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Reference Values of Myocardial Structure, Function and Tissue Composition by Cardiac Magnetic Resonance in Healthy African Americans at 3T and their Relations to Serologic and Cardiovascular Risk Factors

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Abstract

Cardiac magnetic resonance (CMR) is a standard of reference for cardiac structure and function. Recent advances in T1 mapping and spectroscopy also provide assessment of myocardial tissue composition. However, the reference ranges of left ventricular parameters have rarely been assessed in an African American (AA) population without known cardiac disease. To estimate the reference values of myocardial structure, function and tissue composition by CMR and to explore their relationships to serologic factors and cardiovascular risk factors in asymptomatic AAs with low Framingham risk. Between November 2010 and June 2012, 92 healthy AAs aged 21 years or older, from Baltimore, Maryland, were enrolled in an observational study. CMR examination was performed on a 3T scanner. Proton magnetic resonance spectroscopy was performed to noninvasively quantify myocardial triglyceride content. Native T1 values were obtained from modified Look-Locker inversion recovery (MOLLI) sequence. The median age was 37 (IQR: 27–44) years (41% men). The median native T1 time of the myocardium was 1228 ms (IQR: 1200–1263) with no gender difference. The median myocardial fat content was 0.6% (IQR: 0.7–4.6%). Native T1 time was influenced by age, sex and BMI. Among the factors investigated, myocardial fat and elevated CRP (CRP > 2.0 mg/dL) were independently associated with T1 relaxation time. Native T1 time was also independently associated with LV end-diastolic volume indexed to BSA. In conclusion, this study of asymptomatic AAs provides reference ranges for

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cardiovascular structure, function and tissue composition. Alterations in myocardial fat are associated with native T1 time, a CMR measure of interstitial fibrosis.

Keywords

Native cardiac magnetic resonance T1 mapping; African Americans; Myocardial triglyceride content; Myocardial structure and function

Introduction

T1 mapping is emerging as a useful tool for quantitative assessment of myocardial disease, and measurement of myocardial T1 relaxation times with noncontrast magnetic resonance T1 mapping, also known as “native” T1, has demonstrated potential to detect interstitial expansion due to myocardial edema and fibrosis [1-5]. “Native” T1 time of the myocardium has recently been evaluated as a marker of myocardial disease [6,7]. To use T1 mapping for characterizing myocardium, it is critical to obtain “normal or standard” T1 values. Despite the fact that T1 values have been estimated in those who were cardiovascularly asymptomatic without known cardiac disease at 3T [6,7], normal variation of T1 values in healthy African American population has not assessed. Also, factors that are associated with T1 relaxation time in this population should be investigated. The objectives of this investigation were (1) to explore the reference range for T1 relaxation time in healthy African American population; and (2) to identify factors that are independently associated with T1 relaxation time in healthy individuals.

Methods

Between November 2010 and June 2012, as part of a cohort study of heart disease in AAs conducted in Baltimore, 109 AA study participants from the city of Baltimore, Maryland, were enrolled in an observational study investigating factors that are associated with T1 relaxation time.

Inclusion criteria were age ≥ 21 years and African American. Exclusion criteria were (1) any evidence of ischemic heart disease as indicated by clinical history, previous hospitalization for myocardial infarction, angina pectoris, or evidence of valve disease or hypertension, (2) any symptoms believed to be related to cardiovascular disease, (3) a positive urine test for illegal drugs, (4) HIV infection, (5) pregnancy, and (6) history of MRI claustrophobia.

Interviews regarding sociodemographics, medical history, and behaviors were conducted; urine tests for illegal drugs were performed to exclude those with drug abuse, and HIV infection was determined by ELISA and confirmed by Western blot test. Clinical examinations, blood pressure (BP) measurement, cardiac magnetic resonance, and proton magnetic resonance spectroscopy (¹H MRS) were performed; and laboratory tests, including lipid profiles, leptin and high sensitivity C-reactive protein (hsCRP) levels were obtained.

The Johns Hopkins Medicine Institutional Review Board approved the study protocol and consent form, and all study participants provided written informed consent. All procedures

used in this study were in accordance with institutional guidelines. Although the overall investigation is a cohort study, the data presented herein are cross-sectional.

