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PHYSICAL ACTIVITY, HEALTH AND EXERCISE

Attenuation of stress-induced cardiovascular reactivity following high-intensity interval exercise in untrained males

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ABSTRACT

Exaggerated cardiovascular (CV) reactivity to stress is associated with negative cardiovascular outcomes. This study aimed to investigate the effects of acute high-intensity interval exercise (HIIE) and moderate-intensity exercise (MIE) on CV reactivity in response to a stress challenge in untrained males. Thirteen, normotensive males (age: 22.8 ± 2 years, BMI: 21.9 ± 3.6 kg/m^2) underwent three conditions in counter-balanced order: HIIE (bodyweight exercises; 80–90% HRR), MIE (treadmill-jog; 55–60% HRR) and seated rest (CON) separated by 7–10 days. Thirty minutes after performing HIIE, MIE or CON, subjects underwent a 2-min cold pressor task (CPT). Blood pressure (BP) and heart rate (HR) were measured before, during, and after CPT. CV reactivity, i.e., the change in BP and HR responses were compared across conditions. Systolic BP reactivity were attenuated following HIIE (−60%, p = 0.015) and MIE (−42%, p = 0.033) compared to CON, but no differences were observed between HIIE and MIE. HR reactivity was not different across all conditions. We conclude that performing HIIE or MIE 30 minutes prior to acute stress exposure lowers BP reactivity compared to rest in untrained males. These findings highlight the potential benefits of HIIE in lowering stress-induced elevations in blood pressure

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KEYWORDS
Blood pressure; autonomic function; cold pressor test; moderate intensity exercise

Introduction

Cardiovascular reactivity refers to responsiveness of the cardiovascular system towards acute physiological or psychological stressors, eliciting changes to cardiovascular functions e.g., heart rate, cardiac output, and blood pressure, allowing the body to adapt to the stressor (Panaite et al., 2015). Stress-induced cardiovascular reactivity is a result of the interplay between activation of the sympathetic system and hypothalamic–pituitary–adrenal axis, along with parasympathetic or vagal withdrawal (Huang et al., 2013). Following cessation of the stress response, the cardiovascular functions are restored to pre-challenge or baseline levels, termed as cardiovascular recovery. This is mostly attributable to cardiac vagal reactivation, which commonly leads to reduction in blood pressure and heart rate responses (Stanley et al., 2013). Frequent exposure to stressful situations in day-to-day life can lead to cardiovascular responses that are exaggerated, prolonged, and repeatedly expressed resulting in heightened blood pressure and slower heart rate recovery.

Increasing evidence indicates that exaggerated cardiovascular reactivity predicts hypertension and future cardiovascular disease more so than resting blood pressure or heart rate (Grassi et al., 2015). It was reported that every 1 mmHg increase in systolic blood pressure reactivity has been associated with a 0.002 to 0.003 mm increase in carotid artery intima-media thickness, resulting in increased vascular resistance accompanied by permanent blood pressure elevation (Roemmich et al., 2009). Everson et al. (2001) reported in their 11-year prospective study that each 1 mmHg rise in systolic blood pressure during a stressful event corresponded to a 1% rise in stroke risk. Similarly, Carroll et al. (2012) observed that every 1-SD rise in stress-induced blood pressure reactivity was associated with a 3% elevation in cardiovascular mortality in a 16-year follow up. These evidence indicate that even small increments in blood pressure can have a significant implication on cardiovascular health in later years.

Every individual is predisposed to experience cardiovascular reactions in response to stress, but the degree of reactivity, i.e., greater or lesser magnitude, can vary from individual to individual, depending on various factors such as age (Estévez-Báez et al., 2019), obesity (Javorka et al., 2016), sleep deprivation (Massar et al., 2017) and exercise training (Huang et al., 2013). Over the past decade, there has been a considerable interest surrounding high-intensity interval training (HIIT). Reports show it can induce favourable cardiovascular responses, i.e., lower resting heart rate and blood pressure, not just in healthy individuals (Martland et al., 2020) but also in populations with overweight/obesity (Batacan et al., 2017), and those with pre- to established hypertension (Costa et al., 2018; Leal et al., 2020). A recent systematic review also supports the many benefits of this training modality on cardiac autonomic control, resulting in increased vagal tone and decreased sympathetic activity, which may be associated with improved blood pressure control (Abreu et al., 2019).

