказано предположение, что упражнения с динамическим сопротивлением, подкрепленные адекватной нутритивной поддержкой, могут быть наиболее многообещающей стратегией с благоприятными метаболическими эффектами и положительным влиянием на нервную и сердечно-сосудистую системы для улучшения клинического состояния пациентов с остеосаркопенией. Таким образом, установлены прямые взаимосвязи между жировой, мышечной тканями и минеральной плотностью костной ткани. Жировая инфильтрация мышц не только приводит к потере мышечной массы и силы, но и способствует возникновению инсулинорезистентности, неалкогольной жировой болезни печени. Окислительный стресс и хроническое воспаление вызывают атрофию мышц и стрессовые ответы гепатоцитов, что приводит к прогрессированию фиброза печени. Физические упражнения с нагрузкой существенно снижают потерю костной и мышечной массы.

შემაჯამებელი

სარკოპენიასა და ოსტეოპოროზს შორის კავშირი არაალკოჰოლური ცხიმოვანი ღვიძლის დაავადებაში

პივტორაკი კ., მონასტრისკი ვ., კულეშოვი ო., პივტორაკი ნ., ფედჟაგა ი.

სტატიაში გაანალიზებულია სამეცნიერო ლიტერატურა ძვლების, კუნთების და ცხიმოვანი ქსოვილის ურთიერთქმედების მექანიზმის შესახებ ღვიძლის არაალკოჰოლური ცხიმოვანი დაავადების დროს. ხაზგასმულია თანამედროვე შეხედულებები სარკოპენიის, ოსტეოპენიის და ოსტეოპოროზის განვითარების მექანიზმებზე. კუნთებსა და ძვლებს შორის ურთიერთქმედება მოიცავს ძვლებისგან გამოთავისუფლებულ ოსტეოკინებს, კუნთების მიოკინებსა და ციტოკინებს შორის ურთიერთქმედებას, რომლებიც იწვევენ სახსრების სასიგნალო გზებს, რაც იწვევს ფიბროზს, ანთებას, ცილის სინთეზს ან დეგრადაციას. დადგენილია პათოგენეტიკური კავშირი სარკოპენიას, ოსტეოპოროზსა და NAFLD-ს შორის. მრავალი კვლევა იძლევა მტკიცე მტკიცებულებას, რომ მუცლის სიმსუქნე, ჰიპერტენზია, დისლიპიდემია და დისგლიკემია, რომლებიც განიხილება მეტაბოლური სინდრომის კომპონენტებად, მჭიდრო კავშირშია ოსტეოპოროზთან. სარკოპენიის და ოსტეოპოროზის პრევენციისა და მკურნალობის მეთოდები. ვარაუდობენ, რომ დინამიური რეზისტენტობის ვარჯიში, ადეკვატური კვების მხარდაჭერით, შეიძლება იყოს ყველაზე პერსპექტიული სტრატეგია სასარგებლო მეტაბოლური ეფექტებით და დადებითი ეფექტით ნერვულ და გულ-სისხლძარღვთა სისტემებზე ოსტეოსარკოპენიის მქონე ხანდაზმული პაციენტების კლინიკური მდგომარეობის გასაუმჯობესებლად. ამრიგად, დამყარდა პირდაპირი კავშირი ცხიმოვან, კუნთოვან ქსოვილსა და ძვლის მინერალურ სიმკვრივეს შორის. კუნთების ცხიმოვანი ინფილტრაცია არა მხოლოდ იწვევს კუნთების მასისა და სიძლიერის დაკარგვას, არამედ ხელს უწყობს ინსულინრეზისტენტობის დაწყებას, ღვიძლის არაალკოჰოლური ცხიმოვანი დაავადების განვითარებას. ოქსიდაციური სტრესი და ქრონიკული ანთება იწვევს კუნთების ატროფიას და ჰეპატოციტების სტრესულ რეაქციებს, რაც იწვევს ღვიძლის ფიბროზის პროგრესირებას, რომელიც დაკავშირებულია უალკოჰოლო სტეატოჰეპატიტთან, არაალკოჰოლური ცხიმოვანი ღვიძლის დაავადების ერთ-ერთ სტადიასთან. სავარჯიშო ვარჯიშს შეუძლია მნიშვნელოვნად შეამციროს ძვლებისა და კუნთების დაკარგვა.