

The role of sex, training load, and sports type in athletic cardiac remodelling: Insights from T1 and T2 mapping via cardiac magnetic resonance

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ABSTRACT

Background: Cardiovascular magnetic resonance (CMR) imaging, utilising native T1 and T2 mapping, provides a non-invasive method for assessing myocardial tissue properties, contributing to the clinical evaluation of the athlete's heart.

Objective: To evaluate T1 and T2 mapping alterations and their association with sex, training volume, sports type, and other standard CMR parameters of the athlete's heart.

Methods: We conducted a cross-sectional analysis of healthy elite athletes (≥ 10 training hours/week) and sedentary controls (≤ 5 h/week) who underwent detailed cardiology screening. CMR was performed, and native T1 and T2 values were quantified.

Results: Of the 199 healthy participants (115 elite athletes, 24 ± 5 years, 70 % males; 84 sedentary volunteers, 26 ± 3 years, 58 % males), athletes had higher ventricular volumes, left ventricular mass (LVMI), and lower ejection fractions than volunteers. Athletes showed lower T1 values (male athletes: 941 ± 23 ms vs. 960 ± 21 ms, $p < 0.01$; female athletes: 970 ± 20 ms vs. 982 ± 25 ms, $p < 0.01$). T1 negatively correlated with training hours and LVMI (Rho: -0.554 , $p < 0.001$). T1 values were positively associated with female sex with 22 ms (CI 14.3, 29.7, $p < 0.001$) higher values than males, while each additional hour in weekly exercise volume was associated with a 0.5 ms (CI -0.84 , -0.11 , $p = 0.011$) decrease. Compared to strength and mixed athletes, endurance athletes showed more pronounced myocardial adaptation, reflected in lower T1.

Conclusion: Sex, training volume, and type of sport significantly influence CMR-derived T1 and T2 values. This study highlights the critical need for sex- and sport-type-specific reference ranges in assessing myocardial remodelling in athletes, facilitating the distinction between benign athletic remodelling and (early) pathological changes.

1. Introduction

Frequent and intensive exercise induces changes in the structural and electrophysiological adaptations of the heart, collectively referred to as

the 'athlete's heart'. However, the early manifestations of certain myocardial diseases, such as dilated cardiomyopathy (DCM), hypertrophic cardiomyopathy (HCM), and arrhythmogenic cardiomyopathy (ACM), may overlap with the structural changes associated with regular

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exercise, complicating the distinction between physiological and pathological alterations [1–6].

Cardiac magnetic resonance imaging (CMR) is ideally suited for the assessment of subtle cardiac changes. Owing to its precision, whole-heart coverage, and high reproducibility, it has been extensively employed to distinguish between physiological cardiovascular adaptations and pathological conditions. Native T1 and T2 mapping can provide quantitative characterization of tissue properties without the need for contrast material administration [7]. Myocardial necrosis, fibrosis, amyloid deposition, and edema may result in elevated T1 mapping values, whereas lipid and iron accumulation are associated with decreased T1 mapping values. T2 mapping, which is more specific than T1, is particularly useful in identifying myocardial edema across various clinical settings [8].

The relationship between native T1 mapping and physical activity remains unclear. Some authors have reported decreased T1 values among male athletes compared to the general population [9,10]. In contrast, other studies have shown no effect for female athletes, or even increased T1 values among middle-aged endurance athletes [11–15]. Moreover, most studies focus on male athletes, and data on adaptation of female athletes remains insufficient, despite female athletes constituting half of the elite athlete population [16]. Athletes are often compared with controls without specific considerations of the individual exercise levels among the control group, even though sports participation is increasingly popular among the general population. To efficiently utilise mapping tools for distinguishing between physiological adaptation and disease, it is essential to establish a clear baseline for athletes in terms of native T1 and T2 mapping, and to understand the broader trends in the relationship between mapping values and sport-specific adaptations.

Our aim was to characterise T1 and T2 mapping in the context of cardiovascular adaptation in a clinically verified healthy population of highly trained athletes and less active individuals. We sought to investigate the association between T1 and T2 mapping values and covariates such as sex, age, training load, sport discipline, and conventional CMR metrics of cardiac remodelling. Additionally, we aimed to establish sex-specific reference ranges for T1 and T2 mapping in the athletic population.

2. Methods

2.1. Study group

The participants in this study comprised healthy volunteers and athletes who had previously taken part in our institutional cardiology screening programme and underwent CMR examination between July 2019 and June 2023. Ethical approval was obtained from the National Public Health Centre (No: ETT TUKEB IV/10282–1/2020/EKU). All participants provided written informed consent for both the examination and data collection.

All participants were required to complete a questionnaire concerning their medical history, cardiovascular complaints, and sporting activity. A digital 12-lead ECG was performed prior to the CMR examinations. None of the participants reported a history of systemic or cardiovascular diseases, including hypertension, diabetes mellitus, renal disease, or a family history of genetically predisposed conditions, such as cardiomyopathies. All participants denied the use of illicit drugs.

Based on the 2020 European Society of Cardiology Guidelines, athletes who engaged in high levels of sports activity (>10 h per week), were classified as healthy elite athletes, all competing at an international level in various disciplines, such as strength, endurance, or mixed sports [17]. All healthy volunteers participated in ≤5 h of sporting activity per week.

