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Health Promotion Strategies for Managing Exercise Intensity and Duration Among Athletes: A Scoping Review

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ABSTRACT

Introduction: Regular physical exercise offers substantial benefits to cardiovascular health by improving aerobic capacity, cardiac efficiency, and reducing the risk of cardiovascular disease. However, excessive or unregulated training may lead to maladaptive cardiac remodeling and increase the risk of arrhythmias or sudden cardiac death. Understanding the physiological boundaries of exercise-induced cardiac adaptation is therefore crucial for promoting safe and effective athletic performance.
Methods: This scoping review followed a systematic protocol based on the PEOS framework (Population, Exposure, Outcome, Study Design). Literature searches were conducted in PubMed, ScienceDirect, EBSCO, and Google Scholar for studies published between 2000 and 2024 using relevant keywords on cardiac responses to exercise. Inclusion criteria covered original research and reviews focusing on cardiac adaptation, remodeling, and training intensity among athletes. From an initial 512 records, 20 eligible studies were critically appraised and synthesized using a narrative approach.
Results: Endurance training predominantly induced eccentric hypertrophy and increased left and right ventricular volumes without systolic dysfunction, while resistance training produced concentric hypertrophy due to higher pressure load. These structural adaptations were physiological, reversible, and not associated with fibrosis. Conversely, extreme endurance activities were linked to transient cardiac fatigue and elevated cardiac biomarkers. Detraining studies confirmed regression of cardiac mass, highlighting the reversible nature of physiological remodeling.
Conclusion: Cardiac responses to exercise depend on training intensity, duration, and type. Physiological remodeling enhances cardiovascular function, whereas excessive training or genetic predispositions may lead to pathological consequences. Balancing load and recovery, alongside periodic medical evaluation, is essential to optimize performance and prevent cardiovascular complications.

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INTRODUCTION

Exercise has been shown to benefit everyone, even those with cardiovascular disease (CVD). Regular exercise improves CVD risk through various mechanisms, including lowering triglycerides, increasing high-density lipoprotein (HDL) cholesterol, weight loss, lowering blood pressure, improving glucose metabolism, reducing insulin resistance, and reducing inflammatory markers in the blood. Increasing exercise intensity is directly proportional to a reduced risk of CVD. In fact, the more active a person is, the smaller the reduction in risk is associated with increased exercise volume(1,2). However, any exercise or physical activity is better than no exercise at all. Moderate physical activity has been recommended for all individuals at risk for cardiovascular disease. Regardless of the benefits, a doctor should evaluate each individual's status and determine their risk level for sudden cardiac arrest during exercise (3).

In sports cardiology, a relatively new subspecialty, information on the natural history of disease progression and the risk of heart attack during intense exercise is relatively scarce. Prevention of heart attacks during intense exercise or competitive sports always includes a thorough medical examination, including a cardiac examination. In fact, most guidelines rely on the extensive experience of consensus groups, rather than large-scale prospective studies (3).

English: In response to increased stress or load, the heart must work harder than usual. To compensate for this long-term stress, stressed heart muscle cells increase in size and mass. This increase in heart muscle mass is defined as cardiac hypertrophy. At the cellular level, cardiac hypertrophy can be divided into physiological and pathological. Myocytes (cardiac muscle cells), non-myocytes (fibroblasts, endothelial cells, mast cells, vascular smooth muscle cells), and the surrounding extracellular matrix form the structure of the heart. Cardiac hypertrophy is an independent risk factor for myocardial infarction, dysrhythmias, and even sudden cardiac death. In response to chronically increased stress, the heart increases its mass to normalize wall tension and maintain normal cardiovascular function. This mechanism is a form of organ compensation for stressful conditions.

In contrast, hypertrophy in its physiological form represents the refinement of the athlete's heart, a benign increase in heart mass and morphological changes in response to physiological adaptations to chronic exercise. Consistent, intense exercise has been linked to cardiac remodeling, particularly in the athlete's heart. The athlete's heart, as it is called, is the physiological condition of someone who regularly exercises over a long, consistent period (4). Typically, these individuals are asymptomatic. Even as inpatients, heart problems are not the primary reason for their admission. Structural changes in the heart are discovered incidentally during a physical examination or diagnostic imaging (echocardiography, CT scan, MRI, or other chest imaging).

Cardiovascular adaptations to exercise have been studied in two categories: endurance training and strength training. According to Mitchell's classification, exercise can be categorized as high or low intensity, i.e., dynamic or static. Endurance training is described as dynamic, isotonic, or aerobic exercise, such as long-distance running or swimming. Strength training is described as static, isometric, or resistance exercise, such as wrestling, weightlifting, or throwing heavy objects. Furthermore, there are only a few sports that combine the two, such as cycling and rowing (5). The duration, type, volume, frequency, and duration of training have been shown to be major factors in an athlete's cardiac development.

