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Progressive and Accelerated Disability Onset by Race/Ethnicity and Education among Late Midlife and Older Adults

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Abstract

Objectives—This study explores the pace of severe disability onset with an emphasis on the role of race/ethnicity and education. More specifically, this research examines whether race/ethnicity and educational attainment are independent predictors of progressive and accelerated disability onset.

Methods—Using the Health and Retirement Study (HRS) Waves 2–10 (1994–2010), a series of discrete-time Cox proportional hazards models with multiple competing events were created to ascertain whether respondents developed progressive or accelerated disability in subsequent waves.

Results—Black and Hispanic respondents were at an increased risk of developing progressive disability. Respondents without a high school degree were more likely to experience progressive or accelerated disability.

Discussion—Low educational attainment was a particularly strong predictor of accelerated disability onset and may represent an acute lack of resources over the life course. Race and ethnicity were important predictors of progressive disability onset, which may reflect racial/ethnic variations in the disabling process.

Keywords

severe disability onset; progressive disability; accelerated disability; health disparities; The Health and Retirement Study

Introduction

There has been growing emphasis on the dynamic nature of disability including disability trajectories. Ferrucci et al. (1996) conceptualized the disabling process as having two main trajectories to severe disability: progressive and catastrophic disability, where gradual onset represented progressive disability and sudden onset represented catastrophic disability. According to Ferrucci et al. (1996), the type of pathology or disease is the major determinant of progressive and catastrophic disability. For example, progressive disability is thought to develop from degenerative diseases (e.g., arthritis, diabetes, etc.), while catastrophic disability is thought to develop from more sudden medical events (e.g., stroke, hip fracture, etc.). However, more recent research examining disability trajectories have noted several distinct trajectories (Deeg, 2007; Liang, Xu, Bennett, Ye, & Quiñones, 2010; Nusselder, Looman, & Mackenbach, 2006). Furthermore, Nusselder, Looman, and Mackenbach (2005)

identified several nondisease factors associated with nine disability trajectories including psychosocial and behavioral factors. This body of research suggests that the type of pathology is only one factor influencing the pace (or speed) of severe disability development. Distinguishing between disability trajectories provides researchers with the opportunity to investigate the role of contextual factors on the pace of severe disability onset.

While prior research has documented multiple disability trajectories among older adults such as permanent mild disability or healthy/nondisabled trajectories (see Nusselder, Looman, & Mackenbach, 2006 or Liang et al., 2010), this research focuses on the pace of severe disability onset among late midlife adults. Severe disability, defined as having difficulty completing three or more Activities of Daily Living (ADLs), represents a loss of independence and a need for greater in/formal care, which can significantly lower an individual's quality of life and put them at a greater risk of social isolation (Fields & Jette, 2007). Drawing from Ferrucci et al.'s (1996) conceptualization of two broad severe disability trajectories (i.e., progressive and catastrophic disability) and more recent empirical research, the purpose of this research is to examine whether race/ethnicity and educational attainment are independent predictors of progressive and accelerated¹ disability onset. By acquiring a greater understanding of the role of race/ethnicity and educational attainment on the pace of severe disability onset, researchers and clinicians will be better equipped to reduce severe disability disparities and possibly create strategies to slow the speed at which severe disability develops for traditionally underprivileged groups with limited access to resources.

Progressive, Catastrophic, and Accelerated Disability

Ferrucci and colleagues' (1996) introduction of the concepts of progressive and catastrophic disability was significant because it capitalized on clinical definitions of disability and helped draw attention to the notion of time-characterization or the pace of severe disability development. They defined progressive disability as the result of the steady "breakdown of the homeostatic equilibrium" (Ferrucci et al., 1996, p. M123). Progressive disability is often typified as occurring in people with older ages and multiple chronic conditions (Ferrucci et al., 1996; Onder, et al., 2005). It is characterized by worsening functional health from degenerative diseases over many years; whereas, catastrophic disability is characterized by sudden disability onset without previous impairment—usually from a catastrophic medical event (Ferrucci et al., 1996; Guralnik, Ferrucci, Balfour, Volpato, & Iorio, 2001; Onder et al., 2005). Research has noted an increased likelihood of negative health events following catastrophic decline. For example, mortality rates are substantially higher (Ferrucci et al., 1996), hospitalizations are more likely (Ferrucci, Guralnik, Pahor, Corti, and Havlik, 1997), and risk of nursing home stays is greater (Ferrucci et al., 1997; Latham, 2011) following catastrophic disability onset.

The extant literature exploring progressive and catastrophic disability is limited; however, other research exploring disability trajectories, more generally, provides insight into the pace of severe disability onset. Liang et al. (2010) classified five trajectories among US adults 50 years or older including: excellent functional health, good functional health with small increasing disability, accelerated increase in disability, high but stable disability, and persistent severe disability; however, after adjusting for time-varying covariates only three

¹Ferrucci et al. (1996) contend that catastrophic disability occurs from a sudden medical event such as a stroke and the decline in function is immediate; however, the data utilized for this project has a two-year follow-up period. Therefore, the term "accelerated disability" is used to describe a quicker onset of severe disability compared to progressive disability. Accelerated disability may represent a precipitous decline from a catastrophic medical event or precipitous decline from a poorly managed degenerative disease.

trajectories were observed: healthy functioning, moderate functional decrement, and large functional decrement. Nusselder, Looman, and Mackenbach (2006) documented nine disability trajectories among Dutch persons 15–74 years old. Of the nine trajectories, three were associated with severe disability: sudden increase in disability, severely disabled with large increase, and permanently severely disabled. A study by Gill et al. (2010) examined disability trajectories in the last year of life. The authors identified five distinct trajectories of disability: no disability, catastrophic disability, accelerated disability, progressive disability, and persistent severe disability. Although Gill et al.'s (2010) research only examined disability trajectories in the last year of life, it highlights the dynamic nature of severe disability onset. Previous research exploring disability trajectories have revealed variations in the pace of disability onset among various age groups and internationally, yet these studies do not concentrate on severe disability specifically. Nevertheless, taken as a whole, the current disability trajectory literature indicates that there are variations in the time-characterization of severe disability onset with at least one trajectory representing an accelerated decline.

