

Trajectories of Impairment, Social Support, and Depressive Symptoms in Later Life

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Objectives. Research has increasingly focused on the dynamic nature of disability and depressive symptoms in later life. Little research, however, has modeled disability and depressive symptoms as dynamic, related processes. Furthermore, virtually no research has considered social support as dynamic across age. Here, we investigate the relationship between long-term patterns of disability, perceived and received social support, and depressive symptoms in later life.

Methods. We use random coefficient (growth) models of four waves of Duke Established Populations for Epidemiologic Studies of the Elderly data to determine whether disability, support, and depressive symptoms follow linear trajectories across age and whether support mediates the relationship between disability and depressive symptoms.

Results. The results show that (a) trajectories of disability are strongly related to trajectories of depressive symptoms and (b) trajectories of perceived support mediate the relationship between trajectories of disability and depressive symptoms, whereas trajectories of received support do not.

Discussion. Disability, social support, and depressive symptoms are strongly interrelated processes in later life. Our results are consistent with previous research in showing that perceived, rather than received, support mediates the relationship between disability and depressive symptoms, but our results extend previous research in showing that this mediation occurs across time.

ARE long-term patterns of disability related to long-term patterns of depressive symptoms in later life? Can social support be viewed as a process in later life that emerges in response to disability, and, if so, what dimensions of social support (if any) mediate the relationship between disability patterns and depressive symptom patterns? In previous research, disability has increasingly been conceptualized as a chronic stressor affecting individuals persistently over time. For example, contemporary research on the disablement process emphasizes disability as a trajectory of experience over time rather than a collection of static events (Verbrugge & Jette, 1994). Paralleling this recent interest in considering disability as a dynamic process across age, research on depressive symptoms has increasingly discussed life course patterns of symptomatology (e.g., Mirowsky & Ross, 1992). In contrast, social support has yet to be considered a long-term process at the individual level. Yet conceiving of disability as a chronic stressor and social support as a resource that may be mobilized in the face of such a stressor suggests that we should consider social support as patterned in later life.

Drawing on stress theory, we argue that conceptualizing disability, depressive symptoms, and social support as life course processes and measuring them as long-term trajectories can elucidate the way they interrelate over time. In treating disability as a long-term trajectory, we offer an alternative approach to estimating exposure to this chronic stressor and its consequences. That is, the majority of previous research on the deleterious effects of disability has been cross-sectional or short-term longitudinal in design, limiting the ability to understand the process of disability and its interrelationship to other health and psychosocial life course processes. In this

research, we test the mediating effects of long-term patterns of social support rather than the immediate and limited effect of support as a static phenomenon, extending the way mediating effects are tested for chronic stressors.

Disability, Depressive Symptoms, and Social Support

This research is guided by the conceptual outline of the stress process (Pearlin, Menaghan, Lieberman, & Mullan, 1981), which consists of three main components: sources, mediators, and outcomes. This framework posits that stress may impact an individual directly as a life course event or as a chronic strain over time. Also central to this framework is the protective role of mediators such as social support in limiting the impact of stressors on psychological outcomes. Disability has been considered a chronic stressor that is persistent and linked to negative psychological outcomes (Turner & Noh, 1988). Disability *onset* may cause a period of adjustment or disruption, promoting feelings of worthlessness or hopelessness that may fuel depressive symptoms (Boerner, 2004; Bruce, 2001). As a *chronic* stressor, disability produces limitation in the ability to perform social roles and may interfere with social interaction for older individuals (Blazer, 1982; Lowenthal & Haven, 1968; Thompson & Heller, 1990), and its psychological consequences may endure over time. At the same time, it may threaten an individual's ability to live alone and decrease a sense of independence or mastery, which is known to be particularly important for the continued psychological well-being of older adults (Gignac, Cott, & Badley, 2000; Jang, Haley, Small, & Mortimer, 2002; Worobey & Angel, 1990).

Much of disability research has focused on heterogeneity in disability among the elderly and the short-term dynamic nature

of the disability experience (Beckett et al., 1996; Crimmins & Saito, 1993; Maddox, Clark, & Steinhauser, 1994; Manton, 1988). Yet only a handful of studies have *modeled* disability as individual-level trajectories (e.g., Li, Duncan, McAuley, Harmer, & Smolkowski, 2000; Maddox & Clark, 1992; Verbrugge, Reoma, & Gruber-Baldini, 1994). These studies reveal a trend toward treating disability as a trajectory rather than as a single transition or as a static circumstance, reflecting the conceptualization of disability as a process that unfolds in later life.