Veteran athletes who performed endurance exercise at 70% of their maximum heart rate for at least 1 hour, 3 times a week for 5 years, did not exhibit significant left ventricular chamber dilation or left ventricular hypertrophy. In fact, a minimum of 6 hours of weekly exercise is recommended to maintain diastolic function and improve left atrial reservoir function (6). Morphological adaptations of the heart in sedentary young individuals were found after one year of intensive and prolonged endurance training. The observed adaptations resembled concentric hypertrophy in the first 6-9 months and eccentric hypertrophy at the end, but did not reach the level of an athlete's heart (7).

Although it's called athlete's heart, athletes aren't the only ones affected by this condition. Non-athletes who exercise regularly can also experience cardiac remodeling. However, strength, activity level, and duration of exercise play a role as external factors. The objective of this scoping review is to systematically map and synthesize empirical evidence regarding how exercise intensity and duration influence cardiac physiological responses in athletes, and to identify health promotion strategies that support safe and effective training practices.

METHOD

Identifying research questions

Question development is an important step that forms the basis of the entire review protocol in determining the search strategy, inclusion and exclusion criteria and data extraction. This review uses the Population, Exposure, Outcome, Study Design (PEOS) framework to help identify key concepts in the focus of the review. The PEOS framework can be seen in the table below. Guided by the PEOS framework, this review specifically addresses the following question: *'What is known from existing research about the cardiovascular adaptations associated with varying exercise intensities and durations among athletes, and how can these findings inform health promotion strategies for training management?* **'"**

Table 1. PEOS Framework

P (Population)	E (Exposure)	O (Outcome)	S (Study Design)
Athletes with intensive physical activity.	Intensity and duration of physical exercise.	Physiological response of the heart. Exercise and cardiovascular response. Exercise-induced cardiovascular disease. Portion of heart and heart training for athletes.	All research studies/study designs are related to athletes or to intensive physical activity.

Identifying relevant articles

There are three steps in identifying relevant articles. The first step is to determine the database. The databases used are PubMed, Sciences Direct, EBSCO and Google Scholar. The second step is to determine the inclusion and exclusion criteria. Articles are filtered according to the inclusion criteria published in 2000-2024, articles published in English, primary research articles (original research), and no specific country criteria, cross-sectional and descriptive research methods, retrospective, case analysis, and meta-analysis that describe data on athletes or to intensive physical activity, and describe the causes and procedures for its implementation. The exclusion criteria are opinion articles, peer reviews, theses manuscripts and theses. The third step is to determine the keywords that are focused on based on the framework, expanded by determining the Thesaurus and Boolean synonyms (can be seen in table 2).

Table 2 Article search keywords

Database	Keyword Search
PubMed	(athletes with intensive physical activity) OR ("intensity and duration of physical exercise") AND ("physiological response of the heart") ("exercise and cardiovascular response") ("exercise-induced cardiovascular disease") ("portion of exercise and heart of athletes")
Science Direct	(athletes with intensive physical activity) OR ("intensity and duration of physical exercise") AND ("physiological response of the heart") ("exercise and cardiovascular response") ("exercise-induced cardiovascular disease") ("portion of exercise and heart of athletes")
EBSCO	(athletes with intensive physical activity) OR ("intensity and duration of physical exercise") AND ("physiological response of the heart") ("exercise and cardiovascular response") ("exercise-induced cardiovascular disease") ("portion of exercise and heart of athletes")
Google Scholar	(athletes with intensive physical activity) OR ("intensity and duration of physical exercise") AND ("physiological response of the heart") ("exercise and cardiovascular response") ("exercise-induced cardiovascular disease") ("portion of exercise and heart of athletes")

Selection/choice of articles

The screening process is used to assess the relevance of research identified in the search according to the desired literature characteristics. From the search using 4 databases as many as 512 articles. Then the articles are filtered based on duplication, abstract and title and full text reading. So that 20 articles were found to be reviewed.

Furthermore, a critical appraisal was carried out using the Quality Assessment tool from Hawker and the classification of the total quality of the article and the article screening process are stated in PRISMA. The flowchart in Figure 1.

