

## **“Outside the Skin”: The Persistence of Racial Disparities in Early Life Mortality**

Andrea M. Tilstra, University of Colorado Boulder, Iliya Gutin, University of North Carolina – Chapel Hill, Nathan Dollar, University of North Carolina – Chapel Hill, Richard G. Rogers, University of Colorado Boulder, Robert A. Hummer, University of North Carolina – Chapel Hill

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\*Direct correspondence to Andrea M. Tilstra, Population Program, IBS, 483 UCB, University of Colorado, Boulder, CO 80309-0483; e-mail: [Andrea.Tilstra@colorado.edu](mailto:Andrea.Tilstra@colorado.edu); telephone: 616-914-8850.

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## **“Outside the Skin”: The Persistence of Racial Disparities in Early Life Mortality**

### **Abstract** (max 300 words):

We augment current research surrounding rising extrinsic mortality among young- and middle-aged Americans by emphasizing the importance of race and racism as fundamental determinants of mortality early in the life course. We demonstrate that despite overall declines in early life mortality over the past three decades, black-white disparities in relative mortality have remained largely unchanged across a broad range of causes – especially those considered to be more “preventable” and/or “avoidable.” This paper documents trends in cause-specific early life mortality using data from the National Vital Statistics System (NVSS) Multiple Cause of Death files, which serve as the numerator, and population counts from the Surveillance, Epidemiology, and End Result Program (SEER), which serve as the denominator. We use age-standardized death rates to show how early life all-cause and cause-specific mortality has changed from 1990 to 2016 for sex- and race-specific groups of adolescents and young adults. Our findings show an early life black-white mortality ratio that never drops below 1.14 for women and 1.58 for men, and that in recent years has been increasing. This ratio is largely driven by higher rates of homicide mortality for black youth compared to whites. White youth have higher rates of suicide and transport mortality; there are no clear race differences in cancer mortality. We argue that addressing race/ethnic differences in early life mortality will require attention to systemic and institutional factors that operate “outside the skin” to acutely affect mortality.

### **Research Highlights** (3-5 bullet points)

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**Keywords:** Adolescence/Young Adulthood; Mortality; Homicide; Racism; Racial Disparities; National Vital Statistics System (NVSS) Multiple Cause of Death files

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## 1. Introduction

Mortality during adolescence and early adulthood (ages 15-24) poses a number of methodological and substantive challenges for researchers studying trends and disparities in population health. For one, this is a period of life defined by low mortality. The lack of any significant senescent or age-related processes accelerating death at these ages has led some to call this a quiescent phase in mortality wherein the only major threats to life are from external causes such as injuries and violence (Engelman et al. 2017), especially among males (Goldstein 2011; Remund et al. 2018). Furthermore, adolescence and early adulthood encompasses a segment of the U.S. population that has seen some of the most remarkable declines in mortality over past decades (Gore et al. 2011; Patton et al. 2009; Thakrar et al. 2018; Viner et al. 2011). Because of these dramatic declines in mortality and corresponding low numbers of deaths, mortality disparities in early life have received considerably less attention from population health scholars compared to other age groups.

Despite the positive trends in early life mortality, one disparity – which has perhaps been overshadowed by the recent emphasis on growing educational and income gradients in adult mortality (Case and Deaton 2015; Chetty et al. 2016; Sasson 2016) – is the persistent racial gap across virtually all ages and causes of death in early life mortality (Xu et al. 2018). Black-white disparities in healthy life expectancy and mortality continue to define U.S. population health (Hummer and Chinn 2011; Kim and Miech 2009; Levine and Crimmins 2014; Levine et al. 2016; Masters et al. 2014; Olshansky et al. 2012; Williams and Jackson 2005), and are a significant contributor to the relatively low U.S. life expectancy relative to other wealthy nations (Avendano and Kawachi 2014; National Research Council [NRC] 2013). However, only relatively recently have researchers more formally sought to implicate racism – that is, one’s racialized experience in a society – as a fundamental cause of health and mortality disparities in the U.S., where historical and contemporary institutionalized racism continues to define black-white relations across age, gender, geography, and socioeconomic status (Hicken et al. 2018; Phelan and Link 2015).

To date, though, most work on racism and health conceptualizes racism as an all-encompassing and lifelong institution, and in turn focuses on the cumulative mechanisms by which racism operates (Hicken et al. 2018; Phelan and Link 2015). Namely, these life course perspectives argue that persistent exposure to racial discrimination – at both the institutional and individual levels – manifests in multiple dimensions of one’s life, compounds over the life course, and deteriorates minorities’ health over time. In other words, U.S. racism impinges on the health and longevity of minorities through this cumulative process where racism operates by getting “under the skin” (Ferraro and Shippee 2009; Geronimus et al. 2010). Although the large body of research documenting the cumulative processes through which racism gets “under the skin” helps explain black-white disparities in health and longevity later in life, it is less useful for understanding racial disparities in mortality at younger ages. This is especially the case when we consider the 15-24 age group, which captures a critical turning point in the life course where youth establish some independence from their parents and family (Shanahan 2000). This newfound independence brings increased exposure to and risk of death from external causes. Indeed, most deaths in early life are due to external and, to a large extent, preventable causes (e.g. transport accidents, suicide, homicide, and drug poisonings) (Heron 2017). Thus, we seek to augment the current research surrounding rising extrinsic mortality among young- and middle-aged Americans by emphasizing the continued importance of racism as a fundamental determinant of mortality early in the life course.

In this paper we analyze black-white disparities in all-cause and cause-specific mortality among adolescents and young adults (ages 15-24) between 1990 and 2016. We show persistent disparities in all-cause mortality and clear differences in cause-specific external mortality between black and white adolescents and young adults. Additionally, we show that compared to white adolescents, black adolescents are more likely to die from homicides but less likely to die from suicides and transport accidents. There are no clear race differences in cancer deaths. These findings provide evidence that the pernicious effects of racism on minority health begin “outside the skin” and operate as a more acute source of risk at younger ages, rather than as a cumulative or chronic source of health disparities as they do for adults. However, similar to older adults, the racial disparities observed in these largely external causes of death point to a similar set of systemic and institutional determinants – such as individuals’ social, economic, and physical environments – that structure minorities’ exposure to risk and subsequent mortality.

## **Background**

### *Racism as a Fundamental Cause of Disparities in Mortality*

Recent decades have witnessed a considerable narrowing of black-white disparities in health and life expectancy (Xu et al. 2018) – likely due to the combined effects of improvements in socioeconomic opportunity and standing among the black population (Fuchs 2016; Masters et al. 2014; Riddell et al. 2018), coupled with worsening health among white adults (Case and Deaton 2015; Montez and Zajacova 2013; Sasson 2016). Yet we continue to observe sizable black-white differences in health and mortality across virtually all ages (Hummer and Gutin 2018; Williams and Mohammed 2009; Xu et al. 2018), and the ability to close this *persistent* gap in coming years remains uncertain (Olshansky et al. 2012). In an effort to explain the presence of these stark and sustained racial health disparities, recent scholarship has sought to define racism – as embodied in individuals’ racialized experiences in a society where institutional racism is rampant – as a distinct and fundamental cause of disparities in health and mortality throughout the entirety of U.S. history (Hicken et al. 2018; Phelan and Link 2015). Racism has taken on many forms over time – ranging from the more obvious and formalized institutions of slavery, Jim Crow legislation, and pre-Civil Rights era second-class citizenship status to the more subtle and coded institutions of color-blind and embedded systemic racism observed in the U.S.’ financial, education, housing, criminal justice, legislative, and medical institutions (Bonilla-Silva 2017; Reskin 2012). Racism lingers in the U.S. through white supremacy and limited opportunities for the black minority. Consequently, racism has remained relentless in its harmful effect on the health of the black population (Phelan and Link 2015).

