



Published in final edited form as:

Nutr Metab Cardiovasc Dis. 2012 February ; 22(2): 141–148. doi:10.1016/j.numecd.2010.05.006.

Racial/ethnic discrepancies in the metabolic syndrome begin in childhood and persist after adjustment for environmental factors

S.E. Walker, M.J. Gurka, M.N. Oliver, D.W. Johns, and M.D. DeBoer

University of Virginia School of Medicine, Charlottesville, VA; Department of Pediatrics (S.E.W, M.J.G., M.D.D.), Department of Public Health Sciences (M.J.G.), Department of Medicine (D.W.G); Department of Family Medicine (M.N.O.); and Center for Health Disparities (M.N.O.).

Abstract

Background—Evaluation of metabolic syndrome (MetS) characteristics across an age spectrum from childhood to adulthood has been limited by a lack of consistent MetS criteria for children and adults and by a lack of adjustment for environmental factors. We used the pediatric and adult International Diabetes Federation (IDF) criteria to determine whether gender- and race-specific differences in MetS and its components are present in adolescents as in adults after adjustment for socioeconomic status (SES) and lifestyle factors.

Methods—Waist circumference, blood pressure, triglycerides, HDL cholesterol, and fasting glucose measures were obtained from 3,100 adolescent (12-19y) and 3,419 adult (20-69y) non-Hispanic white, non-Hispanic black, and Mexican-American participants of the 1999-2006 National Health and Nutrition Examination Surveys. We compared odds of having MetS and its components across racial/ethnic groups by age group, while adjusting for income, education, physical activity and diet quality.

Results—After adjusting for possible confounding influences of SES and lifestyle, non-Hispanic-black adolescent males exhibited a lower odds of MetS and multiple components (abdominal obesity, hypertriglyceridemia, low HDL, hyperglycemia) compared to non-Hispanic-white and Mexican-American adolescents. Compared to non-Hispanic white adolescent males, Mexican-American adolescent males had less hypertension. There were no differences in MetS prevalence among adolescent females, though non-Hispanic-black girls exhibited less hypertriglyceridemia.

Conclusion—Racial/ethnicity-specific differences in MetS and its components are present in both adolescence and adulthood, even after adjusting for environmental factors. These data help strengthen arguments for developing racial/ethnic-specific MetS criteria to better identify individuals at risk for future cardiovascular disease.

Keywords

metabolic syndrome; adolescence; insulin; race

© 2010 Elsevier B.V. All rights reserved.

Correspondence to Mark D. DeBoer, MD, MSc., MCR; Department of Pediatrics, University of Virginia, P.O. Box 800386, Charlottesville, VA 22908. Office Phone: 434-924-9144 Fax: 434-924-9181 deboer@virginia.edu.

Publisher's Disclaimer: This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final citable form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

Disclosures There are no potential conflicts of interest from any of the authors of this manuscript.

The epidemic of childhood obesity has placed an increasing number of children at risk for developing the metabolic syndrome (MetS)[1]. This clustering of specific components—including abdominal obesity, hypertriglyceridemia, low HDL cholesterol, hypertension and hyperglycemia—appears to be linked by a poorly-understood underlying process that increases risk for type 2 diabetes mellitus (T2DM) and coronary artery disease (CAD)[2,3]. Recently there have been reports of increases in MetS among children[4] and adults[5], and children with MetS are >9 times more likely to exhibit MetS in adulthood[6]. As a result, some have suggested that a diagnosis of MetS could be used to trigger increased targeting of weight loss and other fitness efforts among children with MetS[7].

One feature confounding the importance of a diagnosis of MetS is the presence of discrepancies in the prevalence of individual components among different racial/ethnic groups. For instance, non-Hispanic-black men have lower rates of MetS diagnosis[5], in part, due to lower prevalence of hypertriglyceridemia[8-10] and low HDL[11], despite having higher rates of hypertension[5,11-13]. Nevertheless, non-Hispanic-black men have higher rates of CAD than would be expected given their low prevalence of MetS, suggesting that their lower rates of dyslipidemia are falsely reassuring. Therefore, due to unknown reasons the process underlying MetS may manifest itself differently among racial/ethnic subgroups[9,13,14].

Among children, racial/ethnic differences in obesity have been noted, with increased rates among non-Hispanic-black and Mexican-American children[15-17]. However, gender- and race/ethnicity-specific analyses of individual MetS components in children have been limited in the past by either a lack of pediatric MetS criteria[17] or a lack of adjustment for environmental factors[4].

We hypothesized that racial/ethnic differences in pediatric MetS components would remain significant after adjustment for multiple environmental factors such as socio-economic status (SES), education, nutrition and physical activity. We further hypothesized that these racial/ethnic discrepancies would be seen in a similar degree in both children and adults, suggesting a lifelong nature of the differences in MetS between racial/ethnic groups. To perform this analysis we elected to use the definition for pediatric and adult MetS used by the International Diabetes Federation (IDF)[7,18]. These are the only sets of MetS criteria that have been designed by a single agency to be used in both children (i.e. the IDF pediatric MetS criteria, all individuals<18y) and adults (the IDF adult MetS criteria, all individuals≥18y). We applied these criteria to children and adults from the most recently-available data from the National Health and Nutrition Examination Survey (NHANES), a cross-sectional survey that is representative of the US population and that contains information on socio-economic and lifestyle factors of potential importance in considering MetS. This report comprises the first unified analysis regarding racial-ethnic differences in MetS across the age spectrum from childhood to maturity.

