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## Acute resistance exercise is more effective than aerobic exercise for 24 h blood pressure control in type 2 diabetics

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### Abstract

**Aim.** – The study aimed to analyze blood pressure (BP) responses in individuals with type 2 diabetes (T2D) over a 24 h period following resistance (RES) and aerobic (AER) exercise.

**Methods.** – Ten adults with T2D (age:  $55.8 \pm 7.7$  years; weight:  $79.4 \pm 14.0$  kg; fasting glucose:  $133.0 \pm 36.7$  mg.dL<sup>-1</sup>) underwent: (1) AER: 20 min of cycling at 90% lactate threshold (90% LT); (2) RES: three laps of a circuit of six exercises with eight repetitions at 70% 1-RM and 40 s of recovery; and (3) a control session of no exercise. Heart rate (HR), and systolic (SBP), diastolic (DBP), mean arterial (MAP) and pulse (PP) BP, as well as lactataemia (Lac), VO<sub>2</sub>, respiratory exchange ratio (RER) and rate of perceived exertion (RPE) were measured at rest, during exercise and control (CON) periods, and 60 min after interventions. After each session, BP was also monitored over a 24 h period.

**Results.** – Peak Lac (RES:  $6.4 \pm 1.4$  mM; AER:  $3.8 \pm 1.2$  mM), RER (RES:  $1.1 \pm 0.1$ ; AER:  $0.9 \pm 0.1$ ) and RPE (RES:  $14.0 \pm 1.3$ ; AER:  $11.0 \pm 2.3$ ) were higher following the RES session ( $P < 0.05$ ). Similar VO<sub>2</sub> ( $\sim 70\%$  VO<sub>2peak</sub>) was reached during AER and RES sessions ( $14.0 \pm 3.0$  vs  $14.3 \pm 1.6$  mL.kg.min<sup>-1</sup>;  $P > 0.05$ ). Compared with CON, only RES elicited post-exercise BP reduction that lasted 8 h after exercise. Also, in comparison to pre-exercise rest, the BP dip during sleep was greater following RES ( $P < 0.05$ ).

**Conclusion.** – A single exercise bout decreases BP in T2D patients over a 24 h period, with RES being more effective than AER exercise for BP control.

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**Keywords:** Type 2 diabetes; Physical exercise; Resistance exercise; Aerobic exercise; Ambulatory blood pressure monitoring

### Résumé

L'exercice physique d'endurance est plus efficace que l'exercice aérobie pour le contrôle par la pression artérielle sur 24 heures chez des diabétiques de type 2.

**Objectif.** – Analyser la pression artérielle chez les diabétiques de type 2 (DT2) sur 24 heures après exercice d'endurance et exercice aérobie.

**Méthodes.** – Dix patients atteints de DT2 ( $55,8 \pm 7,7$  ans,  $79,4 \pm 14,0$  kg, glycémie à jeun  $133,0 \pm 36,7$  mg.dL<sup>-1</sup>) ont réalisé : (1) un exercice aérobie de 20 minutes sur bicyclette ergométrique à 90 % du seuil lactate (90 % LT) ; (2) un exercice d'endurance comportant trois tours d'un cycle de six exercices avec huit répétitions à 70 % 1RM et 40 s de récupération ; (3) une session témoin sans exercice. La fréquence cardiaque (HR), la pression artérielle systolique (SBP), diastolique (DBP), moyenne (MAP), la pression pulsée (PP), les lactates plasmatiques, la VO<sub>2</sub>, le quotient respiratoire (RER) et l'intensité de l'effort perçue (RPE) ont été mesurés au repos, pendant les sessions expérimentales et après 60 minutes de récupération. Après chaque session a été réalisée, une surveillance ambulatoire de la pression artérielle sur 24 heures.

**Résultats.** – Le pic des lactates plasmatiques était plus élevé lors de l'exercice d'endurance que lors de l'exercice aérobie ( $6,4 \pm 1,4$  versus  $3,8 \pm 1,2$  mM) de même que le quotient respiratoire ( $1,1 \pm 0,1$  versus  $0,9 \pm 0,1$ ) et l'intensité de l'effort perçue ( $14,0 \pm 1,3$  versus  $11,0 \pm 2,3$ ) ( $P < 0,05$ ). Une VO<sub>2</sub> similaire ( $\sim 70\%$  VO<sub>2</sub> max) a été atteinte au cours de l'exercice aérobie et des séances d'exercice d'endurance ( $14,0 \pm 3,0$  versus  $14,3 \pm 1,6$  mL.kg.min<sup>-1</sup>) ( $P > 0,05$ ). Comparé à la période témoin, l'exercice en endurance seul s'est accompagné d'une réduction de la

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pression artérielle, qui s'est prolongée huit heures après l'exercice. En outre, par comparaison avec la période de repos avant exercice, la baisse de pression artérielle durant le sommeil a été plus nette après l'exercice d'endurance ( $P < 0,05$ ).

