WHY HAS THE BLACK-WHITE LIFE EXPECTANCY GAP RECENTLY DECLINED?

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Abstract

Background

Understanding which age groups and causes of death contribute most to the change in the black-white life expectancy gap may be a first step toward understanding the causes of and remedies for health inequalities between blacks and whites.

Methods

We decomposed the change in the black-white life expectancy gap by sex from 1990 to 2001 for 13 age groups and 27 causes of death using Arriaga's method of combining absolute changes in age-specific mortality with relative changes in the distribution of age-specific causes over time.

Results

From 1990 to 2001 the gap in life expectancy between black and white females decreased from 5.81 to 4.75 years. Most of the decrease was due to relative mortality improvement among black females aged 25-64 (51.0%), with important contributions among infants (16.2%) and those 85 and over (12.9%). With respect to cause, most of the decline was due to relative mortality improvement among blacks in cardiovascular diseases (23.9%), cancers (12.7%), homicide (9.4%), and liver cirrhosis (6.0%). The decline in the black-white life expectancy gap was larger among males, declining from 8.19 to 6.47 years by 2001. The decline among males was primarily due to relative mortality improvement among blacks ages 25-54 (71.9%). Chief among the causes for the decline were relative improvement among blacks in mortality from homicide (22.2%), cancers (15.5%), cardiovascular diseases (8.6%), HIV (7.9%), non-motor vehicle injuries (7.2%), lung cancer (6.1%), and liver cirrhosis (5.5%). Unfortunately, unfavorable changes in other causes of death for blacks relative to whites kept the racial gap

from narrowing further, primarily ischemic heart disease among those 65 and over, HIV among those 45-64, nephritis, breast and prostate cancer, diabetes, and septicemia.

Conclusions

After widening during the late 1980s, the black-white life expectancy gap has narrowed substantially. However, despite recent improvements, future declines in the black-white life expectancy gap will require continued relative mortality declines for blacks in ischemic heart disease, homicide, HIV, and infant mortality.

Two generalizations might be made about historical trends in life expectancy in the United States. The first is that life expectancy has generally been increasing since at least the late 19th century.[1] The second is that, for as long as data on life expectancy have been given by race, the observed life expectancy of blacks has been lower than that of whites.[2,3] But such generalizations tend to obscure the fact that changes in life expectancy are due to mortality changes in different periods that may differ widely with respect to age, gender, race, and major causes of death. Increases in life expectancy from the late 19th century to the mid-20th were largely due to declines in communicable disease mortality, with greater mortality reductions among the young, while a majority of the gain in life expectancy since 1950 has been due to declines in non-communicable disease mortality among those over 50,[4,5] especially from cardiovascular disease.

Similarly, the gap in life expectancy between blacks and whites has varied considerably over the 20th century due to changes in age and cause-specific mortality. The near elimination of typhoid and other waterborne communicable diseases improved black life expectancy in both absolute terms and relative to whites from 1900-1940, but black-white differences stagnated during the 1960s.[6] Blacks again made relative progress during the 1970s and early 1980s, but Kochanek et al.[7] found that black life expectancy declined from 1984 to 1989, primarily due to slower declines in heart disease among older blacks and increases in homicide and human immunodeficiency virus (HIV) among the young.

More recently, life expectancy at birth has increased for all race-gender groups, and the black-white gap in life expectancy has declined once again since the late 1980s, particlarly for males. However, the extent to which certain age groups and causes of death may have contributed to this change is unknown. The purpose of this study was to determine the

component causes of changes in overall U.S. life expectancy and changes in the black-white life expectancy gap between 1990-2001.

Methods

The information used to calculate life expectancy was abstracted from published[4,8] complete life tables for 1990 and 2001 for the total US population and four race-gender groups (black females, black males, white females, white males). Changes in life expectancy are a function of changes in age-specific mortality rates, and we calculated the contribution of mortality changes at each age using the decomposition method developed by Arriaga.[9] For each age group the method estimates how many years of life expectancy at birth are added (or subtracted) due to the change in age-specific mortality rates. In addition, the overall contribution of mortality change for a particular age group may be partitioned by cause of death, under the assumption that the contribution of each cause of death to life expectancy change for an age group is proportional to the contribution of each cause of death to the change in the age-specific central mortality rate.[10] Thus, the contribution of a change in a specific cause of death is a function of both the absolute change in age-specific mortality and the change in the distribution of that cause over time relative to other causes of death. Causes of death that are relatively less frequent in 2001 than in 1990 (i.e., declines in cause-specific mortality rates) will make positive contributions to the overall change in life expectancy at birth, while causes that are relatively more frequent in 2001 (i.e., increases in cause-specific mortality rates) will make negative contributions.

We obtained age and cause-specific mortality data for this study from the National Center for Health Statistics' Compressed Mortality Files for 1990 and 2001.[11,12] We calculated the proportion of deaths due to 28 causes (International Classification of Disease (ICD) codes are available from the authors) in 1990 and 2001 for 13 age groups by gender and race. The

combination of age groups and causes of death led to 364 potential age-cause contributions for each race-gender group. For the sake of clarity, the results section only presents detailed causes of death that contributed at least one month (+/- 0.083 years) to the change in life expectancy from 1990 to 2001, and within a given cause of death only age groups that contributed at least one week (+/- 0.019 years) to the change in life expectancy. Results of the complete decompositions by age and detailed cause-of-death are available from the authors.

