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CLINICAL RESEARCH
Clinical trials

Cardiovascular adaptation to training load in endurance athletes: a longitudinal study

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Abstract

Background and Aims Prior studies on cardiac remodelling associated with exercise have relied on self-reported data of uncertain accuracy. In the present study, exercise duration and intensity were objectively quantified using heart rate (HR) monitors in athletes, and these metrics were correlated with cardiac magnetic resonance findings.

Methods Young (16–23 years, $n = 69$) and middle-aged (45–70 years, $n = 82$) male endurance athletes with ≥80% of training sessions recorded via chest-worn HR monitors over 3 months were included. Training duration, session count, and intensity (classified into five HR zones and expressed as Edwards training impulse in arbitrary units) were analysed. Cardiac magnetic resonance measured indexed left/right ventricular volumes, ejection fraction, and left ventricular mass.

Results Younger athletes trained more than older athletes [169 (127–209) vs 78 (49–114) hours; 23 129 (17 880–28 305) vs 12 620 (7168–17 607) arbitrary units; both $P < .05$] over a 3-month period. In all athletes, light-to-moderate-intensity training exceeded thresholds of >6 or >9 metabolic equivalent of tasks to describe intense activity. Training duration ($r > .33$, $P < .05$ for all) and Edwards training impulse ($r > .29$, $P < .05$ for all) correlated with cardiac dimensions, but the duration always outperformed intensity. Time spent in lower HR zones (1 and 2) correlated more with cardiac dimensions than higher-intensity training. Partial least squares analysis identified training duration in Zones 1&2 and 3 and age as key determinants of cardiac remodelling, whereas intensity was not a significant determinant of cardiac dimensions.

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Conclusions Objective exercise quantification reveals new insights into cardiac remodelling, highlighting total exercise duration as a primary determinant of left/right ventricular volumes, independent of intensity. Traditional questionnaire-based methods may overlook these relationships.

Structured Graphical Abstract

Key Question

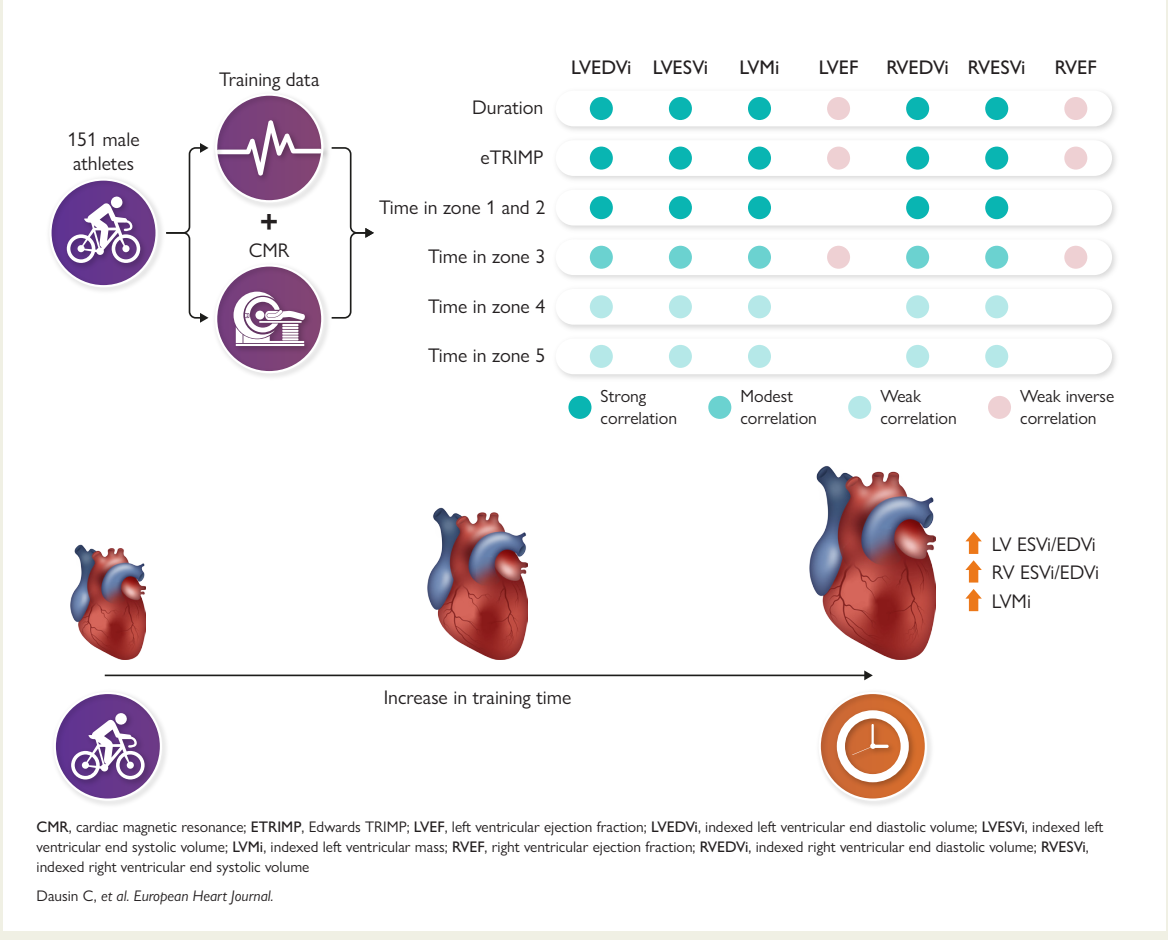
How does duration and intensity of exercise relate to cardiac volume and function?

Key Finding

In a cohort of 151 male endurance athletes ranging from 16 to 71 years of age, with ≥80% of training sessions recorded via chest-worn heart rate monitors over three-months, the main driving factor of increases in left and right ventricular volumes was training duration rather than training intensity.

Take Home Message

Objective exercise quantification reveals new insights into cardiac remodelling, highlighting total exercise duration as a primary determinant of ventricular volumes, independent of intensity. Traditional questionnaire-based methods may overlook these relationships.



CMR, cardiac magnetic resonance; **ETRIMP**, Edwards TRIMP; **LVEF**, left ventricular ejection fraction; **LVEDVi**, indexed left ventricular end diastolic volume; **LVESVi**, indexed left ventricular end systolic volume; **LVMi**, indexed left ventricular mass; **RVEF**, right ventricular ejection fraction; **RVEDVi**, indexed right ventricular end diastolic volume; **RVESVi**, indexed right ventricular end systolic volume

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Keywords Training load • Intensity • Exercise-induced cardiac remodelling

Introduction

Endurance exercise induces significant electrical, structural, and functional cardiac remodelling, enhancing performance but sometimes mimicking cardiac pathology.[1,2] Among athletes, those in endurance sports show the most pronounced adaptations, with substantial individual variation influenced by training load, genetics, and other factors.[1,3]

A key clinical challenge is distinguishing physiological adaptations from potential pathology. When cardiac changes correspond with the athlete's training exposure, they are typically considered benign. In contrast, adaptations that appear excessive relative to training volume or intensity may raise concern for underlying disease.[4]

Despite its importance in these assessments, training load is classically estimated via questionnaires, which are prone to recall bias and

imprecise intensity assessment.[5–8] Exercise intensity is most often quantified from activity-based estimates of metabolic equivalent of tasks (METs) that do not readily allow for the considerable variation within and between individuals for a given task.[9] Subjective training load estimates based on questionnaires may therefore obscure the relationship between training exposure and cardiac remodelling. Wearable activity trackers now allow precise, objective training load quantification.[10,11] We previously developed a software pipeline to semi-automate training load assessment from training files.[8] We also incorporated the Edwards training impulse (eTRIMP), a heart rate-based metric that quantifies internal training load by weighting time spent in predefined heart rate zones according to exercise intensity. To date, no studies have examined the relationship between objectively quantified training load and cardiac remodelling.

