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Living and Dying in the U.S.A.: Sociodemographic Determinants of Death Among Blacks and Whites*

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This paper examines the demographic and social factors associated with differences in length of life by race. The results demonstrate that sociodemographic factors—age, sex, marital status, family size, and income—profoundly affect black and white mortality. Indeed, the racial gap in overall mortality could close completely with increased standards of living and improved lifestyles. Moreover, examining cause-specific mortality while adjusting for social factors shows that compared to whites, blacks have a lower mortality risk from respiratory diseases, accidents, and suicide; the same risk from circulatory diseases and cancer; and higher risks from infectious diseases, homicide, and diabetes. These results underscore the importance of examining social characteristics to understand more clearly the race differences in overall and cause-specific mortality.

There is enormous national concern about the high mortality among blacks. The racial gap in life expectancy, which had been closing slowly over the century, has widened recently. Most attempts to understand this gap have relied on a purely demographic perspective in which black and white mortality rates are compared separately by sex, with adjustment for age. Other social factors, however, may contribute to the black/white difference in mortality in the United States. This study endeavors to identify the key factors that create the racial gap in life expectancy, to determine whether the gap persists across all causes of death, to discover whether the gap is due to blacks' greater propensity to be in disadvantaged categories or to differential survival within the categories, and to illustrate what types of social changes could contribute most to closing the mortality gap.

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Racial Gap in Life Expectancy

Between 1985 and 1988, black life expectancy lost ground slowly but consistently after many years of improvement. The black/white gap in life expectancy at birth was 14.6 years in 1900 and had declined to 5.6 years by 1984, but had risen to 6.4 years by 1988 (National Center for Health Statistics [NCHS] 1991c). The recent divergence is due both to small increases in white life expectancy and to small decreases in black life expectancy, which declined from a high of 69.7 years in 1984 to 69.2 years in 1988 (NCHS 1991c). To reverse this trend, the federal government has formed special task forces and has stated specific goals; for example, one of the five major health goals of the nation is to reduce the racial disparity in life expectancy from more than six years to no more than four years by 2000 (U.S. Dept. of Health and Human Services [DHHS] 1989b). Although preliminary figures show that the gap was closing again for 1989 and 1990 (NCHS 1991a), it is still large, appears in many age groups, and is still evident at ages 25 and over, the age focus of this study. At age 25, for example, white men can expect to live 6.3 years longer than black men; white women can expect to live 4.6 years longer than black women (NCHS 1991c).

The social characteristics hypothesis states that once pertinent social, demographic, familial, and economic characteristics are controlled, race differences in mortality will disappear. This hypothesis seems likely to be valid because race is confounded with social and economic status (Navarro 1990). For example, married persons consistently exhibit lower levels of mortality than those who are not married. Marriage acts to select healthy individuals, to enhance social integration, and to encourage healthful behavior (Gove 1973; Mergenhagen, Lee, and Gove 1985). Blacks, however, are less likely than whites to be married, just as they are less likely to come from small families or to be well housed, well educated, or well employed.

As income increases, mortality decreases, even if other variables are controlled. High income provides access to high-quality health care, diet, housing, and health insurance; 42% of blacks under age 65, but only 19% of whites, lack private health insurance (NCHS 1991b). Once such differences are controlled, black/white differences in mortality should diminish (for examples of male mortality, see Mare 1990; Mott and Haurin 1985).

Race Differences in Cause of Death

In general, blacks have higher mortality than whites at most ages and from most causes of death (Keith and Smith 1988; Potter 1991). The racial gap in life expectancy among adults is due primarily to the following causes: heart disease and stroke, cancer, diabetes, homicide, accidents, and chemical abuse (DHHS 1985). Using cause-substituted life tables, Keith and Smith (1988) show that at all ages, the largest contributors to the racial gap in life expectancy are circulatory diseases for both sexes and homicide and cancer for males. Otten et al. (1990), using age-adjusted mortality rates, find that the major causes contributing to the black/white difference in mortality for individuals aged 35–77 are cardiovascular diseases and cancer.

Table 1 presents age-adjusted cause-specific mortality rates by race and sex. These figures are produced by combining the 1986 National Health Interview and the National Mortality Followback Surveys (NCHS 1988a, 1989; see "Data and Methods" section for further details about the data sets). Black males have the highest total mortality, followed by white males, black females, and white females. This table shows that generally those causes of death which contribute to overall mortality also contribute to the race difference in mortality. Overall, blacks are 50% more likely to die than whites. The racial disparity favors

Table 1. Age-Adjusted Cause-Specific Mortality Rates and Ratios by Race and Sex: U.S. Adults, 1986

