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Racial/ethnic and sex differences in the ability of metabolic syndrome criteria to predict elevations in fasting insulin in adolescents

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Abstract

Objective—To evaluate racial/ethnic and sex differences in the relationship between metabolic syndrome (MetS) diagnosis and fasting insulin among adolescents.

Study design—We analyzed data from the National Health and Nutrition Evaluation Survey 1999–2008 for 3,693 non-Hispanic-white, non-Hispanic-black and Hispanic adolescents (12–19y). We used linear regression to evaluate differences in fasting insulin between those with and without an adolescent adaptation of ATPIII-MetS in a sex- and race/ethnicity-specific basis.

Results—Females had higher insulin levels than males and non-Hispanic blacks and Hispanics had higher levels than non-Hispanic whites. Adolescents with MetS had higher insulin levels than those without MetS. The difference in insulin levels between those with and without MetS was greater among non-Hispanic blacks compared with non-Hispanic whites ($p < 0.05$) but not Hispanics ($p = 0.10$). The sensitivity of MetS for detecting elevated insulin levels was lower among non-Hispanic blacks and females compared with other ethnicities and males, respectively. Correlations between insulin and individual MetS components were similar among ethnicities.

Conclusion—MetS diagnosis performed more poorly in predicting elevated insulin levels among non-Hispanic blacks and among females. These data support the hypothesis that non-Hispanic blacks do not meet current criteria for MetS until they have reached a more advanced degree of insulin resistance.

Keywords

Metabolic syndrome; ethnicity; insulin resistance; adolescents

The metabolic syndrome (MetS) was first described as a cluster of clinical findings that are strongly associated with insulin resistance (1, 2). Nevertheless, measurements of insulin itself are not a criterion for diagnosis of MetS, which is instead based on specific cut-off values used to identify elevated waste circumference (WC), hypertension, low HDL

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cholesterol (HDL-C), hypertriglyceridemia, and elevated fasting glucose (1, 2). Although multiple sets of criteria have been proposed for classifying MetS in children—including one by the International Diabetes Federation (3–5)—the majority of research in this area has utilized criteria based on the adult ATP III criteria (2, 6, 7), with cut-off levels adjusted to be more consistent with adolescent ranges (1, 8–10). The utility of MetS as a clinical tool in pediatrics has been shown in that children with MetS have an odds ratio of 11.5 for developing type 2 diabetes (T2DM) within 30 years (10), and in adults MetS predicts earlier cardiovascular disease (6). This makes a diagnosis of MetS an appealing trigger for early intervention among at-risk youths (5).

Non-Hispanic-black adolescents have lower rates of MetS diagnosis than Hispanics and non-Hispanic whites (4, 7, 9) despite having a higher degree of insulin resistance than seen in non-Hispanic whites (11, 12) and higher rates of T2DM and CVD, as adults (13, 14). Similarly, adolescent females have lower rates of MetS (4, 9) and comparisons of insulin resistance between sexes have been mixed (12, 15). Thus, the value of MetS in identifying females and non-Hispanic-black adolescents with insulin resistance is unclear.

Our goal was to determine whether the relationship between insulin resistance and MetS varied by sex and race/ethnicity among non-Hispanic-white, non-Hispanic-black, and Hispanic adolescents (12–19 y.o.) who participated in the National Health and Nutrition Evaluation Survey (NHANES) '99-'08. On a sex- and race/ethnicity-specific basis, we compared: (1) the degree of insulin resistance (estimated as either levels of fasting insulin or the homeostasis model of insulin resistance, HOMA-IR) in the presence and absence of MetS; and (2) the sensitivity of a MetS diagnosis to detect elevations in fasting insulin. In doing so, we aimed to identify sex- and racial/ethnic variation in the ability of a diagnosis of MetS to identify insulin resistance in adolescents.

Methods

Data were obtained from NHANES (1999–2008), a complex, multistage probability sample of the US population. These annual cross-sectional surveys are conducted by the National Center for Health Statistics (NCHS) of the Centers for Disease Control (CDC), with subjects undergoing anthropometric and blood pressure measurements, answering questionnaires and undergoing phlebotomy (<http://www.cdc.gov/nchs/nhanes.htm>). The NCHS ethics review board reviewed and approved the survey and participants were provided with informed consent prior to participation. WC, blood pressure (BP), and laboratory measures of triglycerides, HDL-C, and glucose were obtained using standardized protocols and calibrated equipment (1, 16). All blood samples used for analyses were obtained from participants asked to fast ≥ 8 hours prior to the blood draw.

