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Association between muscle strength gains and biventricular cardiac remodeling in response to high-intensity resistance training in healthy untrained males: a longitudinal study

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Abstract

Background High-intensity resistance training induces structural and functional adaptations in skeletal muscle, yet its impact on cardiac remodeling remains debated. This study aimed to investigate the longitudinal biventricular cardiac response to a 20-week high-intensity resistance training program in previously untrained, healthy males and examine the association between muscle strength gains and cardiac remodeling.

Methods Twenty-seven male volunteers (aged 18–40 years) participated in a high-intensity resistance training program for 20 weeks. Assessments at baseline, 12 weeks, and 20 weeks included resting blood pressure, electrocardiogram (ECG), three-dimensional transthoracic echocardiography (3DTTE), cardiopulmonary exercise testing ($\dot{V}O_{2peak}$), isokinetic dynamometry for muscle strength, and actimetry recordings. Time effects were analyzed using one-way repeated measures ANOVA ($P < 0.05$).

Results Twenty-two participants completed the study. Resistance training led to significant reductions in arterial systolic and diastolic blood pressure and heart rate. After 20 weeks of training, 3DTTE showed a significant increase in left ventricular (LV) mass (120.1 ± 15.4 g vs. 133.7 ± 16.3 g, $p < 0.001$), without inducing LV hypertrophy. Balanced increases were observed in LV end-diastolic volume (146.4 ± 18.9 ml vs. 157.9 ± 19.6 ml, $p < 0.001$) and right ventricular (RV) end-diastolic volume (119 ± 19.4 ml vs. 129.2 ± 21.6 ml, $p < 0.001$). LV and RV systolic and diastolic function remained unchanged. There were no changes in $\dot{V}O_{2peak}$ or daily activity levels. Maximal muscle strength in the quadriceps, hamstrings, triceps, and biceps was significantly correlated with LV and RV end-diastolic volumes and LV mass ($p \leq 0.001$).

Conclusion The resistance training program resulted in significant and rapid muscle strength gains and reduced blood pressure. Cardiac adaptations, including moderate biventricular dilatation, were observed without changes

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in cardiac function or $\dot{V}O_{2peak}$ and were associated with muscle strength gains. Our study highlights that intensive resistance training in novice male resistance trainers induces an adaptive cardiac response, reflecting a physiological adaptation linked to enhanced muscle performance.

Trial registration ClinicalTrials.gov ID: NCT04187170.

Keywords Athlete's heart, Resistance training, Muscle strength, Cardiac hypertrophy, 3D echocardiography, Biventricular cardiac remodeling

Background

Strength training is recommended to improve health [1], yet uncertainties persist regarding the risk of developing maladaptive cardiac remodeling in strength athletes [2]. Since the postulate of divergent cardiac adaptations in dynamic and static sports [3, 4], a left ventricular (LV) concentric hypertrophic pattern has been documented alongside progressive alterations in diastolic function and vascular endothelial function among elite strength athletes [5–7]. However, directly attributing this potentially maladaptive response to strength exercise alone remains challenging [8]. Evidence suggests that in healthy individuals, intense strength exercise is not associated with significant alterations in LV wall stress due to cardiopulmonary mechanisms that minimize the increase in transmural pressure [9]. Conversely, confounding factors, such as anabolic agent use [10, 11] and a higher prevalence of cardiovascular risk factors like hypertension and obesity among certain strength-trained athlete [12, 13], may contribute to the development of maladaptive cardiac responses.

The current literature on cardiac adaptation in strength athletes predominantly relies on cross-sectional studies, which primarily focus on the left side of the heart [5, 14–17]. These studies often involve varying types of resistance training and individuals with pre-trained hearts, complicating data comparisons. Only a limited number of longitudinal studies have explored the global cardiac response to resistance training, particularly in untrained, healthy individuals without pre-existing cardiac remodeling and with a low likelihood of performance-enhancing substance use [18, 19]. These studies have yielded conflicting results regarding the presence of cardiac adaptation to resistance training and have not fully explored the potential relationship between muscle strength gains and cardiac adaptation due to insufficiently assessments of muscular performance and have not fully explored the potential relationship between muscle strength gains and cardiac adaptation. One potential explanation is the method used to measure strength gains. While 1-RM testing is commonly used in non-laboratory settings, it may not fully capture overall strength changes from resistance training. Isokinetic testing, considered the "gold standard," provides a more comprehensive assessment by evaluating force production across different joint angles and velocities, offering a better understanding neuromuscular adaptations and their potential interaction with cardiac adaptations resulting from the training program. Additionally, the lack of standardized, sufficiently intense training protocols with muscle strength re-assessments during the program further hindered a comprehensive understanding of the expected physiological effects of high-intensity resistance training (i.e. ≥70% of 1-RM) previously validated on cardiac response [17, 18].

Our team previously validated a 20-week high-intensity resistance training program that demonstrated its feasibility and effectiveness in significantly increasing maximal muscle strength in the upper and lower body according to existing results in resistance training [20–22], as measured by the dynamometry method, among previously untrained healthy male volunteers [23]. The present study aimed to describe the longitudinal biventricular cardiac response to this 20-week high-intensity resistance training program in participants without pre-existing cardiac remodeling using 3D echocardiography, and to assess the association between muscle strength gains and both left and right ventricular exercise-induced adaptations.

Methods

Participants

Twenty-seven male volunteers, aged between 18 and 40 years, were recruited for this study among the students and personnel of the University of Caen Normandy (France). Eligible participants were free from known cardiovascular, liver, renal, respiratory, and metabolic diseases, non-smokers, non-hypertensive, non-diabetic, and had a body mass index < 35 $kg.m^{-2}$. None of the participants practiced more than 75 min of sustained physical training (endurance or resistance training) per week in the three years leading up to the study. Individuals displaying abnormalities in the initial examinations were excluded. No participants declared the use of drugs or doping products.

Design

Before starting the 20-week high-intensity resistance training program, all participants underwent a clinical evaluation that included measurements of height, weight, and body composition (mBCA 525 impedance

meter SECA) as previously described [23]. Body composition was determined in the supine position to determine the proportion and distribution of fat and muscle mass. Body mass index was calculated as follows: weight (kg)/height (m)^2. Arterial blood pressure was measured after 10 min of quiet rest in a supine position. Blood pressure was assessed on the left arm using a Dynamap V100 automatic pressure cuff (GE Medical), with the cuff size adapted to each participant's arm circumference. Two consecutive measurements were averaged. Additionally, electrocardiogram (ECG), transthoracic echocardiography (TTE), and isokinetic dynamometry were conducted to assess cardiac and muscular adaptations. These assessments were repeated at 12 and 20 weeks during the training program. Furthermore, a determination of maximal oxygen uptake ($\dot{V}O_{2peak}$) by cardiopulmonary exercise testing was conducted before and after 20 weeks of resistance training. Finally, actimetry recordings were obtained to evaluate the circadian rhythm of the sleep-wake cycle before and after the initial 12 weeks of training. All assessments were conducted at the Sports Medicine Unit of the University Hospital of Caen Normandy, ensuring consistent measurement conditions throughout the study. Participants were instructed to abstain from exercise for 24 h before data collection. No specific instructions were given regarding food intake or sleep prior to testing sessions. The study design is illustrated in Fig. 1 and has been thoroughly documented in a previous publication [23].

