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Research Article

Why the racial gap in life expectancy is declining in the United States

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Abstract

BACKGROUND
Blacks have lower life expectancy than whites in the United States. That disparity could be due to racial differences in the causes of death, with blacks being more likely to die of causes that affect the young, or it could be due to differences in the average ages of blacks and whites who die of the same cause. Prior studies fail to distinguish these two possibilities.

OBJECTIVE
In this study we determine how much of the 2000–10 reduction in the racial gap in life expectancy resulted from narrowing differences in the cause-specific mean age at death for blacks and whites, as opposed to changing cause-specific probabilities for blacks and whites.

METHOD
We introduce a method for separating the difference-in-probabilities and difference-in-age components of group disparities in life expectancy.

RESULTS
Based on the new method, we find that 60% of the decline in the racial gap in life expectancy from 2000 to 2010 was attributable to reduction in the age component,

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largely because of declining differences in the age at which blacks and whites die of chronic diseases.

CONCLUSION

Our findings shed light on the sources of the declining racial gap in life expectancy in the United States, and help to identify where advances need to be made to achieve the goal of eliminating racial disparities in life expectancy.

1. Introduction

The gap in life expectancy between blacks and whites in the United States has been declining. In the first ten years of this century, gains in life expectancy for blacks outstripped gains for whites by nearly two years, reducing the racial gap from 5.7 years in 2000 down to 3.8 years in 2010, an historic low. This decline is attributable largely to the relative mortality improvement of blacks versus whites with respect to heart disease, HIV/AIDS, accidental poisoning (mostly drug-related), and cancer (Harper, Rushani and Kaufman 2012). The life expectancy gap that persists is due primarily to remaining black-white mortality differences in heart disease, homicide (especially for men), cancer, diabetes, HIV/AIDS, and perinatal conditions (Harper et al. 2012; Kochanek, Arias and Anderson 2013; Wong et al. 2002).

What we do not know is how racial differences in heart disease, cancer, etc., contribute to the gap in life expectancy. Cancer, for example, might contribute to the gap because overall death rates for cancer are higher for blacks than for whites, or because black cancer victims tend to be younger than white cancer victims, or both. Although prior studies identify the causes associated with the gap in life expectancy, they do not indicate which mechanism – the difference-in-age component (or simply *age component*) or the difference-in-probability component (or *incidence component*) – is driving the difference. To fashion effective policy for reducing the racial gap, however, we need to know whether the gap is located in black-white differences in cause-specific incidence (indicating differences in the risk of succumbing to given causes) or in differences in the cause-specific mean age at death (indicating differences in the age-profile of risk-exposure).

This paper makes two contributions. The first is methodological: we propose a method for separating the age component from the incidence component. Second, we use that method to uncover the cause-specific dynamics driving the 1.9-year decline in the racial gap in life expectancy from 2000 to 2010 in the United States. We present our results in two parts. In the first part we use period multi-decrement life tables for blacks and whites to determine the distribution and average age at death of blacks and whites

across 19 principal causes of death. We use that information to decompose the racial gap in life expectancy, separating the part due to black-white differences in cause-specific incidence from the part due to black-white differences in cause-specific average age at death. By comparing those results for 2000 to 2010 we uncover why the racial gap in life expectancy declined by one-third over that period. Then, in the second phase of our analysis, we use multi-decrement sex-specific life tables to determine the separate contributions of men and women, by cause, to the narrowing of the overall gap. We conclude the paper by summarizing our major findings and discussing their implications.

2. Cause decomposition as a tool for studying life expectancy

Demographers have a longstanding interest in how specific causes of death contribute to life expectancy. As Beltrán-Sánchez, Preston, and Canudas-Romo (2008) note in their illuminating synthesis, demographers have developed two basic methods for estimating this contribution. One method estimates the contribution of a cause by recalculating the life table with no deaths assigned to that cause (often called "cause-deleted" life tables) (Newman 1987). A second method uses decomposition formulas, derived by Arriaga (1984), Andreev (1982), Pollard (1982), and Pressat (1985) and later extended by Andreev et al. (2002) and Vaupel and Canudas-Romo (2002, 2003), to investigate how specific causes of death contribute to life expectancy.

Neither approach suffices, however, when – as in the United States – the stated goal is to eliminate disparities in life expectancy between different segments of the population, such as blacks and whites (US Department of Health and Human Services 2000). To fashion policies to eliminate black-white disparity, we want to know whether the disparity is located in age differences among blacks and whites who succumb to the same cause, or to differences in the causes of death for blacks and whites (that is, in their incidence). Although cause-deletion and decomposition methods can tell us the portion of the black-white gap in life expectancy that is associated with (say) cancer, neither method can distinguish whether cancer matters because blacks are more likely to die of cancer or because black victims of cancer on average are younger than white victims of cancer. Likewise, in accounting for the narrowing of the black-white gap in life expectancy, standard methods do not tell us whether the contribution of a particular cause is due to convergence in incidence, to convergence in the mean age at death, or to a combination of the two.

Recent studies of the black-white life expectancy gap illustrate these points. Using the Arriaga (1984) method, a data brief from the U.S. National Center for Health Statistics concludes that in 2010 "Life expectancy for the black population was lower

(3.8 years) than life expectancy for the white population because of higher death rates due to heart disease, cancer, homicide, diabetes, and perinatal conditions" (Kochanek et al. 2013: 3). Yet it is possible that the contributions of heart disease, cancer, and so on are not due – or at least not entirely due – to higher death rates for blacks (the incidence component), but to the lower cause-specific mean age at death for blacks, since in the Arriaga method the incidence component is not separated from the age component.

Similarly, Harper et al. (2007) use the Arriaga method to decompose the black-white gap in life expectancy for 1983, 1993, and 2003. They decompose by cause of death and, in a separate decomposition, by age group. The 2003 results from the cause decomposition are similar to those from Kochanek et al. (2013) for 2010 – heart disease, cancer, homicide (especially for men), diabetes (for women), and perinatal death are all major contributors to the life expectancy gap – but again these findings do not distinguish age-at-death differences from differences in incidence. From the Harper et al. (2007) age decomposition we learn that age group 45-64 accounted for about two-fifths of the gap for both men and women in 2003, and that age group 15-44 accounted for another one-fifth of the gap. From these results we conclude that blacks are more likely than whites to die between the ages of 14 and 65, but we do not know why: is it because of differences in cause-specific incidence (for example, blacks are more likely to die of homicide, which predominately affects the young) or because of blacks' earlier deaths from the same causes as whites?

We now describe a method for assessing, for each cause of death, the relative contributions of the age and incidence components to the life expectancy gap. The method uses a logic that is akin to the tradition in demography that separates the contribution of population composition from the contribution of differences in rates (Das Gupta 1993; Kitagawa 1955, 1964).

3. Analytic methods

3.1 Equations for identifying sources of the longevity gap at a point in time

Our method capitalizes on the observation that life expectancy at birth is the sum of probability-weighted cause-specific life expectancies, so life expectancy can be derived using age-specific mortality rates broken down by cause of death (Beltrán-Sánchez, Preston, and Canudas-Romo 2008). We use mortality rates from separate multi-decrement life tables (Keyfitz 1985) for whites and blacks to calculate, for each of 19 causes of death, both the probability (p) of death (from that cause) and the mean age at death ($\bar{x}$). Then from p and $\bar{x}$ we calculate the contribution of each of the causes to the black-white longevity gap in 2000 and 2010, respectively, separating the portion of the

gap attributable to differences in the mean age at death ($\bar{x}_{cW} - \bar{x}_{cB}$) from the portion attributable to differences in the probability of dying of each cause ($p_{cW} - p_{cB}$), where the subscript *c* indexes cause of death, *B* denotes non-Hispanic blacks, and *W* denotes non-Hispanic whites.[6]