Cause of Death	Male				Female			
	1 White ^a	2 Black ^a	3 Black:White (2/1)	4 % Black/White Mortality Rate Difference ^b	5 White ^a	6 Black ^a	7 Black:White (6/5)	8 % Black/White Mortality Rate Difference ^b
Circulatory Disease	497.4	655.9	1.3	26.3	262.5	438.2	1.7	53.3
Cancer	275.6	423.0	1.5	24.5	200.9	235.7	1.2	10.6
Respiratory Disease	88.5	114.5	1.3	4.3	39.1	41.9	1.1	.8
Accidents	60.8	94.3	1.6	5.6	20.6	25.1	1.2	1.3
Suicide	31.7	17.3	.5	-2.4	8.4	4.4	.5	-1.2
Infectious Disease	15.2	31.0	2.0	2.6	6.5	25.1	3.9	5.6
Diabetes	14.7	37.6	2.6	3.8	12.9	39.6	3.1	8.1
Homicide	9.4	87.9	9.4	13.0	3.1	12.7	4.1	2.9
Other	106.2	240.6	2.3	22.3	69.6	130.6	1.9	18.5
Total	1099.5	1702.2	1.5	100.0	623.6	953.2	1.5	100.0

Note: Causes of death may not add to total because of rounding. For discussion of data, see “Data and Methods” section. For classification of causes of death, see Table 2.

Sources: National Health Interview Survey, 1986 (NCHS 1989); National Mortality Followback Survey, 1986 (NCHS 1988a).

^a Age-adjusted to U.S. 1940 total population (rates per 100,000).

^b Calculated by ((cause-specific black mortality rate - cause specific white mortality rate)/ (total black mortality - total white mortality) * 100).

whites for every cause except suicide. Together, the eight causes of death listed contribute to at least 85% of the overall mortality and to about 80% of the racial gap in mortality.

Because circulatory diseases and cancer cause by far the greatest number of deaths, the racial disparity in those disorders makes large contributions to the racial disparity in total mortality. Indeed, more than 50% of the female racial gap in mortality is due to circulatory disease. Among black females the rate of circulatory disease is 1.7 times that of white females. Part of this excess may be due to hypertension, which is a risk factor in heart disease and is a chronic disease more prevalent among blacks than among whites (Jaynes and Williams 1989). Through increased education, screening, and treatment, rates of hypertension have declined in the United States, but blacks are still at greater risk.

Cancer, the second leading cause of death, also displays a substantial excess among blacks, both male and female. Almost one-quarter of the male racial gap in mortality is due to cancer. Compared to white males, black males experience higher rates of lung, esophagus, and prostate cancer. Among women, blacks aged 15 and over are 2.6 times more likely than whites to die from cervical cancer (Centers for Disease Control [CDC] 1990). Several social factors help to explain these differences. For example, black males are more likely to smoke than white males (Jaynes and Williams 1989; Manton, Patrick, and Johnson 1987). Prostate and cervical cancer can be diagnosed and treated, but it is suspected that black people in low socioeconomic strata are less likely than whites to receive preventive services such as pap smears and breast and prostate examinations (Jaynes and Williams 1989). Even those who do receive screening may suffer from disparities in follow-up and treatment (CDC 1990). Moreover, blacks are less knowledgeable than whites about the signs of cancer and are more pessimistic about treatment outcomes; they may delay seeking screening and treatment not only because of economic constraints but also because they perceive cancer as terminal (Manton et al. 1987).

The most striking differences in black/white mortality ratios exist in three causes that account for relatively small numbers of deaths: homicide, diabetes, and infectious diseases. Homicide—which accounts for 13% of the racial gap for males but only 3% for females (see Table 1, Columns 4 and 8)—is a leading cause of death among blacks, especially young blacks. Among adults, black males are nine times more likely than white males to die from homicide (Table 1, Column 3; see also Greenberg 1990). Of course, the causes of homicide are many and complex, but poverty, unemployment, and alcohol and drug use are central social risk factors (Jaynes and Williams 1989; Potter 1991).

Diabetes accounts for more than 8% of the racial gap in mortality for females, but less than 4% for males (Table 1). Diabetes is an important cause of death, a major risk factor in heart disease, and (if not controlled) a contributor to such serious health conditions as kidney failure (DHHS 1985). Two types of diabetes—insulin-dependent diabetes mellitus (IDDM) and noninsulin-dependent diabetes mellitus (NIDDM)—account for 90 to 95% of all diabetes cases in the United States. The excess of diabetes morbidity and mortality among minorities is due overwhelmingly to NIDDM (DHHS 1985). Compared to whites, blacks have a 30% higher incidence of diabetes; black females have especially high rates. NIDDM is related to obesity; blacks, especially black women, are more likely to be obese; and most blacks who are diabetic are overweight women (DHHS 1985; Manton et al. 1987). Blacks are about three times more likely than whites to die from diabetes.

