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A 12-week cycling program improves glucose homeostasis, lipid profile, and body composition in women with insulin resistance: a pilot study

Saša Plećević[1], Miloš Glišić[2], Emilija Stojanović[2,3], Dragana Bubanja[4], Ivan Srejović[2,5,6], Vladimir Živković[2,5,6], Vladimir Fisenko[6], Stefan Zečar[7] and Vladimir Jakovljević[2,5,8]

[1]*Sports Medicine Association of Serbia, Belgrade, Serbia*
[2]*Department of Physiology, Faculty of Medical Sciences, University of Kragujevac, Kragujevac, Serbia*
[3]*Department of Training and Exercise Science, Faculty of Sport Science, Ruhr University Bochum, Bochum, Germany*
[4]*Clinic for Endocrinology, University Clinical Center Kragujevac, Kragujevac, Serbia*
[5]*Center of Excellence for Redox Balance Research in Cardiovascular and Metabolic Disorders, Kragujevac, Serbia*
[6]*Department of Pharmacology, First Moscow State Medical University I.M. Sechenov, Russia*
[7]*Wyoming Seminary College Preparatory School, Kingston, Pennsylvania, USA*
[8]*Department of Human Pathology, First Moscow State Medical University I.M. Sechenov, Russia*

ABSTRACT

This study evaluated the effects of a cycling intervention on glucose homeostasis, lipid profile, and body composition in individuals with insulin resistance and excess body weight. Seven women participants completed a 12-week supervised cycling program (20–30 min/day, 3 days/wk). Body composition assessments and biochemical analyses, including oral glucose tolerance tests, triglycerides, total cholesterol, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, and non-high-density lipoproteins, were conducted pre- and post-intervention. Paired *t*-tests and Hedge's *g* assessed changes in body composition, lipid profile, and homeostatic model assessment for insulin resistance (HOMA-IR), while a 2×4 repeated-measures analyses of variance (ANOVA) analyzed exercise- and time-related effects on glucose and insulin. Post-intervention, significant moderate reductions were observed in body fat mass ($p = 0.020$, $g = -0.52$), body fat percentage ($p = 0.006$, $g = -0.55$), visceral fat area ($p = 0.005$, $g = -0.64$), and waist-to-hip ratio ($p < 0.001$, $g = -0.87$). A significant, small reduction in non-high-density lipoproteins was also noted ($p = 0.016$, $g = -0.34$). A 2×4 repeated-measures ANOVA revealed a significant effect of exercise on glucose concentration ($p = 0.031$; $\eta_p^2 = 0.640$, moderate), with lower post-exercise levels. Additionally, a significant time×exercise interaction was observed for insulin concentration ($p = 0.009$; $\eta_p^2 = 0.30$, moderate). Follow-up tests comparing pre to post-exercise changes revealed significant decreases in insulin levels at 30-min ($p < 0.001$), 60-min ($p = 0.008$), and 120-min ($p = 0.011$) postload. These findings suggest a 12-week cycling intervention improves glucose homeostasis, lipid profile, and body composition in overweight women with insulin resistance.

Keywords: cholesterol, insulin, glucose, exercise, body weight

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Corresponding Author: Emilija Stojanović, PhD

Department of Physiology, Faculty of Medical Sciences, University of Kragujevac, 69 Svetozara Markovica, Kragujevac 34000, Serbia

Tel: 00381 655510466, E-mail: stojanovic.emilija@yahoo.com

Abbreviations:
HOMA-IR: homeostatic model assessment for insulin resistance
HDL: high-density lipoprotein
LDL: low-density lipoprotein
ANOVA: analyses of variance

INTRODUCTION

Insulin resistance, characterized by hyperinsulinemia, serves as a central precursor to several metabolism-related diseases, including metabolic syndrome and Type 2 diabetes. Diabetes is reaching epidemic proportions globally, currently affecting approximately 537 million adults worldwide, with projections indicating this number will rise to 643 million by 2030.[1,2] In healthy individuals, insulin facilitates glucose uptake by peripheral tissues, such as skeletal muscle and adipose tissue, while simultaneously suppressing glucose production in the liver.[3] However, in the early stages of Type 2 diabetes mellitus development, glucose dysregulation arises due to insulin resistance and insufficient insulin secretion.[4] This dysfunction disrupts glucose homeostasis, leading to metabolic abnormalities and elevated circulating lipids, which can ultimately progress to overt diabetes.[5] Furthermore, the resulting dyslipidemia, characterized by elevated triglycerides, decreased high-density lipoprotein (HDL) cholesterol, and increased atherogenic low-density lipoprotein (LDL) particles, is a well-established risk factor for cardiovascular disease.[6] Therefore, targeting lipid profile alterations is crucial in the early management of insulin resistance.

