



Journal of Sports Sciences

ISSN: 0264-0414 (Print) 1466-447X (Online) Journal homepage: https://www.tandfonline.com/loi/rjsp20

Autonomic response to a short and long bout of high-intensity functional training

Brian Kliszczewicz, Cassie Williamson, Emily Bechke, Michael McKenzie & Wade Hoffstetter

To cite this article: Brian Kliszczewicz, Cassie Williamson, Emily Bechke, Michael McKenzie & Wade Hoffstetter (2018) Autonomic response to a short and long bout of high-intensity functional training, Journal of Sports Sciences, 36:16, 1872-1879, DOI: 10.1080/02640414.2018.1423857

To link to this article: https://doi.org/10.1080/02640414.2018.1423857



Published online: 08 Jan 2018.



Submit your article to this journal 🕑

Article views: 368



View related articles



View Crossmark data 🗹

Autonomic response to a short and long bout of high-intensity functional training

Brian Kliszczewicz^a, Cassie Williamson^a, Emily Bechke^a, Michael McKenzie^b and Wade Hoffstetter^a

^aDepartment of Exercise Science and Sport Management, Kennesaw State University, Kennesaw, GA, USA; ^bDepartment of Exercise Science, Winston-Salem State University, Winston-Salem, NC, USA

ABSTRACT

The evaluation of Autonomic Nervous System (ANS) recovery following exercise provides insight into the transient stress placed on the cardiovascular system. High-Intensity Functional Training (HIFT) is a form of intense exercise that is prescribed in various modalities and durations; however, little is known about the influence of HIFT duration on ANS recovery. Ten apparently healthy males (28.1 \pm 5.4 yrs) performed two HIFT sessions (<5-minute and 15-minute) in a crossover fashion. ANS activity was measured using plasma Epinephrine (E) and Norepineprine (NE); Heart Rate Variability markers of the log transformed Root Mean Square of Successive Differences (InRMSSD) and High-Frequency power (InHF). No trial dependent differences were observed in InRMSSD (p = 0.822), InHF (p = 0.886), E (p = 0.078), or NE (p = 0.194). A significant main time effect was observed in both trials with a depression in InRMSSD and InHF following the trials (p < 0.05) and recovering by 2-hours post (p < 0.05) and recovered by 1-hour post (p > 0.999, p > 0.999) respectively. The HIFT bouts examined within this study demonstrated similar transient strain of the ANS.

ARTICLE HISTORY Accepted 1 January 2018

KEYWORDS Autonomic stress; epinephrine; norepinephrine; heart rate variability; exercise

Introduction

Recently, amongst the general population and applied research settings, there has been an increased interest in short-duration and high-intensity bouts of exercise, in part due to commonly reported time constraints. High-Intensity Interval Training or Sprint Interval Training are single-modality exercises that have been shown to lack compliance amongst the general population due to required levels of motivation and discomfort (Gibala, 2007). An alternative to this style of training is High-Intensity Functional Training (HIFT), which is a competitive and functional based exercise style, that has been shown to elicit greater participation within the general population when compared to moderate aerobic and resistance training (Heinrich, Patel, O'Neal, & Heinrich, 2014). HIFT employs multiple energy pathways through the multimodal prescription of resistance and aerobic based conditioning in conjunction to plyometric, and gymnastic movements performed at varying durations (Heinrich et al., 2014). Due to the multiple components, repetition schemes, and durations that makeup HIFT, prescribed bouts can range from body weight exercises (e.g., push-ups, sit-ups, and squats) performed in a timed circuit to more complicated schemes involving Olympic lifts (e.g., cleans, snatch, jerk) for a set number of reps, making the prescription unlimited. HIFT exercise sessions can last as little as 2 minutes, to more than an hour, with the average bout typically lasting around 10 minutes in duration (Paine, Uptgraft, & Wylie, 2010). To this point, it is unknown how these variations influence the magnitude of cardiovascular stress experienced and may lead to concern for potential health risks such as prolonged periods of autonomic nervous system (ANS) imbalance, which has been related to increased likelihoods of acute cardiac events (Albert et al., 2000; Eijsvogels, Fernandez, & Thompson, 2016).

A pragmatic and relatively non-invasive evaluation of ANS activity can be achieved through the measurement of heart rate variability (HRV) and plasma catecholamine concentration. HRV is the measurement and quantification of the timing between consecutive R-R intervals derived from an electrocardiogram or beatto-beat guantifying device (e.g., heart rate monitor) (Malik, 1996). The root mean square of successive differences (RMSSD) and the frequency domain index of high frequency (HF) are markers of parasympathetic activity that can be derived from HRV analysis and are sensitive to periods of physical stress and activity (Buchheit, Papelier, Laursen, & Ahmaidi, 2007; Malik, 1996). Additionally, concentrations of circulating plasma catecholamines, epinephrine (E) and norepinephrine (NE), are sympathetic neurohormones that are elevated during periods of stress (Børsheim, Knardahl, Høstmark, & Bahr, 1998). Exercise is a physiological stressor that results in the depression of HRV and subsequent increase in circulation of E and NE. Upon completion of exercise bouts, a rebound in HRV and clearance of E and NE is observed (Kjaer, Christensen, Sonne, Richter, & Galbo, 1985; Kliszczewicz et al., 2016). Therefore, ANS responsiveness can be used to gauge transient stress of the cardiovascular system following an exercise bout based on the duration of HRV depression and magnitude of catecholamine response following exercise (Kliszczewicz et al., 2016; Seiler, Haugen, & Kuffel, 2007; Spiering et al., 2008). Studies have shown that the degree of ANS responsiveness is influenced by the intensity, duration, and modality of the exercise

