Ideal Cardiovascular Health, Cardiovascular Remodeling, and Heart Failure in Blacks

The Jackson Heart Study

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Background—The lifetime risk of heart failure (HF) is higher in the black population than in other racial groups in the United States.

Methods and Results—We measured the Life's Simple 7 ideal cardiovascular health metrics in 4195 blacks in the JHS (Jackson Heart Study; 2000–2004). We evaluated the association of Simple 7 metrics with incident HF and left ventricular structure and function by cardiac magnetic resonance (n=1188). Mean age at baseline was 54.4 years (65% women). Relative to 0 to 2 Simple 7 factors, blacks with 3 factors had 47% lower incident HF risk (hazard ratio [HR], 0.53; 95% confidence interval [CI], 0.39–0.73; P<0.0001); and those with ≥4 factors had 61% lower HF risk (HR, 0.39; 95% CI, 0.24–0.64; P=0.0002). Higher blood pressure (HR, 2.32; 95% CI, 1.28–4.20; P=0.005), physical inactivity (HR, 1.65; 95% CI, 1.07–2.55; P=0.02), smoking (HR, 2.04; 95% CI, 1.43–2.91; P<0.0001), and impaired glucose control (HR, 1.76; 95% CI, 1.34–2.29; P<0.0001) were associated with incident HF. The age-/sex-adjusted population attributable risk for these Simple 7 metrics combined was 37.1%. Achievement of ideal blood pressure, ideal body mass index, ideal glucose control, and nonsmoking was associated with less likelihood of adverse cardiac remodeling by cardiac magnetic resonance.

Conclusions—Cardiovascular risk factors in midlife (specifically elevated blood pressure, physical inactivity, smoking, and poor glucose control) are associated with incident HF in blacks and represent targets for intensified HF prevention. **(*Circ Heart Fail*. 2017;10:e003682. DOI: 10.1161/CIRCHEARTFAILURE.116.003682.)**

Key Words: blood pressure ■ body mass index ■ heart failure ■ hypertension ■ risk factors

Blacks have a disproportionately higher risk[1] and earlier onset[2] of heart failure (HF) and cardiovascular mortality, relative to the overall American population. A prime contributor to this racial disparity is a higher prevalence and earlier onset of cardiometabolic disease, including obesity, hypertension, diabetes mellitus, and physical inactivity.[3] Professional guidelines reinforcing ideal cardiovascular health target improvements in cardiometabolic health to prevent disease ("Life's Simple 7"). However, recent data in over 5000 blacks from the JHS (Jackson Heart Study) indicate a low prevalence of ideal cardiovascular health characteristics (specifically diet, body mass index [BMI], and physical activity) with only modest improvements over time.[4] In largely non-black populations, achievement of fewer Life's Simple 7 components is associated with more adverse left ventricular (LV) remodeling[5] and greater cardiovascular disease (CVD) and non-CVD mortality.[6,7] Nevertheless, most prior work examining Simple 7 metrics has not focused on blacks to define axes of cardiometabolic health that most impact cardiovascular disease progression in this population.

See Clinical Perspective

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To understand the impact of cardiometabolic health on HF in blacks, we studied the relationship of Life's Simple 7 metrics with incident HF and myocardial structure and function (by cardiac magnetic resonance [CMR]) in the JHS. Given previous results in JHS documenting a low prevalence of physical activity, greater BMI, and poor diet,[4] we hypothesized that the achievement of fewer ideal components of Life's Simple 7 would be associated with greater risk of incident HF and more adverse cardiovascular remodeling by CMR.

Methods

Study Population

The JHS is a population-based prospective study of blacks 21 years or older from the Jackson, Mississippi, tricounty area (Hinds, Madison, and Rankin). The study was designed to identify causes of CVD among blacks.[8,9] Study subjects were examined at a baseline clinic visit (2000–2004) and during 2 additional visits: visit 2 (2005–2008) and visit 3 (2009–2013). Follow-up telephone interviews were performed annually. The study was Institutional Review Board approved. All participants provided written informed consent.

There were 5306 study participants who attended the initial study visit. Our first aim was to examine the association between achievement of Life's Simple 7 metrics and incident HF. For this aim, we sequentially excluded participants with (1) incomplete data on Life's Simple 7 components (n=824 participants) and (2) prevalent coronary heart disease (CHD: self-reported history of myocardial infarction [MI] or MI by 12-lead ECG using the Minnesota Code Classification system[8]) at the baseline clinic visit (n=287 of remaining participants), leaving 4195 participants in our final analytic sample for this first aim.

Our second aim was to assess the relationship of Simple 7 attainment with cardiovascular remodeling by CMR. Of the 4195 individuals included in our first aim, 3112 attended visit 3, of whom 1188 participants had a CMR with complete circumferential systolic strain data. Of note, participants were excluded from CMR imaging for pregnancy, metallic hazards (eg, implanted electric devices, pacemaker, and orbital metal), inability to fit in the scanner, claustrophobia, or refusal to undergo CMR. In addition, we did not include individuals who had CMR at visit 2 because we wanted to ensure that the CMR outcome was measured at the same (and most contemporary) time point across all individuals.

