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Concurrent Effects of High-intensity Interval Training and Mealworm on Oxidative Stress, Muscle Mass, and Hepatic Fat and Glucose Metabolism in Rats with Non-Alcoholic Fatty Liver Disease

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Abstract

Introduction: The present study aimed to evaluate the combined effects of aerobic exercise and Mealworm supplementation on reduced glutathione, hydrogen sulfide, lipid profile, glucose levels, and muscle mass in rats with non-alcoholic fatty liver disease (NAFLD).

Methods: 25 male Wistar rats were randomly assigned to five equal groups: healthy, patient, patient + supplement, patient + exercise, and patient + supplement + exercise. Non-alcoholic fatty liver disease was induced by a high-fat and high-cholesterol diet. High-intensity interval aerobic training was performed for 8 weeks, 30 minutes, 5 days a week, at an intensity equivalent to 50-80% of maximum oxygen consumption, in a progressive overload manner. Mealworm supplement was administered via gavage at a dose of 20 mg/kg body weight. Enzymatic concentrations of reduced glutathione and hydrogen sulfide in the soleus muscle, sarcomere length and count, and serum levels of triglycerides, cholesterol, low-density lipoprotein (LDL), high-density lipoprotein (HDL), glucose, and body weight were measured. Data were analyzed using independent t-tests and two-way ANOVA at a significance level of $P \leq 0.05$

Results: NAFLD significantly altered the studied parameters. Both aerobic exercise and Mealworm supplementation independently led to significant reductions in hydrogen sulfide, triglycerides, cholesterol, LDL, glucose, and body weight, along with significant increases in reduced glutathione, HDL, and muscle mass. The combined intervention produced significant improvements in all measured variables except sarcomere length.

Conclusions: Aerobic exercise combined with Mealworm supplementation may play a crucial role in reducing insulin resistance and managing NAFLD by improving oxidative stress status and regulating hepatic lipid and glucose metabolism.

Key Words: Aerobic Exercise, Mealworm, Glutathione, Lipid Profile Corresponding author:

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1. Introduction

Scientific evidence indicates that non-alcoholic fatty liver disease (NAFLD) is considered a metabolic disorder, as it is closely associated with metabolic abnormalities such as dyslipidemia, obesity, and type 2 diabetes. In recent terminology, this condition has been designated as metabolic dysfunction–associated fatty liver disease (MAFLD) (1). A recent study conducted in 2024 on 3,709 patients reported that the prevalence of MAFLD among individuals with NAFLD was 96.7%. A combination of various factors—including insulin resistance, oxidative stress, lipid peroxidation, mitochondrial dysfunction, endoplasmic reticulum stress, and other mechanisms—contributes to the development of NAFLD. An increase in hepatic triglyceride concentration represents the initial step in the onset and progression of this disease, ultimately leading to more advanced conditions such as liver cirrhosis (1). The fact that insulin resistance and compensatory hyperinsulinemia play a central etiological role in the development of metabolic syndrome and NAFLD highlights the excessive accumulation of triglycerides in the liver and its pivotal role in the onset of NAFLD and metabolic syndrome. The relationship between serum lipid profile and the occurrence of NAFLD is particularly notable in populations with obesity and metabolic disorders. Studies indicate that elevated levels of total cholesterol, low-density lipoprotein (LDL), and triglycerides (TG) are associated with the development of NAFLD. Likewise, lower levels of high-density lipoprotein (HDL) are linked to a higher incidence of NAFLD. Despite these findings, some studies suggest that the lipid profile alone may not be sufficient for diagnosing NAFLD, indicating the need for comprehensive assessments (2 Elevated levels of lipids in the serum and liver, on the other hand, lead to hepatic lipotoxicity, mitochondrial dysfunction, endoplasmic reticulum stress, and ultimately the production of reactive oxygen species (ROS). Oxidative stress in patients with NAFLD represents a major potential threat that can eventually result in hepatocyte death (1). Research indicates that exercise improves the lipid profile by reducing LDL, triglycerides (TG), blood glucose, body weight, and body fat percentage, while increasing HDL levels in inactive and obese men and women. The reduction in body fat in these individuals is accompanied by enhanced antioxidant capacity, decreased oxidative stress, and reductions in markers of muscle damage (3). Among the known endogenous antioxidants, glutathione is the most abundant compound and plays a primary role in cellular defense against oxidative stress. Its major functions include scavenging hydroxyl radicals, neutralizing hydrogen peroxide, and even regenerating cellular antioxidants such as vitamins E and C. Levels of reduced glutathione (GSH) are often decreased in patients with NAFLD, and this reduction is associated with disease severity. Treatment with this antioxidant can reduce liver enzymes and oxidative stress markers (4). In the context of managing oxidative stress in NAFLD, other factors—such as non-enzymatic hydrogen sulfide (H_2S) in the liver—may exert a complex and dual role in the induction of hepatic oxidative stress, acting both as a protective agent and as a detrimental factor in liver pathophysiology (5). At the molecular level, the toxic properties of H_2S are attributed to its inhibition of mitochondrial complex IV. Excessive H_2S may, under certain conditions, exacerbate oxidative stress (5). Moreover, evidence indicates that increased oxidative stress in NAFLD negatively affects skeletal muscle mass and function through multiple mechanisms. Specifically, reactive oxygen species (ROS) can induce muscle atrophy by activating pathways that enhance protein degradation, such as the ubiquitin–proteasome system (6). Furthermore, oxidative stress is associated with insulin resistance, which can exacerbate muscle loss by impairing glucose uptake and utilization in muscle tissues. This relationship creates a vicious cycle in which muscle loss leads to worsening metabolic health, which in turn further increases oxidative stress (7). In NAFLD, skeletal muscle dysfunction also leads to alterations in the myokine profile, which disrupts hepatic metabolism and increases oxidative stress. For example, myostatin, a negative regulator of muscle growth, is elevated in NAFLD and contributes to muscle atrophy, insulin resistance, hepatic lipid accumulation, and the exacerbation of oxidative stress (8). Exercise training, nutritional strategies, and antioxidant therapies are considered key approaches for improving muscle mass, reducing oxidative stress, and managing NAFLD. Low levels of physical activity, high-fat diets, and obesity are regarded as major contributors to the development of NAFLD, and exercise is likely to help treat the disease by counteracting the harmful effects of these behaviors and it was shown that high-intensity interval training (HIIT) has a beneficial effect on antioxidant levels and reduces oxidative stress in patients with NAFLD (9). In this regard, Evangelista (2022) demonstrated that eight weeks of aerobic training in NAFLD mice, in addition to significantly reducing body weight, glucose levels, and insulin resistance and increasing the activity of oxidative enzymes, was able to enhance the expression of genes involved in fatty acid oxidation in muscle tissue (10). In another study

