Trends in the Black-White Life Expectancy Gap in the United States, 1983-2003

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Trends in the Black-White Life Expectancy Gap in the United States, 1983-2003

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Context Since the early 1980s, the black-white gap in life expectancy at birth increased sharply and subsequently declined, but the causes of these changes have not been investigated.

Objective To determine the contribution of specific age groups and causes of death contributing to the changes in the black-white life expectancy gap from 1983-2003.

Design and Setting US vital statistics data from the US National Vital Statistics System, maintained by the National Center for Health Statistics. Standard life table techniques were used to decompose the change in the black-white life expectancy gap by combining absolute changes in age-specific mortality with relative changes in the distribution of causes of death.

Main Outcome Measure The gap in life expectancy at birth between blacks and whites.

Results Among females, the black-white life expectancy gap increased 0.5 years in the period 1983-1993, primarily due to increased mortality from human immunodeficiency virus (HIV) (0.4 years) and slower declines in heart disease (0.1 years), which were somewhat offset by relative improvements in stroke (–0.1 years). The gap among males increased by 2 years in the period 1983-1993, principally because of adverse changes in HIV (1.1 years), homicide (0.5 years), and heart disease (0.3 years). Between 1993 and 2003, the female gap decreased by 1 year (from 5.59 to 4.54 years). Half of the total narrowing of the gap among females was due to relative mortality improvement among blacks in heart disease (–0.2 years), homicide (–0.2 years), and unintentional injuries (–0.1 years). The decline in the life expectancy gap was larger among males, declining by 25% (from 8.44 to 6.33 years). Nearly all of the 2.1-year decline among males was due to relative mortality improvement among blacks at ages 15 to 49 years (–2.0 years). Three causes of death accounted for 71% of the narrowing of the gap among males (homicide [–0.6 years], HIV [–0.6 years], and unintentional injuries [–0.3 years]), and lack of improvement in heart disease at older ages kept the gap from narrowing further.

Conclusions After widening during the late 1980s, the black-white life expectancy gap has declined because of relative mortality improvements in homicide, HIV, unintentional injuries, and, among females, heart disease. Further narrowing of the gap will require concerted efforts in public health and health care to address the major causes of the remaining gap from cardiovascular diseases, homicide, HIV, and infant mortality.

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LIFE EXPECTANCY AT BIRTH (THE average number of years an individual can expect to live under current age-specific mortality rates)[1] has generally been increasing in the United States since at least the late 19th century.[2] Additionally, for as long as data have been given by race/ethnicity, life expectancy of blacks has been lower than that of whites.[3,4] However, overall trends tend to obscure the fact that the gap in life expectancy between blacks and whites has varied considerably during the 20th century. The near elimination of typhoid and other waterborne communicable diseases improved black life expectancy in both absolute and relative terms compared with whites in the period 1900-1940, but black-white differences stabilized during the 1960s.[5]

Blacks again made absolute and relative progress compared with whites during the 1970s and early 1980s, but the study by Kochanek et al[6] found that the black-white gap in life expectancy at birth increased between 1984 and 1989, primarily due to slower declines in heart disease among blacks relative to whites and faster increases in homicide and human immunodeficiency virus (HIV)–related mortality among young blacks. The gap continued to widen until the early 1990s, but since 1993, the black-white gap in life expectancy at birth has declined to historically low levels (from a 7.1-year gap in 1993 to a 5.3-year gap in 2003) (FIGURE 1).[7] The extent to which certain age groups and causes of death may have contributed to the increase and decrease in the black-white life expec-

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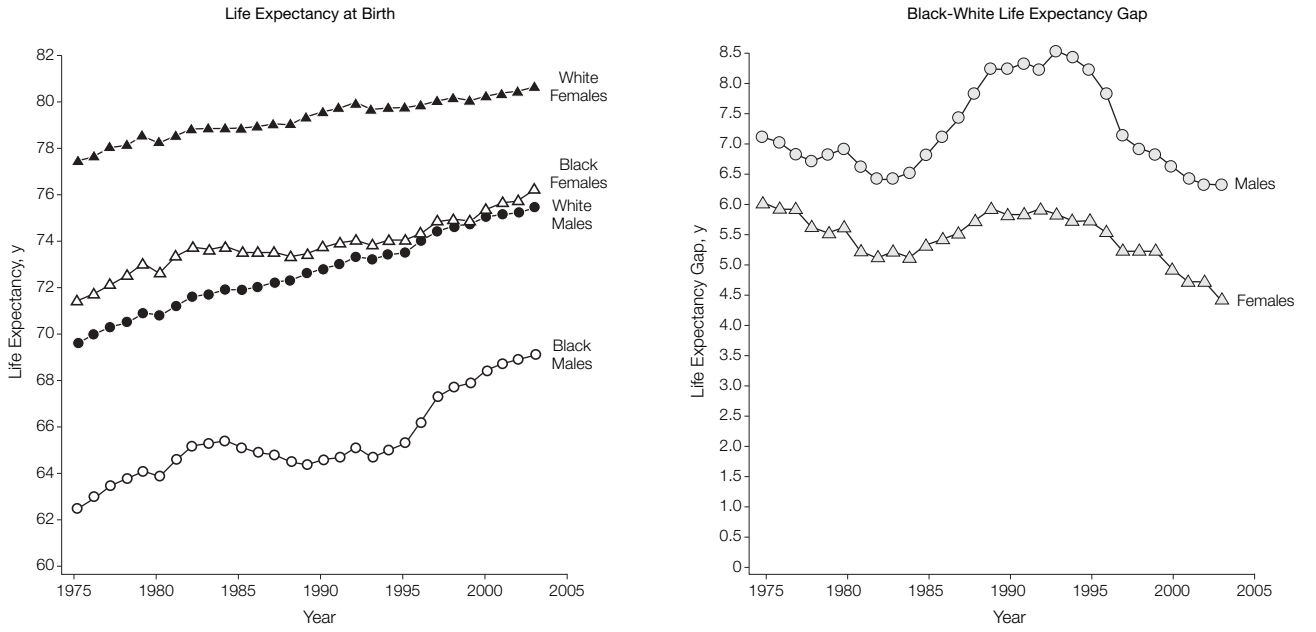
tancy gap since the early 1980s is unknown, and quantifying their contribution may help in formulating public health and health care policies aimed at further reducing black-white mortality differences. Therefore, our goal was to determine the age- and cause-specific components of the widening gap in life expectancy at birth between blacks and whites in 1983-1993, and the narrowing of the gap in 1993-2003.

