Education, Income and Ethnic Differences in Cumulative Biological Risk Profiles in a National Sample of US Adults: NHANES III (1988-1994)

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Abstract

Data from the nationally representative NHANES III cohort were used to examine the hypothesis that socio-economic status is consistently and negatively associated with levels of biological risk, as measured by nine biological parameters known to affect health risks (i.e., diastolic and systolic blood pressure, pulse, HDL and total cholesterol, glycosylated hemoglobin, c-reactive protein, albumin and waist-hip ratio), resulting in greater cumulative burdens of biological risk among those of lower education and/or income. As hypothesized, consistent education and income gradients were seen for biological parameters reflecting cardiovascular, metabolic and inflammatory risk: those with lower education and income exhibiting greater prevalence of high risk values for each of nine individual biological risk factors. Significant education and income gradients were also seen for summary indices reflecting cumulative burdens of cardiovascular, metabolic and inflammatory risks as well as overall total biological risks. There were no significant ethnic differences in the patterns of association between socio-economic status and biological risks. Multivariable cumulative logistic regression models revealed that the education and income effects were each independently and negatively associated with cumulative biological risks, and that these effects remained significant independent of age, gender, ethnicity and lifestyle factors such as smoking and physical activity.

Keywords: biological markers, biological aging, education, ethnicity, income, social class

Abbreviations: AL, allostatic load; CRP, c-reactive protein; DBP, diastolic blood pressure; MEC, mobile examination center; PIR, poverty income ratio; PSU, primary sampling unit; SBP, systolic blood pressure; SES, socio-economic status; WHR, waist-hip-ratio

Socio-economic status (SES) has long been a focus of interest for those seeking to elucidate the patterning of health disparities and the underlying causes for these disparities (1-3). Decades of research have clearly delineated the fact that SES is a consistent and strong predictor of health differentials, with lower SES generally associated with poorer outcomes for most major types of morbidity and mortality (1, 2, 4-6). The data also show that these health disparities reflect a gradient of effects, rather than a threshold effect of poverty versus non-poverty, with those in the middle class experiencing better health than those below them but worse health than those above them (7-10). Existing SES health gradients, however, remain incompletely understood.

A range of different factors have been postulated to contribute to these SES gradients, including differences in health behaviors, availability and quality of health care, differential exposure to environmental hazards and stressors/demands and the presence of fewer available financial and psychosocial resources with which to address life challenges (1, 2, 11). These (and other) SES-related differences in experiences and exposures ultimately "get under the skin" to impact on biological processes in ways that result in increased health risks. Indeed, a large and growing body of evidence documents SES gradients in major cardiovascular risk factors such as obesity, blood pressure, lipid profiles and diabetes (11-17) as well as markers of inflammation (18-20). Largely absent to date, however, has been a focus on the multi-systems nature of these risks (reflect the broad scope of SES gradients in biological risks) nor has there been much attention to the SES differentials in resulting cumulative burdens of biological risk (21-23).

Analyses of data from the MacArthur Studies of Successful Aging have previously shown that a cumulative index of biological risk (allostatic load) is negatively associated with SES, and mediates some 35 percent of the education differentials in mortality (24). Examination

of individual components of the index as well as sub-scales reflecting major systems (e.g., cardiovascular, stress hormones, inflammation) revealed consistent, though more modest effects in all cases. Parallel evidence linking lower SES to greater cumulative biological risk has also been documented in the Normative Aging Study (25).

Several studies also suggest that SES gradients may be stronger among whites as compared with blacks (17, 26), a finding consistent with the hypothesis that minority status (and its associations with discrimination and disadvantage; 27, 28) may result in larger deficits among those of lower SES and smaller gains among those of higher SES. Recent analysis of National Health and Nutrition Examination Survey (NHANES) IV (1999-2002) data by Geronimus and colleagues (29) documented that blacks have significantly higher overall allostatic load scores, even after adjusting for SES (as measured by the poverty income ratio – a measure of household income relative to the US poverty threshold). Data from the NHANES III survey (1988-1994) likewise show higher overall levels of biological risk in non-whites (both blacks and Mexican Americans), though these differences are largely found among those less than 70 years of age (30).

Analyses presented here use data from the National Health and Nutrition Examination Survey (NHANES III) to examine the hypothesis that education and income gradients in biological risks would be evident across a set of nine major biological parameters, resulting in significant differences in cumulative burdens of biological risk. Additional analyses tested for possible ethnic differences in the patterns of these SES-biological risk associations.

MATERIALS AND METHODS

The study population reflects adults aged 20 and older (N=18,825) in the National Health and Nutrition Examination Survey (NHANES) III (1988-1994), a nationally representative sample of the U.S. population with interview, clinical exam, and laboratory components (31). We excluded those with no mobile examine center (MEC) visit (n=2,252) (i.e., missing most biological data) and those missing education information (n=115) (our primary index of SES). The household poverty income ratio (PIR), our second index of SES, was not used as an initial exclusion criteria due to greater missing data for this measure (an additional n=1,413)***;*** rather, analyses based on the PIR were run on the subset of our general analysis sample who also had PIR data. Additional exclusion criteria included pregnancy (n=285) or excessive missing biological data (n=595; see below for details). The final analysis sample included a total of 15,578 NHANES participants.

Biological Indicators

Data based on nine biomarkers were used to create an overall summary index of multi-system risk, to reflect the cumulative effect of physiological dysregulation across multiple systems, or allostatic load, (AL) (22). We also created three subscales based on subsets of biomarkers reflecting inflammatory, metabolic and cardiovascular parameters. The inflammation subscale included C-reactive protein [CRP] (mg/dL) and albumin (g/dL). The metabolic subscale included glycated hemoglobin (%), total cholesterol (mg/dL), HDL cholesterol (mg/dL), and waist-to-hip ratio [WHR]. And, the cardiovascular subscale included systolic blood pressure [SBP] (mm Hg), diastolic blood pressure [DBP] (mm Hg), and resting heart rate (bt/min). Biomarkers available in the NHANES database, but excluded from the present analyses include: fibrinogen (data were only available for individuals aged 40+) and

biomarkers that required fasted samples such as triglycerides, LDL cholesterol and glucose (a smaller and less representative sample was available).

