

# Metabolically Healthy Obesity Is Not Associated with Food Intake in White or Black Men<sup>1–4</sup>

Ruth W Kimokoti,<sup>5\*</sup> Suzanne E Judd,<sup>6</sup> James M Shikany,<sup>7</sup> and PK Newby<sup>8</sup>

<sup>5</sup>Department of Nutrition, School of Nursing and Health Sciences, Simmons College, Boston, MA; <sup>6</sup>Department of Biostatistics, School of Public Health, and <sup>7</sup>Division of Preventive Medicine, School of Medicine, University of Alabama at Birmingham, Birmingham, AL; <sup>8</sup>Program in Environmental Sciences, Harvard University Extension School, Boston, MA

## Abstract

**Background:** Healthy obese individuals may be protected against adverse health outcomes. Diet and race might influence healthy obesity, but data on their roles and interactions on the phenotype are limited.

**Objective:** We compared the food intake of metabolically healthy obese men to those of other weight status–metabolic health phenotypes.

**Methods:** Men ( $n = 4855$ ) aged  $\geq 45$  y with BMI  $\geq 18.5$  kg/m<sup>2</sup> and free of cardiovascular diseases, diabetes, and cancer were evaluated in a cross-sectional study of the REGARDS (REasons for Geographic And Racial Differences in Stroke) study cohort. Food intake was assessed with the use of a food frequency questionnaire. Weight status–metabolic health phenotypes were defined by using metabolic syndrome (MetS) and homeostasis model assessment of insulin resistance (HOMA-IR) criteria. Mean differences in food intake among weight status–metabolic health phenotypes were compared with the use of linear regression.

**Results:** MetS-defined healthy obesity was present in 44% of white obese men and 58% of black obese men; the healthy obese phenotype, based on HOMA-IR, was equally prevalent in both white (20%) and black (21%) obese men. Among white men, MetS-defined healthy and unhealthy obesity were associated with lower wholegrain bread intake and higher consumption of red meat ( $P < 0.001$ ), whereas HOMA-IR–defined healthy and unhealthy obesity were associated with lower red meat intake ( $P < 0.0001$ ) compared with healthy normal weight in multivariable-adjusted analyses that adjusted for sociodemographic, lifestyle, and clinical confounders. However, results were attenuated and became nonsignificant after further adjustment for BMI. Healthy and unhealthy overweight, defined by both criteria, were associated with lower whole grain bread intake ( $P < 0.001$ ) in all models. Among black men, weight status–metabolic health phenotypes were not associated with food intake in all models.

**Conclusion:** Healthy obesity in men is not associated with a healthier diet. Future studies need to consider dietary patterns, which may better inform the holistic effect of diet on healthy obesity, in prospective analyses. *J Nutr* 2015;145:2551–61.

**Keywords:** diet, healthy, metabolic, obesity, phenotype

## Introduction

According to the 1994–2004 NHANES, approximately one-quarter (29%) of obese men in the United States are metabolically

healthy (1). These individuals display high levels of insulin sensitivity, favorable inflammation and lipid profiles, and a low prevalence of hypertension, and are hypothesized to be protected from, or to be at substantially lower risk of, obesity-related metabolic complications, including cardiovascular diseases, type 2 diabetes, and mortality (2–7). There is no consensus on the definition of so-called “healthy obesity,” and researchers have generally used insulin sensitivity denoted by low HOMA-IR, absence of metabolic syndrome (MetS)<sup>9</sup>, or a combination of both, to describe the phenotype (2–5). MetS is a multiplex of metabolic risk factors that includes abdominal obesity, elevated blood pressure, hyperglycemia, low HDL cholesterol, and hypertriglyceridemia (8).

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<sup>4</sup> Supplemental Tables 1–5 are available from the “Online Supporting Material” link in the online posting of this article and from the same link in the online table of contents at <http://jn.nutrition.org>.

\* To whom correspondence should be addressed. E-mail: [ruth.kimokoti@simmons.edu](mailto:ruth.kimokoti@simmons.edu).

<sup>9</sup> Abbreviations used: hs-CRP, high-sensitivity C-reactive protein; MetS, metabolic syndrome; REGARDS, REasons for Geographic And Racial Differences in Stroke; SSB, sugar-sweetened beverage; WC, waist circumference.

Healthy obesity may have a genetic basis, but lifestyle factors are also implicated in the etiology of the phenotype (2–5). Several studies that have examined dietary determinants of healthy obesity have demonstrated conflicting results (9–11). Two studies (9, 11) compared dietary intakes in women and men but none of the studies compared intakes among racial/ethnic subgroups. The epidemiology of obesity, body composition, and energy metabolism are shown to differ by sex and race, as are glucose and lipid metabolism, as well as insulin homeostasis (12–16). In our previous studies of the REGARDS (REasons for Geographic And Racial Differences in Stroke) study cohort, sex and race modified the effects of region on intake of *trans* fat, cholesterol, and calcium (17, 18). Sex- and race-specific studies of dietary intake are thus important when conducting dietary studies.

Our objective in this study was to compare the food intake of metabolically healthy obese men to that of other weight status–metabolic health phenotypes in the REGARDS study.

We hypothesized that healthy obese men consume more healthy foods (e.g., whole grains, fish, and low-fat dairy foods) and fewer unhealthy foods [e.g., fried foods, processed meat, and sugar-sweetened beverages (SSBs)] than their unhealthy obese counterparts but have a food intake similar to metabolically healthy normal-weight men. Weight status–metabolic health phenotypes were defined with the use of both MetS and HOMA-IR criteria.

## Methods

### Study population

Design and recruitment strategies for the REGARDS study are described elsewhere (19). The study was initiated in 2003 as a longitudinal study of stroke and its risk factors. Between 2003 and 2007, 30,239 adults aged  $\geq 45$  y (white: 58%; black: 42%) were recruited via mail and telephone by using a commercially available list of residents. Individuals from the “Stroke Belt” (noncoastal regions of North Carolina, South Carolina, and Georgia, as well as Alabama, Arkansas, Georgia, Louisiana, Mississippi, and Tennessee) (35%) and the “Stroke Buckle” (coastal plains of North Carolina, South Carolina, and Georgia) (21%) were oversampled. The remainder (44%) were enrolled from the other 40 contiguous states. The Institutional Review Board for Human Use at the University of Alabama at Birmingham approved the study protocol and all participants provided written informed consent.

At the baseline examination, 13,551 men participated in the main study. Of these, 11,499 men (85%) aged 45–98 y completed an FFQ and comprised our study. Those with  $>15\%$  missing responses ( $n = 1289$ ) and an implausible energy intake [ $<800$  kcal/d or  $>5000$  kcal/d ( $<3347$  kJ/d or  $>20,920$  kJ/d);  $n = 664$ ] were excluded, leaving a total of 9546 men. The final sample included 4855 men (51%) with BMI  $\geq 18.5$  kg/m<sup>2</sup> who were free of cardiovascular diseases, diabetes mellitus, and cancer, and had data on covariates and metabolic risk factors.

### Exposure and covariate assessment

Sociodemographic, lifestyle, and clinical factors were assessed initially via a computer-assisted telephone interview followed by an in-home examination (2003–2007). Additional information was obtained via self-administered mail-in questionnaires (19). Age, marital status, residential region, education, income, multivitamin use, alcohol intake, smoking status, physical activity, television viewing, and medical information were obtained via computer-assisted telephone interview and mail-in questionnaire (19). Information on medication use was obtained during the in-home examination, and phlebotomy and anthropometric and blood pressure measurements were done. To calculate BMI (weight in kilograms divided by height in meters squared), participants dressed in hospital gowns and without shoes were weighed by using a calibrated digital scale (Salter, Salter Brecknell), and height was measured with a metal tape. Waist circumference (WC) was measured midway between the lowest rib on the right side and the top of the iliac crest on standing

participants by using a cloth tape measure (19). Blood pressure was measured on the participant’s left arm with an aneroid sphygmomanometer with the participant in a sitting position; the mean of 2 measurements was used (19, 20). Fasting serum glucose, HDL cholesterol, and TGs were assessed by calorimetric reflectance spectrophotometry with the use of the Ortho Vitros Clinical Chemistry System 9501RC instrument (Johnson & Johnson Clinical Diagnostics). Serum insulin was measured by electrochemiluminescence immunoassay with the use of the Roche Elecsys 2010 system (Roche Diagnostics). Serum high-sensitivity C-reactive protein (hs-CRP) was analyzed by particle-enhanced immunonephelometry with the use of a Behring Nephelometer II analyzer nephelometer (N High-Sensitivity CRP; Dade Behring) (21).

### Definition of weight status–metabolic health phenotypes

Weight status (normal weight, overweight, or obese) as defined by BMI (normal weight: 18.5 to  $<25.0$  kg/m<sup>2</sup>; overweight: 25 to  $<30$  kg/m<sup>2</sup>; and obese:  $\geq 30$  kg/m<sup>2</sup>) was based on the NIH criteria that were adopted from WHO classification (22). Metabolic status was defined according to both the AHA/National Heart, Lung, and Blood Institute guidelines as outlined in the harmonized Joint Scientific Statement criteria for MetS (8) and HOMA-IR criteria (23, 24). MetS is defined as having  $\geq 3$  of the following individual components: abdominal obesity (WC  $\geq 102$  cm); elevated blood pressure ( $\geq 130/\geq 85$  mm Hg) or drug therapy for hypertension; elevated glucose ( $\geq 5.6$  mmol/L) or drug therapy for hyperglycemia; low HDL cholesterol ( $<1.3$  mmol/L) or drug therapy for reduced HDL cholesterol; and elevated TGs ( $\geq 1.7$  mmol/L) or drug therapy for hypertriglyceridemia (16). Insulin resistance was assessed from fasting glucose and insulin concentrations by using the formula HOMA-IR = fasting glucose (millimoles per liter)  $\times$  fasting insulin (picomoles per liter)/22.5, and was defined as the highest quartile of HOMA-IR scores (23, 24).