METHODS

The underlying data source for our study was the US National Vital Statistics System, maintained by the National Center for Health Statistics, which collects information on all deaths occurring in the United States each year. We calculated mortality rates by single-year-of-age and cause of death between 1983 and 2003 using software from the National Cancer Institute's Surveillance Research Program,[8] for which the National Center for Health Statistics provides the underlying mortality data and population estimates.[9] Using the age-specific mortality rates and data on live births by sex and race of mother obtained from published estimates,[10,11] we calculated life expectancy at birth for black and white males and females using standard life table methods.[1] The black-white gap in life expectancy at birth is a function of black-white differences in age-specific mortality rates. To measure the contribution of age-specific mortality changes to the change in the black-white life expectancy gap, we used the decomposition method developed by Arriaga.[12] We took the estimated values from the life table for infant mortality and each 5-year age group up to age 85 years to calculate the contribution of black-white differences in age-specific mortality rates to the overall gap in life expectancy at birth in 1983, 1993, and 2003. For a given year, the overall gap in life expectancy at birth is the sum of the age-specific components (measured in years), and the contribution of each age group to the change in the overall life expectancy gap for a given period is difference between each age group's contribution in the initial year and the final year.

Because black-white differences in age-specific mortality rates are a reflection of differences in cause-specific mortality, the total number of years a particular age group contributes to the overall life expectancy gap may also be partitioned by cause of death. Assuming that the contribution of each cause of death to the total contribution for an age group is proportional to the contribution of each cause of death to the black-white difference in the age-specific mortality rate, the overall difference in life expectancy at birth is the sum of the age-cause-specific components.[13] In a given year, higher cause-specific mortality rates among blacks make the overall black-white life expectancy gap wider, whereas higher cause-specific mortality rates among whites make the gap smaller. The overall contribution of a cause of death to the black-white life expectancy gap in a given year is the sum of its contribution across all age groups, and the contribution of each cause of death to the change in the gap in life expectancy at birth is the difference between each cause's contribution at the beginning and end of the period. Because mortality has generally been improving over time, even when a cause of death is declining among both blacks and whites, black-white differences in the rate of decline will change

Figure 1. Life Expectancy at Birth Among Black and White Males and Females in the United States and the Black-White Life Expectancy Gap, 1975-2003



Data taken from the United States Life Tables of the National Center for Health Statistics.[7]

the black-white life expectancy gap. That is, for a particular cause of death that is declining, faster mortality declines among blacks contribute to narrowing the overall life expectancy gap, and faster declines among whites contribute to widening the gap.

For the cause-specific analyses, we included the 10 leading causes of death for each sex and racial/ethnic group,[14] with separate categories for major cancers (lung, breast, colorectal, prostate) and major causes of infant mortality (perinatal death, congenital anomalies). For simplicity, we used only the "underlying" cause of death reported on the death certificate, defined as "the disease or injury which initiated the train of events leading directly to death, or the circumstances of the accident or violence which produced the fatal injury."[15] To minimize artifactual changes in cause-specific mortality due to the *International Statistical Classification of Diseases, 10th Revision*, which took effect in 1999, we used the cause-of-death recoding scheme developed by the National Cancer Institute designed to facilitate analysis of long-term trends.[16]

RESULTS

Between 1983 and 1993, the life expectancy gap between black and white females increased by 0.5 years, but between 1993 and 2003, it declined by 1 year from 5.59 to 4.54 years, an 18% reduction. Males experienced somewhat larger changes during both periods, particularly during 1983-1993, when the black-white gap increased by 31% (2 years) compared with only a 10% increase for females. After 1993, relative improvement in the black-white gap among males was similar to females (25%) but the reduction was twice as large in absolute terms (declining by 2.1 years from 8.44 years in 1993 to 6.33 years in 2003).

