Serum adiponectin in relation to body mass index and other correlates in black and white women

Sarah S. Cohen, PhD[1,2], **Marilie D. Gammon, PhD**[1], **Lisa B. Signorello, Sc D**[2,3], **Kari E. North, PhD**[1,4], **Ethan M. Lange, PhD**[5], **Jay H. Fowke, PhD**[3], **Margaret K. Hargreaves, PhD**[6], **Qiuyin Cai, MD, PhD**[3], **Wei Zheng, MD, PhD**[3], **William J. Blot, PhD**[2,3], and **Charles E. Matthews, PhD**[7]

[1]Department of Epidemiology, University of North Carolina, Chapel Hill, NC

[2]International Epidemiology Institute, Rockville, MD

[3]Division of Epidemiology, Department of Medicine, Vanderbilt University Medical Center and Vanderbilt-Ingram Cancer Center, Nashville, TN

[4]Carolina Center for Genome Sciences, University of North Carolina, Chapel Hill, NC

[5]Departments of Biostatistics and Genetics, University of North Carolina, Chapel Hill, NC

[6]Department of Internal Medicine, Meharry Medical College, Nashville, TN

[7]Nutritional Epidemiology Branch, National Cancer Institute, Rockville, MD

Abstract

Purpose—Adiponectin is a promising biomarker linking obesity and disease risk; however, limited data are available regarding adiponectin in black women among whom obesity is highly prevalent.

Methods—A cross-sectional analysis was conducted to assess racial differences and correlates of serum adiponectin measured in 996 black and 996 white women enrolled in the Southern Community Cohort Study through Community Health Centers in twelve southeastern states from 2002–2006.

Results—Blacks had significantly lower adiponectin levels than whites (median 10.9 versus 14.9 ug/ml, Wilcoxon p<0.0001). Among blacks, adiponectin was lower among overweight and obese women compared to healthy weight women but showed no clear decreasing trend with increasing severity of obesity; adjusted geometric means (95% confidence interval) were 15.0 (13.8–16.4), 11.5 (10.6–12.5), 9.7 (9.0–10.6), 11.4 (10.3–12.6), and 10.9 (9.5–12.6) ug/ml for body mass index [BMI] categories of 18.5–24.9, 25–29.9, 30–34.9, 35–39.9, and 40–45 (p for trend<0.0001). In contrast, among whites there was a monotonic reduction in adiponectin over increasing BMI (adjusted geometric means = 19.9 (18.3–21.7), 15.1 (13.9–16.4), 14.3 (13.2–15.5), 12.5 (11.2–13.9), and 11.0 (9.7–12.5) ug/ml, p for trend<0.0001). BMI, age, HDL-cholesterol, and hypertension were important correlates of adiponectin in both groups.

Correspondence: Sarah S. Cohen International Epidemiology Institute 1455 Research Blvd, Suite 550 Rockville, MD 20850 Telephone) 301-279-4279 Fax) 301-424-1053 sarah@ieixmail.com.

Conclusions—Among women, racial differences exist in both the magnitude and form of the adiponectin-BMI association.

Keywords

Adiponectin; obesity; African Americans

INTRODUCTION

Adiponectin is a protein produced exclusively in adipose tissue that appears to play a critical role in mediating physiological effects such as insulin sensitivity, inflammatory response, and cell proliferation. Adiponectin levels are inversely associated with obesity and are thought to decrease in individuals with increased adiposity through down-regulation of adiponectin receptors (1). Adiponectin may also be affected by diet, physical activity, comorbidities, and other environmental factors as well as variation in genes encoding adiponectin or its receptors (1). Because adiponectin is inversely associated with obesity phenotypes as well as several obesity-related diseases (2), there is speculation that adiponectin activity may be a useful target in the prevention of cardiovascular disease, cancer, and type 2 diabetes (1,2). With the highest prevalence of obesity found among non-Hispanic black women (39%) and the lowest among non-Hispanic white women (22%) (3), differences in adiponectin levels across race groups may contribute to disparities in obesity-related diseases between black and white women.

