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ORIGINAL ARTICLE

Autonomic modulation following an acute bout of bench press with and without blood flow restriction

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Abstract

Purpose Traditional resistance exercise decreases vagal tone up to 30 min after an acute bout of resistance exercise, which may increase the risk of cardiovascular events. However, the effects of resistance exercise with blood flow restriction (BFR) on autonomic modulation are unclear. To evaluate autonomic modulation after resistance exercise with and without BFR in resistance-trained men.

Methods Eleven young men volunteered for the study. Autonomic modulation was assessed at rest, 15 (Rec 1), and 25 (Rec 2) minutes after low-load bench press with BFR (LL-BFR), traditional high-load bench press (HL), and a control (CON). Autonomic modulation assessments were expressed as natural logarithm (Ln), and included total power (LnTP), low-frequency power (LnLF), high-frequency power (LnHF), sympathovagal balance (LnLF/LnHF ratio), root mean square of the successive differences (LnRMSSD), and the proportion of intervals differing by > 50 ms from the preceding intervals (LnPNN50). A repeated measures ANOVA was used to evaluate conditions (LL-BFR, HL and CON) across time (Rest, Rec1, and Rec2) on autonomic modulation.

Results There were significant condition by time interactions for LnTP, LnHF, and LnRMSSD such that they were reduced during recovery after LL-BFR and HL compared to Rest and CON. There were no interactions in the LnLF, LnLF/LnHF ratio, and LnPNN50.

Conclusions These data suggest that LL-BFR and HL significantly alter autonomic modulation up to 30 min after exercise with significant reduction after HL compared to LL-BFR when exercise volume is equated.

Keywords Heart rate variability · Vagal modulation · Sympathovagal balance · Resistance exercise

Abbreviations

1RM	1-Repetition maximum
ACSM	American College of Sports Medicine
BFR	Blood flow restriction
BMI	Body mass index
BP	Blood pressure
CON	Control
ECG	Electrocardiogram
HF	High-frequency power
HL	Traditional high-load bench press
HR	Heart rate
HRV	Heart rate variability
LL-BFR	Low-load bench press with blood flow restriction
Ln	Natural logarithm
LnHF	Natural logarithm of high-frequency power
LnLF	Natural logarithm of low-frequency power
LnPNN50	Natural logarithm of number of normal to normal intervals which differed by > 50 ms from adjacent intervals divided by the total number of all normal to normal intervals
LnRMSSD	Natural logarithm of root mean square of successive differences of normal to normal intervals
LnTP	Natural logarithm of total power
LF	Low-frequency power
NN	Normal to normal intervals
PAR-Q	Physical Activity Readiness Questionnaire
TP	Total power

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PNN50	Number of normal to normal intervals which differed by > 50 ms from adjacent intervals divided by the total number of all normal to normal intervals
RMSSD	Root mean square of successive differences of normal to normal intervals

Introduction

The American College of Sports Medicine (ACSM) recommends resistance exercise as a means to develop general health and muscular fitness such as strength, power, and hypertrophy (Position-Stand 2009). Although ACSM recommends that resistance exercise should be performed at 70% 1-repetition maximum (1RM), or greater, to develop muscular hypertrophy, similar improvements in muscular strength and hypertrophy have been shown to occur using resistance exercise with blood flow restriction (BFR) at 20–30% 1RM (Madarame et al. 2008; Takarada et al. 2004; Abe et al. 2005). In addition, low-load resistance exercise with BFR is becoming widely accepted as an alternative exercise modality for individuals who cannot tolerate high-load resistance exercise, or do not have access to heavy weights and gyms (Nakajima et al. 2006).

