

The Impact of HIIT on the cTnT Response in Sedentary Obese Young Men

Behroz Khodayari¹, Mohammad Reza Dehghan^{2*}, Reza Nouri³, Abbas Ali Gaeini⁴,
Mohammad Reza Kordi⁵

¹Assistant Professor of Department of Physical Education, Ahvaz Branch, Islamic Azad University, Ahvaz, Iran.

²Instructor of Department of Exercise Physiology, Shahid Chamran University of Ahvaz, Ahvaz, Iran.

³Assistant Professor of Department of Sport Sciences, Kish International Campus, University of Tehran, Kish, Iran.

⁴Professor of Department of Exercise Physiology, Faculty of Physical Education and Sport Sciences, University of Tehran, Tehran, Iran.

⁵Associate Professor of Department of Exercise Physiology, Faculty of Physical Education and Sport Sciences, University of Tehran, Tehran, Iran.

Abstract

Objective: Evaluation of the effect of 8 weeks of High-Intensity Interval Training (HIIT) on the serum level of cardiac troponin T (cTnT) in sedentary obese young men.

Materials and Methods: Thirty sedentary men were randomly divided into 3 groups: the 30s HIIT training group (n=10), the 60s training group (n=10), and the control group (no exercise) (n=10). Interval training with 90 %VO₂peak was done in 3 sessions for 8 weeks. cTnT was measured 5 times; pre-exercise, 4hrs, and 24hrs after the first session (4hF, 24hF), and 4hrs and 24hrs after the last training session (4hL, 24hL). ANOVA with repeated measures and Bonferroni *post-hoc* tests were used by SPSS 23 with a significance level of ($P < 0.05$).

Results: The significant increase in serum levels of cTnT in the post-tests of the 60s HIIT compared to the first 24hrs and the last 24hrs ($P = 0.0001$). But in the 30s group it was not significant compared to the other measurements ($P \geq 0/05$). In the 2 training groups, serum levels of cTnT in the last 4hr post-tests of the last week were not significantly different from the 4hrs post-tests of the first week ($P \geq 0.05$).

Conclusion: It seems that none of these activities cause heart damage sustained, and changes in cTnT levels in two 4hrs compared to the first and last 24hrs of the 60s group may be due to reversible leakage of cardiac cell membranes, and may continuous exercise will reduce this reduction.

Keywords: Cardiac biomarker, Cardiac troponin T, High-intensity interval training

QR Code:



Citation: Khodayari B, Dehghan M R, Nouri R, Gaeini A A, Kordi M R. The Impact of HIIT on the cTnT Response in Sedentary Obese Young Men. IJDO. 2022; 14 (3) :167-175

URL: <http://ijdo.ssu.ac.ir/article-1-730-en.html>

 10.18502/ijdo.v14i3.10743

Article info:

Received: 12 May 2022

Accepted: 24 July 2022

Published in September 2022



This is an open access article under the (CC BY 4.0)

Corresponding Author:

Mohammad Reza Dehghan, Instructor of Department of Exercise Physiology, Shahid Chamran University of Ahvaz, Ahvaz, Iran.

Tel: (98) 912 943 8932

Email: yadni500@gmail.com

Orcid ID: 0000-0003-2395-9292

Introduction

High-intensity interval training (HIIT), is generally defined as repeated short to middle-time training (several seconds to minutes) that is in high intensity more than the anaerobic threshold, and the repeating of them is separated by relaxing periods or low-intensity activities (1,2).

This kind of exercise training, like other traditional continuous exercise training, could improve the functional and physiological capacities of some organs by improving the structural and biochemical compatible adaptations (3-6). HIIT is more contribute than a continuous exercise in decreasing fat (7). So, many exercise trainings are recommended for weight loss. For instance, Muller et al. (2020) showed that HIIT-based concurrent training programs involving traditional strength training vs. power training are equally effective in improving functionality, cardiorespiratory fitness, and body composition in healthy older men (8). It seems that the absolute risk of HIIT for cardiovascular disease and musculoskeletal injury is low (9). Most studies have shown that doing 30s intervals compared to 60 and 90s intervals in cardiovascular patients leads to feeling more comfortable (10,11) and a lower increase in lactic acid aggregation has been seen (12).

