Postresistance Exercise Blood Pressure Reduction Is Influenced by Exercise Intensity in Type-2 Diabetic and Nondiabetic Individuals

GRAZIELA C. SIMÕES,1 SÉRGIO R. MOREIRA,1 MICHAEL R. KUSHNICK,2 HERBERT G. SIMÕES,1 AND CARMEN S.G. CAMPBELL1

1Graduate Program on Physical Education and Health, Catholic University of Brasilia, Brasilia, Brazil; and 2School of Recreation and Sport Sciences, Ohio University, Athens, Ohio

ABSTRACT

Simões, GC, Moreira, SR, Kushinick, MR and Simões, HG, and Campbell CSG. Postresistance exercise blood pressure reduction is influenced by exercise intensity in type-2 diabetic and nondiabetic individuals. J Strength Cond Res 24(5): 1277–1284, 2010—This study analyzed the postexercise blood pressure (BP) after resistance exercise (RE) on middle-aged type-2 diabetic (T2DM, \( n = 10, 46.6 \pm 13.1 \) years) and nondiabetic subjects (NDM, \( n = 10, 52.0 \pm 13.2 \) years). Participants performed (a) 1 repetition maximum (1RM) strength test; (b) 3 laps in an RE circuit of 6 exercises (16 repetitions at 43% 1RM); (c) 3 laps in an RE circuit (30 repetitions at 23% 1RM); and (d) a control session. The blood lactate concentration ([lac]) (YSI 2700S) and BP (Microlife BP3AC1-1) were measured pre-exercise, after exercise, and at each 15 minutes during the 120 minutes of recovery. Analysis of variance with Bonferroni as a post hoc evidenced that the 43% 1RM session elicited the highest [lac] response for both NDM (7.8 ± 1.8 vs. 6.4 ± 1.8 mmol/L; \( p < 0.05 \)) and T2DM (7.0 ± 1.4 vs. 5.6 ± 1.6 mmol/L; \( p < 0.05 \)). Also, the 43% 1RM session promoted a significant postexercise hypotension (PEH) of systolic blood pressure (SBP) and mean arterial pressure (MAP), whereas the 23% 1RM did not. The highest BP reductions for T2DM and NDM after 43% 1RM were, respectively, 9.5 ± 11.1 and 11.0 ± 7.1 mmHg for SBP and 6.4 ± 7.8 and 7.7 ± 7.9 mmHg for the MAP (\( p \leq 0.05 \)). The PEH of SBP lasted longer (120 minutes) for NDM than for T2DM (90 minutes). The PEH may be associated with [lac] elevation, and the lower hypotensive effect presented by T2DM may be related to endothelial dysfunction usually observed in diabetic individuals. In conclusion, the RE of higher intensity, performed in ~25-minute duration, was more efficient at promoting PEH which, in turn, suggests its use on BP control for middle-aged T2DM and NDM subjects with characteristics similar to those of our participants.

KEY WORDS resistance exercise, diabetes, blood pressure control, postexercise hypotension, blood lactate

INTRODUCTION

Modern lifestyle has been associated with an increase in the incidence of noncontagious diseases. The increase in prevalence of type-2 diabetes mellitus (T2DM) in recent years is a global problem (17), and in particular, this metabolic disease may lead to and increase the severity of several secondary clinical complications, including an elevated blood pressure (BP) (2,11).

Exercise has been suggested as a nonpharmacological intervention for elevated BP, in part because many studies have demonstrated a reduced postexercise BP in relation to pre-exercise resting values (3,10,15). This phenomenon is termed postexercise hypotension (PEH) and has been observed after the performance of different exercise modes in both normotensive (4,19) and hypertensive subjects (9,14,23). However, there is a lack in the literature regarding PEH using resistance exercise (RE). Moreover, to our knowledge, no literature exists on postresistance exercise hypotension in individuals diagnosed with T2DM.

