Applied Physiology, Nutrition, and Metabolism
Physiologie appliquée, nutrition et métabolisme

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Journal:	*Applied Physiology, Nutrition, and Metabolism*
Manuscript ID:	apnm-2015-0055.R1
Manuscript Type:	Article
Date Submitted by the Author:	n/a
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Keyword:	athlete performance, elite athlete performance < athlete performance, exercise intensity < exercise, exercise performance < exercise, exercise physiology < exercise

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Abstract

This study had two specific objectives; 1) to examine the individual variation in the pattern of cardiac troponin I (cTnI) and N-terminal pro-brain natriuretic peptide (NT-proBNP) response to high intensity rowing exercise, and 2) to establish if individual heterogeneity in biomarker appearance was influenced by athletic status (elite vs. amateur). We examined cTnI and NT-proBNP in 18 elite and 14 amateur rowers before and 5 min, 1, 3, 6, 12, and 24 h after a 30 min maximal rowing test. Peak post-exercise cTnI (pre: 0.014 ± 0.030, peak post: 0.058 ± 0.091 $\mu g.L^{-1}$, $p = 0.000$) and NT-proBNP (pre: 15 ± 11, peak post: 31 ± 19 $ng.L^{-1}$, $p = 0.000$) were elevated. Substantial individual heterogeneity in peak and time course data noted for cTnI. Peak cTnI exceeded the upper reference limit (URL) in 9 elite and 3 amateur rowers. No rower exceeding the URL for NT-proBNP. Elite rowers had higher baseline (0.019 ± 0.038 vs. 0.008 ± 0.015 $\mu g.L^{-1}$; $p = 0.003$) and peak post-exercise cTnI (0.080 ± 0.115 vs. 0.030 ± 0.029 $\mu g.L^{-1}$; $p = 0.022$) than amateur rowers but the change with exercise was similar between groups. There were no significant differences in baseline and peak post-exercise NT-proBNP between groups. In summary, marked individuality in cTnI response was noted to a short but high intensity rowing bout. Athlete status did not seem to mediate the change in cardiac biomarkers to high intensity exercise.

Keywords: exercise; cTnI; NT-proBNP; athletic status; rowing, elite athletes, amateur athlete

Introduction

An increasing number of studies have described the elevation of cardiac troponin I (cTnI), a biomarker of cardiac cell necrosis, and N-terminal pro-brain nariuretic peptide (NT-proBNP), a biomarker of cardiac dysfunction, after prolonged and strenuous exercise (Scharhag et al. 2008; Shave et al. 2010a). The cardiac biomarker response to short-duration, high-intensity exercise is largely unknown although some have suggested that within the endurance exercise domain cTnI increased with exercise intensity (Legaz-Arrese et al. 2011; Serrano-Ostáriz et al. 2011). Shave et al. (2010b) are one of the few groups to have studied the cTnI response shorter, high-intensity bouts of exercise. In spite of the limited volume of exercise (30 min all-out treadmill run) cTnI was elevated during recovery in 75% of athletes (Shave et al. 2010b).

Importantly Shave et al. (2010b) observed that the cTnI appearance during recovery was markedly heterogeneous and confirmed similar individuality of response noted in field based studies of prolonged exercise (Shave et al. 2010a) as well as an observation from a meta-analysis (Shave et al. 2007). The percentage of individuals with post-exercise cTnI or cTnT levels above the upper reference limit (URL) has varied from 0% (Roth et al. 2007) to 100% (Middleton et al. 2008) in individual studies but this may partially represent the "lottery" of a single post-exercise blood test. It is important that in on-going cardiac biomarker research that multiple post-exercise blood draws occur to fully understand any heterogeneity in cTnI or NTpro-BNP peak concentrations as well as recovery kinetics (Middleton et al. 2008).

The influence of exercise intensity on NT-proBNP release is less well known. Within the endurance exercise domain data suggests that NT-proBNP increase may be more influenced by exercise duration (Serrano-Ostáriz et al. 2009) but studies involving shorter bouts of high intensity exercise in well-trained athletes are limited.

Individual variability in biomarker response in the extant literature has been speculated to be, at least partially, related to training or "athletic" status. It has been suggested that highly-trained individuals have lower post-exercise cTnI and NT-proBNP release (Mehta et al. 2012; Neilan et al. 2006). Indeed, the only two previous studies on elite athletes reported normal post-exercise cardiac biomarker levels (Bonetti et al. 1996; König et al. 2003). Contrary, we have recently observed in untrained subjects that a controlled endurance training intervention resulted in higher pre- and post-exercise values of cTn with no changes in NT-proBNP (Legaz-Arrese et al., 2015). Currently, the influence of training level on cardiac biomarker release has not yet been evaluated in a controlled study with disparate groups in terms of training or athletic status completing a similar (relative) high intensity exercise bout. Finally, it has been postulated that increases in both biomarkers may be dependent on their respective resting values (Legaz Arrese et al. 2005; Serrano-Ostáriz et al. 2011) although this construct has not been studied in different athlete groups.

