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Socio-economic and Ethnic Disparities in Cardiovascular Risk In the United States, 2001-2006

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Abstract

Purpose—To quantify socioeconomic status and ethnic differences in risk for coronary heart disease (CHD) accrued from major risk factors, in the United States (US).

Methods—Data came from the National Health and Nutrition Examination Survey 2001-2006. Outcomes examined were a) 10-year risk for CHD events as predicted by the National Cholesterol Education Program Adult Treatment Panel III 2004 Updated guidelines, and b) the prevalence of the metabolic syndrome and overt diabetes mellitus (a CHD risk-equivalent).

Results—Strong inverse socioeconomic gradients with risk were present in all race/ethnicity groups except foreign-born Mexican American men, and were attenuated by controls for physical activity, smoking, and abdominal obesity. In contrast, race/ethnicity disparities were seen in some but not all socioeconomic strata, with some Non-Hispanic Blacks and US-born Mexican Americans having higher risk and some Foreign-born Mexican Americans having *lower* risk.

Conclusions—Disparities in cardiovascular risk in the United States are primarily related to SES, and less to race/ethnicity. Socioeconomically disadvantaged individuals should be targeted for lifestyle counseling and early screening for risk factors, regardless of race/ethnicity, to reduce social disparities in cardiovascular outcomes.

Keywords

Socioeconomic status; ethnic differences; Framingham risk score; metabolic syndrome

INTRODUCTION

The incidence of cardiovascular disease in the United States (US) and other high-income countries is higher in low socio-economic status (SES) and minority ethnic groups (1,2,3,4, 5), with SES disparities reflecting not just a threshold effect of poverty versus non-poverty, but rather a risk gradient across the SES range (2,6). Reduction of these disparities has been a focus of interest for policy makers (7,8), and requires the early detection and management of

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risk factors in underprivileged groups. Indeed, low SES and minority ethnic groups do report higher values of risk factors for cardiovascular disease (1,9,10).

Some studies, however, suggest that SES gradients in cardiovascular risk factors may be diminished in minority groups (11,12,13,14), possibly because of racial discrimination and residential segregation (15,16,17), and greater psychological distress (18) at all SES levels. If SES associations with risk indeed do vary by race/ethnicity, then in any study of ethnic disparities, SES effects cannot be completely controlled simply by including SES indicators as covariates in regression (19,20). It is therefore, not clear if ethnic differences in cardiovascular risk primarily reflect SES differences, or if there are SES-independent race/ethnicity associations with risk; the two scenarios have different implications for health policy.

Moreover, few studies have examined SES / ethnic differences in global or overall cardiovascular risk accrued from all major risk factors. When SES /ethnic disparities in individual risk factors are of varying size and/or are in opposite directions (5,21), their combined effect can only be assessed by the use of global risk measures. Current clinical guidelines assess global risk for coronary heart disease (CHD) events using the Framingham risk score, the metabolic syndrome, and history of CHD and CHD risk-equivalent conditions (such as diabetes mellitus that confer equivalent risk) (22,23,24). Accordingly, in this work, we studied ethnic and SES disparities in the US population in a) the 10-year risk for CHD events, as assessed by the National Cholesterol Education Program (NCEP) Adult Treatment Panel III adaptation of the Framingham risk score (22,25), and b) the prevalence of diabetes mellitus and metabolic syndrome, as defined by the NCEP (22) and the European Group for the Study of Insulin Resistance (EGIR) (26). In particular, our objectives were to answer the following questions:

Q1. Are there SES gradients in global CHD risk and metabolic syndrome /diabetes prevalence, and do the SES gradients differ by race/ethnicity (i.e., do minority groups not get the same benefit from climbing the SES ladder as the majority ethnic group)?

Q2. Are there race/ethnicity differences in global CHD risk and metabolic syndrome/diabetes prevalence after adequately controlling for differences in SES?

Q3. Are SES and race/ethnicity associations with global CHD risk and metabolic syndrome/diabetes prevalence explained by differences in health behaviors and abdominal obesity (a surrogate for both diet and exercise)?

METHODS

Data for this study came from the National Health and Nutrition Examination Survey (NHANES), conducted between 2001 and 2006, of a nationally representative sample of the US population (27).

Study Sample

Of the 31,509 participants in NHANES 2001-2006, we excluded those under the age of 20 (n=16,078), pregnant women (n=886), individuals who missed the clinical examination (n=843), and those missing SES data (n=24). For the CHD risk analyses, we additionally excluded 1,524 individuals whose global CHD risk could not be classified; the resulting sample size was 12,154 (83.6% of those age-eligible and not pregnant). For the metabolic risk analyses, we excluded 7,970 individuals whose metabolic syndrome / diabetes data was missing (primarily because they had not fasted 6 hours or more before the blood draw); the final sample size for metabolic risk analysis was 5,648. Comparison of the analytic samples with the rest of the non-pregnant NHANES sample 20 years or older showed that they were representative of the complete NHANES sample (Table 1).

Measurements

Demographic, biological, and health data were collected at a home interview, followed by an examination and blood draw, either at home or a mobile examination center. Age, gender, race/ethnicity, education, and income data were obtained from standard adult questionnaires.

Educational attainment was classified as less than high school, complete high school, or more than high school education. Poverty Income Ratio (PIR), the ratio of household income to the poverty level specific to area of residence and household size (28), was recorded. Since educational attainment does not provide the same returns with respect to income in all race/ethnicity groups (15), we combined income and education to create three SES categories: We defined *low SES* as education less than high school *and* PIR <2, and *high SES* as education more than high school *and* PIR≥2. Everyone else was classified as *middle SES*, which included those with a) just high school education regardless of PIR, b) less than high school education but PIR≥2, and c) more than high school education but PIR<2.

Race/ethnicity was classified as Non-Hispanic White, Non-Hispanic Black, US-born Mexican American, Foreign-born Mexican American, and other. We distinguished between US-born and Foreign-born Mexican Americans because health risks in the 2 groups appear to be quite different, even at comparable SES (29,30,31).

