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Effect of Resistance Training on Heart Rate Variability and Lactate Indices in Type 2 Diabetics

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Abstract

Introduction: Type 2 Diabetes Mellitus (T2DM) is associated with alterations in cardiac autonomic function and lactate metabolism. Heart rate variability (HRV) and blood lactate measurements are useful tools to evaluate these changes, while resistance training (RT) has emerged as an effective non-pharmacological intervention. **Objective:** To investigate the chronic effects of RT on parasympathetic modulation and lactate metabolism in individuals with T2DM. **Methods:** Fourteen volunteers participated, including eight individuals with T2DM (62.37±9.65 years) and six healthy controls (58.67±5.28 years). Participants performed a one-repetition maximum (1RM) test followed by submaximal exercise at 10–50% of 1RM. HRV was analyzed using RMSSD and SD1 indices, and blood lactate was measured at rest and during exercise. The T2DM group completed a 12-week RT program at 60% of 1RM, while controls maintained their usual lifestyle. Bootstrap resampling (1,000 samples) was applied, with significance set at $p<0.05$. **Results:** Lactate responses did not differ significantly before and after training; however, the exercise intensity associated with a significant increase shifted from 30% to 40% of 1RM after RT, suggesting delayed lactate accumulation. No significant lactate changes were observed in controls, and HRV indices remained unchanged in both groups. **Conclusion:** Twelve weeks of RT did not significantly modify cardiac autonomic modulation but may improve oxidative metabolism by delaying lactate accumulation during exercise in individuals with T2DM.

Keywords: Heart rate variability, lactate, resistance training, autonomic nervous system.

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BACKGROUND

Type 2 diabetes mellitus (T2DM) is characterized as a multifactorial metabolic disorder. There are over 400 million people with T2DM, and this number is estimated to increase by 51% by 2045; global public spending on the disease could reach US$ 1.452 billion by 2040[1].

Lactate is a biomarker that can predict the incidence of diabetes independently of other risk factors when measured at absolute rest[2]. Individuals with T2DM tend to exhibit higher baseline blood lactate concentrations due to reduced oxidative capacity, particularly when they have not engaged in physical exercise shortly before sample collection[3]. In addition to its effects on lactate metabolism, T2DM also impacts cardiac autonomic modulation, reducing heart rate variability (HRV), an important tool for assessing the autonomic nervous system (ANS)[4].

Physical exercise (PE) is one of the treatment options for T2DM, it is a beneficial, non-pharmacological lifestyle approach that leads to favorable adaptations. Resistance training (RT) is a form of PE recognized for its efficacy and safety, as it can improve metabolic characteristics and insulin sensitivity[5]. Vascular adaptations resulting from RT can facilitate peripheral blood flow, thereby ameliorating macro and microvascular alterations, furthermore, when performed regularly, RT can contribute to efficient lactate metabolism by enhancing oxidative capacity as well as lactate transport and removal[6]. Thus, the objective of this study was to investigate the effect of resistance training on lactate and parasympathetic HRV indices in individuals with type 2 diabetes.

METHODS

The study was approved by the Research Ethics Committee Involving Human Subjects of the School of Philosophy and Sciences at UNESP, Marília Campus, in accordance with Resolution 466/2012 and its supplementary resolutions issued by the National Health Council (Report number:. 1,779,423). All volunteers were informed about the experimental procedures and the invasive and non-invasive nature of the tests, as well as the confidentiality of the collected data and their identities, and all signed informed consent forms.

