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Adiponectin is associated with risk of the metabolic syndrome and insulin resistance in women

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Abstract

The purpose of this study was to examine insulin resistance, markers of the metabolic syndrome, cardiovascular disease (CVD) risk, and serum adiponectin concentrations in pre-menopausal Hispanic and non-Hispanic White (NHW) women. This cross-sectional study examined 119 pre-menopausal women (76 Hispanic, 45 NHW) for markers of the metabolic syndrome (ATP III criteria), level of insulin resistance (HOMA-IR), CVD risk factors, and serum total adiponectin concentrations. Relationships between variables were assessed using Student's *t*-tests, Pearson's and Spearman's Rho correlations, and stepwise multiple regression analysis. Hispanic women had significantly lower adiponectin concentrations than NHW women, even after controlling for body fat (%) ($P < 0.01$). Number of markers of the metabolic syndrome was inversely related to total adiponectin concentration for all women combined and for NHW women ($P = 0.04$), but not for Hispanic women. Insulin resistance was inversely related to adiponectin for all women and for NHW women ($P < 0.01$), but not significantly associated in Hispanic women. Adiponectin concentration was not significantly associated with number of CVD risk factors for these women. While adiponectin was associated with markers of metabolic syndrome and insulin resistance for all women of this study and despite lower adiponectin concentrations for Hispanic women than NHW women, the role of adiponectin to these conditions among Hispanics remains unclear. There was no significant association between adiponectin and CVD risk for these women. Future research should focus on understanding mechanisms for up-regulating adiponectin secretion and if ethnicity affects adiponectin gene expression and secretion given the beneficial effects derived from elevated adiponectin levels.

Keywords

Hispanic; Pre-menopausal; HOMA; Adiponectin

Introduction

Adipose tissue is known to coordinate a broad range of metabolic, endocrine, and paracrine functions that have wide-ranging effects on food intake, energy expenditure, and carbohydrate/lipid metabolism [1, 2]. Adiponectin, discovered in 1995, is an adipocyte-secreted protein whose release is enhanced by insulin and was thought to be representative of the nutritional status of an organism [3]. Subsequent research has revealed that adiponectin (secreted exclusively by white adipose tissue) affects glucose homeostasis and insulin sensitivity and is significantly lower in type 2 diabetic, insulin resistant, and obese individuals [1, 2, 4, 5]. Individuals with high circulating adiponectin concentrations (meaning, higher than average values reported within the literature), regardless of body mass index (BMI), appear to be protected against obesity-related metabolic disorders [6, 7].

In addition to being protective against insulin resistance, adiponectin is believed to have protective effects against cardiovascular disease (CVD). In recent years, it has become apparent that endothelial dysfunction may be a contributor to the pathogenesis of vascular disease in type 2 diabetes [8]. Chronic hyperglycemia is a major contributor to atherosclerosis and microvascular disease thought to be accomplished through advanced glycation end products (AGEs) which increase arterial stiffness and affect myogenic regulation of blood flow [9]. Adiponectin has been shown to attenuate C-reactive protein and TNF- α release from adipose tissue; and in endothelial cells, adiponectin regulates interleukin-8, vascular cell adhesion molecule-1 (VCAM-1), and reactive oxygen species (ROS) through the cAMP-PKA-dependent pathway [10, 11]. Thus, adiponectin may exert some of its cardioprotective effects through the inhibition of cellular adhesion molecules and markers of inflammation.

The incidence of diabetes has increased in the United States by more than 3 million in approximately 2 years, now affecting almost 24 million people [12]. Another 57 million people are estimated to have pre-diabetes [12], a condition in which individuals have blood glucose levels higher than normal, but not high enough to be classified as diabetes [12]. Additionally, disparities exist among ethnic groups and minority populations including Native Americans, Blacks, and Hispanics. The rate for diagnosed diabetes in Hispanics is 10.4% compared to 6.6% in non-Hispanic Whites [12]. Diabetes is also associated with serious complications such as increased risk for heart disease, stroke, high blood pressure, and endothelial dysfunction. The estimated total direct and indirect costs of diabetes were approximately \$174 billion in 2007 [12].

