Psychosocial Influences in Onset and Progression of Late Life Disability

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Objectives. Disability in older age has been related to several psychosocial characteristics, including social networks, social engagement, and depression. However, the exact role of these characteristics in the disablement process remains uncertain.

Method. Data come from a population-based study of black and white adults aged 265 years (N = 5,306), with up to 9 yearly data on the primary outcome measure, activities of daily living (ADL) disability. We use a two-part regression model to simultaneously test the association between each psychosocial characteristic and both onset and progression of ADL disability, while controlling for demographic variables, education, and mode of interview in the first model and health status variables in the second model.

Results. Social networks were negatively associated with onset of ADL disability but not associated with progression. The association became non-significant after adjustment for health status. Social engagement was negatively associated with both onset and progression of disability, even after adjustment for health status. Depression was significantly associated with onset of disability after adjustment for health status but not with progression of disability.

Discussion. The results suggest a differential role for psychosocial characteristics in the disablement process, with generally stronger associations for transitions to onset of ADL disability than progression of ADL disability.

Key Words: Depression—Disability—Social engagement—Social networks—Statistical methods.

Disability in late life is generally seen as a common outcome of the multiple, often comorbidly occurring, chronic disease processes that affect humans as they age (Manton, Corder, & Stallard, 1997; Pope & Tarlov, 1991). It represents the cumulative impact of these aging-related conditions on a person’s ability to perform basic physical functions and to maintain independence in essential activities of daily living (ADL). Extended models of the disablement process recognize the importance of several layers of “external” influences on late-life disability, including individual-level psychosocial characteristics, social and cultural influences, and environmental factors (Committee on Disability in America, 2007; Verbrugge & Jette, 1994; World Health Organization, 2001). However, despite several decades of research, considerable uncertainty remains about the exact role of some of these external influences in the disablement process. In this article, we focus on three individual-level psychosocial characteristics commonly associated with disability outcomes including social networks, social engagement, and depression and examine how they are related to different aspects of the disablement process. Following recent work in statistical modeling of population-level disability data (Haas & Rohlfsen, 2010; Taylor, 2008, 2010), we formulate a two-stage regression model that allows us to test the degree to which each of these psychosocial characteristics is associated with either onset of disability, progression of disability, or both.

Traditional disability models emphasize the progressive nature of the underlying biological processes that give rise to disabilities in late life (Nagi, 1991; Pope & Tarlov, 1991; Verbrugge & Jette, 1994; Wolf, Hunt, & Knickman, 2005). These processes involve in the earliest stages damage to a molecular and cellular level, which, if sustained and not repaired, will eventually result in active pathology in specific organ systems. Sufficient accumulation of active pathology over time will produce, through either anatomical or physiological changes, impairments in organ function, with injury being another potential cause of such impairment. Impairments in normal organ functioning will develop into specific motor and cognitive deficits that may produce functional limitations at the behavioral level. Functional limitations, in turn, will gradually interfere with the ability to perform the basic self-care and household tasks that are required for maintaining independence in the community.

This conceptualization of the disablement process has posed a number of challenges in modeling changes in disability as they occur in older populations. One important challenge is that this process involves changes in various systems, ranging from dysregulation at basic cellular and tissue levels, to the development of specific functional...
deficits, and the overt behavioral manifestations or consequences of this process that often form its ultimate endpoint. Another challenge is that the rate of progression in this process tends to vary substantially across these different systems. Dysregulation at basic biological levels likely originate many years, if not decades, before onset of older age and tends to progress at a slow rate. By the time changes in basic biological processes become apparent, they often involve the development of sufficient damage to result in clinical disease events or specific functional impairments. In contrast, overt behavioral manifestations may not become apparent until much later in the disablement process and are typically confined to just the later stages of life. A third challenge is that progression in the disablement process is not necessarily uniform; changes may be reversible and result in recovery from temporary episodes of disability.

Population studies of disability have typically emphasized a specific facet of the complex dynamics of the disablement process. For example, some studies have defined disability trajectories in terms of the accumulation and exacerbation of functional or behavioral deficits over time (Haas & Rohlfksen, 2010; Mendes de Leon, Glass, & Berkman, 2003; Taylor, 2008, 2010). This approach adheres most closely to the original disability model proposed by Nagi (1976), which underscores the continuum of deficits that sequentially affect various stages of the process, from pathologies, to impairments, to functional limitations and eventually disabilities in key social tasks and roles. It favors the definition of disability outcomes in quantitative terms in order to capture the progressive nature of the underlying process (Haas & Rohlfksen, 2010; Jette, 1999; Mendes de Leon, Bang et al., 2005; Nagi, 1976; Nusselder, Looman, & Mackenbach, 2006; Verbrugge & Jette, 1994).

Other studies emphasize the development of discrete, overt behavioral changes as manifestation of the disablement process, for example, the inability to perform basic household and self-care tasks (Hardy & Gill, 2004; Pope & Tarlov, 1991; Spillman, 2004; Wolf et al., 2005). In this work, disability is typically defined in terms of distinct transitions from a previous state of no (less) disability to a state of (more) disability, or the return to a state of no (less) disability, that is, recovery. Categorical definitions of discrete disability states have played an important role in descriptive research as a marker of overall health status in older populations (Crimmins, Hayward, Hagedorn, Saito, & Brouard, 2009; Freedman, Martin, & Schoeni, 2002; Manton, Gu, & Lamb, 2006; Seeman, Merkin, Crimmins, & Karlamangla, 2010). In addition, discrete disability states are often considered a general measure of total “morbidity” and used in calculations of population measures of health expectancies that are expressed as active life expectancy or disability-free life expectancy (Branch et al., 1991; Crimmins et al., 2009; Crimmins, Hayward, & Saito, 1996; Jagger, Arthur, Spiers, & Clarke, 2001; Land, Guralnik, & Blazer, 1994). Discrete classifications of disability have also served to inform geriatric care decisions, such as the need for home health care or admission to long-term care facilities (Fortinsky, Covinsky, Palmer, & Landefeld, 1999; Muramatsu et al., 2007; Spillman, 2004). Due to its relative simplicity and relevance for medical decisions, categorical definitions have also been used commonly to characterize the disablement process in prospective population studies, focusing on outcomes such as (new) onset of disability and recovery from disability (Ferrucci et al., 1996; Gill, Allore, & Guo, 2003).