Identifying correlates of adiponectin and ascertaining whether they vary by race is likely to enhance the development of effective adiponectin-related chemopreventive strategies. Thus, the goal of this analysis was to examine associations between adiponectin levels, body mass index (BMI), and other potential correlates and to assess whether associations varied by race.

METHODS AND PROCEDURES

Institutional Review Boards at Vanderbilt University, Meharry Medical College, and the University of North Carolina at Chapel Hill approved this study.

Study population

The Southern Community Cohort Study (SCCS) is a prospective epidemiologic cohort study designed to examine racial disparities in cancer incidence and mortality (4). Study enrollment began in 2002 in 12 southeastern states at Community Health Centers (CHC) which are government-funded facilities providing health services primarily to low-income individuals in medically underserved areas (5). Potential participants included anyone entering the CHC such as patients, persons accompanying patients, and individuals utilizing CHC pharmacy or dental services. Individuals were eligible if they had not been under treatment for cancer in the past year. A personal or family history of cancer or elevated risk factors for cancer was not used to determine eligibility. Additionally, as described previously (4), participants were required to be age 40–79 years and English-speaking. From over 47,000 SCCS participants enrolled through early 2006, a sub-sample of 2,000 women who provided a blood sample at study enrollment and self-reported their race as either `Black/African American' or `White' was selected for further biomarker analyses. This included a random sample of 395 women selected in 2005 within three strata (race, BMI, and smoking status) and a second random sample of 1,605 women selected in 2006 in equal numbers across race, BMI, and menopausal status categories.

Data collection

Trained study interviewers led all participants through a structured questionnaire using a computer-assisted interview with extensive skip patterns and range and logic checks. The interview elicited information including demographics, anthropometrics, and several aspects of health and behavior. Physical activity was measured using a questionnaire developed for the SCCS to comprehensively assess active and sedentary behaviors at the time of the interview. Dietary intake in the year prior to the baseline interview was measured using an 89-item food frequency questionnaire (FFQ) designed specifically for the SCCS to elicit information about foods most commonly eaten in the southeastern United States (6,7). For the 20% of women who were patients in the CHC on the day of the baseline interview, measured height and weight were abstracted from medical records for validation purposes. Participants self-reported diagnoses of medical conditions including diabetes, heart attack or coronary bypass surgery, hypertension, high cholesterol, and depression by responding "Yes" to questions beginning "Has a doctor ever told you that you have…?"

A convenience blood sample was collected at the time of recruitment using one EDTA-containing plasma tube and one serum BD Vacutainer® tube. For this study, the median time (hours) between the last reported meal and blood collection was 6.0 for blacks and 6.3 for whites (p=0.07). Fasting blood, defined as at least 8 hours since last meal, was collected for 44% of the participants. Blood samples were shipped cold to Vanderbilt University in Nashville, TN, where they were processed for storage at −80° C. 84% of the blood samples were received the day after the blood draw and 98% were received within two days. The samples were frozen for an average of 2.6 years (range 3 months to 5 years) prior to analysis.

Laboratory assays

Adiponectin levels were measured in serum by immunoassay using the LINCOplex kit (Luminex® xMAP™ Technology, St. Louis, MO) in the Vanderbilt Hormone Assay and Analytical Services Core Laboratory in duplicate for each woman. The average of the two measurements was used in all analyses. Duplicate sets of samples for five randomly selected women as well as five repeat samples from each of two pooled samples were measured to assess the reliability and validity of the assay. Adiponectin levels were successfully measured in 1,992 of the 2,000 samples (eight samples failed due to a filter plate error or low sample volume). The intra-assay coefficient of variation was 9.4%. High-density lipoprotein (HDL) cholesterol was measured in serum by the Vanderbilt Lipid Laboratory using the ACE Clinical Chemistry System and the ACE HDLC Reagent (#SA1038) following the manufacturer's protocols (Alfa Wassermann, Inc, West Caldwell, NJ). The intra-assay coefficient of variation was 1.6%. Neither adiponectin nor HDL-cholesterol levels differed by fasting status (for adiponectin, p=0.3 for blacks, p=0.6 for whites; for HDL, p=0.5 for blacks, p=0.4 for whites).

