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Effect of different temperatures on blood glucose level, glucose tolerance test, and insulin tolerance test in mice

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Abstract

Objective

Role of temperature on modulation of biological parameters has been increasingly documented on biological species, however, the temperature role on glucose biology and insulin resistance needs further challenging study. The objective of this study is to investigate the effect of different temperatures on the blood glucose level, glucose tolerance test (GTT), and insulin tolerance test (ITT).

Materials and methods

Seventy mice, aged five weeks, were acquired from the university animal house. They were split into two major groups (one to study the effect of different temperature on blood glucose level and second to study the glucose and insulin tolerance) with comparable weight distributions and given either a high-fat diet. Food was constantly available, unless too fast before the oral GTT. These animals will also be separated into four main groups, A1, A2, A3, and A4. Heat therapy is being administered to A1, A2, A3 and A4, with varying heating temperatures of 15°C, 25°C, 35°C, and 45°C. Each group has 7 mice. Prior to treatment, whole animals were fasted for sixteen hours. Its purpose is to measure the mice's blood glucose levels. Following a period of fasting, mice's blood was used to measure their blood sugar levels using an optoelectronic assessment of test strip device. Before the start of the GTT, mice were weighed and their blood glucose levels were tested after a fast. Blood glucose was tested at following the oral bolus of a 50% glucose solution (2.5 g/kg). Five mice were put in to this group.

Results

The p-value of 0.041, which is below the significance level of 0.05. For this reason, the difference is considered statistically significant. In brief, the outcomes of the paired samples analysis suggest that there exists a noteworthy distinction between control and high-fat diet (HFD).

Conclusions

In mice (Mus musculus), heating therapy can lower blood glucose levels and is an alternate treatment for Diabetes Mellitus Type 2. The ideal temperature for heating therapy is 40°C.

Keywords: Ambient temperature, Glucose, Heat stress, Insulin, Type 2 diabetes mellitus

Paper Type: Research Paper.