It is shown that the contribution of produced energy from oxidation in HIIT training is more than 50% of total energy (13), and these trainings resulted in cardiovascular pressure (14). It is caused by changes in some parameters such as preload, afterload, and heart contraction (15).

Cardiac troponin T (cTnT) is a part of regulative proteins that is constituted as a part of contradicting cardiac cells and one of the cardiac cell necrosis indexes, and it is observed that its serum levels increased following long time and intensive exercise trainings (16,17). Although some studies also indicated that endurance exercises lead to an increase in the serum levels of cTnT as well

(18,19). The response of cardiac index to high-intensity and short-time activities is unknown yet. Among the few researchers, Fu et al. (2009) studied cTnT response to the intensity of trainings, and it showed that time and intensity of exercises had notable effects on the releasing of cTnT in serum, and the intensity of exercises is more effective compared to the time (14). Physical pressure during interval training resulted in releasing cTnT in serum (20). Although, no increase in the serum levels of cTnT has to be seen following the interval and continuous activities in cardiovascular patients (21). George et al. (2004) studied the release of cTnT in adult players in rugby and American football and indicated that cTnT level was not accountable immediately and 24hr after the game (22). In one study it has been shown that cTnT concentration was at a maximum level of 4 and 24 hours after exercise activities, and then it will come back to base level (19).

The increase of this index following the exercises causes researchers to attempt to focus on clarifying this index. The mechanism of releasing cardio cTnT following high-intensity and long-term trainings is unknown yet, although, many studies regarding cardio cell injury in response to exercise in athletes, overweight men, and cardiovascular patients after one session of training have been done. Overweighing is one of the most popular risk factors for cardiovascular disease (23). The prevalence of cardiovascular disease in developed and developing countries is increasing (24). It is observed that in relaxing conditions, the risk factor was related to a high cTnT level (25). Some studies indicated that high Body mass index (BMI) might be related to high cTnT in relaxing conditions (26). Since research on the type of HIIT on cardiac indices is limited, this study tries to examine whether an 8-week HIIT program can influence cardiac cellular biochemical index in overweight men?

Materials and Methods

Participants in this quasi-experimental study consisted of 30 – 40 years old men from Ahvaz city. Qualified samples were selected based on the following criteria: no cardiovascular and musculoskeletal disorders, no regular exercise training, and no smoking. Thirty sedentary, healthy overweight men with BMI between 27 to 29 were selected and randomly divided into 3 groups (first group; control group, second group; 30s HIIT group, and third group; 60s HIIT group). For homogenization of 3 groups, they were managed based on aerobic fitness and BMI before the test, and it was determined that there was no significant difference. Before the experiment, all the participants were informed about trainings, procedure steps, and the aim of this study, then written consents were provided for them. Their daily diets were normal and regular. They filled out the food reminder questionnaire, 3 days before exercises and 3 days after the last session. We should notice that samples were determined by using the formula for determining the sample size in experimental studies, then taking into account the first type of error equal to 0.05, and 10 people in each group.

$$n = \frac{S_X^2 \times Z_{\alpha/2}^2}{D^2}$$

In this study, the height was measured by a tape measure (centimeter) and the weight was measured by a scale (Beurer model) (kilograms). BMI was measured using the following formula: weight divided by height squared (in meters). To determine the Vo₂peak and the related distance, every participant had to run, and an increasing test was done for one minute on treadmill (Ti51 Horizon Fitness USA). After starting the test at the speed of 10 km/h, after each minute, one km/h was added to the speed until fatigue. Vo₂peak was measured by a gas analyzer (Quark b2 Cosmed Co Italy). Criteria for determining Vo₂peak was no increase in oxygen consumption despite an increase in speed and an increase in heart rate of more

than 90% of the estimated maximum heart rate (220 -age) (27, 28) (Tables 3 and 4). The heart rate and distance for 2 training groups by the intensity of VO₂peak 90 % in the 30s and 60s training was determined. For preliminary preparedness and before the pilot test, all participants of 2 training groups were done continuous exercises for 10 minutes with an intensity of 50% VO₂peak for 3 sessions in a week. 48 hours after ending the training, one session was held to prepare them for the pilot test, and the frequency and sets for each group were determined. All participants of 2 training groups were done continuous exercises. It is necessary to mention that before the tests 2 training groups were done 10 minutes warming up, and in ending the training, they did 10 minutes cooling down.

