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

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Abstract

This paper models trajectories of depressive symptoms across adolescence and young adulthood. Using data from the National Longitudinal Survey of Adolescent Health, latent curve models (LCM) examined variation in mean depressive symptom trajectories, and the effects of childhood socioeconomic status (SES) and stressful life events (SLE), across racial/ethnic and gender groups. It was found that while all groups exhibit a curvilinear inverted U-shaped trajectory, significant variation between groups exists in both trajectory intercepts and slopes. For all racial/ethnic groups women exhibited higher depression levels. Racial disparity was found for both genders, with whites generally exhibiting lower levels than minorities. Childhood SES was primarily influential on the intercept component of the LCM and its effects, particularly on the trajectory slope components, were substantially mediated by SLE. The effects of SLE were found to be of fairly consistent magnitude across ages. Overall, the results indicate considerable heterogeneity among racial-gender groups for both normative depression trajectories and covariate effects of SES and SLE.

There is now a substantial body of research indicating elevated rates of depressive symptomology in adolescence relative to adulthood (e.g. Schoenbach et al. 1983; Radloff 1991; Wade and Cairney 1997; Ge et al. 2006). Thus, research has shown that 49% of age 15 adolescent females and 34% of males experience weekly depressive symptoms (Scheidt et al., 2000), and it is clear that such symptoms are not uniformly distributed across the population. However, progress has been made toward elucidating the causes and course of depression and depressive symptomology across early life. For instance, replicated findings have suggested that the gender difference in depression emerges in early adolescence (e.g. Petersen et al. 1991; Nolen-Hoeksma and Girgus 1994; Hankin et al. 1998; Angold et al. 1998). Further, an increasing body of literature has suggested a normative curvilinear trajectory of depressive symptomology, rising through early and middle adolescence, peaking in the late adolescence and declining or leveling in early adulthood (e.g. Ge et al. 1994, 2006; Hankin et al. 1998; Mirowsky 1996).

While advances have been made in modeling depression trajectories across adolescence and young adulthood, the body of literature is still relatively small and significant gaps in knowledge remain. For instance, investigations of racial/ethnic differences in adolescent/young adult depression have yielded inconsistent results with studies variously finding higher rates among Blacks (Garrison et al. 1990; Gore and Aseltine 2003), Hispanics (Iwata et al. 2002; Twenge and Nolen-Hoeksema 2002), and whites (Dornbush et al. 1991). Additionally, little research has examined the influence of childhood socioeconomic status (SES) on depression trajectories, despite ample motivating cross-sectional evidence of the SES's influence on mental health in early life (Genetian and Miller 2002; Costello et al. 2003 [see Case 2004]). Finally, though some

studies have examined the effects of stressful life events (SLE) on adolescent depression trajectories (e.g. Ge et al. 1994, 2001, 2006), it is known that SLE are substantially influenced by more distal components of the stress process, such as SES (Perlin 1989; Brown and Harris 1978; McLeod and Kessler 1990; Turner and Lloyd 1999) and this mediating relationship has not yet been examined with trajectory models.

The general intent of the present study was to develop more comprehensive models of depressive symptomology trajectories across the ages 12-26. Specifically, we improve on former small sample research through using a large, nationally representative dataset to examine the influences of gender, race/ethnicity, childhood SES and SLE on the course of early life depression. Our aims were to (a) characterize difference between racial/ethnic and gender groups in normative trajectories of depressive symptoms across ages 11-27, (b) examine racial/ethnic and gender variation in the influence of childhood SES and SLE on trajectories of depressive symptoms, and (c) assess the degree to which SLE mediates the effects of childhood SES. To achieve these aims we employed latent growth curve models (LCMs). Taking advantage of the large sample with minority over-representation, we stratified analysis by racial-ethnic group and gender to allow investigation of effect heterogeneity between groups.

Normative Development, Race/Ethnicity, Gender and Depressive Symptoms

Normative trajectories of depressive symptoms

Though relatively few longitudinal studies of the development of depression during adolescence and young adulthood have been conducted, there is mounting evidence of a normative curvilinear trajectory of depressive symptomology through early life. This

conclusion is supported by longitudinal research finding curvilinear trajectories in samples of individuals moving through adolescence and young adulthood, as well as by research in younger samples showing linear increase through adolescence and studies of young adult samples showing linear decrease or stabilization through the twenties. For instance, analyzing eleven waves of longitudinal data covering ages 12-23, Ge et al. (2006) found curvilinear trajectories of depressive symptoms rising in early and mid adolescence and declining in late adolescence. Likewise, Wight et al. (2004) examined depressive symptoms in three datasets (one adolescent sample and two adult samples) and found increasing levels in the adolescent sample, while the adult samples showed both lower initial levels and a steady decline over time. Using a comparable methodology, Wade et al. (2002) found increasing symptom levels across the ages 11-17 followed by plateauing and decline for ages 17-21 in panel data from three countries. Similar findings of curvilinear trajectories of depressive symptoms across early life have been found in several other analyses (e.g. Poulin et al. 2005; Hankin et al. 1998; Ge et al. 1994). Further, research in adolescent-only samples has shown increasing symptom levels across the teen years (Measelle et al. 2006; Garber et al. 2002; Ge et al. 2001), while analyses of young adult samples have shown linear decrease across ages 18-25 (e.g. Galambos et al. 2006). Cumulatively, the literature strongly indicates an inverted-U curvilinear trajectory of depressive symptoms across adolescence and young adulthood.

Race/Ethnicity and Depressive Symptoms

While much theory suggests that the structural disadvantage endured by minorities is likely to engender stress and poorer mental health, the empirical research to date has been

inconsistent. For instance, Siegel, Aneshensel, and Taub (1998) found that compared to Whites, Blacks or Asian Americans, Hispanic adolescents (ages 12-17) reported more depressive symptoms, even controlling for SES. Further, several other studies have also indicated relatively high levels of depressive symptoms among Hispanics (Gore and Aseltine 2003; Iwata, Turner, and Lloyd 2002; Twenge and Nolen-Hoeksema 2002). Similarly, numerous empirical analyses have indicated comparatively high levels of depression among young African-Americans. In a review of community studies of adolescent depression, Fleming and Offord (1990) reported that in two of five studies, Black adolescents had higher rates of depression and depressed mood than Whites, a finding that has found further support in other studies of adolescents (Garrison et al. 1990) and young adults (Gore and Aseltine 2003). Greenberger and Chen (1996) found that ethnic differences in depressed mood, not evident in the early adolescent sample, emerged in the college sample, with Asian Americans reporting more symptoms compared to White Americans. Other studies examining Asian ethnic groups have also found that these minority groups experience greater levels of depression compared to Whites (e.g. Lam, Pepper, and Ryabchenko 2004; Greenberger et al. 2000).

These findings fit well with current theory as racial minorities are believed to be exposed to more stressors, and to have access to fewer buffering resources, due to structural disadvantages (Williams and Collins 1995; Geronimus et al. 2006; Williams et al. 2003; Kessler et al. 1999). As high levels of stress are known to negatively impact mental health (Cohen, Kessler and Gordon 1997), minorities may reasonably be hypothesized to suffer higher levels of depressive symptoms than the Whites. A major portion of this increased exposure to stress likely derives from racial SES disparities, and

indeed, controlling for SES generally decreases racial disparity in health (Williams et al. 1997; Lillie-Blanton et al. 1996). However, while controlling for SES attenuates racial differences in health outcomes, it rarely eliminates them, leading many researchers to consider other complementary causes such racial discrimination and neighborhood disparities (Williams et al. 1997). Both of these mechanisms have found some support in the literature with Williams et al. (1997) finding significant effects for perceived discrimination. Further, it is known that minorities are disproportionately located in low SES neighborhoods and it has been shown that residence in such neighborhoods is negatively associated with mental health even after controlling for personal characteristics (Ross 2000; Wheaton and Clarke 2003). Similarly, Gore and Aseltine (2003) have indicated that heightened depressed mood among Hispanic and Black high school students is influenced by increasingly disadvantaged pathways into adulthood, characterized by poorer prospects for educational advancement and more problematic relationships.

Although minority disadvantage in depressive symptomology is theoretical plausible and supported by many empirical analyses, there is also substantial body of research contradicting this conclusion. For instance, Nettles and Pleck (1996) reviewed several studies and concluded that although African-American youth are at greater risk for many negative behavioral and health outcomes, rates of depressive symptoms in African-American samples are typically lower than in White youth. In a study of one of the largest multiethnic samples of adolescents, Dornbush et al. (1991) reported that White and Asian-American youth reported more depressive symptoms than African-American or Hispanic-American adolescents, even after controlling for level of stressful life events.

Given such contradictory findings, it is clear that further investigation of racial/ethnic disparity in depressive symptoms is needed and the large, nationally representative minority oversample of the Addhealth data is well-suited for this task.

Gender and Depressive Symptoms

The significant gender difference in depression among adults is one of the most robust findings in the mental health literature (Nolen-Hoeksema 1991). Rates of depression are approximately two to three times higher among women than men cross-culturally, regardless of diagnostic scheme or interview method (Culbertson 1997; Nolen-Hoeksema 1987). This gender differential emerges in early adolescence between the ages 11–15 (e.g. Allgood-Merten et al. 1990; Angold et al., 1998; Ge et al. 1994). Given the emergence of the gender differential during puberty, sex hormones have been considered a likely influence. While some research has supported this hypothesis (Angold et al. 1999), other evidence has not (Yonkers et al., 2000). Other explanations of the gender difference in depression focus on gender differences in the volume of experienced stressors. Some scholars suggest that women's social roles expose them to more stress than men, arguing that women, like individuals with low SES, are often situated in more stressful social environments (Turner and Lloyd 1995, 1999; McLeod and Kessler, 1990). Further, both adolescent and adult research suggests that women are not only more exposed to SLE, but are also more vulnerable to their negative effects on mental health (Ge et al., 1994; Turner and Turner, 1999). However, other experts have argued that the ostensible increased vulnerability to stress among women is actually an artifact caused by failure to distinguish first episodes from recurrence (Kessler 2003). Thus, further research

is needed to resolve whether women have greater sensitivity to stress and the trajectory methods used here, which control baseline depression levels, are well-suited for this task.