For each of the variables, a dichotomous indicator was created, reflecting those with "high risk" values (assigned a score of "1") and "lower risk" values (assigned a score of "0"). Values assigning high and low risk were based on clinically accepted "high risk" criteria (see Table 1).

Insert Table 1

These indicator variables were then summed to create summary scores for each of the subscales (inflammatory, metabolic and cardiovascular). Those missing more than half of the individual components of a particular subscale (i.e., more than one for inflammation and cardiovascular subscales and more than two for the metabolic subscale) were scored as "missing" for that subscale. In addition, individuals who self-reported recent infection (e.g., cold, cough, respiratory infection), or whose white blood cell count was greater than 10,000 per μL, were assigned a missing code for CRP and/or albumin if their respective values were in the high risk range since this could reflect acute infection rather than chronic elevation. In fact, missing data were not a major problem: 81.5 percent of the cohort had no missing data and another 15 percent were missing only one measure. For those with missing data on no more than one component of a subscale (or two in the case of the metabolic indicators), summary scores were calculated by summing available data and then scaling that score by the ratio of "number of items in the subscale" to "number of items with available data".

The summary, multi-system AL score was created by summing the subscale scores. No cross-system imputations were made for missing items; summary AL scores were created only for participants with a score for each of the three subscales. To allow for greater ease of

comparisons across indices of SES and ethnicity, summary scores were rounded to nearest integers. Rounding affected less than five percent of the scores and comparisons of results based on raw score revealed no differences. Additional algorithms for scoring risk, including use of quartile cut-points (24, 32) and using reported medication use as a criterion for "high risk", were also examined. Results were consistent with those found based on the primary scoring system outlined above using clinically defined cut-points and actual levels of biological parameters (i.e., those on medications who have "low risk" levels of a given biological parameter such as blood pressure or glucose are counted as "low risk").

Socio-economic Status

Education was measured in terms of highest level completed (grade school, some high school, complete high school, some college, complete college or more). Indicator variables were created using "completed college or more" as the reference group. Income was measured based on the poverty income ratio (PIR), an index reflecting the ratio of household income to the household poverty level determined by area of residence and household size. (33). PIR values were examined using five categories (<1, 1-1.99, 2-2.99, 3-3.99, 4-4.99 and 5+) with those reporting incomes five (or more) times the poverty ratio serving as the reference group.

Ethnicity

Blacks and Mexican Americans were over-sampled, allowing for separate analysis of these two ethnic groups, along with whites and a fourth "other" category. Hispanics other than Mexican Americans were classified as "other" in NHANES (33).

Covariates

Multivariable analyses included controls for smoking and physical activity. Smoking status was coded by two indicator variables designating current and former smokers (ever

smoked >=100 cigarettes), with never smokers serving as the reference group. Total physical activity was measured by summing weighted scores for all reported moderate and vigorous activities. Weighted scores for each activity reflect the intensity rating (i.e., metabolic equivalents [MET's]) for that activity (34) multiplied by the reported monthly frequency.

Analyses

All analyses were weighted, using the NHANES final examination weight (1), to adjust for probabilities of selection and non-response. Initial descriptive analyses compared characteristics of those included versus excluded from the present analyses. For those included in the analyses, distributions of the various components of AL were also examined by education and PIR levels.

Distributions of total AL and sub-scale scores (inflammatory, metabolic and cardiovascular) were examined within groups defined by levels of education, income, and race, after standardizing the age distribution of each comparison group to the age distribution of the US at the time of the 2000 Census.

Cumulative logistic regression models were then fit to assess relative odds for increasing AL by education and by PIR. For an ordinal variable like AL, this approach models the log of the cumulative odds (i.e., the odds of AL score k or higher against score less than k) as a linear function of predictors, fitting a single cumulative odds ratio per predictor. The cumulative OR for a predictor is the average effect of the predictor on cumulative odds at different levels k.

A series of these cumulative logistic models were fit to estimate the independent effects of education and income after: a) controlling for age and gender; b) additional controls for race (black, Mexican American and other vs. white) and c) further controls for smoking (current,

former vs. never) and physical activity. Terms testing for race-by-SES interactions were also examined.

To account for the complex survey design, we used the SVY procedures in Stata (version 9) to fit the cumulative logistic regression models and to obtain p-values for trends for the overall sample. For ethnic-stratified analyses, where data in some PSUs were sparse, we ran regression models (without the SVY command), accounting for the complex survey design by using sampling weights and robust variance estimation with clustering at the PSU level, using Stata's cluster option.

RESULTS

Table 2 provides descriptive information comparing those included in the analyses to the overall NHANES sample aged 20+ and to those excluded from the current analyses due to missing data, no MEC exam, or pregnancy. As shown, the analysis sample is representative of the overall NHANES sample with respect to age, gender, race/ethnicity, and education and PIR. The analysis sample had a median age of 42, with 48.7 percent male, 76.9 percent white, 10.4 percent black, 5 percent Mexican American and 7.7 percent "other". A majority of the participants had completed high school or more, and reported household incomes twice the poverty level or more.

INSERT TABLE 2

Examination of the individual biological measures revealed that high WHR was the most prevalent high risk factor with 63.1 percent of the cohort having WHR's above the high-risk threshold. Next most frequent high-risk values were for low HDL cholesterol (23.8 percent), high total cholesterol (19.6 percent) and high CRP (17.6 percent). The expected educational

gradient was seen for each of the individual biomarkers, with increasing prevalence of "high risk" values with lower education (with the exception of albumin and diastolic blood pressure) (see Table 3A). Parallel analyses based on the PIR revealed generally similar results, though total cholesterol no longer exhibited a gradient.

INSERT TABLE 3

Parallel education and income gradients were also found for the three subscales and the overall AL index. Due to the skew of the various biological indices (with expected lower frequencies with higher scores), we examined both the scale means as well as "percent with higher scores". Each of the subscales revealed the expected education gradients with respect to both mean scores and "percent with high scores" – those with less education having higher scores (see Table 3B). Analyses based on the PIR revealed similar, though less steep, gradients. For the overall AL index, a nearly linear gradient of increasing cumulative AL with decreasing level of education was evident (see Figure 1); a similar gradient was also seen for the PIR (See Figure 1).