2.2. CMR protocol

All CMR examinations were performed using a 1.5 Tesla magnetic

resonance imaging (MRI) scanner (Magnetom Aera, Siemens Healthcare, Erlangen, Germany). The standardized CMR protocol included the following sequences: balanced-SSFP cine sequences in four-chamber, two-chamber, and three-chamber long-axis (LA) views, as well as a short-axis (SA) stack from the cardiac base to the apex, providing complete coverage of the left and right ventricles. Native T1 mapping relaxation times were measured using long-T1 5(3)3 and short-T1 5(3)3 Modified Look-Locker Inversion recovery (MOLLI) sequences, which are insensitive to heart rate artefacts [18]. T2 mapping was performed using T2-prep balanced steady-state free precession (b-SSFP) in a subset of short-axis images covering the ventricular base, mid-ventricle, and apex.

2.3. Image analysis

All post-processing analyses were performed using the Medis Suite Software (Medis Medical Imaging Software, Netherlands). Left ventricular (LV) and right ventricular (RV) volumes, functions, and masses were calculated from the short-axis (SA) stack using artificial intelligence-based automated contour detection (autoQ application), with manual adjustments made when necessary. Maximal end-diastolic wall thicknesses (EDWT) were assessed and measured manually in each subject. Myocardial native T1 and T2 relaxation times were measured using two segmentation approaches: gold standard septal regions of interest (ROIs) were defined in the mid-ventricular or basal slices (if the mid-ventricular images were technically inadequate for analysis) to measure septal mapping parameters in accordance with SCMR guidelines. ROIs were conservatively sized to reduce susceptibility artefacts from adjacent tissues and to ensure accurate measurements. Specifically, small ROIs (<20 pixels) were avoided, and the ROI size was standardized to approximately 1–2 cm², following best practice recommendations. [19,20] Endocardial and epicardial borders were drawn on basal, mid-ventricular, and apical parametric maps for each subject. The anterior RV insertion point was then defined to automatically divide the slices into 16 segments (excluding the apex), adapted from the American Heart Association (AHA) 17-segment model, to calculate segmental native T1 and T2 values of the LV. After removing segments affected by artefacts, the average relaxation time for the entire myocardium was calculated from the mean of the remaining segments. T1 and T2 mapping inter- and intraobserver agreement was assessed in 20 randomly selected participants. Two observers simultaneously performed analyses to evaluate inter-observer variability, while one observer repeated the analysis after a six-month interval to assess intra-observer variability.

2.4. Statistical analysis

The Shapiro-Wilk test was applied to assess the normality of the data for each continuous variable. Continuous variables that followed a normal distribution are presented as mean and standard deviation (SD), while those that did not (non-normal distribution) are reported as medians and interquartile ranges (IQRs). Categorical variables are presented as frequencies and percentages. Bland-Altman plots were generated to assess inter-method consistency, moreover intraclass correlation coefficients (ICC) were calculated to assess both inter- and intra-observer reliability. Comparisons between participant groups were conducted using independent samples *t*-tests for normally distributed continuous data; sport categories were compared using one-way ANOVA. Chi-squared tests were employed to compare the distributions of categorical data. Correlations were assessed using Spearman's rank test. Associations between mapping values, clinical characteristics and standard CMR parameters were analysed using linear regression analysis for greater comparability. We report raw and standardized coefficients and their corresponding 95 % confidence interval to ensure greater comparability across methods. Forest plots were used to visually summarize effect sizes and confidence intervals. All probability values were two-sided, and *p*-values <0.05 were considered statistically significant.

3. Results

3.1. Baseline characteristics

The study cohort comprised 199 healthy participants, including 115 elite athletes (24 ± 5 years, 70 % males) and 84 sedentary healthy volunteers (26 ± 3 years, 58 % males). None of the participants exhibited borderline or pathological ECG alterations indicative of underlying cardiac pathology.

On average, the athletes performed 20 ± 6 h of training per week, with a mean training experience of 16 years, participating in mixed (53 %), endurance (30 %), and power (17 %) disciplines (Table 1). The healthy volunteers exercised for an average of 2.2 ± 1.8 h per week, with 68 % engaging in regular recreational activity and 32 % not participating in any sports activity.

3.2. Concordance of mapping values across different measurement techniques

Overall, the gold standard septal ROI measurements for T1 mapping demonstrated a strong agreement with the global mid-ventricular endo-epicardial segmentation-derived values (Supplementary Fig. 1). The mean differences in T1 values from the basal slices were slightly higher compared to those from the mid-ventricular slices. Segmental values from the septal regions exhibited strong agreement, whereas the lateral segments displayed greater variability. The endocardial and epicardial contour ROI measurement method for T2 mapping parameters showed minimal mean differences, indicating a high level of consistency (Supplementary Fig. 2). For the analysis of T1 and T2 mapping, the ROI technique was generally used unless otherwise specified. Intra- and interobserver variability analyses showed excellent agreement, further supporting the robustness and reproducibility of the ROI-based measurement approach for T1 and T2 mapping (Supplementary Fig. 3, Supplementary Table 1).

3.3. Comparison of CMR characteristics between athletes and healthy volunteers stratified by sex

Both male and female elite athletes demonstrated increased left ventricular (LV) and right ventricular (RV) volumes, as well as greater myocardial mass, compared to healthy volunteers (Table 2). They also exhibited lower resting LV and RV ejection fractions (male athletes vs. healthy volunteers LVEF: 56 ± 4 vs. 60 ± 4 ; RVEF 55 ± 4 vs. 58 ± 5 ; $p < 0.001$).

Table 1
Baseline characteristics.