A sedentary lifestyle significantly contributes to insulin resistance, a key factor in the development of Type 2 diabetes mellitus.[7,8] Emerging evidence suggests that exercise may be the most effective non-pharmacological strategy in preventing and managing insulin resistance and Type 2 diabetes mellitus.[9] Specifically, various forms of exercise such as aerobic, resistance, combined, and flexibility have been identified as important non-pharmacological approaches for managing diabetes-related health complications.[9] In managing diabetes-related health complications, it is generally recommended to engage in ≥150 minutes of moderate-to-vigorous intensity aerobic activity per week, spread over at least three days, with no more than two consecutive days of inactivity.[8]

Aerobic exercise has emerged as a promising means of counteracting insulin resistance,[10] enhancing glucose metabolism,[10] promoting fat loss,[11] and improving vascular function,[12] all of which may help reduce the risk of developing Type 2 diabetes. Despite the importance of aerobic exercise in counteracting diabetes-related health complications, a recent review[9] reinforces the discrepancies in research attention between exercise modalities, with limited literature available regarding the effect of cycling exercise in Type 2 diabetes management, particularly when compared to other forms of exercise such as walking or jogging. Incorporating aerobic exercise, particularly cycling, is highly recommended for overweight individuals with insulin resistance as it provides numerous benefits while minimizing strain on joints and bones.[13] Cycling is a low-impact activity, making it a safe and effective option for individuals with insulin resistance and excess body weight. Additionally, it can be sustained over the long term and easily integrated into daily routines, such as commuting or recreational activities, making it a practical choice for lifestyle modification aimed at managing insulin resistance and excess body weight. Given the clinical importance of managing excess body weight and improving lipid profiles in individuals with insulin resistance, understanding how cycling influences these parameters could provide valuable insights for targeted lifestyle interventions. Considering the benefits of cycling in minimizing joint

strain, its long-term sustainability, and the limited research attention given to this exercise type, our study aimed to quantify the effect of cycling on glucose homeostasis, lipid profile, and body composition in individuals with insulin resistance and excess body weight.

METHODS

Participants

Seven women participants with insulin resistance and excess body weight were recruited to participate in 12-week supervised cycling program. Participants were recruited from the University Clinical Center Kragujevac where the study was carried out. Participants characteristics are shown in Table 1. Insulin resistance and excess body weight were verified as follows: homeostatic model assessment for insulin resistance (HOMA-IR) >2.5[14,15] and body mass index >25 kg/m^2.[16] Baseline biochemical analyses indicative of renal function, electrolyte balance, liver function, inflammation and thyroid function were within the normal reference ranges in individuals we examined. Blood glucose and 25-hydroxyvitamin D were outside of the reference range. All participants were free from any injuries, diabetic nephropathy, diabetic retinopathy, severe diabetic neuropathy, and severe cardiovascular and cerebrovascular diseases preventing safe participation in this study. Moreover, all were sedentary (no participation in regular moderate and/or intensive exercise in the previous 6 months). The experimental design was explained to all participants and written informed consent was obtained prior to commencing data collection. All procedures were approved by the Ethics Committee of the University Clinical Center Kragujevac (Approval No. 01/24-134).

Table 1 Participants' baseline characteristics (n = 7)

Variable	Pre-exercise	Reference range
Renal function		
Urea (mmol/L)	6.4 ± 1.8	3.5 to 7.2
Creatinine (μmol/L)	86.5 ± 16.2	53 to 97.2
Glomerular filtration rate (mL/min)	77.5 ± 21.7	>60
Electrolyte balance		
Potassium (mmol/L)	4.4 ± 0.4	3.5 to 5.2
Urine sodium (mmol/day)	139.2 ± 2.1	40 to 220
General health or immune system		
Protein (mg/g)	1.1 ± 0.1	<30
Calcium (mmol/day)	2.4 ± 0.1	2.5 to 7.5
25-hydroxyvitamin D (nmol/L)	**23.9 ± 11.8**	>75
Metabolic control		
Blood glucose (mg/dL)	**306.1 ± 175.8**	70.0 to 99.0
Liver function		
Aspartate aminotransferase (U/L)	23.5 ± 9.9	8 to 33
Alanine transaminase (U/L)	25.5 ± 6.6	4 to 36
Gamma-glutamyl transferase (U/L)	31.8 ± 12.2	5 to 40
Inflammation		
C-reactive protein (mg/L)	2.5 ± 1.2	<10
Thyroid function		
Free T4 (pmol/L)	14.9 ± 4.4	10.3 to 24.5
Thyroid-stimulating hormone (mU/L)	2.4 ± 1.1	0.45 to 4.12

Glomerular filtration rate – adjusted for the participant's body surface area; values outside of the reference range are bolded.

Procedures

A pre-post longitudinal experimental design was adopted whereby each participant underwent a 12-week supervised cycling program. Body composition, body height, and biochemical analyses were assessed at baseline and following the 12-week cycling program at the same time of day (7:00 to 9:00 AM) and under similar conditions. Participants were instructed to maintain habitual dietary intake during the study period.

Body composition

Body composition was assessed in each participant using a multifrequency bioelectrical impedance analyzer (Inbody 770; Biospace Co, Ltd, Seoul, Republic of Korea) following the manufacturer's standard measurement procedures. Prior to measurement, participants cleaned their hands and feet with provided antibacterial wipes. Personal data (name/ID, age, sex, and height) were manually entered for each participant into the device's software. While standing upright, participants positioned their feet on the foot electrodes and gripped the hand electrodes, ensuring their arms were extended outward to avoid contact with the torso. This posture was maintained throughout the test. Upon completion, participants were instructed to place the hand electrodes back and step off the device. The validity and reliability of the InBody 770 in measuring percentage body fat has been supported previously.[17] Additionally, a validation study[18] has supported the reliability of InBody device in measuring visceral fat area, with strong correlations to gold-standard methods like computed tomography. While the waist to hip ratio derived from bioelectrical impedance may not match manual measurements precisely, it was considered in our study as it has been shown to provide clinically useful approximations in large-scale epidemiological and health-risk studies.[19] The InBody score was included in the study as an additional tool to capture overall changes in body composition by integrating multiple parameters, such as muscle mass, body fat, and body water, into a single value.

