



Structural Differences in Physiological Cardiac Hypertrophy Induced by Endurance and Resistance Exercise: A Systematic Review

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ABSTRACT

Regular physical exercise induces physiological cardiac hypertrophy, although the structural remodeling pattern differs according to exercise modality. This systematic review compared the structural characteristics of exercise-induced cardiac hypertrophy following endurance and resistance training in healthy humans and animal models. A systematic search of PubMed, ScienceDirect, and SpringerLink was conducted to identify studies published between 1 January 2020 and 10 March 2026, in accordance with the PRISMA 2020 guidelines. Eligible studies included randomized controlled trials and longitudinal interventions involving at least four weeks of endurance or resistance exercise with structural cardiac outcomes. Risk of bias was assessed using the ROBINS-I tool for human studies and the SYRCLE tool for animal studies. Findings were synthesized narratively because of methodological heterogeneity. Nine studies were included in this systematic review, comprising three human studies and six animal studies. All studies evaluating resistance training were conducted in human longitudinal cohorts. Endurance training predominantly induced eccentric remodeling, characterized by increased left ventricular mass and chamber dimensions with preserved relative wall thickness, although concentric and mixed phenotypes were also observed, particularly with higher training intensities. Resistance training generally produced concentric or mixed hypertrophy associated with pressure overload. Among studies reporting cardiac functional outcomes, systolic function was generally preserved. These findings provide preliminary evidence that exercise modality and intensity may influence cardiac remodeling and may help guide future research on individualized exercise prescriptions, particularly in human populations.

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INTRODUCTION

Regular physical exercise is a critical determinant of healthy cardiac aging, promoting structural and functional adaptations that counteract age-related cardiovascular decline (D'Silva & Sharma, 2015). As a physiological stimulus for cardiovascular health, physical exercise induces structural adaptations that enhance myocardial efficiency and resilience (Pittaras et al., 2023). Cardiac adaptation to regular physical exercise is essential for maintaining cardiovascular health and reducing the risk of heart disease (Seo et al., 2020). Exercise-induced cardiac remodeling is generally beneficial, characterized by myocardial enlargement that enhances cardiac function (Kim & Baggish, 2016). It is characterized by adaptive myocyte growth in sizes as exemplified by increases in left ventricular mass, chamber size, or wall thickness, while preserving systolic and diastolic function and generally absence of fibrosis or pathological remodeling (Cheng et al., 2025; Riehle et al., 2014). Distinguishing physiological exercise-induced cardiac remodeling from pathological cardiac hypertrophy is clinically important because exercise-related structural adaptations may overlap phenotypically with cardiomyopathies, particularly hypertrophic cardiomyopathy (HCM). Accurate differentiation is therefore essential to avoid misclassification of physiological adaptation as disease while ensuring that underlying pathological conditions are not overlooked, with implications for clinical evaluation (Hsieh et al., 2025a).

Different exercise modalities impose distinct mechanical and hemodynamic loads on the heart, leading to divergent patterns of cardiac remodeling. Exercise-induced cardiac hypertrophy exhibits distinct structural patterns determined by the relative contributions of dynamic and static exercise components (Martinez et al., 2021). Sporting disciplines can be generally separated as isotonic or endurance training and isometric or resistance training (Hsieh et al., 2025). Endurance exercise predominantly generates a chronic volume overload and is associated with calcium-dependent and energy-sensing pathways, promoting ventricular chamber enlargement and eccentric hypertrophy (Ellison et al., 2012; Muhl et al., 2008). Eccentric hypertrophy represents the typical response to volume overload associated with mechanosensitive growth signalling, including the IGF-1/PI3K–Akt–mTOR pathway, and characterized by ventricular dilation with preserved or reduced wall thickness (Toktarbay et al., 2023). This adaptation serves to maintain stroke volume despite increased preload (Pitoulis & Terracciano, 2020). In contrast, resistance exercise is associated with pressure overload due to transient elevations in arterial blood pressure, favouring increased ventricular wall thickness with relatively preserved chamber size, consistent with concentric remodeling (Cengiz, 2012).

Structural characterization of exercise-induced cardiac hypertrophy commonly relies on echocardiographic and imaging-based parameters that reflect left ventricular geometry. Exercise-induced cardiac remodeling may manifest as eccentric hypertrophy, concentric hypertrophy, or mixed remodeling. Eccentric hypertrophy is characterized by an increase in left ventricular mass (LVM) or left ventricular mass index (LVMI), accompanied predominantly by enlargement of the ventricular chamber, as reflected by an increased left ventricular end-diastolic diameter (LVEDD) or left ventricular end-diastolic volume (LVEDV), with relatively proportional myocardial wall growth (Patel et al., 2017). In contrast, concentric hypertrophy is characterized by increased LVM or LVMI accompanied by greater ventricular wall thickness, including interventricular septal thickness (IVST) and posterior wall thickness (PWT), with relatively preserved chamber dimensions (Shah, 2024). Mixed remodeling describes concurrent features of chamber enlargement and increased wall thickness that do not conform predominantly to either eccentric or concentric patterns (Banks et al., 2021).

