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REVIEW ARTICLE

Physical Exercise Increases Cardiac Troponin (cTn) Levels as a Marker of Myocardial Infarction: A Systematic Review

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Abstract

Objectives. This study aimed to analyze an indication of physical exercise on the increase of cardiac troponin (cTn) levels as a marker of myocardial infarction from the physiological perspective.

Materials and methods. A search of journal databases including MEDLINE-Pubmed, Web of Science, Scopus, and Science Direct was used in this systematic review study. The inclusion criteria were papers that addressed cardiac troponin (cTn), physical activity, and myocardial infarction, and were published within the last five years. Using the databases Science Direct, Pubmed, and Web of Science, 83 recognized publications were found. For this systematic review, finally ten publications that fulfilled the requirements for inclusion were chosen and examined. This study evaluated standard operating procedures using the Preferred Reporting Items for Systematic reviews and Meta-Analyses (PRISMA).

Results. This systematic review reports that physical exercise has been found to increase cardiac troponin (cTn) levels as a marker of myocardial infarction, but this increase is triggered by the body's physiological response during exercise and is not considered as a pathological response.

Conclusions. According to the findings of the study, physical exercise has been proven to elevate the risk of heart muscle damage by triggering an increase in cardiac troponin (cTn). However, this feature occurs in a physiological state and represents an exercise adaptation process. An analysis of the relevant articles reveals that this increase in cardiac troponin (cTn) has not been shown to have a negative influence on developing cardiovascular disease.

Keywords: cardiac troponin, physical exercise, myocardial infarction, healthy lifestyle.

Introduction

Regular physical exercise is strongly connected to better human life expectancy and has an impact on reducing morbidity, as well as mortality in cardiovascular disease (Ekelund et al., 2019). Regular exercise with moderate intensity is proven as a healthy lifestyle that can improve the health status of the human body (Bull et al., 2020). Consequently, according to global guidelines for physical activity, individuals should routinely exercise for 150–300 minutes in a steady state or 75–150 minutes at a high intensity per week (Pelliccia et al., 2021). Acute exercise has long-term benefits, but if done incorrectly it can also have negative side effects, such as sudden cardiac death (Franklin et al., 2020). In addition, after prolonged exercise, endurance athletes may experience depression in left and right ventricular function; Nonetheless, after six hours of exercise, this impact is typical and frequently returns to baseline levels (Paul-Hus & Desrochers, 2019).

There are different viewpoints and debates regarding the pathological or physiological aspects of cTn release brought on by exercise. The increase in cTn after exercise is considered by some researchers to be a physiological reaction to exercise as it is frequent, occurs in the absence of clinical symptoms,

and often returns to normal within 24-72 hours after activity (S. L. Janssen et al., 2023). Increased cTn concentrations have been documented in research results throughout the last thirty years including among others the association with different types, duration, and intensity of exercise. These results have also been noted among a wide range of individuals, including age, gender, health conditions, and exercise type (Donaldson et al., 2019). The high levels of cTn induced by endurance training may compromise the cardiomyocyte sarcolemma It might cause cTn particles to flow into the bloodstream. In addition, the amount of exertion induced by physical exercise that results in increased cTn concentrations has also been linked to the risk of developing occult obstructive coronary artery disease (Kleiven et al., 2020). However, this is still confusing and confusing among researchers.

Regular engagement in physical activity and exercise has been associated with a reduced prevalence of cardiovascular disease. It also has a positive impact on longevity (Aakre & Omland, 2019). Contrary to popular belief, a number of studies have shown that exercise, regardless of duration or level of intensity, is shown to immediately increase troponin concentrations (Aengevaeren et al., 2019). This exercise-induced increase in troponin concentration is considered benign as it occurs immediately after an activity session, will naturally return to normal, is observed in healthy individuals, and does not show any clinical signs. However, much remains to be learned about the processes and practical significance of troponin generated by exercise elevations which are still very complex (Aengevaeren et al., 2019). Therefore, this systematic review will attempt to discuss how physical exercise mechanisms increase cardiac troponin (cTn) levels. In addition, it will also provide recommendations whether this physical exercise with a review of cardiac troponin levels is a natural thing or will actually have a negative impact on health, researchers will discuss in this systematic review.

Materials and methods

Study Design

Searches via journals databases, like Science Direct, Web of Science, MEDLINE-Pubmed, and Scopus are used in this kind of systematic review study. Since it collects papers of scientific significance and influence, it is considered a leading platform worldwide.

Eligibility Criteria

The study's inclusion criteria were papers that discussed cardiac troponin (cTn), physical activity, and myocardial infarction, and were published in the previous five years. In addition, journals that did not meet scientific credibility standards or were not included in the Scopus and Web of Science indexes were excluded from this study.

Procedure

Article Full texts, abstracts, and titles were checked, confirmed, and entered into Mendeley software. A total of 83 publications were found during the first phase using the databases Web of Science, Pubmed, and Science Direct. 36 papers were then selected for screening in the second stage based on whether the title and abstract met the criteria. In the third step, a total of 24 articles were asked to be processed further. We now filtered the articles according to their overall appropriateness. In the last phase of comprehensive investigation and observation, 10 papers that satisfied the inclusion criteria were chosen for analysis. For operational standards, this study adhered to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) assessment.