There is an increasing recognition that a better understanding of the role of various specific determinants of the disablement process may depend on methodological approaches that capture multiple facets of the temporal dynamics of this process. A number of recent studies have taken advantage of newer statistical methods to model the complex nature of disability changes over time. For example, Zimmer and House (2003) conceptualized disability outcomes in terms of new onset as well as various forms of progression of disability over time to specify the role of education and income in disability in more detail. Liang and colleagues used a semi-parametric growth mixture model to identify distinct classes to characterize initial level and rate of change in disability severity over time. Their findings suggest three major classes of trajectories, each with varying degrees of initial level of disability and rate of progression, including a class of individuals who experience generally low levels of disability, a second class of individuals experiencing a slightly elevated initial disability level followed by moderate progression over time, and a third class of those with more severe disability levels initially and a higher rate of progression over time (Liang, Xu, Bennett, Ye, & Quinones, 2010). Another recent approach involves the use of two-stage regression models, designed to simultaneously model discrete transitions in the disablement process as well as gradual accumulations in deficits. For example, based on a life-course theoretical framework, Taylor (2008, 2010) argues that specific socioeconomic resources may differentially promote delay of disability onset or mitigate the progression of this process once it has manifested itself, that is, after first onset. Consistent with Zimmer and House’s earlier findings (Zimmer & House, 2003), she finds that education is a key preventative resource in terms of delaying onset, whereas income affects both onset and progression of disability. Haas and Rohlfksen (2010) use a variation of Taylor’s two-part regression analysis approach to examine the role of childhood and adulthood socioeconomic status in accounting for racial/ethnic differences in disability trajectories in older age.

Although these newer approaches have shown promise in further specifying the role of basic demographic and socioeconomic resources in the disablement process, they have not been applied to some of the other types of resources thought to be involved in this process. In this article, we
consider the domain of individual-level psychosocial resources, with a focus on three specific characteristics that often have been linked to late-life disability: depression, social networks, and social engagement. Our objective is to explore the degree to which the application of a two-stage regression approach to characterize changes in disability serves to refine our understanding of their role in the overall disablement process. According to prevailing disablement models, individual-level psychosocial characteristics are another type of resources that is thought to shape age-associated changes in disability (World Health Organization, 2001; Verbrugge & Jette, 1994). At the same time, these models remain mostly ambiguous about the exact nature of their influence in the disablement process.

Although subject of numerous epidemiological studies, the empirical evidence thus far also provides limited guidance in formulating more specific hypotheses regarding their differential associations with disability changes over time. For example, depression has been found to be predictive of both new onset disability and changes in severity of disability (Carriere et al., 2011; Dunlop, Manheim, Song, Lyons, & Chang, 2005; Fauth, Gerstorf, Ram, & Malmberg, 2012; Geerlings, Beekman, Deeg, Twisk, & Van Tilburg, 2001; Kivela & Pahkala, 2001; Lenze et al., 2005; Penninx, Leveille, Ferrucci, van Eijk, & Guralnik, 1999; van Gool et al., 2005; Wang, van Belle, Kukull, & Larson, 2002; Wolinsky et al., 2007). Other studies, however, have failed to find a prospective association between depression and changes in disability severity and suggest that depression is more likely the result of increases in disability rather than a predictor of such changes (Everson-Rose et al., 2005; Ormel, Rijsdijk, Sullivan, van Sonderen, & Kempen, 2002; Yang & George, 2005). Similarly, social networks and social engagement have been associated with changes in disability status although like for depression, the most consistent evidence has emerged from studies focusing on new onset of disability (Avlund, Lund, Holstein, & Due, 2004; Giles, Metcalf, Glonek, Luszcz, & Andrews, 2004; James, Boyle, Buchman, & Bennett, 2011; Lund, Nilsson, & Avlund, 2010; Mendes de Leon et al., 1999; Michael, Berkman, Colditz, & Kawachi, 2001), whereas findings for progressive changes in disability have been more variable (Mendes de Leon et al., 2003; Mendes de Leon, Gold, Glass, Kaplan, & George, 2001; Unger, Johnson, & Marks, 1997; Unger, McAvay, Bruce, Berkman, & Seeman, 1999).

In sum, our overall objective is to reexamine the role of social networks, social engagement, and depressive symptoms in relation to changes in ADL disability in older adults. To that end, we use a two-part regression method that is a modification of this analytic approach used in previous studies of disability outcomes (Haas & Rohlf, 2010; Taylor, 2008, 2010). The two-part regression method allows us to simultaneously test the prospective association of each psychosocial characteristic with new onset and progression of ADL disability. Based on previous research, we generally hypothesize that these psychosocial factors will show more consistent associations with new onset of ADL disability than with progression of ADL disability, defined as accumulation of ADL limitations after onset.

Method

Study Population

Data for this analysis come from the Chicago Health and Aging Project (CHAP). CHAP is a population-based, longitudinal study of Alzheimer’s disease and other common health conditions in older age (Bienias, Beckett, Bennett, Wilson, & Evans, 2003). Briefly, the original CHAP study population was selected from three adjacent community areas on the south side of Chicago. In 1993, the study began with a complete census of the study area, and all residents aged 65 and older were invited to participate. Of the 7,813 eligible residents, 6,158 (78.8%) agreed. The CHAP study included in-person interviews that were conducted in the participants’ homes in approximately three-year cycles, with each subsequent cycle starting immediately after completion of the previous one. The first cycle was completed by May 1997, the second by February 2000, the third by January 2003, the fourth by April 2006, and the fifth by January 2010.

As of the third interview cycle (March 2000), CHAP began to enroll newly aged participants; that is, community residents that had turned 65 years since the beginning of the study. These residents were identified through the original census and through purchase of commercially available listings as the census had become increasingly outdated over time. As of the fourth cycle, CHAP also began to enroll residents from a neighboring community area, Mount Greenwood, to achieve a more even racial balance in its study population. As of the third interview cycle, CHAP also started to add yearly interviews for all surviving participants of the original cohort and all newly enrolled participants, in order to obtain more frequent data on changes in disability status. To take advantage of these yearly disability data, the present analysis was restricted to data collected in the third and subsequent interview cycles. The CHAP study was approved by the institutional review board of Rush University Medical Center, and all participants (or their legal guardians) provided written informed consent.

