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Original article

Evaluation of the Effect of Moderate Physical Activity on Biochemical Markers of Glucose, Cholesterol, and Enzymes in Rodents

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Abstract
A comparative analysis was conducted to evaluate the effects of an aerobic exercise training program versus a sedentary lifestyle on biochemical markers of high-fat diet–induced metabolic dysregulation in male Wistar rats. Forty rats were randomly assigned to either a training group (n = 20), which performed treadmill exercise five days per week at moderate intensity (15–22 m/min), or a control group (n = 20), which remained sedentary. Blood samples were collected from all rats at baseline and after the 8-week intervention. Assessed parameters included fasting serum glucose, glycosylated hemoglobin (HbA1c), serum lipoproteins (total cholesterol, LDL, HDL, triglycerides), and hepatic and skeletal muscle enzymes (ALT, AST, creatine kinase, LDH). Statistical analyses were performed using paired and independent-sample t-tests (SPSS v26; α = 0.05). Significant reductions were observed in the training group for fasting glucose (−25.5%, $p < 0.001$), LDL (−22.1%), and triglycerides (−24.1%), alongside a significant increase in HDL (+35.4%). Improvements in liver enzymes (ALT: −32.8%, AST: −24.0%) and creatine kinase (+29.9%) were also noted within the physiologic adaptation range. No clinically relevant changes were detected in the control group. These findings suggest that structured, moderate-intensity aerobic exercise can mitigate diet-induced metabolic dysregulation in rodent models and provide preliminary evidence supporting the potential translation of these results to human populations for metabolic disease prevention.
Keywords. Biochemical Markers, Moderate Physical Activity, Wistar Rats, Glucose, Cholesterol.

Introduction

Metabolic syndrome and its range of disorders, such as Type 2 diabetes, dyslipidemia, and non-alcoholic fatty liver disease, is the most significant public health issues facing the world in the twenty-first century [1]. There is a compelling scientific case to increase investigations into how to non-pharmacologically prevent metabolic syndrome based on our knowledge of the underlying contributions to it: sedentary lifestyle, excessive caloric intake, and disordered glucose and lipid metabolism [2-7]. Although a great deal of research has been conducted on physical activity as an effective means of improving metabolic homeostasis, the specific mechanisms through which this occurs remain largely unknown.[2]

The use of rodent models (specifically, Wistar rats) in the laboratory to study the molecular underpinnings of the interactions between exercise and metabolism has been critical. The rapid life cycle, genetic manipulations, and catalogue of metabolic phenotypes enable us to perform precise experiments that could not be completed with human subjects due to both ethical and practical limitations [2]. Rodent models of metabolic disorders due to consumption of a high-fat diet produce many of the same physiological ramifications as humans who have metabolic disorders, including hyperglycemia, hyperinsulinemia, high LDL levels, low HDL levels, and high liver transaminase activity. Thus, rodent models provide a valid experimental laboratory environment for studying the effect of exercise on metabolic syndrome [3].

Evidence suggests that moderate intensity aerobic activity, defined as 40-60% of maximum oxygen consumption (VO_2max) in rodents, generally conducted using a treadmill running at 15-22 meters per minute, produces beneficial effects on insulin sensitivity through stimulation of GLUT-4 mediated glucose uptake, alteration of lipoprotein lipase activity, and upregulation of mitochondrial antioxidant enzyme activities [4]. Despite this evidence, no prior rodent trial has been able to quantitatively assess how an 8-week structured moderate intensity protocol influences all of the included biochemical parameters (glucose homeostasis markers; lipid fractions; hepatic enzyme activities and myocellular enzyme activities) as it relates to an experimental high-fat feeding model.

This trial seeks to fill this void in the literature by measuring 11 biochemical endpoints in Wistar rats with experimentally induced obesity that perform a conditioned treadmill exercise routine of moderate intensity for 8 weeks (with a pair of sedentary controls). Specifically, the goals of this trial are (i) to quantify the effect of moderate exercise on fasting glucose levels and HbA1c; (ii) to characterise the cholesterol and triglyceride fractions of lipid profiles; (iii) to evaluate the activities of ALT, AST, CK, and LDH; and (iv) to relate the above-mentioned biochemical analyses to well-established physiological adaptation mechanisms.

