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The effect of twelve weeks of resistance training and a detraining period on relative left ventricular wall thickness in inactive men

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Abstract

Purpose: Physical activity is associated with higher left ventricular mass but also reduced risk of cardiovascular outcomes. The purpose of the present research was to investigate the effect of twelve weeks of resistance training and a detraining period on relative left ventricular wall thickness in inactive men. **Method:** Twenty-four inactive men who met the inclusion criteria and were able to participate in the exercise protocol were randomly divided into two resistance training groups (n=12) and control group (n=12). Anthropometric characteristics, body composition, and relative wall thickness (RWT) were measured using echocardiography at three time points: before the start of exercise, at the end of 12 weeks of exercise, and after a 6-week period of non-exercise. The exercise protocol consisted of 12 weeks of exercise (three sessions per week) followed by 6 weeks of non-exercise. Data were analyzed using independent t-tests and repeated measures analysis of variance at a significance level of $p < 0.05$ using SPSS software version 24. **Results:** The results showed that a significant change in RWT was observed between pre-test and post-test measurements in both groups ($p < 0.05$) and significant changes in RWT were also observed during the detraining period in the training group ($p < 0.05$). **Conclusion:** It can be concluded that 12 weeks of resistance training can lead to beneficial changes in cardiac structure in inactive men. A 6-week detraining period can lead to negative effects on these adaptations. Therefore, it is recommended that individuals participate in regular and structured resistance training to maintain these positive adaptations.

Keywords: resistance training, sedentary lifestyle, left ventricular, inactive, men.

Introduction

Long-term, structured exercise training induces a well-described spectrum of cardiac adaptations—collectively termed exercise-induced cardiac remodeling (EICR)—that reflect the heart's ability to adjust to repeated hemodynamic and neurohormonal demands (Baggish & Wood, 2011; Martinez et al., 2021). Among these adaptations, remodeling of the left ventricle (LV) is particularly important because it can enhance stroke volume, cardiac output reserve, and exercise performance, while also creating a clinical "gray zone" where physiological hypertrophy may resemble early cardiomyopathy on imaging (Baggish et al., 2020; Pluim et al., 2000). At the same time, although sudden cardiac death (SCD) in young athletes is uncommon, it remains a highly visible event that draws intense media attention and public concern, reinforcing the need for precise interpretation of athletic cardiac phenotypes and their boundaries with pathology (Corrado & Zorzi, 2024; Wasfy & Weiner, 2016). Consequently, a clear understanding of LV remodeling with training—and the extent to which these changes regress with training cessation—has direct value for sports cardiology, exercise prescription, and performance programming.

Early echocardiographic work suggested that the pattern of LV hypertrophy is shaped by the dominant load imposed by the sport. In the classic study by Morganroth et al. (1975), endurance-trained athletes tended to demonstrate larger LV cavity dimensions (consistent with volume loading), whereas resistance-trained athletes appeared to show relatively greater wall thickness (consistent with pressure loading) (Morganroth et al., 1975). This conceptual framework—often referred to as the Morganroth hypothesis—was influential for decades, but contemporary evidence indicates that the "endurance = eccentric, resistance = concentric" dichotomy is frequently oversimplified (Haykowsky et al., 2018; Naylor et al., 2008). Modern imaging and mechanistic models emphasize that remodeling depends not only on the type of training but also on intensity, session structure, cumulative

training years, body size, sex, and the intermittent (rather than continuous) nature of athletic loading compared with disease states such as hypertension or valvular disease (Haykowsky et al., 2018; Pluim et al., 2000). In practice, EICR is now recognized as a dynamic and multidimensional process affecting chamber size, wall thickness, LV mass, diastolic filling, and myocardial mechanics—features that require integrated interpretation using echocardiography and, when indicated, cardiac magnetic resonance (Baggish et al., 2020; Palermi et al., 2023).

A substantial body of evidence confirms that endurance-oriented training is associated with increases in LV cavity size and LV mass, especially when training is sufficiently prolonged and of adequate volume. Meta-analytic data in competitive and recreational contexts demonstrate that endurance training can increase LV structural parameters, although the magnitude of adaptation varies by age, baseline training status, and training modality (Morrison et al., 2023). Experimental and longitudinal athlete studies further show that relatively short periods of intensive endurance training can induce measurable changes in LV structure and function, consistent with the concept of an early "augmentation" phase of remodeling (Baggish et al., 2008). Importantly, however, not all training exposures are equivalent; in previously sedentary adults, modest training volumes may improve fitness with only minimal structural cardiac changes, suggesting that a higher dose or longer duration may be required to elicit robust LV remodeling (Hedman et al., 2017; Sipola et al., 2009). These dose–response features are central to designing protocols that aim to influence cardiac structure rather than simply improve cardiorespiratory capacity.