All studies were performed on a 3.0-T MR scanner (Trio Tim; Siemens, Erlangen, Germany) with a six-channel phased-array torso coil and combined with posterior coil elements resulting in 12 channels of data. Participants were instructed to hold their breath at end expiration during imaging and to breathe normally during spectroscopy. To measure left ventricular (LV) function, the heart was imaged in both long and short-axis orientations, using retrospectively gated steady state free precession cine images. One grid tagged short-axis slice was obtained at the middle LV.

Myocardial ^1H -MRS spectra were obtained with electrocardiogram gating during early systole, with navigator gating to enable free-breathing using a single voxel point-resolved spectroscopy sequence. The spectroscopic volume (6- to 8-mL voxel) was positioned within the interventricular septum. The navigator was placed across the liver-lung interface. For reliable measurement of the low-fat signals, one spectrum was recorded with water suppression (32 averages), and another spectrum (eight averages) was recorded without water suppression.

“Native” (noncontrast) T1 values were obtained by modified Look-Locker inversion recovery (MOLLI) sequence [8]. The MOLLI sequence acquired a set of 11 source four-chamber view images with one breath-hold (17 heartbeats), allowing the reconstruction of one parametric T1 map. The source images were all identical with the same voxel size, image position, and phase of the cardiac cycle, except for different effective inversion recovery times. This was achieved by performing three different ECG-gated inversion recovery-prepared experiments, each followed by several single-shot image acquisition (about 200 ms) at end-diastole cardiac phase.

The distribution of visceral adipose tissue (VAT) and subcutaneous adipose tissue (SAT) were quantified by a single-shot fast spin echo sequence (slice thickness=10mm). Three transverse images were acquired at the level of the fifth lumbar vertebrae during one breath hold.

Left ventricular volumes and mass were determined at end diastole from manual endocardial and epicardial contours traced on a stack of short-axis slices using commercially available software (QMass 7.2, Medis, The Netherlands). Spectral analysis was performed offline using Java based MR user interface (jMRUI version 3.0 software) [9]. Estimated lipid and water signals were fitted by the advanced method for accurate robust and efficient spectral fitting [10] with the assumption of Gaussian distribution. Resonance frequency of lipids at 0.9 and 1.3 ppm were summed to quantify myocardial triglycerides content and related to water in unsuppressed spectra [11]. Fat fraction was calculated as the ratio of total lipids in the water suppressed spectra to water content and reported as a percentage.

Tagged ventricular magnetic resonance images were analyzed by HARP MRI (Diagnosoft, Inc., Palo Alto, California) [12]. The average peak midwall circumferential strain (Ecc, in percentage) in the systolic phase was determined in all myocardial segments at the basal, mid and apical levels as previously described [13]. The value of Ecc is normally negative

during the contraction of the ventricle as it corresponds to relative circumferential shortening. Less negative Ecc (or positive Ecc) indicates diminished regional LV shortening. Tagged images were analyzed by a single reader who was blinded to participants' demographics and steatosis results.

T1 images were processed off-line using MASS research software (Mass, Leiden University Medical Center, Leiden, The Netherlands). Typical pixel-by-pixel fit was performed to a three-parameter model using Levenberg-Marquardt algorithm. Myocardial T1 value was drawn manually from interventricular septum; the same area that the MRS voxel was positioned. To avoid partial volume effects, voxels at the edge of the myocardium, as well as interatrial septum fat and blood pool were excluded from the contour. The blood T1 value was measured from the LV cavity. Visceral and subcutaneous adipose tissue was identified by selecting the region of interest and thresholding the pixel signal intensity using QMASS software. Adipose tissue volumes were quantified by converting the number of pixels to square centimeters and multiplied by the thickness of slices. The total volume of VAT and SAT was calculated by totaling the volumes of the individual slices.

Statistical analysis was performed with SAS (SAS 9.3, SAS Institute, Cary, NC). All continuous parameters were summarized by medians and interquartile ranges (IQRs), and all categorical parameters were summarized as proportions. To compare between-group differences in demographic and clinical characteristics, lipid profiles, and other factors, the non-parametric Wilcoxon 2-sample test was used for continuous variables and the Fisher's exact test was employed for categorical variables.

