Disentangling the Life Course Process of Disability in Later Life: The Role of Socioeconomic Status

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Abstract:

Over the past few decades, disability has become one of the most salient and researched outcomes for older adults in the United States. It is predominantly conceptualized as a life course process and is increasingly measured over time using transitions or trajectories. In this paper, I extend this research by using methods unutilized in aging research to measure disability as both a transition and trajectory, therefore better capturing it as a life course process. Previous theory and research on SES and health suggests that education and financial resources work differently on disability over time. I revisit these arguments, finding that the independent effects of education are mainly protective for onset, where financial resources also impact level of disability given onset. I discuss these findings in terms of past and future research, focusing on what may be gained by conceptualizing and measuring disability in later life as a life course process.

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Among the most notable global demographic trends of the 21st century is population aging, where the mortality and fertility declines seen almost universally across the globe have meant an increasing percentage of the population at older ages (Gavrilov & Heuveline, 2003). Of particular importance to population projections and resources is the measure of active life expectancy. Even with the observed increases in active life (see Freedman, Martin, & Scheoni, 2004; Crimmins, 2004), there will still be an increasing number of elderly in need of services and policy that reflect a valid picture of their disablement processes (Spillman, 2004; Uhlenberg, 1992). Disability, the measure commonly underlying active life expectancy calculations, has therefore become one of the most salient and researched outcomes for older adults. As a variable, it predicts mortality, institutionalization, depression, and other key outcomes for individuals at high levels of significance. By the accepted standards of conceptualization and measurement, disability is nested in the social context of an individual's biography, arguably even more than self-reports of health status. It is partially defined as the inability of any individual to function in his/her respective roles in society. Therefore, it is both extremely powerful as a predictor and inherently sociological as an indicator of health.

Disability is primarily conceptualized as a life course process (see Verbrugge & Jette, 1994). Accepted theory of disablement fits neatly into a life course framework, with its emphasis on transitions that fit into trajectories in a human life. Increasingly, measurement of disability and analytic strategies used to study it have encompassed the dynamic nature of disability over time (Manton, 1988; Taylor & Lynch, 2004). Mapping disability experience longitudinally fits accepted theory on the disablement process and the life course framework.

Given the observed declines in disability prevalence among older adults in the United States, researchers have begun to examine what may be fueling declines. One factor of noted importance is socioeconomic status (Freedman & Martin, 1999; Zimmer & House, 2003). Other recent research has focused on medical technology (Manton, Stallard & Corder, 1997). However, none of this recent research has examined the interplay of mechanisms in the causal pathway leading to disability. In addition, none of this research has addressed disability using long-term individual trajectories, but rather onset, prevalence, and transition.

Two main arguments have been presented in the connection of socioeconomic factors and health. The first suggests that education is primary and robust as a predictor and may work mainly to prevent or delay poor health. The second focuses on financial resources, suggesting that they serve both a preventive role on onset and a "muting" effect on level once onset of poor health has occurred. It is possible that both strains of thought are correct in hypothesizing the protective effects of these measures of socioeconomic status.

In this paper, I bridge theory of the disablement process and the social stratification of health within the framework of the life course to disentangle the effects of socioeconomic status on disability as a life course process in later life. By using methods previously absent in the aging literature I test these effects in turn on transitions and growth in disability over time, therefore conceptualizing *and* measuring disability as a life course process. I argue that understanding the way socioeconomic indicators work on disability over time illuminates the forces behind the observed declines in disability over the last decade. I aim to disentangle these effects as the first step in understanding how socioeconomic status works in the causal pathways leading to differential trajectories of disability.

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". They frame disablement as a process of transitions over time that creates a trajectory of disability in the life of an individual. Given the theoretical model, it is not only the level of disability that is assumed to vary, but rather the whole process, making differential "outcomes" of disability pieces of a dynamic process occurring over time. The Verbrugge and Jette (1994) model provides a theoretical view of disability as a process and social phenomenon, predisposed by individual risk factors and possibly mediated by extra-individual factors and intra-individual characteristics. The research presented in this study focuses mainly on the final stage of the theoretical model rather than the process itself. However, it does take the dynamic nature of the outcome into account, along with its time dependence. In addition, time invariant individual risk factors are investigated in order to distinguish and explicate disability trajectories. Understanding how the most basic of these predictors works in the timing and accumulation of disability as a longitudinal dynamic outcome provides a starting point for research aimed at disentangling these effects within the pathway from pathology to disability.

In conceptualizing and measuring disability as a process that is dynamic and occurring across age or time, it is useful to draw on the theoretical perspective of the life course, since it clearly outlines the assumptions about life events and the human life span. In addition, the life course framework gives a backbone on which to build hypotheses about the differential disability experiences of individuals over time. Elder (1992) describes the term life course as both a theoretical perspective and a concept in itself. As a theoretical perspective, the life course

framework shapes the way researchers identify and formulate questions, choose variables, and develop research design and analytic strategies. As a concept, the life course is defined by short-term transitions (e.g., transition from single to first marriage) nested in long-term trajectories (e.g., marriage trajectory) that give each trajectory its shape and meaning (Elder, 2000). Researchers studying health and health transitions in later life have been contributing to the life course literature for some time, but actual measurement of individual-level trajectories in this field of study is still somewhat rare (George, 2003).

Socioeconomic effects

The study of the impact of social inequalities on health is arguably the foremost subject of research within medical sociology at present. Understanding how social factors influence health and how individuals are stratified on the basis of health has been a primary focus of the discipline for decades. Although there is no dispute of the robustness of this association, the emphasis on measures (education, income, employment status, occupation) and proposed pathways (learned health behaviors, access to health resources, etc.) through which social status affects health vary (see Evans, 2002). One body of research emphasizes the importance of education as a predictor of health. This measure is usually assumed to be time invariant among older adults and has the benefit of marking advantage earlier in the life course (where prospective observations of income do not often allow the study of resources upstream of the observation period).

