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Oxidative phosphorylation in brown adipose tissue in a type II diabetes mellitus mouse model after forced treadmill running

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ABSTRACT

Aim. To study the effect of forced exercises on the content and parameters of oxidative phosphorylation in brown adipose tissue of mice with type II diabetes mellitus.

Materials and methods. To model the disease, we used a high-fat diet and physical exercises in the form of forced treadmill running for 4 weeks. The content of oxidative phosphorylation enzymes in brown adipose tissue was determined by Western blotting.

Results. Modeling diabetes in experimental animals was accompanied by expansion of adipose tissue. However, in brown adipose tissue, the content of all oxidative phosphorylation components decreases. Apparently, during type II diabetes mellitus modeling in mice, there is a decrease in the "energy efficiency" in brown adipose tissue, which is partially offset by an increase in its content in the body.

Regular physical activity in mice with type II diabetes mellitus, in contrast to healthy animals, contributes to a decrease in the content of brown adipose tissue. At the same time, the content of most oxidative phosphorylation components in brown adipose tissue increases, in some casesб it even exceeds the baseline values. The latter is typical of a variable load mode – when the execution time of exercises periodically changes.

Conclusion. The obtained results suggest that metabolic rearrangements in brown adipose tissue may serve as some of the mechanisms of preventive and projective effects of physical activity in type 2 diabetes mellitus.

Keywords: brown fat, running load, diabetes, obesity

Conflict of interest. The authors declare the absence of obvious or potential conflicts of interest related to the publication of this article.

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Окислительное фосфорилирование в ткани бурого жира у мышей с моделью сахарного диабета II типа после принудительных беговых нагрузок

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РЕЗЮМЕ

Цель: изучить влияние принудительных физических нагрузок на содержание и показатели окислительного фосфорилирования в ткани бурого жира у мышей с моделью сахарного диабета (СД) II типа.

Материалы и методы. Для формирования модели заболевания использовалась высокожировая диета, физические нагрузки в виде принудительного бега проводились в течение 4 нед. Содержание ферментов окислительного фосфорилирования в бурой жировой ткани определялось методом вестерн-блоттинга.

Результаты. Формирование диабетических расстройств у экспериментальных животных сопровождается возрастанием количества как белой, так и бурой жировой ткани. Однако в бурой жировой ткани при этом снижается содержание всех компонентов системы окислительного фосфорилирования. По-видимому, при формировании модели СД II типа у мышей происходит снижение «энергетической эффективности» бурой жировой ткани, что частично компенсируется увеличением ее содержания в организме.

Регулярные физические нагрузки у мышей с моделью СД II типа, в отличие от здоровых животных, способствуют снижению содержания бурой жировой ткани. В то же время при этом в буром жире возрастает содержание большинства компонентов системы окислительного фосфорилирования, в некоторых случаях – даже выше исходных значений. Последнее характерно для нагрузок, применяемым в переменном режиме – когда время выполнения нагрузок периодически изменяется.

Заключение. Полученные результаты позволяют предположить, что метаболические перестройки бурой жировой ткани могут служит одним из механизмов профилактических и проекторных эффектов физических нагрузок при сахарном диабете второго типа.

Ключевые слова: бурый жир, беговая нагрузка, сахарный диабет, ожирение

Конфликт интересов. Авторы декларируют отсутствие явных и потенциальных конфликтов интересов, связанных с публикацией настоящей статьи.

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INTRODUCTION

Brown fat (also known as thermogenic fat) is a type of adipose tissue whose main function is thermogenesis [1, 2]. Brown adipose tissue is capable of expending energy, unlike white fat, which stores it. As a result, brown adipose tissue has a regulatory effect on metabolism and may be involved in the control of blood glucose levels [3].

Patients with type II diabetes may have a lack of brown fat, which can lead to poor glucose management and an increased risk of complications. Some

studies suggest that stimulating brown fat storage may improve glucose management and reduce the risk of diabetic complications [4]. Ways to stimulate the growth of brown adipose tissue may include physical activity, consumption of certain foods, and the use of medications [5].