Data Charting

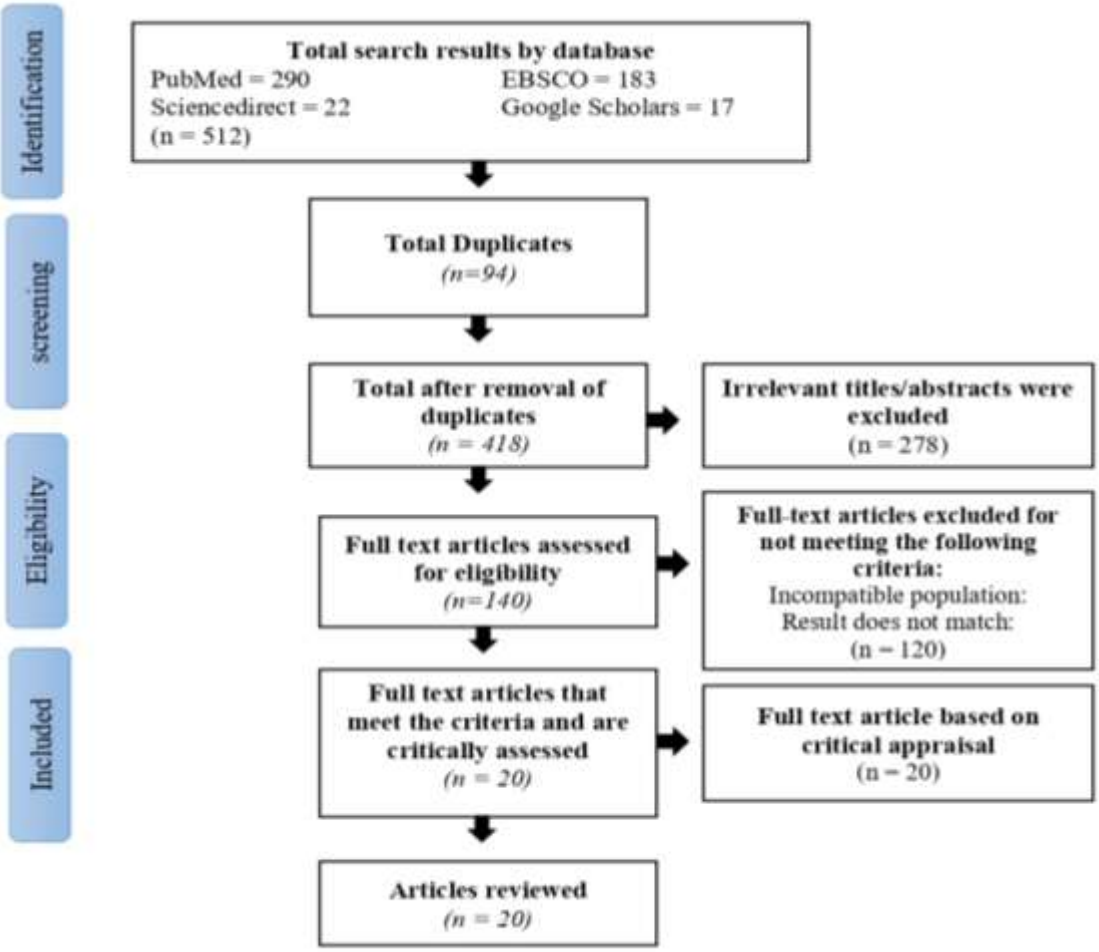

Figure 1. PRISMA Flowchart

RESULT

20 articles result from PRISMA flowchart, from four (4) database including PubMed, ScienceDirect, Google scholar, and EBSCO,

Table 3. Examples of Moderate and High Intensity Aerobic Exercises (16)

Moderate Intensity Aerobic Exercise	High Intensity Aerobic Exercise
Brisk walking (>3 miles/hour)	Uphill or fast walking
Cycling (<10 mph)	Cycling (>10 miles/hour)
Water gymnastics	Running or jogging
Tennis (doubles)	Tennis (singles)

Table 4. Fundamental Differences Between Athlete's Heart and Pathological Hypertrophy

	Athlete's heart	**Pathological hypertrophy**
Etiology	Adaptation to chronic exercise	Hypertension, heart valve disease, sarcomere gene mutations, infarction, etc.
Function	Normal or improved heart function	Cardiac dysfunction
Fibrotic growth	No fibrosis	Fibrosis
Gene expression	Normal gene expression	Myocyte necrosis and apoptosis
Proportional morphology	Proportional room enlargement	Proportional or disproportionate
Disease	There isn't any	Associated with heart failure

Table 5. Literature Synthesis on the Topic of Cardiac Physiological Response to Exercise Intensity and Duration in Athlete's Heart Syndrome

No	**Author/Year**	**Article Name**	**Research Purpose**	**Research Method**	**Sample**	**Result**
1	Arbad-Zadeh et al., 2004 (7)	Cardiac remodeling in response to 1 year of intensive endurance training	To investigate whether prolonged, high-intensity endurance training induces structural and functional cardiac remodeling	Longitudinal study	12 previously inactive healthy individuals (7 men, 5 women; mean age 29±6 years).	1. Increased LV mass secondary to increased wall thickness (concentric hypertrophy) in the first 6 months with low-intensity exercise 2. Increases in LVED volume only occur after 6-9 months of progressive training (eccentric hypertrophy) 3. Increase in RV mass and volume after resistance training (eccentric hypertrophy)

