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Abstract

Purpose: Examine the autonomic cardiovascular modulation in well-trained masters (n = 9, age = 55.6 ± 5.0 years) and young (n = 8, age = 25.9 ± 3.0 years) cyclists following high-intensity interval training (HIT).

Methods: Participants completed a HIT protocol consisting of 6 x 30 sec at 175% of peak power output, with 4.5-minutes rest between efforts. Immediately following HIT, heart rate (HR) and R-R intervals were monitored for of 30 minutes during passive supine recovery. Autonomic modulation was examined by: i) HR recovery in the first 60 seconds of recovery (HRR_{60}); ii) the time constant of the 30 minute HR recovery curve (HRRτ); iii) the time course of the root mean square for successive 30 second R-R interval ($RMSSD_{30}$); and iv) time and frequency domain analysis of subsequent 5 minute R-R interval segments.

Results: No significant between-group differences were observed for HRR_{60} (P = 0.096) or HHRτ (P = 0.617). However, a significant interaction effect was found for $RMSSD_{30}$ (P = 0.021), with the masters cyclists showing higher $RMSSD_{30}$ values following HIT. Similar results were observed in the time and frequency domain analyses with significant interaction effects found for the natural logarithm of the RMSSD (P = 0.008), normalised LF power (P = 0.016) and natural logarithm of the HF band (P = 0.012).

Conclusion: The masters cyclists demonstrated greater post-exercise parasympathetic reactivation when compared to young cyclists following HIT.

Keywords: age; parasympathetic reactivation; cycling; recovery

Introduction

Heart rate (HR) indices of recovery following exercise such as heart rate recovery (HRR), and heart rate variability (HRV) have historically been used as non-invasive measures of autonomic modulation [4-5]. Specifically, autonomic modulation during recovery is characterised by a withdrawal of sympathetic activity and parasympathetic reactivation, which results in a decreasing heart rate [4]. Moreover, parasympathetic activity has previously been reported to play a cardio-protective role through enhanced cardiac electrical stability [2]. Therefore, a delay in parasympathetic reactivation monitored via the HRR and HRV responses during recovery may indicate an autonomic imbalance and has been suggested to be a strong predictor of all-cause mortality [15] and coronary artery disease [13], as well as a suggesting a delay in physical recovery following exercise in athletes [18]. Heart rate recovery and HRV responses to exercise are known to be negatively influenced by age [33], low physical fitness [30] and high exercise intensity [22]. Despite this, physical activity and training in older adults has been shown to improve resting autonomic modulation through increased parasympathetic responsiveness [26, 31], which suggests that physical training into older age can have a positive influence on these autonomic functions.

Masters athletes are defined as older adults who have continued participation in structured physical activity and competitive sport into older age [20]. As a result, masters athletes have demonstrated the ability to attenuate declines in some physical and physiological characteristics into older age [3]. Consequently, there has been increasing research interest in masters athletes as their physically active lifestyle has been suggested to offset the influence of increasing physical inactivity on physical and physiological systems into older age. From a research perspective, comparison of masters athletes and performance-matched younger cohorts allows for further insight into the specific influence of the ageing process. Specifically, masters athletes have demonstrated a maintenance in HRR values following

maximal exercise when compared to similarly trained younger cohorts [8] and improved resting HRV parameters when compared to sedentary age-matched cohorts [31]. These results suggest that maintaining systematic training may preserve cardiovascular autonomic modulation into older age. However, relatively little remains known about the autonomic modulation during recovery immediately following high-intensity interval exercise (HIT).

Regardless of age, it is common for competitive cyclists to compete several times over one day and utilise training practices such as HIT to simulate competition stress and promote desired physiological adaptations [25]. Indeed, the recovery of autonomic modulation to resting values following exercise has been shown to be dependent on exercise intensity [23], and parasympathetic reactivation has been shown to be suppressed in young cohorts immediately following sprint interval cycle when compared to baseline values [27] and sprint interval running exercise when compared to lower intensity exercise protocols [6, 17]. Further, given that parasympathetic reactivation following exercise has also been shown to be influenced by age [33], an investigation into autonomic modulation following HIT in masters and young cyclists is warranted to extend current research examining the influence that physical training can have on preserving autonomic modulation following acute exercise stress into older age. Such knowledge may give insight into possible differences in recovery rates that could not only influence subsequent performance following HIT in masters athletes but also assess possible cardiovascular risks for masters athletes performing HIT.

