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Long-Term Changes in Ventricular Function in Recreational Marathon Runners

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IMPORTANCE The effects of long-term repetitive and strenuous exercise loads on the right ventricle and plasma troponin levels are unclear.

OBJECTIVE To investigate the marathon-induced increase in troponin T and its association with right ventricular remodeling after 10 years of repetitive bouts of strenuous exercise.

DESIGN, SETTING, AND PARTICIPANTS This was a longitudinal, observational cohort study (the Prospective Follow-Up, Marathon, Long-Term, Inflammation, Cardiovascular System [Pro-MagIC] study) conducted from August to December 2019. Participants were male marathon runners recruited from a single center in Germany.

EXPOSURES Repetitive strenuous exercise training and endurance competitions for 10 years.

MAIN OUTCOMES AND MEASURES Cardiac biomarkers and 3-dimensional echocardiography were assessed prerace, immediately, on days 1 and 3 postrace, and at 10-year follow-up.

RESULTS A total of 152 male runners (mean [SD] age, 43 [3] years) were included in this analysis. Right ventricular ejection fraction declined significantly from the prerace (median, 52.4%; IQR, 50.0%-55.1%) to immediate postrace (median, 47.6%; IQR, 44.7%-51.5%; $P < .001$) assessment and 1-day postrace (median, 50.7%; IQR, 48.4%-53.0%; $P = .001$) but recovered at 3-day postrace (median, 51.3%; IQR, 50.4%-53.0%; $P = .18$); it also recovered to the prerace level even at the 10-year follow-up (median, 51.9%; IQR, 49.6%-54.5%; $P = .15$). Left ventricular ejection fraction decreased (median, 59.6%; IQR, 55.6%-64.5% to median, 57.6%; IQR, 54.1%-61.6%; $P < .001$), whereas lateral E/e' ratio, as an index of left ventricular filling pressure, increased (median, 5.1; IQR, 4.3-6.1 to median, 5.4; IQR, 4.5-6.4; $P < .001$) at the 10-year follow-up. No association of exercise-induced troponin T increase after the marathon with right and left ventricular ejection fraction changes at the 10-year follow-up was detected (Pearson $r = -0.10$, $P = .35$ and Pearson $r = -0.09$, $P = .35$, respectively).

CONCLUSIONS AND RELEVANCE Results showed that marathon-induced acute troponin T increase was not associated with right ventricular ejection fraction after 10 years of endurance training and competition. Left ventricular ejection fraction and left ventricular diastolic function changed significantly; however, the values remained within normal limits. These data suggest that repetitive strenuous exercise training and endurance competitions did not induce long-term deterioration of right ventricular function in most recreational male endurance athletes.

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Supplemental content

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The benefits of moderate to vigorous physical activity on health and longevity are supported by several medical societies.[1,2] However, emerging data have implicated the potentially harmful cardiac effects of high-volume exercise workloads, especially right ventricle (RV)–associated fatigue, dysfunction, and tissue injury.[3-5] Furthermore, previous studies showed that exercise-induced acute increase in plasma cardiac troponin concentration was associated with an acute reduction in RV ejection fraction (EF); this may indicate myocardial fiber injury that eventually leads to RV dysfunction.[6] The RV responds favorably to short bouts of exercise; however, there is a concern that repetitive bouts of strenuous, prolonged endurance exercise may cause adverse cardiac remodeling and lead to exercise-induced arrhythmogenic right ventricular cardiomyopathy (EI-ARVC).[7]

However, previous studies were cross-sectional in design, and there are limited longitudinal data from cohort studies of acute exercise-induced changes, such as increases in troponin and the development of RV dysfunction over long-term follow-up in endurance athletes. Therefore, this study aimed to investigate RV remodeling using real-time 3-dimensional (3-D) echocardiography (RT3-DE) immediately after a marathon and after 10 years of repetitive bouts of prolonged, strenuous exercise and determine its association with marathon-induced increases in troponin T (TnT) level. We hypothesized that an exercise-induced increase in TnT levels after marathon competition would be associated with a decline in RVEF after 10 years of training and competition. Furthermore, we analyzed its association with short- and long-term changes in left ventricular EF (LVEF).

Key Points

Question Is marathon-induced troponin T release associated with long-term right ventricular remodeling after 10 years of repetitive endurance exercise?

Findings In this cohort study of 152 male marathon runners, right ventricular ejection fraction declined transiently postrace but recovered within 3 days and remained stable at 10-year follow-up. Troponin T release after the marathon was not associated with right or left ventricular function at long-term follow-up.

Meaning Study results suggest that transient cardiac changes after endurance exercise were reversible and not associated with troponin release, suggesting no persisting myocardial dysfunction in recreational marathon runners.

Methods

Study Design

The Prospective Follow-up, Marathon, Long-Term, Inflammation, Cardiovascular System (Pro-MagIC) study was a prospective observational single-center study that assessed cardiovascular changes induced by prolonged strenuous exercise and repetitive participation in endurance events in recreational athletes over a long-term follow-up period of 10 years. The Pro-MagIC study was conducted from August 2019 to December 2019 and was based on participants from the Beer, Marathon, Genetics, Inflammation and the Cardiovascular System (Be-MaGIC) study, which was conducted from June 2009 to November 2009. The detailed study protocol has been published previously.[8] The methods and study design did not change after the trial commencement. The local Ethics Committee of the University Hospital Klinikum rechts der Isar, Munich, Germany, approved the study. All participants provided written informed consent before participation in the study. This study followed the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) reporting guidelines.