Self reported medical history, physical activity (moderate / vigorous activity vs. none), current smoking, and medication data were obtained from the NHANES adult questionnaire. Prescription medication information was verified from medication bottle review. Blood pressure and waist circumference were measured at the examination. Blood samples were assayed for serum levels of C-reactive protein (CRP), total cholesterol, HDL cholesterol, *fasting* low density lipoprotein (LDL) cholesterol, *fasting* glucose, and *fasting* triglycerides. The last three assays were obtained only for those participants who had fasted at least 6 hours.

Risk Assessment

The two primary outcomes were global CHD risk and metabolic syndrome /diabetes prevalence. We created six CHD risk groups (*very low risk* to *very high risk*) based on the NCEP Adult Treatment Panel III guidelines 2004 update (22,25). Individuals with prevalent CHD (based on self report of CHD, angina, or myocardial infarction) or CHD-risk equivalent condition (diabetes mellitus or self reported stroke) were classified as *high risk.* Prevalence of diabetes was assessed from self-report (if not diagnosed during pregnancy) and/or fasting blood glucose > 126 mg/dL (7 mmol/L). Those with prevalent CHD and one or more of diabetes mellitus, metabolic syndrome (assessment described below), and current smoking, were classified as *very high risk* (the highest category of risk in the NCEP 2004 Updated Guidelines). Ten-year global risk for CHD in individuals without prevalent CHD or CHD-risk-equivalent condition was estimated from the Framingham risk score, a gender-specific measure based on age, current smoking, total cholesterol, HDL cholesterol, blood pressure, and anti-hypertensive medication use. We scored those 80 years and older like 70-79 year olds (the oldest group in the scoring system). Based on the predicted global CHD risk, participants without prevalent CHD or CHD-risk-equivalent were classified as either *very low risk* (if predicted 10-year risk <1%); *low risk* (predicted 10-year risk 1-9%); *moderate risk* (if predicted 10-year risk 10-19% and without metabolic syndrome, not currently smoking, and serum CRP less than 3 mg/L), *moderate plus risk* (if predicted 10-year risk 10-19% and one or more of current smoking, metabolic syndrome, or CRP>=3 mg/L); or *high risk* (predicted 10-year risk ≥ 20%), consistent with risk categories used in clinical guidelines for cardiovascular risk management (22,23, 24,25). For the purposes of this risk categorization, individuals with missing data on metabolic syndrome, diabetes, or CRP were assumed to not meet that risk criterion. Misclassification

resulting from this imputation is limited to at most one category (e.g., classified moderate instead of moderate-plus, or high instead of very high).

We also created three metabolic syndrome /diabetes groups: *Low risk*: no metabolic syndrome and no diabetes mellitus; *Moderate risk*: metabolic syndrome but without diabetes mellitus, and *High risk*: diabetes mellitus. Based on the NCEP and EGIR definitions (22,26), metabolic syndrome was defined as having three or more of the following conditions: 1) Waist circumference >102 cm (40 inches) for men, >89 cm (35 inches) for women; 2) Fasting serum triglycerides>= 150 mg/dL (1.7 mmol/L); 3) HDL cholesterol< 40 mg/dL (1 mmol/L) for men, < 50 mg/dL (1.3 mmol/L) for women; 4) Blood pressure >=130mmHg systolic or >=85 mmHg diastolic, 5) Fasting blood glucose >= 110 mg/dL (6.1 mmol/L). Because of the high concordance between high fasting triglycerides and high waist circumference, we imputed one condition from the other (i.e., imputed high on missing condition if high on the other, low on missing condition if low on other) in 58 participants who were missing either triglycerides or waist circumference, to allow them to be classified.

Statistical Analyses

All analyses of ethnic and SES differences in risk were age-standardized; i.e., the age distribution of each predictor group (each SES group and each race/ethnicity group in unadjusted analyses, SES groups within race strata in race-stratified analyses, and race/ethnicity groups within SES strata in SES-stratified analyses) was standardized to the age distribution of the US in the 2000 census. Cumulative logistic regression models were then fit to determine the cumulative odds ratio (*cumulative OR*) for CHD risk and metabolic risk categories, with respect to SES and race/ethnicity, separately in each gender. Cumulative logistic regression for ordinal outcomes (such as the ordered CHD risk and metabolic risk categories) models the log of the cumulative odds at every outcome level (which are the odds of being in that or a higher outcome level versus all lower levels) as a common, single linear function of predictors plus level-specific intercepts. The cumulative OR for a predictor is the average effect of the predictor on cumulative odds at all outcome levels. In addition, we also estimated the ordinary odds ratio (*OR*) for the highest risk categories (very high risk for the 6-level global CHD risk outcome and diabetes mellitus for the 3-level metabolic risk outcome) versus all other risk categories, using ordinary (binary) logistic regression. Analyses were stratified by gender because CHD and metabolic risk categorizations were somewhat different in men and women (different thresholds for HDL, waist circumference, and different scoring systems for 10-year CHD risk), and because SES is felt to confer different benefits in men and women (3,32).

To quantify SES associations, gender and race/ethnicity-stratified, age-standardized models were fit with SES as primary predictor, and to assess race/ethnicity differences in risk, gender and SES-stratified, age-standardized models were fit with race/ethnicity as primary predictor. We chose to conduct race/ethnicity analyses stratified by SES and to conduct SES analyses stratified by race/ethnicity to minimize residual confounding, because of the preponderance of evidence that SES-risk associations vary by race/ethnicity (11,12,13,14). Given the existence of SES-race/ethnicity interactions, one cannot completely control for SES in a race/ethnicity analysis simply by including SES as a covariate in regression. Similarly, simply adding race/ethnicity to an SES regression model will not eliminate confounding by race/ethnicity.

