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Interlocking Trajectories of Loss-Related Events and Depressive Symptoms Among Elders

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Objectives. As people age, their peers (who are also aging) become increasingly susceptible to health decline and death, implying potential growth in stressful loss-related events over time for the individual. Yet little research has examined trajectories of stress and their relationship to trajectories of depression among elders. The purpose of this research was to determine whether growth in loss-related events occurs for elders and whether stress growth is related to the well-known growth in depressive symptomatology in later life.

Methods. Three waves of National Institute on Aging Established Populations for Epidemiologic Studies of the Elderly (Duke University site) data were used in the analyses. Latent growth curve models were estimated for stress, for depressive symptoms, and for stress predicting depression net of several covariates.

Results. Findings include that (a) loss-events evidence clear growth across age at the aggregate level, but with much variation within the sample, and (b) variation in growth in stress is strongly related to variation in growth in depressive symptoms.

Discussion. The results suggest that stress in later life may be conceived of as a growth process, with strong consequences for trajectories of mental health.

AFTER 40 years of research, the fact that stress is a risk factor for physical and mental illness, especially depression, is well established. A myriad of studies based on both age-heterogeneous samples (e.g., Ensel & Lin, 1991; Simon & Marcussen, 1999; Turner & Lloyd, 1999) and samples of older adults (e.g., Ensel, 1991; Holahan & Moos, 1991; Roberts, Kaplan, Shema, & Strawbridge, 1997) support this conclusion. (For recent reviews, see George, 1996; Wheaton, 1999). Despite the volume of research on stress and depression in later life, unresolved issues remain. This article addresses two of these issues, using longitudinal data obtained from a representative sample of community-dwelling older adults: (a) the relationship between loss-related events and depressive symptoms in later life and (b) using appropriate statistical methods for understanding the dynamic interplay between stress and depression.

The Distinctive Character of Late-Life Depression

There are several distinctive facets of depression in later life. First, rates of depressive disorder are lower among older adults than among young and middle-aged adults (e.g., American Psychiatric Association, 1994; Weissman, Bruce, Leaf, Florio, & Holzer, 1991). In contrast, most investigators report that depressive symptoms are higher among older adults than among their younger peers—especially among the old-old (i.e., persons 80–85 years of age or older; e.g., Blazer, Burchett, Service, & George, 1991; Mirowsky & Ross, 1992; Roberts et al., 1997). Findings from the relatively few long-term longitudinal studies are less clear cut (e.g., Glass, Kasl, & Berkman, 1997; Holahan & Moos, 1991). A limitation of these findings is that they are based on sample means rather than on individual trajectories of symptoms over time. One hypothesis tested in this study is that a substantial minority of older adults will report trajectories of increasing depressive symptoms over time.

As noted above, the experience of stressful life events has been empirically linked to higher levels of depressive symptoms for adults of all ages. Some evidence also suggests, however, that life events are of lesser importance for depression in later life than at earlier ages. Two streams of research support this conclusion. First, in some studies of age differences in the social and behavioral antecedents of depressive symptoms, life events have been observed to have weaker effects for older than for younger and middle-aged adults (e.g., George, 1992; Keith, 1987). Second, older adults report fewer life events, on average, than young and middle-aged individuals (e.g., Goldberg & Comstock, 1980; Hughes, Blazer, & George, 1988). Perhaps as a result of these empirical patterns, recent research on stress and depression in later life has focused more on chronic stressors, especially physical health problems and caregiving responsibilities, than on life events (e.g., Koenig, O'Connor, Guarisco, Zabel, & Ford, 1993; Schulz, Visintainer, & Williamson, 1990). No studies to date, however, have related trajectories of life events to trajectories of depressive symptoms among older persons. Examination of trajectories suggests that the dynamics of life events and depressive symptoms are more complex than can be captured by models in which life events at one point in time are used to predict changes in depression over a subsequent time interval.

The general hypothesis tested in this study is that trajectories of increasing levels of stress will be associated with trajectories of increasing depression. In light of previous evidence that older adults experience fewer life events than do their younger peers, it is unlikely that using a traditional life-events check list will be suitable to the research question. We used a measure of interpersonal loss-related events to test our hypothesis. The losses in the measure include death of a spouse, death of a child, death of a close friend or (other) family member, and serious illness or injury experienced by a close friend or family member. The last event is not a loss in the same sense that death of a loved one is. Nonetheless, we argue that older adults experience serious illness or injury of significant others as tangible reminders of their own mortality and/or the shrinking of their social networks (e.g., an ill or disabled significant other cannot interact in the same ways as previously—a kind of direct loss, as well as a reminder of the precariousness of life in old age). We hypothesized that repeated exposure to such loss-related events will be associated with increasing levels of depressive symptoms.

