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"Outside the Skin": The Persistence of Black–White Disparities in U.S. Early-Life Mortality

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ABSTRACT Research on Black–White disparities in mortality emphasizes the cumulative pathways through which racism gets "under the skin" to affect health. Yet this framing is less applicable in early life, when death is primarily attributable to external causes rather than cumulative, biological processes. We use mortality data from the National Vital Statistics System Multiple Cause of Death files and population counts from the Surveillance, Epidemiology, and End Result Program to analyze 705,801 deaths among Black and White males and females, ages 15–24. We estimate age-standardized death rates and single-decrement life tables to show how all-cause and cause-specific mortality changed from 1990 to 2016 by race and sex. Despite overall declines in early-life mortality, Black–White disparities remain unchanged across several causes—especially homicide, for which mortality is nearly 20 times as high among Black as among White males. Suicide and drug-related deaths are higher among White youth during this period, yet their impact on life expectancy at birth is less than half that of homicide among Black youth. Critically, early-life disparities are driven by preventable causes of death whose impact occurs "outside the skin," reflecting racial differences in social exposures and experiences that prove harmful for both Black and White adolescents and young adults.

KEYWORDS Adolescence/young adulthood • Mortality • Racism • Racial disparities • Homicide

Introduction

Mortality during adolescence and early adulthood (ages 15–24) poses 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 significant congenital or senescent processes 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). Given large-scale reductions in these causes of death in the United States over past decades, adolescents and young adults have

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experienced remarkable declines in mortality (Gore et al. 2011; Patton et al. 2009; Thakrar et al. 2018; Viner et al. 2011). Perhaps because of these dramatic declines in mortality and relatively low numbers of deaths, mortality disparities in early life—and the causes contributing to them—have received considerably less attention from population health scholars compared with other age groups.

Despite the positive trends, the racial gap in early-life mortality accounts for thousands of excess deaths among Black adolescents and young adults relative to their White counterparts (Xu et al. 2018). Indeed, Black–White disparities in healthy life expectancy and mortality throughout the life course are a defining aspect of U.S. population health (Hummer and Chinn 2011; Kim and Miech 2009; Levine and Crimmins 2014; Masters et al. 2014; Olshansky et al. 2012; Williams and Jackson 2005) and are implicated in the United States' low life expectancy relative to other wealthy nations (Avendano and Kawachi 2014; Woolf and Aron 2013). While researchers have long acknowledged the role of racism—that is, individuals' racialized experience in a stratified society—in shaping population health (e.g., the pioneering work of W. E. B. Du Bois (1899)), more recent scholarship has sought to formally theorize it as a *fundamental cause* of health and mortality disparities in the United States, where historical and contemporary racism structures Black–White relations and subsequent health outcomes (Hicken et al. 2018; Hummer 2022; O'Brien et al. 2020; Phelan and Link 2015).

These frameworks are comprehensive and compelling in explaining persistently worse health outcomes among racial minorities in the United States. By conceptualizing racism as an all-encompassing and lifelong institution, they emphasize the many cumulative social and individual mechanisms perpetuating Black–White health disparities across time and place and throughout the life course (Brown and Homan 2022; Goosby et al. 2018; Hicken et al. 2018; Phelan and Link 2015). This large body of research documenting the cumulative, biophysiological processes through which institutional and interpersonal racism gets "under the skin" has proved invaluable for explaining Black–White disparities in morbidity and mortality later in life, wherein chronic disease and the cumulative, lifetime consequences of deleterious health behaviors and exposures contribute to poor health and premature death (Ferraro and Shippee 2009; Geronimus et al. 2010).

Yet, looking "under the skin" is less informative for understanding racial disparities in mortality at younger ages. Adolescence and early adulthood represent a critical turning point in the life course when youth establish some independence from their parents and family (Shanahan 2000). This newfound independence is associated with a substantial increase in exposure to and risk of death from external causes; the leading causes of death in early life are due to more acute and, to a large extent, preventable causes (e.g., accidents, suicide, and homicide) whose etiology is far different from the profile of gradual and cumulative health decline at older ages (Heron 2017; Rogers et al. 2022). In turn, the nature of racial disparities in mortality throughout the life course bears further scrutiny, as researchers strive to be cognizant of how racism acts "*outside* the skin" in shaping cause-specific trends among Black and White males and females at these ages.

Racism, by its nature, exists outside the skin and is external to the individual. It is a fundamental cause that precipitates many and multiple mechanisms that are detrimental to the health and well-being of the Black population. There is nothing inherent

to individuals' race that accounts for racial disparities in morbidity and mortality; rather, Black adults' exposure to adverse *external,* structural conditions produces these population-level differences across multiple dimensions of health. In emphasizing how we hypothesize racism as manifesting outside the skin, we instead draw attention to causes of death that likely reflect more acute and immediate mechanisms consistent with individuals' exposures to unsafe social environments or vulnerability to negative social influences (Beltrán-Sánchez 2011; Elo et al. 2014; Miech et al. 2011; Rogers et al. 2017; Sharkey and Friedson 2019), with a particular interest in racial differences therein. We contend that descriptive evidence of racial disparities in these external causes is crucial to developing a more comprehensive and nuanced understanding of how and why racism shapes disparities in mortality throughout the life course. This requires age- or life stage–appropriate theories on the mechanisms through which racism operates and how this may be reflected in the etiology of deaths driving racial differences. Indeed, this is a gap we seek to address in describing early-life mortality disparities as the product of harmful conditions exerting their acute and immediate influence "outside the skin," as compared with the more chronic and cumulative processes that take their toll "under the skin" in later life.

Recent critical perspectives on "Whiteness" as a determinant of health in and of itself reinforce the importance of these external causes of death in understanding disparities in Black–White life expectancy (Lee and Hicken 2018; Malat et al. 2018; Siddiqi et al. 2019). Whiteness undoubtedly confers many social and health advantages; however, the racialization and internalization of Whiteness as a socially dominant status also harms the health of White adults, as manifest in worse mental health, lower psychological resilience, and self-destructive behaviors—all of which are hypothesized as underlying rising mortality from suicide and drug overdoses (Malat et al. 2018). Thus, knowledge of racial disparities in early life stands to benefit from more careful consideration of how specific causes of death correspond with these theoretical perspectives on racism as a danger to both Black and White lives in early life.