Evidence from both human and animal studies supports exercise-induced physiological cardiac remodeling, although the structural response may vary according to training modality, intensity, duration, and individual characteristics. Human studies in athletes consistently demonstrate increased left ventricular mass compared with sedentary controls, with endurance training associated with larger ventricular chamber dimensions and volumes, indicative of eccentric remodeling. Longitudinal study demonstrates that prolonged intensive endurance training induces substantial and progressive cardiac remodeling in previously sedentary adults. Over one year of structured endurance training, left and right ventricular mass increased significantly, accompanied by improvements in stroke volume and cardiac output (Arbab-Zadeh et al., 2014). Resistance-trained athletes exhibit modest increases in wall thickness and mass without clear evidence of pathological concentric hypertrophy, suggesting overlapping and graded adaptations rather than distinct dichotomous patterns. Evidence from rodent models indicates that resistance exercise induces modality-specific and largely physiological cardiac adaptations without exacerbating adverse remodeling (Rodrigues et al., 2023). Together, human and animal evidence suggests that exercise-induced cardiac remodeling encompasses heterogeneous structural phenotypes, supporting the need to evaluate myocardial mass, chamber dimensions, and wall thickness collectively when characterizing exercise-related cardiac hypertrophy (Utomi et al., 2013).

Recent systematic review systematically compared endurance, resistance, high-intensity interval, and combined training across molecular, structural, and functional cardiovascular outcomes (Lanke Vani et al., 2025). However, the review was restricted to human studies and excluded animal models, while structural cardiac hypertrophy represented only one component of a broader cardiovascular assessment. Although exercise intensity was described as a characteristic of the training interventions, it was not examined as a predefined effect modifier of structural cardiac remodeling. Findings regarding concentric remodeling in resistance-trained individuals are inconsistent, with several studies reporting modest or absent wall thickening, challenging the traditional dichotomy between endurance and strength induced hypertrophy (Hellsten & Nyberg, 2016). The present systematic review addresses this gap by specifically examining structural cardiac hypertrophy induced by endurance and resistance exercise across both human and animal studies. Rather than evaluating cardiovascular adaptations broadly, this review focuses on structural indicators of myocardial remodeling, including left ventricular mass, chamber dimensions, wall thickness, and related measures used to characterize eccentric, concentric, or mixed remodeling. In addition, exercise intensity is considered in interpreting differences in structural adaptation.

This systematic review aimed to explore the structural characteristics of physiological cardiac hypertrophy induced by endurance and resistance exercise across human and animal studies. The review focused on key indicators of structural remodeling, including changes in myocardial mass, ventricular chamber dimensions, wall thickness, relative wall thickness, and other relevant structural parameters to characterize eccentric, concentric, or mixed remodeling patterns. By integrating evidence across species and training conditions, this review sought to clarify the extent to which cardiac structural remodeling differs between endurance and resistance exercise and to address limitations in the scope of previous reviews.

METHODS

This systematic review was conducted and reported in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 guidelines and registered in PROSPERO under separate registration numbers for human studies

(CRD420261364320) and animal studies (CRD420261364521) to enhance transparency and reproducibility of the review process (Page et al., 2021).

Search Strategy

A comprehensive literature search was conducted using PubMed, ScienceDirect, and SpringerLink to identify relevant studies published between 1 January 2020 and 10 March 2026. The search strategy was developed to capture two primary concepts, including exercise modalities and cardiovascular structural adaptations. A combination of keywords and Medical Subject Headings (MeSH) was used to ensure a sensitive and comprehensive search. Keywords within the same concept were combined using the Boolean operator “OR”, while different concepts were combined using “AND”. The search was restricted to articles published in English.

Study Selection

All records retrieved from the database searches were downloaded and imported into Rayyan reference management software for duplicate removal. After deduplication, two independent reviewers (JN and AD) screened all studies in a two-stage process. Initially, titles and abstracts were screened according to the predefined inclusion and exclusion criteria. Studies deemed potentially eligible were subsequently assessed through full-text review. Any disagreements between the two reviewers at either the screening or full-text assessment stages were resolved through discussion. If consensus could not be reached, a third reviewer (NS) independently evaluated the study to make the final decision.

Inclusion and Exclusion Criteria

Inclusion Criteria

Study eligibility was determined using the PECOS (Population, Exposure, Comparator, Outcomes, Study design) framework.

1. Population

Eligible studies included human adults aged ≥ 18 years and healthy animal models without pre-existing cardiovascular disease or pathological cardiac conditions. Human participants could include sedentary or recreationally active individuals, amateur athletes, and elite/professional athletes, provided that they met the predefined health criteria.

2. Exposure

Studies were required to investigate exercise interventions lasting at least 4 weeks. Exercise modalities included endurance exercise, defined as aerobic activities such as running or cycling, across all intensity levels and resistance exercise, defined as strength-based training such as weightlifting or ladder climbing, across all intensity levels.

3. Comparator

Studies were required to include a comparator group, including randomized or non-randomized comparator group (such as sedentary) and longitudinal pre-post comparison (such as baseline pre-intervention measurements).

4. Outcomes

Eligible studies reported quantitative structural cardiac outcomes can be assessed using echocardiography. Outcomes of interest included, but were not limited to, left ventricular hypertrophy, ventricular geometry, wall thickness, chamber dimensions, and vascular or ventricular remodeling. Eccentric hypertrophy was defined as

increased LVM or LVMI accompanied predominantly by ventricular chamber enlargement, reflected by increased LVEDD or LVEDV, with relatively proportional wall growth. Concentric hypertrophy was defined as increased LVM or LVMI accompanied predominantly by increased ventricular wall thickness, including interventricular septal thickness and posterior wall thickness, with relatively preserved chamber dimensions. Mixed remodeling was defined as the concurrent presence of chamber enlargement and increased wall thickness without a clearly predominant eccentric or concentric pattern.