The sample size was determined based on variations in RMSSD values between pre- and post-intervention time points from the study by Pagkalos et al. (2008)[7], 13; assuming a Type I error rate of 5% and 80% power, an initial sample of 6 subjects was estimated. Accounting for a 30% attrition rate, nine volunteers were selected. Subsequently, the study power was calculated to be 56%, based on a difference in mean RMSSD of 2.8 ms, a standard deviation of 3.2 ms, and a sample size of 8 subjects. Individuals eligible for the study included non-insulin-dependent type 2 diabetes (T2DM) patients aged 50 to 75 years (both sexes) who had not engaged in resistance training (RT) in the previous six months and were cleared for physical exercise, presenting with associated comorbidities such as dyslipidemia, obesity, and systemic arterial hypertension. Individuals were deemed ineligible if they were smokers or heavy alcohol consumers, or if they had cardiovascular diseases (deep vein thrombosis, angina, arrhythmias, heart failure, or myocardial infarction), diagnosed neoplasms, or pulmonary, renal, or neurological diseases unrelated to their T2DM condition.

The experiments were conducted during the afternoon (between 2:00 PM and 6:00 PM) to standardize the influence of circadian variations on the body, in a climate-controlled room with temperatures between 22˚C and 24˚C and relative humidity between 40% and 60%. Volunteers wore appropriate clothing for the experiments; they did not consume alcoholic beverages or stimulants for 48 hours prior, did not engage in physical activities outside their daily routine, had a regular night's sleep, and consumed a light meal at least two hours before the experiments. All volunteers underwent data collection familiarization procedures at least 24 hours before the actual data collection. A control group (CG) consisting of six apparently healthy individuals was also included; this group underwent all initial and final assessments, with the exception of the resistance training (RT) protocol intervention. The CG was instructed not to alter their habits during the 12-week period.

Based on body composition assessments, we obtained parameters to characterize overall obesity using the Body Mass Index (BMI). Blood pressure (BP) was measured in the supine position after twenty minutes of rest, using a mercury-column sphygmomanometer and the auscultatory method.

Assessment procedures were conducted over three days—with 48- to 72-hour intervals between them—both before and after the resistance training (RT) protocol. Day 1: Anamnesis and clinical/physical assessment. Day 2: Recording of R-R intervals and test

familiarization. Day 3: Maximal and submaximal load testing.

Linear time-domain indices were recorded during the submaximal load test and calculated using Kubios HRV software (version 3.0; University of Kuopio, Finland). These included the root mean square of successive differences between normal R-R intervals (RMSSD) and the standard deviation of point distances from the y = x diagonal on the Poincaré plot (SD1); the latter relates to short-term variability and represents parasympathetic heart rate activity.

On the day of testing, a warm-up was performed beforehand: five minutes of cycle ergometer exercise at an intensity of 50% of heart rate reserve $^{(220 - age - resting\ HR) \times 0.5) + resting\ HR}$; two 15-second stretching sets for each muscle group to be tested; and 10 repetitions of the exercise using only the weight of the machine's mechanism (unloaded).

Subsequently, a one-repetition maximum (1RM) test for knee extension (seated leg extension machine) was performed. The volunteer remained seated with hips and knees flexed at 90°, back supported against the machine's backrest, and the lever arm's foam pads positioned against the anterior aspect of the legs, two centimeters above the lateral malleoli. To determine the maximum load, the weight established during the familiarization session served as the starting point. Resistance was increased progressively in 5 kg increments until the volunteer could no longer complete a subsequent attempt; at that point, 50% of the load added in the final attempt was subtracted. A maximum of five attempts were performed, with three-minute recovery intervals between them; if the 1RM load could not be determined within these five attempts, the individual returned after 48 hours to repeat the test.

Thirty minutes after determining the 1RM load, participants performed submaximal loads of 10%, 20%, 30%, 40%, and 50% of their 1RM; the protocol involved one minute of rest, one minute performing 20 repetitions at 10% of the load established in the 1RM test, and three minutes of recovery. During the second minute—15 seconds before the exercise ended—the earlobe was cleaned with alcohol; using a lancet and disposable gloves, the first drop of blood was discarded, and the second was placed directly onto the lactate-specific test strip of the Accutrend Plus device (Roche, USA). This equipment was chosen for its clinical applicability, offering a cost-effective method to assess lactate levels during routine exercise physiology assessments in clinical and even hospital settings. This same procedure was repeated for subsequent sets, increasing the load by 10% increments up to 50% of the maximum; HRV data were collected throughout the entire test.