Electrocardiogram analysis

The cardiac electrical response to resistance training was assessed using resting 12-lead ECGs recorded in the supine position during quiet respiration after participants had been resting in that position for at least 10 min. ECGs were recorded at a speed of 25 mm/s and standardized calibration for 10 mm/mV (Schiller Cardiovit AT-102 G2). ECG assessments followed international guidelines and included heart rate, PR interval, QRS axis, QRS interval, and QT interval duration. The corrected QT interval was calculated using Bazett's formula using the tangent method [24]. Cornell voltage criteria for left ventricular hypertrophy (SV3 + RaVL ≥ 2.8 mV in males) was also evaluated [25].

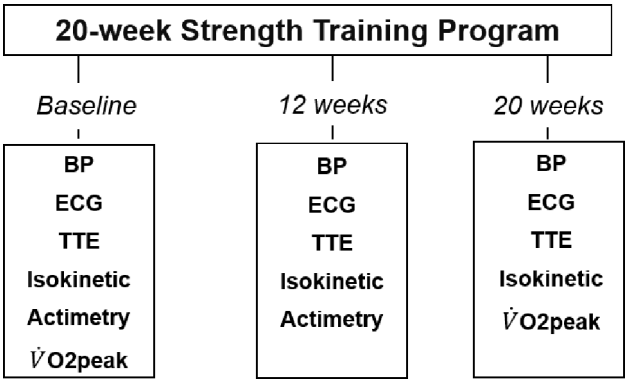

Fig. 1 Study design (BP: blood pressure, ECG: electrocardiogram, TTE: transthoracic echocardiography, $\dot{V}O_{2peak}$: maximal oxygen uptake)

Transthoracic echocardiographic assessment

Cardiac morphological and functional responses to resistance training were studied using a commercially available echocardiographic system (EPIQ 7 equipped with an X5-1 xMATRIX-array transducer, Philips). Participants were scanned at rest in the left lateral decubitus position at each stage of the protocol. All echocardiographic measurements acquired during a brief apnea were stored digitally for offline data analysis performed by a single operator (AH) blinded to the study time point (TOMTEC-Arena TTA2, TOMTEC Imaging Systems GMBH, Germany). Standard two-dimensional (2D), three-dimensional (3D), Doppler, and tissue Doppler imaging (TDI) measurements of chamber structure and function were made in accordance with the current guidelines [26]. Left ventricular (LV) dimensions, LV wall thickness, and aortic root were measured from B-mode acquisitions in a standard parasternal long-axis view. Relative wall thickness (RWT) was calculated as follows: [interventricular septal thickness (mm) + posterior wall thickness (mm)]/LV end-diastolic diameter (mm). Right ventricular (RV) free wall thickness was measured from the subcostal view. Left atrial (LA) and right atrial (RA) end-systolic volumes were measured by the biplane and monoplane methods of disks, respectively. LV and RV 3D volumes, ejection fraction (EF), and LV mass were obtained from separate sequences for the LV and RV 3D acquisitions and post-processed using the TOMTEC analysis software for 3D LV- and RV-Function. LV hypertrophy was defined as an LV mass > 116 g/m^2, classified as concentric hypertrophy if RWT > 0.42 and eccentric hypertrophy if RWT ≤ 0.42 [26]. LV global longitudinal strain (GLS) was obtained from each of the three apical views using speckle tracking. RV regional systolic function was assessed using tricuspid annular plane systolic excursion (TAPSE) in M-mode and peak RV lateral systolic (RV S') myocardial velocity in Doppler tissue imaging (DTI). The diastolic biventricular function was assessed by measuring peak early (E_m) and late (A_m) transmitral, and peak early (E_t) and late (A_t) transtricuspid flow Doppler velocities. Peak early diastolic mitral annulus (e_m') velocity (average of septal and lateral peaks) and peak early diastolic tricuspid lateral annulus (e_t') velocity were obtained from DTI. Doppler parameters were obtained as the average value of three consecutive cardiac cycles.

Muscle strength assessment

Muscle strength was assessed in both the upper and lower limbs using a Con-Trex® isokinetic dynamometer (Con-Trex MJ; Dübendorf, Switzerland) and field-based 1-RM tests. Isokinetic strength was tested in a seated position and evaluated through 4 maximal repetitions (reps) at 60°/s in concentric mode for knee extensors and flexors, and through 4 maximal reps at 150°/s in concentric mode for elbow extensors and flexors. The peak torque (in Newton-meters, N m) from the best repetition was recorded for both the agonist and antagonist muscle groups. All assessments were performed with free breathing. The detailed methodology and results of muscle strength assessments using isokinetic dynamometry have been previously reported [23].

The 1-RM testing protocol began with a load estimated from the participant's perceived strength capacity. Resistance was progressively increased across trials until the participant could no longer complete a full repetition with proper technique [27]. The highest load lifted successfully defined the 1-RM (in kilograms, kg). This measure was subsequently used to tailor training intensity, ensuring consistent high-intensity effort throughout the strength training program. Loads were expressed as percentages of the 1-RM (based on the concentric phase of the lift). The description of the exercises tested and the kinetics of 1-RM strength gains across the training program has been previously published [23]. Investigators provided consistent verbal encouragement to all participants during both isokinetic and 1-RM assessments.

Cardiopulmonary exercise testing

To evaluate the impact of resistance training on aerobic capacity, maximal oxygen uptake ($\dot{V}O_{2peak}$) was assessed through cardiopulmonary exercise testing on an electronically braked ergometer (Ergoline, Ergoline GmbH) both before and after the 20 weeks of resistance training. The cardiopulmonary exercise testing protocol included 1 min of rest in a seated position, followed by a 3-minute warm-up at a load of 50 watts. The workload was then increased by 30 watts every 3 min until exhaustion, despite verbal encouragement. Blood pressure was measured every 2 min using a manual BP cuff with continuous heart rate and 12-lead ECG monitoring. Respiratory gas analysis was conducted on a breath-by-breath basis using the Medisoft gas analyzer (ExpAir Soft). The following parameters were determined by two trained and independent operators: maximum oxygen uptake ($\dot{V}O_{2peak}$), first ventilatory threshold (VT1), and second ventilatory threshold (VT2). Exercise capacity was assessed based on $\dot{V}O_{2peak}$, which was calculated as the average of values obtained during the final 20 s of exercise. The determination of VT1 and VT2 was performed using a graphical method, with VT1 identified at the nadir of the $\dot{V}_E/\dot{V}O_2$ ratio relative to workload, and VT2 determined at the nadir of the $\dot{V}_E/\dot{V}CO_2$ ratio relative to workload [28]. A respiratory exchange ratio greater than 1.1 and a peak exercise heart rate exceeding 85% of the estimated maximal heart rate were used to assess test adequacy measures indexed to body weight. Cardiopulmonary exercise testing was carried out according to guidelines [29, 30].