However, the majority of these studies reported changes either in resting blood pressure between pre- and post-

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training, or in 24-ambulatory BP between intervention and control groups, in the absence of a stress stimulus. Although it has been shown that HIIT is effective for improving resting cardiovascular outcomes, many of these studies do not demonstrate how an acute bout of high-intensity interval exercise can influence the way the cardiovascular system responds to stress. Furthermore, cardiovascular reactivity cannot be predicted from resting or baseline cardiovascular levels (Zanstra & Johnston, 2011). While it also appears that cardiovascular reactivity can be attenuated following acute bouts of moderate-intensity continuous exercise (Alderman et al., 2007; Ebbesen et al., 1992; Hamer et al., 2006; Santaella et al., 2006), it remains unclear whether acute bouts of high-intensity exercise may also provide similar or greater outcomes. The lack of existing data comparing stress-induced cardiovascular reactivity following high-intensity and moderate-intensity exercises resulted in limited understanding of how different types of exercise may influence our cardiovascular reactivity in response to stress.

Considering the gaps in the literature, the aim of the present study was to investigate the acute effects of high-intensity interval exercise (HIIE) and moderate-intensity exercise (MIE) on cardiovascular reactivity and recovery in response to a physiological stress challenge in untrained young adult males. Males were chosen for this study as they have been shown to demonstrate higher peak blood pressure and heart rate responses, in addition to slower blood pressure recovery time compared to females (Dimpka & Ugwu, 2009). In our study, we propose to implement an HIIE protocol using bodyweight exercises conducted in circuit style, as opposed to the majority of HIIE protocols that are typically completed on an ergometer or treadmill. A number of recent findings have supported the advantages of whole-body or bodyweight HIIE in producing positive adaptations in physical fitness (Machado et al., 2019; Schaun et al., 2018). We hope to answer the following questions: i) does an acute bout of HIIE elicit an attenuating effect on cardiovascular reactivity and better cardiovascular recovery when compared to no exercise; ii) is the attenuating effect by HIIE superior to that by moderate-intensity exercise. We hypothesised that a single session of HIIE is effective at lowering cardiovascular reactivity in response to a stress challenge, and that the magnitude of the lowering effect is greater than that produced by moderate-intensity exercise.

Methodology

Subjects

A total of 13 untrained, young adult males participated in this experimental study consisting of three conditions. Those who were between 18 and 30 years of age, normotensive, with resting blood pressure above 90/60 mmHg and below the clinical threshold for hypertension stage 1 (>130/80 mmHg) (Whelton et al. 2018), body mass index (BMI) between 18.5 and 27.5 kg/m^2, did not have a history of diagnosed metabolic or cardiovascular disorders, and who were physically inactive (*i.e.*, less than 60 min of exercise per week) for the past 3 months were included in the study. Habitual physical activity was determined using a 5-item physical activity questionnaire that was adapted from Cho (2016). The exclusion criteria were: BMI >27.5 kg/m^2, smokers, use of medications that can affect blood pressure and heart rate, physical limitations that can affect exercise performance, and were suffering from psychological (*e.g.*, depression, anxiety) disorders or traumatic life events (*e.g.*, abuse, death of a loved one). The characteristics of the subjects are presented in Table 1.

Table 1. General characteristics of subjects (n = 13).

Characteristics	Mean ± SD
Age (years)	22.8 ± 2.0
Weight (kg)	61.1 ± 12.2
Height (cm)	166.5 ± 5.7
Body mass index (kg/m^2)	21.9 ± 3.6
Resting systolic BP (mmHg)	115 ± 7
Resting diastolic BP (mmHg)	69 ± 5
Resting heart rate (bpm)	75 ± 6
Minutes of exercise per week	28 ± 10

Experimental protocol

Subjects were required to complete three experimental conditions in counterbalanced order separated by 7–10 days: high-intensity interval exercise (HIIE), moderate intensity exercise (MIE), or rest (CON) (Figure 1). On the three occasions, they reported to the laboratory in the morning after a light breakfast and proceeded to undergo one of the three protocols: HIIE, MIE and control (CON). The HIIE condition consisted of 10 bodyweight exercises, conducted in circuit style and performed in two sets (*i.e.* set 1: push-ups, jumping jacks, lunges, squats, burpees; set 2: bicycle crunches, plank, mountain climber, high-knees, and V sit-ups). Each exercise was performed for 30 seconds interspersed with 20-s rest intervals. The rest interval between the two sets was 2 min. The entire exercise protocol lasted approximately 20 minutes and was adapted based on the recommendations by Machado et al. (2019). Subjects were required to keep the exercise intensity between 80% and 90% of heart rate reserve (HRR). The MIE condition involved slow-