Limited evidence exists that suggests PEH is influenced by intensity of RE (15,19,20). On the other hand, it has also been demonstrated that RE, at any intensity, may fail to elicit a PEH response (5). Therefore, the purpose of the present study was to compare the effects of the RE at 23 and 43% of an individual’s 1 repetition maximum (1RM) intensity (23% 1RM and 43% 1RM) on the postexercise BP response in individuals with controlled T2DM and healthy subjects without diabetes mellitus (NDM) who were matched for characteristics.

The hypothesis of the present study was that PEH would be observed after RE and that the intensity at which the RE is performed would influence the magnitude of the PEH for...
both T2DM and NDM. It was also hypothesized that PEH would be more evident for NDM than for T2DM because endothelial dysfunction is usually associated with the pathology.

**METHODS**

**Experimental Approach to the Problem**

To verify if the RE would elicit a PEH in T2DM, and if the intensity of the RE influences the lowering effect on the postexercise BP, 10 T2DM and 10 NDM individuals underwent to 2 RE sessions on different days at 23% 1RM and 43% 1RM. Blood pressure was measured before exercise and during 120 minutes of post-RE recovery period. For both trials (23% 1RM and 43% 1RM), the postexercise BP was compared with the pre-exercise resting values, and to the nonexercise control session. Blood lactate was also measured after each exercise trial to analyze the metabolic stress of intensities studied, and if the lowering BP effect of exercise would be related to the peak blood lactate elicited by the REs for both T2DM and NDM.

**Subjects**

Twenty physically active male subjects with >1-year experience using RE completed the current study. Participants were 10 healthy men without Diabetes Mellitus (NDM; 52.0 ± 13.2 years of age) and 10 subjects with documented T2DM (~4.8 years with the disease) according to previous medical screening (T2DM; 46.6 ± 13.1 years of age). The T2DM participants had their blood glucose controlled by nutritional management and/or hypoglycaemic medication (e.g., Sulfonylureas, Metformin 2, Glucovance 2, Elimepirida 1, Actos 1, and Amaryl 1) but were not using exogenous insulin. Their participation was previously approved by a physician. The main characteristics of the participants are presented in Table 1.

The local ethics committee (Catholic University of Brasilia) approved the methods of the study. An informed consent was signed after the details of the procedures, and its benefits were explained to each participant, but before their involvement in the study. The exclusion criteria included diagnosed coronary disease or any cardiac complications; diabetic foot with ulceration or neuropathy; hypertension, established as values above 149 mmHg for systolic (SBP) and 99 mmHg for diastolic (DBP); uncontrolled blood glucose levels, or levels above 250 mg dL\(^{-1}\); and/or orthopedic or other complications that would impair the participant's ability to complete all procedures in the study.

**Procedures**

All data were collected in the Exercise Physiology Laboratory at the Catholic University of Brasilia. The participants underwent 5 experimental sessions with no less than 48 hours between each at approximately the same time of the day (between 8:00 and 12:00 PM). The first session was an incremental exercise test on a cycle ergometer for cardiovascular evaluation and VO\(_{2}\)max determination. The second session was used to determine the participant's maximal strength (kg) for each given lift. Three additional sessions were used for participants to perform REs in randomized order (sessions 3–5) at intensities corresponding to 23% 1RM and 43% 1RM, and a control session without exercise (CON). The 23% 1RM and 43% 1RM sessions were performed in a circuit. Blood lactate and glucose, heart rate (HR), and BP were measured at after 20 minutes of rest, before resistance exercise (PRE), after each circuit (EX), during the 120 minutes of postexercise recovery (REC), and during the CON at the same points. During PRE and REC, and during the entire session for CON, the participants remained seated in a quiet environment.

Two hours before trials 3–5 (23% 1RM, 43% 1RM, and CON), each participant consumed a standardized breakfast. This moderate glycemic index (GI = 73.9) meal consisted of 315.9 kcal as follows: 53 g (67.1%–122 kcal) of carbohydrate; 4.6 g (5.8%–18.4 kcal) of protein; and 9.5 g (27.1%–85.5 kcal) of fat. Participants were instructed to avoid physical exercise during the last 48 hours before experimental sessions. The medicines that participants were taking (e.g., hypoglycemic drugs) were kept normally during the study.

