REVISITING THE 1973 REPORT, "ALARMING INCREASE OF THE CANCER MORTALITY IN THE US BLACK POPULATION (1950–1967)"

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A 1973 review article by Henschke et al has been described as a "landmark," influencing the development of cancer surveillance by race/ethnicity in the United States. The 1973 article showed larger increases in total cancer mortality in Black than White males and larger increases for "non-Whites" than Whites for lung, prostate, pancreas, and various other cancers from 1950 to 1967. A review of data published after 1973 shows that the Black-White disparities in cancer mortality rates have generally increased. Research in the past 30 years supports Henschke et al's emphasis on racial differences in specific risk factors (including tobacco, alcohol, obesity, diet, and infectious diseases) and shows the importance of socioeconomic status in explaining Black-White differences in cancer risk and survival. Continued surveillance is needed to determine if declining cancer mortality rates in 1992–2001 for Blacks will continue. (*Ethn Dis.* 2005;15:779–785)

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INTRODUCTION

A 1973 article, "Alarming Increase of the Cancer Mortality in the US Black Population (1950–1967)" by Henschke et al[1] has been described as a "landmark" in the development of awareness of the cancer burden of African Americans (US Blacks).[2] Harold P. Freeman observed that this article influenced the collection of data according to race in the National Cancer Institute's Surveillance, Epidemiology and End Results (SEER) program established in 1973.[3]

The landmark article[1] showed a 20% increase in the overall age-adjusted cancer mortality rate (per 100,000 per year) from 1950 to 1967 for Blacks but no change for Whites and increases for males (ie, from 147 to 220 or +50% for Blacks, and from 158 to 181 or +16% for Whites) but not for females (from 139 to 126 for Whites and from 146 to 142 for Blacks); thus, in 1967, the Black/White ratio was 1.22 for males and 1.13 for females. For specific sites or types of cancer, the report was limited to the "non-White" population (≈91.3% Black in 1967)[1] with data originally reported by Burbank in 1971.[4] Cancers that showed a larger increase, in mortality among non-Whites than Whites included colon, prostate, and lung, while stomach cancer decreased faster in Whites than non-Whites; only cervical cancer showed a slower decrease, and melanoma of the skin showed a faster increase, in Whites compared to non-Whites[1] (Table 1). For females, declines in rates for two common sites (stomach and uterine cervix) affected trends for all cancers combined, but temporal increases in the non-White/White ratio were evident for several other sites (Table 1).[1]

Actual mortality rates by cancer type or site in non-Whites and Whites were not presented by Henschke et al.[1] Data from Burbank,[4] however, show rising non-White/White ratios of rates by cancer site or type for males and females from 1950 to 1967 (Table 1). Henschke et al[1] concluded that Black-White differences in cancer mortality and incidence should be thoroughly studied and that the rapid increase in Black cancer mortality had largely escaped attention.

This report reviews the expansion of cancer surveillance efforts on Black-White differences in cancer rates after 1973, identifies subsequent literature that cited the findings in the 1973 report, and revisits Henschke et al's[1] explanations for the earlier mortality patterns in the context of subsequent cancer trends and epidemiologic research.

Table 1. Age-adjusted cancer mortality rates (per 100,000) in Whites and non-Whites for selected cancer sites in 1950 vs 1967, from Henschke et al[1] and Burbank[4]

