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INDICATORS OF *MUSCULUS SOLEUS* CONTRACTILITY DISORDER IN OBESE RATS

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Obesity has become a widespread issue across the globe, reaching epidemic proportions. Being overweight is a known risk factor for developing impairments in muscle performance. The aim of the study was to estimate mechanokinetic parameters of musculus soleus contraction in obese animals to better understand the possible impact of obesity on muscle contractile activity, tissue structure and appearance of damage markers in the blood. Experiments were carried out on 40 male white non-linear rats, divided equally into two groups. Control group were fed a standard diet for 10 weeks. Rats in the obesity group were maintained on a high-fat diet for the same time period. At the end of the experiment animals were anesthetized, musculus soleus was dissected, the ventral roots were severed from the spinal cord. Stimulation was performed by electrical impulses generated by a pulse generator. Tissue samples histological analysis was done with the use of Van Gieson's trichrome and Sudan Black staining. Creatinine concentration, creatine phosphokinase (CPK) and lactate dehydrogenase (LDH) activity in the blood was determined. Reduction in musculus soleus maximum contraction force and muscle force impulse, prolonged relaxation time and delayed muscle return to initial state in obese animals as compared to control group were detected indicating on skeletal muscle fatigue. The appearance of intramyocellular lipid droplets and increased amount of intramuscular collagen fibers in the muscle tissue, as well as the elevated creatinine level and increased LDH and CPK activity in the blood, confirmed the impairment of muscle state in obese rat.

K e y w o r d s: musculus soleus, obesity, muscle fatigue, contractile activity, creatinine, phosphokinase, lactate dehydrogenase, histological analysis.

The problem of obesity has spread to almost every corner of the planet to the point of epidemic proportions, with over 4 million people dying every year from comorbidities and harmful effects associated with it. Nowadays, it is widely acknowledged that obesity can lead to significant health complications such as metabolic syndrome, type 2 diabetes, and some types of cancer. In addition, obesity is a strong risk factor for cardiovascular disease, as well as for many other chronic and acute pathological states, leading to further long-term health concerns [1].

Obesity is a well-recognized risk factor for developing functional limitations in muscle performance [2]. An increase in adipose tissue throughout the body is typically accompanied by a concomitant increase in fat deposition within skeletal muscle [3], which is associated with their structural deterioration and the progression of physical disability [4, 5]. Obese individuals are also characterized by a predominance of fast-twitch (type II) muscle fibers and a smaller proportion of type I fibers [6]. 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 [7]. 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 [8]. Furthermore, the type II muscle fiber phenotype is more closely associated with elevated oxidative stress.

The intramyocellular lipid content in healthy individuals is about 1.5% and can reach up to 5% in obese individuals [9]. Recent modeling studies [9, 10] have shown that fat accumulation within

muscle leads to changes in its structural composition and interferes with contraction, thereby decreasing muscle strength. Damage to myocytes, along with increased tissue stiffness caused by obesity, results in a disruption of overall muscle force generation and performance.

Unfortunately, the exact mechanism by which intramuscular fat affects muscle contractile performance remains unclear. There is currently limited information regarding the relationship between biochemical parameters and muscle fatigue during obesity development. For this reason, in this study, we used obese rats to model muscle fatigue. We focused on assessing blood biochemical parameters such as creatinine, creatine phosphokinase (CPK), and lactate dehydrogenase (LDH), which could serve as reliable indicators of biochemical changes in skeletal muscles during fatigue. We also analyzed the most important mechanokinetic parameters of *musculus soleus* (slow-twitch (type I)) contraction (the maximum contraction force, muscle force impulse, and kinetic time characteristics) in obese animals to better understand the impact of obesity on muscle contractile activity. In addition, a histological approach was employed to analyze muscle tissue structure.

Materials and Methods

Experiments were carried out on 40 male white non-linear rats with an initial weight of 135 ± 5 g. Experiments on rats were conducted in compliance with international recommendations for carrying out biomedical research on animals, following the guidelines of the European Convention for the Protection of Vertebrate Animals Used for Experimental and Other Scientific Purposes (Strasbourg, 1986).

Ethical approval. The investigation was conducted in accordance with the Declaration of Helsinki and its amendments. This study was approved by the ESC "Institute of Biology and Medicine", Taras Shevchenko National University of Kyiv (Protocol No.5 from 09.09.2020).

Animals were divided equally into two groups (n = 20): "Control" and "Obesity". Rats in the Control group were fed a standard chow diet for ten weeks (6.7% fat, 21% protein, 55.1% carbohydrates; 15.27 kJ·g^{-1}). Rats in the Obesity group were maintained on a high-fat diet (HFD; 38.8% fat, 28.71 kJ·g^{-1}) for the same period. The HFD consisted of standard chow (60%) supplemented with pork fat (10%), eggs (10%), sucrose (9%), peanut butter (5%), dry milk (5%), and vegetable oil (1%) [11, 12].

