RACIAL/ETHNIC INEQUALITY IN ADULT SURVIVAL: DECOMPOSITION OF AGE AT DEATH VARIATION AMONG U.S. ADULTS

Joseph T. Lariscy[1], Claudia Nau[2], Glenn Firebaugh[3], and Robert A. Hummer[1]

[1] Population Research Center and Department of Sociology, University of Texas at Austin

[2] Johns Hopkins Global Center for Childhood Obesity, Bloomberg School of Public Health, Johns Hopkins University

[3] Population Research Institute and Department of Sociology, Pennsylvania State University

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Correspondence: Joseph T. Lariscy, jlariscy@prc.utexas.edu.

Abstract

Racial and ethnic inequalities in life expectancy (the mean age at death) are well-established. However, considering only differences in longevity misses an important component of mortality inequality: variability in age at death. We use 2009 U.S. National Vital Statistics System data to examine the magnitude of racial/ethnic differences in lifespan variability. We decompose variance in the age at death distribution of Hispanics and non-Hispanic blacks relative to non-Hispanic whites using the variance decomposition technique developed by Nau and Firebaugh (2012). This approach decomposes racial/ethnic differences in lifespan variability into a) the contribution of causes of death and b) the contributions of racial/ethnic differences in cause-specific variability of age at death (spread effect), number of deaths attributable to each cause (allocation effect), and cause-specific mean age (timing effect). Hispanics exhibit less lifespan variability relative to non-Hispanic whites in addition to higher life expectancy. Non-Hispanic blacks, on the other hand, have greater variability and lower life expectancy than the other two racial/ethnic groups. The lower variability among Hispanics relative to whites is largely attributable to allocation effects in cancer, suicide, and other external cause mortality whereas the greater variability among blacks relative to whites is mainly due to spread effects in mortality from heart disease and the residual cause grouping.

Introduction

Racial and ethnic inequalities in life expectancy are well established. Most recently, Arias (2010) determined that Hispanics have the highest life expectancy at birth in the U.S., non-Hispanic blacks have the lowest, and non-Hispanic whites exhibit life expectancy between the two minority groups. However, considering only group differences in life expectancy (i.e., the mean age at death) misses an important component of survival inequality: racial/ethnic differences in the variability of age at death. For instance, if Hispanics' higher life expectancy is matched by a narrower variation in ages at death relative to other groups, they are particularly advantaged, since Hispanics would not only live longer on average but would also do so more homogeneously across individuals. A greater clustering of age at death (known as mortality compression) among a particular racial/ethnic group relative to others would also indicate that groups proceed disproportionately through the epidemiologic transition, where a large proportion of one group experiences "senescent mortality" that strikes at the oldest ages while another group continues to die from "background mortality," causes which strike at earlier adult ages (Bongaarts 2006; Horiuchi and Wilmoth 1998).

This study addresses the following research questions: Do U.S. racial/ethnic groups differ in their variability in age at death? Are these differences due to spread, allocation, or timing effects? How do causes of death underlie such differences? We use 2009 U.S. National Vital Statistics System data to examine the magnitude of racial/ethnic differences in the variability of individual longevity and its cause-specific underpinnings. We decompose the difference in the variance of ages at deaths conditional on survival to age 10 of Hispanics and non-Hispanic blacks relative to non-Hispanic whites using the variance decomposition procedure developed by Nau and Firebaugh (2012). This methodology decomposes racial/ethnic differences in lifespan

variability into a) the contribution of each cause of death and b) the contributions of racial/ethnic differences in (i) cause-specific variability of ages at death (spread effect), (ii) number of deaths attributable to each cause (allocation effect), and (iii) cause-specific mean ages (timing effect).

Sources of Racial/Ethnic Variability in Age at Death

Hispanics in the U.S. on average are socioeconomically disadvantaged relative to non-Hispanic whites. Since a clear inverse gradient exists between socioeconomic (SES) and mortality risk, with greater socioeconomic resources associated with lower risk of death (Baker et al. 2011; Elo 2009; Hummer and Lariscy 2011; Kitagawa and Hauser 1973), one would expect Hispanics' lower socioeconomic profiles to negatively affect their survival. However, Hispanics (particular older adults and immigrants) exhibit lower adult mortality risk relative to non-Hispanic whites. This anomalous finding is known as the "epidemiologic paradox" (Markides and Coreil 1986).

While Hispanics generally have equal or better survival relative to whites and blacks have lower survival relative to whites, these patterns vary by age and by cause of death. Hispanics do not exhibit a clear mortality advantage at all adult ages—young adult Hispanics exhibit higher mortality risk relative to non-Hispanic whites. Researchers have found that the higher Hispanic mortality risk is limited to males ages 15-24 years (Hayes-Bautista et al. 2002; Murphy, Xu and Kochanek 2012) and females 10-19 years (Murphy et al. 2012) and that Hispanic mortality risk is not significantly different from that of non-Hispanic whites throughout early to middle adulthood (Hummer et al. 1999). Beyond roughly age 45, the mortality risk among Hispanics becomes lower relative to that of non-Hispanic whites as chronic causes of death become more prevalent. Regarding cause of death, Hispanics have a greater mortality risk of diabetes, cirrhosis, and infectious diseases relative to whites (Singh and Hoyert 2000) due to

elevated levels of obesity and heavy alcohol consumption, particularly among U.S.-born Hispanics (Hummer et al. 1999). At middle and older adult ages, where chronic causes of death replace infectious and external causes (unintentional injuries and homicide), a Hispanic mortality advantage clearly emerges.

