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Socioeconomic Pathways to Depressive Symptoms in Adulthood: Evidence from the National Longitudinal Survey of Youth 1979

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Abstract

The existence of a direct effect of early socioeconomic position (SEP) on adult mental health outcomes net of adult SEP is still debated. This question demands the explicit modeling of pathways linking early SEP to adult SEP and mental health. In light of this background, we pursue two objectives in this study. First, we examine whether depressive symptoms in adulthood can be fit in a trajectory featuring both an intercept, or baseline range of depressive symptoms that varied between individuals, and a slope describing the average evolution of depressive symptoms over the years. Second, we estimate the direct and indirect pathways linking early SEP, respondents' education and adult household income, with a particular focus on whether early SEP retains a significant direct effect on the trajectory of depressive symptoms once adult SEP is entered into the pathway model. Drawing from 29 years of cohort data from the National Longitudinal Survey of Youth 1979, a survey that has been following a national probability sample of American civilian and military youth (Zagorsky and White, 1999), we used structural equation models to estimate the pathways between parents' education, respondent's education, and latent growth curves of household income and depressive symptoms. We found that the effect of parents' education was entirely mediated by respondent's education. In turn, the effect of respondent's education was largely mediated by household income. In conclusion, our findings showed that the socioeconomic attainment process that is rooted in parents' education and leads to respondent's education and then to household income is a crucial pathway for adult mental health. These results suggest that increasing educational opportunities may be an effective policy to break the intergenerational transmission of low socioeconomic status and poor mental health.

Keywords

depressive symptoms; USA; mental health; life course; structural equation model; latent growth curve; trajectories; socioeconomic position

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In this paper we will examine the socioeconomic pathways leading from parents' education to depressive symptoms in adulthood. Using 29 years of annual or biennial cohort data from the National Longitudinal Survey of Youth 1979, we model adults' depressive trajectories as the result of pathway effects taking root in early socioeconomic position (SEP), which in turn affect adult SEP. Thus, through structural equation modeling, we estimate the direct and indirect effects of parents' education, respondents' own education and household income on adult trajectories of adult depressive symptoms.

In this introductory section, we briefly review causal models in life course epidemiology to contextualize the importance, but dearth of studies on pathway models. Second, we present research on the relationship between age and depressive symptoms to propose a model of the natural (and social) history of depressive symptoms over the life course. Third, we review evidence in life course epidemiology pertaining to the interplay of early and adult SEP in the etiology of major depressive disorders. Finally, we draw from these lines of research to develop the hypotheses tested in this paper.

Causal models in life course epidemiology: The elusive pathway effects

The maturation of birth cohort studies has seen the concomitant development of a bustling area of research in epidemiology concerned with the elucidation of life course processes of disease etiology (Ben Shlomo and Kuh, 2002). A number of life course models have been proposed, such as the critical/sensitive periods model, the accumulation model, and the pathway (or chains of risk) model (Hertzman, 1999; Kuh et al., 2003). Whereas the critical/sensitive period and accumulation models have been substantiated by a wealth of studies, empirical evidence for the pathway effects remains elusive (Lahelma et al., 2004), particularly with regards to depression (Gilman et al., 2002).

Pathway effects occur when social conditions at time $t-1$ affect social conditions at time t , which increases the probability of certain trajectories of cumulative socioeconomic advantage or disadvantage in adulthood with distinct effects on adult health (e.g. Hayward and Gorman, 2004). The pathway model thus postulates that early life environments shape individual trajectories of socioeconomic attainment, which in turn have proximal effects on health. For instance, a typical pathway effect would occur when low SEP in childhood restricts educational opportunities, which in turn impairs entry into the labor force in early adulthood, with dire consequences for accumulated SEP and finally, health in adulthood (Graham, 2002).