Variable	Healthy volunteers (n = 84)	Healthy athletes (n = 115)
Age (years)	26 ± 3	24 ± 5
Male	49(58 %)	80(70 %)
Female	35(42 %)	35(30 %)
HR (bpm)	72 ± 13	61 ± 11
Height (cm)	176 ± 9	182 ± 11
Weight (kg)	72 ± 13	82 ± 15
Sport type (power)	NA	19(17 %)
Sport type (mixed)	NA	61(53 %)
Sport type (endurance)	NA	35(30 %)
Sport type (recreational)	57(68 %)	NA
No sport	27(32 %)	NA
Training hours (hours/week)	2 ± 2	20 ± 6
Training years	NA	16 ± 6

The table presents age, sex distribution, heart rate (HR), height, weight, weekly training hours, and years of training. For athletes, the distribution of sport types are also included. Continuous data are presented as mean $\pm$ standard deviation, and categorical variables as frequency (percentage). NA indicates not applicable.

0.001). In linear models, female sex was associated with higher mapping values (Supplementary Fig. 4, Supplementary Table 2), independent of exercise conditioning (on average by 32 ms for T1, and by 2 ms for T2; $p < 0.001$). Therefore, we analysed mapping values stratified by sex, similar to standard volumetric measurements. Athletes had lower native T1 values compared to healthy volunteers (T1 male athletes: 941 ± 23 ms vs. 960 ± 21 ms; T1 female athletes: 970 ± 20 vs. 982 ± 25 ms; $p < 0.01$ for both). T1 values ranged (5–95 percentile) from 900 to 983 ms in healthy elite males and from 925 to 1006 ms in female elite athletes. In male elite athletes, T2 values were slightly lower compared to healthy volunteers (42 ± 2 vs. 44 ± 2 ms; $p = 0.005$). However, no such difference was observed between female athletes and their less active counterparts.

Both male athletes and healthy volunteers presented with lower global T1 and T2 mapping values compared to females (Supplementary Fig. 5). Segmental T1 and T2 mapping values were represented on a modified 16-segment AHA model (Supplementary Fig. 6, 7).

3.4. Comparison of T1 mapping values in left ventricular hypertrophy

In male elite athletes, there was no significant difference between the group with a maximal wall thickness of <12 mm (mean: 941 ms) and the group with a maximal wall thickness of ≥ 12 mm (mean: 941 ms), $p = 0.867$ (Supplementary Fig. 8).

3.5. Comparison of CMR characteristics in power vs. mixed vs. endurance athletes

The CMR parameters of male athletes, stratified by different sport disciplines, showed that LVESVi, LVMI were significantly lower in power athletes compared to those in mixed and endurance sports, while LVEF was higher in power athletes (LVEF: 60.3 %) than in the other groups ($p < 0.05$). T1 mapping values were lower in endurance athletes compared to mixed and power athletes (Supplementary Table 3). For female athletes, RVEDVi, RVESVi, LVMI values were higher in endurance athletes than in power and mixed athletes ($p < 0.05$ for all). Similar to findings in male athletes, T1 mapping values for female endurance athletes were lower compared to the other groups (Supplementary Table 4).

3.6. Associations of T1 and T2 mapping

We identified a negative correlation between T1 and T2 values and weekly training hours, LVEDVi, LVESVi, LVSVi, and LVMI (Fig. 1). Additionally, there was a negative correlation between T1 values and heart rate, as well as LVEF. T1 values demonstrated a stronger correlation with the aforementioned factors compared to T2 values. The association was particularly pronounced with LVMI (Rho: -0.554 , $p < 0.001$, Fig. 1).

We quantified the associations between T1 and T2 mapping values and key covariates using multivariate linear regression models (Table 3). T1 mapping values demonstrated a strong positive association with female sex, with women exhibiting, on average, 22 ms higher T1 values than age-, training load-, and myocardial mass-adjusted male counterparts (B coefficient [95 % CI]: $22[14.26, 29.73]$; $p < 0.001$). In contrast, each additional hour of exercise was associated with a 0.5 ms decrease in T1 values ($-0.47[-0.84, -0.11]$; $p < 0.01$), even after adjusting for the effects of age, sex, heart rate (HR), and LVMI. T2 mapping values showed a positive association with increasing age and female sex. However, when adjusted for LVMI, exercise load showed no significant association with T2 mapping values (Table 3).

4. Discussion

Our study of 199 young individuals characterises the native myocardial tissue properties of healthy elite athletes and healthy

Table 2
Comparison of healthy volunteers and athletes.

