Race Differences in Depressive Symptoms: 
A Dynamic Perspective on Stress Exposure and Vulnerability*

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The existence, nature, and strength of race differences in mental health remain unclear after several decades of research. In this research, we examine black-white differences in the relationship between acute stressors and depressive symptoms. We reframe the stress exposure and differential vulnerability hypotheses in the context of long-term trajectories of stress and depression, and we hypothesize that trajectories of stress growth will be associated with trajectories of depressive symptom growth. Using latent growth curve analysis of a sample of 1,972 older persons interviewed three times at three-year intervals, we test the hypotheses that (1) growth in exposure to loss-related events will predict growth in depressive symptoms, and (2) African Americans will experience greater stress growth than whites. Results support the hypotheses. Stress growth exhibited a linear increase for blacks but not for whites, and predicted depression growth for both races, but explained more variance for blacks than for whites.

A substantial body of research addresses status differences in mental health and explanations for those differences. The primary social statuses examined to date include socioeconomic status, race, gender, age, and marital status. Differing levels of stress exposure and differential vulnerability to stress have been the major explanations offered to account for status differences in mental health. This paper has two primary purposes: (1) to examine the extent to which stress exposure and vulnerability account for differences in depressive symptoms between older African Americans and whites, as well as the extent to which they explain within-group variability; and (2) to reconceptualize the concepts of stress exposure and stress vulnerability in a prospective, dynamic fashion.

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RACE DIFFERENCES IN DEPRESSION AND DISTRESS

Despite a voluminous research base, the basic question of whether blacks and whites differ in levels of depression and psychological distress remains unclear. The distinction between major depressive disorder and depressive symptoms explains, in part, the inconsistency of findings. African Americans typically report significantly higher numbers of depressive symptoms than white. This pattern is observed in both age-heterogeneous samples (Amato 1991; Frerichs, Aneshensel, and Clark 1981; Jackson 1997; Kessler 1979; Myers et al. 2002; Ulbrich, Warheit, and Zimmerman 1989) and in samples restricted to older adults (Blazer et al. 1998; Cochrane, Brown, and McGregor 1999; Fernandez et al. 1998; Nelson 1989), although a few studies report no race differences (Gallo, Cooper-Patrick, and Lesikar 1998; Husaini et al. 1990). In studies of depressive disorder, in contrast, race differences are either non-significant or African Americans are at lower risk of disorder than
whites. This pattern also is observed in both age-heterogeneous samples (Kessler, Michelson, and Williams 1999; Oquendo et al. 2001; Williams, Takeuchi, and Adair 1992) and in samples of older adults (Blazer et al. 1994; Haralson et al. 2002).

The interrelationships among race, socioeconomic status, and depression/distress have been of particular concern. Again, results are inconsistent, with some studies reporting that race differences in depression are rendered non-significant when socioeconomic status is controlled (e.g., Frerichs, Aneshensel, and Clark 1981) and others finding that socioeconomic status does not fully account for race differences in distress (e.g., Kessler and Neighbors 1987). Moreover, the effects of race and socioeconomic status on depression may be interactive, although, again, the nature of such interactions remains unclear. Some investigators (Kessler and Neighbors 1987; Ulbrich, Warheit, and Zimmerman 1989) report that race differences in depression and distress are strongest among people with low incomes. Cockerham (1990), in contrast, found no race difference in levels of depression at low levels of socioeconomic status, but that blacks had significantly lower levels of depression than whites at high levels of socioeconomic status.

In addition to socioeconomic status, stress exposure and vulnerability have been the primary factors hypothesized to explain race differences in depression. Indeed, stress exposure and vulnerability are the most widely posited explanations for a broad range of status differences in mental health (e.g., gender, socioeconomic status, marital status). According to the stress exposure hypothesis, African Americans report higher levels of depression/distress than whites because they are exposed to greater stress. The differential vulnerability hypothesis posits that, at equal levels of stress, blacks react more strongly to stressors, generating greater increases in depression symptoms than observed among whites. These are not, of course, mutually exclusive hypotheses: African Americans could both experience more stress than whites and react more intensely to it.

Although the stress exposure and vulnerability hypotheses have been tested quite frequently for other statuses, they have received little attention as possible explanations for race differences in depression/distress. In the first empirical study testing these hypotheses, Kessler (1979) found that differential vulnerability was more important in explaining socioeconomic status, gender, and marital status differences in distress, whereas stress exposure was the key determinant for differences between whites and non-whites. Ulbrich and colleagues (1989) report more complex results: Blacks were more vulnerable to depression than whites when confronting undesirable life events; whites were more vulnerable when exposed to chronic economic problems.

Differential vulnerability has received more empirical support than differential exposure in accounting for status differences in depression. A number of hypotheses about possible explanations for differential vulnerability have been tested. The common theme of these hypotheses is that more vulnerable groups have fewer social and personal resources that can be used to eradicate or lessen the negative effects of stress on health. Social integration, social support, and psychosocial resources such as self-esteem and self-efficacy have been the primary factors studied to date, to varying degrees and with varying success.

BROADENING THE DEFINITION OF STRESS EXPOSURE

Recently, the case for expanding the concept of stress exposure has appeared on multiple fronts. Turner and colleagues argue that the boundaries of stress exposure need to be extended beyond recent life events and current chronic stressors, advocating the concepts of "operant stress" and "cumulative stress" (Turner, Wheaton, and Lloyd 1995). Operant stress refers to the total number of stressors affecting an individual at a given point in time, including recent life events, more distal life events that respondents report affect their lives currently, and chronic stressors (Avison and Turner 1988; Turner et al. 1995; Turner and Lloyd 1999). Cumulative stress refers to the combination of current stress and temporally distal traumas that are believed to be so significant that they become permanent sources of stress (Turner et al. 1995; Turner and Lloyd 1995). In studies to date, both operant stress and cumulative stress were (1) stronger predictors of depression than more conventional measures of recent life events and (2) significant mediators of relationships between social statuses (gender, age, marital status, and SES).
and depression (Turner et al. 1995; Turner and Lloyd 1999). Unfortunately, they did not examine the effects of operant and cumulative stress on race differences in depression.

