Published in final edited form as:

Int J Behav Med. 2020 April ; 27(2): 188–199. doi:10.1007/s12529-020-09847-y.

Elucidating the Multidimensionality of Socioeconomic Status in Relation to Metabolic Syndrome in the Hispanic Community Health Study/Study of Latinos (HCHS/SOL)

Tasneem Khambaty, PhD^a, Neil Schneiderman, PhD^b, Maria M. Llabre, PhD^b, Tali Elfassy, MSPH^b, Ashley E. Moncrieft, PhD^b, Martha Daviglus, MD, PhD^c, Gregory A. Talavera, MD, MPH^d, Carmen R. Isasi, MD, PhD^e, Linda C. Gallo, PhD^f, Samantha A. Reina, MS^b, Denise Vidot, PhD^b, Gerardo Heiss, MD, PhD^g

^aDepartment of Psychology, University of Maryland, Baltimore County, Baltimore, MD, United States of America;

^bDepartment of Psychology and Behavioral Medicine Research Center, University of Miami, 5665 Ponce De Leon Boulevard Coral Gables, FL, 33124, United States of America;

^cDepartment of Medicine, University of Illinois, Chicago, IL, United States of America;

^dGraduate School of Public Health, San Diego State University, San Diego, CA, United States of America;

^eDepartment of Epidemiology & Population Health, Albert Einstein College of Medicine, Bronx, NY, United States of America;

^fDepartment of Psychology, San Diego State University, San Diego, CA, United States of America;

^gDepartment of Epidemiology, University of North Carolina at Chapel Hill, Chapel Hill, NC, United States of America;

Abstract

Background: Socioeconomic (SES) factors underlying disparities in the prevalence of metabolic syndrome (MetSyn) and consequently, type 2 diabetes among Hispanics /Latino populations are of considerable clinical and public health interest. However, incomplete and/or imprecise

Corresponding Author: Tasneem Khambaty; Address: 1000 Hilltop Circle, Math/Psychology 326, Baltimore, MD 21250; khambaty@umbc.edu; Phone: 410.455.2304; Fax: 410.455.1055.

Ethical approval: All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Conflict of interest:

The authors declare that they have no conflict of interest.

Informed Consent:

The study protocol was approved by the Institutional Review Board (IRB) at each field center, namely the University of Miami IRB, the University of North Carolina IRB, Albert Einstein College of Medicine IRB, San Diego State University IRB, and Northwestern University IRB. Written informed consent was obtained from all participants.

Availability of data and material:

The dataset used for the current study are available upon reasonable request to the coordinating center via email (hchsadministration@unc.edu).

measurement of the multidimensional SES construct has impeded a full understanding of how SES contributes to disparities in metabolic disease. Consequently, a latent-variable model of the SES-MetSyn association was investigated and compared with the more typical proxy-variable model.

Methods: A community based cross-sectional probability sample (2008–2011) of 14,029 Hispanic/Latino individuals of Puerto Rican, Cuban, Dominican, Central American, South American, and Mexican ancestry living in the U.S was used. SES proxy's education, income, and employment were examined as effect indicators of a latent variable, and as individual predictors. MetSyn was defined using 2009 harmonized guidelines, and MetSyn components were also examined individually.

Results: In multivariate regression analyses, the SES latent variable was associated with 9% decreased odds of MetSyn (95% confidence interval: 0.85, 0.96, $P < .001$) and was associated with all MetSyn components, except diastolic blood pressure. Additionally, greater income, education, and employment status were associated with 4%, 3%, and 24% decreased odds of having MetSyn, respectively (P s $< .001$). The income-MetSyn association was only significant for women and those with current health insurance.

Conclusions: Hispanic/Latinos exhibit an inverse association between SES and MetSyn of varying magnitudes across SES variables. Public health research is needed to further probe these relationships, particularly among Hispanic/Latina women, to ultimately improve healthcare access to prevent diabetes in this underserved population.

Keywords

Health Disparities; Hispanics/Latinos; Latent Models; Metabolic Syndrome; Socioeconomic Status; Women's Health

Background

Metabolic syndrome (MetSyn) is conceptualized as a cluster of cardiometabolic abnormalities related to insulin resistance that confers an increased risk for 2 diabetes and cardiovascular disease [1–3]. MetSyn – consisting of hyperglycemia, abdominal adiposity, hyperlipidemia, and hypertension – can be observed in one-third of all adults in the U.S. [4]. Alarming, MetSyn rates are disproportionately higher among minority race/ethnicity groups. In fact, the highest rates – 36% among women and 34% among men – are observed among Hispanic/Latino populations, the fastest growing ethnic minority group in the U.S [5, 6]. Not surprisingly, factors underlying disparities in the prevalence of MetSyn and consequently, type 2 diabetes among Hispanics/Latinos are of considerable clinical and public health interest.

A recent study compared the relative impact of socioeconomic, local environmental, psychosocial, lifestyle/behavioral, and biophysiological factors on excess type 2 diabetes risk among minority race/ethnicity groups and found that the largest explainable proportion was attributable to socioeconomic status (SES) [7]. SES is a multidimensional, abstract measure of access to material and social resources, and is most commonly operationalized by the proxy variables education, income, and/or employment [8]. A growing number of studies have begun to focus on SES as a key factor implicated in race/ethnicity disparities in

MetSyn [10–16], with good reason. These studies report a consistent inverse association such that as SES increases, risk for cardiometabolic morbidity decreases. This SES gradient in cardiometabolic health is observed across the SES hierarchy (i.e., from the lowest to highest SES categories), across the lifespan (i.e., in children, adults, and older adults), and across racial/ethnic groups [9].

And yet, there are critical limitations of prior studies examining SES and MetSyn: First, while several studies have been conducted in African American [10, 11] and Asian [12–14] samples, there exist only a few studies among Hispanic/Latinos, and those too primarily among Mexican American adults [15, 16]. Given vast heterogeneity in the geographic distribution, ancestry, socioeconomic status (SES), and cultural background of Hispanics/Latinos residing in the U.S. [17], these results from primarily Mexican American samples may not generalize to the wider Hispanic/Latino population. Even rates of MetSyn vary among these groups, with South Americans having the lowest prevalence, and Puerto Ricans and Cubans having the highest prevalence [5]. As a consequence, little is known about how SES contributes to Hispanics/Latinos population of diverse ancestry having a disproportionately higher MetSyn risk than other groups. A second critical limitation of prior studies has been the lack of inclusion of all three major SES proxy variables in the same analysis. Thus, the contribution of education, income, and employment, individually or in combination, towards MetSyn prevalence among Hispanics/Latinos is unclear. To address these limitations, this study examined (a) the association of individual SES proxy variables education, income, and employment with MetSyn and its components, and (b) the combined contribution of SES, in the form of a latent construct, towards MetSyn prevalence. This approach has a distinct advantage in the context of an immigrant population (such as in the current study) among whom discordance is often observed among measured SES indicators. For instance, immigrants are often unable to practice a profession in the US commensurate with the level of education they obtained in a different country for various reasons (e.g., government rules, licensure), which results in lower income than predicted by their education. This approach allows incorporation of this discordance by determining how each of the SES variables fare individually and in comparison.