Infectious diseases are no longer a primary cause of death in the population at large (Crimmins 1981; Rogers and Hackenberg 1987) and do not make a large numerical contribution to the racial disparity in mortality. Yet many adults still die from infectious diseases, and blacks' mortality is more than twice that of whites. Furthermore, more than 5% of the female black/white mortality difference is due to infectious diseases.

Other significant racial disparities exist in accidents. Risk factors for accidental death include alcohol consumption, which can increase risk through home, industrial, and

recreational accidents, and low socioeconomic status, which can increase risk through lack of smoke detectors, inappropriate heating devices, poor housing, and lack of swimming instruction. Blacks are 3.2 times more likely than whites to die in a residential fire, 1.8 times more likely to drown, and 1.6 times more likely to die as pedestrians (Gulaid et al. 1988), but they have lower rates of motor vehicle accidents (NCHS 1990). Gulaid et al. (1988) suggest that because blacks are less likely than whites to own cars, they are less likely to die in cars; conversely, they are more likely to walk and therefore are at greater risk of pedestrian accidents.

Compared to whites, blacks have high rates of pneumonia and influenza but low rates of chronic obstructive pulmonary diseases (NCHS 1990). For respiratory diseases in general, blacks have slightly higher rates of mortality than whites. Among males, blacks are almost 30% more likely than whites to die of respiratory diseases.

Suicide is the only major cause of death for which blacks have lower mortality than whites. Blacks' lower suicide rates and higher homicide rates may indicate differences in the ways blacks and whites solve personal or interpersonal problems (Farley and Allen 1987). Alternatively, if more poor individuals die by homicide, and if more blacks than whites are poor, this disparity could indicate differences in the ways individuals in different socioeconomic strata solve problems.

From a purely demographic perspective, then, blacks have lower life expectancies and higher cause-specific mortality from all major causes of death save suicide. To what should we attribute these differences? In the following tables I examine racial disparities in mortality, controlling for demographic, familial, and socioeconomic status. I attempt to determine how much of the racial gap in life expectancy for adults is due to sociodemographic factors and whether this gap persists across all causes of death.

Data and Methods

The investigation of sociodemographic differences in mortality has been limited by the availability of data on the social, demographic, familial, and economic characteristics of decedents and of comparable surviving populations. Because few national data sets include both sociodemographic and mortality information, I have created one data set from two national samples: the 1986 National Health Interview Survey (NHIS), which provides the population "at risk," and the 1986 National Mortality Followback Survey (NMFS), which includes data on decedents aged 25 and over. Because this article is the first to link these data sets successfully, I detail the linking procedure and the underlying assumptions. (For other new methodological developments in mortality research, see Nam and Myers 1987.)

The NMFS is a nationally representative sample of 18,733 individuals aged 25 and over who died in 1986. This survey includes information from the death certificate and data on social, demographic, and economic characteristics obtained from informants' responses (NCHS 1988a). More than 80% of the NMFS informants were close relatives of the decedent, often surviving spouses (NCHS 1988a). Although NMFS data are derived from informants' responses, several studies have demonstrated that kin provide accurate information (see Kolonel, Hirohata, and Nomura 1977; Marshall et al. 1980). Because the NMFS includes all decedents, whereas the NHIS samples only the civilian noninstitutionalized population, I maintained comparability by excluding from the NMFS decedents who were not residents of the United States, who served full-time in the armed forces at the time of death, who were institutionalized, who were neither white nor black, or for whom no informant filled out a questionnaire. These adjustments netted a total sample size of 14,471 decedents. (For further detail about NMFS design, pretest results, and data level and quality, see Poe et al. 1988; Seeman, Poe, and McLaughlin 1989.)

NHIS data are obtained from a household multistage probability design that permits a continuous sampling of the noninstitutionalized civilian U.S. population through both cluster and stratified sampling techniques (NCHS 1980). It includes 65,052 subjects, 37,917 of whom are aged 25 years and over, and is weighted to represent the midyear 1986 population (NCHS 1989). This weighting is ideally suited for constructing mortality rates, but is inappropriate for comparing those people who started the year and survived with those who died during the year. Thus for the multivariate analyses, I have adjusted the NHIS sample weights to include only whites and blacks who began the year and survived to the end of the year. Because certain subgroups are more likely to die than others, I reduced the weights within each of 300 subgroups (2 races x 2 sexes x 75 one-year age groups).¹