A decrease in vagal modulation is associated with a greater risk of mortality (Tsuji et al. 1996), cardiac events (Pokorny et al. 2011), and ventricular arrhythmias (Vinik et al. 2011). Researchers have demonstrated that high-load (> 70%1RM) whole-body resistance exercise results in reductions in vagal modulation that may last up to 30 min (Kingsley et al. 2014; Heffernan et al. 2006). However, it has also been demonstrated that following either exclusively lower- or upper-body resistance exercise, this may not be the case. Specifically, high-load lower-body resistance exercise results in significant reductions in vagal modulation (Heffernan et al. 2008; Kingsley et al. 2014; Iglesias-Soler et al. 2015), similar to whole-body resistance exercise. Interestingly, this does not appear to be true in regards to high-load upper-body resistance exercise (Kingsley et al. 2014). To our knowledge, there is only the lone study that has investigated the effects of upper-body resistance exercise on the cardiovasculature. Thus, the effects of upper-body resistance exercise on the cardiovasculature are not well-understood, and more data examining the effects of upper-body resistance exercise on the cardiovasculature are pertinent.

It has been suggested that low-load resistance exercise with BFR may result in a rapid restoration of vagal modulation compared to high-load resistance exercise (Okuno et al. 2014), thereby decreasing the risk for cardiac arrhythmias and sudden death (Pokorny et al. 2011). However, low-load BFR compared to high-load resistance exercise has only been investigated in one study (Okuno et al. 2014). This particular study utilized an acute bout of low-load (40% 1RM) lower-body unilateral leg press resistance exercise for 4 sets of 16 repetitions plus 1 set to volitional fatigue with BFR, using a standard blood pressure cuff inflated to 100 mmHg throughout, and compared it to 4 sets of 8 repetitions plus 1 set to volitional fatigue at 80%1RM (Okuno et al. 2014). These data demonstrated that while both occlusion and intensity reduced vagal reactivation, BFR allowed for a more rapid vagal reactivation compared to the high-load resistance exercise. These data suggest that BFR resistance exercise, due to its lower load, compared to high-load resistance exercise, may result in a cardioprotective, rapid reactivation of the vagus. However, this study only utilized lower-body resistance exercise, and the effect of upper-body resistance exercise with BFR on vagal modulation remains unknown.

Sympathovagal balance is significantly increased after whole-body resistance exercise (Kingsley et al. 2014; Heffernan et al. 2006). In addition, a significant augmentation of sympathovagal balance has also been reported to occur after high-load lower-body resistance exercise up to 25 min postexercise (Kingsley et al. 2014), thereby suggesting an increased risk for an acute cardiac event due to decreases in vagal modulation (Tsuji et al. 1996). Okuno et al. (2014) demonstrated that low-load lower-body resistance exercise had no effect on sympathovagal balance using the previously described protocol. In addition, there does not appear to be an increase in sympathovagal balance after high-load upper-body resistance exercise (Kingsley et al. 2014). These data suggest that both the time course, and magnitude of change, for sympathovagal balance are similar for lower-body and upper-body resistance exercise. However, no data have evaluated the effects of low-load BFR on sympathovagal balance when performed in conjunction with upper-body resistance exercise.

Although ACSM recommends that resistance exercise should be performed using multiple major muscle groups, many resistance-trained individuals train one major muscle group per day such as leg day or chest day for their workout routine. Therefore, the purpose of the present study was to evaluate autonomic modulation, specific vagal reactivation, after an acute bout of upper-body resistance exercise with and without BFR. We hypothesized that the global autonomic modulation and vagal modulation would be significantly decreased with no change in sympathovagal balance after traditional high-load bench press without BFR (HL) compared to low-load bench press with BFR (LL-BFR) in resistance-trained men.

