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BIOMECHANICAL AND BIOCHEMICAL CHARACTERISTICS OF *MUSCULUS SOLEUS* CONTRACTION IN OBESE RATS

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Background: Obesity is accompanied by complex metabolic disorders that significantly affect skeletal muscle physiology, including contractile activity and fatigue resistance. However, the mechanisms linking obesity to altered muscle function remain insufficiently characterized.
Objectives: The present study aimed to evaluate the impact of obesity on the biomechanical parameters of *musculus soleus* contraction in rats under fatigue conditions. In parallel, blood levels of creatinine, creatine phosphokinase (CPK), and lactate dehydrogenase (LDH) were measured to assess the relationship between metabolic alterations and neuromuscular dysfunction associated with obesity.
Materials and methods: Experiments were conducted on male white non-linear rats divided into control and high-calorie diet groups. The *musculus soleus* contraction force was recorded using strain gauges attached to the muscle tendons. Electrical stimulation of the L4-L5 efferents was performed with 2 ms impulses at 1 Hz, generated through platinum electrodes controlled by an ADC-DAC system. Muscle fatigue was induced by three stimulation series separated by 5 min rest intervals. The maximum contraction force, time to decrease muscle contraction force by 25% and 50% of the initial level and muscle force impulse were determined. Blood levels of creatinine, CPK, and LDH were measured by commercial kits.
Results: The findings have shown that obesity cause a significant suppression of the contractile activity of rat *musculus soleus*. The concentrations of creatinine, CPK, and LDH in the blood of obese rats increased, indicating impaired neuromuscular function and insufficient recovery during repeated stimulation. These alterations reflect obesity-induced dysfunction in muscle energy metabolism and contractile mechanisms.
Conclusions: Obesity causes pronounced suppression of the contractile activity of *musculus soleus* and disrupts neuromuscular homeostasis, as evidenced by both biomechanical and biochemical markers. The findings contribute to understanding the pathophysiological basis of muscle fatigue in obesity and may support diagnostics of obesity-related muscular dysfunction.
KEY WORDS: *musculus soleus*; obesity; muscle fatigue; contractile activity; biomechanical parameters of muscle contraction; biochemical blood indicators

One of the most important characteristics of the human muscular system is its ability to maintain the level of effort generation, determined by the central nervous system (CNS), for a

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certain period of time. If the factors that induce contractions continue to reach the muscle with constant intensity, and the level of force generated by the muscle gradually decreases, this phenomenon is called muscle fatigue. Muscle fatigue is a protective mechanism of the body against overload and the subsequent development of muscle pain [1]. Its degree is a key factor in the development of adaptation and the improvement of the body's functional and physical capacities. During intense physical activity, the duration of the recovery period (active rest of the muscle) is also an important parameter for maintaining optimal performance and the normal physiological state of contracting muscles [2]. Interestingly, the effects of fatigue are observed not only in the actively working muscle fibers but also in adjacent ones that have not been pre-stimulated. The causes of this effect may include the diffusion of metabolites from active to inactive muscle fibers; reduced effectiveness of neuromuscular transmission due to compression of motor terminals and altered extracellular metabolite composition; and local hypoxia caused by increased intramuscular pressure, which, in turn, alters the excitability and contractile capacity of muscle fibers [3].

Several studies indicate the development of skeletal muscle dysfunction in obesity, such as impaired insulin sensitivity [4]. It has been shown that rats with obesity exhibit disturbances in muscle protein synthesis in response to chronic lipid infiltration [5]. In addition, a decline in oxidative capacity and abnormal organization of muscle fibers has been observed in the skeletal muscles of obese patients [6]. However, the specific features of skeletal muscle contraction dynamics under conditions of obesity remain poorly studied.

Damage to the molecular structure of muscles during the development of obesity may be one of the possible causes of their reduced contractility. Degenerative changes, including partial loss of transverse striation and increased spacing between bundles of muscle fibers, have been observed after the development of obesity [7]. According to the authors, such structural changes may result from oxidative damage, as evidenced by elevated levels of malondialdehyde and lactate dehydrogenase (LDH) in the systemic circulation.