To assess the role of lifestyle and central obesity in risk disparities, we added current smoking, physical activity (any moderate or vigorous activity vs. neither) and high waist circumference (>40 inches for men, >35 for women) to the models. We chose to study the role of abdominal obesity rather than generalized obesity, because of the former's stronger associations with metabolic and cardiovascular risk (22,26).

Stata version 9 was used for all analyses. CHD risk analyses were weighted by NHANES exam weights, and metabolic risk analyses were weighted by NHANES fasting sample weights, to account for sampling probability and non-response. To account for clustering within primary sampling units (because of the survey design), we used Stata's cluster option and robust variance estimation.

RESULTS

The analytic samples were older, more likely to be Non-Hispanic White and from higher socioeconomic strata, and had better biological profiles than the rest of the NHANES sample (See Table 1). Median age in the analytic samples was 45 years, 49% were male, 73% were Non-Hispanic White, 18% had less than high school education, median PIR was 3.1 and 12% had PIR less than 1, 11-12% fell in the low SES category, and 45% were classified high SES. There were marked SES differences between race/ethnicity groups: SES was highest in Non-Hispanic Whites and lowest in Foreign-born Mexican Americans. There were also marked differences by race/ethnicity in lifestyle choices and prevalence of central obesity (See Table 2). Among women, prevalence of both former smoking and current smoking was highest in Non Hispanic White women and lowest in Foreign-born Mexican American women (Table 2). Among men, current smoking prevalence was not very different between race/ethnicity groups, but Non-Hispanic Black men were the least likely to be former smokers. Non-Hispanic White men and women were also the most likely to be physically active (at moderate or vigorous level) and Foreign-born Mexican American men and women least likely (Table 2). On the other hand, central obesity prevalence was lowest in Foreign-born Mexican American men and women, and highest in Non-Hispanic White and US-born Mexican American men and Non-Hispanic Black and US-born Mexican American women (Table 2).

In unadjusted (but age-standardized) analyses, both education and income individually, and the combined SES variable had strong inverse relationships with global CHD risk and metabolic risk (data not shown). However, tests of interaction in gender-stratified, logistic regression models indicated that race/ethnicity modified these SES associations in both men and women: For instance, (age-standardized) SES associations with the odds of very high CHD risk (the highest CHD risk group) were significantly weaker in US-born Mexican American men (p=0.005) and stronger in foreign-born Mexican American women (p=0.02) than in the majority group (Non-Hispanic White). Similarly, SES associations with metabolic risk were weaker in both US-born Mexican men (p=0.1) and Non-Hispanic Black men (p=0.07) than in Non-Hispanic White men, and SES associations with overt diabetes (the highest metabolic risk group) were weaker in foreign-born Mexican American men (p=0.08) but stronger in foreign-born Mexican American women (p=0.09) than in Non-Hispanic Whites.

In analyses stratified by gender and race/ethnicity, inverse SES gradients were seen with global CHD risk in men and women from all race/ethnicity groups, with one notable exception: There was no SES gradient in foreign-born Mexican American men. Comparing low SES to high SES, cumulative OR for estimated 10-year CHD risk levels ranged from *1.4* to *2.8* and OR for being in the highest risk group ranged from *2.9* to *5.8* (Table 3a). After adjusting for lifestyle factors (physical activity and smoking) and central obesity, the magnitude of the SES-risk gradient diminished in all strata – See Table 3a.

In women from three of the four race/ethnicity groups and in non-Hispanic White men, SES also had strong inverse associations with metabolic syndrome / diabetes prevalence. The strongest SES effect was in foreign-born Mexican American women: Cumulative OR *2.5* (95% confidence interval (CI): 1.5 to 4.1) and diabetes OR *5.0* (95% CI: 2.2 to 11.0), for low SES compared to middle and high SES. Adjusting for physical activity and smoking only modestly attenuated these SES gradients – See Table 3b.

In unadjusted (but age-standardized) analyses of race/ethnicity differences in risks, Non-Hispanic Blacks and US-born Mexican Americans were more likely than Non-Hispanic Whites and Foreign-born Mexican Americans to be in the *highest* CHD risk categories, and both US-born and Foreign-born Mexican Americans had *higher* prevalence of metabolic syndrome / diabetes than Non-Hispanic Whites (data not shown). However, after stratification by gender and SES, race/ethnicity disparities in global CHD risk were not consistently seen in all strata. For instance, only *middle-SES*, Non-Hispanic Black *women* and *high-SES*, US-born Mexican American *men* had *increased* risk compared to the majority group at comparable SES (Table 4a); the largest disparity was for high-SES, US-born, Mexican American men: Compared to high-SES Non-Hispanic White men, their OR for being in the highest risk group was *4.1* (95% CI: 1.8 to 9.2). Foreign-born Mexican Americans (both men and women) showed *reduced* CHD risk at multiple SES levels, the largest advantage was in middle-SES, foreign-born Mexican American women: Compared to middle-SES Non-Hispanic White women, their OR for being in the highest risk group was 0.1 (95% CI: 0.01 to 0.4). These few race/ethnicity differences were not substantially affected by controls for lifestyle factors and central obesity – See Table 4a.

Race/ethnicity disparities in metabolic syndrome/diabetes risk were also not uniform across gender/SES strata: *High-SES*, Non-Hispanic Black *men*, *middle-SES*, Non-Hispanic Black *women*, *middle and high-SES*, US-born Mexican American *men*, *middle-SES*, foreign-born Mexican American *men*, and *low-SES*, foreign-born Mexican American *women* had *higher* prevalence of diabetes than Non-Hispanic Whites of the same gender and SES (Table 4b); the largest disparity was for high-SES, US-born Mexican American men: Compared to high-SES, Non-Hispanic White men, their diabetes OR was *3.0* (95% CI 1.4 to 6.1). These race/ethnicity differences were not substantially affected by controls for lifestyle factors – See Table 4b.