Participants

The Pro-MagIC study was a long-term follow-up study based on the Be-MaGIC Marathon study.[9] The inclusion criterion was participation in the Be-MaGIC Marathon Study in 2009 for male runners aged 30 to 70 years. The studies (Be-MaGIC and Pro-MagIC) were conducted in 2009 and 2019, respectively. In the baseline Be-MaGIC study (2009), race and ethnicity were recorded; of the 277 participants, 1 participant (1%) identified as African American, and the remaining participants identified as White. At the 10-year follow-up, race and ethnicity were not reassessed; however, the 1 participant who had identified as African American at baseline did not participate in the follow-up. Therefore, the follow-up cohort consisted entirely of participants who identified as White at baseline.

Visits

Participants were examined 2 weeks before the marathon race in 2009 (termed *prerace*, V0), within 1 hour after finishing the marathon race (termed *immediately postrace*, V1), 1 day after the race (termed *1-day postrace*, V2), 3 days after the race (termed *3-day postrace*, V3), and 10 years after the initial race in 2009 (termed *10-year follow-up*, V4). The short-term follow-up period was defined as the visits during the period from prerace until 3 days postrace, while the long-term follow-up period was defined from the prerace to the 10-year follow-up period.

Cardiovascular Risk Factors, Questionnaire, and Cardiopulmonary Exercise Test

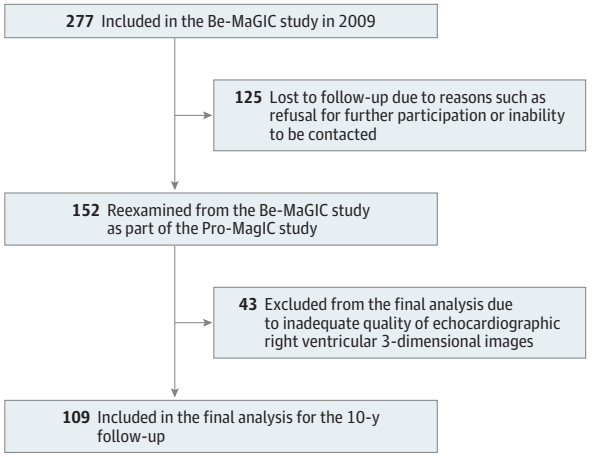
Body mass index (BMI) was calculated based on the measured weight and height, and body fat was calculated from the measurements of 7 skinfolds. Trained staff recorded blood pressure in the supine position. Hypertension was defined according to the current European Society of Cardiology (ESC) guidelines.[10] The participants responded to a 10-year retrospective questionnaire regarding known cardiovascular diseases (CVD) and training history, including average training distance, training hours per week, participation in endurance races (marathons, half-marathons, ultramarathons), family history of CVD, cardiovascular risk factors, and exercise-related symptoms. A cardiopulmonary exercise test (CPET) was performed at the 10-year follow-up (V4) using a cycle ergometer with a ramp protocol to assess cardiorespiratory fitness. An electrocardiogram was recorded continuously during the exercise test to detect arrhythmias, which were classified as exercise induced if

ventricular ectopic beats occurred during exercise or within 5 minutes of recovery. Higher-grade arrhythmias were defined as any triplets or nonsustained ventricular tachycardia.

Echocardiography

A complete echocardiography examination that included M-mode, B-mode, color, pulsed, continuous wave, and tissue Doppler analysis was performed prerace (V0), immediately postrace (V1), 1 day postrace (V2), 3 days postrace (V3), and at the 10-year follow-up (V4). Images and loops were acquired from the parasternal long and short axes, apical 2-, 3-, and 4-chamber views, focused RV view, and subcostal view. Full-volume samples of the LV and RV were derived over 7 consecutive beats from an apical view in the left lateral decubitus position using RT3-DE. Three-dimensional LV and RV recordings from all visits were analyzed using ImageArena, version 4.6 (TomTec Imaging Systems). Experienced sonographers performed the echocardiography and analysis. Intraclass correlation coefficients for interobserver and intraobserver variability ranged from 0.65 to 0.87 and were comparable with those of prior studies.[11] Aortic dilatation was defined as an end-diastolic diameter of the aortic root or ascending aorta greater than 40 mm, according to current ESC guidelines.[12]

Figure 1. Flowchart of Participant Inclusion



Be-MaGIC indicates Beer, Marathon, Genetics, Inflammation, and the Cardiovascular System Study; Pro-MagIC Study, Prospective Follow-up, Marathon, Long-Term, Inflammation, Cardiovascular System study (which is a 10-year follow-up of the Be-MaGIC study).

