Impact of socioeconomic status on longitudinal accumulation of cardiovascular risk in young adults: the CARDIA Study (USA)

Arun S. Karlamangla[a],*, Burton H. Singer[b], David R. Williams[c], Joseph E. Schwartz[d], Karen A. Matthews[e], Catarina I. Kiefe[f], Teresa E. Seeman[a]

[a]Division of Geriatrics, UCLA School of Medicine, 10945 Le Conte #2339, Los Angeles, CA 90095-1687, USA
[b]Office of Population Research, Princeton University, 21 Prospect Avenue, Princeton, NJ 08544-2091, USA
[c]Institute for Social Research, University of Michigan, P.O. Box 1248, Ann Arbor, MI 48106-1248, USA
[d]Department of Psychiatry, SUNY Stony Brook, 153 Putnam Hall, Stony Brook, NY 11794-8790, USA
[e]Department of Psychiatry, University of Pittsburgh, 3811 O'Hara Street, Pittsburgh, PA 15213, USA
[f]Division of Preventive Medicine, Department of Medicine, University of Alabama at Birmingham, and Birmingham Veterans Affairs Medical Center, Birmingham, AL, USA

Abstract

Our objectives were to describe the trajectories of biological risk factors of cardiovascular disease in young adults, and to study the association of socioeconomic status (SES) with aggregate risk scores that summarize longitudinal risk accumulation from multiple risk factors. We used data from a prospective, bi-racial, cohort study of 18–30-year-old adults in the USA, initiated in 1985, with 10-year follow-up. SES was measured by parental education level, financial hardship during the study, and the participant's education level by the end of the study. We studied growth patterns of seven biological risk factors for cardiovascular disease using a semi-parametric, class-mixture model to identify clusters of individuals with distinct growth trajectories. Risk scores that summarize risk from all seven risk factors were created to reflect risk at baseline, longitudinal risk change over 10 years, and total accumulated risk. Multivariable regression was used to study their associations with SES within each race/gender group. We found tracking of all seven risk factors: in each case, the cluster with the highest baseline value maintained its position as the highest-risk cluster over the next 10 years. After adjustment for age, lifestyle, and healthcare access, SES was associated inversely with baseline risk score in women (black and white), with risk change score in all four race/gender groups, and with accumulated risk score in women (black and white) and in white men. Our findings suggest that individuals with high overall cardiovascular risk in midlife can be identified by their relatively higher values of risk factors in younger ages and that socioeconomic differences in cardiovascular risk start accumulating early in life.
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Keywords: Socioeconomic status; Cardiovascular risk factors; Longitudinal risk accumulation; Growth trajectories; Tracking; USA

*Corresponding author. Tel.: +1-310-825-8253; fax: +1-310-794-2199.
E-mail address: akarlamangla@mednet.ucla.edu (A.S. Karlamangla).

Introduction

Low socioeconomic status (SES) is associated with increased risk of cardiovascular disease in both men and

women, and in different ethnic groups (Adler et al., 1994; Wing, Barnett, Casper, & Tyroler, 1992; James, 1984). Risk factors for cardiovascular disease are also more prevalent in lower SES groups, both in the USA (Williams & Collins, 1995) and Europe (Manhem, Dotevall, Wilhelmsen, & Rosengren, 2000; Bobak, Hertzman, Skodova, & Marmot, 1999) and both in men and women (Matthews, Kelsey, Meilahn, Kuller, & Wing, 1989). SES-driven differences may, in fact, begin to accumulate early in life: Childhood social position is associated with adult levels of high density lipoprotein (HDL) cholesterol (Brunner, Shipley, Blane, Smith, & Marmot, 1999) and fibrinogen (Brunner et al., 1996). Also, in young adults, education level is inversely associated with 5-year weight gain (Burke et al., 1996) and 10-year incidence of high blood pressure (Dyer et al., 1999), while financial hardship is associated with 10-year incidence of hypertension (Matthews et al., 2002).

While these data point to SES effects on individual risk factors, there has been little documentation of the impact of SES on longitudinal accumulation of overall risk from multiple risk factors. Moreover, some studies have suggested that one's relative position in risk-factor distributions is determined in childhood (Clarke, Schrot, Leaverton, Connor, & Lauer, 1978; Sanchez-Bayle, Munoz-Fernandez, & Gonzalez-Requejo, 1999). Thus, it is not clear if changes in SES in young adulthood influence risk. Accordingly, our objective was to study the associations of SES and changes in SES with summary scores that reflect risk accumulation over 10 years from increases in multiple risk factors in 18–30-year-old adults. We hypothesized that some young adults who started the study with elevated risk factors would continue to have high values and show increases, and that low SES at baseline and declining SES over the study period would predict risk accumulation.

Methods

Study sample

Data came from the Coronary Artery Risk Development in Young Adults (CARDIA) Study, a prospective, bi-racial, cohort study, initiated in 1985 (Friedman et al., 1988). Briefly, 5115 men and women, ages 18–30 years, were recruited by telephone from two racial groups (non-Hispanic black and white) living in four urban areas in the USA. Sampling was stratified to achieve nearly equal numbers of blacks vs. whites, men vs. women, persons of age $\leqslant 24$ vs. >24 years, and persons with education high school or less vs. more than high school. The baseline examination was conducted in 1985 and included standardized questionnaires, blood pressure and anthropometric measurements, and a

fasting blood draw. Repeat examinations were conducted in 1987, 1990, 1992, and 1995. Overall retention by 1995 was 70% for black men, 81% for white men, 76% for black women, and 82% for white women. In addition, biological measurements were deleted for women who reported being pregnant at the time of the examination, because of the potential impact of pregnancy on these measurements; 78 black women and 101 white women reported being pregnant at least once. Our study sample consisted of the 4149 CARDIA participants who had baseline measurements of education level, financial hardship, and all seven biological risk factors (listed below), *and* had each of the seven risk factors measured at least once more in follow-up.

Measurements

Cardiovascular risk factors

We measured systolic and diastolic blood pressure, fasting glucose, fasting insulin, waist–hip circumference ratio, low density lipoprotein (LDL) cholesterol, and total-to-HDL cholesterol ratio, using standardized methods (Friedman et al., 1988). Glucose and insulin measurements were repeated only in 1992 and 1995; all other risk factors were measured at every visit.

Primary predictors

The SES indicators used as primary predictors were parental education, personal educational attainment, and financial hardship. In this young cohort, occupation category and income level are not as yet, reliable indicators of SES (Matthews et al., 2002).

At the baseline interview, the education level (highest grade/year of regular schooling completed; range 1–20+) of the participant's father (or 'man responsible for you as a child') and mother (or 'woman responsible for you as a child') were obtained, and the higher of the two was reported as the parental education level. Because there are definite socioeconomic benefits associated with specific credentials (such as graduation from high school and college), we categorized education level into four groups: less than high school education (<12 years of regular schooling), high school graduation but no college (12 years of schooling), 1–3 years of college (13–15 years of schooling), and college graduation ($\geqslant 16$ years of schooling). The participants' own educational attainment was obtained at baseline and at every follow-up interview. Because many participants were still in the process of completing their education, analyses with the participant's own education were confined to the older stratum in the study sample; i.e., those individuals who were 25 years of age or older at baseline, and thus likely to have completed their education when the study began. Only 6% of participants in this group had not completed

high school in 1985 and there was no significant difference in summary risk scores between those who had not completed high school and those who had completed only high school. Therefore, we combined the two groups into one: high school or less ($\leqslant 12$ years of regular schooling). In the older stratum, we also studied the effects of further adult education, beyond the level achieved in 1985.