This research primarily focuses on progressive and accelerated disability. Because of data limitations, this research does not distinguish between accelerated and catastrophic disability (i.e., abrupt onset from a medical event), but instead views severe disability onset as falling into two broad trajectories representing a gradual decline versus an accelerated decline. Although it is important to understand all disability trajectories, this research concentrates on progressive and accelerated disability because these trajectories allow for a closer examination of the pace of severe disability onset. Excellent functional health (no disability) and persistent severe disability are important outcomes; however, gaining a greater understanding of accelerated or progressive disability onset, may enable researchers to more fully explore the disabling process. Additionally, greater knowledge about predictors of progressive and accelerated disability may allow researchers to develop important interventions, which could prevent or slow the development of severe disability.

Race/Ethnicity, Education, and the Pace of Severe Disability Onset

It is well established that race/ethnicity and education are important risk factors for disability incidence and prevalence; racial minorities and those with low educational attainment are at increased risk of experiencing disability (Crimmins & Saito, 2001; Mendes de Leon et al., 1997; Ferraro, Farmer, & Wybraniec 1997; Guralnik, Land, Blazer, Fillenbaum, & Branch, 1993; Melzer, Izmirlian, Leveille, & Guralnik, 2001; Stuck et al., 1998; Verbrugge, Gates, & Ike 1991). Racial/ethnic and educational disability disparities are generally thought to be due to a variety of factors including social position, lifestyle choices/health behaviors, psychosocial factors, and access to health services (Melzer et al., 2001). In relation to disability trajectories more specifically, there is evidence that low educational attainment and race/ethnicity are related to steeper disability trajectories (Haas & Rohlfen, 2010; Kelley-Moore & Ferraro, 2004; Li, 2005; Liang et al., 2010; Mendes de Leon, Barnes, Bienias, Skarupski, & Evans, 2005; Pérès, Verret, Alioum, & Barberger-Gateau, 2005; Schoeni, Freedman, & Wallace, 2002).

Accordingly, Taylor (2010) investigated socioeconomic status and disability trajectories in later life and documented an educational and income preventative effect for onset as well as a mediating effect of income on progression. Similarly, Haas and Rohlfen (2010) explored racial and ethnic variations in functional health trajectories—both Blacks and Hispanics, compared to Whites, were more likely to have functional limitations at baseline and Black-White differences in the accumulation in functional limitations remained constant over time. Prior research has demonstrated racial/ethnic and educational variations in disability trajectories, where racial and ethnic minorities and those with low educational attainment are

more likely to have disability onset, greater number of (more severe) disability limitations at baseline, and greater accumulation over time; however, many these studies do not distinguish between different trajectories such as progressive or accelerated trajectories. Liang et al. (2010) provide a noteworthy exception, and they observed increased probabilities of experiencing worse functional health trajectories including accelerated decline among Blacks and Hispanics relative to Whites; however, education mediated the association for Hispanics and accelerated disability.

In the past, disability trajectories were thought to be mostly a function of the type of pathology or health condition, yet more recent empirical data illustrate the potential of nondisease or contextual factors to shape disability trajectories (see Nusselder, Looman, & Mackenbach, 2005). Furthermore, two popular disability models recognize the importance of contextual factors in shaping the pace of disability onset. The Disablement Process (Verbrugge & Jette, 1994) uses the concepts of interventions and exacerbators to discuss how contextual factors could potential influence the speed at which an individual becomes disabled. For example, interventions (e.g., medical care, external supports, special equipment, environmental modifications, and positive changes in lifestyle and psychosocial attributes) may reduce the speed at which a person becomes severely disabled, while exacerbators (e.g., the absence of interventions, adoption of behaviors or attitudes that are harmful to health, and societal impediments) may escalate the speed at which a person develops severe disability (Verbrugge & Jette, 1994). Another popular disability model, the International Classification of Functioning, Disability and Health (ICF) (WHO, 2002), also acknowledges the importance of contextual factors on the development of disability. Similar to the Disablement Process' interventions and exacerbators, the ICF uses the concepts of facilitators and barriers to identify possible contextual factors that influence disability development. Facilitators include substances (e.g., medication) or devices (e.g., prostheses) that alter the nature of the impairment as well as "scene-setting" contextual factors like modifying a room or access to personal help, and social norms/legislation (Badley, 2008). On the other hand, barriers may include "lack of necessary substances or devices" (Badley, 2008, p. 2340). Both the Disablement Process and the ICF model emphasize the importance of contextual factors on the development of disability. The concepts of interventions/exacerbators or facilitators/barriers demonstrate how access to resources and high quality healthcare could impact the pace of severe disability onset. Specifically, exacerbators or barriers exemplify how a significant lack of resources may accelerate the pace of severe disability onset.

