The Shifting Age Pattern of the Black-White Life Expectancy Gap

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Abstract

The black-white life expectancy gap has narrowed dramatically and reached its lowest point since 1990. While previous studies examined cause of death patterns, relatively few focused on the shifting age pattern of the black-white gap. This paper identifies a structural break for the black-white gap in 2004. Prior to 2004, the black-white gap in life expectancy at age 50 was fairly constant, but in the following decade, it narrowed substantially. Mortality at ages 0-50 was the predominant driver of the narrowing gap in life expectancy at birth from 1990-2004, while mortality at ages 50+ became the important factor from 2004-2015. Much of this narrowing in 2004-2015 was due to morality improvements among blacks from chronic diseases for which effective treatment and screening were introduced in the 1980s-1990s. The diffusion of life-saving technologies like statins and cancer screening may have led to a delayed reduction in the gap at older ages.

Introduction

One of the most remarkable demographic events of the last quarter century has been the dramatic narrowing of the black-white life expectancy gap. The gap peaked in the early 1990s at 8.5 years for men and 5.9 years for women, and has since declined to 4.2 years for men and 2.9 years for women in 2014. In other words, the gap more than halved over this 20 year period.

While recent research on this narrowing has focused on cause of death contributions to the narrowing gap, far less attention has been devoted to the age pattern of reductions in the black-white life expectancy gap (Harper et al. 2007, 2012, 2014; Macinko and Elo 2009). Age patterns are important because they help us understand and contextualize contemporaneous and life course impacts on health and mortality. Furthermore, exposure to risk factors for mortality are strongly patterned by age. Finally, people at different ages are often embedded in different institutional contexts, so that for older people, factors like Medicare might be quite important, while for younger people factors like physical safety and neighborhood context may matter more.

This paper examines how the contributions of deaths under age 50 and over age 50 to the narrowing black-white gap have changed over time. The analysis starts in the year 1990 and ends in 2015, and answers three main research questions: (1) What are the recent trends in life expectancy for blacks and whites, and how do the trends in life expectancy at birth differ from those for life expectancy at age 50? (2) How have the contributions of deaths at younger (0-50) and older (50+) ages to the black-white life expectancy gap changed over time? (3) Which causes of death are contributing to the narrowing of the gap at younger versus older ages?

Data

Two types of data are used in this analysis. The first is data from the National Center for Health Statistic's official U.S. life tables for the years 1980 through 2014. These data include life expectancies at birth and at age 50 and are used to produce Figures 1-4. The second type of data are the restricted versions of the NCHS's multiple cause of death microdata files and the U.S. Census Bureau's bridged-race population file for each year between 1990 and 2015. These files include records of each death recorded in the United States, along with the age, race, sex, and cause of death for the decedent. I tabulate deaths by race (black and white), sex (male and female), standard demographic age group for abridged life tables (0-1, 1-4, 5-9, …, 80-84, 85+ years), and 17 broad cause of death categories described in Appendix Table 1.

Methods

The methods used in this paper are standard demographic techniques that can mostly be found in Preston et al. (2001) and the studies cited therein. Each specific method is described below.

Abridged Period Life Tables

Using the multiple cause of death and bridged-race population data, I first compute age-specific death rates by race, sex, and year. Death rates are computed as standard occurrence-exposure ratios and are assumed to equal the life table (model) death rates. Within each year, I

compute period life tables for each race-sex group using the abridged age groups described above. $_na_x$ values are computed using graduation (Keyfitz 1966; Preston et al. 2001), and for non-graduated age groups the values are assumed to equal $\frac{n}{2}$. The open-ended age group is 85+ years.

Adjusting Mortality at the Oldest Ages

Two potential problems with the above approach are that (1) there could be significant age misreporting at ages 85+, and (2) there has been quite a lot of population growth at ages 85+, rendering that age group non-stationary. The first issue is problematic since age misreporting will tend to bias mortality rates, and, depending in part on the direction of the misreporting, the bias itself could be in either direction. The second issue is problematic because, when computing a crude death rate for an open-ended age category and using that rate in a life table, we are implicitly assuming that that death rate is equal to the stationary-equivalent population's death rate. If that age group has been growing rapidly in recent years, as has been the 85+ group, then death rates will tend to be biased downwards, since younger people in the 85+ group will tend to be overrepresented. This will lead to life expectancy estimates that are too high.

Both of these issues are solved in this analysis by applying a growth adjustment to the life expectancy value at age 85 (Horiuchi and Coale 1982). This growth adjustment makes three plausible assumptions: (1) that there is no (or at least very limited) age misreporting below age 85, (2) that the population aged 85+ is approximately stable, and (3) that mortality at ages 85+ follows a Gompertz pattern of exponential increase. The adjustment itself is quite simple:

$$e_{85}^{adj} = {_\infty}M_{85+}^{-1} \cdot e^{\beta \cdot r \cdot {_\infty}M_{85+}^{-\alpha}}$$

where r is the growth rate of the population aged 85+, $_\infty M_{85+}$ is the crude death rate for the open-ended age group, and α and β are parameters specified in Horiuchi and Coale (1982) (assumed to be 1.4 and 0.095, respectively). The remaining life table values can be adjusted using standard relationships (Preston et al. 2001).

Because using only data from two adjacent years to compute the growth rate r can lead to highly erratic estimates, I use a regression smoothing technique to estimate r in each year. First, I estimate the following regression:

$$\log\big(P_{85+}(t)\big) = \gamma_0 + \gamma_1 t + \gamma_2 t^2 + \gamma_3 t^3 + \eta_t$$

where $P_{85+}(t)$ is the population aged 85+ in year t. I then estimate r as the derivative of this equation:

$$\hat{r} = \gamma_1 + 2\gamma_2 t + 3\gamma_3 t^2.$$

Arriaga's Decomposition

Arriaga's decomposition (Arriaga 1984) is a standard method to decompose differences in life expectancy into age and cause of death contributions. I apply this decomposition to the black-white life expectancy gap in a given year, and then compute changes in this decomposition across

periods. I sum up the decomposition values across the abridged age groups to get cause-specific (and all-cause) contributions for age groups 0-50 and 50+.

Decomposition of Diagnosis vs. Treatment

In subsequent analyses (not yet complete), I will use data from the National Health Interview Survey (NHIS) and the National Health and Nutrition Examination Survey (NHANES) to compute a decomposition of black-white differences in cause-specific mortality into diagnosis and treatment components. An age-specific mortality rate for cause of death i (e.g., diabetes mortality) can be written as:

$$_nM_x^i = {}_n\pi_x^i \cdot {}_nM_x^{i,diagnosed} + (1 - {}_n\pi_x^i) \cdot {}_nM_x^{i,undiagnosed}$$

where π^i is the proportion diagnosed with a particular condition (e.g., diabetes) and the two death rates on the right-hand side at the death rates from cause i for people who were diagnosed versus not diagnosed with a particular condition. I would compute these values for both blacks and whites, and decompose the difference between them. If there is no racial difference in timely and correct diagnoses, then one would expect a smaller racial difference in $_nM_x^{i,undiagnosed}$. We can thus use the difference in this value as a proxy for the effect of differential diagnosis timeliness/quality. Similarly, racial differences in $_nM_x^{i,diagnosed}$ can be used as a proxy for the quality and quantity of treatment of the condition. Thus, when applied over time, this decomposition between blacks and whites can give us three quantities: the portion of the change in the black-white cause-specific mortality gap due to treatment, diagnosis, and an interaction between treatment and diagnosis.

Results

Generally, the results indicate improvement in black and white life expectancies, substantial narrowing black-white life expectancy gaps, and rapidly changing age and cause of death contributions to these narrowing gaps.

What are the trends in black and white life expectancies?