The purpose of this cross-sectional study was to examine adiponectin levels in both Hispanic and non-Hispanic White (NHW) pre-menopausal women. We also aimed to determine the association between insulin resistance, markers of the metabolic syndrome, CVD risk factors, and adiponectin levels in these groups. We hypothesized that adiponectin levels

would be lower in the Hispanic women, potentially pre-disposing them to the development of metabolic diseases.

Research design and methods

Participants

Seventy-four Hispanic and 45 NHW pre-menopausal women between the ages of 35 and 50 years volunteered for this study. Participants were included if they reported that both parents and three of four grandparents were of the same ancestry (either Hispanic or NHW). Prior to data collection, each participant signed an informed consent form approved by the Institutional Review Board of the University of Texas at El Paso. Women were excluded from the study for pregnancy, currently nursing, irregular menstrual cycles, amenorrhea, or any known metabolic disease.

Study protocol

Each participant reported to the laboratory between 0600 and 0800 h, at least 12 h post-prandial, and following 6–8 h of sleep. Body mass was measured to the nearest 0.01 kg using a calibrated load cell scale (Tanita Corporation, Tokyo, Japan); height was measured to the nearest 0.1 cm using a stadiometer (Seca Corp., Germany); and BMI (kg/m^2) was calculated. A fasting blood sample was collected followed by measurement of waist and hip circumference, body composition assessment by dual-energy x-ray absorptiometry (DXA), and three supine blood pressure measurements. All laboratory procedures were completed during a single testing session, and each woman was asked to void prior to body composition assessments.

Body composition assessment

For each woman, body composition (DXA-BF) was assessed using DXA (Lunar DPX-NT, GE Lunar Corp., Madison, WI). All measurements were performed in accordance with manufacturer specifications. Women were asked to remove all jewelry and other accessories and were measured in a standard set of gym shorts and t-shirt that were provided by the investigators. A quality assurance test (QA), which calibrates and verifies the correct operation of the densitometer, was performed at the start of each testing day to examine the functionality, accuracy, and precision of the system. The coefficient of variance (CV%) for the DXA system used was 0.23% based on 254 QA test procedures and control measurements.

Blood sample analysis

A fasting blood sample was collected by venipuncture from an antecubital vein into a blank serum vacuum tube for each participant. The blood sample was allowed to clot, centrifuged for 20 min, and serum aliquots were separated into cryovials (Wheaton, Millville, NJ) and frozen at -80°C until analyzed for adiponectin and insulin. Serum total adiponectin concentration ($\mu\text{g/mL}$) was determined by ELISA (Linco, St. Charles, MO) (intra-assay CV %: 7.4, 0.9, and 1.8%; inter-assay CV%: 8.4, 2.4, and 6.2% for low, medium, and high adiponectin standards, respectively) and serum insulin concentration was determined by EIA (LDN, Nordhorn, Germany) (intra-assay CV%: 5.3 and 3.0%; and inter-assay CV%: 9.5 and

4.5% for low and high insulin standards, respectively). Absorbance was assessed using a microtiter plate reader (SpectraMAX 190, Molecular Devices, Sunnyvale, CA). All samples were measured in duplicate, and the average of the two measures was recorded as the adiponectin or insulin concentration.

Serum triglycerides (TG), fasting glucose, and lipid profile were assessed using an automated analyzer (Cholestech LDX, Hayward, CA). The optical system (CV%: <0.85% for the four channels) and the calibration of the instrument were verified prior to each use, using two calibrator standards of known concentration (CV% for all analytes: <6.3% and <9.9% for low and high controls, respectively).