Therefore, the aim of this study was to examine the autonomic cardiovascular modulation by comparing HR and HRV indices following a HIT protocol in well-trained masters and young cyclists.

Methods

Participants

Nine masters and eight young cyclists were recruited from local cycling and triathlon clubs to participate in the study. The participant's demographic data are presented in Table 1. To be eligible for the study all participants were required to be free from injury and medication that may have affected their ability to perform exercise or modify cardiovascular function and must have been involved in competitive cycling over the past two years. Prior to inclusion all participants were informed about the study including potential risks and benefits and were required to give written consent. Participants were matched on training practices (hours per week) and performance [maximal oxygen consumption (VO_{2max}) and peak power output (PPO)]. This study was given ethical clearance by the Central Queensland University Human Ethics Research Panel in accordance with the Helsinki declaration.

INSERT TABLE 1 AROUND HERE

Study Overview

This study was completed over two sessions separated by at least 48 hours. The first session included familiarisation and preliminary testing. The second session involved the collection of resting measures (10 minutes), the completion of a HIT protocol (detailed below), and the immediate recovery period (30 minutes). All exercise testing was conducted using an electromagnetically braked cycle ergometer (Velotron, Racermate; Seattle, USA) and blood lactate was collected via standardised capillary protocols (Accutrend Plus, Roche Diagnostics; Mennheim, Germany). All resting and recovery HR and HRV measures were collected at a frequency of 1000 Hz with the participant laying supine in a quiet dark room in accordance with previous investigations [23, 27]. Measures where obtained using a Polar RS800cx (Polar Electro; Kempele, Finland) which has previously been reported to be a valid and reliable tool to measure HRV [32]. Participants were asked to not perform strenuous exercise over the 48 hours preceding exercise testing and to follow their usual dietary intake.

Preliminary Testing

Preliminary testing consisted of assessing anthropometric measures and a maximal graded exercise test (GXT). The GXT commenced after a standard warm up of 6 minutes at 100 Watts (W). The GXT commenced at 150 W and workload increased 50 W every 3 minutes. VO_{2max} was deemed as attained when the participants reached volitional exhaustion. Expired gas was continuously analysed throughout the GXT with an indirect calorimetry system (TrueOne 2400, Parvo Medics, Inc.; Sandy, USA) which was calibrated according to the manufacturer's instructions prior to each test. PPO (W) was calculated using the following formula [10]:

$PPO = W_{(final)} + (t/180*50)$

Where,

PPO = maximal aerobic power; $W_{(final)}$ = Workload (W) of final completed stage; t = duration of the final workload completed (s)

High-intensity Interval Exercise Protocol

Once resting HR, HRV and lactate measures were collected each participant performed a standardised warm-up before the commencement of the HIT protocol that consisted of 6 x 30 second exercise bouts at an intensity of 175% of previously calculated PPO (Table 1). Each exercise bout was interspersed with 4.5 minutes of rest in which participants were allowed two minutes of active recovery at a self-selected cadence with a resistance of 50 W at a standardised time. The remainder of the between bout recovery was performed passively seated on the cycle ergometer. Upon completion of the HIT, participants stepped off the cycle ergometer and immediately laid (< 5 s) supine on a massage table adjacent to the ergometer where a lactate measure was immediately taken. Participants underwent the 30 minutes of

passive recovery in a quiet and darkened laboratory where HR and HRV were monitored. Respiratory rate was not controlled during the recovery period due to the high-intensity nature of the exercise protocol. The authors postulated that standardising the respiratory rate may have placed participants under greater stress and influenced the return of HR values to baseline during recovery [6].