Data Collection and Measures

CHAP data collection consisted of in-person interviews conducted in the participants’ homes and included standardized questions on demographic information, medical history, and a number of brief self-report instruments of disability, psychological characteristics, and social function, as well as brief performance-based tests of physical and cognitive function. The telephone interviews included the same brief self-report measures of disability that were
part of the in-person interview. The structure of the data set is as follows: for the original CHAP study participants recruited between 1993 and 1997, we used data from their third in-person interview cycle (2000–2002) as baseline source of the main predictor variables (social networks, social engagement, and depressive symptoms) and initial disability status. For participants recruited into the study during the third and subsequent CHAP interview cycles, we used their first CHAP interview as source of baseline data. Follow-up disability data come from all subsequent telephone and in-person interviews, up until January 2010 (the end of the fifth cycle of data in CHAP). For the progression of disability part of the analysis (see Statistical Analysis), we used a time-varying age variable, corresponding to age at onset of disability. We also used time-varying information for the main predictor variables of interest, derived from the last in-person interview participants completed prior to onset of disability.

In this analysis, we focus on disability in ADL as outcome. ADL disability was measured using six items assessing the ability to perform basic ADLs, such as bathing, dressing, and eating (Branch, Katz, Kniepmann, & Papsidero, 1984). Needing help with a task (from another person, special equipment, or both) or inability to perform a task was scored as a disability, and disabilities were added across tasks to create a summary measure of ADL disability (Mendes de Leon, Barnes, Bienias, Skarupski, & Evans, 2005). Social networks were assessed using a brief series on questions about the number of relationships with children, friends, and relatives a participant has and the frequency of interactions with each type of social relationship. Following procedures established previously, we added the number of children, friends, and relatives a participant reported seeing on at least a monthly basis (truncated at 30) to create a summary measure of total social networks (Barnes, Mendes de Leon, Bienias, & Evans, 2004; Mendes de Leon et al., 2001). Social engagement was assessed using four questions on social and productive activities, derived from previous work by Glass, Mendes de Leon, Marottoli, and Berkman (1999) and Glass, Mendes de Leon, Seeman, and Berkman (1997). Participants were asked how often they attended religious services, went to a museum, participated in activities or groups outside the home, and whether they currently worked a part-time or full-time job. As we have done previously with these data, we recoded each item on a 3-point ordinal frequency scale (0–2) and then summed across items to create a summary social engagement score with a range from 0 to 8, and higher scores indicating higher levels of social engagement (Barnes, Mendes de Leon, Bienias, et al., 2004; Mendes de Leon et al., 2003).

Depressive symptoms were measured using the 10-item version of the Center for Epidemiological Studies-Depression (CES-D) scale. The original CES-D includes 20 self-report items and was developed for use in epidemiologic studies in the general population (Radloff, 1977). The 10-item form, developed and tested for the Established Populations for the Epidemiologic Studies of the Elderly (EPESE) site in East Boston, was found to have acceptable reliability and cover the same four dimensions as the original 20-item CES-D (Kohout, Berkman, Evans, & Cornoni-Huntley, 1993). Following procedures established previously in these data, each item in this version was scored as the presence or absence of a symptom (yes/no), and a summary score was derived by adding up the number of positive items, yielding a range from 0 to 10 (Everson-Rose et al., 2005; Skarupski et al., 2005).

We used a series of fixed and time-varying covariates as control variables in the analyses. Fixed variables included age (centered at age 75); male sex; self-reported race, coded as Black versus non-Hispanic white; and education, coded as the number of years of formal schooling completed (centered at 12 years of education). We used fixed and time-varying information for the health-related control variables (described subsequently), which include body mass index (BMI, kg/m²), number of medical conditions, physical function summary score, and cognitive function summary score. They were assessed at each three-yearly CHAP in-person interview. BMI was computed on the basis of measured weight and self-reported height (Mendes de Leon, Hansberry, Bienias, Morris, & Evans, 2006). The number of chronic medical conditions was assessed using standardized questions about six self-reported, physician-diagnosed medical conditions, including hypertension, coronary (ischemic) heart disease, stroke, cancer, diabetes, and hip fracture. Positive responses, indicative of a prevalent condition at baseline, were added to create a summary measure of number of chronic medical conditions. Physical function was based on a summary of three physical performance tests, including timed walk, chair stand, and tandem stand, as described previously (Everson-Rose et al., 2005). Cognitive function was measured using four brief tests, including tests of immediate and delayed recall of 12 ideas contained in the East Boston Story (Albert et al., 1991), a test of perceptual speed (oral version of the Symbol Digit Modalities Test) (Smith, 1982), and the Mini-Mental State Examination as measure of global cognition (Folstein, Folstein, & McHugh, 1975). Raw scores on each test were converted to z scores and then averaged, with higher scores indicating better cognitive function (Wilson et al., 2003). We also created a time-varying indicator variable for telephone interview (in-person interview is referent category) to adjust for the mixed mode of interviews in the study (Kelley-Moore, 2006).

Statistical Analysis

Standard descriptive analyses were used for all study variables. For the main analysis, we made two modifications to our longitudinal data set to accommodate the specific objectives of this report. First, as is commonly done,
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we restricted the onset models to participants with no ADL limitations at baseline. Second, we defined onset of ADL disability on the basis of two consecutive yearly reports of ADL disability, rather than just one, and restricted progression models to participants who met this criterion of onset. This was done to minimize the impact of recovery from disability processes in our models, as they may be subject to determinants that differ from those associated with increases in disability (Clark, Stump, & Wolinsky, 1998; Gill, Robison, & Tinetti, 1997; Hardy, Dubin, Holford, & Gill, 2005; Mendes de Leon et al., 1999). Previous studies have shown high rates of recovery among older adults who report an ADL disability at a single point in time (Hardy et al., 2005; Hardy & Gill, 2004). In our data, 47% of the participants who reported new onset ADL disability during follow-up reported no ADL disability in at least one subsequent interview, whereas 21% reported no ADL disability in any subsequent interview, suggestive of temporary and complete recovery episodes, respectively. The modified definition of ADL disability onset reduced the proportion of temporary recovery episodes after onset to 15% and complete recovery episodes to 5%; all disability data following our definition of onset were retained in the progression models described subsequently, including those from participants who reported no ADL disabilities after two consecutive yearly reports of ADL disability.

Changes in disability status over time were modeled as a two-part process. Transitions to onset of disability were modeled using a binomial distribution with logit link function and accumulation of ADL disabilities using a Poisson distribution with log link function. Because of the nested, longitudinal structure of our data, we modeled person-specific random effects in each regression model, which were allowed to be correlated for the onset and progression models. For the progression model, ADL scores of non-disabled participants were set to missing until the last report of no disability before onset. Thus, progression reflects increases in ADL disability from a state of no disability, as opposed to changes in ADL disability as of the first report of ADL disability.

