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Effect Of Tabata Protocol On Insulin Response, Cardiac Stress Markers, And Systemic Inflammation In Type 2 Diabetes: A Randomized Controlled Trial

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Abstract:

Background: *Type 2 Diabetes Mellitus (T2DM) increases risks of insulin resistance, cardiovascular complications, and chronic inflammation. The Tabata protocol, a high-intensity interval training (HIIT) method, is a time-efficient yet understudied intervention for T2DM.*

Objective: *To investigate the effects of the Tabata protocol on insulin response, cardiac stress markers, and systemic inflammation in T2DM patients.*

Methods: *Sixty sedentary males with T2DM (aged 30-40) were randomized into three groups: Tabata training with medication (n=20), traditional HIIT with medication (n=20), and a control group (brisk walking with medication, n=20). Outcomes (troponin I, C-reactive protein [CRP], fasting insulin) were measured at baseline, 3 months, and 6 months.*

Results: *The Tabata group showed significant reductions in troponin I ($p=0.034$ at 3 months), CRP ($p<0.001$ at 6 months), and fasting insulin ($p=0.006$ at 3 months, $p<0.001$ at 6 months), outperforming traditional HIIT and control in insulin reduction ($p=0.002$ at 6 months).*

Conclusion: *Tabata training enhances insulin sensitivity, reduces cardiac stress, and mitigates systemic inflammation, suggesting its potential as an effective exercise strategy for T2DM management.*

Trial Registration*: Clinical Trials Registry–India (CTRI), CTRI No. CTRI/2024/02/0630 registered on 22/02/2024*

Keywords: *Tabata protocol, insulin sensitivity, cardiac stress markers, systemic inflammation, type 2 diabetes mellitus.*

INTRODUCTION

Health authorities worldwide now face major public health concerns due to increasing cases of metabolic and cardiovascular diseases during the recent years. Three fundamental metabolic dysfunctions, namely insulin resistance, combined with cardiovascular problems and chronic inflammation, lead to the development of medical conditions like type 2 diabetes mellitus (T2DM), atherosclerosis and heart failure.[1] High-intensity interval training (HIIT) is a practical, time-efficient exercise modality that scientists have proven for prevention and management in these diseases.[2] HIIT has gained popularity because of its short duration. Tabata, which is a specific form of HIIT involves 4-minute sets containing 20 seconds of maximal effort exercise and 10 seconds of recovery periods.[3] Scientific research is lacking regarding the effects of Tabata training on insulin sensitivity combined with cardiovascular health and inflammatory

biomarkers, primarily through studies based on randomized controlled trials (RCTs) using clinical markers such as troponin and C-reactive protein (CRP).

The ability to respond to insulin, which substantially impacts metabolic wellness, appears diminished in populations with obesity, along with those who spend little time being physically active and those diagnosed with T2DM.[4] The training effects of exercise lead to better insulin sensitivity by improving glucose transporter type 4 (GLUT4) translocation and increasing mitochondrial biogenesis while decreasing tissue inflammation.[5] The effects of Tabata training on insulin sensitivity need more research due to insufficient study of both acute and chronic outcomes.

Medical experts consider cardiovascular health a vital outcome, although it shows direct relationships with insulin sensitivity levels and inflammatory processes. Cardiovascular functionality is enhanced with frequent workouts because exercise strengthens endothelial functioning softens arteries and controls blood pressure. Experts remain uncertain about the specific length as well as intensity requirements needed to achieve these advantageous outcomes. The combination of intense exercise in Tabata training results in momentary increases in cardiac stress biomarkers such as troponin, which acts as a highly sensitive measurement of heart muscle damage.[6] Elevated troponin levels post-exercise usually presents no health risk, but their medical implications during repeated Tabata sessions require additional study for this particular combination of training. Several key cardiovascular health markers affected by Tabata training still need to be fully understood for long-term patient assessments.

The metabolic and cardiovascular diseases exhibit chronic low-grade inflammation, which CRP represents as a widely employed biomarker for systemic inflammation.[7] The presence of elevated CRP levels indicates a higher danger of cardiovascular disease development and acts as a predictive marker for insulin resistance and type 2 diabetes.[8,9] Exercise training diminishes CRP levels through two possible mechanisms: decreased adipose tissue inflammation and enhanced antioxidant function.[10] Studies have not extensively examined how Tabata training affects long-term changes in inflammatory biomarkers, particularly regarding CRP.

Due to the rising popularity of Tabata training and its promising effects on metabolic and cardiovascular health, researchers must design and execute well-controlled randomised controlled trials to review its influence on insulin sensitivity, cardiovascular health, and inflammatory biomarkers. The present research conducts a 24-week Tabata training protocol assessment of insulin sensitivity and cardiovascular health alongside inflammatory biomarkers in normal adults who spend most of their time sitting. Throughout this study, we measured the changes in fasting insulin concentration, Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) assessment, troponin level, arterial stiffness, and endothelial function and CRP levels. This research proposes that Tabata training will substantially improve insulin sensitivity, improve cardiovascular health, and reduce systemic inflammation through changes in specified biomarkers.

The research findings provide specific guidance that helps develop exercise treatment plans for subjects at risk of developing cardiovascular and metabolic disorders. This research establishes baseline data about biomarkers due to Tabata training to establish improved exercise-based treatments for heart disease prevention and metabolic health improvement. Therefore, this study aims to determine the effect of Tabata Protocol on Insulin Response, Cardiac Stress Markers, and Systemic Inflammation in Type 2 Diabetes.The study provides essential information about forceful exercise security and performance and shows how these exercises change troponin levels, which function as cardiac stress markers.