It is known that obese individuals are characterized by a predominance of fast-twitch (type II) muscle fibers and a smaller proportion of slow-twitch (type I) muscle fibers [8]. Evidence suggests that a relative reduction in type I muscle fibers is associated with impaired metabolic health, increased low-density lipoprotein levels, decreased insulin sensitivity, and reduced arterial elasticity [9]. At the same time, type II muscle fibers exhibit a reduced ability to oxidize lipids, which is linked to lower whole-body lipid oxidation and increased lipid accumulation [10]. Furthermore, the type II muscle fiber phenotype is more closely associated with elevated oxidative stress. Therefore, the lack of data on the effect of obesity on the slow skeletal muscle (*musculus soleus*) contractile activity prompted us to conduct such studies.

Today, it is well established that there is no single, distinct mechanism responsible for the development of the muscle fatigue phenomenon; rather, there exists a wide range of mechanisms operating at different systemic levels [11, 12]. These include disturbances in the functioning of the CNS, dysfunction of peripheral nerves and neuromuscular junctions, as well as reversible physiological changes occurring directly within the actively working skeletal muscles. Naturally, such large-scale physiological alterations are reflected in the composition of blood, the body's main transport medium.

Thus, the present study aimed to comprehensively evaluate the effect of obesity on the biomechanical parameters of *musculus soleus* contraction in rats, including the maximum contraction force, the muscle force impulse, and the time required for the reduction of contraction force to 25% and 50% of the initial values, under conditions of fatigue development. In parallel, we assessed key biochemical markers of muscle performance – creatinine, creatine phosphokinase (CPK), and LDH — in the blood plasma of obese rats to elucidate the relationship between metabolic disturbances and functional impairment of the neuromuscular system.

MATERIALS AND METHODS

Experiments on rats were conducted in compliance with international recommendations for biomedical research involving animals, in accordance with the guidelines of the European Convention for the Protection of Vertebrate Animals Used for Experimental Purposes. The study was approved by the Bioethical Committee of Taras Shevchenko National University of Kyiv (Protocol No. 5, dated 09.09.2020).

Experiments were carried out on male white non-linear rats with an initial weight of 135–140 ± 5 g. During the first week, the rats were provided with standard food and water *ad libitum*. On the eighth day of the experiment, the animals were randomly divided into two groups. The first group (Control, n = 20) received standard food and water, while the second group (High-Calorie Diet, HCD; n = 20) was fed a high-calorie diet consisting of standard food (60%), pork fat (10%), chicken eggs (10%), sucrose (9%), peanut butter (5%), milk (5%), vegetable oil (1%), and water [13, 14]. The rats were sacrificed on the 70^{th} day of the experiment.

The body mass of the rats was recorded once a week, while food and water intake were monitored daily in both groups. The body mass index (BMI) — defined as the ratio of body mass (g) to the square of body length (cm^2) — was calculated at the end of the experiment.

The concentration of insulin in the blood of rats was determined by an enzyme-linked immunosorbent assay (ELISA) [14]. The concentration of glucose in the blood was measured in animals that had been fasted for at least 2 h. Blood samples were collected from the caudal vein using a catheter, and glucose levels were determined with a Glucophot-II glucometer (Ukraine). The concentration of glycosylated hemoglobin in the blood of experimental rats was measured using standard kits manufactured by PLIVA-Lachema Diagnostika (Czech Republic) [14].

The concentration of creatinine in rat blood was measured using the kinetic Jaffe method [15] without deproteinization, for the quantitative determination of serum creatinine. The concentration of CPK was determined by the immunoinhibition method with measurement in the UV region of the spectrum, according to the International Federation of Clinical Chemistry (IFCC) protocol [15]. The concentration of LDH was measured using an optimized IFCC method [15].

The preparation for the experiment included intraperitoneal administration of nembutal (40 mg/kg) for anesthesia. Standard preparation procedures also included cannulation for drug administration and pressure monitoring, tracheotomy, and laminectomy at the level of the lumbar spinal cord. The *musculus soleus* was dissected free from the surrounding tissues and transected transversely at the distal part of its tendon. To prepare for controlled stimulation of the efferent fibers in the L4–L5 segments, the ventral roots were severed at their exit points from the spinal cord. During both surgery and the experimental procedure, heart rate and electrocardiogram amplitude were continuously monitored. Anesthesia was maintained by intraperitoneal injections of a ketamine/xylazine mixture every 30–40 min until the end of the experiment. Body temperature was maintained at 37–38 °C using an infrared lamp [16, 17].

