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Original Article

Biomechanical and functional effects of abdominal obesity on activities of daily living in individuals with low back pain

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Abstract:
Problem Statement. Striking statistics on the prevalence of low back pain among the population are negatively reflected in the medical, insurance and pension statistics of the countries of in the civilised world, as well as in international legal acts on health care. It has been established that a high body mass index is one of the provoking risk factors for low back pain. This has been confirmed by a number of studies which demonstrate significant association between low back pain development and abdominal obesity. Based on the findings of these studies, researchers conclude that abdominal obesity is an independent risk factor for low back pain. This article is an overview of the current theories of the identification and explanation of bio-mechanical and functional roles of abdominal obesity in the low back pain development. **Approach.** Theoretical methods of systematisation, explanation, and generalisation of recent theories of identification and explanation of bio-mechanical and functional roles of abdominal obesity in the development of low back pain were used. **Purpose.** Analysis and description of potential gaps in modern theories of identification and explanation of the bio-mechanical role of abdominal obesity in the development of low back pain. Filling in these gaps would contribute to extend existing recommendations for low back pain therapy. **Results.** There are only a few studies on the musculoskeletal characteristics developed as a result of the significant association between low back pain development and abdominal obesity. Existing knowledge on the identification and explanation of bio-mechanical role of abdominal obesity in the low back pain development, description and evaluation of the movement patterns of people with low back pain and abdominal obesity, and their characteristics compared to the movement patterns of healthy adults need to be supplemented by new data. **Conclusions.** A study of the mechanisms of behaviour of the musculoskeletal structures, investigation of their function, postural tone, proper activation-deactivation patterns during performance of various daily basic and complex movements in adults with abdominal obesity and low back pain will qualitatively extend the existing recommendations for the low back pain therapy.
Key Words: - **body weight, movement, physiotherapy, risk factors, vertebral disk**.

Introduction

According to the Global Burden of Disease (GBD) (Global Health Metrics, 2019; Institute for Health Metrics and Evaluation, 2019), over the past thirty years, chronic low back pain (LBP) has moved up from the 13th to the 9th position in terms of the total burden of disease, such as disability-adjusted life years (DALY), and continues to occupy the first place in terms of years of healthy life lost due to disability (YLD). This conclusion is valid for both women and men. Disability due to back pain was found increased by more than 50% in the general population since 1990. LBP may be of different etiologies. The prevalence of back pain makes it the most common among modern diseases. The number of cases is estimated to increase to 843 million LBP cases by 2050 (Global Health Metrics, 2019; Institute for Health Metrics and Evaluation, 2019). Such calculations indicate the epidemiological nature of low back pain.

Another problem, which, according to experts, is also reaching epidemic proportions, is obesity (Global Health Metrics, 2019; Institute for Health Metrics and Evaluation, 2019). An important role of obesity in the development of LBP has been confirmed by a number of research reports (Shiri et al., 2010; Zhang et al., 2018; Garzillo & Garzillo, 1994; Bener et al., 2003; da Cruz Fernandes et al., 2018; Deyo & Bass, 1989; Leboeuf–Yde et al., 1999; Dario et al., 2015; Ewald et al., 2016). Furthermore, targeted research on the role of abdominal obesity in the development of LBP attracts more and more attention. Rahman Shiri and his colleagues found that abdominal obesity (defined by waist circumference) increased the risk of LBP (Shiri et al., 2019). It is established that individuals with excess abdominal fat mass often report musculoskeletal complaints such as low back pain (Shiri et al., 2010; Uçar et al., 2012). The study conducted by İlyas Uçar and colleagues demonstrated that the abdominal adipose tissue rate was a significant and independent factor in low back pain severity, accounting for 73.5% of the variance (Uçar et al., 2012). Studies have confirmed significant correlations between waist-to-hip ratio (WHR, a measure used to identify and assess abdominal obesity) and LBP (Brooks et al.,

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2016). The results of a recent meta-analysis by You Q. and colleagues confirm that individuals with abdominal obesity tend to have an increased risk of LBP (You, Q. et al., 2022). The increasing body of evidence supports the impact of obesity, both systemic and local, on the manifestation and course of LBP. Nevertheless, assessing the potential bio-mechanical and functional effects of abdominal obesity on LBP and studying the probability of the associated development of specific movement patterns remain relevant.

