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Tryptophan enhances diet-induced improvement of white adipose tissue in obese rats

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Abstract: The study examined the effect of L-tryptophan on white adipose tissue and its potential to enhance the effectiveness of fasting-dietary obesity correction. Forty-eight Wistar rats were divided into four groups: I – control; II – animals that received a high-calorie diet for 12 weeks; III – rats that after 12 weeks of a high-calorie diet received a balanced diet for 4 weeks; IV – rats that after 12 weeks of a high-calorie diet received a balanced diet with the addition of L-tryptophan (80 mg/kg body weight) for 4 weeks. The high-calorie diet contained 45% fat, 9% protein, and 31% carbohydrate. Physiological, morphological, histomorphometric, biochemical, and biophysical methods were used in the experiment. A high-calorie diet induced obesity and structural alterations in white adipose tissue in the rats. Dietary L-tryptophan accelerated restoration of adipocyte composition, decreased connective tissue, improved white adipose tissue vascularization, and normalized visceral fat mass and lipid metabolism markers. L-tryptophan also contributed to a reduction in the disturbance of the bioelectric properties of the white adipose tissue in obesity. This may indicate restoration of adipocyte membrane integrity, adipose tissue hydration, and microstructural organization. The presented information may be applied clinically in the comprehensive treatment of obesity.

Keywords: balanced diet, L-tryptophan, obesity, rats, white adipose tissue

Abbreviations: HCD – high-calorie diet; WAT – white adipose tissue

INTRODUCTION

Obesity is one of the most prevalent diseases worldwide, representing a disruption to the body's overall energy balance and metabolic processes. It is characterized by the accumulation of excess fat deposits within tissues [1]. Obesity develops from multiple factors, including excess food intake, high caloric content, impaired energy metabolism, and low physical activity [2]. The social impact of obesity stems from its association with disability and reduced life expectancy due to frequent comorbidities. These include, but are not limited to, type 2 diabetes mellitus, arterial hypertension, dyslipidemia, atherosclerosis, and related diseases, reproductive dysfunction, gallstone disease, and osteoarthritis [3].

The primary methods of addressing obesity include dietary interventions, increased physical activity, and pharmacological and surgical treatments [4,5]. However, these methods do not always yield the desired result, necessitate long-term use, and frequently engender adverse effects [6]. Investigating alternative strategies to prevent, treat, or accelerate recovery from obesity-related dysfunction is a relevant area of research.

A treatment of alimentary obesity is fasting-dietary therapy based on the reduction of the calorie content of the diet (hypocaloric diet). However, such a diet is often ineffective, and a significant reduction in the calorie content of the diet is impossible for a variety of reasons (age, or the presence of concomitant diseases in patients) [7]. In this regard, the identification of potential novel methods and means to enhance the efficacy of a balanced diet in the context of obesity prevention is paramount. The findings of earlier research suggest that one avenue for further investigation may be the utilization of a dietary supplement comprising L-tryptophan [8].

L-tryptophan is a precursor of serotonin and melatonin, which are frequently employed in the treatment of obesity. An increased level of serotonin reduces stress, thereby contributing to a decrease in food intake and body weight. Serotonin activates gluconeogenesis, suppresses glucose uptake by the liver, and accelerates bile acid metabolism, which reduces calorie intake [9]. The concentration of melatonin in the blood is inversely proportional to the level of leptin and food intake. Melatonin decreases lipid peroxidation, suppresses hepatic lipogenic and fatty acid synthesis enzymes, and preserves mitochondrial respiratory function, preventing oxidative cell damage during a high-calorie diet (HCD) [10].