At the same time, research examining the relationship between disability and depression/depressive symptoms has remained relatively static in design but has established a strong association between disability and depression/depressive symptoms in older adults (Bruce, 2001). Whereas healthy, functional elders have no greater risk of depression than those at younger ages, those with functional impairment have a significantly greater risk (Berkman et al., 1986; Roberts, Kaplan, Shema, & Strawbridge, 1997). Research has shown that the deleterious effects of disability on depressive symptoms are greatest for adults over age 65 (Turner & Noh, 1988) and that disability and poor health account for a majority of depressive symptom incidence among older adults (Kennedy, Kelman, & Thomas, 1990). These findings suggest that disability is one of the most salient factors for the psychological well-being of older adults, yet little is known about how long-term patterning of disability relates to long-term patterning in psychological well-being. The connection between disability and depressive symptoms is somewhat reciprocal (Bruce, 2001; Graney, 2000); however, although depressive symptoms have been shown to predict disability onset (Penninx, Leveille, Ferrucci, van Eijk, & Guralnik, 1999), others have found that change in disability has a stronger effect on depressive symptoms than symptoms has on disability (Ormel, Rysdyk, Sullivan, van Sonderen, & Kempen, 2002).

Personal and social resources have received a great deal of attention for their roles in the stress process in general and specifically the disablement process. Among the most salient and robust of these is social support. Social support has been found to protect individuals from the effects of disability on mental health (Jang et al., 2002). Multiple dimensions of social support have been studied to determine which facets of support provide protective effects for physical and mental health (Glass & Maddox, 1992; Mendes de Leon, Glass, Beckett, Seeman, Evans, & Berkman, 1999; Strawbridge, Cohen, Shema, & Kaplan, 1996).

In this study, we focus on perceived and received support. Perceived support has been found to be highly protective against the effects of stress (Cohen & Wills, 1985; Kessler & McLeod, 1985; Wethington & Kessler, 1986), especially chronic stress (Krause, 1986). In regard to disability, perceived support has been shown to attenuate the effect of disability on depressive symptoms (Allen, Ciambone, & Welch, 2000; Jang et al., 2002; Turner & Noh, 1988). The protective effects of received support, however, remain in question. Despite the existence of some evidence showing received support to be protective against the effects of general stressors for older adults (Krause, 1986), received support has also been found to have no ameliorative effects in the face of disability (Jang et al., 2002). In addition, increased received support has been associated with

increased disability (Mendes de Leon, Gold, Glass, Kaplan, & George, 2001; Seeman, Bruce, & McAvay, 1996). Overall, the role of received support in the connection between disability and depression remains relatively unclear.

Krause (1997) provides a theoretical explanation for the conflicting findings across dimensions of social support. More salient than a general measure of perceived support (which may just measure relationship satisfaction), anticipated support may be the key element for buffering the effects of a stressor on negative psychological outcomes. Anticipated support may act to promote feelings of autonomy (mastery) or personal problem solving since belief in a personal network ready to react in the face of stress may promote personal problem solving (Pierce, Sarason, & Sarason, 1996; Wethington and Kessler, 1986). In addition, the belief that others are ready to help when needed may decrease the strain put on network relationships. In other words, this belief may decrease the burden felt by support networks and increase or maintain feelings of independence in the individual. Conversely, received support may exacerbate the effects of a stressor. This may be due to the fact that personal resources may not be sufficient to cope with the stressor, therefore increasing feelings of dependence or powerlessness (Gore, 1979; Wheaton, 1985). In addition, received support may lead to conflict with one's network due to demands placed on their time and resources (Rook, 1984).

In this study, we do not attempt to disentangle the social processes involved in support and its protective effects. Rather, we attempt to measure the connection between disability and depressive symptoms, and the effects of social support in this connection, as life course processes. Thus, we extend previous research in two ways. First, we determine the connection of individual trajectories of disability and depression over a decade in the lives of older adults. Most research to date either has used cross-sectional data to examine this relationship or has used short-term longitudinal data and cross-lagged (or related) models. Our approach treats disability and depressive symptoms (well-being) as life course processes that are connected throughout the later stage(s) of life. This approach is important because although disability and depressive symptoms may or may not be strongly related at any single point in time, life course patterns in them may well be very strongly interrelated.

Second, we alternately introduce trajectories of perceived and received social support to examine the longitudinal effects of these protective factors. To date, no research has examined trajectories of support in later life. In addition, most research testing the mediating effects of support has been cross sectional or two-wave longitudinal in design. These types of analytic strategies cannot illustrate how the protective effects of social support work function over long periods of time in response to change in disability. We believe that this may account somewhat for the conflicting results concerning both the effects of received support and the type of protective effects perceived support offer.

