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Cancer differentials among US blacks and whites: quantitative estimates of socioeconomic-related risks

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CANCER DIFFERENTIALS AMONG US BLACKS AND WHITES: QUANTITATIVE ESTIMATES OF SOCIOECONOMIC-RELATED RISKS

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This article analyzes 10 studies that assessed the association of socioeconomic status (SES) with cancer occurrence among blacks and whites in the United States. The following summative inferences were made: the associations of SES with cancer are similar among blacks and whites; cancers of organ sites with the most intimate environmental interfaces have the strongest SES-cancer associations (stomach, lung, cervix, and rectum); the prevalence of exposure to low socioeconomic-related risks such as poverty are approximately fourfold greater among blacks; the all-site population attributable risk percent due to low socioeconomic exposure among blacks is estimated to be four times that of whites, and similar data trends were observed for individual cancer sites such as the stomach and lung; and the three cancer sites of the stomach, lung, and cervix uteri account for nearly half of the observed US black-white cancer rate difference. This review also found all 10 of the primary studies in this field to be ecological with respect to socioeconomic exposure measurement, ie, they used aggregate measures (eg, census tract median education or family income) to characterize the individual's exposure. The need for direct empirical validation of such measures to aid in interpretation of the extant data in this field is underscored. (*J Natl Med Assoc.* 1994;86:209-215.)

Key words • socioeconomic status • cancer rates • race • blacks

Reviews of all-cause and cause-specific mortality differentials have demonstrated clearly the relative disadvantaged status of blacks compared with whites in the United States.[1,2] These differences have been observed across the life span from infancy to later life and include the most prevalent morbid and mortal outcomes: cancer, heart disease, and stroke. Racial group incidence, mortality, and survival disparities consistent with a black disadvantage have been observed for all cancer sites combined as well as for numerous specific cancer sites.[3-6] In fact, the gap has been widening between racial groups over the last 50 years. Socioeconomic status (SES), however, also is associated with both cancer occurrence and racial group status in the United States.[7-10] The analytic picture is further complicated by the wealth of epidemiologic evidence for an array of cancer risk factors (eg, dietary, lifestyle, family history, psychosocial, environmental/occupational exposures, stress, etc) that may be associated with both racial group and SES.[11-15] This review offers a perspective on the interrelationship of race, SES, and cancer by critically summarizing the results of 10 studies.

CONCEPTUAL/OPERATIONAL DEFINITION OF RACE AND SES

Five reviews/editorials have provided compelling arguments against the use of race as a biologic construct in epidemiologic research.[16-20] The preponderance of evidence establishes and justifies the use of race as a social rather than as a biologic construct. Indeed, less than 10% of racial group genetic diversity may be

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accounted for by between-race variability, while the remainder accounted for by within-race and between-geographic regional variance. Furthermore, the vast majority of genes from the original premigration pools (eg, Africa and Europe) were not unique to either, and recent evidence suggests that in the United States, even those few alleles that were unique to the original African or European gene pools are now approximately 25% admixed. These two populations, ie, US blacks and whites, are very likely to become more genetically similar with each passing generation.[21,22]

This phenomenon, taken together with the probable polygenetic-environmental control of cancer occurrence and the already noted widening between racial group cancer differential in the United States, strongly suggests a predominantly environmental explanation of cancer differences by race. This review's paradigmatic orientation to the race variable also draws on the recent findings of a review of the use of "race" among studies reported in the *American Journal of Epidemiology* [23]:

> Exposure to social risks are experienced differentially by racial groups in a race-conscious society. This social conceptualization of race then suggests environmental, rather than genetic, causes of disease when racial group differences are observed.

The associations of social class or SES with a variety of health outcomes among studies of the populations of industrialized countries have been observed to be robust to the operational measure of SES used—income, education, or occupation.[24,25] Consequently, we will broadly review studies that incorporated any such SES measures. As for analytic procedures, nearly all modern analyses "control for" social class.[25] A review of the *American Journal of Epidemiology* (1982 through 1985) found that of the chronic disease studies that used some measure of SES, nearly half (42%) incorporated such variables as confounders only.[26]

It has been suggested that adjustment for SES may nearly abolish any observed black-white racial group differences for all-cause mortality and all-cancer incidence/mortality, as well as for site-specific cancer incidence/mortality.[8,10,27,28] Socioeconomic-related variables have been found to account for substantial proportions or nearly all of the observed black-white differentials. From a purely etiologic perspective, such adjustment for "confounding" may diminish our ability to explain group differences, and thus, to effectively intervene. Such an analytic strategy, although theoretically sound, may be misleading from a public health perspective. In the real world, differences exist between the races. For example, the prevalence of poverty among US blacks is approximately threefold the white rate,[15,29,30] and while these differences may be "controlled for" in the abstract world of statistical constructs, such strategies may result in the loss of useful information.