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**Table 1.** Mean ± SD characteristics of the participants with T2DM (n = 10) and those without Diabetes Mellitus (NDM) (n = 10).

<table>
<thead>
<tr>
<th></th>
<th>T2DM</th>
<th>NDM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>46.6 (13.1)</td>
<td>52.0 (13.2)</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>82.2 (12.7)</td>
<td>76.8 (6.3)</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>174.1 (5.6)</td>
<td>171.4 (4.6)</td>
</tr>
<tr>
<td>BMI (kg m(^{-2}))</td>
<td>21.0 (6.3)</td>
<td>19.5 (2.1)</td>
</tr>
<tr>
<td>VO(_{2})max (ml kg(^{-1}) min(^{-1}))</td>
<td>27.1 (4.2)</td>
<td>26.1 (1.4)</td>
</tr>
<tr>
<td>Fasting* blood glucose (mg dL(^{-1}))</td>
<td>29.8 (6.1)</td>
<td>32.3 (5.5)</td>
</tr>
<tr>
<td>Length of T2DM diagnosis (y)</td>
<td>106.6† (22.7)</td>
<td>71.1 (11.3)</td>
</tr>
</tbody>
</table>

BMI = body mass index; T2DM = type-2 Diabetes Mellitus.

*Fasting blood glucose measured under hypoglycemic medication for T2DM.

†p < 0.05 between groups.
Exercise Protocols

Cardiovascular Evaluation. During the first session, an incremental exercise test was performed on an electromagnetic cycle ergometer (Lode Excalibur Sport, Groningen, The Netherlands) with 15 W of initial workload and 15-W increments at each 3-minute stage until volitional exhaustion. The BP was monitored at the end of each stage with a noninvasive sphygmomanometer. Also, the electrocardiogram was monitored over the exercise test.

One Repetition Maximum Test. During the second session, participants reported to the laboratory after no less than 2 days since they last performed RE. Each participant first performed a 5-minute warm-up on the cycle ergometer using a self-selected moderate intensity and a standardized stretching routine. Before each lift, a submaximal attempt of 5–10 repetitions at workload corresponding to approximately 30–60% of their individual body weight was used. Then, the participants performed each lift using the procedures described by Nieman et al. (16) to determine their 1RM for each of the following 6 REs (Power Tech equipment): knee extension (KE), bench press, leg press (LP) 45°, pulley (PU), knee flexion (KF), and pull down (PD). This order, from lower to upper body, was used to minimize the influence of one exercise on the other in the prevention fatigue.

Resistance Exercise Session at 23% One Repetition Maximum. The 23% 1RM circuit of REs was performed in the following sequence: KE, bench press, LP, PU, KF, and PD. The subjects performed 3 circuits of 30 repetitions (1 complete movement in ~2 seconds) with about 20 seconds of active and passive rest between each exercise in which time the participant moved between each station and then began the next on cue from the investigators. After each complete circuit, a 2-minute passive rest was given for blood sampling and BP measurements. The total length of RE was 3 sets × 6 exercises × 30 repetitions; 2-minute rest after each lap in RE circuit.

Resistance Exercise Session at 43% One Repetition Maximum. The 43% 1RM circuit of REs was performed in a fashion similar to the 23% 1RM session. However, each set for each exercise during a circuit consisted of 16 repetitions at 43% 1RM (1 complete movement in ~2 seconds) with ~40 seconds of active and passive rest between each exercise in which time the participant moved between each station and then began the next on cue from the investigators. After each complete circuit, a passive rest (2 min) was given for blood sampling and BP measurements. The total length of RE (3 sets × 6 exercises × 16 repetitions; 2-minute rest) and the total duration of circuits of different intensities was ~25 minutes, and resulted in the same total work in comparison to 23% 1RM session (a nonsignificant variation <0.3% was observed in the total workload).