The muscle contraction force was measured using strain gauges attached to the tendons of the examined muscle. Programmable signal generators of a specific waveform were used to produce the stimulating signals [16]. Stimulation of the efferent fibers in the L4–L5 segments was performed with electrical impulses of 2 ms duration and 1 Hz frequency, generated by a pulse generator controlled by an ADC–DAC system (Analog to Digital Converter – Digital to Analog Converter) and delivered through platinum electrodes.

To induce muscle fatigue, three series of electrical stimulations were applied, with a 5-min rest interval between them. The current intensity at which the muscle began to contract was considered the threshold, and subsequent stimulation was performed at 1.3–1.4 times the

threshold value. The biomechanical parameters, namely the time required for the reduction of *musculus soleus* contraction force by 25% and 50% from the initial level, as well as the maximum contraction force, as markers of muscle damage [17], in both the Control and HCD groups were recorded. The muscle force impulse, calculated as the total area under the force–time curve, was determined using Origin 9.4 software (OriginLab Corp., USA) [17].

Data analysis, including statistical processing and graph plotting, was performed using Origin 8.0 software (OriginLab Corp., USA). Differences were considered statistically significant at $p < 0.05$. The statistical reliability of differences between groups was assessed using Student's *t*-test.

RESULTS AND DISCUSSION

The results of the preliminary metabolic assessment confirmed the successful induction of obesity in rats maintained on a HCD. As shown in Table 1, animals from the HCD group demonstrated a significant increase in body weight, BMI, and blood glucose levels compared to the Control group ($p < 0.05$). Elevated concentrations of glycosylated hemoglobin and insulin further indicated disturbances in carbohydrate metabolism and the onset of insulin resistance. These findings are consistent with earlier reports describing the development of chronic low-grade inflammation and metabolic dysregulation in adipose tissue during obesity [18].

Such systemic alterations are known to profoundly influence skeletal muscle structure and function. The disruption of metabolic homeostasis, accumulation of lipids, and chronic inflammatory background can impair neuromuscular communication and contractile performance. Therefore, in the next stage of the study, a detailed biomechanical analysis of *musculus soleus* contraction dynamics was carried out to evaluate the effect of obesity on its functional state. This approach made it possible to identify characteristic changes in the force generation, fatigue development, and recovery capacity of skeletal muscle under conditions of obesity-induced metabolic imbalance.

Table 1. The key indicators characterizing the disruption of the functioning of metabolic processes associated with the development of obesity in rats

Indicators	Control group	HCD group
Feed consumption, g/day	28 ± 2	33 ± 2*
Water consumption, mL/day	38 ± 2	32 ±2 *
Weight gain, %	108 ± 6	160 ± 8*
BMI, g/cm^2	0.60±0.03	0.84 ± 0.05*
Concentration of glucose, mM	4.5±0.4	7.1±0.5*
Concentration of glycosylated hemoglobin, μmol f/g hem	0.21±0.03	0.81±0.06*
Concentration of insulin, rel.units/mg protein	0.13±0.01	0.26±0.02*

*$p < 0.05$ — the difference is statistically significant compared to the Control group.

Figure 1 shows the mechanograms of *musculus soleus* contraction in the Control and HCD groups, obtained during electrical stimulation at a frequency of 1 Hz in three consecutive series, each separated by a 5-minute rest interval. The time required for a 25% and 50% reduction in contraction force from the baseline level was 14 ± 1 min and 25 ± 1 min, respectively, during the first series of stimulation. In the second and third series, the corresponding values decreased to 9.0 ± 0.5 min and 19 ± 1 min, and 4.0 ± 0.2 min and 11 ± 1 min, respectively.

These data indicate that, unlike the Control group, rats maintained on a high-calorie diet exhibited a progressive decline in muscle endurance, as evidenced by the faster reduction in contraction force across successive stimulation cycles (Fig. 2). It should be noted that the control values remained stable throughout the experiment, confirming the reproducibility of the stimulation protocol and the absence of fatigue accumulation under normal metabolic conditions.

