Food Intake Does Not Differ between Obese Women Who Are Metabolically Healthy or Abnormal^{1–4}

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Abstract

Background: Metabolically healthy obesity may confer lower risk of adverse health outcomes compared with abnormal obesity. Diet and race are postulated to influence the phenotype, but their roles and their interrelations on healthy obesity are unclear.

Objective: We evaluated food intakes of metabolically healthy obese women in comparison to intakes of their metabolically healthy normal-weight and metabolically abnormal obese counterparts.

Methods: This was a cross-sectional study in 6964 women of the REasons for Geographic And Racial Differences in Stroke (REGARDS) study. Participants were aged 45–98 y with a body mass index (BMI; kg/m²) ≥18.5 and free of cardiovascular diseases, diabetes, and cancer. Food intake was collected by using a food-frequency questionnaire. BMI phenotypes were defined by using metabolic syndrome (MetS) and homeostasis model assessment of insulin resistance (HOMA-IR) criteria. Mean differences in food intakes among BMI phenotypes were compared by using ANCOVA.

Results: Approximately one-half of obese women (white: 45%; black: 55%) as defined by MetS criteria and approximately one-quarter of obese women (white: 28%; black: 24%) defined on the basis of HOMA-IR values were metabolically healthy. In age-adjusted analyses, healthy obesity and normal weight as defined by both criteria were associated with lower intakes of sugar-sweetened beverages compared with abnormal obesity among both white and black women ($P < 0.05$). HOMA-IR-defined healthy obesity and normal weight were also associated with higher fruit and low-fat dairy intakes compared with abnormal obesity in white women ($P < 0.05$). Results were attenuated and became nonsignificant in multivariable-adjusted models that additionally adjusted for BMI, marital status, residential region, education, annual income, alcohol intake, multivitamin use, cigarette smoking status, physical activity, television viewing, high-sensitivity C-reactive protein, menopausal status, hormone therapy, and food intakes.

Conclusions: Healthy obesity was not associated with a healthier diet. Prospective studies on relations of dietary patterns, which may be a better indicator of usual diet, with the phenotype would be beneficial. *J Nutr* 2014;144:2018–26.

Keywords: diet, healthy, metabolic, obesity, phenotype

Introduction

Obesity is associated with a high risk of cardiovascular diseases (CVDs)¹⁰, type 2 diabetes, certain forms of cancer, other metabolic disorders, and mortality (1–5). Obesity, however, shows considerable heterogeneity; compared with abnormal obesity, healthy obesity is characterized by a favorable metabolic profile and may confer lower

risk of adverse health outcomes (6–12). Currently, there is no standardized definition of healthy obesity and the phenotype has been defined by using criteria for metabolic syndrome (MetS) (7–15), a cluster of cardiometabolic risk factors (16), and/or insulin sensitivity/resistance measures (7, 8, 10–13, 17, 18). Approximately one-third of women in the United States are obese (19), and an approximately equal proportion of these are metabolically healthy (13).

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⁴ Supplemental Tables 1–3 are available from the “Online Supporting Material” link in the online posting of the article and from the same link in the online table of contents at <http://jn.nutrition.org>.

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¹⁰ Abbreviations used: CVD, cardiovascular disease; 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.

The etiology of healthy obesity is unclear, but genetic and lifestyle factors, sex, and race may influence the phenotype (6, 7). A few studies evaluated dietary intake of healthy obese individuals (20–22). Among U.S. participants of the INTERMAP (International Population Study on Macro/Micronutrients and Blood Pressure), nutrient and food intakes of healthy and unhealthy obese individuals were similar (20). Likewise, energy, carbohydrate, fat, and protein intakes did not differ between obese phenotypes in the third Korean NHANES (21). Conversely, healthy obese women who participated in the 1999–2004 U.S. NHANES had lower fiber intake and higher alcohol consumption than did their normal-weight counterparts (22). Moreover, it is established that obesity epidemiology, body composition, and fat distribution as well as energy, glucose and lipid metabolism, and insulin homeostasis differ by sex and race (23–26). Sex and race also influence dietary intake and dietary patterns due in part to gender-influenced eating behaviors, cultural differences, and socioeconomic status (2, 27–30; SE Judd, AJ Letter, JM Shikany, DL Roth, PK Newby, unpublished results, 2012). To our knowledge, no studies have compared food intakes of healthy obese individuals with those of their abnormal obese counterparts and other BMI phenotypes across race. Although MetS-based and HOMA-IR-defined healthy obesity have been shown to be associated with similar risks of CVDs, diabetes, and mortality (8, 10, 12), it is not known if dietary intakes of the phenotype on the basis of both criteria are similar.

In our study, we examined food intakes of metabolically healthy obese women defined by using both MetS and HOMA-IR criteria in comparison to intakes of other BMI phenotypes in the REasons for Geographic And Racial Differences in Stroke (REGARDS) study. We hypothesized that healthy obesity is associated with higher intakes of healthy foods (e.g., vegetables, fish, low-fat dairy foods) and lower consumption of less healthy foods [e.g., refined grains, processed meat, sugar-sweetened beverages (SSBs)] than abnormal obesity but with food intakes similar to the healthy normal-weight phenotype.