CMR variables	Male					Female				
	Elite athletes (n = 80)		Healthy volunteers (n = 49)		P value	Elite athletes (n = 35)		Healthy volunteers (n = 35)		P value
	Mean ± SD	95 % range	Mean ± SD	95 % range		Mean ± SD	95 % range	Mean ± SD	95 % range	
LVEDVi (ml/m ²)	118.5 ± 13.3	95.4–144.5	95.1 ± 10.7	73.5–112.4	<0.001	104.4 ± 7.9	92.7–118	85.6 ± 10	67.1–103	<0.001
LVESVi (ml/m ²)	51.9 ± 8.4	36–69	38.5 ± 6.3	27.2–49	<0.001	43.2 ± 5.9	32.5–51.9	34.5 ± 6.4	25.1–50.6	<0.001
LVSVi (ml/m ²)	66.5 ± 8.1	54–85	56.5 ± 6.9	43–70.2	<0.001	61 ± 6.6	50.4–73.4	52.1 ± 6.6	39.8–62.7	<0.001
LVEF (%)	56.3 ± 4.2	48–64	59.5 ± 4	53.3–67	<0.001	58.6 ± 4.3	50.8–66.5	60.8 ± 3.4	53.7–66.4	0.018
LVMi (g/m ²)	69.4 ± 13.8	51–100.6	51.3 ± 6.9	39.4–64.9	<0.001	52.9 ± 9	39.4–70.8	38.5 ± 4.8	29.9–48.5	<0.001
EDWT (mm)	10.8 ± 1.2	9–13	8.9 ± 0.8	8–10	<0.001	8.5 ± 1.1	7–10.1	7.2 ± 0.9	6–8.1	<0.001
RVEDVi (ml/m ²)	119.7 ± 14.7	92.4–149.8	95.8 ± 11.7	74.7–117.4	<0.001	103.4 ± 10.3	90–126.7	85.8 ± 11.4	68.4–110.5	<0.001
RVESVi (ml/m ²)	53.7 ± 9.4	36–70.1	40.5 ± 7.8	29–53.3	<0.001	44.4 ± 8.3	29.9–59	37 ± 12.5	24.7–59.4	0.005
RVSVi (ml/m ²)	66 ± 8.2	53.9–84	55.5 ± 7.3	40.4–70.6	<0.001	59.1 ± 6.1	50.5–70.2	50.6 ± 7.2	38.2–62.4	<0.001
RVEF (%)	55 ± 4	48–64	57.7 ± 5	51–66.8	0.002	57.3 ± 5.4	50.7–67.7	59 ± 4.1	51.8–66.2	0.142
RVMi (g/m ²)	19.8 ± 3.5	15–26.6	16.3 ± 2.7	11.2–21.1	<0.001	17.4 ± 3.7	10.5–23.1	14.8 ± 2.6	8.8–20.1	0.001
T1 ROI (msec)	941.4 ± 23.2	900–983.2	960.2 ± 20.5	917.8–991.8	<0.001	969.7 ± 19.6	924.8–1006.1	983.4 ± 16.6	952.4–1009.6	0.003
T1 mid-ventricular global myocardium (msec)	942.2 ± 23.8	899.5–991	954.7 ± 19.8	918.8–989.5	0.002	973.1 ± 22.7	913.5–1008.3	979 ± 20.6	943.9–1018.7	0.262
T1 mid-ventricular anteroseptal segment (msec)	941.4 ± 27.8	884.6–999.4	950.3 ± 24.9	891.8–989.9	0.062	976.6 ± 26.8	927.4–1027.3	982.4 ± 25.4	933.8–1020.8	0.37
T2 ROI (msec)	42.9 ± 1.9	40–46	43.8 ± 1.7	41–47	0.005	44.9 ± 2.1	41–48.3	45.6 ± 2.1	42–49.1	0.146
T2 mid-ventricular global myocardium (msec)	43.6 ± 1.8	41–46.8	44 ± 1.9	40.5–47.2	0.274	45.9 ± 2	42.6–49.2	46 ± 2.3	42.1–49.4	0.871
T2 mid-ventricular anteroseptal segment (msec)	44.3 ± 2.3	40.7–49	45.3 ± 2.4	41–49.7	0.037	47 ± 2.8	42.1–51.4	47.8 ± 3.1	43–53.3	0.289

The table presents mean values ± standard deviation (SD) for various CMR measurements, including left ventricular (LV) and right ventricular (RV) end-diastolic volume index (EDVi), end-systolic volume index (ESVi), stroke volume index (SVi), ejection fraction (EF), and end-diastolic wall thickness (EDWT). Additionally, it includes T1 region of interest (ROI), global and segmental mid-ventricular parameters and T2 ROI, global and segmental mid-ventricular values. The 95 % confidence range is also provided. Statistical significance is indicated by p-values, with $p < 0.05$ considered significant.

volunteers using native T1 and T2 mapping via CMR. We found that both male and female athletes had lower T1 values compared to their less active counterparts, with T2 values also being lower in male athletes, but not significantly different in females. A strong negative association was observed between weekly training hours and T1 and T2 mapping values. T1 and T2 values were generally higher in female participants across both groups. Notably, endurance athletes, particularly males, exhibited the lowest T1 mapping values compared to those in mixed and power sports, showing a more pronounced cardiovascular adaptation. Furthermore, T1 values did not differ significantly between male athletes who exhibited left ventricular hypertrophy and those who did not. Sex, training hours, and myocardial mass were identified as key predictors of T1 values.

Our findings indicate that when analyzing septal segments, T1 values derived from global mid-ventricular *endo*-epicardial contouring closely align with those obtained via septal ROI measurements. This concordance suggests that *endo*-epicardial contouring can serve as a reliable alternative to the ROI method in septal regions.

4.1. Myocardial remodelling in athletes

Our study confirmed that healthy elite athletes exhibit distinct myocardial remodelling, as assessed by CMR, in comparison to less active healthy individuals. This includes increased left and right ventricular volumes, stroke volumes, myocardial mass indices, and end-

diastolic wall thicknesses, all of which are indicative of the heart’s adaptive response to prolonged, intense physical activity. These adaptations are well-documented features of the ‘athlete’s heart’, reflecting enhanced cardiac output and efficiency [21–24].

