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Long-term left atrial adaptations to reduced training load in former elite athletes: a long-term follow-up longitudinal observational study

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ABSTRACT

Objective Our objective was to evaluate the effects of long-term reduced training on the left atrium (LA) in elite endurance athletes and to elucidate sex-specific differences in LA detraining patterns.

Methods In this long-term longitudinal echocardiographic study of 50 active elite endurance athletes a follow-up examination was performed 7 years after retirement from the elite programme. All echocardiographic measurements were indexed for body surface area. We analysed the changes between baseline and follow-up measures using analysis of covariance models adjusted for baseline level, sex and enrolment age as covariates. Results are reported as least squares means with two-sided 95% CIs.

Results LA enlargement (left atrial maximum volume index) remained unchanged from baseline (change from baseline: 1.4mL/m^2, 95% CI: −0.7 to 3.5 mL/m^2) despite significant reductions in VO_2max (change from baseline: −864mL/min, 95% CI: −1091 to −637 mL/min). In contrast, left ventricular (LV) end-diastolic volume was reduced (change from baseline: −8mL/m^2, 95% CI: −11 to −5 mL/m^2), consistent with reduced VO_2max. LA contraction strain was increased (change from baseline: 1.4%, 95% CI: 0.4% to 2.5%), while LV filling pressure increased (E/e' change from baseline: 0.4, 95% CI: 0.1 to 0.7).

Conclusions 7 years of reduced training does not reverse exercise-induced LA enlargement in former elite endurance athletes. LA contractile function improved with higher LV filling pressure, suggesting that age-related LV pressure increases may contribute to chronic LA dilation, though irreversible adaptations like fibrosis cannot be ruled out.

Trial registration number NCT05555849.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ The risk of atrial fibrillation may be elevated up to fivefold in former endurance athletes without an established cause, although limited data specifically constrain definitive conclusions for female athletes.

WHAT THIS STUDY ADDS

⇒ In this large long-term follow-up study of reverse cardiac remodelling in former elite athletes, we find left atrial dilation to be chronic, irrespective of a prolonged period of reduced training, whereas ventricular size tends to decrease by current fitness level. Our results suggest that an age-related decline in left ventricular compliance may contribute to chronic left atrial dilation in former elite endurance athletes. Our findings revealed no sex-specific differences in left atrium (LA) reverse remodelling.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ This study demonstrates that atrial reverse remodelling exhibits distinct characteristics compared with ventricular remodelling. It remains unclear whether chronic dilation of the LA is solely attributable to age-related reductions in compliance or intrinsic structural/functional alterations within the LA (ie, fibrosis).

INTRODUCTION

The long-term consequences of intensive training in elite athletes remain largely unknown due to the limited research available on long-term cardiac detraining.[1] In recent years, a U-shaped association has been suggested linking the risk of atrial fibrillation (AF) to exercise intensity.[2] The benign physiological adaptations, referred to as 'athlete's heart', include dilation of cardiac chambers and mildly reduced contraction at rest, with decreased resting heart rate (HR) and increased HR variability (HRV), reflecting high vagal tone.[3] AF regularly originates near the pulmonary veins in the left atrium (LA),[4] highlighting the importance of LA remodelling. Some features governing LA morphology and function are predetermined (age, sex, ethnicity), while others are highly dynamic (body surface area (BSA), maximal oxygen uptake (VO_2max), HRV). Additionally, left ventricular (LV) compliance (as measured by E/e') is highly age-dependent.[3 5–8] In

otherwise healthy young non-athletes the age-related increase in LA size is primarily attributed to a decline in LV diastolic function, resulting in elevated LA afterload and subsequent LA dilation.[9] A characteristic feature of the athlete's heart is a supranormal diastolic function measured as low E/e' ratio indicating a high degree of LV compliance and LV reduced stiffness.[3] Prior studies in non-athletes have reported an increase in LA size with advancing age, though data on LA size in a young population <30 years is sparse and no longitudinal data on LA size in athletes is available.[10] Periodisation studies in young elite athletes have shown that LA volume adapts quickly to increased exercise over a period of weeks,[11] and any subsequent gains remain solely incremental,[12] while training cessation results in a rapid decline in LA volume and VO_2max within the first weeks of detraining.[11 13]

Distinguishing pathological remodelling in athletes

In a recent population study by Sabo *et al*, an increased atrial/ventricular size ratio was significantly correlated to unfavourable cardiovascular risk factors, including echocardiographic measures suggesting atrial/ventricular size ratio as a potential marker for cardiovascular risk.[14] The long-term consequences of LA enlargement (LAE) vary depending on the type of LA adaptations[15]; however, distinguishing benign from pathological LA remodelling in athletes has proven challenging. The causes of athletic-induced pathological remodelling of the LA remain unclear[16] and have so far been established only in male athletes, with limited data available for female athletes.[17] Benign LAE may be profound in elite athletes and may surpass LA volumes seen in patients with AF,[18] highlighting that even substantial LA dilation may not be a marker of pathology. Other measures of atrial morphology, such as increased atrial stiffness or reduced function, have been suggested as an early marker for LA fibrosis and pathological remodelling in athletes.[19]

To our knowledge, this is the largest long-term follow-up study of LA inverse remodelling in former elite endurance athletes. The objective of this study was to investigate the effects of long-term reduced exercise on key LA parameters in young former elite endurance athletes and evaluate sex-specific differences in LA remodelling over a ≈10-year follow-up period.