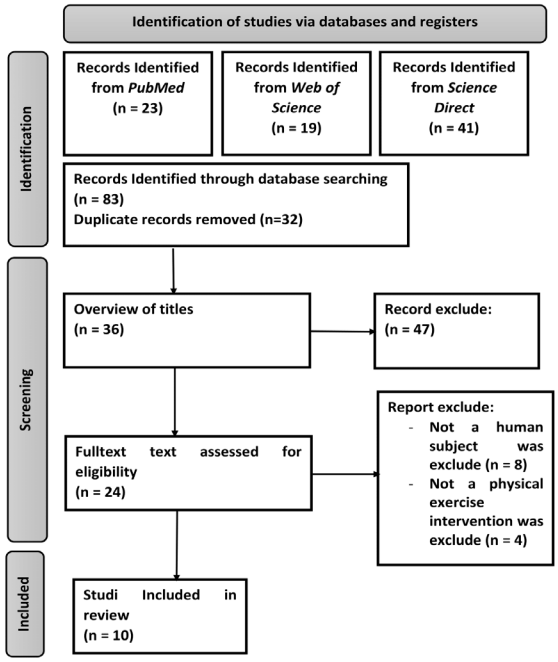

Fig. 1. PRISMA flowchart of the article selection process

Results

Description of Physical Exercise Increase Cardiac Troponin (cTn) Include in Review

The researchers found information regarding increased cardiac troponin (cTn) levels following exercise based on the papers examined. The rise in cardiac troponin levels is influenced by the length and intensity of exercise. The average intervention that has been given is physical exercise with moderate to high intensity. Both have a positive effect on increasing post-exercise cardiac troponin (cTn) levels as a marker of myocardial infarction.

Based on the 10 articles that have been analyzed (Tesema and George, 2021, Nguyen et al., 2023, Kastner et al., 2024, Orn et al., 2020, Bjorkavoll-Bergseth et al., 2024, Janssen et al., 2024, Paana et al., 2019, Cirer-Sastre et al., 2019, Birat et al., 2020, Cirer-Sastre et al., 2021) everything put into practice physical exercise interventions consisting of warm-up, core, and cool-down:

1. Warming up.
2. Moderate Intensity Physical Exercise.
3. High Intensity Physical Exercise.
4. Low Intensity Physical Exercise.
5. Stretching and Relaxation.

The participants of this study were humans consisting of athletes, untrained people, and trained people. This study did

Table 1. Summary of the design and intervention of the studies

Author	Design	Participants	Participants Age	Intervention	Outcome
Tesema & George, 2021	Randomized Controlled Trial	15	19.5 years old	1. Warm up for 5 minutes. 2. In the first week, maintain steady-state endurance running to 70% to 80% HRmax for 35 minutes per session. 3. And increase 2 minutes of exercise duration every week until 12 weeks of intervention.	1. Following exercise, there was a discernible rise in CTnI levels. Measuring Tools: Chemiluminescence Immunoassay. 2. CTnL levels are positively correlated with systolic blood pressure. Measuring Tools: Chemiluminescence Immunoassay. 3. CTnI levels showed a significant effect and positive relationship with resting heart rate. Measuring Tools: Chemiluminescence Immunoassay.
Nguyen et al., 2023	Randomized Controlled Trial	24	18 – 50 years old	High Intensity Training 1. Warm up for 10 minutes. 2. Running interval training on the treadmill every 5 minutes for 20 minutes until reaching VO_2Max. 3. Run by maintaining 80% - 95% of their HRMax for 30 minutes. Moderate Intensity Training. 1. Warm up for 10 minutes. 2. Running exercise on a treadmill maintaining 60% - 70% HRMax for 50 minutes. Low Intensity Training 1. Warm up for 10 minutes. 2. Running exercise on a treadmill maintaining 44% - 55% HRMax for 50 minutes.	1. There was an increase in troponin I and T levels in the group with high intensity training intervention both 2 hours, 4 hours and 6 hours after the intervention. Measuring instruments: ADVIA Centaur high sensitivity Troponin I immunoassay on ADVIA Centaur and STAT high sensitive Troponin I assay. Measuring Instruments: The Elecsys Troponin T hs assay on a Roche Cobas 8000 was used to quantify troponin T (cTnT). 2. There was an increase in troponin I and T 4 hours after the intervention in the moderate intensity training group. Measuring instruments: ADVIA Centaur high sensitivity Troponin I immunoassay on ADVIA Centaur and STAT high sensitive Troponin I assay. Measuring Instruments: The Elecsys Troponin T hs assay on a Roche Cobas 8000 was used to quantify troponin T (cTnT).
Kastner et al., 2024	Randomized Controlled Trial	36	22-24 years old	1. Running competition with a distance of 5 km for girls and 10 km for boys. 2. Ski sprint competition with a distance of 1.1 km performed 4 times by athletes.	1. There was an increase in troponin I in 3 male athletes. Measuring Tools : cobas Elecsys Troponin T hs STAT and High-Sensitivity Troponin I (TnIH) assay.
Orn et al., 2020	Randomized Controlled Trial	991	25-75 years old	1. Research participants completed a 91 km bicycle race in 2014. 2. Race duration was 3.4 - 4.3 hours.	1. After 3 hours of completing the race there was an increase in cTnT levels in 821 participants and 910 participants in the cTnI test that exceeded the 99th percentile limit. Measuring Tools : cTnT assay and cTnI assay. 2. After 24 hours of completing the race there was an increase in cTnI in 168 participants and an increase in cTnT in 264 participants that exceeded the 99th percentile limit. Measuring Tools : cTnT assay and cTnI assay.
Bjorkavoll-Bergseth et al., 2024	Randomized Controlled Trial	40	46-54 years old	Cardiopulmonary Exercise Test 1. Participants warm up for 10 minutes. 2. All participants exercise using an ergocycle that uses electronic brakes. 3. The VO_2max test was carried out for 5-10 minutes with participants doing the test on the ergocycle as much as possible until the VO_2max threshold. 4. Participants cool down. The Nort Sea Race 1. Participants cycled 91 km.	1. There was a significant increase in cTnI in all participants. Measuring Tools: hs-cTnI STAT assay at Architect SR2000i (Abbott Diagnostics, Abbot Park, IL, USA). 2. The highest increase in cTnI levels was found at 3 hours after undergoing both types of exercise interventions. Measuring Tools: hs-cTnI STAT assay at Architect SR2000i (Abbott Diagnostics, Abbot Park, IL, USA). 3. The increase in cTnI levels was higher after the race than during the cardiopulnary exercise test. Measuring Tools: hs-cTnI STAT assay at Architect SR2000i (Abbott Diagnostics, Abbot Park, IL, USA).