L-tryptophan supplementation exhibits protective effects against diet-induced obesity associated with metabolic complications in rats. Multiple studies have shown that L-tryptophan administration reduces visceral fat accumulation and obesity indices in rats fed HCD [11-13]. L-tryptophan effectively prevents fatty degeneration of the pancreas by reducing lipid and triglyceride concentrations in pancreatic tissue while preserving both exocrine and endocrine function [11]. This amino acid supports bone health by maintaining calcium and phosphorus levels and preserving femur biophysical properties in obese rats [12]. Additionally, L-tryptophan reduces brown adipose tissue whitening, a process associated with decreased metabolic activity in obesity [8]. While L-tryptophan normalizes body weight and prevents age-related decline in locomotor activity, it may increase anxiety-like behavior and impair long-term memory [13]. Obesity is associated with white adipose tissue (WAT) dysfunction, including pathological hypertrophy, fibrosis, impaired vascularization, immune cell infiltration, and ectopic lipid accumulation, resulting in chronic low-grade inflammation [14-16]. The efficacy of L-tryptophan in preventing and treating obesity remains unproven, and its impact on the repair of WAT altered by obesity has not been investigated.

In the present study, we investigated the effect of L-tryptophan on the state of WAT and its potential to enhance the efficacy of fasting-dietary interventions for the treatment of visceral obesity.

MATERIALS AND METHODS

Ethics statement

All experiments were approved by the Committee on Biomedical Ethics for the Care and Use of Animals of the O.O. Bogomoletz Institute of Physiology, NAS of Ukraine (protocol No. 5 dated 11/31/19). The rats were euthanized by decapitation under isoflurane anesthesia, in accordance with the European Convention for the Protection of Vertebrate Animals (Strasbourg, 1986).

Rats

The experiment comprised 48 male Wistar rats obtained from the vivarium of the O.O. Bogomoletz Institute of Physiology, NAS of Ukraine. The initial weight of the animals was 360±20 g. The rats were 3 months old at the commencement of the experiment. The rats were maintained under standard conditions and housed separately in cages with mesh partitions. They were randomly divided into four groups: group I – control rats that received a balanced vivarium diet; group II – rats that received a HCD for 12 weeks; group III – rats that after 12 weeks of a HCD were transferred to a vivarium diet for 4 weeks; and group IV – rats that after 12 weeks of a HCD were transferred to a balanced diet enriched with L-tryptophan (Ajinomoto Eurolysine S.A.S., France) for 4 weeks. L-tryptophan was administered to the animals in a daily dose of 80 mg/kg of body weight, with visual confirmation of complete food intake.

Modeling alimentary obesity

The experimental model of obesity in rats utilized a patented HCD [17]. According to this diet, experimental rats from groups II, III, and IV received HCD daily for 12 weeks, with a total daily calorie content of 116 kcal per animal. The composition of the diet, in addition to standard compound feed, included pork lard, white breadcrumbs, and sunflower seeds containing 45% fat, 9% protein, and 31% carbohydrate. Rats were administered a 10% fructose solution every other day in place of water, increasing caloric intake to 140 kcal and expediting obesity development.

After switching to a balanced diet, each rat in the control group and groups III and IV received 20 g/day

of standard feed containing 6% fat, 23% protein, and 55% carbohydrate (K120-1 Rezon-1, Ukraine). The daily calorie content of the diet was 66 kcal, with *ad libitum* access to water. Visceral obesity was assessed post-experimentally by measuring visceral fat weight and blood serum lipid parameters. Visceral fat was extracted from the abdominal cavity.

Assessment of lipids in blood serum

The concentration of total lipids, triglycerides, and cholesterol was determined in rat serum by the colorimetric-enzymatic method using standard reagent sets (Filisit-Diagnostics, Ukraine) on a biochemical analyzer (Sinnowa, China).

Histomorphological analysis of WAT

Samples of visceral WAT from the omentum were randomly selected, and histological preparations were made from the samples. All tissue samples were placed in Bouin's fluid, consisting of 75 mL of saturated picric acid solution, 25 mL formalin, and 5 mL glacial acetic acid, for 48 h. The samples were dehydrated in increasing concentrations of ethanol and embedded in paraffin blocks. Slices measuring 6 μm in thickness were prepared using a sledge microtome (MC-2, Austria). The staining of the sections was conducted in accordance with the Van Gieson method [18]. Analysis of the tissue samples was performed using a light optical microscope (Nikon Eclipse E100, Japan). Morphometric analysis was performed on micropreparations photographed with a Levenhuk T800 digital camera (USA) using ImageJ 1.34p software.