Because overall life expectancy is the probability-weighted sum of cause-specific life expectancies (life table mean age at death for that cause), the gap in life expectancy for blacks and whites, $e_W - e_B$, is:

$$e_W - e_B = \sum_c p_{cW}\, \bar{x}_{cW} - \sum_c p_{cB}\, \bar{x}_{cB}, \tag{1}$$

where $\sum_c p_{cW} = \sum_c p_{cB} = 1$. It follows that $\sum_c p_{cW} k = \sum_c p_{cB}\, k = k$, where k is a constant. In particular, let k be $\bar{e} = \frac{e_W + e_B}{2}$, the simple mean of the life expectancies for blacks and whites. Then, to determine the contribution of specific causes to the longevity gap, we insert $\sum_c p_{cB}\, \bar{e} - \sum_c p_{cW}\, \bar{e}$, a term that equals zero, into (1) and rearrange terms:

$$\begin{aligned} e_W - e_B &= \sum_c p_{cW}\, \bar{x}_{cW} - \sum_c p_{cW}\, \bar{e} - \sum_c p_{cB}\, \bar{x}_{cB} + \sum_c p_{cB}\, \bar{e} \\ &= \sum_c p_{cW}\, (\bar{x}_{cW} - \bar{e}) - \sum_c p_{cB}\, (\bar{x}_{cB} - \bar{e}). \end{aligned} \tag{2}$$

From (2) it follows that the contribution of each cause to the black-white longevity gap is:

$$contribution\ of\ c\text{th}\ cause = p_{cW}(\bar{x}_{cW} - \bar{e}) - p_{cB}(\bar{x}_{cB} - \bar{e}). \tag{3}$$

The first term, $p_{cw}(\bar{x}_{cw} - \bar{e})$, is positive when white victims of cause *c* on average outlive the overall simple average life expectancy ($\bar{e}$) and negative when white victims tend to die before the overall average. Similarly, the second term in (3) is positive when black victims of cause *c* on average outlive the overall average and negative when they generally die before the overall life expectancy. If equation (3) – the first term minus the second term – is positive, then cause *c* increases the racial gap in life expectancy. If the two terms are the same, cause *c* does not contribute to the gap. If the difference is negative, then cause *c* narrows the gap.

From (3) we can see that the effects of mean age at death and probability of death are intertwined because they weight each other. To separate out the age component for

[6] Hispanics generally are treated as a separate group in studies of U.S. mortality because of what is known as the "Hispanic paradox" (Franzini et al. 2001).

a specific cause, we remove the incidence component in (3) by setting p_{cW} and p_{cB} at their mean value ($\bar{p}_c$) for blacks and whites. Thus the age component is:

$$age\ component\ for\ cause\ c = \bar{p}_c(\bar{x}_{cW} - \bar{x}_{cB}) \quad (4)$$

Age differences matter more when they occur in common causes of death than when they occur in rare causes of death, so the equation for the age component weights the age difference $\bar{x}_{cW} - \bar{x}_{cB}$ by the probability of dying of the cause.

We separate out the incidence component in the same way, that is, we remove the influence of differences in age at death in (3) by setting $\bar{x}_{cW} - \bar{e}$ and $\bar{x}_{cB} - \bar{e}$ at their mean, $[(\bar{x}_{cW} - \bar{e}) + (\bar{x}_{cB} - \bar{e})]/2$. Rewriting $[(\bar{x}_{cW} - \bar{e}) + (\bar{x}_{cB} - \bar{e})]/2$ as $\bar{x}_c - \bar{e}$, where $\bar{x}_c = (\bar{x}_{cW} + \bar{x}_{cB})/2$, and inserting this value into (3), the incidence component is:

$$incidence\ component\ for\ cause\ c\ = (\bar{x}_c - \bar{e})(p_{cW} - p_{cB}) \quad (5)$$

Differences in the probability of dying affect the longevity gap more for causes that strike predominately at the tails as opposed to the middle of the age-at-death distribution, so the equation for the incidence component weights the difference in the probabilities by the average distance from the overall mean age.[7]

Summing over all causes in (4) reveals how much of the longevity gap is accounted for by differences in the ages at which blacks and whites succumb to specific causes of death:

$$all\text{-}cause\ age\ component = \sum_c \bar{p}_c(\bar{x}_{cW} - \bar{x}_{cB}) \quad (6)$$

Likewise, summing over all causes in (5) reveals how much of the gap is accounted for by differences in cause-specific incidence for blacks and whites:

[7] This weighting is consistent with conventional practice in demography (Preston, Heuveline, and Guillot 2001: 28–29), and it works well in this study. Because age differences make a greater contribution to the racial gap when they occur in common causes of death than when they occur in rare causes of death, dramatically changing death rates for a given cause (e.g., an epidemic) could boost the contribution of age differences to the racial gap in the absence of any change in the age differences of black versus white victims. Strictly, then, the term "age component" refers to the contribution of age differences to a longevity gap rather than to the size of the age difference itself. Similarly, by "incidence component" we refer to the contribution of differences in cause-specific incidence to the longevity gap rather than to the size of the incidence differences themselves.

$$all\text{-}cause\ incidence\ component = \sum_c (\bar{x}_c - \bar{e})(p_{cW} - p_{cB}) \quad (7)$$

The all-cause age and incidence components sum to the overall longevity gap.

3.2 Equations for identifying sources of change in the longevity gap

Change in the longevity gap is due to change in the age and incidence components. To determine how much of the narrowing of the longevity gap from 2000 to 2010 was due to declines in each of those components, we add subscripts 0 and 1 to indicate the years 2000 and 2010, respectively. Then, from equation (4), change in the age component over time for cause *c* is:

$$\begin{aligned} &change\ in\ the\ age\ component\ for\ cause\ c \\ &= \bar{p}_{c1}(\bar{x}_{cW1} - \bar{x}_{cB1}) - \bar{p}_{c0}(\bar{x}_{cW0} - \bar{x}_{cB0}) \end{aligned} \quad (8)$$

Similarly, change in the incidence component for the c^{th} cause is, from (5):

$$\begin{aligned} &change\ in\ the\ incidence\ component\ for\ cause\ c \\ &= (\bar{x}_{c1} - \bar{e}_1)(p_{cW1} - p_{cB1}) - (\bar{x}_{c0} - \bar{e}_0)(p_{cW0} - p_{cB0}) \end{aligned} \quad (9)$$

To determine how much of the narrowing of the longevity gap is due to change in the contributions of the age components for all causes, we sum over causes in (8). Likewise, to determine how much of the narrowing of the longevity gap is due to change in the contributions of the incidence components for all causes of death, we sum over causes in (9).

4. Data

We draw upon the 2000 and 2010 Multiple Cause of Death Mortality files (National Center for Health Statistics 2007, 2012), which also include census counts of the U.S. population by sex, age, and race. The NCHS mortality data contain information on all deaths occurring among the U.S. resident population during the calendar year, and include information on the underlying cause of death [8] as well as on selected

[8] The underlying cause of death is the specific injury or disease that initiated the train of events that led to death or the circumstances of an event that caused a fatal injury (World Health Organization 2008).

demographic characteristics obtained from death certificates. The 2000 and 2010 population counts are from the U.S. Census Bureau's April 1st bridged modified race census counts (National Center for Health Statistics 2007, 2012). We stratify the mortality data by race and cause of death in order to create the cause-age-specific multiple decrement life table data. After removing Hispanics, we create separate multiple decrement life tables for blacks and whites.[9] By using multiple decrement life tables we eliminate the effect of black-white differences in population size and age composition, and we obtain the life table number of deaths and mean ages at death by cause of death (Carey 1989).[10]

Causes of death were coded using the 10th revision of the International Classification of Diseases (ICD-10). This classification contains over 10,000 individual codes. In order to reduce the number of causes under investigation while still considering the entire landscape of disease, we regrouped causes into broad categories that are sensible from an epidemiological point of view. We began with the coding scheme used to identify the 15 leading causes of death in the National Vital Statistics Report for 2006 (Heron et al. 2009). We then adapted this scheme based on data constraints and on the findings of prior analyses (Harper et al. 2007; Nau and Firebaugh 2012) to include causes that are of particular relevance to the racial gap in life expectancy. Our final cause categorization consists of (a) the 10 leading causes of death in the United States in 2006, with the exception that we substituted traffic accidents and accidental poisoning for the more general category "accidents"; (b) 5 new cause categories – homicide, suicide, infectious diseases other than influenza, septicemia, and HIV/AIDS (which are listed separately), and external causes other than homicide, suicide, traffic accidents, and accidental poisoning (which are also listed separately); (c) two infant and child conditions (perinatal deaths and congenital anomalies), and (d) a residual category of causes not elsewhere classified ("n.e.c."). The residual category assembles all ill-defined causes of death as well as all causes that had too few deaths to stand on their own or that are not of primary interest from a population health point of view. We included accidental poisoning, HIV/AIDS, suicide, and homicide as separate causes because they are highly associated with race in the United States (Appendix Table 2 reports the racial difference in probabilities for each cause). Appendix Table 1 describes our cause grouping in more detail.