Financial hardship at baseline (1985) and at three of the four follow-up exams was assessed by a multiple-choice question about difficulty paying for basics (such as food, medical care, and heating), with response choices: (1) very hard, (2) hard, (3) somewhat hard, and (4) not very hard. Only 3% selected choice 1, with no significant differences in summary cardiovascular risk scores between them and those who selected choices 2 or 3. We, therefore, created a dichotomous variable as follows: somewhat hard or harder (choices 1, 2, or 3) vs. not very hard (choice 4). This information was collected again at three of the four follow-up interviews. To study the effects of ongoing hardship over time, we created the following three-category indicator of ongoing financial hardship: no hardship in any follow-up visit, hardship in at least one follow-up visit, and hardship reported in every follow-up visit.

Covariates

Age, gender, race, lifestyle habits (smoking/alcohol/exercise), and access to medical care are potential confounders and/or plausible mediators of an SES-health link, and were assessed by participant self-reports on interviewer-administered questionnaires. Access to medical care was assessed for the first time at the 4th examination (1992), but the other variables were assessed at baseline (1985). Age was recorded in years (integer number). Because the association between alcohol consumption and cardiovascular risk is generally non-linear or J-shaped (Klatsky, 1995), we created a three-category variable for alcohol consumption: abstainers, moderate drinkers (those who consumed between 0 and 20 ml of absolute alcohol per day) and heavy drinkers (more than 20 ml). A total exercise score was created as the weighted sum of the number of months of frequent or infrequent performance of 13 activities (Jacobs, Hahn, Haskell, Pirie, & Sydney, 1989). Only 30 participants (0.6%) had a score of 0; we therefore stratified the cohort by tertiles of the total exercise score: mild, moderate, and heavy exercisers. Access to medical care was ascertained from participants' answers to questions about having a usual source of medical care, having health insurance over the past 2 years, and whether there had been any time over the past 2 years when he/she did not get medical care because it was too expensive. Answers to the questions were coded as binary (0/1) variables.

Analysis

The baseline (1985) distributions of the seven cardiovascular risk factors were examined. Because their distributions were substantially skewed, baseline and follow-up measurements of fasting glucose and fasting insulin were natural-log transformed, and of total-to-HDL cholesterol ratio were square-root transformed, to make them approach the normal distribution.

Trajectory clustering

We divided the study sample into sub-groups (clusters) based on 10-year trajectories, separately for each of the seven risk factors, using a semi-parametric, latent class-mixture model (Nagin, 1999). This model fits a mixture of sub-groups (clusters) of relatively homogeneous trajectories to the study sample, with each cluster modeled by one trajectory from a parametric family. The use of a class-mixture model does not rest on the belief that the population is composed of distinct groups; instead, the clusters represent an approximation of an underlying continuum (Nagin & Tremblay, 2001). The approximation of a continuous distribution by a discrete mixture is standard practice in non-parametric and semi-parametric statistics (Heckman & Singer, 1984; Follman & Lambert, 1989; Lindsay, 1995). Our purpose was to identify groups of individuals with distinctly different trajectories, and not to suggest that the population is composed of distinct clusters.

We used available software that fits a user-specified number of clusters to the study sample, and computes the maximum likelihood estimates of the modeled trajectory parameters for each cluster (Jones, Nagin, & Roeder, 2001). Since this clustering is influenced substantially by the baseline value of the risk factor, and we were interested in *changes* in the risk factor over time, we ran the trajectory clustering analyses separately within strata defined by the baseline value of the risk factor: stratum 1, bottom quartile; stratum 2, middle two quartiles; and stratum 3, top quartile of the risk factor at baseline. In each of the three strata, we attempted to fit a three-cluster model to the transformed data, expecting to see one group showing substantial positive change over the study period, one group that stayed relatively flat, and one group showing negative change. In this cohort of young adults, in whom risk factors are still fairly low, small changes in risk-factor values are less likely to significantly change overall risk; hence, we sought to identify individuals at the extremes of change. Clustering of trajectories into three groups allowed us to do just that. Except for trajectories of fasting blood glucose, three-cluster models fit the data better than two-cluster models: The Bayesian information criterion (BIC), a goodness-of-fit measure (that rewards parsimony and penalizes models with more clusters) was better (higher) for the three-cluster model

compared to the two-cluster model. For instance, in stratum 1 (the bottom quartile at baseline), where we might not expect enough participants with substantial decline in the risk factor, the Bayes factor ($e^{\text{BIC difference}}$) was 10^{15} or higher in favor of the three-cluster model. In the case of fasting blood glucose, in each stratum, two-cluster models fit the data better than three-cluster models (Bayes factors 10^5 or better in favor of two-cluster models); however, in stratum 1, one of the two clusters had only seven people in it and we instead used a single-cluster model.

Since this is a relatively young cohort, we hypothesized that first-order (linear) growth trajectories would adequately model growth of risk factors. We fit only linear growth terms to all clusters except the fastest growing cluster in stratum 3 (the top quartile at baseline), where we initially included a quadratic term for all risk factors other than fasting glucose and insulin (since these were measured at only three time points). In every case, the quadratic term was either non-significant ($p > 0.4$) or did not substantially affect the BIC; hence, we restricted the models to linear growth for all clusters.

Creating risk summaries

A *Baseline Risk* score (range 0–7) was created for each participant as the simple count of the number of risk factors in the highest quartile at baseline. For each of the seven risk factors, we assigned a trajectory score of 1 to those in clusters with substantial positive growth, -1 to those in clusters with negative growth, if they started from the highest stratum (top quartile at baseline), and 0 to everyone else. This scoring system penalizes growth in the risk factor regardless of starting value, while rewarding declines in the risk factor only if the baseline value was high. Since risk-factor values were fairly low at baseline in the bottom two strata (for example systolic blood pressure 118 or below, fasting blood glucose 87 or below), a decline in risk factor was considered an improvement only for those in the top stratum. The *Risk Change* score (range -7 to $+7$) was obtained by summing trajectory scores over all seven risk factors. An *Accumulated Risk* score was created to capture duration of exposure to high risk in the following fashion. For each risk factor, participants were assigned 1 point for having a high value of the risk factor for only part of the study period (either by starting in the top quartile at baseline and having negative growth or by starting in a lower quartile and having substantial positive growth), 2 points for having a persistently high value of the risk factor throughout the study period, but without substantial growth, and 3 points for starting from the top quartile of the risk factor and also continuing to grow. This scoring strategy penalizes persistence at a high level of the risk factor as well as growth in the risk factor. The *Accumulated Risk* score (range 0–21) was created by summing the points accumulated for all seven risk factors. It can be shown that the three summary risk scores are related to each other as

$$\text{Accumulated Risk score} = 2 \times \text{Baseline Risk score} + \text{Risk Change score}.$$

Socioeconomic status-risk associations

The distributions of the summary risk scores were examined by categories of SES. To test whether some of the differences by SES in summary risk scores may reflect SES differences in lifestyle, we fitted multivariable linear regression models using SES indicators as primary predictors, along with the following covariates: age (in years and as two categories: $\leqslant 24$ and > 24 years), smoking status (never/past/current), number of cigarettes smoked per day by current smokers (square-root transformed to reduce skew, and set to 0 for never-smokers and past smokers), alcohol consumption categories (none/moderate/heavy), physical exercise level (total exercise score and categories: mild/moderate/heavy), and access to medical care (3 yes/no variables). Since the SES indicators used in this study may reflect social standing and economic pressures differently in different race/gender groups, and because the CARDIA sampling was stratified by race and gender, these regression analyses were run separately in the four race/gender groups: black men, white men, black women, and white women. SAS version 8, SAS Institute Inc., Cary, NC, was used for all analyses.