Training programs

Group 1 did not receive any exercises. In the first session, group 2 used a polar watch on the left-hand wrist and ran in 3 repeated 8 sets, so they ran the determined distance in the 30s (heart rate was controlled). Between each repeat, participants relaxed for one minute (walking), and between each set, they rest for 4 minutes in inactive mode. Their walking speed is controlled by HR. Group 3 used the polar watch on the left-hand wrist and ran in 3 repeated 4 sets, so they ran the determined distance in the 60s (heart rate was controlled). Between each repeat, the participant relaxed (walking) for 2 minutes and between each set, they rest for 4 minutes in inactive mode. All the exercises continued 3 sessions a week for 8 weeks. The number of repetitions was increased gradually and according to the principle of overload (Table 1) (29-31).

Collecting the blood samples and measuring the index

Blood samples were taken as follows: before the pilot study, 4 and 24 hours after the first session, and 4 and 24 hours after the last session. It is necessary to mention that based on some reviews, the highlighted times were to recognize the cardiac injury resulting from

appropriate exercise training (20). 10 ml of blood was collected in sitting mode. After centrifugation for 10 min at a speed of 3000 rpm, serum was separated and shed in special microtubules and held at -80 °C. The cTnT level was measured by the LIAISON device and using the Chemiluminescence procedure. The natural range in this method was <0.01 n/ml.

Statistical analysis

The data distribution normality and the homogeneity of the variances were evaluated using the Shapiro-Wilk and Levon's test, respectively ($P \geq 0.05$). Afterward, mean statistical indices and standard deviation were used for descriptive data analysis, and ANOVA with repeated measure and Bonferroni post-hoc tests were used by SPSS 23 with a significance level of ($P < 0.05$) for all tests.

Ethical considerations

This study has been approved by the Ethics Committee of Shahid Chamran University of Ahvaz, Iran (Ethics code: EE/99.3.02.65864 /scu.ac.ir).

Results

The results of one-way ANOVA showed that there is no significant difference between the 3 groups for weight ($P = 0.150$), BMI ($P = 0.770$), and VO₂Peak ($P = 0.987$) in the pre-test that is shown in Table 2. The results of one-way ANOVA showed that there is not any significant differences between the 3 groups for weight in post-test ($P = 0.139$). The results of one-way ANOVA showed that there is a significant difference between the 3 groups for BMI in the post-test ($P = 0.0001$), and the Bonferroni post-doc test showed a significant decrease in the HIIT 30s ($P = 0.0001$) and HIIT 60s groups ($P = 0.0001$) compared to control the group. The results of one-way ANOVA showed that there is a significant differences between the 3 groups for VO₂Peak in the post-test ($P = 0.0001$), and the Bonferroni post-doc test showed a significant decrease in the HIIT 30s ($P = 0.0001$) and HIIT 60s groups ($P = 0.0001$) compared to the control group. The result of the one-way ANOVA for pre-test is shown in Table 3.

Repeated measures ANOVA showed a significant interaction between groups and time for cTnT ($P = 0.0001$, $F = 136.984$). To

Table 1. training protocols

Weeks	Groups	Sets per session	Repetitions per each set
1&2 the week	3	3	8
	4	3	4
3&4 the week	3	3	9
	4	3	5
5&6 the week	3	3	10
	4	3	6
7&8 the week	3	3	11
	4	3	7