As outlined in the Disablement Process and the ICF model, severe disability pacing may be influenced by interventions/exacerbators or facilitators/barriers, which are intimately linked with social position and access to resources. Given the prior theoretical and empirical evidence linking education and race/ethnicity to the pace of severe disability onset, it is anticipated that race/ethnicity and educational attainment will be significant predictors for accelerated and progressive disability onset in both the unadjusted and adjusted (i.e., controlling for healthcare access/utilization, health behaviors, and morbidity status) models. This research contributes to extant literature because it explores the time-characterization of severe disability onset in relation to race/ethnicity and education, whereas previous research examining progressive and catastrophic disability onset has largely ignored race/ethnicity and education, and research exploring multiple trajectories have neglected severe disability onset. By exploring onset among a late midlife cohort followed for many years, this research is able to speak to initial severe disability impairment, which contributes to our understanding of the disabling process.

Methods

Data

This research employs data from Waves 2–10 (1994–2010) of the Health and Retirement Study (HRS). The objectives of the HRS are to describe the lives of older US adults emphasizing information about physical and mental health, health insurance, finances and retirement, and family. The HRS is sponsored by the National Institute of Aging (grant number NIA U01AG009740) and the Social Security Administration and is conducted by the University of Michigan (HRS, 2011). The HRS is an ongoing longitudinal survey of a non-institutionalized late midlife (b. 1931–1941) US cohort and their spouses (regardless of the spouse's age). Key groups were over sampled including: African Americans (1.86:1), Hispanics (1.72:1), and Florida residents (2:1). The original baseline (1992) interviews were conducted as structured face to face interviews with two year follow-up telephone surveys. Following the death of a respondent, proxy interviews were completed by the person “most familiar” with the respondent’s finances, health, and family, which was usually the spouse. The initial sample size was 12,654 people (from nearly 7,600 households). To assist in the data management and analysis, the most recent RAND HRS Data file (version L), a simplified and user-friendly data set created from the original HRS data, was utilized (RAND, 2012). Because the HRS has many years of follow-up data and includes an abundance of health information (e.g., multiple functional health measures), it is ideal for examining disability onset.

Measures

For this study, the dependent variable was severe disability onset. A respondent was considered to have severe disability if they had difficulty completing three or more ADLs. This threshold of three or more ADLs has been used in the previous studies (see Ayis, Gooberman-Hill, Bowling & Ebrahim, 2006; Ferrucci et al., 1996; Ferrucci et al., 1997). Three disability categories were formed based on ADLs summary index created for the RAND HRS, which ranged from 0 to 5 and used five standard ADL measures: 1.) difficulty walking across the room; 2.) difficulty bathing/showering; 3.) difficulty dressing; 4.) difficulty eating; and 5.) difficulty getting in/out of bed. The ADLs were all self-reported. Respondents who had “any difficulty” for each task were assigned a value of “1” for that ADLs task. Respondents who had difficulty completing all of the ADLs tasks were assigned the maximum value of the index (5) and those respondents who had no difficulty completing any of the ADLs tasks were assigned the lowest value of the index (0). For each wave, respondents were assigned into the three disability categories: no disability (ADLs summary index=0), mild disability (ADLs summary index=1 or 2), or severe disability (ADLs summary index=3 to 5). Because of concordance issues with ADL measures in Wave 1 (1992), Wave 1 was omitted from the analysis.

Accelerated and progressive disability categories were formed by using information from the previous two waves (four years); this is very similar to the methodology used by Ferrucci and colleagues (1996). If respondents had no disability from the previous two waves and then acquired severe disability in the subsequent wave, then they were classified as having accelerated disability. Respondents who had mild disability for either of the two previous waves and then acquired severe disability in the following wave were classified as having progressive disability. If respondents did not have information for any of the three waves (per interval) (e.g., a respondent had information for Wave 2 and Wave 4, but did not participate in the study in Wave 3), they were treated as missing. Although this technique increased the amount of missing observations, it prevented misclassifying progressive and accelerated disability. In Ferrucci et al. (1996)’s study, the authors used four years of annual data to establish whether or not a respondent had severe disability, and followed the

respondents without severe disability for seven additional years. This research also used four years of data, though biennial, to identify respondents without severe disability; however, this research was able to follow respondents for multiple waves—a potential of six waves (twelve years). Because it is not possible to establish when a respondent developed severe disability during the two-year interval, there was no distinction made between accelerated and catastrophic disability.

Race/ethnicity and educational attainment were the independent variables of interest. A four-category dummy variable was created for race/ethnicity, where White (reference group), Black, other race, and Hispanic were the categories. Educational attainment was based on credential achievement (e.g., receiving a high school diploma) opposed to years of formal schooling. Credential achievement influences type of employment and income more so than years of formal schooling. Three categories were created for educational attainment: 1) less than high school; 2) high school degree or GED (reference group); 3) college (Associate's Degree or higher). Age, sex, household income, and married/partnered status were also included as covariates. Age was measured in years at time of interview. A dichotomous dummy variable, where female=1 was created for sex. Race/ethnicity, education, sex, and age were all treated as time-fixed variables. Household income and married/partnered status were treated as time-varying covariates. Household income was scaled by \$10,000. Married/partnered status was coded as dichotomous dummy variable with married or partnered (i.e., cohabiting) respondents=1.