In order to account for changes in cause-specific mortality due to the Tenth revision of the International Classification of Diseases (ICD-10), which took effect in 1999, comparability ratios were applied for each cause of death. The comparability ratio was derived by classifying all deaths in 1996 by both ICD-9 and ICD-10 criteria, and was calculated as the number of deaths for a given cause classified by ICD-10 divided by the number of deaths classified by ICD-9.[13] Comparability ratios were calculated for each race-gender group and applied to mortality data in 1990 to obtain comparability-modified mortality rates. Results were essentially similar when using non-comparability-modified data.

Results

Overall United States Life Expectancy

Life expectancy at birth in the United States improved from 75.35 years in 1990 to 77.17 years in 2001, an increase of 1.82 years. The leftmost panel of Table 1 ("Decomposition by Age") shows that all age groups made positive contributions to the change (i.e., age-specific mortality rates declined for all age groups), but a majority of the increase in life expectancy was due to improving mortality among individuals 55 and over (1.07 years or 55.8% of the total).

Notable improvements in mortality among infants and 25-34 year-olds increased life expectancy by 0.19 years (10.4%) and 0.16 years (8.8%), respectively.

The middle panel of Table 1 ("Decomposition by Cause") shows that a large part of the improvement in life expectancy can be attributed to improvements in mortality from cardiovascular-related diseases (CVD)—namely ischemic heart disease (IHD, 44.7%), ischemic stroke (5.8%), and other CVD (5.2%). Improvements in mortality from human immunodeficienty virus (HIV) also made a relatively important contribution to improved life expectancy (7.8%). The simultaneous age and cause decomposition (rightmost panel of Table 1) shows similar results as the decomposition by cause, but highlights the importance of particular age-cause groups as contributors to life expectancy change. Most of the positive contribution for "Unspecified causes" was due to improving infant mortality, improvements in HIV and homicide mortality occurred primarily among those 25-44, while the positive contribution of lung cancer occurred primarily among those 45-64. Overall, most of the negative contributions to the change in life expectancy were due to small unfavorable mortality changes for a number of other causes of death (e.g., Alzheimer's disease, chronic lower respiratory diseases, diabetes) and the residual causes of death not specified in this analysis. As a consequence of aggregating a large number of relatively rare causes of death, the residual category of unspecified causes was relatively large for both positive and negative contributions to life expectancy change.

Black Female Life Expectancy

Life expectancy at birth among black females increased 1.86 years, from 73.60 years in 1990 to 75.47 years in 2001 (Table 2). The age decomposition (data not shown) indicates that the increase in life expectancy for black females was spread across age groups, including infants (15.7%), 25-34 year-olds (10.4%), 55-74 year-olds (34.5%), and those 85 and older (12.2%).

Improvements in mortality from IHD made a substantial contribution to improving life expectancy among black females—28.1% of the total positive contribution. Unspecified causes of death (primarily infant mortality), other cancers, ischemic stroke, and homicide also contributed at least 5% to the total positive contribution. Life expectancy among black females would have increased more were it not for unfavorable mortality changes in Alzheimer's disease, HIV, lung cancer, COPD, diabetes, and septicemia (Table 2, "Other specific causes").

Black Male Life Expectancy

Black male life expectancy improved more than any other race-gender group studied here, increasing from 64.52 years in 1990 to 68.56 in 2001—a gain of 4.03 years (Table 2). With respect to age (data not shown), improving mortality among 25-54 year-olds contributed 1.92 years (47.6%) to the overall increase in life expectancy, while 55-74 year-olds contributed another 1.09 years (26.9%).

Homicide mortality made the largest positive contribution to life expectancy over this period (15.5%), followed by IHD (15.1%), unspecified causes (12.7%), HIV (10.3%) and lung cancer (7.6%). As might be expected, the cause-specific improvements were concentrated among particular age groups. The positive contribution of homicide was primarily among 15-34 year-olds, IHD among men 45 and older, and HIV among 25-44 year-olds. Most of the contribution of unspecified causes of death was due to improvements in infant mortality (0.27 years of the total 0.55 years). Life expectancy among black males would have increased even more were it not for unfavorable mortality changes in diabetes (chiefly among 65-74 year-olds) and other specific causes of death such as HIV (among 45-64 year-olds) and hypertensive heart disease.

White Female Life Expectancy

White females experienced a net improvement of 0.81 years of life expectancy during this period, the smallest gain among the four race-gender groups (Table 3). In terms of age (data not shown), the majority of improvement in life expectancy was due to favorable mortality changes among those 55 and over—0.55 years or 64.7% of the total positive contribution—with an additional 0.12 years (14.5%) added from improved infant mortality. White females were exceptional as the only race-gender group for which an entire age group (35-44 year-olds) made a net negative life expectancy contribution. The increase in the all-cause mortality rate among white females 35-44 between 1990 and 2001—from 119.1 per 100,000 to 130.2—decreased white female life expectancy at birth by 0.04 years.

