Acute Hypotension after High-Intensity Interval Exercise in Metabolic Syndrome Patients

Introduction

Metabolic syndrome (MSyn) affects about 23% of the US adult population [5] and it is associated with poor blood pressure control in a large sample of European outpatients with hypertension [28]. Hypertension, one of the components of MSyn, increases the risk of developing stroke, coronary artery disease [48], heart failure, atrial fibrillation [4] and peripheral vascular diseases. The remaining MSyn components, increased waist circumference, dyslipidemia and impaired fasting glucose also increase the risk of cardiovascular diseases. Some of these factors interact among themselves to raise the risk of suffering cardiovascular diseases. For instance, abdominal obesity and excess weight raise the prevalence of hypertension [49] and thus the occurrence of adverse cardiovascular events [11]. Conversely, even moderate weight loss by caloric restriction results in substantial reductions in systolic and diastolic blood pressure (11 and 6 mmHg, respectively) in MSyn patients [7].

Aerobic exercise training, a non-pharmacological intervention, reduces resting blood pressure [46] even in people with resistant hypertension [12]. However, aerobic training does not lower blood pressure in the absence of body weight loss in obese patients [43] or MSyn patients [2, 50] despite a significant increase in cardiorespiratory fitness. In corroborating, a recent meta-analysis suggests that continuous moderate-intensity aerobic exercise training without concomitant weight loss does not improve arterial stiffness at least in obese adults [35]. In MSyn patients, aerobic training results in mild reductions in systolic blood pressure that are only noticeable when measured at the carotid artery [13].

One way to assess whether exercise lowers blood pressure in MSyn patients independently of the effects of reduced body weight is to study the blood pressure effect of a single bout of exercise (PEH). In healthy and essential hypertensive subjects, a bout of exercise results in transient (PEH) after a bout of cycling exercise using high-intensity interval training (HIIT) in comparison to an isocaloric bout of traditional moderate-intensity continuous exercise (CE). After supine rest 14 obese (31 ± 1 kg·m⁻²) middle-age (57 ± 2 y) metabolic syndrome patients (50% hypertensive) underwent a bout of HIIT and CE in a random order and then returned to supine recovery for another 45 min.

Exercises were isocaloric and compared to a no-exercise trial (CONT) of continuous supine rest for a total of 160 min. Before and after exercise we assessed blood pressure (BP), heart rate (HR), cardiac output (Q), systemic vascular resistance (SVR), intestinal temperature (Tint), forearm skin blood flow (SBF) and percent dehydration. HIIT produced a larger reduction in systolic blood pressure than CE (−20 ± 6 vs. −5 ± 3 mmHg in the hypertensive group and −8 ± 3 vs. −3 ± 2 mmHg in the normotensive group) and larger increases in resting HR (P < 0.005). Percent dehydration was larger after HIIT, and post-exercise Tint and SBF increased only after HIIT (P < 0.05). Our findings suggest that HIIT is a superior exercise method to CE to transiently reduce blood pressure in MSyn subjects.

ABSTRACT

The purpose of this study was to compare the magnitude of post-exercise hypotension (PEH) after a bout of cycling exercise using high-intensity interval training (HIIT) in comparison to an isocaloric bout of traditional moderate-intensity continuous exercise (CE). After supine rest 14 obese (31 ± 1 kg·m⁻²) middle-age (57 ± 2 y) metabolic syndrome patients (50% hypertensive) underwent a bout of HIIT and CE in a random order and then returned to supine recovery for another 45 min. Exercise trials were isocaloric and compared to a no-exercise trial (CONT) of continuous supine rest for a total of 160 min. Before and after exercise we assessed blood pressure (BP), heart rate (HR), cardiac output (Q), systemic vascular resistance (SVR), intestinal temperature (Tint), forearm skin blood flow (SBF) and percent dehydration. HIIT produced a larger reduction in systolic blood pressure than CE (−20 ± 6 vs. −5 ± 3 mmHg in the hypertensive group and −8 ± 3 vs. −3 ± 2 mmHg in the normotensive group) and larger increases in resting HR (P < 0.005). Percent dehydration was larger after HIIT, and post-exercise Tint and SBF increased only after HIIT (P < 0.05). Our findings suggest that HIIT is a superior exercise method to CE to transiently reduce blood pressure in MSyn subjects.
40 min of moderate-intensity aerobic exercise when compared to carefully matched counterparts [39]. High-intensity interval training (HIIT), a training mode originally used to improve athletic performance, has been quickly embraced in health fitness programs as less time-consuming and tedious than moderate-intensity continuous training while producing similar health benefits [15]. HIIT involves 30 s [33] to 4 min [22] periods of high-intensity exercise that elicit muscle accumulation of metabolites and hyperthermia, all of which have been regarded as mediators of vasodilation (i.e., histamine; [19]). However, some studies maintain that exercise intensity does not affect the magnitude or duration of PEH [31, 40]. The few training studies using HIIT on MSyn patients suggest an effect on reducing resting blood pressure along with a concomitant reduction in body weight [36, 52], which makes it difficult to isolate the influence of exercise from weight loss on the reductions in blood pressure.