4.2. Native T1 and T2 mapping and the ‘Athlete’s Heart’

Native T1 relaxation times serve as a non-invasive marker of myocardial tissue characteristics. In elite athletes, lower native T1 values have been observed compared to controls (e.g., 955 ms vs. 983 ms; $p < 0.05$ on 1.5 T), highlighting the adaptive changes of myocardial tissue in response to intense physical activity [25,26]. Shorter T1 times in athletes have been suggested to result from a relative increase in intracellular myocardial content due to training-induced remodelling. McDiarmid et al. compared CMR parameters in 30 male endurance athletes and 15 control subjects. [9] Their findings suggest that changes in the ratio of extracellular to intracellular volumes, particularly a decrease in extracellular volume, may explain the lower T1 mapping values observed in athletes.

However, not all studies have consistently reported lower T1 values in athletes. [11–15] For example, Görmeli et al. and others found no significant differences, or higher T1 mapping values in athletes compared to non-athletes [27]. These discrepancies could be attributed to variations in study design, cohort size, and the type and intensity of physical activity across studies.

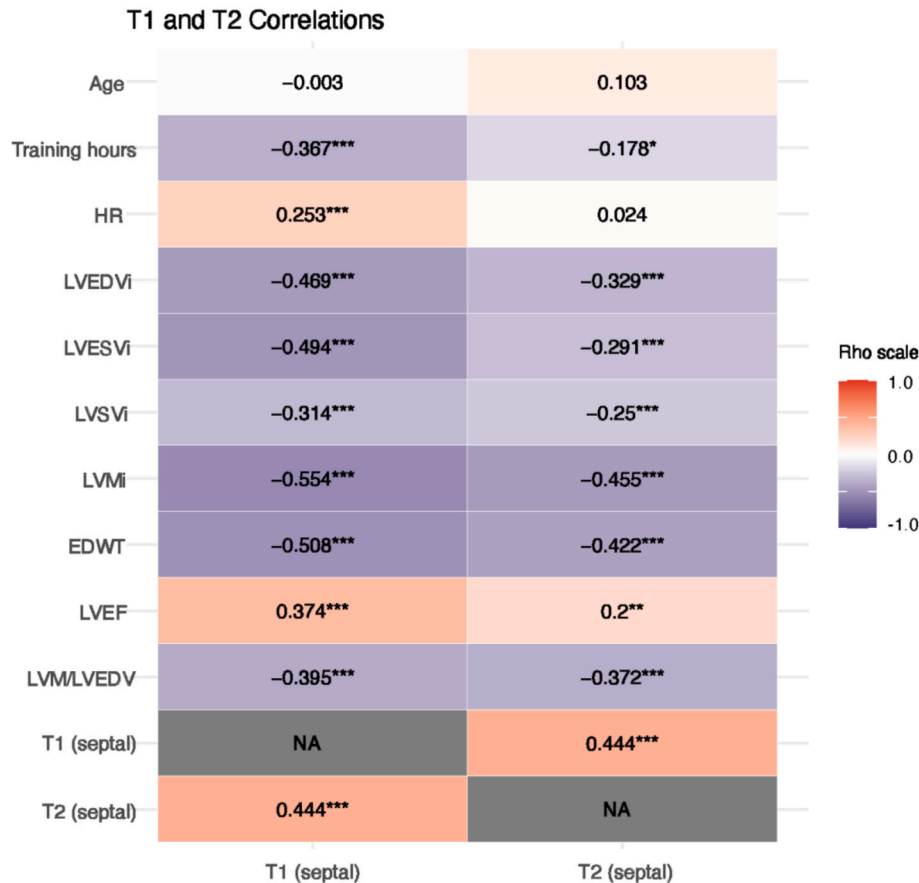


Fig. 1. Correlation between mapping values, standard CMR parameters and participant characteristics. The heatmap displays the correlation coefficients between T1 and T2 septal region of interest values and various factors, including age, training hours, heart rate (HR), left ventricular end-diastolic volume index (LVEDVi), left ventricular end-systolic volume index (LVESVi), left ventricular stroke volume index (LVSVi), left ventricular mass index (LVMi), end-diastolic wall thickness (EDWT), left ventricular ejection fraction (LVEF), and left ventricular mass to left ventricular end-diastolic volume ratio (LVM/LVEDV). Significant correlations are indicated with asterisks (*** $p < 0.001$, ** $p < 0.01$, * $p < 0.05$).

Table 3
Linear regression model for T1 and T2 mapping predictors.

T1 mapping predictors		
Variable	B coefficient [95 % CI]	P value
Age	0.431 [-0.297, 1.158]	0.244
Female sex	21.996 [14.26, 29.732]	<0.001
HR	0.12 [-0.128, 0.368]	0.341
Training hours	-0.474 [-0.838, -0.109]	0.011
LVMi	-0.368 [-0.646, -0.09]	0.01

T2 mapping predictors		
Variable	B coefficient [95 % CI]	P value
Age	0.11 [0.044, 0.175]	0.001
Female sex	1.606 [0.908, 2.304]	<0.001
HR	-0.024 [-0.046, -0.002]	0.034
Training hours	0.002 [-0.03, 0.035]	0.885
LVMi	-0.046 [-0.071, -0.021]	<0.001

The table presents the beta (B) coefficients and 95 % confidence intervals (CI) for the predictors of T1 and T2 mapping values derived from the linear regression model. Variables include age, female sex, heart rate (HR), training hours, and left ventricular mass index (LVMi). A p -value of less than 0.05 is considered statistically significant.