Metabolic syndrome and cardiovascular disease risk assessment

The number of markers of the metabolic syndrome was assessed using the Adult Treatment Panel III (ATP III) Criteria from the National Cholesterol Education Program. Briefly, for women, the risk factors include: (1) a waist circumference ≥ 88 cm; (2) systolic blood pressure ≥ 130 mmHg and/or diastolic blood pressure ≥ 85 mmHg; (3) high-density lipoprotein (HDL) cholesterol <50 mg/dL; (4) total triglycerides ≥ 150 mg/dL; and (5) fasting blood glucose ≥ 100 mg/dL. Individuals with three or more of the above criteria are classified as having the metabolic syndrome, while individuals with two of the above criteria, we defined as borderline metabolic syndrome.

For CVD risk, the number of markers was assessed as defined by the American College of Sports Medicine (ACSM) guidelines [13]. A family history of CVD, smoking history, and physical activity history were assessed by self-report using both a questionnaire and interview by a researcher. Objective markers for CVD risk were hypertension (blood pressure $>140/90$ mmHg), dyslipidemia (total cholesterol >200 mg/dL; LDL >130 mg/dL; and/or HDL <40 mg/dL), impaired fasting glucose (fasting blood glucose >100 mg/dL), and obesity (BMI >30 kg/m²; waist circumference >88 cm; and/or waist: hip ratio >0.86) [13]. Women in this study were classified as low risk for development of cardiovascular disease if they had less than 2 markers and were classified as moderate risk if they displayed two or more risk factors [13].

Insulin resistance was estimated by the Homeostatic Model for the Assessment of Insulin Resistance (HOMA-IR) using fasting serum insulin and glucose concentrations [14].

Statistical analysis

Statistical analyses were conducted using the software package SPSS v16.0 (SPSS, Inc., Chicago, IL). The normal distribution of variables was checked with histograms and Kolmogorov–Smirnov’s test. Preliminary analyses for violations of the assumption of normality, linearity, and homoscedasticity revealed that serum TG, HOMA-IR, and serum total adiponectin were not normally distributed. A Log₁₀ transformation was used to transform TG ($P = 0.03$), HOMA-IR ($P = 0.18$), and adiponectin ($P > 0.20$) data to reflect normality.

Descriptive data were compared between groups using an independent samples *t*-test. A one-way between-groups analysis of covariance was conducted to examine adiponectin levels

between NHW and Hispanic women while controlling for percent body fat. Preliminary checks were done to ensure there were no violations of the assumptions of normality, linearity, homogeneity of variances, homogeneity of regression slopes, and reliable measure of the covariate. Pearson's and Spearman's Rho correlation coefficients were calculated to examine relationships between adiponectin, insulin resistance, markers of the metabolic syndrome, and CVD risk factors. Multiple stepwise linear regression was used to identify those variables with the strongest associative influence on adiponectin. Collinearity between independent variables was assessed using variance inflation factor and a value above 10.0 indicated multicollinearity. Potential outliers were identified by Mahalanobis distances with a critical value of 24.32. Significance was set at an alpha level of <0.05 .

Results

A total of 119 pre-menopausal women (45 NHW and 74 Hispanic) volunteered to participate in this study. An independent samples *t*-test was conducted to compare clinical and biochemical markers between NHW and Hispanic women (Table 1). Hispanic women were of significantly shorter stature ($P < 0.001$) and had a greater body fat percentage ($P = 0.01$) than NHW women. The one-way between-groups analysis of covariance revealed that after adjusting for percent body fat, Hispanic women had significantly lower adiponectin concentrations than NHW women ($P = 0.002$), with approximately 8% of the variance in adiponectin being explained by ethnicity (partial eta squared = 0.08). Although there was no difference in fasting blood glucose ($P = 0.23$) between NHW and Hispanic women, Hispanic women had significantly greater insulin resistance, as estimated from HOMA-IR ($P = 0.01$). Approximately 6% of the variance within HOMA-IR was explained by ethnicity for this group of women (eta squared = 0.06).