Physical activity of varying intensity launches a large number of biochemical, molecular, genetic, and epigenetic mechanisms that underlie the body's adaptive response to physiological stress [6, 7]. In particular, physical activity has been shown to have a positive effect on metabolic disorders [7, 8]. Animal experiments have shown that exercise increases insulin sensitivity and improves high-fat diet-induced glucose tolerance not only in the animals themselves, but also in their offspring [9]. Circadian rhythms have also been shown to influence the effects of exercise. Glucose uptake by muscles and insulin tolerance also have a circadian nature, and physical training does not affect the circadian rhythm of these parameters [10].

In connection with the above, the aim of the research was to study the effect of forced exercises on the content and parameters of oxidative phosphorylation in brown adipose tissue in a mouse model of type II diabetes mellitus.

MATERIALS AND METHODS

Male mice of the C 57bl/6 line were used as the object of the study. The mice were obtained from the vivarium of the Tomsk National Research Medical Center of the Russian Academy of Sciences, Goldberg Research Institute of Pharmacology and Regenerative Medicine. The age of the mice at the beginning of the experiment was 32 weeks. Animal keeping regime: 12 h / 12 h light / dark cycle, light cycle starts at 6 a.m., free access to food and water, room temperature 24 °C.

The study was conducted in accordance with the principles of the Basel Declaration and approved by the Bioethics Committee at the Biology Institute of Tomsk State University (Protocol No. 32 of 2.12.2019). The study was carried out in accordance with the principles of humanity set out in the European Council Directives (86/609/EEC) and the Declaration of Helsinki.

The experiment lasted 16 weeks. Until week 12, the mice were divided into 2 subgroups:

– animals receiving a high-fat diet – 28 mice.

– animals receiving a standard diet – 28 mice.

Type II diabetes mellitus (T2DM) and a high-fat diet for 12 weeks, developed specifically for this experiment, were used to model T2DM. The composition and energy value of the feed are described in detail in our previous work [11].

Starting from week 12 of the experiment, each group of animals was divided into two subgroups – those exposed (main group, n = 21) and those not exposed (control group, n = 7) to forced treadmill running.

Subgroups of mice from the main group were exposed to forced running exercise at different times of the day:

Group A – mice exposed to forced treadmill running during the light cycle (from 8:00 to 10:00), 7 animals;

Group B – mice exposed to forced treadmill running during the dark cycle (from 19:00 to 21:00), 7 animals;

Group C – the time of forced treadmill running alternated (alternating light / dark cycle): weeks 1 and 3 – in the dark cycle (from 19:00 to 21:00), weeks 2 and 4 – in the light cycle (from 8:00 to 10:00), 7 animals.

To normalize physical activity, the BMELAB SID – TM 10 treadmill for mice was used [12].

The animals had been exposed to forced treadmill running 6 times a week for 4 weeks. The duration of the exercise was gradually increased from 10 to 60 minutes during the first 6 days (an increase of 10 minutes per day) and did not change over the next 3 weeks. Every week, the elevation angle of the treadmill (from 0 to 10°) and its rotation speed (from 15 to 18 m / min) were changed. Once a week exercise was not performed (on the 7th day).

Body weight was measured using laboratory scales. Body weight of each animal was measured separately. Measurements were taken 11 times over 16 weeks.

The experimental animals were decapitated 24 hours after the last exercise. White and brown adipose tissue was extracted. The collected samples were weighed, then frozen in liquid nitrogen and stored in the freezer at –80 °C.

To homogenize the adipose tissue, 500 µl buffer per 50 mg of tissue was applied using the Vortex – Genie 2 laboratory mixer for 15 minutes at 4 °C with metal balls. Then the tissue was placed in the refrigerated mini rotary shaker for 1 hour, then left in the shaker rack in the cold for 15 minutes, and then again placed in the Vortex – Genie 2 laboratory mixer for 15 minutes at 4 °C.

After this, the tubes were transferred to a centrifuge at 4 °C and spun for 5 minutes at 8,000 rpm.

Then the lipid layer was carefully removed, placed again in the centrifuge for 15 minutes, after which clear supernatant under the remaining lipid layer was collected. Total protein in the sample was determined by the Bradford method.