Pearlin and colleagues’ work on stress proliferation also calls attention to new ways of thinking about stress exposure (Pearlin, Aneshensel, and LeBlanc 1997). Stress proliferation refers to the propensity for stressors to multiply and “spill over” into life domains beyond that in which the original stressor occurred. Pearlin et al. refer to the original stressor as the “primary” stressor; “secondary” stressors result from the original stressor, but occur in other life domains. Stress proliferation occurs when a primary stressor leads to other primary stressors or generates secondary stressors. Using the experience of providing care for a person with AIDS, Pearlin et al. demonstrated that the primary stressor of taking on caregiving responsibilities often resulted in other primary stressors as the illness progressed (e.g., role overload, role captivity) and in secondary stressors (e.g., work strain, constriction of social and leisure activities). The primary and secondary stressors operated in cumulative fashion to increase depressive symptoms.

We employ another strategy for broadening the scope of stress exposure. Specifically, we use latent growth curve analysis to examine the extent to which trajectories of stress, exhibited over six years, predict trajectories of depression over the same interval. We examine differences between African Americans and whites in both trajectories. The estimates generated by latent growth curve analysis speak directly to the dynamics of stress exposure and vulnerability.

Latent growth curve analysis estimates a number of parameters. The specific estimation procedures we used are described below; in this section, we provide a rationale for interpreting those parameters as indicators of stress exposure and vulnerability to stress. Latent growth curve analysis is a two-level hierarchical model. In the first level, individual trajectories of the variables of interest are constructed for all sample members. The mean trajectory for the sample is estimated. In the second level, heterogeneity around the mean trajectory is modeled to determine the extent to which it is a function of the predictors and covariates of interest.

Latent growth curve analysis generates four estimators for each group (here, African Americans and whites): latent intercepts for both stress and depressive symptoms and coefficients calibrated as growth factors for both stress and depression. The intercepts represent baseline levels of the variables, and the extent to which the intercepts are explained by predictors and covariates is estimated. Thus, a set of covariates is used to predict baseline levels of stress, and the same covariates plus baseline stress levels are used to predict baseline depression scores. This part of the analysis is a cross-sectional analysis of race differences in the predictors of depressive symptoms.

Growth coefficients represent the direction and amount of change in the variables. We predict both stress growth and depression growth over the six years of observation. Predictors of stress growth include the set of covariates and baseline level of stress; predictors of depression growth include the covariates, baseline levels of stress and depression, and stress growth.

In the latent growth curve analysis models reported here, stress exposure is measured as both current/recent stress at baseline and as changes in the direction and amount of stress over a six-year interval. Traditional cross-sectional and short-term longitudinal studies operationalize stress exposure as the number of stressors experienced in the present or recent past. Operant stress expands stress exposure to include distal events that continue to affect the individual’s life. Cumulative stress broadens stress exposure by including early traumas that are believed to have persisting effects on health. None of these methods examines stress growth prospectively. We argue that trajectories of growth or decline in levels of stress over time are another useful way to operationalize stress exposure and to prospectively observe its effects on depression.

What about differential vulnerability using latent growth curve analysis? Covariates and predictors can be used to determine, in the conventional way of most stress research, whether personal characteristics and social resources explain group differences in stress growth, depression growth, or both. However, use of latent growth curve analysis provides another, more dynamic way of thinking about differential vulnerability. If stress growth is differentially associated with growth in depression for African Americans and whites, this may represent group differences in vulnerabil-
ity to stress. For example, if stress growth is a stronger predictor of growth in depressive symptoms for whites than for African Americans, whites may be more vulnerable to stress than African Americans. Thus, differential vulnerability takes on a different meaning when long-term trajectories of stress and depression are the focus of analysis.

HYPOTHESES

These analyses are based on data from a multistage probability sample of black and white adults age 65 and older living in a defined geographic area. Using data from a sample of older adults poses a potential problem in testing the hypothesis that stress growth predicts growth in depressive symptoms because older adults report fewer life events than younger persons (e.g., George 1992). Certain types of life events are more common among older than younger adults, however, including the deaths and illnesses of age peers. To test the hypothesis that stress growth predicts growth in depression, we use a measure of loss-related events. The losses in the measure are death of a spouse, death of a child, death of a friend or other family member, and serious illness or injury experienced by a close friend or family member. The last item is not a loss in the same sense as death of a loved one. We argue, however, that serious illnesses or injuries experienced by significant others are likely to lead to decreased interaction and unwanted changes in the nature of the relationship. In this sense, it is an interpersonal loss.

Our primary hypothesis focuses on the stress exposure hypothesis: We hypothesize that stress growth will predict growth in depressive symptoms over time. We also predict that older African Americans will experience higher levels of growth in loss-related events than older whites, reflecting race differences in health and longevity. We examine vulnerability to stress in terms of whether older blacks and whites differ in the strength of stress as a predictor of depression, but we do not offer a hypothesis with regard to vulnerability.

DATA AND MEASURES

The data for this study are from the Duke Established Population for Epidemiologic Study of the Elderly. The sample consists of whites and an oversample of blacks (approximately 55% of the sample) age 65 and over from five counties in North Carolina, representing both rural and urban areas. The study has a panel design, with seven waves of data collected over six years (1986–1993). In-person interviews were conducted in years 1, 4, and 7, with telephone interviews conducted between in-person waves. The data used in these analyses are from the in-person interviews.

