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Do all components of the metabolic syndrome cluster together in US Hispanics/Latinos? Results from the Hispanic Community Health Study (HCHS/SOL)

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Abstract

Purpose—Metabolic syndrome (MetS), the clustering of several risk factors for cardiovascular disease, is highly prevalent in Hispanics/Latinos. We tested whether all components significantly loaded on the syndrome in Hispanics/Latinos and whether their contribution differed by sex and Hispanic ancestry. We also examined associations of metabolic syndrome with prevalent diabetes and coronary heart disease (CHD) in Hispanics/Latinos.

Methods—Data were obtained from a population-based cohort of N = 15,823 participants in the HCHS/SOL who self-identified as being of Central American, Cuban, Dominican, Mexican American, Puerto Rican, or South American ancestry, aged 18-74 years at screening.

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Maria M. Llabre had full access to all of the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

M.L. also researched the literature, interpreted data, organized and wrote the manuscript, except for the methods. W.A. wrote the methods and critically reviewed, edited, and contributed to the interpretation of data. D.S-A. reviewed the data analysis, and edited and contributed to the interpretation of the data. N.S., L.C.G, G.A.T., and S.F.C. edited and contributed to the interpretation of the data. M.L.D., E.C.C., D.A.C., S.C.R., and G.H. were involved in manuscript preparation.

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Preliminary data from this study were presented in a symposium at the 71st Scientific Sessions of the American Diabetes Association, San Diego, CA, June 24-28, 2012.

Results—A latent variable model of waist circumference, systolic and diastolic blood pressure, triglycerides, high-density lipoprotein cholesterol (HDL-C), and fasting glucose fit the data in men and women, but the contribution of HDL-C was weak. No difference in the latent model of MetS was detected across Hispanic/Latino ancestry groups. MetS was significantly associated with diabetes and CHD.

Conclusions—Our results indicate that similar criteria for MetS may be applied across Hispanic/Latino ancestry groups; but call into question the role of HDL-C in classifying the MetS in Hispanics/Latinos.

Keywords

Metabolic syndrome; Hispanics; Lipids

Introduction

Metabolic syndrome (MetS) refers to the clustering of several risk factors that together confer increased risk for cardiovascular disease (CVD). The interrelated factors include obesity, hyperglycemia, dyslipidemia, and hypertension. According to a unified definition, the presence of three or more of following risk factors results in a diagnosis of MetS: 1) waist circumference 102 cm in US men and 88 cm in US women, 2) systolic/diastolic blood pressure 130/ 85 mm Hg or use of antihypertensive medication, 3) high-density lipoprotein (HDL) cholesterol <40 mg/dL in men and <50 mg/dL in women or use of cholesterol medication, 4) triglycerides 150 mg/dL or use of lipid lowering medication, and 5) fasting glucose 100 mg/dL or use of medication [1]. Presence of MetS increases the risk of CVD [1, 2] and type 2 diabetes (T2D) [3].

Data on the prevalence of MetS among US Hispanics/Latino, based primarily on Mexican Americans, indicate increased rates of MetS relative to whites or blacks[4]. Ancestry group comparisons from the Multi-Ethnic Study of Atherosclerosis showed that Mexican Americans had a higher prevalence of MetS compared to Puerto Ricans and other ancestry groups in the US, suggesting that Hispanic/Latinos do not represent a homogeneous group with respect to MetS prevalence.

Furthermore, the prevalence of some individual MetS components has been shown to be higher for Hispanics/Latinos than non-Hispanic whites, including overweight or obesity and dyslipidemia [4]. Hispanics tend to have lower levels of HDL-C cholesterol in comparison to Blacks or non-Hispanic Whites [5, 6], and higher triglycerides [5]. Interestingly, HDL-C has not been shown to reliably predict myocardial infarction in Hispanics/Latinos, as shown in non-Hispanic Whites [7]. Prevalence of hypertension, on the other hand, has been reported to be lower in Hispanics/Latinos compared to non-Hispanic Whites [4].

Similar to MetS, diabetes appears to be disproportionately prevalent among Hispanic/ Latinos relative to that of whites and blacks [4]. Paradoxically, prevalence of CVD is lower among US Mexican Americans than among whites and blacks [4]. Researchers often refer to the unexpected lower CVD rates in a relatively more disadvantaged population as the "Hispanic Paradox" [8, 9].

