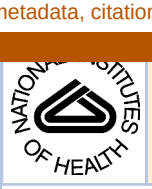

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Structure and Stress: Trajectories of Depressive Symptoms across Adolescence and Young Adulthood

Daniel E. Adkins, **Victor Wang**, and **Glen H. Elder Jr.**

Abstract

Previous research into the social distribution of early life depression has yielded inconsistent results regarding subgroup differences in depression levels and in the etiology of these differences. Using latent curve models and data from the National Longitudinal Study of Adolescent Health, this study investigates gender and racial/ethnic disparities in early life depressive symptoms and the explanatory roles of stress and socioeconomic status (SES). Results show that females and minorities experience higher levels of depressive symptoms across early life compared to males and Whites. Further, childhood SES and stressful life events (SLEs) explain much of the disparity for Blacks and Hispanics. Finally, Blacks, Hispanics, and females show greater sensitivity to the effects of low childhood SES and, in the case of females, SLEs. Overall, this study provides new insight into gender and racial/ethnic differences in the course of early life depression and in the role of the stress process during this important developmental stage.

For nearly three decades, the social determinants of depression have remained a dominant theme in the mental health literature. During this period, studies have provided greater conceptual clarity and more elaborate statistical models that have contributed enormously to our understanding of social disparities in depression. Yet, despite countless inquiries and a myriad of empirical formulations, significant gaps remain in our knowledge of how some of the established relationships develop over the life course.

There is now a sizeable body of evidence to suggest that adolescents report higher rates of depressive symptoms compared to adults (Schoenbach et al. 1983; Radloff 1991; Ge et al. 2006). However, it is becoming evident that depression is not uniformly distributed across adolescent groups. For example, studies find that elevated rates of depression among females compared to males largely emerge in early adolescence (Petersen et al. 1991; Hankin et al. 1998; Angold et al. 1998), with approximately half of adolescent females reporting some depressive symptoms during a given week (Scheidt et al., 2000). Racial and ethnic differences in depressed mood are also documented, although the direction and magnitude of these differences have been inconsistent. For example, studies show that adolescent Blacks (Garrison et al. 1990; Gore and Aseltine 2003), Hispanics (Iwata et al. 2002; Twenge and Nolen-Hoeksema 2002), Asians (Greenberger and Chen 1996), and Whites (Dornbusch et al. 1991) each exhibit higher rates of depression compared to other racial/ethnic groups. Surprisingly, little has been done to integrate these bodies of literature in a unified framework to examine how gender and racial/ethnic differences in depression develop in adolescence.

We draw upon a “stress process” framework to elucidate the antecedents of and changes in depressive affect across early life. Over the past several decades, the stress process has become the central paradigm in sociological research for explaining inequalities in adult mental health (see Pearlin et al. 1981; Thoits 1991; Turner and Lloyd 1999). From a developmental perspective, stress process models are informative because they emphasize the early social-

structuring of stress exposure and vulnerability that ultimately shape long-term patterns of psychological well-being. Indeed, several studies show that childhood socioeconomic status (SES) influences psychological health in early life (McLeod and Shanahan 1993; Costello et al. 2003 [see Case 2004]), and although stressful life events (SLEs) are also known to play a role (e.g., Turner and Avison 2003; Ge et al. 1994, 2001, 2006; Meadows et al. 2006), it is less clear how SES and SLEs exert their effects longitudinally. Recent studies attempt to address this shortcoming by examining depression "trajectories" to better understand the timing, level, and growth of negative affect along with its social etiology (e.g. Ge et al. 1994, 2006; Hankin et al. 1998; Mirowsky 1996). Despite these contributions, research has been slow to integrate the principal components of the stress process with the life course construct of a depression trajectory.

This study uses the largest nationally representative, longitudinal sample of U.S. adolescents and young adults to investigate trajectories of depressive symptoms across age. This research is the first analysis to test the major components of the stress process on age-based trajectories of early life depression by gender and race. Several key questions guide the analyses. First, how do trajectories of depressive symptoms differ by gender and race as adolescents transition to young adulthood? Second, to what extent, and through what mechanisms, does the stress process explain differences in depression trajectories? Specifically, does childhood SES explain race and gender differences in adolescent trajectories of depressive symptoms? Are gender and race differences in depressive symptom trajectories related to differential exposure and/or vulnerability to SLEs? We conclude by discussing the implications of our findings for future research.

Structure, Stress, and the Distribution of Early Life Depression

One of the most robust findings in the mental health literature is the pronounced gender difference in depression (Nolen-Hoeksema 1990). Studies suggest that gender differentials emerge in early adolescence (e.g., Allgood-Merten et al. 1990; Angold et al., 1998; Ge et al. 1994) and that rates of depression are approximately two to three times higher among females than males, regardless of the diagnostic scheme or interview method (Culbertson 1997; Nolen-Hoeksema 1990). Although some evidence suggests that elevated rates of depression among adolescent females can be traced to hormonal changes during puberty (Angold et al. 1998), the predominant explanation identifies gender discrepancies in social status and exposure and vulnerability to stress. It is argued that gendered social roles situate females in stressful environments, which produce greater exposure to stress compared to males (Turner, Wheaton and Lloyd 1995; Turner and Lloyd 1999; McLeod and Kessler 1990). Further, both adolescent and adult research suggests that women are not only more exposed to SLEs, but may also be more vulnerable to their negative effects on mental health (Ge et al., 1994; Turner and Turner, 1999). However, other experts have argued that gender differences in stress vulnerability are actually disorder specific (Aneshensel et al. 1991; Hagan and Foster 2003). Thus, it has been theorized that while females generally react to stress with internalizing symptoms, males often express an equal degree of stress response that is manifested in externalizing behaviors.

Established theory also suggests that the structural disadvantages faced by racial and ethnic minorities produce greater exposure and vulnerability to stress and consequently poorer mental health compared to their advantaged counterparts. Results from both community-level and large-scale longitudinal studies show that Blacks report higher levels of depressed mood than Whites during adolescence and young adulthood (Garrison et al. 1990; Gore and Aseltine 2003). Similar studies also demonstrate that Hispanics and Asian Americans are at an increased risk for depressive symptoms during adolescence and young adulthood compared to Whites (Gore and Aseltine 2003; Greenberger and Chen 1996; Siegel, Aneshensel, and Taub 1998; Twenge and Nolen-Hoeksema 2002). Despite the evidence supporting a minority disadvantage

in depression, some studies contradict this argument. For instance, a review of the literature by Nettles and Pleck (1996) shows that although Black youth are at greater risk for many negative behavioral and health outcomes, their rates of depression are often lower than White adolescents. Likewise, findings from a large multiethnic sample of adolescents indicate that White and Asian-American youth report significantly more depressive symptoms than Black and Hispanic adolescents (Dornbusch et al. 1991).

Inconsistent findings on the distribution of adolescent depression may well be attributed to age variations in the development of the stress process. For instance, there is now mounting evidence that depressive symptoms follow a normative pattern of increase through early and middle adolescence, peak during late adolescence, and decline in early adulthood. This conclusion is supported by longitudinal research finding curvilinear trajectories in samples of individuals aging through adolescence and young adulthood (e.g., Ge et al. 2006), as well as by research in younger samples showing linear increase through middle adolescence (e.g., Garber et al. 2002; Ge et al. 2001), and studies of young adult samples showing linear decrease or stability through the twenties (e.g., Galambos et al. 2006). Despite the collectively strong evidence for a curvilinear trajectory of depression across adolescence and young adulthood, the explanation and social distribution of this pattern are not well understood.

Theoretical Framework and Hypotheses

The *stress process* model has dominated the literature on mental health over the past several decades and remains the prevailing theoretical orientation for understanding the structure and consequences of stressful life events (Pearlin 1981; 1989). A basic premise of the stress-process model is that the social location of individuals (e.g., SES) influences stress exposure and vulnerability that, in turn, produce physical and psychological responses. Among the most salient consequences of stress for adolescents is the manifestation and progression of depressive symptoms. Despite the theoretical import of such a perspective, no studies have incorporated the central components of the stress process in trajectory analyses of early life depression. We propose several hypotheses to describe and explain why gender and race disparities in depression emerge through adolescence.

Hypothesis 1a: Female adolescents will exhibit greater levels of depression than males and the gender difference will persist across early life

Previous studies consistently show that females report higher levels of depression compared to males in both adolescence and young adulthood (Nolen-Hoeksema 1990). This gender gap has been shown to emerge in early adolescence and persist across early adulthood (Allgood-Merten et al. 1990; Ge et al. 1994). Based on these findings, we hypothesize that females will have higher levels of depressive symptoms at baseline and that the gender gap will persist across early life.

Hypothesis 1b: Minority adolescents will exhibit greater levels of depression than Whites, but this disparity will vary across racial/ethnic groups, with Blacks and Hispanics exhibiting persistent disadvantage across early life and Asians experiencing disadvantage primarily in adolescence

Although empirical findings on the topic are mixed, theoretical perspectives suggest diminished psychological well-being among minorities in early life for several reasons, some of which vary by race/ethnicity. For instance, structural theories of disadvantage suggest that the interlocking adversity represented by low SES, racial/ethnic discrimination and neighborhood disadvantage exerts a strong negative effect on minority psychological well-being (Vega and Rumbaut 1991; Ross 2000; Williams and Collins 1995). But while all of these sources of structural disadvantage commonly apply to Black and Hispanic groups, some are

less applicable to Asians. Specifically, though many Asian adolescents report experiencing racial/ethnic discrimination (Romero et al. 2007), as a group they are generally raised in comparatively affluent households buffered from the negative effects of poverty (U.S. Census Bureau 2008). However, previous research has indicated that Asians face unique psychosocial challenges in late adolescence, primarily due to parent-child conflicts arising from high parental expectations for achievement (Greenberger and Chen 1996; Lee and Liu 2001). Likewise, both Asians and Hispanics are often subject to the additional stresses associated with bicultural identity, particularly in adolescence (LaFromboise et al. 1993; Romero et al. 2007). Based on these theoretical perspectives, we predict elevated levels of depression among Asians during adolescence and a persistent disadvantage in depression across early life for Blacks and Hispanics.