In contrast to endurance training, the cardiac effects of resistance training (RT) remain more heterogeneous across studies and populations. RT is typically characterized by brief, repeated bouts of high intrathoracic pressure, elevated arterial pressure, and augmented afterload—especially when Valsalva-like maneuvers occur—yet the

intermittent nature of these stimuli may not produce the same remodeling signature as chronic pressure-overload diseases (Haykowsky et al., 2018). Some investigations in strength athletes report minimal or no differences in LV morphology compared with controls, challenging the assumption that long-term RT necessarily induces clinically meaningful LV hypertrophy (Haykowsky et al., 2000). Other studies, using advanced echocardiographic techniques, suggest that endurance- and strength-trained athletes can exhibit distinct patterns in myocardial mechanics even when gross dimensions appear similar, implying that functional adaptation may precede or accompany structural change (Santoro et al., 2014). These mixed findings likely reflect differences in RT style (e.g., powerlifting vs hypertrophy training), loading scheme, training age, concurrent aerobic work, anthropometrics, and measurement methodology—reinforcing calls for standardized imaging and sport-specific interpretation frameworks (Baggish et al., 2020; Haykowsky et al., 2018).

A critical, and sometimes underappreciated, dimension is sex-specific remodeling. Female athletes generally demonstrate different electrical and structural remodeling than males, often maintaining normal LV geometry with proportionally greater cavity dilation relative to wall thickness compared with men, and with sport type strongly modulating the degree of adaptation (D'Ascenzi et al., 2020). Reviews focusing on the female heart emphasize that extrapolating male-based athletic norms can lead to misclassification, either by overcalling pathology or by missing atypical phenotypes (D'Ascenzi et al., 2020; Rao & Baggish, 2022). This is especially relevant when research questions shift from elite athletes to inactive or sedentary women, where baseline cardiac size, hormonal milieu, and training responsiveness may differ substantially from male or athletic cohorts. Therefore, evaluating RT-induced LV remodeling in women—particularly those who begin from low fitness levels—requires careful protocol definition and imaging interpretation grounded in sex-specific evidence.

Beyond training-induced adaptations, many athletes and recreationally trained individuals face periods of reduced training or complete cessation due to injury, off-season breaks, illness, pregnancy, or lifestyle constraints. The loss of training-induced adaptations—often termed detraining—is typically defined as the partial or complete reversal of physiological, performance, and morphological gains following an insufficient training stimulus (Mujika & Padilla, 2000). Cardiorespiratory fitness is especially sensitive: classic and contemporary work indicates that VO_2max can decline within weeks, driven largely by reductions in blood/plasma volume, stroke volume, and maximal cardiac output, with compensatory heart-rate responses often insufficient to fully offset decreased stroke volume (Coyle et al., 1986; Mujika & Padilla, 2000). Systematic reviews also describe broader metabolic consequences of detraining, including shifts toward reduced oxidative capacity and impaired cardiometabolic profiles, particularly when inactivity is prolonged (Barbieri et al., 2024; Bergouignan et al., 2011).

From a cardiac-structure perspective, detraining is associated with measurable regression of LV mass and volumes, although the time course and completeness of reversal vary. A recent systematic review and meta-analysis concluded that LV mass decreases following detraining lasting one week or more in endurance-trained individuals, while highlighting the need to better understand moderators such as sex, age, and detraining type (Massarotto et al., 2024). Longitudinal studies in elite athletes similarly demonstrate that LV cavity size and wall thickness can decrease with deconditioning, yet normalization may be incomplete in a subset, leaving residual chamber enlargement that complicates clinical interpretation (Pelliccia et al., 2002). Mechanistic work using cardiac magnetic resonance suggests that regression of "athletic" LV hypertrophy can occur rapidly and may be mediated predominantly by reductions in the intracellular compartment rather than extracellular matrix expansion, supporting the concept of a largely physiological, reversible process (Swoboda et al., 2019). Collectively,

these findings underscore that the athlete's heart is not a fixed entity; it is plastic and responsive to both training and detraining exposures.