A third objective of this study was to obtain a better understanding of the SES-MetSyn association by examining sex and health insurance as effect modifiers. There is a need to understand whether a stronger SES-MetSyn association among women than among men reported in studies of other race/ethnicity groups is also present in a diverse sample of Hispanic/Latinos [16, 18, 19]. Moreover, health insurance, or access to health care services, is a critical factor in disparities in cardiometabolic outcomes and an important moderator of health literacy, preventative interventions, and management of disease. Despite the passage of the Affordable Care Act of 2010, and other public health initiatives, the percentage of Hispanics/Latinos with health insurance remains low [20]. These uninsured Hispanic/Latino individuals report lower levels of knowledge and awareness of diabetes and related conditions resulting in higher cardiometabolic morbidity [21]. Yet, other studies indicate that the effect of SES on cardiometabolic morbidity persists even when participants have access to health care and that race/ethnicity disparities in metabolic outcomes remain after adjustment for health care access [9, 22]. These paradoxical findings underscore the need to understand the influence of health insurance on the SES-MetSyn relationship. Results from

these data may advance our understanding of the most relevant factors contributing to disparities in MetSyn and its cardiometabolic sequelae and inform the design of public health interventions at the individual and societal levels targeting these factors, ultimately helping to reduce the disproportionate health burden in this historically underserved population.

Methods

Participants

The Hispanic Community Health Study/ Study of Latinos (HCHS/SOL; <http://www.csc.unc.edu/hchs/>) is a community based cross-sectional probability sample of self-identified Hispanic/Latino individuals aged 18–74 years at screening from randomly selected households in four U.S. field centers (Chicago, IL; Miami, FL; Bronx, NY; San Diego, CA). The sample design and cohort selection has been described previously [23, 24]. Briefly, a stratified two-stage area probability sampling plan was used, such that census block groups were randomly chosen in specified geographic areas of each study site, and households were randomly chosen in each sample census block group. Individuals in each chosen household were eligible for the HCHS/SOL study if they self-identified as Hispanic/Latino, were able to travel to a local field center for examination, were not active military or currently pregnant, and had no plans to move from the study area within three years. These eligibility criteria were assessed via telephone calls or in person visits. Of the individuals screened to be eligible, 42% agreed to enroll and were invited to the baseline examination, occurring at each U.S field center between March 2008 and June 2011. The examination included comprehensive biological (e.g., blood draw, anthropometrics), behavioral (e.g., objective and self-report physical activity, tobacco and alcohol use), and socio-demographic (e.g., education and income, acculturation) assessments. Of note, the 45–74-year age group was oversampled to facilitate examination of cardiometabolic outcomes. The study protocol was approved by the Institutional Review Board at each field center and written informed consent was obtained from all participants. Of the 16,415 total participants enrolled in the HCHS/SOL, those with missing data on key variables (SES proxy variables: N=1,794; MetSyn components: N=627; Hispanic background: N=87; covariates: N=429), and two additional participants with triglycerides values considered to be outliers (6,366 and 3,425mg/dL; next highest value 2,234mg/dL) were excluded, resulting in an analytic sample of 14,029 participants.

Measures

SES.—SES was operationalized in two ways - one based on proxy variables and another based on a latent variable. The proxy variables were (a) yearly household income (categorized as <\$10,000, \$10,000 to \$15,000, >\$15,000 to \$20,000, >\$20,000 to \$25,000, >\$25,000 to \$30,000, >\$30,000 to \$40,000, >\$40,000 to \$50,000, >\$50,000 to \$75,000, >\$75,000 to \$100,000, or >\$100,000), (b) education (years of school completed), and (c) employment status (coded categorically using three dummy vectors for ‘full-time employed’, ‘part-time employed’, and ‘retired’, with ‘not currently employed’ as the reference category). SES data was obtained by interview with certified assessors and via standard questionnaires. The latent variable was created as a more precise definition of SES,

as any one proxy variable alone is not as reliable as the latent construct. Income, education, and employment status were conceptualized as effect indicators of, and thus, manifestations of a latent SES variable. The latent variable was scaled using the metric of the education variable (i.e., years of school completed).

MetSyn.—MetSyn was defined according to a harmonized definition [4], which requires the presence of three or more of the following components: (a) waist circumference (WC) 102 cm in men and 88 cm in women; (b) systolic (SBP) and diastolic (DBP) blood pressures 130 mm Hg and 85 mm Hg, respectively, or use of antihypertensive medication; (c) high-density lipoprotein cholesterol (HDL-C) levels <40 mg/dL in men and <50 mg/dL in women, or use of cholesterol medication; (d) triglyceride (TG) levels 150 mg/dL or use of lipid-lowering medication; and (e) fasting plasma glucose (FPG) level 100 mg/dL or use of medication. WC was measured with a measuring tape at the uppermost lateral border of the right ilium to the nearest 0.1 cm. SBP and DBP were measured three times at 1-minute intervals after a 5-minute rest in a seated position using an automatic sphygmomanometer (Omron model HEM-907 XL, Omron Healthcare Inc., Bannockburn, IL). The average of the three readings was used, consistent with guidelines. HDL-C, TG, and FPG values were obtained from fasting blood samples. TG and FPG values were logarithmically transformed and multiplied by 100 to obtain normalized distributions. Antihypertensive, lipid-lowering, and oral hypoglycemic medication use during the month prior to the baseline visit was assessed via standard questionnaire and interview, and when available, by scanning Universal Product Code bar codes or centralized manual coding. The dichotomous harmonized MetSyn variable, and the six continuous MetSyn components were analyzed as separate dependent variables.

Candidate effect modifiers.—Sex (0 = female, 1 = male), and health insurance (0 = no current health insurance, 1 = yes current health insurance) were assessed through standard questionnaires.

Covariates.—Age, field center, Hispanic/Latino ancestry group, and nativity/length of residence in the U.S. (0 = Foreign born, 0–5 years lived in the U.S., 1 = Foreign born, 6–10 years lived in the U.S., 2 = Foreign born, 11–15 years lived in the U.S., 3 = Foreign born, 16 or more years lived in the U.S., and 4 = Born in the U.S.) were assessed via standard questionnaires and interviews. Age was examined as a continuous variable. Field center was dummy coded to obtain three variables for Bronx, Chicago, and San Diego field centers, with Miami serving as the reference group (the purpose of including this variable was to control site variation, which is invariant to the choice of referent). Hispanic/Latino ancestry group was also dummy coded to obtain six variables for Puerto Rican, Cuban, Dominican, Central American, South American, and more than one/other heritage, with Mexican ancestry as the reference group (given that this was the largest category).

Analytic Plan

First, descriptive analyses were conducted to obtain MetSyn prevalence and MetSyn component means (95% CI) by sociodemographic and SES factors. All prevalence values were weighted for sampling probability, nonresponse, and age standardized to the 2010 U.S.

population. Descriptive analyses were conducted using SAS version 9.3 (SAS Institute) and SUDAAN release 10.0.0 (RTI). Second, to determine whether SES was associated with MetSyn and its components, four sets of logistic or linear regression analyses were performed, one set for each SES proxy or latent predictor variable. In each set, the harmonized MetSyn variable (logistic regression analyses), or each of the six continuous MetSyn components (i.e., WC, TG, HDL-C, SBP, DBP, FPG; linear regression analyses) served as criterion variables in separate models. Along with the respective SES predictor variable, each model also included age, sex, field center, Hispanic/Latino background, nativity/length of residence in the U.S., and health insurance as covariates. Due to the large number of comparisons in these analyses, a conservative alpha ($P < .01$) was used. Finally, to determine if the SES-MetSyn association varied as a function of sex and health insurance, interaction terms between each SES proxy variable and each candidate effect modifier were created and entered one at a time in separate logistic regression models predicting the harmonized MetSyn variable along with the main effects and aforementioned covariates. When models yielded significant interaction terms, analyses stratified by level(s) of the effect modifier were conducted. Linear regression analyses were also performed to test the interaction in models predicting each of the six MetSyn components. Statistical analyses were conducted using Mplus software, version 7.1, SAS version 9.3 (SAS Institute), and SUDAAN release 10.0.0 (RTI), incorporating sampling weights, stratification, and clustering features of the study design.