Covariate and Exposure Definition

We constructed the Simple 7 score for each JHS participant by assigning a score of 1 (ideal status) or 0 (nonideal) for each of the Simple 7 metrics (Table I in the Data Supplement) as previously published.[4,10] For consistency with the previous reports,[4] we used metrics already adjudicated within the JHS (as opposed to ideal, intermediate, and poor categories as defined in other work[4]). Smoking was assessed at baseline by questionnaire (current, never, or former smoker). Former smokers were further subdivided into those who quit smoking <12 months or >12 months before the interview. Family income was adjusted for family size and calendar-year–specific poverty level and stratified into 2 groups: (1) a composite poor and lower-middle–income group and (2) an upper-middle and affluent group. Upper-middle income was defined as at least 1.6× poverty level.[11] Weight was measured (to the nearest kg) using a balance scale. Height was measured in a standing position (to the nearest cm) with a vertical ruler. Dietary intake was assessed with a regionally specific food frequency questionnaire designed for the study population.[12] Physical activity was obtained from a modified Kaiser physical activity survey, and the time per week engaged in moderate or vigorous sports and exercise activity was used to derive the physical activity ideal health score.[4] Sitting blood pressure was calculated as the average of 2 resting blood pressure recordings. Prevalent diabetes mellitus was defined according to the American Diabetes Association criteria as fasting glucose ≥126 mg/dL, hemoglobin A1c ≥6.5%, or use of medications for diabetes mellitus.[13] Hypertension was defined as a systolic blood pressure ≥140 mm Hg, a diastolic blood pressure ≥90 mm Hg, or use of antihypertensive medications.

Venous blood samples were drawn from each subject at JHS baseline examination as previously described.[14] Lipids, fasting plasma glucose, hemoglobin A1c, insulin, and high-sensitivity C-reactive protein were measured using standard laboratory techniques. The homeostasis model assessment of insulin resistance was calculated in molar units as (fasting blood glucose [mg/dL]/18.1×insulin [μU/mL]/22.5).[15] Estimated glomerular filtration rate (in mL min^{-1} 1.73 m^{-2}) was calculated based on the Chronic Kidney Disease Epidemiology Collaboration formula.[16] Assays for aldosterone,[17] renin,[18] leptin,[17] and adiponectin[19] have been previously described.

Primary Outcome Ascertainment

The construction of the cohort for outcomes ascertainment is shown in Figure 1. HF adjudication has been previously described.[20] In brief, incident HF hospitalizations were identified through annual follow-up telephone interviews and compared with annual hospital discharge lists and death certificates by trained and certified HF abstractors. The annual hospital discharge lists were reviewed for (1) International Classification of Diseases, Ninth and 10th Revision HF diagnosis codes (428 and I50, respectively), and (2) radiographic findings consistent with HF, increased jugular venous pressure, dilated ventricle, LV ejection fraction <0.40 by echocardiogram or nuclear scan, or (3) autopsy finding of pulmonary edema or HF. Death certificates were reviewed for causes of death that were suggestive of HF. Trained medical personnel performed final event adjudication.

There was a gap in the period between the baseline visit (September 26, 2000) and the start of formal HF adjudication (in January 1, 2005). Events were formally adjudicated by the JHS from January 1, 2005 to December 31, 2012. We have therefore provided analysis for (1) all incident HF from the time of enrollment, either self-reported or by formal adjudication (after January 1, 2005) and (2) formally adjudicated HF only (from January 1, 2005 to end point or censor).

CMR Imaging

CMR scans included in this study were performed at visit 3 using a 1.5-T system (Siemens Espree; Siemens, Erlangen, Germany; 70 cm bore, advanced cardiac package, TIM Matrix surface coil). The CMR protocol was based and developed in collaboration with the Multi-Ethnic Study of Atherosclerosis CMR protocol to enhance comparability.[21] Assessment of ventricular function and mass was performed using electrocardiographically gated fast gradient echo cine images with steady-state free precession (TrueFISP or TRUFI, Siemens sequence variant Tfi2d1_18) with parameters: repetition time (TR) 45.5 ms, echo time (TE) 1.1 ms, flip angle 78° to 82°, 8-mm slice thickness, matrix 109×192, and field of view 400 mm. LV volume and mass were determined by short-axis volumetric coverage, and LV mass was indexed to height in $m^{2.7}$.[22] Papillary muscles were included in the LV volumes and excluded from LV mass.[23] Cardiac Image Modeler software (University of Auckland, New Zealand) was used to analyze CMR function and morphology data.[24] CMR tagged images were acquired at the base, mid, and apex of the LV using a cine radiofrequency grid–tagging sequence (field of view 400 mm, slice thickness 8 mm, 192×256 matrix, TR 60 ms, TE 4 s, and FA of 12° [Siemens sequence: Tl2d1r5]). Tagging analysis was performed using HARP (Diagnosoft, Morrisville, NC). Global strain was calculated as the average peak circumferential systolic Eulerian strain (Ecc) of the basal, midcavity, and apical segments of the LV. Aortic pulse wave velocity (PWV) was calculated from phase-contrast images acquired in the ascending and descending thoracic aorta, as previously described.[25] intraclass correlation coefficient for interobserver reliability based on 96 scans that were analyzed as new scans after relabeling (to blind analysts) was 0.95, 0.88, 0.85, and 0.96 for LV end-diastolic volume, LV end-systolic volume, LV stroke volume, and LV mass, respectively. The intraclass correlation coefficient for HARP Ecc measures was 0.78 (and for PWV analyses was 0.82) in repeated, blinded analyses of 96 scans.