In this study, we examined the association between cardiac structural and functional parameters and training load derived from heart rate monitors in male endurance athletes. We hypothesized that moderate-to-high-intensity exercise would have a greater impact on cardiac volumes and function than low-intensity exercise and that eTRIMP, as a composite measure of exercise duration and intensity, would better reflect cardiac adaptation than either component alone.

Methods

Study participants

Study participants were recruited from two international multicentre studies: the Prospective Athlete Heart (Pro@Heart) study[3,12] and the Master Athlete's Heart (Master@Heart) study.[13,14] Pro@Heart is a prospective study in which young male and female elite endurance athletes [cycling, distance running (≥1500 m), duathlon, triathlon, rowing, swimming (≥400 m), or cross-country skiing] competing at the national or international level were recruited at a starting age between 16 and 23 years, with the aim to characterize cardiac remodelling during 20 years of follow-up.[3,12]

Master@Heart is a cohort study including middle-aged men between 45 and 70 years participating in endurance sports, aiming to assess the impact of endurance sports on cardiac and vascular structure and function.[14] Individual training data from wearables used by the subjects were voluntarily uploaded and stored on an electronic data recording platform (TrainingPeaks, Peaksware, Boulder, USA).

For the current study, we included male athletes in whom training data during at least 3 months before or after the clinical evaluation were available. At least 80% of training sessions had to be registered with a chest-worn heart rate monitor. Given the design of Master@Heart, only men were included. All participants gave written informed consent. Both studies were approved by the ethics committee research of University Hospitals Leuven (S57241 and S61336) and by the Alfred Hospital Ethics Committee (333/15).

Cardiopulmonary exercise test

All participants underwent an incremental cardiopulmonary exercise test on a cycle ergometer to assess peak oxygen consumption (VO_2peak).[12,14] Initial workload was set at 60 W and increased by 30 W per minute until exhaustion. Respiratory gas exchange was analysed using a breath-by-breath open-circuit ergo-spirometry system. VO_2peak was determined as the highest mean VO_2 measured over 30 s.

Cardiac magnetic resonance

Resting cardiac magnetic resonance (CMR) imaging was performed to assess cardiac systolic and diastolic structure and function.[3,12] All CMR scans were performed on a 1.5 or 3 T magnetic resonance imaging scanner (Philips Medical Systems, Best, The Netherlands; Siemens Healthineers, Erlangen, Germany), equipped with a dedicated cardiac coil and electrocardiographic gating.

Short-axis steady-state free procession cine sequences covering the left and right ventricles were performed with a slice thickness of 8 mm and an interslice gap of 2 mm, according to standardized Society for Cardiovascular Magnetic Resonance (SCMR) recommendations.[15] Cardiac ventricular volumes, function, and left ventricular (LV) mass were quantified using analysis software (suiteHEART®, Version 4.0.6, Neosoft, Pewaukee, WI, USA), according to standardized SCMR recommendations.[15] The software automatically defined the endo- and epicardial LV and endocardial right ventricular (RV) contours and was manually adjusted if the tracking was suboptimal. Trabeculae and papillary muscles were included in the LV cavity.

End-diastolic and end-systolic volumes (EDV/ESV) for the right and left ventricles were indexed by the body surface area, yielding the right ventricular end-diastolic volume index (RVEDVi), right ventricular end-systolic volume index (RVESVi), left ventricular end-diastolic volume index (LVEDVi), and left ventricular end-systolic volume index (LVESVi). The LV mass index (LVMi) was calculated as the difference between epicardial and endocardial volumes at end-diastole multiplied by myocardial density (1.05 g/ml) and indexed to the body surface area.

Training load

All participants were asked to share their recorded training data collected using a chest-worn heart rate monitor for at least 3 months before or after the clinical evaluation. Data were stored on an electronic data recording platform (TrainingPeaks, Peaksware, Boulder, USA). Raw data files were exported and analysed using a custom-developed code in R (R Core Team, Vienna, Austria) as previously described.[8] In short, training files were systematically screened for duplicates, erroneous data, corrupted files, and activities shorter than 1 min. Files were excluded if they met any of the following criteria: average speed of >65 km/h, missing date of activity, unidentified activity, or unclear sport type.

Preference was given to using the 3-month training data prior to clinical evaluation. If insufficient pre-evaluation data were available, data from the 3 months following the evaluation were used. The validity of this approach was confirmed by comparing the agreement of training data from both pre- and post-evaluation in a subset of participants who had data available for both time frames.

For each participant, the total duration and number of training sessions over the 3-month period were calculated. The maximum heart rate (MaxHR) was individually determined using the training data, as previously described.[8] To calculate individualized training intensity, five personalized heart rate zones were established:

- Zone 1: 50%–59% of MaxHR
- Zone 2: 60%–69% of MaxHR
- Zone 3: 70%–79% of MaxHR
- Zone 4: 80%–89% of MaxHR
- Zone 5: 90%–100% of MaxHR

The eTRIMP score was calculated as the sum of the time spent in each heart rate zone multiplied by a corresponding weighing factor (Zone 1 = 1, Zone 2 = 2, Zone 3 = 3, Zone 4 = 4, and Zone 5 = 5).[11,16]

Since Zones 1 and 2 represent physiologically similar low-intensity training levels, they were combined in the analyses (Zone 1&2).

For comparison, exercise intensity in both groups was also calculated as MET, based on the speeds determined by the Global Positioning System and/or speed monitors during exercise. Calculations were made using the 2024 Adult Compendium of Physical Activities (https://pacompendium.com/).[17]

Statistics

Basic statistical analyses were performed using GraphPad Prism (GraphPad Software, San Diego, USA). Data are presented as median and interquartile range (IQR) unless otherwise specified. Pearson's correlations were calculated to examine the associations between training duration, number of sessions, eTRIMP, and CMR-derived cardiac measures. Normality of the data was confirmed using the Shapiro–Wilk test.

Participants were grouped by quartiles, and differences between groups were compared using analysis of variance (ANOVA) with Tukey's honestly significant difference (HSD) *post hoc* correction to compare means. Outliers were identified and excluded using the robust regression and outlier removal method in GraphPad Prism.[18] A two-tailed *P*-value of <.05 was considered statistically significant.

To identify the primary training characteristics associated with cardiac volumes while accounting for the interdependencies among training variables, two advanced statistical methods were used: partial least squares (PLS) regression and Extreme Gradient Boosting (XGBoost). Residual diagnostics were evaluated during the PLS and XGBoost modelling to ensure basic assumptions were met, including the absence of local bias and heteroscedasticity.

