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Timing, Accumulation, and the Black/White Disability Gap in Later Life

A Test of Weathering

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Examining the Black/White disability gap among older adults, this study focuses on the role of timing in racial inequality over time. Using the Duke Established Populations for Epidemiologic Studies of the Elderly (EPESE), the author reexamines Black and White disability trajectories with attention to timing of onset. In addition, known mediators are examined for their relative impact on onset and accumulation of disability. The author finds that diverging trajectories of Black and White disability, evidence of a cumulative disadvantage argument, are fueled solely by differences in onset. A more nuanced picture of racial disparities arises when controls are included, lending support to a weathering hypothesis. Access to health care is primary in explaining the Black/White disparity. The author concludes that timing is integral to the study of health trajectories and that research using cumulative disadvantage benefits from supplemental theories with specific assertions as to timing, including weathering.

Keywords: disability; race; inequality; trajectories; cumulative disadvantage

Research now consistently reports increases in active life in the United States, especially at later ages (Crimmins 2004; Freedman, Martin, and Scheoni 2004). Although disability is decreasing in this age group, findings from the 1999 National Long Term Care Survey report that roughly 20% of Medicare beneficiaries over the age of 65 have some level of chronic disability

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(Manton and Gu 2001). Leveille and others (2000) replicate these findings and report that at age 80 for women and 85 for men, the percentage of disabled elderly increases from roughly 20% to about half. Even with observed increases in active life (see Freedman et al. 2004; Crimmins 2004), there is a growing number of elderly requiring services and policy that reflect their specific health and functional needs (Spillman 2004; Uhlenberg 1992).

However, increases in health and functioning have not been universal; health disparities by race or ethnicity and socioeconomic status have widened among adults (Williams and Collins 1995). For example, Black individuals are at higher risk of morbidity, disability, and mortality compared to Whites almost consistently across the life course, therefore enjoying both fewer years overall and fewer years in good health compared to their White counterparts (Hayward and Heron 1999; Kitagawa and Hauser 1973; Manton, Patrick and Johnson 1987). Racial and ethnic disparities in disability are marked and notable, and stretch into the latest ages. Furthermore, these disparities widened throughout the 1980s (Clark 1997), and differentials in mortality are forecasted to increase (Levine et al. 2001).

A substantial body of research has addressed this topic among older adults, and increasingly this research has focused on whether inequality grows over time as evidence of cumulative disadvantage. Findings from these studies are still inconsistent, highlighting the importance of understanding the shape of disability accumulation among Blacks and Whites, the extent of the disparity over time as individuals transition into the latest ages, and the processes by which these disparities can be explained. Of particular importance to these research questions are issues of disability timing, accumulation, and selective mortality. If not explicitly taken into account, these components of disparities can lead to the confounding of significant effects or differences, possibly even underestimating the level of inequality between racial or ethnic groups (Kelley-Moore and Ferraro 2004).

In this study, I examine the Black/White disability gap in later life in terms of persistent, widening, or diminishing inequality over time as individuals move through later life. Drawing on cumulative disadvantage theory and the weathering hypothesis, I examine both the timing of disability onset and the accumulation of disability severity over a decade using the Duke Established Populations for Epidemiologic Studies of the Elderly (EPESE). I then move on to known mediators of racial disparities in health, focusing in turn on socioeconomic status, chronic conditions, health behaviors, and access to care. I examine the relative impact of these factors on disability timing and accumulation, therefore establishing whether the factors that lead

to differences in becoming disabled for Blacks and Whites are the same as those exacerbating or ameliorating trajectories of disability accumulation thereafter.

Theoretical Framework

A cumulative advantage and/or disadvantage argument is often used in present studies to couch findings on the disability gap in later life. Cumulative advantage is traced back to what Merton (1968) termed "the Matthew Effect." The concept was applied to early recognition in scientific careers leading to greater (or accumulated) resources over time. These resources in turn lead to greater productivity over the career. The theory has since been applied more broadly in research on social stratification of education, income, mobility, and other aspects of socioeconomic status (DiPrete and Eirich 2006; Pallas 2003; O'Rand 1996; Rosenbaum 1979) showing that early advantage leads to accelerating advantage over time. Increasingly, cumulative advantage and/or disadvantage has been used in life course and health research (Crystal and Shea 1990; Dannefer 2003; Lynch 2003; Ross and Wu 1996), applied to studies of meaningful heterogeneity in processes of aging. The core components of support for cumulative advantage and/or disadvantage in this field are trajectories of interindividual inequality, differentials that increase despite comparable levels of status at a given time or age, and increasing inequality arising not from one static social position, but from "an interaction of a complex of forces" (Dannefer 2003, pp. S327) arising from structural stratification and differential access to opportunities and resources (Dannefer 2003; DiPrete and Eirich 2006; Ferraro and Kelley-Moore 2003; O'Rand 1996).

In the health disparities literature, cumulative disadvantage is used to describe accumulated exposure to negative events and stressors across the life course for racial and ethnic minorities, especially Black individuals. The strongest support for cumulative disadvantage has been evidence of a widening gap, or diverging trajectories of health, as individuals age. In a recent review of the literature, DiPrete and Eirich (2006) noted the use of cumulative advantage and/or disadvantage theory in describing a process of widening inequality in health and other literatures, stating that a truer test of the process would necessitate earlier levels of accumulation, predicting accumulation at later time points. In addition, the authors point to the importance of structural factors in facilitating or mediating such processes of growing inequality.

Although the importance of timing is inherent and sometimes addressed directly in studies testing cumulative disadvantage in health (see Dupre 2007; Ferraro and Kelley-Moore 2003), studies of racial disparities in disability usually focus on either differentials in timing of onset (e.g. Hayward and Heron 1999) or on diverging trajectories (e.g., Clark and Maddox 1992). No study to my knowledge has simultaneously addressed the racial disparity in both the timing and trajectory of disability, or has measured the mediating effects of factors known to be important in explaining racial disparities on these two components of accumulated inequality (more years with disability, accelerated accumulation of disability severity over time).