**Conclusion.** – L'exercice physique entraîne une baisse de la pression artérielle chez les patients atteints de DT2, l'exercice en endurance étant plus efficace que l'exercice aérobie.

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**Mots-clés :** Diabète de type 2 ; Exercice physique ; Exercice en endurance ; Exercice aérobie ; Pression artérielle ; Surveillance ambulatoire de la pression artérielle

## 1. Introduction

The incidence of type 2 diabetes (T2D) and systemic arterial hypertension (SAH) has increased greatly worldwide due to sedentary lifestyles and obesogenic dietary habits [1]. T2D represents 90–95% of all cases of diabetes, characterized by decreased insulin sensitivity and poor glycaemic control [2]. Furthermore, T2D presents with associated complications such as endothelial dysfunction [3,4], increased sympathetic tonus and other cardiovascular disorders, including SAH [5], that lead to an increase in morbidity and mortality in diabetic patients [6].

Ambulatory blood pressure monitoring (ABPM) has been used for blood pressure (BP) response analysis during both waking and sleeping hours, contributing to the prognosis of possible cardiovascular events and damage to target organs [7,8]. Elevated daytime BP values, associated with greater nocturnal dips are factors that, when associated with hyperglycaemia, increase cardiovascular dysfunction [9–11].

Physical exercise, a healthy diet and proper medication are important tools in the treatment of T2D and its complications [12,13]. Physical exercise reduces body weight, lipid profile, BP and endothelial dysfunction, and increases insulin sensitivity, glycaemic control and cardiovascular fitness [14,15]. Usually, aerobic exercise programmes are recommended for patients with T2D [16–18]. On the other hand, resistance training has also been recommended [19,20] for promoting increased muscle mass, insulin sensitivity and glucose transporter type 4 (GLUT-4) translocation [17].

It is known that responses to physical training are the result of cumulative acute effects over time. One acute effect of a single exercise session is post-exercise hypotension (PEH), characterized by a reduction in BP during the postexercise recovery period to values below those observed during pre-exercise rest [21]. The effect of exercise on lowering BP can be observed in both normotensive [22] and hypertensive [23] individuals, and can last from 12 [24] to 24 h [25–27] in the latter. Nevertheless, new studies are now needed to assess the intensity, type and duration of physical exercise necessary to optimize BP control in patients with T2D. With this in mind, the objective of the present study was to investigate BP responses during a 24 h postexercise period in T2D patients who had performed both resistance (RES) and aerobic (AER) exercise.

## 2. Patients and methods

### 2.1. Patients

After approval by the local Ethics Committee for Human Research of the Catholic University of Brasilia, 10 volunteers

(three men and seven women) with T2D, aged 45–70 years (Table S1; see supplementary material associated with this article online), gave their written consent to participate in the study. The inclusion criteria were: sedentariness; a T2D diagnosis for at least 1 year; glycaemia controlled by diet and/or hypoglycaemic medication (sulphonylureas, metformin, metformin + glibenclamide, glimepiride, pioglitazone); non-use of exogenous insulin; and no chronic diabetes complications (such as diabetic foot, nephropathy, retinopathy, neuropathies or cardiovascular illnesses).

### 2.2. Protocol

Following clinical evaluation, at-rest electrocardiography (ECG) and a session of familiarization with the experimental procedures, the volunteers were submitted to five experimental sessions on different days: (1) an incremental cycle ergometer test; (2) measurement of one-repetition maximum load (1-RM) on weight machines; (3) a session at 90% of lactate threshold (LT) intensity on a cycle ergometer (AER); (4) a resistance exercise session at 70% 1-RM (RES); and (5) a control session (CON) of no physical exercise. The AER, RES and CON sessions were carried out on alternate days in random order at the same time of day (between 0830–0900 h in the morning) and separated by at least 48 h. In addition, 2 h before every session, the volunteers had a standardized breakfast of 315 kcal that was 66% (55.6 g) carbohydrate, 6% (4.6 g) protein and 27% (9.5 g) fat, and consisting of foods of a moderate (73.9) glycaemic index.