Partial least squares

PLS regression was employed to address multicollinearity among predictor variables.[19] The model included absolute and relative durations spent in training Zones 1&2, 3, 4, and 5, as well as age, as predictor variables. PLS constructs latent factors (LFs) as linear combinations of these predictors to maximize covariance with the outcome variable, in this case, a CMR-derived cardiac index.

Instead of using the original predictors, the derived LFs were then used for model estimation. The number of LFs constructed for the final PLS models was the number that managed to explain a substantial proportion of the variation in predictors and outcomes while not differing significantly from the model with the lowest predicted residual sum of squares.

Wold's variable importance in projection (VIP) scores were calculated for each PLS model to evaluate the contribution of each predictor to the LF construction. Predictors with a VIP of >1.2 were considered influential.

Extreme Gradient Boosting

XGBoost, a supervised machine learning algorithm based on decision trees, was used to explore the relationship between training characteristics and CMR measurements.[20] This algorithm builds models in a step-by-step manner: it starts by creating an initial decision tree to predict the outcome. The prediction errors (residuals) from this first tree are calculated, and in the next step, another tree is created to correct these errors. This process is repeated iteratively, with each new tree focusing on the remaining errors from the previous tree, until a stopping criterion is met, such as reaching the maximum number of trees or achieving minimal improvement in prediction accuracy.

XGBoost was selected for its capability to perform multivariable non-linear regression and its established efficacy in analysing small datasets. In this study, the model was trained using the entire cohort since the aim was to explore associations between training characteristics and CMR-derived indices in athletes, rather than developing a generalized predictive model for clinical application. Due to the limited dataset size, data splitting was not performed.

The model parameters were set to 300 estimators and a learning rate of .02, selected to balance minimizing root mean square error and preventing overfitting. All other hyperparameters were kept at default values. The same training characteristics used in the PLS model were included without standardization, as XGBoost can naturally handle features with varying scales. Two models were trained: one with age as a predictor and one without.

To understand the influence of each training characteristic on CMR measurements, Shapley Additive Explanation (SHAP) values were calculated.[21] SHAP values measure the contribution of each predictor by systematically varying the input values and observing the change in the model's prediction. This approach provides insight into both the importance of each variable and the nature of its relationship with the CMR outcomes.

Results

Demographics

Out of 255 athletes evaluated for eligibility, 198 male athletes had at least 3 months of training data available, including training duration. Among them, 151 athletes recorded more than 80% of their total training time using a chest-worn heart rate monitor (*Table 1*). Master@Heart athletes were older and had a higher body mass index compared to the younger Pro@Heart athletes.

Training characteristics

Training duration ($r = .85$), number of sessions ($r = .88$), and eTRIMP ($r = .85$) demonstrated strong and significant correlations (all $P < .05$; see Supplementary data online, *Figure S1*) in the 3-month period before and after the clinical evaluation.

Pro@Heart athletes engaged in more training sessions [77 (64–99) vs 43 (31–60) sessions] and spent more total training time [169 (127–209) vs 78 (49–114) hours] compared to Master@Heart athletes over the 3-month period ($P < .05$ for both; *Table 1*). In addition, the total training load, as measured by eTRIMP, was significantly higher in Pro@Heart athletes [23 129 (17 880–28 305) vs 12 620 (7168–17 607) arbitrary units (AU); $P < .05$] compared to Master@Heart athletes.

Based on the speed at which exercise was performed, MET values were calculated from the training data (recorded speed) to estimate the average absolute intensity of exercise training for both Pro@Heart and Master@Heart athletes. Notably, all athletes in this study regularly exercised at intensity levels far exceeding those typically used to define moderate-intensity exercise (>6 METs) or intense exercise (>9 METs), highlighting the fact that exercise considered light-to-moderate for well-trained athletes would be considered high-intensity exercise in most medical literature.

Pro@Heart athletes trained at a significantly higher average absolute exercise intensity, measured as METs (14.8 ± 2.4 METs; mean ± standard deviation), compared to Master@Heart athletes (10.0 ± 2.2 METs, $P < .05$; *Figure 1A*). Despite this higher absolute intensity, Pro@Heart athletes spent a larger percentage of their total training time in lower heart rate zones (Zone 1&2) compared to Master@Heart athletes (47.7 ± 13.7% vs 34.0 ± 17%; $P < .05$; *Figure 1B*; see Supplementary data online, *Table S1*). In contrast, Master@Heart athletes devoted more time to training in higher heart rates, particularly Zone 4 (23.6 ± 16.4% vs 15.4 ± 7.7%; $P < .05$; *Figure 1B*; see Supplementary data online, *Table S1*), indicating higher relative intensity. These findings illustrate that while Pro@Heart athletes train at higher absolute exercise intensities, a greater proportion of their training is performed at lower heart rate zones, reflecting a distribution skewed towards lower-intensity efforts despite higher overall training capacity. In contrast, Master@Heart athletes engage in a relatively higher proportion of moderate-to-high-intensity training.

In absolute terms, Pro@Heart athletes trained longer than Master@Heart athletes in all heart rate zones.

Cardiac imaging

Pro@Heart athletes had larger EDV/ESV in both the left and right ventricles compared to Master@Heart athletes (*Table 1*). Also, Pro@Heart athletes had a higher LVMi [86 (79–92) vs 66 (59–72) g/m^2; $P < .05$] and greater left ventricular ejection fraction (LVEF) [55 (52–57) vs 52 (50–57) %; $P < .05$] compared to older Master@Heart athletes. Right ventricular ejection fraction (RVEF) was not different between the two groups.

Relationship between training load and cardiac remodelling

Quartile analyses

We evaluated the relationship between training load and cardiac remodelling. First, we categorized the athlete population into quartiles