MetS was defined by a commonly-used pediatric/adolescent adaptation of the ATP III criteria (1, 8, 9). Participants had to meet ≥ 3 of the following 5 criteria: concentration of triglycerides ≥ 110 mg/dL, HDL-C ≤ 40 mg/dL, WC ≥ 90 th percentile for age/sex (or ATP III limit of 102 cm for males and 88 cm for females, whichever was lower)(2, 17), glucose concentration ≥ 100 mg/dL, and systolic or diastolic BP ≥ 90 th percentile (age, height, and sex-specific)(18).

Data from non-Hispanic-white, non-Hispanic-black, or Hispanic (Mexican-American/other Hispanic) adolescents 12–19y were analyzed. Children <12 y were excluded because fasting values for triglycerides and glucose were only obtained in participants ≥ 12 y. Subjects were excluded if they had known diabetes or unknown diabetes (fasting glucose >125 mg/dL), as each of these can result in limitations in insulin release (19), which would confound our evaluation of the relationships between MetS and insulin levels. Also excluded were

pregnant women, and individuals taking antihyperlipidemic or anti-diabetic medications as these are all likely to alter lipid and insulin levels in a manner that may not reflect baseline MetS-insulin correlations. Individuals taking anti-hypertensive medication were classified as having hypertension.

“High” fasting insulin levels were determined to be the 95th percentile of insulin among normal weight individuals (BMI<85th percentile) in the sample (16 IU/mL), given the influence of highly-prevalent obesity on “normal” insulin values overall. The homeostasis model of insulin resistance (HOMA-IR) was calculated as [(fasting insulin in mU/mL)xfasting glucose in mmol/L/22.5].

Statistical Analysis

Statistical significance was defined as a p-value<0.05. Statistical analysis was performed using SAS (version 9.2, Cary, NC) and SUDAAN (version 10; Research Triangle Institute, Research Triangle Park, NC), which accounts for the survey design when estimating standard errors to obtain population-based estimates. We combined all data sets from the 4 two-year cycles (1999–2008) for statistical analyses to increase our total sample size. Prevalence of MetS was calculated by sex, race/ethnicity, and time period of data collection and compared via chi-square tests. Mean fasting insulin was also compared among these groups using either unpaired t-tests or ANOVA. Pearson’s r correlation coefficients were computed to assess the degree of linear association between each MetS component and ln(insulin), by race/ethnicity. Linear regression was then used to assess the effect of sex, race/ethnicity, and MetS status on levels of ln(insulin). The natural log transformation of fasting insulin was used to achieve normality. All interactions of the three covariates (sex, race/ethnicity, and MetS status) were initially included in the model, but removed in a stepwise fashion if the associated interaction p-value was <0.15. Due to the known effects of poverty (20), education (20) and smoking (21) on insulin levels, each of these covariates were included in the model. Education was classified as the highest level obtained for any household member and categorized as follows: less than high school, high school, and greater than high school. Income-to-need ratio was used to measure poverty. Due to the poor reliability of self-reporting of smoking among adolescents (22), serum cotinine was used to identify smokers, with a cut-off of 15 ng/mL as recommended (23). Geometric means of fasting insulin from the final model were estimated and compared among sex and race/ethnicity, as applicable. Sensitivity of MetS to identify elevated fasting insulin (16 IU/mL, the 95th percentile among normal-weight adolescents) were computed by sex and race/ethnicity. With the exception of the correlation estimates, all analyses incorporated the sampling weights included in NHANES.

Results

The sample of participants consisted of 3,693 non-Hispanic blacks, non-Hispanic whites, and Hispanics age 12–19 y.o. with data for all variables tested, excluding 119 subjects excluded based on the criteria listed above. Analyzing Mexican Americans as a separate group did not yield different results than when combined with other Hispanics. Among US adolescents the prevalence of MetS was greater among males compared with females (11.1% vs. 5.9%, $p<0.05$) and among non-Hispanic whites and Hispanics compared with non-Hispanic blacks (8.8% and 11.2% vs. 4.7%, both $p<0.05$) (Table I; available at www.jpeds.com). The prevalence of MetS did not differ over the test period and there were no consistent trends in MetS-associated variables over the study period (data not shown).

