Socioeconomic Indicators and Mortality from Coronary Heart Disease and Cancer: A 22-Year Follow-Up of Middle-Aged Men

ABSTRACT

Objectives. Data from the Western Collaborative Group Study were used to determine the extent to which the inverse association between socioeconomic status (SES) and mortality can be explained by risk factors for major causes of mortality.

Methods. The relation of education and income to subsequent mortality was studied in 3154 employed, middle-aged men over 22 years of follow-up.

Results. Over the follow-up period, 584 (18.5%) men died, 214 (6.8%) from coronary heart disease and 70 (2.2%) from lung cancer. A significant inverse association with systolic blood pressure, serum cholesterol, and smoking was found only for education. For education, adjustment for risk factors reduced the relative risk for coronary heart disease mortality from 1.80 (95% confidence interval = 1.33, 2.44) to 1.54 (1.13, 2.09), for lung cancer mortality from 1.60 (0.95, 2.70) to 1.38 (0.81, 2.34), and for all-cause mortality from 1.49 (1.09, 1.13) to 1.33 (1.12, 1.60). For income, adjustment for risk factors did not change relative risk for mortality from coronary heart disease (1.27 [0.97, 1.66]) and all causes (1.21 [1.03, 1.43]), but it did increase the relative risk for lung cancer mortality from 1.68 (1.05, 2.68) to 1.83 (1.13, 2.96).

Conclusions. In middle-aged, employed men, the association between SES and mortality is partially but not completely accounted for by major risk factors for mortality. (*Am J Public Health*. 1995;85:1231–1236)

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Introduction

The inverse association between socioeconomic status (SES) and mortality occurs consistently in epidemiology.[1-3] Excess mortality in low socioeconomic groups holds for different cultural groups,[4] different age groups,[2] and both sexes.[2,5,6] But while the strength and consistency of the association have apparent implications for public health policy, important questions concerning the association must first be addressed.

One question relates to the mechanism of the association: do socioeconomic factors operate through known biological and behavioral risk factors for mortality or through other risk factors not identified? Another question is whether socioeconomic factors represent a general susceptibility to disease, or whether their association with mortality is specific to certain diseases.

This article, based on results from a 22-year mortality follow-up of middle-aged men, investigates the role of biological and behavioral risk factors as mediators of the SES–mortality association, and elucidates the independent and combined effect of two socioeconomic indicators, income and education, across several mortality outcomes.

Methods

Subjects and SES Indicators

Analyses were based on data from the Western Collaborative Group Study,[7,8] a large prospective study of coronary heart disease initiated in 1960/61. A total of 3154 men aged 39 to 59 and free from coronary heart disease or other obvious health problems were recruited from 10 California firms in the San Francisco Bay and Los Angeles areas. The study population consisted of exclusively White men employed in different occupational settings such as the aircraft industry (30.0%), sales and banking (27.0%), the petrol industry (23.5%) construction (8.7%), and varied industrial settings (10.8%). At the baseline examination, the following risk factors for coronary heart disease were assessed: blood pressure, serum cholesterol, cigarette smoking, and type A behavior. Detailed information about the study population and risk factor assessments have been published in previous reports.[9]

Also assessed at the baseline examination were sociodemographic characteristics of study participants, including education and income. Education was registered as the highest level of schooling achieved (high school or less, some college, and graduated from college or higher) and was treated in the analysis as a binary variable (college vs no college education). Income, defined as the earnings in 1960/61, was recorded as one of five categories (< \$5000, \$5000 to \$10 000, \$10 000 to \$15 000, \$15 000 to \$25 000, and > \$25 000) and was reduced for analysis to two categories (< \$10 000 and ≥ \$10 000). A combined measure of SES was then constructed from income and education. SES code 1 refers to the men with less than \$10 000 income and no

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TABLE 1—Cumulative Mortality Risks of Men in the 1981/82 Western Collaborative Group Study Cohort after 22 Years of Follow-Up (n = 3154)

Follow-Up Status	No.	%
Died during follow-up, by cause of death	584	18.5
Coronary heart disease	214	6.8
Stroke	37	1.2
Non-lung cancer	186	5.9
Lung cancer	70	2.2
Other	77	2.4
Alive in 1982/83	2532	80.3
Unknown	38	1.2

TABLE 2—Social Class Indicator and Selected Risk Factors among Men in the Western Collaborative Group Study during 22-Year Follow-Up