Table 1 (continued)

Author	Design	Participants	Participants Age	Intervention	Outcome
S. L. J. E. Janssen et al., 2024	Randomized Controlled Trial	58	58-69 years old	Cycling Test 1. Participants begin with a 30-minute warm-up. 2. Exercise is performed at 70% HRmax. 3. Until fatigue, the physical demand was raised by 5% every three minutes. 4. Cooling down for 3 minutes.	1. There was an increase in cTnT and cTnI levels after physical exercise intervention. 2. Measuring Tools : the Cobas 6000 analyzer (Troponin T Gen 5 STAT, Roche Diagnostics, Mannheim, Germany) and the ALINITY ci-series analyzer (Alinity i STAT High Sensitive Troponin-I Reagent Kit, Abbott Diagnostics, Abbott Park, IL). 3. There was an increase in cTnT and cTnI levels at 180 minutes post exercise. Measuring instruments: ALINITY ci-series analyzer (Alinity i STAT High Sensitive Troponin-I Reagent Kit, Abbott Diagnostics, Abbott Park, IL), and Cobas 6000 analyzer (Troponin T Gen 5 STAT, Roche Diagnostics, Mannheim, Germany).
Paana et al., 2019	Randomized Controlled Trial	43	Age: 12 (less than 35), 31 (more than 44) years old	1. MaraCat Study participants were first recruited to take part in the 2018 Paavo Nurmi Marathon in Turku.	1. Post-marathon cTnT concentrations increased below the reference standard in 38 participants. Measuring Tools : Commercial high-sensitive assay (Roche Diagnostics GmbH, Mannheim, Germany). 2. None of the participants reported post-marathon cardiac symptoms. Measuring Tools : Commercial high-sensitive assay (Roche Diagnostics GmbH, Mannheim, Germany). 3. Age significantly correlates with cTnT concentration after marathon. Measuring Tools : Commercial high-sensitive assay (Roche Diagnostics GmbH, Mannheim, Germany).
Cirer-Sastre, Legaz-Arrese, Corbi, López-Laval, et al., 2019	Randomized Controlled Trial	20	Age < > 11.9 years old	1. Research participants warm up for 15 minutes. 2. Physical exercise small soccer 5x5 for 16 minutes with interveal rest 3 minutes every 4 minutes exercise. 3. Then cool down.	1. There was an increase in hs-cTnT levels peaking at 3 hours after exercise in all participants. Measuring Tools : Troponin T hs STAT immunoassay using Roche Diagnostics' Cobas E 601 analyzer (Penzberg, Germany).

