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WHY IS THERE GREATER VARIANCE IN THE LIFE SPANS OF BLACKS THAN OF WHITES IN THE UNITED STATES?

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WHY IS THERE GREATER VARIANCE IN THE LIFE SPANS OF BLACKS THAN OF WHITES IN THE UNITED STATES?

The elimination of racial and ethnic health disparities is a primary goal of U.S. health policy (U.S. Department of Health and Human Services 2011). One of the most significant racial disparities in health is the difference in the life expectancies of non-Hispanic blacks and non-Hispanic whites (hereafter simply "blacks" and "whites"). Life expectancy is shorter (Kochanek et al. 2013), and variance in age at death is greater (Tuljapurkar and Edwards 2011), for blacks than it is for whites in the United States. In other words, not only is the length of life shorter, on average, for blacks than for whites, there is also greater variance in the life spans of blacks. We call this black-white difference in variance in the age at death the *racial variance gap*.[1]

This study investigates the sources of the racial variance gap. As we see subsequently, there is relatively little research on this issue. Yet a deeper understanding of the sources of the racial variance gap would inform policy on alleviating this important racial health disparity. Life itself has value, so inequality in the length of life is a fundamental type of inequality (Tuljapurkar [2011] calls it "the final inequality"). Although inequality in the length of individuals' lives may be, to some extent, biologically ordained, we assume that *black-white differences* in that inequality are not (life expectancy patterns for Hispanics in the United States, for example, are closer to whites than to blacks). If that assumption is correct, reduction of the black-white variance gap is not beyond the reach of public policy.

Why, then, is there greater inequality in the longevity of blacks than in the longevity of whites in the United States? To find out, we use a new decomposition method (Nau and Firebaugh 2012) that enables researchers to investigate how specific causes of death contribute to group differences in the variability of longevity. The Nau-Firebaugh method partitions group differences in variance into three components: a *spread component*, which is the part of the variance gap due to differences in within-cause variability; an *allocation component*, which captures the contribution of the differences in cause-specific incidence in the two populations;

[1] By the scale-invariance property of inequality measures (Allison 1978), a measure of dispersion such as variance serves as a measure of inequality when distributions have the same mean. In the current study, mean age at death differs by less than five percent, so one can think of the racial variance gap here as capturing (approximately) the difference in the inequality of the life spans of blacks versus whites.

and a *timing component*, which is the part of the variance gap due to differences in the average age of death for victims of each cause (the more divergent the average age at death across causes, the greater the overall heterogeneity of life spans in that population).

From prior research we know that women and men both contribute to the racial disparity in life expectancy: White women tend to outlive black women and white men tend to outlive black men (Harper, Rushani and Kaufman 2012). However, we do not know to what extent, and how, differences in the longevity of women and men contribute to the black-white variance gap. In the current study we expand on Nau and Firebaugh (2012) to decompose the two dominant components of the variance gap – spread and allocation – by sex to gain further insight into the processes that generate the racial variance gap.

The paper is organized as follows. First we review the limited literature on racial differences in life span variance and inequality. Next we present equations for the Nau-Firebaugh (2012) decomposition, and extend them to show how the spread and allocation components can be further partitioned by sex. We then present results for each of 17 critical causes of death in 2010. After reporting black-white differences in the timing and incidence of each of the causes of death, we assess the contribution of each of the causes to the racial variance gap. As we will see, the spread and allocation components account for virtually all the difference in the life span variability of blacks and whites in the United States, with the spread component contributing the most. A major finding is that for *all* major causes of death – causes that account for at least 2% of all deaths – there is greater variability in age at death among blacks than there is among whites. In the next section we break the results down further by sex. We conclude by summarizing our findings and suggesting next steps for future research

PRIOR RESEARCH

Inequality in the length of life is one of the most fundamental manifestations of health disparities in the U.S. (Edwards and Tuljapurkar 2005; Engelman, Canudas-Romo and Agree 2010). Despite its significance, there is little research on racial differences in this inequality. Studies of differences in longevity have focused on the first moment of longevity – average age at death – while largely ignoring variance in age at death, the second moment (Edwards, in press). In the case of racial differences, first moments tell us very little about second moments, since substantial differences in inequality exist among populations with similar levels of life

expectancy (Edwards and Tuljapurkar, 2005; Smits and Monden 2008). In short, studies of life expectancy provide little insight about inequality in the survival of individuals (Engelman et al. 2010).

We therefore know relatively little about the black-white gap in the variance of longevity. Only a few studies have measured the racial variance gap directly. Using the interquartile range to measure the gap, Weden (2007) finds greater variability in longevity among nonwhites than among whites throughout the 20th century. The nonwhite-white difference in variability increased during the first three decades of the century, and has declined since, albeit at a slower rate more recently. Based on data from the Berkeley Mortality Database, Lynch, Brown and Harmsen (2003) also find evidence of modest convergence in the inequality of life spans for the white and black populations in America. In another study of the racial variance gap, Go, Brustrom, Lynch and Aldwin (1995) find, based on vital statistics for California and census data for 1970, 1980, and 1990, that blacks exhibited greater variation in age at death than whites, but lower variation than Hispanics. In contrast, Lariscy, Nau, Firebaugh and Hummer (2013) find – based on more recent nationally-representative data – that Hispanics have *lower* life span inequality than either blacks or whites.