Biochemical analysis

Biochemical analyses were performed at baseline and after 12 weeks to assess circulating levels of glucose, insulin, triglycerides, total cholesterol, LDL cholesterol, HDL cholesterol, and non-high-density lipoproteins (non-HDL). Blood samples (5 mL) were drawn from the antecubital vein in test tubes (BD Vacutainer, Becton Dickinson, USA) with seated participants in the morning (7:00–9:00 h) after overnight fasting and at least 24 h after the last exercise session. Oral glucose tolerance tests (OGTTs) were performed in each participant according to standard published protocols.[20] Glucose and insulin samples were collected at fasting (0 min), and at 30 min, 60 min, 90 min, and 120 min postload during the OGTTs, where 75 g of glucose was ingested over a 5-minute period.[20] HOMA-IR was determined as follows:

$$\text{HOMA-IR} = \frac{insulin(mIU/L) \ *glucose(mmol/L)}{22.5}.$$

Triglyceride, total cholesterol, LDL cholesterol, HDL cholesterol and non-HDL were measured using a biochemistry analyzer (Targa BT 4500 Biotecnica instruments S.p.A, Rome, Italy).

Exercise program

A member of the research team supervised the 12-week exercise program, performed on Yesoul M1 bikes (Shenghui International, Fuzhou, Fujian, China), three times per week with no more than two consecutive days off between sessions. Previous studies[21-23] have supported a 12-week exercise duration in patients with Type 2 diabetes mellitus, as this timeframe has been shown to produce significant improvements in glucose homeostasis. The supervised exercise program

consisted of a 15 min warm-up, followed by the main cycling protocol and ended with a 5 min cool-down. The exercise program was performed at an intensity ranging from 55 to 80% of heart rate max (HR_{max}) for 20–30 min per session. The program began at a lower intensity (55–70% HR_{max}) and progressively increased over the 12 weeks, ultimately reaching approximately 70–80% of HR_{max} during the final weeks. Heart rate was monitored by using heart rate fitness tracker bands (Transtek, Zhongshan City, Guangdong, China).

Statistical analysis

Data were analyzed using IBM SPSS (version 20; IBM Corp, Armonk, NY, USA). The Shapiro–Wilk test, quantile–quantile (Q–Q) plots, as well as skewness and kurtosis coefficients were used to verify the normality of all data. Differences in body composition parameters, lipid profile and HOMA-IR between pre- and post-exercise intervention were assessed using paired t tests. The magnitude of these differences was quantified using Hedge's *g* effect size (ES) and interpreted as follows: trivial ≤0.20; small = 0.20–0.49; moderate = 0.50–0.79; large ≥0.80. Separate 2×4 repeated-measures analyses of variance (ANOVAs) with two within-subjects factors were conducted to examine the effects of exercise (pre- vs post-intervention) and time (fasting, 30 min, 60 min, and 120 min postload) on glucose and insulin concentrations. Partial eta-squared (η_p^2) was used as a measure of ES for each repeated-measures ANOVA, and the values were interpreted as follows[24]: no effect ($\eta_p^2 < 0.04$); minimum effect ($0.04 < \eta_p^2 < 0.25$); moderate effect ($0.25 < \eta_p^2 < 0.64$); or strong effect ($\eta_p^2 > 0.64$). Data are presented as mean ± standard deviation (SD) for body composition parameters, glucose and insulin concentrations, or median and interquartile range for cholesterol, triglycerides, HOMA-IR, HDL cholesterol, LDL cholesterol, and non-HDL. Statistical significance for all analyses was set at $p \leq 0.05$.

RESULTS

Mean ± SD for body composition parameters in women participants before and after the 12-week cycling program are shown in Table 2. Hedge's *g* ESs with 95% CI for body composition parameters and lipid profile are shown in Figure 1.

Analyses revealed significant moderate decreases in body fat mass ($p = 0.020$, $g = -0.52$), body fat percentage ($p = 0.006$, $g = -0.55$), visceral fat area ($p = 0.005$, $g = -0.64$), and waist-hip ratio ($p < 0.001$, $g = -0.87$) post-exercise. On the other hand, InBody score displayed significant moderate increase ($p = 0.006$, $g = 0.50$) post-exercise. In addition, significant small decreases were observed in body mass ($p = 0.021$, $g = -0.39$) and body mass index ($p = 0.029$, $g = -0.47$) post-exercise intervention.

Individual data points for each participant and descriptive statistics (minimum, 25th percentile, median, 75th percentile, and maximum) for (A) cholesterol, (B) triglycerides, and (C) HOMA-IR in participants who completed the 12-week cycling program are shown in Figure 2. Individual data points for each participant and descriptive statistics (minimum, 25th percentile, median, 75th percentile, and maximum) for (A) HDL cholesterol, (B) LDL cholesterol, and (C) non-HDL in participants who completed the 12-week cycling program are shown in Figure 3.