5. Study Design

Eligible study designs included randomized controlled trials, in which participants or animals were randomly allocated to an exercise or comparator condition, non-randomized controlled trials, in which an exercise intervention was compared with a concurrent control without random allocation, and longitudinal studies, in which structural cardiac outcomes were measured before and after an exercise intervention without a concurrent control group.

Exclusion Criteria

Studies were excluded if they investigated combined or multimodal exercise interventions without isolating endurance or resistance exercise effects, focused on pathological cardiac hypertrophy or included participants/animals with cardiovascular disease, were published in languages other than English, were review articles, meta-analyses, editorials, conference abstracts, or commentaries, and provided insufficient data for quantitative analysis of structural cardiac outcomes.

Data Extraction and Analysis

Data extraction was performed independently by two reviewers (JN and AD) using a standardized data extraction form. Inter-reviewer agreement was high, with a concordance rate of $\geq 95\%$. Any discrepancies were resolved through discussion and consensus, with consultation from a third reviewer (NS) when necessary. Following consensus, one reviewer (JN) completed data extraction for all included studies. Extracted data were summarized in table form and included the author and year of publication, study design, sample characteristics, exercise modality and intervention details, comparator groups, and main findings related to cardiac structural outcomes. Data were synthesized narratively to compare modality-specific effects of endurance and resistance exercise on structural outcomes.

Quality Assessment

The methodological quality of included studies was independently assessed using validated risk of bias assessment tools. For human subjects, ROBINS-I tool was applied (Higgins JPT et al., 2024). For animal study, quality assessment was performed with SYRCLE (Hooijmans et al., 2014). This tool evaluated bias across the following domains: randomization process, deviations from intended interventions (including allocation concealment and blinding), missing outcome data, measurement of outcomes, and selection of the reported result. Each domain was rated as low risk of bias, some concerns, or high risk of bias. An overall risk of bias judgment was assigned for each study. Risk of bias assessment was conducted independently by two reviewers (JN and AD). Any discrepancies were resolved through discussion and consensus, with consultation of a third reviewer (NS) when necessary.

RESULTS

A total of 2,280 records were identified through database searches, including PubMed (n = 154), SpringerLink (n = 383), and ScienceDirect (n = 1,743). After removal of 43 duplicate records, 2,237 articles remained for screening. Title and abstract screening resulted in the exclusion of 2,174 studies, leaving 63 articles eligible for full-text assessment. Of these 63 articles, 54 studies were excluded due to failure to meet the inclusion criteria. Ultimately, 9 studies met all eligibility criteria and were included in this systematic review. The study selection process is summarized in the PRISMA flow diagram (Figure 1).