[9] Recall that we are comparing the life expectancy of non-Hispanic blacks and non-Hispanic whites. We exclude Hispanics because of their distinctive life expectancy, which differs from that of blacks and of whites. We also exclude the very small percentage of the sample (fewer than 1%) that failed to specify whether or not they were of Hispanic origin.

[10] For each cause, we use age 90 as the midpoint for the open-ended age category of 85 and older because using 90 as the midpoint yields the closest approximation of the overall mean age at death.

5. Decomposition of the racial gap by cause in 2000 and in 2010

To set the stage for the more detailed analyses to follow, Figure 1 presents the all-cause age and incidence components for the overall racial gap in life expectancy in the United States in 2000 and 2010. The age component accounted for about 4.3 years of the racial gap in 2000 and about 3.2 years of the gap in 2010 (Figure 1), so roughly three-fifths of the 1.9-year decline in the racial gap from 2000–2010 was due to narrowing of the difference in the average age at death for blacks and whites who died of the same cause. Convergence in cause-specific incidence for blacks and whites accounted for the remaining two-fifths of the decline in the racial gap.

Figure 1: All-cause age and incidence components for the black-white gap in life expectancy: 2000, 2010, and change from 2000 to 2010

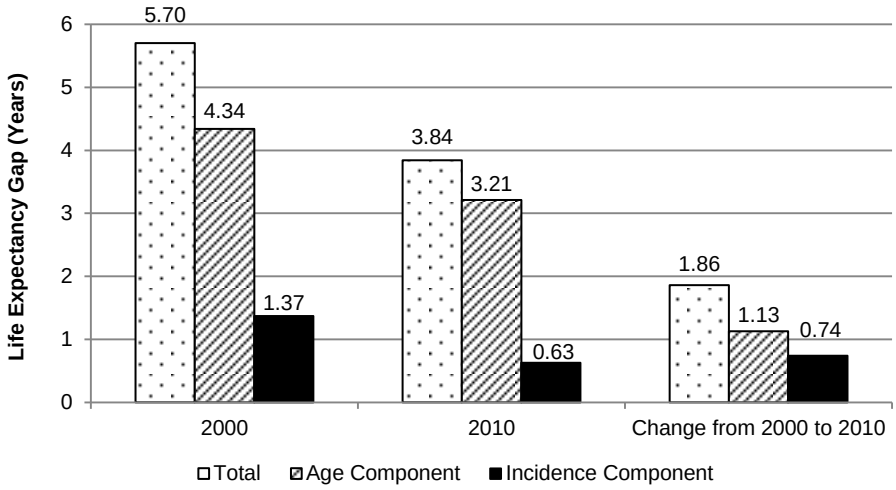


Table 1 presents the contribution of each of the 19 causes to the racial gap in life expectancy in 2000 and in 2010. Several findings stand out. First, in terms of years of life expectancy, virtually all causes of death contributed less to the racial gap in 2010 than in 2000. The greater life expectancy gains for blacks, then, were spread out across the 19 causes of death. (The one exception is trivial: communicable diseases other than HIV/AIDS, septicemia, and influenza contributed 0.01 more years – or four more days of life – to the gap in 2010.)

Table 1: Cause-specific components of the black-white gap in life expectancy in the United States in 2000 and 2010, and total percentages of deaths due to each cause

ICD-10 cause of death		Gap in 2000		Gap in 2010		% of total deaths due to this cause[a]
		Years of gap	% of gap	Years of gap	% of gap	
Chronic diseases						
1	Heart diseases	1.49	26.2	1.02	26.5	28.2
2	Cancers	0.53	9.4	0.41	10.7	22.6
3	Cerebrovascular diseases	0.39	6.8	0.25	6.6	6.7
4	Chronic lower respiratory diseases	0.24	4.1	0.18	4.8	4.6
5	Alzheimer's	0.16	2.8	0.16	4.1	2.8
6	Diabetes	0.08	1.5	0.07	1.7	3.4
7	Nephritis	0.08	1.4	0.06	1.6	2.4
	Totals for chronic diseases	**2.97**	**52.1**	**2.15**	**56.0**	**70.6**
Communicable diseases						
8	Influenza and pneumonia	0.18	3.2	0.08	2.1	2.4
9	Septicemia	0.04	0.6	0.03	0.8	1.7
10	HIV/AIDS	0.40	7.1	0.20	5.3	0.7
11	Other infectious diseases	0.05	0.8	0.06	1.5	0.7
	Totals for communicable diseases	**0.67**	**11.7**	**0.37**	**9.7**	**5.5**
External causes						
12	Homicide	0.53	9.3	0.50	13.1	0.8
13	Suicide	-0.10	-1.8	-0.18	-4.8	0.8
14	Traffic accident	0.00	0.1	-0.01	-0.2	1.1
15	Accidental poisoning	0.01	0.2	-0.19	-4.9	0.7
16	Other external causes	0.14	2.5	0.13	3.4	1.8
	Totals for external causes	**0.59**	**10.3**	**0.25**	**6.5**	**5.0**
Infant and child conditions						
17	Perinatal deaths	0.41	7.2	0.29	7.7	0.5
18	Congenital anomalies	0.05	0.8	0.04	1.0	0.3
	Totals for infant and child conditions	**0.46**	**8.0**	**0.33**	**8.7**	**0.8**
Aggregated minor causes						
19	NEC - Not elsewhere classified	**1.02**	**17.9**	**0.73**	**19.0**	**18.1**
Grand Total		**5.70**	**100.0**	**3.84**	**100.0**	**100.0**

[a] Simple average of the percentage of life table deaths for blacks and whites in 2000 and 2010.

The second notable finding is that the racial gap in life expectancy has multiple sources in both periods, with no single cause of death responsible for the majority of the gap. The largest contributor is heart disease, accounting for 1.5 years of the 5.7-year gap in 2000 and one year of the 3.8-year gap in 2010, or roughly one-fourth of the racial gap each year. These results replicate the findings of Kochanek et al. (2013) and can be attributed to the fact that heart disease accounts for more than one-fourth of all deaths in the United States.

Third, some relatively rare causes of death contribute more to the racial gap than do some common causes of death. Homicide, for example, accounted for more of the racial gap in life expectancy in 2010 than did cancer, even though blacks are about 17 times more likely, and whites about 120 times more likely, to be cancer victims than homicide victims. In both years perinatal deaths accounted for more of the racial gap than did cerebrovascular disease, even though cerebrovascular disease is a much more common cause of death. Homicide, HIV/AIDS, and perinatal deaths are major over-contributors to the racial gap. Suicide and accidental poisoning – also causes of death that disproportionately strike the young – stand out because they narrow, not widen, the racial gap in life expectancy.[11] Of the leading causes of death in the United States, heart disease and cerebrovascular disease contribute to the racial gap roughly in proportion to their share of deaths, whereas cancer contributes proportionately less (23% of deaths, 10% of the racial gap).

Prior studies end here, with the decomposition of group differences in life expectancy by cause of death. We extend those results by using our decomposition method to determine, for each cause of death, how much of the contribution to the overall gap in life expectancy reflects group differences in probabilities and how much reflects group differences in age at death. We report those results in the next section.

6. Decomposition of the racial gap by cause and by age at death vs. incidence

6.1 Cause-specific probability of death and average age at death for blacks and whites in 2000 and 2010

In this section we show that the shorter life expectancy of blacks is the result of (a) racial differences in incidence for select causes and (b) an across-the-board (or nearly so) lower average age at death for blacks than for whites who die of the same cause. To determine how much of the racial gap is due to (a) and how much is due to (b), we

[11] Suicide and accidental poisoning both increased more for whites than for blacks from 2000 to 2010 (Appendix Table 2). This contributed to the narrowing of the life expectancy gap, as we see subsequently.

decompose the racial gaps in 2000 and in 2010 simultaneously by cause of death and by age at death versus incidence.

Results from the multidecrement life tables for 2000 and 2010 (Appendix Table 2) reveal that whites tend to outlive blacks in the United States for virtually every cause of death (accidental poisoning is the only notable exception, with white victims being 3.6 years younger on average in 2000 and 6.6 years younger in 2010). The magnitude of the black-white age difference varies across causes, from a few months in the case of Alzheimer's and HIV/AIDS to over 10 years for homicides in 2010. For most causes, however, the age difference is narrowing over time, particularly for chronic diseases. For heart disease victims the black-white disparity in average age at death declined by 0.7 years, from 4.7 years in 2000 to 4.0 years in 2010. For cancer victims the black-white disparity in age at death declined by 0.6 years over the course of the decade (Appendix Table 2).