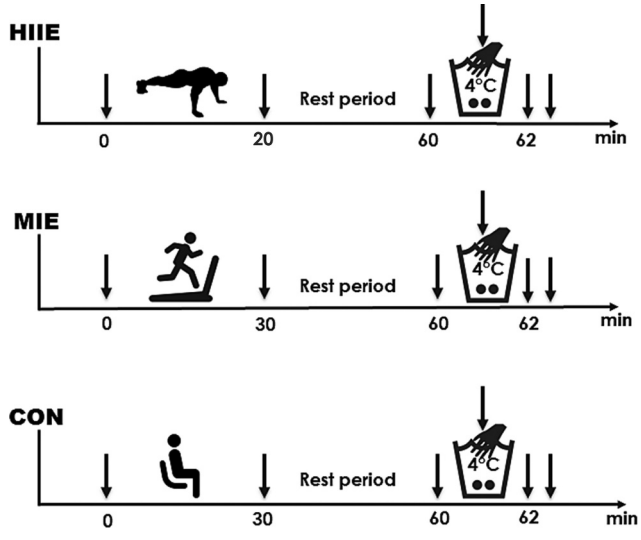

Figure 1. Experimental conditions. HIIE: high-intensity interval exercise; MIE: moderate intensity exercise; CON: control. Arrows indicate measurements of blood pressure and heart rate responses.

jogging on a treadmill at moderate intensity (55–60% of HRR) for 30 minutes. In the CON condition, subjects performed a seated rest for 30 minutes. HR was measured every 5 min during each exercise session and during the corresponding period in the rest session using POLAR® heart rate monitor (Polar RS400, Polar Electro, Oy, Finland). All exercise and rest conditions were followed by a 30-min recovery period, after which subjects underwent the cold pressor task (CPT). The experimental procedures were in line with the Declaration of Helsinki principles and were approved by the local institutional ethics committee (UKM-JEP-2018-239). All subjects gave informed consent prior to participating in the study. Subjects were instructed to refrain from caffeine consumption on the day of the experiment and to abstain from alcohol and strenuous physical activity for 24 h prior.

Cold Pressor Task (CPT)

The CPT is a potent sympathoexcitatory manoeuvre to induce significant increases in blood pressure (Seals, 1990) and is a valid method to evaluate cardiac autonomic function (Wirch et al., 2006). Subjects were instructed to immerse their dominant hand up to the wrist in ice water (0–2°C) for 2 min, which was followed by a one-minute recovery with their hand out of the water. Blood pressure and HR responses were measured before, during and after the CPT.

Cardiovascular reactivity and recovery measurement

Systolic BP (SBP), diastolic BP (DBP), and heart rate (HR) responses were measured at three time points: (i) before the start of CPT i.e., after 30 minutes of seated rest following the exercise/control protocol, (ii) at 1st and 2nd minutes during CPT, and (iii) 2 min after CPT ended (recovery period). All BP measurements were taken in an upright seated position, on the right arm using automated sphygmomanometer (Omron M3 Intellisense, Omron Healthcare, Japan). Mean arterial pressure (MAP) was calculated as one-third of systolic blood pressure plus two-thirds of diastolic blood pressure. Cardiovascular reactivity was determined as the change (Δ) in systolic and diastolic BP, mean arterial pressure (MAP), and HR during the administration of CPT in relation to the pre-CPT (rest) period (Llabre et al., 1991). Cardiovascular recovery was determined as the change (Δ) in systolic and diastolic BP, MAP, and HR during the recovery period in relation to during CPT.