The cause of death contributing most to improved life expectancy among white females was IHD, which accounted for 48% of the total positive contribution. Net improvements in mortality from breast cancer (among those 35-74) and ischemic stroke (among those 65 and over) also made notable positive contributions. Worsening mortality from unspecified causes, chronic lower respiratory disease (CLRD), Alzheimer's disease, and lung cancer together contributed 75.4% of the total negative contribution, most of which occurred among women 55 and over. The net negative contribution for white females 35-44 appears to be due to increased mortality from unspecified causes and unintentional injuries other than motor vehicle accidents, which were large enough to offset a notable improvement in breast cancer mortality for this age group.

White Male Life Expectancy

Life expectancy at birth for white males increased 2.31 years, from 72.72 in 1990 to 75.03 in 2001 (Table 3). The majority (59.8%) of the increase in life expectancy was due to mortality improvement among 55-84 year-olds (data not shown). Other age groups that made relatively

large contributions to the total change included infants (0.18 years or 7.6%) and 25-34 year-olds (0.21 years or 8.9%).

Improvements in mortality from IHD made the largest positive contribution to improved life expectancy among white males (1.1 years), primarily due to improvements among those 55-74 years old. Improvements in IHD mortality among these three age groups alone accounted for 29.1% of the total positive contribution to life expectancy change. Other causes of death that made strong positive contributions to improved life expectancy were HIV (10.9%), lung cancer (7.5%), and unspecified causes of death, primarily among infants (6.4%). White male life expectancy would have increased more were it not for small unfavorable mortality changes for some specific causes of death (e.g., diabetes, non-motor vehicle injuries, Alzheimer's disease) and residual causes of death not specified in this analysis.

Table 1. Rank Ordering of the Contribution of Age Groups and Leading Causes of Death to the Change in Life Expectancy at Birth in the United States, 1990-2001

Decomposition by Age		**Decomposition by Cause of Death**		**Decomposition by Age and Cause of Death**		
Age Group	Change in LE, years (%)	Cause of Death*	Change in LE, years (%)	Cause of Death*	Major Age Groups†	Change in LE, years (%)
All ages	1.818	All causes	1.818	All causes		1.818
		Positive Contribution		**Positive Contribution**		
55-64	0.368 (20.2)	Ischemic heart disease	0.965 (44.7)	Ischemic heart disease	45+	0.966 (39.3)
65-74	0.357 (19.6)	HIV	0.168 (7.8)	Unspecified causes	0-1	0.189 (7.7)
75-84	0.208 (11.4)	Ischemic stroke	0.125 (5.8)	HIV	25-44	0.168 (6.8)
0-1	0.188 (10.4)	Other cancer	0.115 (5.3)	Ischemic stroke	65+	0.125 (5.1)
25-34	0.161 (8.8)	Other CVD	0.112 (5.2)	Lung cancer	45-64	0.121 (4.9)
85+	0.136 (7.5)	Homicide	0.106 (4.9)	Other CVD	55-74	0.115 (4.7)
45-54	0.127 (7.0)	Lung cancer	0.097 (4.9)	Other cancer	55-74	0.115 (4.7)
35-44	0.088 (4.9)	Other specific causes	0.473 (21.9)	Homicide	15-19, 25-34	0.107 (4.3)
15-19	0.062 (3.4)	Total positive	2.161 (100.0)	Other specific causes	None	0.554 (22.5)
20-24	0.041 (2.2)			Total positive		2.460 (100.0)
1-4	0.039 (2.1)					
5-9	0.024 (1.3)					
10-14	0.020 (1.1)					
Total	1.818 (100.0)					
		Negative Contribution		**Negative Contribution**		
		Unspecified causes	-0.086 (24.9)	Unspecified causes	35+	-0.275 (42.7)
		Other specific causes	-0.257 (75.1)	Other specific causes	None	-0.368 (57.3)
		Total negative	-0.343 (100.0)	Total negative		-0.642 (100.0)

Abbreviations: CVD, cardiovascular diseases; HIV, human immunodeficiency virus; LE, Life expectancy.
*Includes causes of death that contributed at least one month (+/- 0.083 years) to the change in life expectancy.
†Includes age groups that contributed at least one week (+/- 0.019 years) to the change in life expectancy.

Table 2. Rank Ordering of the Leading Causes of Death and Major Age Groups Contributing to the Change in Life Expectancy at Birth among Black Males and Females in the United States, 1990-2001

Black Females			Black Males		
Cause of Death*	Major Age Groups†	Change in LE, years (%)	Cause of Death*	Major Age Groups†	Change in LE, years (%)
All causes		1.864	All causes		4.033
Positive Contribution			**Positive Contribution**		
Ischemic heart disease	45+	0.701 (28.1)	Homicide	15-64	0.673 (15.5)
Unspecified causes	0-1, 25-34	0.301 (12.1)	Ischemic heart disease	35+	0.656 (15.1)
Other cancer	45-74, 85+	0.235 (9.4)	Unspecified causes	0-4, 15-64	0.550 (12.7)
Ischemic stroke	55+	0.193 (7.7)	HIV	20-44	0.447 (10.3)
Homicide	15-34	0.180 (7.2)	Lung cancer	35-74	0.328 (7.6)
Other CVD	55-74, 85+	0.123 (4.9)	Other cancer	35-74	0.305 (7.0)
Liver cirrhosis	35-64	0.110 (4.4)	Other injuries	1-4, 25-44	0.208 (4.8)
Other specific causes	None	0.654 (26.2)	Other CVD	35-74	0.189 (4.4)
Total positive		2.497 (100.0)	Liver cirrhosis	35-64	0.161 (3.7)
			Ischemic stroke	45-84	0.146 (3.4)
			Motor vehicle accidents	25-34	0.111 (2.6)
			Prostate cancer	55-84	0.098 (2.3)
			Influenza/pneumonia	35-45	0.087 (2.0)
			Other specific causes	None	0.381 (8.8)
			Total positive		4.339 (100.0)
Negative Contribution			**Negative Contribution**		
Unspecified causes	45-54, 75-84	-0.101 (15.9)	Diabetes	65-84	-0.084 (27.3)
Alzheimer's disease	75+	-0.087 (13.8)	Other specific causes	None	-0.223 (72.7)
Other specific causes	None	-0.446 (70.3)	Total negative		-0.307 (100.0)
Total negative		-0.633 (100.0)			