Changes in the incidence component are more complex. Blacks are about eight times more likely than whites to die of HIV/AIDS and about seven times more likely to be homicide victims. Whites, on the other hand, are about three times more likely to commit suicide and also were more likely, in 2010, to die of accidental poisoning (largely drug-related: Warner et al. 2011). As noted earlier, the effect of these differences on the racial gap in life expectancy depends on whether the cause-specific mean age at death is higher or lower than the overall mean age at death. Because causes of death are competing against each other, the black-white longevity gap would narrow, for example, if a higher proportion of blacks than whites died of Alzheimer's. Homicide has the opposite effect, with a higher proportion of black victims enlarging the gap.

Table 2 reports the decomposition results, based on equations (4) and (5) above. Because black victims tend to be younger than white victims for nearly every cause of death, age components are responsible for most of the longevity gap, accounting for about 76% of the gap in 2000 and 84% of the gap in 2010 (Table 2, last row). This means that if blacks and whites died of the same causes at the same rates – so that they varied only with respect to cause-specific average age at death – three-fourths of the racial gap in life expectancy would persist in 2000 and five-sixths of the gap would persist in 2010.

Table 2: Cause-specific age and incidence components of the black-white gap in life expectancy in the United States in 2000 and 2010, and change from 2000-2010 (in years)

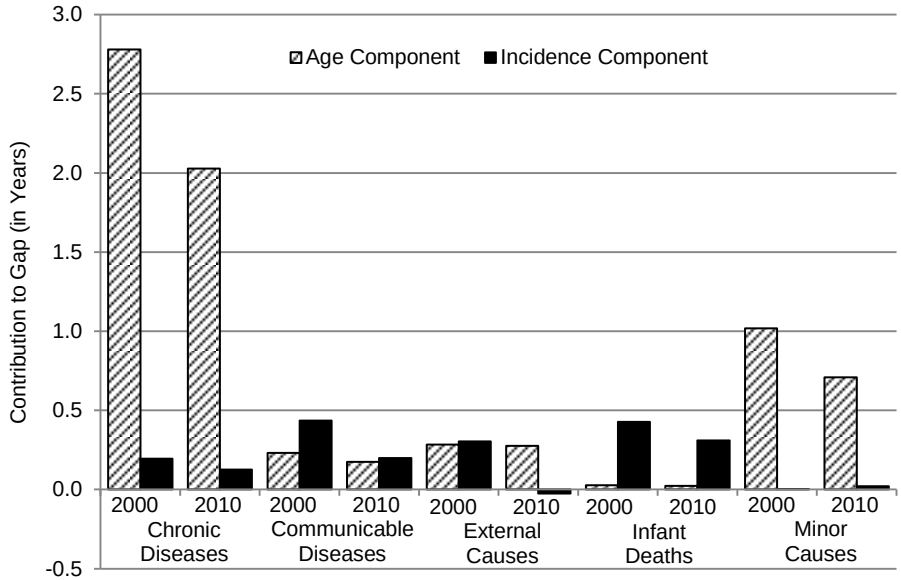
		2000			2010			Change from 2000-2010		
ICD-10 cause of death		**Total**	**Age**	**Incidence**	**Total**	**Age**	**Incidence**	**Total**	**Age**	**Incidence**
Chronic diseases										
1	Heart diseases	1.49	1.44	0.06	1.02	1.03	-0.01	-0.48	-0.40	-0.07
2	Cancers	0.53	0.54	-0.01	0.41	0.41	0.00	-0.12	-0.13	0.01
3	Cerebrovascular diseases	0.39	0.40	-0.01	0.25	0.28	-0.03	-0.14	-0.12	-0.02
4	Chronic lower respiratory diseases	0.24	0.18	0.06	0.18	0.12	0.06	-0.05	-0.06	0.01
5	Alzheimer's	0.16	0.01	0.14	0.16	0.01	0.15	0.00	0.00	0.01
6	Diabetes	0.08	0.10	-0.01	0.07	0.06	0.00	-0.02	-0.03	0.01
7	Nephritis	0.08	0.12	-0.04	0.06	0.11	-0.05	-0.02	-0.01	-0.01
	Totals for chronic diseases	**2.97**	**2.78**	**0.20**	**2.15**	**2.03**	**0.13**	**-0.82**	**-0.75**	**-0.07**
Communicable diseases										
8	Influenza and pneumonia	0.18	0.14	0.04	0.08	0.07	0.01	-0.10	-0.07	-0.03
9	Septicemia	0.04	0.06	-0.03	0.03	0.04	-0.01	0.00	-0.02	0.02
10	HIV/AIDS	0.40	-0.01	0.41	0.20	0.00	0.20	-0.20	0.01	-0.21
11	Other infectious diseases	0.05	0.03	0.01	0.06	0.06	0.00	0.01	0.03	-0.01
	Totals for communicable diseases	**0.67**	**0.23**	**0.44**	**0.37**	**0.18**	**0.20**	**-0.29**	**-0.06**	**-0.24**
External causes										
12	Homicide	0.53	0.06	0.48	0.50	0.06	0.44	-0.03	0.00	-0.03
13	Suicide	-0.10	0.05	-0.16	-0.18	0.06	-0.24	-0.08	0.01	-0.09
14	Traffic accident	0.00	0.03	-0.03	-0.01	0.03	-0.04	-0.01	0.00	-0.01
15	Accidental poisoning	0.01	-0.02	0.03	-0.19	-0.05	-0.14	-0.20	-0.04	-0.16
16	Other external causes	0.14	0.16	-0.02	0.13	0.18	-0.05	-0.01	0.03	-0.03
	Totals for external causes	**0.59**	**0.28**	**0.30**	**0.25**	**0.28**	**-0.02**	**-0.34**	**-0.01**	**-0.33**
Infant and child conditions										
17	Perinatal deaths	0.41	0.00	0.41	0.29	0.00	0.29	-0.12	0.00	-0.12
18	Congenital anomalies	0.05	0.03	0.02	0.04	0.02	0.02	-0.01	-0.01	0.00
	Totals for infant and child conditions	**0.46**	**0.03**	**0.43**	**0.33**	**0.02**	**0.31**	**-0.12**	**-0.01**	**-0.12**
Aggregated minor causes										
19	NEC - Not elsewhere classified	1.02	1.02	0.00	0.73	0.71	0.02	-0.29	-0.31	0.02
Grand Total		**5.70**	**4.34**	**1.37**	**3.84**	**3.21**	**0.63**	**-1.86**	**-1.13**	**-0.73**

Although age at death accounted for the majority of the racial gap in life expectancy in both years, there are important differences when we consider causes by our five general cause categories (Figure 2). The age component dominates for chronic diseases and for minor causes not elsewhere classified: for these causes, then, the racial gap is due almost entirely to differences in the ages at which blacks and whites succumb to the same cause. In the case of infant and childhood conditions and communicable diseases, by contrast, the incidence component is more important. That result is expected for infant deaths because there cannot be much difference in the ages at which black and white infants die, so virtually all of the difference must be due to

differences in incidence. In the case of communicable diseases the incidence component is larger than the age component because of HIV/AIDS. For all other communicable diseases the age component is larger than the incidence component (Table 2).

The general category of external causes of death – homicides, suicides, traffic fatalities, and accidental poisoning – provides important insights about mortality differences for blacks and whites. For external causes collectively the age and incidence components are roughly the same in 2000, each responsible for about 0.3 years of the racial gap in life expectancy. By 2010 the incidence component was negligible (slightly below zero). The disappearance of the incidence component for external causes largely reflects the fact that the black-white difference in homicide rates was, in 2010, offset by the near-tripling of accidental poisoning among whites, as well as by an increase in white suicides, with little or no increase in the incidence of either cause for blacks over the same period. Indeed, in 2010 whites were almost as likely to be victims of accidental poisoning as blacks were to be homicide victims (Appendix Table 2).