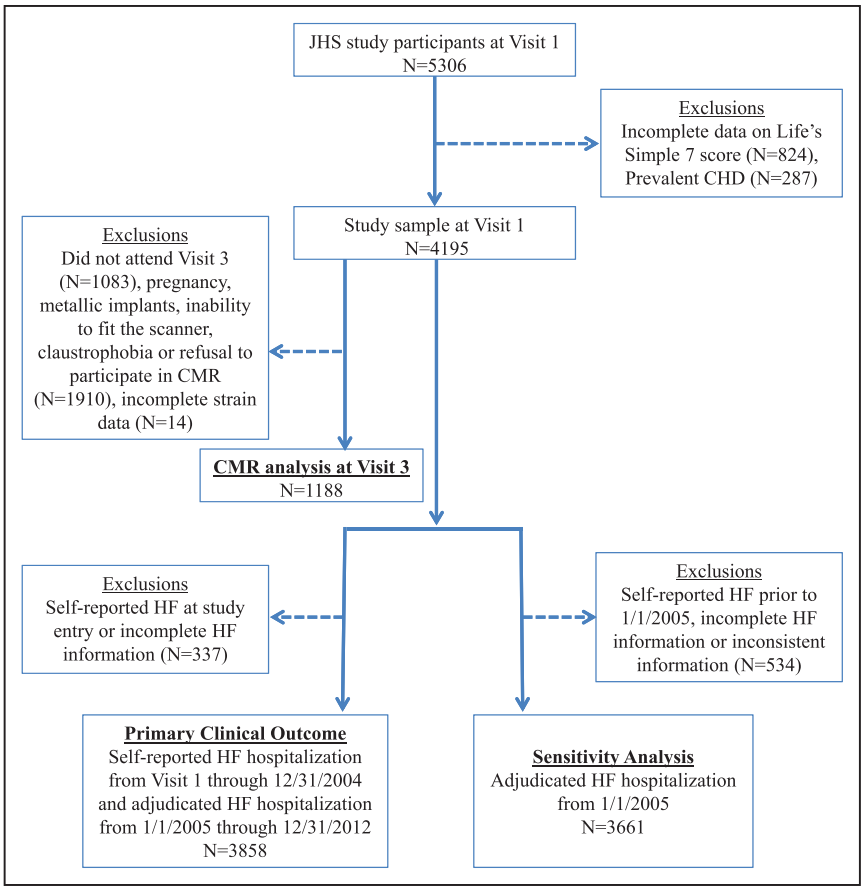

Figure 1. CONSORT diagram. Out of 4195 participants without prevalent coronary heart disease (CHD) with Life's Simple 7 metrics measured at baseline, 1188 were included in our cardiac magnetic resonance (CMR) analyses, and 3858 were followed for incident heart failure (HF). Incomplete HF information refers to missing self-reported HF hospitalization or self-reported physician diagnosis of HF. JHS indicates Jackson Heart Study.

Statistical Analysis

Baseline clinical, demographic, biochemical, and imaging characteristics were stratified by categories of Life's Simple 7 score (0–2, 3, and 4–6). We chose these categories to facilitate an approximately equal number of participants in each bin (0–2: n=1936; 3: n=1354; and 4–6: n=905). The Kruskal–Wallis test (non-normal continuous data) or χ^2 test (categorical) was used for comparisons.

We assessed the relationship between Life's Simple 7 and incident HF using Cox proportional hazards models and Kaplan–Meier survival analysis. For Kaplan–Meier analysis, we stratified Simple 7 score into categories (0–2, 3, and 4–6 ideal factors). To identify which components of Simple 7 were most closely associated with HF, we constructed univariate and multivariate Cox proportional hazards regression models, including each individual Simple 7 component, age and sex. To limit confounding by CHD, we performed additional analyses excluding fatal CHD and incident MI throughout the study period. Fatal CHD and MI were adjudicated by trained personnel on review of medical records.[20] The proportional hazards assumption was verified by the Kolmogorov-type supremum test.

Finally, we used a pooled logistic regression (splitting follow-up time into 5-year bins for each participant) to estimate an adjusted population attributable risk (PAR) for each of the 4 Simple 7 factors that were significantly associated with incident HF in multivariable Cox regression as specified.[26] The Simple 7 factors were left constant through the follow-up period (each 5-year bin). To calculate the adjusted (partial) PAR for each of the 4 Simple 7 metrics, we treated age, sex, and the 3 other Simple 7 variables as nonmodifiable. The SAS macro %PAR[26] was used for this analysis.

To measure the association of Life's Simple 7 with cardiac remodeling by CMR, we constructed separate univariate and age- and sex-adjusted linear models for cardiac structure/function by CMR (measured a median 8.1 years after baseline study visit) as a function of each Simple 7 component. CMR indices included LV mass index, LV mass:volume ratio (LV concentric remodeling index), aortic PWV, and global circumferential LV strain. We performed a Bonferroni correction for multiplicity (to account for multiple models).

We evaluated effect modification by age (median stratified), sex, and obesity status (defined as obese BMI ≥30 kg/m^2 versus nonobese BMI <30 kg/m^2) on the association between the Simple 7 score (modeled using categories of 0–2, 3, and 4–6 factors) and CMR parameters using a multiplicative interaction term. The least squares means in each category of age, sex, and obesity status for each cardiac parameter were graphed to facilitate visual interpretation. Of note, we performed log transformations of LV mass index, LV mass:volume ratio, and aortic PWV to approximate normality before regression; least squares means were exponentiated back after regression to provide clinically meaningful LV mass, concentricity, and aortic PWV values.

Statistical analyses were performed using SAS version 9.4 (SAS Institute, Cary, NC). A 2-tailed *P* value of 0.05 was considered significant.

Results

Baseline Characteristics

Demographic, clinical, biochemical, and imaging characteristics of our study population stratified by Simple 7 score are presented in Table 1. Mean age in the overall study population was 54.4±12.8 years, with 65% female. Median BMI was at least overweight across all Simple 7 score categories, with low prevalence of ideal nutrition and ideal physical activity, as previously reported.[4] Overall, JHS participants with lower Simple 7 scores were more often female and had lower education and income. JHS participants with lower Simple 7 scores had a more adverse cardiometabolic profile, with greater proatherogenic dyslipidemia, insulin resistance (homeostasis model assessment of insulin resistance), systemic inflammation

Table 1. Baseline Characteristics for All Jackson Heart Study Participants Meeting Inclusion Criteria, Stratified by Simple 7 Score Category