Table 1 Demographics and training characteristics

	Pro@Heart	Master@Heart	All athletes
Demographics			
N (male%)	69 (100)	82 (100)	151 (100)
Age (years)	21 (19–23)	55 (49–60)*	47 (21–56)
Weight (kg)	70.0 (66.2–75.0)	73.0 (68.0–78.3)*	71.5 (67.5–77.0)
Height (cm)	180 (176–186)	178 (174–182)*	179 (175–184)
Body mass index (kg/m^2)	21.6 (20.3–22.6)	23.4 (21.8–24.6)*	22.2 (21.1–23.7)
Body surface area (m^2)	1.89 (1.82–1.96)	1.90 (1.83–2.00)	1.90 (1.83–1.98)
Peak oxygen consumption (ml/kg/min)	70 (64–74)	46 (41–52)*	53 (45–68)
Percentage predicted peak oxygen consumption (%)	160 (150–174)	155 (137–171)*	157 (143–173)
Training characteristics			
Data available 3 months before (%)	60 (87)	50 (61)	110 (73)
Maximal heart rate training (b.p.m.)	197 (191–201)	178 (171–186)*	189 (177–197)
Duration (h)	169 (127–209)	78 (49–114)*	116 (65–175)
Hours per week	13 (10–16)	6 (4–9)*	9 (5–13)
Exercise sessions	77 (64–99)	43 (31–60)*	62 (40–88)
Sessions per week	6 (5–8)	3 (2–5)*	5 (3–7)
Edwards training impulse (AU)	23 129 (17 880–28 305)	12 620 (7168–17 607)*	17 333 (10 102–23 426)
Edwards training impulse/week (AU)	1779 (1375–2177)	970 (551–1354)*	1333 (777–1802)
Time Zone 0 (min)	243 (69–613)	97 (19–374)	157 (40–422)
Time Zone 1&2 (min)	4649 (2921–6229)	1498 (888–2499)*	2605 (1184–4805)
Time Zone 3 (min)	2554 (1805–3521)	1514 (699–2491)*	2035 (1149–2965)
Time Zone 4 (min)	1455 (894–1687)	885 (394–1416)*	1143 (566–1603)
Time Zone 5 (min)	236 (133–365)	121 (56–197)	163 (84–297)
Cardiac measures			
Left ventricular end-diastolic volume index (ml/m^2)	138 (126–155)	111 (104–123)*	123 (110–139)
Left ventricular end-systolic volume index (ml/m^2)	62 (55–73)	53 (47–58)*	57 (50–65)
Left ventricular stroke volume index (ml/m^2)	74 (69–82)	59 (55–65)*	66 (59–75)
Left ventricular ejection fraction (%)	55 (52–57)	52 (50–57)*	54 (50–57)
Left ventricular mass index (g/m^2)	86 (79–92)	66 (59–72)*	74 (64–86)
Left ventricular end-diastolic volume/left ventricular mass (ml/g)	1.63 (1.49–1.79)	1.74 (1.58–1.87)*	1.70 (1.54–1.83)
Right ventricular end-diastolic volume index (ml/m^2)	144 (135–156)	117 (107–128)*	131 (114–146)
Right ventricular end-systolic volume index (ml/m^2)	71 (61–80)	57 (50–64)*	62 (54–74)
Right ventricular stroke volume index (ml/m^2)	75 (57–84)	61 (56–66)*	66 (58–75)
Right ventricular ejection fraction (%)	51 (49–55)	52 (48–55)	52 (48–55)

Demographic data, training characteristics measured by chest-worn heart rate monitors and cardiac magnetic resonance characteristics for both Pro@Heart and Master@Heart athletes. Data expressed as median (interquartile range). AU, arbitrary units.
*$P < .05$.

based on total exercise duration. We found a significant stepwise increment in all measures of cardiac remodelling (LVEDVi, LVESVi, LVMi, RVEDVi, and RVESVi) across each quartile of exercise dose (*Figure 2*). The same pattern was observed across eTRIMP quartiles, with increases in LVEDVi, LVESVi, RVEDVi, RVESVi, and LVMi corresponding to greater eTRIMP values (*Figure 3*).

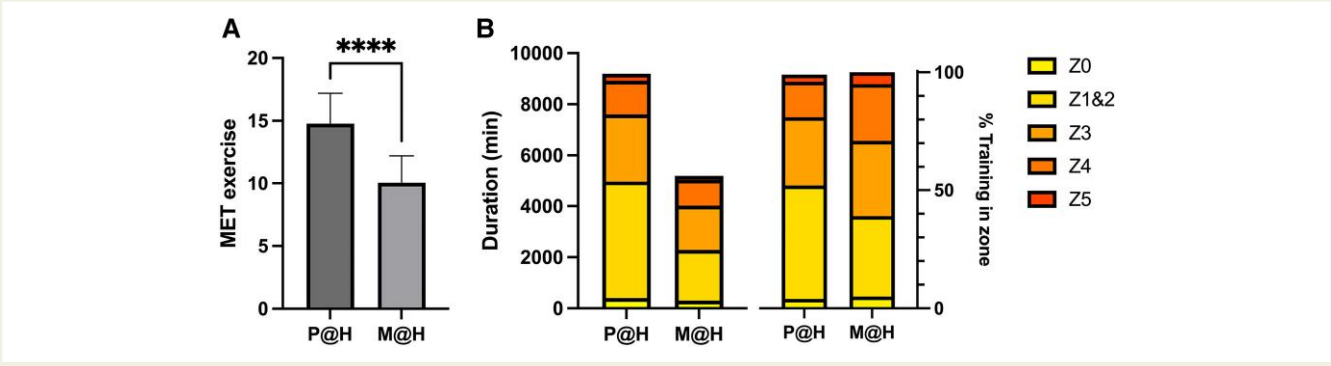

Figure 1 Training intensity distribution in Pro@Heart vs Master@Heart athletes. While Pro@Heart athletes trained at a higher absolute intensity, they spent a larger proportion of their training in lower-intensity zones compared to Master@Heart athletes. In contrast, Master@Heart athletes allocated more time to higher-intensity zones, highlighting differing training strategies between the groups. (*A*) Average metabolic equivalent of task per training session calculated based on average speed recorded during training sessions. (*B*) Duration displayed as minutes and percentage of training time in each training zone. Mean ± standard deviation; ****$P < .0001$