A key premise of the fundamental cause framework is that a fundamental cause operates consistently over time *as well as* through a diverse set of health pathways (Link and Phelan 1995; Phelan et al. 2004). This is in part evidenced by sizable black-white disparities across multiple causes of death in the U.S. (Hummer and Gutin 2018; Xu et al. 2018). Research on the health pathways of racism emphasizes cumulative (dis)advantage as the driving force underlying how and why racism has been a persistent source of racial disparities in mortality. This framework highlights how racism operates through the mechanisms of sustained socioeconomic disadvantage and the harmful consequences of everyday discrimination. For instance, greater educational attainment, especially a college degree, does not offer the same health advantages for blacks as whites, and this

disparity is only compounded with age (Shuey and Wilson 2008). Similarly, accumulated income and wealth disparities over the lifecourse – despite their having the same health benefit for blacks and whites (Shuey and Wilson 2008) – account for a substantial portion of racial disparities in health and mortality (Kahn and Fazio 2005; Lynch 2008; Pais 2014). Independent of socioeconomic status, a large and growing body of research speaks to the cumulative health toll of chronic stress attributable to everyday experiences of racial discrimination (Jackson et al. 2010; Jackson et al. 2011; Williams and Mohammed 2009; Williams and Sternthal 2010; Williams et al. 2003). This repeated psychosocial trauma has very real consequences for physical health, as the resulting systemic inflammation and cardiometabolic dysfunction account for shorter and less healthy lives, beset by more chronic disease and poor mental health, among the black population.

Further compounding the cumulative harm inflicted by chronic stress and systematic disadvantage is the fact that medical and public health advances and interventions, as a key source of improvements in chronic disease mortality over past decades (Ma et al. 2015), exhibit considerable racial inequality in their impact on black-white disparities in mortality. Specifically, extant research testing the conceptual and empirical validity of fundamental cause theory often examines disparities in mortality on the basis of how *preventable* a certain cause of death is. Preventability is determined by the stochasticity, potential for treatment/intervention, and the extent to which the cause of death could have been avoided to begin with, as a function of social, behavioral/lifestyle, and other human created – rather than biological or genetic – factors (Beltran-Sanchez 2011). Just as more preventable causes of death (e.g., heart disease and lung cancer) often exhibit wider SES gradients in mortality (Miech et al. 2011; Masters et al. 2015; Phelan et al. 2004; Rubin et al. 2014), the research extending this approach to the study of racial disparities in mortality consistently finds that causes of death most closely linked to the availability of key medical and public health resources like new knowledge, screening/testing protocols, specific drugs or other forms of treatment (e.g., HIV/AIDS, breast and colorectal cancer) have large, and sometimes widening, black-white disparities (Levine et al. 2010; Rubin et al. 2010; Soneji et al. 2010; Tehranifar et al. 2009; Tehranifar et al. 2016). While research examining the preventability of mortality in the context of race and racism is not as expansive as that for SES, the causes of death identified thus far plainly demonstrate how a more equitable racial distribution of life-saving medical knowledge and resources – which are currently impeded by evidence of systemic racism within medical and health institutions (Phelan and Link 2015; Williams and Mohammed 2009) – could avert thousands of premature deaths.

### *The Importance of Early Life Mortality*

A key limitation of this nascent, yet critical, body of research on racism as a fundamental cause of mortality is the overwhelming focus of both theory and evidence on mechanisms and pathways to health and mortality that do not manifest until later in adulthood. With the exception of infant mortality (Alexander et al. 2008; Rossen and Schoendorf 2014), and only a few studies of child health (Mehta et al. 2013; Turney et al. 2013), our descriptive knowledge of the persistence of racial disparities over time is largely limited to overall life expectancy and adult mortality rather than how these trends have unfolded in early life, let alone across distinct causes of death prevalent at these ages. Relatedly, the effects of the *cumulative* mechanisms singled out as contributing to black-white disparities in health are unlikely to have a measurable impact on mortality until later (Warner and Hayward 2006). This is not to suggest that racism and discrimination do not have a direct effect on health and mortality in early life; indeed, emerging research suggests exposure to

and encounters with racism are an important source of health disparities at these ages. However, the effects are largely confined to psychological health, and the few observed associations with physical health are not severe enough to account for disparities in mortality (Heard-Garris et al. 2018). Likewise, the preventability framework for understanding black-white disparities in mortality has largely focused on *cumulative* chronic disease mortality, along with causes of death most directly amenable to medical intervention. Yet preventability takes on a substantially different meaning in early life wherein the focus is not on preventability as a function of medical care and intervention – as is the case with treating and/or preventing the onset of chronic diseases. Instead, early life preventability is better conceptualized as the relative *safety* and *stability* of one’s social environment in helping to avoid mortality from external causes, many of which are considered to be equally if not more preventable than later-life chronic disease mortality (Beltran-Sanchez 2011; Elo et al. 2014; Miech et al. 2011).

In sharp contrast to the cardiovascular disease, diabetes, and lifestyle-related cancers that dominate mortality at older ages, the leading causes of mortality among adolescents and young adults are almost exclusively external, with accidents, assaults, unintentional injuries, and suicides accounting for the majority of deaths at these ages (Heron 2017; Khan et al. 2018). While sociologists, demographers, and public health scholars have long-emphasized the importance of external causes of death in discussions of U.S. mortality trends (Ho 2013; Miech et al. 2011; NRC 2013), only recently have we observed the national- and population-level impact of increased mortality attributable to these causes occurring earlier in the life course (Case and Deaton 2015; Kochanek et al. 2016; Xu et al. 2018), with the sharp increase in young adult and mid-life mortality accounting for declining U.S. life expectancy over the past three years. Unsurprisingly, these already elevated and increasingly rising external cause mortality rates at younger ages have also been singled out as a key explanation for the U.S.’ poor international performance with respect to population health. While the U.S. often emerges as a world-leader with respect to higher survival rates at older ages for cancer and other causes of where medical technology and innovation is imperative (Glei et al. 2010; Ho and Preston 2010), childhood and young adult health is generally worse in the U.S. than its peer countries (Khan et al. 2018). International comparisons have specifically singled out early life mortality from external causes as a key driver of the U.S.’ relatively poor international standing on life expectancy and other measures of population health (Ho 2013; NRC 2013). Indeed, nearly two-thirds of the male life expectancy gap between the U.S. and other wealthy, industrialized nations, and two-fifths of the female gap, is attributable to its significantly elevated mortality below age 50, across a broad range of external causes of death including homicides, drug overdoses, motor vehicle accidents, and other accidental injuries (Ho 2013).