2	Petek BJ et al., 2022	Cardiac effects of detraining in athletes A narrative review	Effects of detraining on cardiac adaptation	Narrative review	Athlete	LV mass decreased after detraining
3	Mozafary Bazargany MH et al., 2024	Value of cardiac magnetic resonance feature-tracking in Arrhythmogenic Cardiomyopathy (ACM): A systematic review and meta-analysis	CMR feature-tracking on AMC and athletes	Systematic review	16 studies	Strain and dyssynchrony measures analyzed by CMR-FT have the potential to enhance ACM diagnosis in special populations, such as athletes, ACM patients with preserved EF, and those in the grey zone.
4	Matsuo et al., 2020 (39)	Effects of a Low-Volume Aerobic-Type Interval Exercise on VO_{2max} and Cardiac Mass	To compare the effects of low-volume aerobic interval training on cardiorespiratory capacity (VO_2max) and left ventricular (LV) mass compared to traditional continuous aerobic training in sedentary individuals.	Randomized experimental design	42 healthy but inactive men (mean age 26.5 ± 6.2 years)	There was a slight increase in LV mass during aerobic and sprint type interval training, but it was not significant.
5	Burger AL. et al., 2024	Impact of an Ultra-Endurance Marathon on Cardiac Function in Association with Cardiovascular Biomarkers	Effects of ultra endurance on the heart and biomarkers	Prospective observational	15 athletes	Troponin increases RV function decreases temporarily
6	Zujko-Kowalska K. et al., 2024	Revew: Detraining among Athletes—Is Withdrawal of Adaptive Cardiovascular Changes a Hint for the Differential Diagnosis of Physically Active People?	Detraining for differential diagnosis	Narrative review	Athletes and cardiomyopathy patients from previous studies	LV mass decreased significantly

7	Spence et al., 2011. (42)	A prospective, randomized, longitudinal MRI study of left ventricular adaptations to endurance and resistance training in humans	To examine how left ventricular (LV) structure and function adapt to endurance versus resistance exercise training in humans using highly sensitive cardiac MRI, and to test the validity of the "Morganroth hypothesis."	Randomized experimental study	23 untrained young participants	LV mass and LVED volume in resistance training increased significantly compared to resistance training.
8	Aengevaeren VL et al., 2018	Right Heart Remodeling in Olympic Athletes During 8 Years of Intensive Exercise Training	Assessing RV changes in Olympic athletes after 8 years of intensive training	Longitudinal cohort	40 elite athletes	Increased RV and RA without dysfunction
9	Czimbalmos C. et al., 2018	Cardiac Magnetic Resonance-Based Deformation Imaging: Role of Feature Tracking in Athletes with Suspected Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC)	Differentiating physiological versus pathological remodeling in athletes	Cross-sectional	76 total (34 ARVC, 34 healthy athletes, 8 athletes with ARVC)	RV strain increased, without fibrosis
10	Caselli & Pelliccia, 2018	The athlete's heart: a contemporary appraisal. Progress in Cardiovascular Diseases	Assessing LV geometry in athletes	Observational	100 professional athletes	LV mass increased but function normal
11	Swoboda PP et al., 2019	Regression of Left Ventricular Mass in Athletes Undergoing Complete Detraining Is Mediated by Decrease in Intracellular but Not Extracellular Compartments	Assessing LV mass regression after detraining	prospective Longitudinal	23 athletes	Significant decrease in LV mass
12	D'Andrea A. et al., 2021	Review: The Role of Multimodality Imaging in Athlete's Heart Diagnosis: Current Status and Future Directions	Assessing the role of multimodal imaging in the diagnosis of athlete's heart	Narrative review	Elite athlete	Combination of imaging improves diagnostic accuracy

13	Kübler J. et al., 2021	Cardiac MRI Findings to Differentiate Athlete's Heart from Hypertrophic, Arrhythmogenic, and Dilated Cardiomyopathy	Comparing CMR of athletes versus HCM/DMC	Cross-sectional	40 athletes and patients (HCM=14, ARVC=18, DCM=48)	Athlete: no LGE (fibrosis)
14	Dalos D. et al., 2022	Cardiac Remodeling in Ambitious Endurance-Trained Amateur Athletes Older Than 50 Years – An Observational Study	Looking at the effects of endurance training in people aged > 50 years	Observational	45 trained amateur athletes and 24 trained participants	LV and RV physiological dilation
15	La Gerche A. et al., 2022	The Athlete's Heart — Challenges and Controversies	A review of the challenges of cardiac diagnosis	Narrative review	Endurance athlete	RV structural changes are often reversible.
16	Zholshybek N. et al., 2023	Cardiac imaging in athlete's heart: current status and future prospects	Review of current imaging for the athlete's heart	Systematic review	Endurance athlete	Adaptation variations depending on the type of exercise
17	Bryde R. et al., 2021	Cardiac Structure and Function in Elite Female Athletes: A Systematic Review and Meta-Analysis	Meta-analysis of cardiac structure in female athletes	Meta-analysis	25 studies n = 1200	Gender affects cardiac response
18	Bernhard B. et al., 2021	Cardiac magnetic resonance imaging characteristics for the differentiation of athlete's heart from inherited cardiomyopathies	CMR for differential diagnosis of HCM/ARVC	Narrative review	80 athletes	Altel T1 normal; patient: T1 is high
19	Claessen G. et al., 2023	Reduced Ejection Fraction in Elite Endurance Athletes: Clinical and Genetic Overlap With Dilated Cardiomyopathy	Examining low EF in endurance athletes	Observational	281 elite endurance athletes	EF slightly decreased, but normal function
20	Vitiello D. et al., 2021	Review: Marathon-Induced Cardiac Fatigue: A Review over the Last Decade for the Preservation of the Athletes' Health	The effect of marathon on heart function	Systematic review	Athlete	Temporary RV and LV fatigue

DISCUSSION

Physiological Response of the Heart to Exercise

Physical exercise, particularly endurance training, induces various physiological adaptations in the cardiovascular system known as cardiac remodeling. This process is the result of structural and functional adjustments of the heart to the increased hemodynamic load that occurs during repeated physical activity. Arbab-Zadeh et al. (2004) reported that in healthy, previously inactive individuals, there was an increase in left ventricular (LV) mass, preceded by concentric hypertrophy during the first six months of low-intensity exercise, followed by eccentric hypertrophy after six to nine months of progressive exercise. These adaptations reflect the heart's response to the combined increase in blood pressure and blood volume resulting from repeated physical activity (7).

