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High-sensitivity cardiac troponin T release after a single bout of high-intensity interval exercise in experienced marathon runners

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ABSTRACT

Objective: The purpose of this study was to investigate the effects of a single bout of high-intensity interval exercise (HIIE) on high-sensitivity cardiac troponin T (hs-cTnT) release and to explore the potential influencing factors.

Methods: Twenty-one experienced marathon runners completed HIIE on treadmill. Each bout of HIIE included a hard run (15.8 ± 1.3 km·h^{-1}) at 90% vVO_{2max} for 2 min followed by an easy run (8.8 ± 0.7 km·h^{-1}) at 50% vVO_{2max} for 2 min performed 23 times within 92 min. Heart rate (HR) was recorded every 2 min during HIIE. The hs-cTnT level was measured before (pre), immediately after (0 h), and at 4 and 24 h after exercise.

Results: The hs-cTnT level was elevated at 0 h, peaked at 4 h, and had not returned to the baseline value at 24 h after exercise. The response of hs-cTnT at 4 h was positively related to exercise HR. Subjects with a greater increase in hs-cTnT level had a higher exercise HR under fixed exercise intensity.

Conclusion: HIIE at 90% vVO_{2max} interspersed with 50% vVO_{2max} for recovery can elicit hs-cTnT elevation. HR is a good predictor of exercise-induced cardiac troponin (cTn) release under fixed exercise intensity. Further study should consider to correct for HR when constructing impact factors contributing to exercise-induced cTn release.

1. Introduction

Cardiac troponin (cTn) is a highly specific and sensitive biomarker for the identification of cellular damage or injury in the diagnosis of acute myocardial infarction.[1,2] Exercise is possibly the only documented cause of cTn release that is not associated with an adverse clinical outcome.[3] Many reports suggested that cTn increased in 0% to 100% of subjects,[4,5] peaked at 3 to 4 h and rapidly returned to baseline level within 24 h after the completion of intense exercise.[6] Exercise-induced cTn release is related to exercise intensity and duration, age, training experience, cardiovascular disease, and the environment.[7–10] Transient changes in membrane permeability may be responsible for the release of unbound cTn from the cytosolic pool of cardiomyocytes,[11] but the mechanism is still under debate. The factors that may affect the increase in cTn require further investigation to determine the mechanism and clinical relevance of such exercise-induced perturbations.

From the existing evidence, it seems that exercise intensity, mostly evaluated by heart rate (HR), is a predictor of cTn elevation,[12–14] but this has not been confirmed in other studies.[15,16] One reason for this inconsistency is that there seems to be a threshold of exercise intensity at which cTn may not be released at

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a low HR.[17] Furthermore, exercise-induced cTn release, including cardiac troponin T (cTnT) and I (cTnI), subunits of cTn, has been predominantly observed in field-based settings, during which exercise intensity including running velocity and HR are hard to manipulate and instantly record. Consequently, it is impossible to show the respective independent effects of exercise intensity and HR *per se* on the changes of cTn. Actually, cTnT elevation is primarily dependent on HR when tachycardia is present in patients with no coronary disease.[18] Therefore, it is necessary to survey the independent effect of HR on cTn release during exercise.

High-intensity interval exercise (HIIE) performed at an intensity associated with competition pace and maximal oxygen consumption (VO_{2max}) is a training method commonly used by experienced runners. In contrast to constant running at a steady state and submaximal intensity, HIIE places a fluctuated workload on the myocardium, during which intensity can be sustained at a higher level than that attained during constant exercise load.[19] Various HIIE protocols, that of repeated bouts of 1 to 8 min of hard running at 90% to 100% VO_{2max}, have proven to be effective in improving the VO_{2max} of experienced runners,[19–24] and HIIE has also been found to benefit cardiorespiratory fitness in non-athletes.[25] To date, the data on exercise-induced cTn release during interval exercise mode are very limited and inconsistent.[26] In field-based settings, cTn elevation was observed after basketball,[27] floorball,[28] and sprints[29] but not after rugby, football,[30] and indoor soccer matches.[31] Research on cTn change induced by HIIE in a laboratory-based setting remains limited. Lu and colleges reported no significant change in cTnI after 7 intervals of a 2-min run at 90% VO_{2max} interspersed by 1-min recovery periods.[32] Due to the development of a high-sensitivity cardiac troponin T (hs-cTnT) assay, it is now possible to reliably detect changes in cTnT at low levels.[33] Therefore, the effect of acute bouts of HIIE on hs-cTnT release can and should be investigated.

The purpose of this study was to investigate the effect of HIIE on hs-cTnT release and the relationship between hs-cTnT release and exercise HR, physical characteristics, and training information in the setting of the same relative exercise intensity (runs at hard and easy velocities).