In this study, we sought to determine if a bout of cycling exercise using HIIT results in PEH in comparison to an isocaloric bout of traditional moderate-intensity continuous exercise (CE). The hypothesis we maintained is that intense exercise is required to lower blood pressure after exercise in the MSyn population. Identification of the hemodynamic factors that underlie PEH is key to direct strategies that potentiate these factors. We did a full assessment of physiological variables such as post-exercise oxygen consumption, heart rate, cardiac output, systemic vascular resistance, stroke volume, core and skin temperature, and blood flow to identify associations with PEH after each exercise bout.

**Methods**

**Participants**

14 middle-aged (57 ± 8 years) obese subjects (n = 11 males and n = 3 females) with a body mass index of 31 ± 4 kg · m⁻² (> Table 1) and metabolic syndrome (MSyn; [1]) completed this study. Exclusion criteria included the use of medication known to affect heart rate response, (i.e., beta adrenergic receptor blockers or agonists and calcium channel antagonists), untreated thyroid, cardiovascular or renal disease, or any condition associated with exercise intolerance. All subjects provided written, witnessed, informed consent of the protocol approved by the local Ethics Committee in accordance with the World Medical Association Declaration of Helsinki and as required by the journal [21].

**Preliminary testing**

Peak aerobic capacity (VO₂peak) was assessed on an electronically-braked cycle ergometer (Ergoselect 200, Ergoline, Germany) during graded exercise testing (GXT) using indirect calorimetry (Quark b², Cosmed, Italy) with 12-lead ECG monitoring (Quark T12, Cosmed, Italy). The highest heart rate value obtained during the test was considered HRpeak.

**Experimental design**

Using a repeated-measures crossover randomized design, subjects completed 3 trials. One cycling trial consisted of a 10-min warm-up followed by 5 × 4-min intervals at 90% of HRpeak interspersed with 3 min of active recovery at 70% HRpeak (HIIT trial). Another continuous exercise trial was conducted at 60% HRpeak over 70 ± 5 min (CE trial). In a third no-exercise control trial (CONT), subjects rested supine for 70 min. Exercise duration for the HIIT and CE trials was calculated to match trials for energy expenditure (~460 kcals). During exercise, heart rate (HR) and blood pressure were monitored during the initial stages (10–25 min) and during the final stages (38–65 min) of exercise.

**Experimental protocol**

At least 2 h separated the trials and subjects were tested in the morning. 4h before arriving at the laboratory, participants ingested a telemetric thermistor pill (CorTemp™, HQ, Inc., Palmetto, Florida, USA) for the measurement of intestinal temperature (Tint). 2 h before arrival to the laboratory subjects ingested a light breakfast of 500 kcal and 65 g of carbohydrate. Upon arrival to the laboratory subjects voided and nude body weight was recorded (Toledo Hawk, Mettler, Columbus, OH, USA). Urine specific gravity (USG) was measured to confirm euhydration (USG < 1.020; [45]). Then subjects were instrumented with an ECG-gated automated blood pressure sphygmomanometer (Tango™ SunTech Medical, Inc., Morrisville, NC, USA) and a telemetric heart rate monitoring device (Accurex coded, Polar, Finland). Thereafter subjects lay on a stretcher and a laser Doppler flowmeter (Moor Lab, Moor Instruments, Devon, UK) was attached to the dorsal side of the forearm to measure skin blood flow (SBF). Data was expressed as percent of maximal vasodilation elicited at the end of the trial by local heating to 42°C. After 45 min of supine rest, blood pressure, metabolic and cardiovascular measurements were collected before and after exercise. At the end of exercise, subjects toweled dry and nude weight was measured again.