Polyacrylamide gel electrophoresis was carried out under denaturing conditions according to the method described by Laemmil, with 5% stacking and 10% separating gels using the electrophoresis system (electrophoresis cell (Mini – PROTEAN Tetra Cell, USA), current source (PowerPacBasic, USA)). The amount of total protein applied to each well was 10 μg. Using the transfer system (Trans – Blot Turbo, USA), proteins were transferred from the gel to a PVDF membrane (Bio-Rad, USA) with further blocking with 5% skim milk (Bio-Rad, USA) in TBSt 1X (TBS supplemented with 0.1% Tween 20) for 1 h at room temperature.

Target proteins were determined by overnight incubation at 4 °C in 5% dry milk in TBSt with a 1:1000 dilution with rabbit polyclonal antibodies against citrate synthetase (cat. no. ab96600, abcam, UK), with rabbit polyclonal antibodies against hexokinase (cat. no. ab227198, abcam, UK), and a cocktail with antibodies Total OXPHOS Rodent WB (cat. no. ab110413, abcam, UK), containing 5 mouse antibodies, each against the subunits NDUFB8, SDHB, UQCRC2, MTCO1, and ATP5A. The sample was then incubated with secondary antibodies conjugated with horseradish peroxidase (anti-mouse, cat. no. 1706516, anti-rabbit, cat. no. 1706515, Bio-Rad, USA) for 1 h at room temperature in 5% dry milk in TBSt.

Antigen – antibody complexes were visualized using the ECL kit (SuperSigna West Dura, Thermo Scientific, USA) and gel documentation systems (ChemiDoc - It 2, UVP, UK). The densitometric analysis was performed using the ImageJ software. The Western blotting data were presented in relative units compared to the standard sample (the same sample was present on all blots). The values of the standard sample were taken as 100%.

The data were presented as the mean and the error of the mean ($M \pm m$). After checking the normality of data distribution using the Kolmogorov – Smirnov test, the results were analyzed using the two-way Kruskal – Wallis analysis of variance. Statistical processing of the results was carried out using the GraphPad Prism application package.

RESULTS

Figure 1 shows the dynamics of the body weight in the mice during the experiment. Already starting from week 4, a statistically significant increase in the body weight was observed in the mice receiving a high-fat diet ($p < 0.05$). The average body weight of the animals receiving a high-fat diet was 35.2 ± 2.0 g, and the average body weight of the animals receiving a standard diet was 32.7 ± 1.3 g. At week 12, the differences between the high-fat diet group (body weight 44.3 ± 2.6 g) and the standard diet group (32.2 ± 1.2 g) increased.

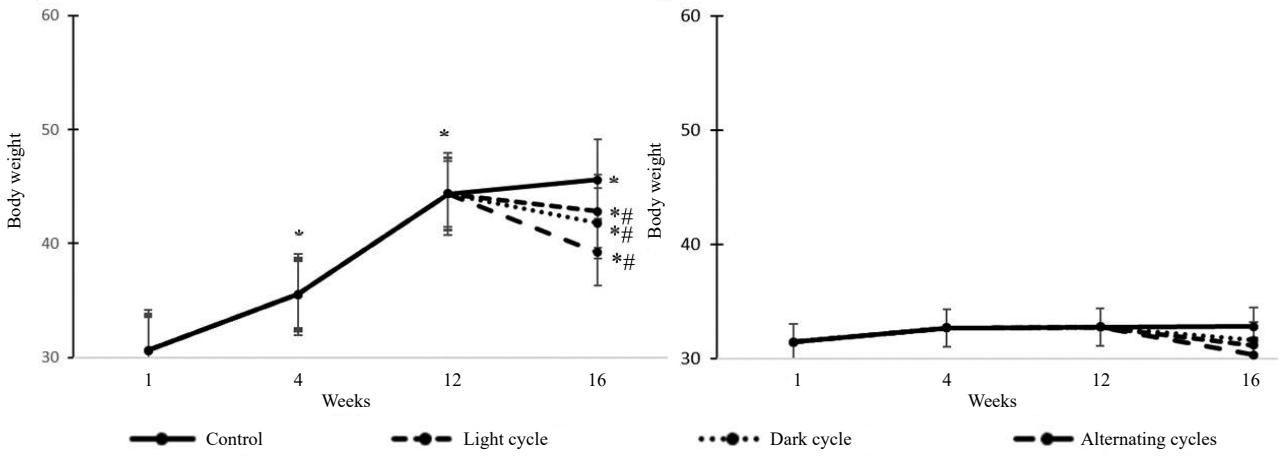

Fig. 1. Changes in the body weight of mice during the experiment: *a* – high-fat diet group; *b* – standard diet group. Here and in Fig. 2, 3, $M \pm m$, $n = 6$. * – statistically significant differences with the values at week 1 ($p < 0.05$), # – statistically significant differences with the group without physical exercises ($p < 0.05$)

