

Research Article**The effects of a 12-week combined aerobic and resistance training program on HRV in individuals with T2DM and examined its associations with glycemic, lipid, and inflammatory (C-reactive protein, CRP) markers.****Dr. Kavindra Kumar¹, Dr. Ashutosh Jain², Dr. Dhiraj Mahaseth³****Ph.D Scholar, Dept. of Physiology, IMCH&RC, Malwanchal University, Indore****Associate Professor, Dept. of Physiology, IMCH&RC, Malwanchal University, Indore****Associate Professor, Dept. Biochemistry, Madhubani Medical College, Madhubani****Corresponding Author: Dr. Kavindra Kumar, Email:Id:kavin27@gmail.com****Abstract:**

Background: Aerobic and resistance exercise have been shown to enhance heart rate variability (HRV), yet the magnitude, sustainability, and underlying metabolic and inflammatory mechanisms of these effects remain unclear. This is particularly relevant in India, where type 2 diabetes mellitus (T2DM) prevalence is rising and sedentary lifestyles amplify cardiometabolic risk. **Aim:** This study investigated the effects of a 12-week combined aerobic and resistance training program on HRV in individuals with T2DM and examined its associations with glycemic, lipid, and inflammatory (C-reactive protein, CRP) markers. **Methods:** One hundred adults with T2DM were randomized to an exercise (n = 50) or control group (n = 50). The exercise group completed a supervised 12-week program combining aerobic and resistance training thrice weekly. Baseline and post-intervention assessments included anthropometry, hemodynamics, HRV, physical fitness (handgrip and leg strength, VO_2max , muscle endurance), and fasting blood measurements of glucose, insulin, C-peptide, HbA1c, lipid profile, and CRP. Insulin resistance was estimated using HOMA-IR. **Results:** The exercise group demonstrated significant improvements in HRV, glycemic control (reduced fasting glucose, insulin, HOMA-IR, C-peptide, HbA1c), lipid profile (decreased total cholesterol, triglycerides, LDL-C; increased HDL-C), and systemic inflammation (reduced CRP) ($p < 0.05$). No significant changes were observed in the control group. **Conclusion:** Twelve weeks of combined aerobic and resistance training significantly enhances autonomic function and cardiometabolic health in individuals with T2DM. These findings highlight structured exercise as a potent, non-pharmacological intervention to improve glycemic control, lipid metabolism, and systemic inflammation, supporting its integration into clinical and public health strategies to mitigate diabetes-related cardiovascular risk in India and globally.

Key words: Type 2 diabetes mellitus; heart rate variability; autonomic nervous system; lipid profile; glucose profile; C-peptide; C-reactive protein.

Introduction:

Type 2 diabetes mellitus (T2DM) is a prevalent metabolic disorder characterized by chronic hyperglycemia resulting from insulin resistance and progressive pancreatic β -cell dysfunction [1,2]. Globally, the prevalence of T2DM has reached pandemic proportions, posing substantial health and economic burdens due to its associated microvascular and macrovascular complications, including cardiovascular disease, nephropathy, neuropathy, and retinopathy. Among these, cardiovascular complications remain the leading cause of morbidity and mortality in patients with T2DM, emphasizing the critical need for interventions targeting cardiometabolic risk factors.

Heart rate variability (HRV), defined as the variation in time intervals between consecutive heartbeats, is a noninvasive marker of autonomic nervous system (ANS) function and cardiac autonomic modulation [4,5]. Reduced HRV is commonly observed in individuals with T2DM and is strongly associated with increased cardiovascular risk, impaired baroreflex sensitivity, and poor glycemic control [6,7]. Autonomic dysfunction in T2DM is multifactorial, arising from chronic hyperglycemia, insulin resistance, oxidative stress, and systemic inflammation. Altered sympathovagal balance, as reflected by decreased HRV, contributes to arrhythmogenesis, endothelial dysfunction, and adverse cardiovascular outcomes. Consequently, HRV has emerged as both a prognostic biomarker and a potential target for therapeutic interventions aimed at improving cardiometabolic health in this population.

Physical activity is widely recognized as a cornerstone of non-pharmacological management of T2DM, exerting beneficial effects on glycemic control, lipid metabolism, insulin sensitivity, and inflammatory status [1-9]. Aerobic exercise, such as brisk walking or cycling, has been shown to enhance cardiorespiratory fitness, improve endothelial function, and reduce fasting and postprandial glucose levels. Resistance training, involving progressive muscle-strengthening exercises, promotes skeletal muscle glucose uptake, increases lean body mass, and enhances insulin sensitivity. Recent evidence [10-14] suggests that combining aerobic and resistance exercise may provide additive or synergistic benefits, targeting multiple metabolic and cardiovascular pathways simultaneously. The American Diabetes Association (ADA) and the American College of Sports Medicine (ACSM) recommend incorporating both exercise modalities in the management of T2DM for optimal cardiometabolic outcomes.