This relevance is also enhanced by the fact that simple mechanical low back pain (pain arising as a result of bio-mechanical dysfunctions) is one of the most widespread in the classification of low back pain (Jenkins, H., 2002). The question about abdominal obesity biomechanical impact in mechanical causes of low back pain development needs to be clarified. The authors aim to define the probably effects of abdominal obesity on each of the links of the pathological chain leading to LBP on the basis of anatomical, physiological, and mechanical prerequisites link with activities of daily life.

Material & methods

In this article we use theoretical methods of systematization, explanation, and generalization of recent theories of identification and explanation of biomechanical and functional roles of abdominal obesity in the development of low back pain. This review comprises the articles that were retrieved by PubMed search using the strategy with no time limits until May 23, 2023 and the following terms: “obesity”, “excess body weight”, “low back pain”, “non-specific back pain”. The literature was ordered according to the relevance to the subjects covered in this review.

Results

It has been established that a high body mass index is one of the provoking risk factors for LBP. This has been confirmed by a number of studies demonstrating significant association between LBP development and abdominal obesity. Based on the findings of these studies, researchers conclude that abdominal obesity is an independent risk factor for LBP. However, there are only a few studies on the musculoskeletal characteristics developed as a result of this association.

Existing knowledge on the identification and explanation of biomechanical role of abdominal obesity in the development of LBP, description and evaluation of the movement patterns of people with LBP and abdominal obesity, their characteristics compared to the movement patterns of healthy adults needs to be supplemented by new data. A study of the mechanisms of behavior of the musculoskeletal structures, investigation of their function, postural tone, proper activation-deactivation patterns during performance of various daily basic and complex movements in adults with abdominal obesity and LBP will qualitatively extend existing recommendations for LBP therapy. However, such data and additional recommendations are also needed for the development of current methods of obesity management in order to properly and effectively organize physical exercise for people with LBP.

Discussion

It has been found that the point prevalence rate of LBP (back pain at the time of the examination) was 35%, while the annual prevalence of LBP (back pain during the year) was 70% (Kohlmann et al., 1995; Hartvigsen et al., 2018; Schmidt & Kohlmann, 2005; Schmidt et al., 2007). The proportion of patients with different localization of back pain was reported in 2004 by G. Waddell. These findings are consistent with other published data (Bogduk & Mercer, 2000; Dreyfuss et al., 2000; Caspar et al., 2003; Deyo et al., 2004) and demonstrate the following distribution of patients: 36% with cervical syndromes, 2% with thoracic syndromes, and 62% with lumbar syndromes. Various types of symptoms of the most common lumbar pain syndrome differ in relative frequency: 93% of patients have low back pain, 5% – back and leg pain, and 2% – alarming spinal symptoms (red flags).

Pain is the key subjective indicator of LBP for an individual. In the course of research, Kuslich and colleagues identified the most sensitive structures to pain in the spine: nerve trunks, the outer dorsal region of the fibrous ring of the intervertebral disc and the posterior longitudinal ligament attched to it, which are innervated by the sinuvertebral nerve (also known as recurrent nerve of Luschka or ramus meningeus nervi spinalis) (Kuslich et al., 1998). The sites of attachment of ligaments and joint capsules were less sensitive to pain, and the yellow ligament, lumbar fascia, vertebral endplates, and facet joint cartilage were completely insensitive to pain. In addition to the spinal motion segment, paravertebral muscles are also sensitive to pain. Most often, non-specific back pain has a mixed etiology. Provocative factors may include psychological, social, genetic, and biophysical factors, as well as the presence of comorbidities (Hartvigsen et al., 2018). The objective of the study was not to shed light on the intricacies of the diagnosis of one or another component of pain, since it is currently impossible to trace the share of each factor in the development of pain syndrome. Instead, the author advocates the idea of expediency and ability to determine the exact effects of abdominal obesity on each of the links of the pathological chain leading to LBP on the basis of anatomical, physiological, mechanical, and metabolic prerequisites.

The study of biomechanical characteristics of movements in people with LBP and abdominal obesity may provide promising results for the following reasons.