Figure 2: Components of the racial gap in life expectancy, by cause category, in 2000 and 2010



Source: Table 2

To summarize, the age component is responsible for most of the racial gap in life expectancy in both 2000 and 2010. In particular, age-at-death differences account for the major part of the longevity gap associated with chronic diseases, which includes the most common causes of death in the United States. The incidence component contributes significantly to the longevity gap in the case of infant deaths and communicable diseases in 2000 and 2010 and external causes in 2000. Based on these findings about the factors that produced the black-white gaps in life expectancy in 2000 and 2010, we can now turn to the question of what accounts for the narrowing of the longevity gap from 2000 to 2010.

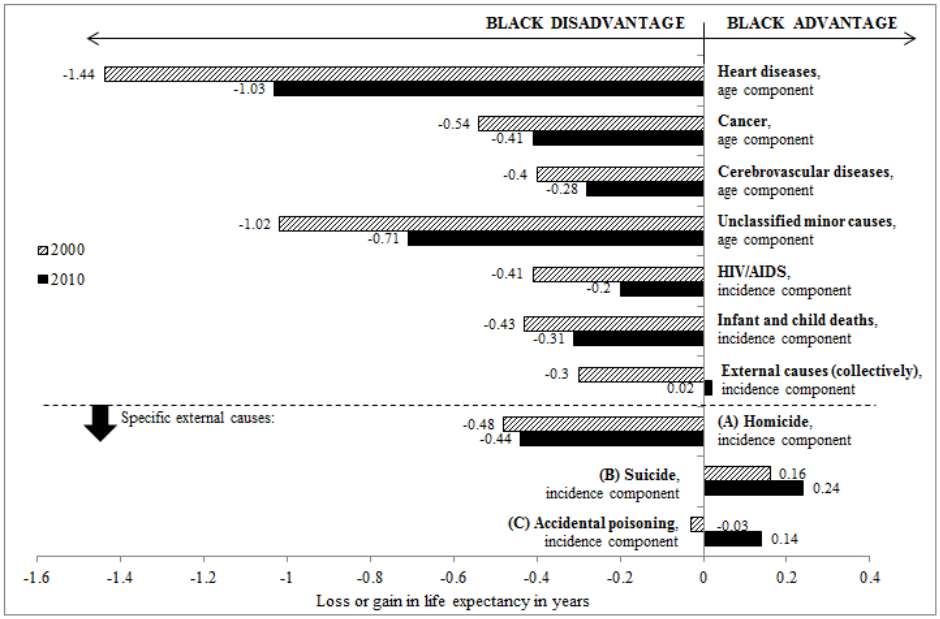
6.2 Narrowing of the longevity gap: Reductions in the age component versus reductions in the incidence component

Because differences in life expectancy between groups are produced by differences in the cause-specific incidence of deaths as well as by differences in the cause-specific mean ages at death, convergence in life expectancy comes from reductions in either or both factors. The final three columns in Table 2 display the results of our decomposition of the change in the life expectancy gap from 2000 to 2010. Change in the age component of heart disease was the largest single contributor to the narrowing of the gap, accounting for 22% (0.40/1.86) of the narrowing. Although other, less frequent, causes of death – such as chronic lower respiratory diseases, diabetes, and influenza – exhibited greater reductions in the black-white difference in average age at death or age gap (Appendix Table 2), change in the age component was significantly greater for heart disease because heart disease is a much more common cause of death in the United States. Change in the age component of minor causes (not elsewhere classified) accounted for another 17% of the reduction in the black-white longevity gap. Change in the incidence component of HIV/AIDS was the next largest single contributor, accounting for 11% of the narrowing of the gap. Change in the incidence component of accidental poisoning and infant deaths reduced the gap by 9% and 6%, respectively. Collectively, then, these five factors accounted for about two-thirds of the narrowing of the gap.

Another way to summarize these findings is to note that the 2000-10 narrowing of the racial gap came from three broad sources (Figure 3). The first source is declining differences in the age at death among blacks and whites who die of the three leading chronic diseases or of minor causes not elsewhere classified. These are the first four causes depicted in Figure 3. The second source is declining racial differences in the probability of dying as infants or children, or as victims of HIV/AIDS, the next two causes depicted in Figure 3. The third source is change in the probability of dying of

external causes. External causes exhibit a unique pattern in that they narrowed the longevity gap from 2000 to 2010 mainly by reducing the life expectancy for whites (bottom of Figure 3). Deaths due to external causes increased from 4.5% to 5.7% for whites while declining from 5.1% to 4.7% for blacks (Appendix Table 2). In this instance, then, the racial disparity in longevity was compressed by rising mortality for distinctively youthful causes of death among whites – certainly not the method for eliminating population health disparities envisioned in *Healthy People 2010* (US Department of Health and Human Services 2000).

Figure 3: Key cause-specific components accounting for the narrowing of the racial gap in life expectancy, 2000-2010



Source: Table 2. Note that the bars depict the age component for some causes (e.g. heart disease, cancer) and the incidence component for other causes (e.g. HIV/AIDS).

7. Decomposition of the racial gap by cause and by sex

In this section we determine the cause-sex-specific contributions to the difference in life expectancies of blacks and whites. This is not to be confused with the cause-specific contributions to the differences in the life expectancy of black men versus white men, and of black women versus white women, the subject of prior investigations. Harper et al. (2012) find, for example, that heart disease accounts for 22% of the 5.4-year gap in life expectancy between black men and white men in 2008, and for 29% of the 3.7-year gap in life expectancy between black women and white women the same year. Although findings such as these add to our understanding of why white men tend to outlive black men, and why white women tend to outlive black women, they do not tell us how much of the overall black-white gap in life expectancy is attributable to cause-specific racial differences in men's mortality, and how much is attributable to cause-specific racial differences in women's mortality – our objective here. By determining the sex-specific underpinnings of the overall gap we identify more precisely where advances are needed to achieve the goal of eliminating racial disparities in life expectancy.

7.1 Equations for identifying sources of the longevity gap at a point in time, by sex

To decompose the black-white longevity gap simultaneously by cause and by sex, let superscripts distinguish men and women, as follows:

$$p_{cW}^{men} = \frac{number\ of\ white\ men\ who\ die\ of\ cause\ c}{total\ white\ deaths}$$

$$p_{cW}^{women} = \frac{number\ of\ white\ women\ who\ die\ of\ cause\ c}{total\ white\ deaths}$$

$$p_{cB}^{men} = \frac{number\ of\ black\ men\ who\ die\ of\ cause\ c}{total\ black\ deaths}$$

$$p_{cB}^{women} = \frac{number\ of\ black\ women\ who\ die\ of\ cause\ c}{total\ black\ deaths}$$

Note that $p_{cW}^{men} + p_{cW}^{women} = p_{cW}$, the probability that a white person will die of cause *c*, and similarly for blacks.

The cause-specific means, $\bar{x}_{cW}$ and $\bar{x}_{cB}$, can also be written as the sums of the cause-specific means for women and men weighted by the relative probabilities of women and men succumbing to cause *c*:

$$for\ whites\colon\ \bar{x}_{cW} = (p_{cW}^{men}/p_{cW})\bar{x}_{cW}^{men} + (p_{cW}^{women}/p_{cW})\bar{x}_{cW}^{women}$$
$$for\ blacks\colon\ \bar{x}_{cB} = (p_{cB}^{men}/p_{cB})\bar{x}_{cB}^{men} + (p_{cB}^{women}/p_{cB})\bar{x}_{cB}^{women}$$

(Note that p_{cW}^{men}/p_{cW} and p_{cW}^{women}/p_{cW} sum to 1.0 for each cause, and similarly for blacks.) From these two equations it follows that the contribution of cause *c* to the longevity gap, $p_{cW}(\bar{x}_{cW} - \bar{e}) - p_{cB}(\bar{x}_{cB} - \bar{e})$, can be partitioned into the part attributable to differences between black women and white women and the part attributable to differences between black men and white men as follows:

$$for\ cause\ c, the\ longevity\ gap\ due\ to\ differences\ between\ black\ women\ and\ white\ women = p_{cW}^{women}(\bar{x}_{cW}^{women} - \bar{e}) - p_{cB}^{women}(\bar{x}_{cB}^{women} - \bar{e})$$

$$for\ cause\ c, the\ longevity\ gap\ due\ to\ differences\ between\ black\ men\ and\ white\ men = p_{cW}^{men}(\bar{x}_{cW}^{men} - \bar{e}) - p_{cB}^{men}(\bar{x}_{cB}^{men} - \bar{e})$$

To determine the sex-specific contributions to the black-white longevity gap, we sum over the causes:

$$part\ of\ longevity\ gap\ due\ to\ racial\ differences\ in\ women's\ mortality = \sum_c p_{cW}^{women}(\bar{x}_{cW}^{women} - \bar{e}) - p_{cB}^{women}(\bar{x}_{cB}^{women} - \bar{e}) \quad (10)$$

$$part\ of\ longevity\ gap\ due\ to\ racial\ differences\ in\ men's\ mortality = \sum_c p_{cW}^{men}(\bar{x}_{cW}^{men} - \bar{e}) - p_{cB}^{men}(\bar{x}_{cB}^{men} - \bar{e}) \quad (11)$$

Equation (10) can be interpreted as the extent to which the overall black-white longevity gap would change if we removed racial differences in women's mortality without changing the average age at death, $\bar{e}$ (similarly for men, in equation (11)). This is not to be confused with the extent to which the gap would change if we removed women, since removing women will change $\bar{e}$ unless men and women happen to have the same life expectancy. The practical implication is that the results given here for men and women (examining the overall racial gap) are not the same as those one would obtain by analyzing men and women separately (examining two racial gaps).