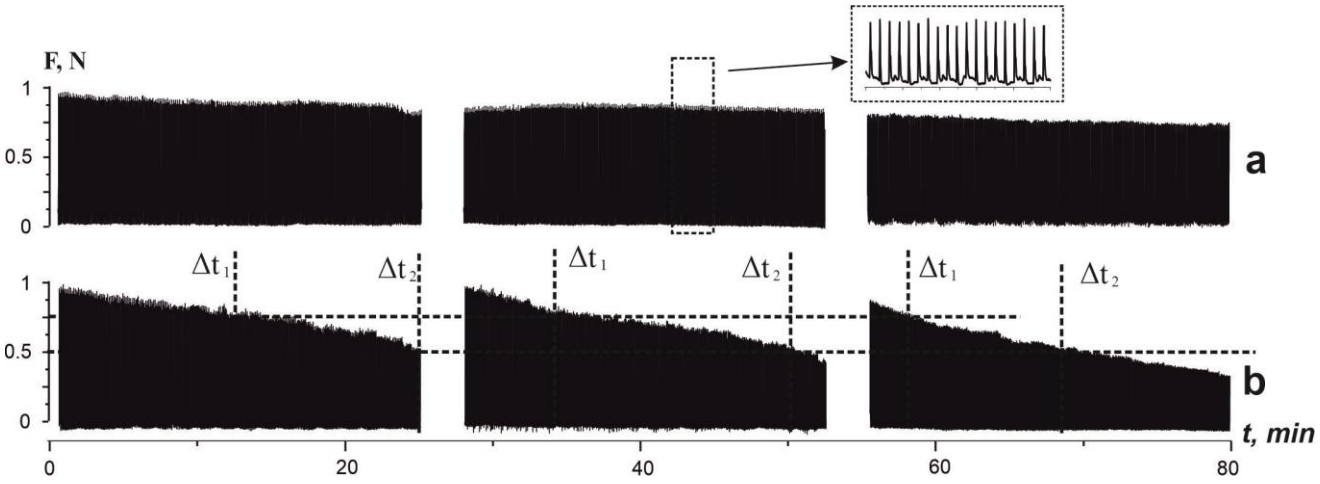

Fig. 1. The force of *musculus soleus* contraction (F, N) caused by electrical stimulation at a frequency of 1 Hz, performed in three series with 5 min relaxation between each: a — Control group; b — HCD group. Δt_1 and Δt_2 — the time of reduction of the muscle force contraction by 25% and 50%, correspondingly, from the initial level.