Methods

Subjects

Based on our previous study, changes in the natural logarithm of high-frequency power (LnHF) after free-weights resistance exercise, an effect size of 0.96 was calculated (Kingsley et al. 2016). With a power at 0.8 and significance level at 0.05, and with the effect size of 0.96, 11 participants were required for the present study. Eleven healthy men (20–30 years) that had been participating in resistance training (6 ± 4 years) for at least 3 days a week for a minimum of 1 year, as assessed via a health history questionnaire, volunteered for the study. Participants were excluded if they were taking any medications or supplements known to affect autonomic modulation. All participants were also non-smokers (< 6 months), and free from obesity (body mass index ≥ 30 kg/m^2), hypertension [resting brachial blood pressure (BP) ≥ 140/90 mmHg], upper-body orthopedic injuries, blood clots, cancer, metabolic syndromes, cardiovascular or vascular disease as assessed via Physical Activity Readiness Questionnaire (PAR-Q) and a Health Participant Questionnaire. This research was approved by the Institutional Review Board and was completed in accordance with the Declaration of Helsinki.

Study design

The first visit consisted of assessments of anthropometrics, body composition, and muscular strength on the bench press. The second visit was verification of muscular strength on the bench press. The first and second visits were separated by at least 72 h. The remaining three visits were completed in a counterbalanced design. Participants reported to the laboratory for either LL-BFR, HL, or a quiet control (CON), on three separate days. All participants were asked to avoid food for 3 h, caffeine and alcohol for 12 h, and strenuous exercise for 24 h prior to data collection. Participants reported to the Cardiovascular Dynamics Laboratory, and rested in the supine position for a period of 15 min followed by assessment of autonomic modulation for 5 min (15–20 min). After the completion of either LL-BFR, HL, or CON, participants returned to the supine position for recovery data collection within 2 min. During recovery, autonomic modulation was collected at 15–20 min (Rec 1) and 25–30 min (Rec 2). To control for diurnal variation, all data collection were conducted between 8 am and 12 pm, and at the same time of day (± 1 h) for each participant. These three visits were separated by at least three days to minimize carryover. Participants were asked not resistance train between sessions.

Anthropometrics

A stadiometer and a balance beam scale were used to assess height and weight, respectively. Height was measured to the nearest 0.1 cm and converted to m. Weight was measured to the nearest 0.1 lb and converted to kg. Body mass index was calculated as kg/m^2. A seven-site skinfold was used to assess body density, and the Brozek equation (Brozek et al. 1963) was used to calculate body composition.

Maximal strength

The 1RM test was used to assess maximal strength on the bench press. According to recommendations from the National Strength and Conditioning Association (Haff and Triplett 2015), participants warmed up at 50% of their body weight followed by increasing 5–20 lbs gradually until the participants could not finish the lifts with no more than 5 attempts. The highest weight lifted between the first and second visits was used for the LL-BFR and HL conditions.

Exercise protocol

The LL-BFR consisted of performing 4 sets of bench press for 30, 15, 15, and 15 repetitions at 30% 1RM with 30 s of rest between sets (Yasuda et al. 2006). Two 77 mm-wide knee wraps (Harbinger, Implus LLC, Durham NC, USA) were wrapped at the proximal end of both upper arms at a rating of tightness at 7 out of 10 to occlude the venous blood flow while maintain arterial blood flow (Wilson et al. 2013). The knee wraps stayed on both upper arms throughout the exercise, and were released immediately after completion of the bench press. The HL consisted of 4 sets of bench press for 8 repetitions at 70% 1RM with 60 s of rest between sets. The protocols were designed to equate exercise volume between LL-BFR and HL. Verbal encouragement was provided during the lifting protocol. The CON consisted of 10 min rest in the supine position.

Autonomic modulation

Autonomic modulation was assessed by heart rate variability (HRV) using a modified CM5 electrocardiogram (ECG) configuration (PowerLab, AD Instruments) at a rate of 1000 Hz. Heart rate (HR) was also recorded at rest and during recovery via the ECG. HRV was assessed for 5 min at rest, 15–20 min (Rec 1), and 25–30 (Rec 2) during recovery following standard procedures (Task-Force 1996). During data collection, the breathing frequency of participants was set at 12 breaths/min using a metronome.