Starting from week 12, both groups were divided into 4 subgroups, in which the animals were exposed to physical activity at different times of the day (light cycle, dark cycle, alternating cycles). At week 16 (final) of the experiment, we observed that in the high-fat diet group (45.6 ± 4.5 g) and standard diet group (32.8 ± 2.4 g), the difference in the body weight remained statistically significant ($p < 0.05$).

In the group receiving a fat diet, statistically significant differences ($p < 0.05$) in the body weight compared to the control group were observed in all 3 subgroups exposed to physical activity. The most effective was alternating exposure to physical activity (39.2 ± 4.4 g). In this group, the body weight was 1.2 times lower than in the control group.

Figure 2 shows the amount of abdominal white adipose tissue and brown adipose tissue in the mice after completion of the experiment. The increase in the body weight in the mice was largely due to an increase in the amount of white adipose tissue, but the content of brown adipose tissue in the body also increased significantly. It is important to note that forced physical exercise, while causing a decrease in the amount of white adipose tissue in all subgroups, had a much weaker effect on the content of brown adipose tissue. Only physical activity performed with a phase shift in the circadian rhythm helped reduce the amount of brown fat by half.

Figure 3 shows the results of determining the content of citrate synthase and OXFOS proteins in brown adipose tissue of the mice. The results are presented as a percentage of the control sample. As can be seen from the presented results, the content of citrate synthase in brown adipose tissue did not depend on the type of nutrition and increased slightly only when forced treadmill running was used during a phase shift in the circadian rhythm (Fig. 3, *a*).

ATP5A concentrations (Fig. 3, *b*) were reduced in the high-fat diet group compared to the standard diet group. Physical exercise in the high-fat diet group helped increase the content of this protein; the effect of evening exercise and training in the alternating regimen was more pronounced. In the standard diet group, on the contrary, physical exercise led to a decrease in the content of this protein.

MTCO1 concentration (Fig. 3, *c*) was significantly reduced in the mice fed with a high-fat diet compared to the standard diet group. Physical exercise in the standard diet group increased the content of this protein if applied in the alternating regimen.

The concentration of SD HB (Fig. 3, *d*) in the high-fat diet group decreased slightly compared to the standard diet group. Physical activity in the high-fat diet group increased the content of this protein. The effect of exercise was more pronounced during the dark cycle and alternating cycles. In the mice fed with a standard diet, on the contrary, physical exercise led to a decrease in the content of this protein.