The following indicators were determined on the WAT micropreparations: relative area of the parenchyma, connective tissue and vessels, stromal-parenchymal index (ratio of relative area of vessels and connective tissue to parenchyma area), trophic index (ratio of relative area of vessels to parenchyma area), size (diameter and area) of adipocytes, area of adipocyte nuclei, density of adipocytes per unit area, thickness of adipocyte walls. Adipocytes were classified by diameter into three types: AI <50 μm, AII 50-100 μm, and AIII >100 μm. One hundred adipocytes were counted on different sections. The number of adipocytes of each type was expressed as a percentage of the total number of counted cells [19,20]. Histomorphometric analysis of visceral WAT was performed on 10 photomicrographs per rat at ×200 magnification.

Bioimpedance analysis

WAT bioelectric properties were assessed using multifrequency bioimpedance measurements [21,22]. Immediately following the isolation of visceral WAT, the samples were subjected to testing using the LCR Meter, Quad Tech 1920 (USA), in the operating mode of the device with a parallel equivalent circuit for a two-electrode installation. The frequency range utilized was from 100 Hz to 1 MHz, with a peak test signal voltage of 50 mV. Absolute impedance values were measured using two flat silver electrodes, each measuring 25 mm^2, with an interelectrode distance of 4 mm. Before and after each measurement, the electrode surface was cleaned using a piece of soft tissue and 70% ethanol. For analysis, impedance values at maximum (10^4 Hz) and minimum (10^6 Hz) polarization, and the phase angle at 10^4 Hz, were used. The results were used to calculate the impedance coefficient and its dispersion, expressed as the ratio of values at low and high frequencies (Z_{10^4}/Z_{10^6}).

Statistical analysis

Data were processed using variational statistics and presented as the mean (M)±standard deviation (SD). Experimental groups were subjected to one-way analysis of variance (ANOVA), followed by the Bonferroni t-tests for multiple comparisons. Statistical significance was defined as $P<0.05$.

RESULTS

Visceral fat weight

Rats that consumed HCD for 12 weeks (group II) exhibited visceral obesity, evidenced by a 2.4-fold increase in visceral fat weight. After switching to a balanced diet, the fat weight of the rats (group III) was 28% ($P<0.05$) higher than that of the control group. Group IV rats that received L-tryptophan in conjunction with a standard diet after HCD exhibited a 17% ($P<0.05$) reduction in visceral fat weight compared to the control group (Supplementary Table S1).

Biochemical study of lipid metabolism indicators in the blood serum

Obesity in group II rats was indicated by elevated serum lipid levels: total lipids +53%, triglycerides +80%, and cholesterol +37% versus controls. After exposure of rats to a balanced diet, the concentration of lipids rose by 21% (P<0.05). Administration of L-tryptophan in conjunction with a standard diet to animals exhibiting visceral obesity (group IV) restored all measured indicators to control levels (Fig. 1).

Microscopic examination of WAT

Visceral WAT in control and experimental rats comprised fat lobules and microvessels, separated by thin layers of loose connective tissue (Fig. 2). White adipocytes form the core of WAT fat lobules, each containing a single crescent-shaped nucleus displaced to the cell periphery. This shift is attributable to the presence of a single, large lipid droplet in the cytoplasm. The adipocytes within the groups exhibited divergent morphologies and dimensions. In the control animals, the adipocytes were predominantly oval, whereas in obese rats, they were often irregular and enlarged (Fig. 3).

Following a 12-week exposure to HCD (group II), significant structural changes were observed in the WAT. Visceral fat increase resulted from adipocyte hypertrophy, with the diameter by +51% and the area by +81% versus controls. This resulted in a 19% (P<0.05) decrease in adipocyte density. Adipocyte wall thickness decreased by 15% (P < 0.05) due to cell expansion. A substantial increase in connective tissue by 63% was observed in the WAT. Concurrently, the area of vessels and the trophic index decreased by 32% (P<0.05) compared to the control (Table 1, Fig. 3B).