Childhood Socioeconomic Status, Stressful Life Events and Depressive Symptoms

Childhood Socioeconomic Status and Depressive Symptoms

Despite consistent findings of association between SES and depression (Lorant et al. 2003 for meta-analysis), recent research has stressed the importance of distinguishing the causal direction of this relationship, or as it is commonly phased in the literature—distinguishing social causation from social selection[1]. While most research on the topic has been non-experimental and unable to allow strong causal inference, the small body of experimental and quasi-experimental evidence to date has generally offered support for social causation in mental health outcomes, though not precluding selection effects (Genetian and Miller 2002; Costello et al. 2003 [see Case 2004]). For instance, Costello et al. (2003) examined data from the Great Smoky Mountains Study, in which a casino opened midway thorough the study giving every American Indian an income supplement. This exogenous shock raised 14% of sample families out of poverty, resulting in a significant reduction in emotional (i.e. depression and anxiety) symptoms for the children transitioning out of poverty. This and other analyses using robust analytic approaches have indicated substantial social causation effects.

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

[1] The social causation model posits that stress associated with low SES leads to increased levels of depression; while the social selection hypothesis asserts that individuals suffering from depression are more likely to drift into, or fail to rise out of, poverty (Dohrenwend et al. 1992).

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 parent's SES, particularly given that a major component of SES—parental education, was generally determined prior to the subjects' births.

Investigating the influence of childhood SES on trajectories of depressive symptoms is further complicated by the problematic nature of operationalizing SES. SES can be measured in several ways (e.g. education level, household income, occupational status) and former research has found that no one variable captures its full effect (Goodman 1999; Duncan et al. 2002; Gilman et al. 2003). Thus, it is generally held that an optimal approach should consider a variety of indicators. The AddHealth data is well-suited in this regard, including various measures of parental SES including educational levels and household income. Thus, after preliminary analyses comparing the effects of the individual indicators, we have assessed the aggregate effect of SES using a latent variable approach. Using a single factor confirmatory factor analysis, we conceptualized SES as the intercorrelation of the various SES indicators. This approach is known to reduce measurement error in the data relative to analyzing the indicators separately (Bollen 1989).

Stressful Life Events and Depressive Symptoms

In the past 30 years many studies have examined the influence of recent SLE on depression, providing consistent evidence of a significant effect (e.g. Paykel 1978;

Costello 1982; Kendler et al. 1999; Ge et al. 2006). While most of this research has examined adult samples, similarly consistent findings have also been found among children and adolescents (e.g. Goodyer, Kolvin and Gatzanis 1985, 1987; Goodyer, Wright and Altham 1990). For instance, using an index of 43 SLE, Ge et al. (2001) found that SLE were highly predictive of depressive symptoms in both genders across 7th to 12th grades. While the consistency of association between event accumulation and disorder clearly demonstrate that SLE indexes yield meaningful estimates of stress exposure (Turner and Wheaton 1995; Thoits 2006), debate remains regarding gender differences in sensitivity to SLE (Dornbush et al. 1991; Ge et al. 1994; Aneshensel et al. 1991). The methods employed in the current investigation will allow an examination of this issue.

Recently, there have been attempts to statistically model the principles of stress process theory through incorporate both proximate risk factors like SLE, and distal ones like SES, in more comprehensive models of environmental risk in depression. The guiding hypothesis of this literature is that poverty leads to increased stress, both in chronic stress and acute SLE, which in turn precipitate depression (Pearlin 1989). This literature has robustly shown that SLE are more prevalent among low SES individuals (e.g. Brown and Harris 1978; McLeod and Kessler 1990; Mickelson and Kubzansky 2003). Further, cross-sectional research has generally shown that the SES effect is partially mediated by SLE (Turner and Lloyd 1999; Turner and Butler 2003). However, this mediating relationship has not yet been examined in the context of trajectory analyses, which correspond more closely to developmental theory (Curran and

Willoughby 2003). This lack highlights the need for further research developing more complete models of proximate and distal environmental risk in depression.

Methods

Sample and procedures

Data from the three waves the National Longitudinal Study of Adolescent Health (Add Health) was used to develop our depressive symptom trajectory models. Add Health is a nationally representative, school-based sample of 20,745 adolescents in grades 7-12 surveyed during the 1994–1995 academic year. The sampling frame consisted of all high schools in the United States. A total of 80 high schools were selected with probabilities proportional to size and a sample of 52 feeder middle schools was attached to the sample of high schools. The response rate for the 134 participating schools was 78.9%. Of the over 90,000 students completed the in-school survey in 1994 a baseline sample of 20,745 adolescents was selected for further data collection. The adolescents were interviewed three times during a 7-year period in 1994–1995, 1995–1996, and 2001–2002. The overall sample is representative of United States schools with respect to region of the country, urbanicity, school type (e.g., public, parochial, private non-religious, military, etc.), and school size. Members of ethnic minority groups were over-sampled. Further details regarding the sample are available at http://www.cpc.unc.edu/projects/adhealth/. Among all the cases in the sample, 9 cases were dropped from the sample due to lack of information on race; 334 Native Americans were dropped because of their problematically small sample size; 1667 first generation immigrants were deleted due to diverse cultural backgrounds and variable language capabilities (Harker 2001); and 8

cases were deleted due to lack of information on depression at any wave. Thus, the analysis sample consists of 18,764 native born Whites, Blacks, Asians and Hispanics.

Measures

Depressive symptoms. The depressive symptoms scale is a 9-item derivative of the CES-D (Radloff, 1977, 1991). Previous research has shown the 20-item CES-D to cluster into four subfactors—somatic-retarded activity, depressed affect, positive affect, and interpersonal relationships. All four components are represented in the 9-item scale used here. Individual items are coded on a four-point scale, from never or rarely (0) to most or all of the time (3) and refer to feelings the respondent had in the past week. The CES-D 9-item scale is consistent across all three waves ($\alpha = 0.79$, wave one; $\alpha = 0.80$, wave two; $\alpha = 0.80$, wave three). The raw score means for the entire Add Health sample by wave are 5.91, 5.80, and 4.60, respectively.

Parental socioeconomic status. Variables measuring resident parent's (generally, the mother's) education originate in the student surveys. Each respondent reports on the highest level of education that his or her resident parent completed. From this information, the variable describing the mother's educational attainment was derived. Additionally, mothers reported their education and that of their current partner, which were used to create a measure father's education. The household income measure was also taken from the parental questionnaire. Income was measured in thousands of dollars of household income in the previous year. Respondents are instructed to include their own income, the income of everyone else in their household, and income from welfare

benefits, dividends, and all other sources. After preliminary analyses of the effects of the individual SES indicators, we moved to a latent variable approach which corresponded move closely to our theoretical premise and also provided greater explanatory power (Appendix 1). SES indicators were mean-centered to aid in model interpretation[2].

Stressful life events. The index of SLE presented in Appendix 2 is derived from the measures developed by Ge et al. (1994). A major challenge of developing the current measure of SLE is to make it longitudinally accountable. As adolescents make the transition into adulthood, a number of stressors become irrelevant (e.g., expelled from school), however a number of new stressors become appropriate (e.g. divorce, entering the military service). To ensure stress is correctly measured at different life stages, we used a slightly different set of items for wave III to capture the different life experiences. Complying with the most common practice for comparability (Turner and Wheaton 1997), the current study selected only the events happened less than a year before the interview. Further, only acute events of sudden onset and of limited duration were included. Similar items (such as miscarriage and still birth , or dissolution of sexual non-romantic relationship, romantic relationship, cohabitation, marriage) were grouped together to avoid making the measurement overly specific, at the same time insuring having enough number of events to form a relatively continuous measurement. Simple, additive indices were then created with raw score means for the entire Add Health sample

[2] In LCM if all covariates are mean-centered the growth factor means describe the overall mean trajectory shape; if covariates are not mean-centered the growth factor means represent the trajectory shape for cases with values of zero on all covariates—as this information is generally less substantively important, it is common practice to mean-center continuous covariates (Bollen and Curran 2006).

by wave equal to 2.6, 1.95, and 1.64, respectively. The indices were standardized in the data analysis.

Race/ethnicity. In keeping with the new census policy, Add Health respondents were allowed to mark as many race/ethnicity categories as they felt applied to them. Approximately 4% of the sample identified as multi-racial/ethnic. Given this, we used the coding method used by the Add Health data manager as a way to obtain mutually exclusive race/ethnicity category for the primary analysis. Thus, a single race is assigned to those reported multiple racial/ethnic backgrounds using the following criteria: if the respondent reported single race/ethnicity, he/she will be coded as is; if the respondent reported more than one race, only one race will be selected from the races the respondent reported in the following order: Hispanic, Black, Asian and White. In a sensitivity analysis, a reduced sample composed of only individuals identifying as one race/ethnicity was used and results were compared for robustness.