Healthcare access and utilization, health risk behaviors, and morbidity status were also included in the analyses as time-varying covariates—each measured at the beginning of the interval. Type of health insurance coverage and healthcare utilization was used as a proxy for access to healthcare. A dichotomous measure for health insurance was created: respondents with either individual or spousal private insurance (=1) and those without private insurance (=0). Health insurance coverage was measured as a time-varying variable. Private health insurance was included in the analysis instead of a dichotomous measure of “any” health insurance coverage since private insurance signifies more access to high quality healthcare and interventions/facilitators (see Porell & Miltiades, 2001). Furthermore, many of the respondents qualified for governmental health insurance. Some respondents qualified for Medicare due to age-eligibility during the study, while some respondents qualified for Medicaid or other need-based governmental programs because of financial and health reasons (Wilper et al., 2009); therefore, having any health insurance coverage would be characteristic of being employed (private insurance), older (Medicare), or experiencing financial/health hardship (Medicaid), which denote differing access to high quality healthcare and levels of health status. Both doctor visits (yes=1) and hospitalizations (yes=1) were self-reported and were measured at the beginning of the interval about the past two years (interval length).

There were three measures of health risk behaviors: 1) physical activity; 2) smoking status; and 3) body mass index (BMI). Physical activity was measured as a dichotomous variable, where participating in vigorous exercise/sports three or more times per week=1. Smoking status was measured as a trichotomous variable comprised of never smoked (reference group), former smoker, and current smoker. A four-category dummy variable was created for Body Mass Index (BMI), where (below 18.5), healthy weight (normal) (18.5–24.9), overweight (25–29.9), and obese (above 30) were the categories. It is important to note that unlike physical activity and smoking, BMI is not a behavior, but reflects a set of health behaviors including diet and physical activity. Health risk behaviors may change substantially over time; therefore, the health risk behaviors were treated as time-varying measures. Morbidity status was measured using chronic conditions; the measures were based on self-reports of physician diagnosis. The chronic conditions were: high blood

pressure, diabetes, cancer, lung disease, heart problems, stroke, arthritis, and psychological problems (i.e., emotional, nervous, or psychiatric problems). Morbidity status was measured as a time-varying.

Analytic Strategy

The sample was restricted to age eligible HRS respondents (born 1931–1941). Discrete-time Cox-proportional hazard models were estimated using multinomial logistic regression with multiple competing events (see Allison, 1984). Because two waves of data were used to establish a pattern of severe disability and a third wave was used to establish onset of severe disability, there were a total of six intervals created from nine waves of data. To demonstrate, an individual who did not report any impairment in Wave 2 or Wave 3, but reported severe impairment (3 or more ADLs) in Wave 4 was classified as having experienced accelerated decline. The risk group at the beginning of each interval was respondents without severe disability, and the possible outcomes included: no severe disability, progressive disability onset, accelerated disability onset, died, and attrited (i.e., respondents lost to follow-up). The models included progressive disability onset vs. no severe disability, accelerated disability onset vs. no severe disability, and died vs. no severe disability onset. Attrited vs. no severe disability was also modeled, but is not presented (results available from author upon request). Cox-proportional hazard ratios were then ascertained for each variable. Additionally, to account for complex stratified sampling, the models were weighted by individual-level sampling weights and strata using PROC SURVEYLOGISTIC procedure and WEIGHT and STRATA statements in *Statistical Analysis Software* (SAS). Robust standard errors were used to adjust for within person clustering.

Sample Characteristics

Table 1 presents the risk group, respondents without accelerated or progressive disability at Wave 4 (N=8,078), sample characteristics. The distributional information was weighted using individual-level sampling weights. The risk group sample was 83.5% White, 9.8% Black, 1.8% other race, and 4.9% Hispanic, and the majority (56.1%) of the sample had a high school degree or GED followed by 23.7% having more than a high school degree (i.e., Associate's Degree or higher), and 20.2% having less than a high school degree (or equivalent). Women represented 53.6% of the risk group, and the average income was \$62,000. Most (75.3%) of the respondents were married or partnered at Wave 4. The average age of a respondent was 61.2 years. More than half (63.8%) of the respondents had private health insurance at Wave 4. The vast majority (93.2%) of respondents had visited a doctor in the past two years, while a little less than 20% of respondents had experienced a hospitalization. Among the health risk behaviors, a little more than half of the sample was active and 18.8% of the sample was current smokers at Wave 4. Being overweight (40.7%) was the modal category followed by 31.2% of the sample being healthy weight, 26.0% being obese, and only 3.4% being underweight. The most prevalent chronic condition was arthritis (47.3%), followed by high blood pressure (40.1%), heart disease (14.4%), diabetes (11.7%), psychological problems (8.6%), cancer (8.1%), lung disease (6.6%), and stroke (3.5%).

Results

Table 2 displays a summary of the findings acquired from multinomial logistic regression analysis for: progressive disability, accelerated, died versus no severe disability (i.e., respondents who did not develop progressive or accelerated disability) by race/ethnicity, education, and age. Model 1 presents the age-adjust hazard ratios for race and ethnicity, while Model 2 presents the age-adjusted hazard ratios for education. Model 3 introduced

both race/ethnicity and education. The Likelihood Ratio and intercept for all models were statistically significant at an alpha level of less than 0.001.