		Average Annual Percent Change[1]		Mortality Rate[4]	
		1950 to 1967		1950	1967
Site/Sex		Non-White	White	Non-White/White	Non-White/White
Prostate	M	+.451	−.090	21.8/18.0 (1.21)	30.8/16.9 (1.82)
Lung 162*	M	+.882	+.652	7.3/22.1 (0.33)	22.1/19.7 (1.12)
	F	+.073	+.078	1.6/1.6 (1.00)	2.9/3.0 (0.97)
Lung 163†	M	+1.306	+.956	8.3/12.1 (0.69)	32.0/29.3 (1.09)
	F	+.183	+.116	2.2/3.1 (0.71)	5.5/5.3 (1.04)
Esophagus	M	+.294	−.013	7.1/4.2 (1.69)	11.3/4.0 (2.83)
	F	+.063	−.003	1.9/1.1 (1.73)	2.6/1.0 (2.60)
Stomach	M	−.498	−.688	29.8/22.2 (1.34)	21.1/10.5 (2.00)
	F	−.303	−.393	15.2/12.2 (1.26)	9.0/5.4 (1.67)
Pancreas	M	+.340	+.157	6.0/7.7 (0.78)	12.1/10.5 (1.15)
	F	+.215	+.059	3.9/5.2 (0.75)	7.4/6.5 (1.14)
Colon	M	+.289	+.088	9.4/15.4 (0.61)	14.2/16.9 (0.84)
	F	+.215	−.092	11.4/17.2 (0.66)	14.3/15.6 (0.92)
Myeloma	M	+.134	+.157	1.1/1.0 (1.10)	3.4/2.2 (1.55)
	F	+.215	+.059	0.8/0.7 (1.14)	2.4/1.5 (1.60)
Cervix	F	−.326	−.208	21.7/9.8 (2.21)	16.1/6.1 (2.64)

* Lung cancer specified as the primary site.
† Lung cancer unspecified as to whether primary or secondary site.
Note: Rates were standardized by using the age distribution of the entire 1960 US population as the standard.[4]

CANCER SURVEILLANCE FOR THE US BLACK POPULATION AFTER THE 1973 REPORT

The SEER program of selected high-quality population-based cancer incidence registries, established in 1973,[5,6] has included several urban areas (Connecticut, metropolitan Atlanta, certain metropolitan areas of California and metropolitan Detroit) with substantial Black populations. Additional areas with both urban and rural Black populations (California, New Jersey, North Carolina, and Louisiana) were added in 2001, increasing coverage to 23% of US Blacks and 26% of the entire US population,[7] but statistics for the newest areas have not yet been included in SEER reports.

SEER reports in 1981[8] and 1996[9] included data on cancer mortality, incidence, and survival rates by race/ethnicity.[8,9] Since 1998, several national agencies and organizations have collaborated to produce an annual report to the nation on the status of cancer, including mortality rates (for the entire United States) and incidence rates (for SEER areas only) by racial/ethnic group. The 2004 report[10] included US cancer mortality data for 1992–2001 (Table 2). Although the mortality rates in Tables 1 and 2 are not directly comparable, because methods of age standardization differ, the racial disparities reported for 1967[1] persisted in 1992–2001, and Black/White ratios in 1992–2001 were higher than non-White/White ratios in 1967 for all sites combined and several major sites, including prostate, lung, pancreas, and colon. Black men had higher mortality rates than White men for all sites combined and for prostate, lung, colon-rectum, oral cavity, stomach, pancreas, esophagus, larynx, and myeloma.[10] Black women also had higher death rates than White women for all sites combined and several sites including breast and cervix (Table 2).[10]

Analyses of trends in US mortality rates by year within the period 1992–2001 (data not shown), however, indicate a larger negative annual percentage change (APC) for Black males (−1.9%) than for White males (−1.4%) for all cancers combined and larger declines for Black than White males for some sites (lung, bladder, esophagus, and oral cavity-pharynx) but not others (prostate, colon-rectum, stomach).[10] For females, the declines were smaller than for males and similar in magnitude to declines for Whites (−0.7% APC) and Blacks (−0.8% APC) for all sites except breast (−2.6% for Whites vs −1.2% for Blacks). As with mortality rates in the past,[1,11] the recent decline in mortality from cervical cancer was larger in Blacks than Whites. For incidence rates during 1992–2001, estimated from SEER data, trends were similar to those for mortality rates, except that for females the total cancer rate increased slightly in Whites (+0.3% APC) but declined slightly in Blacks (−0.4% APC), due in part to increases in breast cancer and melanoma of skin in Whites only.[10]

In 1992–2001, the age-standardized incidence rate for all cancers combined was higher for Black than White males but slightly lower for Black than White females (due largely to the lower rate for breast cancer in Blacks) (Table 2). High Black/White ratios for incidence rates for many sites in both males and females (Table 2) indicate the importance of primary prevention in reducing disparities. Higher Black/White ratios for mortality than for incidence rates,, however, suggest the