Contributions to the Black-White Gap by Cause of Death and Age

TABLE 1 shows the distribution of major causes of death contributing to the black-white life expectancy gap in 1983, 1993, and 2003. For females, heart disease made by far the largest contribution to the overall life expectancy gap, accounting for 1.4 years (28%) of the gap in 1983, 1.6 years (28%) in 1993, and 1.3 years (30%) in 2003. Other important causes of the 4.5-year gap in 2003 include diabetes (0.5 years), stroke (0.4 years), and perinatal death (0.4 years). Among males, the largest contributor to the gap was homicide in 1983 (1.1 years) and 1993 (1.6 years), but by 2003 heart disease became the leading cause, accounting for 1.4 years (21%) of the overall 6.3-year gap, followed by homicide (1 year), HIV (0.6 years), and perinatal death (0.4 years).

TABLE 2 shows the contribution of age groups to the black-white gap in 1983, 1993, and 2003. The leading age group contributing to the gap in 1983 was infants for both males (0.8 years) and females (0.7 years), and it remained the leading cause among females (0.6 years) in 2003. Over time, the black-white gap has become more concentrated at older ages. The contribution of black-white mortality differentials among those individuals 65 years or older increased from 0.6 years (10%) in 1983 to 1.3 years (21%) in 2003 among males, and from 0.9 years (18%) to 1.1 years (24%) among females. In 2003, black-white mortality differentials at ages 45 to 64 years account for the largest share (roughly 40%) of the black-white gap among males (2.6 years) and females (1.9 years).

Change in the Black-White Gap by Age

FIGURE 2 shows the contribution of specific age groups to the change in the black-white life expectancy gap. Each bar indicates the net contribution of mortality changes in each age group to the overall change in the life expectancy gap (in years), and the percentage of the total change is also shown. The pattern was noticeably different by sex. Between 1983 and 1993, increases in mortality among black females aged 25 to 44 years widened the gap 0.3 years (accounting for 61% of the total net change), and increasing mortality for those aged 85 years or older added 0.2 years (45%). The decline in the female gap between 1993 and 2003 occurred across a fairly broad range of ages. Relatively faster mortality improvement among black females ages 20 to 49 years decreased the life expectancy gap by 0.56 years (53%), and another 0.24 years (23%) was due to improving mortality among females ages 65 to 84 years.

Among males, increases in mortality at younger ages and slower mortality declines at older ages among blacks increased the life expectancy gap between 1983 and 1993. The largest contribution was among those individuals aged 15 to 24 years, which added 0.7 years (35%) to the black-white gap, and those individuals aged 65 years or older increased the gap by another 0.6 years (28%). Between 1993 and 2003 in contrast with the more varied age pattern for females, the decline in the life expectancy gap among males was almost entirely due to relative mortality improvement among blacks aged 15 to 49 years, which alone reduced the overall gap by 2 years (95% of the total). Improvements in infant and child (<15 years) death rates also reduced the gap by 0.18 years (8%), but this gain was entirely offset by adverse mortality changes among those individuals aged 60 to 84 years, which widened the gap by 0.25 years (–12%).

Change in the Black-White Gap by Cause of Death

FIGURE 3 presents the decomposition of the change in the gap by underlying cause of death. The 2-year increase in the male life expectancy gap between 1983 and 1993 was mostly due to unfavorable mortality changes among blacks for HIV (1.1 years [55.4%]), homicide (0.5 years [26.0%]), and heart disease (0.3 years [16.0%]). However, the decline in the male gap between 1993 and 2003 did not exactly mirror the period of increase (ie, the causes of the decline in the black-white gap, while similar, were not exactly the causes of the increase between 1983 and 1993). Rapid mortality declines in blacks from homicide and HIV accounted for 55% of the overall decline, narrowing the gap by 0.60 years and 0.57 years, respectively, but changes in mortality from unintentional injures, which played little role in the 1983-1993 increase, narrowed the gap by another 0.33 years (16%). Consistent with these causes,

nearly all of the improvement in these causes resulted from mortality changes among young and middle-aged males (ages 15-49 years). Most of the remaining decline in the life expectancy gap among males (0.4 years) was due to cancers other than lung, colorectal, and prostate (9%), lung cancer (5%), and cirrhosis (5%). For simplicity, Figure 3 only shows the net change for each cause of death across all age groups, but looking at age and cause of death simultaneously also revealed in some cases divergent age patterns for the same cause of death (data not shown). For example, among males, heart disease widened the gap by only 0.03 years. However, this net effect masks the fact that mortality among blacks at younger ages (40-59 years) narrowed the life expectancy gap by 0.15 years, whereas relatively slower mortality declines at older ages (60-84 years) offset this improvement and widened the gap by 0.18 years.