Individual components of MetS are shown in Table I. Compared with non-Hispanic whites and Hispanics, non-Hispanic blacks had lower triglycerides and fasting glucose and higher

HDL and SBP. Hispanics had the highest WC but otherwise did not exhibit differences in individual MetS components compared with non-Hispanic whites.

Insulin levels were higher in adolescent females than males (11.3 vs. 10.5 IU/mL, $p<0.05$) and were higher in non-Hispanic blacks and Hispanics than non-Hispanic whites (12.3 and 12.5 UI/mL vs. 10.1 IU/mL, both $p<0.05$)(Table I). Similarly, HOMA-IR values were higher in non-Hispanic blacks compared with Hispanics and non-Hispanic whites (2.9 and 3.0 vs. 2.4, both $p<0.05$); however, HOMA-IR levels were not significantly different between females and males.

Covariates in the linear model of $\ln(\text{insulin})$ are shown in Table II and geometric means of insulin levels by sex and ethnicity (generated from the model) are shown in Figure 1, A–B. Household education level and smoking were significantly associated with levels of insulin as shown previously (20, 21). Two pairwise interactions were significant and thus remained in the model: MetS x sex ($p=0.06$) and MetS x ethnicity ($p=0.11$). Both non-Hispanic blacks and Hispanics had higher insulin levels than non-Hispanic whites ($p<0.01$ and $p=0.05$, respectively). Across both sex and ethnicity adolescents with MetS exhibited higher insulin values compared with those without ($p<0.01$ for each sex/ethnicity combination, Figure 1, A–B). However, the increase in insulin associated with having MetS was significantly greater for non-Hispanic blacks compared with non-Hispanic whites ($p<0.05$), but not Hispanics ($p=0.10$) for both sexes (Figure 1, C). As was true for overall insulin levels (Table I), females without MetS had overall significantly higher values of insulin compared with males without MetS, a finding which did not vary among ethnicities ($p<0.05$)(Figure 1, A–B). Nevertheless, among adolescents with MetS, males and females across ethnicities had nearly identical levels of insulin (ratio of geometric means comparing males with females =1.01, 95% CI: (0.83, 1.23); $p=0.91$). Thus, there was a trend toward a larger increase in insulin associated with MetS for males compared with females across all ethnicities ($p=0.06$). In substituting HOMA-IR for insulin, the differences between ethnic and sex groups were nearly identical to that found using fasting insulin alone (data not shown).

The sensitivity of a diagnosis of MetS for detecting an elevated fasting insulin (16 IU/mL, the 95th percentile among normal-weight adolescents) by sex and race/ethnicity is shown in Figure 2, A–B. Among adolescent males, a MetS diagnosis displayed a decreased sensitivity for detecting an elevated fasting insulin among non-Hispanic blacks compared with both non-Hispanic whites, Hispanics and those two groups combined (all $p<0.05$). Among adolescent females, a MetS diagnosis had a lower sensitivity for detecting elevated insulin among non-Hispanic blacks compared with Hispanics ($p<0.05$), with a non-significant trend when compared with non-Hispanic whites ($p=0.13$) or non-Hispanic whites and Hispanics combined ($p=0.05$). A MetS diagnosis displayed a decreased sensitivity for detecting elevated insulin among females compared with males, both overall ($p<0.01$) and among non-Hispanic whites ($p=0.01$) and non-Hispanic blacks ($p<0.05$) but not Hispanics ($p=0.08$).

To evaluate the qualitative relationship between fasting insulin and MetS among each ethnicity and sex, we assessed correlations of $\ln(\text{insulin})$ with BMI and individual MetS components (Table III). With the exception of diastolic blood pressure, insulin was significantly correlated with each of the components of MetS in each of the sex/ethnic groups, with strength of association strongest for WC, BMI and triglycerides and weaker for SBP, and fasting glucose. Notably, the strength of these associations was similar between non-Hispanic blacks and the other groups for each of the components and overall lower for females compared with males. Correlations of HOMA-IR with BMI and the individual MetS components were essentially identical to that of fasting insulin (data not shown).