Results

Descriptive statistics

A comparison of the study sample with the rest of the CARDIA cohort (Table 1) revealed no gender differences in study retention, but more blacks than whites were lost to follow-up ($p < 0.0001$). Participants lost to follow-up also had lower SES than those who were retained in the study, with respect to every indicator of SES: parental education ($p = 0.0002$), financial hardship at baseline ($p = 0.001$), and participant education at baseline in the older (25 years of age or older) stratum ($p < 0.0001$). There were no significant differences in cardiovascular risk factors at baseline between the study sample and the rest of the cohort, except for fasting glucose and fasting insulin, which were slightly higher in those who were lost to follow-up.

While 49% of black men and 50% of black women in the study sample were 25 years of age or older, 63% of white men and 64% of white women were in the older stratum. There were also significant differences by race and gender groups in SES, cardiovascular risk factors, and other covariates (Tables 2a and b). Consistent with

Table 1
Descriptive statistics at baseline

Characteristics	Study sample[a] ($N = 4149$)	Full cohort ($N = 5113$)
	Percentage	
Gender—women	54	54
Race—white[b]	51	48
Parent education level[b]		
Less than high school (<12 years)	11	11
1–3 years of college (>12 but <16 years)	16	17
College graduate ($\geqslant 16$ years)	36	35
Financial hardship at baseline[b]	34	35
Participant education level at baseline in older stratum[b,c]		
1–3 years of college (>12 but <16 years)	28	29
College graduate ($\geqslant 16$ years)	41	39
Cardiovascular risk factors at baseline	Median [Q1, Q3[d]]	
Systolic blood pressure (mm Hg)	110 [103, 118]	110 [103, 118]
Diastolic blood pressure (mm Hg)	69 [62, 75]	68 [63, 75]
Fasting blood glucose (mg/dL)[b]	81 [77, 87]	81 [77, 87]
Fasting insulin (μU/mL)[b]	8.7 [6.0, 12.9]	8.7 [6.1, 13.0]
Waist–hip ratio	0.78 [0.72, 0.83]	0.78 [0.72, 0.83]
LDL cholesterol (mg/dL)	106 [87, 128]	106 [87, 127]
Total-to-HDL cholesterol ratio	3.3 [2.7, 4.1]	3.3 [2.7, 4.1]

[a]The study sample is composed of those individuals in the CARDIA cohort who had all seven cardiovascular risk factors, education level, and financial hardship assessed at baseline, and had each of the seven risk factors measured at least once more in follow-up.

[b]Two-sided p-value for test of difference (χ^2 test for categorical variables, t-test for continuous variables) between study sample and rest of cohort <0.05.

[c]Participants 25 years of age or older at baseline ($N = 2350$ in the study sample and 2832 in the full cohort).

[d]Q1 and Q3 refer to the 1st and 3rd quartile cut-off points; i.e., the 25th and 75th percentile values, respectively.

known racial differences in SES in urban communities in the USA, black men and women reported lower levels of SES with respect to every indicator, compared to white men and women. However, more blacks than whites in the older stratum, reported getting additional education over the study period. Black men and white men had higher baseline levels for six of the seven cardiovascular risk factors, compared to black women and white women, respectively. However, black women had the highest baseline values of fasting blood insulin. There were also significant difference by race and gender in lifestyle factors (Table 2b).

Clustering by risk-factor trajectories

Except in the case of fasting glucose trajectories, clustering split the study sample into nine sub-groups (Figs. 1a–g). For every risk factor, the cluster with the highest risk; i.e., the cluster from the top-baseline-quartile stratum with the most positive growth (Cluster 3.3 in Fig. 1) was one of the smallest clusters, with size ranging from 0.7% to 4.1%.

To assess model fit, within each cluster we examined the sample mean (and 95% confidence interval) of the risk factor at every visit (shown for Cluster 3.3 in Fig. 1). As can be seen, except in the case of fasting insulin, the trajectory predicted by the model for Cluster 3.3 closely approximates the trajectory of the cluster mean. Sample means and 95% confidence intervals for the other clusters are not shown in Fig. 1 because they were indistinguishable from the trajectory predicted by the model.

The trajectories of the various clusters from the three strata crossed each other over the study period; however, for each of the seven risk factors, Cluster 3.3 had the highest baseline value of all clusters and did not cross the trajectories of other clusters, indicating that its ranking as the highest-risk cluster was maintained throughout the 10-year period (Fig. 1).

Summary risk scores

The median baseline risk score in the study sample was 1 (25th percentile = 0, 75th percentile = 3); thus at baseline, one half of the sample had a high value of at

Table 2

(a) Descriptive statistics by race and gender for primary predictors and baseline values of cardiovascular risk factors

	Black men (N = 885)	White men (N = 1006)	Black women (N = 1151)	White women (N = 1107)
Primary predictors	Percentage			
Parent education[a]				
Less than high school	15	4	18	6
1–3 years of college	14	15	19	16
College graduate	19	51	20	50
Financial hardship at baseline[a]	40	27	37	32
Ongoing financial hardship[a]				
In some but not all follow-up visits	41	34	42	35
In every follow-up visit attended	13	8	18	12
Education in older stratum				
Participant education at baseline[a]				
1–3 years of college	31	24	38	23
College graduate	21	55	20	55
Additional education by last visit[a]				
Some college (1–3 years)	13	6	15	6
College graduation	6	6	9	9
Cardiovascular risk factors in 1985	Median [Q1, Q3[b]]			
Systolic blood pressure (mm Hg)[a]	116 [109, 122]	114 [107, 121]	107 [102, 114]	104 [99, 110]
Diastolic blood pressure (mm Hg)[a]	71 [64, 78]	71 [65, 77]	67 [61, 73]	66 [61, 71]
Fasting blood glucose (mg/dL)[a]	83 [78, 88]	84 [80, 89]	79 [74, 84]	80 [76, 84]
Fasting insulin (μU/mL)[a]	8.7 [5.8, 13.2]	7.8 [5.6, 11.0]	10.8 [7.4, 16.0]	7.6 [5.7, 10.9]
Waist–hip ratio[a]	0.81 [0.79, 0.84]	0.84 [0.81, 0.87]	0.73 [0.70, 0.77]	0.72 [0.69, 0.75]
LDL cholesterol (mg/dL)[a]	108 [86, 129]	110 [90, 130]	107 [89, 130]	102 [85, 122]
Total-to-HDL cholesterol ratio[a]	3.3 [2.7, 4.1]	3.7 [3.1, 4.7]	3.2 [2.7, 3.8]	3.1 [2.6, 3.7]

(b) Descriptive statistics by race and gender for covariates

Continuous covariates (Units)	Median [Q1, Q3[b]]			
Age (years)[a]	24 [21, 28]	26 [23, 28]	24 [21, 28]	26 [23, 28]
Physical exercise score[a]	462 [264, 708]	469 [295, 672]	228 [103, 400]	365 [211, 550]
Cigarettes/day by current smokers	10 [5, 15]	20 [10, 20]	10 [5, 15]	15 [5, 20]
Categorical covariates	Percentage			
Smoking status[a]				
Past smokers	9	15	8	20
Current smokers	35	25	29	25
Alcohol consumption[a]				
Moderate alcohol (⩽20 ml/day)	42	44	37	49
Heavy alcohol (⩾20 ml/day)	25	32	5	13
Physical exercise level[a]				
Moderate exercise	31	35	31	38
Heavy exercise	45	46	15	31
Access to medical care				
Had usual source of care[a]	82	80	91	91
Had health insurance for 2 years[a]	73	80	83	86
Did not get care—too expensive[c]	9	11	11	15

[a] Two-sided *p*-value for test of difference (χ^2 test for categorical variables, *F*-test for continuous variables) across four groups <0.0001.

[b] Q1 and Q3 refer to the 1st and 3rd quartile cut-off points; i.e., the 25th and 75th percentile values, respectively.