The observed difference in T1 values in athletes is relatively small compared to healthy controls, yet it may serve as an important marker in distinguishing between physiological and pathological conditions. For

instance, in Anderson-Fabry disease, native T1 values are significantly lower due to glycosphingolipid accumulation in the myocardium, and myocardial fat deposition also results in pronounced reductions in T1 times [28–30]. Conversely, hypertrophic cardiomyopathy (HCM) typically causes elevated native T1 values due to extracellular matrix expansion and myocyte disarray [31–33].

The decreased myocardial T2 relaxation times observed in male athletes compared to controls may result from the physiological adaptations associated with intense physical training. However, the lack of significant association between exercise load and T2 mapping values when adjusted for LVMi suggests that the observed differences in T2 times between athletes and controls are likely driven by factors other than exercise alone. Moreover, these results do not suggest exercise-induced inflammation in highly trained athletes.

4.3. Sex differences in myocardial adaptation to sports

Female sex was associated with higher T1 values, independent of age, training hours, and myocardial mass. This finding aligns with studies in sedentary cohorts, which report higher native T1 times in females compared to males [25]. These sex-specific differences in T1 mapping values underline the importance of using sex-specific reference ranges when interpreting mapping alterations. The limited data available on T1 mapping values in athletic populations is consistent with our findings. Research from Daems et al. and Prosperi et al. similarly identified lower T1 mapping values in male athletes compared to females. These results emphasise the necessity for sex-specific reference ranges in

athletes. [25,26]

Disregarding sex-specific reference ranges can result in significant misclassification of cardiovascular conditions and diagnoses in both males and females. A study by Thomas et al. demonstrated that applying a single reference range for both males and females misclassified up to 36 % of healthy females and 37 % of healthy males as having abnormal T1 values, and up to 16 % of healthy females and 12 % of healthy males as having abnormal T2 values [34]. Such misclassification can have serious consequences for the diagnosis of myocardial disease, potentially leading to unnecessary interventions or missed diagnoses. The authors highlight that normal myocardial T1 and T2 values differ significantly between sexes, further reinforcing the critical need for sex-specific reference ranges in clinical practice.

4.4. Influence of sport discipline

Endurance athletes exhibited the lowest T1 values, indicating extensive myocardial remodelling, which is consistent with the chronic volume overload typically seen in this group. [35–37] Similarly, Prosperi et al. reported lower T1 values in endurance athletes, when compared to other types of exercise. In contrast, power athletes demonstrated relatively higher T1 values. This supports the idea that the type and intensity of physical activity play a crucial role in the extent of cardiac remodelling. [26]

4.5. Correlation and predictors of mapping values

The inverse correlation between T1 and T2 values and weekly training hours highlights the dose-response relationship between physical activity and myocardial adaptation. This finding aligns with previous research, which suggests that increased exercise intensity is associated with shorter native T1 times, reflecting enhanced myocardial efficiency resulting from sustained training. [26,38] Additionally, our regression analyses identified key predictors of T1 and T2 mapping values, including age, body surface area (BSA), sex, heart rate (HR), and left ventricular mass. These factors have been shown to significantly influence cardiac structure and function, with studies demonstrating that left ventricular mass and training intensity correlate with native T1 times [36,38]. The importance of considering these variables in comprehensive assessments is emphasised by the potential for misclassification of physiological adaptations as pathological changes. Thus, tailoring assessments to individual characteristics and establishing sport-specific reference ranges is essential for improving diagnostic accuracy and guiding appropriate management strategies for athletes.

4.6. Implications for clinical practice/perspective

Our findings carry significant implications for the differentiation of physiological cardiac adaptations from pathological conditions in athletes. Establishing well-defined normal ranges for T1 and T2 mapping specific to athletes is essential for helping clinicians distinguish benign adaptations from potential early indicators of disease, such as cardiomyopathy. This distinction is critical for informed clinical decision-making, preventing unnecessary restrictions on athletic participation, and guiding appropriate medical interventions when warranted. While the observed changes in T1 and T2 values in athletes generally fall within normal limits, their clinical utility extends beyond simple categorization. When conventional assessments yield borderline results, T1 and T2 mapping can serve as a valuable adjunct tool, improving diagnostic accuracy. Therefore, integrating these techniques into routine cardiovascular evaluations, supported by well-established reference values, provides a more nuanced and individualized approach to understanding cardiac remodelling in athletes. Defining and standardizing these normal values is crucial to ensuring accurate interpretation and optimal application in sports cardiology.

5. Conclusion

In conclusion, this study offers a comprehensive insight into the myocardial tissue characteristics of elite athletes, employing native T1 and T2 mapping via CMR. The reduction observed in T1 and T2 values underscore the significant myocardial remodelling that occurs as a result of high-intensity physical activity. The establishment of sport-specific reference values is crucial in enabling clinicians to distinguish between benign athletic remodelling and early pathological changes.

5.1. Limitations

Our study has the following limitations: the cross-sectional design prevents the establishment of causality, and the potential influence of unmeasured confounding factors cannot be entirely excluded. Furthermore, the reliance on a single CMR scanner model may limit the generalisability of device-specific normal values. Although there was an imbalance in sex distribution between the athlete and sedentary groups, we stratifying our analyses ensures the validity of our results.

Declaration of generative AI and AI-assisted technologies in the writing process

During the preparation of this work the author(s) used [ChatGPT 4o model] for text editing purposes only. After using this tool/service, the author(s) reviewed and edited the content as needed and take(s) full responsibility for the content of the publication.