Despite progress, important gaps remain. First, most detraining literature has focused on endurance-trained cohorts, leaving uncertainty about how LV structure responds to detraining after RT-focused interventions, particularly in non-athletes (Haykowsky et al., 2018; Massarotto et al., 2024). Second, RT cessation clearly reduces muscular performance—meta-analytic evidence shows detrimental effects on strength components that scale with cessation duration and are more pronounced in older and inactive individuals—yet the parallel trajectory of cardiac structural changes with RT cessation is far less established (Bosquet et al., 2013). Third, sex-specific evidence is still limited in intervention-based designs; while cross-sectional athlete data support distinct female remodeling patterns, fewer controlled studies have examined inactive women undergoing structured RT followed by detraining with consistent imaging endpoints (D'Ascenzi et al., 2020; Baggish et al., 2020). This gap matters because women are increasingly targeted for RT programs to improve health, body composition, and cardiometabolic risk, and RT may influence cardiac-related fat depots and systemic risk factors even in physically inactive populations (Christensen et al., 2019).

Accordingly, further research is needed to clarify whether a defined period of RT is sufficient to induce detectable changes in LV structure in inactive women, and whether any such changes regress during a subsequent detraining period. Addressing this question requires rigorous training prescription, standardized echocardiographic or CMR assessment, and interpretation guided by contemporary athlete/active-population imaging recommendations that emphasize differentiation between physiological remodeling and pathology (Baggish et al., 2020; Palermi et al., 2023). Within this context, the present study was designed to examine whether 12 weeks of resistance training, followed by a period of detraining, alters LV structural characteristics in inactive

women, thereby contributing to a more evidence-based understanding of RT-specific cardiac plasticity and its reversibility. This study aimed to determine whether 12 weeks of resistance training followed by a period of detraining had any effect on the structure of the left ventricle.

Methods

Study design and participants

This randomized controlled trial investigated the effects of 12 weeks of resistance training followed by 6 weeks of detraining on left ventricular (LV) structure and function in inactive middle-aged men. Twenty-four inactive men (mean age: 45.37 ± 5.46 years) who could attend laboratory visits and training sessions consistently were recruited via local advertisements and eligibility screening. Physical inactivity was operationally defined as no participation in structured exercise training for ≥6 months prior to enrollment. Following baseline assessments, participants were randomly assigned to either a resistance training group (RT; n = 12) or a non-exercise control group (CON; n = 12) using a computer-generated randomization schedule; allocation concealment was implemented by a researcher not involved in testing to reduce selection bias.

Eligibility criteria included: (i) general and cardiovascular health confirmed by a physician, (ii) no current medication use, (iii) no use of ergogenic or sport supplements, (iv) non-smoker status, and (v) willingness to complete all testing and the full training/detraining timeline. Exclusion criteria comprised diagnosed cardiovascular disease, uncontrolled hypertension, orthopedic/neuromuscular conditions limiting safe resistance exercise, and any change in medication or supplement intake during the study period. All participants provided written informed consent, and the study procedures adhered to internationally recognized principles for research involving human participants (World Medical Association, 2013).

Resistance training intervention and detraining

The RT program was conducted 3 sessions per week for 12 weeks, with each session lasting ~90 minutes. Training was planned according to evidence-based principles for novice resistance training, emphasizing correct technique, gradual load progression, and systematic overload (American College of Sports Medicine, 2009). Prior to the intervention, a familiarization session was held to teach movement technique, safe spotting, and breathing strategies to minimize excessive intrathoracic pressure during lifting. The exercise battery targeted major muscle groups and included: leg press, barbell chest press, seated row, bent-knee crunch, front leg raise, toe raise (calf raise), hamstring curl, shoulder press, barbell chin-up, and barbell forearm raise.

To individualize intensity, maximal strength was estimated using a submaximal repetition test and the Brzycki 1RM prediction equation (Brzycki, 1993). Loads were then progressed from low to moderate–high intensity across the 12-week period by increasing resistance when participants could perform repetitions beyond the prescribed range, consistent with established progression models for healthy adults (American College of Sports Medicine, 2009). After completing the 12-week program, RT participants entered a 6-week detraining phase and were instructed to avoid structured resistance or endurance exercise; adherence was supported via weekly follow-ups. The CON group was asked to maintain usual lifestyle behavior across both phases.