The statistical model was formulated as follows: let the ADL disability score for the ith subject at time j be represented by $Y_{ij}$, with time being expressed in years since baseline. Let $t_j$ denote the time since baseline at which the ith subject reported the first of at least two consecutive reports of an ADL disability score ≥1. The variable $Y_{ij}$ is defined in terms of a two-part process, $A_{ij}$ for onset of disability and $B_{ij}$ for progression of disability as follows:

$$A_{ij} = \begin{cases} 0 & \text{for } j < t_j; \\ 1 & \text{for } j = t_j; \\ \end{cases}$$

$$B_{ij} = Y_{ij} \text{ for } j \geq t_{ij-1}; \text{ missing otherwise}$$

If $A_{ij}$ is assumed to follow a binomial($p_{ij}$) distribution, and $B_{ij}$ a Poisson($\lambda_{ij}$, $k$) distribution, then the two-part model can be characterized as a generalized linear model of the form

$$\logit(a_{ij}) = X_{ij}^T\beta + u_i,$$

and

$$\log(b_{ij}) = X_{ij}^T\gamma + v_i,$$

for design matrices $X_{ij}$ for onset and progression, respectively. The random effects $u_i$ and $v_i$ follow a jointly normal distribution with a common covariance structure. The log-likelihood for the two-part model with a multivariate normal distribution for error terms is given by:

$$l(\beta, \gamma) = \sum_{i=1}^n \log \int f_i(a_i | u_i) f_i(b_i | v_i) g(u_i, v_i) \, du_i \, dv_i.$$

Maximization of the likelihood with respect to the parameters is complicated by the integration over two random effects from a jointly normal distribution. By using binomial and Poisson functional forms, and the random effects as missing data, we can use the Laplace transformation to maximize the likelihood (Pinheiro & Bates, 1995). The data were organized by yearly interview, so that regression coefficients reflect the effect of each predictor variable on change in disability status (onset or progression) at the next interview (1 year later), averaged across all follow-up interviews. Models were fitted using the SAS/STAT software PROC NLMIXED (The SAS Institute, 2005).

The associations of the psychosocial variables with onset and progression of ADL disability were tested in two sequential models. In the first model, they were tested controlling for time since baseline, age, sex, race, education, and mode of interview. In the second model, the health-related variables were added to control for health status. For the onset models, data for the psychosocial and health-related variables were obtained from the baseline interview only. This was done to maximize the duration between exposure (psychosocial status, health status) and onset, as exposure assessments more proximate to the time of onset are more likely to be affected by underlying changes in the disablement process before they become manifest in overt ADL disability. For the progression models, we used time-varying data for age at onset and time-varying data from the most recent face-to-face interview prior to onset of ADL disability for the psychosocial and health-related variables. This was done to obtain a more rigorous test of the association between psychosocial characteristics and progression of disability, as it models the psychosocial and health-related variables with data that reflect their status proximate (but prior) to onset of the progression process, rather than their status more distal to the onset of the progression process (i.e., baseline). In a secondary series of analyses, we examined the influence of attrition due to death on the results by (a) restricting the analysis to survivors only and (b) adding an additional binary control variable for death during follow-up. We also examined the influence of our definition of onset of ADL disability by refitting the primary model after defining onset on the basis of one yearly report, instead of two consecutive yearly reports, of ADL disability.
**RESULTS**

A total of 7,594 participants completed at least one in-person interview during the study period. Of these, 1,142 (15%) reported at least one ADL disability at baseline and were excluded from analysis. We further excluded participants with fewer than three non-missing disability assessments ($N = 1,134$), to have sufficient data for each participant on potential transitions to onset and progression of disability. Another 12 participants were excluded due to missing values on key predictor and demographic control variables, resulting in a final sample size of $N = 5,306$. Participants included in the analysis were at baseline on average 73.2 ($SD, 6.4$) years old; 61% were women, and 65% were African American (see Table 1). The mean number of years of formal schooling completed was 12.7 ($SD, 3.4$). They had a mean BMI of 28.4 ($SD, 5.8$), an average of 1.1 ($SD, 0.9$) medical conditions, and mean physical and cognitive function scores of 11.1 ($SD, 2.9$) and 0.40 ($SD, 0.62$), respectively. At baseline, the mean social network score was 7.3 ($SD, 6.1$), the mean social engagement score was 2.6 ($SD, 1.7$), and the mean CES-D score was 1.3 ($SD, 1.8$). These are the data summaries of the primary predictor variables in the onset models.

A total of 1,302 participants (25% of 5,306) developed new onset ADL disability during follow-up. This is the subset of the cohort whose data were used in the progression models. Their average age was 79.0 years old ($SD, 7.0$), almost 6 years older than the average age of all participants at baseline. Not surprisingly, participants with onset ADL disability also tended to have a poorer health status and psychosocial profile (see Table 1). Average BMI was lower (27.8; $SD$, 6.6), the number of medical conditions was higher (1.4; $SD$, 1.0), and both physical function (8.1; $SD$, 3.3) and cognitive function (−0.03; $SD$, 0.81) were lower among these participants. Similarly, average levels of social networks (6.5; $SD$, 5.7) and social engagement (2.0; $SD$, 1.6) were lower among participants in the progression models, whereas the average CES-D score was higher (1.8; $SD$, 2.1). There were, on average, a total of 6.2 disability assessments per participant (range, 3–9), and 63% of the total number of disability assessments ($N = 32,731$) was obtained during a telephone interview.

**Social Networks**

The first model indicates that the social networks variable has a significant negative association with ADL disability onset (coefficient = −0.018, $p < .05$), after adjusting for time since baseline, basic demographic variables, education, and mode of interview (Table 2, top half). Inspection of the other variables reveals that the risk of ADL disability onset increased over time since baseline (coefficient = 0.324; $p < .001$) and with older age (coefficient = 0.100; $p < .001$), with an additional linear increase in the age effect as a function of each year of follow-up (coefficient age by time interaction term = 0.010, $p < .001$). Men had lower risk (coefficient = −0.211, $p < .05$), and blacks had higher risk of ADL disability onset (coefficient = 0.653, $p < .001$). Converting the coefficients to estimated odds ratios (ORs), men had about 20% lower odds of ADL disability onset (OR = 0.81) relative to women, whereas the odds of developing disability were almost twice as high among blacks as among whites (OR = 1.92). Education was associated with reduced odds of ADL disability onset (coefficient = −0.083, $p < .001$), whereas telephone as mode of interview (coefficient = 0.846, $p < .001$) was associated with increased odds of ADL disability onset.