The HCHS/SOL provides a unique opportunity to examine the MetS in Hispanic/Latinos and address fundamental questions, as well as methodological shortcomings in the quantification of the MetS. Several methodological criticisms related to MetS have been elucidated by Khan et al [10], who point out that there is no empirical rationale for the inclusion (or exclusion) of specific indicators, nor for the cutoff values specified in existing MetS criteria, which appear arbitrary, given that biological levels of the individual MetS components are continuous. It is possible that the risk factors and/or cut-off values do not apply in Hispanics/Latinos and their routine application contributes to the Hispanic Paradox. To overcome these methodological shortcomings, we specify a latent variable model using the current indicators of MetS.

In the most common latent variable measurement model, it is assumed that there is an underlying construct or mechanism that explains the covariation among a set of measures. Each measure or indictor is linearly related to its latent variable and is expected to contain some error or residual variation. The indicators are measured as continuous variables without the need to impose cutoffs. The model allows estimation of the contribution of each component to the underlying construct, the MetS.

We use this latent variable model approach, consistent with the notion of MetS, to address a fundamental question: Do all components of MetS cluster together in Hispanic/Latinos? A related question is whether there are differences in the contribution of each component to the MetS as a function of sex or Hispanic/Latino ancestry group? We will also assess whether MetS, assessed using continuous measures, is associated with prevalent diagnosis of diabetes and CHD in Hispanic/Latinos.

Methods

Participants

The purpose, design, and methods of the HCHS/SOL have previously been reported [11, 12]. Participants were recruited in 4 US communities: the Bronx, NY, Chicago, IL, Miami, FL, and San Diego, CA. A two-stage stratified probability sampling plan was used and previously described [12]. The study protocol was approved by the institutional review board at each site and informed consent was obtained from all participants. Out of the 16,415 eligible participants between 18 and 74 years old who were assessed at baseline between 2008-2011, N = 15,823 identified themselves as being of Central American, Cuban, Dominican, Mexican American, Puerto Rican, or South American ancestry. Those of mixed background or who identified as "other" were excluded from the current analyses. Two participants with missing data on all components of the metabolic syndrome were also excluded from analysis; thus the analytic sample size was N = 15823.

Measures

MetS Components—The indicators used in this study are those consistent with current definitions of metabolic syndrome. Waist circumference was measured to the nearest 0.1 cm at the uppermost lateral border of the right ilium using a measuring tape. After 5 minutes in the seated position, systolic (SBP) and diastolic blood pressure (DBP) were measured 3

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times at 1 minute intervals using an automatic sphygmomanometer (Omron model HEM-907 XL, Omron Healthcare Inc., Bannockburn, IL), and the average of the 3 readings was used. Measurements of high-density lipoprotein cholesterol (HDL-C), triglycerides (TG), and glucose were obtained from fasting blood samples.

Prevalent CHD and Diabetes—Each participant received a standard digital 12-lead electrocardiogram (ECG; GEMSIT MAC 1200 portable electrocardiograph) and readings were electronically transmitted to a Central ECG Reading Center (The Epidemiological Cardiology Research Center of Wake Forest University's School of Medicine). Guidelines to determine wave duration and voltage following the Minnesota Code were used to ascertain possible old myocardial infarction (MI). ECG classification criteria for MI by the Minnesota Code are detailed elsewhere [13]. Self-reported information on angina, heart attack, and coronary procedures (angioplasty, stent, or bypass surgery to the arteries of the heart) was collected via standard questionnaire and interview. Prevalent coronary heart disease (CHD) was specified as a dichotomous variable that combined information from ECG reports of possible old MI as well as self-report of heart attack, coronary procedures, and angina.

Diabetes was also specified as a dichotomous variable based on the American Diabetes Association definition, taking into account serum glucose levels adjusted for fasting time and, if available, glucose level 2-hr after a 75g glucose load, glycosylated hemoglobin A1c %, scanned/transcribed anti-glycemic medication use, or self-report of diabetes.

Covariates—Standard questionnaires and interviews were used to collect information on age, sex, Hispanic/Latino ancestry group, current or previous smoking history, education and total household income. Age was examined as a continuous variable. Sex was examined as a dichotomous variable. Use of lipid lowering, diabetes, and hypertension medication was coded as a dichotomous variable. Hispanic/Latino ancestry group was dummy coded with five vectors for Puerto Rican, Cuban, Dominican, Central American, or South American, with Mexicans serving as the reference group.