Table 2 Body composition parameters before and after the 12-week bicycle training in participants with insulin resistance and excess body weight (n = 7)

Variable	Pre-exercise	Post-exercise	Mean difference
Body mass (kg)	91.2 ± 14.9	84.5 ± 12.7	6.7 ± 5.7
Body fat (kg)	34.7 ± 9.2	29.4 ± 7.9	5.3 ± 4.4
Body fat (%)	37.5 ± 5.5	34.0 ± 5.5	3.6 ± 2.3
Skeletal muscle mass (kg)	31.7 ± 4.5	31.0 ± 3.9	0.7 ± 1.4
Body mass index (kg/m^2)	30.8 ± 4.3	28.5 ± 3.4	2.2 ± 2.1
Visceral fat area (cm^2)	168.6 ± 49.3	133.0 ± 47.3	35.6 ± 22.2
Intracellular water (L)	25.8 ± 3.5	25.3 ± 3.0	0.5 ± 1.1
Extracellular water (L)	15.6 ± 1.9	15.4 ± 1.8	0.2 ± 0.4
Total body water (L)	41.4 ± 5.4	40.6 ± 4.7	0.8 ± 1.5
Waist-hip ratio	1.0 ± 0.1	0.9 ± 0.1	0.1 ± 0
Basal metabolic rate (kcal)	1591.1 ± 158.5	1569.3 ± 140.3	21.9 ± 43.4
Recommended calorie intake	2097.9 ± 396.8	2015.9 ± 350.9	82.0 ± 91.8
Protein (kg)	11.1 ± 1.5	10.9 ± 1.2	0.2 ± 0.5
InBody score (points)	67.4 ± 7.3	71.6 ± 7.3	4.1 ± 2.6

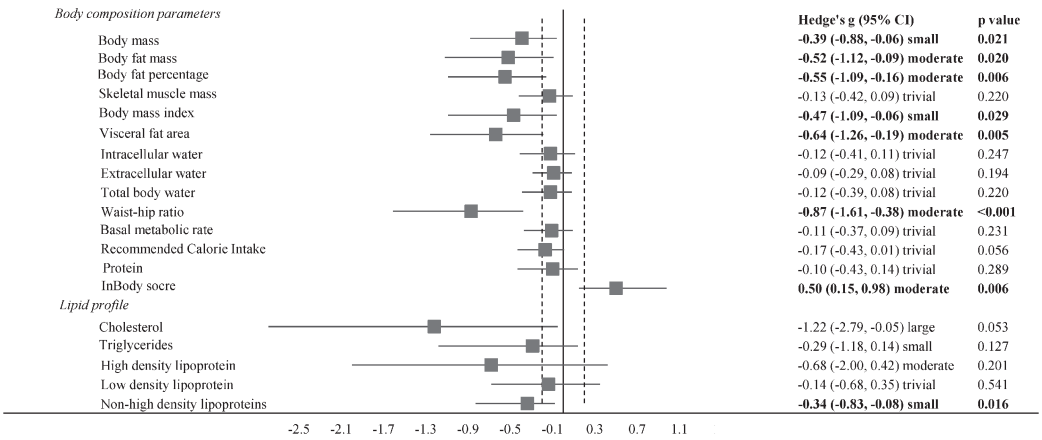

Fig. 1 Hedges' *g* effect sizes (with 95% confidence intervals) for body composition parameters and lipid profile between pre- and post-exercise
Dash lines represents borders for trivial changes.

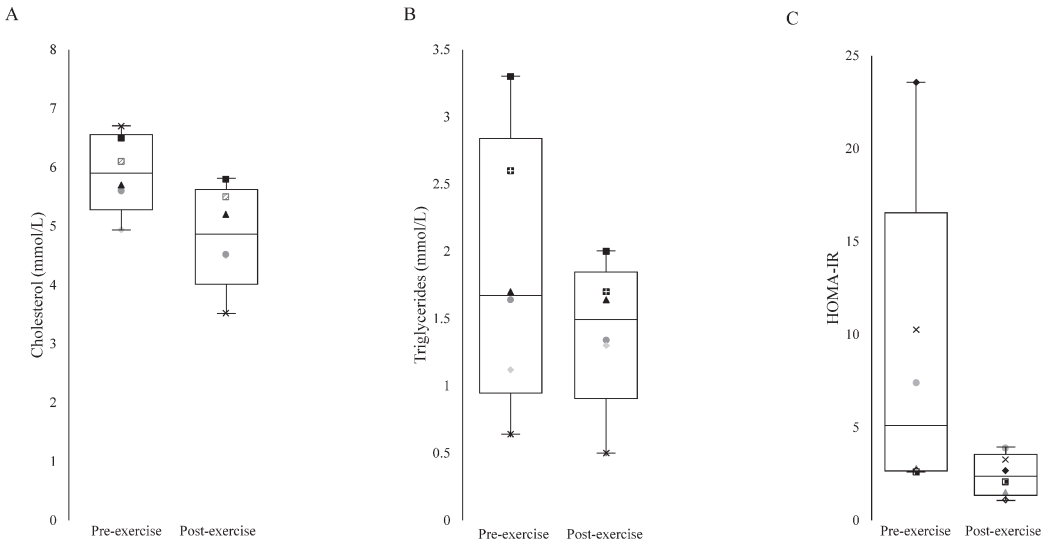

Fig. 2 Cholesterol, triglycerides, and homeostatic model assessment for insulin resistance (HOMA-IR) pre- and post-exercise

Each marker represents a different participant. In the box plots, whiskers indicate the minimum and maximum values, the boundary of the box closest to zero indicates the 25th percentile, the black line within the box indicates the median, and the boundary of the box farthest from zero indicates the 75th percentile.