In this article, we use mortality data from the National Vital Statistics System and population counts from the Surveillance, Epidemiology, and End Result Program to analyze Black–White disparities in all-cause and cause-specific mortality among adolescents and young adults (ages 15–24) between 1990 and 2016. Specifically, we analyze the five most common causes of mortality in this age group: transport accidents, suicide, homicide, drug-related deaths, and cancer. While the first four causes encompass external causes of death, the fifth is driven by intrinsic, biological forces in this age group. We first show persistent disparities in all-cause mortality, by which Black adolescents and young adults die at considerably higher rates than their White counterparts. Second, we show that compared with White adolescents, Black adolescents are more likely to die from homicides but less likely to die from suicides, drug poisonings, and transport accidents. Indeed, the loss of life expectancy at birth associated with eliminating Black male homicide at these ages is more than double any other cause for the race and sex groups examined. Third, we find no clear race differences in cancer deaths.

Although these data are unable to provide direct evidence of anti-Black racism and Whiteness as causal mechanisms underlying racial disparities in early-life mortality, they are consistent with theoretical perspectives on how and why structural racism and the internalized experience of Whiteness operate "outside the skin" as a more

acute and immediate source of risk at younger ages. Moreover, while there is some degree of bifurcation in the causes of death disproportionately affecting Black and White young adults and adolescents, similar to the case with older adults, the racial disparities observed in these external causes of death point to a similar set of systemic and institutional determinants—such as individuals' social, economic, and physical environments—that disproportionately affect early mortality in the Black population.

Background

Racism as a Fundamental Cause of Mortality Disparities

With the important exception of COVID-19-related mortality, recent decades have generally 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), as well as worsening health among White adults (Case and Deaton 2015; Montez and Zajacova 2013; Sasson 2016). Yet there are still sizable Black–White differences in mortality (Hummer and Gutin 2018; Williams and Mohammed 2009; Xu et al. 2018) and pessimism that this gap will ever close (Olshansky et al. 2012). In seeking to explain the persistence of these stark racial health disparities, recent scholarship has sought to define racism as a distinct and fundamental cause of disparities in health and mortality throughout the entirety of U.S. history (Dennis et al. 2021; Hicken et al. 2018; Phelan and Link 2015).

Indeed, 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). Racism is exemplary of this framework, having taken on many overt and more subtle and coded forms, ranging from slavery and Jim Crow legislation to present "color-blind" racism across multiple social, economic, and legislative institutions (Bonilla-Silva 2006; Reskin 2012). Likewise, different population health threats and causes of death emerge and fade over time; yet most exhibit clear racial patterning (Clouston and Link 2021; Gutin and Hummer 2021; Phelan and Link 2015). Unsurprisingly, there is continued evidence of sizable Black–White disparities across multiple causes of death in the United States (Hummer and Gutin 2018; Xu et al. 2018).

Given the emphasis on chronic diseases as the leading causes of death, 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 (Ferraro and Shippee 2009; Geronimus et al. 2010). This framework highlights the gradual and sustained process by which racism gets "under the skin," negatively influencing health through both the indirect mechanisms of sustained socioeconomic disadvantage and the direct harmful consequences of everyday discrimination. For instance, greater educational attainment does not offer the same health advantages for Black as for White Americans, and this disparity is only compounded with age (Boen 2016; Shuey and Willson 2008). Similarly, accumulated income and wealth disparities over the life course account for a substantial portion of racial disparities in older adult health and mortality (Kahn and Fazio 2005; Lynch

2008; Pais 2014). Independent of socioeconomic status, the cumulative health toll of chronic stress attributable to everyday experiences of racial discrimination results in systemic inflammation and cardiometabolic dysfunction that lead to shorter and less healthy lives among the Black population (Jackson et al. 2011; Williams and Mohammed 2009; Williams and Sternthal 2010). As recent work using longitudinal data and biomarkers has shown, these biopsychosocial mechanisms originate early in life (Goosby et al. 2018; Goosby et al. 2015) and have long-lasting physiological consequences well into adulthood (Boen 2020).

Consistent with this emphasis on the cumulative chronic deterioration of health throughout the life course, research testing the conceptual and empirical validity of fundamental cause theory examines disparities in mortality on the basis of how *preventable* a certain cause of death is or the extent to which it could have been avoided as a function of social, behavioral, and lifestyle factors, rather than biology or genetics (Beltrán-Sánchez 2011). This research consistently finds that causes of death most closely linked to the availability of key medical and public health resources (e.g., screening/testing protocols for different cancers or HIV) exhibit large and sometimes widening Black–White disparities (Levine et al. 2010; Rubin et al. 2010; Soneji et al. 2010; Tehranifar et al. 2016; Tehranifar et al. 2009). Although this research primarily examines "medically" amenable causes of death, it speaks to the broader issue of how a more equitable racial distribution of lifesaving medical and public health knowledge and resources could avert thousands of premature deaths.

"Whiteness" and Declining White Life Expectancy

An understanding of the narrowing U.S. Black–White mortality gap is incomplete without acknowledgment of the worsening health in the White population. Prior to the onset of the COVID-19 pandemic, the dominant recent population health narrative in the United States was the increase in "despair"-associated mortality among White males and females (Case and Deaton 2015, 2021; National Academies of Sciences, Engineering, and Medicine (NASEM) 2021) and corresponding decreases in life expectancy starting in 2016 (Xu et al. 2018)—if not earlier for some subgroups of this population (Montez and Zajacova 2013; Sasson 2016). Although there is continued uncertainty in the appropriateness of "despair" as a catchall term for deaths from drug overdoses, accidental poisonings, alcohol-related diseases, and suicide (Gaydosh et al. 2019; Masters et al. 2018), recent scholarship situates these population trends amid a broader discussion of racism's negative consequences for the health of both Black and White adults in the United States, albeit via different mechanisms (Lee and Hicken 2018; Malat et al. 2018).

