Socioeconomic Status Variables Predict Cardiovascular Disease Risk Factors and Prospective Mortality Risk among Women with Chest Pain: The WISE Study

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This study examined the relationship between socioeconomic status (SES), coronary artery disease (CAD) risk factors, and all-cause mortality in a cohort of women with chest pain. A total of 743 women (mean age = 59.6 years) with chest pain who were referred for coronary angiography completed a diagnostic protocol including CAD risk factor assessment, ischemic testing, psychosocial testing, and queries of SES. Patients were followed for about 2 years to track subsequent all-cause mortality. Results indicated that low SES was associated with CAD risk factors, including higher BMI and waist-hip ratios, cigarette smoking, lower reported activity levels, and a greater probability of hypertension. Low income also predicted all-cause mortality (RR = 2.7, 95% CI 1.4, 5.2), including after adjusting for proposed psychosocial and behavioral variables (RR = 5.9, 95% CI 1.2-29.7). Future research will require a thorough a priori focus on potential mechanisms to better understand SES effects on health.

Socioeconomic Status Variables Predict Cardiovascular Disease Risk Factors and Prospective Mortality Risk Among Women With Chest Pain

The WISE Study

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Markers of socioeconomic standing (SES)—most frequently including education, income, and occupation—are among the most consistent psychosocial predictors of morbidity and mortality (Kaplan, Pamuk, Lynch, Cohen, & Balfour, 1996; Syme & Berkman, 1976; Taylor & Seeman, 1999). More than five decades of research indicate that SES is reliably associated with an increased risk of coronary artery disease (CAD), stroke, cancer, and respiratory infections, among other disease endpoints (Anderson & Armstead, 1995; Cohen, 1999; Kaplan & Keil, 1993; Pickering, 1999). The magnitude of SES effects also rivals the risk associated with more commonly considered biomedical risk factors (Marmot, Kogenivas, & Elston, 1987), and SES variables are currently a frequent topic of investigation in the context of studies assessing morbidity and mortality differences between men and women (Matthews, Kelsey, Meilahn, Kuller, & Wing, 1989; Wamala, Mittleman, Schenck-Gustafsson, & Orth-Gomer, 1999), and Black and White populations in the United States (Geronimus, Bound, Waidmann, Hillemeier, & Burns, 1996; Gillum, 1996; Williams & Collins, 1995). Despite these well-documented associations with important clinical outcomes, clinical research often

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fails to consider financial and other SES factors in the estimation of major adverse events.

The mechanisms explaining the association between SES and subsequent disease development and mortality are believed to be many and include behavioral, biological, psychological, and social factors. Reviews of the SES-health literature increasingly call for a focus on identifying these potential mechanisms (Adler et al., 1994; Adler & Ostrove, 1999; Baum, Garofalo, & Yali, 1999; Gallo & Matthews, 1999), specifically citing a need for prospective studies including measures of stress and negative affect, social and behavioral factors, health care access, and biological processes in addition to SES variables. An improved knowledge of behavioral or biological agents responsible for SES effects on health would not only benefit future research in this area but would also better enable health care policy makers to design interventions to potentially modify these factors.

The current study addressed the impact of low SES (i.e., low income and education) on CAD risk factors and mortality risk in a cohort of 743 women with chest pain as part of the multisite Women's Ischemia Syndrome Evaluation (WISE) investigation. WISE participants completed a battery of assessments, including quantitative coronary angiography, CAD risk factor assessment, psychological testing, and measures of SES. In addition, participants were followed over a mean 2.1-year period for the occurrence of all-cause mortality.

METHOD

PARTICIPANT RECRUITMENT AND ENTRANCE CRITERIA

Women were eligible for participation in WISE if they were older than 18 years of age and were referred for a coronary angiogram to evaluate chest pain. Exclusion criteria included current pregnancy, cardiomyopathy, recent myocardial infarction or revascularization procedure (i.e., percutaneous transluminal coronary angioplasty or stenting [PTCA], coronary artery bypass graft [CABG]), a language barrier preventing questionnaire completion, and a history of congenital heart disease, among others (Bairey-Merz et al., 1999). The current

study population represents a total of 743 women who have complete data from the coronary angiogram, core diagnostic protocol, and documented SES information. A detailed description of the assessment procedures and objectives in WISE has been published elsewhere (Bairey-Merz et al., 1999).