In sum, pathway effects postulate probabilistic mechanisms whereby negative outcomes at one stage increase the likelihood of negative outcomes at the next stage, in a process of cumulative disadvantage (Evans, 2002; O'Rand, 1996). Testing of this causal model therefore requires prospective data with multiple time points of data collection, as well as statistical modeling capable of reflecting the time varying and interdependent nature of the exposures and outcomes. In addition, this approach requires that we have a prior understanding of the natural progression of the outcome over the life course. In the next section, we therefore present evidence concerning the trajectories of depressive symptoms over the life course.

Trajectories of depressive symptoms in adulthood

One contemporary development of research on depressive symptoms over the life course has been the study of the association between depressive symptoms and age, and more specifically, of the social factors that might explain this relationship.

Regarding the bivariate association between depressive symptoms and age, most North American studies find a decline in depressive symptoms with age from early to mid-adulthood (Newmann, 1989). For instance, longitudinal results indicate that women from the baby boomer generation experience declining levels of distress from age 35 to 50 (Kasen et al., 2003). However, the shape of the age-depressive symptoms relationship from middle age to older adulthood is more contentious, as some U.S. studies find an upturn of depressive symptoms in middle age, with the inflexion point reported to be anywhere from the mid-forties (Kasen et al., 2003; Kessler et al., 1992; Mirowsky and Ross, 1992) to the early sixties (Schieman et al., 2001), while others in Canada observe an uninterrupted decline (Wade and Cairney, 1997).

These debates about the exact shape of the relationship between age and depressive symptoms have substantive implications, as this research can in fact be seen as bringing together the rich lines of inquiry into the stress process and the life course perspective (Pearlin and Skaff, 1996; Schieman et al., 2001). Most studies on age and depressive symptoms indeed reach beyond the simple description of this relationship to explain it away with the prevalence of life events and roles (and related stressors or protective factors) at those ages.

For instance, Mirowsky and Ross (1992) explain their observation in the United States of a u-shaped relationship of depressive symptoms with age that bottoms out in the mid-forties by the fact that were it not for the gain and loss of partners, jobs, and wealth, there would be little or no fall and rise in the depressive symptoms in successive age groups (Mirowsky and Ross, 1992: 201). They, and others after them (Kessler et al., 1992; Mirowsky and Ross, 2001; Schieman et al. 2001; Wade and Cairney, 1997, 2000), thus attribute much of the decline and increase in depressive symptoms with age to these proximate social and economic statuses and related life events. This u-shaped relationship was replicated in other U.S. and Canadian studies, with further development of the life course timing of the determinants explaining it (Kessler et al., 1992; Mirowsky and Ross, 2001; Schieman et al., 2001; Wade and Cairney 1997, 2000).

However, many of these studies relied on synthetic cohort analysis based on cross-sectional data to establish these patterns, and thus it is not possible to rule out entirely that these observations stem from period or cohort effects rather than aging or life course effects (Muntaner et al., 2004). Furthermore, these studies focused on the adult years, and were therefore not able to assess full pathways of socioeconomic position starting in childhood. In the next section, we turn to studies that have used cohort data to examine the life course epidemiology of depressive disorders from childhood into adulthood.

The life course epidemiology of depressive disorders: The interplay of early life and adult SEP

While the association of SEP with adult major depression is well-established (Lorant et al., 2003), the origins of this association are still debated. More specifically, the extent to which early life SEP effects are partly or fully mediated by adult SEP effects in the etiology of adult depressive disorders is still debated to this day (Mckenzie et al., 2010). In a review of this literature, Muntaner and colleagues (2004) found only eight studies published between 1999 and 2003 that were concerned with the life course epidemiology of depression (Harper et al., 2002; Gilman et al., 2003; Kubzansky et al., 2000; Marmot et al., 2001; Miech et al., 1999; Power et al. 2002; Stansfeld et al., 2003). Most of these studies suggest that both early life and adult SEP are associated with adult depression and anxiety (Muntaner et al., 2004). However, none of these studies explicitly model the pathways that link early SEP to adult SEP, and instead assess the change in magnitude of adult SEP coefficients when early SEP

coefficients are introduced in the model (e.g. Power et al., 2002). In our update of this literature review, we noted that while many of these studies speak to the direct and indirect effects of early and adult SEP on adult mental health, none of the studies we found explicitly estimated these effects while taking into account the causal pathways that lead from early SEP to adult SEP (Benjet et al., 2010; Clark et al., 2007; Elovainio et al., 2007; Green & Benzeval, 2010; Huurre et al., 2005; Kristensen et al., 2010; McKenzie et al., 2010; Melchior et al., 2007; Mossakowski, 2008; Stansfeld et al., 2010; Vasiliadis et al., 2010; Wiggins, 2004).