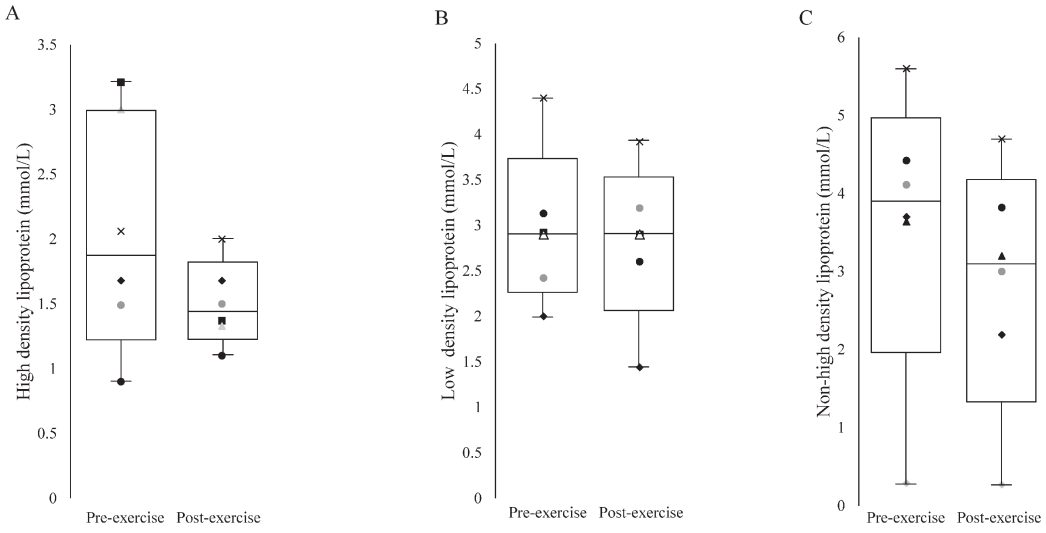

Fig. 3 High density lipoprotein, low density lipoprotein and non-high density lipoprotein pre- and post-exercise

Each marker represents a different participant. In the box plots, whiskers indicate the minimum and maximum values, the boundary of the box closest to zero indicates the 25th percentile, the black line within the box indicates the median, and the boundary of the box farthest from zero indicates the 75th percentile.

A significant, small decrease was observed in non-HDL (p = 0.016, g = −0.34) post-exercise. In addition, non-significant moderate to large decreases post-exercise were observed for cholesterol (p = 0.053, g = −1.22) and HOMA-IR (p = 0.126, g = −0.67).

The results of the repeated-measures ANOVAs, along with the mean ± SD for glucose (A) and insulin concentrations (B) at fasting (0 min), 30 min, 60 min, and 120 min postload during the OGTTs, are presented in Figure 1. The 4×2 repeated-measures ANOVA revealed a significant main effect of time (p = 0.002; η_p^2 = 0.606, moderate) with significantly higher glucose concentrations at 30 min postload compared to fasting (p = 0.030). A significant main effect of exercise was also observed (p = 0.031; η_p^2 = 0.640, moderate), indicating significantly lower glucose concentrations post-exercise.

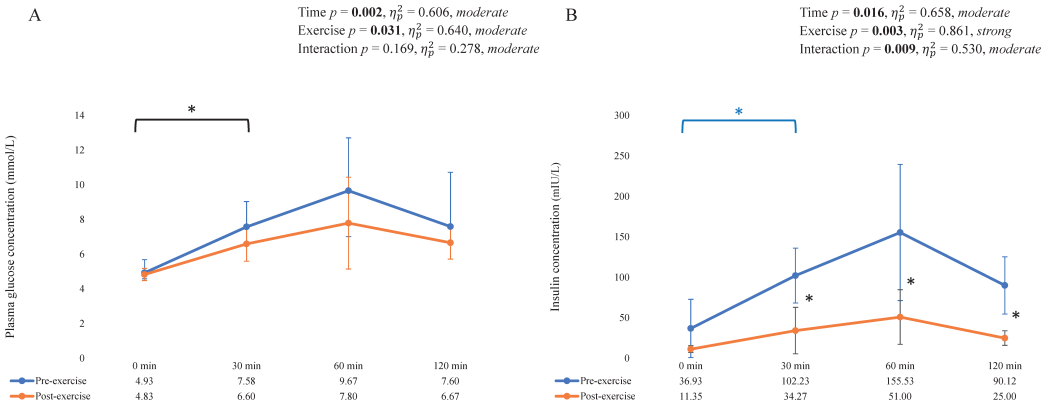

Fig. 4 Glucose and insulin responses during oral glucose tolerance testing
The black bracket indicates a significant main effect of time, showing that participants exhibited significantly (p = 0.030) higher glucose concentrations at 30 minutes postload compared to the fasting condition; the blue bracket indicates a significant (p = 0.030) increase in insulin concentration at 30 minutes postload compared to the fasting condition prior to the exercise intervention.

The 4×2 repeated-measures ANOVA revealed a significant interaction between time and exercise (p = 0.009; η_p^2 = 0.30, moderate) for insulin concentration. Follow-up tests comparing time points revealed significantly higher insulin concentrations 30 min postload compared to fasting (p = 0.030) before the exercise intervention. In addition, follow-up tests comparing pre- to post-exercise changes revealed significant decreases in insulin concentration at 30 min (p < 0.001), 60 min (p = 0.008), and 120 min (p = 0.011) postload.