Food intake was measured daily between 9:00 and 10:00 am. The daily energy intake (kJ·day^{-1}) per rat was calculated as the average amount of food consumed per rat (g) multiplied by the total energy content of the corresponding diet. Relative weight gain was calculated using the formula: relative weight gain (%) = [(final weight at week 10 – initial weight) / initial weight] × 100. The body mass index (BMI) was determined at the end of the experimental period according to the formula: body weight of the rat (g) divided by the square of the nose-to-anus length (cm^2) [11]. After completion of all experimental procedures, rats were euthanized, and adipose tissues (visceral and subcutaneous) were immediately removed and weighed separately. The data were expressed as relative fat mass (% of body weight × 100).

Mechanokinetic parameters, namely the force and time characteristics of muscle contraction, as markers of muscle damage [13], in both the Control and Obesity groups were analyzed at the end of the 10th week of corresponding diet feeding.

Preparation for the experiment included intraperitoneal administration of nembutal (40 mg·kg^{-1}) for anesthesia. Standard surgical preparation also involved cannulation for drug administration and pressure measurement, tracheotomy, and laminectomy at the lumbar spinal cord level. Throughout surgery and the experiment, heart rate and ECG amplitude-frequency were monitored. Body temperature was maintained at 37-38°C using an infrared lamp. *Musculus soleus* was dissected from surrounding tissues and transected at the distal tendon. To enable stimulation of the efferent fibers in the L4-L5 spinal segments, the ventral roots were severed at their exit points from the spinal cord. Stimulation was performed by electrical impulses (2 ms duration, 50 Hz frequency, 6 s train duration) generated by a pulse generator controlled via an analog-to-digital converter (ADC) and delivered through platinum electrodes.

Each experimental animal underwent 20 series of non-relaxation stimulation followed by a 30 min relaxation period after each series. External loading was applied using a mechanical stimulator system – a linear motor under position servo control. The load was transmitted to the muscle through its tendon, which was connected by a ligature to the stimulator. The stimulation parameters were programmed and delivered from the ADC-DAC (analog-to-digital/digital-to-analog converter) complex to the generator [14].

The blood concentrations of creatinine, CPK, and LDH, as markers of muscle damage [15], were determined in the experimental animals using clinical diagnostic equipment – biochemical analyzers RNL-200 and JN-1101-TR2 (Netherlands).

Fragments of *musculus soleus* were fixed in 10% neutral buffered formalin (Caplugs/Evergreen, CA, USA). Half of each muscle sample was oriented to obtain transverse sections, and the other half for longitudinal sections. The fragments were flash-frozen in isopentane pre-cooled with liquid nitrogen. Cryosections 15 μm thick were obtained at −20°C using a Shandon Cryotome FE cryostat (Thermo Fisher Scientific, San Jose, CA, USA) and mounted on glass slides. Complex histochemical staining methods were applied: Van Gieson's trichrome stain was used to evaluate fibrosis [16], and Sudan Black staining was performed to detect lipid content [17]. A 50:50 glycerin-gelatin mixture was used as a mounting medium. All microphotographs were obtained using a light microscope Axio Imager A2 (Carl Zeiss Microscopy GmbH) equipped with 10× and 40× objective lenses. Images were captured with an Axiocam 105 color digital camera (Carl Zeiss Microscopy GmbH) and processed using ZEN 2 (blue edition) software (Carl Zeiss Microscopy GmbH). Morphometric parameters were calculated using ImageJ software (National Institutes of Health, Bethesda, MD, USA).

Data processing, statistical analysis, and graph plotting were performed using Origin 8.0 software (OriginLab Corp., USA). All datasets were first tested for normality of distribution using the Shapiro-Wilk test. Since the data followed a normal distribution, intergroup differences were analyzed using the Student's *t*-test. A *P* value of less than 0.05 was considered statistically significant.

Results

Our findings revealed successful induction of obesity in rats maintained on a high-fat diet for 70 days. As shown in Table 1, by the end of the 10th week, the body weight of animals in the Obesity group was significantly higher than that of the Control group. The BMI in obese rats was also significantly increased compared with the Control group. Moreover, both visceral and subcutaneous fat depots were markedly greater in obese animals compared to controls (Table 1). These changes, along with the pronounced accumulation of adipose tissue, are consistent with a stable diet-induced obesity phenotype.

Table 1. Nutritional profile and some biometric parameters

Parameters	Experimental groups	
	Control, n = 20	Obesity, n = 20
Mean daily food intake, g·day^{-1}	28 ± 2	33 ± 3*
Mean daily energy consumption, kJ·day^{-1}	428 ± 30	948 ± 84*
Total body weight gain, %	108 ± 6	160 ± 8*
BMI, g·cm^{-2}	0.60 ± 0.03	0.84 ± 0.05*
Relative visceral fat mass, %	1.78 ± 0.05	3.13 ± 0.30*
Relative subcutaneous fat mass, %	1.13 ± 0.08	1.87 ± 0.17*

Note. Each data value represents the mean ± SD of 20 animals; *indicates significant difference ($P < 0.05$) from Control group

The adequacy of this model was further confirmed based on our previous studies using the same experimental design, in which the development of systemic metabolic dysfunction – manifested by increased serum triglycerides, glucose, and insulin levels – was demonstrated [11, 12].