Data analysis

Data were tested for normality of the distribution with the Shapiro–Wilk test and were considered normal. Data were analysed for descriptive and inferential statistics. BP and HR responses during the CPT challenge were calculated as the mean of two values taken at the 1st and 2nd minutes during CPT. A one-way analysis of covariate (ANCOVA) was employed to compare the absolute BP and HR responses during the CPT, controlling for rest period (pre-CPT) across three experimental conditions. Cardiovascular reactivity (BP and HR) was calculated as: mean values during CPT – resting value (pre-CPT). Higher values indicate higher cardiovascular reactivity. To obtain a measure of cardiovascular recovery from stress, BP and HR recovery were calculated as: mean values during CPT – post-CPT value. Greater values indicate greater cardiovascular recovery. Using the change scores as measures for cardiovascular reactivity and recovery, one-way repeated measures ANOVA was conducted to compare the differences between the three conditions, followed by Bonferroni's post hoc test for pairwise comparisons. All statistical analyses were performed using SPSS v.20 (IBM Corp., Armonk, USA). Significance was set at $p \leq 0.05$. Data for statistical reporting are presented as mean ± SEM, unless stated otherwise.

Results

General characteristics of subjects

General characteristics of the study subjects at baseline are shown in Table 1. Thirteen (n = 13) subjects successfully completed all three conditions required by the study. Subjects ranged in age from 21 to 28 years (mean age: 22.8 ± 2.0 years) with a mean BMI of 21.9 ± 3.6 kg/m^2. None of the subjects had BP higher than 130/80 mmHg at the time of screening and before the start of experiment.

Cardiovascular responses during and post exercise

Cardiovascular responses during and post HIIE and MIE exercise are presented in Table 2. Mean exercise heart rates for HIIE condition was 175 ± 3 bpm and 155 ± 2 bpm for MIE, with a significant difference between conditions ($p = 0.01$), indicating that the exercise intensity was indeed different. All subjects completed the exercise sessions without difficulties.

Cardiovascular reactivity and recovery

Using data from all conditions combined, we observed significant increases in systolic and diastolic BP, and HR in response to CPT. The overall mean increase for SBP was 11.4 mmHg, DBP 7.5 mmHg, and HR 9.2 bpm. Table 3 summarises the absolute BP and HR responses at rest, during and post-CPT challenge across experimental conditions. Univariate ANCOVA (controlling for pre-CPT values) indicated that there were significant effects of experimental conditions on SBP ($F(3, 35) = 5.61$, $p = 0.008$, $\eta^2 = 0.24$), DBP ($F(3, 35) = 6.18$, $p = 0.002$, $\eta^2 = 0.23$), and MAP ($F(3, 35) = 9.37$, $p < 0.001$, $\eta^2 = 0.20$) responses during CPT. Post-hoc analyses revealed that performing HIIE resulted in lower SBP ($p = 0.009$), DBP ($p = 0.010$) and MAP ($p = 0.014$) responses during CPT compared to CON. SBP responses during CPT were also lower in MIE ($p = 0.044$) compared to CON. Meanwhile, no significant differences were observed between HIIE and MIE conditions, as well as for HR and post-CPT values across experimental conditions.

Table 2. Cardiovascular responses during and 30-min post exercise (n = 13).

Cardiovascular Variables	HIIE	MIE
Mean exercise HR (bpm)	175 ± 3	155 ± 2
Mean HR post-exercise (bpm)	98 ± 3	90 ± 3
MAP post-exercise (bpm)	83 ± 3	85 ± 3

HIIE: high intensity interval exercise; MIE: moderate intensity interval exercise, MAP: mean arterial pressure; HR: heart rate. Data reported as mean ± SEM.

Table 3. Blood pressure and heart rate responses before, during and after CPT challenge across experimental conditions (n = 13).

Condition	Parameter	Pre-CPT (0 min)	During CPT (1st & 2nd min)	Post-CPT (4th min)
HIIE	SBP	115.9 ± 3.5	122.3 ± 3.1*	119.8 ± 2.9
	DBP	68.0 ± 3.7	69.9 ± 1.7 *	68.6 ± 1.7
	MAP	83.6 ±3.3	87.6 ± 2.0 *	85.7 ± 1.7
	HR	98.3 ± 3.2	103.5 ± 1.4	101.8 ± 2.3
MIE	SBP	114.6 ± 3.5	124.8 ± 3.5 *	123.2 ± 3.2
	DBP	69.1 ± 1.9	77.3 ± 3.1	74.2 ± 2.6
	MAP	85.2 ± 2.6	93.7 ± 3.2	90.5 ± 2.4
	HR	90.1 ± 2.6	96.5 ± 1.8	93.0 ± 2.0
CON	SBP	114.6 ± 2.6	132.3 ± 3.0	129.0 ± 3.1
	DBP	67.8 ± 1.9	80.1 ± 2.6	78.1 ± 2.5
	MAP	83.4 ± 1.9	96.9 ± 2.8	95.1 ± 3.3
	HR	85.7 ± 1.4	94.7 ± 2.5	90.8 ± 2.4

CPT: cold pressor task; SBP: systolic blood pressure; DBP: diastolic blood pressure; MAP: mean arterial blood pressure, HR: heart rate; (*) indicates significant difference (p < 0.05) from CON. Data reported as mean ± SEM.

