

Capturing Transitions and Trajectories: The Role of Socioeconomic Status in Later Life Disability

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Objectives. Disability is conceptualized as a life course process and measured using either transitions or trajectories. Previous research does not simultaneously explore both aspects of disablement, accounting for timing and trajectory. The role of education is noted in disability research, but its independent effects over time have not been fully examined. I investigate the effects of education and income on disability onset and progression over a decade.

Methods. I use a latent curve modeling approach with four waves of the Duke Established Populations for Epidemiologic Studies of the Elderly data to independently test the preventive and mediating effects education and income provide for disability.

Results. Education has a preventive effect for disability onset but no significant effect on progression once income is held constant. Income has both a preventive and mediating effect on disability, although preventive effects are stronger for education.

Discussion. Later life disability is measured here using both onset and trajectory. Findings are consistent with health research suggesting that education and income work through shared and independent mechanisms to affect disability over time. These findings also highlight the importance of modeling timing when studying health trajectories.

Key Words: Disability—Education—Socioeconomic status—Stratification and health.

CAN transitions and trajectories be simultaneously examined to better capture the life course process of disability in later life? If so, what is the added benefit in testing the effects of known predictors over time? Do multiple components of socioeconomic status (education and financial resources) differ in their effects on disability onset and accumulation in later life? Disability is primarily conceptualized using a life course framework (Verbrugge & Jette, 1994) with an emphasis on individual transitions and trajectories. Timing is a key component of life course transitions and active life expectancy (Crimmins, Saito, & Ingegneri, 1997). Transitions are embedded in trajectories, giving shape and meaning to the trajectory as a whole. The study of trajectories in life course research is primary in investigating the management of demands and resources over time (Halfon & Hochstein, 2002).

Although transitions and trajectories are interdependent facets of life course processes, they are rarely studied simultaneously. The intersection of life course and health research suggests that key predictors of health work differently on transitions and trajectories (Kuh, Ben-Shlomo, Lynch, Hallqvist, & Power, 2003). Measurement of disability has focused increasingly on its dynamic nature, and analytic strategies capture either transitions or, more recently, individual level trajectories (see Manton, 1988; Taylor & Lynch, 2004). However, no study to the knowledge of the author has addressed both components simultaneously in measuring socioeconomic predictors over time.

Given the observed declines in disability prevalence among older adults in the United States (Freedman, Schoeni, Martin, & Cornman, 2007), researchers have increasingly focused on underlying factors, and particularly education (Freedman & Martin, 1999). The connection of individual-level socioeconomic status and health is central to social stratification and health research across the life course (House, Lantz, & Herd, 2005), particularly the disentangling of education and income effects on the onset and progression of poor health. Research suggests that education works to prevent or delay poor health, whereas income has both a preventive role and a “muting” effect on severity once poor health has occurred. However, the findings for disability outcomes in later life over time are inconsistent (see Grundy & Glaser, 2000; Maddox & Clark, 1992).

In this article, I bridge theories of the disablement process and the social stratification of health within the life course framework to disentangle the effects of socioeconomic status on disability in later life. I test these effects on the first transition (onset) and subsequent trajectory (progression) of disability over time, conceptualizing and measuring disability as a life course process. The analyses presented in this study serve to disentangle these effects as the first step in understanding how socioeconomic status works in the causal pathways leading to differential trajectories of disability in later life with regard to transitions and trajectories. In addition, they serve as an example of a novel approach to trajectory analyses where timing of transition is tested separately from progression of poor health, therefore outlining a

more nuanced analysis of onset, severity, and recovery of other health outcomes over time in future research.

THEORETICAL FRAMEWORK AND PREVIOUS LITERATURE

Verbrugge and Jette (1994) define disablement as “impacts that chronic and acute conditions have on the functioning of specific body systems and on people’s abilities to act in necessary, usual, expected, and personally desired ways in their society” (p. 3). This model provides a theoretical view of disability as a process and social phenomenon, predisposed by individual risk factors and mediated by extra-individual factors and intra-individual characteristics. The life course perspective aids in conceptualizing and measuring disability as a dynamic process, defined by short-term transitions nested in long-term trajectories (Elder, 2000). In the study of transitions, the life course perspective highlights temporal order and sequencing of events. The collection of transition states over time creates a trajectory that is meaningful as a whole and linked to other trajectories.

One of the most salient aspects of studying trajectories is a focus on the management of demands and resources after key transitions. The study of transitions within trajectories has been central to research on life course dynamics for over two decades, but the focus on both tenants of life course processes has not been fully realized in disability research. Although researchers studying health transitions in later life have contributed to the life course literature for some time, actual measurement of individual-level trajectories in this field of study is still somewhat rare (George, 2003). A wealth of literature has emerged on transition into disability, and a growing number of studies investigate disability trajectories. In addition, there is evidence to suggest that failure to capture delayed onset in disability trajectories leads to biased estimates of trajectory slope coefficients (Taylor, 2005, 2008).