Figure 1 plots life expectancy at birth for black and white men and women. In 1990, life expectancy at birth was 64.5 years for black men, 73.6 years for black women, 72.7 years for white men, and 79.4 years for white women. Between 1990 and 1993, there was little to no improvement in black life expectancy for either men or women, though life expectancy did improve for whites. Starting in 1993, life expectancy among blacks increased at an impressive pace of 3.8 years per decade for men and 2.3 years per decade among women. These are quite high compared to the rates of improvement among whites, which were 1.7 years per decade and 0.9 years per decade for men and women, respectively. In short, the pace of improvement in life expectancy for blacks during this period is nothing short of phenomenal. The usual metric for life expectancy improvement is to compare to the "best practice" life expectancy—that is, the rate of improvement among the world leaders. This rate is roughly 2.5 years per decade. The world leaders during most of this period were Japanese men and women, who, over this period, experienced gains of 4.6 and

5.0 years, respectively, compared to the 8.0 and 4.9 year improvements among black men and women.

Part of the reason for the dramatic improvement among blacks may be that they started out at such a low level to begin with, and that the improvement was mostly concentrated at young ages which have an outsize impact on life expectancy. We thus additionally examine trends in life expectancy at age 50.

Figure 2 plots life expectancy at age 50 for black and white men and women. In 1990, life expectancy at age 50 was 22.5 years for black men, 28.2 years for black women, 26.7 years for white men, and 31.6 years for white women. Between 1990 and 1995, black men and women and white women saw moderate increases in life expectancy at age 50, while white men saw more robust rates of improvement. Starting in 1995, black men and women and white women began a period of dramatic improvement in life expectancy at age 50 (while white men continued their pace of impressive improvement). The pace of improvement for black men and women accelerated in the 2000s and surpassed the pace of improvement for white men and women. Nevertheless, by 2014, black life expectancy at age 50 was still much lower than for whites. In fact, the 2014 levels of life expectancy at age 50 for black men were close to the 1994 levels for white men, and the 2014 levels for black women were close to the 1989 levels for white women. In other words, black men still lagged behind white men by two decades and black women lagged behind white women by a quarter century.

Figure 3 plots trends in the black-white life expectancy gap at birth and at age 50 for men and women. The black-white gap in life expectancy at birth in 1990 was 8.2 years for men and 5.8 years for women. This gap increased slightly from 1990-1993, and then began a precipitous decline. By 2014, the black-white gaps in life expectancy at birth were 4.2 years and 2.9 years for men and women, respectively.

One concern is that the narrowing of the black-white gap in life expectancy at birth may not be due to gains in black life expectancy, but may instead be due to stagnation or decline in white life expectancy. Figure 1 indicates that this is unlikely to be the case. Figure 4, which plots the five-year average annualized rate of increase in life expectancy for black and white men and women, provides additional evidence to counter this hypothesis. We see that, with the exception of 2010-2014, the rates of improvement in white life expectancy actually increased since 1995 and are within the normal range of historical life expectancy improvements prior to the opioid epidemic (around 0.1 years of improvement per annum for white women and 0.2 years of improvement per annum for white men). Thus, the reduction of the black-white life expectancy gap isn't due solely to the opioid epidemic or related factors, but is instead being driven by real and robust improvements in black mortality.

The remaining lines in Figure 3 show trends in the black-white gap at age 50. In 1990, the black-white gap in life expectancy at age 50 was 4.2 years for men and 3.4 years for women. This gap stayed fairly constant through the early 2000s, at which point the gaps began to decline. By 2014, the black-white gaps in life expectancy at age 50 were 2.8 years for men and 1.9 years for women.

Once again, this narrowing of the black-white gap in life expectancy at age 50 is not due to poor performance among whites. In fact, in the 2004-2010 period, when most of the narrowing occurred, whites were experiencing the fastest rates of increase in life expectancy at age 50 since 1980. Rather, it was the superlative performance of blacks that led to the narrowing of the black-white gap.

How have the contributions of deaths at younger versus older ages to the black-white gap changed over time?

Next, we examine the age pattern of improvements in the black-white life expectancy gap. This analysis examines two broad age groups: ages 0 to 50 years and ages 50 years and older. Fifty is used as the dividing value because different phenomena tend to determine mortality on either side of age 50. People younger than age 50 are embedded in different work, family, and institutional contexts, and these different contexts, along with different levels of accumulated inflammation and biological risk factors, results in a greater propensity to die of external causes of death relative to people aged 50 and older, who die more often of chronic disease mortality.

Figures 5 and 6 plot the contribution of ages 0-50 and 50+ years to the change in the black-white gap in life expectancy at birth for men and women, respectively. The reference year is 1990, so the values for that year are zero for both ages 0-50 and 50+. The interpretation of these estimates is as follows: if the value of the 50+ contribution is 0.5 years in year t, then the decline in mortality at ages 50+ between 1990 and year t decreased the black-white gap in life expectancy at birth between 1990 and year t by 0.5 years. If the corresponding value for the 0-50 contribution is 0.6 years, then the decline in mortality at ages 0-50 between 1990 and year t decreased the black-white gap in life expectancy at birth by 0.6 years. These two numbers add up to the total decline in the black-white gap in life expectancy at birth, which is 0.6+0.5=1.1 years.

Figure 5 shows that ages 0-50 contributed to a rapid decline in the black-white gap for men starting around 1993. The magnitude of the 0-50 contribution increased quickly through the early 2000s, at which point the pace slowed. Perhaps the most striking finding of this article is the trajectory of the dashed red curve representing the contribution of ages 50+ to the narrowing black-white life expectancy gap. Ages 50+ basically made no contribution at all to the narrowing of the gap from 1990 through 2004. However, in 2004 there was a sharp increase in the magnitude of the 50+ contribution, and that pace of improvement continued through 2015. It is currently unclear what could have caused this sudden takeoff in 2004, but it is likely that a sudden improvement in chronic disease mortality among blacks in 2004-2015 led to ages 50+ making increasingly important contributions to the narrowing black-white gap.

Figure 6 is the corresponding graph for women. Between 1990 and 2004, both age groups, 0-50 and 50+, contributed to the narrowing of the black-white gap in female life expectancy at birth. Relative improvements in mortality at ages 0-50 made the greater contribution, producing almost 2/3 of the narrowing. Starting in 2004, the contribution of ages 0-50 continued to increase in magnitude at roughly the same pace, but the trend for ages 50+ became much steeper. The contribution of ages 50+ increased in magnitude from 2004-2014 at *triple* the rate of the 1990-2004 period. Once again, it is unclear what caused this sudden and dramatic increase in the rate of

change for contribution of mortality at ages 50+ to the narrowing of the black-whtie gap, but it seems likely that chronic disease mortality played a major role.

Which causes of death are contributing to the narrowing of the gap at younger versus older ages?

Figure 7 plots the contribution of various causes of death at ages 0-50 to the narrowing black-white gap in life expectancy at birth for men. Negative contributions indicate that these causes of death contributed to the narrowing of the gap, and positive contributions indicate that these causes of death contributed to the widening of the gap. In the early 1990s, the gap increased slightly due to higher HIV/AIDS mortality among blacks. This reversed in the mid-90s, and by the last year the largest contributors at ages 0-50 to the narrowing of the black-white gap between 1990 and 2015 were: HIV/AIDS (-0.49 years), homicide (-0.48 years), drug overdose (-0.3 years), perinatal conditions (-0.27 years), digestive diseases (-0.24 years), circulatory diseases (-0.22 years), ill-defined conditions (-0.18 years), other external injuries (-0.18 years), mental disorders (-0.13 years), respiratory diseases (-0.12 years), other cancers (-0.12 years), and infectious diseases (-0.11 years). Lung cancer, residual deaths, genitourinary conditions, nervous system disorders and Alzheimer's, and breast, prostate, and colorectal cancers all made contributions of less than -0.1 years.