Clinical Chemistry

Blood samples were obtained from the antecubital vein, and plasma samples were aliquoted and stored in a −80 °C freezer. Datasets from the analysis of TnT and N-terminal pro-brain natriuretic peptide (NT-proBNP) have been published elsewhere.[13] Briefly, TnT level was measured using a high-sensitivity enzyme immunoassay based on electrochemiluminescence technology using a Cobas e 411 analyzer (Roche Diagnostics); the measurement range was 3 to 10 000 ng/L, the upper reference limit in healthy volunteers was set at 14 ng/L, and the interassay coefficient of variation was 6.5% at a concentration of 27 ng/L (to convert TnT to micrograms per liter, divide by 1000 and multiply by 1). NT-proBNP levels were analyzed using an enhanced electrochemiluminescence immunoassay system on a Cobas e 411 analyzer. The measurement range of NT-proBNP was 5 to 35 000 pg/mL, the interassay coefficient of variation was 4.2% at a concentration of 138 pg/mL (to convert NT-proBNP to nanograms per liter, multiply by 1), and the upper reference limit referred to age and sex as follows: males aged 18 to 49 years, 50 to 59 years, and older than 60 years as 65, 125, and 194 pg/mL, respectively.

Outcomes

The primary research question was the association between acute exercise-induced increases in plasma TnT level from prerace to immediately postrace after the initial marathon event in 2009 and changes in the RVEF from the prerace assessment (marathon in 2009) to the follow-up examination 10 years later in recreational endurance athletes. Secondary end points included the following: (1) changes from prerace to 10-year follow-up for the RV (RVEF, RV end-diastolic volume, and RV end-systolic volume); (2) association between acute changes in RVEF in 2009 and 10-year follow-up in relation to cardiovascular risk factors, laboratory parameters, and training history; (3) changes from prerace in 2009 to the 10-year follow-up of the left ventricle: LVEF, LV end-diastolic volume, LV end-systolic volume, E/A wave ratio (E/A ratio), and the ratio of early diastolic mitral inflow velocity to early diastolic mitral annular velocity (E/e'), as an index of LV filling pressure, measured at the lateral LV free wall (E/e' lat) and at the septal insertion of the mitral annulus (E/e' med); and (4) changes from prerace to immediately postrace, as well as at 10-year follow-up of biomarkers (TnT and NT-proBNP). Trained medical staff documented, categorized, and evaluated adverse events. Further outcomes that were recorded as part of the study protocol but not reported here included further cardiovascular assessments such as electrocardiography and vascular measurements.

Sample Size

To detect a relevant association between an acute increase in TnT level immediately after the marathon (defined as the change from prerace to immediately postrace) and a change in the RVEF within the 10-year follow-up (H_0: Pearson $r = 0.20$), a power of at least 90% was obtained with a sample size of 100 participants when a true correlation of 0.5 was assumed (significance level of 5%, 2-sided test). As a moderate number of missing values were expected due to the follow-up interval of 10 years, a total number of 130 participants (from the initial 277 participants of the Be-MaGIC study 2009) were included in the study.

Statistical Analysis

Data are presented as the mean (SD) or median (IQR). Count data are presented as frequencies and/or percentages. Differences in continuous normally distributed data between baseline and follow-up were analyzed using paired *t*-tests. The Wilcoxon signed-rank test was used for data following a skewed distribution, and differences in proportions were analyzed using the McNemar

Table 1. Characteristics of the Participants at Baseline and 10-Year Follow-Up Visits (N = 152)

Characteristic	Baseline (2009)	10-y Follow-up (2019)	*P* value
Anthropometry			
Age, mean (SD), y	43 (3)	53 (3)	
Body mass index, mean (SD)[a]	23.7 (2.1)	24.6 (2.8)	<.001
Total body fat, mean (SD), %	14.8 (4.2)	17.3 (4.7)	<.001
Systolic RR, mean (SD), mm Hg	124.9 (12.6)	127.6 (13.5)	.04
Diastolic RR, mean (SD), mm Hg	81.2 (7.5)	80.0 (8.3)	.08
Marathon run in 2009			
Marathon time (n = 129), mean (SD), h:min:s	3:49:02 (0:30:03)	NA	NA
Min/max race time, h:min:s	2:51:01/5:25:40	NA	NA
HR during race, mean (SD) bpm	157.0 (10.2)	NA	NA
Training history			
Training per week before the marathon in 2009 and 2019, median (IQR), km	50.0 (40-60)	30.0 (20-41)	<.001
Training per year, No. (%), km			
<500	5 (3.9)	11 (8.9)	NA
500-1000	21 (16.4)	31 (25.2)	NA
1000-1500	34 (26.6)	25 (18.7)	NA
1500-2000	35 (27.3)	22 (17.9)	NA
>2000	33 (25.8)	37 (29.3)	NA
Previously completed marathon races, No.			
Before 2009 (n = 129), median (IQR)	3 (1-7)	NA	NA
2009-2019 (n = 131), median (IQR)	NA	5 (2-9)	NA
Peak VO_2 (n = 143), mean (SD) mL/kg/min	NA	42.6 (7.8)	NA
Cardiovascular risk factors			
Diabetes, No. (%)	0	3 (2.0)	NA
Hypercholesterolemia, No. (%)	13 (9.4)	25 (16.4)	.07
Total cholesterol, median (IQR), mg/dL	191.0 (169.0-215.0)	203.5 (183.0-228.5)	.001
Non-HDL-C, median (IQR), mg/dL	134.0 (113.0-156.0)	144.1 (117.2-168.0)	.02
Arterial hypertension, No. (%)	43 (28.3)	40 (26.3)	.78

Abbreviations: HDL-C, high-density lipoprotein cholesterol; HR, heart rate; RR, Riva-Rocci.

