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ABSTRACT

Racial differences in mortality are large, persistent and likely caused, at least in part, by racism. While the causal pathways linking racism to mortality are conceptually well defined, empirical evidence to support causal claims related to its effect on health is incomplete. In this study, we provide a unique set of facts about racial disparities in mortality that all theories of racism and health need to confront to be convincing. We measure racial disparities in mortality between ages 40 and 80 for both males and females and for several causes of death and, measure how those disparities change with age. Estimates indicate that racial disparities in mortality grow with age but at a decreasing rate. Estimates also indicate that the source of racial disparities in mortality changes with age, sex and cause of death. For men in their fifties, racial disparities in mortality are primarily caused by disparities in deaths due to external causes. For both sexes, it is racial disparities in death from healthcare amenable causes that are the main cause of racial disparities in mortality between ages 55 and 75. Notably, racial disparities in cancer and other causes of death are relatively small even though these causes of death account for over half of all deaths. Adjusting for economic resources and health largely eliminate racial disparities in mortality at all ages and the mediating effect of these factors grows with age. The pattern of results suggests that, to the extent that racism influences health, it is primarily through racism's effect on investments to treat healthcare amenable diseases that cause racial disparities in mortality.

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Introduction

Racial differences in health are large, persistent and well documented.[1] In 1980, the age-adjusted death rate of Black males aged 45 and over was 4,169 per 100,000 persons. The analogous figure for White males was 3,454. The implications of this difference are worth noting—for every 100,000 persons of each race, there were 715 fewer Black men than White men alive at these ages. In 2022, the age-adjusted death rates of males aged 45 and over was 3,047 and 2,485 per 100,000 persons for Black and White males, respectively, implying that for every 100,000 persons of each race, there were 562 fewer Black men alive at these ages.[2] The "cost" of this racial disparity in mortality is a staggering half a trillion dollars when valued at conventional estimates of the value of a statistical life.[3] While death rates have declined by approximately 25% for both races, the absolute decline is greater for Black males than for White males, although the relative improvement is modest (153 per 100,000 persons). Among females of the same ages, death rates are markedly (30% to 40%) lower than those among males, but the changes between 1980 and 2022 are qualitatively similar.

It is widely held that racism is a cause of these racial disparities in mortality. Racism can have a direct effect on health through biological mechanisms associated with heightened, sustained stress (Geronimus et al. 1992; McEwen and McEwen 2017; McEwen 2022), and an indirect effect through its impact on distal (e.g., income) and proximate (e.g., quality of medical treatment) causes of health. It is for these reasons that racism has been referred to as a fundamental cause of health (Phelan and Link 2015). As noted, the recent time series of mortality rates of Black people mirrors that of White people suggesting that Black people have been able to improve their health over time by a similar, if not slightly greater, amount than White people. However, because Black people started from a position of higher mortality than White people, their mortality rate remains higher despite significant and relatively greater improvements. One implication of this empirical fact is that, if racism is the underlying cause of the racial disparity in mortality, then its influence seems to limit, not prevent, Black people's ability to invest in health (e.g., medical care) and to benefit from environmental (e.g., declining pollution, safer roads) and other changes that have extended life over the past 20 years.

While most agree that racism is a cause of racial disparities in health, the pathways by which racism exerts its harmful effects are not well elucidated empirically. Identifying the causal pathways of racism on health is a difficult task. One approach to do so is to, first, identify the causal effect of distal and proximate causes of health, and then assess racial disparities in these causes to estimate the extent to which racial disparities in health, say mortality, are a result of racial disparities in these causes. For example, there is some, although not abundant and not uniform, causal evidence that more education lowers mortality (e.g., Davies et al. 2018), and there are known racial disparities in education. Combining the causal effect of education on mortality with the magnitude of the racial disparity in education provides an estimate of the share of the racial disparity in mortality that works through education.[4] Accordingly, eliminating the racial disparity in education will reduce the racial disparity in mortality. This approach can be applied to other distal and proximate causes of health. While some

[1] See, for example: Dwyer-Lindgren et al. 2024; Dwyer-Lindgren et al. 2022; Arias 2019; Roth et al. 2017; Beydoun et al. 2016; Yao and Robert 2011; and Ng-Mak et al. 1999.
[2] Data are from authors' query of CDC Wonder database: https://wonder.cdc.gov/.
[3] There are approximately eight million Black men aged 45 and over implying approximately 45,000 more deaths relative to a similar sized population of white men. Using $10 million value of a statistical life yields $450 billion.
[4] The racial disparity in education may be only partly a result of racism. Thus, identifying the contribution of racial disparities in education to racial disparities in health does not necessarily identify the effect of racism on health that works through education. Only if racial disparities in education were completely the result of racism would this approach identify the effect of racism on health that works through education.

progress has been made along these lines, this approach has not explained much of the racial disparity in health.

To illustrate why little progress has been made using this approach, we can continue with the example of education. Causal estimates of the effect of education on health were derived from small changes in education, for example, less than one additional year of education, at distinct margins, for example, staying in high school instead of dropping out, and for narrow samples, for example, cohorts affected by school reforms in the 1960s or 1970s (Clark and Royer 2013; Davies et al. 2018; Meghir et al. 2018; Janke et al. 2020). These studies are arguably not particularly relevant to the current racial gap in education in the US, where 42% of White adults have a Bachelor's degree or higher compared to 28% of Black adults (U.S Census Bureau 2023). None of these studies estimated the causal effect of a college degree versus a high school degree, or large differences in education, on health, and none assessed whether the causal effects of education are moderated by race. Furthermore, there is limited evidence on the causal effect of education on health over the life cycle, as the effect of education may differ by age (Kaestner et al. 2020). In sum, relatively little is known about whether education is a mediating pathway between racism and health, particularly over the life cycle.[5] Similar characterizations and conclusions apply to income (Cesarini et al. 2016; Miller et al. 2024; Lyu, Wehby, and Kaestner 2024).[6]

More progress has been made with respect to medical treatments for which there are credible (e.g., experimental) estimates of the causal effect of treatment on health, for example, treatments for hypertension, hyperlipidemia, diabetes and some cancer screenings. However, racial disparities for these treatments are relatively small (Aggarwal et al. 2021; Lu et al. 2023; CDC 2024; American Association for Cancer Research 2024). Thus, even though there are reliable, arguably externally valid estimates of the causal effects of some important proximate causes of health, the racial disparity in many of these causes is small. Therefore, these pathways are unlikely to be an important explanation of the effect of racism on health.[7] This conclusion does not rule out the moderating effect of racism on the effectiveness of these treatments, for example, there is evidence that conditional on diagnosis or treatment, there are racial differences in diabetes and hypertension control (e.g., Aggarwal et al. 2021) and guideline concordant cancer care (Fang et al. 2018; Nogueira et al. 2023).

A second approach to measure the effect of racism on health is to directly measure exposure to interpersonal racism, or racial bias among providers, and estimate the impact of these types of racism on health. This line of research is plagued by a lack of credible estimates of causal effects. Few studies have used experimental, or even quasi-experimental, research designs and most rely on unvalidated instruments to measure discrimination exposure such as the Everyday Scale of Discrimination (Bastos and Harnois 2020; Harnois 2022). Putting aside the methodological weaknesses of these types of studies, the evidence from this literature is mixed (e.g., Liu and Kawachi 2017; Chae et al. 2020; Chae et al. 2012; Becares and Zhang 2018; Dunlay et al. 2017; Monk 2015; Williams et al. 2019; Williams and Mohammed 2019). Even if findings of an adverse effect of exposure to racism are to be believed, the magnitudes of estimates from this literature can explain only a small portion of racial disparities in health despite the reduced form nature of these analyses that are intended to measure the total effect of discrimination (Hall et al. 2015;

[5] This conclusion is based on studies that have used experimental or quasi-experimental research designs. There are hundreds of studies that showed that education mediates the negative association between race and health.
[6] Some studies examined short term changes in income on health and found that an increase in income was associated with higher mortality (Evans and Moore 2011; Andersson et al. 2015).
[7] This conclusion is at odds with widespread, almost rote, claims otherwise. Such claims are often vague and not specific about the disparity in diagnosis and treatment. The evidence we reviewed suggests that these claims overstate the importance of disparities in treatment on racial disparities in health. However, as we noted in text, there may be important disparities in the effectiveness and success of treatment that may be important.

Maina et al. 2018; Alsan et al. 2019; Williams and Mohammed 2013). In sum, this line of study has not greatly advanced the understanding of the pathways by which racism affects health.

Overall, while racism is undoubtedly a cause of racial disparities in health, and may be the root, fundamental cause, the extent of racism's influence and the pathways by which racism adversely affects health is not well known for the reasons discussed above. Despite this evidence, most researchers continue to draw conclusions as if the extent of racism's impact and the pathways of its influence are definitively known. Consider the following quote from prominent researchers of racism and health:

> "Systemic racism also can harm health by placing people of color at economic disadvantage. Given the strong and well-documented influence of economic advantage and disadvantage on health,[36] racially discriminatory obstacles to economic resources and opportunities are a major pathway through which systemic racism can harm health.[37,38]" (Braverman et al. 2022a, p. 175)

Here, we see the authors assuming that there is causal evidence that economic advantage (e.g., education and income) affect health and cite this as a "major pathway". As discussed earlier, there is relatively little such evidence, and what evidence there is, is unlikely to be particularly relevant to current (recent) racial disparities in the US. Therefore, despite the strong intuition underlying the argument, the effect of racism on health that works through economic advantage is not well established.

Another example from the same authors:

> "Widespread, entrenched, and pervasive racially discriminatory policies and practices affecting access to quality healthcare also have been shown repeatedly to play a role in racial disparities in health.[124, 125] These disparities often reflect entrenched, deeply rooted prejudice, as well as sometimes unintentional but nevertheless discriminatory policies and practices built into systems." (Braverman et al. 2022b)

Here, the authors must be referring to only part of the evidence base to make their point because, in contrast to the authors' claim, two recent reviews of studies of the effect of racial bias among providers find decidedly mixed and inconsistent evidence of the effect of provider bias on racial disparities in treatment and health (Hall et al. 2015; Maina et al. 2018).

Finally:

> "…., systemic racism can lead to poorer health among people of color at all economic levels by exposing them chronically to race based unfair interpersonal treatment (or the threat thereof); this can produce chronic stress, which has been shown to lead to increased risks for chronic disease.[44]" (Braverman et al. 2022, p. 175)

In this quote, the authors apply the first approach outlined above for measuring the effect of racism on health. They note that there is a causal effect of stress on health. Then, they appeal to intuition that exposure to interpersonal racism causes stress and therefore racism explains racial disparities in health. However, as we noted, there is a lack of credible, consistent evidence linking exposure to interpersonal discrimination to health. In short, while likely correct, the importance of exposure to interpersonal racism in explaining racial disparities in health is largely unknown.

Our intention is not to criticize these distinguished and knowledgeable scholars of racial disparities in health, but to illustrate that claims about the extent of racism's effect on health and the pathways of racism's influence on health are widespread despite there being relatively little empirical evidence to support these causal claims. In this article, we start with the view that the causal pathways

linking racism to health, while conceptually well defined, are not well documented empirically. We also note that few studies have investigated in a detailed, comprehensive and non-parametric way how racial disparities in health evolve over the adult life cycle. Accordingly, we measure the association between race and mortality at several ages between ages 40 and 80 and do so for several causes of mortality and for males and females.[8] We link the empirical analysis of racial disparities in health over the life cycle to the health production function of the human capital model of the demand for health (Grossman 1972). This model explains changes in health over the life cycle—i.e., between any two ages—by two broad factors: investments in health at that point in life and depreciation of health at that age. Racism can affect both components, for example, by reducing economic resources and thus the quantity of investment at a given age, or by causing heightened stress and raising health depreciation at that age. The conceptual model is an organizing framework useful for linking racism, and theories of racism, to empirical facts. For example, if the racial disparity in mortality remains constant between two ages, say between ages 60 and 65, this suggests that there were no racial differences in net investments in health at that age.[9] This fact would need to be explained by a convincing theory of racism and health.

In sum, the goal of our empirical analysis is to produce a set of facts presented through the lens of the human capital model of health that links racial disparities in health to investments in health and the depreciation of health. It is these facts that theories of how racism adversely affects health need to reconcile to be convincing. For example, why would racism have an effect at one age and not another? Or similarly, why would racism have an effect at one age for one cause of mortality but not for another cause of mortality?

We make a few contributions to the extant literature on racial disparities in mortality. Ours is the first study that links the evolution of racial disparities in mortality over the life cycle to the economic model of the demand for health (Grossman 1972). Second, following the theoretical model, and differing from most previous studies, we use a flexible, nonparametric empirical specification that allows racial disparities in mortality to differ by age in a nonlinear, nonproportional way. Almost all past studies imposed strong assumptions about the lifecycle relationship of race and mortality with little theoretical justification. For example, a popular analytical choice is to use the Cox (or other) proportional hazard model to study racial disparities in mortality. However, this model assumes that racial disparities in mortality are a constant proportion of White mortality at all ages. Even studies that allow for there to be a non-constant proportional difference in racial disparities by age do so in a restrictive way that requires age to have a constant proportional effect by race (e.g., Yao and Robert 2011; Beydoun et al. 2016; Ng-Mak et al. 1999). Below, we show that these are statistically invalid restrictions. Third, although many studies have examined racial differences in mortality risk at specific ages, or age ranges, these point-in-time mortality differences reflect the cumulative effects of all prior health investments and depreciation. In this study, we measure racial differences in mortality *at* a specific age for all ages between ages 40 and 80. These estimates measure the effect of racism and other causes of mortality *at* that age and can be linked to the age-specific conceptual model of investments and depreciation at that age. We conduct analyses of three causes of death: diseases that can be effectively treated or prevented by healthcare resources including cardiovascular disease, stroke, COPD, influenza/pneumonia, diabetes, and kidney diseases (hereafter, referred to as healthcare amenable diseases), all cancers, and all other causes which include accidents, Alzheimer's, and other unspecified causes.