Actimetry recording

Actimetry recordings were performed before training and after 12 weeks to assess the sleep-wake cycle. For each participant, the sleep-wake cycle was recorded continuously using the Motion Watch 8 wrist-worn triaxial actigraph (CamNTech Ltd), wore during 7-day continuously, except during water immersion. The sleep diary was used to complement actigraphy in determining when participants were awake and when they were asleep. The following parameters were recorded: (1) bed time; (2) sleep time; (3) wake-up time; and (4) total sleeping time. In addition, the activity during the 5 least active hours and the 10 most active hours were recorded, and the absolute amplitude of rhythm (difference between the 10 most active hours and the 5 least active hours) was calculated and expressed in counts per minute.

Training protocol

The resistance training protocol consisted of three supervised weekly sessions over 20 weeks, conducted at a fully equipped university sports center dedicated to resistance training. The program included concentric, eccentric, and isometric phases, organized into two successive cycles of 12 and 8 weeks, with exercises targeting the upper and lower body muscles at high loads (≥ 70% of 1-RM) following the American College of Sports Medicine's guidelines [30]. This intensity level is consistent with high-intensity resistance training and has been shown to significantly improve muscle strength within a short period (typically between 8 and 14 weeks) in adults with or without diseases [30–32]. The concentric phase consisted of 8 reps at 70% of 1-RM, 6 reps at 80%, 3 reps at 90%, and 2 reps at 90%. The eccentric phase included 4 sets of 6 reps at 90% of 1-RM, followed by progressively heavier eccentric reps (100–130% of 1-RM) paired with concentric reps (50–90% of 1-RM), culminating in 3 sets of 2 eccentric reps at 130% and 10 explosive concentric reps at 50%. The isometric phase involved 5 sets of 8 and 4 stato-dynamic reps at 50% of 1-RM, along with varying isometric holds (6–20 s) combined with explosive concentric reps at 80%. Individualized assessment of muscle training progression was monitored through regular 1-RM assessments, conducted at the beginning of the training program and then repeated at 4, 8, 12, and 20 weeks of training. This approach allowed for adjustments

in exercise intensity for each training cycle to maintain high intensity throughout the program. No breaks were planned to ensure continuous resistance training intensity. The detailed description of the resistance training protocol is available in our study design publication [23].

Statistical analysis

Statistical analyses were performed using JASP (JASP Software, Amsterdam, The Netherlands). The normality of the distribution was verified using the Shapiro-Wilk test. A repeated measures ANOVA was used to compare time effects, with the Bonferroni post hoc test applied to identify specific differences between each evaluation. Prior to the ANOVA, Mauchly's test of sphericity was conducted to assess the assumption of sphericity. If sphericity was violated, the Greenhouse-Geisser correction was applied. Partial eta squared (η^2) was reported for effect size in the muscular isokinetic analysis and classified as small ($\eta^2 = 0.01$), medium ($\eta^2 = 0.06$), or large ($\eta^2 > 0.14$). The correlation between two quantitative variables was assessed using Pearson's correlation coefficient. A p-value of 0.05 was considered the threshold for statistical significance.

Results

Clinical evaluation

The 20-week high-intensity resistance training program was completed by 22 participants. Five participants who did not complete the protocol were excluded from the final analysis (4 participants were lost to follow-up after the 12-week assessment, and 1 participant was excluded for medical reasons outside the protocol).

The characteristics of the participants are presented in Table 1. Body mass index and muscle mass were increased after 12 and 20 weeks of training compared to baseline, without changes in body fat mass and lean mass. Both systolic and diastolic blood pressure significantly decreased after 20 weeks of resistance training compared to baseline. Additionally, systolic blood pressure also significantly decreased between the 12- and 20-week assessments.

The resistance training did not affect $\dot{V}O_{2peak}$ and ventilatory thresholds, but it did result in a significant increase of 8.3% in the mean maximal load (Table 1). Concerning sleep-wake cycle measurement, there were no significant changes observed in total sleeping time, including bedtime or wake-up time, and the mean movements during both the least active (L5) and most active (M10) periods remained unchanged (Table 1).

Cardiac morphological and functional adaptations to resistance training

Resting heart rate, as measured on ECG, demonstrated a significant decrease at both 12 weeks and 20 weeks of training compared to baseline (Table 2). There were no other modifications in the ECG parameters over the 20

Table 1 Physical and cardiac fitness characteristics of participants

Variables	Baseline	12-week	20-week	Interaction
Age (years)	22.8 ± 3.2	-	-	-
Height (m)	1.78 ± 0.06	-	-	-
Weigh (kg)	69.8 ± 10.8	72.1 ± 10.1*	72 ± 11$^{\#\#}$	**< 0.001**
BMI (kg/m^2)	22.1 ± 3	22.8 ± 2.7*	22.8 ± 3$^{\#\#}$	**< 0.001**
Body fat mass (kg)	11.3 ± 6.8	11.7 ± 7	12.4 ± 6.8	0.164
Body muscle mass (kg)	28.9 ± 3.4	30 ± 3.8*	29.5 ± 3.4	**0.003**
Systolic BP (mmHg)	124.6 ± 9.9	121 ± 7 9	114.8 ± 10.1$^{\#\#\ddagger}$	**< 0.001**
Diastolic BP (mmHg)	68.3 ± 8.2	64.6 ± 5.9	62.8 ± 8$^{\#}$	**0.016**
Aerobic capacity fitness				
$\dot{V}O_{2peak}$ ($mLO_2 \cdot min^{-1}$)	2833 ± 574	-	2969 ± 526	0.12
$\dot{V}O_{2peak}$ ($mLO_2 \cdot kg^{-1} \cdot min^{-1}$)	40.9 ± 7.5	-	41.5 ± 5.8	0.634
VT1 ($mLO_2 \cdot kg^{-1} \cdot min^{-1}$)	26.4 ± 8.4	-	26.0 ± 7.8	0.779
VT2 ($mLO_2 \cdot kg^{-1} \cdot min^{-1}$)	33 ± 7.7	-	33.7 ± 6.9	0.602
$\dot{V}O_{2peak}$ maximal load (Watts)	180.9 ± 40.9	-	195.9 ± 36.2$^{\#\#}$	**< 0.001**
Sleep-wake cycle				
Bedtime (hh: min)	00:51 ± 00:54	01:10 ± 00:57	-	0.083
Wake-up time (hh: min)	08:47 ± 00:59	08:31 ± 01:18	-	0.230
Total sleeping time (min)	411.5 ± 59.7	385.3 ± 65.3	-	0.059
L5 (counts per minute)	15.3 ± 9.9	21.3 ± 23.2	-	0.711
M10 (counts per minute)	215.9 ± 55.4	236.6 ± 51.5	-	0.114

Values are presented as mean ± SD. Abbreviations: BMI: body mass index; BP: blood pressure; M10: mean of movement over the least active period; L5: mean of movement over the most active period; $\dot{V}O_{2peak}$: maximum oxygen consumption; VT: ventilatory threshold