The presence of fine granular lipid dystrophy, manifested by the appearance of intramyocellular lipid droplets in the muscles of obese rats (Fig. 1, blue arrows), was clearly demonstrated. An increase in the amount of intramuscular collagen fibers indicates a mild degree of fibrosis in obese rats (Fig. 1, black arrows).

Fig. 2 shows changes in the *musculus soleus* contraction force of obese rats at external loads of 0.5 N and 0.25 N (Fig. 2, *A* and Fig. 2, *B*, respectively). When these stimulation parameters were applied, a reduction in both the maximum contraction force and the muscle force impulse was observed. Analysis of the fixed-force curves after termination of the stimulation signal (t_1, t_2, see Fig. 2) demonstrated a significant increase in the time required for the contraction force to return to baseline in rats from the Obesity group, which may indicate fatigue development in their muscular system. The increase in the duration of the t_2 phase compared with t_1 (Figs. 2 and 3) could be attributed to the reduced elasticity and lability of intramuscular and fascial structures caused by obesity progression. A reduction in the number

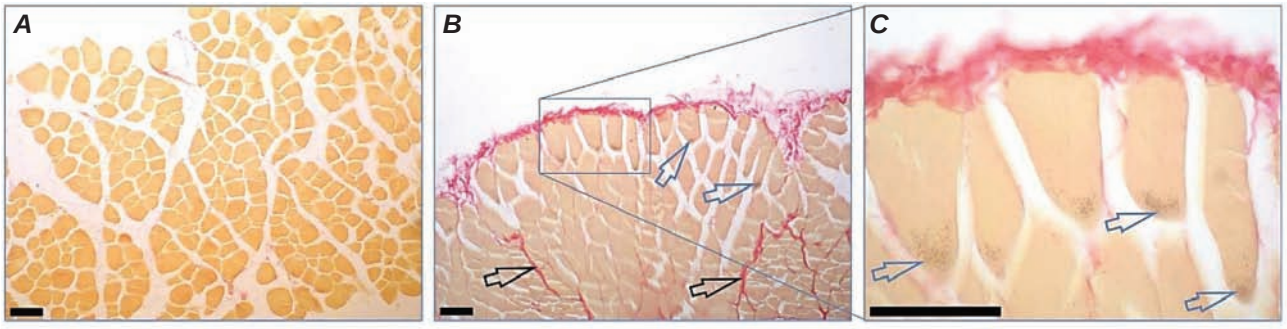

*Fig. 1. Microphotographs of rat musculus soleus tissue (cross-sections) in the Control group (**A**) and Obesity group (**B**, **C**). Combined Van Gieson (red staining – collagen fibers) and Sudan Black (black spots – lipid droplets) histochemical staining. Blue arrows indicate lipid droplets, and black arrows mark massive collagen fibers. Scale bar: 100 μm*

of active contractile components in the muscle led to a decrease in overall muscle strength, which can be observed from the changes in the external load level (Figs. 2 and 3).

The decline in maximum contractile response in experimental animals of the Obesity group began immediately after the first stimulation (Fig. 3). The muscle contraction force during the 20th stimulation series was reduced by 65 ± 4% relative to the baseline force at a load of 0.25 N, and by more than 50 ± 3% at a load of 0.5 N (Fig. 3, *C*). These data indicate a pronounced manifestation of muscle fatigue in obese animals under the applied stimulation conditions.

In order to better understand the dynamics of *musculus soleus* contraction during obesity progression, the specific transformation of descending activity was also examined. Fig. 4 illustrates the transition of active muscle response from incomplete to fused tetanus at different levels of external load. Time intervals between contraction peaks and their maximal amplitude were measured (Fig. 5). These two parameters are crucial for the transition of active muscle from incomplete to fused tetanus. Alterations in these parameters may indicate distinct characteristics of motor unit force generation, as sequential activation allows for finely tuned regulation of the total force produced by the whole muscle.