The racial gap in U.S. mortality is wide and enduring as a result of lower socioeconomic status, residential segregation, and institutional- and individual-level discrimination experienced by African Americans (Hummer and Chinn 2011; Rogers, Hummer and Nam 2000). In 2008, life expectancy at birth among African Americans was 4.5 years less than that among whites (74.0 years versus 78.5) (Miniño et al. 2011). Additionally, the black-white mortality differential is roughly two-to-one in early adulthood but mortality rates converge and a crossover appears to occur in later adulthood (Eberstein, Nam and Heyman 2008; Elo and Preston 1997; Stewart 2008). Blacks exhibit a greater risk of mortality from most causes of death, particularly from heart disease and cancer (causes that account for more than half of all deaths in the United States) and homicide (Rogers et al. 2002). Whites are at a greater risk of death than blacks from motor vehicle accidents, chronic obstructive pulmonary disease, and suicide (Levine et al. 2001; Singh and Siahpush 2001). And although non-Hispanic blacks exhibit lower prevalence of some diseases and behaviors relative to whites, they often experience greater morbidity, earlier onset of disease, greater severity of symptoms, and reduced survival (Hayward et al. 2000; Williams and Sternthal 2010).

Other researchers have previously demonstrated greater variation in longevity among Americans relative to residents of other developed countries and suggest this may partially be a result of the greater racial and ethnic heterogeneity in the U.S. (Edwards and Tuljapurkar 2005; Nau and Firebaugh 2012). While much research has examined racial/ethnic inequalities in cause-

specific mortality risk, studies of racial/ethnic inequalities in variation in cause-specific mortality are lacking. Tuljapurkar and Edwards (2011) compared black-white dispersion in length of life (measuring dispersion as the standard deviation in life table ages at death from all causes combined conditional on survival to age 10, S_{10}) and found that dispersion is greater among blacks (S_{10}=16.7) than whites (S_{10}=14.9). This roughly 2-year gap has persisted since the 1960's (Edwards and Tuljapurkar 2005). Lynch, Brown, and Harmsen (2003) used data from the Berkeley Mortality Database (the predecessor to the Human Mortality Database), with adjustments for data quality issues among older African Americans, to show that from 1972 to 1990, the greater dispersion in age at death among blacks has become more compressed while dispersion has remained stagnant among whites. Nau and Firebaugh (2012:1224) examined the degree to which racial/ethnic heterogeneity accounts for the greater lifespan variability among Americans compared to Swedes and identified "nontrivial differences between non-Hispanic whites and nonwhites in the spread, allocation, and timing effects for some specific causes." Only one analysis to date considers lifespan variation among Hispanics, non-Hispanic blacks, and non-Hispanic whites (Go et al. 1995). Go and colleagues used California vital statistics and Census data for years 1970, 1980, and 1990 to suggest that whites in California had the highest mean age at death (life expectancy) as well as the smallest standard deviation in age at death, African Americans had a lower life expectancy paired with greater variation, and Hispanics exhibited the lowest life expectancy and the largest variation in age at death. Newer analyses are clearly warranted since recent, nationally-representative data show lower mortality risk among Hispanics relative to whites and are available to document up-to-date racial/ethnic differences in lifespan variation.

Measuring Subgroup Differences in Age-at-Death Variability

Racial/ethnic differences in the mean age at death (life expectancy) reveal *between-group* variation but cannot reveal *within-group* variation. In this study, we measure racial/ethnic differences in within-group variation using variance in life table age at death conditional on survival to age 10 (S_{10}^2). Measures of lifespan variation other than variance are available (interquartile range, Theil's index, Gini coefficient, etc.) but these measures are highly correlated (van Raalte and Caswell Forthcoming; Wilmoth and Horiuchi 1999) although in some cases conclusions may depend on the variability indicator used (Shkolnikov, Andreev and Begun 2003). Relative to the other indicators, variance is particularly well-suited for decomposition into spread, allocation, and timing effects. Other studies of within-population variability in age at death rely on modal age at death as the central tendency indicator and the standard deviation above the modal age as the variability indicator. While this approach is less sensitive to the age at left-truncation, it primarily measures variability in mortality due to biological aging (i.e., senescence) without capturing group differences in early-life mortality, which have high potential for increasing age-at-death dispersion.

Nau and Firebaugh (2012) developed an innovative variance decomposition approach to determine why length of life in the United States is more unequal than in Sweden, the nation characterized by best case mortality patterns of high life expectancy paired with low variability in age at death. They found that much of the variance difference between the two nations is due to greater within-cause variance (spread effect) in the U.S. relative to Sweden. Greater incidence of external causes of death among U.S. adults also explained a sizable component of the greater variance in age at death in the U.S. In this paper, we apply Nau and Firebaugh's decomposition

technique to examine differences in lifespan variation among racial and ethnic subpopulations within the U.S.

Methods

Data

We use 2009 U.S. National Vital Statistics System data. Death data come from the 2009 U.S. Multiple Cause of Death file available from the National Center for Health Statistics. Population estimates come from 2009 intercensal, bridged race U.S. population estimate data released by the U.S. Census Bureau. Population estimates for individuals ages 85-100+ years were provided as a NCHS special request file. We include individuals from the United States and U.S. territories, since the special request file of population counts of individuals ages 85-100+ does not distinguish residents by whether they live in the U.S. or U.S. territories. These data were selected because they are current and nationally-representative and include age in single years from 0 to 100+, detailed information regarding race/ethnicity and sex, and underlying cause-of-death.