Procedures

Statistical Analysis—We examined a latent variable model for MetS as most consistent with the notion of a common underlying pathophysiology using confirmatory factor analysis (CFA), where each indicator was assumed to be a manifestation of the syndrome. The commonality or common variance among the indicators is represented by a latent variable. We tested whether such a model represented good fit to the data. Initially, we assumed uncorrelated residuals except for SBP with DBP and HDL-C with triglycerides, but relaxed the assumption based on modification indices suggesting added correlations between waist with HDL-C and DBP. We then examined the correlations between the latent variable of MetS and each of its indicators, also known as factor loadings. The model was first examined in men and women separately, as metabolic syndrome criteria vary by sex. We then conducted a multiple group analysis to determine the equality of factor loadings between sex groups. We systematically compared each unstandardized factor loading using a chi-squared difference test appropriate for the maximum likelihood robust method of

estimation. This test compares two nested models, one where the loading in question is constrained equal between groups to one where the loadings are freely estimated in each group. Similarly, the model was examined in each Hispanic/Latino ancestry group and compared across ancestry groups using multiple group CFA. Lastly, we examined associations between MetS and diagnosis of diabetes and CHD using logistic regression, controlling for smoking history, use of medication, and demographic covariates.

Results

Preliminary Analyses

All variable distributions were examined for the presence of outliers, skewness, and kurtosis and found to approximate normality, once glucose and triglycerides were log transformed. Descriptive statistics were computed on all measures. Table 1 displays the means and standard deviations on all measures for participants with complete data.

The initial analysis was conducted on the sample of men (n = 6316) and women (n = 9507) separately. Correlations among the six indicators are shown in Table 2 for women and men. Several observations are worth noting. The correlations are very similar for both sexes and generally modest in magnitude with the exception of correlations between the two measures of blood pressure and the two lipid measures. The single indicator most consistently associated with the others is waist circumference. Also, of all the indicators, HDL-C has the weakest associations with the others.

Confirmatory Factor Analysis Model

The model specified the existence of a continuous underlying construct (latent variable), responsible for the clustering or covariation among the indicators that comprise it. We selected the CFA model with effect indicators, given that the MetS was assumed to occur on a continuum and all indicators were measured concurrently. To determine whether such a model fits the data from the HCHS/SOL cohort, we conducted a CFA using the software Mplus v7.0 (Muthen & Muthen, 1998-2010) and incorporated the sampling weights, stratification, and clustering features of the study design.

Assessment of model fit was based on several common indices [Comparative Fit Index (CFI > .95), the Root Mean Squared Error of Approximation (RMSEA < .06), and the Standardized Residuals (SRMR < .10)]. We also report the results of the chi-squared test of exact fit, but do not use it in model evaluation because such a test is highly dependent on sample size and expected to be significant with our large sample, even if departures from exact fit are small. The model fit the data for both men [CFI = .97; RMSEA = .06 90%CI (. 051-.069); SRMR = .022; χ^2 (5) = 117.67, p < .01)] and women [CFI = .97; RMSEA = .055 90%CI (0.048-0.063); SRMR = .029; χ^2 (5) = 148.56, p < .01)]. This model included residual correlations between SBP and DBP, both measures of blood pressure and between HDL-C and triglycerides, both indicators of lipids. We also added correlated residuals between waist with DBP and HDL-C.

Unstandardized and standardized factor loadings are reported in Table 3 with corresponding 95% confidence intervals. The loadings were all statistically significant (p < .001), and the

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unstandardized loadings seemed comparable between men and women, except for the loadings associated with systolic blood pressure. The loading for SBP was stronger for women than for men $\chi^{-2}(1) = 192.61$, p < .01.

Standardized loadings represent the correlations between the factor and the corresponding indicator. In the factor analysis literature, it is common to use a criterion of a correlation of . 30 or greater as indicating an adequate indicator of a factor. As shown in Table 3, the standardized loadings associated with HDL-C do not meet this criterion, suggesting that HDL-C does not cluster together as strongly with the other risk factors that define MetS in Hispanic/Latino men or women. At best, HDL-C appears to be a weak indicator of the underlying syndrome in a single factor model.

