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Morphological and Hemodynamic Adaptations of the Athlete's Heart to Chronic Exercise-Induced Cardiac Remodeling

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ABSTRACT

Purpose: The aim of this review is to examine exercise-induced cardiac remodeling within the conceptual framework of the athlete's heart, to clarify the different effects of endurance and strength training on cardiac structure and function, and to distinguish physiological adaptations from pathological cardiac remodeling.

Method: In this study, the existing literature on the topic was systematically reviewed, and studies investigating the effects of chronic exercise on cardiac structure and function were evaluated. As a result of the screening process, a total of 42 scientific studies that met the inclusion criteria were included and analyzed within the scope of this review.

Results: The findings of the literature indicate that endurance training promotes eccentric cardiac remodeling associated with volume overload in the heart. This adaptation is characterized by increases in ventricular volume, stroke volume, cardiac output, and maximal oxygen uptake. In addition, endurance training leads to positive functional outcomes such as reduced resting heart rate and increased cardiac efficiency. In contrast, strength training appears to be more closely associated with concentric hypertrophic adaptations related to pressure overload. These exercise-induced cardiac changes are largely physiological in nature and are generally associated with improved performance outcomes.

Conclusion: Overall, cardiac remodeling resulting from chronic exercise can largely be considered a beneficial and adaptive physiological response. However, excessive or inadequately monitored training loads may contribute to maladaptive cardiac changes in some cases. Therefore, individualized training programs and regular cardiovascular monitoring remain important for maintaining long-term cardiac health in athletes.

Keywords: Athlete's heart, Cardiac remodeling, Cardiovascular adaptation, Exercise, Hemodynamic changes

ÖZET

Kronik Egzersize Bağlı Kardiyak Yeniden Yapılanmaya Sporcu Kalbinin Morfolojik ve Hemodinamik Adaptasyonları

Amaç: Bu derlemenin amacı, sporcu kalbi kavramsal çerçevesi içerisinde kronik egzersizin kardiyovasküler sistem üzerinde oluşturduğu kardiyak yeniden şekillenme süreçlerini incelemek, dayanıklılık ve kuvvet antrenmanlarının kalp yapısı ve fonksiyonları üzerindeki farklı etkilerini ortaya koymak ve egzersize bağlı gelişen fizyolojik adaptasyonları patolojik kardiyak yeniden şekillenmeden ayırt etmektir.

Yöntem: Çalışmada konuya ilişkin mevcut literatür sistematik bir yaklaşımla taranmış ve kronik egzersizin kardiyak yapı ve fonksiyon üzerindeki etkilerini inceleyen araştırmalar değerlendirmeye alınmıştır. Tarama süreci sonucunda dahil edilme kriterlerini karşılayan toplam 42 bilimsel çalışma analiz edilerek derleme kapsamında incelenmiştir.

Bulgular: Literatür bulguları, dayanıklılık antrenmanlarının kalpte hacim yüklenmesine bağlı olarak gelişen eksantrik yeniden şekillenmeyi desteklediğini göstermektedir. Bu adaptasyon; ventrikül hacminde artış, atım hacmi ve kardiyak debide yükselme ile maksimal oksijen tüketiminde artış gibi fizyolojik değişikliklerle karakterizedir. Bununla birlikte dayanıklılık antrenmanı, dinlenim kalp hızının azalması ve kardiyak verimliliğin artması gibi olumlu

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fonksiyonel sonuçlar da ortaya çıkarmaktadır. Öte yandan kuvvet antrenmanlarının daha çok basınç yüklenmesine bağlı konsantrik hipertrofik adaptasyonlarla ilişkili olduğu belirlenmiştir. Egzersiz kaynaklı bu kardiyak değişimlerin büyük ölçüde fizyolojik nitelikte olduğu ve spor performansını destekleyici etkiler taşıdığı görülmektedir.
Sonuç: Genel olarak kronik egzersizin neden olduğu kardiyak yeniden şekillenme, çoğunlukla faydalı ve adaptif bir fizyolojik yanıt olarak değerlendirilmektedir. Bununla birlikte aşırı veya uygun şekilde izlenmeyen antrenman yükleri, bazı durumlarda maladaptif kardiyak değişimlere yol açabilmektedir. Bu nedenle sporcularda kardiyak sağlığın korunması açısından bireyselleştirilmiş antrenman programlarının uygulanması ve düzenli kardiyovasküler izlemin sürdürülmesi önem taşımaktadır.
Anahtar Kelimeler: Egzersiz, Hemodinamik değişiklikler, Kardiyak yeniden yapılanma, Kardiyovasküler adaptasyon, Sporcu kalbi

INTRODUCTION

Physical exercise is widely recognized as one of the principal determinants in the preservation and enhancement of cardiac health through the acute and chronic physiological effects it exerts on the cardiovascular system. Understanding the effects of exercise on cardiac structure and function constitutes a major focus of research in exercise physiology and sport sciences, both for optimizing athletic performance and for reducing cardiovascular disease risk in the general population. Regular physical activity induces significant structural and functional modifications within the cardiovascular system, positively influencing cardiac pump capacity, circulatory efficiency, and overall cardiac performance. In this context, the investigation of exercise-induced cardiac adaptations is of paramount importance for elucidating physiological adaptations in healthy individuals and for distinguishing these changes from pathological conditions.