Statistical Methods

For this cross-sectional analysis, data from 1,992 women with measured adiponectin were analyzed. BMI was calculated from self-reported values as [weight (kg)] / [height (m)^2]. Dietary intakes, total physical activity, and HDL-cholesterol were categorized into quartiles based on the distribution of the entire sample. Other characteristics were categorized as shown in Table 1. The Wilcoxon signed rank test was used to compare adiponectin levels between groups.

Adiponectin had a skewed distribution (Figure 1), and therefore was log-transformed to better meet modeling assumptions (8). To ease presentation, back-transformed values are shown. Linear regression models were constructed for blacks and whites separately. All

models were adjusted for sample selection (395 selected in 2005 versus 1605 selected later) and age at baseline interview. Further adjustment for factors used in the sample selection (cigarette smoking status and menopausal status) did not alter the results and were not included in the models presented here. Categorized covariates with inherent order were modeled using indicator variables rather than as ordinal variables because the assumption of linearity was not generally met.

The first analytic objective was to characterize the relationship between adiponectin and BMI within each race group. BMI (continuous) was regressed on adiponectin, and potential confounders (determined from the literature and categorized as shown in Table 1) were added to the model using backwards model selection with a change-in-estimate criterion of ≥ 5%. Linear trend tests were conducted via F-tests of the continuous BMI variable. Potential modifiers of the BMI-adiponectin relationship were assessed using the likelihood ratio test (LRT) to compare models with and without interaction terms for BMI and potential modifiers. Additionally, adjusted geometric means for adiponectin were calculated from linear regression models that included standard categories of BMI (<18.5, 18.5–24.9, 25–29.9, 30–34.9, 35–39.9, and 40–45 kg/m^2) and the final set of confounders.

The second analytic goal was to determine correlates of adiponectin after adjustment for BMI. BMI was forced into the linear regression model and potential correlates were then added sequentially. Models were compared using Akaike's Information Criterion (AIC) which balances model fit with model complexity (9), and potential correlates were included in the final model as long as their addition resulted in an AIC value at least one unit lower than the AIC for the smaller-order model.

SAS/STAT software, Version 9.1 of the SAS System for Windows (SAS Institute Inc., Cary, NC, USA) was used for all analyses.

RESULTS

Adiponectin levels were 25% lower among black women compared to white women (medians: 10.9 versus 14.9 ug/ml, respectively, Wilcoxon p<0.0001). After adjustment for BMI and age, adiponectin was still significantly lower in blacks (p<0.0001). Unadjusted adiponectin levels decreased with increasing BMI categories in both groups although not as consistently among blacks (Table 1). There was a strong positive association between adiponectin and both age and HDL-cholesterol while adiponectin was slightly lower among women reporting diabetes or hypertension. Unadjusted adiponectin levels increased with increasing alcohol consumption in whites but not in blacks. Adiponectin was not consistently associated with education, income, physical activity, cigarette smoking, or any of the dietary or reproductive indices.

There was a strong linear association between continuous BMI and adiponectin in both race groups (test for linear trend, p<0.0001 for black and white women). Figure 2 illustrates race-specific associations between categories of BMI and adiponectin adjusted for HDL-cholesterol, the only variable found to be a confounder of the BMI-adiponectin relationship. Adjusted adiponectin means for blacks were lower than those for whites within each category of BMI although the differences for women in the highest categories of BMI (35–39.9 and 40+) were small. Among blacks, adiponectin decreased over BMI values up to 30–34.9 before leveling off at the highest levels of BMI. In contrast, among whites, adiponectin levels were lower with each increasing category of BMI. Menopausal status did not modify the BMI-adiponectin relationship (LRT p=0.9 for blacks, p=0.6 for whites) (data not shown). In models restricted to 421 black and 450 white women with fasting blood samples,

the association between BMI and adiponectin was essentially unchanged for both groups (data not shown).