Hispanic/Latino Ancestry Group Comparisons

Hispanic/Latino ancestry group comparisons were performed for men and women, separately, to compare equality of loadings among Mexican (n = 4021 women; n = 2449 men) Puerto Rican (n = 1589 women; n = 1139 men), Central American (n = 1049 women; n = 683 men), Cuban (n = 1250 women; n = 1098 men), Dominican (n = 963 women; n = 510 men), or South American (n = 635 women; n = 437 men). The model with loadings constrained equal across ancestry groups fit the data for men [CFI = .97; RMSEA = .049 CI (.041-.056); SRMR = .08; χ^2 (55) = 192.61, p < .01] and women [CFI = .96; RMSEA = .049 CI (.043-.055); SRMR = .075; χ^2 (55) = 262.44, p < .01], indicating the clustering of risk factors that form MetS is comparable across Hispanic/Latino subpopulations living in the US.

Diabetes and CHD Associations

Lastly, the latent variable of MetS along with age, education, income, current smoker, and previous smoker, was used in a logistic regression to predict prevalent CHD and diabetes in the sample. The results showed significant associations with both CHD and diabetes. With respect to diabetes, adjusting for age, education, income, smoking and use of medication (diabetes, lipid, hypertension), the odds ratio (OR) per standard deviation unit in the standardized MetS latent variable was 2.39 [95% CI 2.25-2.55] for men and 2.78 [95% CI 2.60-2.97] for women. With respect to CHD and the same covariates, the OR was 1.18 [95% CI 1.08-1.29] for men and 1.22 [95% CI 1.11-1.35] for women. This means that for every one standard deviation increase in MetS, the odds of having CHD increase by about 20%, while the odds of having diabetes increase over 130% for both men and women. In unadjusted models (results not shown) these OR's were slightly lower than in the adjusted models when predicting diabetes but slightly higher when predicting CHD.

Discussion

This study confirmed that current MetS indicators measured as continuous variables generally cluster together, as expected, in the largest sample of Hispanics/Latinos studied in the US. However, within a single factor, the data indicate that HDL-C does not load strongly on the MetS factor. Comparative results indicated similarity between men and women except for the contribution of blood pressure to MetS, and across Hispanic/Latino ancestry

groups. Systolic blood pressure was a stronger indicator for women relative to men. Lastly, results confirmed associations between MetS, measured as a continuous latent variable, and prevalent CHD and diabetes diagnoses in Hispanics/Latinos, consistent with prior reports of a dose-response association between MetS components and disease (see [14] for review).

It was expected that the latent model analysis would confirm the clustering of current risk factors known as MetS in a sample of Hispanic/Latinos. Our results were consistent with the work of Shen et al.[15, 16] and others[17] who used similar methodology to support a single factor model of MetS in non-Hispanic samples. We extended that work by demonstrating comparability in the clustering presentation across Hispanic/Latino ancestry groups. Earlier work based on a principal component analysis of the Framingham study data suggested three factors [18], and questioned the notion of a single pathophysiological mechanism underlying the clustering. More recent work in children, with an expanded set of indicators that included multiple measures of glucose metabolism and an optimizing methodology [19], suggested two distinct underlying mechanisms: "impaired glucose metabolism" and "impaired lipid metabolism"

Given our results indicating a single factor model fit the data may lead one to conclude that MetS has a single etiology. However, we caution against this interpretation. In fact, the model that fit the data suggested that beyond the single MetS clustering, waist circumference was associated with diastolic blood pressure and HDL-C, indicating multiple roles of waist circumference, perhaps through different pathophysiological systems, in accounting for associations among components of MetS. Also, while HDL-C did not have a strong loading on the MetS factor, it correlated with waist circumference. In other analyses of the prevalence of MetS components in these data [20], waist circumference was also the most prevalent component, underscoring the salience and complexity of this risk factor in the context of MetS.

The work of Peeters and colleagues with data from overweight children and adolescents [19], would suggest that not a single, but multiple physiological pathways underlie the phenotypic expression of the metabolic syndrome. Certainly, the weak loading of HDL-C on the MetS factor in our own data coupled with the need to correlate residuals associated with the waist circumference indicator, are clues that with a broader set of indicators, a two factor solution might have been replicable in our sample. The choice to restrict our analysis to the current indicators of the MetS prevented us from being able to conduct a fair test of the two factor hypothesis generated by the Peeters et al. study. Further work with a broader set on indicators in a Hispanic/Latino population would be both interesting and important.