Malat and colleagues' work (2018) provides a compelling framework for understanding the many ways White adults experience health advantages and disadvantages as a function of their position as the dominant group in the United States' racial hierarchy. The White advantage in health vis-à-vis Black Americans is seen across numerous conditions and causes of death, but it is neither universal nor unidirectional, as there are key domains and categories of health and mortality where the internalization of Whiteness appears to confer its own risks. Notably, research points to worse mental health and lower levels of psychosocial resilience among White relative to

Black adults in the United States (Keyes 2009), rooted in inflated and often unmet social expectations tied to engrained beliefs about meritocracy and White superiority (Malat et al. 2018; Siddiqi et al. 2019). Impaired mental and psychosocial health, and the racialized social constructs that underlie it, are associated with harmful and self-destructive behaviors and coping strategies that are hypothesized as underlying White Americans' higher rates of substance-abuse, accident-related, and suicide mortality (Case and Deaton 2021; Lee and Hicken 2018; Malat et al. 2018). However, the emphasis on rising mortality and declining health in midlife, and the changing trends in causes of death in the last few years, suggests that our knowledge of the harmful and pervasive aspects of Whiteness in early life remains far less understood (NASEM 2021).

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. Apart from infant mortality (Alexander et al. 2008; Rossen and Schoendorf 2014; Wallace et al. 2017), and a few studies of child health (Mehta et al. 2013; Rogers et al. 2022; Rogers et al. 2017; Turney et al. 2013), 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. Critically, 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 adulthood (Warner and Hayward 2006). Likewise, the preventability framework for understanding Black–White disparities in mortality has largely focused on causes of death most directly amenable to medical intervention. Yet, preventability takes on a different meaning in early life, wherein the focus is not exclusively on preventability as a function of medical care but instead as a function of how the relative safety and stability of adolescents' and young adults' social and physical environments limit exposure to mortality from external causes (i.e., those "outside the skin"), many of which are considered more preventable than later-life chronic disease mortality (Beltrán-Sánchez 2011; Elo et al. 2014; Miech et al. 2011).

Researchers have long emphasized the importance of such external causes of death as accidents, homicides, and suicides at younger ages in discussions of U.S. mortality trends (Ho 2013; Miech et al. 2011; NASEM 2021; Woolf and Aron 2013). Although external sources of mortality have recently received more attention because of their outsized role in declining U.S. life expectancy (Case and Deaton 2015; Xu et al. 2018), high rates of early-life mortality associated with recklessness, violence, and self-harm are an unfortunate example of American exceptionalism. Indeed, nearly two thirds of the male life expectancy gap between the United States and other wealthy, industrialized nations, and two fifths of the female gap, is attributable to 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; NASEM 2021).

Notably, large disparities in who experiences the highest mortality rates across these causes of death further account for the United States' laggard status in both early-life and overall mortality. Despite similarities in leading causes of death among Black and White adolescents and young adults, assault-related deaths account for only 4% of deaths among White females and males ages 15–24 but for more than 40% of deaths among similarly aged Black females and males (Sharkey and Friedson 2019), for whom they are the *leading* cause of death (Heron 2017). By contrast, mortality attributable to accidents, poisonings, and suicide is more prevalent among younger White females and males (Case and Deaton 2015; Heron 2017), which speaks to a very different etiology of underlying social factors (Malat et al. 2018).

Yet research on racial differences in early-life mortality is lacking, and the few extant studies find significantly elevated rates of external mortality for Black adolescents and young adults as compared with their White counterparts, especially among preventable causes of death associated with violence (Kochanek et al. 1994; Rogers et al. 2017; Sharkey and Friedson 2019). However, these studies are limited in their focus on past decades (i.e., 1980s) (Kochanek et al. 1994) or their reliance on survey data (Rogers et al. 2017). Sharkey and Friedson's (2019) recent analysis underscores the large contribution of declines in homicide to the reduction in Black–White disparities in life expectancy from 1991 to 2014, but this was the only cause of death examined in the study. More critically, as we continue to observe overall improvements in U.S. early-life mortality (Khan et al. 2018), we should be cognizant of the extent to which these absolute declines potentially obscure the persistence of relative Black–White disparities, as is the goal of the present study.

Aims

Early life is a unique and important part of the life course, during which race is consequential in its influence on the social and contextual factors that affect health and mortality. Research has invoked the persistence of racial disparities in chronic and degenerative disease mortality at older ages—especially those considered more "preventable"—to illustrate the *fundamental* nature of racism as the cause of Black–White differences in health. Yet, fewer efforts have been made to systematically document the persistence of racial disparities in early-life mortality, where the leading external causes of death have a more immediate etiology. Consequently, in adopting a fundamental cause perspective on racism as a source of Black–White disparities in early-life mortality, our analyses examine racial disparities over time and across a diverse set of preventable causes of death that are dominant earlier in the life course.

Emerging knowledge and theory of how racism negatively affects the health of *both* Black and White adults suggest that the magnitude and direction of these disparities will differ by cause-specific mortality, reflecting the heterogeneous etiology of these causes of death. Although the vital statistics data we use in our analyses cannot definitively speak to the causal mechanisms underlying individuals' mortality, or to the contributing role of various racialized ideologies and institutional factors, we draw on this past work to help interpret these descriptive findings. Specifically, even at younger ages—when accidental or unintentional injury 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 that exhibit patterns consistent with both the internalization of Whiteness and the experience of racism as a product of racial disparities in social circumstances, resources, and broader forms of institutional discrimination. However, the fundamental nature of Black–White mortality disparities in the United States leads us to suspect that the relative size of racial disparities in all-cause mortality will remain consistent over time, indicative of racism's enduring influence on health, regardless of the changing nature of mortality over time.

Specifically, we first highlight how rates in all-cause mortality vary over time for young non-Hispanic Black and White males and females aged 15–24. Second, we contrast these trends against trends in the five leading causes of death for Americans aged 15–24 (four external: transport accidents, homicide, suicide, and drug-related; and one internal: cancer), emphasizing the patterning of Black–White differences. Third, we show how life expectancy at birth (e_0) would change for Black and White males and females if these causes of death were eliminated at ages 15–24. By highlighting the persistent racial disparities specific to distinct external causes of death, we argue that more theoretical attention should be given to understanding the idiosyncrasies of racial disparities in early-life mortality, thereby providing greater nuance in understanding racism as a fundamental cause of health and mortality throughout the life course.