Methods

Data were obtained from NHANES (1999-2006), a complex, multistage probability sample of the US population. These annual surveys are conducted by the National Center for Health Statistics (NCHS) of the Centers for Disease Control (CDC) with data released every 2y. The NCHS ethics review board reviewed and approved the survey and participants were provided with informed consent prior to participation. Participants who were ≥16y self-reported race/ethnicity. A family member reported the race/ethnicity of children younger than 16y. Racial/ethnicity groups for study analysis were categorized as non-Hispanic-black, non-Hispanic-white, and Mexican-American. Survey participants were interviewed in their homes to determine current medical condition followed by physical/laboratory examination in a mobile examination facility.

Waist circumference (WC), blood pressure, and lab measures of triglycerides, HDL cholesterol, and glucose were obtained using standardized protocols and calibrated equipments. All lab values used for analyses were obtained from participants asked to fast for 8hrs prior to the blood draw. The criteria for MetS in children 10-16y as specified by the **pediatric IDF criteria**[7] require the presence of abdominal obesity ($WC \geq 90^{\text{th}}$ percentile[19] or, if lower, adult cut-off) and ≥ 2 of the following components: hypertriglyceridemia ($\geq 1.7 \text{ mmol/l}$ [150mg/dl]); low HDL ($< 1.03 \text{ mmol/l}$ [40mg/dl]); hypertension ($\geq 130 \text{ mmHg}$ systolic or $\geq 85 \text{ mmHg}$ diastolic); elevated fasting blood glucose level ($\geq 5.6 \text{ mmol/l}$ [100mg/dl] or known T2DM). As prescribed in the pediatric IDF criteria, the adult IDF criteria[18] were applied to participants ≥ 16 y of age. The **adult IDF criteria** are similar to the pediatric definition with the following differences: $WC \geq 94 \text{ cm}$ in Europoid males ($\geq 80 \text{ cm}$ Europoid females); low HDL $< 1.29 \text{ mmol/l}$ (50mg/dl) for females and $< 1.03 \text{ mmol/l}$ (40mg/dl) for males; and history of treatment of hypertriglyceridemia, low HDL, diabetes, or hypertension can be used as an alternative to lab values. We included only children ≥ 12 y in our data analyses given that fasting values for triglycerides and glucose were only obtained in participants ≥ 12 y. Racial/ethnic-specific cut-offs were used for waist circumference measurements in adolescents < 16 y[19].

In making race/ethnicity comparisons, we adjusted for several environmental factors with potential effects on MetS components: education level, income-to-poverty-level ratio, diet quality, and physical inactivity. Education level of the adult individual, or the highest level among the homeowner and his/her spouse in the case of children, was classified into three categories: less than high school (< 8 y of school) high school (8-12y of school), and more than high school (> 12 y of school). The US Census Bureau, as a gauge of poverty, uses a ratio of household income-to-poverty level, with lower ratios indicating a more impoverished state. Three categories of income-to-poverty-level ratio were examined: < 1.0 , $1.0-3.0$, > 3.0 . As a means of identifying physically-inactive individuals, physical activities and their associated metabolic equivalent (MET) scores were obtained for each participant. The survey rated > 60 different physical activities including organized sports, aerobic dance, running, jogging, and gardening and their respective intensities defined by MET score. "Physical inactivity" was defined as a total MET score ≤ 3.5 [5].

Diet quality of the individual was measured by the Healthy Eating Index (HEI-2005)[20] using data collected from computer-assisted 24-hour food recall questionnaires (the Automated Multiple-Pass Method) developed by NHANES[21] and USDA[22]. The HEI-2005 was developed to measure compliance with the recommendations of the 2005 Dietary Guidelines for Americans[20]. Scores are assigned to 12 components of diet including intake of grains, vegetables, fruits, milk, and meat/meat alternatives and are assigned a weight; a total score is then calculated from the sum of these weighted scores. The HEI-2005 total score ranges from 0-100, with higher scores representing better diet quality. For the purposes of this analysis, HEI-2005 total scores were categorized into tertiles based on the final sample ("low" score: 0-41.4; "moderate" score: 41.4-53.7; "high" score: 53.7-100). As performed in previous analyses of MetS[10], we excluded self-reported diabetics, pregnant women and individuals taking estrogen-containing medications, anti-hypertensives, diabetes medications, and lipid lowering medications.