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Introduction

There are different forms of diabetes, but Diabetes Mellitus Types 1 and 2 is the most common kind in the general population. The autoimmune death of beta cells is the cause of type 1 diabetes. The majority of people with Type 1 diabetes are also afflicted with an autoimmune disease (Agahi & Murphy, 2013). According to two studies done ten years apart, there is a chance that the prevalence rate of diabetes may rise quickly in the urban population. According to this study, the number of people with diabetes mellitus has increased thrice during the past 20 years (Cockram et al., 2000). There are other forms of diabetes, but diabetes mellitus is the one that is most prevalent in the general population (Sangeetha et al., 2024). The autoimmune death of beta cells is the cause of type 1 diabetes. The majority of people with Type 1 diabetes are also afflicted with an autoimmune disease (Roep et al. 2021). Due to the impacts of the target tissue, type II diabetes, also known as non-insulin-dependent diabetes mellitus (NIDDM), has a decrease in insulin metabolic sensitivity. Insulin resistance will eventually result from decreased sensitivity to insulin. According to other investigations, an electric heating blanket heated to 41.5°C for ten to fifteen minutes (Chung et al. 2008). Heat shock proteins are stress proteins that are produced as a result of heating therapy. The amount of ROS and NO in skeletal muscle can rise with the use of heating therapy. The synthesis of HSP72 and HSP32 may result from this. Adiponectin, a kind of fat cell cytokine that helps to reduce insulin resistance, is synthesized in response to Hsp72 and Hsp synthesis 32 (Gupte et al. 2009). Insulin-induced glucose uptake can proceed because Hsp72 prevents the activation of JNK (June N-terminal kinase) receptors. This prevents insulin resistance from developing. Furthermore, Hsp32 will block JNK activation receptors. A concurrent rise in Hsp32 will result in an increase in the muscle-serving GLUT-4 glucose transporter. Muscle will absorb more glucose as a result of an increase in GLUT-4. Setting the amounts of glucose absorption by skeletal muscle achieves 80-90%. As a result, it is anticipated that heating therapy for skeletal muscle will affect the treatment of diabetes mellitus (Gupte et al. 2009). The researchers used the qualities of heating therapy to start using it as a simple, affordable, and efficient adjuvant therapy for patients with Type 2 Diabetes (Younis et al., 2022). The exact method of using thermal therapy is yet unknown, though. Thus, the purpose of this study is to assess the optimal temperature ranges for heating therapy in patients with diabetes mellitus. This study set out to evaluate the efficacy of heating therapy for lowering blood glucose levels. The objective of this study is to evaluate the effect of different temperature on lowering blood glucose level, and glucose tolerance. The process of exposing an organism to high temperatures is called heating therapy. The goal of heating therapy is to raise an organism's body temperature, which has an impact on the amount of free radicals (ROS and NO) present. In the first-ever heating therapy trial, mice were kept in a water bath at room temperature for 20 minutes, with the goal of raising their rectal temperature (Gupte et al. 2009). Because of their small size, which makes it possible to keep many of them at very low costs, and their genetic manipulation potential, mice are a preferred animal model for studying human diseases (Maloney et. al. 2014). Some mouse strains are very popular for investigating behavioral conditions because they are easily induced to become obese, which in turn causes impaired insulin sensitivity and non-insulin-dependent diabetes (Agahi & Murphy, 2013). There is a growing concern that mice kept in cages exhibit hypertension and increased food consumption, even though mice are commonly utilized in biomedical studies (Martin et al. 2010). This concern stems mostly from the current housing recommendations, which mandate that mice be kept at 21 to 25°C, a temperature far below their thermoneutral zone (TNZ), which is thought to be between 30 and 32°C (Maloney et. al. 2014). Owing to their high body surface to volume ratio and high total heat conductivity, mice have a significantly greater TNZ than humans. It is suggested by this that mice housed at specific temperatures have a significantly higher metabolism, which is accompanied by a higher heart rate. Elevated sympathetic tone causes tachycardia, while the TNZ is dominated by high parasympathetic drive, which is normal for mammals (Swoap et al. 2008). When there is a lack of food and the surrounding temperature drops (Ta), the laboratory mouse (Mus musculus) easily goes into a lethargy. Torpor is characterized by a regulated reduction in metabolic rate, core body temperature (Tb), and physical activity. It is a transient state involving the temporary suppression of metabolic functions (Melvin and Andrews, 2009). While periodic arousals separate multiday periods of hypometabolism in hibernating animals, the hypometabolic state and subsequent decrease in Tb remain for a full day in animals that use daily torpor. Moreover, body temperature acts as a key regulator of glucose metabolism, and temperature changes can lead to glucose hypometabolism, increased insulin, and insulin resistance in mouse models, which is induced through genetic circuits such as Qrfp neurons in the prefrontal cortex (Lee et al., 2025). The expression of thermogenesis genes, including Ucp1, Pgc-1α, and Dio2, in mouse brown adipose tissue is changed by temperature; for example, cold exposure upregulates thermogenesis genes and thereby increasing glucose uptake via Glut4, whereas high temperature downregulates their expression resulting in metabolic disorders. In mice deletion of insulin receptors with temperature changes in the maintenance environment (e.g., a shift from 22 to 30°C) were used as a model to study of insulin resistance and glucose metabolism (Shi et al., 2021; Hao et al., 2024). Finally, the integration of genetic markers with these environmental changes enhances our understanding the impact of temperature on glucose and insulin tolerance tests in animal models of diabetes and helps in the development of targeted therapies (Small et al., 2018). The present study highlighted the role of basic physiological impact of temperature on glucose metabolism in mice model.

Materials and Methods

Animal housing: Seventy mice (5 weeks age, ~200g weight) were kindly provided from the animal house of Tikrit University. The animal were housed at standard conditions (23°C and a 12:12 h light: dark cycle), and they were kept in groups of five and had unlimited access to water (Al-Thanoon and Merkhan, 2024; Halima et al. 2024). They were given a regular laboratory chow meal for the first week of their acclimatization. After that, they were split into two major groups (one to study the effect of different temperature on blood glucose level and second to study the glucose and insulin tolerance) with comparable weight distributions and given either a high-fat diet. Food was constantly available, except during fasting before the oral glucose tolerance test (GTT).