INSERT FIGURE 1

Stratification by ethnicity revealed parallel education gradients for low overall AL (i.e. scores of 0 or 1; see Figure 2A) and for higher AL (i.e., scores of 2 or more; see Figure 2B) and formal tests for ethnicity-by-education interactions were non-significant. Parallel gradients were also found by PIR within each ethnic group (data not shown). Significant education and income gradients were also seen for higher risk scores on most of the subscales (see Tables 4 & 5). For whites and blacks, the strongest gradients were seen for indices of metabolic and cardiovascular risks. For Mexican Americans, the strongest gradients were seen for the inflammation subscale.

Insert Figures 2A & 2B and Tables 4 & 5

Cumulative logistic regression models including both education and PIR confirmed that education and income gradients in cumulative biological risk scores each remained significant after adjusting for the other (as well as for age and gender), and that these significant gradients were largely unchanged by further adjustments for race/ethnicity and lifestyle factors (i.e., smoking and physical activity)(see Table 6). Tests for possible education or income interactions with ethnicity were all non-significant. Main effects for ethnicity indicated significantly increased risks among blacks and marginally increased risks for Mexican-Americans as compared with whites, independent of education and income.

DISCUSSION

Analyses of NHANES III, a nationally representative sample of the US, provide evidence of consistent education and income gradients in biological risks. Indeed, gradients were seen at the level of individual biomarkers, at the level of subscales reflecting major systems of biological activity such as inflammation, metabolism and cardiovascular activity, and –not surprisingly in light of the foregoing – at the level of overall, cumulative biological risk profiles (allostatic load). This pattern of findings is consistent with the proposition that SES is an individual characteristic with broad contextual implications in terms of the conditions of one's life, including both types and frequency of "stressors" as well as resources available to deal with such stressors and that such SES-related differences in life conditions are likely to result in "wear and tear" on many (if not all) of the body's physiological regulatory systems (35).

In contrast to some previous studies (17, 26) suggesting that SES gradients in biological risk profiles were shallower in blacks, gradients based on the NHANES data were found to be similar across whites, Mexican Americans and blacks as well as an undifferentiated group of

"others". The strength of the NHANES data are their national representation and the over-sampling of blacks and Mexican Americans which allows for greater statistical power in assessments of SES gradients within these two important ethnic groups. By contrast, earlier findings from the Coronary Artery Risk Development in Young Adults (CARDIA) study (17) reflect blacks and whites aged only 18-30 at baseline and drawn from four specific cities (Oakland, CA; Chicago, IL; Birmingham, AL; and Minneapolis, MN) while the Watkins study (26), based on data from the Multiple Risk Factor Intervention Trial (MRFIT), included only black and white men at risk for cardiovascular disease. The weaker SES gradients found in these earlier studies could reflect effects of enrollment selection criteria (i.e., risk for heart disease in the case of the MRFIT study and, in the case of the CARDIA cohort, over-sampling of black men and women with high school education or more; [36]). The NHANES data provide a much broader and representative picture of the overall adult US population and the present analyses indicate that education and income gradients in biological risk are similar across major ethnic groups.

Both education and income gradients remained significant, independent of smoking and physical activity, two of the major lifestyle factors often cited as contributors to observed SES health disparities, indicating that other SES-related differentials in life experience must contribute to observed differences in cumulative biological risk profiles. The lack of significant ethnic variation in these SES gradients of biological risk indicates that, regardless of ethnic/minority status, those achieving higher education and income appear to reap benefits with respect to lower cumulative biological risk profiles – an encouraging pattern to the extent that social policies and programs can be further targeted to foster achievement of higher levels of education and income for all members of society.

Strengths of the present analyses include the nationally representative nature of the NHANES III sample and the over-sampling of blacks and Mexican Americans, which allowed for more detailed examination of possible ethnic variation in SES-related gradients in biological risks. The range of available biomarkers also allowed for examination of the consistency of SES gradients across multiple categories of known biological risk factors, including inflammation, metabolism and cardiovascular risk profiles.

Limitations that should be acknowledged include the fact that available biomarker data provide unequal assessments across different physiological systems, ranging from two indicators of inflammation to four indicators of metabolic profiles. Like many large population-based studies, the NHANES also provides biomarker data based solely on a one-shot, static assessment; no information is available on possible differences in system dynamics, nor can we assess the degree to which the available information reflects that individuals "usual" status. Our use of a scoring system based on a simple count of whether or not the individual's value for a given biomarker was in a clinically defined "higher risk" category likely provides a relatively crude index of cumulative biological risks as information on actual levels is lost, as is the potential variability in the contributions of different individual biomarkers to overall risk. Indeed, in previous work, we have demonstrated that more complex and sensitive scoring algorithms that include individual biomarker scores and that allow for unequal weighting of the different biomarkers do result in stronger relationships to health outcomes (37). Nonetheless, the available data do provide information on a range of biomarkers known to influence risks for major health outcomes and the indices created for the current analyses clearly show evidence for consistent education- and income-related gradients in cumulative risks.

The findings presented here underscore the consistent and widespread impact of SES on major physiologic systems that affect our health and functioning. Using both educational and income-related indices of SES, consistent patterns of increasing prevalence of higher biological risk at lower levels of SES were found for individual biological parameters, summary indices reflecting burdens of inflammation, metabolic and cardiovascular risk and for a summary index of overall cumulative biological risk. The fact that these same gradients were seen within all ethnic groups further highlights the degree to which SES plays a significant role in the development of differential profiles of biological risk – profiles that can be seen as warning signs for the increased risks for major disease, disability and mortality outcomes known to be associated with such biological risks.

While examination of individual biomarkers and even individual systems or processes such as metabolism provides important information on that system or biomarker's potential role as a mediator of SES differences in health, it fails to convey the full picture of biological consequences associated with lower SES. Evidence presented here provides a more complete picture, highlighting the wide range of biological consequences of lower SES and suggesting the need to consider this multiplicity of biological consequences in our efforts to both better understand how SES "gets under the skin" and to develop more effective approaches to primary (and secondary) interventions to reduce and/or prevent SES-related health disparities.