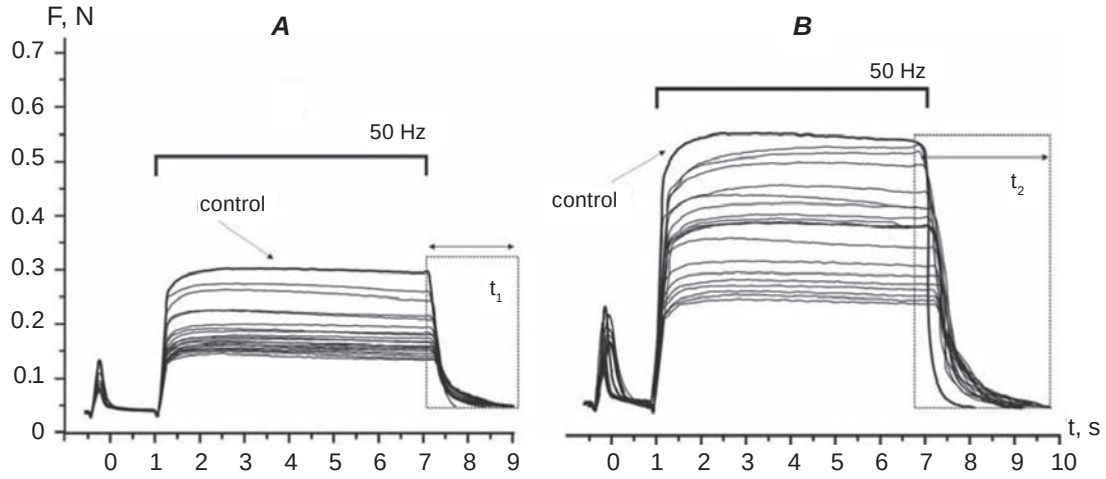

*Fig. 2. Musculus soleus mechanograms of a representative experimental animal from the Obesity group recorded during 20 consecutive non-relaxation electrostimulation signals at 50 Hz. Averaging was performed for 10-15 obtained force curves. Under the same conditions, no significant changes in the strength response were observed in control animals. **A** – external load 0.5 N; **B** – external load 0.25 N; control – contraction of musculus soleus in control animals; t_1, t_2 – time intervals representing the period required for the muscle to return to baseline tension after termination of stimulation*

