Explaining Reduced Cancer Survival among the Disadvantaged

HOWARD P. GREENWALD, EDGAR F. BORGATTA, RUTH McCORKLE, and NAYAK POLISSAR

University of Southern California; University of Washington; University of Pennsylvania; Mountain-Whisper-Light Statistical Consulting

SCIENTISTS AND CLINICIANS HAVE LONG RECOGnized that socially disadvantaged people have shorter life expectancies than the advantaged. Greater incidence of life-threatening illness among the disadvantaged and poorer chances of surviving them both contribute to this phenomenon. Unwillingness to accept continued excess mortality among the disadvantaged partly explains recurring movements in the United States to support universal access to health services. Through analysis of published work and illustrations from a new empirical study, this article is designed to help develop the basis for an improved explanation of excess mortality among socially disadvantaged people with cancer. An extremely prevalent, life-threatening, and expensive disease, cancer exemplifies the problems and issues generally encountered in health care delivery, costs, and outcomes.

For over a century, observers have reported that socially disadvantaged people experience lower cancer survival rates than the socially advantaged. But researchers have determined neither the actual dimensions of social differentiation that affect survival nor the reasons why

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such factors may affect mortality risk. Although race and socioeconomic status (SES)—typically defined as income, education, or a composite of these attributes—are strongly correlated, a single dimension of social differentiation may predominate in reducing or increasing survival rates. Alternatively, several dimensions of social differentiation may affect survival through a variety of mechanisms. The marked differences in treatment indications and prognosis among specific forms of cancer suggest that the actual causes of excess mortality depend on the type of malignancy involved.

Effective measures to reduce excess cancer mortality experienced by the disadvantaged depend on our comprehending the complex relations among social factors, individual behavior, disease processes, and medical interventions. The establishment of clear connections between income and survival would underscore the importance of economic barriers to health care. Evidence for links between educational level and survival independent of income would suggest a more complex, behavioral dimension, involving awareness of symptoms of serious illness, predisposition to use of health services, and feelings of self-efficacy. Determination that race or ethnicity played a role independent of income or education would introduce still more complexity into the posited causal chain between disadvantaged status and poorer survival prospects, suggesting the need to consider possible differences in treatment by health care providers of members of specific status groups, regardless of other attributes. These issues reflect concerns that extend beyond the financing and delivery of health care to basic dilemmas in American social life.

Current Understanding of Social Differences in Cancer Survival

Unanswered Questions

Studies published since the 1960s leave little doubt that *some* factor, or set of factors, associated with social disadvantage reduces the survival chances of cancer patients. Research published thus far has varied greatly in specific malignancies examined, number of observations accrued, dimensions of social differentiation, measures of SES, and sources of data. Although existing studies have produced evidence of an important social problem, they have provided neither empirically consistent findings

nor a comprehensive explanation of excess cancer mortality among the disadvantaged.

Most studies of how social forces affect cancer survival have focused on interracial differences. Comparisons of black and white Americans indicate striking distinctions. Relative five-year survival rates for all cancers diagnosed between 1977 and 1980 were 51 percent for white Americans, 39 percent for blacks. The survival advantage of whites over blacks increased during the 1980s, with 53 percent of whites diagnosed between 1981 and 1987 surviving five years or more, compared with 38 percent of blacks (Boring, Squires, and Tong 1992). Blacks face greater mortality risks than whites in most forms of cancer. Neither late-stage diagnosis nor delay in seeking care fully explains the survival differences (Ragland, Selvin, and Merrill 1991; Coates et al. 1992).

Researchers have periodically reported survival disadvantages among people with low SES. Early studies comparing indigent with nonindigent patient groups found that those in the lower SES categories had poorer prospects of survival (Linden 1969; Lipworth, Abelin, and Connelly 1970; Berg, Ross, and Latourette 1977). Later studies utilizing finer distinctions among SES levels and multivariate statistical models confirm these findings (Chirikos, Reiches, and Moeschberger 1984; Chirikos and Horner 1985; Greenwald 1992). Studies of patient populations that include very few minority group members (Chirikos and Horner 1985; Greenwald 1992) add weight to evidence from multivariate analysis that SES has an effect independent of race, at least on some malignancies.