Figure 8 plots the contribution of the same causes of death at ages 0-50 to the narrowing of the black-white gap in life expectancy at birth for women. Much like for men, excess HIV/AIDS deaths led to an increase in the black-white gap in the early 1990s for women. Starting in the mid-90s, the gap began to narrow, and by 2015 the largest contributors at ages 0-50 to the narrowing of the black-white gap between 1990 and 2015 were: perinatal conditions (-0.26 years), circulatory diseases (-0.22 years), homicide (-0.19 years), drug overdose (-0.17 years), HIV/AIDS (-0.17 years), ill-defined conditions (-0.13 years), and digestive diseases (-0.13 years). The remaining causes of death each contributed less than -0.1 years to the narrowing.

As expected, the story is quite different at older ages, where chronic disease mortality predominates as opposed to infectious and external mortality. Figure 9 plots the contribution of various causes of death at ages 50 and older to the narrowing black-white gap in life expectancy at birth for men. Again, positive bars indicate that a cause of death contributed to the widening black-white gap, while negative bars indicate that a cause of death contributed to the narrowing black-white gap. For each cause of death, there are two bars. The blue bar shows the contribution to the narrowing gap for the period 1990-2004. The red bar shows the contribution to the change between 2004 and 2015. These two periods were chosen since 2004 was the year when the contribution of deaths at ages 50+ to the narrowing black-white gap increased dramatically.

Figure 9 shows a rather striking pattern. In the 1990-2004 period, while some causes of death, like lung cancer, tended to reduce the black-white gap, other causes of death cancelled out these reductions by contributing to a widening of the gap. These latter causes of death included circulatory diseases, HIV/AIDS, breast, prostate, and colorectal cancers, infectious and genitourinary diseases, drug overdose, and residual deaths. In the 2004-2015 period, *every* cause of death at ages 50+ except drug overdose contributed to a decline in the black-white gap. The largest contributors at ages 50+ to the narrowing black-white gap were circulatory diseases (-0.31

years), other cancers (-0.15 years), lung cancer (-0.10 years), digestive diseases (-0.10 years), respiratory diseases (-0.09 years), other external injuries (-0.09 years), HIV/AIDS (-0.06 years), and breast, prostate, and colorectal cancers (-0.05 years). The remaining causes of death at ages 50+ each contributed less than -0.05 years to the narrowing of the black-white gap in life expectancy at birth.

Figure 10 plots the contribution of various causes of death at ages 50 and older to the narrowing black-white gap in life expectancy at birth for women. We observe a similar pattern as for men, wherein some causes of death contributed to a widening gap, while others contributed to a narrowing gap in the 1990-2004 period. Breast, prostate, and colorectal cancers, HIV/AIDS, genitourinary diseases, infectious diseases, and residual deaths each contributed to the widening the gap. This was cancelled out by the larger contributions of the remaining causes of death at ages 50+. In the 2004-2015 period, every cause of death at ages 50+ contributed to a narrowing of the black-white gap in life expectancy at birth. The largest contributors at ages 50+ were: circulatory diseases (-0.48 years), residual deaths (-0.12 years), other external injuries (-0.07 years), respiratory diseases (-0.07 years), digestive diseases (-0.07 years), and infectious diseases (-0.05 years). The remaining causes of death each contributed less than -0.05 years to the narrowing of the gap.

Discussion

One of the surprising findings of this study is that the older adult ages (50 years and older) did not contribute to the narrowing of the black-white life expectancy gap until 2004 for men, and that the age 50+ contribution increased rapidly for women in the years following 2004. It's both unexpected and puzzling that mortality at ages 50+ should take on a greater role following 2004. This finding raises questions about what could have happened either during that period or what made the birth cohorts aged 50+ during that period distinctive from the preceding birth cohorts. There are two types of explanations that may have driven these findings: a life course / cohort effects explanation (Preston et al. 1998), and a technological diffusion / period effects explanation (Levine et al. 2007, 2010).

According to the life course explanation, the disparity in early life conditions between blacks and whites decreased across birth cohorts starting around the late 1940s and early 1950s. These were the birth cohorts who were entering the 50+ age group around 2004. Because these more recent birth cohorts had smaller black-white disparities in early life conditions, this led to improvements in mortality at older ages starting in 2004. This hypothesis has some face validity, since there were many social changes for these birth cohorts that could have plausibly improved their later life mortality. First, these birth cohorts include the early baby boomers, and their fathers could potentially have benefited from the GI Bill, resulting in better jobs and living conditions. Second, these were the birth cohorts that experienced firsthand the civil rights movement and benefited from desegregation and increased levels of schooling. The recognition and enforcement of civil rights could lead to lower accumulated inflammation over the life course, which is a correlate of chronic disease mortality. Increased schooling leads to better jobs, better pay, and better living conditions overall, along with the eschewal of negative health behaviors and adoption of healthy practices, which are also tied to lower mortality. Third, the birth cohorts born in the

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mid-1950s were among the first to benefit from the introduction of Medicaid, which was adopted by most states by 1966 and by all but two states by 1970. Better health care at younger ages could possibly be tied to lower incidence or less severe presentation of chronic disease at older ages.

The technology diffusion explanation is more about instantaneous effects than about life course mechanisms. According to this explanation, life-saving technologies were deployed in the 1980s and 1990s and were offered to and quickly adopted by whites earlier than for blacks. This earlier receipt and adoption of new technologies could have led to earlier declines in chronic disease mortality for whites relative to blacks. Under this hypothesis, blacks would have started benefiting from these technologies in the early 2000s, so that by 2004 their rate of improvement at the older ages would surpass that of whites, leading to a narrowing black-white gap. This hypothesis is also quite plausible. There were several life-saving technologies that were adopted during the 1980s and 1990s, including anti-hypertensives, statins, cancer screening technologies, and drug-eluting stents. If these technologies were offered to whites earlier on average than to blacks, then we would expect to see a more rapid increase in life expectancy at age 50 for whites relative to blacks in the 1990s, and then a more rapid improvement for blacks relative to whites in the 2000s. This is indeed what we see, and this hypothesis also finds support in the cause of death decompositions.