Consequently, the purpose of the present study was to determine the cardiac biomarker response to a short duration, high intensity bout of rowing with specific emphasis on detailing individual responses across multiple assessment points during a 24 hr recovery period. A secondary purpose was to determine the influence of athlete status on cTnI and NT-proBNP release by comparing two cohorts; amateur and elite rowers.

Material and Methods

Participants

Thirty-two male rowers were recruited from a large Rowing Club in Spain through an open invitation to all of its members. Volunteers included elite rowers (n = 18) who had at least 3 yr of competitive history at the national or international level (1 world champion, 1 under-23

world champion, 1 Olympic competitor, 2 Spanish champions, and 3 Spanish sub-champions) and were training ≥5 days per week and non-competitive amateur rowers (n = 14) who trained ≤3 days per week. All rowers provided informed written consent. The study followed the ethical guidelines of the Declaration of Helsinki and was approved by the Research Ethics Committee of the Government of Aragón (CEICA; Spain).

Research Design and Protocols

All rowers attended a preliminary testing session 1 week before the main study was performed. At this initial testing body height was measured to the nearest 0.1 cm (SECA 225 SECA, Hamburg, Germany). Body mass was determined to the nearest 0.05 kg (SECA 861, SECA, Hamburg, Germany). A questionnaire was completed to obtain personal data, performance level, training history, and history of any cardiac symptoms. Exclusion criteria were a significant personal or early family history of cardiovascular disease and/or abnormal ECG at baseline examination.

The rowers then performed a progressive incremental test to exhaustion on a Concept II rowing ergometer (Model C, Morrisville, VT, USA) to determine the maximal heart rate (HR) (Polar Electro Oy, Kempele, Finland). Prior to the test, the rowers completed a self-paced 5-min warm-up (HR <130 beats.min^{-1}). The test began at a workload of 150 W (elite rowers) or 75 W (amateur rowers) with workload increments of 50 W every 3 min until exhaustion. Strong verbal encouragement was provided to all participants.

After a minimum of 7 days all participants returned to the laboratory to complete the 30 min rowing test. All participants were fully habituated to the 30 min all-out rowing test protocol and were asked to abstain from strength training and strenuous exercise for 48 h before testing. All high-intensity testing sessions occurred at 11:00 am in a sports hall at a temperature of 18-21 °C and a relative humidity of 50-60%. The rowers completed a self-

paced 5-min warm-up (HR <130 beats.min^{-1}) followed by a 30-min "all-out" rowing test. Pairs of rowers competed side-by-side to mimic a regular competition and again strong verbal encouragement was provided. During the test HR was recorded continuously via a Polar HR monitor (Polar Electro Oy, Kempele, Finland) and downloaded using Polar Precision Performance software (v. 3.0). The mean power output (W) and distance covered were recorded every 5 min from the rowing ergometer screen. Immediately after the test was completed, the participants rated the test for perceived exertion (RPE) (Borg and Kaijser 2006). Venous blood samples were taken before, immediately after (5 min), as well as 1, 3, 6, 12, and 24 h after exercise to assess serum cardiac-specific biomarkers.

Blood Sampling and Analysis

Blood samples were drawn by repetitive venipuncture from an antecubital vein and quickly centrifuged. The serum and plasma were drawn off and stored at −80 °C for later analysis. cTnI was analyzed from samples of EDTA (ethylenediaminetetraaceitic acid) plasma with the Access AccuTnI assay (Beckman Coulter, Fullerton, CA, USA). The imprecision profile of 839 duplicate samples showed 10% and 20% coefficients of variation values of 0.014 and 0.008 μg.L^{-1}, respectively. The URL for cTnI, defined as the 99th percentile of healthy participants, was 0.04 μg.L^{-1} (Eggers et al. 2007). NT-proBNP was analyzed in the serum with an Elecsys proBNP electrochemiluminescent immunoassay on the Roche Elecsys 1010 (Roche Diagnostics, Lewes, United Kingdom) with an analytical range of 5–35,000 ng.L^{-1} and intra- and interassay imprecisions of 0.7–1.6% and 5.3–6.6%, respectively. The URL for NT-proBNP was considered to be 125 ng.L^{-1} (Silver et al. 2004).

Statistical analysis

Statistical analyses were performed using the IBM Statistical Package for the Social Sciences (IBM SPSS Statistics, v. 20.0 for WINDOWS). Cohort data are presented as the mean ± standard deviation unless otherwise stated. Kolmogorov-Smirnov tests were used to check for normal distribution and data for cTnI and NT-proBNP were log-transformed prior to statistical testing. To measure the impact of sampling time during recovery (pre, 5 min, 1, 3, 6, 12, and 24 h post-exercise) as well as athletes status (elite and amateur) upon cTnI and NT-proBNP mixed model 2-way ANOVAs were performed with post-hoc Bonferroni tests employed when appropriate. The association between the exercise increase in both biomarkers and other relevant variables (e.g., baseline biomarker concentration, mean and max exercise HR) were assessed using bivariate Pearson's product-moment correlation coefficients. The values were considered to be significant if $p < 0.05$.