Table 2. Characteristics of subjects before training

Statistic Groups	N	Age	Weight	BMI	VO ₂ Peak
Control	10	35.4 (±1.78)	85.04 (±3.07)	28.43 (±0.44)	34.24 (±0.75)
HIIT 30s	10	35.4 (±2.66)	86.49 (±4.56)	28.44 (±0.49)	34.24 (±0.18)
HIIT 60s	10	35.3 (±2.41)	87.17 (±3.61)	28.33 (±0.35)	34.21 (±0.31)
<i>P</i>	30	0.994	0.150	0.770	0.987

Table 3. Characteristics of subjects after 8 weeks of training

Statistic Groups	N	Weight	BMI	VO ₂ Peak
Control	10	84.68 (±2.96)	28.44 (±0.41)	34.17 (±0.75)
HIIT 30s	10	82.54 (±4)	27.18 (±0.46)	39.36 (±0.47)
HIIT 60s	10	82.98 (±4.37)	26.95 (±0.46)	39.5 (±0.31)
<i>P</i>	30	0.139	0.0001	0.0001

determine the differences among these 4 tests (4 and 24 hours after the first and last session), ANOVA with repeated measures was applied separately. Sequential tests showed a non-significant difference between pre and post-tests and post-tests together in the 30s groups ($P \geq 0.05$). There were significant differences between the first and the last 4 hours post-test, with pre-test and 24 hours post-tests in the 60s group ($P = 0.0001$). Also, there was no significant difference between 24 hours and pre-test in the 60s group ($P \geq 0.05$). There was no significant difference between 4 hours post-test in first and last session in the 60s group ($P \geq 0.05$). (Figure 1)

Discussion

The results showed that in the post-tests cTnT levels were more than the natural levels, and in the 60s HIIT group, this was higher than in other groups. Against the other clinical myocardial failure studies in which the peak of serum cTnT levels between the first and third day after the myocardial injury was irreversible (32), the result of this study showed that serum levels of cTnT 4 hours after HIIT reached its peak and after 24 hours it came back to the natural level. This result was similar to the study by Legaz et al. (2011), Tian et al. (2006), and Shave et al. (2004) who studied the serum levels of troponin in interval

training, male runners, and scullers (15,19,33). However, our results were not similar to the results of Faramarzi et al. (2007) and König et al. (2003). They found that one session exercise training had not any significant effect on the troponin level (34,35).

They just collected blood samples before and after exercise training. Although the main reason for these differences in troponin changes is not known, maybe the reason for this change was the different types of exercise and physical fitness of participants. Each of these trainings requires specific physiological demand, and heart tissue experience a variety of conditions for different metabolic to prepare their needs. The time of measurement was also effective as the peak of the troponin level was 3 to 6 hours after exercise training (20).

The quick return of serum levels of cTnT to the basic level supported the previous theory that indicated releasing troponin following high-intensity training might show the high diffusion of cytosolic from heart cell membrane injury and did not relate to necrosis of heart mycosis (33). Our results showed that 60s HIIT activities like continuous activities (19) resulted in high pressure on the heart and released the serum levels of cTnT, but not for 30s HIIT. This new finding supported the relationship between the intensity of exercise training and releasing of troponins (36). The