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conducted in 2022, it was reported that six weeks of aerobic training in NAFLD mice led to significant improvements in the blood lipid profile (including TG, total cholesterol, and LDL), enhanced hepatic morphological status, and increased hepatic GSH content (11). In another study, Wang (2017) reported that a long-term 24-week period of aerobic training in NAFLD mice resulted in reductions in body weight, insulin resistance, impaired glucose tolerance, hepatic steatosis, and fibrosis, as well as increases in plasma and hepatic H_2S levels and the ratio of reduced to oxidized glutathione (12). Naghi-Zadeh (2023) reported that regular physical activity and exercise, through the release of catecholamines as an additional modulatory mechanism influencing oxidative stress, promote the upregulation of antioxidant defenses and consequently counteract lipid peroxidation processes (13). In addition to dietary modification, the use of certain nutritional supplements has been proposed as a strategy for managing fatty liver disease. Recently, supplements derived from edible insects, including mealworms, have demonstrated strong antidiabetic and antioxidant properties. Mealworm protein supplementation may influence the expression of genes involved in lipid metabolism and lead to reductions in body weight and fat mass (14). Given that current NAFLD treatments primarily focus on reducing body weight and fat mass, the use of mealworms with antioxidant properties may offer additional benefits in improving NAFLD. In this regard, Lee (2020) demonstrated that a 10-week administration of mealworm larval extract in high-fat-diet–fed mice significantly reduced hepatic fat accumulation, body weight, fat mass, serum glucose levels, lipid profile, and lipogenesis markers, while increasing hepatic antioxidant enzyme activity (15). Nevertheless, a recent report in 2022 indicated that a 4-week mealworm larva–based diet in growing pigs had no significant effect on hepatic, muscular, or plasma glutathione concentrations. Moreover, no meaningful changes were observed in the muscular or hepatic activities of the antioxidant enzymes glutathione peroxidase, SOD, and catalase (16). Although, in a study by Cho (2020), it was reported that a period of supplementation with hydrolyzed mealworm extract was able to reduce hepatic ROS levels by regulating the expression of genes involved in glutathione synthesis (17). Mealworm larvae also provide an accessible biological source of essential amino acids (EAAs), particularly leucine, which activates the mTORC1 pathway and stimulates muscle protein synthesis (18). In rodent models of obesity, mealworm protein supplementation increased lean mass by 12–15% compared with casein-fed controls, an effect attributed to enhanced anabolic signaling (19). While rodent studies are promising—for example, showing a 20% reduction in muscle atrophy markers in NAFLD models (18)—human trials remain limited. A pilot study reported improvements in muscle strength (greater than 8.5%) in older adults consuming high amounts of insect protein (20); however, overall, NAFLD-specific data remain scarce.

Overall, considering the role of mealworm protein supplementation (mealworm larvae) in improving lipid metabolism, insulin resistance, and antioxidant defense and, on the other hand, the effects of regular exercise in enhancing these same factors, it appears that these two interventions may potentially yield a more effective outcome when combined in the treatment of NAFLD. Since no study to date has examined the synergistic effects of these two interventions on oxidative status and hepatic lipid and glucose metabolism in NAFLD, the aim of the present study is to investigate the combined effects of aerobic training and mealworm protein supplementation on GSH levels, H_2S, the length and number of consecutive sarcomeres, and the lipid and glucose profile in rats with NAFLD.

2. Methods

2.1. Participants

Twenty-five adult male Wistar rats, eight weeks old, with an initial mean body weight of 257±4 g, were included in this study. The animals were obtained from the Pasargad Tissue and Gene Center and maintained under controlled environmental conditions, including a temperature of 22.4±1.4∘C, humidity of 55±4%, and a 12:12 h light–dark cycle. All animals had free access to water and standard rodent chow. After a two-week adaptation period, the rats were randomly assigned to five equal groups: healthy, NAFLD, NAFLD + supplement, NAFLD + training, and NAFLD + supplement + training. All procedures related to animal care and handling were conducted in accordance with the Helsinki ethical guidelines (1964) and were approved by the Ethics Committee of Islamic Azad University, Karaj Branch (IR.IAU.K.REC.1403.029).