Intervention

A resistance training program was developed in accordance with American Heart Association recommendations 16. The training is described in Figure 1.

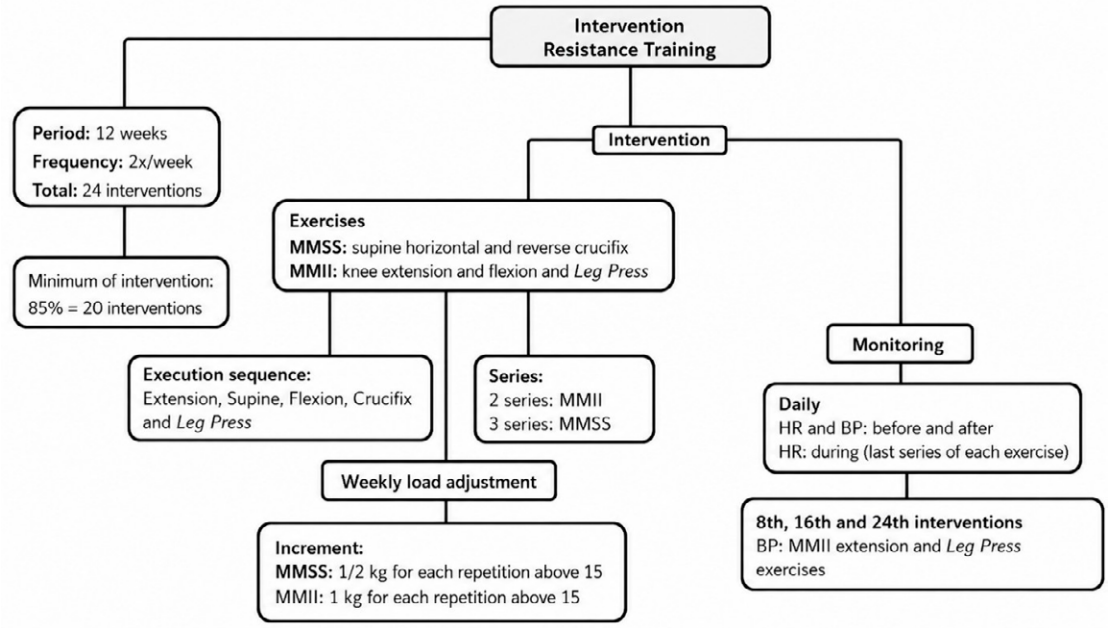

Figure 1: Structure of the intervention protocol. Note: UE: upper limbs; LE: lower limbs; X: times; %: percentage; HR: heart rate; BP: blood pressure; kg: kilograms.

Qualitative variables are described by absolute (f) and relative (%) frequency distributions. Quantitative variables are described by mean and standard deviation (SD) or 95% confidence interval (95% CI). A bootstrapping resampling technique (sample size of 1,000 subjects) was used to determine the 95% confidence interval for the percentile. Paired-samples t-tests were used to compare means between pre- and post-intervention time points within each group. Repeated-measures ANOVA was used to compare means across resting and loaded conditions within each group; the Greenhouse-Geisser correction was applied when Mauchly's test of sphericity was violated. Post-hoc comparisons were performed using the Bonferroni test. A significance level of 5% was adopted, and data were analyzed using SPSS software (version 24.0).

RESULTS

A total of 129 individuals with T2DM and 26 apparently healthy individuals were screened between July 2015 and January 2017. From this sample, eight individuals participated in and completed the training in the T2DM group (GDM2), and six participated in the control group (GC), as shown in the figure below.

The demographic, anthropometric, and resting physiological data of the individuals are presented in Table 1.

Table 1. Demographic, anthropometric, and physiological data at rest prior to training.