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Figure 1 – Mean Overall Biological Risk (Allostatic Load) Scores by Education and Income Groups

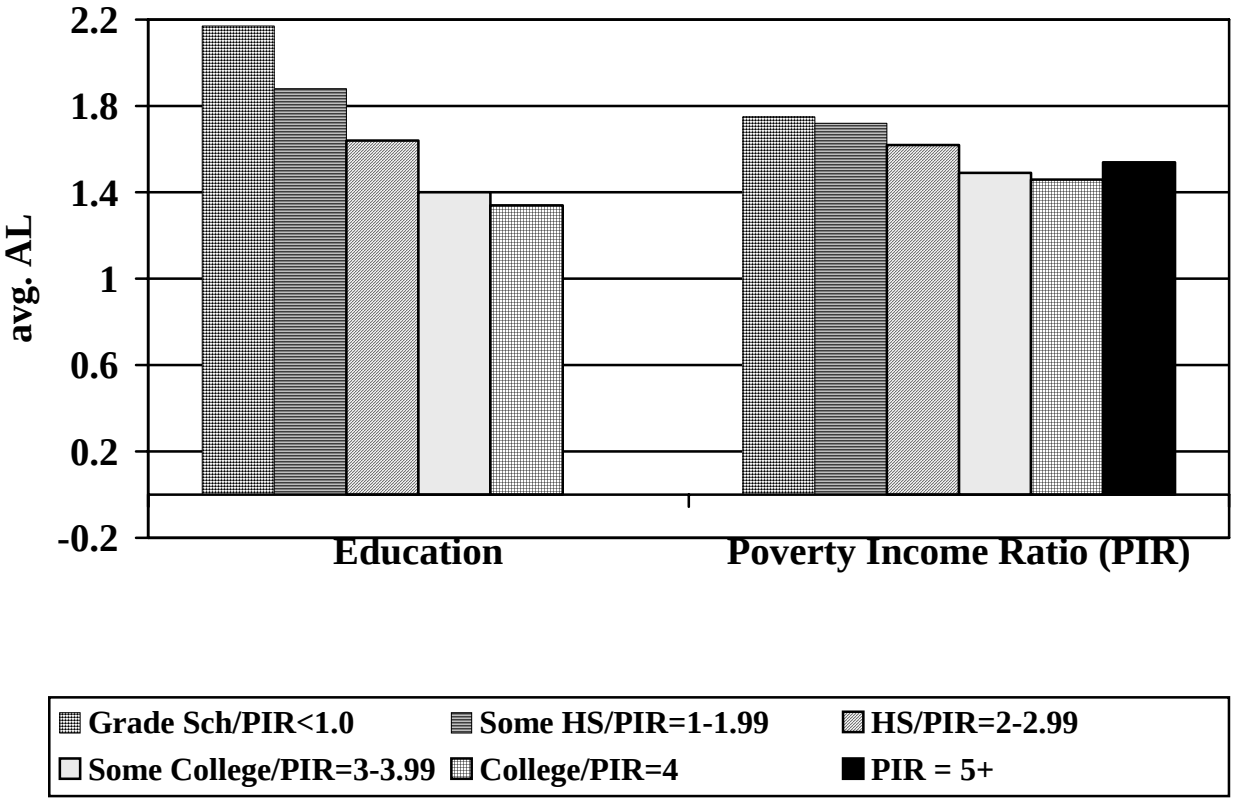

Figure 2A. Age-adjusted percentages with ***Low Overall Biological Risk (Allostatic Load)*** (scores=0-1) by Education and Ethnicity: NHANES III

