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ORIGINAL RESEARCH

Differential cardiac biomarker responses to aerobic exercise in sedentary individuals and trained athletes

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Abstract

Acute aerobic exercise can increase cardiac troponin T (cTn-T) and B-type natriuretic peptide (BNP) concentrations in healthy individuals, but the influence of training status on the magnitude of this response remains unclear. This study compared acute exercise-induced cTn-T and BNP responses in 60 healthy men aged 18–25 years, including 30 professional football players and 30 sedentary individuals. Participants completed 45 min of outdoor aerobic running at 50–80% of age-predicted maximal heart rate, with heart rate continuously monitored. Venous blood was obtained immediately before and after exercise, and serum cTn-T and BNP were measured using commercial ELISA kits. Within-group pre- to post-exercise changes were assessed with paired-samples t-tests, and individual relative changes were compared between groups using independent-samples t-tests. cTn-T increased significantly in both trained (36.70 ± 4.1 to 41.9 ± 4.2 ng/L, $p < 0.0001$) and sedentary participants (31.87 ± 2.1 to 48.34 ± 2.8 ng/L, $p < 0.0001$); the mean individual relative increase was greater in the sedentary group (59.7% vs. 19.6%, $p = 0.002$). BNP also increased significantly in both groups, with a greater relative increase in sedentary participants (39% vs. 19%, $p < 0.05$). These findings indicate an attenuated acute biomarker response in trained participants, which may reflect training-associated cardiovascular adaptation. Because only immediate post-exercise samples were obtained and research-use ELISA assays were used, the findings do not establish biomarker kinetics or equivalence to clinical diagnostic thresholds. The results may help contextualize exercise-related biomarker changes in sports-science research.

Introduction

Exercise and physical activity are widely accepted as cornerstone recommendations in clinical medicine and sports sciences for maintaining health and improving overall well-being (Sietsema et al., 2020). The exercise-induced increase in metabolic demands requires appropriate cardiovascular and respiratory responses to sustain physiological homeostasis (Ward, 2018; Neder et al., 2018; Whipp et al., 2007).

Due to the increased myocardial workload during exercise, cardiac biomarkers such as B-type natriuretic peptide (BNP) and cardiac troponins may provide useful information regarding exercise-induced hemodynamic and myocardial stress (Hamasaki, 2016; Aengevaeren et al., 2021).

BNP is a cardiac-derived peptide hormone that plays a crucial role in regulating both blood volume and pressure through its vasodilatory effects. Circulating BNP concentrations have been shown to increase with the functional severity of heart failure in patients with underlying cardiac disease (Mueller et al., 2019). Beyond its role as a marker in myocardial pathology, BNP is also involved in cardiovascular homeostasis, energy expenditure, and fat metabolism (Hamasaki, 2016).

Aerobic and/or resistance exercise has been shown to increase BNP concentrations in individuals without

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heart disease (Ohba et al., 2001). During exercise, increased intraventricular pressure and ventricular wall stretch are considered potential stimuli for BNP release.

Exercise-associated changes in cardiac troponin concentrations can also provide information about myocardial stress (Aengevaeren et al., 2021; Stavroulakis & George, 2020). Cardiac troponins (cTn), which are integral to cardiomyocyte contraction, can now be detected at low circulating concentrations. Exercise-induced cTn elevation has therefore received increasing attention in both sports science and clinical research (Thygesen et al., 2018; Newby et al., 2012; Jakubiak, 2024).

However, whether regular endurance training modifies the acute response of these biomarkers during exercise remains unclear. Therefore, this study aimed to compare cTn-T and BNP responses to acute aerobic exercise in sedentary individuals and trained athletes and to examine whether training status is associated with differences in the magnitude of these acute biomarker responses.