*$p \leq 0.05$ between baseline and 12-week, $^{\#}p \leq 0.05$ between baseline and 20-week, $^{\#\#}p \leq 0.001$ between baseline and 20-week, $^{\ddagger}p \leq 0.05$ between 12-week and 20-week

Table 2 ECG data

Variables	Baseline	12-week	20-week	Interaction
Heart rate (bpm)	76.8 ± 8.6	66.9 ± 10.6*	70.4 ± 9.4#	**< 0.001**
P duration (ms)	99.8 ± 14.1	98 ± 16.2	104.1 ± 15.1	0.138
P amplitude (mm)	1.65 ± 0.56	1.45 ± 0.51	1.32 ± 0.61#	**0.015**
PR duration (ms)	137.5 ± 22.6	135.6 ± 25	142.9 ± 23.6	0.111
QRS axis (°)	72.6 ± 19.4	73.5 ± 29.4	70.1 ± 21.4	0.211
QRS voltage (mV)	11.9 ± 4.4	12.8 ± 4.8	13 ± 5.1	0.224
QRS duration (ms)	96.1 ± 8.4	93.5 ± 10	97.5 ± 10.5	0.796
QTc duration (ms)	413.3 ± 15	408.1 ± 20.8	408.2 ± 17.9	0.7

Values are presented as mean ± SD

*$p \leq 0.05$ between baseline and 12-week, #$p \leq 0.05$ between baseline and 20-week

weeks of resistance training, except for a slight decrease in the P wave amplitude (Table 2).

The echocardiographic measurements are summarized in Table 3. A significant increase in 3D LV mass was observed after 12 weeks of training, which persisted throughout the training program (Fig. 2). No increase in GLS LV was observed. Furthermore, there was a significant increase in LV septal and posterior wall thickness as well as in RV free wall thickness at the end of training. No participant exhibited LV wall thickness greater than 11 mm. Both LV and RV end-diastolic and end-systolic volumes were significantly increased and the RV/LV ratio remained unchanged. These modifications were observed as early as 12 weeks into the resistance training program (Table 3; Fig. 2). Significant correlations were observed between 3D RV EDV and 3D LV EDV ($r = 0.74$, $p \leq 0.001$), and 3D RV EDV and 3D LV mass ($r = 0.8$, $p \leq 0.001$).

Additionally, LV and RV systolic and diastolic functions were preserved and unchanged throughout the protocol (Table 3). Finally, 20 weeks of training did not induce any changes in left and right atrial volumes or aortic root diameter (30.1 ± 2.6 mm vs. 30.4 ± 2.2 mm, $p = 0.56$).

Table 3 Cardiac morphological and functional parameters at baseline, 12 weeks, and 20 weeks of resistance training

Variables	Baseline	12-week	20-week	Interaction
Left Ventricle (LV)				
LV septal wall thickness (mm)	6.1 ± 1.1	6.6 ± 1.5	7.2 ± 1.4#	**0.027**
LV posterior wall thickness (mm)	5.5 ± 1.3	6.6 ± 1.4*	7.1 ± 1.3##	**< 0.001**
RWT	0.24 ± 0.05	0.28 ± 0.05*	0.29 ± 0.06##	**0.007**
3D LV mass (g)	120.1 ± 15.4	132.9 ± 15.6*	133.7 ± 16.3##	**< 0.001**
3D LV EDV (ml)	146.4 ± 18.9	156.5 ± 20.8*	157.9 ± 19.6##	**< 0.001**
3D LV ESV (ml)	64 ± 10	69.4 ± 12.3*	68.1 ± 11.8#	**0.003**
3D LV ejection fraction (%)	56.5 ± 3.3	55.9 ± 3.8	57 ± 4.5	0.789
LV GLS (%)	20.7 ± 1.8	20.3 ± 1.6	20.4 ± 1.7	0.28
Peak E_m ($cm.s^{-1}$)	76.7 ± 10.4	77.6 ± 12.1	79.6 ± 10.9	0.557
Peak A_m ($cm.s^{-1}$)	44.5 ± 8.9	44 ± 8.2	43.9 ± 7.6	0.926
E_m/A_m ratio	1.79 ± 0.47	1.83 ± 0.5	1.86 ± 0.39	0.770
averaged e_m' ($cm.s^{-1}$)	15.1 ± 1.9	15.4 ± 2.1	15.3 ± 2.6	0.575
Right Ventricle (RV)				
RV wall thickness (mm)	3.33 ± 0.73	3.76 ± 0.71	3.89 ± 0.8#	**0.014**
3D RV EDV (ml)	119 ± 19.4	126.7 ± 18.7*	129.2 ± 21.6##	**< 0.001**
3D RV ESV (ml)	53.9 ± 11.5	58.4 ± 13.3*	58.2 ± 11.3##	**0.004**
3D RV ejection fraction (%)	54.7 ± 6	54.1 ± 5.4	54.9 ± 4.3	0.789
RV/LV EDV ratio	0.81 ± 0.09	0.81 ± 0.08	0.82 ± 0.1	0.950
Peak RV S' ($cm.s^{-1}$)	13.5 ± 2.1	13.1 ± 2.1	13.2 ± 2.2	0.595
TAPSE (mm)	22.5 ± 3.3	24.1 ± 3.5	24.9 ± 3.3	**0.019**
Peak E_t ($cm.s^{-1}$)	49.4 ± 7.3	51.4 ± 7.8	50.5 ± 6	0.582
Peak A_t ($cm.s^{-1}$)	32.1 ± 4.7	30.7 ± 5.8	31.5 ± 8.3	0.761
E_t/A_t ratio	1.55 ± 0.23	1.71 ± 0.3	1.68 ± 0.38	0.353
Lateral e'_t ($cm.s^{-1}$)	12.4 ± 2.9	12.9 ± 2.8	13.2 ± 2.7	0.109
Atrial volumes				
Left atrial volume (mL)	33.2 ± 9.4	32.6 ± 7.6	32.1 ± 8.7	0.778
Right atrial volume (mL)	31.3 ± 8.5	32.9 ± 8.3	33.1 ± 8.7	0.617

Values are presented as mean ± SD. Abbreviations: A_m: late transmitral flow Doppler velocity; A_t: late transtricuspid flow Doppler velocity; e_m': early diastolic mitral annulus (septal and lateral) velocity; e_t': early diastolic tricuspid lateral annulus velocity; E_m: peak early transmitral flow Doppler velocity; E_t: early transtricuspid flow Doppler velocity; EDV: end-diastolic volume; ESV: end-systolic volume; GLS: global longitudinal strain; RV S': right ventricle lateral systolic myocardial velocity; RWT: relative wall thickness; TAPSE: tricuspid annular plane systolic excursion

*$p \leq 0.05$ between baseline and 12-week, #$p \leq 0.05$ between baseline and 20-week, ##$p \leq 0.001$ between baseline and 20-week

