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Effect of Race and Socioeconomic Status on Cardiovascular Risk Factor Burden: The Cooper Center Longitudinal Study

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Abstract

Objectives—This study examines the prevalence of cardiovascular risk factors and chronic disease burden among African Americans compared to Caucasians in a population of higher socioeconomic status.

Design—The current study is a cross-sectional, secondary data analysis of the Cooper Center Longitudinal Study.

Setting—Patients with a medical examination from 1970-2010 at the Cooper Clinic.

Participants—762 African Americans and 40,051 Caucasians who met the criteria.

Outcome Measures—Racial differences in cardiovascular risk factors/burden of disease between African Americans and Caucasians.

Results—African Americans had higher prevalence of evaluated cardiovascular risk factors than did Caucasians after controlling for obesity, tobacco use, and physical fitness. Caucasians had greater likelihood of no risk factors while African Americans were more likely to have all three risk factors. Race was typically predictive of cardiovascular risk factors in African Americans compared to Caucasians.

Conclusions—Findings suggest that health differences persist despite greater socioeconomic status, and further investigations of biopsychosocial causes are warranted. *(Ethn Dis.* 2013;23[1]: 35-42)

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Keywords

Socioeconomic Status; Racial/ethnic Minorities; Preventive Medicine; Cardiovascular Risk Factors

Introduction

Despite the increasing life expectancy for the US population, a greater burden of disease continues to exist among African American (AA) men and women compared to their Caucasian counterparts. In 2007, the age-adjusted death rate was 1.3 times greater for African Americans than Caucasians.[1] African Americans' lifespan is 4.8 years (on average) less than Caucasians.[1] Cardiovascular disease (CVD) in African Americans accounted for 44.6% and 46.9% of deaths in men and women, respectively, in 2006.[2,3] Data from the Centers for Disease Control and Prevention further demonstrate racial/ethnic health disparities by a 32.3% higher mortality rate due to strokes among African Americans compared to Caucasians.[3] In addition to CVD, African Americans also have a higher prevalence of cardiovascular risk factors including hypertension, obesity, diabetes mellitus, and physical inactivity.[2] For example, 48% of African Americans have multiple cardiovascular risk factors vs 37% of the remainder of the population.[4] While race is commonly explored in the health disparities literature, its meaning and relationships to health are routinely discussed for additional clarity in these studies.

The meaning of race has varied over the decades to refer to physical traits or phenotypical features[5] or a social political construct that places certain races above or below another race in a so-called hierarchy.[6,7] The lack of a conceptual consensus, varied methods to assess, and nebulous operational definitions of race across disciplines has led to a growing methodological literature to further clarify the meaning, influences, and related factors to race. For example, the construct of ethnicity, which is not synonymous with race, could replace race in health disparities research as this term refers to values, customs, behaviors, and/or traditions.[8] This construct can be added to a study with an acculturation measure.[9] The influence of genes and/or environment on race and health outcomes has advanced with the growth of technology, genetic research, and quantitative methodologies.[10] Furthermore, theory-driven constructs that are strongly correlated to race or certain races (eg, discrimination), while not reinforcing stereotypes, can be used to better delineate the relationships between race and various outcomes.[11] Unfortunately, race has been confounded by low socioeconomic status (SES) over the past decades where it is not clear if low SES and/or race is contributing to specific poor health outcomes.[12]

Socioeconomic status pertains to occupation, income, employment, and at times ZIP Code and insurance status.[13] While it is optimal to have all of these indicators of SES in health research, survey design, report bias, and privacy of the participant may prevent an actual or valid assessment of all indices. Race as previously defined does not explicitly explain or describe these prior areas of SES. Thus, the relationships are not between race and SES, and their influence on outcomes must be clearly defined in health research. Past research has attempted to do such where racial health inequality may stem predominantly from SES