Age-based trajectory approach

While developmental theory posits age as the appropriate metric in the study of longitudinal change, the Add Health data is not organized by age, but by wave. Thus, given the substantial age variation within each wave of Add Health (table 1), it was necessary to reorganize the data from wave to age in order to address our research aims.[3] This reorganization resulted in a considerable amount of missing data, which was addressed by consolidating adjacent years for ages with less data coverage. In cases

[3] Sensitivity analysis examining the possibility of cohort effects were conducted and no evidence of cohort effects were found.

where there are two data points in the consolidated age interval, the mean of these values is considered the interval value. This procedure resulted in a moderate consolidation of data (8,076 observations, 17.6% of all person-years). Thus, in the restructured data, the unit of age ranges from one to five years, resulting in age intervals: 11-15, 16-17, 18, 19-21, and 22-27.[4] The mean age of the span was used to represent age in statistical models. After this data management, the remaining missing data is addressed using the full-information, or direct, maximum likelihood (FIML) method as recommended by Bollen and Curran (2006). FIML estimates a likelihood function for each individual based on the variables that are present in the data so that all the available data are used. FIML only requires that missingness is not correlated to the dependent variable after controlling for the other variables in the model (i.e. "missing at random"). FIML has been shown to be more efficient and less biased than alternative methods for handling missing data such as listwise deletion, pairwise deletion, and mean imputation (Wothke, 2000).

<Table 1 about here>

Analytic strategy

To model trajectories of depressive symptomology during adolescence and the transition to young adulthood, we employed the latent curve model (LCM), a special case of the structural equation model. 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; Curran 2000). We began by

[4] This specification of age intervals was chosen as it conserved the most person-years among the possible specifications with adequate covariance coverage for model estimation. Sensitivity analysis testing alternate specifications of age intervals (e.g. 11-14, 15-17, 18, 19-21, 22-27) showed no significant differences in the model parameters.

modeling an unconditional LCM in which there were no predictors in order to identify the correct functional form of the trajectory. After testing various functional forms, it was determined that the average trajectory shape followed a curvilinear, inverted U-shaped pattern, with depressive symptomology rising through adolescence and declining in young adulthood. Thus, the unconditional trajectory was modeled as a quadratic function of time.[5] This unconditional model is described in the following equations:

Level 1 model: $$y_{it} = \alpha_i + \beta_{1i}\lambda_t + \beta_{2i}\lambda_t^2 + \varepsilon_{it}$$

Level 2 model: $$\alpha_i = \mu_\alpha + \zeta_{\alpha i}$$

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

$$\beta_{2i} = \mu_{\beta 2} + \zeta_{\beta 2 i}$$

Combined model: $$y_{it} = (\mu_\alpha + \lambda_t\mu_{\beta 1} + \lambda_t^2 \mu_{\beta 2}) + (\zeta_{\alpha i} + \lambda_t\zeta_{\beta 1 i} + \lambda_t^2\zeta_{\beta 2 i} + \varepsilon_{it})$$

In the level 1 model, y_{it} represents the depression measure for person i at time point t; 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_t represents the value of time at time point t, λ_t^2 represents the squared value of time at time point t, and ε_{it} represents the time specific residual for person i at time t. 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; and $\mu_{\beta 2}$ represents the mean quadratic component of the slope

[5] It is important to note that the LCM estimates a distinct trajectory for every case in the sample. Given this, a further advantage of specifying a quadratic functional form is its flexible ability to model not only various curvilinear trajectories among sample cases, but also cases exhibiting patterns of linear or no change.

of the trajectory; $\zeta_{\alpha i}$ represents the residual (or random component) of the intercept term for person I; $\zeta_{\beta 1i}$ represents the residual of the linear component of the slope term for person I and $\zeta_{\beta 1i}$ represents the residual of the quadratic 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 ($\mu_\alpha + \lambda_t\mu_{\beta 1} + \lambda_t^2 \mu_{\beta 2}$) and a random component of growth ($\zeta_{\alpha i} + \lambda_t\zeta_{\beta 1i} + \lambda_t^2\zeta_{\beta 2i} + \varepsilon_{it}$).

After developing an accurate model of the unconditional trajectory of depressive symptomology, we then sequentially introduce predictors, comparing the fit indices of these nested models to select the best fitting model. In this initial model selection analysis, dummy variables were created for each race/ethnicity gender combination. Thus, we first examine the effect of race/ethnicity and gender before examining childhood SES and SLE. In addition to generally testing of the degree to which each additional set of variables improves overall model fit, the sequential entering of covariates allowed an examination of the mediating effects of SLE on SES and of the optimal specification of SLE effects. After identifying the best fitting model for the entire sample, we then stratified the sample by race/ethnicity and gender group and reran this model (minus the race/ethnicity gender dummy variables) for each subgroup in order to probe for differences in the effects of childhood SES and SLE. All analyses were performed in Stata 9 and Mplus 4.2.

Results

Descriptive statistics and bivariate correlations

Table 2 presents means and standard deviations for the analysis variables by race/ethnicity and gender. For both father's and mother's educational attainment a racial/ethnic ordering was found in which Asian had the highest values, followed by Whites, Blacks and lastly Hispanics for both genders. For household income the racial/ethnic ordering was similar: Asian, White, Hispanic and Black. Values on SES variables were generally comparable across genders with the exception household income among Asians in which females reported significantly higher values than males. For all race-gender subgroups the five repeated measures of depressive symptomology show a pattern of moderate increase across the younger ages, peaking at ages 16-18 and relatively sharp decline from ages 18-27. There is a gender gap for all race, with females have noticeably higher values. There is also a clear racial difference with Whites showing lower values at all ages. The depressive symptom means suggest a narrowing of the gender gap over time and also a narrowing of the racial disparity for females. The SLE repeated measures showed some similarities to the depressive symptomology profile with increase in the early teens, peaking at ages 16-17, and decline from 18-27. However, females showed noticeably lower SLE means than males and Asians were the most advantaged racial/ethnic group followed by Whites, Blacks and Hispanics.

<Table 2 about here>

As seen in table 3 bivariate correlations indicate a generally autoregressive pattern for depressive symptomology and SLE repeated measures, with correlations positive and significant within repeated measures, but declining as a function of time elapsed between repeated measures. Depression and SLE repeated measures were also correlated with one another, with correlations strongest between measures from the same age interval and

declining as a function of time elapsed between measures. Childhood SES variables were positively and highly intercorrelated and they were significantly negatively correlated with depressive symptoms and SLE at each repeated measurement—these correlations diminished as age increased.

<Table 3 about here>

Latent curve models of depressive symptomology

To model trajectories of depressive symptomology, we began by examining a series of unconditional LCMs to identify the correct functional form of the growth curve. In preliminary analyses we compared various specifications including a simple linear model, various splines, and two polynomial (i.e. quadratic and cubic) functions. Analyses showed that the quadratic model fit the data well and represented a superior balance of accuracy and parsimony. In this unconditional quadratic LCM, the intercept factor loadings are all set to 1, the linear slope factor loadings are set to 0, 3.5, 5, 7, 12 and the quadratic factor loadings are set to 0, 12.25, 25, 49, 144. The factor loadings reflect the uneven spacing of the age intervals with the mean age of the interval used to represent age. As shown in model 1 of table 4, this quadratic model fit the data well with all growth factor means and variances strongly significant[6]. Thus, for the full sample depressive symptomology repeated measures are well-modeled as a curvilinear trajectory with values rising early in the trajectory, before declining in the mid and later sections. The significant growth factor variances indicate significant variability around this mean trajectory. The R^2 for the depressive symptomology repeated measures ranged from .46-.53

[6] Preliminary analyses indicated that constraining depressive symptomology error variances equal across repeated measures resulted in no significant decrease in model fit; thus, in all presented models this specification is used.

<Table 4 about here>

Next, as shown in model 2 of table 4, we introduced a battery of race/ethnicity-gender dummy variables as time invariant predictors. Thus, each race/gender group was modeled as a dummy variable (resulting in eight variables, seven of which were included in the analysis with White males removed as the reference group) predicting the growth factors. While race/gender differences are examined more thoroughly in the next set of analyses stratified by race/gender group, there are a few patterns worth noting here. Most appreciably, all race/gender groups had significantly higher intercept values than the White male reference group and intercept values were typically higher for females than males. Other male racial/ethnic groups did not differ significantly from White males on either the linear or quadratic slope growth factors. All females differed from White males on the linear slope factor and only Asian females differed on the quadratic slope factor. Again, the growth factor means[7] and variances were all highly significant and indicated a curvilinear, inverted U-shaped trajectory. The depressive symptomology R^2's range from .47-53 and the R^2's of the intercept, linear slope and quadratic slope growth factors are .11, .03, and .02, respectively. As shown in the model comparison given in table 5, the inclusion of race/gender dummy variables as time invariant predictors resulted in an improvement in all fit indices and a statistically significant likelihood ratio test (LRT) ($\Delta \chi^2 = 909.85$ with 21 *df* , $p<.01$), indicating this model to fit significantly better the unconditional model.

The effects of childhood SES were assessed by including childhood SES as a time invariant predictor to the race/gender model described above. Childhood SES was

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

modeled as a confirmatory factor analysis (CFA) latent variable estimated from three indicators: father's educational attainment, mother's educational attainment and household income; as shown in Appendix 3, this childhood SES latent variable fit the data very well (χ^2 (df) = 19.81 (2); CFI = 1.00; RMSEA =.02) and had various advantages over alternative specifications[8]. In the depressive symptomology growth curve childhood SES had a highly significant negative effect on the intercept growth factor, a significant positive effect on the linear slope growth factor and no significant effect on the quadratic growth factor (model 3, table 4). This indicates that the influence of childhood SES on depression trajectories is strong in the early teen years, but this influence weakens over time (as the influence of the quadratic growth factor is predominant in the later portion of the trajectory). Thus, as childhood SES increases the starting point of the depressive symptomology trajectory drops precipitously, while the slope of the early section of the trajectory increases moderately. The effects of the race/gender dummy variables were slightly attenuated by the inclusion of childhood SES, but the significance of these effects was generally robust. The growth factor means and variances were all significant and continued to indicate a curvilinear, inverted U-shaped trajectory. The depressive symptomology R^2's range from .47-53 and the R^2's of the intercept, linear slope and quadratic slope growth factors are .20, .05, and .02, respectively. As shown in table 5, the inclusion of childhood SES as a time invariant predictor resulted in an improvement in model fit with a statistically significant LRT ($\Delta \chi^2$ = 556.41 with 3 *df* , p<.01), indicating this model to fit significantly better the model 2.