Research on Disability Transitions and Trajectories.—Research has increasingly focused on heterogeneity among disabled elderly and the dynamic nature of the disablement process (see Branch, Katz, Knipmann, & Papsidero, 1984; Crimmins & Saito, 1993). Transitions in impairment status have been predicted by age, gender, race, previous functional state, income, and education (Beckett et al., 1996; Maddox, Clark, & Steinhauer, 1994; Schoeni, Freedman, & Wallace, 2002). Other work has focused on demographic and social predictors of recovery (Mendes de Leon et al., 1999). Previous literature has also measured disability as a trajectory, extending the findings of transition studies, emphasizing heterogeneity across individuals and differential risk given demographic and social predictors (see Li, Duncan, McAuley, Harmer, & Smolkowski, 2000; Maddox & Clark, 1992; Taylor & Lynch, 2004; Verbrugge,

Reoma, & Gruber-Baldini, 1994). Bridging the separate findings of previous research on transitions and trajectories in disability, I explore disability as a life course process encompassing both facets (transition and trajectory).

Socioeconomic Effects on Onset and Progression of Morbidity and Disability.—I draw on the conceptual framework of House (2001) for integrating the effects of socioeconomic status on health into a broader model of social inequalities and psychosocial risk factors. According to this framework, socioeconomic status is the product of other ascribed statuses such as gender and race in addition to macrosocioeconomic conditions in early life, working through a number of life course mechanisms to affect health throughout adulthood (see also Alwin & Wray, 2005). The distinction between education and financial resources is highlighted in this research because these components of socioeconomic status likely differ in their effects on health over the life course. The examination of timing (onset) and progression of poor health is also primary because it addresses two important topics for research, policy, and practice:

1. Do education and income solely predict a delay or compression of morbidity (Fries, 1980) in the life course of individuals or do they also serve a muting effect such that the progression of disease or disability is decreased once onset occurs? Understanding which facets of socioeconomic status predict the onset and progression of poor health is vital for policy as well as projections of health among older adults. There is some support for the differential effects of education and income on the onset and progression of disability (Grundy & Glaser, 2000; Zimmer & House, 2003), but trajectory studies show no difference due to the lack of focus on transitions (see Maddox & Clark, 1992). Also common in disability research is the use of education as a sole proxy of socioeconomic status (Melzer, Izmirlian, Leveille, & Guralnik, 2001; Taylor & Lynch, 2004; Waidmann & Liu, 2000) because it is a robust indicator of socioeconomic status that is assumed to be time invariant in later life. If the effects of education and financial resources differ over time, they must be examined independently in future research.

2. Do the causal mechanisms through which education and income affect health differ? If the effects of education and income on the timing and progression of disability differ, it is likely that they have both shared and independent mediators through which they affect health. Researchers focusing on education often note that it is the “root component of social status” yielding accumulated social inequalities (Mirowsky, Ross, & Reynolds, 2000: p. 50). Important mediators of education include work and economic conditions, social-psychological resources, health behaviors/lifestyle, and personal mastery (Mirowsky & Ross, 2003; Ross & Wu, 1995). In sum, the protective effect of education

on health includes resources provided by occupation and income (including health insurance, assets, housing, etc.), but also independently promoting healthy life behaviors and giving individuals a sense of control over life.

Other research emphasizes financial resources and the material components of socioeconomic status (see Lynch & Kaplan, 2000). Income inequality gives rise to the “differential accumulation of exposures and experiences that have their sources in the material world” (Lynch, Davey Smith, Kaplan, & House, 2000: p. 1202). Financial resources therefore not only serve to protect individuals through a decrease in the occurrence of life stressors (delayed onset), but also serve to mute the effects of negative health outcomes through access to multiple forms of capital (assets, medical care, etc.).

The present study seeks to address the first question presented previously because it is only in understanding how components of socioeconomic status work over time for health that we can disentangle the causal pathways through which multiple facets of socioeconomic position affects health. The analysis of this topic among older adults in the United States has been fragmented (transitions or trajectories are studied but not simultaneously) and findings are inconsistent in previous studies that vary by age, gender, race/ethnicity, sample, and methodological approach (Grundy & Glaser, 2000; Maddox & Clark, 1992; Taylor, 2008; Verbrugge et al., 1994; Zimmer & House, 2003). Furthermore, the systematic analysis of education and income in the disablement process over time serves to elucidate the processes by which socioeconomic status is connected with declining disability trends in this country (Freedman & Martin, 1999).

Based on previous literature, I hypothesize that disability may be measured as a trajectory over time in later life. However, I hypothesize that modeling onset and progression in severity as separate processes will lead to more consistent results with regard to timing and progression. Among demographic covariates, men will be both more likely to delay disability and to have lower levels of impairment compared with women (among those who are disabled; Schoeni, Freedman, & Wallace, 2002). White individuals are also expected to delay disability and have lower levels of severity compared with nonwhites (Manton & Gu, 2001; Manton, Patrick, & Johnson, 1987), although the observed mortality crossover by race may weaken these effects over time (see Corti et al., 1999).