	Simple 7 Score of 0–2	Simple 7 Score of 3	Simple 7 Score of 4–6	
	n≤1936*	n≤1354*	n≤905*	*P* Value
Demographics				
Age, y	59.2 (50.1 to 66.3)	53.0 (43.9 to 63.7)	45.5 (38.8 to 54.8)	<0.0001
Female	1288 (66.5)	874 (64.6)	554 (61.2)	0.02
High-school graduate or GED and beyond	1087 (81.2)	891 (86.8)	654 (93.2)	<0.0001
Upper-middle income or greater	956 (57.7)	719 (63.1)	541 (69.1)	<0.0001
Ever smoker	725 (37.5)	359 (26.5)	164 (18.1)	<0.0001
Weight, kg	90.7 (80.0 to 104.0)	87.4 (75.0 to 102.0)	79.8 (67.5 to 92.0)	<0.0001
BMI, kg/m^2	31.9 (28.5 to 36.5)	30.1 (26.6 to 35.3)	27.3 (23.9 to 31.9)	<0.0001
Systolic BP, mm Hg	129.3 (122.0 to 140.3)	124.8 (116.5 to 134.8)	114.7 (107.3 to 122.0)	<0.0001
Estimated GFR, mL/min	92.9 (79.1 to 106.6)	96.7 (83.2 to 110.4)	103.2 (90.5 to 116.2)	<0.0001
Prevalent disease				
Hypertension	1369 (70.7)	691 (51.0)	181 (20.0)	<0.0001
Diabetes mellitus	605 (31.3)	108 (8.0)	25 (2.8)	<0.0001
Simple 7 component				
Nonsmoking	1545 (79.8)	1222 (90.3)	861 (95.1)	<0.0001
Ideal BMI	64 (3.3)	207 (15.3)	340 (37.6)	<0.0001
Ideal nutrition	3 (0.2)	11 (0.8)	16 (1.8)	<0.0001
Ideal physical activity	100 (5.2)	288 (21.3)	444 (49.1)	<0.0001
Ideal BP	53 (2.7)	253 (18.7)	585 (64.6)	<0.0001
Ideal fasting plasma glucose	1112 (57.4)	1275 (94.2)	896 (99.0)	<0.0001
Ideal cholesterol	358 (18.5)	806 (59.5)	760 (84.0)	<0.0001
CMR parameters (n≤1188)†				
LV mass index, g/m$^{2.7}$	32.0 (28.2 to 36.9)	31.1 (27.0 to 35.1)	29.0 (25.3 to 32.5)	<0.0001
LV concentric remodeling index, g/mL	1.1 (1.0 to 1.3)	1.1 (0.9 to 1.3)	0.9 (0.8 to 1.1)	<0.0001
Height-indexed LVEDV, mL/m	68.9 (59.1 to 82.6)	71.7 (59.4 to 85.0)	75.1 (62.7 to 86.2)	0.02
Stroke volume, mL	69.1 (56.9 to 86.2)	72.9 (61.8 to 88.1)	74.2 (63.1 to 88.6)	0.03
LV ejection fraction, %	61.8 (54.7 to 66.8)	61.4 (55.6 to 66.6)	59.8 (54.4 to 65.1)	0.16
Aortic PWV, m/s	6.2 (5.0 to 8.8)	5.9 (4.3 to 8.2)	5.3 (4.0 to 7.4)	<0.0001
Global circumferential strain, %	−15.4 (−16.9 to −13.6)	−15.8 (−17.6 to −14.3)	−16.4 (−17.9 to −14.7)	<0.0001
Biomarkers‡				
LDL, mg/dL	140.5 (115.0 to 162.0)	120.0 (99.0 to 141.0)	110.0 (92.0 to 128.0)	<0.0001
HDL, mg/dL	49.0 (41.0 to 60.0)	51.0 (41.0 to 60.0)	50.0 (42.0 to 59.0)	0.66
Triglycerides, mg/dL	103.0 (76.0 to 145.5)	84.0 (62.0 to 117.0)	69.0 (50.0 to 97.0)	<0.0001
hs-CRP, mg/dL	0.7 (0.7 to 0.8)	0.7 (0.6 to 0.8)	0.7 (0.6 to 0.7)	<0.0001
Hemoglobin A1c, %	5.9 (5.5 to 6.5)	5.5 (5.2 to 5.8)	5.4 (5.1 to 5.6)	<0.0001
HOMA-IR	3.5 (2.5 to 5.0)	3.0 (2.1 to 4.2)	2.5 (1.8 to 3.4)	<0.0001
Plasma renin activity, ng mL^{-1} h^{-1}	0.5 (0.2 to 1.4)	0.4 (0.2 to 0.9)	0.3 (0.2 to 0.8)	<0.0001
Aldosterone, ng/dL	4.7 (2.9 to 8.1)	4.2 (2.5 to 6.8)	3.7 (2.2 to 5.8)	<0.0001
Adiponectin, ng/mL	4041.5 (2557.7 to 6277.4)	4443.6 (2842.8 to 6895.2)	4434.0 (2771.4 to 7032.1)	<0.0001
Leptin, ng/mL	27.0 (13.5 to 43.0)	23.5 (9.7 to 39.4)	15.6 (6.2 to 30.1)	<0.0001

Values are median (25th–75th percentile) or n (%). BMI indicates body mass index; BP, blood pressure; CMR, cardiac magnetic resonance; GED, graduate equivalency diploma; GFR, glomerular filtration rate; HDL, high-density lipoprotein; HOMA-IR, homeostatic model assessment of insulin resistance; hs-CRP, high-sensitivity C-reactive protein; LDL, low-density lipoprotein; LV, left ventricle; LVEDV, left ventricular end-diastolic volume; and PWV, pulse wave velocity.

*Numbers are for Simple 7 score category out of a total 4195 participants in our sample population.

†The greatest number of missing participants with CMR was in aortic PWV with 93 of 1188 missing.

‡The greatest number of missing participants was in plasma renin activity with 2321 of 4195 missing.