Our second set of hypotheses attempts to explain gender and racial/ethnic differences in adolescent depression by incorporating the tenets of the stress process model. Perhaps the most obvious and well-documented association is the link between low SES and depression (see Lorant et al. 2003 for a review).[1] We maintain that SES is a fundamental dimension of the stress process that both structures an individual's exposure to stressful life events and influences one's vulnerability to stressors through available resources and coping mechanisms (Pearlin 1981; 1989; Link and Phelan 1995). From a developmental perspective, adolescence is a period of growth and maturation when childhood SES (i.e., parental status) can exert significant and lasting influences on well-being. Thus, our next objective is to examine how childhood SES mediates racial/ethnic disparity[2] in adolescent depression.

Hypothesis 2: Differences in childhood SES will explain a significant portion of the racial/ethnic gap in depression trajectories for Blacks and Hispanics

Countless studies show that SES is negatively associated with depression (e.g., Dohrenwend et al. 1992; Kessler et al. 1994) and an equal volume of literature shows that Blacks and Hispanics have lower mean levels of SES than Whites (see Williams and Collins 1995). Drawing from these findings, we anticipate that a significant portion of the racial/ethnic disparity in adolescent depression can be explained by SES differences between these racial groups. However, given that former research has found that racial/ethnic structural disadvantage extends beyond SES (Ross 2000; Williams and Collins 1995), we predict that childhood SES will not explain the full extent of Black and Hispanic disadvantage in early life depression.

It is often argued that gender and racial/ethnic inequalities in depression resulting from structural disadvantage are mediated through disparities in stress exposure and adaptive resources (Pearlin 1989; Kessler et al. 1999; Williams and Collins 1995). Among the mechanisms linking structure and depression, perhaps the most studied and robust predictor has been SLEs (e.g., Paykel 1978; Kendler et al. 1999; Ge et al. 2006). The general supposition of this research maintains that low SES leads to greater exposure to SLEs (Brown and Harris 1978; McLeod and Kessler 1990; Mickelson and Kubzansky 2003), which in turn, precipitate depression (Pearlin 1989). Despite the bulk of evidence linking depressed affect and various indices of SLEs among adults (e.g., Turner and Wheaton 1995), research has been slow to

[1]A valid concern in the literature is whether SES affects depression (social causation) or whether depressive disorders influence SES (social selection). Significant social selection effects have also been shown in some mental health research (e.g. Costello et al. 2003; Miech et al. 1999; Dohrenwend et al. 1992) and unfortunately, the effects of selection and causation are notoriously hard to separate in non-experimental, survey research. However, in the current study we have largely avoided the risk of confounding social selection effects through focusing on parental SES during the subject's youth. Thus, social selection effects are likely to be minimized as the children's mental health is generally unlikely to have a dramatic influence on their parents' SES, particularly given that a major component of SES —parental education, was generally determined prior to the subjects' births.

[2]Gender differences in *exposure* to low childhood SES are not examined because mean childhood SES levels are the same—there are no differences in exposure. Gender differences in *sensitivity* to low childhood SES are examined in hypothesis 4a.

focus on adolescents (see Ge et al. 2001; Goodyer, Kolvin and Gatzanis 1985). Moreover, existing studies rarely employ a developmental approach to examine the differential impact of stress exposure on depression trajectories (see Curran and Willoughby 2003). Therefore, we propose:

Hypothesis 3a: Female disadvantage in early life depression trajectories is partially attributable to the greater exposure of SLEs among females

As suggested above, research on both adolescents and young adults has indicated that females experience a higher volume of SLEs than males (e.g., Turner et al. 1995; Hankin and Abramson 2001). According to this perspective, social roles expose them to more stress than males, because females, like individuals with low socioeconomic status, are often situated in social roles where they are expected to perform less desirable tasks with little recognition or reward (Turner and Lloyd 1999; Turner and Avison 1989). Given the substantial evidence supporting this perspective, we hypothesize that a portion of the gender gap in early life depression will be due to greater exposure to SLEs among females.

Hypothesis 3b: Black and Hispanic disadvantage in early life depression trajectories is partially attributable to the greater exposure of these racial/ethnic groups to SLEs

Similar to females, we hypothesize that adolescent Blacks and Hispanics are, on average, exposed to a greater number of SLEs than White adolescents. We find support for this hypothesis in theories of structural disadvantage, which maintain that these minorities experience a greater volume of stressors than Whites due to their marginalized social location and experience of racial/ethnic discrimination (Williams, Neighbors and Jackson 2003; Williams and Collins 1995).

In many cases, however, accounting for gender and racial/ethnic differences in both SES and SLEs fails to fully explain observed health disparities (Gore and Aseltine 2003; Lillie-Blanton et al. 1996; Turner and Turner 1999). Thus, researchers have investigated alternative explanations, including possible gender and racial/ethnic differences in vulnerability to SES and SLEs (Dornbusch et al. 1991; Ge et al. 1994; Aneshensel et al. 1991). We test this argument with the following hypotheses:

Hypothesis 4a: Gender differences in adolescent depression are partially attributed to the greater tendency of females to react depressively to low childhood SES and SLEs

Research from various theoretical perspectives suggests that females respond to adversity with greater depressive affect than males. For instance, the *double jeopardy hypothesis* suggests that characteristics associated with disadvantaged social position, such as low SES and female gender, may interact to produce particularly detrimental mental health outcomes (Dowd and Bengston 1978). This explanation suggests that females generally face chronic adversity due to being situated in less desirable social roles (Turner and Lloyd 1999; Turner and Avison 1989), and that these difficulties deplete females' coping resources, leaving them less able to respond to additional sources of adversity, such as SLEs and low SES (e.g., Turner and Turner 1999). An alternative perspective suggesting greater depressive response to adversity among females is the gendered stress reactivity hypothesis (Aneshensel et al. 1991; Hagan and Foster 2003).This perspective holds that females are not globally more susceptible to the negative effects of adversity, but simply express distress differently than males. Accordingly, studies show that males are more likely to respond to adversity with externalizing behaviors and substance abuse, whereas females tend toward internalizing responses such as depression (Hagan and Foster 2003). Considering support for both perspectives, we hypothesize that females respond more depressively to adversity than males, regardless of the prevailing mechanism.

Hypothesis 4b: Racial/ethnic differences in adolescent depression are partially attributed to the greater tendency of Blacks and Hispanics to react depressively to low childhood SES and SLEs

Much research has suggested that disadvantaged minorities, like females, experience some degree of double jeopardy, or *cumulative disadvantage*, when confronted with multiple sources of adversity (see McLeod and Owens 2004). These perspectives suggest that the persistent adversity associated with minority status, such as the experience of discrimination, leaves Blacks and Hispanics with fewer buffering social and psychological resources than Whites, and thus, more vulnerable to the effects of additional adverse circumstances (e.g., Dowd and Bengston 1978; Williams and Neighbors 2001). Based on these perspectives, we predict that Black and Hispanic individuals will respond more depressively to childhood SES and SLEs than their White counterparts during early life.

Methods

Sample

We use data from three waves of the National Longitudinal Study of Adolescent Health (Add Health). A key feature of Add Health is that it includes the largest nationally representative sample of high schools in the United States with an over-sampling of racial/ethnic minorities. The baseline sample consists of 80 high schools selected with probabilities proportional to size and an additional 52 feeder middle-schools. The overall response rate for the 134 participating schools was 79 percent. Of the over 90,000 students who completed in-school surveys during the 1994–1995 academic year, a sample of 20,745 adolescents in grades 7–12 were followed over a 7-year period in 1994–1995, 1995–1996, and 2001–2002. A questionnaire was also administered to a selected residential parent of each adolescent. Further details of Add Health's sampling design, response rates, and data quality are well documented (http://www.cpc.unc.edu/projects/addhealth/design).

The analytic sample for this study consists of 18,764 native-born White, Black, Asian, and Hispanic adolescents. We dropped 9 cases due to lack of information on race, 334 Native Americans due to limited sample size, 8 cases that lacked information on depression, and 1667 first-generation immigrants due to their diverse cultural backgrounds and language capabilities (Harker 2001).

Measures

Depressive symptoms—Depressive symptoms are measured using a 9-item scale derived from the conventional 20-item Center for Epidemiologic Studies Depression Scale (CES-D) (Radloff 1977). The 20-item CES-D is composed of questions on a number of physical and psychological symptoms of depression, which cluster into four factors: Somatic-Retarded Activity, Depressed Affect, Positive Affect, and Interpersonal Relations (Ensel 1996; Radloff 1977). The scale has been validated in adult samples of Whites and Blacks (Blazer et al. 1998) and samples of adolescents and young adults (Radloff 1991). Previous factor analyses have validated the subscale used in Add Health, indicating it is invariant across subgroups and contains the four factors present in the full scale (Meadows et al. 2006). Fortunately, a 19-item CES-D was collected in the first two waves of Add Health and a comparison with the subscale (9 items) indicated a high correlation ($r = .91$ and .92 in waves one and two, respectively). Individual items are coded on a four-point scale to indicate the frequency of symptoms occurring during the past week, ranging from never or rarely (0) to most or all of the time (3). The reliability of the 9-item scale is consistent across all three waves of data ($\alpha = 0.79$, 0.80, and 0.80, respectively).

Parental SES—We include measures for parental education and household income. We use adolescent reports to determine the educational attainment of the sampled residential parent (typically mother) and use the parental questionnaire to ascertain the educational level of their current partner (typically father). Household income is ascertained from the parental questionnaire and includes all sources of income from the previous year (measured in thousands of dollars). Preliminary analyses indicated that a latent construct of the individual SES measures provided greater explanatory power and was more consistent with our theoretical framework. This approach also reduces the measurement error incurred by analyzing the indicators separately (Bollen 1989). We use mean-centered indicators of SES to assist in model interpretation[3].

Stressful life events—The current study uses an additive index to measure cumulative exposure to stressful life events. Presented in appendix 1, the SLE index used here is derived from one developed by Ge et al. (1994). Established criteria for the development of the SLE index were used in modifying and expanding the measure for the Add Health survey (Turner and Wheaton 1995). For instance, only acute events of sudden onset and of limited duration that occurred within 12 months of the interview were included (Turner and Wheaton 1995). Further, given previous research indicating that undesirable life events are more likely to adversely affect health (Compas 1987; Johnson, 1982), only negative life events were included in the index. To ensure a complete coverage of stressful events, we included almost 50 items from various domains of life (e.g., family, romantic and peer conflicts, academic problems, involvement/exposure to violence, death of family and friends). A major challenge of operationalizing our measure of SLE was to make it longitudinally accountable. As adolescents make the transition into adulthood, some stressors become irrelevant (e.g., expulsion from school) and other stressors become relevant (e.g. divorce or entering military service). Thus, to ensure stress was appropriately measured at different life stages, we used a slightly different set of items for wave III to capture the different life experiences. Finally, similar items (such as miscarriage and still birth) were grouped together to avoid making the measurement overly specific. A simple, additive index was created from the selected items and is standardized in the current analysis.