	No.	%	Mean Systolic Blood Pressure, mmHg	Mean Serum Cholesterol, mg/100 mL	Mean No. Cigarettes Smoked per Day	Mean Height, cm	Mean Age, y
Education[a] (n = 3153)							
No college	1855	58.8	129.5	229.3	13.0	176.7	46.8
College	1298	41.2	127.3	222.2	9.5	177.9	45.4
P			<.0001	<.0001	<.0001	<.0001	<.0001
Income[b] (n = 3150)							
<$10 000	1391	44.2	128.9	227.3	11.9	176.3	45.8
≥$10 000	1759	55.8	128.4	225.6	11.4	177.9	46.6
P			.39	.25	.35	<.0002	<.0001
Socioeconomic status[c] (n = 3149)							
Code 1	1052	33.4	129.4	229.0	12.6	176.1	46.3
Code 2	1140	36.2	129.0	227.4	12.3	177.4	46.7
Code 3	957	30.4	127.4	222.1	9.6	178.2	45.7
P			<.005	<.01	<.0001	<.0005	<.0001

[a]Data on education was missing in one subject.
[b]Data on income was missing in four subjects.
[c]Code 1 = less than $10 000 and no college; code 2 = less than $10 000 and college or $10 000 or more and no college; code 3 = $10 000 or more and college.

college education; SES code 2 refers to the men in the middle of the social class distribution, where a college degree can substitute for higher income to some extent and vice versa (<$10 000 and college or ≥$10 000 and no college); and SES code 3 refers to men with more than $10 000 income and a college education. Five subjects were excluded from the analysis because of missing information for income and/or education.

At a follow-up of the original cohort in 1982/83, 22 years after the baseline examination, vital status was identified for all but 38 (1.2%) study participants. Cause of death was determined from death certificates according to the *International Classification of Diseases* (eighth revision).

Risk Factors and Analyses

Age, systolic blood pressure, and serum cholesterol were the biological risk factors studied because of their confirmed association with mortality in previous analyses of this dataset.[10] Other biological risk factors studied were height, which served as a proxy variable for early-life environmental factors such as nutrition,[11] and body weight. Behavioral risk factors studied were smoking and type A behavior. However, since type A behavior and body weight were not related to coronary heart disease mortality[8] or to any specific cause of mortality,[12] they were excluded from further analysis.

Statistical analysis was done with the SAS program and conducted in two steps.[13] The first step examined the mean of each risk factor at each level of both socioeconomic indicators (income and education) and the combined SES measure. A general linear model using the F test was used to assess the association between each indicator of SES and each biological risk variable.

The second step of the analysis examined mortality in terms of each SES variable using the Cox proportional hazards model.[14] The association between each measure of SES and each mortality outcome was examined. To evaluate the effect of each indicator as different biological risk variables were considered, two different models were fitted. In the first model, only age was entered without controlling for other variables. In the second model, systolic blood pressure, the mean number of cigarettes smoked, serum cholesterol, and height were added. The resulting relative risks for education, income, and the combined SES measure are reported, with the 95% confidence intervals for each variable. All tests of statistical significance in the study were two tailed.

Results

Mortality at Follow-Up

During the follow-up interval, 584 (18.5%) participants died, including 256 (8.1%) from cancer, 214 (6.8%) from coronary heart disease, and 37 (1.2%) from stroke. Among the cancer deaths, 70 (2.2%) were from lung cancer (Table 1). Again, only 38 (1.2%) participants were of unknown vital status in 1982/83.

Socioeconomic Indicators and Biological and Behavioral Risk Variables

The distribution of biological and behavioral risk factors for each socioeconomic indicator is given in Table 2. Education was inversely related to each biological risk factor ($P < .001$ in each case). Compared with college graduates, nongraduates had higher systolic blood pressure and higher mean serum cholesterol; they also smoked more cigarettes and were less tall and older.

Income was inversely related to systolic blood pressure, serum cholesterol, and smoking, but none of these trends was statistically significant. However, correlation of income with age and height was statistically significant.