One of the most prominent examples of such adaptations is described in the literature as the “athlete’s heart.” Athlete’s heart refers to the adaptive structural changes that occur in response to regular and intensive physical training, and it is particularly characterized by increases in left ventricular volume and myocardial hypertrophy. These structural adaptations enhance cardiac output during exercise, facilitating greater delivery of oxygen and nutrients to peripheral tissues and thereby supporting cardiovascular efficiency (Baba Ali et al., 2024). Beyond structural modifications, athlete’s heart is also associated with improved cardiac performance at rest and during exercise, contributing to enhanced endurance capacity and overall athletic performance (Zimmermann et al., 2021). In this regard, athlete’s heart is considered an indicator of physiological adaptation and a key component of a healthy and efficiently functioning cardiovascular system.

One of the fundamental processes underlying these beneficial exercise-induced changes is cardiac remodeling. Cardiac remodeling refers to long-term alterations in myocardial structure and function and is often confused with structural transformations associated with pathological conditions such as heart failure or hypertension. However, remodeling induced by regular exercise differs fundamentally from pathological remodeling, as it represents a physiological and adaptive process. Exercise-induced remodeling enhances structural integrity and functional capacity of the myocardium, thereby supporting cardiovascular performance. Indeed, the "physiological remodeling" observed in healthy individuals following regular exercise contributes to myocardial strengthening and plays a protective role in preventing cardiac atrophy that may develop as a consequence of physical inactivity (Hedge et al., 2024; Bletsa et al., 2023).

Moreover, it has been reported that the effects of different exercise modalities on cardiac remodeling may vary. In particular, specific training methods such as high-intensity interval training (HIIT) have been shown to exert favorable effects on left ventricular hypertrophy and to enhance cardiovascular endurance (Venckūnas et al., 2025; Drummond et al., 2023). Within this framework, interval training has been demonstrated to promote left ventricular hypertrophy and improve cardiovascular endurance capacity (Xiao et al., 2025; Eskandari et al., 2020).

However, it has also been suggested that excessive exercise volume or inadequate recovery periods may adversely affect the cardiac remodeling process, potentially weakening the heart's protective adaptations and increasing the risk of pathological remodeling (Yang et al., 2021). Therefore, the effects of exercise on cardiac structure and function are closely associated with training load, intensity, and overall program characteristics.

In light of these considerations, a comprehensive evaluation of exercise-induced cardiac adaptations is essential for both the preservation of athlete health and the optimization of performance. The aim of this review is to examine the effects of chronic exercise on the cardiac remodeling process in accordance with current literature; to address the morphological and hemodynamic adaptations observed in the athlete's heart from a holistic perspective; and to systematically present the existing evidence by clearly distinguishing between physiological remodeling and pathological alterations. This review aims to provide an updated synthesis of recent evidence on chronic exercise-induced cardiac remodeling, with particular attention to differences between endurance and strength training.

METHODS

This study is a structured literature review designed to comprehensively evaluate the process of chronic exercise-induced cardiac remodeling and the morphological and hemodynamic adaptations observed in the athlete's heart. In order to systematically and reproducibly examine the existing scientific evidence, a planned and structured literature search strategy was implemented.

The literature search was conducted in the PubMed/MEDLINE, Web of Science, Scopus, and Google Scholar databases. To identify relevant studies, the keywords "cardiac remodeling," "athlete's heart," "chronic exercise," "exercise training," "cardiac adaptation," "morphological adaptation," "hemodynamic response," "left ventricular hypertrophy," "endurance training," and "strength training" were searched individually and in various combinations using Boolean operators (AND, OR) (Cumpston et al., 2019). Additionally, to broaden the scope of the search and ensure conceptual consistency, Medical Subject Headings (MeSH) terms were utilized in the PubMed database, and structured searches were conducted based on standardized indexing terms. The literature search was conducted between 21 December 2025 and 29 January 2026 to ensure transparency of the search timeline.

To expand the literature coverage and identify potentially overlooked studies, a reference chain searching method (snowballing) was applied. In this context, the reference lists of the selected articles were examined retrospectively (backward snowballing), and more recent studies citing these publications were evaluated prospectively (forward snowballing).

The review included full-text research articles, narrative reviews, systematic reviews, and meta-analyses published in peer-reviewed journals between 2020 and 2025 in English and Turkish. This time frame was specifically chosen to ensure the inclusion of the most recent and up-to-date literature on the topic. Priority was given to studies investigating chronic exercise-induced cardiac morphological and hemodynamic adaptations in athletes or healthy individuals. Animal studies, research focusing on pathological cardiac conditions in clinical patient populations, studies examining only acute exercise effects, and publications without accessible full texts were excluded.