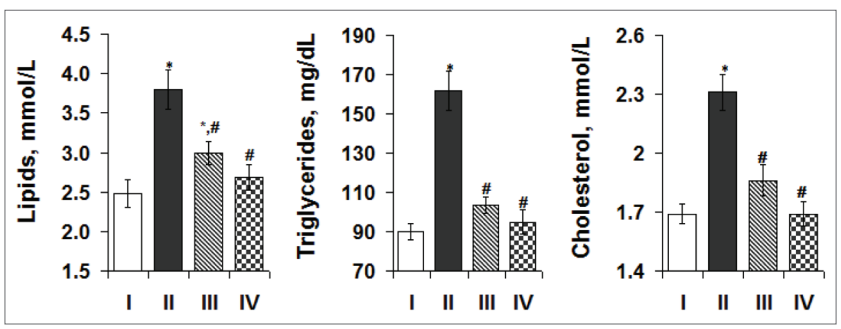

Fig. 1. Indicators of lipid metabolism in rat serum. *P<0.05 – significant differences compared to the group I; #P<0.05 – significant differences compared to the group II. I group – control; II group – animals that received a high-calorie diet (HCD) for 12 weeks; III group – rats that, after 12 weeks of HCD, received a balanced diet for 4 weeks; IV group – rats that, after 12 weeks of HCD, received a balanced diet with additional L-tryptophan for 4 weeks.

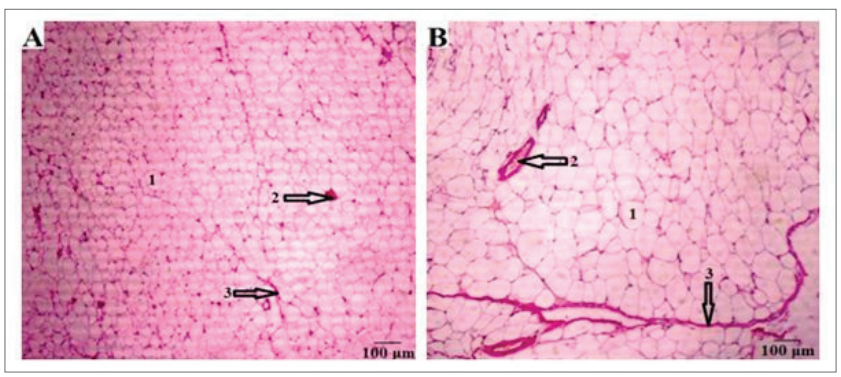

Fig. 2. Visceral WAT of a control rat (**A**), and a rat that received a high-calorie diet (**B**). 1 – hepatocyte; 2 – vessel; 3 – connective tissue that divides adipose tissue into lobules. Staining according to Van Gieson. Magnification ×100.

Rats switched to a balanced diet after HCD (group III) showed smaller changes in WAT. Adipocytes exhibited a slight increase in size compared to the control group, while the nucleus significantly increased by 34% relative to the control. Connective tissue area was 24% greater than controls (P < 0.05). Vascularization remained impaired, with vessel area 29% lower (Table 1).

In rats that received a balanced diet in combination with L-tryptophan after HCD (group IV), an 18% smaller adipocyte area was observed (P<0.05). The adipocyte density was significantly higher by 25% compared to the control group. This finding points to the possibility of an L-tryptophan-induced hyperplastic process in adipocytes. The area of the adipocyte nucleus was 43% larger (P<0.05), suggesting elevated levels of cellular activity. The connective tissue area was 20% larger than in the control group (P<0.05). Vessel area and trophic index matched control values, indicating WAT blood supply restoration (Table 1).