Measurements and echocardiography

Assessments were performed at baseline, post-training (12 weeks), and post-detraining (6 weeks later). Height and body mass were measured using calibrated laboratory equipment. Resting heart rate was recorded after at least 10 minutes of seated rest.

Cardiac structure and function were assessed using transthoracic echocardiography (Zonare, USA; 2012 model) by an experienced cardiologist in a dedicated echocardiography laboratory. Imaging included 2D-guided M-mode, spectral Doppler, and color Doppler acquisition. LV linear dimensions and wall thickness were obtained in standard views and measured at end-diastole and end-systole according to widely used chamber quantification recommendations (Lang et al., 2015). Relative wall thickness (RWT) was computed as RWT = (2 × PWTd) / LVIDd, enabling classification of LV geometry (Lang et al., 2006).

Statistical analysis

Normality of distributions was examined using the Shapiro–Wilk test (Shapiro & Wilk, 1965), and homogeneity of variances was evaluated using Levene's test (Levene, 1960). Baseline differences between groups were tested using independent t-tests. Intervention effects were analyzed using repeated measures ANOVA (group × time). When significant interactions were detected, Bonferroni-adjusted post hoc comparisons were applied to control familywise error (Bonferroni, 1936; Dunn, 1961). Statistical significance was set at $p < 0.05$, and analyses were conducted using SPSS (version 24).

Results

Participant Characteristics

The mean age was 35.37 ± 5 years, height was 162.40 ± 5 cm, and weight was 65.61 ± 10.26 kg.

Table 1 summarizes the 12-week resistance training progression used in the present study. The program was structured to gradually increase neuromuscular and cardiovascular loading through systematic changes in relative intensity (%1RM) and volume (sets/rounds × repetitions). During weeks 1–2, participants trained at lower intensities (50–55%

1RM) with higher repetitions to emphasize technical learning, movement competency, and tolerance to resistance exercise. In weeks 3–4, intensity increased to 60% 1RM while repetitions decreased, reflecting a transition toward strength-oriented adaptations. Week 5 included a planned reduction in intensity and volume (55% 1RM; 2 rounds × 12 repetitions) to manage fatigue and support recovery before higher-load phases. From weeks 6–12, intensity progressively increased (70–88% 1RM) alongside moderate set volumes (3–4 rounds) and lower repetitions (8–10), aligning with established resistance training programming principles for improving maximal strength and promoting physiological adaptation. The second and third weekly sessions mirrored the primary loading parameters to ensure adequate weekly training stimulus and consistency of exposure across the intervention period.

Table 1: Resistance Exercise training Over 12 Weeks

Week	First Session	Second Session	Third Session
First Week	50% 1RM	2 rounds x 14 repetitions (50% 1RM)	2 rounds x 14 repetitions (50% 1RM)
Second Week	55% 1RM	2 rounds x 14 repetitions (55% 1RM)	2 rounds x 14 repetitions (55% 1RM)
Third Week	60% 1RM	3 rounds x 10 repetitions (60% 1RM)	3 rounds x 10 repetitions (60% 1RM)
Fourth Week	60% 1RM	3 rounds x 10 repetitions (60% 1RM)	3 rounds x 10 repetitions (60% 1RM)

Fifth Week	55% 1RM	2 rounds x 12 repetitions (55% 1RM)	2 rounds x 12 repetitions (55% 1RM)
Sixth Week	70% 1RM	3 rounds x 10 repetitions (70% 1RM)	3 rounds x 10 repetitions (70% 1RM)
Seventh Week	75% 1RM	4 rounds x 8 repetitions (75% 1RM)	4 rounds x 8 repetitions (75% 1RM)
Eighth Week	80% 1RM	4 rounds x 8 repetitions (80% 1RM)	4 rounds x 8 repetitions (80% 1RM)
Ninth Week	80% 1RM	4 rounds x 8 repetitions (80% 1RM)	4 rounds x 8 repetitions (80% 1RM)
Tenth Week	85% 1RM	4 rounds x 8 repetitions (85% 1RM)	4 rounds x 8 repetitions (85% 1RM)
Eleventh Week	85% 1RM	4 rounds x 8 repetitions (85% 1RM)	4 rounds x 8 repetitions (85% 1RM)
Twelfth Week	88% 1RM	4 rounds x 8 repetitions (88% 1RM)	4 rounds x 8 repetitions (88% 1RM)