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Table 1 (continued)

Author	Design	Participants	Participants Age	Intervention	Outcome
Birat et al., 2020	Randomized Controlled Trial	12	14 – 15 years old	Session 1 VO_2Max Test 1. The first session subjects took anthropometric measurements and body mass index measurements and continued with the VO_2max test. 2. The test begins with a warm-up using an ergocycle for 4 minutes. 3. Participants perform the test using an ergocycle with 95% HRMax. Session 2 1. A competitive 42.7-kilometer adventure race for volunteers was part of the second session. 2. A variety of endurance-based sports were included in the race: Trail jogging (4.1 km), mountain biking (27.0 km), kayaking (4.4 km), and inline skating (4.2 km). The previous evening, a 5.5 km long run before the marathon. Each competitor traveled 48.2 kilometers in total ("1-day race"). Session 3 1. During the third session, the volunteers competed in a 59.0 km race over two days. 2. By participating in a series of mixed-modality endurance activities spread over two days (32.0 km on Day 1 and 27.0 km on Day 2), comprising 14.0 km of trail running, 27.0 km of mountain biking. 3. a 7.0 km trail run takes place the night before the marathon. Each competitor has traveled 66.0 kilometers in total ("2-day race").	1. There was an increase in cTnI levels in participants during the first race session immediately after the race. The i-STAT cTnI portable analyzer, manufactured by Abbott Point of Care, Inc. in Princeton, NJ, is the measuring tool used.
Cirer-Sastre et al., 2021	Randomized Controlled Trial	32	11 – 52 years old	Swimming 1. Before the intervention, participants warmed up by swimming independently for 5 minutes. 2. Participants performed long-distance swimming for 45 minutes. 3. Participants cool down.	1. There was an increase in cTnT immediately after the swimming intervention.Measuring Tools : Troponin T hs STAT immunoassay in a Cobas E 601 analyzer (Roche Diagnostics, Penzberg, Germany) with a range of 3–10,000 ng/L. 2. There was the highest increase in cTnT 3 hours after the swimming intervention. Troponin T hs STAT immunoassay in a Cobas E 601 analyzer (Roche Diagnostics, Penzberg, Germany) with a range of 3–10,000 ng/L is the measuring tool used.

not include animal participants since the criteria for inclusion were not met (Tesema and George, 2021, Nguyen et al., 2023, Kastner et al., 2024, Orn et al., 2020, Bjorkavoll-Bergseth et al., 2024, Janssen et al., 2024, Paana et al., 2019, Cirer-Sastre et al., 2019, Birat et al., 2020, Cirer-Sastre et al., 2021).

Report results from research Cirer-Sastre et al. (2019) proved that the highest increase in cardiac troponin (cTn) occurred at 3 hours after the administration of physical exercise intervention. Other research from Cirer-Sastre et al. (2021) also proved the same thing, notably, that three hours following the physical activity intervention, the rise in cardiac troponin (cTn) levels peaked. Another report from Bjorkavoll-Bergseth et al. (2024) also said the same thing that 3 hours is the peak time of cardiac troponin increase after physical exercise. Of course, differences in the concentration of cardiac troponin levels are also caused by many factors including the duration of exercise, type of exercise, and research samples, as well as blood sampling methods.

From the overall review results that have been analyzed also prove that There is no correlation between the rise in cardiac troponin brought on by physical exercise treatments and a higher risk of cardiovascular disease. Based on existing data, it proves that cardiac troponin levels will decrease by themselves after peaking in healthy people (Tesema and George, 2021, Nguyen et al., 2023, Kastner et al., 2024, Orn et al., 2020, Bjorkavoll-Bergseth et al., 2024, Janssen et al., 2024, Paana et al., 2019, Cirer-Sastre et al., 2019, Birat et al., 2020, Cirer-Sastre et al., 2021). So the increase in cardiac troponin during physical exercise is not a trigger for cardiovascular disease. However, it is still a debate among researchers whether the rise in heart troponin levels with physical activity is a physiological factor or even a pathological factor is still confusing and needs further exploration.

Intensity and Type of Physical Exercise Influence Differences in Cardiac Troponin (cTn) Concentrations

The type of exercise intervention affects the concentration of cardiac troponin levels. Based on the data that has been taken from the study Nguyen et al., 2023 that high-intensity exercise was shown to increase cardiac troponin levels higher than moderate-intensity exercise 4 hours post-intervention. And low-intensity exercise has lower cardiac troponin levels than high-intensity exercise and moderate-intensity exercise (Nguyen et al., 2023). Elevated cardiac troponin during exercise has a strong effect on increasing cardiovascular risk in people with cardiovascular disease, but not in people without cardiovascular disease (Bjorkavoll-Bergseth et al., 2024).

Research results from Cirer-Sastre et al. (2021) with subjects who were given a swimming intervention with a maximum of 45 minutes proved that cardiac troponin increased shortly after the intervention and peaked at 3 hours post exercise. The value of cardiac troponin (cTn) concentration seems to be lower in young age than old age, and this can be explained that age affects the prevalence of an increase in cardiovascular disease patients (Cirer-Sastre et al., 2019). Interestingly although the increase in cTn peaked 3 hours after the physical exercise intervention, however Cirer-Sastre et al. (2021) in his study found that there was an increase shortly after the highest exercise cTn levels in adults.

It may be that cTn elevations appear earlier compared to teenagers, in adults. Additionally, it is expanding evidence that exercise-induced cTn elevations in apparently healthy athletes may respond physiologically rather than pathologically (Baker et al., 2019). Cardiac troponin is indeed one of the biomarkers of myocardial infarction. However, especially during exercise, cTn levels increase immediately after exercise. This is highly debatable as cTn is essentially a marker of myocardial infarction.