PSYCHOSOCIAL & CAD RISK FACTOR ASSESSMENT

We assessed levels of depression (Beck Depression Inventory [BDI]) (Beck, 1978), cynical hostility (Cook-Medley Hostility Scale) (Cook & Medley, 1954), anger expression (Spielberger Anger Expression Scale) (Spielberger et al., 1985), and social network size (Social Network Scale) (Cohen, Doyle, Skoner, Rabin, & Gwaltney, 1997) using measures with demonstrated predictive validity for cardiovascular disease outcomes. The BDI assesses symptoms related to sadness, feelings of guilt, and perceptions of self-worth, among others, whereas the cynical hostility questionnaire measures a stable lack of trust and bitterness toward others. Anger-in and anger-out styles, respectively, assess a respondent's tendency to inhibit or repress signs of anger versus making outward (vocally or physically) displays of anger. Social network measures provide an indicator of the respondent's degree of social involvement and the number of friends and family in their social sphere.

We focused on relationships between SES variables and CAD risk factors as follows: (a) smoking status, rated as current smoker/current nonsmoker; (b) fasting high-density lipoprotein levels (HDL-C, less than/greater than 50 mg/dl); (c) fasting low-density lipoprotein levels (LDL-C, less than/greater than 160 mg/dl); (d) hypertension history; (e) body mass index (BMI) (less than/greater than 27.0) and waist-hip ratio; (f) functional capacity; and (g) diabetes history. The waist-hip ratio and functional capacity scores were maintained in continuous form in the analyses. Functional capacity score was assessed using the 12-item Duke Activity Status Index (DASI) assessing the patient's ability to perform a variety of activities ranging from basic self-care to strenuous exercise, and it is associated with physical fitness levels (Hlatky et al., 1989). The DASI is scored such that higher scores equal higher functional capacity. Patients taking cholesterol-lowering medi-

cations (approximately 20% of the sample) were excluded from analyses involving LDL-C and HDL-C endpoints.

Control variables included patient's age, menopausal status (a dichotomous yes/no variable), and use of postmenopausal hormone replacement therapy. We also assessed SES effects after controlling for baseline CAD severity as defined by each participant's quantitative angiogram findings. Controlling for race and marital status did not affect the interpretive outcomes of the described analyses.

SES MEASUREMENT

As part of the baseline self-report battery, participants responded to questions indicating their current annual household income, employment status, and education history. Income and education history served as the primary focus in this article. For the purpose of statistical testing, we categorized each of these SES markers into dichotomous variables including (a) high or low income, reflecting participants with an annual income below $20,000 versus those with an annual income exceeding this amount; and (b) high or low education, reflecting those with less than a high school or GED equivalency versus those reporting at least a high school education.

STATISTICAL ANALYSES

Preliminary analyses indicated that observed SES effects resulted primarily from the lowest scoring categories (less than high school education and less than $20,000 annual income, respectively) compared to all other participants, rather than forming a more linear relationship. Therefore, described analyses focused exclusively on these dichotomized SES variables. Occupation was also assessed, but the coding scheme resulted in too many complexities for analysis. Finally, we also explored an interaction variable combining the information from education and income, but this new variable did not improve the predictive models and is not presented.