Notably, large disparities in *who* experiences the highest mortality rates across these different external causes of death helps to further account for the U.S.’s status as a laggard in both early life and adult health and mortality. For instance, despite some similarities in leading causes of death among black and white adolescents and young adults, assault-related deaths (consisting primarily of homicides) only account for approximately four percent of deaths among white women and men ages 15-24, but are the *leading* cause of death among similarly-aged black women and men, accounting for over 40% of deaths (Heron 2017). Similarly, the few extant studies of racial disparities in early life mortality find significantly elevated rates of external mortality for black adolescents and young adults as compared to their white counterparts, with the most dramatic differences observed among the types of preventable causes of death most closely associated with violence and social disorganization (Khan et al. 2018; Singh and Kogan 2006; Rogers et al. 2017;

Singh and Yu 1996). Speaking to the black-white gap in the *types* and *preventability* of deaths driving early life and overall mortality, a counterfactual analysis by Woolf et al. (2004) reached the striking conclusion that “resolving the causes of higher mortality rates among African Americans can save more lives than perfecting the technology of care” (p. 2080). Specifically, the hypothetical number of averted deaths between 1990 and 2000 attributable to secular (or medical) improvements in age-specific mortality was only *one-fifth* the number of averted deaths attributable to equivalent mortality rates among black and white adults. Consequently, as we continue to observe overall improvements in U.S. early life mortality (Khan et al. 2018), we should also be cognizant of the extent to which these absolute declines obscure the persistence of black-white disparities, which is our overall goal of the present study.

### *Current Aims*

In light of the above research, our goal is not to emphasize the potential shortcomings of fundamental cause theory as extended to race and racism; rather it is to demonstrate the *robustness* of this framework even at younger ages, when the population is relatively healthy and exhibits low mortality. Early life, and young adulthood in particular, is a unique and important part of the life course, during which race is a particularly salient determinant of health, shaping the social and contextual factors that influence health and mortality risk. Rather than conceptualizing the “fundamental” aspects of racism as always reflected in chronic and cumulative conditions as at older ages, it is important to consider how these same fundamental social processes – such as residential segregation, ineffective policy and legislation, and inadequate access to health and medical care (Williams and Jackson 2005) – are instead observed across causes of death more concentrated in early life. Namely, the increasingly held view that racism is indeed a fundamental cause of black-white disparities in mortality strongly suggests that our analyses will demonstrate consistent racial disparities over time and across a diverse set of preventable causes of death dominant earlier in the life course. Thus, even at younger ages – where accidental or unintentional mortality is largely the product of *acute*, rather than *chronic* and *cumulative*, processes originating from “outside” the body– we anticipate clear black-white differences in mortality, consistent with the experience of racism as a product of racial disparities in social circumstances, resources, and broader forms of institutional discrimination and segregation.

Consequently, we expect that, despite overall declines in early life mortality over past decades, black-white relative disparities in mortality have remained largely unchanged across a broad range of causes – especially those considered to be more “preventable” and/or “avoidable” (Elo et al. 2014). Specifically, we highlight how rates for all-cause mortality, four leading external causes of death, and one internal cause of death vary over time for young non-Hispanic black and white men and women, ages 15-24. Additionally, we show how life expectancy at birth ( $e_0$ ) would change for black men if homicide mortality – the leading external cause of death for young black men – declined or was eliminated. By highlighting the persistent disparities in external causes of death, we argue that more theoretical attention should be given to understanding racial disparities in early life mortality, thus helping to confirm existing theories of race and racism as a fundamental cause of health and mortality.

## **2. Data & Methods**

We use data from the National Vital Statistics System (NVSS) Multiple Cause of Death files (National Center for Health Statistics [NCHS] 2016) and population counts from the Surveillance, Epidemiology, and End Result Program (SEER) for the years 1990-2016. NVSS mortality data are collected by NCHS and encompass all individuals who die within the United States. SEER population counts are produced by the U.S. Census Bureau and are standardized across time by SEER. The data include age-, race/ethnic-, and gender-specific U.S. population estimates from the midpoint of each year, July 1. We restrict our analyses to U.S.-born non-Hispanic white and non-Hispanic black adolescents and young adults aged 15-24. All together, there are a total of 705,801 deaths for black and white individuals aged 15-24 in the analytic dataset, across the 27-year time frame.

The analysis first focuses on all-cause mortality and then on five specific causes of death. We use the underlying cause of death specified on the NVSS file for cause-specific analyses. We first analyze four external causes: transport accidents, homicide, suicide, and drug-related deaths. Unintentional injury deaths are the leading cause of death for youth, and transport accidents comprise the majority of these deaths (Heron 2017; NCHS 2018). In 2015, suicide was the second and homicide was the third leading cause of death for youth (Heron 2017). Drug-related mortality has been increasing in recent years for both blacks and whites, and while rates are highest in midlife, younger age groups are also adversely affected (Alexander et al. 2018; Masters et al. 2017). These four causes of death are highly preventable, based on their amenability to social, environmental, and/or behavioral intervention (Beltran-Sanchez 2011). However, there are clear racial and gender differences in mortality rates among these four causes of death (Heron 2017). We also analyze cancer as the leading non-external cause of death for this age group (Heron 2017). Critically, the well-established non-treatable nature of cancers at these ages (e.g., brain cancer, leukemia) serves as a contrast to the external causes described before. We standardize across ICD-9 and ICD-10 and code *homicide* (ICD9: 960-969; ICD10: X85-Y09, Y87.1), *suicide* (ICD9: 950-959.9; ICD10: U03, X60-X84.9, Y87.0), *drug-related deaths* (ICD9: 304, 850-858, 962, 980-980.5; ICD10: F11-F16, F19, X40-X45, X85, Y10-Y15), *transport accidents* (ICD9: 800-848, 929.0, 929.1; ICD10: V01-V99, Y85), and *cancer* (ICD9: 140-208, ICD10: C00-C97), respectively.

First, we estimate single-year age-standardized mortality rates by sex, race/ethnicity, and cause of death. Analyses for all-cause and cause-specific deaths include ages 15-24, and race/ethnic- and sex-specific age distributions for the entire study time period, are used in the calculation of the age-standardized rates. We also show the black-white mortality ratio, separately by young men and young women, to examine the persistence of these racial disparities in recent years. Second, we estimate associated single-decrement life tables (ASDT) for the year 2016 to assess what life expectancy at birth ( $e_0$ ) would be for black men if homicide deaths were either eliminated or reduced. Initial analyses highlight homicide as the driving force of black-white disparities at these ages and over time; thus, we believe it is the most salient cause of death to use in this counterfactual example. To calculate ASDTs, we first estimate  $e_0$  for all-cause mortality for both black and white men, then we: (1) estimate  $e_0$  in the absence of cause of death  $i$ ; (2) estimate  $e_0$  in the absence of cause of death  $i$  in *just the sample age range* (ages 15-24); (3) estimate  $e_0$  if the cause were reduced by  $\Delta_{i,j}$ , where  $\Delta_{i,j}$  is the risk ratio between white and black mortality for cause of death  $i$  in each age group  $j$ ; and (4) estimate  $e_0$  if the cause were reduced by  $\Delta_{i,j}$  in *just the sample*



age range (ages 15-24). We use the Chiang method, the most widely accepted method for estimating life expectancy from these data (Chiang 1984).<sup>1</sup>