However, the weak association of HDL-C with the MetS in the current study was unexpected as previous CFA studies of the structure of MetS using non-Hispanic cohorts reported stronger loadings for HDL-C on the MetS factor than what we found [15,16]. In the one study that included Hispanic/Latinos, Shen et al [15] reported similarity between Caucasians and Cubans, but the samples were too small (211 Caucasians and 135 Cubans) to draw definitive conclusions. Our results suggest that, in the context of a single factor model, HDL-C is not a good indicator of MetS in Hispanic/Latinos. HDL-C, however, was also not significantly associated with risk of MI among Hispanics in the NOMAS study or the San

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Antonio Heart Study [7, 21]. Other data from samples of Hispanic/Latinos [22], show that Hispanic/Latinos have low HDL-C levels relative to non-Hispanic whites and blacks. As noted earlier, the age-adjusted prevalence of MetS is higher in Hispanic/Latinos relative to blacks or whites, yet the prevalence of CHD is not. Our findings indicate that using HDL-C to classify the MetS in Hispanics/Latinos may increase the rates of false positive classifications, thus reducing specificity.

If HDL is to be considered in defining MetS, the commonly assayed HDL-C level may not be the most informative parameter, as it may not capture the cardioprotection related to HDL. The heterogeneity in HDL particles which vary in concentration, shape, and size has led to investigations suggesting that particle-based measures [23] rather than cholesterol measures might be more promising in identifying successful therapies [24, 25]. Indices that capture cholesterol efflux capacity to reverse cholesterol transport from macrophages have shown associations with presence of CHD [26]. While HDL-C levels are correlated with HDL efflux capacity, the correlation is not perfect and has not been reported in Hispanic/ Latinos. The HDL-C hypothesis is currently being debated and supplemented by the HDL efflux hypothesis[27]. A recent large-scale study of HDL particle subclasses on the basis of size (very large, large, medium, small, very small) provides a more nuanced examination of the differential associations between HDL particle subclasses and incident CHD [24]. These notions need to find their way into the incorporation of other HDL indices in MetS definitions.

A recent publication on CHD risk factors in the HCHS/SOL reported heterogeneity among the Hispanic/Latino ancestry groups with respect to levels of the individual MetS components [28]. This heterogeneity in MetS component levels across ancestry groups does not necessarily translate to differential contribution of the specific components to the MetS. In fact, we found a striking similarity in the contribution of the MetS components among all ancestry groups for both men and women.

We also raise the question of sex differences in the role of blood pressure in the MetS. Given that this study was limited to Hispanics/Latinos, it will be important to examine whether a similar pattern of sex differences in the contribution of blood pressure to the MetS is present in other racial/ethnic groups to determine the generalizability of this finding. Furthermore, the target population was limited to four field centers in four communities, which are among the ten metropolitan areas with the largest Hispanic/Latino concentration in the US. Thus the geographic representation is limited.

Two other important limitations must be considered. Medication use for blood pressure and diabetes is taken into account in the MetS classification. In our analyses, participants on medication may have negatively biased covariance estimates, as their levels on specific indicators would likely regress towards the mean as a result of medication. To assess the extent of this potential problem, we repeated the initial models excluding participants on medication. As the pattern of results did not change, we believe our conclusions are not biased by medication. Nonetheless, how to best account for medication in a latent variable model remains a topic for future work. Controlling for medication alone is not sufficient in cases where patterns might be moderated by medication. Also, we did not have prospective

data at this time on incident CHD and diabetes in the cohort, thus our findings reflect crosssectional associations. With the recent funding for the collection of important follow-up data, we will be able to report on the extent to which our MetS model can predict future diagnoses and/or events.

Despite these limitations, our analysis demonstrates the advantages of a latent variable model approach to defining the MetS. First, it is consistent with the concept and allows specific examination of the importance of each component. Prior studies based on the current criteria have assumed that all components are equally important. Yet we showed that this assumption is not necessarily valid in Hispanics/Latinos with respect to HDL-C. Given that the components are examined as continuous variables, this latent variable approach does not impose an arbitrary cutoff and provides information on degree rather than simply presence or absence of risk factors. Further work is needed to examine the sensitivity and specificity of various cut-points, particularly for Hispanics/Latinos.