The general framework emphasizing education proposes that it works through various mediators to affect health. These include work and economic conditions, social-psychological resources, and health behaviors/lifestyle (Ross & Wu, 1995). This perspective treats education as the "root component of social status" yielding accumulated social inequalities thereafter

(Mirowsky, Ross, & Reynolds, 2000). More recently, the focus of this research has shifted to conceptualizing education as *learned effectiveness*. The authors (Mirowsky & Ross, 2003) propose that the protective effect of education on health, net of the effect of income, is the result of education fostering healthy life behaviors and giving individuals a sense of control over their lives. Other research emphasizes the material components of socioeconomic status, namely income or financial resources (see Lynch & Kaplan, 2000). In this perspective, 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). Financial resources therefore not only serve to protect individuals through a decrease in the occurrence of life stressors (prevention), but also serve to mute the effects of negative outcomes both on health and of health.

It should be noted that socioeconomic status is one of the strongest factors thus far linked to the observed disability declines (Freedman & Martin, 1999). In specific, education has been shown to be most salient for the transition into disability over time, where income has been shown to be protective in transitions within level of disability for adults (Zimmer & House, 2003). Therefore, previous research using disability as an outcome has noted the independent effects of these factors and their differential effects.

Research on Disability Transitions and Trajectories

Researchers have been increasingly focused on the heterogeneity among disabled elderly and the dynamic nature of the disablement process (see Crimmins & Saito, 1993). The work of Manton in 1988 is one of the earliest classic pieces on disability change/transition in the gerontological literature, focused mainly on gender differences (see also Branch, 1984). Transitions in impairment status have also been predicted by age, race, previous functional state,

income, and education (Beckett et al. 1996; Maddox, Clark, & Steinhauser, 1984; Schoeni, Freedman, & Wallace, 2002).Other work has focused on demographic and social predictors of recovery (Kempen et al. 2001; Mendes de Leon et al. 1999).

Previous literature has also focused on disability measured as a trajectory. These studies mirror 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 et al., 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.

Due to the previous literature on disability trajectories, I hypothesize that disability will increase for individuals on average over time (i.e. positive growth). In addition, women will be both more likely to transition into disability and to have a higher level of impairment compared to men given onset (see Leveille et al., 2000; Schoeni, Freedman & Wallace, 2002). Nonwhite individuals are also expected to be disadvantaged on both onset and level (Hayward & Heron, 1999; Manton, Patrick, & Johnson, 1987; Manton & Gu, 2001), although the observed mortality crossover by race may weaken these effects (see Corti et al., 1999; Wing et al., 1985).

Due to the previously discussed arguments on the differential protective effects of education and financial resources, I hypothesize that education will work mainly to delay disability onset (prevention) and that financial resources will work both to delay disability onset and to decrease the level of disability once onset has occurred. In addition, I hypothesize that results will vary by cohort, with older cohorts experiencing greater levels of onset and level given onset.

RESEARCH DESIGN

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 project was sponsored by NIA and was conducted at multiple cites to investigate predictors of mortality, chronic disease, disability, and service utilization including long-term care. The Duke sample consists of individuals aged 65 and older residing in the community. Respondents participated in 4 in-person and 4 telephone based interviews. Only in-person interviews are used in the following analyses[1]. The baseline survey was conducted in 1986-7, with follow-up in-person data collected in modal years 1989, 1992, and 1996. The original Duke Sample consisted of 4,162 individuals. Of these, 4,030 had some report (non-missing) of disability status at baseline in 1986-7. This loss of 132 cases comprised only 3.2% of the total sample. Further, 63 cases were deleted for missingness on key predictor variables leaving a sample size of 3,967[2]. I include age (years), gender (male=1), and race (white=1, non-white=0) as demographic controls. In addition, I split the sample into cohorts (65-74, 75-84, 85+) in order to examine cohort differences in demographic and socioeconomic effects.[3]

Disability was measured using Activities of Daily Living and Instrumental Activities of Daily Living (Katz et al., 1963, Lawton & Brody, 1969). The ADL index includes a list of daily activities such as walking, bathing, eating, dressing, etc. A scale of these items produced by Manton was used ranging from 1 to 6 (1=no IADL's or ADL's, 2=IADL limitations only, 3=1-2

[1] Although respondents were asked a wide variety of questions during telephone interviews, they were not asked about Instrumental Activities of Daily Living. This measure is one of the main variables of interest in these analyses. Therefore, only in-person interviews are used. Proxy responses *are* included in Waves 2-4.
[2] The analytic strategy handles attrition over time due to all sources, therefore individuals were allowed to stay in the analyses until they dropped out.
[3] Alternate analyses were included using cohort as a control in the model and revealed similar results. Due to multicollinearity concerns and the possibility that cohorts may have distinct shapes of average trajectories over time I chose the split sample.

ADL limitations, 4=3-4 ADL limitations, 5=5-6 ADL limitations, and 6=Institutionalized). This scale was chosen due to the fact that it was based on how individuals accumulate disability across age (see Manton & Gu, 2001). All analyses were replicated with a summed scale (i.e. Spector & Fleishman, 1998) and a separated sum of ADL's and IADL's, respectively, with similar substantive results (models not shown).