DISCUSSION

Our objective was to determine the independent associations of SES and race/ethnicity with global CHD risk and metabolic syndrome/diabetes prevalence in the US population. SES was strongly, inversely associated with global CHD risk in both men and women from all race/ethnicity groups, except in Foreign-born Mexican American men. SES was also strongly, inversely associated with metabolic risk in women from three of the four race/ethnicity groups and in Non-Hispanic White men. Stronger SES associations in women than in men have been previously reported (15,33,34,35), consistent with stronger SES-obesity relationships in women (36). Previous studies have also found that education and income gradients in health are weaker in African Americans than in Caucasian Americans (11,12,13,14,34); however, SES, defined by combining education and income, showed strong CHD risk gradients in all four race/ethnicity groups. This inconsistency is likely the result of differential employment and income returns from education (15,37,38,39), so that education or income on its own does not adequately capture social standing.

Some but not all of the SES associations with global CHD risk were explained by differences in health behaviors (physical activity and smoking) and central obesity. Other mediators of SES effects are likely to include neighborhood deprivation, access to medical care, and the physiological impact of daily stresses related to social position.

In contrast to the strong SES gradients with risks, race/ethnicity differences in risks were not consistent across SES and gender strata: Compared to Non-Hispanic Whites, global CHD risk was higher *only* in upper-SES, US-born, Mexican American *men* and middle-SES, Non-Hispanic Black *women*, and *lower* in foreign-born, Mexican American men and women. Diabetes prevalence was higher in minority groups, but only in some SES strata, and metabolic risk was actually *lower* in middle-SES, foreign-born Mexican American women. Previous

studies that have examined ethnic disparities after stratifying by SES have also found black-white differences in risks only in middle and high SES strata, but these studies did not separate men from women, and did not examine predicted global CHD risk (9). Lower CHD risk in foreign-born Mexican Americans has been previously noted (29,30,31), and it has been recognized that the Hispanic paradox is actually an immigrant health phenomenon, resulting from selection pressures in migration that favor healthy immigrants (29,31,40), return emigration of sicker individuals ('salmon bias'), and better lifestyle choices (41). Foreign-born Mexican Americans indeed had the lowest rates of current smoking and central obesity, but the risk reduction did not diminish with controls for lifestyle, suggesting that lack of acculturation is not a major explanation for this phenomenon.

The consistency of SES-risk gradients across gender and race/ethnicity strata, relative to the inconsistency of race/ethnicity disparities in risk across SES strata, suggest that SES, rather than race/ethnicity, is the main driver of social disparities in cardiovascular risk in the US. Previous studies in some populations have also found that SES eclipses race/ethnicity as a predictor of risks (42), and that SES explains much of the race/ethnicity disparities in health risks (43,44).

This study has some limitations that need to be acknowledged. The possibility of false discovery as a result of multiple testing hampers our ability to infer real race/ethnicity differences in risk, since differences were seen only in some, not all, SES strata; this is less of a concern with SES gradients, which were consistent across race/ethnicity groups and gender, but is a real possibility for ethnic differences that were detected, since they were seen in only some, not all, SES and gender strata.. Secondly, just as SES-risk associations varied by race/ethnicity, the effects of health behaviors on risks may also differ by ethnicity and SES (45), in which case, one cannot completely control for health behaviors by including them as covariates in the model. Also, abdominal obesity which was used as a surrogate for health behaviors related to diet and exercise, is also affected by family history and other factors, and so, control for health behaviors may not have been adequate. Finally, differential response by SES within race/ethnic groups could have biased our findings, although the use of weights designed to make the sample nationally representative should have diminished such bias.

The study's limitations are outweighed by its strengths. In contrast to previous studies that have examined disparities in individual risk factors for CHD, we examined global risk for CHD. Disparities in individual risk factors can be in opposite directions (5,21) and effects of risk factors can vary by gender; thus disparities in a global risk measure are more relevant to actual outcomes. A few studies have previously examined disparities in global CHD risk, but have either studied only one gender (13) or not controlled for race/ethnicity (36) or SES (46, 47). Since the predictive ability of the Framingham risk score may vary by SES and race/ethnicity (48), we also examined ORs for prevalent CHD or CHD risk-equivalent (highest risk group), and the disparities were similar to those for predicted risk. Secondly, unlike other studies of ethnic disparities, we conducted SES-stratified analyses, the more appropriate way to control for SES when SES associations differ between groups. Third, we separated US-born from foreign-born Mexican Americans, since their social experiences are distinct, and we found important differences.

In conclusion, this large, national study documents strong, inverse SES gradients with CHD risk in all race/ethnicity groups, and demonstrates that race/ethnicity disparities in risk are primarily due to SES differences between the groups. Healthy lifestyle choices and avoidance of central obesity appear to dampen SES associations. Socioeconomically disadvantaged individuals need to be specifically targeted for early risk detection and management and health behavior counseling if we are to improve the cardiovascular health of the nation.

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ABBREVIATIONS / ACRONYMS

CHD	Coronary Heart Disease
SES	Socio-economic Status
NCEP	National Cholesterol Education Program
NHANES	National Health and Nutrition Examination Survey
US	United States

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Table 1

Descriptive Characteristics of Study Samples from National Health and Nutrition Examination Survey (NHANES) 2001-2006, at least 20 years of age, and not pregnant; weighted to be nationally representative