(Kliszczewicz et al., 2016). For instance, Heffernan, Kelly, Collier, and Fernhall (2006) demonstrated that a resistance-based exercise bout (3 sets of 10-repetitions of various exercises) created a greater depression of HRV when compared to a 30-minute continuous aerobic bout (65% of VO₂ peak). Parekh and Lee (2005) examined the influence of exercise intensity, low (50% VO₂max) vs. high (80% VO₂max), on HRV recovery and found that the high-intensity group had greater HRV depression. Previous research performed in this lab demonstrated that when matched for time and intensity, a HIFT bout created a greater disturbance in HRV when compared to a treadmill bout (Kliszczewicz et al., 2016). However, the examination of ANS activity comparing differences within the HIFT modality is understudied.

Currently, the number of studies that evaluate recovery of the ANS following bouts of HIFT are limited and the examination of its varying arrangements have yet to be examined. Research in this area may provide important information regarding safe, effective and time efficient prescription of HIFT programming for those seeking to participate. Therefore, the purpose of this study was to examine the acute ANS response in physically active men following two common styles of HIFT that are of short and long duration.

Methods

Experimental design

All data collection was performed in the Exercise Physiology Laboratory (EPL). Participants were asked to arrive at the EPL on three separate occasions at roughly the same time, each visit taking place within one week of each other between the times of 6 AM and 11 AM. The initial visit consisted of a review of the procedures, signing of the informed consent, completion of the health history questionnaire, and a graded exercise test (GXT) to determine aerobic capacity. The remaining two sessions were performed in a randomized crossover fashion between the SHORT (<5-min) and LONG (15-min) bouts of HIFT with preexercise and post-exercise measurements of HRV collected through a heart rate monitor (Polar Team²). Additionally, preexercise and post-exercise concentration of E and NE were obtained through plasma sampling. Finger capillary sticks were used to collect Lactate (La) in order to gauge exercise intensity, while hemoglobin (Hb), and hematocrit (Hct) were collected to calculate plasma volume shifts. After baseline samples were obtained, participants engaged in a 5-minute, self-selected warm-up followed by the exercise bout of SHORT or LONG. After the completion of the bouts, designated blood draws and HRV markers were collected. Experimental design can be seen in Figure 1. Participants were not allowed to eat until 3-hours following the exercise bout.

Participants

The University Institutional Review Board approved all testing protocols and procedures of this study. Fifteen physically fit males with a minimum of 3-months of HIFT experience participated in this study. Those who reported having any orthopedic problems, conditions, or were suffering from any cardiovascular, pulmonary, or metabolic diseases were excluded from the study as determined through a PAR-Q and Health History Questionnaire. Participants were required to be able to perform all of the prescribed movements and complete 30 clean & jerks at 61.4 kg in less than 5-minutes in order to be included in this study. Those who were taking prescription medications known to influence autonomic or metabolic activity or had any symptoms or contraindications of health were excluded from this study. Prior to participation, each participant reviewed and signed the informed consent. Prior to the first testing session, participants were asked to wear light, comfortable clothing, fast for a minimum of fourhours, abstain from alcohol and exercise for 24-hours, and avoid caffeine and over the counter supplementation 12hours. Prior to each HIFT trial, participants were instructed to abstain from exercise for 48-hours, alcohol for 24-hours, and caffeine for 12-hours. Participants were asked to eat a moderate breakfast of their choice before their arrival in order to avoid post-exercise hypoglycemia. Additionally, participants were asked to repeat this meal for consistency between trials.

Anthropometric measurements & graded exercise test (GXT)

Aerobic capacity (VO₂max) was assessed during the first visit through a graded exercise test (GXT) on a treadmill (Woodway USA, Waukesha, WI) using a modified ramp protocol at a self-selected speed with increases in elevation (percent grade) of one percent (1%) every minute until completion of the test. Expired gas fractions were sampled using a portable metabolic system (Cosmed K4b², Rome, Italy). VO₂max test completion criteria require two of the following: RER > 1.10, heart rate (HR) within 10 bpm of age-predicted HRmax, lactate (La) concentration \geq 8 mmol/L, volitional fatigue, or a plateau in VO₂ with increasing workload. Resting and maximal effort La levels (mmol/L) were collected before and after the GXT using a portable lactate analyzer (Lactate Plus, Nova Biomedical, Waltham, MA).

Anthropometric measurements were collected using an electronic physicians scale (Tanita WB 3000, Arlington Heights, IL) to measure height (cm) and weight (kg). Body composition analysis (percent body fat and fat free mass) was assessed using dual-energy x-ray absorptiometry scan

Crossover						
PRE	Warma Lin	SHORT	IDE	1HR	2110	211D
PRE	Warm-Up	LONG	IPE	IHK	2HR	3HR
Blood Draw Finger Stick 10-min R-R	Self-selected		Blood Draw Finger Stick 45-min R-R	Blood Draw Finger Stick	10-min R-R	Blood Draw Finger Stick

1874 🛞 B. KLISZCZEWICZ ET AL.

Table 1. Participant characteristics.