The sample size for the study was 4,162 at baseline. However, we report results for survivors and non-attriters to wave 3, reducing our final sample to 1,972. Of the 2,190 missing, 1,310 died prior to last interview, and 752 attrited. The attritors included individuals for whom proxy interviews were required because of severe cognitive or physical impairment, persons who moved out of the area, and those who refused continued participation. One hundred twenty-eight (6%) of the remaining sample members were missing on one or more measures used in the analysis. Logistic regression analyses suggested that the missing did not differ much from our subsample. Compared to survivors, decedents were more likely to be older (OR = 1.08) and male (OR = 1.93). Comparing the analysis sample with attriters, the latter were slightly older (OR = 1.06), more likely to be white (OR = 1.25), and had slightly less education (OR = .962). There were no significant differences between the subsample used in the analysis and the 128 sample members missing on one or more items.

If the missing data cause any bias, it is likely to be a conservative one. The outcome measure is depressive symptoms, which are related to general health and mortality risk. Thus, excluding decedents leaves a more robust sample that may tend to have fewer depressive symptoms and less growth in them over time. Also, we are estimating a late-life age pattern, so the exclusion of decedents and attriters should not affect the estimated age pattern. Differences in selective survival or attrition between races could, arguably, generate racial differences in growth patterns for stress and depression. We have several responses to this argument, however. First, our theory concerns a late-life pattern that is irrelevant to decedents. Thus, eliminating the decedents, the
majority of missing cases, introduces no sub-
stantive bias. Second, in addition to the analy-
ses reported here, we conducted Bayesian ran-
dom coefficient models, which permitted
inclusion of deceased and attrited sample
members until they exited the study. The
results of those models did not differ substan-
tively from those reported here. Third, most
techniques for handling missing data (e.g.,
imputation) assume that the data are either
missing completely at random or missing at
random (see Allison 2002 for a discussion of
this distinction). If either selective survival or
attrition are related to either stress or depres-
sion, the missing at random assumption is vio-
lated. There are few techniques to handle this
problem, and, to our knowledge, there are no
structural equation modeling packages avail-
able which incorporate them. If the missing
data are missing at random, the only result of
listwise deletion (used here) is a loss of statis-
tical power.

The two primary measures used in the study
are loss-related events and depressive symp-
toms. Four life events from a 22-item scale
measure loss-related events: death of spouse,
death of a child, death of a friend or other fam-
ily member, and serious illness or injury of a
close friend or family member. We summed
the items to produce a count of types of events
experienced at each wave (range 0–4), and we
then adjusted to compensate for differences in
risk (e.g., persons who do not have a spouse or
child are not at risk for those losses). The
adjustment consisted of dividing the count of
event types by the number of events for which
the respondent was at risk and multiplying by
100 to obtain the percentage of possible events
experienced.

There are two primary limitations to this
measurement. First, respondents were inter-
viewed at three-year intervals, but life events
were asked about only for the preceding year.
Thus, we miss two-thirds of the total stress
experience. Second, the questions do not
assess how frequently the event occurred in the
previous year. This is unlikely to be a problem
for the deaths of spouses, but some sample
members undoubtedly had more than one
friend, child, or other family member die or
become ill during the year prior to being inter-
viewed. The result of these limitations is that
we underestimate growth in loss-related
events.

Depressive symptoms were measured with
the Center for Epidemiologic Studies
Depression Scale (CES-D). The version of the
scale used in the Duke study (Blazer et al.
1991) consists of 20 dichotomous items indi-
cating whether the respondent experienced
depressive symptoms in the past week. The
scale has excellent internal consistency at all
waves (Kronbach's alpha = .89, .87, and .86).
Three variables were used as controls: sex
(female = 0, male = 1), education (in years),
and functional impairment status, as measured
at baseline. Functional status was measured
using the Instrumental Activities of Daily
Living Scale developed in the Framingham
Disability Study (Branch et al. 1984).

Age and race enter the analyses in funda-
mental ways. Age forms the basis for the
growth curve models. Six cohorts, based on
baseline age, were created: ages 65–67, 68–70,
71–73, 74–76, 77–79, and 80 and older. The
data were further disaggregated by race (black
and white), yielding a final set of 12 subsam-
plings (6 age groups X 2 race groups). Table 1
provides the sample sizes and descriptive sta-
tistics for the covariates by age-race subgroup,
as well as subgroup means for stress and
depressive symptoms.

METHODS
We use latent growth curve analysis to
determine (1) whether levels of stress are relat-
ed to depressive symptoms, and (2) whether
growth patterns in stress are related to growth
patterns in depressive symptoms. The first
question is typically answered by cross-sec-
tional (or short-term panel), regression-based
analyses; the latter question must be answered
using dynamic models. To date, very few stud-
ies have examined the relationship between
stress and depression using dynamic models. A
standard approach to examining the life course
pattern in depression, for example, is to treat
some function of age as a covariate in a cross-
sectional data set and then estimate a standard
regression model (e.g., Mirowsky and Ross
1992). This is not a true individual-level
approach, however, because only the aggregate
pattern of depressive symptoms is revealed: in-
dividual heterogeneity in life course patterns
is ignored. Furthermore, if stress is used as a
predictor of depression, it is treated statically
and tells us only whether stress at one point in
time is related to depressive symptoms at a
### TABLE 1. Means for Variables in Analyses by Age-Race Subgroup

<table>
<thead>
<tr>
<th></th>
<th>Baseline Age Group among Whites</th>
<th>Baseline Age Group among Blacks</th>
<th>Row Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>66.0</td>
<td>69.0</td>
<td>72.0</td>
</tr>
<tr>
<td>Male</td>
<td>.47</td>
<td>.37</td>
<td>.31</td>
</tr>
<tr>
<td>Education</td>
<td>10.6</td>
<td>10.4</td>
<td>10.7</td>
</tr>
<tr>
<td>Impairment</td>
<td>.25</td>
<td>.21</td>
<td>.22</td>
</tr>
<tr>
<td>Stress by Age</td>
<td>17.1</td>
<td>17.1</td>
<td>18.9</td>
</tr>
<tr>
<td></td>
<td>21.2</td>
<td>17.8</td>
<td></td>
</tr>
<tr>
<td></td>
<td>21.0</td>
<td>19.8</td>
<td>19.4</td>
</tr>
<tr>
<td></td>
<td>22.9</td>
<td>20.3</td>
<td>16.1</td>
</tr>
<tr>
<td>Depression by Age</td>
<td>2.3</td>
<td>2.6</td>
<td>2.7</td>
</tr>
<tr>
<td></td>
<td>2.0</td>
<td>2.4</td>
<td>2.7</td>
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<tr>
<td></td>
<td>2.1</td>
<td>1.6</td>
<td>2.8</td>
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<td>2.7</td>
<td>2.3</td>
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<td>3.0</td>
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</tr>
<tr>
<td></td>
<td>3.3</td>
<td>3.0</td>
<td></td>
</tr>
<tr>
<td>n</td>
<td>267</td>
<td>184</td>
<td>166</td>
</tr>
</tbody>
</table>
later time at the aggregate level. Again, individual-level heterogeneity in the stress-depression relationship across the life course is ignored.