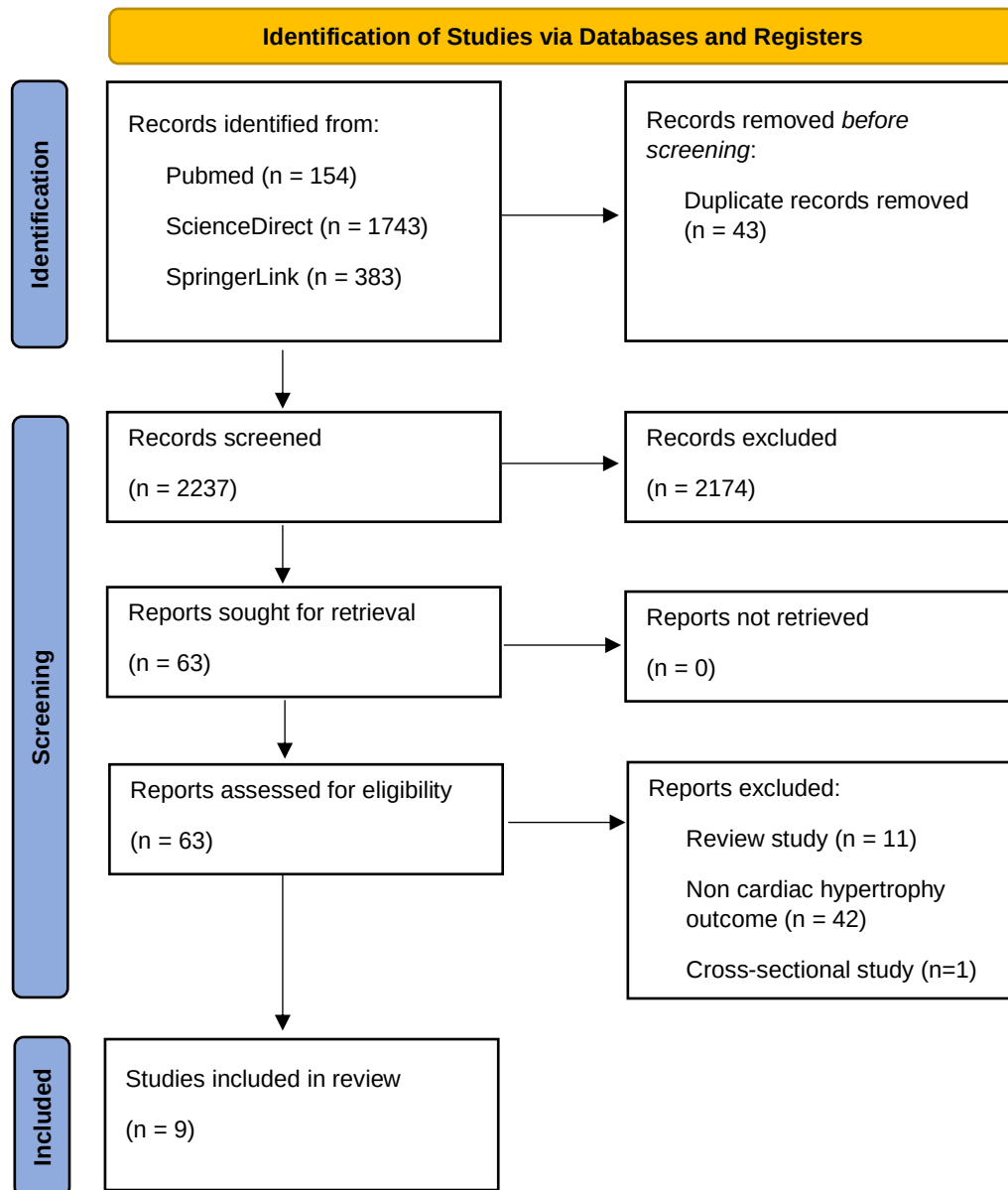


Figure 1. PRISMA Flowchart of The Study Selection Process

Adapted from Page MJ, McKenzie JE, Bossuyt PM, et al. The PRISMA 2020 statement: an updated guideline for reporting systematic reviews (Page et al., 2021).

Characteristics of Included Studies

A total of 9 studies met the inclusion criteria and were included in the qualitative synthesis (Figure 1). The included studies comprised human studies (n = 3) and controlled experimental

animal studies ($n = 6$) with results focusing on the cardiac structural adaptations to endurance or resistance training exercise modality. The sample size in human studies ranged from 8 to 24 healthy adults, predominantly amateur athletes or previously untrained individuals undergoing structured training programs. Animal studies primarily used rodent models (Wistar rats, Sprague–Dawley rats, and C57BL/6 mice), with sample sizes ranging from 14 to 72 animals and allocated to exercise and sedentary control groups.

In terms of exercise modality, endurance training was the most frequently investigated intervention ($n = 7$), including continuous aerobic exercise (treadmill running, swimming, voluntary wheel running) and high-intensity interval training (HIIT). Resistance training was examined in a smaller number of studies ($n = 2$), all of which were conducted in human participants. The duration of interventions ranged from 5 to 20 weeks. Across all studies, echocardiography was the primary method used to assess cardiac hypertrophy, focusing on left ventricular mass index, wall thickness, chamber dimensions, and systolic function. In several animal studies, echocardiographic findings were further supported by histological and molecular analyses to evaluate cardiomyocyte hypertrophy and associated signalling pathways. The results reported quantitative cardiac structural outcomes assessed by echocardiography, describing cardiac remodeling and related morphological changes as shown in Table 1.

Among the included studies, endurance exercise was generally associated with physiological left ventricular remodeling, with patterns ranging from predominantly eccentric features at low-to-moderate intensities to concentric or mixed features in some higher-intensity or HIIT protocols. Resistance training demonstrated structural hypertrophic adaptations characterized by concentric or mixed remodeling patterns. However, these modality-specific patterns should be considered preliminary given the limited number of resistance-training studies and the predominance of animal evidence.

Structural Changes

Across the included studies, exercise training consistently induced structural cardiac remodeling ($n=9$; 100%). However, the magnitude and pattern of these adaptations varied according to exercise modality, intensity, duration, and study population. Endurance exercise was the most frequently investigated modality ($n = 7$) and was generally associated with increases in left ventricular mass and chamber dimensions, with several studies demonstrating predominantly eccentric remodeling characterized by increased LV end-diastolic dimensions or volumes. However, some higher-intensity endurance or high-intensity interval training (HIIT) protocols demonstrated concentric or mixed hypertrophy. These findings suggest that the structural response to endurance exercise may extend beyond a simple eccentric remodeling pattern and may be influenced by exercise intensity and training duration. Several experimental endurance-training studies also reported structural findings that may indicate a transition from adaptive remodeling toward potentially adverse tissue responses under specific exercise conditions. In particular, high-intensity or prolonged exercise protocols were associated with increased collagen deposition or myocardial fibrosis in some animal models (Maleki & Mehrabani, 2024; Santos-Lozano, 2022; Yan et al., 2021). Resistance training was investigated in only two studies, both conducted in human participants. The reported structural responses were characterized by increases in myocardial mass and/or ventricular wall thickness, with patterns described as predominantly concentric or mixed remodeling. However, the limited evidence base for resistance training restricts the extent to which distinct modality-specific structural adaptations can be established.

The six animal studies evaluated endurance exercise and demonstrated a broader range of structural responses. Histological evaluations demonstrated increased cardiomyocyte cross-sectional area and myocardial mass (Medina et al., 2020; Yoshizaki et al., 2026). At the molecular level, exercise-induced cardiac remodeling was associated with activation of adaptive signalling pathways, including PI3K–AKT signalling (Yoshizaki et al., 2026), ERK1/2 pathways (Yan et al., 2021), and exercise-responsive microRNAs such as miR-1 and miR-133 (Fathi et al., 2020), which are known regulators of hypertrophy. Overall, the human and animal evidence demonstrated overlapping but heterogeneous structural responses to exercise. The available evidence was predominantly derived from animal models and endurance-training protocols, whereas evidence for resistance training was limited to two human studies.