Statistical Methods

Preliminary data analyses comparing individual NHANES surveys from 1999-2006 did not demonstrate significant differences or trends in the prevalence of MetS or its components in adolescents or adults. We combined all data sets for statistical analyses, increasing sample size and power. Prevalence of MetS and its components was calculated by age, gender, and race/ethnicity (non-Hispanic-white, non-Hispanic-black, and Mexican-American). Age was

divided into 3 groups: adolescent (12-19y), early-adulthood (20-34y), and mid-adulthood (35-64y), as used previously[5]. Standard errors and confidence intervals were also computed. Multiple logistic regression was applied to the data stratified by age and gender to estimate odds ratios (OR's) of MetS and its components by race/ethnicity, education level, income:poverty-level ratio, carbohydrate intake, and physical inactivity. Statistical significance was defined as a $p < 0.05$. Statistical analysis was performed using SUDAAN (version 10; Research Triangle Institute, North Carolina). SUDAAN was used to apply sampling weights in order to produce national estimates.

Results

Sample Characteristics

The sample of participants who met inclusion criteria consisted of 6,519 non-Hispanic black, Mexican Americans, and non-Hispanic whites age 12-64y with data for all variables tested. Of these, 47.6% were adolescents (ages 12-19y) and 52.4% were adults (20-64y) with the following racial composition: 27.6% non-Hispanic black, 33.0% Mexican American, and 39.3% non-Hispanic white (Table 1). Whites in each age range were more likely than the other two groups to have more than a high school (HS) education and an income:poverty ratio > 3.0 , while Mexican Americans in each age range were more likely to have less than a HS education ($p < 0.05$, data not shown), as previously noted[23].

MetS Prevalence

Among US adolescent males the prevalence of MetS was 8.4%, 9.4%, and 2.5% for non-Hispanic whites, Mexican Americans, and non-Hispanic blacks respectively (Figure 1, Table 1). The prevalence of MetS among adolescent females was 4.4%, 6.4%, and 4.2% among non-Hispanic whites, Mexican Americans, and non-Hispanic blacks respectively. This prevalence increased significantly with age among adults in all racial/ethnic groups (Table 1 and Supplementary Figure). Race comparisons in the form of unadjusted odds ratios are located on the first three rows of Table 2. During adolescence, non-Hispanic-black males had a lower odds of MetS compared to both non-Hispanic white (OR=0.3; $p < 0.01$) and Mexican-American males (OR=0.2, $p < 0.01$). This lower prevalence of MetS in non-Hispanic blacks was also seen across age groups: 20-34y (OR=0.4; $p < 0.01$) and 35-64y (OR=0.6; $p < 0.05$). There was no significant difference by race/ethnicity in odds of MetS observed among adolescent females. In contrast to non-Hispanic-black males across the age-spectrum, non-Hispanic-black females did not have lower rates of MetS at any age. Mexican-American women age 35-64 had a higher odds of MetS compared to non-Hispanic-white women.

Logistic Regression Models for MetS and MetS Components

Multiple logistic regression models of MetS and its components for males and females in each of three age groups (12-19y, 20-34y, and 35-64y) are presented in Table 2 and Figure 2. Age along with race/ethnicity, education level, income-to-poverty-level ratio, food quality (HEI), and physical activity were included as covariates in the models (Table 2). Adjusting for these covariates did not confound the raw observed racial differences, with the exception of non-Hispanic black men age 35-64, who did not exhibit a lower odds of MetS after adjustment for the covariates.

Adjusted odds ratios for the components of MetS among the races are shown in Figure 1. After adjusting for socio-economic and lifestyle factors as in the MetS models, non-Hispanic-black adolescent males were less likely to have elevated WC (OR 0.6; $p < 0.05$), hypertriglyceridemia (OR=0.2; $p < 0.001$), low HDL (OR=0.3; $p < 0.001$), or hyperglycemia (OR=0.5; $p < 0.05$) compared to non-Hispanic whites (Figure 2). Non-Hispanic black

adolescent males were also less likely to have these outcomes—with the exception of hyperglycemia—compared to Mexican Americans ($p<0.05$). All of these observations except HDL were evident in at least one phase of adulthood. Mexican-American adolescent males were more likely to have abdominal obesity compared to non-Hispanic-black males ($p<0.05$) with a trend toward increased abdominal obesity compared to whites ($OR=1.4$, $p=0.08$). This trend widened in early-adulthood (OR vs. non-Hispanic whites, 3.9; $p<0.001$). Mexican-American adolescent males were less likely to have hypertension compared to whites ($OR=0.3$; $p<0.01$), and this difference was also seen in early adulthood.

Compared to non-Hispanic whites, non-Hispanic-black adolescent females were less likely to have hypertriglyceridemia (OR 0.3; $p<0.01$), a difference which did not extend into adulthood (Figure 2). There were no significant differences in odds of MetS components between non-Hispanic-black and Mexican-American adolescent females.

Discussion

Racial/ethnic differences in individual components of the MetS have been reported previously, raising the possibility of a genetic component of MetS among these populations, despite widespread genetic overlap among racial/ethnic groups in the United States[24]. Nevertheless, significant differences in SES and other lifestyle factors exist between the racial/ethnic groups evaluated in this study, and many of these environmental factors are known to affect components of MetS including effects of SES, diet and activity level on obesity[25-27], hypertension[28] and T2DM[15]. While studies of adults with MetS have adjusted for these factors[5], to date no study has evaluated whether socioeconomic and lifestyle factors may be responsible for racial/ethnic differences in the prevalence of MetS or its components among children. In addition, there was previously a lack of unique pediatric MetS criteria designed to be in continuum with adult criteria, preventing an analysis for differences in MetS across an age spectrum that included childhood.