disadvantages in education, income, and employment status (ie, lower SES).[14,15] The literature has attempted to address this prior relationship by statistically controlling for SES which has worked for some health outcomes (asthma) compared to others (hypertension).[16,17] Thus, other factors can be influencing racial group differences. Discrimination at the personal and health care system level, medical mistrust, limitations in environmental protection, economic development, and limited access to health care also may contribute.[18-22] Inadequate health insurance and access to quality health care are critical elements that impact medical care among racial/ethnic minorities.[23] Studies demonstrate strong relationships between these sociodemographic and psychosocial factors and greater CVD mortality in African Americans.[24,25] There are limited investigations in racial/ethnic minorities with higher SES, comparable to Caucasians, receiving preventive medical services.[22,23] Thus it is not clear if racial/ethnic minorities compared to Caucasians, both with high SES levels, have similarly good health outcomes or the racial/ethnic minorities have poorer health similar to findings of minorities with lower SES in the health inequality literature. These issues may prevent a full understanding of the factors contributing to health and disease disparities between racial/ethnic minorities and non-racial/ethnic minorities. Studies assessing higher SES (ie, college educated and/or currently employed) for racial/ethnic minorities are warranted.

Our study compares the prevalence of cardiovascular risk factors and chronic disease burden between African Americans and Caucasians of higher SES and their access to prevennve health care.

Methods

The Cooper Center Longitudinal Study (CCLS) is a large cohort of men and women who underwent a preventive medical examination at the Cooper Clinic in Dallas, Texas from May 1970 to July 2010.[26] Reported results are obtained from 762 self-identified African Americans, 61% men, and 40,051 Caucasians, 68% men, with complete cross-sectional data. Cooper Clinic patients are of higher SES and are self-referred or executives referred by their employer for these extensive preventive medicine examinations. Prior CCLS papers have described this database as primarily White, higher SES, and male.[27,28] For the purpose of this investigation, high SES was defined as education attainment and ability to obtain said examination. This data set presents a unique opportunity to examine two racial groups (one being a historically defined minority group) from similar, higher SES backgrounds (Table 1). Patients consented and approved the use of their data for research. The study was approved by The Cooper Institute Institutional Review Board.

The medical exam included self-reported personal and family history and demographic information, physical examination by a physician, anthropometric measurements, electrocardiogram, blood chemistry analyses after an overnight fast of 12 hours, blood pressure assessment, and maximal exercise treadmill test. Weight and height were obtained using a standard physician's scale and stadiometer and used to calculate body mass index (BMI) in kilograms per meter squared (kg/m^2). Obesity was defined as a BMI of ≥ 30 kg/m^2. Waist circumference was measured on a horizontal plane at the level of the umbilicus with a tape measure. Blood pressures were measured using the auscultatory technique with a

mercury sphygmomanometer. Blood chemistry was analyzed using automated methods that corresponded to standards set by the CDC's Lipid Standardization Program. Cardiorespiratory fitness (CRF) was determined by the maximal time on the treadmill during the modified Balke protocol. A correlation of r=.92 was established between V02 max and Balke suess test time in minutes.[29] A 5-level self-report physical activity (PA) index was calculated where 0 indicated no regular PA; 1, PA other than walking, jogging, or running; 2, walking, jogging, or running <10 miles per week; 3, 10 to 20 miles per week; and 4, >20 miles per week. This scale has been used as a valid measure of PA within the CCLS population.[30,31] The Center for Epidemiologic Studies-Depression scale assessed the presence of depressive symptoms over the previous week. Scores range from 0-30; ≥10 indicates clinically significant depression warranting evaluation.[32]

The three risk factors for cardiovascular disease are defined below. Dyslipidemia was clinically defined as total cholesterol ≥200mg/dL, and/or high density lipoprotein cholesterol (HDL-C) ≤40mg/dL, and/or low density lipoprotein cholesterol (LDL-C) ≥130mg/dL, and/or triglycerides ≥150mg/dL, and/or a self-reported history of hyperlipidemia. Hypertension and diabetes were based on self-reported history and/or SBP >140 and/or DBP >90 and self-reported history and/or fasting blood glucose >126 mg/dL, respectively.