2.2. Study design

This experimental and fundamental study was designed to investigate the effects of high-intensity interval aerobic training and mealworm extract supplementation on antioxidant markers, lipid and glucose metabolism, and sarcomeric characteristics in an animal model of non-alcoholic fatty liver disease (NAFLD). The NAFLD model was induced using a high-fat diet containing animal fat, cholesterol, and cholic acid, based on the protocol described by Wang (2004) (21), and the disease status was confirmed through biochemical indicators according to established diagnostic criteria (22). Following the induction of NAFLD, the aerobic training

intervention was implemented according to the protocol of Li et al. (2022) (23), and the mealworm extract supplementation was administered following previously reported procedures (24, 25). All experimental procedures were approved by the Ethics Committee of Islamic Azad University, Karaj Branch (IR.IAU.K.REC.1403.029) and conducted in accordance with the Helsinki ethical guidelines (1964).

2.3. Sample size calculation

Based on a paired-sample F-test power analysis, a minimum sample size of 25 subjects was required to achieve a statistical power of at least 0.80 with an alpha level of 0.05 and a medium effect size (d = 0.6). Accordingly, in the present study, a total of 25 adult male Wistar rats, aged 6 to 8 weeks, were selected as the statistical population.

2.4. Nutritional intervention / Supplementation protocol

To establish the NAFLD model, the diseased groups were fed a high-fat diet containing animal fat, cholesterol, and cholic acid, whereas the healthy group received standard rodent chow (AIN-93G), which provides approximately 18.8% of energy from protein, 16.4% from fat, and about 65.1% from carbohydrates. Based on the composition of standard rodent chow, the high-fat diet used for NAFLD induction was prepared according to the 12-week protocol described by Wang (2004) (21) and consisted of the standard rodent diet supplemented with 15% animal fat, 1% cholesterol (Sigma, USA), and 0.5% cholic acid (Sigma, USA). The high-fat diet was freshly prepared daily by the Pasargad Tissue and Gene Company and provided to the animals ad libitum.

To confirm NAFLD induction in the high-fat diet groups, blood samples were collected from the tail tip at the end of the feeding period and prior to the initiation of the main interventions. Biochemical parameters, including glucose, lipid profile (triglycerides, total cholesterol, low-density lipoprotein [LDL], and high-density lipoprotein [HDL]), and hepatic enzymes alanine aminotransferase (ALT) and aspartate aminotransferase (AST), were assessed as key diagnostic indicators of fatty liver disease. Elevated levels of these markers—serum ALT above 60 U/L, AST above 140 U/L, glucose above 160 mg/dL, triglycerides and total cholesterol above 200 mg/dL, LDL above 100 mg/dL, and HDL below 40 mg/dL—confirmed NAFLD induction (22). The mean body weight of the animals after model establishment reached 399.1±4.1 g.

2.5. Exercise protocol

Following the transfer of the animals to the laboratory, a two-week familiarization period (three sessions per week) was allocated for the exercise-training groups prior to receiving the high-fat diet. During this period, the rats in the training groups were accustomed to treadmill running at a speed of 10 m/min. One day before initiating the high-intensity interval endurance training protocol, a pilot test was conducted on five rats to estimate an appropriate VO_2max for the training program. Additionally, another five rats were selected for a pilot assessment of maximal running speed on the treadmill. For this purpose, a graded exercise performance test was performed at a 0° incline, beginning at 5 m/min and increasing by 2 m/min every 2 minutes until the animals were unable to continue running (exhaustion). In this test, a speed of 20 m/min was identified as the exhaustion threshold.

After determining the maximal running speed, the training groups performed the endurance interval training program according to the protocol of Li et al. (2022) for eight weeks, five sessions per week (23) (Table 1).

Table 1. High-Intensity Interval Aerobic Training Protocol

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Groups	Familiarization Period	Warm-up & Cool-down (min)	Main Training Duration (min)	Work Bouts per Session	Work-to-Rest Ratio (R:W)	Progressive Overload	Intensity (%VO_2max)
Diseased + Exercise	2 weeks	Running at 5 m/min for a total of 10 min	30 min	Week 1: Ten 1-min work bouts at 10 m/min with 2-min rest intervals at 5 m/min	1:2	Progressive overload applied to running speed during work :bouts Week 1: 10 m/min Week 2: 12 m/min Week 3: 14 m/min Weeks 4–8: 16 m/min	**50%** **60%** **70%** **80%**

2.7. Dietary control and physical activity standardization

Rats in the Disease + Supplement and Disease + Supplement + Exercise groups received the mealworm protein extract at a dose of 20 mg per kilogram of body weight, administered by gavage for 8 weeks, five sessions per week, scheduled in the morning hours, similar to the training days (24). The nutritional composition of this protein supplement, based on 100 g of dried mealworm, was as follows: moisture 5%, protein 50%, fat 28%, fiber 6%, carbohydrate 1.73%, iron 2.06%, and an energy value of 515.6 kcal per 100 g (25).