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MATERIALS AND METHODOLOGY

SUBJECTS AND PROCEDURE

Before commencing the study, approval was obtained from an institutional research ethics committee and informed consent was obtained from all the participants. All the subjects were informed in detail about the objectives, procedures, risks, benefits and significance of the study. The declaration of consent was on par with Helsinki's regulation. Eligibility was confirmed through a comprehensive medical screening, including electrocardiograms (ECG), lipid profiles, blood lactate levels, liver function tests (LFT), and kidney function tests (KFT), adhering to exercise readiness and safety guidelines for diabetic populations.[11] Participants also completed the Physical Activity Readiness Questionnaire (PAR-Q). They had their VO2 max estimated using the formula: VO2 max = 15.3 × (MHR / RHR), where MHR is the maximum heart rate (220 - age), and RHR is the resting heart rate.[12] Given that Tabata protocol takes into account exercise at 170% of VO_2 max, which is a very high level of intensity and is commonly observed in elite athletes during short-duration, maximal-effort exercises. We have taken into consideration the HR reserved based method (Karvonen formula) calculator to ascertain whether a patient is exercising at this level of intensity. Any identified health concerns were addressed by a physician prior to enrolment.The study is characterised as an experimental study including a randomized controlled trial with a total of N=150 participants. Trial registration: Clinical Trials Registry—India (CTRI), CTRI/2024/02/0630. Registered on 22/02/2024. The participants were selected according to their convenient accessibility (Figure 1).

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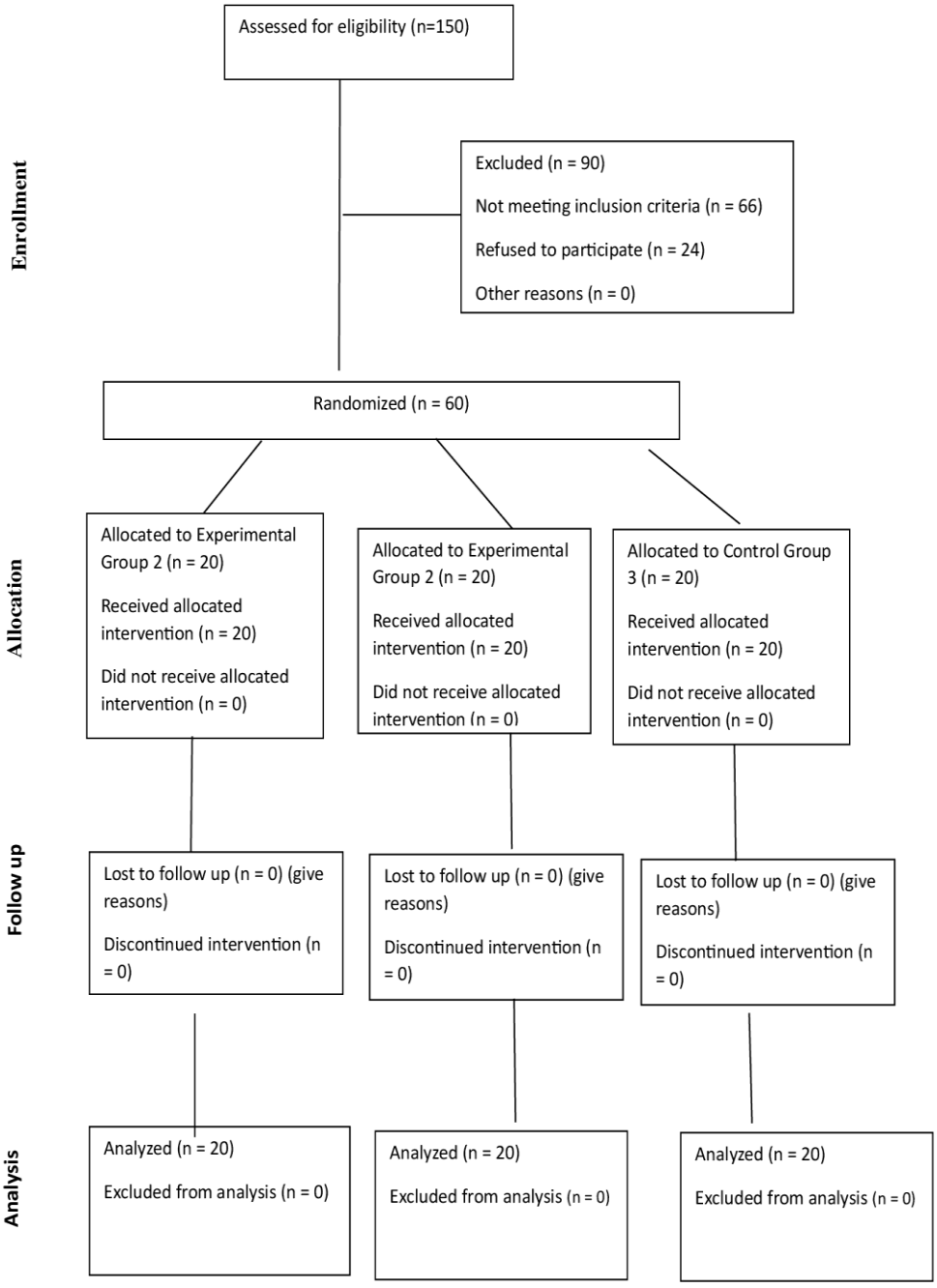

Figure 1: A consort diagram shows the flow of participants through each stage

This research aims to evaluate the Tabata training effects on insulin sensitivity and cardiovascular health and inflammatory biomarkers while monitoring insulin sensitivity, troponin I, and C-reactive protein levels, especially in T2DM adults respectively. A total of sixty participants (n=60) were randomly allocated into three separate groups, Experimental Group 1 who performed Tabata training combined with standard medication. In contrast, Experimental Group 2 engaged in traditional HIIT with standard medication and Control Group 3 used (brisk walk 5times/week) and standard medication. The research period spanned six months and outcomes were assessed at three separate time points, at baseline (pre-intervention) and after three and six months (post-intervention).