Aneshensel (1992) proposed a distinction between "random stressors" and "systemic stressors." Random stressors are those that occur across all individuals, regardless of social location or resources. As such they are important for understanding risk of mental health problems, but will not explain the social patterning of mental illness. Although this is a valuable distinction, it ignores life-course patterns of stressors. Interpersonal losses, for example, generally are not experienced randomly across the life course. Although relationships can and do end at all ages, the loss of significant others is likely to increase dramatically in late life, as friends and relatives die or become disabled. In addition, the loss of important age peers and perhaps significant others who are younger than oneself is likely to trigger concerns about one's own health and longevity. Both the grief of losing significant others and anticipating one's own health declines and death may increase psychological distress. For these reasons, we used trajectories of loss-related events as predictors of trajectories of depressive symptoms.

Interpersonal losses are not, of course, the only kinds of losses that commonly occur in late life. Exits from the labor force, for example, also are concentrated in late adulthood. We restricted our measure of stress to interpersonal losses for both conceptual and methodological reasons. Conceptually, because the majority of exits from the labor force are voluntary, we would not expect them to have the same effects on psychological well-being as the loss of friends and family. Moreover, research consistently reports that retirement has no substantial effects on mental and physical health (e.g., Kasl, 1980). Methodologically, we wish to demonstrate that trajectories of increasing or repeated stress are associated with increasing levels of depression. Consequently, the stressors examined must be ones that have the possibility of increasing over time. In general, we cannot expect individuals' exits from the labor force to increase over time—for most people, retirement is a "one-shot" transition. In contrast, although only a few individuals will experience widowhood multiple times over 6 years, other interpersonal losses have the potential to increase (as well as to decrease or remain at baseline levels).

Stress, of course, is not the only risk factor associated with depressive symptoms in previous research. Therefore, our model inclues a number of covariates that have been associated with depressive symptoms in previous research: age, sex, race, education, physical health, and functional status. Because we are studying trajectories of depressive symptoms, we will be able to examine the extent to which these covariates are associated with both baseline levels of depression and patterns of change over time.

Appropriate Methods for Modeling Long-Term Patterns of Stress and Depression

Despite efforts to broaden the conceptualization and measurement of stress exposure, issues of timing and duration have been largely ignored in research to date. In part, this reflects limitations in the data available to many investigators: Much previous research is based on cross-sectional data and the relatively few longitudinal studies available are often restricted with regard to times of measurement, intervals between measurements, and total duration of the study. But the relative neglect of timing and duration also results in part from a failure to conceptually address the dynamics of stress exposure. An exception to this is found in Pearlin and colleagues' work on stress proliferation, a process in which stressors multiply and "spill over" into life domains beyond that in which the original stressor occurred (Pearlin, Aneshensel, & LeBlanc, 1997). Again, however, the data available to these investigators permitted little opportunity to model dynamics—baseline reports of caregiving and secondary stressors were used to predict depressive symptoms 7 months later. Although the secondary stressors undoubtedly emerged over time, their dynamics remain empirically uncharted.

One approach to modeling change and stability in both the independent (here, loss-related events) and dependent (depression) variables, when more than two waves of data are available, is to model trajectories using latent growth analysis. The specific question tested in this study is whether trajectories characterized by increasing levels of loss-related events are associated with trajectories characterized by increasing levels of depressive symptoms. Because individual trajectories of both stressors and depressive symptoms are estimated for each sample member, attention to heterogeneity is much greater than in more conventional regression techniques.

We are aware of only one other study using this approach, and it examined trajectories of stress and depression during adolescence (Ge, Lorenz, Conger, Elder, & Simons, 1994). Ge and colleagues hypothesized that trajectories of increasing numbers of uncontrollable life events would be associated with trajectories of increasing depression symptoms for adolescents. They report that, at each time of measurement, number of uncontrollable life events is significantly associated with level of depressive symptoms for both boys and girls. Trajectories of increasing stress, however, explain changes in depressive symptoms over time, but only for adolescent girls. Their hypothesis is similar to ours with the exception that they suggest that uncontrollable life events are especially salient for adolescent mental health, whereas we argue that interpersonal loss-related

events are particularly important for mental health in late life.

Methods

Sample

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–funded multicenter, collaborative research program designed to prospectively investigate the physical, mental, social, and cognitive functioning of persons age 65 and older in several communities. EPESE data have been collected in four sites: New Haven, CT (Yale University), East Boston, MA (Harvard University), rural Iowa (University of Iowa), and north central North Carolina (Duke University).

The Duke EPESE data include seven times of measurement over 6 years. In-person interviews, averaging 2 hr in length, were collected at baseline (Year 1), Year 4, and Year 7. Brief telephone surveys were conducted in Years 2, 3, 5, and 6. The data used in these analyses are from the three in-person interviews.