Race/ethnicity—Add Health allows respondents to indicate as many race and ethnic categories as deemed applicable. Approximately 4% of the participants report a multiracial/ethnic identity. Following criteria developed by Add Health data administrators, we assign one racial identity for persons reporting multiple backgrounds (http://www.cpc.unc.edu/projects/addhealth/data/using/code/race). This method combines Add Health's five dichotomous race variables and the Hispanic ethnicity variable as following: respondents identifying a single race are coded accordingly; respondents identifying as Hispanic were coded as such regardless of racial designation; respondents identifying as "black or African American" and any other race were designated as Black; respondents identifying as Asian and any race other than Black were coded as Asian. Sensitivity analyses comparing this coding approach with another in which only individuals identifying as one race/ethnic group were coded as such and all other individuals were coded as multiracial suggest that findings are generally robust across coding schemes.[4]

Analytic Strategy

Add Health is typical among longitudinal datasets, in that it is organized by wave of assessment with variability in chronological age at each wave. This presents a challenge to our current

[3]It is common practice with LCM to use mean-center continuous independent variables (Bollen and Curran 2006). If covariates are not mean-centered, the mean growth factors represent the trajectory for cases with values of zero on all covariates, rather than the shape of the mean trajectory for the sample.

purpose because developmental theory is clear that age is a more meaningful time metric than wave for the study of depression trajectories (e.g., Hankin et al. 1998; Ge et al. 1994). Accordingly, we restructure the three waves of Add Health data to provide age-based measurements for the trajectory analyses. Fortunately, addressing the issues associated with analyzing data that has been restructured from wave to age has been an active topic in the methodological literature on latent curve modeling and effective strategies are now well-established (e.g., Mehta and West 2000; Bollen and Curran 2006; see Muthén and Muthén 2000 for an empirical example).

The fundamental problem encountered with restructuring data from wave to age is the creation of a special case of missing data, termed "missing by design" (Muthén and Muthén 2007). Data that are missing by design are considered "missing completely at random" according to Rubin's (1976, 1987) typology of missing data. Thus, it can be analyzed using direct, or full information, maximum likelihood estimation (FIML), which requires only that the data satisfy the less restrictive "missing at random" assumption (Bollen and Curran 2006). FIML is generally considered a superior method for handling missing data as it maintains optimal properties under less restrictive assumptions than do more traditional approaches, such as listwise deletion (Allison 2002). The key idea of the FIML approach is that the likelihood function is calculated for each case using only variables that are available for that case. The total likelihood is then the sum of the likelihood values for each case (Arbuckle 1996; Bollen and Curran 2006). The FIML approach has several desirable features including the asymptotic properties of maximum likelihood estimators (e.g., consistency, unbiasedness, normality, and efficiency) and permits the calculation of asymptotic standard errors for significance testing (Arbuckle 1996). Most importantly, FIML makes use of all available information in the data without imputation—no cases are dropped or imputed and all the values for all variables available for a case are used. See Bollen and Curran (2006) chapter 3 for a more detailed treatment of converting time metrics from wave to age and handling the resulting missing data in latent curve models.

This analysis uses age-based latent curve models (LCMs), a special case of the structural equation model, to model trajectories of depressive symptoms across adolescence and young adulthood. The LCM is a flexible approach to modeling developmental trajectories, in which the observed repeated measures are considered indicators of an unobserved growth trajectory (Willet and Sayer 1994). We begin by fitting a series of unconditional growth models (i.e., age-based trajectories of depressive symptoms with no predictors) to identify the correct functional form of the trajectory. A test of various specifications (e.g., linear, quadratic, and a set of piecewise linear models) indicated that the shape of the average trajectory followed a curvilinear pattern (inverted U-shape), with depressive symptoms increasing through adolescence and declining in young adulthood. Thus, the unconditional trajectory is modeled as a quadratic function of age and is described in the following equations:

$$\text{Level 1 model:} \qquad Y_{ia}=\alpha_i+\beta_{1i}\lambda_a+\beta_2\lambda_a^2+\varepsilon_{ia}$$

[4]As shown in appendix 2, we conducted a final sensitivity analysis testing the robustness of our racial/ethnic coding scheme. In this model, instead of coding multiracial/ethnic individuals as a single race/ethnicity, we coded only individuals who designated a single race/ethnicity as such, and coded all individuals designating multiple race/ethnicities as multiracial/ethnic. The results of this analysis show that the estimates from the final, preferred model 4 were generally robust in terms of direction, significance and magnitude, regardless of the treatment of multiple race/ethnicity. The only exception to this is the SES × Hispanic interaction effect, which declined in magnitude and lost statistical significance in the sensitivity analysis. None of the multiracial effects were statistically significant, perhaps due to a relatively small sample size or heterogeneity in racial/ethnic background. Overall, the sensitivity analysis results support our use of the Add Health racial/ethnic coding scheme.

Level 2 model:

$$\alpha_i = \mu_\alpha + \zeta_{\alpha i}$$
$$\beta_{1i} = \mu_{\beta 1} + \zeta_{\beta 1 i}$$

Combined model:

$$y_{ia} = \left(\mu_\alpha + \lambda_a \mu_{\beta 1} + \lambda_a^2 \beta_2\right) + \left(\zeta_{\alpha i} + \lambda_t \zeta_{\beta 1 i} + \varepsilon_{ia}\right)$$

In the level 1 model, y_{ia} represents the CES-D measure for person i at age a; α_i represents the intercept of the growth trajectory for person i; β_{1i} represents the linear component of the slope of the trajectory for person i; β_{2i} represents the quadratic component of the slope of the trajectory for person i; λ_a represents the value of age at age a, λ_a^2 represents the squared value of age at age a, and ε_{ia} represents the age-specific residual for person i at age a. In the level two model, μ_α represents the mean (or fixed) intercept of the trajectory, $\mu_{\beta 1}$ represents the mean linear component of the slope of the trajectory; $\zeta_{\alpha i}$ represents the residual (or random component) of the intercept term for person I; and $\zeta_{\beta 1 i}$ represents the residual of the linear component of the slope term for person i. The combined model clarifies that the observed repeated measures of y can be expressed as an additive combination of a fixed component of growth ($\mu_\alpha + \lambda_t \mu_{\beta 1} + \lambda_t^2 \beta_2$) and a random component of growth ($\zeta_{\alpha i} + \lambda_t \zeta_{\beta 1 i} + \varepsilon_{ia}$).[5]

After developing an accurate model of the unconditional trajectory of depressive symptoms for the full sample, we then sequentially introduce predictors in order to test each hypothesis in the order they are presented. We begin by examining trajectory differences by gender and race/ethnicity. We then include childhood SES, assessing the degree to which it explains racial/ethnic differences in CES-D trajectories. Next, we examine the degree to which exposure to SLEs mediates the effects of gender and race/ethnicity. Cohort and cohort × age effects are then examined. Finally, we investigate the degree to which race/ethnicities and genders differ in their sensitivity to childhood SES and SLEs by introducing a series of interactions. We performed data management and descriptive statistics with Stata 9.2 and latent curve modeling with Mplus 4.2.

Results

Descriptive Statistics

Table 1 presents means and standard deviations by wave and age for the 9-item CES-D. Two notable trends are evident. First, there is a strong trend of increasing symptom levels through early adolescence, slowing in late adolescence, and declining in young adulthood. A strong cohort trend is also present, with lower symptom levels among younger cohorts. Descriptive statistics are presented for the model predictors in table 2. Similar to CES-D levels, SLEs are shown to increase in frequency across adolescence and then decline in young adulthood. Demographically, the sample is about equally split between genders and Add Health's minority oversample is apparent with Blacks and Hispanics representing higher proportions of the sample than the national population. Finally, the measures of SES show that the mean yearly

[5]One technical modeling issue worth noting is the decision to model the quadratic component of growth as fixed (i.e., set the quadratic growth factor variance equal to zero). This decision was made based on several considerations. First, while the unconditional model was estimable with the quadratic growth factor specified as random (i.e., the quadratic growth factor variance freely estimated), optimization difficulties were encountered for the more complex conditional models for this specification. Constraining the quadratic factor variance stabilized model optimization and was considered a reasonable restriction because a) the quadratic growth factor variance estimated for the unconditional model was very close to zero, b) all other parameter estimates were extremely robust regardless of whether this growth factor was modeled as fixed or random and c) the quadratic growth factor variance is of little substantive importance in the present analysis.

household income for respondents is approximately $47,000 and the mean highest parental educational attainment is slightly greater than a high school degree for both mothers and fathers.

Latent curve models of depressive symptoms

We find that a quadratic unconditional LCM of depressive symptoms best fits the data and represents a superior balance of accuracy and parsimony.[6] As shown in model 1 of table 3, this quadratic model fits the data well with highly significant growth factor means.[7] The mean R^2 for the 15 CES-D repeated measures is .48.

Model 2 tests hypotheses 1a and 1b by examining the effects of race/ethnicity and gender on depression trajectories. Dummy variables predicting each of the growth factors are included for Black, Asian, Hispanic and female, with White and male being the reference groups. Visualized in figure 1, key findings include significant, positive effects for Blacks, Hispanics and females on the intercept growth factor. Among racial/ethnic groups, only Asians differ on the slope growth factors, exhibiting a significantly higher linear slope and more negative quadratic slope. Again, the growth factor means[8] are all highly significant and indicated a curvilinear, inverted U-shaped trajectory. The average R^2 for CES-D is .48 and the R^2's for the intercept and linear slope growth factors are .06 and .05, respectively. CFI, TLI, and RMSEA fit indices all indicate good model fit. Overall, these results support hypotheses 1a and 1b, indicating that females and racial minorities experience higher levels of depression than Whites and males. Specifically, results indicate a widening then narrowing of the gap between Asians and Whites, while the gaps for Blacks and Hispanics are persistent across early life.

