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**Impact of high-intensity interval and moderate-intensity continuous exercise on heart rate variability and cardiac troponin.**

**Impact of high-intensity interval and moderate-intensity continuous exercise on heart rate variability and cardiac troponin**

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**ABSTRACT**

BACKGROUND: It remains uncertain whether exercise modality (high-intensity interval [HIE]; moderate-intensity continuous [MCE]) mediates exercise-induced changes in markers of pro-arrhythmogenic state and/or cardiac damage. This study examines heart rate variability (HRV) and cardiac troponin T (cTnT) kinetic responses to HIE and MCE.

METHODS: Fourteen sedentary, overweight/obese females completed two trials including HIE (2-min running at 90%$VO_{2max}$ followed by 2-min running at 50%$VO_{2max}$, repeated for 60min) and MCE (70%$VO_{2max}$ steady-state running for 60min) in a randomized, counterbalanced fashion. Supine HRV was evaluated as root mean square of successive differences (RMSSD), normalized low-frequency (LF) and high-frequency (HF) spectral power, as well as the LF/HF ratio before (PRE), immediately (0HR), 3 (3HR) and 24 (24HR) hours after exercise. Serum cTnT was assessed using a high-sensitivity assay at the same time-points and the values were corrected for plasma volume changes.

RESULTS: Exercise temporarily altered all HRV indices (i.e. RMSSD and HF decreased; LF and LF/HF ratio increased at 0HR, all $p<0.05$) but a rebound increase of RMSSD was observed at 24HR, and the kinetic responses of HRV were similar between exercise modalities. The cTnT was significantly elevated ($p<0.05$) after exercise at 3HR (by 688%) and 24HR (by 374%) with no between-modality differences. There was no significant correlation between delta change in cTnT and HRV metrics.

CONCLUSIONS: Exercise modality (workload-equivalent HIE vs MCE) did not mediate exercise-induced alteration in autonomic activity and cTnT elevation, and it seems these are largely separate exercise-induced phenomena.

**Keywords**: Exercise; Heart, Autonomic nervous system, Troponin T

## Introduction

High-intensity interval exercise (HIE) typically involves repeated bouts of relatively intense exercise interspersed by short periods of recovery and is growing in popularity as evidenced by the fact that HIE training was recently named the top fitness trend worldwide in an annual survey by the American College of Sports Medicine.[1] With increasing popularity has come some degree of concern about the risk of adverse cardiac events due to the greater cardiovascular strain associated with HIE compared to moderate-intensity continuous exercise (MCE).[2] Acute bouts of higher-intensity exercise may transiently potentiate a pro-arrhythmogenic state and increase the risk of acute coronary syndrome, particularly among at-risk individuals.[3] It is, therefore, important to determine the impact of HIE and MCE on potential markers of a pro-arrhythmogenic state and biomarkers of cardiac damage associated with acute coronary syndrome.

Heart rate variability (HRV) analysis provides non-invasive markers of cardiac autonomic activity and reduced HRV has been associated with an unfavourable electrophysiological milieu, such as increasing the risk for a malignant arrhythmia.[4] Acute exercise transiently depresses HRV and higher-intensity exercise attenuates post-exercise HRV recovery [5]. In addition, exercise has also been shown to result in elevated levels of circulating cardiac troponin (cTn, cTnT and/or cTnI),[6,7] which is a highly specific biomarker of cardiomyocyte insult that is used in the evaluation of acute coronary syndromes.[8] An association between exercise-related changes in HRV and cTnT was reported by Aagaard *et al*.[9] These authors noted that, following cross-country running race, cTnT levels were higher in trained subjects with a larger decline in HRV post-exercise and a delayed return to baseline HRV.[9] Whilst ecologically valid, this approach based on field-based competition has made it difficult to control many factors such as the duration and intensity of effort and training status of subjects and environmental conditions. Therefore, the nature and consistency of any association between HRV and cTnT require further study in a lab-based setting.