Post data collection, the ECG signals were imported into WinCPRS (Absolute Aliens, Turku, Finland) following inspection of noise, ectopics and artifacts. HRV was

assessed in both the frequency and time domains. For the frequency domain, total power (TP) of HRV was used as a global index of autonomic modulation. Low-frequency power (LF 0.04–0.15 Hz) was represented as both sympathetic activation and vagal modulation (Task-Force 1996). High-frequency power (HF 0.15–0.4 Hz) was used to measure vagal modulation (Task-Force 1996). The LF/HF ratio was used to measure of sympathovagal balance (Task-Force 1996). For the time domain, root mean square of successive differences of normal to normal (NN) intervals (RMSSD) and the number of NN intervals which differed by > 50 ms from adjacent intervals divided by the total number of all NN intervals (PNN50) were used as measures of vagal modulation (Task-Force 1996).

A Shapiro–Wilk normality test revealed that TP, LF, HF, LF/HF, RMSSD, and PNN50 were not normally distributed, therefore, these variables were logarithmically (Ln) transformed to meet the assumptions of parametric testing. The intraclass correlation coefficients of LnTP, LnLF, LnHF, LnLF/HF, LnRMSSD, and LnPNN50 were 0.683, 0.536, 0.831, 0.738, 0.853, and 0.925, respectively.

Statistics

A one-way ANOVA was used to determine if there were any significant difference at rest between conditions. A 3 × 3 repeated measures ANOVA was used to determine the effects of conditions (LL-BFR, HL, and CON) across time (rest, Rec 1, and Rec 2) on HR, LnTP, LnLF, LnHF, LnLF/HF, LnRMSSD, and LnPNN50. If the ANOVA noted a significant interaction, paired *t* tests were used to determine significance using Bonferroni correction for post hoc comparisons. Partial eta squared (η_p^2) was used to assess the effect size of each variable. Significance was set at $p \leq 0.05$. Values are presented as mean ± standard deviation (SD). All statistical analyses were completed using IBM SPSS version 24 (Armonk, NY, USA).

Results

The participant characteristics are presented in Table 1.

Measures of autonomic modulation are presented in Table 2. There was a significant condition-by-time interaction for HR ($F_{4,40} = 11.13$, $p < 0.001$, $\eta_p^2 = 0.527$) such that it was significantly increased at Rec1 after HL compared to CON, and Rec2 after HL compared to the rest, LL-BFR, and CON.

In the frequency domain, there were significant condition-by-time interactions for LnTP ($F_{4,36} = 3.54$, $p = 0.015$,

Table 1 Participant characteristics ($N = 11$)

	Participants
Age (years)	23 ± 3
Height (m)	1.77 ± 0.1
Weight (kg)	80.2 ± 9.3
BMI (kg/m^2)	25.9 ± 2.5
Body fat (%)	13.3 ± 4.8

Data are mean ± SD

BMI body mass index

Table 2 Heart rate, and frequency and time domains of heart rate variability at rest and after low-load bench press with BFR (LL-BFR), traditional high-load bench press (HL) and a control ($N = 11$)