Methods

Study population

The REGARDS study is a national prospective cohort study that was designed to examine geographic and racial variations in stroke and its risk factors. The design and methods of the study are described elsewhere (31). Briefly, between 2003 and 2007, 30,239 white (58%) and black (42%) adults aged ≥ 45 y were enrolled via mail and telephone by using commercially available lists of residents. The cohort oversampled 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%); the rest (44%) were recruited 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.

This study included 13,433 women aged 45–98 y who completed an FFQ at the baseline examination (81% of women in the main study; $n = 16,632$). After excluding those with $>15\%$ missing responses ($n = 988$) and implausible energy intakes [<500 kcal/d or >4503 kcal/d (<2093 kJ/d or $>18,841$ kJ/d); $n = 355$], a total of 12,090 women remained. Of these, 6964 (58%) had a BMI (kg/m^2) ≥ 18.5 , were free of CVDs, diabetes mellitus, and cancer and had data on covariates and cardiometabolic risk factors.

Exposure and covariate

Upon enrollment (2003–2007), individuals underwent a computer-assisted telephone interview (CATI) followed by an in-home examination. Additional information was obtained via self-administered mail-in questionnaires (31). Age, marital status, residential region, education,

income, multivitamin use, alcohol intake, smoking status, physical activity, television viewing, hormone therapy, menopausal status, as well as medical information were obtained via CATI and mail-in questionnaire (31). An inventory of current medications, anthropometric and blood pressure measurements, and phlebotomy were performed during the in-home examination. Participants, dressed in hospital gowns and without shoes, were weighed with the use of a calibrated digital scale (Salter; Salter Brecknell) and height was measured by using a metal tape. BMI [$\text{weight} (\text{kg})/\text{height} (\text{m}^2)$] was calculated from measured height and weight. Waist circumference (WC) was measured on standing participants by using a cloth tape measure midway between the lowest rib on the right side and the top of the iliac crest (32). Blood pressure was determined by using the average of duplicate measurements with the use of an aneroid sphygmomanometer with the participant in a sitting position (31, 32). Fasting serum glucose, HDL cholesterol, and TGs were assessed by calorimetric reflectance spectrophotometry by using the Ortho Vitros Clinical Chemistry System 9501RC instrument (Johnson & Johnson Clinical Diagnostics). Serum insulin was measured by electrochemiluminescence immunoassay by using the Roche Elecsys 2010 system (Roche Diagnostics). Serum high-sensitivity C-reactive protein (hs-CRP) was analyzed by particle-enhanced immunonephelometry by using the BNII nephelometer (N High Sensitivity CRP; Dade Behring) (33).

Definition of BMI phenotypes

BMI categories were determined on the basis of NIH criteria (normal weight: 18.5 to <25.0 ; overweight: 25 to <30 ; obese: ≥ 30) (4). Metabolic status was defined according to both the American Heart Association/National Heart, Lung, and Blood Institute guidelines as outlined in the harmonized Joint Scientific Statement criteria for MetS (16) and HOMA-IR criteria (17, 18). MetS is defined as having ≥ 3 of the following individual components: abdominal obesity (WC ≥ 88 cm), elevated blood pressure ($\geq 130/\geq 85$ mm Hg) or drug therapy for hypertension, elevated glucose (≥ 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 (≥ 1.7 mmol/L) or drug therapy for hypertriglyceridemia (16). Insulin resistance was assessed from fasting glucose and insulin concentrations by using the formula $\text{HOMA-IR} = \text{fasting glucose (mmol/L)} \times \text{fasting insulin (pmol/L)}/22.5$ and was defined as the highest quartile of HOMA-IR scores (17, 18).

Dietary assessment

Dietary intakes were assessed by using the self-administered, semiquantitative 1998 Block (Block 98) FFQ. For each FFQ item, a common serving size of the food or beverage is specified (e.g., 64 g carrots), and participants are asked the frequency of consumption, on average, of this amount during the previous year. Individuals selected from 9 possible frequencies ranging from “never or less than once per month” to “1 (or 2) or more times per day” and selected the appropriate portion size. Portion size for unitary items (e.g., eggs) was queried as “1, 2, or 3” and the number consumed each time reported. For nonunitary foods, a photo was provided to aid in estimating 4 different portions. For each food, an amount was assigned on the basis of 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 (Berkeley, CA) for processing. The amount of each food consumed was calculated by multiplying the reported frequency by the portion size for each food item. We classified the 107 FFQ items into 56 food categories on the basis of culinary use (e.g., high- and low-fat milk) and similarities in nutrient content [e.g., fruit juice-flavored beverages such as Hi-C (Minute Maid, Coca-Cola) and SSBs] (Supplemental Table 1) (28; SE Judd, AJ Letter, JM Shikany, DL Roth, PK Newby, unpublished results, 2012). The Block 98 FFQ has not been validated in the REGARDS study cohort but it has been validated among Canadian women. FFQ reliability was high (Pearson correlation coefficients ranged from 0.57 to 0.9; median = 0.75) and validity was moderate to high (deattenuated Pearson correlation coefficients: 0.11–0.73; median = 0.59) (34).