Further adjustment for health-related control variables in the second onset model (Table 2, top half) reduced the association for the social networks variable to a non-significant level (coefficient = −0.003, $p = .75$). Addition of the health-related variables also reduced the estimates for the effects of male sex and black race to non-significant levels. BMI was not associated with ADL disability onset, whereas higher number of medical conditions and lower physical and cognitive function scores were associated with increased ADL disability onset.

The social networks variable was not associated with the rate of progression in ADL disability after onset, either before or after adjustment for health-related variables (Table 2, bottom half). Some of the other variables in these

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**Table 1. Baseline Characteristics of Study Population**

<table>
<thead>
<tr>
<th>Participant data</th>
<th>Mean $(SD)/N (%)$</th>
<th>Range (minimum, maximum)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Onset of disability analysis $(N = 5,306)$</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>73.1 (6.4)</td>
<td>64.3, 101.2</td>
</tr>
<tr>
<td>Females</td>
<td>3,220 (61%)</td>
<td>—</td>
</tr>
<tr>
<td>Blacks</td>
<td>3,470 (65%)</td>
<td>—</td>
</tr>
<tr>
<td>Years of education</td>
<td>12.7 (3.4)</td>
<td>0, 30</td>
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<tr>
<td>Body mass index (BMI, kg/m$^2$)</td>
<td>28.4 (5.8)</td>
<td>9.9, 60.3</td>
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<tr>
<td>Number of medical conditions</td>
<td>11.0 (9.9)</td>
<td>0, 5</td>
</tr>
<tr>
<td>Physical function score</td>
<td>11.1 (2.9)</td>
<td>0, 15</td>
</tr>
<tr>
<td>Cognitive function score</td>
<td>0.40 (62)</td>
<td>−4.3, 1.7</td>
</tr>
<tr>
<td>Social network</td>
<td>7.3 (6.1)</td>
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</tr>
<tr>
<td>Social engagement</td>
<td>2.6 (1.7)</td>
<td>0, 8</td>
</tr>
<tr>
<td>CES-D score</td>
<td>1.3 (1.8)</td>
<td>0, 10</td>
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<tr>
<td>Progression of disability analysis $(N = 1,302)$</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>79.0 (7.0)</td>
<td>64.7, 103.5</td>
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<tr>
<td>Body mass index (BMI, kg/m$^2$)</td>
<td>27.8 (6.6)</td>
<td>9.9, 57.1</td>
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<tr>
<td>Number of medical conditions</td>
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<tr>
<td>Physical function score</td>
<td>8.1 (3.3)</td>
<td>0, 15</td>
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<tr>
<td>Cognitive function score</td>
<td>−0.03 (81)</td>
<td>−3.07, 1.45</td>
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<tr>
<td>Social network</td>
<td>6.5 (5.7)</td>
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<tr>
<td>Social engagement</td>
<td>2.0 (1.6)</td>
<td>0, 7</td>
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<tr>
<td>CES-D score</td>
<td>1.8 (2.1)</td>
<td>0, 10</td>
</tr>
</tbody>
</table>

**Follow-up data**

| Total number of interviews | 32,731 | — |
| Number of telephone interviews | 20,670 (63%) | — |
| Mean number of interviews (range) | 6.2 (3–9) | — |

*Note. CES-D = Center for Epidemiological Studies-Depression.*
### Table 2. Onset and Progression of Disability in Relation to Social Networks

<table>
<thead>
<tr>
<th></th>
<th>First model coefficient (SE)</th>
<th>Second model coefficient (SE)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Onset of disability</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Social networks</td>
<td>-0.018* (0.008)</td>
<td>-0.003 (0.008)</td>
</tr>
<tr>
<td>Time (in years since baseline)</td>
<td>0.324*** (0.030)</td>
<td>0.405*** (0.033)</td>
</tr>
<tr>
<td>Age (centered at 75)</td>
<td>0.100*** (0.010)</td>
<td>0.030*** (0.011)</td>
</tr>
<tr>
<td>Age x time</td>
<td>0.010*** (0.003)</td>
<td>0.012*** (0.003)</td>
</tr>
<tr>
<td>Male</td>
<td>-0.211* (0.087)</td>
<td>-0.123 (0.092)</td>
</tr>
<tr>
<td>Black</td>
<td>0.653*** (0.106)</td>
<td>-0.149 (0.113)</td>
</tr>
<tr>
<td>Education (centered at 12)</td>
<td>-0.083*** (0.014)</td>
<td>-0.002 (0.014)</td>
</tr>
<tr>
<td>Telephone</td>
<td>0.846*** (0.078)</td>
<td>0.885*** (0.084)</td>
</tr>
<tr>
<td>Body mass index</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number of medical conditions</td>
<td>0.437*** (0.052)</td>
<td></td>
</tr>
<tr>
<td>Physical function</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cognitive function</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Progression of disability</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Social networks</td>
<td>0.003 (0.004)</td>
<td>0.004 (0.004)</td>
</tr>
<tr>
<td>Time (in years since onset)</td>
<td>0.202*** (0.007)</td>
<td>0.207*** (0.007)</td>
</tr>
<tr>
<td>Age (centered at 75)</td>
<td>0.001 (0.007)</td>
<td>-0.021*** (0.004)</td>
</tr>
<tr>
<td>Age x time</td>
<td>0.007*** (0.001)</td>
<td>0.007*** (0.001)</td>
</tr>
<tr>
<td>Male</td>
<td>0.154*** (0.040)</td>
<td>0.129*** (0.040)</td>
</tr>
<tr>
<td>Black</td>
<td>0.059 (0.056)</td>
<td>-0.078 (0.050)</td>
</tr>
<tr>
<td>Education (centered at 12)</td>
<td>-0.003 (0.006)</td>
<td>0.007 (0.006)</td>
</tr>
<tr>
<td>Telephone</td>
<td>0.292*** (0.027)</td>
<td>0.297*** (0.028)</td>
</tr>
<tr>
<td>Body mass index</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number of medical conditions</td>
<td>-0.078*** (0.021)</td>
<td></td>
</tr>
<tr>
<td>Physical function</td>
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<tr>
<td>Cognitive function</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Variance parameters</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Onset random effect</td>
<td>1.543*** (0.158)</td>
<td>1.474*** (0.158)</td>
</tr>
<tr>
<td>Progression random effect</td>
<td>0.454*** (0.036)</td>
<td>0.435*** (0.025)</td>
</tr>
<tr>
<td>Covariance</td>
<td>0.170 (0.134)</td>
<td>-0.149* (0.074)</td>
</tr>
</tbody>
</table>

Note. ***p < .001. **p < .01. *p < .05.

models showed somewhat different associations with progression of ADL disability compared with their effect on ADL disability onset. For example, men had a higher rate of progression of ADL disability, even after controlling for health-related variables in the second model (coefficient = 0.129, p < .01). Race, education, and physical function were not significantly associated with progression in ADL disability. Somewhat unexpectedly, higher BMI levels (coefficient = -0.009, p < .01) and higher number of medical conditions (coefficient = -0.078, p < .001) were associated with slower rates of progression in ADL disability. Higher cognitive function scores were associated with a slower rate of progression (coefficient = -0.150, p < .001).