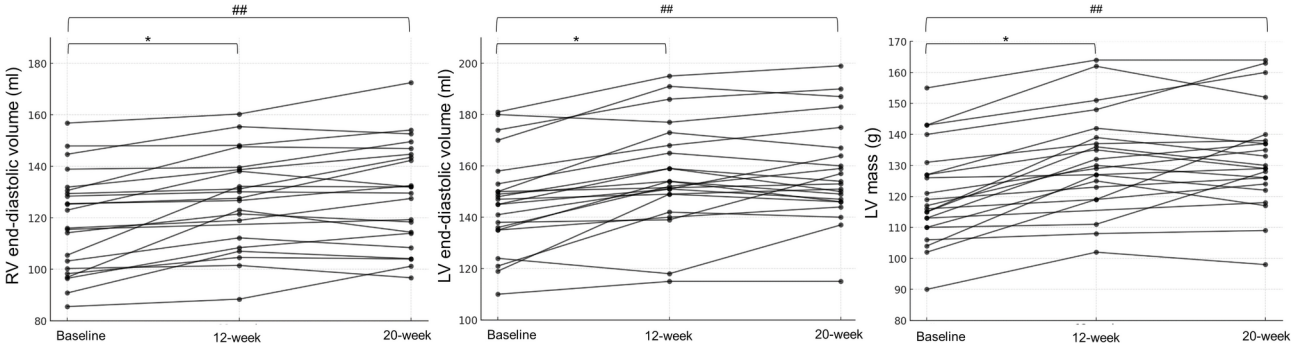

Fig. 2 Kinetics of LV mass and biventricular end-diastolic volumes assessed using 3D echocardiography over the training period. $^{*}p \leq 0.05$ between baseline and 12-week, $^{\#\#}p \leq 0.001$ between baseline and 20-week

Table 4 Correlations between cardiac structure and muscle variables

Variables	3D RV EDV		3D LV EDV		3D LV mass	
	r	*P*	*r*	*P*	*r*	*P*
Body muscle mass	0.70	**≤ 0.001**	0.87	**≤ 0.001**	0.87	**≤ 0.001**
Muscle strength						
Triceps strength	0.47	**≤ 0.001**	0.62	**≤ 0.001**	0.72	**≤ 0.001**
Biceps strength	0.43	**≤ 0.001**	0.64	**≤ 0.001**	0.71	**≤ 0.001**
Quadriceps strength	0.42	**≤ 0.001**	0.58	**≤ 0.001**	0.64	**≤ 0.001**
Hamstring strength	0.45	**≤ 0.001**	0.51	**≤ 0.001**	0.61	**≤ 0.001**
1-RM assessment						
Bench press	0.54	**≤ 0.001**	0.65	**≤ 0.001**	0.70	**≤ 0.001**
Biceps curl on a desk	0.38	**0.0017**	0.53	**≤ 0.001**	0.60	**≤ 0.001**
Leg-extension	0.46	**≤ 0.001**	0.57	**≤ 0.001**	0.67	**≤ 0.001**
Leg-curl	0.59	**≤ 0.001**	0.67	**≤ 0.001**	0.75	**≤ 0.001**
Leg press	0.52	**0.017**	0.48	**≤ 0.001**	0.52	**0.016**

Pearson's correlation coefficient ($P < 0.05$). Abbreviations; EDV: end-diastolic volume, LV: left ventricle, RV: right ventricle

Relationship between muscle strength gains and cardiac bi-ventricular adaptation

Significant correlations were observed between cardiac morphological 3D echocardiographic parameters and muscular performance indices recorded throughout the entire protocol (Table 4). First of all, body muscle mass was significantly correlated with 3D measurements of RV EDV, LV EDV, and LV mass. The overall dynamometry assessments of maximal muscle strength in the biceps, triceps, quadriceps, and hamstrings were also significantly correlated with these 3D cardiac parameters. Additionally, significant correlations were also found between these 3D cardiac parameters and 1-RM assessments for the bench press, biceps curl on desk, leg extension, leg curl, and leg press (Table 4).

Discussion

The main findings of our study, conducted through a longitudinal protocol within a healthy, untrained male population, revealed that resistance training, implemented with a controlled high-intensity exercise regimen resulted in rapid and substantial gains in maximal force without concomitant improvements in aerobic capacity or sleep-wake cycle activities, was associated with several noteworthy outcomes. Firstly, there was a significant reduction in both systolic and diastolic arterial blood pressure and resting heart rate. Secondly, a physiological, moderate biventricular cardiac response was observed, characterized by 3D echocardiography as biventricular enlargement and a balanced increase in LV mass and cardiac volumes with preserved systolic and diastolic functions. These changes were significantly correlated with muscle strength gains, as assessed by isokinetic testing and 1-RM. The results of this study contribute to a deeper understanding of the physiological cardiovascular impacts of high-intensity resistance training in previously untrained males, and for the first time, report a relationship between biventricular geometric cardiac modifications and maximal strength gains, as recorded using a reference technique such as dynamometry.

Positive impact of high-intensity resistance training on arterial blood pressure

In our previously physically inactive, healthy young male cohort, we observed a significant reduction in resting systolic and diastolic blood pressure following resistance

training. Although we cannot entirely exclude the influence of psychological factors at baseline on these cardiovascular parameters, a consistent and progressive decrease in blood pressure was noted throughout the follow-up period. Only one participant initially presented with high systolic blood pressure at 140/65mmHg, according to international guidelines [33], which normalized during the evaluations at 12 and 20 weeks. Numerous studies have consistently demonstrated the effectiveness of dynamic and isometric resistance training in reducing blood pressure in individuals both with and without cardiovascular disease [34, 35]. The mechanism underlying the effect of resistance training on reducing blood pressure appears to be associated with improvements in vascular conductance and endothelial function [36]. The American Heart Association endorses progressive resistance training for improving cardiovascular health in adults with and without cardiovascular disorders [1]. Our data aligns with these findings, providing additional evidence of the positive impact of short-term resistance training on blood pressure levels, particularly in high-intensity resistance training with rapid increments in a previously untrained population at low cardiovascular risk. As previously reported in this population, bioelectrical impedance data indicated that our participants maintained an average body fat mass of about 16%, and consequently an average lean body mass of approximately 84%, further substantiating their low-risk profile [23]. When analysing cardiovascular responses in athletes, it is essential to consider individual cardiovascular risk factors, particularly in strength-based discipline as these factors can lead to cardiac functional impairments [37]. Athletes with increased body fat percentage, hypertension, dyslipidemia, and impaired fasting glucose may face an increased risk of developing early endothelial dysfunction and cardiovascular events [38]. Recently, Tso et al. showed an association between collegiate athletes engaged in competitive American-style football and an increase in the diameter of the aortic root, noted within the first year of the study and up to 3 years of follow-up [7]. The authors concluded that this maladaptive pattern of aortic remodeling appeared to be more closely linked to a higher level of cardiovascular risk factors among these young athletes rather than the type of resistive or non-resistive training, as described based on their position on the field. While our follow-up was limited to 20 weeks, and the assessment did not include the evaluation of vascular function, our high-intensity resistive training conducted in a healthy, low-cardiovascular risk population does not seem to impact the aortic root dimension.

