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ORIGINAL ARTICLE

Lack of cardiac remodelling in elite endurance athletes: an unexpected and not so rare finding

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Abstract

Purpose Endurance elite athletes are expected to present a cardiac remodelling, characterized by eccentric hypertrophy (EH), may be associated with higher sportive performances. However, not all can present a cardiac remodelling.
The study aimed to identify endurance athletes without cardiac remodelling characterizing their physiologic and clinical features.

Methods We studied 309 endurance athletes (cycling, rowing, canoeing, triathlon, athletics, long-distance swimming, cross-country skiing, mid-long distance track, pentathlon, biathlon, long-distance skating and Nordic-combined) examined during period of training, by clinical evaluation, ECG, echocardiogram and exercise-stress test. Sport career achievements (Olympic\World championship medals or national\world records) were recorded.

Results EH was found in most of athletes, (n = 126, 67% of males; n = 85, 68.5% of females). A significant proportion,, exhibited normal geometry (NG) (n = 59, 31.3% in males; n = 39, 31.4% in females). At stress test, significant differences between EH and NG athletes were found in peak power (317.1 ± 71.2W in NG vs. 342.2 ± 60.6W in EH, p = 0.014 in males and 225.1 ± 38.7W in NG vs. 247.1 ± 37W in EH, p = 0.003 in females), rest heart rate (66.1 ± 13 in NG vs. 58.6 ± 11.6 in EH, p = 0.001 in males and 68 ± 13.2 in NG vs. 59.2 ± 11.2 in EH, p = 0.001 in females) with similar ventricular extrasystoles (p = 0.363 in males and p = 0.492 in females). However, no significant differences in athletic achievements were registered.

Conclusion Our study demonstrates a relatively high prevalence of NG in endurance athletes, in addition to the expected EH. Athletes with NG perform worse in exercise-stress test and exhibit some less advantageous functional heart characteristics. However, the type of heart geometry is not associated with negative clinical findings.

Keywords Athlete · Remodelling · Endurance · Training

Abbreviations

BMI	Body mass index
BSA	Body surface area
CH	Concentric hypertrophy
CMR	Cardiac magnetic resonance
CR	Concentric remodelling
EF	Ejection fraction
EH	Eccentric hypertrophy
LAD	Left atrial diameter
LAV	Left atrial volume
LV	Left ventricular
LVEDD	Left ventricle end-diastolic diameter
LVEDV	Left ventricle end-diastolic volume
LVESV	Left ventricle end-systolic volume
LVM	Left ventricular mass
LVMi	Left ventricular mass indexed
NG	Normal geometry

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PW	Pulsed wave Doppler
RAA	Right atrial area
RVOT	Right ventricle outflow tract
RWT	Relative wall thickness
TDI	Tissue Doppler imaging
TTE	Transthoracic echocardiography

Introduction

Cardiac remodelling in athletic individuals, generally referred to as the "athlete's heart," is the combination of structural and functional cardiac changes in response to chronic exercise training, which has a profound impact on performance (Pelliccia et al. 2021). The physiologic adaptations help to optimize cardiac performance, to improve oxygen delivery to working muscles, and to enhance athlete's functional capacity. In highly-trained athletes, differences in cardiac remodelling have been described according to sport discipline (Mihl et al. 2008).

In endurance sports, which typically showcase the "athlete's heart" at its highest expression, the prolonged and sustained hemodynamic load associated with regular training results in an increase in left ventricular (LV) cavity size\LV mass indexed (LVMi) ratio with normal relative (RWT) wall thickness, which allows for an increased stroke volume, and cardiac output to the working muscles (Brown et al. 2020). Indeed, the enhanced diastolic function, characterized by higher ventricular filling and lower end-diastolic pressures, allows for a more efficient and rapid filling, making it possible to maintain a higher cardiac output even at high heart rates during strenuous exercise (Dalos et al. 2022; Pelliccia et al. 2018; Arbab-Zadeh et al. 2014).

Actually, eccentric (EH) and concentric hypertrophy (CH) represent divergent cardiac adaptations in athletes. However, this classification can be sometimes misleading since the evidence of overlapping phenotypes according to heterogeneity in sporting disciplines, gender, different training programmes and volumes and genetic response to exercise (Martinez et al. 2021; Naylor et al. 2008; Rossi et al. 2024).

EH is a more typical characteristic of endurance athletes and emerges from chronic volume overload by widening the ventricular cavities while maintaining wall thickness, thus increasing stroke volume and cardiac output, which are essential for sustained aerobic performance (Oxborough et al. 2010). In contrast, CH is seen in strength or power athletes, where high-resistance training induces pressure overload. This leads to an increase in myocardial wall thickness without significant chamber enlargement (Maron et al. 2006). This adaptation optimizes the heart for high-force, short-duration efforts, improving systolic function (Finocchiaro et al. 2017). Both responses are initially adaptive and reversible, however, excessive or unbalanced dilation or hypertrophy can mask co-existent pathological conditions. In addition, the autonomic resetting with enhanced vagal tone contributes to maintain a larger stroke volume during exercise and ultimately improves cardiac performance (Ellison et al. 2012).