The ATP III criteria markers were used for determining risk for development of metabolic syndrome. Individuals with 3 or more markers were classified as having metabolic syndrome, while individuals with 2 markers were classified as borderline metabolic syndrome. Overall, 21 women (6 NHW, 15 Hispanic) from this study were classified as having metabolic syndrome, while 16 women (5 NHW, 11 Hispanic) were borderline. We elected to analyze the number of markers (0–4 markers) rather than the metabolic syndrome category (no, borderline, or metabolic syndrome) to allow for greater resolution of the risk continuum (5 marker levels vs. 3 disease categories). When examining the number of markers overall, Hispanic women, on average, had significantly more markers than NHW women ($P = 0.037$). When examining differences in individual markers of the metabolic syndrome between the two ethnicities, the only significant difference was in waist circumference ($P = 0.01$), suggesting that Hispanic women had more visceral adiposity than the NHW women. To further evaluate this, an independent samples *t*-test revealed that Hispanic women had significantly greater trunk fat (kg) determined by DXA than NHW women ($P = 0.022$). Similar to metabolic syndrome markers, we again elected to analyze the number of risk factors (0–3 + risk factors) rather than CVD risk category (low or moderate risk) to allow for greater resolution of the risk continuum (4 risk factor levels vs. 2 risk categories). There was no significant difference in the number of CVD risk factors between the Hispanic and NHW women.

The relationship between adiponectin and markers of metabolic and CVD risk can be seen in Table 2. Overall, adiponectin was inversely related to markers of adiposity, fasting glucose, and insulin resistance, while positively associated with HDL cholesterol. However, for Hispanic women, adiponectin was inversely correlated with waist-to-hip ratio and positively correlated with HDL cholesterol, while no significant association was seen with the other markers. Adiponectin was inversely related to the number of markers of the metabolic syndrome (Fig. 1) for all the women combined ($\rho = -0.30$, $P = 0.001$) and for the NHW women ($\rho = -0.31$, $P = 0.040$). There was no significant relationship between adiponectin and markers of the metabolic syndrome for Hispanic women ($\rho = -0.16$, $P = 0.17$). Total adiponectin was not significantly related to CVD risk (Fig. 2) for either NHW ($\rho = -0.15$, $P = 0.110$) or Hispanic women ($\rho = -0.09$, $P = 0.463$) or for the group overall ($\rho = -0.15$, $P = 0.110$).

A one-way between-groups analysis of variance was conducted to explore the impact of adiponectin on waist circumference, waist-to-hip ratio, HDL cholesterol, fasting blood glucose, triglyceride concentration, and insulin resistance (HOMA-IR). All participants were divided into tertiles according to adiponectin concentration. There was a statistically significant difference in waist circumference, waist-to-hip ratio, HDL cholesterol, and level of insulin resistance (HOMA-IR) for the three adiponectin tertiles (Table 3). The effect size, calculated using eta squared, was large for waist-to-hip ratio and HDL cholesterol concentration, suggesting that these differences may be clinically relevant. A Kruskal–Wallis test revealed a statistically significant difference in the number of markers of the metabolic syndrome across adiponectin tertiles ($\chi^2 = 9.37$, $df = 2$, $P = 0.009$). The individuals with the lowest measured adiponectin concentrations (1st tertile) had the greatest number of metabolic syndrome markers. There was no significant difference in the number of CVD risk markers across adiponectin tertiles ($\chi^2 = 4.16$, $df = 2$, $P = 0.125$).