	LL-BFR			HL			CONTROL		
	Rest	Rec 1	Rec 2	Rest	Rec 1	Rec 2	Rest	Rec 1	Rec 2
Heart rate (bpm)	59 ± 12	63 ± 14	63 ± 14	59 ± 12	66 ± 14‡	68 ± 13*†‡	58 ± 10	54 ± 8†	54 ± 8
LnTP (ms^2)	8.3 ± 0.9	7.8 ± 0.9‡	7.4 ± 0.9†‡	8.5 ± 1.6	7.6 ± 0.8	7.3 ± 1.0†‡	8.4 ± 0.6	8.4 ± 0.7	8.4 ± 0.7
LnLF (ms^2)	6.9 ± 1.0	6.4 ± 1.1	6.1 ± 1.0	7.0 ± 1.9	6.3 ± 0.8	5.9 ± 1.0	6.7 ± 0.7	7.0 ± 1.0	7.1 ± 0.8
LnHF (ms^2)	7.0 ± 1.4	6.3 ± 1.5	6.3 ± 1.3	7.4 ± 1.8	5.7 ± 1.4	5.6 ± 1.3*†‡	7.1 ± 0.8	7.2 ± 1.3	7.1 ± 1.1
LnLF/HF (ratio)	4.27 ± 0.81	4.70 ± 0.77†	4.59 ± 0.80†	4.17 ± 1.09	5.06 ± 0.96†	5.04 ± 0.91†	4.19 ± 0.82	4.50 ± 0.65†	4.59 ± 0.61†
LnRMSSD (ms)	4.14 ± 0.71	3.76 ± 0.79	3.61 ± 0.68†‡	4.37 ± 1.03	3.38 ± 0.74*‡	3.31 ± 0.70*‡	4.14 ± 0.47	4.24 ± 0.58	4.20 ± 0.54
LnPNN50 (%)	3.37 ± 0.85	3.15 ± 0.99	2.95 ± 1.10	3.74 ± 0.65	2.30 ± 1.27	1.83 ± 1.93	3.70 ± 0.52	3.67 ± 0.90	3.68 ± 0.72

Data are mean ± SD

LnHF natural logarithm high-frequency power, *LnLF* natural logarithm low-frequency power, *LnLF/LnHF* natural logarithm low-frequency power/ natural logarithm high-frequency power ratio, *LnPNN50* number of NN intervals differed by > 50 ms from adjacent interval divided by the total number of all NN intervals, *LnRMSSD* natural logarithm root mean square of successive differences of NN intervals, *LnTP* natural logarithm total power

*$p \leq 0.05$, different from LL-BFR

$^{\dagger}p \leq 0.05$, different from rest

$^{\ddagger}p \leq 0.05$, difference from control

$\eta_p^2 = 0.283$) and LnHF ($F_{4,36} = 6.11$, $p = 0.001$, $\eta_p^2 = 0.404$) such that they were both significantly decreased at Rec a and Rec 2 after LL-BFR and HL compared to the rest with no differences between LL-BFR and HL. Both LL-BFR and HL were significantly reduced compared to the CON at Rec1 and Rec 2. There was a main effect ($F_{2,20} = 7.50$, $p = 0.004$, $\eta_p^2 = 0.428$) of time in LnLF/HF such that it was significantly increased at Rec 1 and Rec 2 after HL and LL-BFR compared to rest. However, there was no difference in LnLF after LL-BFR or HL.

In the time domain, there was a significant condition-by-time interactions for LnRMSSD ($F_{4,36} = 8.12$, $p < 0.001$, $\eta_p^2 = 0.474$) such that they were significantly decreased at Rec 1 and Rec 2 compared to rest after LL-BFR and HL. LnRMSSD did not change after the CON, and was thus augmented at Rec 1 and Rec 2 compared to LL-BFR and HL. There were no statistical differences in LnPNN50 after LL-BFR, HL, or the CON.

Discussion

The purpose of the present study was to evaluate autonomic modulation after acute upper-body resistance exercise with and without BFR. The main finding of the present study was (a) that vagal modulation was significantly decreased up to 30 min after both LL-BFR and HL compared to rest and CON, with significant reductions after HL compared to LL-BFR, (b) that sympathovagal balance was significantly increased up to 30 min in the HL compared to rest, the control and the LL-BFR. These data suggest that an acute bout of low-load upper-body resistance exercise, with and without BFR, may result in an increase for a cardiovascular event due to the reduction in vagal modulation, and the significant increase in sympathovagal balance.

