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Resistance Exercise Elicits Acute Blood Pressure Reduction in Type 2 Diabetics

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ABSTRACT

Sales MM, Segundo PR, Moreira SR, Santana HAP, Moraes JFVN, Asano RY, Motta DF, Dullius J, Simões HG, Campbell CSG. Resistance Exercise Elicits Acute Blood Pressure Reduction in Type 2 Diabetics. **JEPonline** 2012;15(1):98-109. Different exercise modes have been suggested as non-pharmacological treatment for the prevention and/or control of elevated blood pressure and glycemia. Therefore, with objective of the analyze the occurrence of post-exercise hypotension (PEH) to aerobic (AE) and resistance exercises (RE) in type-2 diabetics, nine type 2 diabetics performed: 1) 20-min of cycling (AE) at lactate threshold intensity; 2) RE at 70% of 1RM; and 3) control session (CONT). Heart rate (HR), blood pressure (BP), blood lactate ([lac]), oxygen consumption (VO_2) and rate of perceived exertion (RPE) were measured at rest, exercise or CONT and post-session recovery. Mean VO_2 ($mL \cdot kg^{-1} \cdot min^{-1)}$ of RE (10.6 ± 1.7) was lower than AE (13.3 ± 1.4) ($P<0.05$). VO_2 peak measured immediately after each RE bout (17.5 ± 3.4) was higher than in AE ($P<0.05$). The RE but not AE elicited PEH ($P<0.05$). The mean arterial pressure decrease after 1 hr of recovery in RE by ~5.3 mmHg, while AE elicited a non-significant decrease of ~1.9 mmHg. Only the 70% of 1RM RE circuit training model promoted PEH in type 2 diabetic individuals, perhaps, due to the higher cardiovascular and metabolic stress when compared to the AE session.

Key Words: Diabetes, Blood Pressure, Post-Exercise Hypotension

INTRODUCTION

Type 2 diabetes (T2D) is statistically associated with the development of cardiovascular diseases, nephropathies, neuropathies, retinopathies, and other dysfunctions (1,2). Individuals with T2D may also present systemic arterial hypertension due to structural changes in the endothelium and smooth muscle of the blood vessels (25). From a non-pharmacological point of view, different types of exercise have been used as an aid for the prevention and/or control of hypertension and glycemia (6,15,24,30). Improvement in glycemic control and the post-exercise hypotension (PEH) have been observed in numerous studies on health and hypertensive subjects (4,8,17,23,26,28,31,32) and aerobic exercise (AE) at ~60% of maximum oxygen uptake (VO_2 max) as treatment for T2D.

However, by comparison, there are relatively few studies have evaluated the physiological effects of resistance exercise (RE) on T2D (1,29) and, in particular, PEH after RE in both the healthy and hypertensive subjects (26,31). Only one study has compared resistance exercise with aerobic exercise in BP control in T2D subjects (21). However, the intensities of exercise (AE and RE) seem to have been relatively very different (i.e., 90% of anaerobic threshold, AT, vs. 70% of 1RM). Also, until present there are no studies comparing the rate of perceived exertion, oxygen consumption and blood lactate ([Lac]) responses to AE and RE for T2D and the subjects PEH response.

Thus, the purpose of the study was to determine the effects of both aerobic cycling at lactate threshold intensity (AE) and resistance exercises performed at 70% of 1RM (RE) on hemodynamic and metabolic responses and perceived exertion in T2D subjects. The occurrence of PEH was also analyzed once it was hypothesized that RE would promote a higher cardiovascular and metabolic stress, resulting in a significant PEH effect in the T2D subjects.