We hypothesize that disability and depressive symptoms will generally increase across age but will exhibit marked variation across individuals, as suggested by previous research (Li et al., 2000; Lynch & George, 2002). Furthermore, we hypothesize that growth patterns in disability will be strongly associated with growth patterns in depressive symptoms, meaning that individuals with increasing trajectories of disability will also be more likely to have increasing trajectories of depressive

Table 1. Descriptive Statistics of Variables from the Duke EPESE Sample, Including Percentages or Means, Standard Deviations, and Ranges

Variable	Wave 1	Wave 2	Wave 3	Wave 4
Age (y)	73.2 (6.4) [64, 101]	—	—	—
Male (%)	34.8	—	—	—
Female (%)	65.2	—	—	—
Black (%)	53.7	—	—	—
Non-Black (%)	46.3	—	—	—
Married (%)	39.0	—	—	—
Not married (%)	61.0	—	—	—
Education (y)	8.6 (4.1) [0, 17]	—	—	—
Disability	0.93 (1.9) [0, 12]	1.1 (2.2) [0, 12]	1.4 (2.4) [0, 12]	1.7 (2.8) [0, 12]
Depression	1.5 (1.8) [0, 9]	1.3 (1.8) [0, 9]	1.4 (1.8) [0, 9]	1.5 (1.8) [0, 9]
Perceived support	5.5 (0.9) [2, 6]	5.4 (1.0) [2, 6]	5.6 (0.9) [2, 6]	5.6 (0.9) [2, 6]
Received support	8.8 (2.7) [0, 12]	9.0 (2.7) [0, 12]	9.3 (2.6) [0, 12]	9.1 (2.6) [0, 12]
<i>N</i>	3,876 ^a	3,039	2,245	1,475

^aSample size for time-varying measures at baseline was 3,870.

Notes: Standard deviations are in parentheses and range in brackets. EPESE = Established Population for Epidemiologic Studies of the Elderly.

symptoms, whereas individuals with decreasing trajectories of disability will have declining trajectories of depressive symptoms. Finally, we hypothesize that social support trajectories will mediate the connection between disablement and distress over time (Turner & Noh, 1988). We expect the effect of perceived support to be stronger than the effect of received support, as suggested by previous literature (Krause, 1997).

METHODS

Sample and Variables

The data for this study are from the Established Populations for Epidemiologic Studies of the Elderly (EPESE) at Duke University. The EPESE Project is a National Institute on Aging–sponsored multicenter research program geared to investigate the health of persons ages 65 or older at baseline. The data include eight periods of measurement, four of which were in-person interviews and four of which were telephone interviews. We use only the in-person data, given that certain key measures were not collected via the telephone interviews. The baseline survey was conducted in 1986–87, with follow-up in-person data collected in 1990, 1993, and 1996.

At baseline, the survey consisted of 4,162 respondents. Of these, 162 were not directly interviewed and were not used here. Of the 4,000 remaining, 124 persons did not have a valid measure for disability status at any wave or for at least one of the social support measures. After deleting observations without valid data, the analytic sample consisted of 3,876 persons (6.9% missing). We include age, sex (male = 1), race (Black = 1), baseline marital status (married = 1), and years of formal education as covariates. Income is not included in the analyses because (a) over 25% of the sample was missing on the income item at baseline, (b) income is unstable in later adulthood (whereas education is generally stable), and (c) the causal direction of the relationship between income and health is unclear, especially in late life. No individuals were missing on these background variables.

We measure disability at each wave as a sum of seven activities of daily living (ADL; Katz et al., 1963) and seven instrumental ADL (IADL; Lawton & Brody, 1969) measures. ADL items included whether the respondent needed help to (a)

walk across a small room, (b) bathe, (c) groom him/herself, (d) dress him/herself, (e) eat, (f) get from a bed to a chair, and (g) use the toilet. The IADL items included whether the respondent could (a) use the telephone, (b) drive a car, (c) shop for groceries, (d) prepare meals, (e) do housework, (f) take medicine, and (g) handle money without help. The sum of these items has been shown to be effective in capturing a full range of disability (Spector & Fleishman, 1998).

We measure depressive symptoms at each wave using the Center for Epidemiologic Studies—Depression (CES-D) Scale, a well-known and highly reliable scale (Radloff, 1977; Cronbach α across the four waves: .83, .87, .88, .89). However, recent research suggests that certain items from the CES-D Scale may be biased by physical impairment (Grayson, Mackinnon, Jorm, Creasey, & Brae, 2000); nonetheless, results obtained using a reduced version of the scale did not differ substantially from those we present here.

We measured perceived and received social support at each wave. The perceived support measure was the sum of two items: one asking whether the respondents could count on at least some family or friends for support in times of trouble and the other asking whether the respondents had at least some family or friends with whom they could discuss their problems. The received support measure was the sum of 12 items regarding whether the respondent had received support during the last year from family or friends for a variety of issues. Descriptive statistics for all variables used in the analyses at all waves can be found in Table 1.