### 3. Results

Figure 1 shows all-cause age-adjusted mortality rates for black and white men and women, from 1990 to 2016. Rates for adolescent black men declined rapidly across the 1990s, followed by stalled improvements across the 2000s and 2010s; between 2014 and 2016, rates rapidly rose from 140 to 172 deaths per 100,000. Conversely, rates declined modestly for black and white women and for white men until the early 2010s, when improvements in overall mortality rates stalled. Like black men, mortality rates for these four groups rose between 2014 and 2016. To better compare racial trends, Figure 2 shows the large and consistent black-white mortality ratio for young men and women over this time period. Specifically, the ratio for men increased until 1993, when it reached a peak of 2.66 and then decreased quite drastically until 2011, reaching a low of 1.58. The ratio followed a similar pattern for women, though the fluctuation was more modest. Notably, there was also a slight increase in the ratio for women between 2007 and 2008, from 1.17 to 1.32. Reflecting the lack of continued declines in mortality rates for both men and women, the black-white mortality ratio did not decline after 2011. In fact, the ratio for men rose dramatically between 2014 and 2015 (from 1.58 to 1.73), followed by modest declines between 2015 and 2016; the ratio for women, on the other hand, has been increasing since 2013.

*[Figures 1 & 2 about here]*

Figure 3 reveals differences in cause-specific mortality rates for black and white men and women. Though the rates of homicide have declined for all four groups, young black persons continue to have much higher rates of homicide mortality than their white counterparts. In 2016, the rates for black and white men were 85.90 and 4.40 deaths per 100,000, respectively, and the rates for black and white women were 9.81 and 1.84 deaths per 100,000, respectively. In the same year, the black-white mortality ratio for homicide deaths was in 19.5 for men and 5.33 for women. Conversely, white males and females have higher rates of transport mortality, though these rates are also improving for all four groups. Suicide rates are higher for white than black adolescents and young adults. These improvements have been minimal over time for all groups, with some worsening since 2014, mirroring trends among older adults (Curtin et al. 2016). Relative rates of these causes have changed over time and across groups. Notably, homicide rates have dropped dramatically for black females, and are now lower traffic death rates (2016: 9.51 and 10.88 deaths per 100,000, respectively). For white men and women, as transport mortality declines and suicide mortality increases, the two causes have converged. In 2016, transport and suicide mortality rates were identical for white men, 24.30 deaths per 100,000, and for white women they were 10.45 and 6.07 deaths per 100,000, respectively.

*[Figure 3 about here]*

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<sup>1</sup> We also estimate ASDT using linear and exponential life table methods. Results were nearly identical across approaches.

Trends in drug-related mortality also mirror that observed in older populations (Alexander et al. 2018; Masters et al. 2017), where rates for all populations increased beginning in the late 1990s – especially so for white men (Figure 4). The most rapid increase in drug-related mortality for young black and white men began in 2013. Between 2013 and 2016, there has been a 50% increase in drug mortality for young white men (15.98 to 24.00 deaths per 100,000), and, although much lower overall, the rate for black men has increased by 144% (3.28 to 8.00 deaths per 100,000). Rates for young women have also increased rapidly since 2013, but not as quickly as for men. There has been a 44% increase for white women (6.24 to 8.97 deaths per 100,000) and a 127% increase for black women (1.68 to 3.81 deaths per 100,000). In contrast to these external causes, Figure 5 presents cancer mortality death rates for this age group. Unlike, homicides, motor vehicle accidents, suicides, and drug poisonings, we see that trends in cancer deaths have been declining modestly – albeit consistently – over time and lack clear racial disparities. Women do have consistently lower rates than men across this time period, and although white women appear to have the lowest rates, there are several years where rates actually cross with those for black women.

*[Figures 4 & 5 about here]*

Given the central role of homicide mortality as a driving force of black-white mortality disparities at these ages – for both men and women – we consider how life expectancy might differ for black and white U.S. adults under a series of counterfactual scenarios about hypothetical changes to black homicide mortality in early life. Figure 6 shows results from the associated single decrement life tables in which black men’s homicide rates are changed to reflect these assumptions, and Figure 7 shows the same for black women’s homicide rates. In light gray we see actual life expectancy at birth ( $e_0$ ) in 2016 for white and black men, at 76.33 and 71.95 years, respectively. The subsequent lines then show how black men’s  $e_0$  would have improved with each of the four ASDT approaches. If black men had the homicide rate of white men in early life, we would expect their  $e_0$  to be 72.05 years (solid black), but if black men had the homicide rate of white men at all ages their  $e_0$  would be 72.13 years (solid red). In the absence of all homicide deaths in early life, we would expect black men’s  $e_0$  to be 72.48 years (checkered), and, finally, in the absence of all homicides, their  $e_0$  would be 73.21 years (dark gray). That is to say, under the best possible circumstances – the elimination of black homicide – the gap in life expectancy at birth between black and white men would decrease by 1.26 years.

*[Figures 6 & 7 about here]*

#### **4. Discussion**

The considerable black-white disparities in adult mortality are an unfortunate reality of population health in the United States (Hummer and Gutin 2018; Xu et al. 2018), and evidence increasingly situates the origins of these disparities earlier in the lifecourse (Khan et al. 2018; Rogers et al. 2017; Singh and Kogan 2006), at ages typified by good health and high survival. The persistence of these disparities – over time, via multiple mechanisms, across many outcomes, and, as we argue, throughout the lifecourse – has motivated scholars to theorize racism as a fundamental cause of health disparities in the U.S. (Phelan and Link 2015). As a reflection of the systemic, institutional, and intrapersonal mechanisms limiting the advancement of black men and women relative to their

white peers, racism proves pervasive in both society and in its adverse effect on health and mortality. Consequently, most extant research underlying racism as a fundamental cause has focused on the *cumulative* mechanisms through which it operates to get “under the skin” of black adults, such as accumulated social disadvantage and chronic stress and discrimination (Jackson et al. 2011). Yet we contend that these cumulative mechanisms do not adequately characterize racial differences in mortality in early life where disparities are best understood as a function of the kinds of acute exposures and events that drive mortality at these ages – i.e., those causes occurring “outside the skin.” To assess this argument, we analyze overall and cause-specific disparities in mortality among non-Hispanic black and white youth, aged 15-24, for the years 1990-2016. By examining single-year age-standardized mortality rates for all-cause and five distinct causes of death (homicide, suicide, transport- and drug-related, and cancer), as well as gender specific black-white mortality ratios for all-cause mortality, we document cause-specific trends underlying overall disparities at these ages. Furthermore, we use associated single-decrement life tables to estimate hypothetical changes to life expectancy at birth among black men in the absence or reduction of homicide rates, as the dominant cause underlying disparities at these ages.