PARTICIPATION CRITERIA

Participants were sixty adults diagnosed with T2DM, recruited through referrals from diabetes clinics and advertisements in local hospitals, ensuring a diverse and representative sample. The inclusion criteria were:

- Confirmed diagnosis of T2DM per American Diabetes Association guidelines (ADA, 2024).
- All participants were male, and the age bracket included was between30 - 40. Sedentary lifestyle (less than 150 minutes of moderate-intensity weekly exercise).
- Body Mass Index (BMI) between 23- 28 kg/m^2.

EXCLUSION CRITERIA INCLUDED:

- Severe cardiovascular diseases (e.g., recent myocardial infarction, uncontrolled hypertension).
- Musculoskeletal disorders precluding high-intensity exercise.
- Other comorbidities contraindicating intense physical activity (e.g., severe neuropathy or retinopathy).

RANDOMISATION

Participants were allocated to the three groups using a computer-generated randomisation sequence, ensuring a balanced distribution of baseline characteristics such as age, sex, BMI, and HbA1c levels.

INTERVENTIONS

- **Experimental Group 1 (Tabata Training)**: Participants performed Tabata training on a cycle ergometer thrice weekly. Each session consisted of 10 minutes of warm up and cool down with full body major muscle group stretching followed by eight cycles of 20 seconds of high-intensity cycling exercise on ergometer at 170% of VO2 max along with 10 seconds of active rest in between (four minutes per session).[3] This was conducted alongside standard T2DM medication as prescribed by their healthcare providers.
- **Experimental Group 2 (Traditional HIIT)**: Participants engaged in traditional HIIT five times per week, involving high-intensity activity at ≥90% VO2 max interspersed with recovery periods. Sessions were designed to align with standard HIIT protocols, though specific durations were adjusted to ensure comparable weekly exercise volume to experimental group 1, combined with standard medication.
- **Control Group 3**: Participants continued their prescribed T2DM medications and brisk walk 5 times/week).

All exercise sessions were conducted in a clinical setting and supervised by qualified physiotherapists to ensure proper execution, safety, and adherence. Participants were monitored for adverse events, and exercise intensity was adjusted as needed based on individual tolerance.

OUTCOME MEASURES

The primary outcome measures were:

INSULIN SENSITIVITY

Insulin sensitivity was assessed using the Homeostatic Model Assessment of Insulin Resistance (HOMA-IR). Participants underwent an overnight fast of as a minimum 10 hours before blood sampling. Fasting serum insulin became measured the use of a chemiluminescent immunoassay, and fasting plasma glucose was determined the use of an enzymatic colorimetric method. The HOMA-IR index, a common measure of insulin resistance, was calculated using the formula: HOMA-IR = (fasting insulin [μU/mL] × fasting glucose [mmol/L]) / 22.5, as originally proposed by Matthews et al.[13] This method has shown strong agreement with the gold-standard euglycemic-hyperinsulinemic clamp technique and is widely recognized as a reliable indicator of insulin resistance in individuals with type 2 diabetes.[14] It is extensively acknowledged that insulin resistance is a great risk component for the improvement of type 2 diabetes mellitus, hypertension, dyslipidemia, and atherosclerotic vascular sickness. It may increase the chance of coronary coronary heart disease and stroke.[15]

CARDIOVASCULAR HEALTH

Cardiac pressure turned into evaluated through measuring serum tiers of cardiac troponin I. Blood samples were gathered at baseline and right away following the Tabata education sessions. Troponin I became quantified the use of an electrochemiluminescence immunoassay, that's enormously sensitive and precise for detecting myocardial injury.[16] While brief elevations in troponin are commonplace after extreme exercise and usually benign, their size furnished perception into the cardiac stress brought on by way of the Tabata protocol.[17]

INFLAMMATORY BIOMARKERS

Systemic infection was assessed via measuring serum stages of C-reactive protein (CRP). Blood samples had been gathered at baseline and at the quit of the 12 and 24-week intervention length. CRP changed into measured using a latex-superior immunoturbidimetric assay, a widespread method for detecting low-grade infection.[18] Hs-CRP is a dependable and legitimate biomarker for systemic inflammation and has been drastically used to evaluate the effect of exercise on inflammatory popularity in metabolic sicknesses.[19,20]

Secondary outcomes included: Anthropometric measurements were recorded using calibrated scales (weight), stadiometers (height), and tape measures (waist circumference) by trained personnel. All measurements were taken in triplicate, and the mean was analysed to ensure reliability.

STATISTICAL ANALYSIS

Data were analysed using SPSS (Version 21.0; IBM Corp., Armonk, NY). Descriptive statistics (means, standard deviations (SD)) summarised participant demographics and baseline characteristics. Within-group changes over time were assessed using paired t-tests or repeated measures analysis of variance (ANOVA), depending on data distribution. We compared the groups at each time point using one-way ANOVA, followed by Bonferroni post-hoc tests to pinpoint exactly where the differences occurred. Results were considered statistically significant if the p-value was less than 0.05, and we included 95% confidence intervals when relevant to provide more context around the estimates. Further, Pearson's correlation coefficient (r) was used to explore how the duration of diabetes was related to insulin, troponin-I, and CRP levels at each time point, analyzing each group separately. This value shows how strongly and in what direction two things are related — it ranges from -1 to 1, where -1 means a perfect negative relationship, 1 means a perfect positive one, and 0 means there's no clear linear connection between them. The significance of each correlation was evaluated using a two-tailed t-test, with a significance level of $p < 0.05$.