Results

Descriptive Results

The prevalence of MetSyn, the number of MetSyn components above diagnostic threshold [4], and mean (95% CI) values of each MetSyn component are shown in Table 1 by age, sex, Hispanic background, and health insurance. As expected, MetSyn prevalence was higher, and number of components above threshold were greater with increasing age range and significantly higher than in the 18–44 age range (all p s $< .001$). Interestingly, although men and women did not differ significantly on overall MetSyn prevalence ($P = .295$), men had significantly worse values on all MetSyn components than women (all P s $< .001$). This can occur when cutpoints are created for dichotomous variables and continuous information is lost. Although individuals with versus without current health insurance had significantly lower TG values ($P < .001$), overall MetSyn prevalence, or number of components above threshold did not differ by health insurance status (P s $< .452$).

The prevalence of MetSyn, the number of MetSyn components above diagnostic threshold, and mean (95% CI) values of each MetSyn component are shown in Table 2 by SES proxy variables. In these unadjusted analyses, individuals with less than a high school education had significantly higher MetSyn prevalence and more components above threshold than those with a high school diploma or better (all P s $< .001$). MetSyn prevalence and number of components above threshold were significantly lower for individuals in the highest versus lowest income category ($> \$75,000$ vs. $< \$20,000$; P s $< .001$). Among the MetSyn component values, only TG values were better among those earning the highest versus lowest incomes ($P < .003$). Lastly, both full-time and part-time employed participants had

significantly lower MetSyn prevalence and number of components above threshold than those unemployed ($P < .001$). Further, full-time employees exhibited significantly better values on WC and FPG ($P < .002$), while part-time employees exhibited significantly better values on all MetSyn components other than HDL-C and TG ($P < .001$) compared to those unemployed.

SES latent variable in relation to MetSyn and its components

Unstandardized factor loadings, representing the covariance between each of the indicators and the latent variable, were statistically significant (all p -values $< .01$). Moreover, standardized factor loadings indicated that each was an adequate indicator of the latent variable: income: $\beta = 0.82$, 95% Confidence Interval (CI): 0.73, 0.90, education: $\beta = 0.36$, 95% CI: 0.32, 0.41, full-time employed: $\beta = 0.87$, 95% CI: 0.75, 1.00, part-time employed: $\beta = 0.28$, 95% CI: 0.18, 0.38, and retired: $\beta = -0.24$, 95% CI: -0.44 , -0.37 . The latent SES variable was significantly and inversely related to MetSyn ($OR = 0.91$, 95% CI: 0.85, 0.96, $P < .001$). Specifically, for each unit increase in SES, the odds of having MetSyn decreased by 9%. Moreover, this latent SES variable was significantly related to five of the six MetSyn components in the expected directions: WC (unstandardized beta = -0.61 , $R^2 = 0.07$, $P < .001$), SBP (unstandardized beta = -0.61 , $R^2 = 0.30$, $P < .001$), HDL-C (unstandardized beta = 0.61 , $R^2 = 0.10$, $P < .001$), TG (unstandardized beta = -0.80 , $R^2 = 0.13$, $P = .002$), and FPG (unstandardized beta = -0.50 , $R^2 = 0.11$, $P < .001$); SES was not related to DBP (unstandardized beta = 0.17 , $R^2 = 0.13$, $P = .136$).

SES proxy variables in relation to MetSyn and its components

The odds of having MetSyn decreased by 4% (Odds Ratio (OR) = 0.96, 95% CI: 0.93, 0.98, $P < .001$; Table 3, column 1) with increasing income bracket. Income was significantly associated with all MetSyn components with the exception of DBP and TG in the expected directions ($P < .007$; Table 3, columns 2–7). Furthermore, each additional year of education was associated with 3% decreased odds of having MetSyn (OR = 0.97, 95% CI: 0.96, 0.98, $P < .001$; Table 3, column 1). Education was associated with WC, HDL-C, SBP, and FPG ($P < .001$) with magnitudes generally similar to those in the income models (Table 3, columns 2–7). Lastly, part time (OR = 0.78, 95% CI: 0.64, 0.94, $P < .001$) and full time (OR = 0.76, 95% CI: 0.67, 0.85, $P < .001$) employed persons had 22% and 24% decreased odds of having MetSyn compared to unemployed persons, respectively (Table 3, column 1). When compared to unemployed status, full time status was associated with all metabolic syndrome components except SBP and DBP ($P < .001$; Table 3, columns 2–7). Of note, the above SES-MetSyn associations did not differ by Hispanic background at the $\alpha < .01$ level (data not shown).

Effect modification analyses

Table 4 reports mean (SE) of education, income, and the percentage of full-time and part-time employed participants by sex and insurance status. These descriptive analyses reveal that despite a lack of significant differences between men and women on education ($P = .24$), there were significant differences in both income ($P < .001$) and occupation ($P < .001$). Specifically, men had higher incomes and a greater percentage reported full-time occupation than women. Moreover, current health insurance was associated with significantly higher

education ($P=.002$), and greater income ($P<.001$), but not the percentage of full-time work ($P=.23$).

Income.—Separate logistic regression models revealed significant effect modification of the income-MetSyn association by sex and health insurance, as evidenced by significant income $\times$ sex and income $\times$ health insurance interaction terms ($P_s < .001$). Stratified analyses demonstrated that women in higher income brackets had 8% decreased odds of having MetSyn, while no such relationships were observed among men (Table 3, column 1). Similarly, participants with current health insurance also had 8% decreased odds of having MetSyn, while no such relationship was observed among those without current health insurance (Table 3, column 1). Examining MetSyn components, sex was a significant effect modifier of the associations between income and WC and HDL-C ($P_s < .002$), and health insurance was a significant effect modifier of the associations of income with WC, SBP, and FPG ($P_s < .003$; Table 3, columns 2, 5, 7). For example, moving from one to the next higher income bracket was associated with a 0.54 cm decrease in WC among women, while no association was found among men. Similarly, moving from one to the next higher income bracket was associated with a 0.42 cm decrease in WC for those with current health insurance, while no association was found for those without current health insurance.

Education.—There was no effect modification of the association between education and MetSyn by sex ($P=.02$) or health insurance ($P=.02$; Table 3, column 1). However, the associations of education with WC, TG, HDL-C, and SBP were significant among women but not men ($P_s < .004$; Table 3, columns 2–5), and the association of education with HDL-C was significant for those with health insurance ($P=.002$). For example, each additional year of education was associated with a 0.40 cm decrease in WC among women, while no association was found among men.