METHODS

Study design and methods

In this long-term longitudinal observational cohort study, we consecutively invited athletes in sports of high cardiac demands (Mitchell Sports Classification 1C-2C-3C,[20] corresponding to the endurance category in the updated recommendations on the evaluation of athlete's heart[21] from 'The Danish Athletes Heart Cohort' (baseline results published in 2016 and 2024[22 23] to achieve a target of 50 included athletes. The only inclusion criterion was: Retired from the elite training programme at Team Danmark >6 months ago. Only athletes with prior diagnosed cardiac conditions were excluded. All participants received a clinical examination by the designated physician and were interviewed on medical and training history, including a detailed description of recreational sports participation and estimated mean training hours since retirement from the elite training programme.

Echocardiographic analysis

A standard transthoracic echocardiographic examination (TTE) was performed by an experienced sonographer according to current guidelines[24] (GE Vivid E9 and GE Vivid S6, Vingmed, Horten, Norway). The methods used for standard echocardiographic analysis in this cohort have already been described in previous work by this group.[23] Ventricular (LV end-diastolic and systolic volume index: LVEDVi) and atrial volumes (left atrial maximum and minimum volume: maxLAVi, minLAVi) were indexed to BSA. Reference limits for E/e' (<8.0 m/s) were defined according to the current British Society of Echocardiography guidelines for screening of athletes.[25] No E/A reference limits exist for athletes; therefore, an E/A ratio of <0.8 or >2.0 was considered abnormal according to current general guidelines.[24] The upper limit of normal maxLAVi in athletes was defined as 34 mL/m^2 in accordance with the current guidelines.[24] No defined reference limits exist for minLAVi in athletes or the background population. Reduced left atrial reservoir strain (LAre) was defined as <34.6 and reduced left atrial contraction strain (LAct) as <11.6, based on a meta-analysis of 408 athletes.[26] LA Stiffness Index was calculated as (E/e')/LAre.[27]

Cardiopulmonary exercise test

Baseline VO_2max data from each participant were collected from the in-house seasonal testing at the Danish National Elite Sports organisation (Team Danmark). Follow-up cardiopulmonary exercise testing (CPET) was performed at our clinic using a standard bicycle ergometer test protocol with continuously increasing resistance (Vyire Vyntus CPX, Lode Corival, Netherlands). Recognition of VO_2max required meeting three of the following criteria: individual perception of exhaustion, respiratory exchange rate >1.15, VO_2 curve plateau, HR approaching age-predicted maximum or inability to maintain a pedalling frequency above 80 rpm.

Holter monitoring

HRV was evaluated using the Cortrium C3+Holter unit (Cortrium Aps, Denmark). The SD of R-R intervals and root mean square of successive differences of R-R intervals were assessed individually and compared with reference values of the background population.[28] Additionally, a number of premature supraventricular (PSVB) and ventricular (PVB) beats were documented, and any cardiac arrhythmias were diagnosed by an experienced cardiologist.

Biomarkers

Troponin T (TnT) and pro-brain natriuretic peptide (proBNP) were measured before exercise testing at our

in-hospital laboratory. The upper reference limit for TnT was 14 nmol/L, and for proBNP, 125 pmol/L for this age group.[29] According to hospital standards, the reference limits were 8.3–10.5 mmol/L for haemoglobin and 60–105 µmol/L for creatinine.

Statistical methods and sample size

Analysis of covariance models were employed to assess change from baseline to follow-up measures based on models adjusted for the level at baseline, sex and age at enrolment (applied as covariates). P values are reported as two-sided. We did not apply explicit adjustments for multiplicity but analysed, reported and interpreted the secondary objectives in a prioritised order.[30] With a power of 90% and a sample size of 49, we estimated that we should be able to determine a mean change in maxLAVi with a precision (95% CI) of ±1.3 mL.

Patient and public involvement

Recruitment and distribution of conclusions were planned in collaboration with The Danish National Elite Sports Organisation (Team Danmark). The project was executed and designed independently at our clinic.

RESULTS

Demographics

The recruitment process is described in figure 1. At baseline, the participants were all active elite endurance athletes with a mean VO_2max>140% of the expected level for this age group (table 1). The mean follow-up time was 12±3 years, and the time since retirement from the elite programme was 7±3 years. Elite career duration was 11±4 years. Compared with baseline, the athletes were training significantly fewer hours per week. Males had a larger BSA, but otherwise the characteristics were similar between the sexes.