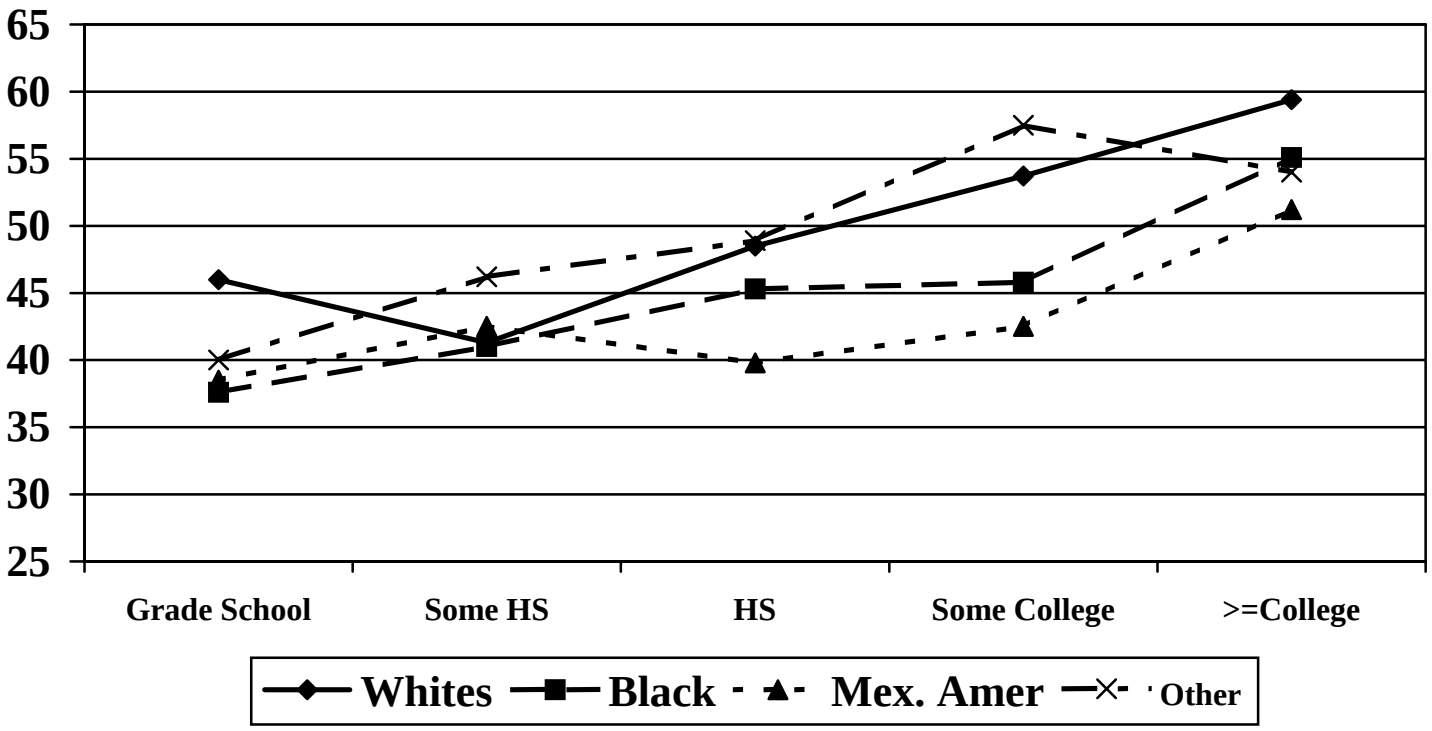

Figure 2B. Age-adjusted percentages with ***Higher Total Biological Risk (Allostatic Load)*** (scores=2+) by Education and Ethnicity: NHANES III

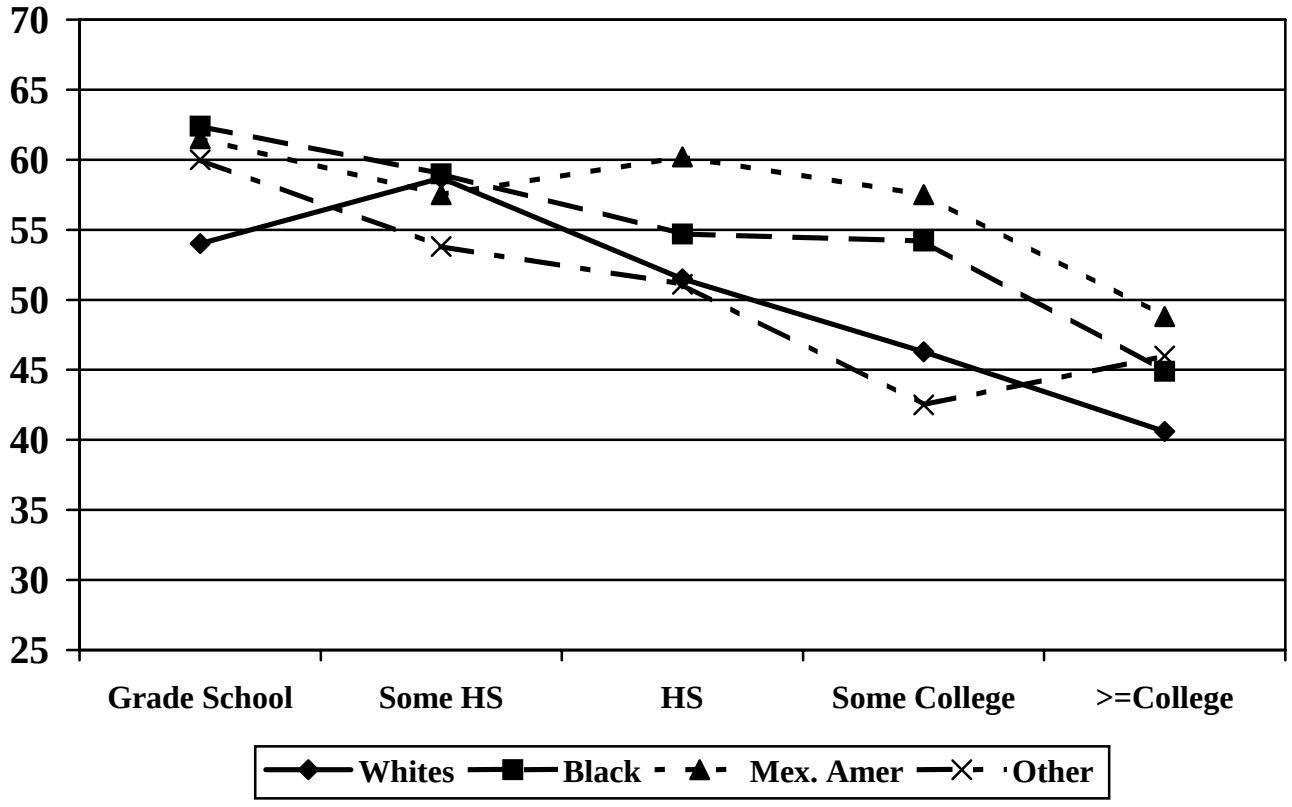

Table 1. Clinically-defined "high risk" criteria for biologic risk factors

Variable	High Risk Clinical
Albumin (g/dL)	<3.8 g/dL (ref. 38)
CRP (mg/dL)	>=0.3 (ref. 39)
Waist:Hip	>0.90 for men; >0.85 for women (ref. 40)
Total cholesterol (mg/dL)	>=240 (ref. 41)
HDL (mg/dL)	<40 (ref. 41)
Glycated Hemoglobin (%)	>=6.4 (refs. 42; 43)
Resting Heart Rate (bt/min)	>=90 (ref. 44)
Systolic BP (mm Hg)	>=140 (ref. 45)
Diastolic BP (mm Hg)	>=90 (ref. 45)

Table 2. Characteristics of the NHANES III and study samples, based on data for adults aged 20 or more years from the National Health and Nutrition Survey, 1988-1994*