Progressive Disability vs. No Severe Disability

In Model 1, race/ethnicity (i.e., Black and Hispanic) had a significant association with developing progressive disability. Black respondents (hazard ratio=1.86) and Hispanic respondents (hazard ratio=1.89) were more likely to develop progressive disability compared to White respondents, net of age. Similarly, in Model 2, respondents with less than high school education (hazard ratio=1.76) were more likely to develop progressive disability, while having more than high school education (hazard ratio=0.69) was associated with a lower likelihood of progressive decline compared to respondents with a high school or equivalent education. Model 3 introduced both race/ethnicity and education. Black (hazard ratio=1.60) and Hispanic (hazard ratio=1.48) respondents remained more at risk of progressive disability onset relative to White respondents even after controlling for educational attainment; however, the associations were attenuated. Educational attainment also persisted in Model 3. Having low educational attainment continued to be linked to a greater likelihood of progressive disability, while high educational attainment was linked to a lower likelihood.

Accelerated Disability vs. No Severe Disability

The age-adjusted hazard ratios for Black and Hispanic respondents were 1.93 and 1.92, respectively. Compared to Whites, Blacks and Hispanics were almost two times more likely to experience accelerated decline, net of age. In Model 2, respondents with less than a high school education were substantially more at risk of accelerated disability onset with a hazard ratio of 2.92. With the introduction of education (Model 3), race and ethnicity were no longer associated (mediated) with accelerated disability onset. Respondents with less than high school education (hazard ratio=2.72) continued to be considerably more at risk of experiencing accelerated disability. Age was associated with accelerated disability in all three models with older ages being associated with greater risk of accelerated decline.

Died vs. No Severe Disability

In the first model, being black (hazard ratio=1.56) was associated with increased risk of death, net of age, whereas, in Model 2, having a less than high school education (hazard ratio=1.85) was associated with increased likelihood of death. In Model 3, Black (hazard ratio=1.33) respondents remained more at risk of death; however, the strength of the relationship was attenuated with the inclusion of education. From Model 2 to Model 3, having a less than high school education remained relatively stable with a hazard ratio of 1.84. In all three models, being older was associated with greater risk of death during the study.

Table 3 displays a summary of the findings acquired from multinomial logistic regression analysis for: progressive disability, accelerated, died versus no severe disability (i.e., respondents who did not develop progressive or accelerated disability) by race/ethnicity, education, socio-demographic characteristics, health risk behaviors, healthcare access & utilization, and morbidity status. Model 1 included all socio-demographic characteristics, while Model 2 introduced health risk behaviors. Model 3 represented the fully adjusted model and introduced morbidity status. The Likelihood Ratio and intercept for all models were statistically significant at an alpha level of less than 0.001.

Progressive Disability vs. No Severe Disability

In Model 1, race/ethnicity (i.e., Black and Hispanic) and education (i.e., less than high school and college) had a significant association with developing progressive disability, net of other socio-demographic characteristics. Black respondents (hazard ratio=1.42) and Hispanic respondents (hazard ratio=1.32) were more likely to develop progressive disability relative to White respondents. Respondents with less than high school education (hazard ratio=1.45) were more likely to develop progressive disability compared to respondents with a high school or equivalent education. Having a higher household income (hazard ratio=0.93) had a protective effect on developing progressive disability. In Model 2, health risk behaviors were introduced. Black and Hispanic respondents remained at a greater risk of progressive disability; however, the association for Black respondents (hazard ratio=1.31) was slightly attenuated, while the association for Hispanic respondents (hazard ratio=1.30) remained relatively stable across Model 1 and Model 2. Less than high school education (hazard ratio=1.38) continued associated with progressive disability; however, the strength of the association was also slightly attenuated with the introduction of health risk factors. Income (hazard ratio=0.94) continued to have a protective effect on progressive disability onset. Three health risk behavior measures were associated with progressive disability; physical activity (hazard ratio=0.52) had a protective effect, while being a current smoker (hazard ratio=1.41) or obese (hazard ratio=2.01) were associated with greater likelihood of progressive disability onset.

Model 3 included healthcare access and utilization measures and morbidity status. After adjusting for these measures, being Black (hazard ratio=1.49) or Hispanic (hazard ratio=1.63) was associated with a greater risk of progressive disability onset—the magnitude of these associations increased from Model 2, suggesting a potential suppressor effect. Although the relationship for less than high school education (hazard ratio=1.19) and progressive disability persisted in Model 3, much of the association was mediated with the inclusion of healthcare access/utilization and morbidity status. In the fully adjusted model, higher income and being physically active remained linked to a lower likelihood of progressive disability onset, while being a current smoker or obese remained linked to a greater likelihood. Private insurance and hospitalizations were significantly associated with progressive disability onset, where having private insurance (hazard ratio=0.75) was linked to decreased risk of onset and hospitalizations (hazard ratio=1.45) were linked to increased risk of onset. All of chronic conditions with the exception of high blood pressure and heart problems were associated with higher likelihood of progressive disability onset with arthritis (hazard ratio= 2.76), stroke (hazard ratio= 2.15), and psychological problems (hazard ratio= 2.10) having the most robust associations.

Accelerated Disability vs. No Severe Disability

Model 1 included socio-demographic characteristics, and having less than high school education (hazard ratio=2.48) was strongly associated with increased likelihood of accelerated disability onset. Respondents without a high school degree were nearly two and a half times more likely to experience an accelerated decline. Age was also significantly linked with accelerated disability with older respondents (hazard ratio=1.06) being at a greater risk of onset. After adjusting for health risk behaviors (Model 2), low educational attainment remained highly significant. Similarly, being older continued to be associated with accelerated disability onset. Education (i.e., less than high school) and age remained relatively stable across the models with the inclusion of health risk behaviors. Among health risk behaviors, only physical activity was associated with accelerated disability onset. Physically active respondents were less likely to experience accelerated disability.