### Dietary assessment

Food intake was assessed with the use of the 107-item semiquantitative Block 1998 FFQ. For each FFQ item, a common serving size of the food or beverage is specified (e.g., 15 g spinach), and participants indicate the frequency of consumption, on average, of the portion during the preceding year. There are 9 response categories, ranging from “never or less than once per month” to “1 (or 2) or more times per day,” and individuals selected the appropriate serving size. Portion size for unitary items (e.g., eggs) was queried as “1, 2, or 3,” and the number consumed each time was reported. For nonunitary foods, a photo was provided to aid in estimating 4 different portions. For each food, an amount was assigned based on the gram weight of the volume for the selected portion-size model. Participants completed the FFQs at home and mailed them to the study center, where they were checked for completeness and scanned. Scanned FFQ files were then sent to NutritionQuest for processing. The amount of each food consumed was calculated by multiplying the reported frequency by the portion size for each food item. The 107 FFQ items were categorized into 56 food groups based on nutrient similarities (e.g., SSBs and beverages containing some juice, such as Hi-C) and culinary use (e.g., high- and low-fat milk) (Supplemental Table 1) (17, 18). The Block 1998 FFQ has not been validated in the REGARDS study cohort, but a validation study in the nationally representative Eating at America’s Table Study showed moderate to high validity (de-attenuated Pearson correlation coefficient: 0.24–0.77 in men) (25).

Compared with men who did not complete an FFQ ( $n = 1731$ ), those who completed one ( $n = 4855$ ) were somewhat older, more likely to be married and to reside in the Stroke Belt, to have a college degree and annual income  $\geq \$35,000$ , and to be physically active, and less likely to smoke. Additionally, FFQ completers had a higher prevalence of normal weight and overweight, lower prevalence of total obesity, hypertension, and hypertension treatment, and lower blood pressure, mean HOMA-IR index, and insulin concentration (all  $P < 0.01$ , data not shown).

### Statistical analysis

Given the race differences in dietary exposures, we conducted race-specific analyses a priori (17, 18).

**Characteristics.** Participant characteristics analyzed included age, energy intake, BMI, WC, blood pressure, glucose, insulin, HOMA-IR, HDL cholesterol, TGs, and hs-CRP in their continuous form. Residential region [Stroke Belt or other (Stroke Buckle and other 40 contiguous states)], marital status (married or other), education level (<college degree or ≥college degree), annual income (<\$35,000 or ≥\$35,000), alcohol intake (none, moderate, or heavy) (26, 27), multivitamin use (yes or no), cigarette smoking status (nonsmoker or current smoker), physical activity (0 times/wk, 1–3 times/wk, or ≥4 times/wk), television viewing (0 h/wk, 1–6 h/wk, or ≥1 h/d), weight status categories (normal weight, overweight, or obese), elevated WC (yes or no), elevated blood pressure (yes or no), hypertension medication (yes or no), elevated glucose (yes or no), HOMA-IR quartiles (1–3 or 4), low HDL cholesterol (yes or no), elevated TGs (yes or no), and lipid-lowering medication (yes or no) were analyzed as categorical variables. ANOVA was used to compare mean differences in continuous variables between white and black men, as well as to calculate pairwise mean differences in the weight status–metabolic health phenotypes (28). A chi-square test was used to compare differences in proportions of categorical variables in the subgroups of men and to compute pairwise differences in proportions between weight status–metabolic health phenotypes (28). Results were summarized as means ± SEMs for continuous measures and percentages for categorical variables.

**Food intake.** ANCOVA was used to compute age-adjusted least-squares means of food intake and to calculate pairwise mean differences in food intake between white and black men. Linear regression was used to compute age-adjusted and multivariable-adjusted least-squares means of food intake (vegetables, fruits, whole grain bread, refined grains, beans, fish, poultry, red meat, processed meat, fried foods, low-fat dairy, high-fat dairy, 100% fruit juice, and SSBs) and to identify pairwise mean differences in the weight status–metabolic health phenotypes (healthy normal weight, unhealthy normal weight, healthy overweight, unhealthy overweight, healthy obese, or unhealthy obese). The SAS procedure PROC GLM was used to fit models (29). Five hierarchical models were fitted: model 1 adjusted for age; model 2 additionally adjusted for marital status, residential region, education level, income, alcohol intake, multivitamin use, cigarette smoking status, physical activity, television viewing, hs-CRP, and food intake; model 3 further adjusted for BMI; model 4 additionally adjusted for energy intake; and model 5 adjusted for variables in model 3 excluding lifestyle factors (alcohol intake, multivitamin use, cigarette smoking status, physical activity, and television viewing). Data are presented as means ± SEMs.

All analyses were performed with the use of Statistical Analysis Software (version 9.2) (29).  $P < 0.01$  was considered statistically significant. All statistical tests were 2-sided.

## Results

**Characteristics.** White men relative to black men were older and more likely to be married, have a college degree, and an annual income of ≥\$35,000 ( $P < 0.0001$ ). They consumed more alcohol, had higher multivitamin use, and exercised more but smoked less than black men ( $P < 0.001$ ). Additionally, white men compared with black men were more likely to be normal weight and overweight; had a higher prevalence of elevated WC, low HDL cholesterol, hypertriglyceridemia, and lipid-lowering treatment; and had a higher mean TG concentration ( $P < 0.001$ ). Black men, in contrast, had higher mean BMI, systolic and diastolic blood pressure, HOMA-IR index, and concentrations of insulin and hs-CRP. They were also more likely to be obese, insulin resistant, hypertensive, and on hypertensive treatment than white men ( $P < 0.01$ ). There were no differences in energy intake, television viewing, mean WC, glucose concentration, or prevalence of hyperglycemia between white and black men (Table 1).

Intake of vegetables, red meat, and low-fat dairy was higher among white men, whereas consumption of refined grains,

processed meat, fried foods, fruit juice, and SSBs was higher in black men ( $P < 0.0001$ ) (Table 2).

**MetS-defined healthy obesity: Prevalence, clinical characteristics, and food intake.** Defined by the absence of MetS, 43.9% of white obese men and 58% of black obese men were metabolically healthy (Table 3).

Both white and black healthy obese and overweight men had lower mean concentrations of HDL cholesterol than their healthy normal-weight counterparts, but higher concentrations than unhealthy obese and overweight men. Conversely, they had higher mean WCs than did healthy normal-weight men, but lower concentrations than their unhealthy obese and overweight counterparts. Among black men, mean systolic blood pressure and TG concentrations of healthy obese, overweight, and normal-weight men were lower than those of unhealthy obese and overweight men. BMI and diastolic blood pressure of healthy and unhealthy obese men, as well as the BMI of healthy and unhealthy overweight men, were higher than those of their healthy normal-weight counterparts (Table 3).

In age-adjusted analyses, white healthy and unhealthy obese and overweight men consumed significantly lower amounts of whole grain bread than did healthy normal-weight men ( $P < 0.01$ ). They also had a significantly higher intake of red meat and fried foods than did healthy normal-weight men ( $P < 0.0001$ ). Obese men similarly consumed significantly more processed meat and SSBs than did healthy normal-weight men ( $P < 0.0001$ ). Among obese men, findings for whole grain bread and red meat were maintained in multivariable-adjusted analyses ( $P < 0.001$ ). However, these results were attenuated and became nonsignificant after additional adjustment for BMI. Further adjustment for energy intake did not qualitatively alter the results. Among overweight men, results for whole grain bread were maintained in all multivariable-adjusted models ( $P < 0.001$ ). In age-adjusted but not multivariable-adjusted models, fruit consumption among unhealthy obese and overweight men was lower than that of healthy normal-weight men ( $P < 0.001$ ) (Table 4 and Supplemental Table 2).

In both age-adjusted and multivariable-adjusted models, weight status–metabolic health phenotypes were not associated with food intake among black men (Supplemental Table 3).

**HOMA-IR-defined healthy obesity: Prevalence, clinical characteristics, and food intake.** Based on HOMA-IR criteria, the prevalence of healthy obesity was comparable in obese white (20.3%) and black (21.4%) men, in contrast to with the MetS criteria (Table 5).

Both white and black healthy obese, overweight, and normal-weight men had higher mean concentrations of glucose and insulin, and a higher HOMA-IR index than unhealthy obese and overweight men. White and black healthy obese and overweight men likewise had higher mean BMI and WCs than did their healthy normal-weight counterparts, but these were lower than those of unhealthy obese and overweight men. Among white men, mean concentrations of HDL cholesterol in healthy obese and overweight men were lower than those of their healthy normal-weight counterparts but higher than those of unhealthy obese and overweight men. Also, mean systolic and diastolic blood pressure were higher in healthy and unhealthy obese men than in their healthy normal-weight counterparts (Table 5).

In age-adjusted analyses, white healthy and unhealthy obese and overweight men had a significantly lower intake of whole

**TABLE 1** Characteristics of men from the REGARDS study<sup>1</sup>

Characteristic	White	Black	P
<i>n</i>	3726	1129	
Sociodemographic			
Age, y	64.7 ± 0.1	62.8 ± 0.3	<0.0001
Stroke belt	53.6	49.3	0.0108
Married	85.4	68.6	<0.0001
≥College degree	53.5	32.9	<0.0001
≥\$35,000	67.7	52.6	<0.0001
Lifestyle			
Energy intake, <sup>2,3</sup> Mcal/d	1.92 ± 0.01	1.87 ± 0.02	0.10
Alcohol intake <sup>4</sup>			<0.0001
None	42.7	52.8	
Moderate (≤28 g/d)	51.1	42.3	
Heavy (>28 g/d)	6.2	5.0	
Multivitamin use	69.0	56.1	<0.0001
Current smokers	10.3	19.4	<0.0001
Physical activity			0.0005
0 times/wk	22.3	25.9	
1–3 times/wk	38.2	40.9	
≥4 times/wk	39.5	33.2	
Television viewing			0.05
0 h/wk	0.9	0.3	
1–6 h/wk	11.4	12.9	
≥1 h/d	87.7	86.8	
Clinical			
BMI, kg/m <sup>2</sup>	27.7 ± 0.1	28.4 ± 0.1	<0.0001
Weight status category			<0.0001
Normal weight (BMI 18.5 to <25 kg/m <sup>2</sup> )	26.8	25.2	
Overweight (BMI 25 to <30 kg/m <sup>2</sup> )	48.6	43.8	
Obese (BMI ≥30 kg/m <sup>2</sup> )	24.6	31.0	
Waist circumference, cm	98.2 ± 0.2	97.7 ± 0.4	0.33
Elevated waist circumference (≥102 cm)	82.6	77.9	0.0003
Systolic blood pressure, mm Hg	126 ± 0	129 ± 1	<0.0001
Diastolic blood pressure, mm Hg	76.9 ± 0.1	79.3 ± 0.3	<0.0001
Elevated blood pressure (≥130/≥85 mm Hg)	45.4	61.0	<0.0001
Hypertension medication	35.6	50.7	<0.0001
Serum glucose, <sup>5</sup> mmol/L	5.19 ± 0.01	5.21 ± 0.02	0.34
Elevated serum glucose (≥5.6 mmol/L)	25.7	28.3	0.08
Serum insulin, <sup>6</sup> pmol/L	78.5 ± 1.4	88.9 ± 2.1	<0.0001
HOMA-IR index	26.7 ± 0.4	30.6 ± 0.9	<0.0001
Insulin-resistant (HOMA-IR quartile 4)	48.3	54.0	0.0032
Serum HDL cholesterol, <sup>7</sup> mmol/L	1.19 ± 0.01	1.27 ± 0.01	<0.0001
Low serum HDL cholesterol (<1.3 mmol/L)	67.7	57.3	<0.0001
Serum TGs, <sup>8</sup> mmol/L	1.52 ± 0.01	1.27 ± 0.03	<0.0001
Elevated serum TGs (≥1.7 mmol/L)	30.2	16.7	<0.0001
Lipid-lowering medication	28.9	24.9	0.0081
Serum high-sensitivity C-reactive protein, <sup>9</sup> mg/L	28.0 ± 1.0	41.0 ± 4.0	0.0008

<sup>1</sup> Values are means ± SEMs or percentages. Variables are unadjusted. ANOVA was used to compare mean differences in continuous variables between white and black men. A chi-square test was used to compare differences in percentages of categorical variables in the subgroups of men. REGARDS, REasons for Geographic And Racial Differences in Stroke.