Study design: Male adult mice that have been hand-picked, approximately three months old, weighing roughly 20 grams, and in somatic and mental well-being with comprehensive morphology. Methods used in the simple random sampling research group allocation. These animals will also be separated into four main groups, A1, A2, A3, and A4. Heat therapy is being administered to A1, A2, A3, and A4, with varying heating temperatures of 15°C, 25°C, 35°C, and 45°C. Each group has 7 mice. Prior to treatment, whole animals were fasted for sixteen hours (Pederson et al. 2014). Its purpose is to measure the mice's blood glucose levels. Following a period of fasting, mice's blood was used to measure their blood sugar levels using an optoelectronic assessment of test strip device (Perlitasari et al., 2016). The mice were given 1 milliliter of prepared glucose solution, or as much high glucose chow as could be calculated. The mice were then given one hour to relax. Following an hour, the mice gave back blood that had been taken from the end of the tail to measure the glucose concentration of blood of the mice (Prieto et al. 2004). After that, mice received the proper care from each group for 20 minutes. The negative control group is called A1 Group. A container made up of wood that was sealed and had small air gaps was filled with mice. The room temperature in the city is indicated by the thermometer in the wooden box enclosure. There was no heating or cooling provided to this group. The temperature within the box adjusted to about 25°C, the outside temperature. Mice in the A2 group were housed in a container with small air openings sealed inside. The room temperature in the city is indicated by the thermometer in the box. Cooling is applied to the group until the temperature in the box reaches 15°C. A box of ice cubes can be distributed throughout the room by controlling the cooling process until the thermometer registers a temperature of about 15°C. The A3 mice were housed in a wooden box with small air openings that was sealed. The temperature of the room is indicated by the thermometer in the box. The temperature in this group was changed to match the mice's body temperature. The mice's body temperature was approximately 35°C. The room's heating bias was adjusted by applying targeted, low-power radiation until the thermometer registered about 35°C. A4 mice are housed in a sealed container with minimal ventilation holes. The temperature of the room is indicated by the reference thermometer in the box. The group was assigned a temperature of 45°C for heating. The temperature of the space can be adjusted to achieve a specific level of light power heating until the thermometer registers around 45°C. Following a 20-minute treatment period, the rectal temperatures of each mouse were taken right away. following another blood glucose measurement on the animals. Additionally, the experiment's outcomes were examined to see how heating therapy can lower blood sugar.

Body weight: The mice used for GTT had an initial body mass of 21.9 g, while the mice used for insulin tolerance test (ITT) and blood glucose level had an initial body mass of 20.5g. The GTT mice's body mass grew to 28.7 g on the control diet and 36g on the High fat diet after six weeks on the experimental diets. The mice utilized for ITT and blood glucose level had increases in body mass throughout the same period, reaching 26 g on the control and 33g on the High fat diet, respectively. Each group control GTT, HFD GTT, control ITT, and HFD ITT has 10 mice. A total of 40 mice used in this model.

Measurement of blood glucose and insulin: A tail tip incision was used to draw blood for glucose and insulin testing. The ultra-sensitive mouse insulin ELISA kit was used to assess plasma insulin. Measurements of blood glucose were taken with an Accu-Check Mobile glucometer (Hamed et al 2022).

Glucose tolerance tests: Before the start of the GTT, mice were weighed and their blood glucose levels were tested after a fast. Blood glucose was tested at following the oral bolus of a 50% glucose solution (2.5 g/kg). Five mice were assigned to this group. Blood glucose was measured 20, 40, 60, and 120 minutes later. Five mice were put in to this group (Mustafa and Al-Thanoon, 2024).

Insulin tolerance test: Following the mice's weight measurements, an intraperitoneal injection of insulin (0.05 UI/ml) at a dose of 0.5 UI/kg was administered. After injection, blood glucose was measured 20, 40, 60, and 120 minutes later. Five mice were put in to this group (Esmaeil et al. 2024).

Ethical considerations: Approval was obtained from the Northern Technical University (Iraq).

Results and discussion

The blood glucose level in A3 group 3 at 35°C 9.2±0.24 was significantly ($p<0.05$) higher as compared to all other groups. The blood glucose in A4 group at 45°C (39.3±0.2) was significantly ($p<0.05$) higher as compared to lower temperature exposed mice (15°C and 25°C) (Figure 1).