Table 2. Age-adjusted cancer mortality rates and incidence rates per 100,000 per year, relative risk (RR) of death from cancer among cancer patients, for Blacks and Whites by sex, 1992–2001, and established or likely risk factors

Site, Type	Sex	Death Rate*			Incidence Rate†	RR of Death in Patients‡	Risk Factors§
		Black (B)	White (W)	B/W Ratio	B/W Ratio	B/NHW Ratio	
Sites/Types with Higher Rates in Blacks							
All	M	364.7	254.3	1.43	1.25	1.26	
	F	200.1	168.6	1.19	0.95	1.52	
Oral cavity, pharynx	M	8.4	4.2	2.00	1.30	1.67	A, D, HPV, T
	F	2.2	1.7	1.29	0.97	1.32	
Esophagus	M	13.2	7.1	1.86	1.73	1.29	A, D, O, T
	F	3.5	1.6	2.19	2.20	—‖	
Stomach	M	14.3	6.4	2.23	1.74	1.33	B, D, T
	F	6.7	3.0	2.23	1.90	0.86	
Colon-rectum	M	35.0	26.1	1.34	1.13	1.29	A, D, DM, O, PI, T
	F	24.9	18.0	1.38	1.22	1.18	
Liver, bile	M	9.1	5.8	1.57	1.60	1.20	HBV, HCV, RI, O, T
	F	3.7	2.7	1.37	1.38	1.14	
Pancreas	M	16.6	12.0	1.38	1.44	1.15	A, D, DM, O, PI, T
	F	12.9	8.9	1.45	1.53	1.09	
Larynx	M	5.8	2.5	2.32	1.79	1.44	A, D, OE, T
	F	1.0	0.5	2.00	2.00	—‖	
Lung	M	110.7	80.0	1.38	1.51	1.09	AP, D, OE, T, RI
	F	39.2	41.0	0.96	1.05	1.07	
Breast	F	36.4	28.3	1.29	0.87	1.75	A, PI, SH
Cervix	F	6.3	2.7	2.33	1.36	1.21	HIV, HPV, SH, T
Corpus	F	7.0	3.9	1.79	0.69	1.82	DM, O, PI, SH
Prostate	F	74.9	31.8	2.35	1.62	1.31	SH
Myeloma	M	9.2	4.5	2.04	1.98	1.01	RI
	F	6.6	2.9	2.28	2.40	1.00	
Sites/Types with Rates Lower in Blacks, or Similar to Whites							
Melanoma of skin	M	0.5	4.4	0.11	0.06	0.88	RU, FS
	F	0.5	2.0	0.25	0.05	1.82	
Testis	M	0.1	0.3	0.33	—‖	—‖	MT
Bladder	M	5.9	8.0	0.74	0.51	1.43	D, HI, OE, T
	F	3.0	2.3	1.30	0.76	1.38	
Kidney	M	6.2	6.2	1.00	1.15	1.06	HI, O, T
	F	2.8	2.9	0.97	1.15	0.99	
Brain, Nervous	F	3.3	6.1	0.54	0.55	0.98	RI
	F	2.3	4.1	0.56	0.59	0.88	
Thyroid	M	0.3	0.4	0.75	0.55	1.15	O, RI
	F	0.5	0.5	1.00	0.53	0.94	
Lymphoma							
Hodgkin	M	0.6	0.7	0.86	0.85	—‖	EBV
	F	0.4	0.4	1.00	0.78	—‖	
Non-Hodgkin	M	7.5	10.9	0.69	0.75	1.21	HIV
	F	4.5	7.2	0.63	0.67	1.29	
Leukemia	M	9.4	10.7	0.88	0.75	1.40	HI, RI, T
	F	5.5	6.1	0.90	0.80	1.36	

* Death rates are for the entire United States, age-standardized by using the age distribution of the entire 2000 US population.[10]

† Incidence data are for the areas covered by the SEER Program; incidence rates are not shown here.[10]