Characteristic	$Values \pm SD$
Age (y)	28.1 ± 5.4
Height (cm)	176.1 ± 8.0
Weight (kg)	88.0 ± 10.4
Body Fat (%)	17.9 ± 5.0
VO_2 max (mL × kg ⁻¹ × min ⁻¹)	43.5 ± 5.2
GXT Max HR (b \times min ⁻¹)	186.3 ± 11

(GE Lunar iDXA). Participant characteristics and body composition data are presented in Table 1.

Short HIFT trial

The SHORT bout chosen for this study was the CrossFit® named workout "Grace." The workout "Grace" consists of 30 power clean & jerks at 61.4 kg using an Olympic barbell. The beginning of the movement starts with the barbell on the ground, a power clean is performed, followed by a shoulder to overhead movement (i.e., jerk). Full extension was necessary at the end of the movement for the repetition to count. These movements and standards were repeated for 30 repetitions at a self-selected pace to complete the workout as fast as possible. Participants were allowed to rest ad libitum. This protocol was chosen due to its common prescription amongst the participants, full body muscle recruitment, and provided a time based inclusion criteria (i.e., <5 minutes to complete the workout). A circuit was not selected for the SHORT bout to avoid potential over pacing and because it was believed that a "complete for time" trial would have yielded greater effort. Prior to the exercise bout, a 10-minute baseline R-R interval recording to measure HRV was taken. In addition, resting lactate (mmol/L), hemoglobin (g/dL), hematocrit (Alere Hemopoint 2, Waltham, Massachusetts), and a pre-exercise blood draws were taken. Upon completion of the workout, participants were placed in a seated position and postexercise blood draws for E and NE were taken at designated time points. Following blood draws, participants remained in the seated position and R-R interval recordings for HRV were taken at the designated time points.

Long HIFT trial

The LONG bout chosen for this study was a 15-minute circuit created by a Level I certified CrossFit® Coach. This circuit consisted of a 250-meter row on a rowing ergometer (Concept 2), 20-kettlebell swings at 16 kg, and 15-dumbbell thrusters with two 13.6 kg dumbbells. The objective of the workout was to complete as many repetitions as possible within the 15-minutes. Participants were allowed to rest ad libitum. For scoring purposes, every 10-meters on the rowing ergometer equaled one repetition. The standard resistance for the rowing ergometer was for the damper to be set at six. Kettlebell swings began with the kettlebell at the starting position between the legs and off the ground. The kettlebell is then swung overhead until achieving an upright position with the kettlebell directly overhead with arms in the locked position. The dumbbell thruster consisted of holding the dumbbells in the front rack position, completing a full front squat into an overhead press with hips open and arms locked at the end of the movement. The participant could not continue onto the next movement until all prescribed repetitions were completed. All movements had to be completed within the standards in order for the repetition to be counted. The LONG bout was not designed as a "complete for time" scheme in that a longer duration workout would have yielded too great of a variation in the time to completion. The current protocol was selected due to its duration, full body make up, and ability to ensure that participants completed at the same time. Prior to the exercise bout, a 10-minute baseline R-R interval recording to measure HRV was taken. In addition, resting lactate, hemoglobin, hematocrit, and pre-exercise blood draws were taken. Upon completion of the workout, participants were placed in a seated position and post-exercise blood draws for E and NE were taken at designated time points. Following blood draws, participants remained in the seated position and R-R interval recordings for HRV were taken at the designated time points.

Heart rate variability

During all time points of HRV measurement, participants were placed in a quiet, dimly lit room and instructed to remain as still as possible while in a seated position. HRV recordings were collected using the Polar Team² system (Lake Success, NY). Three separate HRV recordings were obtained throughout the second and third visit; two 10-minute HRV recordings taken prior to exercise (PRE) and 2-hours post-exercise (2HP), one 45-minute recording was taken immediately following the exercise bout. HRV recordings obtained were divided into 5-minute segments for analysis: the PRE and 2-HP time points measured the last 5-minutes of the 10-minute recordings; the 45-minute post recording was broken down into eight POST 5-minute segments at 5-10, 10-15, 15-20, 20-25, 25-30, 30-35, 35-40, and 40-45 minutes. The presence of artifact noise was filtered through a piecewise cubic spline interpolation method using a "low artifact correction" with a sensitivity set to identify any R-R abnormality ±0.35 sec compared to the local average through a function available in the Kubios software (Kubios V 2.2, Joensuu, Findland) (Nakamura et al., 2017; Tarvainen, Niskanen, Lipponen, Ranta-Aho, & Karjalainen, 2014). In order to avoid distortion of analysis, any segments containing three or more irregular R-R intervals (e.g., artifact or ectopic beats) (Giles, Draper, & Neil, 2016) were excluded from analysis.

The HRV markers selected for this study were the time domain index of the root mean square of successive differences (RMSSD) and the frequency domain index of high frequency (HF) of the power spectral density (0.15– 0.40 Hz). Acquired R-R interval recordings were transformed into time and frequency domain components using specialized online HRV software (Kubios, Version 2.2). In order to assess RMSSD, R-R intervals were converted into a tachogram, which plots the successive R-R intervals (y-axis) against the number of beats within the total number of beats in the recording (x-axis). Five-minute recordings were sampled from the tachogram in order to analyze RMSSD. HF was analyzed through power spectral analysis through the application of a fast Fourier transformation of the R-R interval recording with a window width of 256 s and overlap of 50%. RMSSD and HF are widely accepted markers of parasympathetic activity and are commonly used to assess vagal activity following exercise (Buchheit et al., 2007; Malik, 1996; Stanley, Peake, & Buchheit, 2013).