The quality and reliability of the included studies were systematically assessed using structured risk of bias tools. Specifically, AMSTAR 2 was applied for systematic reviews and meta-analyses, ROBINS-I for non-randomized studies, and the Cochrane Risk of Bias tool for randomized trials. These tools allow a comprehensive, transparent, and reproducible

evaluation of potential sources of bias, including selection, measurement, and reporting biases. Each study was evaluated across relevant domains: AMSTAR 2 provided an overall confidence rating (high, moderate, low, critically low) for systematic reviews; ROBINS-I assessed non-randomized studies for confounding, participant selection, and intervention classification; and the Cochrane RoB tool evaluated randomization, allocation concealment, blinding, and outcome reporting. This structured assessment enabled a clear understanding of the strengths and limitations of the existing evidence and informed the interpretation of findings in the context of chronic exercise-induced cardiac adaptations.

The initial screening of titles and abstracts, followed by full-text evaluation, was conducted by the author alone, with careful cross-checking to ensure consistency and minimize bias. Data extraction was systematically performed by the author using a predefined extraction form.

The identified studies were initially screened at the title and abstract level, and those directly relevant to the topic were assessed in full text. Following the eligibility assessment, a total of 42 studies met the inclusion criteria and were included in the final analysis. The distribution of these studies by publication year is presented in Figure 1.

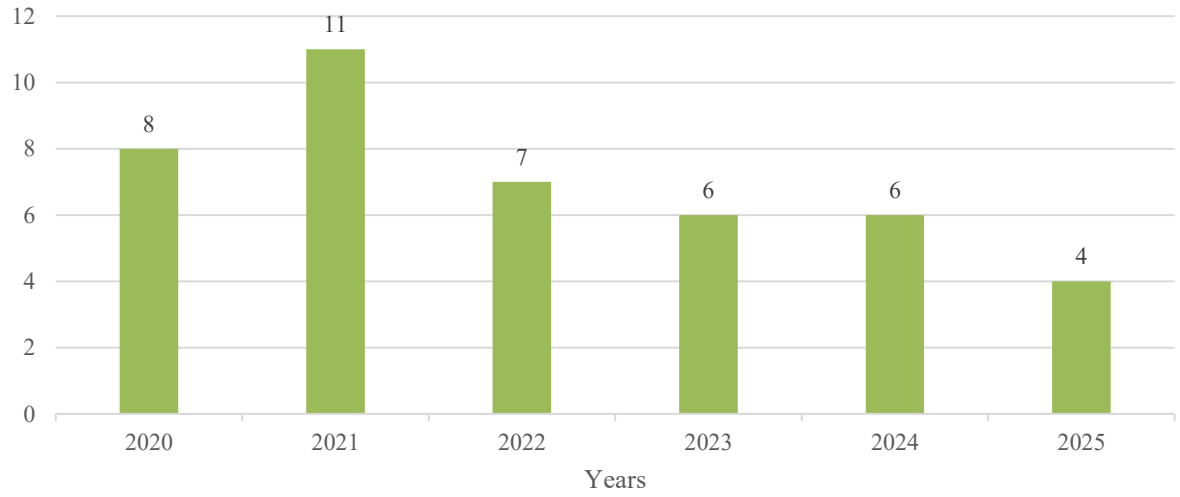

Figure 1. Distribution of Included Studies by Publication Year

The selected publications were categorized according to the themes of cardiac remodeling mechanisms, morphological changes, hemodynamic adaptations, differences according to training modalities, and the distinction between physiological and pathological remodeling. Findings were synthesized and interpreted using a thematic approach. A detailed PRISMA-compliant flowchart outlining the identification, screening, eligibility assessment,

and final inclusion processes of the reviewed studies is presented in Figure 2 to enhance the transparency and reproducibility of the review methodology.