Post-exercise Heart Rate Recovery

The HR (bpm) and R-R interval data (time period between successive heart beats [ms]) related to the HRV measures were extracted to a personal computer using the *Polar Pro Trainer 5* software (Polar Electro; Kempele, Finland). HRR was analysed in two ways, both of which are considered to be representative of parasympathetic reactivation following exercise [5]. Firstly, by calculating the absolute difference between end of exercise HR (HRpeak) and HR 60 seconds into recovery (HRR_{60}) [5]. Secondly, by applying a mono-exponential decay curve using the non-linear curve fitting function in *Origin Pro 2015* software (Origin Lab; Northampton, USA) to 30 minutes of HRR values [5, 17]. This method calculated the time constant of the HRR curve using an orthogonal distance regression algorithm using the following equation:

$$Y(t) = Y_{(FR)} + Ae^{-(t - TD)/\tau}$$

Where,

where Y(t) represents HR at any given time, $Y_{(FR)}$ is the final HR recovery value, A is the amplitude of the HR recovery curve, t is the time (s), TD is the time delay of exponential component, and τ is the time constant of the HR recovery curve.

The goodness of the curve fit was assessed by the sum of squares due to error (SSE) and the R-squared (R^2) value. An SSE equalling zero represents no variation or random error between

measured values and modelled values and an R^2 equalling one represents perfect curve modelling [16]. To reduce the SSE of the resultant time constant (τ) and time delay (TD), the initial parameters for amplitude (amplitude = final exercise HR – final recovery HR) and final recovery HR value of the curves were fixed using recorded data.

Post-exercise Heart Rate Variability

Data taken from the *Polar Pro Trainer 5* software were converted to a text file and analysed with *HRV Analysis Software v2.2* (Biosignal Laboratory, University of Kuopio, Finland) [28]. Occasional ectopic beats were examined and erratic data were identified and replaced with interpolated adjacent R-R intervals using a medium artefact correction (± 0.25 seconds of local mean). Due to methodological limitations of performing conventional HRV analysis with rapidly fluctuating autonomic conditions such as directly after exercise [29], during the first ten minutes of recovery both the R-R interval and the root mean square of successive differences of R-R intervals (RMSSD) were obtained over subsequent 30 second non-overlapping segments ($RMSSD_{30}$). $RMSSD_{30}$ was calculated as a time varying vagal parameter to monitor parasympathetic reactivation immediately following exercise [9].

Following the initial five minutes of recovery, subsequent 5 minute R-R interval segments were analysed in both the time and frequency domains until cessation of the 30 minute recovery period. The mean R-R interval and the natural logarithm of the RMSSD (LnRMSSD) for each 5 minute segment were obtained from time domain analysis. Power frequency analysis of the 5 minute segments were performed using Fast-Fourier Transform after data were de-trended and resampled at 5 Hz to calculate the low frequency (LF) band (0.04 - 0.15 Hz) and high frequency (HF) band (0.15 – 0.4 Hz). The natural logarithm of the LF (LnLF) and HF (LnHF) power as well as the normalised LF power (LFnu = LF [ms^2]/(total power [ms^2] – VLF [ms^2]), normalised HF power (HFnu = HF [ms^2]/(total power [ms^2] – VLF [ms^2])

were derived for each 5 minute segment in the frequency domain. Low frequency measures have recently been shown to be a measure of baroreflex function [19] and HF measures are commonly accepted measures of parasympathetic activity [29].

Statistical Analyses

All data are presented as mean ± standard deviations (SD) unless stated otherwise. The distribution of all data was tested with the Shapiro-Wilk normality test. When data were skewed, data were transformed using the natural logarithm. Natural logarithm data were taken for RMSSD, LF and HF parameters. Independent t-tests were used to compare between-group differences for demographic data, HR derived data, and lactate values. For $RMSSD_{30}$ a 2 x 21 repeated measures ANOVA, and for short term HRV parameters 2 x 6 repeated measures ANOVA were utilised to examine the effects of age and time. When statistical significance was identified, independent t-tests were used to further delineate between group differences. All data were assessed using Mauchly's test for sphericity and whenever a test was violated the Greenhouse-Geisser test was used. All statistical analyses were conducted using IBM SPSS Statistics (Version 22, IBM Corporation; New York, USA) and statistical significance was accepted at $P < 0.05$ level.