In the present study we applied the pediatric and adult IDF MetS criteria to NHANES data (1999-2006) to define trends in MetS between adolescents and adults. These criteria differ from the ATP III definition of MetS predominantly in using race-specific elevations in WC as a prerequisite for MetS diagnosis[7,18], though they have been noted to detect similar racial/ethnic discrepancies as seen in other MetS criteria[29]. We utilized the IDF criteria for our analysis because in the absence of a consensus criteria for pediatric MetS, the IDF criteria are the only set of MetS criteria released by a single agency for use in children[7] or adults[18].

Thus, our evaluation is unique in 1) applying a set of MetS criteria with cut-off values designed to gradually change between children and adults and 2) adjusting for socioeconomic and lifestyle factors across the age spectrum. We found that MetS discrepancies among racial/ethnic groups are present in children as well as in adults and that these differences persist after adjustment for these multiple potential environmental confounders, including sizable differences in income and education between racial/ethnic groups. This argues that development of MetS or its components may be influenced by racial differences between groups and not solely environmental factors.

In particular, we found that non-Hispanic-black boys and young men are less likely than their non-Hispanic-white and Mexican-American counterparts to be diagnosed with MetS or to exhibit elevated WC, hypertriglyceridemia, low HDL, or hyperglycemia. These findings are overall similar to prior reports that did not adjust for lifestyle factors[4]. The lower prevalences of MetS and individual MetS components among non-Hispanic black adolescents could, at first glance, be construed as reassuring, suggesting that non-Hispanic

black young men are less likely to suffer later consequences of MetS, including T2DM and CAD. However, data from non-Hispanic black men reveal higher rates of CAD[14] suggesting a false sense of reassurance given the low rates of diagnosed MetS. Although long-term studies will be needed to demonstrate whether non-MetS processes may be responsible for these increases in CAD in non-Hispanic blacks, there is concern that MetS may be underdiagnosed in these individuals[13].

The prognostic importance of a diagnosis of MetS in childhood has been limited by the difficulty in gathering prospective data linking childhood disease state with adult disease. It is important to note that the data we present here are cross-sectional in nature and cannot, in isolation, confirm persistence of these traits with increasing age in a given individual or cohort. As is common in pediatric disease, the prolonged time necessary before children develop clinically-significant disease hinders the ability for prospective studies to answer these questions. Nevertheless, the overall importance of a diagnosis of MetS in childhood has been demonstrated by retrospective analysis of longitudinal data, including the Bogalusa Heart Study[30], the Princeton Lipids Research Clinic Study[6], and other such studies[31]. These studies reveal that a diagnosis of childhood MetS increases the likelihood of developing adult MetS (over 9-fold)[6] and T2DM (3-fold)[6] and of exhibiting increased carotid artery intimal thickness (26% increase in intimal thickness)[32]. In addition, individual features of childhood MetS also predict an increased risk for developing adult MetS and T2DM, including increased BMI[33,34], fasting glucose[33,34], triglycerides[34], low HDL[33,34], and systolic blood pressure[34]. Still, the combination of multiple MetS components in childhood provide a stronger predictor of future disease[6,33,34].

This ability of pediatric MetS to predict adult disease has led some researchers to propose using MetS diagnosis in childhood as a tool to trigger increased intervention[7]. The presence of modifiable cardiovascular risk factors as individual components of MetS makes this an attractive approach to prevent future CAD[7,17,33]. While fitness interventions and lifestyle adjustments remain challenging, the ability of a MetS diagnosis to predict future disease may prove to be an important motivating tool for patients and their families[8]. Given that non-Hispanic black boys and men are less likely to be diagnosed with MetS despite their increased lifetime risk of T2DM and CAD, it is likely that they would inappropriately miss out on opportunities for early intervention.

The appearance of racial/ethnic discrepancies in MetS components in both childhood and adulthood may help to strengthen arguments for racial/ethnic-specific MetS criteria that could better identify individuals at risk for future CAD[9,10,13]. Despite a high degree of racial admixture between groups[24], it is possible that specific population subgroups within each racial/ethnic category have a genetic predisposition toward the presence or absence of particular MetS components. This may account for the trends in both adolescence and adulthood as observed in this study. Future identification of CAD-associated polymorphisms that differ among racial/ethnic subgroups could lead to early identification of individuals, such as non-Hispanic black boys, who are at risk for CAD despite reassuring MetS indices[35]. Until investigators clearly define these genetic variations, race/ethnicity-specific modifications of MetS criteria may better identify children and adults at increased risk for future disease[8,10,11].

Further research may also pinpoint whether individual components of MetS are more sensitive predictors of future disease among children or whether non-MetS risk factors underlie the increased risk for CAD among non-Hispanic blacks[13]. Inherent in this need is research to determine racial/ethnic differences in the ability of MetS to predict risk among adolescents. Indeed, there is a growing need in our society for new tools of risk-identification, such that increased efforts could be targeted at intervention for at-risk youths

to reverse cardiovascular risk. These efforts would include increased fitness and dietary counseling, efforts which often require a significant amount of resources and are likely to be best directed at those children who would benefit the most[8].

