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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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, from Baltimore, MD, 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 sequence. The median age was 37 (interquartile range IQR 27 to 44) years (41% men). The median native T1 time of the myocardium was 1,228 ms (IQR 1,200 to 1,263) with no gender difference. The median myocardial fat content was 0.6% (IQR 0.7% to 4.6%). Native T1 time was not influenced by age, sex, and body mass index. Among the factors investigated, myocardial fat and elevated C-reactive protein (>2.0 mg/dL) were independently associated with T1 relaxation time. Native T1 time was also independently associated with left ventricular end-diastolic volume indexed to body surface area. In conclusion, this study of asymptomatic AAs provides reference ranges for cardiovascular structure, function, and tissue composition. Alterations in myocardial fat are associated with native T1 time, a CMR measure of interstitial fibrosis. **© 2014 Elsevier Inc. All rights reserved.** **(Am J Cardiol 2014;114:789−795)**

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 because of 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. Although 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 (AA) 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 AA population and (2) to identify factors that are independently associated with T1 relaxation time in healthy subjects.

Methods

Between November 2010 and June 2012, as part of a cohort study of heart disease in AAs conducted in Baltimore, 92 AA study participants from the city of Baltimore, MD, were enrolled in an observational study investigating factors that are associated with T1 relaxation time.

Inclusion criteria were age ≥21 years and AA. 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) hypertension and/or diabetes; (4) a positive urine test for illegal drugs; (5) HIV infection; (6) pregnancy; and (7) history of magnetic resonance imaging (MRI) claustrophobia.

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See page 794 for disclosure information.

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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
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

ACC/AHA high risk, the 2013 ACC/AHA risk score ≥7.5%; 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; BMI, body mass index (kg/m^2); 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.

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 enzyme-linked immunosorbent assay and confirmed by Western blot test. Clinical examinations, blood pressure (BP) measurement, cardiac magnetic resonance (CMR), and proton magnetic resonance spectroscopy 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 here are cross-sectional.

All studies were performed on a 3T MR scanner (Trio Tim; Siemens, Erlangen, Germany) with a 6-channel phased-array torso coil and combined with posterior coil elements resulting in 12 channels of data. Participants were

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