The degree to which SES may account for interracial and interethnic differences in survival remains uncertain. Several important studies, all of which adjust for stage at diagnosis, suggest that race makes no difference in survival after SES has been controlled (Page and Kuntz 1980; Dayal, Polissar, and Dahlberg 1985; Bassett and Krieger 1986; Cella et al. 1991; Ansell et al. 1993). A widely cited report by the American Cancer Society (ACS) Subcommittee on Cancer in the Economically Disadvantaged concludes that ethnic differences (particularly white versus black) are probably secondary to socioeconomic factors (American Cancer Society 1986). But numerous studies, which include both racial and SES variables in statistical models, continue to suggest that race and ethnicity influence survival independently of SES (Wegner, Kolonel, and Nomura 1982; LeMarchand, Kolonel, and Nomura 1984; Vernon et al. 1985; Steinhorn et al. 1986; Dayal et al. 1987; Eley et al. 1994).

Harold Freeman, chairman of the ACS subcommittee that compiled the report, has observed that, at least in the case of some highly prevalent malignancies (viz. bladder and uterine corpus), an independent role for race in determining survival cannot be ruled out (Freeman 1989).

An absence of knowledge about intervening variables—for example, differences in treatment—that may link social disadvantages with adverse disease outcomes constitutes perhaps the most significant gap in current understanding. Studies have demonstrated that black women receive less aggressive interventions for breast cancer than whites (McWhorter and Mayer 1987) and that non-small-cell lung cancer patients lacking private medical insurance receive surgery less often than those with private medical insurance (Greenberg et al. 1988). But no investigation has yet explained adverse survival patterns among the disadvantaged on the basis of care received.

Methodological Limitations to Explaining Survival

Design features of the studies cited here limit their ability to support a comprehensive understanding. The methodological diversity of these investigations precludes ready synthesis of their findings, a problem that hinders our ability to determine the effects of SES. Bassett and Krieger (1986) and Eley et al. (1994) both study mortality in breast cancer, but they reach different conclusions about the relative importance of SES and race. These divergent findings may arise from methods used to measure SES. Bassett and Krieger employ a "working-class" versus "non-working-class" distinction, based on several census-block characteristics. Eley et al. utilize a "poverty index," combining household income and family size.

Methods for assessing SES and representing it in statistical models, furthermore, tend to be inadequate for determining the impact of distinct SES dimensions like income and education. Even studies that employ finely graded SES scales often utilize composite variables, including both income and education as predictors of survival (Wegner et al. 1982; LeMarchand, Kolonel, and Nomura 1984; Bassett and Krieger 1986). Studies utilizing more specific SES variables tend to report data on only one dimension of SES, making it impossible to determine whether other SES dimensions play an independent role.

An almost universal practice among the studies cited here of inferring SES measures from surrounding community characteristics (e.g., ZIP code or census tract) limits their ability to detect the effects of SES. This procedure recalls the well-known ecological fallacy, or the erroneous inference of individual characteristics from those of groups (Robinson 1950). Empirically, Greenwald et al. (1994) report that assessment of individual SES from aggregate data may lead to serious underestimation of the effects of SES on survival. These investigators report correlations, ranging from .33 to .40, between the individual's SES characteristics and aggregate indicators like median family income and percentage of high school graduates in his or her census tract. Given the error that these correlations imply in measurement of individual-level SES, researchers would require samples of about 1,500 cases to detect differences in mortality risk as high as 50 percent at conventionally accepted levels of statistical significance and power ($p < .05$, power = .80).

Many investigators report total observations lower than 1,500. In studies including several different malignancies, numbers actually used in statistical analyses may be considerably lower. Associated limitations in statistical power become most serious when the effects of race and SES on mortality risk are compared. Based on direct observation rather than inference from the aggregate, racial characteristics may appear to have stronger effects simply because they are more accurately measured.

Toward a Comprehensive Understanding

Focus on a limited number of studies (Wegner et al. 1982; LeMarchand, Kolonel, and Nomura 1984; Dayal, Polissar, and Dahlberg 1985; Steinhorn et al. 1986; Dayal et al. 1987) helps lay the groundwork for a better understanding of excess cancer mortality among the disadvantaged. These studies (summarized in table 1) assess the effects of race and SES within the same statistical models. Although all infer individual SES from surrounding community characteristics, they generally utilize numbers of observations capable of supporting detection of moderate to strong relations between SES and survival. Most important, these studies allow comparison of the effects of race and SES on different types of cancer, using identical methods of data collection and analysis.