Figure 9, for example, shows that changes in cardiovascular disease mortality at ages 50+ in the 1990s led to a growing black-white life expectancy gap for men. One could imagine that this was the result of white men being prescribed statins at a higher rate than black men. In the 2000s, as statins began to diffuse to the black population, black men experienced rapid improvements in cardiovascular disease mortality, leading to sizeable contributions of mortality at ages 50+ to the narrowing black-white life expectancy gap. We see similar changes for other causes of death for which new, effective screening and treatment options were introduced or more widely disseminated in the 1990s and early 2000s, including prostate and colorectal cancer, HIV/AIDS, and diabetes (which is in the residual deaths category). Each of these causes of death contributed to a growing black-white disparity in the 1990-2004 period and a narrowing gap in the 2004-2015 period, which is consistent with a later adoption of life-saving technologies among blacks than whites. Figure 10 indicates that this may have been partly the case for women, too, particularly for diabetes mortality, HIV/AIDS and other infectious diseases, and breast and colorectal cancer.
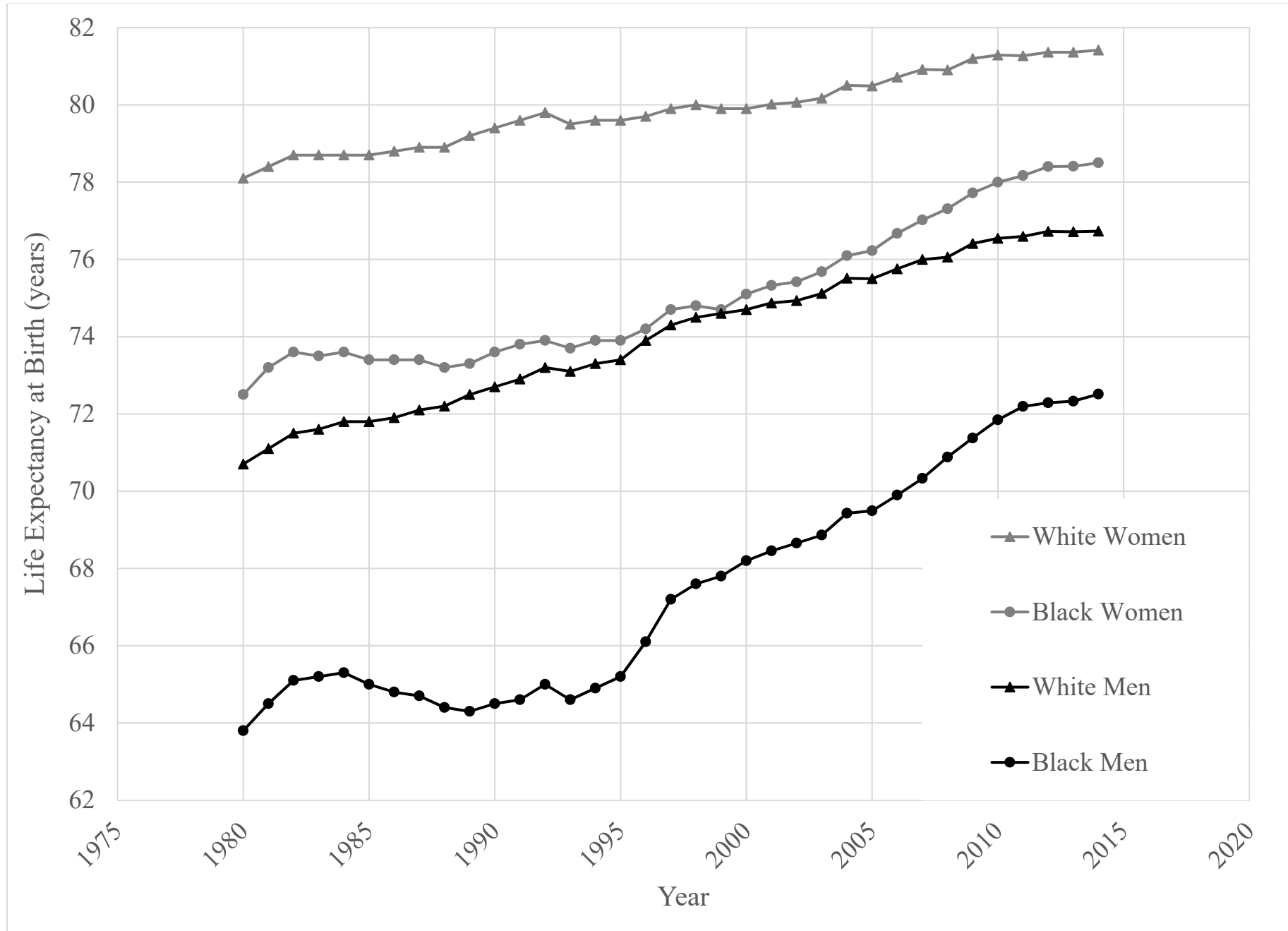
Next Steps

The next steps of this project will be to use data from the National Health Interview Survey (NHIS) and the National Health and Nutrition Examination Survey (NHANES) to test whether the differential adoption by race of new technologies like statins and cancer screening led to earlier mortality improvements among whites relative to blacks. In addition, I plan to use data from the NHIS, along with the diagnosis/subsequent mortality decomposition described in the methods section, to decompose the narrowing of black-white gaps in cause-specific mortality into diagnosis and treatment components. These analyses will shed light on the relative contribution of black-white disparities in initial diagnosis versus receipt of treatment to trends in black-white mortality gaps.