Figure 2 presents the magnitude of change in BP and HR reactivity and recovery across experimental conditions. There were significant main effects in SBP [F(2, 24) = 3.900, p = 0.001, η^2 = 0.442], DBP [F(2, 24) = 5.114, p = 0.014, η^2 = 0.299] and MAP [F(2, 24) = 6.366, p = 0.006, η^2 = 0.347] reactivity across all conditions. Post-hoc analyses showed that SBP reactivity was ~60% attenuated following HIIE (+6.4 ± 2.7 mmHg) compared to CON (+17.7 ± 2.8 mmHg; p = 0.015). HIIE also resulted in lower DBP (+1.9 ± 2.1 mmHg; p = 0.005) and MAP (+3.9 ± 2.5 mmHg; p = 0.007) reactivity compared to CON (DBP: +12.3 ± 2.2 mmHg; MAP: +13.5 ± 1.9 mmHg). SBP reactivity was also attenuated following MIE (+10.3 ± 2.4 mmHg; p = 0.033) compared to CON, but not different when compared with HIIE. However, findings showed no significant differences in HR reactivity and cardiovascular recovery parameters across all experimental conditions.

Discussion

The present study sought to determine if a prior bout of high-intensity interval exercise (HIIE) or moderate intensity exercise (MIE) can attenuate cardiovascular reactivity during exposure to a physiological stressor (*i.e.*, cold pressor task) in untrained males. A secondary aim was to compare the attenuation effects on cardiovascular reactivity between HIIE and MIE. The main highlight of the study was that BP reactivity during a stress challenge was attenuated following both HIIE and MIE compared to control. In other words, performing exercise, either of

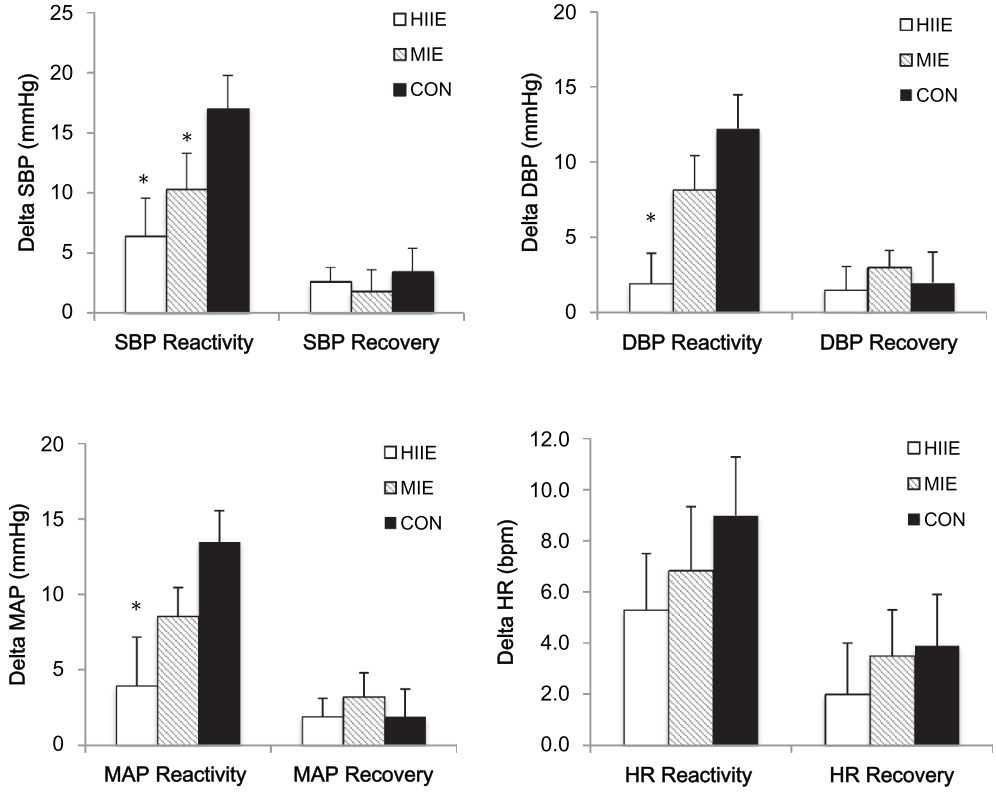

Figure 2. Cardiovascular reactivity and recovery in response to cold pressor task (CPT). Cardiovascular reactivity was calculated as difference (Δ) between during CPT and rest (pre-CPT). Cardiovascular recovery was calculated as difference (Δ) between during CPT and post-CPT. (*) indicates significant difference from CON (p < 0.05). Data reported as mean ± SEM.