Discussion

This study aims to examine the relationship in between exercises that can increase cardiac troponin (cTn) levels as one of the biomarkers of myocardial infarction. From the journal database search, 10 relevant articles were found according to the inclusion criteria. Due to the limited research related to cardiac troponin, the author is more focused and concentrated on this topic. Report results from Nguyen et al. (2023) proved that high-intensity exercise had higher cardiac troponin levels than low- and moderate-intensity exercise. We all know that regular exercise is a therapy that is very beneficial for the body and has a protective effect in reducing the risk of cardiovascular disease, However, other studies claim that following cardiac troponin (cTn) levels rise with physical exercise, which causes heart injury (Celeski et al., 2024).

Troponin is a complex protein that is attached to tropomyosin found in all skeletal and cardiac muscle cells (Aakre & Omland, 2019). The increase in cTn levels after physical exercise is highly variable based on how long and how hard the workout is (Aengevaeren, Baggish, Chung, George, Kleiven, Mingels, Ørn, Shave, Thompson, et al., 2021). Several investigations have been conducted to explore what factors influence and contribute to the high release of specific biomarkers of myocardial infarction markers. The high levels of cTn were found to be influenced by several factors including age, trained person, blood pressure, surrounding conditions, level of exertion, and length of exercise (Aengevaeren, Baggish, Chung, George, Kleiven, Mingels, Ørn, Shave, Thompson, et al., 2021). During physical exercise, the heart's oxygen demand in the body increases significantly (Heusch, 2019). The sole biomarker recommended for the diagnosis of myocardial infarction is cardiac troponin (cTn) in clinical practice (Taggart & Chapman, 2024).

Exercise training has been shown to have a beneficial impact on the cardiovascular system. The overall physiological changes that result from physical exercise induce a rise in cardiac output, heart rate, and stroke volume in response to exercise (Chen et al., 2022). Moreover, physical activity of the right intensity and duration can provide benefits in improving enhancing respiratory and cardiovascular fitness and lowering the risk of cardiovascular disease and other causes of death (Ezzatvar et al., 2021). WHO recommendations in 2020 that physical exercise at a moderate to high intensity is recommended to be done routinely and regularly (Bull et al., 2020). Regular physical exercise also has a beneficial effect on the occurrence of cardiac muscle hypertrophy, a condition in which the mass of the heart muscle increases and results in increased heart protection (Chen et al., 2022). Exercise-induced hypertrophy physiologically has different features and characteristics than pathologically induced hypertrophy (Li et al., 2022).

Researchers disagree on the rise in cardiac troponin after exercise. It is known that an increase in cTn during exercise is normal because it is a physiological response and

not a pathological response (Aengevaeren, Baggish, Chung, George, Kleiven, Mingels, Ørn, Shave, & Thompson, 2021). It should be noted that physical exercise is proven to improve the health status of the human body (Hussey & Gupta, 2023). During exercise, the body will activate the sympathetic nervous system (Moscatelli et al., 2022) and releases the hormone catecolamin (Er et al., 2024). The function of the heart is significantly improved during physical exercise (Lavie et al., 2023). The working mechanism of the heart muscle is also very extra to fulfill the supply of oxygen to all capillaries (Paluch et al., 2024). From the results of the study mentioned that the increase in cTn levels during physical exercise has an impact on the occurrence of cardiac muscle hypertrophy and exercise can be concluded to induce a temporary increase in troponin caused by a temporary increase in cardiomyocyte membrane permeability (Aengevaeren et al., 2019).

It is known that an increase in cTn is synonymous with an increased risk of cardiovascular disease. However, this does not apply and does not occur in people who have done physical exercise. Because the results of several studies did not mention any disorders related to increased cardiovascular system disease (Cirer-Sastre et al., 2019; Birat et al., 2020; Janssen et al., 2024). Post-physical exercise there were also no problems with the heart despite the increase in cTn (Nguyen et al., 2023). Further analysis and research is needed regarding the duration or other factors that influence the increase in cTn after physical exercise. cTn levels rise and are shown to also decrease and return to normal limits some time after the increase in cTn triggered by physical exercise (Tesema & George, 2021). So it can be said that the increase in cTn levels is normal because it is a physiological response to exercise and not pathological. Now the impact of the increase in cTn during exercise can be further analyzed and researched in order to provide understanding to academics and practitioners about its effect on other factors.

Strenght and Limitations

One of the advantages of this systematic review is that it only examines controlled trials conducted at random, which are as the most trustworthy type of scientific data because there is no possibility of unclear causal relationships. The provision of regular physical exercise and its impact on cTn improvement is very relevant with a detailed discussion of its role so as to recommend physical exercise as a preventive measure and can improve the workings of the cardiovascular system. The restriction we ran across is that there are scarcely any recent studies that address this issue, which show that the increase in cTn levels after physical exercise has a beneficial impact, has not received much attention, even though the improvement of human quality of life can be improved depending on how we can regularly perform physical exercises that can lower the risk of degenerative diseases like heart disease which is a serious problem globally.