Control Session. The volunteers remained seated, without performing any exercise, for the same period of time as the exercise sessions (23% 1RM and 43% 1RM) with the same blood sampling and BP measurements to control for the effects of time (CON).

Blood Sample Collections and Measurements
Each participant had 25 μL of capillary blood being drawn from the earlobe at the end of the 20-minute pre-exercise rest (PRE), at the end of each complete circuit (INT), and every 15 minutes after the completion of the circuits for the 23% 1RM and 43% 1RM and rest for the CON trials during the 120 minutes of recovery (REC). The blood was collected in microcapillary tubes coated with heparin and deposed in microtubes (Eppendorfs) containing 50 μL of sodium fluoride (1%) for measuring blood lactate and glucose. All analyses for each individual’s trial were performed later through an electrochemical analyzer (2700 STAT, Yellow Springs Instruments, Yellow Springs, Ohio, USA). The analyzer was calibrated and maintained according to the manufacturer’s recommendation. Blood glucose was monitored to avoid severe hypoglycemia during exercise. The HR was also monitored by an HR monitor (Polar, Kempele, Finland).

During the exercise, the BP measurements were done noninvasively by sphygmomanometry (Tycos Welch Allyn, New York, USA). However, at each 5 minutes of the 20-minute PRE to take the average and at each 15 minutes of the REC, the BP was measured using a calibrated and automated unit (Microlife BP9AC1-1, Microlife, Heerbrugg, Switzerland). All BP measurements were made according to the procedures of JNC 7 (7) on the participant’s left arm while they were seated with their feet on the ground and arm resting comfortably at the level of the heart. Furthermore, before determination, it was ensured that the BP cuff encircled no less than 80% of their upper arm.

Statistical Analyses
Data are presented as mean ± SD. A repeated measures analysis of variance was applied for comparison within sessions for the results of the postexercise recovery to pre-exercise resting for each group (T2DM and NDM). Comparisons between exercise sessions (23% 1RM; 43% 1RM and CON) were done for the delta of BP (REC value minus the PRE value) and delta% (as the percent change of the REC value as compared with the PRE value) at each 15 minutes of REC within each trial. Significant differences were identified through a Bonferroni test as a post hoc, or a Dunn’s multiple comparisons test was applied if the results were not parametric. Additionally, comparisons between groups (T2DM and NDM) for the delta and delta% of BP during REC from each exercise intensity (23% 1RM and 43% 1RM) were done by the Student t-test. The level of significance was set at p < 0.05, and all statistical analyses were done using the Instat GraphPad software.

Results
The total weight lifted for each participant during 23% 1RM was not different from the 43% 1RM, nor was the time for each circuit within each session different. However, the total
**Table 2.** Mean (± SD) of SBP (mmHg) results for the 23% and 43% 1RM exercise sessions and control (CON) for the participants with T2DM (n = 10) and those without Diabetes Mellitus (NDM) (n = 10).