[8] The latent variable approach offered the following advantages over using the SES indicators themselves: 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 (Appendix 1); 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.

In model 4 we introduce the SLE index (hereafter SLE) as a time variant predictor of the repeated measures of depressive symptomology. As shown in table 4, the effects of SLE are large, positive and highly significant at each age interval. Also, the SLE coefficient values decline slightly as age increases. Of particular note is the attenuation of the effects of childhood SES caused by the inclusion of SLE. In the presence of SLE the effect of childhood SES on the intercept growth factor declined by approximately 20% and the SES effect of the linear slope factor declined by approximately 40%—becoming statistically nonsignificant. Thus, SLE are indicated as a principal mediator of childhood SES's influence on depressive symptoms through early life and the magnitude of this mediation increases over time. However, as shown in figure 1, though reduced by the inclusion of the SLE measure, childhood SES's direct effect on the intercept factor remains large and significant. Otherwise, the model was robust to the inclusion of SLE, with little change in the significance of the race/gender effects or the growth factor means and variances. Depressive symptomology R^2's range from .46-52 and the R^2's of the intercept, linear slope and quadratic slope growth factors are .23, .05, and .03, respectively. As shown in table 5, the inclusion of SLE as a time variant predictor resulted in an improvement in model fit with a statistically significant LRT ($\Delta \chi^2 = 1843.92$ with 5 *df*, $p<.01$), indicating this model to fit significantly better the model 3.

<Figure 1 about here>

In the final model of this first set of analyses we tested whether the effects of SLE could be constrained equal across age intervals. As seen in model 5 of table 4, this resulted in a SLE coefficient estimate which was positive, highly significant and close to the mean of the five coefficients for the freed SLE coefficient estimates shown in model

4. Otherwise, there was virtually no change in the other model parameters or the R^2's. Turning to the model comparison in table 5, we see that constraining SLE coefficients equal across age intervals does not clearly worsen the fit of the model. While the LRT does indicate a marginally poorer fitting model ($\Delta \chi^2 = 16.47$ with 4 *df*, $p<.01$), it is known that this test exaggerates nested model differences when sample sizes are as large as present (Bollen 1989; Bollen and Curran 2006). Further, when comparing the models' sample-size adjusted Bayesian Information Criterion fit index (Adj. BIC), an index known to be more robust at larger sample sizes than the χ^2, the models are shown to fit identically (Δ Adj. BIC = .18). On balance, given the extremely large analysis sample size, the marginal significance of the LRT p value and the equivalence of all other fit indices, we felt the increased parsimony of the equal SLE justified its selection as the best fitting model of the series.

Probing for racial/ethnic and gender differences

Having selected our preferred model (figure 2), we then moved to probe for race/gender differences, particularly in the effects of SLE and SES, by fitting the preferred model to sub-samples stratified by race/gender group. Table 6 shows results for males as a whole and stratified by race/ethnicity. For all males groups fit indices indicated good overall model fit and depressive symptomology R^2's indicate good component model fit[9]. Growth factor R^2's were considerably lower than in the previous set of models, highlighting race/gender group as the best predictor of the depressive symptomology trajectory. The mean trajectories for males (all and stratified by

[9] Though there was some variation in depressive symptomology R^2's with component fit best for Hispanics and worst for Black males.

race/ethnicity) are described by the growth factor means in table 6 and are presented graphically in figure 3. Here the results from the first set of analyses are corroborated, showing the mean trajectories for male racial/ethnic groups to differ primarily in intercept. Thus, all male groups show a generally curvilinear pattern of early rising and mid/late decline, but Whites are shown to have lower predicted values at ever age. Other notable characteristics displayed in figure 3 include a precipitous drop late in the trajectory for Hispanic males and a relatively high trajectory for Asian males. Growth factor variances were fairly comparable across race/ethnicities with the only the intercept growth factor evidencing significant variance for all groups.

<Table 6 about here>

<Figure 2 about here>

<Figure 3 about here>

There was some heterogeneity in the effects of childhood SES among male racial/ethnic groups. For all male groups except Asians childhood SES had a significant negative effect on the intercept growth factor—again indicating childhood SES as a very influential protective factor against depressive symptomology. However, the magnitude of childhood SES's influence varied considerably across racial/ethnic groups, with the coefficient smaller among White males, larger among Hispanics and largest among Blacks. There were no significant effects of childhood SES on the linear and quadratic slope growth factors for any male groups. The effects of SLE on depressive symptomology were large, positive and significant among all racial/ethnic groups. There was also racial/ethnic heterogeneity in the magnitude of the SLE effects, with Whites showing smaller coefficients, followed by Asians, Blacks, and Hispanics. Thus, as

shown in Figure 4, disadvantaged male minorities (Blacks and Hispanics) show greater sensitivity to SLE than White and Asian males.

<Figure 4 about here>

Results for the full model fitted to female subsamples are presented in table 7. For all females groups fit indices indicated good overall model fit and depressive symptomology R^2's indicate good component model fit[10]. Growth factor R^2's were fairly low, again indicating race/gender group as the best predictor of the depressive symptomology trajectory. The mean trajectories for females (all and stratified by race/ethnicity) are described by the growth factor means in table 7 and are presented graphically in figure 5. Here all female groups, other than Asians, exhibit curvilinear trajectories of monotonic, accelerating decline. Asian females evidence an inverted U-shaped trajectory more similar to that of the male groups. Trajectory shapes for non-Asian female groups differ primarily in the intercept. Asians females exhibited a unique trajectory distinguished by increasing depressive symptomology levels in the early portion of the trajectory, steeper decline in later ages, and an overall more curvilinear shape. The trajectory for Hispanic females is also notable for its extremely high intercept and more accelerated decline—thus, while the mean intercept for Hispanic females is over a point higher the other racial/ethnic groups, by the end of the age span examined their mean levels have converged with that of the other groups. Growth factor variances for non-Asian females were fairly comparable with the only the intercept growth factor evidencing significant variance. Asian females showed larger variances for all growth factors and also showed statistically significant variance in the quadratic growth factor.

[10] Though there was some variation in depressive symptomology R^2's with component fit best for Asians and worst for Hispanic females.

<Table 7 about here>

<Figure 5 about here>

There was considerable heterogeneity in the effects of childhood SES among female racial/ethnic groups. For Asian females, there were no significant effects of childhood SES on any of the depressive symptomology growth factors. All non-Asian female groups evidenced large, negative effects of childhood SES on the intercept growth factor of similar magnitude and significance. The effect of childhood SES on the linear growth factor was significant (and positive) only for White females and there were no significant effects of SES on the quadratic slope growth factors for any female group. The effects of SLE on depressive symptomology were large, positive and significant among all female racial/ethnic groups. There was racial/ethnic heterogeneity in the magnitude of the SLE effects, with Blacks showing smaller coefficients, followed by Asians, Whites, and Hispanics. As shown in Figure 4, females show markedly greater sensitivity to SLE than males across racial/ethnic group.

Discussion

Despite repeated calls for more comprehensive modeling of depression in early life, research to date has generally lacked the statistical power to thoroughly examine the influence of race/ethnicity, gender, and the stress process on trajectories of depression. The present investigation aimed to address this shortcoming using Add Health, a large, nationally representative, longitudinal dataset with minority over-representation. Using latent growth curve models we examined racial/ethnic and gender differences in the

effects of childhood SES and SLE on trajectories of depressive symptomology across the ages 11-27. The following findings emerged from these analyses.

Depressive symptomology trajectories are curvilinear for all racial/ethnic and gender groups. However, major gender and racial/ethnic differences are evident in these curvilinear trajectories. On average, females have persistently higher levels of depressive symptomology than males in every race/ethnicity group. The mean trajectory shape also differs between females and males, with females showing monotonic, accelerating decline and males showing initial increases before decline in the mid and late portions of the depressive symptomology trajectory. Thus, the longstanding finding of female disadvantage in depression was supported (Nolen-Hoeksema 1987, 1990) and the age marking the beginning of this disparity is indicated to occur prior to age 13.

Consistent patterns of racial/ethnic disparity in depressive symptomology were evident and indicated a general advantage for Whites of both genders. Thus, theories of structural disadvantage were supported as minority status was consistently associated with reduced psychological well-being even after adjusting for childhood SES and SLE (Williams et al. 1997; Williams and Collins 1995). Asians and Hispanics were shown to be the most disadvantaged racial/ethnic groups over the majority of trajectory. However, Hispanics of both genders showed strong evidence of converging with Whites toward the end of the trajectory, suggesting adolescence is a particularly difficult period for Hispanics, but after navigating the transition to adulthood they typically experience greater improvements in psychological well-being relative to other racial/ethnic groups. One potential explanation of this finding may be found in the *assimilation optimism hypothesis*, suggesting that the transition into school is marked by initially taxing cultural

adjustments, followed by adaptation as young adult Hispanics move into mainstream society (Kao and Tienda 1995; Landale, Oropesa and Llanes1998).