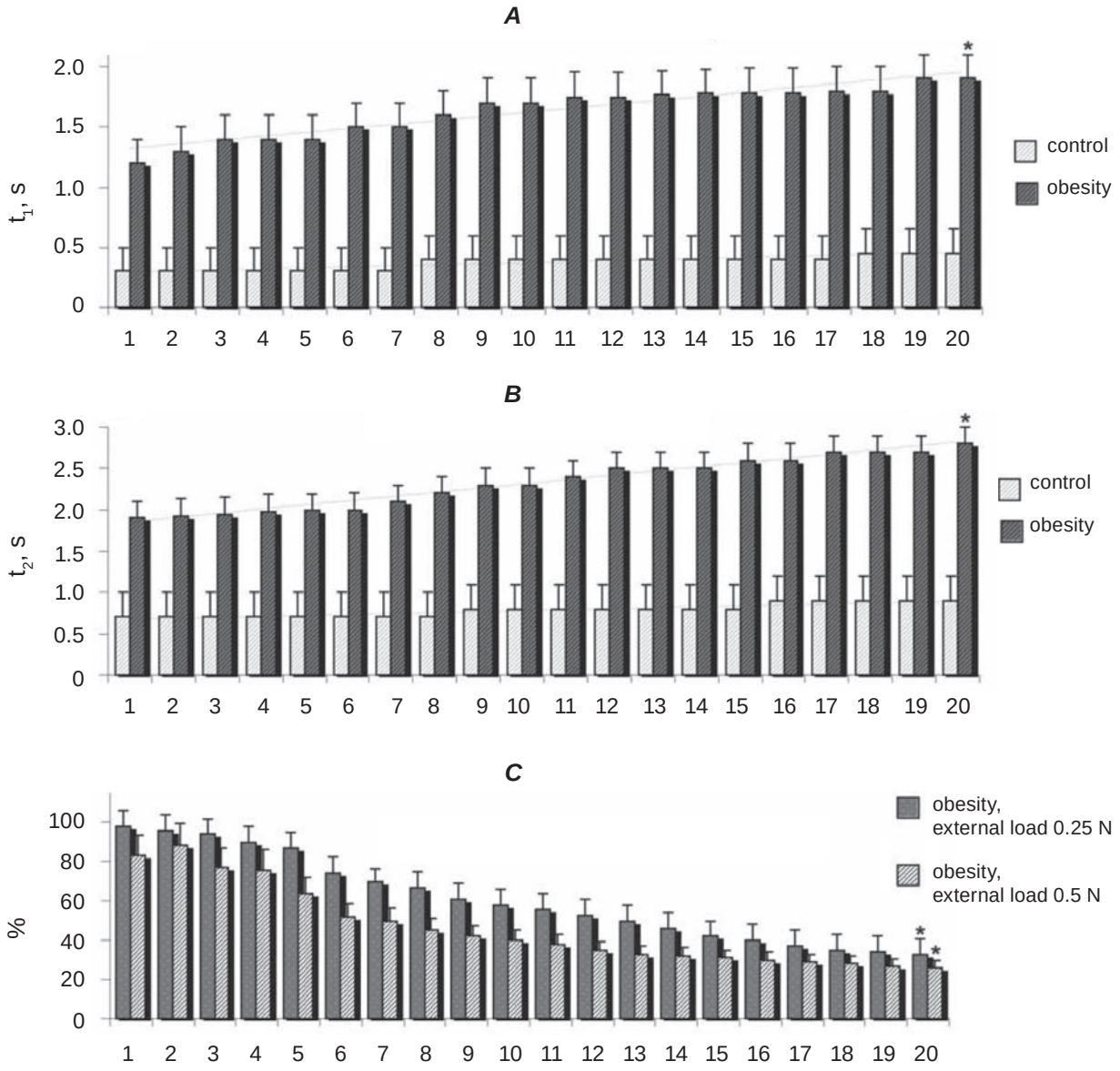

*Fig. 3. Time required for musculus soleus contraction to return to the initial state (**A** – t_1; **B** – t_2) and maximum contraction force expressed as a percentage of the control value (**C**) after non-relaxation stimulation at 50 Hz for 6 s with external loads of 0.25 and 0.5 N. Numbers 1-20 correspond to consecutive muscle contractions. *P < 0.05, significant difference compared with the Control group*

Response time fluctuations for the five maximal contractions decreased from 51 ± 4 to 36 ± 2 ms in control animals, leading to the development of fused tetanus after 3.36 ± 0.30 s from the beginning of stimulation (Fig. 5, *A*). In obese rats, this parameter changed from 55 ± 4 to 41 ± 3 ms, resulting in a prolonged period of complete tetanus formation – 3.59 ± 0.30 s. Such dysfunction could be caused either by an imbalance in Ca^{2+} influx/efflux or by an increase in non-contractile elements within the muscle tissue. Since Ca^{2+} pump dysfunction has not been widely reported in obesity, we assume that the observed contractile impairment results from increased muscle stiffness. An increase in the time interval between contractions under higher external load (0.5 N) to (57-46) ± 4 ms is in good agreement with this assumption.

Another piece of evidence supporting this hypothesis is the reduction in force response observed under obesity (Fig. 5, *B*). In control animals, the force difference was 355 ± 19 mN during the first contraction and 456 ± 29 mN during the fifth one. In obese

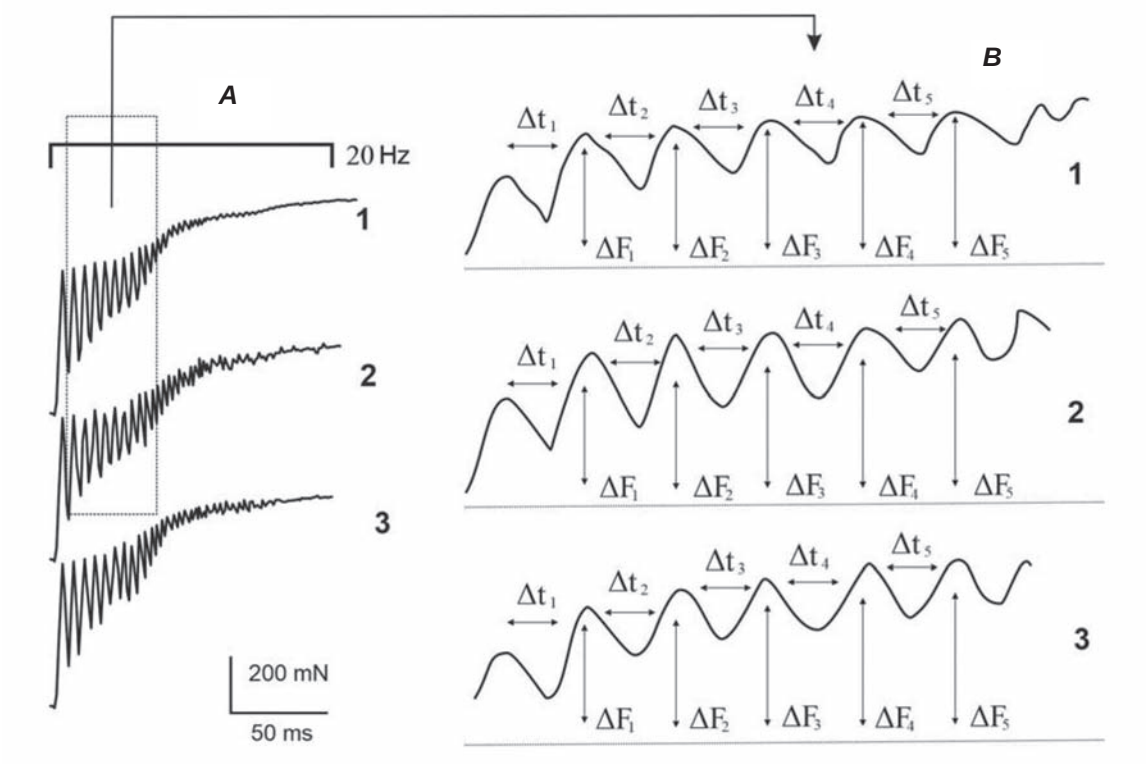

*Fig. 4. Typical curve of the musculus soleus contractile response to stimulation with impulses applied at 20 Hz for 6 s (**A**); development of incomplete tetanus during the first five peaks of the musculus soleus force response to 20 Hz stimulation in obese rats (**B**). 1 – contraction curves recorded in control animals; 2 – external load 0.25 N; 3 – external load 0.5 N; Δt_1 - Δt_5 – time intervals between sequential contraction peaks; ΔF_1 - ΔF_5 – amplitude of force generated during sequential contractions of musculus soleus*

rats, this parameter decreased to (340-425) ± 17 mN at an external load of 0.25 N and was further reduced to (331-375) ± 15 mN under an external load of 0.5 N. In this case, such a decrease in contraction force may reflect a compensatory mechanism associated with increased muscle stiffness.