Employment status.—There was no effect modification of the association between employment and MetSyn by sex ($P=.014$) or health insurance ($P=.225$; Table 3, column 1). However, significant interactions were observed between employment and sex in separate models predicting TG, HDL-C, and SBP ($P_s < .001$, Table 3, columns 3–5). For example, when compared to unemployed women, full-time employed women had a 2.98 mg/dL increase in HDL-C, while no association was found among men.

Conclusions

The primary finding based on analysis of the HCHS/SOL data was a significant inverse relationship between SES and MetSyn among Hispanic/Latino adults of diverse ancestry groups. Multivariate regression analyses revealed that higher income and education, and full-time employment status versus unemployed status were associated with a 4%, 3%, and 24% decreased odds of having MetSyn, respectively. It is important to note that the larger OR for employment status than for income or education is likely due to the employment status variable being modeled categorically instead of continuously. Moreover, when a latent variable was examined, SES was associated with 9% decreased odds of having MetSyn. SES was also significantly associated with all of the MetSyn components except DBP, possibly because DBP is less responsive than SBP to changes in social (e.g. SES) and behavioral

(e.g., exercise) factors [25, 26]. Furthermore, the association of income with MetSyn was only significant for women and those with current health insurance. Sex and health insurance were less consistent effect modifiers of the associations between SES and MetSyn components with no clear patterns. Overall, these findings suggest that the deleterious effect of low SES exists at the preclinical level for cardiometabolic risk factors that comprise MetSyn, and further, may partly explain the previously documented associations between SES and the more distal clinical endpoint of type 2 diabetes.

Almost all prior investigations of the SES-MetSyn association among Hispanics/Latinos are based on Mexican American samples. Fortunately, there is an emerging literature of studies conducted using HCHS/SOL data that have investigated the relationship between SES and cardiometabolic disease outcomes. These studies have quantified the overall prevalence of MetSyn in the HCHS/SOL cohort by age, sex, and Hispanic/Latino background [5], reported on the relative contribution of individual MetSyn components towards the overall syndrome [27, 28], and established an inverse relationship of some SES components with diabetes [29]. One recent HCHS/SOL study sought to understand whether psychosocial risk (i.e., depressive symptoms, social support) and resource (inter and intrapersonal) factors mediated the association between SES and MetSyn [30]. As hypothesized, the researchers found that psychosocial risk partly mediated the SES-MetSyn relationship. Notable differences between this and the current study are a) the use of the smaller HCHS/SOL-Sociocultural Ancillary Study cohort (N=5,313), the participants of which additionally completed self-report assessments of psychosocial and sociocultural factors, b) the exclusion of occupation from their latent SES factor, and c) the examination of MetSyn as three separate latent variables reflecting blood pressure, lipid, and metabolic factors. The current report extends the SES-MetSyn literature by creating a latent SES factor comprising all three major proxy variables, examining this factor's association with both MetSyn and its individual components, and finally, investigating whether sociodemographic factors (i.e., sex, health insurance, sex) modify the SES-MetSyn association. Nevertheless, the primary finding in this study of an inverse SES-MetSyn association is consistent with the abovementioned HCHS/SOL studies, and two previous studies conducted among Mexican American men and women [15, 16]. Interestingly, this inverse association has also been documented in studies conducted among other minority groups living in the U.S. [10, 11, 16], which may speak to a similarity in contextual factors related to SES that contribute to this consistent finding. Taken together with the disproportionately higher rates of diabetes in minority populations than in Whites in the U.S., and the similar inverse association seen in diabetes incidence and prevalence, this data may point to the greater need for community- and national- level interventions over individual-level interventions aimed at improving health related knowledge and resources.

Also consistent with this study's results, a stronger SES-MetSyn association has been previously reported among women than among men [16, 18, 19, 31, 32]. With little known about whether certain MetSyn components account for these sex differences, this study's findings suggest that stronger associations of SES with WC and HDL-C among women than among men may be responsible. Given that abdominal obesity is a major contributor of MetSyn among Hispanic/Latino women compared to men [5], it is reasonable that WC accounts for the sex differences observed. Moreover, studies have documented a stronger

association of different SES markers with HDL-C levels among women than among men [18, 19, 31]. Potential reasons for these sex differences are likely multifactorial and interacting. One reason is the differential relationships of income with health behaviors among Hispanic women versus men [19]. Among women, higher income may translate into healthier food choices, while this may not be true for men due to cultural values (e.g., fatalism) and gender roles (i.e., *Machismo*), which are related to negative health behaviors [33]. In turn, increased consumption of fruits and vegetables, and lower consumption of processed foods among women would have an effect on both WC and HDL-C, and may partially explain the associations we observed between these MetSyn components and income in women but not in men. A second explanation is the association of lower SES with higher psychosocial stress among Latina women. Income below the poverty threshold in women has been associated with increased stress and depression [16, 18, 34], which in turn is related to higher cardiometabolic risk through neuroendocrine perturbations. Altogether, these findings highlight the clinical relevance of SES for women, and suggest that women may be disproportionately vulnerable to the effects of low SES on cardiometabolic risk.

A more surprising finding was that the inverse association of income with MetSyn was only observed among those with current health insurance. A notion that has been previously postulated is that health insurance buffers the ill effects of lower SES on cardiovascular risk factors [35]. Despite lower income, having adequate health insurance may be associated with greater health literacy [36, 37], access to important preventative health services [38], opportunity for early diagnosis and treatment, and ultimately, better cardiometabolic health [29, 39, 40]. However, the results we observed do not lend themselves to the above interpretation as we found an inverse income-MetSyn association only among individuals *with* current health insurance. Thus, a more likely interpretation is that health insurance serves as a prerequisite without which the cardiometabolic health benefits of a higher SES are attenuated [41]. That is, in the presence of health insurance, individuals with high income may have greater protection against cardiometabolic risk than those with lower income. However, in the absence of current health insurance, the income-related SES gradient is less relevant for cardiometabolic risk. From a public health standpoint, this interpretation is concerning because of the percentage of Hispanics/Latinos who do not have health insurance. Prior to the Affordable Care Act of 2010, the uninsured rate among Hispanics/Latinos was 29%, a rate that is twice the national average, and much higher than rates found for non-Hispanic Whites and Blacks [20]. The present analyses indicated that only 50% of the HCHS/SOL sample reported having health insurance, which is consistent with the dates of data collection (2008–2011) encompassing the passage of the Affordable Care Act. Additional studies are needed to tease apart the specific relationships among SES, health insurance, and MetSyn.

Mechanisms that underlie the inverse SES-MetSyn association have been proposed. Prior studies indicate that lifestyle/behavioral factors such as physical activity, and diet account for 33–45% of the effect of SES on diabetes [7, 42]. These factors are also implicated in the SES-MetSyn association. Low SES is associated with excess nutrient intake, increased consumption of saturated fats through highly processed foods, and less time spent in leisure time physical activity [43]. These maladaptive behaviors can promote adiposity and insulin resistance, a process by which fat, muscle, and liver tissue exhibit reduced glucose uptake,

consequently increasing glucose levels in the blood stream [2]. This study found significant consistent effects of both latent SES and all SES proxies on FPG, suggesting potential initiation of the insulin resistance process. In turn, insulin resistance is a key process underlying the cardiometabolic abnormalities associated with MetSyn and its trajectory towards type 2 diabetes [2]. However, as few studies have empirically tested these potential mechanisms among Hispanic/Latina women, these questions remain important for future research.