**Metabolic and cardiovascular measurements**

Before and after exercise, O₂ consumption (VO₂) and CO₂ production (VCO₂) were measured for 14 min using indirect calorimetry and energy expenditure was calculated [6]. Blood pressure (BP) was measured in triplicate on the left arm (Tango™ SunTech Medical, Inc., Morrisville, NC, USA) and mean arterial pressure (MAP) calculated. HR was recorded every 5 min before, after and during exer-

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**Table 1** Subject characteristics.

<table>
<thead>
<tr>
<th></th>
<th>Hypertensive (n = 7; 50 %)</th>
<th>Normotensive (n = 7; 50 %)</th>
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<tbody>
<tr>
<td>Age (yrs)</td>
<td>59 ± 6</td>
<td>55 ± 9</td>
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<tr>
<td>Weight (kg)</td>
<td>95.1 ± 15.8</td>
<td>79.9 ± 9.1 *</td>
</tr>
<tr>
<td>BMI (kg · m⁻²)</td>
<td>33.0 ± 4.8</td>
<td>29.1 ± 3.6</td>
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<tr>
<td>Waist circumference (cm)</td>
<td>112.6 ± 10.7</td>
<td>98.0 ± 8.2 *</td>
</tr>
<tr>
<td>Glucose (mg · dl⁻¹)</td>
<td>107.6 ± 14.2</td>
<td>97.1 ± 8.7</td>
</tr>
<tr>
<td>Triglycerides (mg · dl⁻¹)</td>
<td>143.4 ± 61.5</td>
<td>116.9 ± 56.8</td>
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<tr>
<td>HDL-c (mg · dl⁻¹)</td>
<td>49.3 ± 8.8</td>
<td>51.3 ± 10.1</td>
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<tr>
<td>HOMA IR</td>
<td>3.2 ± 1.8</td>
<td>1.8 ± 0.6</td>
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<tr>
<td>MSyn factors (number)</td>
<td>3.6 ± 1.0</td>
<td>3.0 ± 0.6</td>
</tr>
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Data presented as mean ± SD for 14 metabolic syndrome patients. * Significant difference between groups. * P < 0.05 Hypertensive vs. Normotensive
cise. Before and after exercise, cardiac output (Q) was measured in duplicate using the rebreathing technique (Innokor™, Innovation, Odense, Denmark). Stroke volume (SV) was calculated by dividing the Q between HR. Systemic vascular resistance (SVR) was calculated as MAP/SV.

Thermoregulatory measurement
Percent dehydration during exercise was calculated from changes in body weight. None of the participants urinated or ingested fluids during the trials and thus no corrections were needed. T_rit and SKBF were collected continuously before and after exercise and data averaged every 5 min. Cutaneous vascular resistance was calculated as MAP/SKBF and expressed in arbitrary units.

Statistical analysis
Normality was evaluated by the Shapiro-Wilk test. Data collected over time (PRE- and POST-exercise) in the 3 trials was analyzed using 2-way (treatment-by-time) repeated measures ANOVA. After a significant F test, pairwise differences were identified using post hoc Tukey’s HSD. Cohen’s formula for effect size (ES; [9]) was used, and the results were based on the following criteria: >0.70 large effect; 0.30–0.69 moderate effect; ≤0.30 small effect. Minimal detectable change (MDC) was calculated as: MDC = Z-score (90 % CI) × SEM × √2 according to Costa et al. [10]. Data are presented as mean ± SEM. Statistical significance level was set at P ≤ 0.05.

Results
▶ Table 1 shows that when subjects were divided into hypertensive and normotensive groups, they were not different in age, BMI or in the number of MSyn factors. However, the weight and waist circumference were higher in the hypertensive subjects.

Arterial pressure responses
Before exercise (i.e., PRE), systolic blood pressure (SBP) was similar among trials for both the hypertensive and normotensive groups. After HIIT there was a reduction in SBP in the hypertensive group (134 ± 7 to 114 ± 1 mmHg; P = 0.023, ES = 1.14; ▶ Fig. 1a) and in the normotensive group (114 ± 4 to 107 ± 3 mmHg; P = 0.045, ES = 0.95; ▶ Fig. 1c). However, CE and CONT did not affect SBP in either of the groups (▶ Fig. 1a, c). After exercise (POST) there was no difference among trials in SBP in the hypertensive group (▶ Fig. 1a), whereas in the normotensive group POST HIIT was lower than CE and CONT (P = 0.008 and P = 0.002, respectively; ▶ Fig. 1c).