Data and Methods

We employ data from the National Vital Statistics System (NVSS) Multiple Cause of Death files (National Center for Health Statistics (NCHS) 2018) 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. The U.S. Census Bureau produces age-, race/ethnic-, and sex-specific U.S. population estimates from the midpoint of each year, July 1. SEER creates a data set that standardizes these population counts across time (SEER 2020). We restrict our analyses to U.S.-born non-Hispanic White and non-Hispanic Black adolescents and young adults aged 15–24. Across the 27-year time period, there were 705,801 deaths among Black and White individuals aged 15–24.

In addition to all-cause mortality, 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 mortality. Unintentional injury deaths are the leading cause of death in this age range, and transport accidents and drug-related deaths account for most of these deaths (Heron 2017). In 2015, suicide was the second and homicide was the third leading cause of death among males and females aged 15–24 (Heron 2017). These four causes of death are highly preventable owing to their amenability to social, environmental, or behavioral intervention (Beltrán-Sánchez 2011). We then analyze cancer, the leading intrinsic cause of death for this age group (Heron 2017). The well-established nontreatable nature of cancers at these ages (e.g., brain cancer, leukemia) serves as a contrast to the external causes described. That is, while the four external causes of death are clearly preventable,

the experience of cancer at younger ages is less preventable and thus perhaps less socially patterned. We use cause of death codes from the International Classification of Disease (ICD) and standardize across the ninth and tenth versions. We code *homicide* (ICD9: 960–969; ICD10: X85–Y09, Y87.1), *suicide* (ICD9: 950–959.9; ICD10: U03, X60–X84.9, Y87.0), *transport accidents* (ICD9: 800–848, 929.0, 929.1; ICD10: V01–V99, Y85), *drug-related deaths* (ICD9: 304, 850–858, 962, 980–980.5; ICD10: F11–F16, F19, X40–X45, X85, Y10–Y15), and *cancer* (ICD9: 140–208, ICD10: C00–C97).

First, we estimate single-year age-standardized mortality rates by sex and race/ethnicity for all-cause and cause-specific mortality among Black and White males and females aged 15–24 in 1990–2016. Estimates are age-standardized to the race/ethnicity- and sex-specific age distributions for the entire study time period. We also show the Black–White mortality ratio, separately by young males and females, to examine the persistence of racial disparities. Second, we demonstrate the implications of these relative differences for absolute differences in life expectancy. Specifically, 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 and White males and females if the five analyzed causes of death were eliminated at ages 15–24. To calculate ASDTs, we first estimate e_0 for all-cause mortality for each race/sex group, then we estimate e_0 in the absence of the cause of death i in 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 1960).[1]

Results

To examine Black–White differences in all-cause mortality, Figure 1 illustrates all-cause age-adjusted mortality rates (panel a) and ratios (panel b) for Black and White males and females, from 1990 to 2016. Panel a shows that rates for adolescent Black males declined consistently 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 remained stable or slightly declining for Black and White females and for White males until the early 2010s, when trends began to reverse and mortality rose between 2014 and 2016. Panel b shows the large and consistent Black–White mortality ratio for young males and females over the study time period. The ratio for males 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 closing of this gap was largely driven by reductions in Black male mortality across the 1990s and early 2000s. The ratio followed a similar pattern for females, with a more modest fluctuation. Reflecting the lack of continued declines in mortality rates for both males and females, the Black–White mortality ratio did not decline after 2011. In fact, the ratio for males rose between 2014 and 2016 (from 1.58 to 1.73), and the ratio for females has been increasing since 2013, driven by increases in Black

[1] We also estimate ASDT using linear and exponential life table methods. Results were nearly identical across approaches.