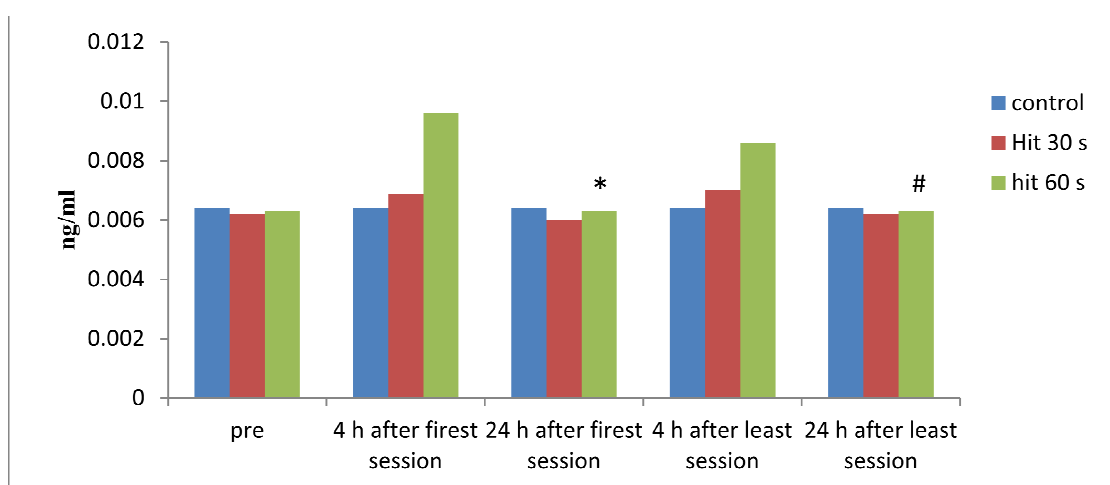


Figure 1. Levels of cTnT in control and training groups (pretest, posttest; 4hr and 24hr after the first session and the last session). * and # indicated differences between two 4hrs post-test of the 60s HIIT compared to the first 24hrs and the last 24hrs, respectively.

intensity of exercise training is one of the most strong predictive factors in increasing the cTnT levels (37).

Our results were not similar to the results of the studies by George et al. (2004) and Rejaee et al. (2010) who reported that troponin levels in men players after American football and active men were not increased (22,38). They evaluated blood sample of participants immediate and 24 hours after training while Legaz et al. (2015) observed that 3 to 6 hours after the exercise training, the troponin level was in peak (36). The increase in heart exercise training could temporally accelerate the apoptosis process and myocardial recovery (39). The heart injury at a low level and following recovery is a part of natural myocardia, and it leads to a temporal increase of troponins level after any exercise trainings, this increase could be assigned to kidney disorders for purifying the troponin. During exercise training, the circulation in the abdominal region and kidney decreased (40,41).

Then the capacity of the kidney to secrete substances will be reduced, and will probably lead to a slight increase in cTnI serum concentrations (41). However, the diffusion mechanism of cTnT is unknown. The previous studies (17,42,43) indicated different mechanisms such as increasing the permeability of the myocardial membrane and pressure on myositis (44,45), increasing the production of free radicals (46), or changing the base–acid balance (47). Our results showed that the increase of cTnT level in the last session after 8 weeks of training was lower than in the first session, and this result was more obvious in the 60s interval group. This finding was similar to the study results by Fortescue et al. (2007) and Mahta et al. (2012) who reported that releasing troponins after exercise in sedentary people was more than in others (48,49).

This result could be obtained by the adaptation of myocardial cells with exercise programs and lead to high-intensity activities in the future (48,49). With repeating training,

skeletal muscle fibers are subjected to stress (50) that is caused muscle fiber recovery and connective tissue changes; therefore, skeletal muscles are protected against more efforts (50). If it is for the heart as well, the lower level of cTnT in serum after exercise programs are justifiable (49). Legaz et al. (2015) reported different results and found that troponin levels of participants were higher than new ones before and after exercise trainings (36). On other hand, no significant relation was reported between training levels and releasing the troponins by Eijssvogels et al. (2015) (51). This contradiction might be related to exercise procedures and the frequencies of blood samples in recovery. It is observed that the increased level of cTnT in the 30s HIIT was lower than in other groups and was not significant.

This condition was brought by less pressure on the heart in this kind of exercise training, this result was similar to the results of Normandin et al. (2013), who studied the acute response to 30s interval training in heart failure patients (21). It is indicated that the heart patients felt more relaxed in short time interval exercise training (the 30s with inactive recovery) compared to the long term intervals (60s and 90s) (10,11). In sedentary people, 30s HIIT resulted in less aggregation of lactic acid (12). It seems that performing both HIITs (the 30s and 60 s) has the same effect on cTnT levels in obese men, and these 2 intensities affected this regulative protein after 8 weeks. It is recommended that a similar study could be performed on women, and other factors related to heart function in normal or obese individuals could be measured as well.

Conclusions

Generally, this study indicated although the increase of serum cTnT in two 4hrs compared to the first and last 24hrs of the 60s group was higher, after 24 hours, its level came back to base level. None of the both HIITs (the 30s and 60 s) in overweight men did change constantly cTnT. However, future searches can

study the exact mechanism of cardiac troponin diffusion.