Analytic Strategy

We use random coefficients (growth) models to capture disability, depressive symptoms, and social support trajectories across age. In a growth model, time-specific (t) individual level (i) measures (y_{it}) are viewed as a composite of the individual's latent growth process and normally distributed random error. If the latent growth process is assumed to be linear, then two latent parameters define the process. Thus,

$$y_{it} \sim N(\alpha_i + \beta_{it}, \sigma^2).$$

This specification defines the random error as normally distributed with a time-constant variance of σ^2 . The mean of

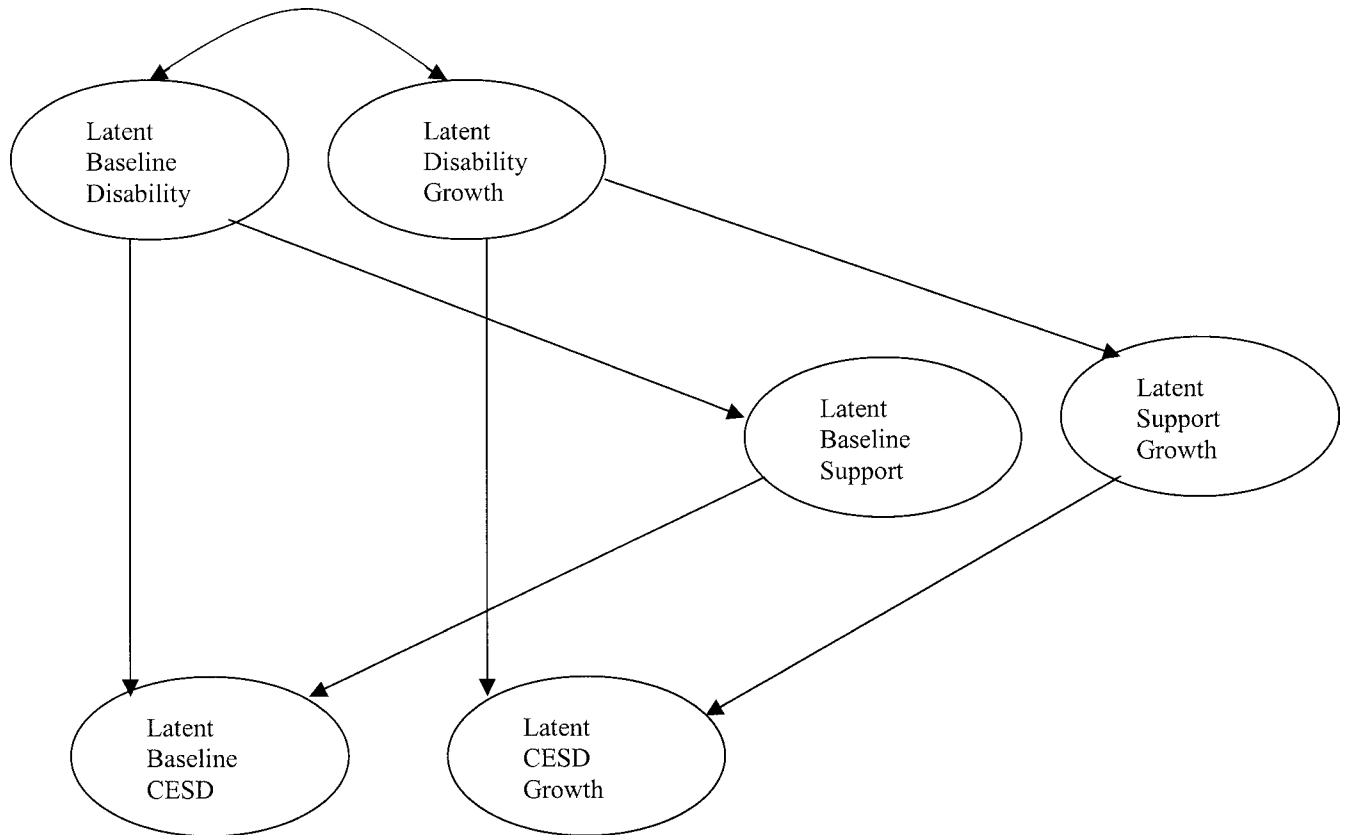


Figure 1. Conceptual diagram of disability, social support, and depressive symptom random effects model. CESD = Center for Epidemiologic Studies—Depression scale.