We find that from ages of 45 to 80, Black men and women have higher mortality rates than White men and women, respectively. The absolute racial difference in mortality *at* each age from ages 50 to 75

[8] We begin at age 40 because mortality is very low prior to this age.

[9] There may be different amounts of (gross) investment and depreciation, but net investment (gross investment minus depreciation) would be the same and there would be no change in the racial disparity in health between ages 60 and 65.

is positive, which is consistent with an effect of racism on mortality at that age, and remains relatively constant, if not declining from ages 50 to 75. The relative racial difference in mortality *at* each age declines, which is inconsistent with most prior empirical analyses that assume a proportional effect. Together, these results rule out the hypothesis that racism has an increasing effect on mortality over the lifecycle.

While racial disparities in mortality are positive at each age through age 75, the underlying causes of death driving those disparities shift during these ages and in distinct ways for males and females. For men in their fifties, the greatest contributor to the racial disparity in mortality is from causes of death other than healthcare amenable diseases or cancer. At subsequent ages, it is the growing disparities in healthcare amenable mortality and, to a lesser extent, cancer mortality that account for overall racial disparities in mortality for men. For women, racial disparities in healthcare amenable mortality account for almost all the disparity in mortality, as cancer and other causes of death are very small and do not contribute meaningfully to the racial disparity in all-cause mortality.

Varying theories about the causes of racial disparities in health and mortality can be evaluated against these set of facts. Our conceptual framework focuses on the changing quantity and quality of investments and depreciation with age. Viewed through this lens, results suggest that racism adversely affects younger, Black men's health behaviors, which causes racial disparities in mortality from other (e.g., external) causes of death that are less amenable to healthcare management. As people age, racial disparities in healthcare amenable causes of death suggests that racism limits the quantity and effectiveness of investments in treatments known to be effective. And the smaller role of racial disparities in cancer mortality throughout our analysis for both males and females is consistent with this explanation, as there are fewer and less effective treatments for cancer. Finally, for both males and females, adjustment for economic resources, and to a lesser extent health, mediators virtually eliminate disparities in mortality and the effects of these mediators grows with age. The growing impact of economic mediators with age is consistent with the hypothesis that racism's primary influence on mortality is through reduced investment including differential effectiveness and quality of treatment because of the strong link between education, income and investment (treatment).

Conceptual Framework--The Demand for Health

The human capital model of the demand for health (Grossman 1972) is the conceptual framework most often used in economics to study the determinants of health. In this model, health is a form of human capital that individuals augment through investments in health such as diet, nutrition, other health behaviors, and medical care. Investments in health come at a cost, as both the monetary and time resources needed for investment have alternative uses such as the forgone consumption of other goods and forgone returns on capital (e.g., interest). People invest in health because health is valued for itself—the value of being in good health—and because health influences income and other resources that generate value (e.g., consumption). Optimal investment in health occurs where the marginal benefit of investment equals the marginal cost of investment. Working in the opposite direction from investment is depreciation, for example, illness, that diminishes the stock of health capital (i.e., health). It is this process of investment and depreciation that causes the evolution of health over the life cycle.

The demand for health—i.e., the quantity of health capital at any age—can be derived using the optimal investment conditions, and following is a slightly revised version of the Grossman (1972) formulation of the demand for health capital at age t:

(1) $\frac{U_H}{\lambda} + Y_H = MC_{I(t-1)}(\delta_t + r) - \Delta MC_I$ [10]

The left-hand side of equation (1) represents the marginal benefit of a given level of health capital (H). The marginal benefit includes the monetary value of the utility (U_H / λ) and the greater income (Y_H) derived from the health stock (H). The right-hand side of equation (1) represents the marginal (user) cost of health capital—i.e., the opportunity cost of producing additional health (H). The marginal cost depends on: the price of health investment (p); the marginal product of investment in producing health (f_I); the rate of interest (r); the depreciation rate on stock of health (δ_t); and the change in MC_I (ΔMC_I) between ages t-1 and t. Despite the economic jargon, the intuition of these determinants of the marginal cost of health capital are not hard to understand. It costs money to invest in health and that money could be used for other purposes. The return on the money spent on investment depends on the productivity of investment. For example, the effectiveness of statins in reducing cardiovascular disease. The value of investment also depends on how fast health depreciates and erodes additional health from any investment. The optimal amount of health at age t is achieved when the marginal benefit of health (capital) is equal to its marginal cost.

In most formulations of the model, the only exogenous factors that vary with age (t) in equation (1) are the depreciation rate and MC_I. Depreciation of health generally grows with age, although this may not be the case with mental health. MC of investment may change, for example, because of price changes. To maintain the equilibrium in (1) with rising depreciation (i.e., aging), the level of health stock must decline with age so that $\frac{\partial U}{\partial H}$ and $\frac{\partial Y}{\partial H}$ increase. Eventually, as depreciation grows sufficiently large and investment too costly the health stock declines to a level where death occurs (Grossman 1972).

Most studies assume that the marginal cost of investment (MC_I) is constant with age, for example, because prices are assumed constant. However, this is unlikely to be the case and not just because prices are changing, but because the productivity of investment (f_I) is likely to change. Up to now, we have used the generic term investment to refer to activities and goods that augment health, but investments in health take many forms, such as nutrition, exercise and medical care. It is also likely that the types of investment in health change with age as the nature of the depreciation of health (illness) changes. For example, exercise may be more important at younger ages than older ages because the depreciation of health at younger ages may be due to moderate, slowly evolving biological effects of aging more than abrupt changes in health due to the onset of illness. Conversely, medical care may be more important at older ages than younger ages because the depreciation of health that occurs at older ages may be due to serious illness and be more amenable to medical care than exercise. Accordingly, the productivity of investments is likely to change with age simply because the types of investments change,

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[10] This formulation also appears in an early work in Kaestner et al. (2020). Similar to Grossman (1972), the lifetime utility function depends on health (H) and consumption (Z): $U = U(H_0,...,H_t,Z_0,...,Z_t)$. The production function of health depends on investment (I) and depreciation (δ): $H(t+1) = f(I_t) + [1-\delta(t)]H(t)$, $f_{It} = \frac{\partial f}{\partial I_t}$. The budget constraint is: $\sum_t \frac{p_t I_t + Z_t}{(1+r)^t} = \sum_t \frac{Y_t(H_t)}{(1+r)^t}$. The price of consumption is 1 and income is exogenous and depends on health ($Y_H = \frac{\partial Y}{\partial H}$). In equation (1), the following definitions are used:

$U_H = \frac{\partial U}{\partial H}(1+r)^t$, $MC_{It} = \frac{p_t}{f_{It}}$, $\Delta MC_I = MC_{I(t-1)} - MC_{It}$

but also because of other reasons such as the fact that older people are more experienced investors (made prior investments of same type).

If the productivity of investments changes with age, then the life cycle pattern of investment and health may differ significantly from that predicted based on a model with only an increasing rate of depreciation with age. For example, assume that the productivity of investment declines with age. Then the health stock will decrease more rapidly with age than predicted by a model with only depreciation rising with age. Alternatively, if the productivity of investment increases with age, then this increase in productivity will offset some, perhaps all, of the higher cost of investment associated with a rising depreciation rate with age and investment will be higher, and the health stock will decline more slowly with age, than in the model with just an increasing rate of depreciation with age. In this case, healthcare spending is more likely to increase, perhaps sharply, with age even as health deteriorates (i.e., when the depreciation rate is high). In fact, empirically, there is a strong negative correlation between health and health care spending that changes with age. Of course, how the productivity of investment changes with age is not really known and it may change non-monotonically with age.

Race, Investments in Health, Depreciation and Health

Before analyzing how investments in health, and thus health, differ by race and age, we note here that we view race as a social construct and as an indicator for exposure to racism both interpersonal and systemic. Like Vanderweele and Robinson (p. 5, 2014), we see race as a marker for "… biologic effects of skin color (e.g. darker skin protecting against ultraviolet light), the person's understanding of her skin color and other physical features and how this affects her identity and health behaviors, and also, importantly, how others react to the person's physical features, (e.g., discrimination or feelings of affinity)." This and similar conceptualizations address the debate in the causal inference literature that factors that cannot be manipulated such as sex and race cannot be causes (Holland 2003). In our view, racism is a cause that can theoretically be manipulated and race is a marker for racism. For the remaining parts of the article the reader should interpret our use of the word race as described above—as a marker for racism.

Racism may affect health through its effect on depreciation and investments in health. First, racism may have a direct biological effect that raises the rate of depreciation, for example, as hypothesized by Geronimus and colleagues (1992, 1996, 2006), and others (e.g., Jackson and Knight 2010; Berger and Sarnyai 2015; McEwen 2017). This would raise the marginal cost of health capital, lower the benefits of investment in health and reduce health relative to those unaffected by racism. Geronimus and colleagues (1992, 1996, 2006) hypothesized that the biological weathering due to racism will grow with age because of cumulative effects. Second, racism may lower the productivity of investment, for example, because of provider bias and differential treatment, or because of reduced access to high-quality providers (Hall et al. 2015; Maina et al. 2018). This too would raise the marginal cost of health capital investment, lower investment and reduce health. Another pathway linking racism to health is through income and lifetime wealth. Lower income and wealth due, for example, to discrimination, would reduce the benefits of health (or raise the marginal cost of health) because benefits come at the expense of a greater loss of utility from foregone consumption. As a result, there would be less investment and lower health. These potential pathways from racism to health, and others, are widely accepted across disciplines (e.g., Williams et al. 2019), but here expressed in terms of economic theory.

It is also likely that racism will affect health differently at different ages (Gee et al. 2012; Jones et al. 2020). This hypothesis stems from the likelihood that the types and productivity of investments in health change over the life cycle. For example, if racism has its most important influence on the productivity (quality or effectiveness) of medical care, which is an assumption, then as medical care grows more important to health, racial disparities stemming from racism will grow. In contrast, if racism

has its most important influence on health depreciation, then racial disparities in health will tend to be consistent and growing throughout the life cycle, although racism may have particularly large biological effects at some ages than others.

Measuring the Effect of Racism (Race) on Health over the Lifecycle

In this section, we outline how to measure the relationship between racism and health empirically. We focus on the health production function that relates investments in health and the depreciation rate to the stock of health (determined as in equation 1). The effect of racism on health is through investment and depreciation and therefore reflected in the production function.[11] One description of the health production function at age t is as follows:

$$(2) \quad H_t = H_0(1-\delta_0)...(1-\delta_{t-1}) + \alpha_0 I_0(1-\delta_1)...(1-\delta_{t-1}) + ... + \alpha_{(t-1)} I_{t-1}$$

In equation (2), health at age t is a function of initial health (H_0), the quantity of all past investments in health (I_0, …, I_{t-1}), the effects (productivity) of those investments (α_t) and the depreciation rate in each period (δ_0, …, δ_{t-1}). Health is determined by a cumulative process in which investments in health augment the stock of health and depreciation erodes the stock. Note that the increment to health from an investment, for example, medical care at a specific age, will differ for those at different ages because of depreciation.[12] The effect of an investment, α_t, may also differ by age because the productivity of an investment—the same investment and the same amount of investment—may be age-specific for biological reasons. For example, calorie and nutrient consumption at earlier ages may add more to health than at older ages because of differences in biological development at these ages, or for clinical reasons, for example, if effects of pharmacotherapy differ by age (Goldman and Lakdawalla 2005).

A point to note about equation (2) is that the health production function is age specific. One empirical implication of this is that it may be inappropriate to use samples of people of different ages without allowing the effects of (the same) investments to differ by age (for both reasons noted earlier). The upshot of this point is that the effect of racism on health is unlikely to be constant (proportional) over the life cycle.

The same approach applies to health at age t+1:

$$(3) \quad H_{t+1} = H_0(1-\delta_0)...(1-\delta_t) + \alpha_0 I_0(1-\delta_1)...(1-\delta_t) + ... + \alpha_{(t)} I_t$$

And the difference in health between ages t+1 and t is:

$$(4) \quad H_{t+1} - H_t = \alpha_t I_t - \delta_t H_t$$

The difference in health between two ages depends on net investment, which is the last period's investment ($\alpha_t I_t$) minus the extra depreciation ($\delta_t H_t$) that has occurred on past investments from being

[11] The following section draws heavily form a previously published article by Kaestner et al. 2020.

[12] To illustrate this, consider two people, one who is age 40 and one who is age 54. The health production function for these two individuals are given by:

$$H_{40} = H(1-\delta_0)...(1-\delta_{39}) + \alpha I_0(1-\delta_1)...(1-\delta_{39}) + \alpha I_1(1-\delta_2)...(1-\delta_{39}) + ... + \alpha I_{38}(1-\delta_{39}) + \alpha I_{39}$$

$$H_{54} = H(1-\delta_0)...(1-\delta_{53}) + \alpha I_0(1-\delta_1)...(1-\delta_{53}) + \alpha I_1(1-\delta_2)...(1-\delta_{53}) + ... + \alpha I_{52}(1-\delta_{53}) + ... + \alpha I_{53}$$

As shown, the increase in health caused by an investment at any age (e.g., 39) will differ for those age 40 and age 54 because depreciation of health capital between ages 40 to 54 has eroded the value of previous investments (e.g., age 39) for those age 54 more than for those age 40.

one year older. Note that if depreciation is zero then the difference in health between ages is just the addition to health from last period investment.