Our data demonstrate significant decreases in vagal modulation after LL-BFR and HL. In addition, there was a significant difference between the LL-BFR and HL, which was in agreement with our hypothesis. These data demonstrate that vagal modulation is attenuated for at least 30 min, following LL-BFR and HL resistance exercise. We only found one study that examined the autonomic modulation after resistance exercise with BFR (Okuno et al. 2014). In agreement with the present study, Okuno et al. (2014) suggested that there was a significant increase in HR and a decrease in LnHF after either high-load (80% 1RM) resistance exercise and low-load (40% 1RM) resistance exercise with BFR which indicates vagal modulation was significantly decreased. Following the high-load and the low-load resistance exercise, the reduction in vagal modulation was sustained up to one hour postexercise. However, Okuno et al. (2014) suggested that there were significant interactions for HR as well as LnHF between high-load resistance exercise and low-load resistance exercise with BFR which are contrary to our results. It is difficult to make comparisons between the present study and Okuno et al. (2014) due to different study designs, especially the different exercise intensities, repetitions and resistance exercises, upper- versus lower-body, which might alter the autonomic modulation responses.

Due to lack of data on HRV comparing the responses to resistance exercise with and without BFR on autonomic modulation, we sought to compare the data from the present study to resistance exercise without BFR that utilized just the upper body. Accordingly, we found one study that investigated autonomic modulation after upper-body resistance exercise (Kingsley et al. 2014). Kingsley et al. (2014) showed a significant reduction in vagal modulation up to 30 min after performing high-load upper-body (seated row and chest press with 3 sets at the 10 RM, with 2 min of rest) resistance exercise. Therefore, it is clear that an acute bout of low-load upper-body resistance exercise with BFR, as well as moderate to high-load upper-body resistance exercise without BFR significantly reduces vagal modulation, which may have implications for cardiovascular risk following acute resistance exercise. In addition, this is the first study to show significant increases in sympathovagal balance after an acute bout of low-load upper-body resistance exercise and after high-load upper-body resistance exercise. Specifically, we noticed a significant increase in sympathovagal balance (LnLF/HF ratio) after HL compared to LL-BFR due to a significant decrease in LnHF, with no change in LnLF after HL, which was contrary to our hypothesis. Okuno et al. (2014) showed there was no change in LF/HF ratio after both high-load resistance exercise as well as low-load resistance exercise with BFR. Iglesias-Soler et al. (2015) reported that no change in Ln LF/HF ratio after high-load resistance exercise, however, both studies (Iglesias-Soler et al. 2015; Okuno et al. 2014) performed only one resistance exercise. Studies (Heffernan et al. 2006; Teixeira et al. 2011; Kingsley et al. 2014, 2016) that utilized multiple exercises demonstrated significant increases in Ln LF/HF ratio, or LF/HF ratio, after high-load resistance exercises. In turn, this indicates that total exercise volume might play an important role in regulation of sympathovagal balance.

Potentially, the different responses in autonomic modulation after an acute bout of resistance exercise might be from different exercise protocols (exercise, set, repetition, load), times of measurements were taken, and body positions (seated or supine) making it difficult to compare responses across studies. The low-load resistance exercise with BFR reduced vagal modulation by 10.0% in the present study while Okuno et al. (2014) showed a 14.1% reduction in vagal modulation after performing unilateral leg press for 4sets of 16 repetitions plus 1 set to volitional fatigue at 40% 1RM with BFR, achieved using standard blood pressure