### 2.3. Experimental tests

#### 2.3.1. Incremental test (IT) on cycle ergometer

The IT was carried out on a cycle ergometer (Excalibur Sport; Lode Medical Technology, Groningen, The Netherlands), and started with a 1 min warmup at 0 watts (W), followed by 15-W increments every 3 min until voluntary exhaustion. At rest and during the final 10 s of each stage of the IT, blood samples were collected for blood lactate analysis (Lac) using a YSI 2700 biochemistry analyzer (YSI Inc, Yellow Springs, OH, USA). In addition, BP was measured during IT by the auscultatory method, using a mercury column sphygmomanometer (Tycos Instrumentos Hospitalares, São Paulo, Brazil), heart rate (HR) was verified with a Polar® S810i heart-rate monitor (Polar Electro Oy, Kempele, Finland), perceived exertion (RPE) was rated on the 20-point Borg scale [28] and ventilatory variables were also recorded (MetaLyzer 3B System; Cortex, Leipzig, Germany). The IT allowed iden-

tification of the LT [29] and measurement of the  $VO_{2peak}$  [30].

### 2.3.2. One-repetition maximum test (1-RM)

The one-repetition maximum test (1-RM) [31] was carried out on weightlifting equipment (Righetto; Powertec, São Paulo, Brazil) with six exercises in the following order: leg extensions; bench press; leg press; seated pulley; leg curls; and rowing machine (Table S1; see supplementary material associated with this article online).

## 2.4. Acute exercise and control sessions

### 2.4.1. Aerobic exercise (AER) at 90% LT intensity

The AER session consisted of a 20 min cycle ergometer exercise (Excalibur Sport; Lode Medical Technology) at a constant load, corresponding to 90% of the LT (90% LT). During the exercise (at 10 and 20 min), BP, HR, Lac and RPE were measured. Ventilatory variables were also measured throughout the entire AER session (MetaLyzer 3B System; Cortex).

### 2.4.2. Resistance exercise (RES) at 70% 1-RM intensity

The RES session consisted of three sets of resistance exercise at 70% 1-RM intensity. Each set of RES was performed in the same order and with the same exercises as mentioned above for the 1-RM test (leg extensions, bench press, leg press, seated pulley, leg curls and rowing machine). Eight repetitions were performed (1 s in the concentric phase, and 1 s in the eccentric phase) of each exercise, with a recovery period of 40 s between exercises and 1 min between sets. During the recovery between RES sets, BP, HR, Lac and RPE data were collected. As with the AER, ventilatory variables were measured throughout the RES session (MetaLyzer 3B System, Cortex).

### 2.4.3. Control session (CON)

During the CON session, participants remained at rest in a seated position for 20 min and performed no exercise. However, the same data collection procedures were carried out, as described for the other sessions.

## 2.5. Pre- and postexercise session procedures

Prior to the AER, RES and CON sessions, the participants remained seated at rest for 20 min for BP (Dyna-MAPA<sup>®</sup>, Cardios Sistemas, São Paulo, Brazil) and HR (Polar<sup>®</sup> S810i, Polar Electro Oy) measurements, while capillary blood samples were collected for Lac determination (YSI 2700). In the postsession period, volunteers remained seated and the same variables (BP, HR and Lac) were measured every 15 min up to 60 min. Pulse pressure (PP) was also determined.

### 2.5.1. 24 H ambulatory blood pressure monitoring (ABPM)

After 60 min of recovery, the volunteers were allowed to perform their own personal-hygiene routines to prepare for placement of the ABPM monitor (Dyna-MAPA<sup>®</sup>, Cardios Sistemas), which measured BP over 24 h after every session (AER, RES and CON). Systolic (SBP) and diastolic blood pressure

(DBP), mean arterial pressure (MAP) and PP were determined and analyzed separately, according to periods of pre-exercise rest, waking and sleeping hours, and total 24 h average. Recordings were considered valid for interpretation only when  $\geq 90\%$  of the values were valid. Volunteers were instructed to continue with their usual daily routines and, while BP was being measured, to keep the cuff on their non-dominant arm and in a relaxed position. ABPM was carried out after sessions until 2300 h, with measurements taken every 30 min and, after that time, hourly until 0700 h, as per the IV Guidelines For Ambulatory Blood Pressure Monitoring [8].

