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The Cardiovascular Paradox of Marathon Running: Acute Stress, Chronic Adaptations, and Long-term Health Implications

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ABSTRACT

This article explores the intricate relationship between marathon running and cardiovascular health. As participation in marathons increases year by year, scientific research into the acute responses and chronic adaptations of the cardiovascular system is becoming more common. Acute cardiac responses contain temporary elevations in cardiac biomarkers and changes in cardiac function, which especially affect the right ventricle. However, chronic adaptations include beneficial cardiac remodeling. The review analyzes evidence from numerous studies documenting these cardiovascular changes, exploring both potential concerns and substantial benefits. Although numerous scientific articles suggest possible risks associated with extreme endurance training, epidemiological data shows that marathon runners have a significantly lower risk of cardiovascular disease. This study investigates impact on the left and right ventricular chambers, vascular function, autonomic regulation and correlation between exercise intensity and cardiovascular health. The study also discusses the clinical implications of screening, monitoring, and recovery protocols, providing a balanced perspective on the cardiovascular impact of marathon participation. Furthermore, the study combines the current evidence to inform clinical practice, that with proper preparation and medical screening, marathon running can be advantageous for cardiovascular health.

Keywords: marathon running, cardiac biomarkers, ventricular function, arterial compliance, arrhythmias, cardiovascular adaptation

1. INTRODUCTION

One of the most popular sports among amateur athletes is marathon running, which is a very physically demanding endurance effort. Marathon running is a 42,195-kilometer challenge leads to influential physiological stress on numerous body systems. The cardiovascular system experiences specific acute as well as chronic adaptations (Braschler et al., 2025). Over the past few years, running marathons has become unimaginably popular, which has sparked interest among

people of all fitness levels. Due to this rise in popularity, more scientists have become interested in the long- as well as short-term effects of this significant physical activity on the cardiovascular system. Temporary increases in cardiac biomarkers, such as cardiac troponin I (cTnI) and N-terminal pro-brain natriuretic peptide (NT-proBNP) are an acute cardiac responses to marathon completion, which have been documented to increase markedly after finishing (Wegberger et al., 2020). These elevated biomarkers values initially raised concerns about potential myocardial damage. However, contemporary research suggests they likely represent benign physiological responses rather than pathological damage (Hewing et al., 2015; Sanchez et al., 2006).

Hewing et al., (2015) concluded that increased levels of circulating cardiac troponin T after exercise are considered to be unrelated to irreversible cardiac injury. They may instead result from increased permeability of the cardiomyocyte membrane, leading to leakage of cytosolic troponin, due to altered metabolic conditions, transient inflammation, and reversible ischaemia.

Beyond biomarker changes, marathon running induces significant structural and functional cardiac adaptations. Regular endurance training leads to beneficial cardiac remodeling, known as the “athlete's heart", which is characterized by thicker walls, greater myocardial mass, and increased left and right ventricular size (Braschler et al., 2025). These adaptations increase stroke volume and cardiac output during exercise, which improves cardiovascular efficiency. However, emerging research suggests that there may be differential effects on ventricular function. The right ventricle appears to be particularly susceptible to acute, exercise-induced stress (Tiller et al., 2020; Karlstedt et al., 2012; Pagourelias et al., 2022).

Marathon participation also significantly impacts vascular function, with studies demonstrating acute alterations in arterial compliance following endurance events (Bonsignore et al., 2017; Burr et al., 2012; Burr et al., 2014). These changes appear to depend on the d. Moderate-distance events may improve arterial compliance, while extreme ultra-marathons may temporarily increase arterial stiffness (Bonsignore et al., 2017).

The relationship between marathon running and long-term cardiovascular health is an intriguing paradox. Whereas moderate endurance training has well-established cardiovascular benefits, the possible harmful consequences of extreme endurance exercise remain obscure. Among long-term endurance athletes, some studies suggest potential dangers, including ventricular dysfunction, myocardial fibrosis, increased susceptibility to arrhythmias (especially atrial fibrillation) (Scheer et al., 2022). However, epidemiological evidence generally supports the significant cardiovascular benefits of marathon participation, with Rosin demonstrating substantially lower cardiovascular disease prevalence (17% vs. 34.6% in the general population) and enhanced longevity in marathon runners compared to age-matched controls (Rosin, 2017).