cuff that inflated to 100 mmHg. This is the only study we found that measured autonomic modulation after an acute bout of low-load resistance exercise with BFR. Unlike Okuno et al. (2014), we used elastic knee wraps as a mean to restrict blood flow in the present study, and it is possible that the pressures might change over sets. Okuno et al. (2014) used nylon blood pressure cuffs which maintained the same pressures over sets. The different percentage of vagal withdraw might be from different pressures of cuffs and resistance exercises which the upper-body exercise has been reported to elicit greater strains in HR and BP than lower-body exercise (Toner et al. 1983; Per-olof et al. 1965) which might decrease autonomic modulation. In the present study, participants performed bench press resistance exercise at 30% 1RM, making the load lower than other previous studies (Heffernan et al. 2006; Kingsley et al. 2014, 2016; Teixeira et al. 2011) that completed multiple resistance exercises at high load. The traditional high-load resistance exercise decreased vagal modulation (LnHF) by 23.0% in the present study while Heffernan et al. (2006) and Kingsley et al. (2014) reported that vagal modulation reduced, measured via LnHF, by 26.7% and 19.0% after completing 3 sets of 10 repetitions at an intensity of 10RM on 8 and 4 resistance exercises machines, respectively. Kingsley et al. (2016) found vagal modulation, measured via LnHF, decreased by 26.8% after performing 3 sets of 10 repetitions at 75% 1RM on 3 free-weights resistance exercise, including the bench press, squat, and deadlift, which was drastically different than the present study. Teixeira et al. (2011) accomplished 3 sets of 20 repetitions at 50% 1RM on 6 resistance exercises machines, and vagal modulation, measured via LnHF, dropped by 43.5%. It is possible that greater activities of main (Escamilla et al. 2001), agonist (McCaw and Friday 1994), and stabilizing (Santana et al. 2007) muscle groups using higher load, free weights, and/or multiple resistance exercises in protocols, might lead to greater reduction in vagal modulation compared to low-load resistance exercise with BFR (Okuno et al. 2014). However, it is not surprising that our results demonstrate a significant decrease in vagal modulation compared with other aforementioned studies even though our participants underwent only one free-weight resistance exercise at a lower load with BFR. The range of different times of measurements were taken is from immediately post (Iglesias-Soler et al. 2015), 10–15 min (Okuno et al. 2014), 25–30 min (Heffernan et al. 2006; Kingsley et al. 2014, 2016), to 60 min (Teixeira et al. 2011) after an acute bout of resistance exercise. In the present study, we collected HRV from 15 to 20 and 25–30 min during recovery. In addition, we noted significant decreases in LnTP and LnRMSSD up to 30 min during recovery after both LL-BFR and HL while significant reduction in LnHF and LnRMSSD after HL compared to LL-BFR. In addition, HRV was measured with participants resting in the supine position in the present study. The positions during HRV measurements were in seated positions (Iglesias-Soler et al. 2015; Okuno et al. 2014; Teixeira et al. 2011) or supine position (Heffernan et al. 2006; Kingsley et al. 2014, 2016). Moreover, significantly higher vagal modulation is found in the supine position than in the seated position (Barak et al. 2010; Young and Leicht 2011) due to orthostatic load (Perini and Veicsteinas 2003) and control of BP and HR via the baroreflex (Task-Force 1996).

The present study is not without limitations. First, we determined the tension of the knee wraps using a rating of perceived pressure. The rating of perceived pressure at 7 of 10 may have different responses on autonomic modulation across our participants due to differing percentages of BFR. Second, we recruited resistance-trained individuals, but the differences of training statues are varied which may cause different physiological changes in response to an acute bout of resistance exercise with BFR in terms of autonomic modulation.

In conclusion, although it is difficult to compare across studies due to different study designs, we observed significant reductions in vagal modulation up to 30 min after an acute bout of traditional high-load bench press resistance exercise as well as an acute bout of low-load bench press resistance exercise with BFR, with significant decreases after traditional high-load bench press resistance exercise compared to low-load bench press resistance exercise with BFR, without differences in exercise volume between conditions in the present study. Futures studies should evaluate the difference between upper- and lower-body low-load resistance exercise with BFR, and the difference between knee wraps and nylon cuffs on autonomic modulation. In addition, it is important to exam acute and chronic low-load resistance exercise with blood flow restriction on autonomic modulation.

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Author contributions YLT and JDK conceived and designed. YLT, EMM, AG, JCP, and LS conducted experiments. YLT and JDK analyzed data. YLT and JDK wrote the manuscript. All authors read and approved the manuscript.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

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