Furthermore, the athlete's heart, within all its declinations, is directly related to improved physical performance. EH in endurance athletes may result in larger ventricular volumes, facilitating greater stroke volume and increased cardiac output thus increasing oxygen-carrying capacity, directly contributing to a high peak oxygen uptake ($\dot{V}O_2$ peak), as highlighted by Fagard et al. (1996) who found a significant relationship between LV mass and $\dot{V}O_2$ max in cyclists. Similarly, CH in strength athletes, although less directly linked to peak $\dot{V}O_2$, improves cardiac efficiency during high-intensity, short-duration efforts, as shown by Arbab-Zadeh et al. (2014) for what concerns peak power. Both forms of remodelling optimize the heart's performance based on the specific needs of different sports disciplines.

Actually, while it is widely accepted that endurance sports are commonly associated with the described physiologic adaptations, however, it is recognized that not all athletes exhibit such cardiac remodelling (Boraita et al. 2022), despite being able to successfully participate in endurance-type events. In fact, a recent work by Boraita et al. (2022) on 3,282 elite athletes practicing different sports revealed that the majority (85.4%) had normal LV geometry (NG), with a smaller proportion (13.4%) showing EH. Notably, concentric remodelling (CR) and CH were rare, observed in less than 0.8% of athletes. However, definitive data on endurance elite athletes are lacking at the moment.

In the present analysis, therefore, we sought to investigate the prevalence and physiologic characteristics of elite endurance athletes that did not show the anticipated cardiac EH remodelling, in order to identify the functional features and possible clinical significance.

Materials and methods

The Institute of Sports Medicine and Science in Rome is the medical division of the Italian National Olympic Committee, where elite athletes undergo medical examinations before the international competitions and Olympics. All elite athletes who competed in major international events in the period from the London 2012 Summer Games to the Beijing 2022 Winter Games were considered for the present analysis.

We studied 309 endurance athletes (59.9% male), mean age 26.7 ± 4.6 years, mostly Caucasians (10, 3.2% were Afro-Caribbean), engaged in a spectrum of different disciplines, according to the ESC guidelines (D'Ascenzi et al.

2017, Di Gioia et al. 2023, Pelliccia et al. 2021): cycling (65, 21%), rowing (53, 17.1%), canoeing 22 (7.1%), triathlon 11 (3.6%), athletics (56, 18.1%), long distance swimming (14, 4.5%), cross-country skiing (28, 9.1%), mid-long distance track (22, 7.1%), pentathlon (6, 1.9%), biathlon (18, 5.8%), long-distance skating (6, 1.9%) and Nordic combined (8, 2.6%).

The cardiovascular (CV) routine evaluation included a complete physical examination, a 12-lead ECG, transthoracic echocardiography (TTE), and a bicycle ergometer maximal exercise test. Athletes were evaluated within three months prior to the Olympic events, during a period of intense training characterized by high and constant training load (with both general and specific-discipline preparations). Athletes in detraining period, loading phase or in tapering (usually 2 to 3 weeks before Olympics, depending from sporting discipline) were excluded from the study.

All athletes were world-class competitors, and a substantial proportion (34%) had consecutively participated in more than one Olympic Game.

We also investigated sport career achievements in terms of Olympic or World championship medals or exceptional performance (previous or still unbeaten national or world record holder).

For each athlete, anthropometric measurements, body mass index (BMI) and body surface area (BSA) were recorded. Body composition and fat mass percentage were measured using Bioelectric Impedance Analysis (BIA 101 Quantum, Akern, Pisa, Italy) using constant sinusoidal current at an intensity of 50 kHz and 400 µA. Blood pressure was measured in a sitting position before exercise testing (Piepoli et al. 2016; Caselli et al. 2016). Standard 12-lead ECGs were recorded and interpreted according to international criteria for athletes (Drezner et al. 2017). The maximal exercise ECG test was conducted on a cycle ergometer; the starting load was 0.5 W/kg, with increase of 0.5 W/kg every two minutes until physical exhaustion, identified as the time when athletes was unable to maintain the required pedalling frequency (70 rpm) despite repetitive encouragement (Caselli et al. 2016).

Images were obtained and interpreted by two different expert sports cardiology consultants (GDG, AP). Intra-observer and inter-observer variability was assessed in a sample of 50 athletes, selecting randomly one every 20 athletes on our database, independently from sex and sporting discipline. Two investigators (GDG and AP) blinded measured the same exam. Both the investigators repeated the analysis 2 days later, blinded to the previous measurements. Interclass correlation coefficients (ICC) were 0.93 for intra-observer and 0.91 for inter-observer agreement. Echocardiography was performed in the left lateral recumbency at rest. Measurements of LV cavity dimensions (left ventricle end-diastolic diameter, LVEDD; left ventricle end-systolic diameter, LVESD), interventricular septum (IVS), posterior free wall thickness (PWT), LV ejection fraction (LVEF), RWTLVMi and indexes of LV diastolic function were obtained according to current recommendations on athletes (Lang et al. 2015).

Specifically, linear internal measurement of LV and wall thickness was performed in parasternal long-axis view, measuring immediately below the level of the mitral valve leaflet, by positioning callipers tracker on the interface between the wall and the pericardium and myocardial wall and the cavity (Lang et al. 2015).

Moreover, LVM was calculated according to M-mode Cube formula $= 0.8\ (1.04\ ([\mathrm{IVS} + \mathrm{PWT} + \mathrm{LVEDD}]^3 - [\mathrm{LVEDD}]^3)) + 0.6$ g (Lang et al. 2015).