These findings were reinforced by Spence et al. (2011), who demonstrated that endurance training resulted in significant increases in LV mass and volume compared with resistance training. Aengevaeren et al. (2018) also found increases in LV and right ventricular (RV) volume in Olympic athletes after eight years of intensive training without any evidence of systolic dysfunction. This phenomenon suggests that cardiac remodeling in athletes is physiological and reflects an adaptive mechanism to improve cardiac pumping efficiency and oxygen delivery capacity to peripheral tissues. A similar finding was expressed by Caselli and Pelliccia (2018), who introduced the concept of athlete's heart, where increased LV mass in professional athletes is not accompanied by impaired systolic function. Matsuo et al. (2020) added that both aerobic interval training and continuous aerobic training can increase LV mass, although these increases are not always statistically significant. Thus, the degree of cardiac adaptation is highly dependent on the duration, intensity, and type of exercise performed (8,9,10,11).

Histologically and molecularly, the heart muscle (myocardium) is composed of myocytes, blood vessels, and an interstitial collagen matrix. Cardiac remodeling is characterized by changes in the proportions and composition of these compartments. Remodeling can occur in response to pathological stress, such as myocardial ischemia or infarction, or as a physiological adaptation to activity such as exercise. In the context of endurance training, increased cardiac output and blood volume cause repeated stretching of the ventricular wall, triggering the activation of various molecular signaling pathways that regulate cardiac cell growth and tissue reorganization (4,9,10).

The main factors involved in cardiac remodeling include oxidative stress and energy metabolism. An imbalance between oxygen demand and supply can reduce the energy available for ATPase proteins, thereby increasing the production of reactive oxygen species (ROS). The accumulation of ROS leads to lipid peroxidation, fibroblast proliferation, DNA damage, matrix metalloproteinase activation, and apoptosis stimulation. These processes influence calcium ion transit and the activation of hypertrophy signaling pathways. Under conditions of chronic pressure overload, sarcomeres are added in parallel, resulting in concentric hypertrophy. Conversely, volume overload causes serial sarcomeres to be added, resulting in eccentric hypertrophy (4,6).

Interestingly, in physiological hypertrophy, such as that seen in endurance athletes, eccentric or concentric hypertrophy patterns can also occur, but without contractile dysfunction. This distinguishes physiological hypertrophy from pathological hypertrophy. These adaptations are beneficial in improving cardiac function and hemodynamic efficiency. Multiple molecular pathways involved in exercise-induced cardiac remodeling include activation of insulin-like growth factor-1 (IGF-1), phosphorylation of PI3K (phosphatidylinositol 3-kinase), and AKT (protein kinase B), all of which play a role in increasing cardiomyocyte proliferation and growth. In fact, short-term aerobic exercise has been shown to reprogram cardiac remodeling through increased AKT activity (4,6,10).

According to Mitchell's classification, exercise is distinguished by two main components: the dynamic component (measured by maximal oxygen uptake or $MaxO_2$) and the static component (measured by maximal voluntary muscle contraction or MCV). The combination of these two components results in four main categories: low dynamic–low static (LD–LS), low dynamic–high static (LD–HS), high dynamic–low static (HD–LS), and high dynamic–high static (HD–HS). LD–LS exercises, such as golf or billiards, generally do not show significant cardiac adaptations and are often used as control groups. In contrast, HD–HS exercises, such as rowing, cycling, and triathlon, generate high volume and pressure stress, resulting in the most pronounced cardiac remodeling (5,12,26).

Dynamic exercises, such as long-distance running and swimming, increase cardiac output and decrease peripheral vascular resistance, creating a volume challenge for the ventricles. Meanwhile, static exercises such as weightlifting increase vascular resistance without significantly increasing cardiac output, creating a pressure challenge. Chronically, this combination of pressure and volume load induces enlargement of both ventricles and the

left atrium, as reported in professional rowers and cyclists. Approximately 75% of the variation in cardiac chamber size is related to non-genetic factors such as body size, sport category, sex, and age (9,12,25,26).

In aerobic exercise, particularly at moderate to high intensity (3–6 METs and ≥6 METs), increased oxygen demand stimulates an increase in cardiac output within the first few minutes, reaching a steady state. Increased end-diastolic volume increases stroke volume, while active muscle vasodilation decreases total peripheral resistance. Shear stress from increased blood flow stimulates the endothelium to release nitric oxide (NO), which improves vascular function. Furthermore, mechanical stretching of cardiac myocytes during exercise increases the release of humoral factors such as angiotensin II, endothelin-1, cardiotrophin-1, IGF-1, and TGF-β, which play a role in cardiac cell growth and adaptation (4,9,10,12).