<table>
<thead>
<tr>
<th>Group and session</th>
<th>PRE</th>
<th>EX</th>
<th>r 15</th>
<th>r 30</th>
<th>r 45</th>
<th>r 60</th>
<th>r 75</th>
<th>r 90</th>
<th>r 105</th>
<th>r 120</th>
</tr>
</thead>
<tbody>
<tr>
<td>T2DM 23%</td>
<td>122.8 (14.0)</td>
<td>133.8 (17.9)</td>
<td>120.3 (17.1)</td>
<td>116.6 (20.8)</td>
<td>120.3 (18.4)</td>
<td>116.3 (17.8)</td>
<td>117.8 (13.6)</td>
<td>118.6 (12.1)</td>
<td>119.5 (11.5)</td>
<td>123.0 (14.4)</td>
</tr>
<tr>
<td>NDM 23%</td>
<td>118.0 (11.1)</td>
<td>136.0* (10.2)</td>
<td>118.4 (9.8)</td>
<td>115.9 (10.4)</td>
<td>111.6 (8.6)</td>
<td>114.3 (7.5)</td>
<td>115.3 (10.7)</td>
<td>115.4 (7.2)</td>
<td>116.8 (7.3)</td>
<td>121.5 (6.2)</td>
</tr>
<tr>
<td>T2DM 43%</td>
<td>122.6 (17.9)</td>
<td>138.8 (18.1)</td>
<td>116.1 (18.8)</td>
<td>113.4* (17.2)</td>
<td>113.9* (20.2)</td>
<td>115.3 (20.6)</td>
<td>115.4 (18.5)</td>
<td>113.1* (17.2)</td>
<td>114.6 (18.8)</td>
<td>117.8 (16.0)</td>
</tr>
<tr>
<td>NDM 43%</td>
<td>120.6 (9.8)</td>
<td>141.0* (18.7)</td>
<td>114.9 (7.9)</td>
<td>112.5* (11.5)</td>
<td>114.4 (10.0)</td>
<td>115.9 (11.2)</td>
<td>109.6* (10.5)</td>
<td>111.9* (7.7)</td>
<td>115.8 (10.8)</td>
<td>111.4* (9.4)</td>
</tr>
<tr>
<td>T2DM CON</td>
<td>121.2 (15.3)</td>
<td>117.6 (15.3)</td>
<td>120.9 (16.4)</td>
<td>121.8 (12.4)</td>
<td>125.6 (18.7)</td>
<td>120.1 (16.3)</td>
<td>121.8 (17.9)</td>
<td>125.8 (17.9)</td>
<td>119.3 (18.3)</td>
<td>123.5 (16.9)</td>
</tr>
<tr>
<td>NDM CON</td>
<td>117.2 (11.1)</td>
<td>115.4 (10.2)</td>
<td>115.5 (9.8)</td>
<td>119.8 (10.4)</td>
<td>117.6 (8.6)</td>
<td>117.0 (7.5)</td>
<td>120.6 (10.7)</td>
<td>117.3 (7.2)</td>
<td>116.0 (7.3)</td>
<td>116.3 (6.2)</td>
</tr>
</tbody>
</table>

PRE = pre-exercise resting; EX = end of exercise circuits; REC15–120 min = recovery each 15 minutes of postexercise recovery period (r15, r30, r45, r60, r75, r90, r105, and r120 minutes); SBP = systolic blood pressure; T2DM = type-2 Diabetes Mellitus; 1RM = one repetition maximum.

*<i>p</i> < 0.05 in relation to PRE.

**Table 3.** Mean (±SD) of DBP (mmHg) results for the 23% and 43% 1RM exercise sessions and control (CON) for the participants with T2DM (n = 10) and those without Diabetes Mellitus (NDM) (n = 10).