To better situate the current research questions regarding timing and accumulation, I draw on the weathering hypothesis to supplement the theoretical framework of cumulative disadvantage. The weathering hypothesis stems from the epidemiology literature with an emphasis on the unique experience of Black women, suggesting that they undergo greater health deterioration during reproductive years as a result of prolonged exposure to disadvantage (Geronimus 2001). The theoretical concept of weathering may be seen in other research that focuses on unique stressors and "high energy" coping strategies in explaining morbidity among middle-class Blacks (Geronimus et al. 2006, 2001; James et al. 1992; Light et al. 1995; Williams, Jackson, and Anderson 1997). Weathering also posits a premature aging process for disadvantaged groups, suggesting that they undergo both an earlier onset of poor health and an acceleration of poor health once the process of deterioration begins (Geronimus 2001). This process also selects Black individuals out of observation at certain ages due to selective mortality, therefore leaving relatively healthy Black individuals to be compared with both healthy and unhealthy Whites. Since weathering presents a clear structure in regard to timing and accumulation of disability in addition to selective mortality as a competing force, I argue for its use alongside a cumulative disadvantage argument when testing the Black/White disability gap among older adults over time. Some evidence has been found for weathering in disability outcomes in terms of active life expectancy (Geronimus 2001), using a dichotomous indicator of disability without focusing on increased severity in disability over time. Ferraro and others (2006) have also referred to a similar "cascade" of disadvantage in health outcomes occurring both earlier and more severely for Black individuals.

Disability in Later Life and Black/White Differences

Research has increasingly focused on the heterogeneity among disabled elderly and the dynamic nature of the disablement process (see Crimmins

and Saito 1993). Transitions in impairment status have also been predicted by age, gender, race, previous functional state, income, and education (Beckett et al. 1996; Maddox, Clark, and Steinhauser 1994; Manton 1988; Schoeni, Freedman, and Wallace 2002). Other work has focused on demographic and social predictors of recovery (Kempen et al. 2001; Mendes de Leon et al. 1999). Increasingly, studies have focused on disability trajectories, extending findings on heterogeneity across individuals and differential risk given demographic and social predictors (see Li et al. 2000; Maddox and Clark 1992; Taylor and Lynch 2004; Verbrugge, Reoma, and Gruber-Baldini 1994).

A wealth of literature has emerged on Black/White disparities in disability in later life. Drawing back to the double jeopardy hypothesis (Dowd and Bengston 1978), these studies have examined whether the Black/White disparity persists into and across later life and whether these differentials are widening as in a cumulative disadvantage argument (Dannefer 1987; Kelley-Moore and Ferraro 2004) or lessening due to some leveling effect (see Dowd and Benston 1978 and Ferraro and Farmer 1996). The results from this body of research are fragmented and inconsistent, with some studies finding a persistent gap at the oldest ages (Clark and Maddox 1992; Kelley-Moore and Ferraro 2004), one that increases across time or age (Clark 1997; Liao et al. 1999), convergence at the latest ages (Guralnik et al. 1993; Johnson 2000), or no significant differences by race when controls are included (Taylor and Lynch 2004). These discrepancies may be attributed to a number of different factors (disability measurement, inclusion of other explanatory variables, selective mortality), but they are also likely affected by the methodological approaches undertaken by scholars. Arguments of health deterioration are difficult to test when only dichotomous indicators of disablement are used over time (Ferraro and Farmer 1996), and it is likely that mediating variables such as education and income work differently for onset, progression, and recovery of disability in later life (Ferraro and Farmer 1996; Mendes de Leon et al. 1997; Taylor 2005a; Zimmer and House 2003).

Methodologically, failure to estimate random timing of onset in growth models may also lead to confounding in findings of increasing racial inequality in disability. Figure 1 shows two hypothetical observed scenarios of trajectories (solid lines) of the Black/White disability gap in later life along with their hypothetical model estimated trajectories (dashed lines) to illustrate this possibility. The left part of the figure (A) reflects what has been previously found in bivariate analyses of race and disability over time as evidence of cumulative disadvantage, namely diverging trajectories of disability with Black individuals having both a significantly higher level of

disability at each wave on average, and a significantly higher rate of increase in disability over time. In this scenario, the estimated trajectories (dashed lines) accurately estimate what has been observed. This model assumes that both Black and White individuals "start" their disability trajectories at the first time point observed (here, age 65). However, it is very possible that White individuals have consistently later onset of disability compared to Blacks. The right part of the figure (B) reflects this alternate scenario (solid lines) along with the biased estimated trajectories (dashed lines). In this scenario, observed disability trajectories all start at 0 and increase at the same rate for Black and White individuals on average. In other words, the trajectories are no different in level or rate of accumulation, but the shift in timing of onset (Whites start their trajectories later on average) makes the estimated average trajectories (dashed lines) for the two groups diverge, producing a significantly higher estimated slope for Blacks that is an artifact of differential timing. Notice that a third solid line was also added in part B, indicating the likelihood that on average, Blacks enter later life with some disability (left censoring) compared to their White counterparts. This would lead to a biased estimate of the latent intercept (interpreted as estimated level of disability at baseline) in a traditional growth curve model along with the biased latent slope explained above. In sum, the model estimated disparity in the growth of disability for Black and White individuals over time is the same in both models, showing a significantly greater intercept and significantly greater slope for Blacks. What differs in the two scenarios are the observed experiences of Black and White individuals producing these estimates, where in part A the estimated trajectories predict the observed trajectories accurately and in part B the estimated trajectories are an artifact of differential timing only.