Limitations of this study warrant discussion. First, although the three most common indicators of SES were included in this study, other indicators like social status were not examined. However, social status is related to employment, and is likely influenced by retirement status (i.e., decreases after retirement). Second, because of the cross-sectional study design, conclusions regarding the directionality of observed relationships cannot be made, and reverse causality is possible. Third, although a major strength of this data is the diverse sample of Hispanic/Latinos, HCHS/SOL did not include a non-Hispanic/Latino cohort. Consequently, obtained results cannot be compared or generalized to non-Hispanic/Latino populations. Finally, there is a possibility that the latent SES construct is not invariant across Hispanic/Latino ancestry groups. Measurement invariance in the latent SES variable was assumed but not tested as no differences were expected in the loadings of the SES indicators (education, income, and occupation) on the latent variable across groups.

To conclude, this examination of the HCHS/SOL data revealed an inverse association between SES and MetSyn among Hispanic/Latinos, with income-MetSyn associations only significant for women and those with health insurance. The latter finding is unexpected and suggests that current health insurance is a necessary prerequisite after which income becomes relevant for cardiometabolic risk. More broadly, these findings suggest that the deleterious effect of low SES exists at both preclinical and clinical levels of diabetes. Overall, this study contributes to the limited existing knowledge of socioeconomic disparities in the distribution of MetSyn in the diverse Hispanic/Latino population, and helps to clarify the relationship between SES and metabolic risk by suggesting stronger associations of SES with some MetSyn components. Importantly, the study addressed some major criticisms of previous SES-MetSyn investigations by evaluating a latent SES variable, and analyzing MetSyn components as continuous rather than dichotomous variables. These findings highlight the need for additional research to understand the significance of these relationships for public health policy, as well as the need to address SES related factors, particularly among Hispanic/Latina women, at both the individual and societal level, through interventions aimed at improving healthcare access and health literacy to prevent diabetes in this underserved population.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgements:

The authors thank the more than 250 staff members of the HCHS/SOL for their dedication and expertise. The study website is <http://www.csc.unc.edu/hchs/>.

Funding:

The HCHS/SOL was carried out as a collaborative study supported by contracts from the National Heart, Lung, and Blood Institute(NHLBI) to the University of North Carolina (N01-HC25233), University of Miami (N01-HC65234), Albert Einstein College of Medicine (N01-HC65235), Northwestern University (N01-HC65236), and San Diego State University (N01-HC65237). The following institutes/centers, contributed to the baseline HCHS/SOL funding period through a transfer of funds to the NHLBI: National Institute on Minority Health and Health Disparities, National Institute on Deafness and Other Communication Disorders, National Institute of Dental and Craniofacial Research, National Institute of Diabetes and Digestive and Kidney Diseases, National Institute of Neurological Disorders and Stroke, and National Institute of Health Office of Dietary Supplements. T.E., T.K., A.M., S.R. and D.V. were supported by T32 HL07426. These funding sources were not involved in the study design, data collection, analysis and interpretation of data, in the writing of this manuscript, or in the decision to submit it for publication.

References

1. Go AS, et al., Heart disease and stroke statistics--2013 update: a report from the American Heart Association. *Circulation*, 2013 127(1): p. e6–e245. [PubMed: 23239837]
2. Ford ES, Li C, and Sattar N, Metabolic Syndrome and Incident Diabetes Current state of the evidence. *Diabetes care*, 2008 31(9): p. 1898–1904. [PubMed: 18591398]
3. Ballantyne CM, et al., Metabolic syndrome risk for cardiovascular disease and diabetes in the ARIC study. *International journal of obesity* (2005), 2008 32(Suppl 2): p. S21. [PubMed: 18469836]
4. Alberti K, et al., Harmonizing the Metabolic Syndrome A Joint Interim Statement of the International Diabetes Federation Task Force on Epidemiology and Prevention; National Heart, Lung, and Blood Institute; American Heart Association; World Heart Federation; International Atherosclerosis Society; and International Association for the Study of Obesity. *Circulation*, 2009 120(16): p. 1640–1645. [PubMed: 19805654]
5. Heiss G, et al., Prevalence of metabolic syndrome among Hispanics/Latinos of diverse background: the Hispanic Community Health Study/Study of Latinos. *Diabetes care*, 2014 37(8): p. 2391–2399. [PubMed: 25061141]
6. Ennis S, Rios-Vargas M, and Albert N, The Hispanic Population 2010: 2010 Census Briefs. Washington, DC: US Census Bureau; 2011. 2014.
7. Piccolo RS, et al., Relative Contributions of Socioeconomic, Local Environmental, Psychosocial, Lifestyle/Behavioral, Biophysiological, and Ancestral Factors to Racial/Ethnic Disparities in Type 2 Diabetes. *Diabetes Care*, 2016 39(7): p. 1208–1217. [PubMed: 27330127]
8. Bradley RH and Corwyn RF, Socioeconomic status and child development. *Annual review of psychology*, 2002 53(1): p. 371–399.
9. Adler NE and Stewart J, Health disparities across the lifespan: meaning, methods, and mechanisms. *Annals of the New York Academy of Sciences*, 2010 1186(1): p. 5–23. [PubMed: 20201865]
10. Lucove JC, Kaufman JS, and James SA, Association between adult and childhood socioeconomic status and prevalence of the metabolic syndrome in African Americans: the Pitt County Study. *American Journal of Public Health*, 2007 97(2): p. 234–236. [PubMed: 17194854]
11. Chichlowska KL, et al., Individual and Neighborhood Socioeconomic Status Characteristics and Prevalence of Metabolic Syndrome. The Atherosclerosis Risk in Communities (ARIC) Study. *Psychosomatic medicine*, 2008 70(9): p. 986–992. [PubMed: 18799428]
12. Kang H-T, et al., Employment is associated with a lower prevalence of metabolic syndrome in postmenopausal women based on the 2007–2009 Korean National Health Examination and Nutrition Survey. *Menopause*, 2014 21(3): p. 221–226. [PubMed: 23760431]
13. Yang X, et al., The impact of socioeconomic status on the incidence of metabolic syndrome in a Taiwanese health screening population. *International journal of public health*, 2012 57(3): p. 551–559. [PubMed: 22349293]
14. Gupta R, et al., Association of educational, occupational and socioeconomic status with cardiovascular risk factors in Asian Indians: a cross-sectional study. 2012.
15. Gallo LC, et al., Socioeconomic status, psychosocial resources and risk, and cardiometabolic risk in Mexican-American women. *Health Psychology*, 2012 31(3): p. 334. [PubMed: 22059620]