To date, few studies have compared post-exercise HRV recovery and/or cTn elevation between acute bouts of HIE and MCE.[5-7]. Perkins *et al*.[10] and Mourot *et al*.[11] reported HRV recovery was slower after HIE than MCE, but it is still unclear whether intensity mediated any difference. In addition, data for post-exercise cTn kinetics is limited and equivocal. When matched for total work, two recent studies from our group noted that HIE and MCE induced similar elevations in cTn after acute exercise,[12,13] but Ranjbar *et al*.[14] showed that MCE resulted

in a higher cTnT elevation than HIE. Notably, the three studies did not assess cTn data at 24h post-exercise, which may be important, considering that an extended timescale for the evaluation of cTn kinetics is a fundamental component of clinical acute coronary syndrome assessment.[8] To the best of our knowledge, no study has determined the association between HRV indices and cTn kinetics post-exercise when comparing HIE and MIE. An integrated understanding of the response of HRV and cTn data after MCE and HIE could inform the debate about the risk/benefit assessment of these exercise models as well as add new evidence about the potential clinical significance of exercise-induced cTn elevation. This is particularly important for sedentary obese individuals who are considered to be at high risk of exercise-induced adverse cardiovascular events.[15] Indeed, this group has delayed post-exercise vagal reactivation [16] and higher resting cTn concentrations.[17] It is also worth noting that HIE training is increasingly prescribed for losing weight.[18]

Consequently, the present study characterized the change in HRV indices and cTn kinetics up to 24 h after acute bouts of MCE and HIE, matched for the total workload, in sedentary, overweight/obese young females. We hypothesized that exercise modality (matched for workload) will not mediate exercise-induced changes in HRV and cTnT. Still, there is an association(s) between exercise-related changes in HRV and cTnT.

## Materials and methods

### Participants

Sixty female volunteers were recruited through local advertisements to participate in the study. In total, 18 females were eligible according to the following inclusion criteria: (1) age range of 18–23 years; (2) body mass index (BMI) ≥ 23 kg $m^{-2}$, which is the overweight cut-off for Asian adults;[19] (3) eumenorrheic for the past six months; (4) no regular physical activities, based on International Physical Activity Questionnaire in the last 6 months;[20] (5) no history of smoking; and (6) no history of hormonal, orthopaedic, or cardiovascular diseases, diabetes, hyperlipidaemia, hypertension or polycystic ovary syndrome; and (7) no current use of prescribed medication (including contraceptive pills in the last 6 months), as assessed by a medical screen prior to the study. After receiving a thorough briefing, all participants gave their written informed consent to participate. The experiment was approved by the local ethics committee and was conducted in accordance with the Helsinki declaration 1975 (revised 2013).

Experimental design and procedures

On the first and second visits to the laboratory, separated by three days, MCE (70% of age-predicted $HR_{max}$, 60 min) and HIE (80/40% of age-predicted $HR_{max}$, 2/2min, repeated for 60 min) trials were performed to acclimatize the participants to corresponding running sessions. We excluded four subjects who could not complete a simulated trial, which left a total of 14 participants (mean ± SD: aged 20.8 ± 1.1 years; body mass 72.1 ± 4.9 kg; height 167.2 ± 4.9 cm; BMI 25.7 ± 1.6 [range: 23.2−28.7]; and $VO_{2max}$ 34.3 ± 3.0 $ml.kg^{-1}.min^{-1}$). At least five days later, the assessment of maximal oxygen uptake ($VO_{2max}$) was completed. One week after base assessments, all participants commenced experimental trials. The two trials were completed in a randomized (by lottery) crossover design of exercise modality (HIE and MCE) and separated by one week.

During each exercise session, heart rate (HR) was collected for a period of 5 min for resting HRV assessments before exercise (PRE), 5 min after (0HR), as well as at 3 h (3HR) and 24 h (24HR) after exercise. HR was also continuously recorded throughout each exercise bout. In addition, after a 5-min rest period in a seated position, venous blood samples were also drawn before exercise (PRE), immediately after (0HR), as well as at 3h (3HR) and 24h (24HR) after exercise to assess cTnT, haemoglobin (Hb) and haematocrit (Hct). The timing for the post-exercise blood cTnT samples was in accordance with serial cTn testing recommended by the latest European Society of Cardiology guidelines[8] and our previous work that demonstrated blood concentrations for cTnT peaked 3 h after exercise.[21]

One day prior to each trial, participants were provided with the same meals at 20:00. The last meal before testing was a light snack (~400 kcal) consumed at 08:00 and water was allowed ad libitum. All exercise tests started at 11:00 and were performed in an air-conditioned laboratory (20 °C and 50% relative humidity). All participants were asked to maintain their usual physical activity, diet and sleeping habits, and avoid any additional exercise and alcohol/coffee consumption throughout the study period.