A stepwise multiple regression was performed to determine which variables had the strongest association with total adiponectin concentration for all women combined. Body mass index, waist circumference, waist-to-hip ratio, HDL cholesterol, triglycerides, fasting glucose, and insulin resistance were the independent variables. The R for regression (0.50) was significantly different from zero ($P < 0.001$), with R^2 at 0.25 indicating that approximately one-quarter of the variability in adiponectin is predicted by waist-to-hip ratio, HDL cholesterol, and fasting glucose (Table 4). HDL cholesterol made the strongest unique contribution to the model (~26%) and accounted for approximately 5% of the total variance in adiponectin that is uniquely explained by HDL levels.

Discussion

The purpose of this study was to examine adiponectin levels in both NHW and Hispanic premenopausal women as well as determine associations of adiponectin with insulin resistance, markers of the metabolic syndrome, and CVD risk factors. The major findings of our study were that adiponectin concentrations were lower in these Hispanic women and that for all women, adiponectin was inversely related to insulin resistance and risk for the metabolic syndrome. However, within our data, there was no association between adiponectin and markers of CVD risk.

Adiponectin, a 30-kDa protein secreted exclusively by white adipose tissue, appears to be associated with lower risk for the development of insulin resistance, type 2 diabetes, and other metabolic consequences of obesity [3, 5, 6, 15]. Certain ethnic groups have a greater predisposition to develop these diseases compared to their NHW counterparts. Specifically, Hispanic adults have an increased prevalence of the metabolic syndrome and type 2 diabetes compared to NHW adults [16]. Hispanics also have an increased prevalence of overweight and obesity compared to NHW adults, and recent data indicates these ethnic disparities are more prevalent among women than men, with Mexican-American women much more likely than NHW women to be obese [17]. In our sample, Hispanic pre-menopausal women had lower total adiponectin concentrations (~25%) than NHW pre-menopausal women, even after controlling for differences in percentage body fat between the two groups. Low adiponectin concentrations have been reported in the Arizona Pima Indians [18], a group characterized by very high rates of obesity, insulin resistance, and type 2 diabetes. African-American obese and non-obese women also have lower adiponectin concentrations than Caucasian non-obese women; however, non-obese African-American, obese African-American, and obese Caucasian women had similar adiponectin concentrations [19]. Caucasians were also reported to have higher baseline adiponectin concentrations than African-American and Hispanic participants in the Diabetes Prevention Program [20]. Hispanics were found to have higher adiponectin concentrations than African-American adults in a large, cross-sectional analysis of data collected in the IRAS Family Study [21]. However, when controlled for visceral adipose tissue, there was no significant difference in adiponectin concentration between the two groups [21]. The combined results of these studies [20, 21] and ours may indicate why there is an increased prevalence of insulin resistance and type 2 diabetes among African-Americans and Hispanics, given that low adiponectin concentrations are associated with increased risk for development of these diseases.

Hispanic women have the highest prevalence of the metabolic syndrome compared to other ethnic groups [16]. Additionally, insulin resistance is thought to be highly associated with the development of the metabolic syndrome. The Hispanic women of our study had a significantly greater degree of insulin resistance (estimated by HOMA-IR) and, on average, had more metabolic syndrome markers than the NHW women, suggesting that Hispanic women may be at higher risk for development of the metabolic syndrome. Additionally, our results demonstrated statistically significant negative correlations between adiponectin, BMI, waist circumference, fasting glucose, and insulin resistance for the NHW women, but there were no significant correlations between these variables for the Hispanic women (Table 2). Although the relationship between adiponectin and insulin sensitivity has been well documented, these data suggest that this relationship is not consistent across ethnicities. Hulver et al. [19] also found no significant association between adiponectin and BMI, insulin concentration, or insulin resistance (HOMA-IR) within their African-American participants and suggested that population-specific criteria may be necessary for assessing disease risk in ethnic populations. The Hispanic women in our study had a significantly greater waist circumference than their NHW counterparts. Our trunk fat measurement encompasses tissue between the neck and pelvis and does not solely estimate abdominal fat. Because these Hispanic women had significantly greater trunk fat and waist circumference