Pulsed wave Doppler (PW) and tissue Doppler imaging (TDI) were used for the assessment of diastolic function, including measurements of ventricular septal peak systolic velocity (S'), peak early diastolic velocity (e') and peak late diastolic velocity (A') (Maragiannis et al. 2015). The right ventricle was also evaluated according to the updated guidelines and measurements were then indexed to the BSA (Mor-Avi et al. 2011), calculated with Du Bois and Du Bois formula: 0.007184 x (height(cm)^0.725) x (weight(kg)^0.425) (Burton 2008). The RV outflow tract (RVOT) diameter was measured in the parasternal long-axis (LAX) view (at a proximal level from the anterior RV wall to the RV septum) (Rudski et al. 2010). In the apical four-chamber view, end-diastolic and end-systolic areas were measured by tracing the endocardial contour, from which fractional area change (FAC) was calculated and expressed as a percentage (Rudski et al. 2010). TAPSE was measured as an index of RV longitudinal systolic function (Rudski et al. 2010).

Different types of LV remodelling were defined based on the measurements obtained, including NG defined as LVM ≤ 115 g/m^2 in male or ≤ 95 g/m^2 in female and RWT ≤ 0.42; CR as LVM ≤ 115 g/m^2 in male or ≤ 95 g/m^2 in female and RWT > 0.42; CH as LVM > 115 g/m^2 in male or > 95 g/m^2 in female and RWT > 0.42 and EH, as LVM > 115 g/m^2 in male or > 95 g/m^2 in female and RWT ≤ 0.42. The study design of the present investigation was evaluated and approved by the Review Board of the Institute of Medicine and Sports Science. All athletes included in this study were fully informed of the types and nature of the evaluation and signed the consent form, according to Italian Law and Institute policy. All clinical data assembled from the study population are maintained in an institutional database. The study was performed in accordance with the ethical standards as laid down in the 1964 Declaration of Helsinki.

Statistical analysis

Categorical variables were presented as frequencies and percentages, and their comparisons were conducted using Fisher's exact test or Chi-squared test, as appropriate. For continuous variables, normality criteria were checked, and results were expressed as mean ± standard deviation (SD), otherwise median and interquartile range were used. Comparison between groups was performed using Student's t-test or Mann–Whitney test, depending on normality criteria of data distribution. Effects sizes are reported as Cramer's V (contingency tables) or Cohen's d (continuous variables). A two-tailed P value < 0.05 was considered statistically significant. ICCs were calculated to assess inter-observer and intra-observer agreement.

Statistical analysis was performed with STATA Statistics software for Windows (SE, version 17).

Results

Of the overall group of endurance athletes (n = 309), 211 athletes (68.3%) presented an EH (126 males and 85 females).

No case of CR or CH were observed in female. In male, 2 athletes (1.1%) presented CH and 1 (0.5%) CR. A not negligible proportion of endurance athletes, however, showed a NG (98 athletes, 31.7%; including 59 males and 39 females). Figure 1 shows a graphical distribution of elite endurance athletes, according to LVMi and RWT, in order to classify heart remodelling as NG, CH, CR and EH both in male **(Panel A)** and females **(Panel B)**. Table 1 shows a comparison between endurance athletes with NG and those with EH. Figure 2 shows the percentage of endurance athletes with EH, according to a sporting discipline practised.

Table 1 summarizes differences between athletes with EH and those with NG in both sexes. Male athletes with EH were generally older (27.3 ± 4.8 vs. 25.8 ± 3.7 years, p = 0.035). No significant differences were observed, in both sexes, for anthropometric parameters (weight, BMI, BSA), or body composition (percent fat mass), or volume of training as expressed by weekly hours, or eventually cardiovascular risk factors (smoking and family history for CV disease). As expected from the definition, athletes presenting EH showed morphological differences: larger LVEDD (p = 0.001 in males and females), end-diastolic volume (LVEDV, p = 0.001 in males and females) and end-systolic volume (LVESV, p = 0.005 in males and p = 0.001 in females), higher LVMi, (p = 0.001 in males and females) and greater LV wall thickness values (IVS and PWT, both p = 0.001 in male and female athletes), but similar ejection fraction (EF, p = 0.292 in males and p = 0.147 in females).

Also, as expected, concordant remodelling of other cardiac chambers was observed. Indeed, larger left atrial diameter (LAD, p = 0.001 in males and females) and volume (LAV, p = 0.001 in males) were observed. Furthermore, functional parameters were also different in the EH phenotype; specifically, improved diastolic filling with higher E-wave velocities (p = 0.028) and a higher E/A ratio (p = 0.032), was observed in males. In female athletes, improved LV relaxation parameters were observed, with lower e' septal velocities (p = 0.002) and higher E\e' ratio (p = 0.025) compared to athletes with NG. Finally, the morphological changes also involved the right chambers with EH athletes presenting a larger RV outflow tract (RVOT, p = 0.011 in males and p = 0.001 in females) and larger right atrial area (RAA, p = 0.007 in males and p = 0.001 in females).

Along with the morphological changes, functional differences were elicited by the exercise ECG test. Specifically, athletes with EH presented lower heart rates (p = 0.001 in both males and females) and tendentially a higher systolic blood pressure at peak exercise. The exercise capacity was definitely higher in EH athletes, as shown by the higher maximum absolute peak load achieved (p = 0.014 in males and p = 0.003 in females) and a higher normalized peak as expressed by Watt/kg ratio (p = 0.001 in males and p = 0.004 in females).

A few subsets of athletes presented ventricular premature (10.2% in NG vs 15.1% in EH, p = 0.0363 in male; 7.7% in NG vs 11.8% in EH, p = 0.492 in females) or supraventricular beats during exercise (6.8% in NG vs 7.9% in EH, p = 0.782 in male; 0% in NG vs 5.9% in EH, p = 0.324 in females), without any significant difference between the two groups.