Finally, Edwards and Tuljapurkar (2005) use life table deaths to calculate the standard deviation in ages at death for Americans who survive until age 10. After finding persistently greater inequality among blacks, with some black-white convergence from 1970 to the mid-1980s, they conclude that "a deeper understanding … of the sources of adult life span variance is an important new frontier in social science" (Edwards and Tuljapurkar 2005, page 670). In that vein, the current study is meant to deepen our understanding of the sources of adult life span variance by decomposing the racial variance gap among those who survive until age 10.

METHODS

In order to understand the sources of the greater life span variance for blacks in the United States, we extend the decomposition method presented in Nau and Firebaugh (2012). Using period life table data broken down by cause of death, the Nau-Firebaugh method partitions the difference in the life span variances for two populations into the part due to differences in the cause-specific variances for the two populations (spread component), the part due to differences

in the proportions dying of each cause (allocation component), and the part due to differences in the average age at death for each cause (timing component).

To understand how to decompose *differences* in life span variances for two populations, we first consider how standard ANOVA would partition the life span variance for a single population into its within-cause and between-cause components. Denoting the age at death for the i^{th} victim of cause c as X_{ic}, the overall mean age at death in the population as $\bar{X}$, and the mean age at death for victims of cause c as $\bar{X}_c$, the difference between X_{ic} and $\bar{X}$ can be expressed as the sum of two differences, $x_{ic} = X_{ic} - \bar{X}_c$ and $\bar{x}_c = \bar{X}_c - \bar{X}$ (a within-cause part and a between-cause part). Based on this notation, the ANOVA equation for the total variance in age at death, by cause, is:

$$\sigma^2 = \sum_{c=1}^{C}\sum_{i=1}^{N_c}\frac{(X_{ic}-\bar{X})^2}{N} = \sum_{c=1}^{C}\sum_{i=1}^{N_c}\frac{x_{ic}^2}{N} + \sum_{c=1}^{C}\sum_{i=1}^{N_c}\frac{\bar{x}_c^2}{N}$$
$$= ANOVA\ within-cause\ component + ANOVA\ between-cause\ component \quad (1)$$

where the $c = 1, 2, \ldots, C$ causes of death are mutually exclusive and exhaustive.

Among those who are victims of a given cause, the variance in age at death is:

$$\sigma_c^2 = \sum_{i=1}^{N_c}\frac{x_{ic}^2}{N_c} \qquad (2)$$

Thus:

$$\sum_{i=1}^{N_c} x_{ic}^2 = N_c\sigma_c^2 \qquad (3)$$

Substituting (3) into the within-cause component in (1), we see that the ANOVA within-cause component is the incidence-weighted sum of the within-cause variances, σ_c^2:

$$\sum_{c=1}^{C}\sum_{i=1}^{N_c}\frac{x_{ic}^2}{N} = \sum_{c=1}^{C}\frac{N_c\sigma_c^2}{N} = \sum_{c=1}^{C} p_c\sigma_c^2 \qquad (4),$$

where $p_c = N_c/N$, the proportion of deaths due to cause c. The between-cause component in ANOVA is likewise an incidence-weighted sum of dispersion (in this case of $\bar{x}_c^2$, a measure of the dispersion of the cause-specific means):

$$\sum_{c=1}^{C}\sum_{i=1}^{N_c}\frac{\bar{x}_c^2}{N} = \sum_{c=1}^{C}\frac{N_c\bar{x}_c^2}{N} = \sum_{c=1}^{C} p_c\,\bar{x}_c^2 \qquad (5)$$

The difference in the variances for two populations is the difference in the ANOVA within- and between-variances for the two populations, so it follows from (4) and (5) that the difference in the overall variances of life spans of two populations *A* and *B*, broken down by cause, is:

$$\sigma_A^2 - \sigma_B^2 = \sum_{c=1}^{C} p_A \sigma_{cA}^2 - \sum_{c=1}^{C} p_B \sigma_{cB}^2 + \sum_{c=1}^{C} p_{cA}\, \bar{x}_{cA}^2 - \sum_{c=1}^{C} p_{cB}\, \bar{x}_{cB}^2 \qquad (6)$$

Because positive differences are more intuitive than negative differences, we designate the population with the greater variance as *A*, so $\sigma_A^2 - \sigma_B^2 > 0$.