## 2.6. Data analysis

The data are presented as means ( $\pm$  SD) and as absolute variations (delta absolute), as indicated by the variance between the pre- and postexercise periods of rest. Two-way analysis of variance (ANOVA) was used for repeated measures, and the Bonferroni post-hoc test (Statistica<sup>®</sup> software, version 5.0) was used to compare values among experimental sessions. The level of significance was set at  $P < 0.05$ .

## 3. Results

The participants' clinical characteristics, and parameters of aerobic and anaerobic fitness obtained during the AER, RES and CON sessions, are presented in Tables S1 and S2 (see supplementary material associated with this article online). Table S3 (see supplementary material associated with this article online) presents the mean values for SBP and DBP, as well as MAP, at rest and during the 24 h ABPM for all experimental sessions. The post-RES exercise SBP, DBP and MAP over 24 h, and during waking hours, were statistically different from the matching values obtained after the CON ( $P < 0.05$ ). Despite no differences in sessions, reductions in SBP, DBP and MAP was observed during sleep after RES whereas, after AER, there was a reduction in MAP only compared with pre-exercise resting values for the same sessions ( $P < 0.05$ ). No nocturnal decrease was observed after the CON session, as values did not differ between pre-exercise rest and waking hours ( $P < 0.05$ ). Also, no differences were observed in PP across all sessions ( $P > 0.05$ ).

Fig. 1 (A–C) presents the absolute variation (delta) in relation to pre-exercise rest of SBP, DBP and MAP during the 24 h post-experimental sessions. SBP reduction was observed at 0–2 h and at 6–8 h post-RES compared with the CON ( $P < 0.05$ ; Fig. 1A). Furthermore, compared with pre-exercise rest, a reduced SBP was observed at 18–20 h after RES ( $P < 0.05$ ). Fig. 1B shows a significant reduction ( $P < 0.05$ ) in DBP at 0–2 h and 6–8 h post-RES exercise compared with the CON. There was also nocturnal dipping of the DBP at 14–16 h only after RES compared with pre-exercise rest ( $P < 0.05$ ). A significant reduction ( $P < 0.05$ ) was also observed in MAP at 0–2 h following RES compared with the CON (Fig. 1C). At 14–16 h and at 18–20 h, there was a drop in MAP post-RES compared with pre-exercise values ( $P < 0.05$ ). Following AER, MAP was also reduced at 16–18 h and 18–20 h compared with pre-exercise values ( $P < 0.05$ ). How-