Underlying Mechanisms and Confounding Factors in Health Disparities Research

Multiple causal mechanisms are used to account for racial disparities in health across the life course: socioeconomic status (SES), differential health behaviors or lifestyle choices, increased exposure to stressors and lack of resources, and biological or genetic disadvantage. The SES argument is often emphasized in racial disparities, with many studies "explaining away" race effects when education and/or income variables are introduced into models (Kahn and Fazio 2005). In fact, there is difficulty in teasing out the effects of race and SES since these processes of disadvantage are so closely linked (Hummer 1996). Another concentration of literature focuses on differences

Figure 1
Hypothetical Scenarios of Racial Inequality in Disability Over Time
Including Observed and Model Estimated Trajectories (A): Diverging
Trajectories (B): Differential Timing
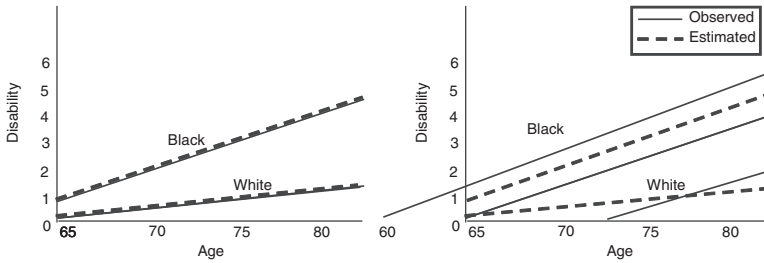
Note: The left part of the figure (A) reflects diverging trajectories of disability for Blacks and Whites. Here, the estimated trajectories (dashed lines) accurately estimate what is observed (solid lines). The right part of the figure (B) reflects an alternate scenario where the estimated trajectories are identical to (A), but are biased due to observed differences in timing of onset, including the likelihood that Blacks enter the observation period with disability more often (left censoring).

in lifestyle or health behaviors, linking higher prevalence of smoking, inactivity, and overweight among minority populations to increased morbidity and mortality, since chronic disease is so closely linked to disability and death (Bolen et al. 2000). A more sociological perspective situates racial disparities in a context of disadvantages, or stressors, accumulating over the life course to yield both poorer physical and mental health (Link and Phelan 1995; Williams 2005). This argument includes the increasingly recognized influence of racism as a predominant chronic stressor over the life course (Kessler, Mickelson, and Williams 1999; Jackson 2005; Williams 2004). In addition, access to health care and other health promoting resources have been emphasized in racial disparities, since preventive care and insurance status are such strong predictors of health outcomes (Blustein 1995; Fang and Alderman 2004) and racial minorities are less likely to have access to these resources (Freeman et al. 1987; Lurie 2002; Williams and Collins 1995) over and above traditional socioeconomic indicators (Ferraro et al. 2006; Mutchler and Burr 1991; Weinick, Zuvekas, and Cohen 2000). Finally, biological or genetic factors are historically argued to be at the root of racial disparities research in this country (Krieger 1987) and still emerge as underlying explanations when other demographic and social factors have been controlled (Kaufman, Cooper, and McGee 1997).

Researchers advocating for the extension and further application of cumulative disadvantage in health and life course research note the importance of causal pathways and structural mechanisms at work in placing minority groups at risk over time. In addition, the weathering hypothesis posits a premature aging process among racial minorities fueled by high energy coping mechanisms used in response to accumulated individual and structural disadvantage in society. Therefore, I test the mediating roles of SES, chronic conditions, health behaviors, and access to care in trajectories of disability for Black and White older adults.

Research Questions

In the present study, I examine the Black/White disability gap over time for older adults. Previous research has found inconsistent support for a persistent, widening, and converging gap. Although many studies have specifically addressed the nature of disability trajectories in later life (see Taylor and Lynch 2004), they have not specifically accounted for timing of onset in trajectory models. It has been shown that this may confound findings and show an accelerated accumulation of disability among a disadvantaged group when only a delay in onset is at work (Taylor 2005a). In addition, the weathering hypothesis posits that trajectories of poorer health among racial minorities are at least in part fueled by the earlier onset of poor health. Therefore I examine the disability gap over time in two components, the differential timing of disability onset, and the trajectory of disability level over time once initial onset has occurred. I expect that timing will play a crucial role in the Black or White disability gap, and that race differences in onset will be robust to mediators. In addition, I expect findings for race to be consistent with the weathering hypothesis, such that Black individuals will undergo a process of functional impairment that occurs both earlier than for Whites, and is increasingly more severe over time compared to Whites.

I will also test the relative roles of SES, chronic conditions, health behaviors, and access to care in explaining disparities in disability onset and accumulation. SES has traditionally been one of the greatest mediators in explaining health disparities and is more predictive of disability among older adults than race in some studies (Kahn and Fazio 2005; Taylor and Lynch 2004). Chronic conditions, and especially their incidence over time, have also been shown to explain the Black/White disability gap in later life (Kelley-Moore and Ferraro 2004). Health behaviors are also often used in explaining Black/White health disparities at younger ages in adulthood

(Bolen et al. 2000). Finally, access to care is important as a structural component of status differentials, over and above individual-level SES, and racial minorities are more likely to be disadvantaged in access to care (Williams and Collins 1995). I expect SES and chronic conditions to have the largest mediating effects due to the most recent findings for these mediators in the literature on disability trajectories among older adults (Kelley-Moore and Ferraro 2004; Taylor 2005b).

Data and Measures

Data from the in-person 1986 to 1996 Duke *Established Populations for Epidemiologic Studies of the Elderly* (EPESE) are used to examine the role of timing in the Black/White disability gap in later life. The Duke EPESE is a multisite survey designed to investigate chronic disease, mortality, morbidity, and other measures of well-being among older adults. The Duke sample includes individuals aged 65 and older residing in the community of five counties in central North Carolina. In-person interviews were conducted in 1986/1987, 1989, 1992, and 1996. The Duke EPESE data has been used in many previous analyses of disability trajectories over time (see Kelley-Moore and Ferraro 2004; Taylor and Lynch 2004) and in testing racial disparities in other health trajectories (see George and Lynch 2003). This study extends previous literature using this robust data source to disentangle the separate processes of timing of initial onset and progression of disability thereafter, since it is probable that the causal mechanisms for these processes may hold shared and independent mediators (see House, Lantz, and Herd 2005) and since it is possible that failure to capture timing effects may lead to bias in estimates of disability trajectories over time (see Taylor 2005a).

