Association of Socioeconomic Status and Exercise Capacity in Adults With Coronary Heart Disease (from the Heart and Soul Study)

Beth Cohen, MD, MAS[a,c,*], Eric Vittinghoff, PhD[a,b], and Mary Whooley, MD[a,b,c]

Lower socioeconomic status (SES) was associated with reduced treadmill exercise capacity and predicted adverse cardiovascular outcomes. Why patients with low SES had reduced exercise capacity and whether this relation existed in patients with coronary heart disease (CHD) was not known. Using data from the Heart and Soul Study, the association of 4 indicators of SES (household income, education, housing status, and occupation) with treadmill exercise capacity was analyzed in 943 men and women with stable CHD. In multivariable linear regression models adjusted for demographic variables, co-morbidities, medication use, and health behaviors (smoking, alcohol use, body mass index, physical activity, and medication adherence), exercise capacity significantly decreased in a graded fashion from the highest to lowest categories of each SES variable (p <0.001 for all trends). Differences in exercise capacity between the lowest and highest SES categories were 2.4 METs for household income, 1.8 METs for education, 2.3 METs for housing, and 1.3 METs for occupation. In similarly adjusted logistic regression models comparing the lowest with the highest categories of SES, low SES was strongly associated with impaired exercise capacity (defined as <5 METs; odds ratios for income 5.5, 95% confidence interval [CI] 1.9 to 16.0; education 4.3, 95% CI 2.0 to 9.5; housing 4.5, 95% CI 2.1 to 9.6; and occupation 2.8, 95% CI 1.4 to 5.7, p ≤0.001 for all trends). In conclusion, 4 indicators of low SES were strongly associated with decreased exercise capacity in patients with CHD. Differences in traditional cardiac risk factors and health behaviors did not explain this association. **(Am J Cardiol 2008;101:462–466)**

Lower socioeconomic status (SES) was linked with increased cardiovascular morbidity and mortality, but the reasons for this association were not well understood.[1–7] We examined the association of multiple SES indicators (income, education, housing status, and occupation) with treadmill exercise capacity in 943 ambulatory outpatients with coronary heart disease (CHD) from a wide range of socioeconomic backgrounds. We sought to determine whether the relation between SES and exercise capacity was explained by demographic factors, co-morbidities, medication use, or health behaviors.

Departments of [a]Medicine and [b]Epidemiology & Biostatistics, University of California, San Francisco; and [c]Veterans Affairs Medical Center, San Francisco, California. Manuscript received June 22, 2007; revised manuscript received and accepted September 21, 2007.

The Heart and Soul Study was supported by the Department of Veterans Affairs, Washington, DC; the National Heart Lung and Blood Institute (R01 HL079235), Bethesda, Maryland; the American Federation for Aging Research (Paul Beeson Scholars Program), New York, New York; the Robert Wood Johnson Foundation (Faculty Scholars Program), Princeton, New Jersey; the Ischemia Research and Education Foundation, South San Francisco, California; and the Nancy Kirwan Heart Research Fund, San Francisco, California.

*Corresponding author: Tel: 415-221-4810, ext. 4851; fax: 415-379-5573.

E-mail address: Beth.Cohen@ucsf.edu (B. Cohen).

Methods

The Heart and Soul Study is prospective cohort study of psychosocial variables and health outcomes in patients with CHD. The complete methods of the study were described previously.[8] Briefly, subjects were recruited from 2 Department of Veterans Affairs Medical Centers (San Francisco Veterans Affairs Medical Center and the Veterans Affairs Palo Alto Health Care System, California), 1 university medical center (University of California, San Francisco), and 9 public health clinics (Community Health Network of San Francisco). To be eligible, participants needed ≥1 of: history of myocardial infarction, angiographic evidence of stenosis ≥50% in ≥1 coronary vessel, evidence of exercise-induced ischemia using treadmill electrocardiogram or stress nuclear perfusion imaging, or history of coronary revascularization. Those who were unable to walk 1 block or were planning to leave the area within 3 years were excluded. From September 2000 to December 2002, a total of 1,024 ambulatory men and women with established CHD enrolled. This cross-sectional analysis was limited to the 943 subjects who completed exercise treadmill testing. The protocol was approved by the appropriate institutional review boards, and all subjects provided written informed consent.