Comparison of findings within individual studies indicates that specific social factors differ in their impact on survival in various forms of

TABLE 1
Findings from Selected Studies of Socioeconomic Status, Race, and Cancer Survival[a]

Citation	Database	Cancer site	SES and race variables	Risk ratios
Wegner et al. 1982	Hawaii Tumor Registry	Colon (N = 1,446)	SES (composite of income and education, census tract):	
			low–high	1.22
			med–high	1.04
			RACE:	
			Hawaiian–Japanese	1.56*
			Filipino–Japanese	1.47*
LeMarchand, Kolonel, & Nomura 1984	Hawaii Tumor Registry	Breast (N = 2,956)	SES (composite of income and education, census tract):	
			low–high	1.23
			med–high	.96
			low–med	1.28*
			RACE:	
			Filipino–Japanese	1.65*
			Hawaiian–Japanese	1.68*
			Filipino–Caucasian	1.61*
			Hawaiian–Caucasian	1.63*
			Hawaiian–Chinese	1.58*

Dayal, Polissar, & Dahlberg 1985	Selected comprehensive cancer centers	Prostate (N = 2,500)	SES (education, % high school graduates in census tract):	
			<25	1.86*
			25–49	1.50
			50–74	1.49
			75 or greater	1.00
			RACE:	
			Black–white	1.13
Dayal et al. 1987	Selected comprehensive cancer centers	Colon (N = 3,617)	SES (education, % high school graduates in census tract):	
			low	.97
			medium	.90
			high	1.00
			RACE:	
			Black–white	1.28*
Steinhorn et al. 1986	SEER, San Francisco/ Oakland, Detroit, Atlanta	Uterine corpus, adenocarcinoma (N = 5,018)	SES (income, education, census-tract level):	
			Median family income	
			<$10,000	1.33*
			$10,000 or greater	1.00

continued

TABLE 1 *continued*

Citation	Database	Cancer site	SES and race variables	Risk ratios
			Mean education	
			<high school	1.18
			high school or greater	1.00
			RACE:	
			Black–white	2.03*
Steinhorn et al. 1986	SEER, San Francisco/ Oakland, Detroit, Atlanta	Uterine corpus, sarcoma (N = 174)	SES	
			Median family income	
			<$10,000	0.83
			$10,000 or greater	1.00
			Mean education	
			<high school	1.86*
			high school or greater	1.00
			RACE:	
			Black–white	1.45

[a]To simplify and standardize presentation of mortality risk findings, only risk ratios are presented and indicated by an asterisk (*) as significant at least at the .05 level. Because many published articles present risk ratios with significance levels but no confidence intervals, only risk ratios are presented here. All risk ratios have been adjusted for the effects of stage at diagnosis.
*$p < .05$

cancer. Wegner et al. (1982) and LeMarchand, Kolonel, and Nomura (1984) utilize case records from the Hawaii Tumor Registry, combined with census-tract-level SES data, to study survival in colon and breast cancer. They report that an SES indicator combining income and education predicts survival in breast cancer even after race has explained all it can. Race, but not SES, however, predicts survival in colon cancer. Utilizing data from selected comprehensive cancer centers, Dayal, Polissar, and Dahlberg (1985) and Dayal et al. (1987) report that SES, but not race, predicts survival in prostate cancer, whereas race, but not SES, predicts survival in colon cancer; the latter finding confirms the work of Wegner et al. (1982). Steinhorn et al. (1986) utilize records combining information from population-based tumor registries and census tracts to study two types of cancer of the uterine corpus: adenocarcinoma and sarcoma. They report that both race and SES (income) predict survival in adenocarcinoma, but that SES, not race, predicts survival in sarcoma.

Evidence that race and SES vary across individual forms of cancer as independent predictors of mortality adds weight to the proposition that the characteristics of individual diseases condition the links between social factors and survival. Of course, the studies summarized in table 1 comprise a very limited range of malignancies. Like the literature in general, these studies provide little information about specific dimensions of SES and do not address the role treatment may play in connecting social factors and survival.

Focusing on specific dimensions of SES, the next section provides additional evidence that distinct social factors have varying effects on survival across malignancies. The data presented suggest that each social factor affects cancer survival by conditioning detection and treatment in a disease-specific manner.