[c] Two-sided *p*-value for test of difference (χ^2 test for categorical variables, *F*-test for continuous variables) across four groups = 0.0006.

least one risk factor, and a quarter of the sample was in the high group for three or more risk factors. The median risk change score was 0 (25th percentile = −1, 75th percentile = +1); thus, a quarter of the sample showed substantial positive growth in one or more factors, and a quarter of the sample showed decline from a high baseline in one or more risk factors. The median accumulated risk score was 3 (25th percentile = 1, 75th percentile = 5; thus, three quarters of the sample had at least one risk factor elevated for at least part of the study period. We examined the distribution of the summary risk scores by gender and by race, and found that men had higher scores than women: medians of 2, 0, and 4, respectively, for baseline risk, risk change, and accumulated risk, respectively, in both black men and white men, compared to 1, 0, and 2, respectively, in black women and 1, 0, and 1, respectively, in white women.

Association of socioeconomic status with baseline risk score

There was a statistically significant trend for decreasing baseline risk score with increasing parental education, in three of the four race/gender groups; in the 4th group (black men), the trend was only marginally significant (Fig. 2a). White women who had no financial hardship had lower baseline risk; the difference was not statistically significant in the other three groups (Fig. 2b). In those who were 25 years of age or older at baseline (the older stratum), there was an inverse and significant association between baseline education and baseline risk only in white women (p < 0.0001), and not in the other three race/gender groups (Fig. 2c).

After adjustment for age and lifestyle habits as described in Methods, women with less educated parents had higher baseline risk than women whose parents had higher education (p = .04 in black women and p = .002 in white women): High school education in the parent was associated with a reduction in the baseline risk score by 0.34 in white and black women, and college graduation by a parent was associated with a further reduction in the score by 0.23 (standard error = 0.09) in white women. In white women, financial hardship at baseline was also associated with a 0.18 increase in baseline risk score (p = .03), and older white women who were themselves college graduates at baseline had lower baseline risk scores than those who had high school or less education (difference in score = −0.52, p < 0.0001). The associations between baseline risk and baseline SES that were seen in unadjusted analyses in other groups did not persist after adjustment.

Association of socioeconomic status with risk change score

In white men and in black and white women, but not in black men, lower parental education and the presence of financial hardship were both associated with higher risk change score, adjusted for baseline risk score (Table 3). In older women, there was an inverse and significant association between the participant's own baseline education and risk change score. There was a similar marginally significant association in white men but not in black men (Table 3).

Socioeconomic status change and risk change score

After adjusting for baseline risk score and baseline financial hardship, ongoing hardship was associated with higher risk change scores in black women. A similar trend, that was only marginally significant, was seen in white women. No such associations were seen in men (Table 3). There were no significant associations between risk change score and additional education over the study period in the older stratum, adjusted for baseline education level and baseline risk score, in any of the four race/gender groups (data not shown).

Adjustment for age, lifestyle habits, and access to medical care weakened some SES effects but did not eliminate them (Table 4). After adjustment, beneficial associations with parental education were seen only in white men (p = .05) and white women (p = .004); financial hardship at baseline was associated positively with longitudinal risk accumulation in all four race/gender groups (though this effect was only marginally significant in white men), and the benefits of the participant's own baseline education among the older stratum was confined mainly to women, with a marginally significant association in white men and no association in black men. The adjusted association with ongoing financial hardship however, was only marginally significant in black men (Table 4).
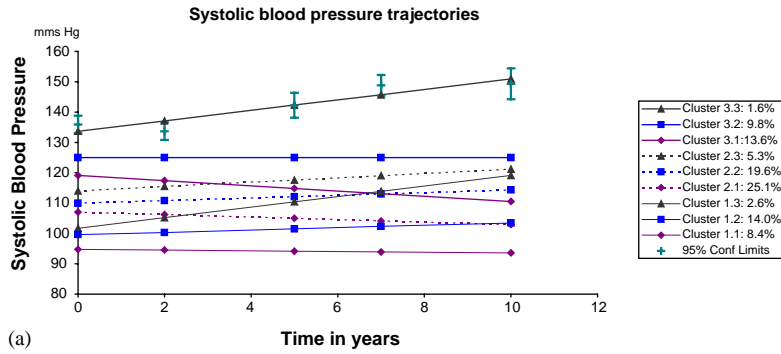
Association of socioeconomic status with accumulated risk score

The unadjusted associations of childhood and baseline SES (in Fig. 3) with accumulated risk score were similar in pattern to the associations with baseline risk score (in Fig. 2); except that here all associations are statistically significant in both black women and white women. As before, the only SES association in men was with parental education, and it was only marginally significant in black men (Fig. 3a). In women, the associations of accumulated risk score with markers of childhood and baseline SES persisted even after adjusting for age, lifestyle variables, and access to medical care (Table 5). In white men, the association with parental education seen in unadjusted analyses was seen again after adjustment; however in black men, there were no accumulated risk score association with markers of either education or financial hardship.
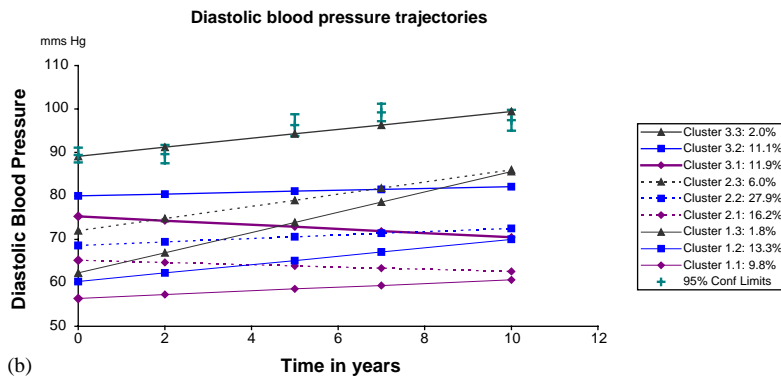
Socioeconomic status change and accumulated risk score
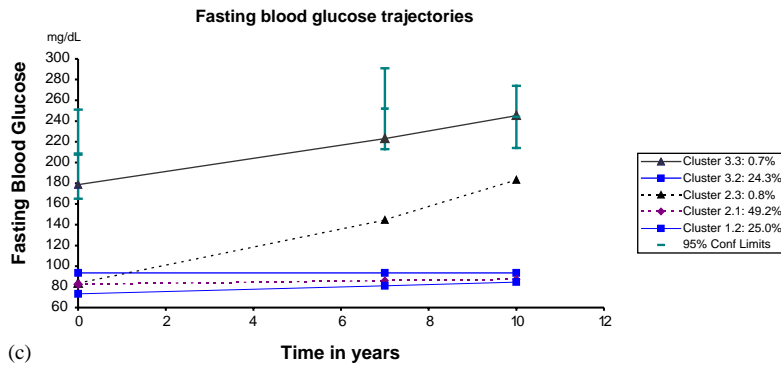 Ongoing financial hardship was associated with high-er accumulated risk, *independent of baseline hardship*,

only in white women: Compared to those who reported no financial hardship in any follow-up visit, white women who reported financial hardship in every
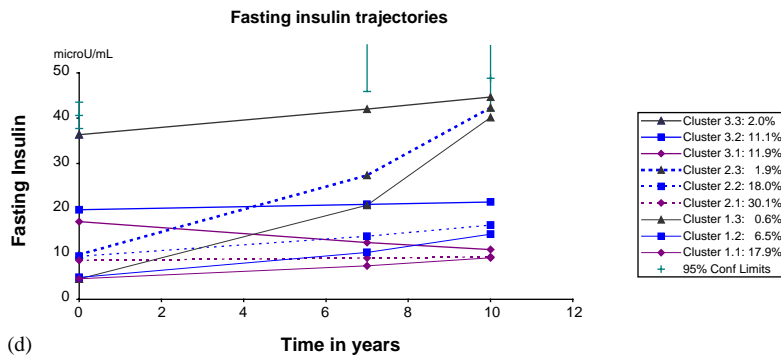