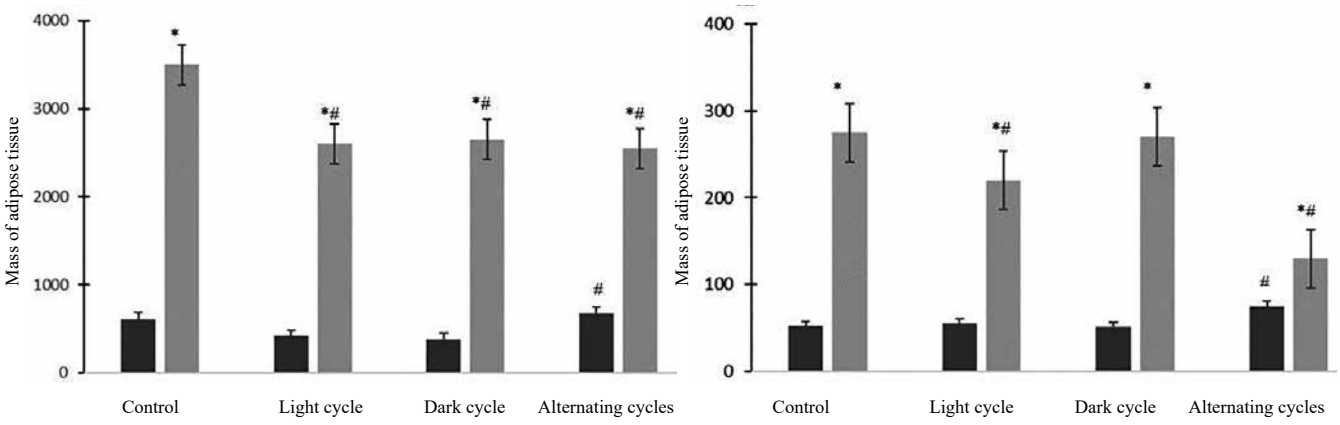

Fig. 2. Mass of white and brown adipose tissue in mice at 16 weeks of the experiment: light columns – high-fat diet group; dark columns – standard diet group. Here and in Fig. 3: * – statistically significant differences with the standard diet group ($p < 0.05$), # – statistically significant differences with the control group ($p < 0.05$).

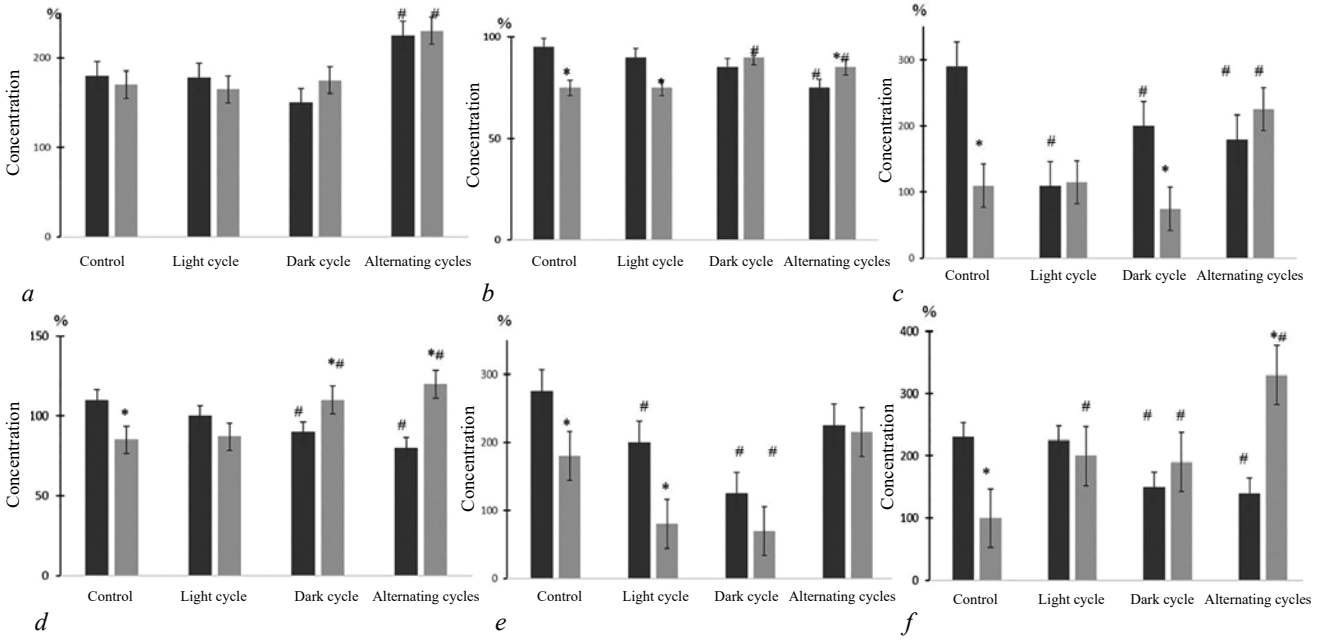

Fig. 3. Content of citrate synthase and OXFOS proteins in brown adipose tissue of mice: light columns – high-fat diet group, dark columns – standard diet group. Citrate synthase (*a*), ATP5A (*b*), MTCO1 (*c*), SD HB (*d*), UQCRC2 (*e*), NDUFB8 (*f*)

The concentration of UQCRC2 (Fig. 3, *e*) was reduced in the high-fat diet group compared to the standard diet group. Exercise in the high-fat diet group resulted in a significant a decrease when applied during the light or dark cycle, and in an increase when applied during the alternating cycles. In the mice fed with a standard diet, physical exercise led to similar, but less pronounced changes.