This modeling strategy amounts to estimating only the direct pathways between a predictor and the outcome, net of the effects of other variables. Thus, the indirect pathways are not explicitly estimated, and the underlying covariance and potential error correlation structure existing between the variables is ignored. In situations where there is unmeasured confounding, measurement error and where conceptual constructs are complex, this can lead to biased statistical estimates and erroneous conclusions concerning the existence of mediation (Shaver, 2005). The existence of these potential biases is particularly germane to the situation at hand, since there obviously exists a complex web of causation from early SEP to adult SEP that is also fraught with measurement error and unobserved confounding (Gilman, 2007). This could explain the persistence of a debate around the existence (or not) of a direct effect of SEP on adult mental health that remains after adjustment for adult SEP.

Conceptual model

These lines of research point to the following fruitful directions for this investigation. First, research on depressive symptoms in adulthood suggests that depressive symptoms over the life course should be measured as a trajectory, and this with cohort data. Second, following the life course epidemiology literature, it appears that depressive symptoms in adulthood are affected by both early life and adult exposure to deleterious socioeconomic conditions, and that these relationships should be modeled as pathway effects. Finally, the potential bias in the estimation of mediating effects noted above suggests that we need to turn to a statistical method that allows to explicitly model the error terms in the variables in the model, as well as the potential covariance existing between them.

In light of these observations, we will draw from cohort data from the National Longitudinal Survey of Youth 1979 and use structural equation modeling to first examine whether depressive symptoms in adulthood can be fit in a trajectory featuring both an intercept, or baseline range of depressive symptoms that varied between individuals, and a slope describing the average evolution of depressive symptoms over the years; and second, to test the direct and indirect pathways linking early SEP, respondents' education and adult household income, with a particular focus on whether early SEP will retain a significant direct effect on the trajectory of depressive symptoms once adult SEP is entered into the pathway.

Research Design

Study Population

Data are drawn from the publicly available files of the 1979 National Longitudinal Survey of Youth (NLSY79), an ongoing longitudinal panel survey that has been following since 1979 a national probability sample of American civilian and military youth aged 14 to 21 years old in 1978 (Zagorsky and White, 1999).

The NLSY79, sponsored by the Bureau of Labor Statistics, was originally designed to gather longitudinal information on the socioeconomic status and labor force experiences of young

American men and women. As such, the NLSY79 is particularly well-suited for the study of stratification outcomes, as it includes data about social origins and traces a comprehensive, prospective and continuous work history of its respondents from their teens/early 20s to well into their forties. Furthermore, with the recent addition of supplementary health modules, the NLSY79 has now become an important source for the study of social inequalities in health over the life course.

Not surprisingly, with such a long period of follow-up, the NLSY79 sample has suffered some attrition over the years. In an analysis of attrition in the NLSY79, MaCurdy and colleagues (1998) found that, despite being nonrandom, attrition did not introduce biases in the estimation of earnings and other labor-market variables. However, no comparable studies have been conducted as of yet on the impact of attrition on health in the NLSY79. Previous studies on health-related attrition (not specific to the NLSY79) have found that attriters were more likely to be in poor health (see the five-paper series in the *Journal of Clinical Epidemiology*, 2002, volumes 55 and 56, introduced by Deeg, 2002, as well as Norris, 1987), which, if it were the case in the NLSY79 as well, would tend to introduce a conservative bias to our analyses by limiting health variation to the healthier range of the distribution. More analyses are obviously needed to assess the impact of attrition on health in the NLSY79. However, given that there were no attrition biases in labor force outcomes, and based on prior studies of mental health-related attrition bias in panel studies (Osler et al., 2008), it is unlikely that this nonrandom attrition will introduce discernable bias in the estimation of the model (Deeg, 2002; Norris, 1987).