The original Duke sample consisted of 4,162 individuals. Black individuals were oversampled (54%) and very few individuals reported a race other than White or Black. These individuals were dropped from analyses (26 cases). In addition, another 195 cases were dropped due to the fact that they had no report (missing) of disability status at baseline. Missingness on dependent variables after baseline is allowed in the present analyses through the use of a FIML estimator, which is default in *Mplus*, such that individuals are included in analyses even if they drop out of the sample (attrition) or are considered missing due to no longer being at risk (in the case of the onset variable). Missingness on independent variables comprised another 10% of the original sample (418 cases) and due to the dichotomous nature of the onset variable, individuals with missing values

on the independent variables could not be included in the model using the FIML estimator. Tests revealed that missingness was informative, therefore missing values were imputed to the mean of each variable and dichotomous indicators were included for each covariate where missingness was non-random in bivariate tests. In the final models, the only missing indicators that remained significant were those missing on heart attack and diabetes, therefore other indicators were dropped. All analyses are unweighted since the variables used for sample selection criteria were included as control variables in the models (Winship and Radbill 1994). Sensitivity analyses reveal that weighted analyses provide similar results, as do those including a control for mortality selection (see Ferraro and Kelley-Moore 2003).

The analyses first aim to examine the Black/White disability gap over time in this age group while taking into account both timing (random onset) and trajectories of disability level (growth) over the study period. Therefore, the outcome variable (disability level) was split into two variables. The first is a dichotomous indicator of disability onset at each wave, with individuals no longer at risk coded as missing. The second is a continuous measure of disability level, with nondisabled individuals coded as missing. Therefore, individuals only contribute to the disability trajectories once they had some nonzero value of disability. Disability is measured with a summed index (0-14) of Activities of Daily Living (ADLs) and Instrumental Activities of Daily Living (IADLs) that has been found to tap into the spectrum of disablement among older adults (Katz et al. 1963; Lawton and Brody 1969, Spector and Fleishman 1998). The ADL items include walking, bathing, grooming, dressing, eating, transferring, and toileting. The IADL items include using the telephone, driving and/or traveling, shopping, preparing meals, housework, taking medication, and handling finances. Both ADLs and IADLs measure whether an individual requires any help (from a person or device) for activities. Sensitivity analyses splitting ADL and IADL indexes reveal similar findings to those presented here, except that in models using ADLs only the disparity was smaller in magnitude and nonsignificant for the intercept. I argue that due to its small range (0-5 observed in this sample) and the use of combined indexes in previous work using methodological techniques with normality assumptions (Maddox and Clark 1992; Taylor and Lynch 2004), the combined index is most appropriate here.

The key independent variable is race (Black = 1) and therefore models including this variable and controlling for age (years) represent the first step of analyses in determining the nature of racial inequality in disability over time. Gender (female = 1) and marital status at baseline (married = 1) were included in subsequent analyses. Original analyses included controls for

widowhood, social support, depressive symptoms, and religious attendance, with similar results (models not shown). To explain disparities in onset and trajectories of disability level, nested models are used, including variables commonly found to explain race differences in the outcome variable. Socioeconomic variables include education, occupational prestige, employment, income, and assets. Education is measured in years. Occupational prestige is scored with the Hodge, Siegel, and Rossi (1964) scale, corresponding to the occupation the respondent reported as the longest job/career of their life. In addition, a dichotomous indicator of being a homemaker for the majority of life was included. Current employment is also measured at baseline with a dichotomous indicator for any type (full- or part-time) of employment. Income is self-reported at baseline for the past year representing both individual's and spouse's income from all sources (wages, salaries, Social Security, retirement benefits, help from relatives, rent from property, etc.) and recoded into thousands. Assets are measured through a sole dichotomous indicator of homeownership. Chronic conditions measures include heart attack, stroke, hypertension, cancer, diabetes, and hip fracture. Dichotomous indicators are created for each disease to represent having the disease or health event during the entire study period. Health behaviors include an indicator of obesity calculated from BMI at baseline, and two dichotomous indicators of smoking (current or former smoker) at baseline. Access to health care includes an indicator for urban status (urban = 1) measured from census tract in 1990. In addition, limited access to physician visits is measured at baseline through a single item asking whether the respondent ever refrained from visiting a physician due to cost. Dichotomous indicators are also included for Medicaid insurance and supplemental (additional to Medicare) health insurance at baseline.

Analytic Plan

The analytic strategy used here is a novel approach to growth curve models where the timing of initial onset is measured independently from the growth in disability level thereafter. It is possible that traditional growth models may reveal significantly divergent trajectories of degeneration in health or functioning where merely a shift in timing is occurring (see Figure 1) (Taylor 2005b). Previous literature (Melzer, Izmirlian, Leveille and Guralnik 2001; Zimmer and House 2003) also suggests delayed onset is an important factor in modeling disability trajectories over time and that the protective effects of covariates may differ for onset and growth and/or accumulation of

disability severity once initial onset occurs. The analytic strategy used here allows the separate but simultaneous testing of how mechanisms work over time. To model onset in a latent variable framework, I use observed binary event indicators to create a discrete-time survival model. For discussion on binary variables and thresholds in SEM and growth modeling, see Muthén (1996). The hazard probabilities (u_{ij}), expressed as a function of the observed covariates, are equivalent to the logistic regression of η_{ui} on the observed covariates:

$$P(u_{ij} = 1) = \frac{1}{1 + e^{-(\beta_j + \lambda'_{uj}\eta_{ui})'}} \ , \tag{1}$$

where β_j is the time-specific logit intercept parameter, λ_{uj} is a $f \times 1$ logit parameter vector that may vary across time and η_{ui} is a continuous latent variable vector representing onset (u) for each individual i. Since a common assumption in discrete-time survival modeling is that of a constant hazard, this assumption was tested to determine the appropriate functional form. The models presented here allow for an "unstructured" hazard across time, analogous to a piecewise hazard in continuous-time survival analysis (see Allison 1995; Masyn 2005). The unstructured model allows the hazard to take any functional form across time points. For discussion of discrete-time survival analysis and assumptions using latent variables, along with applications, see Muthén and Masyn (2005). For instruction on data manipulation and the interpretation of the hazard using a latent variable approach, see Masyn (2005). The binary latent variables modeling the effects of the covariates on disability onset constitute the first portion of the overall model. The second portion is a traditional two-factor latent growth process estimating the growth in the level of disability over time for each individual *given the individual's disability onset*.