16. Loucks EB, et al., Socioeconomic disparities in metabolic syndrome differ by gender: evidence from NHANES III. *Annals of epidemiology*, 2007 17(1): p. 19–26. [PubMed: 17140811]
17. Gallo LC, et al., The Hispanic community health study/study of Latinos sociocultural ancillary study: Sample, design, and procedures. *Ethnicity & disease*, 2014 24(1): p. 77. [PubMed: 24620452]
18. Kavanagh A, et al., Socioeconomic position, gender, health behaviours and biomarkers of cardiovascular disease and diabetes. *Social science & medicine*, 2010 71(6): p. 1150–1160. [PubMed: 20667641]
19. Dallongeville J, et al., Household income is associated with the risk of metabolic syndrome in a sex-specific manner. *Diabetes care*, 2005 28(2): p. 409–415. [PubMed: 15677801]
20. Ruiz JM, et al., The Hispanic health paradox: From epidemiological phenomenon to contribution opportunities for psychological science. *Group Processes & Intergroup Relations*, 2016 19(4): p. 462–476.
21. González HM, et al., Diabetes awareness and knowledge among Latinos: does a usual source of healthcare matter? *Journal of general internal medicine*, 2009 24(3): p. 528. [PubMed: 19842002]
22. Heisler M, et al., Mechanisms for racial and ethnic disparities in glycemic control in middle-aged and older Americans in the health and retirement study. *Archives of internal medicine*, 2007 167(17): p. 1853–1860. [PubMed: 17893306]
23. Sorlie PD, et al., Design and implementation of the Hispanic community health study/study of Latinos. *Annals of epidemiology*, 2010 20(8): p. 629–641. [PubMed: 20609343]
24. LaVange LM, et al., Sample design and cohort selection in the Hispanic Community Health Study/Study of Latinos. *Annals of epidemiology*, 2010 20(8): p. 642–649. [PubMed: 20609344]
25. Kagura J, et al., Association of socioeconomic status change between infancy and adolescence, and blood pressure, in South African young adults: Birth to Twenty Cohort. *BMJ Open*, 2016 6(3): p. e008805.
26. Blumenthal JA, et al., Effects of the dash diet alone and in combination with exercise and weight loss on blood pressure and cardiovascular biomarkers in men and women with high blood pressure: The encore study. *Archives of Internal Medicine*, 2010 170(2): p. 126–135. [PubMed: 20101007]
27. Arguelles W, et al., Characterization of metabolic syndrome among diverse Hispanics/Latinos living in the United States: Latent class analysis from the Hispanic Community Health Study/Study of Latinos (HCHS/SOL). *International journal of cardiology*, 2015 184: p. 373–379. [PubMed: 25745986]
28. Llabre MM, et al., Do all components of the metabolic syndrome cluster together in U.S. Hispanics/Latinos? Results from the Hispanic Community Health study/Study of Latinos. *Annals of epidemiology*, 2015 25(7): p. 480–485. [PubMed: 25818844]
29. Schneiderman N, et al., Prevalence of diabetes among hispanics/latinos from diverse backgrounds: the hispanic community health study/study of latinos (HCHS/SOL). *Diabetes care*, 2014 37(8): p. 2233–2239. [PubMed: 25061138]
30. McCurley JL, et al., Psychosocial Factors in the Relationship between Socioeconomic Status and Cardiometabolic Risk: the HCHS/SOL Sociocultural Ancillary Study. *Annals of Behavioral Medicine*, 2017 51(4): p. 477–488. [PubMed: 28130624]
31. Salsberry PJ, Corwin E, and Reagan PB, A complex web of risks for metabolic syndrome: race/ethnicity, economics, and gender. *American journal of preventive medicine*, 2007 33(2): p. 114–120. [PubMed: 17673098]
32. Elovainio M, et al., Socioeconomic differences in cardiometabolic factors: social causation or health-related selection? Evidence from the Whitehall II Cohort Study, 1991–2004. *American journal of epidemiology*, 2011: p. kwr149.
33. Galanti G-A, The Hispanic family and male-female relationships: An overview. *Journal of Transcultural Nursing*, 2003 14(3): p. 180–185. [PubMed: 12861920]
34. Thurston RC, et al., Is the association between socioeconomic position and coronary heart disease stronger in women than in men? *American journal of epidemiology*, 2005 162(1): p. 57–65. [PubMed: 15961587]

35. Rosero-Bixby L and Dow WH, Surprising SES gradients in mortality, health, and biomarkers in a Latin American population of adults. *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences*, 2009 64(1): p. 105–117.
36. Kutner M, et al., *The Health Literacy of America's Adults: Results from the 2003 National Assessment of Adult Literacy*. NCES 2006–483. National Center for Education Statistics, 2006.
37. Schumacher JR, et al., Potentially preventable use of emergency services: the role of low health literacy. *Medical care*, 2013 51(8): p. 654–658. [PubMed: 23703649]
38. DeVoe JE, et al., Receipt of preventive care among adults: insurance status and usual source of care. *American journal of public health*, 2003 93(5): p. 786–791. [PubMed: 12721145]
39. DeVoe JE, et al., Is health insurance enough? A usual source of care may be more important to ensure a child receives preventive health counseling. *Maternal and child health journal*, 2012 16(2): p. 306–315. [PubMed: 21373938]
40. US Department of Health Human Services. HHS action plan to reduce racial and ethnic health disparities: A nation free of disparities in health and health care. 2011; Available from: http://minorityhealth.hhs.gov/npa/files/plans/hhs/hhs_plan_complete.pdf.
41. McClurkin MA, et al., Health Insurance Status as a Barrier to Ideal Cardiovascular Health for US Adults: Data from the National Health and Nutrition Examination Survey (NHANES). *PloS one*, 2015 10(11): p. e0141534. [PubMed: 26535890]
42. Stringhini S, et al., Contribution of modifiable risk factors to social inequalities in type 2 diabetes: prospective Whitehall II cohort study. 2012.
43. Pampel FC, Krueger PM, and Denney JT, Socioeconomic disparities in health behaviors. *Annual review of sociology*, 2010 36: p. 349–370.

Table 1.

Prevalence of Metabolic Syndrome and Mean (95% CI) of Metabolic Syndrome Indicators by Sociodemographic Factors in the HCHS/SOL, 2008–2011 (N=14,029) *