(high-sensitivity C-reactive protein), a more dysfunctional adiposity phenotype (higher leptin and lower adiponectin), and greater renin–angiotensin–aldosterone system activation (all $P<0.0001$). Of note, individuals who had CMR imaging performed in JHS were in general younger, with a higher Simple 7 score (Table II in the Data Supplement).

Life's Simple 7 Is Associated With Incident HF

Over a median 9.9-year follow-up from study enrollment (25th–75th percentile 9.0–10.7 years), 239 incident HF events occurred in 3858 participants. After adjustment for age and sex, achievement of more components of ideal health was associated with a reduced hazard of incident HF (hazard ratio [HR], 0.53; 95% confidence interval [CI], 0.39–0.73; $P<0.0001$ for Simple 7 score of 3 and HR, 0.39; 95% CI, 0.24–0.64; $P=0.0002$ for Simple 7 score of ≥4 relative to a Simple 7 score 0–2; Table 2; Figure 2).These associations remained after incident MI or fatal CHD were excluded (Table 2). Finally, when only adjudicated HF hospitalizations beginning January 1, 2005 were included (n=3661 total subjects; Figure 1), we found a similar association between Life's Simple 7 and incident HF (Table III in the Data Supplement).

When evaluating all Simple 7 components simultaneously in an age- and sex-adjusted Cox model, elevated blood pressure (HR, 2.32; 95% CI, 1.28–4.20; $P=0.005$), physical inactivity (HR, 1.65; 95% CI, 1.07–2.55; $P=0.02$), smoking (HR, 2.04; 95% CI, 1.43–2.91; $P<0.0001$), and impaired glucose control (HR, 1.76; 95% CI, 1.34–2.29; $P<0.0001$) were associated with incident HF (Table 3). After adjustment for age and sex, the population attributable fraction for elevated blood pressure, physical inactivity, smoking, and impaired glucose control was 37.1% (95% CI, 20.0%–51.9%; Table 3, with the greatest PAR% for elevated blood pressure (16.0%; 95% CI, 5.4–26.3).

Simple 7 Components Are Associated With LV Function and Structure in Blacks

In 1188 JHS participants with CMR imaging over a median 8.1 years (25th–75th percentile 7.5–8.9 years) after study entry, ideal blood pressure, BMI, glucose control, and nonsmoking were consistently associated with cardiac phenotypes (Table 4). Ideal blood pressure was associated with lower LV mass and lower concentricity (both $P<0.0001$). Ideal BMI was associated with lower LV mass, lower concentricity, and strain (all $P<0.0001$). Nonsmoking was associated with lower LV concentric remodeling ($P<0.0001$), lower LV strain ($P=0.0002$), and lower aortic PWV ($P=0.0001$). Ideal glucose was associated with lower LV mass ($P=0.002$).

We further examined whether sex, age, or BMI at the time of CMR modified the relationship between categories of Simple 7 score and these parameters. Although males and obese individuals had greater hypertrophy, concentric remodeling, and decreased strain (relative to female and nonobese counterparts), we did not observe any evidence of effect modification (Figure 3).

Discussion

In a large, community-based population of blacks with comprehensive cardiometabolic phenotyping, we found that the achievement of fewer components of Life's Simple 7 at baseline was strongly associated with incident HF over ≈10 years of follow-up. Specifically, elevated blood pressure, physical inactivity, smoking, and impaired glucose control contributed to HF risk development, highlighting these factors as essential for HF prevention. In addition, nonideal cardiovascular health status was strongly associated with adverse cardiac remodeling in blacks ≤8 years later, including LV hypertrophy, concentric remodeling, and LV dysfunction—all subclinical phenotypes contributing to HF development. Collectively, in a large, contemporary, community-based black cohort, cardiovascular health practices in midlife in black influence HF progression across the spectrum of subclinical CVD to manifest HF. Improved cardiovascular health behaviors early in life may impact long-term HF development in blacks.

Since the definition of "ideal cardiovascular health" as 7 separate targets of prevention by the American Heart Association,[10] a wealth of data attesting to achievement of ideal Life's Simple 7 metrics and its consequences on cardiometabolic disease and CVD has emerged.[27,28] Blacks may be at especially high risk of achieving fewer components of ideal health relative to the general population: recent results from Djoussé et al[4] in over 5000 blacks in JHS demonstrate that over 60% of JHS participants had fewer than 3 components of Life's Simple 7, with a dramatically low prevalence of ideal diet (particularly sodium intake). The higher prevalence of HF and LV remodeling in blacks warrants a direct investigation of the impact of health characteristics and behaviors on comprehensive cardiometabolic phenotypes.

Table 2. Association of Life's Simple 7 With Incident HF

	Model 1 HR	95% CI	*P* Value	Model 2 HR	95% CI	*P* Value
Simple 7 score of 0–2	Referent	Referent	Referent	Referent	Referent	Referent
Simple 7 score of 3	0.53	(0.39–0.73)	<0.0001	0.52	(0.36–0.74)	0.0003
Simple 7 score of 4–6	0.39	(0.24–0.64)	0.0002	0.42	(0.24–0.72)	0.002
No. of participants	3858			3749		
No. of events	239			185		

Model 1 is adjusted for age and sex. Model 2 excludes adjudicated incident myocardial infarction and fatal coronary heart disease at any time in JHS (Jackson Heart Study) and is adjusted for age and sex. Incident HF was defined as self-reported HF hospitalizations (before January 1, 2005) and formally adjudicated HF (after January 1, 2005).CI indicates confidence interval; HF, heart failure; and HR, hazard ratio.