Algebraic manipulation of (6) separates out the spread component, which captures the part of the total racial variance gap that is due to cause-specific differences in the variances of the two populations; the allocation component, which accounts for the contribution of differences in cause-specific incidence; and the timing component, which isolates the effect of differences between *A* and *B* in the variance of the mean age at death across causes. Taking population *B* as the reference, and summing over all *C* causes, the components are (Nau and Firebaugh 2012):

$$spread\ component\ = \sum_{c=1}^{C} (\sigma_{cA}^2 - \sigma_{cB}^2) p_{cB}$$

$$allocation\ component = \sum_{c=1}^{C} (p_{cA} - p_{cB})(\sigma_{cB}^2 + \bar{y}_{cB}^2)$$

$$timing\ component = \sum_{c=1}^{C} (\bar{y}_{cA}^2 - \bar{y}_{cB}^2) p_{cB}$$

$$joint\ component\ = \sum_{c=1}^{C} (p_{cA} - p_{cB})[(\sigma_{cA}^2 - \sigma_{cB}^2) + (\bar{y}_{cA}^2 - \bar{y}_{cB}^2)] \qquad (7)$$

The joint component captures the amount of the differential in the population variances that is due to simultaneous differences in incidence and in cause-specific variances for the two populations.[2] The components add up exactly to the overall difference in variance, $\sigma_A^2 - \sigma_B^2$. By

[2] The joint component could be eliminated by weighting the S-A-T differences in (7) by averages for the two populations – in effect replacing population *B* values with average values as the

dividing each of the components by $\sigma_A^2 - \sigma_B^2$, then, we can express them as proportions of the total difference in variance.

Our objective is to partition $\sigma_{Blacks}^2 - \sigma_{Whites}^2$, the difference in the variances of age at deaths for blacks and whites in 2010, into its spread, allocation, and timing components. Because $\sigma_{Blacks}^2 - \sigma_{Whites}^2$ is a positive number, a positive value in our results indicates that the component in question – say the spread effect for heart disease – contributes to the greater variance for blacks, whereas a negative value indicates that the component operates in the opposite direction, to compress the difference in the variance for blacks and whites. Because we use whites as the reference population, the magnitude of a component indicates how big the gap between blacks and whites would have been if blacks had the same values as whites on all other components, and had differed only with respect to the particular factor under investigation (e.g., the spread effect for heart disease).

SAT equations by sex

To anticipate a major result of this study, we find that the total spread component constitutes about 87 percent of the difference in variance for the black and the white populations, while the total allocation component constitutes about 12 percent of the difference. The timing and joint components are relatively small, indicating that most of the gap in life span inequality is caused by either spread effects or allocation effects but rarely by a combination of the two. Hence we focus on the spread and allocation components, probing them further to determine whether they arise largely from differences between black men and white men or from differences between black women and white women.

The spread and allocation components can be broken down by subpopulations – in our case, by sex – in a straightforward manner. In the case of the allocation component, $p_{cBlacks} = p_{cBlackWomen} + p_{cBlackMen}$, and similarly for whites, so the allocation component for cause *c* is (from Eq. 7):

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reference. Our research question here, however, is "Why is there *greater variance* in age at death among blacks than among whites?" so the interpretation of results will be more straightforward if we use whites as the reference.

$$allocation\ component\ for\ cause\ c = (p_{cBlacks} - p_{cWhites})(\sigma^2_{cWhites} + \bar{y}^2_{cWhites})$$
$$= (p_{cBlackWomen} - p_{cWhiteWomen})(\sigma^2_{cWhites} + \bar{y}^2_{cWhites}) + (p_{cBlackMen} - p_{cWhiteMen})(\sigma^2_{cWhites} + \bar{y}^2_{cWhites}) \qquad (8)$$

We turn now to the spread component. With whites as the reference population, the formula for the c^{th} cause of death is $(\sigma^2_{cBlacks} - \sigma^2_{cWhites})p_{cWhites}$. Because the sum of squares for blacks is the sum for women (*SSBlackWomen*) plus the sum for men (*SSBlackMen*), the numerator of $\sigma^2_{cBlacks}$ can be partitioned by sex:

$$\sigma^2_{cBlacks} = \frac{SSBlackWomen}{N_{cBlacks}} + \frac{SSBlackMen}{N_{cBlacks}}, \qquad (9)$$

where *SSBlackWomen* = $\sum_{i=1}^{N_{cBlacks}}(X_{iBlackFemale} - \bar{X}_{Blacks})^2$ and similarly for black men. $N_{cBlacks}$ is the number of black victims of cause *c*, and $\bar{X}_{Blacks}$ is the mean age of those victims.

The variance for whites is partitioned in the same way, so the *difference* in the within-cause variance for white and black victims is:

$$\sigma^2_{cBlacks} - \sigma^2_{cWhites} = \frac{SSBlackWomen}{N_{cBlacks}} + \frac{SSBlackMen}{N_{cBlacks}} - (\frac{SSWhiteWomen}{N_{cWhites}} + \frac{SSWhiteMen}{N_{cWhites}})$$
$$= \left(\frac{SSBlackWomen}{N_{cBlacks}} - \frac{SSWhiteWomen}{N_{cWhites}}\right) + \left(\frac{SSBlackMen}{N_{cBlacks}} - \frac{SSWhiteMen}{N_{cWhites}}\right) \qquad (10)$$

By weighting the differences in eq 10 by $p_{cWhites}$, the proportion of all white deaths due to the c^{th} cause of death, we determine the part of the spread component for cause *c* attributable to differences between black women and white women versus the part attributable to differences between black men and white men. The two parts add up exactly to the spread component for a particular cause, so by summing these components over the 17 causes we obtain the part of the overall spread component that is attributable to differences between white and black women and the part that is attributable to differences between white and black men.