Table IV (available at www.jpeds.com) shows mean values for MetS components by ethnicity among individuals with and without MetS, along with rates above/below the cutoffs of these components for which MetS is defined. Non-Hispanic-black adolescents with MetS had a higher BMI than either other group; however, there was no difference between groups in WC. Non-Hispanic blacks with MetS had a higher mean SBP than did Hispanics, and there was a higher prevalence of hypertension among non-Hispanic blacks with MetS than among either other groups. There were no other significant differences in MetS components between groups. Table V (available at www.jpeds.com) shows sex-specific MetS components among individuals with and without MetS. Females with MetS had a greater proportion of elevated WC than males, and males had higher fasting blood glucose values and a greater proportion of hypertension.

Discussion

Currently-used pediatric MetS criteria exhibit ethnic differences in their ability to identify elevations in fasting insulin. Even though non-Hispanic blacks and Hispanics overall have higher fasting insulin levels (and higher HOMA-IR levels) than non-Hispanic whites, what is more striking is that a MetS diagnosis confers a greater elevation beyond baseline in insulin levels among non-Hispanic-black adolescents than is seen among non-Hispanic whites but not Hispanics. These larger differences in insulin levels between non-Hispanic blacks with and without MetS could be interpreted as a MetS diagnosis favorably detecting individuals with insulin resistance. However, when used as a screening test, MetS exhibits lower sensitivity for detecting elevated insulin levels among non-Hispanic black males than other ethnicities, with a similar trend among non-Hispanic black females ($p=0.05$). Together these findings support the hypothesis that currently-used criteria to diagnose MetS are less effective at identifying insulin resistance among non-Hispanic blacks than among non-Hispanic whites and that non-Hispanic blacks may not be diagnosed with MetS until they have progressed to a more extreme degree of insulin resistance. Given the importance of insulin resistance for the development of T2DM and CVD, these data suggest that MetS may not adequately identify all non-Hispanic blacks at elevated risk for future disease.

Using criteria that are based on ATP III guidelines, MetS is diagnosed based on an individual exceeding set cut-off points in at least three individual components of MetS, findings that are all associated with insulin resistance (1, 2, 6, 8, 9). Although MetS diagnosis is a good predictor of future T2DM (10), the use of cut-off points to diagnose MetS is problematic, resulting in a high degree of instability in MetS classification during adolescence (24). Further, the use of cut-off points ignores potential ethnic variation in specific components of MetS because these cut-off values are determined according to population-based norms and not ethnicity-specific norms. Non-Hispanic blacks have lower baseline levels of triglycerides and higher baseline levels of HDL-C than whites (25, 26) and are less likely to exceed currently cut-off values to diagnose MetS (4, 7, 9). It is possible that these differences in baseline lipid levels result in the lower rate of MetS diagnosis in non-Hispanic blacks despite increased rates of insulin resistance, T2DM and CVD (11–14).

We tested ethnicity-specific correlations between insulin, triglycerides and HDL-C to determine whether the relationship between insulin resistance and dyslipidemia is different in non-Hispanic-black adolescents compared with other ethnicities. Instead we found similar correlations between fasting insulin (and HOMA-IR), triglycerides between ethnicities (Table III), findings are similar to what has been noted among adults (26). Thus, it appears that non-Hispanic blacks with increasing degrees of insulin resistance are likely to experience excursions from baseline in triglycerides and HDL-C, though they still may be less likely to exceed current cut-off limits to contribute toward a MetS diagnosis (25).

One explanation for the exaggerated increase in insulin levels among non-Hispanic blacks with MetS is that by the time non-Hispanic blacks exceed current cut-off values for dyslipidemia, they already exhibit a more progressive condition of MetS, with greater excursions from baseline in individual components of MetS and greater insulin resistance. To investigate this, we evaluated BMI and the individual MetS components by ethnicity among adolescents diagnosed with MetS. Overall, each ethnicity exhibited similar levels of the individual components of MetS and similar proportions of subjects with elevations in any given component (Table IV). This included a similar prevalence of hypertriglyceridemia and low HDL-C levels among non-Hispanic black adolescents with MetS (72.2% and 73.3%, respectively) as is seen among non-Hispanic whites (85.7% and 70.8%) and Hispanics (87.5% and 78.3%), and non-Hispanic blacks without MetS had lower rates of dyslipidemia than the other ethnicities. Thus, despite more favorable baseline levels of triglycerides and HDL-C, non-Hispanic blacks with MetS have a similar proportion of dyslipidemia as other ethnicities.