<sup>2</sup> To convert kcal to kJ multiply by 4.184.

<sup>3</sup> To convert Mcal to kcal multiply by 1000.

<sup>4</sup> A standard drink is equal to 14 g. For men, moderate alcohol consumption is defined as having up to 2 drinks/d; heavy drinking is defined as consuming ≥15 drinks/wk (26, 27).

<sup>5</sup> To convert glucose to conventional units (mg/dL) divide by 0.0555.

<sup>6</sup> To convert insulin to conventional units (μU/mL) divide by 6.945.

<sup>7</sup> To convert cholesterol to conventional units (μU/mL) divide by 0.0259.

<sup>8</sup> To convert TGs to conventional units (μU/mL) divide by 0.0113.

<sup>9</sup> To convert C-reactive protein to conventional units (μU/mL) divide by 10.

grain bread, as well as a higher consumption of red meat, processed meat, and fried foods ( $P < 0.0001$ ), than did healthy normal-weight men.

Among obese men, results for red meat were significant in multivariable-adjusted analyses ( $P < 0.0001$ ), but these were attenuated and became nonsignificant after further adjustment

**TABLE 2** Age-adjusted mean food intake of men from the REGARDS study<sup>1</sup>

Food groups	White	Black	P
<i>n</i>	3726	1129	
Vegetables, g/d	201 ± 3	156 ± 5	<0.0001
Fruits, g/d	113 ± 2	117 ± 4	0.50
Whole grain bread, g/d	16.7 ± 0.4	18.1 ± 0.7	0.08
Refined grains, g/d	25.0 ± 0.6	33.0 ± 1.1	<0.0001
Beans, g/d	14.6 ± 0.4	16.5 ± 0.8	0.0348
Fish, g/d	21.1 ± 0.5	20.1 ± 0.9	0.34
Poultry, g/d	12.9 ± 0.3	12.9 ± 0.6	0.98
Red meat, g/d	43.8 ± 0.7	31.9 ± 1.3	<0.0001
Processed meat, g/d	21.0 ± 0.4	27.0 ± 0.7	<0.0001
Fried foods, g/d	24.8 ± 0.5	40.9 ± 1.0	<0.0001
Low-fat dairy, g/d	107 ± 3	23.6 ± 5.6	<0.0001
High-fat dairy, g/d	120 ± 3	115 ± 5	0.40
100% fruit juice, g/d	138 ± 3	204 ± 6	<0.0001
Sugar-sweetened beverages, g/d	183 ± 5	254 ± 9	<0.0001

<sup>1</sup> Values are means ± SEMs. ANCOVA was used to compute age-adjusted least-squares means of food intake and to calculate pairwise mean differences in food intake between white and black men. REGARDS, REasons for Geographic And Racial Differences in Stroke.

for BMI. Additional adjustment for energy intake did not alter the findings. Among overweight men, findings for whole grain bread were sustained in all multivariable-adjusted models ( $P < 0.001$ ), as with MetS criteria. Intake of SSBs among unhealthy obese and overweight men was higher than that of healthy normal-weight men, whereas consumption of fruits among healthy obese and healthy normal-weight men was higher than that of their unhealthy obese and overweight counterparts in age-adjusted but not multivariable-adjusted models ( $P < 0.001$ ) (Table 6 and Supplemental Table 4).

Weight status–metabolic health phenotypes were not associated with food intake in black men in both age-adjusted and multivariable-adjusted models (Supplemental Table 5).

## Discussion

Among obese men in the REGARDS study, the prevalence of MetS-defined healthy obesity was higher in black men (58%) than in their white counterparts (43.9%); the healthy obese phenotype, based on HOMA-IR, was equally prevalent in both black (21%) and white (20%) men. Healthy obesity in white men was associated with food intake in age-adjusted but not fully-adjusted analyses. Healthy overweight was associated with lower intake of whole grain bread in all models.

Data on race-specific prevalence of healthy obesity based on HOMA-IR measures among men in the US are not available. Prevalence of the MetS-defined phenotype among black men in the Howard University Family Study was lower (29%) than that of their counterparts in our study (30). Our findings are, however, higher than those of men in other cohorts based on MetS criteria (24–32%) (31–37). The prevalence of healthy obesity in white men from the REGARDS study, nonetheless, is lower than that of men in studies that compared MetS- and HOMA-IR-defined healthy obesity, i.e., the Uppsala Longitudinal Study of Adult Men (36, 37) and CoLaus (Cohorte Lausannoise) Study (35) (prevalence: 25–32%).

Our findings are consistent with those observed in REGARDS study women in our earlier study (38) in that they show a lack of association between healthy obesity and food intake. Conversely,

high SSB intake (median of 7 SSB servings/wk) relative to non-consumption in the Framingham Offspring/Spouse cohort was associated with a lower prevalence of healthy obesity (7.6% compared with 13.2%), as well as a higher risk of unhealthy obesity (OR: 1.9; 95% CI: 1.1, 3.4) (39). Unlike the REGARDS study cohort, the Framingham Offspring/Spouse cohort was exclusively white. Moreover, the analyses were not sex-specific and other dietary factors were not examined.

As expected, the prevalence of MetS-defined healthy obesity was higher than that based on HOMA-IR in black men from the REGARDS study. Evidence shows that MetS criteria are not ideal when used for black individuals and underdiagnose the syndrome (40). Similarly, use of different MetS definitions and different MetS components, as well as varying cutoffs for obesity and MetS components in the REGARDS study, Howard University Family Study, CoLaus Study, and Uppsala Longitudinal Study of Adult Men, may account for the disparities in prevalences (2, 5, 31). Furthermore, the use of BMI to measure body fatness in black populations may overestimate the prevalence of obesity (41).

The null findings in our study suggest that healthy overweight and obesity may be intermediate transient phenotypes (2, 5). Furthermore, our findings may be attributable in part to single food analysis that fails to take into account interrelationships among dietary factors, as well as biological interactions between diet and other metabolic factors (16, 42, 43). Whole grains and fruits contain dietary fiber, minerals, vitamins, phytochemicals, and other bioactive compounds that have antioxidant and anti-inflammatory activities (44–46). Phytochemicals and related bioactive components, though, are thought to confer the health benefits, and it is suggested that phytochemicals in whole grains and fruits have a synergistic effect when these food items are consumed together (44, 46). Whole grain and fruit consumption likewise tends to be associated with favorable dietary and lifestyle behaviors, including higher fiber and micronutrient intake, higher diet quality, moderate alcohol consumption, dietary supplement use, less smoking, and regular physical activity (47–52). By contrast, red meat, processed meat, and SSB intake are invariably related to a less healthy lifestyle that includes higher alcohol and *trans* fat consumption and lower whole grain, dietary fiber, vegetable, fruit, and fish intake, as well as increased smoking and less exercise (53, 54). This clustering of lifestyle factors makes it difficult to identify the effect of a single food item (53–55); coupled with single food analysis, this may explain the lack of association between whole grains, fruits, fruit juice, meat, and other foods and healthy obesity. The dietary pattern approach, which considers the entire pattern of dietary intake (16, 42, 43), may thus be more suitable for evaluating relations between diet and healthy obesity. Nonetheless, our aim was to examine food intake among weight status–metabolic health phenotypes of men from the REGARDS study first, and then proceed to dietary pattern analysis in future studies. We used select food groups derived from the Block 1998 FFQ in the current study, but more detailed groups were used to create dietary patterns for future studies.

Our study has several strengths, including a national, well-characterized cohort, a large sample size of men of broad age range, a large number of black men, and regional data that includes the Stroke Belt and Stroke Buckle, which are areas with known health disparities. As well, food intake was evaluated with the use of 2 definitions of healthy obesity and models adjusted for many covariates. Other studies examined diet only, but, to our knowledge, our study is unique in evaluating the effects of race and diet on healthy obesity in a large, diverse cohort.