DATA

We use information on the number of deaths by cause, age, sex and race from the 2010 Multiple Cause of Death data archive from the National Center for Health Statistics (NCHS). The NCHS mortality data contains information from death records of the entire U.S. resident population of a given calendar year. As suggested by Edwards and Tuljapurkar (2005), we consider only deaths

that occur after age 10, because childhood mortality is etiologically different from adult mortality.[3]

Causes of death are coded according to the 10th revision of the International Classification of Diseases (ICD-10). This classification contains over 10,000 individual codes. In order to reduce the number of causes under investigation, we regrouped causes into broad, epidemiologically sensible, categories. The template for our taxonomy was the coding scheme used to identify the 15 leading causes of death in the National Vital Statistics Report for 2006 (Heron et al., 2009). In addition, our categorization of causes reflects findings from prior analyses regarding specific causes of death that are particularly germane to racial differences in mortality (Harper et al. 2007; Warner et al. 2011).

Our final classification consists of 17 cause categories, beginning with the 10 leading causes of death in the United States in 2006. To this list we added one category by substituting traffic accidents and accidental poisoning for the more general category "accidents." We also made HIV/AIDS, homicide, and suicide separate categories because they are highly associated with racial mortality differentials in the United States (Harper et al. 2007). To capture the effect of all infectious diseases, we added a category for infectious diseases other than those already listed in our classification scheme (i.e., influenza, septicemia, and HIV/AIDS). Similarly, to separate out the effect of all external causes, we added a category for external causes other than those already on our list (homicide, suicide, traffic accidents, and accidental poisoning). Finally, to be exhaustive, we added a residual category of causes that we call "minor causes, not elsewhere classified (n.e.c.)." This residual category aggregates all ill-defined causes of death as well as those causes that have too few deaths to be considered independently, or that are not of primary interest in this analysis.

The NCHS files also contain population counts from the U.S. Census Bureau's April 1st modified race census counts for 2010 (NCHS 2010). These counts serve as population denominators for calculating age-race and sex-specific mortality rates by cause category. We

[3] Conceivably, small differences in mortality in the very young could strongly affect the variance of the age-at-death distribution. In supplementary analysis not shown, we replicated Harper et al.'s (2012) finding that nearly all deaths before age 10 fall under two new cause categories, "prenatal deaths" and "congenital anomalies," so including pre-10 deaths has virtually no effect on the relative proportions in our cause categories. Consequently, our essential results are the same whether or not we include those who died before their tenth birthday.

compute multi-decrement life tables for non-Hispanic blacks and non-Hispanic whites to eliminate effects of differences in population size and age-composition (Das Gupta 1993). Ages are aggregated into five-year intervals, with the open-ended age category being 85+.[4] The life-table numbers of deaths by age are then used to calculate the statistics that are needed for the SAT decomposition (cause-specific means, variances, and proportions of deaths).

RESULTS

As shown in Figure 1, the variance in life spans is greater for blacks than for whites in the United States. Our findings closely resemble those of Edwards and Tuljapurkar (2005) and Tuljapurkar and Edwards (2011). Among those who survive past age 10, the variance of life table deaths is 244.1 for blacks and 199.2 for whites, so the difference in variance is 44.9. That is the difference we decompose along both a cause-of-death axis and a spread-allocation-timing axis.[5]

Figure 1 about here

The first step is to determine, for all 17 causes, the differences in the proportion of whites and blacks who die of each cause as well as the differences in the average age at death of the white and black victims (Table 1). With one exception (accidental poisoning), the difference in average ages is positive, reflecting the fact that, for 16 of our 17 cause categories, white victims are older than black victims on average.

Table 1 about here

We group the 17 causes into four major categories: chronic diseases (which account for almost 70% of all deaths), communicable diseases (5% of all deaths), external causes (5% of all deaths), and aggregated minor causes (20% of all deaths). Of the four major categories, "communicable diseases" is the most racially imbalanced, with blacks being 33% more likely (than whites) to die of a communicable disease. This difference is due entirely to racial

[4] For the open-ended age category being 85+, we have used age 90 as the mean age at death because age 90 is the approximately the mean age for those who survived until age 85 for both racial groups and sex.

[5] As suggested by Figure 1, the racial variance gap is due almost entirely to racial differences in death rates among the young and middle-aged; very little is due to the black/white crossover in death rates at older ages. Because the crossover is due partly to age misreporting (Fenelon 2013), we performed simulations based on downward adjustments of the age at death among older blacks. These adjustments reduced the difference in black-white variance very little.

differences in septicemia and HIV/AIDS, diseases that disproportionately affect blacks. Proportions for the other two categories of communicable diseases – influenza and pneumonia, and other infectious diseases – are comparable for blacks and whites.