The biggest difference separating non-Hispanic black adolescents with MetS from non-Hispanic whites and Hispanics was a higher BMI. Non-Hispanic blacks also had higher BMI among the entire sample, as has been well-documented previously (27). Although non-Hispanic blacks have been noted to have on average 3% more lean mass per unit body weight, the magnitude of difference in BMI among adolescents with MetS (approximately 10%) would suggest a higher amount of adipose tissue as well (27). It has been well-documented that measures of WC are better predictors of insulin resistance than BMI (27); although non-Hispanic blacks with MetS had the highest WC of the races/ethnicities, this was not statistically significant. Thus, the cause of worsened insulin resistance among non-Hispanic black adolescents with MetS remains unclear. Non-Hispanic blacks with MetS also had a higher SBP than either of the other ethnicities, though the association between SBP and insulin resistance in non-Hispanic blacks is weaker than for other MetS components (Table III and (28)).

We noted higher fasting insulin levels among adolescent females than males, which was also true among females without MetS. This occurred despite significantly lower rates of MetS diagnosis among female compared with male adolescents (5.9% vs. 11.1%). Worsened insulin resistance in female vs. male adolescents has been reported during pubertal progression (15), and among adults, males have higher fasting insulin levels (16). Among adolescents with MetS, males and females of each ethnic group had similar fasting insulin levels, producing a trend ($p=0.06$) toward a wider difference in fasting insulin between males with and without MetS than in females. Together these data suggest a higher degree of insulin resistance among female adolescents without MetS than males. Nevertheless, females in this sample did not have significantly higher HOMA-IR values, partly related to higher fasting glucose levels among males. The cause of these discrepancies by sex is uncertain though higher overall insulin levels in females may be related to a higher proportion of elevated WC among females without MetS.

This study had several limitations. Although powerful, the NHANES data we used did not contain information regarding pubertal status or body composition, which are of clear importance in the consideration of insulin resistance. We excluded subjects with unknown diabetes (fasting BG >125 mg/dL) because by the time individuals have developed diabetes they have already lost some of their insulin secretory ability (19). We also excluded those on anti-hyperlipidemic and anti-diabetic medication because these treatments may have unequally influenced the levels of insulin and/or the components of MetS to complicate interpretation of the underlying relationships. These exclusion criteria may have excluded the most severely-affected subjects of each ethnicity and sex with potential influence on our findings. We used fasting insulin as an estimate of the degree of insulin resistance in this

cohort. Even though the presence of an elevated fasting insulin level has poor specificity for picking up individuals who will go on to develop T2DM, it does have utility in population-based studies, given its strong overall association with MetS and its association with risk for later development of diabetes (29). When we substituted fasting insulin for HOMA-IR (an estimate of insulin resistance that uses both fasting insulin and fasting glucose), this yielded similar results for all measures. Although not ideal methods of estimating insulin sensitivity, these measures overall correlate with the hyperinsulinemic-euglycemic clamp method, the gold standard for assessing insulin resistance (30). Finally, females and non-Hispanic blacks had higher fasting insulin levels overall; thus in testing for sensitivity of MetS for detecting elevated insulin, these groups were more likely to exceed our population-based cut-off without clear knowledge that their higher insulin levels correspond to higher risk on a sex- and race/ethnicity- specific basis.

The most important consideration regarding racial/ethnic discrepancies in MetS diagnosis is that if the current pediatric MetS criteria were used to identify children at risk for adult disease who required increased intervention efforts (5, 10), non-Hispanic-black adolescents may disproportionately miss out on these additional efforts despite their elevated higher lifetime risk for T2DM and CVD. One solution to this would be to identify other markers besides MetS that are accurate among all ethnicities at identifying risk to help target children and adolescents for increased intervention. Alternatively, another approach could be ethnicity-specific MetS criteria (for example, with ethnicity-specific cut-off values for each of the components) which could be designed that could better identify non-Hispanic blacks at risk (4, 25).

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Figure 1.

Adjusted geometric means of insulin by sex, ethnicity and MetS status. Estimated geometric means (95% CIs) for A, males and B, females among adolescents with and without MetS, for non-smokers with a high school degree and an income-to-needs ratio = 2. Ratio of adjusted geometric means (and 95% CIs) of fasting insulin (MetS+/MetS-) by ethnicity for C, males and D, females. Comparisons between ethnic groups by corresponding MetS status are : * $p < 0.05$, ** $p < 0.01$, both vs. non-Hispanic whites.