<table>
<thead>
<tr>
<th>Group and session</th>
<th>PRE</th>
<th>EX</th>
<th>r 15</th>
<th>r 30</th>
<th>r 45</th>
<th>r 60</th>
<th>r 75</th>
<th>r 90</th>
<th>r 105</th>
<th>r 120</th>
</tr>
</thead>
<tbody>
<tr>
<td>T2DM 23%</td>
<td>75.5 (5.8)</td>
<td>81.7 (8.2)</td>
<td>80.3 (14.2)</td>
<td>72.4 (7.0)</td>
<td>75.5 (9.0)</td>
<td>75.4 (7.1)</td>
<td>72.6 (6.9)</td>
<td>75.3 (5.3)</td>
<td>76.0 (6.3)</td>
<td>76.1 (7.5)</td>
</tr>
<tr>
<td>NDM 23%</td>
<td>76.3 (13.0)</td>
<td>78.8 (18.7)</td>
<td>75.9 (13.5)</td>
<td>76.1 (16.0)</td>
<td>74.8 (14.2)</td>
<td>76.6 (15.4)</td>
<td>77.3 (11.5)</td>
<td>75.4 (12.2)</td>
<td>77.5 (11.2)</td>
<td>80.8 (9.7)</td>
</tr>
<tr>
<td>T2DM 43%</td>
<td>76.5 (7.3)</td>
<td>83.6 (11.3)</td>
<td>74.0 (6.0)</td>
<td>71.4 (5.7)</td>
<td>71.1 (6.7)</td>
<td>71.5 (7.4)</td>
<td>71.4 (7.6)</td>
<td>71.9 (5.5)</td>
<td>74.5 (8.2)</td>
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<tr>
<td>NDM 43%</td>
<td>79.0 (15.8)</td>
<td>76.1 (15.3)</td>
<td>72.6 (13.8)</td>
<td>75.0 (12.7)</td>
<td>77.5 (16.0)</td>
<td>75.6 (14.8)</td>
<td>77.0 (15.4)</td>
<td>76.5 (15.0)</td>
<td>80.3 (13.5)</td>
<td>80.5 (15.1)</td>
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<tr>
<td>T2DM CON</td>
<td>73.1 (7.7)</td>
<td>74.1 (7.8)</td>
<td>75.3 (7.4)</td>
<td>78.8 (8.1)</td>
<td>74.9 (8.0)</td>
<td>77.0 (7.3)</td>
<td>76.5 (5.9)</td>
<td>76.8 (5.5)</td>
<td>78.6 (6.0)</td>
<td>78.5 (4.9)</td>
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<tr>
<td>NDM CON</td>
<td>74.9 (15.3)</td>
<td>76.5 (15.3)</td>
<td>79.9 (16.4)</td>
<td>80.4 (12.4)</td>
<td>80.0 (18.7)</td>
<td>81.0 (16.3)</td>
<td>77.8 (17.9)</td>
<td>81.8 (17.9)</td>
<td>80.6 (18.3)</td>
<td>74.9 (16.9)</td>
</tr>
</tbody>
</table>

PRE = pre-exercise resting; EX = end of exercise circuits; REC15–120 min = recovery each 15 minutes of postexercise recovery period (r15, r30, r45, r60, r75, r90, r105, and r120 minutes); DBP = diastolic blood pressure; T2DM = type-2 Diabetes Mellitus; 1RM = one repetition maximum.

*<i>p</i> < 0.05 in relation to PRE.
amount lifted for each of these trials was greater than the CON trial, whereas the time for the CON trial was similar to the other trials.

The mean blood lactate values obtained at the end of 23% 1RM (6.4 ± 1.8 mM), 43% 1RM (7.8 ± 1.8 mM), and CON (1.4 ± 0.7 mM) sessions for NDM were not significantly different from those observed at 23% 1RM (5.6 ± 1.6 mM), 43% 1RM (7.0 ± 1.4 mM), and CON (1.4 ± 0.5 mM) for the T2DM participants (p > 0.05). However, the mean blood lactate elicited by the 43% 1RM session was significantly higher than that of the 23% 1RM and CON for both groups (p < 0.05), which supports the notion of a higher metabolic stress.

The SBP and DBP results are presented in Tables 2 and 3, respectively, for the T2DM and NDM participants. The SBP, DBP, and mean blood pressure (MBP) during the REC from the 23% 1RM, 43% 1RM, and CON are presented in Figure 1 as delta (difference between REC value and resting value) for each T2DM and NDM participant.

Postexercise hypotension of SBP was observed for T2DM and NDM during REC of the 43% 1RM as compared with the REST value for this trial. In fact, the PEH of SBP within the 43% 1RM trial occurred not only at REC 30 and 90 minutes for both groups, but also 45 minutes in the T2DM participants and at 75 and 120 minutes in the NDM participants. Moreover, besides being observed for both groups and
intensities, the lowering effect on postexercise BP (delta variation in relation to resting) in relation to CON was more significant for NDM than for DM-2 and after the higher intensity session (43% IRM).

**DISCUSSION**

The present study compared the effects of 25 minutes of REs, performed in a circuit at intensities of 23% and 43% 1RM, on the postexercise BP responses of T2DM and NDM participants as compared with a pre-exercise (PRE) BP and a control trial. The characteristics and intensities of the exercise sessions of the present investigation followed previous recommendations for T2DM and hypertensive patients (1), and the main findings were that RE of moderate intensity and repetitions (43% 1RM) elicited a significant lowering effect on postexercise BP (PEH) in men with controlled T2DM, and in those without.