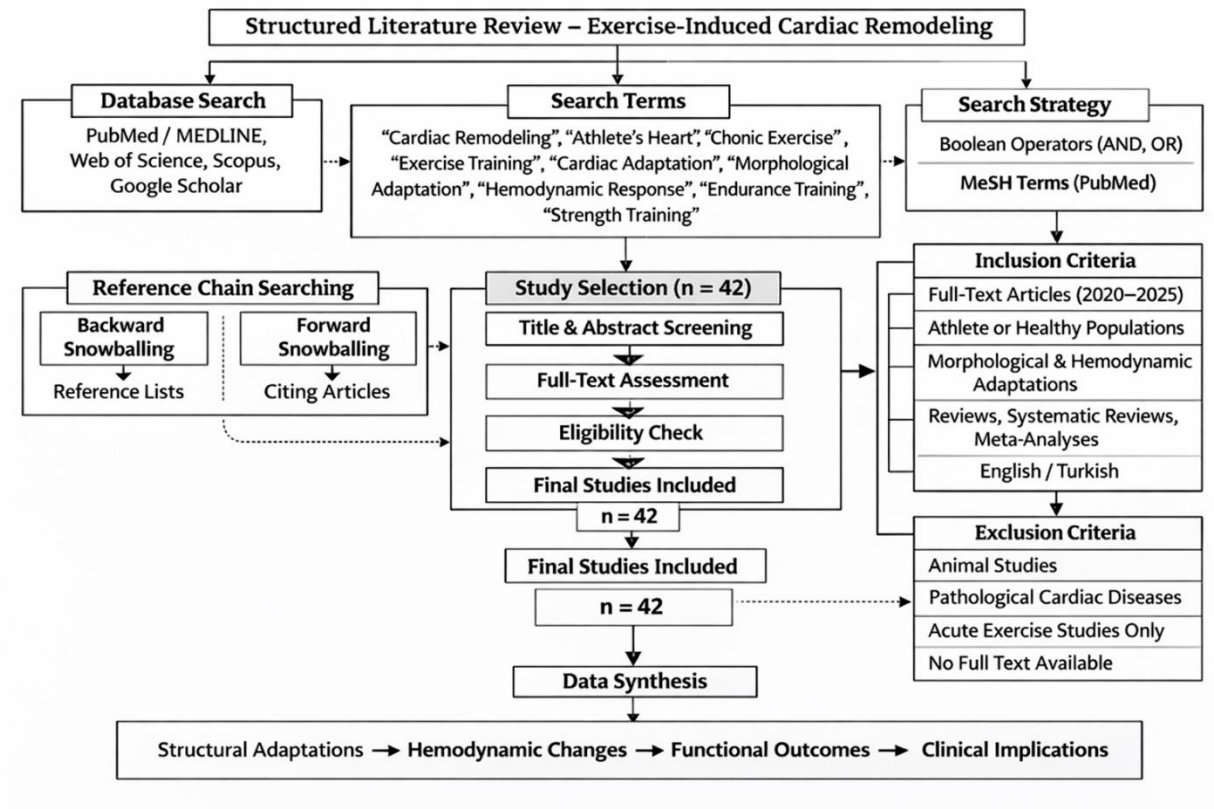

Figure 2. PRISMA-Compliant Flow Diagram of Study Selection

EVIDENCE SYNTHESIS

Physiological Mechanisms of Exercise-Induced Cardiac Remodeling

Hemodynamic loading conditions that occur during exercise are primary determinants of the cardiac remodeling process, leading to adaptive structural and functional changes in the heart. In this context, increases in preload and afterload play a central role. Preload refers to the volume of blood filling the ventricles during diastole, whereas afterload represents the vascular resistance encountered by the heart during systole. With exercise, increased venous return has generally been linked to elevations in preload (Dowrick et al., 2022), while elevated arterial pressure is generally associated with higher afterload (Kusy et al., 2021). These hemodynamic stressors collectively trigger morphological and functional adaptations in the myocardium.

Intensive exercise loading may stimulate myocardial hypertrophy; however, these adaptations vary depending on the type of exercise performed. Sprint-based training is more commonly associated with increases in left ventricular wall thickness, whereas endurance

training is primarily linked to ventricular dilation (Kusy et al., 2021; Kirsch et al., 2024). These differences reflect the specific loading patterns imposed by distinct exercise modalities. Sustained elevations in preload and afterload are generally related to the development of physiological hypertrophy and appear to enhance cardiac pump function (Lasocka et al., 2024; Kusy et al., 2021; Liang et al., 2021). Moreover, mechanical loading activates hormonal systems, including natriuretic peptides, which contribute to the regulation of fluid and electrolyte balance (Yu et al., 2020; Ditterline et al., 2020).

The integrated outcome of these adaptations is commonly interpreted as the "athlete's heart," whereby regular exercise may support cardiac endurance and could lower the risk of cardiovascular disease (Hulshof et al., 2020; Szabó et al., 2022). Nevertheless, excessive and uncontrolled training loads may potentially lead to maladaptive remodeling, underscoring the importance of appropriate training prescription and monitoring (Huang et al., 2021; Kambič et al., 2021).