DISCUSSION

The findings of this study add to the limited knowledge concerning the effects of a 12-week cycling exercise program on body composition, lipid profile, and glucose homeostasis in women patients with insulin resistance and excess body weight. Specifically, we observed significant moderate improvements in body composition, lipid profile (non-HDL and cholesterol), and glucose homeostasis (glucose and insulin concentration) following the 12-week exercise program. Altogether, our data provide exercise-specific insight into the efficacy of a 12-week cycling program in improving body composition, lipid profile, and glucose homeostasis.

Evidence suggests that aerobic exercise can beneficially influence diabetes-related health complications. Our results indicate that a 12-week cycling program may help counteract diabetes-related health complications, with significant moderate decreases in body fat percentage ($p = 0.006$, 3.6 ± 2.3%), body fat mass ($p = 0.020$, 5.3 ± 4.4 kg), visceral fat area ($p = 0.005$, 35.6 ± 22.2 cm^2), and waist-hip ratio ($p < 0.001$, 0.1 ± 0). The benefits observed in our study exceeded those reported by Gholami[25] who detected a 1.5% reduction in body fat percentage following a 12-week predominantly moderate cycling program (3 days/wk, 45 min/day) in patients with Type 2 diabetes and peripheral neuropathy. The observed variations may be underpinned by the lower baseline body fat percentage of participants (27.4 ± 3.2% vs 37.5 ± 5.5%) recruited by Gholami et al.[25] Similarly, Bourne et al[26] reported only a 0.2% reduction in body fat following a 12-week electrically assisted cycling program (median: 66 min/wk, 10.4 km/wk) in patients with Type 2 diabetes mellitus. Disparities between our results and those reported by Bourne et al[26] may be attributed to differences in cycling type (electrically assisted vs regular bike), which can influence the load encountered, thereby affecting calorie expenditure and body composition improvements. On the other hand, our data align with findings by de Oliveira et al[27] and Maillard et al[28] who demonstrated a 2–3% reduction in body fat among Type 2 diabetic patients following 12-week (3 days/wk, 20–50 min/day)[27] and 16-week cycling programs (2 days/wk, 20 min/day high intensity or 40 min/day moderate intensity).[28] Altogether, these findings support the effectiveness of a 12-week cycling exercise program in providing sufficient stimuli to improve body composition in patients with insulin resistance and excess body weight.

The improvements in the lipid profile support the inferences observed in body composition. Specifically, our data revealed small to moderate decreases in non-HDL ($p = 0.016$, 0.76 ± 0.52 mmol/L) and total cholesterol ($p = 0.053$, 1.08 ± 1.05 mmol/L) following the 12-week cycling program. Our results partially align with those of de Oliveira et al[27] and Madsen et al[29] who reported nonsignificant reductions in total cholesterol of 0.45 mmol/L[27] and 0.38 mmol/L,[29] respectively, following cycling interventions. The variation in findings may arise from differences in exercise intensity and duration, or from baseline cholesterol levels of the recruited participants across studies. For instance, Madsen et al[29] implemented a protocol involving 10×60-second high-intensity intervals (with 1-minute active recovery periods),[29] performed for 20 min/day, 3 days/wk, over 8 weeks.[29] The greater reliance on the glycolytic system in short, high-intensity intervals may explain the smaller cholesterol reduction observed in Madsen's study. Further, de Oliveira et al[27] recruited participants with lower baseline cholesterol levels (4.74 mmol/L vs 5.92 mmol/L), potentially accounting for the smaller effect observed in their study. The beneficial effects of exercise on non-HDL and total cholesterol observed in our study are mediated by enhanced lipoprotein metabolism (increasing lipoprotein lipase activity, promoting clearance of triglyceride-rich lipoproteins and reducing non-HDL levels),[30] improved insulin sensitivity,[31] increased fat oxidation (promoting fatty acid utilization, reducing lipid accumulation in the liver and improving overall lipid metabolism),[32] and reduced hepatic cholesterol synthesis (through the downregulation of key enzymes involved in cholesterol biosynthesis, such as 3-hydroxy-3-methylglutaryl-coenzyme A reductase [HMG-CoA reductase]).[33] These mechanisms collectively contribute to better lipid regulation. Therefore, practitioners are encouraged to consider a 12-week cycling intervention as a strategy to improve the lipid profile.

A main effect of exercise was observed ($p = 0.031$; $\eta_p^2 = 0.640$, moderate), with significantly lower glucose and insulin concentration post-exercise. Improved insulin sensitivity and metabolic efficiency were partially supported by a trend toward reduced insulin resistance (HOMA-IR, −2.46; $p = 0.126$). Research has also examined different cycling interventions in patients with Type 2 diabetes, reporting either similar (fasting glucose −34 mg/dL and glycosylated hemoglobin −0.7%, $p < 0.05$[25]) or lower improvements in glucose homeostasis (HOMA-IR, −1.28; $p = 0.4$[34],

−0.54[29], −0.28[26]). This variability underscores that while exercise is beneficial, the magnitude of its impact can differ based on the specific intervention and patient characteristics. Exercise appears to stimulate muscle glucose transport by upregulating the glucose transporter 4 (GLUT4) isoform in skeletal muscle.[35] This acute effect is followed by a sustained improvement in insulin sensitivity.[36] Together, these adaptations result in enhanced insulin sensitivity and responsiveness following exercise. Additionally, exercise improves muscle blood flow through capillary recruitment, both crucial factors for better glucose management. Collectively, our findings suggest that a 12-week cycling intervention improves insulin sensitivity in women with insulin resistance and excess body weight, in part by promoting GLUT4 translocation and enhancing muscle blood flow, which are crucial adaptations in managing glucose levels and insulin resistance.