As a concept, MetS is important in that it postulates that there is an underlying insidious process that links multiple metabolic derangements in an as-yet poorly described manner. Since this process appears to manifest differently among racial/ethnic groups, the development of racial/ethnic-specific MetS criteria or other risk-identification tools may lead to a deeper understanding of fundamental processes contributing to MetS and its sequelae. In addition, research efforts should be initiated to reveal why non-Hispanic blacks have a higher risk of CAD despite low rates of hypertriglyceridemia and low HDL. These efforts may also assist in the development of improved guidelines to better identify which children need additional fitness interventions in order to avoid progression toward adult complications of MetS.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

Funding Sources

NIH T32H207956 (to SEW) NIH HD060739-01 (to MDD)

References

1. Weiss R, Dziura J, Burgert TS, Tamborlane WV, Taksali SE, Yekkel CW, Allen K, Lopes M, Savoye M, Morrison J, Sherwin RS, Caprio S. Obesity and the metabolic syndrome in children and adolescents. *N Engl J Med*. 2004; 350:2362–2374. [PubMed: 15175438]
2. Reaven GM. Banting lecture 1988. Role of insulin resistance in human disease. *Diabetes*. 1988; 37:1595–1607. [PubMed: 3056758]
3. DeBoer MD, Gurka MJ. Ability among adolescents for the metabolic syndrome to predict elevations in factors associated with type 2 diabetes and cardiovascular disease: Data from the National Health and Nutrition Examination Survey (NHANES) 1999-2006. *Metabolic Syndrome and Related Disorders*. 2010 In Press.
4. Johnson WD, Kroon JJ, Greenway FL, Bouchard C, Ryan D, Katzmarzyk PT. Prevalence of risk factors for metabolic syndrome in adolescents: National Health and Nutrition Examination Survey (NHANES), 2001-2006. *Arch Pediatr Adolesc Med*. 2009; 163:371–377. [PubMed: 19349567]
5. Park YW, Zhu S, Palaniappan L, Heshka S, Carnethon MR, Heymsfield SB. The metabolic syndrome: prevalence and associated risk factor findings in the US population from the Third National Health and Nutrition Examination Survey, 1988-1994. *Arch Intern Med*. 2003; 163:427–436. [PubMed: 12588201]
6. Morrison JA, Friedman LA, Wang P, Glueck CJ. Metabolic syndrome in childhood predicts adult metabolic syndrome and type 2 diabetes mellitus 25 to 30 years later. *J Pediatr*. 2008; 152:201–206. [PubMed: 18206689]
7. Zimmet P, Alberti G, Kaufman F, Tajima N, Silink M, Arslanian S, Wong G, Bennett P, Shaw J, Caprio S. The metabolic syndrome in children and adolescents. *Lancet*. 2007; 369:2059–2061. [PubMed: 17586288]
8. DeBoer MD. Underdiagnosis of the Metabolic Syndrome in Non-Hispanic Black Adolescents: A Call for Ethnic-Specific Criteria. *Current Cardiovascular Risk Reports*. 2010 In press.
9. Giannini E, Testa R. The metabolic syndrome: all criteria are equal, but some criteria are more equal than others. *Arch Intern Med*. 2003; 163:2787–2788. [PubMed: 14662635]