Variable	T2DM Group (n = 8)	Control Group (n = 6)
Age (years)	62.50 ± 9.72	58.67 ± 5.28
Body mass (kg)	70.50 ± 13.41	72.42 ± 7.34
Height (m)	1.57 ± 0.06	1.62 ± 0.08
Body mass index (kg/m²)	28.24 ± 5.03	27.86 ± 4.65
Fasting blood glucose (mg/dL)	118.13 ± 24.03	96.83 ± 6.65
Total cholesterol (mg/dL)	181.25 ± 37.36	213.17 ± 20.94
Low-density lipoprotein cholesterol (LDL-C, mg/dL)	91.13 ± 37.51	117.17 ± 38.25
High-density lipoprotein cholesterol (HDL-C, mg/dL)	49.63 ± 17.25	70.40 ± 38.88
Triglycerides (mg/dL)	202.75 ± 85.05	128.00 ± 44.50
Systolic blood pressure (mmHg)	135.88 ± 15.73	118.33 ± 4.27
Diastolic blood pressure (mmHg)	80.00 ± 10.31	84.33 ± 5.57
Resting heart rate (beats/min)	76.38 ± 5.32	71.85 ± 5.42

Note: kg = kilograms; m = meters; kg/m² = kilograms per square meter; cm = centimeters; mg/dL = milligrams per deciliter; TC = total cholesterol; LDL = low-density lipoprotein; HDL = high-density lipoprotein; TG = triglycerides; mmHg = millimeters of mercury; SBP = systolic blood pressure; DBP = diastolic blood pressure; bpm = beats per minute; HR = heart rate.

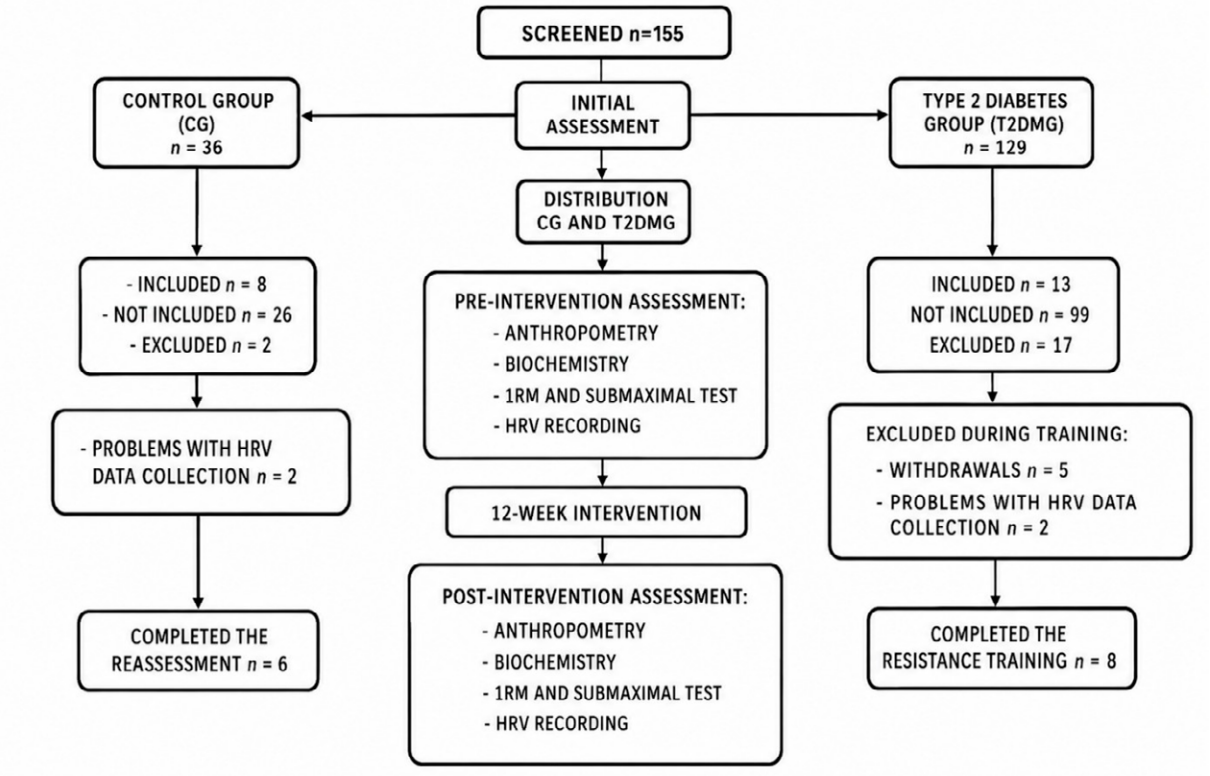

Figure 2: Flowchart of the study development.