A. Demographic Characteristics

Variables	*NHANES aged 20+ (n=18,825)*	*Study Sample (n=15,578)*	*Excluded Sample (n=3,247)*
	%	%	%
Age (Median)	41.0	42.0	32.0
Age Groups			
20-29	22.2	21.3	40.2
30-39	24.0	23.8	29.1
40-49	18.7	19.1	10.5
50-59	12.4	12.8	6.1
60-69	11.4	11.7	5.7
70-79	7.8	8.0	4.9
80+	3.4	3.4	3.5
Male	47.6	48.7	26.7
Race/Ethnicity			
White	76.2	76.9	63.0
Black	10.9	10.4	21.6
Mexican American	5.1	5.0	7.3
Other	7.7	7.7	8.1
Education			
Grade School	11.6 (11.6)†	11.6	10.9 (12.0) †
Some High School	13.3 (13.3)	13.4	11.4 (12.6)
Complete High School	33.5 (33.7)	33.6	32.1 (35.5)
Some College	20.6 (20.7)	20.7	19.3 (21.4)
>=Complete College	20.6 (20.7)	20.8	16.8 (18.6)
Missing	0.5		9.5
Poverty Income Ratio (median)	2.9	2.9	2.8
Poverty Income Ratio			
<1.0	11.8	11.5	17.4
1.0-1.99	19.8	19.7	22.3
2.0-2.99	19.8	20.1	13.5
3.0-3.99	16.8	17.0	13.0
4.0-4.99	9.9	10.2	5.3
>=5.0	15.4	15.4	16.1
Missing	6.5	6.2	12.4

* Weighted by NHANES MEC weight (WTPFEX6)

† Percentage excludes those missing.

Table 2(cont'd) B. Biological Indicators

Variables	*NHANES aged 20+ (n=18,825)*	*Study Sample (n=15,578)*	*Excluded Sample (n=3,247)*
	%	%	%
Allostatic Summary Score (Mean (Median)) (range: 0.-8)	1.6 (1.0)	1.6 (1.0)	0.91 (1.0)
Inflammatory Score (Mean (Median)) (range: 0 -2)	0.25 (0)	0.25 (0)	0.60 (0)
Metabolic Score (Mean (Median)) (range: 0-4)	1.1 (1.0)	1.1 (1.0)	0.97 (1.0)
Cardiovascular Score (Mean (Median)) (range: 0-3)	0.28 (0)	0.28 (0)	0.31 (0)
C-Reactive Protein			
High Risk (>=0.3 mg/dL)	17.7	17.6	21.5
Low Risk (<0.3 mg/dL)	71.3	71.6	57.0
High Risk and Infection	11.0	10.8	21.5
Missing	[4.8]‡	[2.1] ‡	[60.3] ‡
Albumin			
High Risk (<3.8 g/dL)	6.8	6.3	28.9
Low Risk (>=3.8 g/dL)	89.3	90.2	42.6
High Risk and Infection	4.0	3.5	28.6
Missing	[5.1] ‡	[2.4] ‡	[60.3] ‡
Glycosylated Hemoglobin			
High Risk (>=6.4%)	5.8	5.9	0.6
Low Risk (<6.4%)	94.2	94.1	99.4
Missing	[3.2] ‡	[0.5] ‡	[58.6] ‡
Total Cholesterol			
High Risk (>=240 mg/dL)	19.7	19.6	23.5
Low Risk (<240 mg/dL)	80.3	80.4	76.5
Missing	[4.2] ‡	[1.4] ‡	[59.8] ‡
HDL Cholesterol			
High Risk (<40 mg/dL)	23.5	23.8	6.7
Low Risk (>=40 mg/dL)	76.5	76.2	93.3
Missing	[5.0] ‡	[2.2] ‡	[60.2] ‡
Waist to-Hip Ratio			
High Risk (men >0.9, women>0.85)	62.8	63.1	56.8
Low Risk (men<=0.9,women<=0.85)	37.2	36.9	43.2
Missing	[4.1] ‡	[3.6] ‡	[13.2] ‡

‡ Number in brackets indicates percent with missing data; percentages for remaining groups total to 100%

Table 2 (cont'd) B. Biological Indicators

Variables	*NHANES aged 20+ (n=18,825)*	*Study Sample (n=15,578)*	*Excluded Sample (n=3,247)*
	%	%	%
Systolic Blood Pressure			
High Risk (>=140 mm Hg)	14.5	14.6	11.0
Low Risk (<140 mm Hg)	85.6	85.4	89.1
Missing	[0.2] ‡	[0.2] ‡	[0.4] ‡
Diastolic Blood Pressure			
High Risk (>=90 mm Hg)	6.6	6.6	6.4
Low Risk (<90 mm Hg)	93.4	93.4	93.7
Missing	[0.3] ‡	[0.3] ‡	[0.5] ‡
Pulse Rate			
High Risk (>=90 bt/min)	6.6	6.2	13.4
Low Risk (<90 bt/min)	93.5	93.8	86.6
Missing	[2.7] ‡	[2.6] ‡	[3.8] ‡

‡ Number in brackets indicates percent with missing data; percentages for remaining groups total to 100%

Table 3a. Percent with "high-risk" values for individual biomarkers by levels of education and poverty income ratio, based on data for adults aged 20 or more years from the National Health and Nutrition Survey, 1988-1994*

	CRP	Albumin	Glyco. Hemo globin.	Total Chol.	HDL Chol	WHR	SBP	DBP	Pulse
	% with "high-risk" values								
Education									
Grade School	22.3%	6.4%	10.6%	23.3%	29.7%	75.4%	16.9%	6.3%	8.6%
Some High School	24.4%	7.9%	8.5%	21.2%	27.8%	71.8%	16.9%	7.1%	9.9%
Complete High School	22.7%	7.3%	6.0%	21.5%	24.5%	65.9%	16.1%	7.0%	5.9%
Some College	20.3%	6.5%	5.6%	20.1%	21.8%	62.2%	15.7%	6.7%	5.5%
>=Complete College	14.8%	6.6%	3.6%	17.3%	20.7%	57.6%	13.7%	7.2%	3.9%
P-value for Trend	0.003	0.9	<0.0001	0.02	<0.0001	<0.0001	0.004	0.4	<0.0001
Poverty Income Ratio									
<1.0	28.1%	9.7%	10.1%	20.6%	26.4%	71.0%	19.9%	7.4%	8.4%
1.0-1.99	25.1%	7.4%	7.7%	20.2%	25.7%	66.8%	16.4%	7.3%	8.4%
2.0-2.99	19.1%	6.4%	6.4%	21.5%	25.8%	66.4%	14.8%	7.0%	6.5%
3.0-3.99	18.2%	5.4%	5.0%	19.8%	22.5%	64.6%	14.5%	6.2%	5.5%
4.0-4.99	17.9%	6.2%	4.5%	20.4%	18.8%	62.8%	15.3%	5.7%	5.0%
>=5.0	16.6%	8.1%	4.7%	18.3%	21.8%	56.7%	14.6%	6.9%	3.7%
P-value for Trend	<0.0001	0.06	<0.0001	0.3	0.003	<0.0001	<0.0001	0.3	<0.0001

* All values are weighted by NHANES MEC weight: WTPFEX6 and age standardized to the 2000 US Census population. P-values for trend were determined based on regression models that considered education and poverty as ordinal variables in the models.