Statistical Analyses

Descriptive analyses, chi-square, and Student's *t* tests, when appropriate, were conducted on sociodemographics (eg, age, SES, education and employment, marital status), health behaviors (eg, tobacco use, PA), self-reported chronic diseases, and phenotypic parameters (eg, BMI, glucose, blood pressure). *T* tests were adjusted using Satterthwaite method to account for unequal sample size and variance when appropriate.[33] Race/ethnicity findings were reported by sex. Multivariate logistic regression determined the association of race with three relevant cardiovascular risk factors (hypertension, dyslipidemia, and diabetes mellitus). The models were adjusted for race, age, SES, tobacco, obesity, and/or CRF. The number of cardiovascular risk factors (0-3) was determined for sex and racial strata. $P \leq .05$ was considered significant. Analyses were performed in SAS versiOn 9.1 (SAS Institute, Cary, NC).

Results

Demographic and clinical baseline characteristics (average age = mid-40s) are summarized in Table 1. Among women, there were significant racial differences in age, education, employment, and personal perception of overall health. African American women were younger, more likely to be employed, but less likely ro be married than Caucasian women. African American men were younger, more likely to be employed, and used less tobacco than Caucasian men. Caucasians self-reported fewer chronic diseases, other than cancers, vs African Americans. African Americans had a significancly greater prevalence of self-reported hypertension (27.4% vs. 17.5% for men [X^2 = 31.3, $P \leq .0001$] and 27.10/0 vs. 13.5% for women [X^2 = 45.3, $P \leq .000l$]) and diabetes (5.4% vs. 2.0% for men [X^2 = 26.0, $P \leq .0001$] and 4.4% vs. 1.9% for women [X= 9.27, P=.002]). African Americans had higher

prevalence of obesity as well as greater systolic blood pressure and lower CRF than their Caucasian counterparts (Table 1).

Figures 1a and 1b illustrate the burden of cardiovascular risk factors by absolute numbers, comparing African Americans with Caucasians by sex. A greater proportion of Caucasian men reported none of the identified cardiovascular risk factors (20.5% vs 15.8% in African Americans, *P*=.012). A similar pattern was identified in women (Caucasian [39.9%] vs. African-American [29.1%], *P*<.001). A greater proportion of African American men (4.6% vs 2.4%, *P*=.00l) had all three of the risk factors.

Adjusted logistic regression models in Table 2 for African American men show greater odds for diabetes (OR= 1.94, 95% CI, 1.37, 2.75, *P*=.002) and hypertension (OR= 1.35, 95% CI, 1.11, 1.64, *P*=.002). The odds of prevalent hypertension in African American women in the unadjusted model was higher (OR=2.20, 95% CI, 1.74, 2.78, *P*<.001) and diabetes (OR=2.19, 95% CI, 1.33, 3.62, *P*=.002). After adjusting for obesity, however, the odds of diabetes in African American women, while greater than Caucasian, becomes statistically non-significant (OR= 1.58, 95% CI, .094, 2.66, *P*= .087).

Discussion

Our study explored the relationship between sociodemographic factors including race and higher SES on CVD risk burden in African Americans and Caucasians with access ro a preventive medical examination. African Americans presented with a greater prevalence of the self-reported chronic diseases. African Americans had greater odds of prevalent hypertension and diabetes mellitus as compared ro Caucasians. Past research indicated greater morbidity from diabetes and hypertension for African Americans than Caucasians as well.[34] In multivariate analyses, obesity strongly influences the risk of diabetes in African American women; in contrast, African Americans had lower risk for the lipid disorders.[35] Our srudy found sex-specific health disparities between African Americans and Caucasians, and we examined the impact of additional covariates (obesity, tobacco use, age, SES, and CRF) that can increase cardiovascular risk factor burden. Race as a predictor appeared to be independent, given that our SES was homogenous and should not confound our analyses of other prevalent cardiovascular risk factors in the models. Addressing the overall burden of cardiovascular risk factors as a sum, Caucasians overwhelmingly had greater prevalence of being free of these factors. Despite being socioeconomically similar, African Americans had higher prevalence of multiple risk factors (3 factors). Similar to other health disparities research, our findings demonstrated racial/ethnic differences in chronic disease risk factor burden such as hypertension.[14,36,37]