There are five major findings from our analyses. First, although the black-white mortality ratio for both males and females improved across much of the study period, the ratio has never dropped below 1.14 for women and 1.58 for men, and instead increased in recent years. Second, this persistent ratio is almost entirely driven by homicide rates for blacks, which are nearly 20 times higher and 6 times higher compared to white males and females, respectively. In fact, if homicide mortality were eliminated entirely for black men, we would see an improvement in their life expectancy at birth of nearly 1.3 years. Third, we find that, across the study time period, suicide rates are consistently higher among white males and females than their black counterparts, consistent with literature on the racial “suicide paradox,” in reference to lower black suicide rates despite their lower social standing (Rockett et al. 2010). Fourth, similar to patterns observed among older populations (Alexander et al. 2018; Masters et al. 2017), we find that drug-related mortality is increasing rapidly for all four groups; though adolescent and young adult white males have seen the most sustained and steepest growth over this time period, recent years have seen increases for black men and women as well – largely undocumented in extant research. Fifth, unlike these primarily external causes of death, we find no clear racial differences in cancer mortality at these ages, as the modest decline in rates over time is true for all race-gender groups in our analysis.

Undoubtedly, extending the fundamental cause perspective to be inclusive of racism provides a powerful and parsimonious framework for understanding black-white disparities in U.S. health and mortality, emphasizing the systemic and intrapersonal racism rampant in the U.S. that so negatively impacts the health of its black population (Phelan and Link 2015). Although most research underscores the cumulative mechanisms by which racism gets “under the skin” to influence adult health and mortality, our study is among the first to emphasize the significance of external mechanisms – acting “outside the skin” – as the primary source of racial disparities in mortality in early life. Specifically, our findings demonstrate that among the leading causes of death for adolescents and young adults, racial disparities are most apparent for those causes external to the body – namely, homicide, suicide, transport- and drug-related mortality. While not all of the cause-specific racial disparities favor white youth, such as their higher suicide and similar transport-related rates relative to young black men and women, the net effect is a persistent black disadvantage in mortality over time, emblematic of the “*multiplicity of mechanisms*” and “*systematic asymmetry*” by which a fundamental cause negatively influences health (Freese and Lutfey 2011). The absence of racial disparities in cancer deaths at these ages further highlights the

lack of evidence that racism truly gets “under the skin” in early life, as there is less time for the biological effects of racism to be experienced and manifest as “chronic” morbidities.

The differing patterns of racial disparities observed across homicides, suicides, transport-, and drug-related deaths as compared to cancer also underscore the importance of the *preventability* of death at these ages, and how we use this knowledge to best address racial disparities in early life mortality. Brain cancer, leukemia, lymphoma, and other cancers of the neurological and immune systems most prevalent at these ages are, for the most part, randomly occurring conditions; though survival rates have steadily improved over time – as seen in our results – their survival rates are still far lower than many cancers common at older ages (Howlader et al. 2017). The difficulty of both prevention and treatment render this a category of deaths with *low-preventability* at these ages. While racism is prevalent in medical institutions and care, the lack of clear racial disparities in early life cancer mortality mirrors the lack of racial disparities among other hard-to-treat and/or prevent cancers at older ages (Tehraniifar et al. 2009; Tehraniifar et al. 2016).

Critically, these findings place greater emphasis on the external causes of death examined in our study, indicating the more immediate, acute, and *preventable* pathways through which racism affects the health and wellbeing of young Americans. We single out homicide as the most significant pathway preserving racial disparities in mortality at these ages, as young black individuals, especially males, continue to have homicide rates an order of magnitude higher than their white counterparts. Exposure to violence is much higher for young blacks than whites (Crouch et al. 2000), and research suggests that race/ethnic disparities in homicide mortality can be partially explained by the increased propensity for violent and/or criminal activity catalyzed by economic deprivation and social disorganization (Kennedy et al. 1998; Krueger et al. 2004) – which are, themselves, the product of structural racism. This structural racism also manifests in the concentration of black Americans in low-resource, low-opportunity neighborhoods (Massey et al. 1994), which negatively impact black health and mortality through acute pathways engendered by persistent social disadvantage, often across multiple generations (Goosby and Heidbrink 2013).

Indeed, racism operates somewhat differently as a fundamental cause of health disparities in early life than traditionally conceptualized under fundamental cause theory. As seen in our analyses, racism largely operates through a *single, acute* mechanism (i.e., homicide) to maintain racial disparities in mortality in early life, rather than through *many* mechanisms and through *cumulative* (dis)advantage as it does in later life. However, the acute and preventable processes through which racism adversely affects health and mortality in adolescence and young adulthood are preventable and thus amenable to interventions, making this an important place for policy work. Though points of potential intervention are not drastically different than those highlighted in fundamental cause theory and racial disparities literature in adulthood – such as “reduc[ing] racial differences in SES, in neighborhoods, in freedom, in power and prestige, in health care” (Phelan and Link 2015: 325) - it is important to consider which interventions are most consequential for early life health and mortality. Reducing racial disparities in health care access and availability is less critical at these ages; conversely, subverting the institutional and intrapersonal discrimination that limits the socioeconomic and geographic mobility of young black men and women may help to break the cyclical patterns of violence that continue to account for the overwhelming majority of black-white disparities in mortality at this critical stage in the life course. Consequently, further public policy attention ought to be given to gun laws and neighborhood disadvantage, and how their design and/or implementation implicitly channels systemic racism writ large, to adversely affect racial/ethnic minorities.

Our analyses are not without limitations. For example, we do not consider competing risks in our ASDT analyses. That is, we do not statistically consider that someone who would have died from homicide might be at a greater risk of dying early from a different cause of death. Additionally, our analyses are not multivariate, so we are unable to determine what specific contributors (e.g. socioeconomic status, neighborhood effects, crime, emergency response time) might affect observed racial differences. Nevertheless, our results serve as an entrée into future work in this area using survey datasets that cover this age range and are able to track health and/or mortality (e.g., NHIS-LMF). Although results must be interpreted with these limitations in mind, our results suggest that differences in specific external causes of death are driving observed black-white differences in adolescent and young adult mortality. Because these causes of death originate outside of the body – and largely represent ‘acute,’ rather than ‘chronic’ and/or cumulative mechanisms – it is important to consider the unique pathways by which race and racism impact mortality for this age group. By demonstrating that racial disparities are largely attributable to differences in preventable or avoidable sources of mortality related to young peoples’ *social, behavioral, and policy* environments (Elo et al. 2014) – such as unsafe neighborhoods, exposure to violence, or firearm-related policies – we argue that addressing racism as a determinant of mortality disparities at younger ages requires a greater focus on reducing the *immediate* rather than *long-term* consequences of racism on health. Interventions targeting systemic – rather than individual-level racism – are liable to have a greater impact.