This review examines the relationship between SES and cancer among both blacks and whites. Race will be treated as a potential effect modifier, and neither SES nor race will be controlled for, but rather, described as fully as the body of research will allow. Our concern was with the relationship between SES and cancer among both blacks and whites in the United States. Consistent with the theoretical underpinnings of this study, it was hypothesized that: 1) SES acts similarly on cancer among both blacks and whites, ie, the SES-cancer rate ratio (RR) is the same in both populations, 2) the prevalence of risk exposure (ie, to disadvantaged or low SES) is significantly greater among blacks, and 3) the population attributable risk percent (PAR%) of cancer due to SES is greater among blacks compared with whites in the United States.

METHOD

Sampling

An initial group of studies, potentially relevant to the question of the association between SES and cancer occurrence among both blacks and whites, was retrieved through computer searches of the following databases (1960 through 1992): Medline, Catline, Sociological, Social Work and Psychological Abstracts, Dissertation Abstracts International, and government document indices (Monthly Catalog, ASI, SRI, NTIS, and CIS). A maximally broad keyword scheme was used (cancer or neoplasm; racial stocks, blacks, or minority groups; and socioeconomic status or social class). Computer searches then were augmented with a bibliographic review of retrieved manuscripts. Ten manuscripts were collected that met the population (ie, United States), relational (ie, black or nonwhite versus white), constructual (ie, SES-cancer relation among both blacks and whites), and statistical (ie, effect sizes such as the rate ratio [RR], odds ratio [OR], or prevalence ratio [PR] reported, or could be estimated from the reported data) demands of the central review question. In short, 10 independent studies were retrieved that included both black and white samples, and rather than control for SES, they reported the strength of the SES-cancer association for both black and white samples. These 10 studies are the database for this review's analysis (Table 1).

TABLE 1. 10 STUDIES REPORTING THE STRENGTH OF THE SES-CANCER ASSOCIATION FOR BOTH BLACK AND WHITE SAMPLES

Authors	Title	Year	Publication
Baquet, Horm, Gibbs, & Greenwald	Socioeconomic factors and cancer incidence among blacks and whites	1991	*J Natl Cancer Inst*
Blot & Fraumeni	Geographic patterns of lung cancer: industrial correlations	1976	*Am J Epidemiol*
Boring, Squires, & Heath	Cancer statistics for African Americans	1992	*CA Cancer J Clin*
Devesa & Diamond	Association of breast cancer and cervical cancer incidences with income and education among whites and blacks	1980	*J Natl Cancer Inst*
Devesa & Diamond	Socioeconomic and racial differences in lung cancer incidence	1983	*Am J Epidemiol*
Ernster, Selvin, Sacks, Austin, Brown, & Winkelstein	Prostatic cancer: mortality and incidence rates by race and social class	1978	*Am J Epidemiol*
Ernster, Winkelstein, Selvin, Brown, Sacks, Austin, et al	Race, socioeconomic status and prostatic cancer	1977	*Cancer Treat Rep*
Levin, Connelly, & Devesa	Demographic characteristics of cancer of the pancreas: mortality, incidence, and survival	1981	*Cancer*
Miller & Chapman	Reviewing cancer in American blacks: a Baltimore study	1981	*J Natl Med Assoc*
Wright, Bernstein, Peters, Garabrant, & Mack	Adenocarcinoma of the stomach and exposure to occupational dust	1988	*Am J Epidemiol*

Sample Restriction

The present study is centrally concerned with the socioeconomic explanation of black-white cancer risk differences among sites where disease occurrence is greater for blacks. The data displayed in Table 2 suggest black predominance for all sites combined and for 10 specific cancer sites: prostate, lung, esophagus, cervix, stomach, pancreas, multiple myeloma, oral, colon, and larynx. Studies of these sites were eligible for inclusion in this review.