7.2 Men's and women's contributions to the racial gap, by cause: 2000, 2010, and change from 2000 to 2010

In this section we use the equations presented above to determine how much of the racial gap in life expectancy was generated by differences in cause-specific mortality for black men versus white men, and for black women versus white women, respectively, in 2000 and in 2010. We then use those results to separate the part of the decline in the gap due to change in the racial differences in mortality patterns for men from the part due to change in the racial differences in mortality patterns for women.

In 2000 the racial gap was generated equally by racial differences in mortality among men and among women (2.9 and 2.8 years, respectively: Table 3). However, this overall similarity masks notable cause-specific differences in the contributions of men and women. The contribution of homicide, for instance, is primarily due to racial differences in mortality for men, not women, with the difference for men accounting for 0.45 years of the total homicide contribution of 0.53 years in 2000 (the pattern is similar in 2010). This means that if black men and white men died of homicide following the same pattern, all else being constant, the overall racial gap would decrease by 0.45 years, whereas equalizing the pattern for women would reduce the gap only by 0.08 years. The pattern of mortality for HIV/AIDS is likewise more strongly racially shaped for men than for women.

Although men and women contributed equally to the racial gap in life expectancy in 2000, women made a greater contribution to the narrowing of the gap in the ensuing decade. By 2010 women's contribution to the gap had declined by 1.1 years, while men's contribution to the gap had declined by only 0.75 years. Changing racial patterns in chronic diseases for women reduced the overall racial gap by 0.65 years, thus accounting for over one-third of the 1.9-year overall reduction, while changing racial patterns in chronic diseases for men contributed less than 0.2 years to the reduction. Racial differences in heart disease mortality in particular declined much more for women than for men. As a result, change in the racial disparity in heart disease among women accounted for about 0.4 years of the 1.9-year decline in the longevity gap, whereas men's heart-disease contribution was only about 0.1 years.

The reverse is true for external causes, where changing racial patterns for men contributed more to the narrowing of the black-white gap in life expectancy. Men's greater contribution with respect to external causes is attributable largely to accidental poisoning, as poisoning fatalities increased among whites, especially among white males. Gender also played a role in the contribution of HIV/AIDS to the narrowing of the black-white longevity gap, with men again contributing more than women.

Table 3: Cause-specific male and female contributions to the black-white gap in life expectancy in the United States in 2000 and 2010, and change from 2000-2010 (in years)

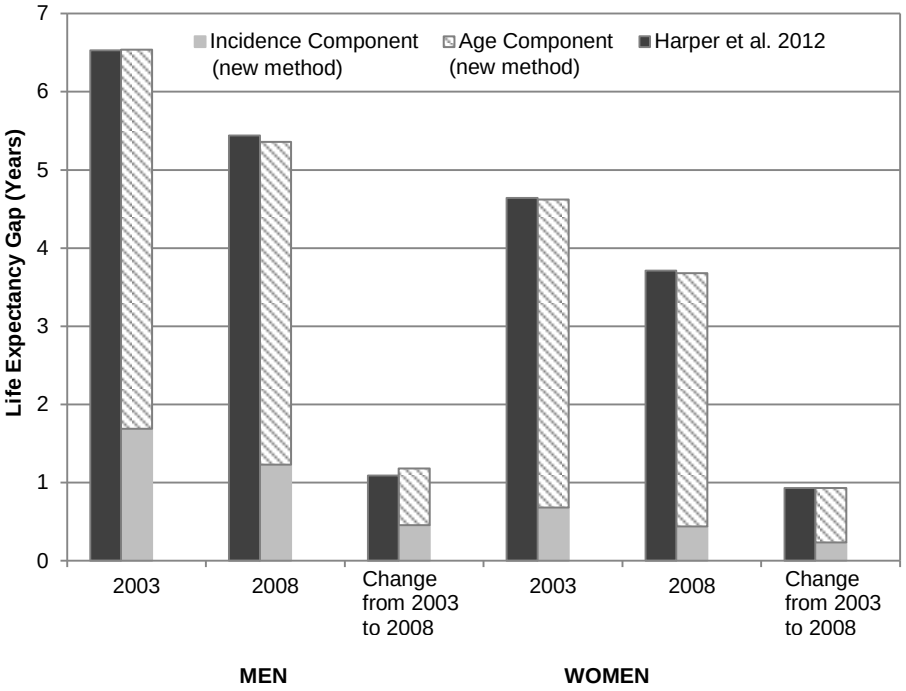
		2000			2010			Change from 2000-2010		
	ICD-10 cause of death	**Total**	**Male**	**Female**	**Total**	**Male**	**Female**	**Total**	**Male**	**Female**
Chronic diseases										
1	Heart diseases	1.49	0.66	0.83	1.02	0.57	0.45	-0.48	-0.09	-0.39
2	Cancers	0.53	0.25	0.28	0.41	0.20	0.21	-0.12	-0.05	-0.07
3	Cerebrovascular diseases	0.39	0.16	0.23	0.25	0.14	0.12	-0.14	-0.02	-0.11
4	Chronic lower respiratory diseases	0.24	0.10	0.13	0.18	0.08	0.11	-0.05	-0.03	-0.03
5	Alzheimer's	0.16	0.04	0.12	0.16	0.05	0.11	0.00	0.02	-0.01
6	Diabetes	0.08	0.06	0.02	0.07	0.06	0.01	-0.02	0.00	-0.02
7	Nephritis	0.08	0.05	0.03	0.06	0.06	0.01	-0.02	0.00	-0.02
	Totals for chronic diseases	**2.97**	**1.33**	**1.65**	**2.15**	**1.15**	**1.00**	**-0.82**	**-0.17**	**-0.65**
Communicable diseases										
8	Influenza and pneumonia	0.18	0.08	0.10	0.08	0.05	0.03	-0.10	-0.03	-0.07
9	Septicemia	0.04	0.03	0.01	0.03	0.03	0.01	0.00	0.00	0.00
10	HIV/AIDS	0.40	0.26	0.14	0.20	0.13	0.08	-0.20	-0.14	-0.06
11	Other infectious diseases	0.05	0.03	0.02	0.06	0.03	0.03	0.01	0.00	0.01
	Totals for communicable diseases	**0.67**	**0.40**	**0.27**	**0.37**	**0.23**	**0.15**	**-0.29**	**-0.17**	**-0.12**
External causes										
12	Homicide	0.53	0.45	0.08	0.50	0.45	0.06	-0.03	-0.01	-0.02
13	Suicide	-0.10	-0.08	-0.03	-0.18	-0.14	-0.04	-0.08	-0.06	-0.02
14	Traffic accident	0.00	0.01	0.00	-0.01	0.00	-0.01	-0.01	-0.01	0.00
15	Accidental poisoning	0.01	0.01	0.01	-0.19	-0.13	-0.06	-0.20	-0.14	-0.07
16	Other external causes	0.14	0.07	0.07	0.13	0.06	0.07	-0.01	-0.01	0.00
	Totals for external causes	**0.59**	**0.47**	**0.12**	**0.25**	**0.24**	**0.02**	**-0.34**	**-0.23**	**-0.10**
Infant and child conditions										
17	Perinatal deaths	0.41	0.23	0.18	0.29	0.16	0.13	-0.12	-0.07	-0.05
18	Congenital anomalies	0.05	0.02	0.02	0.04	0.02	0.02	-0.01	0.00	-0.01
	Totals for infant and child conditions	**0.46**	**0.25**	**0.20**	**0.33**	**0.18**	**0.15**	**-0.12**	**-0.07**	**-0.05**
Aggregated minor causes										
19	NEC - Not elsewhere classified	1.02	0.45	0.57	0.73	0.34	0.39	-0.29	-0.11	-0.18
Grand total		**5.70**	**2.89**	**2.81**	**3.84**	**2.14**	**1.70**	**-1.86**	**-0.75**	**-1.11**

In short, racial differences in mortality narrowed for virtually all major causes of death from 2000 to 2010. Although this was true for both women and men, women overall contributed more to the narrowing than men did, primarily because the convergence in the racial patterning of chronic diseases was greater among women. In the case of external causes, by contrast, there was greater convergence in the racial patterning for men than for women. Homicide contributed virtually nothing to the narrowing of the racial gap because, for both men and women, the racial patterning of homicide changed very little.