Childhood SES was shown to be highly influential on the level but not shape of depression trajectories in all groups but Asians, where it was shown to have no significant influence. Given our exogenous conceptualization of socio-economic environment as childhood SES, these findings provided strong evidence that the direction of the effect here is SES→depression, lending support to social causation theories of depression (e.g. Link and Phelan 1995; Mirowsky and Ross 2003). Further, there were gender differences in the effect of childhood SES, with females being far more influenced than males, indicating multiplicative disadvantage for low SES females. In line with stress process theory, the influence of childhood SES on trajectories of depressive symptomology was shown to be partially mediated by SLE across the ages examined (Perlin 1989; House and Williams 1995). Thus, we find that childhood SES has large, protective, direct impact on depression trajectories and also a substantial indirect effect through reducing the likelihood of SLE occurrence. This corroborates research positing the occurrence of stressful events as primary paths through which poverty reduces psychological well-being (Turner and Lloyd 1999; Turner and Butler 2003; Lantz et al. 2005).

As alluded to above, across racial/ethnic and gender groups SLE were found to promote depressive symptomology. These effects were found be fairly consistent across ages and to show strong evidence of gender disparity. Thus, SLE, though occurring less frequently among females, were shown to have more deleterious effects on this gender compared to males. It seems females are more prone to react to the effects of poverty and

stress with depression than males. This finding is consistent with research indicating females to be more apt to internalize adversity than their male counterparts (e.g. Compas and Wagner 1991; Dornbush et al. 1991).

Although these analyses offer some of the first comprehensive trajectory models of depressive symptomology in early life for both genders and all primary American racial/ethnic groups, the study is nevertheless limited in several respects. First, additional waves of data would allow further refinement and extension of these findings. The present investigation analyzed five age intervals based on the three waves of data currently available from the Add Health study. Further understanding of process of depression would be facilitated through additional waves of data extending the age interval further into adulthood. Fortunately, such analyses will soon be possible using the data used here, as the fourth wave of data collection is now underway for Add Health (http://www.cpc.unc.edu/projects/addhealth/design_focus/wave4). When released this data will allow the extension of the models presented here into the participants late 20's and early 30's.

Another shortcoming of the current study was our partial conceptualization of stress. Here we limited our modeling of the stress process to only SLE. However, 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; Wheaton 1994). Future research could improve the model presented here through a more exhaustive modeling of the stress process including chronic stressors and buffering psychological resources as other predictors of depression and mediators of the of the SES→depression effect. Another

potential improvement in the measurement of stress could be achieved through disaggregating the SLE index into various domains (e.g. Ge et al. 2006).

Despite these limitations, the present study improves our understanding of racial/ethnic and gender differences in normative trajectories of depressive symptoms and in the effects of childhood SES and SLE, over the ages 11-27. The results emerging here indicate that, consistent with research by Ge et al. (1994, 2006) and Hankin et al. (1998), trajectories of depressive symptoms during adolescence and young adulthood follow a normative curvilinear pattern with females disadvantaged at all ages. Consistent with research by Dornbush et al. (1991) and Ge et al. (1994) and, females are also found to react more depressively to adversity in the forms of both low childhood SES and SLE, though these factors are influential among males as well. Empirical support for the mediating role of SLE on the childhood SES→depressive symptomology was also found (Turner and Lloyd 1999; Turner and Butler 2003; Lantz et al. 2005). A distinguishing advantage of this study was its ability to demonstrate such a mediation using longitudinal data covering a wide age range.

By using a large, nationally representative dataset with minority over-sample, this study allowed the identification of racial/ethnic differences in depressive symptomology trajectories. Thus, the current findings shed light on an important debate in the field of mental health by showing a clear minority disadvantage in depressive symptomology in early adulthood. The large statistical power of the analyses offered a unique contribution through allowing the investigation of racial/ethnic groups, such as Asians and Hispanics, which many longitudinal studies are under-powered to investigate. In sum the findings of this study indicate that, unfortunately, the primary dimensions of inequality in America—

race/ethnicity, socio-economic status and gender, continue to be important determinants of psychological well-being.

Tables

Table 1. Frequency Distribution of Age, by Wave
(Counts and Percentages)

	Wave I (1995)		Wave II (1996)		Wave III (2001-2002)	
Age	Freq	Pct	Freq	Pct	Freq	Pct
11	10	0.05				
12	482	2.57	9	0.07		
13	2122	11.33	577	4.3		
14	2602	13.89	1800	13.41		
15	3357	17.92	2189	16.31		
16	3656	19.52	2797	20.84		
17	3461	18.48	2921	21.76		
18	2572	13.73	2172	16.18	121	0.87
19	402	2.15	796	5.93	1331	9.59
20	55	0.29	141	1.05	1888	13.61
21	10	0.05	19	0.14	2297	16.56
22					2586	18.64
23					2630	18.96
24					2185	15.75
25					713	5.14
26					102	0.74
27					17	0.12
Total	18729	100	13421	100	13872	100

Note: The respondents of Add Health in Wave I contain a wide range of age, with majority of them in Middle school or High school.

Table 2: Descriptive Statistics of Analysis Variables by Race/Ethnicity and Gender Group

	All		White		Black		Asian		Hispanic	
Male	N=9251		N=5341(28.5%)		N=2132(11.4%)		N=438(2.3%)		N=1340(7.1%)	
	Mean	*SD*	Mean	*SD*	Mean	*SD*	Mean	*SD*	Mean	*SD*
Father's education	5.50	2.32	5.77	2.11	5.59	2.29	6.32	2.28	3.97	2.58
Mother's education	5.62	2.40	5.90	2.26	5.44	2.30	6.37	2.29	4.19	2.68
Household Income	46.60	52.40	52.21	54.56	34.15	38.46	57.85	42.90	37.61	58.69
CES-D 11-15	4.77	3.35	4.41	3.22	5.21	3.44	5.25	3.71	5.46	3.42
CES-D 16-17	5.51	3.78	5.15	3.74	5.76	3.81	6.38	3.52	6.22	3.80
CES-D 18	5.50	3.90	5.14	3.85	5.77	3.85	6.63	3.59	6.12	4.09
CES-D 19-21	4.54	3.84	4.17	3.65	5.15	4.14	4.53	2.90	5.21	4.22
CES-D 22-27	4.19	3.80	3.83	3.59	4.64	3.98	5.11	3.72	4.56	4.17
SLE 11-15	2.55	2.73	2.37	2.60	2.81	2.67	1.75	2.69	3.18	3.25
SLE 16-17	3.02	3.16	2.60	2.83	3.58	3.33	2.57	2.85	4.02	3.81
SLE 18	2.85	3.09	2.48	2.76	3.50	3.39	2.26	2.70	3.60	3.62
SLE 19-21	1.95	2.29	1.79	2.16	2.34	2.51	1.23	1.69	2.26	2.55
SLE 22-27	1.85	1.95	1.65	1.80	2.35	2.13	1.63	2.15	1.95	1.98
Female	N=9507		N=5448(29.0%)		N=2388(12.7%)		N=351(1.9%)		N=1320(7.0%)	
	Mean	*SD*	Mean	*SD*	Mean	*SD*	Mean	*SD*	Mean	*SD*
Father's education	5.47	2.33	5.74	2.17	5.44	2.37	6.45	2.26	4.12	2.47
Mother's education	5.57	2.42	5.85	2.30	5.33	2.35	6.88	1.92	4.19	2.62
Household Income	46.77	51.49	53.12	55.49	33.60	34.06	67.77	92.42	36.42	36.65
CES-D 11-15	6.22	4.30	5.83	4.17	6.44	4.25	6.22	4.26	7.60	4.66
CES-D 16-17	6.84	4.45	6.53	4.42	7.05	4.43	7.85	4.66	7.41	4.46
CES-D 18	6.62	4.57	6.16	4.50	6.86	4.37	7.45	5.01	7.84	4.75
CES-D 19-21	5.33	4.48	4.95	4.35	5.64	4.51	6.41	4.85	6.19	4.76
CES-D 22-27	4.97	4.34	4.64	4.19	5.60	4.68	5.04	4.42	5.23	4.17
SLE 11-15	1.89	2.26	1.67	2.07	2.17	2.24	1.28	1.89	2.50	2.98
SLE 16-17	2.10	2.25	1.93	2.05	2.39	2.43	1.87	2.46	2.31	2.55
SLE 18	1.92	2.06	1.71	1.88	2.38	2.38	1.63	1.86	2.15	2.15
SLE 19-21	1.54	1.66	1.43	1.60	1.81	1.80	1.30	1.66	1.59	1.57
SLE 22-27	1.52	1.55	1.39	1.48	1.91	1.68	1.28	1.25	1.47	1.58

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

Table 3. Bivariate Correlations of Analysis Variables

	CES-D 11-15	CES-D 16-17	CES-D 18	CES-D 19-21	CES-D 22-27	SLE 11-15	SLE 16-17	SLE 18	SLE 19-21	SLE 22-27	Father's Education	Mother's Education	Household Income
CES-D 11-15	1												
CES-D 16-17	0.55	1											
CES-D 18	0.39	0.55	1										
CES-D 19-21	0.37	0.36	0.59	1									
CES-D 22-27	0.35	0.34	0.37	0.36	1								
SLE 11-15	0.31	0.25	0.19	0.17	0.21	1							
SLE 16-17	0.25	0.26	0.20	0.17	0.15	0.71	1						
SLE 18	0.25	0.22	0.25	0.26	0.13	0.45	0.68	1					
SLE 19-21	0.17	0.16	0.21	0.26	0.08	0.40	0.41	0.66	1				
SLE 22-27	0.18	0.14	0.15	0.18	0.22	0.42	0.39	0.41	0.37	1			
Father's Education	-0.14	-0.12	-0.14	-0.12	-0.10	-0.11	-0.12	-0.11	-0.08	-0.07	1		
Mother's Education	-0.17	-0.12	-0.12	-0.10	-0.10	-0.15	-0.13	-0.10	-0.11	-0.09	0.56	1	
Household Income	-0.10	-0.07	-0.07	-0.08	-0.05	-0.11	-0.10	-0.08	-0.10	-0.08	0.28	0.33	1