Growth curves offer a flexible way to examine relationships between life course processes at the individual level that are heterogeneous across individuals, and to identify sources of heterogeneity. In a typical growth curve model, individual-level time-specific measures are assumed to be indicators of the latent process of interest and to contain input from two sources: the latent process and measurement error. The process is governed by two unobserved parameters: an intercept (\( \alpha \)) and a growth factor (\( \beta \)). At the first level of a growth curve model, the process is modeled to determine the extent of heterogeneity in the sample:

\[
\begin{bmatrix}
y_1 \\
y_2 \\
\vdots \\
y_k
\end{bmatrix} = \begin{bmatrix} f(t_1) \\
1 \\
f(t_2) \\
1 \\
f(t_k) \\
1
\end{bmatrix} \begin{bmatrix} \alpha \\
\beta \\
e_1 \\
e_2 \\
e_k
\end{bmatrix}
\]

In this equation, the errors for the time (k)-specific outcome measures (y) are assumed to be normally distributed random noise with a mean vector of 0 and covariance matrix \( \sigma_e \). The \( \sigma_e \) matrix can be diagonal (implying \( e_i \) and \( e_j \) are uncorrelated \( \forall i \neq j \)), or it can incorporate additional sources of unmeasured heterogeneity (e.g., via error correlations). For a linear specification, \( f(t) \) measures the distance from baseline (in years, months, or other units), with \( t_1 = 0 \) (so \( f(t) \neq t \)). \( \alpha \) and \( \beta \) are considered to be normally distributed with a mean vector of \( \mu_{\alpha,\beta} \) and covariance matrix \( \Sigma \). This equation thus models variability in individual trajectories around the mean trajectory for the subsample. This variability (heterogeneity) then can be modeled at a second level, by allowing \( [\alpha, \beta]^T \) to be a function of covariates and predictors:

\[
\begin{bmatrix}
\alpha \\
\beta
\end{bmatrix} = \begin{bmatrix} \mu_{\alpha} \\
\mu_{\beta}
\end{bmatrix} + \begin{bmatrix} \gamma_{\alpha x_1} & \gamma_{\alpha x_2} & \cdots & \gamma_{\alpha x_4} \\
\gamma_{\beta x_1} & \gamma_{\beta x_2} & \cdots & \gamma_{\beta x_4}
\end{bmatrix} \begin{bmatrix} x_1 \\
x_2 \\
\vdots \\
x_4
\end{bmatrix} + \begin{bmatrix} \xi_\alpha \\
\xi_\beta
\end{bmatrix}
\]

In this equation, the mean vector has been decomposed into an adjusted mean and a linear combination of covariates \( [x_1, x_2, \ldots, x_4]^T \), which are, in this case, assumed to be measured without error. The second level error vector, \( [\xi_\alpha, \xi_\beta]^T \), is assumed to have a mean vector of 0 and a covariance matrix \( \psi \), which represents unexplained between-individual heterogeneity.

These models can be estimated using standard structural equation modeling software such as LISREL. If the covariates and predictors are allowed to contain measurement error, then the full latent variable model presented by Bollen (1989) is used. In our case, we estimate growth curve models for both stress and depression and allow the stress curve to predict the depression curve, exploiting the full latent variable model.

Figure 1 displays the full model we estimate. We assume that stress and depression follow a growth process across age. We assume the pattern for stress is linear and the depression pattern is quadratic (we establish \( f(t) = t^2 \) for depression). We assume the stress pattern is linear because of the truncated measurement; we assume the depression pattern is quadratic because this pattern has been found in previous research (e.g., Mirowsky and Ross 1992). With three waves of data, it is generally difficult to distinguish between linear and quadratic patterns; however, as discussed below, our approach using missing data techniques allows us to capture a quadratic pattern. We also modeled a linear growth pattern for depression; those models produced results almost identical to those reported here. They also did not fit the data quite as well as the models presented in this paper. In general, the results we report here are equivalent to estimating a linear growth curve on the square root of depressive symptoms.

We use age as the basis for our growth curves. Recall from equation 1 that \( y_k \) represents the measurement of the outcome variable at time k. Time k can represent either a specified wave (e.g., \( y_{1986} \)), or an age (e.g., \( y_{65} \)). Aside from random birthday fluctuations, t is not affected by the growth curve's basis. In panel data, all individuals are typically measured at each wave (attritors and decedents deleted), but not all individuals are measured at all ages. Thus, if we use age as the basis for our growth curves and we wish to estimate our model across the complete age range covered by the data, we will have missing data at the individual level for ages we do not observe in the sample—the portion of data collected in a panel is only a diagonal band through a lexis surface. Such a model is estimable, however, using missing data techniques in multiple
group models. For such models, we separate the data into groups based on baseline age (as discussed above), each of which is observed at three time points. We specify a complete growth curve model for the entire age range represented by the sample, but we must alter the paths from the latent intercept and growth factors in each group to reflect (1) the ages for which we observe the group and (2) the ages for the group for which the data are missing. For the observed group, we set the paths from the latent intercept to the observed data equal to 1 and from the latent growth factor to the observed data equal to \( f(t) \), where \( t \) represents years from the baseline age range of 65–67. Since the measurement periods are evenly spaced, we need only set these parameters proportional to \( t^2 \) for depression (we use 0, 1, 4, 9, 16, 25, 36, and 49), and \( t \) for stress (we use 0, 1, 2, 3, 4, 5, 6, and 7). For the group with missing data, we set the paths from both the latent intercept and growth factors equal to 0. We also set the error variance for these missing variables equal to 1. Finally, we adjust the covariance matrices that are used as the input data for estimation such that the missing variables have a mean of 0 and a variance of 1, and we establish the covariances between the missing variables and all other variables in the model to be 0. These changes force the model to perfectly replicate the missing data, while the observed data and the model specification for the paths to them allow estimation of the growth curve across the entire age range.