Results

The characteristics of the elite and amateur rowers are shown in Table 1. The elite rowers had more years of training, greater weekly training frequency, and higher weekly training volume (all $p < 0.05$). Performance during the grade rowing test was greater in the elite rowers (294 ± 18 W vs. 211 ± 44 W; $p = 0.000$) whereas maximum HR was similar between groups (elite: 196 ± 7 beats.min^{-1}; amateur 193 ± 9 beats.min^{-1}; $p = 0.372$).

Maximal 30-min rowing test

All of the subjects completed the maximal 30-min rowing test and every blood draw. Performance during the 30 min all out test was substantially greater in the elite athlete (Table 2). Whilst mean HR was higher in the elite (180 ± 7 beats.min^{-1}) compared to the amateur rowers (171 ± 12 beats.min^{-1}; $p = 0.023$) there was no difference in the maximum HR (elite

rowers: 195 ± 7 beats.min^{-1}, amateur rowers: 188 ± 11 beats.min^{-1}; p = 0.061) or RPE (elite rowers: 8.7 ± 0.5, amateur rowers: 8.6 ± 0.5; p = 0.536).

cTnI release

A significant main effect of sampling time was observed for cTnI with an elevation at 3-, 6-, and 12-h post-exercise compared to baseline (p = 0.000) (Table 3). All participants presented with an increase in cTnI post-exercise with the URL for cTnI exceeded by 2 rowers at all measurements points and another 10 rowers (8 elite and 2 amateur) having sporadic data points above the URL during recovery (Fig. 1). The maximum post-exercise cTnI was observed at 3 h in 11 individuals, 6 h in 19 individuals, and 12 h of recovery in 2 individuals. A significant main effect for athlete status was observed with cTnI data higher in elite rowers including pre-exercise values (amateur: 0.008 ± 0.015 μg.L^{-1}; elite: 0.019 ± 0.038 μg.L^{-1}; p = 0.003). There was no significant interaction of test time and athlete status with respect to cTnI (p = 0.311). In support of this the maximal increase in cTnI (peak-baseline) was not significantly different between groups (elite: 0.062 ± 0.083 μg.L^{-1}; amateur: 0.023 ± 0.021 μg.L^{-1}; p = 0.145). The absolute post-exercise cTnI values were significantly correlated with the basal values (r = 0.88, p = 0.000) as well as mean exercise HR (r = 0.35, p = 0.048)

NT-proBNP release

There was a main effect of time with an increase in NT-proBNP from pre-exercise at 5 min, 1-, 3-, 6-, 12-, and 24-h post-exercise (p = 0.001; Table 3). There was a rise in NT-proBNP post exercise in all subjects but the URL was not exceeded by any subject (Fig. 2). The maximum post-exercise NT-proBNP values were observed at 5 min in 10 individuals, 1 h for 4 individuals, 6 h for 7 individuals, 12 h for 4 individuals, and 24 h for 11 individuals. There was no significant main effect of athlete status on NT-proBNP data and there was no time by

athlete status interaction effect. In support of this latter point there was no difference between the elite and amateur rowers with respect to the peak NT-proBNP increase (14 ± 11 vs. 18 ± 13 $ng.L^{-1}$, respectively; $p = 0.470$). Basal NT-proBNP values were significantly correlated with the maximum post-effort values ($r = 0.83$, $p = 0.000$) but there was no correlation between change in NT-proBNP and cTnI data.

Discussion

The main findings of this study were; (1) a single 30-min bout of "all-out" rowing exercise resulted in a significant increase in the cTnI and NT-proBNP in both elite and amateur rowers, (2) significant individual heterogeneity in peak cTnI during recovery was noted with the URL for cTnI exceeded in 12/32, (3) less individual variability was apparent in peak NT-proBNP response with no data point exceeded the URL, (4) baseline and post-exercise cTnI data were higher in elite rowers, but (5) the rowing-induced changes in cTnI and NT-proBNP were independent of athlete status.

Post-exercise cTnI peak and kinetics in elite and amateur rowers

Our results in rowers extend the findings of Shave et al. (2010b) who employed a 30 min high intensity run and demonstrate that cTnI is elevated following short-duration, high-intensity exercise in non-elite athletes. An elevation in cTnI occurred in all participants despite the relatively short duration and limited exercise volume. In prolonged exercise there is some evidence to suggest that cTnI release is positively associated with exercise intensity (Fu et al. 2009; Serrano-Ostáriz et al. 2011; Shave et al. 2007). Whilst the current study does not compare exercise intensities it adds to the extant data that different types and intensities of exercise can stimulate an increase in circulating cTnI. According to the results of Shave et al. (2010b) cTnI release following short-duration intense exercise may be as common as

when prolonged exercise trials are studied and the current study supports this contention. This also underscores the necessity to complete blood draws during recovery (Middleton et al. 2008).