To model disability growth given onset over time, the *y* variable (representing disability level) was modified such that an individual with no disability becomes missing on variable *y* (rather than having a value of zero as in traditional growth models) until the time period (*t*) when disability is observed, at which time individual *i* would have some nonzero value for variable *y*. The values for *y* would be nonmissing thereafter. The separate examination in growth of severity handles the confounding of timing shifts that may bias effects on the slope of growth over time (see Figure 1) since initial onset is both modeled separately and treated as time variant (known as random onset).

The level 2 equations may be expressed as follows:

$$\alpha_{yi} = \mu_{\alpha y} + \gamma_{\alpha_y \chi i} + \zeta_{\alpha_{yi}} \qquad (2)$$

$$\beta_{yi} = \mu_{\beta y} + \gamma_{\beta_y \chi i} + \zeta_{\beta_{yi}} \qquad (3)$$

For this analysis, a linear model fits best (models not shown), such that the factor loadings of η_{yt} are fixed at 0, 1, 2, & 3. For consistency with models in previous research, decreases in level of disability subsequent to first transition are modeled with the y variable even though u = missing on all time periods subsequent to onset. Therefore, any recovery is captured through the growth portion of the model. The onset and growth of disability are not allowed to covary in these models due to identification restrictions, a noted limitation of this type of analysis. Future research using alternate techniques should address the connection between the timing of onset and the progression of disability. For examples of growth modeling including random onset, see Albert and Shih (2003) and Taylor (2005b). All analyses are performed using *Mplus* version 4.0.

Results

Descriptive analyses in Table 1 provide comparative estimates of outcome and predictor variables for both Blacks and Whites. When considering disability totals at each wave using the summed scale (0 to 14), Black individuals have higher disability at each wave. In fact, Black individuals have .32 higher items at Wave 1 compared to Whites, and this disparity grows to .81 items at Wave 4. However, when the disability measure is split into onset and some level of disability among those reporting initial onset (prior to or at each wave), it is apparent that timing of onset is the force driving inequality in disability. Black individuals have 3.4% to 12.4% higher percentage of onset compared to Whites at each wave. This difference is substantial at the first wave (10.3% difference) indicating that Black individuals are more likely to enter the study disabled compared to Whites (left censoring). When considering level of disability given report of onset, White individuals have higher levels at the first two waves (.10 to .15 item difference) with Blacks having slightly higher levels at the last two (.05 to .21 item difference).

Analyses of predictor variables show differences in SES, chronic diseases, health behaviors, and access to care along with controls. Fewer Black individuals report being married compared to Whites in the sample. Black individuals are more disadvantaged across all measures of SES. Blacks also

Table 1
Descriptive Statistics for Dependent and Predictor Variables

	Total ($N = 3{,}941$)	Blacks ($n = 2{,}145$)	Whites ($n = 1{,}796$)
Black	54.4%	N/A	N/A
Age	73.4(6.6)	73.5(6.7)	73.3(6.4)
Female	65.2%	65.2%	65.1%
Married	38.3%	33.9%	43.6%
Education (years)	8.55 (4.08)	7.35 (4.02)	9.98 (3.67)
Prestige	35.56 (13.71)	31.38 (13.00)	40.56 (12.83)
Homemaker	13.3%	8.5%	19.0%
Employed	12.1%	11.1%	12.5%
Income ($)	10,346.01	7,118.62	14,200.55
	(9,983.06)	(6,328.29)	(11,983.71)
Home ownership	63.0%	55.9%	71.4%
Heart attack	20.3%	18.6%	22.4%
Stroke	16.8%	17.7%	15.8%
Hypertension	65.6%	70.9%	59.3%
Cancer	19.8%	12.5%	28.5%
Diabetes	23.5%	28.3%	17.8%
Hip fracture	7.8%	4.9%	11.3%
Obese	18.0%	23.8%	11.0%
Current smoker	17%	16.3%	18.0%
Former smoker	26.5%	23.1%	30.6%
Urban	55.8%	56.3%	55.1%
Physician access	19.7%	19.9%	19.5%
Medicaid	7.1%	10.5%	3.0%
Supplemental insurance	51.9%	33.3%	74.2%
Disability 1986	1.11 (2.31)	1.26 (2.38)	0.94 (2.20)
Disability 1989	1.64 (3.00)	1.79 (3.03)	1.47 (2.94)
Disability 1992	2.17 (3.49)	2.49 (3.61)	1.78 (3.31)
Disability 1996	2.95 (4.06)	3.32 (4.19)	2.51 (3.86)
Onset 1986	33.5%	38.2%	27.9%
Onset 1989	25.4%	27.0%	23.6%
Onset 1992	26.6%	32.7%	20.3%
Onset 1996	31.1%	34.2%	28.4%
Disability level 1986 (0 = mis)	3.35 (2.92)	3.31 (2.86)	3.40 (3.03)
Disability level 1989 (0 = mis)	4.08 (3.51)	4.02 (3.41)	4.17 (3.66)
Disability level 1992 (0 = mis)	4.65 (3.83)	4.67 (3.78)	4.62 (3.92)
Disability level 1996 (0 = mis)	5.59 (4.87)	5.67 (4.09)	5.46 (4.04)
Disability 1986	1.11 (2.31)	1.26 (2.38)	0.94 (2.20)
Disability 1989	1.64 (3.00)	1.79 (3.03)	1.47 (2.94)
Disability 1992	2.17 (3.49)	2.49 (3.61)	1.78 (3.31)
Disability 1996	2.95 (4.06)	3.32 (4.19)	2.51 (3.86)
Onset 1986	33.5%	38.2%	27.9%
Onset 1989	25.4%	27.0%	23.6%
Onset 1992	26.6%	32.7%	20.3%
Onset 1996	31.1%	34.2%	28.4%
Disability level 1986 (0 = mis)	3.35 (2.92)	3.31 (2.86)	3.40 (3.03)
Disability level 1989 (0 = mis)	4.08 (3.51)	4.02 (3.41)	4.17 (3.66)
Disability level 1992 (0 = mis)	4.65 (3.83)	4.67 (3.78)	4.62 (3.92)
Disability level 1996 (0 = mis)	5.59 (4.87)	5.67 (4.09)	5.46 (4.04)