2.8. Laboratory methods

To obtain tissue samples, all animals were sacrificed under identical conditions 48 hours after the final training session to eliminate any acute effects of exercise. Euthanasia and surgical removal of the soleus muscle were performed in accordance with ethical guidelines for laboratory animal euthanasia and surgical procedures. Deep anesthesia was induced by intraperitoneal injection of ketamine (60 mg/kg body weight) and xylazine (5 mg/kg body weight) at a 5:2 ratio, ensuring that the rats reached a surgical plane of anesthesia and did not recover from it, thereby allowing pain-free tissue extraction and adherence to ethical euthanasia standards.

After confirming deep anesthesia, the thoracic cavity was opened, and 10 mL of blood was directly collected from the heart. The samples were centrifuged at 3000 rpm for 12 minutes, and the obtained serum was immediately transferred and stored at –80 °C.Serum glucose concentration was measured using the enzymatic glucose-oxidase method (Zist Shimi, Tehran, Iran) with an approximate sensitivity of 0.1–1 mg/dL, employing a digital spectrophotometer (Spectronic 20, USA). In addition, total cholesterol (sensitivity: 1–5 mg/dL), triglycerides (TG) (sensitivity: 0.8–2 mg/dL), LDL (sensitivity: 1–5 mg/dL), and HDL (sensitivity: 0.5–2 mg/dL) were quantified using the corresponding commercial kits (Zist Shimi, Tehran, Iran) according to the manufacturer's instructions.

Following blood collection, the soleus muscle was carefully dissected, rinsed with distilled water, weighed, and immediately frozen at –70 °C. The frozen muscle tissue was then pulverized in liquid nitrogen and homogenized in protease buffer (PBS, pH 7.4). The homogenates were centrifuged at 14,740 × g for 20 minutes at 4 °C. Tissue GSH concentration was measured using a rat-specific glutathione ELISA kit (MyBioSource, USA; Cat. No. MBS265966, sensitivity 0.5 µg/mL) according to the ELISA protocol. Tissue H_2S concentration was similarly quantified using a rat-specific hydrogen sulfide ELISA kit (MyBioSource, USA; Cat. No. MBS724525, sensitivity 0.1 µmol/L) following the manufacturer's instructions.

For histological evaluation of changes in sarcomere length and the number of consecutive sarcomeres, hematoxylin and eosin (H&E) staining was performed on muscle tissue sections. To prepare the hematoxylin solution, 1000 mL of distilled water was heated to boiling, removed from the heat source, and alum potassium powder was gently added. The mixture was reheated and stirred until fully dissolved. Separately, hematoxylin powder was dissolved in 50 mL of 96% ethyl alcohol. The temperature of the alum–water solution was then raised to approximately 85 °C, after which the hematoxylin solution was added and mixed. Mercuric oxide powder was subsequently

incorporated, and the container was rapidly cooled under running tap water while stirring with a glass rod. Then, 10 mL of glacial acetic acid was added, and the solution was allowed to stand for 24 hours before use. To prepare the eosin solution, 10 g of eosin powder was dissolved in 100 mL of distilled water, followed by dilution in 900 mL of 96% ethyl alcohol. Finally, 10 mL of glacial acetic acid was added. Using H&E staining, cell nuclei appeared blue, whereas the cytoplasm exhibited a pink coloration.

2.9. Statistical analysis

The collected data were processed and analyzed using SPSS software, version 24, with the level of significance set at $P < 0.05$. All results were expressed as mean ± standard deviation. The normality of data distribution was first assessed using the Shapiro–Wilk test. Subsequently, an independent t-test was applied to determine between-group differences (healthy control vs. diseased control) and to examine baseline assumptions. To evaluate the simultaneous effects of aerobic training combined with mealworm protein supplementation on reduced glutathione (GSH), hydrogen sulfide (H_2S), sarcomere length and number, lipid profile, and glucose levels in rats with non-alcoholic fatty liver disease, a two-way analysis of variance (two-way ANOVA) was performed.

3. Results

3.1. Participants' characteristics and compliance

The experimental animals consisted of male Wistar rats allocated to healthy control, diseased control, disease + supplement, disease + exercise, and disease + supplement + exercise groups. All animals completed the full duration of the study without mortality or major protocol deviations. Adherence to the training, supplementation, dietary standardization, and blood/tissue sampling protocols was high, with animals in the intervention groups receiving all scheduled exercise sessions and gavage administrations throughout the 8-week period. No adverse events related to aerobic training, mealworm protein supplementation, anesthesia, or tissue collection procedures were observed.

3.2. Primary outcomes

The descriptive findings of the study variables in the post-test phase for the research samples are presented in Table 2.