### Social Engagement

Social engagement showed more robust associations with changes in disability over time. With respect to disability onset, higher social engagement was associated with a reduced risk of developing ADL disability (coefficient = -0.259, p < .001) in the first model (Table 3, top part). This coefficient means that each standard unit (1.6) higher level of social engagement is associated with a 34% reduced odds of developing disability (OR = 0.66). Further adjustment for health-related variables in the second model reduced this association by about 45% (coefficient = -0.139, p < .001), but it remained statistically significant. The fully adjusted coefficient translates in a reduction of about 13% in the odds of developing ADL disability (OR = 0.87) per standard unit difference in social engagement scores. Higher social engagement was also associated with a slower rate of progression in ADL disability after onset (coefficient = -0.054, p < .001). This association was reduced by about 40% (coefficient = -0.033, p < .05) after adjustment for health-related control variables (Table 3, bottom part). Associations of the other variables with onset and progression of ADL disability were mostly similar to those observed in the first set of models for social networks, although there were some differences in magnitude, in particular for male sex and black race.

### Depressive Symptoms

Depressive symptoms were significantly associated with disability onset (coefficient = 0.186, p < .001), such that those with higher symptoms levels have an increased risk of developing ADL disability (Table 4, top part). The coefficient translates into a 40% increased odds (OR = 1.40) of developing ADL disability per standard unit increase in CES-D scores. Adjustment for health-related variables in the second model reduced this association by more than half (coefficient = 0.084, p < .001), but it remained statistically significant. The fully adjusted coefficient translates into an OR = 1.16 per standard unit increase in CES-D scores. In contrast, the progression models showed no association between depressive symptoms and rate of progression after onset of ADL disability before or after adjustment for health-related variables (Table 4, bottom part).

### Tests of Mutually Adjusted Associations

In the final series of models, we first examined the degree to which each psychosocial characteristic is associated with onset or progression of ADL disability independent of the other two characteristics. In Table 5, we show a summary of the results of the fully adjusted models without (first column) and with (second column) adjustment for the other two characteristics; the coefficients in the first column are identical to the ones presented for these variables in Tables 2–4. For onset of disability, the association for social networks variable remains non-significant after mutual adjustment, whereas the effects for social engagement (coefficient = -0.131, p < .001) and depressive symptoms (coefficient = 0.068, p < .01) were attenuated by about 6% and 20%, respectively. Mutual adjustment did not materially change the association for social networks or depressive symptoms in the model for progression of ADL disability. The association for social engagement was...
slightly increased (coefficient = −0.033, \(p < .001\)) relative to the model without mutual adjustment.

We next examined the potential effect of attrition due to death during follow-up on the results. When we restricted the analysis to those who survived until the end of follow-up, substantively the same pattern of results emerged, despite some minor variations in the size of the specific coefficients: social engagement and depressive symptoms were significantly associated with onset, whereas only social engagement was associated with progression (see Table 5, third column). If instead we added a binary indicator for death during follow-up as additional control variable, we again obtained a very similar pattern (data not shown): social engagement was significantly associated with both onset (coefficient = −0.101, \(p < .001\)) and progression of ADL disability (coefficient = −0.035, \(p < .01\)), and depressive symptoms only with onset (coefficient = 0.067, \(p < .01\)). Finally, we examined the effect of our definition of onset of ADL disability. In a separate analysis, we redefined onset on the basis of just one yearly report, instead of two consecutive yearly reports of ADL disability. Again, the results were mostly similar to the mutually adjusted associations in the total cohort, even if the effect of social engagement on onset was slightly attenuated (Table 5, fourth column).

**Discussion**

We investigated the prospective association between psychosocial characteristics and ADL disability changes over time in a large cohort of older adults. Our general approach was informed by a conceptual framework of the underlying disablement process that identifies two distinguishable processes, one of which involves the transition to initial onset of disability and the other the rate of disability accumulations after initial onset. The analysis itself focuses on three key psychosocial factors previously associated with late-life disability—social networks, social engagement, and depressive symptoms—and aims to more precisely define their role in the disablement process. Our findings indicate that higher social networks was associated with a reduced risk of developing ADL disability, but this effect appeared to be attributable mostly to health-related differences between those with higher and lower social networks levels. The size of an older adult’s social networks did not have any protective effect on progression of ADL disability. In contrast, higher levels of social engagement were associated with lower risk of developing ADL disability and with a lower rate of disability accumulations after onset, suggesting that social engagement represents an important protective factor in the disablement process in older age.
Depressive symptoms were associated with increased risk of new onset ADL disability but not with the rate of accumulation after onset. The observed associations for each of these characteristics were not altered substantively when considered conjointly with the other two psychosocial characteristics.

A common interpretative challenge in disability research is that the disablement model encompasses concepts and processes that are formulated and operationalized at different levels of abstraction, ranging from biological mechanisms at basic intra- and extracellular levels to behavioral consequences and adaptations in particular social environments. A biological perspective tends to emphasize the progressive nature of this process, and in general offers little substantiation for qualitatively distinct stages of development. Accordingly, evidence regarding the causal determinants
of this process is most reliably derived from studies that prospectively link putative risk factors with differences in the rate of disability accumulations or severity progressions over time. In contrast, a perspective that focuses on behavioral consequences or outcomes may characterize the disability process as a breakdown in the behavioral management of, or adaptation to underlying physical and cognitive deficits that result in the (perceived) inability to perform specific basic, self-care tasks (Fried, Young, Rubin, & Bandeen-Roche, 2001; Jette, Assmann, Rooks, Harris, & Crawford, 1998; Mendes de Leon, Guralnik, & Bandeen-Roche, 2002; Wolinsky, Miller, Andresen, Malmstrom, & Miller, 2005). In other words, gradual accumulation and progression of physical and cognitive deficits may reach a threshold at which point a person no longer is able to perform these tasks. This perspective more readily enables the definition of clearly defined, discrete stages in the disability process, leading to formulations of transitions into or recovery from a disabled state, such as impaired mobility or ADL disability (Clarke, Ailshire, Bader, Morenoff, & House, 2008; Ferrucci et al., 1996; Gill & Gahbauer, 2005; Hardy & Gill, 2004; Koster et al., 2005; Simonsick, Guralnik, Volpato, Balfour, & Fried, 2005; Visser et al., 2005). It is important to recognize that transitions to a behavioral outcome such as ADL disability may be less “discrete” in their onset than they appear. That is, these transitions may follow a period of behavioral accommodation and adaptation to progressively worsening functional limitations through, for example, performance adjustments that depend on increasingly more time and effort to complete self-care tasks, or adoption of alternative methods (Gill, Robison, & Tinetti, 1998; Hoenig et al., 2006; Verbrugge, Rennert, & Madans, 1997). The absence of detailed information on these aspects of behavioral performance may give the appearance of a discrete behavioral transition which, at its basis, is a more progressive process that gradually evolves over time.