Socioeconomic Indicators and Mortality

The age-adjusted mortality rates according to the socioeconomic categories showed an inverse gradient for each outcome (coronary heart disease, stroke,

TABLE 3—Age-Adjusted Mortality Rates per 1000 Person-Years, by Socioeconomic Categories, among Men in the Western Collaborative Group Study during 22-Year Follow-Up

			All Causes		Coronary Heart Disease		Stroke		Lung Cancer		Non-Lung Cancer	
	No.	Person-Years	No. Deaths	Mortality Rate	No. Deaths	Mortality Rate	No. Deaths	Mortality Rate	No. Deaths	Mortality Rate	No. Deaths	Mortality Rate
Education												
No college	1 855	34 273	406	11.73	157	4.35	29	1.43	50	1.34	73	2.53
College	1 298	25 255	178	7.79	57	2.34	8	0.61	20	0.81	43	2.22
P				<.0001		.0002		.04		.08		.67
Income												
<$10 000	1 391	26 082	269	11.06	102	3.91	16	1.17	38	1.45	51	2.57
≥$10 000	1 759	33 396	313	9.31	112	3.35	21	1.07	32	0.86	63	2.25
P				.03		.08		.81		.03		.51
Socioeconomic status[a]												
Code 1	1 052	19 516	225	12.21	88	4.61	14	1.47	31	1.57	45	2.63
Code 2	1 140	21 282	224	10.02	83	3.42	17	1.08	26	1.07	33	2.41
Code 3	957	18 660	133	7.88	43	2.44	6	0.76	13	0.70	36	2.12
P				<.0001		.0007		.15		.01		.48

[a]Code 1 = less than $10 000 and no college; code 2 = less than $10 000 and college or $10 000 or more and no college; code 3 = $10 000 or more and college.

TABLE 4—Adjusted Relative Risk (RR) and 95% Confidence Interval (CI) for Mortality from Selected Causes, by Socioeconomic Status, among Men in the Western Collaborative Group Study during 22-Year Follow-Up

Socioeconomic Indicators	Control Variables: 2 Models	RR (95% CI) All Causes	Coronary Heart Disease	Stroke	Lung Cancer	Non-Lung Cancer
Education: college/no college	Age	1.49 (1.09, 1.13)	1.80 (1.33, 2.44)	2.25 (1.03, 4.95)	1.60 (0.95, 2.70)	1.09 (0.75, 1.59)
	Systolic blood pressure Cholesterol Smoking Height	1.33 (1.12, 1.60)	1.54 (1.13, 2.09)	2.07 (0.94, 4.57)	1.38 (0.81, 2.34)	1.06 (0.72, 1.55)
Income: <$10 000/ ≥$10 000	Age	1.19 (1.01, 1.41)	1.27 (0.97, 1.66)	1.07 (0.56, 2.06)	1.68 (1.05, 2.69)	1.14 (0.79, 1.65)
	Systolic blood pressure Cholesterol Smoking Height	1.21 (1.03, 1.43)	1.27 (0.97, 1.66)	1.08 (0.56, 2.08)	1.83 (1.13, 2.96)	1.15 (0.79, 1.67)
Socioeconomic status (SES)[a]: first vs third level of the combined SES measure	Age	1.57 (1.26, 1.94)	1.89 (1.31, 2.73)	2.09 (0.80, 5.46)	2.20 (1.15, 4.21)	1.16 (0.75, 1.80)
	Systolic blood pressure Cholesterol Smoking Height	1.45 (1.17, 1.81)	1.67 (1.16, 2.41)	1.97 (0.75, 5.18)	2.08 (1.08, 3.99)	1.14 (0.73, 1.78)

[a]Code 1 = less than $10 000 and no college; code 2 = less than $10 000 and college or $10 000 or more and no college; code 3 = $10 000 or more and college.

lung cancer, non-lung cancer, and total mortality), with higher mortality at lower educational and income levels and at lower levels of the combined SES measure (Table 3). Results of the statistical analysis of SES and mortality using the Cox model are shown in Table 4.

Education. In the analysis adjusted for age, there was a statistically significant inverse association of SES with mortality from all causes, coronary heart disease, and stroke. Adjustment for other risk factors reduced the relative risk of death from all causes from 1.49 to 1.33, which was still significant ($P < .05$). Similarly, the age-adjusted relative risk of death from coronary heart disease was reduced from 1.80 to 1.54, which was also significant ($P < .05$). However, the addition of other risk factors reduced the age-adjusted risk of death from lung cancer from 1.60 to 1.38, which was not statistically significant, and reduced the age-adjusted risk of death from stroke from 2.25 to 2.07, which was also not significant. No significant association between death from non-lung cancer and education was found.