Since the ordinary least-squares regression model minimizes the sum of all the residuals and thus is sensitive to outliers, a robust regression model with the least trimmed squares (LTS) estimation method was used to provide robust (stable) results in the presence of outliers [14]. Both the Framingham risk score and the newly developed 2013 cardiovascular risk were calculated to estimate the CAD risk [15,16]. Univariate robust regression models were first fitted to evaluate the crude association between T1 and each of the factors—including age, sex, total serum cholesterol, HDL-cholesterol, LDL-cholesterol, serum triglycerides, high-sensitivity C-reactive protein (hsCRP), leptin, cigarette smoking, alcohol use, glucose level, systolic BP, diastolic BP, body mass index (BMI), Framingham risk score, and myocardial triglyceride content individually. Those factors that were significant at the $P < 0.30$ level in the univariate models were put into the multivariate robust regression models to identify the ones independently associated with the presence of cardiac steatosis. Those variables that ceased to make significant contributions to the models based on these criteria were deleted in a stagewise manner and a new model was refitted. This process of eliminating, refitting, and verifying continued until all of the variables included were statistically significant, yielding a final model. To examine whether relaxation time influence LV- function, a univariate robust regression model was first fitted for each LV function parameter and then multivariate robust regression analysis was performed for each parameter, adjusting for age, sex, BMI, and Framingham risk score. Since T1 relaxation time is influenced by heart rate, heart rate was treated as a potential confounding factor in the regression models. The p-values reported are two-sided. A p-value < 0.05 indicated statistical significance.

Results

The general and clinical characteristics of the study participants are presented in Table 1. Among these 92, 31 (33.7%) were normal weight ($18.5 \leq \text{BMI} < 25$), 23 (25.0%) were overweight ($25 \leq \text{BMI} < 30$), and 38 (41.3%) were obese ($\text{BMI} \geq 30$). The median myocardial triglyceride content was 0.6 (IQR: 0.3-1.0). The distribution of T1 relaxation time was moderately skewed (skewness=0.79) (Figure 1).

Compared with males, females had significantly higher C-reactive protein, BMI, leptin, subcutaneous fat, visceral fat, hepatic fat, and myocardial fat, and significantly lower LV end-diastolic volume, LV end-systolic volume; LV stroke volume, LV ejection fraction, LV end-diastolic mass, LV mass to volume ratio, LV end-diastolic volume indexed to body surface area, LV end-systolic volume indexed to body surface area, LV stroke volume indexed to body surface area, and LV end-diastolic mass indexed to body surface area.

The reference range for myocardial T1 relaxation time was between 1,132 and 1,332 ms based on the mean $\pm$ standard deviation of $1,232 \pm 51$ ms. The 90th and 95th percentiles of T1 relaxation time were 1,294 and 1,309 ms, respectively.

According to robust regression analyses, age and BMI had no impact on T1 relaxation time (Figures 2 and 3). The difference between males and females in T1 relaxation time was not statistically significantly different (the medians with IQRs of T1 are presented in Table 1, the means and standard deviations of T1 for males and females are $1,224 \pm 49$ ms and $1,239 \pm 51$ ms, respectively, p-value for the t-test: 0.17).

As shown in Figure 4, T1 relaxation time was significantly associated with the heart rate. By univariate robust regression analyses, factors associated with T1 relaxation time at the 0.30 level included age, male sex, leptin, hsCRP, $\text{hsCRP} > 2 \text{ mg/dL}$, BMI, total cholesterol, high-density lipoprotein cholesterol, subcutaneous fat, visceral fat, hepatic fat, and myocardial triglyceride content. The final robust regression model indicated that only $\text{hsCRP} > 2 \text{ mg/dL}$ and myocardial triglyceride content were independently associated with the T1 relaxation time (Table 2, Figure 5).

Multivariate robust regression models, in which heart rate, age, male sex, ACC/AHA new risk score, myocardial triglyceride content, and $\text{hsCRP} > 2 \text{ mg/dL}$ were adjusted for, indicated that T1 relaxation time was independently associated with LV end-diastolic volume indexed to BSA (Table 3).