Subjects completed questionnaires with detailed demographic data. We assessed the 4 different SES variables of annual household income (6 categories) divided by household size (5 categories), level of education (8 categories), housing status (4 categories), and occupation (9 categories).

0002-9149/08/$ – see front matter
doi:10.1016/j.amjcard.2007.09.093

Table 1
Characteristics of 943 subjects by exercise capacity (in METs)

Characteristics	Normal Exercise Capacity (≥5 METS) (n = 714)	Impaired Exercise Capacity (<5 METS) (n = 229)	p Value
Age (yrs)	65.4 ± 10	71.0 ± 11	<0.001
Men	602 (84%)	183 (80%)	0.17
White race	424 (59%)	150 (65%)	0.25
Hypertension	483 (68%)	178 (78%)	0.004
Diabetes mellitus	160 (22%)	76 (33%)	0.001
Heart failure	100 (14%)	56 (24%)	<0.001
Myocardial infarction	380 (53%)	121 (54%)	0.90
Coronary artery bypass grafting	265 (37%)	81 (35%)	0.63
Percutaneous revascularization	307 (43%)	69 (30%)	0.001
Chronic pulmonary disease	100 (14%)	47 (21%)	0.02
Depression	114 (16%)	57 (25%)	0.002
Exercise-induced wall motion abnormality	158 (22%)	70 (31%)	0.008
Resting left ventricular ejection fraction (%)	62 ± 9	61 ± 10	0.01
Medication use			
Aspirin	572 (80%)	169 (74%)	0.05
Renin-angiotensin system inhibitor	349 (49%)	135 (59%)	0.008
β Blocker	411 (58%)	139 (61%)	0.39
Statin	488 (68%)	127 (56%)	<0.001
Systolic blood pressure (mm Hg)	131 ± 19	134 ± 19	0.04
Diastolic blood pressure (mm Hg)	75 ± 10	72 ± 11	0.002
Total cholesterol (mg/dl)	177 ± 41	177 ± 47	0.82
Low-density lipoprotein cholesterol (mg/dl)	104 ± 33	101 ± 34	0.21
High-density lipoprotein cholesterol (mg/dl)	46 ± 14	44 ± 14	0.06
Triglycerides (mg/dl)	138 ± 131	150 ± 124	0.20
Current tobacco use	132 (19%)	52 (23%)	0.17
Heavy alcohol use	218 (31%)	59 (26%)	0.15
Body mass index	28 ± 5	29 ± 6	<0.001
Physical inactivity	210 (29%)	116 (51%)	<0.001
Medication nonadherence	58 (8%)	16 (7%)	0.58

Table 2
Difference in mean exercise capacity between highest and lowest categories of socioeconomic status (SES)*

SES Variable	Age-Adjusted		Adjusted for Confounders†		Adjusted for Confounders Plus Health Behaviors		Adjusted for Confounders, Health Behaviors, and Other SES Variables	
	Mean Difference in METs (95% CI)	p Value Trend	Mean Difference in METs (95% CI)	p Value Trend	Mean Difference in METs (95% CI)	p Value Trend	Mean Difference in METs (95% CI)	p Value Trend
Income	3.4 (2.6–4.2)	<0.001	2.6 (1.8–3.4)	<0.001	2.4 (1.6–3.1)	<0.001	1.8 (1.0–2.6)	<0.001
Education	2.9 (2.1–3.6)	<0.001	2.1 (1.4–2.8)	<0.001	1.8 (1.1–2.5)	<0.001	1.1 (0.2–1.9)	0.02
Housing	3.3 (2.4–4.1)	<0.001	2.6 (1.8–3.4)	<0.001	2.3 (1.5–3.0)	<0.001	1.9 (1.1–2.7)	<0.001
Occupation	2.4 (1.6–3.1)	<0.001	1.7 (1.0–2.3)	<0.001	1.3 (0.6–1.9)	<0.001	0.3 (−0.4–1.1)	0.20

* n = 726 for analyses including occupation, n = 850 for all others.