Socioeconomic variables included education and financial resources. Education was self-reported in years and recoded into categories representing credentials (0-8, 9-12, 13+ years). Preliminary analyses for this data set were replicated using a continuous measure of education, and substantive findings did not differ. Financial resources were self-reported for the past year by individuals representing both individual's and spouse's income from all sources and recoded into 3 categories (less than $10,000 per year, $10,000 to less than $20,000 per year, and $20,000 and greater) with cutpoints based on 150% of the poverty level for this age group at baseline to reflect those least advantaged (<$10,000) and the most advantaged (10%) of the sample fell into the highest category ($20,000+). The reference category was less than $10,000. Analyses were replicated using continuous measures of both education and income with no substantive difference. I chose these categories to reflect marked differences in achievement and for ease of interpretation. In addition, the recode of income allowed it to be treated as time invariant, since only a maximum of 5% of individuals transitioned between income categories at any wave.

Discrete-time Survival Analysis using Latent Variables

Previous findings (author citation; Zimmer & House, 2003) suggest delayed onset is an important factor in modeling disability trajectories over time and that the protective effects of covariates may differ for onset and growth of disability level once onset occurred. However, none of this research has examined these effects both in terms of transitions *and* long term

trajectories among older adults. I turn to latent growth curve modeling to explore these different processes, however, the approach toward growth taken here is slightly different than that used previously in aging research. Using latent variables, I estimate individual onset and growth simultaneously as two separate processes through the inclusion of binary observed variables in structural equation modeling (in addition to continuous variables).[4]

General equations: Discrete time Survival and Two-factor Growth Process

If we define **u** as a vector of binary measures, then we may define $\mathbf{u}_i^* = (u_{i1}^*, u_{i2}^*, \ldots. u_{ir}^*)'$ as the continuous response propensities underlying **u**. As expressed, u_j^* reflects u_j through the parameter threshold τ_j such that:

$$P(u_{ij} = 1) = \frac{1}{1 + e^{-(-\tau_j + u_{ij}^*)'}} \qquad (2)$$

The higher τ is, the higher u^* must be in order to exceed it. For further discussion on binary variables and thresholds in SEM and growth modeling, see Muthén (1996).

In order to model onset as a discrete-time survival model in a latent variable framework, it is possible to use binary (0/1) event history indicators $u_j, j = 1,2,\ldots J$) where $u_{ij} = 0$ if individual i is at risk for the event at time j, but the event did not occur, and $u_{ij} = 1$ if individual i experiences the event at time j, where J is the last time period of data collection. The hazard probabilities expressed as a function of the observed covariates is equivalent to the logistic regression of $\mathbf{u}_i$ on the observed covariates. Therefore, Equation 2 may be written as:

$$P(u_{ij} = 1) = \frac{1}{1 + e^{-(\mathbf{logit}_{ij})'}} \qquad (3) \qquad \text{where}$$

[4] All analyses were performed using *Mplus*.

$$\text{logit}_{ij} = \beta_j + \lambda'_{uj}\,\eta_{ui}, \qquad (4)$$

$$\eta_{ui} = \alpha_u + \gamma'_u\,x_i \qquad (5)$$

where β_j is the time-specific logit intercept parameter ($\beta_j = -\tau_j$), λ_{uj} is a f x 1 logit parameter vector that may vary across time, α_u is a f x 1 time-invariant logit parameter logit, γ_u is a q x 1 time-invariant logit parameter vector, and η_{ui} is a continuous latent variable vector representing onset (u) for each individual i. For further discussion of discrete-time survival analysis and assumptions using latent variables, along with applications, see Muthén and Masyn (2005). For instruction on data manipulation and the interpretation of the hazard using the latent variable approach, see Masyn (2004). The discrete-time survival analysis modeling the effects of the covariates on disability onset is the first portion of the overall model. The second portion is a traditional two factor latent growth process estimating the growth in the level of disability over time for each individual *given* the individual's onset.

In order to model disability level given onset, the y variable (representing disability level) was modified such that an individual with no disability was missing on variable y (rather than having a value of zero as in traditional growth models) until the time period (j) when onset occurred, at which time individual i would have some nonzero value for variable y. The values for y would be nonmissing thereafter. Only initial onset was used in these analyses (individuals were not considered at-risk of onset subsequent to initial onset) for consistency with other event history models, therefore decreases in level of disability subsequent to first onset were modeled with the y variable even though u = missing on all time periods subsequent to onset.

The general level 1 equation for growth may be expressed as:

$$y_{it} = \alpha_{yi} + \beta_{yi}\lambda_{yt} + \varepsilon_{yit} \qquad (6)$$

where y_{it} is a vector of repeated measures representing disability level (y) for each individual i at time t, α_{yi} is a vector of latent intercepts for variable y (disability level) for each individual i, β_{yi} is a vector of latent slopes for variable y (disability level) for each individual i, λ_{yt} is a vector of fixed/freed loadings for variable y representing time (t), and ε_{yit} is a vector of disturbance terms assumed to have equal variances.

The level 2 equations may be expressed as:

$$\alpha_{yi} = \mu_{ay} + \gamma_{\alpha_y} x_i + \zeta_{\alpha_{yi}} \qquad (7)$$
$$\beta_{yi} = \mu_{\beta y} + \gamma_{\beta_y} x_i + \zeta_{\beta_{yi}} \qquad (8)$$

where μ_{ay} and $\mu_{\beta y}$ are the mean intercept and slope for growth in disability level (y) across all cases, γ_{α_y} and γ_{β_y} are vectors of the parameter coefficients of covariate x pertaining to the latent intercept and slope, and $\zeta_{\alpha_{yi}}$ and $\zeta_{\beta_{yi}}$ are disturbance terms assumed to have means of 0 and be uncorrelated with ε_{yit}. The onset and growth of disability are not allowed to covary in these models due to identification reasons, a noted limitation of this type of analysis.