Following the previously discussed arguments on the differential protective effects of education and financial resources, I hypothesize that education will work mainly to delay disability (prevention) and that financial resources will work both to delay disability and to decrease the progression of disability among those disabled. In addition, I hypothesize that the results will vary by cohort (Manton, Gu, & Lowrimore, 2008), with younger cohorts more likely to delay and have lower levels over time due to the decreasing

trends in disability in the United States. Gender and race differences are expected to decrease with older cohorts due to selective mortality of men and nonwhite individuals. It is also likely that socioeconomic effects will be greater in younger cohorts due to a stronger return from education (Lynch, 2003) and the possibility that as individuals age into very late life they may spend down assets.

METHODS

Study Sample and Variables

The data source used to test these hypotheses is the Established Populations for Epidemiologic Studies of the Elderly (EPESE) at Duke University. The EPESE is a multisite, National Institute on Aging sponsored project aimed at investigating predictors of mortality, chronic disease, disability, and service utilization. The Duke sample is representative of those individuals aged 65 and older residing in the community in a five-county area of central North Carolina at baseline. Respondents participated in four in-person and four telephone-based interviews. Only in-person interviews are used, including proxy responses. The baseline survey was conducted in 1986–1987, with follow-up in-person data collected in 1989, 1992, and 1996. The original sample consisted of 4,162 individuals, and of these, 4,016 had some report (nonmissing) of disability status at baseline in 1986–1987. This loss of 146 cases comprised 3.5% of the total sample. Another 61 cases were dropped for missingness on key predictor variables leaving a sample size of 3,955. Attrition during the observation period was modeled using a full information maximum likelihood estimator that calculates the likelihood at each time point among those individuals contributing to that time point. Therefore, individuals were allowed to contribute to the onset and trajectory portions of the model until they dropped out. Attrition over the decade was due primarily to mortality (Table 1). Those who died over the observation period were more likely to be men, nonwhite, older, and have less resources in terms of education and income at each wave. Sensitivity analyses (not shown) suggest estimates of disability were not substantially different when comparing models with attriters at each wave compared with those remaining in the study. I include age (years), gender (*men* = 1), and race (*white* = 1, *nonwhite* = 0) as demographic controls. In addition, I split the sample into cohorts (65–74, 75–84, 85 years and older) in order to examine cohort differences in covariate effects.

Disability was measured using activities of daily living (ADL) and instrumental activities of daily living (IADL; Katz, Ford, Moskowitz, Jackson, & Jaffe, 1963; Lawton & Brody, 1969). The ADL items include walking, bathing, grooming, dressing, eating, transferring, and toileting. The IADL items include using the telephone, driving/traveling, shopping, preparing meals, housework, taking medication,

Table 1. Frequencies, Means, and Standard Deviations of Established Populations for Epidemiologic Studies of the Elderly Data

1912–1921 (cohort age 65–74)	1986–1987	1989	1992	1996
	<i>N</i> = 2,456	<i>N</i> = 1,978	<i>N</i> = 1,593	<i>N</i> = 1,211
Men (%)	39.58	—	—	—
White (%)	45.72	—	—	—
Age	69.07 (2.86)	—	—	—
Education (years)	8.89 (3.97)	—	—	—
Income (\$)	11512.49 (10495.67)	—	—	—
Mortality (%)	40.80	—	—	—
Disability (ADL/IADL)	0.72 (1.86)	1.05 (2.38)	1.41 (2.81)	2.18 (3.58)
Nondisabled (%)	75.37	69.51	63.21	56.25
Disability given onset	2.92 (2.75)	3.41 (3.23)	3.83 (3.48)	4.96 (3.93)
1902–1911 (cohort age 75–84)	1986–1987	1989	1992	1996
	<i>N</i> = 1,226	<i>N</i> = 915	<i>N</i> = 651	<i>N</i> = 363
Men (%)	27.98	—	—	—
White (%)	45.35	—	—	—
Age	78.67 (2.73)	—	—	—
Education (years)	8.11 (4.12)	—	—	—
Income (\$)	8720.19 (8957.95)	—	—	—
Mortality (%)	62.72	—	—	—
Disability (ADL/IADL)	1.46 (2.53)	2.34 (3.40)	3.55 (4.14)	5.09 (3.43)
Nondisabled	57.42	46.89	35.18	22.59
Disability given onset	3.42 (2.89)	4.39 (3.56)	5.46 (4.00)	6.55 (4.11)
1881–1901 (cohort age 85–105)	1986–1987	1989	1992	1996
	<i>N</i> = 273	<i>N</i> = 170	<i>N</i> = 86	<i>N</i> = 30
Men (%)	24.18	—	—	—
White (%)	41.39	—	—	—
Age	87.95 (3.13)	—	—	—
Education (years)	7.34 (4.42)	—	—	—
Income (\$)	6870.02 (7218.37)	—	—	—
Mortality (%)	85.71	—	—	—
Disability (ADL/IADL)	3.10 (3.36)	4.71 (4.03)	5.99 (4.15)	6.93 (4.38)
Nondisabled	30.40	17.06	10.47	16.67
Disability given onset	4.45 (3.19)	5.67 (3.75)	6.69 (3.81)	8.32 (3.34)