Data and Measures

Because of the staggered nature of the measurement of CES-D (see below), we reshaped the data to anchor measurements on respondents' ages rather than the survey year. Coupled with the Full Information Maximum Likelihood (FIML) estimator described at the end of this section, this strategy allowed us to maximize both sample size and period of observation, while also providing more intuitive anchor points from a life course perspective for the trajectories studied here. Finally, because of the sparseness of data at certain ages, we used age ranges to capture the different points of the trajectories of depressive symptoms and household income, as described in greater detail below. The models presented here are robust to various specifications on these age ranges, as well as to an analysis based on survey year (results not shown), and all models control for year of birth (although the inclusion of this variable to the model did not substantively affect the results).

Depressive symptoms—Depressive symptoms are measured with the Center for Epidemiologic Studies Depression scale (CES-D; Radloff, 1977). In the NLSY79, this variable was measured in 1992, 1994, either in 1998, 2000, 2002, 2004 or 2006 (whenever the respondents reached 40 years of age) and in 2008 (only for those respondents having reached 50 years of age). With the data reshaped with age as an anchor, we then took the individual mean of CES-D when respondents were between 1. 28 and 34 years old, 2. 35 and 39 years old and 3. 40–51 years old. In alternative models not shown here we used the maximum CES-D reported during these periods with similar substantive findings. We elected to keep the model with mean values to avoid capturing mental health shocks due to non-recurrent events, which the maximum CES-D had the potential to do.

In addition, while the 1992 wave uses the full CES-D scale of 20 items, later waves use a 7 items subscale validated by Mirowsky and Ross (1989). Thus, for comparability, we will use only the 7-item scales that are common to all those waves of data collection in these analyses.

These seven questions ask the respondents how often during the past week they had: had poor appetite, trouble keeping mind on tasks, been depressed, thought everything took extra effort, suffered from restless sleep, been sad, and been unable to get going. Responses on a 4-point likert scale ranged from rarely/none of the time/1 day to Most/All of the time/5–7 days. These items were summed up to create scales ranging from 0 to 21, and because they were all negatively worded items, higher values indicate more depressive symptoms.

Cronbach alphas on those summed indices ranged between 0.77 and 0.84, which is slightly lower than previously reported for the full scale (Radloff, 1977) but nevertheless satisfactory given that only seven items were available. These tests indicate that the internal consistency of the scales is acceptable, and suggest that the indices are measuring a single unidimensional (primarily somatic) latent construct.

Early socioeconomic position—*Mother's and Father's highest grade completed* measure parental socioeconomic status. These measures range from 0 to 20 years of education. The NLSY79 has a number of siblings from the same family in the sample. Thus, when the information was missing on these variables for one sibling, values were imputed from other siblings (and averaged if there were more than one sibling). When values were missing for all siblings or if there were no siblings available (in the general sample), the sample's gender-specific mean value of education for the relevant parent was imputed. We also include a dummy variable to capture the effect of having a missing value for each parent's education.

Socioeconomic status in adulthood—Respondents' years of completed *Education* were measured as the highest education reported between 24 and 26 years old (in sensitivity analyses, we also estimated this model with education at age 25, without any substantive modifications to the results), and range from 0 to 20 years. This measure of educational attainment very closely approximates the highest degree attained during the study period, as almost three-fourths (72%) of the respondents had attained their final educational status at that point.

Annual household income, in \$10,000 increments was measured from 28 years old to up to 50 years old (depending on the age of the respondent at the last survey). A household-level variable was preferred over individual income in these analyses because this is a prime childbearing period for the women in this sample, and using a household measures circumvents the fact that they may temporarily leave the labor force due to pregnancy.