the error is 0, but the expected value for the structural aspect of the model is a function of an individual-specific random intercept (α_i) and slope (β_i) and time (t). This level equation separates the time-specific measures into a structural component (the trajectory) and random variation around the trajectory (captured by σ^2). A second-level equation allows for the inclusion of covariates to differentiate individuals' trajectories:

$$\alpha_i \sim N(X_i\gamma, \tau_\alpha)$$

$$\beta_i \sim N(Z_i\delta, \tau_\beta).$$

In this specification, a different set of covariates may explain latent baseline and growth factors (hence X and Z differ), and their effects are expected to vary as well (hence γ and δ differ). Unexplained between-individual variance in trajectories is captured by τ_α and τ_β , thus enabling us to distinguish between error at the individual level and heterogeneity at the between-individual level. Thus, this model is an ideal technique for analyzing panel data because, unlike standard regression approaches, it is able to distinguish between change that occurs at the individual level and heterogeneity that is attributable to between-individual differences.

Our first model estimates trajectories of disability and depressive symptoms. Included in the X and Z covariate vectors for the depressive symptom trajectory are the random effects from the disability trajectory. Our second and third models include a trajectory of social support (received and perceived

support, respectively) as a mediator between disability and depressive symptom trajectories. Thus, these models include an influence of the random effects of disability on the random effects for support and depressive symptoms, and the depressive symptom trajectory also includes the random effects for the support trajectories as predictors. Figure 1 provides a conceptual diagram of the model.

Over the course of the study, a number of individuals died or otherwise left the sample. Some of those who attrited at earlier waves returned at later waves. In order to compensate for the missing data without deleting observations with any missing data, we estimated our models using a full-information maximum likelihood estimator within a structural equation modeling framework (Mplus; Muthen and Muthen, 1998; Willett and Sayer, 1994).

RESULTS

Table 2 shows the results of the random effects models with trajectories of disability predicting trajectories of depressive symptoms. The top portion of the table shows these effects along with the effects of the background variables, and the bottom portion of the table shows the explained variance for each of the time-specific measures.

Regarding overall model fit, we report two measures: the model χ^2 and the root mean squared error of approximation (RMSEA). For a well-fitting model, the χ^2 should be nonsignificant (indicating little discrepancy between the

Table 2. Growth Models of the Influence of Disability Trajectories on Depressive Symptom Trajectories

Covariates	Disability		Depressive Symptoms	
	Intercept (α)	Slope (β)	Intercept (α)	Slope (β)
Intercept	-4.0 (.39)***	-.79 (.09)***	2.24 (.34)***	.13 (.07) [#]
Age	.07 (.01)***	.02 (.001)***	-.001 (.004)	-.002 (.001)**
Male	-.03 (.07)	-.03 (.02)*	-.32 (.06)***	.01 (.01)
Black	.02 (.06)	.004 (.01)	-.02 (.06)	.01 (.01)
Education	-.06 (.01)***	-.01 (.002)***	-.05 (.01)***	.001 (.001)
Married	-.04 (.07)	-.03 (.02) [#]	-.26 (.06)***	.03 (.01)**
Disability I.	—	—	.22 (.02)***	—
Disability S.	—	—	—	.14 (.02)***
R^2 (latent)	.11	.17	.19	.18
R^2 (observed)				
R^2 Time 1	.79		.44	
R^2 Time 2	.73		.45	
R^2 Time 3	.80		.46	
R^2 Time 4	.78		.51	
χ^2	356.32 (45)***			
RMSEA	.042			

[#] $p < .1$, * $p < .05$, ** $p < .01$, *** $p < .001$, $n = 3,876$.

Notes: I. = intercept; S. = slope; RMSEA = root mean squared error of approximation.

observed and predicted data)—although larger sample sizes tend to produce significant χ^2 values despite adequate fit—and the RMSEA should be $<.1$ (and preferably $<.05$; Bollen, 1989). In the first model, we observe a significant χ^2 but an RMSEA of .042, indicating good fit.

In the second level of the model, age has a positive effect on latent baseline disability, as is expected. Men and married persons evidence slightly less baseline disability than women and the unmarried, whereas Blacks show slightly more baseline disability than Whites, although none of these differences are significant. Years of education has a strong negative effect on baseline disability. In terms of the latent growth rates (the slope) for disability, age increases the rate of growth in disability symptoms, indicating either nonlinear growth in disability across age or cohort differences in the rate of growth in disability. Men have significantly slower growth in disability across age ($\delta = -.03$, $p < .05$), as do married persons ($\delta = -.03$, $p < .1$). Blacks are predicted to have higher growth rates in disability, although the coefficient for Black is not significant. Finally, education reduces the growth rate significantly ($\delta = -.01$, $p < .001$). The covariates as a whole explain 11% of the variance in latent baseline disability and 17% of the latent growth in disability across the survey period, and the explained variances for each observed measure of disability are quite high (ranging from 73% at Time 2 to 80% at Time 3), indicating a good fit of the linear growth specification.