Abbreviations: COPD, chronic obstructive pulmonary disease; CVD, cardiovascular diseases; HIV, human immunodeficiency virus; LE, Life expectancy.
*Includes causes of death that contributed at least one month (+/- 0.083 years) to the change in life expectancy.
†Includes age groups that contributed at least one week (+/- 0.019 years) to the change in life expectancy.

Table 3. Rank Ordering of the Leading Causes of Death and Major Age Groups Contributing to the Change in Life Expectancy at Birth among White Males and Females in the United States, 1990-2001

White Females			White Males		
Cause of Death*	Major Age Groups†	Change in LE, years (%)	Cause of Death*	Major Age Groups†	Change in LE, years (%)
All causes		0.806	All causes		2.312
Positive Contribution			**Positive Contribution**		
Ischemic heart disease	45+	0.890 (48.0)	Ischemic heart disease	35+	1.070 (37.7)
Breast cancer	35-74	0.171 (9.2)	HIV	25-54	0.310 (10.9)
Ischemic stroke	65+	0.127 (6.8)	Lung cancer	45-74	0.213 (7.5)
Unspecified causes	0-1	0.126 (6.8)	Unspecified causes	0-1	0.182 (6.4)
Other cancer	55-74	0.105 (5.7)	Other CVD	55-84	0.159 (5.6)
Other specific causes	None	0.434 (23.4)	Motor vehicle accidents	15-34	0.108 (3.8)
Total positive		1.852 (100.0)	Ischemic stroke	65+	0.108 (3.8)
			Prostate cancer	65-84	0.097 (3.4)
			Other cancer	55-74	0.086 (3.0)
			Homicide	25-34	0.085 (3.0)
			Other specific causes	None	0.419 (14.8)
			Total positive		2.837 (100.0)
Negative Contribution			**Negative Contribution**		
Unspecified causes	25+	-0.404 (38.6)	Unspecified causes	25+	-0.249 (47.4)
CLRD	65+	-0.154 (14.7)	Other specific causes	None	-0.276 (52.6)
Alzheimer's disease	75+	-0.132 (12.6)	Total negative		-0.525 (100.0)
Lung cancer	65-74	-0.100 (9.5)			
Other specific causes	None	-0.256 (24.5)			
Total negative		-1.046 (100.0)			

Abbreviations: CLRD, chronic lower respiratory diseases; CVD, cardiovascular diseases; HIV, human immunodeficiency virus; LE, Life expectancy.
*Includes causes of death that contributed at least one month (+/- 0.083 years) to the change in life expectancy.
†Includes age groups that contributed at least one week (+/- 0.019 years) to the change in life expectancy.