Model 3 examines hypothesis 2 by including childhood SES as a time invariant predictor to the race/gender model described above. Childhood SES is modeled as a confirmatory factor analysis (CFA) latent variable estimated from three indicators: father's educational attainment, mother's educational attainment and household income.[9] Key findings from this model include a large and highly significant negative effect of childhood SES on the intercept growth factor, a significant positive effect on the linear slope growth factor, and no significant effect on the quadratic growth factor. Also notable is the substantial attenuation of the Black and Hispanic intercept coefficients when including the childhood SES variable, with the value of the Black and Hispanic coefficients decreasing by 24% and 36%, respectively. Despite this attenuation, both of these coefficients remain significant. Conversely, the Asian coefficient increases with the inclusion of childhood SES by 53%, to statistically significant levels. The growth factor means are all significant and continue to indicate an inverted U-shaped trajectory. The average R^2 for CES-D is .48 and the R^2's for the intercept and linear slope growth factors are .10 and .10, respectively. Fit indices again confirm good model fit. These results support hypothesis 2, indicating that racial differences in childhood SES explain much of the White-minority gap in early life depression trajectories for Blacks and Hispanics.

Models 4 and 5 test hypotheses 3a and 3b by introducing the SLE index as a time variant predictor of CES-D. In model 4, the effects of SLEs are allowed to vary across ages. Results

[6]In this unconditional quadratic LCM, the intercept factor loadings are all set to 1, the linear slope factor loadings are set to 0, 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14 and the quadratic factor loadings are set to 0, 1, 4, 9, 16, 25, 36, 49, 64, 81, 100, 121, 144, 169, 196.

[7]Preliminary analyses indicated that constraining CES-D error variances equal across repeated measures resulted in a significant decrease in model fit; thus, in all presented models CES-D error variances are freely estimated. No model derived variance estimates are presented, but are available upon request.

[8]In this case the growth factor means only represent the White male reference group while the variances apply to the full sample.

[9]This childhood SES latent variable fit the data very well (CFI = 1.00; TLI = 1.00; RMSEA =.00) and had the following advantages over alternative specifications of childhood SES: 1) it measured the desired theoretical construct—not disparate aspects of the construct; 2) it has greater predictive power than any one of its indicators; and 3) it circumvents the problem of coefficient interpretation that arises because the three indictors are highly correlated and compete for the same growth factor variance.

from this model indicate that the effects of SLEs are large, positive, and highly significant at each age, with a modest degree of variation in effect size across ages. In model 5, the effects of SLEs are constrained equal for all ages; thus estimating only a single coefficient for the effect of SLEs on CES-D across all ages. This model again shows the effects of SLEs to be large, positive and highly significant for all ages. Other than differences in the modeling of SLE effects, parameter estimates are essentially equivalent across the two models, but fit indices indicate that the more parsimonious model 5 offers a superior fit to the data. Thus, all subsequent models use the SLEs specification used in model 5.

Substantively, model 5 shows further attenuations of the Black and Hispanic intercept coefficients, with the combined effects of childhood SES and SLEs diminishing the Black and Hispanic intercept effects by 49% and 58%, respectively. Conversely, controlling for SLEs increases the intercept coefficients for Asians and females. Despite the mediating effect of SLEs, all racial/ethnic and gender intercept coefficients remain significant. Also noteworthy is the moderate attenuation of the effects of childhood SES after the inclusion of SLEs. In the presence of SLEs, the effects of childhood SES on both the intercept and linear slope growth factors decline by approximately 20%. Thus, SLEs are indicated as a mediator of childhood SES's influence on depressive symptoms through early life. Otherwise, the model is robust to the inclusion of SLEs, with only minor changes in the growth factor means and the various measures of model fit. In sum, hypothesis 3a for females is not supported and hypothesis 3b is supported as racial differences in exposure to SLEs explain much of the racial gap in early life depressive symptom trajectories for Blacks and Hispanics.

Model 6 in table 3 examines the influence of cohort, specified as a time invariant continuous variable.[10] Large, highly significant cohort effects are present on each of the three growth factors. These results indicate that older cohorts are characterized by higher levels of depressive symptomology at all ages. Further, the trajectories of older cohorts are flatter, or less peaked, across the time interval examined. Otherwise, parameter estimates are generally consistent and model fit is slightly improved through modeling the effect of cohort.

Racial/ethnic and gender differences in the stress process of depression

To investigate whether racial/ethnic and gender groups differ in their depressive response to childhood SES and SLEs, we expand the preferred model 6 from the previous set of analyses (figure 2) to include sets of interactions. We first investigate whether the effects of childhood SES differ by racial/ethnic and gender groups in models 1 and 2 of table 4, before examining racial/ethnic and gender heterogeneity in the effects of SLEs in model 3. Model 4 presents results from the final, trimmed model with nonsignificant interactions excluded.

Results from model 1 of table 4 indicate significant negative interactions for Blacks, Hispanics, and females on the effect of childhood SES on the trajectory intercept. Other than changes in the SES effect on the trajectory intercept, parameter estimates are consistent with the former model and measures of model fit remain good. Model 2 introduces interactions between SES and race/ethnicity and gender on the linear and quadratic growth factors to test if differences in the effects of childhood SES vary in magnitude across early life. Results indicate that racial/ethnic and gender differences do not vary in magnitude, with all newly introduced interactions nonsignificant and model fit declining slightly relative to the previous model. Overall, the results of these two models indicate that, relative to Whites and males, Blacks, Hispanics, and females are more sensitive to the influence of childhood SES on depressive symptoms. Further,

[10]Preliminary analyses specifying cohort as a series of dummy variables indicated that influence of cohort on depressive symptoms is roughly linear.

the magnitude of this increased sensitivity to SES among Blacks, Hispanics, and females is constant across early life, showing no evidence of change in magnitude.

Model 3 in table 4 examines differential sensitivity to SLEs. Findings indicate a large, positive, highly significant interaction effect for females, but no significant interactions for any racial/ethnic groups. Thus, females show greater sensitivity to SLEs relative to males. Otherwise, parameter estimates are consistent and fit indices continue to indicate good fit. The last model shown, model 4, describes the overall preferred, trimmed model. Here, interactions between SES × race/ethnicity, SES × gender, and SLEs × gender are modeled along with the base model. All significant interactions from the former three models held in this model. Illustrated in figure 3, findings again indicate that Blacks, Hispanics and females show greater sensitivity to childhood SES. Compared to Whites, the estimated effects of SES for Blacks and Hispanics are 35% and 29% larger, respectively. Further, the estimated influence of SES among females is approximately 26% larger than among males. Similarly, as shown in figure 4, the estimated influence of SLEs is 94% larger among females than among males. Other than differences in the influence of SES and SLEs, parameter estimates were consistent between the final model and the base model from table 3 and all measures of fit indicate that the final model fits the data well. In sum, this set of models partially supports hypotheses 5a and 5b, as females, Blacks, and Hispanics show increased sensitivity to the effects of childhood SES and females show increased sensitivity to the effects of SLEs. However, modeling the interaction effects does not substantially narrow the gender and racial/ethnic gaps in early life depression.

Discussion

The sociological study of mental health has long emphasized the importance of the structural underpinnings of the stress-depression relationship. Research in this vein has advanced race/ethnicity, gender, and SES as the primary dimensions of inequality structuring exposure to stress and the development of depression. Although much research has demonstrated the role of these dimensions in structuring the stress-depression relationship, fundamental questions about the function of the stress process across early life have remained unanswered. Using Add Health, a large, nationally representative, longitudinal dataset with minority over-representation, this study addresses limitations in current knowledge regarding the course of gender and racial/ethnic disparity in early life depression and the role of stress and childhood SES in explaining these subgroup differences.

Results from our analyses reveal several major findings. First, females and minorities experience higher levels of depressive symptoms across early life compared to males and Whites. Second, childhood SES and SLEs explain much of the minority disadvantage in depressive symptoms for Blacks and Hispanics. Third, females, Blacks, and Hispanics react more depressively to low childhood SES than males and Whites. Fourth, females react more depressively to SLEs than males, despite the greater exposure to SLEs among males. These findings contradict previous evidence of White disadvantage in early life depression (Nettles and Pleck 1996; Dornbusch et al. 1991) and female disadvantage in SLEs exposure (Turner, Wheaton and Lloyd 1995; Turner and Lloyd 1999; McLeod and Kessler, 1990), while providing support to research on gender differences in early life depression (Nolen-Hoeksema 1990) and theories of racialized structural disadvantage (e.g., Williams and Collins 1995).

We find that trajectories of depressive symptoms are curvilinear across adolescence and young adulthood and that there are major gender and racial/ethnic variations in this pattern. Notably, mean trajectory shapes for all genders and racial/ethnic groups show an initial increase in depressive symptoms before declining in the later stages of the trajectory. Thus, our results corroborate previous findings that depression levels peak in mid to late adolescence (Wight et al. 2004; Ge et al. 2006), with trajectory apices occurring around ages 15–17. Substantively,

these findings highlight the emotional difficulties encountered in adolescence and the beneficial effects of many of the events associated with the transition to adulthood, such as achieving independence, establishing stable relationships, and an increased sense of control (Mirowsky and Ross 1992; Schieman et al. 2001).

Although this study is based on non-clinical measures of depressive symptoms, it also has important public health implications due to the strong relationship between depressive symptoms in early life and the development of clinical depression in later life (Petersen et al. 1993). In fact, as Achenbach notes (1991), depressed mood is the single most powerful symptom differentiating clinically referred and nonreferred youth. Additionally, depressive symptoms have been linked to various other psychological problems such as anxiety and social withdraw (Petersen et al. 1993). Given the elevated levels and considerable variability of symptoms observed among adolescents in Add Health, we suggest that this life stage represents a particularly good target for policy intervention to relieve psychological distress and prevent the development of more debilitating clinical disorders in later life.

As expected, females exhibit persistently higher levels of depressive symptoms than males. However, we observed a substantial narrowing of the gender gap across young adulthood in several models. While studies have indicated some narrowing of the gender gap in the transition to adulthood (Hankin et al. 1998; Ge et al. 2006), this is the first study to show such a dramatic degree of convergence. Although these results should be considered tentative due to inconsistent statistical significance across models, given the relative superiority of the data employed here over that used in former examinations, we believe this anomalous finding deserves further investigation. That said, it is important to note that the trajectories presented here should not be extrapolated beyond the ages included in the sample. Given the overwhelming evidence of gender depression differentials across adulthood (e.g., Mirowsky 1996), we suspect that any narrowing of the gender gap must either stabilize or reverse at some point in early adulthood. In sum, our findings support the longstanding finding of female disadvantage in depression (Nolen-Hoeksema 1990) and provide a more nuanced picture of the fluctuation in the gender gap across early life.