Women who completed an FFQ ($n = 6964$), compared with those who did not complete an FFQ ($n = 2146$), were more likely to be from the Stroke Belt, to be married, to have a college degree and higher annual

income ($\geq \$35,000$), and to be physically active as well as to be postmenopausal and taking hormone therapy. FFQ completers, relative to FFQ noncompleters, also had lower BMI, a smaller WC, higher prevalence of normal weight and overweight, lower prevalence of total obesity and abdominal obesity, and a better metabolic profile (lower blood pressure; lower mean HOMA-IR index; lower concentrations of insulin, glucose, and hs-CRP; higher concentrations of HDL cholesterol; as well as lower prevalence of hypertension, hypertension treatment, hyperglycemia, insulin resistance, and low HDL cholesterol) (all $P < 0.05$; data not shown).

Statistical analysis

Race-specific analyses were conducted a priori, given the race differences in dietary intakes (27–30; SE Judd, AJ Letter, JM Shikany, DL Roth, PK Newby, unpublished results, 2012).

Characteristics. ANOVA was used to compare mean differences in continuous variables between white and black women (35). A chi-square test was used to compare differences in proportions of categorical variables in the subgroups of women (35). Participant characteristics 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, other), marital status (married, other), educational level (less than college degree, college degree or higher), annual income ($< \$35,000$, $\geq \$35,000$), alcohol intake (none, moderate, heavy) multivitamin use (yes, no), cigarette smoking status (nonsmoker, current smoker), physical activity (0, 1–3, ≥ 4 times/wk), television viewing (0 h/wk, 1–6 h/wk, ≥ 1 h/d), BMI categories (normal weight, overweight, obese), elevated WC (yes, no), elevated blood pressure (yes, no), hypertension medication (yes, no), elevated glucose (yes, no), HOMA-IR quartiles (1, 3/4), low HDL cholesterol (yes, no), elevated TGs (yes, no), lipid-lowering medication (yes, no), hormone therapy (yes, no), and postmenopausal status (yes, no) were analyzed as categorical variables. Results were summarized as means $\pm$ SEMs for continuous measures and percentages for categorical variables.

Food intake. ANCOVA 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, SSBs) and to identify pairwise mean differences in the BMI phenotypes (healthy normal weight, abnormal normal weight, healthy overweight, abnormal overweight, healthy obese, abnormal obese) by using Tukey's honestly significant difference test (which adjusted for multiple comparisons being examined). The SAS procedure PROC GLM was used to fit ANCOVA models (36). Three hierarchical models were fitted: model 1 adjusted for baseline age; model 2 additionally adjusted for BMI, marital status, residential region, educational level, income, alcohol intake, multivitamin use, cigarette smoking status, physical activity, television viewing, hs-CRP, menopausal status, hormone therapy, and food intakes; and model 3 further adjusted for energy intake. Results were summarized as means $\pm$ SEMs.

All analyses were performed by using the SAS (version 9.2, 2008) (35). $P < 0.05$ was considered significant. All statistical tests were 2-sided.

Results

Characteristics. Compared with black women, white women were older and were more likely to be married, have a college degree and an annual income of $\geq \$35,000$, and live in the Stroke Belt ($P < 0.0001$). White women were more likely to consume alcohol, use multivitamins, and to exercise but less likely to smoke and watch television than black women ($P < 0.01$). More white women were also postmenopausal and taking hormone therapy relative to black women ($P < 0.05$). Mean BMI, WC, systolic and diastolic blood pressure, HOMA-IR, as well as mean concentrations of insulin, glucose, and hs-CRP were lower in white women than in black women as was the prevalence of

total obesity, abdominal obesity, hypertension, hypertension treatment, hyperglycemia, and insulin resistance ($P < 0.0001$). Conversely, the prevalence of normal weight, overweight, lipid-lowering treatment, and hypertriglyceridemia, as well as mean TG concentrations were lower in black women ($P < 0.05$). White and black women did not differ in energy intake, mean HDL-cholesterol concentration, and prevalence of low HDL cholesterol (Table 1). White women had higher intakes of vegetables, red meat, low-fat dairy, and high-fat dairy as well as lower intakes of fruits, refined grains, processed meat, fried foods, fruit juice, and SSBs than did black women ($P < 0.05$) (Table 2).

MetS-defined healthy obesity: prevalence and food intake.

On the basis of MetS criteria, 45% of white obese women and 55% of black obese women were metabolically healthy (Tables 3 and 4). In age-adjusted analyses, white healthy obese and healthy normal-weight women had similar intakes of SSBs, which were significantly lower than those of abnormal obese women ($P < 0.0001$). By contrast, healthy and abnormal obese women consumed comparable amounts of red meat and fried foods that were higher than those of healthy normal-weight women ($P < 0.0001$). Intake of processed meat among healthy obese women was lower than that of their abnormal obese counterparts but higher relative to that of healthy normal-weight women ($P < 0.0001$). However, findings were attenuated and became nonsignificant in multivariable-adjusted models ($P \geq 0.05$) (Table 3). In age-adjusted analyses, black healthy obese and healthy normal weight women consumed lower quantities of SSBs that were similar to those consumed by abnormal obese women ($P < 0.001$).

Conversely, healthy obese and abnormal obese women had comparable significantly higher intakes of processed meat and fried foods that were higher than those of healthy normal-weight women ($P < 0.0001$). Red meat consumption among healthy obese women was, however, higher than that of their normal-weight counterparts but lower than that of abnormal obese women ($P < 0.05$). Results were attenuated and became nonsignificant in multivariable-adjusted analyses ($P \geq 0.05$) (Table 4).

HOMA-IR-defined healthy obesity: prevalence and food intake.