2. Methods

2.1. Subjects

After approval of this study by the local Ethical Committee, 21 experienced marathon runners were recruited from the Department of Physical Education at a local university. They were free of diseases, did not smoke, and had not taken drugs or antioxidant supplements in the month before the study. Training history, volume, and personal best time in a marathon race (within 6 months) were self-reported. Table 1 summarises these physical characteristics and training information. An initial medical screening and examination were performed by a team of medical doctors and technicians. None of the subjects had a history of cardiac symptoms, and all had normal resting blood pressures and electrocardiographic results. All subjects provided their written consent and were fully informed about the purposes, procedures, and potential cardiovascular risks of this study.

2.2. Preliminary testing

All tests took place in a local sports science research centre between October and December at 14:00 and 18:00. Air conditions were similar for each test with small variations in temperature (20.9 ± 1.6°C) and humidity (43.9% ± 9.5%). Subjects were asked to refrain from intense exercise and alcohol intake for 48 h before and after each test and were allowed to freely ingest pure water during the test. Before the HIIE protocol, VO_{2max} (Max-II, Physio-Dyne Instrument Corp., Quogue, NY) and corresponding velocity of VO_{2max} (vVO_{2max}) for each subject were determined on a treadmill (Pulsar 4.0, h/p/cosmos sports & medical GmbH, Nussdorf-Traunstein, Germany) at a 2% slope. After a general warm up, the initial speed of 12 $km \cdot h^{-1}$ was increased by 1 $km \cdot h^{-1}$ every 3 min until the test subject's respiratory exchange ratio reached 1.00. The speed was then increased every 2 min by 1 $km \cdot h^{-1}$. The test was stopped either when the increase in VO_2 was less than 2.1 $ml \cdot kg^{-1} \cdot min^{-1}$ while the respiratory exchange ratio was 1.15 or greater, or when exhaustion was reached.[38] The VO_{2max} and maximal heart rate (HR_{max}) were the average of the highest value over 30 s, and the vVO_{2max} was the minimal speed at which VO_{2max} was reached, but only if this speed was sustained for at least 1 min.

2.3. Experimental trial

Each bout of HIIE consisted of a 2-min hard run at 90% vVO_{2max} followed by a 2-min easy run at 50% vVO_{2max} for recovery performed 23 times within 92 min.

2.4. Measurements

During HIIE, the subjects' HR (S810, Polar, Finland) was recorded before exercise in rest and during exercise every 2 min. Venous blood samples (5 ml) from the antecubital vein were collected before HIIE (pre), immediately after (0 h) and at 4 h and 24 h after exercise. The blood was allowed to clot at room temperature and was then centrifuged at 3000*g* for 15 min. The separated serum was then collected and stored at −80°C for further analysis.

The hs-cTnT analysis method is based on a new electrochemiluminescence technology that uses Elecsys 2010 automated batch analysers (Roche Diagnostics, Basel, Switzerland).[33] The measurement range was 3 to 1000 $pg \cdot ml^{-1}$. Data lower than the minimal detection limit was recorded as 3 $pg \cdot ml^{-1}$ when statistical analysis was conducted. The 99th percentile cut-off concentration and the level at the 10% coefficient of variation were 14 and 13 $pg \cdot ml^{-1}$, respectively, and the upper reference limit (URL) was set at 14 $pg \cdot ml^{-1}$.

2.5. Statistical analyses

Data are presented as means ± standard deviations (SD) unless otherwise stated. Log-transformation was applied to the hs-cTnT values. A one-way repeated analysis of variance was used to determine the effect of time and identify the peak level of hs-cTnT across sampling points with *post hoc* Bonferroni tests when appropriate. The relationship between the increase of hs-cTnT at 4 h after exercise (delta scores of 4-h post- and pre-exercise values, $\Delta hs\text{-}cTnT_{4\ h}$) and the relevant variables, physical characteristics, training information and exercise HR and velocity were assessed by bivariate Pearson's product-moment correlation coefficients.

Depending on the magnitude of $\Delta hs\text{-}cTnT_{4\ h}$, the subjects were defined as a high responder (n = 7) with a large increase in 4-h hs-cTnT ($\Delta hs\text{-}cTnT_{4\ h}$: 32.0 to 95.0; 4-h hs-cTnT: 35.0 to 98.0 $pg \cdot ml^{-1}$), a medium responder (n = 7) with a moderate increase in 4-h hs-cTnT ($\Delta hs\text{-}cTnT_{4\ h}$: 15.1 to 31.9; 4-h hs-cTnT: 18.1 to 38.0 $pg \cdot ml^{-1}$) and a low responder (n = 7) with a small increase in 4-h hs-cTnT ($\Delta hs\text{-}cTnT_{4\ h}$: 0.3 to 13.0; 4-h hs-cTnT: 5.8 to 16.0 $pg \cdot ml^{-1}$). A mixed between-subjects and within-subject analysis of variance was conducted to explore the differences between the three groups in hs-cTnT and HR of hard and easy runs and average values, and a one-way between-groups analysis of variance was used to analyse the differences in subjects' physical characteristic, training information variables and percent of HR reserve (% HRR). The level of