Table 2. Descriptive statistics of the post-intervention data presented as mean ± standard deviation

Groups	Healthy	Diseased	Diseased + Exercise	Diseased + Supplement	Diseased + Supplement + Exercise
LDL (mg/dL)	33.20 ± 8.04	242.4 ± 14.03	144.8 ± 7.52	163.8 ± 6.34	105.2 ± 8.16
HDL (mg/dL)	60.20 ± 3.34	39 ± 2.44	51.40 ± 3.50	49.2 ± 3.56	57.4 ± 1.34
TG (mg/dL)	53.40 ± 9.09	313.4 ± 12.4	157 ± 10.5	174.4 ± 9.04	108.8 ± 10.52
Total cholesterol (mg/dL)	93.40 ± 5.50	281.4 ± 13.37	162.6 ± 5.01	196.4 ± 2.5	214.2 ± 8.26
Glucose (mg/dL)	91 ± 4.63	174.2 ± 8.01	113.4 ± 3.27	130.8 ± 8.73	135.6 ± 1.51
Weight (g)	256.3 ± 3.8	399.1 ± 4.1	325.5 ± 6.1	348.5 ± 4.5	327.3 ± 9.9
GSH (mM/L)	8.33 ± 0.15	0.52 ± 0.06	1.86 ± 0.17	1.36 ± 0.11	4.53 ± 0.41
H_2S (mg/L)	1.01 ± 0.045	2.54 ± 0.061	1.78 ± 0.187	1.62 ± 0.088	1.19 ± 0.041
Sarcomere number	89,940 ± 1418.1	43,253.2 ± 2145.9	54,354.8 ± 4766.05	48,996.6 ± 3188.8	74,327.6 ± 1993.2
Sarcomere length (µm)	4.64 ± 0.20	1.44 ± 0.10	2.98 ± 0.24	1.96 ± 0.07	3.94 ± 0.11

3.3. Secondary outcomes

One of the assumptions of this study was that the development of NAFLD would influence the alterations in the measured variables in the experimental rats. To examine this assumption and determine the differences between the healthy and diseased groups, an independent t-test was employed. The results of this analysis are presented in Table 3.

Table 3. Independent t-test results for determining the differences between the healthy group and the NAFLD model

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Variable	p-value	df	t-value
GSH (mM/L)	0.000*	8	48.49
H_2S (mg/L)	0.000*	8	−44.67
HDL (mg/dL)	0.000*	8	11.43
LDL (mg/dL)	0.000*	8	−28.92
TG (mg/dL)	0.000*	8	−37.71
Total cholesterol (mg/dL)	0.000*	8	−29.07
Glucose (mg/dL)	0.000*	8	−20.09
Weight (g)	0.000*	8	−25.52
Sarcomere number	0.000*	8	18.15
Sarcomere length (µm)	0.000*	8	14.01

Indicates a significant difference compared with the healthy group ($P \leq 0.05$).

The results of the independent t-test assessing between-group differences showed that the levels of H_2S, LDL, TG, total cholesterol, glucose, and body weight were significantly increased in the diseased (NAFLD model) group compared with the healthy group. In contrast, the levels of GSH, HDL, sarcomere length, and the number of consecutive sarcomeres were significantly reduced in the diseased group relative to the healthy group ($P \leq 0.05$).

The results of the two-way ANOVA at a significance level of $P \leq 0.05$ for determining the between-group changes in the measured variables are presented in Table 4.

Table 4. Two-way ANOVA results

Variable	Group Factor	SS	df	F	p	Effect Size (η^2)
GSH (mM/L)	Exercise	25.38	1	93.66	0.0001*	0.85
	Supplement	15.31	1	56.49	0.0001**	0.78
	Exercise × Supplement	4.19	1	15.48	0.001***	0.49
	Error	4.33	16	—	—	—
H_2S (mg/L)	Exercise	1.77	1	146.07	0.0001*	0.90
	Supplement	2.82	1	232.09	0.0001**	0.93
	Exercise × Supplement	0.137	1	11.23	0.004***	0.41
	Error	0.195	16	—	—	—
LDL (mg/dL)	Exercise	30498.05	1	338.4	0.0001*	0.95
	Supplement	17464.05	1	193.8	0.0001**	0.92
	Exercise × Supplement	1901.25	1	21.1	0.0001***	0.56
	Error	1441.6	16	—	—	—
HDL (mg/dL)	Exercise	530.45	1	64.68	0.0001*	0.80
	Supplement	328.05	1	40.004	0.0001**	0.71
	Exercise × Supplement	22.05	1	2.68	0.121	0.14
	Error	131.2	16	—	—	—
TG (mg/dL)	Exercise	61605	1	537.09	0.0001*	0.97
	Supplement	43804.8	1	381.9	0.0001**	0.96
	Exercise × Supplement	10305.8	1	89.85	0.0001***	0.84
	Error	1835.2	16	—	—	—
Total cholesterol (mg/dL)	Exercise	23461.2	1	336.8	0.0001*	0.95
	Supplement	12650.4	1	181.62	0.0001**	0.91
	Exercise × Supplement	1394.4	1	20.02	0.0001***	0.55
	Error	1114.4	16	—	—	—
Glucose (mg/dL)	Exercise	5379.2	1	140.1	0.0001*	0.89

Variable	Group Factor	SS	df	F	p	Effect Size (η^2)
	Supplement	3920	1	102.1	0.0001**	0.86
	Exercise × Supplement	561	1	14.64	0.001***	0.47
	Error	614	16	—	—	—
Weight (g)	Exercise	11233.8	1	51.39	0.0001*	0.76
	Supplement	2976.8	1	13.61	0.002**	0.46
	Exercise × Supplement	3432.2	1	15.70	0.001***	0.50
	Error	3497.5	16	—	—	—
Sarcomere number	Exercise	1659167928.45	1	32.01	0.0001*	0.67
	Supplement	826653678.05	1	15.95	0.001**	0.50
	Exercise × Supplement	253094780.45	1	4.88	0.043***	0.23
	Error	829241548.00	16	—	—	—
Sarcomere length (µm)	Exercise	15.45	1	135.72	0.0001*	0.90
	Supplement	2.75	1	24.17	0.001**	0.60
	Exercise × Supplement	0.23	1	2.08	0.168	0.11
	Error	1.82	16	—	—	—

Indicates a significant main effect of aerobic exercise on the study variables; ** indicates a significant main effect of mealworm supplementation on the study variables; *** indicates a significant interactive effect of aerobic exercise and mealworm supplementation on the study variables; significance level set at $P \leq 0.05$.