The sampling frame for the Duke EPESE project was a five-county area of north central North Carolina consisting of one urban county and four contiguous rural counties. Sampling procedures were designed to generate a stratified random sample of approximately 4,000 adults age 65 and older. The sample was stratified on the basis of race with the intent of African Americans comprising half of the sample (compared with approximately 35% of the population in the sampling frame). First-stage sampling consisted of selection of segments in the five counties; second-stage sampling consisted of selection of households within the segments. Households selected were rostered for all residents age 65 and older. Predetermined selection grids were used to randomly select one respondent from each household, based on number of eligible persons (Kish, 1965). The baseline sample consisted of 4,162 persons age 65 and older; the response rate was 80%. Sample weights were initially used in these analyses to adjust for the oversampling of African Americans; however, the results did not vary from those without the weights, and hence unweighted analyses are reported.

The sample size for the analyses reported here is 1,972. This represents all respondents who participated in the three in-person interviews. Of the 2,190 missing cases, 1,310 died prior to the third wave, and another 752 were either administered proxy interviews at subsequent waves, moved out of the interview area and were administered telephone interviews, or were simply lost at follow-up. An additional 128 (3%) were missing on one or more measures used in the analysis. Logistic regression analyses were conducted to examine systematic differences between the missing and nonmissing. These analyses suggested that the missing contribute little if any bias. In the first analyses, persons who died prior to Wave 3 were compared with survivors. Those who died were more likely to be older (odds ratio [OR] = 1.08), about twice as likely to be male (OR = 1.93), and made slightly less income (although statistically significant, the difference is substantively 0). Among survivors, analyses suggested that those who attrited for other reasons were again slightly older (OR = 1.06), were more likely to be White (OR = 1.25), and had slightly lower education (OR = .962). Among the remainder, there were no significant differences between the 128 missing on one or more items and those for whom we had complete data. Clearly, the majority of attrition was due to death or incapacity. Exclusion of these persons at worst produces a negative bias, making the results of the analyses conservative. That is, if stress increases the risk of illness and/or death and we eliminate those persons who experienced death and severe disability, we necessarily underestimate the influence of stress.

Measures

Loss-related events.—Life events were assessed in the EPESE with a scale of 22 major events that has been used in previous research (Hughes et al., 1988; Landerman, George, Campbell, & Blazer, 1989). Respondents were asked about the occurrence of each event during the year prior to the interview. We extracted four loss-related events from the scale: death of a spouse, death of a child, death of a close friend or (other) family member, and serious illness or injury of a close friend or family member. These items were summed to produce a count of loss-related events at each interview. Because of differential risk (i.e., some respondents had no spouse and/or children), the count was rescaled into percent of possible loss-related events the respondent experienced. In other words, the denominator for a person who had no spouse or any children would be 2, rather than 4. Making respondents comparable, therefore, required using percent of possible loss events experienced.

A larger problem for these analyses is the fact that life events are available for only 3 years. Given the 1-year time frame for reports of the events, we have no information about loss events that may have occurred during Years 1 to 2, 2 to 3, 4 to 5, and 5 to 6. Although this lack of full data about loss-related events across the 6 years is unfortunate, unless these events are temporally patterned, the three measures can be viewed as samples of the occurrence of events across time. Another problem with the loss-related event measures is that they sum the number of types of events experienced rather than a full count of all events. It is unlikely that respondents lost more than one spouse or child during a 1-year interval, but the measure undoubtedly underestimates the number of close friends and (other) relatives who died or experienced serious illness or injury for some respondents. The effect of both problems, however, is for the analyses to underestimate the effects of the stressors on depression.

Depressive symptoms.—Number of depressive symptoms was measured using the Center for Epidemiologic Studies Depression scale (CES-D; Radloff, 1977), a well established and widely used scale. The CES-D inquires about 20 symptoms using the time frame of the past week. In the Duke EPESE, the CES-D response categories were truncated to a simple yes–no format (in the original scale there are four response categories assessing frequency of symptoms). The validity of this modified form of the CES-D is

documented in Blazer and colleagues (1991). The internal consistency of the CES-D was excellent at all three interviews (α = .89, .87, and .86, respectively).

Control variables.—Age was coded in years; at baseline, the age range was 64 to 95 with a mean of 71.6 years. Race was coded as a dichotomous variable (0 = non-White, 1 = White; 45.6% were White), as was gender (1 = male, 31.7%). Education was measured as years of formal schooling (*M* 9.1, *SD* 4.0). Physical health was measured using an index developed by Duke EPESE investigators. The index is based on the presence or absence of five chronic conditions: cardiovascular disease, hypertension, diabetes, stroke, and cancer (specific type or site of cancer is known). The five chronic conditions are weighted for severity and prognosis and the weighted scores are summed. Across the survey period, the weighted scores increase slightly, reflecting worsening health (Wave 1 *M* = 33.8, *SD* = 28.4; Wave 2 *M* = 37.7, *SD* = 31.0; Wave 3 *M* = 44.5, *SD* = 33.8). Analyses by the Duke EPESE investigators show that the index is a stronger predictor of functioning and mortality than a simple count of the chronic conditions. Functional status was measured using the instrumental ADL Scale developed in the Framingham Disability Study (Branch, Katz, & Kniepman, 1984). Internal reliability for the scale is very high (α = .98). This scale also reveals worsening health across time (Wave 1 *M* = .37, *SD* = .99; Wave 2 *M* = .52, *SD* = 1.14; Wave 3 *M* = .98, *SD* = 1.67).