Conclusions

Considering the related articles that we discovered, it can be concluded that regular physical training can increase cTn levels after exercise. However, this is a physiological adaptation response to exercise that does not increase the risk of myocardial infarction.

Conflicts of Interest

The authors declare no conflict of interest.

References

Ekelund, U., Tarp, J., Steene-Johannessen, J., Hansen, B. H., Jefferis, B., Fagerland, M. W., Whincup, P., Diaz, K. M., Hooker, S. P., Chernofsky, A., Larson, M. G., Spartano, N., Vasan, R. S., Dohrn, I. M., Hagströmer, M., Edwardson, C., Yates, T., Shiroma, E., Anderssen, S. A., & Lee, I. M. (2019). Dose-response associations between accelerometry measured physical activity and sedentary time and all cause mortality: Systematic review and harmonised meta-analysis. *The BMJ,* 366, 1-10. https://doi.org/10.1136/bmj.l4570

Bull, F. C., Al-Ansari, S. S., Biddle, S., Borodulin, K., Buman, M. P., Cardon, G., Carty, C., Chaput, J. P., Chastin, S., Chou, R., Dempsey, P. C., Dipietro, L., Ekelund, U., Firth, J., Friedenreich, C. M., Garcia, L., Gichu, M., Jago, R., Katzmarzyk, P. T., … Willumsen, J. F. (2020). World Health Organization 2020 guidelines on physical activity and sedentary behaviour. *British Journal of Sports Medicine, 54*(24), 1451-1462. https://doi.org/10.1136/bjsports-2020-102955

Pelliccia, A., Sharma, S., Gati, S., Bäck, M., Börjesson, M., Caselli, S., Collet, J.-P., Corrado, D., Drezner, J. A., Halle, M., Hansen, D., Heidbuchel, H., Myers, J., Niebauer, J., Papadakis, M., Piepoli, M. F., Prescott, E., Roos-Hesselink, J. W., Graham Stuart, A., … Rakhit, D. (2021). 2020 ESC Guidelines on sports cardiology and exercise in patients with cardiovascular disease. *European Heart Journal, 42*(1), 17-96. https://doi.org/10.1093/eurheartj/ehaa605

Franklin, B. A., Thompson, P. D., Al-Zaiti, S. S., Albert, C. M., Hivert, M. F., Levine, B. D., Lobelo, F., Madan, K., Sharrief, A. Z., & Eijsvogels, T. M. H. (2020). Exercise-Related Acute Cardiovascular Events and Potential Deleterious Adaptations Following Long-Term Exercise Training: Placing the Risks into Perspective-An Update: A Scientific Statement from the American Heart Association. *Circulation, 141*(13). https://doi.org/10.1161/CIR.0000000000000749

Paul-Hus, A., & Desrochers, N. (2019). Acknowledgements are not just thank you notes: A qualitative analysis of acknowledgements content in scientific articles and reviews published in 2015. *PLoS ONE, 14*(12), 1-13. https://doi.org/10.1371/journal.pone.0226727

Janssen, S. L., Berge, K., Luiken, T., Aengevaeren, V. L., & Eijsvogels, T. M. (2023). Cardiac troponin release in athletes: what do we know and where should we go? *Current Opinion in Physiology, 31*(January), 100629. https://doi.org/10.1016/j.cophys.2022.100629

Donaldson, J. A., Wiles, J. D., Coleman, D. A., Papadakis, M., Sharma, R., & O'Driscoll, J. M. (2019). Left Ventricular Function and Cardiac Biomarker Release—The Influence of Exercise Intensity, Duration and Mode: A Systematic Review and Meta-Analysis. *Sports Medicine, 49*(8), 1275-1289. https://doi.org/10.1007/s40279-019-01142-5

Kleiven, Ø., Omland, T., Skadberg, Ø., Melberg, T. H., Bjørkavoll-Bergseth, M. F., Auestad, B., Bergseth, R., Greve, O. J., Aakre, K. M., & Ørn, S. (2020). Occult obstructive coronary artery disease is associated with prolonged cardiac troponin elevation following strenuous exercise. *European Journal of Preventive Cardiology,*

27(11), 1212-1221. https://doi.org/10.1177/2047487319852808

Aakre, K. M., & Omland, T. (2019). Physical activity, exercise and cardiac troponins: Clinical implications. *Progress in Cardiovascular Diseases, 62*(2), 108-115. https://doi.org/10.1016/j.pcad.2019.02.005

Aengevaeren, V. L., Hopman, M. T. E., Thompson, P. D., Bakker, E. A., George, K. P., Thijssen, D. H. J., & Eijsvogels, T. M. H. (2019). Exercise-Induced Cardiac Troponin I Increase and Incident Mortality and Cardiovascular Events. *Circulation, 140*(10), 804-814. https://doi.org/10.1161/CIRCULATIONAHA.119.041627

Tesema, G., & George, M. (2021). Associations between cardiac troponin i and cardiovascular parameters after 12-week endurance training in young moderately trained amateur athletes. *BMJ Open Sport and Exercise Medicine, 7*(1), 14-16. https://doi.org/10.1136/bmjsem-2021-001065