Table 1. Outcome and Population Characteristic of Included Studies

Reference (Country)	Sample Size	Outcome Measures	Intervention		Frequency (days/week)/total duration (weeks)	Conclusion
			Control Group	Experimental Group		
Yoshizaki, A. et al., 2026 (Brazil)	72 rats	- Echocardiographic LV parameters - Hemodynamic assessment - Myocardial mechanics - Cardiomyocyte width and nuclear volume - Gene expression (PI3K/Akt pathway) - miR expression	UT	Endurance training: - Running (treadmill exercise) - Swimming (swimming exercise at 75% VO₂max)	5/8	Swimming training induced physiological cardiac eccentric hypertrophy with LVEDD and LVESD increased by 12% and 13% respectively
Venckunas et al., 2025 (Lithuania)	8 amateur athletes	- Echocardiographic LV cardiac structure (LV mass, LV wall thickness, interventricular septum thickness) - Cardiac function parameters	No control group (longitudinal study)	Endurance training: Volume progressive HIIT running program	3/7	Concentric cardiac hypertrophy with increase in LV wall thickness of 11% and LV mass of 24%
Pamart et al., 2025 (France)	22 UT males	Echocardiographic cardiac structure and function (LV mass, LV wall thickness, LV and RV end-diastolic volume)	No control group (longitudinal study)	High-intensity resistance training ≥70% 1-RM	3/20	Physiological and mixed remodeling characterized by increase in LV mass by 11% accompanied by increases in both LVEDD and LVESD (8-10%)
Maleki and Mehrabani, 2024 (Iran)	32 rats	- Echocardiographic cardiac structure in LV and RV - Histology (cardiomyocyte diameter, fibrosis)	UT	16 weeks intensive endurance exercise 12 weeks intensive + 4 weeks	5/16	Intensive endurance training induced physiological concentric RV hypertrophy

				moderate exercise • 12 weeks intensive + 4 weeks detraining		with RVPTW increased by 80% and eccentric LV hypertrophy with increased LVEDD by 46%
Grandperrin et al., 2024 (France)	24 UT adults	- Echocardiographic LV parameters - Body composition	No control group (longitudinal study)	Resistance training (70% 1-RM)	3/16	Physiological concentric LV remodeling with LVMi increased by 20-25% and RWT increased by 6-10%
Török et al., 2021 (Hungary)	32 rats	Echocardiographic LV parameters	UT	Endurance swimming exercise progressively increased until 200 min/day	5/12	Physiological concentric LV remodeling with increase in LVMi by 30-40% and LV wall thickness
Yan et al., 2021 (China)	72 rats	- Echocardiographic LV cardiac structure - Histology (cardiomyocyte diameter, fibrosis)	UT	- Low-intensity treadmill running - Medium-intensity treadmill running - High-intensity treadmill running	5/8	Low and moderate intensity induced LV eccentric hypertrophy with LVEDD increased by 10% and high-intensity induced mixed hypertrophy and accompanied by fibrosis
Medina et al., 2020 (Argentina)	25 mice	- Echocardiographic cardiac structure - Histology (Fibrosis) - Molecular analysis	UT	Endurance voluntary wheel running	Voluntary/ 5	Physiological cardiac remodeling with increase in LVMi, interventricular septal thickness, and posterior wall thickness
Fathi et al., 2020 (Iran)	14 rats	- Echocardiographic cardiac structure - Molecular analysis (miR-1, miR-133)	UT	Endurance treadmill training (75% VO ₂ max)	6/14	Eccentric physiological LV hypertrophy with increased LVESD by 25%

UT = untrained; LV= left ventricle; RV= right ventricle; LVEDD= left ventricular end-diastolic diameter; LVESD= left ventricular end-systolic diameter; RVPTW= right ventricular posterior wall thickness; LVMi= left ventricular mass index; RWT= relative wall thickness; miR= microRNAs; RM= repetition maximum; HIIT= high-intensity interval training; VO₂max= maximum oxygen consumption; PI3K/Akt= phosphoinositide 3-kinase/protein kinase B.

Endurance Exercise Induced Structural Cardiac Remodeling

Across the 7 studies of endurance exercise protocols, endurance training produced physiological cardiac remodeling that varied according to exercise intensity. Endurance based interventions including swimming, treadmill running, voluntary wheel running, and high-intensity interval training (HIIT) were performed for approximately 5–16 weeks with sessions lasting about 60 minutes per day and intensities ranging from 60–85% VO_2max (Maleki & Mehrabani, 2024; Yan et al., 2021; Yoshizaki et al., 2026). Among the seven endurance-training studies, six were conducted in animal models and one in humans, indicating that current evidence on endurance exercise-induced structural remodeling is largely preclinical. Most endurance interventions induced eccentric LV hypertrophy. Yoshizaki et al. stated that swimming training at approximately 75% VO_2max for 8 weeks significantly increased LV cavity diameter, myocardial mass, and cardiomyocyte size, which indicates an eccentric LV hypertrophy (Yoshizaki et al., 2026). A similar study using voluntary wheel running for 5 weeks significantly increased 35% of the heart weight per tibia length ratio from 6.54 ± 0.20 to 8.81 ± 0.24 and increased 40% of the cardiomyocyte cross-sectional area from 200.6 ± 8.9 to $281.9 \pm 24.1 \mu\text{m}^2$, without signs of declining cardiac function indicating clear physiological hypertrophy (Medina et al., 2020). In contrast, some endurance studies reported predominantly concentric adaptations despite being aerobic-based protocols. Study by Venckunas et al. explored that seven weeks HIIT program increased LV posterior wall thickness from 7.5 ± 0.7 to 8.2 ± 0.4 mm (11%) and interventricular septum thickness from 7.6 ± 0.7 to 8.6 ± 0.9 mm (13%), indicating a mild concentric component without significant chamber dilation (Venckunas et al., 2025).