In WAT of group II rats after 12 weeks on HCD, A1 adipocytes (<50 µm)

Table 1. Histomorphometric parameters of WAT (n=12, M±SD)

Indicators	Control	High-calorie diet	High-calorie diet followed by a balanced diet	High-calorie diet followed by a balanced diet with L-tryptophan
Relative area (%): parenchyma connective tissue vessels	 90.3±2.4 4.1±0.3 5.6±0.2	 89.5±1.8 6.7±0.2* 3.8±0.1*	 90.9±1.2 5.1±0.3*# 4.0±0.4*	 89.7±1.6 4.9±0.2*# 5.4±0.2#
Adipocyte diameter, μm	44.9±1.3	67.7±1.1*	47.7±1.3#	41.1±1.3#
Adipocyte area, μm²	1999±104	3628±164*	2074±155#	1643±72*#
Adipocyte nucleus area, μm²	17.4±1.1	18.9±1.0	23.3±0.9*#	24.8±1.1*#
Density of adipocytes, no./mm²	291±11	235±12*	300±13#	364±11*#
Adipocyte wall thickness, μm	1.65±0.06	1.40±0.04*	1.50±0.03	1.60±0.09
Stromal-parenchymal index	0.107±0.002	0.117±0.005	0.100±0.002	0.115±0.005
Trophic index	0.062±0.001	0.042±0.002*	0.044±0.002*	0.060±0.003#

*P<0.05 – significant compared with the control; #P<0.05 – significant compared with the high-calorie diet.

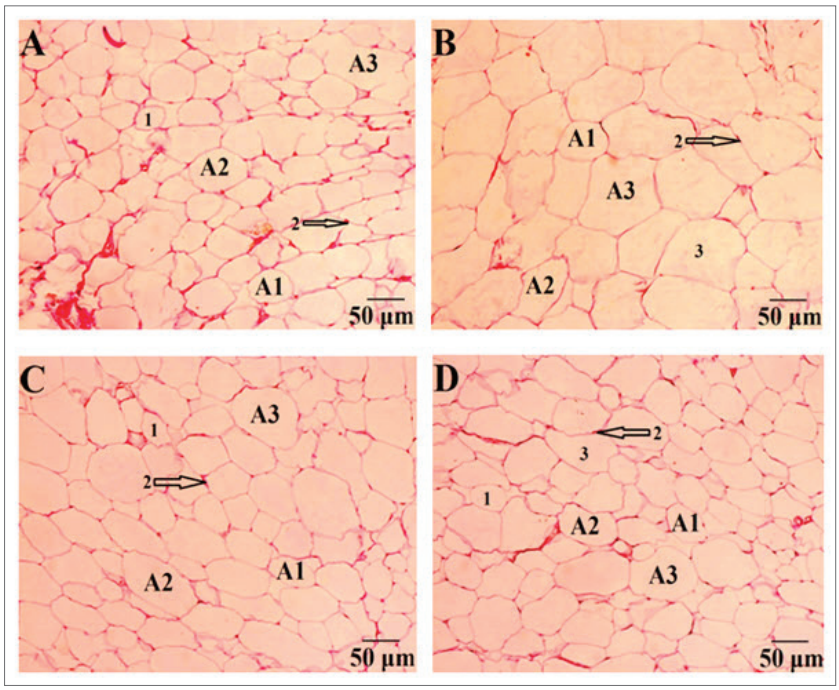

Fig. 3. Visceral WAT from a control rat (**A**), after high-calorie diet (HCD) exposure (**B**), a balanced diet post-HCD (**C**), and a balanced diet with L-tryptophan post-HCD (**D**). A1 – hepatocyte diameter <50 μm, A2 – hepatocyte diameter 50-100 μm, A3 – hepatocyte diameter >100 μm. 1 – oval-shaped hepatocyte; 2 – flattened nucleus, which is shifted to the periphery of the cell; 3 – hypertrophied hepatocyte of irregular shape. Staining according to Van Gieson. Magnification ×200.

decreased by 28%, A2 (50-100 μm) decreased by 30%, and A3 (>100 μm) increased 6.2-fold versus controls. In group II rats switched to a balanced diet after HCD, A1 adipocytes were 16% lower ($P < 0.05$), A2 increased 16%, and A3 rose 4.2-fold versus controls. In group IV rats receiving a balanced diet with L-tryptophan after HCD, A1 adipocyte numbers matched controls, A2 decreased by 18%, and A3 increased 2.8-fold (Figs. 3 and 4). Adding L-tryptophan to a balanced diet accelerated adipocyte composition recovery more than the diet alone.