Methods

Study Design and Ethical Approval

This study included 60 male participants, 30 sedentary and 30 trained, matched for age, height, weight, and body mass index (BMI). Ethical approval for the study was obtained from the Fırat University Non-Interventional Research Ethics Committee (approval no.: 05/20/14). All participants provided written informed consent after being thoroughly informed about the potential benefits and risks of the acute running exercise. The study was conducted in accordance with the principles of the Declaration of Helsinki.

Participants

Participants were male volunteers aged 18–25 years. Participants were classified into trained and sedentary groups according to habitual physical activity and sports participation. The trained group consisted of professional football players, whereas the sedentary group consisted of individuals who did not participate in regular exercise or organized sports.

Before enrollment, all participants underwent a medical examination by a physician to confirm their general health status and suitability for participation in the prescribed exercise protocol. Inclusion criteria required participants to be physically healthy and free of any acute illness that could influence exercise performance or study outcomes. Exclusion criteria included cardiovascular, respiratory, metabolic, or musculoskeletal disorders; acute or chronic diseases such as diabetes, obesity, or cardiac insufficiency; and regular use of medications, alcohol, or cigarettes. Participants with any medical condition considered incompatible with the exercise protocol during the physician examination were not included. Participants were instructed to refrain from substances that could affect exercise performance, including non-steroidal anti-inflammatory drugs (NSAIDs) and vitamin supplements, for two weeks before testing and to avoid caffeine and strenuous exercise for 24 h before the experimental session.

Sample Size and Statistical Power

No formal a priori sample-size calculation was performed before participant recruitment. The study included 60 eligible volunteers who met the predefined inclusion criteria and completed the experimental protocol, with 30 participants in each group. To evaluate the statistical sensitivity of the available sample, a sensitivity power analysis was performed for the between-group comparison of exercise-induced changes. With 30 participants per group, a two-sided alpha level of 0.05 and 80% statistical power, the study was capable of detecting an effect size of approximately Cohen's d = 0.74 or greater.

Exercise Protocol

Predicted maximal heart rate (HRmax) was estimated using the age-predicted equation HRmax = 220 − age, which was the equation prospectively used during the experimental sessions. The individual target heart-rate range was calculated as 50–80% of predicted HRmax (target HR = predicted HRmax × 0.50–0.80). Participants performed 45 min of outdoor aerobic running between 08:00 and 10:00 a.m. Heart rate was continuously monitored throughout the exercise session using a Polar T31-Coded heart-rate monitor to ensure that participants remained within the prescribed intensity range. The monitor was used for real-time intensity control; mean and peak heart rate, running distance, pace, and rating of perceived exertion were not systematically recorded as study variables.

Blood Collection and Biochemical Analysis

Blood samples (5 mL) were collected from the antecubital vein immediately before and immediately after the exercise session. All samples were centrifuged at 4500 rpm for 5 min at 4°C, and the obtained serum was stored at −80°C until analysis. Serum cardiac troponin T (cTn-T) concentrations were measured

using a commercially available Human Troponin T ELISA kit (SunRed Biological Technology Co., Ltd., Shanghai, China; catalog no.: 201-12-1255; detection range: 18–6000 ng/L; sensitivity: 15.125 ng/L). Serum BNP concentrations were measured using a Human BNP ELISA kit (SunRed Biological Technology Co., Ltd., Shanghai), according to the manufacturer's instructions.

Statistical Analysis

Statistical analyses were performed using IBM SPSS Statistics for Windows, Version 26.0 (IBM Corp., Armonk, NY, USA). Data are presented as mean ± standard error (SE). The normality of data distribution was assessed using the Kolmogorov–Smirnov test. Within-group changes in serum cTn-T and BNP concentrations from pre- to post-exercise were evaluated using paired-samples t-tests. To assess whether the magnitude of the exercise-induced response differed between trained and sedentary participants, the relative change was calculated separately for each participant as [(post-exercise value – pre-exercise value) / pre-exercise value] × 100. These individual percentage changes were subsequently averaged within each group and compared between the trained and sedentary groups using independent-samples t-tests. All tests were two-sided, and statistical significance was accepted at $p < 0.05$.