Notes: ADL = activities of daily living; IADL = instrumental activities of daily living.

and handling finances. Both indexes measure whether an individual requires help for activities. A summed index (0–14 items) was created based on previous research (Spector & Fleishman, 1998) that has been shown to capture a full range of disability (Cronbach's $\alpha > .91$ for all waves). A logged summed scale, and a separated sum of ADLs and IADLs with similar substantive results (models not shown).

Socioeconomic variables included education and financial resources. Education was self-reported at baseline in years. Financial resources were 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.). Income was recoded to thousands. Analyses were replicated using categorical measures of education (credentials) and income (150% of poverty line, etc.) with no substantive difference. Income is treated as time invariant in these analyses due to the interest in causal order. Roughly 15%–25% of individuals had an increase or decrease of \$5,000 or more in income between waves. Previous research has shown that wealth may

be a more robust indicator of material resources and that wealth may vary more by gender and race/ethnicity (Robert & House, 1996). It is also possible that wealth has protective effects for health that are independent of income. I use income alone because the measures in the EPESE are inadequate in measuring all assets (there is one home ownership indicator) and because I argue assets are a mechanism through which financial resources convey health advantage (and should be included as mediators in subsequent analyses).

All analyses are unweighted because the variables used for sample selection criteria were included in the models (Winship & Radbill, 1994). I include an indicator for mortality to handle selective attrition due to mortality. Other recent research has used similar approaches to show that failure to account for mortality or attrition may bias results in this type of model and data source (Kelley-Moore & Ferraro, 2004). Results including this indicator replicate previous results using a Heckman selection correction technique (Kelley-Moore & Ferraro, 2004). All substantive findings were replicated without the inclusion of the correction, but model fit and stability increased when it was

included. An overall attrition indicator and time varying mortality were tested with no substantive difference. Table 1 provides descriptive statistics for the three cohorts. The Duke EPESE oversampled men (24%–40%) and Black respondents. White individuals comprised 41%–46% of the sample. The mean levels of education were between 7 and 9 years and decreased slightly with older cohorts. On average, mean disability levels increased over the decade for all cohorts, and the percentage of those nondisabled decreased. Mortality across the decade ranged from 41% to 86% by cohort, suggesting a large proportion of the sample died at some point during the observation period.

Analytic Method

Previous findings (Melzer et al., 2001; Zimmer & House, 2003) suggest delayed disability is an important factor in modeling disability trajectories and that covariate effects may differ for prevention and progression of disability. I utilize latent curve modeling to estimate individual transitions and trajectories simultaneously as separate processes through the inclusion of binary onset variables (analyses performed using Mplus Version 3.0). The models described below allow the simultaneous estimation of transition and trajectory. Substantively, it is likely that important predictors of health outcomes vary in their impact on the timing and progression of disease or disability. Methodologically, failure to take timing into account when measuring trajectories may bias estimation of the trajectories leading to inaccurate conclusions of where health disparities lie in cumulative processes (Taylor, 2008).

In order to model transition in a latent variable framework, a discrete-time survival model was used. The timing of first onset was modeled as a hazard probability with a corresponding hazard odds ratio. The hazard probabilities expressed as a function of the observed covariates is equivalent to the logistic regression of \mathbf{u}_t on the observed covariates:

$$P(\mathbf{u}_{ij} = 1) = \frac{1}{1 + e^{-(\text{logit}_i)'}} \quad (1)$$

The models presented allow for an “unstructured” hazard across time, analogous to a piecewise hazard in continuous-time survival analysis (see Allison, 1995; Masyn, 2004). For discussion of discrete-time survival analysis using latent variables see Muthén and Masyn (2005) and Masyn (2004). The binary latent variables capturing the covariate effects on disability onset is the first portion of the model. The second portion is a traditional two-factor latent process estimating the change in the level of disability over time for each individual given the individual’s disability onset.

In order to model disability trajectories given onset over time, the y variable (representing disability level) was modified such that an individual with no disability was missing on variable y (rather than a value of zero) until the time period (t) when disability was observed. The values for y were not-

missing thereafter. Consistent with previous research, disability decreases subsequent to first transition were modeled with the y variable. Although recovery is an important transition, the choice not to model it as a separate transition was based on the lack of consensus on recovery measurement (overall decrease or transition to no disability) and the added instability to the model. A nonlinear model fit best (models not shown), the loadings of λ_{yt} were fixed at 0, 1, 2, and 3.8.