RESULTS[5]

Effects of Demographic Covariates[6]

As a reminder, the measurement of disability level no longer includes a value for "no disability" since this is captured by the onset portion of the model (i.e. the lowest level of

[5] For the sake of brevity, descriptive statistics will not be discussed here. Information on the composition of the Duke EPESE sample on variables of interest is both available in previous literature (see Taylor and Lynch 2004) and available from the author on request.

[6] The results for the unconditional models of the predicted hazard and latent growth model are not discussed for the sake of brevity. These analyses, and their discussion, are available from the author.

disability growth here is IADL level disability). Therefore if individuals had an initial disability level of 1.5 on average, it would mean that among individuals reporting first onset the initial level of disability on average was between IADL level (1) and 1-2 ADL's (2). Table 1 reports the coefficients of the effects of demographic covariates on the onset and growth of disability for each cohort. The slope of average disability increase was steeper in older cohorts, with those in the 1912-1921 cohort increasing by .37 units per wave where those in the 1902-1991 and the 1881-1901 cohort increase by .63 and .76 units per wave respectively.

The effects of covariates on the onset of disability must be interpreted differently than on growth since η_u is a latent variable with dichotomous indicators. The coefficients are presented here for consistency, but the values must be exponentiated in order to produce the proportional hazard odds ratio. For example, the effect of gender in the 1912-1921 cohort is -0.198. Therefore $Hazard\ OR_{gender}$ = exp (-0.198) = 0.82. As a reminder, the proportionality assumption holds in these models, therefore the effects of covariates are assumed to be equal across time points. I will proceed by discussing the effects of covariates on η_u in terms of the proportional hazard odds ratios. The effects of covariates on the latent intercept (α_y) and latent slope (β_y) are more easily interpreted given the coefficient since these are continuous latent variables with continuous indicators.

For the 1912-1921 cohort, males were 18% less likely than females to experience the onset of disability and white individuals were 56% less likely to experience onset compared to nonwhites. For each year of age, the likelihood of onset increased by 8% among this cohort. Among those with disability onset, there was no significant difference in either the latent intercept or the latent slope by gender. White individuals had a .12 unit increase per wave on

average in the slope of disability compared to nonwhites. However, there was no significant difference in the initial level (latent intercept) of disability by race. For each year of age the slope of disability increased .02 units per wave. However, there was no significant difference in the intercept of disability by age among this cohort.

- Table 1 about here -

For the 1902-1911 cohort, males were 20% less likely to experience onset compared to females. White individuals were 54% less likely to experience onset compared to their nonwhite counterparts and each year of age increased the likelihood of onset by 14%. The effects of covariates were similar to those found in the youngest cohort on the latent intercept of disability. That is, once onset was modeled separately, the covariates did not significantly predict initial level of disability. The effects of gender and race on the latent slope of disability were not significant. However, with each year of age the slope of disability increased by .05 units per wave.

As might be expected given the sample size andmortality selection, the significant effects of demographic covariates were sparse in the oldest (1881-1901) cohort. In addition, it has been noted that the individual variation around the mean latent slope is not significant. The only significant effect was that of age on the onset of disability. Each year of age increased the likelihood of onset by 22% within this cohort.

Effects of Socioeconomic Covariates

Table 2 reports the results from the nested model including education. The effects of education were highly significant on the onset of disability among the youngest (1912-1921) cohort, with at least some high school education decreasing the likelihood of onset by 45%. Having at least some college education decreased the onset of disability by 63% compared to

those having less than high school education. Once education was included in the model, the effects of gender on the onset of disability increased, with males 32% less likely to experience onset compared to females. The effects of race, however, were partially mediated. Whites were 20% less likely to experience onset compared to their nonwhite counterparts. The effects of age remained similar, with each year increasing the likelihood of onset by 7% within the cohort.

For this cohort, the effects of education were nonsignificant on growth with the exception of some college education on the initial level of disability given onset. Individuals with some college education had a .24 unit increase in initial disability level compared to those with less than high school education. The effects of race and age on the slope of disability remained roughly stable with the inclusion of education.

- Table 2 about here -

For the 1902-1911 cohort, the effects of education on onset were similar to those of the youngest cohort. Having at least some high school decreased the likelihood of disability onset by 42% and having at least some college decreased the likelihood of onset by 59%. The effects of gender remained relatively stable when education was included in the model, with males 21% less likely than females to experience onset. Similar to the youngest cohort, the effects of race decreased when education was introduced. Whites were 18% less likely than nonwhites to experience onset. Each year of age increased the likelihood of onset by 14%.

The effects of education on disability growth were interesting for this cohort. Given first onset, the initial level of disability was .18 units higher for those with some high school education. Similarly, those with some college education had an initial disability level of .279 units higher than the reference group. For this cohort, however, education did have some negative effect of the growth of disability over time. Individuals with some college education

had, on average, a slope of disability that was .27 units less per wave compared to those with less than high school education.

The effects of the demographic covariates on the intercept of disability remained nonsignificant when education was introduced into the model. However, the effects of gender and race on the slope of disability became significant with males having a .15 unit decrease per wave on the slope of disability compared to females. White individuals actually had a .15 unit increase in slope on average compared to nonwhites when education was included. Age retained its effect on the slope of disability in this model.

The oldest cohort (1881-1901) had surprisingly strong effects of education on onset. Individuals with some high school education had 50% less likelihood and those with some college had 52% less likelihood of onse. The effects of gender on onset became significant in this model, with males 45% less likely than females to experience onset. The effect of age remained similar, with each year increasing the likelihood of onset by 24%. As with the 1992-1911 cohort, education did have some positive effect on the initial level of disability growth given onset. Individuals with some high school education had a .326 unit increase in the latent intercept compared to those with with less than high school education. Similar to the previous models, none of the covariates predicted the latent slope of disability growth in this cohort.