than the NHW women, it is reasonable to infer that our Hispanic women had greater abdominal adiposity. Obesity is one of the defining criteria of the metabolic syndrome, and the use of waist circumference measurements over BMI or total body fat measurements in determining risk may be advantageous because abdominal fat is more closely correlated to metabolic disease than total body fat [22, 23]. For our women, insulin resistance was significantly correlated to waist circumference and waist-to-hip ratio for both NHW and Hispanic women (data not shown). Hanley et al. [21] also showed that Hispanics had greater visceral adipose tissue than African-Americans and that there was no difference in adiponectin concentrations between these groups when visceral adipose tissue was controlled. Increased visceral adiposity is also associated with an increase in metabolic risk factors including dyslipidemia, elevated free fatty acids, and elevated clinical markers of inflammation [24]. Therefore, it may be possible to suggest that the increased incidence of the metabolic syndrome in Hispanics is largely due to the higher amounts of visceral adipose tissue found in this population.

The relationship between adiponectin and CVD risk has been well studied, but with disparate results [10, 25–31]. In general, higher adiponectin concentrations are associated with a decreased risk for development of CVD [10]. We found no relationship between adiponectin and the number of CVD risk markers for either the Hispanic or NHW women. Laughlin et al. [27] found no association between adiponectin and cardiovascular events in women and a weak association between the two variables among men. No association between adiponectin and CVD was also reported from a 4-year follow-up of cardiovascular events in the Strong Heart Study [32] and in the British Women's Heart and Health Study [33]. However, adiponectin was positively correlated with HDL cholesterol in both the Hispanic and NHW women of our study. This result corresponds well with previous literature which suggested a strong positive association between adiponectin and HDL levels [25–27, 29–31]. It is possible then that the anti-atherogenic property of adiponectin may be partially attributed to the action of HDL in reverse cholesterol transport [34].

This study has several limitations that should be considered. First, we used an indirect estimate of insulin resistance with the HOMA-IR model. It is possible that a direct measurement of insulin resistance by the euglycemic hyperinsulinemic clamp method may have yielded different associations with adiponectin. However, the HOMA-IR model is widely used by researchers and has acceptable validity for estimating insulin resistance [14]. Further, we measured only total adiponectin concentration. The high molecular weight form of adiponectin has a stronger inverse association with insulin resistance and positive association with HDL cholesterol concentrations [10]. Therefore, had we determined the high molecular weight form of adiponectin, we may have observed a significant negative relationship with insulin resistance for our Hispanic women. Finally, this study is cross-sectional in nature, and as such, we cannot develop conclusions about adiponectin concentrations and disease development. However, several longitudinal studies in both animal models and humans have shown that with low adiponectin concentration, there is a greater prevalence of metabolic and cardiovascular diseases [1, 6, 7, 10, 11, 25].

Additionally, several of the 'common' relationships seen between adiponectin and metabolic diseases and biomarkers were observed within our NHW population, but lost within our

Hispanic population. Although commonly used as a racial descriptor, Hispanic is an ethnic term and describes a heterogeneous population comprised of Caucasian (European), Native American, and West-African ancestry. Recent developments in genome mapping now allow for identification of disease-causing variants within a population (admixture mapping) [35]. The use of admixture mapping can allow for the characterization of the heterogeneity of the Hispanic population. Knowing that Native Americans and African-Americans have a greater incidence of diabetes compared to NHW [12], it could be theorized that Hispanics with closer ancestry to Native Americans are at an increased risk for development of diabetes and other metabolic diseases compared to Hispanics with more European ancestry. Our study did verify Hispanic ancestry through self-report but showed no relationship between adiponectin and insulin resistance, metabolic syndrome, or CVD risk. Similar to most survey information, our questionnaire was limited by the women's subjective self-identification of ethnicity as opposed to genetically determined racial characteristics. A study's inclusion of admixture mapping will greatly enhance our understanding of ethnic health disparities.