Finally, we investigated personal sport achievements differences (in terms of Olympic or World championship medals or national\world record holder) between EH and NG athletes (Table 2) and no significant differences were observed both in male and female athletes.

Discussion

This study presents a comprehensive analysis of a large cohort of elite athletes focusing on cardiac remodelling and yields insights into how different types of sports influence the heart's structure and function. EH was the most prevalent remodelling in endurance athletes (67% in males, 68.5% in females). Notably, a considerable proportion (31.3% in males and 31.4% in females) of endurance athletes had NG, indicating individual variability in cardiac adaptation and various grade of exercise induced cardiac remodelling, according to different sporting disciplines, all classified as "endurance sports" (Fig. 2). For what concerns prevalence, the aforementioned work of Boraita et al. (2022) was a

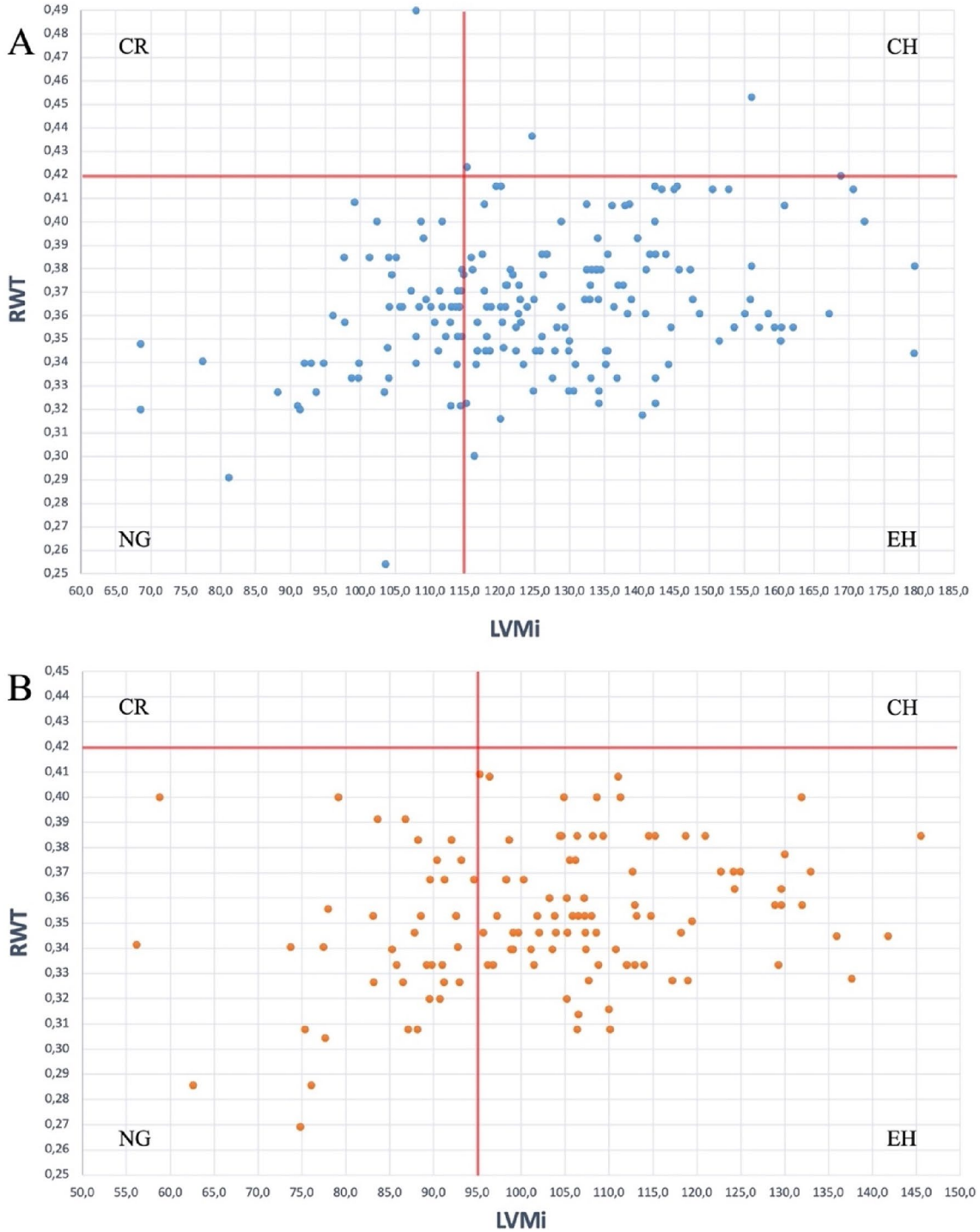

Fig. 1 Left ventricular remodeling in endurance athletes. Types of left ventricular remodeling. NG: LVM ≤ 115 g/m^2 in male and ≤ 95 g/m^2 in female, RWT ≤ 0.42; CR: LVM ≤ 115 g/m^2 in male and ≤ 95 g/m^2 in female, RWT > 0.42; CH: LVM > 115 g/m^2 in male and > 95 g/m^2 in female, RWT > 0.42; EH: as LVM > 115 g/m^2 in male and > 95 g/m^2 in female, RWT ≤ 0.42. **A**: males; **B**: females. *CH* concentric hypertrophy, *CR* concentric remodeling, *EH* eccentric hypertrophy, *LVMi* left ventricular mass indexed, *NG* normal geometry, *RWT* relative wall thickness

recent single-center, retrospective study on 3,282 Spanish elite athletes, in which individuals were grouped by sport and its relative dynamic/static component. They found a concrete different prevalence when compared to our cohort: 85.4% presented NG, with a smaller proportion of athletes (13.4%) showing EH.