Protocol and measurements

*Graded exercise test*

The protocol for the graded treadmill running test has been described previously [21]. Briefly, subsequent to a general warm-up, participants ran on a motor-driven treadmill (Pulsar,

h/p/cosmos sports and medical GmbH, Traunstein, Germany) at a gradient of 0% and with an initial speed of 4 $km.h^{-1}$. During the test, running speed was increased in increments of 0.8 $km.h^{-1}$ every minute until the participant reached volitional exhaustion. Ventilatory and metabolic data were recorded continuously using an automated online metabolic cart (MetaMax 3b; Cortex Biophysik, Leipzig, Germany). HR was recorded continuously using an HR monitor (Polar RS400, Polar Electro, Kempele, Finland) and recorded at 5-s intervals. $\dot{V}O_{2max}$ and maximum HR ($HR_{max}$) were recorded as the highest 30-s average value of $O_2$ uptake ($V\dot{}O_2$) and HR, respectively, during the test. Following the graded exercise test, a treadmill running speed that elicited approximately 90%, 50% and 70% $\dot{V}O_{2max}$, was selected from the linear relationship of steady-state $\dot{V}O_2$ versus running speed to be used in subsequent HIE and MCE trials.

*Acute exercise*

Each exercise session consisted of 60 min of either interval (HIE) or continuous (MCE) exercise on a treadmill (Pulsar, h/p/cosmos sports and medical GmbH, Traunstein, Germany). The HIE protocol comprised 60 min running on a treadmill with intensity alternating between 90% (2 min) and 50% (2 min) of $\dot{V}O_{2max}$, resulting in a mean intensity of 70% $\dot{V}O_{2max}$ ([(90% × 2) + (50% × 2)] ÷ 4). By contrast, the MCE protocol consisted of 60 min of running on the same treadmill at 70% of $\dot{V}O_{2max}$. In each exercise session, participants completed an identical 10 min warm-up and 5-min cool-down at ~50% of $HR_{max}$. All exercise sessions were supervised. Supervisors provided verbal encouragement during the exercise bouts and ensured that the participants exercised at the intended intensity.

*Heart rate variability assessments*

Beat-to-beat HR was collected with 1 ms resolution for a period of 5 min at each designated time using an HR monitor (Polar RS800CX, Polar Electro, Kempele, Finland). This instrument has been previously validated for the accurate measurement of R-R intervals.[22] The participant was instructed to remain in a supine position, resting, awake and silent in a quiet and dimly lit room. Respiratory rate was not controlled so as not to interfere in post-exercise recovery, although they were asked to avoid irregular respiration. Spontaneous respiratory rate does not result in significantly different HRV indices compared with controlled breathing.[23]

The heartbeats recorded were transmitted to Polar Precision Performance software (Polar Electro, Kempele, Finland) to generate the R-R interval time series. Occasional ectopic

beats were examined, and erratic data were identified and replaced with interpolated adjacent R–R intervals.[24] The HRV was calculated in time and frequency using HRV software (Kubios 2.0, Kubios Oy, Finland). In the time domain, RMSSD was determined. The RMSSD index is defined as the root mean square of successive differences between adjacent normal R-R intervals in ms. For the frequency domain, fast Fourier transform analysis was used to calculate low (LF; 0.04–0.15 Hz) and high frequency (HF; 0.15–0.40 Hz) bands of the spectrum and reported in normalized units (nu), as well as the LF/HF ratio.[24] Among these parameters, the RMSSD and HF represent vagal activity, LF is regarded as reflecting predominantly sympathetic and, to a lesser extent vagal activity, and LF/HF ratio is a measure of the sympathovagal balance.[24]