Human immunodeficiency virus was the major reason for the increase among females between 1983 and 1993, adding 0.4 years to the black-white gap (84%), with another 0.4 years added collectively from heart disease (26%), diabetes (22%), breast cancer (20%), and homicide (13%). Some of the increase in the gap was offset by relative improvements in stroke (–29%), cirrhosis (–15%), and chronic lower respiratory diseases (–11%), which decreased the gap by 0.3 years. Similar to the pattern for males, the causes contributing most to the 1993-2003 decline among females were similar to, but not exactly, those responsible for the 1983-1993 increase. However, in contrast with males, the leading contributor to the decline among females was heart disease, primarily among those individuals aged 45 to 74 years (Figure 3), which reduced the gap by 0.21 years (20% of the total decline). Other causes making notable contributions were homicide (16%), unintentional injuries (14%), cancers other than

Table 1. Causes of Death Contributing to the Black-White Life Expectancy Gap in 1983, 1993, and 2003 by Sex

	Life Expectancy Gap, Years (%)					
	Males			Females		
Cause of Death	1983	1993	2003	1983	1993	2003
Cardiovascular	1.51 (23.5)	1.81 (21.5)	1.92 (30.3)	2.03 (40.1)	2.10 (37.6)	1.90 (41.9)
Heart disease	1.00 (15.6)	1.32 (15.6)	1.35 (21.3)	1.41 (27.8)	1.55 (27.7)	1.34 (29.5)
Hypertension	0.06 (1.0)	0.08 (1.0)	0.15 (2.3)	0.08 (1.7)	0.13 (2.3)	0.16 (3.5)
Stroke	0.45 (7.0)	0.40 (4.7)	0.39 (6.2)	0.55 (10.8)	0.40 (7.1)	0.37 (8.2)
Other CVD	–0.01 (–0.1)	0.02 (0.2)	0.03 (0.5)	0.00 (–0.1)	0.03 (0.6)	0.03 (0.7)
Cancer	1.14 (17.8)	1.24 (14.7)	0.99 (15.6)	0.54 (10.6)	0.64 (11.5)	0.57 (12.6)
Colorectal	0.03 (0.4)	0.08 (1.0)	0.12 (1.9)	0.05 (1.1)	0.11 (2.0)	0.13 (2.8)
Lung	0.39 (6.1)	0.42 (4.9)	0.30 (4.8)	0.02 (0.5)	–0.01 (–0.2)	–0.01 (–0.2)
Breast	0	0	0	0.05 (1.0)	0.15 (2.7)	0.20 (4.4)
Prostate	0.20 (3.1)	0.27 (3.1)	0.27 (4.3)	0	0	0
Other	0.52 (8.1)	0.48 (5.6)	0.29 (4.6)	0.41 (8.0)	0.39 (7.0)	0.25 (5.6)
Infectious	0.40 (6.3)	1.53 (18.1)	0.89 (14.1)	0.23 (4.5)	0.72 (12.9)	0.56 (12.4)
Flu/pneumonia	0.19 (2.9)	0.17 (2.0)	0.10 (1.6)	0.02 (0.4)	0.06 (1.0)	0.02 (0.5)
Septicemia	0.09 (1.5)	0.11 (1.3)	0.19 (2.9)	0.13 (2.5)	0.13 (2.2)	0.20 (4.4)
HIV	0	1.12 (13.2)	0.55 (8.7)	0	0.44 (7.8)	0.31 (6.7)
Other	0.12 (1.9)	0.13 (1.5)	0.06 (0.9)	0.08 (1.6)	0.10 (1.8)	0.03 (0.7)
Other causes	0.42 (6.6)	0.45 (5.4)	0.50 (7.9)	0.50 (9.8)	0.46 (8.3)	0.41 (9.0)
Alzheimer	–0.01 (–0.1)	–0.01 (–0.1)	–0.02 (–0.3)	–0.01 (–0.3)	–0.03 (–0.6)	–0.08 (–1.7)
CLRD	–0.02 (–0.4)	0.02 (–0.2)	–0.02 (–0.3)	–0.08 (–1.5)	–0.13 (–2.4)	–0.22 (–4.8)
Diabetes	0.14 (2.1)	0.22 (2.6)	0.30 (4.7)	0.30 (5.9)	0.41 (7.4)	0.45 (9.9)
Nephritis	0.12 (1.8)	0.12 (1.4)	0.24 (3.8)	0.16 (3.1)	0.16 (2.8)	0.27 (5.8)
Cirrhosis	0.20 (3.2)	0.10 (1.2)	0	0.13 (2.6)	0.05 (1.0)	–0.01 (–0.2)
Injuries	1.17 (18.2)	1.82 (21.6)	0.83 (13.0)	0.27 (5.3)	0.36 (6.4)	0.04 (0.9)
Homicide	1.07 (16.6)	1.59 (18.8)	0.99 (15.6)	0.25 (5.0)	0.32 (5.8)	0.15 (3.4)
Suicide	–0.17 (–2.6)	–0.12 (–1.5)	–0.19 (–2.9)	–0.09 (–1.8)	–0.07 (–1.2)	–0.07 (–1.5)
Unintentional injuries	0.27 (4.2)	0.36 (4.2)	0.02 (0.4)	0.11 (2.1)	0.11 (1.9)	–0.04 (–1.0)
Infant mortality	0.53 (8.2)	0.53 (6.3)	0.49 (7.8)	0.49 (9.6)	0.47 (8.3)	0.40 (8.9)
Congenital anomalies	0.03 (0.5)	0.04 (0.5)	0.05 (0.8)	0.03 (0.5)	0.04 (0.7)	0.03 (0.6)
Perinatal death	0.50 (7.7)	0.49 (5.8)	0.44 (7.0)	0.46 (9.1)	0.43 (7.6)	0.37 (8.2)
Residual	1.25 (19.4)	1.05 (12.4)	0.71 (11.2)	1.01 (20.0)	0.84 (15.0)	0.65 (14.4)
Total	**6.42 (100)**	**8.44 (100)**	**6.33 (100)**	**5.07 (100)**	**5.59 (100)**	**4.54 (100)**