Echocardiographic findings

Both maxLAVi and minLAVi adjusted for sex and age were unchanged at follow-up, despite reductions in training volume and VO_2max (figure 2 and table 2). Overall, maxLAVi increased in 28/50 athletes during the study period. At follow-up, 19/50 athletes had maxLAVi exceeding the reference limits for LAE; this was unchanged from baseline. Of these, 11/50 athletes had LAE at both baseline and follow-up, while 16/50 athletes showed a change in LAE status between examinations. Changes in primary and key secondary variables in individual athletes are illustrated in figure 3. Training hours per week did not differ between the eleven athletes with LAE both at baseline and follow-up and those without LAE at any time. During their active careers, athletes with LAE trained 19.0±5.1 hours/week compared with 19.8±4.9 hours/week for those without LAE; at follow-up, these figures were 7.0±5.8 hours/week and 5.6±3.5 hours/week, respectively. Postcareer training hours did not affect maxLAVi at follow-up (p=0.228). Age-adjusted maxLAVi and LVEDVi were indifferent between the most

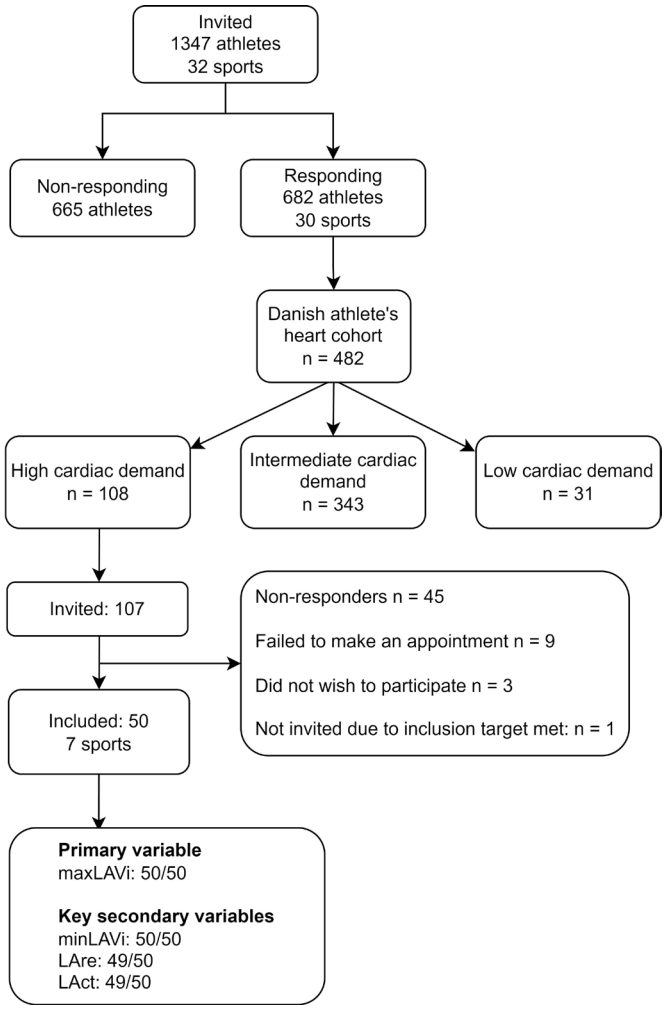

Figure 1 Flow diagram of the recruitment process. Detailed description of population recruitment for the Danish Athlete's Heart Cohort was published in 2014 DOI: 10.1111/sms.12405. LAct, Left atrial contraction strain; LAre strain, left atrial reservoir strain; MinLAVi, left atrial minimum volume index; MaxLAVi, left atrial maximum volume index.

postcareer active athletes (weekly training hours >5; n=21; maxLAVi=34.5 mL/m^2; LVEDVi: 75.3 mL/m^2) compared with their less active counterparts (weekly training hours 5 hours or less; n=29; maxLAVi=32.2 mL/m^2; p=0.264; LVEDVi: 73.1 mL/m^2; p=0.452).

Additionally, LA filling function (as measured by LAre) was unchanged since baseline with one-third of the athletes demonstrating reduced LAre beyond reference limits (baseline: 17/50; follow-up 15/50). Mean LA emptying function (as measured by LAct) did see a small increase since baseline (table 2), though >50% of the athletes exhibited LAct below the reference limits (baseline: 31/50; follow-up: 26/50). LA Stiffness Index was increased from baseline (table 2).

Determinants for LA size and function

Training volume at follow-up was reduced ≈60%, while VO_2max was 20% lower than at baseline (table 2). Mean LV filling pressure (as measured by E/e') increased over