In Model 3, healthcare and morbidity measures were introduced. Even after controlling for various health measures, respondents without a high school degree were still two times more likely to experience accelerated disability onset; however, the strength of this association was somewhat weakened. Age was no longer significant in Model 3. Physical activity continued to have a protective effect on accelerated disability onset. Of the healthcare access and utilization measures, only private health insurance (hazard ratio=0.66) was significantly associated with accelerated disability. Having private health insurance was linked to lower risk of accelerated disability onset. Five of the eight chronic conditions (i.e., arthritis, cancer, diabetes, psychological problems, and stroke) were associated with greater risk of accelerated disability with stroke (hazard ratio=2.56) and psychological problems (hazard ratio=2.17) being particularly strong predictors.

Died vs. No Severe Disability

In the first model, having a less than high school education (hazard ratio=1.66) and being older (hazard ratio=1.07) were all associated with increased likelihood of death. Hispanic respondents (hazard ratio=0.68) and female respondents (hazard ratio=0.55) were less likely to experience death during the study. Additionally, higher household incomes (hazard ratio=0.96) and being married/partnered (hazard ratio=0.64) were associated with a lower risk of death. In Model 2, after adjusting for health risk behaviors, Black respondents (hazard ratio=1.22) were more likely to experience death. Being Hispanic was no longer significantly associated with death. Older respondents (hazard ratio=1.07) continued to have an increased risk of death. Similarly, being female, having a higher household income, and being married/partnered continued to have a protective effect on death. All of the health risk behaviors were significant predictors of death. Physically active (hazard ratio=0.40), overweight (hazard ratio=0.72), and obese (hazard ratio=0.74) respondents were less likely to experience death during the study, where former smokers (hazard ratio=1.74), current smokers (hazard ratio=2.64), and underweight (hazard ratio=2.88) were more at risk of death.

In the final model (Model 3), Black respondents and respondents without a high school degree remained more at risk of death, net of healthcare access and utilization and morbidity status. With the inclusion of the health care and morbidity measures, the association for Black respondents strengthened, while the association for low education attainment attenuated. The relationships for the other socio-demographic characteristics (i.e., age, sex, income, married/partnered) persisted in Model 3. Likewise, all of the health risk behaviors continued to be associated with death. Among the healthcare access and utilization measures, hospitalizations (hazard ratio=2.47) were strongly associated with increased risk of death, while doctor visits were associated with decreased risk of death. With the exception of arthritis, all of the chronic conditions were associated with greater likelihood of death; cancer (hazard ratio=2.93), lung disease (hazard ratio=2.10), diabetes (hazard ratio=1.95), and stroke (hazard ratio=1.91) had the most robust associations.

Discussion and Conclusions

The results of this study suggest that race/ethnicity (i.e., Black and Hispanic) and education (i.e., less than high school) are both important predictors of progressive disability onset, while education (i.e., less than high school) is a very important predictor of accelerated disability onset. It is important to note that race/ethnicity were significant predictors of accelerated disability onset in the model that only adjusted for age; however, education mediated those associations. Even after controlling for key contextual factors like other socio-demographic characteristics, healthcare access and utilization, health risk behaviors, and morbidity status, race/ethnic and educational disparities persisted in the full models. The strength of the education association and lack of race/ethnic associations in relation to

accelerated disability are particularly noteworthy findings. Previous research has suggested that racial and ethnic minorities experience steeper disability trajectories compared to Whites (Kelly-Moore & Ferraro, 2004; Li, 2005); however, this research suggests that among individuals who developed severe disability onset, low educational attainment is a more salient factor of accelerated decline.

Racial and ethnic minorities may have an *overall* steeper disability trajectory, but when progressive and accelerated disability are distinguished, educational attainment is a more powerful predictor of accelerated disability onset. This finding is somewhat similar to Liang et al. (2010), where Hispanics were no longer at risk of accelerated disability when education was introduced. After controlling for numerous covariates, respondents with a less than high school education were more than two times as likely to experience an accelerated decline. A study conducted by Herd, Goesling, and House (2007) demonstrates the importance of education for onset of health problems including functional limitations and chronic conditions; educational attainment occurs earlier in the life course and denotes varying psychosocial resources that are powerful determinants of health status. Although racial and ethnic disparities exist in regard to managing chronic conditions and disability (Ferraro & Farmer, 1996, 2005; Geronimus, Hicken, Keene, & Bound, 2006; Ndao-Brumblay & Green, 2005), educational attainment, reflecting variation in exposure to life chances over the life course (see Ferraro & Shippee, 2009; Wadsworth, 1997), may drive the acceleration of the disabling process. Alternatively, educational attainment may be more predictive of the types of underlying conditions leading to accelerated disability. Progressive disability onset represents a more gradual decline compared to accelerated disability onset, yet both outcomes are indicative of severe disability. A greater likelihood of progressive disability versus accelerated disability, net of educational attainment, among racial and ethnic minorities (i.e., Blacks and Hispanics) may reflect racial and ethnic variations in the disabling process (see Zsembik, Peek, & Peek, 2000). It is possible that the mechanisms leading to progressive disability versus accelerated disability stem from differences in the stages of the disabling process (e.g., health condition, physical functional limitation, or cognitive function limitation).