Our findings regarding the importance of these selected psychosocial characteristics in relation to progressive changes in disability after onset appear somewhat equivocal. We found no evidence that social networks or depressive symptoms are associated with disability changes after initial onset, suggesting that these factors do not have a clear influence on the underlying progressive nature of the disability process, at least not to the extent that it is defined by accumulations in ADL limitations after first onset. Although this is consistent with some previous research (Everson-Rose et al., 2005; Mendes de Leon et al., 2001; Ormel et al., 2002), others have shown significant associations between these factors and progressive changes in disability over time (Lenze et al., 2005; Unger et al., 1999; van Gool et al., 2005; Wang et al., 2002; Wolinsky et al., 2007). Previous studies have also considered the impact of depression on progressive changes in other stages of the disability process. For example, Everson-Rose and colleagues (2005) found no significant effect of depression on changes in physical function, which is an outcome that conceptually may be seen as more proximal to the underlying biological process than ADL disability. Another study, however, found that depression accelerated progressive changes through the entire spectrum of disability development, especially in the earlier and later stages of this process (van Gool et al., 2005). In sum, although the case for a clear, protective effect of social networks on progressive changes in disability remains weak, specifying the exact role of depression across the continuum of changes in disease, functional limitations, and disability will require further investigation.

Our results regarding social engagement differed notably from those for the other two psychosocial characteristics, indicating that more active social involvement appears to slow down at least some of the mechanisms involved in the disability process. Social engagement is likely to be strongly correlated with initial health status, which may be an important confounder of this association. Adjustment for several important markers of overall health, BMI, number of medical conditions, and summary measures of physical and cognitive function may mitigate this concern to some extent. Although residual confound due to unobserved health differences at baseline remains a possibility, the statistically reliable associations suggest that this effect may have survived even more rigorous health adjustments. Social engagement could also be part of a lifestyle pattern that includes higher levels of physical activity, which is associated with changes in disability (Boyle, Buchman, Wilson, Bienias, & Bennett, 2007; Pahor et al., 2006; Simonsick et al., 1993, 2005) and could therefore be another potential confounder that needs to be considered in future studies. Another possibility is that social engagement is primarily predictive of higher recovery rates of ADL disability, which would have contributed to a slower average rate of accumulations in ADL limitations after onset. However, in an additional analysis of our data, we did not find any evidence for a higher recovery rate associated with social engagement, suggesting that the observed protective effect due to social engagement is not linked to recovery from disability. Social engagement is also protective against cognitive decline (Barnes, Mendes de Leon, Wilson, Bienias, & Evans, 2004; Bassuk, Glass, & Berkman, 1999; Fratiglioni, Paillard-Borg, & Winblad, 2004; Ghisletta, Bickel, & Lovden, 2006; Saczynski et al., 2006; Zunzunegui, Alvarado, Del Ser, & Otero, 2003) and mortality in older adults (Berkman et al., 2004; Glass et al., 1999; Kiely, Simon, Jones, & Morris, 2000; Ramsay et al., 2008; Thomas, 2012), suggesting that the ability to remain socially involved may be a critically important resource in older age.

Consistent with the general pattern in the literature, both depression (Carriere et al., 2011; Dunlop et al., 2005; Geerlings et al., 2001; Lenze et al., 2005; Penninx et al., 1999) and social engagement (James et al., 2011; Michael et al., 2001; Nilsson, Lund, & Avlund, 2008; Unger et al., 1997) were found to be predictive of transitions to onset of
ADL disability. This pattern is compatible with an interpretation that underscores the role of these factors in accelerating or postponing the breakdown in behavioral adaptation to deficits that threaten the performance of basic self-care tasks. A similar role has previously been identified for educational attainment, and these two factors may well be among the social and behavioral mechanisms through which education is suspected to prevent or postpone onset of ADL disability (House, Lantz, & Herd, 2005; Taylor, 2010; Zimmer & House, 2003). Moreover, education tends to be strongly associated with both depression and levels of social involvement (Gallo & Matthews, 2003; House et al., 2005), providing further support for the notion that education exerts its protective effect on disability onset in part through psychosocial mechanisms that include aspects of mental health and social involvement. For the purpose of this article, we did not test the degree to which these two psychosocial factors mediate the association between education and onset of ADL disability. However, our own findings regarding education were very similar to those reported previously (Haas & Rohlfsen, 2010; Taylor, 2010; Zimmer & House, 2003), showing a considerably more robust association with onset than with progression of disability. They further indicate that health status differences associated with education are likely to be another important mechanism for the protective role of education in onset of disability, as rigorous adjustment for health-related variables that included assessment of physical functions substantially reduced the effect of education on disability onset in our analysis.

The Verbrugge and Jette extension of Nagi’s original disablement model underscores the importance, whether through protective or harmful influences, of person-specific attributes, types and degree of social involvement, and factors related to the physical, social, and cultural environment in modulating this process (Verbrugge & Jette, 1994). In the context of this model, onset of disability may be conceived as the moment at which the protective net sum of these human, material, environmental, and cultural resources becomes insufficient to fend off the injurious effects of accumulating physiological and functional deficits. Our approach in this article aims to offer additional empirical validation and refinement of this model: factors exogenous to the disability process itself, such as individual-level psychosocial attributes, appear to have the potential to influence the timing, or level of severity, at which accumulating deficits in basic biological and physical functions manifest themselves in overt behavioral consequences. At the same time, they may have much less direct influence on the rate of decline in biological and physical functions that form the basis of the disability process in late life. It is worth noting that this pattern of findings has been observed in other contexts as well: for example, education is a robust protective factor for the development of Alzheimer’s disease (Evans et al., 1997; Gatz et al., 2001; Stern et al., 1994; Wilson et al., 2009) but is associated with a faster rate of cognitive decline after onset of Alzheimer’s disease (Wilson et al., 2004).