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Abbreviations

MetS	Metabolic syndrome
CVD	cardiovascular disease
T2D	type 2 diabetes
SBP	systolic
DBP	diastolic blood pressure
HDL-C	high-density lipoprotein cholesterol
TG	triglycerides
ECG	electrocardiogram
MI	myocardial infarction
CFA	confirmatory factor analysis
CFI	Comparative Fit Index
RMSEA	Root Mean Squared Error of Approximation
SRMR	Standardized Residuals

References

- Alberti KG, Eckel RH, Grundy SM, Zimmet PZ, Cleeman JI, Donato KA, 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. Oct 20; 2009 120(16):1640–5. PubMed PMID: 19805654. [PubMed: 19805654]
- Executive Summary of The Third Report of The National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, And Treatment of High Blood Cholesterol In Adults (Adult Treatment Panel III). JAMA : the journal of the American Medical Association. May 16; 2001 285(19):2486–97. PubMed PMID: 11368702. Epub 2001/05/23. eng.
- Lorenzo C, Okoloise M, Williams K, Stern MP, Haffner SM. The metabolic syndrome as predictor of type 2 diabetes: the San Antonio heart study. Diabetes Care. Nov; 2003 26(11):3153–9. PubMed PMID: 14578254. Epub 2003/10/28. eng. [PubMed: 14578254]
- Roger VL, Go AS, Lloyd-Jones DM, Adams RJ, Berry JD, Brown TM, et al. Heart disease and stroke statistics--2011 update: a report from the American Heart Association. Circulation. Feb 1; 2011 123(4):e18–e209. PubMed PMID: 21160056. [PubMed: 21160056]
- 5. Haffner SM, Stern MP, Hazuda HP, Rosenthal M, Knapp JA. The role of behavioral variables and fat patterning in explaining ethnic differences in serum lipids and lipoproteins. American journal of epidemiology. May; 1986 123(5):830–9. PubMed PMID: 3962965. [PubMed: 3962965]
- Burchfiel CM, Hamman RF, Marshall JA, Baxter J, Kahn LB, Amirani JJ. Cardiovascular risk factors and impaired glucose tolerance: the San Luis Valley Diabetes Study. American journal of epidemiology. Jan; 1990 131(1):57–70. PubMed PMID: 2293753. [PubMed: 2293753]
- Willey JZ, Rodriguez CJ, Carlino RF, Moon YP, Paik MC, Boden-Albala B, et al. Race-ethnic differences in the association between lipid profile components and risk of myocardial infarction: The Northern Manhattan Study. Am Heart J. May; 2011 161(5):886–92. PubMed PMID: 21570518. Pubmed Central PMCID: 3095911. Epub 2011/05/17. eng. [PubMed: 21570518]
- Franzini L, Ribble JC, Keddie AM. Understanding the Hispanic paradox. Ethnicity & disease. 2001; 11(3):496–518. Autumn. PubMed PMID: 11572416. Epub 2001/09/27. eng. [PubMed: 11572416]
- Sorlie PD, Backlund E, Johnson NJ, Rogot E. Mortality by Hispanic status in the United States. JAMA : the journal of the American Medical Association. Nov 24; 1993 270(20):2464–8. PubMed PMID: 8031341. Epub 1993/11/24. eng.
- Kahn R, Buse J, Ferrannini E, Stern M, American Diabetes A, European Association for the Study of D. The metabolic syndrome: time for a critical appraisal: joint statement from the American Diabetes Association and the European Association for the Study of Diabetes. Diabetes care. Sep; 2005 28(9):2289–304. PubMed PMID: 16123508. [PubMed: 16123508]
- Sorlie PD, Aviles-Santa LM, Wassertheil-Smoller S, Kaplan RC, Daviglus ML, Giachello AL, et al. Design and implementation of the Hispanic Community Health Study/Study of Latinos. Annals of epidemiology. Aug; 2010 20(8):629–41. PubMed PMID: 20609343. Pubmed Central PMCID: 2904957. [PubMed: 20609343]
- Lavange LM, Kalsbeek WD, Sorlie PD, Aviles-Santa LM, Kaplan RC, Barnhart J, et al. Sample design and cohort selection in the Hispanic Community Health Study/Study of Latinos. Annals of epidemiology. Aug; 2010 20(8):642–9. PubMed PMID: 20609344. Pubmed Central PMCID: 2921622. [PubMed: 20609344]
- 13. Prineas, RJ.; Crow, RS.; Zhang, Z. Standards and Procedures for ECG Measurement in Epidemiologic and Clinical Trials. Springer; 2009. The Minnesota Code Manual of Electrocardiographic Findings: Including Measurement and Comparison with the Novacode.
- Meigs JB. Epidemiology of type 2 diabetes and cardiovascular disease: translation from population to prevention: the Kelly West award lecture 2009. Diabetes care. Aug; 2010 33(8):1865–71. PubMed PMID: 20668155. Pubmed Central PMCID: 2909080. [PubMed: 20668155]
- Shen BJ, Goldberg RB, Llabre MM, Schneiderman N. Is the factor structure of the metabolic syndrome comparable between men and women and across three ethnic groups: the Miami Community Health Study. Annals of epidemiology. Feb; 2006 16(2):131–7. PubMed PMID: 16257230. Epub 2005/11/01. eng. [PubMed: 16257230]