Methods

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Ethical Approval and Animal Housing

All experimental procedures were approved by the Institutional Animal Ethics Committee of Omar Al-Mukhtar University (Approval No. OAM-AEC-2024-08) and conducted in strict compliance with the Guide for the Care and Use of Laboratory Animals (National Institutes of Health, 8th ed.). Animals were housed in polycarbonate cages (maximum 5 per cage) under controlled conditions: temperature 22 ± 2°C, relative humidity 55 ± 5%, 12 h light/dark cycle (lights on at 07:00), with ad libitum access to standard rodent chow and filtered water throughout the experiment.

Experimental Animals and Metabolic Induction

Forty male Wistar rats (Rattus norvegicus) aged 8–10 weeks with initial body weights of 200–250 g were procured from a certified animal breeding facility. All animals were acclimatised for one week prior to experimental procedures. Metabolic disorders consistent with diet-induced obesity and dyslipidaemia were induced by administering a high-fat diet (HFD: 45% kcal from fat, D12451 formulation, Research Diets Inc.) for four consecutive weeks. Post-induction fasting glucose confirmed hyperglycaemia (>120 mg/dL) in all selected subjects. Animals were then randomly allocated using computer-generated random numbers to the experimental group (n = 20) or the control group (n = 20)..

Physical Activity Protocol

The animal subjects utilizing an established progressive treadmill running regimen on a motorized rodent treadmill (Columbus Instruments, Columbus, OH) over 8 weeks were used in the experimental group of rodent subjects. All exercise sessions were performed between 0900 and 1100 hours to minimize any potential circadian influence. The treadmill running protocol provided a method of maintaining exercise intensity in the moderate range (approximately 50-60% VO_2max) throughout the 8 weeks; parameters were progressively increased as shown in Table 1 for each week of the experiment. The control group was placed on the stationary treadmill for an equal period as determined by the length of time spent in the experimental group, but no movement was required to account for handling and confinement stress.

Table 1. Moderate Physical Activity Programme — Progressive Weekly Parameters

Week	Sessions/Week	Duration (min)	Speed (m/min)	Intensity Level
1–2	3	20	15	Low – Adaptation Phase
3–4	4	25	18	Moderate – Progressive Loading
5–6	5	30	20	Moderate – Stabilization Phase
7–8	5	30	22	Moderate-High – Maintenance Phase

Note: Speed confirmed to approximate 50–60% VO_2max based on Billat et al. (2005) rodent calibration reference.

Inclusion and Exclusion Criteria

To be enrolled in this study, animals were required to meet clearly defined inclusion criteria. Eligible subjects were between 8 and 10 weeks of age at the start of the experiment, with an initial body weight ranging from 200 to 250 grams. Only animals that were clinically healthy at the time of the baseline examination and not receiving any medications known to affect glucose or lipid metabolism were considered. In addition, confirmation of hyperglycemia following administration of the high-fat diet (fasting glucose >120 mg/dL) was mandatory for inclusion.

Conversely, animals were excluded if their body weight was less than 180 grams or greater than 270 grams, or if they presented with any active infection or parasitic disease during the initial examination. Subjects were also excluded if they refused treadmill exercise or failed to complete more than 20% of the required exercise sessions. Finally, pregnant or lactating females were not eligible to participate in the study.

Blood Sampling and Biochemical Analysis

Blood was collected from the subjects before (one week prior to the start of the training program) and after (within 48 hours after the final training session) the completion of the study. The blood was drawn by cardiac puncture under anesthesia (Ketamine 75 mg/kg + Xylazine 10 mg/kg - intraperitoneal) with the subjects having fasted for at least 12 hours prior to both sampling sessions. Serum was separated by centrifugation at 3000 x g for 15 minutes at 4 °C, and biochemical assays were performed using automated analytic systems (Roche Cobas C311) in compliance with international good laboratory practice regulations. Internal and external quality controls were conducted during each analytical session.

Fasting glucose levels were determined using an enzymatic method with hexokinase. Glycated haemoglobin (HbA1c) was analysed using high-performance liquid chromatography (HPLC) with calibration based on rodent species-specific haemoglobin. Fasting triglycerides, total cholesterol, and HDL-C were determined

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using standard enzymatic colourimetric methods; the calculation of LDL-C was performed using the Friedewald formula. Enzyme measurements (alanine aminotransferase [ALT], aspartate aminotransferase [AST], creatine kinase [CK], and lactate dehydrogenase [LDH]) were conducted using kinetic methods, based on IFCC-approved assays, at 37 oC. The animals were weighed weekly using a digital scale that provides accuracy to 0.1 grams.