The Possibility of the Biomechanical and Functional Impact of Abdominal Obesity on the Intervertebral Disc as a Pain-Provoking Factor in LBP Patients

The intervertebral discs form the largest continuous structures in the adult body that have no blood supply. Unlike the surrounding tissues, the inner area of the disc is subject to the influence of both a high hydrostatic pressure and a high oncotic pressure. These two types of pressure ensure fluid movement in opposite directions, outside and inside the disk, respectively. There is an inverse relationship between the concentration of dissolved molecules and pressure gradient in the outer layers of the disc. The mechanical pressure outside the disc and the absorptive pressure inside it are in one part of this relationship, while the mechanical pressure inside the disc and the absorptive pressure outside are in the other. If one or the other part predominates, fluids and molecules move into or out of the disk. The inverse relationship between hydrostatic and oncotic pressure has important consequences for the supply of nutrients to the intervertebral disc and the function of the spinal motion segment. Thus, it makes sense to study the mechanical impact of excess subcutaneous and visceral adipose tissue on the osmotic system of the disc. Abdominal obesity, as a mechanical factor locally concentrated above the pelvis, due to its localization may create an excessive load on the intervertebral disc, when the body in a vertical position, thus contributing to excessive outflow of fluid from the disc and preventing fluid inflow into it. The impairment of nutritional supply to the disc tissue provokes its accelerated degeneration, 'wear and tear'. This can lead to a pain syndrome due to damage to the dorsal outer region of the intervertebral disc, compression of the posterior longitudinal ligament and local neural structures by the protruding disc, as well as an increase in the strain, inclination of the facet joints, and overstretching of the facet joint capsules due to a decrease of disk height (Schulitz et al., 1983). Thus, a significant factor in the form of abdominal obesity is added to the main cause of generalized disc degeneration in humans, that is senile tissue bradytrophy caused by natural static and mechanical forces. We hypothesize that a long-term increased anterior load in the form of a hanging belly can cause a compensatory anterior tilt of the pelvis (flexion), an increase in lumbar lordosis, and, therefore, a constant mechanical load and deterioration of the nutrition supply to the dorsal regions of the lumbar intervertebral discs. In addition, the disc tissues may enter into the "claws" formed by the posterior edges of the vertebral bodies that leads to an increase in the pain syndrome in LBP patients.

The Possibility of the Biomechanical and Functional Impact of Abdominal Obesity on the Facet (Zygapophyseal) Joints as a Pain-Provoking Factor in LBP Patients

The facet (zygapophyseal) joints are the most important functional components of the spine. They have mostly a dynamic role. In case of axial compression of the spine with a symmetrical decrease in the height of the discs, the articular surfaces fold with each other in the craniocaudal direction like a spyglass. A high strain of the facet joint capsule occurs mainly with additional load against the background of increased lumbar lordosis (during extension or reclination). During extension, a reduction in the joint volume is observed, because the articular surfaces meet more tightly.

The mechanism provoking a local pain syndrome in the lumbar region in patients with abdominal obesity may be as follows. First, by increasing axial vertical compression due to the mechanical load of additional weight, abdominal obesity contributes to a reduction in the disc height and an increase in the inclination of the facet joints and capsular strain. Secondly, due to the displacement of the center of mass of the body and the provocation of an increase in lumbar lordosis, the previous effect is duplicated, thus resulting in additional inclination of the facet joints and capsular strain. As a result, pain syndrome in LBP patients increases.

The Possibility of the Biomechanical and Functional Impact of Abdominal Obesity on the Muscular Structures as a Pain-provoking Factor in LBP Patients

Reduction in the elasticity of disc fibers and dehydration of the disc matrix are the causes of the most frequent functional disorder in the spinal motion segment, i.e., intervertebral hypermobility. At an early stage, this disorder can be compensated by contraction of the trunk muscles. If the functional reserve of the muscles is exhausted, a dull pain occurs that is a sign of muscle failure. The limited ability of trunk muscles to compensate for invertebral hypermobility can cause dysfunction of the spinal motion segment even in elite athletes (Rauschning, 1987).

Longitudinal muscles of the back and neck (paravertebral muscles) are among the least trained muscles of the body, despite the fact that they are under a constant significant load in modern working conditions. Abdominal obesity provokes even greater tension of the paravertebral muscles and increases ischemia and pain, by contributing to the forward shift of the center of mass. The shortening of the posterior group of trunk muscles that is occurred in response to their overstrain provokes an increase in curvature (lordosis), rather than just flattening of the spine in the lumbar region (Kapandji, 1974; Pérez-González & Llop-Harillo, 2019). This further increases improper positioning of the facet joints, stretching of the capsules of the intervertebral joints and, as a result, lower back pain, which is also known as facet joint syndrome. Overstrain of the paravertebral muscles further increases the pressure on the intervertebral discs (especially on their dorsal parts, where structures highly sensitive to nociception (pain sensitivity) are located), thus reducing the nutrient supply to the discs, which is

known to be maintained by osmosis and diffusion from the surrounding tissues. In addition, the transport of nutrients from ischemic (and sometimes inflamed) tissues can also be difficult.