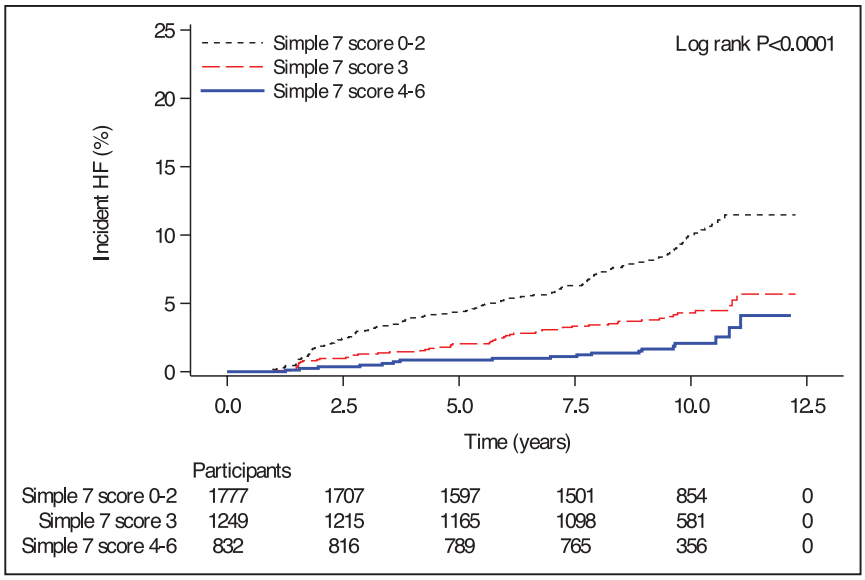

Figure 2. Survival curves for incident heart failure (HF) by Simple 7 score category. Kaplan–Meier survival curves for HF-free survival stratified by Simple 7 scores of 0 to 2, 3, and ≥4. In this analysis, incident HF was defined as either self-reported or formally adjudicated incident HF. Two-hundred thirty-nine incident HF events occurred in 3858 participants.

In the ARIC study (Atherosclerosis Risk in Communities), Shah et al[5] used echocardiography to demonstrate greater LV mass, impaired LV strain, increased HF, and CVD risk with a decline in the number of ideal health components achieved over time. Although this study included 24% blacks (1416 from Jackson, given overlap between ARIC and JHS), it did not address specific CMR-based parameters of structure/function, and it did not investigate the associations with individual components of ideal cardiovascular health. Our study expands these previous efforts by focusing squarely on the largest cohort of blacks with detailed CMR assessments of phenotypes central to HF development. We found that the achievement of fewer components of ideal health was associated with greater incident HF risk, independent of age, sex, or interval CHD development. In multivariable models for HF, we found that elevated blood pressure, physical inactivity, smoking, and impaired glucose control were associated with incident HF. Although hypertension, smoking, and diabetes mellitus were significant contributors to incident HF risk in both Black and White Americans in ARIC,[29] the impact of physical activity is not well defined. In our study, physical inactivity was not associated with LV structure and function but was associated with incident HF, suggesting a possible myocardial-independent contribution of physical inactivity on HF risk,[30] a finding that motivates further prospective investigation.

Of the different pillars of Life's Simple 7, similar factors to those associated with incident HF, including hypertension, impaired glucose control, smoking, but also BMI in the overweight or obese range, were associated with a constellation of myocardial phenotypes classically observed before the onset of frank HF (specifically HF with preserved ejection fraction), including greater hypertrophy,[31] concentric LV remodeling, and higher LV strain by CMR. In the black community, hypertension is highly prevalent and an established risk factor for LV hypertrophy and ensuing HF development.[32,33] We add to the current literature by identifying obesity and insulin resistance as correlates of unfavorable

Table 3. Simple 7 Metrics Associated With Incident HF

	Univariate Analysis			Multivariate Analysis			
	HR	95% CI	*P* Value	HR	95% CI	*P* Value	Adjusted PAR %
Age	1.08	(1.06–1.09)	<0.0001	1.07	(1.06–1.09)	<0.0001	
Female sex	1.14	(0.86–1.49)	0.36	1.08	(0.81–1.43)	0.61	
Smoking	1.43	(1.03–2.01)	0.04	2.04	(1.43–2.91)	<0.0001	12.5 (6.7–18.2)
Nonideal BMI	1.07	(0.74–1.55)	0.72	1.11	(0.75–1.64)	0.62	
Nonideal nutrition	1.67	(0.24–11.93)	0.61	1.82	(0.25–13.00)	0.55	
Nonideal physical activity	2.37	(1.55–3.65)	<0.0001	1.65	(1.07–2.55)	0.02	6.2 (1.0–11.4)
Nonideal BP	5.25	(2.94–9.38)	<0.0001	2.32	(1.28–4.20)	0.005	16.0 (5.4–26.3)
Nonideal fasting plasma glucose	2.44	(1.88–3.16)	<0.0001	1.76	(1.34–2.29)	<0.0001	8.7 (5.0–12.4)
Nonideal total cholesterol	1.41	(1.09–1.84)	0.01	1.01	(0.77–1.31)	0.96	
All 4 significant Simple 7 factors							37.1 (20.0–51.9)

All ideal cardiovascular health factors were included simultaneously in the multivariate model. Each PAR was adjusted for the remaining 3 Simple 7 factors (that were significantly associated with HF; eg, BMI, physical activity, blood pressure, and glucose), age, and sex. BMI indicates body mass index; BP, blood pressure; CI, confidence interval; HF, heart failure; HR, hazard ratio; and PAR, population attributable risk.