Of 92 participants, data for regional myocardial circumferential strain for each left ventricular wall segment (anterior, anteroseptal, inferior, inferoseptal, inferolateral, and anterolateral) were available from 82 participants. Multivariate robust regression analysis suggested that T1 relaxation time was not independently associated with regional strain (Table 4).

Discussion

This study estimated the reference range for native T1 relaxation time in healthy AAs at 3T. Although the normal variation of MRI T1 relaxation was estimated in healthy volunteers at 1.5T [17], it was only estimated at 3T in two studies: one with normal range of $1,070 \pm 55$ ms (N=30) in UK [6], and another with normal range of 967 ± 34 ms (N=52) in Italy [7]. The normal range of T1 values for AAs derived from this study was $1,232 \pm 51$ ms, which was significantly higher than those reported from these two studies. The impact of age, sex, and BMI on T1 relaxation time has not been reported. In this study, we demonstrated that age (median 37 with IQR: 27-44 years), sex, and BMI (median 28 kg/m^2 with IQR: $23\text{-}34 \text{ kg/m}^2$) had no impact on noncontrast T1 relaxation time. This study shows that the 95th percentile of T1 relaxation time was 1,309 ms. Thus, we may use 1,300 ms as an optimal cutpoint to define normal range in healthy AA population.

The main findings of our study are (1) Native T1 relaxation time was independently associated with myocardial triglyceride content in cardiovascularly asymptomatic AAs recruited from the community; and (2) native T1 relaxation time was independently associated with *LV end-diastolic volume indexed to BSA, a marker of LV remodeling*. This is the first study to examine factors that are independently associated with T1 relaxation time and to explore the relationships between T1 relaxations time and LV function in healthy adults.

Tissue characterization using native T₁ mapping has drawn the attention of researchers and clinicians because this technique does not require the administration of contrast agents and is accessible in the context of renal impairment or contrast allergies. In the study by Dass et al, native T1 values were significantly elevated in patients with dilated or hypertrophic cardiomyopathy [3]. Using similar techniques, Puntmann et al reported that native T1 values provided greater distinction between healthy and diseased myocardium than did post contrast T1 [7].

This study reveals that among factors investigated, only myocardial triglyceride content and elevated CRP (CRP >2 mg/dL) were independently associated with T1 values. Overaccumulation of myocardial triglyceride (cardiac steatosis) may lead to cardiac dysfunction and cardiomyopathy [18-20]. Focal myocardial fat is frequently seen at cardiac computed tomography and MRI examinations in the normal and in diseased hearts with healed myocardial infarction [21]. Most adult hearts develop varying amounts of physiologic fat with aging or pathological fat as part of disease processes [22]. Increased precontrast myocardial T1 values have been observed in the hyperenhanced areas of acute and chronic infarction due to the expansion of extracellular volume [23]. Furthermore, increased fat content in areas of chronic myocardial infarction has also been found using MRI [24], computed tomography [25], and histological examinations [26]. Theoretically, myocardial fibrosis expands the extracellular space and increases the native T1 values, while the fat deposition should, on the contrary, reduce the T1 values of the area. Little is known about the physiology of the fibro-fatty replacement as well as the amount of fat in chronic scar. Based on reports of elevated T1 with chronic scar, it is reasonable to assume that the fibrosis outweighs the fatty replacement in this T1 competing process [21]. The exact mechanism by

which T1 relaxation time is related to myocardial triglyceride content remains to be understood.

This study also indicates that T1 relaxation time was independently associated with elevated CRP. It was observed that CRP levels at the acute phase of myocardial infarction revealed inflammation not only of the infarcted area but even more of the surrounding pericardial tissue [27]. An increase in native T1 values may be due to myocardial inflammation or diffuse myocardial fibrosis [28]. However, the relationship between native T1 and elevated CRP has not been reported.

This study has several limitations. First, because all of the study participants were AAs, the results may not be generalized to other race/ethnic groups without caution. Second, the data presented here are cross-sectional only and causality cannot be inferred. Third, due to the nature of a cross-sectional design, some hidden confounding factors, such as socioeconomic status, were not adjusted for. Longitudinal studies are certainly warranted to further investigate this association.