Table 3 presents the final nested model including income, education, and the demographic covariates. For the youngest (1912-1921) cohort, income did have a significant negative effect on onset. Those making $10-20,000 per year were 47% less likely to experience onset and those making $20,000 dollars and more per year were 45% less likely to experience onset. The effects of income partially mediated the effects of education on onset. Those with some high school education were now 38% less likely than those with less than high school

education to experience onset. Individuals with some college were 49% less likely to experience onset. The effects of gender and race on onset in this cohort were reduced and became nonsignificant once income was introduced in the model. The effects of age remained similar with each year increasing the likelihood of onset by 6%.

- Table 3 about here -

The effects of income on the initial level of disability given onset were nonsignificant. However, the positive effect of education apparent in the previous model was reduced to nonsignificance. Income did have a negative impact on the slope of disability, with those making $10 to 20,000 having a .21 unit decrease on average per wave and those making $20,000 or more had a .19 unit decrease in slope. The positive effect of race remained on the slope of disability growth, with white individuals having a .19 unit increase in slope on average per wave. Each year of age increased the slope of disability by .02 units per wave.

For the 1902-1911 cohort, the effects of income on disability were weaker compared to the youngest cohort. There was no significant effect of making $10 to 20,000. However, those making $20,000 or more per year were 33% less likely to experience onset compared to the reference group. The effects of education on onset were mediated slightly but remained substantial for this cohort. Individuals with some high school education were 40% less likely to those having less education to experience onset. Individuals having some college education were half as likely as those with less than high school education to experience onset.

Once income was included in the model, the effects of gender and race were reduced to nonsignificance. The effects of age remained similar, with each year increasing the likelihood of onset by 13%. The effects of income on the growth of disability were nonsignificant. The positive effects of education remained even after income was included in the model, with some

high school education increasing the latent intercept of disability by .21 units compared to those with less education. Some college education increased the latent disability intercept by .37 units compare to those with less than high school education. Income mediated the effects of education on the slope of disability growth, reducing the effect to nonsignificance. In addition, the protective effect of gender was also mediated. The positive effect of race on the slope of disability was decreased slightly but remained significant, with white individuals having a .16 unit increase in disability per wave on average. Again, the effect of age remained similar, with each year increasing the slope of disability by .05 units per wave.

For the oldest cohort (1881-1901), the effects of income on the onset of disability were not significant. The effect of some college education was mediated when income was introduced into the model, reducing it to nonsignificance. However, the effect of some high school education remained significant, with these individuals having 48% less likelihood of onset. Consistent with the other two cohorts, the protective effect of gender on disability onset was mediated when income was introduced into the model and the effect of age remained the same. Income had a positive effect on initial level of disability so that individuals making $10 to 20,000 had on average .64 units increase in disability level. The effect of making $20,000 or more, however, was not significant. With the inclusion of income, the positive effect of education on the initial level of disability was reduced in magnitude and became nonsignificant. The effects of the other covariates remained nonsignificant on the latent intercept of disability and there were still no significant effects on the slope of disability.

Figure 1 presents the mean estimated hazard plots of the independent effects of education over time on the onset of disability (taken from the full model). As apparent in Tables 2 and 3, these effects on onset are strong and persistent across cohort. As is expected, the hazard of

disability onset is lower overall for younger cohorts compared to their older counterparts. It is noticeable, however, that the gap between those at the lowest levels of education (here, less than high school) increases with older cohorts compared to those of younger cohorts with higher educational attainment. The effects of income on onset are not presented here since their effects were highly significant only for the youngest (1912-1921) cohort and slightly significant in the 1902-1911 cohort. These findings on onset suggest that the proximate protective effects of income may diminish with age while the distal protective effects of education may remain more salient for disability onset until the latest ages.

- Figure 1 about here -

DISCUSSION

Disability is predominantly conceptualizied in the literature as a life course process, and previous studies have analyzed it as either an independent collection of transitions or as a trajectory of growth encompassing a baseline and slope over time or age. The goal of this paper was to measure disability both as a transition and a growth trajectory, better illustrating the life course process of disablement and better illuminating the way known predictors work on disability over time. Findings show support for both conceptualization and measurement of disability using this analytic strategy, previously absent in aging research.

Concerning the role of socioeconomic status on disability, the findings support *both* strains of thought on how education and financial resources protect individuals differentially from disability. Education was found to play a strong preventive role in delaying onset of disability. In fact, even in the final model, individuals with higher levels of education were generally 38-50% less likely to experience onset compared to those with the least education. This suggests that education's role in disablement is indeed preventive and likely works through a

number of social and behavioral mechanisms independently of income to delay disability. The independent effects of income were significantly preventive, but were consistent and robust compared to those of education on disability onset. Income worked mainly to decrease the level of disability given onset. This suggests that income may also serve as a protective role, but is primary in decreasing disability level once onset occurs through access to resources in the material world (treatment, prescriptions, etc.).

The findings on gender suggest that women are more likely to experience onset (replicating findings using data of this type) but these effects were mediated by income. In addition, given the onset of disability, women generally did not have significantly higher levels over time compared to men. This finding supports previous work by Manton (1988) and others on women's higher disability prevalence caused by duration. The findings on race were somewhat sporadic in these findings across model and cohort. This is not surprising given the known mortality crossover that exists at older ages. More analyses will be needed to disentangle the story of race.