Before exercise (PRE), diastolic blood pressure (DBP) was similar among trials in the hypertensive (▶ Fig. 1b) and normotensive (▶ Fig. 1d) groups. With exercise, DBP was reduced in the hypertensive group only in the HIIT trial (81 ± 3 to 73 ± 1 mmHg; P = 0.01, ES = 0.90; ▶ Fig. 1b). After exercise (POST), DBP in the hypertensive group was lower in HIIT than in the CONT trial (P = 0.03; ▶ Fig. 1b), whereas there was no difference in DBP among trials POST in the normotensive group (▶ Fig. 1d).

Minimal detectable change (MDC) analysis of blood pressure
Systolic blood pressure MDC analysis resulted in 71 % (10 out of 14) of the subjects reaching the systolic MDC (i.e., 6.72 mmHg reduction) following HIIT and 21 % (3 out of 14) following CE. Diastolic blood pressure MDC analysis resulted in 57 % (8 of 14) of the subjects reaching the diastolic MDC (i.e., 3.23 mmHg reduction) following HIIT and 29 % (4 of 14) following CE.

Metabolic and cardiovascular responses
There was no significant difference in cardiac output (Q) among trials PRE or POST. However, HIIT tended to increase Q from 5.7 ± 0.2 to 6.3 ± 0.4 L·min⁻¹ (P = 0.23, ES = 0.34; ▶ Fig. 2a). After exercise (POST), stroke volume (SV) was reduced in the HIIT trial (95.2 ± 6.3 to 70.9 ± 5.2 mL·bt⁻¹; P = 0.001, ES = 1.18) and maintained in the CE and CONT trials. As a result, SV was lower post-exercise in HIIT than in CONT (P = 0.002). From a similar pre-exercise value in all trials, HR increased after exercise in the HIIT and CE trials (62 ± 2 to 89 ± 2 beat·min⁻¹; P = 0.001, ES = 3.00 and 60 ± 3 to 64 ± 3 beat·min⁻¹; P = 0.02, ES = 0.69; respectively) but not in the CONT (▶ Fig. 2b). The increases were larger in the HIIT trial, resulting in higher post-exercise HR than in CE and CONT (P < 0.001).

There was no difference in systemic vascular resistance (SVR) among trials PRE-exercise, however post-exercise HIIT was lower than CE (14 ± 4 vs. 18 ± 6 mmHg·(L·min⁻¹)⁻¹; P = 0.01; ▶ Fig. 3a). Cutaneous vascular resistance (CVR) before exercise (PRE) was increased in the CE trial. However, after exercise (POST), there was a reduction in CVR only in the HIIT trial (422 ± 53 to 101 ± 11 arbitrary units; P = 0.001, ES = 1.91; ▶ Fig. 3b). Although trials were isocaloric, HIIT resulted in larger EPOC than CE (0.211 ± 0.041 vs. 0.095 ± 0.026 mLO₂·min⁻¹; P < 0.05).

Thermoregulatory responses
Percent dehydration measured from body weight losses was larger in the HIIT than in the CE trial (0.85 ± 0.07 vs. 0.55 ± 0.07 %, respectively; P = 0.001; ES = 1.15). SKBF was similar pre-exercise in all trials. SKBF only increased post-exercise in the HIIT trial being, above the rest of the trials (27 ± 4 to 92 ± 7 %, ES = 2.78, P < 0.001; ▶ Fig. 4a). Pre-exercise T INT was similar in all trials. However, post-exercise T INT increased only after HIIT (36.8 ± 0.2 to 37.7 ± 0.2 °C; P = 0.001, ES = 2.56), being larger than in the CE and CONT trials (▶ Fig. 4b).

Discussion
We conducted this study to determine the magnitude of PEH after a single bout of high-intensity interval training (HIIT; [22]) in comparison to a isocaloric bout of traditional moderate-intensity continuous exercise (CE). HIIT has increased its popularity after reports showing training adaptations that promote health in young, healthy subjects [15]. In MSyn subjects, a 14–16 week HIIT program lowers blood pressure [36, 52] and improves endothelial function [52]. Concomitantly, those HIIT programs reduce body weight, which by itself is associated with lowering resting blood pressure [53]. By testing the effects of a single bout of HIIT on blood pressure, we avoided the confounding effects of body weight loss. We found in a sample of MSyn patients that a bout of HIIT induces PEH in normotensive and hypertensive subjects (▶ Fig. 1). In the hypertensive subjects, the magnitude of PEH was similar to that reported in the literature (i.e., SBP = 20 mmHg and DBP = 8 mmHg; [8]; ▶ Fig. 1a, b). In the normotensive subjects, PEH was of a lesser magni-
tude than in the hypertensive subjects, as shown previously in other studies [8, 27]. PEH after HIIT was significantly larger than after CE in diastolic pressure for hypertensive subjects and in systolic pressure for the normotensive subjects. Our findings suggest that HIIT is a superior exercise method to acutely combat hypertension than an isocaloric bout of CE, at least in the obese MSyn population.