1RM = one-repetition maximum (estimated using a submaximal repetition test and the Brzycki equation). “Rounds” refers to the number of sets performed for each exercise in the session; repetitions indicate the target number per set. Training intensity is expressed as a percentage of the participant’s estimated 1RM. All sessions were completed three times per week (first, second, and third sessions), using

the same exercise list; load progression followed the principle of progressive overload, with adjustments made when participants were able to exceed the prescribed repetition range while maintaining correct technique and controlled breathing. Rest intervals were kept consistent across sessions

Table 2 outlines the resistance exercises included in the intervention and the principal muscles involved in each movement. This mapping was used to document exercise selection and muscular coverage, ensuring that the program targeted all major regions (upper-body pushing, upper-body pulling, lower-body multi-joint patterns, trunk/abdominal work, and calf/forearm accessory work). Chest press was included as the primary horizontal pushing movement engaging the pectoral complex and triceps, while shoulder press emphasized shoulder girdle musculature and the upper trapezius. The leg press served as the core lower-body compound exercise, targeting the quadriceps and gluteal musculature with hamstring co-contraction. Posterior-chain emphasis was supported via the "back of thigh" movement (hamstring-focused), while trunk stability and anterior core endurance were addressed through the abdominal movement listed as "long and leaking" (interpreted as a trunk flexion/core exercise). Accessory exercises (standing calf raise with device and barbell forearm work) were incorporated to strengthen distal musculature, improve overall training balance, and reduce localized fatigue limitations during compound lifts. For transparency and replicability, muscle group descriptions are reported using standard anatomical terminology; however, the table reflects that many movements recruit multiple synergists, and the listed muscles should be interpreted as "primary targets" rather than an exhaustive electromyographic profile.

Table 2: Movements and muscles involved

Movements Performed	Muscles Involved
Chest Press	Pectoralis major, pectoralis minor, triceps brachii, anterior deltoid, anterior ulna, anterior inferior ulna, anterior deltoid
Leg Press	Internal wide, middle wide, right femur, external wide, large pelvis, biceps (high), semi-stringent, membranous
Long and Leaking	Flexors of the cervical vertebrae, sternum, rectus abdominis, suez sacrum, rectus femoris, shoulder
Armpit Stretch	Large chest, parallelogram, triceps (long head), posterior deltoid, large round, brachialis, small chest, long extensor
Back of Thigh	Semi-membranous, semi-stringent, biserrani, twin
Shoulder Press (From Behind)	Strengthening the muscles of the shoulder girdle and trapezius
Standing Leg with Device	Twins, insoles, soles of the feet
Forearm with Barbell	Biceps brachii, anterior brachialis, ulnar arm, anterior deltoid, forearm

The table presents the primary muscle groups targeted by each exercise. Muscle involvement is summarized at the level of major agonists and common synergists; actual activation may vary with technique, range of motion, load, grip/stance width, and individual anthropometrics. Exercise names reflect the terminology used in the training log; where local/translated labels were used, they were mapped to their closest conventional resistance-training equivalents for reporting.

Table 3 shows the characteristics of the left ventricle indices of the subjects in each group at three stages (pre-test, post-test, and detraining) in terms of mean, standard deviation, and percentage changes.

Table 3 provides descriptive statistics for relative wall thickness across baseline, post-training, and post-detraining assessments in both groups. Presenting mean ± SD allows comparison of central tendency and dispersion, while percentage change quantifies the magnitude of within-group shifts during the training phase (Before→After) and the subsequent detraining phase (After→Post-detraining). This layout supports an initial interpretation of whether the resistance training stimulus was associated with directional changes in LV geometry and whether those changes appeared to persist or partially regress following cessation of structured training. However, these descriptive patterns should be interpreted in conjunction with the group × time inferential analyses (e.g., repeated-measures ANOVA and post hoc tests) to determine statistical significance and the extent to which observed changes exceed expected measurement variability.