(a) Systolic blood pressure trajectories

(b) Diastolic blood pressure trajectories

(c) Fasting blood glucose trajectories

(d) Fasting insulin trajectories

LDL Cholesterol trajectories

Legend:
- Cluster 3.3: 3.6%
- Cluster 3.2: 14.5%
- Cluster 3.1: 7.0%
- Cluster 2.3: 7.7%
- Cluster 2.2: 25.2%
- Cluster 2.1: 17.1%
- Cluster 1.3: 5.1%
- Cluster 1.2: 15.9%
- Cluster 1.1: 3.9%
- 95% Conf Limits

Total-to-HDL Cholesterol ratio trajectories

Legend:
- Cluster 3.3: 4.1%
- Cluster 3.2: 13.1%
- Cluster 3.1: 7.8%
- Cluster 2.3: 6.0%
- Cluster 2.2: 22.8%
- Cluster 2.1: 21.2%
- Cluster 1.3: 3.1%
- Cluster 1.2: 12.6%
- Cluster 1.1: 9.3%
- 95% Conf Limits

Waist-to-Hip circumference ratio trajectories

Legend:
- Cluster 3.3: 3.5%
- Cluster 3.2: 13.4%
- Cluster 3.1: 8.1%
- Cluster 2.3: 13.5%
- Cluster 2.2: 21.8%
- Cluster 2.1: 14.6%
- Cluster 1.3: 4.0%
- Cluster 1.2: 13.8%
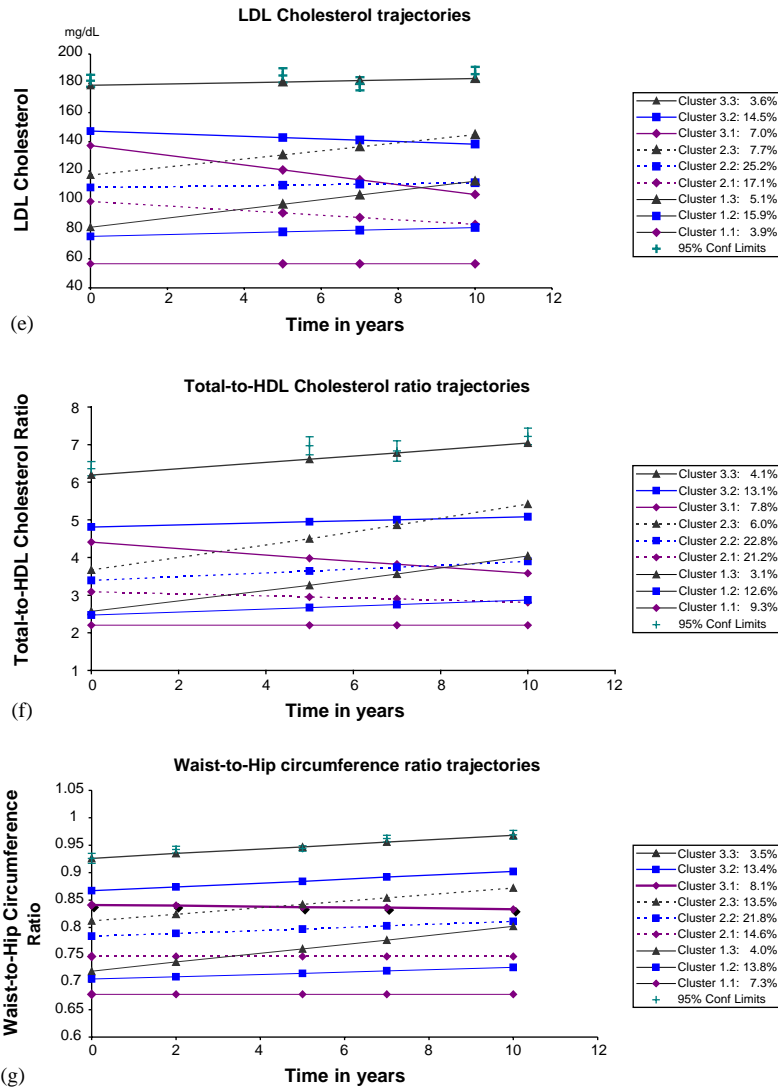- Cluster 1.1: 7.3%
- 95% Conf Limits

Fig. 1. (*Continued*)

follow-up visit had accumulated risk scores that were higher by 0.70 (standard error 0.23, $p = 0.002$). After adjusting for age, lifestyle habits, and access to medical care, the effect size fell to 0.56 ($p = 0.03$). Continuing adult education over the study period was associated with accumulated risk score in the older stratum,

Fig. 1. Growth trajectories for seven cardiovascular risk factors, after clustering within strata defined by the baseline value of the risk factor. Model trajectories for the clusters for stratum 1 (bottom quartile at baseline) are shown with thin solid lines, the trajectories for the clusters from stratum 2 are shown with broken lines, and the trajectories for the clusters from stratum 3 (top quartile at baseline) are shown with thick solid lines. Clusters are numbered $x.y$, where x refers to the stratum number and y refers to the cluster number within the stratum: $y = 3$ for the cluster with the largest slope, $y = 2$ for the cluster with intermediate slope, and $y = 1$ for the cluster with smallest slope. The percentage of the study sample assigned to each cluster is indicated in the legend accompanying each panel. Error bars on the model trajectory for the Cluster 3.3 represent the 95% confidence interval for the sample mean in Cluster 3.3 at each visit. The upper confidence limit is outside the scale of panel d: 94.4 in Year 7 and 56.2 in Year 10. Sample means and 95% confidence intervals for the other clusters are not shown because they are indistinguishable from the trajectory predicted by the model. Cluster membership was used to create the *risk change score*. For each risk factor, clusters 1.3, 2.3, and 3.3 were assigned trajectory scores of $+1$; Cluster 3.1 was assigned trajectory score of -1; and all other clusters scored 0. The *risk change score* equals the sum of trajectory scores over all seven risk factors.