high or moderate intensity, 30 minutes prior to undergoing a stress challenge resulted in lower magnitude of BP reactivity compared to no-exercise. The findings of this study lends further support for the position that a prior bout of exercise is beneficial in lowering exaggerations in BP in response to physical and/or psychological stress.

Previous research has pointed out the benefits of an acute session of moderate-intensity exercise in reducing cardiovascular sympathetic responsiveness following a stress challenge (Hamer et al., 2006). However, very few studies have examined whether an acute bout of HIIE is associated with lower cardiovascular reactivity, similar to that observed with moderate-intensity exercise. Our findings showed that systolic BP reactivity induced by the cold pressor task (CPT) was ~60% lower when subjects performed HIIE prior to the stress challenge, compared to control. The magnitude of the increase in mean arterial pressure during CPT was only ~4 mmHg in HIIE compared to ~14 mmHg in control. The observed magnitude of attenuation was comparable to those observed in previous studies involving moderate-intensity aerobic exercise (Hamer et al., 2006), resistance exercise (Moreira et al., 2014) and HIIE (Ketelhut et al., 2016). We also observed that the attenuation effect in systolic BP reactivity in HIIE was accompanied by a lowering of diastolic BP reactivity. This indicates that the stress-buffering effect involved both the systolic and diastolic components, an observation that is consistent with previous studies on stress-induced BP reactivity (Alderman et al., 2007; Ketelhut et al., 2016; Moreira et al., 2014; Rauber et al., 2014).

While it was beyond our scope to examine the underlying haemodynamic mechanisms that accounts for the attenuated reactivity response, some studies have attempted to examine the potential mechanisms. Since BP is the function of cardiac output and systemic vascular resistance, the changes in BP response could be explained by changes to either the neural (autonomic nervous system) or peripheral (vascular) mechanisms (Halliwill, 2001). Brownley et al. (2003) demonstrated a diminished sympathetic neural activity, evidenced by reduced norepinephrine responses following an acute bout of exercise, supporting the neural mechanism theory. Similar studies also corroborated this hypothesis by reporting negative inotropic sympathetic effects *i.e.*, lower stroke volume and cardiac output responses (Neves et al., 2012). Other studies have also shown enhanced postexercise vagal reactivation, favouring a lower stress-induced BP reactivity with acute exercise (Cunha et al., 2015; Gauche et al., 2017; Michael et al., 2017). On the other hand, enhanced vasodilator response mediated by β2-adrenergic receptor responsiveness and increased nitric oxide release have been associated with vascular mechanisms responsible for the blunted BP reactivity following acute exercise (Brownley et al., 2003; Campbell et al., 2011).

The present study also investigated the effects of prior exercise on HR reactivity to a stress challenge; however, the responses were not statistically different. It was worth noting that the absolute HR values prior to stress exposure were higher following HIIE compared to CON due to the intensity of the exercise, delaying the recovery to baseline. Initially, this was thought to contribute to a greater increase in HR reactivity during the subsequent stress exposure. Our observations, however, showed that despite the heightened HR responses following HIIE, the magnitude of HR reactivity was seemingly lower than that of CON. Although our data were insignificant, similar findings from Alderman et al. (2007) affirmed that reductions in HR reactivity during stress exposure were evident even with higher HR values following intense exercise. On the other hand, there also seems to be incongruities when it comes to reports of stress-induced HR reactivity, with some studies reporting reduction (Alderman et al., 2007; Meireles et al., 2020), no differences (Gauche et al., 2017; Neves et al. 2012), while others showed increased reactivity (Moreira et al., 2014; Rauber et al., 2014). One of the reasons for these discrepancies in findings could be attributed to differences in fitness status as untrained subjects were shown to have a blunted stress-induced HR responses compared to trained subjects (Ifuku et al., 2007). Furthermore, although it is generally believed that changes in BP is brought about by changes in HR, this is not always the case. A considerable number of studies have reported post-exercise hypotension (Forjaz et al., 1998; Moreira et al., 2014; Oliveira et al., 2018), or lack of changes in BP (Gauche et al., 2017; Meireles et al., 2020) despite accelerated HR, displaying the uncoupled responses between HR and BP following moderate or high-intensity exercises.