The GTT is significantly ($p<0.05$) reduced at all temperatures in HFD group as compared to control group. Moreover, in the HFD group, the GTT levels were significantly ($p<0.05$) lower in A2 (6.9±0.25), A3 (7.2±0.2), and A4 (6.9±0.24) as compared to A1(8.7±0.001). Similarly, in the control group, the GTT levels were significantly ($p<0.05$) lower in A2 (8.6±0.61), A3 (8.4±0.414), and A4 (8.5±0.17) as compared to A1(11.9±1.67). The ITT is significantly ($p<0.05$) elevated at all temperatures in HFD group as compared to control group. Moreover, in the HFD group, the ITT levels were significantly ($p<0.05$) lower in A2 (9.4±0.15), A3 (9.3±0.08), and A4

(9.6±0.16) as compared to A1(10.3±0.11). Similarly, in the control group, the GTT levels were significantly ($p<0.05$) lowered in A2 (7.2±0.25), A3 (7.4±0.207), and A4 (7.2±0.24) as compared to A1(8.7±0.001) (Figure 2).

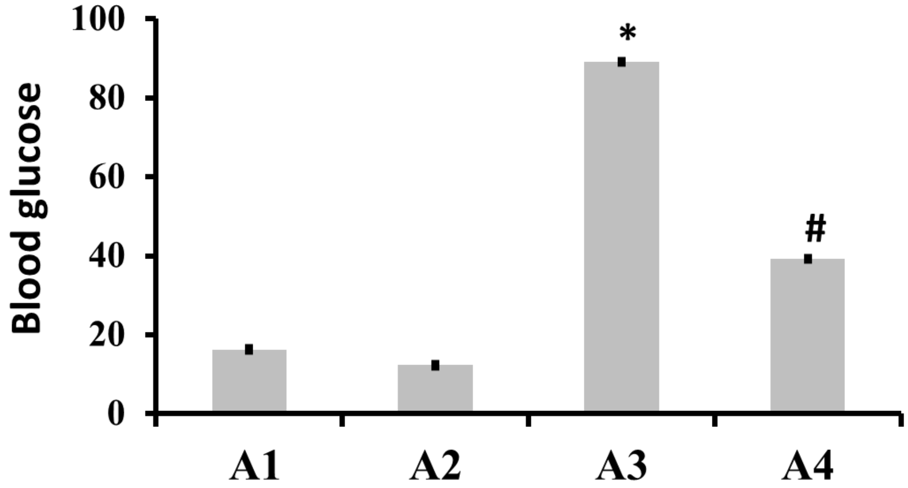

Figure 1. Effects of various temperature on blood glucose levels. Histogram bar indicates mean and standard deviation at different temperatures (A1=15°C, A2=25°C, A3=35°C, and A4=45°C). *# indicates significant differences at p value less than 0.05 using one way ANOVA test followed by Bonferroni test, *As compared to all other groups. # as compared to A1 and A2 groups

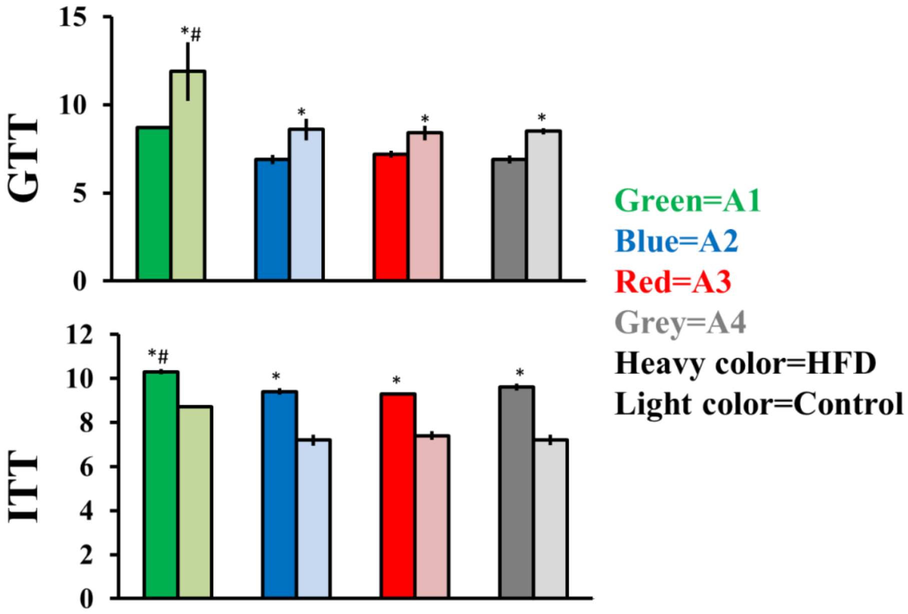

Figure 2. The GTT and ITT levels at various temperature in HFD versus control group. Histogram bar indicates mean and standard deviation at different temperatures (A1=15°C, A2=25°C, A3=35°C, and A4=45°C). *# indicates significant differences at p value less than 0.05 using one way ANOVA test followed by Bonferroni test, *As compared to HFD groups. # as compared to all other groups