In studies of the double relationship between obesity and LBP and its consequences, the issue of ischemic and inflamed tissues attracts special attention. A number of studies on the pathogenesis of obesity have demonstrated the presence of systemic inflammation manifested as an increased level of CRR in the blood of the studied subjects (Dekker et al., 2020; Saal, 1995; Li et al., 2016; Hashem et al., 2018; Wiet et al., 2017). Such inflammation is considered as latent by researchers. But in the light of the manifestation of specific pathological condition, as in the case of LBP, such inflammation can certainly have an enhancing effect, in particular, on ischemic or simply inflamed tissues. In fact, systemic inflammation, which is diagnosed in the vast majority of obese individuals, has a negative impact on local inflammation accompanying LBP. Moreover, excess of visceral fat as a metabolically active tissue can have negative effects due to its inherent pro-inflammatory influence on the surrounding tissues. This fact highlights the potential risks of abdominal obesity to LBP due to local accumulation of fat deposits. And although the scientific interest of the author concerns the study of the skeletal and muscular structures of patients with LBP and abdominal obesity from the standpoint of biomechanics, the author's findings can extend the data on systemic and local inflammation in persons with obesity and LBP. In particular, the potential pro-inflammatory influence of adipose tissue can affect muscle function that can be assessed using biomechanical tools.

According to the clinical practice guidelines for low back pain interventions, the 2021 recommendations provided by George S.Z. and colleagues for the use of exercise as a therapeutic intervention in LBP patients of different age groups are considered to have level A evidence (George et al.,2021). These authors point out that for the correct and efficient use of physical exercises to treat LBP, abdominal obesity should be considered as a significant biomechanical factor. The study of the mechanisms of behavior of the musculoskeletal structures, investigation of their function, postural tone, proper activation and inactivation during performance of various daily basic and complex movements in adults with abdominal obesity may be a qualitative addition to the recommendations provided by George et al. (2021). The prospect of organizing such studies would reinforce the view expressed in a series of articles in the Lancet in 2018 regarding the need to reduce variability of care to improve the quality of LBP treatment through the synthesis of available evidence and use of evidence-based interventions (Hartvigsen et al., 2018; Foster et al., 2018). Accordingly, the prospect of investigating the biomechanical and functional impact of abdominal obesity on activities of daily living in people with LBP would add to the existing recommendations, making them more targeted and accurate on the basis of the obtained data (Simonet et al., 2020). At the same time, such data could contribute to increasing the level of safe use of physical exercise in the system of excessive body weight management in patients with LBP (Andrieieva & Nahorna A., 2020; Nahorna et al., 2020).

The Possibility of the Biomechanical and Functional Impact of the Postural Changes in Individuals with Abdominal Obesity as a Provoking Factor for LBP

The key muscles as the main dynamic factor that can worsen or improve the postural state in individuals with abdominal obesity are the muscles that can be conventionally divided into two groups: muscles, the contraction and/or fascial shortening of which contribute to increased lumbar lordosis and pelvic flexion, and those, the contraction and/or fascial shortening of which promote flattening of lumbar lordosis and pelvic extension. Postural changes, which are manifested as increased lordosis and pelvic flexion, will undoubtedly increase the likelihood of LBP (Firmento et al., 2012; Franklin & Conner-Kerr, 1998).

In accordance with the above, we list in the table below potential postural risks of abdominal obesity in relation to the muscles that control pelvic tilt and lumbar curve (Table I).

Table I. The potential postural risks of abdominal obesity in relation to the muscles that control pelvic tilt and lumbar curve

Postural risks			
Muscle	Topography	Function	Postural effects of abdominal obesity
Increased lumbar lordosis and pelvic flexion			
Trunk muscles (mainly deep and intermediate layers)	Transversospinales muscles: according to Winkler, the fascicles originate from the vertebral plates and spinous processes of the four superior vertebrae and insert on the transverse processes of each inferior vertebra. Interspinales muscles: are located laterally to the midline and connect the spinous processes of the contiguous vertebrae. Spinalis muscles: lie medially to the interspinales and	Lumbar spine extension. With stabilized sacrum, the muscles strongly pull back lumbar and thoracic parts of the spine in the lumbosacral and thoracolumbar joints. In addition, they increase the lumbar lordosis, because they partially or completely form the muscle strings in the arc	Given the function of these muscles and a shift forward of the center of mass due to abdominal obesity, the muscles are overstrained to keep the trunk in a normal position, thereby significantly increasing the lordosis in the lumbar spine.