Low Dynamic Low Static (LD-LS) Non-athletes (all workouts require <40% MaxO2 and <20% MVC) Billiards Bowling Golf Cricket Curly	**Low Dynamic High Static (LD-HS)** (All exercises require <40% MaxO2 and >50% MVC) Martial arts Gymnastics Climbing Weightlifting Water skiing
High Dynamic Low Static (HD-LS) (All exercises require >70% MaxO2 and <20% MVC) Badminton Hiking Long distance running Tennis Football	**High dynamic High static (HD-HS)** (All exercises require >70% MaxO2 and >50% MVC) Boxing Canoeing Bicycle Rowing Triathlon

Figure 2. Sports classification (5)

Cardiovascular Adaptations to Training and Endurance and Resistance

Cardiovascular adaptations to training depend on the characteristics of the training stimulus. Endurance training, which emphasizes increasing volume load, tends to induce eccentric hypertrophy, whereas resistance training, which increases pressure load, produces concentric hypertrophy. Spence et al. (2011) suggested that endurance training has a greater impact on increasing LV volume and mass than resistance training, consistent with the Morganroth hypothesis (8).

Research by Burger et al. (2024) showed that ultra-endurance training can cause increased troponin levels and a temporary decrease in RV function, indicating cardiac fatigue. However, a systematic review by Vitiello et al. (2021) found that this cardiac fatigue is transient and reversible, confirming that the athlete's heart is capable of adapting to extreme physiological stress (14).

Conversely, detraining indicates a regression of cardiac adaptations. Swoboda et al. (2019) and Petek et al. (2022) reported a significant decrease in LV mass after a period of detraining (15,16). Zujko-Kowalska et al. (2024) also showed that LV mass decreased significantly after detraining, confirming that cardiac structural adaptations are highly dependent on the continuation of the training stimulus (17). Thus, cardiovascular adaptations to training are dynamic and reversible depending on the intensity and duration of the training.

Cardiovascular Diseases Caused by Exercise

One of the key issues in sports cardiology is distinguishing between physiological cardiac remodeling that occurs as a result of intense training and pathological changes that indicate the presence of cardiovascular disease, such as hypertrophic cardiomyopathy (HCM) and arrhythmogenic right ventricular cardiomyopathy (ARVC). This condition is known as athlete's heart, where the heart undergoes morphological and functional adaptations due to repeated increased workload during high-intensity physical activity. Czimbalmos et al. (2018) reported that elite athletes showed increased right ventricular (RV) strain without myocardial fibrosis, so that ventricular enlargement and increased mass are considered a form of physiological adaptation, not a sign of cardiomyopathy (1).

Other studies by Kübler et al. (2021) and Bernhard et al. (2021) using cardiac magnetic resonance (CMR) confirmed these findings by showing that athletes did not exhibit late gadolinium enhancement (LGE) or increased T1 values, two parameters typically indicative of myocardial fibrosis in patients with cardiomyopathy. D'Andrea et al. (2021) emphasized the importance of a multimodal approach combining three-dimensional echocardiography, CMR, and strain imaging to distinguish physiological remodeling from pathological conditions with high accuracy (4).

However, the adaptation of an athlete's heart can pose a diagnostic dilemma, particularly because its symptoms mimic several forms of cardiomyopathy, such as HCM, dilated cardiomyopathy (DCM), and ARVC. An electrocardiogram (ECG) often shows similar findings, such as left ventricular enlargement or ambiguous T-wave changes. To differentiate between the two, physicians can perform a deconditioning test, which involves temporarily stopping exercise to assess for a decrease in cardiac mass or assess for an absolute left ventricular (LV) diastolic dimension greater than 55 mm, as an indicator of athlete's heart. However, these tests cannot be performed in an emergency setting and require further evaluation with other non-invasive imaging technologies.

Exercise intensity is also an important factor influencing the risk of cardiovascular disease. Moderate-intensity exercise requires an energy expenditure of 3.0–5.9 METs, while high-intensity exercise requires ≥6 METs. Epidemiological studies have shown that increasing the volume of moderate-intensity exercise consistently reduces the risk of cardiovascular disease and mortality, while very high-intensity exercise (>11 METs/week) provides no significant additional benefit and can potentially lead to excessive cardiac stress (6–8). At the cellular level, the increased mechanical load on the myocardium during strenuous exercise triggers the release of various humoral factors such as angiotensin II, endothelin-1 (ET-1), cardiotroponin-1 (CT-1), insulin-like growth factor-1 (IGF-1), and transforming growth factor-β (TGF-β). These factors induce intracellular signaling pathways that promote myocardial cell hypertrophy, an adaptive process that, under extreme conditions, can progress to pathological changes (4).