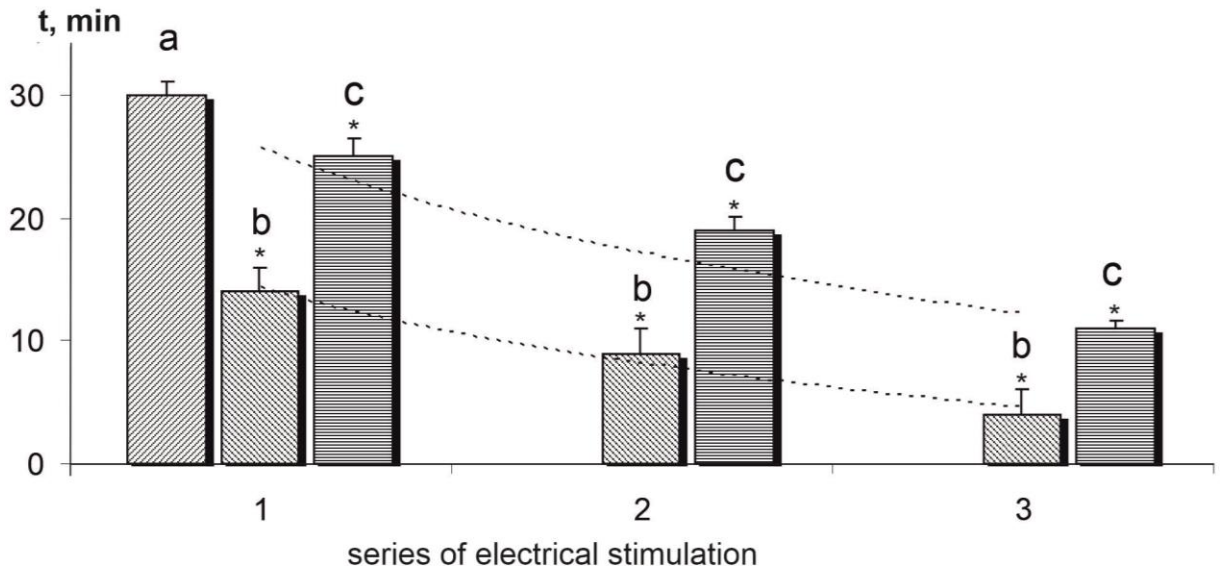

Fig. 2. The time (t, min) of reduction of *musculus soleus* contraction force by 25% (b) and 50% (c) from the initial level in obese rats caused by electrical stimulation at a frequency of 1 Hz, performed in three series (1, 2 and 3) with 5 min relaxation between each. $*p < 0.05$ — the difference is statistically significant compared to the Control group (a).

The maximum contraction force generated by an active muscle reflects its ability to perform rapid, untargeted movements and serves as an indicator of the overall functional state of the muscular system [16]. As shown in Figure 3, the maximum contraction force of the *musculus soleus* in the HCD group decreased to 82 ± 5% during the first series of contractions, while in the second and third series it dropped further to 63 ± 3% and 38 ± 2%,

respectively, compared with the Control group. Such a progressive reduction in force generation suggests a significant impairment of neuromuscular performance in obese rats. The observed changes may result from disturbances in both neural regulation and myogenic mechanisms contributing to the pathology under investigation [2, 19].

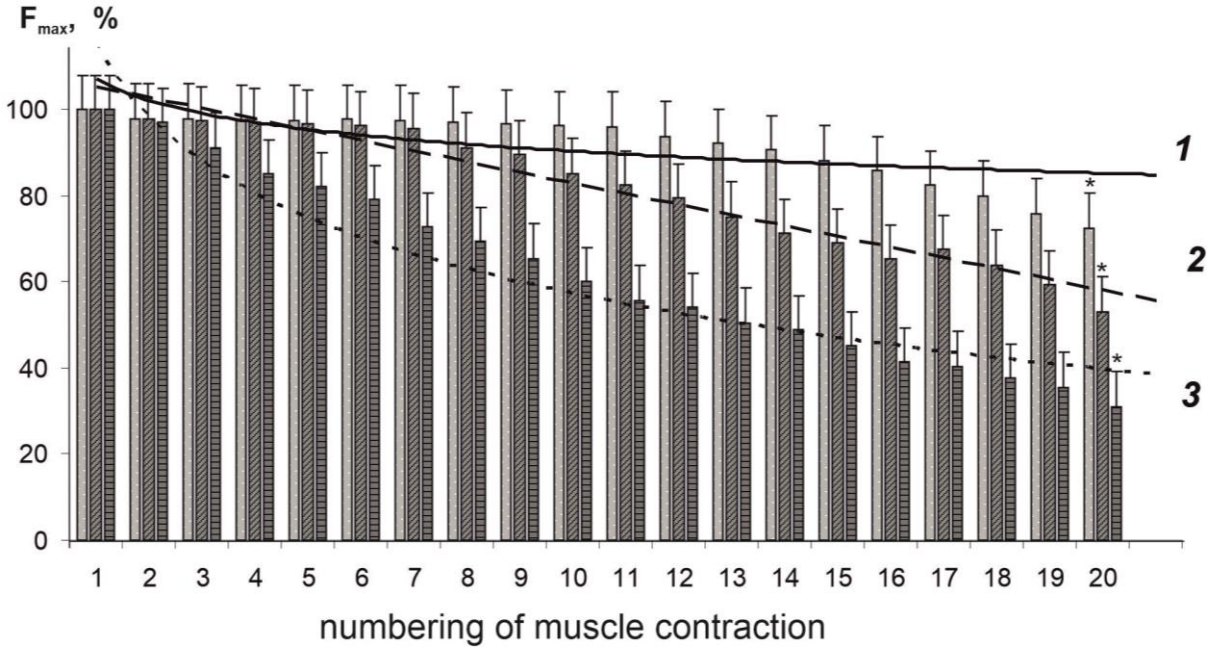

Fig. 3. The maximum force of *musculus soleus* contraction (F_{max}, presented as a percentage of the control levels taken for 100%) in obese rats caused by electrical stimulation at a frequency of 1 Hz, performed in three series (1, 2 and 3) with 5 min relaxation between each. 1–20 — the consecutive muscle contraction; $*p < 0.05$ — the difference is statistically significant compared to the Control group.