Does race, SES, or combination affect health disparities between African Americans and Caucasians?[14] Although SES is a complex variable to measure, it is seen as a predictor of racial/ethnic minority health outcomes. Past reports were inconsistent regarding health disparities after controlling for SES.[38,39] We conrrolled for SES, but racial health disparities still existed. Greater understanding of these health disparities and correlates is needed. Our study suggests that being African American has unique medical implications, independent of SES, which must be taken into account by health care providers.

Recently, a debate has risen regarding the impact of race and ethnicity on health and disease status, stemming from the lack of clarity of the role of race/ethnicity within the genetic, environmental, psychosocial, and cultural milieu.[40,41] Some factors can be linked to theories about the psychosocial and cultural implications of race.[42,43] One theory is the "diminishing return hypothesis," suggesting that higher education or SES attained by African Americans does not lead to similar positive health outcomes comparable to Caucasians due to an incongruent relation between a person's daily experiences and economic status that lead to conflict and stress.[42,44] This has rarely been explored in the literature.[14] Our findings provide evidence for this theory. This research gap may be attributed to several factors: 1) prevailing focus on the "minority poverty hypothesis," which suggests disadvantages due to race and lower SES,[45,46] 2) targeted recruitment of lower SES, and 3) under-recruitment of a range of SES in African-American samples. The current study provides a unique opportunity to stan investigations of the diminishing return hypothesis in a cohort of highly educated African Americans with health care access.

To fully understand this hypothesis, our sample's sociodemographics and relations to chronic disease outcomes must be revisited. Despite comparable SES status, African Americans had more self-reponed chronic disease and cardiovascular risk factors. A plausible interpretation of these findings can come from the diminishing return hypothesis. But, our cohort may have experienced poorer health outcomes because of negative psychosocial factors such as stress, mistreatment/discrimination, less marital support, and/or lower quality of life. Overall health (a proxy for quality of life) is statistically lower for African American vs Caucasian women.[47] However, our study found a lack of depressive symptoms (a proxy for stress) based on CES-D in both races.

With higher SES comes greater expectations of certain lifestyles and quality of life.[42,43] If these expectations aren't met, an individual may experience a greater level of stress and/or allostatic load. [48,49] Stress has been associated with increased CVD.[24] A concept, "John Henryism," suggests that active coping methods may result in a counterintuitive activation of the sympathetic nervous system in some African Americans.[50,51] This may lead to greater negative health outcomes. These theories and hypotheses further support the importance of examining psychosocial factors and health outcomes in racial/ethnic minorities across the SES spectrum. This study provides one of the first epidemiological investigations supporting the diminishing return hypothesis as a plausible explanation of these findings.

There are several strengths of this study. The CCLS provides a large sample of African Americans and Caucasians of both sexes with sociodemo-graphic information, self-reported chronic disease history, and phenotypic data at a preventive medicine visit. This is the first CCLS study examining the effects of race, health behaviors, and SES on chronic burden cardiovascular risk factors. These data on African Americans in the CCLS are comparable to other published data such as the Dallas Heart Study and the National Health and Nutrition Examination Survey (Table 3).[52-55] While the absolute disease burden in our sample is lower, the relative burden is consistent with previous literature.[52-55] Our data present clinically relevant information for physicians and social scientists. African Americans deserve a careful evaluation of their cardiovascular risk factors and disease even when presenting with higher SES (eg, education). The risk factors reviewed in this study are

modifiable through health promotion programs (eg, diet, physical activity, stress management).