Results

No significant differences between groups were found for demographic data or HIT workload ($P > 0.05$), except for age ($P < 0.001$). All HRR and lactate data are presented in Table 2. The sum of the squares due to error was low for masters (2.94 ± 0.80sec) and young cyclists (3.19 ± 0.73sec) and the R-square values were all > 0.98 suggesting good curve fits. No significant between-group differences were observed for HRR or lactate values except for HRpeak ($P = 0.004$) and HRRamp ($P = 0.014$).

INSERT TABLE 2 AROUND HERE

The time courses of the R-R intervals and $RMSSD_{30}$ for the first 10 minutes of recovery are presented in Figures 1 & 2. A significant interaction effect was found for the $RMSSD_{30}$ ($F_{20, 300} = 1.792$, P = 0.021) time course with the masters group exhibiting higher $RMSSD_{30}$ values compared to the young group. The between-group differences at specific time points for the time course of the $RMSSD_{30}$ are represented in Figure 2.

INSERT FIGURES 1 & 2 AROUND HERE

Between-group HRV indices for the 30 minute recovery time period are presented in Table 3. Significant interaction effects were found for LnRSMMD (F = 4.595, P = 0.008), LnHF (F = 4.429, P = 0.012) and LFnu (F = 3.980, P = 0.016). No significant interaction effects were found for R-R intervals (P = 0.693), LnLF (P = 0.472) and HFnu (P = 0.136), however, main effects for time were found (F = 151.10, P < 0.0001; F = 43.85, P < 0.0001, F = 9.915, P = 0.0001). Post hoc analyses found significant between group differences for $LnLF_{25-30}$ (0.031), $LnHF_{5-10}$ (P = 0.037), $LFnu_{5-10}$ (P = 0.015), $LFnu_{10-15}$ (P = 0.033) $LFnu_{15-20}$ (P = 0.043), $HFnu_{5-10}$ (P = 0.039) and $HFnu_{10-15}$ (P = 0.032) with the masters athletes exhibiting higher HF and lower LF values

INSERT TABLE 3 AROUND HERE

Discussion

The aim of this study was to examine and compare autonomic cardiovascular modulation following a high-intensity interval training bout in well-trained masters and young cyclists. The present results suggest no significant differences in HR based indices of parasympathetic reactivation (HRR_{60} and HRRτ) between masters and young cyclists following HIT. However, significant differences were observed within the HRV indices of autonomic modulation. Specifically, the masters cyclists showed significantly higher $RMSSD_{30}$ values suggesting greater parasympathetic reactivation in the first 10 minutes of recovery. Furthermore,

significant between-group differences in HFnu and LFnu over the 30-minute recovery period also suggest a greater parasympathetic reactivation following HIT in the masters cyclists compared to the young cyclists. Therefore, the present findings suggest an altered autonomic modulation in the masters cyclists compared to young cyclists following HIT with the masters cyclists in the present study exhibiting greater parasympathetic reactivation following HIT.

As expected, the present study found significantly lower HRpeak and HRRamp in the masters cyclists which can be attributed to the age-related differences in maximal heart rate [14]. However, despite lower HRpeak in the masters cyclists no significant between-group differences for HRR_{60} and HRRτ where observed following HIT. Similar dissociations between HRpeak and HRR parameters were reported by Nakamura et al. [17] who found no difference in HRR_{60} and HRRτ despite differences in HRpeak induced by different exercise protocols in national level handball players (n = 13; 23.5 ± 4.1 years). Comparable HRR_{60} and HRRτ between young (24 ± 2 years) and older (51 ± 2 years) trained participants were also reported by Darr et al. [8] who performed linear regression analysis on the fast and slow phases of HRR curve. The authors reported no significant differences between groups in the HRR slopes [8]. Despite the different methodologies utilised, the results of the seminal Darr et al. [8] study support those of the present study suggesting that parasympathetic reactivation as measured via HRR after HIT appear to be maintained in athletes who continue systematic training into older age.