Table 2 shows race-specific prediction models for adiponectin. Based on the AIC values, BMI was an important correlate of adiponectin in both race groups although the regression coefficient was larger in magnitude among whites. Age, HDL-cholesterol, and hypertension were also significant correlates of adiponectin in both groups based on the AIC comparisons. None of the other factors examined added additional predictive value to the models.

As both diabetes and treatment for diabetes have been shown to affect adiponectin levels (10), we repeated our main analyses in the subset of non-diabetic women in this study (N=776 black and N=832 white). Among the non-diabetics, median adiponectin levels remained significantly lower in blacks compared to whites (median: 11.0 versus 15.3 ug/ml, respectively, Wilcoxon p<0.0001). Adjusted geometric means for adiponectin across categories of BMI were also not appreciably different in either race group among the non-diabetic women compared to the patterns observed in the entire sample (for BMI categories of 18.5–24.9, 25–29.9, 30–34.9, 35–39.9, and 40–45, adjusted adiponectin means were 15.3, 11.4, 9.7, 11.2, and 9.9 in non-diabetic black women and 20.1, 15.4, 14.7, 12.0, and 10.8 in non-diabetic white women).

DISCUSSION

In this largest to-date, cross-sectional study of black and white women from similar geographic and socioeconomic situations, we observed that blacks have lower adiponectin levels than whites even after adjustment for BMI. Our results expand upon previous studies that have also found lower levels of adiponectin in blacks but were limited either by small numbers of black participants (11–14) or limited age ranges (15–17), indicating that racial differences in adiponectin exist across the adult age spectrum.

Adiponectin has consistently been found to be negatively correlated with obesity in white and Asian populations (18–20). Despite racial differences in the prevalence of obesity and risk for obesity-related disease, few large studies have examined adiponectin in relation to obesity in blacks. As did our study, several small studies (12,14,21) as well as the Atherosclerosis Risk in Communities Study (15) found that adiponectin decreased over categories of BMI in blacks and that adjusted adiponectin values were lower for black women compared to white women in each BMI category. Our analysis included larger numbers of women with higher values of BMI than in previous studies, and we showed for the first time that the form of the BMI-adiponectin association may differ by race with white women showing a consistent decline in adiponectin over all levels of BMI while among blacks, adiponectin was lower among overweight and obese women compared to healthy weight women but there was little trend with increasing severity of obesity.

Our first-ever extensive examination of potential correlates of adiponectin found few factors that were strongly associated with adiponectin. Adiponectin levels rose with advancing age, consistent with earlier studies (18,22). The direction of this association has previously been noted to be paradoxical; abdominal fat, which is inversely associated with adiponectin, increases with age indicating that adiponectin levels should decrease with age. One potential explanation for this seeming contradiction is that estrogen, thought to inhibit adiponectin, decreases with age allowing adiponectin to rise (18). We also observed a strong positive association between HDL-cholesterol and adiponectin, as have others (18,22). Mechanistically, it is hypothesized that decreased adiponectin levels may affect hepatic insulin resistance leading to increased hepatic lipase activity and decreased HDL levels

(18,22). While the cross-sectional nature of our data did not allow us to examine the temporality of the HDL-adiponectin relationship, our data demonstrate that this strong association holds across race and age groups.

Low adiponectin levels have been inversely linked to hypertension (23–25) including in one study of blacks (25), a finding we also observed in both race groups. A few prior studies have found this association only among participants with insulin resistance, but at least some evidence indicates that low adiponectin levels may affect the development of hypertension at an early stage, without involvement of insulin resistance (24).