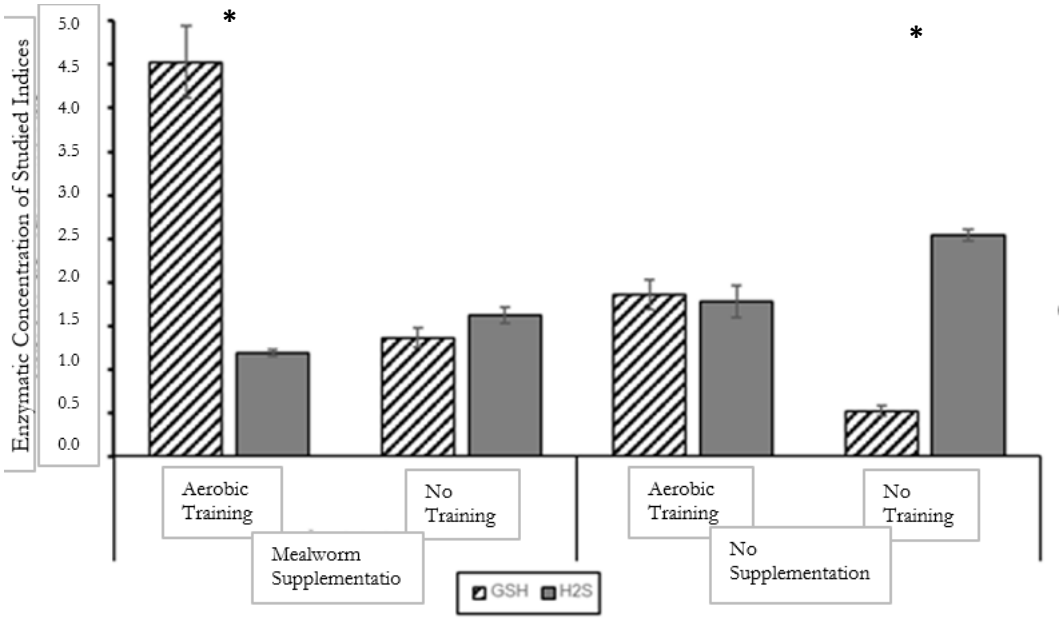

Figure 1. Changes in enzymatic concentrations of GSH and H_2S following the interactive intervention of high-intensity interval training and mealworm supplementation

4. Discussion

The findings of the present study demonstrated that the development of NAFLD in rats led to increased levels of H_2S, LDL, TG, total cholesterol, glucose, and body weight, along with decreased levels of GSH, HDL, and a reduction in both the number and length of consecutive sarcomeres. These results are consistent with previous studies (1, 5). Several molecular pathological mechanisms have been proposed in this regard. NAFLD is considered a consequence of insulin resistance, and elevated plasma glucose and fatty acid concentrations may enhance hepatic uptake and synthesis of fatty acids and triglycerides while impairing hepatic β-oxidation. Increased lipid levels induce hepatic lipotoxicity, mitochondrial dysfunction, endoplasmic reticulum

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stress, and ultimately the generation of ROS (1). Under such conditions, hepatic GSH levels are often reduced in NAFLD patients (4). Additional factors, such as non-enzymatic H_2S production in the liver, may exert a complex and dual role in hepatic oxidative stress. Elevated hepatic H_2S concentrations, as observed in NAFLD, competitively bind to complex IV of the mitochondrial electron transport chain, thereby increasing ROS production (5). Moreover, the collective evidence indicates that elevated oxidative stress in NAFLD significantly contributes to reduced skeletal muscle mass and impaired muscle function (6).

The main findings of the present study indicate that a period of endurance training and the administration of mealworm extract supplementation, each independently, resulted in significant increases in GSH and HDL levels, as well as in the number and length of consecutive sarcomeres. Moreover, both interventions led to significant reductions in H_2S, LDL, TG, total cholesterol, glucose, and body weight in rats with NAFLD.

In line with these findings, Evangelista et al. (2022) reported that eight weeks of moderate-intensity continuous aerobic training in NAFLD model mice resulted in significant reductions in body weight, glucose levels, and insulin resistance, along with increased activity of oxidative enzymes. Moreover, this intervention enhanced the expression of genes involved in fatty acid oxidation in the soleus muscle (10). Similarly, Rowan et al. (2022) demonstrated that six weeks of low-, moderate-, and high-intensity aerobic training in high-fat-diet–induced NAFLD mice significantly improved the blood lipid profile (triglycerides, total cholesterol, and LDL), hepatic morphology, and hepatic GSH content, with the most pronounced effects observed in the moderate-intensity training group (11).