Figure 2 presents a lexis diagram for the data that are observed and missing. At the bottom of the figure, the coefficients for the paths from the latent intercept and growth factors for depressive symptoms are listed for each group. As the figure illustrates, there is considerable overlap in the age ranges covered by the age groups within race. For example, we observe three different groups in four of the age ranges and two different groups in two of the age ranges. At the extreme ages (65–67 and 86+) we have information from only one group each. For groups that overlap, we constrain the measurement error variances to be equal in
### FIGURE 2. Lexis Diagram of Cohorts and Ages Covered by Waves 1, 2, and 3 of the Duke EPESE, and First-Level Growth Curve Specifications

<table>
<thead>
<tr>
<th>Wave/Cohort</th>
<th>65-67</th>
<th>68-70</th>
<th>71-73</th>
<th>74-76</th>
<th>77-79</th>
<th>80-82+</th>
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<tr>
<td>1</td>
<td>X</td>
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<tr>
<td>2</td>
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<tr>
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<td></td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
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**Scaled time from age 65 =**

<table>
<thead>
<tr>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>1</td>
<td>4</td>
<td>0</td>
<td>0</td>
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</tr>
<tr>
<td>0</td>
<td>0</td>
<td>9</td>
<td>4</td>
<td>16</td>
<td>0</td>
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</tr>
<tr>
<td>0</td>
<td>0</td>
<td>0</td>
<td>9</td>
<td>9</td>
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<td>36</td>
</tr>
</tbody>
</table>

**Scaled paths from latent growth factor to age-specific indicator by baseline age group to fit quadratic pattern**

**Note:** The Os in the Lexis diagram indicate that some persons in this age group (80-82) are older than 82 at baseline.
those age ranges, just as the paths from the latent variables are constrained to be equal. Additionally, we constrain the means and variances for the latent variables to be equal across all age groups (hence leading to a single curve across all age groups), as well as the effects of the covariates (e.g., education, sex, and functional status), but we allow them to vary across races. This assumes that there are no cohort differences in measurement or growth patterns, or in the effect of covariates on stress and depression, but this is a reasonable assumption for the age groups covered. Also, this assumption is typical in research that attempts to estimate life course patterns using data on multiple cohorts. For more discussion of these methods, see Allison (1987) and McArdle and Hamagami (1992).

The benefit of this approach is that we are able to estimate the relationship between stress and depression patterns across a much larger age range than would otherwise be possible. Furthermore, the additional time points allow us to estimate more flexible patterns than a simple linear one. There are drawbacks as well, however: It is more difficult to obtain convergence with these models, and some modifications may be needed. In this case, we ultimately had to free the variance of the latent stress variables for the oldest age group within each race to achieve convergence. However, this seems a reasonable approach because the oldest group includes a wider range of ages (ages 80+ at baseline versus 3 year groups for those under 80).

RESULTS

Table 1 presents descriptive statistics for all variables used in the analyses for each age-race subgroup. The percent male declines across each age group, as might be expected given gender differences in survival. Mean educational attainment is considerably lower for blacks in all age groups; within each race there appears to be a slight negative trend, indicating higher educational attainment across later birth cohorts. The functional impairment measure evidences an upward trend across age groups for both races, reflecting declining health across age (or improving health across cohorts). Blacks evidence greater impairment than whites at all ages.

The table presents the means for stress and depression for each age-race group across the three waves of the study (in columns). The table also presents the means for stress and depression at each age (a weighted average across rows). Examining the means within age groups across time reveals little about the overall late life patterns in stress and depression. For some age groups, stress clearly increases; for others, the shape is less clear. For depression, almost all age groups evidence a quadratic depression pattern, with wave 2 depression scores being lower than those in waves 1 and 3. The averages by age, however, show a clearer pattern. Stress for whites appears to increase slightly across waves, from 17.1 percent of possible events at age 65 to 19.1 percent by age 86. There is a slight upward pattern at the aggregate level, but the pattern evidences considerable fluctuation. The age pattern for blacks is more clearly linear and increasing. At age 65–67 blacks experienced 18.9 percent of possible events, increasing with relatively little reversal to 23.1 percent of by age 86. The depressive symptom pattern for both blacks and whites shows increases in symptoms across age.

The first level equations of the growth curve model estimate the aggregate pattern for stress and depression across age, as well as heterogeneity around the pattern. Table 2 reports the indicator reliabilities from the level one equations in the full growth curve model. These reliabilities indicate the proportion of the total variance in the indicators that is accounted for by the estimated aggregate pattern. Recall from the equations above that there are two levels of variance in the growth curve model: within-individual variance and between individual variance. For a model in which the growth curve captures considerable within-individual variance (meaning the specified pattern fits at the individual level), the indicator reliabilities will be high. If there is considerable between individual heterogeneity that can be captured by the growth curve, there will be a large between-individual variance as well. However, if the specified growth pattern doesn’t hold well at the individual level, the indicator reliabilities will be low (indicating that the individual patterns aren’t fit well by the specified growth pattern), and between-individual heterogeneity will not be very large either, suggesting that most of the variance in the time-specific indicators is within-individual variation that cannot be captured by the specified
TABLE 2. Growth Curve Model Level 1 Equation Results: Indicator Reliabilities