Table 2 Clinical Characteristics, Risk Factors, and Current Medication Use of Participants in the T2DM and Control Groups

Variables	T2DM Group (n = 8)	Control Group (n = 6)
Clinical conditions and risk factors		
Dyslipidemia	6 (75.0%)	0 (0%)
Hypertension	7 (87.5%)	0 (0%)
Type 2 diabetes mellitus	8 (100%)	0 (0%)
Obesity (based on BMI)	5 (62.5%)*	0 (0%)
Physical inactivity (sedentary lifestyle)	8 (100%)	6 (100%)
Current medication classes		
Lipid-lowering agents	7 (87.5%)	0 (0%)
Antiplatelet agents	3 (37.5%)	0 (0%)
Beta-blockers	2 (25.0%)	0 (0%)
Diuretics	1 (12.5%)	0 (0%)
Calcium channel blockers	1 (12.5%)	0 (0%)
Angiotensin-converting enzyme (ACE) inhibitors	1 (12.5%)	0 (0%)
Antacids	1 (12.5%)	1 (16.7%)
Hormone replacement therapy	1 (12.5%)	1 (16.7%)

Abbreviations: BMI, Body Mass Index; T2DM, Type 2 Diabetes Mellitus.

DISCUSSION

This study aimed to investigate the effect of resistance training (RT) on lactate levels and parasympathetic indices of heart rate variability (HRV) in individuals with type 2 diabetes compared to control subjects (free from metabolic diseases).

Initial subject characterization revealed homogeneity between groups regarding anthropometric variables such as body mass and Body Mass Index (BMI); the sample fell, on average, within the overweight range, with the T2DM group (GDM2) showing a mean BMI of 28.24 ± 5.05 kg/m² and the control group (GC) showing 27.86 ± 4.56 kg/m². This similarity in anthropometric profiles is important to ensure that the observed physiological changes stem primarily from the pathophysiological condition of Type 2 Diabetes Mellitus (T2DM) rather than from discrepancies in general body composition.

Table 3 RMSSD, SD1, and Blood Lactate Responses During the Exercise Protocol in the T2DM and Control Groups Before and After the Intervention