To concatenate these two data sets into one data set of individuals who began 1986 and either survived the entire year or died sometime during the year, I divided both the NHIS and the NMFS sample weights by the NHIS mean sample weight. These adjustments 1) maintain the nationally representative character of the data set, 2) represent the original NHIS, 3) scale down the NMFS to represent the number of deaths expected in the NHIS sample, and 4) produce results similar to NCHS published figures. For example, I calculated life expectancies using Smith's (1988) SURVIVAL life table program, and my calculations are similar to or slightly higher than NCHS life expectancies (see NCHS 1988b): my figures were .4 year higher for white males, 1.0 year higher for white females, the same for black males, and .9 year higher for black females. These differences are acceptable in view of differences in data collection (NMFS does not include deaths from Oregon), sample size, and life table methods. The two files create a data set of 50,754 total records, representing 36,046 individuals who survived 1986 and 444 who died in 1986.² Although this linked cross-sectional data set is not longitudinal, it has the advantages of a large sample with numerous covariates and a substantial number of deaths (this data set has more than 30 times as many deaths as would be available in the NHIS, a sample of more than 36,000 adults).

Classification of Causes of Death

The analyses use eight major causes of death plus a residual category (see Table 2). These codes are based on the three-digit categories presented in the ninth revision of the *International Classification of Diseases*, or ICD (U.S. Department of Health and Human

Table 2. Cause of Death Coding Assignments Based on the Three-Digit ICD Categories

Cause of Death	ICD Code
Infectious Diseases	ICD 001-139
Cancer	ICD 140-239
Diabetes	ICD 250
Diseases of the Circulatory System	ICD 390-459
Diseases of the Respiratory System	ICD 460-519
Accidents and Violence	
Accidents	ICD E800-949, E970-999
Suicide	ICD E950-959
Homicide	ICD E960-969
All Other Causes	ICD 240-249, 251-389, 520-799

Note: Based on *International Classification of Diseases*, ICD (U.S. Department of Health and Human Services 1989a).

Services 1989a). These categories highlight causes of death that are associated with the racial disparity in life expectancy and that contribute disproportionately to general mortality. Approximately 80% of the excess deaths among black adults result from these eight causes. The coding balances the substantive need for detailed cause-of-death information with the methodological need for reasonable sample size.³

Circulatory diseases, the primary cause of death, include heart and cerebrovascular diseases (stroke). Rather than selecting the broad category of social pathologies—comprising homicides, suicides, cirrhosis of the liver, and accidents—I retained more detail because blacks are more likely than whites to die from homicide, but less likely to commit suicide. Infectious disease, no longer a major cause of death, includes such disorders as tuberculosis and syphilis. Respiratory diseases include pneumonia, influenza, and chronic obstructive pulmonary disease (e.g., bronchitis, emphysema, asthma).

Statistical Calculations

To examine race relations with mortality within a multivariate framework, I performed dichotomous and polytomous logistic regression analyses with the statistical package BMDP (Dixon et al. 1990). The NHIS data set is based on a household multistage probability design; therefore the sampling frame should be included in the analysis to determine the appropriate error terms. Yet because I combined data sets, and because I am using polytomous logistic regression where the software does not allow for adjustments for the sampling frame, I have not modified how standard errors are calculated. This assumption will not affect the coefficients or the odds ratios, but may bias the standard errors. To estimate the coefficients properly, I used the weighted sample, but for hypothesis testing, I calculated the standard errors from the unweighted sample (see Wolinsky and Johnson 1991).

The coefficients indicate, through their signs, whether the relations with mortality are direct or inverse. The odds ratios for categories, which are calculated by taking the antilogs of the estimated coefficients, compare one group to another when the other correlates of mortality are controlled. An odds ratio of 1.0 indicates no difference between categories, a ratio above 1.0 indicates a positive difference, and a ratio below 1.0 indicates a negative difference.

Results

Covariates of Mortality

Table 3 presents the description and means of the covariates for people who survived 1986 and for those who died in 1986. A comparison of the two columns shows that death is more likely to occur among those who are black, older, male, unmarried, and poorer.⁴ The crude death ratio, 12.3, shows that for every 1,000 adults who survived 1986, 12 died.

Family size includes household members who are related by blood or marriage. NHIS measures family size in 1986; NMFS requests family size in 1985. Measuring family size in two different years might bias results, but actually could improve them. Because some individuals may be institutionalized in part of their last year of life, it is reasonable to seek information in a less disruptive year. In addition, family size is related directly to family income, the one income variable available in both data sets.

The NMFS and the NHIS asked for the family's income in 1985. Family income includes money from jobs, retirement income, social security, unemployment payments, and public assistance, as well as from interest and dividends (NCHS 1988a, 1989). Again,

Table 3. Description and Means of Covariates: U.S. Adults, 1986

Variable	Categories	Alive	Dead
Independent			
Demographic			
Race ^a	0 = White	10.8	12.4
	1 = Black		
Age	5-year age groups for ages 25 +	5.1	9.6
Sex ^a	0 = Female	47.1	54.8
	1 = Male		
Familial			
Marital status ^a	0 = Married	29.1	49.7
	1 = Not Married		
Family size	Ranges from 1 to 9 or more family members in household	2.9	2.2
Socioeconomic Status			
Family income (in ten thousands)	0.7 = <\$11,000	2.8	1.7
	1.3 = \$11,000–14,999		
	1.7 = \$15,000–18,999		
	2.2 = \$19,000–24,999		
	4.0 = >\$25,000		
Dependent			
Mortality ^b	0 = Alive		12.3
	1 = Dead		

Sources: National Health Interview Survey, 1986 (NCHS 1989); National Mortality Followback Survey, 1986 (NCHS 1988a).