<table>
<thead>
<tr>
<th>Stress by Age</th>
<th>Baseline Age Group among Whites</th>
<th>Baseline Age Group among Blacks</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>65-67</td>
<td>68-70</td>
</tr>
<tr>
<td>65</td>
<td>.12</td>
<td>.19</td>
</tr>
<tr>
<td>68</td>
<td>.10</td>
<td>.10</td>
</tr>
<tr>
<td>71</td>
<td>.09</td>
<td>.09</td>
</tr>
<tr>
<td>74</td>
<td>.11</td>
<td>.11</td>
</tr>
<tr>
<td>77</td>
<td>.15</td>
<td>.15</td>
</tr>
<tr>
<td>80</td>
<td>.21</td>
<td>.21</td>
</tr>
<tr>
<td>83</td>
<td>.19</td>
<td>.19</td>
</tr>
<tr>
<td>86</td>
<td>.19</td>
<td>.19</td>
</tr>
<tr>
<td>n</td>
<td>267</td>
<td>184</td>
</tr>
</tbody>
</table>

Note: The off-diagonal elements have 0 R-squares, reflecting that all paths leading to the 'missing data' are constrained to be 0 (hence error variance = total variance). R-squares are the same across baseline age groups measured at overlapping ages, because the error variances were constrained to be equal within age, except at the oldest baseline ages (80+). Rounding error in the estimation process led to slight variations in R-squares.

growth curve. In these analyses, the indicator reliabilities for stress for both blacks and whites suggests that much of the variability in the indicators is not captured by the growth curve. The error variances for the indicators were constrained to be equal across age groups measured at the same age (e.g., stress at age 71–73 was measured for three baseline age groups: group 1 was 71–73 at wave 3, group 2 was 71–73 at wave 2, and group 3 was 71–73 at wave 1). Thus, in Table 2, the reliabilities are the same across rows within each race (slight variation is attributable to rounding error in the estimation algorithm). For whites, the reliabilities range from .09 to .21, indicating that about 10 to 20 percent of the total variance in individual-level stress experience is captured by our linear growth specification. For blacks, the reliabilities range from .14 to .25—a slightly better fit than for whites. The reliabilities for both racial groups are much better for depression. For whites, the depression indicator reliabilities ranged from .44 to .93; for blacks, they ranged from .43 to .71. The high reliabilities suggest that the quadratic specification we used fits the data well at the individual level. These findings have several implications. First, the hypothesis that stress will increase linearly across late life receives little support. However, this may be due to the measurement limitations discussed above. It could also be due to a reduction in at-riskness at extreme old ages—by age 80 or so, individuals may have very few network members left to lose. Second, the low reliabilities imply a small between-individual variance in the second level equation, making it difficult for stress growth to explain depression growth (as in OLS, small variance in x makes it difficult to explain variance in y).

Table 3 reports the results from the second level equations. We used three criteria to assess overall model fit: the model chi-square, the root mean squared error of approximation (RMSEA), and the goodness-of-fit index (GFI). The model chi-square was significant, suggesting a poor fit of the model to the data. However, this test tends to be significant in large datasets. The ratio of the chi-square to its degrees of freedom suggested the model did not fit poorly (i.e., the ratio was 1.16; ratios less than 4 are considered acceptable). The RMSEA was 0, suggesting excellent fit, and the GFI was .97, also indicating good fit.

Figure 3 presents observed aggregate means (from Table 1) and predicted aggregate stress and depression patterns from the full model. The predicted scores for the stress growth curve are computed in two steps: first, the latent intercept and growth factors are computed as the linear combination of the estimated coefficients and the covariates set to their
TABLE 3. Growth Curve Model Level 2 Equation Results: Effect of Stress Growth and Covariates on Depression Growth (n = 1,972)

<table>
<thead>
<tr>
<th>Outcomes</th>
<th>Covariates</th>
<th>Stress α</th>
<th>Stress β</th>
<th>Depression α</th>
<th>Depression β</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blacks</td>
<td>Intercept (μ)</td>
<td>19.23***</td>
<td>.093</td>
<td>2.82***</td>
<td>.012</td>
</tr>
<tr>
<td></td>
<td>Education</td>
<td>.02</td>
<td>.03</td>
<td>-15***</td>
<td>-.001</td>
</tr>
<tr>
<td></td>
<td>Male</td>
<td>-3.72**</td>
<td>.66</td>
<td>-82***</td>
<td>-.03</td>
</tr>
<tr>
<td></td>
<td>Impairment</td>
<td>.28</td>
<td>-.09</td>
<td>.51***</td>
<td>-.01</td>
</tr>
<tr>
<td></td>
<td>Stress α</td>
<td>.07***</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Stress β</td>
<td>.05***</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>ψ³76.8****</td>
<td>2.93</td>
<td></td>
<td>4.70****</td>
<td>.004</td>
</tr>
<tr>
<td></td>
<td>R²50+ age group</td>
<td>98.0</td>
<td>.11</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>ψβ -9.7</td>
<td></td>
<td></td>
<td>-0.07*</td>
<td></td>
</tr>
<tr>
<td></td>
<td>R²50+ age group</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>R² covariates only model</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>R² covariates only model</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Whites</td>
<td>18.30***</td>
<td>-.54</td>
<td>.71</td>
<td>.02</td>
</tr>
<tr>
<td></td>
<td>Education</td>
<td>.15</td>
<td>.04</td>
<td>-16***</td>
<td>-.001</td>
</tr>
<tr>
<td></td>
<td>Male</td>
<td>-2.40†</td>
<td>.91*</td>
<td>-38</td>
<td>-.02</td>
</tr>
<tr>
<td></td>
<td>Impairment</td>
<td>.56</td>
<td>.04</td>
<td>.15</td>
<td>.005</td>
</tr>
<tr>
<td></td>
<td>Stress α</td>
<td>.18***</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Stress β</td>
<td>.03***</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>ψ³38.46**</td>
<td>1.66</td>
<td></td>
<td>4.37****</td>
<td>.005***</td>
</tr>
<tr>
<td></td>
<td>R²60+ age group</td>
<td>370.52†</td>
<td>9.37</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>ψβ -3.39</td>
<td></td>
<td></td>
<td>-0.06†</td>
<td></td>
</tr>
<tr>
<td></td>
<td>R²65-79 age groups</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>R² covariates only model</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Model x² (df)</td>
<td>723.49 (626)**</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>RMSEA</td>
<td>0.0</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>GFI</td>
<td>.97</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

† p < .1, * p < .05, ** p < .01, *** p < .001
* These are the adjusted means for the growth curves (adjusted for effect of covariates).
# These are the adjusted means for the growth curves (adjusted for effect of covariates).