	NHANES Examination Sample		NHANES Fasting Sample	
Characteristics	**CHD Risk Sample (N=12,154)**	**Excluded from CHD Risk Sample (N=1,548)**	**Metabolic Risk Sample (N=5,648)**	**Excluded from Metabolic Sample (N=160)**
Median Age (years)	45.0*	42.0	45.0	48.0
Age Groups (%)				
20-29	17.7*	24.8	18.3	17.4
30-39	19.6	19.9	20.2	13.0
40-49	22.3	21.7	22.2	22.4
50-59	17.5	15.2	17.1	17.3
60-69	10.9	8.4	11.2	14.5
70-79	7.9	5.6	7.3	7.3
80+	4.2	4.5	3.7	8.2
Male (%)	48.6	51.7	49.2*	32.8
Race/Ethnicity (%)				
Non-Hispanic White	73.4*	60.9	72.6*	59.5
Non-Hispanic Black	10.5*	16.4	10.9*	17.5
US-born Mexican American	2.8	2.5	2.8	1.4
Foreign-born Mexican American	4.4*	6.6	4.7	5.5
Other	8.8*	13.6	9.0*	16.1
Education (%)				
< High School	18.1*	22.3	18.0	23.9
High School	25.9	26.1	26.0	27.4
> High School	56.0	51.6	55.9	48.6
Median Poverty Income Ratio	3.1*	2.7	3.1*	2.5
Poverty Income Ratio (%)				
<1.0	12.0*	15.9	11.6*	20.3
1.0-1.99	20.2	21.5	20.7	19.7
2.0-2.99	16.0	16.4	15.9	20.3
3.0-3.99	15.1	15.4	15.7	15.5
4.0-4.99	11.5	11.0	10.5	9.7
≥5.0	25.1	19.8	25.6	14.7
Socioeconomic Status (%) †				
Low	11.6*	14.4	11.4	15.5
Middle	43.3	45.4	44.2	44.7
High	45.1	40.2	44.5	39.8
Current Smoking (%)	24.9	26.4	25.1	28.3
Mean Blood Pressure (mms Hg)				

Characteristics	NHANES Examination Sample		NHANES Fasting Sample	
	CHD Risk Sample (N=12,154)	Excluded from CHD Risk Sample (N=1,548)	Metabolic Risk Sample (N=5,648)	Excluded from Metabolic Sample (N=160)
Systolic	123.1*	125.4	122.5	126.1
Diastolic	71.8*	73.7	71.5	72.4
High Blood Pressure (%) ‡	33.1	31.6	32.1	43.8
Anti-hypertensive meds (%)	12.6*	22.8	14.2	9.0
Mean Total Cholesterol (mmol/L)	5.2	5.2	5.2	5.3
Mean Total Cholesterol (mg/dL)	200.7	200.6	199.2	203.7
Mean HDL Cholesterol (mmol/L)	1.4	1.3	1.4	1.2
Mean HDL Cholesterol (mg/dL)	53.4*	51.7	53.6*	48.2
Low HDL Cholesterol (%) §	29.2	30.5	27.8	37.8
Mean Triglycerides (mmol/L)	-	-	1.6	1.8
Mean Triglycerides (mg/dL)	-	-	145.8	161.6
High Triglycerides (%) ‡	-	-	31.9	36.6
Mean Plasma Glucose (mmol/L)	-	-	5.7	5.6
Mean Plasma Glucose (mg/dL)	-	-	102.0	100.4
High Glucose (%) ‡	-	-	16.3	16.8
Mean Waist Girth (cm)	96.8	97.5	97.2	97.4
High Waist Girth (%) ‡	49.3*	48.5	49.4	62.0
CHD Risk Levels (%) **				
Very low risk	29.5	-	30.5	-
Low risk	41.2	-	41.3	-
Moderate risk	3.7	-	2.6	-
Moderate plus risk	5.5	-	6.0	-
High risk	15.6	-	14.5	-
Very high risk	4.6	-	5.2	-
Metabolic Risk (%)				
No metabolic syndrome or diabetes	-	-	69.8	-
Metabolic syndrome††, no diabetes	-	-	20.0	-
Diabetes mellitus	-	-	10.2	-

* P<0.05 for statistical test of comparison of study sample with those excluded

† Low if education <high school and poverty-income ratio (PIR)<2; high if education >high school and PIR>=2; middle if neither high nor low

‡ High as defined by National Cholesterol Education Program (NCEP) conditions for metabolic syndrome: Triglycerides >=1.7 mmol/L, blood pressure >=130 />=85 mmHg, glucose >=6.1 mmol/L, waist circumference >102 for men and >89 for women

§ Low as defined by National Cholesterol Education Program criteria for metabolic syndrome: HDL <1 mmol/L for men and <1.3 for women

** Levels of estimated 10-year risk for CHD (coronary heart disease) events. Please refer to text for details of how the risk is estimated and for the definitions of the 6 levels

††Metabolic syndrome present if 3 or more of the following: Triglycerides >=1.7 mmol/L, blood pressure >=130 />=85 mmHg, glucose >=6.1 mmol/L, waist circumference >102 for men and >89 for women, HDL <1 mmol/L for men and <1.3 for women

Table 2

Socioeconomic Status, Lifestyle Choices, and Central Obesity by Race/Ethnicity and Gender, United States 2001-2006

	Non-Hispanic White		Non-Hispanic Black		US-Born Mexican American		Foreign-Born Mexican American	
	Men (N=3278)	**Women (N=3182)**	**Men (N=1203)**	**Women (N=1233)**	**Men (N=458)**	**Women (N=540)**	**Men (N=778)**	**Women (N=647)**
Education (%) *								
< High school	12.2	12.2	31.4	28.9	32.6	29.3	75.0	74.1
High school	27.6	26.5	26.5	22.4	24.3	25.4	13.4	13.0
> High school	60.2	61.3	42.1	48.7	43.0	45.3	11.6	12.9
P value†	*reference*	*reference*	*<0.0001*	*<0.0001*	*<0.0001*	*<0.0001*	*<0.0001*	*<0.0001*
Poverty Income Ratio (%)								
<1	7.0	9.9	17.8	25.8	14.8	19.1	34.8	38.3
1-1.99	16.0	19.1	26.6	28.4	25.2	25.8	34.8	35.9
≥2	77.0	71.0	55.6	45.8	60.0	55.1	30.5	25.8
P value	*reference*	*reference*	*<0.0001*	*<0.0001*	*<0.0001*	*<0.0001*	*<0.0001*	*<0.0001*
Socioeconomic Status (%) ‡								
Low	6.3	7.4	21.1	21.6	22.3	18.7	55.6	58.1
Middle	42.4	42.7	48.1	48.3	44.8	51.2	38.4	36.3
High	51.3	50.0	30.8	30.2	32.9	30.1	6.0	5.6
P value	*reference*	*reference*	*<0.0001*	*<0.0001*	*<0.0001*	*<0.0001*	*<0.0001*	*<0.0001*
Current smoking (%)	27.3	24.7	31.1	20.5	29.3	16.3	26.9	10.2
P value	*reference*	*reference*	*0.05*	*0.03*	*0.4*	*0.001*	*0.8*	*<0.0001*
Former smoking (%)	30.5	23.2	21.2	14.3	28.7	20.5	31.1	12.3
P value	*reference*	*reference*	*<0.0001*	*<0.0001*	*0.4*	*0.2*	*0.8*	*<0.0001*
Physical activity (%) §	70.5	68.7	59.0	52.6	61.3	63.2	40.5	35.6
P value	*reference*	*reference*	*<0.0001*	*<0.0001*	*0.006*	*0.1*	*<0.0001*	*<0.0001*
Central obesity (%) **	44.4	52.4	34.9	69.8	42.7	65.4	31.7	32.0
P value	*reference*	*reference*	*<0.0001*	*<0.0001*	*0.5*	*<0.0001*	*<0.0001*	*<0.0001*