‡ Relative risk (RR) of cancer death, adjusted for age (all cancers combined) and for both age and stage at diagnosis (individual cancer sites or types).[10]

§ Code for risk factors: A (alcohol, or heavy alcohol use); AP (air pollution); B (bacteria); D (dietary factors); DM (diabetes mellitus); EBV (Epstein-Barr virus); FS (fair skin color and poor tanning ability); H (body height); HI (history of infection); HBV (hepatitis B virus); HBC (hepatitis C virus); HIV (HIV virus); HPV (human papilloma virus); MT (maldescent of testes); O (obesity or overweight); OE (occupational exposures); PI (physical inactivity); RI (radiation, ionizing) RU (radiation, ultraviolet); SH (sex hormone levels or related factors); T (Tobacco). Socioeconomic status is not listed, but is associated with these risk factors, physical inactivity may be a risk factor for several cancers, along with family history of cancer.[14,15]

‖ Data were not published for relatively uncommon sites, or rates were statistically unreliable due to small numbers.[10,57]

NHW=non-Hispanic white.[10]

potential importance of early detection, treatment, and/or other factors associated with socioeconomic status (SES) and/or prognosis.

EXPLANATIONS FOR BLACK-WHITE DIFFERENCES IN CANCER RATES

In considering reasons for the increase in Black cancer mortality, Henschke et al[1] discussed errors in census enumeration or inaccuracies in denominators of cancer rates, underreporting cancer in death certificates, genetic differences, cure rates, and (most importantly) environmental factors. Accuracy of population estimates by race/ethnicity is still a concern to the SEER program.[12] With regard to underreporting, temporal increases in Black/White ratio for certain cancers such as pancreas[13] and myeloma could reflect improvements in diagnosis, which affect Blacks more than Whites, but this hypothesis is difficult to evaluate because of low autopsy rates.

Henschke et al[1] focused on racial differences in specific environmental factors, rather than on SES disparities (a more fundamental explanation). The large group of cancers with higher mortality rates in Blacks than Whites, however, includes many that are strongly associated with specific risk factors (such as tobacco and heavy alcohol use, various infections, inadequate diet and obesity)[14] (Table 2) also linked to lower SES. Tobacco use is an accepted risk factor for a growing list of cancers, although its role as a risk factor is uncertain for others (eg, colorectal).[15] Until recent years, Blacks had higher current-smoking prevalence rates than Whites, and many cancers have long latency periods, so the effect of past Black-White disparities in smoking prevalence may persist; in addition, continuing Black-White and SES disparities in smoking cessation rates are a major public health problem. For prostate cancer risk, the association with SES has been less clear.[16–18]

A report published in 1980 examined SES factors but used pre-SEER cancer incidence data for 1969–71 from the Third National Cancer Survey to show that using race-specific population indicators of SES (from the US census) for the census tract of residence at diagnosis greatly reduced the Black-White differences in risk of cervical cancer.[19] Subsequent reports have shown that most Black-White disparities in cancer risk are eliminated or reduced by adjusting for ecologic indicators of SES.[16–18,20]

Genetic variability within African Americans, not mentioned by Henschke et al,[1] has been well documented, and the search for biological or genetic explanations for racial differences in cancer risk and prognosis has been elusive.[21] Henschke et al's conclusion about the limited role for inherited genetic factors in explaining such a large, rapid increase in non-White or Black cancer mortality rates remains valid, and this conclusion may also be valid for most of the Black-White differences in cancer patterns evident in 1992–2001.

Results of studies on polymorphisms in the androgen-receptor gene in relation to the higher risk of prostate cancer risk in Blacks than Whites have been inconsistent,[22] possibly because of genetic variability among African Americans and/or limitation of the association to younger ages at diagnosis. The African-American Hereditary Prostate Cancer Study Network, with NIH funding to Howard University, has focused on the HPC-1 gene, but few African-American families have been analyzed.[23,24] Recent studies of prostate cancer risk,[25] however, have suggested that Black-White differences in such factors as diabetes, obesity and/or body fat distribution, diet, history of infectious disease, and smoking history need closer examination as explanations for Black-White differences in prostate cancer risk.