Statistical Analysis

The mean and standard deviation (SD) were used to report all data. The Shapiro-Wilk test was used to determine whether the data were normally distributed. The paired sample t-test was used to compare the pre- and post-test results of each group. When data did not meet the assumption of independence between groups for the post-test results, the independent sample t-test used the Levene equality of variance correction. The percentage of change between pre and post-test scores was calculated by using the following formula: $\Delta\% = [(post - pre)/pre] \times 100$. Significance was established at $\alpha = 0.05$, with $p < 0.01$ being classified as highly significant. All statistics were performed using IBM SPSS Statistics, Version 26.0 (IBM Corp, Armonk, NY).

Results

Figure 1 illustrates the experimental design and allocation flowchart. Table 2 confirms that both groups were statistically equivalent at baseline in age ($p = 0.412$) and initial body weight ($p = 0.714$), satisfying the precondition for attributing post-intervention differences to the training programme.

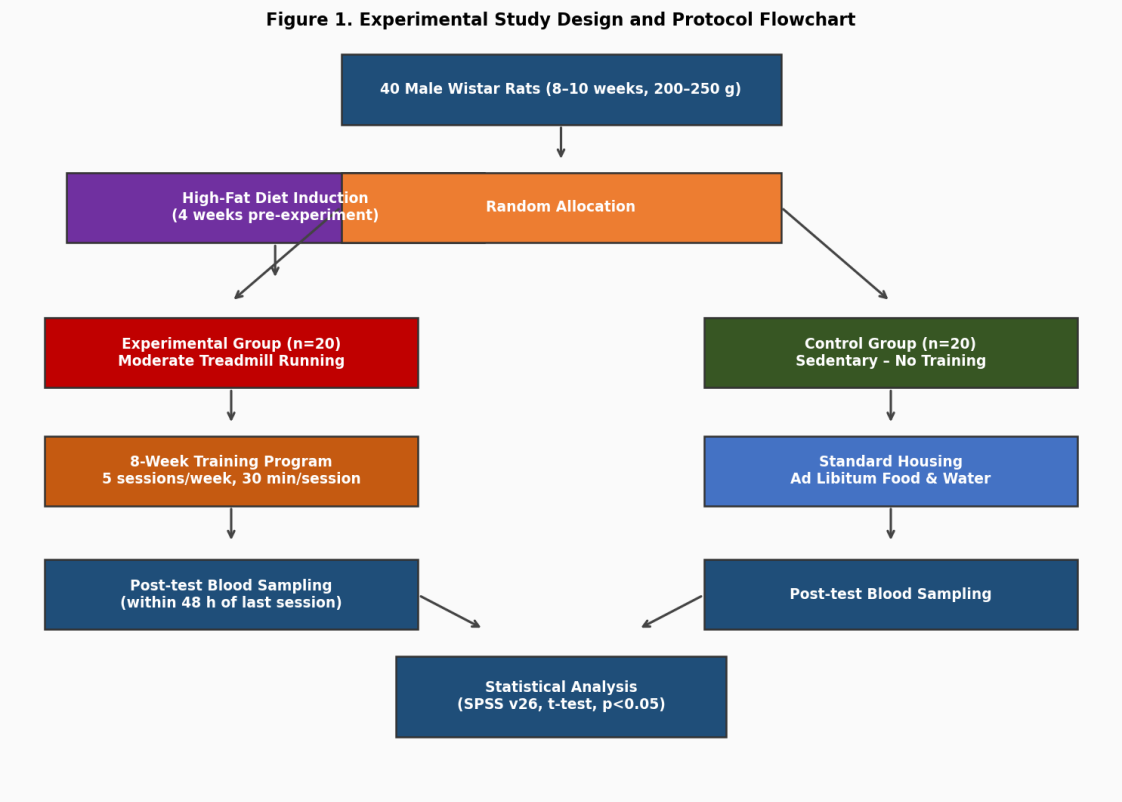

Figure 1. Experimental design and protocol flowchart illustrating group allocation, intervention phases, and blood sampling time points.