Analysis of selected blood biochemical parameters after intense muscle stimulation can provide valuable insight into biochemical alterations occurring in active muscle tissue. The typical markers of fatigue development, a process leading to muscular dysfunction, include creatinine, CPK, and LDH [18].

In our study, we observed that following stimulatory muscle contractions, the blood creatinine concentration was elevated in the Obesity group by 50 ± 3% compared with the Control group (Fig. 6). The levels of both CPK and LDH were also significantly increased (by 20 ± 1% and 17 ± 1%, respectively) after the development of skeletal muscle fatigue in obese animals compared with controls.

Discussion

It is well known that obesity is associated with functional limitations in muscular efficiency and an increased risk of developing physical disability. Regardless of age, individuals with obesity exhibit greater absolute maximal muscular strength than non-obese ones, indicating that obesity acts as a chronic overload stimulus for antigravitational muscles, thereby increasing muscle volume [2, 5].

However, at the same time, there is a progressive destruction of muscle fibers and a decline in overall muscular performance in obese individuals [7, 8]. These alterations may be caused by reduced myocyte mobility, neuronal adaptations, and changes in muscle morphology. Another important factor influencing muscle contractility during obesity development is the enhanced lipid accumulation, which was demonstrated in our study (Fig. 1) as well as in others [3, 8, 9, 19]. It should be noted that fat possesses stiffer material properties than muscle tis-