In conclusion, adiponectin was found to be positively associated with HDL cholesterol in both Hispanic and NHW women. In NHW women only, adiponectin was inversely associated with insulin resistance and number of markers of the metabolic syndrome. For all women, those with the lowest adiponectin concentration (1st tertile) had the greatest number of markers of the metabolic syndrome. This study has also shown that Hispanic pre-menopausal women have lower fasting total adiponectin concentrations than their NHW counterparts, potentially increasing the risk in Hispanic women for development of the metabolic syndrome and type 2 diabetes. Interventions designed to up-regulate adiponectin secretion or gene expression may be beneficial within populations at higher risk for development of the metabolic syndrome and type 2 diabetes.

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Fig. 1.

The number of markers of the metabolic syndrome per person was inversely related to total adiponectin concentration for non-Hispanic White women ($\rho = -0.31$, $P = 0.04$), but not for Hispanic women ($\rho = -0.16$, $P = 0.17$). With all women combined, there was a significant inverse correlation between adiponectin and number of markers of the metabolic syndrome ($\rho = -0.30$, $P < 0.01$)

Fig. 2.

The number of cardiovascular disease (CVD) risk factors was not significantly related to total adiponectin concentration for all of these women ($\rho = -0.15$, $P = 0.11$), non-Hispanic White women ($\rho = -0.16$, $P = -0.31$), or Hispanic women ($\rho = -0.09$, $P = 0.46$)

Table 1
Descriptive clinical and biochemical data for pre-menopausal Hispanic and non-Hispanic White women

	Overall			Non-Hispanic White			Hispanic			P-value
	N	Mean	SE	N	Mean	SE	N	Mean	SE	
Age (y)	119	43.09	± 0.41	45	43.63	± 0.63	74	42.76	± 0.54	0.31
Height (cm)	119	162.9	± 0.6	45	165.5	± 0.9	74	161.3	± 0.7	<0.01
Weight (kg)	119	68.41	± 1.29	45	66.36	± 1.84	74	69.65	± 1.74	0.22
BMI (kg/m ²)	119	25.77	± 0.46	45	24.18	± 0.60	74	26.73	± 0.62	0.01
Body Fat (%)	119	40.18	± 0.77	45	37.58	± 1.22	74	41.77	± 0.95	0.01
Waist circumference (cm)	119	80.5	± 1.1	45	76.9	± 1.6	74	82.7	± 1.4	0.01
Hip circumference (cm)	119	104.5	± 1.0	45	102.9	± 1.3	74	105.4	± 1.4	0.22
Waist: hip ratio	119	0.77	± 0.01	45	0.75	± 0.01	74	0.78	± 0.01	<0.01
Systolic BP (mm Hg)	119	119	± 1	45	117	± 2	74	120	± 2	0.35
Diastolic BP (mm Hg)	119	78	± 1	45	77	± 1	74	78	± 1	0.51
Total cholesterol (mg/dL)	119	207.92	± 4.01	45	217.00	± 6.25	74	202.39	± 5.13	0.07
HDL cholesterol (mg/dL)	118	59.01	± 1.36	44	62.75	± 2.11	74	56.78	± 1.74	0.03
LDL cholesterol (mg/dL)	114	127.15	± 3.65	41	131.93	± 6.11	73	124.47	± 4.55	0.33
VLDL cholesterol (mg/dL)	108	22.56	± 1.15	39	20.92	± 1.57	69	23.48	± 1.57	0.29
Triglycerides (mg/dL)	119	117.86	± 8.05	45	119.16	± 16.21	74	117.07	± 8.48	0.62
Fasting blood glucose (mg/dL)	119	85.87	± 1.36	45	83.76	± 1.42	74	87.16	± 2.00	0.23
Fasting insulin (pmol/L)	119	108.80	± 6.49	45	89.17	± 5.22	74	120.75	± 9.72	<0.01
HOMA-IR	119	3.40	± 0.22	45	2.69	± 0.18	74	3.83	± 0.33	0.01
Total adiponectin (µg/mL)	119	10.55	± 0.41	45	12.37	± 0.68	74	9.45	± 0.47	<0.01*