We initially hypothesised that HIIE would elicit a greater attenuating effect in cardiovascular reactivity compared with MIE. Although the current findings showed reductions in BP reactivity in HIIE and MIE compared to control, the differences between these two exercise intensities were not significant. To the extent of our knowledge, very few studies thus far have compared the effects of moderate-intensity and high-intensity exercises on cardiovascular reactivity. A recent study by Meireles et al. (2020) found no differences between moderate-intensity (30-min cycling at 50%–60% of HRR) and high-intensity interval exercise (3-min cycling intervals at 80%–90% of HRR) on SBP/DBP reactivity to CPT in overweight adolescents. On the other hand, Rauber et al. (2014) reported lower BP reactivity to CPT following playing traditional games (VO_2: 23.1 ± 4.0 $ml.kg^{-1}.min^{-1}$) compared to an interactive dance video game (VO_2: 11.7 ± 1.3 $ml.kg^{-1}.min^{-1}$) in children. In an earlier study, Alderman et al. (2007) reported that both low and high-intensity exercises (50–55% and 75–80% VO_2max) resulted in attenuated BP reactivity to a psychological stressor compared to control, with high-intensity exercise showing greater reduction. Hamer et al. (2006) in their review indicated that the attenuation effects of stress-induced BP responses following a bout of exercise seemed to be evident in submaximal exercise bouts lasting at least 30 min at 50% VO_2max, with higher intensities tending to show greater effects. In addition to exercise intensity, it was recently suggested that exercise modality could also play a moderating factor in exercise-induced reductions in BP (Caminiti et al., 2019), and that different exercise modalities have different effects on central haemodynamics, vascular and cardiac functions in healthy and clinical populations (Michael et al., 2017; Pierce et al., 2018; Y. Zhang et al., 2018).

Our current findings show that HIIE resulted in a 10-mmHg attenuation in absolute SBP response to stress, whereas MIE attenuated SBP by 7.5 mmHg. Although the attenuation effects between the exercise conditions were not statistically different, the individual data trends in which the same subjects

underwent all conditions seemed to indicate a consistent tendency towards greater attenuation effects with HIIE compared to MIE. Based on these observations, it is plausible that HIIE may potentially exert a more pronounced stress-buffering effect on BP reactivity. We postulate that perhaps performing bodyweight exercises in HIIE led to a more robust neural and/or peripheral mechanism, compared to the slow-jog exercise in MIE, thereby supporting the exercise modality premise. It may also be conceivable that the number of subjects in this study presents a limitation, causing the study to be slightly underpowered, which results in the inability to detect differences between the two exercise conditions. Nonetheless, we do not undervalue the benefits of moderate-intensity exercise on cardiovascular reactivity. Although the attenuation effect produced by MIE was not statistically meaningful in this study, one could not dismiss the fact that the magnitude of reduction on SBP reactivity was ~42% lower than CON, a magnitude that may suggest clinical relevance in mitigating stress-induced surges in blood pressure over time.

Panaite et al. (2015) in their comprehensive meta-analytic review reported that poor cardiovascular recovery from laboratory challenges predicts future adverse cardiovascular outcomes. We had expected that reduced BP reactivity (resulting from HIIE) would be accompanied by a greater BP and HR recovery in the study, and yet our finding seems to suggest otherwise. It is unclear why the recovery effect was not observed in the present study. In contrast to our findings, Moreira et al. (2014) reported greater BP and HR recovery one-minute post-CPT following resistance exercise when compared to control. They also demonstrated that the attenuation degree of BP reactivity during a CPT challenge was moderately associated with the degree of BP recovery post-exercise, indicating that these two measures were related. However, Morissette et al. (2020) recently demonstrated that BP and HR responses following CPT were not altered when preceded by an acute bout of anaerobic exercise compared to control. These findings may suggest that exercise intensity and/or modality could influence the nature of cardiovascular recovery following a stress challenge.