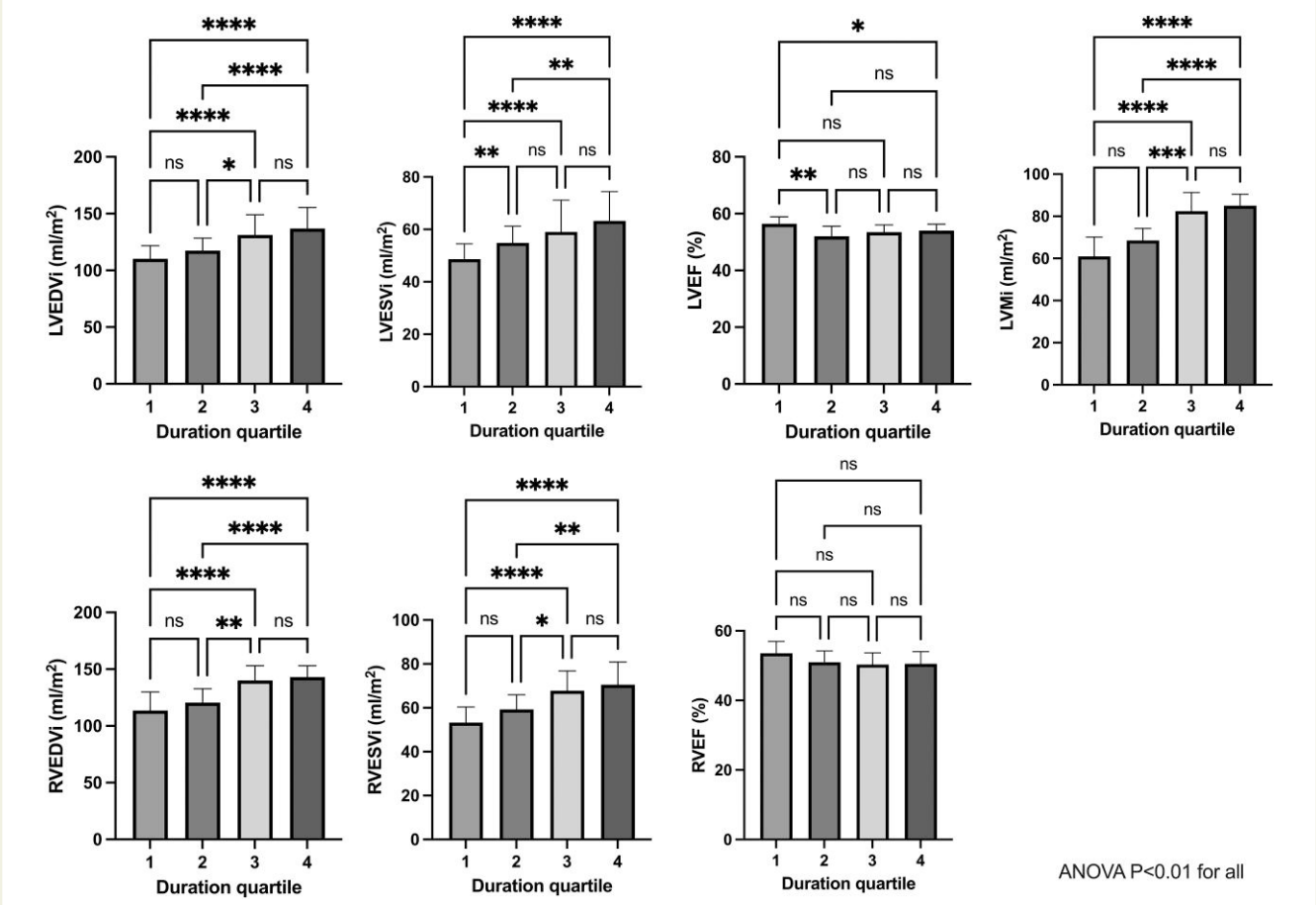

Figure 2 Exercise duration measured by chest-worn heart rate monitors and exercise-induced cardiac remodelling in four quartiles of athlete exercise duration. Indexed left and right ventricular end-diastolic volume and end-systolic volume, left and right ventricular ejection fraction, and indexed left ventricular mass. An analysis of variance with Tukey's honestly significant difference *post hoc* correction was performed to compare the means across groups. Median ± interquartile range; *$P < .05$, **$P < .005$, ***$P < .0005$, and ****$P < .0001$ between groups

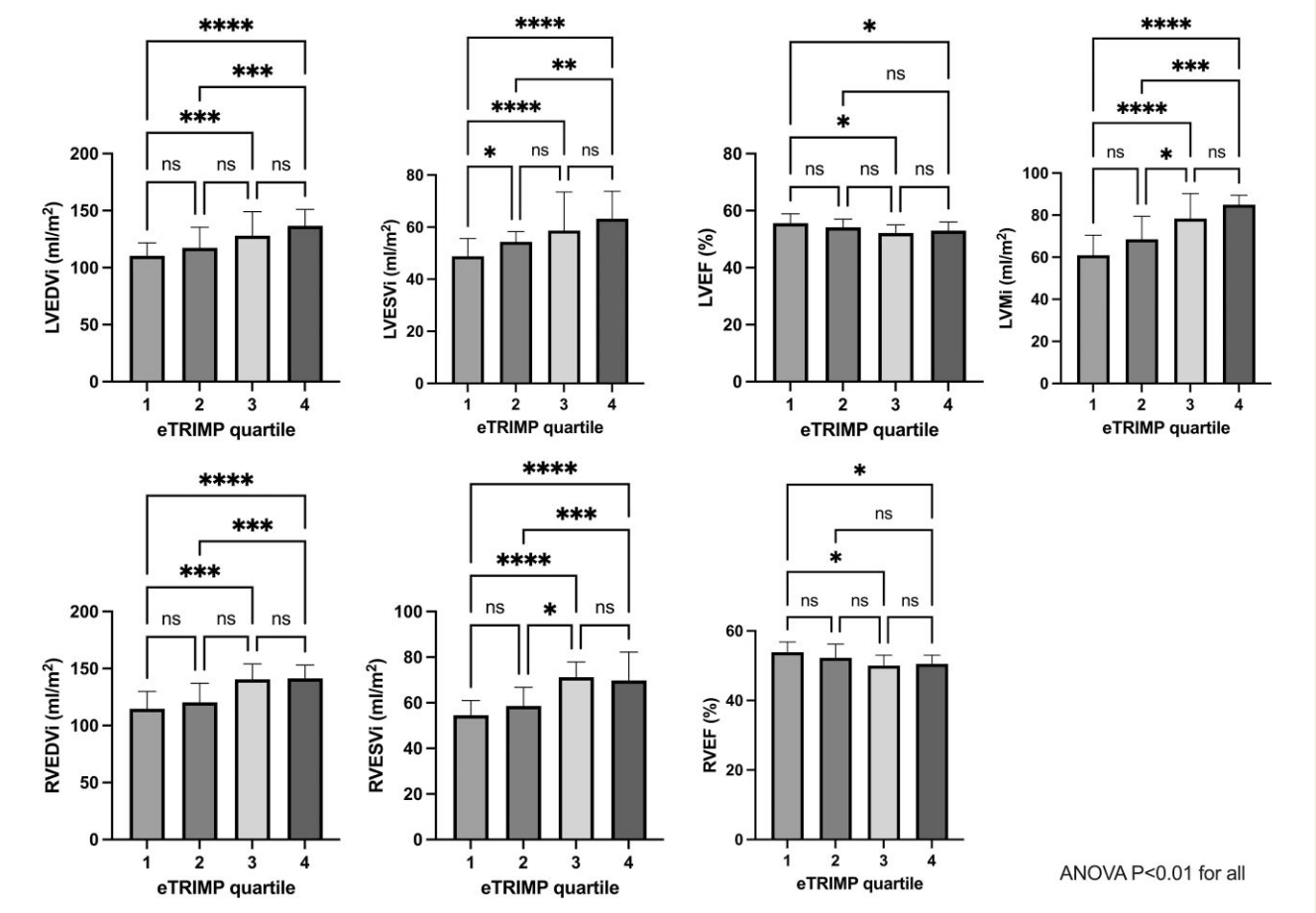

Figure 3 Edwards training impulse measured by chest-worn heart rate monitors and exercise-induced cardiac remodelling in four quartiles of athlete Edwards training impulse. Indexed left and right ventricular end-diastolic volume and end-systolic volume, left and right ventricular ejection fraction, and indexed left ventricular mass. An analysis of variance with Tukey's honestly significant difference *post hoc* correction was performed to compare the means across groups. Median ± interquartile range; *$P < .05$, **$P < .005$, ***$P < .0005$, and ****$P < .0001$ between groups

Correlation analyses

Significant correlations were found between total training duration and several cardiac parameters: LVEDVi ($r = .51$), LVESVi ($r = .50$), LVMi ($r = .53$), RVEDVi ($r = .47$), and RVESVi ($r = .45$). In contrast, both LVEF ($r = -.18$) and RVEF ($r = -.19$) showed significant inverse correlations. Comparable results were observed with eTRIMP (*Table 2*). The time spent in Zone 1&2 showed the strongest correlation with LVEDVi ($r = .49$), LVESVi ($r = .45$), left ventricular stroke volume index (LVSVi) ($r = .41$), LVMi ($r = .52$), RVEDVi ($r = .47$), and RVESVi ($r = .42$), compared to time spent in higher-intensity zones. Notably, only time in Zone 3 showed a positive correlation with LVEF. After adjustment of age, total exercise duration remained significantly associated with increased LVEDVi, LVESVi, LVMi, RVEDVi, and RVESVi, while LVEF was inversely related to training volume (*Table 3*).