Acknowledgments

This study was conducted at the expense of the researchers.

Funding

There is no funding.

Conflict of Interest

The authors declare that there is no conflict of interest.

References

- Gibala MJ, McGee SL. Metabolic adaptations to short-term high-intensity interval training: a little pain for a lot of gain?. *Exercise and sport sciences reviews*. 2008;36(2):58-63.
- Briand J, Tremblay J, Thibault G. Can Popular High-Intensity Interval Training (HIIT) Models Lead to Impossible Training Sessions?. *Sports*. 2022 ;10(1):10.
- Burgomaster KA, Howarth KR, Phillips SM, Rakobowchuk M, MacDonald MJ, McGee SL, et al. Similar metabolic adaptations during exercise after low volume sprint interval and traditional endurance training in humans. *The Journal of physiology*. 2008;586(1):151-60.
- Burgomaster KA, Heigenhauser GJ, Gibala MJ. Effect of short-term sprint interval training on human skeletal muscle carbohydrate metabolism during exercise and time-trial performance. *Journal of applied physiology*. 2006;100(6):2041-7.
- Burgomaster KA, Hughes SC, Heigenhauser GJ, Bradwell SN, Gibala MJ. Six sessions of sprint interval training increases muscle oxidative potential and cycle endurance capacity in humans. *Journal of applied physiology*. 2005;98(6):1985-90.
- Yakut H, Dursun H, Felekoğlu E, Başkurt AA, Alpaydın AÖ, Özalevli S. Effect of home-based high-intensity interval training versus moderate-intensity continuous training in patients with myocardial infarction: a randomized controlled trial. *Irish Journal of Medical Science (1971-)*. 2022:1-0.
- Bartels MN, Bourne GW, Dwyer JH, Sandel ME. High-Intensity Exercise for Patients in Cardiac Rehabilitation After Myocardial Infarction. *PM&R*. 2010;2(2):151-5.
- Müller DC, Boeno FP, Izquierdo M, Aagaard P, Teodoro JL, Grazioli R, et al. Effects of high-intensity interval training combined with traditional strength or power training on functionality and physical fitness in healthy older men: A randomized controlled trial. *Experimental Gerontology*. 2021;149:111321.
- Quindry JC, Franklin BA, Chapman M, Humphrey R, Mathis S. Benefits and risks of high-intensity interval training in patients with coronary artery disease. *The American journal of cardiology*. 2019;123(8):1370-7.
- Guiraud T, Juneau M, Nigam A, Gayda M, Meyer P, Mekary S, et al. Optimization of high intensity interval exercise in coronary heart disease. *European journal of applied physiology*. 2010;108(4):733-40.
- Meyer KA, Samek LA, Schwaibold MA, Westbrook SA, Hajric RA, Beneke RA, et al. Interval training in patients with severe chronic heart failure: analysis and recommendations for exercise procedures. *Medicine and science in sports and exercise*. 1997;29(3):306-12.
- Gosselin LE, Kozlowski KF, DeVinney-Boymel L, Hambridge C. Metabolic response of different high-intensity aerobic interval exercise protocols. *The Journal of Strength & Conditioning Research*. 2012;26(10):2866-71.
- Billaut F, Bishop D. Muscle fatigue in males and females during multiple-sprint exercise. *Sports medicine*. 2009;39(4):257-78.
- Fu F, Nie J, Tong TK. Serum cardiac troponin T in adolescent runners: effects of exercise intensity and duration. *International journal of sports medicine*. 2009;30(03):168-72.
- Carranza-García LE, George K, Serrano-Ostáriz E, Casado-Arroyo R, Caballero-Navarro AL, Legaz-Arrese A. Cardiac biomarker response to intermittent exercise bouts. *International journal of sports medicine*. 2011;32(05):327-31.
- S Scharhag J, George K, Shave R, Urhausen A, Kindermann W. Exercise-associated increases in cardiac biomarkers. *Medicine and science in sports and exercise*. 2008;40(8):1408-15.
- Shave R, Baggish A, George K, Wood M, Scharhag J, Whyte G, et l. Exercise-induced cardiac troponin elevation: evidence, mechanisms, and implications. *Journal of the American College of Cardiology*. 2010;56(3):169-76.
- Middleton N, George K, Whyte G, Gaze D, Collinson P, Shave R. Cardiac troponin T release is stimulated by endurance exercise in healthy humans. *Journal of the American College of Cardiology*. 2008;52(22):1813-4.