The level 2 equations may be expressed as:

$$\alpha_{yi} = \mu_{\alpha y} + \gamma_{\alpha y} x_i + \zeta_{\alpha_{yi}} \quad (2)$$

$$\beta_{yi} = \mu_{\beta y} + \gamma_{\beta y} x_i + \zeta_{\beta_{yi}} \quad (3)$$

The onset and trajectory of disability are not allowed to covary 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.

RESULTS

Baseline (Unconditional) Model Fit

The unconditional model for the trajectory of disability (without onset) yielded good fit (1912–1921 cohort: comparative fit index (CFI) = 0.952, Tucker-Lewis index (TLI) = 0.943, root mean square error of approximation (RMSEA) = 0.057; 1901–1911 cohort: CFI = 0.953, TLI = 0.953, RMSEA = 0.054; 1881–1901 cohort: CFI = 1.00, TLI = 1.00, RMSEA = 0.00). Separate models for the baseline hazards were also tested (1912–1921 cohort: log likelihood = -3061.41(11); 1901–1911 cohort: -1460.47(11); 1881–1901 cohort: -224.56(11)). The effects of covariates on disability onset must be interpreted differently than on the trajectory coefficients because the hazard (η_u) is a latent variable with dichotomous indicators. The coefficients were exponentiated in order to produce proportional hazard odds ratios. The proportionality assumption holds in these models, therefore the effects of covariates are assumed to be equal across time points. The latent intercept (α_y) and latent slope (β_y) are continuous latent variables and should be interpreted as the level and slope of disability across time given that the individual had onset of disability.

Effects of Demographic and Socioeconomic Covariates

Previous models including only demographic effects (models not shown) generally revealed a protective effect of gender and race (men and white) for the onset of disability, although race effects weakened in the oldest cohort, consistent with selective mortality. Age significantly delayed disability and suppressed the slope of disability; however, the intercept (or level) of disability was slightly higher among younger individuals. This finding supports previous studies on the compression of morbidity in younger cohorts (Fries, 1980).

Table 2. Coefficients (*SEs*) for demographic predictors and education effects

Covariate model including education	Hazard OR	95% CI	Intercept	<i>SE</i>	Slope	<i>SE</i>
1912–1921 (cohort age 65–74, <i>N</i> = 2,456)						
Men	0.638***	0.549–0.727	0.033	0.202	–0.055	0.108
White	0.852***	0.735–0.969	–0.208	0.206	0.208*	0.104
Age	1.051***	1.027–1.076	–0.069***	0.033	0.050***	0.017
Education	0.889***	0.871–0.906	0.019	0.026	–0.031***	0.013
Mortality	2.646***	2.262–3.030	1.570***	0.199	0.447***	0.126
Intercept	—	—	6.213***	2.308	–2.456***	1.165
Variance	—	—	5.501***	0.506	0.885***	0.154
Covariance	—	—	–0.660***	0.257		
	1986–1987	1989	1992	1996		
Threshold (τ)	3.814*** (0.817)	3.874*** (0.818)	3.622*** (0.818)	3.008*** (0.817)		
Log likelihood	–8576.758	(28)		BIC	17372.093	
1902–1911 (cohort age 75–84, <i>N</i> = 1,226)						
Men	0.674***	0.532–0.815	0.294	0.264	–0.344***	0.155
White	0.834	0.672–0.995	–0.104	0.243	–0.012	0.129
Age	1.110***	1.070–1.149	0.023	0.041	0.087***	0.023
Education	0.906***	0.883–0.929	0.008	0.030	–0.037***	0.016
Mortality	1.992***	1.594–2.390	1.734***	0.251	0.531***	0.143
Intercept	—	—	–0.168	3.244	–5.197***	1.823
Variance	—	—	5.895***	0.641	0.854***	0.203
Covariance	—	—	–0.183	0.326		
	1986–1987	1989	1992	1996		
Threshold (τ)	7.943*** (1.413)	8.039*** (1.409)	7.542*** (1.409)	6.588*** (1.415)		
Log likelihood	–5711.851	(28)		BIC	11622.825	
1881–1901 (cohort age 85–105, <i>N</i> = 273)						
Men	0.469***	0.210–0.728	–0.579	0.545	0.178	0.485
White	0.979	0.478–1.480	–0.140	0.468	–0.333	0.324
Age	1.208***	1.080–1.336	0.161***	0.070	0.068	0.056
Education	0.928	0.873–0.982	0.026	0.051	0.069	0.037
Mortality	2.344***	0.911–3.778	2.395***	0.682	0.535	0.323
Intercept	—	—	–12.232***	6.053	–4.619	4.854
Variance	—	—	6.868***	1.275	0.490***	0.211
Covariance	—	—	0.272	0.634		
	1986–1987	1989	1992	1996		
Threshold (τ)	7.943*** (1.413)	8.039*** (1.409)	7.542*** (1.409)	6.588*** (1.415)		
Log likelihood	–1297.187	(28)		BIC	2745.829	

Notes: CI = confidence interval; OR = odds ratio; BIC = Bayesian information criterion.