Table 3b. Age-adjusted means and inter-quartile ranges and percent with high cumulative biological risk scores by levels of education and poverty income ratio, based on data for adults aged 20 or more years from the National Health and Nutrition Survey, 1988-1994*

	Total AL	Inflammation	Metabolism	Cardiovascular	% AL = 2+	% Inflammation = 1+	% Metabolism = 1+	% Cardiovascular = 1+
	Mean (25th, 75th percentiles)				**% high scores**			
Education								
Grade School	1.93 (1.0, 3.0)	0.26 (0, 0)	1.37 (1.0, 2.0)	0.32 (0, 0)	58.1	22.9	81.7	24.3
Some High School	1.89 (1.0, 3.0)	0.29 (0, 0)	1.28 (1.0, 2.0)	0.34 (0, 1.0)	57.8	24.6	76.5	27.9
Complete High School	1.72 (1.0, 3.0)	0.28 (0, 0)	1.17 (0, 2.0)	0.29 (0, 0)	52.0	24.2	73.1	24.0
Some College	1.60 (1.0, 2.0)	0.25 (0, 0)	1.09 (0, 2.0)	0.28 (0, 0)	47.2	21.8	68.9	23.1
>=Complete College	1.43 (0, 2.0)	0.21 (0, 0)	0.99 (0, 2.0)	0.25 (0, 0)	41.5	17.9	64.7	19.7
P-value for Trend	<0.0001	0.03	<0.0001	0.001	<0.0001	0.04	<0.0001	0.001
Poverty Income Ratio								
<1.0	1.95 (1.0, 3.0)	0.34 (0, 1.0)	1.27 (1.0, 2.0)	0.36 (0, 1.0)	57.6	29.0	76.6	27.9
1.0-1.99	1.79 (1.0, 3.0)	0.30 (0, 1.0)	1.19 (0, 2.0)	0.33 (0, 1.0)	53.2	25.9	73.1	26.0
2.0-2.99	1.70 (1.0, 3.0)	0.24 (0,0)	1.19 (0, 2.0)	0.28 (0,0)	51.5	20.6	72.1	23.9
3.0-3.99	1.59 (1.0, 2.0)	0.22 (0,0)	1.11 (0, 2.0)	0.27 (0,0)	47.1	19.3	71.4	21.9
4.0-4.99	1.53 (0, 2.0)	0.23 (0,0)	1.05 (0, 2.0)	0.26 (0,0)	44.9	20.7	68.2	21.5
>=5.0	1.48 (0, 2.0)	0.23 (0,0)	1.00 (0, 2.0)	0.25 (0,0)	43.0	19.3	64.9	19.9
P-value for Trend	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001

* All values are weighted by NHANES MEC weight: WTPFEX6 and age standardized to the 2000 US Census population. P-values for trend were determined based on regression models that considered education and poverty as ordinal variables in the models.

Table 4 Ethnic-specific gradients in inflammation, metabolic risk and cardiovascular subscales by education, based on data for adults aged 20 or more years from the National Health and Nutrition Survey, 1988-1994*

	Inflammation		**Metabolic Risk**		**Cardiovascular Risk**	
	0	**1+**	**0**	**1+**	**0**	**1+**
Whites – N's	4,728	1,507	2,317	4,212	4,517	2,003
	%	**%**	**%**	**%**	**%**	**%**
Grade School	80.6	19.4	24.2	75.8	77.3	22.7
Some HS	78.9	21.1	22.8	77.2	71.9	28.1
HS	77.4	22.6	26.3	73.7	77.0	23.0
Some College	79.5	20.5	31.3	68.7	77.2	22.9
>=College	83.2	16.8	36.0	64.0	80.9	19.1
P-Value	0.2		<0.0001		0.005	
Blacks- N's	2,662	1,268	2,037	2,188	2,900	1,318
	%	**%**	**%**	**%**	**%**	**%**
Grade School	66.0	34.0	24.2	75.8	60.9	39.1
Some HS	67.0	33.0	26.7	73.3	63.4	36.6
HS	65.1	34.9	33.9	66.1	66.8	33.3
Some College	67.5	32.5	32.1	67.9	72.7	27.3
>=College	66.6	33.4	37.1	62.9	71.5	28.5
P-Value	0.9		<0.0001		<0.0001	
Mexicans – N's	3,069	959	1,525	2,676	3,233	959
	%	**%**	**%**	**%**	**%**	**%**
Grade School	73.0	27.1	14.2	85.8	74.5	25.6
Some HS	74.2	25.8	18.5	81.5	77.1	22.9
HS	76.3	23.8	18.5	81.5	72.7	27.4
Some College	82.3	17.7	21.6	78.4	70.5	29.5
>=College	87.7	12.3	23.4	76.6	77.2	22.8
P-Value	0.001		<0.0001		0.08	
Others - N's	465	130	260	362	486	136
	%	**%**	**%**	**%**	**%**	**%**
Grade School	80.6	19.4	14.2	85.8	76.0	24.0
Some HS	67.2	32.8	25.4	74.6	83.3	16.7
HS	76.8	23.2	23.8	76.3	77.6	22.5
Some College	84.9	15.1	27.2	72.8	88.8	11.2
>=College	75.8	24.2	31.0	69.0	82.1	18.0
P-Value	0.9		0.006		0.1	

* All values are weighted by NHANES MEC weight: WTPFEX6 and age standardized to the 2000 US Census population. P-values for trend were determined based on regression models that considered education as an ordinal variable in the model.