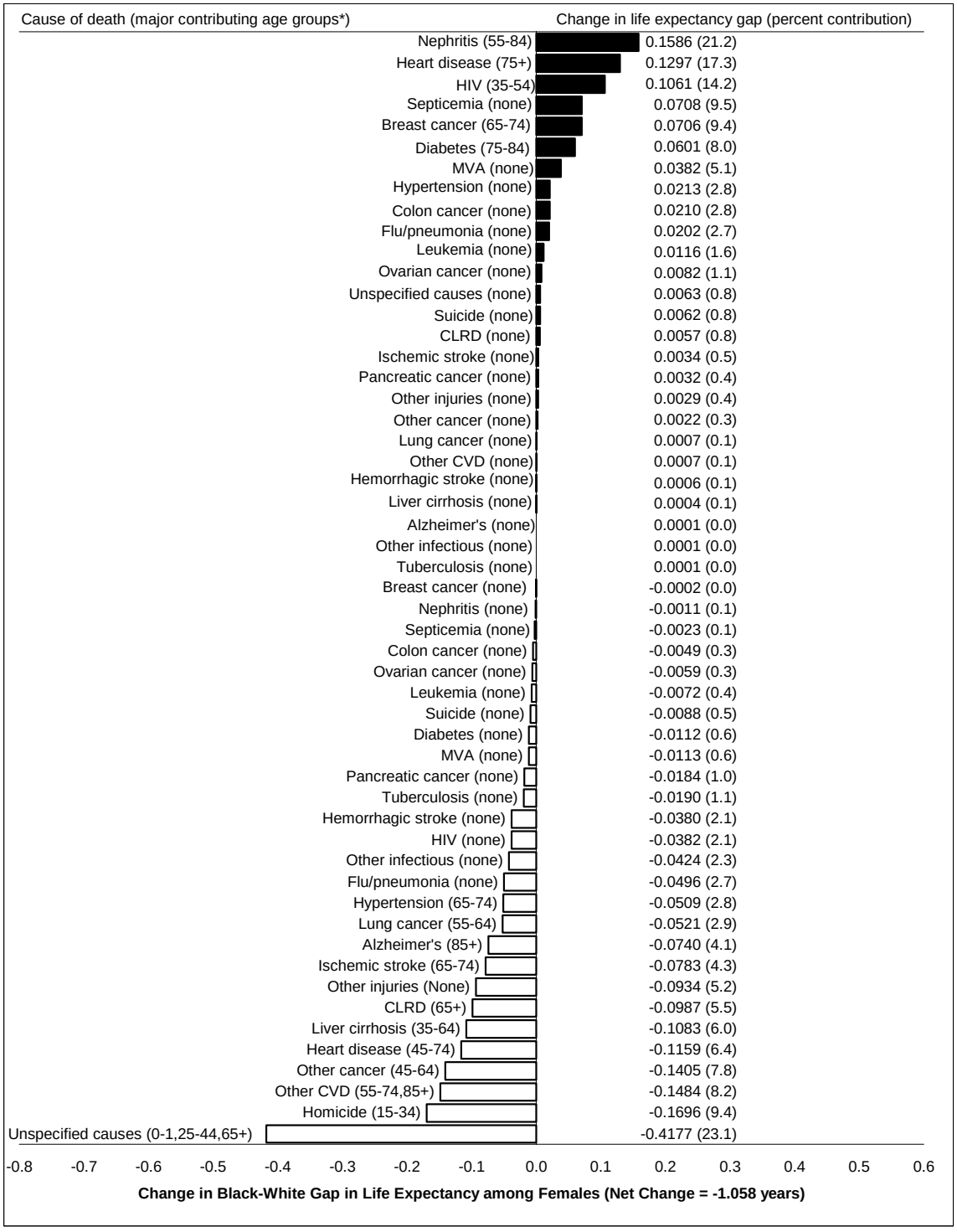
Changes in the Black-White Gap in Life Expectancy

From 1990 to 2001 the gap in life expectancy at birth between blacks and white declined for both males and females. For females the gap was 5.81 years in 1990 and 4.75 years in 2001, a decline of 1.06 years. With respect to age (data not shown), most of the disparity change was due to relative mortality improvement among black females 25-64 (51.0%), with important contributions by infants (16.2%) and those 85 and over (12.9%). Figure 1 presents the decomposition of this change by age and detailed cause of death, and shows that most of the positive contribution to the decline in the black-white gap was due to improvements in homicide (9.4%), other CVD (8.2%), other cancers (7.8%), IHD (6.4%), liver cirrhosis (6.0%), and small improvements for a number of other specific causes. Relative improvement in infant mortality from unspecified causes among black females made the largest contribution of any age-cause group, accounting for 8.8% of the change in the female black-white gap. The upper part of Figure 1 also shows that the black-white life expectancy gap among females would have declined even more were it not for unfavorable changes in black mortality (relative to whites) from nephritis, IHD among those 75 and over, and HIV primarily among those 35-54.

The decline in the black-white life expectancy gap was larger among males, declining from 8.19 years in 1990 to 6.47 years in 2001. In contrast to the more varied age decomposition pattern for females, the disparity decline among males was almost entirely due to relative mortality improvement among blacks ages 25-54 (71.9%), with a smaller contribution of overall infant mortality (6.0%) and mortality changes at older ages actually contributing to widening the gap (data not shown). Figure 2 shows that relative improvement among black males in mortality from homicide (22.2%) and unspecified causes (19.9%) accounted for a large portion of the decline in the black-white life expectancy gap among males. The contribution of homicide