We explored the hemodynamics behind the difference in PEH between HIIT and CE by measuring cardiac output (Q; ▶ Fig. 2a) and calculating systemic vascular resistances (SVR). During exercise, Q increases to satisfy the energy demands of the contracting muscle, and SVR declines in muscle and skin. In the return from exercise to rest, SVR remained at a lower level after the HIIT bout (▶ Fig. 3a). Apparently the systemic vasodilation after HIIT lowered central venous pressure, signaling low-pressure baroreceptors to increase Q [20] to prevent further lowering of blood pressure. Interestingly, our middle-aged deconditioned MSyn patients increased Q by increasing heart rate (i.e., chronotropic effect; ▶ Fig. 2b) rather than by increasing cardiac contractility and stroke volume, similar to what has been found in a younger, trained population [47].

An HIIT bout reduced cutaneous vascular resistance in parallel with SVR (▶ Fig. 3b). Thus, the incomplete recovery of resting vasoconstriction tone after the HIIT bout does not seem to be restricted to active skeletal muscle, but affects inactive vascular beds like the skin. It is tempting to conclude that the larger heat accumula-

![Fig. 1](https://example.com/fig1.png)

▶ Fig. 1 a Systolic and b diastolic blood pressure in the hypertensive group and c systolic and d diastolic blood pressure in the normotensive group, before (PRE) and after (POST) a bout of continuous exercise (CE) or high-intensity interval exercise (HIIT) equaled by total energy expenditure (~460kcal) or a control no-exercise trial (CONT). Data are means ± SEM for 14 middle-aged metabolic syndrome subjects.

* Significantly different from PRE-exercise for that trial (P < 0.05). † Difference from CONT at that time point (P < 0.05). ‡ Difference from CE at that time point (P < 0.05).
the HIIT bout. To our knowledge, ours is the first study to integrate EPOC as a possible factor in the PEH response.

It is well documented that PEH occurs upon prolonged moderate-intensity continuous aerobic exercise [51]. However, there is no a clear consensus on the type, intensity and duration of exercise that elicits the larger and more sustained PEH response. Initial reports in hypertensive older humans maintain that the magnitude of PEH is larger the higher the exercise intensity [16]. However, in middle-aged hypertensive [40] or normotensive [31] adults, PEH was not different after mild- or moderate-intensity continuous cycling. Those studies led to early recommendations of light to moderately intense exercise to combat hypertension [41], which continues to be the prevailing recommendation. However, this view has started to change recently upon the appearance of studies showing that vigorous exercise results in larger PEH than moderate aerobic exercise [14]. Furthermore, PEH is maintained longer after more intense exercise [3, 26]. Many studies that find larger PEH with intense exercise use a graded exercise test (GXT) bout [8, 14, 42]. GXT requires participants exercise to the point of volitional fatigue, which results in marked elevations in BP, HR, Q, and muscle metabolite and heat accumulation. Some of these factors were likely also elicited during the high-intensity bouts of the HIIT trial.

One study stands alone in reporting the PEH in MSyn patients [39]. The researchers found that in an MSyn group the PEH response was much attenuated when compared to overweight, middle-aged, hypertensive counterparts with a similarly low cardiovascular fitness level. They reasoned that MSyn-impaired insulin actions for glucose transport (tested by HOMA) may also reflect a blunted insulin-vasodilating effect. Corroborating Pescatello’s data, we re-

![Fig. 2](image-url) a Cardiac output and b heart rate, before (PRE) and after (POST) a bout of continuous exercise (CE) or high-intensity interval exercise (HIIT) equaled by total energy expenditure (~460 kcals) or a control no-exercise trial (CONT). Data are means ± SEM for 14 middle-aged metabolic syndrome subjects. * Significantly different from PRE-exercise for that trial (P<0.05). † Difference from CONT at that time point (P<0.05). ‡ Difference from CE at that time point (P<0.05).