Abbreviations: CLRD, chronic lower respiratory disease; CVD, cardiovascular disease; HIV, human immunodeficiency virus.

lung, colorectal, and breast (13%), and HIV (13%). The above 5 causes together accounted for 0.8 years (76%) of the 1-year net decline in the female life expectancy gap. Finally, Figure 3 also shows that, despite the overall reductions in the black-white life expectancy gap since 1993 among both males and females, the gap would have declined even more were it not for unfavorable changes in black mortality (relative to whites) from nephritis, septicemia, breast cancer, and diabetes.

COMMENT

A previous study[6] showed that changes in heart disease, HIV, and homicide widened the black-white life expectancy gap between 1984 and 1989. Our study confirms that this pattern continued through 1993, but reversed course thereafter. However, the recent decline in the black-white gap does not appear to be due to general mortality improvement among blacks. Rather, the decline reflects relative mortality improvement for specific age groups and causes of death. Among males, changes in homicide, HIV, and unintentional injuries accounted for 71% of the decline in the black-white gap, 91% of which occurred among males aged 15 to 49 years. These same causes also accounted for 43% of the decline in the gap among females and, if heart disease is included, the proportion explained among females increased to 63% for these 4 causes of death.

Homicide

Homicide remains a dominant determinant of black-white mortality patterns,[6,17] but the causes of changes in homicide mortality likely differ by age[18] and time period.[19] There is strong evidence that the increase in homicide during the mid-1980s was linked to the spread of crack-cocaine markets,[20,21] and the homicide decreases observed in our analysis likely resulted from some combination of changes in the crack market, increased incarceration rates, increases in the size of police forces, and economic changes.[18,22] Despite these decreases, homicide remains a leading contributor to the black-white life expectancy gap among males, and strategies to reduce homicide mortality among blacks are urgently needed.

Human Immunodeficiency Virus

Mortality from HIV disproportionately contributed to the widening of the black-white life expectancy gap in the late 1980s,[6] but made a substantial contribution to narrowing the black-white life expectancy gap since 1993, particularly among males. Although the relative mortality decline (ie, the percentage reduction) was larger for whites, the absolute decline was far larger for blacks. The steep declines in HIV mortality were due to the widespread and rapid introduction of highly-active antiretroviral therapy (HAART).[23,24] Given evidence

Table 2. Age Groups Contributing to the Black-White Life Expectancy Gap in 1983, 1993, and 2003 by Sex