In contrast, whites are more likely than blacks to die of external causes. Whites are three times more likely to die of suicide, almost twice as likely to die of accidental poisoning, and 50% more likely to die of unclassified external causes (i.e., external causes other than suicide, accidental poisoning, traffic accidents, and homicide). These differences offset the higher homicide rate among blacks (blacks are nearly six times more likely to be homicide victims). Although homicide is relatively uncommon, it could be a significant source of the racial variance gap since the differences across race are notable for both their incidence (proportions) and for the ages of the victims (homicide disproportionately affects the young).

About 90% of all deaths fall under the two general categories of "chronic diseases" and "minor causes, not elsewhere classified." With respect to chronic diseases, the two leading causes of death, heart disease and cancer, account for almost 50% of the total number of deaths for both racial groups (48% for whites, 49% for blacks). The proportion of blacks who die of heart disease closely matches the proportion for whites who die of heart disease, and similarly for cancer. At least with respect to allocation, then, we do not expect heart disease and cancer to be major contributors to the racial variance gap. With regard to the other chronic diseases, whites are more likely to die of chronic lower respiratory diseases or of Alzheimer's, while blacks are more likely to die of diabetes, nephritis, or cerebrovascular disease. These differences largely offset each other, however, so in general whites and blacks are almost equally likely to die of some type of chronic disease (69.0% for whites, 69.4% for blacks).

Turning now to the age gaps between blacks and whites, note that, for almost all causes of death, blacks tend to die at a younger age than whites do. The only exception is accidental poisoning, where black victims are almost 7 years older, on average. For Alzheimer's and HIV/AIDS the racial age gap is negligible, with the advantage of whites being 4 months or less. The remaining 14 causes of death all contribute to the racial gap in life expectancy, but their contribution varies considerably. For example, the advantage for whites ranges from about 2 years for cancers and diabetes, to almost 11 years for homicide. Besides homicides, the age advantage for whites is particularly notable for suicide (7.7 years), "other external causes" (7.1

years), "other infectious diseases" (5.4 years), cerebrovascular diseases (4.7 years), nephritis (4.1 years), and heart disease (4.0 years).

Decomposition of the racial variance gap

Table 2 reports the results of the 17 causes of death partitioned into their respective spread, allocation, timing, and joint components. The sum of these components for each cause, displayed in the final column, is the portion of the black-white variance gap attributable to that cause of death (expressed as a percentage). The decomposition shows that four-fifths of the racial variance gap would have persisted if the mortality regime of blacks and whites differed only with respect to heart disease (about 30%) and homicide (about 50%). Deaths due to accidental poisoning, on the other hand, reduce the racial variance gap. In other words, if the mortality regime for blacks and whites differed only with respect to accidental poisoning, the variance in age at death would be greater for whites, not blacks. The largest contributors to the black-white variance gap are, from higher to lower: homicide, heart disease, minor causes not elsewhere classified, HIV/AIDS, cerebrovascular diseases, and diabetes. The largest compressors of the black-white variance gap are accidental poisoning and suicide.

Table 2 About Here

The SAT decomposition enables us to unpack the dynamics underlying the overall contributions of each cause. For example, the spread component for homicide, the largest single contributor to the racial variance gap, is negligible, indicating that black-white differences in the variability of the ages of homicide victims did not contribute notably to the racial gap in variance. Homicide nonetheless contributes greatly to the variance gap because blacks are six times more likely to be homicide victims, and homicide victims tend to be young, particularly in the case of black victims (Table 1). These differences translate into an allocation component of 38% and a joint component of 10.2% (Table 2). The allocation component for homicide indicates that the black-white difference in the incidence of homicide alone constitutes 38% of the racial variance gap, that is, 38% of the observed gap would persist if the black and white mortality regimes were otherwise equivalent. The joint component captures the simultaneous effect of black-white differences in both incidence and the prematurity of the deaths of homicide victims. Although homicide tends to result in premature deaths for both whites and blacks, the effect on variance in age at death is greater for blacks because black homicide victims are particularly young (about 11 years younger for blacks than for whites, on average).

On the basis of homicides, then, one might think that the variance gap is due largely to racial differences in the causes of death, i.e., the allocation component. Yet in fact the total allocation component accounts for only about one-eighth (12.4%) of the greater variance in age at death among blacks (last row of Table 2). The total allocation component is smaller than the allocation component for homicides alone because the allocation components for some of the other causes are negative. Whites are three times more likely than blacks to commit suicide, for example, and twice as likely to die of accidental poisoning (mainly narcotics-related: Warner et al. 2011). Because suicide and poisoning victims tend to be relatively young, the greater incidence of suicide and poisoning deaths among whites stretches the age-at-death distribution for whites relative to the age-at-death distribution for blacks, thus reducing the overall gap in variance of the two distributions. Together, the negative allocation components for suicide and accidental poisoning (-36.8%) largely offset the positive allocation component for homicide (+38%).