Anatomically, the greatest changes in cardiac chamber size and wall thickness were observed in athletes in sports with a combination of high-dynamic and high-static activity, such as rowing, skiing, cycling, and swimming. Multivariate analysis showed that approximately 75% of the variation in cardiac chamber size was influenced by non-genetic factors such as body size, sport category, gender, and age. Left atrial remodeling commonly occurs in athletes with high-static and high-dynamic sports, such as cycling and rowing. La Gerche et al. (2022) reported that changes in right ventricular (RV) structure due to endurance training were reversible and not indicative of cardiovascular pathology (22).

In addition to structural changes, exercise can also trigger arrhythmias. Both athletes and non-athletes can experience bradyarrhythmias (slow heart rate) and tachyarrhythmias (fast heart rate) in response to increased physical activity. A large-scale study by Furtado and Araujo found that 43% of respondents experienced arrhythmias during cardiopulmonary exercise testing (CPET), with supraventricular extrasystoles being the most common type. Approximately 3% of respondents experienced polymorphic ventricular extrasystoles, which have a poor prognosis and can precede complex ventricular arrhythmias. Meanwhile, athletes with high levels of dynamic training often exhibit sinus bradycardia, first-degree AV block, left ventricular hypertrophy (LVH), or ST-segment elevation on the ECG, which generally reflect increased physiological vagal tone (23).

Sudden cardiac death (SCD) in athletes is a rare but nonetheless concerning phenomenon. Although most cases of cardiac remodeling do not result in sudden death, prolonged, extreme exercise can increase cardiovascular risk in certain individuals. Therefore, pre-competition evaluation with a 12-lead electrocardiogram is highly recommended to detect abnormalities such as long QT syndrome, HCM, ARVC, or Marfan syndrome (14). However,

interpretation of these results requires caution because the distinction between physiological adaptations and pathology is often difficult to ascertain.

Exercise-related emergencies have also been reported, including cardiac arrest, palpitations, heatstroke, hypovolemic shock, and severe dehydration. One interesting example is a case of polymorphic ventricular tachycardia that occurred after performing a treadmill test using the modified Bruce protocol. Initially, the ECG showed several multiform premature ventricular complexes (PVCs), but after three minutes of jogging, polymorphic ventricular tachycardia appeared, which disappeared immediately after the exercise was stopped. Coronary angiography was normal, but genetic analysis revealed a mutation in the RYR2 gene, which plays a role in the mechanism of Catecholaminergic Polymorphic Ventricular Tachycardia (CPVT) (16).

During strenuous exercise, elevated serum catecholamines can trigger excessive intracellular calcium ion release through increased cyclic adenosine monophosphate (cAMP), causing delayed afterdepolarization and disruption of the cell's refractory period. This condition facilitates reentry and abnormal automaticity that can trigger severe arrhythmias such as Right Ventricular Outflow Tract Ventricular Tachycardia (RVOT-VT), ARVC, CPVT, and even acute coronary syndrome (16). In addition to RYR2 gene mutations, mutations in CASQ2, which plays a role as a major store of calcium ions in the sarcoplasmic reticulum, have also been linked to CPVT (17). Although patients with CPVT often have normal cardiac morphology and ECG, they remain at high risk of sudden cardiac death during exercise (14,17).

A large multicenter study showed that the difference between peak heart rate and heart rate 1–2 minutes after stopping exercise, especially if the difference reaches ≥80% of the predicted maximum heart rate, can help predict the possibility of symptomatic CPVT, making this evaluation important in the assessment of athletes with symptoms of palpitations (16,17).

Proportions and Principles of Cardiac Training in Athletes

Cardiac adaptation to exercise is a complex process involving structural, functional, and molecular changes, known as cardiac remodeling. This process is greatly influenced by the intensity, frequency, duration, and type of exercise performed. The principle of progressive overload is the basis for achieving optimal cardiac adaptation without causing myocardial damage. Arbab-Zadeh et al. (2004) reported that physiological cardiac remodeling develops gradually through progressive exercise stimuli over several months, whereby gradually increasing cardiovascular workload stimulates balanced growth and adjustment of cardiac structure (7).

Research by Dalos et al. (2022) showed that in amateur athletes over 50 years of age, resistance training still provided benefits in the form of physiological dilation of the left ventricle (LV) and right ventricle (RV) without significant systolic dysfunction. (6) These findings reinforce the idea that cardiovascular adaptations to training can occur in older adults, as long as the training intensity is adjusted to individual capacity. Meanwhile, Bryde et al. (2021) emphasized that cardiac responses to training differ by gender, with female athletes experiencing a milder increase in heart mass and volume than male athletes, likely due to hormonal influences and body composition (25). Therefore, designing training programs should consider individual biological factors such as age, gender, and underlying health conditions (3).

In general, a combination of moderate-intensity endurance training for at least 150 minutes per week and strength training two to three times per week is recommended to maintain optimal cardiovascular function (1). In professional athletes, it is important to maintain a balance between the loading and recovery phases to prevent overtraining syndrome and long-term myocardial fatigue (14,16).

Exercise-induced cardiac remodeling is multifactorial. Influencing factors include the type of exercise, training volume, intensity, frequency, duration, body surface area, gender, age, comorbidities, and genetic factors (4). The duration of training plays a major role in the rate of cardiac adaptation. Increases in left ventricular mass and wall thickness generally begin to appear after six months of endurance training, while increases in left ventricular end-diastolic diameter (LVED) can be detected after three months of consistent training (7).