Low to moderate intensity endurance training predominantly induced eccentric LV hypertrophy, characterized by LV chamber dilation accompanied by proportional wall thickening, which is typical of the athlete heart phenotype. However, the pattern was not uniform across studies. High-intensity interval training showed mixed remodeling, where LV wall thickening occurred with a little to none substantial chamber enlargement (Venckunas et al., 2025). In Yan et al. (2021) study, higher-intensity endurance protocols increased collagen deposition or myocardial fibrosis. Additionally, comparisons between endurance modalities indicated that swimming training produced greater increases in myocardial mass and LV cavity size than running, suggesting modality-specific differences in structural adaptation (Yoshizaki et al., 2026). The time course of structural adaptation also differed by intensity. High-intensity endurance and HIIT protocols induced detectable increases in LV wall thickness and mass within 4–8 weeks, whereas moderate continuous endurance training generally required 12–16 weeks to achieve comparable changes (Yan et al., 2021).

Overall, endurance exercise was predominantly associated with eccentric structural remodeling in animal models, whereas the available human evidence demonstrated a predominantly concentric pattern following HIIT. However, the distribution of evidence was highly uneven, with six animal studies and only one human study, and therefore the observed differences between human and animal findings should be considered in the context of differences in study design, exercise protocols, and outcome assessment.

Resistance Training Induced Structural Cardiac Remodeling

Resistance training was evaluated in two human longitudinal studies and was associated with measurable structural cardiac adaptations. Both studies involved previously untrained or sedentary adults and used relatively high training loads ($\geq 70\%$ of one-repetition maximum).

However, the structural responses were not limited to concentric adaptation and included changes in both ventricular mass and chamber dimensions. The 20-week high-intensity resistance training program in previously untrained men produced 11% increase in LV mass along with mild increases in both LV and right ventricular end-diastolic volumes, indicating early structural remodeling (Pamart et al., 2025). Similarly, a longitudinal study demonstrates LV and left atrial enlargement occurring during a 16-week strength training intervention in previously untrained adults, while ventricular systolic and diastolic functions remain preserved (Grandperrin et al., 2024).

Across the two studies, resistance training was therefore associated with increases in myocardial mass and alterations in ventricular geometry, with findings encompassing concentric and mixed remodeling features. Training duration also contributes to the remodeling adaptation, with measurable cardiac changes emerging after 16–20 weeks of structured resistance training in previously untrained individuals (Pamart et al., 2025). Training intensity are also key determinants in which programs utilizing high loads ($\geq 70\%$ of one-repetition maximum) and multi-joint exercises performed multiple times per week tend to produce more pronounced structural adaptation (Grandperrin et al., 2024). At the molecular level, physiological hypertrophy induced by resistance exercise is generally mediated by anabolic growth signalling pathways, including IGF-1/PI3K-Akt-mTOR signalling, which promotes cardiomyocyte protein synthesis and myocardial growth while preserving cardiac function.

Risk of Bias

Each study was assessed for quality using ROBINS-I tool (Sterne et al., 2016) for human studies (n=3) and SYRCLE tool (Hooijmans et al., 2014) for animal studies (n=6). From Figure 2, all three human studies were judged to have a moderate risk of bias due to confounding (D1), primarily because their longitudinal intervention designs lacked a concurrent non-training control group, limiting the ability to distinguish intervention-related changes from temporal effects and other unmeasured factors. For missing data (D5), Venckunas et al. (2025) was judged to have a low risk of bias, as participants completed at least 95% of the prescribed training sessions and no substantial loss to follow-up was reported. In contrast, Pamart et al. (2025) was judged to have a moderate risk of bias because 5 of 27 participants did not contribute to the final 20-week analysis, including four participants lost to follow-up and one excluded for medical reasons, although the reasons for non-completion were transparently reported. Grandperrin et al. (2024) was also judged to have a moderate risk of bias due to missing data, as 10 of 34 recruited participants did not complete the intervention, leaving 24 participants for the final analysis. The six included animal studies were generally judged to have a low overall risk of bias. Nevertheless, several individual domains were rated as having an unclear risk of bias, primarily because methodological procedures were not sufficiently reported. These uncertainties involved baseline characteristics, allocation concealment, random outcome assessment, incomplete outcome data, and other potential sources of bias across individual studies. Despite these reporting limitations, no study was judged to have a high overall risk of bias, and all six studies were considered to have a low overall risk of bias, as shown in Figure 3.