As mentioned previously, this paper is a first step in disentangling the causal pathways involved in disability trajectories in later life. Revisiting known predictors (as was undertaken here) sheds light on the tests of life course processes that are available. Future research now underway will study these pathways using the Verbrugge and Jette (1994) model, examining chronic conditions leading to disablement along with mediating and moderating factors such as social support, lifestyle behaviors, and treatment.

The effects of cohort generally supported the hypothesis presented and previous research on cohort effects in disability declines (Manton, Stallard, & Corder, 1997). However, it should be noted that it is not possible to disentangle the specific effects of age and cohort here. Future

research should and will address the role of age, period, and cohort in a more detailed manner in light of the findings presented here. Although the analyses included all individuals who reported on variables at baseline (i.e. individuals were included in the sample until they dropped out), a detailed analysis of mortality is not presented here[7]. Mortality should be viewed as both a selective and competing force in comparison to disability in later life, therefore, future research should address transitions into mortality along with those into disability.

A notable limitation of these analyses is the time invariant nature of predictor variables. Although income was used at baseline, the categories used were roughly stable over time. Future research should include time varying predictors known to affect disability, particularly marital status and social support. Measuring these predictors at baseline would not give and adequate picture of their effects over a decade, and therefore their dynamic nature must be taken into account in the future.

[7] Analyses on selection due to mortality are available from the author.

Table 1: Coefficients (S.E's) for Demographic Predictors of Disability over Time

1881-1901	**Cohort**	**Age 85-105**		**N=279**		
	α_v	S.E.	β_v	S.E.	η_u	S.E.
Male	-0.151	(0.140)	-0.063	(0.180)	-0.468	(0.262)
White	0.108	(0.118)	0.207	(0.128)	-0.186	(0.236)
Age	0.027	(0.018)	0.000	(0.024)	0.223***	(0.053)
Intercept	-0.799	(1.572)	0.714	(2.127)	---	---
Var.	0.464***	(0.161)	0.139	(0.074)	---	---
Cov. (α, β)	0.054	(0.099)				
	Onset 86/87	Onset 89	Onset 92	Onset 96		
Threshold (τ)	18.434***	18.551***	18.417***	20.309***		
	(4.584)	(4.545)	(4.557)	(4.661)		
Loglikelihood	-916.922	(24)		BIC	1968.994	
1912-1921	**Cohort**	**Age 65-74**		**N=2,458**		
	α_v	S.E.	β_v	S.E.	η_u	S.E.
Male	0.024	(0.047)	-0.040	(0.047)	-0.198***	(0.066)
White	0.030	(0.059)	0.121***	(0.045)	-0.449***	(0.065)
Age	0.004	(0.010)	0.018***	(0.007)	0.079***	(0.011)
Intercept	1.079	(0.683)	-0.881	(0.516)	---	---
Var.	0.446***	(0.062)	0.232***	(0.052)	---	---
Cov. (α, β)	-0.097***	(0.045)				
	Onset 86/87	Onset 89	Onset 92	Onset 96		
Threshold (τ)	6.285***	6.508***	6.409***	6.080***		
	(0.781)	(0.781)	(0.780)	(0.778)		
Loglikelihood	-6608.145	(24)		BIC	13403.660	
1902-1911	**Cohort**	**Age 75-84**		**N=1,230**		
	α_v	S.E.	β_v	S.E.	η_u	S.E.
Male	0.003	(0.076)	-0.154	(0.079)	-0.220***	(0.102)
White	0.098	(0.068)	0.103	(0.062)	-0.422***	(0.090)
Age	0.011	(0.012)	0.046***	(0.012)	0.135***	(0.017)
Intercept	0.570	(0.949)	-3.050***	(0.903)	---	---
Var.	0.362***	(0.095)	0.273***	(0.068)	---	---
Cov. (α, β)	-0.006	(0.065)				
	Onset 86/87	Onset 89	Onset 92	Onset 96		
Threshold (τ)	10.672***	10.930***	10.607***	9.944***		
	(1.354)	(1.348)	(1.345)	(1.344)		
Loglikelihood	-4262.557	(24)		BIC	8659.868	

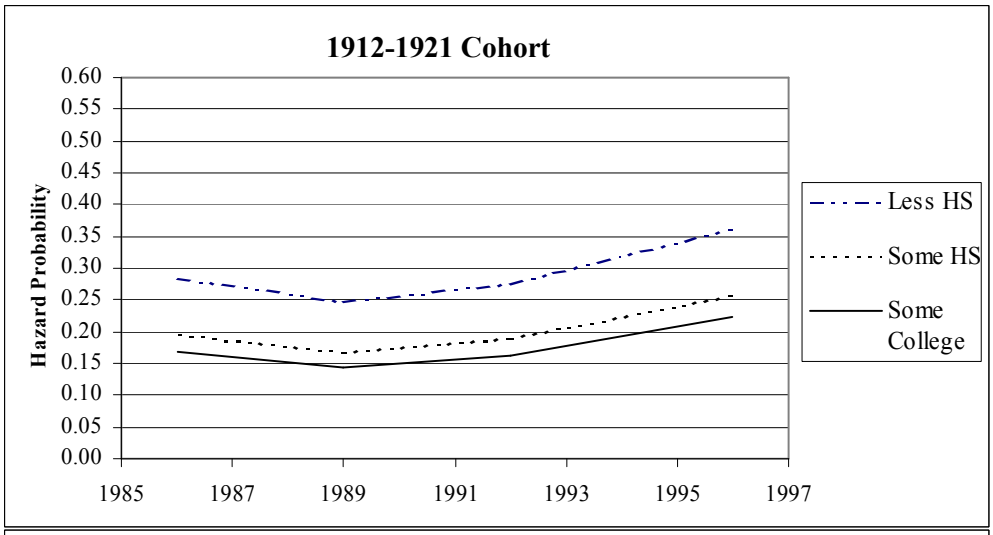
Table 2: Coefficients (S.E.'s) for Demographic Predictors and Education Effects