Table 1 Baseline characteristics stratified by sex

	Male n=35	Female n=15	Total n=50
Age, years	22.8±4.3	23±7	23±5
Training hours per week, hours	18.0 (16; 21)	22.0 (17; 25)	19.5 (16.5; 22.0)
VO_2max, mL/min	5119±451	3864±393	4668±746
BSA, m^2	2.02±0.11	1.74±0.17	1.94±0.2
SBP, mm Hg	127±12	116±6	124±11.5
DBP, mm Hg	72±8	68±9	71±8.2
Heart rate, beats/min	54±7	53±7	54±7
Primary outcome measure			
MaxLAVi, mL/m^2	31.5±4.9	32.5±5.1	31.8±4.9
Key secondary outcome measures			
MinLAVi, mL/m^2	16.8±3.7	16.5±3.0	16.7±3.5
LAre strain, %	38±7	42±9	39±8
LAct strain, %	10±3	11±3	10±3
Other secondary outcome measures			
LA Stiffness Index, ratio	0.12±0.03	0.13±0.05	0.12±0.03
LAct/minLAVi, ratio	0.55 (0.43; 0.74)	0.67 (0.57; 0.94)	0.65 (0.45; 0.81)
LVEDVi, mL/m^2	84.4±10.1	77.7±11.1	82.4±10.8
LVESVi, mL/m^2	36.4±7.9	32.4±6.7	35.2±7.7
Peak E, cm/s	81±2	91±8	84±16
Peak A, cm/s	40±10	43±8	41±9
Average e', cm/s	20±3	19±3	19±3
E/A, ratio	2.08±0.42	2.22±0.73	2.12±0.53
E/e', ratio	4.23±0.82	4.91±0.98	4.44±0.92
LVEF, %	57.1±6.9	58.5±4.3	57.5±6.3
LV GLS, %	17.1±1.9	19.2±1.5	17.8±2.0
RWT, %	0.36±0.04	0.35±0.05	0.36±0.04
LV mass, g/m^2	106.5±15.4	100.1±17.9	104.6±16.3

All measures are reported as means and SDs unless otherwise indicated. Training hours per week and LAct/minLAVi were not normally distributed and are thus reported as median and upper/lower quartile. Left Atrial Stiffness Index calculated as E/e'/LAre.
BSA, body surface area; DBP, diastolic blood pressure; LAct, left atrial contraction strain; LAre strain, left atrial reservoir strain; LVEDVi, left ventricular end systolic volume indexed to BSA; LVEDVi, left ventricular end-diastolic volume index; LVEF, left ventricular ejection fraction; LVESVi, left ventricular end-systolic volume index; LV GLS, left ventricular global longitudinal strain; MaxLAVi, left atrial maximum volume index; MinLAVi, left atrial minimum volume index; RWT, relative wall thickness; SBP, systolic blood pressure; VO_2max, maximum oxygen uptake.

the course of the study period, though no athletes exhibited an E/e' ratio outside the reference limits (table 2). We found a high prevalence of E/A ratio beyond the reference limits at both time points (baseline: 25/50; follow-up: 19/50), though the mean E/A ratio did not change over the course of the study (table 2). We found no sex-specific differences in the primary or key secondary outcome variables (table 3).

Multiple regression analysis of variables of interest for LA size

In multiple regression analysis of clinically determined variables (age, sex, BSA, VO_2max, E/e', SD of R-R intervals, figure 2), current VO_2max and age were identified as independent predictors for maxLAVi at follow-up. However, in univariate analysis only age was an independent predictor of current maxLAVi.

Abnormal biomarkers and arrhythmias

Chronic cardiac stress (evaluated by proBNP) was within normal parameters in all athletes (5.9±4.9), while high TnT was documented in one athlete; additional examination determined that this increase was of non-cardiac origin. Mean haemoglobin was 9.0±0.8 mmol/L and creatinine 81±16 µmol/L. We found no change in resting HR over the study period (table 2). We assessed vagal activity