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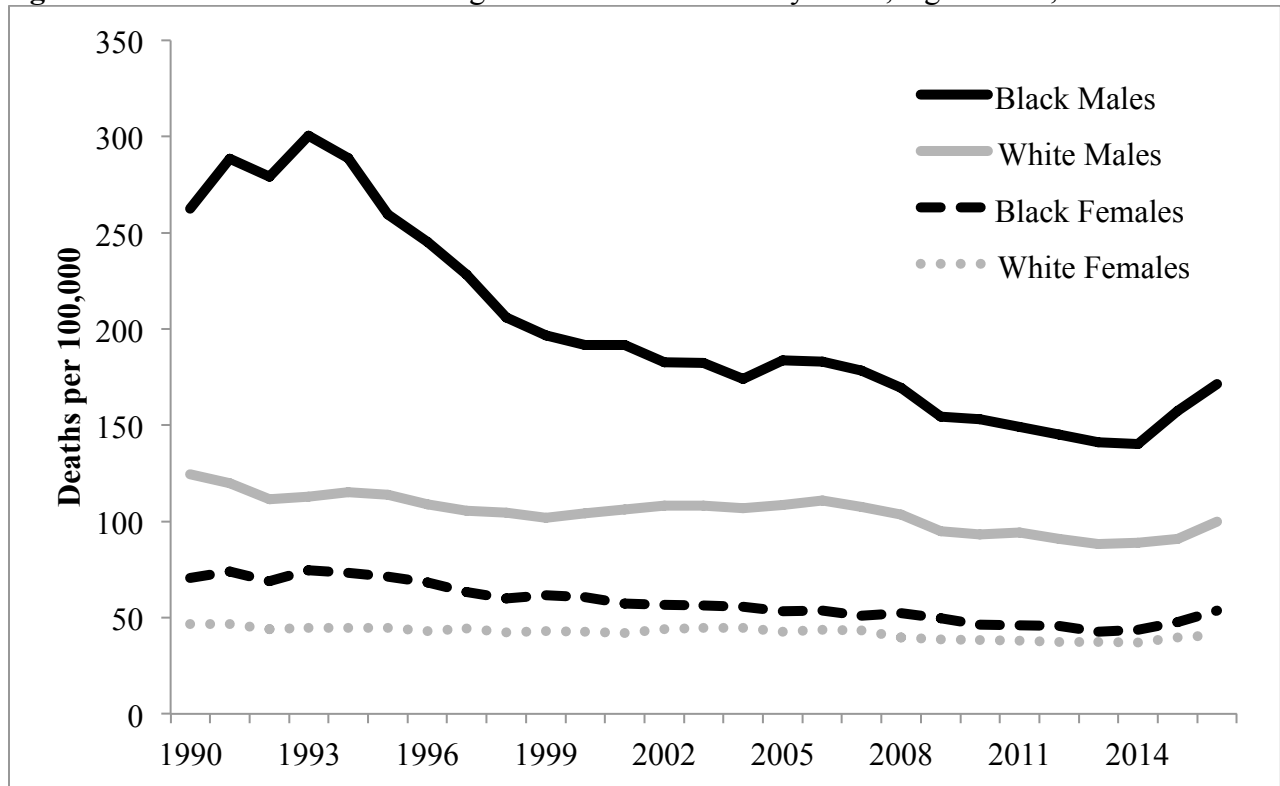
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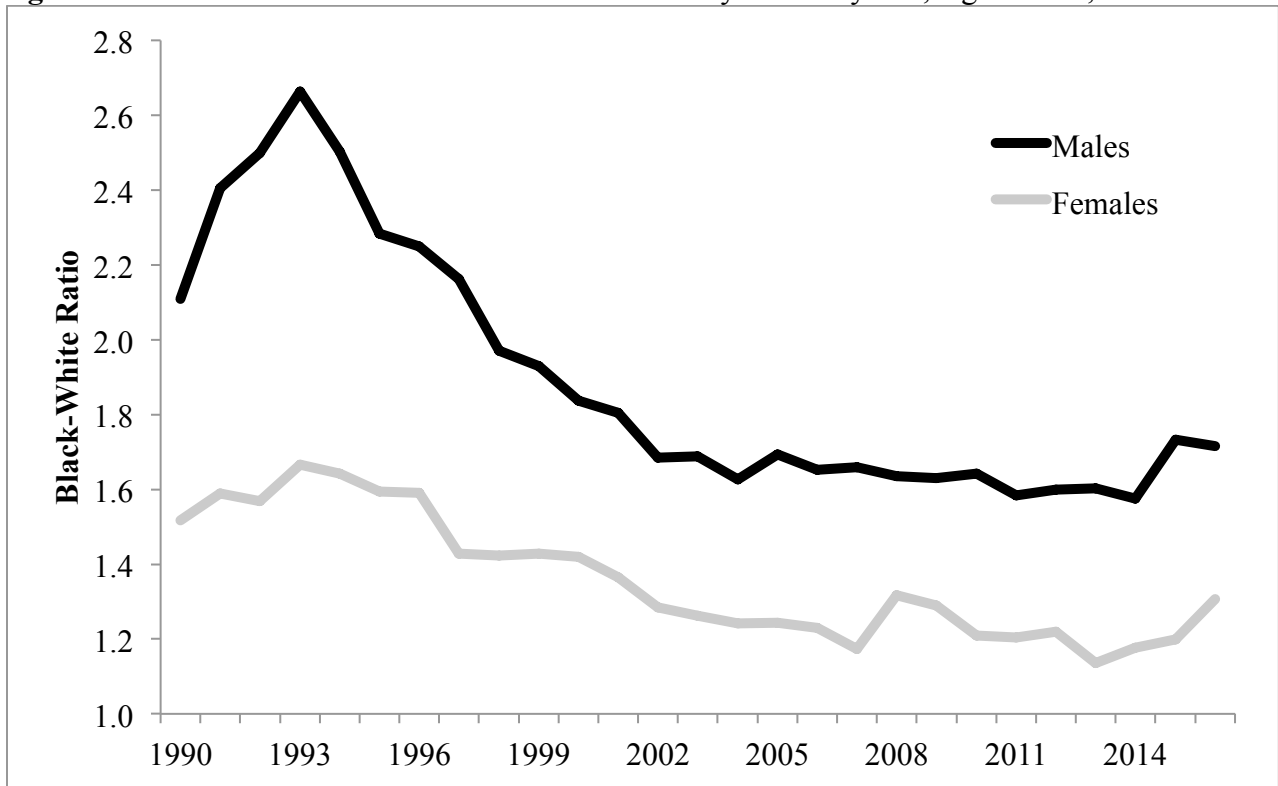
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**Figure 1.** United States All Cause Age-Standardized Mortality Rates, Ages 15-24, 1990-2016