* $p < .05$; ** $p < .01$; *** $p < .001$.

Table 2 reports the results from the nested model including demographic variables and education. The effects of education were highly significant in reducing the odds of disability onset in all but the oldest cohort. The effects of education were also significant for the progression of disability, such that each year of education decreased the slope of disability by .03 to .04 items per wave. The effects of race on disability onset decreased from previous models (not shown) by roughly half in the 1912–1921 cohort and became nonsignificant in the 1902–1911 cohort. In the 1902–1911 cohort, white individuals experienced a .21 unit increase in disability at each wave compared with nonwhites. This suggests that racial disparities in disablement over time are nuanced and may reflect greater compression among whites, highlighting the importance of testing timing of disability within trajectories.

Table 3 presents the final nested model including income, education, and the demographic covariates. The findings for

income were interesting in the youngest (1912–1921) cohort. Income had a significant effect in delaying disability onset and a muting effect on the slope of disability over time, such that with each thousand dollar increase, individuals accumulated disability at a rate that was .01 units less per wave. However, the effect of income on the intercept of disability was positive, suggesting a compression effect. With each thousand dollar increase, the intercept (disability level at baseline given onset) increased by .03 units.

The independent effects of income were restricted to disability onset for the 1902–1991 cohort, and no significant effect was observed in the oldest (1881–1901) cohort. Upon the inclusion of income in the models, the protective effects of education remained highly significant and stable in magnitude for the youngest two cohorts and remained nonsignificant in the oldest cohort. This supports previous findings on the robustness of education's effects, and suggests that

Table 3. Coefficients (SEs) for Demographic Predictors and Socioeconomic Status Effects

Covariate model including education and income	Hazard OR	95% CI	Intercept	SE	Slope	SE
1912–1921 (cohort age 65–74, N = 2,456)						
Men	0.702***	0.600–0.804	–0.102	0.210	0.024	0.108
White	0.928	0.795–1.060	–0.361	0.215	0.294***	0.104
Age	1.047***	1.022–1.072	–0.062	0.033	0.045***	0.017
Education	0.907***	0.887–0.926	–0.009	0.028	–0.016	0.013
Income	0.981***	0.972–0.991	0.032***	0.014	–0.018***	0.007
Mortality	2.604***	2.226–2.982	1.605***	0.199	0.428***	0.1260
Intercept	—	—	5.743***	2.309	–2.140	1.165
Variance	—	—	5.414***	0.504	0.857***	0.153
Covariance	—	—	–0.614***	0.255		
	1986–1987	1989	1992	1996		
Threshold (τ)	3.557*** (0.821)	3.614*** (0.821)	3.357*** (0.822)	2.735*** (0.821)		
Log likelihood	–8564.519	(31)		BIC	17371.033	
1902–1911 (cohort age 75–84, N = 1,226)						
Men	0.724***	0.564–0.884	0.328	0.277	–0.339***	0.108
White	0.866	–1.003 to 2.734	–0.091	0.244	–0.008	0.104
Age	1.107***	1.068–1.146	0.022	0.041	0.087***	0.017
Education	0.919***	0.894–0.945	0.014	0.034	–0.036	0.019
Income	0.986***	0.973–1.000	–0.008	0.019	–0.001	0.007
Mortality	1.996***	1.597–2.395	1.736***	0.251	0.532***	0.126
Intercept	—	—	–0.123	3.248	–5.205***	1.825
Variance	—	—	5.899***	0.641	0.856***	0.203
Covariance	—	—	–0.186	0.327		
	1986–1987	1989	1992	1996		
Threshold (τ)	7.869*** (1.414)	7.962*** (1.410)	7.463*** (1.410)	6.499*** (1.416)		
Log likelihood	–5709.521	(31)		BIC	11639.499	
1881–1901 (cohort age 85–105, N = 273)						
Men	0.538***	0.224–0.852	–0.381	0.564	0.175	0.495
White	1.068	0.503–1.634	–0.031	0.475	–0.325	0.342
Age	1.214***	1.086–1.343	0.165***	0.069	0.068	0.056
Education	0.946	0.884–1.007	0.057	0.057	0.067	0.039
Income	0.974	0.940–1.009	–0.053	0.042	0.003	0.033
Mortality	2.280***	0.877–3.683	2.372***	0.680	0.540	0.324
Intercept	—	—	–12.611***	6.045	–4.598	4.854
Variance	—	—	6.807***	1.267	0.486***	0.211
Covariance	—	—	0.274	0.632		
	1986–1987	1989	1992	1996		
Threshold (τ)	16.069*** (4.717)	15.974*** (4.683)	15.709*** (4.703)	17.024*** (4.831)		
Log likelihood	–1295.187	(30)		BIC	2758.658	

Notes: CI = confidence interval; OR = odds ratio; BIC = Bayesian information criterion.

* $p < .05$; ** $p < .01$; *** $p < .001$.

there are independent mediators through which income and education work to affect disability.