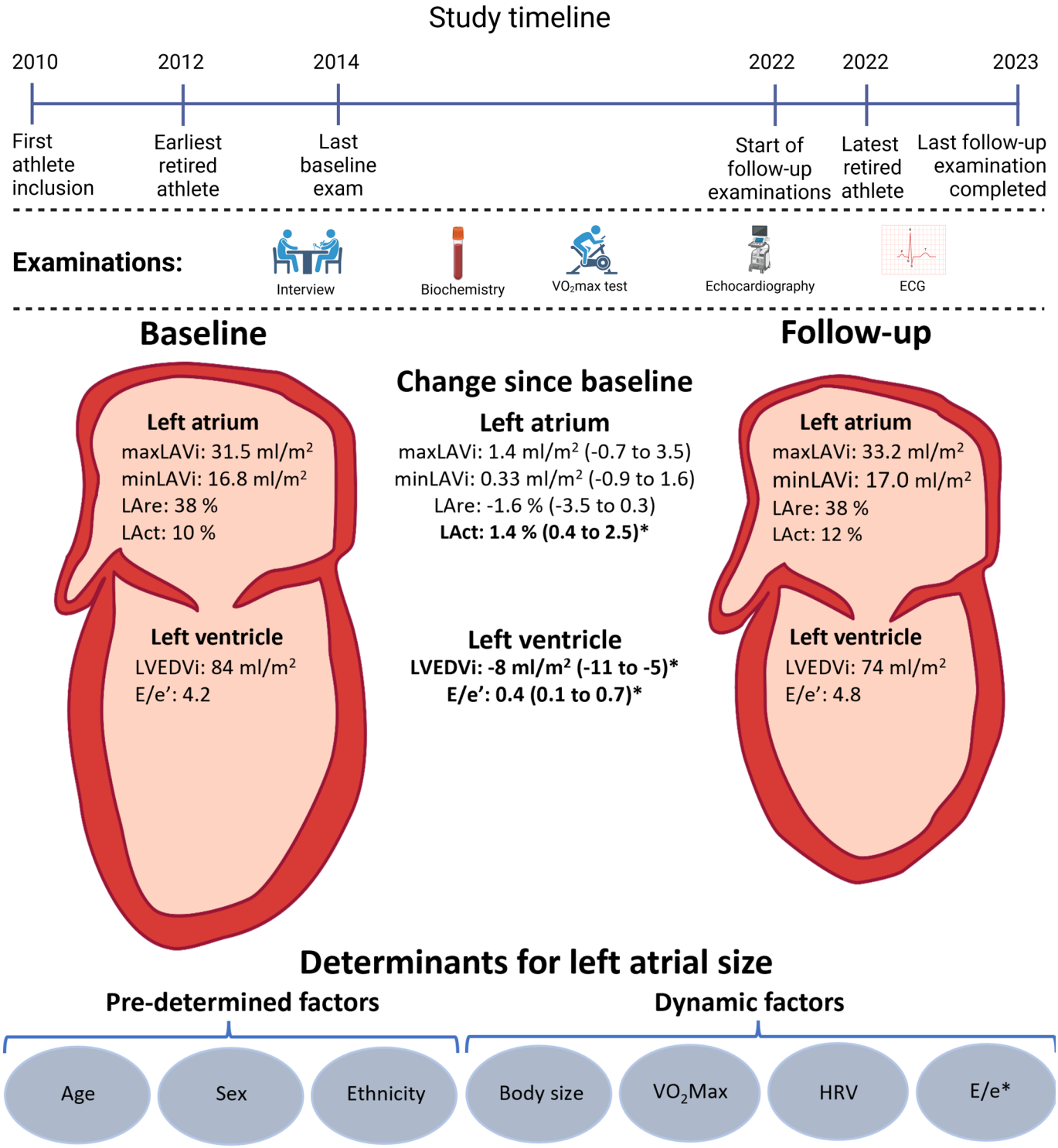

Figure 2 Graphical abstract. Determining factors of left atrial remodelling. *Denotes significant change since baseline. All measures were adjusted for age and sex. LV filling pressure (estimated by E/e'). Created with BioRender.com. HRV, heart rate variability; LAct, left atrial contraction strain; LAre strain, left atrial reservoir strain; LVEDVi, left ventricular end diastolic volume index; MaxLAVi, left atrial maximum volume index.

by SD of R-R intervals at follow-up (males: 216±51 ms; females: 211±68 ms) and root mean square of successive differences of R-R intervals (males: 57±27 ms; females: 58±31 ms) during 24 hours Holter monitoring. We identified no cardiac rhythm disorders, and no athlete was recorded with >100 PSVB. Three athletes presented with >100 PVB, one of whom presented with >1000 PVB/day; this athlete was also one of three athletes exhibiting a high number of PVBs with inferior axis and right bundle branch block on ECG during CPET. Additional examinations (including MRI) revealed no structural heart disease, though both LVEDVi and maxLAVi were in the

Table 2 All outcome measures collected after ≈10 years adjusted for sex and age (in years)

	Follow-up	Change from baseline
Training hours per week, hours	6.1 (4.6 to 7.6)	–13 (–15 to –12)*
VO_2max, mL/min	3804 (3576 to 4031)	–864 (–1091 to –637)*
BSA, m^2	1.98 (1.96 to 2.01)	0.05 (0.02 to 0.07)*
SBP, mm Hg	126 (123 to 130)	3 (–1 to 6)
DBP, mm Hg	77 (74 to 80)	6 (3 to 10)*
Heart rate, beats/min	57 (54 to 59)	3 (–0 to –5)
Primary outcome measure		
maxLAVi, mL/m^2	33.2 (31.1 to 35.2)	1.4 (–0.7 to 3.5)
Secondary outcome measures		
minLAVi, mL/m^2	17.0 (15.8 to 18.2)	0.33 (–0.9 to 1.6)
LAre strain, %	38 (35.6 to 39.4)	–1.6 (–3.5 to 0.3)
LAct strain, %	12 (10.6 to 12.8)	1.4 (0.4 to 2.5)*
Other secondary outcome measures		
LA Stiffness Index, ratio	0.13 (0.12 to 0.14)	0.01 (0.01 to 0.02)*
LAct/minLAVi, ratio	0.72 (0.64 to 0.81)	0.07 (–0.01 to 0.16)
LVEDVi, mL/m^2	74 (71 to 77)	–8 (–11 to –5)*
LVESVi, mL/m^2	33 (31 to 34)	–2 (–4 to –1)*
Peak E, cm/s	77.8 (74.3 to 81.4)	–6.3 (–9.8 to –2.7)*
Peak A, cm/s	41.8 (39.1 to 44.4)	1.1 (–1.5 to 3.8)
Average e', cm/s	16.5 (15.6 to 17.4)	–2.9 (–3.8 to –2.0)*
E/A, ratio	2.0 (1.8 to 2.1)	–0.2 (–0.3 to 0.0)
E/e', ratio	4.8 (4.6 to 5.1)	0.4 (0.1 to 0.7)*
LVEF, %	55.6 (54.3 to 57.0)	–2 (–3 to –1)*
LV GLS, %	18.5 (17.8 to 19.2)	0.6 (–0.1 to 1.3)
RWT, %	0.34 (0.32 to 0.37)	–0.01 (–0.03 to 0.01)
LV mass, g/m^2	86 (81 to 91)	–19 (–24 to –14)*