Analytic Strategy

In this analysis, we used latent growth curve methods to examine trajectories of stress and depressive symptoms and to determine whether trajectories of stress predict trajectories of depressive symptoms. In a latent growth curve model, individual trajectories are constructed; here, time-specific measures of stress and depression are seen as observed indicators of a latent growth process. Across individuals, stress and depression trajectories may vary considerably, both in terms of levels of stress and depressive symptoms at baseline and in terms of the rate of true growth in stress and depressive symptoms over time. In a typical growth curve model, one assumes that observed measures contain input from two components: the parameters of the latent process and measurement error. If the process is assumed to follow a linear trajectory over time, two growth parameters define the trajectory: a latent intercept and a slope (α and β, respectively). Thus, in hierarchical modeling language the Level 1, or within individual, model is:

$$\begin{bmatrix} y_1 \\ y_2 \\ \vdots \\ y_k \end{bmatrix} = \begin{bmatrix} 1 & t_1 \\ 1 & t_2 \\ \vdots & \vdots \\ 1 & t_k \end{bmatrix} \begin{bmatrix} \alpha \\ \beta \end{bmatrix} + \begin{bmatrix} e_1 \\ e_2 \\ \vdots \\ e_k \end{bmatrix}. \quad (1)$$

The errors (e) for the each time (k)-specific measure (y) are assumed to be normally distributed with a mean of 0 and covariance matrix σ_e, where the σ_e matrix can incorporate heteroscedasticity and cross-variable error correlation. For a linear growth model, t_j measures time from baseline (hence $t_1 = 0$). α and β are assumed to be normally distributed with mean vector

$$\begin{bmatrix} \mu_\alpha \\ \mu_\beta \end{bmatrix}$$

and covariance matrix Σ_μ. This Level 1 equation thus models individual trajectories (of stress and depression). The mean vector relates the average initial level of stress or depression and change in stress or depression across time, whereas the covariance matrix Σ_μ relates between-individual variation in trajectories. This interindividual variation can be modeled in a Level 2 model. If the regressors are without measurement error, then the following equation holds:

$$\begin{bmatrix} \alpha \\ \beta \end{bmatrix} = \begin{bmatrix} \mu_\alpha \\ \mu_\beta \end{bmatrix} + \begin{bmatrix} \gamma_{\alpha x_1} & \gamma_{\alpha x_2} \cdots \gamma_{\alpha x_j} \\ \gamma_{\beta x_1} & \gamma_{\beta x_2} \cdots \gamma_{\beta x_j} \end{bmatrix} \begin{bmatrix} x_1 \\ x_2 \\ \vdots \\ x_4 \end{bmatrix} + \begin{bmatrix} \zeta_\alpha \\ \zeta_\beta \end{bmatrix}. \quad (2)$$

From this equation, an individual's latent intercept and slope can be viewed as a linear combination of the aggregate mean of the true intercept and slope (which is now adjusted for covariate values of 0), the effect of regressors (here, unlike in Willett & Sayer, 1994, we do not deviate regressors from their means), and a normally distributed error term with a 0 mean vector and some covariance matrix (say ψ). These equations (and extensions) can be estimated easily with existing structural equation modeling (SEM) software, such as LISREL (see e.g., Jöreskog & Sörbom, 1993; Willett & Sayer, 1994). Furthermore, relationships between multiple growth curves can be estimated. In that case, the vector of regressors exploits the full latent variable SEM model (see Bollen, 1989), with a measurement model for the regressor growth curve specified through the x measurement portion of the SEM model (similar to Equation 1). Finally, the growth curve approach can be extended to incorporate various patterns of nonlinear growth; this can be done in at least two ways. First, one can incorporate a third latent factor (say β_2), with the necessary changes in the dimensionality of Equations 1 and 2. Similarly, the t_j parameters must be altered to reflect the desired curvilinearity (i.e., for a quadratic curve, one would square the t_j for the new factor). Second, one can directly incorporate the nonlinearity in the first β term (see Ge et al., 1994; McArdle & Epstein, 1987) by modifying the t_j parameters as needed. The former approach is perhaps preferable, while the latter approach may be necessary for identification purposes (see Bollen, 1989; for more discussion of growth curve modeling, see Meredith & Tisak, 1990; Rogosa & Willett, 1985).