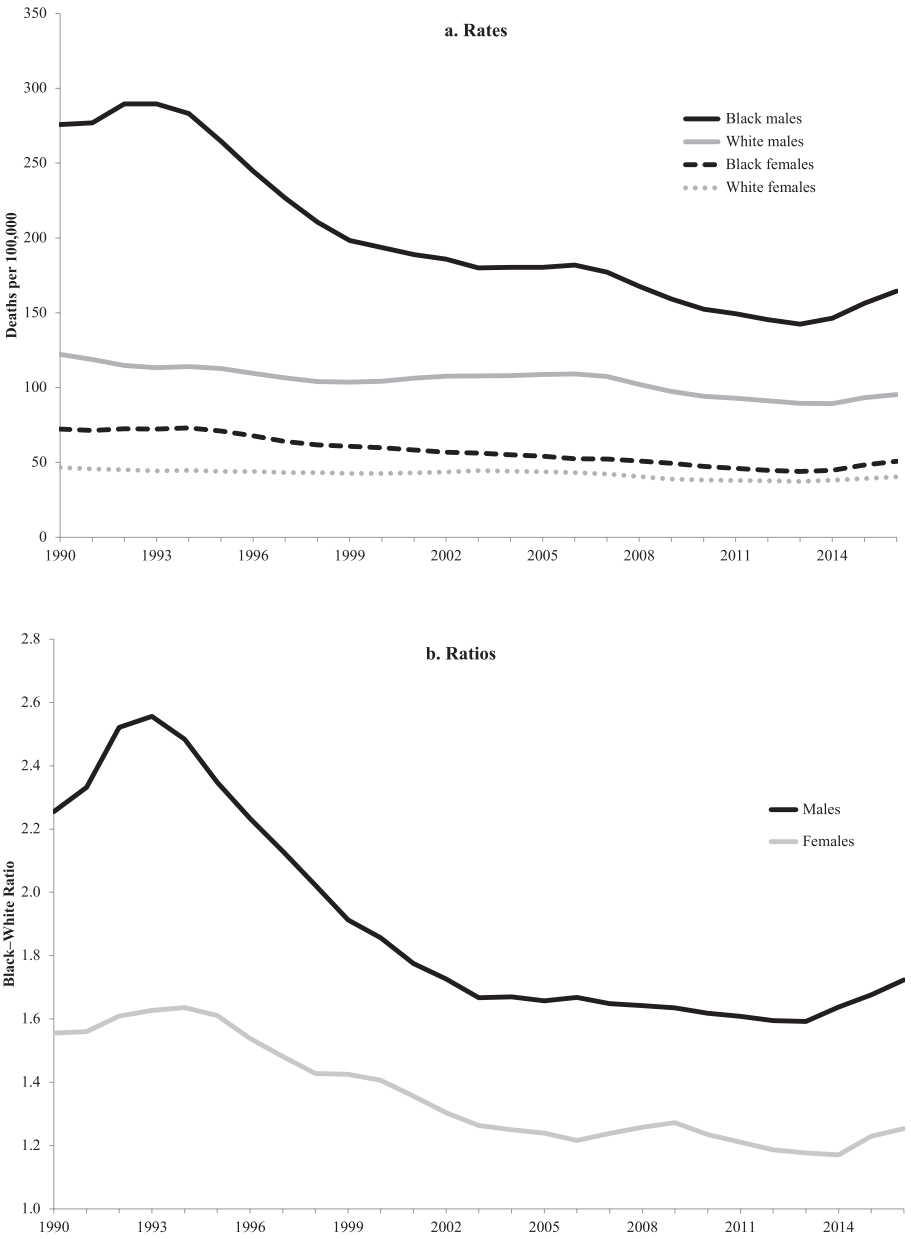

Fig. 1 All-cause age-standardized mortality rates (panel a) and ratios (panel b), ages 15–24, United States, 1990–2016. Data are from National Vital Statistics System mortality files and Surveillance, Epidemiology, and End Result Program population counts. Data represent three-year moving averages.

females' mortality. In the online supplementary Figures S1 and S2, we disaggregate these trends by age group and show comparable patterns. We note that the trends are largely driven by higher rates of mortality among the older ages.

Figure 2 shows trends in cause-specific external mortality rates for Black and White males and females. Rates of homicide declined for all four groups across the 1990s and

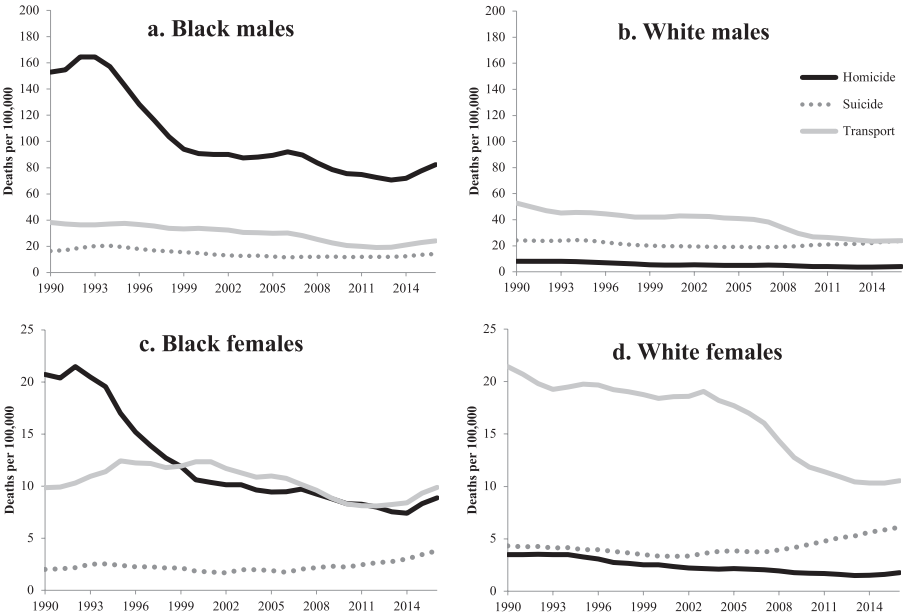

Fig. 2 External age-standardized mortality rates, ages 15–24, United States, 1990–2016. Data are from National Vital Statistics System mortality files and Surveillance, Epidemiology, and End Result Program population counts. Data represent three-year moving averages.

2000s, with recent increases for all since 2014. However, young Black persons continued to have much higher rates of homicide mortality than their White counterparts. In 2016, the rates for Black and White males were 85.90 and 4.40 deaths per 100,000, respectively, and the rates for Black and White females were 9.81 and 1.84 deaths per 100,000, respectively. In the same year, the Black–White mortality ratio for homicide deaths was 19.5 for males and 5.33 for females. Conversely, White males and females had higher rates of transport mortality than their Black counterparts, despite declining rates for all four groups, except for a slight increase for Black females since 2014. Suicide rates were also higher for White adolescents and young adults, though trends across racial/ethnic and sex groups were comparable. The declines in suicide mortality have been minimal for all groups, with some worsening since 2014, mirroring trends among older adults (Curtin et al. 2016). In 2016, transport and suicide mortality rates were identical for White males, at 24.30 deaths per 100,000; for White females, they were 10.45 and 6.07 deaths per 100,000, respectively.

Figure 3 presents trends in drug-related mortality. Like trends observed in older ages (Alexander et al. 2018; Tilstra et al. 2021), rates for all subgroups increased beginning in the late 1990s. This increase was especially pronounced among White males, who between 2013 and 2016 experienced a 50% increase in drug-related mortality (from 15.98 to 24.00 deaths per 100,000). Although rates were much lower for Black males, drug-related mortality also increased substantially between 2013 and 2016, from 3.28 to 8.00 deaths per 100,000, an increase of 144%. Rates for young females have also increased since 2013, to a lesser degree than for young males. Drug-related mortality increased by 44% for White females (from 6.24 to 8.97 deaths per 100,000) and by 127% for Black females (from 1.68 to 3.81 deaths per 100,000).