**TABLE 3** Clinical characteristics of men from the REGARDS study according to weight status–metabolic health phenotype defined by metabolic syndrome criteria<sup>1</sup>

	Weight status–metabolic health phenotypes					
	Normal weight		Overweight		Obese	
	Metabolically healthy	Metabolically unhealthy	Metabolically healthy	Metabolically unhealthy	Metabolically healthy	Metabolically unhealthy
<b>White men</b>						
<i>n</i> (%) <sup>2</sup>	933 (93.4)	66 (6.6)	1,432 (79.1)	378 (20.9)	403 (43.9)	514 (56.1)
BMI, kg/m <sup>2</sup>	23.1 ± 0.1 <sup>e</sup>	23.7 ± 0.3 <sup>e</sup>	27.2 ± 0.1 <sup>d</sup>	27.9 ± 0.1 <sup>c</sup>	32.9 ± 0.1 <sup>b</sup>	33.8 ± 0.1 <sup>a</sup>
Waist circumference, cm	87.6 ± 0.3 <sup>e</sup>	93.9 ± 1.0 <sup>d</sup>	96.4 ± 0.2 <sup>d</sup>	101.4 ± 0.4 <sup>c</sup>	108.3 ± 0.4 <sup>b</sup>	112.1 ± 0.4 <sup>a</sup>
Systolic blood pressure, mm Hg	123 ± 1 <sup>c</sup>	132 ± 2 <sup>a</sup>	124 ± 0 <sup>b,c</sup>	131 ± 1 <sup>a</sup>	126 ± 1 <sup>b</sup>	131 ± 1 <sup>a</sup>
Diastolic blood pressure, mm Hg	74.2 ± 0.3 <sup>d</sup>	77.5 ± 1.1 <sup>a,b,c</sup>	76.1 ± 0.2 <sup>c</sup>	79.2 ± 0.5 <sup>a,b</sup>	78.4 ± 0.5 <sup>b</sup>	80.5 ± 0.4 <sup>a</sup>
Hypertension medication	23.2	69.1	27.2	61.5	30.8	60.4
Serum glucose, <sup>3</sup> mmol/L	5.02 ± 0.02 <sup>c</sup>	5.57 ± 0.07 <sup>a</sup>	5.00 ± 0.02 <sup>b</sup>	5.57 ± 0.03 <sup>a</sup>	5.12 ± 0.03 <sup>b</sup>	5.48 ± 0.03 <sup>a</sup>
Serum insulin, <sup>4</sup> pmol/L	45.8 ± 2.1 <sup>d</sup>	104 ± 8 <sup>b</sup>	65.3 ± 2.1 <sup>c</sup>	105 ± 4 <sup>b</sup>	95.1 ± 3.5 <sup>b</sup>	136 ± 3 <sup>a</sup>
Serum HDL cholesterol, <sup>5</sup> mmol/L	1.35 ± 0.01 <sup>a</sup>	0.98 ± 0.04 <sup>d</sup>	1.24 ± 0.01 <sup>b</sup>	0.97 ± 0.02 <sup>d</sup>	1.19 ± 0.02 <sup>c</sup>	0.96 ± 0.02 <sup>d</sup>
Serum TGs, <sup>6</sup> mmol/L	1.15 ± 0.03 <sup>c</sup>	2.42 ± 0.10 <sup>a</sup>	1.33 ± 0.02 <sup>b</sup>	2.23 ± 0.04 <sup>a</sup>	1.37 ± 0.04 <sup>b</sup>	2.20 ± 0.04 <sup>a</sup>
Lipid-lowering medication	22.8	28.6	29.9	29.8	33.1	31.3
Serum high-sensitivity C-reactive protein, <sup>7</sup> mg/L	23.4 ± 1.8 <sup>b</sup>	35.4 ± 7.0 <sup>a,b</sup>	23.8 ± 1.5 <sup>b</sup>	34.9 ± 2.9 <sup>a</sup>	31.8 ± 2.8 <sup>a,b</sup>	35.7 ± 2.5 <sup>a</sup>
<b>MetS components</b>						
Elevated waist circumference (≥102 cm)	0.7	12.3	14.4	49.7	63.8	89.4
Elevated blood pressure (≥130/≥85 mm Hg)	31.2	82.5	34.1	81.5	37.3	75.2
Elevated serum glucose (≥5.6 mmol/L)	13.5	57.9	18.6	59.2	13.0	49.9
Low serum HDL cholesterol (<1.3 mmol/L)	18.0	75.4	22.9	74.2	18.9	74.3
Elevated serum TGs (≥1.7 mmol/L)	12.3	84.2	18.9	73.6	15.8	68.1
<b>Black men</b>						
<i>n</i> (%) <sup>2</sup>	272 (95.4)	13 (4.6)	415 (84.0)	79 (16.0)	204 (58.3)	146 (41.7)
BMI, kg/m <sup>2</sup>	23.0 ± 0.2 <sup>c</sup>	23.5 ± 0.7 <sup>c</sup>	27.4 ± 0.1 <sup>b</sup>	27.8 ± 0.3 <sup>b</sup>	33.8 ± 0.2 <sup>a</sup>	34.4 ± 0.2 <sup>a</sup>
Waist circumference, cm	85.8 ± 0.6 <sup>e</sup>	87.8 ± 2.5 <sup>e</sup>	94.5 ± 0.5 <sup>d</sup>	99.1 ± 1.1 <sup>c</sup>	109 ± 1 <sup>b</sup>	112 ± 1 <sup>a</sup>
Systolic blood pressure, mm Hg	128 ± 1 <sup>b</sup>	133 ± 4 <sup>a,b</sup>	128 ± 1 <sup>b</sup>	135 ± 2 <sup>a</sup>	127 ± 1 <sup>b</sup>	133 ± 1 <sup>a</sup>
Diastolic blood pressure, mm Hg	77.3 ± 0.6 <sup>c</sup>	75.6 ± 2.5 <sup>a,b,c</sup>	78.2 ± 0.5 <sup>b,c</sup>	79.5 ± 1.1 <sup>a,b,c</sup>	80.1 ± 0.7 <sup>a,b</sup>	82.0 ± 0.8 <sup>a</sup>
Hypertension medication	35.8	46.2	47.4	70.8	52.6	76.7
Serum glucose, <sup>3</sup> mmol/L	5.07 ± 0.04 <sup>b</sup>	5.69 ± 0.17 <sup>a</sup>	5.11 ± 0.03 <sup>b</sup>	5.58 ± 0.08 <sup>a</sup>	5.13 ± 0.04 <sup>b</sup>	5.66 ± 0.06 <sup>a</sup>
Serum insulin, <sup>4</sup> pmol/L	54.2 ± 4.2 <sup>d</sup>	113 ± 19 <sup>b</sup>	75.0 ± 3.5 <sup>c</sup>	117 ± 8 <sup>b</sup>	104 ± 5 <sup>b</sup>	154 ± 6 <sup>a</sup>
Serum HDL cholesterol, <sup>5</sup> mmol/L	1.44 ± 0.02 <sup>a</sup>	0.95 ± 0.10 <sup>c</sup>	1.32 ± 0.02 <sup>b</sup>	1.02 ± 0.04 <sup>c</sup>	1.28 ± 0.03 <sup>b</sup>	1.05 ± 0.03 <sup>c</sup>
Serum TGs, <sup>6</sup> mmol/L	1.10 ± 0.04 <sup>d</sup>	2.66 ± 0.18 <sup>a</sup>	1.08 ± 0.04 <sup>d</sup>	1.89 ± 0.08 <sup>b</sup>	1.17 ± 0.05 <sup>d</sup>	1.59 ± 0.06 <sup>c</sup>
Lipid-lowering medication	18.3	30.8	27.1	34.9	24.3	29.3
Serum high-sensitivity C-reactive protein, <sup>7</sup> mg/L	32.8 ± 8.6 <sup>b</sup>	57.2 ± 36.1 <sup>a,b</sup>	30.9 ± 6.9 <sup>b</sup>	96.2 ± 16.0 <sup>a</sup>	43.1 ± 9.8 <sup>b</sup>	47.0 ± 11.7 <sup>a,b</sup>
<b>MetS components</b>						
Elevated waist circumference (≥102 cm)	0.0	0.0	10.1	43.9	60.7	86.3
Elevated blood pressure (≥130/≥85 mm Hg)	48.1	76.9	55.5	90.9	56.7	87.9
Elevated serum glucose (≥5.6 mmol/L)	20.4	69.2	17.4	62.1	15.2	70.2
Low serum HDL cholesterol (<1.3 mmol/L)	10.8	84.6	18.5	71.2	13.5	61.3
Elevated serum TGs (≥1.7 mmol/L)	8.7	76.9	7.8	59.1	9.6	37.9

<sup>1</sup> Values are means ± SEMs or percentages. Variables are unadjusted. ANOVA was used to compute means of continuous variables and to calculate pairwise mean differences in the weight status–metabolic health phenotypes. A chi-square test was used to compute proportions of categorical variables and to calculate pairwise differences in proportions between weight status–metabolic health phenotypes. Labeled means and percentages in a row without a common letter differ, *P* < 0.01. Metabolically healthy normal weight: BMI 18.5 to <25.0 kg/m<sup>2</sup> and <3 MetS components; metabolically healthy overweight: BMI 25 to <30 kg/m<sup>2</sup> and <3 MetS components; metabolically healthy obese: BMI ≥30 kg/m<sup>2</sup> and <3 MetS components; metabolically unhealthy normal weight: BMI 18.5 to <25.0 kg/m<sup>2</sup> and ≥3 MetS components; metabolically unhealthy overweight: BMI 25 to <30 kg/m<sup>2</sup> and ≥3 MetS components; and metabolically unhealthy obese: BMI ≥30 kg/m<sup>2</sup> and ≥3 MetS components. MetS, metabolic syndrome; REGARDS, REasons for Geographic And Racial Differences in Stroke.

<sup>2</sup> Percentage of each weight status–metabolic health phenotype.

<sup>3</sup> To convert glucose to conventional units (mg/dL) divide by 0.0555.

<sup>4</sup> To convert insulin to conventional units (μU/mL) divide by 6.945.

<sup>5</sup> To convert cholesterol to conventional units (mg/dL) divide by 0.0259.

<sup>6</sup> To convert TGs to conventional units (mg/dL) divide by 0.0113.

<sup>7</sup> To convert C-reactive protein to conventional units (mg/dL) divide by 10.