These variables were transformed first to constant 2000 dollars, and secondly by taking the natural log (plus a small constant) to reflect the non-linear functional relationship between income and health. To avoid reverse causality, household income was lagged by one calendar year relative to the measurement of the CES-D. Finally, as with the CES-D, we used aggregated measurements over three age groupings: 1. 28–34 years old; 2. 35–39 years old; 3. 40–51 years old, but in this case, we relied on the median income over these periods as is common in stratification research to avoid undue influence by outlier income years (both high and low).

Control variables—We originally estimated each model separately by gender (models not shown). However, the findings were substantively similar and so for parsimony, we present models including both men and women, controlling on gender (Male=1). We also control in the models for race (Non-Hispanic White=0/Black=1/Hispanic=1). In addition, we control for history of parents' depression (reported on the ICD-9 index by respondents when they were 40 years or over) and for respondent's early depression (reported as an ICD-9 coded

condition that would keep one from being able to work in the years 1979–1982, or when the respondents were in their late teens/early 20s).

Analyses

Analyses were conducted using latent growth curves to measure how changes in SEP over the life course affect changes in depressive symptoms over the life course (Duncan et al., 1999). Using structural equation modeling, latent growth curves allow the estimation of within-individual trajectories of change over the life course, as well as between-individual differences in the initial level of and rate of change in both depressive symptoms and SEP. Thus, we first chart the individual progression of depressive symptoms over time and the shape of this trajectory (Mirowsky and Ross, 1992). Second, we examine the progression of financial resources as both early and concurrent income trajectories stretching from young to mid-adulthood. Finally, we run a series of nested models on the depressive symptoms trajectory to assess the direct, indirect and total effects of early and adult SEP. To respect temporality, we will first assess the impact of sociodemographic control variables and early SEP while controlling for parents' and respondent's history of depressive symptoms on initial level and change in depressive symptoms. The second model introduces the respondent's education, and the third model introduces the household income trajectory. It should be noted that although we emphasize the social causation hypothesis here (following notably results from Power et al., 2002), we include the effects of respondent's early depression on achievement occurring thereafter (income trajectory) in order to account for selection bias of mental health on future status attainment.

Models

Unconditional Depressive Symptoms Trajectory: The first step in our analytic design was to establish that depressive symptoms could be measured as a two-factor latent growth process (or trajectory) for the adults in this sample. Indeed, actual measurement of individual-level trajectories in this field of study is still rare (George, 2003). For instance, depressive symptoms have been modeled successfully as linear trajectories among older adults (George and Lynch, 2003; Taylor and Lynch, 2004) and adolescents (Ge et al., 1994).

The general level 1 equation may be expressed as:

$$y_{it} = \alpha_{yi} + \beta_{yi}\lambda_{yt} + \varepsilon_{yit} \quad (1)$$

where y_{it} is a vector of repeated measures, α_{yi} is a vector of latent intercepts, β_{yi} is a vector of latent slopes, λ_{yt} is a vector of fixed/freed loadings representing time, and ε_{yit} is a vector of disturbance terms assumed to have equal variances. It is assumed that ε_{yit} has a mean of zero and is homoscedastic for all individuals at each time point, and is uncorrelated with α_{yi} , β_{yi} , λ_{yt} and ε_{yit} . Figure 1 presents a graphical representation of this model.

In addition, it is necessary to test whether income may also be modeled as two factor latent growth process. The general equation (1) may be generalized to the estimation of this trajectory. We refer to the unconditional trajectory of depressive symptoms as model 1a, and to that of household income as model 1b.

Conditional Depressive Symptoms Trajectories: Because of the complexity of the models tested here (notably with latent growth trajectories causing and being caused by other variables), we provide a graphical description of the models tested in Figure 2.

In model 2 we establish the link between gender, race, parents' education and the depressive symptoms trajectory in adulthood while controlling for parents' and respondent's history of depressive symptoms.