In another study, Wang et al. (2017) showed that a prolonged 24-week period of moderate-intensity aerobic training in NAFLD mice led to reductions in body weight, insulin resistance, impaired glucose tolerance, hepatic steatosis, and fibrosis. Additionally, the intervention significantly improved hepatic mitochondrial function and β-oxidation, increased plasma and hepatic H_2S levels, and elevated the reduced-to-oxidized glutathione ratio (12).

Zhang et al. (2020) further reported that a 12-week combined exercise program substantially increased muscle mass (assessed via DEXA) while simultaneously reducing hepatic fat content (26).

Moreover, Lee et al. (2020) demonstrated that ten weeks of mealworm larval extract supplementation in high-fat-diet mice significantly reduced hepatic fat accumulation, body weight, fat mass, serum glucose levels, blood lipid profile, and lipogenesis markers, while enhancing hepatic antioxidant enzyme activity (15).

In a study by Cho et al. (2020), supplementation with hydrolyzed mealworm extract reduced hepatic ROS levels through the regulation of catalase expression and genes involved in glutathione synthesis (17).

Additionally, Han et al. (2024) showed that administration of mealworm extract in dexamethasone-induced muscle atrophy model mice significantly increased muscle strength and muscle fiber size (19).

Nevertheless, contrary to our findings, a recent study in 2021 reported that a four-week diet based on mealworm larvae in growing pigs did not produce any significant changes in hepatic, muscular, or plasma glutathione concentrations. Moreover, no significant alterations were observed in the activities of the antioxidant enzymes glutathione peroxidase, SOD, and catalase in either liver or muscle tissue (16). This discrepancy appears to be primarily attributable to differences in the pathological status of the experimental models, as the present study involved NAFLD-induced animals, whereas the referenced investigation was conducted on healthy, growing pigs.

Aerobic exercise plays a crucial role in improving lipid profile and insulin resistance in individuals with NAFLD through several molecular mechanisms. These mechanisms include the modulation of fatty acid and glucose metabolism, reduction of insulin resistance, attenuation of hepatic inflammation, and enhancement of mitochondrial function, all of which collectively contribute to improved metabolic health. Aerobic exercise, particularly interval-based training, increases the expression of peroxisome proliferator-activated receptor-γ coactivator-1α (PGC-1α), a key regulator of mitochondrial biogenesis. Through improved mitochondrial function, PGC-1α contributes to enhanced insulin sensitivity, increased fatty acid oxidation, reduced body weight, and improvements in hepatic steatosis, inflammation, and fibrosis (27).

The role of exercise in reducing oxidative stress via PGC-1α activation has also been highlighted. Moderate-intensity exercise activates the AMPK/PGC-1α pathway, which plays a central role in regulating cellular GSH content by influencing mitochondrial biogenesis and antioxidant gene expression. Several key mechanisms have been proposed through which exercise-induced PGC-1α regulates glutathione levels: PGC-1α upregulates multiple mitochondrial antioxidant genes, including manganese superoxide dismutase (SOD2), catalase, and glutathione peroxidase-1 (GPx1). This positive regulation enhances GSH synthesis. Additionally, improved mitochondrial function leads to better ROS management and increased GSH availability (28).

PGC-1α also influences the expression of enzymes involved in H_2S metabolism, such as ethylmalonic encephalopathy protein 1 (ETHE1), which is responsible for H_2S degradation. By stimulating the activity of these enzymes, PGC-1α helps maintain lower H_2S levels, thereby reducing the potential oxidative effects of H_2S on mitochondrial components (29).

Recent evidence further suggests that aerobic exercise can be expected to play a substantial role in improving muscle mass and function—reflected in sarcomere adaptations—depending on the intensity, duration, and type of aerobic training (30). In this regard, it has been shown that interval aerobic training not only stimulates muscle protein synthesis but also modulates inflammatory responses within muscle tissue, which may help prevent muscle atrophy and enhance recovery. Improved insulin sensitivity induced by such training supports better nutrient uptake and utilization in muscle cells, thereby promoting overall muscle health (24).

The molecular mechanisms through which mealworm larvae counteract conditions such as NAFLD and obesity—primarily by improving oxidative stress—include the modulation of antioxidant enzymes, enhancement of detoxification processes, and regulation of metabolic pathways. The beneficial effects of mealworm supplementation on lipid metabolism and oxidative stress, mediated through PGC-1α activation, have been highlighted in several studies (14). Incorporating mealworm larvae into high-fat diets has been shown to reduce adiposity and improve metabolic parameters, accompanied by increased PGC-1α levels.

Furthermore, consumption of mealworm larvae may influence signaling pathways associated with PGC-1α, mainly through effects on lipogenesis and lipid metabolism. Mealworm larval extract can inhibit adipogenesis in adipocytes by activating AMPK signaling and mitogen-activated protein kinases (MAPKs) (31). In addition, PGC-1α activation induced by mealworm supplementation may modulate the expression of antioxidant genes, enzymes involved in H_2S metabolism, and enhance the activity of H_2S-degrading enzymes—particularly ETHE1—while increasing cellular GSH content (28, 29). Improved mitochondrial function driven by elevated PGC-1α activity also plays a key role in enhancing cellular access to GSH (28).