Blood samples & storage

Blood samples were collected to determine catecholamines, E and NE, pre-exercise (PRE), immediately post-exercise (IPE), 1hour post exercise (1-HR), 2-hours post exercise (2-HR), and 3hours post exercise (3-HR). Blood draws were performed by a trained phlebotomist and taken via venipuncture through the antecubital vein while the participant was in a seated position. Blood was collected in 12-mL heparinized tubes and samples were immediately centrifuged at 2500 rpm for 15-minutes. The samples were then aliquoted and stored in an ultralow freezer at -80°C until the assays were performed. Hb and Hct were determined via finger capillary sticks at each blood draw time point (Alere Hemopoint 2). Participants were asked to abstain from food until after the 2-HR time point.

All samples were assayed for E and NE using a commercially available ELISA kit (DLD, Hamburg, Germany). Protocols followed the instructions provided by the manufacturer. In order to account for plasma volume shifts that occurred following the HIFT bouts, all samples were normalized based on the established protocols of Dill and Costill (1974) through Hb and Hct levels. The procedures and findings for plasma catecholamines were previously reported and permissions were granted by the publishing journal (Kliszczewicz, Buresh, Bechke, & Williamson, 2017).

Statistical analysis

HRV data was entered into a statistical software program SPSS 19.0 (Chicago, Illinois, USA). In order to determine normal distribution of the HRV data set, a Shapiro-Wilk test was performed. A violation of normality occurred and therefore a natural logarithmic transformation was performed on RMSSD and HF prior to further statistical analysis and reported as InRMSSD and InHF. In order to assess differences from resting to post-exercise, as well as between HIFT trials (SHORT and LONG), a 2 (trial) × 10 (time) repeated-measures analysis of variance (ANOVA) with a Bonferroni adjustment was ran for both InRMSSD and InHF. A paired samples t-test was run in order to further assess differences between same trial-time points. The statistical significance was set to an alpha of <0.05. The data is presented in the mean \pm standard deviation.

Participant plasma E and NE data was entered into statistical software program SPSS. In order to assess differences between resting and post-exercise, as well as between HIFT trials (SHORT and LONG), a 2 (trial) \times 4 (time) repeated measures ANOVA with a Bonferroni adjustment was ran on plasma E and NE concentration. A paired-samples t-test was run in order to further assess differences between same trial-time points. The statistical significance was set to an alpha of <0.05. The data is presented in the mean ± standard deviation (SD).

Effect sizes were determined using the recommend guidelines of Quintana (Quintana, 2017) for HRV analysis. Effect sizes thresholds were categorized as small effect (<0.25), moderate effect (0.50), and large effect (0.90).

Results

Of the 15 original participants, 10 completed all pre-exercise and post-exercise HRV analyses and blood draws for SHORT and LONG HIFT bouts: Two participants were excluded due to vasovagal responses to the phlebotomy procedure, two participants withdrew due to injury experienced outside the study, and one due to a scheduling conflict. The completed 10 participant characteristics can be seen in mean \pm SD in Table 1. Performance outcome measures, La, average HR, time to completion or repetitions completed for the SHORT and LONG bouts of HIFT can be seen in Table 2. HRV presented as InRMSSD (Figure 2(a)) and InHF (Figure 2(b)); plasma catecholamines are presented in E (Figure 3 (a)) and NE (Figure 3(b)) (Kliszczewicz et al., 2017).

Heart rate variability

Repeated measures ANOVA showed no main trial-dependent differences between the SHORT and LONG bout of HIFT (Figure 2). However, a paired-samples t-test revealed only one point of significant difference between the trials at 5–10 min post in InHF (Table 3). A main time-dependent effect was observed in both InRMSSD and InHF. For instance, InRMSSD experienced a significant reduction in all post-exercise measures (p < 0.05) and did not return to baseline until 2-HP (p = 0.141) when compared to pre-exercise values. Similarly, InHF experienced a significant reduction in all post-exercise measures (p < 0.05) and did not return to baseline until 2-HP (p > 0.999) when compared to pre-exercise values.

Catecholamines

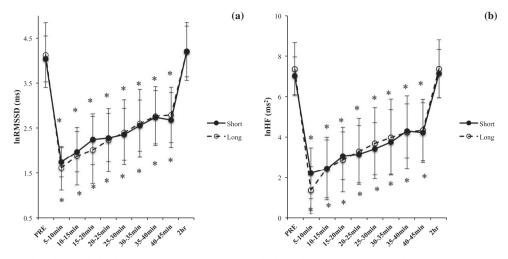
Repeated measures ANOVA revealed no trial-dependent differences between the SHORT and LONG bout of HIFT (Table 3). However, a significant main time-dependent effect was observed. For instance, E significantly increased at IPE (p = 0.002) and returned to baseline by 1-HP (p > 0.999), while NE significantly increased at IPE (p < 0.001) and returned to baseline by 1-HP (p > 0.999).