The depressive symptom portion of the model shows that age has a nonsignificant effect on baseline depressive symptoms, controlling for other factors. Males have significantly fewer depressive symptoms at baseline, as do married persons. Blacks have fewer symptoms at baseline as well, although the race difference is not significant. Education has a strong negative effect. Finally, latent baseline disability has a significant and strong effect on baseline depressive symptoms ($\gamma = .22$, $p < .001$). This coefficient implies that a 1-unit increase in baseline level of disability translates into a .22-symptom difference in baseline symptoms.

Relatively few variables affect the growth rate in depressive symptoms. Only age, marital status, and disability growth have an effect, with age's effect being negative (indicating either a slowing of the rate of increase in depressive symptoms across age or cohort differences in the rate of symptom increase) and marital status and disability growth both increasing the growth rate in symptoms. The effect of marriage is fairly large but is overshadowed by the effect of disability ($\delta = .14$, $p < .001$). The coefficient for disability growth implies that a 1-unit increase in the growth rate for disability yields a .14-unit increase in the growth rate for depressive symptoms. As a whole, the covariates (including the latent trajectory parameters for disability) explain 19% of the variance in latent baseline depressive symptoms and 18% of the variance in latent growth in symptoms, and the explained variance in the observed measures is quite large (ranging from 44% at Time 1 to 51% at Time 2). Disability trajectories account for a large proportion of this explained variance; without disability trajectories, the covariates alone explain 10% and 3% of the variance in latent baseline symptoms and growth in symptoms, respectively (results not reported in table).

Tables 3 and 4 show the results of adding social support trajectories as mediators of the disability–depressive symptom relationship. Table 3 contains the results of including trajectories of received support, whereas Table 4 contains the results of including trajectories of perceived support. In both tables, the coefficients for predicting disability trajectories change very little over those results presented in Table 2. This is to be expected because we have not added any additional information to help explain disability patterns.

In terms of received support trajectories, men report receiving less support at baseline, and education also reduces received baseline support. All other covariates have a positive effect. Blacks report receiving more support, as do married persons. Finally, latent baseline disability has a strong positive effect on baseline received support, as may be expected: Those who need more support tend to receive it. In terms of the

Table 3. Growth Models of the Influence of Disability and Received Support Trajectories on Depressive Symptom Trajectories

Covariates	Disability		Depressive Symptoms		Received Support	
	Intercept (α)	Slope (β)	Intercept (α)	Slope (β)	Intercept (α)	Slope (β)
Intercept	-4.0 (.39)***	-.79(.09)***	2.15 (.38)***	.14 (.07) [#]	8.35 (.53)***	-.12 (.11)
Age	.07 (.005)***	.02 (.001)***	-.01(.004)	-.003 (.001)**	.003 (.01)	.002 (.001)***
Male	-.03 (.07)	-.03 (.02)*	-.31(.06)***	.001 (.01)	-.76 (.09)***	.05 (.02)*
Black	.02 (.06)	.004 (.01)	-.02 (.01)	.007 (.01)	.28 (.08)***	.003 (.02)
Education	-.06 (.01)***	-.01 (.002)***	-.05 (.01)***	.001 (.001)	-.02 (.01) [#]	.001 (.002)
Married	-.04 (.07)	-.03 (.02) [#]	-.26 (.06)***	.03 (.01)**	.76 (.09)***	-.04 (.02)*
Disability I.	—	—	.21 (.02)***	—	.28 (.03)***	—
Disability S.	—	—	—	.13 (.03)***	—	.12 (.04)***
Support I.	—	—	.01 (.02)	—	—	—
Support S.	—	—	—	0.08 (.12)	—	—
R ² latent	.11	.17	.19	.18	.14	.16
R ² (observed)						
R ² Time 1	.79		.45		.38	
R ² Time 2	.72		.45		.42	
R ² Time 3	.80		.46		.45	
R ² Time 4	.78		.51		.51	
χ^2	523.09 (89)***					
RMSEA	.035					

[#] $p < .1$, * $p < .05$, ** $p < .01$, *** $p < .001$, $n = 3,876$.

Notes: I. = intercept; S. = slope; RMSEA = root mean squared error of approximation.

growth rate in support, men have slightly greater growth rates in received support, whereas married persons report slightly lower growth rates in received support than unmarried persons. Finally, disability growth has a significant and strong effect on growth in received support. Overall, 14% of the variance in latent baseline received support is explained by the covariates, whereas 16% of the variance in growth in received support is explained. Furthermore, the explained variance for received support is fairly high—ranging from 38% at Time 1 to 51% at Time 4—indicating good fit of the linear specification for growth in support.