have higher percentages of stroke, hypertension, and diabetes while Whites report more heart attacks, cancer, and hip fracture. Black individuals are more likely to be obese, but less likely to smoke compared to Whites. In addition, Black individuals report more support from Medicaid and far less supplemental insurance compared to their White counterparts.

Table 2 shows bivariate models, controlling for age, replicating previous conventional growth models (A) supporting findings of increasing inequality in disability between Blacks and Whites (Kelley-Moore and Ferraro 2004; Liao et al. 1999). As evidenced by the significant-intercept term, Blacks have .28 more disability items compared to Whites at baseline. In addition, Black individuals accumulate disability at a rate of .13 items per wave faster than Whites. The results presented for the conventional growth model lend support to the cumulative disadvantage argument often used in this literature, since the trajectories of Blacks and Whites diverge significantly over time. In other words, the findings show a process of accelerated functional decline over time among Blacks compared to Whites.

When analyses were rerun modeling timing of onset and growth in disability level thereafter over time (B), the inequality shifts from the trajectory of disability level (intercept or slope) to a substantial and significant difference in onset. The hazard ratios are presented here for ease of interpretation. Therefore, Black individuals have 54% higher odds of onset than their White counterparts, but are not significantly different in the intercept or slope of disability level over time. This means that although evidence from conventional growth models shows a widening gap in the accumulation of disability, Black individuals do not have significantly higher levels of disability compared to Whites given initial onset. In addition, they do not have an accelerated rate of functional impairment over time. Rather, they live with comparable levels of disability for more years in later life compared to Whites. Fit statistics also show that the model incorporating random onset into the measurement of disability growth provides better fit (difference in BIC = 49,356.48) for these analyses.

Nested models including controls and known mediators of the Black to White disability gap are presented in Table 3. When gender and marital status are included (Model 1) the hazard ratio for Blacks decreases slightly (a decrease of 5.5%). Married individuals are less likely to experience onset (23% less odds) and have a .27 decrease in the slope of disability over time given initial onset. Model 2 includes socioeconomic controls. Individual indicators of socioeconomic position, including education, employment, income, and assets are highly significant in this model and work primarily to decrease the likelihood of disability onset. Education, being a homemaker

Table 2
Traditional Growth Curve (A) and Growth Curve With Random Onset (B) Models for the Black to White Disability Gap

$N = 3,941$	(A) Growth Curve		(B) Growth Curve With Random Onset		
	Intercept	Slope	Hazard OR	Intercept	Slope
Black	0.28*	0.13*	1.54*	0.07	–0.02
Age	0.10*	0.10*	1.11*	0.07*	0.06*
Intercept	–6.34*	–6.06*	—	–2.93*	–3.38*
Variance	3.84*	1.28*	—	6.89*	1.30*
Covariance	0.47*	—	—	0.88*	—
R^2	0.10	0.24	—	0.04	0.11
γs	1,1,1,1	0,1,2,3	1,1,1,1	1,1,1,1	0,1,2,3
LL (npar)	–40,673.84 (13)			–15,970.76 (19)	
BIC	81,414.00			32,038.46	

Note: OR = Odds Ratio; LL = Log Likelihood; BIC = Bayesian Information Criterion; npar = Number of Parameters.
*$p < .001$.

for most of life, and assets are among the strongest effects, with each year of education decreasing the odds of disability by 9%, those reporting being a homemaker having 37% more odds, and those reporting home ownership having 29% less odds. The effects of current employment are also substantial in magnitude but the causal direction is less clear for this control variable. The effects of SES on the growth of disability among those with initial onset are confined to education (each year of education decreases the slope of disability .03 items), employment (those reporting employment at baseline have 1.07 fewer disability items), and assets (individuals who report owning their own homes have .30 fewer disability items). In this model the racial inequality in onset is reduced by 70.5%, placing Black individuals at 15% greater odds of onset compared to Whites at comparable levels of SES. It should also be noted that the inclusion of socioeconomic variables reduces the effects of marital status to nonsignificance for both odds of onset and slope of disability given onset.

When chronic conditions are included (Model 3), all but cancer significantly increase the odds of onset. Stroke and hip fracture are the strongest predictors of onset, level, and accumulation of disability in this model. Hypertension and heart attack increase the likelihood of disability onset but are not significant in disability trajectories given onset. The race differential in onset increases when chronic conditions are controlled (an increase of 40%) and remained significant. The inclusion of health behaviors (Model 4)

Table 3
Nested Models for Mediators of the Black to White Disability Gap in Later Life