Variable	T2DM Pre (Mean; 95% CI)	T2DM Post (Mean; 95% CI)	p-value	Control Pre (Mean; 95% CI)	Control Post (Mean; 95% CI)	p-value
RMSSD – Rest	15.30 (10.17–20.93)	16.47 (10.59–24.87)	0.523	16.20 (11.03–24.68)	18.62 (15.87–21.35)	0.649
RMSSD – 10%	11.23 (7.93–14.39)	11.80 (7.22–16.46)		10.60 (5.18–15.80)	11.02 (5.60–17.65)	
RMSSD – 20%	11.04 (7.60–14.84)	12.09 (7.23–16.95)		8.62 (5.15–13.05)	9.55 (4.78–15.02)	
RMSSD – 30%	11.31 (7.02–16.50)	12.09 (7.00–17.56)		7.48 (5.02–9.77)	9.15 (4.87–15.15)	
RMSSD – 40%	10.47 (6.74–14.20)	11.11 (5.87–17.07)		7.12 (4.90–9.25)	9.38 (5.27–14.35)	
RMSSD – 50%	10.49 (8.84–12.33)	10.79 (5.44–16.96)	0.126	6.03 (4.27–7.73)	7.97 (4.02–12.82)	0.515
SD1 – Rest	10.83 (7.21–14.78)	11.69 (7.51–17.63)	0.520	11.48 (7.83–17.07)	13.22 (11.38–15.15)	0.654
SD1 – 10%	7.89 (5.61–10.16)	8.47 (5.19–11.81)		7.67 (3.97–11.37)	7.92 (4.10–12.51)	
SD1 – 20%	7.96 (5.46–10.71)	8.69 (5.19–12.18)		6.22 (3.72–9.28)	6.83 (3.42–10.78)	
SD1 – 30%	8.16 (5.07–11.90)	8.69 (5.04–12.61)		5.47 (3.78–7.08)	6.57 (3.48–10.93)	
SD1 – 40%	7.57 (4.97–10.21)	7.97 (4.17–12.27)		5.20 (3.60–6.68)	6.73 (3.82–10.27)	
SD1 – 50%	7.54 (6.36–8.87)	7.74 (3.90–12.16)	0.119	4.50 (3.20–5.73)	5.68 (2.85–9.15)	0.524
Blood Lactate – Rest	3.00 (2.57–3.43)	3.10 (2.49–3.77)	0.015*	2.38 (1.73–3.20)	2.50 (2.00–2.90)	0.410
Blood Lactate – 10%	2.77[a] (2.41–3.11)	3.08[a] (2.33–3.86)		1.90 (1.67–2.20)	2.88 (2.25–3.75)	
Blood Lactate – 20%	2.71[a] (2.37–3.01)	3.09[a] (2.34–3.84)		2.20 (1.82–2.67)	3.10 (2.40–3.80)	
Blood Lactate – 30%	3.24[b] (2.94–3.47)	3.53 (2.77–4.37)		2.55 (2.08–3.03)	4.00 (3.20–4.80)	
Blood Lactate – 40%	4.21[b] (3.69–4.84)	4.02[b] (3.21–4.94)		3.20 (2.60–3.75)	3.95 (2.65–5.25)	
Blood Lactate – 50%	4.45[b] (3.97–4.97)	4.27 (3.20–5.47)	0.001*	3.82 (3.13–4.50)	4.88 (2.90–6.85)	0.120

Abbreviations: T2DM, Type 2 Diabetes Mellitus; RMSSD, root mean square of successive differences between normal RR intervals; SD1, standard deviation of the instantaneous beat-to-beat variability derived from the Poincaré plot; 95% CI, 95% confidence interval. Data were processed using the bootstrap resampling technique (1,000 samples) with a 95% percentile confidence interval. * indicates a significant difference between loads (including rest) at the same time point (repeated-measures ANOVA, $p < 0.05$). Different letters indicate a significant difference between loads (including rest) at the same time point (Bonferroni post-hoc test, $p < 0.05$). ł indicates a significant difference relative to the pre-intervention time point within each group (Student's t-test, $p < 0.05$). RMSSD: root mean square of successive differences between adjacent normal iRR intervals, expressed in milliseconds; SD1: standard deviation of point distances from the y = x diagonal on the Poincaré plot. Lac: lactate.

Epidemiological studies demonstrate that individuals with T2DM frequently present with excess weight and obesity at the time of diagnosis, highlighting the close association between increased BMI, insulin resistance, and the development of the disease. Paul et al. observed that patients with T2DM had significantly higher BMI values than individuals without diabetes, reinforcing the role of adiposity in the disease's pathophysiology[8].

The T2DM group (GDM2) exhibited elevated fasting blood glucose and triglyceride levels, as well as reduced HDL levels, compared to the control group (GC). This pattern reflects the classic metabolic derangement associated with insulin resistance, characterized by increased hepatic production of triglyceride-rich lipoproteins, reduced HDL-cholesterol, and qualitative changes in LDL particles, culminating in a highly atherogenic profile associated with increased cardiovascular risk[9].

It was also observed that the GC exhibited higher concentrations of Total Cholesterol (213.17 ± 20.9) and LDL (117.17 ± 38.2). Although counterintuitive at first glance, this finding can be explained by the continued use of lipid-lowering pharmacological therapy (such as statins) among individuals with T2DM—a standard practice in clinical guidelines for managing cardiovascular risk in this population (Table 2).