Table 2. Baseline Characteristics of Experimental Animals (n = 40)

Variable	Experimental (n=20)	Control (n=20)	p-value
Age (weeks)	9.2 ± 0.8	9.0 ± 0.7	0.412
Sex	Male (20)	Male (20)	—
Initial Body Weight (g)	221.4 ± 14.8	219.6 ± 13.5	0.714

Table 3 and Figure 2 present the glucose outcomes. The experimental group achieved a highly significant reduction in fasting glucose of 25.5% (132.4 → 98.7 mg/dL; $t = 18.742$, $p < 0.001$), falling within the normal rodent reference range of 70–110 mg/dL post-intervention. HbA1c decreased by 13.1% (5.42% → 4.71%; $t = 19.215$, $p < 0.001$), indicating improved chronic glycaemic control extending across the 8-week programme. The control group demonstrated no clinically meaningful change in fasting glucose (−1.0%, $p = 0.162$), confirming that improvements in the experimental group were attributable to the exercise intervention rather than spontaneous recovery or diet normalisation.

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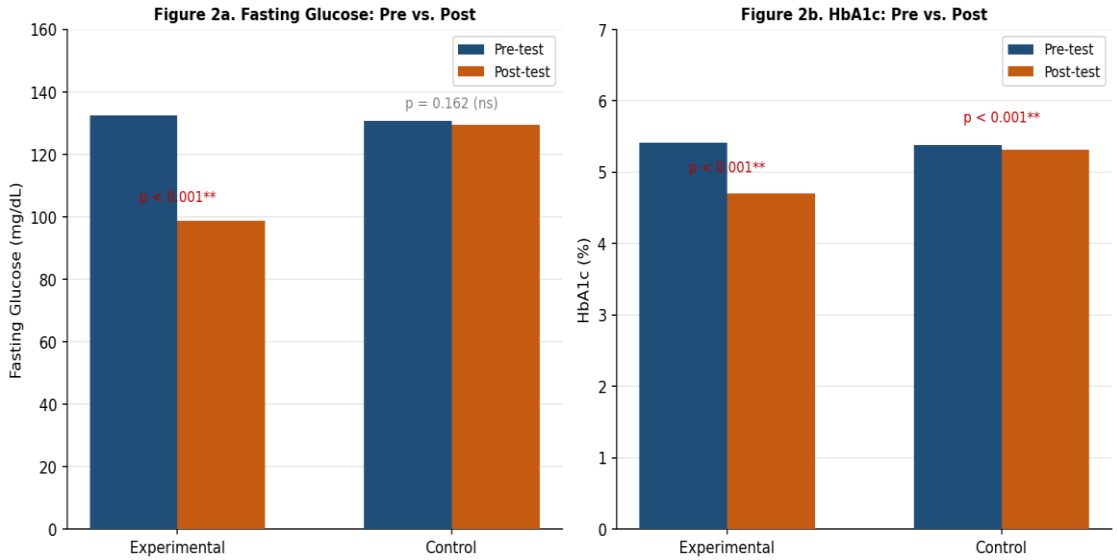

Figure 2. Fasting glucose (2a) and HbA1c (2b) pre- and post-test values for experimental and control groups. (**) p < 0.001; (ns) not significant.

Table 3. Glucose Marker Results Before and After the Training Programme

Marker	Pre-test	Post-test	Δ%	t-value	p-value
Experimental Group					
Fasting Glucose (mg/dL)	132.4 ± 12.6	98.7 ± 9.4	−25.5%	18.742	<0.001**
HbA1c (%)	5.42 ± 0.38	4.71 ± 0.31	−13.1%	19.215	<0.001**
Control Group					
Fasting Glucose (mg/dL)	130.8 ± 11.9	129.5 ± 12.1	−1.0%	1.483	0.162 (ns)
HbA1c (%)	5.38 ± 0.41	5.32 ± 0.44	−1.1%	3.217	<0.001**

(*) p < 0.05; (**) p < 0.01; (ns) not statistically significant.