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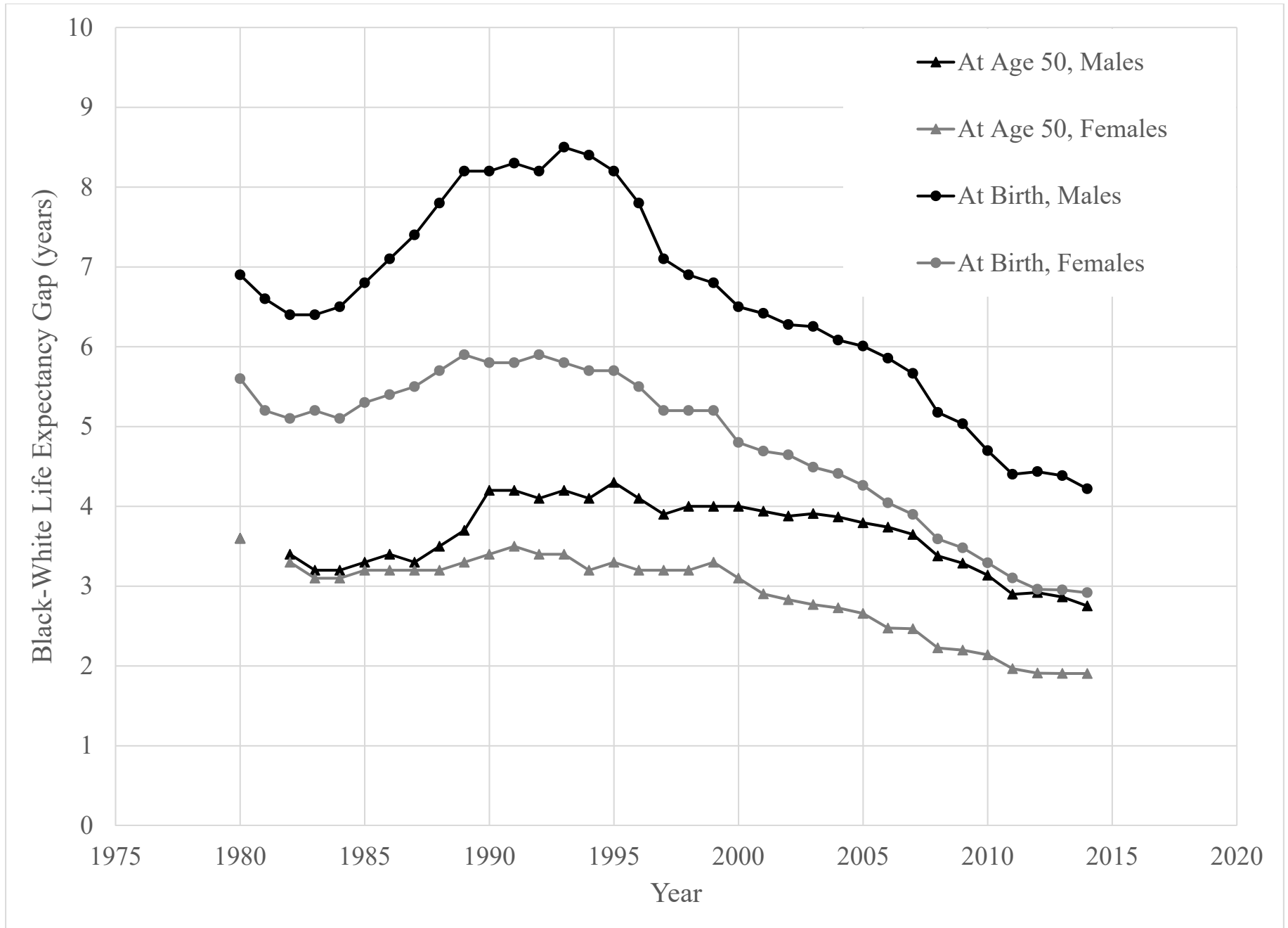
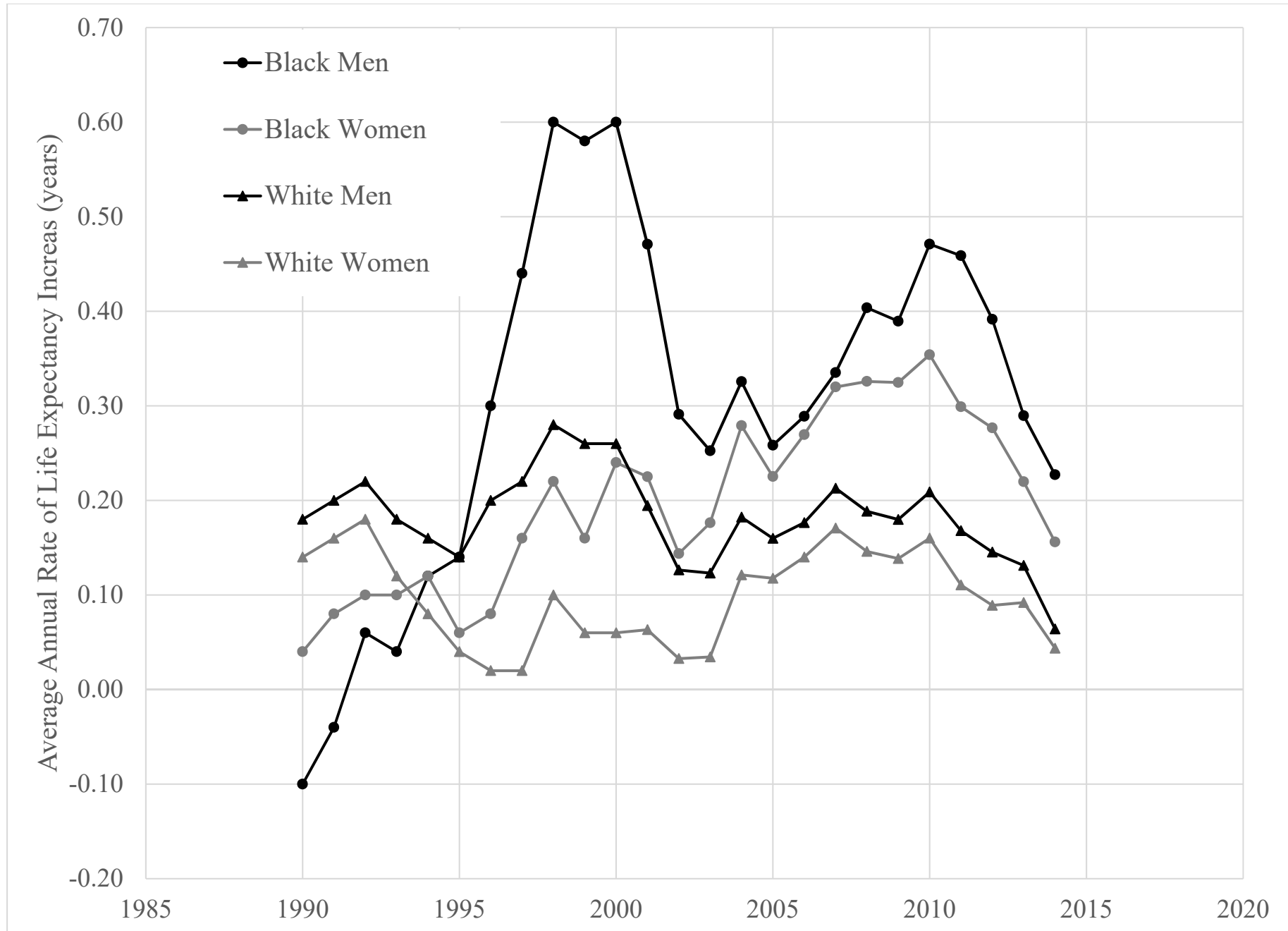**Figure 1**. Life Expectancy at Birth by Race and Sex, 1980-2014

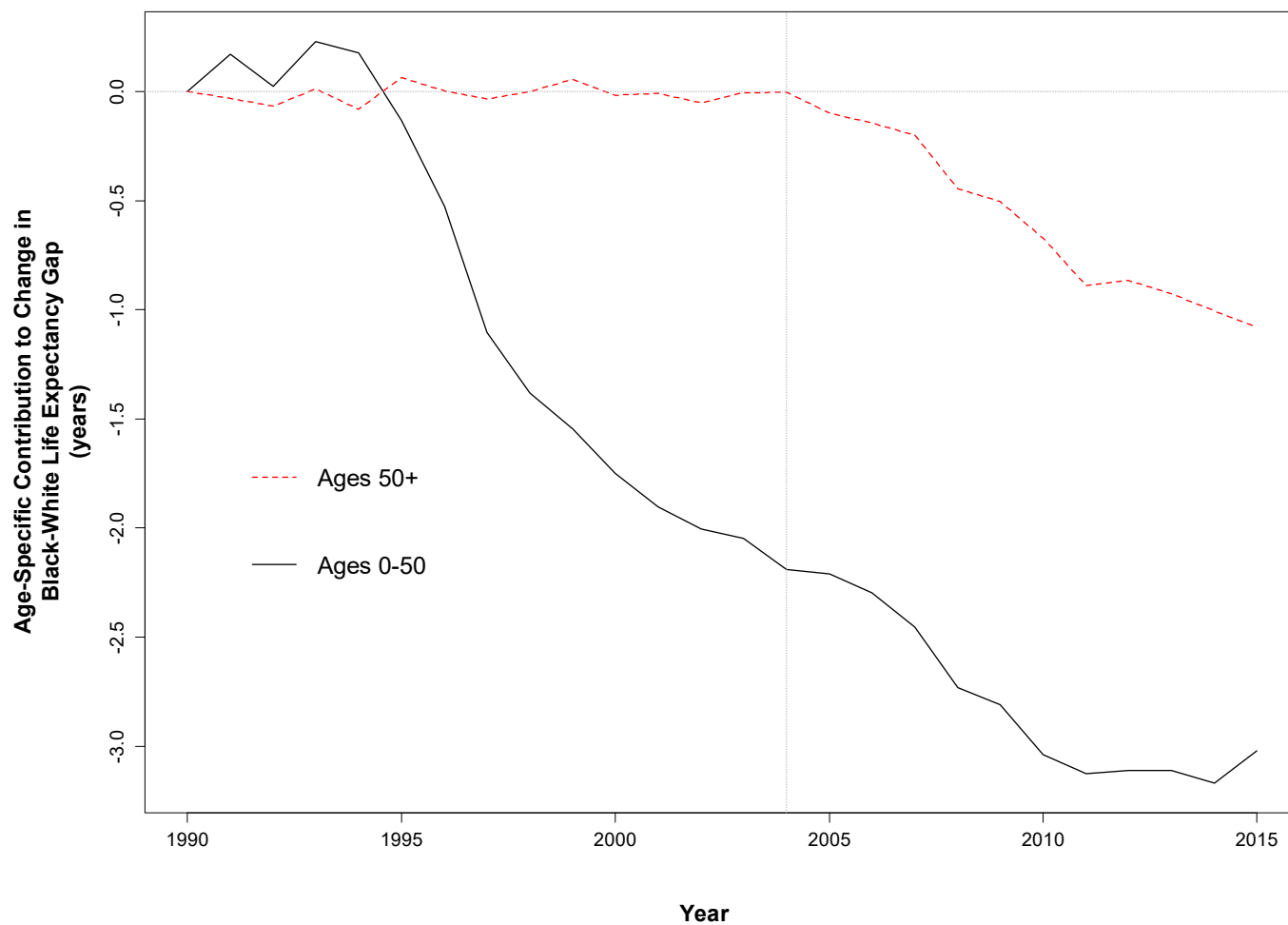**Figure 2**. Life Expectancy at Age 50 by Race and Sex, 1980-2014