Figure 2.

Sensitivity of a diagnosis of MetS to detect elevated fasting insulin by sex- and ethnicity. A “high” fasting insulin was defined as the 95th percentile for non-overweight adolescents (16.0 IU/mL). Comparisons between racial/ethnic groups by corresponding sex and MetS status are : * $p < 0.05$ vs. non-Hispanic whites; # $p < 0.05$ vs. Hispanics. Comparisons between sexes: § $p < 0.05$ females vs. males.

NHANES 1999–2008 Characteristics of subjects 12–19 years old with data on all outcomes of interest (n=3693)

Table 1

	n	% with MetS	Age (Years)	BMI	Waist (cm)	Triglycerides (mg/dL)	Mean (95% CI)					
							HDL (mg/dL)	SBP (mmHg)	DBP (mmHg)	Fasting Glucose (mg/dL)	Fast Insulin (IU/mL)	HOMA score
Overall	3,693	8.6	15.5 (15.4, 15.6)	23.4 (23.1, 23.7)	81.1 (80.3, 82.0)	90.0 (87.1, 93.0)	50.9 (50.3, 51.5)	109.8 (109.1, 110.5)	61.9 (61.3, 62.5)	93.2 (92.7, 93.7)	10.9 (10.4, 11.3)	2.5 (2.4, 2.6)
Sex												
Males	1,949	11.1	15.5 (15.4, 15.7)	23.2 (22.8, 23.6)	81.6 (80.5, 82.7)	91.8 (88.0, 95.6)	48.8 (48.0, 49.6)	112.5 (111.6, 113.4)	60.6 (59.9, 61.4)	95.1 (94.6, 95.5)	10.5 (9.8, 11.1)	2.5 (2.3, 2.7)
Females	1,744	5.9	15.5 (15.3, 15.7)	23.6 (23.2, 24.0)	80.7 (79.6, 81.7)	88.2 (84.2, 92.2)	53.1 (52.2, 54.0)	107.0 (106.2, 107.8)	63.3 (62.6, 64.0)	91.2 (90.6, 91.9)	11.3 (10.7, 11.9)	2.6 (2.4, 2.7)
p-value *		0.0003	0.5042	0.2174	0.1795	0.1752	<.0001	<.0001	<.0001	<.0001	0.0393	0.3937
By Ethnicity												
Non-Hispanic White	1,045	8.8	15.6 (15.4, 15.7)	23.0 (22.6, 23.4)	80.9 (79.8, 82.0)	93.6 (89.8, 97.4)	50.1 (49.3, 51.0)	109.5 (108.6, 110.4)	62.4 (61.6, 63.1)	93.4 (92.8, 94.0)	10.1 (9.5, 10.7)	2.4 (2.2, 2.5)
Hispanic	1,486	11.2	15.4 (15.2, 15.6)	23.9 (23.4, 24.4)	82.7 (81.5, 83.9)	94.8 (88.8, 100.9)	50.2 (49.4, 51.0)	109.3 (108.3, 110.2)	60.3 (59.6, 61.1)	94.3 (93.7, 95.0)	12.5 (11.7, 13.4)	3.0 (2.8, 3.2)
Non-Hispanic Black	1,162	4.7	15.4 (15.2, 15.6)	24.5 (24.1, 25.0)	80.4 (79.3, 81.5)	68.3 (65.9, 70.7)	55.1 (54.1, 56.1)	111.9 (111.1, 112.6)	61.7 (60.9, 62.6)	91.1 (90.6, 91.7)	12.3 (11.6, 13.1)	2.8 (2.6, 3.0)
p-value **		0.0001	0.2930	<.0001	0.0069	<.0001	<.0001	<.0001	0.0004	<.0001	<.0001	<.0001

* Chi-square test comparing MetS prevalence, t-test comparing outcomes of interest