The level 1 model expressed previously in equation (1) still applies to this model. However, the level 2 equations may now be expressed as:

$$\alpha_{yi} = \mu_{\alpha y} + \gamma_{\alpha y,1} \text{male} + \gamma_{\alpha y,2} \text{black} + \gamma_{\alpha y,3} \text{faed} + \gamma_{\alpha y,4} \text{moed} + \gamma_{\alpha y,5} \text{modep} + \gamma_{\alpha y,6} \text{fadep} + \gamma_{\alpha y,7} \text{rdep} + \zeta_{\alpha yi} \quad (2)$$

$$\beta_{yi} = \mu_{\beta y} + \gamma_{\beta y,1} \text{male} + \gamma_{\beta y,2} \text{black} + \gamma_{\beta y,3} \text{faed} + \gamma_{\beta y,4} \text{moed} + \gamma_{\beta y,5} \text{modep} + \gamma_{\beta y,6} \text{fadep} + \gamma_{\beta y,7} \text{rdep} + \zeta_{\beta yi} \quad (3)$$

In turn, model 3 introduces respondent's education to explore the pathway through which early SEP is mediated by respondent's education in affecting the depressive symptoms trajectory in adulthood.

Again, the level 1 model expressed previously (1) still applies to this model. However, the level 2 equations may now be expressed as:

$$\alpha_{yi} = \mu_{\alpha y} + \gamma_{\alpha y,1} \text{male} + \gamma_{\alpha y,2} \text{black} + \gamma_{\alpha y,3} \text{faed} + \gamma_{\alpha y,4} \text{moed} + \gamma_{\alpha y,5} \text{modep} + \gamma_{\alpha y,6} \text{fadep} + \gamma_{\alpha y,7} \text{rdep} + \gamma_{\alpha y,8} \text{edu} + \zeta_{\alpha yi} \quad (4)$$

$$\beta_{yi} = \mu_{\beta y} + \gamma_{\beta y,1} \text{male} + \gamma_{\beta y,2} \text{black} + \gamma_{\beta y,3} \text{faed} + \gamma_{\beta y,4} \text{moed} + \gamma_{\beta y,5} \text{modep} + \gamma_{\beta y,6} \text{fadep} + \gamma_{\beta y,7} \text{rdep} + \gamma_{\beta y,8} \text{edu} + \zeta_{\beta yi} \quad (5)$$

Finally, in model 4, we test the hypothesis that both early life and education impact the trajectory of depressive symptoms in midlife through the trajectory of income.

Missing data and Fitting Function—Missing data were handled with a Full Information Maximum Likelihood estimator that calculates the likelihood for each individual given their available information. This allows individuals missing on independent or dependent variables to remain in the analytic sample, and more importantly, allows individuals to contribute to the trajectory portions (income and depression) of the model until they drop out. This resulted in an analytic sample size of 9,348 respondents. Data were weighted with longitudinal sample weights created expressly for the purposes of our analysis to account for attrition, and standard errors were adjusted for the intra-household clustering design of the NLSY79. All analyses were carried out with STATA v.10 and MPlus v. 6.11.

Results

Descriptive Statistics

Table 1 shows the unweighted descriptive statistics for the variables included in the models. Roughly half the sample was male and black and Hispanic individuals respectively made up 30% and 20% of the sample. These high proportions are due to oversampling, which we control for in the analytic models with sample weights. Parents had an average of 10.8 years of education while respondents averaged 12.7 years of education. The prevalence of parental history of depression was very low (about 1% of the sample), as was respondents' early history of depression (0.2 %). Despite these low counts, we still included these variables in the models due to their theoretical importance in assessing causality.

Unconditional Models

Results from Table 2 show that linear trajectories fit the data extremely well for both depressive symptoms and household income. For the two-factor latent growth model, the chi-squares tests are not significant, meaning that the observed covariance structure is not significantly different from the structural model we tested. Other measures also suggest

excellent fit: in both cases, the TLI and IFI are close to 1, and the RMSEA are below 0.05 (depressive symptoms: TLI=1.001, IFI=1.000, RMSEA=0.007; household income: TLI=0.998, IFI=0.998, RMSEA=0.000). For depressive symptoms, the mean of the latent intercept is 3.840 ($p<.001$) and the mean of the latent slope is -0.303 ($p<.05$) showing that on average, individuals begin the trajectory (around ages 28 to 34) with a mean of approximately 3.8 on the depressive symptoms scale and decline by 0.3 units at each wave. In contrast, the positive slope of the income trajectory suggests that household income tends to increase by 11,084\$ ($\exp(0.103)*10,000$) per time point of measurement from an intercept of about 49,579\$ ($\exp(1.601)*10,000$) as individuals age from their early 30s into their early 50s.