METHODS

Subjects

The ethical committee of the Catholic University of Brasília approved this study. Nine sedentary subjects with T2D (5 men; 4 women; 46.6 ± 13.1 yr with ~5 yr of T2D) participated in the study. The subjects' blood glucose was controlled by nutritional management and/or hypoglycemic medication (1 Sulfonylureas, 3 Metformin, 2 Metformin+Glibenclamide, 3 Glimepiride, 1 Pioglitazone Chloridrate). None of the subjects was using exogenous insulin. Some subjects with T2D were classified as prehypertensive during previous medical screening according to the JNC 7 (5), but were not taking medicines for BP control. All subjects were able to participate in this study after a physician's approval by a prior medical screening that included an ECG during an incremental exercise test. The subjects` characteristics are presented in Table 1.

Table 1. Subject demographics. Data expressed in mean and ± standard deviation (n = 9).

Age (yr)	Weight (kg)	Height (cm)	BMI ($kg \cdot m^{-2}$)	BF (%)	Time T2D (yr)	FBG ($mg \cdot dL^{-1}$)	VO_2 peak ($mL \cdot kg^{-1} \cdot min^{-1}$)
56.4	88.5	163.5	32.6	32.8	5.6	153.0	19.6
± 9.1	± 25.1	± 10.3	± 7.2	± 8.6	± 2.4	± 58.3	± 4.5

BMI = body mass index; BF = body fat; FBG = fasting blood glucose; VO_2 peak = peak oxygen consumption.

Before the subjects engaged in the study, an informed consent was signed after the procedures and benefits of participation were explained. A medical screening was performed before the tests. The exclusion criteria included: coronary disease or any cardiac complications; diabetic feet ulcerations or

neuropathy hypertension (>140/90 mmHg) (5); uncontrolled blood glucose levels or routine fasting blood glucose values above 250 mg?dL^{-1}, and/or orthopedic or other complications that would impair the subjects' ability to complete all procedures in the study.

Procedures

All subjects performed: 1) an incremental test on a cycle ergometer to determine the lactate threshold (LT) and VO_2 max; 2) a resistance exercise familiarization; 3) one repetition maximum strength test (1RM); 4) a 20-min submaximal AE session at lactate threshold intensity (100% LT); 5) a resistance exercise circuit training session (RE) performed at 70% of 1RM; 6) a control session without exercise (CONT) (Figure 1). The AE, RE, and CONT sessions were performed on different days and in a randomized order. Oxygen consumption (VO_2) was measured continuously during the experimental sessions by a portable device (Cortex, Metamax3B). Blood lactate concentrations ([Lac]) were measured using an electro-enzymatic analyzer (2.700 S Yellow Springs Instruments). Blood pressure was measured using a portable automated device (Microlife BP3AC1-1).

The subjects were asked to participate in six experimental sessions during the morning on distinct days at least 48 hr apart. A standardized breakfast was consumed 2 hr before trials 4 (AE), 5 (RE), and 6 (CON). The moderate glycemic index meal (GI = 73.9) consisted of 315.9 kcal as follows: 53 g (67.1%, 212 kcal) of carbohydrate; 4.6 g (5.8%, 18.3 kcal) of protein; and 9.5 g (27.1%, 85.6 kcal) of fat. Subjects were instructed to avoid physical exercise and alcohol consumption during the 24 hr before all experimental sessions.

Incremental Exercise Test in Cycle Ergometer (IT)

During the first session, an incremental exercise test was performed on an electromagnetic cycle ergometer (Lode Excalibur Sport – Netherlands) with 15 watts of initial intensity followed by a 15-watt increment at each 3-min stage until volitional exhaustion. The subjects' heart rate (HR) and BP were monitored at the end of each stage by an electrocardiogram and a sphygmomanometer, respectively. Lactate threshold was identified during the IT (33).

RE Familiarization

Subjects underwent 3 sets of each exercise (leg extension, bench press, leg press, lat pull down, leg flexion, and seated row). In the first set, the subjects performed 20 repetitions with a light load. In the 2nd and 3rd sets, more intense loads were used a specific familiarization to the 1RM test.