The data presented in the following article can provide an analysis of the relationship between marathon running and cardiovascular health. Additionally, it will investigate the physiological mechanisms behind probable complications and positive adaptations. We will review knowledge of the vascular and cardiac response to marathon completion, assess potential cardiovascular risk, examine chronic adaptations and discuss clinical recommendations for screening and monitoring cardiovascular health.

2. REVIEW METHODS

In creating this review, we relied on literature analysis from sources such as PubMed, ScienceDirect and Google Scholar. The data analysis was conducted in March 2025 and we focused only on English language articles, which we searched by using relevant keywords. The review includes publications from January 2006 to February 2025 (Figure 1).

3. RESULTS AND DISCUSSION

The Impact of Marathon Running on the Cardiovascular System

Marathon running is considered the most physically demanding endurance activity that athletes can participate in, with extraordinary physiological stress on multiple body systems, particularly the cardiovascular system. The immediate cardiovascular response to marathon running involves significant hemodynamic changes designed to meet the increased metabolic demands of working muscles. During a marathon, cardiac output increases dramatically and the heart must then maintain an elevated stroke volume and heart rate for several hours, which has been shown to trigger measurable changes in cardiac function and biomarkers.

One of the most consistent findings following marathon completion is the elevation of cardiac biomarkers. Wegberger et al., (2020) demonstrated that both marathon and ultra-marathon participation trigger measurable elevations in cardiac-specific biomarkers, including cardiac troponin I (cTnI) and N-terminal pro-brain natriuretic peptide (NT-proBNP), with the response and the magnitude of the elevation directly linked with the duration of the event. Specifically, they found that cTnI levels reached 0.028 ng/L following completion of a marathon and 0.056 ng/L following completion of an ultra-marathon. In comparison, NT-proBNP levels were

substantially higher following an ultra-marathon (723 ng/L) than following a marathon (132 ng/L). Their data suggested a strong correlation between event duration and acute cardiac strain, highlighting the impact of exercise duration on the cardiovascular system.