	MetSyn % (95% CI)	Components above Threshold ^b	WC (cm)	TG (mg/dL) ^a	HDL-C (mg/ dL) ^a	SBP (mmHg)	DBP (mmHg)	FPG (mg/dL) ^a
Age					Mean (95% CI)			
18–44 years	21.8 (20.3 – 23.4)	1.5 (1.5 – 1.6)	96.1 (95.5 – 96.8)	122.4 (119.0, 125.8)	47.7 (47.3 – 48.2)	113.8 (113.4 – 114.3)	70.1 (69.7 – 70.6)	96.3 (95.4 – 97.1)
45–64 years	45.9 (44.1 – 47.7)	2.4 (2.3 – 2.4)	99.7 (99.3 – 100.1)	152.7 (149.1 – 156.3)	49.1 (48.6 – 49.6)	126.3 (125.8 – 126.9)	76.0 (75.6 – 76.4)	109.3 (107.8 – 110.8)
65+ years	58.7 (54.1 – 63.1)	2.8 (2.7 – 2.9)	101.1 (100.2 – 102.1)	143.3 (136.4 – 150.2)	50.7 (49.5 – 51.8)	138.0 (136.1 – 139.9)	72.7 (71.7 – 73.6)	112.9 (110.2 – 115.7)
Sex								
Women	35.2 (33.6 – 36.8)	2.0 (2.0 – 2.1)	97.1 (96.4 – 97.7)	121.9 (119.5 – 124.2)	52.0 (51.5 – 52.5)	117.5 (117.0 – 118.0)	71.0 (70.6 – 71.4)	100.2 (99.2 – 101.3)
Men	34.0 (32.5 – 35.6)	1.9 (1.8 – 1.9)	98.9 (98.4 – 99.4)	149.3 (145.4 – 153.2)	45.0 (44.5 – 45.4)	124.8 (124.3 – 125.3)	74.0 (73.6 – 74.4)	105.5 (104.5 – 106.6)
Hispanic background								
Dominican (N= 1209)	30.3 (27.7 – 33.1)	1.8 (1.8 – 1.9)	96.9 (95.4 – 98.3)	109.1 (105.0 – 113.2)	50.5 (49.6 – 51.4)	123.7 (122.5 – 125.0)	75.0 (74.2 – 75.8)	101.0 (98.6 – 103.4)
Central American (N= 1466)	34.6 (31.7 – 37.6)	2.0 (1.9 – 2.1)	95.7 (94.9 – 96.4)	148.9 (142.1 – 155.7)	48.4 (47.5 – 49.3)	123.3 (122.2 – 124.4)	73.3 (72.5 – 74.0)	103.6 (101.3 – 105.9)
Cuban (N= 1932)	34.9 (32.6 – 37.3)	2.0 (1.9 – 2)	97.2 (96.3 – 98.1)	139.8 (133.8 – 145.8)	47.4 (46.7 – 48.1)	122.6 (121.8 – 123.4)	74.6 (74.0 – 75.2)	100.6 (99.4 – 101.9)
Mexican (N= 5770)	34.9 (32.9 – 36.9)	2.0 (1.9 – 2.1)	98.6 (98.0 – 99.2)	140.5 (136.5 – 144.5)	48.4 (47.9 – 49.0)	119.1 (118.5 – 119.7)	70.4 (69.9 – 70.9)	104.7 (103.0 – 106.5)
Puerto Rican (N= 2278)	37.5 (34.6 – 40.5)	2.0 (2.0 – 2.1)	99.7 (98.7 – 100.7)	129.0 (122.1 – 135.9)	48.0 (47.0 – 49.0)	122.0 (121.2 – 122.9)	73.6 (72.9 – 74.2)	102.9 (101.2 – 104.6)
South American (N= 950)	25.6 (22.2 – 29.2)	1.6 (1.5 – 1.7)	93.4 (92.3 – 94.4)	131.4 (125.6 – 137.1)	50.3 (49.2 – 51.4)	118.7 (117.5 – 120.0)	70.0 (69.1 – 70.8)	98.7 (97.1 – 100.2)
Mixed/other (N= 424)	36.4 (29.8 – 43.5)	2.0 (1.8 – 2.1)	99.6 (97.2 – 102.0)	128.4 (117.7 – 139.1)	49.6 (47.8 – 51.3)	120.6 (118.6 – 122.6)	73.0 (71.6 – 74.4)	103.9 (99.4 – 108.4)
Health Insurance								
No	34.7 (32.9 – 36.5)	2.0 (1.9 – 2.0)	97.5 (96.9 – 98.0)	141.3 (138.0 – 144.5)	48.2 (47.7 – 48.8)	121.6 (120.9 – 122.3)	72.6 (72.2 – 73.1)	103.2 (102.0 – 104.4)

	MetSyn % (95% CI)	Components above Threshold ^b	WC (cm)	TG (mg/dL) ^a	HDL-C (mg/ dL) ^a	SBP (mmHg)	DBP (mmHg)	FPG (mg/dL) ^a
Yes	34.6 (33.0 – 36.3)	2.0 (1.9 – 2.0)	98.4 (97.8 – 99.0)	129.2 (125.7 – 132.7)	49.0 (48.6 – 49.5)	120.6 (120.1 – 121.1)	72.3 (71.9 – 72.7)	102.2 (101.1 – 103.2)

Abbreviations: CI: Confidence Interval; DBP: Diastolic Blood Pressure; FPG: Fasting Plasma Glucose; HCHS/SOL: Hispanic Community Health Study/Study of Latinos; HDL-C: High-density lipoprotein cholesterol; MetSyn: Metabolic Syndrome; SBP: Systolic Blood Pressure; TG: Triglycerides; WC: Waist Circumference.

* Continuous data are presented as mean (95% CI), and categorical data are presented as a percentage (95% CI). Values are weighted for study design and nonresponse, and are adjusted for age standardized to the US 2010 population.

^aSI conversions: To convert triglycerides to mmol/L, multiply by 0.0113; HDL-cholesterol to mmol/L, multiply by 0.0259; fasting glucose level to mmol/l, divide by 18

^bNumber of MetSyn components above diagnostic threshold, according to the harmonized definition of MetSyn

Table 2.

Prevalence of Metabolic Syndrome and Mean (95% CI) of Metabolic Syndrome Indicators by Socioeconomic Status in the HCHS/SOL, 2008–2011 (N=14,029)*

	MetSyn % (95% CI)	Components above Threshold ^b	WC (cm)	TG (mg/dL) ^a	HDL-C (mg/ dL) ^a	SBP (mmHg)	DBP (mmHg)	FPG (mg/dL) ^a
<i>Education</i>								
< High School	37.9 (36.2 – 39.7)	2.1 (2.0 – 2.1)	98.4 (97.7 – 99.0)	139.7 (136.2 – 143.2)	48.1 (47.5 – 48.7)	121.8 (121.2 – 122.5)	72.3 (71.9 – 72.8)	105.3 (103.7 – 106.8)
High School	34.9 (32.8 – 36.9)	2.0 (1.9 – 2.0)	98.2 (97.5 – 98.9)	133.9 (129.2 – 138.7)	48.2 (47.6 – 48.8)	121.3 (120.5 – 122.0)	72.4 (71.9 – 73.0)	103.1 (101.6 – 104.6)
> High School	32.1 (30.3 – 34.1)	1.9 (1.8 – 1.9)	97.5 (96.9 – 98.1)	133.0 (129.3 – 136.7)	49.3 (48.7 – 49.8)	120.3 (119.7 – 120.9)	72.7 (72.2 – 73.1)	100.9 (99.8 – 102.0)
<i>Household Income</i>								
< \$20,000	36.3 (34.6 – 38.0)	2.1 (2.0 – 2.1)	98.0 (97.4 – 98.5)	137.3 (133.8 – 140.8)	48.4 (47.9 – 49.0)	121.3 (120.7 – 121.9)	72.7 (72.3 – 73.1)	103.4 (102.2 – 104.6)
\$20,001–\$40,000	35.4 (33.6 – 37.3)	2.0 (1.9 – 2.0)	98.1 (97.5 – 98.8)	137.5 (133.3 – 141.6)	48.2 (47.7 – 48.7)	121.1 (120.5 – 121.8)	72.5 (72.0 – 73.0)	103.1 (101.7 – 104.4)
\$40,001–\$75,000	31.8 (28.8 – 34.9)	1.8 (1.8 – 1.9)	97.8 (96.9 – 98.6)	128.7 (123.3 – 134.2)	49.3 (48.4 – 50.2)	120.7 (119.9 – 121.6)	72.4 (71.8 – 73.0)	101.2 (99.2 – 103.2)
> \$75,000	26.1 (21.8 – 30.9)	1.6 (1.5 – 1.8)	97.8 (96.2 – 99.3)	122.2 (112.8 – 131.7)	49.2 (47.8 – 50.6)	119.7 (118.2 – 121.2)	71.0 (69.5 – 72.5)	100.4 (97.4 – 103.5)
<i>Employment Status</i>								
Unemployed	49.0 (39.6 – 58.5)	2.5 (2.3 – 2.8)	105.4 (100.4 – 110.3)	142.9 (127.9 – 157.9)	47.6 (45.3 – 49.9)	125.8 (122.4 – 129.3)	76.3 (73.5 – 79.0)	49.0 (39.6 – 58.5)
Retired	37.7 (35.8 – 39.7)	2.1 (2.1 – 2.2)	98.6 (98.0 – 99.3)	139.5 (136.3 – 142.7)	48.5 (48 – 49.1)	120.5 (119.9 – 121.2)	72.5 (72.1 – 73.0)	37.7 (35.8 – 39.7)
Part time employed	31.5 (28.6 – 34.6)	1.9 (1.8 – 1.9)	96.8 (95.9 – 97.7)	126.7 (122.5 – 130.8)	49.3 (48.7 – 50.0)	119.4 (118.5 – 120.3)	71.1 (70.4 – 71.7)	31.5 (28.6 – 34.6)
Full time employed	31.2 (28.8 – 33.6)	1.8 (1.8 – 1.9)	97.4 (96.6 – 98.2)	134.5 (130.3 – 138.8)	48.4 (47.8 – 48.9)	122.6 (121.7 – 123.5)	73.3 (72.8 – 73.8)	31.2 (28.8 – 33.6)