To our knowledge, this study is the first to demonstrate cTnI release with exercise in elite athletes with values that exceed the URL in some, but not all, participants. Previously, only two studies had evaluated cTnI release in elite athletes. Bonetti et al. (1996) analyzed 25 cyclists participating in the Giro d'Italia and reported detectable cTnT values in only 5 athletes; moreover, these values were below the cut-offs considered to be indicative of myocardial insult. Similarly, König et al. (König et al. 2003) reported normal post-exercise cTnT levels in 11 professional road cyclists. Both studies were constrained by limited blood sampling (pre- and post-exercise design) and by less-sensitive measurement equipment.

Despite the fact that all participants experienced a rise in cTnI post-exercise the magnitude of peak post-exercise levels was variable, which also supports the data from Shave et al. (2010b). Recent studies have also demonstrated "positive" high sensitivity cTnT (hs-cTnT) values after prolonged exercise in most subjects (86-94%) (Mingel et al. 2009; Saravia et al. 2010; Scherr et al. 2011; Tian et al. 2012), but with marked heterogeneity in peak hs-cTnT (Scherr et al. 2011; Tian et al. 2012). It is not known what personal, environmental or exercise-related factors mediate the heterogeneity and this requires on-going study. Whilst we observed variability in the peak cTnI values recorded 94% of participants recorded their peak cTnI between 3 or 6 h which suggests some consistency in cTnI kinetics and agrees with previous data gathered after a treadmill run (Tian et al. 2012).

As in previous studies (Legaz-Arrese et al. 2011; Serrano-Ostáriz et al. 2011), it is interesting that the main factor that significantly predicted post-exercise values of cTnI was their respective pre-exercise values. In a broad range of pathologies and patient groups baseline cTn values are repeatedly and robustly associated with adverse cardiovascular prognosis and

mortality (deFilippi et al. 2010). In healthy population little attention has been focused to the variability of baseline cTn values and whether this variability may have clinical significance. On this matter, our results provide that the athletic status may be one of the factors that determine the heterogeneity in baseline cTnI. Further research into the factors associated with the inter-subject variability in the baseline values of cTn are required.

Certain authors suggest that the post-exercise cTnI release is greater in less well-trained individuals (Fortescue et al. 2007; Mehta et al. 2012; Mingels et al. 2009; Neilan et al. 2006). However, other studies did not observe any relationship between training level and cTnI release (Eijsvogels et al. 2015; Hubble et al. 2009; Jassal et al. 2009; Scherr et al. 2011; Serrano-Ostáriz et al. 2009). Our results demonstrating greater pre- and post-exercise values of cTn in elite rowers than in amateur rowers. These data are consistent with our recent controlled endurance training intervention (Legaz-Arrese et al. 2015) and a field based study with marathoners (Saravia et al. 2010). Contradiction with previous studies may relate to differences in exercise regime, training status as well as the limited by the number of blood samples taken during the recovery period, in past work.

There has been some descriptive association between peak post-exercise cTnI and mean exercise HR (Fu et al. 2009; Legaz-Arrese et al. 2011; Serrano-Ostáriz et al. 2009). Conversely, the higher absolute and relative work performed by the elite rowers in the 30 min exercise bout did not result in a greater change in cTnI during recovery when compared to amateur rowers. Overall there is no convincing evidence that exercise intensity mediated the cTnI response within the current research design.

We do not know the reasons behind the higher cTnI baseline levels in elite vs. amateur rowers. A previous study also showed that runners with detectable hs-cTnT were significantly better trained than runners in whom hs-cTnT was non-detectable (Saravia et al. 2010). Also, we observed that a controlled endurance training intervention resulted in higher

pre-exercise values of hs-cTnT (Legaz-Arrese et al. 2015). One hypothesis is that this effect is due to the successive training sessions with limited recuperation time for elite athletes. However, this seems unlikely to be a factor in this study because subjects were required to abstain from vigorous athletic activity for 48 h before each exercise test. Furthermore, if the greater baseline cTnI values were a consequence of incomplete recuperation, they ought to have similarly increased baseline levels of NT-proBNP, based on the results observed in this study. In a previous study, a significantly higher baseline hs-cTnT concentration was obtained in males compared to females (Mingels et al. 2009). Given that the mean heart size is larger for male and elite athletes than for female and amateur athletes (Legaz-Arrese et al. 2006; Legaz Arrese et al. 2005), it is reasonable to expect different cTn reference values between these groups. Future research may wish to address this issue.