Income. In the first model, which included age as a control variable, income was significantly related to mortality from all causes (relative risk = 1.19) and lung cancer (relative risk = 1.68). There was a marginally nonsignificant association between income and risk of death from coronary heart disease (relative risk = 1.27). The association of income with non-lung cancer was small and not statistically significant.

When systolic blood pressure, serum cholesterol, cigarette smoking, and height were added to the model, the association was virtually unchanged for all mortality outcomes except lung cancer, for which it increased, and it was significant for all-cause mortality and lung cancer.

Combined income–education measure (SES). In the analysis adjusted for age, the SES indicator combining income and education was highly significantly associated with the relative risk of death from all causes, coronary heart disease, and lung cancer. When all risk factors were entered into the model, the relative risk of death from these three mortality outcomes was reduced but remained statistically significant (at least $P < .05$ for all). Independently of other risk factors, men at the lowest level of the combined three-level SES index had a 1.45-fold increased risk of death from all causes, a 1.67-fold increased risk of death from coronary heart disease, and a 2.08-fold increased risk of death from lung cancer compared with men in the highest SES category. For all three outcomes, the risk ratios were higher when the combined SES measurement was applied instead of education or income as single SES measurements for social class. Death from all cancers combined but excluding lung cancer was not related to the combined SES variable.

Discussion

Do Biological Risk Factors Mediate the SES–Mortality Association?

The first finding with bearing on this question is that individuals at lower income levels and with less education had higher levels of biological and behavioral risk factors for mortality, the one exception being that individuals with less income were also younger. These results are consistent with findings from other studies. Higher levels of risk factors for coronary heart disease mortality, such as systolic blood pressure and smoking, have been confirmed for lower occupational groups,[11,15–18] and education has been shown to be inversely related to smoking[19] and hypertension.[20] In two studies from the United Kingdom, however, and in contrast to our findings, cholesterol levels were higher in upper-class groups.[11,16] Data from US cohort studies are conflicting: some studies report no association between education and total cholesterol,[18] whereas others report a direct association[21] or confirm our findings of an inverse association between the two.[22]

The second finding was the observed change in the association between socioeconomic indicators and mortality when the biological and behavioral risk variables were controlled for in the analyses. For mortality from all causes, coronary heart disease, stroke, and lung cancer, a similar pattern was seen. When other risk factors were considered, the strength of the association between education and all-cause, coronary heart disease, and lung cancer mortality was reduced by between 8% and 15% for each mortality category. This reduction is similar to the one found by another US study.[18] For income, however, the association was unchanged for mortality from all causes, coronary heart disease, stroke, and non-lung cancer but increased for mortality from lung cancer. Thus, in this study, biological and behavioral risk factors beside age did not account for the income–mortality association.

Is SES Related to Specific Causes of Mortality?

Several authors[1,2] have suggested that SES factors are related to the entire range of disease, which would suggest that these factors influence the general susceptibility of the individual. The present study, in accordance with many others, shows an association of all three SES measures with mortality from all causes, coronary heart disease, and lung cancer. However, the small number of specific causes within other categories precludes any more specific analysis.

Residual Association

In the present study, education, income, and the combined SES index were associated with mortality even after accounting for known risk variables. This relationship was found regardless of the relatively crude indexing of socioeconomic variables and the relatively homogeneous and highly selected study group of exclusively White, employed, middle-class men. The residual associations for mortality from coronary heart disease, stroke, and lung cancer in particular were quite substantial. In the Whitehall study of civil servants, occupational status was a very strong and independent predictor of coronary heart disease, and the residual association was bigger than the contribution of all known risk factors.[23] In the British Regional Heart Study[16] and the three epidemiological studies of education and mortality from Chicago,[18] however, the residual association was about 50% smaller than that found in the Whitehall study, and similar residual associations have been found in studies from Scandinavia[24,25] and the Netherlands.[26]

It is important to note that the measurements of the different associations of interest—SES variables and outcome, risk factors and outcome, and SES variables and risk factors—are all influenced by measurement errors. Because these errors can result in a "regression dilution bias"[27] of these associations, the independent association between an SES variable and an outcome variable can be the product of residual confounding.

