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FULL RESEARCH PAPER
Exercise physiology

Effects of endurance exercise training on left ventricular structure in healthy adults: a systematic review and meta-analysis

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Aims
To determine the impact of endurance training (ET) interventions on left ventricular (LV) chamber size, wall thickness, and mass in healthy adults.

Methods and results
Electronic databases including CINAHL, MEDLINE, PsycINFO, SPORTDiscus, Cochrane library, and EBM Reviews were searched up to 4 January 2022. Criteria for inclusion were healthy females and/or males (>18 years), ET intervention for ≥2 weeks, and studies reporting pre- and post-training LV structural parameters. A random-effects meta-analysis with heterogeneity, publication bias, and sensitivity analysis was used to determine the effects of ET on LV mass (LVM) and diastolic measures of interventricular septum thickness (IVSd), posterior wall thickness (PWTd), and LV diameter (LVDd). Meta-regression was performed on mediating factors (age, sex, training protocols) to assess their effects on LV structure. Eighty-two studies met inclusion criteria ($n = 1908$; 19–82 years, 33% female). There was a significant increase in LVM, PWTd, IVSd, and LVDd following ET [standardized mean difference (SMD) = 0.444, 95% confidence interval (CI): 0.361, 0.527; $P < 0.001$; SMD = 0.234, 95% CI: 0.159, 0.309; $P < 0.001$; SMD = 0.237, 95% CI: 0.159, 0.316; $P < 0.001$; SMD = 0.249, 95% CI:0.173, 0.324; $P < 0.001$, respectively]. Trained status, training type, and age were the only mediating factors for change in LVM, where previously trained, mixed-type training, young (18–35 years), and middle-aged (36–55 years) individuals had the greatest change compared with untrained, interval-type training, and older individuals (>55 years). A significant increase in wall thickness was observed in males, with a similar augmentation of LVDd in males and females. Trained individuals elicited an increase in all LV structures and ET involving mixed-type training and rowing and swimming modalities conferred the greatest increase in PWTd and LVDd.

Conclusion
Left ventricular structure is significantly increased following ET. Males, young and trained individuals, and ET interventions involving mixed training regimes elicit the greatest changes in LV structure.

Lay summary
Heart structure significantly increases the following endurance training (ET) ≥2 weeks.

- Changes in heart structure were most prominent in males, who are young (18–35 years), already trained, and following concurrent continuous and interval training.
- Changes in heart size were not shown in older individuals (>55 years) compared with young and middle-aged individuals.
- While both males and females similarly increase their cavity size and heart mass, sex differences were revealed for wall thickness where significant increases were seen in males but not females.

Keywords Endurance exercise training • Left ventricle • Athletes • Sex differences

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Introduction

Participation in regular endurance training (ET) is associated with structural and functional cardiac adaptations that, in turn, improve cardiorespiratory fitness needed for higher peak oxygen uptake required for enhancing athletic performance.[1,2] These structural adaptations include increases in left ventricular (LV) chamber size, wall thickness, and mass (LVM) and require careful consideration when differentiating between normal physiological adaptation and potential pathology.[1] Previous cross-sectional literature has demonstrated that endurance-trained individuals have an increased LVM compared with age- and sex-matched controls,[3–8] but that these physiological cardiac adaptations can be mediated by gender, training volume, and sport type.[5,6,8,9] Evidence suggests that male athletes,[5,8] those with greater training volume,[5,8] and those that participate in high dynamic–low static sports and high dynamic–high static sports have the largest LVM.[6,10] Cross-sectional data are, however, limited by selection bias and cannot determine the cause-and-effect relationship between ET and cardiac phenotype. This requires intervention-based, longitudinal training studies. There is a growing evidence-base available suggesting that longitudinal training studies can lead to cardiac adaptation, at least qualitatively similar to that observed in cross-sectional, athlete-control studies. For example, a 1-year intensive ET programme in sedentary individuals resulted in a significant increase in LVM, approaching similar levels to that seen in elite endurance-trained athletes.[3]

Although research has demonstrated increases in LV chamber size, wall thickness, and mass in response to ET, these changes have been highly variable, study-to-study, and there has been limited systematic evaluation of potential factors that may mediate the cardiac structure ET response. Confounding factors such as age, sex, ET history (untrained or previously trained), modality of ET (i.e. running, cycling, rowing, swimming, or combination of more than one modality), or type of training programme (continuous, interval, or mixture of both types) require further study and the complexity and breadth of potential confounders lends itself to a systematic review and meta-analysis. Therefore, the aim of the current systematic review was to determine the impact of prospective ET interventions on LV structure in healthy adults. The secondary purpose was to assess how variation in potential confounders may mediate change in LVM. We hypothesized that LV structure would be significantly greater following ET, with males, younger, trained individuals, and ET interventions involving high dynamic–high static modalities eliciting greater changes in LVM and other structural variables.

Methods

The review is reported in accordance with the PERSiST (implementing PRISMA in Exercise, Rehabilitation, Sport medicine and SporTs science) consensus statement.[11] This review is registered with Prospero (CRD42017072090).

Eligibility criteria

Studies included had to meet the following inclusion criteria: (i) ET interventions in healthy men and women ≥18 years; (ii) LV structural parameters reported prior to and after an ET intervention; and (iii) ≥2 weeks in duration. Cross-sectional studies comparing training methods across sporting disciplines and observational longitudinal studies, including multi-day races, were excluded, as were conference abstracts, editorial, and review papers. Studies that included clinical populations were excluded; however, if there was a healthy control population that underwent ET, the control population results were included. Individually or in any combination, ET that included running, aerobics, cycling, swimming, rowing, soccer, or other ET modality of at least moderate intensity were eligible. Studies that were solely strength-based, or low-intensity ET, were excluded. Interventions that reported an element of strength training within their moderate or higher intensity ET intervention were included. There were no restrictions on the setting in which the ET were performed (i.e. classes, supervised individual, or group training). Furthermore, exercise could not be combined with pharmacological interventions. In the event the same study had multiple publications, the report that included the greatest sample size of the eligible outcome measures was included. In studies that assessed more than two time-points during the intervention, the pre-training and last training block measurements were included in the analysis.[3,12–22] In instances where studies only broadly discussed the training phases (i.e. pre-season training, competitive season, maintenance phases), and did not prospectively prescribe the training programme, they were excluded as observational studies.

Search strategy

The systematic search was conducted by a librarian (E.K.) who had expertise in systematic reviews and verified by a senior librarian (D.D.). The databases searched included CINAHL, MEDLINE, PsycINFO, SPORTDiscus, Cochrane (Database of Systematic Reviews, Clinical Answers, Central Register of Controlled Trials, Methodology Register), and EBM Reviews (Health Technology Assessment, NHS Economic Evaluation Database, ACP Journal Club, Database of Abstracts of Reviews of Effects). These databases were last searched on 4 January 2022 and yielded 3481 results. The search strategy combined the two concepts of intervention and outcome, investigating the impact of ET (intervention) on LV structure (outcome). Keyword searches, with the phrase and truncation indications, were performed in the Title and Abstract fields. Subject headings were employed where available. See Supplementary material online, *Table S1* for the full list of keywords and subject headings used in each database, according to their interface (EBSCO or Ovid). The English-language filter was the only limiter applied to the search results; results were not limited according to date or publication type.

Selection process

A total of 3481 articles were identified through database searching with an additional 13 articles identified through reference lists and hand searching. The articles were imported into a reference management software (Endnote X9) where duplicates were removed. The remaining 1631 results were imported into a systematic review management software (COVIDENCE, Melbourne, Australia) for screening so that investigators could work independently. Two reviewers (N.S., A.R.) reviewed all study titles and abstracts and excluded studies according to the pre-specified criteria with a unanimous result required to exclude a study (*Figure 1*). Two investigators (A.T.C., B.N.M.) reviewed the full-text articles and assessed them according to the search criteria for inclusion. Disagreement in any of the stages was resolved by consensus between the two reviewers and a third reviewer (K.G.) if consensus could not be reached.

Data extraction

One investigator (B.N.M.) extracted data using a standardized data extraction form agreed upon by three reviewers (A.T.C., K.G., B.N.M.). A second investigator (L.R.) reviewed the data for accuracy, and any inconsistencies were resolved by discussion between these investigators, and a third investigator (A.T.C.) solved any disagreements. General study information (authors, publication year, sample size), participant information (age, sex), imaging modality (echocardiography, echo; cardiac magnetic resonance imaging, CMR), LV structural parameters (interventricular septum thickness in end-diastole, IVSd; posterior wall thickness in end-diastole, PWTd; LV end-diastolic diameter, LVDd), and LV functional parameters [(VO_{2max}, end-diastolic volume, EDV; end-systolic volume, ESV; and stroke volume, SV), ET intervention features: trained status, modality (running/aerobics, cycling, swimming, rowing, soccer, or any combination of these), type of training, inclusion of strength training, duration of the study (weeks), frequency of training (days/week), time per session (h); total weekly hours] were extracted from included studies. Participants' trained status were classified as *untrained* (individuals who had not previously exercised regularly, on an organized basis or competed in previous competitions) and *trained* (individuals who followed a regular training programme or previously competed, including those who were at the beginning an ET programme (i.e. college matriculation, training for a marathon) and athletes who went through a period of detraining prior to the ET intervention). The type of training included: *continuous* (*CONT*) training defined as ET that performed with a

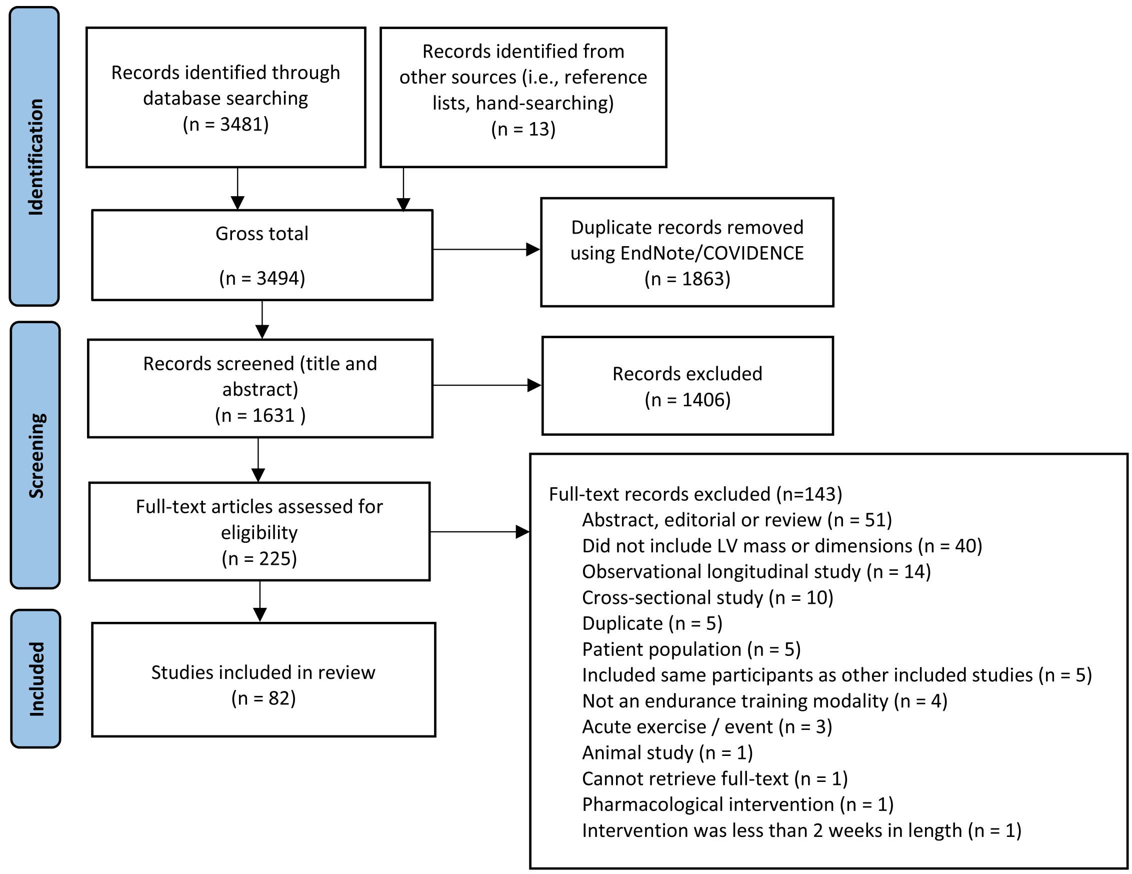

Figure 1 PRISMA flow diagram of the systematic process in article selection.

continuous intensity throughout that did not involve rest periods, *interval (INT)* training that included alternating bouts of high intensity with rest periods, and *mixed (MIX)* training that included a combination of these, either within the same session or during separate training sessions. Studies assessing more than one independent group were extracted as individual interventions for the analyses. Where full-text articles could not be found, or if data were unavailable from tables or the results section, the authors of the study in question were contacted by email. If the authors could not be contacted, the study was excluded. In instances where the training details were not reported, these studies were left out of the respective sub-analysis.