Partial least squares results

To investigate the relation between training characteristics and cardiac remodelling in more detail, we applied PLS analysis using time and percentage of time spent in different heart rate zones (see Supplementary data online, *Table S2*).

- *With age included*: age was the dominant influential feature (VIP of >1.2) for nearly all CMR measurements except RVEF. Minutes in Zone 1&2 remained influential for LV and RV volumes, while time spent in Zone 3 was influential for predicting LVESVi, RVESVi, LVEF, and RVEF. The PLS models explained 4.6%–47.8% of the variance in CMR indexes (see Supplementary data online, *Table S2*).
- *Without age*: minutes in Zone 1&2 emerged as the most influential predictor (VIP of >1.2) of nearly all CMR measurements except LVEF and RVEF, for which Zone 3 time was most important. The models explained 4.0%–29.8% of the variance (see Supplementary data online, *Table S3*).

Extreme Gradient Boosting results

Using SHAP values from the XGBoost supervised machine learning algorithm, minutes spent in Zones 1&2 and 3 emerged as the most influential training characteristics for nearly all CMR measurements (*Figure 4*). For each CMR outcome, we identified the relative importance of each training load metric; from top to bottom, the overall influence of each training load metric on the final prediction gradually decreases. The direction and consistency of these relationships are illustrated by uniform colour gradients, with blue representing negative associations and red representing positive associations between the training characteristics and the CMR outcomes. For example, an increase in time spent in Zone 1&2 pushes the prediction to higher LVEDVi.

Table 2 Correlation matrix

	Left ventricular end-diastolic volume index	Left ventricular end-systolic volume index	Left ventricular stroke volume index	Left ventricular ejection fraction	Left ventricular mass index	Right ventricular end-diastolic volume index	Right ventricular end-systolic volume index	Right ventricular stroke volume index	Right ventricular ejection fraction
Duration (min)	**.51***	**.5***	**.37***	**−.18***	**.53***	**.47***	**.45***	**.33***	**−.19***
Sessions	**.45***	**.4***	**.36***	−.09	**.47***	**.39***	**.32***	**.33***	−.06
Edwards training impulse (AU)	**.46***	**.47***	**.31***	**−.2***	**.48***	**.43***	**.43***	**.29***	**−.21***
Time in									
Zone 1&2	**.49***	**.45***	**.41***	−.1	**.52***	**.47***	**.42***	**.35***	−.12
Zone 3	**.34***	**.39***	**.18***	**−.24***	**.34***	**.32***	**.36***	**.19***	**−.24***
Zone 4	**.23***	**.25***	.12	−.16	**.24***	**.22***	**.24***	.13	−.14
Zone 5	**.23***	**.18***	**.22***	.03	**.25***	**.2***	**.17***	**.19***	−.02

Correlation matrix (r) of left ventricle and right ventricle function compared to training load measured by chest-worn heart rate monitors. Indexed left and right ventricular end-diastolic volume and end-systolic volume, left and right ventricular ejection fraction, and indexed left ventricular mass. AU, arbitrary units.
*Highlighted $P < .05$.

Table 3 Association between cardiac magnetic resonance-measured cardiac volumes and function and training characteristics

		Effect size of training characteristics on cardiac magnetic resonance measures						
Training characteristic	Total training (per 1000 min)	**1.69 ± .39 (*P* < .0001)***	**1.28 ± .24 (*P* < .0001)***	**−.37 ± .09 (*P* = .0003)***	**1.19 ± .25 (*P* < .0001)***	**1.56 ± .42 (*P* < .0001)***	**1.18 ± .23 (*P* < .0001)***	−.30 ± .11 (*P* = .0099)
	Minutes in Zone 1&2 (per doubling)	**5.40 ± 1.21 (*P* < .0001)***	**3.80 ± .79 (*P* < .0001)***	−1.00 ± .32 (*P* = .0022)	**3.72 ± .82 (*P* < .0001)***	**5.28 ± 1.34 (*P* = .0001)***	**3.71 ± .93 (*P* = .0001)***	−.83 ± .37 (*P* = .026)
	Minutes in Zone 3 (per doubling)	**5.72 ± 1.59 (*P* = .0004)***	**4.68 ± 1.01 (*P* < .0001)***	**−1.55 ± .41 (*P* = .0002)***	3.69 ± 1.08 (*P* = .0008)	5.77 ± 1.74 (*P* = .0011)	**4.76 ± 1.19 (*P* < .0001)***	−1.42 ± .47 (*P* = .0028)
	Minutes in Zone 4 (per doubling)	1.85 ± 1.78 (*P* = .30)	2.05 ± 1.15 (*P* = .077)	−.90 ± .45 (*P* = .048)	1.44 ± 1.20 (*P* = .23)	2.05 ± 1.93 (*P* = .29)	2.27 ± 1.33 (*P* = .090)	−.82 ± .51 (*P* = .11)
	Minutes in Zone 5 (per doubling)	1.17 ± 1.18 (*P* = .32)	1.05 ± .77 (*P* = .17)	−.46 ± .30 *P* = .13	.74 ± .80 (*P* = .36)	.68 ± 1.29 (*P* = .60)	.62 ± .89 (*P* = .49)	−.18 ± .34 (*P* = .59)
		Left ventricular end-diastolic volume index (ml/m²)	**Left ventricular end-systolic volume index (ml/m²)**	**Left ventricular ejection fraction (%)**	**Left ventricular mass index (g/m²)**	**Right ventricular end-diastolic volume index (ml/m²)**	**Right ventricular end-systolic volume index (ml/m²)**	**Right ventricular ejection fraction (%)**
		Cardiac magnetic resonance variable						

Multivariable-adjusted association between cardiac magnetic resonance-derived cardiac left ventricle and right ventricle function and training characteristics. Values represent effect sizes ± standard error, which indicate how much the cardiac magnetic resonance index changes for a given increase or for a doubling in the training variable.
*Significant *P*-value after correcting for multiple testing, for which the threshold for significance was .00062 (=.05/81 tests).