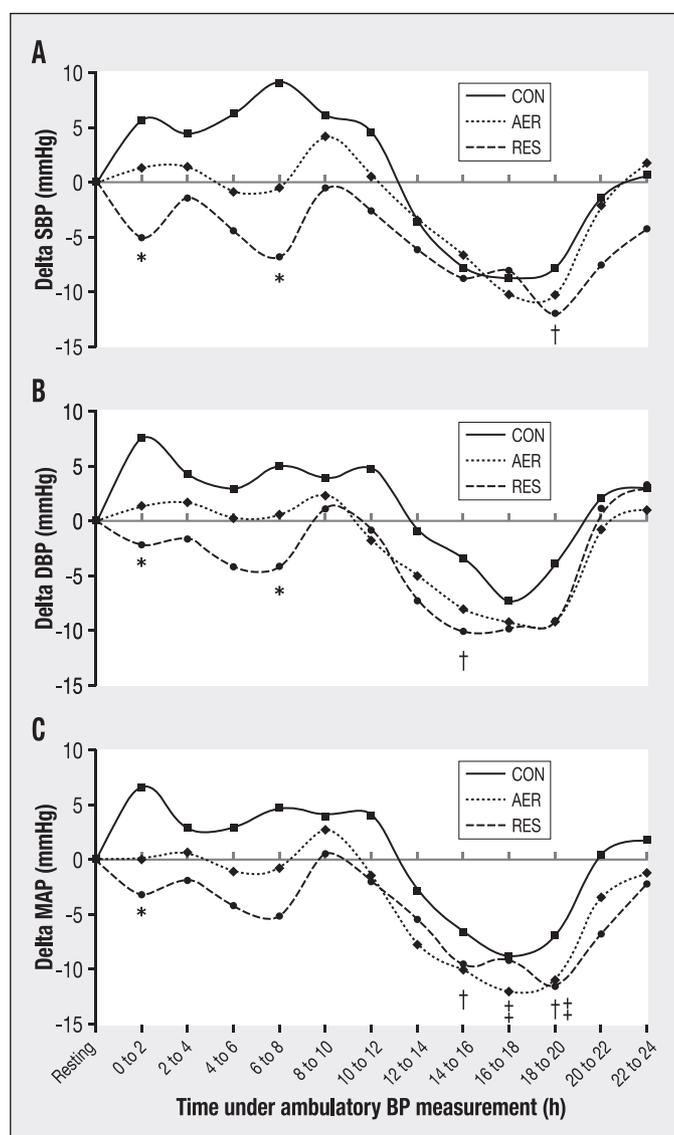


Fig. 1. Delta variations of: (A) systolic blood pressure (SBP), (B) diastolic blood pressure (DBP), and (C) mean arterial pressure (MAP), postexperimental sessions. AER: aerobic exercise at 90% LT; RES: resistance exercise at 70% 1-RM; CON: control session with no exercise. \* $P < 0.05$  vs each corresponding point of the CON session; † $P < 0.05$  vs pre-exercise rest in the same session; †† $P < 0.05$  vs pre-exercise rest in the AER session.

ever, no differences were seen in SBP, DBP or MAP post-AER exercise compared with the RES or CON during the 24 h ABPM ( $P < 0.05$ ).

#### 4. Discussion

The results of the present study demonstrate that, compared with no exercise, RES performed at 70% 1-RM was more effective than AER in promoting protective effects in SBP, DBP and MAP in T2D patients, as measured by 24 h ABPM (Table S3; see supplementary material associated with this article online). In addition, RES brought about significant reductions in BP variables during sleeping hours (Table S3; see supplementary material associated with this article online; Fig. 1). Also

worth noting is that, compared with at-rest values, exercise performed at 90% LT (AER) was effective for reducing MAP during sleeping hours (Table S3; see supplementary material associated with this article online; Fig. 1C). Despite the fact that a nocturnal drop in BP is a normal physiological response in healthy subjects, this phenomenon was not significant in our participants for any BP variable after the CON as, during sleeping hours, BP variables did not differ from either pre-exercise rest or waking values ( $P < 0.05$ ; Table S3; see supplementary material associated with this article online; Fig. 1).

Studies verifying postexercise BP-lowering have been performed in both normotensive [17] and hypertensive [18] individuals, as well as in patients with T2D [4]. Nevertheless, until now, no investigation of BP has been carried out in T2D patients with 24 h ABPM postexercise (aerobic and resistance). For this reason, little is known of the intensity, type and duration of exercise that might promote better BP control in such a population.

In the present study, despite the fact that both sessions (AER and RES) reduced BP, it is suggested that both exercise type and metabolic stress played important roles in the reduction of BP in patients with T2D, with resistance training being the exercise mode that offered more benefits for BP control over the 24 h postexercise period.

Strasser et al. [32] investigated the BP effects of 16 weeks of resistance training, with a workload corresponding to 10–15 maximum repetitions, in patients with T2D. They verified, by ABPM, a reduction in MAP during the day and night following the intervention. Although the present study has only verified the effects of a single exercise session, a link to the study by Strasser et al. [32] is conceivable, as the ‘chronic’ effects of exercise may be considered simply the cumulative effect of several single exercise sessions.

Both hypertensive and normotensive individuals can benefit from the acute effects of exercise on BP. Melo et al. [33], who studied 20 hypertensive women together with an angiotensin-converting enzyme (ACE) inhibitor, reported large BP reductions (SBP:  $-12.0 \pm 3.0$  mmHg; DBP:  $-6.0 \pm 2.0$  mmHg) during the 120 min postexercise recovery period after low-intensity RES ( $3 \times 20 \times 40\%$  1-RM). Such reductions persisted for up to 10 h postexercise (waking hours), but were not seen after a control session with no exercise. Bermudes et al. [34] investigated 25 sedentary normotensive individuals, aged 40–50 years, and found that RES at  $3 \times 25 \times 40\%$  1-RM reduced BP only during sleep.