^a Percentages.

^b Per 1,000.

because income could be a consequence rather than a cause of poor health, it is fortuitous that income is measured in the previous year. Furthermore, this analysis examines the civilian noninstitutionalized population rather than long-term institutionalized individuals, so that those individuals who have major health problems, and possibly tenuous financial conditions, are not included. Moreover, Mutchler and Burr (1991) showed that incomes are relatively stable over the course of the year for most elderly individuals, even among those who become institutionalized or who die. Because values were missing for family size and income, and because more values were missing for blacks than for whites, I imputed codes for the missing values, using separate regression equations by each sex-race subpopulation.⁵

Race Differences in Adult Mortality, with Controls for Sociodemographic Factors

Table 4 reports the race differences in mortality, controlling simultaneously for other covariates, through four cumulative models. I begin with the demographic characteristics, then sequentially add family and socioeconomic characteristics. Comparisons among these models allow us to address a number of important questions. A comparison of Models 1 and 2 shows how much the effects of race are mediated by marital status and family size; a comparison of Models 1 and 3, how much racial differences are influenced by socioeconomic factors. Finally, the full model shows how much the racial gap in mortality

Table 4. Sociodemographic Differences in Mortality: U.S. Adults, 1986 (N = 50,754)

Status	Model 1		Model 2		Model 3		Model 4	
	Coefficient	Odds Ratio	Coefficient	Odds Ratio	Coefficient	Odds Ratio	Coefficient	Odds Ratio
Demographic								
Race	0.390***	1.48	0.252***	1.29	0.156***	1.17	0.011	1.01
Age	0.439***	1.55	0.432***	1.54	0.378***	1.46	0.392***	1.48
Sex	0.568***	1.76	0.740***	2.10	0.665***	1.95	0.772***	2.16
Familial Status								
Marital status			0.586***	1.80			0.435***	1.54
Family size			0.115***	1.12			0.189***	1.21
Socioeconomic Status								
Income					-0.348***	.71	-0.354***	.70
Log-Likelihood		-2760.60		-2745.20		-2723.20		-2705.70
Constant		-8.03		-8.55		-6.82		-7.59

Sources: National Health Interview Survey, 1986 (NCHS 1989); National Mortality Followback Survey, 1986 (NCHS 1988a).

*** $p \leq .001$.

is affected by the combined effects of demographic, familial, and socioeconomic characteristics.

Model 1 shows that if one does not control for other factors, all the demographic variables appear to be related significantly to mortality. Mortality is significantly higher for blacks than for whites, for old than for young adults, and for males than for females. The odds ratios show that blacks are 48% more likely than whites to die.

Controlling for family size and marital status, as in Model 2, reduces differences in mortality by race. In this model, blacks are 29% more likely than whites to die (for similar results for males, see Mare 1990; Mott and Haurin 1985).⁶ Married individuals enjoy lower mortality than nonmarried persons, and small families enjoy lower mortality than large families (see also Rogers 1991). Blacks are less likely than whites to be married and to live in small families (Farley and Allen 1987); thus the racial gap in life expectancy is due in part to a disadvantageous household configuration among blacks. If proportionately more blacks marry, remain married, and have fewer children, racial differences in life expectancy should narrow.

Controlling for socioeconomic status, as in Model 3, further reduces the racial gap in mortality.⁷ In this case, blacks are 17% more likely than whites to die. Persons with high incomes can expect lower mortality than those with low incomes, but blacks are more likely than whites to be unemployed or underemployed. Therefore, if blacks could increase their employment opportunities and thereby improve their incomes, they could reduce their mortality concomitantly.

Controlling simultaneously for five key sociodemographic factors—age, sex, marital status, family size, and income, as in Model 4—eliminates race differences in mortality.⁸ Controlling for socioeconomic factors reduces the importance of marital status but underscores that of family size: again, the larger the family, the higher the mortality. Family income, which represents the family's ability to provide proper diets, quality housing, and superior health care, is related strongly to mortality.

If blacks reduce their marital dissolution rates and family sizes, and increase their employment opportunities and income, racial differences in mortality should narrow further. Unfortunately, the proportion of married people in the United States has declined, and has declined faster for blacks than for whites. Between 1960 and 1984, for instance, the proportion of women aged 15–44 who lived with a spouse declined from 69% to 55% for whites, but from 52% to 28% for blacks (Smock 1990). In addition, the economic gap between black and white families has widened, even though the incomes of black individuals have slowly approached those of white individuals. Black and white females now earn about the same; the earnings gap between black and white males closed between 1979 and 1989, as white males' earnings declined. During this period, however, the racial gap in family income widened, in part because the proportion of black families headed by women increased (U.S. Bureau of the Census 1991). Thus the relatively high proportions of single-parent families, the low marriage rates, and the high marital dissolution rates among blacks have eroded the economic gains, and also may diminish their chances for survival.

The racial gap in mortality may not only be due to blacks' greater propensity to be in disadvantaged social categories (measured by direct effects), but also could be the result of differential survival within those categories (measured by interactions). Therefore I tested for interactions between race and age (see Nam, Weatherby, and Ockay 1978) and between race and income (see Wilson 1987).⁹ These interactions produced significant Wald statistics, but they did not contribute significantly to the model fit through the likelihood ratio test. Because Hauck and Donner (1977) demonstrate that the Wald test can provide misleading information, I followed their recommendation to rely on the likelihood ratio test and therefore present only results for direct effects. To understand race differences in mortality more completely, we examine causes of death by race.

Race Differences in Cause of Death, with Controls for Sociodemographic Factors

Other studies (and my Table 1) found that blacks have higher rates of mortality than whites from every cause save suicide. With sociodemographic controls, however, most black cause-specific mortality rates approach those of whites more closely. Even so, blacks are still more likely than whites to die from diabetes, homicide, infectious diseases, and "other causes;" they are less likely to die from accidents, respiratory diseases, and suicide; and they are as likely as whites to die from circulatory disease and cancer.

Circulatory disease and cancer, two major causes of death, contribute to more than 50% of the race gap in mortality. From a purely demographic perspective, blacks are more than 30% more likely than whites to die from circulatory disease, and at least 17% more likely to die from cancer (Table 1). Blacks, especially poor blacks, are less likely than whites to know about the signs of cancer or to receive cancer preventive services, follow-up, or treatment (CDC 1990; Jaynes and Williams 1989; Manton et al. 1987). In the United States, cigarette advertising is now directed to minorities. Also, while more lower-class minorities are beginning to smoke, more upper-class groups are beginning to quit or to smoke less (Susser, Watson, and Hopper 1985). When familial and socioeconomic factors are controlled, however, the mortality from circulatory disease and cancer is the same for blacks as for whites (Table 5). Thus social programs—increased education, employment, and health insurance—could diminish these hazards.

Blacks are over 70% more likely than whites to die from infectious diseases such as tuberculosis, cholera, measles, and syphilis. These diseases are often prevented or treated with proper medical care—immunizations, vaccinations, and antibiotics—and through higher standards of living. This point implies that infectious disease mortality among blacks could be reduced by reducing poverty and its concomitants—overcrowding, poor housing, inadequate nutrition and sanitation, and limited access to health care.

Blacks are 65% more likely than whites to die of diabetes (Table 5). Because diabetes is associated closely with obesity, the control of obesity could prevent an estimated 300,000 cases of diabetes a year (DHHS 1985) and might prevent this excess. Diabetes can be treated and managed if individuals with diabetes are identified early, and diabetic individuals seem better able to manage their disease if they are married (Gove 1973). Fewer blacks than whites, however, have access to preventive health care, and fewer blacks than whites are married.

Blacks are over three times more likely than whites to die from homicides. This contrast suggests cultural and possibly social differences in the ways blacks and whites handle personal and interpersonal problems, and in the ways they perceive life and deal with death. Potter (1991) argues that blacks are more likely than whites to be residentially isolated and economically disenfranchised, and these structural constraints promote violent or risk-taking behavior. Providing blacks with more job and income opportunities will reduce, but will not close, the race gap in homicide. Indeed, Bell (1991) found that among women who work, black women are more likely to be killed than white women.

Whites are three times more likely than blacks to die from suicides and 20% more likely to die from accidents. Guilaud et al. (1988) suggested that blacks are less likely than whites to die from motor vehicle accidents because they are less likely to own cars. Therefore, implementing social programs to increase black income may unintentionally increase black mortality due to accidents.

Conclusion

Most of the racial gap in life expectancy can be traced to blacks' greater likelihood of falling into high-risk social categories. Statistical controls for demographic, familial, and

Table 5. Sociodemographic Differences in Cause-Specific Mortality: U.S. Adults, 1986
(Log-Likelihood = -3,567, N = 50,754)

	Circulatory Disease	Cancer	Respiratory Disease	Accidents	Suicide	Infectious Disease	Diabetes	Homicide	Other
A. Coefficients									
Demographic									
Race	-0.027	0.017	-0.273***	-0.221*	-1.100***	0.551***	0.501***	1.337***	0.208***
Age	0.510***	0.356***	0.531***	0.051***	0.048**	0.270***	0.326***	-0.119***	0.318***
Sex	0.791***	0.499***	1.101***	1.262***	1.756***	0.894***	0.329*	1.794***	0.717***
Familial Status									
Marital status	0.448***	-0.029	0.425***	0.881***	0.972***	1.047***	0.440**	1.210***	0.674***
Family size	0.221***	0.143***	0.246***	0.137***	0.060	0.182***	0.215***	0.070	0.136***
Socioeconomic Status									
Income	-0.300***	-0.343***	-0.358***	-0.483***	-0.411***	-0.281***	-0.537***	-0.637***	-0.442***
Constant	-9.683	-8.166	-11.840	-8.328	-9.422	-11.450	-10.630	-9.514	-9.014
B. Odds Ratios									
Demographic									
Race	.97	1.02	.76***	.80*	.33***	1.73***	1.65***	3.81***	1.23***
Age	1.66***	1.43***	1.70***	1.05***	1.05**	1.31***	1.39***	.89***	1.37***
Sex	2.21***	1.65***	3.01***	3.53***	5.79***	2.44***	1.27*	6.01***	2.05***
Familial Status									
Marital Status	1.57***	.97	1.53***	2.41***	2.64***	2.85***	1.55**	3.35***	1.96***
Family size	1.25***	1.15***	1.28***	1.15***	1.06	1.20***	1.24***	1.07	1.15***
Socioeconomic Status									
Income	.74***	.71***	.70***	.62***	.66***	.75***	.58***	.53***	.64***

Note: For classification of causes of death, see Table 2.

Sources: National Health Interview Survey, 1986 (NCHS 1989); National Mortality Followback Survey, 1986 (NCHS 1988a).

p ≤ .05; ** p ≤ .01; *** p ≤ .001.

socioeconomic factors *eliminate* any differences in general mortality between blacks and whites. Furthermore, once such controls are applied, the racial gaps narrow, though they do not close completely, within every cause-of-death category except suicide, where blacks have lower rates than whites. Such a finding suggests that if blacks improve their socioeconomic status (with higher incomes), modify their household status (by increasing their propensity to marry and remain married), and reduce their family size, their life expectancies should increase and the racial gap in life expectancy should decrease (see also Hollingsworth 1981; Jaynes and Williams 1989; Potter 1991; Susser et al. 1985).

The poor suffer higher mortality than the affluent, and among the poor, blacks suffer higher mortality than whites. Many poor families may not be able to muster the necessary resources to assure health, to ensure recovery of sick members, and to prevent mortality. For example, blacks are more likely than whites to contract a serious illness, but less likely to see a physician, to be hospitalized, to have private health insurance, or to have access to health care providers (Blendon et al. 1989). Yet federal programs that provided health services for the poor were cut back in the 1980s, and some hospitals have limited the number of Medicaid and Medicare patients they will treat (Cockerham 1988). Such actions will accentuate the risk of mortality among the poor, and may widen further the mortality gap between whites and blacks (Marmot, Kogevinas, and Elston 1987).

Most solutions for reducing the black/white gap in life expectancy call for medical remedies—better screening, treatment, or surgical methods. Yet it is also possible to attack the social causes of mortality, including inadequate family support, education, employment, income, and access to and use of quality medical care. For instance, lung cancer may cause mortality, but often smoking contributes to lung cancer. Medical science in many cases cannot cure lung cancer, but social scientists (and public health officials) can work to prevent smoking. Similarly, either medical professionals can screen, identify, and treat hypertension, or social programs can reduce stress and the causes of hypertension by initiating exercise and diet programs, educating the illiterate, aiding families with dependent children, providing accessible health care, and employing the jobless. Both avenues are important; neither is expendable.

It is simplistic to assume that once blacks know that sociodemographic factors improve their life chances, they will move voluntarily to more favored statuses, or that such a change will instantaneously improve their survival prospects. Unfortunately there are individual, social structural, and cohort obstacles to overcoming social inequalities. Some individuals are unable—because of age, residential location, transportation limitations, or dependent care responsibilities—to take advantage of increased employment or pay opportunities. Blacks also may contend with previous or current discrimination in education, training, hiring, retraining, or promotion (Farley and Allen 1987). In addition, period-specific social changes are never synchronized perfectly with the groups or cohorts they are intended to help, a phenomenon that Riley (1990) termed “asynchrony.” Thus social programs implemented to help blacks in general may help only young blacks, educated blacks, or those of higher socioeconomic status. Yet these pitfalls should not overshadow the fact that this paper, by identifying ways to improve blacks’ survival chances, has suggested a step toward closing the racial gap in life expectancy. Sociodemographic factors—age, sex, marital status, family size, and income—profoundly affect race differences in mortality. Everyone is at risk of dying, but how adeptly we deal with, protect against, and recover from that risk determines whether we live or die.

Notes

¹ To create the population at the beginning of the year that survived to the end of the year, I forward survived the midyear population by one-half year, using the formula in Shryock and Siegel

(1976) and the survival rates from NCHS. L_x values by age, sex, and race are provided in annual publications for ages 25 to 85, but not for ages 85 and over. Thus I used L_x values for ages 25 to 85 based on 1986 data (NCHS 1988b) and L_x values for ages 86 to 99 and over based on 1980 data (NCHS 1985), the most recent information available. The survival rates based on 1980 data tend to underestimate the population slightly: the forward-survived population is .08% (30 persons) smaller than it would have been if 1986 survival rates had been available for all ages.

² My sample includes 14,471 weighted records that represent 444 deaths. The analyses that follow would be impossible with 444 deaths because the cell sizes would be too small. Where possible, I calculated the results using two different weighting procedures, weighting the records either to the NHIS sample size or to the U.S. adult population. For example, I calculated life expectancies and created Table 1 using 1) the NHIS weighted to the U.S. adult midyear population ($N=139,969,762$) with the NMFS weighted to the U.S. adult decedents ($N=1,713,644$), and 2) the NHIS weighted to the NHIS midyear sample ($N=36,283$) and the NMFS weighted to the expected number of adult decedents in the NHIS data set ($N=444$). The findings were identical.

The multivariate calculations presented in Table 3 and the coefficients presented in Tables 4 and 5 were based on the NHIS sample. I used a weighted sample of 36,490 individuals that has been rescaled to the 50,754 actual observations.

³ The linked data set, which oversampled blacks, includes information on 3,955 black decedents and on 10,516 white decedents. Among the blacks there were 1,738 deaths due to circulatory disease, 883 to cancer, 254 to respiratory disease, 170 to accidents, 32 to suicide, 87 to infectious disease, 111 to diabetes, 154 to homicide, and 526 to other causes.

⁴ NMFS coded income, a variable with an intrinsically continuous metric, into nonequal intervals. The nonequal-interval values can be kept or the values can be collapsed into dummy codes, but at the expense of losing information. For readability, I analyzed these data using the midpoints of the within-category intervals.

⁵ There were no missing values for family size for survivors, but family size was missing in 4% of the decedent cases. The regression equation for imputing missing family size values for decedents had an R^2 of .16 and was

$$\text{famsiz} = 3.82 - .12(\text{age}) + .48(\text{race}) - .19(\text{sex}) - .87(\text{marry}).$$

Information on income was missing for 14% of the survivors and 22% of the decedents. The regression equations for imputing missing family income values for survivors and decedents had R^2 's of .22 and .16 respectively and were

$$\begin{aligned} \text{income}_{\text{survivor}} &= 2.76 + .15(\text{age}) - .02(\text{age}^2) - .70(\text{race}) \\ &+ .13(\text{sex}) - .73(\text{marry}) + .06(\text{famsiz}), \text{ and} \\ \text{income}_{\text{decedent}} &= 1.92 + .02(\text{age}) - .005(\text{age}^2) - .67(\text{race}) \\ &- .08(\text{sex}) - .36(\text{marry}) + .15(\text{famsiz}). \end{aligned}$$

Regardless of the type of value used, the final conclusions remain. For example, I reanalyzed Model 4 of Table 4 by 1) excluding missing values, 2) using the mean for missing values, and 3) allocating missing values through regression. All of the odds ratios for race were all between .98 and 1.01.

⁶ I could have included more detailed marital status categories, but because the main focus of the study is on racial differences, and because the more detailed categories (married, widowed, divorced/separated, and never married) did not contribute statistically to the full model (Model 4), I have retained the simpler marital status codes.

⁷ Although information on education was also available, previous studies have shown that income and education are highly intercorrelated. Thus income and education cannot be included together in the same multivariate analyses of mortality. Further, income contributes substantially more to the model fit than does education (Otten et al. 1990; Rogers 1991; Zick and Smith 1991).

⁸ I contend that because the black/white difference in mortality disappears when key socioeconomic factors are controlled, the racial disparity is due to these factors. A competing explanation maintains that blacks are more likely than whites to overstate their ages, especially at the greatest ages (see, for example, Rosenwaike and Logue 1983), and this age overstating artificially depresses black mortality. This explanation, if true, could dampen but would not eliminate my results.

⁹ I also tested the significance but did not find support for adding an age-squared or an age-logged term (for similar results, see Rogers 1991; Zick and Smith 1991).

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