The effect of racism on health between age t+1 and age t is:

$$(5)\quad \frac{\partial(H_{t+1}-\partial H_t)}{\partial R}=\frac{\partial \alpha_t}{\partial R}I_t+\frac{\partial I_t}{\partial R}\alpha_t-\delta_t\frac{\partial H_t}{\partial R}-\frac{\partial \delta_t}{\partial R}H_t$$

As described by equation (5), the effect of racism (*R*) on health between two ages consists of the effect of racism on the productivity of health investment, on the amount of health investment at age t, on the initial (i.e., period t) stock of health at age t, and on the depreciation rate at age t.

An estimate of equation (5) can be obtained from a regression model such as the following:

$$H_{i(t+1)}-H_{it}=\pi_0+\pi_{t+1}RACE_i+[e_{i(t+1)}-e_{it}]$$

$$(6)\quad \pi_{t+1}=\frac{\partial \alpha_t}{\partial R}I_t+\frac{\partial I_t}{\partial R}\alpha_t-\delta_t\frac{\partial H_t}{\partial R}-\frac{\partial \delta_t}{\partial R}H_t$$

Note that the dependent variable in equation (6) is a first difference of health between two ages. The model, however, still includes race (*RACE*), which is age invariant. This is because race (racism) may modify the effect and quantity of investment in every period (racism has a time varying effect) and change depreciation in each period. The parameter estimate from the first-difference model measures the difference-in-difference effect of race (racism) on health *at* age t+1.

Estimates of π_{t+1} from equation (6) measures the racial difference in health between two ages (a difference-in-differences) and not necessarily the effect of racism. Only if all differences ($\frac{\partial \alpha_t}{\partial R},\frac{\partial I_t}{\partial R},\frac{\partial H_t}{\partial R},\frac{\partial \delta_t}{\partial R}$) in equation (6) are solely a result of racism is π_{t+1} an estimate of the effect of racism. We return to this point below.

Empirical Implementation

Studies of racial differences in health and mortality over the life cycle make several estimation choices and assumptions that often have little theoretical justification. For example, most choose to use a Cox (or other) proportional hazard model to estimate the association between race and mortality, which assumes that race has the same proportional difference on mortality at all ages. In addition, most studies do not allow the association between race and mortality to differ by age (Deshpande et al 2009; Feinstein et al. 2012; Signorello et al. 2014; Hummer and Chinn 2011; Louie et al. 2021; Luo et al. 2021), and when they do, they do so in the context of a proportional hazard model that assumes that the effect of age is linear (or quadratic) and a constant proportion (e.g., Yao and Robert 2011; Beydoun et al. 2016; Ng-Mak et al. 1999; Sautter et al. 2012; Howard et al. 2000).

We use a more flexible estimation approach that is consistent with our conceptual framework. Before describing that approach, we briefly describe the data we use. The data come from the National Health Interview Survey (NHIS) from 1997 to 2014 that linked respondents to the National Death Index (NDI), which provides the date of death for those who died by 2019. We limit the sample to non-Hispanic Black and non-Hispanic White individuals aged 40 to 70 at baseline (the time they were observed in the

NHIS survey) and stratify the analysis by sex. We describe the data in more detail below. With this sample, we estimate the following discrete-time, hazard regression model separately by sex:

$$(7)\quad H_{imkt} = \alpha_k + \lambda_t + \sum_{m=40}^{80} \gamma_m AGE_{imt} + \sum_{m=40}^{80} \beta_m (AGE_{imt} * BLACK_i) + e_{imkt}$$

In equation (7), for person *i* who was observed in NHIS survey year k, the hazard rate of dying at age *m* in calendar year *t* depends on: survey year dummy variables (α_k), calendar year dummy variables (λ_t), age dummy variables (*AGE*), an indicator that the person's race is Black (*BLACK*), and the interactions between age dummy variables and the indicator that the person's race is Black. Basically, equation (7) estimates the mean hazard rate of dying by age and race adjusted for survey year and calendar year.[13] We obtain estimates of equation (7) using ordinary least squares methods.[14]

From equation (7), the difference-in-differences (DiD) estimate of the association between race and the hazard rate of dying from age *m* to *m+1* ($\tilde{\pi}_{t+1}$, see equation 6) is:

$$(8)\quad \begin{aligned} \pi_{m+1} &= \frac{\partial \alpha_t}{\partial R} I_t + \frac{\partial I_t}{\partial R} \alpha_t - \delta_t \frac{\partial H_t}{\partial R} - \frac{\partial \delta_t}{\partial R} H_t \\ &= \beta_{m+1} - \beta_m \end{aligned}$$

Equation (8) shows the difference in the change in hazard rate of dying between ages *m+1* and *m* for Black vs. White people. It is a difference-in-differences estimate and can be obtained from estimates of equation (7).[15]

Equation (7) and (8) show the DiD estimates for each single age. To improve statistical precision, we use five-year age dummy variables, specifically, the age dummy variables for ages 41 to 45, 46 to 50, 51 to 55, 56 to 60, 61 to 65, 66 to 70, 71 to 75, and 76 to 80. The estimates on these 5-year-age dummy variables are very similar to estimates using one-year age dummy variables.

Additionally, to test the sensitivity of our estimates to the model specification, we estimate several alternative models that: 1) allow the effects of survey year and calendar year to vary by race (*BLACK*) by including their interactions with race in addition to their main effects; 2) use birth year fixed effects in place of the survey year fixed effects; and 3), use birth year fixed effects in place of the calendar year fixed effects. Estimates from these models are nearly identical and are presented in the Appendix.

Interpreting Estimates of the Association Between Race and the Hazard Rate of Dying over the Lifecycle

Equation (8) describes the components of the estimate of the association between race and the hazard rate of dying *at* a given age ($\pi_{m+1} = \beta_{m+1} - \beta_m$). Those components are the racial difference in the quantity of investment; the racial difference in the productivity of investment; the racial difference in the

[13] This specification implicitly controls for baseline age, which is completely determined by survey year, calendar year and age.

[14] The use of ordinary least squares (OLS) may be less efficient than other approaches (e.g., maximum likelihood logit) but only if the probability distribution of other approaches is correct, which is unknown. OLS also does not impose any proportionality restrictions.

[15] Note that the standard errors of difference-in-differences estimates account for the covariance of estimates across ages.

depreciation rate; and the racial difference in the baseline hazard rate of dying. The estimate of π_{m+1} can be positive indicating that race has a negative association with health at that age (i.e., higher hazard rate of dying); zero indicating that race has no association with health at that age; and negative indicating that race has a positive association with health at that age. A positive estimate is consistent with the hypothesis that racism adversely affects health at that age.

The empirical analyses we conduct produce a large set of estimates of the association between race and the hazard rate of dying by age (equation 8) including cause-specific estimates and separate estimates by sex. This collection of results can be informative about the underlying role of racism on mortality, particularly if viewed through the human capital model of health. For example, consider the finding reported below that, for "other" causes of death, estimates of the association between race and the hazard rate of dying are virtually zero after age 55 for males and zero from ages 40 to 80 for females. A conclusion consistent with this set of results is that, except for younger males, racism has almost no adverse effect on mortality of Black people from these causes of death even though these causes account for approximately one-third of all deaths. What theory of the effect of racism on health can explain this fact? Why would there be no differences by race in investment and depreciation related to these diseases, but racial differences in investment and depreciation for healthcare amenable diseases and, to a lesser extent, cancer? If we assume that depreciation is a general, biological phenomenon that affects frailty and susceptibility to many types of diseases in a similar way, then the cause-specific differences in racial differences in the hazard rate of dying are more likely to be the result of racial differences in investment than racial differences in depreciation.

The example just described provides an illustration of the value of our empirical analysis, and specifically, the value of conducting analyses by age, sex and cause of disease. It also illustrates the value of a conceptual model for interpreting empirical facts.

Mediation Analysis

The pathways linking racism to health include mediating effects of distal causes of health such as education, employment, financial status and health insurance among others. [16] There is a long tradition of assessing how much of racial disparities in health can be explained by these mediating factors (Kaufman and Copper 2001; Nuru-Jeter et al. 2018). Interpretation of the results of a mediation analysis, however, is difficult.

There are two extremes related to potential mediating pathways between racism and health. One is that racism is the sole cause of racial disparities in the mediating factors, for example, education. If so, then, if one can show a causal effect of education on health it is possible to measure the effect of racism on health that works through education. To do so, one multiplies the causal effect of education on health by the racial disparity in education to obtain the causal effect of racism on health that works through education. Or alternatively, measuring the change in the coefficient on race (Black) before and after adjusting for education measures the effect of racism on health that works through education (this is the traditional mediation analysis).[17] The other extreme is that racism has no causal effect on racial disparities

[16] Several studies of the Black-white disparity in mortality risk over the lifecycle have investigated the role of mediating factors such as education, income, or baseline health (Beydoun et al. 2016; Signorello et al. 2014; Duru et al. 2012; Sudano and Baker 2006). However, these studies typically, do not interact proposed mediators with age, and, as such, restrict the mediating effect on race to be unchanging over time.

[17] In this case, the unadjusted coefficient on race (without controls for mediating factors such as education) measures the overall effect of racism, while the adjusted race coefficient when education is included measures the effect of racism that work through channels other than education.

in mediating factors. If so, then adjusting for education and other confounding factors implies that the adjusted race coefficient measures the (direct) effect of racism on health.

The truth likely lies between these two extremes. It is undoubtedly true that racism causes racial disparities in education, income and other potential mediating factors. However, there is variation in education, income and other influences of health within homogenous populations defined by race and ethnicity, and even family income. If so, then there is a cause of variation in these mediating factors that is independent of race and racism. Some possibilities include the market economy (unemployment), public funding of schools, social support for families adversely affected by illness (mental, addiction) and other aspects of the social, political and economic environment. These causes of disadvantage do not originate solely from racism because we see them in populations that are arguably not affected by racism. If this view is correct, then the interpretation of the results from mediation analyses are ambiguous.

Despite the ambiguity, it remains true that the coefficient on race after adjusting for potential mediating factors measures the effect of racism on health other than through the mediating pathways.[18] Thus, for example, if the coefficient on race is zero, it suggests that much (all) of the effect of racism on health works through these mediating factors. Further, because mediating factors such as education and income affect mostly investment in health (e.g., medical care), changes in the coefficient on race after adjusting for these factors indicates how important racism is to the investment pathway. This may differ by cause of death and sex and providing a comprehensive set of results helps identify the most likely ways that racism is adversely affecting the health of Black people.

The mediation analysis we conduct proceeds along the same lines as described above. Specifically, we add controls for educational attainment (four categories); indicators for whether a person received income from interest, income from dividends, income from both interest and dividends, or income from neither interest nor dividends;[19] an indicator for whether a person had health insurance; an indicator that a person was employed; and an indicator that self-rated health was excellent or very good. All these potential mediating variables are measured at baseline and the association between these variables and the hazard rate of dying is allowed to vary by age. The augmented regression model is:

$$H_{imkt} = \alpha_k + \lambda_t + \sum_{m=40}^{80} \gamma_m AGE_{imt} + \sum_{m=40}^{80} \beta_m (AGE_{imt} * BLACK_i) + \sum_{m=40}^{80} \gamma_m (AGE_{imt} * EDUC_i) + e_{imkt} \quad (9)$$

Equation (9) reflects the addition of potential mediators as indicated by the inclusion of the variable *EDUC* in the model. To conserve on notation, we do not show explicitly all the additional variables. As noted, the association between these mediating variables and hazard rate of dying is allowed to vary by age. Therefore, we can assess where in the lifecycle, if at all, these factors mediate the effect of race. For example, Kaestner et al. (2020) present evidence that the beneficial effect of education on mortality does not appear until roughly age 55 and grows in magnitude after that age. This suggests that education is unlikely to mediate racial differences in mortality prior to age 55. Similar with Equation (7), in the main analysis, we use five-year age dummy variables instead of single-year age dummy variables.

Data

[18] Adjusting for education and income are imperfect controls for mediating pathways from racism to health because of measurement issues (Nuru-Jeter et al. 2018). Thus, the coefficient on race may still reflect some of these effects.
[19] Income is poorly measured in the NHIS and missing in many cases. Income from interest and/or dividends is highly correlated with income and have lower item nonresponse rates.

The data for the analysis is from the National Health Interview Survey (NHIS) linked with the National Death Index (NDI). The NHIS, is a large, nationally representative, cross-sectional annual household survey that collects information on demographic and socioeconomic characteristics, as well as a broad range of health topics, including measures of health and health insurance coverage. The NDI contains a national database of death records collected from state vital statistics offices. Death records up to 2019 were linked for respondents of the 1986-2018 NHIS surveys.