*Blood sampling procedures*

At each sample time point, 5 ml of venous blood was drawn from the antecubital vein by venipuncture with the participants in a seated position. An aliquot was obtained for the determination of whole blood Hb and Hct using a Sysmex XP-100 analyser (Sysmex Corporation, Kobe, Japan). The remaining blood was allowed to clot at room temperature and then centrifuged at 3500 g for 20 min. The serum was drawn off and stored at −80 °C for later analysis of cTnT, which was measured based on electrochemiluminescence technology using a Cobas E 601 analyser (Roche Diagnostics, Penzberg, Germany). The cTnT assay is the 5$^{th}$ generation high-sensitivity immunoassay and has a lower detection limit of 3 ng.l$^{-1}$ with an upper limit of 10,000 ng.l$^{-1}$. Serum cTnT concentrations that were below the limit of detection are reported as 1.5 ng.l$^{-1}$.[21] The coefficient of variation at a mean cTnT concentration of 13.5 ng.l$^{-1}$ is 5.2%. The upper reference limit for cTnT, defined as the 99$^{th}$ percentile of healthy participants, was 14 ng.l$^{-1}$.[8] Results for cTnT were corrected for percent change in plasma volume, calculated from levels of Hb and Hct in two trials, as described elsewhere.[13]

Statistical analysis

A 2 × 4 two-way ANOVA with repeated measures was used to examine the changes in HR, RMSSD, LF, HF and LF/HF ratio across the two modalities (HIE and MIE) and four observed points (PRE, 0HR, 3HR, and 24HR). *Post-hoc* analyses, using the Newman–Keuls test[21] were performed for cases in which the main effect was significant. Group differences between HIE and MCE in mean HR and %HRmax during each exercise bout were analysed by pair t-tests.

The Kolmogorov–Smirnov test was used to evaluate the normality of the data. The non-parametric Friedman's test was used to compare cTnT across the four observed points (PRE, 0HR, 3HR, and 24HR) because of the skewed distribution of the cTnT data. Wilcoxon signed ranks tests were completed for pairwise comparisons between different time points or modalities where appropriate. The percentages of subjects with cTnT exceeding the upper reference limit of 14 $ng.l^{-1}$ at each assessment point were compared using Fisher's exact test. Correlations between delta values for log-transformed cTnT and HRV indices were determined via Pearson's product-moment bivariate correlation analysis. Statistical significance was assumed at a level of $p < 0.05$. Values are reported as mean ± SE unless otherwise indicated. Data analysis was performed using the statistical software package SPSS 20.0 (IBM Corp., Armonk, NY, USA).

Data availability

The data associated with the paper are not publicly available but are available from the corresponding author on reasonable request.

**Results**

The acute exercise data, including mean HR during exercise (HIE: 140±2 vs MCE: 138±3 $beat.min^{-1}$) and $\%HR_{max}$ during exercise (HIE: 72.9±0.9% vs MCE: 72.1±0.9%), were similar (both $p>0.05$) between trials. HRV data for the two trials are presented in Table 1. Markers of parasympathetic (RMSSD, HF and HR) and sympathetic (LF and LF/HF ratio) activity were significantly ($p<0.05$, Cohen's $d$: 0.70–3.30) reduced and increased just after exercise, respectively and returned to baseline values at 3HR. At 24HR, significantly higher RMSSD ($p<0.05$, Cohen's $d$: 0.56–1.39) and lower HR ($p<0.05$, Cohen's $d$: 0.42–1.42) values were observed, compared with PRE and 3HR. The values of all HRV indices were not significantly ($p>0.05$) different between HIE and MCE at any assessment point.

cTnT data for the two trials are presented as cohort data in Table 2 and as individual data points in Fig. 1. cTnT values increased ($p<0.05$) after the exercise bout in both trials with no between-trial differences at any time point ($p>0.05$). Peak post-exercise cTnT values occurred (61% of cases) at 3HR (Fig. 1). Although most of the cTnT values at 3HR were followed by a marked decrease, data in both trials had not returned to baseline at 24HR. The percentage of participants with maximum cTnT elevations greater than the upper reference

limit of 14 ng.l$^{-1}$ was similar between the two trials (HIE, 36% [5 of 14] vs MCE, 50% [7 of 14], $p$>0.05).

There were only small correlations (all $p$>0.05) between delta change (PRE to post-exercise peak) in cTnT and delta changes (PRE to 0HR, PRE to 3HR and PRE to 24HR) in RSMMD (r: -0.069–0.348), LF (r: -0.320–0.145), HF (r: 0.014–0.322), and LF/HF ratio (r: -0.133–0.324) when the two trials were combined.