We consider lifespan variability among the three largest racial/ethnic groups in the United States: Hispanics, non-Hispanic blacks, and non-Hispanic whites. Although American Indians/Alaska Natives, Asians/Pacific Islanders, or individuals of other races/ethnicities are included in the death and population data, their small population sizes preclude reliable calculations of life table functions. Non-Hispanic whites are the reference group in both decomposition analyses. This complicates interpretation somewhat since age-at-death variability is greater among blacks than whites and lower among Hispanics than whites. However, using whites as the reference group is the convention in racial/ethnic health disparities research. Positive effect values indicate a source of greater variability in age at death for the group with

less variability (Hispanics in the Hispanic-white decomposition and whites in the black-white decomposition).

Hispanic life expectancy estimates using unadjusted vital statistics data are potentially too high as a result of underascertainment (Hispanics reported as non-Hispanic on death certificates). This issue reduces the death count in the numerator of Hispanic death rate calculations and thus biases Hispanic death rates downward and life expectancy upward. However, Arias and colleagues (2010; 2008) found that Hispanic ethnicity reporting on death certificates is reasonably good and that "adjustment for death certificate misclassification did not significantly affect minority-majority mortality differentials" (Arias et al. 2008:1). In the first official release of U.S. Hispanic life tables, Arias (2010) applied classification ratios derived from the National Longitudinal Mortality Study to correct for ethnic misclassification on death certificates and made other adjustments to show that Hispanics continue to exhibit a greater life expectancy at birth relative to whites. We did not adjust our data using Arias' approach because the adjustments are for all-cause mortality and we focus on specific causes of death. Additionally, access to the Medicare data required to make Arias' corresponding adjustments to mortality rates among older non-Hispanic blacks and whites is restricted. Readers should be aware of these data quality concerns when interpreting our results.

We measure racial/ethnic differences in lifespan dispersion with variance in life table age at death conditional on survival to age 10 (S_{10}^2). We limit our sample to individuals aged 10 years and older since including infant and child mortality would bias the variance estimates. Although racial/ethnic mortality differences exist in infancy and childhood (and are quite wide among non-Hispanic blacks relative to non-Hispanic whites) (Hummer et al. 2007; Mathews and MacDorman 2013; Powers et al. 2006) relatively few deaths occur at these young ages.

Additionally, the causes of death that strike at ages 0-10 are very distinct from the dominant causes among older adults. The mortality schedule in developed countries has a bimodal functional form, with a relatively high risk in the first year of life, low risk throughout much of childhood and adolescence, and a gradual increase throughout adulthood (Murphy et al. 2012). Including infant and child mortality in the decomposition would severely skew the age at death distribution (Edwards and Tuljapurkar 2005). Truncation past infant ages is necessary to analyze variance around the old-age mode.

Analytic Approach

The decomposition process we use is developed and described in detail elsewhere (Nau and Firebaugh 2012). Briefly, this methodology decomposes racial/ethnic differences in lifespan variability into a) the contribution of each cause and b) the contributions of racial/ethnic differences in (i) cause-specific variability of ages at death (spread effect), (ii) number of deaths attributable to each cause (allocation effect), and (iii) cause-specific mean ages (timing effect). To generate the necessary input for the decomposition process and remove racial/ethnic differences in age structure, we constructed multi-decrement life tables for Hispanic, non-Hispanic black, and non-Hispanic white females and males ages 10 years and older. Underlying cause of death is classified according to the 10th revision of the International Statistical Classification of Diseases, Injuries, and Causes of Death (ICD-10). We exclude a relatively small number of deaths (2,736, 0.11% of total deaths) with missing underlying cause of death information. The sixteen cause of death groupings include heart diseases, malignant neoplasms (cancer), cerebrovascular diseases, respiratory diseases, traffic accidents, homicide, suicides, other external causes, influenza/pneumonia, sexually-transmitted or needle-transmitted diseases, septicemia, other infectious diseases, diabetes, nephritis, Alzheimer's, and a residual category of

other causes of death not elsewhere classified (N.E.C). This is the same cause-of-death scheme used by Nau and Firebaugh except that we distinguish suicide from other external causes since non-Hispanic blacks have lower risk of death from suicide relative to non-Hispanic whites (Harper, Rushani and Kaufman 2012; Rogers 1992; Singh and Siahpush 2001). After removing suicides, the external causes group mostly consists of falls (old age deaths) and poisoning through narcotics (young to middle ages).

Results

Racial/Ethnic Differences in Lifespan Mean and Variability

Figure 1 reports e_{10} (mean age of death conditional on survival to age 10) and S_{10} (standard deviation of age at death conditional on survival to age 10) and plots the d(x) function for each racial/ethnic population. Although we decompose variance (S_{10}^2) differences rather than standard deviation (S_{10}) differences, here we report standard deviation since this is the convention established by Edwards and Tuljapurkar (2005) and differences in standard deviation are easier to interpret than in variance. Hispanics (S_{10}=14.36 years) exhibit the lowest standard deviation of all three groups as well as the highest life expectancy (e_{10}=73.6 years). Non-Hispanic blacks exhibit lower life expectancy (e_{10}=65.3 years) and higher standard deviation (S_{10}=16.54 years) than the other two racial/ethnic groups. The difference in S_{10} between Hispanics and non-Hispanic whites is small (0.70) whereas the difference between non-Hispanic blacks and whites is much more substantial (1.47). Thus, while we decompose 100% of the difference in age-at-death variance for both minority groups relative to non-Hispanic whites, the black-white difference in variance is greater than the Hispanic-white difference.