We use the 1997-2014 NHIS surveys because the NHIS went through a major redesign in 1997 and because linkage eligibility to the NDI changed from 2015 onward.[20] For our analysis, we focus on non-Hispanic White and non-Hispanic Black respondents aged 40 to 70 years at the time of the NHIS survey. To identify their mortality status, we limit the sample to those who were eligible to be linked to NDI (92.42% of the sample). The characteristics of the linked sample and the full NHIS sample are nearly identical for a wide range of characteristics including baseline health (Appendix Table 1), suggesting that our sample retains the national representativeness of the NHIS survey. In the linkage-eligible sample, those with a death record in the NDI are known to have died and those without are considered still alive in 2019. Using these data, we created a longitudinal data set that follows individuals from the time they were observed in the NHIS until the time of death, or if the person did not die until 2019 or age 80. We create an indicator variable for whether an individual died, which equals zero in the years the individual was still alive and one in the year the individual died. We excluded observations who died in the same year they were surveyed in the NHIS (e.g., everyone is alive in year 0) and those who had missing baseline characteristics. The average length of follow-up is 12 years.

Demographic (e.g., age, sex, race) and socioeconomic characteristics (e.g., education, employment, financial status), along with self-reported health status and health insurance coverage are all measured in the NHIS surveys. Based on the sources of income, we create a set of indicators to measure a person's financial status (whether the individual had no interest or dividend income, had interest income but no dividend income, had dividend income but no interest income, or had both interest and dividend income). Economic resources, such as education, employment, and financial status, are shown to be important determinants of health outcomes (Whitman et al. 2022). Thus, in the mediation analysis, we include indicator variables for whether an individual had a high school degree only, some college education, or a Bachelor's or greater education, whether they were employed, and our financial status indicators as proxy measures for economic resources.

For individuals who died, ICD-10 diagnostic codes were used to classify the cause of death into three groups: healthcare amenable diseases including cardiovascular disease, stroke, COPD, influenza/pneumonia, diabetes, and kidney diseases; all cancers; and other causes of death. Healthcare amenable diseases accounted for approximately 41% of the deaths, cancer for approximately 31% of the deaths, and other causes for approximately 28% of the deaths.[21]

[20] Prior to 2015, all NHIS respondents providing information on their social security number, name, sex, and date of birth were eligible to be linked to the NDI. From 2015 onward, only "sample adult" respondents (i.e., one selected adult respondent per household) providing this information were eligible.

[21] To protect respondent identity, the public linked mortality records include the use of synthetic data for NHIS respondents considered at risk for identification (NCHS 2019). This synthetic data either perturbed the year of death or the underlying cause of death, replacing original values with values from mortality records with similar characteristics. Importantly for our analysis, there is no measurable difference in all-cause mortality hazard ratios by age, sex, race and ethnicity, or education, or by age, sex, and education stratified by race and ethnicity, between the actual, unperturbed data and the synthetic data file.

Table 1 presents sample characteristics. For both males and females, the average baseline age is approximately 51 and slightly older for White people than Black people, which likely reflects the lower mortality rate among the former. There are substantial racial disparities in education attainment, being employed, and financial status. For example, Black males are 16 percentage points less likely to have a Bachelor's degree or greater education than White males, while Black females are 11 percentage points less likely than White females to have a Bachelor's degree or greater education. Black males are 10 percentage points less likely to be employed, although the corresponding difference for females is two percentage points. Both Black males and females are approximately 30 percentage points less likely to have interest or dividend income. As for baseline health, both Black males and females report worse health than their White counterparts. At follow-up, approximately 18.8% of Black males died before or at age 80 compared to 15.4% of White males, and 13.7% of Black females died compared to 10.9% of White females. Correspondingly, the mean of the variable death indicator in the longitudinal data set equals 0.017 for Black males, 0.013 for White males, 0.012 for Black females, and 0.009 for White females.

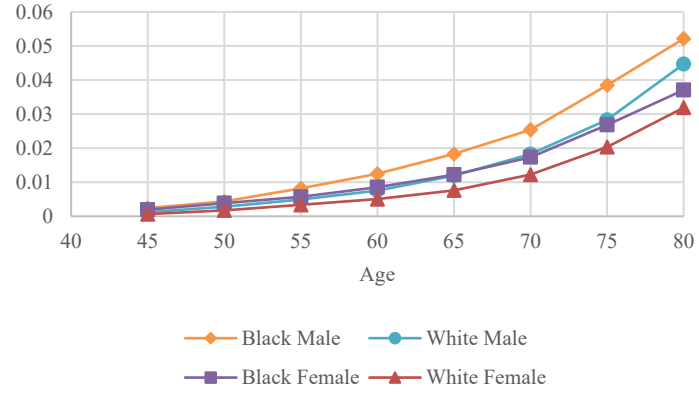
Results

Hazard Model Estimates

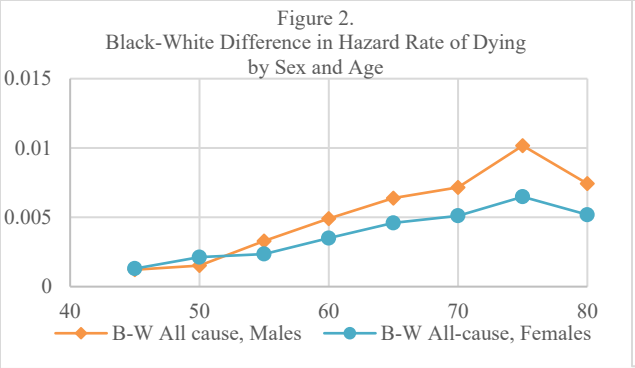
The first results we present are the hazard rate of dying from all causes by race and sex. We report the predicted hazard rate by age from a discrete-time, OLS regression after adjusting for the survey year that the person was observed in the NHIS data and the calendar year of the observation. The unit of observation in these analyses was a person-year.

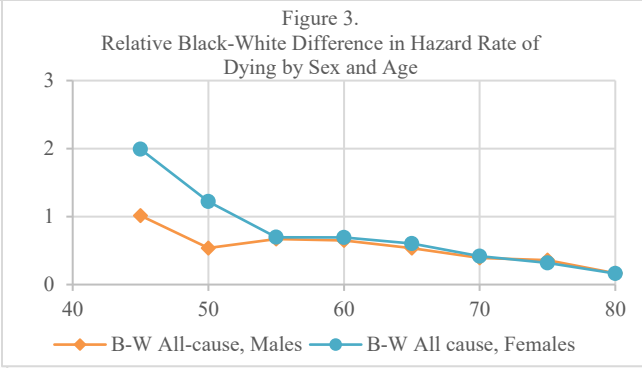
Figure 1 shows the predicted hazard rates. The hazard rate has the typical relationship with age starting very low at age 45, rising to one percentage point around age 60, and then rising to around three to five percentage points by age 80 depending on the person's sex and race. The hazard rate of dying is higher for males than for females and is higher for Black people than for White people. Of note, the hazard rate of dying for Black females is approximately equal to that of White males—there is no mortality disadvantage of Black females relative to White males.

Figure 1.
All-cause Hazard Rate of Dying by Sex, Race and Age

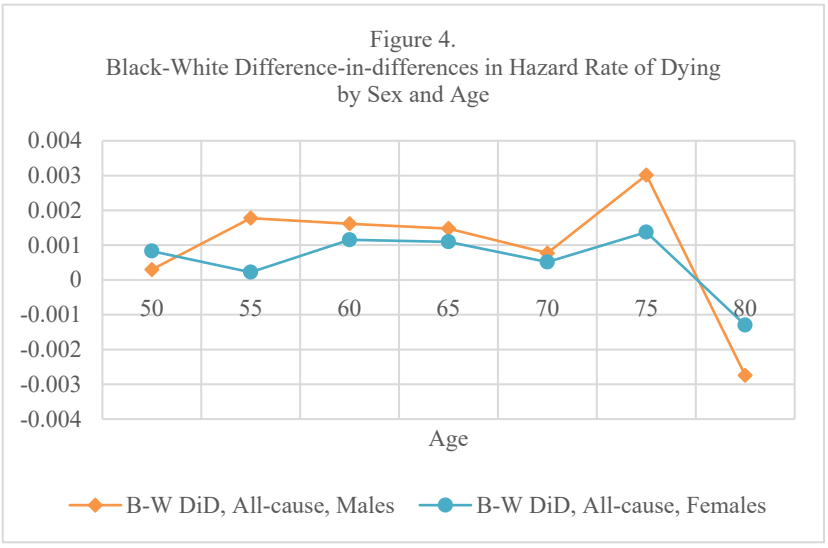


Next, we show the Black-White differences in the predicted hazard rate of dying by sex and age. We show the absolute differences in Figure 2 and the relative (to White) differences in Figure 3. For both males and females, the absolute racial difference in the hazard rate of dying increases with age up to age 75 but at a generally decreasing rate, and the racial difference is larger for males than females. However, the relative racial difference in the hazard rate of dying (Figure 3) decreases with age for both males and females and is approximately the same for males and females from age 55 onward. The declining proportional racial disparities in mortality are inconsistent with standard applications of proportional hazard regression models that assume covariates (i.e., race) have a constant, proportional effect on the hazard rate at all ages.





Changes in the racial differences in the hazard rate of dying by age can most clearly be observed using estimates of the racial difference-in-differences (DiD) in the hazard rate of mortality. DiD estimates measure the racial difference in the hazard rate of dying between two ages and, at least partly, of the effect of racism *at* each age. Figure 4 presents the DiD estimates by sex and age. Estimates in Figure 4 indicate that the racial DiD estimate in the hazard rate of dying for males is positive and statistically significant at age 55 (see column (1) in Appendix Table 2), indicating that the racial difference in the hazard rate grows between ages 50 and 55.[22] This result suggests that racism influences health at this age, but not before this age (from 45 to 50). DiD estimates for males remain positive and mostly statistically significant from ages 55 to 70 but decline by almost 50% in relative terms over these ages. From age 70 to 80, DiD estimates increase and decrease sharply with the age 80 estimate being imprecisely estimated and statistically insignificant.



A similar pattern characterizes racial difference-in-differences (DiD) in the hazard rate of mortality among females, although DiD estimates are smaller, statistically significant only at ages 60 and 65, and the pattern is shifted to the right by five years.

Overall, DiD estimates in Figure 4 suggest that racism is adversely affecting the health of Black females at each age roughly from ages 55 to 75, although effects are mostly statistically insignificant, and that its influence is declining modestly over this age range.

Conceptually, racism adversely affects health by decreasing the quantity and productivity (effectiveness) of investments in health and by raising the rate of depreciation on health. The DiD estimates in Figure 4 suggest that racism is causing a decrease in net investment in health, which is investment net of depreciation, by a roughly similar (slightly declining) absolute magnitude at each age after age 50. Net investment in health is smaller for Black than White people between ages 55 and 75 and

[22] In Appendix Table 2, we provide estimates from three alternative specifications including: allowing the effect of calendar year and survey year to vary by Black; using birth year in place of survey year; using birth year in place of calendar year. The estimates are nearly identical.

the racial difference in the hazard rate of dying grows with age. The smaller amount of net investment by Black people is approximately constant from ages 60 to 75 for females, and approximately constant or slightly declining from ages 55 to 70 for males.

How can this pattern be explained by racism? At a fundamental level, the patterns found in Figure 4 must be explained by the effect of racism on investment in health and depreciation. One theory, or at least what we will refer to as the strong version of that theory, that seems to be ruled out by the patterns in Figure 4 is that racism has an increasingly negative effect on health as people age, as suggested by theories of cumulative advantage (Dannefur 2003; Di Prete and Eirich 2006) and weathering (Geronimus et al. 2006). As implied by estimates in Figure 4 and shown in Figure 2, the racial difference in the hazard rate of dying is growing with age, as reflected in positive DiD estimates, but the growth is smaller in absolute and relative terms with age. In short, racism does not appear to be having a growing effect on racial disparities as people age.

Understanding the cause of the DiD estimates is central to identifying how racism impacts Black people's health over the life course. Is it through decreased investment? Greater depreciation? Or both? And why do we not observe much of an effect prior to age 50? It is helpful for thinking about how racism affects Black people's health to recognize that the incidence of major causes of death—heart disease including stroke (CVD), cancer and chronic obstructive pulmonary disease (COPD)—grow with age although the share of deaths from these diseases remains roughly constant with age.[23] For example, cancer accounts for approximately 30% of deaths at ages 45 and 75. These three causes of death account for approximately 60% of all deaths. The growing incidence of these diseases with age increases the importance of treatments of these diseases (investments), which also grow with age. For example, hypertension grows with age (depreciation) as does the use of anti-hypertensive medications (investments). If racism was mainly inhibiting treatment, or the effectiveness of treatment, of these diseases, it is likely that DiD estimates would reflect this and become larger with age as the scope for treatment and amount of treatment becomes greater. For example, if provider bias limited the effectiveness of treatment for Black people, then the increasing amount of treatment with age would imply an increasing racial difference with age. Similarly, if reduced economic resources because of racism was causing less investment (treatment) by Black people, then we may expect an increasing racial difference with age as the amount of treatment grows.

This is not what we observe. The roughly constant DiD estimates in Figure 4 indicate a roughly constant increment to the racial difference in the hazard rate of dying by age, for example, a 0.002 higher increase in the hazard rate for Black people at each age. If this racial difference in the growth of the hazard rate by age was due to reduced investment or the effectiveness of investment, then it implies that there is a constant racial difference in the amount of effective investment despite the known rising quantity of investment with age. In sum, if the explanation of racial differences in mortality is because racism causes less investments, then the pattern of estimates in Figure 4 suggests that Black people's investments in health increase with age the same as it does for White people but at each age fall a bit short of the level of investment by White people.