10. Sumner AE, Cowie CC. Ethnic differences in the ability of triglyceride levels to identify insulin resistance. *Atherosclerosis*. 2008; 196:696–703. [PubMed: 17254586]
11. Anuрад E, Chiem A, Pearson TA, Berglund L. Metabolic syndrome components in African-Americans and European-American patients and its relation to coronary artery disease. *Am J Cardiol*. 2007; 100:830–834. [PubMed: 17719328]
12. Saad MF, Lillioja S, Nyomba BL, Castillo C, Ferraro R, De Gregorio M, Ravussin E, Knowler WC, Bennett PH, Howard BV, et al. Racial differences in the relation between blood pressure and insulin resistance. *N Engl J Med*. 1991; 324:733–739. [PubMed: 1997839]
13. Sumner AE. Ethnic Differences in Triglyceride Levels and High-Density Lipoprotein Lead to Underdiagnosis of the Metabolic Syndrome in Black Children and Adults. *Journal of Pediatrics*. 2009; 155:e7–e11. [PubMed: 19732569]
14. Yancy CW, Benjamin EJ, Fabunmi RP, Bonow RO. Discovering the full spectrum of cardiovascular disease: Minority Health Summit 2003: executive summary. *Circulation*. 2005; 111:1339–1349. [PubMed: 15769779]
15. Lieb DC, Snow RE, DeBoer MD. Socioeconomic factors in the development of childhood obesity and diabetes. *Clin Sports Med*. 2009; 28:349–378. [PubMed: 19505621]
16. Ogden CL, Carroll MD, Flegal KM. High body mass index for age among US children and adolescents, 2003–2006. *Jama*. 2008; 299:2401–2405. [PubMed: 18505949]
17. Winkleby MA, Robinson TN, Sundquist J, Kraemer HC. Ethnic variation in cardiovascular disease risk factors among children and young adults: findings from the Third National Health and Nutrition Examination Survey, 1988–1994. *JAMA*. 1999; 281:1006–1013. [PubMed: 10086435]
18. Alberti KG, Zimmet P, Shaw J. Metabolic syndrome--a new world-wide definition. A Consensus Statement from the International Diabetes Federation. *Diabet Med*. 2006; 23:469–480. [PubMed: 16681555]
19. Fernandez JR, Redden DT, Pietrobello A, Allison DB. Waist circumference percentiles in nationally representative samples of African-American, European-American, and Mexican-American children and adolescents. *J Pediatr*. 2004; 145:439–444. [PubMed: 15480363]
20. Guenther, PM.; Reedy, J.; Krebs-Smith, SM.; Reeve, BB.; Basiotis, PP. Development and Evaluation of the Healthy Eating Index-2005: Technical Report. <http://www.cnpp.usda.gov/HealthyEatingIndex.htm>; accessed May 13, 2010
21. CDC. Dietary Interview Component, NHANES. http://www.cdc.gov/nchs/data/nhanes/nhanes_07_08/dietaryrecall_e.pdf; accessed May 13, 2010
22. USDA. Features of AMPM. <http://www.ars.usda.gov/Services/docs.htm?docid=7711>; accessed May 13, 2010
23. Merkin SS, Karlamangla A, Crimmins E, Charette SL, Hayward M, Kim JK, Koretz B, Seeman T. Education differentials by race and ethnicity in the diagnosis and management of hypercholesterolemia: a national sample of U.S. adults (NHANES 1999–2002). *Int J Public Health*. 2009; 54:166–174. [PubMed: 19219403]
24. Hernandez, L.; Blazer, D. *Genes, Behavior and the Social Environment: Moving Beyond the Nature/Nurture Debate*. Washington, DC: National Academies Press: 2006.
25. Ambrosini GL, Huang RC, Mori TA, Hands BP, O'Sullivan TA, de Klerk NH, Beilin LJ, Oddy WH. Dietary patterns and markers for the metabolic syndrome in Australian adolescents. *Nutr Metab Cardiovasc Dis*. 20:274–283. [PubMed: 19748245]
26. Delva J, Johnston LD, O'Malley PM. The epidemiology of overweight and related lifestyle behaviors: racial/ethnic and socioeconomic status differences among American youth. *Am J Prev Med*. 2007; 33:S178–186. [PubMed: 17884566]
27. Babio N, Bullo M, Basora J, Martinez-Gonzalez MA, Fernandez-Ballart J, Marquez-Sandoval F, Molina C, Salas-Salvado J. Adherence to the Mediterranean diet and risk of metabolic syndrome and its components. *Nutr Metab Cardiovasc Dis*. 2009; 19:563–570. [PubMed: 19176282]
28. Hernelahti M, Levalahti E, Simonen RL, Kaprio J, Kujala UM, Uusitalo-Koskinen AL, Battie MC, Videman T. Relative roles of heredity and physical activity in adolescence and adulthood on blood pressure. *J Appl Physiol*. 2004; 97:1046–1052. [PubMed: 15145916]

29. Ford ES, Li C, Zhao G, Pearson WS, Mokdad AH. Prevalence of the metabolic syndrome among U.S. adolescents using the definition from the International Diabetes Federation. *Diabetes Care*. 2008; 31:587–589. [PubMed: 18071007]
30. Tzou WS, Douglas PS, Srinivasan SR, Bond MG, Tang R, Chen W, Berenson GS, Stein JH. Increased subclinical atherosclerosis in young adults with metabolic syndrome: the Bogalusa Heart Study. *J Am Coll Cardiol*. 2005; 46:457–463. [PubMed: 16053958]
31. Schubert CM, Sun SS, Burns TL, Morrison JA, Huang TT. Predictive ability of childhood metabolic components for adult metabolic syndrome and type 2 diabetes. *J Pediatr*. 2009; 155:S6 e1–7.
32. Iannuzzi A, Licenziati MR, Acampora C, De Michele M, Iannuzzo G, Chiariello G, Covetti G, Bresciani A, Romano L, Panico S, Rubba P. Carotid artery wall hypertrophy in children with metabolic syndrome. *J Hum Hypertens*. 2008; 22:83–88. [PubMed: 17928879]
33. Franks PW, Hanson RL, Knowler WC, Moffett C, Enos G, Infante AM, Krakoff J, Looker HC. Childhood predictors of young-onset type 2 diabetes. *Diabetes*. 2007; 56:2964–2972. [PubMed: 17720898]
34. Huang TT, Nansel TR, Belsheim AR, Morrison JA. Sensitivity, specificity, and predictive values of pediatric metabolic syndrome components in relation to adult metabolic syndrome: the Princeton LRC follow-up study. *J Pediatr*. 2008; 152:185–190. [PubMed: 18206687]
35. Assimes TL, Knowles JW, Basu A, Iribarren C, Southwick A, Tang H, Absher D, Li J, Fair JM, Rubin GD, Sidney S, Fortmann SP, Go AS, Hlatky MA, Myers RM, Risch N, Quertermous T. Susceptibility locus for clinical and subclinical coronary artery disease at chromosome 9p21 in the multi-ethnic ADVANCE study. *Hum Mol Genet*. 2008; 17:2320–2328. [PubMed: 18443000]

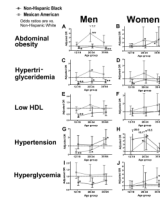


FIGURE 1.