In this analysis, we first estimated a univariate growth curve for stress to determine whether growth in loss-related events occurs for elders. Next, we estimated a similar univariate growth curve for depression. Finally, we estimated a set of models that relate growth in stress to growth in depression,

net of age, gender, race, education, physical health, and functional status. Figure 1 provides a graphic depiction of the full SEM model to be estimated.

Results

We first conducted within-individual ordinary least squares (OLS) regressions for each individual in the sample to examine individual patterns of stress and depression across time. The estimated individual slopes for both stress and depression across time were then coded as either increasing, stable, or decreasing, on the basis of their sign, yielding a 3 × 3 crosstab. Tables 1 and 2 contain both the aggregate means for stress and depression at each wave and a 3 × 3 table of the OLS-produced within-individual stress and depressive symptom patterns.

The aggregate means for stress reveal a clear increasing pattern. The mean percent of stressful events experienced by the sample at baseline was 17.64%, and that percent increased across the 3 waves to 21.29% by Wave 3. The aggregate means for depressive symptoms did not have a monotonically increasing pattern across waves. At Wave 1, the average depressive symptoms for the sample was 2.85; at Wave 2, the average had declined to 2.41; by Wave 3, the average symptoms had increased to 2.86. Heterogeneity in individual-level patterns makes it difficult to determine whether the quadratic aggregate pattern is the result of quadratic individual patterns or linear individual patterns. However, although an individual-level quadratic life course pattern for depression is well-known (e.g., see Mirowsky and Ross, 1992), the typical individual-level pattern tends to be linear and increasing for the age range covered by our sample, and hence we assume that the aggregate pattern is an artifact of heterogeneity in the sample (e.g., so that individual and aggregate patterns differ). Furthermore, although the quadratic aggregate pattern is statistically significant, the curvature is very slight and substantively unimportant.

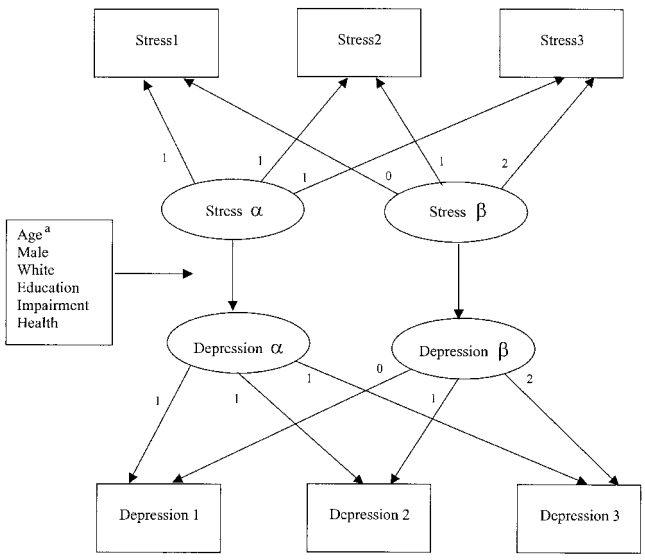

Figure 1. Full growth curve model of stress growth predicting depression growth. All models in the analysis are subsets of this model. The univariate growth curve model for stress is the upper half of the figure; the growth curve for depression is the lower half of the figure; the model of stress predicting depression is both the upper and lower half of the figure; the final model with covariates includes the covariates to the left of the main figure. [a]These covariates affect all latent variables (growth curve intercepts and slopes) in the model; however, all paths are not included in the figure in order to keep the figure simple. Similarly, measurement errors and covariances are omitted.

Table 1. Aggregate Means and Standard Deviations for Stress and Depression at Each Wave

	Stress		Depression	
Wave	*M*	*SD*	*M*	*SD*
1	17.64	18.8	2.85	3.24
2	20.17	18.0	2.41	3.28
3	21.29	17.9	2.86	3.50

The goal of the analyses is to determine whether individual-level patterns of stress predict individual-level patterns of depressive symptoms. The cross-tabulation of the OLS regressions discussed above provides a preliminary answer to this question (see Table 2). The table reveals a clear pattern, with approximately 40% of the sample falling on the diagonal, indicating that individuals' stress patterns are related to their depression patterns. Another 40% fall just off the diagonal, experiencing no growth in depressive symptoms, but either positive or negative growth in stress, or experiencing no growth in stress, but either positive or negative growth in depressive symptoms. A simple chi-square test revealed a significant association between these crudely-measured stress and depression trajectories, χ^2 (4, N = 1,972) = 28.17, $p < .001$.