Results

Serum cTn-T concentrations increased significantly following acute exercise in both groups. In the trained group, cTn-T increased from 36.70 ± 4.1 ng/L at baseline to 41.9 ± 4.2 ng/L post-exercise ($p < 0.0001$). In the sedentary group, cTn-T increased from 31.87 ± 2.1 ng/L at baseline to 48.34 ± 2.8 ng/L post-exercise ($p < 0.0001$). When relative change was calculated separately for each participant and subsequently averaged within each group, the mean individual percentage increase in cTn-T was 19.6% in the trained group and 59.7% in the sedentary group. The exercise-induced relative increase in cTn-T was significantly greater in the sedentary group than in the trained group ($p = 0.002$).

Serum BNP concentrations also increased significantly following acute exercise in both groups. In the trained group, BNP increased from 2.20 ± 0.11 pg/mL at baseline to 2.61 ± 0.14 pg/mL post-exercise ($p < 0.05$). In the sedentary group, BNP increased from 2.00 ± 0.09 pg/mL at baseline to 2.73 ± 0.10 pg/mL post-exercise ($p < 0.05$). Baseline BNP concentrations did not differ significantly between the trained and sedentary groups ($p = 0.26$). When relative change was calculated separately for each participant and subsequently averaged within each group, the mean individual percentage increase in BNP was 19% in the trained group and 39% in the sedentary group. The exercise-induced relative increase in BNP was significantly greater in the sedentary group than in the trained group ($p < 0.05$).

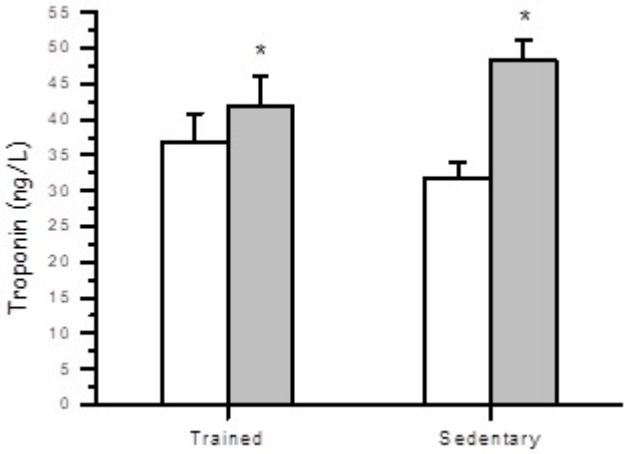


Figure 1. Serum cTn-T concentrations before and immediately after acute exercise in the trained and sedentary groups. White bars represent pre-exercise values, and gray bars represent post-exercise values. *p < 0.05 versus the corresponding pre-exercise value.

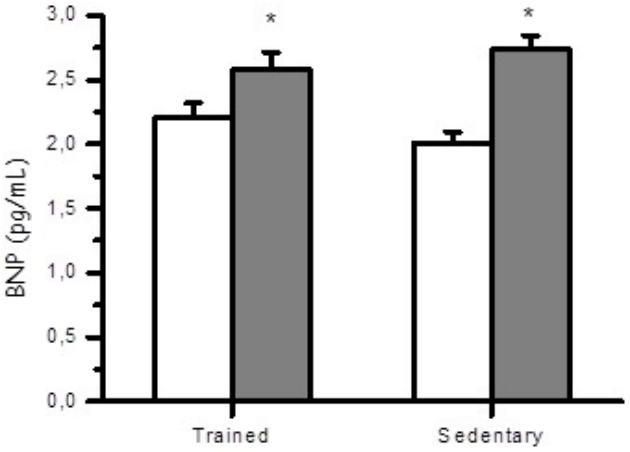


Figure 2. Serum BNP concentrations before and immediately after acute exercise in the trained and sedentary groups. White bars represent pre-exercise values, and gray bars represent post-exercise values. *p < 0.05 versus the corresponding pre-exercise value.