The muscle force impulse, which represents the integrated mechanical work performed by the muscle during a series of contractions, serves as an indicator of its overall functional performance [16]. The obtained results show that this parameter decreased to 83 ± 5%, 67 ± 3%, and 49 ± 2% in the first, second, and third series of contractions, respectively, compared with the Control group (Fig. 4). This progressive decline may be associated with increased intramuscular collagen deposition, the presence of non-functional muscle fibers, and inflammatory processes that develop against the background of obesity [20, 21].

As can be seen, in contrast to the Control group, the biomechanical parameters of muscle contraction in obese rats began to decline from the first minutes of activation (Figs. 1–4). Thus, obesity leads to a pronounced dysfunction or desynchronization of these biomechanical characteristics. As a result, the muscle, functioning as a dynamic system, is unable to adequately respond to neuronal impulses originating from the CNS [19] and therefore cannot maintain the target position or maximal force output throughout the stimulation cycle.

Changes in the chemical composition of blood during the development of fatigue processes reflect the biochemical alterations occurring in skeletal muscle [22]. One of the main biochemical indicators of muscle fatigue is the blood concentration of creatinine, the amount of which depends on muscle mass and the level of its activity. In our study, we observed an increase in creatinine concentration with the development of skeletal muscle fatigue in the Control group after each of the three series of contractions (Fig. 5). The difference in creatinine content between the first and third series was 10 ± 1%, reflecting the physiological fatigue of the control muscles.

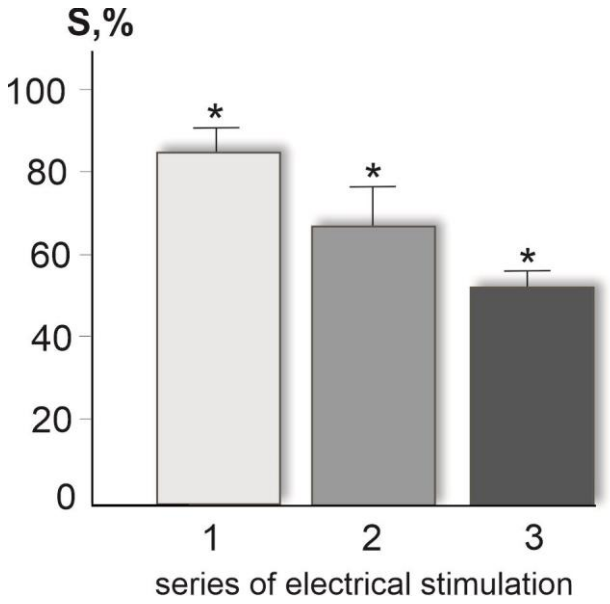

Fig. 4. The muscle force impulse (S, presented as a percentage of the control values taken for 100%) caused by electrical stimulation at a frequency of 1 Hz, performed in three series (1, 2 and 3) with 5 min relaxation between each. *$p < 0.05$ — the difference is statistically significant compared to the Control group.

In contrast, the blood creatinine concentration in obese rats was significantly higher than in the Control group across all series of the experiment. After the first and third stimulation series, the levels increased by 38 ± 2%, indicating a more pronounced metabolic response (Fig. 5). It should be noted that the observed increase in blood creatinine levels in fatigued rats was not associated with any nephrotoxic effects of the barbiturates used for anesthesia, since in the Control group the rise in creatinine remained within the physiological range.

Another marker of muscle fatigue development is the content of CPK. When muscle damage or intense activity occurs, this enzyme is released from muscle cells, leading to an increase in its concentration in the blood. In our study, we observed a slight elevation in CPK levels after the development of muscle fatigue in the Control group, which indicates the presence of physiological muscle fatigue (Fig. 5). In contrast, the CPK concentration in obese rats increased by 32 ± 2% between the first and third series of contractions (Fig. 5), suggesting a significant impairment of the neuromuscular system in these animals.

In our opinion, the elevated blood CPK fraction reflects a non-specific physiological disruption of the myocyte membrane integrity, accompanied by the partial release of intracellular enzymes into the extracellular space. This process is likely associated with increased energy demands of the muscle to overcome the enhanced stiffness of active muscle components.