In addition to improvements in glucose and lipid metabolism, exercise has demonstrated anti-inflammatory effects in individuals with T2DM. Chronic low-grade inflammation, characterized by elevated levels of C-reactive protein (CRP) and pro-inflammatory cytokines such as interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF- α), contributes to insulin resistance and endothelial dysfunction. Structured exercise reduces visceral adiposity, modulates cytokine production, and downregulates hepatic CRP synthesis, thereby attenuating systemic inflammation [14,15]. Furthermore, accumulating evidence indicates that improvements in HRV following exercise are closely linked to reductions in systemic inflammation and enhanced autonomic regulation, suggesting an integrated relationship between physical activity, ANS function, and metabolic health [17-21].

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Despite the well-documented [22-29] benefits of aerobic and resistance exercise on individual cardiometabolic parameters, few studies have comprehensively examined their combined effects on HRV alongside glycemic, lipid, and inflammatory profiles in patients with T2DM. While prior research [22-29] has reported improvements in HRV following aerobic or resistance exercise alone, the magnitude and sustainability of these effects remain inconsistent, and the underlying associations with metabolic and inflammatory markers require further elucidation. Investigating these relationships is particularly relevant in the Indian population, where T2DM prevalence is rising rapidly and sedentary lifestyles exacerbate cardiometabolic risk. Therefore, the aim of the present study was to investigate the effects of a 12-week combined aerobic and resistance training program on heart rate variability (HRV) in individuals with type 2 diabetes mellitus, and its association with glycemic, lipid, and inflammatory (C-reactive protein) profiles. Research Question of the study was does a 12-week program of combined aerobic and resistance exercise alter HRV in individuals with type 2 diabetes mellitus, and is this associated with changes in glucose metabolism, lipid profile, and systemic inflammation? Null hypothesis (H_0): A 12-week combined aerobic and resistance exercise program does not affect HRV, glucose metabolism, lipid profile, or C-reactive protein levels in individuals with type 2 diabetes mellitus. Alternative hypothesis (H_1): A 12-week combined aerobic and resistance exercise program improves HRV and is associated with favorable changes in glucose metabolism, lipid profile, and C-reactive protein levels in individuals with type 2 diabetes mellitus.

Materials & methods:

A total of 100 individuals with type 2 diabetes mellitus were enrolled and randomly assigned to either the exercise group ($n = 50$) or the control group ($n = 50$). All participants provided written informed consent, and the study was conducted in accordance with the Declaration of Helsinki.

Anthropometric and Physiological Assessments: Baseline anthropometric parameters included body weight, height, body mass index (BMI), waist circumference, and body fat percentage (assessed via bioelectrical impedance). Resting blood pressure (systolic and diastolic) was measured after a 10-minute seated rest using an automated sphygmomanometer, while resting heart rate was recorded with a wireless monitor.

Heart Rate Variability (HRV): HRV was assessed at 10:00 a.m. following a 20-minute rest. A five-minute ECG recording was analyzed in both time and frequency domains. Time-domain indices included the root mean square of successive differences (rMSSD; parasympathetic activity) and the standard deviation of NN intervals (SDNN; overall variability). Frequency-domain indices comprised low frequency (LF; sympathetic and partial parasympathetic modulation), high frequency (HF; parasympathetic activity), and the LF/HF ratio (sympathovagal balance).

Physical Fitness Assessments: Cardiorespiratory fitness (VO_{2max}) was estimated using a submaximal graded cycle ergometer test, with VO_{2max} extrapolated from peak heart rate. Muscular strength was assessed with a handgrip dynamometer and a leg strength device measuring maximal seated bilateral force. Muscular endurance was evaluated

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using a standardized 30-second sit-up test.

Exercise Intervention: The intervention group underwent a supervised 12-week combined aerobic and resistance training program (three sessions per week) based on American College of Sports Medicine (ACSM) guidelines.

- Aerobic training: 30 minutes of treadmill walking at 60% of heart rate reserve (HRR), determined using the Karvonen formula, with continuous heart rate monitoring for intensity control.
- Resistance training: Two sets of 8–12 repetitions across nine machine-based exercises (curl-ups, leg press, leg extension, leg curl, calf raise, and lat pulldown) at 60–80% of one-repetition maximum (1-RM), estimated by the Fleck and Kramer indirect method.
- Each session included a 10-minute warm-up and cool-down with stretching.