Table 5 Ethnic-specific gradients in inflammation, metabolic risk and cardiovascular subscales by poverty income ratio, based on data for adults aged 20 or more years from the National Health and Nutrition Survey, 1988-1994*

	Inflammation		**Metabolic Risk**		**Cardiovascular Risk**	
	0	**1+**	**0**	**1+**	**0**	**1+**
Whites – N's	4,413	1,385	2,189	3,877	4,224	1,834
	%	%	%	%	%	%
<1.0	73.4	26.6	24.5	75.5	75.0	25.0
1.0-1.99	76.5	23.5	26.3	73.7	75.5	24.5
2.0-2.99	80.7	19.3	28.4	71.7	76.5	23.6
3.0-3.99	81.9	18.1	28.1	72.0	78.4	21.6
4.0-4.99	80.1	19.9	32.1	67.9	78.7	21.3
>=5.0	81.0	19.0	35.2	64.9	80.1	19.9
P-Value	0.02		<0.0001		0.004	
Blacks – N's	2,411	1,154	1,870	1,963	2,641	1,186
	%	%	%	%	%	%
<1.0	62.2	37.8	29.6	70.5	61.9	38.1
1.0-1.99	66.3	33.7	32.7	67.3	65.7	34.4
2.0-2.99	68.2	31.8	29.8	70.2	67.0	33.0
3.0-3.99	66.4	33.6	36.3	63.7	67.7	32.3
4.0-4.99	71.2	28.8	33.2	66.8	75.7	24.4
>=5.0	68.0	32.0	36.5	63.5	74.1	25.9
P-Value	0.03		0.02		<0.0001	
Mexican-Americans – N's	2,716	836	1,368	2,332	2,874	818
	%	%	%	%	%	%
<1.0	71.9	28.1	15.1	84.9	76.2	23.8
1.0-1.99	74.7	25.4	18.6	81.5	76.8	23.2
2.0-2.99	78.0	22.1	19.6	80.4	72.1	27.9
3.0-3.99	79.0	21.0	17.6	82.4	70.8	29.2
4.0-4.99	76.4	23.6	19.9	80.1	72.2	27.8
>=5.0	83.5	16.5	23.6	76.4	76.0	24.0
P-Value	0.02		0.005		0.1	

* All values are weighted by NHANES MEC weight: WTPFEX6 and age standardized to the 2000 US Census population. P-values for trend were determined based on regression models that considered poverty as an ordinal variable in the model.

Table 5 (cont'd) Ethnic-specific gradients in inflammation, metabolic risk and cardiovascular subscales by poverty income ratio, based on data for adults aged 20 or more years from the National Health and Nutrition Survey, 1988-1994*

	Inflammation		**Metabolic Risk**		**Cardiovascular Risk**	
	0	**1+**	**0**	**1+**	**0**	**1+**
Others – N's	423	118	242	323	441	124
	%	%	%	%	%	%
<1.0	73.2	26.8	19.2	80.8	71.9	28.2
1.0-1.99	72.4	27.6	29.4	70.6	73.7	26.4
2.0-2.99	82.0	18.0	26.0	74.0	80.1	19.9
3.0-3.99	78.9	21.1	29.1	70.9	81.1	18.9
4.0-4.99	81.2	18.8	33.8	66.3	87.3	12.7
>=5.0	89.1	10.9	49.0	51.0	97.0	3.0
P-Value	0.1		0.04		<0.0001	

* All values are weighted by NHANES MEC weight: WTPFEX6 and age standardized to the 2000 US Census population. P-values for trend were determined based on regression models that considered poverty as an ordinal variable in the model.

Table 6. Estimated relative cumulative odds of higher total allostatic load score (AL) by education and income from multi-nomial ordinal logistic regression models, based on data for adults aged 20 or more years from the National Health and Nutrition Survey, 1988-1994

	Basic Model*	***Adjusted for Ethnicity*** †	***Adjusted for Lifestyle Factors***‡
Education			
Grade School	1.8 (1.5-2.3)	1.8 (1.4-2.3)	1.6 (1.3-2.1)
Some High School	1.8 (1.4-2.2)	1.8 (1.4-2.2)	1.7 (1.3-2.0)
Complete High School	1.6 (1.4-1.9)	1.6 (1.4-1.9)	1.6 (1.3-1.8)
Some College	1.4 (1.2-1.6)	1.4 (1.2-1.6)	1.4 (1.2-1.6)
>=Complete College	Reference	Reference	Reference
P-Value for Trend	<0.0001	<0.0001	<0.0001
Poverty Income Ratio			
<1.0	1.7 (1.4-2.1)	1.6 (1.3-2.0)	1.6 (1.2-2.0)
1.0-1.99	1.3 (1.0-1.6)	1.2 (1.0-1.5)	1.2 (1.0-1.5)
2.0-2.99	1.1 (1.0-1.4)	1.1 (0.9-1.3)	1.1 (0.9-1.3)
3.0-3.99	1.0 (0.8-1.1)	1.0 (0.8-1.1)	0.9 (0.8-1.1)
4.0-4.99	1.0 (0.8-1.3)	1.0 (0.8-1.3)	1.0 (0.8-1.3)
>=5.0	Reference	Reference	Reference
P-Value for Trend	<0.0001	<0.0001	0.001
Race/Ethnicity			
Black		1.2 (1.1-1.5)	1.3 (1.1-1.5)
Mexican American		1.2 (1.0-1.4)	1.2 (1.0-1.4)
Other		1.2 (0.9-1.6)	1.2 (0.9-1.6)
White		Reference	Reference
Smoking			
Current			1.1 (0.9-1.2)
Former			1.1 (1.0-1.2)
Never			Reference
Physical Activity Score			1.0 (1.0-1.0)

* Adjusted for age (using age-standardized weights, based on the US 2000 Census Population distribution, as well as age as a categorical variable) and gender.
† Additional adjustment for race/ethnic groups (black, Mexican American, other vs. white)
‡Additional adjustment for smoking (now, former vs. never) and physical activity (overall moderate and vigorous activity score).