Our approach involved a number of important limitations that need to be considered for a proper interpretation of the findings. First of all, the present results are derived from an observational study design, and causal inference therefore depends on a critical consideration of both measured and unmeasured confounders. Health status is likely among the more important candidate confounding variables. Our statistical models included reasonably rigorous adjustment for various markers of health status, yet the possibility of residual confound due to unmeasured health differences associated with psychosocial characteristics cannot be excluded entirely. Clearly, intervention studies may offer much stronger evidence regarding causality, but experimental manipulation of psychosocial characteristics is not always feasible or ethical, although interventions aimed at reducing depressive symptoms may offer some opportunities in this context. In addition, there is the possibility of other omitted variables that may have biased the observed relationships in our analysis. Another important design issue is that attrition due to death or other forms of dropout during follow-up may produce biased estimates of the associations between the psychosocial measures and disability outcomes. This may be true particularly when both the main predictor variables and outcome variable are strongly predictive of mortality, as is the case in this analysis (Brock, Lemke, Branch, Evans, & Berkman, 1994; Buchman, Wilson, Bienias, & Bennett, 2009; Giles, Glonek, Luszcz, & Andrews, 2005; Glass et al., 1999; Holt-Lunstad, Smith, & Layton, 2010; Schoevers et al., 2009; Wilson, Bienias, Mendes de Leon, Evans, & Bennett, 2003). We found that the overall pattern of results remained reasonably consistent across a series of sensitivity analyses that we conducted to investigate this form of potential bias. A number of more advanced statistical methods have recently been proposed to account for selective attrition during follow-up in longitudinal studies (Chiba & VanderWeele, 2011; Weuve et al., 2012), but these have yet to be adapted for application in two-stage regression models.

A third important design issue is that our analytic methods were not designed to model recovery from disability as a separate, informative transition in disability trajectories. A high proportion of participants with new onset of ADL disability during follow-up reported no ADL disability in at least one subsequent interview, a subset of whom reported no disability in any subsequent interview. For this reason, we defined onset of ADL disability on the basis of two consecutive yearly reports of at least one ADL limitation, rather than just one. This was done to focus the progression analysis more clearly on those whose disability levels tended to increase over time, rather than on a mixture of older adults with worsening and recovered disability trajectories after onset. A sensitivity analysis of onset and progression based
on a single yearly report of ADL disability revealed a similar pattern, suggesting that our findings do not depend on our particular definition of onset of ADL disability. However, future work will be needed to incorporate recovery and other types of disability-relevant transitions, such as death or losses to follow-up, into these statistical models.

Another design issue pertains to the use of a mixed mode of interviews to ascertain disability outcomes during follow-up. Telephone interviews tended to elicit considerably higher reports of ADL disability levels than in-person interviews, suggesting a source of bias due to interview mode. A selection effect may account for these differences: participants in poorer health may have been more likely to agree to telephone than in-person interviews. However, participation rates during follow-up were uniformly high—about 85% for in-person interviews and >90% for telephone interview—suggesting that a health selection effect is likely to be minimal. Another possibility is the duration of the interview; the in-person interviews were much longer, which may have induced fatigued participants to under-report functional limitations (assessed toward the end of the interview). Although we cannot exclude this possibility, it is of note that a comparable study in older adults found exactly the opposite effect: higher ADL disability reports during in-person interviews than telephone interviews (Kelley-Moore, 2006). That finding prompted the speculation that face-to-face interviews taking place in the respondents’ home may have allowed the interviewers to develop a rapport facilitating the reporting of limitations in basic self-care tasks, which otherwise may be more difficult to acknowledge. In sum, we remain uncertain about the precise nature of the mode of interview effect, and future studies should continue to carefully evaluate such effects on the overall pattern of their data.

Another limitation is that we considered only three psychosocial characteristics in this analysis of onset and progression of disability. Previous studies have focused on other important modulators of the disability process, including race/ethnicity, education, and income (Haas, Krueger, & Rohlfsen, 2012; Haas & Rohlfsen, 2010; Taylor, 2008, 2010; Zimmer & House, 2003), but more work is needed to extend this approach to other important psychosocial modulators of the disablement process. The exact influence of each of these factors may further depend on important individual-level “structural” contexts, such as gender or race/ethnicity, and future studies should consider the intersection between demographic characteristics and psychosocial influences in the disablement process in further detail. Although mutual adjustment for one another did not change the overall pattern appreciably, it remains possible that they influence disability trajectories through more complex additive or synergistic mechanisms. Finally, the present analysis is only a first step in more detailed investigations of the disability process and its psychosocial determinants; a logical next step would be to build on these and related statistical techniques to see if we can identify subject-specific threshold levels in basic physical functions at which overt instrumental activities of daily living or ADL disabilities become apparent, such as strength, balance, fine motor skills, and basic mobility, and then test whether psychosocial characteristics predict individual differences in these threshold levels (Jette et al., 1998; van Gool et al., 2005). Another extension of this work may wish to consider medical devices and other assistive technologies that may modulate onset of overt behavioral disability manifestations as well as its rate of progression afterward (Freedman, Martin, Corman, Agree, & Schoeni, 2009).

Despite these limitations, our approach may offer new opportunities to advance our understanding of the role of psychosocial factors in disability trajectories defined by accumulation and progression of deficits, as well as transitions between discrete states of disability. Although our findings suggest that, with the possible exception of social engagement, psychosocial characteristics may not directly affect disability trajectories if conceptualized as a progressive process, consideration of these resources may still offer substantive opportunities for the prevention and postponement of disability outcomes that commonly affect humans in late life.

**Funding**

This research was supported by grants from the National Institute of Environmental Health Sciences (ES 10902) and the National Institute on Aging (AG 11101, AG 032247).

**Acknowledgments**

C. F. Mendes de Leon conceived the theoretical framework and specific hypotheses. He also took primary responsibility for the writing of the article. K. B. Rajan developed the analytic models and conducted all statistical analyses. He wrote the section on the statistical analysis plan and contributed to the interpretation of the findings. He also provided critical feedback on all versions of the manuscript. We thank George Dombrowski and Todd Beck for their assistance in data management and data analysis and Jennifer Tarpey, project coordinator, and all field staff of the Chicago Health and Aging Project. We also thank the reviewers for their excellent feedback and comments on earlier versions of this article.

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**References**


of social interactions and physical activity. *Annals of Behavioral Medicine, 19*, 152–160. doi:10.1007/BF02833332