Data synthesis and analysis

Meta-analysis

The raw data extracted from the studies were transformed into effect sizes and were calculated as standardized mean differences (SMD) between pre- and post-training values using sample size and paired *P*-values with a meta-analysis software, using a random-effects model (Comprehensive Meta-Analysis V3). This model was selected due to different imaging techniques used to assess functional and structural variables as well as differences in the estimation equations to derive LVM across the studies. Absolute values for all variables of interest were the preferred value for the meta-analysis. Indexed values were used if absolute values or body surface area (BSA, pre- and post-measurement) were not reported. Since the model calculates the difference between pre- and post-values, the inclusion of the indexed values in this manner was justified. In cases where exact *P*-values were not reported, significant levels of $P < 0.05$, $P < 0.01$, and $P < 0.001$ were reported as 0.05, 0.01, and 0.001, respectively, and when reported as non-significant or $P > 0.05$, the values were reported as 0.999. An overall meta-analysis was conducted for each of the LV structural outcomes (LVM, LVDd, IVSd, PWTd) and additional meta-analyses were performed independently for each moderator variable to investigate their effect on LV structures. The planned moderators to be assessed were sex, age group [young (18–35 years), middle-aged (36–55 years), or older (>55 years)], training status, type of training, and mode of training. A meta-regression was conducted to ascertain if any effect moderator variable influenced the SMD in LV structures.

Heterogeneity among studies was assessed using I^2 statistics (the percentage of total variation between studies due to heterogeneity rather than by chance) and classified as low, moderate, and high at <50, 51–75, and >75%, respectively.[23] Relative influence of each study on the SMD was assessed by omitting one study at a time for sensitivity analysis ('one study removed analysis'). Publication bias was investigated by funnel plots for a visual inspection of asymmetry and statistically assessed using Egger's test and the trim-and-fill method.[24,25] The National Heart, Lung, and Blood Institute (NHLBI) quality assessment tool for pre-post studies with no control group to assess for potential flaws in study methods including sources of bias, sampling, cofounding variables, study power, and other relevant factors.[26] Each study was judged as 'good', 'fair', or 'poor' quality based on ratings from 11 items included in the tool. The 12th item was not included in this assessment as it pertained to group-level interventions (e.g. a whole hospital, a community) which was not relevant in the current review. Globally, a good study had the least risk of bias and was considered valid. A fair study is prone to some bias, but insufficient to invalidate its findings, varying in its strengths and weaknesses. A poor-quality study has a high risk of bias and is considered invalid. If a study was rated 'poor', it was removed from the analysis to determine its influence on the SMD. If the removal of the poor-rated study did not change the overall outcome of the

SMD when the study was removed, the study remained in the analysis. The quality assessment was performed by two reviewers (B.N.M. and R.J.M.), with a third reviewer (A.T.C.) involved in cases of uncertainty.

Descriptive analysis

The pre- and post-values for structural and functional values were reported as estimated pooled means and were calculated using a weighted sample dependent upon the sample size of each study [pooled average = $((n_1 \times \text{mean}_1) + (n_2 \times \text{mean}_2)\ldots(n_k \times \text{mean}_k))$/sum of all samples] and pooled standard deviations were calculated as:

$$s_{\text{pooled}} = \sqrt{\frac{(n_1-1)s_1^2 + (n_2-1)s_2^2 + \cdots + (n_k-1)s_k^2}{n_1 + n_2 + \cdots + n_k - k}}$$

When the SD was not reported, it was estimated from the standard error of the mean, 95% confidence interval (CI), or IQR as suggested in the Cochrane handbook.[27] In situations where provided measures were not able to be converted to SD, they were excluded from the pooled means table and the sample size and number of studies were adapted accordingly. In instances where the relative VO_{2max} (mL/kg/min) was not provided, it was estimated by dividing the group absolute mean VO_2 (L/min) by the group mean body weight (L/min/kg × 1000). In studies where the SV was not reported, it was calculated using the following equation SV = LVEDV —LVESV. Per cent change was reported in consideration of different measurement techniques and modalities and calculated as ((post − pre)/pre) × 100 for all LV structural variables (LVM, IVSd, PWTd, LVDd) and their mediating factors (i.e. sex, age group, training status, type of training, mode of training). Independent samples *t*-test or one-way ANOVA was used to determine differences between groups with the Tukey *post hoc* analysis. The Pearson correlation was used to establish associations between training status and protocols with changes in LVM. Statistical significance was considered statistically significant at a *P*-value of <0.05. Statistical analyses were performed using SPSS software Version 27.0 (IBM Corp., Armonk, NY, USA).

Results

Study selection and characteristics

The PRISMA flow diagram (*Figure 1*) illustrates the process of article selection, removal, and inclusion. Eighty-two studies satisfied the inclusion criteria ($n = 1908$; 19–82 years, 33% female).[3,12–22,28–97] Baseline characteristics for the included studies are reported in *Table 1*. There were 19 studies that included more than one population. Specifically, 8 studies reported more than one type[16,43,44,51,53,62,65] or mode[31] of ET intervention and 11 studies reported more than one population type (i.e. sex,[19,60,63] age,[69] trained status,[14,21,50,54] menopause status,[41] genotype[30]) for a total of 105 analyses. Of these studies, 15 were randomized to either the ET or control group.[28,31,36,37,48,51,53,63,66,67,76,77,89,96] Left ventricular structural characteristics were assessed before and after ET using echo or CMR. *Table 2* summarizes the ET interventions. The average duration (range) of the ET interventions was 16.3 weeks (2–52 weeks), 4 sessions per week (1–8), and 4.8 h per week (0.2–32.5), with the majority of ET sessions between 30 and 60 min in duration. There were 11 studies that reported an element of strength training within their ET intervention.[18,32,39,57,61,72,73,75,78,91,93] Trained individuals engaged in a significantly greater number of hours per week compared with untrained individuals (14.3 ± 8.8 vs. 3.1 ± 2.2; $P < 0.001$, respectively), with similar length of ET programs (18.9 ± 11.9 vs. 15.9 ± 12.3 weeks; $P = 0.378$). *Table 3* summarizes pooled means and % change for before and after ET intervention for all structural and functional parameters in untrained and trained individuals. *Figure 2A–E* illustrates the per cent change for all LV structural variables and their mediating factors.

Overall ET had a high tolerability, with three participants reporting the training to be burdensome and 19 reporting musculoskeletal injuries out of 10 studies. No studies reported major adverse outcomes; however, there were four cardiac abnormalities identified (i.e. three dysrhythmias and one left bundle branch block) in two studies. There were 127 (7%) dropouts amongst all studies, although 48 studies did not report adherence.

Effect of endurance training on left ventricular structure

Meta-analysis

The results of all meta-analyses including the overall SMD for LV structures and their independent moderators, heterogeneity, and publication bias are reported in Supplementary material online, *S2–S5*. The meta-analyses revealed a significant increase in all LV structures (LVM: SMD = 0.444; $P < 0.001$, IVSd: SMD = 0.237; $P < 0.001$, PWTd: SMD = 0.234; $P < 0.001$, LVDd: SMD = 0.249; $P < 0.001$) (see Supplementary material online, *S9–S11*), with the greatest increase in LVM (*Figure 3*). Left ventricular mass and LVDd increased in both males and females (LVM: SMD = 0.498; $P < 0.001$ and SMD = 0.280; $P = 0.004$; LVDd: SMD = 0.216; $P < 0.001$ and SMD = 0.245; $P = 0.003$, respectively), whereas PWTd and IVSd only increased in males. Egger's test revealed publication bias for LVM ($P = 0.001$) and LVDd ($P = 0.013$). Using the Trim and Fill method in those with publication bias, the SMD decreased slightly for LVM (0.368, 95% CI: 0.279, 0.458) and LVDd (0.188, 95% CI: 0.107, 0.269). The heterogeneity was low to moderate in all studies. None of the studies had high (>75%) heterogeneity. Each of the included studies were of good ($n = 33$), fair ($n = 47$), and poor ($n = 2$) quality as indicated by the NHLBI quality assessment and agreed upon by both reviewers. The removal of the two studies with 'poor' quality did not elicit a significant change in the SMD for LV structural parameters; therefore, these studies remained in the analyses. Sensitivity analyses using the one study removed protocol revealed that the omission of any single study showed no change in the overall statistical significance, indicating that the presented results are statistically robust.