Although no significant between-group differences were found for HRR, significant differences were observed in a number of HRV indices. These results support previous research suggesting differences between HRR and HRV measures of parasympathetic activity in trained athletes following exercise [11]. The significantly lower recovery $RSMMD_{30}$ values in the young cyclists from 90 seconds to 10 minutes during recovery (expect the 450

second time-point) suggests a delayed parasympathetic reactivation following the HIT protocol (Figure 2). The lower parasympathetic activity in the young cyclists compared to the masters continued into the short-term HRV measures with the young cyclists showing significantly lower $HFnu_{5-10}$ and $HFnu_{10-15}$ during recovery. It should be noted that the masters athletes in the current study demonstrated high variability in their HRV responses to HIT which suggests that the recovery of autonomic modulation as measured by HRV following HIT may be highly individualised in master athletes and may be representative of this cohort only.

Unfortunately, to the authors' knowledge, no previous studies have compared the autonomic modulation of masters and young athletes following exercise. Leti and Bricout [12] noted that the sympathetic modulation recorded via nocturnal HRV indices in the 10 senior runners (51 ± 5 years) they tested over 12 weeks training were lower than other values reported in current literature, regardless of age or training status. Taken together with work by Ueno & Moritani [31] who reported greater parasympathetic activity and baroreflex function in aged endurance runners (66.9 ± 0.8 years) compared to their sedentary age-matched counterparts (65.4 ± 1.0 years), the present study supports current research suggesting that maintaining physical training into older age has a beneficial influence on the autonomic control of the cardiovascular system.

Although the ability of physical activity and structured exercise to improve HRV indices of parasympathetic activity in older adults is well documented [1], the greater parasympathetic reactivation in the masters cyclists compared to the young cyclists in the current study was unexpected. A slowed parasympathetic reactivation has been suggested to be significantly related to the contribution of the anaerobic energy system due to the associated plasma metabolites [6]. However, the standardised relative workloads (175% PPO) administered in the tests used in the present study resulted in similar lactate concentrations (masters: 14.5 ±

5.3 mM; young: 13.9 ± 4.9 mM) between groups at the cessation of exercise. Therefore, it is unlikely that the delayed parasympathetic reactivation of the young cyclists was due to an increased anaerobic contribution at the end of exercise. The masters cyclists did, however, exhibit a significantly lower HRpeak at the end of exercise which is thought to be due to an age-related decrease in sensitivity to catecholamines [21]. Theoretically, a decreased sensitivity to catecholamines may have led to a decreased cardiovascular sympathetic drive during the HIT in the masters cyclists which could have could have led to a seemingly stronger post-exercise parasympathetic response. This suggests that perhaps a greater sympathetic drive during HIT in the young cyclists may have led to a blunted parasympathetic reactivation during recovery potentially explaining the between-group differences.

A further possible explanation for the between-group differences in the HRV response could relate to the baroreflex response and blood pressure regulation following the HIT. The authors acknowledge that a limitation of the present study was that the blood pressure response was not measured during recovery as blood pressure changes can influence HR modulation [7] and has been suggested to be effected by age [24]. Additionally, previous studies have reported that blood pressure responses mainly occur in the first 15 minutes following HIT exercise [27], which is when the majority of the between-group differences in autonomic function in the present study were observed (Figure 2 & Table 3). Considering that the present study found significant differences in LFnu5-10, LFnu10-15 and LFnu15-20, which suggests differences in the baroreflex function between masters and young cyclists, blood pressure regulation immediately following exercise may have attributed to the different autonomic responses between groups and warrants further investigation. Additionally, another limitation of the current investigation is that respiration rate was not controlled or monitored during recovery. Therefore, between-group differences in respiration rate could

have influenced the autonomic cardiovascular regulation during recovery following the HIT protocol [4]. Future research should look to standardise and monitor respiration rate as well as monitor blood pressure following exercise sessions to gain further insights into age-related differences in the autonomic response of masters and young athletes.