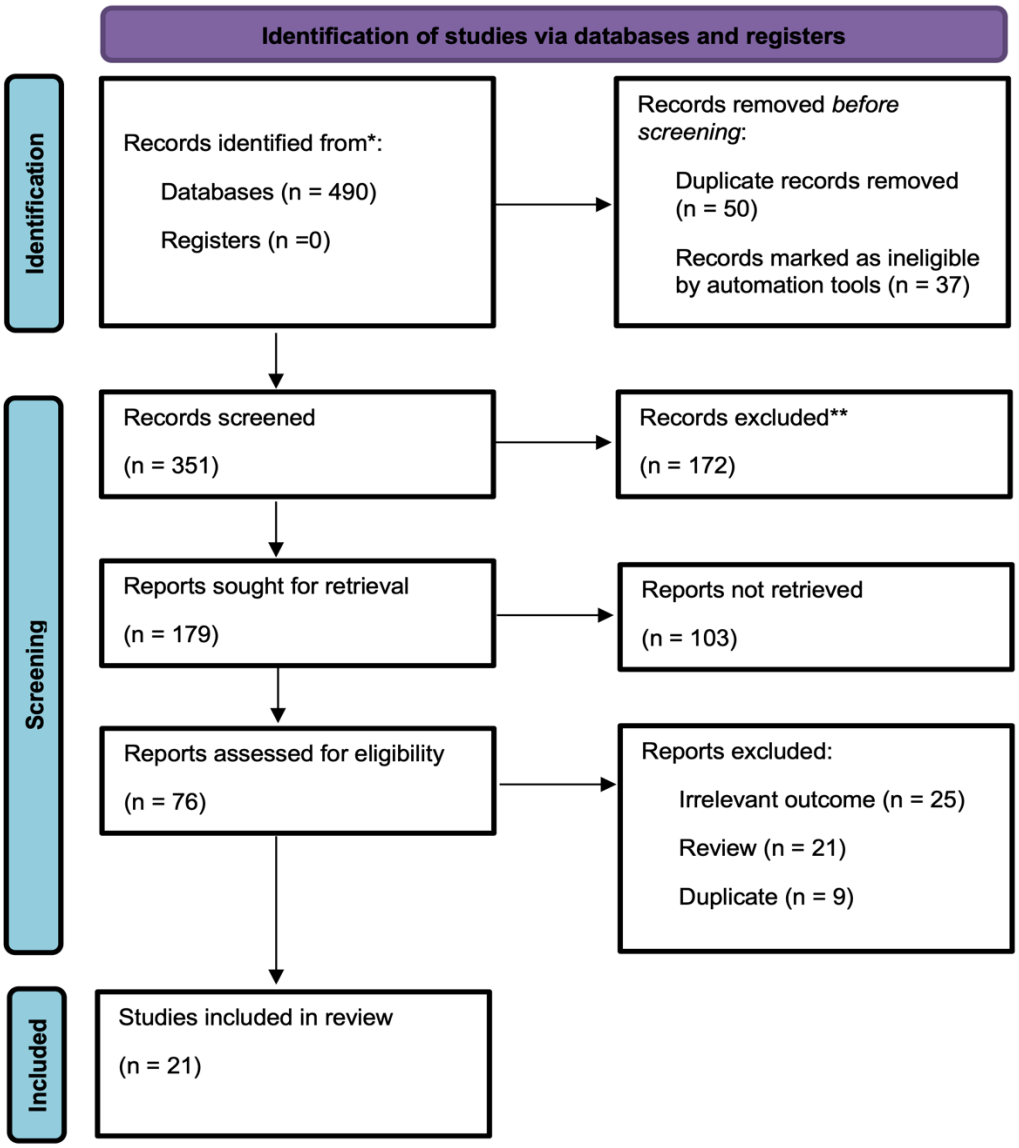

Fig 1. PRISMA flow diagram of selected studies (*PubMed, ScienceDirect and Google Scholar)

These biomarker elevations raised concerns about potential myocardial injury and clinical implications for the athletes in the future. However, contemporary research suggests they likely represent benign physiological responses rather than pathological damage. Hewing et al. concluded that increased levels of circulating cardiac troponin T following exercise are not considered to be indicative of irreversible cardiac injury, but may result from increased permeability of the cardiomyocyte membrane, leading to the leakage of cytosolic troponin due to altered metabolic conditions, transient inflammation, and reversible ischemia (Hewing et al., 2015). This interpretation, supported by Leers et al., (2006) who observed that the elevation of troponin levels typically normalizes within 24 hours after the marathon, may suggest a mechanism of transient myocardial stress rather than permanent damage to the heart.

Sanchez et al., (2006) further clarified that troponin assays are recommended over creatine kinase-MB (CK-MB) in order to assess potential myocardial injury, and unnecessary investigations should be avoided in runners who present no symptoms after the exercise, unless prompted by specific symptoms such as chest pain or syncope.

Marathon running also induces temporary alterations in cardiac function, with the right ventricle (RV) appearing particularly susceptible to the prolonged acute stress. Significant post-race reductions in all RV function metrics, including ejection fraction,

following ultramarathon events were presented in a study by Tiller et al., (2020). They observed that, although left ventricular (LV) function was retained across race types, there were significant post-race reductions in all metrics of right ventricular (RV) function, including ejection fraction. Pertinently, RV stroke volume remained depressed at 1-week follow-up. In contrast, the left ventricle demonstrates greater resilience to the stress of marathon training. Kean et al., (2006) found that recreational athletes had preserved left ventricular (LV) function following marathon completion, no significant changes in ejection fraction or contractility indices were observed and that marathon running by a group of well-trained recreational athletes does not result in impairment of LV systolic or diastolic function.

The differential ventricular response likely reflects the disproportionate increase in pulmonary vascular resistance during prolonged exercise, which places greater relative strain on the thin-walled right ventricle. These findings were confirmed by Karlstedt et al., (2012) in a study of older marathon runners (>50 years). The study noted significant right atrial and ventricular enlargement with reduced RV function after the race, which returned to normal after one week. Their study found that most runners showed no evidence of myocardial damage. However, two participants exhibited late gadolinium enhancement on cardiac MRI, suggesting possible fibrosis associated with underlying coronary artery disease.

Pagourelias et al., (2022) provided further insights using advanced deformation analysis after a 246 km ultra-marathon, observing slight decreases in right and left ventricular longitudinal strain post-race, mainly driven by impairment of basal segments with preservation of apical regions. They concluded that these minor strain changes are probably functional adaptations rather than signs of irreversible myocardial damage or cardiomyopathy. They also concluded that the heart remains functional after such demanding endurance activity, with only minimal and likely reversible functional impairment. Another characteristic of physiological alterations in marathon running is vascular function. While the moderate-distance runs (80km) tend to improve large arterial compliance, the extreme ultra-marathons (195km and more) have been found to increase arterial stiffness. This suggests a potential dose-response relationship, in which moderate endurance exercise enhances vascular elasticity, whereas extreme volumes may temporarily stiffen the arteries (Bonsignore et al., 2017).