Discussion

The present study showed that acute exercise was associated with significant increases in both cardiac troponin T (cTn-T) and B-type natriuretic peptide (BNP) concentrations in trained and sedentary

individuals. The magnitude of these responses was greater in sedentary participants, suggesting that training status is associated with the acute cardiac biomarker response to exercise.

Consistent with previous literature, circulating cTn concentrations increased following acute exercise in both groups. Exercise-associated cTn elevations have been reported in healthy individuals and may occur in the absence of clinically apparent myocardial injury (Aengevaeren et al., 2021). The greater relative increase observed in sedentary individuals may indicate a greater biomarker response to the same prescribed exercise-intensity range; however, the present study did not directly measure myocardial injury, cardiac function, or exercise-load equivalence between groups.

Several mechanisms have been proposed to explain exercise-associated cTn release, including increased cardiomyocyte membrane permeability, release from the cytosolic troponin pool, and non-necrotic cellular stress (Hammarsten et al., 2018). In trained individuals, attenuated cTn release may be related to improved myocardial perfusion, antioxidant capacity, or reduced mechanical strain, although these mechanisms were not directly measured in the present study. Our findings are consistent with reports of measurable cTn increases after physical activity in healthy populations (Clerico et al., 2024). Because blood was sampled only immediately after exercise, the present data cannot establish the duration, peak, or reversibility of the observed cTn response. During exercise, increased heart rate and stroke volume alter myocardial oxygen consumption, coronary blood flow, preload, and afterload (Diaz-Garzon et al., 2021), and these hemodynamic and metabolic changes may contribute to exercise-associated cTn release (Celeski et al., 2024).

The greater relative increase in cTn-T observed in sedentary participants may reflect differences in physiological adaptation to exercise. Trained individuals generally exhibit cardiovascular adaptations that can reduce relative physiological strain during exercise, whereas sedentary individuals may experience a greater acute cardiovascular demand at a given prescribed relative intensity (Aengevaeren et al., 2021). Nevertheless, the present protocol did not include direct measures of cardiac function or detailed internal and external exercise load; therefore, the mechanisms underlying the between-group difference cannot be established from these data.

One proposed mechanism for exercise-associated cTn release is a transient increase in cardiomyocyte membrane permeability. Mechanical and metabolic stress may temporarily alter the sarcolemmal membrane and facilitate release of a small cytosolic troponin pool (Hammarsten et al., 2018). This mechanism has been proposed to occur without irreversible myocyte necrosis; however, the present study was not designed to distinguish among possible mechanisms of troponin release.

The cytosolic troponin pool may also contribute to circulating concentrations. A small fraction of total cellular troponin is present in a soluble cytosolic form and may be released more readily under conditions of increased membrane permeability and cellular stress (Wu et al., 2018). This mechanism has been proposed as one explanation for early post-exercise increases in circulating troponin.

Subclinical cardiomyocyte stress or injury has also been discussed in the context of prolonged or high-intensity endurance exercise. Mild myofibrillar disruption and other reversible cellular changes have been proposed as potential contributors to post-exercise cTn elevations, particularly after prolonged endurance events (Shave et al., 2010). These observations from the literature should not, however, be interpreted as evidence that the elevations observed in the present study were necessarily reversible or clinically benign.

Mechanical factors such as myocardial wall stress and stretch may also contribute. Exercise-induced changes in venous return, preload, and afterload can increase ventricular wall tension and activate stretch-responsive pathways (Middleton et al., 2008; Donaldson et al., 2019).

Oxidative and metabolic stress during exercise may further influence cardiomyocyte membrane integrity. Increased production of reactive oxygen species during intense or prolonged exercise has been proposed as one contributor to transient cellular alterations and biomarker release (Powers et al., 2011).

Alterations in renal hemodynamics during exercise may also influence circulating troponin concentrations. Redistribution of blood flow during exercise can transiently alter renal clearance and may therefore affect biomarker kinetics (Eijsvogels et al., 2015).