Table 4. Associations Between Life's Simple 7 Components and CMR Imaging Indices

	LV Mass Index, g/m$^{2.7}$*		LV Concentric Remodeling Index, g/mL*		Peak Global Strain (%)		Aortic PWV, m/s*	
Ideal Cardiovascular Health	LS Mean	*P* Value	LS mean	*P* Value	LS Mean	*P* Value	LS Mean	*P* Value
Nonsmoking	28.95	0.01	0.98	<0.0001	−16.11	0.0002	5.20	0.0001
Smoking	30.32		1.07		−15.31		6.09	
Ideal BMI	26.65	<0.0001	0.97	<0.0001	−16.12	<0.0001	5.90	0.01
Nonideal BMI	32.93		1.09		−15.29		5.36	
Ideal nutrition	29.65	0.98	0.98	0.34	−15.89	0.66	4.61	0.02
Nonideal nutrition	29.60		1.07		−15.53		6.87	
Ideal physical activity	29.61	0.94	1.03	0.95	−15.78	0.37	5.71	0.33
Nonideal physical activity	29.64		1.03		−15.64		5.54	
Ideal BP	28.38	<0.0001	0.98	<0.0001	−15.80	0.28	5.51	0.17
Nonideal BP	30.92		1.07		−15.62		5.75	
Ideal fasting plasma glucose	28.86	0.002	1.01	0.15	−15.95	0.01	5.70	0.51
Nonideal fasting plasma glucose	30.41		1.04		−15.47		5.56	
Ideal total cholesterol	29.97	0.06	1.02	0.35	−15.72	0.90	5.62	0.94
Nonideal total cholesterol	29.29		1.03		−15.70		5.63	

Linear models were constructed for each imaging parameter (dependent variable) as a function of age, sex, and each Simple 7 component to obtain least squares means. All Simple 7 components were included simultaneously in the same model. *P* values of <0.007 were considered significant using Bonferroni correction. BMI indicates body mass index; BP, blood pressure; CMR, cardiac magnetic resonance; LS, least squares; LV, left ventricle; and PWV, pulse wave velocity.

*Least squares means of log-transformed variables (LV mass index, LV concentric remodeling index, and aortic PWV) were exponentiated.

LV phenotypes and possible targets for personalized HF prevention in blacks in keeping with a previous report by Shah et al[5] and from others.[34,35]

The strengths of our study include the use of a large cohort of blacks with long-term follow-up, extensive cardiometabolic phenotyping, and careful adjudication of Life's Simple 7 ideal

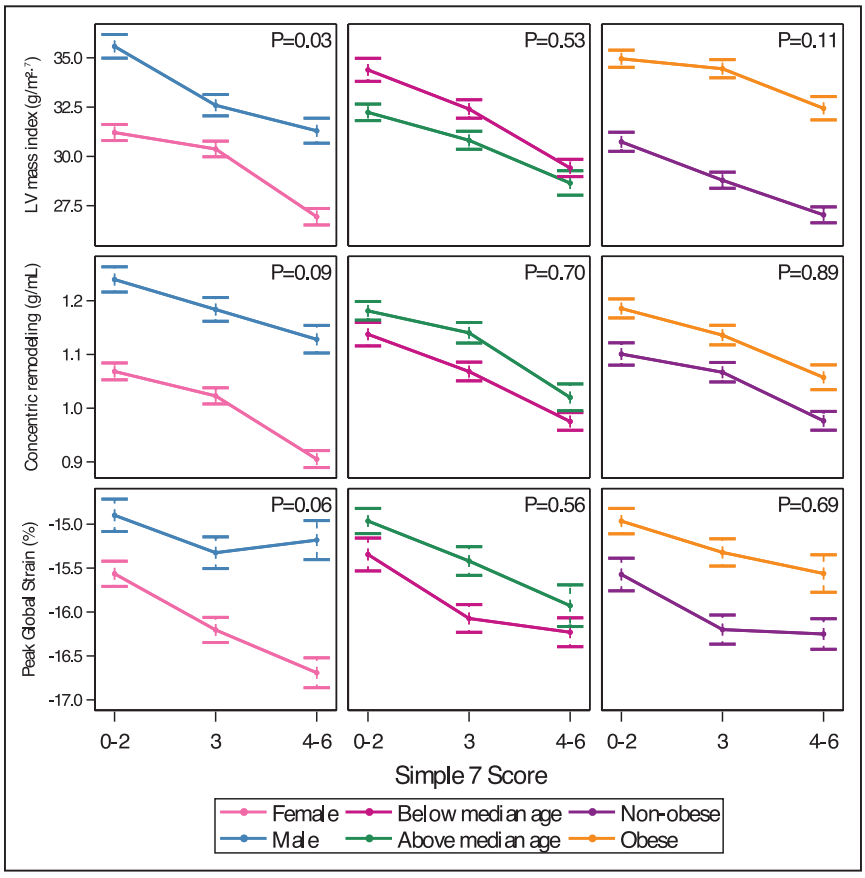

Figure 3. Cardiac magnetic resonance measures of cardiac remodeling, stratified by median age, sex, and obesity. Data points represent least squares means (obtained in adjusted linear models), with error bars representing SE of the mean. *P* values for interaction between Simple 7 score category and each stratum are presented. LV indicates left ventricle.

health metrics. Nevertheless, the results of our study should be viewed in the context of its design. Although low rates of incident HF in JHS may have limited our ability to assess relationships in select subgroups (eg, by presence/absence of atherosclerotic CVD), we nonetheless observed significant associations between achievement of fewer ideal health metrics and HF. Future studies might focus on the prognostic relevance of Life's Simple 7 metrics in blacks with established atherosclerotic CVD. Although we did not observe significant associations between dietary habits and incident HF, we may have been limited by power, given the low proportion of participants achieving ideal nutrition. An inherent limitation of the JHS study is that CMR imaging was performed in a subset of the JHS participants who achieved more ideal health metrics in general compared with participants who did not undergo CMR imaging. Despite these limitations, the study population represents the largest population of blacks with detailed imaging-based phenotyping of cardiometabolic risk. In addition, we chose to investigate cardiovascular phenotypes as a function of baseline Life's Simple 7 metrics and did not consider changes in these metrics over time, which require further longitudinal follow-up and are a fruitful area of future investigation. The PARs denoted here are adjusted for age and sex, and there may be other unmeasured confounders that influence the magnitude of attributable risk; nevertheless, our results are consistent with the common theme of hypertension and diabetes mellitus as critical to HF development. Finally, although HF determination was complex (formal adjudication after January 1, 2005 and self-report before January 1, 2005), observed associations remained robust to the exclusion of self-reported HF.