In the final models, the effects of gender on onset were reduced in magnitude but remained significant as hypothesized. The protective effects of race were also mediated; however, a significant positive effect of race (white) remained for disability progression in the youngest cohort, even after controlling for selective mortality. The effects of age were almost unchanged with the inclusion of income in the models. In order to aid in the interpretation of socioeconomic effects on timing and trajectory of disability in these models, results for the 1912–1921 final models are plotted in Figure 1.

The hazard probabilities are plotted for disability onset in the first section of the figure, where the effects of covariates are fixed to their respective means. The robust independent

effects of education become clear when expressed in terms of credentials. Compared with the mean, the effect of 12 years of education (high school) are similar to roughly double an individual’s annual (household) income. The effect of 16 years of education (college) was comparable with quadruple an individual’s income such that the probability of onset decreases by almost half. The second part of the figure presents the significant findings of income on the progression of disability given an individual’s initial onset. On average for this cohort, individuals report an initial disability level of 2.2 given they have experienced disability onset. Individuals with higher income actually report higher levels of disability given onset, but increase at a slower rate such that an individual with double the mean income would expect a disability level that is 12% lower compared with the mean at the end of the decade.

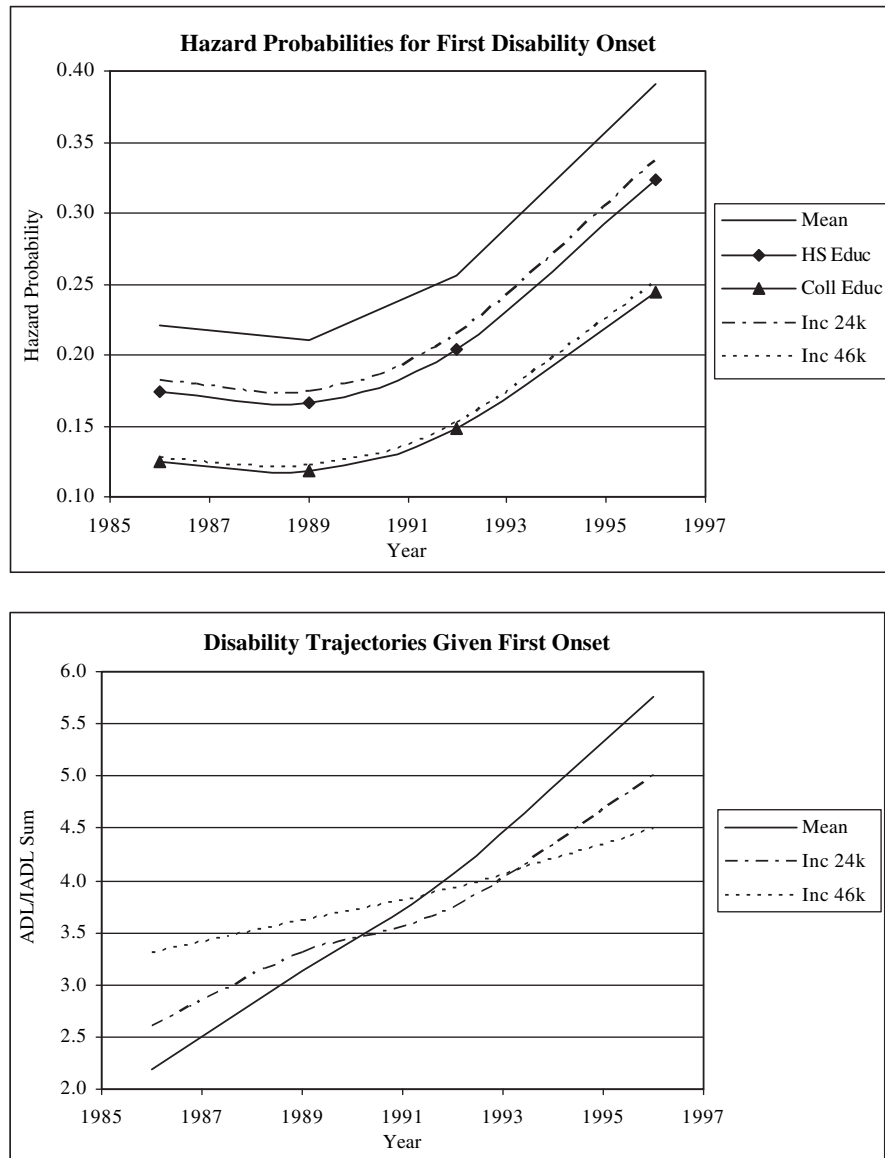


Figure 1. Socioeconomic Status effects in the 1912–1921 cohort.

An individual with quadruple the mean income would expect a 21% decrease in disability level compared with the mean.