8. What is new here: Comparison with a prior study

To underscore how our approach differs from prior approaches, we used it to reanalyze and extend the findings of Harper et al. (2012), who used the Arriaga decomposition method to determine why the black-white gap in life expectancy narrowed from 2003 to 2008. They analyzed men and women separately. Figure 4 summarizes key results. For women, Harper et al. (2012) find that the racial gap declined from 4.64 years down to 3.71 years, compared to our finding of 4.62 years down to 3.69 years (the small difference most likely is due to our use of updated 2010 census population figures for the life table denominators). By employing our method we can decompose those racial differences further. We find that the age component accounts for 85% of the racial gap in 2003 and 88% of the gap in 2008, and that three-fourths of the narrowing of the gap was due to declining differences in the mean age at death for black versus white women who die of the same causes. For men we also find that the convergence in life expectancy is due primarily to narrowing differences in cause-specific mean age at death. These findings are unavailable in prior studies because those studies rely on cause decompositions that do not separate age and incidence components. The new method permits researchers to quantify age and incidence components not only for all causes collectively (equations 6 and 7 above) but also for specific causes (equations 4 and 5 above).

Figure 4: Narrowing of the racial gap in life expectancy from 2003-2008: What the new method contributes



9. Discussion and conclusion

This study introduces a general method to account for group differences in life expectancy by separating the portion of the gap due to group differences in the probability of dying from given causes from the portion due to group differences in cause-specific average age at death. We use the method to determine why whites tend to outlive blacks in the United States, and why that racial difference has declined from 2000 to 2010. We find that the average age at death for whites is higher for virtually all causes of death. As a result the age component amounts to 76% of the racial gap in life expectancy in 2000 and to 84% of the gap in 2010. The remainder of the gap is attributable to racial differences in the probability of dying from specific causes of

death, which we call the incidence component. In contrast to our results for the cause-specific age components, we find significant offsetting effects for the cause-specific incidence components. In particular, racial differences in the incidence of HIV/AIDS, homicide, and perinatal deaths widen the racial gap in life expectancy, whereas racial differences in the incidence of suicide narrow the gap. Racial differences in accidental poisoning also narrow the gap, but only in 2010.

Blacks' life expectancy increased faster than whites' life expectancy from 2000 to 2010 largely because gains in years of life expectancy were greater for blacks for nearly all chronic diseases, the most prevalent causes of death in the United States. As a result, 60% of the narrowing of the longevity gap was attributable to the age component, with black-white age differences declining for three leading causes of death (heart disease, cancer, and cerebrovascular disease) as well as for minor causes of death not elsewhere classified. The remaining 40% of the reduction in the life expectancy gap was associated with change in the distribution of the causes of death among blacks and whites. In this case, heart disease and other types of chronic disease played only a minor role: the reduction in the incidence component instead was produced almost entirely by greater declines among blacks (than whites) in HIV/AIDS and in perinatal deaths, and by the near-tripling of deaths due to accidental poisoning among whites. Although homicide is a significant contributor to the racial gap in life expectancy in the United States, it contributed very little to the narrowing of the racial gap from 2000 to 2010 because the racial patterning of homicide changed very little over this decade.

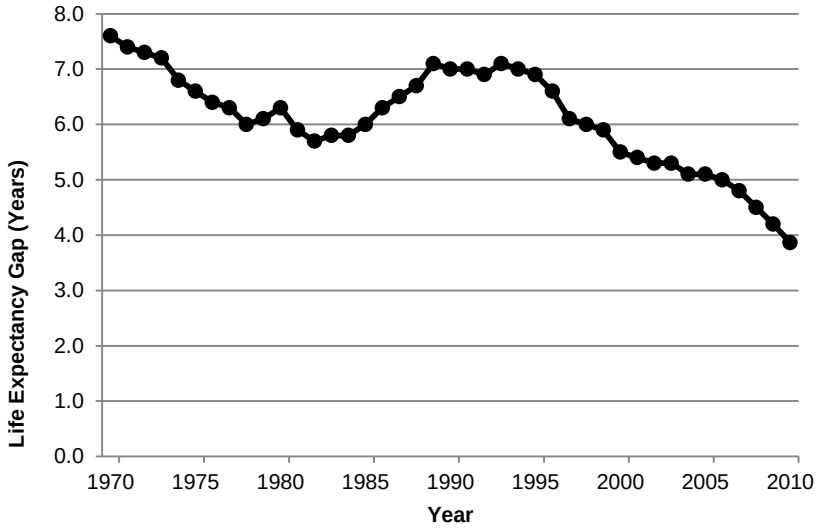
For most major causes of death, racial differences in mortality narrowed from 2000 to 2010 for both women and men. Women, however, contributed more to the narrowing of the racial gap because of greater convergence in their racial patterning of chronic diseases. The effect of this trend on the gap in life expectancy was particularly amplified because chronic diseases account for the large majority of all deaths.

In interpreting our results, readers should bear in mind the limitations of studies based on the underlying cause of death. The underlying cause may be hard to determine, particularly among the elderly, where multiple diseases at the time of death are more likely (Israel, Rosenberg, and Curtin 1986). Where cause of death is miscoded, the error has been shown to be correlated with race (Noymer, Penner, and Saperstein 2011). In addition, population estimates could be biased, especially for minority populations, by census undercounts, age misreporting, and race misclassification. By using five-year age categories and relatively broad cause groups, we reduce the effect of these misclassifications. We focused on (1) the most common causes of death, and (2) those causes, such as homicide, HIV/AIDS, accidental poisoning, and suicide, that are less common but disproportionately afflict blacks or whites. Although information is lost due to the aggregation of causes in our classification scheme, it is important to note that, under the method used here,

disaggregating a particular cause category further would not affect results for the other causes.

Our study has implications for policy and for future research. With regard to research, the method developed here can be used in subsequent studies to shed light on the factors producing longevity gaps between countries as well as between demographic or socioeconomic groups within countries. The method could also be applied to the racial longevity gap in the United States over a longer span of time. The time period we analyzed in this study is strategic because it corresponds to an initiative in the United States to eliminate racial health disparities (U.S. Department of Health and Human Services 2000). The analysis could be pushed back in time, however, because official life tables by cause of death have been available for the United States since the late 1960s (U.S. Department of Health, Education and Welfare 1968). As Figure 5 shows, the trend in the racial life expectancy gap in the United States has been erratic over the past 40 years, declining in the 1970s, rising in the 1980s, and then declining again from the mid-1990s. The method described in this paper could be used to determine the reasons for this undulating trend.

Figure 5: Black-white gap in life expectancy, 1970-2010



Source: CDC/NCHS, National Vital Statistics System (1970-2008 reported in Arias 2012). Life expectancies for 2009 and 2010 calculated from the CDC/NCHS data.

Our study also bears on policy. By clarifying the factors behind the racial gap in life expectancy in the United States at two points in time, we identify where significant advances have been made with respect to the goal of eliminating racial disparities in life expectancy, reveal places where advances still need to be made to achieve the goal, and inform policy regarding the most strategic targets for intervention to reduce disparities. Consider the implications of our findings for heart disease, for example. The incidence component is negligible, indicating that blacks and whites are equally likely to succumb to heart disease. Heart disease nonetheless accounts for about one-fourth of the racial gap in life expectancy in 2010 because black victims tend to be younger than white victims. Together, these findings point to policy interventions focusing on identifying heart disease and equalizing treatment of those with heart disease. Indeed, for chronic diseases in general – including cancers, cerebrovascular diseases, Alzheimer's, diabetes, and nephritis – the age component dominates, indicating that treatment may be important in reducing disparities. In the case of HIV/AIDS, by contrast, the racial gap is driven by differences in incidence, not age at death. In that case prevention is the key to reducing disparities.