For multiple myeloma, SES accounts for at least part of the Black-White difference in risk, and although speculation about inherited genetic factors exists,[26] more studies of environmental (including occupational) risk factors are needed. For cancer of the pancreas, a large population-based, case-control epidemiologic study in three states (Atlanta, Ga; Detroit, Mich; and 10 New Jersey counties) found that Black-White differences in prevalence of specific risk factors (tobacco and alcohol use, obesity, and diabetes mellitus) explained the Black-White disparity in risk.[27] In studies without such detailed information on risk factors, results showing that Black-White differences persist despite statistical adjustment for SES are often emphasized without critical review of the adequacy of such adjustment.[28,29]

SURVIVAL RATES

Henschke et al[1] reported that slightly lower "cure" rates, as estimated from the 1969 Third National Cancer Survey and 1967 mortality statistics, in Blacks than Whites could not explain all the Black-White differences in cancer mortality. However, they noted that "few Black families have the economic resources... required for optimal cancer care."[1] In 1973, lack of data on SES of patients was noted in a report of lower survival rates for Black than White pediatric leukemia patients in a large US clinical trial.[30] The same problem was mentioned in a 2003 report of lower survival of Black vs White childhood leukemia patients diagnosed in SEER areas.[31] A study with a smaller sample of Black children seen at a single institution that provides broad access to treatment, however, found no Black-White difference in survival.[32] The lower survival not only of Black but also American Indian/Alaskan Native and Hispanic children compared to non-Hispanic Whites in the SEER data[31] suggests a role for SES and/or other factors related to healthcare access.

Another hypothesis for the lower survival of Black vs White patients with pediatric leukemia involves possible racial-ethnic differences in biological response to drugs, which is related to genetic polymorphisms affecting drug detoxification.[31] However, genetic differences between racial groups are based on large numbers of polymorphic gene loci, each with small differences in frequency among populations,[33] so that pharmacogenetic studies may be more useful in developing individualized drug treatment (regardless of patient race/ethnicity).[34]

Since 1978[35] and the early 1980s,[36] five-year relative survival rates (RSRs) have been routinely used to compare cancer patients by race/ethnicity in the SEER program, because RSRs adjust for expected mortality in the general US population for persons of comparable age, sex, and race. The term ''cure'' is rarely used because mortality rates among cancer survivors may not decline to the level found in the general population until many years after cancer diagnosis. A 1978 report by Axtell and Myers[35] used data from SEER Program and its predecessor, the End Results Program (with data from Connecticut, California, University of Iowa Hospitals, and Charity Hospital in New Orleans, La), to show that survival for Black patients was less favorable than that for Whites for all types of cancers except multiple myeloma. The report,[35] often cited in the literature, concluded that explanations should be explored,[36] and cited Henschke et al[1] in suggesting the roles of immunologic reactions, nutrition, economic class, accessibility of medical care, and environmental dangers.[35]

Both Henschke et al[1] and Axtell and Myers[35] were cited in a 1985 report, in which Black-White differences in survival of breast cancer patients were explained by clinical factors (including tumor grade and size) at diagnosis.[37] Obesity was also noted as a prognostic indicator,[37] a finding confirmed as a partial explanation for Black-White differences in survival.[38] Although overall age-adjusted breast cancer incidence rates are lower in Blacks than Whites (Table 2), incidence rates are higher for Black than White women at younger ages, when prognosis tends to be poorer; the explanation may involve racial differences in reproductive history.[39,40]

In 1985, Henschke et al[1] was the first citation in a report[41] that used data from the 22 officially designated US comprehensive cancer centers, showing that the Black-White differences in risk of death (all causes) among prostate cancer patients was minimal and not statistically significant after adjusting for stage at diagnosis and educational attainment (at the zip-code level). A later report from the San Francisco Bay area of the SEER Program found that stage and census-tract-level SES variables explained most of the excess of death among Black vs White prostate cancer patients, except for deaths from prostate cancer among patients <65 years at diagnosis, but the potential limitations of ecologic variables in adjusting for SES were acknowledged.[42]