Biophysical study of WAT

In control rats, WAT showed relatively low impedance values, consistent with tissue containing large lipid droplets and low hydration. The impedance dispersion coefficient ($Z10^4/Z10^6$) did not exceed 1.34±0.01, and the phase angle was 9.28±0.61° (Fig. 5).

In group II rats with alimentary obesity, impedance increased significantly at 10^4 Hz (+47%) and 10^6 Hz (+80%) versus controls ($P < 0.05$). The phase angle decreased by 21% ($P<0.05$), indicating impaired cell membrane integrity (Fig. 5).

After four weeks of dietary correction (group III), impedance values decreased relative to the untreated obese rats, reflecting partial recovery of tissue properties. However, values remained above controls: impedance +21% at 10^4 Hz, +42% at 10^6 Hz ($P<0.05$), with phase angle 17% lower (Fig. 5).

In rats with alimentary obesity that received a balanced diet supplemented with L-tryptophan (group

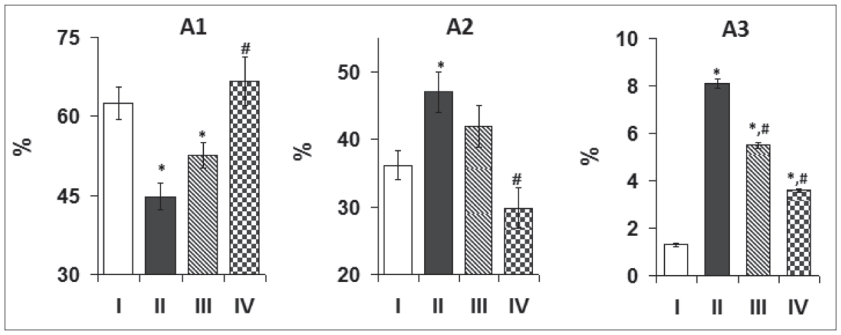

Fig. 4. Number of different types of adipocytes in white adipose tissue of rats. **A1** – diameter <50 μm, **A2** – diameter 50-100 μm, **A3** – diameter >100 μm. The number of cells of each type is presented as a percentage of the total number of counted adipocytes. *P<0.05 – significant differences compared to the group I; #P<0.05 – significant differences compared to the group II. I group – control; II group – animals that received a high-calorie diet (HCD) for 12 weeks; III group – rats that, after 12 weeks of HCD, received a balanced diet for 4 weeks; IV group – rats that, after 12 weeks of HCD, received a balanced diet with additional L-tryptophan for 4 weeks.