SES and Cancer Survival: Two Detailed Examples

A population-based study of symptoms, treatment, and survival in cancers of the lung, prostate, pancreas, and uterine cervix provided data for a detailed examination of the effects of social factors in two specific malignancies. The study's location—King and Pierce Counties, Washington, where Seattle and Tacoma are located—encompasses a broad range of residential areas, including high-density urban to rural–

agricultural. After dropping cancers of the pancreas and uterine cervix from the analysis because of low initial accrual, the research team focused on non-small-cell lung and prostate cancer. Survival of all study patients was monitored for at least ten years.

Differences between non-small-cell lung and prostatic cancer make them useful for the purpose of identifying disease-specific predictors of survival. Many people with these diseases remain alive long after diagnosis. Often occurring at middle age, non-small-cell lung cancer is frequently cured through surgery if detected in its early stage (Lin and Ihde 1992). Although effective surgical treatment was available at the time of this study, reliable methods for early detection were not. Prostate cancer typically occurs in late adulthood, often developing slowly, and, even in the absence of treatment, allowing a high percentage of patients to survive until competing causes of mortality (e.g., heart disease) intervene. Digital rectal examination constituted a standard method for early detection at the time these patients were diagnosed. But treatment was highly controversial, as the medical literature failed to provide consistent evidence for the superiority of surgery, radiation, or multimodal interventions in extending life (de Kernion 1985).

Methods

Data. Between 1980 and 1982, the investigators collected data on patients 20 through 80 years of age newly diagnosed with the four cancers specified above. The researchers identified the patients through the Cancer Surveillance System (CSS), a population-based tumor registry maintained by the Fred Hutchinson Cancer Research Center in Seattle. Operating under contract with the National Cancer Institute's Surveillance, Epidemiology, and End Results (SEER) program, the CSS attempts to register all cancer cases that occur in a 13-county area in northwest Washington state within three months of diagnosis. Data were obtained from the CSS records themselves and from face-to-face interviews with patients.

The CSS constitutes a highly reliable means of identifying cancer patients and monitoring their survival. Shortly before this study was conducted, Smith, Francis, and Polissar (1980) determined that the registry missed less than 2.5 percent of malignancies incident in its catchment area. The National Cancer Institute requires the registry to

update the status of all living patients within 18 months of diagnosis or last contact (Young, Percy, and Asire 1981) by contacting the hospitals at which patients were originally treated and the physicians who cared for them (Horm, Asire, and Young 1984) and reviewing state death certificate files (Riles, Pollack, and Young 1983).

The investigators monitored the CSS and interviewed patients until prespecified quotas for the four cancers were filled. A total of 877 living individuals were identified during an 18-month accrual period. Human subjects considerations required obtaining permission from each potential subject's physician before requesting an interview. Permission was granted to contact 599 of these patients, of whom 536 agreed to interviews and provided sufficient data to be included in some part of the analysis. Of those interviewed, 260 had lung cancer; 201, prostatic cancer; 25, cancer of the pancreas; and 50, cancer of the cervix.

In addition to vital status, the CSS file provided basic data on age (years at time of diagnosis), histology, stage, date of diagnosis, gender, and location of residence. Stage was coded as I (localized), II (regionally disseminated), and III (distantly metastasized) according to CSS schema establishing these summary designations on the basis of site-specific clinical staging systems in widespread use (Fritz and Roffers 1991). Histology was coded according to the International Classification of Diseases for Oncology (ICD-O) (World Health Organization 1976). With respect to gender, males were coded 1, females 2. Face-to-face interviews were conducted utilizing an extensive questionnaire with closed-ended items on income (family income in the year preceding diagnosis), education (years of formal schooling), race and ethnicity (self-described), therapy received, and functional status according to a collapsed version of the Karnofsky scale (Karnofsky and Burchenal 1949), which classifies patients according to ten categories ranging from fully functional to dead.

The researchers carried out two procedures to assess possible bias in the sample of cancer patients obtained. First, they asked physicians who withheld permission to contact specific patients about their reasons for refusal. Almost always, the physicians indicated that these patients were moribund or incapable of answering interview questions for other reasons. Second, at the midpoint of the accrual period, the investigators compared patients they had succeeded in interviewing with records of all patients with the same diagnoses entered in the CSS since the beginning of the study. Patients who were interviewed were more likely to

have received surgery than those not interviewed, but they did not differ in age, race, and marital status.