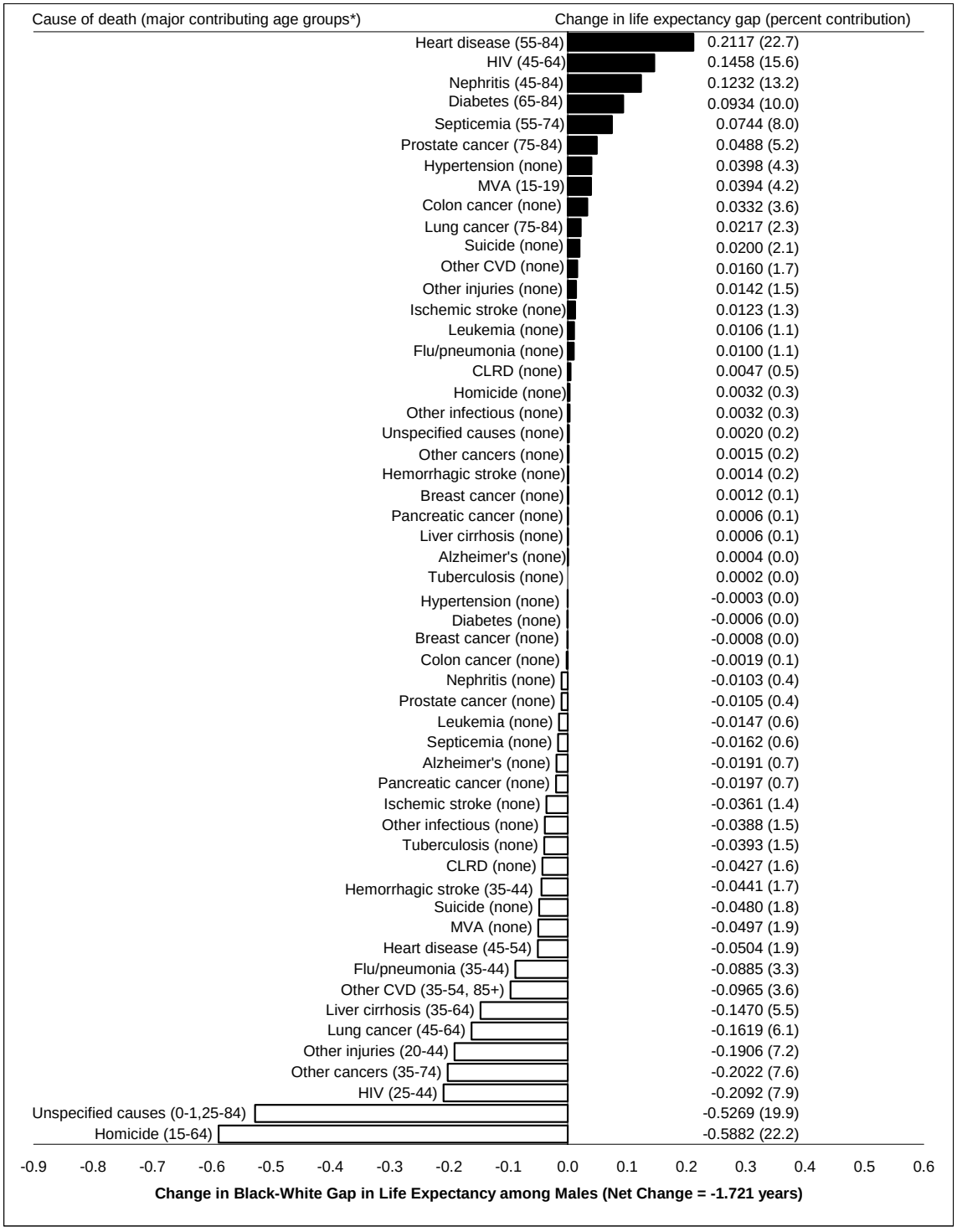
to the declining gap among males 15-34 was more dramatic, nearly 48% (results not shown). Other causes that made relatively important contributions to the decline in disparity were HIV among those 25-44 (7.9%), and, primarily among those 35-64, other cancers (7.6%), non-motor vehicle injuries (7.2%), lung cancer (6.1%), and liver cirrhosis (5.5%). Changes in other causes of death kept the black-white gap among males from narrowing even further than it did, primarily IHD, HIV, nephritis, diabetes, and septicemia. It is worth noting that among men HIV contributed to both narrowing (ages 25-44) and widening (ages 45-64) the black-white life expectancy gap, indicating diverging age-specific mortality changes.

Figure 1. Leading Causes of Death and Major Age Groups Contributing to the Change in the Black-White Gap in Life Expectancy at Birth among Females in the United States, 1990-2001



Abbreviations: CLRD, chronic lower respiratory diseases; CVD, cardiovascular diseases; HIV, human immunodeficiency virus; MVA, motor vehicle accidents. *Includes age groups that contributed at least one week to the change in life expectancy.

Figure 2**.** Leading Causes of Death and Major Age Groups Contributing to the Change in the Black-White Gap in Life Expectancy at Birth among Males in the United States, 1990-2001



Abbreviations: CLRD, chronic lower respiratory diseases; CVD, cardiovascular diseases; HIV, human immunodeficiency virus; MVA, motor vehicle accidents. *Includes age groups that contributed at least one week to the change in life expectancy.

Discussion

Life expectancy in the United States improved from 1990 to 2001 primarily due to continuing declines in cardiovascular mortality among those ages 45 and over, as well as improvements in infant mortality, HIV and homicide mortality among the young, lung cancer among those 45-64, and small improvements for a number of other causes of death. Improvement in mortality from ischemic heart disease was by far the most important cause contributing to increased U.S. life expectancy, accounting for nearly 40% of the total net gain.

There has been some concern that the steady declines in ischemic heart disease mortality observed since the late 1960s had begun to slow during the 1990s.[14,15] Our analysis suggests that such a slowing, if actual, was nevertheless transitory, as IHD mortality improvement since the late 1990s has been impressive. Based on the age-adjusted IHD mortality rates published in Health, United States 2004,[16] the average annual mortality decline was 4.4% from 1999-2002, a relative decline as steep or steeper than those seen during previous decades.[17] This begs the question of why the pace of mortality decline for IHD recently increased. A recent large international study[18] and studies of large U.S. cohorts[19,20] showed that at least 75% of IHD mortality at the population level can be attributed to its major risk factors—smoking, dyslipidemia, hypertension, and diabetes history. It would follow that recent improvement in IHD mortality decline should be linked to population changes in major risk factors.