Effect of Different Temperature on GTT And ITT: When compared to the control group, the effects of varying temperatures on fasting blood glucose levels before to the GTTs and IITs were significantly greater in the HFD animals. At 15C, 20C, 25C, and 30C, the p-value was 0.04. Nutrition and temperature have a big impact on fasting glucose. The mean difference between the paired samples is 1.72. The 95% confidence interval gives us 95% confidence that the true difference is between 0.13 and 3.31. The p-value of 0.041, which is below the significance level of 0.05. For this reason, the difference is considered statistically significant. In brief, the outcomes of the paired samples analysis suggest that there exists a noteworthy distinction between control and HFD. Fasting blood glucose during the tests. The blood glucose levels are measure in (mmol/L). The glucose tolerance test and insulin tolerance test is done at four different temperatures 15, 25, 35 and 45C. The mice were fed on high fat diet for six weeks in HDF group and standard diet in control group. The mean and SD of the data are displayed. A significant and noteworthy impact of temperature on insulin tolerance. Three blood glucose readings were taken, the first one coming after a 16-hour fast in the mice. the next half-hour following the mice's meal. Third, with temperature, thirty minutes after treatment. The homogeneity minutes are tested with fasting blood glucose levels. While the difference in the mice's blood glucose levels between when they were fed glucose for 30 minutes and after heat therapy is used to assess how much the mice's blood sugar levels decreased. The study revealed that the application of heating therapy significantly reduces blood glucose levels. The results of this experiment showed that treatment at 35°C (group A3) was considerably different from that at 15°C (group A1) and 25°C (group A2). It did not differ appreciably from the 45°C treatment (group A4). In the earliest heating therapy trials, mice were kept in a heated environment with a water bath for 20 minutes, or until their rectal temperatures reached 41°C. According to other investigations, an electric heating blanket heated to 41.5°C for ten to fifteen minutes (Chung et al., 2008). But based on the analysis's findings, these tests suggest that 37-40°C is the optimal temperature range for heating therapy to work. However, 37°C was the study's effective temperature. This leads to the conclusion that a temperature that doesn't exceed a person's body temperature can effectively lower glucose levels. It is thought that around 40°C, the body temperature will approach fatigue, verge on hyperthermia, and shorten the endurance period. Heating therapy is the process of subjecting an organism to high temperatures. The goal of heating therapy is to raise an organism's body temperature, which has an impact on the amount of free radicals present. This is one of the factors contributing to the drop in blood glucose levels. Furthermore, administering heating therapy to individuals with diabetes mellitus reduced blood glucose levels and enhanced insulin sensitivity. One is the protein kinase B (PKB)-mediated insulin signal transduction pathway, and these molecules' activity increases in response to heating therapy (Gupte et al. 2009). Heat shock proteins are stress proteins that are produced as a result of heating therapy. The amount of reactive oxygen molecules and Nitric oxide in skeletal muscle can rise with the use of heating therapy. The synthesis of HSP72 and HSP32 may result from this. Conversely, Hsp72 and Hsp32 levels are lowered in diabetes mellitus (Kurucz et al. 2002). As a matter of fact, this protein plays a protective function within the cell, assisting it in adjusting to environmental perturbations like those associated with diabetes. Heat shock proteins are a type of stress protein that are produced in response to heating therapy. ROS will enhance heat shock protein 72 transcription and activate HSF. Nitric oxide (NO) levels are raised by heating therapy, which is the second process. Nitric oxide triggers the Phosphokinase C (PKC) pathway, which in turn triggers HSF and raises the synthesis of ROS in Hsp72. By increasing the expression of Hsp72, insulin resistance can be prevented from occurring. According to Chung et al. (2008), heating therapy can help patients with reduced glucose tolerance and hyperglycemia. The cell's NO levels may rise as a result of heating therapy, here was also the existence of a reactive oxygen species, for instance, O_2 (Zuo et al. 2000).