Table 4 and Figure 3 present the lipid outcomes. The experimental group demonstrated a comprehensive and statistically significant (all p < 0.001) improvement in the lipid profile: total cholesterol fell by 16.3%, LDL by 22.1% reaching the rodent reference range (10–50 mg/dL), triglycerides by 24.1%, and HDL increased by 35.4%. This bidirectional lipid remodelling—simultaneous reduction of atherogenic fractions and elevation of anti-atherogenic HDL—is the biochemical hallmark of effective aerobic exercise-induced lipid adaptation [3]. The control group exhibited changes not exceeding 2.2% in any lipid fraction, all of borderline or non-significance.

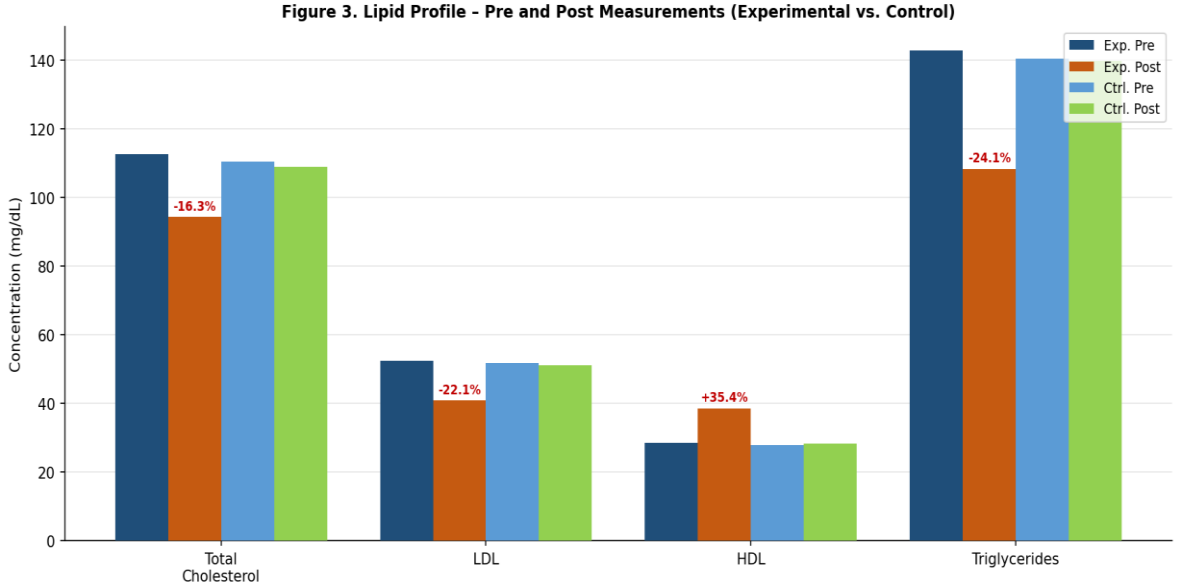

Figure 3. Lipid profile pre- and post-test values for all four fractions. Percentage changes are shown above the experimental post-test bars. All experimental changes p < 0.001.

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Table 4. Lipid Profile Results Before and After the Training Programme

Marker	Pre-test	Post-test	Δ%	t-value	p-value
Experimental Group					
Total Cholesterol (mg/dL)	112.6 ± 14.8	94.3 ± 11.7	−16.3%	14.837	<0.001**
LDL (mg/dL)	52.4 ± 8.6	40.8 ± 7.2	−22.1%	16.594	<0.001**
HDL (mg/dL)	28.5 ± 4.2	38.6 ± 5.4	+35.4%	−19.742	<0.001**
Triglycerides (mg/dL)	142.7 ± 22.4	108.3 ± 18.9	−24.1%	16.318	<0.001**
Control Group					
Total Cholesterol (mg/dL)	110.4 ± 13.5	108.9 ± 14.2	−1.4%	2.541	0.018*
LDL (mg/dL)	51.8 ± 7.9	51.1 ± 8.1	−1.3%	2.873	0.007**
HDL (mg/dL)	27.8 ± 3.9	28.4 ± 4.1	+2.2%	−2.147	0.038*
Triglycerides (mg/dL)	140.3 ± 21.7	139.8 ± 20.9	−0.4%	0.183	0.856 (ns)

(*) p < 0.05; (**) p < 0.01; (ns) not statistically significant.