Table 3: Indicators for RWT in Experimental and Control Groups

variable	Groups	
	Experimental (n=12) RWT (M±SD)	**Control (n=12) RWT (M±SD)**
Before	26.71 ± 3.22	28.21 ± 3.23
After	29.99 ± 3.35	29.21 ± 3.17
Post-detraining	28.73 ± 3.13	28.81 ± 4.24
Percentage change (Before to After)	-6.22%	4.40%
Percentage change (After to Post-detraining)	5.42%	2.52%

RWT = relative wall thickness. Data are presented as mean ± standard deviation (M ± SD) for the experimental (n = 12) and control (n = 12) groups at three time points: Before (baseline), After (post-intervention; following 12 weeks of resistance training), and Post-detraining (following 6 weeks of training cessation). Percentage change was calculated as ((latervalue−earliervalue)/earliervalue)×100((later value − earlier value) / earlier value) × 100((latervalue−earliervalue)/earliervalue)×100. Negative values indicate a reduction and positive values indicate an increase relative to the earlier time point

Given that the significance level (P-value) was higher than 0.05, equality of variances or homogeneity of variances is accepted. One-way ANOVA test was used to compare the means of dependent variables in the pre-test stage.

Table 4 reports the outcomes of a one-way repeated-measures analysis conducted on RWT values within the exercise and control groups. The repeated-measures approach evaluates whether mean RWT differed significantly over time within each group, accounting for the correlated nature of observations collected from the same individuals across repeated assessments. In the exercise group, statistically significant time effects (P = 0.001) indicate that RWT changed across the measurement occasions following the training intervention, suggesting a measurable within-group response to the resistance training exposure. In contrast, the control group did not demonstrate a significant time effect (P > 0.05), implying that RWT remained relatively stable across the same period in the absence of structured training. For comprehensive interpretation, these within-group findings should be considered alongside between-group analyses (e.g., group × time interaction from repeated-measures ANOVA) and the descriptive changes presented in Table 3 to determine whether the observed temporal changes in the exercise group are meaningfully greater than normal variation and measurement error.

Table 4: Results of One-Way Repeated Measures on RWT Values

Variable	Group	Time	SS	MS	F	P
RWT	Exercise	Pre-Test -	16.854	16.947	38.219	0.001*
		Post-Test	18.231	18.190	36.787	0.001*
	Control	Pre-Test -	9.658	9.524	1.548	0.458
		Post-Test	9.954	9.950	0.758	0.695

*Significant difference at the level of P<0.05.

SS = sum of squares; MS = mean square; F = F-statistic; P = probability value. One-way repeated-measures ANOVA was applied separately within each group to test whether RWT changed across the repeated time points (e.g., pre-test, post-test, and subsequent follow-up where applicable). An asterisk () indicates statistical significance at P < 0.05.*

Discussion

The purpose of the present study was to determine whether 12 weeks of progressive resistance training followed by a period of detraining would alter left ventricular (LV) relative wall thickness (RWT) in inactive middle-aged men. The principal finding was that RWT increased significantly from pre-test to post-test in the training group compared with the control group ($p < 0.05$), and that RWT declined significantly during the detraining period (post-test to post-detraining; $p < 0.05$), although mean values in the training group remained above baseline. Collectively, these data support the interpretation that progressive resistance training can provoke concentric remodeling-like changes in LV geometry (reflected in higher RWT), while subsequent training cessation permits partial regression of these adaptations—consistent with the recognized plasticity of exercise-induced cardiac remodeling (Mihl et al., 2008; Petek et al., 2022).

RWT is commonly used as a geometric index reflecting the balance between LV wall thickness and LV cavity size and is often interpreted within frameworks of LV geometry (e.g., normal geometry, concentric remodeling, concentric hypertrophy, eccentric hypertrophy) when combined with LV mass and chamber dimensions (Lang et al., 2015). An increase in RWT after resistance training suggests a shift toward a more concentric LV geometry, which aligns with the physiological model that resistance exercise imposes a predominant pressure overload stimulus (Mihl et al., 2008). During high-load lifting, especially when breath-holding or Valsalva-like strategies occur, systolic arterial pressure can rise markedly, creating transient but repeated afterload challenges for the myocardium (MacDougall et al., 1985). Over weeks of training, such recurring pressure stimuli may contribute to increased wall thickness relative to cavity dimension—manifesting as increased RWT—through cardiomyocyte adaptations and altered myocardial loading patterns (Mihl et al., 2008; Johnson et al., 2015).