One Repetition Maximum Test (1RM)

The 1RM test measured the greatest amount of weight that could be performed in a single repetition (22) for each of the following a specific exercises: knee extension, bench press, leg press, pull down, leg curl, and seated row. The test consisted of a warm up for 5 min on a cycle ergometer at 60% of VO_2 max, muscle stretching, and a warm up of 5 to 10 repetitions on the leg press and bench press, with moderate weight between 40 and 60% of the estimated 1RM with a 2-min interval. The first attempt after load was increased was performed alternating the leg press and the bench press with a 2-min interval from one exercise to the next. The maximum number of attempts was 4 for each exercise with 2 min of rest between each.

Resistance Exercise Session at 70% of 1RM (RE)

The exercises were performed in a circuit training model alternating muscle groups by segment. The intensity was set at 70% of 1RM and consisted of 3 laps on circuit. The circuit was composed by the

same 6 exercises used to assess 1RM. Order was kept as described in the 1RM section with 8 repetitions at each exercise with a 50-sec interval between them. A resting period of 1 min was required between each circuit lap. Each repetition lasted approximately 2 sec. The RE session was completed in ~21 min.

Aerobic Exercise Session at LT Intensity (AE)

Subjects performed 20 min of AE on a cycle ergometer (Lode Excalibur Sport – Netherlands) at a previously determined intensity that corresponded to LT. The LT was visually identified by the inflection point of the [lac] versus the workload curve.

Control Session (CONT)

Although the CONT session followed the same procedures used in the AE and RE sessions, the subjects remained in the seated position without exercising.

Measurements

At the end of the 20-min of the pre-exercise rest period in all the experimental sessions, each subject had 25 μL of capillary blood drawn from the earlobe followed by analysis of expired gases. During the incremental test on the cycle ergometer, blood and expired gases were collected during the last 20 sec of each 3-min incremental stage. Expired ventilation, VO_2 and volume of carbon dioxide produced (VCO_2) were measured (Cortex Metamax3B) before, during, and after all sessions, including the RE as shown in Figure 1. Expired gases were measured during both exercise (AE and RE) and control sessions. For the VO_2 measurement, the mean response during the entire time of the exercise sessions was determined (AE: 20 min of cycling; RE: ~21 min of resistance exercises plus the recovery period between exercises; CONT: 20 min of rest). However, the peak VO_2 reached immediately after the completion of each exercise for the RE session was also determined for comparison to AE.

Blood samples were collected during the RE session at the end of each completed circuit lap, as well as during the AE session at the 10th and 20th-min of cycling. The blood samples were collected in heparinized microcapillary tubes and stored in microtubes (Eppendorfs) containing 50 μL of sodium fluoride (1%) for [Lac] analyses. The [Lac] concentrations were measured in an electrochemical analyzer (2700 S Yellow Springs Instruments). All equipment were calibrated and used in accordance to the manufacturer's recommendation.

During the AE, RE, and CONT sessions, BP (Microlife BP3AC1-1), HR (S810i Polar®, Finland), and rate of perceived exertion (RPE) were determined. All BP measurements were performed according to the procedures of the JNC 7 (5) using the subjects' left arm while they were seated with the feet on the floor and the arm resting comfortably at the level of the heart. The BP cuff encircled no less than 80% of the arm.

Statistical Analyses

After exploratory data analysis, we used the Kolmogorov-Smirnov test to investigate the hypothesis of normality of the data. The assumption of sphericity in data distribution was checked using the Mauchly's test and whenever the test was violated, technical corrections were done applying the Greenhouse-Geisser test. Data are presented as mean ± standard deviation or deltas (absolute variation from rest to post-exercise values). Split Plot ANOVA was used to compare the results between and within sessions. Fisher LSD test was adopted as a *Post hoc* to identify the differences.

The level of significance was set at P = 0.05. All calculations were performed using the Statistical Package for Social Sciences (SPSS) for Windows, version 15.0.

RESULTS

The 1 RM results were 75.0 ± 26.6 kg for leg press, 53.9 ± 19.3 kg for bench press, 101.7 ± 28.1 kg for knee extension, 68.2 ± 22.5 kg for pull-down, 80.3 ± 21.1 kg for leg curl and 62.6 ± 18.9 kg for seated row. The metabolic, hemodynamic, and rate of perceived exertion results at RE (70% of 1-RM), AE (LT intensity) and CONT sessions are presented in Table 2.