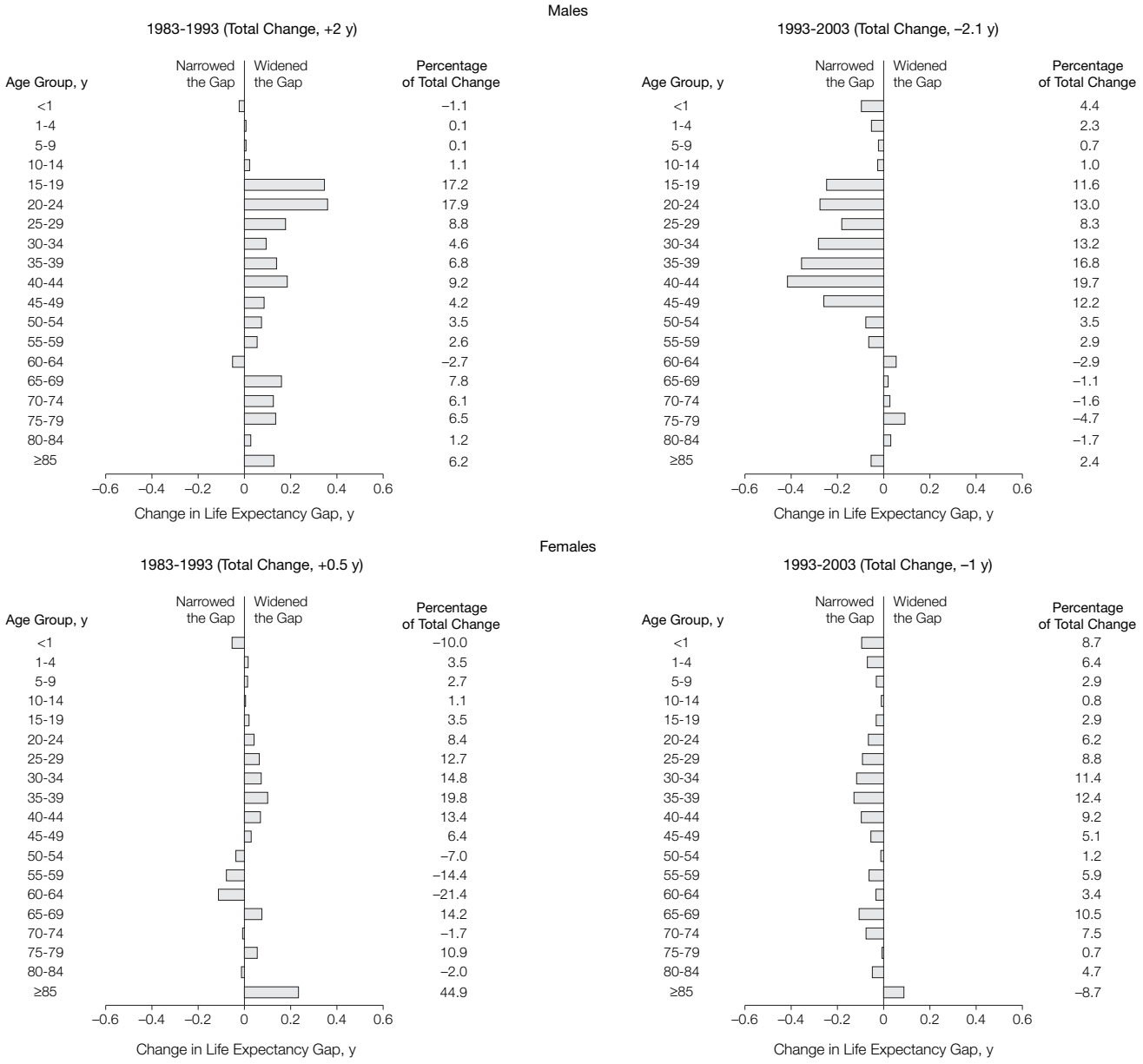
	Life Expectancy Gap, Years (%)					
	Males			Females		
Age Group, y	1983	1993	2003	1983	1993	2003
<1	0.80 (12.4)	0.77 (9.2)	0.68 (10.8)	0.73 (14.4)	0.68 (12.1)	0.59 (12.9)
1-14	0.17 (2.7)	0.20 (2.4)	0.12 (1.8)	0.15 (3.0)	0.19 (3.4)	0.08 (1.8)
1-4	0.11 (1.7)	0.11 (1.3)	0.06 (1.0)	0.09 (1.8)	0.11 (2.0)	0.04 (0.9)
5-9	0.04 (0.5)	0.04 (0.4)	0.02 (0.3)	0.04 (0.7)	0.05 (0.9)	0.02 (0.5)
10-14	0.03 (0.5)	0.05 (0.6)	0.03 (0.5)	0.02 (0.4)	0.03 (0.5)	0.02 (0.4)
15-44	2.01 (31.3)	3.31 (39.2)	1.57 (24.8)	1.04 (20.4)	1.41 (25.3)	0.88 (19.4)
15-19	–0.01 (–0.1)	0.34 (4.0)	0.10 (1.5)	0.01 (0.1)	0.03 (0.5)	–0.01 (–0.1)
20-24	0.16 (2.5)	0.52 (6.2)	0.25 (3.9)	0.09 (1.8)	0.13 (2.4)	0.07 (1.5)
25-29	0.31 (4.8)	0.48 (5.7)	0.31 (4.9)	0.14 (2.8)	0.21 (3.7)	0.12 (2.5)
30-34	0.45 (7.0)	0.54 (6.4)	0.26 (4.2)	0.20 (4.0)	0.28 (5.0)	0.16 (3.5)
35-39	0.51 (7.9)	0.65 (7.6)	0.29 (4.6)	0.26 (5.1)	0.36 (6.5)	0.23 (5.2)
40-44	0.59 (9.2)	0.77 (9.2)	0.36 (5.7)	0.33 (6.6)	0.40 (7.2)	0.31 (6.8)
45-64	2.82 (44.0)	2.97 (35.3)	2.64 (41.8)	2.24 (44.1)	2.05 (36.6)	1.88 (41.4)
45-49	0.69 (10.8)	0.78 (9.2)	0.52 (8.2)	0.43 (8.5)	0.46 (8.3)	0.41 (9.0)
50-54	0.70 (11.0)	0.77 (9.2)	0.70 (11.1)	0.53 (10.6)	0.50 (8.9)	0.49 (10.7)
55-59	0.73 (11.3)	0.78 (9.2)	0.72 (11.4)	0.62 (12.3)	0.55 (9.8)	0.49 (10.7)
60-64	0.70 (10.9)	0.64 (7.6)	0.71 (11.1)	0.65 (12.8)	0.54 (9.6)	0.50 (11.1)
≥65	0.62 (9.6)	1.18 (14.0)	1.32 (20.8)	0.92 (18.1)	1.26 (22.6)	1.11 (24.4)
65-69	0.37 (5.8)	0.53 (6.3)	0.55 (8.7)	0.48 (9.5)	0.55 (9.9)	0.44 (9.8)
70-74	0.30 (4.7)	0.42 (5.0)	0.46 (7.2)	0.48 (9.4)	0.47 (8.4)	0.39 (8.5)
75-79	0.06 (0.9)	0.19 (2.2)	0.29 (4.5)	0.23 (4.5)	0.29 (5.1)	0.28 (6.1)
80-84	0.06 (0.9)	0.08 (1.0)	0.12 (1.9)	0.17 (3.4)	0.16 (2.9)	0.11 (2.5)
≥85	–0.17 (–2.7)	–0.04 (–0.5)	–0.10 (–1.5)	–0.44 (–8.7)	–0.21 (–3.7)	–0.12 (–2.5)
Total	**6.42 (100)**	**8.44 (100)**	**6.33 (100)**	**5.07 (100)**	**5.59 (100)**	**4.54 (100)**