Several limitations must be taken into account. The comparison samples are unbalanced with respect to absolute numbers of African Americans and Caucasians, although this was addressed with statistical methods. The study is cross-sectional which limits causality. The chronic disease history is self-reported although confirmed by the clinic physician. Education and employment were used as proxies to define SES; however, actual income levels were not available. Our study did not include commonly used stress or quality of life measures such as the Perceived Stress Scale and SF-36.[56,57]

There are three important implications to aid physicians and social scientists working with African Americans. The assumption cannot be made that higher SES levels buffer poorer health outcomes. Health care providers need to proactively address health promotion with African Americans regardless of higher SES. Further research should be directed towards longitudinal epidemiologic evaluation of higher SES African Americans including the complex biopsychosocial needs and the impact on their health outcomes.[24] Additional health disparity research examining the diminishing return hypothesis is warranted in other racial/ethnic minorities of higher SES. Our study presents data on African Americans in the CCLS, which addresses the relationship of race with health among higher SES individuals. Our study will improve the understanding of cardiovascular health issues of African Americans withhigher SES who are typically underrepresented in the literature.

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Our study compares the prevalence of cardiovascular risk factors and chronic disease burden between African Americans and Caucasians of higher SES and their access to preventive health care.

Despite comparable SES status, African Americans had more self-reported chronic disease and cardiovascular risk factors.

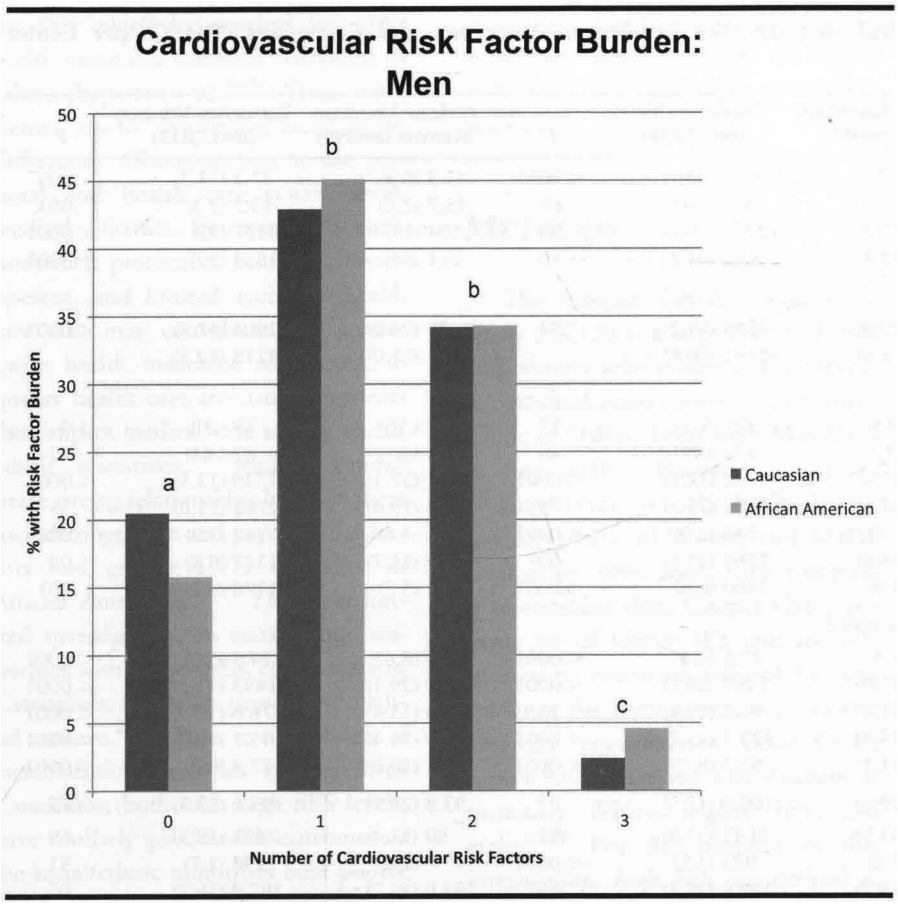

Fig 1a.