Impact of muscle strengthening on cardiac structure and function

The impact of sustained resistance training on cardiac structure and function has been inconsistently reported in longitudinal studies. Kanakis and Hickson [39] showed over 30 years ago that a ten-week high-intensity resistance training program, performed five days per week and focused on lower-extremity strength (alternating exercises, including 3 to 5 sets of 5 reps each of parallel squats, leg flexions, leg extensions, leg presses, and 3 sets of 20 reps of calf raises) in young, untrained males significantly increased LV mass and fractional shortening while reducing post-training heart rate, though it did not improve maximal oxygen uptake. Conversely, Au et al. found no changes in LV size or function after 12 weeks of resistance training in young, healthy males, consisting in 20–25 reps/set to failure at ~30-50% of 1-RM in higher-rep group and 8–12 reps/set to failure at ~75-90% in lower-rep group, potentially due to prior resistance training history of participants [40]. Studies examining longer durations of resistance training have also displayed conflicting results. Spence et al. observed no significant biventricular changes after 24 weeks of resistance training in young, untrained men, possibly due to insufficient exercise intensity [17, 18], whereas the study by Sharf et al. noted balanced biventricular dilatation after only 22 weeks in untrained middle-aged men, though muscle strength was not assessed [19]. Our results align with the notion that prolonged high-intensity resistance training could induce a moderate, balanced biventricular cardiac response, characterized by slight increases in LV mass and biventricular enlargement, without changes in systolic or diastolic function. Despite a small rise in LV relative wall thickness from 0.24 to 0.29, no participant in our study exhibited LV hypertrophy or concentric hypertrophic pattern as previously defined [26]. The absence of LV electrical hypertrophy on ECG supports these findings. Unlike Scharf et al. [19], we observed no changes in atrial dimensions, possibly due to the lower sensitivity of 2D echocardiography compared to magnetic resonance imaging in detecting small volumetric changes.

Although our cohort differs from long-term strength athletes, our findings are consistent with cross-sectional studies suggesting that concentric LV hypertrophy is uncommon among elite strength athletes [41, 42]. This study underscores the importance for clinicians to thoroughly assess strength athletes for risk factors associated with maladaptive cardiac remodeling, particularly when a concentric LV hypertrophic pattern is accompanied by early signs of myocardial impairment. Such findings warrant a detailed investigation to rule out underlying pathology. The observations provided by this study are particularly valuable in sports medicine practice, as they enhance our understanding of the distinction

between physiological and potentially pathological cardiac adaptations. Our longitudinal data further demonstrate that 3D RV and LV EDV as well as LV mass, closely correlate with muscle strength gains. This corroborates Miranda et al.'s cross-sectional findings showing a correlation between RV basal diameter and 1-RM bench press strength among bodybuilders [43]. However, generalizing the cardiovascular response to strength training remains challenging, as adaptations vary depending on training modalities, which can significantly differ between strength disciplines, particularly between weightlifters and bodybuilders [8]. The latter typically engage in strength training with a greater dynamic component, leading to higher preload and stroke volume adaptation. Although our resistance training protocol was based on a high-load, low-repetition approach, our data suggest that slight RV enlargement with preserved function can be a physiological adaptation if it occurs alongside balanced LV remodeling within normal ranges following such training. The nature of our training program, which incorporated distinct concentric, eccentric, and isometric phases, was designed to maximize muscle strength gains while minimizing musculoskeletal injury risk in a population of novice resistance trainers as recommended by the American College of Sports Medicine [30]. This could explain the more homogeneous cardiac response with moderate wall thickening associated with biventricular enlargement observed in our study population compared to high-intensity training protocols relying essentially on concentric exercises. Furthermore, for the first time, this study highlights the relationship between cardiac volumetric changes and enhanced muscle function, assessed using both isokinetic dynamometry and 1-RM tests. While the 1-RM test is a reliable measure of muscle performance, it does not assess maximal muscle strength across different angular velocities and may overestimate strength gains, particularly at higher strength levels, compared to the gold-standard isokinetic evaluation [44]. Although our high-intensity resistance training shares principles with strength-based disciplines such as weightlifting, powerlifting, strongman, and high-intensity functional training, further studies using dedicated exercises are needed to confirm these findings.

Finally, unchanged assessments of both aerobic capacity and actimetry support that the observed cardiac changes were primarily attributable to resistance training, excluding the influence of covert endurance training during the study follow-up. Improvements in aerobic capacity are typically associated with endurance training or resistance training focused on muscular endurance. In contrast, resistance training designed to increase maximal muscle strength does not significantly enhance maximal aerobic capacity, which aligns with our findings [39, 45]. In addition, the sleep-wake cycle and activity parameters showed no significant changes in daily physical activity outside of training periods, further supporting participants' adherence to the prescribed protocol. Although the measurement was only carried out after 12 weeks, the first period of training program lasted 3 months and included 34 training sessions, which is in line with the protocols usually used in adapted physical activity and chronobiology in the literature, showing that this is sufficient to produce changes in the sleep-wake cycle [46]. While actimetry data were collected only at the 12-week mark, our findings suggest that the majority of physiological adaptations occurred early in the program, as expected in previously untrained individuals undertaking high-intensity resistance training. Additionally, Langeard et al. demonstrated that 12 weeks of high-intensity resistance training in older adults did not promote more active daily behavior, partly due to the muscular fatigue induced by the training [47]. Lastly, our training protocol did not alter participants' sleep patterns, which is particularly relevant, as poor sleep quality is known to impair muscle gains and performance [48].

Limitations

This study included only male participants, limiting the generalizability to female populations. However, this selection aligns with established sex differences in cardiac adaptation to training, as men are more prone to developing exercise-induced concentric ventricular hypertrophy compared to women [49]. Examining this population, which is more susceptible to such remodeling, allowed us to assess whether this hypertrophy represents a physiological response to high-intensity resistance training. The absence of such a response in our cohort further supports our conclusion. Similarly, our findings may not apply to older populations. However, comparable cardiac responses have been previously reported in untrained middle-aged men [19]. Finally, although the participants did not exhibit concentric myocardial hypertrophy, our protocol was confined to a 20-week training period. Consequently, we cannot rule out the possibility that a more prolonged practice could yield a different cardiac response. However, the longitudinal follow-up indicated a decrease in the kinetics of the cardiac response with the subject's training after 12 weeks. Moreover, previous observations among strength athletes revealed that 12 weeks of participation in university American-style football competition were sufficient to induce concentric LV hypertrophy with impaired diastolic function [5], suggesting that our 20-week high-intensity resistance training program was adequate to significantly impact cardiac response.