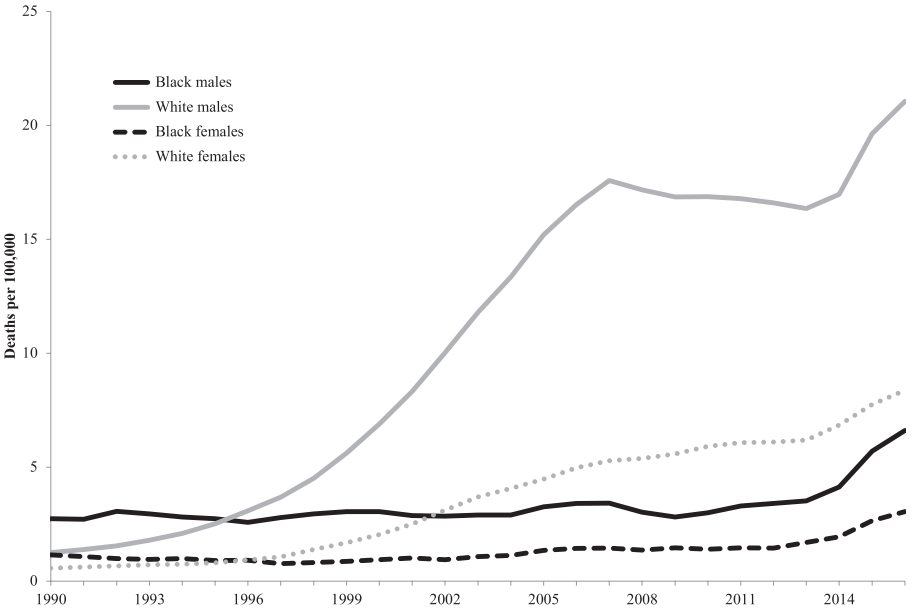

Fig. 3 Drug-related age-standardized death rates, ages 15–24, United States, 1990–2016. Data are from National Vital Statistics System mortality files and Surveillance, Epidemiology, and End Result Program population counts. Data represent three-year moving averages.

In contrast to these external causes, Figure 4 presents cancer mortality rates, the leading internal cause of death for this age group. Unlike trends in homicides, motor vehicle accidents, and suicides, trends in cancer deaths have remained stable or declined minimally over time, with rates that fluctuated between 2.5 and 6.5 deaths per 100,000 for all groups. Females had consistently lower cancer mortality rates than males, and on average, White adolescents and young adults had rates that were only slightly less (about 1 death per 100,000) than those of Black adolescents and young adults.

Next, we consider how life expectancy might improve for Black and White U.S. adults under hypothetical elimination of cause-specific mortality at ages 15–24. Figure 5 displays results from associated single-decrement life tables in which homicide, suicide, transport- and drug-related, and cancer mortality are eliminated among Black males (panel a), White males (panel b), Black females (panel c), and White females (panel d); life expectancy at birth (e_0) is calculated for all four subgroups. The starkest finding is the magnitude of improvement for e_0 for Black males if homicide mortality were eliminated at ages 15–24. In the absence of homicide, life expectancy for Black males would improve by 0.53 years. However, even under this best-case scenario, the gap between Black and White male life expectancy at birth would still be 3.85 years. Second, White male and female life expectancy would be most improved with the elimination of transport- and drug-related mortality and suicide mortality. The magnitude of improvement is consistent across all three causes for White males (0.24 years) and is 0.20, 0.19, and 0.17 years, respectively, for White females.

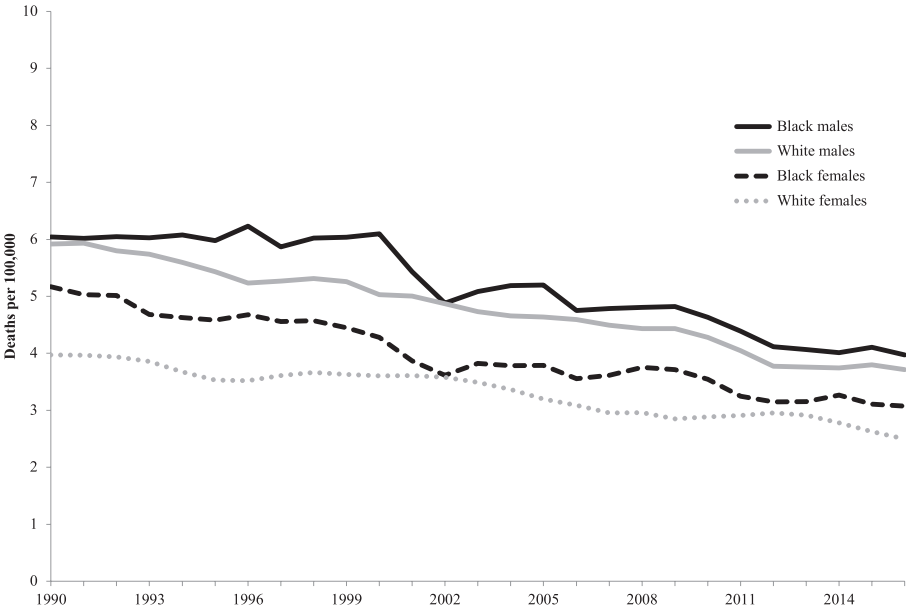

Fig. 4 Cancer age-standardized death rates, ages 15–24, United States, 1990–2016. Data are from National Vital Statistics System mortality files and Surveillance, Epidemiology, and End Result Program population counts. Data represent three-year moving averages.

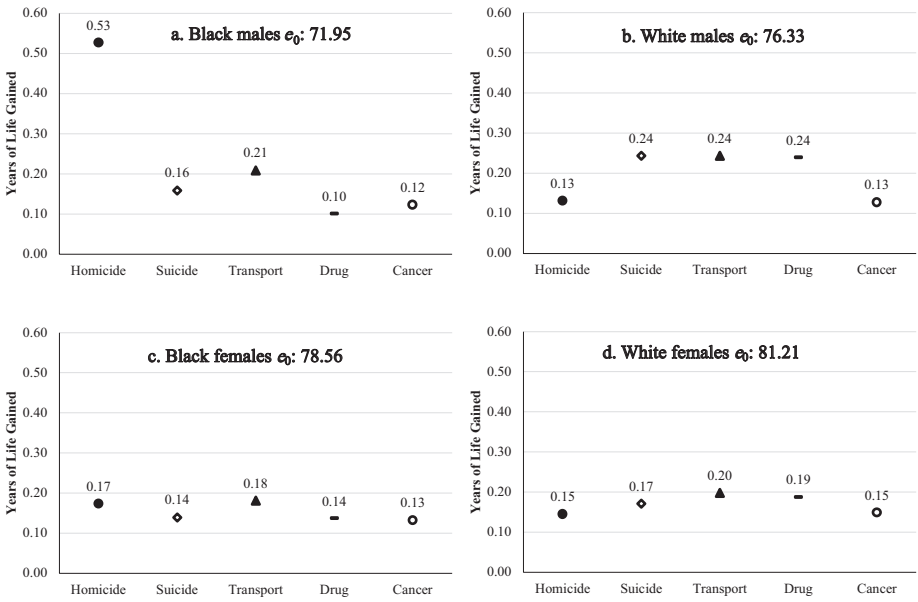

Fig. 5 Life expectancy (e_0) improvements from cause-specific mortality elimination at ages 15–24, United States, 2016. Data are from National Vital Statistics System mortality files and Surveillance, Epidemiology, and End Result Program population counts.