(a)   **Means of Baseline Risk Score by Parental Education Level**

(b)   **Means of Baseline Risk Score by Baseline Financial Hardship Category**

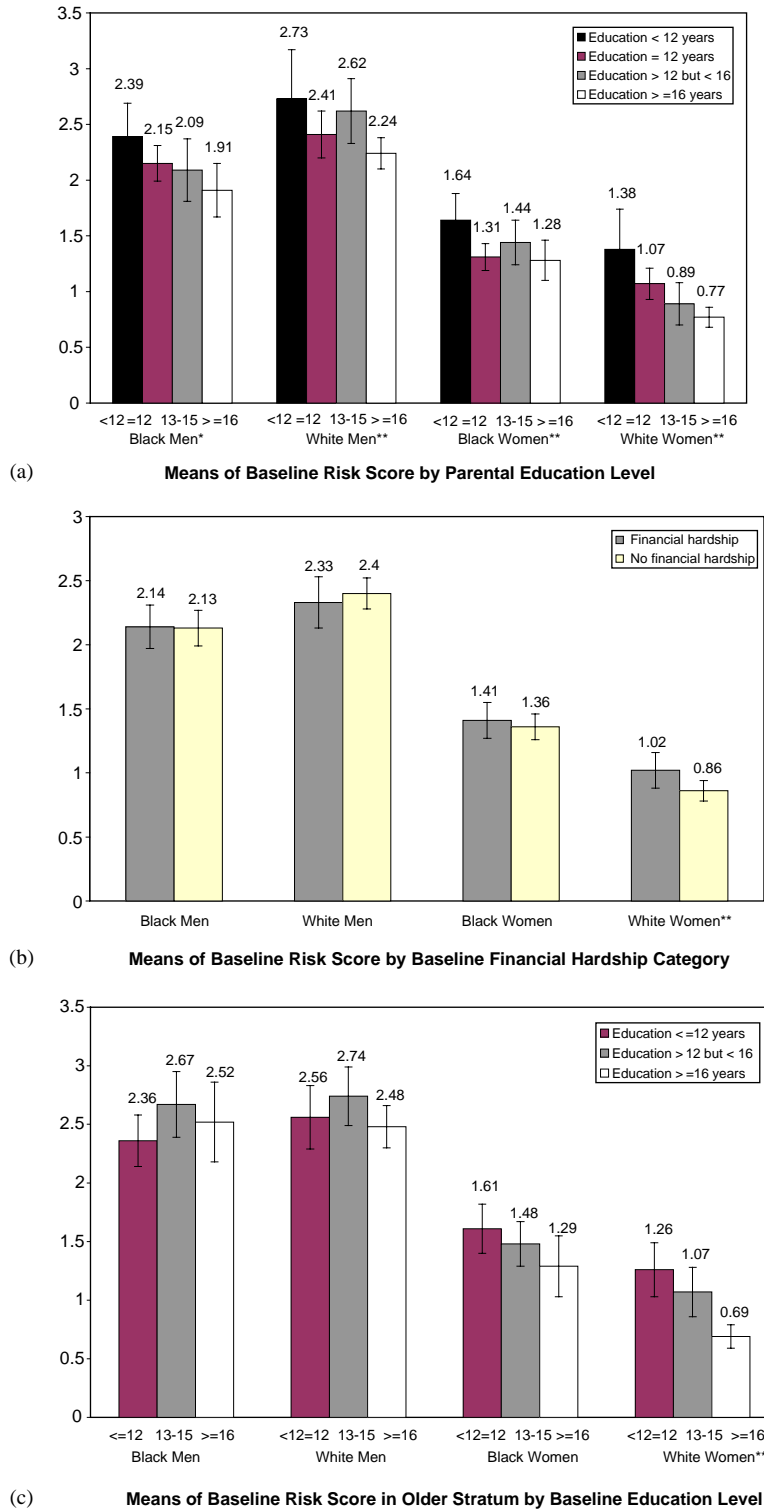(c)   **Means of Baseline Risk Score in Older Stratum by Baseline Education Level**

Fig. 2. Unadjusted associations between indicators of SES and baseline risk score by race and gender. Height of bars represents the point estimate of the mean score within each SES category; range shown is the 95% confidence interval. Panel: (a) association with parental education; (b) association with financial hardship at baseline; (c) association with participant's education at baseline in participants who were 25 years of age or older at baseline. *Two-sided p-value (for test of difference across SES categories) <0.10 but not less than 0.05. **Two-sided p-value (for test of difference across SES categories) <0.05.

Table 3
SES effects on risk change score

SES variable	Magnitude of effect[a] (standard error) (p-value)			
	Black men	White men	Black women	White women
Parent education[b]				
Less than high school	+0.16 (0.15)	+0.33 (0.23)	+0.34 (0.11)	+0.06 (0.13)
1–3 years of college	+0.10 (0.15)	+0.08 (0.15)	−0.03 (0.11)	−0.01 (0.09)
College graduate	−0.04 (0.13)	−0.22 (0.11)	−0.12 (0.11)	−0.26 (0.07)
	(NS)	(p = .01)	(p = .002)	(p = .0002)
Financial hardship at baseline[c]	+0.11 (0.10)	+0.25 (0.10)	+0.36 (0.08)	+0.14 (0.06)
	(NS)	(p = .01)	(p < .0001)	(p = .02)
Ongoing financial hardship[d]				
Some but not all follow-up visits	+0.11 (0.11)	+0.01 (0.10)	+0.10 (0.08)	+0.00 (0.07)
Every follow-up visit attended	−0.15 (0.16)	+0.06 (0.19)	+0.34 (0.11)	+0.20 (0.10)
	(NS)	(NS)	(p = .01)	(p = .10)
In older stratum[e]				
Participant education at baseline[f]				
1–3 years of college	+0.06 (0.17)	−0.20 (0.17)	−0.18 (0.13)	−0.10 (0.11)
College graduate	+0.10 (0.19)	−0.33 (0.15)	−0.60 (0.15)	−0.38 (0.09)
	(NS)	(p = .07)	(p = .0006)	(p < .0001)

[a]Difference in risk change score compared to the reference group, adjusted for baseline risk score. Effect of ongoing financial hardship was also adjusted for baseline financial hardship.

[b]Reference group is composed of those who had high school education only (12 years of regular schooling).

[c]Reference group is composed of those who reported no financial hardship.

[d]Reference group composed of those reporting no financial hardship at any follow-up visit. Effect adjusted also for baseline hardship.

[e]Participants who were 25 years of age or older at baseline.

[f]Reference group is composed of those who had high school or less education (≤12 years of regular schooling).

independent of baseline education level, only in white men ($p = .01$), in whom, advancing education status to 1–3 years of college during the study period was associated with a lower accumulated risk score (lower by 1.80, standard error 0.68) as was interim college graduation (accumulated risk score lower by 1.04, standard error 0.63). Similar but non-significant trends were seen in the other three groups. Adjusting for age, lifestyle habits, and access to medical care did not change the trends or effect sizes substantially (Table 5).

Discussion

In this study, we identified clusters of growth trajectories of cardiovascular risk factors in young adults, and examined the impact of SES on longitudinal risk accumulation from increases in multiple factors. Mean trajectories from different clusters crossed each other, but the cluster with the highest baseline value maintained its position as the highest risk cluster throughout the 10-year study period, confirming our hypothesis that many young adults who start with elevated risk factors, continue to have the highest-risk

profiles in later life. Other studies have documented similar stability over time of relative ranking within risk-factor distributions, both in children (Clarke et al., 1978; Sanchez-Bayle et al., 1999) and in young adults (McTigue, Garrett, & Popkin, 2002; Wilsgaard et al., 2001). This phenomenon, which has been called *tracking* (Ware & Wu, 1981), suggests that one should be able to identify in young adulthood and target for early intervention, the individuals who will go on to be at increased risk of cardiovascular disease (Yong, Kuller, Rutan, & Bunker, 1993; Tate, Manfreda, Krahn, & Cuddy, 1995).

As hypothesized, our investigation demonstrated inverse associations between indicators of SES and summary scores of baseline risk, risk change, and accumulated risk. Parental education, an indicator of childhood SES, had the most consistent associations with risk scores in all race/gender groups. The associations were strongest in white women and weakest in black men, in whom there was only a marginally significant association with baseline risk score and with accumulated risk score, and no association with risk change score. SES at baseline (the start of the study) was associated inversely with baseline score only in

Table 4
SES effects on risk change score, adjusted for age, lifestyle, and access to medical care

SES variable	Magnitude of effect[a] (standard error) (p-value)			
	Black men	White men	Black women	White women
Parent education[b]				
Less than high school	+0.09 (0.16)	+0.22 (0.25)	+0.17 (0.11)	+0.08 (0.13)
1–3 years of college	+0.16 (0.16)	+0.03 (0.16)	+0.02 (0.11)	+0.01 (0.09)
College graduate	−0.03 (0.15)	−0.23 (0.11)	−0.11 (0.11)	−0.20 (0.07)
	(NS)	(p = .05)	(NS)	(p = .004)
Financial hardship at baseline[c]	+0.22 (0.11)	+0.17 (0.11)	+0.21 (0.08)	+0.15 (0.07)
	(p = .05)	(p = .10)	(p = .007)	(p = .03)
Ongoing financial hardship[d]				
Some but not all follow-up visits	+0.20 (0.12)	+0.00 (0.11)	+0.06 (0.09)	+0.03 (0.07)
Every follow-up visit attended	−0.09 (0.18)	+0.02 (0.21)	+0.18 (0.12)	+0.18 (0.11)
	(p = .10)	(NS)	(NS)	(NS)
In older stratum[e]				
Participant education at baseline[f]				
1–3 years of college	+0.13 (0.19)	−0.25 (0.18)	−0.07 (0.13)	+0.03 (0.12)
College graduate	+0.13 (0.22)	−0.35 (0.16)	−0.45 (0.17)	−0.23 (0.11)
	(NS)	(p = .10)	(p = .02)	(p = .012)

[a]Difference in risk change score compared to the reference group, adjusted for age, smoking, alcohol consumption, physical exercise, access to medical care, and baseline risk score. Effect of ongoing financial hardship was also adjusted for baseline financial hardship.