We tested SES models predicting all-cause mortality over follow-up using Cox proportional hazard analyses. Covariate terms were force entered at step 1 of each model followed by SES predictor vari-

TABLE 1
CAD Risk Factors and Demographic Variables Among WISE Participants (*N* = 743) by Education and Income Groups

	Mean (SD)	*Low Income*	*High Income*	*Low Education*	*High Education*
Age (years)	59.6 (11.6)	59.5	59.5	63.6	58.6**
LDL-C (mg/dl)	114.3 (40.3)	114.4	114.8	118.8	114.0
HDL-C (mg/dl)	53.0 (12.8)	53.0	53.4	52.5	53.4
Current smokers (%)	19.7 25.3	16.8**	22.0	19.0	
Body mass index	30.2 (6.7)	31.2	29.6**	31.3	29.9*
Waist-hip ratio	0.87 (.11)	0.88	0.86**	0.91	0.86**
Functional capacity (DASI)[a]	19.5 (14.1)	14.6	22.3**	15.5	20.5**
History of hypertension (%)	58.5	66.7	53.5**	73.5	54.6**
Race: White/non-White (%)	80.4/19.6	67/33	87/13**	67/33	87/13**
Annual income > $20,000 (%)	64.2	—	—	33.8	72.0**
Completed high school (%)	79.8	62.4**	90**	—	—
Married (%)	60.5	35.0	74.6**	45.0	65.0**
With significant CAD[b] (%)	39.0	40.7	38.9	48.2	36.6*

NOTE: LDL = low-density lipoprotein levels; HDL = high-density lipoprotein levels; DASI = Duke Activity Status Index; CAD = coronary artery disease.
a. Range is 0 to 58.2.
b. Percentage with maximum coronary stenosis ≥ 50%.
*$p < .05$. **$p < .001$.

ables. Risk ratio point estimates and associated 95% confidence intervals are described. Power analyses, calculated using $\alpha = .05$ and a minimum n of 743, indicated that our probability to detect large effects (e.g., risk ratio values > 2.0) was greater than 99% and that power levels were smaller but also acceptable (> .80) for effects of moderate size (e.g., risk ratio values > 1.5).

RESULTS

CLINICAL CHARACTERISTICS

A description of the WISE cohort is provided in Table 1. Clinical characteristics are shown for the complete sample and broken down by education (less than high school education versus high school

diploma or greater) and income (less than $20,000 annual income versus greater than that amount) groups. An assessment of these values indicated a marked disparity toward poorer resources and heightened CAD risk for participants in the low-income and low-education groups, with these groups showing higher BMI scores and waist-hip ratios, hypertension history rates, smoking rates, and lower functional capacity levels.

In addition to the above, we further tested for differences on several additional factors proposed to account in part for low SES effects, including the presence of medical insurance, social network size, psychological distress (anger expression, depression, and hostility scores), and recent stress levels. The percentage of uninsured participants was substantially higher for both low-education and low-income groups (15.8 and 13.9%, respectively) compared to their more educated and wealthier peers (3.7 and 2.1%, respectively). Low SES participants also reported significantly fewer social contacts (means 18.5 and 18.1 for low education and income groups versus 20.5 and 21.2; p's < .001), higher cynical hostility (12.6 and 11.1 for low education and income groups versus 9.0 and 8.9, p's < .001), and depression scores (13.8 and 13.1 for low education and income groups versus 9.9 and 9.5, p's < .001). The low SES group did not differ with respect to self-reported stress levels from the preceding 5 years or on anger expression scores.

SES AND ALL-CAUSE MORTALITY

We observed a total of 45 deaths in the WISE sample (4.7%) over the mean 2.1 years of follow-up. Among the SES indicators, low-income status was associated with a greater than two and a half fold increase in all-cause mortality risk (RR = 2.6, 95% CI 1.4, 5.0). Education status, however, did not predict mortality outcomes ($p > .4$).