Abbreviations: CI: Confidence Interval; DBP: Diastolic Blood Pressure; FPG: Fasting Plasma Glucose; HCHS/SOL: Hispanic Community Health Study/Study of Latinos; HDL-C: High-density lipoprotein cholesterol; MetSyn: Metabolic Syndrome; SBP: Systolic Blood Pressure; TG: Triglycerides; WC: Waist Circumference.

* Continuous data are presented as mean (95% CI), and categorical data are presented as a percentage (95% CI). Values are weighted for study design and nonresponse, and are adjusted for age standardized to the US 2010 population.

^aSI conversions: To convert triglycerides to mmol/L, multiply by 0.0113; HDL-cholesterol to mmol/L, multiply by 0.0259; fasting glucose level to mmol/l, divide by 18

N_q Number of MetSyn components above diagnostic threshold, according to the harmonized definition of MetSyn

Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript

Table 3.

Association of the SES Proxy Variables with Metabolic Syndrome and its Components by Sex and Health Insurance in the HCHS/SOL, 2008–2011 (N=14,029)[‡]

	MetS	WC (cm)	TG (mg/dL) ^a	HDL-C (mg/dL) ^a	SBP (mmHg)	DBP (mmHg)	FPG (mg/dL) ^a
	OR (95% CI) ^b			Unstandardized Beta (95% CI) ^b			
Income	0.96 (0.93 – 0.98)*	-0.22 (-0.38 – -0.07)*	-0.36 (-0.64 – -0.08)	0.24 (0.11 – 0.38)*	-0.21 (-0.36 – -0.05)*	-0.04 (-0.14 – 0.07)	-0.14 (-0.24 – -0.05)*
Males	0.99 (0.95 – 1.02)	0.10 (-0.12 – 0.31)	---	0.03 (-0.15 – 0.20)	---	---	---
Females	0.92 (0.90 – 0.95)*	-0.54 (-0.75 – -0.33)*	---	0.44 (0.25 – 0.63)*	---	---	---
No Current Health Insurance	1.01 (0.98 – 1.05)	0.10 (-0.14 – 0.33)	---	---	0.02 (-0.19 – 0.23)	---	0.04 (-0.11 – 0.19)
Current Health Insurance	0.92 (0.89 – 0.95)*	-0.42 (-0.62 – -0.22)*	---	---	-0.33 (-0.53 – -0.14)*	---	-0.26 (-0.38 – -0.14)*
Education	0.97 (0.96 – 0.98)*	-0.19 (-0.28 – -0.10)*	-0.15 (-0.29 – 0.00)	0.12 (0.05 – 0.19)*	-0.27 (-0.37 – -0.17)*	0.04 (-0.02 – 0.10)	-0.15 (-0.21 – -0.09)*
Males	---	0.04 (-0.08 – 0.15)	0.16 (-0.08 – -0.39)	-0.02 (-0.12 – 0.08)	-0.18 (-0.33 – -0.04)	---	---
Females	---	-0.40 (-0.53 – -0.27)*	-0.40 (-0.57 – -0.23)*	0.25 (0.14 – 0.36)*	-0.30 (-0.43 – -0.18)*	---	---
No Current Health Insurance	---	---	---	-0.02 (-0.13, 0.08)	---	---	---
Current Health Insurance	---	---	---	0.25 (0.15, 0.34)*	---	---	---
Full-time Employed versus Unemployed	0.76 (0.67 – 0.85)*	-1.44 (-2.25 – -0.64)*	-3.22 (-4.45 – -1.98)*	1.58 (0.91 – 2.25)*	0.21 (-0.59 – 1.00)	0.46 (-0.12 – 1.05)	-1.21 (-1.70 – -0.72)*
Males	---	---	-2.21 (-4.24 – -0.18)*	0.14 (-0.81 – 1.09)	0.04 (-0.97 – 1.05)	---	---
Females	---	---	-4.91 (-6.39 – -3.42)*	2.98 (2.00 – 3.96)*	-0.22 (-1.28 – 0.83)	---	---
No Current Health Insurance	---	---	---	---	---	---	---
Current Health Insurance	---	---	---	---	---	---	---

Abbreviations: DBP: Diastolic Blood Pressure; FPG: Fasting Plasma Glucose; HCHS/SOL: Hispanic Community Health Study/Study of Latinos; HDL-C: High-density lipoprotein cholesterol; MetSyn: Metabolic Syndrome; SBP: Systolic Blood Pressure; TG: Triglycerides; WC: Waist Circumference.

Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript

$P < .01$
*

^aSI conversions: To convert triglycerides to mmol/L, multiply by 0.0113; HDL-cholesterol to mmol/L, multiply by 0.0259; fasting glucose level to mmol/L, divide by 18.

^bAll models adjusted for age, sex, field center, Hispanic/Latino background, health insurance, and nativity/length of residence in the U.S.

Table 4.

Differences in SES components Household Income, Education, and Occupation by Sex and Health Insurance in the HCHS/SOL, 2008–2011 (N=14,029)[§]

	Income		Education		Occupation	
	M (SE)		M (SE) years	% Full-time	% Part-time	
Sex						
Women	3.9 (0.1)		11.9 (0.1)	25.7% (0.7)	19.9% (0.7)	
Men	4.6 (0.1) [*]		12.0 (0.1)	46.7% (1.1) [*]	14.8% (0.6) [*]	
Health Insurance						
No	4.0 (0.1)		11.7 (0.1)	36.7% (0.9)	20.5% (0.7)	
Yes	4.6 (0.1) [*]		12.1 (0.1) [*]	35.2% (1.0)	14.4% (0.7) [*]	

Abbreviations: DBP: Diastolic Blood Pressure; FPG: Fasting Plasma Glucose; HCHS/SOL: Hispanic Community Health Study/Study of Latinos; HDL-C: High-density lipoprotein cholesterol; SBP: Systolic Blood Pressure; TG: Triglycerides; WC: Waist Circumference.

[§]Values are weighted for study design and nonresponse

^{*}Significant difference from other sex or health insurance category at the $p < .05$ level

^aSI conversions: To convert triglycerides to mmol/L, multiply by 0.0113; HDL-cholesterol to mmol/L, multiply by 0.0259; fasting glucose level to mmol/l, divide by 18