† Adjusted for all variables listed in Table 1 except health behaviors (tobacco use, heavy alcohol use, body mass index, physical inactivity, and medication nonadherence).

CI = confidence interval.

For each SES variable, categories with comparatively few responses were combined with the nearest category. Household income was adjusted for household size by dividing the median of the income category by the median of the household size category. For occupation, those answering "other" were excluded from analyses involving occupation. A sensitivity analysis including the other occupation category produced similar results. This yielded 5 categories for adjusted annual household income (<\$10,000, \$10,000 to \$19,999, \$20,000 to \$29,999, \$30,000 to \$39,999, and >\$40,000), 5 categories for level of education (high school not completed, high school completed, <4 years of college, 4 years of college completed, and graduate or professional school completed), 3 categories for housing status (from lowest to highest SES: hotel room, boarding house, or shelter; apartment or retirement community; and house), and 5 categories for occupation, which were grouped using Census-based methods from previous work on SES and heart disease (from lowest to highest SES: service and laborers; clerical and sales; craftsmen, foremen, manufacturing, and transportation; managers, officials, and proprietors; and professional and technical).[3]

All subjects completed a graded exercise treadmill test limited by symptoms according to a standard Bruce protocol. To achieve maximum heart rate, subjects unable to continue the standard Bruce protocol (for orthopedic or other reasons) were switched to slower settings on the treadmill and encouraged to exercise for as long as possible. Maximum exercise capacity was calculated as total number of METs achieved at peak exercise (1 MET = 3.5 mL/kg/min of oxygen consumption). We defined impaired exercise capacity as <5 METs and normal exercise capacity as ≥5 METs.[9] An echocardiogram was obtained before and after the treadmill test, and the presence of new wall motion abnormalities at peak exercise were recorded. Left ventricular ejection fraction was measured during echocardiography at rest. Technicians performing the treadmill tests were blinded to the study goals.

Demographic variables and medical history were assessed using a self-reported questionnaire. Subjects were instructed to bring their medication bottles to the study appointment, and study personnel recorded all current medications. Fasting (12-hour) venous samples were drawn to measure high- and low-density lipoprotein cholesterol. Systolic, and diastolic blood pressure when supine and at rest were measured using a standard sphygmomanometer. We measured height and weight and calculated body mass index as weight divided by the square of height in meters. We measured alcohol use with the Alcohol Use Disorders Identification Test Consumption questions (AUDIT-C) questionnaire, with a score ≥4 indicating heavy use.[10] To assess depressive symptoms, we administered the 9-item Patient Health Questionnaire and defined depression as a score ≥10, which has sensitivity of 88% and specificity of 88% for major depressive disorders.[11] Subjects rated their physical activity during the previous month using a 6-point Likert scale. Those responding "not at all active" or "a little active" were classified as physically inactive. Subjects rated their medication adherence during the past month using a 5-point Likert scale. Those who responded that they took their medication "all of the time" or "nearly all of the time" were considered adherent.[12]