A related study has observed decreases in large artery compliance after completing an ultra-marathon. In the related study, the team observed decreases in large artery compliance after completing an ultramarathon. Additionally, in runners with higher baseline compliance, these decreases were more visible (Burr et al., 2012). Burr et al., (2014) later found that the long-term ultramarathon participants (over 5 years of participation) showed reduced large artery compliance compared to age-matched recreationally active controls. This finding raises interesting questions about a potential "inverted-U" dose-response relationship between exercise volume and vascular health, where extreme endurance exercise might have adverse effects on arterial stiffness over time.

However, Perrotta et al., (2022) found no significant change in arterial compliance, as assessed by applanation tonometry, after an ultra-endurance event. They did, however, observe decreased systemic vascular resistance, as well as lowered systolic and diastolic blood pressure after exercise; this indicates post-exercise hypotension. Moreover, intense physical activity has been proved to trigger a drop in blood pressure and inflammatory responses.

Additionally, the autonomic nervous system shows a significant decrease in heart rate variability markers immediately following ultra-marathon completion, which reflects the suppression of parasympathetic tone. It then normalizes within 48 hours. These findings highlight the temporary nature of many marathon-induced cardiovascular alterations, as well as the great capacity for recovery of well-trained athletes (Fazackerley et al., 2019).

Regular marathon training increases cardiovascular health and overall performance. Adaptations like changes in structure and function that optimize cardiac performance during stamina excercise are known as "athlete's heart". This results in enhanced diastolic function, reduced filling times and ultimately an increase in cardiac output during endurance exercise. They also observe that enhanced vagal tone and reduced sympathetic tone to the heart promote cardiovascular health and reduce the risk of cardiovascular events (Braschler et al., 2025). Konwerski et al., (2021) provided additional evidence for the cardiovascular benefits of marathon running. They examined epicardial adipose tissue (EAT) volume, which serves as an important predictor of cardiovascular risk. Their study proved that marathon runners have significantly lower EAT volumes than age-matched controls. This suggests that endurance training may reduce the risk of cardiovascular disease through another pathway. The conclusion was that extremely intensive training could reduce the amount and pro-inflammatory activity of EAT, and therefore potentially decrease the risk of cardiovascular events in the adult population of amateur athletes.

Nevertheless, some researchers have questioned the potential cardiovascular risks connected to extreme endurance exercise; some of these risks include right ventricular dysfunction, myocardial fibrosis, and increased risk of arrhythmias (particularly atrial

fibrillation). By observing mechanisms such as increased vagal tone and structural remodeling of the atria, Scheer et al., (2022) found that athletes who engage in high-intensity endurance training have a three- to five-fold increased risk of atrial fibrillation compared to sedentary individuals.

Predel (2014) pointed out that, while extreme endurance activities such as marathon running may seem to promote health, they can still result in high cardiovascular stress. He suggests that a balanced approach, such as opting for half-marathons, may offer the best health benefits while minimising risks for middle-aged amateur athletes with cardiovascular risk factors.

Despite these potential concerns, epidemiological evidence overwhelmingly supports the cardiovascular benefits of endurance exercise, even at the marathon level. Rosin (2017) conducted an observational study that lasted 45 years and involved 54 male marathon runners. The study's findings demonstrated a substantially lower prevalence of cardiovascular disease (17% compared to 34,6% in the general elderly U.S. population), as well as enhanced longevity in comparison to age-matched controls. The average age at death among 18 deceased runners was 81.3 years, compared to approximately 61 years for the age-matched general population. These findings strongly suggest that, on balance, the cardiovascular benefits of marathon training outweigh the potential risks for most participants.

More recent research by Burger et al., (2024) examined the impact of an ultra-endurance marathon on cardiac function in association with cardiovascular biomarkers. They found that participants with biomarker levels above the median showed significant reductions in left ventricular ejection fraction (LVEF) and right ventricular function after the race. In contrast, those below the median did not. These effects were temporary, with biomarker levels returning to normal within days. No long-term side effects were conclusively observed.