Figure 3. Black-White Gaps in Life Expectancy at Birth and at Age 50 by Sex, 1980-2014

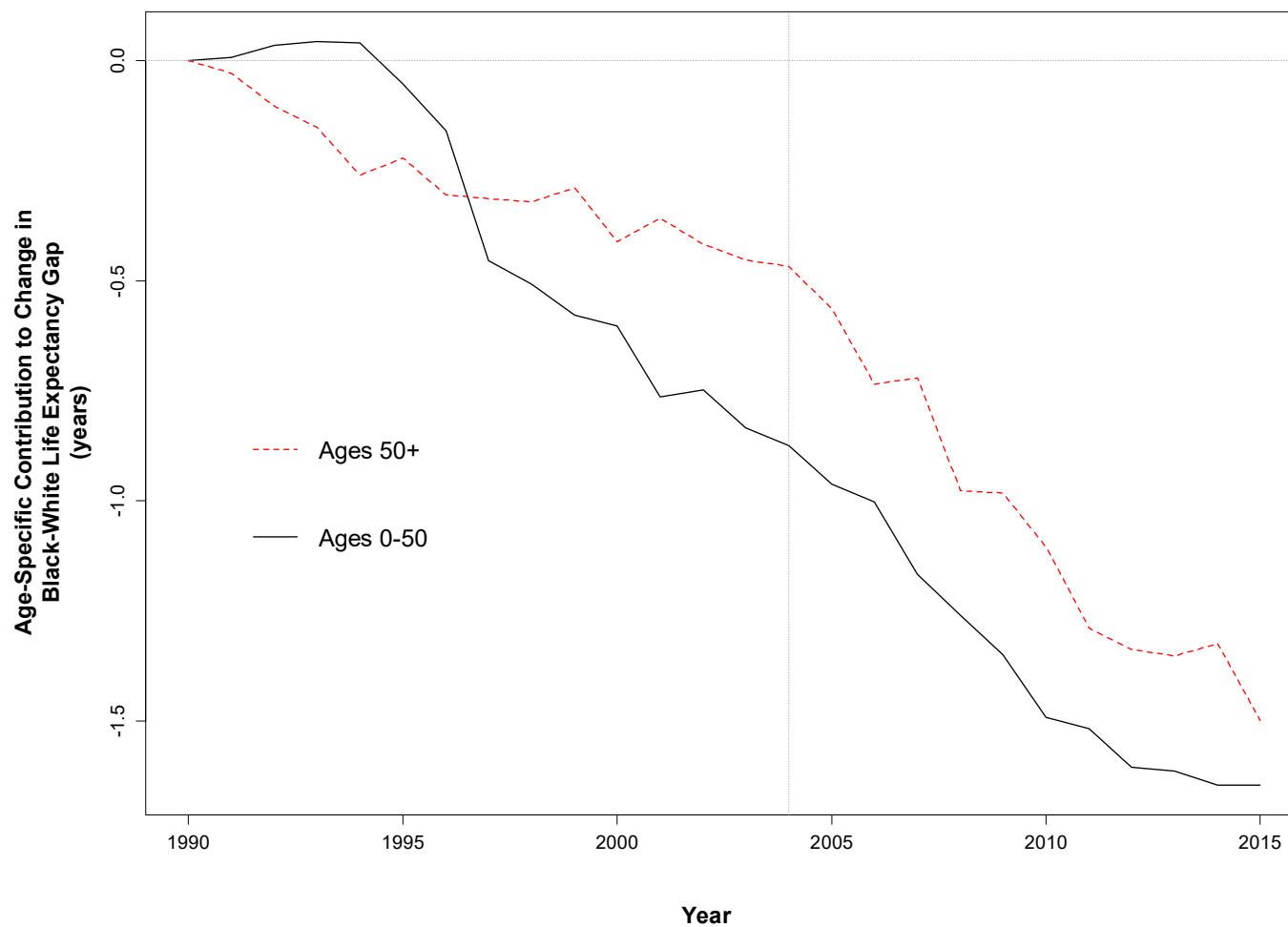**Figure 4**. Average Annual Rate of Increase in Life Expectancy by Race and Sex, 1990-2014

Figure 5. Contributions of Ages 0-50 and 50+ years to Changes in the Black-White Life Expectancy Gap, Men, 1990-2015