A major limitation of this study is the two-year interval; it cannot be determined at what point during the two-year interval respondents developed severe disability. Furthermore, it is possible that respondents experienced severe disability, but recovered during the interval period. Annual data would have led to more precise estimates of accelerated and progressive disability; however, utilizing the HRS data was advantageous for a number of reasons including many waves of data. Another potential limitation of this research is attrition due to lost to follow-up. Attrition was modeled as a competing event, and it appears that attrition was more likely among those with poor health and limited resources, which implies that these findings may underestimate accelerated and progressive disability onset. The inclusion of time-varying covariates in the models also had many advantages, yet the use of time-varying covariates also created potential limitations. For example, time-varying covariates are more difficult to interpret than time-fixed covariates, and they are more susceptible to reverse causation. Due to data confines, other important confounders such as cognition were not included in the analyses. An additional limitation stems from not being able to control for the underlying condition(s) that led to disability onset. Individual measures of chronic conditions are included in the models, but it is not possible to identify the original health condition or combination of comorbidities that led to developing accelerated or progressive disability. Finally, the comparison group, respondents without severe disability, is heterogeneous. Within the same comparison group, a respondent may have no disability or mild disability. Despite these limitations, this research provides evidence that pace of severe disability onset varies by race/ethnicity and education.

Understanding the pace of severe disability onset in relation to racial/ethnic and educational disparities contributes to the extant literature in numerous ways. The pace of severe disability onset or trajectory has been shown to influence mortality, hospitalization and institutionalization rates (Ferrucci et al., 1996; Ferrucci et al., 1997; Latham, 2011). This study suggests that traditionally disadvantaged groups like racial/ethnic minorities and those with low educational attainment are more likely to experience severe disability onset (whether progressive or accelerated), but it appears that low educational attainment places them at an even greater risk of accelerated decline. Given the lack of resources among those without a high school education (or more advanced degrees) over the life course, it is expected to observe an accelerated pace of severe disability development. Accordingly, there is evidence that healthcare access and utilization and health behaviors have potential to reduce severe disability onset. Both private health insurance and physical activity significantly reduced the risk of progressive and accelerated disability onset, while current smokers and obese individuals were more likely to experience progressive disability. Interventions with an emphasis on access to high quality healthcare and health promotion throughout the life course may prevent or slow the onset of severe disability. According to the Disablement Process and the ICF model, access to high quality healthcare and health behaviors are essential contextual factors that shape the pace of disability onset through interventions/exacerbators or facilitators/barriers. In the future, the author suggests prospective studies exploring disability trajectories using smaller interval periods aimed at understanding the role of contextual factors. Additionally, the author recommends research exploring specific interventions targeted at at-risk individuals (i.e., individuals with low educational attainment) and communities that attempt to prevent or slow the onset of severe disability.

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Table 1

Descriptive Statistics of Risk Group (N=8,078)

	Distribution Information ^{a,b}
Socio-demographic Characteristics	
Race/Ethnicity:	
White	83.5%
Black	9.8%
Other Race	1.8%
Hispanic	4.9%
Educational Attainment:	
Less than High School	20.2%
High School Degree or Equivalent	56.1%
More than High School	23.7%
Age (years)	61.2 (2.8)
Sex (female=1)	53.6%
Income (Scaled \$10,000)	6.2 (9.0)
Married or Partnered (yes=1)	75.3%
Health Risk Behaviors:	
Physically Active (yes=1)	50.2%
Smoking Status:	
Never Smoked	37.7%
Former Smoker	43.6%
Current Smoker	18.8%
Body Mass Index (BMI):	
Underweight	2.1%
Healthy Weight	31.2%
Overweight	40.7%
Obese	26.0%
Healthcare Access & Utilization:	
Private Health Insurance (yes=1)	63.8%
Visit Doctor (past 2 years) (yes=1)	92.0%
Hospitalization (past 2 years) (yes=1)	19.3%
Morbidity Status:	
Chronic Conditions (yes=1):	
Arthritis	47.3%
Cancer	8.1%
Diabetes	11.7%
Heart Problems	14.4%
High Blood Pressure	40.1%
Lung Disease	6.6%
Psychological Problems	8.6%
Stroke	3.5%

Source: RAND Health and Retirement Study (HRS) Data (1998)

Notes:

^aPercentage distributions are shown for categorical variables; means and (standard deviations) are shown for continuous variables;

^bDistribution information is weighted.

Table 2

Cox Proportional Hazard Ratios of Progressive and Accelerated Disability Onset and Death, by Race/Ethnicity, Education, and Age (Risk Group: N=8,078)^{a,b}