Age-adjusted and sex-adjusted ANCOVA models values for all variables assessed at follow-up. All echocardiographic measures are reported as least squares means with 95% CI.
*Denotes significant change since baseline. Left atrial stiffness calculated as E/e'/LAre.
ANCOVA, analysis of covariance; BSA, body surface area; DBP, diastolic blood pressure; LAct, left atrial contraction strain; LAre strain, left atrial reservoir strain; LVEDVi, left ventricular end systolic volume indexed to BSA; LVEDVi, Left ventricular end-diastolic volume indexed to BSA; LVEF, left atrial ejection fraction; LVESVi, left ventricular end-systolic volume index; LV GLS, Left ventricular global longitudinal strain; MaxLAVi, left atrial maximum volume indexed to BSA; MinLAVi, left atrial minimum volume indexed to BSA; RWT, relative wall thickness; SBP, systolic blood pressure; VO_2max, maximum oxygen uptake.

top 10% of this cohort and had increased since retirement from the elite programme. This athlete was later diagnosed with AF. Six athletes were referred for additional examination (cause of additional examination: blood pressure: 1 CPET: 3, TTE: 1, biomarkers: 1).

DISCUSSION

This longitudinal study highlights several notable observations regarding long-term inverse LA remodelling in former elite endurance athletes. The main findings were: (1) LAE was chronic regardless of reduced training load, while LV size decreased in response to reduced training volume and VO_2max. (2) LA contractile function and LV filling pressure increased over the study period, while LAre was unchanged. (3) No sex-specific differences in LA outcome variables were observed.

Causes of LAE in former athletes

Cardiac chamber volumes have been found to correlate with VO2maxx in athletes,[31] reflecting increased preload, with LA volumes exceeding reference limits frequently observed in active elite endurance athletes.[32] Prior studies show that exercise-induced LAE in athletes may rival the LA volumes seen in non-athletes with AF,[18] making the distinction between benign and pathological LAE challenging. The pattern of cardiac remodelling and reverse remodelling differs significantly in athletes and non-athletes[3]. In non-athletes, LA size increases with