Analysis

First, the strength of the SES-cancer association, as characterized by the RR calculated by taking disease rates of the high SES group over the low one), was summarized across studies and by cancer site for black and white samples. Socioeconomic status was dichotomized at median (Md) quantile breaks for this analysis or as close to this analytic goal as the data reported for each individual study would allow. Summary or across study estimates were simple arithmetic mean (M) rate ratios; the original studies generally did not report data sufficient for pooling estimates by Mantel-Haenszel procedures.[31] The PAR% or etiologic fraction then was used as a summary measure to more fully describe the SES-cancer relationship among blacks and whites.[32,33] The PAR% is defined as the fraction of disease experience in a population that would not have occurred if the effect associated with the exposure of interest (eg, low SES) were absent. As the PAR% is a function of both the strength of the association of a given risk factor with disease (ie, the RR) and the prevalence (P) of that risk factor among the population at risk,[32] it has implications for both disease etiology and public health planning. The etiologic fraction is calculated by the formula:

$$[P(RR\text{-}1)/1 + P(RR\text{-}1)] \times 100.$$

RESULTS

Characteristics of the Reviewed Studies

The 10 retrieved studies were published between 1976 and 1992 (Md = 1981; M = 1982.7). They are all retrospective cohorts, that is, cumulative incidence studies, with typically 5 years of incidence data (M = 6.2 years). Nine studies assessed cancer incidence, one mortality only, and three studies measured both incidence and mortality among white and black samples. One study used a nonwhite sample that included blacks predominantly. Perhaps more importantly, none of the reviewed studies measured SES at the level of the individual; all of them were ecological in this regard. Nine of the studies evaluated socioeconomic variables at the level of census tracts and one at the county level.

SES and Cancer

Table 3 summarizes the results of the 10 epidemiologic studies that included samples of both blacks and whites and observed the SES-cancer association among each.

TABLE 2. BLACK-WHITE CANCER RATE DIFFERENCES (RD) AND RATE RATIOS (RR) ACROSS THE 20 MOST COMMON SITES, WITH PROPORTION (%) OF THE RD ACCOUNTED FOR BY SITE, 1978-1981: SEER, AGE- AND GENDER-ADJUSTED TO 1970 US STANDARD*

	Incidence			Mortality		
Site	**RD†**	**%**	**RR†**	**RD**	**%**	**RR**
All sites	37.5		1.11	44.9		1.27
Prostate	19.3	25.2[1]‡	1.60[5]	9.5	21.7[1]	2.09[3]
Lung and bronchus	18.3	23.9[2]	1.36[8]	9.4	21.5[2]	1.23[10]
Esophagus	8.5	11.1[3]	3.83[1]	6.6	15.1[3]	3.54[1]
Cervix uteri	6.6	8.6[4]	2.30[3]	3.2	7.3[5]	2.75[2]
Stomach	5.8	7.8[5]	1.73[4]	4.7	10.7[4]	1.89[6]
Pancreas	4.7	6.1[6]	1.53[6]	2.6	5.9[6]	1.31[9]
Multiple myeloma	4.5	5.9[7]	2.32[2]	2.6	5.9[7]	2.08[4]
Buccal cavity and pharynx	3.6	4.7[8]	1.33[9]	2.4	5.5[8]	1.73[7]
Colon	3.3	4.3[9]	1.10[10]	0.7	1.6[10]	1.04[11]
Larynx	2.0	2.6[10]	1.43[7]	1.2	2.7[9]	1.92[5]
Kidney	−0.2[11]		0.97[11]	−0.5[16]		0.84[16]
Leukemias	−1.0[12]		0.90[12]	−1.0[18]		0.85[15]
Hodgkin's disease	−1.2[13]		0.59[18]	−0.2[15]		0.78[18]
Ovary	−2.1[14]		0.70[15]	−0.8[17]		0.79[17]
Brain and central nervous system	−2.4[15]		0.60[17]	−1.7[19]		0.56[20]
Rectum	−3.3[16]		0.78[14]	0.0[13]		1.00[12]
Non-Hodgkin's lymphoma	−3.8[17]		0.64[16]	−1.9[20]		0.63[19]
Corpus uteri	−6.1[18]		0.53[20]	0.6	1.4[11]	1.45[8]
Breast (female)	−6.4[19]		0.84[13]	0.3	0.7[12]	0.99[13]
Bladder	−6.8[20]		0.56[19]	−0.1[14]		0.97[14]

Abbreviations: SEER = Surveillance, Epidemiology, and End Results program.
*Table adapted from Baquet et al[3] and Pickle et al.[5]
†RD = $I_b - I_w$ and RR = I_b/I_w where I_b and I_w are cumulative incidence rates per 100 000 black and white populations. For sites that affect a single gender (eg, prostate, breast, cervix uteri, corpus uteri, and ovary), RRs were estimated among the "at risk" gender and RDs were estimated for the entire population, both female and male.
‡Superscripts indicate the rank order of the between racial group difference or ratio.