Lung cancer cases analyzed below (N = 125) included only persons aged 75 and under with non-small-cell disease, stages I or II. Patients with small-cell lung cancer and stage III non-small-cell lung cancer were omitted from the analysis because of very high short-term mortality rates. Non-small-cell lung cancer patients over 75 years old were omitted because of the possibility that omission of curative surgery because of age alone might confound effects of SES. Prostate cancer cases analyzed (N = 200) included all those accrued except for one case dropped owing to an error in diagnostic information. Survival of all patients was monitored until mid-1993.

Statistical Methods. The investigators used the Cox proportional hazards model to assess effects of specific SES dimensions on mortality risk. They used logistic regression and cumulative logistic analysis to predict stage at detection in lung and prostate cancer, respectively, and they relied on logistic regression to predict receipt of surgery and radiation therapy. Throughout the analysis, separate models were developed for non-small-cell lung and prostate cancer. Cases with missing data on any variable to be included in a specific equation were dropped, resulting in an estimation based on variable numbers of observations (95–120 for lung cancer; 157–196 for prostate cancer).

Findings

Table 2 displays means and standard errors for the variables included in the analysis below, plus five-year survival rates for the non-small-cell lung cancer (stages I and II) and prostate cancer patients followed in this study. Cancer stage (stage at diagnosis) is represented as a series of dichotomous variables, coded 0 or 1 as appropriate for each observation.

Table 3 presents coefficients from Cox proportional hazards models of mortality risk. The variables represent age, sex (lung cancer only), and stage, plus income and education in separate equations. In the estimation of these and subsequent models, income was coded in increments of $5,000. Coefficients on dichotomous variables representing stage indicate the effects of diagnosis in stages II and III compared with stage I, the reference category in the models.

Among patients with non-small-cell lung cancer, greater income predicts decreased mortality hazard at the .01 level of significance, whereas

TABLE 2
Selected Characteristics of Subjects by Cancer Diagnosis[a]

Characteristics	Non-small-cell lung	Prostate
Mean age (years)	59.9 (0.80)	68.0 (5.18)
Mean income (thousands of dollars)	24.2 (1.39)	28.3 (1.21)
Mean education (years)	12.2 (0.24)	12.7 (0.26)
Sex (percent male)	51.2 (4.49)	100.0 (0)
Treatment (percent receiving each modality)		
Surgery	84.8 (3.22)	47.5 (3.54)
Radiation	38.2 (4.65)	37.9 (3.69)
Cancer stage (percent diagnosed in each stage)		
I	43.2 (4.45)	55.4 (3.67)
II	56.8 (4.45)	16.8 (2.77)
III	—	27.7 (3.31)
Mean survival (months after diagnosis)	57.9 (4.77)	89.8 (3.36)
Five-year survival (percent surviving)	39.0 (4.42)	65.8 (3.40)

[a]Standard errors are in parentheses.

years of education is not a significant predictor of mortality risk. An equation that included both income and education was also run but not presented here. In this equation, education was not a statistically significant predictor of mortality risk, but income predicted decreased mortality more strongly than in the equation summarized in table 3 (coefficient = −.137, SE = .049).

Among patients with prostate cancer, additional years of education rather than greater income appear to reduce mortality risk by a statistically significant margin ($p < .01$). Again, an additional equation was run that included both income and education. In this equation, income was not a statistically significant predictor of mortality risk, but education predicted decreased mortality more strongly than the equation summarized in table 3 (coefficient = −.076, SE = .027).

Because the equations in table 3 include stage, this variable cannot have fully accounted for SES-related differences in mortality risk. But stage may still provide a partial explanation. To explore the possibility that stage serves as an intervening variable between specific SES dimensions and survival, the investigators fitted logistic regression models to

TABLE 3
Socioeconomic Status Variables and Relative Mortality Risk:
Proportional Hazards Models for Survival