CRediT authorship contribution statement

Dorottya Balla: Writing – review & editing, Writing – original draft, Visualization, Investigation, Formal analysis, Data curation, Conceptualization. **Liliana Szabo:** Writing – review & editing, Writing – original draft, Visualization, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Francesca Graziano:** Writing – review & editing, Investigation. **Csongor Mesko:** Writing – review & editing, Investigation, Data curation. **Zsolia Dohy:** Writing – review & editing, Investigation, Formal analysis. **Vencel Juhasz:** Writing – review & editing, Investigation, Data curation, Conceptualization. **Dina Amirifard:** Writing – review & editing, Investigation. **Nora Sydo:** Writing – review & editing, Investigation. **Emese Csulak:** Writing – review & editing, Investigation. **Iván Petrov:** Writing – review & editing, Investigation. **Orsolya Kiss:** Writing – review & editing, Investigation. **Mark Zamodics:** Writing – review & editing, Investigation. **Mate Babity:** Writing – review & editing, Investigation. **Bela Merkely:** Investigation, Formal analysis. **Hajnalka Vago:** Writing – review & editing, Supervision, Investigation, Formal analysis, Conceptualization.

Declaration of competing interest

None.

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Appendix A. Supplementary data

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References

- [1] E. Androulakis, P.P. Swoboda, The role of cardiovascular magnetic resonance in sports cardiology; current utility and future perspectives, *Curr. Treat. Options Cardiovasc. Med.* 20 (2018).
- [2] S. Palermi, et al., Athlete's heart: a cardiovascular step-by-step multimodality approach, *Rev. Cardiovasc. Med.* 24 (2023), <https://doi.org/10.31083/j.rcm2405151>. Preprint at.
- [3] C. Czimbalmos, et al., The demanding grey zone: sport indices by cardiac magnetic resonance imaging differentiate hypertrophic cardiomyopathy from athlete's heart, *PLoS One* 14 (2019).
- [4] M. Galderisi, et al., Themulti-modality cardiac imaging approach to the Athletes heart: an expert consensus of the European association of cardiovascular imaging, *Eur. Heart J. Cardiovasc. Imaging* 16 (2015), 353-353T.
- [5] F. Graziano, et al., May strenuous endurance sports activity damage the cardiovascular system of healthy Athletes? A narrative review, *J. Cardiovasc. Dev. Dis.* 9 (2022), <https://doi.org/10.3390/jcdd9100347>. Preprint at.
- [6] L. Szabo, et al., Certainties and uncertainties of cardiac magnetic resonance imaging in athletes, *J. Cardiovasc. Dev. Dis.* 9 (2022), <https://doi.org/10.3390/jcdd9100361>. Preprint at.
- [7] G. Constantine, K. Shan, S.D. Flamm, M.U. Sivananthan, Role of MRI in clinical cardiology, *Lancet* 363 (2004) 2162-2171.
- [8] D.R. Messroghli, et al., Clinical recommendations for cardiovascular magnetic resonance mapping of T1, T2, T2 and extracellular volume: A consensus statement by the Society for Cardiovascular Magnetic Resonance (SCMR) endorsed by the European Association for Cardiovascular Imaging (EACVI), *J. Cardiovasc. Magn. Reson.* 19 (2017), <https://doi.org/10.1186/s12968-017-0389-8>.
- [9] A.K. McDiarmid, et al., Athletic cardiac adaptation in males is a consequence of elevated myocyte mass, *Circ. Cardiovasc. Imaging* 9 (2016).
- [10] S. Pujadas, et al., Myocardial remodelling and tissue characterisation by cardiovascular magnetic resonance (CMR) in endurance athletes, *BMJ Open Sport Exerc. Med.* 4 (2018).
- [11] C.A. Görmeli, et al., Assessment of myocardial changes in athletes with native T1 mapping and cardiac functional evaluation using 3 T MRI, *Int. J. Cardiovasc. Imaging* 32 (2016) 975-981.
- [12] L.A. Malek, et al., Cardiovascular magnetic resonance with parametric mapping in long-term ultra-marathon runners, *Eur. J. Radiol.* 117 (2019) 89-94.
- [13] O. Missenard, et al., Absence of cardiac damage induced by long-term intensive endurance exercise training: a cardiac magnetic resonance and exercise echocardiography analysis in masters athletes, *Am. J. Prev. Cardiol.* 7 (2021) 100196.
- [14] P.P. Swoboda, et al., Assessing Myocardial Extracellular Volume by T1 Mapping to Distinguish Hypertrophic Cardiomyopathy from Athlete's Heart, *J. Am. Coll. Cardiol.* 67 (2016) 2189-2190, <https://doi.org/10.1016/j.jacc.2016.02.054>.
- [15] I. Mordi, D. Carrick, H. Bezerra, N. Tzemos, T1 and T2 mapping for early diagnosis of dilated non-ischaemic cardiomyopathy in middle-aged patients and differentiation from normal physiological adaptation, *Eur. Heart J. Cardiovasc. Imaging* 17 (2016) 797-803.
- [16] GenderEqualOlympics: Celebrating full gender parity on the field of play at Paris 2024. <https://olympics.com/ioc/news/genderequalolympics-celebrating-full-gender-parity-on-the-field-of-play-at-paris-2024>, 2024.