## Discussion

*Overview of findings*

The present study provides a direct comparison of the kinetics of changes in HRV and cTnT after workload-matched HIE and MCE. Using serial observations in a randomized crossover design, we demonstrated that acute HIE or MCE induced a transient reduction in HRV and a rebound of RMSSD at 24 h after exercise, with no between-exercise trial differences. In addition, both HIE and MCE resulted in a similar elevation in cTnT that remained above baseline at 24 h. The changes in HRV and cTnT after both exercise trials were not associated temporally.

*HRV change with different exercise trials*

In the present study, immediately following acute exercise, vagal (RMSSD and HF) and sympathetic (LF and LF/HF) indices were decreased and increased, respectively, indicating parasympathetic withdrawal and sympathetic predominance following exercise.[24] These findings are consistent with published studies.[5] The period of reduced HRV at post-exercise is a vulnerable time with a higher risk of adverse cardiac events[24] and thus the magnitude and duration of disrupted autonomic balance are important regarding the safe prescription of exercise. In this regard, exercise intensity has been shown to be the key determinant of autonomic recovery, with greater and/or longer disruptions being observed at higher intensities, but which is almost entirely based on data from single-modality and continuous-based exercise.[5] In the current study, HRV changes and recovery were nearly identical following workload-matched bouts of HIE and MCE. This is a novel finding that contradicts the only two previous studies[10,11] of a slower return of parasympathetic activity after HIE than MCE. The HIE protocols of Perkins *et al*.[10] and Mourot *et al*.[11] were, however, different when compared with ours, as HIE in theirs was performed at a higher average intensity than the MCE, though

Mourot *et al*.[11] matched the total physical work. The HIE and MCE in the current study were performed at the same average intensity (70% $VO_{2max}$). Thus, the present study adds to the previous work of Perkins *et al*.[10] and Mourot *et al*.[11] and provides additional evidence to support the notion that post-exercise autonomic recovery depends on the exercise intensity and not on the modality of exercise (HIE vs MCE), despite considerable intensity fluctuation in HIE. It would be of interest, in the future, to ascertain whether the critical role of exercise intensity in determining cardiac parasympathetic recovery is independent of total workload in different protocols commonly used in the prescription of HIE.

In the present study, all HRV indices had returned to baseline 3h after the termination of the exercises, suggesting that the sympathovagal balance altered by acute exercise had rapidly returned to resting conditions, and any possible increased risk of adverse cardiac events was mitigated.[4] Further, enhanced vagal regulation was observed one day after both exercise trials, as reflected by the higher RMSSD at 24 h post-exercise compared to PRE. It is now well established that the adaptive increased parasympathetic activity induced by exercise training may reduce the risk of lethal ventricular arrhythmias.[4] Thus, the current finding supports the notion that benefits traditionally associated with training may be realized quite rapidly,[25] albeit with repetition required. Indeed, a marked cardioprotective effect (e.g. smaller infarct size) may be evoked by a single bout of exercise and has been termed "exercise preconditioning".[25] Whether the cardioprotective effect of exercise preconditioning includes improved cardiac autonomic function has not been systematically investigated. Although the rebound increase of parasympathetic activity was also observed 24 h after a cross-country endurance race in athletes,[26] the well-trained experience might have confounded the preconditioning effect. The present lab-based study seems to suggest exercise preconditioning mediated by acute HIE or MCE may include enhanced vagal changes in sedentary, overweight/obese young females. Nevertheless, further work is required to confirm and validate these findings.

*cTnT change with different exercise trials and its association with HRV*

All participants in this study had elevated levels of cTnT after the two bouts of exercise. This is in keeping with findings from a previous meta-analysis describing a high prevalence of post-exercise cTnT elevation.[6] This study also confirmed previous studies that found that wide inter-individual variation exists in cTnT response to acute exercise,[6,7] but a large portion (> 90%) of variations cannot be explained by current understanding of participant attributes.[27] Moreover, we observed workload-matched HIE and MCE displayed similar peak post-exercise

cTnT values. The present findings support our recent studies[12,13] and extend to the matching of HIE and MCE for total mechanical work to average physical workload. Some studies suggest that increasing exercise intensity within the scope of continuous endurance exercise results in increased post-exercise cTnT levels.[6,7] The current work does not support a role for exercise intensity in determining an exercise-induced cTnT elevation in workload matched HIE and MCE. It would seem that post-exercise cTnT elevation in the current study might be related to the overall cardiac work rather than to any variation in exercise intensity. It should be noted that the current data are in contrast with the study by Ranjbar *et al*.,[14] who showed that MCE resulted in a higher cTnT elevation than HIE. Notably, this study employed heart rate during exercise to equalize the cardiac work of HIE and MCE. Given that heart rate lag and inertia at exercise onset and cessation occur during HIE,[28,29] the comparison of results from this study above should be interpreted with some caution.