Moreover, the presence of glutathione S-transferase (GST) in mealworm larvae contributes significantly to detoxification of harmful compounds and protection of cellular membranes against oxidative damage, thereby reducing oxidative stress (32). Mealworms are rich in protein and essential amino acids, which are critical for muscle protein synthesis. The amino acid profile of mealworm larvae is comparable to that of animal proteins and supports muscle growth and repair. Studies indicate that diets containing mealworm larvae improve nutrient digestibility in animals, leading to better absorption and utilization of nutrients for muscle development (33).

The additional findings of the present study indicate that the combined effect of exercise and supplementation significantly improved the measured variables; however, this interactive effect was not significant for sarcomere length. For sarcomere number, a slight but statistically significant increase was observed, although the effect size was small.

There is limited evidence regarding the concurrent effects of exercise and mealworm supplementation on lipid profile and oxidative stress status in metabolic disorders such as NAFLD. It appears that both interventions may contribute to improvements in lipid and glucose metabolism, insulin resistance, and oxidative stress through molecular mechanisms dependent on PGC-1α activation (14, 27). In addition, numerous studies have examined other mechanisms through which exercise influences NAFLD treatment. Exercise enhances metabolic cooperation between adipose tissue and skeletal muscle, improves hepatic regulation of lipid metabolism, and promotes overall metabolic health. Aerobic exercise increases adiponectin release from adipose tissue, which, by acting on other tissues—particularly skeletal muscle and liver—is associated with improved hepatic fat content and insulin sensitivity (34).

Conversely, mealworm larvae or edible insects may improve adiponectin levels in NAFLD or obesity through several mechanisms. These larvae have been shown to inhibit adipogenesis by activating AMPK signaling and mitogen-activated protein kinases, thereby reducing lipid accumulation and triglyceride content in adipocytes, which may indirectly influence adiponectin levels (31). Mealworms are also rich in unsaturated fatty acids and essential amino acids, contributing to their beneficial effects on metabolic health. These nutrients can enhance antioxidant capacity and improve lipid metabolism, potentially leading to increased adiponectin levels (35). Moreover, mealworm consumption has been associated with reduced body fat and improved lipid profiles in animal models—factors that may positively influence adiponectin levels. Overall, a combination of improved lipid metabolism, reduced lipogenesis, and enhanced antioxidant status may

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underlie the beneficial effects of mealworm supplementation on adiponectin levels in NAFLD and obesity (18).

Taken together, aerobic exercise alongside mealworm supplementation or mealworm-based diets may exert synergistic effects on improving lipid metabolism and oxidative stress in NAFLD through adiponectin-dependent pathways as well as PGC-1α-related mechanisms. However, further studies are needed to clarify the precise cellular mechanisms involved.

In contrast, given that in the present study the interaction between aerobic exercise and mealworm supplementation did not produce a statistically significant increase in muscle mass, several potential cellular mechanisms may be considered. Aerobic exercise is itself a strong activator of PGC-1α through multiple pathways, including calcium/calmodulin-dependent protein kinase (CaMK) and p38 MAPK signaling (36). Evidence also suggests that mealworm supplementation activates PGC-1α (31). Therefore, high-intensity interval aerobic training alone may be sufficient to maximally stimulate PGC-1α expression, leaving limited potential for additional enhancement through supplementation.

On the other hand, because mealworm larvae are rich in protein, many studies have proposed their use as an effective alternative protein source for stimulating muscle protein synthesis (33). Hermans et al. (2021) demonstrated that consuming smaller amounts of insect-derived protein in a meal increased muscle protein synthesis rates both at rest and during post-exercise recovery (37). Consequently, it is plausible that the essential amino acids present in protein-rich mealworm larvae may, unlike aerobic exercise—which strongly activates AMPK/PGC-1α signaling—partially interfere with this signaling axis (38).

5. Conclusion

Given that aerobic exercise and mealworm supplementation, both independently and interactively, produced favorable changes in lipid profile, blood glucose, and antioxidant and oxidative stress indices in NAFLD, it appears that moderate-intensity interval aerobic training combined with mealworm extract may play a substantial role in reducing insulin resistance and improving NAFLD through the enhancement of oxidative stress status and the regulation of hepatic lipid and glucose metabolism.

Considering the limited number of studies examining the interactive effects of exercise and mealworm supplementation on lipid metabolism capacity and their fundamental role in NAFLD treatment, further research is warranted. Therefore, it is recommended that future studies investigate similar interventions in models of obesity or type 2 diabetes, conditions closely associated with NAFLD.

6. Highlights

Given that aerobic exercise and mealworm supplementation, both independently and interactively, produced favorable changes in lipid profile, blood glucose, and antioxidant and oxidative stress indices in NAFLD, it appears that moderate-intensity interval aerobic training combined with mealworm extract may play a substantial role in reducing insulin resistance and improving NAFLD through the enhancement of oxidative stress status and the regulation of hepatic lipid and glucose metabolism.

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Conflicts of Interest:

The author declares that there is no conflict of interest regarding this article.

Authors' Contributions:

S. Paykari designed the study, conducted the experiments, and collected the data. A. Rahimi supervised the project, contributed to the methodological framework, and revised the final manuscript. F. Aghaei assisted in laboratory procedures and biochemical analyses. F. Feizolahi contributed to data interpretation.

All authors read and approved the final manuscript.

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