Taken together, these mechanisms provide a plausible framework for exercise-associated cTn elevation (Baker et al., 2019; Conesa-Milian et al., 2023). However, the present design does not permit classification of the observed elevations as physiological or pathological, nor does it establish their clinical significance. In addition, values generated with a research-use ELISA assay should not be interpreted

against clinical diagnostic thresholds without evidence of analytical equivalence.

BNP concentrations also increased significantly in both groups following acute exercise, with a greater relative increase in sedentary participants. BNP release is influenced by myocardial wall stress and ventricular stretch, which vary with preload and afterload. The larger BNP response in sedentary individuals may therefore reflect a greater acute hemodynamic response to the exercise bout, although cardiac loading conditions were not measured directly in this study (Hamasaki, 2016; Ohba et al., 2001).

The attenuated BNP response observed in trained individuals may be consistent with structural and functional adaptations associated with regular endurance training, including greater ventricular compliance, improved diastolic function, and more efficient stroke-volume responses. Such adaptations could reduce relative hemodynamic stress during exercise; however, these mechanisms were not directly assessed in the present study (Eijsvogels et al., 2015; König et al., 2007).

The observed increases in cTn-T and BNP are consistent with exercise-associated biomarker responses described in healthy and athletic populations. Because only immediate post-exercise samples were obtained, the present study cannot determine peak concentrations, recovery kinetics, or whether the elevations were transient and reversible. Moreover, published findings obtained with clinical or high-sensitivity assays should not be assumed to be analytically equivalent to the research-use ELISA measurements used here (Janssen et al., 2024).

Taken together, the combined evaluation of cTn-T and BNP provides complementary information about the acute biomarker response to exercise. The attenuated responses observed in professional football players are consistent with training-associated cardiovascular adaptation, but the cross-sectional design does not establish causality or a protective effect of training.

Limitations

This study has several limitations. The sample was limited to 60 young male participants, which restricts generalizability to women and other age groups. Blood sampling was limited to pre-exercise and immediately post-exercise; therefore, peak biomarker concentrations, recovery kinetics, and the duration or reversibility of the responses could not be determined. Exercise intensity was prescribed using age-predicted rather than directly measured HRmax. Detailed training history (e.g., years of professional participation and weekly training volume) and formal physical-fitness measures were not systematically recorded, limiting quantitative characterization of training status.

The prescribed exercise-intensity range was relatively broad, and mean and peak heart rate, running distance, pace, and perceived exertion were not systematically recorded; consequently, the internal and external exercise load could not be characterized in detail. In addition, cTn-T and BNP were quantified using research-use ELISA assays. Therefore, the absolute concentrations reported in this study should not be considered analytically equivalent to values generated by standardized clinical cardiac biomarker assays or interpreted directly against clinical diagnostic thresholds.

Conclusion

Acute aerobic exercise was associated with significant increases in cTn-T and BNP concentrations in both trained and sedentary individuals, with greater relative increases in sedentary participants. These findings suggest that training status is associated with the magnitude of the acute cardiac biomarker response and are consistent with training-associated cardiovascular adaptation. Because samples were obtained only immediately after exercise, peak concentrations, recovery kinetics, and reversibility cannot be determined. In addition, values generated by research-use ELISA assays should not be interpreted against clinical diagnostic thresholds without evidence of analytical equivalence. Future studies incorporating serial post-exercise sampling, directly measured exercise intensity, and detailed training and fitness characterization are warranted.

Ethical Approval

The study was approved by the Fırat University Ethics Committee for Non-Interventional Research (Approval Number: 2014/05/20) and was conducted in accordance with the principles of the Declaration of Helsinki.

Author Contributions

Study Design: SA, SBB; Data Collection: SA, OO; Statistical Analysis: SA, SBB; Manuscript Preparation: SA, SBB; Funds Collection: SA, SBB.

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Conflict of Interest

The authors declare that they have no conflicts of interest regarding the publication of this study.

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