19. Tian Y, Nie J, Tong TK, Cao J, Gao Q, Man J, et al. Changes in serum cardiac troponins following. *Journal of sports medicine and physical fitness*. 2006;46(3-4):481-8.
20. Nie J, Tong TK, Shi Q, Lin H, Zhao J, Tian Y. Serum cardiac troponin response in adolescents playing basketball. *International journal of sports medicine*. 2008;29(06):449-52.
21. Normandin E, Nigam A, Meyer P, Juneau M, Guiraud T, Bosquet L, et al. Acute responses to intermittent and continuous exercise in heart failure patients. *Canadian Journal of Cardiology*. 2013;29(4):466-71.
22. George KP, Dawson E, Shave RE, Whyte G, Jones M, Hare E, et al. Left ventricular systolic function and diastolic filling after intermittent high intensity team sports. *British journal of sports medicine*. 2004;38(4):452-6.
23. Gelber RP, Gaziano JM, Orav EJ, Manson JE, Buring JE, Kurth T. Measures of obesity and cardiovascular risk among men and women. *Journal of the American College of Cardiology*. 2008;52(8):605-15.
24. James WP. WHO recognition of the global obesity epidemic. *International journal of obesity*. 2008;32(7):S120-6.
25. Wallace TW, Abdullah SM, Drazner MH, Das SR, Khera A, McGuire DK, et al. Prevalence and determinants of troponin T elevation in the general population. *Circulation*. 2006;113(16):1958-65.
26. Saunders JT, Nambi V, De Lemos JA, Chambless LE, Virani SS, Boerwinkle E, et al. Cardiac troponin T measured by a highly sensitive assay predicts coronary heart disease, heart failure, and mortality in the Atherosclerosis Risk in Communities Study. *Circulation*. 2011;123(13):1367-76.
27. Dupont G, Blondel N, Lensel G, Berthoin S. Critical velocity and time spent at a high level of for short intermittent runs at supramaximal velocities. *Canadian journal of applied physiology*. 2002;27(2):103-15.
28. Asghari E, Damirchi A. A comparison between Effects Of high intensity and high volume training on lactate accumulation, time performance and vo2peak in 10-14 year old distance runners. *Journal of Sport Biosciences*. 2013;5(3):29-40.
29. Burgomaster KA, Hughes SC, Heigenhauser GJ, Bradwell SN, Gibala MJ. Six sessions of sprint interval training increases muscle oxidative potential and cycle endurance capacity in humans. *Journal of applied physiology*. 2005 .
30. Apple FS, Rogers MA, Sherman WM, Costill DL, Hagerman FC, Ivy JL. Profile of creatine kinase isoenzymes in skeletal muscles of marathon runners. *Clinical chemistry*. 1984;30(3):413-6.
31. Gibala MJ, Little JP, MacDonald MJ, Hawley JA. Physiological adaptations to low-volume, high-intensity interval training in health and disease. *The Journal of physiology*. 2012;590(5):1077-84.
32. Antman E, Bassand JP, Klein W, Ohman M, Lopez Sendon JL, Rydén L, et al. Myocardial infarction redefined-a consensus document of the Joint European Society of Cardiology/American College of Cardiology committee for the redefinition of myocardial infarction: the Joint European Society of Cardiology/American College of Cardiology Committee. *Journal of the American College of Cardiology*. 2000;36(3):959-69.
33. Shave R, Dawson E, Whyte GR, George KE, Gaze DC, Collinson PO. Altered cardiac function and minimal cardiac damage during prolonged exercise. *Medicine & Science in Sports & Exercise*. 2004;36(7):1098-103.
34. Faramarzi M, Gaeini A, Kordi M. Effect of intense interval physical activity and carbohydrate supplement on biomarkers of cardiac (cTnI, CK-MB) in soccer players. *Olympic*. 2007;15(3):35-44.
35. König D, Schumacher YO, Heinrich L, Schmid A, Berg AL, Dickhuth HH. Myocardial stress after competitive exercise in professional road cyclists. *Medicine and science in sports and exercise*. 2003;35(10):1679-83.
36. Legaz-Arrese A, López-Laval I, George K, Puente-Lanzarote JJ, Moliner-Urdiales D, Ayala-Tajuelo VJ, et al. Individual variability in cardiac biomarker release after 30 min of high-intensity rowing in elite and amateur athletes. *Applied Physiology, Nutrition, and Metabolism*. 2015;40(9):951-8.
37. Eijssvogels TM, Veltmeijer MT, George K, Hopman MT, Thijssen DH. The impact of obesity on cardiac troponin levels after prolonged exercise in humans. *European journal of applied physiology*. 2012;112(5):1725-32.
38. Rajaei F, Mojtahedi H, Akbari A, Marandi M. Comparison of effects of three types of endurance, resistance and combination training on (cTnT and CKMB) in active males. *British Journal of Sports Medicine*. 2010;44(Suppl 1):i21.
39. Beltrami AP, Barlucchi L, Torella D, Baker M, Limana F, Chimenti S, et al. Adult cardiac stem cells are multipotent and support myocardial regeneration. *cell*. 2003;114(6):763-76.
40. Thijssen D, Steendijk S, Hopman M. Blood redistribution during exercise in subjects with spinal cord injury and controls. *Medicine+ Science in Sports+ Exercise*. 2009;41(6):1249.
41. Qamar MI, Read AE. Effects of exercise on mesenteric blood flow in man. *Gut*. 1987;28(5):583-7.
42. Hickman PE, Potter JM, Aroney C, Koerbin G, Southcott E, Wu AH, et al. Cardiac troponin may be released by ischemia alone, without necrosis. *Clinica chimica acta*. 2010;411(5-6):318-23.