that the effectiveness of HAART is similar in blacks and whites,[25,26] differences in the relative mortality declines most likely result from differential access to and quality of treatment among blacks.[27-29] Despite this success, HIV mortality remains a leading contributor to the remaining black-white gap (Table 1). The delivery of proven primary prevention interventions, cost-effective voluntary testing, and expanded access to HAART, especially for minorities, should remain high public health priorities.[30-34]

Unintentional Injuries

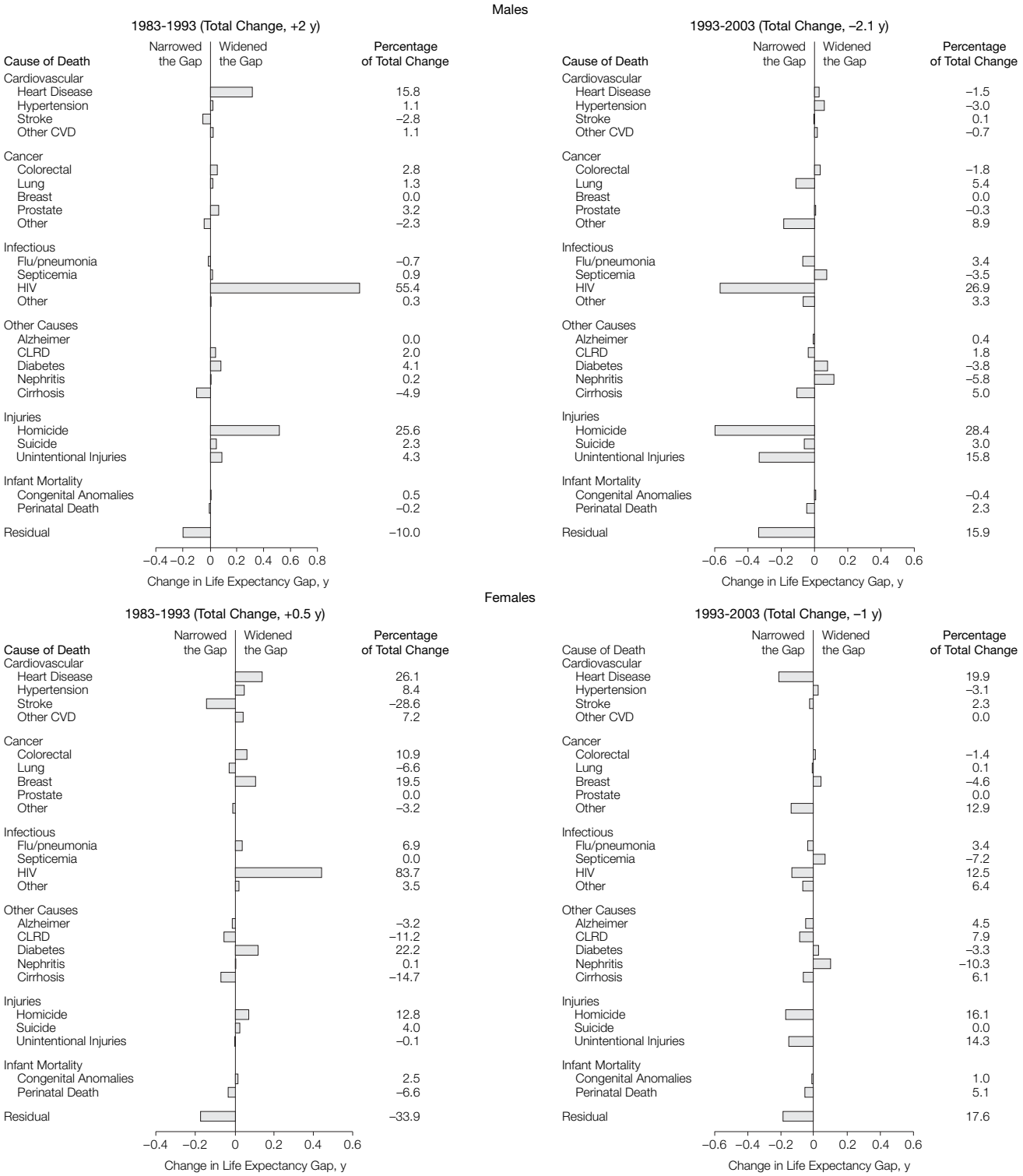
Changes in mortality from unintentional injuries were the third most important cause of the decline in the gap. Underlying mortality trends showed not only faster mortality declines among blacks, but also, in contrast with the patterns for homicide and HIV, increasing mortality rates among middle-aged whites. This finding is likely due to increases among whites in deaths due to unintentional poisoning, which is the