The smaller numbers of deaths and population at mid-year among Hispanics and non-Hispanic blacks are responsible for the less smooth d(x) distributions and less-defined modal age

at death relative to non-Hispanic whites. However, we chose not to artificially smooth the age at death distribution. The life expectancy at birth (roughly e_{10} + 10) is lower than the modal age at death (the maximum point of the d(x) function) because of each group's long negative skew (Canudas-Romo 2010).

Leading Causes of Death and Their Contribution to Age-at-Death Variability

In the two decomposition tables, causes of death are listed in order of incidence for the minority group, with the most common causes (heart disease, cancer) at the top and least common causes of death at the bottom. Thus, the cause of death ordering in the tables is slightly different since the ranking of causes of deaths among Hispanics is different from that among non-Hispanic blacks. For instance, among blacks nephritis is the sixth most common cause of death whereas among Hispanics nephritis ranks tenth. Gross contributions (presented in the next three columns) are the sum of spread, allocation, timing, and joint effects and represent the percentage of the difference in variance explained by each cause of death. Incidences of causes do not necessarily match their contribution to racial/ethnic group differences in age-at-death variation. That is, some causes are overcontributors while others are undercontributors.

Among cause-specific contributions to the Hispanic-white difference in variance (Table 1), other external causes and suicide are major overcontributors, accounting for 94.8% of the gross Hispanic-white difference in lifespan dispersion but only 3.4% of deaths. Heart disease and respiratory disease also overcontribute somewhat. This may be a result of lower smoking prevalence among Hispanics relative to non-Hispanic whites since some mortality from both of these causes are attributable to smoking. Diabetes, sexually- or needle-transmitted diseases, and homicide are undercontributors and have negative gross effects. The percentage of deaths from the leading causes of death (heart disease, cancers, N.E.C., and cerebrovascular diseases) roughly

matches their gross contribution. Interestingly, 91.6% of the difference would remain if only women differed in mortality. In the black-white analysis (examined below), women also account for the majority of the racial difference in variance though to a smaller degree (67.7%).

Unlike our findings regarding Hispanic-white differences above, heart disease, the leading causes of death, overcontributes to the black-white difference in lifespan dispersion (Table 2). For instance, heart disease accounts for 28.8% of black deaths but 39.2% of the black-white difference in age-at-death variability. Among the other leading causes, cancer undercontributes while cerebrovascular disease is a slight overcontributor. Many of the external causes of death that generally strike at younger ages (suicide, traffic accidents, and other external causes) are undercontributors but some (diseases transmitted sexually or by needles and homicide) are overcontributors. Negative gross effects for respiratory disease, other external causes, traffic accidents, and suicide suggest that if the mortality profile of blacks resembled that of whites for these causes of death, the greater variability in age at death among blacks would increase rather than narrow.

Spread-Allocation-Timing Decomposition

When we decompose Hispanic-white differences in age-at-death variance by spread, allocation, and timing components (Table 1), we find that allocation effects explain much of the difference in age-at-death variance, especially for other external causes, cancer, and suicide. In fact, the total allocation effect exceeds 100 (131.79), but the negative total effects for spread and timing offset the excess 31.79% so that the total gross effect is 100%. Diabetes, sexually- and needle-transmitted disease, and homicide mortality have sizable, negative allocation effects, indicating that if incidence of these causes among Hispanics became like that of whites, age-at-death variance for Hispanics would increase toward that of whites rather than narrow. Cancer

presents a unique case; spread and timing effects are negative and narrow the difference while allocation and joint effects are positive and expand the difference. The small Hispanic-white difference in age-at-death variance revealed in Figure 1 may account for the particularly large joint effects for cancer (7.7), suicide (10.2), and homicide (-10.8) since now, two relatively small differences in allocation-spread or allocation-timing effects will amount to an interaction effect that explains a high percentage of the small Hispanic-white variance gap.

In the black-white variance decomposition (Table 2), spread effects are the primary contributor to the greater variability in age at death among blacks relative to whites. 84% of the greater variance in longevity of whites would remain if blacks and whites differed only in within-cause variability. Heart diseases and causes not elsewhere classified alone are responsible for more than half of the difference in variance. The spread effect for heart disease is 30.4, indicating that 30.4% of the difference in variance would remain if blacks and whites differed only in variation in heart disease mortality. The heart disease spread effect among males (19.8%) is nearly twice the effect among females (10.7%). Figure 2 shows this greater dispersion in heart disease deaths among blacks relative to whites for females and males.

Effects are generally similar for females and males. However, there are some substantial sex-specific differences in variance by race/ethnicity. It's particularly noteworthy that in the Hispanic-white decomposition, total spread and timing effects operate in opposite directions for females and males and have effects of relatively large magnitude. Additionally, in the black-white decomposition analysis, we observe large homicide allocation effect for males only. The male allocation effect for homicide is 38.2, suggesting that 38.2% of the difference in variance would remain if blacks and whites differed only in incidence of male homicide mortality.