Conclusion

Our longitudinal study confirmed that, in a population of novice male resistance trainers, 20 weeks of high-intensity resistance training was sufficient to induce a physiological cardiac response. This response included reductions in arterial pressure and heart rate, along with moderate and balanced biventricular remodeling, preserved cardiac function, and no significant ventricular hypertrophy. These changes were associated with improvements in muscle performance and maximal strength, underscoring the physiological link between muscle strength and cardiac remodeling in response to resistance training. Our study advances the understanding of chronic physiological cardiac responses to resistance training and contributes to the ongoing debate on ventricular hypertrophy patterns in strength- versus endurance-trained male athletes. The mechanisms underlying the development of cardiovascular risk factors and maladaptive cardiac phenotypes observed in some strength athletes remain poorly understood. Future studies focusing on athletes engaged in purely isometric disciplines, considering sex and age differences, could help isolate the factors and pathways contributing to maladaptive remodeling, thereby improving our understanding of the balance between physiological and pathological adaptations in strength athletes.

Abbreviations

2D	Two-dimensional
3D	Three-dimensional
A_m	Peak late transmitral flow Doppler velocity
A_t	Peak late transtricuspid flow Doppler velocity
ANOVA	Analysis of variance
ECG	Electrocardiogram
e_m'	Peak early diastolic mitral annulus velocity
e_t'	Peak early diastolic tricuspid lateral annulus velocity
E_m	Peak early transmitral flow Doppler velocity
E_t	Peak early transtricuspid flow Doppler velocity
EF	Ejection fraction
GLS	Global longitudinal strain
LA	Left atrium
LV	Left ventricle
TTE	Transthoracic echocardiography
RA	Right atrium
Rep	Repetition
RM	Repetition maximum
RV	Right ventricle
RV S′	Peak right ventricle lateral systolic myocardial velocity
RWT	Relative wall thickness
TAPSE	Tricuspid annular plane systolic excursion
TDI	Tissue Doppler imaging
$\dot{V}O_{2peak}$	Maximal oxygen uptake
VT	Ventilatory threshold

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Author contributions

JD, ER, AG, and AH contributed to conceptualization. NP, JD, AR, FT, ER, AG, and AH were involved in the methodology. NP, JD, HA, MR, KL, ES, ER, AG, and AH were involved in the investigation. AR and AH performed data curation. NP and AH were involved in writing and original draft preparation. JD, ER, AG, and AH contributed to writing, reviewing, and editing. AG and AH were involved in supervision. AH contributed to project administration and funding acquisition. All the authors approved the final version of the manuscript.

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Data availability

The datasets generated during and analyzed during the current study are available from the corresponding author upon reasonable request.

Declarations

Ethics approval and consent to participate

All procedures achieved in this study were performed in accordance with the ethical standards as laid down in the 1964 Declaration of Helsinki and its later amendments or comparable ethical standards. The study was approved by the French research ethics committee (EUDRACT: 2019-A01235-52). Informed consent was obtained from all participants involved in the study. All the collected data were anonymous. A compensatory allowance of €150 was provided for all participants, subject to their participation in the entirety of the study.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

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References

1. Paluch AE, Boyer WR, Franklin BA, Laddu D, Lobelo F, Lee DC, et al. Resistance exercise training in individuals with and without cardiovascular disease: 2023 update: A scientific statement from the American heart association. Circulation. 2024;149(3):e217–31.
2. La Gerche A, Wasfy MM, Brosnan MJ, Claessen G, Fatkin D, Heidbuchel H, et al. The athlete's Heart-Challenges and controversies: JACC focus seminar 4/4. J Am Coll Cardiol. 2022;80(14):1346–62.
3. Morganroth J, Maron BJ, Henry WL, Epstein SE. Comparative left ventricular dimensions in trained athletes. Ann Intern Med. 1975;82(4):521.
4. Morganroth J, Maron BJ. The athlete's heart syndrome: a new perspective. Ann N Y Acad Sci. 1977;301:931-41.
5. Baggish AL, Wang F, Weiner RB, Elinoff JM, Tournoux F, Boland A, et al. Training-specific changes in cardiac structure and function: a prospective