Moderator analysis

Several moderator variables influenced the SMD of the LV structures. The meta-regression bubble plots are reported in Supplementary material online S6-S8. *LVM:* There was a significant association between age group and LVM ($P = 0.0408$), with young and middle-aged individuals ($B = 0.2836$, $P = 0.0185$; $B = 0.3444$, $P = 0.0256$, respectively) conferring the greatest change in LVM compared with older individuals. Trained individuals conferred a greater change in LVM ($B = 0.2778$, $P = 0.0110$) than untrained and mixed training ($B = 0.2976$, $P = 0.0335$) elicited the greatest increase in LVM compared with continuous- and interval-type raining. *IVSd:* Sex influenced the SMD of IVSd, with males demonstrating the greatest change in IVSd compared with females ($B = 0.2981$, $P = 0.0053$). Training status also demonstrated a significant association, with those that were trained ($B = 0.2362$, $P = 0.0261$) conferring a greater change than untrained individuals. *PWTd:* There was a significant association between sex and SMD of PWTd, with males conferring the greatest change in PWTd ($B = 0.1954$, $P = 0.0443$). There was also a significant association between trained status ($P = 0.0010$), training type ($P = 0.0492$), and mode of exercise ($P = 0.0046$) on SMD of PWTd. Those that were trained ($B = 0.3162$, $P = 0.0010$) had a greater change compared with those that were untrained, mixed, and continuous training regimes elicited the greatest change ($B = 0.2650$, $P = 0.0263$; $B = 0.2404$, $P = 0.0215$, respectively) compared with interval training, and swimming, rowing, and running ($B = 1.4014$, $P = 0.0093$; $B = 0.3084$, $P = 0.0340$; $B = 0.2831$, $P = 0.0022$, respectively) conferred the greatest change compared with cycling. *LVDd:* Those that were trained ($B = 0.2344$, $P = 0.0328$) conferred a greater increase compared with the untrained. Of the training types, only those that

Table 1 Baseline characteristics of journal articles meeting eligibility criteria

Study (author, year)	n	Control (n)	Female (%)	Age [mean ± SD or (range)]	Population	VO_{2max} [mL/min/kg) (mean ± SD or (range)]	Imaging modality (dimensions included)
Untrained							
Adams_1981[28]	22	9[a]	0	22 (18–25)	College students (no endurance training > 3 months in last 5 years)	48.6 ± 6.0	Echo (IVSd, PWTd, LVDd)
Aksakai_2013[29]	34	n/a	0	22 ± 2	No history of prior exercise exposure	DNR	Echo (LVM, IVSd, PWTd, LVDd)
Alves_2009_1, 2, 3, 4[30]	83	n/a	0	27 ± 1	Brazilian policeman	47–50	Echo (LVM, IVSd, PWTd, LVDd)
Andersen_2010_A[31]	18	10[a]	100	37 ± 8[b]	Sedentary (no physical training for at least 2 years)	35.5 ± 5.9	Echo (IVSd, PWTd, LVDd)
Andersen 2010_B[4]	19	10[a]	100	37 ± 8[b]	Sedentary (no physical training for at least 2 years)	32.5 ± 4.6	Echo (IVSd, PWTd, LVDd)
Arbab-Zadeh_2014[3]	12	n/a	42	29 ± 6	Untrained (< 30 min/day, < 3×/week regularly using either dynamic or static exercise)	40.3 ± 5.5	CMR (LVM)
Bates_2013[c33]	10	n/a	40	39 ± 12	Untrained	27.6 ± 6.3	CMR (LVM)
Boone_2014[35]	9	n/a	0	27 ± 3	Moderately active (< 3×/week of any training)	53	Echo (LVM, IVSd, LVDd)
Camargo_2008[36]	6	7[a]	0	29 ± 4	Healthy (no regular physical training in previous year, 35–42 VO_{2peak} (mL/min/kg)	38.1 ± 2	CMR (LVM)
Cornelissen_2011[37]	16	n/a	44	59 (55–71)[b]	Sedentary	21.8 ± 0.8	CMR (LVM)
Cox_1986[38]	11	5[a]	55	23 ± 1	Sedentary, inactive (no formal training for at least 3 months prior)	40.8 ± 2.1	Echo (LVM, IVSd, PWTd, LVDd)
Dart_1992[12]	10	n/a	50	20–30	Sedentary	38	Echo (LVM, IVSd, PWTd, LVDd)
DeMaria_1978[40]	24	n/a	46	26	Sacramento Police Academy	35.5 ± 1.7	Echo (LVM, IVSd, PWTd, LVDd)
Egelund_2017_1[41]	36	n/a	100	49 ± 2	Late pre-menopausal, sedentary (<2 h of physical training/week during previous 2 years, < 40 VO_2 (mL/min/kg)	30.1 ± 4.4	Echo (LVM, IVSd, PWTd, LVDd)
Egelund_2017_2[41]	37	n/a	100	53 ± 3	Early post-menopausal, sedentary (<2 h of physical training/week during previous 2 years, < 40 VO_2 (mLl/min/kg)	30.4 ± 3.3	Echo (LVM, IVSd, PWTd, LVDd)
Ehsani_1991[13]	10	n/a	0	64 ± 3	Sedentary	29.6 ± 4.1	Echo (LVM, PWTd, LVDd)
Esfandiari_2014_A[43]	8	n/a	0	25 ± 3	Untrained (<2 h of <6 METs)	39.5 ± 7.1	Echo (LVM, IVSd, PWTd, LVDd)
Esfandiari_2014_B[43]	8	n/a	0	26 ± 5	Untrained (<2 h of <6 METs)	39.9 ± 5.9	Echo (LVM, IVSd, PWTd, LVDd)
Eskelinen_2016_A[44]	14	n/a	0	47 ± 9	Untrained (< 2×/week, no active training background, < 40 VO_2 (mL/min/kg)	34.7 ± 4.1	CMR (LVM)
Eskelinen_2016_B[44]	14	n/a	0	48 ± 10	Untrained (< 2×/week, no active training background, < 40 VO_2 (mL•/min•/kg)	33.7 ± 3.9	CMR (LVM)
Fujimoto_2010[47]	9	n/a	33	71 ± 3	Sedentary (≤ 30 min, 3×/week)	22.8 ± 3.4	CMR (LVM)
Fujimoto_2013[46]	15	13[a]	71	67 ± 6	Sedentary (≤ 30 min, 3×/week)	23.0 ± 4.7	CMR (LVM)
Grace_2018_1[14]	22	n/a	0	62 ± 5	Sedentary (no participation in formal exercise training)	28.3	Echo (LVM, IVSd, PWTd, LVDd)

Continued

Table 1 ***Continued***

Study (author, year)	*n*	Control (*n*)	Female (%)	Age [mean ± SD or (range)]	Population	VO_{2max} [mL/min/kg) (mean ± SD or (range)]	Imaging modality (dimensions included)
Haykowsky_2005[48]	8	8[a]	100	66 ± 3	No regular participation in aerobic or strength training	22	Echo (LVM, IVSd, PWTd, LVDd)
Hedman_2017[49]	21	21[a]	100	34 ± 7	No regular participation in aerobic training within last year	DNR	Echo (LVM, IVSd, PWTd, LVDd)
Hickson_1982_1, 2[50]	15	n/a	40	29 ± 5	Moderately active in recreational sports, but no training in last 6 months	39.6–45.2	Echo (LVM, IVSd, PWTd, LVDd)
Holloway_2018[15]	12	9[a]	0	21 ± 2	Healthy	42.5	Echo (IVSd, PWTd)
Huang_2019_1[51]	18	9[a]	0	22 ± 1	Sedentary (exercise <1/week, < 20 min)	20.4 ± 1.4	Echo (LVM, IVSd, LVDd)
Huang_2019_2[51]	18	9[a]	0	22 ± 0	Sedentary (exercise <1/week, < 20 min)	21.0 ± 1.0	Echo (LVM, IVSd, LVDd)
Hulke_2012_1[52]	42	n/a	100	20 ± 2	Healthy (DNR physical activity criteria)	37.4 ± 3.3	Echo (LVM, IVSd, PWTd, LVDd)
Hulke_2012_2[52]	43	n/a	0	20 ± 1	Healthy (DNR physical activity criteria)	45.1 ± 5.9	Echo (LVM, IVSd, PWTd, LVDd)
Hulke_2012_A[16]	14	12	DNR	20 ± 1	Healthy (DNR physical activity criteria)	34.1 ± 6.1	Echo (LVM, IVSd, PWTd, LVDd)
Hulke _2012_B[16]	13	12	DNR	20 ± 1	Healthy (DNR physical activity criteria)	33.3 ± 8.5	Echo (LVM, IVSd, PWTd, LVDd)
Hwang_2016_1[53]	15	14[a]	67	65 ± 1	Sedentary	23.1 ± 0.7	Echo (LVM, IVSd, PWTd, LVDd)
Hwang_2016_2[53]	14	14[a]	50	66 ± 2	Sedentary	25.9 ± 1.9	Echo (LVM, IVSd, PWTd, LVDd)
Kanakis_1982_1,2[54]	12	n/a	58	23 (19–32)	Moderately active in recreational sports	35.9–42.5	Echo (IVSd, PWTd, LVDd)
Kiflom_2022[55]	20	n/a	0	19–23	No regular participation in exercise training	n/a	Echo (IVSd, PWTd, LVDd)
Kivisto_2006[56]	14	n/a	71	43 (23–58)	Sedentary	n/a	CMR (LVM)
Krzeminski_1989[17]	18	n/a	0	21 ± 8	No active participation in sports	46.3 ± 4.3	Echo (PWTd, LVDd)
Landry_1985[59]	20	n/a	60	25 ± 4	Sedentary (never trained)	37	Echo (LVM, IVSd, PWTd, LVDd)
Lane_2014_1[60]	28	n/a	0	24 ± 1	Sedentary (<30 min, < 1/week)	38	Echo (LVM)
Lane_2014_2[59]	25	n/a	100	24 ± 1	Sedentary (<30 min, < 1/week)	30	Echo (LVM)
Lusiani_1986[61]	13	9[a]	0	20 ± 7	Prior to beginning training program	DNR	Echo (LVM, IVSd, PWTd, LVDd)
Mahdiabadi_2013_A[62]	10	n/a	0	21 ± 2	Non-athletic	DNR	Echo (IVSd, PWTd, LVDd)
Mahdiabadi_2013_B[62]	10	n/a	0	21 ± 1	Non-athletic	DNR	Echo (IVSd, PWTd, LVDd)
Marsh_1983[64]	12	12	100	28 ± 4	Running on unorganized basis <6 months – 4 years	46.4 ± 3.6	Echo (LVM, IVSd, PWTd, LVDd)
Marsh_2021_1[63]	46	COC	100	24 ± 5	Untrained (< 150 min/week of organized exercise)	DNR	CMR (LVM)
Marsh_2021_2[63]	26	COC	0	28 ± 6	Untrained (< 150 min/week of organized exercise)	DNR	CMR (LVM)
Masoomeh_2012[97]	10	9	100	25 ± 4	Non-athletic (no previous regular exercise training)	DNR	Echo (LVM, IVSd, PWTd, LVDd)
Matsuo_2014_A[65]	14	n/a	0	26 ± 7	Sedentary (no participation in regular activities for 1 year)	43.9 ± 6.7	CMR (LVM)