Table 2 illustrates the results of Cox regression equations in which we statistically adjusted for age, hormone replacement therapy use, and menopause status. Low income remained a reliable predictor, with adjusted risk ratio (RR) values comparable to those we observed for diabetes and hypertension history, and angiogram-based CAD

TABLE 2
Significant Independent Predictors of All-Cause Mortality Risk Following Adjustment for Age, Menopause Status, and Hormone Replacement Therapy Use

Predictor	*RR*	*95% CI values*
Diabetes history	2.8	(1.4, 5.2)
Hypertension history	3.0	(1.3, 7.0)
Stenosis > 50%[a]	2.8	(1.1, 7.2)
Annual income < $20,000	2.7	(1.4, 5.2)

a. Maximum stenosis value ≥ 50% in ≥ 1 coronary artery based on quantitative angiogram results.

severity. None of the other CAD risk factors, demographic variables, or psychosocial variables detailed in Table 1 were associated with mortality risk. Of note, inclusion of CAD severity as an additional covariate eliminated diabetes history as a significant predictor of all-cause mortality, but hypertension and stroke history and low income (adjusted RR = 2.3, 95% CI 1.2, 4.7) remained as independent predictors of all-cause mortality.

We subsequently explored the stability of the SES findings after controlling for differences in insurance coverage, psychological distress, and social network resources. The forced entrance of these factors into the regression hazard models actually increased the magnitude of the risk ratio point estimate for low-income effects although slightly attenuating the associated probability (RR = 5.9, 95% CI 1.2, 29.7, $p = .03$). Therefore, despite differences in medical care access, psychological distress, and social resources, low SES continued to have a substantive impact on the estimation of mortality.

Our final regression model tested the effects of low income after controlling for baseline covariates (age, menopause status, and use of hormone replacement therapy), the psychosocial variables from the previous model, and all CAD risk factors from Table 1 that differed significantly for low- and high-income groups (i.e., smoking, hypertension, BMI, and functional capacity levels). The inclusion of these additional covariates, however, had no effect on the predictive power of low income (adjusted RR = 6.9, $p = .04$).

DISCUSSION

This article described SES relationships (i.e., income and education) with CAD risk factors and prospective all-cause mortality in a sample of women with chest pain. Participants in the low-income group, defined by an annual income less than or equal to $20,000, were at greater than a two and a half fold all-cause mortality risk in comparison to wealthier participants, a finding that held true after controlling for factors such as age, menopause status, hormone replacement therapy, CAD risk factors, and baseline CAD severity based on quantitative angiogram findings. Mortality examinations with the WISE cohort are, at this stage, somewhat exploratory due to the early period of follow-up (mean 2.1 years) and low mortality incidence (4.7%). In the prediction of mortality outcomes, low SES and only a handful of the most robust biomedical variables, including a history of diabetes and hypertension and baseline CAD severity, offered predictive value after covariate adjustment.

Consistent with previous research (Cohen, 1999; Kaplan & Keil, 1993; Syme & Berkman, 1976), we observed a pattern of relationships between low SES categorization and elevated CAD risk factors in the form of higher smoking and hypertension rates, physical impairment levels, and BMI scores. These differences in risk factor classification held true for both education and income variables, but the relationships showed only a weak linear trend. Instead, the associations between SES and CAD risk factors observed here were largely differences between the lowest categories of SES membership in reference to all other participants. Although a number of previous investigations have reported more linear SES-health relationships (Marmot, Shipley, & Rose, 1984; Pincus, Callahan, & Burkhauser, 1987), this difference is likely a result of the smaller sample described in the WISE study, in which only the strongest finding—that of the poorest groups—was sufficiently powered.

The WISE protocol also included a number of psychosocial variables believed, in part, to explain SES-health associations (Baum et al., 1999; Gallo & Matthews, 1999; Taylor & Seeman, 1999). Increasingly, experts in the SES field are calling for longitudinal studies that more explicitly attempt to identify and test potential mechanisms, which largely fall into categories of chronic stress, differences

in health behavior patterns, and access to health care (Pickering, 1999). This list of potential variables is large, and their conceptualization(s) complex; no single study can hope to provide a comprehensive assessment. In the current study, we focused on measures of negative affect (depression, hostility, anger expression), social function (social networks and marriage), and health care access in the form of medical insurance status. The psychosocial measures used in WISE have shown strong relationships with CAD risk factors and disease severity in previous articles (Rutledge et al., 2001a, 2001b). Predictably, we observed large differences in these variables between our low and high SES groups, with low SES participants expressing greater distress, fewer social and interpersonal resources, and more difficulties receiving basic medical coverage. Statistically controlling for these differences, however, did not appreciably change the relationship between SES and all-cause mortality, indicating that other, unmeasured factors require further attention in understanding this association.