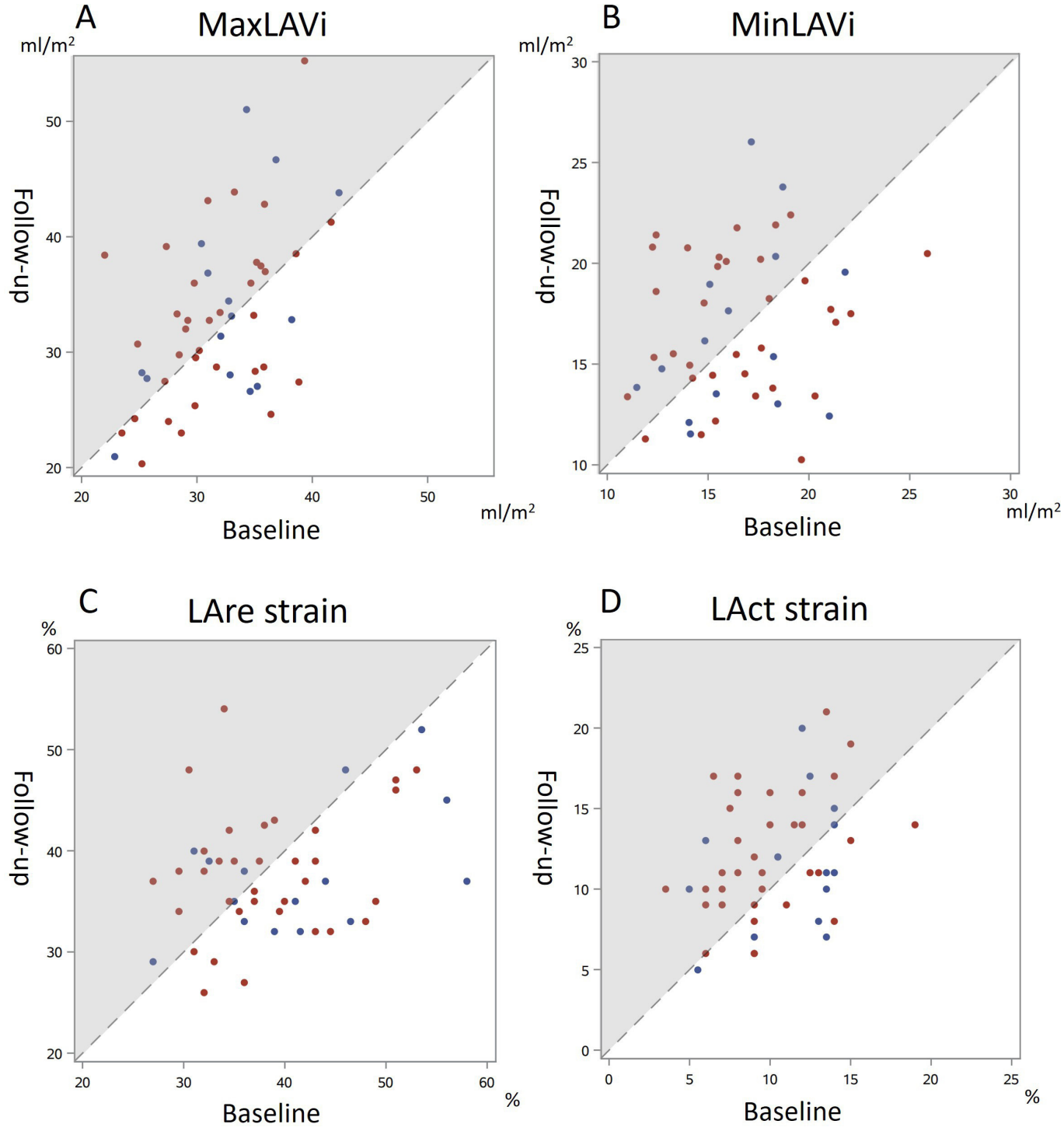

Figure 3 Change in primary and key secondary variables. Scatterplots indicating the ratio between baseline and follow-up values in primary and key secondary variables. Grey area marks increased values throughout the study period. (A) MaxLAVi, left atrial maximum volume indexed to BSA. (B) MinLAVi, Left atrial minimum volume indexed to BSA. (C) LAre, left atrial reservoir strain. (D) LAct, left atrial contraction strain. Male and female athletes are depicted in red and blue, respectively. BSA, body surface area.

age[10]. A similar trend has been found in athletes, at a rate in which maxLAVi in young athletes may be comparable to non-athletes 30 years their senior,[10] though data on the age-associated increase in maxLAVi in a <30-year-old healthy population is limited. A recent meta-analysis by Christou and O'Driscoll indicated that the observed age-related increase in LA size in active athletes may be an artefact of increased training years rather than age.[33] During the study period, athletes from our cohort ceased their elite training regimen and thereby significantly reduced training load. A reduction in maxLAVi since baseline would therefore be expected. As such,

Table 3 Change from baseline comparing sexes for primary and key secondary outcome measures adjusted for age in years and the level at baseline

	Change among males	Change among females	Difference (95% CI)	P value
Primary outcome measure:				
MaxLAVi, ml/m^2	1.3 (–0.9 to 3.5)	1.4 (–2.0 to 4.8)	0.1 (–4.0 to 4.1)	0.978
Key secondary outcome measures:				
MinLAVi, mL/m^2	–0.8 (–0.5 to 2.2)	–0.2 (–2.2 to 1.9)	–1.0 (–3.5 to 1.5)	0.424
LAre strain, %	–1.1 (–3.2 to 1.0)	–2.2 (5.4 to 1.0)	–1.1 (–5.0 to 2.8)	0.569
LAct strain, %	2.1 (0.9 to 3.3)	0.8 (–1.1 to 2.6)	–1.3 (–3.6 to 0.9)	0.231
LA Stiffness Index, ratio	0.01 (0.00 to 0.02)	0.02 (0.00 to 0.03)	0.01 (–0.01 to 0.02)	0.511
LAct/minLAVi, ratio	0.09 (-0.01 to 0.18)	0.06 (–0.08 to 0.21)	–0.02 (–0.20 to 0.15)	0.782

Comparison between sexes in age-adjusted primary and secondary variables. All measures are reported as least squares means with 95% CI.
LAct, left atrial contraction strain; LAct/minLAVi, left atrial contraction to minLAVi ratio; LAre, left atrial reservoir strain; MaxLAVi, left atrial maximum volume indexed to BSA; MinLAVi, left atrial minimum volume indexed to BSA.

the age effect may contribute to the observed increase in maxLAVi since baseline. Other dynamic, not sports-specific factors also contribute to LA dilation (figure 2), including increased LV filling pressure, increased BSA and high HRV.

Short-term longitudinal studies of exercise-induced dilation of cardiac chambers have documented rapid and reversible adaptations to current exercise demands within weeks in young elite athletes.[11 34] Our cohort of athletes reported continued high training load after retirement from the elite training programme, although it was significantly reduced compared with baseline levels. No athlete had continued structured competitive training in their professional sports discipline. As such, the training load (volume and intensity) at follow-up was significantly lower after retirement from the elite training programme.

At follow-up, HRV in our population of former athletes remained in the upper quartile compared with the background population,[28] suggesting sustained high vagal tone despite significantly reduced VO_2max and a self-reported reduction in training load, indicating successful detraining. The results from our cohort suggest that exercise-induced LA dilation tends to remain chronic, even after a prolonged period of reduced training load, despite reductions in LV size consistent with decreased VO_2max and training volume.