Consistent patterns of racial/ethnic disparities in depressive symptoms indicate a general advantage for Whites of both genders. Thus, theories of structural disadvantage are supported as minority status is consistently associated with reduced psychological well-being even after adjusting for childhood SES and SLEs (Williams et al. 1997; Williams and Collins 1995). However, important differences between racial/ethnic minority groups also suggest variations in the social etiology of depression across these groups.

Childhood SES is highly influential on depressive symptoms for the entire sample, and although this effect is persistent across early life, we find some evidence that it lessens over time. There are also considerable differences in childhood SES levels between racial/ethnic groups, and thus, controlling for childhood SES decreased the Black-White depressive symptoms gap by about one quarter and decreased the Hispanic-White gap by well over a third. Conversely, as Asians report a higher level of childhood SES than Whites, control of childhood SES results in an over one half increase in the Asian-White disparity. Controlling for SLEs has similar effects to SES on the racial disparity in depressive symptoms. Specifically, as Blacks and Hispanics have higher, and Asians lower, levels of SLEs than Whites, adjusting for life events further reduces White-Black and White-Hispanic disparity in early life depressive symptoms while increasing the White-Asian gap.

Collectively, controlling for childhood SES and SLEs reduces the racial/ethnic disparity by 49 percent for Blacks and 58 percent for Hispanics and increases the gap by 78 percent among Asians. These findings corroborate work by Williams and others (e.g., Williams et al. 1997;

Williams and Collins 1995) suggesting that minority well-being is undermined by several converging sources of structural adversity. Thus, while childhood SES and early life SLEs are major factors explaining the Black-White and Hispanic-White depression gaps, these factors actually obscure the Asian-White disparity. It is only after controlling for these factors that it becomes apparent that Asians experience a comparable degree of disadvantage in depressive symptoms as other racial/ethnic minority groups. Given the substantial effects of perceived racism and discrimination reported in the literature (e..g., Williams et al. 2003; Romero 2007), we suggest that these unmeasured sources of adversity likely explain much of the observed residual racial disparities.

In addition to factors common to the experiences of minorities, the Asian experience in early life is uniquely characterized by relatively elevated levels of depressive symptoms in late adolescence and sharper declines during young adulthood. This finding is consistent with previous research and likely points to the unique psychosocial challenges that Asians face in late adolescence due to parent-child conflicts arising from high parental expectations for achievement and obedience (Greenberger and Chen 1996; Lee and Liu 2001). However, unlike previous research on the topic, we were able to leverage the longitudinal design and wide age range of Add Health to model this phenomenon in life course context, showing that the relative difficulties Asians face in late adolescence fade with the transition to adulthood and the increased independence this brings (Mirowsky and Ross 1992; Schieman et al. 2001).

Beyond examining racial/ethnic differences in depression trajectories and the roles of childhood SES and SLE exposure in explaining these disparities, we also investigate the issue of differential sensitivity to childhood SES and SLEs across subgroups. Results indicate that Blacks and Hispanics are substantially more sensitive to the effects of childhood SES than Whites. This finding supports several related theories suggesting that the experience of persistent adversity, such as the experience of racial/ethnic discrimination, weakens individuals' ability to respond to additional sources of adversity—low SES in this case. For our purposes, these perspectives may be reasonably classified under the rubric of *cumulative disadvantage* (see McLeod and Owens 2004), but include a wide array of theories including double jeopardy (Dowd and Bengston 1978), the weathering hypothesis (Geronimus et al. 2006), heightened vigilance (Williams and Neighbors 2001), as well as classic stress process theory (Turner et al. 1995). Despite their differences, these perspectives are uniformly consistent with our findings in their suggestion that the depletion of personal physical and psychological resources caused by the many pernicious social factors associated with disadvantaged minority status tends to weaken individuals' ability to cope with other sources of adversity such as socioeconomic disadvantage.

Gender differences in the function of the stress process in early life depression are also evident. For instance, contradicting most previous research on the topic, we find that males experience greater exposure to SLEs than females in early life (see Turner and Lloyd 1999; McLeod and Kessler 1990). Thus, while former studies have often suggested greater SLE exposure among females as a primary explanation of the gender gap in depression, this is clearly not the case in our analysis of the Add Health data. Rather, we find that controlling for SLEs increases the gender gap by almost 25 percent. We suspect that this finding reflects elevated risk-taking tendencies among young males and the disproportionate representation of events related to such risk-taking behaviors in the SLE index used here. Specifically, due to limitations in the information available in Add Health, the SLEs include a substantial proportion of the events that are likely to be "self-generated" rather than "fateful" stressors (see Turner and Wheaton 1995). Given that young males more frequently engage in behaviors increasing the risk of self-generated stressors (Crick and Zahn-Waxler 2003), it may be that the SLE index captures stressful events more common to males than females.

Regarding gender differences in sensitivity to adversity, although occurring less frequently among females, the SLEs measured here are shown to have almost twice the impact on females compared to males. Similarly, females react more depressively to low childhood SES than males. In interpreting these findings we suggest two distinct, though not mutually exclusive, potential explanations. First, it may be the case that females, like disadvantaged minorities, experience some degree of cumulative disadvantage. This explanation suggests that women's social roles expose them to more adversity than men because women are often situated in social roles where they are expected to perform less desirable tasks with little recognition or reward (Turner and Lloyd 1999; Turner and Avison 1989). Similar to disadvantaged minorities, it is theorized that the difficulties associated with females' social roles deplete their coping resources, leaving them less able to respond to additional sources of adversity, such as SLEs and low SES (e.g., Turner and Turner 1999).

Another potential explanation lies in the possibility of gendered differences in stress reactivity (Aneshensel et al. 1991; Hagan and Foster 2003). This perspective holds that females are not more susceptible to the negative effects of adversity, but simply respond to stressors in different ways than males. Support for this perspective is found in studies showing that males are more likely to respond to adversity with externalizing behaviors and substance abuse, while females tend toward internalizing responses such as depression (Hagan and Foster 2003). Unfortunately, we can not empirically adjudicate between these potential explanations, and given that both have received substantial support in previous research, it is not clear which perspective is more compelling theoretically. It is worth noting, however, that these two explanations are not necessarily contradictory—it is possible that when faced with adversity females experience some degree of double jeopardy *and* this is expressed primarily as internalizing symptoms. We leave further investigation of this issue to future research, but note that Add Health contains the externalizing and substance abuse measures necessary for a more thorough examination of the topic.

Given the large magnitude of the gender differential in SLE sensitivity, an obvious question is whether there are specific events that males and females respond differently to. To investigate this issue we conducted additional analyses, summarized in appendix 3, in which we looked for gender × life event interaction effects on depressive symptomology for each individual life event included in the index. Interestingly, the results suggest that gender differences in SLE sensitivity are fairly pervasive across all events measured. Thus, females react more depressively to a wide range of events including experiences of violence, relationship problems, sexual abuse, academic problems, and involvement with the criminal justice system. Collectively, these results suggest that regardless of the life domain, females generally respond more depressively to stressful events in early life than males. As previously discussed, it is not clear whether this reflects heightened sensitivity to adversity among females, gendered differences in stress response, or both.

The current analyses offer some of the first comprehensive trajectory models of depressive symptoms in early life for both genders and all primary American racial/ethnic groups. However, the study is nevertheless limited in several respects. First, additional waves of data would allow further refinement and an extension of our understanding of how depressive symptoms develop over a longer period of the life course. The present investigation was limited to ages 12–26 based on three waves of data that are currently available from the Add Health study. Fortunately, the fourth wave of data collection for Add Health is now underway (http://www.cpc.unc.edu/projects/addhealth/design_focus/wave4) and will allow an elaboration of the models presented here to include participants in their late 20's and early 30's.

Another shortcoming of the study was our conceptualization of stress being limited to SLEs. It has been demonstrated that other aspects of the stress process - including chronic stressors and buffering resources - are also important components of the stress-depression relationship (e.g. Perlin 1989; McLean and Link 1994). We encourage future research to improve upon the current analyses with more exhaustive models that integrate chronic stressors and buffering psychosocial resources as predictors and mediators in the stress process. Another potential improvement in the measurement of stress could be achieved through disaggregating the SLE index into various life domains (e.g., Ge et al. 2006).

Despite these limitations, the present study improves our understanding of racial/ethnic and gender differences in early life depression trajectories and in the degree that differences in exposure and sensitivity to SLEs and low childhood SES explain these subgroup differences. Specifically, our results show that females and racial/ethnic minorities experience relatively higher levels of depressive symptoms across early life, and that females, Blacks, and Hispanics generally react more depressively to the effects of social adversity. In sum, these findings demonstrate the utility of combining large population-based data with trajectory models to investigate trends in social psychological development. As we demonstrate, this combination of data and method enables elegant and nuanced models of variation in health disparities, and their causes, across the life course. We hope future research will continue to employ this approach to further elucidate the origins and course of these disparities.