The results of the OLS analyses, although interesting, suffer from at least two limitations. First, regressing individuals' measures on time ignores within-individual autocorrelation across time. Second, if we were to proceed and regress the depressive symptom slopes on the stress slopes, we would, at a minimum, be ignoring error in the estimation of these slopes. Thus, the second step in the analysis was to refine these preliminary analyses by estimating latent growth curve models for stress and depression independently, and

Table 2. Within-Individual OLS Estimated Slopes for Stress and Depression

Variable	*n*	%
Stress +		
Depression +	305	15.5
Depression 0	149	7.6
Depression −	250	12.7
Stress 0		
Depression +	279	14.2
Depression 0	202	10.2
Depression −	297	15.1
Stress −		
Depression +	169	8.6
Depression 0	88	4.5
Depression −	233	11.8

Notes: OLS = ordinary least squares. Within-individual OLS regressions were conducted to determine individuals' trends in stress and depression. These slopes were then classified as 0, positive, or negative, and were cross-tabulated. Table χ^2 (4, N = 1,972) = 28.17, $p < .001$.

then estimating models with stress predicting depression net of covariates. Results of these models can be found in Tables 3 and 4.

Table 3 presents the results of univariate growth curve models for stress and depression. Univariate growth curves require only estimation of the Level 1 equations presented above, as their purpose is to estimate the aggregate growth pattern in the data and variation around the average. In Model 1, a univariate growth model for stress is presented. In terms of overall model fit, this model fit the data quite well. The model chi-square was small, χ^2 (1, N = 1,972) = 2.51, 1 p = .11, and other measures of model fit suggested excellent fit. Here we use two additional measures of model fit—the incremental fit index (IFI) and the root mean squared error of approximation (RMSEA). The IFI for a well-fitting model should be above at least .9 and preferably above .95; the RMSEA should be below .1, and preferably below .05 for a well-fitting model (see Bollen, 1989). For this model, the IFI was .99, and the RMSEA was .028. Although the measurement parameters were fixed to provide a linear specification, the results of interest include the mean and variance of the latent growth parameters (α and β). The latent intercept parameter (α) for stress had a mean of 17.87, and the latent slope parameter (β) had a mean of 1.82. Both are highly significant ($p < .001$ for each), suggesting that at baseline, elders experienced about 17.87% of possible events on average. The positive latent slope parameter suggests that the average sample member experienced a slight growth in loss-related events of approximately 2% per wave of the study, hence supporting our hypothesis that these types of events occur with increasing frequency across age. However, the variance around each of these means is significant in both initial stress experience and in growth in stress over time. Specifically, the variance parameter for baseline stress indicates that most of the sample experienced between 0% and 36% of possible stressful events at baseline, and the variance parameter for growth in stress experience indicates that trajectories range from approximately −8% to 11% growth in stress per wave. In sum, the growth model for stress fits the data well, with significant variability in baseline stress and growth across the sample.

Table 3. Growth Curve Model Level 1 Equation Results: Univariate Growth Curves for Stress and Depression (Duke EPESE Data, n = 1,972)

	Model 1 (Stress)		Model 2 (Depression)	
Parameter	α	β	α	β
μ	17.87***	1.82***	2.71***	−.01
σ^2	78.86***	22.41**	5.65***	.68***
cov(α, β)	−24.07***		−0.35	
y1[a]	1	0	1	0
y2	1	1	1	1
y3	1	2	1	2
Model χ^2 (*df*)	2.51(1)		51.27(1)***	
RMSEA	.028		.16	
IFI	.99		.96	

Note: Level 1—individual level—Equations: $y_k = \alpha + t_k\beta + e_k$.

[a]Measurement parameters in these equations are fixed for a linear growth specification; only mean and variances of latent variables are estimated. Effects run from latent variables (α,β) to the observed indicators.

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

Table 4. Growth Curve Model Level 2 Equation Results: Effects of Stress and Covariates on Depression Growth (Duke EPESE Data, n = 1,972)

	Model 1 (S→D)		Model 2[a] (X→S)		S, X→D	
Parameter	α	β	α	β	α	β
Intercept (μ)[b]	1.13***	−.19**	28.46***	−6.4	1.76	−.43
Age			−.17*	.12*	.01	.01
Male			−2.53**	1.12	−.85***	.03
White			.53	−.82	−.05	.04
Education			.17	−.15	−.14***	−.01
Impairment			.41	−.13	.42***	−.16***
Poor Health			.02	.00	.01***	.00
Stress α	.09***				.086***	
Stress β		.1***				.095***
R^2	.12	.42	.04	.03	.29	.46
Model χ^2 (*df*)	77.93(9)***		87.46(21)***			
RMSEA	.062		.04			
IFI	.96		.97			

Notes: Level 2 Equations: $\alpha = \mu_\alpha + x\Gamma + \zeta_\alpha$ and $\beta = \mu_\beta + x\Gamma + \zeta_\beta$ Model changes for inclusion of stress growth model as predictor (see Willett and Sayer, 1994).

[a]In this model, covariates (x) influence both stress and depression growth curves, hence requiring the additional columns.