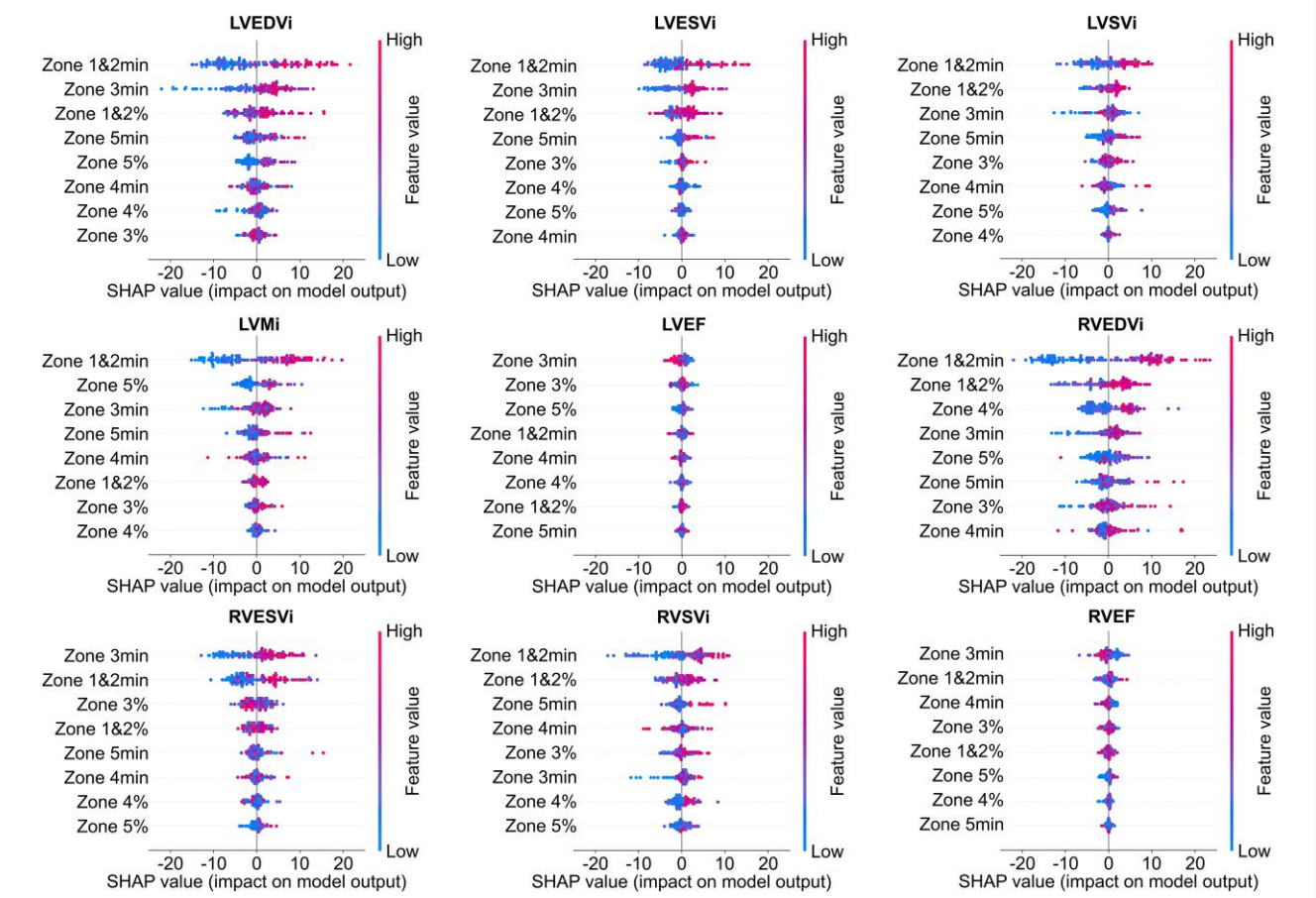

Figure 4 Explainability analysis of the Extreme Gradient Boosting model for each cardiac magnetic resonance measurement, showing the effect of training characteristics on cardiac metrics. Shapley Additive Explanation values indicate the impact of each training characteristic for individual participants. Red indicates a higher value in the training characteristic for the study participant, while blue indicates a lower value. The sign of the Shapley Additive Explanation value shows the direction of the effect: positive values indicate that a higher training characteristic increases the cardiac magnetic resonance metric, while negative values indicate that it decreases the cardiac magnetic resonance metric. Training characteristics are ranked by influence from most to least impactful on the model's output

- Increased time in Zone 1&2 was associated with higher LVEDVi, LVESVi, LVMi, LVSVi, and RVEDVi.
- Time in Zone 3 was the strongest predictor of LVEF and RVESVi.
- Relative duration in Zone 1&2 consistently ranked among the top three predictors, while for LVMi, absolute duration in Zone 5, and for RVEDVi, absolute duration in Zone 4 also emerged as important predictors.

When age was included, it emerged as the dominant predictor for nearly all CMR measurements except RVEF (see Supplementary data online, *Figure S2*). Younger age was associated with larger LVEDVi, higher LVMi, and greater RV volumes, as well as higher LVEF. Nonetheless, time in Zones 1&2 and 3 remained important contributors, particularly for LVESVi, RVESVi, and biventricular ejection fractions. A summary of the SHAP analysis is provided in *Table 4*.

Discussion

This study is the first to objectively assess the relationship between cardiac adaptation to endurance sports and training load using heart rate monitors, offering new insights for clinical cardiologists. Contrary to our initial hypothesis, we found that total training duration, not exercise intensity, was the predominant determinant of cardiac remodelling (*Structured Graphical Abstract*). This challenges the conventional focus on high-intensity exercise as the main driver of cardiac adaptation. The limited impact of high-intensity training (Zones 4 and 5) likely reflects the shorter time spent in these zones. Additionally, age significantly influenced cardiac remodelling, although its effect was challenging to isolate due to younger athletes engaging in more extensive training. These findings highlight the need to evaluate total training volume alongside intensity when assessing cardiac remodelling in endurance athletes, informing more nuanced risk stratification and management in sports cardiology.

The relationship between cardiac adaptation and high-level endurance sports has primarily been evaluated using exercise questionnaires,[5–7] but accurate quantification poses a significant challenge when relying on self-reported questionnaires to estimate the duration and intensity of exercise. Conventionally, the speed of exercise is employed to estimate METs as a proxy for exercise intensity. However, as we identified in our cohort of elite athletes, the speeds at which the athletes typically train would all be considered high intensity, and all nuance

Table 4 Summary of the most important training characteristics per cardiac magnetic resonance measurement based on the Shapley Additive Explanation values from Extreme Gradient Boosting models

	Left ventricular end-diastolic volume index	Left ventricular end-systolic volume index	Left ventricular ejection fraction	Left ventricular mass index	Left ventricular stroke volume index	Right ventricular end-diastolic volume index	Right ventricular end-systolic volume index	Right ventricular stroke volume index	Right ventricular ejection fraction
Without age									
Most important training characteristic	Zone 1&2$_{min}$ Zone 3$_{min}$ Zone 1&2$_{\%}$	Zone 1&2$_{min}$ Zone 3$_{min}$ Zone 1&2$_{\%}$	Zone 3$_{min}$	Zone 1&2$_{min}$ Zone 5$_{\%}$	Zone 1&2$_{min}$ Zone 1&2$_{\%}$	Zone 1&2$_{min}$ Zone 1&2$_{\%}$ Zone 4$_{min}$	Zone 3$_{min}$ Zone 1&2$_{min}$	Zone 1&2$_{min}$	Zone 3$_{min}$

The table lists the predictors that showed a clear relation with the cardiac magnetic resonance measurement. Zone 1&2$_{min}$, training time in minutes in Zone 1&2; Zone 3$_{min}$, training time in minutes in Zone 3; Zone 4$_{min}$, training time in minutes in Zone 4; Zone 1&2$_{\%}$, percentage of training time in Zone 1&2; Zone 5$_{\%}$, percentage of training time in Zone 5.

regarding training load would be diluted or lost (*Figure 1A*). The elite athletes in our study could maintain higher speeds at a relatively low percentage of their MaxHR and/or VO_2peak.[9] This observation also explains why elite young Pro@Heart athletes had a higher frequency of activity within lower heart rate zones compared to the older Master@Heart athletes (*Figure 1B*).