Based on previous examination in the literature as well as plausible roles in the physiologic pathways through which adiponectin is believed to act, we selected additional medical conditions for inclusion in this study, but they were ultimately not found to be important correlates of adiponectin in our final multivariate model. We found that adiponectin levels were only slightly lower among diabetics, and after adjustment for BMI and age, diabetes and adiponectin were not significant associated. This was somewhat unexpected since adiponectin and diabetes incidence have previously been shown to be inversely linked (10). One explanation may be that many SCCS women with prevalent diabetes were receiving treatment for their diabetes (among diabetics in this study, 75% of white women and 87% of black women reported taking medication for their diabetes), thus reducing effects of hyperinsulinemia on adiponectin levels. We also observed slightly lower adiponectin levels among women reporting depression, a finding consistent with the literature to date that has demonstrated lower adiponectin among patients with major depressive disorder and anxiety scores (26); however, in our study, this difference was non-significant after inclusion in the final multivariate model.

Neither physical activity nor dietary factors were predictive of adiponectin levels despite their known roles as major components of energy balance. In contrast, a prior study found that physical activity was associated with increased adiponectin levels, with moderate/high intensity activity showing stronger effects than low intensity activity (27). It is possible that the activity levels in our population were too low overall to detect effects on adiponectin levels; in fact, less than 20% of the women in either race group in this study met the recommended guidelines for physical activity (28). Little research has been conducted regarding associations between dietary factors and adiponectin. One study found no association between total calories or macronutrient intake and adiponectin (29) while fiber was found to be positively associated with adiponectin among diabetics (30,31). It seems likely that there are many intermediaries in the pathways linking diet and physical activity to adiponectin, making the detection of associations difficult in our cross-sectional dataset. Additionally, it is possible that once age, BMI, and HDL-cholesterol were included in models for adiponectin, minimal additional predictive value was added by factors such as diabetes, diet, and physical activity which are reasonably expected to be predictive of adiponectin but are also strongly associated with age, BMI, and HDL-cholesterol. While standard in large epidemiologic studies, the physical activity and dietary intakes obtained via questionnaire rather than objective measures may also have limited the detection of associations with adiponectin.

This study is potentially limited by our measurement of adiponectin. The high-molecular weight (HMW) form of adiponectin has been suggested to be the more biologically active (32) and thus, we may have been unable to detect certain associations because we (like most other large, population-based studies) did not specifically measure HMW adiponectin. Additionally, adiponectin was measured only at a single point in time. Studies in white and Chinese individuals found that adiponectin levels measured one year apart were highly correlated, indicating that a single measurement of adiponectin is likely sufficient for large

epidemiologic studies (33,34). Another possible limitation of our adiponectin measurement is that it was not conducted exclusively in fasting samples; however, analyses limited to samples provided more than 8 hours since the last meal did not show any appreciable differences from those using the entire population.

The use of self-reported height and weight measures is also a potential limitation. A recent review indicates that among women, height tends to be over-reported and weight under-reported (35). However, data from the 1999–2004 National Health and Examination Survey show that despite errors in self-report, BMI categories based on self-reported values still generally demonstrate good agreement with BMI categories from measured values (36). These data also showed that under-reporting was more common in whites and among well-educated women (36) which suggests that the BMI values calculated from self-report in the SCCS may be less vulnerable to bias than in other studies of more educated, white participants. Furthermore, in the SCCS, BMI values calculated from self-reported height and weight were very highly correlated with BMI values calculated from medical record data overall (Pearson correlation coefficient > 0.95) as well as across strata of BMI, race, education, and income, indicating that the self-reported values are generally of good quality. An additional study limitation is the absence of a measurement of central obesity as it has been shown that the amount of centrally-deposited adipose tissue differs between black and white women at the same level of BMI (37) and further, that there are differences in the proportion of the various isoforms of adiponectin in relation to measures of central obesity across race groups (38).