Adjusted Odds Ratios for Metabolic Syndrome Components by Age, Race, and Gender.

OR's for non-Hispanic blacks and Mexican Americans are shown using non-Hispanic whites as the comparator group after adjustment for education level, income:poverty ratio, physical inactivity and dietary quality (Healthy Eating Index). Data are from NHANES '99-'06 for all individuals with complete information regarding IDF MetS components.

* $p < 0.05$ vs. white non-Hispanic; ** $p < 0.01$ vs. white non-Hispanics

Table 1

Prevalences of IDF Metabolic Syndrome and its Components by Age, Gender, and Race.

	n	Percent (Standard Error)					
		Metabolic Syndrome	Abdominal Obesity	High Triglyceride Level	Low HDL	High Blood Pressure	Hyperglycemia
<i>Males</i>							
12-19 years old							
Non-Hispanic White	470	8.4 (1.5)	22.0 (2.0)	13.5 (2.1)	24.2 (2.0)	7.4 (1.4)	16.9 (2.0)
Mexican American	601	9.4 (1.4)	29.6 (1.6)*	12.8 (1.4)	20.8 (1.8)	3.7 (0.7)*	21.5 (2.0)
Non-Hispanic Black	587	2.5 (0.7)**	15.8 (1.5)**	3.5 (0.7)**	9.8 (1.5)**	9.9 (1.3)#	10.3 (1.4)**
20-34 years old							
Non-Hispanic White	356	19.3 (2.0)	43.9 (2.7)	28.1 (2.8)	28.4 (2.6)	21.2 (2.1)	19.1 (2.2)
Mexican American	261	21.3 (2.9)	61.9 (4.0)*	31.7 (3.3)	27.8 (2.4)	10.6 (2.2)*	28.1 (2.5)*
Non-Hispanic Black	184	7.9 (2.0)**	35.9 (3.5)#	13.9 (2.6)**	16.4 (3.1)**	25.3 (3.7)#	9.7 (2.6)**
35-64 years old							
Non-Hispanic White	612	36.6 (2.5)	69.1 (2.3)	37.4 (2.5)	26.9 (2.3)	31.5 (2.0)	39.3 (2.2)
Mexican American	279	44.9 (3.9)	77.0 (2.3)*	48.9 (3.3)*	28.5 (3.0)	32.8 (3.3)	48.2 (4.4)*
Non-Hispanic Black	220	23.4 (2.9)**	50.0 (3.5)**	15.8 (2.6)**	15.2 (2.6)**	44.7 (3.6)**	30.7 (3.1)**
<i>Females</i>							
12-19 years old							
Non-Hispanic White	397	4.4 (1.2)	38.9 (2.7)	8.1 (1.8)	25.3 (2.6)	1.0 (0.6)	6.7 (1.4)
Mexican American	588	6.4 (0.8)	47.9 (2.3)*	10.4 (1.2)	25.8 (1.7)	0.7 (0.2)	7.1 (1.2)
Non-Hispanic Black	457	4.2 (1.1)	44.2 (2.6)	3.5 (1.1)**	21.6 (2.2)	3.5 (0.8)**	4.7 (1.1)
20-34 years old							
Non-Hispanic White	250	14.9 (2.4)	66.1 (3.2)	15.5 (2.7)	39.9 (3.4)	3.3 (1.3)	9.0 (1.8)
Mexican American	182	19.5 (3.5)	83.7 (2.7)*	21.6 (3.4)	48.8 (4.8)	3.1 (1.3)	12.8 (3.5)
Non-Hispanic Black	157	16.6 (3.2)	75.4 (3.3)*	9.9 (2.3)#	42.9 (4.3)	10.6 (2.3)**	8.3 (2.3)
35-64 years old							
Non-Hispanic White	479	22.9 (2.3)	72.9 (2.1)	21.9 (2.0)	33.1 (2.4)	21.6 (1.8)	17.5 (2.0)
Mexican American	243	36.6 (3.8)*	90.4 (1.8)*	27.0 (3.6)	47.7 (4.4)*	22.5 (2.9)	34.1 (3.3)*

	n	Percent (Standard Error)					
		Metabolic Syndrome	Abdominal Obesity	High Triglyceride Level	Low HDL	High Blood Pressure	Hyperglycemia
Non-Hispanic Black	196	30.7 (3.6)	86.8 (2.3)*	12.4 (2.6)*#	34.5 (4.0)	33.2 (3.7)*#	22.8 (3.0)#