SI conversion factors: To convert total cholesterol and HDL-C to millimoles per liter, multiply by 0.0259.

[a] Calculated as weight in kilograms divided by height in meters squared.

test. Differences between defined groups were analyzed using unpaired *t*-tests or the Mann-Whitney *U* test.

Pearson correlation coefficients were calculated to evaluate the associations between changes in RVEF or LVEF and the number of marathon races in the last 10 years, baseline risk factors (prerace age and BMI), and blood parameters (prerace non-high-density lipoprotein cholesterol [non-HDL-C], increase in NT-proBNP level, and prerace vs immediately postrace).

Changes in the parameters of the RV over time were assessed using a linear mixed model with time as a fixed effect and a diagonal covariance matrix. Significant effects of time and post hoc contrasts were assessed by testing a given measurement against the prerace. Furthermore, the relative RVEF changes were classified into the following 3 categories for exploratory analysis: decline, increase, and test variability (unchanged), defined as when the RVEF prerace to 10-year follow-up declined by more than 2%, increased by more than 2%, and was between a decline and an increase of 2%, respectively.[11]

A significance level of 5% was used in all statistical tests. Statistical analyses are consistent with the Checklist for Statistical Assessment of Medical Papers (CHAMP) statement and were conducted using SPSS Statistics for Windows, version 25 (IBM Corp) and R software, version 4.1.0 (R Foundation for Statistical Computing).[14]

Results

Participant Characteristics

Of the 277 male participants, 152 (55%; mean [SD] age, 43 [3] years) were contacted and included in the follow-up study (**Figure 1**). Valid RV data were available for 143 of 152 participants (94.1%) at the 10-year follow-up. Paired RV data at baseline (V0) and at the 10-year follow-up (V4) were available for 109 of these 143 participants (76.2%). Furthermore, 51 participants had data from prerace until 3 days postrace, and 30 participants had data across all time points (Figure 1). The characteristics of the 277 initial participants have been published elsewhere.[9] The baseline characteristics of the participants adhering to the study requirements at baseline and follow-up are presented in **Table 1**, as are those of the participants at baseline included in the short-term follow-up (prerace until 3 days postrace) and long-term follow-up (eTables 1 and 2 in Supplement 1). The baseline characteristics of participants who did (n = 152) and did not (n = 125) participate in the 10-year follow-up (Pro-MagIC study) significantly differed only in age (mean [SD] age, 43 [3] vs 40 [9], respectively; *P* = .005) (eTable 3 in Supplement 1). Of the 152 participants who were included in the current analysis, 140 stated that they continued

Table 2. Changes in Functional Parameters From the Prerace Visit to the 10-Year Follow-Up Visit[a]