Continued

Table 1 Continued

Study (author, year)	n	Control (n)	Female (%)	Age [mean ± SD or (range)]	Population	VO_{2max} [mL/min/kg) (mean ± SD or (range)]	Imaging modality (dimensions included)
Matsuo_2014_B[65]	14	n/a	0	27 ± 6	Sedentary (no participation in regular activities for 1 year)	41.9 ± 5.6	CMR (LVM)
Matsuo_2014_C[65]	14	n/a	0	26 ± 6	Sedentary (no participation in regular activities for 1 year)	42.0 ± 6.8	CMR (LVM)
Morrison_1986[66]	17	8	100	52 ± 4	Untrained	27.3 ± 4.6	Echo (IVSd, PWTd, LVDd)
O'Driscoll_2018[67]	40	COC	0	21 ± 2	Inactive/sedentary (<2.5 MET-h/week, > 8 h/day sitting time)	43.2 ± 5.2	Echo (LVM, IVSd, PWTd, LVDd)
Park_2003[68]	8	n/a	100	63 ± 2	Sedentary	21.6 ± 2.6	Echo (LVDd)
Perrault_1982_1[69]	11	n/a	0	19 ± 1	DNR	DNR	Echo (IVSd, PWTd, LVDd)
Perrault_1982_2[69]	13	n/a	0	40 ± 3	DNR	DNR	Echo (IVSd, PWTd, LVDd)
Pickering_1997[70]	10	n/a	60	62 ± 2	Sedentary	25.0 ± 3.2	Echo (IVSd, PWTd, LVDd)
Rahimi_2018_1[71]	10	n/a	100	32 ± 7	Non-athlete (no specific exercise activities)	DNR	Echo (LVM, IVSd, PWTd, LVDd)
Rodrigues_2006[72]	23	n/a	0	31 ± 4	Sedentary	39 ± 5	Echo (LVM, IVSd, PWTd, LVDd)
Rojek_2015[73]	21	0	24	33 ± 6	Starting to prepare for triathlon competition	DNR	Echo (LVM, IVSd, PWTd, LVDd)
Rubal_1987_1[19]	10	n/a	100	19–31	No participation in regular programme of physical conditioning	38 ± 8	Echo (LVM, IVSd, PWTd, LVDd)
Rubal_1987_2[19]	10	n/a	0	19–30	No participation in regular programme of physical conditioning	42 ± 7	Echo (LVM, IVSd, PWTd, LVDd)
Saadatnia_2016[96]	12	10[a]	0	23 ± 3	Untrained (currently no regular exercise), non-athlete	37.6	Echo (LVM, IVSd)
Sagiv_1989[74]	20	n/a	0	67 ± 4	Physically active in supervised aerobic programme for > 1 year, 3×/week	29.9	Echo (IVSd, PWTd)
Sayevand_2015[76]	10	10[a]	100	23 ± 1	No regular training in the previous year	DNR	Echo (LVM, IVSd, PWTd, LVDd)
Scharf_2015[77]	42	n/a	0	44 ± 5	Sedentary (<3 h/week of physical training)	DNR	CMR (LVM)
Shapiro_1983[20]	15	15	0	26	No recent participation in sport	48.6 ± 4.0	Echo (LVM, IVSd, PWTd, LVDd)
Skattebo_2020[79]	12	n/a	42	29 ± 6	Untrained (≤ 1 exercise training session/week during previous year)	44.5	Echo (LVM, IVSd, PWTd, LVDd)
Slordahl_2004[80]	12	n/a	100	22 ± 1	Sedentary (no formal training programme <3 months prior to the study)	42.6 ± 2.9	Echo (LVM, IVSd, PWTd, LVDd)
Soto_2008[81]	12	n/a	50	69 ± 6	Sedentary (≤30 min/day, <2 days/week)	23 ± 3	Echo (LVM)
Spence_2011[82]	10	n/a	0	27 ± 1	<3 h/week of structured activity	45.8 ± 1.6	CMR (LVM, IVSd, PWTd, LVDd)
Spina_1992[84]	17	n/a	41	27 ± 16	Sedentary (no regular exercise for 6 months prior to study)	40.9	Echo (LVDd)
Spina_1997[86]	8	n/a	0	66 ± 5	Sedentary (no regular exercise for 6 months prior to study)	28.7	Echo (LVM, IVSd, PWTd, LVDd)
Spina_2000[85]	10	n/a	100	54 ± 3	Sedentary (<2×/month of regular physical activity)	22.0 ± 0.5	Echo (LVM, IVSd, PWTd, LVDd)

Continued

Table 1 *Continued*

Study (author, year)	*n*	Control (*n*)	Female (%)	Age [mean ± SD or (range)]	Population	VO_{2max} [mL/min/kg) (mean ± SD or (range)]	Imaging modality (dimensions included)
Spina_2004[83]	22	14[a]	50	82 ± 4	Sedentary	17 ± 3	Echo (LVM, IVSd, PWTd, LVDd)
Vance_2014[87]	19	22[a]	0	22–37	Untrained (<2 h/week of aerobic training prior to enrolment,<50 mL/kg/min)	37.1 ± 7.1	Echo (LVM, IVSd, PWTd, LVDd)
Vanhees_1992[88]	27	n/a	0	38 ± 2	Sedentary (<1 h/week of sport activities)	n/a	Echo (LVM, IVSd, PWTd, LVDd)
Wieling_1981_1[21]	9	17[d]	0	20 ± 2	Freshmen (active in high school sports, but no daily training programme)	58 ± 8	Echo (IVSd, PWTd, LVDd)
Windecker_2002[92]	8	n/a	0	36 ± 5	Healthy (DNR prior physical activity levels)	46 ± 6	Echo (LVM, IVSd, PWTd, LVDd)
Wolfe_1979[22]	12	10[a]	0	37	No engagement in regular exercise for at least 5 years prior	42.4 ± 2.4	Echo (LVM, IVSd, PWTd, LVDd)
Wolfe_1992[93]	7	4[a]	100	19 ± 2	Sedentary (no previous participation in endurance-type sport)	41.3 ± 3.3	Echo (LVM, IVSd, PWTd, LVDd)
Younis_1987[94]	19	n/a	0	19	Non-athlete, active	53 ± 6	Echo (LVM, IVSd, PWTd, LVDd)
Trained							
Baggish_2009[e32]	38	n/a	50	20 ± 1	University students participating in official competitive athletics	DNR	Echo (LVM, IVSd, PWTd)
Bonaduce_1998[34]	15	15[d]	0	21 ± 4	High level bicyclists competing in Italian amateur national teams for >3 years	62 ± 4	Echo (LVM, IVSd, PWTd, LVDd)
D'Ascenzi_2015[39]	91	n/a	0	23 ± 6	Professional athletes (soccer, basketball, volleyball) competing in national or international level	DNR	Echo (LVM, IVSd, PWTd, LVDd)
Ehansi_1978[42]	8	n/a	13	17–19	University swim team (no regular exercise for 2–7 months before the beginning of training)	51.8 ± 2.0	Echo (LVM, PWTd, LVDd)
Fagard_1983[45]	12	12[d]	0	24 ± 1	Belgian cyclists (professional and amateur)	60.9 ± 1.5	Echo (IVSd, PWTd, LVDd)
Grace_2018_2[14]	17	n/a	0	61 ± 5	Masters athletes (national competitors, in triathlon, athletics, sprint cycling, racquet sports)	40.4	Echo (LVM, IVSd, PWTd, LVDd)
Kleinnibbelink_2021_1[57]	19	n/a	0	26 ± 4	Elite (Olympic) rowers	DNR	Echo (LVM, IVSd, PWTd, LVDd)
Kleinnibbelink_2021_2[57]	8	n/a	100	27 ± 2	Elite (Olympic) rowers	DNR	Echo (LVM, IVSd, PWTd, LVDd)
Lamont_1980[58]	11	n/a	100	21 ± 4	University swim team	DNR	Echo (LVM, IVSd, PWTd, LVDd)
Naylor_2005[18]	22	12[a]	23	20 ± 3	Elite rowers	DNR	Echo (LVM, IVSd, PWTd, LVDd)
Rahimi_2018_2[71]	10	n/a	100	31 ± 9	Athlete (>5×/week, 2 h)	DNR	Echo (LVM)
Sareban_2018[75]	15	n/a	0	20 ± 3	National or international level rowers	67 ± 6	Echo (LVM, LVDd)
Shah_2018[78]	9	n/a	0	19 ± 1	Newly matriculated, recruited members of varsity rowing	39.2	Echo (LVM, IVSd, PWTd, LVDd)
Venckunas_2006[89]	12	11[a]	0	25 ± 7	Distance runners	DNR	Echo (LVM, IVSd, PWTd, LVDd)
Wasfy_2018[90]	8	4[d]	0	DNR	Newly matriculated, recruited members of varsity rowing	DNR	Echo (LVM)

Continued

Table 1 Continued

Study (author, year)	n	Control (n)	Female (%)	Age [mean ± SD or (range)]	Population	VO_{2max} [mL/min/kg) (mean ± SD or (range)]	Imaging modality (dimensions included)
Wasfy_2019[91]	17	n/a	47	19 ± 0	Collegiate swimmers	DNR	Echo (LVM, LVDd)
Wieling_1981_2[21]	14	17[d]	0	22 ± 6	Senior oarsmen (completed at least 1 rowing season)	62.1 ± 7.6	Echo (IVSd, PWTd, LVDd)
Zilinski_2015[95]	45	n/a	0	48 ± 7	Recreational athletes	44.6 ± 5.2	Echo (LVM, PWTd)

Articles assessing independent training groups were evaluated as individual studies and were distinguished by letters (A, B, C, D); articles assessing independent population groups were evaluated as individual studies and were distinguished by number (1, 2, 3, 4).COC, cross-over control study; CMR, cardiac magnetic resonance imaging; DNR, did not report; Echo, echocardiography.
[a]Underwent pre- and post-measurement.
[b]Only reported age as a group.
[c]Control group in a clinical study that underwent intervention.
[d]Pre-measurement only.
[e]Only included group with parents who did not have hypertension.

participated in a mixed training regime elicited an increase in SMD of LVDd ($B = 0.3459$, $P = 0.0053$). Furthermore, only those that participated in swimming and rowing conferred an increase in SMD of LVDd ($B = 0.6527$, $P = 0.0056$; 0.3839, $P = 0.0198$, respectively) compared with running.

Impact of training protocol and status on left ventricular structural change

The length of the ET intervention was not associated with an increase in LVM when all studies from 2 to 52 weeks were evaluated together ($r = 0.103$; $P = 0.349$). The dose–response association between the length of the training programme and LVM between sexes are presented in *Figure 4A* and *B*. In the males, there was a significant positive association between the length of study and change in LVM ($r = 0.424$; $P < 0.005$), whereas in females, there was not ($r = 0.264$; $P = 0.274$). In the studies that reported VO_{2max}, all reported an increase in VO_{2max} except for one group of elite rowers.[75] The mean per cent change for VO_{2max} was higher in the untrained ($N = 69$) vs. the trained ($N = 8$) group (14.7 ± 7.6 and 8.6 ± 5.3; $P = 0.014$, respectively); however, the mean per cent change for LVM was higher in the trained ($N = 15$) vs. untrained ($N = 71$) group (19.7 ± 11.7 and 9.9 ± 10.1; $P = 0.007$, respectively). Additionally, pre-intervention VO_{2max} and LVM ($N = 63$) were positively associated with the change in LVM ($r = 0.285$, $P = 0.024$).

Discussion

This systematic review and meta-analysis synthesized data from 82 studies and 1908 participants examining the impact of ET intervention on LV structure in healthy men and women. The main findings were: (i) all LV structures increased in response to ET; (ii) LVM increased in young and middle-aged individuals but not in older individuals; (iii) a significant increase in wall thickness was observed in males, whereas an increase in LVM and LVDd was similarly augmented in males and females; (iv) trained status was associated with a greater increase in all LV structural parameters compared with untrained; (v) type of training influenced the adaption of LVM, PWTd, and LVDd with mixed-type training eliciting the greatest change; and (vi) mode of training conferred an increase in PWTd and LVDd with rowing and swimming eliciting the greatest increase (*Figure 5*). These findings in the context of influencing factors are discussed below.

Age

Ageing is associated with numerous cardiac changes, such as an increase in wall thickness and a decline in ventricular contractility.[3,98,99] Additionally, LVM and LVM index increases naturally with age in both males and females.[100] It is accepted that intense and long-lasting ET increases wall thickness beyond age-associated norms with a concomitant increase in cavity size in younger individuals; however, these changes have not been consistently observed in older individuals initiating an ET programme.[47,98] In a study comparing the impact of 6 months of unsupervised training on cardiac structure in younger (mean age 29 ± 4) and older (mean age 46 ± 7) individuals preparing for a marathon, both groups demonstrated a similar increase in LVM; however, the younger group also saw an increase in LV cavity size.[98] Another study reported that in sedentary seniors (71 ± 3) compared with Masters athletes (68 ± 3), the sedentary seniors' baseline LVM was significantly less than the Masters athletes'.[47] After 1 year of progressive training, the sedentary seniors' LVM increased closer to the LVM of the Masters athletes; however, they did not see a change in their mass–volume ratio.[47] A potential reason for this could be that long and intense programmes required to elicit such changes are cautiously initiated in the previously inactive older population due to the concern of causing a musculoskeletal injury or an adverse cardiac event. However, it would be important to determine if high intensity and longer types of ET could elicit positive remodelling in these older age groups, safely to prevent age-associated cardiac structural and functional changes such as LV hypertrophy and LV stiffening.[99] In order to prevent the age-related, cross-linked advanced glycation end products in the vascular and LV walls and to elicit change in the number and volume of cardiac myocytes, exercise training needs to start early, and if started later in life, may be too late.[101] Secondly, sedentary adults may need a longer period of time to see improvements in cardiac structure and function compared with their younger counterparts.[101] In the present study, the average length of study in the older group was 24.1 ± 18.7 (6–52) weeks and the average hours of ET per week was 2.5 ± 1.3 h. Further research examining the age ET needs to be undertaken to attenuate age-related decline in cardiac structure and function and thereby cardiac risk as well as determining age-appropriate training programmes.