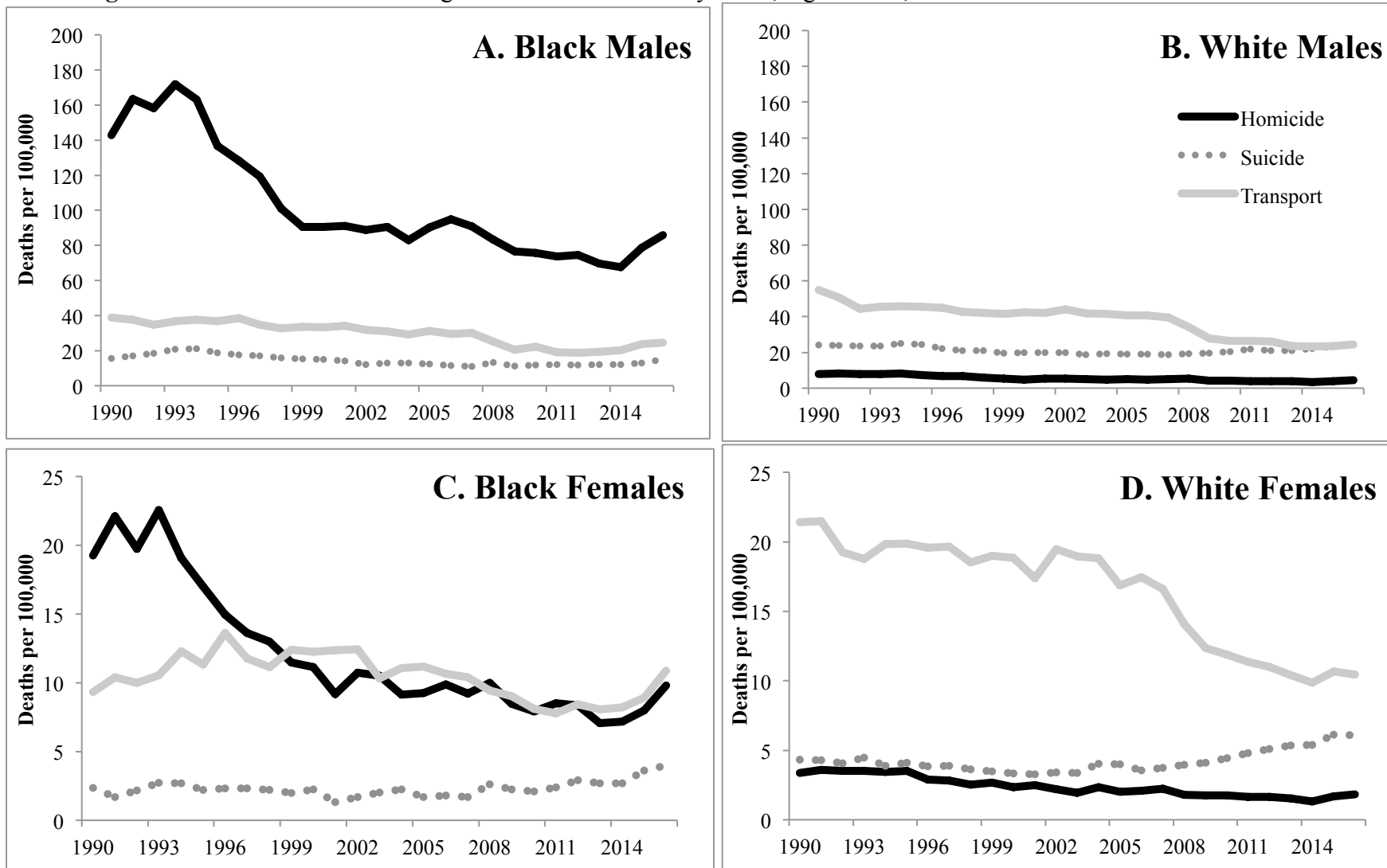
*Notes:* Data come from National Vital Statistics System mortality files and Surveillance, Epidemiology, and End Result Program population counts.

**Figure 2.** United States Black-White All Cause Mortality Ratios by Sex, Ages 15-24, 1990-2016



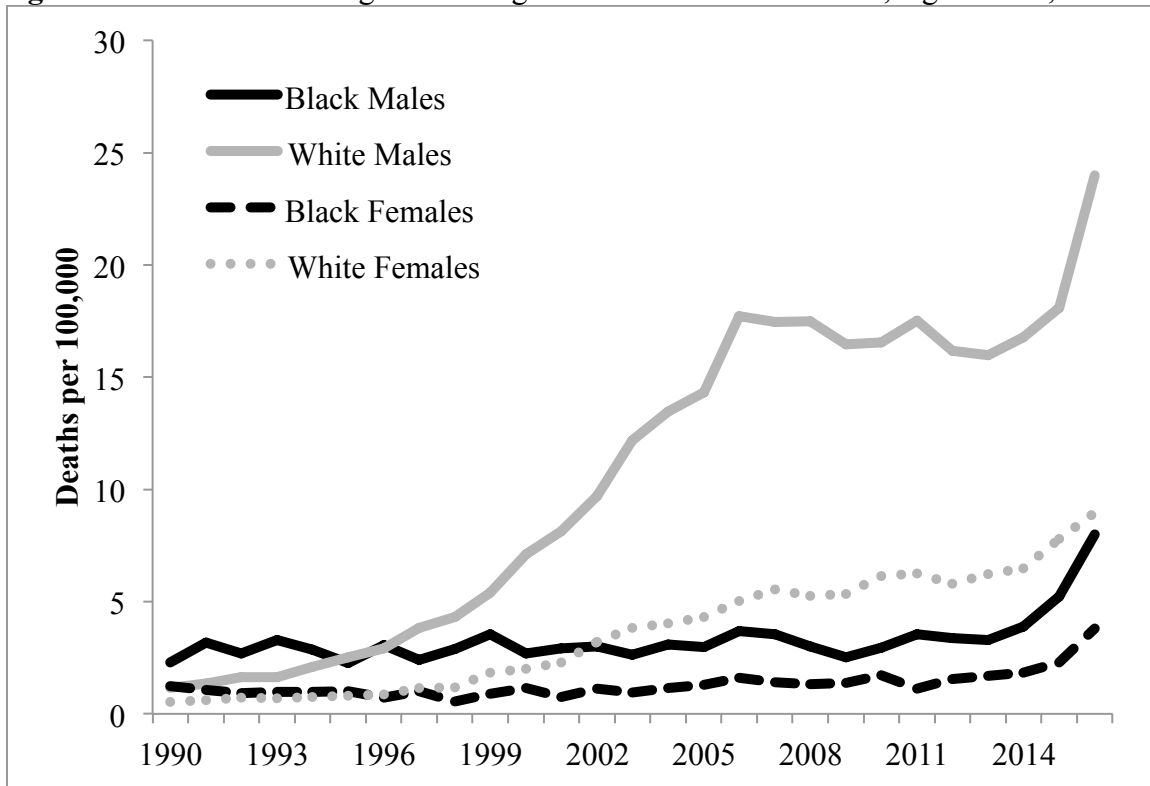
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**Figure 3.** United States External Age-Standardized Mortality Rates, Ages 15-24, 1990-2016