Table 1 General, anthropometrics and echocardiographic differences between endurance athletes showing normal left ventricle geometry and those with eccentric hypertrophy according to gender

	Male, n = 185		P value	Effect size	Female, n = 124		P value	Effect size
	NG	EH			NG	EH		
n, (%)	59 (31.3)	126 (67)			39 (31.4)	85 (68.5)		
Age, years*	25.8 ± 3.7	27.3 ± 4.8	0.035	− 0.334	25.9 ± 5.6	26.7 ± 4.2	0.379	− 0.171
Weight, kg	75.7 ± 12.2	75.8 ± 11.6	0.957	− 0.008	57.9 ± 8.2	59.2 ± 13.8	0.587	− 0.105
BMI, kg/m^2	22.9 ± 2.5	22.6 ± 2.7	0.472	0.114	20.6 ± 2.1	21 ± 3.8	0.540	− 0.118
Fat mass, %	9.61 ± 2.9	10.3 ± 3.4	0.180	− 0.212	18.7 ± 4.1	17.6 ± 3.9	0.154	0.277
Afro-Caribbean, n (%)	3 (5.1)	5 (4)	0.711	0.025	2 (5.1)	0 (0)	0.097	0.189
Smokers, n (%)	0 (0)	1 (0.8)	0.999	− 0.050	0 (0)	0 (0)	0.999	0.000
Familiarity for CVD, n (%)	9 (15.2)	28 (22.2)	0.269	− 0.199	6 (15.4)	14 (16.5)	0.879	− 0.014
Training hours	27.5 (17.9–37.1)	27.7 (19.2–36.2)	0.721	− 0.056	24.0 (15.1–32.9)	23.5 (17.0–30.5)	0.778	0.054
BSA	1.95 (1.92–1.96)	1.94 (1.92–1.96)	0.752	0.050	1.64 (1.63–1.65)	1.64 (1.63–1.65)	0.999	0.001
LVEDDi, mm/m2*	28.0 (26.2–30.5)	30.2 (27.8–32.6)	0.001	− 0.941	29.6 (27.5–31.5)	32.0 (29.8–34.2)	0.001	− 1.102
LVESDi, mm/m2*	17.4 (15.3–19.5)	18.3 (15.5–21.0)	0.039	− 0.328	18.5 (16.5–20.7)	19.0 (16.8–21.2)	0.263	− 0.217
LVEDVi, mL/m2*	72.6 (64.0–81.4)	87.4 (69.3–105)	0.001	− 0.522	66.0 (50.6–81.4)	78.4 (61–95.5)	0.001	− 0.732
LVESVi, mL/m2*	26.1 (19.2–32.0)	31.4 (25.4–37.5)	0.005	− 0.445	22.3 (17.5–27.0)	27.5 (20.5–34.5)	0.001	− 0.800
LVMi/m^2*	103.5 (92.5–114)	135 (121.5–148.5)	0.001	− 2.287	84.3 (75.0–93.5)	111.5 (100–123)	0.001	− 2.504
RWT*	0.35 (0.32–0.38)	0.37 (0.34–0.40)	0.001	− 0.667	0.34 (0.31–0.37)	0.36 (0.34–0.38)	0.001	− 0.848
EF, %	63.3 ± 4.7	64.2 ± 5.7	0.292	− 0.166	65.9 ± 5.4	64.6 ± 4.2	0.147	0.282
IVS, mm*	9.9 (9.1–10.7)	10.9 (10.1–11.7)	0.001	− 1.250	8.6 (8.0–9.2)	9.6 (9.0–10.2)	0.001	− 1.667
PW, mm*	9.6 (8.8–10.4)	10.7 (10–11.4)	0.001	− 1.487	8.3 (7.6–9.0)	9.3 (8.7–9.9)	0.001	− 1.580
LA, mm*	36.4 (32–40)	40.0 (35.6–44.2)	0.001	− 0.991	33.3 (30.2–36.5)	35.6 (32.2–38.0)	0.001	− 0.725
LAVi, mL/m2*	22.3 (17–27.6)	30.2 (22–38.5)	0.001	− 1.056	21.3 (15.5–27.4)	23.1 (17.5–29.6)	0.153	− 0.278