Whereas limitations of the cross-sectional design are well established, because the causal relation cannot be determined, we elected to examine associations between weight status–metabolic health phenotypes and race and food consumption at baseline before conducting longitudinal studies. The Block 1998

FFQ is rather old, but it was one of the major dietary assessment tools for racially diverse populations when the REGARDS study started in 2003 (17, 18). A more culturally specific FFQ designed for Southern black populations was unavailable at the time (56). Therefore, the Block 1998 FFQ may not have adequately

**TABLE 4** Adjusted mean food intakes of white men from the REGARDS study according to weight status–metabolic health phenotype defined by metabolic syndrome criteria<sup>1</sup>

Food group	Weight status–metabolic health phenotypes					
	Normal weight		Overweight		Obese	
	Metabolically healthy	Metabolically unhealthy	Metabolically healthy	Metabolically unhealthy	Metabolically healthy	Metabolically unhealthy
<i>n</i> (%) <sup>2</sup>	933 (93.4)	66 (6.6)	1,432 (79.1)	378 (20.9)	403 (43.9)	514 (56.1)
Vegetables, g/d						
Model 1 <sup>3</sup>	211 ± 5	205 ± 20	202 ± 4	192 ± 9	198 ± 8	191 ± 7
Model 2 <sup>4</sup>	228 ± 11	246 ± 20	221 ± 10	222 ± 12	208 ± 13	206 ± 13
Fruits, g/d						
Model 1 <sup>3</sup>	127 ± 4 <sup>a</sup>	75 ± 15 <sup>c</sup>	115 ± 3 <sup>a,b</sup>	100 ± 6 <sup>b,c</sup>	111 ± 6 <sup>a,b,c</sup>	105 ± 5 <sup>b,c</sup>
Model 2 <sup>4</sup>	109 ± 9	71 ± 16	111 ± 8	108 ± 9	124 ± 11	123 ± 11
Whole grain bread, g/d						
Model 1 <sup>3</sup>	20.6 ± 0.7 <sup>a</sup>	14.6 ± 2.7 <sup>b</sup>	15.9 ± 0.6 <sup>b</sup>	13.3 ± 1.2 <sup>b</sup>	16.2 ± 1.1 <sup>b</sup>	15.1 ± 1.0 <sup>b</sup>
Model 2 <sup>4</sup>	17.2 ± 1.8 <sup>a</sup>	12.6 ± 3.1 <sup>b</sup>	13.8 ± 1.6 <sup>b</sup>	12.5 ± 1.8 <sup>b</sup>	15.5 ± 2.0 <sup>a,b</sup>	14.7 ± 2.0 <sup>a,b</sup>
Refined grains, g/d						
Model 1 <sup>3</sup>	25.4 ± 1.0	26.6 ± 3.9	23.7 ± 0.8	24.7 ± 1.7	26.5 ± 1.6	25.1 ± 1.4
Model 2 <sup>4</sup>	29.2 ± 2.5	32.3 ± 4.4	27.8 ± 2.3	29.3 ± 2.6	29.7 ± 2.9	28.4 ± 2.9
Beans, g/d						
Model 1 <sup>3</sup>	16.4 ± 0.8	13.2 ± 3.0	14.0 ± 0.6	13.2 ± 1.3	14.2 ± 1.2	14.2 ± 1.1
Model 2 <sup>4</sup>	20.2 ± 1.9	15.9 ± 3.3	19.7 ± 1.7	18.9 ± 1.9	20.7 ± 2.2	21.0 ± 2.2
Fish, g/d						
Model 1 <sup>3</sup>	20.4 ± 0.9	19.4 ± 3.3	21.2 ± 0.7	19.5 ± 1.4	22.0 ± 1.3	22.1 ± 1.2
Model 2 <sup>4</sup>	18.7 ± 2.0	20.2 ± 3.5	18.2 ± 1.8	17.9 ± 2.1	17.0 ± 2.3	19.3 ± 2.3
Poultry, g/d						
Model 1 <sup>3</sup>	12.4 ± 0.6	8.1 ± 2.4	13.6 ± 0.5	11.5 ± 1.0	14.7 ± 1.0	11.5 ± 0.9
Model 2 <sup>4</sup>	8.7 ± 1.5	5.5 ± 2.7	10.6 ± 1.3	10.0 ± 1.6	13.1 ± 1.7	10.2 ± 1.7
Red meat, g/d						
Model 1 <sup>3</sup>	35.6 ± 1.4 <sup>d</sup>	48.4 ± 5.2 <sup>a,b,c</sup>	42.2 ± 1.1 <sup>c</sup>	45.1 ± 2.2 <sup>b,c</sup>	52.3 ± 2.1 <sup>a,b</sup>	53.1 ± 1.9 <sup>a</sup>
Model 2 <sup>4</sup>	50.3 ± 3.1	53.6 ± 5.5	48.6 ± 2.8	49.5 ± 3.2	48.1 ± 3.6	49.4 ± 3.6
Processed meat, g/d						
Model 1 <sup>3</sup>	17.7 ± 0.7 <sup>c</sup>	22.0 ± 2.5 <sup>a,b,c</sup>	20.8 ± 0.5 <sup>c</sup>	21.1 ± 1.1 <sup>b,c</sup>	24.3 ± 1.0 <sup>a</sup>	23.9 ± 0.9 <sup>a</sup>
Model 2 <sup>4</sup>	19.5 ± 1.5	20.0 ± 2.7	20.4 ± 1.4	19.4 ± 1.6	19.4 ± 1.8	18.6 ± 1.8
Fried foods, g/d						
Model 1 <sup>3</sup>	19.2 ± 0.9 <sup>c</sup>	24.9 ± 3.5 <sup>a,b</sup>	24.3 ± 0.7 <sup>b</sup>	27.4 ± 1.5 <sup>a,b</sup>	30.0 ± 1.4 <sup>a</sup>	29.7 ± 1.2 <sup>a</sup>
Model 2 <sup>4</sup>	22.7 ± 2.1	22.4 ± 3.6	23.9 ± 1.8	24.5 ± 2.1	24.4 ± 2.4	21.7 ± 2.4
Low-fat dairy, g/d						
Model 1 <sup>3</sup>	113 ± 7	92 ± 26	116 ± 5	85 ± 11	98 ± 10	97 ± 9
Model 2 <sup>4</sup>	96 ± 16	113 ± 28	94 ± 14	82 ± 16	65 ± 18	79 ± 18
High-fat dairy, g/d						
Model 1 <sup>3</sup>	122 ± 6	154 ± 22	111 ± 5	129 ± 10	113 ± 9	140 ± 8
Model 2 <sup>4</sup>	147 ± 14	159 ± 25	133 ± 13	134 ± 15	118 ± 16	137 ± 16
100% fruit juice, g/d						
Model 1 <sup>3</sup>	153 ± 6	156 ± 21	140 ± 5	128 ± 9	122 ± 9	129 ± 8
Model 2 <sup>4</sup>	128 ± 14	146 ± 25	123 ± 13	122 ± 15	116 ± 16	129 ± 16
Sugar-sweetened beverages, g/d						
Model 1 <sup>3</sup>	152 ± 9 <sup>c</sup>	268 ± 35 <sup>a</sup>	167 ± 7 <sup>b,c</sup>	205 ± 15 <sup>a,b</sup>	201 ± 14 <sup>a,b</sup>	230 ± 12 <sup>a</sup>
Model 2 <sup>4</sup>	167 ± 23	255 ± 40	150 ± 20	162 ± 24	141 ± 26	162 ± 26

<sup>1</sup> Values are means ± SEMs. Linear regression was used to compute age-adjusted and multivariable-adjusted least-squares means of food intake and to calculate pairwise mean differences in the weight status–metabolic health phenotypes. Labeled means in a row without a common letter differ,  $P < 0.01$ . Metabolically healthy normal weight: BMI 18.5 to  $<25.0$  kg/m<sup>2</sup> and  $<3$  MetS components; metabolically healthy overweight: BMI 25 to  $<30$  kg/m<sup>2</sup> and  $<3$  MetS components; metabolically healthy obese: BMI  $\geq 30$  kg/m<sup>2</sup> and  $<3$  MetS components; metabolically unhealthy normal weight: BMI 18.5 to  $<25.0$  kg/m<sup>2</sup> and  $\geq 3$  MetS components; metabolically unhealthy overweight: BMI 25 to  $<30$  kg/m<sup>2</sup> and  $\geq 3$  MetS components; and metabolically unhealthy obese: BMI  $\geq 30$  kg/m<sup>2</sup> and  $\geq 3$  MetS components. MetS, metabolic syndrome; REGARDS, REasons for Geographic And Racial Differences in Stroke.

<sup>2</sup> Percentage of each weight status–metabolic health phenotype.

<sup>3</sup> Adjusted for baseline age.

<sup>4</sup> Adjusted for baseline age, BMI, marital status (married or other), residential region (Stroke Belt or other), education ( $<$ college degree or  $\geq$ college degree), annual income ( $<$ \$35,000 or  $\geq$ \$35,000), alcohol intake (none, moderate, or heavy), multivitamin use (yes or no), cigarette smoking status (nonsmoker or current smoker), physical activity (0 times/wk, 1–3 times/wk, or  $\geq 4$  times/wk), television viewing (0 h/wk, 1–6 h/wk, or  $\geq 1$  h/d), high-sensitivity C-reactive protein, and food intake (vegetables, fruits, whole grain bread, refined grains, beans, fish, poultry, red meat, processed meat, fried foods, low-fat dairy, high-fat dairy, 100% fruit juice, and sugar-sweetened beverages; each food item was adjusted for all other food intake).

**TABLE 5** Clinical characteristics of men from the REGARDS study according to weight status–metabolic health phenotype defined by HOMA-IR criteria<sup>1</sup>