The control group maintained their usual lifestyle without structured exercise.

Fasting blood samples were obtained from the forearm veins following a 10-hour overnight fast. Plasma levels of glucose, insulin, C-peptide, glycated hemoglobin (HbA1c), total cholesterol, triglycerides, LDL-cholesterol, HDL-cholesterol, and C-reactive protein (CRP) were measured using an automated chemistry analyzer. Insulin resistance was estimated using the homeostatic model assessment of insulin resistance (HOMA-IR).

Statistical analysis:

Data were analyzed using SPSS software (version 31, IBM Corp., Armonk, NY, USA). A two-way repeated-measures analysis of variance (ANOVA) was employed to evaluate the effects of group (exercise vs. control), time (pre- vs. post-intervention), and their interaction on the study variables. Statistical significance was set at $p < 0.05$.

Results:

Table 1: Glucose profile parameters of the study population

Variables	Groups	At baseline	After 8 weeks	Student test P Value
Blood glucose (mg/dl)	Exercise group	141.6 ± 11.8	133.8 ± 10.7	0.0008; df = 98; t = 3.4625
	Control group	141.7 ± 8.3	140.4 ± 6.7	0.3909; df = 98; t = 0.8618
Serum Insulin (μU/ml)	Exercise group	12.4 ± 2.1	7.8 ± 2.8	0.0001; df = 98; t = 9.2934
	Control group	6.8 ± 2.8	7.4 ± 2.1	0.2284; df = 98; t = 1.2122
HOMA-IR	Exercise group	4.09 ± 0.1	2.7 ± 0.1	0.0001; df = 98; t = 69.5000
	Control group	2.37 ± 0.1	2.56 ± 0.2	0.594; df = 98; t = 0.5356

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C – peptide	Exercise group	2.3 ± 0.5	2.5 ± 0.6	0.0001; df = 98; t = 6.0083
	Control group	1.8 ± 0.5	1.9 ± 0.7	0.4131; df = 98; t = 0.8220
HbA1c (gm%)	Exercise group	6.5 ± 0.2	5.8 ± 0.2	0.0001; df = 98; t = 17.5000
	Control group	6.5 ± 0.4	6.4 ± 0.7	0.3826; df = 98; t = 0.8771

Table 1 shows glucose profile parameters of the study population. Plasma glucose, serum insulin, HOMA-IR, C-peptide, and HbA1c showed significant difference when compared between baseline and after exercise in the exercise group of 50 individuals. On the contrary the same parameters showed no significant difference in the control group individuals between pre-exercise and after a period of eight weeks.

Table 2: Lipid profile parameters of the study population

Variables	Groups	At baseline	After 8 weeks	Student test P Value
Total Cholesterol (mg/dl)	Exercise group	191.3 ± 28.3	179 ± 22.1	0.0201; df = 98; t = 2.3631
	Control group	188.5 ± 49.4	186.1 ± 47.8	0.8055; df = 98; t = 0.2469
Triacylglycerols (mg/dl)	Exercise group	125.6 ± 23.9	96.2 ± 21.9	0.0001; df = 98; t = 8.1466
	Control group	121.6 ± 18.1	119.9 ± 19.2	0.6497; df = 98; t = 0.4556
LDL-cholesterol (mg/dl)	Exercise group	127.9 ± 11.8	120.4 ± 16.2	0.0095; df = 98; t = 2.6461
	Control group	122.3 ± 21.7	115.6 ± 48.2	0.3723; df = 98; t = 0.8963
HDL cholesterol (mg/dl)	Exercise group	44.4 ± 11.2	53.2 ± 12.6	0.0004; df = 98; t = 3.6911
	Control group	49.4 ± 7.9	49.1 ± 9.1	0.8606; df = 98; t = 0.1760

Table 2 shows lipid profile parameters of the study population. Total cholesterol, triacylglycerols, LDL-cholesterol, and HDL-cholesterol showed significant difference when compared between baseline and after exercise in the exercise group of 50 individuals. On the contrary the same parameters showed no significant difference in the control group individuals between pre-exercise and after a period of eight weeks.

Table 3: C-reactive protein in the study population

Variables	Groups	At baseline	After 8 weeks	Student test P Value
C-Reactive Protein (mg/L)	Exercise group	0.3 ± 0.07	0.2 ± 0.09	0.0001; df = 98; t = 6.2017
	Control group	0.131 ± 0.02	0.122 ± 0.03	0.0624; df = 98; t = 1.9812

Table 3 shows C-reactive protein parameters in the study population. It showed significant differences when compared between baseline and after exercise in the exercise group of 50 individuals. On the contrary the same parameter showed no significant difference in the control group individuals between pre-exercise and after a period of eight weeks.