	deep to the transversospinales muscles. They are attached from below to the spinous processes of the two upper lumbar vertebrae and the two lower thoracic vertebrae and inserts into the spinous processes of the first ten thoracic vertebrae. Longissimus muscle (M. longissimus thoracis): lies laterally to the spinalis muscles and inserts into the lower 10 ribs along the posterior wall of thoracic and into the transverse processes of the lumbar and thoracic vertebrae. Iliocostalis muscle (M. iliocostalis): lies deep and laterally to the aforementioned muscles. At the bottom, it is attached to the posterior part of the lateral sacral ridge, extends to the angles of the 10 lower ribs and further to the transverse processes of the three cervical vertebrae. Serratus posterior muscle: Belongs to the intermediate layer. It originates from the spinous processes of the first three lumbar vertebrae and the two lower thoracic vertebrae, extends obliquely upwards and laterally, and inserts in the inferior borders and lateral surface of the four lower ribs.	formed by the lumbar region of the spine. Thus, the posterior muscles of the trunk pull it back and increase the bend in the lower back.	
Iliopsoas muscle (mostly the iliacus belly)	It is attached proximally to the transverse processes of the lumbar vertebrae, inserts into the bodies of the T12 and L5 vertebrae and extends to the top of the lesser trochanter of the femur.	When the femurs are stabilized with both these muscles and the hip joints are stabilized with the contraction of other periarticular muscles, it has a significant effect on the lumbar spine manifested as the bending and formation of lumbar hyperlordosis (due to proximal attachment ventrally at the top of the lumbar lordosis).	Constant overstrain or fascial shortening of these muscles of the lower limb can potentiate the development of lumbar hyperlordosis, when combined with other pathological biomechanical outcomes associated with abdominal obesity.
Muscles of the lower limb	Rectus femoris muscle: originates from the anterior inferior iliac spine and supraacetabular groove and extends distally to form a tendon that inserts into the quadriceps femoris tendon. Sartorius muscle: the proximal part attaches to the anterior superior iliac spine and part of the notch between the anterior superior iliac spine and anterior inferior iliac spine. The distal end of the muscle forms the tendon that inserts into the superomedial surface of the tibia. Tensor fasciae latae: originates from the outer lip of the iliac crest and inserts into the iliotibial tract, which attaches to the tibia.	In addition to the main functions, increases pelvic flexion and thereby provokes hyperlordosis, when the lower limbs are stabilized and the above muscles are contracted.	Constant overstrain or fascial shortening of these muscles of the lower limb can potentiate the development of lumbar hyperlordosis, when combined with other pathological biomechanical outcomes associated with abdominal obesity.
Flattening of lumbar lordosis and pelvic extension			
Anterior abdominal wall	The muscles arise from the ribs, costal cartilages of ribs V to VII, and the xiphoid process and attach to the	They are simultaneously strong trunk flexors, pelvic extensors, and the	Excess of adipose tissue in the abdominal cavity leads to stretching of the

muscles (mainly the rectus abdominis muscle and, to a lesser extent, the internal and the external oblique muscles)	superior border of the pubic crest and the pubic symphysis.	muscles that flatten lumbar lordosis.	rectus abdominis muscle and constant increase in the distance between its attachment sites, which is accompanied by pelvic flexion and caudal movement of the distal attachment site at the pubic symphysis.
Gluteus maximus	The muscle arises from the posterior region of the outer surface of the ilium and from posterior surface the lower part of the sacrum and inserts into the gluteal tuberosity of the femur.	In addition to the main function of the muscle, i.e. extension of the hip with external rotation, these muscles flatten lumbar lordosis due to pelvic extension and verticalization of the sacrum, when the lower limbs are stabilized.	Taking into account other pathological and biomechanical changes associated with abdominal obesity, the weakness of the large gluteal muscles, can contribute to lumbar hyperlordosis.
Muscles of the posterior thigh	Semimembranosus muscle: originates from the ischial tuberosity and inserts into the tibia. Semitendinosus muscle: begins from the lower and medial impression on the upper part of the tuberosity of the ischium and from the sacrotuberous ligament and inserts into the tibial tuberosity. Biceps femoris: the long head originates from the ischial tuberosity and inserts into the fibula.	In addition to the main function of the muscle, i.e. extension of the lower leg with external rotation, these muscles flatten lumbar lordosis due to pelvic extension, when the lower limbs are stabilized.	Pathological and biomechanical changes associated with abdominal obesity can provoke stretching of the muscles of the posterior thigh due to the increase in the distance between their attachment sites (as a result of pelvic flexion and cranial movement of the ischial tuberosity) that in turn provokes even greater hyperlordosis.