Differences in characteristics between subjects with and without impaired exercise capacity were compared using *t* tests for continuous variables and chi-square tests for dichotomous variables. We used multivariable linear regression models to evaluate the association of income, education, housing, and occupation, treated as multilevel categorical variables, with exercise capacity, treated as a continuous variable. We adjusted for multiple possible confounding variables, including demographics (age, sex, and race/ethnicity), co-morbidities (depression based on 9-item Patient Health Questionnaire scores and history of hypertension, diabetes mellitus, chronic obstructive pulmonary disease, and congestive heart failure), severity of cardiac disease (previous myocardial infarction, coronary artery bypass graft, and percutaneous transluminal coronary angioplasty; exercise-induced wall motion abnormality; and left ventricular ejection fraction), current medication use (aspirin, β blockers, angiotensin-converting enzyme inhibitors/angiotensin receptor blockers, and statins), and other cardiovascular risk factors (systolic blood pressure, diastolic blood pressure, total cholesterol, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, and triglycerides). To determine the relative effect of potential confounding variables and mediating health behaviors on the association between SES and exercise capacity, we constructed serially adjusted multivariable linear regression models. The first model adjusted for only age, the second model adjusted for age and the potential confounders listed, and the third model adjusted for age, confounders, and health behaviors (tobacco use, heavy alcohol use, body mass index, physical activity, and medication adherence).

We used multivariable logistic regression to assess the association of income, education, housing, and occupation, treated as multilevel categorical variables, with impaired exercise capacity, treated as a dichotomous variable (<5 vs ≥5 METS), adjusting for covariates included in the linear models. We used standard orthogonal linear contrasts to assess trend in the log odds ratio or fitted mean across the ordinal SES categories. We tested for interactions between income, education, housing, and occupation in predicting treadmill exercise capacity. To examine whether an individual SES variable could predict exercise capacity independent of the other SES variables, we constructed linear and logistic models that included all 4 SES predictors simultaneously. Analyses were performed using STATA, version 9.0 (SAS Institute Inc, Cary, North Carolina). All tests were 2 tailed, and $p < 0.05$ was considered statistically significant.

Results

Of 943 subjects, 229 (24%) had impaired exercise capacity (METS <5). Characteristics of subjects with normal versus impaired exercise capacity are listed in Table 1. Lower levels of income, education, housing, and occupation were associated with significantly lower exercise capacity in age-adjusted models (p for trend <0.001 for each SES measure; Table 2). After adjustment for potential confounding factors, the mean difference in exercise capacity between the highest and lowest categories for each SES variable ranged from 1.7 to 2.6 METs. The increase in exercise capacity occurred in a graded fashion over nearly all levels of income, education, housing, and occupation (Figure 1).

In logistic regression models adjusting for the same potential confounders, the odds of having impaired exercise capacity were significantly higher in those with the lowest versus highest levels of income, education, housing, or occupation (p for trend <0.001 for each SES measure; Table 3).

In staged multivariable models, adding the 5 lifestyle factors to models that included confounders further reduced estimates of the difference in mean exercise capacity in the highest versus lowest categories of SES by 8% for income, 14% for education, 12% for housing, and 24% for occupation. However, the association with SES and exercise capacity remained highly significant (Table 2). In staged logistic regression models, adding the lifestyle factors further reduced odds ratios for impaired exercise capacity by 10% for income, 4% for education, 12% for housing, and 15% for occupation. Again, the association of lower SES with impaired exercise capacity remained strong (Table 3).