Although PEH after REs has been reported for normotensive and hypertensive nondiabetic subjects (19,20), the current findings of PEH for participants with T2DM after REs are novel. Moreover, the results indicating a more substantial reduction in BP after a higher intensity exercise have not previously been reported in T2DM participants. In the present study, the greatest mean postexercise reduction in SBP was observed at the 90th minute of recovery with a decrease of 7.4% (~9.5 mmHg) for T2DM, and 9.0% (~11 mmHg) for NDM after the 43% 1RM session (Figures 1A, B and Table 2). On the other hand, no significant within-trial lowering effect in SBP was observed after the 23% 1RM session for both groups. However, compared with the no-exercise trial (CON), the SBP was lower in absolute values by 5% (6.5 mmHg) in relation to pre-exercise resting values in the T2DM participants at minute 60 and for NDM at minute 45 after RE performed at 23% 1RM. This suggests that the low-intensity RE may induce PEH and be clinically important in an individual's cardiovascular health.

Taken together, 25 minutes of RE lowers BP in NDM and in patients with T2DM and therefore may be an important nonpharmacologic adjunct in the maintenance of cardiovascular health. According to the JNC 7 (7) a 10-mmHg reduction in SBP of T2DM is associated with a 15% reduction in the risk of overall death, an 11% reduction in the incident of myocardial infarction, and a 13% reduction of other associated vascular complications such as retinopathy and nephropathy. Therefore, the translation of the effects of RE, such as a moderate bout of a whole-body multiple repetition (e.g., 43% 1RM) session, may promote SBP reduction equivalent to the values related to the benefits described previously. However, although the present study only identified the acute effects (through 120 minutes post-RE), it is yet to be demonstrated if routine RE is as beneficial in the maintenance of BP as aerobic training, especially in participants with T2DM.

Previous studies have shown that REs performed at 40% 1RM can promote PEH (19,20) with a decrease of 6–12 mmHg for the SBP in normotensive nondiabetic participants. The 43% 1RM session of the present investigation induced a physical higher stress and metabolic demand, as evidenced by a greater blood lactate concentration ([lac]) when compared with the 23% 1RM and CON sessions for both the T2DM (7.0 ± 1.4 vs. 5.6 ± 1.6 and 1.4 ± 0.5 mM) and NDM (7.8 ± 1.8 vs. 6.4 ± 1.8 and 1.4 ± 0.7 mM). Crisafulli et al. (8) demonstrated that the higher blood lactate elevation after higher intensity exercise is related to more pronounced responses known to be associated with PEH (i.e., peripheral vascular resistance decrease). Such findings corroborate the greater BP lowering effect of the 43% 1RM session in relation to 23% 1RM for the participants of this present study.

The immediate postexercise DBP did not differ from pre-exercise resting values. However, the decrease of DBP (mmHg) after exercise sessions (in recovery) differed significantly from the control session (Figures 1C, D) with 7.7% diminution for T2DM (~6.3 ± 7.4 mmHg) and 6.6% for NDM (~5.2 ± 5.1 mmHg) after the 43% 1RM session and 2.2% diminution for T2DM (~1.3 ± 4.3 mmHg) and 3.9% for NDM (~3.1 ± 6.8 mmHg) after 23% 1RM session, whereas there was an increase of 9.3 and 8.2% after CON for T2DM and NDM, respectively. Despite a failure to elicit any significant reductions in DBP after exercise in this investigation, the RE (43% 1RM) lowered both SBP and DBP of individuals with and without T2DM. Previous studies identified PEH of DBP in healthy young individuals after exercise sessions at intensities between 30 and 40% 1RM (13,20), whereas at higher RE intensity (80% 1RM), a smaller effect was observed (15). Moreover, the MAP was significantly decreased postexercise over the 43% 1RM (~6.6 and ~8.2%) for both T2DM and NDM (Figures 1E, F). Previous research has documented mean postexercise reductions of 3% or more in MAP in relation to pre-exercise resting values (18). When considering that postexercise reductions of ~2 to 4% in MAP have been attributed to a lower risk of stroke (8–14% lower risk) and death by coronary heart disease (5–9% lower risk) (18), the current findings of a reduction of 3.4 and 6.2% after the 23% 1RM session for T2DM and NDM, respectively, although nonsignificant, may be physiologically and clinically significant.