- Shen BJ, Todaro JF, Niaura R, McCaffery JM, Zhang J, Spiro A 3rd, et al. Are metabolic risk factors one unified syndrome? Modeling the structure of the metabolic syndrome X. American journal of epidemiology. Apr 15; 2003 157(8):701–11. PubMed PMID: 12697574. Epub 2003/04/17. eng. [PubMed: 12697574]
- Stevenson JE, Wright BR, Boydstun AS. The metabolic syndrome and coronary artery disease: a structural equation modeling approach suggestive of a common underlying pathophysiology. Metabolism: clinical and experimental. Nov; 2012 61(11):1582–8. PubMed PMID: 22626764. [PubMed: 22626764]
- Meigs JB, D'Agostino RB Sr. Wilson PW, Cupples LA, Nathan DM, Singer DE. Risk variable clustering in the insulin resistance syndrome. The Framingham Offspring Study. Diabetes. Oct; 1997 46(10):1594–600. PubMed PMID: 9313755. [PubMed: 9313755]
- Peeters CF, Dziura J, van Wesel F. Pathophysiological domains underlying the metabolic syndrome: an alternative factor analytic strategy. Annals of epidemiology. Oct; 2014 24(10):762– 70. PubMed PMID: 25238942. [PubMed: 25238942]
- Heiss G, Snyder ML, Teng Y, Schneiderman N, Llabre MM, Cowie C, et al. Prevalence of metabolic syndrome among Hispanics/Latinos of diverse background: the Hispanic Community Health Study/Study of Latinos. Diabetes care. Aug; 2014 37(8):2391–9. PubMed PMID: 25061141. Pubmed Central PMCID: 4113166. [PubMed: 25061141]
- Hanley AJ, Williams K, Stern MP, Haffner SM. Homeostasis model assessment of insulin resistance in relation to the incidence of cardiovascular disease: the San Antonio Heart Study. Diabetes care. Jul; 2002 25(7):1177–84. PubMed PMID: 12087016. Epub 2002/06/28. eng. [PubMed: 12087016]
- 22. Rodriguez C, Pablos-Mendez A, Palmas W, Lantigua R, Mayeux R, Berglund L. Comparison of modifiable determinants of lipids and lipoprotein levels among African-Americans, Hispanics, and Non-Hispanic Caucasians > or =65 years of age living in New York City. The American journal of cardiology. Jan 15; 2002 89(2):178–83. PubMed PMID: 11792339. Epub 2002/01/17. eng. [PubMed: 11792339]
- 23. Nicholls SJ, Puri R. Is it time for HDL to change its tune? Circulation. Sep 10; 2013 128(11): 1175–6. PubMed PMID: 24002796. [PubMed: 24002796]
- Mora S, Glynn RJ, Ridker PM. High-density lipoprotein cholesterol, size, particle number, and residual vascular risk after potent statin therapy. Circulation. Sep 10; 2013 128(11):1189–97. PubMed PMID: 24002795. Pubmed Central PMCID: 3807967. [PubMed: 24002795]
- Akinkuolie AO, Paynter NP, Padmanabhan L, Mora S. High-density lipoprotein particle subclass heterogeneity and incident coronary heart disease. Circulation Cardiovascular quality and outcomes. Jan; 2014 7(1):55–63. PubMed PMID: 24248942. [PubMed: 24248942]
- 26. Khera AV, Cuchel M, de la Llera-Moya M, Rodrigues A, Burke MF, Jafri K, et al. Cholesterol efflux capacity, high-density lipoprotein function, and atherosclerosis. The New England journal of medicine. Jan 13; 2011 364(2):127–35. PubMed PMID: 21226578. Pubmed Central PMCID: 3030449. Epub 2011/01/14. eng. [PubMed: 21226578]
- 27. Rader DJ, Tall AR. The not-so-simple HDL story: Is it time to revise the HDL cholesterol hypothesis? Nature medicine. Sep; 2012 18(9):1344–6. PubMed PMID: 22961164. Epub 2012/09/11. eng.
- Daviglus ML, Talavera GA, Aviles-Santa ML, Allison M, Cai J, Criqui MH, et al. Prevalence of major cardiovascular risk factors and cardiovascular diseases among Hispanic/Latino individuals of diverse backgrounds in the United States. JAMA : the journal of the American Medical Association. Nov 7; 2012 308(17):1775–84. PubMed PMID: 23117778. Pubmed Central PMCID: 3777250.