LVED volume is influenced by diastolic filling, intrinsic myocardial relaxation, ventricular filling pressure, and ventricular wall compliance. LVED volume can be measured using echocardiography or cardiac magnetic resonance imaging (CMR).(20) In a population of patients with type 2 diabetes, exercise duration of approximately nine months did not cause significant changes in cardiac biomarkers such as high-sensitivity cardiac troponin T (hs-

cTnT) and N-terminal pro–B-type natriuretic peptide (NT-proBNP), indicating that this period is still too short to trigger significant structural changes in the heart (13).

Age is an important determinant of cardiac remodeling. In children aged 12–14 years, resistance training increases VO_2max and ventilatory threshold (VT) by approximately 9%, accompanied by an increase in left ventricular diameter without significant wall thickening (11). This mechanism is explained by the Frank–Starling law, where increased venous return during aerobic exercise increases left ventricular preload and stroke volume (9). Left ventricular wall thickening generally occurs more slowly, as it relies on hormonal support such as testosterone, which is not yet optimal at a young age (25).

Comorbidities also influence cardiac adaptation. Cohort studies of populations at cardiovascular risk have shown increased B-type natriuretic peptide (BNP) levels after prolonged moderate-intensity brisk walking (3). However, regular exercise is still recommended because its benefits on endothelial function and aerobic capacity outweigh the risks of transient increases in these biomarkers (1,13).

According to Arbab-Zadeh et al. (2004), concentric hypertrophy typically occurs within the first six months of low-intensity exercise in response to increased blood pressure and afterload (7). In contrast, long-term endurance exercise causes eccentric hypertrophy, characterized by proportional increases in left and right ventricular volume and mass (8). Left ventricular mass index and relative wall thickness (RWT) are used to determine ventricular geometry: RWT $\leq$0.42 indicates eccentric hypertrophy, while RWT >0.42 indicates concentric hypertrophy (10).

The type of exercise also plays a role. Exercise with high dynamic–high static (HD–HS) components, such as rowing and cycling, produces the greatest increases in ventricular mass and volume compared to other forms of exercise. (12) Vogelsang et al. reported a 13% increase in left ventricular mass after eight weeks of 30-minute rowing exercise, three times per week, accompanied by increases in end-diastolic volumes of both ventricles. HD–HS training provides a synergistic combination of pressure and volume loading, resulting in balanced biventricular remodeling (9).

High-intensity aerobic exercise such as uphill running or long-distance cycling (HD–LS) can also induce eccentric hypertrophy, although it takes longer than HD–HS exercise (10). Meanwhile, moderate-intensity exercise, such as jogging 30 minutes three times per week for three months, is often not enough to cause significant changes in LVED volume. However, an increase in myocardial mass can still occur as part of a gradual physiological adaptation (8,11,27-29).

CONCLUSION

Physical exercise, particularly endurance and high-intensity dynamic-static combination training, triggers complex physiological adaptations in the cardiovascular system through cardiac remodeling. These adaptations involve physiological structural, functional, and molecular changes in the heart, characterized by an increase in ventricular mass and volume without impairment of systolic function. The pattern of cardiac hypertrophy depends on the type of training stimulus concentric hypertrophy results from pressure loading, while eccentric hypertrophy results from volume loading. Factors such as intensity, duration, frequency, age, gender, and training status significantly influence the degree of these adaptations.

Physiological remodeling in athletes must be distinguished from pathological conditions such as hypertrophic cardiomyopathy and arrhythmogenic right ventricular cardiomyopathy, as both can present with similar morphological features but have different clinical implications. Excessive exercise at extreme intensities can lead to cardiac fatigue, arrhythmias, or even sudden cardiac death, although these events are rare and generally reversible.

Thus, regular, progressive, and individual-appropriate physical exercise has been shown to provide optimal benefits for heart health. The principle of progressive overload and the balance between training and recovery phases are key to maintaining physiological cardiac remodeling, increasing heart pumping efficiency, and preventing the pathological effects of excessive cardiovascular stress.

AUTHOR'S CONTRIBUTION STATEMENT

Conceptualization: DNK, IP, EKG. Data curation: MT, NN and YY. Investigation: DNK, NN and EKG. Methodology: DNK, IP, MT, EKG, Project administration: DNK, YY, IP and EKG. Supervisors: IP, MT and NN Validation: DNK, YY, and EKG. Writing—original draft: All authors Writing—review & editing: All authors.

CONFLICTS OF INTEREST

The authors declare that they have no conflict of interest

DECLARATION OF GENERATIVE AI AND AI-ASSISTED TECHNOLOGIES IN THE WRITING PROCESS

The authors declare that Generative Artificial Intelligence (AI) and AI-assisted technologies were utilized solely to improve the clarity, grammar, structure, and readability of this article. All ideas, analyses, interpretations, and conclusions presented in this manuscript are entirely the authors' original work.

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