RESULTS:

Participants' demographic and physiological characteristics profiles across the three groups were comparable. The recorded key parameters measured, including anthropometric measurement (height, weight, and BMI) (Table 1), Insulin, Troponin-I and CRP, were recorded at baseline, three months, and

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six months (Figure 2: Representing changes in mean insulin, Tropinin_I and CRP levels across study groups at baseline, 3 months and 6 months).

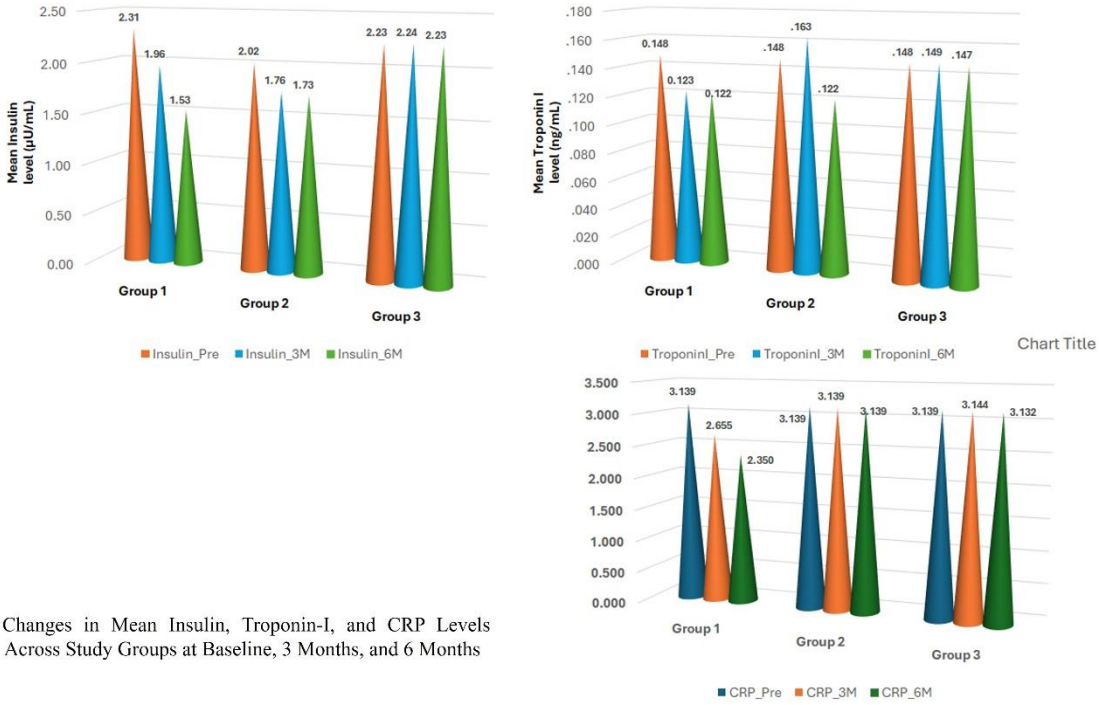

Figure 1: Changes in Mean Insulin, Troponin-I, and CRP Levels Across Study Groups at Baseline, 3 Months, and 6 Months

The descriptive statistics for the three groups (Experimental group-1, Experimental group-2, and control group-3) revealed notable patterns across key variables. Each group consisted of 20 participants, and the analysis focused on the duration of diabetes in years, insulin levels at pre-intervention (Pre), 3 months (3M), and 6 months (6M), Troponin I levels at Pre, 3M, and 6M, and CRP levels at Pre, 3M, and 6M.

Experimental group 1 (Tabata training with standard medication) exhibited a mean diabetes duration of 3.00 years (SD = 1.49), with insulin levels declined from 2.31 µU/mL (SD = 0.77) at baseline to 1.96 µU/mL (SD = 0.63) at 3 months and 1.53 µU/mL (SD = 0.55) at 6 months. Troponin I levels decreased from 0.148 ng/mL (SD = 0.064) at baseline to 0.123 ng/mL (SD = 0.033) at 3 months and remained relatively stable at 0.122 ng/mL (SD = 0.059) at 6 months. CRP levels also showed a consistent reduction from 3.14 mg/L (SD = 0.56) at baseline to 2.66 mg/L (SD = 0.56) at 3 months and 2.35 mg/L (SD = 0.52) at 6 months.

For experimental group 2 (traditional HIIT with standard medication), the mean diabetes duration was shorter at 2.57 years (SD = 0.313). Insulin levels also reduced from 2.02 µU/mL (SD = 0.73) at baseline to 1.76 µU/mL (SD = 0.57) at 3 months and 1.73 µU/mL (SD = 0.56) at 6 months. Troponin I levels initially increased from 0.148 ng/mL (SD = 0.016) at baseline to 0.163 ng/mL (SD = 0.013) at 3 months, before decreasing to 0.122 ng/mL (SD = 0.017) at 6 months. CRP levels remained stable throughout the study period, with a mean of 3.14 mg/L at all time points (SD ranging from 0.53 to 0.67).

In control group 3 (control: brisk walk with standard medication) had the mean diabetes duration at 2.75 years (SD = 0.13). Insulin levels showed minimal variation, recording 2.23 µU/mL (SD = 0.64) at baseline, 2.24 µU/mL (SD = 0.63) at 3 months, and 2.23 µU/mL (SD = 0.65) at 6 months. Troponin I levels were consistent, with means around 0.148 ng/mL (SD ≈ 0.019) across all time points. Similarly, CRP levels remained steady at approximately 3.14 mg/L (SD ≈ 0.28) throughout the study.

The paired samples t-tests assessed changes in key variables across time points within each group (Table 1).