N = 3,941	Model 1 Hazard OR	Intercept	Slope	Model 2 Hazard OR	Intercept	Slope	Model 3 Hazard OR	Intercept	Slope	Model 4 Hazard OR	Intercept	Slope	Model 5 Hazard OR	Intercept	Slope
Black	1.51*	0.07	−0.03	1.15*	0.20	−0.15	1.21*	0.26	−0.07	1.19*	0.23	−0.08	1.09	0.10	−0.09
Female	1.10	−0.30	−0.02	1.06	−0.38*	−0.01	1.05	−0.32	−0.02	1.08	−0.50	−0.21	1.08	−0.07	−0.19
Age	1.10*	0.08*	0.06*	1.09*	0.08*	0.05*	1.09*	0.08*	0.05*	1.09*	0.07*	0.05*	1.09*	0.07*	0.05*
Married	0.77*	0.19	−0.27*	0.94	0.18	−0.20	0.94	0.23	−0.20	0.94	0.19	−0.19	0.92	0.24	−0.21
Education	—	—	—	0.91*	0.01	−0.03*	0.92*	0.00	−0.03	0.92*	0.01	−0.03	0.93*	0.01	−0.02
Occupational prestige	—	—	—	1.00	0.01	0.00	1.004	0.01	0.00	1.004	0.01	0.00	1.00	0.01	0.00
Homemaker	—	—	—	1.37*	0.19	0.09	1.38*	0.20	0.12	1.37*	0.18	0.11	1.32*	0.17	0.01
Employed	—	—	—	0.57*	−1.07*	0.09	0.59*	−0.93*	0.09	0.60*	−0.63*	−0.02	0.61*	−0.57*	−0.03
Income	—	—	—	0.99*	0.02	0.00	0.99*	0.02	−0.01	0.99*	0.01	0.00	0.99	0.01	0.00
Own home	—	—	—	0.71*	−0.30*	−0.07	0.73*	−0.28	−0.07	0.74*	−0.13	0.10	0.79*	0.03	−0.12
Heart attack	—	—	—	—	—	—	1.33*	−0.06	0.02	1.34*	−0.02	0.03	1.30*	−0.04	0.05
Stroke	—	—	—	—	—	—	2.06*	1.34*	0.41*	2.03*	1.05*	0.49*	2.03*	1.05*	0.48*
Hypertension	—	—	—	—	—	—	1.13*	−0.25	−0.05	1.13*	−0.06	−0.10	1.14*	−0.07	−0.08
Cancer	—	—	—	—	—	—	1.04	−0.02	0.04	1.04	0.07	0.03	1.03	0.04	0.03
Diabetes	—	—	—	—	—	—	1.27*	0.44*	0.09	1.26*	0.47*	0.08	1.23*	0.45*	0.09
Hip fracture	—	—	—	—	—	—	2.11*	0.66*	0.61*	2.14*	0.64*	0.64*	2.16*	0.62*	0.64*
Obese	—	—	—	—	—	—	—	—	—	1.21*	−0.21	0.09	1.21*	−0.20	0.09
Pack years	—	—	—	—	—	—	—	—	—	1.00	0.00	−0.01*	1.00	0.00	−0.01*
Urban	—	—	—	—	—	—	—	—	—	—	—	—	0.91	0.06	−0.05
Limited access	—	—	—	—	—	—	—	—	—	—	—	—	1.42*	−0.29*	0.06
Medicaid	—	—	—	—	—	—	—	—	—	—	—	—	1.30*	0.63*	−0.27
Supplemental insurance	—	—	—	—	—	—	—	—	—	—	—	—	0.72*	−0.15	−0.18
Intercept	—	−3.24*	−2.89*	—	−3.25*	−2.31*	—	−3.93*	−2.41*	—	−3.32*	2.33*	—	−3.17*	−2.28*
Variance	—	6.82*	−3.32*	—	6.76*	1.25*	—	6.36*	1.20*	—	−0.44	−2.71*	—	5.37*	1.25*
Covariance	—	1.25*	—	—	−0.85*	1.07*	—	−0.99*	—	—	−0.88*	—	—	−0.92*	—
R²	—	0.04	0.12	—	0.06	0.14	—	0.12	0.19	—	0.21	0.21	—	0.22	0.21
γs	1,1,1,1	1,1,1	0,1,2,3	1,1,1,1	1,1,1	0,1,2,3	1,1,1,1	1,1,1	0,1,2,3	1,1,1,1	1,1,1	0,1,2,3	1,1,1,1	1,1,1	0,1,2,3
LL (npar)	−15,948.11 (25)			−15,744.77 (43)			−15,517.80(67)			−15,367.38 (76)			−15,320.84 (88)		
BIC	32,023.76			31,708.91			31,377.41			31,122.48			31,090.62		

Note: OR = Odds Ratio; LL = Log Likelihood; BIC = Bayesian Information Criterion; npar = Number of Parameters.
*p < .001.

also does little to mediate the race differential in the onset of disability. Obesity does increase the odds of disability onset by 21%, and being a current smoker actually has a .01 item decrease in slope of disability given onset.

Model 5 includes variables on access to care including urban status, lack of physician access due to cost, and health insurance. Those reporting limited access to a physician have 42% increased odds of onset, yet this decreases an individual's level of disability by .29 items at each wave on average. Supplemental health insurance is also very protective for disability onset, decreasing an individual's odds by 28% even after controlling for individual-level SES indicators. The addition of access to health care variables in the model also reduces the somewhat small effect of income on onset (.01% decrease in odds for every thousand dollars) to nonsignificance. With the inclusion of these variables the effect of race on the onset of disability is reduced by roughly 53% and becomes nonsignificant. The inclusion of access to care variables therefore has a slightly smaller but substantial independent impact on the race differential on onset compared to SES. These findings indicate that access to care, along with SES, has the largest mediating effect on the Black/White disability gap in timing of disability onset in later life.

Conclusions

In sum, analyses suggest that the Black/White disability gap is primarily fueled by onset of disability when no other key mediators are controlled. These results provide reconciliation to previously inconsistent findings on increasing, persistent, and decreasing inequality in the Black/White disability gap in later life. Although this finding does not refute a cumulative disadvantage argument, it does call into question the way researchers test cumulative disadvantage over time for this and other topics in health and aging research. In addition, it suggests that measuring timing (random onset) in health trajectories is crucial in the policy and intervention-related implications researchers draw in conclusion to results. The findings from race-only models (controlling on age) in Table 2 provide clear but limited support for the weathering hypothesis, showing that without taking other structural and individual factors into account, Black individuals live more years with disability compared to Whites but that they do not suffer from significantly higher levels of disability, nor do they accumulate disability severity faster than their White counterparts over time once onset has occurred.