Table 1. Fundamental Hemodynamic Mechanisms of Exercise-Induced Cardiac Remodeling

Mechanism	Definition	Exercise-Induced Change	Cardiac Effect	References
Increased Preload	Increased ventricular filling during diastole	Elevated venous return and expanded plasma volume during exercise	Ventricular dilation, increased stroke volume (SV), development of eccentric hypertrophy	Kusy et al., 2021; Lasocka et al., 2024; Liang et al., 2021
Increased Afterload	Vascular resistance encountered by the left ventricle during systole	Elevated arterial pressure and peripheral vascular resistance, particularly during high-intensity or resistance exercise	Increased wall thickness, concentric hypertrophy	Dowrick et al., 2022; Kusy et al., 2021; Kirsch et al., 2024
Mechanical Stress	Volume and pressure overload imposed on the myocardium	Repetitive hemodynamic loading due to chronic training	Physiological myocardial hypertrophy and structural remodeling	Lasocka et al., 2024; Kirsch et al., 2024
Hormonal Activation	Activation of natriuretic peptides and neurohormonal pathways in response to myocardial stretch	Stretch-induced secretion of atrial and brain natriuretic peptides (ANP, BNP)	Regulation of fluid-electrolyte balance and hemodynamic homeostasis	Yu et al., 2020; Ditterline et al., 2020
Outcome: Physiological Remodeling ("Athlete's Heart")	Adaptive structural and functional cardiac remodeling secondary to regular exercise	Chronic adaptation to sustained preload and afterload stimuli	Enhanced pump capacity, improved cardiac efficiency, reduced cardiovascular risk	Hulshof et al., 2020; Szabó et al., 2022

Exercise-Induced Morphological Cardiac Adaptations

Exercise, particularly endurance and strength training, induces morphological cardiac adaptations characterized by increases in ventricular volume, myocardial hypertrophy, and alterations in cardiac mass. This physiological remodeling process is defined in the literature

as the “athlete’s heart” and is generally interpreted as an indicator of healthy cardiovascular adaptation (Diaz-Rodriguez et al., 2024).

Endurance-based training has generally been associated with increases in left ventricular volume. Studies have generally reported ventricular enlargement in endurance athletes (Slankamenac et al., 2022; Zimmermann et al., 2021), and increases in left ventricular diastolic dimensions and cardiac mass index appear to occur in elite judokas (Zimmermann et al., 2021). These structural adaptations may contribute to increased stroke volume and improved exercise efficiency.

Prolonged and high-intensity training loads also seem to promote left ventricular hypertrophy and increases in myocardial mass, which are generally interpreted as physiological adaptations to elevated preload and resistance loading (Jiang et al., 2021; Gastl et al., 2020; Diaz-Rodriguez et al., 2024). However, differentiating athlete’s heart from pathological hypertrophy remains clinically important, and regular cardiac monitoring is recommended (Gioia et al., 2023; Gastl et al., 2020). Depending on the sport discipline, endurance sports are more commonly associated with ventricular dilation, whereas strength-based sports tend to be linked to increased wall thickness (Johnson et al., 2023; Gastl et al., 2020).

Table 2. Morphological Cardiac Adaptations Associated with Chronic Exercise

Morphological Parameter	Observed Adaptation	Exercise Type Predominance	Physiological Significance	References
Left Ventricular End-Diastolic Diameter (LVEDD)	Significant increase	Predominantly endurance training	Enhances stroke volume and diastolic filling capacity	Slankamenac et al., 2022; Zimmermann et al., 2021
Left Ventricular Wall Thickness	Mild to moderate increase	More evident in strength training	Reflects adaptation to pressure overload	Gastl et al., 2020; Johnson et al., 2023
Left Ventricular Mass Index (LVMI)	Increased	Both endurance and strength athletes	Indicates physiological myocardial hypertrophy	Jiang et al., 2021; Diaz-Rodriguez et al., 2024
Ventricular Dilatation	Chamber enlargement	Primarily endurance athletes	Supports higher cardiac output during exercise	Slankamenac et al., 2022; Kusy et al., 2021
Myocardial Hypertrophy	Symmetrical and proportional hypertrophy	Both types (pattern differs)	Adaptive structural remodeling without functional impairment	Gastl et al., 2020; Kirsch et al., 2024
Cardiac Geometry	Eccentric geometry (endurance); concentric geometry (strength)	Exercise-type specific	Reflects dominant hemodynamic stimulus (volume vs pressure load)	Kusy et al., 2021; Johnson et al., 2023
Cardiac Mass	Overall increase	Both	Contributes to improved pump performance	Diaz-Rodriguez et al., 2024; Jiang et al., 2021
Clinical Consideration	Differentiation from pathological hypertrophy required	All competitive athletes	Important for distinguishing athlete’s heart from cardiomyopathy	Gioia et al., 2023; Gastl et al., 2020