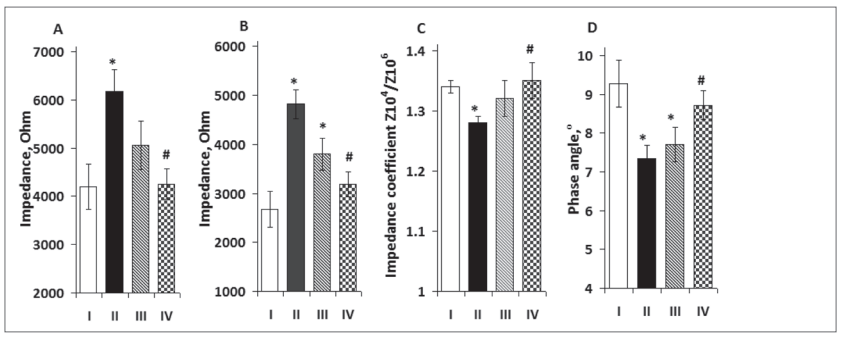

Fig. 5. Bioimpedance measurements of white adipose tissue in rats. **A** – impedance at a frequency of 10^4 Hz; **B** – impedance at a frequency of 10^6 Hz; **C** – impedance dispersion coefficient; **D** – phase angle. *P<0.05 – significant differences compared to the group I; #P<0.05 – significant differences compared to the group II. I group – control; II group – animals that received a high-calorie diet (HCD) for 12 weeks; III group – rats that received a balanced diet for 4 weeks after 12 weeks of HCD; IV group – rats that received a balanced diet with additional L-tryptophan after 12 weeks of HCD.

IV), WAT bioimpedance parameters did not differ significantly from those of control animals, indicating complete restoration of electrical properties (Fig. 5).

DISCUSSION

It is hypothesized that weight reduction in conjunction with dietary intervention enhances WAT functionality. Dietary interventions for the treatment of obesity are associated with beneficial outcomes, including improved endothelial function, reduced systemic inflammation and insulin resistance markers, and a decline in symptoms associated with the metabolic syndrome [23]. However, the treatment of obesity in conjunction with a diet necessitates a protracted period, with patients experiencing a gradual loss of weight and a high incidence of relapse [24]. Thus, identifying additional methods to enhance diet therapy, accelerate obesity treatment, and restore WAT function is a promising avenue. WAT is both a heterogeneous tissue and an endocrine organ regulating metabolic and immune homeostasis, making it a key target in obesity research [16].

In the present study, it was demonstrated that exposure of rats to HCD resulted in substantial alterations in the structure of the WAT. Adipocytes enlarged markedly, reflected by a more than 6-fold increase in cells with diameters >100 μm. A proliferation of connective tissue was observed, and impaired WAT blood supply. As has been demonstrated in obesity, hypertrophied adipocytes create localized micro-hypoxic zones in WAT due to increased oxidative stress and impaired mitochondrial respiration. Insufficient oxygen supply to WAT due to the reduced blood flow leads to the activation of hypoxia-inducible factor-1α (HIF-1α) [25]. Sun et al. suggested that existing hypoxic processes result in the accumulation of collagen and the development of fibrosis [26].

The findings of this study showed that adding L-tryptophan to a balanced diet after 12 weeks of HCD accelerated restoration of WAT structural and biophysical properties compared with diet alone. Results showed restored adipocyte composition, improved blood supply, and reduced connective tissue proliferation. L-tryptophan was more efficient in restoring obesity indicators (visceral fat weight, lipid, triglyceride, and cholesterol concentrations) to their normal levels.

Our results demonstrate that bioimpedance analysis can distinguish qualitative differences in adipose tissue between healthy and obese rats. In control rats, WAT displayed relatively low impedance values and stable dispersion. In obese rats, the impedance values increased,

while dispersion and phase angle decreased. These changes indicate impaired tissue electrical conductivity due to obesity-related morphological alterations, including adipocyte hypertrophy, expanded extracellular space, reduced microcirculation, and increased connective tissue [22]. A lower phase angle indicates reduced cell membrane integrity and number, reflecting diminished adipocyte functional activity [27].

After four weeks on a balanced diet, the impedance values of WAT decreased compared to untreated obese rats, indicating some recovery of tissue hydration and cellular function. However, notable differences from controls remained, showing that dietary correction alone did not fully restore adipose tissue morpho-functional state within this period. Of particular interest is the effect of L-tryptophan supplementation. In obese rats that received a balanced diet enriched with L-tryptophan, the bioimpedance parameters of WAT were comparable to those of control animals. The normalization of impedance values and the phase angle suggests that L-tryptophan enhanced the beneficial effects of dietary correction, promoting restoration of adipocyte membrane integrity, tissue hydration, and microstructural organization.