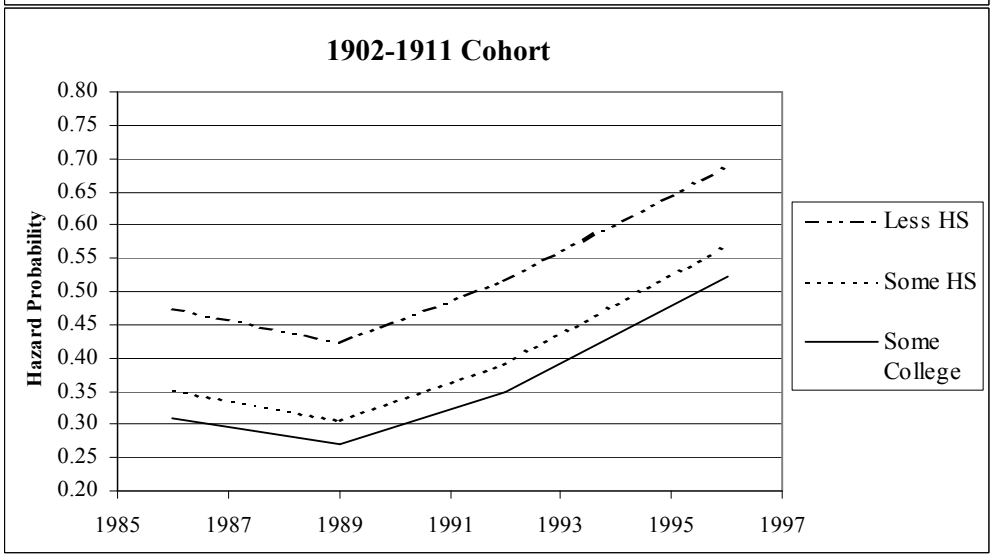
1912-1921	**Cohort**		**Age 65-74**		**N=2,458**	
	α_y	S.E.	β_y	S.E.	η_u	S.E.
Male	0.034	(0.058)	-0.049	(0.047)	-0.252***	(0.068)
White	-0.010	(0.061)	0.143***	(0.048)	-0.229***	(0.068)
Age	0.005	(0.010)	0.017***	(0.007)	0.069***	(0.011)
Educ. 9 to 12	0.093	(0.067)	-0.084	(0.049)	-0.598***	(0.073)
Educ. 13 to 17	0.243***	(0.108)	-0.076	(0.075)	-0.987***	(0.108)
Intercept	1.016	(0.682)	-0.797	(0.510)	---	---
Var.	0.433***	(0.061)	0.220***	(0.051)	---	---
Cov (α, β)	-0.086	(0.045)				
	Onset 86/8	Onset 89	Onset 92	Onset 96		
Threshold (τ)	5.351***	5.544***	5.418***	5.048***		
	(0.794)	(0.794)	(0.794)	(0.791)		
Loglikelihood	-6544.402	(30)	BIC	13323.017		
1902-1911	**Cohort**	**Age 75-84**		**N=1,230**		
	α_y	S.E.	β_y	S.E.	η_u	S.E.
Male	0.022	(0.076)	-0.155**	(0.078)	-0.239***	(0.104)
White	0.041	(0.071)	0.150***	(0.065)	-0.197***	(0.097)
Age	0.010	(0.012)	0.046***	(0.012)	0.129***	(0.017)
Educ. 9 to 12	0.180***	(0.088)	-0.110	(0.074)	-0.549***	(0.111)
Educ. 13 to 17	0.279***	(0.115)	-0.274***	(0.098)	-0.899***	(0.137)
Intercept	0.572	(0.948)	-2.986***	(0.901)	---	---
Var.	0.356***	(0.092)	0.260***	(0.065)	---	---
Cov (α, β)	0.004	(0.063)				
	Onset 86/8	Onset 89	Onset 92	Onset 96		
Threshold (τ)	10.042***	10.252***	9.883***	9.165***		
	(1.378)	(1.372)	(1.369)	(1.369)		
Loglikelihood	-4229.518	(30)		BIC	8672.185	
1881-1901	**Cohort**	**Age 85-105**		**N=279**		
	α_y	S.E.	β_y	S.E.	η_u	S.E.
Male	-0.101	(0.141)	-0.026	(0.183)	-0.602***	(0.271)
White	0.072	(0.121)	0.077	(0.143)	0.045	(0.254)
Age	0.026	(0.018)	0.004	(0.024)	0.214***	(0.053)
Educ. 9 to 12	0.326***	(0.151)	0.307	(0.171)	-0.688***	(0.291)
Educ. 13 to 17	-0.017	(0.183)	0.179	(0.203)	-0.736***	(0.344)
Intercept	-0.814	(1.558)	0.346	(2.143)	---	---
Var.	0.440***	(0.171)	0.129	(0.078)	---	---
Cov (α, β)	0.025	(0.107)				
	Onset 86/8	Onset 89	Onset 92	Onset 96		
Threshold (τ)	17.398***	17.398***	17.244***	19.069***		
	(4.597)	(4.558)	(4.571)	(4.674)		
Loglikelihood	-907.267	(30)		BIC	1983.471	