![Fig. 3](image-url) a Systemic vascular resistance and b cutaneous vascular resistance, before (PRE) and after (POST) a bout of continuous exercise (CE) or high-intensity interval exercise (HIIT) equaled by total energy expenditure (~460 kcals) or a control no-exercise trial (CONT). Data are means ± SEM for 14 middle-aged metabolic syndrome subjects. * Significantly different from PRE-exercise for that trial (P<0.05). † Difference from CONT at that time point (P<0.05). ‡ Difference from CE at that time point (P<0.05).
ported that 70 min of moderate-intensity exercise (i.e., CE trial) only reduces diastolic blood pressure in our MSyn participants. However, the blunted PEH response after CE was not found after the isocaloric HIIT bout (> Fig. 1). The factors behind the different PEH response after HIIT vs. CE are not clear. However, as advanced by Pescatello and colleagues, the cardiovascular insulin actions may be involved in PEH. We have recently found that shorter-interval exercise (i.e., 4 Wingate tests) improves insulin sensitivity 30 min after exercise [37] whereas isocaloric continuous moderate-intensity exercise does not. Thus, it is possible that the vasodilating effects of insulin are facilitated by HIIT but not by lower exercise intensity (i.e., CE).

It is not clear if total energy expended during exercise influences the magnitude of PEH [23, 38]. Our bouts of exercise were isocaloric and however PEH was markedly different after HIIT and CE. Thus, our data suggest that something about HIIT caused the more favorable PEH response. HIIT is not only different from CE in exercise intensity but also in the exercise pattern that produces sequential changes in blood pressure and thus shear forces in the vasculature during exercise. It has been proposed that those shear forces are the main stimulus for the production and release of nitric oxide (NO) by the endothelium, which is an strong vasodilator [24]. It has been found that NO plasma concentration is higher during intense exercise in association with higher PEH in hypertensive women [44]. However, NO blockade experiments question how much of the PEH response could be attributed to NO [20].

We performed a minimal detectable change (MDC) analysis of the blood pressure response because it has been reported that PEH may be a heterogeneous physiological phenomenon with responders and non-responders, rendering it impossible to appreciate the potential effects of different exercise methods (i.e., continuous vs. interval [10]) on PEH. However, 10 out of 14 subjects (71 %) responded to HIIT with detectable reductions in systolic blood pressure, whereas only 3 out of 14 (21 %) responded with reductions after CE. Data for diastolic pressure followed the same pattern. These results coincide with our ANOVA analysis, confirming that the effects of exercise mode are stronger than the possible biological variability in the response to different exercise methods. Costa et al. [10] tested young, healthy normotensive males, whereas our participants were middle-aged, hypertensive (50 % of the sample) and suffered from metabolic syndrome (all of them). Undoubtedly, the scope for blood pressure improvement was larger in our sample, which could explain the more homogeneous blood pressure responses, unveiling the statistical differences between exercise methods (i.e., continuous vs. interval).

There are some limitations to our study. We tested PEH 45 min after exercise because it has been reported that the PEH nadir occurs around that time. However, we did not follow the blood response to exercise during the following 12 waking hours, and thus the clinical potential to manage hypertension with HIIT bouts is uncertain. Our results pertain to one bout of exercise and suggest that interval intense exercise is superior to elicit PEH. However, there are some reports stating that high-intensity training over months has less final effect on reducing blood pressure than milder but longer bouts of training [17, 34].

In summary, long-term training treatments in hypertensive people may not be justified without first demonstrating an acute response. Thus, the study of the effect of a HIIT and CE bout on post-exercise hypotension (PEH) is important to deliver efficient exercise advice to hypertensive individuals. Our data suggest that exercise intensity and exercise method (interval vs. continuous) are determinant factors of PEH, whereas total energy expenditure during exercise has a marginal effect on PEH. The underlying mechanisms that trigger this differential PEH response are unclear but could be related to the higher vascular shear forces during exercise, the higher excess post-exercise oxygen consumption, and/or the greater heat accumulated after HIIT in comparison with CE. From a practical point of view, our findings suggest that HIIT is a superior exercise method to acutely combat hypertension than an isocaloric bout of CE, at least in the obese metabolic syndrome population.
Acknowledgements

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Conflict of interest

There is no conflict of interest.

References