Variable/risk	Coeff	SE	Risk ratio	*p* value
Non-small-cell lung cancer				
SES variable: income				
Income	−0.129	0.046	0.879	<.01
Age	0.006	0.014	0.674	ns
Sex	−0.378	0.241	0.685	ns
Cancer stage:				
I	—	—	1.0	
II	0.944	0.243	2.569	<.01
SES variable: education				
Education	−0.012	0.038	0.988	ns
Age	0.009	0.013	1.009	ns
Sex	−0.143	0.212	0.866	ns
Cancer stage:				
I	—	—	1.0	
II	0.887	0.226	2.427	<.01
Prostate cancer				
SES variable: income				
Income	−0.012	0.034	0.988	ns
Age	0.032	0.016	1.032	ns
Cancer stage:				
I	—	—	1.0	
II	0.618	0.259	1.854	<.05
III	1.333	0.209	3.796	<.01
SES variable: education				
Education	−0.069	0.025	0.933	<.01
Age	0.025	0.014	1.025	ns
Cancer stage:				
I	—	—	1.0	
II	0.551	0.253	1.734	<.05
III	1.260	0.205	3.524	<.01

Abbreviations: coeff, coefficient; ns, nonsignificant; SE, standard error.

predict stage in lung cancer (represented here as a dichotomous variable) and cumulative logistic models to predict stage in prostate cancer (represented as a three-level ordinal variable). Results appear in table 4. No statistically significant relation was detected between SES and stage for lung cancer. But in prostate cancer, education predicted lower stage detection. A statistically significant relation was not detected between income and stage in this disease.

Although education predicts stage at detection in prostate cancer, stage does not explain a major part of the relation between education and mortality risk. In a Cox model that included only age and education, the coefficient on education was −.074 (SE = .023), a magnitude only slightly larger than that presented in table 3.

To assess the possibility that treatment serves as an intervening variable between SES and survival, the investigators performed logistic regression analysis to predict treatment on the basis of SES. Table 5 presents findings from this analysis. In the models represented here, dependent variables were coded 1 if the therapy was received, 0 if not. Among non-small cell lung cancer patients, higher income is positively

TABLE 4
Socioeconomic Status and Cancer Stage: Coefficients from Logistic Regression and Cumulative Logistic Analysis[a]

Predictor	Non-small-cell lung[b]		Prostate[b]	
Income	−.060	—	−.065	—
	(0.069)		(0.051)	
Education	—	−.029	—	−.115
		(0.070)		(0.043)**
Age	.015	.016	−.015	−.021
	(0.023)	(0.022)	(0.023)	(0.020)
Sex	−.798*	−.638	—	—
	(0.406)	(0.375)		

[a]This table presents coefficients from logistic regression for lung cancer and cumulative logistic analysis for prostate cancer. In the LOGIST module of the SAS system utilized for estimation, logistic regression is used to model binary dependent variables (such as the stage variable for lung cancer used here) and cumulative logistic analysis is used for ordinal variables (such as the three-level stage variable for prostate cancer presented here).
[b]Standard errors are in parentheses.
*$p < .05$; **$p < .01$.
Source: SAS Institute (1990).

TABLE 5
Socioeconomic Status and Cancer Treatment: Logistic Regression Coefficients[a]

Predictor	Surgery		Radiation	
Non-small-cell lung				
Income	.133 (0.107)	—	−.022 (0.091)	—
Education	—	.013 (0.097)	—	−.063 (0.089)
Age	.010 (0.031)	.009 (0.029)	−.050 (0.032)	−.039 (0.029)
Sex	.659 (0.578)	.590 (0.550)	−.404 (0.517)	−.330 (0.476)
Cancer stage:				
I	—	—	—	—
II	−1.656* (0.674)	−1.546* (0.666)	2.313** (0.578)	2.449** (0.559)
Prostate				
Income	.017 (0.052)	—	.198** (0.061)	—
Education	—	−.092* (0.045)	—	.102* (0.049)
Age	−.050* (0.024)	−.058* (0.022)	.018 (0.028)	−.008 (0.025)
Cancer stage:				
I	—	—	—	—
II	−.275 (0.434)	−.226 (0.424)	.080 (0.490)	.239 (0.458)
III	.075 (0.358)	−.107 (0.361)	−1.448** (0.481)	−1.419** (0.469)

[a]Standard errors are in parentheses.
*$p < .05$; **$p < .01$.

related to receiving surgery and negatively related to receiving radiation therapy. While income is the stronger predictor of treatment in non-small-cell lung cancer, none of the relations estimated are statistically significant. Among those with prostatic cancer, income predicts an increased likelihood of receiving surgery and radiation, whereas education negatively predicts receipt of surgery and positively predicts radiation therapy. The relations of income to radiation and education to surgery and radiation are statistically significant.