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Figure 1.

Distribution of T1 Relaxation Time.

The histogram of the T1 relaxation time, suggesting the distribution of T1 relaxation time was moderately skewed (skewness=0.79).

Figure 2.

The relationship between age and T1 relaxation time.

Univariate robust regression with the LTS estimator was run to investigate the relationship between age and T1 relaxation time. Robust regression $R^2 = 0.06$, $p = 0.13$, not significant.

Figure 3.

The relationship between BMI and T1 relaxation time.

Univariate robust regression with the LTS estimator was run to investigate the relationship between BMI and T1 relaxation time. Robust regression $R^2 = 0.002$, $p=0.23$, not significant.

Figure 4.

The relationship between heart rate and T1 relaxation time.

Univariate robust regression with the LTS estimator was run to investigate the relationship between heart rate and T1 relaxation time. Robust regression $R^2 = 0.15$, $p = 0.008$.

Figure 5.

The relationship between myocardial triglyceride content and T1 relaxation time.

Univariate robust regression with the LTS estimator was run to investigate the relationship between myocardial triglyceride content and T1 relaxation time. Robust regression $R^2 = 0.15$, $p < 0.0001$.

Table 1
Demographic and Clinical Characteristics of Study Participants*

Characteristic	Total (N = 92)	Male (N=38)	Female (N=54)	P-value
Age (years)	37 (27-44)	34 (26-42)	37 (28-47)	0.52
Family history of CAD	28%	16%	37%	0.03
Cigarette smoking	62%	82%	48%	0.001
Years of cigarette smoking	5 (0-15)	12 (2-20)	0 (0-10)	0.001
Alcohol use	50%	66%	39%	0.01
Years of alcohol use	0 (0-11)	5 (0-15)	0 (0-5)	0.016
Hematocrit (%)	37.1 (34.6-41.2)	41.5 (40.0-41.8)	35.1 (33.4-36.5)	0.0009
hsCRP 2 mg/dL	38%	21%	50%	0.005
hsCRP (mg/dL)	1.2 (0.4-3.5)	0.6 (0.2-1.5)	2.2 (0.6-4.8)	0.0004
Systolic BP (mm Hg)	114 (106-124)	117 (107-126)	112 (105-120)	0.19
Diastolic BP (mm Hg)	65 (59-74)	64 (60-73)	67 (58-75)	0.96
Glucose (mg/dL)	81 (77-89)	80 (77-89)	82 (78-89)	0.56
BMI (kg/m ²)	28 (23-34)	25 (21-30)	31 (26-38)	0.0005
Leptin (ng/mL)	10.8 (3.9-37.0)	3.7 (2.6-5.0)	35.8 (12.7-47.8)	<0.0001
Total cholesterol (mg/dL)	168 (148-191)	161 (147-186)	172 (149-198)	0.39
LDL-C (mg/dL)	94 (77-114)	87 (72-102)	100 (79-119)	0.09
HDL-C (mg/dL)	58 (49-64)	54 (45-64)	59 (51-64)	0.28
TG (mg/dL)	70 (56-102)	70 (56-115)	70 (56-98)	0.63
Subcutaneous fat (ml)	796 (476-1295)	441 (253-733)	1105 (730-1420)	<0.0001
Visceral fat (ml)	402 (294-582)	348 (267-458)	473 (337-650)	0.001
Hepatic TG content (%)	1.3 (0.7-4.6)	1.0 (0.4-1.5)	2.1 (0.9-5.4)	0.008
Myocardial TG content (%)	0.6 (0.3-1.0)	0.4 (0.3-0.8)	0.7 (0.4-1.1)	0.024
Framingham risk score	2 (1-3)	3 (2-4)	1 (1-2)	<0.0001
Framingham score <10.0	100%	100%	100%	-
ACC/AHA new risk score	1.2 (1.0-3.3)%	2.5 (1.3-4.8)%	0.2 (0.02-1.53)%	<0.0001
ACC/AHA high risk	5.4%	10.5	1.9	0.07
LV EDV (ml)	153 (134-174)	173 (157-194)	143 (122-160)	<0.0001
LV ESV (ml)	70 (57-82)	79 (72-88)	62 (51-71)	<0.0001
LV SV (ml)	83 (74-99)	92 (81-110)	80 (71-90)	0.0005
CO (L)	5.8 (5.0-6.8)	5.8 (5.1-6.8)	5.7 (4.7-6.7)	0.09
LV ejection fraction (%)	55 (52-59)	54 (51-58)	56 (52-62)	0.03
LV EDM (g)	118 (91-134)	134 (122-148)	102 (84-118)	<0.0001
LV mass to volume ratio (g/ml)	0.75 (0.67-0.81)	0.77 (0.71-0.84)	0.70 (0.64-0.77)	0.0008
LV EDV indexed to BSA (ml/m ²)	82 (74-91)	90 (84-97)	76 (69-81)	<0.0001
LV ESV indexed to BSA (ml/m ²)	38 (31-41)	41 (39-44)	33 (28-39)	<0.0001