Discussion

Population health researchers have documented racial/ethnic disparities in life expectancy (the mean age at death), but less attention has focused on racial/ethnic disparities in variability around that mean. Our results suggest that the mortality profile of U.S. Hispanics is characterized by a higher life expectancy and lower lifespan variability relative to non-Hispanic whites whereas non-Hispanic blacks exhibit lower life expectancy and greater age-at-death variability relative to whites. While the Hispanic-white difference in S_{10} is small, the observation of less variability in age-at-death among Hispanics relative to whites despite Hispanics' lower socioeconomic status represents an additional component for the epidemiologic paradox. That is, Hispanics are socioeconomically disadvantaged relative to non-Hispanics whites and lifespan dispersion is generally lower among groups with greater levels of socioeconomic status (i.e., educational attainment and income) compared to their socioeconomically disadvantaged counterparts (Brown et al. 2012; Edwards and Tuljapurkar 2005; van Raalte et al. 2011; van Raalte et al. 2012), yet Hispanic adults exhibit a mortality advantage for dispersion in age at death. Our results using recent nationally-representative data, contrast with those of Go and colleagues (1995), who found that Hispanics in California had greater lifespan dispersion and lower longevity relative to non-Hispanic whites and blacks.

Our findings of greater age-at-death variability among blacks than among whites closely match those of Edwards and Tuljapurkar (2005), Tuljapurkar and Edwards (2011), and Lynch et al. (2003) and warrant continued concern for black-white mortality differentials. Decomposition of variance along both a cause-of-death axis and spread-allocation-timing axis will inform efforts aimed at reducing black-white age-at-death variability. For instance, two of the primary contributors to blacks' greater dispersion relative to whites are heart disease spread effects and

homicide allocation effects (particularly among males) Not only do these causes have very different etiologies and age patterns but they contribute to overall variance through different components of variance, spread and allocation, respectively.

While lifespan variability declined during the epidemiologic paradox, progress since the 1950's has been stable (Kannisto 2000; Ouellette and Bourbeau 2011; Wilmoth and Horiuchi 1999). Mortality compression among some populations may be slowing down or stagnant and there is the potential for decompression. The Hispanic advantage in age-at-death variability is not guaranteed to continue. Changes in the composition of the U.S. Hispanic population, such as a reduction of the foreign-born population (who are generally healthier than the U.S.-born Hispanic population at the time of migration) or acculturation to health-compromising U.S. behavioral norms with increased duration in the U.S. could expand Hispanic variability. Alternatively, the small difference in lifespan variability between Hispanics and non-Hispanic whites resembles the shifting mortality scenario, in which lifespan variability has reached such a low level that decreasing mortality is associated with the d(x) curve maintaining its shape rather than becoming further compressed as life expectancy increases (Canudas-Romo 2008). Hispanics and non-Hispanic whites may have maximized length of life in the current socioenvironmental context. However, lifespan variability among U.S. adults is greater relative to most other industrialized nations, suggesting the potential for greater mortality compression among U.S. Hispanics and whites. Lynch et al. (2003) found that from 1972 to 1990, mortality compression occurred among blacks but not among whites, suggesting that the black-white gap in age-at-death variability could be decreasing even though little progress has been made in reducing the black-white mortality gap over the past few decades (Hummer and Chinn 2011; Satcher et al. 2005). Future research should examine longitudinal trends in racial/ethnic

differences in lifespan dispersion.

Heart disease and respiratory disease overcontribute to Hispanics' reduced lifespan dispersion relative to non-Hispanic whites, mostly due to allocation effects. This may be a result of lower smoking prevalence among Hispanics relative to non-Hispanic whites since mortality from both of these causes are attributable to smoking. Lower smoking prevalence among Hispanics, particularly foreign-born Hispanics, may account for a substantial amount of the Hispanic mortality advantage (Blue and Fenelon 2011; Fenelon Forthcoming). Future studies should experiment with distinguishing cancers of the lung, bronchus, and trachea from other cancers or to isolate the proportion of deaths attributable to smoking (Preston, Glei and Wilmoth 2010; Rostron 2013) to further explore the degree to which racial/ethnic smoking differentials contribute to Hispanics lower age-at-death variability. Future research should explore alternative cause of death groupings, such as "actual causes of death" (Mokdad et al. 2004), preventable versus non-preventable causes (Phelan et al. 2004), or causes amenable to medical care or public health intervention (Macinko and Elo 2009).

Decomposition of variance rather than an alternative variability indicator was key to identifying cause-specific effects on racial/ethnic differences in variability. If we used standard deviation above the modal age at death or another indicator that focuses on old-age mortality rather than mortality in young and middle adulthood, black-white differences in lifespan variability would likely be somewhat obscured. U.S. blacks are characterized by much higher mortality in young adulthood (by a factor of 2 to 1), but at older adult ages, African Americans appear to have comparable or superior mortality profiles relative to whites, due to either data quality issues or the survival of the most robust individuals of an initially heterogeneous black population. That is, the frailest members of the population exhibit a greater mortality risk and die

at younger ages whereas the more robust individuals are exhibit lower mortality risk.