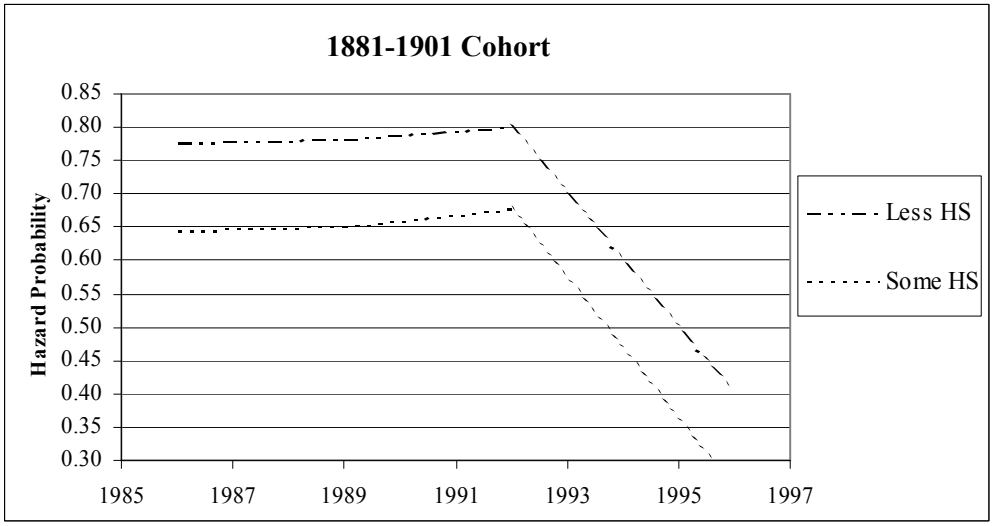
Table 3: Coefficients (S.E.'s) for Demographic Predictors and SES Effects

1912-1921	**Cohort**		**Age 65-74**		**N=2,458**	
	α_y	S.E.	β_y	S.E.	η_u	S.E.
Male	0.016	(0.060)	-0.009	(0.047)	-0.119	(0.071)
White	-0.034	(0.064)	0.193***	(0.051)	-0.083	(0.072)
Age	0.006	(0.010)	0.015***	(0.007)	0.060***	(0.012)
Educ. 9 to 12	0.069	(0.068)	-0.042	(0.049)	-0.485***	(0.075)
Educ. 13 to 17	0.217	(0.118)	0.024	(0.082)	-0.670***	(0.121)
Inc. 10 to 20	0.154	(0.088)	-0.221***	(0.067)	-0.627***	(0.092)
Inc. 20,000+	0.059	(0.118)	-0.191***	(0.086)	-0.593***	(0.123)
Intercept	0.966	(0.681)	-0.703	(0.506)	---	---
Var.	0.431***	(0.060)	0.214***	(0.048)	---	---
Cov (α, β)	-0.083	(0.043)				
	Onset 86/87	Onset 89	Onset 92	Onset 96		
Threshold (τ)	4.760***	4.942***	4.800***	4.404***		
	(0.804)	(0.804)	(0.804)	(0.802)		
Loglikelihood	-6509.671	(36)		BIC	13186.017	
1902-1911	**Cohort**	**Age 75-84**		**N=1,230**		
	α_y	S.E.	β_y	S.E.	η_u	S.E.
Male	0.046	(0.078)	-0.144	(0.079)	-0.183	(0.107)
White	0.050	(0.072)	0.164***	(0.066)	-0.148	(0.099)
Age	0.008	(0.012)	0.045***	(0.012)	0.126***	(0.018)
Educ. 9 to 12	0.201***	(0.088)	-0.094	(0.074)	-0.506***	(0.112)
Educ. 13 to 17	0.372***	(0.135)	-0.221	(0.114)	-0.694***	(0.165)
Inc. 10 to 20	-0.216	(0.121)	-0.097	(0.105)	-0.242	(0.148)
Inc. 20,000+	-0.115	(0.182)	-0.086	(0.156)	-0.399*	(0.201)
Intercept	0.737	(0.950)	-2.903***	(0.904)	---	---
Var.	0.343***	(0.092)	0.256***	(0.065)	---	---
Cov (α, β)	0.008	(0.062)				
	Onset 86/87	Onset 89	Onset 92	Onset 96		
Threshold (τ)						
	(1.382)	(1.377)	(1.373)	(1.373)		
Loglikelihood	-6509.671	(36)		BIC	13186.017	
1881-1901	**Cohort**	**Age 85-105**		**N=279**		
	α_y	S.E.	β_y	S.E.	η_u	S.E.
Male	-0.119	(0.143)	-0.013	(0.184)	-0.532	(0.278)
White	0.008	(0.123)	0.066	(0.152)	0.167	(0.269)
Age	0.027	(0.017)	0.001	(0.024)	0.215***	(0.053)
Educ. 9 to 12	0.293	(0.151)	0.262	(0.176)	-0.652***	(0.293)
Educ. 13 to 17	-0.072	(0.205)	0.218	(0.214)	-0.494	(0.385)
Inc. 10 to 20	0.637***	(0.121)	0.267	(0.290)	-0.727	(0.419)
Inc. 20,000+	-0.150	(0.359)	-0.282	(0.375)	-0.475	(0.534)
Intercept	-0.884	(1.532)	0.593	(2.147)	---	---
Var.	0.408***	(0.163)	0.129	(0.077)	---	---
Cov (α, β)	0.033	(0.103)				
	Onset 86/87	Onset 89	Onset 92	Onset 96		
Threshold (τ)	17.512***	17.476***	17.361***	19.136***		
	(4.606)	(4.567)	(4.581)	(4.684)		
Loglikelihood	-899.790	(36)		BIC	2002.304	

Figure 1: Plotted Hazard Probabilities for Education Effects by Cohort







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