Third, the elimination of drug-related mortality at ages 15–24 would result in greater improvements in White than in Black life expectancy. The improvement would be 2.4 times as great for White males as for Black males (0.24 years vs. 0.10 years) and 1.36 times as great for White females as for Black females (0.19 years vs. 0.14 years). A final noteworthy finding from Figure 5 is the comparability of expected improvement in life expectancy if cancer were eliminated at ages 15–24. For all four subgroups, the elimination of cancer would result in life expectancy improvements of only 0.12 (for Black males) to 0.15 years (for White females).

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 early in the life course (Khan et al. 2018; Kochanek et al. 1994; Rogers et al. 2022; Rogers et al. 2017), at ages typified by relatively good health and high rates of survival. The persistence of these disparities—over time, via multiple mechanisms, across many outcomes, and, as we argue, throughout the life course—has motivated scholars to theorize racism as a fundamental cause of health disparities in the United States (Phelan and Link 2015). As a reflection of the systemic, institutional, and interpersonal mechanisms limiting the advancement of Black males and females relative to their White peers, racism proves pervasive in both society and in its adverse effect on health and survival. Consequently, most research that examines 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, chronic stress, and discrimination (Jackson et al. 2011).

Yet we contend that these cumulative mechanisms inadequately characterize racial differences in mortality in early life, when disparities are best understood as a function of the kinds of acute exposures and events that drive mortality at these ages—that is, those causes occurring "outside the skin" in the sense that they more directly reflect the immediate consequences of unsafe or unstable social environments rather than sustained exposures operating through biophysiological pathways. To provide evidence that advances 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-related, drug-related, and cancer), as well as 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 and White males and females in the absence of cause-specific deaths at these ages.

There are four 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 never dropped below 1.14 for females and 1.58 for males, and instead increased in recent years. Second, this persistent ratio is almost entirely driven by homicide rates among Black adolescents and young adults, which are nearly 20 times

as high and 6 times as high as among White males and females, respectively. In fact, eliminating homicide mortality entirely for Black males at ages 15 to 24 would produce an improvement in their life expectancy at birth of 0.53 years, nearly 0.30 years greater than any other cause of death. Third, we find that, across the study period, suicide and transport-related rates are consistently higher among White males and females than among their Black counterparts, and drug-related deaths have been consistently higher—and the racial gap has been widening—since 2000. This is in keeping with findings of lower rates of suicide in the Black versus the White population (Rockett et al. 2010; Tilstra et al. 2021) and is consistent with arguments that Whiteness is implicated in the surging opioid epidemic (Malat et al. 2018). Fourth, unlike these primarily external causes of death, we find no clear racial differences in cancer mortality among adolescents and young adults, as the rates are relatively stable over time for all race–sex groups in our analysis.

Extending the fundamental cause perspective to include racism provides a powerful and parsimonious framework for understanding Black–White disparities in U.S. health and mortality, emphasizing the systemic and interpersonal racism rampant in the United States that so negatively impacts the health of the 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 emphasizes the oft-neglected significance of acute and 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 and indicative of more immediate dangers—specifically, homicide, suicide, drug-related, and transport-related mortality—but that the directionality and magnitude of these disparities differ depending on the cause of interest.

The Whiteness framework is particularly helpful for understanding why rates of suicide and drug-related deaths are consistently higher among White adolescents and young adults than among their Black counterparts. Consistently higher and increasing mortality rates associated with harmful and self-destructive behaviors and beliefs suggest that the lower psychological resilience and negative coping patterns implicated in rising "despair" mortality among middle-aged White adults may have their origins even earlier in the life course (Malat et al. 2018; NASEM 2021; Siddiqi et al. 2019). Even though adolescents and young adults are less likely to have directly experienced the status loss and socioeconomic distress implicated in so-called deaths of despair (Case and Deaton 2015, 2020), their exposure and proximity to these racialized, societal narratives and adverse psychosocial environments in their communities may be reflected in the racial disparities observed in this analysis. Certainly, vital statistics data cannot directly provide evidence of these causal pathways, but the widening of the Black–White disparity in drug-related deaths in our descriptive analyses perfectly coincides with the increase in White midlife mortality observed in past research, much of which is attributable to the influx of painkillers and growing dependence on both prescription and illegal opioids in predominantly White communities across the United States (Monnat 2018).

While these results suggest that the harmful aspects of Whiteness may be observed in early-life suicide and drug mortality, it is perhaps more important to contextualize the contributions of these various causes in terms of their "net effect" on health.

Indeed, the large and persistent Black disadvantage in early-life mortality over time is emblematic of the "multiplicity of mechanisms" and "systematic asymmetry" by which a fundamental cause negatively influences health (Freese and Lutfey 2011). Under this fundamental cause perspective, there is no requirement that all mechanisms and outcomes exhibit the same patterns (Link and Phelan 1995). Whiteness, with respect to racism as a fundamental cause, is entirely consistent with the "countervailing mechanisms" perspective on socioeconomic status as a fundamental cause; as Lutfey and Freese (2005) demonstrated, there are idiosyncratic reasons and mechanisms that lead socially advantaged individuals to have worse health in some ways relative to less advantaged individuals.