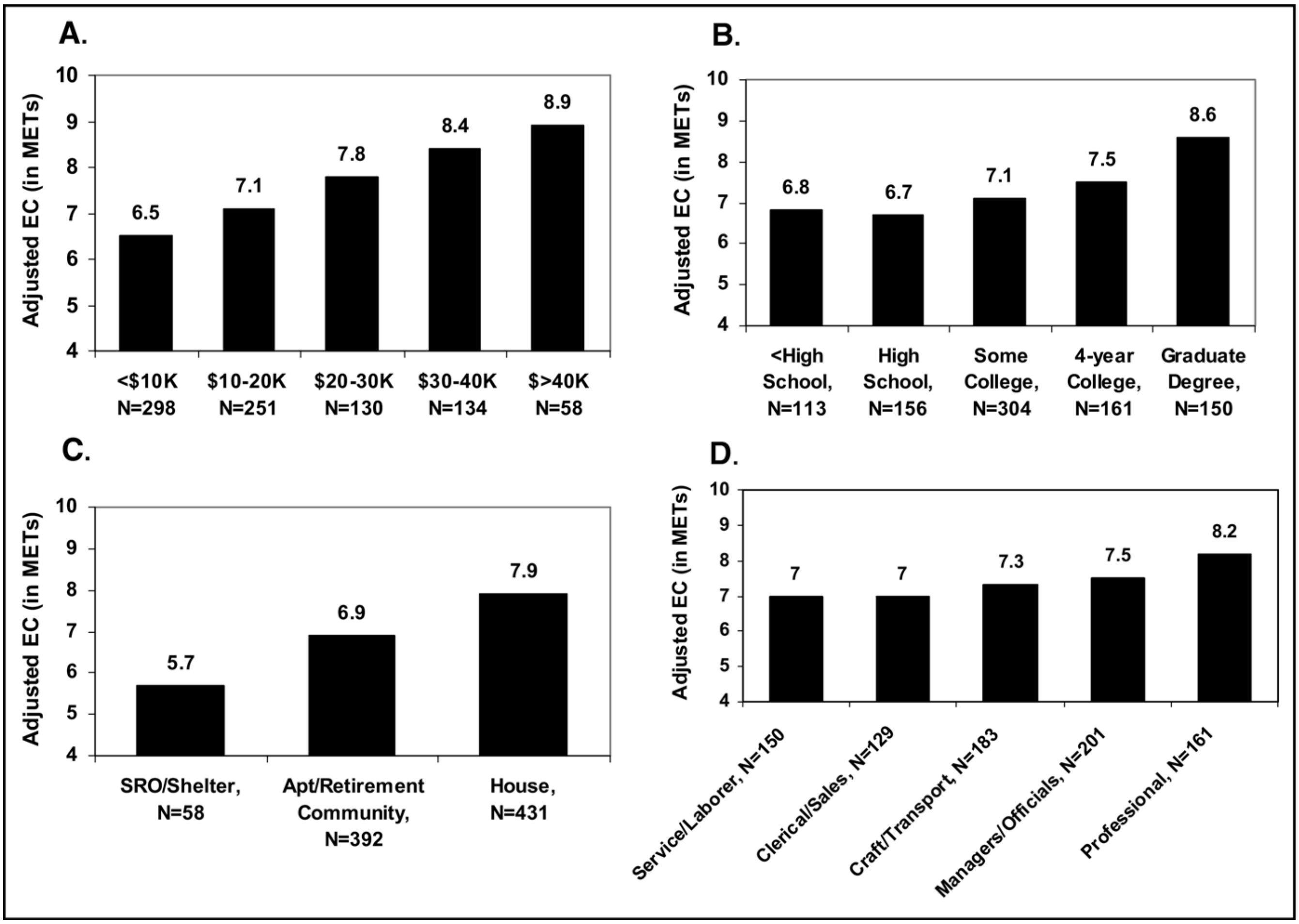

Figure 1. Exercise capacity (EC) values expressed in METs are based on multivariable linear regression models adjusted for all variables listed in Table 1. Adjusted exercise capacity was calculated for each category of *(A)* household income, *(B)* level of education, *(C)* housing status, and *(D)* occupation.

Table 3
Association of lowest (vs highest) category of socioeconomic status (SES) with impaired exercise capacity (METS <5)*

SES Variable	Age-Adjusted		Adjusted for Confounders†		Adjusted for Confounders Plus Health Behaviors		Adjusted for Confounders, Health Behaviors, and Other SES Variables	
	Odds Ratio‡ (95% CI)	p Value Trend	Odds Ratio (95% CI)	p Value Trend	Odds Ratio (95% CI)	p Value Trend	Odds Ratio (95% CI)	p Value Trend
Income	6.7 (2.5–17.8)	<0.001	6.1 (2.1–17.8)	<0.001	5.5 (1.9–16.0)	<0.001	3.4 (1.1–10.6)	0.02
Education	4.2 (2.2–8.2)	<0.001	4.5 (2.1–9.6)	<0.001	4.3 (2.0–9.5)	<0.001	2.9 (1.0–8.4)	0.03
Housing	6.5 (3.4–12.5)	<0.001	5.1 (2.5–10.4)	<0.001	4.5 (2.1–9.6)	<0.001	3.5 (1.4–8.4)	0.006
Occupation	3.3 (1.9–5.9)	<0.001	3.3 (1.7–6.4)	<0.001	2.8 (1.4–5.7)	0.001	1.3 (0.6–2.9)	0.32