* Significantly different (p-value < 0.05) than non-Hispanic White

Significantly different (p-value < 0.05) than Mexican-American

Table 2
Odds of Metabolic Syndrome by Gender and Age Group Race/Ethnicity Comparisons: Unadjusted and Adjusted Odds Ratios*

	Males				Females				
	12-19 Years	20-34 Years	35-64 Years	12-19 Years	20-34 Years	35-64 Years	12-19 Years	20-34 Years	35-64 Years
<i>Unadjusted Race/Ethnicity Comparisons</i>									
Non-Hispanic Black vs. Non-Hispanic White	0.29 (0.15, 0.55)	0.36 (0.20, 0.64)	0.63 (0.37, 0.76)	0.95 (0.40, 2.25)	1.13 (0.58, 2.21)	1.50 (0.99, 2.26)	0.95 (0.40, 2.25)	1.13 (0.58, 2.21)	1.50 (0.99, 2.26)
Mexican-American vs. Non-Hispanic White	1.14 (0.72, 1.80)	1.13 (0.77, 1.67)	1.41 (0.98, 2.05)	1.47 (0.74, 2.93)	1.38 (0.73, 2.63)	1.95 (1.25, 3.03)	1.47 (0.74, 2.93)	1.38 (0.73, 2.63)	1.95 (1.25, 3.03)
<i>Adjusted Race/Ethnicity Comparisons**</i>									
Non-Hispanic Black vs. Non-Hispanic White	0.22 (0.10, 0.49)	0.43 (0.20, 0.91)	0.67 (0.39, 1.15)	1.32 (0.39, 4.44)	1.72 (0.74, 4.00)	1.80 (0.98, 3.28)	1.32 (0.39, 4.44)	1.72 (0.74, 4.00)	1.80 (0.98, 3.28)
Mexican-American vs. Non-Hispanic White	1.01 (0.57, 1.78)	1.80 (0.97, 3.33)	1.26 (0.72, 2.22)	1.42 (0.45, 4.45)	1.87 (0.82, 4.26)	2.03 (1.10, 3.76)	1.42 (0.45, 4.45)	1.87 (0.82, 4.26)	2.03 (1.10, 3.76)
<i>Logistic Model Covariates</i>									
Education Level [†]									
Less than HS vs. More than HS	1.07 (0.41, 2.80)	0.41 (0.17, 0.98)	0.99 (0.41, 2.38)	0.61 (0.23, 1.60)	0.90 (0.34, 2.43)	1.45 (0.64, 3.27)	0.61 (0.23, 1.60)	0.90 (0.34, 2.43)	1.45 (0.64, 3.27)
HS vs. More than HS	1.11 (0.53, 2.32)	0.80 (0.38, 1.71)	0.94 (0.58, 1.55)	0.48 (0.19, 1.20)	1.17 (0.44, 3.11)	1.95 (0.94, 4.01)	0.48 (0.19, 1.20)	1.17 (0.44, 3.11)	1.95 (0.94, 4.01)
Income: Poverty Ratio									
< 1.0 vs. > 3.0	0.98 (0.37, 2.59)	0.24 (0.07, 0.75)	0.98 (0.36, 2.62)	1.35 (0.42, 4.30)	0.62 (0.19, 2.09)	1.87 (0.75, 4.66)	1.35 (0.42, 4.30)	0.62 (0.19, 2.09)	1.87 (0.75, 4.66)
1.0-3.0 vs. > 3.0	1.22 (0.54, 2.80)	1.00 (0.54, 1.87)	1.44 (0.92, 2.27)	2.26 (0.56, 9.08)	0.95 (0.48, 1.92)	0.98 (0.46, 2.07)	2.26 (0.56, 9.08)	0.95 (0.48, 1.92)	0.98 (0.46, 2.07)
Healthy Eating Index (2005)									
Low vs. High	1.28 (0.64, 2.58)	1.68 (0.82, 3.46)	1.39 (0.87, 2.22)	0.85 (0.30, 2.47)	1.17 (0.50, 2.70)	0.64 (0.32, 1.29)	0.85 (0.30, 2.47)	1.17 (0.50, 2.70)	0.64 (0.32, 1.29)
Moderate vs. High	1.20 (0.60, 2.39)	1.09 (0.50, 2.41)	1.03 (0.59, 1.79)	1.22 (0.42, 3.58)	1.05 (0.42, 2.65)	0.92 (0.54, 1.56)	1.22 (0.42, 3.58)	1.05 (0.42, 2.65)	0.92 (0.54, 1.56)
Physically inactive vs. Active	0.77 (0.09, 6.81)	2.38 (1.07, 5.28)	1.44 (0.90, 2.30)	0.55 (0.12, 2.45)	2.69 (1.23, 5.87)	2.52 (1.30, 4.89)	0.55 (0.12, 2.45)	2.69 (1.23, 5.87)	2.52 (1.30, 4.89)

* Statistical significance ($p < 0.05$) indicated by odds ratios in bold / 95% CI not containing 1

** Race comparisons adjusted for model covariates listed below via logistic regression (education, income, healthy eating index category, activity level category)

† Highest among household (person who owns/rents house or his/her spouse)

NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript