Health Differentials between Blacks and Whites: Recent Trends in Mortality and Morbidity

KENNETH G. MANTON, CLIFFORD H. PATRICK, and KATRINA W. JOHNSON

Duke University;
Veterans Administration Medical Center,
Durham, North Carolina;
National Heart, Lung, and Blood Institute

DIFFERENCES IN THE HEALTH STATUS OF BLACKS and whites have been documented in the United States as long as health data have been collected. These differences have persisted despite large increases in life expectancy and improvements in the health status of the general population. Although some indicators of health differentials have declined, others persist and several have increased. Sources of these health differentials have been linked to (1) differences in lifestyle and risk-factor exposures (e.g., alcohol, smoking, and nutrition); (2) health consequences of low socioeconomic status (e.g., economic barriers to access to health services and a lack of health insurance due to chronic unemployment); (3) poorer knowledge of health practices; (4) more hazardous occupations and environmental exposures (e.g., exposure of children to lead); and (5) genetic factors (e.g., sickle cell trait). Because of the interaction of these multiple factors (e.g., the interactions of low education, smoking, and nutrition), the exact contribution of each factor to black-white differentials in health—or its change over time—remains unknown. Of particular importance because of its policy implications is knowledge of how

The Milbank Quarterly, Vol. 65, Suppl. 1, 1987

low socioeconomic status interacts with physical risk factors to increase differentially mortality and morbidity risks for blacks and whites.

Although we do not know the precise etiology underlying health differentials between blacks and whites, the nature of those differentials and their recent changes can be documented. In this article, we discuss health differentials as they vary by sex, age, and severity of medical conditions. Many of these health differentials are related to the generally poorer economic condition of blacks relative to whites. In our analysis, however, we do not attempt to explain the etiology of health differentials—just to document them. Therefore, we will not attempt to standardize for the effect of income and other economic differentials. A complete etiological study of the wide range of conditions we examined is beyond the scope of this overview. Furthermore, because of the paucity of economic measures in mortality and other important national health data sets, it probably could not be done comprehensively.

Black-white differentials in health will be examined by sex because of well-documented differences in health between males and females. Female life expectancy, which was about 10 percent greater than that of males in 1980, has increased more rapidly than male life expectancy for both blacks and whites. In addition, differences in the prevalence of chronic conditions and disabilities between males and females (Verbrugge 1983) suggest that we need to determine whether the factors underlying sex differentials in health are the same for blacks and whites when age is accounted for.

Health differentials between blacks and whites are examined within four broad age categories. *First*, differentials in the survival and health of infants and children are important for both their immediate health consequences and their impact on physical and mental developmental processes that affect health late in life. *Second*, differences in health among teenagers and young adults are examined because differences in economic conditions, health behavior, and risk-factor exposures will affect both current health risks (e.g., homicide, morbidity from alcohol and drug use) and will have long-range implications for the prevalence of such chronic diseases as cirrhosis, lung cancer, and diabetes. *Third*, health differentials in middle and late adulthood are important because chronic diseases most often manifest themselves at those ages. *Fourth*, health differentials for the elderly are interesting because of the observed mortality "crossover" (black mortality rates are lower than those of whites at advanced ages), the role of Medicare in this phenomenon,

and its economic and health care implications. The analysis also examines health conditions of varying severity.

In the following, we first consider differences in the demographic structure of the black and white populations—both now and projected into the future. We then examine mortality differentials and their effect on life expectancy. Next, we examine differentials in chronic and acute morbidity and associated disability. Finally, we discuss differentials in health care access, utilization, and risk-factor exposures that may affect future health differentials.

Methods and Data

We examine the black-white differentials in health using a number of methodologies and data sources. Among the methods used are several types of life tables, indicators of excess mortality, and measures of the relative risk of morbidity and disability. The data sources used include national vital statistics data, the National Health Interview Survey (NHIS), National Health and Nutrition Examination Survey (NHANES), National Long-term Care Survey (NLTCS), National Nursing Home Survey (NNHS), and National Health Examination Survey (NHES). We make only limited use of subnational data sources such as community epidemiological studies because very few have sufficient numbers of blacks to make valid comparisons with whites in the same environment. We also did not consider the many social surveys such as the Longitudinal Retirement History Study and the National Longitudinal Survey (or Parnes study) because health information was a secondary interest in those studies. Thus, we have relied on readily available national data sets whose major focus is some aspect of health.

We examined mortality changes over the periods of 1969 to 1971 and 1979 to 1981—periods unique in American history in terms of achieved life expectancy and the management of the health effects of chronic degenerative diseases at advanced ages. The morbidity, disability, and risk-factor analyses will be both cross-sectional (e.g., HIS survey results for 1979 to 1981; NLTCS 1982) and longitudinal (e.g., HES and HANES for 1960 to 1980).

Mortality is analyzed in several ways, each providing incremental information on the differentials between blacks and whites. Life ex-

pectancy at birth is perhaps the most common measure of the impact of mortality. We examine this figure as well as the life expectancy at age 65, an indication of the impact of mortality among the elderly. To indicate the differential impact of selected causes of death on life expectancy, we examine the change in life expectancy at birth which would occur if a given cause of death were eliminated. This has obvious policy implications for the allocation of health resources. We also analyze age-adjusted and age-specific death rates for different causes of deaths to see which segments of the population are affected by those causes, e.g., homicide among young black adult males.

To get a more comprehensive sense of cause-specific differentials, rates are used to calculate the number of "excess deaths" in the black population, indicating the number of deaths that would have been avoided had the age-specific death rates for a given cause for blacks been the same as those for whites. This excess death measure is a better indicator of the size of the disparity in health than are differences in mortality rates because the former indicator combines mortality rates with the size of the population at risk to determine the absolute number of deaths that are "excess." The absolute number of excess deaths more directly reflects the size of the public health problem and the resources required to correct it than does the differential in rates. The excess death measure has to be interpreted carefully because of two factors. First, only the rates for which blacks exceeded whites were analyzed. For some conditions like suicide, whites have higher mortality rates than blacks. Such white "excess" deaths were not subtracted from the black excess since white excesses were relatively infrequent at younger ages and black-white excesses must eventually "balance out" because both groups have a finite, and presumably similar life span. Second, white excess mortality risks should not be used as a norm or standard for blacks (i.e., we would obviously not wish to increase black mortality to the same levels of mortality for the conditions with white excess deaths).

More absolute health standards to assess the mortality status of blacks and whites are desirable. One such standard might be that of the lowest mortality rate achieved for a disease globally. Therefore, one could compare black infant mortality rates against those in certain Scandinavian countries. A more absolute standard based upon the best clinical judgment about what deaths are preventable is the Sentinel Health Events methodology (Rutstein et al. 1976). We used the

Sentinel Health Events (SHE) methodology to generate estimates of both black *and* white excess mortality based on expert judgment of the best achievable levels given current medical science. The policy implications of these measures are important for health delivery and utilization and other social issues (e.g., health insurance, gun control, drug use). We feel each measure of mortality risk differences is necessary in order to identify different aspects of health differentials existing between blacks and whites.

In the mortality analysis, two types of cause-specific mortality data prepared by the National Center for Health Statistics (NCHS) are examined: (1) underlying cause of death, and (2) multiple cause-of-death data. Underlying cause-of-death data contain only one medical condition reported for each of the two million deaths occurring yearly in the United States and are used in most official vital statistics publications (e.g., National Center for Health Statistics 1986a). Multiple cause-of-death mortality data, however, contain *all* causes of death listed on the death certificate by the physician (Manton and Stallard 1984; Israel, Rosenberg, and Curtin 1986). These data are useful to describe: (1) mortality at advanced ages where there is a high prevalence of multiple conditions, and (2) conditions such as diabetes that appear primarily in a contributory role on the death certificate.

One must exercise care in interpreting cause-specific mortality data because some black-white differentials could be affected by the quality of either the diagnoses reported on the death certificates or by the age reporting of the population data used to form the rates. Furthermore, care must be taken not to interpret the multiple cause data as direct measures of morbidity—they directly reflect only the reported prevalence of conditions at the time of death. Thus, although the differentials may be suggestive of morbidity differentials, the confirmation of multiple cause patterns requires the use of detailed epidemiological data. Nonetheless, the much greater information content of the multiple cause data makes them important in epidemiological studies (Israel, Rosenberg, and Curtin 1986).

In the underlying cause-of-death analysis, all deaths during the two three-year periods—1969–1971 and 1979–1981—were summed (roughly six million deaths for each interval) and an annual average computed. Then 1970 and 1980 population data were used with the averaged deaths to produce more stable estimates of mortality rates—especially for rarer causes of death among blacks. Conditions examined

were cancer, heart disease, stroke, diabetes, cirrhosis, homicide, and infant mortality. Infant mortality as used herein includes all deaths occurring in the first year of life regardless of the cause.

In our multiple cause-of-death analysis, we examine individual deaths from 1969 to 1980, yielding data on nearly 26 million deaths and nearly 70 million reported medical conditions for the total population. Among these were about 2.5 million deaths for blacks. Since both the 8th and 9th revisions of the International Classification of Disease (ICD) (World Health Organization 1969, 1977) were employed over the study period, only comparable disease categories based upon National Center for Health Statistics comparability studies (1975, 1980) were examined.

We examined morbidity using data from major national surveys of health. For example, the NHIS—an annual survey conducted since 1957—typically covered 42,000 households and 111,000 individuals. A number of changes were made in the survey methodology and questionnaire over this period that make cross-temporal comparisons of disease prevalence with NHIS data difficult. For the single time interval of 1979 to 1981, however, we examined the ratio of black-white prevalence rates for conditions that were significant in the mortality analyses. To improve the reliability of our prevalence estimates, only reports of disease involving either health service contact or associated disability were used.

The 1982 NLTC survey reports on the characteristics of community-dwelling persons aged 65 and over who manifested chronic (over 90 days) disability according to reported limitations on activities of daily living (ADL) or instrumental activities of daily living (IADL). A telephone screen of 36,000 persons over the age of 65 drawn from the Medicare Health Insurance master file yielded 6,393 persons who reported (or anticipated) an ADL or IADL limitation lasting three months or more. An intensive household interview of the 6,393 persons (representing five million elderly persons) was conducted. Results on differences in the functional limitations of elderly blacks and whites are discussed in this article.

The NHES and NHANES (1960–1962, 1971–1974, 1976–1980) involve direct clinical measurements of certain important physiological variables. For example, these surveys provide nationally representative data on trends in blood pressure, serum cholesterol, and obesity for blacks and whites. In addition, national surveys of the utilization of

acute-care hospitals (National Hospital Discharge Survey), nursing homes (NNHS for 1977 and 1973–1974, resident place surveys in 1963 and 1969), and physician visits (National Ambulatory Medical Care Survey) were employed. These were supplemented by data from select epidemiological studies and registries. For example, the mortality crossover was examined using 20-year follow-up data on total mortality from the Evans County, Georgia, study of 1,919 whites and 1,183 blacks aged 15 to 74 in 1960 (Wing, Tyroler, and Manton 1985) and in the 25-year follow-up data on total mortality for 1,388 whites and 786 blacks in the Charleston, South Carolina, heart study (Wing et al. 1987). These two closed cohort studies had extremely high rates of subject follow-up.

The primary registry source is the population-based tumor registries of the surveillance, epidemiology, and end results program of the National Cancer Institute. In 1981 the 11 registries in this program covered almost 13 percent of the entire United States population (National Cancer Institute 1984). Data in some registries have been collected since 1973. A comparison of mortality in the registry population shows good concordance with that of the American population, suggesting that the registry data are representative of national patterns.

In order to calculate rates, age-, sex-, and race-specific population estimates are needed. These estimates are subject to considerable error, especially for blacks at older ages. In forming rate estimates, we used either U.S. Bureau of the Census population figures adjusted for various race, sex, and age reporting errors (Siegel 1979) or the sum of population weights used in various surveys (e.g., the NHIS).

The Demographic Structure of the United States Black and White Population

We examined the age structure of the American white and black population by sex as estimated for 1985 and projected for the years 2000 and 2050. The age structure of the population is very different so that even if the age- and sex-specific morbidity and mortality rates were the same in the two populations, the total health needs of the two populations would be quite different. The population structure differences arise because the black population has significantly higher fertility rates than the white, resulting in the black population being

much younger on average and having higher proportions of its population at younger ages. Thus, diseases of infancy and conditions affecting adolescents and young adults (e.g., homicide, accidents, drug abuse) will be proportionately more important in the black population because of the greater proportions of persons at young ages who have the highest risk of those conditions.

The difference in the age structure of the American black and white populations estimated in 1985 and projected to 2000 and 2050 is reported separately by sex in table 1.

The table contains the number of persons in each age category, the proportion that number is of the total race- and sex-specific population, the underlying fertility rate, and the mean age. The black fertility rate is 22 percent greater than that for whites in 1985. Even if there were no excess mortality risks, the relative number of infant deaths would be higher for blacks. For persons under the age of 5 and for those aged 15 to 24, the proportion of the black population is 31 and 18 percent higher than for whites. This increases for blacks the relative significance of diseases and causes of death that are more prevalent at these ages—i.e., for infants, teenagers, and young adults.

Table 1 also contains the projected population age structure of the black and white population for the years 2000 and 2050. The age structure of the black population is projected to continue to be younger than for whites, though because of the assumed convergence of the fertility rates the differentials become smaller. For example, the 10.1 year difference in the median age for females declines to 6.8 years by 2000 and 4.9 years by 2050. While the differential in median age does decrease, it remains substantial to 2050. Both black and white populations age rapidly, however, so that the aggregate health needs of both groups will become increasingly weighted toward the chronic disease risks of middle and late ages and the health differences at younger ages will become less significant—even without changes in morbidity and mortality rates. Nonetheless, significant age-related health differentials will continue to exist between blacks and whites in the coming decades.

Mortality

Life Expectancy: At Birth and in Late Life

Life expectancy has risen dramatically in the United States in recent years for both blacks and whites. In figure 1, the sex-specific increases in life expectancy at birth for blacks and whites from 1950 to 1982 are presented.

All four race-sex groups have experienced large gains, with blacks experiencing faster gains than whites. Nonetheless, significant black-white differences in life expectancy remain for both males and females. For example, in 1980, a 6.3-year difference in life expectancy (70.6 vs. 64.3 years) remained between white and black males and a 5.1-year difference (78.2 vs. 73.1 years) existed between white and black females. Both values represent substantial declines in the sex-specific differences from 1970.

Recently, both races have had significant increases in life expectancy at advanced ages. Changes in life expectancy at age 65 from 1960 to 1983 are presented in table 2.

Significant life expectancy gains at age 65 have been made for all race and sex groups, especially from 1970 to 1983. Between 1960 and 1970 the pace of change for males was slow. From 1970 to 1983 it accelerated, increasing 1.4 years for white males and 0.7 years for black males. For white females, the rapid gain from 1960 to 1970 accelerated from 1970 to 1983. The pace of improvement after 1970 for black females was slower than for white females—but a little faster than for white males. Gains after 1980 for both black males and females have been very rapid.

While life expectancy *at birth* for blacks and whites converged over time, until recently life expectancy after age 65 diverged. For males, this divergence between blacks and whites is considerable, with the differences in life expectancy increasing from 1960 to 1983. For females, the difference between blacks and whites increased from 1960 to 1980, then dropped slightly in 1983. The divergence could be explained in a number of ways. First, the divergence could be due to better recent age reporting for blacks at advanced ages, which reduced recent estimates of the elderly black population, thereby increasing estimates of mortality rates. An improvement in age-reporting accuracy could mask gains in life expectancy for blacks. Second, rapid

TABLE 1

Number of Persons (in Thousands) by Age Group, Sex, and Race; Percentage; Median Age; and Fertility Rates for Females 1985, 2000, and 2050, Middle Series

Age group	1985 White males	1985 White females	1985 Black males	1985 Black females	2000 White males	2000 White females	2000 Black males	2000 Black females	2050 White males	2050 White females	2050 Black males	2050 Black females
					NUMBER							
<1	1,561	1,479	319	312	1,394	1,321	312	305	1,346	1,275	331	323
1–4	6,041	5,732	1,225	1,200	5,713	5,416	1,245	1,218	5,440	5,156	1,338	1,306
5–14	14,004	13,305	2,521	2,483	15,634	14,836	3,243	3,178	13,918	13,199	3,439	3,359
15–24	16,784	16,098	2,870	2,862	14,843	14,160	2,852	2,819	14,042	13,377	3,470	3,410
25–44	31,565	31,376	4,050	4,566	33,324	32,622	5,450	5,678	29,629	28,748	6,920	6,965
45–64	18,870	20,420	1,907	2,431	25,589	26,676	2,879	3,599	28,691	28,948	5,920	6,290
65–74	6,636	8,554	579	831	6,953	8,636	615	974	11,257	12,685	1,979	2,424
75–84	3,034	5,176	271	459	4,212	6,881	334	641	7,131	10,100	1,109	1,750
85+	697	1,780	60	129	1,217	3,227	110	302	4,046	9,326	545	1,419
Total	99,192	103,920	13,802	15,273	108,879	113,775	17,040	18,714	115,500	122,814	25,051	27,246
					PERCENTAGE							
<1	1.57%	1.42%	2.31%	2.04%	1.28%	1.16%	1.83%	1.63%	1.17%	1.04%	1.32%	1.19%
1–4	6.09	5.52	8.88	7.86	5.25	4.76	7.31	6.51	4.71	4.20	5.34	4.79
5–14	14.12	12.80	18.27	16.26	14.35	13.04	19.03	16.99	12.05	10.75	13.73	12.33
15–24	16.91	15.50	20.79	18.74	13.63	12.45	16.73	15.06	12.16	10.90	13.85	12.52
25–44	31.81	30.19	29.34	29.90	30.61	28.66	31.98	30.34	25.65	23.41	27.62	25.55
45–64	19.02	19.65	13.82	15.91	23.50	23.45	16.90	19.23	24.84	23.56	23.63	23.09
65–74	6.69	8.23	4.20	5.44	6.39	7.59	3.61	5.20	9.75	10.33	7.90	8.90
75–84	3.09	4.98	1.96	3.01	3.87	6.05	1.96	3.43	6.17	8.22	4.43	6.42
85+	0.70	1.71	0.43	0.84	1.12	2.84	0.65	1.61	3.50	7.59	2.18	5.21
Total	100.00	100.00	100.00	100.00	100.00	100.00	100.00	100.00	100.00	100.00	100.00	100.00
					MEDIAN AGE							
	31.1	37.7	24.9	27.6	36.1	38.8	28.5	32.0	40.5	44.8	36.3	39.9

	Fertility rates					
	1985		2000		2025	2050
	White Females	Black Females	White Females	Black Females	White Females	Black Females
10–14	0.8	4.2	0.7	3.0	0.7	1.8
15–19	45.0	98.5	44.9	85.5	44.9	65.2
20–24	109.9	145.5	112.6	140.7	114.0	128.2
25–29	115.7	113.0	120.4	118.3	121.8	120.7
30–34	65.2	68.0	74.0	76.5	71.9	73.1
35–39	19.8	26.3	23.1	28.9	22.9	25.8
40–44	3.6	6.0	3.6	5.5	3.6	4.6
45–49	0.2	0.3	0.2	0.2	0.1	0.2
Total	1,800.6	2,309.1	1,897.4	2,292.7	1,900.0	2,098.1

Source: U.S. Bureau of the Census 1984.

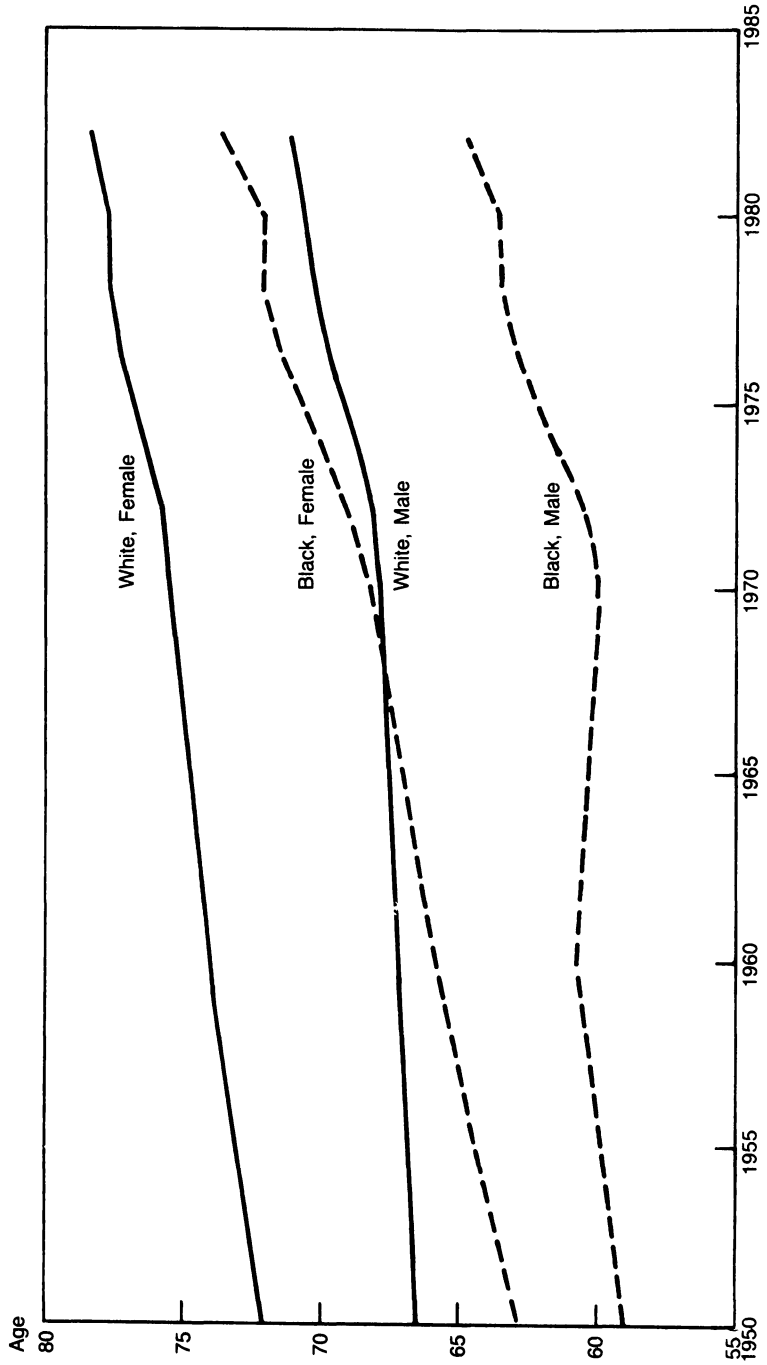

FIG. 1. Life expectancy at birth, according to race and sex: United States, 1950–1983. *Source:* U.S. Department of Health and Human Services 1985, 66 (figure 2).

TABLE 2
Life Expectancy at Age 65: Changes 1960 to 1983

Year	Males		Difference between whites & blacks	Females		Difference between whites & blacks
	Whites	Blacks		Whites	Blacks	
1960	12.9	12.7	0.2	15.9	15.1	0.8
1970	13.1	12.5	0.6	17.1	15.7	1.4
1980	14.2	12.9	1.3	18.5	16.5	2.0
1983	14.5	13.2	1.3	18.9	17.2	1.7
	Number of years change between time interval					
1960–1970	0.2	−0.2		1.2	0.6	
1970–1980	1.1	0.4		1.4	0.8	
1980–1983	0.3	0.3		0.4	0.7	
1960–1983	1.6	0.5		3.0	2.1	

decreases in mortality at younger ages may have increased the number of blacks with chronic conditions surviving to advanced ages. An increase in the prevalence of chronic conditions among elderly blacks would make mortality reductions at those advanced ages more difficult to achieve. Third, special health care and nutritional programs initiated in the 1960s and 1970s may have served to improve the relative health status of black infants and mothers in some areas of the country. Less program specialization and outreach specifically for black elderly may have served to retard their improvement relative to that of whites, as suggested by large differentials in institutionalization rates above the age of 85—trends which may be accelerated by current efforts to reduce Medicaid and Medicare benefits. Identifying the contribution of each of these three factors to the slower rate of improvement in black life expectancy at later ages is a topic for further research; currently, data on black-white health differentials at later ages are insufficient to answer these questions.

A second implication of recent life expectancy gains at advanced ages is the rapid growth of the "oldest old" population (i.e., those aged 85 and over) with its attendant high per capita needs for both acute and long-term care (LTC) health services (Manton and Soldo 1985). The rapid growth of the oldest old population, shown in table 1 to occur for blacks as well as whites, is important to remember

when examining black-white differentials in disability rates from the 1982 NLTCS and nursing home rates from the 1977 NNHS.

The higher disability rates of the black elderly, their rapid growth in numbers, and smaller socioeconomic resources suggest the need to develop programs to provide LTC services that are targeted to the special family and social conditions of minority elderly. Whether privately or publicly funded, paid LTC services are normally viewed as a supplement to the informal care resources of the individual (Liu, Manton, and Liu 1985). Children represent a major source of informal care for elderly females (Maonton and Liu 1984). It will be important to determine differences in the availability of such informal care resources for elderly black and white females. For example, do the generally lower socioeconomic resources of black children hinder them from performing informal care services at the same rate as for whites? On the other hand, does the more fluid, extended family structure of blacks serve to increase the availability of informal care resources?

The Trajectory of Black and White Mortality Rates at Later Ages

One issue often raised when examining black and white mortality differences at advanced ages is the so-called black-white mortality crossover (Manton 1980; Nam and Ockay 1977). U.S. national vital statistics show that black mortality rates at advanced ages converge with and then drop below white mortality rates. The reality of the crossover has been debated by a number of authors. Ryder (1985), for example, argues that such a crossover may be due to higher rates of age misstatement at later ages. The age at which the crossover occurs has increased in more current data, suggesting that the crossover may not be real and will disappear as the quality of age reporting for blacks at later ages is improved. Life expectancy among blacks at later ages, however, is also increasing; indeed, between 1980 and 1983 it increased faster for black than white females. Thus, if the crossover was due to higher rates of systematic mortality selection among blacks at early ages, then as life expectancy increases (and greater proportions of blacks survive to later ages), the effects of selection also ought to disappear and the crossover should advance to later ages.

The trajectories of mortality rates that produce the crossover are

manifest even in middle age. An age-reporting explanation of the crossover would involve age misreporting in early middle age and would have to affect causes of death differently, since the crossover can be identified with disease-specific components of the total force of mortality. With higher black mortality rates at younger ages, a crossover must occur if the life span of both racial groups is the same and the maximum observed ages to which blacks and whites survive is the same. For example, Fries (1980, 1983) has argued that we are currently near a rectangularization of the survival curve in the United States and other developed countries, with deaths at very advanced ages (e.g., 85 and over) increasingly due to basic biological senescence. If the deaths of both blacks and whites at extreme ages are largely due to biological senescence, and the genetically determined life span is the same for both blacks and whites, then a crossover must eventually occur to compensate for higher black mortality rates at younger ages. Others argue that we are not currently manifesting a rectangularization of the mortality curve at advanced ages (e.g., Schneider and Guralnik 1986; Myers and Manton 1984a, 1984b; Manton 1986). In this case there would be no absolute requirement that mortality crossover must occur because of life span constraints.

Whatever arguments are made, there is probably no way to resolve whether the crossover exists using census and vital statistics data alone. An alternative approach is to examine epidemiological data where (a) one is examining a closed cohort for a lengthy period of time, and (b) one has covariate information on the risk-factor characteristics of people who die at different ages. The existence of observed risk-factor heterogeneity and its effect on mortality trajectories has been examined in a number of longitudinal studies (Manton et al. 1985). The extreme heterogeneity of risk-factor exposure within the black population could produce a crossover through systematic mortality selection. We will examine results from an analysis of the sex-specific mortality patterns for blacks and whites in two large, closed-cohort epidemiological studies (i.e., the Charleston heart study and the Evans County, Georgia, study [Wing et al. 1987]). Because these are closed-cohort designs with lengthy follow-up, it is possible to see if a crossover occurs during the course of observation. If so, then no assumption needs to be made about the quality of age reports, even though such data are probably much better in these epidemiological studies than in vital statistics.

TABLE 3
Results from a Pooled Analysis of Total Mortality in the Evans County and Charleston Heart Studies

	Gompertz Hazard	Weibull Hazard
A. One degree of freedom χ^2 associated with hypothesis that shape parameters of hazard function are equal for blacks and whites (scale parameters were separately estimated for blacks and whites to allow for different levels of risk).		
Males	2.93	4.47
Females	11.90	13.87
Total (2 d.f.)	14.83	18.34
B. Value of shape parameters (value for Gompertz is multiplied by 100)		
White males	7.92	6.02
Black males	6.98	5.23
White females	9.51	7.32
Black females	7.52	5.77

Cohort mortality was modeled using separate sex-specific hazard functions for blacks and whites in a pooled analysis of the two data sets. Two alternate hazard functions, the Gompertz and the Weibull, were used to see if the form of the hazard function affected the results. Separate constants were used for each subpopulation and a test made to see if the same exponential parameter, which determines the shape of the hazard function across age, could be used across race within sex. The results are presented in table 3.

The hypothesis of equal shape parameters (one-tailed directional test) is rejected for both males and females with the shape parameter for whites being higher than that for blacks. The higher value of the white shape parameter means that mortality rates rise faster with age for whites; the whites had a smaller constant parameter meaning their initial mortality is lower—hence the "crossover." Demonstrating a crossover in two tightly controlled closed-cohort studies which, because of their relative homogeneity, should probably show less of a tendency to converge and crossover than the national data is important evidence for a crossover. Preliminary examination of published tabls from other

studies (e.g., Alameda County [Berkman and Breslow 1983]) also seem suggestive of a "crossover."

Cause-specific Mortality

Given the rapid improvements in life expectancy for all groups and the persistence of a relative deficit for blacks, it is important to identify factors that underlie mortality changes. One factor is the difference in infant mortality. Black rates are about twice as high as white rates. The pattern of decline in infant mortality rates for blacks and whites from 1950 to 1982, portrayed in figure 2, indicates this relatively constant difference in levels.

For both blacks and whites, the rate of decline accelerates about 1965. The rapid rate of decline persists to at least 1982. Despite the decline in infant mortality rates, black-white differentials persist. Even the United States white infant mortality rate is high relative to many other developed nations. The 1984 infant mortality rate in Japan is 6.0 (World Health Organization 1985), 40 percent lower than the 1982 United States white infant mortality rate of 10.1 per 1,000 live births (U.S. Bureau of the Census 1985). Thus, the American black infant mortality rate appears even more excessive when compared to the rate of other developed nations. Indeed, the 1982 American black infant mortality rate of 19.6 per 1,000 (U.S. Bureau of the Census 1985) does not compare favorably against less developed countries such as Cuba (infant mortality rate of 18.5 per 1,000 in 1981), Costa Rica (24.3 in 1979), or Panama (20.4 in 1983) (World Health Organization 1982, 1985), if we accept their reported rates as accurate. Indeed, infant mortality rates in certain states (e.g., Illinois and Michigan both with a black infant mortality rate of 24.6 per 1,000, or the District of Columbia with 24.1 [U.S. Bureau of the Census 1985]) begin to approach those in some African nations (e.g., 27 per 1,000 in Mauritius in 1982 [World Health Organization 1985]).

The black-white differential in infant mortality has persisted because of a number of factors. These include socioeconomic differences between black and white mothers which contribute to poorer prenatal care and nutrition, higher rates of teenage pregnancy, greater parity, and other risk factors.

Infant mortality differentials, while contributing significantly to

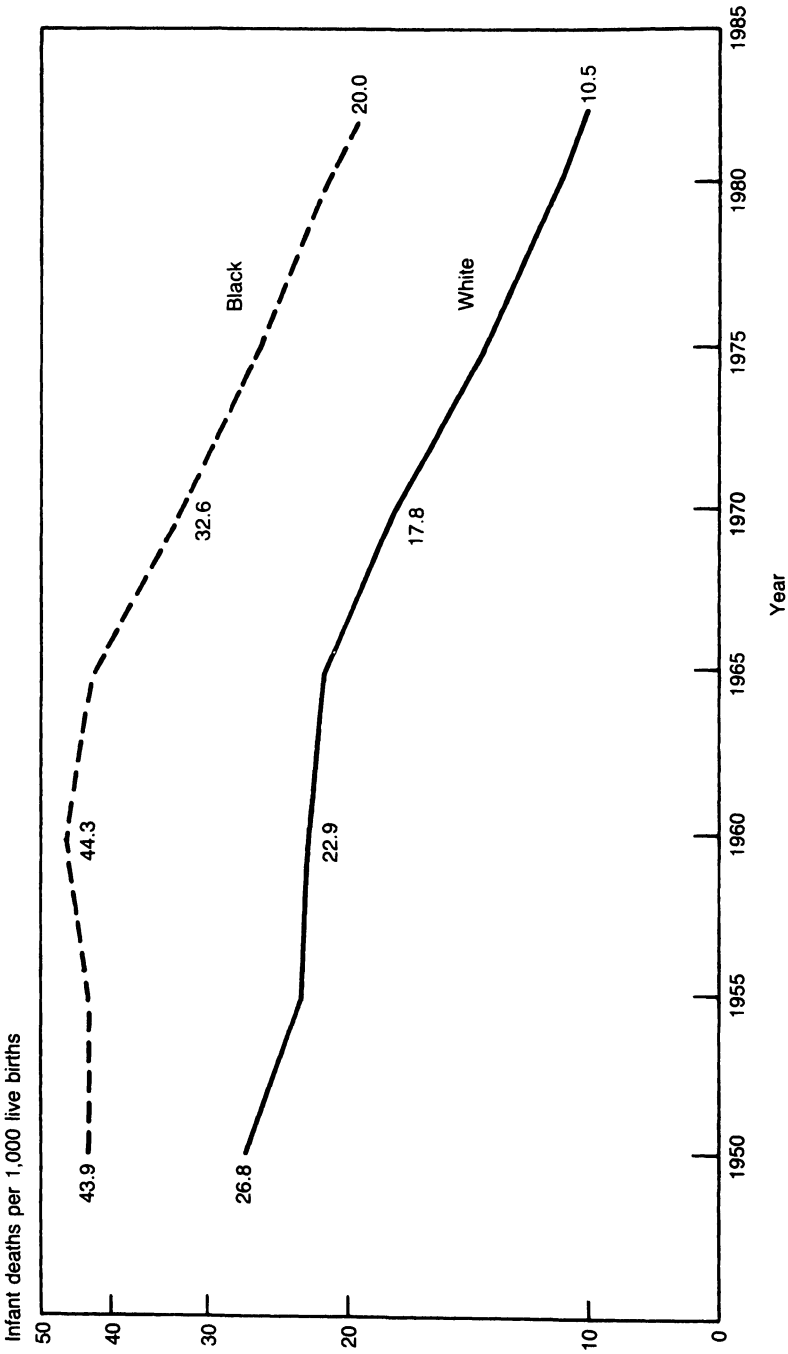

FIG. 2. Infant mortality rates, 1950–1982.
Source: U.S. Department of Health and Human Services 1985, 172 (figure 14).

black and white life expectancy differences, are only one source. A second major source results from chronic diseases, conditions, and external events that produce mortality primarily in mid and late life. This is illustrated in the age-standardized cause-specific mortality rates in table 4.

The age-adjusted death rates for blacks are about 50 percent higher than those of whites for both males and females. The biggest black-white differential is for homicide, an external cause of death most prevalent among adolescents and young adults. Diabetes is the second most elevated cause of adult death for blacks, probably reflecting nutritional factors, including higher prevalence of obesity among black females and possible differences in the management of disease. Cirrhosis, associated with alcohol abuse, is twice as high for blacks, while stroke and heart disease, and circulatory conditions associated with hypertension and diabetes, are also higher.

We also examined mortality differentials in multiple-cause mortality data (data on all conditions reported on the death certificate). Multiple-cause data are important in describing mortality at later ages where the prevalence of multiple chronic diseases is higher and for certain diseases (e.g., diabetes, cirrhosis, pneumonia, septicemia) frequently reported as contributing to death but not being the underlying cause (Israel, Rosenberg, and Curtin 1986).

Table 5 contains the proportion of deaths for American blacks and whites in 1980 from selected causes (diabetes mellitus, stroke, pneumonia) where the condition is: (1) the underlying cause of death (UCD); and (2) mentioned anywhere on the certificate as contributing to death (TM). Table 5 also shows the ratio of the total occurrence on the death certificate of the selected causes to only their underlying cause occurrence. The proportions are estimated for life table populations so the effect of population structure has been removed. The ratio of the two proportions indicates the relative importance of the condition as a contributing cause of death. These statistics are reported for four ages (birth, 45, 65, 85).

In general, whites have a higher ratio of total mentions to underlying-cause mentions than blacks. The ratios can be quite large (e.g., for white males there are 4.5 times as many deaths where diabetes is recorded on the death certificate than there are deaths where diabetes is selected as the underlying cause). Significant reporting of conditions in a nonunderlying-cause role was observed for most major chronic

TABLE 4
Age-adjusted Death Rates by Selected Cause, Race, and Sex, 1980 (per 100,000 population)

	Black male	White male	Ratio	Black female	White female	Ratio
Total deaths (all causes)	1,112.8	745.3	1.5	631.1	411.1	1.5
Heart disease	327.3	277.5	1.2	201.1	134.6	1.5
Stroke	77.5	41.9	1.9	61.7	35.2	1.8
Cancer	229.9	160.5	1.4	129.7	107.7	1.2
Infant mortality	2,586.7	1,230.3	2.1	2,123.7	962.5	2.2
Homicide	71.9	10.9	6.6	13.7	3.2	4.3
Accidents	82.0	62.3	1.3	25.1	21.4	1.2
Cirrhosis	30.6	15.7	2.0	14.4	7.0	2.1
Diabetes	17.7	9.5	1.9	22.1	8.7	2.5

Source: National Center for Health Statistics 1983, tables 9, 15.

TABLE 5

A Comparison of Underlying and Multiple-cause Mortality Reporting for Diabetes, Stroke, and Pneumonia: 1980

	White males			Black males			White females			Black females		
Age	UCD*	TM**	RATIO***	UCD	TM	RATIO	UCD	TM	RATIO	UCD	TM	RATIO
						DIABETES						
Birth	1.3	5.8	4.5	1.7	5.5	3.2	2.0	7.8	3.9	3.4	10.2	3.0
45	1.4	6.1	4.4	1.8	6.0	3.3	2.0	8.0	4.0	3.6	10.8	3.0
65	1.4	6.3	4.5	1.8	6.1	3.4	2.0	8.1	4.1	3.4	10.4	3.1
85	1.1	4.8	4.4	1.2	4.1	3.4	1.4	5.5	3.9	2.4	6.6	2.8
						STROKE						
Birth	7.3	12.5	1.7	8.4	13.4	1.6	12.2	19.7	1.6	12.6	20.1	1.6
45	7.8	13.4	1.7	9.2	14.8	1.6	12.6	20.4	1.6	13.2	21.2	1.6
65	8.9	15.4	1.7	10.6	17.2	1.6	13.6	22.0	1.6	14.3	23.1	1.6
85	11.7	19.4	1.7	11.5	18.4	1.6	15.7	25.1	1.6	15.6	24.2	1.6
						INFLUENZA AND PNEUMONIA						
Birth	3.0	9.3	3.1	3.0	8.2	2.7	3.6	9.7	2.7	2.7	7.1	2.6
45	3.1	9.8	3.2	3.2	8.8	2.8	3.7	9.9	2.7	2.7	7.2	2.7
65	3.6	11.0	3.1	3.6	9.7	2.7	4.0	10.5	2.6	3.0	7.8	2.6
85	6.3	15.7	2.5	5.5	12.6	2.2	5.6	13.3	2.4	4.1	9.8	2.4

*UCD = Underlying cause of death on the death certificate
**TM = Total mentions of causes on the death certificate
***RATIO = TM/UCD

diseases; even for cancer there are 15 percent more deaths (or 60,000 per year) affected than are recorded in underlying-cause statistics (Manton 1986). Part of the greater multiple-cause occurrences for whites may be due to differences in death certification and medical care that leads to more complete diagnosis and treatment of conditions among whites. Another source of the higher rate of multiple-cause reporting among whites may be better survival of whites with chronic disease (because the case fatality rates for many chronic diseases such as cancer are higher for blacks than for whites). Consequently, whites may live longer with a chronic disease, thereby increasing the period of exposure for a second (or multiple) chronic disease to emerge.

In examining the individual conditions, the ratio of total to underlying-cause mentions for diabetes is lower for blacks than for whites. Females of both races have proportionally more deaths affected by diabetes, with over 10 percent of black female deaths associated with diabetes. The lower rate of multiple-cause reporting of diabetes for blacks may occur because the disease is more lethal for blacks, possibly due to later diagnosis and less access to adequate medical and personal resources for the long-term management of the disease. Alternatively, the lower rate may be due to less complete reporting of diabetes on death certificates for blacks. The latter explanation seems less likely because higher proportions of black female death certificates report diabetes overall than do those of white females. One would not expect an excess of diabetes among black females were it less completely reported.

The proportion of deaths due to diabetes in the life table population declines between ages 65 and 85, indicating that much of the mortality due to diabetes occurs in that age range. This occurs both for the underlying-cause and total occurrences of diabetes on the death certificate. thus, diabetes is less significant, either as an underlying or associated cause of death, at advanced ages for all race/sex groups. As the mortality risks for conditions associated with diabetes such as stroke decline, diabetes reported at death at advanced ages may increase (Baum and Manton 1987).

The underlying-cause-of-death rate for stroke has declined 44 percent from 1968 to 1982. Besides being an important cause of death, stroke is an important determinant of disability and nursing home use, accounting for about 7 percent of all nursing home days used in 1977 (Liu and Manton 1984). Compared to diabetes (a condition with which

it is strongly associated), stroke is more important as an underlying cause of death at advanced ages—i.e., the proportion of deaths affected by stroke increases with age. After age 85 almost a quarter of both black and white female deaths are affected by stroke. Despite being a frequent underlying cause of death, stroke affects about 60 percent more deaths than is reported in the underlying-cause-of-death data. The proportion of deaths due to stroke in the life table population is similar for black and white females (even though the mortality rates are 80 percent higher for blacks) because the white female population has a higher life expectancy and stroke is predominantly a cause of death at very advanced ages. The stroke mortality rate for black males is much higher than for white males up to age 65 though we can again see the impact of greater white male survival to later ages, which increases the proportion of deaths from chronic diseases. Thus, the life table results show different aspects of the impact of the disease than the age-adjusted rates.

Influenza and pneumonia are frequent contributory factors in the deaths of debilitated, elderly persons (Besdine 1984). Within sex, blacks have smaller proportions of deaths affected by pneumonia, consistent with higher disease-specific case-fatality rates (or deaths per 100 illnesses) for blacks. This implies a shorter exposure period for the acquisition of complicating conditions like pneumonia.

The multiple-cause data can also be examined over time. Figure 3 records four plots which show the change from 1968 to 1980 in the life table proportion of deaths affected by four conditions (septicemia, cirrhosis, pneumonia, and stroke) for blacks and whites.

Septicemia is frequently a complication of chronic debilitation and has been rapidly increasing as a mortality risk in the United States—especially at advanced ages (Manton 1986). It may also reflect inadequate medical care leading to complicating infections. Septicemia has increased as a cause of death for all race/sex groups, but it affects more nonwhite deaths (e.g., nearly 5 percent of nonwhite female deaths in 1980), and increases more rapidly for nonwhites (figure 3). Table 6 contains sex and age-specific septicemia mortality life table statistics for blacks and whites.

The absolute increases in the impact of septicemia on mortality have been larger for blacks at all ages than for whites. If the increase in septicemia mortality was due to increases in the prevalence of chronic diseases at advanced ages (associated with rapid increases in

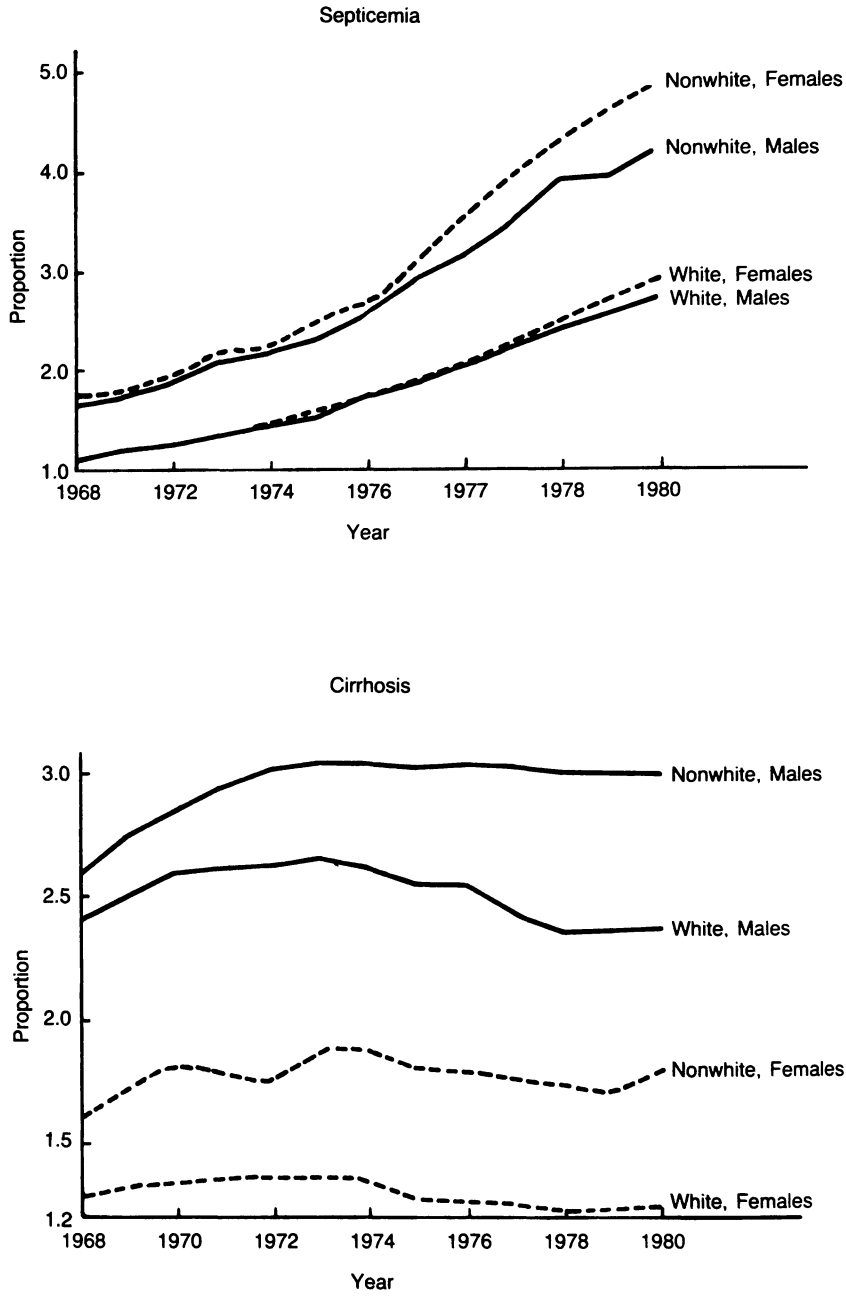

FIG. 3. Total proportion of deaths affected by four conditions, 1968 to 1980, for U.S. white and nonwhite males and females.
Source: Tabulations of national multiple cause of death files.

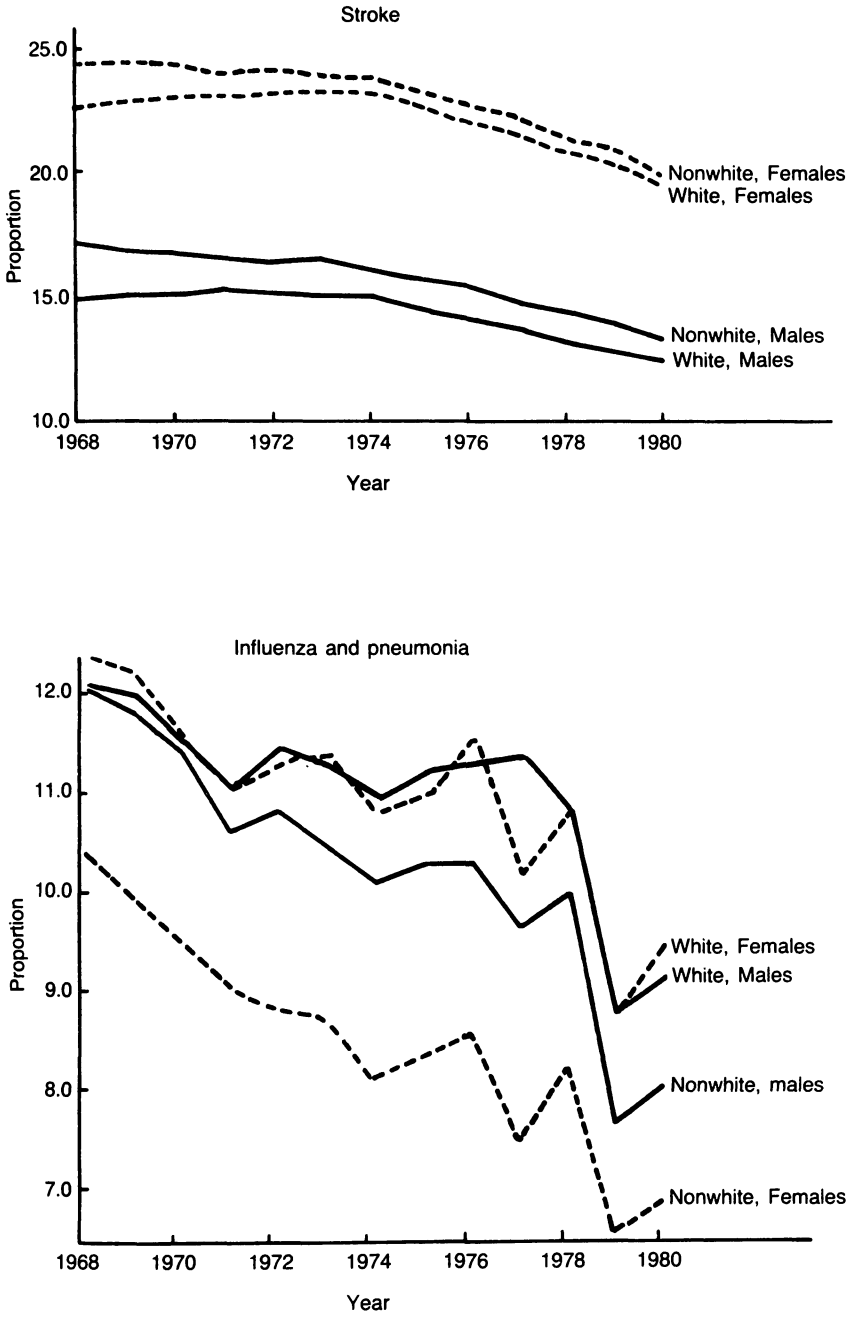

FIG. 3. Continued.

TABLE 6
Proportion of Deaths Due to Septicemia Expected after a Given Age in a Life Table Population: Underlying Cause (UC) and Total Mentions (TM), 1968 and 1980

Age	1968		1980		1968		1980	
	Underlying cause	Total mentions	Underlying cause	Total mentions	Underlying cause	Total mentions	Underlying cause	Total mentions
	WHITE MALES				WHITE FEMALES			
At Birth	0.12	1.08	0.41	2.67	0.12	1.08	0.48	2.87
45	0.10	1.03	0.43	2.73	0.10	1.01	0.48	2.88
65	0.10	1.03	0.45	2.79	0.09	0.93	0.49	2.83
75	0.10	1.00	0.49	2.87	0.08	0.85	0.50	2.76
85	0.11	0.92	0.52	2.93	0.07	0.69	0.49	2.55
	NONWHITE MALES				NONWHITE FEMALES			
At Birth	0.22	1.67	0.79	4.16	0.21	1.75	0.95	4.81
45	0.18	1.65	0.84	4.40	0.17	1.61	0.98	4.91
65	0.19	1.64	0.91	4.71	0.16	1.47	1.02	4.93
75	0.18	1.56	0.97	4.85	0.15	1.34	1.07	4.94
85	0.18	1.34	1.10	5.12	0.16	1.31	1.07	4.73

Source: Tabulation of U.S. mortality data.

life expectancy at later ages), one would have expected white females at very advanced ages to be at higher risk. The more rapid increase for blacks suggests that health differentials not related to age, but possibly to inappropriate or inadequate medical care, may explain recent increases.

In figure 3, cirrhosis shows little evidence of a systematic trend after rising to its 1972 levels. Males of both races have greater proportions of death affected by cirrhosis than females; within sex, blacks have more cirrhosis mortality. One major risk factor for cirrhosis is excessive alcohol consumption so that the pattern observed for cirrhosis suggests greater alcohol abuse among blacks.

Like septicemia, pneumonia and influenza are infectious diseases that affect debilitated persons. In contrast to septicemia, however, pneumonia is (1) decreasing in the proportion of deaths affected (part of the decrease from 1978 to 1979 is due to the changes in ICD revision); and (2) is less significant for blacks. This difference may result because septicemia seems to affect persons at all ages, whereas death from pneumonia is concentrated at later ages (Manton 1986). Hence, current problems with infectious diseases as contributing causes of death are becoming more related to race (increasing septicemia risks at all ages) and becoming less related to age (decreasing pneumonia risks at later ages).

Stroke is a major cause of death that has declined rapidly as an underlying cause of death. The pattern of change is also evident in figure 3 in its multiple-cause occurrences, with significant change after 1972, the year in which the national initiatives on hypertensive control began (Robins and Baum 1981). Interestingly, stroke mortality is less important for black males than for white females despite the latter's better control of hypertension (Wassertheil-Smoller et al. 1979).

How do various conditions contribute to life expectancy differentials between blacks and whites, and how do those contributions change over time? We present in table 7 two life table statistics, the mean age at death and the proportion of deaths caused by a given condition in a life table population for seven underlying causes of death. By examining the mean age at death in a life table population, differences in the mean age at death for a condition due to population structure are removed. By removing the effects of these differences, the cause-specific risk may be compared for 1970 and 1980.

The mean age at death from stroke increased for both blacks and

TABLE 7
Mean Age at Death and at Birth for Underlying Cause Occurrence of Seven Causes of Death, and Proportion of Deaths Due to Each Cause by Race, 1970 to 1980

Cause of death	1970	1980	1970	1980
	MEAN AGE AT DEATH			
	White males		Black males	
Stroke	76.5	78.4	70.2	72.1
Cancer	68.3	70.5	65.6	67.7
Lung cancer	66.5	68.8	62.9	65.1
Heart disease	72.1	74.3	68.7	70.3
Homicide	40.5	38.7	36.6	37.9
Diabetes	70.6	72.5	64.6	67.6
Cirrhosis	58.8	60.4	49.9	52.5
	White females		Black females	
Stroke	81.3	83.5	75.7	79.0
Cancer	69.8	71.9	66.4	69.6
Lung cancer	66.7	68.3	62.9	65.1
Heart disease	80.2	82.4	75.5	78.2
Homicide	41.9	42.0	37.3	38.7
Diabetes	75.3	77.6	69.3	73.8
Cirrhosis	59.9	62.9	49.8	54.2
	PROPORTION OF DEATHS DUE TO CONDITION			
	White males		Black males	
Stroke	9.5	7.2	10.5	8.1
Cancer	16.9	21.0	15.3	21.5
Lung cancer	4.9	6.9	4.2	6.8
Heart disease	42.2	41.0	31.3	32.5
Homicide	0.5	0.7	4.8	4.4
Diabetes	1.5	1.3	1.6	1.6
Cirrhosis	1.7	1.5	2.1	2.1
	White females		Black females	
Stroke	15.1	12.3	16.0	12.6
Cancer	16.0	18.6	13.6	17.3
Lung cancer	1.3	2.6	1.0	2.1
Heart disease	42.1	42.5	37.0	38.8
Homicide	0.2	0.2	1.0	0.9
Diabetes	2.5	2.0	3.7	3.4
Cirrhosis	0.9	0.8	1.3	1.2

Source: Tabulations of U.S. underlying cause data.

whites (e.g., 2.2 years for white females and 3.4 years for black females). The increase is evident even at age 60 for both blacks and whites. Large declines in the proportion of deaths due to stroke for both blacks and whites are also evident. Hence, improvements have occurred in both measures for stroke for blacks. The rate of improvement is more rapid for blacks than whites, though much of the *excess* risk for black males continues to 1980.

Significant race differentials are noted for cancer. The mean age at death from cancer increased for both blacks and whites, although it was lower for blacks in 1970 and 1980. The mean age at death from cancer varies by five years among the four race/sex groups. The proportion of deaths due to cancer increases for all race and sex groups, but more rapidly for blacks. The increase is largest for black males, with the proportion of cancer deaths increasing from 15.3 to 21.5 percent. This proportion is lower for black females at birth, but rises at later ages to white female levels.

The proportionate contribution of lung cancer to mortality is similar for both black and white males (4.9 and 4.2 percent, respectively), with both groups experiencing large increases. The contribution to mortality for females increased faster over the period, doubling for both races. White female lung cancer is significantly higher than black female lung cancer, in contrast to males. Because these trends can be traced to smoking patterns in different cohorts, continued increases in female lung cancer mortality rates for 20 years are likely, while white male rates may have reached their peak (Harris 1983).

Cancer trends are difficult to evaluate because they represent a large number of different diseases. For example, much of the increase in cancer mortality is due to lung cancer, which is driven by cohort patterns of smoking habits (Harris 1983). Crude breast cancer rates, especially for white females, are also increasing, possibly because of later ages at first pregnancy (MacMahon et al. 1970). In contrast, many types of cancer have declining mortality rates, such as stomach cancer and leukemia. Different types of cancer have different rates of incidence and different risk factors.

As a consequence, black-white differentials have to be examined for specific cancer types and for different types of rates. For example, there are higher age-adjusted black mortality rates for a number of cancers such as cancer of the bladder, cervix, esophagus, lung, prostate, and stomach. Among whites, there are higher crude mortality rates

from cancer of the breast, leukemia, lymphoma, and pancreatic cancer (National Center for Health Statistics 1986a). The crude rates reflect the different age structure of the black and white populations, with whites having higher crude rates for cancer types that are prevalent at later ages.

Though the range of cancer types makes the study of black-white differences complex in contrast to many other chronic diseases, good race-specific data on the incidence and case fatality of specific diseases are available from the National Cancer Institute's (1984) SEER program, a system of population-based tumor registries covering 11 areas representing almost 13 percent of the American population. We examined the site-specific cancer data to identify the causes of mortality differences to determine if they are a result of differences in disease incidence, case fatality differences due to different patterns of treatment, or due to poorer general health.

The SEER data for 1978-1981 show an 11 percent higher cancer incidence rate for American blacks relative to American whites, with most of the excess occurring in black males (+25 percent). Blacks experience higher age-adjusted incidence for cancer of the cervix, esophagus, larynx, pancreas, and stomach. Of the 25 types of cancer for which SEER data are collected, blacks have poorer case fatality rates for 22. Five-year relative survival for all cancer types is 38 percent for blacks and 50 percent for whites. Cancer case fatality rates are particularly high in blacks for cancer of the bladder, breast, corpus uteri, prostate, and stomach.

Several reasons are cited for the greater incidence and poorer case fatality rates of blacks with cancer. Risk factors can be identified for increased incidence of certain cancer types. For example, the threefold mortality excess of esophageal cancer for blacks (3.5 times the incidence) may be due to higher alcohol consumption among blacks (consistent with their higher cirrhosis rates). The 45 percent higher lung cancer mortality rates for black males may be due to a combination of higher levels of smoking and higher rates of exposure in environmentally hazardous occupations. Food quality, use of smokeless tobacco, and alcohol consumption are possibly responsible for stomach cancer mortality rates being 1.5 times higher and incidence being twice as high among blacks. Black males have a 60 percent higher incidence and 100 percent higher mortality rates from prostate cancer than white males. Black females have cervical mortality and incidence 2.5 times higher

than the general population. The elevation of both prostatic cancer and cervical cancer may be linked to higher rates of early sexual activity and the possibility of early infection with viral and other venereal diseases (Fraumeni 1975). Higher breast cancer mortality rates, in contrast, have been linked with later age at onset of sexual activity, lower fertility, and later age at first pregnancy (MacMahon et al. 1970).

Differences in case fatality rates (i.e., deaths as a proportion of those with the disease) can be traced to several factors. Knowledge, attitudes, and practices relating to cancer care-seeking behavior (cancer screening, detection, treatment, and rehabilitation) differ by race. National surveys suggest that blacks overestimate the fatality of cancer and underestimate its prevalence (U.S. Department of Health and Human Services 1985). Blacks are less educated about cancer signs and more pessimistic about treatment. Combining these results with possible economic constraints, blacks may delay seeking diagnosis and treatment because of their conception of cancer as a terminal disease process, thus leading to a higher relative prevalence of cancer diagnoses at advanced stages (National Cancer Institute 1984).

Other factors beyond behavior (which we presume to be partly based on knowledge and attitudes toward cancer) contribute to poorer black survival. Studies indicate that survival differences remain between blacks and whites *even* at the same stage of disease (Wilkinson et al. 1979). Socioeconomic factors explain part of the differential (Berg 1977), but even after socioeconomic factors and disease characteristics are controlled, deficits for blacks remain due to differences in treatment (Page and Kuntz 1980), immune competency, histologic type, general health status, nutrition, and other factors (Savage et al. 1981). Thus, blacks have higher cancer rates than whites because of (1) higher risk-factor exposures for many (but not all) cancer types, (2) their image of cancer as a lethal disease, which causes them not to be diagnosed early and to less aggressively pursue treatment, (3) economic and medical access variables that cause them to be less effectively treated, (4) differences in disease type, with many of the histological types affecting blacks being more aggressive, and (5) general differences in health status resulting in lower host resistance to the tumor.

The cancer life table statistics may be compared with the other diseases in table 7. There is an increase in the age at death from heart disease for both blacks and whites. The mean age at death in 1980

varies over 12 years across the four race/sex groups, indicating greater race and sex variation in heart disease risks than in cancer risks. Despite significant declines in the mortality *rates* for heart disease (e.g., 24 percent between 1968 and 1978), the life table proportion of all deaths expected from heart disease declined only slightly for white males, increased slightly at early ages for black males and females, and is nearly constant for white females. Heart disease is a less prevalent cause of death for both black males and females than for whites (due to their higher risk of death at earlier ages from other causes), though heart disease has increased for black males at younger ages.

Homicide, in absolute terms, is a frequent cause of death among black males, being nearly as likely to cause death as lung cancer. Furthermore, it is a cause of death affecting younger persons, with the mean age at death for homicide victims ranging from 37.9 years for black males to 42.0 years for white females in 1980. Between 1970 and 1980, the mean age at death from homicide increased for black males and for females of both races. In contrast, the mean age at death from homicide for white males decreased between 1970 and 1980 (from 40.5 to 38.7 years), suggesting an increase in homicide risks at early ages for white males. The proportion of all deaths due to homicide is much higher for blacks than whites, being over six times higher for black males in 1980 and nearly five times higher for black females.

The proportion of deaths due to diabetes shows a small drop for white males and no change for black males. Declines are also registered for females, although the proportion of black females affected is greater than that of white females. There has been a large increase in the mean age at death for blacks (i.e., 3.0 and 4.5 years), suggesting better control at earlier ages delaying death for blacks.

Cirrhosis is a greater mortality risk for blacks than whites starting at younger ages. For black males, excessive alcohol consumption begins early but occurs primarily after age 30, whereas for white males the prevalence is higher from ages 18 to 25 and then declines (U.S. Department of Health and Human Services 1985). Thus, it appears to be continued high alcohol consumption in adulthood that contributes most to cirrhosis in blacks. There are also both more alcohol abstainers and heavy consumers among blacks (U.S. Department of Health and Human Services 1985). Thus, exposure seems to be concentrated in certain black subpopulations.

Patterns of risk-factor exposures may be related to both geographic and sociocultural differences. For example, exposure to certain types of risks (e.g., homicide, cirrhosis, lung cancer) are probably higher in metropolitan areas than rural areas and in certain regions (e.g., Northeast and Far West) than in other regions (e.g., Southeast), although for lung cancer we see that southeastern regions are converging in risk to that in the Northeast (Manton et al. 1985). An important protective factor in rural areas and the Southeast may be sociocultural and religious norms against alcohol and drug abuse. More detailed study of such geographic differences in cause-specific mortality and risk-factor exposures could potentially produce much useful information on sociocultural and economic differences in health risks between blacks and whites.

The change in the life-table mean age at death for a disease and the proportion of deaths it causes determine the change in life expectancy anticipated if a given condition were eliminated and persons were only exposed to the risks of the remaining conditions. This statistic is important in determining the potential benefit of controlling or eliminating a condition. Two types of such "cause elimination" life expectancy gains are presented in table 8. The first, labelled "Total Population," is the usual statistic presented in the demographic literature that shows the impact (in years) of eliminating the condition on the life expectancy of the population (e.g., Chiang 1968; Keyfitz 1977). The second, labelled "Saved Population," is the effect on the life expectancy of persons who died of the disease—an alternate form of competing risk adjustment. The latter statistic allows comparison of the survival effect of common and rare causes of death and better measures the impact of the disease on the individual (Manton, Patrick, and Stallard 1980). It indicates the years of life lost by a typical decedent from the specific cause of death and is calculated by adjusting the usual-cause-elimination life-expectancy-gain figure (i.e., the "Total Population" figure) for differences in the proportion of deaths expected from the condition as reported in the bottom panel of table 7.

The cause elimination effects of the six conditions for the total population reflect the proportion of deaths caused by the disease. Hence, gain in life expectancy due to the elimination of heart disease is the largest. In contrast, the "saved population" measure describes the effect of premature mortality from a condition on the life span of the individual. Therefore, it is greatest for deaths that occur at early ages—e.g., accidental deaths and homicides.

TABLE 8
Life-Expectancy Increases (in years) Due to Elimination of Selected Causes of Death by Race and Sex, 1969–1971 and 1979–1981

	White males		Black males		White females		Black females	
Cause of death	1969–1971	1979–1981	1969–1971	1979–1981	1969–1971	1979–1981	1969–1971	1979–1981
CANCER								
Total population	2.39	2.97	2.46	3.37	2.68	3.18	2.61	3.21
Saved population	14.12	14.12	16.08	15.70	16.79	17.13	19.26	18.55
HEART DISEASE								
Total population	6.97	6.56	6.16	5.75	6.69	7.19	8.23	7.92
Saved population	16.52	15.97	19.71	17.71	15.89	16.93	22.24	20.41
DIABETES MELLITUS								
Total population	0.18	0.16	0.24	0.23	0.30	0.25	0.61	0.51
Saved population	11.87	11.74	15.02	13.81	12.17	12.43	16.42	14.90
STROKE								
Total population	0.96	0.69	1.51	1.04	1.64	1.33	2.62	1.79
Saved population	10.07	9.58	14.35	12.78	10.82	10.82	16.41	14.23
ACCIDENT								
Total population	1.74	1.60	2.20	1.59	0.80	0.70	0.94	0.69
Saved population	30.22	32.14	30.03	28.12	24.08	26.57	29.96	27.05
HOMICIDE								
Total population	0.16	0.26	1.54	1.43	0.06	0.10	0.38	0.37
Saved population	32.76	36.13	31.98	32.88	38.02	40.05	37.50	39.47

Large increases in the impact of cancer on total-population life expectancy occurred between 1969–1971 and 1979–1981, with black males having the largest increase (0.91 years). White males had the smallest saved-population gain because they had the highest mean age at death from cancer (relative to their total life expectancy). Blacks of both sexes experienced decreases, suggesting that cancer deaths occurred at older ages in 1979–1981 than in 1969–1971. White females had an increase, suggesting that cancer deaths occurred at younger ages in 1979–1981.

Heart disease shows declines in impact in both the saved and total population measures for three of the four groups, suggesting decreased population and individual impact between 1969–1971 and 1979–1981. Only white females show an increased impact over this period.

Diabetes mellitus and stroke show declines in both measures for all four groups consistent with the linkage of the two conditions. Accidental deaths show declining population impact, but increasing individual (i.e., "saved" population) impact. The impact of homicide increased on both the population and individual level for whites, but declined for blacks.

Excess Deaths

The preceding analyses showed black-white differentials in life expectancy, the underlying and multiple-cause mortality factors that produced those differentials, and recent changes in those differentials. To summarize differentials in mortality risks and the mean age at death in terms of the current impact on health, an index of "excess deaths" was calculated. Excess deaths in the black population are the number of deaths due to a particular condition above the level that would have occurred if blacks had experienced the same age-, sex-, and cause-specific mortality rates as whites. An age cutoff of 70 years was used in aggregating excess deaths. Since the maximum life span is probably not much different for blacks and whites, high early mortality among blacks must produce higher white mortality rates at later ages and negative "excesses" at advanced ages. This phenomenon would have lessened the value of the index as an indicator of disparities between blacks and whites that might be amenable to interventions at earlier ages (i.e., the age ranges where deaths are clearly premature).

TABLE 9
Excess Deaths[a] in the Black Population by Sex and Cause from Birth to Age 69, 1969–1971 and 1979 –1981

	Males		Females	
Disease	1969–1971	1979–1981	1969–1971	1979–1981
All causes	39,925	35,321	32,895	23,621
Cancer	3,674	5,782	2,175	2,268
Diabetes	696	646	1,693	1,204
Heart disease	4,258	5,633	8,898	7,235
Stroke	4,202	2,837	4,566	2,477
Accidents	3,822	2,261	1,159	559
Homicide	6,496	6,708	1,347	1,380
Cirrhosis	1,205	1,373	863	783
Infant mortality & congenital anomalies	5,145	2,155	4,260	1,847

[a] Actual deaths − expected deaths, where expected deaths = white death rates × black population (up to age 70).

The changes in the number of excess deaths between 1969–1971 and 1979–1981 are presented in table 9.

Rates of deaths changed markedly for both races during this period. As a result, the number of excess deaths drops from 73,000 to 59,000; over 65 percent of the drop occurred in black females. Large decreases are noted for stroke, accidents, and infant mortality, but large increases in excess deaths occurred for cancer (especially in males).

Observed, expected, and excess deaths by age for black males and females are presented in table 10 for the 1979–1981 period.

In table 10 the annual number of deaths expected in 1979–1981 if blacks were subjected to white mortality rates, the actual number of deaths, and the annual difference or excess are shown for six causes. For example, black males under the age of 70 would have 49,378 deaths if they had the same mortality rates as white males—84,699 black male deaths were observed. Consequently, 35,321 *excess* black male deaths occurred annually. Thus, 42 percent of black deaths in the 1979–1981 period were excess and potentially avoidable. Of the 35,321 excess deaths in males, 24 percent (8,470) were due to cardiovascular disease, 16 percent were due to cancer, and 19 percent to homicide.

The contribution of each cause to excess mortality is quite different for those aged under 45 than for persons aged 45 to 69. Under the age of 45, 15 percent of the excess male mortality is due to infant mortality (deaths occurring under one year of age). This drops to 6 percent for all excess deaths under the age of 70. Likewise, the importance of homicide drops from 37 to 19 percent. From the age of 45 to 69 the male excess due to homicide is only 6 percent. On the other hand, 58 percent of excess male mortality from the age of 45 to 69 is due to cardiovascular disease or cancer.

The excess death measure has the disadvantage that it is based upon the current level of white mortality as the "norm" or "standard." This raises the question of what our targets or goals should be when contemplating corrective actions. For example, there is excess mortality among whites for certain diseases (e.g., suicide for white males and breast cancer for white females). If there is no black excess for a cause, should we conclude that the current mortality level among blacks is acceptable? Furthermore, we know that white infant mortality is much higher in the United States than in many European countries and Japan. This means that the black infant mortality excess is less than that if compared to the lowest observed levels. Even the Japanese and European mortality levels may not represent the lowest achievable level given the current state of medical science. This suggests that we should posit an absolute health standard against which we could evaluate the health of both blacks and whites. We, therefore, calculated a second measure, the SHE index, which represents deaths due to medical conditions that were judged on the basis of known risk factors or the current level of treatment efficacy to be either preventable or curable (Rutstein et al. 1976). These measures are presented for several major age categories for the four race/sex groups in table 11.

The table confirms that there are large numbers of deaths for whites (17.6 percent for males and 12.2 percent for females) that could be prevented. Against absolute standards American whites are doing nearly as poorly as blacks in terms of the proportions of total mortality resulting from preventable or curable diseases. The age-adjusted mortality rate for the SHEs is 185.3 for black males and 177.5 for white males with 17.6 percent and 18.2 percent of all white and black male deaths being preventable.

For women the SHE mortality rate is identical (100.4). However, because a greater proportion of the black female population is at

TABLE 10
Three Measures of the Average Annual Number of Deaths by Disease Category and Sex, for U.S. Blacks, 1979–1981

	CVD[a]	Cancer	Cirrhosis	Diabetes	Homicide	Infant mortality & congenital anomalies	All others	Total
				BIRTH TO AGE 44				
Males: Observed	3,236	1,587	962	202	6,486	4,832	13,789	31,094
Expected[b]	1,339	1,204	259	86	1,017	2,685	9,777	16,367
Excess	1,897	383	703	116	5,469	2,147	4,012	14,727
Percentage of total excess[c]	13%	3%	5%	1%	37%	15%	27%	100%
Observed	2,093	1,789	551	184	1,488	3,996	7,130	17,231
Expected[b]	676	1,366	131	77	344	2,150	4,278	9,022
Excess	1,417	423	420	107	1,144	1,846	2,852	8,209
Percentage of total excess	17%	5%	5%	1%	14%	22%	35%	100%
				AGE 45 TO 69				
Males: Observed	21,677	14,530	1,745	989	1,448	55	13,161	53,605
Expected[b]	15,104	9,131	1,075	459	209	47	6,986	33,011
Excess	6,573	5,399	670	530	1,239	8	6,175	20,594
Percentage of total excess[c]	32%	26%	3%	3%	6%	—	30%	100%
Females: Observed	15,698	10,156	976	1,602	308	47	7,918	36,705
Expected[b]	7,403	8,311	613	505	72	46	4,343	21,293
Excess	8,295	1,845	363	1,097	236	1	3,575	15,412
Percentage of total excess[c]	54%	12%	2%	7%	2%	—	28%	100%

	BIRTH TO AGE 69							
Males: Observed	24,913	16,117	2,707	1,191	7,934	4,887	26,950	84,699
Expected[b]	16,443	10,335	1,334	545	1,226	2,732	16,763	49,378
Excess	8,470	5,782	1,373	646	6,708	2,155	10,187	35,321
Percentage of total excess[c]	24%	16%	4%	2%	19%	6%	29%	100%
Females: Observed	17,791	11,945	1,527	1,786	1,796	4,043	15,048	53,936
Expected[b]	8,079	9,677	744	582	416	2,196	8,621	30,315
Excess	9,712	2,268	783	1,204	1,380	1,847	6,427	23,621
Percentage of total excess[c]	41%	10%	3%	5%	6%	8%	27%	100%

[a] CVD = cardiovascular disease and combined heart disease and stroke
[b] Calculated from the rate observed in the white population
[c] May not add to 100 due to rounding

TABLE 11
Annual Number of Deaths from Sentinel Health Events (SHE), Proportion That SHE Are of All Deaths and Rate for SHE, 1979–1981

	Males			Females		
	Whites	Blacks	Ratio of rates of SHE	Whites	Blacks	Ratio of rates of SHE
Age 0–24	11,261	4,672		8,293	3,824	
Percentage	20.0%	31.9%		28.8%	41.6%	
Rate	29.7	70.6	2.38	22.7	57.1	2.52
Age 25–44	4.541	1,511		3,566	1,088	
Percentage	8.2%	9.2%		13.7%	13.6%	
Rate	17.5	47.4	2.71	13.6	29.1	2.14
Age 45–64	42,669	7,543		22,055	3,633	
Percentage	18.9%	19.3%		17.2%	14.1%	
Rate	229.6	403.1	1.76	108.9	157.4	1.45
Age 65–69	23,992	2,874		10,706	1,216	
Percentage	20.8%	19.9%		15.1%	11.1%	
Rate	689.1	866.4	1.26	247.2	273.0	1.10
Age 70+	80,268	6,595		52,448	4,269	
Percentage	17.0%	15.5%		9.7%	9.2%	
Rate	1398.3	1280.6	0.92	558.2	537.0	0.96
All ages	162,732	23,194		97,068	14,030	
Percentage	17.6%	18.2%		12.2%	14.0%	
Rate	177.5	185.3		100.4	100.4	

younger ages, the proportion of deaths from SHEs is higher for blacks (14.0 percent) than for whites (12.2 percent). For example, the mortality rate from SHEs is 57.1 at ages 0 to 24 for black females and only 22.7 for white females—a ratio of 2.52 to 1.0. This ratio declines with age until above age 70 where the risk from SHEs is higher for white females (558.2) than for black females (537.0). This suggests that even though white females have a higher overall life expectancy, proportionately more preventable deaths at advanced ages occur for white females—an observation based upon an epidemiological and clinical evaluation of the cause of death mix at different ages that is consistent with the crossover in the total mortality rates observed earlier.

Similar age patterns are noted for males except that the peak age range for excess black risk is age 25 to 44 (i.e., 2.71) reflecting the effect of homicide and other external causes of death. It is also interesting to compare the mortality rates for SHEs across sex within race. For example, we see that up to age 44, white females have greater proportions of deaths due to preventable causes than white males—a pattern also found for black males and females. Past the age of 45 males of both races have considerably greater proportions of deaths from preventable causes.

Morbidity and Disability

In the preceding section we discussed mortality differences between blacks and whites. Though mortality is an excellent indicator of serious health problems, many nonlethal conditions generate considerable disability in the population. Such conditions are described in the National Health Interview Survey. Owing to methodological changes in this survey over time, it is difficult to determine how the prevalence of various conditions has changed. However, the data on disease prevalence at a given time probably accurately describe black-white differentials in the health burden of different conditions. Thus, we present the ratio of age-, sex-, and race-specific prevalence rates or relative risks for selected conditions (hypertension, circulatory disease, diabetes, arthritis, and mental and nervous disorders) in table 12.

Hypertension is a risk factor for many other conditions such as stroke, heart disease, and renal failure. Overall, blacks are two to four times as likely to report hypertension as whites. The relative risks are highest at early ages (i.e., before 45) with systematic declines in the relative risks for both sexes at later ages. The higher risk of hypertension for blacks is consistent with the greater risk of stroke mortality documented in earlier tables.

Both black males and females have a greater probability of circulatory disease than whites. The relative risk is higher for black females and is more elevated for both sexes below the age of 45 (consistent with the pattern for hypertension). Analyses of only ischemic heart disease, in contrast, showed a considerable white male excess.

Diabetes is a morbid condition which, like hypertension, is a risk

TABLE 12
Relative Risks Based on White Morbidity Rates for Blacks for Five Conditions, 1979–1981

	Hypertension		Diseases of circulatory system		Diabetes		Arthritis		Nervous & mental disorders	
Age	Males	Females	Males	Females	Males	Females	Males	Females	Males	Females
1–14	4.03	3.73	1.60	2.33	0.22	1.15	0.78	0.36	0.65	1.05
15–24	3.37	3.76	1.88	2.31	0.39	0.68	0.73	0.36	1.39	1.19
25–44	2.73	4.54	1.86	2.91	0.23	2.16	0.65	1.16	1.63	1.41
45–64	2.67	3.20	1.41	2.31	2.43	2.82	1.71	1.64	1.69	1.32
65–69	2.53	2.31	1.40	1.68	2.30	2.87	1.57	1.40	1.19	0.91
70+	2.39	2.22	1.33	1.62	1.85	2.22	1.71	1.54	1.19	1.10

Source: Tabulation of the National Health Interview Survey.

factor for many circulatory diseases. Blacks had over a twofold excess risk of diabetes past the age of 45 (table 12). Much of this risk is associated with the onset of adult diabetes related to obesity. The relative risks are higher for females and decline for both sexes at advanced ages. The decline in the relative risk may be a result of a more rapid mortality selection of black diabetics at earlier ages relative to whites. The greater prevalence of diabetes among blacks and the excess of female diabetes over the male rates within race are consistent with the previously reported mortality excesses.

A condition that causes considerable disability, especially at advanced ages, is arthritis. Past the age of 45, there is over a 50 percent greater risk of arthritis for blacks. Below the age of 25 (45 for males) arthritis is less often reported for blacks. For both races, the prevalence of arthritis is much greater for females. Arthritis has been found to be one of the most prevalent causes of activity limitation in the 1982 National Long-term Care Survey.

Mental and nervous disorders are also chronic nonlethal conditions seriously affecting quality of life—especially in middle age. Blacks report a higher prevalence of such problems between the ages of 24 to 64. At advanced ages the racial differential declines markedly.

The World Health Organization (1980) classification of impairments, disability, and handicaps describes the consequences of disease. Such consequences are particularly important for the elderly population inasmuch as the demand for long-term care services is driven by disability related to specific chronic conditions. Thus, a second important aspect of the comparison of black and white health differentials is the comparison of these disease consequences—i.e., the disability and functional impairment they produce.

Disability is often measured by the number of activities of daily living (ADL) or instrumental activities of daily living (IADL) that the person is chronically limited in performing. ADLs are assumed to be ordered according to their acquisition by a child during socio-biological development (Katz and Akpom 1976). To generate a global measure of disability we summed the number of ADLs for which a person has impairments. The sex- and age-specific disability rates are represented in table 13.

The table records that community-dwelling elderly blacks at almost all ages, disability levels, and for both sexes report higher disability rates than whites. In general, the relative risks of disability for blacks

TABLE 13

Number and Percentage of Disabled Population in 1982 at Three Age Groups and Four Disability Levels, U.S. Blacks and Whites*

Disability level	Age 65 to 74			Age 75 to 84			Age 85 and older		
	White	Black	Ratio	White	Black	Ratio	White	Black	Ratio
				MALES					
IADL only	239	44	1.88	167	26	1.66	53	8	1.76
	(4.1)	(7.7)		(6.4)	(10.6)		(8.1)	(14.3)	
1–2 ADL	200	28	1.40	172	16	0.99	82	13	1.84
	(3.5)	(4.9)		(6.6)	(6.5)		(12.6)	(23.2)	
3–4 ADL	90	18	2.00	62	6	1.00	44	4	1.07
	(1.6)	(3.2)		(2.4)	(2.4)		(6.7)	(7.1)	
5–6 ADL	132	27	2.04	112	11	1.04	41	5	1.42
	(2.3)	(4.7)		(4.3)	(4.5)		(6.3)	(8.9)	
Total disabled	661	117	1.81	513	59	1.22	220	30	1.59
	(11.4)	(20.6)		(19.8)	(24.1)		(33.7)	(53.6)	
Total population in each age group (in thousands)	5,773	569		2,590	245		652	56	

				FEMALES					
IADL only	346	64	1.71	330	48	1.62	122	11	1.23
	(4.7)	(8.0)		(7.4)	(12.0)		(7.7)	(9.5)	
1–2 ADL	353	55	1.44	408	51	1.38	216	19	1.20
	(4.8)	(6.9)		(9.2)	(12.7)		(13.6)	(16.4)	
3–4 ADL	135	29	2.02	172	19	1.21	90	12	1.27
	(1.8)	(3.6)		(3.9)	(4.7)		(5.7)	(7.2)	
5–6 ADL	136	26	1.81	171	26	1.71	126	23	2.31
	(1.8)	(3.3)		(3.8)	(6.5)		(7.9)	(18.3)	
Total disabled	970	174	1.66	1,081	144	1.48	554	65	1.60
	(13.1)	(21.8)		(24.3)	(35.9)		(35.0)	(56.0)	
Total population in each age group (in thousands)	7,400	797		4,448	401		1,585	116	

* Figures in parentheses are percentages

Source: Population counts are 1982 figures from U.S. Bureau of the Census, 1984, 38 (table 5). Disabled population counts are from tabulations of the National Long-term Care Survey.

seem to be higher at ages 65 to 74 than at later ages, although the ratios are higher above the age of 85—i.e., above age 85 over half of community-dwelling blacks have some disability in contrast to about a third of whites.

An important factor in studying the health status of the elderly is the interaction of morbidity, disability, and low income. We examined this factor using data from the Georgia Adult Health Services program where we had information on the health and functional status of 583 persons over the age of 65. This population represents a group of low-income elderly persons on Medicaid long-term care benefits who still resided in the community. Thus, it gives us a much more detailed look at the health and functional status of elderly blacks and whites in a high-intensity and long-term service use category. We analyzed these with a procedure called grade of membership (GOM) analysis (Woodbury and Manton 1982) that could identify subgroups among the population using a wide range of disease and functional status measures. The population could be described by five basic health and functional status profiles. These are described in table 14.

The first group or profile has chronic obstructive lung disease and digestive problems, few chronic conditions, and is relatively free of functional dependence. This group is predominantly white, relatively young, and more likely to be male than the overall study population.

The second group has diabetes and heart problems, requires few services, but tends to have four ADL limitations. This group is relatively more black, heavily female, and older than the study population in general.

The third group has circulatory and urinary tract problems and the highest frequency of dementia and psychological problems. It also has by far the largest number of behavioral problems and the greatest range of therapeutic services used. It is also disproportionately nonwhite and has a high level of ADL impairment.

The fourth group has the highest prevalence of cancer, stroke, neurological disorders, and hip fracture. It has by far the greatest number of medical treatments and is the most bedfast. It is highly functionally impaired. It is *not* distinguished by race, though it is a heavily male group and relatively young.

The final group has sensory problems and arthritis, though few impairments, and requires few services. It is female and though by far the oldest group is somewhat more likely to be black. Given its

TABLE 14
Five Health and Functional Status Profiles Determined by the Grade of Membership Technique in the Georgia Adult Health Services Program

	Frequency	1	2	3	4	5
VARIABLES USED TO DEFINE GROUPS						
I. DIAGNOSES						
1. Cancer	5.15	12.28	0.0	0.0	20.04	0.0
2. Diabetes	19.90	0.0	67.05	0.0	0.0	0.0
3. Anemia	4.97	0.0	0.0	3.82	5.02	15.49
4. Dementia	4.63	0.0	0.0	30.54	0.0	0.0
5. Psychosis, Neurosis	6.00	2.80	0.0	25.72	2.81	6.72
6. Neurological disorders	5.66	0.0	0.0	0.0	38.79	0.0
7. Eye disorders	5.15	0.0	0.0	0.0	0.0	21.34
8. Hypertension	38.77	0.0	88.12	41.90	0.0	0.0
9. Heart disease	43.91	31.72	100.00	78.06	0.0	0.0
10. Cerebrovascular disease	16.64	0.0	0.0	0.0	88.92	0.0
11. Arteriosclerosis	3.60	0.0	0.0	0.0	0.0	16.06
12. Chronic obstructive lung disease	8.58	45.64	0.0	4.08	0.0	0.0
13. Ulcers	2.74	15.71	0.0	0.0	0.0	0.0
14. Digestive disorders	3.43	18.64	0.0	1.65	0.0	0.0
15. Nephritis, Nephrosis	3.60	0.0	3.77	5.00	11.14	0.0
16. Urinary tract disorders	5.32	0.0	0.0	21.31	7.17	5.57
17. Joint problem	30.19	0.0	0.0	0.0	0.0	100.00
18. Hip fracture	2.74	5.61	0.0	0.0	11.27	0.0
19. Residual	19.90	25.87	0.0	23.62	40.62	23.04

TABLE 14—(*Continued*)
Five Health and Functional Status Profiles Determined by the Grade of Membership Technique in the Georgia Adult Health Services Program

	Frequency	1	2	3	4	5
II. CONDITIONS						
1. Decubiti	2.78	0.0	0.0	0.0	17.54	0.0
2. Bowel incontinence	22.74	0.0	0.0	76.75	100.00	0.0
3. Bladder incontinence	43.83	0.0	28.02	100.00	100.00	25.91
4. Agitation	5.20	0.0	0.0	36.34	0.0	0.0
5. Confusion	26.69	0.0	0.0	100.00	0.0	0.0
6. Cooperative	71.58	72.46	100.00	10.43	52.54	100.00
7. Depression	12.31	22.37	0.0	59.27	0.0	0.0
8. Forgetfulness	43.50	20.65	38.74	100.00	0.0	52.63
9. Alertness	46.97	72.43	69.97	0.0	31.65	48.04
10. Noisiness	1.73	1.38	0.0	10.68	0.0	0.0
11. Nonresponsiveness	0.35	0.0	0.0	0.0	2.21	0.0
12. Vacillating behavior	3.29	8.61	0.0	11.05	0.0	1.27
13. Violent behavior	0.17	0.97	0.15	0.0	0.0	0.0
14. Wandering	4.51	0.0	0.0	34.14	0.0	0.0
15. Withdrawal	2.77	0.0	0.0	18.89	0.0	0.0
16. Dependence	42.46	0.0	23.09	100.00	82.95	19.84
17. Independence	8.67	48.59	0.0	0.0	0.0	0.0
18. Anxiety	11.61	44.71	0.0	24.40	0.0	0.0
19. Disorientation	6.59	0.0	0.0	41.26	6.71	0.0
20. Inappropriateness	1.21	0.0	0.0	6.65	0.0	1.31

21. Sight impairment						
Severe	17.37	0.0	0.0	0.0	0.0	58.43
Moderate	49.65	0.0	69.14	100.00	41.32	41.57
Mild	25.44	76.78	25.89	0.0	41.24	0.0
None	7.54	23.22	4.97	0.0	17.44	0.0
22. Hearing						
Severe	8.58	0.0	0.0	6.44	0.0	32.80
Moderate	33.45	12.66	28.28	42.26	30.29	49.74
Mild	32.57	37.11	43.67	51.30	28.91	0.0
None	25.39	50.24	28.05	0.0	40.80	17.47
23. Speech						
Severe	3.68	0.0	0.0	0.0	24.16	0.0
Moderate	9.46	0.0	0.0	0.0	62.12	0.0
Mild	13.13	0.0	0.0	73.83	13.72	0.0
None	73.73	100.00	100.00	26.17	0.0	100.00
24. Limited Mobility						
Severe	20.45	0.0	0.0	0.0	100.00	0.0
Moderate	59.62	0.0	100.00	68.70	0.0	100.00
Mild	14.69	66.63	0.0	31.30	0.0	0.0
None	5.24	33.37	0.0	0.0	0.0	0.0
25. Paralysis						
Severe	3.33	0.0	0.0	0.0	26.77	0.0
Moderate	7.53	0.0	0.0	0.0	60.58	0.0
Mild	4.90	0.0	0.45	19.24	12.65	0.0
None	84.24	100.00	99.55	80.76	0.0	100.00
III. SERVICES						
1. Fluid intake	5.03	0.0	0.0	0.04	30.68	0.0
2. Fluid output	5.03	0.0	0.0	0.06	30.66	0.0

TABLE 14—(*Continued*)
Five Health and Functional Status Profiles Determined by the Grade of Membership Technique in the Georgia Adult Health Services Program

	Frequency	1	2	3	4	5
3. I.V.	0.52	0.0	0.0	0.0	3.29	0.0
4. Suction	0.52	0.0	0.0	0.0	3.32	0.0
5. Catheter	2.95	0.0	0.0	0.0	19.92	0.0
6. Sterile dressing	1.56	7.73	0.0	1.86	0.0	0.0
7. Colostomy	0.87	0.0	0.0	0.0	5.44	0.0
8. Bedfast	6.24	0.0	0.0	0.0	44.59	0.0
9. Physical therapy	19.19	0.0	0.0	100.00	31.04	0.0
10. Occupational therapy	4.93	0.0	0.0	36.77	0.0	0.0
11. Remotive therapy	5.63	0.0	0.0	45.21	0.0	0.0
12. Reality therapy	9.68	0.0	0.0	89.75	0.0	0.0
13. Speech therapy	0.88	0.0	0.0	6.37	0.0	0.0
14. Bowel & bladder training	1.76	0.0	0.0	9.68	2.98	0.0
15. Activity program	12.85	0.0	0.0	100.00	0.0	0.0
IV. FUNCTIONAL LIMITATIONS						
1. Eating	33.22	0.0	0.0	100.00	100.00	0.0
2. Confined to wheelchair	28.10	0.0	0.0	0.0	100.00	0.0
3. Transference	74.96	0.0	100.00	100.00	100.00	100.00
4. Bathing	94.08	62.87	100.00	100.00	100.00	100.00
5. Ambulatory limitations	84.67	32.95	100.00	100.00	67.62	100.00
6. Dressing	74.91	0.0	100.00	100.00	100.00	100.00

EXTERNAL VARIABLES						
V. SOCIODEMOGRAPHIC						
1. Race						
White	48.61	97.65	29.42	39.84	49.50	41.90
Nonwhite	51.39	2.35	70.58	60.16	50.50	58.10
2. Age						
65–69	15.44	41.87	14.28	0.0	29.28	0.0
70–74	16.12	49.87	14.13	2.77	23.42	0.0
75–79	18.01	0.0	29.05	17.37	2.07	26.89
80–84	24.36	8.26	23.94	39.54	29.47	23.15
85–89	15.44	0.0	18.59	31.44	0.0	22.20
90+	10.63	0.0	0.0	8.89	15.76	27.76
3. Sex						
Male	22.20	50.49	0.0	21.83	61.52	2.02
Female	77.80	49.52	100.00	78.17	38.48	97.98
4. Nursing Home Admission	10.12	19.31	3.78	11.15	13.93	8.25

extreme age but few explicit acute medical problems and no dementia (most of its difficulties appear related to musculoskeletal conditions), it is representative of relatively intact extreme elderly survivors.

In the first group of "young" elderly persons with few functional impairments and few chronic conditions, we found a disproportionate probability of being white. Groups two and three with chronic conditions and significant frailty tend to be more heavily black while the fourth group, associated with serious acute medical problems that are medically intensive, is not distinguished by race. The fifth group of extreme elderly with only moderate levels of dependency and few acute medical problems is disproportionately black. The pattern of association of race, functional limitation, and medical problems suggests that the distribution of serious acute medical problems (i.e., group 4) at advanced ages is not strongly differentiated by race, while the distribution of functional impairment over age is. This suggests that social and economic factors may be more important for low-income blacks using community-based Medicaid long-term care services than for whites. Whites may tend to use nursing homes more as a source of care for the extreme elderly, frail person than blacks who apparently are retained in the community, possibly in an extended family milieu.

Risk Factors

Up to this point black and white differentials have been described in terms of manifest morbid changes. An examination of differentials in the distribution of physical risk factors and health behavior will help to explain the pattern of health problems currently observed and suggest the patterns of health differentials that can be expected in the future. The risk factors discussed are (1) smoking, (2) obesity, (3) blood pressure, (4) serum cholesterol, (5) birth weight, and (6) health habits.

Smoking

Cigarette consumption is correlated with the risk of many different chronic conditions—e.g., lung cancer, chronic obstructive lung disease, and heart disease. Table 15 contains the percentages of blacks and whites who are current smokers for the period of 1965 to 1983.

The decline in the proportion of those who smoke is rapid for both black and white males; although black males are still more likely to be smokers, the differential has decreased. The declines in the proportion of the population who smoke are less for females, and the current differential between black and white females (2.7 percent) is less than for males (7.9 percent). For black females overall, there has been relatively little change since 1965, although there are interesting age patterns that reflect cohort-specific smoking habits by sex. For females over the age of 65, there has actually been an increase in the proportion of smokers—reduction in smoking for females has lagged 20 years behind that of males (Harris 1983). With the lengthy latency of many smoking-associated diseases, increases in the risks of those diseases can be expected for a number of years to come for females of both races.

Obesity

Being overweight is a risk factor for circulatory disease, both directly by increasing circulatory load, and indirectly by increasing the likelihood of hypertension and diabetes. Figure 4 records the percentage of those obese by age, sex, and race.

The most striking feature of figure 4 is the greater prevalence of obesity among black females compared to white females. The rate of obesity for black females is twice that of white females above the age of 55. The high prevalence of obesity explains why black females have high mortality and morbidity from diabetes. Black males also have a higher prevalence of obesity than white males. In the group aged 55 to 64, black males have a 46 percent greater likelihood of obesity. Above the age of 65, black males have a 72 percent greater chance of being obese.

Hypertension

Elevated blood pressure is an important risk factor for stroke, renal failure, and heart disease. The race-specific trends for 1960 and 1980 in hypertension are shown in table 16.

The prevalence of hypertension has declined over the interval. Consistent declines from 1960 were noted for females, but not for males. For both white and black males the hypertensive proportion increased

TABLE 15

Percentage of Persons Currently Smoking Cigarettes, 20 Years of Age and Over, According to Sex, Race, and Age: United States, 1965, 1976, 1980, and 1983 (data based on household interviews of a sample of the civilian noninstitutionalized population)

	Males				Females			
	1965	1976	1980	1983	1965	1976	1980	1983
				WHITE				
Age 20 and over (age adjusted)	51.3	41.0	37.1	34.7	34.5	32.4	30.0	29.8
20–24	58.1	45.3	39.0	36.1	41.9	34.4	33.3	37.5
25–34	60.1	47.7	42.0	38.6	43.4	37.1	31.6	32.2
35–44	57.3	46.8	42.4	40.8	43.9	38.1	35.6	34.8
45–64	51.3	40.6	40.0	35.0	32.7	34.7	30.6	30.6
65 and older	27.7	22.8	16.6	20.6	9.8	13.2	17.4	13.2
				BLACK				
Age 20 and over (age adjusted)	59.6	50.1	44.9	42.6	32.7	34.7	30.6	32.5
20–24	67.4	52.8	45.5	41.0	44.2	34.9	32.3	37.0
25–34	68.4	59.4	52.0	39.9	47.8	42.5	34.2	38.0
35–44	67.3	58.8	44.2	45.5	42.8	41.3	36.5	32.7
45–64	57.9	49.7	48.8	44.8	25.7	38.1	34.3	36.3
65 and older	36.4	26.4	27.9	39.0	7.1	9.2	9.4*	13.1

* Relative standard error >30%.

Note: A current smoker is a person who has smoked at least 100 cigarettes and who now smokes; includes occasional smokers. 1982 values are final estimates based on data for the last 6 months of 1980.

Source: National Center for Health Statistics 1986c.

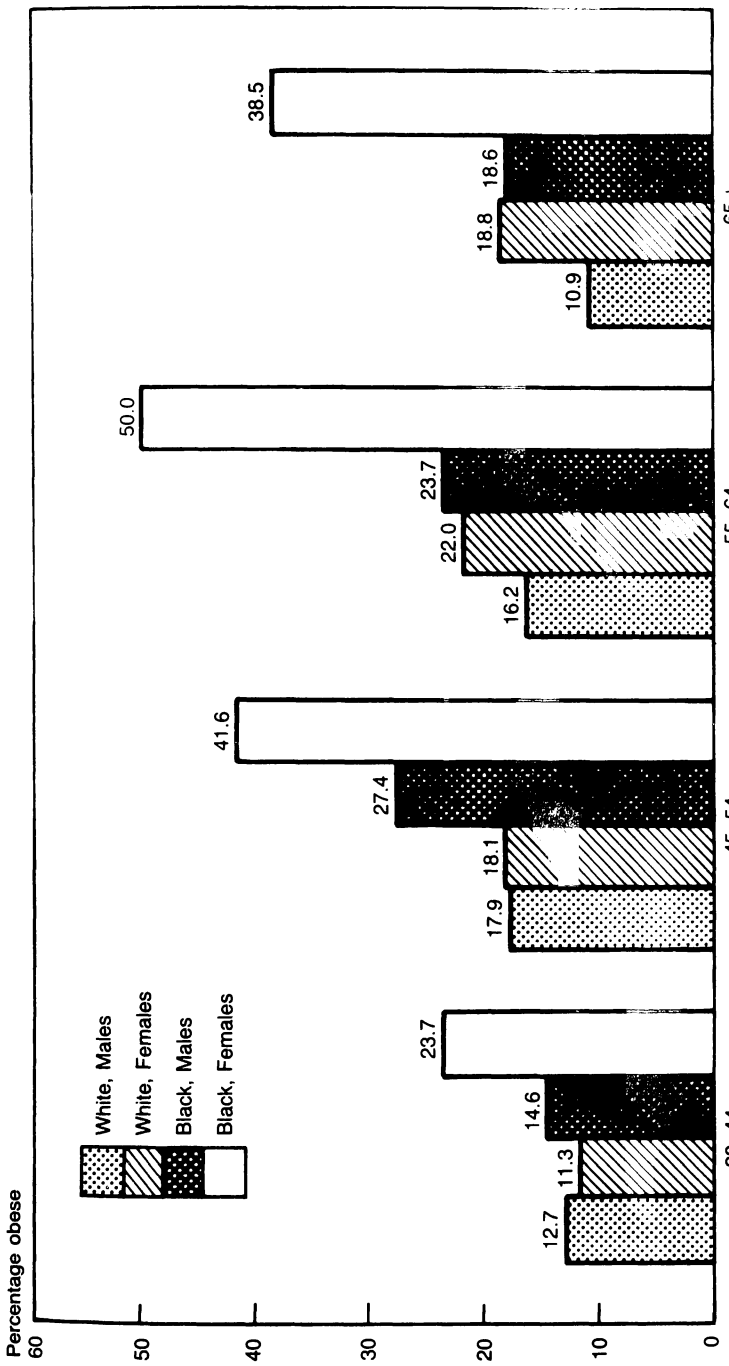

FIG. 4. Percentage of people in the most obese group, 1976.
Note: Percentage of people in the most obese group (Body Mass Index 28.49+ for males, 35.29+ for females): United States, 1976.
Sources: Bonham and Brock 1985; U.S. Department of Health and Human Services 1985, 151 (figure 11).

TABLE 16

Percentage of Persons Aged 25 to 74 with Elevated Blood Pressure, According to Race, Sex, and Age: United States, 1960–1962, 1971–1975, and 1976–1980 (data based on physical examinations of a sample of the civilian noninstitutionalized population)

	Race and period					
	White			Black		
Sex and age	1960–1962	1971–1975	1976–1980	1960–1962	1971–1975	1976–1980
MALE						
All ages, 25-74 years*	14.8	18.5	16.3	32.2	36.5	23.6
25–34 years	3.8	7.5	8.4	12.5	16.4**	11.7
35–44 years	11.8	14.0	10.6	26.5	36.3**	22.3
45–54 years	17.3	22.6	21.2	30.9	36.7**	23.0
55–64 years	21.5	25.2	22.3	44.6	58.6	39.2
65–74 years	27.4	30.8	24.5	66.0	43.3*	27.5
FEMALE						
All ages, 25–74 years*	17.1	14.5	11.3	33.6	31.4	25.5
25–34 years	2.3	2.2	2.3	8.6**	12.4	4.3**
35–44 years	6.2	6.6**	6.5	25.7	23.8**	17.6
45–54 years	15.5	13.9	12.1	42.0	39.7	37.3
55–64 years	21.0	27.6	18.3	41.1	45.6	36.4
65–74 years	48.6	34.9	26.3	71.0	46.3	43.4

* Age adjusted by the direct method to the 1970 civilian noninstitutionalized population 25–74 years of age, using 5 age groups

** Figure does not meet standards of reliability or precision

Note: Elevated blood pressure includes readings of either systolic pressure of at least 160 mmHg or diastolic blood pressure of at least 95 mmHg or both. All blood pressures are the average of 3 measurements.

Source: National Center for Health Statistics 1984, 92 (table 37).

from 1960–1962 to 1971–1975, and then declined. The recent declines can be attributed to the initiation in 1972 of national hypertension control programs (Robins and Baum 1981) which have been linked to a decline of 15 percent in stroke incidence. Fully 80 percent of the hypertension control efforts have been initiated since 1972 (Baum and Manton 1987). Still, the proportion of persons with hypertension in the 1976–1980 period is 45 percent higher for black males and 126 percent higher for black females.

Serum Cholesterol

Table 17 presents trends in the prevalence of elevated serum cholesterol from 1960 to 1980.

The overall trends in the percentage of the population with elevated serum cholesterol have been downward for all groups except black males. Black males between the ages of 35 and 64 show increases occurring from 1960–1962 to 1971–1974. Large declines have been occurring for older females of both races.

Health Behavior

In the Alameda County study (Berkman and Breslow 1983) health behavior is prospectively linked to both morbidity and mortality. A summary measure of health behavior called the "health practices index" was used to present health habits for several types of health behavior (e.g., cigarette smoking, obesity, drinking, physical activity, and loss of sleep). The distribution of these scores (and associated mortality rates) are presented for blacks and whites in table 18.

Blacks generally score lower on the health behavior index than whites (e.g., over 25 percent of blacks score 0–2 versus 16 to 17 percent of whites). At most levels of the health practices index, the mortality rates for blacks are higher than for whites, suggesting differentials in other factors not represented in the index.

Infant Birth Weight

The infant mortality rate of blacks is twice that of whites. A major risk factor for infant mortality is birth weight. While only 7 percent of babies have low birth weight, they produce nearly 60 percent of all infant deaths. The proportion of low birth weight (less than 2,500

TABLE 17

Percentage of Persons Aged 25 to 74 with Elevated Serum Cholesterol Levels, According to Race, Sex, and Age: United States, 1960–1962, 1971–1974, and 1976–1980 (data based on physical examinations of a sample of the civilian noninstitutionalized population)

	Race and period					
	White			Black		
Sex and age	1960–1962	1971–1974	1976–1980	1960–1962	1971–1974	1976–1980
MALE						
All ages, 25–74 years*	20.6	17.4	16.8	14.2	20.0	19.4
25–34 years	10.8	7.8	8.4	9.0	14.0	9.3
35–44 years	21.7	16.5	15.4	8.4	20.7	23.6
45–54 years	26.4	25.0	20.8	21.1	20.4	25.3
55–64 years	24.6	19.4	22.8	13.7	23.0	24.2
65–74 years	21.5	20.7	19.1	22.9**	25.8	18.7
FEMALE						
All ages, 25–74 years*	26.7	20.7	20.0	24.1	20.7	20.0
25–34 years	6.8	5.6	5.9	12.0	5.4	6.5
35–44 years	12.9	9.4	9.9	12.1	10.7	13.3
45–54 years	28.0	24.6	23.8	31.0	27.2	25.8
55–64 years	51.9	36.2	35.8	29.1	34.7	32.0
65–74 years	51.4	41.2	35.6	50.1**	36.0	30.3

* Age adjusted by the direct method to the 1970 civilian noninstitutionalized population 25–74 years of age, using 5 age intervals
** Figure does not meet standards of reliability or precision
Note: Elevated serum cholesterol includes cholesterol levels of at least 260 mg/100 ml.
Source: National Center for Health Statistics 1984, 93 (table 28).

TABLE 18
Age-Adjusted Mortality Rates from All Causes (per 100): Health Practices* Index and Race, Males and Females, Aged 30–69, 1965–1974

Number of low-risk health practices*	Whites	Percentage	Blacks	Percentage
	MALES			
0–2	15.9 (307)**	17.2%	19.4 (73)	25.9%
3	10.7 (580)	32.4	10.0 (110)	39.0
4–5	5.5 (900)	50.4	7.3 (99)	35.1
Total	9.2 (1787)	100.0	11.2 (282)	100.0
	FEMALES			
0–2	11.5 (307)	15.6	10.7 (91)	25.4
3	6.6 (626)	31.7	11.7 (134)	37.3
4–5	3.4 (1040)	52.7	8.4 (134)	37.3
Total	5.8 (1973)	100.0	10.3 (359)	100.0

* Cigarette abstinence, light or no alcohol use, at least moderate physical activity, sleeping 7–8 hours/night, average height and weight
** Numbers in parentheses indicate *N* values
Source: Berkman and Breslow 1983, 103 (table 3–17).

grams at birth) babies has dropped less for blacks (from 13.8 percent to 12.5 percent) than whites (from 6.8 percent to 4.7 percent) from 1969–1971 to 1979–1981. The prevalence of low birth weight babies remains over twice as high for blacks. The size of this differential is consistent with the differential in infant mortality rates.

There is significant geographic variation of the incidence of low birth weight infants, probably due to socioeconomic differences between areas. The proportion of low birth weight infants varies from 6.2 percent in the west-north central regions to 8.0 percent in the mountains for whites in the period of 1969 to 1971. For blacks the prevalence varied from 14.0 percent in the middle Atlantic to 12.4 percent in the Pacific regions. For blacks in the period of 1979 to 1981 these

proportions dropped to 13 percent in the eastern-north central to 11.1 percent in the Pacific region.

In sum, blacks currently exhibit much poorer health potential than whites because of significantly elevated risk factors. The elevation of major risk factors explains, in part, the mortality differentials observed earlier and suggests that health differentials will persist. They suggest the potential among blacks for improvement by intervention in the exposure to risk factors. Differentials in three risk factors—smoking, hypertension, and obesity—can be addressed by direct prevention activities (e.g., public education programs, increased clinical screening, and better disease management and control). Low birth weight is also clearly an important risk factor linked with socioeconomic and cultural factors affecting maternal health. Serum cholesterol and obesity reflect risk factors associated with nutritional status.

Health Service Utilization

Differential rates of health service utilization represent an additional risk factor for poor health. In the past, health service utilization differed by race and constituted a major risk factor for poor health among blacks. Since nursing home utilization rates are lower for blacks, they become a special risk factor for the disabled elderly because current Medicare benefits do not include long-term care services. In this section, trends in three measures of health service utilization—nursing home use, physician use, and acute hospital visits—are discussed.

Nursing Home Use

A significant factor in the generally higher disability rates for blacks in the community are the differentials in nursing home utilization. The racial differences from 1963 to 1977 by age can be examined in table 19.

Nursing home utilization is higher for whites than blacks, although the differential declines with time. In 1963 the nursing home rates for those aged 65 and older were 160 percent higher for whites. This differential declined to 60 percent by 1977. The differentials vary by age. For persons aged 65 to 74 the rate of nursing home entry in 1977 is actually higher for nonwhites—a result of a trend that began

TABLE 19
Nursing Home and Personal Care Home Residents Aged 65 and Over and Number per 1,000 Population, According to Race: United States, 1963, 1969, 1973–1974, and 1977 (data based on a sample of nursing homes)

Year and age	White	All other	Ratio
1963			
65 years and over	26.6	10.3	2.6
65–74 years	8.1	5.9	1.4
75–84 years	41.7	13.8	3.0
85 years and over	157.7	41.8	3.8
1969			
65 years and over	38.8	17.6	2.2
65–74 years	11.7	9.6	1.2
75–84 years	54.1	22.9	2.4
85 years and over	221.9	52.4	4.2
1973–74*			
65 years and over	47.3	21.9	2.2
65–74 years	12.5	10.6	1.2
75–84 years	61.9	30.1	2.1
85 years and over	269.0	91.4	2.9
1977**			
65 years and over	49.7	30.4	1.6
65–74 years	14.2	16.8	0.8
75–84 years	70.6	38.6	1.8
85 years and over	229.0	102.0	2.2

* Excludes residents in personal care or domiciliary care homes
** Includes residents in domiciliary care homes
Note: For data years 1963 and 1969, Hispanic origin was not designated; therefore, Hispanics may be included in either the white or all other category. For data years 1973–1974 and 1977, Hispanics were included in the white category.
Source: National Center for Health Statistics 1984, 116 (table 55).

in 1963. This, along with the higher disability rates at ages 65 to 74 (table 13) suggests higher disability rates for all blacks (i.e., in both the nursing home and community populations) at younger ages. Such a trend toward institutionalization among blacks at ages 65 to 74 may also suggest deficits in the provision of long-term care services which, more recently, has tended to focus on improving the delivery of long-term care to persons in the community to reduce the risk of institutionalization where possible and thereby possibly improve social autonomy and quality of life among the elderly.

Above the age of 85 whites are twice as likely to be institutionalized as nonwhites, which explains part of the higher prevalence of disability in the community for blacks over the age of 85. Even when the disabled and institutional populations are summed, however, a greater proportion of blacks (about 65 percent) are functionally dependent *or* institutionalized than whites (57 percent).

In assessing the implications of the higher black disability rates it is important to know the contribution of different medical conditions to disability. For example, cognitive impairment and dementia pose special management problems for informal care givers in the community, raising the likelihood of institutionalization. To know if black-white differences in institutionalization are appropriate requires detailed analyses of what particular medical condition produced the disability in elderly black and white populations.

Physician and Hospital Visits

Table 20 records the interval since the last physician visit and the discharge rates, days of care, and average length of stay by race for 1964, 1977, and 1982.

The table shows a large increase (from 56 to 73.3 percent) in the proportion of nonwhites who visited a physician in the last year. In 1982 the proportion of nonwhites using a physician was roughly the same as for whites. White hospitalization rates (age adjusted) dropped between 1964 and 1982, while rates for blacks increased significantly. The levels of hospitalization are higher for blacks for 1982, reflecting greater black mortality.

Though the greater morbidity of blacks causes blacks to consume greater amounts of health services (except for nursing homes) than whites, blacks still have much lower life expectancy than whites and higher mortality rates for conditions operating very early on in life (like infant mortality) which cause much premature mortality. Thus, the health services blacks receive are still failing to respond adequately to a variety of medical problems. The greater consumption of health services produces economic burdens on state and federal health programs in that a large proportion of health services provided to blacks must come from Medicaid and other government programs because of blacks' poor economic status. For example, among blacks only 49.3 percent have full-year private health insurance compared to 68.7 percent for

TABLE 20

Interval since Last Physician Visit (percent of population) and Discharges, Days of Care, and Average Length of Stay in Short-stay Hospitals (per 1,000 population) for U.S. Whites and Blacks, 1964, 1977, and 1982

	1964			1977			1982		
	Whites	Blacks	Ratio	Whites	Blacks	Ratio	Whites	Blacks	Ratio
INTERVAL SINCE LAST PHYSICIAN VISIT									
<1 year	67.3	57.0	1.2	75.3	74.5	1.0	74.9	73.3	1.0
1–2 years	13.7	14.6	0.9	11.1	12.1	0.9	10.6	12.2	0.9
2+ years	17.0	21.8	0.8	12.6	12.0	1.1	12.8	12.3	1.0
HOSPITAL USE*									
Discharges/1,000	133.8	106.3	1.3	121.4	133.0	0.9	123.2	147.3	0.8
Days of care/1,000	1053.4	1141.2	0.9	962.9	1354.9	0.7	907.0	1401.4	0.6
Average length of stay	7.9	10.7	0.7	6.9	10.0	0.7	6.5	9.3	0.7

* Excludes deliveries

Source: National Center for Health Statistics 1984, 98 (table 43), 109 (table 51).

whites. For blacks, there is a greater dependence on Medicaid insurance (22.6 percent) than for whites (3.7 percent) (U.S. Public Health Service 1985).

Conclusions

In recent years, large gains in life expectancy and improvements in health status have been noted for the American population, including gains in life expectancy at advanced ages—a historically unique phenomenon. In this article, we have examined the differences between American blacks and whites on a number of parameters of health, including life expectancy, morbidity, disability, and underlying risk factors. From that evaluation, a number of inferences can be drawn. One immediate inference is that more studies of black-white health differences are needed, especially for the elderly. Particularly limited were data from longitudinal, community-based epidemiological studies which often contain little or no data on blacks (e.g., the Framingham study). A second inference is that from a policy standpoint health resources probably are not optimally distributed in terms of closing the racial gaps in health.

Though both blacks and whites exhibited increases in life expectancy and health improvements, blacks remain significantly disadvantaged on a broad range of health measures. The health differentials could be traced to a number of different factors. Exposure to risk factors contributed to a higher incidence of certain chronic diseases. Among these risk factors were cigarette consumption, alcohol consumption, socioeconomic differentials, differences in sexual and reproductive behavior, nutritional and dietary differences, hypertension, and obesity. These factors produced excess risk for cancer, stroke, cardiovascular disease, diabetes, cirrhosis, and infant mortality. In terms of premature or excess deaths, the greatest impact is found for chronic conditions between the ages of 45 and 70.

Although the emphasis in many federal minority health programs is toward improving infant and maternal health, our analysis suggests that more attention needs to be given to reducing chronic disease risk factors for minority populations. This point received emphasis from

our examination of black and white population projections which showed that, while the black population will remain significantly younger than the white population, both populations will undergo considerable "aging" and that the differences in age structures will lessen. Clearly, Medicare health benefits do not fully resolve the special health problems of black elderly—especially long-term care service needs and provision of health benefits at younger ages that could significantly affect the health of the future black elderly.

The most important factors that have an impact on the life expectancy of individuals are infant mortality, heart disease, accidents, and homicide. In the analysis of risk factors, there appeared to be specific high-risk segments of the black population with high rates of alcohol consumption, smoking, and homicide. Thus, part of the black-white differential seems to be attributed to specific groups in the black population with multiple risk-factor exposures. For example, to the extent that intravenous drug abuse is relatively higher in black young adults, deaths due to drug overdose, AIDS, and hepatitis will be growing problems in this population.

Though there are many black subpopulations with high exposure to major risk factors, black excess mortality exists despite a considerable exposure to these same risk factors in the white population. An assessment of excess mortality using an absolute health standard (i.e., SHE) shows about as much excess mortality among whites as blacks. The black excess, however, seems to be concentrated at younger ages produced in subpopulations by conditions (e.g., infant mortality, homicide, drug use) that are more sensitive to the lower socioeconomic status of blacks.

One major uncertainty, of course, in attributing this excess early disease risk to risk-factor exposures is the degree to which the early risk differentials are a product of genetic differences. There are certain well-known genetic factors (e.g., sickle cell trait) that contribute to black-white health differentials. The degree to which there are more diffuse, but highly prevalent genetic differences in the determinants of, say, hypertension or obesity (leading to increased circulatory-disease mortality risks for blacks) is currently not known, though there seems to be some cross-national evidence to support the existence of such factors; efforts to explain fully differences by purely socioeconomic factors in longitudinal studies seem not to be successful.

Furthermore, a major contributor to black excess mortality for certain diseases (e.g., cancer) is poorer quality medical care. For example, even after controlling for many individual and disease characteristics (e.g., stage of disease), there remains an elevated case fatality rate for blacks for many types of cancer. Black life expectancy continues to remain lower and mortality rates remain higher for many treatable conditions, even though the absolute volume of acute care services delivered appears to be similar in the two populations.

In addition to deficiencies in the medical services delivered to blacks, public health educational programs also are not apparently adequately reaching the black population. For example, black attitudes toward disease, treatment, screening, and outcome may significantly affect their help-seeking behavior—especially with economic constraints. Black case-fatality rates often are higher than white rates because diseases are generally presented for treatment at much later stages and because there is lower compliance with treatment. Because of higher case-fatality rates at earlier ages, there appears to be a lower prevalence of multiple chronic conditions reported at advanced ages for blacks, though black disability rates at those ages are higher.

Health service utilization data (especially on hospitalization) show the economic consequences of the poorer health of blacks. Since blacks have lower income and less private insurance coverage (U.S. Public Health Service 1985), they are more likely to utilize governmental sources of health care—especially Medicaid. Thus, the potential benefits of government intervention to improve the relative health status of blacks at multiple levels (i.e., prevention, education, improvement of acute and long-term care services) seems large. It is important to realize that while generating economic equality might require tax and other governmental policies to more equitably redistribute wealth, population health status is not a "zero-sum" game. That is, the health of the black population could be improved relative to that of the white population without in any way adversely affecting the health status of whites.

In conclusion, the differentials in health between blacks and whites are pervasive and longstanding, despite recent advances in black life expectancy. The reasons for this are numerous, and the prescriptions for change are to a large extent long term. Nonetheless, it appears clear to us that unless federal policy takes into account such factors as the relation of lower education and socioeconomic status to em-

ployment, health insurance, and access to quality medical care, as well as the detrimental impact of drugs, alcohol, and handguns on the health of young blacks, the plight of blacks relative to whites is not likely to improve. Given the direct and indirect economic consequences of allowing such health differentials to continue, a failure to reallocate federal and state resources to alleviate racial health differentials seems to be unjustified in fiscal as well as humanitarian grounds.

References

Baum, H.M., and K.G. Manton. 1987. National Trends in Stroke-related Mortality: A Comparison of Multiple Cause Mortality Data with Survey and Other Data. *Gerontologist* 27:293–300.

Berg, J.F. 1977. Economic Status and Survival of Cancer Patients. *Cancer* 39:467–77.

Berkman, L.F., and L. Breslow. 1983. *Health and Ways of Living: The Alameda County Study.* New York: Oxford University Press.

Besdine, R.W. 1984. Functional Assessment of the Elderly: Relationship between Functional Diagnoses. Paper presented at the Fifth Annual Symposium on the Elderly and Their Health—Disability in the Aged: Medical and Psychosocial Aspects, Department of Epidemiology and Preventive Medicine, University of Maryland School of Medicine, Baltimore, October 9.

Bonham, G.S., and D.W. Brock. 1985. The Relationship of Diabetes with Race, Sex and Obesity. *American Journal of Clinical Nutrition* 41:776–83.

Chiang, C.L. 1968. *Introduction to Stochastic Processes in Biostatistics.* New York: Wiley.

Fraumeni, J.F. 1975. *Persons at High Risk of Cancer.* New York: Academic Press.

Fries, J.F. 1980. Aging, Natural Death, and the Compression of Morbidity. *New England Journal of Medicine* 303:130–35.

———. 1983. The Compression of Morbidity. *Milbank Memorial Fund Quarterly/Health and Society* 61:397–419.

Harris, J.E. 1983. Cigarette Smoking among Successive Birth Cohorts of Men and Women in the United States during 1900–1980. *Journal of the National Cancer Institute* 71:474–79.

Israel, R.A., H.M. Rosenberg, and L.R. Curtin. 1986. Analytical Potential for Multiple Cause-of-Death Data. *American Journal of Epidemiology* 124:161–79.

Katz, S., and C.A. Akpom. 1976. A Measure of Primary Sociobiological Functions. *International Journal of Health Services* 6:493–508.

Keyfitz, N. 1977. What Difference Would It Make if Cancer Were Eradicated?: An Examination of the Taeuber Paradox. *Demography* 14:411–18.

Liu, K., and K.G. Manton. 1984. The Characteristics and Utilization Pattern of an Admission Cohort of Nursing Home Patients (II). *Gerontologist* 24:20–76.

Liu, K., K.G. Manton, and B. Liu. 1985. Home Health Care Expenses for the Disabled Elderly. *Health Care Financing Review* 7(2):51–58.

MacMahon, B., P. Cole, T.M. Lin, C.R. Lowe, A.P. Mirra, B. Ravnihar, E.J. Salber, V.G. Valaovas, and S. Yuasa. 1970. Age at First Birth and Breast Cancer. *Bulletin of the World Health Organization* 43:209–21.

Manton, K.G. 1980. Sex and Race-specific Mortality Differentials in Multiple Cause of Death Data. *Gerontologist* 20:480–93.

———. 1986. Past and Future Life Expectancy Increases at Later Ages: Their Implications for the Linkage of Chronic Morbidity, Disability, and Mortality. *Journal of Gerontology* 41:672–81.

Manton, K.G., and K. Liu. 1984. The Future Growth of the Long-term Care Population: Projections Based on the 1977 National Nursing Home Survey and the 1982 Long-term Care Survey. Paper presented at the Third National Leadership Conference on Long-term Care Issues, Hillhaven Foundation, Washington, March 7–9.

Manton, K.G., C.H. Patrick, and E. Stallard. 1980. Population Impact of Mortality Reduction: The Effects of Elimination of Major Causes of Death on the 'Saved' Population. *International Journal of Epidemiology* 9:111–20.

Manton, K.G., and B.J. Soldo. 1985. Dynamics of Health Changes in the Oldest Old: New Perspectives and Evidence. *Milbank Memorial Fund Quarterly/Health and Society* 63:206–85.

Manton, K.G., and E. Stallard. 1984. *Recent Trends in Mortality Analysis.* New York: Academic Press.

Manton, K.G., E. Stallard, J.P. Creason, W.B. Riggan, and M.A. Woodbury. 1985. Compartment Model Approaches for Estimating the Parameters of a Chronic Disease Process under Changing Risk Factor Exposures. *Environmental Health Perspectives* 19:151–69.

Myers, G.C., and K.G. Manton. 1984a. Compression of Mortality: Myth or Reality? *Gerontologist* 24: 346–53.

———. 1984b. Recent Changes in the U.S. Age at Death Distribution: Further Observations. *Gerontologist* 24:572–75.

Nam, C.B., and K.A. Ockay. 1977. Factors Contributing to the Mortality Crossover Pattern. Paper presented at the International Union for the Scientific Study of Population, Mexico City.

National Cancer Institute. 1984. *SEER Program: Cancer Incidence and Mortality in the United States, 1973–81.* NIH pub. no. 85–1837. Bethesda, Md.

National Center for Health Statistics. 1975. *Comparability of Mortality Statistics for the Seventh and Eighth Revisions of the International Classification of Diseases, United States.* DHEW pub. no. (HRA) 76-1340. Rockville, Md.

———. 1980. Estimates of Selected Comparability Ratios Based on Dual Coding of 1976 Death Certificates by the Eighth and Ninth Revision of the International Classification of Diseases. *Monthly Vital Statistics Report* 28(11). DHEW pub. no. (PHS) 80–1120. Hyattsville, Md.

———. 1983. *Health: United States, 1983.* Washington.

———. 1984. *Health: United States, 1984.* Washington.

———. 1986a. *Vital Statistics of the United States, 1981.* Vol. 2, Mortality, part B. DHHS pub. no. (PHS) 85–1102. Washington.

———. 1986b. Trends in Smoking, Alcohol Consumption, and Other Health Practices Among U.S. Adults, 1977 and 1983. *Advance Data,* no. 118, Public Health Service. Washington.

———. 1986c. *Health: United States, 1985.* Washington.

Page, W.F., and A.J. Kuntz. 1980. Racial and Socioeconomic Factors in Cancer Survival: A Comparison of Veterans Administration Results with Selected Studies. *Cancer* 45:1029–40.

Robins, M., and H.M. Baum. 1981. The National Survey of Stroke: Incidence. *Stroke* 12:45–57.

Rutstein, D.D., W. Berenberg, T.C. Chalmers, C.G. Child III, A.P. Fishman, and E.B. Perrin. 1976. Measuring the Quality of Medical Care: A Clinical Methodology. *New England Journal of Medicine* 294:582–88.

Ryder, N.B. 1985. Notes on Stationary Populations. *Population Index* 41:3–28.

Savage, D.G., J. Lindenbaum, E. Osserman, J. VanRyzin, and T. Garret. 1981. Survival of Black and White Patients with Multiple Myleoma at Two Hospitals (Abstract). *Proceedings of the American Association of Cancer Research* 22:537.

Schneider, E.L., and J.M. Guralnik. 1987. The Compression of Morbidity: A Dream Which May Come True Someday! *Gerontologica Biomedica Acta.* (Forthcoming.)

Siegel, J.S. 1979. Prospective Trends in the Size and Structure of the Elderly Population: Impact of Mortality Trends and Some

Implications. *Current Population Reports,* series P–23, no. 78. Washington.

U.S. Bureau of the Census. 1984. Projections of the Population of the United States, by Age, Sex, and Race: 1983 to 2080. *Current Population Reports,* series P–25, no. 952. Washington.

———. 1985. *Statistical Abstract of the United States: 1986.* Washington.

U.S. Department of Health and Human Services. 1985. *Report of the Secretary's Task Force on Black and Minority Health.* Vol. I. Washington.

U.S. Public Health Service. 1985. Changes in Health Status: Full-Year and Part-Year Coverage. *Data Preview* 21. DHHS pub. no. (PHS) 85-3377. Rockville, Md.

Verbrugge, L.M. 1983. The Social Roles of the Sexes and Their Relative Health and Mortality. In *Sex Differentials in Mortality: Trends, Determinants and Consequences,* ed. A. Lopez and L. Ruzicka, 221–45. Canberra: Department of Demography, Australian National University.

Wassertheil-Smoller, A., A. Apostolides, M. Miller, et al. 1979. Recent Status of Detection, Treatment, and Control of Hypertension in the Community. *Journal of Community Health* 5:82–93.

Wilkinson, G.S., F. Edgerton, H.J. Wallace, J. Reese, J. Patterson, and R. Priore. 1979. Delay, Stage of Disease and Survival from Breast Cancer. *Journal of Chronic Disorders* 32:365–73.

Wing, S., K. G. Manton, E. Stallard, J. E. Keil, H. A. Tyroler, and H. A. Hames. 1987. The Black/White Mortality Crossover in Two Community-based Studies. Chapel Hill: University of North Carolina, School of Public Health.

Wing, S., H. A. Tyroler, and K. G. Manton. 1985. The Participant Effect: Mortality in a Community-based Study Compared to Vital Statistics. *Journal of Chronic Disease* 38(2): 135–44.

Woodbury, M.A., and K.G. Manton. 1982. A New Procedure for the Analysis of Medical Classification. *Methods of Information in Medicine* 21:210–20.

World Health Organization. 1969. *International Classification of Diseases: Manual of the International Statistical Classification of Diseases, Injuries, and Causes of Death.* 8th revision. Geneva.

———. 1977. *Manual of the International Statistical Classification of Diseases, Injuries, and Causes of Death.* 9th revision. Geneva.

———. 1980. *International Classification of Impairments, Disabilities, and Handicaps: A Manual of Classification Relating to the Consequences of Disease.* Geneva.

———. 1982. *Demographic Yearbook 1980.* New York: Department of International Economic and Social Affairs, Statistical Office.

———. 1985. *World Health Statistics Annual.* Geneva.

Acknowledgments: Dr. Manton's efforts in this research were supported by NIA grant no. AG01159, and U.S. DHHS Task Force on Black and Minority Health contract nos. 263-SCX13345, 263-SGX13190, and 263-SGX12302. Dr. Patrick's efforts were supported by the Veterans Administration Health Services Research and Demonstration Field Program, Durham, N.C. He was formerly the data resources manager for the U.S. Department of Health and Human Services Task Force on Black and Minority Health. Dr. Johnson, currently with the National Heart, Lung and Blood Institute, was formerly the study director, U.S. Department of Health and Human Services Task Force on Black and Minority Health.

Address correspondence to: Kenneth G. Manton, Ph.D., Research Professor, Center for Demographic Studies, Duke University, 2117 Campus Drive, Durham, NC 27706.