In the present study, 20 min of aerobic exercise at 90% LT intensity failed to reduce SBP and DBP in T2D patients during the 24 h postexercise period compared with the CON session. Lima et al. [35], on analyzing postexercise hypotension following aerobic exercise at different intensities in volunteers with T2D, observed significant reductions in the 2 h postexercise SBP following 20 min at both 90% LT and 100% LT.

However, these authors also verified that only higher-intensity aerobic exercise was able to reduce DBP and MAP. It has also been shown that higher-intensity resistance training in circuit mode (three laps of 16 repetitions at 43% 1-RM vs three laps of 30 repetitions at 23% 1-RM) can promote greater

reductions of BP during the 2 h postexercise period in individuals with or without T2D [36]. BP control was also seen in the present study during the 24 h after RES, which proved to be more intense than moderate AER, especially on analyzing Lac, RER and RPE (Table S2; see supplementary material associated with this article online).

According to MacDonald et al. [37], the accumulation of metabolites induced by exercise is one of the main factors responsible for muscle vasodilation, with a subsequent drop in peripheral vascular resistance that persists after exercise. In the present study, this effect was probably higher with the RES compared with AER. In contrast, in the Bermudes et al. [34] study, normotensive individuals achieved greater reductions in BP post-AER exercise at an intensity of 60–80% HR<sub>max</sub> than post-RES at 40% 1-RM. The differences in these studies might be due to: (a) the intensity reached during RES; (b) the muscle mass involved; and (c) the clinical characteristics of the population studied.

The present investigation was conducted with diabetic patients who presented with endothelial dysfunction [3] and a reduced capacity to release vasodilatory substances [38], suggesting that the possible mechanism for lowering peripheral vascular resistance could be damaged in this population. On the other hand, after intense RES, as demonstrated in the present study, this limitation does not occur. In addition, unlike moderate AER, the intense RES performed in our study (Table S2; see supplementary material associated with this article online) involved the large-muscle groups, thereby perhaps bringing about greater recruitment of motor units that, consequently, induced greater endothelium-dependent dilatation and a larger reduction in BP during both waking and sleeping hours (Table S3; see supplementary material associated with this article online; Fig. 1), even in our T2D participants, all of whom presented with endothelial dysfunction.

Nevertheless, despite this demonstration of the greater beneficial effects of intense RES on BP for 24 h in patients with T2D, further studies are necessary to clarify the causal mechanisms behind BP-lowering following intense RES in such a population.

One of the limitations of the present study is that variables such as cardiac output, peripheral vascular resistance and autonomic nerve activity were not measured during the experimental sessions. Rezk et al. [39] demonstrated, in healthy adults, that after RES at 40% and 80% 1-RM, the drop in BP was due to a reduction in cardiac output that remained uncompensated for by an increase in peripheral vascular resistance, together with a rise in sympathetic activity and HR.

Another interesting finding of the present study was that the VO<sub>2</sub> and %VO<sub>2 peak</sub> achieved during the RES and AER sessions (Table S2; see supplementary material associated with this article online) suggested that both types of exercise offered similar cardiovascular stress in patients with T2D. If this is so, then it is possible that one session of RES, even at high intensity, may provide an aerobic workout that is similar to a session of moderate AER. RES, therefore, could have induced more neuromuscular and metabolic stress, which then could have led to greater BP-lowering effects. Furthermore, this suggests that, per-

formed on a regular basis (long-term resistance training), RES might promote both cardiovascular and metabolic benefits in addition to neuromuscular benefits, such as gains in strength and hypertrophic effects on skeletal muscles [40].

Considered altogether, the results of the present investigation suggest that acute RES may be used as a non-pharmacological therapy for BP control for patients with T2D. It must be pointed out, however, that high-intensity RES, as applied in our study, is suitable only for T2D patients whose BP is controlled and who have no physical or cardiometabolic complications that might represent an increased risk to their overall functioning (and, even then, only after undergoing clinical examinations to attest to their physical and cardiometabolic fitness).

In conclusion, a single session of resistance exercise was effective in reducing BP throughout the following 24 h in our present study participants, and resistance exercise could represent a useful tool for BP control in patients with T2D.

### Conflict of interest statement

We declare that there were no pertinent conflicts of interest.

### Acknowledgements

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### Appendix A. Supplementary data

Supplementary material (table S1) (table S2) (table S3) associated with this article can be found at <http://www.sciencedirect.com>, at doi:10.1016/j.diabet.2010.08.008.

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