[b]Reference group is composed of those who had high school education only (12 years of regular schooling).

[c]Reference group is composed of those who reported no financial hardship.

[d]Reference group composed of those reporting no financial hardship at any follow-up visit. Effect also adjusted for baseline hardship.

[e]Participants who were 25 years of age or older at baseline.

[f]Reference group is composed of those who had high school or less education ($\leqslant$12 years of regular schooling).

white women, with risk change score in three of the four race/gender groups (the exception being black men), and with accumulated risk score only in women. Upward SES mobility during the study was associated inversely with risk scores in selected groups: with risk change score in black women and with accumulated risk in white women and white men. This pattern is consistent with other studies that have found cardiovascular mortality to be more sensitive to early adversity than to adult circumstances (Davey Smith, Hart, Blane, Gillis, & Hawthorne, 1997) and suggests that SES effects on biological risk factors are long lasting and that it may take time for the effects of change in SES to be seen. Alternately, the pattern seen here may be related to the small number of participants in our study with upward SES mobility. However, others have also found that childhood adversity is negatively associated with physical health in young adulthood (Power, Manor, & Fox, 1991; Power & Matthews, 1998) and in middle age (Singer & Ryff, 1999).

SES associations were weakened but not all eliminated after adjusting for differences in age, lifestyle habits and in access to medical care. After adjustment, SES was associated inversely with baseline risk score in women (black and white), with risk change score in all four race/gender groups, and with accumulated risk score in women (black and white) and in white men. Our findings are consistent with other studies that have found that adjustment for health behaviors does not eliminate the association between SES and heart disease (Pincus & Callahan, 1995) and with studies that have documented these gradients even in countries with universal health coverage (Marmot, Kogevinas, & Elston, 1987). Factors other than health behaviors and healthcare access that could mediate the observed associations between SES and cardiovascular risk, include social stresses, lack of social support, environmental exposures, and access to public resources (Luepker et al., 1993).

Consistent with previous studies that have documented gender differences in cardiovascular risk factors (Winkelby, Fortmann, & Barrett, 1990; Reynes, Lasater, Feldman, Assaf, & Carleton, 1993; Pamuk, Makuc, Heck, Reuben, & Lochner, 1998; Colhoun, Hemingway, & Poulter, 1998; Winkelby, Cubbin, Ahn, & Kraemer, 1999; Molarius, Siedell, Sans, Tuomilehto, & Kuulasamaa, 2000; Wardle, Maller, & Jarvis, 2002), we found that the associations between SES and cardiovascular

(a)   **Means of Accumulated Risk Score by Parental Education Level**

(b)   **Means of Accumulated Risk Score by Baseline Financial Hardship Category**

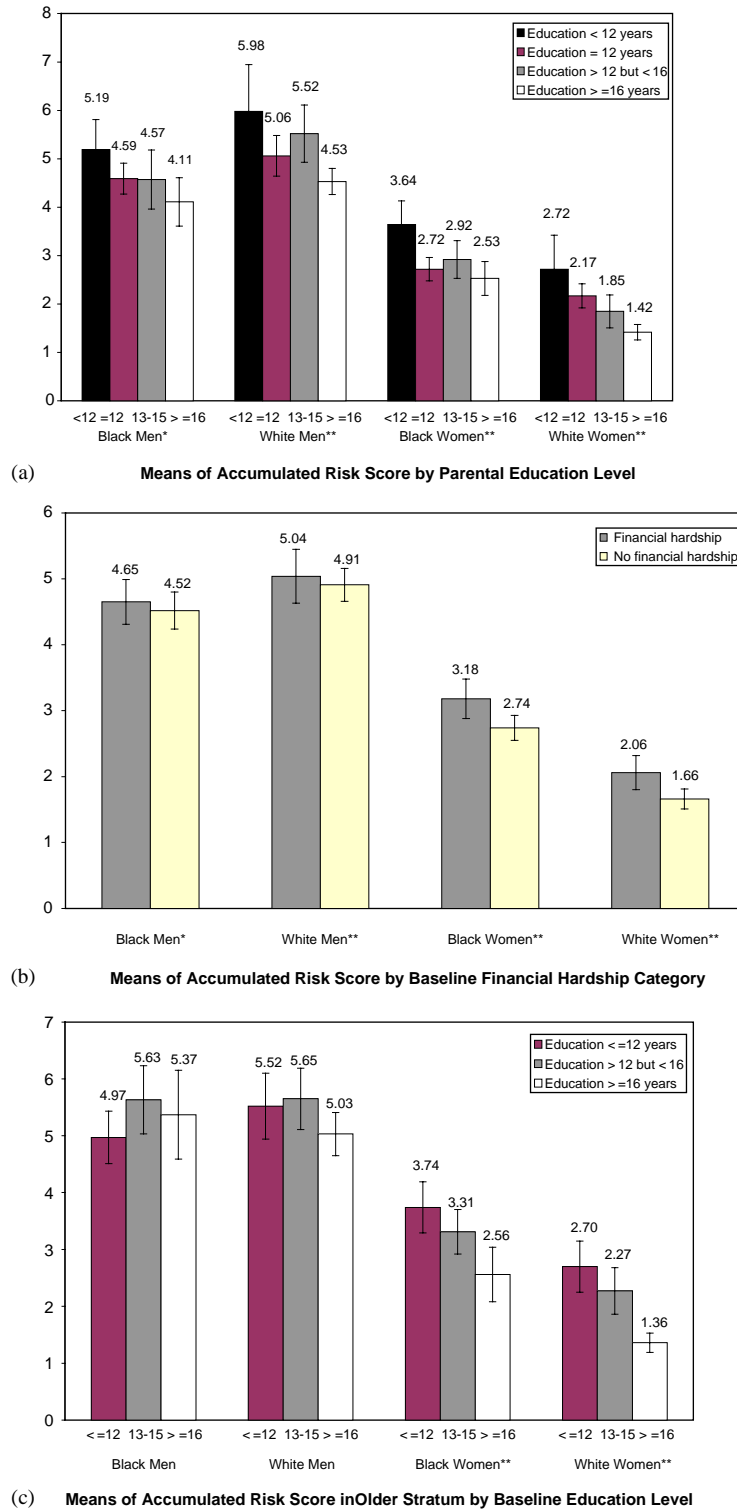(c)   **Means of Accumulated Risk Score inOlder Stratum by Baseline Education Level**

Fig. 3. Unadjusted associations between indicators of SES and accumulated risk score by race and gender. Height of bars represents the point estimate of the mean score within each SES category; range shown is the 95% confidence interval. Panel: (a) association with parental education; (b) association with financial hardship at baseline; (c) association with participant's education at baseline in participants who were 25 years of age or older at baseline. *Two-sided p-value (for test of difference across SES categories) <0.10 but not less than 0.05. **Two-sided p-value (for test of difference across SES categories) <0.05.