The concentration of NDUFB8 (Fig. 3, *f*) was reduced in the high-fat diet group compared to the standard diet group. Physical exercise in the high-fat diet group increased the content of this protein, especially if used in the alternating regimen. In the mice fed with a standard diet, on the contrary, physical exercise led to a decrease in the content of this protein.

DISCUSSION

The results obtained indicate that the use of a high-fat diet in mice leads to the increase in body weight and development of obesity (body weight was more than 25% greater than in the control group). Forced physical exercise in the form of daily treadmill running has a pronounced effect on metabolism in mice with a model of type II diabetes mellitus. First of all, this manifested itself by a decrease in the body weight of the animals and depended on the time of the day when the exercise was performed. Both gain and loss of body weight mainly occurred due to changes in the amount of white abdominal fat; the content of brown fat changed to a lesser extent.

At the same time, significant metabolic changes were observed in brown adipose tissue. Modeling type II diabetes mellitus was accompanied by a decrease in the content of all oxidative phosphorylation system (OXPHOS) components. The content of MTCO1 and NDUFB8 decreased to the greatest extent. Apparently, during the formation of a model of type II diabetes mellitus in mice, there was a decrease in the metabolic efficiency of brown adipose tissue, which was partially compensated by an increase in its content in the body.

The effects of exercise on brown fat levels have been somewhat controversial. According to the literature, regular physical activity helps increase the amount of brown fat in the body [13]. Thus, healthy young men who exercised daily for 12 weeks had more active brown fat than those who did not exercise [14]. We observed similar results in the mice without metabolic disorders fed with a standard diet – they showed an increase in the brown fat content after regular forced physical activity. However, in the mice with a model of type II diabetes mellitus, we observed the opposite effect – the content of brown fat in the body decreased with regular physical activity. This may be due to the fact that in these animals, during the development of pathology, the content of brown fat increased fivefold.

However, a decrease in the content of brown fat after exercise in the mice with a model of type II diabetes mellitus was accompanied by an increase in the content of most OXPHOS components; in some cases, their values even exceeded the baseline ones. The latter is typical of exercise applied in the alternating regimen – when the time of exercise performance periodically changes. Similar changes were observed for citrate synthase. Apparently, in metabolic disorders, the effects of physical activity can be realized not by increasing the amount of brown adipose tissue, but by improving its metabolic efficiency.

As mentioned above, promoting brown fat storage may improve glucose management and reduce the risk of diabetic complications [15]. Apparently, metabolic changes in brown adipose tissue may serve as some of the mechanisms for preventive and predictive effects of physical activity in T2DM.

CONCLUSION

The results obtained allow to draw several important conclusions.

Firstly, the development of diabetic disorders in experimental animals is accompanied by an increase in the amount of both white and brown adipose tissue. However, in brown adipose tissue, the content of all oxidative phosphorylation system components decreases. The content of MTCO1 and NDUFB8 decreases to the greatest extent. Apparently, during the formation of a model of type II diabetes mellitus in mice, there is a decrease in the metabolic efficiency of brown adipose tissue, which is partially compensated by an increase in its content in the body.

Secondly, regular physical exercise in mice with a model of type II diabetes mellitus, in contrast to healthy animals, helps reduce the content of brown adipose tissue. At the same time, the content of most OXPHOS components in brown fat increases, in some cases even above the baseline values. The latter is typical of exercise performed in the alternating regimen, when the time of exercise performance periodically changes. Similar changes are observed for citrate synthase.

The results obtained suggest that metabolic changes in brown adipose tissue may serve as some of the mechanisms for the preventive and predictive effects of physical activity in type II diabetes mellitus.

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Authors' contribution

Kapilevich L.V. – research supervision, conception of the study, editing of the manuscript, final approval of the manuscript for publication. Zakharova A.N. – conception and design, analysis and interpretation of the data, drafting of the manuscript. Milovanova K.G., Kollantay O.V., Orlova A.A., Shuvalov I.Yu. – collection and analysis of the data, processing and interpretation of the results.

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