The physiological mechanisms involved in PEH need to be better understood. However, it is likely that part of this is attributed to a decreased peripheral vascular resistance or activity of the sympathetic nervous system (7,12,14), in addition to the diminution of blood flow to areas of the brain involved in cardiovascular regulation such as the insular cortex as evidenced by Williamson et al. (24). Although there may be differences in the mechanisms responsible for PEH in T2DM patients as compared with individuals without T2DM, the present study demonstrated that PEH is a phenomenon in this population that may prove to have significant clinical applications.
The postexercise reductions on SBP, DBP, and MAP were higher after 43% 1RM session, confirming previous findings that higher exercise intensity may promote a more significant postexercise BP reduction (19,20). Moreover, despite both T2DM and NDM participants having demonstrated PEH, the T2DM participants in the present study had a somewhat less great magnitude of PEH in terms of absolute change and duration (Table 2). One explanation may be related to the vascular release of nitric oxide (NO), which has been noted to be lower in T2DM patients during and postexercise (22). A blunted NO release may reduce the T2DM participants’ peripheral vasodilatation during and postexercise as compared with that of NDM participants in the present study. Moreover, regular participation in physical exercise exerts a protective effect on endothelial function (14,21) and contributes to preserve the capacity to release and activate vasodilatation substances such as peptides of the kallikrein–kinin system that may have a role on postexercise BP reduction (6). Therefore, a smaller postexercise BP reduction for T2DM of the present study may have been the result of variations in physical fitness and routine physical activity that were not controlled for.

The main contributions of the present investigation include the demonstration that RE of a moderate intensity may elicit a PEH in male participants with and without T2DM, and that the exercise intensity may have a role in the magnitude of this lowering effect. Future investigations should help to demonstrate the chronic effect of RE performed at various intensities on postexercise BP, especially in patients with T2DM. In conclusion, RE performed at an intensity of 43% 1RM over 25 minutes in a circuit promoted a significant reduction in postexercise SBP that lasted for at least 2 hours for NDM and 90 minutes for T2DM participants. Therefore, in terms of nonpharmacological management of BP, the current data suggest that acute sessions of RE at moderate intensity may be an alternative to traditional aerobic exercise. Additional studies should address the chronic effects of such exercise modes on postexercise BP responses for individuals with diagnoses of T2DM and hypertension.

**Practical Applications**

The findings of present study revealed the RE to be effective for controlling BP in both T2DM and NDM individuals. Thus a moderate-intensity resistance exercise would be performed in a circuit model to reduce BP of T2DM. The prescription would be done in a circuit model, consisting of ~3 sets of 10 exercises with 16 repetitions each at an intensity of about 40–45% 1RM. The recovery between exercises would be approximately 40 seconds in which the participant should change the exercise, and the muscle groups involved would be alternated (preferable). The selected exercises could be those involving large muscle groups as it was for the present study and should be performed at a frequency of no more than 2–3 times a week, with no less than 48 hours apart to avoid injuries because of insufficient recovery among training sessions.

These recommendations may have practical applications for BP control for individuals with T2DM and nondiabetic individuals with characteristics similar to the participants of the present study. Despite those exercise intensities being considered low to moderate and thus secure in terms of cardiovascular stress, a previous medical screening, including an orthopedic, cardiovascular, and metabolic evaluation, is strongly recommended.

Finally, exercise may be an addictive to the effect of medication for both BP and blood glucose reduction. So, we also recommended a fine tuning of the medical-patient feedback to enable a better adjustment (usually reduction) of the medication doses for controlling BP and the doses of hypoglycemic agents administered to the patients that participate in effective exercise training programs.

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