All of the cause-specific *spread* components, by contrast, are positive or close to zero. For most causes of death there is more variability in the age at death of black victims than of white victims, and the exceptions are negligible. Because the cause-specific spread components are all positive or negligible, the spread component constitutes the vast majority – about 87% – of the gap. In other words, among those who die of the same cause, there typically is greater variance in the age at which blacks succumb than in the age at which whites succumb, and cumulatively that greater within-cause variance for blacks is the main driver of the black-white variance gap.

Decomposition of the racial variance gap by sex

As the final step of our analysis we use equations 8-10 (above) to determine what parts of the racial variance gap arise from differences between white women and black women and what parts arise from differences between white men and black men. We focus on spread and allocation because the spread and allocation components together account for over 99% of the entire black-white variance gap in 2010 (Table 2, last row).

Table 3 reports the results for the gender-race decomposition. Overall, women contribute twice as much to the racial variance gap as men do (last row of Table 3). Indeed, women's spread component alone accounts for about 62.5% of the total racial variance gap. This indicates that, regardless of the cause of death, age at death tends to be more variable for black women

than for white women. This is true for all but three causes of death (homicide, traffic accident, and accidental poisoning), for which the variance of age at death is marginally larger for white women. The female differences in variance are most significant for heart disease and minor causes not elsewhere classified, each accounting for about 21% of the total gap in racial variance.

Table 3 About Here

We find the same cause-specific dynamic for men. That is, for all causes of death, age at death is more variable for black men than for white men or – if the variance is greater for white men – the differences are relatively small. Compared to disparities for women, however, the racial differences in variance are much smaller for heart disease and minor causes for men. In the case of heart disease, for example, the female spread component accounts for 21.4% of the racial variance gap whereas the male spread component accounts for only 7.4% of the gap.

In short, most of the racial variance gap is attributable to black-white differences in the variance in age at death within causes of death. While this is true for both men and women, the racial differences are significantly greater for women than for men with regard to heart disease and minor causes not elsewhere classified. The net result is that differences in cause-specific variances for women are responsible for 62.5% of the overall difference in the variance in longevity for blacks versus whites in the United States.

The total allocation component is relatively small for both women and men. In the case of women, the total allocation component accounts for less than 4% of the overall racial variance gap. This result, however, masks several race-sex-specific differences in cause-specific mortality. For instance, while black women are more likely than white women to die of diabetes, HIV/AIDS, or homicide, white women are more likely than black women to die of chronic lower respiratory diseases, suicide, or accidental poisoning. These contrasting patterns offset each other, producing the small overall contribution of allocation for women.

The allocation component among men accounts for less than 9% of the total racial difference in the variance of age at death, with most causes of death contributing only marginally. However, there are four causes for which the allocation component among males is substantial. The first such cause is homicide: Male homicide is the greatest single contributor to the overall black-white difference in variability of age at death. Although deaths due to homicide represent less than 0.3% of all white deaths and less than 1.3% of all black deaths, the allocation

component for males constitutes over one third of the overall black-white variance gap. This result is even more striking when we consider that the male allocation effect for homicides contributes more to the racial variance gap than does heart disease, even though heart disease accounts for over 1 of every 4 deaths.

The second cause with a substantial allocation effect for males is HIV/AIDS, which adds another 10% to the racial variance gap. Allocation components for two other causes of death for men – suicide (-17.1%) and accidental poisoning (-9.2%) – work in the opposite direction, to reduce the racial variance gap. Because suicide and accidental poisoning victims tend to be relatively young, thus stretching out the variance in age at death, the greater incidence of suicide and accidental poisoning among white men compresses the racial variance gap. As a result, the overall allocation component for men is not as large as one might expect in light of the magnitude of racial differences in homicide and HIV/AIDS death rates.

Figure 2 plots the life table number of deaths per 100,000 by age for the cause-specific components whose contribution amounts to 10% or more of the racial variance gap. We plot the results separately for men and women. Because of differences in incidence, the scale for the y-axis (number of deaths per 100,000) differs from one cause of death to another. The scale is the same for women and men for each cause of death, however, so gender differences are readily apparent.

Figure 2 about here

Figure 2 shows that racial differences in mortality are particularly pronounced for men, with mortality rates above 500 per 100,000 for black men in their mid-twenties. The allocation component for HIV/AIDS is also readily apparent in the graphs. In this case, however, the racial difference is pronounced for both men and women. As in the case of homicides, HIV/AIDS contributes to the racial variance gap due to allocation. Among HIV/AIDS victims, the variance in age at death is the same for black men versus white men, and for black women versus white women.