In summary, the present study is the first to examine autonomic cardiovascular modulation in well trained masters and young cyclists. This study provides novel insight into the effect of age and continued sports participation on autonomic modulation following exercise, with masters cyclists demonstrating a greater parasympathetic response following HIT. Practically, these results suggest that the HIT protocol utilised in the current study did not induce negative influences on autonomic cardiovascular modulation in the masters cyclists during recovery. This finding suggests that HIT training is a safe training modality for masters athletes, however, without blood pressure responses being measured it remains difficult to draw comprehensive conclusions related to overall autonomic recovery in masters athletes. Additionally, the current findings related to autonomic cardiovascular modulation suggest that the duration for autonomic modulation to return to resting values in masters athletes may be similar to young athletes and that masters athletes can follow similar acute recovery procedures.

Conclusion

The current study is the first to compare a number of HR and HRV derived indices of autonomic cardiovascular modulation in training- and performance-matched masters and young cyclists. Although no between-group differences were found in HR derived measures of parasympathetic reactivation, HRV indices demonstrated a stronger parasympathetic reactivation in masters cyclists compared to young cyclists during recovery from HIT. These

findings suggest that continued physical training into older age has regulatory effects on autonomic modulation following exercise.

Conflicts of Interest

The authors report no conflicts of interest.

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Table and Figure Legend

Table 1 Demographic and workload data for masters and young cyclists

Table 2 Mean ± standard deviation of the baseline and recovery heart rate and lactate measures throughout the high-intensity interval training protocol for masters (*n* = 9) and young (*n* = 8) cyclists

Table 3 Mean ± standard deviation of heart rate variability indexes for baseline and the 30 minute recovery period broken into 5 minute segments following high-intensity interval training in masters (*n* = 9) and young (*n* = 8) cyclists

Figure 1 Mean ± standard deviation of the R-R intervals during the first 10 minutes of recovery for masters (*n* = 9) and young (*n* = 8) cyclists following high-intensity interval training. The R-R interval was calculated as the mean R-R intervals over the 30s segment.

Figure 2 Mean ± standard deviation of the root mean square of successive difference of the R-R intervals measured on successive 30-s segments ($RMSSD_{30s}$) during the first 10 min recovery period for masters (*n* = 9) and young (*n* = 8) cyclists following high-intensity interval training. *Significant group difference between masters and young cyclists ($P < 0.05$)

Table

Table 1 Demographic and workload data for masters and young cyclists

	Age (years)	**Height (cm)**	**Body Mass (kg)**	**VO_2max ($mL \cdot kg \cdot min^{-1}$)**	**Peak Power Output (W)**	**Distance per Week (km/wk)**	**HIT Workload (W)**
Masters (*n* = 9)	55.9 ± 5.2*	178.5 ± 8.7	82.0 ± 8.9	54.2 ± 10.3	348.0 ± 29.9	214.4 ± 58.1	609.0 ± 52.3
Young (*n* = 8)	25.9 ± 3.0	177.8 ± 5.8	79.1 ± 4.93	62.0 ± 9.8	364.2 ± 37.0	213.1 ± 128.7	673.3 ± 64.7

All values presented in means ± SD. *Significant group difference between masters and young cyclists ($P < 0.05$). HIT = High-intensity Interval Training.