A major strength of this study is the utilization of the SCCS resource. While the women included in this study do not reflect the distribution of all women in the US due to the recruitment of SCCS participants via CHCs, the SCCS participants are generally reflective of the 14 million users of CHCs in the US who represent a largely understudied group of individuals at high risk for many diseases. More importantly, within-population comparisons such as those presented here, are valid regardless of generalizability. Indeed, by design, the black and white participants in this study arose from similar geographic and SES backgrounds facilitating the examination of racial differences by minimizing the potential role of SES-driven confounding, a limitation that clouds the interpretation of results from many previous studies. Our study also overcomes limitations from previous studies which were hampered by small numbers of black participants, narrow age ranges, and few participants with BMI greater than 35.

Analysis of this large population of highly comparable black and white women shows that adiponectin levels are lower in blacks than in whites and that adiponectin is inversely associated with obesity but the shape of the BMI-adiponectin association differs by race. Additionally, we demonstrated that age, HDL-cholesterol, and hypertension are strong correlates of adiponectin in both race groups. Future work within the SCCS and other studies with diverse populations will be guided by these findings as the mechanistic role played by adiponectin in the development of disease is examined. Further, efforts to develop treatments that can alter adiponectin levels or even therapeutic applications of adiponectin itself for diseases such as cardiovascular disease, cancer, and diabetes may be guided by the racial differences in adiponectin levels and its correlates observed in this study.

Acknowledgments

ABBREVIATIONS AND ACRONYMS

SCCS	Southern Community Cohort Study
CHC	Community Health Center
BMI	Body mass index
FFQ	Food frequency questionnaire
HDL	High-density lipoprotein
AIC	Akaike's Information Criterion
HMW	High molecular weight

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Figure 1.
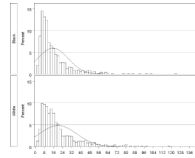Distribution of adiponectin levels (ug/ml) in 996 black and 996 white women in the Southern Community Cohort Study, 2002–2006
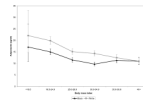
Figure 2.
Adjusted geometric means and 95% confidence intervals for adiponectin levels (ug/ml) by body mass index categories in 996 black and 996 white women in the Southern Community Cohort Study, 2002–2006. Geometric means are adjusted for age, sample selection, and HDL-cholesterol.

Table 1

Descriptive Statistics for Unadjusted Serum Adiponectin Levels (ug/ml) Among 1,992 Women in the Southern Community Cohort Study, 2002–2006

	Black women			White women		
	N	Median	25th–75th percentile	N	Median	25th–75th percentile
All participants	996	10.9	7.0 – 19.1	996	14.9	9.0 – 24.7
Body mass index (kg/m²)						
< 18.5	8	22.3	8.8 – 36.2	10	28.9	13.5 – 48.3
18.5 – 24.9	240	15.7	9.6 – 25.7	239	21.8	13.6 – 36.6
25.0 – 29.9	248	11.0	7.4 – 18.7	248	15.7	9.8 – 25.0
30.0 – 34.9	250	8.9	5.9 – 14.4	249	13.7	8.7 – 20.9
35.0 – 39.9	165	10.6	6.0 – 17.9	146	10.7	7.7 – 18.5
40.0 +	85	9.2	6.0 – 16.6	104	9.5	5.9 – 15.4
Age at interview						
40 – 44	330	9.8	6.5 – 18.0	341	13.9	8.5 – 20.8
45 – 49	265	10.3	6.7 – 18.7	243	13.9	8.2 – 21.6
50 – 54	148	12.2	7.2 – 19.7	136	15.4	8.6 – 28.0
55 – 59	105	11.5	7.3 – 21.5	120	16.2	10.1 – 30.1
60 – 64	53	13.6	8.6 – 19.1	93	17.3	10.6 – 28.2
65 – 69	50	12.2	7.4 – 25.0	29	20.9	11.8 – 32.9
70 – 74	25	12.6	8.4 – 25.3	24	21.1	13.7 – 43.3
75 – 79	20	18.7	10.4 – 21.4	10	29.5	24.8 – 51.4
Household income						
< $15K	620	10.9	7.0 – 19.0	594	14.5	9.4 – 24.1
$15 – $25K	236	10.7	6.9 – 18.3	205	14.4	8.6 – 25.3
$25 – $50K	115	12.5	6.8 – 20.6	129	16.5	8.6 – 29.9
> $50K	16	9.9	6.1 – 18.8	64	16.0	11.0 – 22.9
Education (years)						
< 9	78	11.9	7.7 – 22.4	85	14.7	8.3 – 22.0
9 – 11	241	11.0	7.1 – 18.9	224	14.2	8.9 – 25.2
12	415	11.4	7.2 – 19.8	411	14.6	9.0 – 25.5
> 12	262	10.3	6.1 – 18.2	276	15.6	9.4 – 22.7