AR, mm*	31.4 (28.6–34.2)	33.3 (30.4–36.0)	0.001	− 0.662	28.0 (25.5–30.5)	28.7 (25.9–32.4)	0.255	− 0.221
AA, mm*	28 (25–31.2)	29.5 (26.8–32.1)	0.005	− 0.448	25.3 (22.5–28.4)	27 (24.8–29.5)	0.001	− 0.646
E wave, cm/sec*	78.6 (63.3–89.8)	83.6 (68.4–98.1)	0.028	− 0.350	87.8 (72–103.5)	86.7 (74.5–98.2)	0.734	0.066
A wave, cm/sec	44.7 (35.5–54.1)	43.1 (33.2–53.9)	0.289	0.168	47.9 (35.5–60.0)	44.5 (34.8–54)	0.115	0.307
E/A*	1.83 (1.30–2.25)	2 (1.45–2.55)	0.032	− 0.340	1.88 (1.25–2.49)	2 (1.48–2.45)	0.247	− 0.225
e', m/sec*	12.2 (10.5–14.7)	12.6 (10.5–14.5)	0.206	− 0.200	13.2 (11.8–14.9)	12 (10.5–14.2)	0.002	0.618
E/e'*	6.57 (5.2–7.8)	6.7 (5.4–7.8)	0.557	− 0.097	6.7 (5.5–8.2)	7.4 (6.8–9)	0.025	− 0.437
A', m/sec	6.5 (5.5–7.5)	6.8 (5.5–8.0)	0.127	− 0.242	6 (4.8–7.2)	6.2 (4.9–7.5)	0.428	− 0.154
S', m/sec	8.2 (7–9.5)	8.4 (7.2–9.6)	0.331	− 0.154	8.1 (6.8–9.3)	7.7 (6.5–9.0)	0.114	0.307
TAPSE, mm*	27.8 (20.5–34.2)	28.3 (24.5–32.7)	0.604	− 0.082	24.2 (19.5–30.1)	27.8 (24.1–30.8)	0.001	− 0.792
FAC, %*	47.2 (40.7–54.3)	48.2 (40.8–55.9)	0.412	− 0.130	49.8 (42.1–57.5)	45.9 (38.4–53.6)	0.004	0.565
RVOTi LAX, mm/m2*	15.9 (13.4–18.4)	16.8 (14.7–18.9)	0.011	− 0.402	17 (14.3–19.5)	18.7 (16.5–21)	0.001	− 0.699
Rai, mm2/m2*	11 (8.4–13.3)	11.9 (10–14.2)	0.007	− 0.426	10.2 (9–11.5)	12.1 (10.2–14.5)	0.001	− 0.978
VEB, n (%)	6 (10.2)	19 (15.1)	0.363	− 0.067	3 (7.7)	10 (11.8)	0.492	− 0.062
SVEB, n (%)	4 (6.8)	10 (7.9)	0.782	− 0.020	0 (0)	5 (5.9)	0.324	− 0.139
Rest HR, bpm*	66.1 ± 13	58.6 ± 11.6	0.001	0.622	68 ± 13.2	59.2 ± 11.2	0.001	1.184
Peak HR, bpm*	164.9 ± 12.1	160.8 ± 11.8	0.030	0.344	165.6 ± 8.7	164.2 ± 10.6	0.473	0.139
Rest SBP, mmHg	111.9 ± 10.7	113.2 ± 12.1	0.481	− 0.111	101.7 ± 9.8	102.8 ± 10.6	0.584	− 0.106
Rest DBP, mmHg	70 ± 7.9	69.4 ± 7.9	0.631	0.076	66 ± 6.9	64.2 ± 6.8	0.176	0.264
Peak SBP, mmHg*	185.1 ± 19.8	191.3 ± 20.6	0.055	− 0.305	165.1 ± 16.5	172.6 ± 15.4	0.015	− 0.476
Peak DBP, mmHg	74.4 ± 9	73.8 ± 9.9	0.693	0.062	70 ± 9.2	70.9 ± 7.6	0.568	− 0.111
Watt*	317.1 ± 71.2	342.2 ± 60.6	0.014	− 0.391	225.1 ± 38.7	247.1 ± 37	0.003	− 0.586
Watt/kg*	4.21 ± 0.8	4.6 ± 0.7	0.001	− 0.532	3.89 ± 0.6	4.23 ± 0.6	0.004	− 0.567