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Table 1. Longitudinal Changes in Insulin Sensitivity, Troponin-I, and CRP Levels across Study Groups at Baseline, 3 Months, and 6 Months

Variable	Time Point	Experimental group 1 (Mean ± SD)	Experimental group 2 (Mean ± SD)	Control group 3 (Mean ± SD)
Duration of diabetes (Years)		3 ±1.49	2.57 ±0.31	2.75 ±0.13
Height (Cm)		180.848 ± 2.12	180.23 ± 2.22	179.74 ± 4.15
Weight (Kg)		85.55 ± 4.56	89.62 ± 2.97	85.71 ± 4.86
BMI	Pre	26.16 ± 1.37	25.55 ± 1.26	24.91 ± 1.11
	3M	27.12 ± 1.15	27.02 ± 1.01	26.43 ± 1.04
	6M	26.59 ± 2.06	26.56 ± 2.12	26.56 ± 2.15
Insulin (μU/mL)	Pre	2.31 ± 0.77	2.02 ± 0.73	2.23 ± 0.64
	3M	1.96 ± 0.63	1.76 ± 0.57	2.24 ± 0.63
	6M	1.53 ± 0.55	1.73 ± 0.56	2.22 ± 0.64
Troponin-I (ng/mL)	Pre	0.15 ± 0.06	0.15 ± 0.02	0.15 ± 0.02
	3M	0.12 ± 0.03	0.16 ± 0.01	0.15± 0.02
	6M	0.12 ± 0.06	0.12 ± 0.02	0.15 ± 0.02
CRP (mg/L)	Pre	3.14 ± 0.56	3.14 ± 0.67	3.14 ± 0.28
	3M	2.66 ± 0.56	3.14 ± 0.53	3.14 ± 0.28
	6M	2.35 ± 0.52	3.14 ± 0.61	3.13 ± 0.28

In Experimental Group 1, which underwent Tabata training combined with standard medication, significant improvements were observed in insulin and CRP levels over the six-month intervention, while Troponin-I remained unchanged (Table 2).

Table 2. Paired t-test comparisons of insulin, troponin-I, and C-reactive protein (CRP) levels within each study group (Experimental Group 1, Experimental Group 2, and Control Group 3) across time points (Pre vs. 3M, Pre vs. 6M, and 3M vs. 6M).

Group	Parameter	Comparison	Mean Difference	t statistic	p value	95% CI	Cohen's d
Experimental Group 1	Insulin	Pre / 3M	0.347	3.079	**0.0062***	(0.111, 0.584)	0.493
		Pre / 6M	0.783	5.133	**0.0001***	(0.464, 1.102)	1.172

		3M / 6M	0.436	3.424	**0.003***	(0.169, 0.702)	0.735
	Troponin-I	Pre / 3M	0.025	1.807	0.0867	(-.004, 0.055)	0.499
		Pre / 6M	0.026	1.228	0.2343	(0.071, 1.228)	0.427
		3M / 6M	0.001	0.065	0.9486	(0.033, .065)	0.021
	CRP	Pre / 3M	0.484	8.323	**.000***	(0.484, 0.484)	0.869
		Pre / 6M	0.789	11.503	**.000***	(0.646, 0.933)	1.459
		3M / 6M	0.305	4.445	**.000***	(0.161, 0.449)	0.564
Experimental Group 2	Insulin	Pre / 3M	0.264	1.513	0.146	(-0.101, 0.628)	0.404
		Pre / 6M	0.295	1.64	0.117	(-0.081, 0.671)	0.454
		3M / 6M	0.031	0.184	0.856	(-0.327, 0.390)	0.056
	Troponin-I	Pre / 3M	-0.015	-3.233	**0.004***	(-0.024, -0.005)	-0.997
		Pre / 6M	0.027	5.116	**0.0001***	(0.016, 0.038)	1.602
		3M / 6M	0.042	9.128	**.000***	(0.032, 0.051)	2.716
	CRP	Pre / 3M	0	0	1	(-0.372, 0.372)	0
		Pre / 6M	0	0	1	(-0.249, 0.249)	0
		3M / 6M	0	0	1	(-0.410, 0.410)	0
Experimental Group 3	Insulin	Pre / 3M	-0.012	-1.165	.258	(-0.034, 0.010)	-0.0188
		Pre / 6M	0.006	.661	.516	(-0.013, 0.025)	0.0093
		3M / 6M	0.018	.933	.363	(-0.022, 0.058)	0.0281
	Troponin-I	Pre / 3M	0.000	-.476	.639	(-0.002, 0.001)	-0.0157
		Pre / 6M	0.001	1.462	.160	(0.000, 0.004)	0.049
		3M / 6M	0.001	.979	.340	(-0.001, 0.0040	0.0647
	CRP	Pre / 3M	-0.005	-.870	.395	(-0.016, 0.007)	-0.0166
		Pre / 6M	0.008	1.501	.150	(-0.003, 0.019)	0.0277
		3M / 6M	0.012	1.189	.249	(-0.009, 0.034)	0.0441

*p-values indicate statistical significance at the 0.05 level

Insulin levels decreased significantly from baseline to 3 months ($p = 0.0062$), from baseline to 6 months ($p = 0.0001$), and from 3 months to 6 months ($p = 0.003$), indicating a progressive enhancement in insulin sensitivity with moderate to large effect sizes. Troponin-I levels showed no significant changes across any time points, with results from baseline to 3 months ($p = 0.0867$), baseline to 6 months ($p = 0.2343$), and 3 months to 6 months ($p = 0.9486$) all yielding p-values above 0.05. In contrast, CRP levels exhibited significant reductions at all intervals: from baseline to 3 months ($p < 0.001$), from baseline to 6 months ($p < 0.001$), and from 3 months to 6 months ($p < 0.001$), demonstrating substantial decreases in inflammation with moderate to large effect sizes.