The prevalence of healthy obesity when defined by HOMA-IR criteria was lower among both white obese (28%) and black obese (24%) women in comparison to MetS criteria. Furthermore, prevalence was lower among black women than in white women in contrast to the MetS criteria. Similar to results with MetS criteria, relations between food intakes and BMI phenotypes were evident in age-adjusted analyses only (Supplemental Tables 2 and 3).

White healthy obese and healthy normal-weight women had similar significantly higher intakes of fruits and low-fat dairy products than those of abnormal obese women ($P < 0.001$); they also had comparable intakes of SSBs, which were lower than those of abnormal obese women ($P < 0.0001$). However, obese phenotypes were associated with higher consumption of processed meat than that of healthy normal-weight women ($P < 0.0001$). Intakes of red meat and fried foods among healthy obese women were lower than those of their obese counterparts but higher than those of healthy normal-weight women ($P < 0.0001$). Findings were attenuated and became nonsignificant in multivariable-adjusted models ($P \geq 0.05$) (Supplemental Table 2).

Black healthy obese and healthy normal-weight women consumed comparable lower amounts of SSBs than did abnormal obese women ($P < 0.001$). By contrast, healthy and abnormal obese women had similar intakes of processed meat and fried

TABLE 1 Characteristics of women in the REGARDS study¹

Characteristic	White (n = 4598)	Black (n = 2366)	P
Sociodemographic			
Age, y	63.9 ± 0.1	62.6 ± 0.2	<0.0001
Residing in Stroke Belt, %	61.2	52.3	<0.0001
Married, %	59.1	35.9	<0.0001
College degree or higher, %	38.3	30.5	<0.0001
Income ≥\$35,000, %	49.5	36.9	<0.0001
Lifestyle			
Energy intake, ² Mcal/d	1.58 ± 0.01	1.6 ± 0.01	0.07
Alcohol intake, %			<0.0001
None	58.7	74.4	
Moderate (≤14 g/d)	35.6	23.5	
Heavy (>14 g/d)	5.7	2.2	
Multivitamin use, %	34.8	22.5	<0.0001
Current smokers, %	12.6	15.8	0.0002
Physical activity, %			0.0019
0 times/wk	33.9	37.7	
1–3 times/wk	38.1	37.3	
≥4 times/wk	28.0	25.0	
Television viewing, %			0.0015
0 h/wk	1.1	0.3	
1–6 h/wk	14.5	13.5	
≥1 h/d	84.5	86.2	
Clinical			
BMI, kg/m ²	27.6 ± 0.1	31.2 ± 0.1	<0.0001
BMI category, %			<0.0001
Normal weight (BMI 18.5 to <25.0)	36.9	16.4	
Overweight (BMI 25 to <30)	35.2	33.3	
Obese (BMI ≥30)	27.9	50.4	
Waist circumference, cm	86.8 ± 0.2	94.1 ± 0.3	<0.0001
Elevated waist circumference (≥88 cm), %	42.3	64.0	<0.0001
Systolic blood pressure, mm Hg	122.2 ± 0.2	128.2 ± 0.3	<0.0001
Diastolic blood pressure, mm Hg	74.4 ± 0.1	78.2 ± 0.2	<0.0001
Elevated blood pressure (≥130/≥85 mm Hg), %	43.6	68.2	<0.0001
Hypertension medication, %	37.7	61.9	<0.0001
Serum glucose, ³ mmol/L	5.06 ± 0.01	5.18 ± 0.01	<0.0001
Elevated serum glucose (≥5.6 mmol/L), %	17.6	26.0	<0.0001
Serum insulin, ⁴ pmol/L	68.8 ± 0.7	95.1 ± 1.4	<0.0001
HOMA-IR index	23.0 ± 0.3	32.6 ± 0.6	<0.0001
Insulin-resistant (HOMA-IR quartile 4), %	40.7	60.5	<0.0001
Serum HDL cholesterol, ⁵ mmol/L	1.54 ± 0.01	1.52 ± 0.01	0.08
Low serum HDL cholesterol (<1.3 mmol/L), %	29.7	30.1	0.76
Serum TGs, ⁶ mmol/L	1.51 ± 0.01	1.14 ± 0.01	<0.0001
Elevated serum TGs (≥1.7 mmol/L), %	30.4	12.3	<0.0001
Lipid-lowering medication, %	25.6	23.3	0.0259
Serum high-sensitivity C-reactive protein, ⁷ mg/L	40.0 ± 1.0	56.0 ± 1.0	<0.0001
Hormone therapy, %	66.1	49.8	<0.0001
Postmenopausal, %	89.9	88.3	0.0282

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

² To convert kcal to kJ, multiply by 4.184.

³ To convert glucose to conventional units, divide by 0.0555.

⁴ To convert insulin to conventional units, divide by 6.945.

⁵ To convert cholesterol to conventional units, divide by 0.0259.

⁶ To convert TGs to conventional units, divide by 0.0113.

⁷ To convert C-reactive protein to conventional units, divide by 10.

foods that were significantly higher than those of healthy normal-weight women ($P < 0.0001$). Results were attenuated and became nonsignificant in multivariable-adjusted analyses ($P \geq 0.05$) (Supplemental Table 3).

Among both white and black women, no relations were observed between the other phenotypes and food intakes. Multivariable-adjusted models with and without energy adjustment were similar in both sets of analyses (data not shown).