Black-White differences in clinical-pathologic stage (ie, tumor size and extent of cancer spread) at diagnosis contribute to Black-White differences in survival, and the stage differences are largely explained by SES. Temporal trends in both stage at diagnosis of cancer patients and cancer screening rates in the general population in recent years have indicated some reductions in Black-White disparities.[10] The annual report to the nation on cancer in 2004[10] showed elevated relative risk (RR) of death from cancer, adjusted for stage (as well as age) at diagnosis, for Black vs non-Hispanic White patients in the SEER program for all sites combined and for oral cavity-pharynx, larynx, breast, uterine corpus, prostate, and bladder but not for cancers for which treatments have been of little efficacy (eg, pancreas, lung, myeloma, kidney, and brain) (Table 2). Treatment was not included in the analyses[10] because routine SEER data on treatment (especially adjuvant therapies) are incomplete.

Treatment and overall quality of cancer care for Blacks and other minority groups are now major research issues; evidence for disparities in treatment is considerable, as reviewed elsewhere.[43–46] For Black and White cancer patients comparable in both treatment and stage at diagnosis, a metaanalysis of published studies found statistically insignificant Black-White differences in survival rates, adjusted for mortality rates in the general US population (by age, sex, and race), for each cancer except for breast, bladder, and uterus.[47] However, the results of both clinical trials and observational studies suggest that treatment and SES may account for the Black-White disparity in breast cancer survival.[48] The metaanalysis[47] did not control for SES, which may be associated with survival largely through stage and treatment, but also independently through diet or other factors related to environmental quality or host factors.

Clinical trials of cancer treatments usually provide extensive data on all patients (eg, clinical-pathologic or prognostic features, comorbidity, and/or functional status at the start of the trial) and provide equal access to specific treatment(s). As noted earlier, however, statistical control for SES is often absent or limited. After including SES indicators at the zip-code level, however, the Black-White difference in survival in a clinical trial of treatment for advanced prostate cancer persisted but was no longer statistically significant.[49,50] In such analyses, statistical control for SES is needed, with geographic units smaller than zip codes,[44] and/or SES indicators for individual patients.

Even when the proportion of Black patients in clinical trials of treatments is close to that in the general population,[51] the number of Black patients may be small. NIH guidelines (since 1993) have called for including larger numbers of minority patients in clinical trials in order to analyze differences in treatment effects.[52] A trial of treatments for advanced lung carcinoma included only 46 Black (and 458 non-African-American) patients, although Black-White differences in

outcome disappeared after adjusting for health status at diagnosis.[53] A single, large adjuvant chemotherapy trial for colon cancer found similar risks of recurrence and death in 344 Blacks and 3380 Whites, after adjustment for stage, treatment assignment, and performance (ambulatory) status.[54] Such analyses are needed from clinical trials for other types of cancer.

CONCLUSIONS

Henschke et al's[1] report influenced the development of surveillance of racial-ethnic disparities in cancer,[3,46] and their observations on reasons for the ''alarming'' increases in Black mortality rates have a remarkably contemporary ring. Cancer surveillance by race-ethnicity has expanded greatly since 1973, but the importance of monitoring cancer mortality rates in populations (and not just survival rates among cancer patients) has been supported.[55] Surveillance is needed to determine if the larger decline during 1992–2001 in overall cancer mortality rates in Blacks compared to Whites continues. Several major programs aimed at reducing racial disparities in cancer have only been recently initiated.[20,46]

Elevated Black/White ratios for incidence rates for many cancer sites in 1992–2001 suggest the need for expanded programs in primary prevention in the Black population. Higher Black/White ratios for mortality rates than for incidence rates emphasize the need to address inequities in stage, treatment, and/or SES. Comparable Black-White survival rates after statistical adjustments (for stage, treatment, and/or SES) in many studies show what might be achieved if access to health care and other resources were more equitable. Cancer data on US minority groups other than Blacks may aid in interpreting Black-White disparities, as well as in defining both cultural and SES factors involved in racial-ethnic disparities in cancer risks and survival.[56]

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