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

Table 4. Latent Growth Curve Models of CES-D predicted by race, gender, SES and SLE (N= 18,764)

	Model 1		Model 2		Model 3		Model 4		Model 5	
Parameter	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE
Means										
Intercept (α)	5.86**	0.04	4.70**	0.07	4.83**	0.07	4.69**	0.07	4.71**	0.07
Linear slope (β)	0.05**	0.01	0.11**	0.03	0.10**	0.03	0.13**	0.03	0.12**	0.03
Quadratic slope (β^2)	-0.01**	0.00	-0.02**	0.00	-0.02**	0.00	-0.02**	0.00	-0.02**	0.00
Variances										
α	7.37**	0.29	6.57**	0.28	5.99**	0.28	4.57**	0.27	4.59**	0.27
β	0.15**	0.05	0.16**	0.05	0.15**	0.05	0.10*	0.05	0.10*	0.05
β^2	0.00**	0.00	0.00**	0.00	0.00**	0.00	0.00**	0.00	0.00**	0.00
Time invariant predictors										
White female → α			1.46**	0.10	1.45**	0.10	1.77**	0.10	1.75**	0.10
Black male → α			0.73**	0.14	0.54**	0.14	0.34**	0.13	0.36**	0.13
Black female → α			2.08**	0.13	1.82**	0.13	1.98**	0.12	1.96**	0.12
Asian male → α			1.11**	0.28	1.40**	0.28	1.51**	0.26	1.50**	0.27
Asian female → α			2.05**	0.31	2.39**	0.31	2.74**	0.30	2.71**	0.30
Hispanic male→α			1.05**	0.17	0.27	0.18	0.01	0.17	0.02	0.17
Hispanic female →α			3.15**	0.16	2.40**	0.17	2.54**	0.16	2.51**	0.16
White female→β			-0.09**	0.04	-0.09**	0.04	-0.11**	0.04	-0.11**	0.04
Black male→β			-0.03	0.05	-0.02	0.05	-0.06	0.05	-0.05	0.05
Black female→β			-0.13**	0.05	-0.11*	0.05	-0.13**	0.05	-0.12**	0.05
Asian male→β			0.00	0.10	-0.02	0.10	-0.03	0.09	-0.03	0.09
Asian female→β			0.25*	0.11	0.23*	0.11	0.20	0.11	0.20	0.11
Hispanic male→β			0.03	0.06	0.08	0.06	0.04	0.06	0.04	0.06
Hispanic female→β			-0.18**	0.06	-0.14*	0.06	-0.12*	0.06	-0.12*	0.06
White female→β^2			0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Black male→β^2			0.00	0.00	0.00	0.00	0.01	0.00	0.00	0.00
Black female→β^2			0.01*	0.00	0.01	0.00	0.01	0.00	0.01	0.00
Asian male→β^2			0.00	0.01	0.00	0.01	0.00	0.01	0.00	0.01
Asian female→β^2			-0.03**	0.01	-0.03**	0.01	-0.03**	0.01	-0.03**	0.01
Hispanic male→β^2			-0.01	0.01	-0.01	0.01	0.00	0.01	-0.00	0.01
Hispanic female→β^2			0.00	0.00	0.00	0.01	0.00	0.01	0.00	0.01
SES→α					-0.50**	0.03	-0.40**	0.03	-0.41**	0.03
SES→β					0.03**	0.01	0.02	0.01	0.02	0.01
SES→β^2					0.00	0.00	0.00	0.00	0.00	0.00
Time variant predictors										
SLE→CES-D (across ages)									0.96**	0.02
SLE→CES-D ages 11-15							1.00**	0.04		
SLE→CES-D ages 16-17							1.01**	0.04		
SLE→CES-D age 18							1.02**	0.06		
SLE→CES-D ages 19-21							0.96**	0.05		
SLE→CES-D ages 22-27							0.84**	0.04		
R^2										
CES-D ages 11-15	0.46		0.47		0.47		0.46		0.45	
CES-D ages 16-17	0.52		0.52		0.52		0.51		0.51	
CES-D ages 18	0.53		0.53		0.53		0.52		0.52	
CES-D ages 19-21	0.52		0.52		0.52		0.52		0.52	
CES-D ages 22-27	0.49		0.49		0.49		0.49		0.50	
α			0.11		0.20		0.23		0.23	
β			0.03		0.05		0.05		0.05	
β^2			0.02		0.02		0.03		0.03	

Note: SES = childhood socioeconomic status; SLE = stressful life events index; CES-D = Center for Epidemiologic Studies Depression Scale, 9 item derivative.
*p < .05. **p < .01.

Table 5. Fit Indices and Nested Model Likelihood Ratio Tests (N = 18,764)

Model Description	*df*	χ^2	CFI	RMSEA	Adj. BIC	$\Delta\chi^2$	Δdf
Model 1: Unconditional CES-D growth curve	111	4290.30	.72	0.05	575814.90	-	-
Model 2: Model 1 with race/gender dummy variables predicting growth factors	90	3380.45	.78	0.04	575044.95	909.85	21
Model 3: Model 2 with SES latent variable predicting growth factors	87	2824.04	.82	0.04	574508.53	556.41	3
Model 4: Model 3 with SLE index predicting CES-D repeated measure	82	980.13	.94	0.02	572697.92	1843.91	5
Model 5: Model 4 with SLE index effects constrained equal across ages	86	996.60	.94	0.02	572697.74	-16.47	-4

Note: CFI = comparative fit index; RMSEA = root mean square error of approximation; Adj. BIC = sample-size adjusted Bayesian Information Criterion; SLE = stressful life events index; CES-D = Center for Epidemiologic Studies Depression Scale, 9 item derivative.

** p <.01.

Table 6. Latent Growth Curve Models of CES-D predicted by SES and SLE, stratified by Race/Ethnicity (Males)

	All Male (N=9253)		White Male (N=5342)		Black Male (N=2133)		Asian Male (N=438)		Hispanic Male (N=1340)	
Parameter	Est	SE	Est	SE	Est	SE	Est	SE	Est	SE
Means										
Intercept	5.01**	0.05	4.65**	0.06	5.35**	0.10	5.86**	0.25	5.70**	0.14
Linear Slope	0.12**	0.02	0.12**	0.02	0.10**	0.04	0.09	0.09	0.15**	0.05
Quadratic Slope	-0.02**	0.00	-0.02**	0.00	-0.02**	0.00	-0.02*	0.01	-0.02**	0.00
Variances										
Intercept	3.44**	0.31	3.38**	0.38	2.04**	0.69	4.24*	1.66	3.79**	0.83
Linear Slope	0.05	0.06	0.07	0.07	-0.10	0.13	0.06	0.26	0.26	0.16
Quadratic Slope	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00*	0.00
Time invariant predictors										
SES→Intercept	-0.32**	0.04	-0.27**	0.06	-0.47**	0.10	-0.50	0.25	-0.35*	0.09
SES→Linear Slope	0.01	0.02	0.00	0.02	0.02	0.04	0.09	0.09	0.02	0.03
SES→Quadratic Slope	0.00	0.00	0.00	0.00	-0.00	0.00	-0.01	0.01	0.00	0.00
Time variant predictor										
SLE→CES-D (all ages)	0.77**	0.03	0.69**	0.04	0.89**	0.06	0.77*	0.13	0.95*	0.08
R^2										
CES-D ages 11-15	0.39		0.39		0.31		0.43		0.43	
CES-D ages 16-17	0.52		0.53		0.45		0.44		0.54	
CES-D ages 18	0.53		0.54		0.47		0.42		0.57	
CES-D ages 19-21	0.53		0.54		0.48		0.39		0.57	
CES-D ages 22-27	0.53		0.49		0.49		0.50		0.62	
Intercept	0.07		0.04		0.20		0.09		0.13	
Linear Slope	0.00		0.00		0.00		0.01		0.00	
Quadratic Slope	0.00		0.00		0.00		0.01		0.00	
Fit Indices										
χ^2 *df* = 58	276.01		188.41		115.02		88.80		93.42	
CFI	.96		.96		.95		.95		.95	
RMSEA	.02		.02		.02		.03		.02	

Note: SES = childhood socioeconomic status; SLE = stressful life events index; CES-D = Center for Epidemiologic Studies Depression Scale, 9 item derivative; CFI = comparative fit index; RMSEA = root mean square error of approximation.
*p < .05. **p < .01.