Conclusion: In mice (Mus musculus), heating therapy can lower blood glucose levels and is an alternate treatment for Diabetes Mellitus Type 2. The ideal temperature for heating therapy is 40°C. HFD mice's GTTs are impacted by realistic room temperatures in labs, but neither the ITTs nor the control mice are affected. However, when undertaken at varying ambient temperatures, the results may vary among animal models (e.g., changed insulin production or reduced tissue insulin sensitivity) and consequently may lead to diverse conclusions.

Author Contributions

Conceptualization: MAMA; Methodology: HAH and GFM; Data analysis: GFM; Resources: HAH and GFM; Writing-original draft preparation: HAH, MAMA, and GFM; All authors have read and agreed to the published version of the manuscript.

Data Availability Statement

Available on request.

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Ethical Considerations

Approval was obtained from the Northern Technical University (Iraq).

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Conflict of Interest

The authors declare no conflict of interest.

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تأثیر دماهای مختلف بر سطح گلوکز خون، آزمون تحمل گلوکز و آزمون تحمل انسولین در موش‌ها

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چکیده

هدف: نقش دما در تعدیل شاخص‌های زیستی در گونه‌های مختلف به‌طور فزاینده‌ای گزارش شده است؛ با این حال، تأثیر دما بر زیست‌شناسی گلوکز و مقاومت به انسولین نیازمند بررسی‌های بیشتری است. هدف این مطالعه بررسی اثر دماهای متفاوت بر سطح گلوکز خون، آزمون تحمّل گلوکز (GTT) و آزمون تحمّل انسولین (ITT) بود.

مواد و روش‌ها: هفتاد موش پنج‌هفته‌ای از حیوان‌خانه دانشگاه تهیه شد. آن‌ها به دو گروه اصلی تقسیم شدند: گروه اول برای بررسی اثر دماهای مختلف بر سطح گلوکز خون و گروه دوم برای بررسی تحمّل گلوکز و انسولین. همه موش‌ها از نظر وزن همگن بوده و برخی از آن‌ها با رژیم غذایی پرچرب تغذیه شدند. غذا به‌طور دائم در دسترس بود، مگر در زمان ناشتایی پیش از GTT. حیوانات همچنین به چهار گروه اصلی A1، A2، A3 و A4 تقسیم شدند. برای هر گروه به ترتیب دماهای ۱۵، ۲۵، ۳۵ و ۴۵ درجه سانتی‌گراد به‌عنوان تیمار گرمایی اعمال شد. در هر گروه ۷ موش قرار داشت. پیش از تیمار، همه حیوانات به‌مدت ۱۶ ساعت ناشتا نگه داشته شدند تا سطح گلوکز خون آن‌ها اندازه‌گیری شود. پس از دوره ناشتایی، نمونه خون از موش‌ها گرفته شد و سطح گلوکز خون با دستگاه اپتوالکترونیک نوار تست اندازه‌گیری شد. قبل از شروع GTT، موش‌ها وزن شدند و سطح گلوکز ناشتا در آن‌ها

اندازه‌گیری شد. سپس محلول ۵۰٪ گلوکز به‌صورت خوراکی با دوز ۲/۵ گرم بر کیلوگرم تجویز شد و سطح گلوکز در زمان‌های مشخص اندازه‌گیری شد. پنج موش برای این مرحله استفاده شدند.

نتایج: مقدار p برابر ۰/۰۴۱ بود که کمتر از سطح معنی‌داری ۰/۰۵ است. بنابراین، تفاوت مشاهده‌شده از نظر آماری معنی‌دار تلقی می‌شود. به‌طور خلاصه، نتایج تحلیل نمونه‌های جفت‌شده نشان می‌دهد که بین گروه کنترل و گروه دریافت‌کننده رژیم پرچرب (HFD)تفاوت قابل توجهی وجود دارد.

نتیجه‌گیری: در موش‌ها (Mus musculus)، تیمار گرمایی می‌تواند سطح گلوکز خون را کاهش دهد و به‌عنوان یک روش درمانی جایگزین برای دیابت نوع ۲ مطرح است. دمای بهینه برای تیمار گرمایی ۴۰ درجه سانتی‌گراد تعیین شد.

کلمات کلیدی: انسولین، تنش گرمایی، دمای محیط ، دیابت ملیتوس نوع ۲، گلوکز

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