TABLE 2 Age-adjusted mean food intake of women in the REGARDS study¹

Food group	Intake, g/d		P
	White (n = 4598)	Black (n = 2366)	
Vegetables	213 ± 2	175 ± 3	<0.0001
Fruits	132 ± 2	140 ± 3	0.0327
Whole-grain bread	15.9 ± 0.3	15.7 ± 0.4	0.63
Refined grains	20.1 ± 0.4	24.1 ± 0.6	<0.0001
Beans	12.2 ± 0.3	12.4 ± 0.5	0.72
Fish	18.4 ± 0.4	18.7 ± 0.5	0.60
Poultry	13.7 ± 0.3	13.8 ± 0.4	0.82
Red meat	30.1 ± 0.4	23.2 ± 0.6	<0.0001
Processed meat	12.7 ± 0.3	19.0 ± 0.4	<0.0001
Fried foods	15.4 ± 0.4	32.4 ± 0.6	<0.0001
Low-fat dairy	108 ± 3	27 ± 4	<0.0001
High-fat dairy	98.5 ± 2.3	84.9 ± 3.2	0.0006
100% Fruit juice	105 ± 3	187 ± 4	<0.0001
SSBs	113 ± 4	223 ± 6	<0.0001

¹ 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 intakes between white and black women. REGARDS, REasons for Geographic And Racial Differences in Stroke; SSBs, sugar-sweetened beverages.

Discussion

Healthy obesity was prevalent among REGARDS study women and varied by definition, being higher with MetS criteria (white: 45%; black: 55%) than with HOMA-IR criteria (white: 28%; black: 24%). Although food intakes were associated with healthy obesity and other BMI phenotypes in simple models, results were attenuated and no longer significant in models adjusted for potential confounders.

There are no published data on the prevalence of white healthy obese women in the United States. The prevalence of the phenotype in black women on the basis of absence of MetS in the Howard University Family Study (28%) (37) and on HOMA-IR in Ohio (33%) (38) was higher than that for black women in our study. Globally, reported prevalences of MetS-defined healthy obesity in women (23–42%) (9, 14, 15, 39) are lower relative to those for the women in the REGARDS study. Conversely, the prevalence of the HOMA-IR–defined phenotype was comparatively higher (43%) in the CoLaus (Cohorte Lausannoise) Study (39).

Consistent with findings in REGARDS study women, food intakes of healthy and unhealthy obese INTERMAP study adults did not differ (20). However, our study is unique in comparing food consumption across BMI phenotypes of white and black women defined by using more than one criteria. The higher prevalence of MetS-based healthy obesity compared with that defined by HOMA-IR among black women might be attributable to the fact that lipid measures are not predictive of insulin resistance in blacks (characterized by normal TGs and low HDL cholesterol), which results in underdiagnosis of MetS (40). Likewise, the variable prevalences among studies may be due to the age of participants as well as the use of different protocols for insulin sensitivity and varying cutoffs for obesity and MetS components (7).

The lack of association between diet and BMI phenotypes may underscore inadequacies of single-food analysis that fail to consider interactions and cumulative effects of foods and nutrients as well as biological interactions of diet with other metabolic factors (40, 41). Nevertheless, we chose to begin our assessment of dietary intakes among BMI phenotypes of

REGARDS study women with food intakes before proceeding to the dietary pattern level in future research. The dietary pattern approach, which considers total diet and related interactions, complements single-food analysis (27, 41, 42) and may better inform the relation between diet and healthy obesity. For instance, the relations of SSBs (43–47) and high-fat foods, including high-fat dairy (48–50) and meat (51), with obesity remain controversial and evidence is inconclusive. Some studies demonstrate an effect of SSBs on body weight, although the effect size is small and of equivocal significance. In a comprehensive review of the relation between high-fat dairy foods and obesity (50), high-fat dairy, consumed within distinctive dietary patterns, was found to protect against obesity. Bioactive substances in dairy fat (FAs such as butyric acid, phytanic acid, *cis*- and *trans*-palmitoleic acid) as well as pasture/grass-based dairy cow feeds (vs. grain/concentrate-based fodder) are hypothesized to confer health benefits. The only review (51) on meat intake and obesity demonstrated a positive association with both red and processed meat consumption; however, the heterogeneity among studies precludes a definitive conclusion being drawn. Moreover, there is insufficient evidence regarding the effect of SFAs and MUFAs, the main components of dairy and meat fat, on body weight (52–55). The proposition is that the weight gain associated with SSBs and high-fat foods that is observed in some studies is attributable to increased overall energy intake rather than to SSBs and high-fat foods (47, 48, 53, 56). Likewise, evidence suggests that lifestyle factors, including unhealthy diet, immoderate alcohol and tobacco use, physical inactivity, and sedentary behavior, tend to cluster, making it challenging to tease out the effect of a single component (43, 57).

Strengths of our study include a large, well-characterized, national, and diverse population as well as comparisons of food consumption with the use of 2 definitions of healthy obesity. The study comprised women of a broad age range with a large representation of black women and regional data. In contrast to other studies, associations of race and diet with healthy obesity were examined and analyses were also adjusted for a broad range of covariates.