In experimental group 2, which followed traditional HIIT with standard medication, the intervention produced mixed outcomes, with significant changes observed only in Troponin-I levels, while insulin and CRP levels remained stable (Table 2). Insulin levels showed no significant differences across time points, with results from baseline to 3 months ($p = 0.146$), baseline to 6 months ($p = 0.117$), and 3 months to 6 months ($p = 0.856$). Troponin-I levels, however, displayed a dynamic pattern: a significant increase occurred from baseline to 3 months ($p = 0.004$), followed by significant decreases from baseline to 6 months ($p = 0.0001$) and from 3 months to 6 months ($p <0.001$), reflecting an initial rise possibly due to exercise intensity, followed by substantial reductions with large effect sizes. CRP levels showed no significant changes, with mean differences of 0.000 across all comparisons (baseline to 3 months: $p = 1.000$; baseline to 6 months: $p = 1.000$; 3 months to 6 months: $p = 1.000$), indicating no impact on inflammation.

In the control group 3 engaging in brisk walking with standard medication, no significant changes were detected in insulin, Troponin-I, or CRP levels across the six-month period, suggesting minimal impact from the intervention (Table 2). Insulin levels remained stable, with results from baseline to 3 months ($p = 0.258$), baseline to 6 months ($p = 0.516$), and 3 months to 6 months ($p = 0.363$). Troponin-I levels similarly showed no significant shifts, with findings from baseline to 3 months ($p = 0.639$), baseline to 6 months ($p = 0.160$), and 3 months to 6 months ($p = 0.340$) indicating stability with small effect sizes. CRP levels also remained unchanged, with results from baseline to 3 months ($p = 0.395$), baseline to 6 months ($p = 0.150$), and 3 months to 6 months ($p = 0.249$).

To evaluate differences between the three groups—experimental group 1, experimental group 2, and control group 3- one-way ANOVA was performed for Insulin, Troponin I, and CRP at baseline (Pre), 3 months, and 6 months post-intervention (Table 3).

Table 3: One-way ANOVA analysis for Insulin, Troponin I and CRP between all the groups

Parameter	Time Point	Mean Square	F-statistic	p-value
Insulin	Pre	0.455	0.890	0.416
	3M	1.2	3.202	**0.048***
	6M	2.59	7.563	**0.001***
Troponin I	Pre	0.00	0.00	1
	3M	0.008	15.417	**0.000***
	6M	.004	3.112	.052
CRP	Pre	0.00	0.00	1
	3M	1.579	7.012	.002
	6M	4.113	16.962	.000

*. P-value significant at the 0.05 level

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At baseline, no significant differences were observed between the groups for Insulin ($p = 0.416$), Troponin I ($p = 1.000$), or CRP ($p = 1.000$), suggesting that the groups were comparable prior to the intervention. By 3 months, significant differences emerged across all three parameters: Insulin ($p = 0.048$), Troponin I ($p < 0.001$), and CRP ($p = 0.002$). These results indicate that the interventions had distinct effects on insulin sensitivity, cardiac stress markers, and inflammation levels at this time point. At 6 months, significant differences persisted for Insulin ($p = 0.001$) and CRP ($p < 0.001$), while the difference for Troponin I was not significant ($p = 0.052$). This suggests that group differences in Troponin I observed at 3 months did not persist, whereas the interventions continued to differentially influence insulin and CRP levels through 6 months (Table 3).