However, recent trends in the major risk factors for IHD are diverse. Rates of current smoking continue to decline, but the rate of decline has slowed from 0.6 percentage points per year during 1970 to 1990 to 0.3 per year during 1995-2002.[21] In a reversal of substantial declines since the 1970s,[22] recent trends indicate that the prevalence of hypertension has increased since the early 1990s among adults[23] and is increasing among children and

adolescents.[24] Similar secular increases are observed in the prevalence of diabetes, overweight, and obsesity.[25,26] However, mean levels of total cholesterol and the proportion of individuals with high serum cholesterol have continued to decline,[27] albeit at a somewhat slower pace than in previous decades. Given the unfavorable recent trends in some risk factors, it is thus somewhat surprising that IHD mortality declines appear to have steepened. But this may in part reflect the fact that for IHD population changes in risk factors are unlikely to produce immediate changes in mortality, so risk factor changes further back in time may be more important than more recent trends.

Recent evidence from population-based cohorts have demonstrated faster declines in IHD mortality for in-hosptial compared to out-of-hospital events,[28,29] nonsudden compared to sudden coronary death,[30,31] and among those with preexisting IHD compared to those without.[29,30] Such differences are often taken to reflect changes in secondary rather than primary prevention, though this distinction is somewhat artificial. One important development during the mid-1990s was the publication of the major statin trials, which demonstrated their effectiveness at decreasing serum cholesterol and reducing IHD and stroke events among those with and without pre-existing IHD.[32-35] The use of cholesterol-lowering medications has substantially increased during the past decade,[36] 90% of which are now estimated to consist of statins.[37] Nationally, between 1995-96 and 2001-02 the number of physician office and outpatient visits with cholesterol-lowering drugs prescribed, provided, or continued increased approximately 200% among those 45 and over. This was the largest absolute and relative increase among all 20 major drug classes.[16] In addition, the age-adjusted percentage of participants in the National Health and Nutrition Survey taking drugs for hypercholesterolemia more than doubled between 1988-94 and 1999-2000, from 3.1% to 7.9%.[27] While the overall prevalence would appear too low to

account One might thus speculate about a potential role for the increased use of statins in the recent declines IHD mortality, but there is as yet no quantitative evidence for such a population-wide effect. Despite the apparent increase in the rate of IHD mortality decline, further declines in IHD mortality will depend on reversing the unfavorable recent trends for some risk factors through primary and secondary prevention.

In addition to the overall gains in life expectancy, the life expectancy gap between blacks and whites decreased substantially for both males and females, reversing the widening of the gap observed during the late 1980s.[7] Among single causes of death, relative declines in homicide made the largest contribution to declining black-white disparities over this period, particularly among males where it accounted for almost one quarter of the net decline. Homicide thus continues to be an important determinant of black-white mortality patterns among the young, as it has during the latter part of the 20th century. Elo and Drevenstedt found that among those ages 15-39 differing homicide trends accounted for 60% of the increase in black-white all-cause mortality inequality from 1960-67, 23% of the decrease in inequality from 1968-83, and 54% of the increase from 1984-95.[38] In Kochanek and colleagues' analysis of the change in the black-white life expectancy gap from 1984-89,[7] homicide accounted for 17.2% of the overall widening life expectancy gap among males. However, the trends observed during these earlier periods now seem to have reversed, though homicide rates are still much higher in blacks than whites.

Despite the similar contribution of homicide to the rises and falls in black-white mortality inequality since the 1960s, the causes of changes in homicide mortality likely differ by age and across time.[39] For example, while there is strong evidence that the rise in homicide during the mid-1980s was linked to the spread of crack cocaine markets and primarily affected black males 15-34,[40,41] the rise homicide during the 1960s also affected black males 35-64 and was more

likely due to the initial spread of handguns and political unrest in the postwar period.[39,42] The decline of the crack market is also thought to play a role in the homicide declines of the 1990s, while other potential explanations include increased incarceration rates, increases in the size of police forces, and demographic changes.[43,44] Most controversially, some have also argued that the increased availability of abortion in the 1970s played a role in the crime declines of the 1990s.[45] However, a more recent test of this hypothesis failed to uncover race-specific effects that would explain why the decline was so much steeper for blacks than for whites.[46] While additional research is needed to clarify the factors responsible for the decline in homicide, any such explanations will need to account for why the decrease was greater among blacks than whites.

Mortality from HIV, particularly among those ages 25-44, disproportionately contributed to the widening of the black-white life expectancy gap in the late 1980s,[7] but also to the subsequent narrowing observed in this analysis. Inspection of the age-specific mortality patterns revealed that HIV mortality was rising rapidly (particularly among blacks) until the mid-1990s, declined dramatically from 1995 to 1998, and has since stabilized. There is strong evidence that the recent steep mortality declines from HIV were due to the widespread and rapid introduction of highly-active antiretroviral therapy (HAART).[47,48] However, relative mortality declines have been larger for whites than blacks, though not large enough to overwhelm the contribution to improved life expectancy of the absolute mortality declines among younger blacks. As there is evidence that the effectiveness of HAART is similar in blacks and whites,[49,50] differences in the relative mortality declines most likely result from differential access to and quality of treatment among blacks.[51-53] There is evidence to suggest that the US AIDS epidemic is now becoming more concentrated among older ages and minorities,[54] which is consistent with the results of the

current analysis showing HIV mortality trends at older ages contributing to widening the black-white life expectancy gap. Two recently published studies found compelling evidence for the cost-effectiveness of expanded systematic voluntary screening for HIV in health care settings.[55,56] This is particularly important given recent reports of increases in sexually transmitted infections[57] which, paradoxically, may be related to the dramatic effects of HAART.[58]