Table 2. Mean (± SD) values of blood lactate, oxygen consumption, blood pressure, rate of perceived exertion, caloric expenditure, rate-pressure product and duration of the CONT, RE (70% of 1RM) and AE (100% LT) sessions (n = 9).

	CONT	RE (70% 1RM)	AE (100% LT)
Lac (mM)	2.0 ± 0.6**	7.5 ± 3.0*	4.2 ± 1.5
Mean VO_2 ($mL \cdot kg^{-1} \cdot min^{-1}$)	3.3 ± 0.6**	10.6 ± 1.7*	13.3 ± 1.4
Exercise VO_2 ($mL \cdot kg^{-1} \cdot min^{-1}$)	--------	17.5 ± 3.4*	13.3 ± 1.4
Peak VO_2 ($mL \cdot kg^{-1} \cdot min^{-1}$)	--------	17.7 ± 3.6*	14.8 ± 1.00
Session RER	0.87 ± 0.07	1.09 ± 0.13***	1.00 ± 0.09
Peak RER	1.00 ± 0.14	1.28 ± 0.18***	1.00 ± 0.14
Caloric Expenditure (Kcal)	27.8 ± 7.2**	101.5 ± 29.4	116.4 ± 30.6
RPE	--------	13.6 ± 1.2	13.6 ± 2.0
SBP (mmHg)	122.0 ± 19.5**	145.8 ± 19.0	156.4 ± 13.5
DBP (mmHg)	79.2 ± 8.8	77.3 ± 9.3*	85.6 ± 5.3
HR ($beats \cdot min^{-1}$)	78.9 ± 9.2**	131.4 ±18.1	138.6 ± 20.8
RPP	8456.0 ± 1644.0**	19411.0 ± 4326.0	21664.0 ± 3613.0
Duration (min)	20.0 ± 0.0	21.8 ± 3.8	20.0 ± 0.0

Lac – Mean blood lactate results measured during experimental sessions. Mean VO_2 – Mean of oxygen consumption measured during the entire RE session (including recovery period between sets). Exercise VO_2 – oxygen consumption measurement of the experimental session without considering recovery between exercise and sets in RE. Max VO_2 – Oxygen consumption measured immediately after the execution of exercise. Session RER – Respiratory exchange rate of the whole period of the experimental session. Peak RER – Greatest value reached in the experimental session. RPE – Rate of perceived exertion. SBP - systolic blood pressure, DBP - diastolic blood pressure measured during the experimental sessions. HR – Heart Rate. RPP – Rate pressure product. *P = 0.05 in relation to AE; **P = 0.05 in relation to RE and AE;***P = 0.05 in relation to CONT.

The mean VO_2 results measured during the entire time of intervention (AE: 20 min of cycling; RE: ~21 min of resistance exercises plus the recovery period between exercises; CONT: 20 min of rest) are presented in Table 2. In spite of no differences being found between AE and RE for the total duration of the session, the total amount of real exercise performed during RE was only ~4.8 min. If only considered such period of time for the analyses, the corresponding mean VO_2 result (VO_2 max), reached specifically at the completion of exercise (refer to Table 3), revealed the RE to elicit a higher

oxygen consumption in comparison to AE. For the other measurements performed during exercise (RPE, SBP, DBP, Mean Arterial Pressure - MAP and Rate Pressure Product - RPP) the average values are also presented in Table 2.