The level of LDH is an important marker of tissue damage and cellular destruction, and its concentration in the blood typically increases during intense muscular activity. In our study, the LDH concentration increased following the development of muscle fatigue in the Control group (Fig. 5), which is consistent with previous reports [23] describing similar elevations during high physical activity or muscle injury.

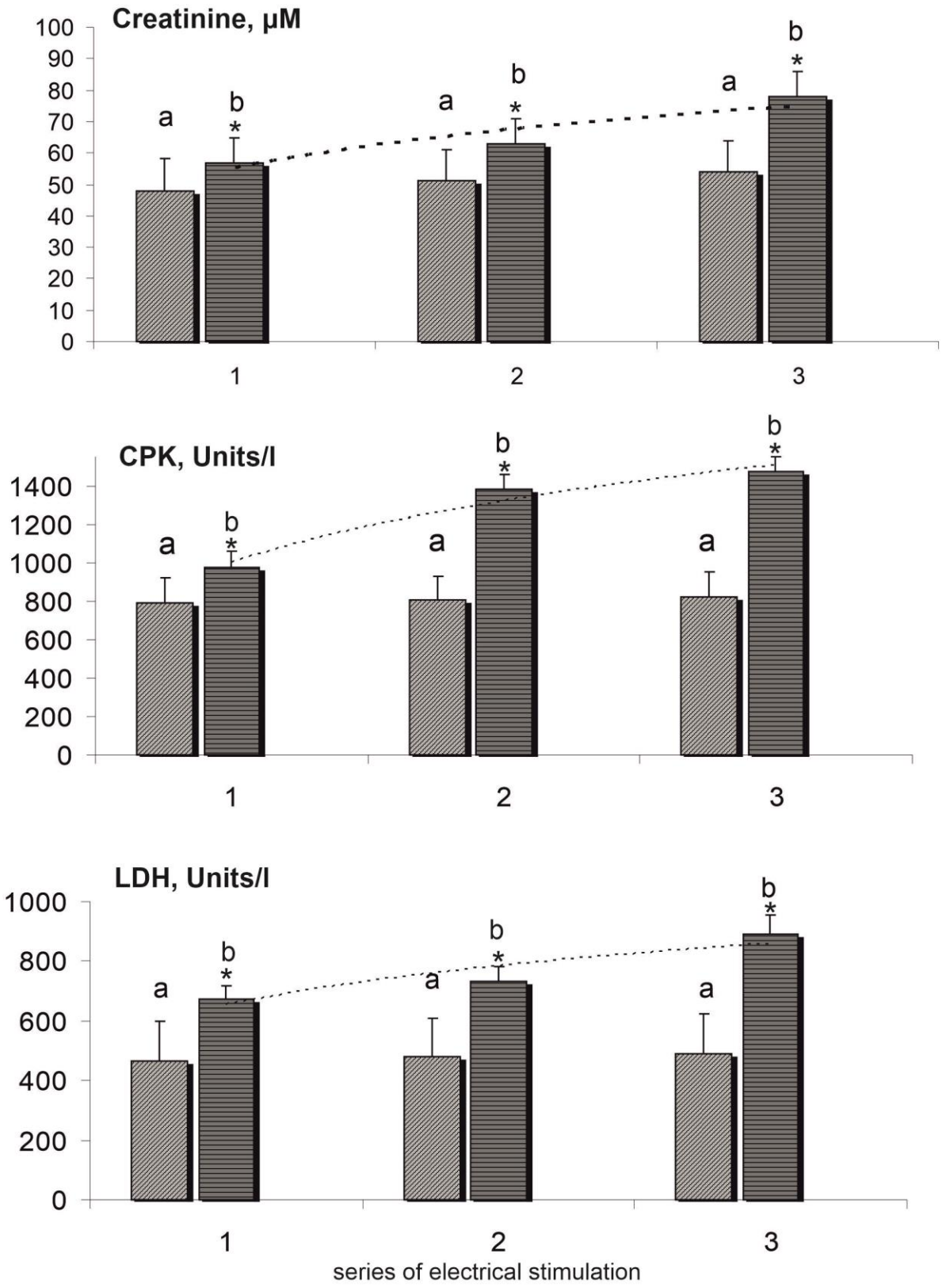

Fig. 5. The concentrations of creatinine, CPK and LDH in the blood plasma of rats after the *musculus soleus* contraction caused by electrical stimulation at a frequency of 1 Hz, performed in three series (1, 2 and 3) with 5 min relaxation between each: a — Control group; b — HCD group. *$p < 0.05$ — the difference is statistically significant compared to the Control group.