[b]The intercept term is for the dependent variable, before considering influences by regressors. For example, for Model 1 the equation for stress growth predicting depression growth is: [Depression β = −.19 + .1∗(Stress β)]. Similarly, the equation for initial stress level predicting initial depression level is: [Depression α = 1.13 + .09∗(Stress α)].

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

Model 2 in Table 3 presents results of a linear growth model for depression. In comparison with the stress model, the overall model fit indices suggest that the depression model did not fit as well. The chi-square was large and significant, χ^2 (1, N = 1,972) = 51.27, $p < .001$, and the RMSEA was large (.16). The IFI, however, indicates acceptable fit (IFI = .96). The mean and variance parameters for the depression model indicate that average baseline experience of depression was 2.71 symptoms, and the variance around this mean is significant, with most persons experiencing between 0 and 8 symptoms at baseline. The mean growth parameter for depression for the sample was 0, indicating that on average there was no growth in depression across the survey period. However, whereas the average growth rate was nonsignificant, the variance around this mean was highly significant, with most of the sample experiences ranging between a loss of 2 symptoms per wave and a gain of 2 symptoms per wave.

The second step in the growth analysis was to determine whether growth in stress and a set of fixed covariates explain the variation in individual level growth patterns in depressive symptoms (the Level 2 equations from above). Table 4 presents the results of these models. In the first model, the growth model for stress was used to predict growth in depressive symptoms. In the second model, age, gender, race, education, functional status, and health were added to determine whether the relationship between stress and depressive symptoms is spurious and whether these factors

contribute to symptoms. In Model 1, the overall model fit indices indicate the model had acceptable fit. Although the chi-square was large and significant (probably the result of a large sample), the RMSEA was reasonable (.06) and the IFI suggested the fit was acceptable (.96). The effect of baseline stress on baseline symptoms was significant (Γ = .09, $p < .001$) and explained 12% of the variance in baseline symptomatology. The effect of stress growth on depressive-symptom growth was also highly significant (Γ = .1, $p <$ .001) and explained 42% of the variance in depressive-symptom growth.

To provide some indication of the substantive implications of these results, one can calculate a predicted depressive symptom growth rate by taking the intercept for the β parameter (−.19), and adding .1 * the stress growth rate. If an individual's stress growth rate were 20% per wave, then the expected growth rate in depressive symptoms would be −.19+(.1*20) = 1.81 symptoms per wave. Significantly, an individual with 0% growth in stress would have an expected loss of .19 depressive symptoms per wave. The means for both the latent intercept and slope of depressive symptoms can also be recovered from this model by calculating the equations with the means for the stress parameters. The estimated mean for the latent intercept of stress was 17.87 (from Table 3, Model 1). Hence, the mean for the latent intercept for depressive symptoms is estimated as 1.13 + (.09*17.87) = 2.74, which is within rounding of the estimate of 2.71 in Table 3, Model 2. Similarly, the estimated mean for the latent slope for depressive symptoms is estimated as −.19 + (.1*1.82) = −.01, which is equivalent to the estimate of −.01 in Table 3, Model 2.

In Model 2, additional covariates were introduced, and these covariates were allowed to influence both the stress growth curve and the depressive-symptom growth curve. The overall model fit indices suggest this model had good fit. The chi-square was large and again significant; however, the RMSEA was quite small (.04) and the IFI was large (.97). In terms of the effect of stress on depression, the covariates reduced the influence of baseline stress on baseline symptoms and the influence of stress growth on symptom growth, but only slightly: These effects remained strong and highly significant. The additional covariates appear to have the greatest effect on baseline symptoms, more than doubling the explained variance to 29%. However, the covariates had relatively little influence on growth in symptoms, increasing explained variance from 42% in the model with stress alone to 46% in the full model. Men had lower baseline depressive symptoms, but gender had no influence on the symptom growth rate. Education also had a negative influence on baseline depressive symptoms, but no significant influence on growth. Functional impairment and poor health each had positive effects on the baseline symptom count, but only impairment influenced growth in symptoms. That influence was negative, which was probably an artifact of regression toward the mean. In sum, the effect of the covariates is largely to influence baseline depressive symptomatology, but these results may be attributed to the fixed measurement of the covariates. However, it should be noted that additional models were constructed that treated functional impairment and poor health as time varying (not reported in table), and the results of those models did not differ substantially from those presented here.

The covariates in Model 2 also influence stress, but the effect of the covariates on stress was limited. Age and gender each negatively influenced baseline stress, and only age affected growth in stress (effect is positive). Relatively little variance in baseline stress and stress growth was explained (4% and 3%, respectively). In sum, it appears that stress trajectories are strongly related to depression trajectories, accounting for much of the variance in depressive symptom growth, and that this relationship does not appear spurious given the covariates introduced.