A limitation of the study is its cross-sectional design, which has known limitations. Nonetheless, our objective was to evaluate the interrelations of BMI phenotypes and race on food intakes before performing prospective studies. The Block 98 FFQ is somewhat old but is ideal for the REGARDS study cohort because it specifically comprised foods contributing to diets of U.S. blacks (28, 30) and was among the main tools used to collect dietary data on racially diverse populations around the time the study was initiated (2003). However, it is likely the FFQ did not adequately discriminate between dietary intakes among white and black adults, potentially misclassifying and attenuating true differences in dietary intakes. Tucker et al. (58) developed a regional FFQ for use among Southern black populations, which contains more culturally specific foods and may have been a more suitable instrument, but the FFQ was not available at the inception of the REGARDS study. Furthermore, dietary self-report errors may have influenced our findings (59). The majority of women who completed an FFQ (58%) were from the Stroke Belt, possibly because individuals from this region were oversampled. In addition, FFQ completers were more likely to be married, to have a higher educational level and socioeconomic status, to be postmenopausal and taking hormone therapy, as well as to have a healthier lifestyle and better metabolic and health profiles. Educated people and those with higher socioeconomic status tend to be healthy and to participate more in studies, indicating greater awareness and trust in science. They are

TABLE 3 Adjusted mean food intake of white women in the REGARDS study according to BMI phenotype defined by MetS criteria¹

	BMI phenotype					
	Normal-weight		Overweight		Obese	
	Metabolically healthy	Metabolically abnormal	Metabolically healthy	Metabolically abnormal	Metabolically healthy	Metabolically abnormal
<i>n</i> (%) ²	1587 (93.2)	115 (6.8)	1226 (76.0)	388 (24.0)	577 (45.0)	705 (55.0)
Food group, g/d						
Vegetables						
Model 1	218 ± 4	186 ± 15	216 ± 5	202 ± 9	218 ± 7	205 ± 6
Model 2	204 ± 10	196 ± 16	208 ± 9	207 ± 11	205 ± 11	193 ± 11
Fruits						
Model 1	141 ± 3 ^a	99 ± 12 ^c	132 ± 4 ^{a,b}	119 ± 7 ^{b,c}	132 ± 6 ^{a,b}	129 ± 5 ^b
Model 2	130 ± 8	105 ± 13	122 ± 8	122 ± 9	127 ± 9	134 ± 9
Whole-grain bread						
Model 1	16.3 ± 0.5	14.6 ± 1.9	15.7 ± 0.6	15.2 ± 1.0	16.6 ± 0.8	15.9 ± 0.8
Model 2	18.3 ± 1.4	18.3 ± 2.2	17.5 ± 1.3	17.0 ± 1.5	17.8 ± 1.6	17.0 ± 1.6
Refined grains						
Model 1	19.4 ± 0.7	18.9 ± 2.5	19.3 ± 0.8	18.7 ± 1.4	20.8 ± 1.1	22.7 ± 1.0
Model 2	24.3 ± 1.7	24.2 ± 2.9	23.9 ± 1.6	23.4 ± 2.0	23.9 ± 2.0	25.6 ± 2.0
Beans						
Model 1	13.4 ± 0.5 ^{a,b}	15.6 ± 2.0 ^a	11.1 ± 0.6 ^c	11.1 ± 1.1 ^{b,c}	11.6 ± 0.9 ^{a,b,c}	11.7 ± 0.8 ^{a,b,c}
Model 2	13.1 ± 1.4	16.2 ± 2.3	11.7 ± 1.3	12.0 ± 1.6	11.8 ± 1.6	12.3 ± 1.6
Fish						
Model 1	18.3 ± 0.6	14.2 ± 2.1	18.6 ± 0.7	17.2 ± 1.2	18.8 ± 1.0	18.6 ± 0.9
Model 2	19.6 ± 1.5	19.0 ± 2.5	19.9 ± 1.4	19.8 ± 1.7	20.3 ± 1.7	20.9 ± 1.7
Poultry						
Model 1	13.4 ± 0.5	8.2 ± 1.7	14.0 ± 0.5	13.8 ± 1.0	13.9 ± 0.8	13.7 ± 0.7
Model 2	10.3 ± 1.2	7.9 ± 2.0	11.3 ± 1.1	12.6 ± 1.3	12.3 ± 1.4	12.6 ± 1.4
Red meat						
Model 1	26.3 ± 0.8 ^d	26.0 ± 2.8 ^{c,d}	30.4 ± 0.9 ^{b,c}	29.7 ± 1.6 ^{b,c}	32.9 ± 1.3 ^{a,b}	35.8 ± 1.1 ^a
Model 2	30.9 ± 1.9	30.9 ± 3.2	32.5 ± 1.8	31.1 ± 2.2	31.8 ± 2.2	33.4 ± 2.3
Processed meat						
Model 1	11.1 ± 0.4 ^c	11.1 ± 1.3 ^{b,c}	12.5 ± 0.4 ^b	13.0 ± 0.7 ^b	13.7 ± 0.6 ^b	15.6 ± 0.5 ^a
Model 2	13.1 ± 0.9	11.4 ± 1.5	12.5 ± 0.9	12.5 ± 1.1	11.6 ± 1.1	12.5 ± 1.1
Fried foods						
Model 1	12.2 ± 0.5 ^d	14.8 ± 1.8 ^{c,d}	15.0 ± 0.5 ^c	16.2 ± 1.0 ^{b,c}	18.7 ± 0.8 ^{a,b}	19.3 ± 0.7 ^a
Model 2	13.0 ± 1.2	13.9 ± 2.0	13.3 ± 1.2	12.9 ± 1.4	14.2 ± 1.4	12.5 ± 1.5
Low-fat dairy						
Model 1	117 ± 5 ^a	96 ± 19 ^{a,b}	116 ± 6 ^a	101 ± 11 ^{a,b}	101 ± 9 ^{a,b}	84 ± 8 ^b
Model 2	75 ± 13	72 ± 22	82 ± 13	74 ± 15	74 ± 15	67 ± 16
High-fat dairy						
Model 1	91 ± 4 ^b	95 ± 15 ^{a,b}	97 ± 5 ^b	96 ± 9 ^{a,b}	105 ± 7 ^{a,b}	116 ± 6 ^a
Model 2	96 ± 11	83 ± 18	104 ± 10	91 ± 12	108 ± 13	109 ± 13
100% Fruit juice						
Model 1	109 ± 4	123 ± 13	104 ± 4	106 ± 7	100 ± 6	105 ± 5
Model 2	97 ± 10	119 ± 16	104 ± 9	106 ± 11	110 ± 11	116 ± 12
SSBs						
Model 1	91 ± 6 ^b	132 ± 23 ^{a,b}	103 ± 7 ^b	147 ± 13 ^a	106 ± 10 ^b	154 ± 9 ^a
Model 2	140 ± 17	147 ± 27	134 ± 16	149 ± 19	113 ± 19	140 ± 19

