



Evidence of accelerated aging among African Americans and its implications for mortality



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ABSTRACT

Blacks experience morbidity and mortality earlier in the life course compared to whites. Such premature declines in health may be indicative of an acceleration of the aging process. The current study uses data on 7644 black and white participants, ages 30 and above, from the third National Health and Nutrition Examination Survey, to compare the biological ages of blacks and whites as indicated from a combination of ten biomarkers and to determine if such differences in biological age relative to chronological age account for racial disparities in mortality. At a specified chronological age, blacks are approximately 3 years older biologically than whites. Differences in biological age between blacks and whites appear to increase up until ages 60–65 and then decline, presumably due to mortality selection. Finally, differences in biological age were found to completely account for higher levels of all-cause, cardiovascular and cancer mortality among blacks. Overall, these results suggest that being black is associated with significantly higher biological age at a given chronological age and that this is a pathway to early death both overall and from the major age-related diseases.

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1. Introduction

Race is linked to striking health disparities in the United States. Overall, blacks experience death and disease much earlier in the life course than do whites, which may suggest that on average blacks are aging faster (Hayward et al., 2000). Because the progression of physiological deterioration that accompanies aging may be strongly related to environmental factors (Finch and Tanzi, 1997), it is conceivable that the various social, economic, mental, and physical factors encountered by many racial minorities throughout their lives may be capable of causing an acceleration of the aging process.

A number of factors have been shown to contribute to racial differences in morbidity and mortality: socioeconomic status (SES) (Hayward et al., 2000; Franks et al., 2006), neighborhood (Williams and Collins, 2001; Acevedo-Garcia et al., 2008), availability of quality healthcare (Mayberry et al., 2000; 2008 National Healthcare Disparities Report, 2009), behaviors (Jackson et al., 2010), and psychological stress (McEwen, 1998). Over time, these factors have the ability to get “under the skin” and alter physiological functioning (Taylor et al., 1997; Kuzawa and Sweet, 2009). Blacks also

experience more discrimination, have less economic security and often live in worse neighborhoods, offering fewer nutritional options, worse air quality, and less access to recreational activities (Krieger et al., 1993; Ellen et al., 2001; Bell and Ebisu, 2012). These experiences may lead to higher levels of both physical and psychological stress with the potential to cause a myriad of biological changes with implications for aging. Finally, the higher prevalence of dangerous health behaviors, such as obesity, among blacks relative to whites (Flegal et al., 2012), are also believed to contribute to progressive breakdowns in biological tissues and systems, leading to widening gaps in physiological function. Growing disparity in physiological functioning due to the continual exposure to adverse conditions is the premise of the “Weathering Hypothesis”, which suggests that the negative effects of exposure to hazardous physical, social, and economic environments of socially disadvantaged racial groups accumulate over the lifespan and contribute to premature health deterioration, which may be indicative of an acceleration of the biological aging process (Geronimus et al., 2006).

The pace of age-related deterioration, potentially resulting from the accumulation of tissue and cellular damage to molecules like DNA and proteins, may be strongly influenced by the amount of wear and tear the body undergoes over time (Selye and Tuchweber, 1976). As a result, individuals exposed to hazardous environments may presumably age quicker, causing them to appear biologically

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older at a given chronological age. In fact, previous research examining race differences in cumulative biological risk have shown that on average, blacks have the same number of “high-risk” (indicated using clinically established cutoffs) physiological indicators as whites who are significantly older chronologically (Geronimus et al., 2006; Crimmins et al., 2007).

The earlier onset of aging-related deteriorations in physiological functioning is believed to also give rise to premature incidence of mortality. It has been reported that life expectancy for blacks is about 5 years less than whites (Arias, 2007) and contributes to approximately 100,000 excess deaths per year (Levine et al., 2001). Additionally, dramatic racial disadvantages have been found across multiple domains of health. Even in middle-age, blacks have been shown to have significantly higher prevalence of both fatal and non-fatal chronic conditions than whites (Hayward et al., 2000) and being black is often associated with earlier onset of many age-related chronic diseases (Bibbins-Domingo et al., 2009). This shift in the age curve—in which the onset of death and disease occurs earlier in life—is thought to explain why racial disparities in mortality risks cross-over in late life, resulting from mortality selection at earlier ages (Johnson, 2000; Manton et al., 1979). Taken together, this may suggest that a majority of those in the black population may be aging faster than the white population in the U.S.