Appendix 1. List of Items in Stressful Life Events Index

Wave I, II, and III items	**Wave I and II items only**
Death of a parent	Was expelled from school
Suicide attempt resulting in injury	Suffered a serious injury
Friend committed suicide	Father received welfare
Relative committed suicide	Mother received welfare
Saw violence	Was raped
Threatened by a knife or gun	Ran away from home
Was shot	Nonromantic sexual relationship ended
Was stabbed	Suffered verbal abuse in a romantic relationship
Was jumped	Suffered physical abuse in a romantic relationship
Threatened someone with a knife or gun	Suffered verbal abuse in a nonromantic sexual relationship
Shot/stabbed someone	Suffered physical abuse in a nonromantic sexual relationship
Was injured in a physical fight	
Hurt someone in a physical fight	**Wave III items only**
Unwanted pregnancy	Evicted from residence, cutoff service
Abortion, still birth, or miscarriage	Entered full time active military duty
Had a child adopted	Discharged from the armed forces
Death of a child	Cohabitation dissolution
Romantic relationship ended	Received welfare
Had sex for money	Involuntarily dropped from welfare
Contracted a STD	Marriage dissolution
Skipped necessary medical care	Baby had major health problems at birth

Wave I, II, and III items	Wave I and II items only
Juvenile conviction	Death of a romantic partner
Adult conviction	Death of a spouse
Served time in jail	

Appendix 2. Sensitivity Analysis Comparing Racial/ethnic Coding Schemes

	Mutually exclusive race/ethnicity Coding		Race/ethnicity coding with multiracial/ethnic category	
Parameter	**Estimate**	**SE**	**Estimate**	**SE**
Means				
Intercept (α)	4.47***	0.21	4.48***	0.21
Linear slope (β)	0.26***	0.07	0.26***	0.07
Quadratic slope (β^2)	−0.03***	0.00	−0.03***	0.00
Time invariant predictors				
Black → α	0.40**	0.16	0.44**	0.16
Asian → α	0.76*	0.36	0.79*	0.38
Hispanic → α	0.53**	0.21	0.60*	0.31
Multiracial → α			0.37	0.23
Female → α	1.77***	0.13	1.77***	0.13
Black → β	−0.05	0.05	−0.06	0.05
Asian → β	0.21*	0.11	0.22*	0.11
Hispanic → β	0.03	0.07	0.14	0.10
Multiracial → β			0.02	0.07
Female → β	−0.01	0.04	−0.01	0.04
Black → β^2	0.00	0.00	0.01	0.00
Asian→ β^2	−0.02*	0.01	−0.02*	0.01
Hispanic → β^2	0.00	0.00	−0.01	0.01
Multiracial → β^2			0.00	0.01
Female → β^2	−0.01*	0.00	−0.01*	0.00
SES → α	−0.34***	0.06	−0.35***	0.06
SES → β	0.03	0.02	0.04	0.02
SES → β^2	0.00	0.00	0.00	0.00
Cohort → α	0.31***	0.07	0.31***	0.07
Cohort → β	−0.06***	0.01	−0.06***	0.01
Cohort → β^2	0.01***	0.00	0.01***	0.00
Time variant predictors				
SLE → CES-D	0.69***	0.03	0.69***	0.03
Interactions				
SES×Black → α	−0.12*	0.05	−0.14**	0.05

	Mutually exclusive race/ethnicity Coding		Race/ethnicity coding with multiracial/ethnic category	
Parameter	**Estimate**	**SE**	**Estimate**	**SE**
SES×Asian → α	0.08	0.11	0.10	0.11
SES×Hispanic → α	−0.10*	0.05	−0.05	0.08
SES×Multiracial → α			−0.07	0.06
SES×Female → α	−0.09*	0.04	−0.09*	0.04
SLE×Female → CES-D	0.65***	0.04	0.65***	0.04
R^2				
α		0.13		0.13
β		0.17		0.18
y	0.49	0.11	0.49	0.11
Fit Indices				
CFI		0.93		0.93
TLI		0.92		0.92
RMSEA		0.10		0.10

Note: SES = childhood socioeconomic status; SLE = stressful life events index; CES-D = Center for Epidemiologic Studies Depression Scale, 9 item subscale.

* p < .05

** p < .01

*** p < .001

Appendix 3. OLS Regression of Female × Individual SLE on CES-D

	Wave I			Wave II			Wave III		
SLE items	**Coef.**		**SE**	**Coef.**		**SE**	**Coef.**		**SE**
Death of a parent	0.43		0.25	0.04		0.28	0.11		0.17
Suicide attempt resulting in injury	1.13	*	0.48	0.59		0.62	0.09		0.32
Friend committed suicide	−0.29		0.26	−0.20		0.31	0.09		0.32
Relative committed suicide	0.68		0.47	−0.04		0.56	2.02	**	0.56
Saw violence	0.47	**	0.10	−0.10		0.14	0.85	**	0.23
Threatened by a knife or gun	0.70	**	0.12	0.31	*	0.15	1.00	**	0.23
Was shot	−1.10	*	0.44	0.91		0.53	1.09		0.74
Was stabbed	0.43	*	0.18	0.78	**	0.27	1.95	**	0.65
Was jumped	0.55	**	0.12	0.52	**	0.17	0.80	**	0.30
Threatened someone with a knife or gun	0.82	**	0.18	1.03	**	0.21	1.70	**	0.52
Shot/stabbed someone	0.44		0.28	0.98	**	0.34	3.71	**	0.89
Was injured in a physical fight	0.69	**	0.16	0.02		0.27	1.51	**	0.27
Hurt someone in a physical fight	0.44	**	0.06	0.56	**	0.21	1.57	**	0.25

	Wave I			Wave II			Wave III		
SLE items	**Coef.**		**SE**	**Coef.**		**SE**	**Coef.**		**SE**
Unwanted pregnancy	NE			−1.58		2.21	0.40	**	0.17
Abortion, still birth, or miscarriage	NE			NE			0.22		0.15
Had a child adopted	NE			NE			NE		
Death of a child	NE			NE			NE		
Romantic relationship ended	0.47	**	0.10	0.20		0.12	−0.01		0.11
Had sex for money	−0.78		0.99	3.31		2.00	1.62	**	0.41
Contracted a STD	0.58		0.39	0.17		0.32	0.11		0.20
Skipped necessary medical care	0.39	**	0.11	0.49	**	0.12	0.13		0.11
Juvenile conviction	−0.43		0.61	−0.42		0.75	0.00		1.27
Adult conviction	0.53		0.32	0.72	*	0.37	0.61	*	0.31
Served time in jail	0.56		0.32	0.73	*	0.37	0.74		0.48
Was expelled from school	0.53	**	0.20	0.37		0.44	NA		
Suffered a serious injury	0.28	*	0.12	0.42	**	0.15	NA		
Father received welfare	−0.17		0.28	0.28		0.34	NA		
Mother received welfare	−0.02		0.14	0.16		0.18	NA		
Was raped	0.88	**	0.27	0.46		0.42	NA		
Ran away from home	0.38	*	0.16	0.03		0.22	NA		
Nonromantic sexual relationship ended	0.54	*	0.25	0.52		0.29	NA		
Suffered verbal abuse in a romantic relationship	0.00		0.13	0.18		0.14	NA		
Suffered physical abuse in a romantic relationship	0.18		0.19	0.49	*	0.22	NA		
Suffered verbal abuse in a nonromantic sexual relationship	0.71	*	0.30	0.61		0.32	NA		
Suffered physical abuse in a nonromantic sexual relationship	0.25		0.42	0.49		0.50	NA		
Evicted from residence, cutoff service	NA			NA			−0.20		0.46
Entered full time active military duty	NA			NA			−0.15		0.44
Discharged from the armed forces	NA			NA			−0.24		1.61
Cohabitation dissolution	NA			NA			0.31	**	0.12
Received welfare	NA			NA			1.41	**	0.41
Involuntarily dropped from welfare	NA			NA			1.00		0.64
Marriage dissolution	NA			NA			−0.41		0.50
Baby had major health problems at birth	NA			NA			0.05		0.76
Death of a romantic partner	NA			NA			−0.26		1.36

	Wave I			Wave II			Wave III		
SLE items	**Coef.**		**SE**	**Coef.**		**SE**	**Coef.**		**SE**
Death of a spouse	NA			NA			NE		

NA = not applicable, item was not collected. NE = did not estimate due to no variation in interaction term.

* p<.05

** p<.01

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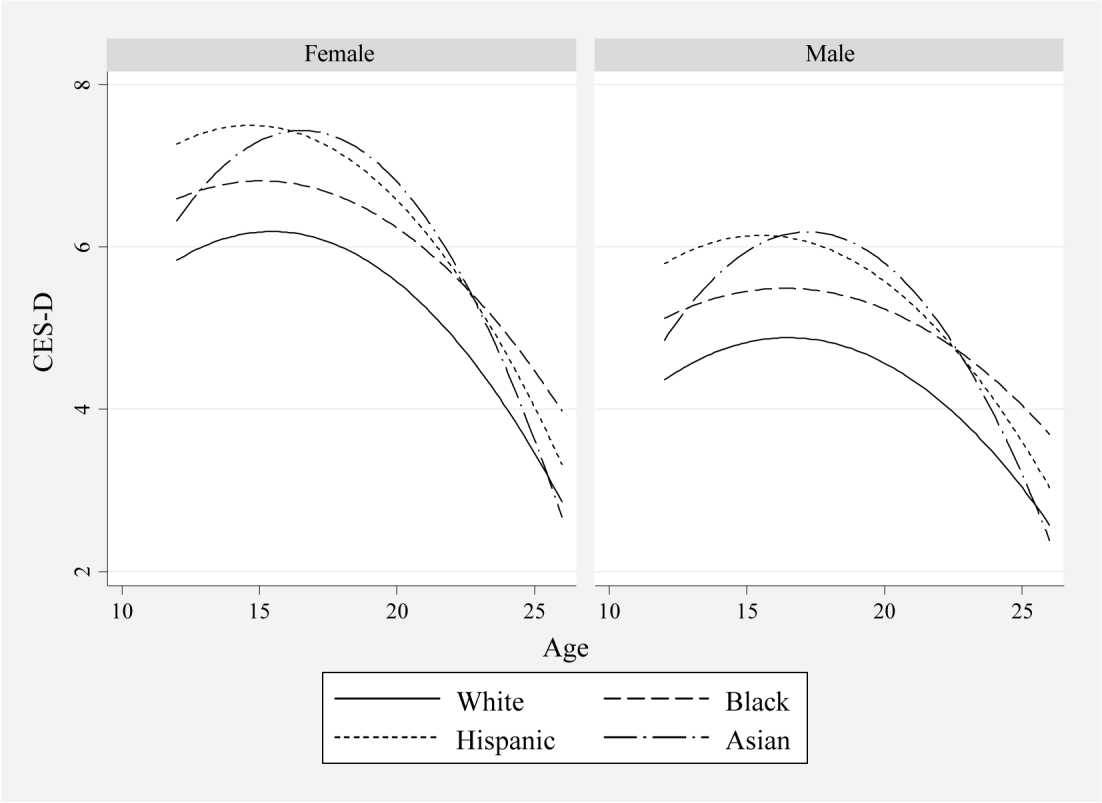