In conclusion, in this contemporary, large cohort of blacks, achievement of fewer components of ideal health was associated with adverse cardiovascular remodeling and HF development. These findings highlight the importance of ideal cardiovascular health in blacks and provide potential, specific targets for personalized HF prevention in this population.

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Disclosures

Dr Murthy has minor stockholdings in General Electric. The other authors report no conflicts.

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CLINICAL PERSPECTIVE

The lifetime risk of heart failure (HF) is higher in the black population than in other racial groups in the United States. We studied the relationship of the American Heart Association Life's Simple 7 ideal cardiovascular health metrics with cardiac structure and HF over 10 years in the Jackson Heart Study. Achievement of fewer ideal Simple 7 components in blacks was strongly associated with greater incident HF risk, as well as abnormal subclinical cardiac remodeling by cardiac magnetic resonance imaging. Elevated blood pressure, physical inactivity, smoking, and impaired glucose control were associated with increased HF risk in blacks. These findings highlight the importance of ideal cardiovascular health in blacks and provide potential specific targets for personalized HF prevention.

Ideal Cardiovascular Health, Cardiovascular Remodeling, and Heart Failure in Blacks: The Jackson Heart Study

Aferdita Spahillari, Sameera Talegawkar, Adolfo Correa, J. Jeffrey Carr, James G. Terry, João Lima, Jane E. Freedman, Saumya Das, Robb Kociol, Sarah de Ferranti, Donya Mohebali, Stanford Mwasongwe, Katherine L. Tucker, Venkatesh L. Murthy and Ravi V. Shah

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SUPPLEMENTAL MATERIAL

Supplemental Table 1. Construction of Life's Simple 7 score.

Metric	Ideal	Non-ideal
Smoking	Never or quit ≥ 12 months ago	Current or quit <12 months ago
BMI	< 25 kg/m^2	≥25 kg/m^2
Nutrition[*]	4-5 healthy components	0-3 healthy components.
Physical activity	≥150 min/week moderate intensity, or ≥75 min/week vigorous intensity, or ≥150 min/week of combined moderate and vigorous intensity	<150 min/week moderate intensity or <75 min/week vigorous intensity or < 150 min/week of combined moderate and vigorous intensity
BP	Untreated and <120/<80 mmHg	Treated to <120/<80 mmHg or untreated ≥ 120/ ≥ 80 mmHg
Fasting plasma glucose	Untreated and fasting plasma glucose <100 mg/dL or HbA1c < 5.7%	Fasting plasma glucose ≥ 100 mg/dL, HbA1c ≥ 5.7% or treated
Cholesterol	Untreated and < 200 mg/dL	< 200 mg/dL if treated or ≥ 200 mg/dL

[*]Based on 5 health dietary metrics (≥ 4.5 cups of fruits and vegetables a day, ≥ two 3.5 ounces of fish servings per week, ≥3 ounce servings of fiber rich whole grains a day, < 1500 mg sodium per day, and <36 fluid ounces per week of sugar-sweetened beverages). BMI = Body mass index; BP = blood pressure.

Supplemental Table 2. Baseline characteristics for JHS participants who underwent CMR imaging at Visit 3 versus those who did not undergo CMR imaging at Visit 3.

	No CMR Imaging at Visit 3 n = 2993[*]	CMR imaging at Visit 3 n=1202[†]	p value
Demographics			
Age, y	56.8 (45.4-65.7)	50.6 (43.4-59.1)	<0.0001
Female	1960 (65.5)	756 (62.9)	0.11
BMI, kg/m^2	30.6 (26.9 - 35.8)	30.1 (26.5-34.7)	0.009
Simple 7 score component			
Non-Smoking	2568 (85.8)	1060 (88.2)	0.04
Ideal BMI	444 (14.8)	167 (13.9)	0.43
Ideal Nutrition	22 (0.7)	8 (0.7)	0.81
Ideal Physical Activity	556 (18.6)	276 (23.0)	0.001
Ideal BP	569 (19.0)	322 (26.8)	<0.0001
Ideal Fasting Plasma Glucose	2268 (75.8)	1015 (84.4)	<0.0001
Ideal Cholesterol	1320 (44.1)	604 (50.3)	0.0003

P values compare CMR imaging to no imaging. Values are median (25th-75th percentile) or n (%). Nonparametric tests (continuous variables) or chi-square testing (categorical variables) were used to determine P values. BMI = Body mass index; BP = Blood Pressure; CMR = Cardiac magnetic resonance. [*]Of note, some of these individuals may have had CMR at Visit 2. [†]Of 1202 participants who underwent CMR, 1188 had complete data on strain, and were included in our CMR analyses.

Supplemental Table 3. Analysis of the association of Life's Simple 7 with formally adjudicated incident HF.

	Model 1 HR	95% CI	p value	Model 2 HR	95% CI	p value
Simple 7 score 0-2	referent	referent	referent	referent	referent	referent
Simple 7 score 3	0.52	(0.35, 0.75)	0.0006	0.47	(0.30, 0.73)	0.0009
Simple 7 score 4-6	0.42	(0.24, 0.76)	0.004	0.45	(0.24, 0.85)	0.01
Number of participants	3661			3568		
Number of events	163			125		

Model 1 is adjusted for age and sex. Model 2 excludes adjudicated incident MI and fatal CHD at any time in JHS and is adjusted for age and sex. Incident HF was defined as formally adjudicated HF (after 1/1/2005). Abbreviations: CI = confidence interval; HR = hazard ratio; HF = heart failure