While this pilot study introduces a novel perspective by focusing on a clinically underrepresented population, ensuring participant safety, adherence, and exercise progression representing a crucial step in evaluating the feasibility, responsiveness, and clinical relevance of supervised cycling interventions, it has certain limitations. First, the absence of a control group limits our ability to draw definitive conclusions, highlighting the need for future research incorporating a control condition. This would enable more robust comparisons and a clearer understanding of the intervention's effectiveness. Second, the results are specific to women with insulin resistance and excess body weight, and may not be generalizable to other populations. Third, the study focused on short-term outcomes related to the processes of care and measurements, without assessing long-term morbidity and mortality indicators. Future studies should also evaluate the sustainability of the observed effects over time and explore broader health impacts, including quality of life and clinical endpoints.

CONCLUSION

The results of the present study demonstrate that a 12-week cycling program improved body composition, lipid profile, and glucose homeostasis in women with insulin resistance and excess body weight, even in the absence of concomitant energy restriction. These findings may help practitioners design exercise programs that promote beneficial adaptations and mitigate the progression of diabetes-related health complications.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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REFERENCES

1 Ogurtsova K, Guariguata L, Barengo NC, et al. IDF diabetes Atlas: Global estimates of undiagnosed diabetes in adults for 2021. *Diabetes Res Clin Pract*. 2022;183:109118. doi:10.1016/j.diabres.2021.109118

2 Magliano DJ, Boyko EJ; IDF Diabetes Atlas 10th edition scientific committee. Global picture. In: Magliano DJ, Boyko EJ; IDF Diabetes Atlas 10th edition scientific committee, eds. *IDF Diabetes Atlas*. 10th ed.

International Diabetes Federation; 2021:chap 3. Accessed August 20, 2025. https://www.ncbi.nlm.nih.gov/books/NBK581934/
3 Samuel VT, Shulman GI. The pathogenesis of insulin resistance: integrating signaling pathways and substrate flux. *J Clin Invest*. 2016;126(1):12–22. doi:10.1172/JCI77812
4 Wang Q, Holmes MV, Davey Smith G, Ala-Korpela M. Genetic support for a causal role of insulin resistance on circulating branched-chain amino acids and inflammation. *Diabetes Care*. 2017;40(12):1779–1786. doi:10.2337/dc17-1642
5 Chandrasekaran P, Weiskirchen R. Cellular and molecular mechanisms of insulin resistance. *Curr Tissue Microenviron Rep.* 2024;5:79–90. doi:10.1007/s43152-024-00056-3
6 Du Z, Qin Y. Dyslipidemia and cardiovascular disease: current knowledge, existing challenges, and new opportunities for management strategies. *J Clin Med*. 2023;12(1):363. doi:10.3390/jcm12010363
7 Andersson DP, Kerr AG, Dahlman I, Rydén M, Arner P. Relationship between a sedentary lifestyle and adipose insulin resistance. *Diabetes*. 2023;72(3):316–325. doi:10.2337/db22-0612
8 Sato Y. Overview of exercise prescription for diabetes patients and its application in Japan. *J Sci Sport Exerc*. 2020;2(3):226–235. doi:10.1007/s42978-020-00061-6
9 Pan B, Ge L, Xun YQ, et al. Exercise training modalities in patients with type 2 diabetes mellitus: a systematic review and network meta-analysis. *Int J Behav Nutr Phys Act*. 2018;15(1):72. doi:10.1186/s12966-018-0703-3
10 Motahari-Tabari N, Ahmad Shirvani M, Shirzad-E-Ahoodashty M, Yousefi-Abdolmaleki E, Teimourzadeh M. The effect of 8 weeks aerobic exercise on insulin resistance in type 2 diabetes: a randomized clinical trial. *Glob J Health Sci*. 2014;7(1):115–121. doi:10.5539/gjhs.v7n1p115
11 Jayedi A, Soltani S, Emadi A, Zargar MS, Najafi A. Aerobic Exercise and Weight Loss in Adults: A Systematic Review and Dose-Response Meta-Analysis. *JAMA Netw Open*. 2024;7(12):e2452185. doi:10.1001/jamanetworkopen.2024.52185
12 Campbell A, Grace F, Ritchie L, Beaumont A, Sculthorpe N. Long-term aerobic exercise improves vascular function into old age: a systematic review, meta-analysis and meta regression of observational and interventional studies. *Front Physiol*. 2019;10:31. doi:10.3389/fphys.2019.00031
13 Usui K, Yamanouchi K, Asai K, et al. The effect of low intensity bicycle exercise on the insulin-induced glucose uptake in obese patients with type 2 diabetes. *Diabetes Res Clin Pract.* 1998;41(1):57–61. doi:10.1016/s0168-8227(98)00068-0
14 Singh Y, Garg MK, Tandon N, Marwaha RK. A study of insulin resistance by HOMA-IR and its cut-off value to identify metabolic syndrome in urban Indian adolescents. *J Clin Res Pediatr Endocrinol.* 2013;5(4):245–251. doi:10.4274/Jcrpe.1127
15 Cho SK, Huh JH, Yoo JS, Kim JW, Lee KJ. HOMA-estimated insulin resistance as an independent prognostic factor in patients with acute pancreatitis. *Sci Rep*. 2019;9(1):14894. doi:10.1038/s41598-019-51466-5
16 Yoo HJ. Body mass index and mortality. *J Obes Metab Syndr*. 2017;26(1):3–9. doi:10.7570/jomes.2017.26.1.3
17 McLester CN, Nickerson BS, Kliszczewicz BM, McLester JR. Reliability and agreement of various InBody body composition analyzers as compared to dual-energy X-ray absorptiometry in healthy men and women. *J Clin Densitom.* 2020;23(3):443–450. doi:10.1016/j.jocd.2018.10.008
18 Ogawa H, Fujitani K, Tsujinaka T, et al. InBody 720 as a new method of evaluating visceral obesity. *Hepatogastroenterology.* 2011;58(105):42–44.
19 Song Y, Hwang JA, Shin J, et al. Waist–hip ratio measured by bioelectrical impedance analysis as a valuable predictor of chronic kidney disease development. *BMC Nephrol*. 2022;23(1):349. doi:10.1186/s12882-022-02981-7
20 Eyth E, Basit H, Swift CJ. Glucose tolerance test. *StatPearls.* StatPearls Publishing; 2023. Accessed August 20, 2025. https://www.ncbi.nlm.nih.gov/books/NBK532915/
21 Umpierre D, Ribeiro PA, Kramer CK, et al. Physical activity advice only or structured exercise training and association with HbA1c levels in type 2 diabetes: a systematic review and meta-analysis. *JAMA*. 2011;305(17):1790–1799. doi:10.1001/jama.2011.576
22 Mannucci E, Bonifazi A, Monami M. Comparison between different types of exercise training in patients with type 2 diabetes mellitus: a systematic review and network metanalysis of randomized controlled trials. *Nutr Metab Cardiovasc Dis*. 2021;31(7):1985–1992. doi:10.1016/j.numecd.2021.02.030
23 Garcia SP, Cureau FV, Iorra FQ, et al. Effects of exercise training and physical activity advice on HbA1c in people with type 2 diabetes: A network meta-analysis of randomized controlled trials. *Diabetes Res Clin Pract*. 2025;221:112027. doi:10.1016/j.diabres.2025.112027
24 Ferguson CJ. An effect size primer: A guide for clinicians and researchers. *Prof Psychol Res Pr*. 2009;40(5):532–538. doi:10.1037/a0015808