Post-exercise NT-proBNP peak and kinetics in elite and amateur rowers

This investigation is, to our knowledge, the first study that demonstrates NT-proBNP release as a consequence of a short-duration, high-intensity exercise in elite athletes. Increased NT-proBNP has been reported in multiple prolonged endurance exercise studies (Legaz-Arrese et al. 2011; Neilan et al. 2006; Sahlén et al. 2008; Serrano-Ostáriz et al. 2009), and the current data extend this phenomenon to short-duration, high-intensity exercise. The observed increase are somewhat smaller than previous (ultra) endurance exercise studies (Neilan et al. 2006; Serrano-Ostáriz et al. 2009) which may not be surprising when one considers that BNP is elevated in response to volume overload and myocyte stretch (Shave et al. 2007) and this is likely to be stressed to a much greater extent in endurance exercise.

Our results demonstrate that like to cTnI, NT-proBNP values post exercise, as well as overall kinetic of appearance, is subject to a degree of heterogeneity. In agreement with the above mentioned study of Tian et al. (2012), levels of NT-proBNP increased immediately after

exercise and were still elevated at 24 h. The elevation in NT-proBNP at 24 h reflects an increase beyond the kinetics of NT-proBNP and its half-life (Silver et al. 2004). Other factors associated with strenuous exercise, such as a temporary reduction in kidney function and changes in cardiac function and hemodynamics, have been suggested to contribute to a sustained elevation in NT-proBNP (Tian et al. 2012), but this requires further study.

Our results show that although peak NT-proBNP data was heterogeneous the URL was not exceeded by any subject. Contrary to the data for cTnI there was no apparent difference in NT-proBNP between subject groups. In previous studies the influence of training level or athletic status on NT-proBNP release has been controversial (Herrmann et al. 2003; Legaz-Arrese et al. 2011, 2015; Neilan et al. 2006; Scharhag et al. 2006; Serrano-Ostáriz et al. 2009), likely because of the inability to precisely control for several variables, such as effort duration. Specifically, our study confirms previous results showing that the baseline NT-proBNP is a key factor related to exercise-induced NT-proBNP increase (Carranza-García et al. 2011; Legaz-Arrese et al. 2011, 2015; Sahlén et al. 2008; Serrano-Ostáriz et al. 2011). Interestingly, we observed greater individual variability in time to peak NT-proBNP than for cTnI, and consequently, previous studies may significantly underestimate NT-proBNP release if a single post-exercise sample is taken. Future studies should be performed to determine NT-proBNP kinetics differences among individuals after different types of exertion.

Implications

The fact that cTnI elevation was observed in all, in the absence of any other signs or symptoms of cardiovascular disease as well as with a rapid onset of accumulation and recovery within the study period would add to the suggestion that this phenomenon is a normal physiological process. Clinicians should be aware regardless of athletic status, it is

possible to observe cTnI but not NT-proBNP values exceeding the URL in the first hours of recovery after a short-duration, high-intensity exercise period in a high percentage of individuals. Since cTnI is recommended as a sensitive and specific marker for cardiac damage in the diagnosis of acute myocardial infarction, caution should be taken when interpreting post-exercise cTnI levels. The results of this study are relevant for clinicians as it could improve medical decision making.

Strengths and limitations

Strengths of the present study include the controlled exercise regimen, matched elite and amateur rowers, serial blood sampling, and the inclusion of cTnI and NT-proBNP values. However, several limitations should be considered. Two of the rowers had cTnI above the URL pre-exercise. The study is limited by only having analyzed associations between biomarkers and athletic status in young male rowers. The impact of age and sex should be studies as factors that may partially mediate the release of cardiac biomarkers with exercise (Scharhag et al. 2008; Shave et al. 2010). The observed differences in the values of cTnI and NT-proBNP between elite and amateur rowers may have resulted from differences in the level of training but could also be associated with other factors, such as genetic differences. To resolve this issue, because of the difficulty of establishing a control group with athletes, it would be also interesting to observe in previously untrained subjects, the effect of training programs on exercise-induced cardiac biomarker release.

Conclusions

In conclusion, our results show that 30 min of high-intensity rowing results in the elevation of both cTnI and NT-proBNP across a 24 h recovery period. Whilst a rise in cTnI and NT-proBNP was observed in all rowers, the peak values recorded were highly variable with some

353 cTnI data above URL. Kinetic data for cTnI were more consistent and there does not appear
354 to be an important role for athlete or training status in mediating exercise biomarker
355 responses beyond the impact of potential group differences in baseline data.
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357 ***Acknowledgments***
358 We gratefully acknowledge the participation and collaboration of all elite and amateur rowers
359 from Club Nautic Amposta (Spain). This study was supported by the DEP 2010-16767 grants
360 from the National Plan for Research, Development and Innovation (R + D + i) MICINN.
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476

477 ***Figure Legends***

Fig. 1. Individual data points for cTnI ($\mu g.L^{-1}$) in elite (*n* = 18) (*a*) and amateur (*n* = 14) (*b*) rowers at pre-exercise (PRE), as well as 0, 1, 3, 6, 12, and 24 h (0HR, 1HR, 3HR, 6HR, 12HR, 24HR, respectively) after a 30 min maximal rowing test. The horizontal dotted line is the upper reference limit (99th percentile) at 0.04 $\mu g.L^{-1}$.