A number of limitations must be considered when interpreting our findings. The sixteen causes of death we examine in our decomposition analyses do not operate independently but rather compete against one another. Individuals who die from causes most prevalent at young adult ages are removed from the population at risk of the chronic and degenerative causes that strike at older adult ages. Also, racial/ethnic or age differences in cause-of-death reporting could influence our results in unobserved ways (Noymer, Penner and Saperstein 2011). Classification of underlying cause of death among older adults can be uncertain given that they may experience multiple contributing factors (Rosenberg 1999). Also, the full mortality burden of diseases that are generally regarded as contributing factors rather than the underlying cause of death (particularly diabetes) is often unclear.

As we report above, data quality issues (i.e., selective in- and out-migration, racial/ethnic misclassification, age misstatement, differential record linkage, Census undercount, etc.) potentially bias mortality estimates for U.S. minority populations (Arias 2010; Elo et al. 2004; Lariscy 2011; Markides and Eschbach 2005; Palloni and Arias 2004; Preston et al. 1996; Turra and Elo 2008). Hispanic life expectancy estimates using unadjusted vital statistics data may be too high as a result of ethnic misclassification on death certificates (Hispanics reported as non-Hispanic) and salmon bias (Hispanics may die after returning to their country of origin and not have a U.S. death certificate). These issues reduce the death count in the numerator of Hispanic death rate calculations and bias Hispanic death rates downward and life expectancy upward. Our estimates of life expectancy conditional on survival to age 10 are greater than the e_{10} estimates that incorporate Arias' adjustments (Arias 2010). We did not adjust our data using Arias' approach because the adjustments are for all-cause mortality and we focus on specific causes of

death. Additionally, access to the Medicare data required to make Arias' corresponding adjustment to mortality rates among older non-Hispanic blacks and whites is restricted. Readers should be aware of these data quality concerns when interpreting our results.

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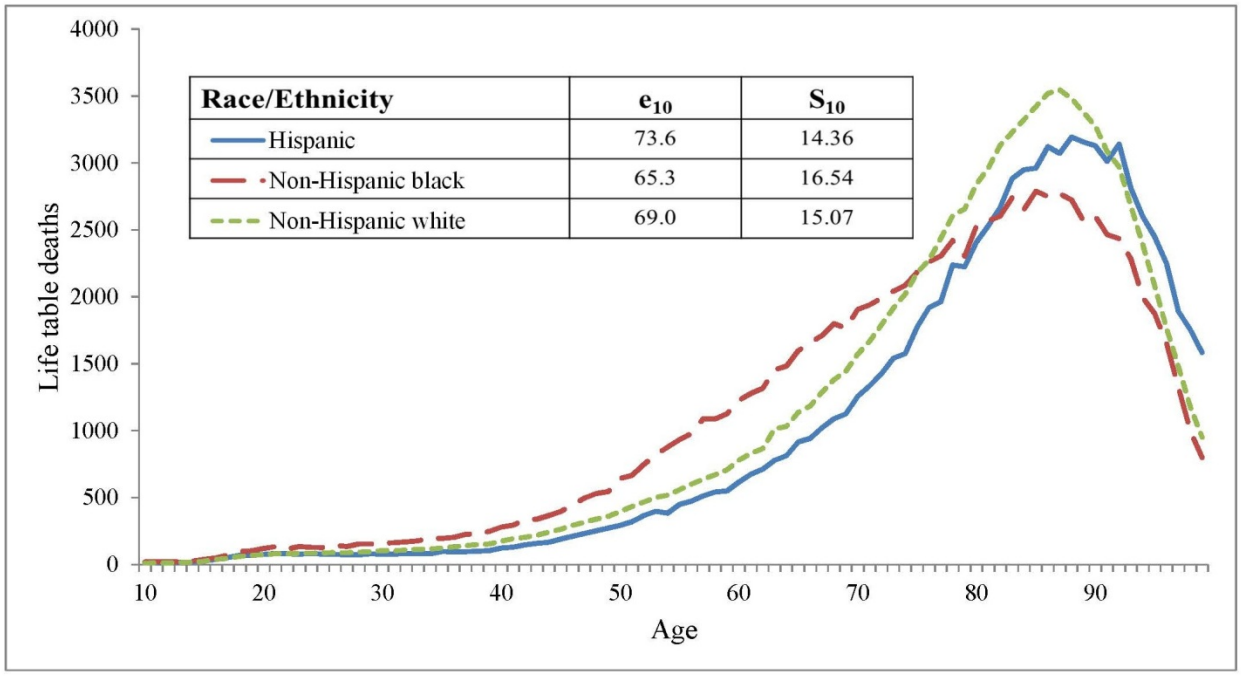
Figure 1. Mean age at death and variability in age at death by race/ethnicity



Source: 2009 U.S. National Vital Statistics System data.