Figure 1 (A, B and C) presents the delta of variation (mmHg) of SBP, DBP and MAP at each 15-min of the 120-min of post-exercise recovery period. PEH of SBP was observed until 75 min after the RE (70% of 1RM) session, with a highest lowering BP effect of ~11.7 mmHg (60 min recovery) in comparison to the pre-exercise values. For the CONT and the AE (100% LT) sessions, the larger reductions were observed at the 30th-min after sessions (~5 mmHg of SBP reduction). Significant differences in SBP were also observed at the 60th and 120th-min of the post-exercise recovery period with a more pronounced decrease after the RE session when compared to AE ($P = 0.05$). No PEH of DBP was observed after any session (Figure 1C, $P > 0.05$). However, when the DBP results were compared at corresponding moments of recovery for both sessions, it was possible to verify significant differences between AE (100% LT) and RE (70% 1RM) with a significantly higher reduction of DBP at the 75th and 90th-min of recovery after RE ($P = 0.05$).

The MAP responses are presented in Figure 4. PEH of MAP was observed from the 15th through the 105th-min (except at the 75th-min) of recovery in the RE session ($P = 0.05$). On the other hand, similar results were not observed after AE and CONT sessions ($P > 0.05$). The MAP values were significantly higher during recovery in AE than in RE ($P = 0.05$). When compared to the CONT session, the delta of variation of MAP at RE was also lower at the 30th, 60th, and 90th-min of post-exercise recovery. Additionally, no differences were observed between AE and CONT for SBP, DBP and MAP at corresponding moments of the post-exercise recovery period ($P > 0.05$).

DISCUSSION

The post-exercise blood pressure as well as the metabolic and perceived responses to aerobic and resistance exercise were analyzed in individuals with T2D. The main finding of this study was that RE at 70% 1RM promoted PEH of SBP and MAP while the AE performed at an intensity corresponding to the LT did not (Figure 1A, B, and C). During the exercises, SBP, RPP, HR, and RPE responses did not differ between AE and RE sessions (Table 2). The LT occurred around 63% VO_2 max (Table 3). The choice of the AE session at 100% LT was based on recommendations for non-athletes individuals, usually 50-70% VO_2 max, during 20-60 min (1,29). The protocol adopted for RE (70% of 1RM) was similar to those suggested for glycemic regulation in individuals with T2D (6). The contribution of this study was the comparison of the lowering effects on BP of both RE and AE in T2D individuals. The results showed PEH of SBP and MAP after RE.

Table 3. Mean (±SD) values corresponding to the lactate threshold and time until exhaustion during the incremental test in cycle ergometer (n = 9).

Pmax (W)	HR max (beats·m^{-1})	Duration (min)	VO_2 max (mL·kg^{-1}·min^{-1})	LT (W)	LT (%VO_2 max)	LT (%Pmax)
103.3	166.4	22.1	19.6	63.3	63.0	60.9
±34.7	±20.3	±6.7	±4.5	±24.6	±7.7	±10.3

Pmax = maximum Power; HR max = maximum heart rate; VO_2 max = maximum oxygen consumption; LT = lactate threshold.