Biological age measures were developed to quantify multi-system age-related changes on a physiological level and may be useful as proxies for the pace or extent of aging of an individual. While the concept of combining multiple measures into a single variable to model the rate of aging was proposed over fifty years ago, recent techniques have been found to be promising predictors of aging-related health outcomes (Cho et al., 2010; Levine, 2013). These measures utilize information from multiple biomarkers to determine where an individual lies on an aging trajectory. Typically, the trajectory is determined using a data driven approach that calculates age-associated differences in the various markers within a large representative sample (Bae et al., 2008; Krøll and Saxtrup, 2000; Nakamura and Miyao, 2007). As a result, biological age reflects the chronological age which on average is characterized by the specified biological profile. For example, someone with a biological age of 50 has the physiological functioning of the average 50 year old within the population. An individual's chronological age can be subtracted from biological age to determine whether the pace of aging for an individual or group is accelerated (i.e. they are older biologically than they are chronologically). For this reason, although it is not an actual marker of mortality risk, the concept of biological age may be useful for examining health disparities, as it allows us to directly estimate the degree of aging, or the difference between biological and chronological age, of disadvantaged groups, as well as compare biological age for race groups at varying chronological ages.

Using data from the National Health and Nutrition Examination Survey (NHANES III), this study examines 1) the racial difference in the pace of aging across ages and by ten-year age groups, to determine if blacks are aging biologically faster than whites and whether disparities in the pace of aging decline or cross-over in later life; and 2) whether these differences in the pace of aging, account for racial disparities in age-specific risks of all-cause mortality, cardiovascular disease (CVD) mortality, and cancer mortality. Overall, we hypothesize that blacks will have higher levels of accelerated aging compared to whites. However, these differences should decrease with age since the most disadvantaged are selected out of the population earlier. Finally, we hypothesize that racial differences in pace of aging will account for the higher mortality risk among blacks.

2. Materials and methods

2.1. Study population

We use data from NHANES III, a nationally representative, cross-sectional study conducted by the National Center for Health Statistics (NCHS) between 1988 and 1994. Data were collected from at-home interviews and examinations taking place at a Mobile Examination Center (MEC). Further details of recruitment, procedures, population characteristics and study design are available through the Centers for Disease Control and Prevention (U.S. Department of Health and Human Services, 2001). Our analytic sample ($N = 7587$) was restricted to black and white subjects ages 30–89. Hispanics were excluded because, although they have slightly higher life expectancy than whites, nativity is believed to be may be a major factor in this observation, and there is evidence that these differences may be explained by the “salmon hypothesis” which suggests that many Hispanics may return to their country of origin once they become ill and thus their mortality is not observed (Crimmins et al.). Those over age 89 were excluded given that NHANES III top-codes age at 90. Complete biomarker data was available for approximately 70% of the age-eligible sample. However, excluded subjects were more likely to be black, have lower education, were older, and were more likely to die between baseline and follow-up.

2.2. Biological age measure

Our estimation was calculated using information for ten biomarkers—C-Reactive Protein (CRP), Serum Creatinine, Glycosylated Hemoglobin (HbA1c), Systolic Blood Pressure, Serum Albumin, Total Cholesterol, Cytomegalovirus Optical Density (CMV), Serum Alkaline Phosphatase, Forced Expiratory Volume at 1 s (FEV1), and Serum Urea Nitrogen. These markers were selected because they had been suggested as potential biomarkers of aging, used in prior estimations of Biological Age using the NHANES III sample (Levine, 2013), or had been found to significantly correlate with chronological age at $r > 0.10$. Together, these biomarkers provide an indication of metabolic, cardiovascular, inflammatory, kidney, liver, and lung functioning.

Biological age was calculated in accordance with the method proposed by Klemmer and Doulal (2006). This method has been shown to predict death more accurately than other well-known Biological Age algorithms, such as Multiple Linear Regression and Principle Component Analysis, and was found to be a better indicator of mortality risk than chronological age (Levine, 2013). The estimated Biological Age calculation combines information from $m = 10$ regression lines of the $m = 10$ biomarker indicators regressed on chronological age (Eq. (1)).

$$BA = \frac{\sum_{j=1}^m (x_j - q_j) \frac{k_j}{s_j^2} + \frac{CA}{s_{BA}^2}}{\sum_{j=1}^m \left(\frac{k_j}{s_j} \right)^2 + \frac{1}{s_{BA}^2}} \quad (1)$$

In Equation (1), k_j and q_j are the slope and intercept, respectively, for each biomarker regressed on chronological age, x_j is the participant's measured value for a given biomarker, s_j is the root mean squared error of a biomarker regressed on chronological age, and CA represents chronological age. Additionally, s_{BA}^2 is the variance of the random variable, R_{BA} , which represents the difference between participants' biological and chronological age.

2.3. Sociodemographic characteristics

Chronological age, race, sex, education, and smoking were based on self-reports. Subjects were categorized into two race

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