** Chi-square test comparing MetS prevalence, ANOVA comparing outcomes of interest (overall difference among the groups)

Table II

Linear Model Results of ln(Fasting Insulin)^{*}

Model Covariate	Estimate	95% CI	p-value
Intercept	1.98	(1.88, 2.07)	< 0.01
Education ^{**}			
Less than High School	0.10	(0.02, 0.18)	0.01
High School	-0.03	(-0.10, 0.05)	0.46
Income-to-Needs Ratio	-0.02	(-0.04, 0.00)	0.12
Current Smoker	-0.22	(-0.31, -0.12)	< 0.01
Females (vs. Males)	0.21	(0.14, 0.28)	< 0.01
Race/Ethnicity [†]			
Hispanic	0.08	(0.00, 0.17)	0.05
Non-Hispanic Black	0.11	(0.04, 0.19)	< 0.01
MetS vs. No MetS – Females			
Non-Hispanic White	0.77	(0.53, 1.02)	< 0.0001
Hispanic	0.83	(0.62, 1.04)	< 0.0001
Non-Hispanic Black	1.01	(0.80, 1.23)	< 0.0001
MetS vs. No MetS – Males			
Non-Hispanic White	0.97	(0.81, 1.13)	< 0.01
Hispanic	1.03	(0.83, 1.23)	< 0.01
Non-Hispanic Black	1.21	(1.01, 1.42)	< 0.01

^{*} Final model included MetS x sex interaction (p = 0.06) and MetS x ethnicity interaction (p = 0.11); model R² = 0.170

^{**} Highest among household (person who owns/rents house or his/her spouse), values indicate differences from “more than high school” category

[†] Values indicate difference from Non-Hispanic White

Table 3

Correlations Between MetS components and ln(Insulin). *

Sex	Ethnicity	Pearson r (95% CI)						
		BMI	WC	SBP	DBP	Triglycerides ^{***}	HDL	Fasting Glucose
Overall	Non-Hispanic White	0.48 (0.43, 0.52)	0.49 (0.45, 0.54)	0.27 (0.21, 0.32)	-0.04 (-0.11, 0.02)	0.34 (0.28, 0.39)	-0.21 (-0.26, -0.15)	0.30 (0.24, 0.35)
	Hispanic	0.58 (0.55, 0.61)	0.58 (0.54, 0.61)	0.22 (0.17, 0.27)	0.08 (0.03, 0.13)	0.43 (0.39, 0.47)	-0.28 (-0.32, -0.23)	0.28 (0.23, 0.33)
	Non-Hispanic Black	0.55 (0.51, 0.59)	0.55 (0.51, 0.59)	0.16 (0.11, 0.22)	0.02 (-0.03, 0.08)	0.35 (0.30, 0.40)	-0.25 (-0.31, -0.20)	0.29 (0.24, 0.34)
Males	Non-Hispanic White	0.56 (0.50, 0.61)	0.59 (0.53, 0.64)	0.33 (0.25, 0.40)	-0.05 (-0.14, 0.03)	0.42 (0.35, 0.49)	-0.32 (-0.40, -0.25)	0.33 (0.25, 0.40)
	Hispanic	0.61 (0.57, 0.66)	0.63 (0.59, 0.67)	0.23 (0.16, 0.30)	0.05 (-0.02, 0.12)	0.48 (0.42, 0.53)	-0.25 (-0.32, -0.19)	0.34 (0.27, 0.40)
	Non-Hispanic Black	0.54 (0.49, 0.59)	0.56 (0.50, 0.61)	0.24 (0.16, 0.31)	0.00 (-0.08, 0.08)	0.38 (0.31, 0.45)	-0.28 (-0.35, -0.21)	0.39 (0.32, 0.46)
Female	Non-Hispanic White	0.38 (0.30, 0.45)	0.37 (0.29, 0.44)	0.25 (0.17, 0.33)	-0.05 (-0.13, 0.04)	0.23 (0.14, 0.31)	-0.12 -0.21, -0.03)	0.32 (0.24, 0.40)
	Hispanic	0.54 (0.49, 0.59)	0.52 (0.47, 0.57)	0.29 (0.23, 0.36)	0.08 (0.01, 0.15)	0.37 (0.31, 0.43)	-0.35 (-0.41, -0.28)	0.31 (0.24, 0.37)
	Non-Hispanic Black	0.53 (0.47, 0.59)	0.53 (0.47, 0.59)	0.21 (0.12, 0.29)	-0.00 (-0.09, 0.08)	0.33 (0.25, 0.41)	-0.26 (-0.34, -0.18)	0.33 (0.25, 0.40)

* Correlation estimates and corresponding 95% CI's; significant correlations (p<0.05) in **bold**.

** Natural log of variable was used to achieve normality.