Table 7. Latent Growth Curve Models of CES-D predicted by SES and SLE, stratified by Race/Ethnicity (Females)

	All Female (N=9511)		White Female (N=5449)		Black Female (N=2390)		Asian Female (N=351)		Hispanic Female (N=1321)	
Parameter	Est	SE	Est	SE	Est	SE	Est	SE	Est	SE
Means										
Intercept	6.52**	0.06	6.12**	0.07	6.71**	0.11	6.66**	0.32	7.83**	0.16
Linear Slope	0.02	0.02	0.02	0.03	-0.01	0.04	0.38**	0.12	-0.08	0.06
Quadratic Slope	-0.01**	0.00	-0.01**	0.00	-0.01**	0.00	-0.04**	0.01	-0.01**	0.00
Variances										
Intercept	5.69**	0.44	5.09**	0.55	5.67**	0.87	7.12**	2.37	5.68**	1.31
Linear Slope	0.11	0.08	0.06	0.10	0.17	0.15	0.67	0.40	0.02	0.22
Quadratic Slope	0.00	0.00	0.00	0.00	0.00	0.00	0.01*	0.00	0.00	0.00
Time invariant predictors										
SES→Intercept	-0.50**	0.05	-0.53**	0.07	-0.43**	0.10	0.08	0.23	-0.59**	0.16
SES→Linear Slope	0.03	0.02	0.06*	0.02	-0.02	0.03	-0.09	0.09	0.01	0.06
SES→Quadratic Slope	0.00	0.00	0.00	0.00	0.00	0.00	0.01	0.01	0.00	0.00
Time variant predictor										
SLE→CES-D (all ages)	1.17**	0.03	1.21**	0.04	1.06**	0.07	1.17**	0.18	1.29**	0.09
R^2										
CES-D ages 11-15	0.43		0.44		0.42		0.49		0.42	
CES-D ages 16-17	0.48		0.49		0.46		0.58		0.47	
CES-D ages 18	0.49		0.49		0.47		0.60		0.47	
CES-D ages 19-21	0.49		0.49		0.47		0.59		0.46	
CES-D ages 22-27	0.47		0.44		0.53		0.56		0.38	
Intercept	0.09		0.10		0.08		0.00		0.10	
Linear Slope	0.02		0.02		0.01		0.04		0.01	
Quadratic Slope	0.00		0.01		0.01		0.04		0.00	
Fit Indices										
χ^2 *df* = 58	491.10		297.13		187.59		81.12		155.30	
CFI	.94		.94		.91		.90		.90	
RMSEA	.03		.03		.03		.03		.04	

Note: SES = childhood socioeconomic status; SLE = stressful life events index; CES-D = Center for Epidemiologic Studies Depression Scale, 9 item derivative; CFI = comparative fit index; RMSEA = root mean square error of approximation.
*p < .05. **p < .01.

Figures

Figure 1. Predicted surface of the effect of SES on Depressive symptomology trajectory

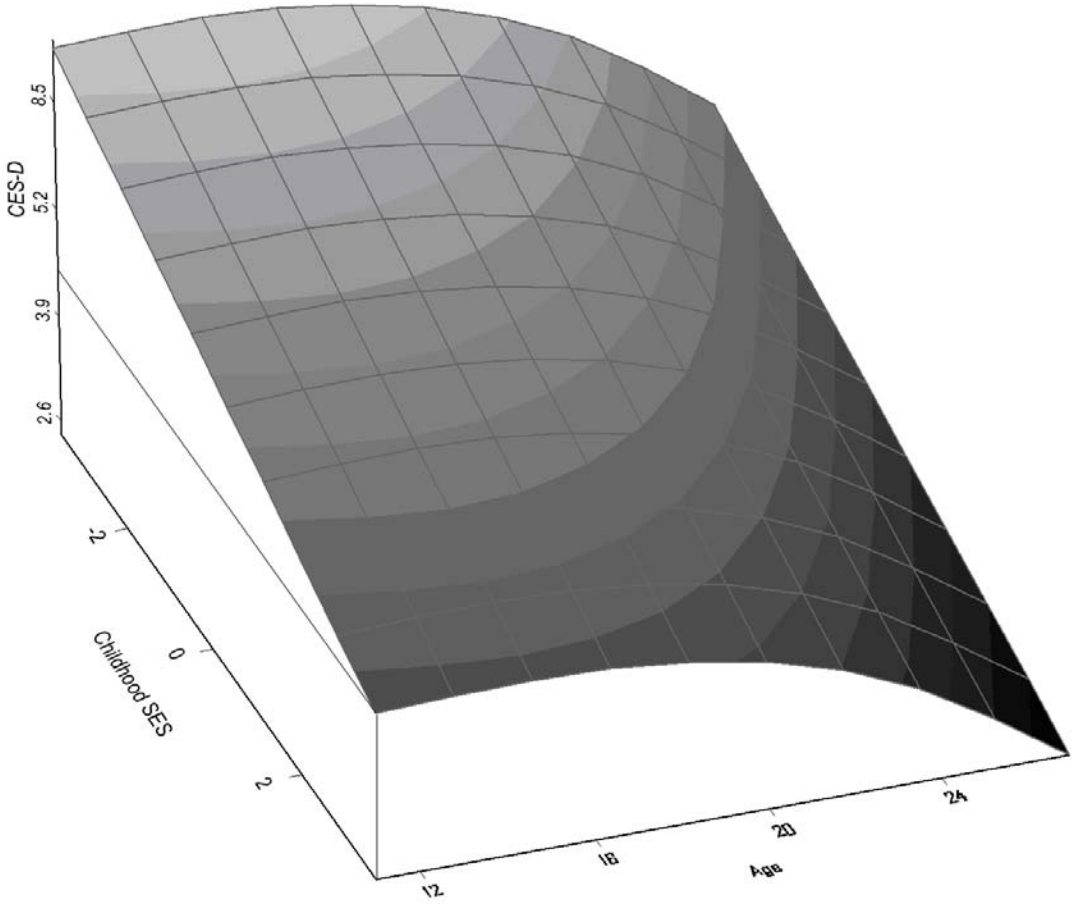


Figure 2. Path diagram of conditional depressive symptomology LCM predicted by time invariant SES and time variant SLE

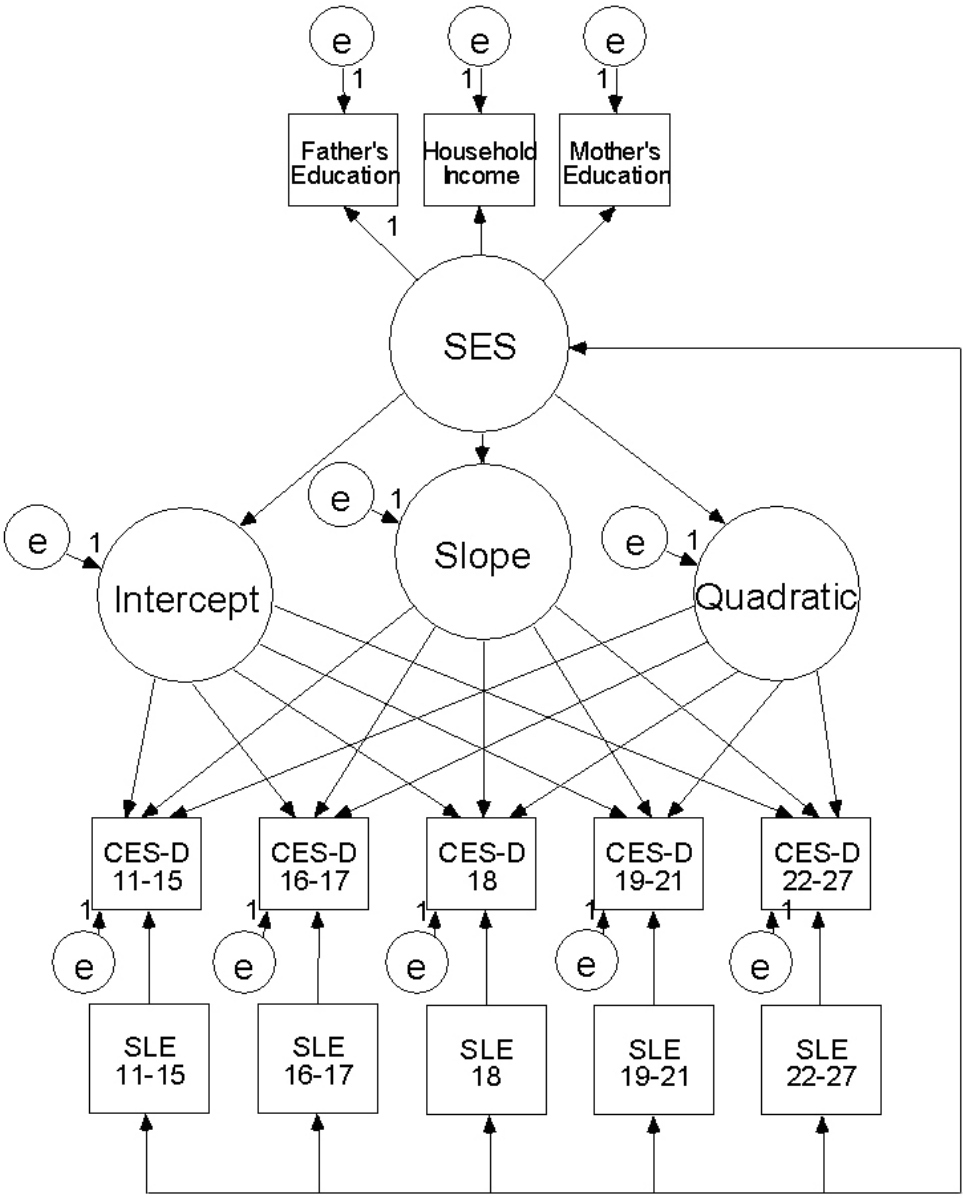


Figure 3. Predicted Depressive Symptomology Trajectories by Race (Males)