Figure 2. Major Age Groups Contributing to the Change in the Black-White Gap in Life Expectancy at Birth Among Males and Females in the United States, 1983-1993 and 1993-2003



Each bar indicates the net contribution of mortality changes in each age group to the overall change in the life expectancy gap in years.

Figure 3. Leading Causes of Death Contributing to the Change in the Black-White Gap in Life Expectancy at Birth Among Males and Females in the United States, 1983-1993 and 1993-2003



CVD indicates cardiovascular disease; HIV, human immunodeficiency virus; CLRD, chronic lower respiratory disease. Each bar indicates the net contribution of mortality changes in each cause of death to the overall change in the life expectancy gap in years.

leading cause of unintentional injury death among US middle-aged adults.[35] The majority of unintentional poisoning deaths, which began increasing in the mid-1980s,[36,37] are related to narcotics.[35] However, since the mid-1990s, the rate of increase appears to be steepening among whites but not blacks, which is consistent with reported increases in drug abuse–related emergency department episodes,[38] opioid abuse,[39] and unintentional death[40,41] from prescription drugs among non-Hispanic white populations.

Causes of the Current Black-White Gap

The current difference in life expectancy at birth between blacks and whites remains substantial (6.3 years for males and 4.5 years for females). Homicide, HIV, and perinatal death, although demonstrating favorable trends, continue to keep the black-white gap unnecessarily large, and unfavorable mortality trends in nephritis and septicemia are currently contributing to widening the gap. However, cardiovascular-related diseases remain the leading cause of black-white differences in life expectancy. If all cardiovascular causes and diabetes are considered together, they account for 35% and 52% of the gap for males and females, respectively. Trends in heart disease among males younger than 60 years and most female age groups reduced the life expectancy gap, but trends among males 60 years or older widened the gap. Previous articles documented slower absolute and relative declines in heart disease mortality among blacks from the mid-1970s to the early 1990s,[42,43] but our results suggest some recent improvement among blacks at younger ages.

Despite some indications of improvement, future efforts to reduce the black-white gap should be aimed at reducing existing inequalities in cardiovascular risk factors, particularly hypertension, and improving access to effective treatments for existing disease that remain underused.[44] For example, Cutler et al[45] estimate that effective use of existing antihypertensive drugs would have reduced approximately 90 000 premature deaths from cardiovascular disease in 2001.

Limitations

Life expectancy at birth is a weighted summary of age-specific mortality rates and is sensitive to mortality changes at young ages.[1] Analyses of changes using different summary measures of population health, such as disability-adjusted life years, might provide different results. Using a single underlying cause of death simplifies the fact that many deaths are due to multiple causes, and our analysis may underestimate the impact of common conditions associated with multiple causes of death (eg, diabetes, influenza, and pneumonia).[46] However, given that a large part of the observed decline in the black-white gap was due to mortality at younger ages, the use of multiple-cause data are unlikely to alter our basic findings.

We used 1983 and 1993 for this analysis because they generally represented the most recent low and high points in the black-white life expectancy gap. Mortality rates are not subject to sampling error,[7] but analyses using a different period of time could alter the estimated contribution of specific causes of death. However, given the dramatic changes in mortality from homicide and HIV during the 1990s, it seems unlikely that using different years would fundamentally change our results.

Finally, our analysis focused on quantifying the age- and cause-specific contributions to the declining life expectancy gap but did not investigate potential causes of changes in black-white mortality patterns. Future research is needed to elicit such causes, but our results suggest that the decline in the black-white life expectancy gap is due to factors specific to particular causes of death rather than changes in general social and economic circumstances. Social and economic progress among blacks was at best uneven during the 1990s,[47] and some recent studies suggest that the 1990s were a particularly bad decade for young black males relative to other groups, with increasing rates of incarceration, stagnant wages, and declines in the fraction of the population working.[48,49] Despite these unfavorable developments, black males gained more years of life expectancy than any other group in our analysis, which suggests a complex and dynamic rather than deterministic relationship between mortality and broader social processes.[50]

CONCLUSION

Recent life expectancy trends[51] indicate positive movement toward one of the major US public health goals of eliminating black-white mortality inequalities,[52] but substantial inequalities and challenges remain. Reducing social and individual risk factors for major causes of death and improving access and quality of care for blacks, particularly for cardiovascular diseases, should be a pressing priority for public health and health care.

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