Although the error variances of the latent variables are held constant across all age groups, slight differences (in the hundredths place) were still allowed by LISREL in the variances themselves, yielding R-squares that vary slightly across the groups. We present the average R-squares across the groups. The error variances for the latent stress factors were allowed to vary for the oldest age group, hence their R-squares are substantially different.

The model chi-square degrees of freedom have been adjusted to reflect the fact that the missing data are constructed to have a mean of 0 and a variance 1.

LISREL reports the GFI for each group. We present the average GFI across all groups.

means; second, the predicted aggregate patterns are computed by using equation (1). That is, the predicted time 1 measure for stress is simply the computed latent intercept; the predicted time t score is computed by multiplying f(t) by the estimated growth factor mean and adding the result to the predicted latent intercept. For the linear stress model, f(t) = t. The predicted scores for the depression growth curve are only slightly more difficult to compute, given that they are a function of the stress growth curve and the growth pattern is assumed to be quadratic. The computation is identical, except that the predicted stress latent intercept and the predicted stress latent growth factor are included in the computation of the estimated latent depression intercept and growth factor. The figure demonstrates that the aggregate stress pattern is more variable for whites than for blacks. Also, stress growth appears steeper for blacks than for whites. For depression, both black and white patterns are consistent with the quadratic specification. However, while blacks evidence greater symptoms at every age, the curvature of the symptom growth across age appears less than that for whites. Table 3 details these results and describes how the stress and depressive symp-
tom growth patterns relate to each other for blacks and whites.

In the model presented in Table 3, we allowed the covariates to influence the latent parameters for both stress and depression, implying that there may be indirect effects of the covariates on depression growth through stress growth. We estimated another model in which the covariates were only allowed to influence the latent parameters for depression (making the stress growth curve fully exogenous), but this model did not fit as well. The results we present suggest, however, that the effect of the covariates on stress was weak, predicting little variance in the latent intercept for stress ($R^2 = .04$ for blacks and $R^2 = .05$ for whites in all but the oldest age groups; $R^2 = .03$ for blacks and $R^2 = 0$ for whites in the oldest age groups) and little variance in the latent growth rate for stress, except for the oldest group of blacks ($R^2 = .04$ for blacks and $R^2 = .10$ for whites in all but the oldest groups; $R^2 = .52$ for blacks and $R^2 = .01$ for whites in the oldest age groups). In terms of specific effects for blacks, only gender had a significant effect on latent baseline stress, with males experiencing considerably less stress ($\gamma = -3.72, p < .01$). For whites, this effect was nearly significant and of similar magnitude ($\gamma = -2.4, p < .1$). None of the covariates was a significant predictor of stress growth for blacks, and only gender was significant in predicting stress growth for whites ($\gamma = .91, p < .05$).

Covariates had stronger effects on the latent intercepts for depression for both racial groups. For blacks, education reduced latent baseline depressive symptoms ($\gamma = -.15, p < .001$). Black males evidenced fewer baseline depressive symptoms than black females ($\gamma = -.82, p < .001$), and functional impairment increased latent baseline symptoms ($\gamma = .51, p < .001$). For whites, all three covariates had similar signs as for blacks, but only education had a significant effect ($\gamma = -.16, p < .001$). Net of the covariates, latent baseline stress had a significant effect on latent baseline depression for both blacks and whites, but this effect was significantly greater for whites ($t = 2.42, p < .05$). Overall, the covariates and stress explained 21 percent of the variance in latent baseline depressive symptoms for blacks and 27 percent for whites, with the exception of the oldest group (with 17 percent explained variance for the oldest blacks and 74 percent for the oldest whites).

In terms of latent growth in depressive symptoms, no covariate had a significant effect for either racial group. However, stress growth
had a significant effect on depressive symptom growth for both blacks ($\gamma = .05$) and whites ($\gamma = .03$). A t-test on the difference between these parameters was non-significant. However, stress growth accounted for considerably more variance in depressive symptom growth for blacks than for whites in all but the oldest age groups ($R^2 = .67$ for blacks and $R^2 = .21$ for whites). In the oldest age group, stress growth accounted for more variance in depressive symptom growth for whites than for blacks ($R^2 = .59$ for whites, and $R^2 = .16$ for blacks). Once again, we also report the results for the model with covariates only, and these results were consistent with those for the latent depressive symptom intercept results: Most of the explained variance is attributable to stress growth and not the covariates. These results suggest (1) that stress growth is an important predictor of late-life growth in depressive symptoms and (2) that the effect of stress growth appears similar for whites and blacks, but that stress growth accounts for more between-individual variation in depressive symptom patterns for blacks than for whites, except at the oldest ages. This latter finding is likely a result of greater individual variance unaccounted for by the level one equations for stress among whites.

**DISCUSSION**

The purpose of this paper was to examine race differences in the relationship between stress and depressive symptoms over a six-year interval, using latent growth curve analysis to estimate the effects of trajectories of stress on trajectories of depression. This is a considerably different approach than has been used in previous research. Previous studies have been based on cross-sectional or short-term longitudinal data, although investigators have previously examined the effects of current or recent stress on changes in depressive symptoms, long-term patterns of change and stability in both stressors and depressive symptoms have seldom been studied.