Discussion

The purpose of this study was to examine the acute ANS response in physically active men following two common styles of HIFT that are of short and long duration through the analysis of HRV and plasma catecholamines. The primary

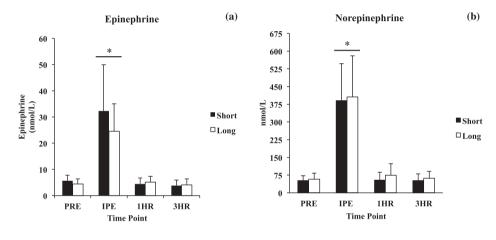
Table 2. Performance measures.	
--------------------------------	--

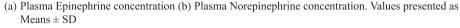
MEASURES	SHORT	LONG
Lactate (mmol/L)		
PRE	1.1 ± 0.4	0.99 ± 0.6
POST	14.3 ± 2.0	13.7 ± 1.5
Average HR (bpm)	172.4 ± 6.3	170 ± 9.5
%HRmax	92.7 ± 4	91.3 ± 3
Score (seconds)	206.4 ± 60.2	-
Score (repetitions)	-	274 ± 48.6



(a) Time domain index lnRMSSD. (b) Frequency Domain index lnHF. Values presented as Means ± SD * = Significantly different from PRE

Figure 2. Heart rate variability log transformed.





* = Significantly different from PRE

Figure 3. Plasma catecholamines.

findings of this study revealed that there were no trialdependent differences in either marker of ANS activity. However, a significant time-dependent difference was observed in both SHORT and LONG bouts of HIFT. These results support the literature demonstrating that significant depressions in InRMSSD and InHF with concurrent increases in plasma E and NE occur following bouts of high-intensity exercise (Kliszczewicz et al., 2016; Stanley et al., 2013). An unexpected outcome of this study was that the different variations of HIFT did not influence the magnitude of ANS disruption.

It is well established that the onset of exercise results in a shift in autonomic balance caused by a withdrawal of PNS activity and concurrent increase in SNS drive (Borresen & Lambert, 2008; Buchheit et al., 2007; Kliszczewicz et al., 2016). The exercise induced disruption of ANS activity is generally innocuous (Eijsvogels et al., 2016); however becomes a point of concern during periods of prolonged disruption that occurs during or

following bouts of exercise, which can trigger arrhythmias in those with underlying conditions (Franciosi et al., 2017). Though it is important to note the concerns associated with prolonged ANS disruption, the purpose of this study was not to assess cardiovascular safety, but rather to establish the degree of ANS perturbation in order to more efficiently prescribe HIFT like workouts. As a general rule, the degree and duration of which ANS activity is disrupted is based on the make up of the exercise bout (e.g., duration, modality, intensity) (Heffernan et al., 2006; Kliszczewicz et al., 2016; Parekh & Lee, 2005). With the recent development of mixed-modality programs such as HIFT, it becomes more difficult to identify and establish which factors elicit the greatest perturbations in ANS activity. Exercise intensity has been shown to be the most influential factor in measures of HRV, with greater disruptions being observed at higher intensities (Stanley et al., 2013). In the current study, HRV depression and recovery following the SHORT and LONG bout of HIFT were nearly identical, despite the differences in make up and duration,

Table 3. Between trial comparison of autonomic nervous system markers of stress.

Marker	SHORT	LONG	р	Cohen's d
InRMSSD (ms)				
PRE	4.03 ± 0.51	4.12 ± 0.71	0.591	0.14
5–10 min	1.74 ± 0.32	1.61 ± 0.48	0.486	0.31
10–15 min	1.97 ± 0.45	1.88 ± 0.64	0.362	0.16
15–20 min	2.25 ± 0.56	2.02 ± 0.74	0.183	0.35
20–25 min	2.28 ± 0.54	2.23 ± 0.70	0.742	0.07
25–30 min	2.36 ± 0.58	2.40 ± 0.72	0.770	0.06
30–35 min	2.56 ± 0.56	2.60 ± 0.75	0.739	0.06
35–40 min	2.74 ± 0.58	2.77 ± 0.65	0.810	0.04
40–45 min	2.68 ± 0.61	2.79 ± 0.61	0.338	0.18
120 min	4.20 ± 0.56	4.20 ± 0.64	0.964	0.00
InHF (ms ²)				
PRE	7.01 ± 0.92	7.34 ± 1.31	0.206	0.29
5–10 min	2.21 ± 1.25	1.38 ± 1.17	0.020 ^a	0.68
10–15 min	2.43 ± 1.42	2.45 ± 1.55	0.956	0.013
15–20 min	3.07 ± 1.18	2.89 ± 1.59	0.578	0.12
20–25 min	3.16 ± 1.42	3.29 ± 1.63	0.718	0.08
25–30 min	3.42 ± 1.29	3.70 ± 1.76	0.403	0.18
30–35 min	3.76 ± 1.60	3.99 ± 1.87	0.466	0.13
35–40 min	4.31 ± 1.32	4.24 ± 1.78	0.849	0.04
40–45 min	4.23 ± 1.46	4.35 ± 1.50	0.590	0.08
120 min	7.12 ± 1.19	7.36 ± 1.43	0.207	0.18
E (ng·ml ^{−1})				
PRE	5.70 ± 2.06	4.44 ± 1.92	0.066	0.63
IPE	32.27 ± 17.67	24.52 ± 10.52	0.079	0.53
1-HP	4.44 ± 2.31	5.10 ± 2.23	0.355	0.29
3-HP	3.82 ± 2.13	4.04 ± 2.29	0.654	0.09
NE (ng⋅ml ⁻¹)				
PRE	51. 97 ± 20.17	57.82 ± 25.22	0.430	0.25
IPE	390.75 ± 155.8	406.16 ± 173.8	0.656	0.09
1-HP	53.61 ± 33.45	74.09 ± 49.31	0.094	0.48
3-HP	51.22 ± 28.82	61.09 ± 30.07	0.280	0.33