Parameters	V0	V1	V2	V3	V4	Mean difference (95% CI) V0/V1	V0/V2	V0/V3	V0/V4
Right ventricle, No.	191	62	62	51	143				
RVEF, %	52.4 (50.0 to 55.1)	47.6 (44.7 to 51.5)	50.7 (48.4 to 53.0)	51.3 (50.4 to 53.0)	51.9 (49.6 to 54.5)	−4.3 (−5.2 to −3.4)	−1.6 (−2.5 to −0.6)	−0.7 (−1.7 to 0.3)	−0.5 (−1.2 to 0.2)
P value	NA	NA	NA	NA	NA	<.001	.001	.18	.15
RVEDVi, mL/m^2	54.8 (46.2 to 66.8)	61.5 (54.5 to 71.6)	64.4 (53.9 to 74.3)	66.7 (58.5 to 75.8)	54.0 (46.4 to 62.9)	3.1 (0.1 to 6.1)	4.9 (1.9 to 7.9)	7.0 (3.8 to 10.3)	−1.3 (−3.5 to 1.0)
P value	NA	NA	NA	NA	NA	.04	.002	<.001	.27
RVESVi, mL/m^2	26.3 (21.3 to 31.7)	32.6 (27.1 to 37.9)	31.4 (26.0 to 37.7)	31.9 (26.0 to 37.6)	25.6 (21.7 to 30.9)	4.1 (2.6 to 5.7)	3.2 (1.7 to 4.8)	3.7 (2.0 to 5.4)	−0.3 (−1.4 to 0.9)
P value	NA	NA	NA	NA	NA	<.001	<.001	<.001	.648
Left ventricle, No.	198	187	183	160	136	NA	NA	NA	NA
LVEF, %	59.6 (55.6 to 64.5)	58.6 (54.5 to 62.6)	60.1 (55.5 to 64.2)	58.9 (55.7 to 63.4)	57.6 (54.1 to 61.6)	−1.6 (−2.7 to −0.6)	0.0 (−1.0 to 1.0)	−0.8 (−1.9 to 0.3)	−2.7 (−3.8 to −1.5)
P value	NA	NA	NA	NA	NA	.002	>.99	.16	<.001
LVEDVi, mL/m^2	51.2 (44.4 to 58.4)	50.5 (44.5 to 56.9)	53.3 (45.0 to 58.9)	49.5 (43.8 to 57.6)	61.7 (54.0 to 70.3)	−1.0 (−2.8 to 0.7)	0.8 (−0.9 to 2.6)	−0.4 (−2.2 to 1.5)	10.1 (8.1 to 12.1)
P value	NA	NA	NA	NA	NA	.25	.36	.68	<.001
LVESVi, mL/m^2	19.8 (17.3 to 23.7)	20.5 (17.9 to 24.9)	20.5 (17.1 to 24.3)	20.3 (17.2 to 23.6)	25.8 (21.3 to 31.4)	0.7 (−0.1 to 1.5)	0.7 (−0.2 to 1.5)	0.5 (−0.4 to 1.4)	6.0 (5.1 to 7.0)
P value	NA	NA	NA	NA	NA	.10	.13	.25	<.001
Diastology, No.	249	216	216	208	146				
E/A	1.5 (1.2 to 1.8)	1.0 (0.9 to 1.2)	1.5 (1.3 to 1.9)	1.6 (1.3 to 2.0)	1.2 (1.0 to 1.5)	−0.5 (−0.6 to −0.5)	0.0 (−0.0 to 0.1)	0.1 (0.1 to 0.2)	−0.3 (−0.4 to −0.2)
P value	NA	NA	NA	NA	NA	<.001	.44	<.001	<.001
E/e' lat	5.1 (4.3 to 6.1)	5.2 (4.3 to 6.2)	5.6 (4.8 to 6.6)	5.4 (4.5 to 6.4)	5.4 (4.5 to 6.4)	0.1 (−0.2 to 0.4)	0.4 (0.2 to 0.7)	0.4 (0.1 to 0.6)	1.2 (0.9 to 1.5)
P value						.186	.001	.007	<.001
E/e' med	7.7 (6.6 to 9.0)	7.7 (6.5 to 8.6)	8.0 (6.8 to 9.5)	8.2 (7.1 to 9.7)	8.4 (7.4 to 10.6)	−0.1 (−0.4 to 0.2)	0.2 (−0.1 to 0.5)	0.4 (0.1 to 0.7)	1.0 (0.6 to 1.4)
P value	NA	NA	NA	NA	NA	.57	.18	.02	<.001
Biomarkers, No.	251	216	217	212	146				
NT-proBNP, pg/mL	24.8 (13.3 to 43.9)	99.8 (61.1 to 174.7)	87.0 (61.8 to 145.1)	35.9 (21.24 to 58.8)	36.3 (21.6 to 67.4)	102.0 (92.1 to 111.8)	80.9 (71.0 to 90.7)	13.6 (3.7 to 23.6)	19.3 (8.0 to 30.7)
P value						<.001	<.001	.007	.001
Troponin T, ng/L[1]	3 (3.0 to 5.0)	33.7 (19.1 to 48.5)	9 (5.0 to 14.8)	3.5 (3.0 to 6.9)	7.0 (6.0 to 8.0)	39.9 (35.8 to 44.0)	7.3 (3.2 to 11.4)	1.4 (−2.7 to 5.5)	2.9 (−1.7 to 7.6)
P value	NA	NA	NA	NA	NA	<.001	.001	.51	.22

Abbreviations: E/A, E wave/A wave ratio; E/e', early diastolic mitral inflow velocity/early diastolic mitral annulus velocity; LVEF, left ventricular ejection fraction; LVEDVi, left ventricular end-diastolic volume/body surface area (Dubois); LVESVi, left ventricular end-systolic volume/body surface area (Dubois); NT-proBNP, N-terminal pro-brain natriuretic peptide; RVEDV, right ventricular end-diastolic volume/body surface area (Dubois); RVEF, right ventricular ejection fraction; RVESV, right ventricular end-systolic volume/body surface area (Dubois).

SI conversion factors: To convert NT-proBNP to nanograms per liter, multiply by 1; troponin to micrograms per liter, divide by 1000 and multiply by 1.