As noted earlier, accidental poisoning and suicide work to narrow the racial difference in variability of age at death. This happens for two reasons: first, these causes of death are more common for whites than for blacks; and second, the causes disproportionately affect the young. As seen in Figure 2, whites are more likely than blacks to commit suicide or die of accidental poisoning. For both causes of death the pattern is similar across gender, but racial differences

are more pronounced among men. Moreover, like homicide and HIV/AIDS, these causes of death are most common among the young and middle-aged, thus enlarging the variance of the age-at-death distribution. Because suicide and death due to accidental poisoning are more common among whites, the allocation components in this case *reduce* the black-white variance gap by enlarging the variance for whites more than for blacks.

Finally, heart diseases and minor causes not elsewhere classified are characterized by a particularly strong spread effect, especially for women. For both cause categories, then, the distribution of age at death is more spread out for blacks than for whites. Although blacks and whites are about equally likely to die of heart disease or of causes not elsewhere classified (Table 1), blacks are more likely than whites to die of these causes at a younger age – when they are in their 40s, 50s, or 60s, farther from the overall average age at death (Figure 2). Deaths for whites, on the other hand, are more concentrated at older ages – over 80 years for heart diseases and 75 years for aggregated minor causes – closer to the overall average. Combined with the fact that heart disease and causes not elsewhere classified account for such a high proportion of deaths, the greater vulnerability of blacks at younger ages generates the substantial spread component for both cause categories, especially among women, for whom this trend is more pronounced.

DISCUSSION AND CONCLUSION (to be finished later)

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APPENDIX I – CAUSE-GROUPING

Table A.1. Cause-grouping and corresponding ICD-10 codes

ICD 10 Cause grouping		
17 category Cause-group	**ICD10 4 digit code range**	**Contents of Cause-group (described using ICD10 cause description)**
(1) **Chronic diseases**		
Heart diseases	I00-I09.9 I11-I11.9 I13-I13.9 I20-I25.9 I26-I28.9 I30-I51.9	Acute rheumatic fever, chronic rheumatic heart disease Hypertensive heart disease Hypertensive heart and renal disease Ischemic heart disease Pulmonary heart disease and diseases of pulmonary circulation Other forms of heart disease
Cancers	C00-D48.9	Chapter II: Neoplasms
Cerebrovascular diseases	I60-I69.8	Cerebrovascular diseases
Chronic lower respiratory diseases	J40-J47	Chronic lower respiratory diseases
Alzheimer's	G30-G30.9	Alzheimer's disease
Diabetes	E10-E14.9	Diabetes mellitus
Nephritis, Nephrosis, Nephrotic Symptom	N00-N07.9 N17-N19 N25-N27.9	Nephritis, Nephrosis, Nephrotic Symptom†
(2) Communicable diseases		
Influenza and pneumonia	J10-J18.9	Influenza and pneumonia
Septicemia	A40-A41.9	Streptococcal and other septicemia
HIV/AIDS		
Other Infectious Diseases	A00-B99	Chapter I: Certain infectious and parasitic diseases, with the exception of septicemia and STD/NTD
(3) External causes		
Homicide	X85-Y09.9	Assault

Suicide		
Traffic Accidents	V02-V04.9 V09-V09.9 V12-V14.9 V19-V19.9 V20-V79.9 V80-V89.9	Pedestrian injured in accident with motor vehicle Pedestrian injured in other and unspecified transport accidents Pedal cyclist injured in accident with motor vehicle, Pedal cyclist injured in unspecified transport/traffic accident Motor-cyclist, occupant of three-wheeled vehicle, car occupant, occupant of pick-up truck, occupant of heavy transport vehicle, or bus occupant injured in transport accident Other land transport accidents
Accidental poisoning		
Other External Causes	Remaining codes V01-V01.9, V90-Y89.9 excluding of Assault	Remainder of chapter XX: External causes of morbidity and mortality, with exception of Assault
(4) Aggregated minor causes		
NEC – Not elsewhere classified		

Table 1. Distribution of deaths, and average age at death, by cause: Whites versus blacks in the U.S., 2010.

		Proportions			Mean age		
ICD-10 cause of death		White	Black	Diff	White	Black	Diff
Chronic diseases							
1	Heart diseases	0.257	0.263	-0.006	81.4	77.4	4.0
2	Cancers	0.226	0.227	-0.002	75.4	73.6	1.8
3	Cerebrovascular diseases	0.056	0.063	-0.007	82.9	78.2	4.7
4	Chronic lower respiratory diseases	0.064	0.034	0.030	79.6	77.3	2.3
5	Alzheimer's	0.044	0.029	0.014	86.8	86.5	0.2
6	Diabetes	0.024	0.044	-0.020	77.2	75.2	1.9
7	Nephritis, nephrosis, nephrotic symptoms	0.020	0.035	-0.015	81.9	77.8	4.1
	Total proportion, chronic diseases	**0.690**	**0.694**	**-0.005**			
Communicable							
8	Influenza and pneumonia	0.022	0.020	0.002	83.1	80.2	2.9
9	Septicemia	0.013	0.022	-0.009	79.0	76.8	2.2
10	HIV/AIDS	0.001	0.009	-0.008	51.2	50.9	0.3
11	Other infectious diseases	0.009	0.008	0.001	76.8	71.4	5.4
	Total proportion, communicable	**0.045**	**0.060**	**-0.015**			
External causes							
12	Homicide	0.002	0.013	-0.011	44.2	33.3	10.9
13	Suicide	0.012	0.004	0.008	50.0	42.4	7.7
14	Traffic accident	0.010	0.009	0.001	49.2	46.6	2.5
15	Accidental poisoning	0.011	0.006	0.004	42.5	49.3	-6.8
16	Other external causes	0.022	0.014	0.008	76.4	69.3	7.1
	Total proportion, external causes	**0.057**	**0.046**	**0.011**			
Aggregated minor causes							
17	NEC - Not elsewhere classified	0.209	0.200	0.009	80.7	77.7	3.0
Overall[b]		**1.000**	**1.000**	**0.000**	**78.7**	**75.3**	**3.3**