These studies gathered data from six national US samples and four regional or statewide ones (three from California and one from Maryland). Across analytic studies, cancer sites, and SES indices, the risk associated with low SES versus high SES was found to be similar among blacks (RR = 1.19) and whites (RR = 1.17). As for risk factor prevalence, blacks were observed to experience approximately a fourfold greater exposure to low SES (52.8% versus 12.3%); consequently, the estimated PAR% of cancer occurrence due to low SES among blacks is more than fourfold the white estimate (9.1% versus 2%).

The strongest SES-cancer associations were observed for stomach and lung cancers, which interestingly were the sites with the most intimate environmental interfaces. These associations again were found to be similar among blacks and whites: stomach—black (RR = 2.12) and white (RR = 2.14), and lung—black (RR = 1.31) and white (RR = 1.37). Also, the black PAR% of both stomach and lung cancers due to low SES exposure were estimated to be approximately threefold the white estimate: stomach (37.2% versus 12.3%) and lung (14.2% versus 4.4%).

One cancer site, cervix uteri, demonstrated a markedly dissimilar socioeconomic gradient among blacks (RR = 1.30) and whites (RR = 2.10). However, because of the much greater low socioeconomic exposure among blacks, the PAR% of cancer of the cervix due to low SES seems to be similar for the two groups (13.7% and 11.9%, respectively). Two competing explanations for this phenomenon may be conjectured. It may be that the black and white socioeconomic gradients for cervical cancer really are similar, but the rather homogeneous distribution of SES among blacks relative to whites bias the black RR toward the null. For example, one study[S6] assessed SES across quintiles among whites, but only across tertiles for blacks. It is also possible that cultural or genetic factors interact with socioeconomic ones to produce the differential observed effects.

DISCUSSION

A number of general data trends were revealed by this review. First, the magnitude of the associations

TABLE 3. SUMMARY OF STUDIES EVALUATING THE RELATIONSHIP OF SOCIOECONOMIC STATUS (SES) WITH CANCER INCIDENCE/MORTALITY AMONG BLACK AND WHITE POPULATIONS*

Study†	Exposure	Site	RR		Risk Factor Prevalence (%)	
			Black	White	Black	White
					Low SES	
Ernster, Selvin, Sacks, et al Ernster, Winkelstein, Selvin, et al	SES‡	Prostate	1.00	1.00	52.8	18.4
Wright, Bernstein, Peters, et al	SES§	Stomach	3.84	4.00		
					<$15 000/year	
Baquet, Horm, Gibbs, et al	Income	All sites	1.11	1.16	51.1	4.2
		Rectum	1.43	1.22		
		Stomach	1.31	1.20		
		Cervix	1.20	2.08		
		Lung	1.15	1.50		
		Prostate	0.90	1.07		
		Colon	0.89	1.05		
Boring, Squires, & Heath	Income	All sites	1.14	1.16		
					<$9000/year	
Devesa & Diamond	Income	Cervix	1.32	2.01	63.0	15.5
Devesa & Diamond	Income	Lung	1.13	1.20		
Levin, Connelly, & Devesa	Income	Pancreas	1.00	1.00		
Miller & Chapman	Poverty‖	All sites	1.37	1.27		
					<12 years	
Baquet, Horm, Gibbs, et al	Education	All sites	1.18	1.11	32.6	5.9
		Cervix	1.41	2.32		
		Lung	1.34	1.36		
		Stomach	1.21	1.23		
		Prostate	1.07	1.08		
		Colon	0.99	0.89		
		Rectum	0.94	0.96		
Boring, Squires, & Heath	Education	All sites	1.13	1.13		
					<11 years	
Devesa & Diamond	Education	Cervix	1.25	1.98	64.7	17.7
Devesa & Diamond	Education	Lung	1.16	1.16		
Levin, Connelly, & Devesa	Education	Pancreas	1.00	1.00		
Blot & Fraumeni	Urban res.	Lung	1.79	1.63		
Summary (mean) estimates						
		All sites	1.19	1.17	52.8	12.3
		Stomach	2.12	2.14		
		Lung	1.31	1.37		
		Cervix	1.30	2.10		
		Rectum	1.19	1.09		
		Pancreas	1.00	1.00		
		Prostate	0.99	1.05		
		Colon	0.94	0.97		