AA ascending aorta, *AR* aortic root, *BMI* body mass index, *BSA* body surface area, *CH* concentric hypertrophy, *CR* concentric remodeling, *CVD* cardiovascular disease, *DBP* diastolic blood pressure, *EF* ejection fraction, *EH* eccentric hypertrophy, *FAC*: fractional area change, *HR* heart rate, *IVS* interventricular septum, *LA* left atrium, *LAVi* left atrial volume indexed, *LVEDDi* left ventricular end-diastolic diameter indexed, *LVEDVi* left ventricular end-diastolic volume indexed, *LVESDi* left ventricular end-systolic diameter indexed, *LVESVi* left ventricular end-systolic volume indexed, *LVMi* left ventricular mass indexed, *NG* normal geometry, *P* posterior wall, *RA* right atrium, *RVOT* right ventricle outflow tract, *RWT* relative wall thickness, *SBP* systolic blood pressure, *SVEB* supra-ventricular ectopic beats, *TAPSE* tricuspid annular plane systolic excursion, *VEB* ventricular ectopic beats

*Indicates variables with $P < 0.05$. Continuous data are shown as mean ± standard deviation or median (interquartile range) and tested accordingly

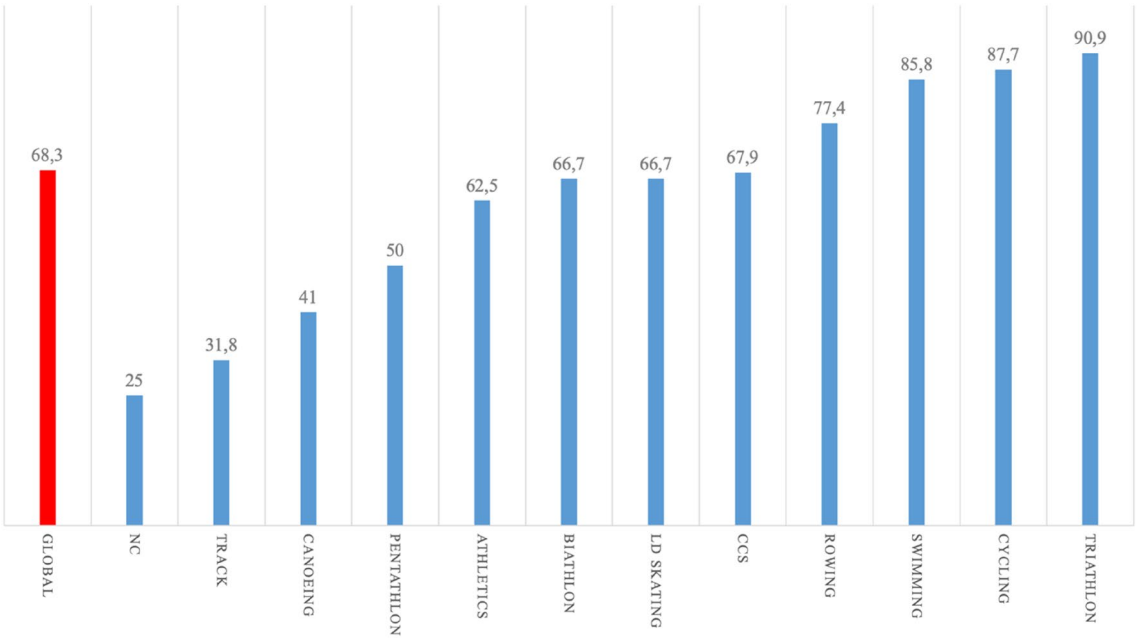

Fig. 2 Endurance athletes with eccentric hypertrophy according to sporting discipline practiced. *CCS* cross-country skiing, *LD* long-distance, *NC* nordic combined

With the due comparative limitations of multiple variables heterogeneity, the difference between our results and Boraita et al. was mainly on the inclusion criteria: in fact, we exclusively included endurance athletes, while mixed sporting disciplines were also included in the abovementioned study (Boraita et al. 2022). Moreover athletes were studied during different training periods: intense training in our study, while Colleagues enrolled athletes in preparatory period (Boraita et al. 2022).

Nevertheless, the concept of cardiac remodelling in endurance athletes was pioneered by John Morganroth in 1975, when he introduced the concept of type-specific cardiac remodelling, describing the EH pattern in highly trained swimmers (Morganroth et al. 1975, 1977). This hypothesis was further supported by several echocardiographic and, more recently, by cardiac magnetic resonance (CMR) observations (Conti et al. 2021; Pujadas et al. 2018). Of note, Spence et al. (2011) found that endurance-trained individuals showed LV EH, whereas resistance-trained participants did not show significant changes in heart structure. Recently, Morrison et al. (2023) conducted a meta-analysis evaluating the impact of exercise training on LV remodelling: the main determinants of remodelling depended on age, gender and endurance type of training. Young adults (compared to middle-aged) exhibited greater cardiac remodelling and males experienced a greater increase in wall thickness compared to females.

On what concerns age-related remodelling, in a study by Kusy et al. (2021), exercise-induced cardiac remodeling in master athletes shifted toward NG in sprinters and toward CR in endurance runners. Also Morrison et al. (2023) highlighted that changes in heart geometry were not shown in master athletes compared with young and middle-aged individuals.

In our cohort, athletes with EH were older than those with NG (even if in this age range, characterized by the highest physical fitness level and sports performance the difference observed in our study has no practical\clinical importance) and males experienced greater increases in wall thickness than females, and no cases of CR or CH were observed in females. Additionally, male EH athletes presented morphological differences, including larger LVEDDi and LVEDVi, and greater LV wall thickness, consistent with the gender-specific remodelling trends noted by Morrison et al. (2023). Also, the impact of endurance training on cardiac remodelling was a concordant point. In fact, our study underscores a significant prevalence of EH, particularly in disciplines like cycling, rowing, and athletics.

Our results focus also on the morphological and functional differences in athletes with EH. We found that EH athletes exhibited larger LVEDDi, LVEDVi, LVESVi, LVMi, and greater LV wall thickness, with comparable LVEFs, characteristic of EH, reflecting an adaptive response to chronic volume overload (Oxborough et al. 2006). For what concerns functional differences, we found that these individuals present improved diastolic function, with higher E-wave velocities and E/A ratio in males and a higher E/e' ratio in females (due to lower E' velocities).

Table 2 Personal achievement differences of endurance athletes according to left ventricle remodelling type and gender

	NG combined group 98 (31.7)	EH combined group 211 (68.3)	P value	Effect size	NG Males 59 (31.9)	EH Males 126 (68.1)	P value	Effect size	NG Females 39 (31.4)	EH Females 85 (68.5)	P value	Effect size
Olympic medal, n (%)	17	39	0.809	− 0.014	10	27	0.478	− 0.052	7	12	0.582	0.049
World medal, n (%)	19	47	0.564	− 0.033	14	33	0.720	− 0.026	5	14	0.600	− 0.047
WR, n (%)	1	6	0.438	− 0.057	1	6	0.433	− 0.075	0	0	0.999	0.000
NR, n (%)	5	13	0.800	− 0.021	3	8	0.735	− 0.025	2	5	0.866	− 0.015

CH concentric hypertrophy, *CR* concentric remodeling, *EH* eccentric hypertrophy, *NG* normal geometry, *NR* national record, *WR* world record

Furthermore, they presented global heart remodeling, showing larger LA diameter and volume, and right chamber changes. This comprehensive remodelling is indicative of global cardiac adaptation to endurance training.