Cardiovascular risk factor burden of disease in African American vs Caucasian men, Cooper Center longitudinal Study, 1970-2010. Risk factor burdens adjusted for obesity, and include diabetes, hypertension, and high cholesterol

[a]*P*=.012

[b]not significant

[c]*P*=.001

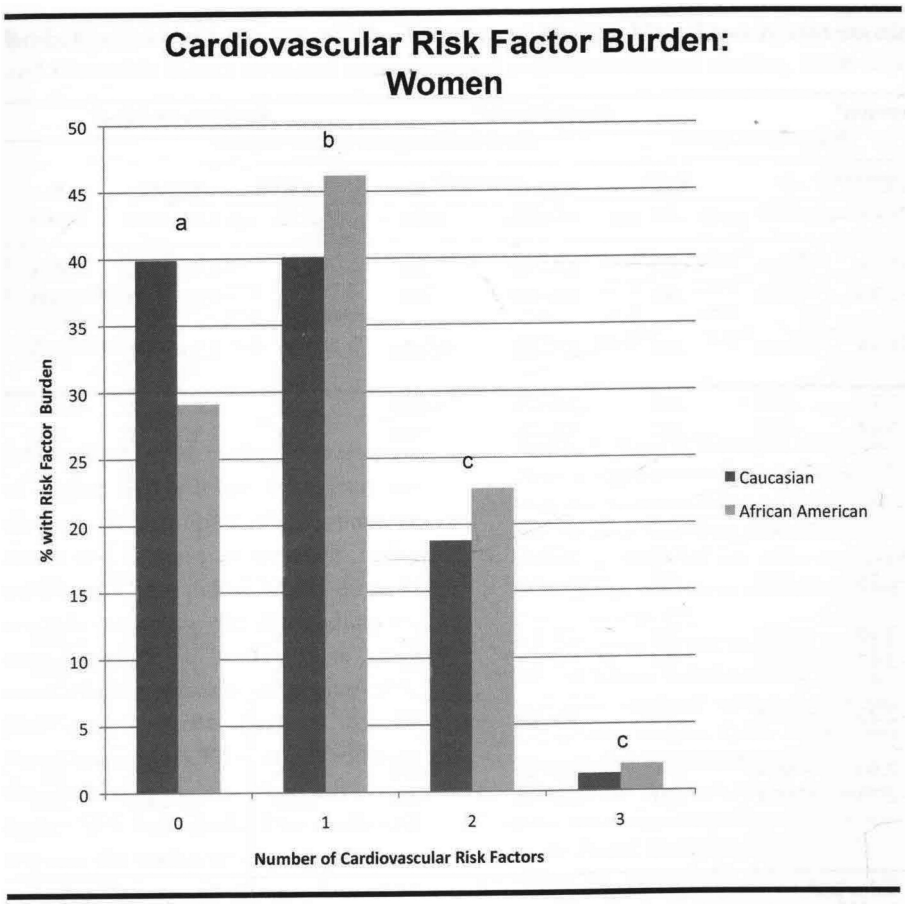

Fig 1b.

Cardiovascular risk factor burden of disease in African American vs Caucasian women, Cooper Center Longitudinal Study, 1970-2010. Risk factor burdens adjusted for obesity, and include diabetes, hypertension, and high cholesterol

[a]*P*<.001

[b]*P*=.034

[c]not significant(ns)

Table 1

Demographic and baseline clinical characteristics for African Americans and Caucasians, The Cooper Center Longitudinal Study, 1970-2010