Table 5
Adjusted SES effects on accumulated risk score

SES variable	Magnitude of effect[a] (standard error) (p-value)			
	Black men	White men	Black women	White women
Parent education[b]				
Less than high school	+0.11 (0.36)	+0.65 (0.55)	+0.79 (0.26)	+0.64 (0.30)
1–3 years of college	+0.13 (0.36)	+0.43 (0.35)	+0.35 (0.25)	−0.23 (0.21)
College graduate	−0.25 (0.32)	−0.35 (0.25)	−0.06 (0.25)	−0.57 (0.16)
	(NS)	(p = .03)	(p = .009)	(p < .0001)
Financial hardship at baseline[c]	+0.13 (0.25)	−0.07 (0.25)	+0.29 (0.18)	+0.44 (0.15)
	(NS)	(NS)	(p = .10)	(p = .003)
Ongoing financial hardship[d]				
Some but not all follow-up visits	−0.26 (0.26)	+0.29 (0.24)	+0.24 (0.20)	+0.13 (0.16)
Every follow-up visit attended	−0.44 (0.40)	+0.35 (0.45)	+0.22 (0.28)	+0.56 (0.25)
	(NS)	(NS)	(NS)	(p = .08)
In older stratum[e]				
Participant education at baseline[f]				
1–3 years of college	+0.56 (0.43)	−0.03 (0.43)	−0.29 (0.31)	−0.22 (0.27)
College graduate	−0.02 (0.48)	−0.55 (0.39)	−1.07 (0.39)	−1.12 (0.24)
	(NS)	(NS)	(p = .02)	(p < .0001)
Additional education by last visit[g]				
Some college (less than 3 years)	−0.40 (0.58)	−2.03 (0.69)	−0.38 (0.44)	−0.25 (0.42)
College graduation	−0.37 (0.77)	−0.79 (0.64)	−0.70 (0.52)	−0.23 (0.34)
	(NS)	(p = 0.01)	(NS)	(NS)

[a]Difference in accumulated risk score compared to the reference group, adjusted for age, smoking, alcohol consumption, physical exercise, and access to medical care. Effect of SES change also adjusted for baseline SES variable.

[b]Reference group is composed of those who had high school education only (12 years of regular schooling).

[c]Reference group is composed of those who reported no financial hardship.

[d]Reference group composed of those reporting no financial hardship at any follow-up visit. Effect also adjusted for baseline hardship.

[e]Participants who were 25 years of age or older at baseline.

[f]Reference group is composed of those who had high school or less education ($\leqslant$12 years of regular schooling).

[g]Reference group composed of those whose education level did not change over the study. Effect also adjusted for baseline education.

risk accumulation were stronger in women than in men. It has been postulated that such differences are a result of underlying gender differences in biological vulnerability, social coping mechanisms, and access to material, social, and psychological resources (MacIntyre & Hunt, 1997). There are also documented gender differences in behavioral and physiological responses to stress, which might explain the heightened sensitivity of biology to SES in women (Taylor et al., 2000). We also found racial differences, with whites showing greater SES-risk associations than blacks. This is consistent with previous work that has found that the effects of SES on cardiovascular risk factors are larger in whites than in blacks and may even be in the opposite direction in black men (Jacobs et al., 1988; Watkins, Neaton, & Kuller, 1986; Kraus, Borhani, & Franti, 1980). Given that health differences between ethnic groups are largely a result of social rather than genetic differences (American Association of Physical Anthropology, 1996), these findings imply that SES indicators such as education and hardship do not fully reflect the complexities of social circumstances faced by minority groups. Others have noted for instance, that blacks report more stress than whites at comparable levels of SES (Kessler & Neighbors, 1986), have less income at comparable levels of education (Krieger, Rowley, Herman, Avery, & Phillips, 1993), and have less wealth and purchasing power at comparable levels of income (Williams, 1996; Kaufman, Cooper, & McGee, 1997). In addition, residential segregation leads to racial differences in environmental exposure, community resources, employment opportunities, access to healthier foods, and ease of access to cigarettes, alcohol, and recreational drugs (Brown 1995; Winkelby et al., 1999; LaVeist & Wallace, 2000). Residential segregation also leads to differences in the quality of education, so that returns from education (both employment opportunities and

income) are lower in blacks (Williams, 1996; Wilhelm, 1987; Williams & Collins, 2001), and this disparity is greater in men than women (Williams & Collins, 1995).

In addition, the health benefits of increased SES may be attenuated in blacks because of increased racial discrimination at higher SES (Clark, Anderson, Clark, & Williams, 1999; Williams & Neighbors, 2001). Institutional barriers that thwart prosperity and power, and "everyday discrimination" events such as being followed in stores for suspicion of shoplifting or not being able to hail a cab, contribute to the stresses faced by blacks in American society (Clark et al., 1999; Meyer, 2003). Because men may be exposed to greater discrimination and/or respond to discrimination differently from women (say, suppress anger vs. talking about it), the attenuation of SES benefits may be more pronounced in black men than in black women, as suggested by our study, and the SES gradient may even be reversed in black men, as seen in some cross-sectional studies (Watkins et al., 1986; Knox, Jacobs, Chesney, Raczynski, & McCreath, 1996; Burke et al., 1990; Freedman, Strogatz, Williamson, & Aubert, 1992).

There are some limitations of our study that need to be acknowledged. Attrition (19%) is one issue—individuals lost to follow-up tended to be lower in SES than those who were retained in the study. Attrition was highest in black men, which may have reduced our power to detect SES differences in cardiovascular risk accumulation in black men. Another issue relates to the fact that many participants were still in school, and social and economic stressors during years of schooling differ considerably from those in the workplace. We attempted to adjust for these differences by including a categorical age term ($\leqslant 24$ and > 24 years) in the multivariable model. Thirdly, the summary risk scores used in the study weighted each risk factor equally and have not been validated. However, others have successfully used un-weighted counts of the number of elevated cardiovascular risk factors to create summary risk scores, and have documented both inverse associations with SES (Winkelby et al., 1990; Helmert, Herman, Joeckel, Greiser, & Madans, 1989) and positive associations with adverse health outcomes (Seeman, Singer, Rowe, Horwitz, & McEwen, 1997; Trevisan, Liu, Bahsas, & Menotti, 1998; Berenson et al., 1998; Ford, 2004). In future work, we will investigate how the summary risk scores created here, predict cardiovascular outcomes in this cohort. Lastly, this was a study of young adults in the USA, and it is not clear how some of these findings would translate to populations in other countries. In particular, the racial differences in SES effects that were seen in this study may, at least partially, be a result of the history of race relations in the USA (Cain & Kington, 2003), and not be generalizable to other societies. However, the consistency of SES-health gradients in studies from different countries suggests that our primary finding of SES effects on risk accumulation is likely to generalize to young adults in other countries.

One of the strengths of this study is derived from the large numbers of participants in each race/gender group, and the diversity of SES represented in the cohort. Also, because this is a young cohort, there was minimal risk of reverse confounding by poor cardiovascular health on SES.

In conclusion, this study found that young adults with the highest values of a risk factor at baseline had further increases in the risk factor over the next 10 years, that accumulated risk was greatest for those with lowest SES, independent of differences in lifestyle and healthcare access, and that SES effects were strongest in white women and weakest in black men. Changes in SES over the study period were also weakly associated with accumulation of risk, independent of baseline SES. Our findings imply that risk-factor trajectories may be determined early in life, and that SES effects on risk may be long lasting, though upward SES mobility may lead to some risk reduction.

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