Nguyen, D. K., Ellingsen, Ø., Grenne, B., Fremo, T., Hov, G. G., Røsbjørgen, R., & Mikkelsen, G. (2023). Treadmill running intensity and post-exercise increase in plasma cardiac troponin I and T—A pilot study in healthy volunteers. *Scandinavian Journal of Medicine and Science in Sports, 33*(12), 2499-2508. https://doi.org/10.1111/sms.14484

Kastner, T., Frohberg, F., Hesse, J., & Wolfarth, B. (2024). *Exercise-Induced Troponin Elevation in High-Performance Cross-Country Skiers*. https://doi.org/10.3390/jcm13082335

Orn, S., Melberg, T. ., Omland, T., Skadberg, O., Bjorkavoll-Bergseth, M. ., Erevik, C. ., Hansen, M. ., Auestad, B., Bergseth, R., Aakre, K. ., & Kleiven, O. (2020). Is cardiac troponin elevation following strenuous exercise clinically relevant in healthy subjects? *European Heart Journal, 41*(Supplement_2), 3121. https://doi.org/10.1093/ehjci/ehaa946.3121

Bjorkavoll-Bergseth, M., Erevik, C., Kleiven, Ø., Wiktorski, T., Auestad, B., Skadberg, Ø., Aakre, K. M., Eijsvogels, T. M. H., & Ørn, S. (2024). The relationship between workload and exercise-induced cardiac troponin elevations is influenced by non-obstructive coronary atherosclerosis. *Scandinavian Journal of Medicine and Science in Sports, 34*(5), 1-13. https://doi.org/10.1111/sms.14667

Janssen, S. L. J. E., De Vries, F., Mingels, A. M. A., Kleinnibbelink, G., Hopman, M. T. E., Mosterd, A., Velthuis, B. K., Aengevaeren, V. L., & Eijsvogels, T. M. H. (2024). Exercise-induced cardiac troponin release in athletes with versus without coronary atherosclerosis. *American Journal of Physiology - Heart and Circulatory Physiology, 326*(4), H1045-H1052. https://doi.org/10.1152/ajpheart.00021.2024

Paana, T., Jaakkola, S., Bamberg, K., Saraste, A., Tuunainen, E., Wittfooth, S., Kallio, P., Heinonen, O. J., Knuuti, J., Pettersson, K., & Airaksinen, K. E. J. (2019). Cardiac troponin elevations in marathon runners. Role of coronary atherosclerosis and skeletal muscle injury. The MaraCat Study. *International Journal of Cardiology,* 295, 25-28. https://doi.org/10.1016/j.ijcard.2019.08.019

Cirer-Sastre, R., Corbi, F., López-Laval, I., Carranza-García, L. E., & Reverter-Masià, J. (2021). Exercise-induced release of cardiac troponins in adolescent vs. Adult swimmers. *International Journal of Environmental Research and Public Health, 18*(3), 1-6. https://doi.org/10.3390/ijerph18031285

Cirer-Sastre, R., Legaz-Arrese, A., Corbi, F., George, K., Nie, J., Carranza-García, L. E., & Reverter-Masià, J. (2019). Cardiac biomarker release after exercise in healthy children and adolescents: A systematic review and meta-analysis. *Pediatric Exercise Science, 31*(1), 18-36. https://doi.org/10.1123/pes.2018-0058

Cirer-Sastre, R., Legaz-Arrese, A., Corbi, F., López-Laval, I., Puente-Lanzarote, J., Hernández-González, V., & Reverter-Masià, J. (2019). Effect of training load on post-exercise cardiac troponin T elevations in young soccer players. *International Journal of Environmental Research and Public Health, 16*(23). https://doi.org/10.3390/ijerph16234853

Birat, A., Bourdier, P., Dodu, A., Grossoeuvre, C., Blazevich, A. J., Amiot, V., Dupont, A. C., Nottin, S., & Ratel, S. (2020). Effect of Long-Duration Adventure Races on Cardiac Damage Biomarker Release and Muscular Function in Young Athletes. *Frontiers in Physiology, 11*(February), 1-9. https://doi.org/10.3389/fphys.2020.00010

Baker, P., Leckie, T., Harrington, D., & Richardson, A. (2019). Exercise-induced cardiac troponin elevation: An update on the evidence, mechanism and implications. *IJC Heart and Vasculature,* 22, 181-186. https://doi.org/10.1016/j.ijcha.2019.03.001

Celeski, M., Segreti, A., Piscione, M., Monticelli, L. M., Di Gioia, G., Fossati, C., Ussia, G. P., Pigozzi, F., & Grigioni, F. (2024). The current paradigm of cardiac troponin increase among athletes. *Monaldi Archives for Chest Disease.* https://doi.org/10.4081/monaldi.2024.2878