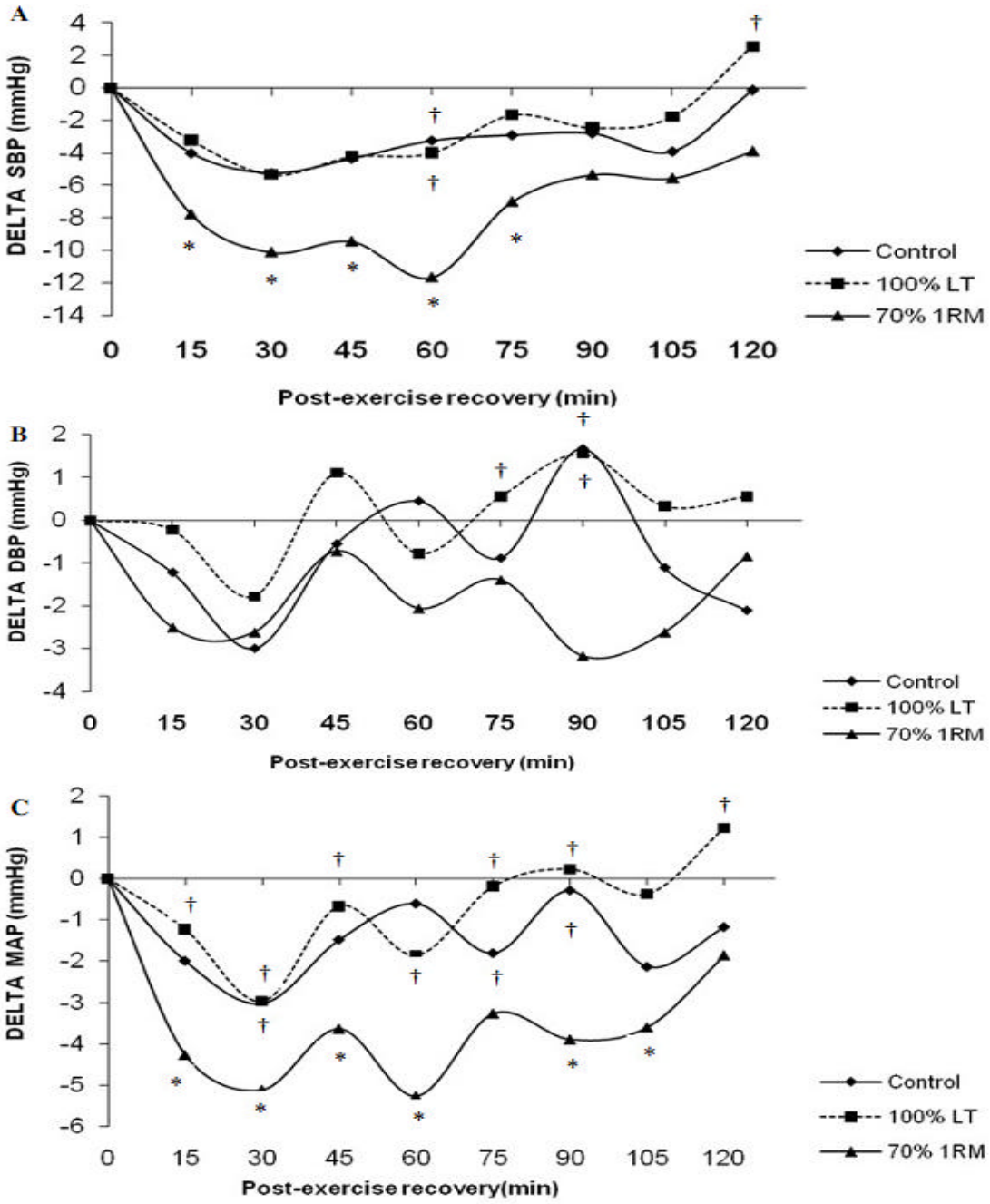

Figure 1 (A, B, and C). Delta of SBP, DBP, and MAP variation during post-exercise recovery in relation to pre-exercise resting (post-exercise recovery - pre-exercise rest). 100%LT = Aerobic exercise performed at lactate threshold intensity; 70% 1RM = Resistance exercise performed at 70% of one repetition maximum; *P = 0.05 in relation to rest; †P = 0.05 in relation to 70% 1RM (RE) session.

The results suggest that RE is an important non-pharmacological control instrument for individuals with T2D, who present augmented risk of developing systemic arterial hypertension due to changes in the secondary structures of endothelium and blood vessels of smooth muscle (25). Dustan et al. (6) has show that that resistance training can improve muscle strength as well as the glycaemic control and prevent sarcopenia in individuals with T2D. Besides that, for presenting a minor quantity of slow-twitch muscle fibers, with consequent greater proportion of glycolytic muscle fibers (10), individuals

with T2D could be more responsive to strength training, enhancing the health benefits from this type of intervention.