Finally, a few notes are in order regarding additional models that were conducted but not reported here. First, nonlinear growth specifications were tried for the depression models; the results did not change significantly. The lack of change across linear and nonlinear specifications is largely the result of having only 3 waves of data—the nonlinear specification used here only changes one parameter. We argue that, although it is well known that depression experiences a quadratic increase in later life, much of the curvature occurs prior to ages 65 to 70 (see Mirowsky & Ross, 1992). Thus, as stated above, we assume that the nonlinear aggregate pattern for depressive symptoms is likely an artifact of sample heterogeneity, and beyond age 65, individual-level growth in depressive symptoms is theoretically linear and can be captured adequately through a linear model. The excellent fit of our full model suggests that a linear specification is appropriate. Nonetheless, given the poor fit of the univariate linear growth model for depression, future research should investigate this curvilinearity further; doing so, however, will require more than 3 waves of data.

Second, given the uniqueness of our stress measure (although it is theoretically motivated), we conducted sensitivity analyses to determine whether the results were an artifact of measurement. We conducted four sets of analyses, each involving reestimation of the full model (Table 4, Model 2) using a modified stress measure obtained by omitting each of the four constituent stress measures. The results of these models did not alter the overall results. R^2 for depression alpha ranged from .28 to .37; R^2 for depression beta ranged from .35 to .59. Similarly, the effect of baseline stress on baseline depression ranged from .06 to .16, and the effect of stress growth on depression growth ranged from .07 to .16. RMSEAs ranged from .037 to .041, and IFIs were all .97. The model chi-square ranged from 79 to 90. Thus, although some coefficients changed, the overall results and substantive implications remained the same.

Discussion

The purpose of this article was to contribute to our understanding of the relationship between stress and depression in late life using a trajectory approach and latent growth curve analysis to examine issues of change over time and sample heterogeneity. This is a substantially different strategy than the more typical regression analysis in which stress is examined as a predictor of current levels of depression or of changes in levels of depressive symptoms. As hypothesized, the results indicate that trajectories of growth in loss-

related events are strong predictors of trajectories of growth in depressive symptoms over a 6-year interval. In contrast, trajectories of stable or decreasing stress are associated with trajectories of decline in depressive symptoms.

One of the reasons that patterns of change are highlighted so clearly using latent growth curve analysis is the explicit attention to heterogeneity provided by the construction of individual trajectories. As expected, we observed wide variation in trajectories of both loss-related events and depressive symptoms, with stability, increases, and decreases well represented in the sample. Indeed, growth in events was experienced by a minority of sample members, albeit a sizeable minority (approximately 40%). Using latent growth curve analysis, however, we were able to test the hypothesis that growth in loss events over time will be associated with increases in depressive symptoms. Thus, we did not simply test the hypothesis that increases in both loss-related events and depression are typical of the older population. Rather, we also tested the hypothesis that those persons who experience increases in loss-related events will be at increased risk for increases in depressive symptoms.

Although not the primary focus of this article, the effects of the covariates on trajectories of depression are of interest. The results indicated that all of the covariates except age and race were significantly related to the intercept of the trajectory of depressive symptoms, but only functional impairment was significantly associated with the slope of the trajectory. The relationships between the covariates and the intercept are comparable to observations in previous cross-sectional and short-term longitudinal studies: Women report higher levels of depressive symptoms, as do the those with lower levels or education, higher numbers of chronic illnesses, and higher levels of functional impairment. In contrast, all the covariates but one were unrelated to the slope of the trajectory of depressive symptoms. Thus, for example, with the effects of loss-related events statistically controlled, women were no more likely than men to exhibit trajectories of increasing numbers of depressive symptoms over time. The single covariate significantly related to the slope of the depression trajectory was functional impairment, and its effect was negative (i.e., high levels of impairment were associated with decreasing depression symptoms over time). This finding is contrary to the usual expectation that functional impairment will increase the risk of depression. But other explanations are possible—for example, this may represent a form of regression toward the mean or it may be that individuals adjust to their impairments over time, reducing psychological distress.

This study is not without limitations. Obviously, the sample was restricted to older adults living in north central North Carolina. As such, replications are needed with either national samples or samples from diverse geographic areas, as well as with other age groups. The latter is especially important, because loss-related events may not be as common among middle-aged adults, and, in those age groups, other kinds of stressors may be stronger predictors of increases in depressive symptoms. We also would have preferred to have a longer series of data. As noted above, with only three measurement points, nonlinearity can be examined in only a truncated fashion. Finally, better measures of stress—ones which more accurately capture stress experience—would be desirable. As mentioned before, our measures were truncated both in terms of failing to provide a count of each type of event between waves and in terms of failing to cover the entire interval between waves of data collection. Nonetheless, we have argued that these effects should have only negatively biased the relationship between stress and depression.

The relationship between stressors and depression has been a major research issue for more than three decades. It may be tempting to assume that we already know all that we need to about this relationship. This article has attempted to demonstrate that on both theoretical (i.e., incomplete attention to stress exposure) and statistical grounds there is more to be learned.

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