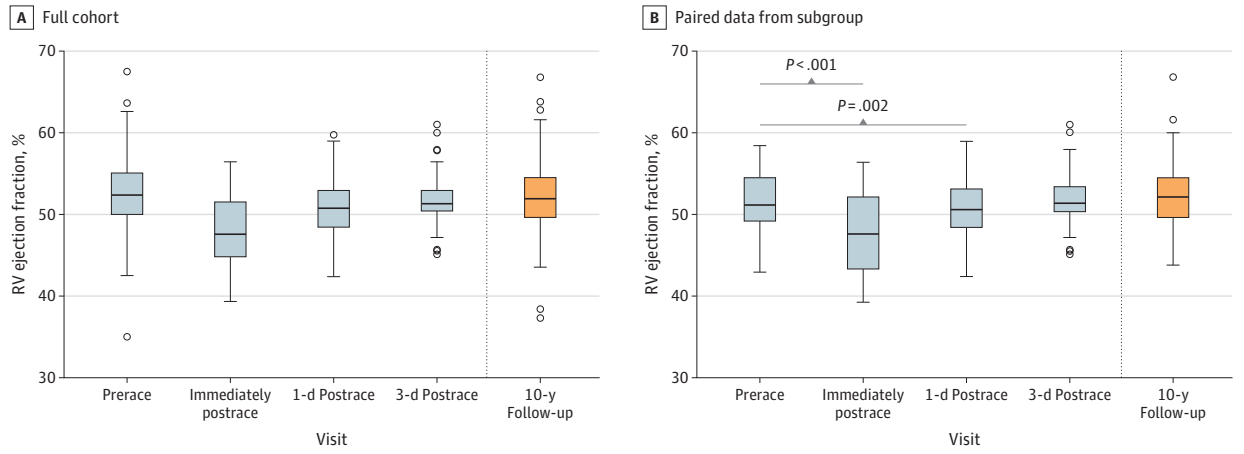
[a] V0, V1, V2, V3, and V4 represent the prerace, immediate postrace, 1-day postrace, 3-day postrace, and 10-year follow-up visits, respectively. Data are presented as median (IQR). Effect of the time and linear mixed-model analysis of each visit compared with prerace; post hoc corrections with Sidak are presented for each visit vs prerace. *P* values for the models are <.001.

to participate in other endurance races after the 2009 Munich Marathons and had a mean (SD) peak oxygen uptake (peak VO_2) of 42.6 (7.8) mL/kg/min at follow-up.

Changes in RV, LV, NT-proBNP, and TnT Level

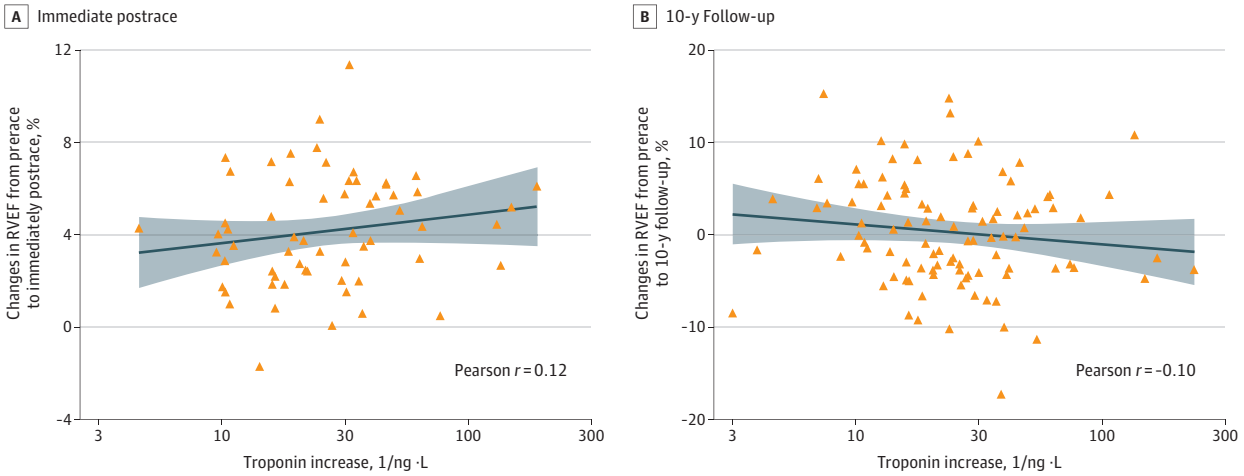
The RVEF declined significantly from the prerace (median, 52.4%; IQR, 50.0%-55.1%) to the immediate postrace (median, 47.6%; IQR, 44.7%-51.5%) assessment (n = 62; mean change, −4.2%; 95% CI, −4.8% to −3.6%; *P* < .001) (**Table 2** and eTable 4 in Supplement 1) and 1-day postrace (median, 50.7%; IQR, 48.4%-53.0%; *P* = .001) assessment but recovered at the 3-day postrace (median, 51.3%; IQR, 50.4%-53.0%; *P* = .18) assessment. The change in RVEF from the prerace to the 10-year follow-up (median, 51.9%; IQR, 49.6%-54.5%) was not significant (−0.5%; 95% CI, −1.2% to 0.2%; *P* = .15) (**Figure 2**). Paired data from prerace to 10-year follow-up are shown in eTable 5 in Supplement 1. Even after dividing the analyzed cohort into 3 groups, corresponding to individuals who no longer participated in any endurance race, those who continued to participate in 10 or more endurance races (high performers), and those who continued to participate in fewer than 10 endurance races (low performers), no significant differences were observed between the high performers and no participation groups (eTable 6 in Supplement 1). LVEF decreased (median, 59.6%; IQR, 55.6%-64.5% to median, 57.6%; IQR, 54.1%-61.6%; *P* < .001), whereas the E/e' lat increased

Figure 2. Right Ventricular (RV) Ejection Fraction at Prerace, Immediate Postrace, and Follow-Up Visits



A, Data from the full cohort (sample sizes at each visit identical to those in Table 2). B, Paired data from the subgroup of participants (n = 30) with complete measurements across all time points.