In addition to providing a more complete view of the dynamics of stress and depression, analyses reported here present challenges to traditional ways of thinking about stress exposure and vulnerability, as well as opportunities to broaden our conceptualization and measurement of those concepts. Multiple investigators note that stress exposure has not received fully appropriate testing in previous research because the stress universe has not been adequately sampled (Wheaton 1994). Important advances have been made in conceptualizing and measuring stress exposure more broadly, but all of those efforts rest on cross-sectional or short-term longitudinal data and fail to consider long-term patterns of change in the rate of stress exposure. We contend that such long-term patterns are an additional dimension of stress exposure, and our findings suggest that growth in loss-related events predicts growth in depressive symptoms over time.

Differential vulnerability to stress is more complex in the context of latent growth curve analysis. Inclusion of appropriate covariates permits us to examine their effects on both stress growth and growth in depression—a strategy compatible with traditional views of stress vulnerability which emphasize social and personal resources as determinants of differential vulnerability. We did not include measures of social and personal resources in these analyses. The three covariates examined—gender, education, and functional status—were very poor predictors of both stress growth and depression growth, this was not a test of differential vulnerability in the traditional sense.

However, stress vulnerability can be viewed another way in latent growth curve analysis. Evidence that stress growth is a stronger predictor of increasing depression for some groups than for others can reasonably be interpreted as evidence of differential vulnerability to stress. Conceptualizing it in these terms, there was modest evidence of differential vulnerability in our results. Although the coefficients for stress growth as a predictor of growth in depressive symptoms were significant for both blacks and whites, stress growth explained more variance in depression growth for blacks than for whites.

Overall, the race differences observed in these analyses are modest. The strongest race difference was observed for the relationships between the covariates and the depression intercept. For African Americans, all of the covariates, as well as baseline levels of stress, were significant predictors of depression at baseline. For whites, education was the only significant covariate, although baseline levels of stress predicted the depression intercept, as
was true for blacks. Because the depression intercept reflects baseline levels of depression, however, these are cross-sectional results and are less important than those based on change over time. Other race differences were less strong, but significant: Stress growth exhibited a more linear pattern for blacks than for whites, as hypothesized, and stress growth explained more variance in growth of depression symptoms for blacks than for whites.

Although these analyses illustrate the importance of using long-term trajectories of stress to predict trajectories of depressive symptoms, this has been only a beginning effort. The sample was restricted to older adults living in central North Carolina. Stress was operationalized in terms of loss-related events, an issue relevant to late life where survival is typically accompanied by a loss of significant others. Other stressors, such as disability and physical health problems, also are likely to exhibit “growth” over time in samples of older adults. It also is likely that other kinds of stressors are more strongly related to growth in depression in other age groups.

This study also had methodological limitations as a result of the database available for analysis. Although these analyses are based on a longer time frame than previous studies, we would have preferred an even longer time frame, with additional times of measurement. With only three times of measurement, non-linear patterns are severely truncated. Our measure of stress growth also was truncated, in terms failing to provide a count of each type of event between waves and failing to cover the entire interval between times of measurement. We believe, however, that these problems have only negatively biased our estimation of the relationship between stress and depression. The covariates included in the analyses are only a subset of the social factors known to be related to depression in late life. In future studies, other covariates such as social support and psychosocial resources should be examined. Finally, if the analytic strategy employed here is to be used to better understand group differences in the relationship between stress and depression, future studies must not only replicate and extend the findings reported here for African Americans and whites, but they must also compare groups based on other statuses such as gender and socioeconomic status, as well as other racial/ethnic groups.

NOTES

1. Another way to capture nonlinearity is to add an additional latent growth factor and specify the t parameters for it appropriately. If an additional factor is added, the t parameters for it can be set equal to (time since baseline)^m, where m is the degree of curvature (e.g., for quadratic, m = 2). Other functions (e.g., exp(t)) could also be used. If an additional factor is not added, the t parameters for the single growth factor can be modified directly (e.g., change from 0,1,2,3 . . . to 0,1,4,9 . . . for quadratic growth). The former method is preferable but may not be possible due to identification and convergence problems. In fact, we experienced this problem here.

2. One reviewer directed us to a paper by Mehta and West (2000) that discusses several approaches to, and problems with, the specification of time in growth curve models. Using age groups, as we do, introduces error into the variance of the latent intercept and possibly the growth factors. The reason for this is quite obvious: If we assume a life course process, individuals at different ages will be at different places in this process, making the estimate of the intercept more variable than if single years of age were used. We do not define the age groups in this analysis by single years of age, but believe that the process of increasing loss-related events with age is a general one that is linked to segments of the life course, not necessarily specific ages. For example, it is unlikely that the friends and family members of 65-year-olds are significantly younger than those of 67-year-olds. Thus, the precision of single-year age groups is not needed. Also, we conducted models using multiple specifications for time and age and obtained similar results across those models.

3. The model degrees of freedom were adjusted to compensate for the fact that much of the “data” were missing and created to have a mean of 0 and a variance of 1 (see Allison 1987).

4. Throughout this section, we report the R-square values for the structural equations. Technically, these R-square values cannot be interpreted in terms of explained variance, as in OLS regression, unless the sys-
tem is recursive (the matrix of coefficients relating endogenous latent variables is subdiagonal) and the error variance matrix is diagonal (meaning no error correlations). Here, our equation system is recursive, but the error variance matrix contains error correlations between latent intercept and growth parameters for depression and for stress. We estimated additional models without these error correlations, and the R-square values for those models were similar to those reported.

5. We used a standard test for examining differences in coefficients: \( t = \frac{(b_1 - b_2)}{\sqrt{\text{se}(b_1)^2 + \text{se}(b_2)^2}} \). We attempted to estimate a full model constraining the effect of stress to be the same across races in order to conduct a chi-square difference test, but we experienced convergence problems and unreasonable estimates for numerous model parameters. This is another indication that the effects are different across races.

REFERENCES


RACE DIFFERENCES IN DEPRESSIVE SYMPTOMS


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