Fig. 2. Individual data points for NT-proBNP ($ng.L^{-1}$) in elite (*n* = 18) (*a*) and amateur (*n* = 14) (*b*) rowers at pre-exercise (PRE), as well as 0, 1, 3, 6, 12, and 24 h (0HR, 1HR, 3HR, 6HR, 12HR, 24HR, respectively) after a 30 min maximal rowing test. All values were lower than the URL (125 $ng.L^{-1}$).

Table 1. Participant characteristics by athletic status.

	Age (years)	Weight (kg)	Height (cm)	Rowing training history (years)	Rowing training frequency (sessions/week)	Rowing training volume (hours/week)
Elite rowers	21.0 ± 4.1	77.9 ± 6.0	181.4 ± 6.0	8.2 ± 5.4*	6.9 ± 0.3*	22.1 ± 6.6*
Amateur rowers	21.2 ± 2.0	76.6 ± 8.7	177.0 ± 9.0	3.7 ± 1.5	1.6 ± 0.5	2.9 ± 0.8

Note: Values are means ± standard deviations (elite rowers: $n = 18$; amateur rowers: $n = 14$). * Significant differences between elite and amateur rowers.

Table 2. Performance during the maximal 30-min rowing test.

	0-5 min power (W)	5-15 min power (W)	15-25 min power (W)	25-30 min power (W)	Mean power (W)	Percentage of max power (%)
Elite rowers	260 ± 23*	254 ± 22*	251 ± 23*	286 ± 27*	259 ± 23*	88 ± 3*
Amateur rowers	165 ± 48	156 ± 37	157 ± 33	179 ± 37	161 ± 36	76 ± 5

Note: Values are means ± standard deviations (elite rowers: n = 18; amateur rowers: n = 14). * Significant differences between elite and amateur rowers. Similar pacing strategy was observed in both groups, with a significant increase in rowing performance in the last 5 min.

Table 3. cTnI (μg.L^{-1}) and NT-proBNP (ng.L^{-1}) before and after 30 min of high-intensity rowing exercise.

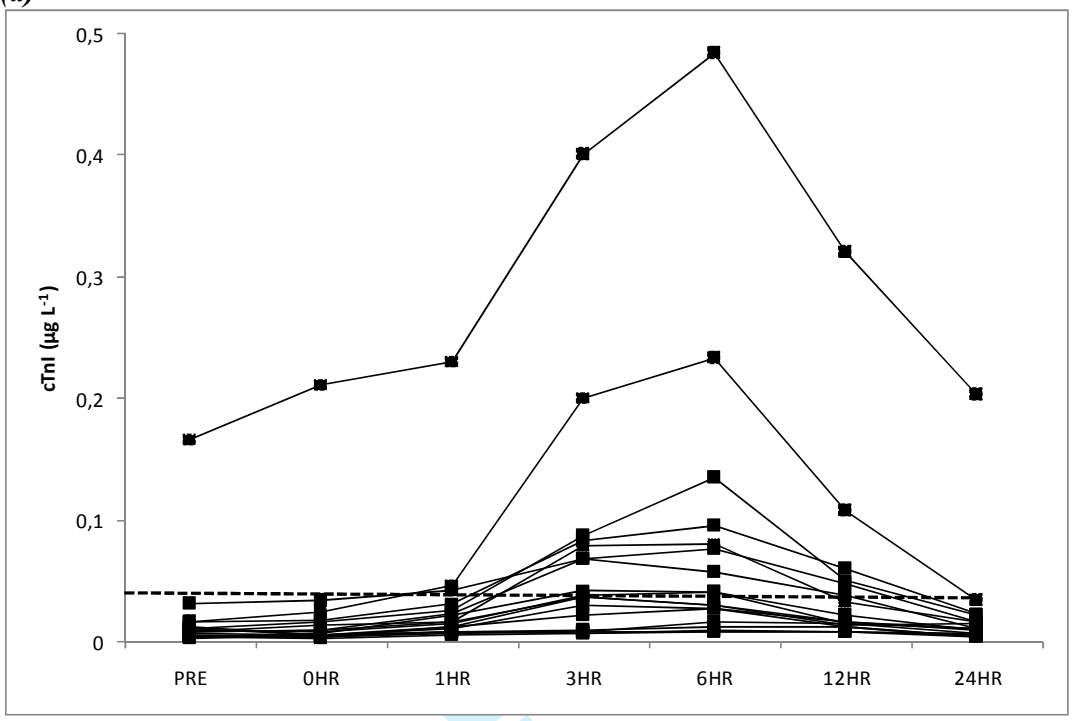
		Pre-exercise	5 min post	1 h post	3 h post	6 h post	12 h post	24 h post	*p* value		
									Time	Group	Time x Group
cTnI	Elite rowers	0.019 ± 0.038 (6)	0.022 ± 0.048 (6)	0.030 ± 0.051 (17)	0.069 ± 0.095 (44)	0.079 ± 0.116 (50)	0.045 ± 0.073 (28)	0.023 ± 0.046 (6)	0.000	0.010	0.311
	Amateur rowers	0.008 ± 0.015 (7)	0.008 ± 0.013 (7)	0.011 ± 0.018 (7)	0.025 ± 0.028 (14)	0.028 ± 0.029 (21)	0.020 ± 0.019 (21)	0.007 ± 0.007 (7)			
NT-proBNP	Elite rowers	14 ± 11 (0)	25 ± 18 (0)	21 ± 16 (0)	19 ± 14 (0)	18 ± 12 (0)	18 ± 11 (0)	19 ± 14 (0)	0.001	0.322	0.171
	Amateur rowers	17 ± 12 (0)	25 ± 19 (0)	25 ± 17 (0)	26 ± 18 (0)	26 ± 18 (0)	28 ± 18 (0)	27 ± 17 (0)			