*Summary estimates are simple across-study arithmetic means. The studies generally did not report data sufficient for pooling estimates by Mantel-Haenszel procedures.[31]
†From studies cited in Table 1.
‡Percentage of individuals 25 years of age or older residing in census tract with some college education.
§Derived SES score—a function of mean income and years of education reported by adult census tract residents.
‖ Defined by federally established criteria.

between SES and cancer occurrence were generally found to be similar for blacks and whites in the United States. However, this review also found that prevalent exposure to low SES-related risks among blacks are

four times greater than that observed among whites. From a public health perspective, then, it becomes clear that although SES, in an etiologic sense, impacts cancer among blacks and whites similarly, the attribution of cancer risk due to low SES exposure or the PAR% is probably four times greater among blacks. The reviewed data also allow for the inference that among blacks in the United States, perhaps as many as one of every 10 cancer occurrences are related to low SES. Similar inferences by site are as follows: stomach (one of every three occurrences) and lung or cervical cancer (one of seven occurrences).

Finally, this article's summary of findings replicated those of previous research[34] in demonstrating that the greatest socioeconomic gradients were for cancers of the stomach, lung, and cervix uteri. In fact, these three sites account for nearly half of the US black-white cancer rate difference (Table 2). Although none of the original 10 studies measured specific correlates of SES, which may explain the SES-cancer relationship, the data trends described above seem to strongly implicate the following: any policy designed to bridge the between-race socioeconomic gap also will serve to diminish the differences that exist between racial groups on cancer morbidity and mortality.

Study Limitations

All 10 primary studies that evaluated the SES-cancer association among blacks and whites were ecological with respect to socioeconomic exposure measurement, that is, they used aggregate measures (eg, census tract Md education or family income) to characterize the individual's exposure. Such an operational strategy is based on the assumption that the tracts, for example, are homogeneous on SES, a premise that has been demonstrated to be tenuous at best, particularly in urban centers.[35] The ecological measure will misclassify individuals to the degree that the areal unit is indeed heterogeneous on SES. Even in the likely case of nondifferential misclassification on disease outcome, such bias may operate to underestimate or overestimate the true SES-cancer association.[36-39] Without external validation, the likely direction of the potential bias, as well as its magnitude, will remain equivocal.[40] The consistency of results across the 10 primary studies lend credence to the validity of this review's findings. However, empirical validation of ecological SES measures would still greatly aid in interpreting the extant data in this field.

Future Research

Two studies have indirectly assessed the validity of using ecological measures of SES, and they suggest that their use may tend to underestimate the true SES-health outcome association at the level of the individual.[39,41] To our knowledge, no previous study has directly validated an ecologic measure with a standard traditional individual measure of SES (eg, census tract Md years of education versus the years of education completed by each individual in the study). Such a validation study would allow essentially for estimation of the sensitivity and specificity of ecological SES measures, and thus, for estimation of the "true" SES-cancer association among blacks and whites, ie, rate ratios that are corrected for errors due to exposure misclassification.[42] Because the prevalence of exposure may be related to the magnitude of misclassification bias[43] and the prevalence of low socioeconomic exposure differs greatly between blacks and whites, ecological measures of SES ought to be validated separately for each racial group.

The size of the areal unit used in a study is necessarily related to the issue of misclassification bias; the smaller the level of measurement, the more likely it will be a homogeneous entity and so, the less potent the bias. Future studies that use ecological measures should consider the use of smaller areal units, eg, block-groups (average $n = 1000$) versus census tracts (average $n = 4000$).[44] Also, the use of multiple aggregate measures, block-group and census tract, for example, together with individual measures, may further aid in the estimation and interpretation of ecological bias.[45] In addition to these methodological needs for future research, this domain of inquiry has yet to address the socioeconomic-cancer associations for a number of sites that demonstrate significant black-white rate differences (Table 2): esophagus, multiple myeloma, oral, and larynx. Future studies should do so.

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