Table

Table 2 Mean ± standard deviation of the baseline and recovery heart rate and lactate measures throughout the high-intensity interval training protocol for masters (n = 9) and young (n = 8) cyclists

	Masters	**Young**	**P-value**
HRbase (bpm)	59.8 ± 7.3	65.1 ± 10.7	0.258
HRpeak (bpm)	145.0 ± 18.2*	168.9 ± 6.6	0.004
HRR_{60} (bpm)	28.0 ± 9.8	35.5 ± 7.5	0.096
HRRτ (sec)	99.57 ± 40.65	92.05 ± 15.57	0.617
HRRamp (bpm)	69.00 ± 15.28*	86.38 ± 9.98	0.014
[La]rest (mM)	1.9 ± 0.6	2.1 ± 0.9	0.441
[La]peak (mM)	14.5 ± 5.3	13.9 ± 4.9	0.807

All values presented in means ± SD. HR = heart rate, HRR_{60} = heart rate recovery in 60 seconds, HRR_{τ} = time constant of heart rate recovery curve, HRR_{amp} = amplitude of the heart rate recovery curve, [La] = Concentration of blood lactate.
*Significant group difference between masters and young cyclists ($P < 0.05$).

Table

Table 3 Mean ± standard deviation of heart rate variability indexes for baseline and the 30 minute recovery period broken into 5 minute segments following high-intensity interval training in masters (n = 9) and young (n = 8) cyclists

	Baseline		**5-10 Minutes**		**10-15 Minutes**		**15-20 Minutes**		**20-25 Minutes**		**25-30 Minutes**	
	Masters	Young	Masters	Young	Masters	Young	Masters	Young	Masters	Young	Masters	Young
RR (ms)	1022 ± 25	950 ± 128	675 ± 92	604 ± 39	700 ± 113	611 ± 28	724 ± 127	635 ± 38	735 ± 125	639 ± 39	775 ± 144	684 ± 48
SNDD (ms)	50.6 ± 12.9	79.6 ± 32.2	15.0 ± 6.6	14.5 ± 4.8	17.7 ± 10.8	22.3 ± 11.4	21.7 ± 14.5	18.5 ± 8.6	33.1 ± 15.0	39.4 ± 8.1	31.1 ± 16.5	42.8 ± 15.8
LnRMSSD (ms)	3.4 ± 0.5	4.0 ± 0.8	2.3 ± 0.6	1.8 ± 0.6	2.1 ± 0.6	1.7 ± 0.4	2.3 ± 0.6	1.9 ± 0.5	2.6 ± 0.5	2.5 ± 0.2	2.4 ± 0.5	2.8 ± 0.5
LnLF	6.1 ± 1.5	7.3 ± 0.8	3.3 ± 1.5	3.8 ± 1.0	3.8 ± 1.4	4.3 ± 0.8	4.2 ± 1.4	4.5 ± 1.0	5.3 ± 1.0	5.8 ± 0.6	5.4 ± 0.9*	6.4 ± 0.8
LnHF	5.9 ± 1.2	7.0 ± 1.7	3.9 ± 0.8*	2.7 ± 1.2	3.5 ± 1.1*	2.5 ± 0.8	3.6 ± 1.0	2.8 ± 1.0	4.1 ± 1.1	3.9 ± 1.0	4.1 ± 1.0	4.4 ± 0.6
LFnu	54.2 ± 24.1	54.1 ± 23.0	40.2 ± 27.6*	69.9 ± 26.4	56.2 ± 31.0*	83.6 ± 11.4	62.3 ± 26.7*	82.7 ± 8.6	73.0 ± 20.9	88.5 ± 6.2	76.2 ± 15.0	87.5 ± 5.2
HFnu	46.0 ± 24.4	45.9 ± 23.0	59.8 ± 27.6*	30.1 ± 26.4	43.8 ± 31.0*	16.4 ± 11.4	37.8 ± 26.7	17.3 ± 8.6	27.0 ± 20.9	11.5 ± 6.1	23.8 ± 4.2	12.5 ± 5.2

All values are presented in means and ± SD. RR = R-R interval, SNDD = standard deviation of normal R-R intervals, LnRMSSD = natural logarithm of the root mean square of successive difference of R-R intervals, LFnu = normalised low frequency power of R-R intervals, HFnu = normalised high frequency power of R-R intervals. *Significant between-group difference between masters and young cyclists ($P < 0.05$).