Note: Values are means ± standard deviations (elite rowers: *n* = 18; amateur rowers: *n* = 14). In brackets the percentage of subjects with serum

cardiac biomarkers exceeding the URL.

Fig. 1

(a)


0,5
0,4
0,3
0,2
0,1
0
cTnI (µg L-1)
PRE
0HR
1HR
3HR
6HR
12HR
24HR

(b)

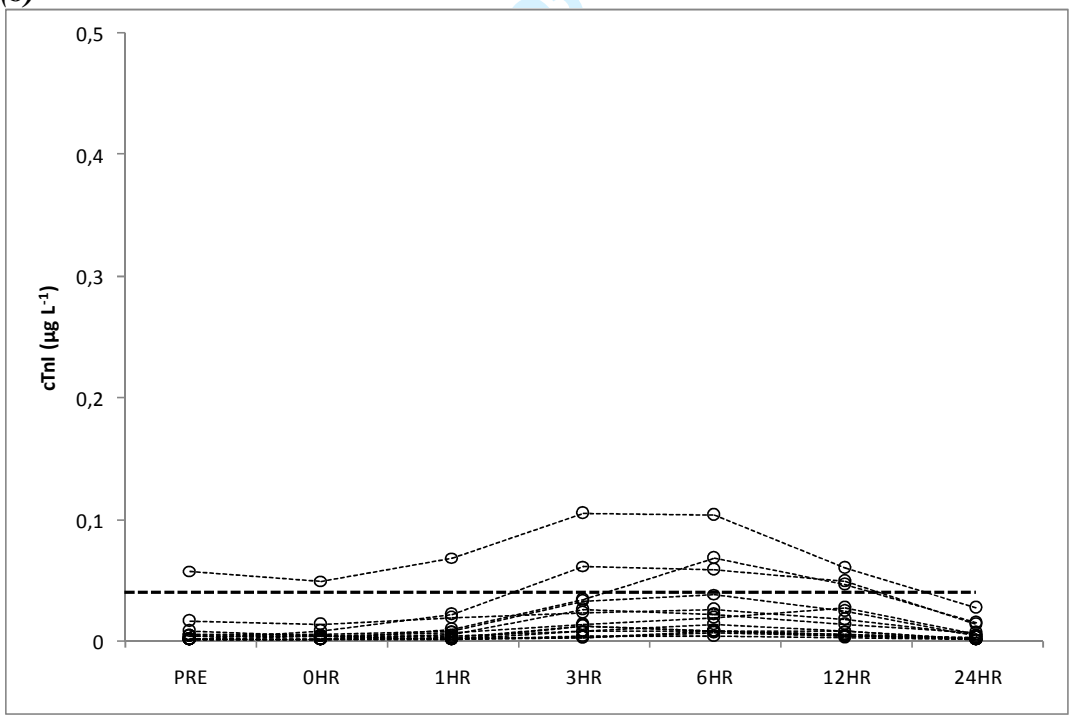