	Developed Progressive Disability ^c			Developed Accelerated Disability ^c			Died ^c		
	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3
Race/Ethnicity:									
White (ref.)									
Black	1.86***		1.60***	1.93***		1.49	1.56***		1.33**
Other Race	1.06		1.10	2.21		2.13	0.69		0.68
Hispanic	1.89***		1.48***	1.92*		1.25	0.97		0.74
Education:									
Less than High School		1.76***	1.60***		2.92***	2.72***		1.85***	1.84***
High School/GED (ref)									
More than High School		0.69***	0.70***		1.03	1.02		0.91	0.92
Age	1.02	1.01	1.01	1.08***	1.07**	1.07**	1.10***	1.08***	1.08***
Observational Intervals:									
Interval 1 (ref.)									
Interval 2	0.78*	0.78*	0.78*	0.64	0.65	0.65	1.15	1.16	1.16
Interval 3	0.67***	0.67***	0.68***	0.96	0.99	0.99	0.74*	0.75*	0.75*
Interval 4	0.56***	0.56***	0.57***	0.77	0.79	0.79	1.03	1.05	1.05
Interval 5	0.62***	0.63***	0.64***	1.15	1.18	1.19	1.27	1.30*	1.31*
Interval 6	0.65***	0.66***	0.67***	2.33***	2.43***	2.46***	2.04***	2.10***	2.11***
Intercept	-3.97***	-3.53***	-3.67***	-10.17***	-9.80***	-9.95***	-8.57***	-8.43***	-8.38***
Likelihood Ratio	324.00***	406.68***	458.51***	324.00***	406.68***	458.51***	324.00***	406.68***	458.51***
Degrees of Freedom	9	8	11	9	8	11	9	8	11

Source: RAND Health and Retirement Study (HRS) Data (1994–2010)

Notes:

^aRisk group = no severe disability;

^b N=24,444.25 weighted person-intervals;

^c Compared to persons who did not develop progressive or accelerated disability;

* 0.05 p < 0.01;

** 0.01 p < 0.001;

*** p 0.001

Table 3

Cox Proportional Hazard Ratios of Progressive and Accelerated Disability Onset and Death, by Race/Ethnicity, Education, Socio-demographic Characteristics, Health Risk Behaviors, and Morbidity Status (Risk Group: N=8,078)^{a,b}

	Developed Progressive Disability ^c			Developed Accelerated Disability ^c			Died ^c	
	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3	Model 1	Model 2
Race/Ethnicity:								
White (ref.)								
Black	1.42***	1.31***	1.49***	1.26	1.23	1.26	1.17	1.22*
Other Race	1.04	1.10	1.25	2.03	1.95	2.13	0.67	0.63
Hispanic	1.32*	1.30*	1.63***	1.12	1.05	1.04	0.68*	0.75
Education:								
Less than High School	1.45***	1.38***	1.19*	2.48***	2.42***	2.10***	1.66***	1.52***
High School/GED (ref)								
More than High School	0.87	0.93	0.99	1.21	1.24	1.34	0.97	1.05
Age	1.00	1.01	0.99	1.06*	1.06*	1.04	1.07***	1.07***
Sex (female=1)	1.03	1.03	0.88	0.86	0.81	0.77	0.50***	0.48***
Income (Scaled \$10,000)	0.95***	0.94***	0.95***	0.94	0.95	0.96	0.96***	0.97**
Married/Partnered (yes=1)	0.95	0.99	1.05	0.71	0.73	0.77	0.64***	0.73***
Health Risk Behaviors:								
Physical Activity		0.52***	0.61***		0.50***	0.57**		0.40***
Smoking Status:								
Never Smoked (ref.)								
Former Smoker		1.13	0.95		1.01	0.90		1.74***
Current Smoker		1.41***	1.24*		0.92	0.88		2.64***
Body Mass Index (BMI):								
Underweight		1.22	1.05		1.32	1.14		2.88***
Healthy Weight (ref.)								
Overweight		1.10	1.03		0.87	0.81		0.72***
Obese		2.01***	1.65***		1.01	0.81		0.74***

	Developed Progressive Disability ^c			Developed Accelerated Disability ^c			Died ^c		
	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3
Healthcare Access & Utilization:									
Private Insurance			0.75***			0.66*			0.94
Doctor Visits (past 2 years)			1.21			0.60			0.52***
Hospitalizations (past 2 years)			1.45***			1.27			2.47***
Morbidity Status:									
Chronic Conditions:									
Arthritis			2.76***			1.48*			1.05
Cancer			1.26**			1.64*			2.93***
Diabetes			1.24**			1.68**			1.95***
Heart Problems			1.14			1.25			1.41***
High Blood Pressure			1.03			1.16			1.33***
Lung Disease			1.64***			0.87			2.10***
Psychological Problems			2.10***			2.17***			1.50***
Stroke			2.15***			2.56***			1.91***
Observational Intervals:									
Interval 1 (ref.)									
Interval 2	0.79**	0.79**	0.77***	0.65	0.65	0.61	1.17	1.12	1.14
Interval 3	0.69**	0.68***	0.60***	0.99	0.99	0.85	0.74*	0.77	0.60**
Interval 4	0.57***	0.50***	0.43***	0.79	0.68	0.55*	1.05	0.92	0.75*
Interval 5	0.64***	0.56***	0.44***	1.19	1.01	0.75	1.30*	1.14	0.87
Interval 6	0.68**	0.59**	0.43***	2.44***	2.05**	1.44	2.09***	1.88***	1.28
Intercept	-2.54***	-3.25***	-2.96***	-8.51***	-8.09***	-6.82***	-6.83***	-7.19***	-6.29***
Likelihood Ratio	666.81***	1120.07***	2127.14***	666.81***	1120.07***	2127.14***	666.81***	1120.07***	2127.14***
Degrees of Freedom	14	20	31	14	20	31	14	20	31

Source: RAND Health and Retirement Study (HRS) Data (1994–2010)

Notes:

^aRisk group = no severe disability;
^bN=24,444.25 weighted person-intervals;
^cCompared to persons who did not develop progressive or accelerated disability;
^{*} 0.05 p < 0.01;
^{**} 0.01 p < 0.001;
^{***} p 0.001