Table 1. Spread-Allocation-Timing decomposition results for the difference in variance of life table ages at death for Hispanics relative to non-Hispanic whites

Cause of death	% of deaths among Hispanics	Gross contribution			Spread effects			Allocation effects			Timing effects			Joint effects		
		Total	F	M	Total	F	M	Total	F	M	Total	F	M	Total	F	M
Heart disease	28.80	35.11	24.28	10.83	13.84	8.62	5.22	7.22	-1.30	8.52	13.95	16.67	-2.72	0.10	0.30	-0.20
Cancers	19.47	10.42	7.37	3.05	-15.04	-6.17	-8.87	38.86	19.19	19.67	-21.06	-8.82	-12.24	7.66	3.17	4.49
N.E.C.	19.25	14.63	19.41	-4.78	-1.52	4.30	-5.82	14.41	7.08	7.33	1.55	8.92	-7.36	0.18	-0.90	1.08
Cerebrovascular	6.34	-1.66	2.55	-4.21	-4.72	-2.15	-2.57	-1.21	-0.30	-0.91	4.44	4.96	-0.52	-0.17	0.04	-0.21
Diabetes	4.57	-11.66	-5.77	-5.89	2.93	1.57	1.36	-14.76	-8.58	-6.17	-1.31	-0.13	-1.18	1.48	1.37	0.10
Alzheimer's	4.22	8.78	6.98	1.80	0.49	0.54	-0.05	1.92	1.41	0.51	6.92	5.45	1.46	-0.55	-0.42	-0.12
Respiratory	4.01	13.72	7.77	5.94	1.12	1.20	-0.08	13.86	7.85	6.01	-1.36	-1.34	-0.02	0.10	0.06	0.04
Influenza/pneumonia	2.92	-0.16	0.88	-1.04	0.41	0.56	-0.14	-3.20	-1.86	-1.34	2.18	1.78	0.40	0.44	0.40	0.04
Other external	2.87	65.87	25.47	40.40	2.43	6.16	-3.73	65.15	19.81	45.34	-0.54	3.56	-4.09	-1.18	-4.06	2.88
Nephritis	2.51	-3.49	-0.82	-2.67	-1.77	-0.44	-1.33	-2.40	-1.44	-0.96	0.81	0.95	-0.14	-0.13	0.11	-0.24
Septicemia	1.47	-1.40	0.11	-1.50	-0.99	0.01	-0.99	0.12	0.07	0.05	-0.54	0.03	-0.57	0.01	0.00	0.02
Traffic accidents	1.04	-5.18	2.45	-7.64	-0.59	0.18	-0.77	7.83	3.24	4.59	-13.79	-1.11	-12.68	1.36	0.15	1.21
STD/NTD	0.72	-13.96	-3.64	-10.32	-0.33	-0.05	-0.28	-11.12	-3.77	-7.35	-0.96	0.10	-1.06	-1.55	0.08	-1.63
Other infectious	0.72	-1.16	-0.06	-1.09	-1.36	-0.66	-0.70	0.30	0.31	-0.02	-0.13	0.24	-0.38	0.04	0.04	-0.01
Suicide	0.57	28.97	7.24	21.73	-4.83	-1.05	-3.78	36.77	8.98	27.79	-13.20	-4.00	-9.20	10.23	3.31	6.91
Homicide	0.52	-38.83	-2.65	-36.18	0.06	0.14	-0.08	-21.97	-0.87	-21.10	-6.15	-1.62	-4.52	-10.77	-0.29	-10.47
Total	100.00	100.00	91.56	8.44	-9.85	12.75	-22.60	131.79	49.82	81.96	-29.19	25.62	-54.82	7.26	3.36	3.90

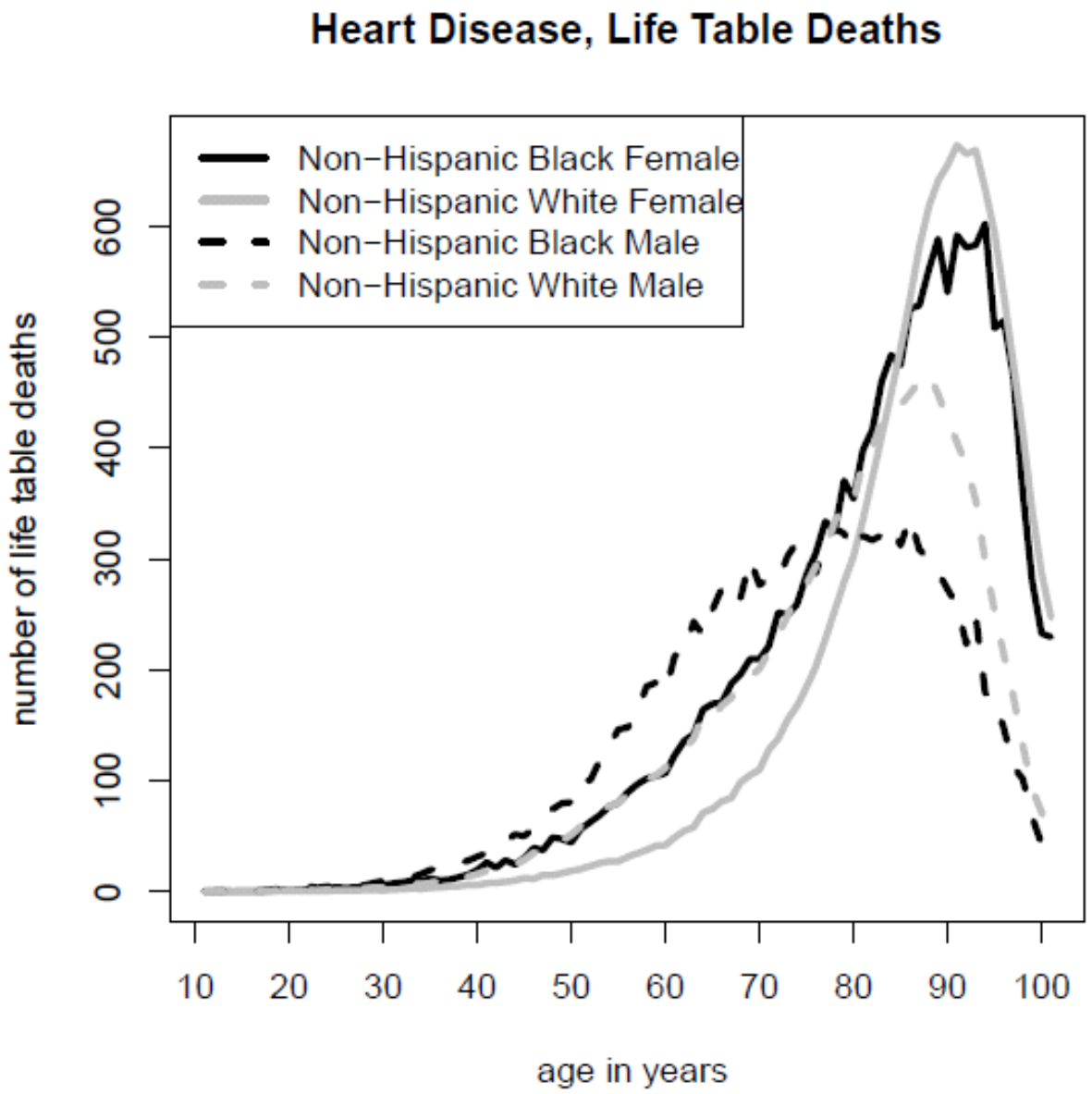
Source: 2009 U.S. National Vital Statistics System data.