For the sake of brevity, we do not discuss the effects of all background covariates on depressive symptom trajectories; rather, we concentrate only on the effects of disability and received support. Received support has positive but non-significant effects on both latent baseline depressive symptoms ($\gamma = .01, p > .05$) and growth in depressive symptoms ($\delta = .08, p > .05$), indicating that received support has little to do with depressive symptom trajectories. The effect of support also has relatively little effect on reducing the relationship between disability and depressive symptom trajectories. The effect of latent baseline disability on latent baseline depressive symp-

Table 4. Growth Models of the Influence of Disability and Perceived Support Trajectories on Depressive Symptom Trajectories

Covariates	Disability		Depressive Symptoms		Perceived Support	
	Intercept (α)	Slope (β)	Intercept (α)	Slope (β)	Intercept (α)	Slope (β)
Intercept	-3.99 (.39)***	-.79 (.09)***	7.62 (.55)***	.20 (.10)*	5.46 (.18)***	.04 (.04)
Age	.07 (.01)***	.02 (.001)***	-.004 (.005)	-.003 (.001)**	0 (.002)	0 (.001)
Male	-.03 (.07)	-.03 (.02)*	-.46 (.06)***	-.01 (.02)	-.14 (.03)***	-.01 (.01) [#]
Black	.02 (.06)	.004 (.01)	-.18 (.06)**	.02 (.02)	-.16 (.03)***	.01 (.01) [#]
Education	-.06 (.01)***	-.01 (.002)***	-.03 (.01)***	.001 (.002)	.02 (.004)***	0 (.001)
Married	-.04 (.07)	-.03 (.02) [#]	-.22 (.06)***	.02 (.01)	.04 (.03)	-.002 (.01)
Disability I.	—	—	.18 (.02)***	—	-.03 (.01)***	—
Disability S.	—	—	—	.06 (.05)	—	-.05 (.01)***
Support I.	—	—	-.99 (.08)***	—	—	—
Support S.	—	—	—	-1.70 (.82)*	—	—
R ² (latent)	.11	.17	.35	.51	.11	.22
R ² (observed)						
R ² Time 1	.79		.44		.29	
R ² Time 2	.73		.44		.28	
R ² Time 3	.80		.46		.36	
R ² Time 4	.78		.51		.38	
χ^2	502.0 (89)***					
RMSEA	.035					

[#] $p < .1$, * $p < .05$, ** $p < .01$, *** $p < .001$, $n = 3,876$.

Notes: I. = intercept; S. = slope; RMSEA = root mean squared error of approximation.

toms is .21 and remains significant ($p < .001$), and the effect of disability growth on depressive symptom growth also remains large and significant ($\delta = .13, p < .001$). Thus, compared with the results in the previous table, the addition of received support only slightly decreases the effect of disability trajectories on depressive symptom trajectories. More to the point, the explained variance in both of the latent parameters for depressive symptom trajectories is unaffected.

The results for perceived support presented in Table 4 reveal a different story. Latent baseline disability has a negative effect on latent baseline perceived support ($\gamma = -.03, p < .001$), and latent disability growth has a negative effect on latent growth in perceived support ($\delta = -.05, p < .001$). Latent baseline perceived support has a strong negative effect on baseline depressive symptoms ($\gamma = -.99, p < .001$), and the effect of growth in perceived support on depressive symptom growth is negative and even stronger ($\delta = -1.70, p < .001$). In this model, the direct effect of baseline disability on baseline depressive symptoms remains significant ($\gamma = .18, p < .001$) but is reduced by 18% (1 - [.18/.22]). However, the effect of disability growth is reduced to nonsignificance. These results indicate that trajectories of perceived support completely mediate the effect of disability growth on depressive symptom growth, whereas the negative relationship between disability trajectories and perceived support trajectories indicates that individuals may feel that support is inadequate to help them cope with their levels of disability.

The increase in the explained variance for depressive symptoms from the first model (disability and depressive symptoms only) to the model with received support included tells a similar story. When latent baseline perceived support is included (see Table 4), the explained variance for latent baseline depressive symptoms increases from .19 to .35 (an increase of 84%). In terms of latent symptom growth, the explained variance increases from 19% to 51% (an increase of 168%). These results also support the perspective that perceived support accounts for variation in trajectories of depressive symptoms, whereas the results from Table 3 indicate that received support does not.