Characteristic	Total	Male	Female	P-value
	(N = 92)	(N=38)	(N=54)	
LV SV indexed to BSA (ml/m ²)	44 (40-51)	48 (44-54)	42 (39-47)	0.0002
CO indexed to BSA (L/m ²)	3.1 (2.7-3.4)	3.2 (2.7-3.6)	3.1 (2.6-3.4)	0.23
LV EDM index to BSA (g/m ²)	61.5 (52.8-68.4)	68.8 (65.2-75.6)	54.2 (47.4-60.6)	<0.0001
T1 times (ms)	1228 (1200-1263)	1219 (1187-1252)	1228 (1201-1274)	0.18
Heart rate (beats/minute)	64 (58-72)	60 (58-69)	66 (58-74)	0.12

Abbreviations: ACC/AHA new risk score, the new calculator for assessing the 10-year risk of atherosclerotic cardiovascular disease assessed by the 2013 ACC/AHA guidelines; ACC/AHA high risk, the 2013 ACC/AHA risk score 7.5%; BMI, body mass index (kg/m²); BP, blood pressure; BSA, body surface area; CAD, coronary artery disease; CO, Cardiac output; Framingham score, Framingham risk score; glucose, fasting glucose; HDL-C, high density lipoprotein cholesterol; hsCRP, high-sensitivity C-reactive protein; LDL-C, low density lipoprotein cholesterol; LV, left ventricular; LV EDM, LV end-diastolic mass; LV EDV, LV end-diastolic volume; LV ESV, LV end-systolic volume; LV SV, LV stroke volume; T1 times, Non contrast T1 relaxation times; TG, triglycerides.

* Median (interquartile range) for continuous variables, proportion (%) for categorical variables.

Table 2
Demographic, Laboratory, and Clinical Factors in Relation to Noncontrast T1 Relaxation Time, Robust Regression Analysis with LTS Estimation

Variable	Noncontrast T1 relaxation times (ms)	
	Bi-variate regression estimate (SE) *	p-value
Age (year)	0.485 (0.416)	0.24
Male sex	-18.85 (9.020)	0.037
Cigarette smoking	-7.216 (9.280)	0.44
Years of cigarette smoking	0.208 (0.396)	0.60
Alcohol use	9.039 (8.963)	0.31
Years of alcohol use	0.312 (0.400)	0.44
Hematocrit (%)	0.578 (2.640)	0.83
Leptin (ng/mL)	0.244 (0.182)	0.18
hsCRP (mg/dL)	1.388 (0.925)	0.13
hsCRP>2 mg/dL	25.428 (8.955)	0.005
Systolic BP (mmHg)	-0.264 (0.371)	0.48
Diastolic BP (mm Hg)	0.270 (0.505)	0.59
Fasting glucose (mg/dL)	0.375 (0.548)	0.49
BMI (kg/m ²)	0.895 (0.616)	0.15
Total cholesterol (mg/dL)	-0.145 (0.134)	0.28
LDL-C (mg/dL)	-0.055 (0.153)	0.72
HDL-C (mg/dL)	-0.387 (0.285)	0.17
Triglycerides (mg/dL)	-0.036 (0.118)	0.76
Framingham score	1.575 (2.971)	0.60
ACC/AHA new risk score	-12.405 (148.762)	0.93
ACC/AHA high risk	18.424 (19.362)	0.34
Hepatic triglyceride (%)	1.110 (0.838)	0.19
Subcutaneous fat (mL)	0.011 (0.008)	0.14
Visceral fat (mL)	0.025 (0.021)	0.25
Myocardial triglyceride content (%)	22.538 (5.149)	<0.0001