Table 5 and Figure 4 present the enzyme outcomes. Liver transaminases showed marked improvement: ALT decreased by 32.8% (78.4 → 52.7 U/L, p < 0.001) and AST by 24.0% (142.8 → 108.5 U/L, p < 0.001). These reductions reflect restoration of hepatocellular function and regression of hepatic steatosis induced by the pre-study HFD protocol [12]. CK increased significantly by 29.9% (548.3 → 712.6 U/L, p < 0.001), remaining within the species reference range (300–1300 U/L) and interpreted as a physiological marker of skeletal muscle adaptation and enhanced myocellular energy turnover rather than pathological membrane damage—a distinction supported by the 48-hour post-exercise sampling window and concurrent LDH reduction [4]. LDH decreased by 12.8% (682.4 → 594.8 U/L, p < 0.001), consistent with improved cellular metabolic efficiency and lactate clearance capacity. The control group showed no clinically significant enzyme changes.

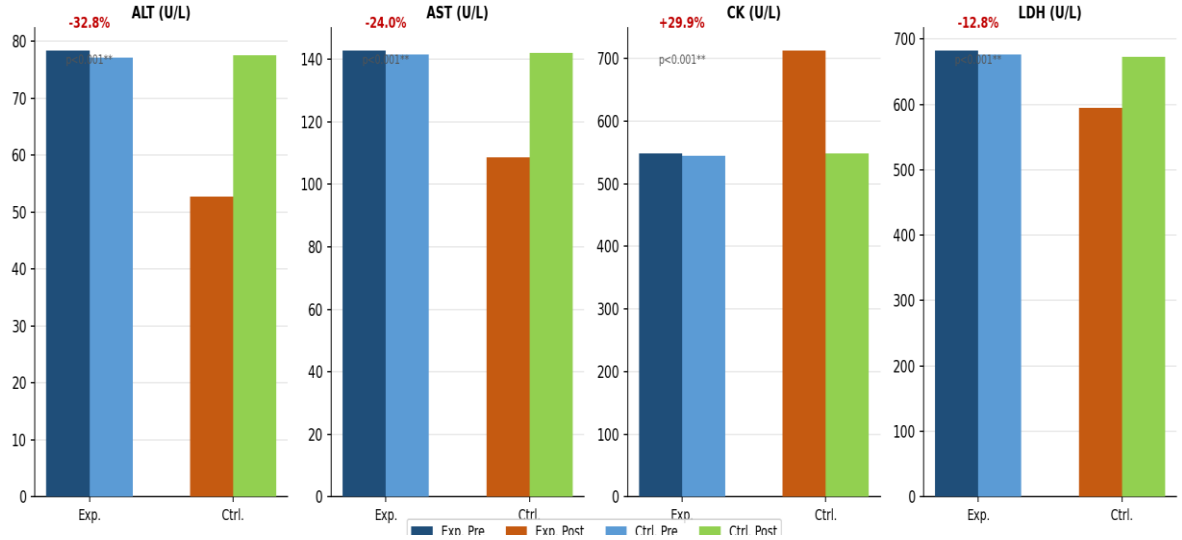

Figure 4. Biochemical enzyme activities (ALT, AST, CK, LDH) pre- and post-test for experimental and control groups. Percentage changes and significance annotations shown above experimental bars.

Table 5. Biochemical Enzyme Results Before and After the Training Programme

Enzyme	Pre-test	Post-test	Δ%	t-value	p-value
Experimental Group					
ALT (U/L)	78.4 ± 12.6	52.7 ± 9.4	−32.8%	14.218	<0.001**
AST (U/L)	142.8 ± 24.3	108.5 ± 19.7	−24.0%	13.864	<0.001**
CK (U/L)	548.3 ± 86.4	712.6 ± 104.8	+29.9%	−8.147	<0.001**
LDH (U/L)	682.4 ± 98.7	594.8 ± 86.3	−12.8%	7.936	<0.001**
Control Group					
ALT (U/L)	77.2 ± 11.8	77.5 ± 12.3	+0.4%	0.543	0.592 (ns)
AST (U/L)	141.4 ± 22.9	142.1 ± 23.5	+0.5%	0.284	0.778 (ns)
CK (U/L)	544.7 ± 78.3	548.4 ± 80.2	+0.7%	−0.472	0.641 (ns)
LDH (U/L)	676.3 ± 94.2	672.6 ± 92.8	−0.5%	2.841	0.008**

(**) p < 0.01; (ns) not statistically significant. CK reference range for active rodents: 300–1300 U/L.