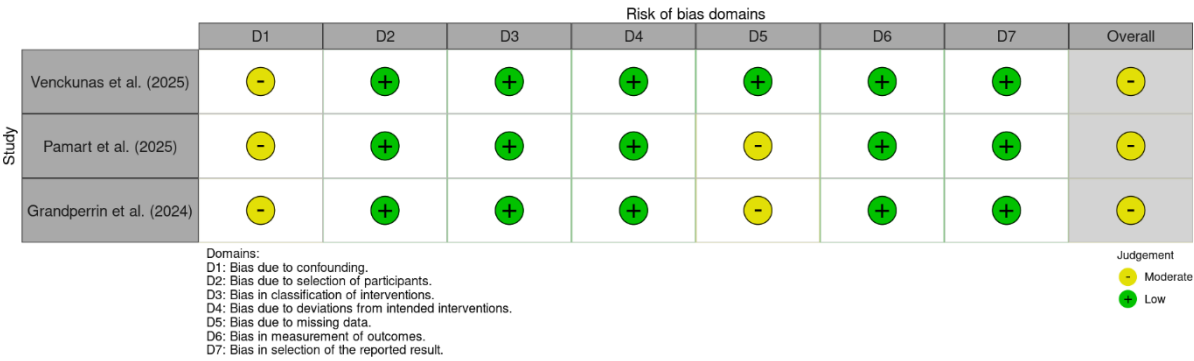


Figure 2. Traffic Light Plot Risk of Bias for Human Studies (McGuinness & Higgins, 2020)

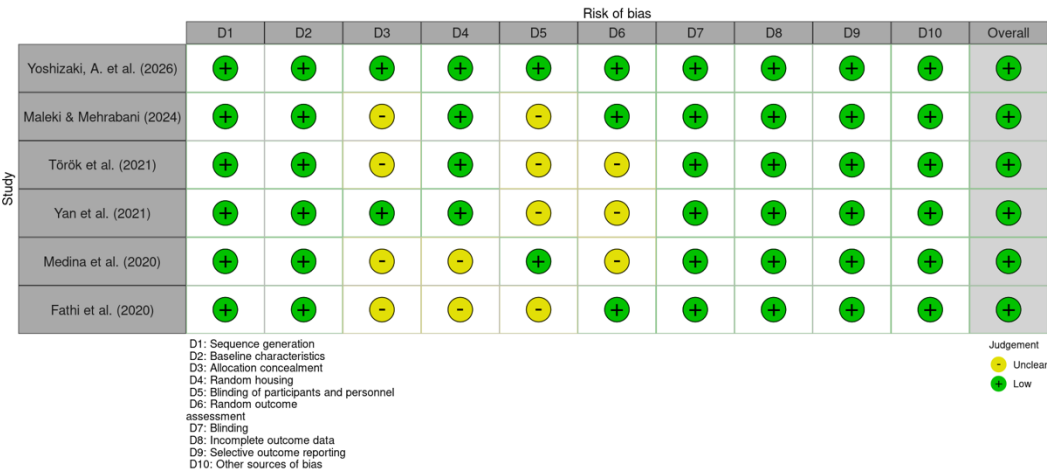


Figure 3. Traffic Light Plot Risk of Bias for Animal Studies (McGuinness & Higgins, 2020)

DISCUSSION

This systematic review synthesized evidence from 9 studies, including human trials (n = 3) and controlled experimental animal studies (n = 6), to delineate the structural differences in physiological cardiac hypertrophy induced by endurance and resistance exercise. The findings demonstrate that exercise induced cardiac remodeling represents a graded and modality dependent adaptive spectrum, rather than a rigid dichotomy between eccentric and concentric hypertrophy (Weiner & Baggish, 2012).

Endurance exercise was evaluated in seven studies, six of which used animal models and one of which involved human participants. The animal studies generally demonstrated increases in cardiac mass and ventricular chamber dimensions, consistent with predominantly eccentric remodeling. For instance, Fathi et al. reported a 25% increase in LV end-diastolic diameter without significant wall thickening, accompanied by 45% substantial improvements in stroke volume (Fathi et al., 2020). Similarly, Yoshizaki and Yan demonstrated early increases in LV volume and chamber dilation, reflecting a volume-overload stimulus (Yoshizaki et al., 2026). The available human evidence demonstrated a somewhat different structural pattern. Venckunas et al. (2025) reported increases in interventricular septal and posterior wall thickness following seven weeks of progressive HIIT, without substantial chamber enlargement. Thus, whereas eccentric remodeling predominated among the animal

endurance studies, the single human endurance study demonstrated predominantly concentric structural features. Differences in cardiac physiology between rodents and humans are also relevant when translating these findings. Rodents have substantially higher resting heart rates than humans. The resulting shorter cardiac cycle, particularly the shorter diastolic filling period, means that ventricular filling and the relationship between heart rate, preload, and stroke volume differ substantially between species (Kass, 2024; Zhao et al., 2026). Consequently, swimming and treadmill-running protocols in rodents should not be considered direct physiological equivalents of human endurance exercise based solely on the exercise duration or intensity. Therefore, structural adaptations observed in animal models provide mechanistic and experimental evidence but may not quantitatively reproduce the magnitude or phenotype of cardiac remodeling occurring in humans.

The potential mechanisms underlying these structural differences are supported primarily by external literature rather than directly established by the included studies. Endurance exercise induces cardiac remodeling driven by chronic volume overload and diastolic wall stress, whereby increased venous return elevates LV end-diastolic volume and induces stretch signaling that promotes serial sarcomere addition and cardiomyocyte elongation rather than thickening (Cheng et al., 2025b). The heterogeneity observed in endurance training cardiac remodeling across the included studies might be associated with variability in the effective hemodynamic load imposed during training (Pittaras et al., 2023). Low to moderate intensity endurance training predominantly increases venous return and diastolic wall stress further pushes IGF-1/PI3K/Akt growth pathway toward longitudinal cardiomyocyte growth and chamber dilation. In contrast, high intensity or interval-based endurance protocols introduce transient elevations in blood pressure and vascular resistance, making a pressure overload component onto the volume stimulus, resulting in increased wall thickness and a more concentric or mixed phenotype (Conti et al., 2021; Hsieh et al., 2025a; Maxwell & Oxborough, 2025).