Clinical studies and international guidelines demonstrate that regular statin use substantially reduces plasma LDL-cholesterol concentrations and the risk of major cardiovascular events in individuals with diabetes; consequently, this therapeutic class is widely recommended for both primary and secondary prevention in this population[10,11].

Regarding cardiac autonomic modulation—assessed via RMSSD and SD1 indices (indicators of short-term parasympathetic activity)—the data revealed similar resting and exercise responses (ranging from 10% to 50%) between the Pre- and Post-intervention conditions, with no statistically significant differences ($p > 0.05$).

Although the T2DM group exhibited a slightly higher resting heart rate (HR) at baseline—suggesting the mild sympathetic predominance commonly reported in the literature—the absence of significant acute or chronic changes in HRV indices indicates that the intervention or the applied submaximal exercise protocol was insufficient to induce statistically detectable autonomic remodeling or fatigue.

In this context, the literature shows that persistent exercise-induced cardiovascular adaptations depend on structured programs performed regularly, with appropriate intensity, volume, and progression [12-16]. The American College of Sports Medicine and the American Diabetes Association emphasize that significant autonomic and cardiometabolic improvements are generally observed after weeks or months of systematic training, particularly when progressive overload and sufficient weekly frequency are employed to promote chronic physiological adaptations[12,13].

Blood lactate (Lac) concentration patterns differed between the groups in the present study. While the GC maintained a stable lactate curve with no statistically significant variations throughout the incremental exercise ($p = 0.410$ at Pre and $p = 0.120$ at Post), the GDM2 group demonstrated marked changes at both time points. At the Pre time point for the DM2 group ($p = 0.015^*$), a stepwise increase in lactate is observed as intensity progresses from rest to 50%, evidenced by the subsequent letters (a and b in Table 3). This phenomenon is even more pronounced at the Post time point ($p = 0.001^*$). The early rise in lactate in individuals with DM2 during submaximal loads reflects probable mitochondrial dysfunction and reduced efficiency in the transition from aerobic to anaerobic lactic metabolism.

The greater statistical significance observed at the Post time point ($p = 0.001^*$) suggests that the intervention performed may have sensitized or modified the dynamics of lactate production and/or removal (clearance) in this group, making the metabolic response to incremental exercise evident and distinct compared to the baseline state.

These results are consistent with studies demonstrating higher lactate concentrations during exercise in individuals with T2DM compared to healthy controls, possibly due to alterations in mitochondrial function, reduced skeletal muscle oxidative capacity, and lower metabolic flexibility. Furthermore, recent evidence indicates that physical training programs can positively modulate lactate metabolism by increasing its clearance and improving metabolic efficiency during exertion[14,15].

CONCLUSION

Based on our results, we conclude that although a 12-week resistance training (RT) intervention failed to induce significant changes in cardiac autonomic modulation, the proposed RT suggests an improvement in

blood lactate oxidative capacity during exercise in patients with T2DM, even though the observed improvements in lactate metabolism did not translate into reductions in resting concentrations.

Ethics Statement and Conflict of Interest Disclosures

Financial support and sponsorship: The authors declare that no specific funding, grant, or other financial support was received from any public, commercial, or not-for-profit funding agency for the conduct of this study.

Ethics Consideration: The authors declare that all the procedures and experiments of this study respect the ethical standards in the Helsinki Declaration of 1975, as revised in 2008(5), as well as the national laws. Written informed consent was provided by the patient participant in this study. This study was approved by the Institutional Research Board and Ethics Committee.

Conflict of interest: No known conflict of interest correlated with this publication.

Availability of data and materials: The data used and/or analyzed throughout this study are available from the corresponding authors upon reasonable request.

Competing interests: The authors declared that they have no competing interests.

The use of generative AI and AI-assisted technologies: The authors did not use in this article generative AI and AI-assisted technologies.

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