Previous studies have applied bioimpedance methods to distinguish between adipose tissue characteristics related to gender [28], type of fat (white, brown, beige), and anatomical depot (visceral, subcutaneous) [29], as well as changes during long-term storage and under external influences [30]. By extending these applications to obesity, our study highlights the potential of bioimpedance analysis as a versatile method for characterizing the structural and functional state of adipose tissue under different physiological and pathological conditions, and for monitoring the efficacy of interventions aimed at tissue recovery.

In our previous studies [20], rats received HCD for 12 weeks with L-tryptophan supplementation at 80 mg/kg body weight. L-tryptophan inhibited visceral fat accumulation and reduced serum lipid elevation in experimental rats. Furthermore, L-tryptophan attenuated the severity of the morphological and functional changes in WAT caused by obesity. Thus, L-tryptophan may reduce visceral obesity, demonstrating potential efficacy in lowering obesity risk when combined with HCD [20].

The findings of this study align with those of Shipelin et al. [13], who found that L-tryptophan at a dose of 250 mg/kg brought serum triglyceride concentrations closer to control values. In mice with alimentary obesity, oral L-tryptophan (1 mg/mL in water) reduced body weight, adipose tissue, and serum cholesterol, accompanied by lower inflammation [31]. L-tryptophan diluted in water to 0.4% and 0.8% suppressed appetite, reduced triglyceride levels, and increased lipolysis in piglets [32]. Studies have demonstrated the significance of L-tryptophan and its metabolites in regulating WAT function, showing that tryptamine, indoleamine 2,3-dioxygenase-1, and serotonin influence fat accumulation in adipocytes, lipid metabolism, and inflammatory responses [33-35]. Targeting L-tryptophan regulation may support novel therapies for obesity and related metabolic disorders, warranting further study.

Studying L-tryptophan and its metabolites in WAT during obesity offers new prospects for developing novel treatment approaches. This finding has the potential to transform current approaches in the prevention and treatment of obesity. Future research should explore how L-tryptophan and its metabolites affect WAT and brown adipose tissue at the molecular level, whether modifying gut microbiota can enhance its effects, the consequences of excessive or deficient intake, and the impact of systemic inflammation on tryptophan metabolism in obesity.

CONCLUSIONS

Twelve weeks on HCD induced visceral obesity in rats, evidenced by increased visceral fat weight and altered serum lipid parameters. The structural alterations observed in visceral WAT included hypertrophy of adipocytes, as well as disturbances in their qualitative composition, an increase in the amount of connective tissue, and a decrease in tissue vascularization. The inclusion of L-tryptophan (80 mg/kg) in a balanced diet resulted in a significant restoration of the qualitative composition of adipocytes. This was accompanied by reduced connective tissue, restored WAT blood supply, and normalized visceral fat weight and lipid parameters compared to diet alone. L-tryptophan also mitigated obesity-induced alterations in WAT bioelectric properties. These findings suggest potential

clinical applications of L-tryptophan for treating visceral obesity and restoring WAT structure and function.

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Author contributions: Both authors participated in the formation and design of this study. RY: methodology, software, validation, formal analysis, investigation, writing – original draft preparation. ML: conceptualization, methodology, validation, formal analysis, investigation, resources, data curation, writing – review and editing, visualization, supervision, project administration, funding acquisition. Both authors have read and agreed to the published version of the manuscript.

Conflict of interest disclosure: There is no conflict of interest to declare.

Data availability: The data supporting this article are available in the online dataset: https://www.serbiosoc.org.rs/NewUploads/Uploads/Yanko%20and%20Levashov_Dataset.pdf.

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SUPPLEMENTARY MATERIAL

Supplementary Table S1. Visceral fat weight, g (n=12, M±SD)

Control	High-calorie diet	High-calorie diet followed by a balanced diet	High-calorie diet followed by a balanced diet with L-tryptophan
18.5±0.8	44.4±1.8*	23.6±1.2*#	15.3±1.0*#

*P<0.05 – significant compared with the control; #P<0.05 – significant compared with the high-calorie diet.