Conditional Models

In Table 3 we present the results of the three conditional models, respectively assessing the impact of parents' education on their offspring's depressive symptoms (model 2), the mediating role of respondents' education to this relationship (model 3) and finally the full pathway model assessing the contribution of household income (model 4). For ease of interpretation, we present only the substantively important variables, and thus exclude year of birth and indicator variables for not knowing parents education; none of these variables had effects that were either substantively important in terms of magnitude or statistically significant (in contrast with parents' depression for instance, which, although they are not statistically significant, have relatively strong effects in terms of magnitude, and hence were kept in the table).

All the models show excellent fit, both in terms of the Chi-square (despite the fact that it reaches significance with model 4, which is to be expected with sample sizes >400) and the other fit statistics. Because the models are built incrementally and estimate full mediating pathways, some variables (such as respondents' education) can be both predictors and outcomes in the same model, as shown in Figure 2. To represent this complex pathway structure, we list the dependent (outcome) variables for each model across the second row of the table in the order of appearance in the causal pathway, as well as the predictors in the usual first column position. Hence, in model 2, depressive symptoms is the only outcome variable, while in model 3, respondents' education is also an outcome of parents' education and other sociodemographic characteristics. In model 4, the income trajectory is predicted by both early SEP and respondents' education.

We will first briefly describe the effect of background and control variables, and then turn to the socioeconomic pathways in these models. Black individuals tend to have a higher depressive symptoms intercept than white individuals, but there is no difference on the slope. In contrast, while Hispanic individuals do not differ from White individuals on the intercept of depressive symptoms, they have a faster rate of decline of symptoms across this period. Furthermore, while the effect for Black individuals disappears with the inclusion of income trajectories in the model, the Hispanic effect is robust throughout all model specifications. Similarly, male respondents have a lower intercept of depressive symptoms than females, and this effect persists from model 2 to model 4. Finally, early depression proves to be a very strong predictor of an increased intercept of depressive symptoms (though it has no effect on the slope).

Turning now to early SEP, in model 2, we see that both father's education and mother's education tend to decrease the intercept of depressive symptoms, though they do not significantly affect the slope. In addition, the direct effect of mother's education is 46% larger in magnitude than that of father's education. However, in model 3, these direct effects lose their statistical significance and substantially decrease in magnitude, indicating that much of the direct effect of parents' education on their offspring's adult mental health

observed in model 2 is in fact mediated by this offspring's educational attainment. Adding income to the model (in model 4) does not affect this relationship.

In model 3, respondent's education has a strong negative direct effect on the intercept of depressive symptoms, but this effect is reduced by 65% when income is introduced in model 4. This suggests that much, though not all, of the effect of education on depressive symptoms in this sample is mediated by the more proximal determinant of income.

Finally, in model 4, we see that the income intercept and slope respectively have strong negative direct effects on the depressive symptoms trajectory intercept and slope. More specifically, each unit increase on the logged income scale (corresponding to about 27,000\$) leads to a reduction in the intercept of 2 units on the depressive symptoms scale, and 1.6 units on the slope. In comparative terms, it would take 20 years of education (as per the effect size in model 4) to achieve the same reduction in risk.

We can also decompose these effects into their direct, indirect and total components, as shown in Table 4. These direct, indirect and total effects were calculated with depressive symptoms intercept as the outcome, and provide a relative magnitude of the various effects pathways. First, as would be expected, the total effects calculated in this model 4 approximate very closely the direct effects estimated in model 2 and model 3 for mother and father's education and respondent's education respectively. Secondly, this decomposition confirms that most of the effect of parents' education is in fact indirect, as mediated by respondent's own education (including the pathway from education to income), and that the bulk of the respondent's education effect is itself mediated by income.