	African American Men (*n*=463)	Caucasian Men (*n*=27,238)	*P*	African American Women (*n*=299)	Caucasian Women (*n*=12,813)	*P*
Age, years, mean (SD)	44.1 (8.7)	46.3 (10.1)	<.0001	45.3 (8.9)	47.2 (11.2)	.003
Education, years, mean (SD)	16.0 (2.4)	16.1 (2.4)	.68	15.7 (2.6)	15.2 (2.3)	.003
Employment, yes, *n* (%)	348 (75.2)	19215 (70.5)	.03	194 (65)	5427 (42)	<.0001
Marital history, yes, *n* (%)	428 (92.4)	25667 (94.2)	.10	251 (84)	11838 (92)	<.0001
Overall health, *n* (%)						
Poor/fair	83 (20.4)	4393 (19.2)	.54	78 (29.0)	2004 (17.9)	<.0001
Good/excellent	324 (79.6)	18491 (80.8)		191 (71.0)	9218 (82.1)	
Chronic diseases – Self-reported history – *n* (%)						
Heart attack	2 (.43)	325 (1.2)	.13	1 (.33)	49 (.38)	1.0
Stroke	1 (.22)	133 (.49)	.40	2 (.67)	82 (.84)	.72
Hypertension	127 (27.4)	4752 (17.5)	<.0001	81 (27.1)	1730 (13.5)	<.0001
High cholesterol	114 (24.6)	6852 (25.2)	.79	58 (19.4)	2788 (21.8)	.33
Diabetes	25 (5.4)	546 (2.0)	<.0001	13 (4.4)	242 (1.9)	.002
Metabolic syndrome	74 (16.0)	5768 (21.1)	.007	35 (11.7)	1127 (8.8)	.08
Cancer	9 (1.9)	1800 (6.6)	<.0001	11 (3.7)	1178 (9.2)	.001
Phenotypic parameters – mean (SD) unless otherwise stated						
Body mass index, kg/m^2	29.7 (5.3)	27.6 (4.3)	<.0001	28.2 (6.6)	24.5 (5.0)	<.0001
BMI ≥30 kg/m^2, *n* (%)	162 (35.0)	5469 (20.1)	<.0001	87 (29.1)	1453 (11.3)	<.0001
Waist circumference, cm	96.0 (11.4)	95.2 (11.4)	.26	82.8 (13.4)	76.8 (11.9)	<.0001
SBP, mm Hg	126.2 (14.4)	123.3 (13.7)	<.0001	120.6 (16.4)	115.7 (16.0)	<.0001
DBP, mm Hg	85.8 (11.1)	82.7 (9.7)	<.0001	81.4 (10.8)	77.3 (9.8)	<.0001
Glucose, mg/dL	102.4 (29.3)	100.0 (17.5)	.09	93.9 (20.1)	93.6 (13.9)	.77
100-125, *n* (%)	139 (31.5)	9641 (37.4)	.01	50 (17.4)	2191 (18.3)	.69
>125, *n* (%)	31 (7.0)	903 (3.5)	<.0001	7 (2.4)	198 (1.7)	.31
Total cholesterol, mg/dL	198.7 (40.2)	199.6 (38.2)	.65	193.8 (36.7)	197.9 (36.7)	.06
LDL, mg/dL	128.3 (36.4)	125.4 (33.5)	.08	113.8 (30.7)	112.8 (32.3)	.58
HDL, mg/dL	50.0 (11.9)	47.3 (12.1)	<.0001	64.5 (17)	65.0 (16.5)	.57
TG, median (Q_1,Q_4), mg/dL	90.0 (69.0,123.0)	112.0 (80.0,164.0)	<.0001	74 (56.0,102.0)	86 (64.0,125.0)	<.0001
CES-D (total scale)	3.9 (4.0)	4.0 (4.3)	.7	4.0 (4.6)	5.3 (5.2)	.24
Health behaviors - mean (SD)						
PA Index	1.5 (1)	1.5 (1.1)	.46	1.3 (.9)	1.5 (1.1)	<.0001
CRF (METs)	10.6 (2.2)	11.5 (2.4)	<.0001	8.5 (1.8)	9.6 (2.1)	<.0001

BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; LDL, low density lipoprotein; HDL, high density lipoprotein; TC, triglycerides; CES-D, Center for Epidemiologic Studies Depression Scale; CRF, Cardiorespiratory fitness; METs, metabolic equivalents, 1 MET = 3.5ml 0_2 uptake/kg/min.