Table 2. Spread-Allocation-Timing decomposition results for the difference in variance of life table ages at death for non-Hispanic blacks relative to non-Hispanic whites

Cause of death	% of deaths among blacks	Gross contribution			Spread effects			Allocation effects			Timing effects			Joint effects		
		Total	F	M	Total	F	M	Total	F	M	Total	F	M	Total	F	M
Heart disease	28.75	39.16	27.32	11.84	30.43	19.75	10.68	7.08	6.92	0.16	-0.96	-1.91	0.95	2.61	2.56	0.04
Cancers	22.35	5.18	5.39	-0.21	9.17	5.95	3.21	0.72	1.20	-0.48	-4.82	-1.88	-2.94	0.12	0.12	0.00
N.E.C.	17.39	20.62	17.31	3.31	26.56	17.55	9.02	-6.11	-1.31	-4.80	1.83	1.61	0.22	-1.66	-0.54	-1.12
Cerebrovascular	6.32	10.26	6.41	3.85	7.11	4.84	2.28	2.46	1.29	1.17	-0.29	-0.27	-0.03	0.98	0.55	0.43
Diabetes	4.33	9.04	5.97	3.07	0.91	0.42	0.49	7.28	4.74	2.53	0.00	0.14	-0.14	0.85	0.67	0.19
Nephritis	3.45	8.33	5.30	3.03	2.15	1.07	1.08	4.62	3.16	1.46	-0.10	-0.03	-0.07	1.67	1.10	0.56
Respiratory	3.27	-1.81	-1.62	-0.20	9.36	5.20	4.16	-6.89	-4.24	-2.65	0.43	0.41	0.02	-4.72	-2.98	-1.74
Alzheimer's	2.91	2.05	1.98	0.08	0.37	0.10	0.28	-3.21	-2.27	-0.94	6.96	5.62	1.34	-2.07	-1.47	-0.60
Other external	2.36	-39.10	-10.29	-28.80	-5.22	-0.87	-4.35	-33.20	-9.15	-24.05	-5.32	-1.15	-4.17	4.65	0.88	3.76
Septicemia	2.30	5.38	3.45	1.93	0.99	0.56	0.44	3.48	2.19	1.29	0.10	0.13	-0.03	0.81	0.57	0.24
Influenza/pneumonia	2.03	1.92	1.43	0.49	2.93	1.88	1.05	-0.78	-0.41	-0.37	0.04	0.12	-0.09	-0.27	-0.17	-0.10
STD/NTD	1.34	15.77	6.35	9.42	0.11	0.01	0.10	13.88	4.49	9.39	0.21	0.30	-0.09	1.58	1.55	0.03
Homicide	1.30	46.79	4.34	42.44	-0.47	-0.16	-0.31	38.21	3.66	34.55	1.54	0.39	1.15	7.51	0.45	7.06
Traffic accidents	0.94	-6.87	-2.29	-4.58	-1.53	-0.56	-0.97	-3.38	-1.50	-1.88	-2.36	-0.39	-1.97	0.40	0.16	0.25
Other infectious	0.57	0.99	0.52	0.47	1.54	0.93	0.62	-0.38	-0.24	-0.14	0.10	0.01	0.09	-0.27	-0.17	-0.10
Suicide	39.00	-17.73	-3.91	-13.82	-0.18	0.03	-0.21	-19.20	-4.23	-14.97	4.52	1.04	3.48	-2.87	-0.74	-2.13
Total	100.00	100.00	67.66	32.34	84.25	56.70	27.55	4.57	4.29	0.28	1.87	4.14	-2.27	9.31	2.53	6.78

Source: 2009 U.S. National Vital Statistics System data.

Figure 2. Variability in age at death from heart disease by sex and race



Source: 2009 U.S. National Vital Statistics System data.