Thus, the negative biomechanical impact of abdominal obesity can be manifested as an increase in pelvic flexion and the inability to compensate for it sufficiently due to overstretched abdominal muscles.
Another characteristic and typical postural effect of excessive body weight, including abdominal obesity, which can provoke LBP, is club-foot (secondary, as a result of axial load due to excessive body weight, or primary, which begins to progress largely due to axial loading of excess weight). This pathology can progress depending on the rate of body weight gain, amount of excessive body weight, adaptive capacity of the tissues (muscles, tendons, ligaments and joints) to increased axial load, anatomical and physiological disposition to club-foot, amount of vertical physical load, and even the footwear. One of the consequences of club-foot is valgus collapse, when the medial surface of the ankle joint shifts inward and the lateral part of the foot moves outwards, that can create pathological biomechanical conditions contributing to the development of LBP, which are presented for better visualization in the figure below (Figure 1).

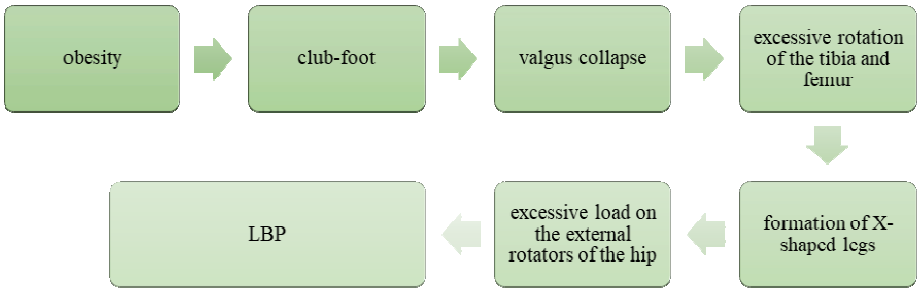

Figure 1. The model of the potential postural risks of the obesity

The piriformis muscle (external rotator of the thigh) originates on the anterior surface of the sacrum and from the second and third sacral openings, passes through the greater sciatic foramen, and inserts onto the the greater trochanter of the femur. Constant overload caused by the above-mentioned negative postural changes leads to the overstrain of this muscle, formation of trigger points, development of edema, then ischemia, tendinitis, and, eventually, of piriformis syndrome. Given that the piriformis muscle is attached to the sacrum, it can cause pain not only in the buttock and trochanter region, but also in the lower back. The author have described only a few possible negative postural consequences of excessive body weight, including abdominal obesity. Further research in this field is required to extend existing knowledge.

Conclusions

Overall, we can state that abdominal obesity is a significantly acquired negative bio-mechanical factor. The results of studying the current state of the issue have shown that excess fatty tissue can limit the natural movement of joints. An increased amount of fat in the region of the abdominal cavity can topographically create an extra load on the intervertebral disc when a person is in all vertical positions. Abdominal obesity can provoke an increase in the deflection in the lower back as a compensation for the displacement of the centre of mass. Besides, an increased deflection in the lower back (hyperlordosis), in turn, can cause muscle overstrain and, thereby, cause pain. As a result of the mechanical pressure of excess weight, the height of the intervertebral discs can be decreased, and the intervertebral structures wear out more quickly - such degenerative processes can later cause lower back pain. An excess of adipose tissue provokes systemic inflammation in the body. Although this inflammation is latent, it can increase the number of LBP manifestation degree.

As summaries, we can highlight that the study of abdominal obesity impact on the biomechanics of the movement in people with LBP are required. The analysis of their movement patterns and comparison with the movement patterns of healthy people, the study of muscle activation-deactivation patterns and the analysis of the obtained data can provide promising results from the point of view of extending and improving current recommendations for LBP prevention and treatment by detailing them for individuals with abdominal obesity.

Conflicts of interest — The authors certify that there is no conflict of interest with any financial organization regarding the material discussed in the manuscript.

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