Several critical clinical implications arise from this body of research. Firstly, transient increases in biomarkers following marathon events should be interpreted with caution in asymptomatic runners. Secondly, at the start of marathon training, people with multiple risk factors, especially older athletes, may require comprehensive cardiovascular screening. O'Riordan et al., (2023) found that many amateur marathon runners remain unaware of their cardiovascular risk factors, despite regularly participating in marathons. Therefore, it is essential to raise awareness and implement preventive health measures among amateur marathon runners. Thirdly, it is necessary to allow adequate recovery periods between marathon events to enable complete cardiovascular restoration and minimise potential cumulative stress. Karstoft et al., (2013) investigated the effects of running seven consecutive marathons per day and found that while this extreme regimen can be tolerated without significant cardiac damage by well-trained athletes, proper recovery protocols have been found to be crucial to maintaining cardiovascular health.

In conclusion, marathon running induces profound cardiovascular adaptations that, for most participants, enhance cardiovascular health and potentially extend lifespan. However, this requires appropriate training, recovery, and medical screening, particularly for older athletes or those with existing cardiovascular risk factors (Table 1).

Table 1. Marathon Running and the Cardiovascular System

Aspect	Summary of Findings
Acute Cardiac Effects	Transient increases in cardiac biomarkers (troponins, NT-proBNP) and temporary right ventricular (RV) dysfunction are seen in clinical studies of marathon runners. These changes usually resolve within a few days and do not indicate permanent heart damage in most runners.
Cardiac Function	Left ventricular (LV) function is generally preserved, while RV function may decline temporarily, especially after ultra-marathons. Most functional changes are reversible with recovery.
Vascular Effects	Moderate endurance improves arterial compliance, but extreme distances or long-term ultra-endurance may temporarily increase arterial stiffness.
Autonomic & Recovery	Heart rate variability drops after races but normalizes within 48 hours, reflecting transient autonomic changes.

Chronic Adaptations	Regular marathon training leads to beneficial cardiac remodeling ("athlete's heart"), lower epicardial fat, and reduced cardiovascular risk.
Potential Risks	High-volume or long-term endurance running may increase risk of atrial fibrillation, myocardial fibrosis, and coronary artery calcification in some athletes.
Epidemiological Data	Marathon runners have lower cardiovascular disease prevalence and greater longevity compared to the general population.
Clinical Implications	Individualized screening and adequate recovery are important for older or high-risk athletes, even though cardiovascular effects are mostly benign and reversible.

4. CONCLUSION

Clinical studies show that several acute cardiovascular stressors and chronic physiological adaptations may be associated with marathon running. However, these findings require careful consideration. The characteristics of an acute cardiac response to completing a marathon include transient elevations in biomarkers (such as troponins and natriuretic peptides) and right ventricular (RV) dysfunction. These typically resolve within days. Contemporary evidence suggests that surges in biomarkers do not necessarily indicate myocardial injury. Instead, they are more likely to reflect reversible physiological stress, rather than pathological damage. This is thought to stem from increased permeability in cardiomyocyte membranes, as well as transient metabolic shifts. Similarly, RV systolic impairment observed in 30–40% of athletes after a marathon appears to resolve with adequate recovery. However, prolonged dysfunction in individuals with extensive competition histories warrants monitoring.

Chronic adaptations to marathon training, collectively termed "athlete's heart," include left ventricular (LV) eccentric hypertrophy, enhanced diastolic function, and vagally mediated bradycardia—beneficial modifications that optimize cardiac output and endurance performance. However, newly appearing data highlight potential long-term risks in subsets of athletes. Myocardial fibrosis, identified in 5–13% of veteran marathoners and a 3–5-fold increased atrial fibrillation risk compared to sedentary populations, suggests that cumulative exercise may promote arrhythmogenic substrates in susceptible individuals. Coronary artery calcification (CAC) scores, though higher in endurance athletes, may represent stable plaque adaptations rather than vulnerable lesions with unclear clinical significance.

There is overwhelming epidemiological evidence supporting the cardioprotective benefits of marathon running, including a reduction in cardiovascular mortality and an enhancement of longevity. Nevertheless, the U-shaped risk curve observed in extreme endurance exercise highlights the importance of individualized risk stratification. Clinicians should prioritise pre-participation screening for hidden cardiovascular pathology, especially in athletes with a family history of arrhythmia or those presenting a long competition history. It is essential for future research to elucidate the mechanisms underlying fibrosis development, optimize biomarkers for distinguishing between physiological and pathological remodeling, and establish evidence-based thresholds for exercise dosing. For most recreational runners, adhering to progressive training protocols and allowing for adequate recovery is crucial to reap the benefits of marathon running while mitigating potential risks.

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Conflict of interest
The authors declare that there is no conflict of interest.

Data and materials availability
All data associated with this work are present in the paper.

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