Note: Reference year is 1990, so that the contributions are to changes in the gap between 1990 and year t. The vertical dotted line is at 2004. Age-specific contributions were computed using Arriaga's decomposition.
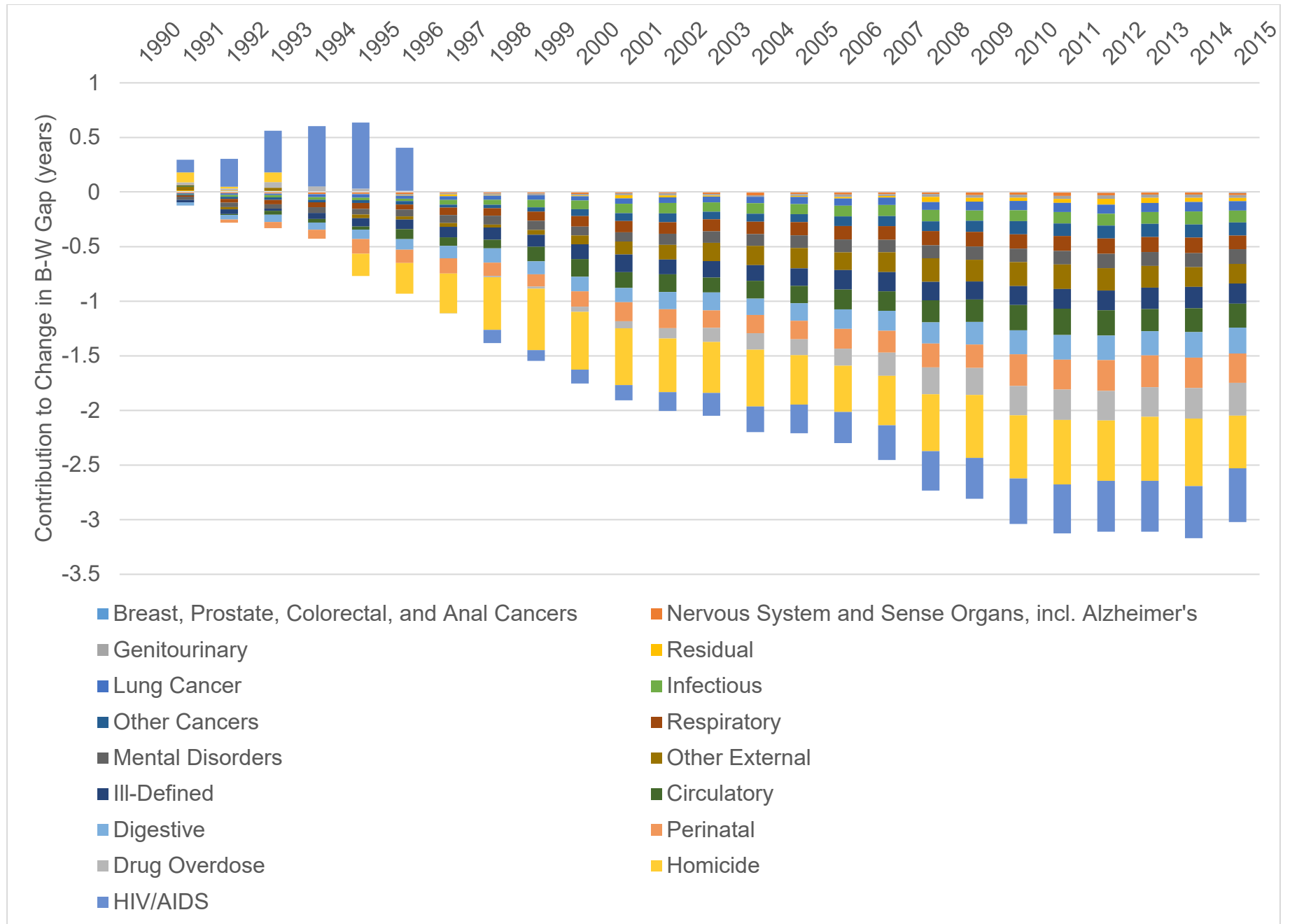
Figure 6. Contributions of Ages 0-50 and 50+ years to Changes in the Black-White Life Expectancy Gap, Women, 1990-2015
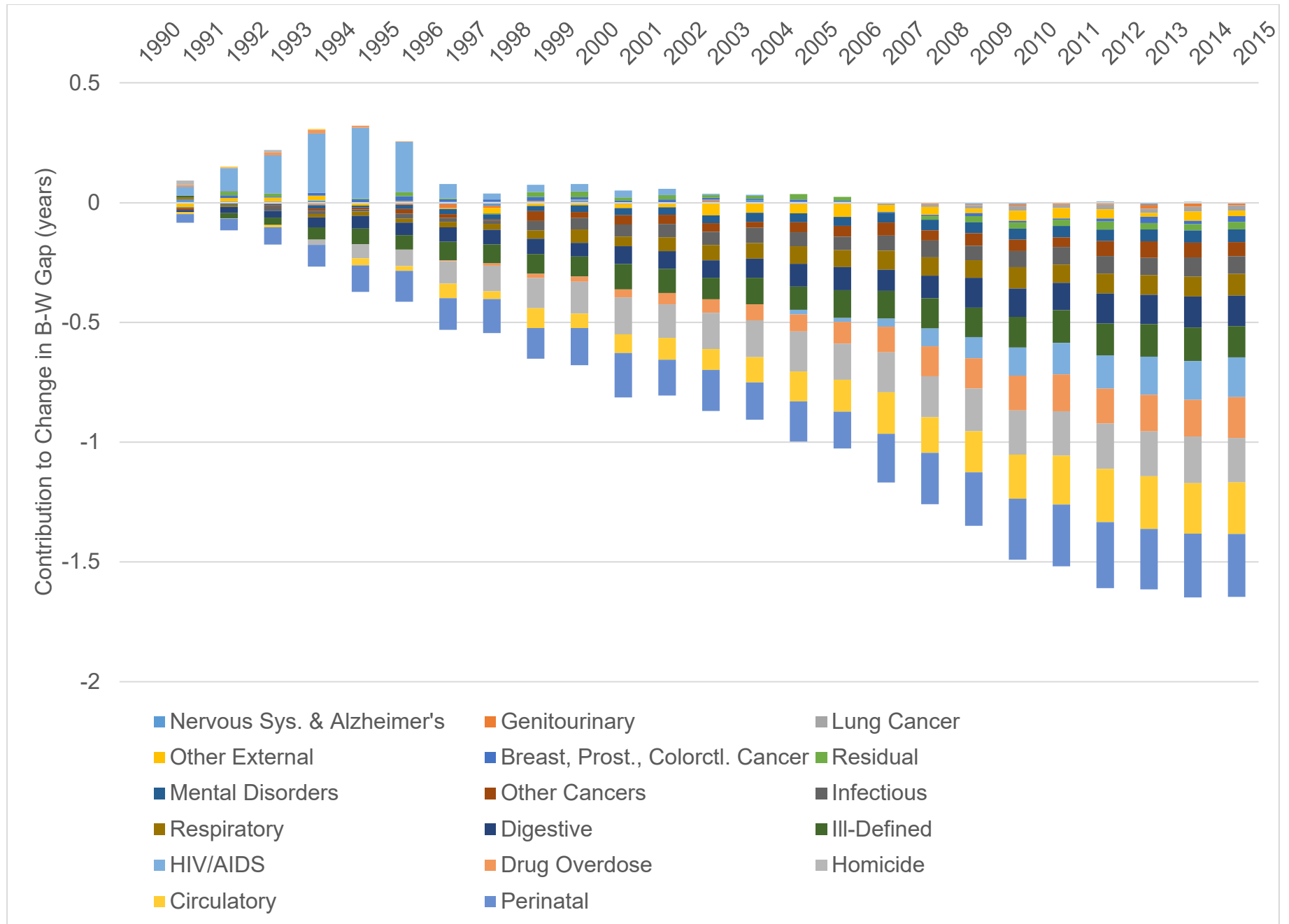