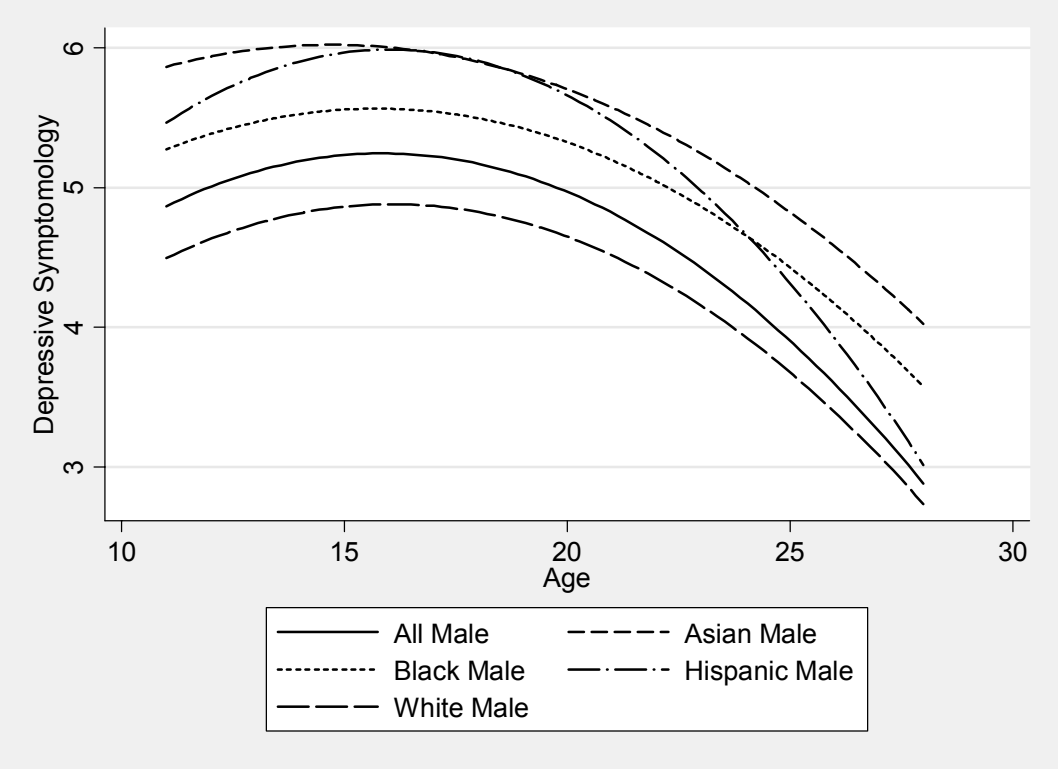


Figure 4. Effect of SLE on Depressive symptomology by Race and Gender

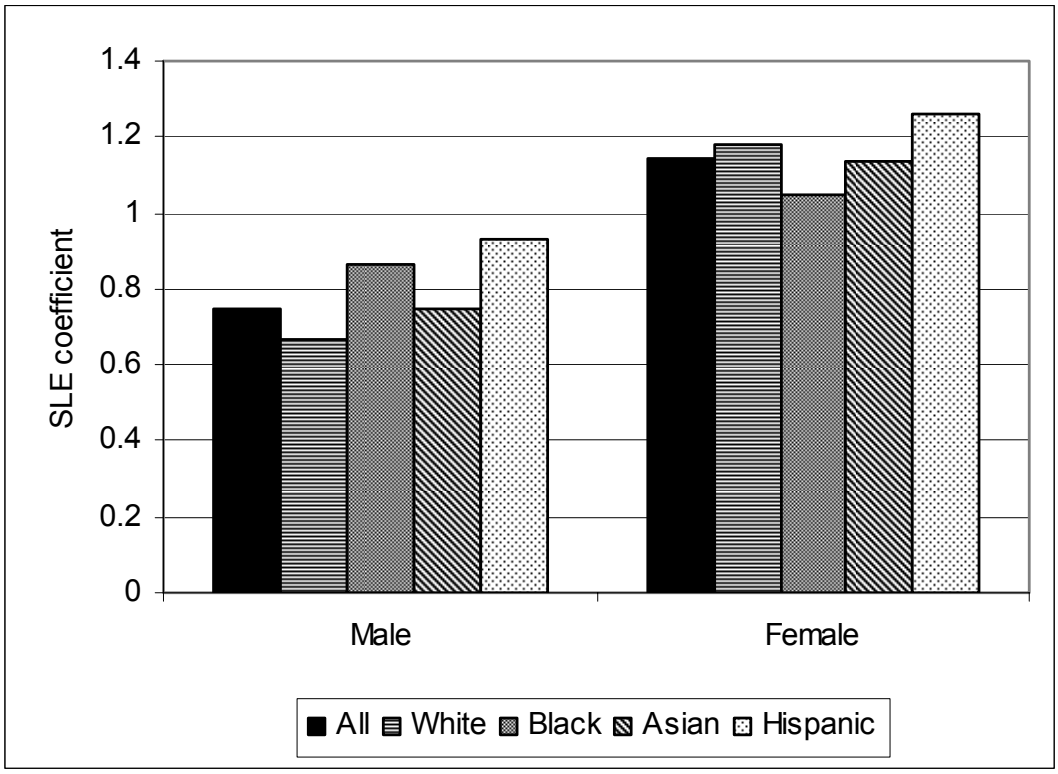
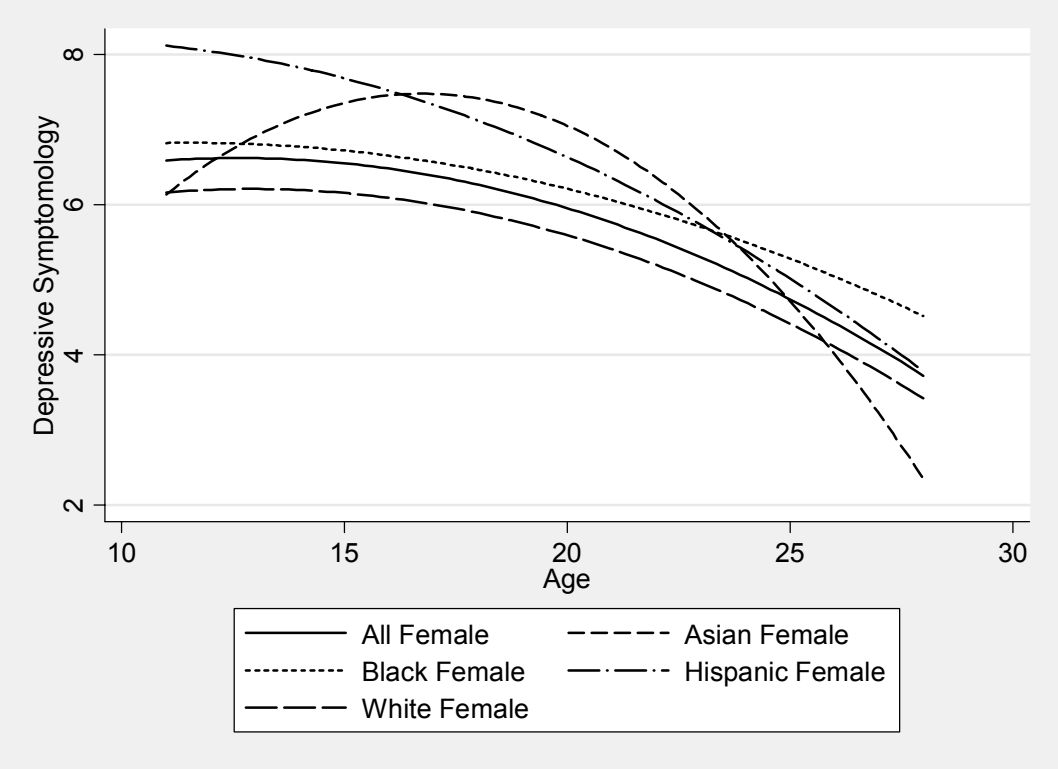


Figure 5. Predicted Depressive Symptomology Trajectories by Race (Females)



Appendices

Appendix 1. Comparison of predictive power and model fit of various SES specifications as time invariant predictors of depressive symptomology LCM

	SES CFA	Father's Education	Mother's Education	Household Income	All SES indicators separately
R^2					
Intercept	0.11	0.05	0.07	0.02	0.08
Linear Slope	0.02	0.00	0.03	0.00	0.03
Quadratic Slope	0.01	0.00	0.01	0.00	0.01
Fit Indices					
CFI	0.97	0.94	0.94	0.93	0.94
RMSEA	0.02	0.04	0.04	0.04	0.03

Note: SES = childhood socioeconomic status; CFA = confirmatory factor analysis; CFI = comparative fit index; RMSEA = root mean square error of approximation.

Appendix 2, List of Stressful Life Event Items in Each Wave

Items Available in All Three Waves
Parent death
Self attempted suicide resulting in injury (outcome)
Friend attempted suicide (unsuccessful)
Friend attempted suicide (with success)
Relative attempted suicide (unsuccessful)
Relative attempted suicide (with success)
Involving in fighting or violence
Unwanted pregnancy
Abortion, still birth, or miscarriage
Having a child adopted
Death of a child
Romantic relationship ended
Giving sex in exchange for drugs or money
STD
Skip Medicare
Juvenile conviction
Adult conviction
In jail (during the past year, assuming jail time starts right after conviction)
Wave I and II
Having a serious injury
Expelled from school
Run away from home
Parents receive welfare
Nonromantic sexual relationship ended (w3 doesn't distinguish the romantic and nonromantic sexual relationships)
Rape (gender difference, and no time available)
Abuse in romantic or nonromantic sexual relationship
Wave III
Receiving welfare
Baby having major health problems at birth
Marriage dissolution
Cohabitation dissolution
Death of a romantic partner
Eviction, cutoff service
Entering full time active military duty
Discharged from the armed forces

Note: All items are coded as 1 year before the interview

Appendix 3. CFA Measurement Model of Childhood SES (N=16,110)

Parameter	Estimate	SE
SES Factor		
Mean	-0.05**	0.02
Variance	2.64**	0.10
Factor loadings		
SES→Father's educational attainment	1.00	-
SES→Mother's educational attainment	1.18**	0.04
SES→Yearly household income	12.57**	0.39
R^2		
Father's educational attainment		0.15
Mother's educational attainment		0.63
Yearly household income		0.49
Fit Indices		
χ^2 (df)		19.81 (2)
CFI		1.00
RMSEA		0.02

Note: Factor loading of SES by father's educational attainment was constrained equal to 1 as a standard identifying assumption. SES = childhood socioeconomic status; CFI = comparative fit index; RMSEA = root mean square error of approximation.
*p < .05. **p < .01