Figure 3. Association of the Exercise-Induced Troponin Increase With Right Ventricular Ejection Fraction (RVEF) changes



Changes at the immediate postrace (n = 62) (A) and 10-year follow-up (n = 101) (B) time points. Changes in exercise-induced troponin were calculated from that recorded immediately postrace to the concentration recorded prerace. Changes in RVEF were calculated from the RVEF recorded prerace to that quantified immediately postrace or at the 10-year follow-up visit.

(median, 5.1; IQR, 4.3-6.1 to median, 5.4; IQR, 4.5-6.4; $P < .001$) at the 10-year follow-up.

Changes in RV and LV volumes and function and TnT and NT-proBNP levels are shown in Table 2 and the eFigure in Supplement 1, and intergroup differences are presented in eTables 6 and 7 in Supplement 1. Sensitivity analysis for changes in systolic and diastolic LV function within the 3 subgroups showed no significance; likewise, no significant differences were observed between participants with less than 1500 km per year and those with greater than or equal to 1500 km per year. In addition, when participants were stratified by 10-year peak VO_2 (median split ≤41.7 vs >41.7 mL/kg/min), no significant differences in changes in RVEF or LVEF from prerace to 10-year follow-up were observed (eTable 8 in Supplement 1).

Associations Between TnT Level and RVEF or LVEF

The association between exercise-induced increase in TnT level, measured immediately after the marathon in 2009, and acute changes in RVEF showed no significant association (n = 62: Pearson $r = 0.12$, $P = .36$) (**Figure 3**A). After 10 years, the exercise-induced increase in TnT level during the marathon in 2009 was not significantly associated with the RVEF or LVEF change from prerace to the 10-year follow-up (n = 101: Pearson $r = -0.10$, $P = .35$ and Pearson $r = -0.09$, $P = .35$, respectively) (Figure 3B). The change in RVEF from prerace to the 10-year follow-up was not significantly associated with the number of marathon races (n = 96: Pearson $r = 0.06$; $P = .58$), cardiovascular risk factors at baseline (n = 109: age, Pearson $r = -0.17$, $P = .07$; n = 109: BMI, −0.08, $P = .42$; n = 98: non-HDL-C, Pearson $r = -0.06$,

P = .52), or increased NT-proBNP postrace (n = 109: Pearson r = −0.06, P = .55). The change in LVEF from prerace in 2009 to 10-year follow-up was not significantly associated with the number of marathon races (n = 94: Pearson r = −0.04, P = .72) and cardiovascular risk factors at baseline (n = 106: age, Pearson r = 0.08, P = .41; n = 106: BMI = 0.17, P = .09; n = 96: non-HDL-C, Pearson r = 0.08, P = .46), increase in NT-proBNP postrace (n = 106: Pearson r = 0.15, P = .13), or increase in TnT postrace (n = 106: Pearson r = −0.09, P = .35). Heart rate immediately postmarathon correlated with the acute change in E/A (Pearson r = −0.17, P = .02), and heart rate at 24 hours postmarathon correlated with the acute change in E/A (Pearson r = 0.17, P = .01). Furthermore, the acute change in heart rate from premarathon to postmarathon was significantly correlated with the acute change in E/A (Pearson r = −0.23, P = .002).

Clinical Changes in RVEF

Of the participants who completed the 10-year follow-up, a decline of more than −2% in RVEF was observed in 50 (46%), whereas in 46 (42%), the RVEF was increased (>2%), and in 13 (12%), it remained unchanged (−2% to 2%). An RVEF of less than 45% was observed in 5 participants (3.3%), of whom 3 had an RVEF of 44%. One participant with an RVEF of 44% presented with a higher grade of exercised-induced arrhythmia (ventricular and supraventricular) after 10 years of follow-up. Coronary angiography did not find occlusive coronary artery disease but a myocardial bridging of the left anterior descendent (LAD) coronary artery, and later pulmonary vein isolation was performed due to atrial fibrillation and flutter. The other 4 participants showed no clinically relevant arrhythmia at the 10-year follow-up. However, in 1 clinically asymptomatic participant with an RVEF of 37%, a 2-chamber pacemaker was implanted with permanent RV pacing due to congenital high-degree atrioventricular block at the 10-year follow-up.

Cardiovascular Adverse Events and Further Findings

No adverse events occurred during the 2009 Munich Marathons. Exercise-induced arterial hypertension was the most common cardiovascular clinical finding during the 10-year follow-up period (61.8% of the participants), followed by ventricular extrasystole (40.8%). Left atrial dilatation was observed in 45.4% of participants, and dilatation of the aorta was found in 9.2%. Atrial fibrillation was observed in 4 participants (2.6%), and arrhythmias during CPET or within the recovery phase were detected in 58 of 152 participants (39%), of whom 4 exhibited higher-grade exercise-induced arrhythmias. Among these 4, 1 participant presented with the aforementioned RVEF of 44%. All cardiovascular events are presented in eTable 9 in Supplement 1.