Sex differences

Many of the mechanisms that contribute to cardiac sex differences are unclear, including the magnitude and temporal sequence of cardiac

Table 2 Endurance training intervention details

Study (author, year)	Length of study (weeks)	Modality	Intensity	Progressive training	Type of training	Frequency (days/week)	Time per session, min	Total hours (h/week)
Untrained								
Adams_1981[28]	11	RUN[a]	85% HR_{max}	DNR	CONT	5	50	4.2
Aksakai_2013[29]	26	RUN	11 →≥17 RPE (Borg scale)	↑ volume	CONT	DNR	60–240	DNR
Alves 2009_1,2,3,4[30]	12	RUN	HR between VT → HR slightly above RCP	DNR	CONT	3	60	3
Andersen_ 2010_A[31]	16	RUN	82% MHR	DNR	CONT	2–3	60	2.5[c]
Andersen_2010_B[31]	16	SOCCER	Football session	DNR	MIX	2–3	60	2.5[c]
Arbab-Zadeh_2014[3]	52	RUN, CYCLE, or SWIM	5 training zones (base pace – intervals)	↑ duration ↑ intensity	MIX	3–6	30–180	8[c]
Bates_2013[c33]	16	CYCLE	70–80% VO_{2max} and RPE 12–14 (Borg scale)	Reassess VO_{2max} at 8 wks, work adjusted to achieve THR	CONT	3	30	1.5
Boone_2014[35]	6	CYCLE	4 intensity profiles from 50 to 130% $Watts_{max}$	DNR	MIX	3.5	60	3.5[c]
Camargo_2008[36]	12	RUN	70% MHR	DNR	CONT	3	35	1.5
Cornelissen_2011[37]	10	WLK, RUN, CYCLE, or STEP	66% HRR	DNR	CONT	3	60	3
Cox_1986[38]	7	RUN, CYCLE	85–90% VO_{2peak} (cycle ergometer) – 100% VO_{2max} (5 × 5 min intervals)	↑ 11 W/week	MIX	6	40	4
Dart_1992[12]	4	CYCLE	70% VO_{2max}	Work adjusted to maintain 70% VO_{2max}	CONT	3	30	1.5
DeMaria_1978[40]	11	RUN	70% HR_{max} (ED)	Work adjusted to maintain 70% HR_{max}	CONT	4	60	4
Egelund_2017_1,2[41]	12	CYCLE	60–96% VO_{2max}	↑ intensity	MIX	3	50	2.5
Ehsani_1991[13]	52	RUN, CYCLE	60–100% VO_{2max}	↑ VO_{2max} + supplement with intervals + re-measure VO_{2max} every 3 months	MIX	5	60	5
Esfandiari_2014_A[43]	2	CYCLE	95–100% VO_{2max}	↑ #intervals	INT	3	20[c]	1[c]
Esfandiari_2014_B[43]	2	CYCLE	65% VO_{2max}	↑duration	CONT	3	105[c]	5.25[c]
Eskelinen_2016_A[44]	2	CYCLE	Maximal effort	↑ #intervals	INT	3	18.5[c]	1
Eskelinen_2016_B[44]	2	CYCLE	60% peak workload	↑duration	CONT	3	50[c]	2.5[c]
Fujimoto_2010[47]	52	RUN	Below VT (~75–85 HR_{max}) – VT threshold (~85–90% HR_{max}) + intervals (5–10 beats below HR_{max})	↑intensity + duration + intervals	MIX	5[c]	25–60	
Fujimoto_2013[46]	52	RUN	70–80% HR_{max}	↑duration ↑ frequency	CONT	3–4	25–40	2.3
Grace_2018_1,2[14]	6	CYCLE	50% PPO	DNR	INT	1	18	0.3
Haykowsky_2005[48]	12	CYCLE	60–80% HRR	↑ duration	CONT	3	15–42.5	2.1

Continued

Table 2 Continued

Study (author, year)	Length of study (weeks)	Modality	Intensity	Progressive training	Type of training	Frequency (days/week)	Time per session, min	Total hours (h/week)
Hedman_2017[49]	12	CYCLE	Alternating increases in resistance and cadence until peak effort achieved	DNR	CONT	3	45–60	3
Hickson_1982_1,2[50]	10	RUN, CYCLE	Up to 100% VO_{2max} by end of exercise session	↑ intensity to reach VO_{2max}	MIX	6	30–45	4
Holloway_2018[15]	6	CYCLE	90% VO_{2max}	Adjustment of training load	INT	3	13–15	0.7[c]
Huang_2019_1[51]	6	CYCLE	60% VO_{2max}	Adjustment of training load	CONT	5	30	2.5
Huang_2019_2[51]	6	CYCLE	80% VO_{2max}	Adjustment of training load	INT	5	30	2.5
Hulke_2012_1,2[52]	16	RUN	Somewhat hard (RPE scale)	DNR	CONT	8	60	8
Hulke_2012_A[16]	20	RUN	59.5% HR_{max} (age-predicted)	DNR	CONT	8	25–30	4
Hulke_2012_B[16]	20	RUN	74.9% HR_{max} (age-predicted)	DNR	CONT	8	25–30	4
Hwang_2016_1[53]	8	ROW[b]	90% HR_{peak}	DNR	INT	4	25	1.7[c]
Hwang_2016_2[53]	8	ROW[b]	70% HR_{peak}	DNR	CONT	4	32	2.1[c]
Kiflom_2022[55]	12	RUN, CYCLE, SKIPPING, BALL games	50–75% MHR	DNR	CONT	3	60–80	3.5[c]
Kivisto_2006[56]	12	CYCLE	60–75% HR_{max}	↑ intensity ↑ duration	CONT	3–4	30	1.75[c]
Krzeminski_1989[17]	13	CYCLE	50–75% VO_{2max}	↑ intensity	CONT	3	30	1.5
Landry_1985[59]	20	CYCLE	60–85% HRR	↑ intensity	CONT	4–5	40–45	3.4
Lane_2014_1,2[60]	8	CYCLE or ELL or RUN	>60–90% HR_{max}	DNR	CONT	3	30–60	2.25[c]
Lusiani_1986[61]	11	RUN	DNR	DNR	MIX	3	DNR	DNR
Mahdiabadi_2013_A[62]	8	RUN	70% HR_{max}	DNR	CONT	3	45	2.25
Mahdiabadi_2013_B[62]	8	RUN	70% HR_{max}	DNR	INT	3	45	2.25
Masoomeh_2012[97]	8	RUN	65–80% HR_{max}	↑ intensity ↑ duration	CONT	3	16–30	1.2[c]
Marsh_1983[64]	DNR	RUN	70% HR_{max}	↑ distance Repeat examinations when mileage changed from 30 and 50 miles from baseline	CONT	5.5	DNR	DNR
Marsh_2021_1,2[63]	12	RUN, CYCLE	60–90% HR_{max}	Periodized progressive macrocycle (×3) plan	MIX	3	60	3
Matsuo_2014_A[65]	8	CYCLE	120% VO_{2max}	Recalculation of workload (W) as per equation	INT	5	10	0.8
Matsuo_2014_B[65]	8	CYCLE	50–90% VO_{2max}	Recalculation of workload (W) as per equation	INT	5	18	1.5
Matsuo_2014_C[65]	8	CYCLE	60–65% VO_{2max}		CONT	5	45	3.75

Continued

Table 2 *Continued*

Study (author, year)	Length of study (weeks)	Modality	Intensity	Progressive training	Type of training	Frequency (days/week)	Time per session, min	Total hours (h/week)
				Remeasurement of VO_2 every 2 weeks				
Morrison_1986[66]	35	RUN	65–75% HRR	↑ intensity to maintain HR	CONT	3	40	2
O'Driscoll_2018[67]	2	CYCLE	100% effort	N/A	INT	3	5.5	0.3
Park_2003[68]	36	RUN, CYCLE	50–60% HRR	Reperform ET and adjust intensity	CONT	3	60	3
Perrault_1982_1[69]	20	RUN	80% HR_{max} (ED)	Adjustment of training load to maintain intensity	CONT	3	30	1.5
Perrault_1982_2[69]	20	CYCLE	80% HR_{max} (ED)	Adjustment of training load to maintain intensity	CONT	3	30	1.5
Pickering_1997[70]	16	CYCLE	50% VO_{2max} – HR @ lactate threshold	↑ intensity ↑ duration	MIX	3	20–55	2.75[c]
Rahimi_2018_1[71]	12	SWIM (water aerobics)	60–80% HR_{max}	↑ intensity ↑ duration	CONT	3	30–60	2[c]
Rodrigues_2006[72]	26	RUN	HR_{AT} – HR 10% lower than RCP – HR @ RCP	↑ intensity	CONT	3	40–45	2.25
Rojek_2015[73]	52	RUN, CYCLE, SWIM	DNR	DNR	DNR	5	> 90	11.2[c]
Rubal_1987_1,2[19]	10	RUN	>70% HR_{max}	DNR	CONT	3.5	> 30	2[c]
Saadatnia_2016[96]	10	RUN	Maximal effort	↑ #intervals	INT	3	8–16	0.2[c]
Sagiv_1989[74]	12	RUN	70% VO_{2max}	Adjustment of training load to maintain intensity	CONT	3	30	1.5
Sayevand_2015[76]	4	CYCLE	80% VO_{2max}	Adjustment of training power output	INT	3	40	2
Scharf_2015[77]	16	RUN	65–90% HR_{max}	↑ frequency	MIX	2–4	DNR	DNR
Shapiro_1983[20]	6	RUN	DNR	DNR	CONT	5	15–30	1.8[c]
Skattebo_2020[79]	10	CYCLE	70–90% HR_{peak}	↑ #intervals	MIX	3	36–60	0.8[c]
Slordahl_2004[80]	8	RUN	50–95% HR_{max}	DNR	INT	3	25	1.25[c]
Soto_2008[81]	44	RUN, CYCLE	60–100% VO_{2max}	Re-measure VO_2 every 3 months	MIX	4–5	60	4.5[c]
Spence_2011[82]	24	RUN	Individualized (based on VO_{2peak} and time trial performances) periodized programme, included 3 phases [preparatory phase (low–moderate intensity), specific phase (hill running, short intervals), competition phase (intensity maintained, volume reduced)]	Variations in intensity, volume	MIX	3	60	3