Studies indicate that individuals with T2D typically have endothelial dysfunction (19,3). Thus, the hypothesis suggests that this population could does not present PEH due to an endothelium dysfunction. This way, the AE performed at 100%LT was not enough to induce PEH in T2D. While, the RE performed at 70% 1RM induced PEH in SBP and MAP, with significant ($P = 0.05$) changes in various moments post-exercise (refer to Figure 1B and C) when compared to the other experimental sessions (100% LT and CONT). According to demonstrated in Figure 1B, the SBP was reduced in more than 11 mmHg after 70% 1RM, while the AE session (63% VO_2 max) did not promote a significant reduction (~5.3 mmHg). Figueroa et al. (7) found that 20 min of walking in treadmill at 65% VO_2 max in 50 yr-old obese women with T2D induced PEH in 6 mmHg for SBP in 20 min of recovery and they assigned this result to a reduction in the peripheral vascular resistance.

Villa-Caballero et al. (34) after a maximal incremental test in treadmill with sedentary and active T2D individuals found PEH in only active, justified by a greater reactivation of parasympathetic tonus and speculating possibilities of a better endothelial function in consequence of regular physical training. Moraes et al. (20) analyzed, in normotensive and hypertensive individuals, the association of blood pressure responses to the concentrations of bradykinin and plasmatic kallikrein activities after different protocols of exercise with 35 min of duration in a cycle ergometer (70% reserve HR) and in a circuit training RE session (50% 1RM). The results demonstrated that both exercise protocols reduced SBP and MAP in the same magnitude, and suggested that the kallikrein kinin system is involved in PEH in hypertensive as well as in normotensive individuals. This disagreement could be due to a minor duration of exercise in the cycle ergometer used on this study (20 min), once the exercise intensities were similar (69% HR reserve at 100% LT vs. 70% HR reserve).

Mach et al. (18) evaluated the PEH in different exercise durations in a cycle ergometer at 80% of the ventilatory threshold and verified that longer sessions promoted a greater magnitude and larger duration of the hypotensive effect. In our study, the possible lack of statistical differences in PEH after the AE session could be due to its short duration (20 min), since it is known that T2D individuals usually present endothelial dysfunction and this could impair the decrease in BP (35). Ageing may also contribute to damage the endothelium-dependent vasodilatation. Lauer et al. (14) demonstrated that individuals with 58.0 ± 2.0 yr presented a reduced vascular capacity to increase plasmatic nitrite to the AE stimulus. Thus a vascular deterioration could result in a reduced bioavailability of nitric oxide and, in consequence, a lower PEH. In the present study, PEH occurred only after RE when compared to AE. The experimental sessions adopted in the methodology of the present study involved distinct models of exercise. Unlike expected, some parameters during exercise related to hemodynamic, metabolic and perceived effort did not show significant differences when the 70% 1RM and 100% LT sessions were compared (Table 2), suggesting that RE is one important tool for treatment and control of blood pressure in individuals with T2D.

Although mean VO_2 in the 70% of 1RM session presented lower values when compared to the 100% LT (<20%; $P = 0.05$), the pause moments between sets and laps have to be considered. On the other hand, when the exercise VO_2 was calculated only in the moments during the muscular contractions at 70% 1RM, a statistically greater mean in relation to AE in LT was observed (Table 2). Besides that, greater values of blood lactate in the RE session at 70% 1RM (>78%) were verified when compared to the AE session at 100%LT (Table 2). The ventilatory (VO_2), metabolic (Lac) and respiratory exchange rate (RER, Table 2) responses to exercise, demonstrated greater values in RE at 70% 1RM when compared to 100%LT in AE. One of the hypothesis for PEH after RE when compared to AE could be due to a higher intensity performed in RE as well as a greater involvement of muscular

groups, suggesting possibilities of larger peripheral vasodilatation with consequently lower peripheral resistance in the recovery period after exercise (17,16). This would suggest that more intense exercises could optimize the benefits of PEH in individuals with T2D.

CONCLUSIONS

We conclude that RE performed at an intensity of 70% of 1RM promoted PEH in T2D individuals, with similar hemodynamic and perceived stress during exercise when compared to the AE at 100%LT, although with higher metabolic stress.

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