Exercise-Induced Hemodynamic and Functional Adaptations

Sport participation and regular physical activity induce pronounced hemodynamic and functional adaptations in the cardiovascular system, particularly in parameters such as stroke volume, cardiac output, resting bradycardia, and VO_2max. In response to increased metabolic demand, endurance training in particular has generally been associated with increases in stroke volume, with reported improvements reaching up to 30% (Cahalin et al., 2022; Pittaras et al., 2023). Strengthening of the myocardium and expansion of ventricular volume through aerobic training may contribute to elevations in both stroke volume and cardiac output (Weberruß et al., 2022; Pittaras et al., 2023). Additionally, improvements in vascular responsiveness appear to support the maintenance and enhancement of overall cardiovascular function (Perales et al., 2025). Another common adaptation associated with endurance training is resting bradycardia. Due to enhanced pump efficiency, the heart is generally able to maintain adequate cardiac output at lower heart rates, a phenomenon that may be related to improved energetic efficiency (Yuan et al., 2021; Pittaras et al., 2023). In elite athletes, resting heart rate can fall below 60 beats per minute (bpm), which is generally interpreted as a physiological adaptation rather than a pathological condition (Cahalin et al., 2022; Perales et al., 2025). Reduced heart rate has generally been associated with increased stroke volume and cardiac output (Weberruß et al., 2022; Pittaras et al., 2023). Furthermore, regular aerobic and high-intensity interval training have generally been associated with increases in VO_2max by approximately 15–20%. Such improvements may positively influence cardiovascular endurance, oxygen transport capacity, and overall physical performance (Pittaras et al., 2023; Perales et al., 2025; Weberruß et al., 2022; Yuan et al., 2021; Cahalin et al., 2022).

Table 3. Comparison of Structural Cardiac Adaptations According to Exercise Type

Parameter	Endurance Sports (Aerobic Training)	Strength/Sprint Sports (Resistance Training)	References
Primary Hemodynamic Load	Volume overload (predominantly increased preload)	Pressure overload (predominantly increased afterload)	Kusy et al., 2021; Kirsch et al., 2024
Left Ventricular Cavity Size	Marked increase (ventricular dilation)	Normal or mildly increased	Slankamenac et al., 2022; Johnson et al., 2023
Left Ventricular Wall Thickness	Proportional or mild increase	Pronounced wall thickening	Gastl et al., 2020; Johnson et al., 2023
Type of Hypertrophy	Eccentric hypertrophy	Concentric hypertrophy	Kusy et al., 2021; Kirsch et al., 2024
Cardiac Mass	Increased	Increased	Diaz-Rodriguez et al., 2024; Jiang et al., 2021
Stroke Volume (SV)	Markedly increased	Mild to moderate increase	Pittaras et al., 2023; Kirsch et al., 2024
Resting Heart Rate	Significant bradycardia	Less pronounced bradycardia	Cahalin et al., 2022; Kirsch et al., 2024
VO_2max	Substantial increase (up to 15–20%)	Limited increase	Pittaras et al., 2023; Perales et al., 2025
Persistence of Functional Adaptation	Long-term and sustained adaptations	Predominantly acute pressure-related responses	Meah et al., 2020; Lässing et al., 2025

Cardiac Adaptations According to Different Training Modalities

Different training modalities appear to produce specific structural and hemodynamic adaptations in the heart, thereby potentially influencing athletic performance, endurance capacity, and overall cardiovascular function. Endurance and strength training may modify cardiac loading patterns in distinct ways, leading to different adaptive mechanisms in cardiac structure and function.

Endurance sports involve prolonged and continuous physical activity and are generally associated with adaptations that seem to enhance cardiac pump capacity. These training modalities have generally been linked to increases in left ventricular volume and stroke volume (Lässing et al., 2025; Kirsch et al., 2024), and may contribute to elevations in heart rate response, stroke volume (SV), and cardiac output (CO) during exercise (Kirsch et al., 2024; Javad, 2021). Parallel improvements in pulmonary capacity and oxygen utilization are generally associated with increases in VO_2max (Kodli, 2023). Structurally, enlargement of ventricular cavity dimensions accompanied by relatively thinner wall profiles may support more efficient cardiac function (Meah et al., 2020), while resting bradycardia is generally interpreted as a typical marker of this physiological adaptation (Kirsch et al., 2024).

In contrast, strength training is characterized by short-duration but high-intensity loading, primarily generating pressure-based stress that could induce hypertrophic changes. During such training, the heart may operate transiently under elevated pressure and stroke volume conditions (Lässing et al., 2025; Kodli, 2023). However, some findings suggest that these responses might represent predominantly acute adaptations rather than sustained functional enhancements (Meah et al., 2020). Compared to endurance athletes, bradycardia is generally less pronounced in strength-trained individuals, and cardiac responses tend to reflect short-term hemodynamic loading patterns (Kirsch et al., 2024; Lässing et al., 2025; Meah et al., 2020).

Clinical Distinction Between Physiological and Pathological Cardiac Remodeling in Athletes

In athletes, the heart generally undergoes adaptive physiological remodeling in response to regular and intensive training; however, excessive or uncontrolled loading may potentially lead to pathological alterations. Accurate differentiation between these two processes is of critical clinical importance for safeguarding athlete health and ensuring safe performance participation.

Physiological remodeling is defined as the "athlete's heart" and is generally characterized by increased left ventricular volume and appears to enhance myocardial efficiency, especially in response to endurance training (Fang et al., 2020; Allwood, 2022). Increased ventricular dimensions and stroke volume may support cardiac performance and could improve exercise tolerance (Albaeni et al., 2021). Echocardiography remains the primary diagnostic modality for monitoring these adaptations (Maestrini et al., 2020; Locatelli et al., 2024).