Although post-exercise elevations in circulating cTn have been widely observed, the clinical significance of this phenomenon remains controversial.[6,7] It seems logical that an increase in cTnT might reflect a detrimental cardiac insult in the setting of impaired cardiac autonomic function.[9] In the present study, however, the peak increase in cTnT did not temporally match with reduced HRV at post-exercise. Moreover, cTnT was still significantly elevated at 24 h post-exercise despite a 'hypervagal' status at the same time point, the latter suggesting adequate recovery of cardiac perturbations from the last exercise bout.[5] These findings suggest that exercise-induced cTnT elevation and alteration in cardiac autonomic activity are likely to be two separate events. Therefore, the current findings further support the current mainstream view of a physiological substrate responsible for the post-exercise elevation of cTnT.[7]

It should be noted that our findings are at odds with those of Aagaard *et al*.,[9] who observed, after a 30-km cross-country running race, a significant association between post-exercise HRV reduction and cTnT elevation, which implied the magnitude of cTnT might carry clinically information. The subjects of Aagaard *et al*.[9] were, however, much older than ours (mean 50.2 vs 20.8 years old). Considering advancing age is associated with endothelial dysfunction and an impaired vascular function responsiveness to acute sympathetic activation during exercise,[30] we may speculate that aging may have facilitated the stronger association between post-exercise HRV and cTnT. Future studies are required to confirm this speculation.

*Implications*

There remains some reservation concerning the prescription of HIE to the untrained population (and clinical groups), due to increased risk of arrhythmic events as exercise intensity increases.[2] From the data presented here, in a sedentary, overweight/obese young female cohort, it is reassuring that HIE does not seem to induce a higher acute risk of adverse cardiac events compared to MCE, as reflected by similar HRV and cTnT kinetics (and no association between these markers). This information may be helpful in developing safer and more effective exercise strategies for improving fitness, especially in untrained obese individuals. In addition, current findings also provide new information that might assist clinicians with decision-making if faced with interpreting post-exercise values of cTnT in individuals after MCE and HIE with a similar workload, e.g. the appearance of a much larger increase in cTnT after HIE than MCE should raise concern for further clinical investigation.

*Limitations*

The current study has certain limitations that also provide some guidance for further research, beyond those already noted. Firstly, the participants in our study were physically able to finish the required HIE and MCE, and thus may not be fully representative of the general sedentary obese population. Secondly, although we attempted to control for menstrual cycle health (no oral contraceptive users and no one with menstrual dysfunction) in the female participants, we could not constrain testing to specific phases of the menstrual cycle on each exercise bout observed. Nevertheless, recent studies showed that the menstrual cycle phase did not mediate the post-exercise vagal recovery[31] and cTnT elevation.[13] Finally, current autonomic measurements were not taken during the exercise bouts, as traditional analysis methods of HRV require the processed signal to be stationary.[24] There is a need to use new technology such as the short-time Fourier transform[32] to overcome this limitation.

## Conclusion

In sedentary overweight/obese young females, a post-exercise reduction in vagal activity was transient and a rebound increase in vagal outflow occurred 24 h after acute HIE and MCE. In addition, cTnT was significantly elevated 3 h after HIE and MCE, and remained above baseline at 24 h post-exercise. The magnitude and time-course of changes in HRV indices and cTnT were not mediated by exercise modality (i.e., equal-workload HIE vs MCE).

The current data also support the contention that exercise-induced alteration in cardiac autonomic activity and cTnT elevation are largely separate phenomena.

## Conflicts of interest

The authors certify that there is no conflict of interest with any financial organization regarding the material discussed in the manuscript**.**


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## Authors contribution

CH, ZK, JN, and KG conceived and designed research; CH and MP performed experiments; CH, ZK, JN, HZ, QS and KG analyzed data; CH, ZK, JN, HZ, QS and KG interpreted results of experiments; CH, ZK, JN and KG wrote the manuscript; All authors have read and approved the final version of the manuscript, and agree with the order of presentation of the authors.