Continued

Table 2 *Continued*

Study (author, year)	Length of study (weeks)	Modality	Intensity	Progressive training	Type of training	Frequency (days/week)	Time per session, min	Total hours (h/week)
Spina_1992[84]	12	RUN and CYCLE	50–95% VO_{2max}	Re-measure VO_2 every 3 weeks ↑ pace and power	MIX	6	40–45	4.25[c]
Spina_1997[86]	36	RUN, CYCLE	60–95% VO_{2max}	Re-measure VO_2 every 3 months and adjust intensity	MIX	5	60	5
Spina_2000[85]	48	RUN, CYCLE	60–95% VO_{2max}	Re-measure VO_2 every 3 months and adjust intensity	MIX	5	60	5
Spina_2004[83]	12	RUN, CYCLE, ROW	65–90% VO_{2max}	DNR	MIX	3	10–60	3[c]
Vance_2014[87]	16	RUN	Half-marathon training	DNR	DNR	DNR	DNR	DNR
Vanhees_1992[88]	16	RUN, CYCLE	70% HRR	Encouraged to improve performance	CONT	3	60	3
Wieling_1981_1[21]	30	Non-specific endurance, ROW	Non-specific endurance, interval training, rowing training	DNR	MIX	DNR	DNR	9[c]
Windecker_2002[92]	22	RUN or CYCLE	80% VO_{2peak}	DNR	CONT	≥4	≥60	≥4
Wolfe_1979[22]	26	RUN[a]	60–80% HR_{max}	↑ intensity ↑ duration	CONT	4	10–30	2[c]
Wolfe_1992[93]	11	RUN	80–85% HR_{max}	↑ frequency ↑ duration	CONT	3–5	20–45	2.2[c]
Younis_1987[94]	26	SWIM, RUN, T/F, basic sports, ball games	≥70% HR_{max}	DNR	CONT	DNR	≥2	12
Trained								
Baggish_2009[d32]	13	ROW	DNR (college training plan followed)	DNR (college training plan followed)	CONT	DNR (college training plan followed)	60–180	11.1[c]
Bonaduce_1998[34]	20	CYCLE, AER	DNR (followed training plan)	DNR (followed training plan)	CONT	7	180	21
D'Ascenzi_2015[39]	18	SOC, VB, BB	Low training period (high volume/low intensity and sprinting); peak training (70–95% HR_{max})	DNR (followed training plan)	MIX	DNR	DNR	12–20
Ehansi_1978[42]	9	SWIM	DNR (followed swim team training plan)	DNR	DNR	6	180	12
Fagard_1983[45]	DNR	CYCLE	DNR (followed competitive cycling training plan)	DNR	DNR	DNR	DNR	DNR
Kleinnibbelink_2021_1,2[57]	36	ROW	DNR (followed elite rowing training programme consisted of high intensity and endurance)	DNR	MIX	DNR	DNR	24–35

Continued

Table 2 *Continued*

Study (author, year)	Length of study (weeks)	Modality	Intensity	Progressive training	Type of training	Frequency (days/week)	Time per session, min	Total hours (h/week)
Lamont_1980[58]	13	SWIM	DNR (followed swim training programme)	DNR	MIX	5	180–240	16
Naylor_2005[18]	26	ROW, AER	80–90% HR_{max}	DNR (followed rowing training programme	CONT	6–7	270	29.3
Rahimi_2018_2[71]	12	SWIM (water aerobics)	60–80% HR_{max}	↑ intensity ↑ duration	CONT	3	30–60	2[c]
Sareban_2018[75]	11	ROW, RUN, CYCLE, SWIM	DNR (followed preparatory training period for elite athletes)	DNR	DNR	DNR	DNR	11.8[c]
Shah_2018[78]	13	ROW	DNR (collegiate rowing training programme)	DNR	CONT	5–6	60–180	11.9[c]
Venckunas_2006[89]	52	RUN	Increased training volume ~ 50% (20–125) in first 2 months and then continued at this training volume for remaining 10 months, participated in no more than once every 2 months, allowed to alternate mileage of sessions as desired	↑ volume	CONT	DNR	~120	DNR
Wasfy_2018[90]	12	ROW	DNR (collegiate rowing training programme)	DNR	DNR	DNR	DNR	13
Wasfy_2019[91]	13	SWIM	DNR (followed collegiate training programme)	DNR	CONT	7	60–180	16.3
Wieling_1981_2[21]	30	ROW	DNR (followed collegiate rowing training programme)	DNR	MIX	DNR	DNR	12[c]
Zilinski_2015[95]	18	RUN	DNR (followed marathon training programme)	↑ distance	CONT	4–5	DNR	4

Articles assessing independent training groups were evaluated as individual studies and were distinguished by letters (A, B, C, D); articles assessing independent population groups were evaluated as individual studies and were distinguished by number (1, 2, 3, 4). AER, aerobics; BB, basketball; CYCLE, cycling; ED, exercise-determined; ELL, elliptical; ET, exercise test; MHR, maximum heart rate; NR, not reported; PPO, peak power output; RCP, respiratory compensation point; ROW, rowing; RUN, outdoor running or treadmill; SOC, soccer; STEP, stationary stepping; T/F, track and field; THR, target heart rate; VB, volleyball; VT, ventilatory threshold.

[a]Bicycle ergometer substituted if injury or inclement weather.

[b]Arm/leg non-weight-bearing ergometer.

[c]Mean if hours or frequency reported as range, and when mean hours reported for entire study population.

Table 3 Pooled means for left ventricular structure and function

LV parameter (units)	Descriptives (*N* analyses; *N* participants)	Pre-intervention (mean ± SD)	Post-intervention (mean ± SD)	% change ± SD (range)
Untrained				
LV structure				
LVM (g)	57; 1035	139.7 ± 25.6	149.4 ± 27.0	7.7 ± 7.3 (−4.6 to 32.1)
LVMi (g/m^2)	39; 824	77.7 ± 12.6	83.8 ± 14.0	8.3 ± 8.3 (−5.13 to 31.7)
IVSd (mm)	65; 1144	9.0 ± 1.4	9.3 ± 1.2	3.6 ± 6.3 (−19.5 to 20.3)
PWTd (mm)	63; 1139	8.8 ± 1.1	9.2 ± 1.1	4.6 ± 6.3 (−5.7 to 28.9)
LVDd (mm)	64; 1125	47.9 ± 1.9	48.8 ± 1.8	2.0 ± 2.8 (−6.1 to 13.2)
LV function				
VO_{2max} (mL/min/kg)	61; 1017	37.9 ± 4.5	44.1 ± 4.5	15.0 ± 7.6 (0.4 to 33.8)
VO_{2max} (L/min)	7; 132	2.4 ± 2.4	2.6 ± 2.5	12.2 ± 4.4 (8.8 to 21.5)
End-diastolic volume (mL)	33; 546	125.5 ± 18.0	131.6 ± 42.8	5.5 ± 5.3 (−1.3 to 21.7)
End-systolic volume (mL)	31; 524	49.2 ± 9.4	50.0 ± 8.8	1.2 ± 7.3 (−17.5 to 14.4)
Stroke volume (mL)	40; 702	76.3 ± 12.4	82.7 ± 12.4	9.0 ± 9.8 (−7.2 to 33.5)
Trained				
LV structure				
LVM (g)	10; 229	196 ± 43.8	217.7 ± 50.8	11.2 ± 5.5 (3.8–19.5)
LVMi (g/m^2)	13; 281	111.2 ± 17.7	124.4 ± 18.7	11.5 ± 7.7 (−0.9 to 29.4)
IVSd (mm)	11; 240	10.0 ± 1.6	10.8 ± 2.5	7.6 ± 4.7 (1.1 to 18.2)
PWTd (mm)	13; 293	9.7 ± 1.1	10.4 ± 1.1	6.3 ± 5.8 (−2.0 to 20.0)
LVDd (mm)	13; 270	52.1 ± 2.1	53.3 ± 2.0	2.6 ± 2.9 (−1.16 to 8.8)
LV function				
VO_{2max} (mL/min/kg)	7; 118	53.5 ± 3. 8	57.3 ± 4.9	8.9 ± 5.7 (0 to 15.5)
VO_{2max} (L/min)	1; 17	3.2 ± 0.6	3.5 ± 0.5	7.8
End-diastolic volume (mL)	6; 167	133 ± 29.4	142.2 ± 30.3	7.5 ± 10.2 (−8.5 to 21.4)
End-systolic volume (mL)	5; 159	55.6 ± 16.0	60.4 ± 15.4	6.9 ± 9.6 (−6.9 to 18.5)
Stroke volume (mL)	4; 61	89.4 ± 17.0	92.5 ± 18.0	3.1 ± 9.3 (−9.6 to 12.1)

changes in response to ET. In the present study, both males and females demonstrated a significant increase in LVM; however, the SMD was greater in males than females. This is in-fitting with other reports where LVM was shown to increase in both males and females.[102] Additionally, a positive correlation was observed between the length of the study programme and change in LVM in males, but not in females. Few studies have explored cardiac adaptation over time between the sexes. One study investigated sex differences in cardiac structure every 3 months over 1 year of an intensive ET intervention.[103] They found that both sexes demonstrated an increase in LVM (scaled to fat-free mass); however, the females demonstrated the greatest increase in LVM within the first 3 months of a 12-month training programme, compared with the males where LVM progressively increased throughout the entire 12 months, with the greatest increase in the first 6 months.[103] Additionally, they found that with an increasing exercise stimulus, LVM was markedly augmented in males, whereas in females, it was attenuated. Analysis of PWTd, IVSd, and LVDd revealed that only PWTd and IVSd significantly increased in males, whereas LVDd was similarly augmented in both males and females. Previous research in athletes demonstrates that when females LVDd is indexed for BSA, they exhibit a higher cavity size. When involved in dynamic sports, females predominately exhibit eccentric LV hypertrophy, whereas a significant proportion of males in dynamic sports demonstrate concentric LV remodelling.[104] Some potential mechanisms contributing to these sex differences are that males have a higher circulating concentration of testosterone and a higher density of myocardial testosterone receptors.[105] Additionally, a higher exercise-related systolic blood pressure in males may play a role in the development of LV hypertrophy.[106]

Training status

Early studies comparing athletes to inactive individuals demonstrated that exercise leads to increased wall thickness, LVM, and LV cavity size.[4,7,107] In the present analysis, this was supported by an increase in all LV structural parameters in both the untrained and trained groups. However, the trained group had a greater increase compared with the untrained group, suggesting that prior training and/or cardiorespiratory fitness is an important factor in cardiac adaptation. It can be speculated that previously trained individuals would have less capacity to increase their LVM due to already having a well-adapted heart. Possible explanations for this could be that athletes train at a higher overall volume of exercise eliciting a greater response, have a superior genetic endowment, and have a greater capacity to push their bodies to their limits.[107] Another factor could be the use of prohibited substances; however, this was not reported in any of the studies. Our data support the suggestion that a greater number of training hours per week may stimulate enhanced cardiac adaption. Additionally, a higher baseline VO_{2max} was significantly correlated with greater changes in LVM, and the post-intervention VO_{2max} in the untrained group was less than that of the baseline VO_{2max} seen in the trained group. Thus, these findings may also represent