DISCUSSION

To date, the gerontologic literature has often discussed disability and depressive symptomatology as life course processes in later life; our theories and hypotheses regarding them have increasingly emphasized them as such, but our research designs and analyses have remained effectively cross-sectional. Furthermore, regarding social support, literature conceptualizing support as a life course process, especially in later life, has been virtually nonexistent. Yet, just as stress can be conceptualized as a process—indeed, the notion of chronic stress implies a process—social support can be considered a life course process that emerges/develops in response to stress processes such as growth in disability in later life. In this research, we have explicitly modeled stress, depressive symptoms, and social support as processes in later life. Our goal has been to examine the relationship between long-term patterns of disability at the individual level—a type of chronic stressor—and long-term patterns of one of its psychological consequences—depressive symptoms. In addition, we considered long-term patterns of social support, both received/actual support and perceived

availability of support, as potential temporal—and not simply static—mediators of this relationship.

The results strongly support the conceptualization of disability, depressive symptoms, and support as linear processes in later life. Specifically, a considerable amount of the variance in the observed individual-level time-specific measures was explained via a linear growth specification for each (nonlinear specifications were also attempted but produced similar results). Furthermore, a considerable amount of variability in trajectories of disability, support, and symptoms was evident across the sample. The first model showed a strong relationship between trajectories of disability and trajectories of depressive symptoms, such that persons with increasing trajectories of disability tend to have increasing trajectories of depressive symptoms. In general, these results are fairly consistent with previous research showing a relationship between disability and depression or depressive symptoms (Berkman et al., 1986; Bruce, 2001; Roberts et al., 1997). However, our results extend previous research by demonstrating that long-term *patterns* of disability are related to long-term *patterns* of depressive symptoms at the individual level, a finding that is important to advancing a life course understanding of the interrelationship between late-life health processes.

Subsequent models including trajectories of received and perceived support showed that trajectories of perceived support, but not received support, tend to match trajectories of disability and provide a buffer against the effect of disability on depressive symptoms. The salience of perceived support as a stress mediator is not a new finding (Cohen & Wills, 1985; Kessler & McLeod, 1985). However, this work contributes to existing literature in at least two ways. First, this research is the first to demonstrate that this mediation extends over time and not simply at one point in time. In fact, this is the first research, to our knowledge, that considers treating social support as a process. Much of the previous research has examined the relationship between social support and depressive symptoms using cross-sectional data or an inherently cross-sectional model on short-term longitudinal data (e.g., by regressing depressive symptoms on prior measures of support). Yet, to the extent that chronic stressors such as disability (or repeated occurrences of life event stressors) (Lynch & George, 2002) can be viewed as life course processes, we can expect social support to evolve in response as well, and the results indicate they do.

Our results indicate that trajectories of received support do not buffer the effect of disability on depressive symptoms but that perceived support completely mediates the relationship between growth in disability and growth in symptoms. This somewhat supports the work of Krause (1997) on anticipated and received support. Perceived support had the expected effect, but received support did not. Although individuals in our sample were more likely to report higher levels of received support as their disability levels increased (Mendes de Leon et al., 2001; Seeman et al., 1996), received support did not exacerbate the effects of disability on depressive symptoms, as suggested by Krause (1997).

An important finding regarding perceived social support was that the effect of latent baseline disability on baseline depressive symptoms remained significant, while the effect of growth in disability on growth in depressive symptoms was eliminated by the inclusion of perceived support. This finding

underscores the importance of using a truly longitudinal method for modeling and understanding life course processes. Our finding shows that the relationship between *growth* in disability and *growth* in depressive symptoms is mediated by *growth* in perceived social support, whereas baseline disability's effect on baseline depressive symptoms is not mediated by baseline perceived support. Using a cross-sectional design or analysis therefore would not fully reveal the importance of social support across the life course.

Despite the significance of the findings, one of the greatest limitations of these analyses is that, even with our multiwave longitudinal design, direction of causality cannot be conclusively established between disability, social support, and depressive symptoms. An important feature of growth modeling is that the method is more consistent with the concept of life course processes than traditional cross-sectional or similar methods. However, a limitation is that causal direction cannot be established. Indeed, this is always a problem regardless of the method used, but it is also true with growth models, because the multiple measures are reduced to (often) two parameters per individual so that the temporal priority of one set of measures versus another is lost. We believe, however, that disability most likely produces the need for social support and therefore increases the actual receipt of support while perhaps simultaneously reducing the actual perception of support. Nonetheless, although the perception of support may be reduced in the face of disability, it may still be sufficient (as our results indicate) to reduce depressive symptoms. Of course, we cannot rule out the possibility that those with the highest levels of depressive symptoms perceive the least support—the so-called “contamination hypothesis” (George, Blazer, Hughes, & Fowler, 1989). Although previous research has demonstrated less support for this hypothesis than our causation hypothesis, even with long-term longitudinal data, the direction of causality cannot be fully ascertained. Our conclusion is that high levels of, and rapid growth in, disability may produce feelings that an individual's support is inadequate for handling disability but may nonetheless be sufficient to mediate the effect of disability growth on depressive symptom growth.

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