and longitudinal assessment of competitive athletes. J Appl Physiol (1985). 2008;104(4):1121-8.
6. Agrotou S, Karatzi K, Papamichael C, Fatouros I, Mitrakou A, Zakopoulos N, et al. Effects of chronic anaerobic training on markers of sub-clinical atherosclerosis. Hellenic J Cardiol. 2013;54(3):178–85.
7. Tso JV, Turner CG, Liu C, Prabakaran G, Jackson M, Galante A, et al. Longitudinal aortic root dilatation in collegiate American-Style football athletes. J Am Heart Assoc. 2023;12(12):e030314.
8. Haykowsky MJ, Dressendorfer R, Taylor D, Mandic S, Humen D. Resistance training and cardiac hypertrophy: unravelling the training effect. Sports Med. 2002;32(13):837-49.
9. Haykowsky M, Taylor D, Teo K, Quinney A, Humen D. Left ventricular wall stress during leg-press exercise performed with a brief Valsalva maneuver. Chest. 2001;119(1):150–4.
10. Grandperrin A, Schuster I, Moronval P, Izem O, Rupp T, Obert P, et al. Anabolic steroids use is associated with impairments in atrial and ventricular cardiac structure and performance in athletes. Med Sci Sports Exerc. 2022;54(5):780-8.
11. Baggish AL, Weiner RB, Kanayama G, Hudson JI, Lu MT, Hoffmann U, et al. Cardiovascular toxicity of illicit anabolic-androgenic steroid use. Circulation. 2017;135(21):1991-2002.
12. Berge HM, Isern CB, Berge E. Blood pressure and hypertension in athletes: a systematic review. Br J Sports Med. 2015;49(11):716–23.
13. Otsuki T, Maeda S, Iemitsu M, Saito Y, Tanimura Y, Ajisaka R, et al. Relationship between arterial stiffness and athletic training programs in young adult men. Am J Hypertens. 2007;20(9):967–73.
14. Pluim BM, Zwinderman AH, van der Laarse A, van der Wall EE. The athlete's heart. A meta-analysis of cardiac structure and function. Circulation. 2000;101(3):336–44.
15. Utomi V, Oxborough D, Ashley E, Lord R, Fletcher S, Stembridge M, et al. The impact of chronic endurance and resistance training upon the right ventricular phenotype in male athletes. Eur J Appl Physiol. 2015;115(8):1673–82.
16. Haykowsky MJ, Teo KK, Quinney AH, Humen DP, Taylor DA. Effects of long term resistance training on left ventricular morphology. Can J Cardiol. 2000;16(1):35–8.
17. Spence AL, Naylor LH, Carter HH, Buck CL, Dembo L, Murray CP, et al. A prospective randomised longitudinal MRI study of left ventricular adaptation to endurance and resistance exercise training in humans. J Physiol. 2011;589(Pt 22):5443–52.
18. Spence AL, Carter HH, Murray CP, Oxborough D, Naylor LH, George KP, et al. Magnetic resonance imaging-derived right ventricular adaptations to endurance versus resistance training. Med Sci Sports Exerc. 2013;45(3):534–41.
19. Scharf M, Oezdemir D, Schmid A, Kemmler W, von Stengel S, May MS, et al. Myocardial adaption to HI(R)T in previously untrained men with a randomized, longitudinal cardiac MR imaging study (Physical adaptions in untrained on strength and heart trial, PUSH-trial). PLoS ONE. 2017;12(12):e0189204.
20. American College of Sports M. American college of sports medicine position stand. Progression models in resistance training for healthy adults. Med Sci Sports Exerc. 2009;41(3):687–708.
21. Schoenfeld BJ, Grgic J, Ogborn D, Krieger JW. Strength and hypertrophy adaptations between low- vs. high-load resistance training: a systematic review and meta-analysis. 2017. pp. 3508-23.
22. Schoenfeld BJ, Grgic J, Van Every DW, Plotkin DL. Loading recommendations for muscle strength, hypertrophy, and local endurance: A Re-Examination of the repetition continuum. Sports (Basel). 2021;9(2).
23. Pamart N, Drigny J, Azambourg H, Remilly M, Macquart M, Lefevre A, et al. Effects of a 20-Week High-Intensity strength training program on muscle strength gain and cardiac adaptation in untrained men: preliminary results of a prospective longitudinal study. JMIR Form Res. 2023;7:e47876.
24. Sharma S, Drezner JA, Baggish A, Papadakis M, Wilson MG, Prutkin JM, et al. International recommendations for electrocardiographic interpretation in athletes. Eur Heart J. 2018;39(16):1466–80.
25. Casale PN, Devereux RB, Alonso DR, Campo E, Kligfield P. Improved sex-specific criteria of left ventricular hypertrophy for clinical and computer interpretation of electrocardiograms: validation with autopsy findings. Circulation. 1987;75(3):565–72.
26. Lang RM, Badano LP, Mor-Avi V, Afilalo J, Armstrong A, Ernande L, et al. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American society of echocardiography and the European association of cardiovascular imaging. J Am Soc Echocardiogr. 2015;28(1):1–e3914.
27. Grgic J, Lazinica B, Schoenfeld BJ, Pedisic Z. Test-retest reliability of the one-repetition maximum (1RM) strength assessment: a systematic review. Sports Med Open. 2020;6(1):31.
28. Binder RK, Wonisch M, Corra U, Cohen-Solal A, Vanhees L, Saner H, et al. Methodological approach to the first and second lactate threshold in incremental cardiopulmonary exercise testing. Eur J Cardiovasc Prev Rehabil. 2008;15(6):726–34.
29. American Thoracic S. American college of chest P. ATS/ACCP statement on cardiopulmonary exercise testing. Am J Respir Crit Care Med. 2003;167(2):211–77.
30. Liguori G. ACSM's Guidelines for Exercise Testing and Prescription, 11th Edition. 2021.
31. Morishita S, Tsubaki A, Takabayashi T, Fu JB. Relationship between the rating of perceived exertion scale and the load intensity of resistance training. Strength Cond J. 2018;40(2):94–109.
32. Thomas R, Baechle RWE. Essentials of strength training and conditioning. (U.S.) NSCA. editor: Human Kinetics; 2008.
33. Mancia G, Kreutz R, Brunstrom M, Burnier M, Grassi G, Januszewicz A, et al. 2023 ESH guidelines for the management of arterial hypertension the task force for the management of arterial hypertension of the European society of hypertension: endorsed by the international society of hypertension (ISH) and the European renal association (ERA). J Hypertens. 2023;41(12):1874–2071.
34. Bonekamp NE, May AM, Halle M, Dorresteijn JAN, van der Meer MG, Ruigrok YM, et al. Physical exercise volume, type, and intensity and risk of all-cause mortality and cardiovascular events in patients with cardiovascular disease: a mediation analysis. Eur Heart J Open. 2023;3(3):oead057.
35. Edwards JJ, Deenmamode AHP, Griffiths M, Arnold O, Cooper NJ, Wiles JD, et al. Exercise training and resting blood pressure: a large-scale pairwise and network meta-analysis of randomised controlled trials. Br J Sports Med. 2023;57(20):1317-26.
36. Fecchio RY, Brito LC, Pecanha T, de Moraes Forjaz CL. Potential mechanisms behind the blood Pressure-Lowering effect of dynamic resistance training. Curr Hypertens Rep. 2021;23(6):35.
37. La Gerche A, Heidbuchel H. Can intensive exercise harm the heart? You can get too much of a good thing. Circulation. 2014;130(12):992–1002.
38. Guo J, Zhang X, Wang L, Guo Y, Xie M. Prevalence of metabolic syndrome and its components among Chinese professional athletes of strength sports with different body weight categories. PLoS ONE. 2013;8(11):e79758.
39. Kanakis C, Hickson RC. Left ventricular responses to a program of lower-limb strength training. Chest. 1980;78(4):618-21.
40. Au JS, Oikawa SY, Morton RW, Phillips SM, MacDonald MJ, Stohr EJ. Unaltered left ventricular mechanics and remodelling after 12 weeks of resistance exercise training - a longitudinal study in men. Appl Physiol Nutr Metab. 2019;44(8):820–6.
41. Pelliccia A, Maron BJ, Spataro A, Proschan MA, Spirito P. The upper limit of physiologic cardiac hypertrophy in highly trained elite athletes. N Engl J Med. 1991;324(5):295–301.
42. Haykowsky MJ, Quinney HA, Gillis R, Thompson CR. Left ventricular morphology in junior and master resistance trained athletes. Med Sci Sports Exerc. 2000;32(2):349–52.
43. Miranda DPAW, Lopes HHMC, deSantana VJ, Bocchi EA, Salemi VMC. Association between right heart dimensions and muscle performance and cardiorespiratory capacity in strength and endurance athletes. Transl Sports Med. 2021;4:470–80.
44. Feiereisen P, Vaillant M, Eischen D, Delagardelle C. Isokinetic versus one-repetition maximum strength assessment in chronic heart failure. Med Sci Sports Exerc. 2010;42(12):2156-63.
45. Sloan RP, Shapiro PA, DeMeersman RE, Bagiella E, Brondolo EN, McKinley PS, et al. The effect of aerobic training and cardiac autonomic regulation in young adults. Am J Public Health. 2009;99(5):921–8.
46. Milot E, Rehel S, Langeard A, Bigot L, Pasquier F, Matveeff L, et al. Effectiveness of multi-modal home-based videoconference interventions on sleep in older adults: study protocol for a randomized controlled trial. Front Public Health. 2024;12:1326412.
47. Langeard A, Bigot L, Loggia G, Bherer L, Chastan N, Gauthier A. Ankle dorsiflexors and plantarflexors neuromuscular electrical stimulation training impacts gait kinematics in older adults: A pilot study. Gait Posture. 2021;84:335–9.
48. Knowles OE, Drinkwater EJ, Urwin CS, Lamon S, Aisbett B. Inadequate sleep and muscle strength: implications for resistance training. J Sci Med Sport. 2018;21(9):959-68.

49. Lasocka-Koriat Z, Lewicka-Potocka Z, Kaleta-Duss A, Siekierzycka A, Kalinowski L, Lewicka E, et al. Differences in cardiac adaptation to exercise in male and female athletes assessed by noninvasive techniques: a state-of-the-art review. Am J Physiol Heart Circ Physiol. 2024;326(5):H1065–79.

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