	Weight status–metabolic health phenotypes					
	Normal weight		Overweight		Obese	
	Metabolically healthy	Metabolically unhealthy	Metabolically healthy	Metabolically unhealthy	Metabolically healthy	Metabolically unhealthy
<b>White men</b>						
<i>n</i> (%) <sup>2</sup>	801 (80.2)	198 (19.8)	940 (51.9)	870 (48.1)	186 (20.3)	731 (79.7)
BMI, kg/m <sup>2</sup>	23.0 ± 0.1 <sup>f</sup>	23.6 ± 0.1 <sup>e</sup>	27.0 ± 0.1 <sup>d</sup>	27.6 ± 0.1 <sup>c</sup>	32.1 ± 0.2 <sup>b</sup>	33.7 ± 0.1 <sup>a</sup>
Waist circumference, cm	87.3 ± 0.3 <sup>f</sup>	90.8 ± 0.6 <sup>e</sup>	96.0 ± 0.3 <sup>d</sup>	99.0 ± 0.3 <sup>c</sup>	107.2 ± 0.6 <sup>b</sup>	111.2 ± 0.3 <sup>a</sup>
Systolic blood pressure, mm Hg	123 ± 1 <sup>c</sup>	125 ± 1 <sup>b,c</sup>	125 ± 1 <sup>b</sup>	126 ± 1 <sup>b</sup>	128 ± 1 <sup>a,b</sup>	129 ± 1 <sup>a</sup>
Diastolic blood pressure, mm Hg	74.3 ± 0.3 <sup>d</sup>	75.0 ± 0.6 <sup>c,d</sup>	76.5 ± 0.3 <sup>b,c</sup>	77.1 ± 0.3 <sup>b</sup>	79.4 ± 0.7 <sup>a</sup>	79.6 ± 0.3 <sup>a</sup>
Hypertension medication	23.3	38.1	30.4	38.1	40.4	49.5
Serum glucose, <sup>3</sup> mmol/L	5.00 ± 0.02 <sup>b</sup>	5.46 ± 0.04 <sup>a</sup>	5.01 ± 0.02 <sup>b</sup>	5.41 ± 0.02 <sup>a</sup>	4.93 ± 0.04 <sup>b</sup>	5.42 ± 0.02 <sup>a</sup>
Serum insulin, <sup>4</sup> pmol/L	35.4 ± 2.1 <sup>d</sup>	107 ± 4 <sup>b</sup>	43.1 ± 2.1 <sup>c</sup>	105 ± 2 <sup>b</sup>	47.2 ± 4.2 <sup>c,d</sup>	136 ± 2 <sup>a</sup>
Serum HDL cholesterol, <sup>5</sup> mmol/L	1.38 ± 0.01 <sup>a</sup>	1.13 ± 0.02 <sup>c</sup>	1.26 ± 0.01 <sup>b</sup>	1.11 ± 0.01 <sup>c</sup>	1.15 ± 0.02 <sup>c</sup>	1.04 ± 0.01 <sup>d</sup>
Serum TGs, <sup>6</sup> mmol/L	1.13 ± 0.03 <sup>d</sup>	1.63 ± 0.06 <sup>b</sup>	1.31 ± 0.03 <sup>c</sup>	1.72 ± 0.03 <sup>b</sup>	1.58 ± 0.07 <sup>b</sup>	1.90 ± 0.03 <sup>a</sup>
Lipid-lowering medication	21.7	29.3	28.0	31.9	28.1	33.1
Serum high sensitivity C-reactive protein, <sup>7</sup> mg/L	24.5 ± 1.9 <sup>b</sup>	22.7 ± 3.9 <sup>b</sup>	23.4 ± 1.8 <sup>b</sup>	28.8 ± 1.9 <sup>a,b</sup>	29.9 ± 4.1 <sup>a,b</sup>	35.1 ± 2.1 <sup>a</sup>
<b>HOMA-IR measures</b>						
HOMA-IR index	11.2 ± 0.8 <sup>c</sup>	38.5 ± 1.7 <sup>b</sup>	13.8 ± 0.8 <sup>c</sup>	36.8 ± 0.8 <sup>b</sup>	14.9 ± 1.7 <sup>c</sup>	47.8 ± 0.9 <sup>a</sup>
Insulin-resistant (HOMA-IR quartile 4)	0.0	100.0	0.0	100.0	0.0	100.0
<b>Black men</b>						
<i>n</i> (%) <sup>2</sup>	213 (74.7)	72 (25.3)	231 (46.8)	263 (53.2)	75 (21.4)	275 (78.6)
BMI, kg/m <sup>2</sup>	22.9 ± 0.2 <sup>e</sup>	23.4 ± 0.3 <sup>e</sup>	27.1 ± 0.2 <sup>d</sup>	27.8 ± 0.2 <sup>c</sup>	32.6 ± 0.3 <sup>b</sup>	34.5 ± 0.2 <sup>a</sup>
Waist circumference, cm	85.0 ± 0.7 <sup>f</sup>	88.5 ± 1.1 <sup>e</sup>	93.2 ± 0.6 <sup>d</sup>	97.1 ± 0.6 <sup>c</sup>	107.6 ± 1.1 <sup>b</sup>	111.0 ± 0.6 <sup>a</sup>
Systolic blood pressure, mm Hg	127 ± 1	130 ± 2	128 ± 1	130 ± 1	128 ± 2	131 ± 1
Diastolic blood pressure, mm Hg	76.9 ± 0.7 <sup>b</sup>	78.2 ± 1.1 <sup>b</sup>	78.2 ± 0.6 <sup>b</sup>	78.7 ± 0.6 <sup>b</sup>	79.3 ± 1.1 <sup>a,b</sup>	81.3 ± 0.6 <sup>a</sup>
Hypertension medication	37.5	33.3	44.3	57.5	50.0	66.1
Serum glucose, <sup>3</sup> mmol/L	4.95 ± 0.04 <sup>b</sup>	5.53 ± 0.08 <sup>a</sup>	4.89 ± 0.04 <sup>b</sup>	5.44 ± 0.04 <sup>a</sup>	4.88 ± 0.07 <sup>b</sup>	5.47 ± 0.03 <sup>a</sup>
Serum insulin, <sup>4</sup> pmol/L	37.5 ± 4.2 <sup>c</sup>	115.3 ± 7.6 <sup>b</sup>	42.4 ± 4.2 <sup>c</sup>	117.4 ± 4.2 <sup>b</sup>	46.5 ± 7.6 <sup>c</sup>	145.8 ± 4.2 <sup>a</sup>
Serum HDL cholesterol, <sup>5</sup> mmol/L	1.46 ± 0.03 <sup>a</sup>	1.29 ± 0.04 <sup>b,c</sup>	1.32 ± 0.03 <sup>b</sup>	1.22 ± 0.02 <sup>c</sup>	1.26 ± 0.04 <sup>b,c</sup>	1.16 ± 0.02 <sup>c</sup>
Serum TGs, <sup>6</sup> mmol/L	1.10 ± 0.05 <sup>b</sup>	1.41 ± 0.09 <sup>a</sup>	1.08 ± 0.05 <sup>b</sup>	1.32 ± 0.05 <sup>a,b</sup>	1.09 ± 0.09 <sup>b</sup>	1.41 ± 0.05 <sup>a</sup>
Lipid-lowering medication	18.2	21.0	18.0	38.0	16.9	28.9
Serum high sensitivity C-reactive protein, <sup>7</sup> mg/L	31.9 ± 9.7	40.6 ± 16.6	32.2 ± 9.3	49.5 ± 8.9	37.5 ± 16.5	46.6 ± 8.5
<b>HOMA-IR measures</b>						
HOMA-IR index	11.8 ± 1.7 <sup>c</sup>	41.5 ± 3.0 <sup>b</sup>	13.4 ± 1.7 <sup>c</sup>	41.3 ± 1.6 <sup>b</sup>	14.5 ± 2.9 <sup>c</sup>	51.8 ± 1.5 <sup>a</sup>
Insulin-resistant (HOMA-IR quartile 4)	0.0	100.0	0.0	100.0	0.0	100.0

<sup>1</sup> Values are means ± SEMs or percentages. Variables are unadjusted. ANOVA was used to compute means of continuous variables and to calculate pairwise mean differences in the weight status–metabolic health phenotypes. A chi-square test was used to compute proportions of categorical variables and to calculate pairwise differences in proportions between weight status–metabolic health phenotypes. Labeled means and percentages in a row without a common letter differ,  $P < 0.01$ . Metabolically healthy normal weight: BMI 18.5 to <25.0 kg/m<sup>2</sup> and <3 MetS components; metabolically healthy overweight: BMI 25 to <30 kg/m<sup>2</sup> and <3 MetS components; metabolically healthy obese: BMI ≥30 kg/m<sup>2</sup> and <3 MetS components; metabolically unhealthy normal weight: BMI 18.5 to <25.0 kg/m<sup>2</sup> and ≥3 MetS components; metabolically unhealthy overweight: BMI 25 to <30 kg/m<sup>2</sup> and ≥3 MetS components; and metabolically unhealthy obese: BMI ≥30 kg/m<sup>2</sup> and ≥3 MetS components. MetS, metabolic syndrome; REGARDS, REasons for Geographic And Racial Differences in Stroke.

<sup>2</sup> Percentage of each weight status–metabolic health phenotype.

<sup>3</sup> To convert glucose to conventional units (mg/dL) divide by 0.0555.

<sup>4</sup> To convert insulin to conventional units (μU/mL) divide by 6.945.

<sup>5</sup> To convert cholesterol to conventional units (mg/dL) divide by 0.0259.

<sup>6</sup> To convert TGs to conventional units (mg/dL) divide by 0.0113.

<sup>7</sup> To convert C-reactive protein to conventional units (mg/dL) divide by 10.

discriminated between dietary intake among white and black adults, possibly attenuating true differences in dietary intake. Moreover, dietary self-report errors (57) and response bias may have affected our findings.

Adults from the Stroke Belt were oversampled; thus, most men who completed an FFQ (52%) were from this region. Moreover, FFQ completers were older, more likely to be married, had a higher educational level and socioeconomic status, and exhibited healthier lifestyle and metabolic profiles; these characteristics are consistent with study participation (58–60). The “healthy cohort” effect may thus somewhat limit the general-

izability of findings. Likewise, the study sample was relatively healthy, which might limit generalizability of the results. Lastly, HOMA-IR results need to be interpreted with caution, because insulin measures are not standardized (61).

In conclusion, healthy obesity was present in about one-quarter to one-half of obese men from the REGARDS study, and it was not associated with food intake. Healthy overweight was similarly not associated with food intake. Future studies need to consider the dietary pattern approach, which may better inform the holistic effect of diet on healthy obesity, in prospective models.



**TABLE 6** Adjusted mean food intakes of white men from the REGARDS study according to weight status–metabolic health phenotype defined by HOMA-IR criteria<sup>1</sup>

Food group	Weight status–metabolic health phenotypes					
	Normal weight		Overweight		Obese	
	Metabolically healthy	Metabolically unhealthy	Metabolically healthy	Metabolically unhealthy	Metabolically healthy	Metabolically unhealthy
<i>n</i> (%) <sup>2</sup>	801 (80.2)	198 (19.8)	940 (51.9)	870 (48.1)	186 (20.3)	731 (79.7)
Vegetables, g/d						
Model 1 <sup>3</sup>	215 ± 6	193 ± 11	202 ± 5	199 ± 6	201 ± 12	192 ± 6
Model 2 <sup>4</sup>	230 ± 12	229 ± 15	222 ± 11	221 ± 11	207 ± 15	206 ± 13
Fruits, g/d						
Model 1 <sup>3</sup>	128 ± 4 <sup>a</sup>	105 ± 9 <sup>b</sup>	114 ± 4 <sup>a,b</sup>	110 ± 4 <sup>b</sup>	128 ± 9 <sup>a</sup>	102 ± 5 <sup>b</sup>
Model 2 <sup>4</sup>	108 ± 9	99 ± 12	109 ± 8	110 ± 9	135 ± 12	118 ± 10
Whole grain bread, g/d						
Model 1 <sup>3</sup>	21.2 ± 0.8 <sup>a</sup>	16.2 ± 1.5 <sup>b</sup>	16.4 ± 0.7 <sup>b</sup>	14.3 ± 0.7 <sup>b</sup>	16.2 ± 1.6 <sup>b</sup>	15.4 ± 0.8 <sup>b</sup>
Model 2 <sup>4</sup>	17.5 ± 1.8 <sup>a</sup>	13.7 ± 2.2 <sup>b</sup>	13.8 ± 1.6 <sup>b</sup>	12.7 ± 1.6 <sup>b</sup>	14.9 ± 2.3 <sup>a,b</sup>	14.7 ± 2.0 <sup>a,b</sup>
Refined grains, g/d						
Model 1 <sup>3</sup>	26.3 ± 1.1	22.0 ± 2.2	23.8 ± 1.0	24.0 ± 1.1	27.3 ± 2.3	25.3 ± 1.2
Model 2 <sup>4</sup>	30.3 ± 2.5	27.1 ± 3.2	28.0 ± 2.3	27.9 ± 2.3	31.0 ± 3.2	27.7 ± 2.9
Beans, g/d						
Model 1 <sup>3</sup>	17.1 ± 0.9	12.4 ± 1.7	13.8 ± 0.8	13.8 ± 0.8	12.8 ± 1.8	14.6 ± 0.9
Model 2 <sup>4</sup>	20.1 ± 1.9	17.6 ± 2.4	19.5 ± 1.7	19.3 ± 1.8	18.9 ± 2.4	21.5 ± 2.1
Fish, g/d						
Model 1 <sup>3</sup>	20.4 ± 0.9	20.2 ± 1.9	20.7 ± 0.9	21.1 ± 0.9	22.0 ± 1.9	22.1 ± 1.0
Model 2 <sup>4</sup>	18.6 ± 2.0	20.0 ± 2.5	18.0 ± 1.8	18.6 ± 1.9	17.5 ± 2.6	18.6 ± 2.3
Poultry, g/d						
Model 1 <sup>3</sup>	12.5 ± 0.7	10.4 ± 1.4	13.6 ± 0.6	12.7 ± 0.7	15.2 ± 1.4	12.3 ± 0.7
Model 2 <sup>4</sup>	8.6 ± 1.5	7.4 ± 1.9	10.7 ± 1.4	10.0 ± 1.4	13.0 ± 1.9	10.9 ± 1.7
Red meat, g/d						
Model 1 <sup>3</sup>	36.0 ± 1.5 <sup>c</sup>	38.3 ± 3.0 <sup>b,c</sup>	42.4 ± 1.4 <sup>b</sup>	43.2 ± 1.4 <sup>a,b</sup>	51.5 ± 3.1 <sup>a</sup>	53.1 ± 1.6 <sup>a</sup>
Model 2 <sup>4</sup>	51.1 ± 3.2	50.1 ± 4.0	49.3 ± 2.9	48.1 ± 2.9	49.9 ± 4.0	47.8 ± 3.5
Processed meat, g/d						
Model 1 <sup>3</sup>	17.5 ± 0.7 <sup>c</sup>	19.6 ± 1.4 <sup>b,c</sup>	20.4 ± 0.7 <sup>b</sup>	21.3 ± 0.7 <sup>b</sup>	22.3 ± 1.5 <sup>a,b</sup>	24.5 ± 0.7 <sup>a</sup>
Model 2 <sup>4</sup>	19.1 ± 1.6	20.5 ± 2.0	20.1 ± 1.4	20.4 ± 1.4	18.3 ± 2.0	19.7 ± 1.7
Fried foods, g/d						
Model 1 <sup>3</sup>	18.8 ± 1.0 <sup>c</sup>	22.7 ± 2.0 <sup>b,c</sup>	23.6 ± 0.9 <sup>b</sup>	26.4 ± 0.9 <sup>b</sup>	26.5 ± 2.0 <sup>a,b</sup>	30.8 ± 1.0 <sup>a</sup>
Model 2 <sup>4</sup>	22.2 ± 2.1	23.6 ± 2.6	23.5 ± 1.9	24.7 ± 1.9	22.3 ± 2.7	23.7 ± 2.3
Low-fat dairy, g/d						
Model 1 <sup>3</sup>	115 ± 7	99 ± 15	118 ± 7	101 ± 7	106 ± 15	95 ± 8
Model 2 <sup>4</sup>	96 ± 16	102 ± 20	98 ± 15	85 ± 15	66 ± 20	74 ± 18
High-fat dairy, g/d						
Model 1 <sup>3</sup>	119 ± 6	144 ± 13	115 ± 6	115 ± 6	101 ± 13	135 ± 7
Model 2 <sup>4</sup>	146 ± 14	162 ± 18	138 ± 13	131 ± 13	111 ± 18	136 ± 16
100% fruit juice, g/d						
Model 1 <sup>3</sup>	155 ± 6	147 ± 12	138 ± 6	138 ± 6	120 ± 12	127 ± 6
Model 2 <sup>4</sup>	129 ± 14	129 ± 18	120 ± 13	126 ± 13	112 ± 18	128 ± 16
Sugar-sweetened beverages, g/d						
Model 1 <sup>3</sup>	152 ± 10 <sup>b</sup>	188 ± 20 <sup>a,b</sup>	157 ± 9 <sup>b</sup>	193 ± 10 <sup>a</sup>	189 ± 21 <sup>a,b</sup>	225 ± 11 <sup>a</sup>
Model 2 <sup>4</sup>	172 ± 23	179 ± 29	144 ± 21	166 ± 21	145 ± 29	158 ± 26

<sup>1</sup> Values are means ± SEMs. Linear regression was used to compute age-adjusted and multivariable-adjusted least-squares means of food intake and to calculate pairwise mean differences in the weight status–metabolic health phenotypes. Labeled means in a row without a common letter differ,  $P < 0.01$ . Metabolically healthy normal weight: BMI 18.5 to <25.0 kg/m<sup>2</sup> and <3 MetS components; metabolically healthy overweight: BMI 25 to <30 kg/m<sup>2</sup> and <3 MetS components; metabolically healthy obese: BMI ≥30 kg/m<sup>2</sup> and <3 MetS components; metabolically unhealthy normal weight: BMI 18.5 to <25.0 kg/m<sup>2</sup> and ≥3 MetS components; metabolically unhealthy overweight: BMI 25 to <30 kg/m<sup>2</sup> and ≥3 MetS components; and metabolically unhealthy obese: BMI ≥30 kg/m<sup>2</sup> and ≥3 MetS components. MetS, metabolic syndrome; REGARDS, REasons for Geographic And Racial Differences in Stroke.

<sup>2</sup> Percentage of each weight status–metabolic health phenotype.

<sup>3</sup> Adjusted for baseline age.

<sup>4</sup> Adjusted for baseline age, BMI, marital status (married or other), residential region (Stroke Belt or other), education (<college degree or ≥college degree), annual income (<\$35,000 or ≥\$35,000), alcohol intake (none, moderate, or heavy), multivitamin use (yes or no), cigarette smoking status (nonsmoker or current smoker), physical activity (0 times/wk, 1–3 times/wk, or ≥4 times/wk), television viewing (0 h/wk, 1–6 h/wk, or ≥1 h/d), high-sensitivity C-reactive protein, and food intake (vegetables, fruits, whole grain bread, refined grains, beans, fish, poultry, red meat, processed meat, fried foods, low-fat dairy, high-fat dairy, 100% fruit juice, and sugar-sweetened beverages; each food item was adjusted for all other food intake).