Somewhat surprisingly, aside from lung cancer, major cancers did not figure significantly in the declines in the black-white life expectancy gap, while breast and prostate cancer trends made moderate contributions to widening the gap. However, among both males and females changes in working-age mortality from relatively less common cancers were an important contributor to declining black-white inequalities. A closer inspection of the mortality trends for specific cancers among the "other cancers" group in this analysis revealed sharp declines in black-white differences occurring for a number of cancers that are thought to be causally related to smoking.[59] Among males and females 45-64 this is apparent for cancers of the oral cavity and pharynx, esophagus, stomach, larynx, kidney, and bladder, and among females for uterine and cervical cancer. Other cancers for which there is as yet no sufficient evidence for a causal relation to smoking[59] (e.g., liver and brain) do not appear to show similar declines in black-white inequality.[48] The extent to which such changes may be linked to race-specific cohort changes in smoking initiation[60] or cessation[61] is worth further exploration.

This analysis primarily focused on causes of death that contributed to the change in the black-white life expectancy gap, but it is important to note that the extent to which a particular cause of death may contribute to the change in the black-white gap may not correspond to its contribution to the overall gap at a particular time. For example, changes in mortality from

unintentional injuries other than motor vehicle accidents ("Other injuries" in Figure 2) made the 5th largest contribution to the decline in disparity among males (7.2%), but it only ranks 14th in terms of the causes of death contributing to the black-white gap in life expectancy in 2001 (results not shown). Other causes of death may make important contributions to narrowing the gap and yet remain those most in need of further declines. For example, while most of the progress in reducing the gap between black and white males has been due to improvements in mortality from infant mortality, homicide, and HIV, in 2001 they are still among the leading causes contributing to the black-white life expectancy gap among males, and are causes of death that should be targeted to further decrease the black-white life expectancy gap. More troubling is the fact that ischemic heart disease remains a primary reason for black-white differences in life expectancy (14.2% of the gap among women in 2001, 9.5% among men) and continues to contribute to widening black-white inequalities, particularly for men 55 and over and women 75 and over. Thus, slower declines in IHD among blacks, which have been observed for some years,[17,62] appear to be continuing, and reducing the risk factors for ischemic heart disease among blacks should remain a public health priority.

While the decomposition method used in this analysis enables the partitioning of life expectancy changes by age and cause of death, it has some limitations. The use of a single underlying cause of death simplifies the fact that many deaths that are due to multiple causes. This may be particularly true for deaths at older ages when comorbid conditions are more prevalent. To the extent that some causes of death are more likely to be mentioned as contributing causes (e.g., diabetes, influenza and pneumonia),[63] this analysis may underestimate the impact of common multiple-cause conditions of life expectancy changes. However, given

that a large part of the observed changes in the black-white gap are due to mortality at younger ages, the use of multiple-cause data appers unlikely to alter the basic findings of this analysis.

Assessments of change between two time periods may potentially mask important trends occurring within the time period analyzed. The year 1990 was used as a beginning for this analysis because a previous decomposition of the racial life expectancy gap[7] ended in 1989, the last year during which life expectancy for blacks actually decreased. However, despite a reversal of the negative trend in life expectancy for blacks, the black-white life expectancy gap remained constant among females and continued to increase among males after 1989, reaching 8.5 years by 1993, a gap last seen in 1944. Thus, the current analysis actually understates the rapidity with which the black-white life expectancy gap has recently declined, especially among males. Had 1993 been used as the starting point a decline in the black-white life expectancy gap among males of 2.2 years rather than 1.8 years would have been observed, and declines in homicide and HIV, which continued to increase disproportionately among blacks during the early 1990s, would have made an even larger contributon to this decline.

Finally, the use of 2001 as the ending year of this analysis includes deaths due to the September 11, 2001 terrorist attacks, which were categorized as "unspecified causes" rather than as homicides to minimize the effect of the change from ICD-9 to ICD-10. These deaths occurred disproportionately among young whites (particularly males) and are likely to have increased the contribution of "unspecified causes" to the decline in black-white inequality.

Summary

After widening during the late 1980s, the life expectancy gap between blacks and whites declined during the next decade, and the most recent data indicates this trend is continuing.[64] This indicates important progress toward one of the major public health goals of eliminating

health inequalities,[65] and is evidence that health inequalities are not immutable and are linked to changes in the broader social environment. But the observation that the black-white gap in overall life expectancy declined gives no indication of how or why it declined; life expectancy is a summary measure of diverse age and cause-specific mortality components, so there are multiple pathways to life expectancy change. Additonally, just knowing the gap declined does not provide any guidance for formulating interventions that might further reduce a gap that is still too wide. The decomposition analysis used here allows for a more effective understanding of life expectancy change. By breaking life expectancy change down into its age, gender, race, and cause-specific components, decomposition analysis facilitates a more specific focus on the most important dimensions of mortality change and provides a tool for generating hypotheses about the mechanisms driving such changes. Because it focuses on net changes, it also sheds light on unfavorable changes that may be masked overall. For example, despite the narrowing of the life expectancy gap, black-white changes in ischemic heart disease continue to prevent the gap from narrowing further and should remain a public health priority. The goal of efficiently and effectively eliminating black-white mortality inequalities will require knowing where prevention and treament efforts should be targeted. As a first step, the analysis presented here suggests a useful method for generating the knowledge required for making progress toward that goal.

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