We used the actual training load derived by heart rate monitors using a chest strap to accurately quantify training load characteristics in all athletes. We chose to focus on data derived from 3-month training having previously demonstrated that training load calculated over 3 months closely approximated annual training load data.[8] In addition, we showed that the timing of the 3-month period is not particularly important, with excellent agreement when training load was assessed prior to or after the cardiac evaluation (see Supplementary data online, *Figure S1*).

Our finding that cardiac remodelling is predominantly determined by duration, rather than intensity, might suggest that quantification of exercise load could be simplified to inquiry about exercise duration. However, exercise intensity was somewhat consistent among this highly conditioned cohort, whereas exercise intensity may be more important when comparing between populations with greater variance in fitness levels. Moreover, our findings also emphasize the need for objective measurement of training duration, as our previous work has demonstrated that self-reporting of exercise duration is inaccurate.[8]

There is a logical basis for the observation that the total duration of exercise exposure, rather than exercise intensity alone, is the primary driver of cardiac remodelling. Cardiac imaging studies consistently demonstrate that ventricular EDV, representing volume load, is maximal during low-to-moderate-intensity exercise and decreases slightly at peak exercise intensity.[22] This suggests that prolonged exposure to sub-maximal heart rates, where ventricular volume is maximized, may be a potent and underappreciated stimulus for cardiac remodelling.

Our finding that exercise duration outweighs intensity carries important clinical implications. Current guidelines for sport participation often prioritize exercise intensity, while exercise duration is given less weight. Our data suggest that both exercise intensity and total exercise duration should be jointly considered when providing training recommendations, particularly for athletes and patients at risk for cardiac remodelling. This dual consideration could enhance individualized exercise prescriptions, improving both safety and performance outcomes.

It is known that RV remodelling appears more sensitive to the intensity of exercise than LV remodelling.[23] A 1-year training intervention in previously sedentary individuals demonstrated differential ventricular responses to an incremental exercise programme: the left ventricle exhibited initial concentric remodelling followed by eccentric hypertrophy, while the right ventricle experienced dilation from the onset of the exercise regimen.[24] Our study highlights the importance of using objectively quantified training load to better understand this relationship, revealing that the primary factor influencing RV emptying fraction was the volume of training performed in Zone 3. This emphasizes the necessity of precise training load assessment, as subjective estimates may not capture the nuanced impact of different intensity zones on RV function.

Another critical aspect to consider is the influence of age on cardiac volumes. In our study, older athletes had smaller EDV/ESV. However, interpreting age in isolation is problematic, since age was strongly linked to training load and group allocation: the younger Pro@Heart athletes accumulated substantially higher training exposure than the older Master@Heart athletes. Thus, the observed 'age effect' likely reflects cumulative training load rather than chronological age alone. Genetic predisposition may also contribute to these intergroup differences. Taken

together, these findings highlight the importance of considering both age and training load when assessing cardiac remodelling in athletes to avoid misattributing training-induced adaptations to age-related cardiac decline.

Accurate quantification of training load is essential to determine whether the extent of cardiac remodelling is proportional to the athlete's training exposure. Ventricular dilation and mildly reduced ejection fraction are prevalent conditions among highly trained athletes,[3] and our current study demonstrates that both are associated with greater training, particularly in heart Zones 1&2 and 3. Objective quantification of training load may therefore assist clinicians in discerning physiological adaptation from potentially pathological remodelling, especially when structural changes appear disproportionate to training volume, which may suggest subclinical cardiomyopathy. Although we did not study the relationship of training load with arrhythmias, we have previously noted a high prevalence of atrial and ventricular arrhythmias in endurance athletes.[25,26] Similarly, investigators have noted an association between exercise dose and life-threatening arrhythmias in arrhythmogenic cardiomyopathy,[27] and there are data to suggest that higher intensity of exercise training may be particularly deleterious.[28] Using our current quantitative training load approach, we demonstrate robust associations between training duration, intensity, and cardiac remodelling, providing a foundation for future studies exploring the link between exercise training characteristics and arrhythmias.

Limitations

This cross-sectional study provides valuable insights into the relationship between training load and cardiac remodelling but cannot establish causality due to its observational design and lack of longitudinal follow-up. Nevertheless, the use of objective training load quantification via heart rate monitors enhances data reliability and reduces recall bias.

We combined data from two distinct cohorts: young elite (Pro@Heart) and older predominantly recreational (Master@Heart) athletes. Despite differences in age and competition level, both groups demonstrated excellent VO_2peak values, confirming their athletic status. Importantly, the relationship between training load and cardiac remodelling remained robust after adjusting for age, supporting the generalizability of our findings. However, combining cohorts may have masked subtle cohort-specific effects. Future studies with larger sample sizes should aim to analyse these cohorts separately to better explore potential age- or cohort-dependent remodelling patterns.

Wearable data were provided by the athletes themselves, and not all training sessions may have been captured. However, any underestimation of training load was likely non-differential and consistent across participants. Although training load was quantified over the 3 months preceding cardiac imaging, we cannot exclude the influence of longer-term or earlier training history on the observed cardiac remodelling. However, previous validation of this time frame, along with consistent training load patterns before and after evaluation, supports its use as a proxy for habitual training exposure in this population.[8] Future longitudinal studies with detailed lifetime training histories will be needed to better disentangle chronic vs acute effects of training on cardiac structure. From an analytical perspective, the PLS and XGBoost models may have been overfitted as a consequence of the relatively low sample size. As such, the variable importance rankings should be interpreted cautiously and confirmed in larger cohorts. Nevertheless, our observations in the PLS and XGBoost modelling aligned with the multivariable-adjusted linear regression models, suggesting robustness of the associations observed. Finally, this study included only male athletes. Further research is needed to assess whether these findings apply to female athletes.

Conclusions

In well-trained athletes, total exercise duration was positively correlated with LV and RV volumes, especially the time spent in lower heart rate zones. Younger athletes showed larger volumes, likely due to higher training durations. Categorization of exercise intensity according to chart-based estimation of METs had limited utility in this population as most exercise would be interpreted as high intensity. These findings highlight how direct quantification of exercise intensity and duration can provide new insights into cardiac remodelling assessments.

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Supplementary data

Supplementary data are available at *European Heart Journal* online.

Declarations

Disclosure of Interest

All authors declare no disclosure of interest for this contribution.

Data Availability

The data that support the findings of this study are available upon reasonable request.

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Ethical Approval

Both studies were approved by the ethics committee research of University Hospitals Leuven (S57241 and S61336) and by the Alfred Hospital Ethics Committee (333/15).

Pre-registered Clinical Trial Number

The pre-registered clinical trial numbers are as follows: Master@Heart (NCT03711539) and Pro@Heart (NCT05164328, ACTRN12618000716268).

Appendix

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