Notably, controlling for CAD risk factors in addition to the psychosocial mechanisms had little influence on the effects of low income in this sample. Although we observed large differences in low- and high-income groups in smoking behavior, BMI, hypertension history, and self-reported functional capacity, our analyses suggested that low income retained an independent effect on mortality. SES, in this article, was defined in terms of previous education experiences and income levels at the time of study participation. In practice, education, income, and occupation measures tend to show moderate but consistent interrelationships and are often used interchangeably as measures of SES. We did not attempt to collect historical income or occupation information regarding income or occupation from WISE participants, data that some researchers argue may be more valuable in light of the theory of SES-health relationships as an effect of gradual, lifelong accumulation (Gallo & Matthews, 1999).

Our inability to explain SES-mortality associations based on measured behavioral, psychosocial, and biomedical factors raises the question: What additional variables or methods are necessary in future research? These suggestions are many. The use of data analytic methods that are better suited to handling complex interactions between SES and mechanism variables, improved definitions of SES that incorporate multiple sources (e.g., combining education, income, and

occupation data into a single composite), further efforts to capture SES influences over the course of the lifetime—especially childhood SES information—and data providing insight into person- and community-level factors such as work environments, pollution exposure, and chronic stress are among the research directions advocated by leading SES researchers (Adler et al., 1994; Kaplan & Keil, 1993; Pickering, 1999; Taylor & Seeman, 1999). In the current article, we explicitly examined possible interaction effects between income and education, but this combination did not contribute additional insight above the separate SES measures.

LIMITATIONS

Unfortunately, the sample size and early stage of follow-up limit our ability to ask certain more detailed but equally interesting questions concerning possible race by SES interactions (Geronimus et al., 1996; Williams & Collins, 1995) and SES links to specific causes of death. The WISE sample, although recruited from several sites in the United States, is predominantly Caucasian, making it difficult to categorize an already modest percentage of African American participants into yet further categories of SES membership. Investigations beyond the veil of all-cause mortality, in contrast, will be possible in subsequent years as follow-up continues to accrue outcome data on WISE participants.

The primary objectives of WISE (Bairey-Merz et al., 1999) are to improve diagnostic procedures and decision making among women with chest pain or suspected myocardial ischemia. All participants were recruited on the basis of the presence of cardiac symptoms, although a large percentage proved to be free of significant CAD based on angiogram findings. The nature of the sample, therefore, limits our ability to generalize findings to the larger population of U.S. women, as the WISE cohort is higher than average on CAD risk factors, related somatic symptoms, and psychological distress. There is no data to suggest, however, that WISE recruitment was substantially influenced by SES factors, and we noted distributions of women across the spectrum of income, education, and occupation that were consistent with the targeted recruitment areas.

SUMMARY

The findings reported here reinforce the health risk associated with low SES in women and are consistent with the current emphasis in SES research to provide an increased focus on biobehavioral mechanisms that may be amenable to public health interventions. Low SES status, particularly low income, was associated with CAD risk factors and prospective mortality incidence over a mean 2.1 years of follow-up interval among women with suspected myocardial ischemia. Our attempts to identify potential explanatory mechanisms for these relationships indicated that SES was associated with a number of psychosocial, behavioral, and medical access variables, including higher levels of depression, hostility, and social isolation, smoking frequency, BMI, hypertension diagnosis rates, and lower levels of insurance coverage. The effects of low income, however, proved independent of these factors in predicting mortality risk, suggesting that a more detailed and comprehensive assessment of behavioral, psychosocial, and economic variables is necessary to understand the influence of SES on health.

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