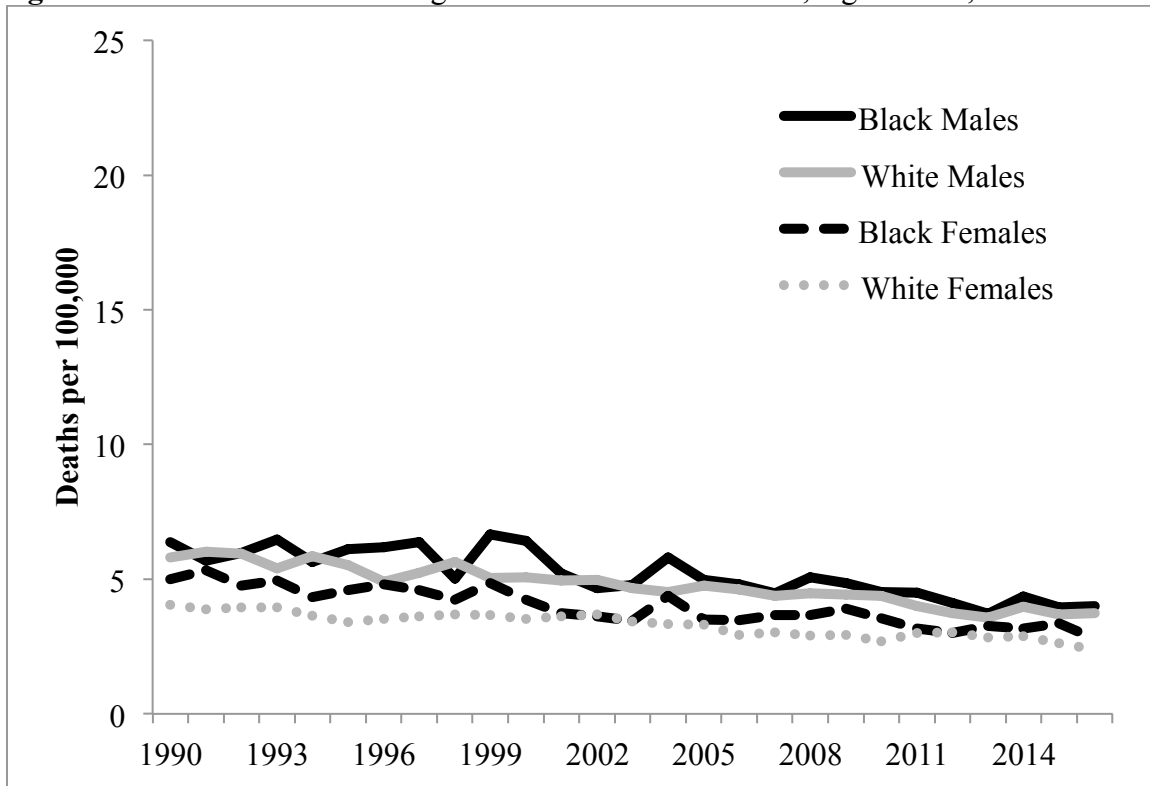
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**Figure 4.** United States Drug Related Age-Standardized Death Rates, Ages 15-24, 1990-2016



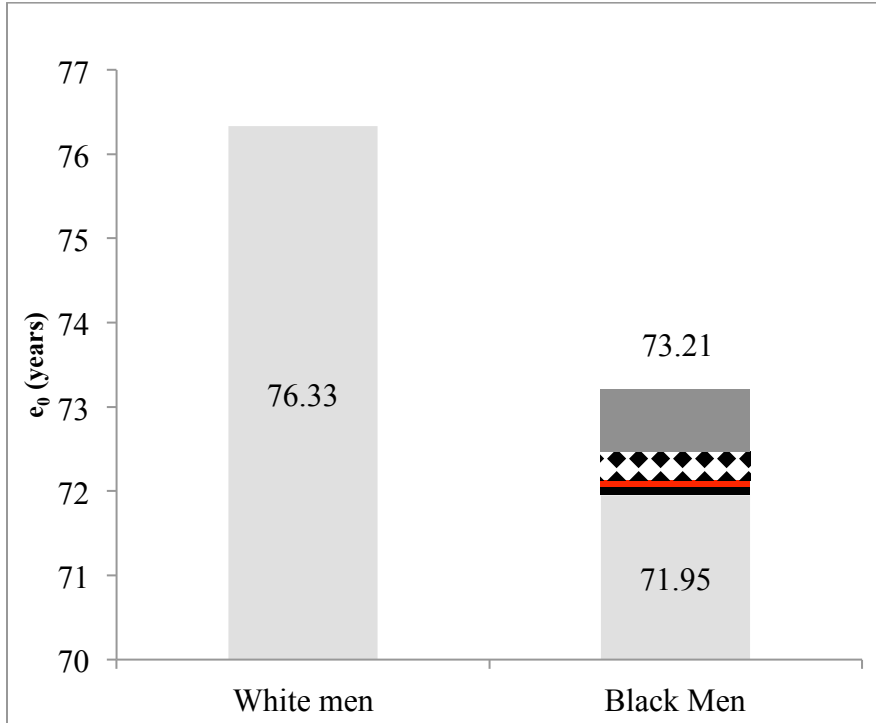
*Notes:* Data come from National Vital Statistics System mortality files and Surveillance, Epidemiology, and End Result Program population counts.

**Figure 5.** United States Cancer Age-Standardized Death Rates, Ages 15-24, 1990-2016



*Notes:* Data come from National Vital Statistics System mortality files and Surveillance, Epidemiology, and End Result Program population counts.

**Figure 6.** Life Expectancy at Birth ( $e_0$ ), White and Black Men, United States 2016



*Notes:* Data come from National Vital Statistics System mortality files and Surveillance, Epidemiology, and End Result Program population counts.

Light gray: actual  $e_0$

Solid black:  $e_0$  gain if black men had homicide rate of white men 15-24 (72.05 years)

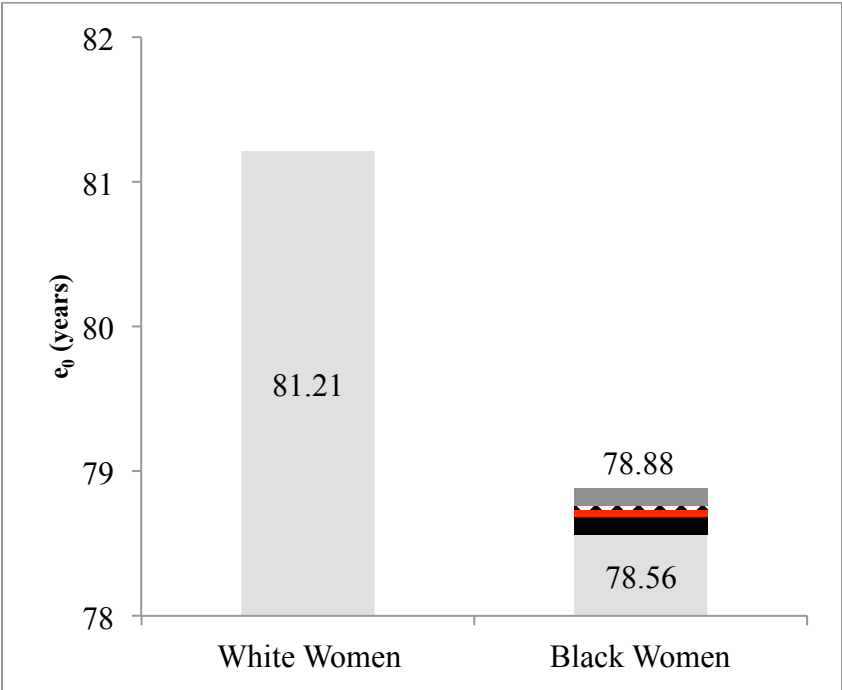
Solid red:  $e_0$  gain if black men had homicide rate of white men at all ages (72.13 years)

Checkered:  $e_0$  gain if black men had no homicides 15-24 (72.48 years)

Dark gray:  $e_0$  gain if black men had no homicides at all ages (73.21 years)



**Figure 7:** Life Expectancy at Birth ( $e_0$ ), White and Black Women, United States 2016



*Notes:* Data come from National Vital Statistics System mortality files and Surveillance, Epidemiology, and End Result Program population counts.

- Light gray: actual  $e_0$
- Solid black:  $e_0$  gain if black women had homicide rate of white women 15-24 (78.69 years)
- Solid red:  $e_0$  gain if black women had homicide rate of white women at all ages (78.74 years)
- Checkered:  $e_0$  gain if black women had no homicides 15-24 (78.76 years)
- Dark gray:  $e_0$  gain if black women had no homicides at all ages (78.88 years)