43. Koller A, Schobersberger W. Post-exercise release of cardiac troponins. *Journal of the American College of Cardiology*. 2009;53(15):1341.
44. McNeil PL, Khakee R. Disruptions of muscle fiber plasma membranes. Role in exercise-induced damage. *The American journal of pathology*. 1992;140(5):1097.
45. Legaz-Arrese A, George K, Carranza-García LE, Munguía-Izquierdo D, Moros-García T, Serrano-Ostáriz E. The impact of exercise intensity on the release of cardiac biomarkers in marathon runners. *European journal of applied physiology*. 2011;111(12):2961-7.
46. Goette A, Bukowska A, Dobrev D, Pfeiffenberger J, Morawietz H, Strugala D, et al. Acute atrial tachyarrhythmia induces angiotensin II type 1 receptor-mediated oxidative stress and microvascular flow abnormalities in the ventricles. *European heart journal*. 2009;30(11):1411-20.
47. Sahlin EH. Acid-base balance during exercise. *Exercise and sport sciences reviews*. 1980;8(1):41-128.
48. Fortescue EB, Shin AY, Greenes DS, Mannix RC, Agarwal S, Feldman BJ, et al. Cardiac troponin increases among runners in the Boston Marathon. *Annals of emergency medicine*. 2007;49(2):137-43.
49. Mehta R, Gaze D, Mohan S, Williams KL, Sprung V, George K, et al. Post-exercise cardiac troponin release is related to exercise training history. *International journal of sports medicine*. 2012;33(05):333-7.
50. Ebbeling CB, Clarkson PM. Exercise-induced muscle damage and adaptation. *Sports medicine*. 1989;7(4):207-34.
51. Eijsvogels TM, Hoogerwerf MD, Maessen MF, Seeger JP, George KP, Hopman MT, et al. Predictors of cardiac troponin release after a marathon. *Journal of science and medicine in sport*. 2015;18(1):88-92.