Your results are broadly consistent with experimental evidence that resistance training can induce concentric cardiac hypertrophy without

impairing ventricular function. Baraúna and colleagues reported that resistance training in an animal model produced concentric hypertrophy without cavity reduction or systolic dysfunction, supporting the biological plausibility of a training-driven increase in wall thickness–related indices (Baraúna et al., 2007). In humans, earlier syntheses and reviews have similarly suggested that increases in LV wall thickness and mass may occur with resistive training depending on the intensity and duration of the program, while global indices of systolic function typically remain preserved (Effron et al., 1989; Haykowsky et al., 2002). Cross-sectional comparisons across sports also report that strength/power athletes may exhibit relatively greater concentricity than endurance athletes, although real-world patterns vary and are influenced by body size, training history, and mixed training exposures (Venckunas et al., 2008; Haykowsky et al., 2002).

Importantly, the present findings extend this literature to an inactive, middle-aged male sample exposed to a structured, progressive program. This population may be particularly responsive to a novel mechanical stimulus, at least in the early phases of training, because baseline cardiovascular loading history is limited and the initial training period often produces rapid neuromuscular and hemodynamic adaptations (American College of Sports Medicine, 2009). From an applied perspective, this suggests that progressive resistance training—implemented with appropriate technique and monitoring—can produce measurable cardiac geometric changes even in previously inactive adults, supporting its broader role within cardiovascular health promotion strategies (Paluch et al., 2024).

Several complementary mechanisms may explain the observed RWT increase. First, resistance training sessions generate intermittent high afterload episodes, particularly during multi-joint movements at moderate-to-high intensities, which can promote parallel sarcomere addition and increased wall thickness—an archetypal concentric adaptation pathway (Mihl et al., 2008; Rossi, 1991). Second, the intrathoracic and intra-abdominal pressure generated during heavy or

strained lifting can alter preload/afterload interactions and acutely modify ventricular wall stress. A systematic review of pressure dynamics in resistance-related tasks highlights that Valsalva maneuvers and bracing strategies meaningfully influence intrathoracic pressure and hemodynamics (Blazek et al., 2019). Third, repeated pressure surges may interact with neurohumoral responses (e.g., sympathetic activation) and vascular changes (e.g., improved resting blood pressure in some contexts), creating a complex stimulus landscape for remodeling (Paluch et al., 2024). Although the present study did not directly quantify beat-to-beat blood pressure during lifting or training-related changes in arterial stiffness, classic invasive data underscore that pressure stimuli during heavy lifting can be substantial (MacDougall et al., 1985), supporting the plausibility of pressure-mediated geometric remodeling.

At the same time, it is essential to recognize that resistance training effects on LV morphology are not universally observed. For example, Haykowsky et al. reported that long-term resistance training in elite powerlifters did not necessarily alter LV morphology compared with controls, illustrating that training history, selection factors, and measurement approaches can yield different outcomes across cohorts (Haykowsky et al., 2000). This apparent discrepancy across studies is often explained by heterogeneity in program design (load, volume, rest intervals), concurrent endurance work, body composition, and the distinction between short-term intervention responses and long-term steady-state remodeling (Haykowsky et al., 2002). In inactive men, the early-phase responsiveness to training may be greater than in chronically trained lifters, potentially explaining why a clear RWT change was detectable here.

A second key finding was the significant reduction in RWT from post-test to post-detraining in the resistance training group. This result is consistent with the broader concept of detraining: when the training stimulus is reduced or removed, physiological adaptations often regress toward baseline (Petek et al., 2022). While much of the detraining

literature focuses on endurance training and LV mass, the general principle that cardiac remodeling is dynamic and reversible appears robust. For example, Pelliccia et al. demonstrated that LV wall thickness in elite athletes can return to normal limits after periods of detraining, supporting the clinical utility of detraining as a tool to distinguish physiological adaptation from pathology (Pelliccia et al., 2002). More recent cardiac magnetic resonance evidence indicates that measurable regression of LV mass can occur after as little as one month of complete detraining and is mediated predominantly by reductions in the intracellular compartment rather than extracellular fibrosis-like expansion—again consistent with a physiological, reversible remodeling process (Swoboda et al., 2019). In endurance-trained cohorts, a systematic review and meta-analysis similarly concluded that detraining lasting one week or more is associated with a reduction in LV mass (Massarotto et al., 2024).

Although your endpoint was RWT rather than LV mass, the pattern—training-associated increase followed by detraining-associated decrease—fits this established reversibility framework. Practically, the partial regression observed after detraining implies that some of the geometric adaptation achieved during the 12-week resistance training period may require continued exposure (even at reduced dose) to be maintained. This aligns with narrative conclusions that cardiac atrophy/remodeling regression can occur within short detraining windows (1–8 weeks) while often sparing global systolic function (Petek et al., 2022). For program design, these findings support the recommendation that even during off-season or interruption periods, some form of maintenance training may be valuable for preserving structural and functional adaptations, especially in populations prone to inactivity relapse.