An alternative explanation of the pattern in Figure 4 is that racism adversely affects depreciation of health and causes Black people to experience a greater depreciation of health at each age. Again, however, the patterns in Figure 4 imply that the higher increment of depreciation of health experienced by Black people as they age is roughly constant, if not declining, despite the growing amount of depreciation that comes with age (i.e. growing mortality rate). The patterns in Figure 4, if to be explained by depreciation, suggest that racism causes Black people's health to decline by a fixed amount more than White people from ages 55 to 75. For example, if White people's hazard rate of dying grows by 0.004

[23] Authors' calculations from NHIS data.

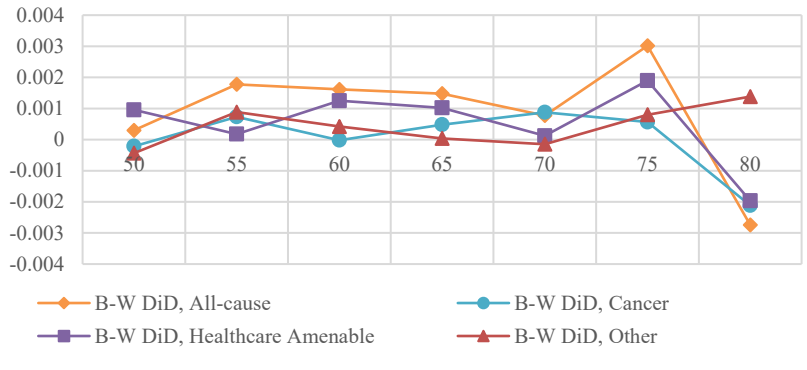
between ages 60 and 65, then Black people's hazard rate of dying grows by 0.006 between those ages. Similarly, if White people's hazard rate of dying grows by 0.006 between ages 65 and 70, then Black people's hazard rate of dying grows by 0.008 between those ages. The difference in the depreciation rate, 0.002, does not grow with age.

Finally, with respect to Figure 4, there is a significant racial convergence of mortality between ages 75 and 80 for both males and females. At these ages, Black people have a mortality advantage and racial disparities in the hazard rate of dying diminish markedly. However, we note that the DiD estimates at age 80 are quite imprecise. The Black mortality advantage is referred to in the literature as crossover and one explanation for it is selective mortality (Markides and Machalek 1984; Yao and Robert 2011; Wing et al. 1985; Roth et al. 2017; Fenelon 2014; Arias 2019). To test whether the (statistically insignificant) crossover observed in Figure 4 is consistent with selective mortality — whether unmeasured frailty (depreciation) is relatively low for Black people at later ages because of higher mortality rates at earlier ages, resulting in a Black mortality advantage at later ages — we conducted an analysis where we examined how the hazard rate of dying differed by age when first observed in NHIS. Specifically, we estimated a discrete-time, hazard model controlling for calendar year fixed effects, birth year fixed effects, and a full set of interactions between indicators for baseline age, indicators for duration, and an indicator for race. We then predict the hazard rate of dying by baseline age, age at follow-up, and race, holding constant the birth year and the calendar year.

Evidence consistent with selective mortality would be that the hazard rate of dying at any given age would be lower for those observed at an older age at baseline—those at older ages have "made it" to that age and are likely to be healthier than those first observed at younger ages. We find limited evidence of selective mortality for White people and even less evidence of selective mortality for Black people. This finding suggests that selective mortality is unlikely to explain the (statically insignificant) crossover observed.

Next, we show DiD estimates by cause of death. We begin with males. Figure 5 presents estimates for males. We divided the causes of death into three groups: healthcare amenable diseases including cardiovascular diseases, stroke, COPD, diabetes, kidney diseases, and influenza/pneumonia related causes; cancer; and other causes (e.g., accidents, Alzheimer's, or other unspecified causes).

Figure 5.
Black-White Difference-in-differences of Hazard Rate of Dying by Cause and Age, Male Sample

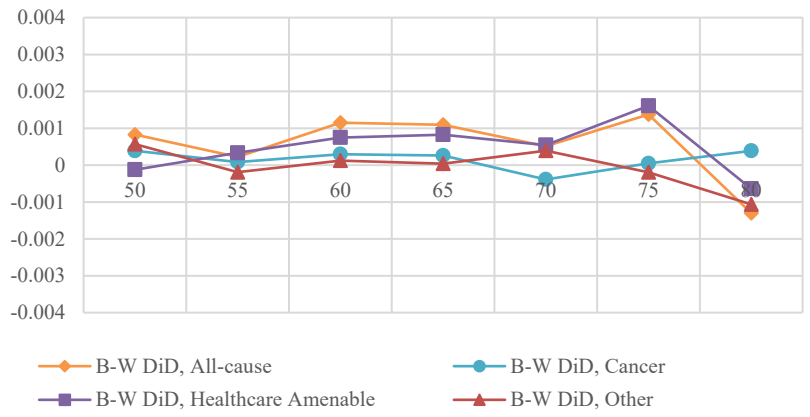


There is a clear pattern to the estimates. Prior to age 60 (ages 51 to 55 specifically), the growing racial disparity in all-cause mortality (Figures 2 and 4) is mainly caused by a statistically significant racial disparity in other causes of death (see Appendix Table 3). From ages 60 to 75, racial differences in other causes of mortality have virtually no influence on the overall racial disparity in mortality. Rather, the growing racial disparity in all-cause mortality after age 60 is caused by growing racial disparities in healthcare amenable diseases and cancer, with the racial disparity in healthcare amenable causes of death having more of an influence about five years earlier than cancer. However, most of the estimates related to cancer mortality are relatively small and not statistically

significant (see column (3) in Appendix Table 3). It is notable that except for age 70, the racial disparity in cancer mortality is insignificant even though cancer mortality accounts for approximately one-third of all deaths at all ages.

Figure 6 presents DID estimates by cause of death for females. As previously noted, DID estimates of racial disparities in mortality are relatively small and most are not statistically significant. For females, racial disparities in other causes of death are virtually zero except for age 75. And cancer deaths also contributed little to the racial disparity in mortality at all ages. The growing racial disparity in all-cause mortality with age (Figures 2 and 4) from ages 60 to 75 are mostly a result of significant racial disparities in healthcare amenable mortality.

Figure 6.
Black-White Difference-in-differences of Hazard Rate of Dying by Cause and Age, Female Sample



How can racism explain the patterns found in Figures 5 and 6? For example, among males, racial disparities in other causes of death account for much of the overall racial disparity in mortality at ages 51-55. For females, this is not the case, and in fact, racial disparities in other causes of death account for very little of the overall racial disparity in mortality at any age. The largest single component of other causes of death are external causes including accidents, injuries, homicides, drug overdoses and suicide. Males (of any race) have higher rates of suicide, drug abuse, homicides and accidents and injuries than females. Thus, there is more scope for racism to impact these causes of death and racism may be the cause of these higher rates for males. In addition, external causes of death are not very treatable and investments to reduce the probability of experiencing such deaths are primarily personal health behaviors. Thus, DiD estimates for males in Figure 5 suggest that racism adversely affects health behaviors of males that lead to early deaths from these causes in their fifties. This interpretation is consistent with the hypothesis and evidence provided by Jackson and colleagues (Jackson et al. 2010; Jackson et al. 2011). The results in Figure 5 suggest that racism does not have the same impact on Black female health behaviors. If racism is the explanation for these findings, then it must be that racism is more virulent for younger males than older males and females.

Personal health behaviors (e.g., diet) also have a relatively strong association with deaths from healthcare amenable diseases but medical treatments for these diseases are also quite effective. DID estimates in Figures 5 and 6 indicate that racial disparities in healthcare amenable causes of death become larger *at* older ages. This pattern is consistent with a racism explanation in which racism limits the amount of investment, both personal (e.g., diet) and medical (e.g., anti-hypertensive use), in health. This interpretation is consistent with some evidence that shows that racial differences in healthcare spending grow modestly with age (Charron-Chenier and Mueller 2018; Dieleman et al. 2021; Baik et al. 2024). Cancer deaths, on the other hand, are not strongly related to personal health behaviors other than smoking, and effective treatment primarily means early detection. Therefore, estimates in Figures 5 and 6 showing that racial disparities in cancer become more important as Black people age, although racial disparities in cancer deaths contribute relatively little to overall racial disparities in mortality, are consistent with a racism explanation focused on treatment—screening and early detection. However, there

are relatively small differences in rates of screening for cancer by race, which are consistent with the small racial disparities in cancer mortality (American Association for Cancer Research 2024).

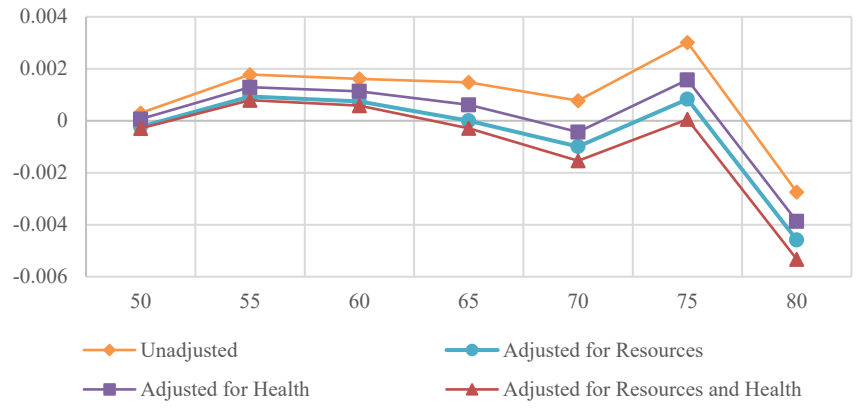
Taking stock, estimates shown in Figures 2, 4, 5 and 6, indicate that racial disparities in mortality grow with age, but at a decreasing rate. They also indicate that while racial disparities in mortality are present at ages 55 to 75, the underlying causes of those disparities change over the life course and do so in slightly different ways for males and females. While estimates in Figure 4 suggest a somewhat constant increment to racial disparities in male mortality between ages 55 and 75, estimates in Figures 5 reveal that the source of those disparities is changing, for example, racial disparities in other causes of death (e.g., external) contribute most to the overall racial disparity in male mortality at younger ages (e.g., 55). And the increasing racial disparities in male healthcare amenable mortality with age and, to a much lesser extent, cancer deaths, as the incidence of these illnesses grows and the scope for treatment of these illnesses grow is consistent with an explanation that racism adversely affects investments in health and has a larger influence as the amount of investment grows with age. A similar pattern characterizes racial disparities in female mortality, although racial disparities in other causes of death and cancer make little meaningful contribution to overall racial disparities in female mortality. Finally, the racial convergence of hazard rates of dying that is apparent for nearly all causes of death and for males and females at older ages (e.g., 75) are not consistent with the evidence we found on selective mortality. It is likely that estimates at these ages are too imprecise to be informative.

Mediation Analysis Estimates

Figures 2 through 6 presented evidence of racial disparities in mortality *at* individual ages that are not adjusted for any racial differences in well-known correlates of health such as education. We now present estimates of racial disparities after adjusting for several potential mediating and/or confounding factors. As discussed earlier, it is not clear whether correlates of mortality such as education are mediators of the effect of racism on mortality or confounders of the association of racism on mortality. Despite this ambiguity, adjusting for correlates of mortality help identify how much of the racial disparity in mortality is net of these influences.

Figure 7 presents the Black-White difference-in-differences (DID) estimates of hazard rate of dying from all causes by age for males from four models: a model that does not adjust for any covariates (same as Figure 5); a model that adjusts for baseline education, employment, receipt of income from interest or dividends , and health insurance coverage, which we refer to as resources; a model that adjusts for baseline self-reported health, which we refer to as health; and a model that adjusts for both resources and health. Figure 7 presents the estimates in graphical form and Appendix Table 4 shows coefficient estimates and their standard errors.

Figure 7.
Black-White Difference-in-differences in Hazard Rate of Dying by Age
All-Cause Mortality, Male Sample



There are a few notable patterns in Figure 7. First, adjustment for potential mediators has a growing impact on racial disparities in health as people age. Second, adjustment for resources has a slightly larger impact than adjustment for health. Third, after adjusting for both resources and health, there are no statistically significant racial disparities in all-cause mortality and most estimates are quite small— less than 13% of the White mean. This finding suggests (but does not prove) that virtually all the effect of racism, to the extent it matters, works through these mediating factors.