Resistance training was evaluated in only two studies, both conducted in human participants. Pamart et al. reported mixed hypertrophy, with simultaneous increases in LV mass, wall thickness, and chamber volume (Pamart et al., 2025). In Grandperrin et al. (2024), LV mass increased by 20–25% alongside significant increases in RTW, while Pamart et al. (2025) demonstrated balanced biventricular dilation (8–10%). Pamart et al. (2025) study showed evidence that resistance training–induced LV mass increases are proportional to lean body mass and lack functional changes, suggest that resistance exercise elicits a heterogeneous, mixed remodeling phenotype rather than a purely concentric adaptation. However, because only two human studies met the inclusion criteria, the structural response to resistance training remains insufficiently characterized. The findings can be considered within the framework of the Morganroth hypothesis, which proposes predominantly eccentric hypertrophy in response to endurance exercise and concentric hypertrophy following resistance exercise (Morganroth et al., 1975). Eccentric remodeling predominated among the animal endurance studies, whereas the available human studies demonstrated concentric and mixed structural features following both endurance and resistance exercise. These findings suggest that the relationship between exercise modality and cardiac remodeling may be more complex than a strict dichotomy.

The magnitude of structural adaptation differs between modalities. The findings suggest that higher training intensities, which are associated with a potential risk of maladaptive responses, may also induce maladaptive features, including fibrosis, as observed in high-intensity endurance training, indicating that intensity can override modality-specific patterns (Maleki & Mehrabani, 2024; Yan et al., 2021). However, the included studies were not

designed to systematically compare different exercise intensities within the same population. Therefore, the independent contribution of intensity cannot be established from the present evidence. Animal studies by Yan et al. (2021) and Maleki et al. (2024) reported an increased collagen deposition or myocardial fibrosis following intensive endurance exercise. These findings raise the possibility that the relationship between exercise exposure and cardiac adaptation may involve a dose-dependent continuum. The presence of collagen deposition or fibrosis in the included animal studies should not automatically be equated with established pathological cardiac remodeling. The biological significance of collagen accumulation depends on its extent, distribution, persistence, and relationship with cardiac function. Whether these findings represent transient adaptation, early maladaptation, or a transition toward pathological remodeling remains uncertain and requires further studies. Comparatively, these results resonate with recent meta-analyses with high-intensity exercise may not uniformly confer beneficial adaptations, likely driven by repetitive microinjury and sustained wall stress leading to extracellular matrix remodeling (Zhang et al., 2020). This may promote chronic hemodynamic stress, autonomic imbalance, and inflammatory signalling, which are known pathways linked to fibrotic remodeling and maladaptive hypertrophy when recovery is insufficient (De Oude et al., 2025).

The distinction between physiological and pathological remodeling is clinically relevant because exercise-induced structural adaptations may overlap phenotypically with cardiac disease. Increased LV mass, wall thickness, or chamber dimensions may occur as part of physiological exercise-induced cardiac remodeling but can also be observed in cardiomyopathies (Maxwell & Oxborough, 2025). The heterogeneous structural patterns identified in this review further support the concept that physiological cardiac remodeling exists along a continuum rather than within strictly discrete eccentric and concentric. At the same time, the findings of collagen deposition and myocardial fibrosis in some animal studies indicate that the boundary between adaptive and potentially maladaptive remodeling may depend on the magnitude and duration of the exercise stimulus. These findings need further investigation rather than supporting a definitive threshold for pathological remodeling.

This review has several limitations. First, the evidence base was small, comprising nine included studies, which limits the precision and generalizability of the conclusions. Second, the evidence was unevenly distributed across exercise modalities, with seven studies evaluating endurance exercise and only two evaluating resistance training. The evidence was also predominantly derived from animal models, with six animal studies compared with three human studies. Third, substantial heterogeneity was present in exercise modality, intensity, frequency, duration, progression, and participant or animal characteristics. Cardiac assessment methods also varied, including echocardiographic measurements, histological assessments, and molecular analyses, which limited direct comparison across studies and constrained quantitative synthesis. Fourth, the human evidence included longitudinal studies without concurrent control groups, limiting causal inference. Finally, the restriction to English-language publications may have introduced language bias. Future research should prioritize well-controlled longitudinal studies in humans that directly compare endurance and resistance exercise while systematically varying exercise intensity, duration, and training volume. Standardized assessment of LV mass, chamber dimensions, wall thickness, and relative wall thickness would facilitate more consistent classification of eccentric, concentric, and mixed remodeling. Further investigation of intensive exercise is particularly warranted to determine whether a reproducible dose-response relationship exists between exercise exposure and potentially adverse structural changes such as myocardial fibrosis.

CONCLUSION

In summary, this review indicates that endurance and resistance exercises induce overlapping but heterogeneous patterns of physiological cardiac hypertrophy, which may vary according to exercise modality and training intensity. Endurance exercise predominantly promotes eccentric hypertrophy, while resistance exercise demonstrates predominantly concentric or mixed hypertrophy associated with pressure overload. However, these patterns were not uniform across studies. Given the small and heterogeneous evidence base, including the predominance of animal studies and the limited number of human studies, these findings should be considered preliminary. Well-controlled longitudinal studies with standardized assessment of cardiac structure and exercise dose are needed to clarify the long-term structural effects of different exercise modalities and to determine whether specific exercise intensity or duration thresholds influence the transition between adaptive and potentially maladaptive remodeling.

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