In contrast, pathological remodeling is generally characterized by maladaptive hypertrophy, fibrosis, and impaired cardiac function (Allwood, 2022; Fang et al., 2020; Albaeni et al., 2021), and may be triggered by excessive training loads (Maestrini et al., 2020). Conditions such as arrhythmogenic right ventricular cardiomyopathy can clinically resemble athlete's heart, necessitating careful differential diagnosis (Allwood, 2022; Locatelli et al., 2024). In this regard, cardiac magnetic resonance imaging (MRI) may provide substantial clinical value in distinguishing physiological adaptations from pathological conditions (Albaeni et al., 2021; Locatelli et al., 2024).

Table 4. Physiological and Pathological Remodeling

Parameter	Physiological Remodeling ("Athlete's Heart")	Pathological Remodeling	Clinical Assessment Tools	References
Left Ventricular Cavity Size	Symmetrical enlargement, proportional to body size and training load	May be normal, reduced, or asymmetrically enlarged	Echocardiography	Fang et al., 2020; Allwood, 2022
Wall Thickness	Mild-to-moderate, symmetrical increase	Marked or asymmetric hypertrophy (e.g., septal predominance)	Echocardiography, Cardiac MRI	Allwood, 2022; Locatelli et al., 2024
Left Ventricular Function (EF)	Preserved or enhanced systolic and diastolic function	Reduced or impaired function	Echocardiography, MRI	Albaeni et al., 2021; Maestrini et al., 2020
Myocardial Fibrosis	Absent	Present (late gadolinium enhancement on MRI)	Cardiac MRI	Albaeni et al., 2021; Locatelli et al., 2024
Diastolic Function	Normal or improved filling pattern	Diastolic dysfunction may be present	Doppler Echocardiography	Fang et al., 2020; Allwood, 2022
Arrhythmia Risk	Low in healthy athletes	Increased risk, particularly in cardiomyopathies (e.g., ARVC)	ECG, Holter Monitoring, MRI	Allwood, 2022; Locatelli et al., 2024
Reversibility	Partial regression with detraining	Typically non-reversible	Follow-up Imaging	Maestrini et al., 2020
Clinical Approach	Periodic monitoring and screening	Further diagnostic evaluation and possible treatment	Multimodal cardiac evaluation	Albaeni et al., 2021; Locatelli et al., 2024

DISCUSSION and CONCLUSION

The findings of the present review comprehensively indicate that regular, structured, and long-term exercise elicits multidimensional structural, hemodynamic, and functional adaptations within the myocardium, and that these adaptations represent fundamental

determinants of cardiovascular efficiency and performance enhancement in athletes. Collectively, the available evidence suggests that exercise-induced cardiac remodeling, when occurring within physiological limits, differs substantially from pathological remodeling processes and is most appropriately characterized as a reversible and adaptive phenomenon commonly referred to as the “athlete’s heart” (Fang et al., 2020; Allwood, 2022). This concept underscores the notion that the trained heart undergoes coordinated and proportional modifications that serve to optimize circulatory performance in response to repetitive hemodynamic stress.

From a morphological standpoint, endurance-oriented training modalities have consistently been associated with significant increases in left ventricular end-diastolic volume and overall cardiac mass. These changes contribute to an augmented stroke volume and enhanced pump capacity, particularly during sustained dynamic exercise (Slankamenac et al., 2022; Zimmermann et al., 2021; Diaz-Rodriguez et al., 2024). Prolonged and intensive loading conditions further promote physiological myocardial hypertrophy, which reflects an adaptive response to chronically elevated preload and intermittent pressure stress rather than a maladaptive structural alteration (Jiang et al., 2021; Gastl et al., 2020). Importantly, these structural adaptations are typically symmetrical and proportionate, preserving ventricular geometry and systolic-diastolic function. When interpreted in conjunction with functional improvements, the morphological remodeling observed in trained athletes appears to enhance mechanical efficiency, allowing the heart to generate greater cardiac output with relatively lower energetic cost under both resting and exercise conditions.

Furthermore, it is essential to acknowledge that cardiac remodeling exhibits sex-specific characteristics. According to Lasocka et al. (2024), while males predominantly display concentric hypertrophy, females are more likely to experience eccentric remodeling. Ventricular changes are accompanied by atrial dilatation and mild reductions in function, which are more pronounced in males than in females. These physiological differences are influenced in part by the cardioprotective effects of estrogen in female athletes, contributing to lower susceptibility to pathological remodeling and arrhythmias. Incorporating these sex-specific insights into the synthesis enhances the understanding of physiological adaptation and emphasizes the need for tailored cardiovascular monitoring strategies for both male and female athletes.