Figure 8.
Black-White Difference-in-differences in Hazard Rate of Dying by Age
All-Cause Mortality, Female Sample

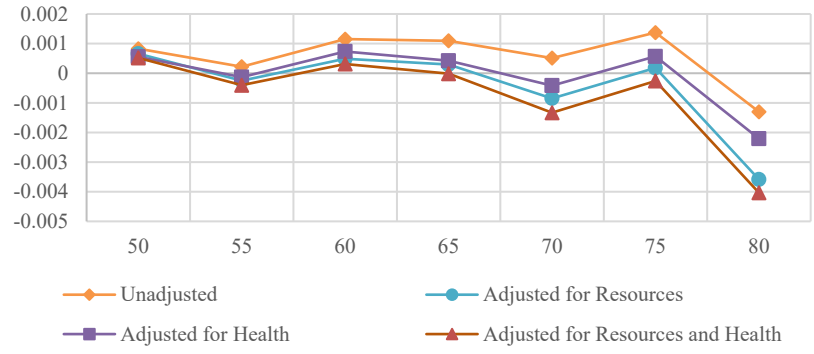
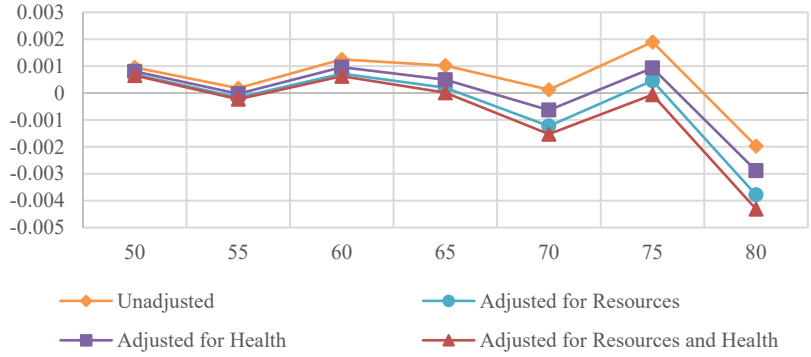


Figure 8 shows analogous estimates for females and the patterns are similar to those for males, although the disparities are approximately half the size as they are for men (although proportionally similar). After adjusting for mediating factors, racial disparities in all-cause mortality are zero or negative at every age except 60. For this sample too, all the effect of racism, to the extent it matters, works through these mediating factors. However, we note that, even before we adjust for potential mediators, there were few statistically significant racial disparities in mortality among females.

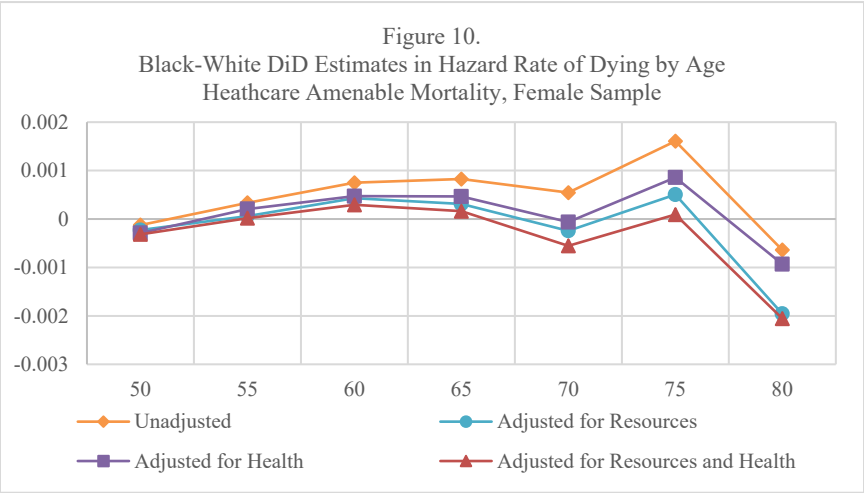
Figure 9 presents the unadjusted and adjusted Black-White difference-in-differences (DID) estimates of hazard rate of dying from healthcare amenable causes by age for males (corresponding estimates and standard errors are reported in Appendix Table 5). Not surprisingly, given that disparities in healthcare amenable mortality are the most important component of overall racial disparities, the impact of adjusting for economic and health mediators has a similar impact as described above.

The impacts of adjustment for mediating factors grow with age and largely eliminate racial disparities in healthcare amenable mortality at all ages except 50.

Figure 9.
Black-White DiD Estimates in Hazard Rate of Dying by Age
Healthcare Amenable Mortality, Male Sample

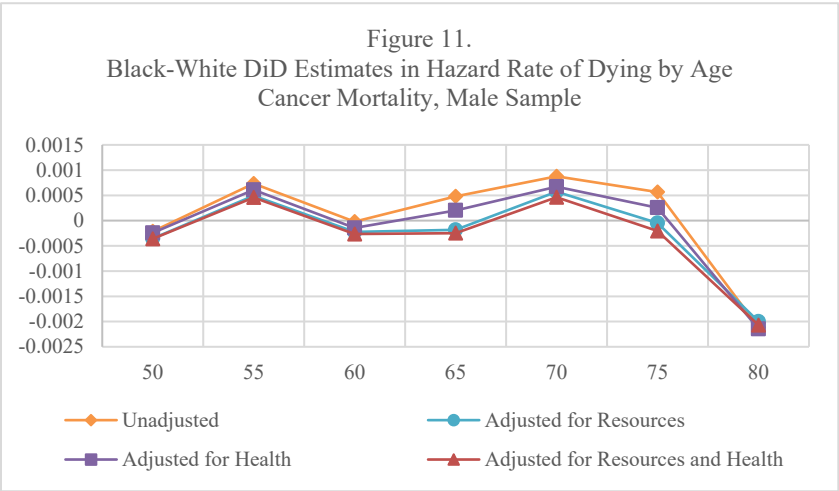


Estimates of unadjusted and adjusted Black-White difference-in-differences estimates of racial disparities in healthcare amenable mortality of females are shown in Figure 10. As with males, adjustments for resources and health have a growing impact with age, and each set of variables have about the same effect. Absent any adjustment, only the disparities at ages 60 and 70 were statistically significant, and after adjustment none of the disparities are statistically significant and the estimates are small in magnitude – approximately 25% of the White mean (Appendix Table 5).



As documented in Figures 5 and 6, racial disparities in healthcare amenable mortality are the most important component of overall racial disparities and racial disparities in these causes of death tend to grow with age. This is the case for both males and females. Also, adjustments for resources (education, income and health insurance) and health (self-rated health) have about the same impact on racial disparities in healthcare amenable mortality and tend to have a growing impact with age. It is reasonable to assume that socioeconomic variables such as education and income are most closely linked to the ability to invest in health, for example, through the quantity and quality of treatment. Given that the importance of treatment of healthcare amenable diseases grows with age because of the growing incidence of these diseases with age and the proven effectiveness of some treatments (e.g., statins and anti-hypertensive drugs), the growing impact of socioeconomic variables on racial disparities by age suggests (not prove) that much of the disparity is because of racial disparities in socioeconomic factors and differential treatment caused by socioeconomic disparities. In the case of adjustments for health, it is reasonable to assume that these variables are most closely linked to the frailty and depreciation of health. The growing impact of health variables on racial disparities by age suggests (not prove) that a smaller share of the disparity is because of racial disparities in depreciation.

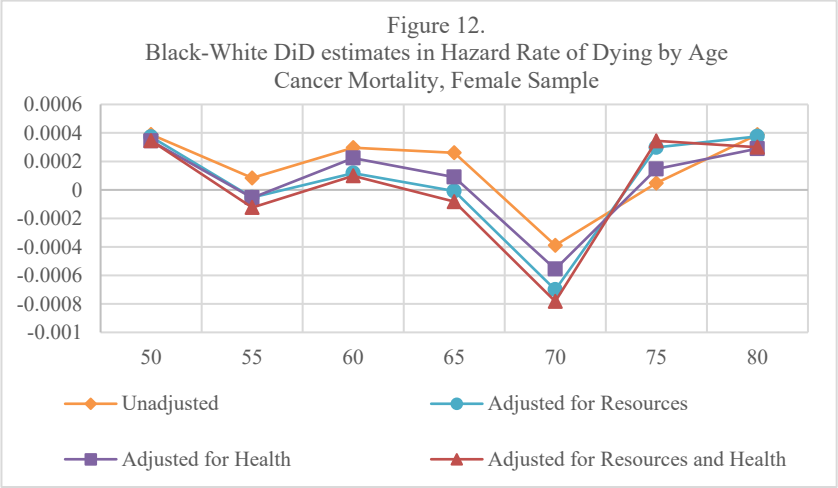
Racism is plausibly linked to both racial disparities in economic mediators and health mediators. So, while there is not much evidence of an effect of racism (race) net of these mediators, racism may be the fundamental cause of the racial disparities in these mediators. And the pattern of results is consistent with an explanation in which racism affects both the quality and quality of investment—through resource mediators—and, to a lesser extent, the rate of depreciation—through health mediators.[24] Of course, how much of the racial disparities in these mediators is caused by racism is largely unknown. There is clear evidence of racial bias in the hiring process (e.g., Kline et al. 2024), but there is no similar causal evidence related to broader measures of socioeconomic standing such as earnings and education.



Adjusted and unadjusted Black-White difference-in-differences estimates pertaining to cancer mortality are shown in

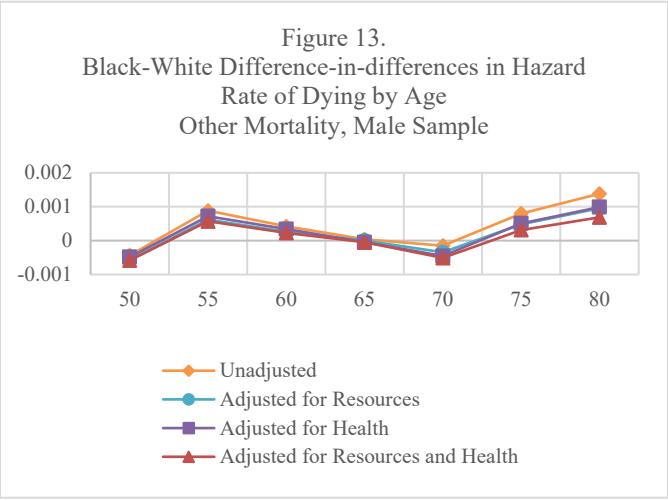
[24] Racial disparities in baseline health may reflect racial disparities in economic mediators that influenced baseline health, but there are quite small racial disparities in mortality prior to age 50.

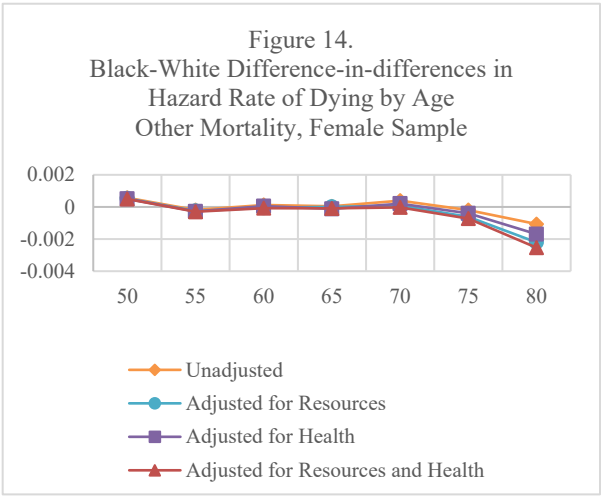
Figures 11 (males) and 12 (females). For both males and females, racial disparities in cancer mortality are relatively small and all but one of the Black-White DiD estimates are not statistically significant even before adjustment for potential mediators (Appendix Table 6). Race, and thus racism, does not seem to be an important influence on cancer mortality.



The absence of significant racial disparities in cancer mortality is an interesting fact. It is consistent with the etiology of cancer and the limited knowledge about the causes (as opposed to risk factors) of the most prevalent cancers (e.g., breast, prostate, colon, lung) other than lung cancer. Cancer mortality, as compared to cardiovascular mortality, seems to be more influenced by genetic factors than treatment and behavior. If so, then the small racial disparities in cancer mortality would be expected because racism influences health investments (health behaviors and treatment) and, beyond screening and early detection, treatment for cancers are less effective (life extending) than treatments for cardiovascular disease. Moreover, there are small racial disparities in screening rates (Islami et al. 2022).

The final set of results we present are unadjusted and adjusted Black-White difference-in-differences estimates for other causes of death (Figures 13 and 14; Appendix Table 7). As discussed above, there were few significant estimates of racial disparities in other causes of death (Appendix Table 2, column 4). It was only for males when they were in their fifties that significant racial disparities were found. Given the generally small, insignificant unadjusted effects, adjusting for potential mediators has little effect.





Conclusion

In this article, we extended the human capital model of the demand for health by Grossman (1972), which links age-specific investments and age-specific depreciation to the change in health from

one age to the next, to study the lifecycle relationship between race and mortality. Our analyses show that racial disparities in mortality are primarily due to disparities in healthcare amenable causes of death and that racial disparities in cancer and other causes of death are generally small and not statistically significant with two exceptions—death from other (external) causes at age 55 and cancer mortality at age 70 among males. This is a notable finding given that cancer and other causes of death account for approximately half of all deaths. At each age from ages 40 to 80, absolute differences in racial disparities in healthcare amenable mortality are positive, which indicates that racial disparities are growing at those ages, although by a roughly constant, if not declining amount, and relative differences in racial disparities in healthcare amenable mortality are positive but declining. These results indicate that, to the extent that racism is adversely affecting this cause of mortality, it is doing so at all ages from ages 40 to 75 by limiting the amount and/or productivity of investment or exacerbating health depreciation by a similar, or even slightly decreasing, amount at each age. There is no evidence that, with respect to mortality, racism has an increasingly important impact over the lifecycle.

The relatively constant increment to racial disparities in mortality as people age can viewed in the context of the substantial decrease in death rates that have occurred over recent decades. While racial disparities persist, there has been a substantial decline in death rates for both Black and White persons and some relative improvement in death rates of Black versus White people. Racism has not prevented Black people from benefiting from the general declines in death rates. Similarly, the results we presented indicate that racism, if present, is limiting, not preventing, investment (treatment) in health and/or the efficacy of that investment. As illness and disease grow with age, the use of healthcare also grows and becomes more central to maintaining health. Treatment grows for both Black and White people, but the increase in racial disparities in mortality at each age suggest that at each age Black people are falling short in the amount and efficacy of treatment relative to their White counterparts.