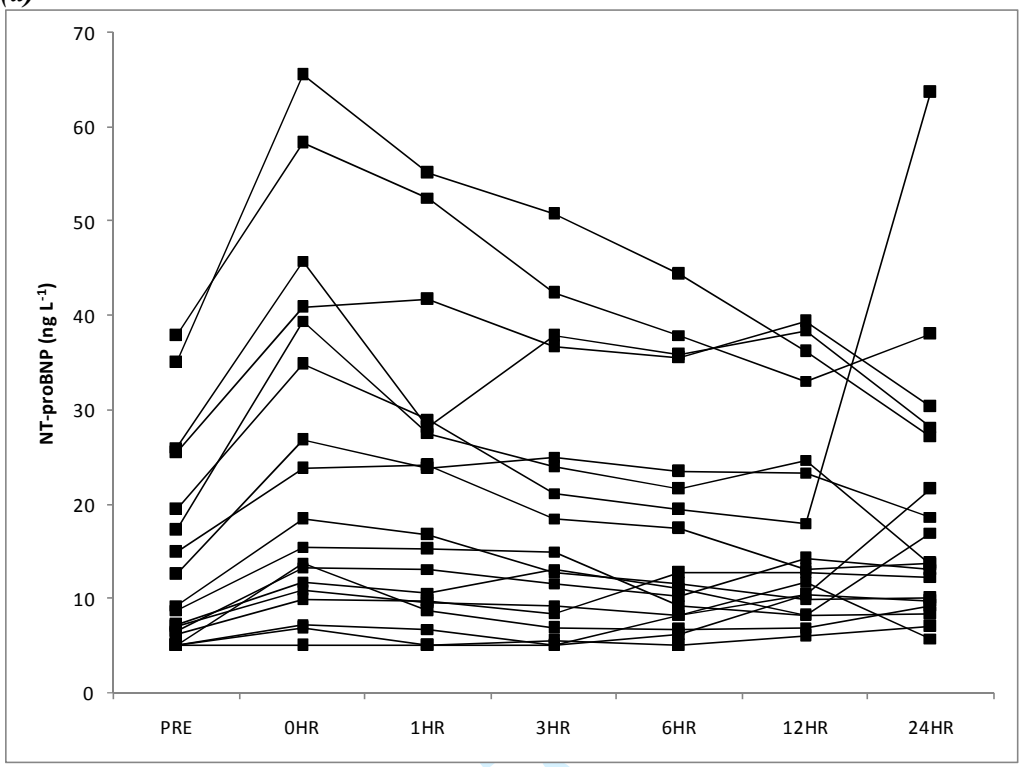
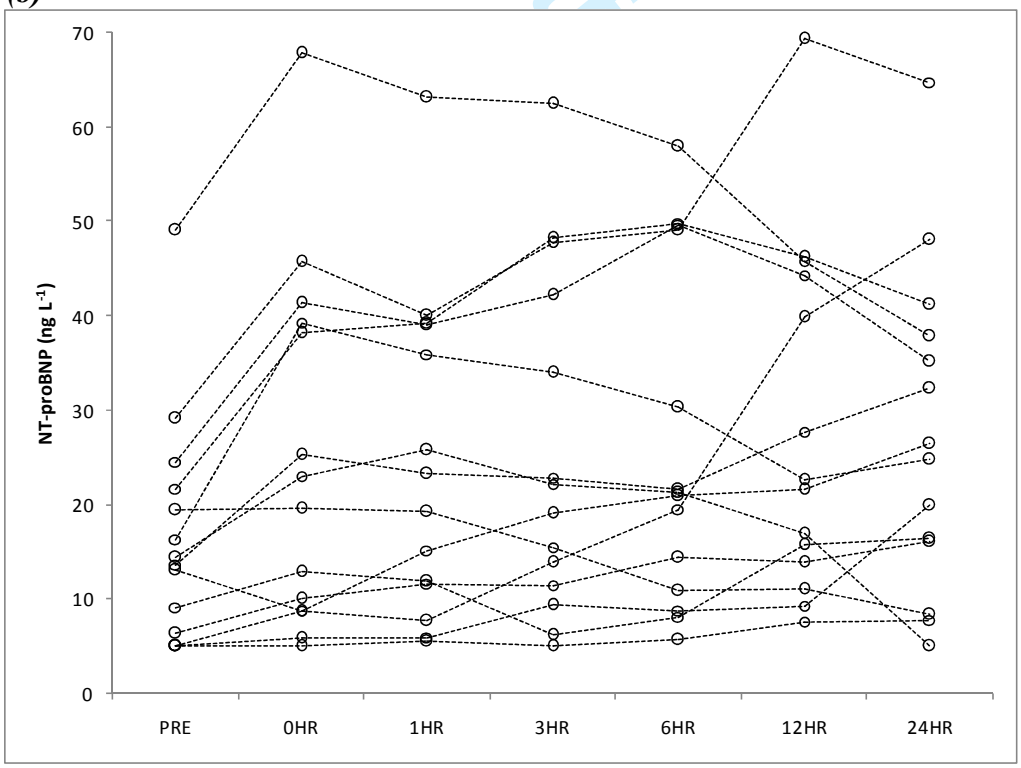

0,5
0,4
0,3
0,2
0,1
0
cTnI (µg L-1)
PRE
0HR
1HR
3HR
6HR
12HR
24HR

Fig. 2

(a)


NT-proBNP (ng L-1)
70
60
50
40
30
20
10
0
PRE
0HR
1HR
3HR
6HR
12HR
24HR

(b)


NT-proBNP (ng L-1)
70
60
50
40
30
20
10
0
PRE
0HR
1HR
3HR
6HR
12HR
24HR