¹ Values are means ± SEMs. ANCOVA was used to compute least-squares means of food intakes and to calculate pairwise mean differences in BMI phenotypes. Labeled means in a row without a common letter differ, $P < 0.05$. Metabolically healthy normal-weight (BMI (kg/m²) 18.5 to < 25.0 and <3 MetS components); metabolically healthy overweight (BMI 25 to <30 and <3 MetS components); metabolically healthy obese (BMI ≥30 and <3 MetS components); metabolically abnormal normal-weight (BMI 18.5 to <25.0 and ≥3 MetS components); metabolically abnormal overweight (BMI 25 to <30 and ≥3 MetS components); metabolically abnormal obese (BMI ≥ and ≥3 MetS components). Model 1 adjusted for baseline age. Model 2 adjusted for baseline age, BMI, marital status (married, other), residential region (Stroke Belt, other), education (less than college degree, college degree or higher), annual income (<\$35,000, ≥\$35,000), alcohol intake (none, moderate, heavy), multivitamin use (yes, no), cigarette smoking status (nonsmoker, current smoker), physical activity (0, 1–3, ≥4 times/wk), television viewing (0 h/wk, 1–6 h/wk, ≥1 h/d), high-sensitivity C-reactive protein, menopausal status (yes, no), hormone therapy (yes, no), and food intakes (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, SSBs; each food item was adjusted for all other food intakes). MetS, metabolic syndrome; REGARDS, REasons for Geographic And Racial Differences in Stroke; SSBs, sugar-sweetened beverages.

² Percentage of each BMI category.

similarly apt to have healthy lifestyles, as are older and married individuals (60–62). Our study sample might thus constitute a somewhat healthier subgroup and hence limit the

generalizability of our results. Finally, HOMA-IR results require cautious interpretation given the lack of standardization of insulin measures (63).

TABLE 4 Adjusted mean food intake of black women in the REGARDS study according to BMI phenotype defined by MetS criteria¹