Data on racial disparities in the use of healthcare offers some auxiliary evidence to our findings. For example, Dickman et al. (2022) and Deilman et al. (2021) reported that after adjustment for age and other factors (sex in Dickman et al. 2022), Black adults used less ambulatory care but more emergency room and inpatient care than White adults. The lower use of ambulatory care is consistent with our findings and the explanation that it is through less investment that racial disparities persist. On the other hand, there were few racial disparities in prescription drug use (Dielman et al. 2022) or in non-financial barriers to access (Caraballo et al. 2020; Caraballo et al. 2022). In addition, as previously noted, racial disparities for treatments for hypertension, hyperlipidemia, diabetes and some cancer screenings are relatively small (Aggarwal et al. 2021; Lu et al. 2023; CDC 2024; American Association for Cancer Research 2024). These facts along with our results suggest that racism, to the extent it is having an influence, is likely reducing the productivity (effectiveness) of treatment (investment), and to a lesser extent, the quantity of investment, particularly for cardiovascular and other healthcare amenable diseases (Lu et al. 2023). It also merits noting that the quantity and efficacy of treatment stems from both personal and provider actions. Racism may affect both, but the implications for effective interventions to address this source of disparity differ by whether it is personal or provider actions that limit the quantity and effectiveness of investment.

Racial differences in economic resources and health at baseline explain virtually all the racial differences in mortality due to healthcare amenable causes (or other causes) and the mediating effect of these variables grows with age, which is consistent with the growing incidence of these diseases with age and the coincident growing importance of effective treatment of these diseases. Overall, results of the analysis suggest that, to the extent that racism is a cause of racial disparities in mortality, its effect is declining with age and is working primarily through economic resources that at each age from ages 50 to 75 reduce personal and medical investments in healthcare amenable diseases of Black relative to White people.

One policy implication of our findings and our hypothesized explanation is to broaden programs that reduce racial disparities in the efficacy of high-value treatment, for example, as illustrated by the well-known barbershop experiment (Victor et al. 2019) that focused on treatment of hypertension, or the initiative in Delaware that largely eliminated racial disparities deaths due to colon cancer (Grubbs et al. 2013). Notably, in both examples, the successful intervention focused mostly on the individual and determinants of personal health behaviors.

Unfortunately, there are relatively few other programs with demonstrated success at reducing racial disparities. Here is a quote from Willimas (2012) in a an aptly titled article "Miles to Go before We Sleep: Racial Inequities in Health":

> "Despite thousands of published studies, our current knowledge is limited with regard to the most effective strategies to reduce health inequities, and there is an urgent need to develop a science base to guide societal interventions." (Willimas 2012, p. 279)

While written over a decade ago, William's conclusion remains applicable today. Specific, successful interventions to significantly reduce racial disparities in health are not readily available. And while our results also suggest that economic resources have significant effect on racial disparities in mortality, progress on improving Black economic status has been slow and meaningful change may require large-scale social changes that are not easily brought about.

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Tables

Table 1.
Mean Sample Characteristics by Sex and Race

	Male		Female	
	Black	White	Black	White
Baseline Characteristics				
Baseline age	51.52	52.13	51.53	52.38
Education				
Less than a high school degree	0.17	0.08	0.17	0.07
High school degree	0.38	0.31	0.36	0.34
Some college	0.27	0.27	0.31	0.30
Bachelor's degree or greater education	0.17	0.33	0.17	0.28
Employed	0.69	0.79	0.64	0.66
Financial status:				
Had no interest or dividend income	0.82	0.52	0.85	0.54
Had interest but no dividend income	0.11	0.22	0.10	0.22
Had dividend but no interest income	0.02	0.04	0.01	0.03
Had both interest and dividend income	0.05	0.22	0.04	0.20
Self-reported very good to excellent health	0.48	0.64	0.42	0.62
Had health insurance	0.82	0.90	0.83	0.91
At follow-up				
Share of individuals who died before age 81	0.188	0.154	0.137	0.109
Number of individuals	30,121	164,540	39,918	174,252
Number of observations in follow-up	339,422	1,975,314	464,454	2,135,241
Died (=1 in the year died only, =0 otherwise)	0.017	0.013	0.012	0.009

Notes: the sample includes White and Black survey respondents aged 40 to 70 years old in the 1997 to 2014 NHIS surveys (i.e., baseline), who were followed until their death or 2019, or age 80, whichever occurs first. Observations with missing values for baseline characteristics were dropped.

Appendix Table 1.
Sample Characteristics for the NHIS Sample and the NDI Link-eligible Sample

Baseline Characteristics	NHIS sample	NHIS sample eligible for linkage to NDI
Baseline Age	53.13	53.18
Male	0.48	0.48
White	0.83	0.83
Education		
Less than a high school degree	0.10	0.10
High school degree	0.33	0.33
Some college	0.29	0.29
Bachelor's degree or greater education	0.28	0.28
Employed	0.68	0.67
Financial Status		
Had no interest or dividend income	0.62	0.62
Had interest but no dividend income	0.18	0.19
Had dividend but no interest income	0.03	0.03
Had both interest and dividend income	0.17	0.17
Self-reported very good to excellent health	0.57	0.57
Had health insurance		
Number of NHIS respondents	430,614	408,831

Notes: the NHIS sample includes White and Black survey respondents aged 40 to 70 years old in the 1997 to 2014 NHIS surveys. Among them, those providing sufficient identifying information were eligible for linkage to the NDI. The analysis sample includes individuals who were eligible to be linked to NDI. Observations with missing values for baseline characteristics were dropped.

Appendix Table 2. All-cause Mortality
Black-White Difference-in-differences of Hazard Rate of Dying by Age and Sex

Panel A. Male					
Age Group	Baseline White All-Cause Mortality Rate	(1): Main Model	(2): Alternative Model 1	(3): Alternative Model 2	(4): Alternative Model 3
50 (46-50)	0.0038	0.0003	0.0005	0.0003	0.0003
		(0.0006)	(0.0006)	(0.0006)	(0.0006)
55 (51-55)	0.0057	0.0018**	0.0021**	0.0018**	0.0018**
		(0.0005)	(0.0005)	(0.0005)	(0.0005)
60 (56-60)	0.0082	0.0016**	0.0018**	0.0016**	0.0016**
		(0.0006)	(0.0006)	(0.0006)	(0.0006)
65 (61-65)	0.0125	0.0015**	0.0016**	0.0015**	0.0015**
		(0.0007)	(0.0007)	(0.0007)	(0.0007)
70 (66-70)	0.0188	0.0008	0.0008	0.0008	0.0008
		(0.0010)	(0.0010)	(0.0010)	(0.0010)
75 (71-75)	0.0288	0.0030**	0.0031**	0.0031**	0.0031**
		(0.0015)	(0.0015)	(0.0015)	(0.0015)
80 (76-80)	0.0450	-0.0027	-0.0024	-0.0026	-0.0026
		(0.0025)	(0.0025)	(0.0025)	(0.0025)
Panel B. Female					
Age Group	Baseline White All-Cause Mortality Rate	(1): Main Model	(2): Alternative Model 1	(3): Alternative Model 2	(4): Alternative Model 3
50 (46-50)	0.0023	0.0008	0.0012**	0.0008	0.0008
		(0.0004)	(0.0005)	(0.0004)	(0.0004)
55 (51-55)	0.0038	0.0002	0.0004	0.0002	0.0002
		(0.0004)	(0.0004)	(0.0004)	(0.0004)
60 (56-60)	0.0054	0.0012**	0.0013**	0.0012**	0.0012**
		(0.0004)	(0.0004)	(0.0004)	(0.0004)
65 (61-65)	0.0080	0.0011**	0.0012**	0.0011**	0.0011**
		(0.0005)	(0.0005)	(0.0005)	(0.0005)
70 (66-70)	0.0126	0.0005	0.0005	0.0005	0.0005
		(0.0007)	(0.0007)	(0.0007)	(0.0007)
75 (71-75)	0.0206	0.0014	0.0014	0.0014	0.0014
		(0.0010)	(0.0010)	(0.0010)	(0.0010)
80 (76-80)	0.0321	-0.0013	-0.0012	-0.0013	-0.0013
		(0.0018)	(0.0018)	(0.0018)	(0.0018)
Calendar Year FE		Yes	Yes	Yes	
Survey Year FE		Yes	Yes		Yes
Birth Year FE				Yes	Yes
Calendar Year by Black			Yes		
Survey Year by Black			Yes		

Notes: This table shows the Black-White difference-in-differences estimate in the hazard rate of dying over 5-year age groups. For example, the estimate for Age group 50 represents the Black-White difference-in-differences in the hazard rate of dying from ages 41-45 to 46-50; and the estimate for Age group 55 represents the Black-White difference-in-differences in the hazard rate of dying from ages 46-50 to 51-55; the corresponding baseline White mortality rate for Age group 50 is the mortality rate among Whites for ages 41-45. All models also control for the main effect of being Black, age dummy variables, and the interaction terms between age dummy variables and being Black. " <0.0001" indicates that the absolute value of the estimate is less than <0.0001. "**" indicates p-value <0.05.

Appendix Table 3. Mortality by Cause:
Black-White Difference-in-differences of Hazard Rate of Dying by Age and Sex

Panel A. Male					
Age Group	Baseline White All-Cause Mortality Rate	(1) All-cause	(2): Healthcare Amenable Diseases	(3): Cancer	(4): Other causes
50 (46-50)	0.0038	0.0003	0.0010**	-0.0002	-0.0004
		(0.0006)	(0.0004)	(0.0003)	(0.0004)
55 (51-55)	0.0057	0.0018**	0.0002	0.0007**	0.0009**
		(0.0005)	(0.0003)	(0.0002)	(0.0003)
60 (56-60)	0.0082	0.0016**	0.0012**	<0.0001	0.0004
		(0.0006)	(0.0004)	(0.0003)	(0.0003)
65 (61-65)	0.0125	0.0015**	0.0010**	0.0005	<0.0001
		(0.0007)	(0.0005)	(0.0004)	(0.0004)
70 (66-70)	0.0188	0.0008	0.0001	0.0009	-0.0001
		(0.0010)	(0.0007)	(0.0006)	(0.0005)
75 (71-75)	0.0288	0.0030**	0.0019	0.0006	0.0008
		(0.0015)	(0.0010)	(0.0009)	(0.0007)
80 (76-80)	0.0450	-0.0027	-0.002	-0.0021	0.0014
		(0.0025)	(0.0018)	(0.0014)	(0.0014)
Panel B. Female					
Age Group	Baseline White All-Cause Mortality Rate	(1) All-cause	(2): Healthcare Amenable Diseases	(3): Cancer	(4): Other causes
50 (46-50)	0.0023	0.0008	-0.0001	0.0004	0.0006**
		(0.0004)	(0.0003)	(0.0002)	(0.0003)
55 (51-55)	0.0038	0.0002	0.0003	0.0001	-0.0002
		(0.0004)	(0.0002)	(0.0002)	(0.0002)
60 (56-60)	0.0054	0.0012**	0.0007**	0.0003	0.0001
		(0.0004)	(0.0003)	(0.0003)	(0.0002)
65 (61-65)	0.0080	0.0011**	0.0008**	0.0003	<0.0001
		(0.0005)	(0.0003)	(0.0003)	(0.0003)
70 (66-70)	0.0126	0.0005	0.0005	-0.0004	0.0004
		(0.0007)	(0.0005)	(0.0004)	(0.0003)
75 (71-75)	0.0206	0.0014	0.0016**	<0.0001	-0.0002
		(0.0010)	(0.0007)	(0.0006)	(0.0005)
80 (76-80)	0.0321	-0.0013	-0.0006	0.0004	-0.0011
		(0.0018)	(0.0012)	(0.0009)	(0.0010)

Notes: This table shows the Black-White difference-in-differences estimate in the hazard rate of dying over 5-year age groups. For example, the estimate for Age group 50 represents the Black-White difference-in-differences in the hazard rate of dying from ages 41-45 to 46-50; and the estimate for Age group 55 represents the Black-White difference-in-differences in the hazard rate of dying from ages 46-50 to 51-55; the corresponding baseline White mortality rate for Age group 50 is the mortality rate among Whites for ages 41-45. "Healthcare Amenable Diseases" includes cardiovascular diseases, strokes, COPD, diabetes, influenza/pneumonia, and nephritis. "Other" diseases includes accidents, Alzheimer's, and unspecified causes of death. The model for each column controls for survey year effects and calendar year effects in addition to the main effect of being Black, age dummy variables, and the interaction terms between age dummy variables and being Black. " <0.0001" indicates that the absolute value of the estimate is less than <0.0001. "**" indicates p-value <0.05.