	Black women			White women		
	N	Median	25th–75th percentile	N	Median	25th–75th percentile
Physical activity (Met-hrs/day)						
Q1 (<10.2)	248	11.6	7.5	20.3	247	14.0	8.7	21.9
Q2 (10.2–18.3)	256	11.6	6.9	19.5	240	16.2	9.8	27.2
Q3 (18.4–29.2)	255	10.6	6.9	18.5	242	14.7	8.8	24.9
Q4 (>29.2)	233	10.8	6.8	18.5	262	15.4	8.8	25.7
HDL cholesterol (mg/dl)						
Q1 (<43)	170	7.1	5.0	10.8	314	10.2	6.6	16.8
Q2 (43–50)	231	8.7	5.8	14.9	272	14.2	9.6	21.9
Q3 (51–60)	245	12.9	8.5	21.4	220	16.7	10.8	29.4
Q4 (>60)	342	14.7	9.3	24.4	182	24.2	16.2	40.9
Total energy intake (kcal/day)						
Q1 (<1352)	213	10.0	6.2	18.0	268	15.4	9.2	27.2
Q2 (1352–1875)	226	11.7	7.6	18.9	255	15.1	8.8	26.5
Q3 (1876–2644)	217	10.9	7.2	22.7	264	14.5	8.9	21.9
Q4 (>2644)	294	11.4	7.2	18.0	187	15.0	8.7	22.8
Total fat intake (g/day)						
Q1 (<49)	215	9.7	6.1	18.0	266	15.4	9.6	29.8
Q2 (49–71)	234	10.9	7.5	18.7	247	14.6	8.7	22.6
Q3 (72–103)	223	11.9	7.0	22.4	258	14.3	8.9	23.2
Q4 (>103)	278	11.7	7.3	18.4	203	16.1	8.8	23.2
Carbohydrate intake (g/day)						
Q1 (<173)	208	10.3	6.4	18.8	273	15.4	9.4	27.5
Q2 (173–240)	226	11.9	7.7	21.6	255	15.4	9.1	27.5
Q3 (241–335)	227	10.4	6.5	18.7	254	14.0	8.5	22.1
Q4 (>335)	289	11.5	7.5	18.5	192	14.9	8.8	22.8
Protein intake (g/day)						
Q1 (<49)	221	9.7	6.1	18.2	260	15.2	8.7	27.6
Q2 (49–70)	227	10.6	6.8	17.9	254	14.2	8.8	23.2
Q3 (71–100)	234	12.4	7.3	23.1	247	15.2	9.7	26.0
Q4 (>100)	268	11.7	7.4	18.6	213	14.6	8.7	22.0