Figure 1.
Predicted CES-D Trajectories by Gender and Race/ethnicity

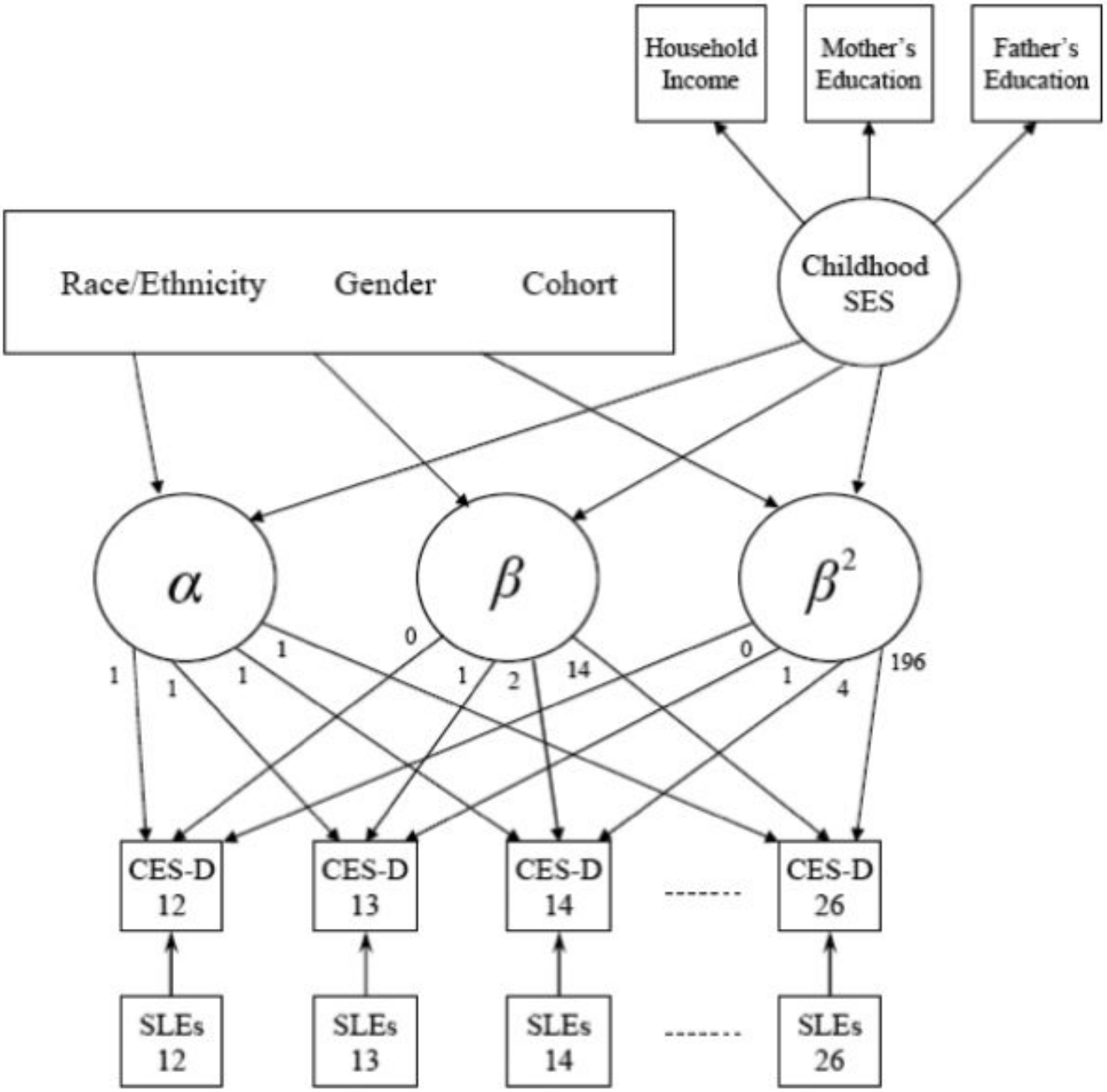

Figure 2.
Simplified Path Diagram of Preferred Model without Interactions

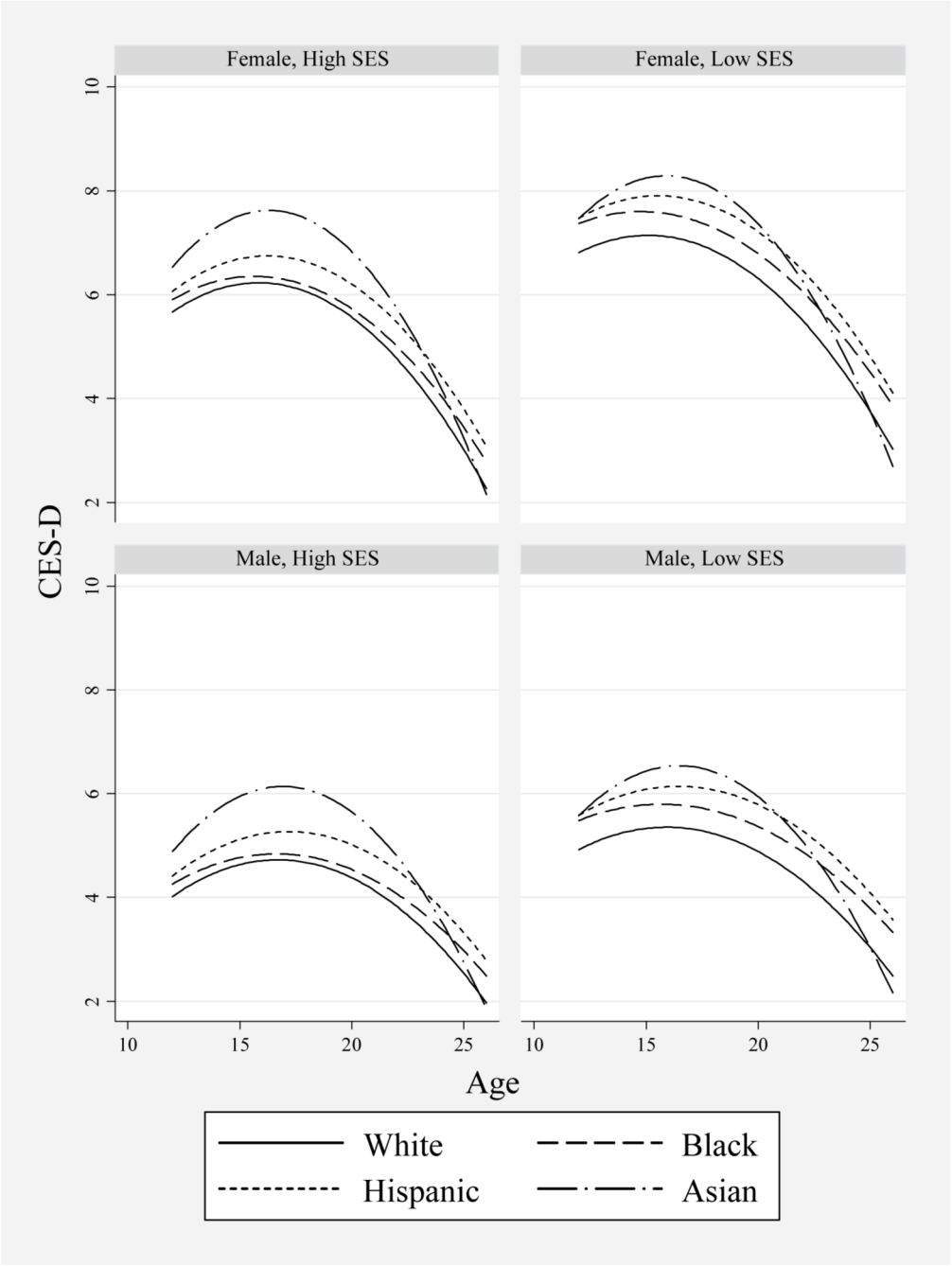

Figure 3.
Predicted CES-D Trajectories by Gender, Childhood SES and Race/ethnicity

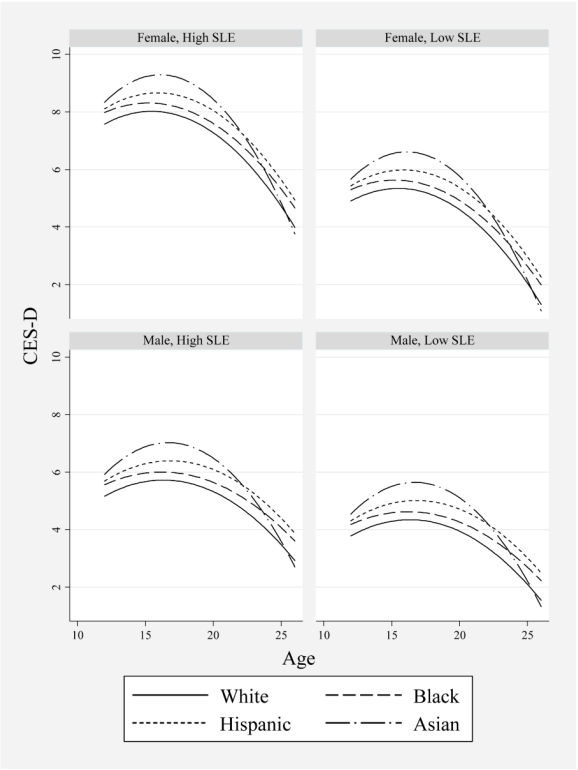

Figure 4.
Predicted CES-D Trajectories by Gender, SLE and Race/ethnicity

Table 1

CES-D Means and Standard Deviations by Age and Wave (N=18,764)

	Wave I (1994–5)			Wave II (1995–1996)			Wave III (2001–2002)		
Age	N	Mean	SD	N	Mean	SD	N	Mean	SD
12	482	4.86	3.65	9	5.44	4.10			
13	2121	4.93	3.84	577	4.62	3.63			
14	2602	5.56	4.26	1800	5.05	4.07			
15	3354	5.92	4.23	2186	5.68	4.29			
16	3654	6.24	4.33	2797	6.01	4.25			
17	3460	6.29	4.34	2921	6.13	4.24			
18	2569	6.16	4.29	2170	6.08	4.31	121	4.56	3.83
19	402	6.67	4.29	795	6.33	4.55	1331	4.63	4.01
20	54	7.00	4.64	141	7.23	4.79	1888	4.71	4.18
21	10	5.80	4.08	19	5.42	3.93	2296	4.54	4.06
22							2586	4.73	4.17
23							2629	4.60	4.09
24							2184	4.37	4.02
25							713	4.56	4.02
26							102	5.65	4.63

Note: CES-D = Center for Epidemiologic Studies Depression Scale, 9 item subscale.