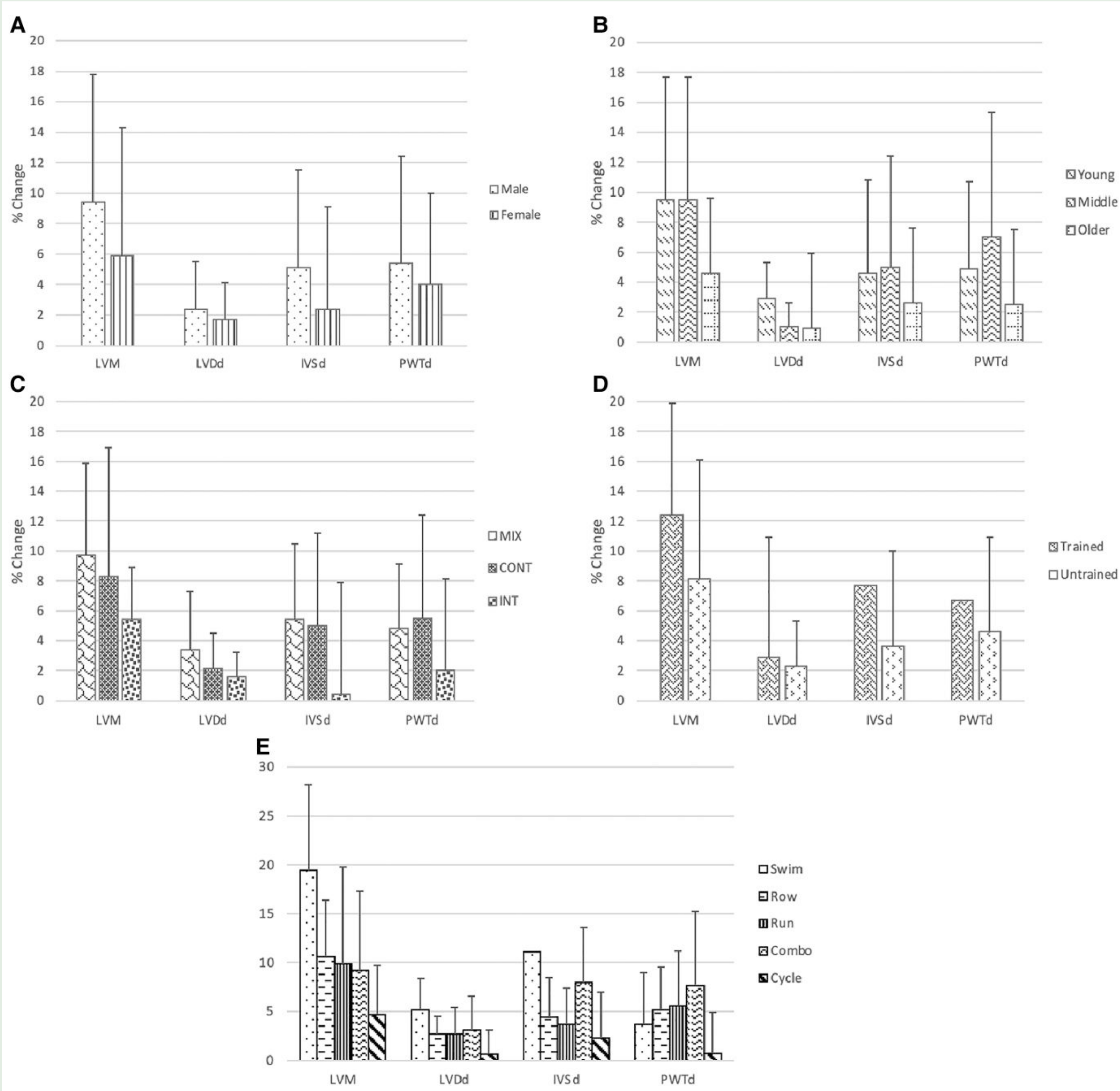

Figure 2 Change in left ventricular structure by (*A*) sex; (*B*) age group; (*C*) training type; (*D*) trained status; and (*E*) training mode.

in part a genetic component driving the enhanced adaptions seen in trained individuals.

Type of training

The type of training programme may influence the magnitude of cardiac remodelling. Previous research suggests that interval training involving shorter intervals and rest periods primarily improves the oxidative capacity of peripheral muscles, whereas continuous and aerobic interval training involving longer intervals targets central adaptations such as cardiac function.[65,108] Previous studies have shown continuous and aerobic interval training is characterized by both an increase in LVDd and LV wall thickness, and consequently LVM.[10,65] Increases in LVM have been observed in short/sprint interval training and can occur with submaximal heart rates to allow optimal CA^{2+} cycling,[108] increased training volumes, high baseline cardiorespiratory fitness, and longer duration programmes (i.e. >3 months).[56,65,82] In the present meta-analysis, mixed-type training elicited the greatest increase in LVM, PWTd, and LVDd, compared with interval- and continuous-type training. It is possible that changes in these structural parameters were not seen in interval-type training as they are often shorter duration as a time-efficient method to improve VO_{2max}, and, therefore, the length of study and training volume were not sufficient to induce these cardiac structural changes. Additionally, the present analysis did not report sprint and aerobic interval training separately; therefore, we were not able to determine cardiac structural changes specific to these two interval training types.

Study name	Std diff in means	Lower limit	Upper limit	Total	Relative weight
Aksakai 2013	0.619	0.252	0.99	34	2.07
Alves 2009_1	0.263	0.001	0.52	58	4.07
Alves 2009_2	0.514	0.008	1.02	17	1.09
Alves 2009_3	0.263	0.001	0.52	58	4.07
Alves 2009_4	0.413	0.004	0.82	25	1.67
Arbab-Zadeh 2014	1.709	0.822	2.60	12	0.35
Baggish 2009	0.329	0.002	0.66	38	2.62
Bates 2013	1.512	0.605	2.42	10	0.34
Bonaduce 1998	0.859	0.267	1.45	15	0.80
Boone 2014	0.531	-0.167	1.23	9	0.57
Camargo 2008	-0.001	-0.801	0.80	6	0.44
Cornelissen 2011	0.327	-0.176	0.83	16	1.10
Cox 1986	0.672	0.018	1.33	11	0.65
D'Ascenzi 2015	0.427	0.212	0.64	91	6.05
Dart 1992	0.000	-0.619	0.62	10	0.73
DeMaria 1978	0.769	0.314	1.22	24	1.34
Egelund 2017_1	0.599	0.244	0.95	36	2.22
Egelund 2017_2	0.548	0.202	0.89	37	2.34
Ehsani 1978	1.425	0.441	2.41	8	0.29
Esfandiari 2014_A	0.000	-0.692	0.69	8	0.58
Esfandiari 2014_B	0.000	-0.692	0.69	8	0.58
Eskelinen 2016_B	0.041	-0.483	0.57	14	1.02
Fujimoto 2010	0.833	0.075	1.59	9	0.49
Fujimoto 2013	0.000	-0.506	0.51	15	1.09
Grace 2018_A	0.000	-0.418	0.42	22	1.60
Grace 2018 _B	0.000	-0.475	0.48	17	1.23
Haykowsky 2005	-0.000	-0.693	0.69	8	0.58
Hickson 1982_1	0.836	0.031	1.64	8	0.43
Hickson 1982_2	0.925	0.040	1.81	7	0.36
Hosseini 2012	0.000	-0.619	0.62	10	0.73
Huang 2019_1	0.497	0.008	0.99	18	1.16
Hunag 2019_2	0.497	0.008	0.99	18	1.16
Hulke 2012_1	0.000	-0.302	0.30	42	3.05
Hulke 2012_2	0.000	-0.299	0.30	43	3.12
Hulke 2012_A	0.000	-0.523	0.52	14	1.02
Hulke 2012_B	0.000	-0.543	0.54	13	0.94
Hwang 2016_1	0.000	-0.506	0.51	15	1.09
Hwang 2016_2	0.000	-0.523	0.52	14	1.02
Kanakis 1982_1	1.049	0.053	2.05	6	0.28
Kanakis 1982_2	0.001	-0.800	0.80	6	0.44
Kivisto 2006	0.805	0.202	1.41	14	0.77
Kleinnibbelink 2021	0.713	0.291	1.14	27	1.56
Lamont 1980	0.956	0.242	1.67	11	0.55
Landry 1985	0.868	0.354	1.38	20	1.05
Lane 2014_1	-0.000	-0.371	0.37	28	2.03
Lusiani 1986	0.847	0.214	1.48	13	0.69
Marsh 1983	0.000	-0.565	0.57	12	0.87
Marsh 2021_1	0.553	0.243	0.86	46	2.90
Marsh 2021_2	0.547	0.135	0.96	26	1.64
Matsuo 2014_A	0.708	0.122	1.29	14	0.81
Matsuo 2014_B	0.805	0.202	1.41	14	0.77
Matsuo 2014_C	0.248	-0.284	0.78	14	0.99
Naylor 2005	0.443	0.005	0.88	22	1.45
O'Driscoll 2018	0.040	-0.270	0.35	40	2.90
Rahimi 2018_1	0.000	-0.619	0.62	10	0.73
Rahimi 2018_2	-0.000	-0.620	0.62	10	0.73
Rodrigues 2006	0.928	0.439	1.42	23	1.17
Rojek 2015	0.552	0.093	1.01	21	1.32
Rubal 1987_1	0.000	-0.619	0.62	10	0.73
Rubal 1987_2	0.000	-0.619	0.62	10	0.73
Saadatnia 2016	1.281	0.517	2.04	12	0.48
Sareban 2018	0.441	-0.089	0.97	15	0.99
Sayevand 2015	0.000	-0.619	0.62	10	0.73
Scharf 2015	0.547	0.223	0.87	42	2.65
Shah 2018	1.680	0.666	2.70	9	0.27
Shapiro 1983	0.769	0.193	1.34	15	0.84
Skattebo 2020	0.635	0.015	1.26	12	0.72
Slordahl 2004	0.954	0.272	1.64	12	0.60
Spence 2011	1.167	0.363	1.97	10	0.43
Spina 1997	1.086	0.213	1.96	8	0.37
Spina 2000	0.144	-0.479	0.77	10	0.72
Spina 2004	0.072	-0.347	0.49	22	1.59
Vance 2014	0.672	0.174	1.17	19	1.13
Vanhees 1992	0.713	0.291	1.14	27	1.56
Venckunas 2006	1.281	0.517	2.04	12	0.48
Waggoner 2008	-0.000	-0.566	0.57	12	0.87
Wasfy 2018	1.912	0.747	3.08	8	0.21
Wasfy 2019	1.245	0.612	1.88	17	0.70
Windecker 2002	1.571	0.535	2.61	8	0.26
Wolfe 1979	0.000	-0.565	0.57	12	0.87
Wolfe 1992	0.000	-0.740	0.74	7	0.51
Younis 1987	0.828	0.307	1.35	19	1.03
Zilinski 2015	0.526	0.214	0.84	45	2.87
	0.402	0.349	0.45		