Note: Reference year is 1990, so that the contributions are to changes in the gap between 1990 and year t. The vertical dotted line is at 2004. Age-specific contributions were computed using Arriaga's decomposition.
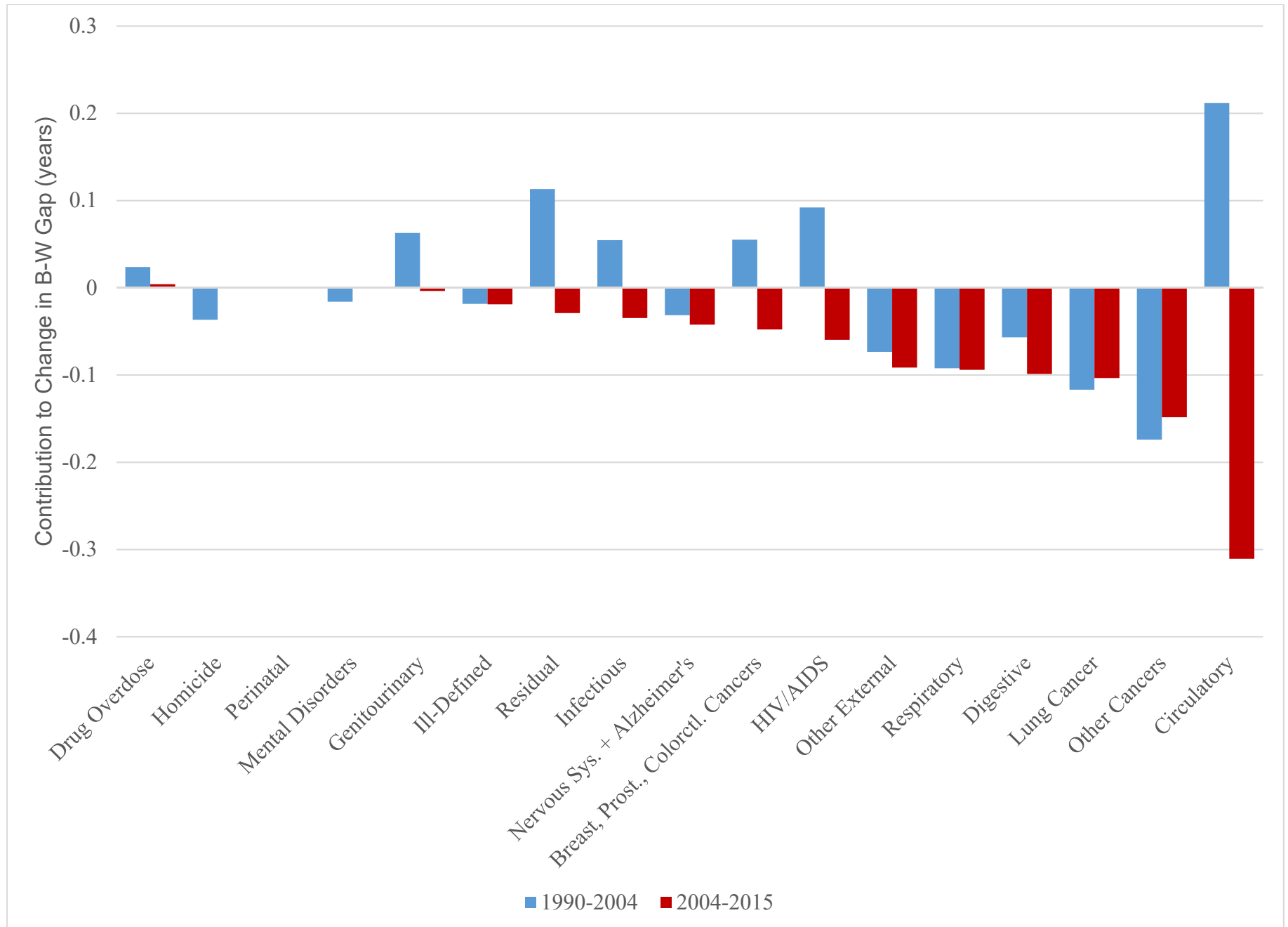
Figure 7. Causes of Death at Ages 0-50 Contributing to Narrowing Black-White Life Expectancy Gap, Men, 1990-2015
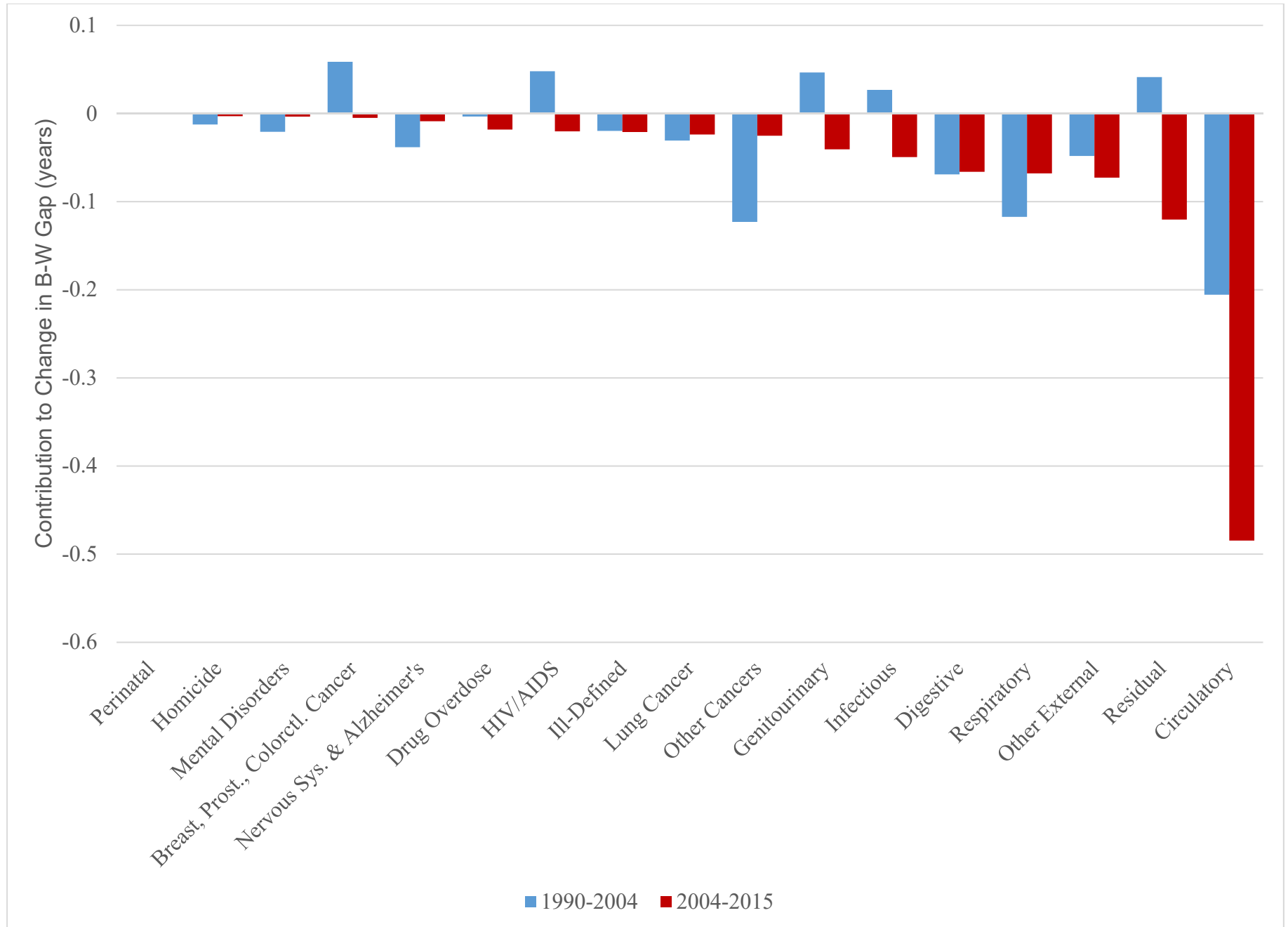
Figure 8. Causes of Death at Ages 0-50 Contributing to Narrowing Black-White Life Expectancy Gap, Women, 1990-2015

Figure 9. Causes of Death at Ages 50+ Contributing to the Narrowing Black-White Life Expectancy Gap, Men, 1990-2004 and 2004-2015

Figure 10. Causes of Death at Ages 50+ Contributing to the Narrowing Black-White Life Expectancy Gap, Men, 1990-2004 and 2004-2015

Appendix Table 1. Cause of Death Categories and Corresponding ICD-9 and ICD-10 Codes

Cause of death	ICD-9	ICD-10
1. HIV/AIDS	042-044	B20–B24
2. Other infectious diseases	001-139, excluding 042-044	A00–B99, excluding B20–B24
3. Lung cancer	162	C33, C34
4. Breast, prostate, colorectal, and anal cancer	174-175, 185, 153-154	C50, C61, C18–C21
5. All other cancers	140-239, excluding 174-175, 185, 153-154	C00–D48, excluding C33, C34, C50, C61, C18–C21
6. Mental disorders	290-319	F01–F99
7. Nervous system and sense organ, incl. Alzheimer's	320-389	G00–G98, H00–H57, H60–H93
8. Circulatory diseases	390-459	I00–I99
9. Respiratory diseases	460-519	J00–J98
10. Diseases of the digestive system	520-579	K00–K92
11. Diseases of the genitourinary system	580-629	N00–N98
12. Perinatal conditions	760-779	P00–P96
13. Homicide*	E960-E969, except E962.0	X86–Y09, Y87.1
14. Drug overdose	E850–E858, E950.0–E950.5, E962.0, E980.0–E980.5	X40–X44, X60–X64, X85, Y10–Y14
15. Other external causes	E800-E999, excluding E850–E858, E950.0–E950.5, E960-E969, E980.0–E980.5	V01–Y89, excluding X40-X44, X60-X64, X85–Y14, Y87.1
16. Symptoms, signs, and ill-defined conditions	780-799	R00–R99
17. All other causes	240-279, 280-289, 630-679, 680-709, 710-739, 740-759	E00–E90, D50–D89, G99, H58-H59, J99, O00–O99, L00–L98, M00–M99, N99, Q00–Q99, U00–U99, Y90-Y98

*except Assault by drugs, medicaments and biological substances