The researchers then assessed the possibility that treatment differences explained effects of specific dimensions of SES on survival by adding treatment variables to the models presented in table 3. In table 6, the variable of surgical treatment is added to the model predicting survival in non-small-cell lung cancer in table 3. Individuals who received surgery were less than one-third as likely to die in any given month after diagnosis than those who did not receive surgery. The coefficient on income is more than one-third lower than in table 3, and is no longer a statistically significant predictor of mortality.

Table 7 represents a similar analysis for prostate cancer, presenting the Cox models in table 3 with the addition of treatment variables. Addition of treatment variables did not reduce the coefficient on education, the SES dimension found to affect survival in prostate cancer, as presented in table 3. Magnitudes on the education variable in table 7 were slightly greater than in table 3.

TABLE 6

Socioeconomic Status and Relative Mortality Risk for Non-Small-Cell Lung Cancer: Proportional Hazards Model Including Treatment (Surgery)

Predictor	Coeff	SE	Risk ratio	*p* value
Income	−0.087	0.046	0.916	ns
Age	0.016	0.015	1.016	ns
Sex	−0.132	0.250	0.876	ns
Surgery	−1.273	0.325	0.280	<.01
Cancer stage:				
I	—	—	1.0	
II	0.784	0.251	2.191	<.01

Abbreviations: see table 3.

TABLE 7
Socioeconomic Status and Relative Mortality Risk for Prostatic Cancer: Proportional Hazards Model Including Treatment

Predictor	Coeff	SE	Risk ratio	*p* value
Treatment variable: surgery				
Education	−0.073	0.026	0.930	<.01
Age	0.023	0.014	1.023	ns
Surgery	−0.131	0.184	0.877	ns
Cancer stage:				
I	—	—	1.0	
II	0.557	0.253	1.745	<.05
III	1.247	0.206	3.479	<.01
Treatment variable: radiation				
Education	−0.087	0.027	0.917	<.01
Age	0.031	0.015	1.031	<.05
Radiation	0.051	0.215	1.052	ns
Cancer stage:				
I	—	—	1.0	
II	0.716	0.277	2.046	<.01
III	1.498	0.224	4.473	<.01

Abbreviations: see table 3.

The investigators estimated a separate set of models in prostate cancer, focusing on a treatment variable representing receipt of radiation only. In logistic regression equations, education predicted the receipt of only radiation at the .05 level of significance (coefficient = .154, SE = .066). When added to the survival model for prostate cancer in table 3, "only radiation" slightly reduced the relation of education to mortality (coefficient = −.065, SE = .026).

Findings presented in tables 3 to 7 are robust across level of functional status, rural and urban location, and age group. Addition of scores from the Karnofsky scale to our equations produced very little change in the magnitudes of the coefficients. Coefficients remained essentially unchanged when a variable indicating residence in an urban versus rural census tract—itself not a predictor of stage, treatment, or survival—was added to the equations. In equations predicting prostate cancer mortal-

ity risk, coefficients on term representing interaction between education and age were higher, but consistently negative, in the younger age groups.

Reestimation utilizing only cases with no missing values on any variable specified in table 2 yielded highly similar results. Coefficients from Cox proportional hazards models identical to those presented in table 3 for lung cancer (N = 95) were −.128 (SE = .047) on income and −.045 (SE = .043) on education. Coefficients from these models for prostate cancer (N = 157) were −.014 (SE = .036) on income and −.091 (SE = .027) on education. Neither income nor education predicted stage in lung cancer at a statistically significant level; education (coefficient = −.117, SE = .046), but not income (coefficient = −.082, S.E. = .054), predicted stage in prostate cancer. Relations between SES indicators and treatment variables were highly similar to those specified in table 5.

Measures of SES other than those utilized here produced much weaker results. The investigators substituted median home value in each patient's census tract according to the 1980 U.S. Census, and occupational status according to the Nam–Powers measures (Powers 1982) for income and education in the equations represented above. Almost none of the resulting coefficients were statistically significant.