[a] Source: Based on life table deaths for those who survive to age 10 (CDC, ***)
[b] Total for proportions and grand mean for age at death

Table 2. SAT components of the black-white variance gap in age at death, by cause, in 2010 (in percentages)[a]

ICD-10 cause of death		Spread (S)	Allocation (A)	Timing (T)	Joint (J)	S+A+T+J
Chronic diseases						
1	Heart diseases	28.7	2.0	-1.7	0.6	29.6
2	Cancers	7.5	0.6	-3.7	0.0	4.4
3	Cerebrovascular diseases	8.1	1.9	-1.2	0.8	9.6
4	Chronic lower respiratory diseases	8.1	-6.9	0.5	-4.0	-2.4
5	Alzheimer's	0.2	-3.1	5.8	-2.0	0.9
6	Diabetes	0.9	7.6	-0.1	0.6	9.0
7	Nephritis, nephrosis, nephrotic symptoms	2.3	3.9	-0.2	1.5	7.5
Communicable						
8	Influenza and pneumonia	2.1	-0.5	0.2	-0.2	1.6
9	Septicemia	0.7	3.2	0.1	0.5	4.5
10	HIV/AIDS	0.1	16.2	-0.3	-2.2	13.7
11	Other infectious diseases	0.7	-0.2	0.2	-0.1	0.6
External causes						
12	Homicide	-0.6	38.0	2.2	10.2	49.8
13	Suicide	-1.0	-21.8	7.2	-4.2	-19.8
14	Traffic accident	-2.1	-3.9	-1.0	0.4	-6.6
15	Accidental poisoning	-0.4	-15.0	-14.9	6.4	-23.9
16	Other external causes	5.0	-5.9	1.5	-2.3	-1.6
Aggregated minor causes						
17	NEC - Not elsewhere classified	27.2	-3.7	0.7	-1.2	23.0
TOTAL		**87.2**	**12.4**	**-4.7**	**5.0**	**100.0**

[a] The standard deviation in age at death is 15.62 among blacks and 14.11 among whites, so the difference in variance is 15.62^2 minus 14.11^2, or 44.9.

Table 3. Difference in the variance of life table ages at deaths for blacks versus whites in the U.S., 2010: Spread and allocation components, broken down by sex.

		Spread Component (%)			Allocation Component (%)		
	ICD-10 cause of death	Total	Female	Male	Total	Female	Male
Chronic diseases							
1	Heart diseases	28.7	21.4	7.4	2.0	4.0	-2.0
2	Cancers	7.5	6.8	0.7	0.6	1.4	-0.8
3	Cerebrovascular diseases	8.1	5.0	3.1	1.9	1.1	0.8
4	Chronic lower respiratory diseases	8.1	3.1	5.0	-6.9	-4.1	-2.8
5	Alzheimer's	0.2	0.2	0.0	-3.1	-2.0	-1.1
6	Diabetes	0.9	1.2	-0.3	7.6	5.2	2.4
7	Nephritis, nephrosis, nephrotic symptoms	2.3	1.6	0.7	3.9	2.6	1.3
Communicable							
8	Influenza and pneumonia	2.1	1.0	1.1	-0.5	-0.2	-0.3
9	Septicemia	0.7	0.5	0.2	3.2	2.0	1.2
10	HIV/AIDS	0.1	0.1	0.0	16.2	5.8	10.3
11	Other infectious diseases	0.7	0.5	0.2	-0.2	-0.3	0.0
External causes							
12	Homicide	-0.6	-0.4	-0.2	38.0	4.0	34.0
13	Suicide	-1.0	0.0	-1.0	-21.8	-4.7	-17.1
14	Traffic accident	-2.1	-0.7	-1.3	-3.9	-1.6	-2.2
15	Accidental poisoning	-0.4	-0.4	-0.1	-15.0	-5.8	-9.2
16	Other external causes	5.0	2.1	2.9	-5.9	-3.5	-2.4
Aggregated minor causes							
17	NEC - Not elsewhere classified	27.2	20.7	6.5	-3.7	-0.2	-3.5
TOTAL		**87.2**	**62.5**	**24.7**	**12.4**	**3.7**	**8.7**