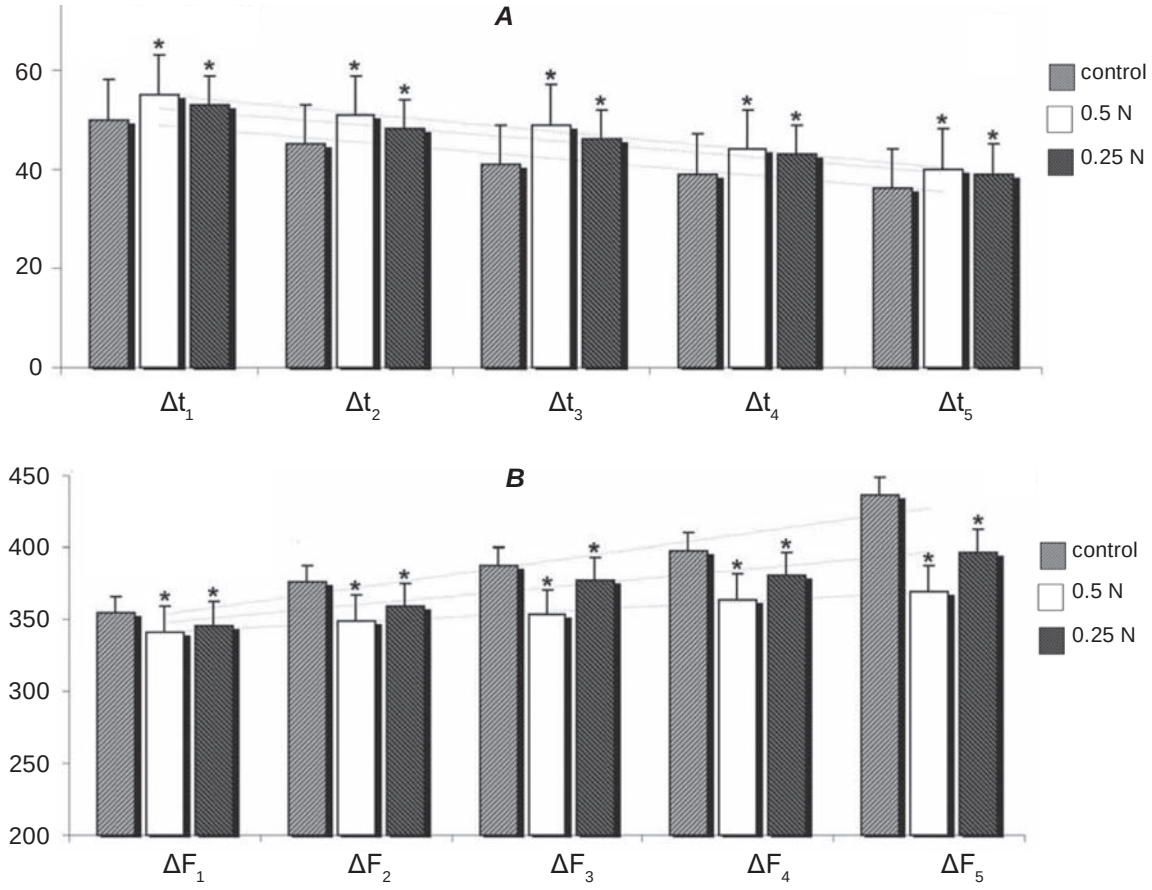

*Fig. 5. Time intervals (Δt_1 - Δt_5, ms) between maximal force responses of musculus soleus (A) and amplitudes of musculus soleus contractions (ΔF_1 – ΔF_5, mN) (B) during five sequential contractions forming incomplete (unfused) tetanus at 20 Hz stimulation. Control – control animals; 0.25 and 0.5 N – external loads of 0.25 and 0.5 N, respectively. *P < 0.05, significant difference compared with the Control group*

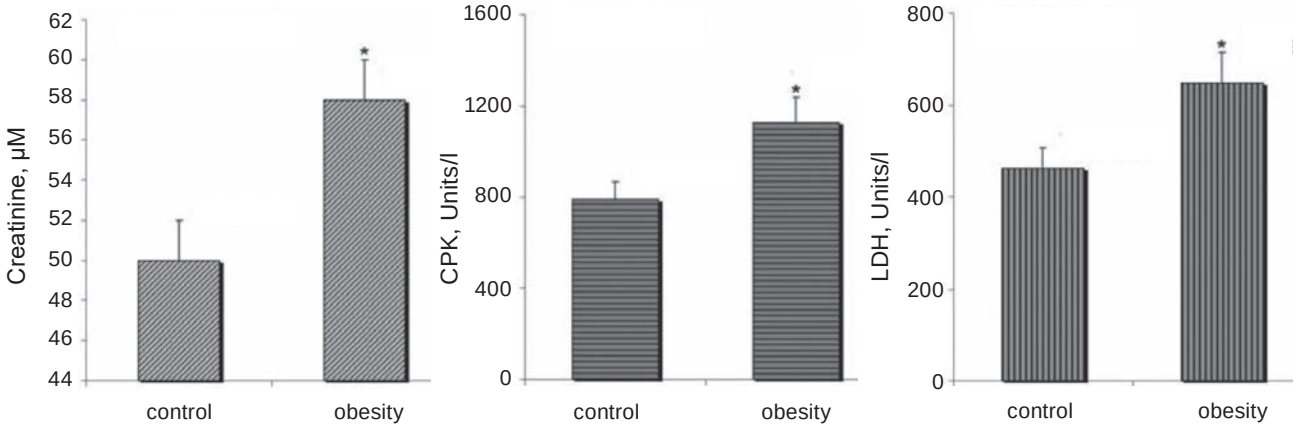

*Fig. 6. Creatinine concentration and levels of creatine phosphokinase (CPK) and lactate dehydrogenase (LDH) in rat blood after non-relaxation electrostimulation of musculus soleus at a frequency of 50 Hz for 6 s with an external load of 0.5 N. *P < 0.05, significant difference compared with the Control group*

sue [20]. Therefore, the accumulation of fat within skeletal muscles can lead to an increase in muscle stiffness, acting to resist muscle fiber shortening and transverse thickening during contraction.

Our findings revealed a lower muscle contraction force in obese rats (Figs. 2, 3) and altered overall skeletal muscle performance (Figs. 4, 5). These results may be directly related to increased muscle stiffness caused by the incorporation of fat components into muscle tissue. Moreover, our data indicate the development of muscle fatigue during obesity progression.

Previous studies have shown that, with the development of obesity, adaptive mechanisms, such as reorganized neuronal activation and modifications of muscle structural properties, may act to improve the efficiency of movements requiring maximal tension [21]. According to these data, muscle efficiency during high-intensity contractions depends on the effectiveness of force transmission from contractile elements to the skeleton, which may serve to reduce the pathological effects of increased muscular rigidity. Other studies have demonstrated that obesity can reduce skeletal muscle force generation due to elevated intramuscular lipid content, thereby disrupting the relationship between muscle mass and contractile function [22].

Research on the role of muscle fascia in maintaining steady-state muscle activity has shown that muscle fatigue itself is not the primary factor [23]. It has been suggested that disruption of fascial rigidity plays a crucial role in the development and maintenance of muscle tension, as well as in changes in interstitial pressure. This may explain our observation of a prolonged relaxation time and delayed return of the muscle to its initial position during obesity progression (Fig. 3).

An important aspect of muscle performance is the response to increasing frequencies of efferent input, which underlies the transition from incomplete to complete (fused) tetanus. The greater inertia of muscle contraction observed in obesity requires stronger stimulation from motor neurons. Therefore, the delay in achieving fused tetanus may represent a sensitive indicator of muscle pathology progression under obesity conditions. This rationale underlies our investigation of the specific transformation of *musculus soleus* descending activity in obese rats (Fig. 4).