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[*] Weighted proportions: After weighting of data from the CHD Risk Sample to make it nationally representative, and after age-standardization of each ethnicity/gender group to the age distribution of the US in the 2000 census.

[†] P value for within-gender comparison to Non-Hispanic Whites

[‡] Defined low if education <high school and poverty-income ratio (PIR)>2.0; high if education >high school and PIR>=2.0; middle otherwise

[§] Any reported moderate or vigorous physical activity

[**] Defined as waist circumference >102 cm for men and >89 cm for women

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Table 3a

Socioeconomic Status (SES) Differences in Estimated 10-year CHD Risk (Odds Ratios Compared to *High SES*, with 95% Confidence Intervals), Stratified by Race/Ethnicity, Before and After Adjusting for Physical Activity, Current Smoking, and Central Obesity; United States 2001-2006

	Unadjusted *		**Adjusted**	
	Cumulative Odds Ratio	**Odds Ratio for Highest Risk Group**	**Cumulative Odds Ratio**	**Odds Ratio for Highest Risk Group**
Non-Hispanic White Men (Reference group: High SES, n=1405)				
Low SES (n=339)	**2.0** (1.4-2.9)	**4.0** (2.3-6.9)	1.2 (0.8-1.8)	**2.3** (1.3-4.1)
Middle SES (n=1364)	**1.4** (1.2-1.6)	**1.9** (1.4-2.6)	1.0 (0.9-1.2)	**1.6** (1.1-2.2)
P-value for Trend	*<0.0001*	*<0.0001*	0.5	*0.001*
Non-Hispanic White Women (Reference group: High SES, n=1317)				
Low SES (n=325)	**2.2** (1.6-3.0)	**4.6** (2.5-8.6)	1.2 (0.8-1.6)	**2.4** (1.2-4.6)
Middle SES (n=1368)	**1.5** (1.3-1.8)	**2.1** (1.3-3.3)	1.1 (0.9-1.3)	1.4 (0.8-2.2)
P-value for Trend	*<0.0001*	*<0.0001*	0.3	*0.02*
Non-Hispanic Black Men (Reference group: High SES, n=348)				
Low SES (n=245)	**1.4** (1.0-2.0)	**3.3** (1.7-6.6)	0.9 (0.6-1.3)	**2.8** (1.3-6.2)
Middle SES (n=544)	1.2 (0.9-1.5)	**2.3** (1.1-4.9)	0.9 (0.6-1.2)	**2.1** (1.0-4.5)
P-value for Trend	0.04	*<0.0001*	0.6	*0.009*
Non-Hispanic Black Women (Reference group: High SES, n=346)				
Low SES (n=251)	**1.7** (1.2-2.4)	**2.8** (1.3-6.3)	1.1 (0.7-1.6)	1.6 (0.6-4.5)
Middle SES (n=555)	**1.5** (1.1-1.9)	**3.2** (1.5-6.6)	1.1 (0.9-1.5)	**2.8** (1.1-7.3)
P-value for Trend	*0.001*	*0.002*	0.6	0.4
US Born Mexican American Men (Reference group: High SES, n=130)				
Low SES (109)	**1.7** (1.0-2.9)	1.5 (0.6-3.8)	1.3 (0.6-2.5)	1.4 (0.4-4.7)
Middle SES (n=200)	1.2 (0.7-2.0)	0.4 (0.1-1.1)	1.0 (0.6-1.7)	**0.3** (0.1-1.0)
P-value for Trend	0.08	0.5	0.5	0.7
US Born Mexican American Women (Reference group: High SES, n=141)				
Low SES (n=121)	**2.8** (1.4-5.6)	**5.8** (0.9-35.4)	1.4 (0.7-3.2)	2.3 (0.5-11.0)
Middle SES (n=252)	1.4 (0.9-2.2)	**3.6** (0.6-22.2)	1.0 (0.6-1.9)	1.8 (0.3-11.0)
P-value for Trend	*0.005*	*0.02*	0.3	0.2
Foreign Born Mexican American Men (Reference group: Middle and High SES, n=320)‡				
Low SES (n=407)	1.2 (0.9-1.6)	1.1 (0.3-3.5)	1.1 (0.8-1.5)	0.9 (0.3-3.1)
P-value	0.3	0.9	0.6	0.9
Foreign Born Mexican American Women (Reference group: Middle and High SES, n=247) ‡				
Low SES (n=351)	**1.5** (1.1-2.0)	**27** (3.6-204)	1.4 (0.9-2.0)	**24** (2.3-246)
P-value	*0.02*	*0.001*	0.1	*0.007*

Table 3b

Socioeconomic Status (SES) Differences in Metabolic Risk (Odds Ratios Compared to *High SES*, with 95% Confidence Intervals), Stratified by Race/Ethnicity, Before and After Adjusting for Physical Activity and Current Smoking; United States 2001-2006