* In bi-variate analyses and multivariate analyses, heart rate was adjusted for. Multivariate analysis showed that only hsCRP (regression estimate: 21.182, SE: 8.263, p=0.01) and myocardial triglyceride content (regression estimate 20.949, SE: 5.026, P<0.0001) were independently associated with native T1 relaxation time.

Abbreviations: BP, blood pressure; BMI, body mass index (kg/m²); Framingham score, Framingham risk score; HDL-C, high density lipoprotein cholesterol; LDL-C, low density lipoprotein cholesterol; hsCRP, high-sensitivity C-reactive protein; SE, standard error.

Table 3
Association between LV functions and Noncontrast T1 Relaxation Times, Robust Regression Analysis with LTS Estimation*

Outcome Variables	Noncontrast T1 relaxation time			
	Univariate regression estimate (SE)*	P-value	Multivariate regression estimate (SE)*	p-value
LV end-diastolic volume	0.012 (0.066)	0.85	0.070 (0.063)	0.26
LV end-systolic volume	-0.023 (0.037)	0.54	0.044 (0.034)	0.20
LV stroke volume	0.035 (0.040)	0.38	0.019 (0.041)	0.64
Cardiac output	-0.001 (0.003)	0.84	0.001 (0.003)	0.77
LV ejection fraction	0.011 (0.012)	0.38	-0.013 (0.013)	0.32
LV end-diastolic mass	-0.030 (0.060)	0.61	0.023 (0.051)	0.66
Concentricity index	-0.000 (0.000)	0.38	-0.000 (0.000)	0.45
LV end-diastolic volume indexed to BSA	0.007 (0.027)	0.80	0.050 (0.023)	0.027
LV end-systolic volume indexed to BSA	-0.011 (0.018)	0.53	0.025 (0.015)	0.09
LV stroke volume indexed to BSA	0.016 (0.016)	0.32	0.012 (0.016)	0.46
Cardiac output indexed to BSA	-0.000 (0.001)	0.89	0.001 (0.001)	0.33
LV end-diastolic mass indexed to BSA	-0.013 (0.024)	0.61	0.016 (0.019)	0.42

* In multivariate analyses, heart rate, age, male sex, ACC/AHA new risk score, myocardial triglyceride content, and CRP>2 were adjusted for.

Abbreviations: LV, left ventricular; BSA, body surface area.

Table 4
Association between mid-wall peak circumferential strain (Ecc) of left ventricular segments and T1 relaxation time, generalized estimating equation (GEE) analysis (N=86)

Variable	Outcome variable: Mid-wall peak circumferential strain (Ecc)			
	Univariate model		Multivariate model*	
	regression estimate (SE)	p -value	regression estimate (SE)	p-value
Age (year)	0.001 (0.030)	0.97	0.008 (0.038)	0.98
Male gender	2.371 (0.552)	<0.0001	2.252 (0.666)	0.0007
Heart rate	0.0493 (0.030)	0.10	0.068 (0.025)	0.007
ACC/AHA new risk	19.745 (10.195)	0.05	4.948 (14.036)	0.72
CRP>2.0 (mg/dL)	-0.610 (0.633)	0.34	-0.466 (0.610)	0.45
Myocardial triglyceride content	-0.245 (0.424)	0.56	-0.105 (0.471)	0.82
T1relaxation time*	-0.014 (0.007)	0.054	-0.003 (0.007)	0.67

Abbreviations: ACC, American College of Cardiology; AHA, American Heart Association; ACC/AHA new risk, new ACC/AHA risk score; CRP, C-reactive Protein