Table 2

Odds ratios for associations of race on cardiovascular risk factors in men and women, Cooper Center Longitudinal Study, 1970-2010

	Hypertension[a]			Dyslipidemia[b]			Diabetes Mellitus[c]		
Men									
	OR	95% CI	*P*	OR	95% CI	*P*	OR	95% CI	*P*
Unadjusted (Caucasian[e], African American)	1.53	1.28-1.84	<.001	.79	.66-.96	.02	2.15	1.54-3.00	<.001
Adjusted risk for age	1.73	1.43-2.08	<.001	.81	.67-.98	.03	2.69	1.92-3.79	<.001
Adjusted risk for age and tobacco use (no[e], yes)	1.74	1.44-2.10	<.001	.82	.68-.99	.04	2.70	1.92-3.80	<.001
Adjusted risk for obesity[d] (BMI ≤29.9[e], BMI ≥30)	1.32	1.09-1.59	.005	.69	.57-.84	<.001	1.72	1.22-2.41	.002
Adjusted for age and fitness	1.43	1.18-1.73	.002	.65	.54-.79	<.001	2.03	1.43-2.87	<.001
Adjusted risk for age, tobacco (no[e], yes), obesity (BMI ≤29.9[e], BMI ≥30), SES, fitness	1.35	1.11-1.64	.002	.62	.52-.76	<.001	1.94	1.37-2.75	.002
Women									
	OR	95% CI	*P*	OR	95% CI	*P*	OR	95% CI	*P*
Unadjusted (Caucasian[e], African American)	2.20	1.74-2.78	<.001	.85	.67-1.07	.16	2.19	1.33-3.62	.002
Adjusted risk for age	2.87	2.24-3.68	<.001	.93	.73-1.17	.52	2.49	1.51-4.13	<.001
Adjusted risk for age and tobacco use (no[e], yes)	2.87	2.24-3.67	<.001	.93	.73-1.18	.56	2.50	1.51-4.14	<.001
Adjusted risk for obesity[d] (BMI ≤29.9[e], BMI≥30)	1.75	1.37-2.25	<.001	.71	.56-.90	.005	1.42	.85-2.38	.18
Adjusted risk for age and fitness	2.30	1.79-2.96	<.001	.74	.58-.94	.014	1.84	1.10-3.07	.02
Adjusted risk for age, tobacco (no[e], yes), obesity (BMI ≤29.9[e], BMI≥30), SES, fitness	2.11	1.63-2.73	<.001	.70	.55-.90	.005	1.58	.94-2.66	.087

[a] Hypertension = self-reported history and/or SBP >140 and/or DBP >90.

[b] Dyslipidemia = self-reported history and/or high density lipoprotein cholesterol <40 mg/dL and/or low density lipoprotein cholesterol >130 mg/dL and/or triglycerides >150mg/dL.

[c] Diabetes mellitus = self-reported history and/or fasting blood glucose >126 mg/dL.

[d] Obesity = BMI ≥30 kg/m^2.

[e] Referent group.

Table 3

Comparisons of absolute prevalence of obesity, hypertension, hyperlipidemia, and diabetes of African American (AA) and Caucasian (Cauc) men and women among population-based studies, 1970-2010, (%)

	Cooper Center Longitudinal Study				Dallas Heart Study				National Health and Nutrition Examination Study			
	Men		Women		Men		Women		Men		Women	
	AA	Cauc	AA	Cauc	AA	Cauc	AA	Cauc	AA	Cauc	AA	Cauc
Obesity	35	20.1	29.0	11.3	36.0[52]	34.0[52]	59.0[52]	42[52]	37.3[53]	31.9[53]	49.6[53]	33.0[53]
Hypertension	27.4	17.5	27.1	13.5	52.0[52]	28.0[52]	66.0[52]	33[52]	42.4[58]	45.8[58]	57.6[58]	54.2[58]
Dyslipidemia	24.6	25.2	19.4	21.8	2.0	7.4	1.1	2.8	45.6[54]	55.0[54]	47.7[54]	58.7[54]
Diabetes Mellitus	5.4	2.0	4.4	1.9	16.0[52]	7.0[52]	20.0[52]	8.0[52]	7.0[55]	9.0[55]	14.0[55]	7.0[55]