Increased LV filling pressure as a consequence of age-related decline in LV compliance[35 36] may be a cause for impaired LA reservoir function and increased reliance on atrial contraction to sustain LV output.[36 37] Furthermore, increased LV filling pressure may cause excessive LA afterload, which in turn contributes to dilation and hence increases the long-term risk of AF.[38 39] Maximum HR (and consequently HR reserve) decreases with advancing age,[40] which intensifies reliance on large chamber volumes to boost cardiac output and sustain a high VO_2max, causing further increases in LA preload and dilation. Reductions in LA functional parameters (including LAct and LAre) have been suggested as possible non-invasive measures of pathological remodelling.[23] In our cohort of relatively young, former endurance athletes, we found a small increase in E/e' and LAct, suggesting a heightened reliance on contraction for atrial emptying, consistent with age-dependent remodelling. However, passive atrial emptying, indicated by the E/A ratio and LAre, was only borderline reduced at follow-up. The increased reliance on LA active emptying highlights a similar trend seen in the background population,[41] though the baseline levels in LA active and passive emptying seen in our athletic population differ from the background population.[41] These findings are consistent with the still relatively young age of the population. A similar pattern of LA dilation with increased LA stiffness and reliance on active atrial emptying may be observed in patients with myocardial fibrosis. However, since fibrosis was not assessed in this study, its presence cannot be excluded. These results corroborate findings by Cousergue *et al*, who found increased maxLAVi in older competitive athletes compared to younger athletes.[36] Additionally, Luthi *et al* documented that maxLAVi was significantly enlarged in former professional cyclists (age: 66±7 years) compared to a control group matched for age and present physical activity. This enlargement persisted despite a mean of 38 years since the cyclists' retirement from professional competition. The authors noted, however, that the study cohort included cyclists who were active during a period when performance-enhancing drugs were highly prevalent in the sport.[42]

The population was still highly active, and no participant had completely ceased physical exercise; therefore, it is possible that even reduced levels of training may be sufficient to maintain LA dilation, though both training volume and intensity were significantly reduced since baseline in line with the observed reductions in VO2max, LV size, suggesting long-term reductions in cardiac

preload. Therefore, the observed LA dilation may be a result of increased afterload or irreversible adaptations of the LA. An increase in LA Stiffness Index could indirectly indicate the latter.

Sex-specific differences in LA inverse remodelling

Cross-sectional studies have suggested a potential sex-specific difference in LA function among athletes[23 43] and that female athletes may be less prone to training-induced arrhythmias than their male counterparts.[16] However, this perceived sex-based difference has been challenged due to insufficient data on female athletes.[44] In this longitudinal study, we found no differences in LA inverse remodelling between male and female athletes, suggesting that the long-term consequences of elite endurance training may be similar between male and female athletes. However, given the relatively young age of this cohort, the study may be unable to detect temporal sex-specific differences in LA reverse remodelling over the study period.

Clinical implications

We provide essential insights into the consequences of elite endurance training on male and female athletes, identifying age-related increases in LV stiffness as a potential mechanism for chronic LA dilation in elite endurance athletes. However, longer-term longitudinal studies are needed to determine whether chronic LA dilation in former elite athletes is associated with an increased AF risk in master athletes. Although previous studies suggest that AF risk is primarily observed in males, extended follow-up is needed to clarify the long-term outcomes in former elite endurance athletes of both sexes.

Limitations

The longitudinal cohort study design with no control group presents inherent limitations in establishing causality. In particular the age-related contribution to changes in maxLAVi over the study period cannot be identified. In the general population, LA size tends to increase with age; though data on very young healthy individuals remain scarce. Furthermore, no longitudinal studies have been published on LA size in young elite endurance athletes during the transition from active career to retirement from elite sports. The studied population of former elite athletes was highly homogeneous in age and ethnicity, limiting the generalisability to other populations. A ≈10-year follow-up period may be insufficient to fully capture age-dependent changes in cardiac morphology and function. Retirement from elite sports typically occurs at a relatively young age, often preceding the onset of significant age-related cardiac alterations. CPET at follow-up was conducted according to a standardised in-clinic protocol. As baseline CPET was not performed, we obtained archived testing data from the annual in-house testing at 'Team Danmark'. It is probable that lean body mass changed over the study period, even though BSA was unchanged. However, body composition was not evaluated in this study.

Conclusions

In former elite endurance athletes, persistent LA dilation was observed after long-term reduced training load despite significant reductions in training load and measured VO_2max. LA contractile function increased, while LA reservoir function remained unchanged. LV filling pressure and, consequently, LA afterload increased over the study period. These changes may contribute to sustained LA dilation despite the reduction in LA load. No sex-related differences in detraining patterns were identified, suggesting that the long-term cardiac effects of extreme endurance exercise are comparable between male and female athletes.

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Patient and public involvement Patients and/or the public were involved in the design, or conduct, or reporting, or dissemination plans of this research. Refer to the Methods section for further details.

Patient consent for publication Not applicable.

Ethics approval The study was approved by the Regional Ethics Committee (H-21043707), and data collection was approved by the Danish Data Protection Agency (P-2021-722). Before inclusion all participants signed an informed consent form after receiving a verbal and written study outline. All data were stored in an encrypted online-based database (REDCap).

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