HRV markers are presented as the mean ± SD. InRMSSD, root mean square of the SD of consecutive N-N intervals; HF, frequency domain of high frequency (0.15– 0.40 Hz). Catecholamine markers are presented as the mean ± SD. E, plasma Epinephrine. NE, plasma Norepinephrine. ^aRepresents significance of difference between SHORT and LONG time points.

which may suggest that the exercise intensity was the greater influencing factor. The percent HRmax measured during the SHORT bout was 92.7 \pm 4%, while the LONG bout elicited a 91.3 \pm 3% HRmax; each bout being classified as vigorous intensity (Garber et al., 2011), supporting the notion that the degree of HRV depression is greatly influenced by exercise intensity.

Modality has also been shown to be an influencing factor in ANS disruption, with resistance-based exercise resulting in the greatest disruptions (Heffernan et al., 2006). When compared to aerobic based training, resistance exercises generally cause a greater depression in markers of HRV (Heffernan et al., 2006). In some cases, HRV can be depressed up to 72hours following maximal resistance based bouts (i.e., 1-RM testing) (Chen et al., 2011). Even lower resistance such as body weight training has been shown to elicit greater HRV depression when compared to a time and intensity-matched aerobic based bout (Kliszczewicz et al., 2016). Despite HIFT exercise being categorized under a similar scope, the modalities within the SHORT and LONG bouts vary greatly (e.g., power clean vs. row, kettlebell swing, thruster). The HIFT bouts in this study used light to moderate resistance load with varying whole body movements and demonstrated no differences in HRV depression. This suggests that resistance based modalities that are whole body inclusive provide a greater potential for disruption in HRV. Currently, there is a lack of research regarding resistance based modalities, the intensity in which they are performed and its influence on HRV. Further investigation is needed.

Plasma catecholamines are commonly elevated following exercise in order to meet the increased demands of the cardiovascular system (Franciosi et al., 2017); however, the magnitude at which they are elevated has been shown to vary greatly between differing bouts (Kliszczewicz et al., 2016). Generally speaking, exercise intensity elevates plasma E and NE in a linear fashion (Bahr, Høstmark, Newsholme, Grønnerød, & Sejersted, 1991; Børsheim et al., 1998); however, this relationship does not always apply. Intensity-matched bouts of treadmill running and HIFT elicited different catecholamine responses following the trials in which plasma E increased ~33%, ~200% and NE increased ~150%, ~300%, respectively (Kliszczewicz et al., 2016). The findings of the current study demonstrated significant elevations of plasma E (685 \pm 601%, 620 \pm 358) and NE (779 \pm 313%, 736 \pm 271%) at IPE following the SHORT and LONG bouts of HIFT; however, no significant differences between the SHORT and LONG catecholamine concentrations were observed. These results demonstrate that the catecholamine response was not greatly influenced by the duration of the bouts. Regardless of intensity and modality of exercise, it has been well established that the rate of clearance of plasma catecholamines occurs within 1-hour of bout termination (Børsheim et al., 1998). The findings of the current study supported the literature, demonstrating that catecholamine levels returned to resting levels by 1-HP following both SHORT and LONG bouts of HIFT.

To date, few studies have observed a duration-dependent relationship between bouts of exercise and their influence over ANS activity. These studies that have been performed show conflicting results in magnitude and duration of ANS depression. For instance, Pichon, De Bisschop, Roulaud, Denjean, and Papelier (2004) observed linear decreases in HRV through power spectral density analysis with increasing exercise durations (3-min, 6-min, and 9-min) at various intensities (60%, 70%, and 80% PVO₂max). However, this relationship was not observed by Seiler et al. (2007) who demonstrated that exercise performed below and above ventilatory threshold at various durations elicited similar changes in HF and RMSSD. When examining the SNS activity markers, E and NE, Galbo, Christensen, and Holst (1977) observed a linear increase throughout continuous running at 60% of VO2max. Though not necessarily conflicting with findings of durations influence, increases in exercise intensity have been found to yield a more robust increase in plasma catecholamines (Kjaer et al., 1985). The available literature suggests that duration may not be as influential as other factors (e.g., modality, intensity), which is consistent with the findings of the current study. It is important to note that the majority of research examines the effect of duration on single-modality and continuousbased exercise (Børsheim et al., 1998; Galbo et al., 1977; Seiler et al., 2007). The current study is unique due to its comparison of a short $(3.44 \pm 1 \text{ min})$ and moderate-duration (15-min) bout of HIFT, both of which encompass selfselected rest periods and a variance of resistance-based exercise. The duration and modality of the examined bouts appeared to have little influence over the ANS response, which presents a viable option for populations that are less trained in technical, resistance-based movements (i.e., power clean and jerk) and who seek to induce equivalent ANS training stimulus.