P-values reflect independent *t*-test comparisons made between non-Hispanic White and Hispanic women

* Adiponectin concentrations between groups were examined by one-way ANCOVA controlling for body fat (%)

Pearson product correlations between adiponectin and markers of metabolic and cardiovascular disease risk for pre-menopausal Hispanic and non-Hispanic White women

Table 2

Variable	Overall (N = 119)		Non-Hispanic White (N = 45)		Hispanic (N = 74)	
	r	P-value	r	P-value	r	P-value
BMI	-0.280	0.002	-0.412	0.002	-0.134	0.253
Waist circumference	-0.337	<0.001	-0.430	0.003	-0.203	0.083
Waist: hip ratio	-0.402	<0.001	-0.326	0.029	-0.340	0.003
Total cholesterol	0.033	0.723	-0.192	0.206	0.080	0.499
HDL cholesterol	0.396	<0.001	0.359	0.017	0.358	0.002
LDL cholesterol	-0.052	0.584	-0.175	0.273	-0.034	0.772
Fasting glucose	-0.273	0.003	0.360	0.015	-0.228	0.051
HOMA-IR	-0.308	0.001	-0.439	0.003	-0.194	0.100

Metabolic risk defined by Adult Treatment Panel III (ATP III) Criteria from the National Cholesterol Education Program; cardiovascular disease risk defined by ACSM's Guidelines for Exercise Testing and Prescription, 7 ed

Table 3

One-way ANOVA to determine impact of adiponectin concentration on markers of metabolic and cardiovascular disease risk of pre-menopausal Hispanic and non-Hispanic White women

Variable	Adiponectin tertile			P-value
	1st (Low)	2nd	3rd (High)	
Adiponectin (µg/mL)	6.19 ± 0.21	9.93 ± 0.19	15.63 ± 0.56	–
Waist circumference (cm)	83.59 ± 1.84	82.97 ± 1.88	75.15 ± 1.48 ^{†‡}	0.001
Waist-to-hip ratio	0.78 ± 0.01	0.78 ± 0.01	0.74 ± 0.01 ^{†‡}	<0.001
HDL cholesterol (mg/dL)	52.55 ± 2.44	57.57 ± 1.71	66.57 ± 2.37 ^{†‡}	<0.001
Triglycerides (mg/dL)	133.41 ± 17.54	128.30 ± 14.56	92.25 ± 7.27	0.057
Glucose (mg/dL)	89.46 ± 3.33	86.27 ± 1.70	81.97 ± 1.59	0.079
HOMA-IR	3.79 ± 0.33	3.60 ± 0.33	2.38 ± 0.15 [†]	0.010

Metabolic risk defined by Adult Treatment Panel III (ATP III) Criteria from the National Cholesterol Education Program; cardiovascular disease risk defined by ACSM's Guidelines for Exercise Testing and Prescription, 7 ed

Values presented are mean ± SE. Post hoc comparisons were calculated using Tukey's HSD test Stated P-value describes the main effect

[†]Significantly different from 1st tertile

[‡]Significantly different from 2nd tertile

Table 4

Stepwise multiple regression examining predictors of adiponectin among pre-menopausal Hispanic and non-Hispanic White women

Dependant variable	Independent variable	R^2	Beta	P-value
Adiponectin	Waist-to-hip	0.250	-0.251	0.007
	HDL		0.257	0.005
	Glucose		-0.176	0.037

Variables entered into the model: waist-to-hip ratio, high-density lipoprotein (HDL) cholesterol, fasting blood glucose, body mass index, waist circumference, insulin resistance (HOMA-IR), triglycerides