Table 2

Means, Standard Deviations and Range for Model Predictors (N=18,764)

Variable	Mean/%	SD	Min	Max
SLE 12	1.68	1.88	0	11
SLE 13	1.83	2.24	0	21
SLE 14	2.04	2.46	0	21
SLE 15	2.33	2.76	0	22
SLE 16	2.55	2.89	0	22
SLE 17	2.60	2.85	0	22
SLE 18	2.42	2.71	0	20
SLE 19	2.01	2.41	0	25
SLE 20	1.66	1.90	0	14
SLE 21	1.60	1.73	0	15
SLE 22	1.70	1.83	0	15
SLE 23	1.69	1.76	0	13
SLE 24	1.65	1.71	0	10
SLE 25	1.63	1.67	0	9
SLE 26	1.86	1.66	0	8
Female	50.7%		0	1
White	57.5%		0	1
Black	22.7%		0	1
Asian	3.8%		0	1
Hispanic	14.1%		0	1
Household Income	46.687	51.956	0	999
Mother’s Education	5.599	2.408	0	9
Father’s Education	5.484	2.323	0	9

Note: SLE = stressful life events index; CES-D = Center for Epidemiologic Studies Depression Scale, 9 item subscale

Table 3

Parameter Estimates of Latent Growth Curve Models of CES-D (N=18,764)

	Model 1		Model 2		Model 3		Model 4		Model 5		Model 6	
Parameter	**Estimate**	**SE**	**Estimate**	**SE**	**Estimate**	**SE**	**Estimate**	**SE**	**Estimate**	**SE**	**Estimate**	**SE**
Means												
Intercept (α)	5.56***	0.07	4.36***	0.12	4.48***	0.12	4.22***	0.11	4.22***	0.11	4.41***	0.21
Linear slope (β)	0.20***	0.02	0.23***	0.04	0.22***	0.04	0.27***	0.04	0.27***	0.04	0.27***	0.07
Quadratic slope (β^2)	−0.03***	0.00	−0.03***	0.00	−0.03***	0.00	−0.03***	0.00	−0.03***	0.00	−0.03***	0.00
Time invariant predictors												
Black→α			0.76***	0.16	0.58***	0.16	0.42*	0.16	0.39*	0.16	0.44***	0.16
Asian→α			0.49	0.36	0.75*	0.36	0.86*	0.35	0.87*	0.35	0.86*	0.35
Hispanic→α			1.43***	0.21	0.91***	0.22	0.61**	0.21	0.60**	0.21	0.61**	0.21
Female→α			1.47***	0.14	1.47***	0.14	1.78***	0.13	1.80***	0.13	1.82***	0.13
Black →β			−0.06	0.05	−0.05	0.05	−0.07	0.05	−0.05	0.05	−0.06	0.05
Asian→β			0.28*	0.11	0.25*	0.11	0.23*	0.11	0.23*	0.11	0.21*	0.11
Hispanic→β			−0.03	0.07	0.00	0.07	0.01	0.07	0.02	0.07	0.01	0.07
Female→β			−0.02	0.04	−0.03	0.04	−0.01	0.04	−0.03	0.04	−0.03	0.04
Black→β^2			0.01	0.00	0.01	0.00	0.01	0.00	0.00	0.00	0.01	0.00
Asian→β^2			−0.02**	0.01	−0.02**	0.01	−0.02*	0.01	−0.02*	0.01	−0.02*	0.01
Hispanic→β^2			0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Female→β^2			0.00	0.00	0.00	0.00	−0.01*	0.00	−0.01	0.00	−0.01	0.00
SES→α					−0.56***	0.06	−0.47***	0.06	−0.46***	0.06	−0.44***	0.06
SES→β					0.05**	0.02	0.03*	0.02	0.04*	0.02	0.04*	0.02
SES→β^2					0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Cohort→α											0.32***	0.07
Cohort→β											−0.06***	0.01
Cohort→β^2											0.01***	0.00

	Model 1		Model 2		Model 3		Model 4		Model 5		Model 6	
Parameter	**Estimate**	**SE**	**Estimate**	**SE**	**Estimate**	**SE**	**Estimate**	**SE**	**Estimate**	**SE**	**Estimate**	**SE**
Time variant predictors												
SLE→CES-D age12							0.72***	0.03				
SLE→CES-D age13							0.89***	0.07				
SLE→CES-D age 14							1.07***	0.06				
SLE→CES-D age 15							0.96***	0.05				
SLE→CES-D age 16							0.99***	0.05				
SLE→CES-D age 17							1.06***	0.05				
SLE→CES-D age 18							1.05***	0.06				
SLE→CES-D age 19							1.06***	0.08				
SLE→CES-D age 20							0.95***	0.09				
SLE→CES-D age 21							0.82***	0.08				
SLE→CES-D age 22							0.85***	0.08				
SLE→CES-D age 23							0.84***	0.08				
SLE→CES-D age 24							0.77***	0.08				
SLE→CES-D age 25							0.66***	0.14				
SLE→CES-D age 26							1.01*	0.48				
SLE→CES-D ages 12–26									0.95***	0.02	0.94***	0.02
R^2												
α			0.06		0.10		0.11		0.11		0.13	
β			0.05		0.10		0.09		0.08		0.18	
Y	0.48	0.12	0.48	0.12	0.48	0.12	0.48	0.11	0.48	0.11	0.49	0.11
Fit Indices												
CFI	0.93		0.94		0.94		0.94		0.94		0.94	
TLI	0.95		0.93		0.94		0.91		0.91		0.94	
RMSEA	0.02		0.02		0.02		0.02		0.01		0.01	

Note: SES = childhood socioeconomic status; SLE = stressful life events index; CES-D = Center for Epidemiologic Studies Depression Scale, 9 item subscale.

* $p < .05$

** $p < .01$

*** $p < .001$

Table 4

Parameter Estimates of Latent Growth Curve Models of CES-D: Testing Interactions (N=18,764)

	Model 1		Model 2		Model 3		Model 4	
Parameter	**Estimate**	**SE**	**Estimate**	**SE**	**Estimate**	**SE**	**Estimate**	**SE**
Means								
Intercept (α)	4.40***	0.21	4.40***	0.21	4.48***	0.21	4.47***	0.21
Linear slope (β)	0.27***	0.07	0.27***	0.07	0.26***	0.07	0.26***	0.07
Quadratic slope (β^2)	−0.03***	0.00	−0.03***	0.00	−0.03***	0.00	−0.03***	0.00
Time invariant predictors								
Black→α	0.43**	0.16	0.43**	0.16	0.41**	0.16	0.40**	0.16
Asian→α	0.79*	0.36	0.56	0.41	0.85*	0.35	0.76*	0.36
Hispanic→α	0.58**	0.21	0.54*	0.22	0.55**	0.21	0.53**	0.21
Female→α	1.82***	0.13	1.84***	0.13	1.77***	0.13	1.77***	0.13
Black→β	−0.06	0.05	−0.06	0.05	−0.05	0.05	−0.05	0.05
Asian→β	0.21*	0.11	0.27*	0.13	0.21*	0.11	0.21*	0.11
Hispanic→β	0.01	0.07	0.03	0.07	0.04	0.07	0.03	0.07
Female→β	−0.03	0.04	−0.03	0.04	−0.01	0.04	−0.01	0.04
Black→β^2	0.01	0.00	0.01	0.00	0.00	0.00	0.00	0.00
Asian→β^2	−0.02*	0.01	−0.02*	0.01	−0.02*	0.01	−0.02*	0.01
Hispanic→β^2	0.00	0.00	0.00	0.01	0.00	0.00	0.00	0.00
Female→β^2	−0.01	0.00	0.00	0.00	−0.01*	0.00	−0.01*	0.00
SES→α	−0.32***	0.06	−0.32***	0.09	−0.44***	0.05	−0.34***	0.06
SES→β	0.04*	0.02	0.03	0.03	0.04*	0.02	0.03	0.02
SES→β^2	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Cohort→α	0.31***	0.07	0.31***	0.07	0.31***	0.07	0.31***	0.07
Cohort→β	−0.06***	0.01	−0.06***	0.01	−0.06***	0.01	−0.06***	0.01
Cohort→β^2	0.01***	0.00	0.01***	0.00	0.01***	0.00	0.01***	0.00

	Model 1		Model 2		Model 3		Model 4	
Parameter	**Estimate**	**SE**	**Estimate**	**SE**	**Estimate**	**SE**	**Estimate**	**SE**
Time variant predictors								
SLE→CES-D	0.94***	0.02	0.94***	0.02	0.72***	0.03	0.69***	0.03
Interactions								
SES×Black→α	−0.13**	0.05	−0.02	0.14			−0.12*	0.05
SES×Asian→α	0.07	0.11	0.38	0.29			0.08	0.11
SES×Hispanic→α	−0.11*	0.06	−0.12	0.16			−0.10*	0.05
SES×Female→α	−0.14***	0.04	−0.21*	0.11			−0.09*	0.04
SES×Black→β			−0.03	0.05				
SES×Asian→β			−0.07	0.10				
SES×Hispanic→β			0.01	0.05				
SES×Female→β			−0.03	0.04				
SES×Black→β^2			0.00	0.00				
SES×Asian→β^2			0.00	0.01				
SES×Hispanic→β^2			0.00	0.00				
SES×Female→β^2			0.00	0.00				
SLE×Black→CES-D					−0.09	0.05		
SLE×Asian→CES-D					−0.05	0.10		
SLE×Hispanic→CES-D					−0.04	0.06		
SLE×Female→CES-D					0.66***	0.04	0.65***	0.04
R^2								
A	0.13		0.13		0.13			0.13
B	0.18		0.19		0.18			0.18
y	0.49	0.11	0.49	0.11	0.49	0.11	0.49	0.11
Fit Indices								
CFI	0.94		0.94		0.93			0.93
TLI	0.94		0.93		0.93			0.94
RMSEA	0.01		0.01		0.01			0.01

Note: SES = childhood socioeconomic status; SLE = stressful life events index; CES-D = Center for Epidemiologic Studies Depression Scale, 9 item subscale.

* $p < .05$

** $p < .01$

*** $p < .001$