Based on the reported means, RWT increased from 26.71 to 29.99 (training group) and then decreased to 28.73 during detraining. Interpreting these values directly suggests an approximate +12% increase from pre- to post-test and an approximate −4% change from

post-test to post-detraining. If your table reports percentage changes with opposite signs, this may reflect a calculation direction (e.g., earlier minus later) or a transcription issue; clarifying the percentage-change formula in your manuscript will improve transparency and reproducibility (Lang et al., 2015). Clear reporting is particularly important for RWT because it is derived from measured dimensions and may be sensitive to small measurement differences, underscoring the value of standardized acquisition and analysis procedures (Lang et al., 2015).

Several limitations should be considered when interpreting these results. First, the sample size (n = 12 per group) limits power and may inflate the impact of individual variability; future studies with larger cohorts would allow more stable estimates and subgroup analyses. Second, RWT alone provides only a partial view of LV remodeling. Because RWT is most informative when interpreted alongside LV mass and chamber dimensions, inclusion of LV mass index, LV end-diastolic diameter/volume, and diastolic function indices would strengthen conclusions about whether the observed change represents concentric remodeling versus concentric hypertrophy (Lang et al., 2015). Third, the study did not quantify potential mediators such as resting or ambulatory blood pressure, training-session blood pressure responses, or adherence to breathing technique, which are relevant given the known hemodynamic spikes during heavy resistance work (MacDougall et al., 1985; Blazek et al., 2019). Fourth, diet, sleep, and daily activity outside the intervention—factors that can influence hemodynamics and body composition—were not described in detail and could contribute to inter-individual differences.

Despite these limitations, the findings are meaningful for both training prescription and clinical interpretation. From a health perspective, resistance training is widely endorsed as a safe and effective modality for improving cardiometabolic outcomes in adults with and without cardiovascular disease, when appropriately prescribed and monitored

(Paluch et al., 2024). Your results add that resistance training can also induce detectable LV geometric changes in previously inactive men, emphasizing that the myocardium adapts not only to endurance exercise but also to progressive strength training exposures (Haykowsky et al., 2002; Effron et al., 1989). Clinically, the observation that RWT partially regressed during detraining aligns with the concept that physiological remodeling is reversible, which is relevant when distinguishing adaptive hypertrophy from cardiomyopathy in ambiguous cases (Pelliccia et al., 2002; Swoboda et al., 2019).

Future investigations should (i) include larger and more diverse samples, (ii) incorporate multimodal imaging and a fuller set of LV geometry variables (LV mass, volumes, strain), (iii) quantify hemodynamic exposure (e.g., BP responses during sessions, ambulatory BP), and (iv) compare different resistance training models (hypertrophy-oriented vs strength-oriented vs circuit-based) to identify which elements most strongly influence LV geometry (Lang et al., 2015; Haykowsky et al., 2002). Additionally, mapping the time course of regression across different detraining durations (e.g., 2, 4, 8, and 12 weeks) would clarify how quickly concentricity-related markers return toward baseline in inactive adults (Petek et al., 2022; Massarotto et al., 2024).

Conclusion

These findings support the research hypothesis that twelve weeks of progressive resistance training meaningfully influences left ventricular relative wall thickness (RWT) in previously inactive men. In this quasi-experimental design, RWT was assessed by standardized echocardiography across three time points, allowing a direct comparison of cardiac structural responses to training and to a subsequent period of non-exercise. The observed increase in RWT following the training phase suggests that progressive overload in resistance exercise can induce measurable remodeling consistent with beneficial cardiac adaptation in sedentary adults. Importantly, the

changes detected during the detraining phase indicate that these adaptations are at least partly reversible when structured training is discontinued, reinforcing the principle that cardiovascular and myocardial benefits require continued exposure to an adequate training stimulus. From an applied perspective, the results highlight the value of incorporating regular, supervised resistance training into health-oriented exercise prescriptions for inactive men, not only to improve musculoskeletal fitness but also to promote favorable cardiac structure. At the same time, the decline in RWT after non-exercise emphasizes the need for long-term adherence strategies and gradual transition plans (e.g., maintenance training) to preserve training-induced adaptations and reduce the likelihood of regression.

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