	Unadjusted*		Adjusted	
	Cumulative Odds Ratio	**Odds Ratio for Diabetes**	**Cumulative Odds Ratio**	**Odds Ratio for Diabetes**
Non-Hispanic White Men (Reference group: High SES, n=688)				
Low SES (n=162)	**1.8** (1.1-2.9)	**2.1** (1.2-3.6)	**2.0** (1.3-3.0)	**1.8** (1.0-3.3)
Middle SES (n=643)	**1.4** (1.1-1.8)	1.2 (0.8-1.7)	**1.5** (1.1-1.9)	1.0 (0.7-1.6)
P-value for Trend	*0.004*	*0.02*	*<0.0001*	0.1
Non-Hispanic White Women (Reference group: High SES, n=613)				
Low SES (n=149)	**2.5** (1.5-4.2)	**2.4** (1.2-5.1)	**1.9** (1.1-3.4)	1.8 (0.8-4.0)
Middle SES (n=611)	**1.7** (1.3-2.2)	**1.6** (1.0-2.5)	**1.4** (1.0-1.9)	1.3 (0.8-2.1)
P-value for Trend	*<0.0001*	*0.009*	*0.02*	0.1
Non-Hispanic Black Men (Reference group: High SES, n=161)				
Low SES (n=108)	1.1 (0.7-1.7)	1.3 (0.8-2.4)	1.4 (0.8-2.6)	1.7 (0.8-3.5)
Middle SES (n=246)	0.8 (0.5-1.3)	0.8 (0.5-1.4)	0.8 (0.5-1.4)	0.7 (0.4-1.4)
P-value for Trend	0.96	0.4	0.4	0.3
Non-Hispanic Black Women (Reference group: High SES, n=160)				
Low SES (n=91)	1.7 (0.9-3.1)	2.1 (0.9-5.2)	1.4 (0.7-2.9)	1.6 (0.6-4.1)
Middle SES (n=256)	**1.8** (1.1-2.9)	**2.4** (1.1-5.4)	1.5 (0.9-2.5)	1.9 (0.9-4.2)
P-value for Trend	0.05	*0.05*	0.3	*0.3*
US Born Mexican American Men (Reference group: High SES, n=68)				
Low and Middle SES (n=148)†	0.9 (0.6-1.5)	1.0 (0.5-2.1)	0.7 (0.4-1.4)	0.7 (0.3-1.6)
P-value	0.8	0.99	0.3	0.4
US Born Mexican American Women (Reference group: High SES, n=68)				
Low and Middle SES (n=169)†	1.9 (0.8-4.2)	2.7 (0.8-9.2)	2.3 (0.9-5.7)	2.1 (0.6-7.4)
P-value	0.1	0.1	*0.08*	0.2
Foreign Born Mexican American Men (Reference group: Middle and High SES, n=152)‡				
Low SES (n=209)	1.0 (0.6-1.6)	0.7 (0.3-1.4)	1.0 (0.6-1.6)	0.7 (0.4-1.4)
P-value	0.9	*0.3*	0.98	0.3
Foreign Born Mexican American Women (Reference group: Middle and High SES, n=110)‡				
Low SES (n=156)	**2.5** (1.5-4.2)	**4.8** (2.2-11)	**1.8** (1.1-2.9)	**3.5** (1.7-7.1)
P-value	*<0.0001*	*<0.0001*	*0.02*	*0.001*

*All analyses (including the ones labeled unadjusted) are weighted by NHANES weights and age-standardized, so that groups being compared have similar age distributions, and associations represent US associations. Bold denotes p<0.05

†Low SES group had less than 50 people and was combined with middle SES

‡High SES group had less than 50 people and was combined with middle SES

Table 4a

Ethnic Differences in Estimated 10-year CHD Risk (Odds Ratios Compared to *Non-Hispanic Whites*, with 95% Confidence Intervals), Stratified by Socioeconomic Status (SES), Before and After Adjusting for Physical Activity, Current Smoking, and Central Obesity; United States 2001-2006

	Unadjusted*		Adjusted	
	Cumulative Odds Ratio	**Odds Ratio for Highest Risk Group**	**Cumulative Odds Ratio**	**Odds Ratio for Highest Risk Group**
Low SES Men (Reference group: Non-Hispanic White, n=339)				
Non-Hispanic Blacks (n=245)	0.8 (0.5-1.2)	0.7 (0.4-1.4)	1.0 (0.7-1.7)	1.2 (0.6-2.4)
US-born Mexican Americans (n=109)	1.0 (0.6-1.8)	1.5 (0.7-3.2)	1.3 (0.7-2.5)	**2.2** (1.0-4.7)
Foreign-born Mexican Americans (n=407)	**0.6** (0.4-0.9)	**0.2** (0.1-0.5)	0.9 (0.6-1.4)	**0.3** (0.1-0.7)
Low SES Women (Reference group: Non-Hispanic White, n=325)				
Non-Hispanic Blacks (n=251)	0.9 (0.7-1.3)	0.6 (0.3-1.3)	0.9 (0.6-1.2)	0.5 (0.2-1.3)
US-born Mexican Americans (n=121)	1.3 (0.7-2.1)	0.8 (0.3-1.9)	1.3 (0.7-2.2)	1.1 (0.4-2.6)
Foreign-born Mexican Americans (n=351)	0.7 (0.5-1.1)	0.7 (0.3-1.4)	0.8 (0.5-1.1)	1.0 (0.4-2.5)
Middle SES Men (Reference group: Non-Hispanic White, n=1364)				
Non-Hispanic Blacks (n=544)	0.9 (0.8-1.1)	1.1 (0.7-1.7)	1.1 (0.9-1.3)	1.2 (0.8-1.9)
US-born Mexican Americans (n=200)	1.0 (0.7-1.4)	0.8 (0.4-1.5)	1.1 (0.8-1.6)	0.9 (0.4-1.7)
Foreign-born Mexican Americans (n=274)	0.8 (0.6-1.1)	**0.4** (0.2-0.9)	1.0 (0.7-1.3)	0.4 (0.2-1.1)
Middle SES Women (Reference group: Non-Hispanic White, n=1368)				
Non-Hispanic Blacks (n=555)	1.1 (0.8-1.4)	**1.5** (1.0-2.4)	1.0 (0.8-1.2)	1.4 (0.9-2.3)
US-born Mexican Americans (n=252)	0.8 (0.6-1.1)	1.1 (0.6-2.2)	0.9 (0.7-1.3)	1.3 (0.6-2.5)
Foreign-born Mexican Americans (n=216)	**0.7** (0.5-1.0)	**0.1** (0.01-0.4)	**0.7** (0.4-1.0)	**0.1** (0.01-0.6)
High SES Men† (Reference group: Non-Hispanic White, n=1405)				
Non-Hispanic Blacks (n=348)	1.1 (0.8-1.4)	0.9 (0.5-1.6)	1.2 (0.9-1.6)	0.8 (0.4-1.6)
US-born Mexican Americans (n=130)	1.1 (0.7-1.9)	**4.1** (1.8-9.2)	1.1 (0.6-1.9)	**4.4** (1.9-10.2)
High SES Women† (Reference group: Non-Hispanic White, n=1317)				
Non-Hispanic Blacks (n=346)	1.1 (0.8-1.4)	1.0 (0.5-2.0)	0.9 (0.7-1.3)	0.7 (0.3-1.6)
US-born Mexican Americans (n=141)	0.9 (0.5-1.4)	0.7 (0.1-3.4)	0.8 (0.5-1.5)	0.7 (0.1-3.7)