	BMI phenotype					
	Normal-weight		Overweight		Obese	
	Metabolically healthy	Metabolically abnormal	Metabolically healthy	Metabolically abnormal	Metabolically healthy	Metabolically abnormal
<i>n</i> (%) ²	346 (89.6)	40 (10.4)	597 (74.8)	201 (25.2)	651 (55.1)	531 (44.9)
Food group, g/d						
Vegetables						
Model 1	180 ± 9	143 ± 26	185 ± 6	166 ± 11	173 ± 6	168 ± 7
Model 2	166 ± 20	140 ± 29	173 ± 18	162 ± 20	163 ± 18	168 ± 19
Fruits						
Model 1	138 ± 8	151 ± 26	141 ± 6	130 ± 11	140 ± 6	138 ± 7
Model 2	119 ± 21	163 ± 31	117 ± 19	118 ± 21	111 ± 19	116 ± 20
Whole-grain bread						
Model 1	15.2 ± 1.1	12.3 ± 3.5	15.6 ± 0.8	14.7 ± 1.5	15.4 ± 0.8	16.3 ± 0.9
Model 2	11.2 ± 3.1	9.6 ± 4.6	12.2 ± 2.9	11.1 ± 3.1	12.9 ± 2.9	14.6 ± 2.9
Refined grains						
Model 1	22.6 ± 2.0	25.3 ± 6.1	25.2 ± 1.5	26.3 ± 2.6	24.3 ± 1.4	23.5 ± 1.6
Model 2	24.6 ± 5.1	28.9 ± 7.7	24.6 ± 4.8	25.3 ± 5.2	21.7 ± 4.8	20.3 ± 4.9
Beans						
Model 1	13.6 ± 1.4	9.9 ± 4.4	12.2 ± 1.1	14.8 ± 1.8	12.6 ± 1.0	10.8 ± 1.1
Model 2	15.2 ± 3.1	15.5 ± 4.6	15.5 ± 2.9	18.5 ± 3.2	16.0 ± 2.9	14.5 ± 3.0
Fish						
Model 1	17.9 ± 1.6	12.7 ± 5.0	18.7 ± 1.2	17.2 ± 2.1	20.9 ± 1.2	18.3 ± 1.3
Model 2	19.5 ± 4.1	18.0 ± 6.1	18.5 ± 3.9	17.8 ± 4.2	20.0 ± 3.9	17.6 ± 3.9
Poultry						
Model 1	12.8 ± 1.2	8.8 ± 3.7	13.5 ± 0.9	13.1 ± 1.5	15.2 ± 0.8	14.7 ± 0.9
Model 2	11.9 ± 3.0	9.6 ± 4.4	10.8 ± 2.8	11.3 ± 3.0	11.9 ± 2.8	12.1 ± 2.9
Red meat						
Model 1	19.4 ± 1.6 ^c	19.9 ± 4.9 ^{a,b,c}	23.0 ± 1.2 ^{b,c}	21.9 ± 2.0 ^{b,c}	23.8 ± 1.1 ^b	27.5 ± 1.3 ^a
Model 2	26.1 ± 4.0	24.1 ± 6.0	27.1 ± 3.7	24.7 ± 4.1	25.5 ± 3.7	27.6 ± 3.8
Processed meat						
Model 1	15.0 ± 1.2 ^d	17.3 ± 3.6 ^{a,b,c,d}	17.5 ± 0.9 ^{c,d}	18.8 ± 1.5 ^{b,c}	20.5 ± 0.8 ^{a,b}	22.3 ± 0.9 ^a
Model 2	19.3 ± 2.8	17.6 ± 4.2	19.3 ± 2.6	19.3 ± 2.9	19.4 ± 2.6	18.9 ± 2.7
Fried foods						
Model 1	22.8 ± 2.1 ^c	21.7 ± 6.6 ^{b,c}	31.3 ± 1.6 ^b	33.9 ± 2.8 ^{a,b}	35.0 ± 1.5 ^{a,b}	38.4 ± 1.7 ^a
Model 2	26.4 ± 5.2	27.3 ± 7.7	31.6 ± 4.8	33.1 ± 5.3	31.9 ± 4.8	32.6 ± 4.9
Low-fat dairy						
Model 1	30.6 ± 5.1	40.5 ± 15.8	25.6 ± 3.8	15.5 ± 6.6	30.8 ± 3.7	19.9 ± 4.1
Model 2	23.6 ± 13.6	47.2 ± 20.3	17.4 ± 12.7	10.3 ± 13.9	21.9 ± 12.7	12.7 ± 13.0
High-fat dairy						
Model 1	91 ± 7	101 ± 23	78 ± 6	75 ± 10	88 ± 5	85 ± 6
Model 2	92 ± 20	108 ± 30	72 ± 19	63 ± 21	78 ± 19	68 ± 19
100% fruit juice						
Model 1	205 ± 12	182 ± 37	180 ± 9	198 ± 16	182 ± 9	178 ± 10
Model 2	236 ± 32	193 ± 47	210 ± 30	227 ± 32	203 ± 30	198 ± 30
SSBs						
Model 1	193 ± 18 ^c	226 ± 55 ^{a,b,c}	200 ± 13 ^c	257 ± 23 ^{a,b}	218 ± 13 ^{b,c}	276 ± 14 ^a
Model 2	155 ± 46	127 ± 68	169 ± 43	195 ± 47	168 ± 43	206 ± 44

¹ Values are means ± SEMs. ANCOVA was used to compute least-squares means of food intakes and to calculate pairwise mean differences in BMI phenotypes. Labeled means in a row without a common letter differ, *P* < 0.05. Metabolically healthy normal-weight [BMI (kg/m²) 18.5 to < 25.0 and <3 MetS components]; metabolically healthy overweight (BMI 25 to <30 and <3 MetS components); metabolically healthy obese (BMI ≥30 and <3 MetS components); metabolically abnormal normal-weight (BMI 18.5 to <25.0 and ≥3 MetS components); metabolically abnormal overweight (BMI 25 to <30 and ≥3 MetS components); metabolically abnormal obese (BMI ≥ and ≥3 MetS components). Model 1 adjusted for baseline age. Model 2 adjusted for baseline age, BMI, marital status (married, other), residential region (Stroke Belt, other), education (less than college degree, college degree or higher), annual income (<\$35,000, ≥\$35,000), alcohol intake (none, moderate, heavy), multivitamin use (yes, no), cigarette smoking status (nonsmoker, current smoker), physical activity (0, 1–3, ≥4 times/wk), television viewing (0 h/wk, 1–6 h/wk, ≥1 h/d), high-sensitivity C-reactive protein, menopausal status (yes, no), hormone therapy (yes, no), and food intakes (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, SSBs; each food item was adjusted for all other food intakes). MetS, metabolic syndrome; REGARDS, REasons for Geographic And Racial Differences in Stroke; SSBs, sugar-sweetened beverages.

² Percentage of each BMI category.

In conclusion, approximately one-half of obese women in the REGARDS study as defined by MetS criteria and approximately one-quarter defined on the basis of HOMA-IR were metabolically healthy. Food intakes were not associated with healthy obesity or with other BMI phenotypes. Prospective studies on relations of dietary patterns, which may be a

better indicator of usual diet, with healthy obesity would be beneficial.

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