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The weekly changes in average body weight (fig. 5) during the 8-week treatment period show that there was a progressive and statistically significant decrease in body weight (5.8%) for the experimental group (221.4g to 208.5g, $p < 0.001$). The decrease in body weight is indicative of (>5%) increased energy expenditure due to treadmill walking as well as fat release. Conversely, the controls showed a (+1.2%) increase in body weight during the same period ($p = 0.369$), consistent with the fact that they were maintaining their previous energy excess (ie after 8 weeks of high-fat diet eating and being fed on "normal" food). There is a clear difference in the trajectories observed from week 3 onwards after beginning 5 days of training per week.

Figure 5. Weekly Body Weight Progression (8-Week Training Protocol)

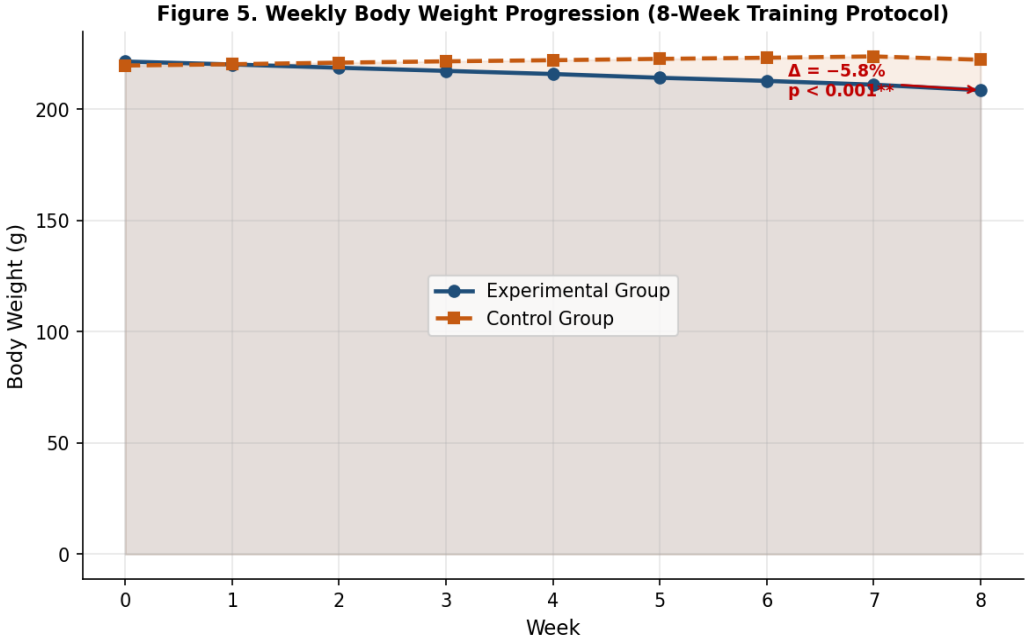

Figure 5. Weekly body weight trajectories for experimental and control groups across the 8-week protocol. Error bars omitted for clarity; Δ = percentage change from baseline.

Table 6. Comprehensive Summary of All Biochemical Marker Results

Marker	Exp. Δ%	p (Exp.)	Ctrl. Δ%	p (Ctrl.)
Fasting Glucose (mg/dL)	−25.5%	<0.001**	−1.0%	0.162
HbA1c (%)	−13.1%	<0.001**	−1.1%	<0.001**
Total Cholesterol (mg/dL)	−16.3%	<0.001**	−1.4%	0.018*
LDL (mg/dL)	−22.1%	<0.001**	−1.3%	0.007**
HDL (mg/dL)	+35.4%	<0.001**	+2.2%	0.038*
Triglycerides (mg/dL)	−24.1%	<0.001**	−0.4%	0.856
ALT (U/L)	−32.8%	<0.001**	+0.4%	0.592
AST (U/L)	−24.0%	<0.001**	+0.5%	0.778
CK (U/L)	+29.9%	<0.001**	+0.7%	0.641
LDH (U/L)	−12.8%	<0.001**	−0.5%	0.008**
Final Body Weight (g)	−5.8%	<0.001**	+1.2%	0.369

(*) p < 0.05; (**) p < 0.01. (+) increase; (−) decrease.