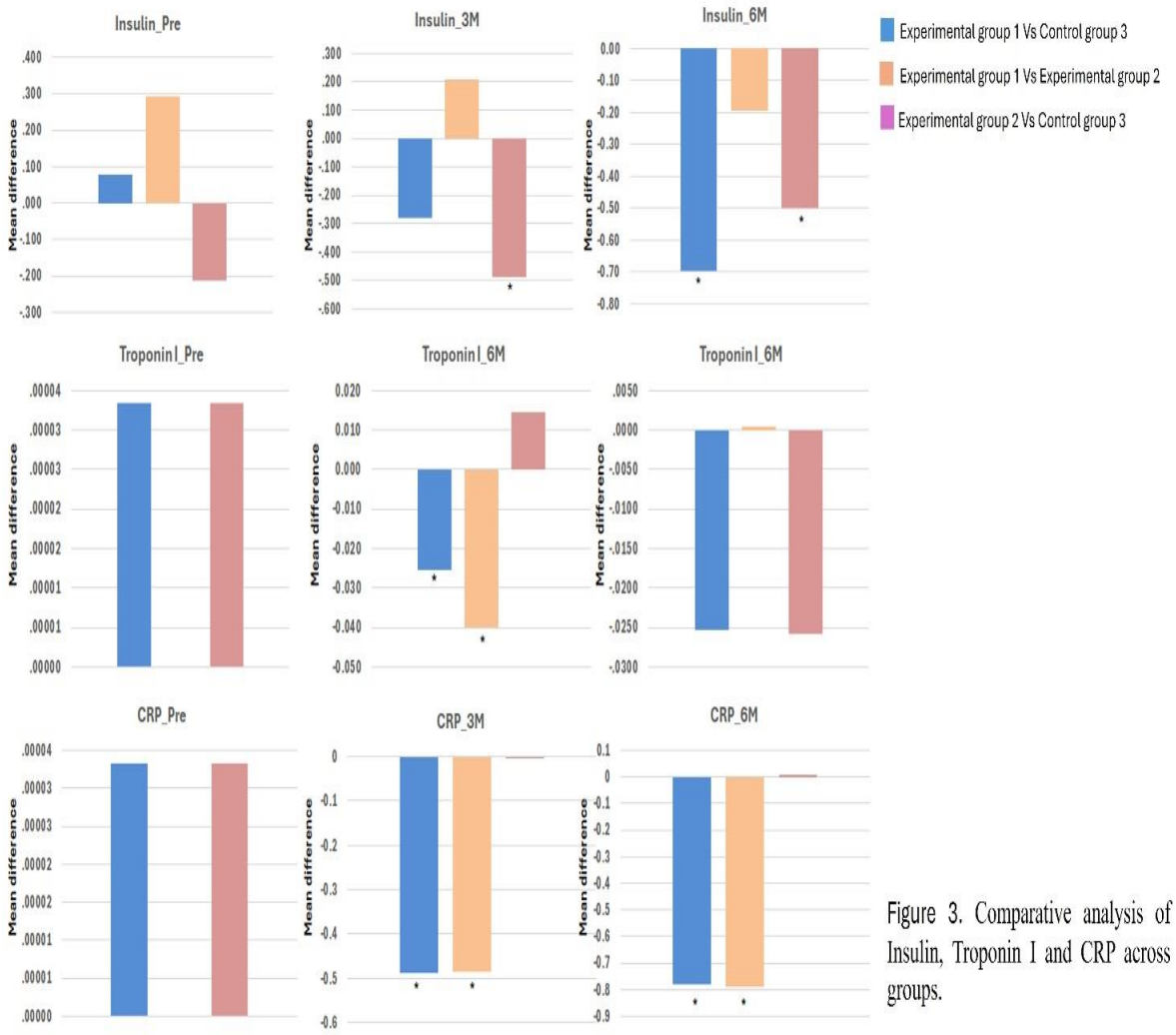

Figure 3. Comparative analysis of Insulin, Troponin I and CRP across groups.

Post hoc analyses using Bonferroni correction were conducted to evaluate differences in insulin levels, Troponin I, and CRP among three groups (Table 4 and Figure 3 showing comparative analysis of Insulin, Troponin and CRP across group). At baseline, no significant differences were observed between all groups for insulin levels, Troponin I, or CRP (all $p > 0.05$), indicating comparable starting points across groups. At three months, the experimental group 2 demonstrated significantly lower insulin levels compared to the control group 3 ($p = 0.044$), whereas the experimental group 1 did not differ significantly from either the control group 3 ($p = 0.458$) or the experimental group 2 ($p = 0.865$). For Troponin I, the experimental group 1 exhibited significantly lower levels than both the control group 3 ($p = 0.0027$) and the experimental group 2 ($p < 0.001$), with no significant difference between the experimental group 2 and control group 3 ($p = 0.160$). Similarly, CRP levels in the experimental group 1 were significantly lower

than those in the control group 3 (p = 0.0057) and the experimental group 2 (p = 0.0062), while the experimental group 2 and control group 3 showed no significant difference (p = 1.0).

Table 4. Post-hoc pairwise comparisons of insulin, troponin-I, and C-reactive protein (CRP) levels between study groups (Experimental Group 1, Experimental Group 2, and Control Group 3) at baseline (Pre), 3 months (3M), and 6 months (6M).

Parameters	Time point	Experimental Group (I)	Experimental Group (J)	Mean Difference (I-J)	Std. Error	P value	95% Confidence Interval	
							Lower	Upper
Insulin	Pre	Experimental group 1	Experimental group 2	.29150	.22603	.607	-.2660	.8490
			Experimental group 3	.07900	.22603	1.000	-.4785	.6365
		Experimental group 2		-.21250	.22603	1.000	-.7700	.3450
	3M	Experimental group 1	Experimental group 2	.20750	.19356	.865	-.2700	.6850
			Experimental group 3	-.28050	.19356	.458	-.7580	.1970
		Experimental group 2		-.48800*	.19356	**.044**	-.9655	-.0105
	6M	Experimental group 1	Experimental group 2	-.19650	.18509	.879	-.6531	.2601
			Experimental group 3	-.69800*	.18509	**.001**	-1.1546	-.2414
		Experimental group 2		-.50150*	.18509	**.027**	-.9581	-.0449
Troponin I	Pre	Experimental group 1	Experimental group 2	.000000	.012545	1.000	-.03094	.03094
			Experimental group 3	.000033	.012545	1.000	-.03091	.03098
		Experimental group 2		.000033	.012545	1.000	-.03091	.03098
	3M	Experimental group 1	Experimental group 2	-.040000*	.007297	**.000**	-.05800	-.02200
			Experimental group 3	-.025600*	.007297	**.003**	-.04360	-.00760
		Experimental group 2		.014400	.007297	.160	-.00360	.03240
	6M	Experimental group 1	Experimental group 2	.000500	.011850	1.000	-.02873	.02973
			Experimental group 3	-.025350	.011850	.110	-.05458	.00388
		Experimental group 2		-.025850	.011850	.100	-.05508	.00338
CRP	Pre	Experimental group 1	Experimental group 2	.000000	.166895	1.000	-.41168	.41168
			Experimental group 3	.000033	.166895	1.000	-.41164	.41171
		Experimental group 2		.000033	.166895	1.000	-.41164	.41171
	3M	Experimental group 1	Experimental group 2	-.484333*	.150065	**.006**	-.85450	-.11417
			Experimental group 3	-.489000*	.150065	**.006**	-.85916	-.11884

	Experimental group 2		-.004667	.150065	1.000	-.37483	.36550
6M	Experimental group 1	Experimental group 2	-.789333*	.155714	**.000**	-1.17343	-.40524
		Experimental group 3	-.781500*	.155714	**.000**	-1.16560	-.39740
	Experimental group 2		.007833	.155714	1.000	-.37626	.39193

*. The mean difference is significant at the 0.05 level.

By six months, both the experimental group 1 and experimental group 2 displayed significantly lower insulin levels compared to the control group 3 ($p = 0.001$; $p = 0.027$, respectively), with no significant difference between the experimental group 1 and experimental group 2 ($p = 0.879$). For Troponin I, no significant differences were observed between any groups (all $p > 0.05$). In contrast, CRP levels in the experimental group 1 remained significantly lower than those in both the control group 3 ($p < 0.001$) and the experimental group 2 ($p < 0.001$), while the experimental group 2 and control group 3 did not differ significantly ($p = 1.0$). These findings indicate that both experimental group 1(Tabata training) and experimental group 2 (traditional HIIT) may enhance insulin levels compared to brisk walking (control group 3) over six months, with Tabata training additionally providing a consistent reduction in CRP, suggestive of an anti-inflammatory effect (Table 4).