Aengevaeren, V. L., Baggish, A. L., Chung, E. H., George, K., Kleiven, Ø., Mingels, A. M. A., Ørn, S., Shave, R. E., Thompson, P. D., & Eijsvogels, T. M. H. (2021). Exercise-Induced Cardiac Troponin Elevations: From Underlying Mechanisms to Clinical Relevance. *Circulation, 144*(24), 1955-1972. https://doi.org/10.1161/CIRCULATIONAHA.121.056208

Heusch, G. (2019). Myocardial ischemia: Lack of coronary blood flow, myocardial oxygen supply-demand imbalance, or what? *American Journal of Physiology - Heart and Circulatory Physiology, 316*(6), H1439-H1446. https://doi.org/10.1152/ajpheart.00139.2019

Taggart, C., & Chapman, A. R. (2024). Cardiac troponin and the diagnosis of type 2 myocardial infarction and acute non-ischaemic myocardial injury. *Journal of Laboratory and Precision Medicine,* 9. https://doi.org/10.21037/jlpm-23-40

Chen, H., Chen, C., Spanos, M., Li, G., Lu, R., Bei, Y., & Xiao, J. (2022). Exercise training maintains cardiovascular health: signaling pathways involved and potential therapeutics. *Signal Transduction and Targeted Therapy, 7*(1). https://doi.org/10.1038/s41392-022-01153-1

Ezzatvar, Y., Izquierdo, M., Núñez, J., Calatayud, J., Ramírez-Vélez, R., & García-Hermoso, A. (2021). Cardiorespiratory fitness measured with cardiopulmonary exercise testing and mortality in patients with cardiovascular disease: A systematic review and meta-analysis. *Journal of Sport and Health Science, 10*(6), 609-619. https://doi.org/10.1016/j.jshs.2021.06.004

Li, H., Trager, L. E., Liu, X., Hastings, M. H., Xiao, C., Guerra, J., To, S., Li, G., Yeri, A., Rodosthenous, R., Silverman, M. G., Das, S., Ambardekar, A. V., Bristow, M. R., González-Rosa, J. M., & Rosenzweig, A. (2022). LncExACT1 and DCHS2 Regulate Physiological and Pathological Cardiac Growth. *Circulation, 145*(16), 1218-1233. https://doi.org/10.1161/CIRCULATIONAHA.121.056850

Hussey, C., & Gupta, A. (2023). *Physical activity and its impact on the health of young people.* December.

Moscatelli, F., Sessa, F., Valenzano, A., Polito, R., Eronia, S., & Monda, V. (2022). The Influence of Physical Exercise, Stress and Body Composition on Autonomic Nervous System: A Narrative Review. *Sport Mont, 20*(2), 131-134. https://doi.org/10.26773/smj.220620

Er, F., Yilmaz, C., Polat, M., Kuzucu, Ö. E., & Suveren, C. (2024). Effect of Physical Exercise on Catecholamine Levels in Individuals over 65 Years of Age. *International Journal of Human Movement and Sports Sciences, 12*(3), 463-470. https://doi.org/10.13189/saj.2024.120302

Фізичні вправи підвищують рівень серцевого тропоніну (cTn) як маркера інфаркту міокарда: Систематичний огляд

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Авторський вклад: A – дизайн дослідження; B – збір даних; C – статаналіз; D – підготовка рукопису; E – збір коштів

Реферат. Стаття: 8 с., 1 табл., 1 рис., 34 джерела.

Мета дослідження. Метою цього дослідження було проаналізувати показник впливу фізичних вправ на підвищення рівня серцевого тропоніну (cTn) як маркера інфаркту міокарда з фізіологічної точки зору.

Матеріали та методи. У цьому систематичному оглядовому дослідженні використовували пошук видань, що індексуються у наукометричних базах даних, включаючи MEDLINE-Pubmed, Web of Science, Scopus і Science Direct. Критеріями включення були роботи, які стосувалися дослідження серцевого тропоніну (cTn), фізичної активності та інфаркту міокарда, а також були опубліковані впродовж останніх п'яти років. За допомогою наукометричних баз даних Science Direct, Pubmed та Web of Science було знайдено 83 авторитетні публікації. З метою проведення систематичного огляду було відібрано та досліджено десять публікацій, які відповідали критеріям включення. У цьому дослідженні проведена оцінка стандартних методів роботи із використанням «Переважних елементів звітності для систематичних оглядів і мета-аналізів» (PRISMA).

Результати. У цьому систематичному огляді зазначається, що фізичні вправи підвищують рівень серцевого тропоніну (cTn) як маркера інфаркту міокарда, однак це підвищення спричинено фізіологічною реакцією організму під час виконання фізичних вправ і не розглядається як патологічна реакція.

Висновки. Згідно з результатами дослідження, доведено, що фізичні вправи збільшують ризик пошкодження серцевого м'яза внаслідок підвищення рівня серцевого тропоніну (cTn). Однак цей ефект спостерігається у фізіологічному стані і представляє собою процес адаптації до фізичних навантажень. Аналіз відповідних наукових статей показує, що таке підвищення серцевого тропоніну (cTn) не має негативного впливу на розвиток серцево-судинних захворювань.

Ключові слова: серцевий тропонін, фізичні вправи, інфаркт міокарда, здоровий спосіб життя.

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