DISCUSSION

Disability is predominantly conceptualized in the literature as a life course process, and previous studies have analyzed it as either a collection of independent transitions or a continuous trajectory of progression encompassing a baseline level and slope over time or age. The goal of this article was to measure disability both as a transition and trajectory, capturing the life course process of disablement and further illuminating the way socioeconomic factors work on disability over time. Findings show support for both conceptualization and measurement of disability using this analytic approach, previously absent in sociological research.

Concerning the role of socioeconomic status on disability, the results generally support previous findings by House and others (House, 2001; House et al., 2005) on how education and financial resources protect individuals differentially. Education played a strong, persistent, preventive role in delaying disability. In fact, even in the final model, individuals with higher levels of education (credentials) were generally 23%–45% less likely to experience disability compared with those with mean levels of education. This suggests education's role in disablement is indeed preventive and likely works through a number of social and behavioral mechanisms independently of income such as health behaviors and mastery. Occupation was not included in the analyses because the majority (88%) of individuals were not employed during the survey period and women comprised most of the sample. Further exploration of occupation's role

in the pathway of socioeconomic status and disability is reserved for future analyses.

The independent effects of income were significantly preventive but were not consistent and robust compared with those of education. However, the greatest support for a compression effect was found for income, where disability was delayed such that higher levels of income predicted higher levels of disability once onset occurred. This effect was not replicated in older cohorts; therefore, generalization should be made with caution. It is possible also that this effect is fueled by increased income affecting reporting of disability (i.e., individuals report more need because they can afford it). Sensitivity analyses using this and other data and alternate methods suggest that these compression effects are not artifacts of the data or models. This finding generates support, however, for further exploration of income effects on disability, net of education, for the health of older adults over time.

Income also had a significant muting effect on the slope of disability over time for the youngest cohort. This suggests that financial resources not only serve a preventive role, but are also primary in decreasing disability accumulation over time once it occurs through access to material resources (formal and informal care, prescriptions, etc.). Although the effects of income were observed in the youngest cohort, they diminished in older cohorts, suggesting effects may either decrease with age or increase among younger cohorts. It is also possible that this measure becomes less valid as individuals age, spend down, or move into institutional living arrangements. The underlying causes will be addressed in future research specifically examining mediators of socioeconomic variables and cohort and mortality effects.

The findings on gender and race suggest that overall, men and white individuals delay disability longer than their counterparts but do not necessarily experience a slower progression of disability decline. This is consistent with some but not all recent findings on disability trajectories in the literature (Liang et al., 2008; Taylor, 2008). The negative effects for nonwhite individuals were mediated when socioeconomic variables were included. It should also be noted that marital status at baseline was included in sensitivity analyses with no substantive differences (models not shown). I argue that marital status is a support mechanism and therefore should be included as a mediating mechanism of socioeconomic status in subsequent research. In addition, this variable should be considered time varying across the decade in this sample, and the analyses presented here were reserved to time invariant covariates.

The effects of cohort generally supported the hypothesis presented, and previous research on cohort effects in disability declines (Manton et al., 2008). However, it should be noted that it is not possible to disentangle the specific effects of age and cohort here. Future research should address the role of age, period, and cohort in a more detailed manner in light of these findings. Although correction for selective mortality was included in the analyses presented, direct measurement of the competing risk of death was not addressed. Sensitivity analy-

ses suggest that mortality selection did not significantly bias estimates of disability progression but it should be noted that findings in the older cohorts should be taken with caution due to the high attrition rates. Mortality should be viewed as both a selective and competing force in comparison with disability in later life; therefore, future research should address transitions into mortality along with those into and within disability.

A notable limitation of these analyses is the time-invariant nature of predictor variables. Although income at baseline was used, it remained stable over time in terms of large changes. Future research should include time-varying predictors known to affect disability, particularly marital status and social support. As mentioned previously, this article is a first step in disentangling the causal pathways involved in disability trajectories in later life. Future research now underway will study these pathways, examining chronic conditions along with mediating and moderating factors such as lifestyle behaviors, social support, and health care access.

The present study shows that education and income do indeed differ in their effects on disability among older adults over time, speaking to inconsistent findings in accordance with the life course perspective. Dissentangling these effects highlights the importance of transitions and trajectories as conceptualized by previous theory on social stratification and health (House, 2001). These findings may serve as a foundation for research addressing the pathways through which socioeconomic factors work through shared and independent mechanisms such as health behaviors and access to care. Furthermore, the present study highlights the usefulness of disaggregating transitions and trajectories in studies of health outcomes over time where the hypothesized processes affecting onset and progression may differ.

FUNDING

The research on which this article is based was performed pursuant to Contract Number N01-AG-1-2102 with the National Institute on Aging, in support of the Established Populations for Epidemiologic Studies of the Elderly (Duke University). M.G.T. was supported by National Institute on Aging (grants F32AG026926 and K99AG030471).

ACKNOWLEDGMENTS

I thank Linda K. George, Kenneth C. Land, Angela O'Rand, Scott M. Lynch, George K. Maddox, and four anonymous reviewers for their comments on this research.

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