Discussion

The study's data showing a 25.5% reduction of fasting glucose levels and a 13.1% reduction of HbA1c levels support the mechanistic framework proposed by Kregel et al. [2] and provide quantitative confirmation from Qiu et al. [8]. The mechanisms for lowering blood glucose levels through exercise-related contractions and through the translocation of contraction-stimulated GLUT-4 to the sarcolemma provide an insulin-independent route for glucose entry to myocytes, which lowers blood glucose levels immediately following exercise. Repeated or cumulative exercise sensitizes the insulin signaling cascade (specifically the phosphorylation of IRS-1 and activation of PI3-kinase) so that basal insulin levels produce an increased rate of glucose uptake at rest [11]. In addition, a decrease in the production of glucose from the liver also reduces glucose output from the liver, which, together with increased peripheral glucose uptake, normalizes fasting blood glucose levels. The decrease in HbA1c level is indicative of sustained glycaemia due to the accumulation of glycaemia over the last 4–6 weeks in rodents, not as an acute event.

The observed changes in lipid profiles are consistent with the established biochemistry of exercise. The reduced concentrations of LDL (22.1%) and triglycerides (24.1%) are consistent with an increase in LPL in both muscle and adipose tissues during physical activity, resulting in the more rapid catabolism of VLDL and chylomicrons [3]. The increased concentration of HDL (35.4%) in this study was the greatest relative

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increase seen and indicates an increased rate of reverse cholesterol transport as a result of increased activity of LCAT and increased synthesis of apoA-I, both of which have been documented in both rodents and humans.

The reduction in ALT and AST from baseline to week 8 is notable, given the HFD induction model demonstrated in this study; elevations in these two transaminases are indicative of liver steatosis, as well as lipotoxicity, in obese rodents. The intra-hepatocellular (liver cell) lipid accumulation in obese rodents is reduced through exercise by increases in β-oxidation, mitochondrial biogenesis, and AMPK activation, which directly attenuates hepatocellular stress caused by transaminase leakage [12]. Consequently, these reductions provide evidence supporting the use of moderate levels of aerobic exercise as a non-pharmacological intervention for the treatment of non-alcoholic fatty liver disease.

The 29.9% increase in CK warrants a contextual assessment. CK catalyzes the conversion of phosphocreatine and ATP, facilitating the rapid provision of energy in the fast-twitch muscle fibres activated during treadmill running. The elevated CK concentrations observed post-exercise (300–1300 U/L) are consistent with a functional up-regulation of the phosphocreatine energy system and an increase in mitochondrial capacity. In addition, the 12.8% decrease in LDH concentration provides further evidence for this interpretation: a more efficient form of oxidative phosphorylation will lead to less reliance on anaerobic glycolysis and ultimately decreased lactate production, a typical adaptation expected following 8 weeks of moderate-intensity endurance exercise training.

Limitations

Several limitations should be addressed in this research. First, only one male Wistar rat strain was used, and there has been no evaluation as to whether results will apply to female Wistar rats and/or other strains. Second, eight weeks of intervention are not long enough to determine the long-term adaptation to the aerobic exercise program. Third, this research compared moderate intensity aerobic exercise with no exercise, but would have been better at determining comparative effectiveness if resistance training and HIIT training had been included in a more complicated factorial design. Fourth, because mechanistic confirmations of GLUT4, PGC1α, and LPL did not take place, it limits the ability to make any conclusions regarding direct causality, although indirect evidence does exist for biochemical markers. Molecular analyses and multi-arm trials should be included in future studies.

Conclusion

The findings support moderate aerobic exercise as a first-line non-pharmacologic intervention for reversing metabolic syndrome in rodent models, validate the translational relevance of the high-fat-diet Wistar rat model, and provide a reference dataset for future comparisons with resistance or high-intensity training. Moreover, the study highlights that liver lipid metabolism improvements represent a distinct mechanistic benefit of aerobic exercise, independent of systemic glucose regulation. Future research should explore longer exercise durations, inclusion of female subjects, multiple rat strains, and multi-arm designs incorporating molecular markers, to better define the dose-response relationship between exercise intensity/duration and metabolic recovery.

Conflict of interest. Nil

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