Std diff in means and 95% CI

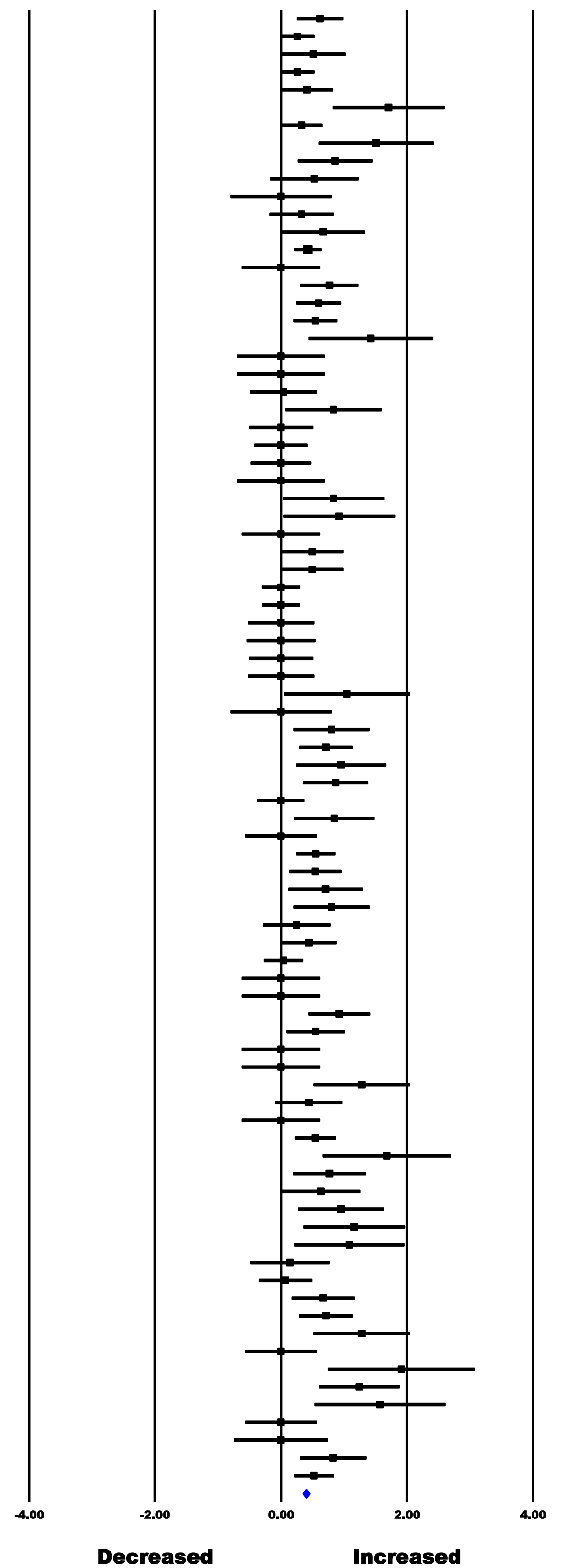

Figure 3 Forest plot of standardized mean difference between pre- and post-training left ventricular mass.

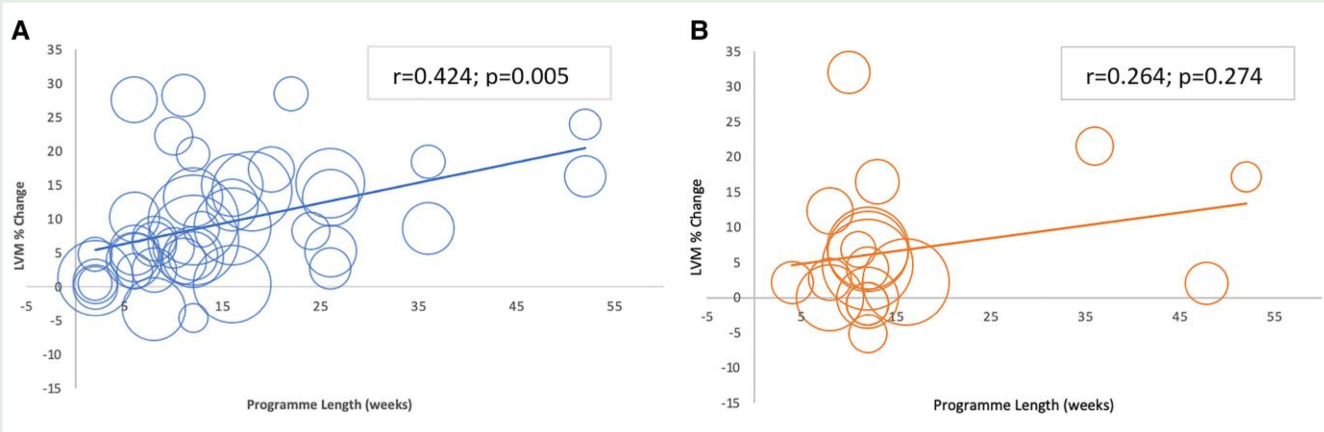

Figure 4 Dose–response association between the length of training programme and left ventricular mass in (*A*) males and (*B*) females.

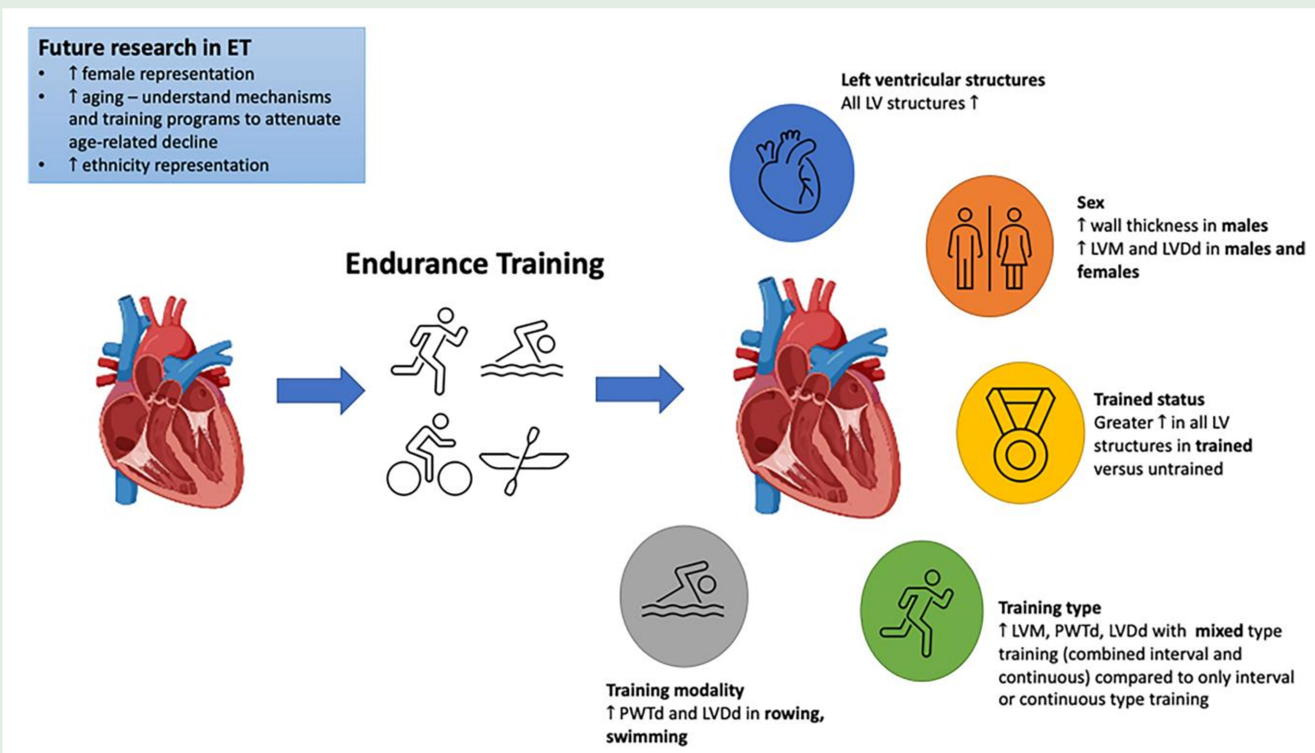

Figure 5 Schematic diagram of the influence of moderator variables (sex, age group, training status, training type, mode of training) on left ventricular structures.

Mode of training

Left ventricular structural changes are influenced by the degree of static and dynamic components involved in the sport modality.[109] We found all modes of exercise increased LVM; however, the mode of exercise influenced the adaptation of PWTd and LVDd with swimming and rowing eliciting the greatest increase and cycling conferring the smallest change. Modalities that combine both dynamic and static components (i.e. rowing) require increases in cardiac output, heart rate, SV, systolic blood pressure, and mean arterial pressure, thereby generating the greatest changes in cavity dimensions and LV wall thickness.[109] Conversely, studies that are predominately dynamic (i.e. running) have lower afterload, and therefore will observe smaller LV structural changes.[109] Although there were only three studies that included swimming as the training stimulus, all elicited a significant increase in LVM, PWTd, and LVDd.[42,58,91] Swimming is a unique sport, due to the physiological response of being immersed in water, and less gravitational forces being exerted on the swimmer.[110] The horizontal position of swimming aids venous return, which is increased with the kicking of the legs. There is a concomitant increase in preload, increasing SV, and cardiac output which can generate an increase in both wall thickness and

LVDd.[110] In studies that have compared swimming to other sporting disciplines, the results are unequivocal;[4,111–116] however, when age and BSA are accounted for, swimming is associated with greater cardiac dimensions.[115,116] Although there is a small sample size of swimmers in this analysis, the present findings confirm previous reports. Interestingly, cycling elicited the smallest changes across LV structures, which does not conform to previous literature where cyclists have been shown to elicit the greatest increases in LVM compared with other sport types.[10,116–118] In the present analysis the majority (93%) of the studies utilising cycling as their training modality were in previously sedentary individuals, therefore, the disparity seen in this study compared to other studies that investigated the impact of cycling on LVM is likely due to the other studies including elite male and female cyclists.[116,117] The stimulus in the cycling studies included in the present analysis was likely not intense or long enough to elicit large increases in LVM compared with that observed in previous studies.

Limitations

The present analysis has some limitations. First, it is important to note that these results pertain to healthy populations, and may not translate to clinical populations. Secondly, the impact of ethnicity could not be determined as only four studies reported ethnicity. Thirdly, while we compared the impact of ET in males and females from individual studies, there were very few within-study sex comparisons. Our overall female sample size was much smaller than that of males, and the Egger's test for publication bias was significant in males, warranting further studies exploring sex differences. Fourthly, a possible confounding factor seen in the trained group was that several of these studies reported strength training as part of their training programme for their specific sports which may have impacted LV structural parameters seen in this group. We are unable to isolate the impact of this additional strength training on cardiac adaptations within an ET intervention;, however, a previous meta-analysis of cross-sectional studies reported no significant difference in LV structural parameters between athletes that are combined endurance-trained *and* strength-trained compared with those that are only endurance-trained.[10] Fifthly, we found cycling interventions elicited the smallest change in LVM, contrary to previous cross-sectional studies where cyclists demonstrated greater LVM compared with other sports. This observation may be due to most cycling studies included in this meta-analysis were conducted with untrained participants, therefore may not represent the competitive cyclist response to an ET. Sixthly, swimming elicited the greatest increase in LVM; however there were only three studies and a very small sample size within each one; therefore, these results need to be interpreted with caution and confirmed in additional studies. Lastly, there was inconsistent reporting between studies with respect to whether they indexed for BSA, fat-free mass, or allometric scaling; therefore, we could not determine how the LV adaptations would be altered when indexed following changes in body mass or composition.

Conclusion

From this review, we confirm our hypothesis that LV structure is significantly increased following ET. Males, younger, trained individuals and ET interventions involving mixed training regimes elicit the greatest changes in LVM and other LV structural variables. Understanding these mediating factors during ET is important in developing effective training programmes and can help delineate between physiological adaptations to ET and potential pathology.

Authors' contributions

B.N.M. contributed to the design, screening, data extraction, and analysis, and wrote this review. K.G. contributed to design, data analysis, and interpretation, and critically reviewed the paper. E.K. and D.D. contributed to the design, search strategy, and performed the literature search. L.R. and R.J.M. contributed to the screening and data analysis. A.T.C. conceived the project, contributed to the design, review, data analysis and interpretation, resolved conflicts, and critically reviewed the paper. All authors reviewed the manuscript and gave final approval for submission.

Supplementary material

Supplementary material is available at *European Journal of Preventive Cardiology*.

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Conflict of interest: None declared.

Data availability

The data sets generated from the review are available from the corresponding author upon reasonable requests.

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