Discussion

This cohort study presents the first longitudinal data, to our knowledge, on the 10-year association of endurance exercise with cardiac function, with a particular focus on the RV in male runners, and assessed the influence of marathon-induced elevations in plasma TnT. These data suggest an acute reduction in RV function accompanied by an associated increase in plasma TnT levels directly after the marathon race; however, the study revealed no impairment of RV function after 10 years of training and long-distance races or associations with postrace increases in the plasma concentration of TnT. Therefore, these data suggest that long-term strenuous exercise workloads do not impair RV function in the vast majority of recreational endurance athletes.

RV Remodeling and Increase in Troponin Levels

We observed an acute decline in RVEF, associated with an increase in circulating TnT levels in our cohort after the marathon; however, RVEF did not change after 10 years, and no significant association was observed between RVEF and TnT level. Our findings are consistent with those of previous studies[15,16] showing that a large proportion of endurance athletes (>50%) experience transiently increased circulating levels of TnT and RV dysfunction after a demanding exercise workload; however, it is in contrast with those[3,6] showing an association between exercise-induced RV dysfunction and elevated plasma concentrations of cardiac biomarkers. Nevertheless, there is a debate on the clinical significance of exercise-induced troponin increase and long-term cardiovascular sequelae and whether increased troponin levels may predict adverse events. However, based on our data, exercise-associated increases in plasma TnT appear to be more benign in general and similar to those in skeletal muscle injury.[17]

Exercise-Induced Arrhythmogenic RV Cardiomyopathy

In our study, RVEF did not change significantly over the 10-year follow-up. These results are consistent with those of previous cross-sectional studies[18,19] in healthy volunteers, which did not reveal any consistent age-related decline in RVEF.

However, the phenomenon of EI-ARVC has been reported previously.[20] The pathophysiology of EI-ARVC is complex and not fully understood. The 2-hits hypothesis was postulated, stating that the combination of a genetic predisposition and high-volume exercise workloads may lead to the phenotypic expression of ARVC.[21] Noteworthy, in some symptomatic endurance athletes with arrhythmias originating in the RV, the prevalence of desmosomal gene variants was lower than expected.[22]

Generally, we could not confirm the consistent development of EI-ARVC in our cohort. Most of exercise-induced arrhythmia (in total, in 39% of the participants) were not higher grade. However, we found a single participant with a reduced RVEF less than 45% and higher-grade ventricular arrhythmia over the 10-year follow-up period. Coronary angiography revealed no obstructive coronary artery disease but did show myocardial bridging of the LAD. The arrhythmia may have represented precursors of the subsequently diagnosed atrial fibrillation and flutter. However, as differential diagnoses, an EI-ARVC or the myocardial bridging of the LAD were also considered, and the participant was advised to start β-blocker therapy.

LV Remodeling

We observed significant short- and long-term alterations in LVEF and diastolic function in our study. The short-term changes are consistent with a previous meta-analysis[23] of 372 athletes reporting a transient 2% decline in LVEF and alteration of diastolic function after prolonged and strenuous exercise.

Furthermore, we observed a decline in LVEF and a change in diastolic function over the 10-year follow-up. The values remained within the reference range for RT3-DE 60 ± 5% in nonathletes and 53% to greater than 74% in athletes without association to exercise-induced changes in TnT level.[24,25] Comparable with recently published data,[26] we observed no participants with the development of nonischemic symptomatic heart failure.

Our observed change in LVEF over 10 years is supported by the findings of a previous cross-sectional study by Cain et al[27] in a nonathletic population. In contrast to our results, a study[28] of 46 ultramarathon runners (mean [SD] age, 43 [7] years) showed that LVEF did not significantly decline over 10 years.

However, changes in LVEF are small, and the comparability of these studies with ours is limited due to different study designs and methods. Because diastolic function was also altered at the long-term follow-up (in line with previous studies on myocardial stiffness and ventricular compliance alterations with age in nonathletes), we hypothesized that the mild decline in LVEF over 10 years could be age related in our study population of recreational male endurance athletes.[29-31]

Study Limitations

This study had some limitations. First, our study included only male participants, which limited its applicability to the entire population. Furthermore, we could not include all participants from the original marathon race cohort for a 10-year follow-up. Thus, we cannot exclude the possibility of cardiovascular events in patients in whom these events were not reported. Our data could have been improved by multimodal imaging, including cardiovascular magnetic resonance, thereby assessing tissue characterization and overcoming the limitations of echocardiography. However, RT3-DE is an adequate tool for determining the LV and RV changes.[11,32-34]

Conclusions

Results of this cohort study showed that the increase in TnT levels and a transient decline in RVEF acutely associated with a marathon race were not prognostic indicators of long-term RV dysfunction in endurance athletes. Nonetheless, LVEF was significantly decreased, and LV diastolic function was impaired at the 10-year follow-up; however, values remained within normal limits. Therefore, these findings suggest that repetitive strenuous exercise training and endurance competitions do not result in long-term deterioration of RV function in the vast majority of recreational male endurance athletes.

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