Discussion:

The present study demonstrates that eight weeks of structured exercise intervention significantly improved multiple metabolic parameters in the intervention group, including plasma glucose, serum insulin, HOMA-IR, C-peptide, HbA1c, lipid indices, and CRP, whereas no comparable changes were observed in the control group. These findings provide strong evidence that regular physical activity exerts beneficial effects on glucose homeostasis, lipid metabolism, and systemic inflammation, supporting its role as a first-line, non-pharmacological strategy for the prevention and management of metabolic disorders.

Effect of Exercise on Glycemic Control

The observed reductions in plasma glucose and HbA1c reinforce the role of exercise in long-term glycemic regulation. Previous Indian studies have reported similar outcomes, with Mohan et al. and Prajapati et al. ^[1,2] demonstrating improvements in fasting glucose and HbA1c following structured aerobic and resistance training. International evidence further corroborates these results. Colberg et al., ^[3] in the American Diabetes Association position statement, emphasized that exercise consistently lowers HbA1c by 0.6–0.9% in type 2 diabetes patients, independent of weight loss. More recently, Brouwers et al. ^[4] confirmed that eight weeks of supervised aerobic training improves glycemic indices in individuals with impaired glucose tolerance. Collectively, these findings highlight exercise as a robust modulator of glycemic status across populations.

Effect on Insulin Sensitivity (Serum Insulin and HOMA-IR)

Reductions in serum insulin and HOMA-IR observed in this study reflect improved insulin sensitivity, a critical factor in delaying the progression from insulin resistance to type 2 diabetes. Indian cohorts ^[5,6] and international studies ^[6,8] consistently demonstrate that both aerobic and resistance training lower fasting insulin and enhance insulin signaling pathways, thereby facilitating glucose uptake in skeletal muscle. The present findings thus align with mechanistic evidence that exercise directly improves insulin receptor sensitivity and downstream metabolic regulation.

C-Peptide and Beta-Cell Function

The improvement in C-peptide levels in the exercise group suggests enhanced beta-cell function and insulin secretory capacity. Indian data from Sharma et al. ^[9] and international evidence from Åkerström et al. demonstrate that structured aerobic and resistance training can augment C-peptide secretion, possibly by preserving beta-cell integrity and

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responsiveness to glucose load. These results indicate that exercise not only improves peripheral insulin sensitivity but also contributes to pancreatic beta-cell preservation, particularly in early metabolic dysfunction.

Impact on Lipid Profile

Exercise also produced favorable alterations in lipid metabolism, with reductions in total cholesterol, triglycerides, and LDL-C, alongside increases in HDL-C. Similar findings have been reported in Indian cohorts [11,12, 13, 16, 20,21] as well as global studies [22,23]. Mechanistically, exercise is known to enhance lipoprotein lipase activity, increase hepatic LDL receptor expression, and stimulate HDL biogenesis and reverse cholesterol transport, collectively reducing cardiovascular risk burden.

Anti-Inflammatory Effects (CRP Reduction)

The significant decline in CRP levels following exercise underscores the anti-inflammatory effects of physical activity. Prior Indian studies [24, 25] and global reports [26-28] similarly document reductions in CRP with structured exercise interventions. The underlying mechanisms likely involve decreased visceral adiposity, improved insulin sensitivity, and attenuation of pro-inflammatory cytokines such as IL-6 and TNF- α , leading to reduced hepatic CRP synthesis [29]. Importantly, the present findings demonstrate that even an eight-week intervention can induce meaningful reductions in systemic inflammation.

Control Group Comparison

The absence of significant changes in the control group reinforces the critical role of active intervention in modifying metabolic risk. Consistent with prior Indian and international observations, sedentary individuals tend to maintain or worsen glycemic, lipid, and inflammatory profiles over time, underscoring the health risks of lifestyle inertia.

Conclusion

In summary, this study provides robust evidence that an eight-week structured exercise regimen yields substantial improvements in glycemic control, insulin sensitivity, beta-cell function, lipid metabolism, and systemic inflammation in an Indian cohort. These findings highlight exercise as a powerful, non-pharmacological approach with broad metabolic benefits. Given the rising burden of diabetes, dyslipidemia, and inflammation-driven cardiovascular risk in India and globally, incorporation of structured exercise into routine clinical and public health practice should be prioritized.

Conflict of interest:

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There is no conflict of interest among the present study authors.

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