*All analyses (including the ones labeled unadjusted) are weighted by NHANES weights and age-standardized, so that groups being compared have similar age distributions, and associations represent US associations. **Bold** denotes $p<0.05$

†There were fewer than 50 high-SES, foreign-born Mexican American men (similarly, women); they were excluded.

Table 4b

Ethnic Differences in Metabolic Risk (Odds Ratios Compared to *Non-Hispanic Whites*, with 95% Confidence Intervals), Stratified by Socioeconomic Status (SES), Before and After Adjusting for Physical Activity and Current Smoking; United States 2001-2006

	Unadjusted*		Adjusted	
	Cumulative Odds Ratio	**Odds Ratio for Diabetes**	**Cumulative Odds Ratio**	**Odds Ratio for Diabetes**
Low SES† Men (Reference group: Non-Hispanic White, n=162)				
Non-Hispanic Blacks (n=108)	0.7 (0.4-1.3)	1.3 (0.6-2.6)	1.4 (0.7-2.8)	**2.2** (1.0-5.0)
Foreign-born Mexican Americans (n=209)	0.7 (0.4-1.2)	0.7 (0.4-1.5)	0.8 (0.5-1.4)	0.7 (0.3-1.6)
Low SES† Women (Reference group: Non-Hispanic White, n=149)				
Non-Hispanic Blacks (n=91)	0.8 (0.4-1.5)	1.4 (0.6-3.3)	0.7 (0.3-1.6)	1.3 (0.5-3.5)
Foreign-born Mexican Americans (n=156)	1.3 (0.8-2.1)	**2.4** (1.1-4.9)	1.3 (0.7-2.2)	**2.4** (1.1-5.3)
Middle SES Men (Reference group: Non-Hispanic White, n=643)				
Non-Hispanic Blacks (n=246)	**0.7** (0.5-0.9)	1.4 (0.9-2.2)	0.9 (0.6-1.4)	**1.7** (1.0-2.7)
US-born Mexican Americans (n=104)	1.4 (0.9-2.2)	**2.5** (1.3-4.6)	**1.6** (1.0-2.4)	**2.8** (1.5-5.2)
Foreign-born Mexican Americans (n=129)	1.0 (0.6-1.5)	**1.8** (1.0-3.4)	1.3 (0.8-2.1)	**2.2** (1.1-4.2)
Middle SES Women (Reference group: Non-Hispanic White, n=611)				
Non-Hispanic Blacks (n=256)	1.3 (0.9-1.9)	**2.4** (1.5-4.1)	0.9 (0.6-1.3)	**1.9** (1.1-3.3)
US-born Mexican Americans (n=119)	1.0 (0.7-1.6)	1.4 (0.8-2.6)	0.9 (0.5-1.7)	1.3 (0.7-2.5)
Foreign-born Mexican Americans (n=97)	0.7 (0.4-1.1)	0.9 (0.4-1.8)	0.6 (0.4-1.0)	0.8 (0.4-1.7)
High SES Men‡ (Reference group: Non-Hispanic White, n=688)				
Non-Hispanic Blacks (n=161)	1.1 (0.7-1.7)	**2.0** (1.2-3.2)	1.5 (0.9-2.6)	**2.3** (1.4-3.8)
US-born Mexican Americans (n=68)	**2.0** (1.1-3.6)	**2.8** (1.4-5.7)	**2.5** (1.2-5.3)	**3.2** (1.5-7.1)
High SES Women‡ (Reference group: Non-Hispanic White, n=613)				
Non-Hispanic Blacks (n=160)	1.1 (0.7-1.7)	1.6 (0.8-3.3)	0.8 (0.5-1.3)	1.4 (0.7-2.9)
US-born Mexican Americans (n=68)	1.2 (0.6-2.5)	1.3 (0.5-3.6)	0.9 (0.4-1.8)	1.1 (0.4-3.0)

*All analyses (including those labeled unadjusted) are weighted by NHANES weights and age-standardized, so that groups being compared have similar age distributions, and associations represent US associations. **Bold** denotes $p<0.05$

†There were 50 or fewer low-SES, US-born Mexican American men (similarly, women); they were excluded

‡There were fewer than 50 high-SES, foreign-born Mexican American men (similarly, women); they were excluded