Actually, our results suggest, however, that not all elite, highly trained endurance athletes show the expected eccentric remodelling, raising questions about the clinical and functional significance of such a lack of LV remodelling. However, individuals without the expected EH reached the same athletic achievements (in terms of medals or records) as the other ones with evident LV remodelling (Table 2).

While the exact mechanisms remain unclear, the duration and intensity of training, genetic predispositions, as well as peripheral vascular function may represent determinants that can influence the extent of cardiac adaptations.

First, the duration and intensity of endurance exercise likely modulates the extent of cardiac remodelling. The specific, individual training agenda followed by non-remodelled athletes may had been different from those with remodelling. Elements including exercise volume, frequency, duration, and intensity, as well as the presence of interval or resistance training components, could had affected the physiological heart responses in the individual athlete.

Additionally, athletes who have engaged in endurance training for shorter durations or had intermittent training bouts throughout their careers may not have reached the threshold for significant cardiac remodelling. It is plausible that long-term adaptations occur gradually over time and athletes with shorter training conditioning have not yet reached that threshold for a significant remodelling.

A relevant aspect is that genetic predisposition probably plays a significant role. Genetic factors may be associated, with some gene variations known to be associated with LV hypertrophy (Mihl et al. 2008; Ellison et al. 2012; Weiner et al. 2012). Some athletes may possess genetic traits that make them less prone to developing significant structural cardiac changes despite engaging in intense training. We can speculate that these genetic factors could influence various aspects, including myocardial hypertrophy, collagen synthesis and remodelling pathways, ultimately influencing the cardiac response of the individual athlete. Eventually, other physiological factors could also contribute to the absence of structural changes in the heart. Improved oxygen utilization, due to skeletal muscle adaptations, with enhanced peripheral vascular adaptations, may represent other mechanisms that may explain the lack of eccentric cardiac remodelling.

As shown, gender plays an important role, with female athletes generally showing less remodelling than males, even after correcting for body size (Mihl et al. 2008; Ellison et al. 2012; Weiner et al. 2012). Ethnicity also influences remodelling, with Afro-Caribbean athletes generally having thicker

LV walls than white athletes (Mihl et al. 2008; Ellison et al. 2012; Weiner et al. 2012).

Regarding the exercise capacity, the differences observed in heart rate, blood pressure and maximum exercise capacity between athletes with EH and those with NG support the efficiency of EH to improve athletic performance. Athletes with EH demonstrated a lower heart rate during exercise, which can be associated primarily to an increased stroke volume and then to a highly efficient vascular remodelling capable of delivering more blood more and more efficient oxygen delivery to peripheral muscles. Additionally, these athletes achieved higher maximum workload, indicating greater overall power and performance capability.

Additionally, one more aspect to consider is related to the intrinsic heterogeneity of the "endurance" sport classification. In fact, despite most of sporting disciplines classified as endurance have a predominant aerobic component, also a psychological, tactic or technical component cannot be underestimated in others (i.e. shooting in pentathlon and biathlon), influencing sports achievements and cardiovascular parameters. In fact, as shown in Fig. 2, non-EH percentages vary in different sporting disciplines, according also to aforementioned components. However, in our study a noticeable percentage of non-EH athletes was observed also in "strongly" endurance disciplines.

Another final consideration regards the role of echocardiography in the physiologic evaluation of athletes: by assessing cardiac sizing, systolic and diastolic function through echocardiography, trainers and sports physiologists would gain valuable insights into an athlete's cardiovascular fitness and ultimately its impact on performance.

Limitations

This study presents some limitations. First, it is designed to be retrospective, suggesting that data collection and analysis were based on previously achieved information. Secondly, although athletes were evaluated during a period of training, the seasonal variability has not been specifically considered. It is reasonable that athletes adjust their training load throughout the season. The timing and frequency of competitions can help shape the heart, as periods of intense competition can lead to more significant adaptations, consequently representing a discerning factor. Moreover, we included an important time frame to overcome this limitation.

Additionally, there is a lack of detailed information on training intensity, duration, and frequency in the individual athlete, which limits the ability to establish a direct relationship between training load and cardiac remodelling. Eventually, the gold standard evaluation for aerobic fitness is the VO_2 max, but the present study did not include a cardiopulmonary exercise test to eventually correlate the VO_2 max. Finally, we evaluated Italian elite athletes including mostly Caucasians; further studies are needed to compare Afro-Caribbean and Caucasian differences.

Conclusions

Our study demonstrates a relatively high prevalence of elite endurance athletes without expected EH, with similar rates in males and females, and challenge the traditional hypothesis of cardiac remodelling in this setting. Athletes with NG perform worse in exercise stress test and exhibit some less advantageous functional heart characteristics. However, the type of heart geometry is not associated with negative clinical findings or lower achievement of sport results.

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Author contribution All authors have approved the final article and approved the submission. GDG, SPC, MRS: conception and design of the study; SM, VM, DO, AP, AS, EL, AN: acquisition of data, or analysis and interpretation of data; EL, MRS, AS, AN: drafting the article or revising it critically for important intellectual content; GDG, AP: final approval of the version to be submitted.

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Data availability De-identified participant data are available upon reasonable request from the corresponding author.

Declarations

Conflict of interest None.

Ethical approval The study design of the present investigation was evaluated and approved by the Review Board of the Institute of Medicine and Sports Science. All athletes included in this study were fully informed of the types and nature of the evaluation and signed the consent form, according to Italian Law and Institute policy. All clinical data assembled from the study population are maintained in an institutional database.

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