The inclusion of control and mediating variables in subsequent models provides results that coincide with previous findings, but also emphasizes

the importance of access to health care. Controlling for education, occupational prestige, current employment, income, and assets reduces the race differential in the odds of disability onset by roughly 70%, but this reduction decreases to 63% when chronic conditions and health behaviors are accounted for in the model. These findings on SES suggest some support for a cumulative disadvantage argument since they suggest a cumulative process of socioeconomic disadvantage for Blacks occurring from early life (education), through adulthood (occupational prestige, homemaker), into later life (income, assets) that explains part but not all of the race differential in disability onset. Support for the weathering hypothesis in previous research has also focused on the poorer health of Black individuals over the life course at comparable levels of SES (Geronimus et al. 2001; James et al. 1992; Light et al. 1995; Williams and Collins 1995).

Chronic conditions provide little explanation for the disability gap in these models, even when sensitivity analyses were run using time varying incidence variables at each wave (models not shown), a departure from previous literature (Kelley-Moore and Ferraro 2004). Health behaviors also had little effect on the magnitude of disadvantage for Blacks over time. Of the mediators included in analyses other than SES, access to health care had the largest effect on racial inequality in disability over time. Even when individual-level SES was held constant, limited access to a physician and health insurance had strong, independent, significant effects on the onset of disability trajectories. In addition, it was access to care that reduced the racial disparity in disability onset to nonsignificance. The preventive effects of access to care may also be seen in the counterintuitive effects of access to a physician on the intercept of disability, where those with limited access actually have lower levels of disability once initial onset occurs. This finding mirrors those on the compression of morbidity (Fries 1980) where individuals delay poor health and functioning such that they have considerable limitations once onset occurs.

The importance of access to care in these analyses points to both theoretical and substantive implications in light of previous research. Theoretically, structural components of disadvantage are at the heart of cumulative disadvantage and should be tested alongside individual achievement (education, income) in the causal pathway to health inequality. Weathering, like cumulative disadvantage, posits a cumulative process of risk exposure with roots both at the individual (coping strategies such as poor health behaviors) and structural (institutional racism, lower access to care, poor and segregated neighborhoods) levels. Substantively, although Social Security and Medicare were created to decrease poverty and poor health among older adults, inequality in

access and quality of care remain among the strongest predictors of func-
tioning among older adults. In addition, the finding that onset fuels the
Black/White disability gap, explained mainly through SES and access to
care, suggests that both selective left censoring (more Black individuals
aging into later life with disability compared to Whites) and a process of
socioeconomic position and access to resources earlier in the life course
(childhood, early adulthood) are central in the explanation of the disability
gap in later life, since Black individuals are both more likely to begin the
disablement process earlier than Whites and are less likely to have accu-
mulated resources that buffer or ameliorate the process in later life.

Although the findings on timing and accumulation present a significant
contribution to the future testing of cumulative disadvantage in health tra-
jectories over time, it should be noted that a truer test of cumulative disad-
vantage would include a significant effect of timing of onset *on* accumulation
(DiPrete and Eirich 2006). In other words, the most pure form of a cumula-
tive disadvantage process would be one where early onset was significantly
connected to a faster rate of accumulation. Unfortunately, these effects can-
not be tested here due to the way onset and accumulation are necessarily
coded in the analyses (an individual was only nonmissing on disability level
once they were no longer at risk (missing) of onset, making estimation of
this causal pathway impossible). Future analyses using alternate method-
ological approaches, however, should test this relationship for disability
(see Ferraro and Kelley-Moore 2003 for an example using exposure to obesity).

The current study has a number of other limitations worth mentioning
here. For simplicity and temporality reasons, I chose to incorporate time
invariant control and mediating mechanisms measured at baseline. The
exceptions to this were chronic conditions, since the incidence of these
variables is substantial in explaining the Black to White disability gap in
recent research (Kelley-Moore and Ferraro 2004). Future research should
examine more closely the changes that occur in marital status, income and
assets, and access to care (for example) and whether these changes matter
more in explaining racial disparities in timing and accumulation than base-
line indicators alone. Although individuals who died or dropped out over
the study period remained in the analyses and Blacks were oversampled at
each age, selective mortality occurring prior to the observation period can-
not be assessed in the present analyses, nor can the extent of selective left
censoring, or the amount of time individuals lived with disability prior to
baseline. It is most likely, however, that the findings here are conservative
in estimating racial inequality, since selective mortality would most likely
leave the most robust Black individuals surviving into the observation

period. Finally, the role of both individual and structural factors is noted here as primary to examining a cumulative disadvantage process, however, socioeconomic position is measured with commonly used, individual-level, observed indicators (education, income, occupation, assets) without emphasis on what these indicators provide distinctly in terms of health benefits or what may be left unmeasured. In addition, structural characteristics (other than urban residence) are measured only at the individual level. Future research should incorporate contextual variables, such as neighborhood-level poverty or access to care, to ascertain their role in the causal pathway between race and disability trajectories across the life course.

Increasingly, studies on health trajectories use a cumulative disadvantage framework to couch findings. With the findings presented here, I show that timing and accumulation are important and distinct factors that deserve explicit attention in testing the nature and magnitude of cumulative disadvantage. In addition, I incorporate the concept of weathering to supplement cumulative disadvantage theory as it is commonly used in health research, with specific assertions on racial inequality over time in reference to the timing and accumulation of disability in later life. I conclude that future research (both empirical and theoretical) using cumulative disadvantage theory in the health literature should draw on supplemental concepts such as weathering (Geronimus et al. 2001) or cascading disadvantage (Ferraro et al., 2006), to aid in a more explicit and directed testing of the theory in terms of timing and accumulation in health.

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