In obese rats, the LDH content increased by 27 ± 1% between the first and third series of contractions (Fig. 5), indicating that the 5-minute rest interval was insufficient for the restoration of muscle biomechanical parameters. Consequently, further stimulation resulted in both impaired activation of motoneuron pools and a significant reduction in the energy components of muscle contraction.

CONCLUSIONS

Thus, the effects of obesity on the dynamics of *musculus soleus* fatigue development and associated blood biochemical parameters in rats were investigated. The results showed that obesity causes a significant suppression of the contractile activity of the rat *musculus soleus*, with the analyzed biomechanical parameters reduced by an average of 50 ± 5%. Moreover, an increase in the concentrations of the studied biochemical markers in the blood of obese rats by an average of 32 ± 3% indicates a marked impairment of neuromuscular function.

The obtained results provide new insights into the mechanisms of skeletal muscle dysfunction under metabolic overload and may contribute to the diagnosis and prevention of pathological conditions of the muscular system associated with obesity.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

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БІОМЕХАНІЧНІ ТА БІОХІМІЧНІ ХАРАКТЕРИСТИКИ СКОРОЧЕННЯ *MUSCULUS SOLEUS* ЩУРІВ З ОЖИРІННЯМ

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Актуальність. Ожиріння супроводжується складними метаболічними змінами, які суттєво впливають на фізіологію скелетних м'язів, зокрема їх скоротливу активність і стійкість до втоми. Проте механізми, що поєднують ожиріння з порушенням м'язової функції, залишаються недостатньо з'ясованими.

Мета. Метою цього дослідження було оцінити вплив ожиріння на біомеханічні параметри скорочення *musculus soleus* щурів за умов розвитку втоми. Поряд з цим визначали рівні креатиніну, креатинфосфокінази (КФК) та лактатдегідрогенази (ЛДГ) у крові для оцінки взаємозв'язку між метаболічними порушеннями та нервово-м'язовою дисфункцією, асоційованою з ожирінням.

Матеріали та методи. Експерименти проводили на білих нелінійних самцях щурів, розділених на контрольну та групу, що перебувала на висококалорійному харчуванні. Силу скорочення *musculus soleus* реєстрували за допомогою тензодатчиків, прикріплених до сухожиль м'яза. Електричну стимуляцію еферентів сегментів L4-L5 здійснювали імпульсами тривалістю 2 мс з частотою 1 Гц через платинові електроди, керовані системою ADC-DAC. М'язову втому індукували трьома серіями стимуляцій,

розділеними 5-хвилинними інтервалами відпочинку. Визначали максимальну силу скорочення, час зниження сили скорочення м'яза на 25% і 50% від початкового рівня та імпульс сили м'яза. Рівні креатиніну, КФК і ЛДГ у крові визначали за використання комерційних наборів.

Результати. Встановлено, що ожиріння спричиняє значне пригнічення скоротливої активності *musculus soleus* щурів. Підвищення концентрацій креатиніну, КФК та ЛДГ у крові тварин із ожирінням свідчить про порушення нервово-м'язової функції та недостатнє відновлення під час повторних стимуляцій. Такі зміни відображають розвиток дисфункції енергетичного метаболізму та скоротливих механізмів м'язів під впливом ожиріння.

Висновки. Ожиріння призводить до вираженого пригнічення скоротливої активності *musculus soleus* і порушення нервово-м'язового гомеостазу, що підтверджується біомеханічними та біохімічними показниками. Отримані результати поглиблюють розуміння патофізіологічних основ розвитку м'язової втоми при ожирінні та можуть бути використані для діагностики ожиріння-асоційованих м'язових дисфункцій.

КЛЮЧОВІ СЛОВА: *musculus soleus*; ожиріння; м'язова втома; скоротлива активність; біомеханічні параметри скорочення м'яза; біохімічні показники крові.