* n = 726 for analyses including occupation, n = 850 for all others.
† Adjusted for all variables listed in Table 1 except health behaviors (tobacco use, heavy alcohol use, body mass index, physical inactivity, and medication nonadherence).
‡ Odds ratios calculated using the lowest versus highest category for each SES variable.
CI = confidence interval.

When all 4 SES variables were included in the linear and logistic models, income, education, and housing remained significant independent predictors of exercise capacity (Tables 2 and 3). Associations with occupation were no longer significant. No significant interactions were observed among the 4 SES variables (all p values for interaction > 0.80).

Discussion

Exercise capacity is a well-established predictor of cardiovascular and overall mortality.[9,13] We found that 4 measures of SES (income, education, housing, and occupation) were significantly associated with exercise capacity in a cohort of patients with CHD. This association was reduced, but remained highly significant, after extensive adjustment for demographic variables, co-morbid conditions, severity of cardiac disease, blood pressure, lipids, medication use, and health behaviors. The graded increase in exercise capacity over nearly all levels of each SES variable showed that the association was not driven solely by differences in the extreme categories of SES.

The 1.3- to 2.4-MET decreases in adjusted exercise capacity present in the lowest categories of the 4 SES variables were clinically significant. Myers et al[9] found that in patients with cardiovascular disease, peak exercise capacity was a stronger predictor of death than the traditional cardiac risk factors of history of diabetes, myocardial infarction, congestive heart failure, and exercise-induced ST-segment depression or ventricular arrhythmia. Every 1-MET decrease in baseline exercise capacity decreased the likelihood of survival by 12% over a mean 6.2 years of follow-up.[9] Therefore, the differences we observed could result in substantially increased mortality in patients with cardiac disease and lower SES.

In models that included all 4 SES variables, only occupation did not remain a significant independent predictor of exercise capacity. This may be caused by the older age of our population, reflecting that many subjects were retired, and perhaps the impact of their previous occupation was diminished. However, other studies showed that variables acting earlier in life, such as parental occupation or childhood social status, could predict future cardiovascular risk factors and disease independent of contemporary SES.[14–16] It is possible that previous occupation influenced exercise capacity, but this effect was captured by the other variables, such as income or education.

Only 1 previous study examined the association between SES and exercise capacity. Shishehbor et al[17] studied 30,043 patients referred to a tertiary-care center for treadmill testing. They found that lower geographically defined SES (a composite score based on characteristics of the subject's census block) was associated with impaired exercise capacity independent of known cardiac risk factors and other potential confounders. Exercise capacity and heart rate recovery, a measure of cardiac autonomic function, explained 47% of the association between SES and total mortality.[17] This large study suggested exercise capacity may be an important mediator in the relation between SES and mortality. However, the study population was derived from a closed tertiary-care center, and this may have limited the socioeconomic diversity. Most subjects were privately insured, and the lowest SES quartile had a median annual household income >$30,000.

Our study expanded upon this important previous work by showing a strong association between low SES and poor exercise capacity in patients with established CHD. This association was present across 4 different measures of individual-level SES in a socioeconomically diverse patient population. The relation was not explained by differences in health behaviors across the SES strata. In addition, because we did not use a composite SES score, we were able to determine the independent predictive value of each SES variable. This is important because these measure various aspects of SES, such as economic resources and prestige, which may influence cardiac function and mortality through different pathways.