However, in the case of racism, there is a valid concern that White centricity clouds our understanding of public health and how our society prioritizes and addresses disparities and their root causes (Lee and Hicken 2018). The focus on "despair" in the White population is understandable given the thousands of preventable deaths lost to these causes, but our results emphasize how the recent emphasis on these causes neglects the long-standing and disproportionate impact of homicide deaths on early-life mortality among Black females and males. The narrative shift in public health policy toward preventing despair-related deaths among disaffected White adults only further exacerbates the seeming lack of interest in addressing an equally preventable cause of death like homicide. Indeed, the decline in U.S. homicide up until the 2010s was rooted in many positive structural and demographic changes that improved social and material conditions in the Black population (Sharkey 2018; Sharkey and Friedson 2019).[2] However, the primary issue is the lack of a policy interest or resources in pursuing a systematic effort to capitalize on this past knowledge, or to implement novel approaches directly confronting violence, and reduce homicides further (Sharkey and Friedson 2019).

By explicitly singling out homicide as the most significant pathway preserving racial disparities in mortality at these ages, we contend that racism operates somewhat differently as a fundamental cause of health disparities in early life when compared to its conceptualization in past research on later life. As seen in our analyses, racial disparities in early-life mortality are largely maintained by a single, acute mechanism (i.e., homicide), rather than through many mechanisms and through cumulative (dis)advantage as hypothesized in later-life mortality. Homicides are particularly pernicious criminal acts of violence that can exert devastating long-term effects on an entire community. Nevertheless, these acute causes of death in adolescence and young adulthood are preventable and thus amenable to interventions, making this an important area for policy work.

Though points of potential intervention are not drastically different than those highlighted in fundamental cause theory and the racial disparities literature in adulthood—such as "reduc[ing] racial differences in SES [socioeconomic status], 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

[2] Though we emphasize positive mechanisms underlying the decline, it is important to acknowledge that more negative changes—with respect to their far-ranging consequences for Black adults in the United States—such as increases in policing and incarceration rates, may have had an impact as well.

and availability is perhaps less critical at these ages, though not entirely irrelevant when it comes to the issue of violence-related deaths and racial disparities in access to trauma centers (Tung et al. 2019). However, we emphasize how subverting the institutional and interpersonal discrimination that hinders the socioeconomic and geographic mobility of young Black males and females may help reduce the violence that continues 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 should be given to gun laws and neighborhood disadvantage, and how their design and implementation implicitly channel systemic racism writ large to adversely affect racial/ethnic minorities.

Our analyses are not without limitations. First, 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 and thus help bring causal, racism-related mechanisms to light. Recent work by Brown, Homan, and colleagues brings much-needed attention to the importance of measuring structural racism as a pervasive *contextual* determinant of racial disparities (Brown and Homan 2022; Homan et al. 2021). We contend that these structural conditions are particularly important for mortality at younger ages, wherein minimizing the availability of firearms and harmful substances could avert numerous deaths. Nevertheless, our results serve as an entrée into future work in this area using survey data sets that cover this age range, track health or mortality, and, ideally, incorporate longitudinal measures of structural racism that provide more direct evidence of how racism is implicated in these early-life deaths and which contextual determinants appear most consequential.

Despite these limitations, 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 are external to the body, and largely represent acute rather than chronic or cumulative mechanisms, it is important to consider the unique pathways by which racial disparities in mortality emerge for this age group, and what they suggest about the broader role of racism as a fundamental cause of these disparities. 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), we argue that addressing racism as a determinant of mortality disparities at younger ages requires a greater focus on reducing the immediate rather than the long-term consequences of racism on health.

Moreover, recent work on the collateral consequences of untimely and early deaths suggests that reducing mortality among young people—and eradicating disparities therein—is essential to improving the health and well-being of the family, friends, and communities who are affected by these tragic losses. Umberson and colleagues provided compelling evidence that these racial differences in early death are a crucial, if understudied, part of the chronic and cumulative pathway underlying worse health among Black Americans (Umberson 2017; Umberson et al. 2017). The sudden and unexpected death of a child, sibling, spouse, or parent—all of which are

possibilities given the age range under study—is associated with greater psychological distress, worse physical health, cognitive decline, and higher risk of mortality (Donnelly et al. 2019; Liu et al. 2022; Umberson and Donnelly 2022; Umberson et al. 2020). Critically, because these early deaths occur with far greater frequency among Black adolescents and young adults, these sustained losses likely exert a chronic and cumulative toll that explains a significant proportion of racial disparities in health.

More broadly, we stress that a fundamental "delinking" of race and health is likely to yield mortality dividends for both Black and White adolescents and young adults (Phelan and Link 2015). As suggested by the etiology of the causes of death in this analysis, the internalization of Whiteness and marginalization of Blackness likely play a key role in stratifying pathways to mortality in this age group. In turn, these causes of death are themselves racialized and perceived as the product of separate etiologies that largely affect one group but not the other. However, these preventable causes of death stem from the same underlying racialization of society.

An example of direct relevance to our findings is the racialized perception of the significance of firearms for population health. Racist perceptions of gun violence in America undoubtedly contribute to public misperception of the threat of homicide as compared with suicide, despite the fact that gun-related suicides occur at nearly double the rate of homicides (Morgan et al. 2018). The framing of firearm-related homicide as a "Black" issue in the United States, where Black lives are viewed as expendable (Cacho 2012; Mills 2001; Vargas 2010), discounts the substantial toll of firearms on White mortality and contributes to our collective inability to reduce access to firearms and thus reduce gun-related deaths among Black and White adolescents and young adults. Indeed, these inaccurate perceptions of homicide and violence likely contribute to increased firearm ownership and thus greater risk of suicide mortality in the White population (Morgan et al. 2018).

Although this is but one example, it is perhaps the clearest example of how institutional and systemic racism—and its effect on public beliefs and policy imperatives—impedes progress on solutions that would benefit all people, regardless of race. It is of paramount importance that we reduce early-life mortality, in large part by better acknowledging the importance of factors "outside the skin," such as external causes of death that are shaped by the social contexts in which adolescents and young adults are enmeshed. Although this continues to be a time in the life course marked by relatively good health and low mortality, both Black and White young males and females in the United States experience unnecessarily high mortality from a diverse set of causes that are fundamentally rooted in their exposure to racialized environments and sources of risk. ■

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