Figure

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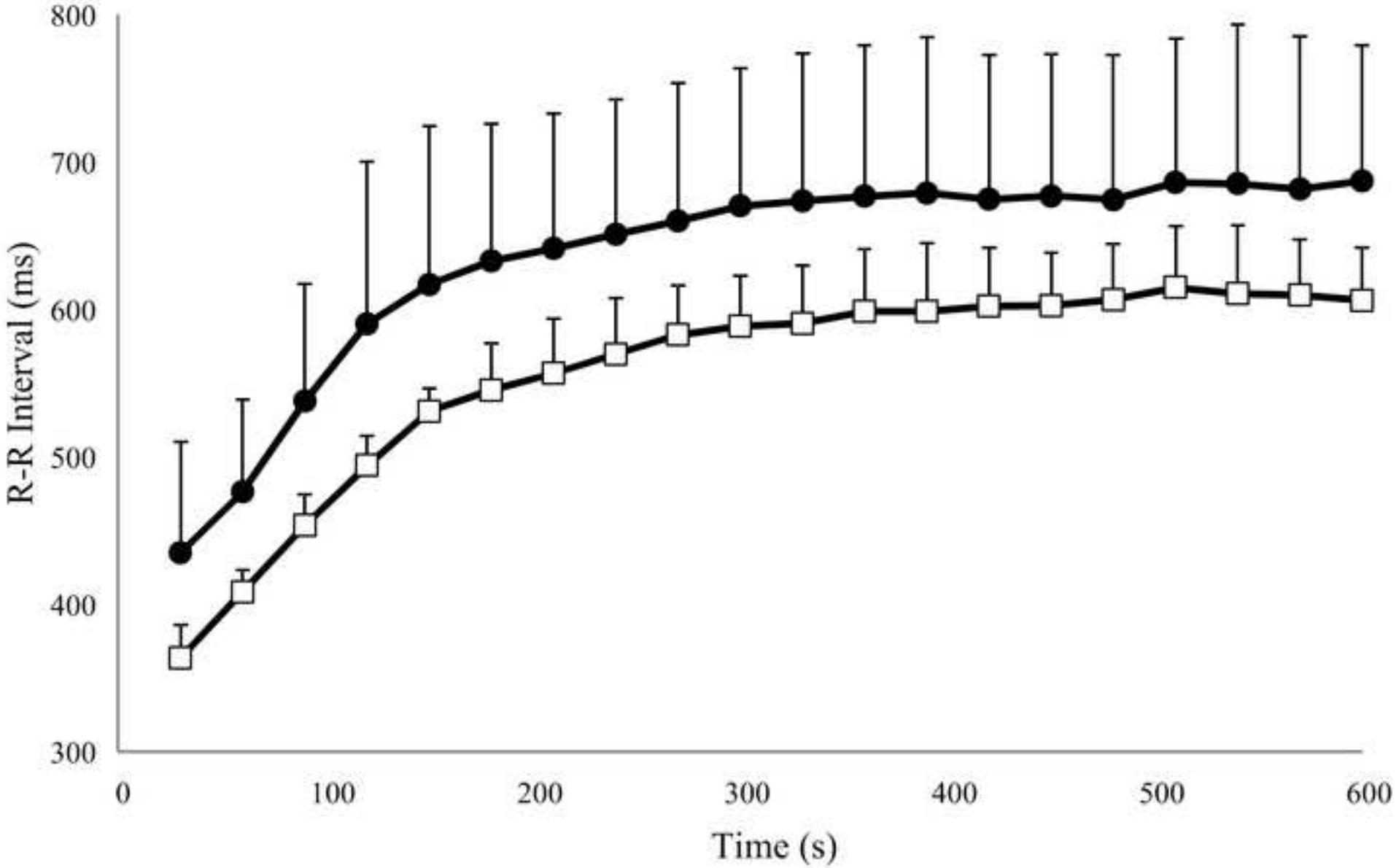

Figure

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