International Science Group

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INTERNATIONAL SCIENTIFIC AND PRACTICAL CONFERENCE "PROBLEMS OF MODERN SCIENCE AND PRACTICE"

Boston, USA

September 21 – 24, 2021

ISBN 978-1-63972-061-3

DOI 10.46299/ISG.2021.II.I

PROBLEMS OF MODERN SCIENCE AND PRACTICE

Abstracts of I International Scientific and Practical Conference

Boston, USA
September 21 – 24, 2021

Library of Congress Cataloging-in-Publication Data

UDC 01.1
The I International Science Conference «Problems of modern science and practice», September 21 – 24, 2021, Boston, USA. 487 p.

ISBN - 978-1-63972-061-3
DOI - 10.46299/ISG.2021.II.I

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BONE COMPONENT OF BODY WEIGHT IN PATIENTS WITH NON-ALCOHOLIC FATTY LIVER DISEASE

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Obesity and insulin resistance are considered to be major risk factors for nonalcoholic fatty liver disease (NAFLD), and the global prevalence of NAFLD increases with obesity and type 2 diabetes. NAFLD increases the risk of hepatocellular carcinoma [1]. There is a deterioration of bone homeostasis in patients with NAFLD, its etiology and pathogenesis is not yet fully understood. Recent epidemiological studies have shown that NAFLD is associated with diseases that are not normally dependent on obesity, such as sarcopenia and osteoporosis [2]. Recent studies by Chinese scientists suggest that NAFLD may increase the risk of osteoporosis [3].

It is believed that bone loss often accompanies aging. The size and shape of the bones are accurately modeled and changed throughout life to ensure the structure and integrity of the skeleton [4]. However, the rate of bone loss depends on different factors in different people. Some hormones are closely linked to the growth, maturation and renewal of the skeleton. Thus, IL-6, IL-1β and TNFα in combination activate osteoclasts through chronic inflammation, which leads to bone resorption. These cytokines have a common pathogenetic mechanism of chronic inflammation [5].

Some hormones are closely linked to the growth, maturation and renewal of the skeleton. These include hormones from the pituitary gland, gonads, thyroid gland and adipocytes.

Mediators of a specific link between the liver, adipose tissue and bone, in addition to TNF-α, are leptin and adiponectin [6].

Studies in humans have shown a decrease in serum adiponectin levels in patients with NASH compared with patients with steatosis [7], which provides convincing evidence that decreased adiponectin production by adipocytes plays an important role in the progression of NAFLD. The role of leptin in the regulation of inflammation has become apparent in recent years. Leptin causes a pro-inflammatory effect in models of auto-inflammatory diseases. The concentration of leptin in the serum of patients with NAFLD with overweight and obesity was statistically significantly higher compared with almost healthy individuals [7].

We have established the features of the component composition of body weight in patients with NAFLD in comparison with almost healthy people of the first adult age [8]. A comprehensive survey and analysis of anthropometric data of 112 patients with NAFLD of the first mature age of Podolskyi region and compared them with anthropometric data of practically healthy people, which were taken from the database of materials of the research center of National Pirogov Memorial Medical University, Vinnytsia. The necessary anthropometric parameters for determining the absolute amount of adipose tissue, the absolute amount of muscle tissue, the absolute amount of bone component in body weight were determined using the formulas of J. Matiegka. The inverse of medium-strength correlations betwe en Matejko's bone mass and the body mass index have been established. Our results, along with the known increase in body fat in NAFLD, showed a significant change in muscle and bone mass in the direction of decrease, which allows us to identify new potential therapeutic targets [9]. Also, with progressive liver cirrhosis, higher levels of circulating sclerostein were observed than in healthy people or patients with early liver cirrhosis [10]. Sclerostin is produced by osteocytes. It reduces bone formation by inhibiting osteoblast differentiation and proliferation.

The skeletal response in NAFLD is complex and depends on a variety of factors, such as mechanical stress, type of obesity, location of adipose tissue, sex, age, bone tissue and secreted cytokines, and that these factors may be of paramount importance for bone health [11].

Bone loss due to reduced bone formation in most cases is a direct or indirect toxic effect on the differentiation and survival of osteoblasts. The prevalence of osteoporosis and sarcopenia is significantly higher in patients with liver disease than in patients without liver disease. Osteoporosis and sarcopenia affect morbidity and mortality in liver disease, but these disorders of the musculoskeletal system are often not taken into account in the clinical practice of patients with NAFLD [12].

Conclusions. The pathogenesis of NAFLD is associated with impaired bone formation, elevated levels of leptin and sclerostin and decreased levels of adiponectin.

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Scientific publications

Materials of the I International Science Conference
«Problems of modern science and practice», Boston, USA. 487 p.
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ISBN – 978-1-63972-061-3
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The recommended citation for this publication is: Savenko V., Dubynka O., Nesterenko I., Honcharenko T., Shatruva I. Necessary factors for the effictive development of building organization // Problems of modern science and practice. Abstracts of I International Scientific and Practical Conference. Boston, USA. 2021. Pp. 18-20.

URL: https://isg-konf.com.