There were too few members of racial minority groups in this study to include race as a variable of primary interest. Of the 536 cases originally collected, only 28 were racial minorities: six of these were African American, and the others were divided among a large number of groups. Race neither predicted survival in equations similar to those presented above nor explained relations between SES and variables indicating detection, treatment, or survival.

Discussion

Focusing on individual dimensions of SES, the findings reported here are consistent with implications of earlier studies that specific social factors affect survival differently across malignancies. In addition, these findings suggest that the mechanisms responsible for these effects vary among different forms of cancer.

Findings on non-small-cell lung cancer are consistent with an "economic" model of survivorship. Income directly predicts survival. The

strength of the relation between income and survival decreases when surgery, the treatment associated with curative intent, is controlled. This decrease suggests that treatment may serve as a mechanism through which income influences survival, although the relation between income and surgery itself is not statistically significant.

Compared with the findings on lung cancer, those on prostate cancer are more consistent with a model emphasizing cognition and behavior. The observation that education, but not income, predicts early stage detection suggests the importance of "predisposing" rather than "enabling" factors in prostatic cancer survival. People with relatively advanced education are more predisposed to pursue lifestyles and behavior patterns—like seeing physicians for examinations in the absence of symptoms and responding promptly to illness—that increase the likelihood of early detection.

Interactions among pathophysiology, detection, treatment-related technology, and individual behavior offer explanations of the variations in the impact of specific SES dimensions across cancers. Often effective, treatment for early non-small-cell lung cancer may mediate between SES and survival among the patients studied here. It is not surprising, on the other hand, that cancer-specific treatment explains a smaller part of the relation between SES and survival in prostate cancer. When the patients studied here were diagnosed, treatment of prostate cancer was (and remains today) a subject of medical controversy. Recent research on long-term survival of prostate cancer continues to cast doubt on the benefits of early detection and treatment (Johansson, Adami, and Andersson 1992).

Interaction between disease characteristics (including patterns of comorbidity) and individual behavior may help determine the relation between specific SES dimensions and cancer detection. Urinary symptoms among men with benign prostatic hypertrophy may cause them to seek medical intervention and obtain early-stage diagnosis of prostate cancer incidental to treatment. In lung cancer there are typically no distressing early symptoms and no symptom-producing, nonmalignant diseases as common and likely to lead to early-stage diagnosis as benign prostatic hypertrophy. Education, a variable closely related to prompt response to symptoms, would be more likely to explain early detection of prostate than of lung cancer.

The empirical findings presented here extend earlier understanding by suggesting alternative, disease-specific models as guidelines for con-

tinuing research. Unfortunately, these findings provide no concrete information on the thinking, actions, or options available to individuals that actually link SES with survival. Although this discussion raises the possibility that income contributes to lung cancer survival through enhancement of access to care, income may in fact reflect several interrelated factors like wealth, insurance status, and occupation. Similarly, a diffuse set of properties corresponding to years of education, like favorable health behavior, compliance with medical regimens, and self-care, may best explain survival in prostatic cancer. It must be acknowledged that the sample we have studied tends to omit the most critically ill individuals, and may thus under- or overrepresent relations among variables that prevail within the general population of lung and prostate cancer patients.

The findings on SES reported here are useful when viewed alongside earlier work on race. The impression that racial effects differ across cancer sites receives support from the observation of differences across sites in the effects of specific SES dimensions. Findings on SES suggest that mechanisms responsible for poorer survival among minorities may also vary across cancer sites. Such mechanisms appear likely to be even more complex than those related to SES, involving, for example, possible variations in provider behavior toward specific racial groups.

Support for the proposed alternative models through continuing studies would have mixed implications for reducing survival differences between advantaged and disadvantaged people in cancer and other serious diseases. Applicability of both models across a broad range of diseases would imply that increasing access to health care would not alone equalize outcomes. Acknowledgment of this limitation does not diminish the importance of basic access to health services. But those concerned with improving outcomes for the disadvantaged must also acknowledge the need for promoting healthy behavior, awareness of symptoms, informed consumer choice of treatment options, compliance, and self-care. Significantly improved access would not eliminate the need for outreach and monitoring efforts focused on major public health risks.

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Address correspondence to: Howard P. Greenwald, Ph.D., Sacramento Center, University of Southern California, 1201 J Street, Sacramento, CA 95814-2919. (E-mail: Greenwa@mizar.usc.edu)