Appendix Table 4. All-cause Mortality:
Black-White Difference-in-differences of Hazard Rate of Dying by Age and Sex

Panel A. Male					
Age Group	Baseline White Mortality Rate	(1): Baseline	(2): (1) + Resources	(3): (1) + Health	(4): (1) + Resources + Health
50 (46-50)	0.0038	0.0003	-0.0002	0.0001	-0.0003
		(0.0006)	(0.0006)	(0.0006)	(0.0006)
55 (51-55)	0.0057	0.0018**	0.0009	0.0013**	0.0008
		(0.0005)	(0.0005)	(0.0005)	(0.0005)
60 (56-60)	0.0082	0.0016**	0.0007	0.0011	0.0006
		(0.0006)	(0.0006)	(0.0006)	(0.0006)
65 (61-65)	0.0125	0.0015**	<0.0001	0.0006	-0.0003
		(0.0007)	(0.0008)	(0.0007)	(0.0008)
70 (66-70)	0.0188	0.0008	-0.001	-0.0004	-0.0015
		(0.0010)	(0.001)	(0.001)	(0.001)
75 (71-75)	0.0288	0.0030**	0.0008	0.0016	0.0001
		(0.0015)	(0.0015)	(0.0015)	(0.0015)
80 (76-80)	0.0450	-0.0027	-0.0046	-0.0039	-0.0053**
		(0.0025)	(0.0026)	(0.0026)	(0.0026)
Panel B. Female					
Age Group	Baseline White Mortality Rate	(1): Baseline	(2): (1) + Resources	(3): (1) + Health	(4): (1) + Resources + Health
50 (46-50)	0.0023	0.0008	0.0007	0.0006	0.0005
		(0.0004)	(0.0005)	(0.0004)	(0.0004)
55 (51-55)	0.0038	0.0002	-0.0003	-0.0001	-0.0004
		(0.0004)	(0.0004)	(0.0004)	(0.0004)
60 (56-60)	0.0054	0.0012**	0.0005	0.0007	0.0003
		(0.0004)	(0.0004)	(0.0004)	(0.0004)
65 (61-65)	0.0080	0.0011**	0.0003	0.0004	<0.0001
		(0.0005)	(0.0006)	(0.0005)	(0.0006)
70 (66-70)	0.0126	0.0005	-0.0008	-0.0004	-0.0013
		(0.0007)	(0.0007)	(0.0007)	(0.0007)
75 (71-75)	0.0206	0.0014	0.0002	0.0006	-0.0003
		(0.001)	(0.0011)	(0.0011)	(0.0011)
80 (76-80)	0.0321	-0.0013	-0.0036	-0.0022	-0.0040**
		(0.0018)	(0.0019)	(0.0018)	(0.0019)

Notes: This table shows the Black-White difference-in-differences estimate in the hazard rate of dying over 5-year age groups. For example, the estimate for Age group 50 represents the Black-White difference-in-differences in the hazard rate of dying from ages 41-45 to 46-50; and the estimate for Age group 55 represents the Black-White difference-in-differences in the hazard rate of dying from ages 46-50 to 51-55; the corresponding baseline White mortality rate for Age group 50 is the mortality rate among Whites for ages 41-45. Models including "Resources" include baseline indicators for employment, education, and income from dividends or interest and insurance coverage. . Models with "Health" controls include baseline indicators for self-reported health status and insurance coverage. For all models, the inclusion of each control includes both main effects and interactions with each age group. "<0.0001" indicates that the absolute value of the estimate is less than <0.0001. "**" indicates p-value <0.05.

Appendix Table 5. Healthcare Amenable Mortality
Black-White Difference-in-differences Estimates of Hazard Rate of Dying by Age and Sex

Panel A. Male					
Age Group	Baseline White Mortality Rate	(1): Baseline	(2): (1) + Resources	(3): (1) + Health	(4): (1) + Resources + Health
50 (46-50)	0.0013	0.0010**	0.0007	0.0008**	0.0007
		(0.0004)	(0.0004)	(0.0004)	(0.0004)
55 (51-55)	0.0020	0.0002	-0.0002	<0.0001	-0.0002
		(0.0003)	(0.0004)	(0.0003)	(0.0003)
60 (56-60)	0.0032	0.0012**	0.0007	0.0010**	0.0006
		(0.0004)	(0.0004)	(0.0004)	(0.0004)
65 (61-65)	0.0050	0.0010**	0.0002	0.0005	<0.0001
		(0.0005)	(0.0005)	(0.0005)	(0.0005)
70 (66-70)	0.0081	0.0001	-0.0012	-0.0006	-0.0015
		(0.0007)	(0.0007)	(0.0007)	(0.0007)
75 (71-75)	0.0130	0.0019	0.0005	0.0009	-0.0001
		(0.0010)	(0.0011)	(0.0010)	(0.0011)
80 (76-80)	0.0204	-0.002	-0.0038**	-0.0029	-0.0043**
		(0.0018)	(0.0018)	(0.0018)	(0.0019)
Panel B. Female					
Age Group	Baseline White Mortality Rate	(1): Baseline	(2): (1) + Resources	(3): (1) + Health	(4): (1) + Resources + Health
50 (46-50)	0.0007	-0.0001	-0.0002	-0.0003	-0.0003
		(0.0003)	(0.0003)	(0.0003)	(0.0003)
55 (51-55)	0.0012	0.0003	0.0001	0.0002	<0.0001
		(0.0002)	(0.0002)	(0.0002)	(0.0002)
60 (56-60)	0.0017	0.0007**	0.0004	0.0005	0.0003
		(0.0003)	(0.0003)	(0.0003)	(0.0003)
65 (61-65)	0.0028	0.0008**	0.0003	0.0005	0.0002
		(0.0003)	(0.0004)	(0.0004)	(0.0004)
70 (66-70)	0.0049	0.0005	-0.0002	-0.0001	-0.0006
		(0.0005)	(0.0005)	(0.0005)	(0.0005)
75 (71-75)	0.0085	0.0016**	0.0005	0.0009	0.0001
		(0.0007)	(0.0008)	(0.0007)	(0.0008)
80 (76-80)	0.0138	-0.0006	-0.0020	-0.0009	-0.0021
		(0.0012)	(0.0013)	(0.0013)	(0.0013)

Notes: This table shows the Black-White difference-in-differences estimate in the hazard rate of dying over 5-year age groups. For example, the estimate for Age group 50 represents the Black-White difference-in-differences in the hazard rate of dying from ages 41-45 to 46-50; and the estimate for Age group 55 represents the Black-White difference-in-differences in the hazard rate of dying from ages 46-50 to 51-55; the corresponding baseline White mortality rate for Age group 50 is the mortality rate among Whites for ages 41-45. "Healthcare Amenable Diseases" includes cardiovascular diseases, strokes, COPD, diabetes, influenza/pneumonia, and nephritis. "Other" diseases includes accidents, Alzheimer's, and unspecified causes of death. Models including "Resources" include baseline indicators for employment, education, and income from dividends or interest and insurance coverage. . Models with "Health" controls include baseline indicators for self-reported health status and insurance coverage. For all models, the inclusion of each control includes both main effects and interactions with each age group. " <0.0001" indicates that the absolute value of the estimate is less than <0.0001. "**" indicates p-value <0.05.

Appendix Table 6. Cancer Mortality:
Black-White Difference-in-differences of Hazard Rate of Dying by Age and Sex

Panel A. Male					
Age Group	Baseline White Mortality Rate	(1): Baseline	(2): (1) + Resources	(3): (1) + Health	(4): (1) + Resources + Health
50 (46-50)	0.0008	-0.0002	-0.0004	-0.0002	-0.0004
		(0.0003)	(0.0003)	(0.0003)	(0.0003)
55 (51-55)	0.0015	0.0007**	0.0005	0.0006**	0.0005
		(0.0002)	(0.0003)	(0.0002)	(0.0003)
60 (56-60)	0.0025	<0.0001	-0.0002	-0.0001	-0.0003
		(0.0003)	(0.0003)	(0.0003)	(0.0003)
65 (61-65)	0.0044	0.0005	-0.0002	0.0002	-0.0002
		(0.0004)	(0.0004)	(0.0004)	(0.0004)
70 (66-70)	0.0064	0.0009	0.0006	0.0007	0.0005
		(0.0006)	(0.0006)	(0.0006)	(0.0006)
75 (71-75)	0.0094	0.0006	<0.0001	0.0003	-0.0002
		(0.0009)	(0.0009)	(0.0009)	(0.0009)
80 (76-80)	0.0132	-0.0021	-0.0020	-0.0021	-0.0021
		(0.0014)	(0.0014)	(0.0014)	(0.0014)
Panel B. Female					
Age Group	Baseline White Mortality Rate	(1): Baseline	(2): (1) + Resources	(3): (1) + Health	(4): (1) + Resources + Health
50 (46-50)	0.0008	0.0004	0.0004	0.0003	0.0003
		(0.0002)	(0.0002)	(0.0002)	(0.0002)
55 (51-55)	0.0015	0.0001	-0.0001	-0.0001	-0.0001
		(0.0002)	(0.0002)	(0.0002)	(0.0002)
60 (56-60)	0.0021	0.0003	0.0001	0.0002	0.0001
		(0.0003)	(0.0003)	(0.0003)	(0.0003)
65 (61-65)	0.0032	0.0003	<0.0001	0.0001	-0.0001
		(0.0003)	(0.0003)	(0.0003)	(0.0003)
70 (66-70)	0.0047	-0.0004	-0.0007	-0.0006	-0.0008
		(0.0004)	(0.0004)	(0.0004)	(0.0004)
75 (71-75)	0.0068	<0.0001	0.0003	0.0001	0.0003
		(0.0006)	(0.0006)	(0.0006)	(0.0006)
80 (76-80)	0.0082	0.0004	0.0004	0.0003	0.0003
		(0.0009)	(0.0010)	(0.0009)	(0.001)

Notes: This table shows the Black-White difference-in-differences estimate in the hazard rate of dying over 5-year age groups. For example, the estimate for Age group 50 represents the Black-White difference-in-differences in the hazard rate of dying from ages 41-45 to 46-50; and the estimate for Age group 55 represents the Black-White difference-in-differences in the hazard rate of dying from ages 46-50 to 51-55; the corresponding baseline White mortality rate for Age group 50 is the mortality rate among Whites for ages 41-45. Models including "Resources" include baseline indicators for employment, education, and income from dividends or interest and insurance coverage. . Models with "Health" controls include baseline indicators for self-reported health status and insurance coverage. For all models, the inclusion of each control includes both main effects and interactions with each age group. " <0.0001" indicates that the absolute value of the estimate is less than <0.0001. "**" indicates p-value <0.05.

Appendix Table 7. Other Mortality:
Black-White Difference-in-differences of Hazard Rate of Dying by Age and Sex

Panel A. Male					
Age Group	Baseline White Mortality Rate	(1): Baseline	(2): (1) + Resources	(3): (1) + Health	(4): (1) + Resources + Health
50 (46-50)	0.0017	-0.0004	-0.0006	-0.0005	-0.0006
		(0.0004)	(0.0004)	(0.0004)	(0.0004)
55 (51-55)	0.022	0.0009**	0.0006	0.0007**	0.0006
		(0.0003)	(0.0003)	(0.0003)	(0.0003)
60 (56-60)	0.0026	0.0004	0.0003	0.0003	0.0002
		(0.0003)	(0.0004)	(0.0003)	(0.0004)
65 (61-65)	0.0032	<0.0001	<0.0001	<0.0001	<0.0001
		(0.0004)	(0.0004)	(0.0004)	(0.0004)
70 (66-70)	0.0045	-0.0001	-0.0003	-0.0005	-0.0005
		(0.0005)	(0.0005)	(0.0005)	(0.0005)
75 (71-75)	0.0070	0.0008	0.0005	0.0005	0.0003
		(0.0007)	(0.0008)	(0.0007)	(0.0008)
80 (76-80)	0.0128	0.0014	0.0010	0.001	0.0007
		(0.0014)	(0.0015)	(0.0014)	(0.0015)
Panel B. Female					
Age Group	Baseline White Mortality Rate	(1): Baseline	(2): (1) + Resources	(3): (1) + Health	(4): (1) + Resources + Health
50 (46-50)	0.0008	0.0006**	0.0005	0.0005	0.0005
		(0.0003)	(0.0003)	(0.0003)	(0.0003)
55 (51-55)	0.0012	-0.0002	-0.0003	-0.0003	-0.0003
		(0.0002)	(0.0002)	(0.0002)	(0.0002)
60 (56-60)	0.0016	0.0001	-0.0001	<0.0001	-0.0001
		(0.0002)	(0.0002)	(0.0002)	(0.0002)
65 (61-65)	0.002	<0.0001	<0.0001	-0.0001	-0.0001
		(0.0003)	(0.0003)	(0.0003)	(0.0003)
70 (66-70)	0.0031	0.0004	0.0001	0.0002	<0.0001
		(0.0003)	(0.0004)	(0.0004)	(0.0004)
75 (71-75)	0.0056	-0.0002	-0.0006	-0.0004	-0.0007
		(0.0005)	(0.0006)	(0.0005)	(0.0006)
80 (76-80)	0.0108	-0.0011	-0.0022**	-0.0017	-0.0025**
		(0.0010)	(0.0010)	(0.0010)	(0.0010)

Notes: This table shows the Black-White difference-in-differences estimate in the hazard rate of dying over 5-year age groups. For example, the estimate for Age group 50 represents the Black-White difference-in-differences in the hazard rate of dying from ages 41-45 to 46-50; and the estimate for Age group 55 represents the Black-White difference-in-differences in the hazard rate of dying from ages 46-50 to 51-55; the corresponding baseline White mortality rate for Age group 50 is the mortality rate among Whites for ages 41-45. Models including "Resources" include baseline indicators for employment, education, and income from dividends or interest and insurance coverage. . Models with "Health" controls include baseline indicators for self-reported health status and insurance coverage. For all models, the inclusion of each control includes both main effects and interactions with each age group. " <0.0001" indicates that the absolute value of the estimate is less than <0.0001. "**" indicates p-value <0.05.