Several mechanisms during the course of exercise assist in the regulation of the ANS, such as changes in blood pressure, metabolites, and muscle afferents (Mitchell, Kaufman, & Iwamoto, 1983). To this point, exercises that utilize greater muscle recruitment create a greater demand on the ANS (Mitchell et al., 1983), which is also known as the pressor reflex. Previously, we made note that both HIFT modalities involved full-body muscle recruitment (SHORT: Clean & Jerk, LONG: Row, Kettlebell swings, and Dumbbell thrusters), which would have engaged this reflex, providing the rationale for the similar ANS outcomes observed. In addition to the exercise modality, load becomes an important variable to consider when evaluating muscle recruitment, in that higher loads equal greater recruitment (i.e., size principle) (Enoka & Stuart, 1984). Both HIFT bouts exhibited some variation of load onto the participants. While total load was not calculated, it is clear that load per repetition (SHORT: 61.4 kg vs. LONG: 16 kg, 13.6 kg) was vastly different. This difference in load may have created a compounding effect on ANS markers (HRV and catecholamines), which could have resulted in the net similarity in post-exercise ANS disruption. A counter point to this is that fatigue-inducing exercise can result in similar muscle recruitments independent of increasing loads (Spiering et al., 2008), and could be a mechanism related to the LONG bout of HIFT. Because it was outside of the scope of the study to explore the differences in mechanisms in ANS recovery

between differing variations of HIFT, we can only use the aforementioned mechanisms to postulate that similar muscle recruitment may have occurred between the bouts, leading to the observed findings. Further investigation into the individual mechanisms involved in ANS disruption is needed.

This study, to the best of the authors' knowledge is the first to examine the effect of HIFT variation on markers of ANS disruption and recovery. Though this was a novel attempt to explore this relationship it was not without its limitations. The sample size of the current study was relatively small and is considered a limitation, thus future studies should incorporate a larger sample size. The general make-up of HIFT makes it difficult to compare the differences in variations, which created a limitation for the comparison of the SHORT and LONG bouts examined. The objective of the SHORT bout was to complete a designated number of repetitions as fast as possible, while the LONG bout was a set 15minute circuit to complete as many repetitions as possible. It is important to note that the variance within HIFT presents challenges when controlling for duration, intensity, or modality, which makes equivocal comparisons difficult to achieve. However, future studies should investigate similar time based objective workouts in order to better understand the complexities of HIFT. In regards to analysis, future research should standardize the warm up protocols amongst the participants, which would allow for the evaluation of the ANS response prior to the HIFT trial. Additionally, an evaluation of the resting periods should be considered in future studies, in that pace and rest could ultimately influence ANS activity and recovery. Lastly, the participants of this study were well-trained individuals with previous exposure to HIFT, and because the ANS response has been shown to be population specific (Borresen & Lambert, 2008), future studies should examine general populations.

In conclusion, the results of this study show that the parasympathetic markers (i.e., InRMSSD, InHF) were significantly altered following both SHORT and LONG bouts, but were not significantly different from each other and recovered by 2-HP. Additionally, SNS markers (i.e., plasma E and NE) were significantly elevated following both bouts and returned to baseline within 1-HP, with no significant differences between the trials. Though the SHORT and LONG bouts of HIFT were sufficient enough to alter ANS activity, no R-R recordings demonstrated irregularities indicative of ectopic beats. Therefore, the variations of the prescribed HIFT bouts did not alter the duration of ANS disruption and subsequently created equivalent cardiovascular stress. With common time restraints, there is a greater appeal for shorter duration workouts that provide similar training stimulus to longer duration bouts. Therefore, the findings of this study suggest that the SHORT bout of HIFT provides a viable option for those with time restrictions in regards to these measures.

Disclosure statement

No potential conflict of interest was reported by the authors.

References

Albert, C. M., Mittleman, M. A., Chae, C. U., Lee, I.-M., Hennekens, C. H., & Manson, J. E. (2000). Triggering of sudden death from cardiac causes by vigorous exertion. *New England Journal of Medicine*, 343(19), 1355–1361.