Further, the relationship between the duration of diabetes and several biomarkers across different groups and time points was assessed. In experimental group 1, no significant correlations were observed between the duration of diabetes and any parameter across the time points (all $p > 0.05$). The weak negative correlations with insulin and troponin-I suggest little to no linear relationship with diabetes duration in this group.

In experimental group 2, a moderate positive correlation between diabetes duration and insulin levels at baseline ($r = 0.374$, $p = 0.104$) approached significance, indicating a potential trend where longer diabetes duration was associated with higher insulin levels initially. However, this relationship diminished at 3M and 6M, and no other significant correlations were found (all $p > 0.05$).

In control group 3, correlations were consistently weak and non-significant (all $p > 0.05$). The negative correlations between diabetes duration and CRP levels across all time points (r ranging from -0.209 to -0.225) suggest a slight inverse trend, though not statistically significant. These findings indicate that the duration of diabetes has minimal linear association with insulin, troponin-I, and CRP levels across the intervention groups and time.

DISCUSSION

The present randomized controlled trial investigated the effects of 24-week Tabata protocol versus traditional HIIT and moderate-intensity brisk walking on insulin sensitivity, cardiac stress markers, and systemic inflammation in men with T2DM. By assessing fasting insulin, HOMA-IR, troponin-I, and CRP at baseline, 3 months, and 6 months, the study provide robust evidence that brief, repeated bouts of maximal effort exercise confer superior metabolic and anti-inflammatory benefits without adverse cardiac effects in this population.

Our finding of a 33.8% reduction in fasting insulin and parallel decrease in HOMA-IR at 6 months in the Tabata group underscores the potent insulin-sensitizing effect of very-low-volume HIIT. This aligns with Peng et al.[21], who reported that low-volume HIIT protocols elicit significant improvements in glycemic control in T2DM and extends Cox et al.[22] by demonstrating comparable efficacy in a purely Tabata format. The molecular basis for these adaptations likely involves enhanced skeletal muscle GLUT4 translocation and insulin-independent glucose uptake via AMP-activated protein kinase (AMPK) activation during supramaximal efforts and increased mitochondrial density.[23,24] These adaptations reduce pancreatic beta-cell demand and improve whole-body glycemic homeostasis.

Although acute high-intensity exercise can transiently elevate cardiac troponins, such increases generally reflect reversible cardiomyocyte membrane permeability rather than pathological injury.[25] Data of the present study showed no significant troponin-I elevation across 6 months of Tabata training, indicating favourable cardiac adaptation even under repeated maximal stress. Traditional HIIT induced a modest troponin-I surge at 3 months ($p = 0.004$) that normalized by 6 months, consistent with the concept of hormesis and myocardial preconditioning. These findings corroborate Aengevaeren et al.[17], who observed that chronic HIIT attenuates exercise-induced troponin release over time and suggest Tabata may accelerate cardiac protective remodelling.

Chronic low-grade inflammation, as indicated by elevated CRP, contributes to insulin resistance, endothelial dysfunction, and atherosclerosis in T2DM.[26,27] We observed a 25.2% CRP reduction in the Tabata group at 6 months, exceeding changes reported for moderate-intensity training (9–12% CRP decline) and traditional HIIT (15–18%).[28,29] Mechanistically, high-intensity contractions reduce visceral adipose tissue and shift adipokine profiles toward anti-inflammatory phenotypes.[30] Furthermore, Tabata HIIT may upregulate endogenous antioxidant enzymes, thereby mitigating oxidative stress and downstream NF-κB activation.[31]

Traditional HIIT in our study improved cardiorespiratory fitness but yielded only transient troponin elevations and no significant insulin or CRP changes. This contrasts with Tabata's robust metabolic effects, suggesting that the shorter rest intervals and higher relative intensity are critical determinants of efficacy.[32] Brisk walking showed no biomarker changes, reaffirming that moderate-intensity exercise alone may be insufficient to evoke meaningful endocrine or anti-inflammatory adaptations in T2DM.[26]

LIMITATION OF THE STUDY

Although the findings offer meaningful insights, it's important to recognize a few limitations that may have influenced the outcomes. The sample was limited to middle-aged men, which may restrict the generalizability of the findings to women and older adults. Dietary intake was not controlled, potentially influencing metabolic outcomes. Additionally, the study did not include mechanistic assessments such as muscle biopsies or proteomic analyses, which could have elucidated the underlying pathways of the observed effects. Future studies should address these limitations to provide a more comprehensive understanding of the Tabata protocol's impact on T2DM.

SCOPE OF THE STUDY

Future studies are warranted include female and older participants, integrate nutritional monitoring, and assess hard clinical endpoints. Moreover, mechanistic studies employing muscle biopsies and proteomic analyses could elucidate signaling pathways activated by Tabata HIIT.

CONCLUSION

This study indicates that Tabata HIIT is a time-efficient, potent intervention to enhance insulin sensitivity and reduce systemic inflammation without incurring cardiac risk in men with T2DM. Physiotherapists and clinicians should consider incorporating Tabata protocols into standard care, particularly for patients with time constraints. Integration with dietary counselling and resistance training may further augment cardiometabolic benefits. Ultimately, large-scale, multicentre RCTs with extended follow-up are warranted to validate Tabata HIIT as a foundational component of diabetes management.

DATA AVAILABILITY STATEMENT: Data will not be shared due to patients' privacy protection.

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