Clinical Implications

Our results indicate that similar criteria for MetS may be applied across Hispanic/Latino ancestry groups; however the role of HDL-C in the classification is called into question by our data. It may be prudent to revisit current guidelines. Also, with respect to blood pressure, the gender difference needs further investigation.

Table 1

Means and (standard deviations) for continuous measures and % for categorical variables by gender and total sample

Measures	Total sa (N = 158	mple 823)	Men (n = 631	.6)	Women (n =950	7)
Age (years)	41.34	(14.99)	40.55	(14.82)	42.07	(15.11)
Education						
<high school<="" td=""><td>33.02</td><td></td><td>32.47</td><td></td><td>33.53</td><td></td></high>	33.02		32.47		33.53	
High school graduate	28.62		30.67		26.73	
Some college	22.26		20.66		23.73	
College degree	16.10		16.20		16.01	
Annual family income (\$)						
<20,000	42.44		38.49		46.06	
20,000-50,000	36.77		39.63		34.15	
>50,000	11.44		14.50		8.63	
Not reported	9.36		7.38		11.17	
Waist circumference (cm)	97.37	(14.50)	98.27	(14.14)	96.55	(14.77)
SBP (mmHg)	120.00	(17.27)	123.50	(15.06)	116.80	(18.50)
DBP (mmHg)	72.22	(10.89)	73.62	(10.93)	70.94	(10.70)
Triglycerides (mg/dL)	133.90	(118.87)	149.10	(150.69)	120.00	(76.87)
HDL-C (mg/dL)	48.42	(12.76)	44.75	(11.45)	51.77	(12.88)
Fasting glucose (mg/dL)	102.10	(33.35)	104.70	(34.45)	99.68	(32.11)
Cigarette use						
Never	61.46		51.12		70.93	
Previous smoker	17.37		22.14		13.00	
Current smoker	21.17		26.75		16.07	
Medication use						
Antihypertensive	.131		12.2		13.8	
Lipid lowering	.093		8.6		10.0	
Glucose lowering	.084		7.9		8.9	
CHD	5.95		6.64		5.32	
Diabetes	15.72		15.04		16.35	

Table 2

Correlation matrix for women and men

Variable	Waist	SBP	DBP	Triglycerides	HDL-C	Glucose
Waist	1	.164	.287	.292	245	.253
SBP	.184	1	.709	.279	.042	.244
DBP	.344	.704	1	.233	041	.157
Triglycerides	.307	.160	.287	1	403	.287
HDL-C	255	.032	071	462	1	124
Glucose	.229	.158	.127	.257	078	1

Note: Correlations for women are above the diagonal and for men are below the diagonal. SBP = systolic blood pressure. DBP = diastolic blood pressure. HDL-C= high density lipoprotein cholesterol.

Table 3

Factor loadings for single latent variable model of metabolic syndrome in women and men

	Unstandardized (9	5%CI)	Standardized (95%CI)		
	Women	Men	Women	Men	
Variable					
Waist	1	1	.46 (.42–.50)	.53 (.47–.59)	
SBP	1.32 (1.15–1.48)	0.68 (.57–.78)*	.48 (.45–.51)	.34 (.29–.39)	
DBP	0.55 (.4762)	0.65 (.5774)	.34 (.30–.38)	.45 (.40–.50)	
Triglycerides	0.20 (.1823)	0.21 (.17–.24)	.60 (.56–.64)	.60 (.55–.65)	
HDL-C	-0.18 (2709)	-0.20 (3010)	09 (1504)	13 (1907)	
Glucose	0.066 (.06–.07)	0.052 (.04–.06)	.48 (.44–.52)	.41 (.37–.45)	

Note: Waist circumference was used to set the metric for the latent variable with a loading of 1. SBP = systolic blood pressure. DBP = diastolic blood pressure. HDL-C= high density lipoprotein cholesterol.

 $\hat{} = p < .05$ for sex difference.