It is also possible that the reduced contraction force observed in obese rats (Fig. 2) may be partially modulated by systemic inflammation [24], as adipose tissue functions as an endocrine organ secreting various cytokines. This is supported by increased levels of creatinine, CPK, and LDH (Fig. 6), which likely reflect the release of intracellular enzymes into the extracellular space following myocyte damage. This hypothesis is consistent with previous reports of elevated anti-inflammatory cytokines associated with obesity, particularly interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α) [24]. Such changes may also be related to increased muscle energy expenditure required to compensate for heightened stiffness of active muscle components.

It is also important to note that elevated serum levels of LDH, CPK, and creatinine in obesity could potentially reflect not only skeletal muscle fiber destruction but also systemic metabolic disturbances or mild renal dysfunction accompanying obesity. These enzymes and metabolites are not entirely specific markers of muscle fatigue; rather, they reflect broader cellular damage or altered energy metabolism. However, several studies indicate that under controlled experimental conditions, when changes in renal function or systemic inflammation are not pronounced, increases in these markers primarily originate from skeletal muscle stress or injury. For example, Goodpaster et al. [3] and Rahemi et al. [9] reported that intramuscular lipid accumulation and structural remodeling in obesity lead to mechanical strain and myocyte membrane leakage, resulting in elevated serum LDH and CPK. Moreover, elevated creatinine in diet-induced obesity has been interpreted as a byproduct of enhanced muscle catabolism rather than renal pathology [25]. Therefore, while alternative explanations cannot be completely excluded, we believe that the biochemical profile observed in our study most likely reflects muscle fiber disruption and metabolic fatigue rather than systemic renal impairment. This interpretation is supported by histological evidence of myocyte damage and fibrosis in the same animals.

Conclusion. It was established that during the development of obesity, the reduction in skeletal muscle strength generation is closely associated with increased intramuscular lipid accumulation, which disrupts the relationship between muscle mass and contractile function. *In vivo* studies have shown that a prolonged increase in external load on the muscles leads to adaptive hypertrophy of muscle fibers. However, the infiltration of intramuscular fat in-

duces structural and metabolic alterations in muscle tissue, resulting in marked disturbances in blood biochemical parameters and significant impairment of biomechanical indices of muscle activity. Thus, a comprehensive biomechanical analysis, including detailed evaluation of the force and time characteristics of muscle contraction, supported by histological and biochemical studies, can serve as an effective tool for assessing the degree of pathological alterations in the muscular system associated with obesity.

Conflct of interest. Authors have completed the Unified Conflicts of Interest form at http://ukrbiochemjournal.org/wp-content/uploads/2018/12/coi_disclosure.pdf and declare no conflict of interest.

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ПОКАЗНИКИ ПОРУШЕННЯ СКОРОТЛИВОЇ ЗДАТНОСТІ *MUSCULUS SOLEUS* У ЩУРІВ З ОЖИРІННЯМ

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Ожиріння набуло глобального поширення, досягнувши масштабів епідемії. Надмірна маса тіла є відомим фактором ризику розвитку порушень м'язової функції. Метою дослідження було оцінити механокінетичні параметри скорочення *musculus soleus* у тварин з ожирінням для кращого розуміння можливого впливу ожиріння на скоротливу активність м'яза, структуру тканини та появу маркерів ушкодження в крові. Експерименти проведено на 40 самцях білих нелінійних щурів, яких порівну розподілили на дві групи. Тварини контрольної групи протягом 10 тижнів отримували стандартний раціон. Щури з групи ожиріння утримувалися на високожировій дієті протягом аналогічного періоду. Наприкінці експерименту тварин піддавали евтаназії, ізолювали *musculus soleus*, після чого вентральні корінці відсікали від спинного мозку. Скоротливу активність м'яза досліджували за умов електричної стимуляції імпульсами, згенерованими імпульсним генератором. Гістологічний аналіз м'язової тканини проводили з використанням трихромного забарвлення за Ван-Гізоном та забарвлення суданом чорним. У крові визначали концентрацію креатиніну, а також активність креатинфосфокінази (КФК) та лактатдегідрогенази (ЛДГ). У тварин з ожирінням, порівняно з контрольною групою, виявлено зниження максимальної сили скорочення *musculus soleus* та імпульсу м'язової сили, подовження часу релаксації і сповільнене повернення м'яза до вихідного стану, що свідчило про розвиток втоми скелетного м'яза. Наявність у цитоплазмі міоцитів ліпідних включень і збільшення кількості колагенових волокон у тканині м'яза, а також підвищений рівень креатиніну й зростання активності ЛДГ та КФК у крові підтвердили порушення функціонального стану м'язової тканини у щурів з ожирінням.

Ключові слова: *musculus soleus*, ожиріння, м'язова втома, скоротлива активність, креатинін, креатинфосфокіназа, лактатдегідрогеназа, гістологічний аналіз.

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