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## References

1. Wildman RP, Muntner P, Reynolds K, McGinn AP, Rajpathak S, Wylie-Rosett J, Sowers MR. The obese without cardiometabolic risk factor clustering and the normal weight with cardiometabolic risk factor clustering: prevalence and correlates of 2 phenotypes among the US population (NHANES 1999–2004). *Arch Intern Med* 2008;168:1617–24.
2. Stefan N, Häring HU, Hu FB, Schulze MB. Metabolically healthy obesity: epidemiology, mechanisms, and clinical implications. *Lancet Diabetes Endocrinol* 2013;1:152–62.
3. Primeau V, Coderre L, Karelis AD, Brochu M, Lavoie ME, Messier V, Sladek R, Rabasa-Lhoret R. Characterizing the profile of obese patients who are metabolically healthy. *Int J Obes (Lond)* 2011;35:971–81.
4. Blüher M. Are there still healthy obese patients? *Curr Opin Endocrinol Diabetes Obes* 2012;19:341–6.
5. Phillips CM. Metabolically healthy obesity: definitions, determinants and clinical implications. *Rev Endocr Metab Disord* 2013;14:219–27.
6. Ortega FB, Lee DC, Katzmarzyk PT, Ruiz JR, Sui X, Church TS, Blair SN. The intriguing metabolically healthy but obese phenotype: cardiovascular prognosis and role of fitness. *Eur Heart J* 2013;34:389–97.
7. Ogorodnikova AD, Kim M, McGinn AP, Muntner P, Khan U, Wildman RP. Incident cardiovascular disease events in metabolically benign obese individuals. *Obesity (Silver Spring)* 2012;20:651–9.
8. Alberti KG, Eckel RH, Grundy SM, Zimmet PZ, Cleeman JJ, Donato KA, Fruchart JC, James WP, Loria CM, Smith SC, Jr, et al. Harmonizing the metabolic syndrome: a joint interim statement of the International Diabetes Federation Task Force on Epidemiology and Prevention; National Heart, Lung, and Blood Institute; American Heart Association; World Heart Federation; International Atherosclerosis Society; and International Association for the Study of Obesity. *Circulation* 2009;120:1640–5.
9. Hankinson AL, Daviglus ML, Van Horn L, Chan Q, Brown I, Holmes E, Elliott P, Stamler J. Diet composition and activity level of at risk and metabolically healthy obese American adults. *Obesity (Silver Spring)* 2013;21:637–43.
10. Lee K. Metabolically obese but normal weight (MONW) and metabolically healthy but obese (MHO) phenotypes in Koreans: characteristics and health behaviors. *Asia Pac J Clin Nutr* 2009;18:280–4.
11. Manu P, Ionescu-Tirgoviste C, Tsang J, Napolitano BA, Lesser ML, Correll CU. Dysmetabolic signals in “metabolically healthy” obesity. *Obes Res Clin Pract* 2012;6:e9–20.
12. Wizemann TM, Pardue ML. Editors Committee on Understanding the Biology of Sex and Gender Differences, Board on Health Sciences Policy, Institute of Medicine (2001). Exploring the biological contributions to human health: Does sex matter? Washington (DC): National Academy Press [Internet]. [cited 2015 Jan 9.] Available from: <http://www.nap.edu/catalog/10028.html>.
13. Lovejoy JC, Sainsbury A; Stock Conference 2008 Working Group. Sex differences in obesity and the regulation of energy homeostasis. *Obes Rev* 2009;10:154–67.
14. Kimokoti RW, Newby PK, Gona P, Zhu L, McKeon-O'Malley C, Pablo Guzman J, D'Agostino RB, Millen BE. Patterns of weight change and progression to overweight and obesity differ in men and women: implications for research and interventions. *Public Health Nutr* 2013;16:1463–75.
15. Magkos F, Wang X, Mittendorfer B. Metabolic actions of insulin in men and women. *Nutrition* 2010;26:686–93.
16. Newby PK, Tucker KL. Empirically derived eating patterns using factor or cluster analysis: a review. *Nutr Rev* 2004;62:177–203.
17. Newby PK, Noel SE, Grant R, Judd S, Shikany JM, Ard J. Race and region are associated with nutrient intakes among black and white men in the United States. *J Nutr* 2011;141:296–303.
18. Newby PK, Noel SE, Grant R, Judd S, Shikany JM, Ard J. Race and region have independent and synergistic effects on dietary intakes in black and white women. *Nutr J* 2012;11:25.
19. Howard VJ, Cushman M, Pulley L, Gomez CR, Go RC, Prineas RJ, Graham A, Moy CS, Howard G. The reasons for geographic and racial differences in stroke study: objectives and design. *Neuroepidemiology* 2005;25:135–43.
20. Levine DA, Calhoun DA, Prineas RJ, Cushman M, Howard VJ, Howard G. Moderate waist circumference and hypertension prevalence: the REGARDS Study. *Am J Hypertens* 2011;24:482–8.
21. Lakoski SG, Le AH, Muntner P, Judd SE, Safford MM, Levine DA, Howard G, Cushman M. Adiposity, inflammation, and risk for death in black and white men and women in the United States: the Reasons for Geographic and Racial Differences in Stroke (REGARDS) study. *J Clin Endocrinol Metab* 2011;96:1805–14.
22. Jensen MD, Ryan DH, Apovian CM, Ard JD, Comuzzie AG, Donato KA, Hu FB, Hubbard VS, Jakicic JM, Kushner RE, et al. 2013. AHA/ACC/TOS Guideline for the management of overweight and obesity in adults: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and The Obesity Society. *Circulation* 2014; 129(25 Suppl 2):S102–38.
23. Balkau B, Charles MA. Comment on the provisional report from the WHO consultation. European Group for the Study of Insulin Resistance (EGIR). *Diabet Med* 1999;16:442–3.
24. Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC. Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. *Diabetologia* 1985;28:412–9.
25. Subar AF, Thompson FE, Kipnis V, Midthune D, Hurwitz P, McNutt S, McIntosh A, Rosenfeld S. Comparative validation of the Block, Willett, and National Cancer Institute food frequency questionnaires: the Eating at America's Table Study. *Am J Epidemiol* 2001;154:1089–99.
26. Centers for Disease Control and Prevention. Alcohol and public health [Internet]. [cited 2015 Feb 10.] Available from: <http://www.cdc.gov/alcohol/faqs.htm#standDrink>.
27. NIH. National Institute on Alcohol Abuse and Alcoholism (NIAAA) [Internet]. [cited 2015 Feb 10.] Available from: <http://www.niaaa.nih.gov/alcohol-health>.
28. Lomax RG. An introduction to statistical concepts. Mahwah, NJ: Lawrence Erlbaum Associates, Publishers, 2007.
29. Statistical Applications Software, SAS Institute, Inc. Cary, NC: Version 9.1.
30. Doumatey AP, Bentley AR, Zhou J, Huang H, Adeyemo A, Rotimi CN. Paradoxical hyperadiponectinemia is associated with the metabolically healthy obese (MHO) phenotype in African Americans. *J Endocrinol Metab* 2012;2:51–65.

31. Rey-López JP, de Rezende LF, Pastor-Valero M, Tess BH. The prevalence of metabolically healthy obesity: a systematic review and critical evaluation of the definitions used. *Obes Rev* 2014;15:781–90.
32. Hwang LC, Bai CH, Sun CA, Chen CJ. Prevalence of metabolically healthy obesity and its impacts on incidences of hypertension, diabetes and the metabolic syndrome in Taiwan. *Asia Pac J Clin Nutr* 2012;21:227–33.
33. St-Pierre AC, Cantin B, Mauriège P, Bergeron J, Dagenais GR, Després JP, Lamarche B. Insulin resistance syndrome, body mass index and the risk of ischemic heart disease. *CMAJ* 2005;172:1301–5.
34. Hadaegh F, Bozorgmanesh M, Safarkhani M, Khalili D, Azizi F. Predictability of body mass index for diabetes: affected by the presence of metabolic syndrome? *BMC Public Health* 2011;11:383.
35. Velho S, Paccaud F, Waeber G, Vollenweider P, Marques-Vidal P. Metabolically healthy obesity: different prevalences using different criteria. *Eur J Clin Nutr* 2010;64:1043–51.
36. Arnlöv J, Ingelsson E, Sundström J, Lind L. Impact of body mass index and the metabolic syndrome on the risk of cardiovascular disease and death in middle-aged men. *Circulation* 2010;121:230–6.
37. Arnlöv J, Sundström J, Ingelsson E, Lind L. Impact of BMI and the metabolic syndrome on the risk of diabetes in middle-aged men. *Diabetes Care* 2011;34:61–5.
38. Kimokoti RW, Judd SE, Shikany JM, Newby PK. Food intake does not differ between obese women who are metabolically healthy or abnormal. *J Nutr*. 2014;144:2018–26. *J Nutr* 2014;144:2018–26.
39. Green AK, Jacques PF, Rogers G, Fox CS, Meigs JB, McKeown NM. Sugar-sweetened beverages and prevalence of the metabolically abnormal phenotype in the Framingham Heart Study. *Obesity (Silver Spring)* 2014;22:E157–63.
40. Yu SS, Castillo DC, Courville AB, Sumner AE. The triglyceride paradox in people of African descent. *Metab Syndr Relat Disord* 2012;10:77–82.
41. Wagner DR, Heyward VH. Measures of body composition in blacks and whites: a comparative review. *Am J Clin Nutr* 2000;71:1392–402.
42. Jacobs DR, Jr., Tapsell LC. Food, not nutrients, is the fundamental unit in nutrition. *Nutr Rev* 2007;65:439–50.
43. Moeller SM, Reedy J, Millen AE, Dixon LB, Newby PK, Tucker KL, Krebs-Smith SM, Guenther PM. Dietary patterns: challenges and opportunities in dietary patterns research an Experimental Biology workshop, April 1, 2006. *J Am Diet Assoc* 2007;107:1233–9.
44. Jonnalagadda SS, Harnack L, Liu RH, McKeown N, Seal C, Liu S, Fahey GC. Putting the whole grain puzzle together: health benefits associated with whole grains—summary of American Society for Nutrition 2010 Satellite Symposium. *J Nutr* 2011;141:1011S–22S.
45. Slavin J. Fiber and prebiotics: mechanisms and health benefits. *Nutrients* 2013;5:1417–35.
46. Liu RH. Health-promoting components of fruits and vegetables in the diet. *Adv Nutr* 2013;4:384S–92S.
47. Steffen LM, Jacobs DR, Jr., Stevens J, Shahar E, Carithers T, Folsom AR. Associations of whole-grain, refined-grain, and fruit and vegetable consumption with risks of all-cause mortality and incident coronary artery disease and ischemic stroke: the Atherosclerosis Risk in Communities (ARIC) Study. *Am J Clin Nutr* 2003;78:383–90.
48. Wang L, Gaziano JM, Liu S, Manson JE, Buring JE, Sesso HD. Whole- and refined-grain intakes and the risk of hypertension in women. *Am J Clin Nutr* 2007;86:472–9.
49. He M, van Dam RM, Rimm E, Hu FB, Qi L. Whole-grain, cereal fiber, bran, and germ intake and the risks of all-cause and cardiovascular disease-specific mortality among women with type 2 diabetes mellitus. *Circulation* 2010;121:2162–8.
50. Joshipura KJ, Ascherio A, Manson JE, Stampfer MJ, Rimm EB, Speizer FE, Hennekens CH, Spiegelman D, Willett WC. Fruit and vegetable intake in relation to risk of ischemic stroke. *JAMA* 1999;282:1233–9.
51. Liu S, Manson JE, Lee IM, Cole SR, Hennekens CH, Willett WC, Buring JE. Fruit and vegetable intake and risk of cardiovascular disease: the Women's Health Study. *Am J Clin Nutr* 2000;72:922–8.
52. McCullough ML, Bandera EV, Patel R, Patel AV, Gansler T, Kushi LH, Thun MJ, Calle EE. A prospective study of fruits, vegetables, and risk of endometrial cancer. *Am J Epidemiol* 2007;166:902–11.
53. Micha R, Michas G, Mozaffarian D. Unprocessed red and processed meats and risk of coronary artery disease and type 2 diabetes—an updated review of the evidence. *Curr Atheroscler Rep* 2012;14:515–24.
54. Malik VS, Popkin BM, Bray GA, Després JP, Hu FB. Sugar-sweetened beverages, obesity, type 2 diabetes mellitus, and cardiovascular disease risk. *Circulation* 2010;121:1356–64.
55. Kimokoti RW, Newby PK. Dietary patterns, smoking, and cardiovascular diseases: a complex association. *Curr Nutr Rep* 2013;2:113–25.
56. Tucker KL, Maras J, Champagne C, Connell C, Goolsby S, Weber J, Zaghoul S, Carithers T, Bogle ML. A regional food-frequency questionnaire for the US Mississippi Delta. *Public Health Nutr* 2005;8:87–96.
57. Willett W. *Nutritional Epidemiology*, 3rd edition. New York: Oxford University Press, 2012.
58. Galea S, Tracy M. Participation rates in epidemiologic studies. *Ann Epidemiol* 2007;17:643–53.
59. Tolonen H, Dobson A, Kulathinal S. WHO MONICA Project. Effect on trend estimates of the difference between survey respondents and non-respondents: results from 27 populations in the WHO MONICA Project. *Eur J Epidemiol* 2005;20:887–98.
60. Waite LJ, Lehrer EL. The benefits from marriage and religion in the United States: a comparative analysis. *Popul Dev Rev* 2003;29:255–76.
61. Marcovina S, Bowsher RR, Miller WG, Staten M, Myers G, Caudill SP, Campbell SE, Steffes MW. Insulin Standardization Workgroup. Standardization of insulin immunoassays: report of the American Diabetes Association Workgroup. *Clin Chem* 2007;53:711–6.