In addition to structural remodeling, hemodynamic and functional adaptations provide a complementary framework that explains the superior cardiovascular performance of trained

individuals. Endurance training has been shown to increase stroke volume by as much as 30%, accompanied by significant elevations in maximal cardiac output and improved systemic oxygen transport capacity (Cahalin et al., 2022; Pittaras et al., 2023; Weberruß et al., 2022). These changes are mediated not only by ventricular enlargement and improved contractile efficiency but also by peripheral vascular adaptations that facilitate enhanced oxygen delivery. Resting bradycardia, frequently observed in well-trained athletes, emerges as a physiological consequence of increased stroke volume and augmented parasympathetic tone, and is widely regarded as a hallmark of improved cardiac efficiency (Yuan et al., 2021; Perales et al., 2025). Furthermore, increases in VO_2max—often ranging between 15% and 20% depending on baseline fitness and training intensity—are directly associated with improvements in aerobic capacity, endurance performance, and overall cardiovascular resilience (Pittaras et al., 2023; Perales et al., 2025). Taken together, these hemodynamic and metabolic adaptations reinforce the concept that exercise-induced remodeling is functionally advantageous.

Moreover, it should not be overlooked that the observed structural and hemodynamic changes are underpinned by cellular and genetic mechanisms. Regular exercise promotes adaptations in cardiomyocytes, including hypertrophy, mitochondrial remodeling, and oxidative stress responses. These processes are closely linked to gene expression and epigenetic regulation, providing cardioprotective effects. In individuals with genetic predispositions, exercise can modulate pathways such as PKC-α/NFAT, reducing hypertrophy and fibrosis (Wu et al., 2021; Wang et al., 2022). When considered together with the observed ventricular remodeling and functional adaptations, these mechanisms demonstrate that exercise induces a multi-level, coordinated, and holistic adaptation process in the heart.

An important consideration emerging from the literature is that the specific pattern and direction of cardiac remodeling are largely determined by the predominant hemodynamic stimulus imposed by different training modalities. Endurance sports, characterized by sustained volume overload, are primarily associated with eccentric remodeling and ventricular chamber dilation. In contrast, strength- and resistance-based sports, which impose repetitive pressure overload, are more frequently linked to concentric hypertrophy with increased wall thickness (Lässing et al., 2025; Kirsch et al., 2024; Meah et al., 2020). These sport-specific adaptations reflect distinct loading conditions and emphasize the necessity of discipline-sensitive approaches in training prescription, monitoring strategies, and cardiovascular evaluation. Recognition of these physiological distinctions is particularly relevant for

clinicians and sports scientists aiming to differentiate adaptive remodeling from early manifestations of pathology.

Despite the predominantly beneficial nature of exercise-induced cardiac remodeling, the distinction between physiological and pathological processes remains a critical clinical concern. Excessive, prolonged, or inadequately monitored training loads may, in susceptible individuals, contribute to maladaptive structural alterations, including disproportionate hypertrophy, myocardial fibrosis, and functional impairment. (Maestrini et al., 2020; Fang et al., 2020). However, precise thresholds for these maladaptive changes are difficult to define due to individual variability and limited clinical data, highlighting the need for careful monitoring and personalized training plans. Moreover, certain cardiomyopathies may phenotypically resemble the athlete's heart, thereby complicating differential diagnosis (Allwood, 2022; Locatelli et al., 2024). In this context, comprehensive cardiovascular screening assumes paramount importance. Echocardiography remains the primary diagnostic modality for evaluating chamber dimensions, wall thickness, and systolic-diastolic function, while cardiac magnetic resonance imaging provides superior tissue characterization and facilitates the detection of myocardial fibrosis when indicated (Albaeni et al., 2021; Locatelli et al., 2024). Systematic and periodic assessment is therefore essential to ensure safe sport participation and to mitigate the risk of adverse cardiac events, including sudden cardiac death.

Conclusion

In conclusion, the cumulative evidence synthesized in this review supports the interpretation that chronic exercise-induced cardiac remodeling in athletes predominantly represents a beneficial, adaptive, and performance-enhancing physiological process. By increasing pump efficiency, optimizing hemodynamic responses, and improving aerobic capacity, such remodeling contributes not only to superior athletic performance but also to long-term cardiovascular health. Importantly, sex-specific differences must be acknowledged, as males and females exhibit distinct remodeling patterns that influence functional outcomes and clinical risk profiles. Nevertheless, the maintenance of these favorable adaptations depends on appropriate training load management, sufficient recovery periods, and regular clinical surveillance. These findings may help coaches and clinicians interpret exercise-related cardiac changes more accurately during routine athlete monitoring. Future research should specifically address gaps such as the lack of longitudinal studies assessing long-term cardiac adaptation, training responses in athletes with specific genetic predispositions, and the

integration of advanced imaging techniques for precise tissue characterization. Such integrated approaches are essential for maximizing athletic performance while safeguarding long-term cardiovascular integrity.

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