For example, the inaccuracy inherent in using single measurements of risk factors as proxy measures of lifetime exposure may produce the residual association. Baseline measurements for an individual may vary from one time to the next and may also be subject to errors in the measurement procedure. The effects of intraindividual variability and measurement errors on a one-time assessment of risk factors may lead to an underestimation of the association of these risk factors with an outcome variable and to an underassessment of their contribution as intervening variables to explain the SES mechanism. Such an underassessment would depend on the size of the regression dilution effect. Some analyses using data from other studies estimated this effect to be quite strong—as large as 60% for diastolic blood pressure and coronary heart disease mortality.[27] Although a previous analysis of the Western Collaborative Group Study, based on the 8.5-year coronary heart disease incidence data, indicated that the regression dilution effect was 5% for cholesterol and 10% for systolic blood pressure, the mediating effect of the biological variables may have been underassessed.[28] Another example relates to cigarette smoking as measured in this study. Cigarette smoking was measured simply in terms of number of cigarettes smoked per day. This measure ignores the type of cigarette smoked (e.g., filter vs nonfilter, high vs low nicotine) as well as the typology of the individual's smoking habit (e.g., extent of inhalation). Thus, the residual association of lung cancer and SES variables could partly be attributed to the inaccurate measurement of exposure to carcinogens from smoking.

On the other hand, imprecise measurements of SES can also cause an observed pattern of confounding to be overestimated.[29] Because various social class measurements are used, this could explain why some studies have failed to find an independent association of SES and mortality after controlling for biological risk factors.

An additional regression dilution effect may result from intraindividual changes in biological and behavioral risk variables over larger periods of time. Such changes (e.g., the lowering of blood pressure through treatment, the cessation of cigarette smoking) would attenuate the measured effect of these variables so that their contribution as intervening variables would again be underassessed. Previous analyses from the Western Collaborative Group Study for coronary heart disease mortality[10] indicate a reduction in the strength of association for cigarette smoking, but not for systolic blood pressure and cholesterol. This suggests that smoking in particular could account for a greater portion of the SES mortality association than the statistical analysis here indicates.

Another explanation of residual associations lies with the possibility that a change in biological risk factors over time is related to SES indicators. Three major studies from the United States[30–32] that covered nearly the same period of interest have shown the decline in mortality from coronary heart disease to be more rapid among men of higher SES. There is some evidence that this trend among better-educated men is partly owing to a more favorable change in their biological and behavioral (smoking) risk factor profile. However, while changes in smoking behavior are clearly important, the present study found that smoking rates of survivors at the 22-year follow-up were cut in half among both high school and college graduates. Thus, differential exposure over time probably did not affect our estimates.

Another explanation advanced to explain SES differences in mortality is health selection. However, this bias seems unlikely given that all subjects went through a rigorous examination at study entrance and only healthy individuals were allowed to enter the study.

Other Potential Risk Factors

Finally, it is possible that the unexplained relationship between social class and mortality may be partly attributable to factors that were not considered in this study. Based on analyses not presented in this report, we eliminated several variables that were measured in the Western Collaborative Group Study; these included type A behavior, body mass index, height (as a proxy variable for early-life environmental factors such as nutrition), job activity, and occupational exposure. Other potential risk factors that were not considered in this study are elevated fibrinogen level, elevated low-density lipoprotein cholesterol, and decreased high-density lipoprotein cholesterol.

We have speculated about these unconsidered risk factors. For example, in the Western Collaborative Group Study, individuals in the lower education/income groups were more typically employed in industrial settings with possible chemical exposure. Such exposure is more likely among low-salaried employees and may explain the association of lung cancer deaths with income. Another possibility is that the low income/education groups differ in psychosocial factors, such as job stress,[33] resources to control external demands,[34,35] or social support.[36,37]

Given these considerations, we are uncertain as to whether the biological and behavioral variables account for only a portion of the SES–mortality association or whether more could be explained with more precise measures of these variables. However, it is also possible that other factors may be involved, such as increased risks from occupational exposure.

Conclusions

This study suggests that the association between SES and mortality may operate at least partially through traditional risk factors. As a practical conclusion from these data obtained from employed middle-aged men, health promotion at the worksite should focus more on promoting lifestyle changes such as improved diet, controlled blood pressure, and especially reduced smoking among the less educated. Programs to this end seem justified, given that subjects of lower SES have higher mortality from diseases that are largely related to lifestyle (smoking) and to differences in biological and behavioral risk factors.

However, a portion of the inverse association between SES and major disease (coronary heart disease and lung cancer) outcomes was not explained by traditional risk variables in our model. Future studies should therefore include more refined measures of SES components and repeated measures of risk factor exposure. Additionally, research focusing on deprivation in early childhood, later risk for chronic disease, and further psychosocial influences on disease risk seems necessary. □

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