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**Table 1.** Heart rate variability parameters [mean±SE (95% Confidence Interval)] before (PRE) and immediately (0HR), 3h (3HR) and 24 h (24HR) after high-intensity interval exercise (HIE) and moderate-intensity continuous exercise (MCE)

| | PRE | 0HR | 3HR | 24HR |
|---|---|---|---|---|
| **HR (beat.min$^{-1}$)** | | | | |
| HIE | 71±1 (68-74) | 95±2 (89-100)† | 75±2 (72-79)† | 66±2 (62-70)† |
| MCE | 72±2 (69-76) | 96±3 (90-102)† | 75±2 (71-80)† | 68±2 (64-73)† |
| **RMSSD (ms)** | | | | |
| HIE | 41.5±4.2 (32.4-50.5) | 9.8±1.5 (6.5-13.2)† | 32.5±3.0 (26.1-38.9) | 53.5±5.1 (42.4-64.6)† |
| MCE | 34.1±4.0 (25.5-42.6) | 7.9±1.0 (5.7-10.0)† | 31.7±4.5 (21.9-41.4) | 54.0±8.6 (35.4-72.6)† |
| **LF (nu)** | | | | |
| HIE | 52.3±4.7 (42.3-62.4) | 69.0±4.3 (59.7-78.4)† | 51.8±3.3 (44.7-59.0) | 43.3±5.3 (31.8-54.7) |
| MCE | 56.0±4.3 (46.7-65.3) | 75.3±2.8 (69.2-81.3)† | 50.1±2.9 (43.9-56.3) | 53.8±5.4 (42.1-65.5) |
| **HF (nu)** | | | | |
| HIE | 47.5±4.7 (37.4-57.6) | 30.8±4.3 (21.4-40.1)† | 48.0±3.3 (40.9-55.1) | 56.6±5.3 (45.1-68.1) |
| MCE | 43.9±4.3 (34.6-53.2) | 24.6±2.8 (18.5-30.6)† | 49.8±2.9 (43.6-56.0) | 45.8±5.4 (34.2-57.5) |
| **LF/HF ratio** | | | | |
| HIE | 1.44±0.30 (0.79-2.08) | 4.06±1.44 (0.93-7.18)† | 1.23±0.16 (0.89-1.57) | 1.28±0.48 (0.24-2.31) |
| MCE | 1.54±0.22 (1.06-2.02) | 4.90±0.78 (3.23-6.60)† | 1.09±0.12 (0.83-1.35) | 1.61±0.30 (0.97-2.25) |

**HR,** heart rate**; RMSSD**, root mean square of successive differences between RR intervals; **LF**, low-frequency power of heart rate variability, **HF**, high-frequency power of heart rate variability

† Significantly different from corresponding PRE value, $p < 0.05$

**Table 2.** Serum cardiac troponin T [$ng.l^{-1}$, median (range)] before (PRE) and immediately (0HR), 3h (3HR) and 24 h (24HR) after high-intensity interval exercise (HIE) and moderate-intensity continuous exercise (MCE)

| | **PRE** | **0HR** | **3HR** | **24HR** |
|---|---|---|---|---|
| HIE | 1.50 (1.50-9.00) | 3.84 (1.31-13.47) | 7.04 (1.43-44.93)† | 7.17 (1.43-29.98)† |
| MCE | 1.50 (1.50-5.00) | 2.64 (1.03-30.61) | 13.83 (1.39-52.24)† | 4.16 (1.41-85.07)† |

† Significantly different from corresponding PRE value, $p < 0.05$

## FIGURE LEGENDS

**Fig 1.** The cardiac troponin T levels (cTnT, ng.l$^{-1}$) in individuals (n=14) before (PRE) and immediately (0HR), 3h (3HR) and 24 h (24HR) after moderate-intensity continuous exercise (MCE) and high-intensity interval exercise (HIE).

Note: Individual data points are presented by symbols with values for the same participant connected by lines for each trial, and each participant has the same symbol and line across two trials. The horizontal dotted line is the upper reference limit (14 ng.l$^{-1}$).