- Bahr, R., Høstmark, A. T., Newsholme, E. A., Grønnerød, O., & Sejersted, O. M. (1991). Effect of exercise on recovery changes in plasma levels of FFA, glycerol, glucose and catecholamines. *Acta Physiologica Scandinavica*, 143(1), 105–115.
- Borresen, J., & Lambert, M. I. (2008). Autonomic control of heart rate during and after exercise: Measurements and implications for monitoring training status. Sports Medicine (Auckland, N.Z.), 38(8), 633–646.
- Børsheim, E., Knardahl, S., Høstmark, A. T., & Bahr, R. (1998). Adrenergic control of post-exercise metabolism. Acta Physiologica Scandinavica, 162(3), 313–323.
- Buchheit, M., Papelier, Y., Laursen, P. B., & Ahmaidi, S. (2007). Noninvasive assessment of cardiac parasympathetic function: Postexercise heart rate recovery or heart rate variability? *American Journal of Physiology*. *Heart and Circulatory Physiology*, 293(1), H8–H10.
- Chen, J.-L., Yeh, D.-P., Lee, J.-P., Chen, C.-Y., Huang, C.-Y., Lee, S.-D., ... Kuo, C.-H. (2011). Parasympathetic nervous activity mirrors recovery status in weightlifting performance after training. *Journal of Strength and Conditioning Research*, *25*(6), 1546–1552.
- Dill, D. B., & Costill, D. L. (1974). Calculation of percentage changes in volumes of blood, plasma, and red cells in dehydration. *Journal of Applied Physiology*, *37*(2), 247–248.
- Eijsvogels, T. M. H., Fernandez, A. B., & Thompson, P. D. (2016). Are there deleterious cardiac effects of acute and chronic endurance exercise? *Physiological Reviews*, 96(1), 99–125.
- Enoka, R. M., & Stuart, D. G. (1984). Henneman's "size principle": Current issues. *Trends in Neurosciences*, 7(7), 226–228.
- Franciosi, S., Perry, F. K. G., Roston, T. M., Armstrong, K. R., Claydon, V. E., & Sanatani, S. (2017). The role of the autonomic nervous system in arrhythmias and sudden cardiac death. *Autonomic Neuroscience*, 205, 1–11.
- Galbo, H., Christensen, N. J., & Holst, J. J. (1977). Catecholamines and pancreatic hormones during autonomic blockade in exercising man. *Acta Physiologica Scandinavica*, 101(4), 428–437.
- Garber, C. E., Blissmer, B., Deschenes, M. R., Franklin, B. A., Lamonte, M. J., & Lee, I.-M.; American College of Sports Medicine. (2011). American College of Sports Medicine position stand. Quantity and quality of exercise for developing and maintaining cardiorespiratory, musculoskeletal, and neuromotor fitness in apparently healthy adults: Guidance for prescribing exercise. *Medicine and Science in Sports and Exercise*, 43(7), 1334–1359.
- Gibala, M. J. (2007). High-intensity interval training: A time-efficient strategy for health promotion? *Current Sports Medicine Reports*, 6(4), 211–213.
- Giles, D., Draper, N., & Neil, W. (2016). Validity of the Polar V800 heart rate monitor to measure RR intervals at rest. *European Journal of Applied Physiology*, *116*(3), 563–571.
- Heffernan, K. S., Kelly, E. E., Collier, S. R., & Fernhall, B. (2006). Cardiac autonomic modulation during recovery from acute endurance versus resistance exercise. *European Journal of Cardiovascular Prevention & Rehabilitation*, 13(1), 80–86.
- Heinrich, K. M., Patel, P. M., O'Neal, J. L., & Heinrich, B. S. (2014). Highintensity compared to moderate-intensity training for exercise

initiation, enjoyment, adherence, and intentions: An intervention study. *BMC Public Health*, 14, 789.

- Kjaer, M., Christensen, N. J., Sonne, B., Richter, E. A., & Galbo, H. (1985). Effect of exercise on epinephrine turnover in trained and untrained male subjects. *Journal of Applied Physiology*, 59(4), 1061–1067.
- Kliszczewicz, B., Buresh, B., Bechke, E., & Williamson, C. (2017). Metabolic biomarkers following a short and long bout of high-intensity functional training in recreationally trained men. *Journal of Human Sport and Exercise*, 12(3), 710–718.
- Kliszczewicz, B., Esco, M. R., Quindry, J., Blessing, D., Oliver, G., Taylor, K., & Price, B. (2016). Autonomic responses to an acute bout of high-intensity body weight resistance exercise vs. treadmill running. *Journal of Strength and Conditioning Research*, 30(4), 1050–1058.
- Malik, M. (1996). Heart rate variability: Standards of measurement, physiological interpretation and clinical use. Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. *Circulation*, 93(5), 1043–1065.
- Mitchell, J. H., Kaufman, M. P., & Iwamoto, G. A. (1983). The exercise pressor reflex: Its cardiovascular effects, afferent mechanisms, and central pathways. *Annual Review of Physiology*, 45, 229–242.
- Nakamura, F. Y., Pereira, L. A., Cal Abad, C. C., Cruz, I. F., Flatt, A. A., Esco, M. R., & Loturco, I. (2017). Adequacy of the ultra-short-term HRV to assess adaptive processes in youth female basketball players. *Journal of Human Kinetics*, 56, 73–80.
- Paine, J., Uptgraft, J., & Wylie, R. (2010). CrossFit study (Special Report No. 0704–0188). Fort Leavenworth, KS: Command and General Staff College.
- Parekh, A., & Lee, C. (2005). Heart rate variability after isocaloric exercise bouts of different intensities. *Medicine and Science in Sports and Exercise*, 37(4), 599–605.
- Pichon, A. P., de Bisschop, C., Roulaud, M., Denjean, A., & Papelier, Y. (2004). Spectral analysis of heart rate variability during exercise in trained subjects. *Medicine and Science in Sports and Exercise*, 36(10), 1702–1708.
- Quintana, D. S. (2017). Statistical considerations for reporting and planning heart rate variability case-control studies. *Psychophysiology*, 54 (3), 344–349.
- Seiler, S., Haugen, O., & Kuffel, E. (2007). Autonomic recovery after exercise in trained athletes: Intensity and duration effects. *Medicine and Science in Sports and Exercise*, 39(8), 1366–1373.
- Spiering, B. A., Kraemer, W. J., Anderson, J. M., Armstrong, L. E., Nindl, B. C., Volek, J. S., & Maresh, C. M. (2008). Resistance exercise biology. *Sports Medicine*, 38(7), 527–540.
- Stanley, J., Peake, J. M., & Buchheit, M. (2013). Cardiac parasympathetic reactivation following exercise: Implications for training prescription. *Sports Medicine*, 43(12), 1259–1277.
- Tarvainen, M. P., Niskanen, J.-P., Lipponen, J. A., Ranta-Aho, P. O., & Karjalainen, P. A. (2014). Kubios HRV – Heart rate variability analysis software. *Computer Methods and Programs in Biomedicine*, 113(1), 210–220.