Our study had several important limitations. First, because our data were cross sectional, we cannot draw inferences about the causal direction of the association between SES and exercise capacity. Second, all patients in this cohort had a history of CHD. Although this extended previous findings to an important group of patients at high risk of cardiovascular morbidity and mortality, our results were not necessarily generalizable to patients without CHD. Third, our socioeconomic variables were based on self-report, rather than review of individual records. Last, many of our confounding variables and health behaviors were also measured using self-report. Therefore, it is possible that residual confounding remained and more thorough measurement of these covariates would have led to a weaker association between SES and exercise capacity.

1. Tonne C, Schwartz J, Mittleman M, Melly S, Suh H, Goldberg R. Long-term survival after acute myocardial infarction is lower in more deprived neighborhoods. *Circulation* 2005;111:3063–3070.
2. Diez Roux AV. Persistent social patterning of cardiovascular risk: rethinking the familiar. *Circulation* 2005;111:3020–3021.
3. Diez Roux AV, Merkin SS, Arnett D, Chambless L, Massing M, Nieto FJ, Sorlie P, Szklo M, Troyler HA, Watson RL. Neighborhood of residence and incidence of coronary heart disease. *N Engl J Med* 2001;345:99–106.
4. Lantz PM, House JS, Lepkowski JM, Williams DR, Mero RP, Chen J. Socioeconomic factors, health behaviors, and mortality: results from a nationally representative prospective study of US adults. *JAMA* 1998; 279:1703–1708.
5. Anderson RT, Sorlie P, Backlund E, Johnson N, Kaplan GA. Mortality effects of community socioeconomic status. *Epidemiology* 1997;8:42–47.
6. Yen IH, Kaplan GA. Neighborhood social environment and risk of death: multilevel evidence from the Alameda County Study. *Am J Epidemiol* 1999;149:898–907.
7. Smith GD, Hart C, Watt G, Hole D, Hawthorne V. Individual social class, area-based deprivation, cardiovascular disease risk factors, and mortality: the Renfrew and Paisley Study. *J Epidemiol Community Health* 1998;52:399–405.
8. Ruo B, Rumsfeld JS, Hlatky MA, Liu H, Browner WS, Whooley MA. Depressive symptoms and health-related quality of life: the Heart and Soul Study. *JAMA* 2003;290:215–221.
9. Myers J, Prakash M, Froelicher V, Do D, Partington S, Atwood JE. Exercise capacity and mortality among men referred for exercise testing. *N Engl J Med* 2002;346:793–801.
10. Bush K, Kivlahan DR, McDonell MB, Fihn SD, Bradley KA, for the Ambulatory Care Quality Improvement Project (ACQUIP). The AUDIT alcohol consumption questions (AUDIT-C): an effective brief screening test for problem drinking? *Arch Intern Med* 1998;158:1789–1795.
11. McManus D, Pipkin SS, Whooley MA. Screening for depression in patients with coronary heart disease (data from the Heart and Soul Study). *Am J Cardiol* 2005;96:1076–1081.
12. Gehi A, Haas D, Pipkin S, Whooley MA. Depression and medication adherence in outpatients with coronary heart disease: findings from the Heart and Soul Study. *Arch Intern Med* 2005;165:2508–2513.
13. Chang JA, Froelicher VF. Clinical and exercise test markers of prognosis in patients with stable coronary artery disease. *Curr Probl Cardiol* 1994;19:533–587.
14. Wannamethee SG, Whincup PH, Shaper G, Walker M. Influence of fathers' social class on cardiovascular disease in middle-aged men. *Lancet* 1996;348:1259–1263.
15. Lynch JW, Kaplan GA, Salonen JT. Why do poor people behave poorly? Variation in adult health behaviours and psychosocial characteristics by stages of the socioeconomic lifecourse *Soc Sci Med* 1997;44:809–819.
16. Power C, Hertzman C. Social and biological pathways linking early life and adult disease. *Br Med Bull* 1997;53:210–221.
17. Shishehbor MH, Litaker D, Pothier CE, Lauer MS. Association of socioeconomic status with functional capacity, heart rate recovery, and all-cause mortality. *JAMA* 2006;295:784–792.