- [17] A. Pelliccia, et al., 2020 ESC guidelines on sports cardiology and exercise in patients with cardiovascular disease, *Eur. Heart J.* 42 (2021) 17-96. Preprint at, <https://doi.org/10.1093/eurheartj/ehaa605>.
- [18] G. Reiter, C. Reiter, C. Kräuter, M. Fuchsjäger, U. Reiter, Cardiac magnetic resonance T1 mapping. Part 1: Aspects of acquisition and evaluation, *Eur. J. Radiol.* 109 (2018) 223-234. Preprint at, <https://doi.org/10.1016/j.ejrad.2018.10.011>.
- [19] J. Schulz-Menger, et al., Standardized image interpretation and post-processing in cardiovascular magnetic resonance - 2020 update: Society for Cardiovascular Magnetic Resonance (SCMR): Board of Trustees Task Force on Standardized Post-Processing, *J. Cardiovasc. Magn. Reson.* 22 (2020), <https://doi.org/10.1186/s12968-020-00610-6>.
- [20] D.R. Messroghli, et al., Clinical recommendations for cardiovascular magnetic resonance mapping of T1, T2, T2 and extracellular volume: A consensus statement by the Society for Cardiovascular Magnetic Resonance (SCMR) endorsed by the European Association for Cardiovascular Imaging (EACVI), *J. Cardiovasc. Magn. Reson.* 19 (2017), <https://doi.org/10.1186/s12968-017-0389-8>.
- [21] A. La Gerche, A.J. Taylor, D.L. Prior, Athlete's heart: the potential for multimodality imaging to address the critical remaining questions, *JACC Cardiovasc. Imaging* 2 (2009) 350-363.
- [22] I. Csics, et al., The impact of sex, age and training on biventricular cardiac adaptation in healthy adult and adolescent athletes: cardiac magnetic resonance imaging study, *Eur. J. Prev. Cardiol.* 27 (2020) 540-549.
- [23] R. Bryde, et al., Cardiac structure and function in elite female athletes: a systematic review and meta-analysis, *Phys. Rep.* 9 (2021).
- [24] D.L. Prior, A. La Gerche, The athlete's heart, *Heart* 98 (2012) 947-955.
- [25] D. Jjn, et al., 1076 Elite Athlete Status, Gender and Mitchell Sports Classification Strongly Influence Native T1 Mapping Times. https://academic.oup.com/eurjpc/article/30/Supplement_1/zwad125.035/7175728.
- [26] S. Prosperi, et al., CMR native T1 and T2 Mapping in Olympic athletes: the influence of sport discipline and sex. 1 Running Title: CMR native T1 and T2 mapping in Olympic athletes. 2 3 4, 2025, <https://doi.org/10.1093/ehjci/jeae247/7764168>.
- [27] C.A. Görmeli, et al., Assessment of myocardial changes in athletes with native T1 mapping and cardiac functional evaluation using 3 T MRI, *Int. J. Card. Imaging* 32 (2016) 975-981.
- [28] A. Ponsiglione, et al., Cardiovascular magnetic resonance native T1 mapping in Anderson-Fabry disease: a systematic review and meta-analysis, *J. Cardiovasc. Magn. Reson.* 24 (2022), <https://doi.org/10.1186/s12968-022-00859-z>. Preprint at.
- [29] D.M. Sado, et al., Identification and assessment of Anderson-fabry disease by cardiovascular magnetic resonance noncontrast myocardial T1 mapping, *Circ. Cardiovasc. Imaging* 6 (2013) 392-398.
- [30] R.B. Thompson, et al., T1 mapping with cardiovascular MRI is highly sensitive for fabry disease independent of hypertrophy and sex, *Circ. Cardiovasc. Imaging* 6 (2013) 637-645.
- [31] S. Dass, et al., Myocardial tissue characterization using magnetic resonance noncontrast T1 mapping in hypertrophic and dilated cardiomyopathy, *Circ. Cardiovasc. Imaging* 5 (2012) 726-733.
- [32] V.O. Puntmann, et al., Native T1 Mapping in Differentiation of Normal Myocardium from Diffuse Disease in Hypertrophic and Dilated Cardiomyopathy, 2013.
- [33] Reiter, G., Reiter, C., Kräuter, C., Fuchsjäger, M. & Reiter, U. Cardiac magnetic resonance T1 mapping. Part 1: Aspects of acquisition and evaluation. *Eur. J. Radiol.* vol. 109 223-234 Preprint at doi: <https://doi.org/10.1016/j.ejrad.2018.10.011> (2018).
- [34] K.E. Thomas, et al., Misclassification of females and males in cardiovascular magnetic resonance parametric mapping: the importance of sex-specific normal ranges for diagnosis of health vs. disease, *Eur. Heart J. Cardiovasc. Imaging* 25 (2024) 339-346.
- [35] B. Brown, et al., Left ventricular remodeling in elite and sub-elite road cyclists, *Scand. J. Med. Sci. Sports* 30 (2020) 1132-1139.
- [36] L.A. Malek, et al., Cardiovascular magnetic resonance with parametric mapping in long-term ultra-marathon runners, *Eur. J. Radiol.* 117 (2019) 89-94.
- [37] I. Csics, et al., The impact of sex, age and training on biventricular cardiac adaptation in healthy adult and adolescent athletes: cardiac magnetic resonance imaging study, *Eur. J. Prev. Cardiol.* 27 (2020) 540-549.
- [38] A.K. McDiarmid, et al., Athletic cardiac adaptation in males is a consequence of elevated myocyte mass, *Circ. Cardiovasc. Imaging* 9 (2016).