25 Gholami F, Nazari H, Alimi M. Cycle Training improves vascular function and neuropathic symptoms in patients with type 2 diabetes and peripheral neuropathy: A randomized controlled trial. *Exp Gerontol*. 2020;131:110799. doi:10.1016/j.exger.2019.110799
26 Bourne JE, Leary S, Page A, et al. Electrically assisted cycling for individuals with type 2 diabetes mellitus: a pilot randomized controlled trial. *Pilot Feasibility Stud*. 2023;9(1):60. doi:10.1186/s40814-023-01283-5
27 de Oliveira VN, Bessa A, Jorge ML, et al. The effect of different training programs on antioxidant status, oxidative stress, and metabolic control in type 2 diabetes. *Appl Physiol Nutr Metab*. 2012;37(2):334–344. doi:10.1139/h2012-004
28 Maillard F, Rousset S, Pereira B, et al. High-intensity interval training reduces abdominal fat mass in postmenopausal women with type 2 diabetes. *Diabetes Metab*. 2016;42(6):433–441. doi:10.1016/j.diabet.2016.07.031
29 Madsen SM, Thorup AC, Bjerre M, Jeppesen PB. Does 8 weeks of strenuous bicycle exercise improve diabetes-related inflammatory cytokines and free fatty acids in type 2 diabetes patients and individuals at high-risk of metabolic syndrome? *Arch Physiol Biochem.* 2015;121(4):129–138. doi:10.3109/13813455.2015.1082600
30 Stanton KM, Kienzle V, Dinnes DLM, et al. Moderate-and high-intensity exercise improves lipoprotein profile and cholesterol efflux capacity in healthy young men. *J Am Heart Assoc*. 2022;11(12):e023386. doi:10.1161/JAHA.121.023386
31 Gylling H, Hallikainen M, Pihlajamäki J, et al. Insulin sensitivity regulates cholesterol metabolism to a greater extent than obesity: lessons from the METSIM Study 1. *J Lipid Res*. 2010;51(8):2422–2427. doi:10.1194/jlr.P006619
32 Haus JM, Solomon TP, Kelly KR, et al. Improved hepatic lipid composition following short-term exercise in nonalcoholic fatty liver disease. *J Clin Endocrinol Metab*. 2013;98(7):E1181–E1188. doi:10.1210/jc.2013-1229
33 Zhao J, Song Y, Zeng Y, et al. Improvement of hyperlipidemia by aerobic exercise in mice through a regulatory effect of miR-21a-5p on its target genes. *Sci Rep*. 2021;11(1):11966. doi:10.1038/s41598-021-91583-8
34 Mohajeri Tehrani MR, Tajvidi M, Kahrizi S, Hedayati M. Does endurance training affect IGF-1/IGFBP-3 and insulin sensitivity in patients with type 2 diabetes? *J Sports Med Phys Fitness*. 2015;55(9):1004–1012.
35 Holloszy JO. A forty-year memoir of research on the regulation of glucose transport into muscle. *Am J Physiol Endocrinol Metab*. 2003;284(3):E453–E467. doi:10.1152/ajpendo.00463.2002
36 Holloszy JO. Exercise-induced increase in muscle insulin sensitivity. *J Appl Physiol (1985)*. 2005;99(1):338–343. doi:10.1152/japplphysiol.00123.2005