It is still largely unknown which exercise modality and intensity causes the greatest reduction of cardiovascular reactivity or enhanced cardiovascular recovery. From a methodological point of view, our findings are noteworthy and offers scope for future investigations to compare the effects of different exercise intensities and modalities on stress-induced cardiovascular reactivity and recovery. In relation to the design of the exercise protocol, our study employed bodyweight exercises for HIIE, which seems to be a practical exercise modality for the general population compared to the traditional HIIT methods utilising specialised laboratory or gym-based equipment. Despite the limited number of studies relating to bodyweight or whole-body HIIT that have been published (Machado et al., 2019), we believe that apart from inducing positive adaptations in physical fitness, bodyweight HIIT can be a viable, low-cost alternative to conventional exercises, especially for those who have limited access to gym facilities or are constrained with time. Certainly, precautions should be exercised when implementing this type of training in high-risk populations. One of the main limitations of the present study was that we measured stress-induced cardiovascular reactivity within 30 min of post-exercise; therefore, it is unknown how these responses could be different or maintained over longer periods of time. Studies by Ketelhut et al. (2016) and Alderman et al. (2007) reported that the attenuation effect in stress-induced BP reactivity could still be detected within 45–60 min after completion of high-intensity exercise. Another interesting ground to explore is whether the attenuation in cardiovascular reactivity would be maintained if individuals were to be exposed to stressful stimuli repeatedly. With regard to cardiovascular recovery, the duration of our observation was one-minute post-CPT, while other studies have looked at longer periods of recovery time up to five-minute post-stress stimuli (Meireles et al., 2020; Molina et al., 2016; Moreira et al., 2014: Morissette et al., 2020; M. Zhang et al., 2013). It is possible that if the duration of the observation was lengthened, we would be able to see more noticeable changes. In addition, longer observations would allow for determination of the time taken for BP and HR to return to baseline levels. Panaite et al. (2015) highlighted that the duration of stress-induced CV reactivity may better predict CVD risk than the absolute magnitude of reactivity. Further research is warranted to answer these questions.

Second, assessment of cardiovascular reactivity to CPT was only conducted after HIIE and MIE sessions, and not prior, thus not allowing for pre- and post-exercise comparisons within the group. Previous reports have demonstrated significant attenuations in stress-induced BP reactivity after an exercise session compared to pre-exercise session, highly suggestive of the protective effect of exercise on cardiovascular reactivity (Meireles et al., 2020; Moreira et al., 2014, Neves et al., 2012; Brownley et al., 2003). With regard to measurements of cardiovascular reactivity, we used heart rate monitors and automated oscillometric BP devices to determine HR and BP, respectively, at predetermined intervals, which only reflected cardiovascular responses at specified times. Therefore, we do not disagree that more accurate observations of cardiovascular responses like heart rate variability and beat-to-beat blood pressure variability would have benefitted the robustness of the present study. The present findings must also be interpreted in view of the stressor used in the study. The cold pressor test has been shown to increase heart rate, cardiac contractility, and vascular resistance through sympathetic activation which in turn results in increased blood pressure (Ifuku et al., 2007). It is possible that different stressor tasks such as physiological (*e.g.*, cold pressor task), cognitive (*e.g.*, Stroop task, mental arithmetic task), and psychosocial stressors (*e.g.*, public speaking, mock job interview) may elicit different cardiovascular reactivity responses (Hamer et al., 2006; Zanstra & Johnston, 2011). Furthermore, measurements of stress-induced cardiovascular reactivity and recovery have been largely conducted under controlled laboratory conditions; thus, future studies should consider measuring ambulatory or beat-to-beat blood pressure reactivity in real-life stressors, which could yield more close-to-life responses than those produced in the laboratory. Finally, the subjects of the study were physically inactive, normotensive young adult males, and thus, the present observations may differ across different populations of varying clinical conditions, fitness,

age and gender. Males especially have been shown to exhibit higher sympathetic activity and a lower parasympathetic tone in men compared to females (Voss et al., 2015).

Despite these limitations, our current investigation expands previous findings on acute exercise and cardiovascular reactivity, demonstrating that an acute bout of HIIE is capable of preventing exaggerated cardiovascular responses in stress-induced conditions even in normotensive individuals. From a public health perspective, these findings not only support HIIE as a suitable exercise modality in promoting cardiovascular health, but also that the beneficial effects of HIIE are not only limited to chronic training but to acute bouts as well.

Disclosure statement

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