In short, by isolating the age and incidence components for principal causes of death, we are in a better position to determine where policy interventions are likely to have the greatest payoff for narrowing racial differences in life expectancy. One thing is clear: continued reductions in the black-white longevity gap are unlikely to come about automatically, but will require conscious effort, informed by an understanding of the sources of the gap. The methodology and results of this study are designed to further that understanding.

10. Acknowledgements

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Appendix

Table A1: Cause-grouping and corresponding ICD-10 codes

ICD 10 Cause grouping		
19- Category Cause-Group	**ICD10 4 digit code range**	**Contents of Cause-group (described using ICD10 cause description)**
(1) **Chronic diseases**		
Heart diseases	I00-I09.9	Acute rheumatic fever, chronic rheumatic heart disease
	I11-I11.9	Hypertensive heart disease
	I13-I13.9	Hypertensive heart and renal disease
	I20-I25.9	Ischemic heart disease
	I26-I28.9	Pulmonary heart disease and diseases of pulmonary circulation
	I30-I51.9	Other forms of heart disease
Cancers	C00-D48.9	Neoplasms
Cerebrovascular diseases	I60-I69.8	Cerebrovascular diseases
Chronic lower respiratory diseases	J40-J47	Chronic lower respiratory diseases
Alzheimer's	G30-G30.9	Alzheimer's disease
Diabetes	E10-E14.9	Diabetes mellitus
Nephritis, Nephrosis, Nephrotic Symptom	N00-N07.9 N17-N19 N25-N27.9	Nephritis, Nephrosis, Nephrotic Symptom
(2) Communicable diseases		
Influenza and pneumonia	J10-J18.9	Influenza and pneumonia
Septicemia	A40-A41.9	Streptococcal and other septicemia
HIV/AIDS	B20-B24.9	HIV disease
Other Infectious Diseases	A00-B99	Certain infectious and parasitic diseases, with the exception of septicemia and STD/NTD
(3) External causes		
Homicide	X85-Y09.9	Assault
Suicide	X60-X84.9	Suicide
	X60-X69	Intentional self-poisoning by drugs and alcohol
	X70-X78	Intentional self-harm by suffocation and by objects
	X80-X82	Intentional self-harm by jumping or lying before moving objects
	X83-X84.9	Intentional self-harm by other specified/unspecified means

Table A1: (Continued)

ICD 10 Cause grouping 19- Category Cause-Group	ICD10 4 digit code range	Contents of Cause-group (described using ICD10 cause description)
(3) External causes		
Traffic Accidents	V02-V04.9	Pedestrian injured in accident with motor vehicle
	V09-V09.9	Pedestrian injured in other and unspecified transport accidents
	V12-V14.9	Pedal cyclist injured in accident with motor vehicle, Pedal cyclist injured in unspecified transport/traffic accident
	V19-V19.9	Motor-cyclist, occupant of three-wheeled vehicle, car occupant, occupant of pick-up truck, occupant of heavy transport vehicle, or bus occupant injured in transport accident
	V20-V79.9	Other land transport accidents
	V80-V89.9	
Accidental poisoning	X40-X49.9, Y10-Y19.9	Accidental poisoning by drugs and alcohol Poisoning by drugs and alcohol, undetermined intent
Other External Causes	Remaining codes V01-V01.9, V90-Y89.9 excluding assault	External causes of morbidity and mortality, with exception of assault
(4) Infant and child conditions		
Perinatal deaths	P00-P96	Conditions originating in the perinatal period
Congenital Anomalies	Q00-Q99	Conditions originating from congenital absence or malformation
(5) Aggregated minor causes (not elsewhere classified)		
NEC – Not elsewhere classified	All codes not listed above	Examples: F1-F 19.9 - Different types of dementia/mental disorder F20-F48 - Other mental disorders including schizophrenia, delusional disorder, psychosis, bipolar affective disorder, depression, anxiety disorder, etc. F50 - Anorexia nervosa

Table A2: Probability of death, and average age at death, for 19 causes of death: Blacks versus whites in 2000 and 2010[a]

		Probability in 2000			Mean Age in 2000			Probability in 2010			Mean Age in 2010		
	ICD-10 cause of death	**White**	**Black**	**Diff**	**White**	**Black**	**Diff**	**White**	**Black**	**Diff**	**White**	**Black**	**Diff**
Chronic diseases													
1	Heart diseases	0.314	0.299	0.015	80.4	75.7	4.7	0.255	0.260	-0.004	81.3	77.3	4.0
2	Cancers	0.230	0.226	0.004	73.7	71.4	2.4	0.224	0.224	0.000	75.3	73.5	1.8
3	Cerebrovascular diseases	0.075	0.076	-0.001	82.4	77.1	5.3	0.056	0.062	-0.006	82.8	78.1	4.8
4	Chronic lower respiratory diseases	0.057	0.029	0.028	78.3	74.2	4.1	0.063	0.033	0.030	79.6	77.1	2.4
5	Alzheimer's	0.026	0.013	0.013	85.9	85.2	0.7	0.043	0.029	0.014	86.8	86.5	0.2
6	Diabetes	0.025	0.045	-0.020	76.2	73.4	2.8	0.023	0.043	-0.020	77.1	75.2	2.0
7	Nephritis, nephrosis, nephrotic symptoms	0.015	0.026	-0.011	80.4	74.6	5.8	0.020	0.034	-0.014	81.8	77.6	4.2
	Total probability, chronic diseases	**0.741**	**0.713**	**0.027**				**0.685**	**0.686**	**0.000**			
Communicable diseases													
8	Influenza and pneumonia	0.030	0.024	0.006	83.4	78.2	5.1	0.022	0.020	0.002	82.9	79.6	3.3
9	Septicemia	0.012	0.022	-0.010	78.7	75.0	3.7	0.013	0.022	-0.009	78.6	76.2	2.4
10	HIV/AIDS	0.002	0.016	-0.014	45.0	45.6	-0.6	0.001	0.009	-0.008	51.2	50.9	0.3
11	Other infectious diseases	0.004	0.006	-0.002	69.4	62.9	6.6	0.009	0.008	0.000	75.7	68.7	7.0
	Total probability, communicable	**0.048**	**0.068**	**-0.020**				**0.045**	**0.060**	**-0.015**			
External causes													
12	Homicide	0.002	0.015	-0.012	39.5	32.6	6.9	0.002	0.013	-0.011	40.5	32.2	8.3
13	Suicide	0.010	0.004	0.006	50.0	41.9	8.1	0.012	0.004	0.008	50.0	42.4	7.6
14	Traffic accident	0.013	0.012	0.001	45.5	43.0	2.5	0.010	0.009	0.001	48.2	45.3	2.9
15	Accidental poisoning	0.004	0.005	-0.001	42.4	46.0	-3.6	0.011	0.006	0.004	42.4	49.0	-6.6
16	Other external causes	0.017	0.015	0.002	69.9	60.4	9.5	0.023	0.015	0.008	74.3	64.8	9.5
	Total probability, external causes	**0.045**	**0.051**	**-0.005**				**0.057**	**0.047**	**0.010**			
Infant deaths													
17	Perinatal Deaths	0.003	0.008	-0.006	0.6	0.6	0.1	0.002	0.006	-0.004	0.7	0.6	0.2
18	Congenital Anomalies	0.003	0.003	0.000	27.6	18.8	8.7	0.003	0.003	0.000	27.5	19.6	7.9
	Total probability, infant deaths	**0.006**	**0.012**	**-0.006**				**0.005**	**0.009**	**-0.004**			
Aggregated minor causes													
19	NEC - Not elsewhere classified	0.160	0.156	0.004	78.0	71.6	6.4	0.208	0.198	0.009	80.3	76.8	3.5
Overall		**1.000**	**1.000**	**0.000**	**77.0**	**71.3**	**5.7**	**1.000**	**1.000**	**0.000**	**78.1**	**74.3**	**3.8**

[a] Based on multi-decrement life tables (see text)