	Black women			White women		
	N	Median	25th–75th percentile	N	Median	25th–75th percentile
Fiber intake (g/day)						
Q1 (<11)	220	10.0	6.8–17.6	261	15.0	8.3–24.1
Q2 (11–16)	227	10.6	6.9–19.9	254	14.4	9.2–24.3
Q3 (17–24)	229	12.9	7.0–20.5	252	15.4	9.5–28.8
Q4 (>24)	274	11.1	7.3–18.7	207	14.5	8.9–22.2
Alcohol consumption (drinks per day)						
0	496	10.9	7.1–19.7	591	14.0	8.6–23.5
1 – < 2	386	11.0	7.0–19.1	366	15.7	9.4–25.3
2+	111	10.5	6.4–18.7	37	18.2	13.2–36.5
Duration of cigarette smoking (years)						
Never smoker	414	10.6	6.8–18.0	328	14.7	8.8–25.6
<20	133	10.9	6.6–19.3	121	15.4	9.8–25.1
20–29	236	10.9	7.1–18.3	248	15.0	9.3–22.6
30+	209	12.6	7.2–22.5	293	14.7	8.9–26.2
Menopausal status						
Pre-	499	10.3	6.5–17.6	497	13.8	8.4–21.6
Post-	497	11.9	7.5–20.4	499	16.1	10.3–28.6
Number of live births						
None	101	12.4	7.2–22.8	96	15.3	9.1–28.6
1–2	332	11.7	7.2–20.6	477	15.0	8.7–23.2
3–4	358	10.2	6.5–18.4	323	14.5	9.6–25.2
5+	205	10.8	7.1–16.7	99	15.4	8.9–25.1
Age at menarche (years)						
<12	186	10.6	6.6–18.2	217	13.6	8.3–22.1
12	258	10.4	7.0–19.6	277	13.6	8.6–22.5
13	219	10.9	6.6–19.2	255	16.2	10.3–27.2
14	131	12.4	8.8–20.0	93	15.8	8.9–33.0
15+	197	10.8	7.0–19.3	147	15.8	9.6–24.3
Diabetes[a]						
Yes	220	10.0	6.2–19.8	164	13.0	7.5–22.1

	Black women			White women		
	N	Median	25th–75th percentile	N	Median	25th–75th percentile
No	776	11.0	7.1	19.0	832	15.3	9.3	25.1
Heart attack or coronary bypass surgery[a]						
Yes	41	12.3	8.6	23.8	51	15.0	9.8	21.5
No	955	10.9	6.9	18.9	945	14.9	9.0	25.0
Hypertension[a]						
Yes	600	10.7	6.5	18.7	462	13.3	7.9	22.5
No	396	11.5	7.7	19.7	534	16.2	9.9	25.5
High cholesterol[a]						
Yes	280	10.9	6.8	18.8	371	13.7	8.6	22.1
No	715	10.9	7.0	19.1	624	15.6	9.3	25.6
Depression[a]						
Yes	214	10.6	6.5	18.0	479	14.4	9.0	23.1
No	782	11.0	7.1	19.4	515	15.4	9.1	25.4

[a] Has a doctor ever told you that you have….?

Table 2

Linear Regression Results from Prediction Models[a] for Log-adiponectin (ug/ml) in 1,992 Black and White Women in the Southern Community Cohort Study, 2002–2006

Predictor	Black women				White women			
	Beta	Std err	p-value	Partial R^2	Beta	Std err	p-value	Partial R^2
Body Mass Index (kg/m^2)	−0.017	0.0036	<0.0001	0.05	−0.029	0.003	<0.0001	0.11
HDL-cholesterol (mg/dl)				0.10				0.10
Q1 (<43)	Referent				Referent			
Q2 (43–50)	0.17	0.067	0.01		0.29	0.053	<0.0001	
Q3 (51–60)	0.53	0.067	<0.0001		0.41	0.057	<0.0001	
Q4 (>60)	0.59	0.064	<0.0001		0.67	0.063	<0.0001	
Age at interview (years)	0.010	0.003	<0.0001	0.01	0.015	0.003	<0.0001	0.02
Hypertension	−0.085	0.046	0.07	0.003	−0.072	0.044	0.10	0.002
Adjusted model R^2				*0.17*				*0.27*

[a]Model estimates for the intercept and sample selection term are not shown