Discussion

In this paper, we assessed the direct and indirect effects of early SEP, respondent's education and household income on trajectories of depressive symptoms in mid-adulthood. In addition to explicitly modeling these pathway effects through structural equation modeling, we used growth curve models to render the dynamic nature of both income growth and the evolution of depressive symptoms in early and mid-adulthood.

We had hypothesized, and indeed found, that depressive symptoms in adulthood could be captured through two dimensions, namely an intercept, or baseline range of depressive symptoms that varied between individuals, and a slope describing the average evolution of depressive symptoms over the years. Moreover, in keeping with previous studies on the relationship between age and depressive symptoms (Kasen et al., 2003; Kessler et al., 1992; Mirowsky and Ross, 1992; Newmann, 1989; Schieman et al., 2001; Wade and Cairney, 1997; 2000), we found that depressive symptoms tended on average to decrease in the sample, as the respondents aged from their 30s to their 40s and 50s. This is an important confirmation with longitudinal cohort data of findings that were often limited to analyses with cross-sectional data.

In turn, we found limited support for a direct effect of early SEP on adult depressive symptoms. Instead, much of the effect of parents' education appeared to be mediated by respondent's own education, which in turn was largely mediated by income. This observation goes against a number of studies having found a persistent direct effect of early SEP on adult mental health (see notably Luo and Waite, 2005; Power et al., 2002; Stansfeld et al., 2010). However, as we argue in the introduction, the explicit modeling of pathways in SEM allows us to take into account correlated error terms and to model the covariance structure between interrelated predictors, and thus to limit the potential bias in the estimation of those effects. Therefore, it may be that the previous observations of only partial mediation of early SEP by adult SEP were in fact due to biased estimates.

These results should in no way be taken to mean that early SEP does not affect adult depressive symptoms however. Indeed, we do show that parents' education has an important total effect on adult outcomes, albeit a primarily indirect one. As such, the message to take away from these analyses is that the socioeconomic pathway from parents' education to respondent's education and then to household income is a crucial mechanism for adult mental health. Moreover, by drawing parallels from this model to those examined in the age-depressive symptoms literature, we come to similar conclusions through the use of a very different method. Thus, we can interpret our findings as indicating that the life course evolution of proximate statuses, in this case, the tendency for income to increase in mid-adulthood, is strongly associated with the evolution of depressive symptoms as individuals age, or in the present case, the average decline in depressive symptoms.

We can also relate these finding to the early work done by Blau and Duncan (1967) elucidating the relationship between family origins, educational attainment, and adult occupational outcomes. More specifically, Blau and Duncan showed that while the effect of educational attainment on adult status is direct, the effect of parental background is indirect and mainly mediated by the offspring's own education. Thus, this model suggests that advantage is maintained from one generation to the next through family background processes encouraging the educational attainment of their children. The corollary of this finding is that children from lower status families could ascend the ladder of success via educational attainment, given the structural opportunity to do so. If we extrapolate from this perspective to our findings, it could be argued that the adverse consequences of low childhood socioeconomic status on adult mental health could be mitigated by policies that increase structural educational opportunities. The G.I. Bill in the U.S. constitutes an example of a policy that had a substantial impact on higher education (Jencks and Riesman, 1968), but recent evidence tends to point to the more profound and lasting effects of intervening at the other end of the educational spectrum, notably through the development of early childhood education programs in subsidized daycare settings to ensure that preschoolers begin elementary school on an equal footing (Mayer and Peterson, 1999).

However, we must also highlight some limitations in this paper that may cast doubt on the waning effect of parental education over the life course. It may be that parents' education is too crude an indicator to capture the effect of early SEP, or the wrong indicator with which to do so.

Interestingly though, the fact that mother's education had a stronger effect on depressive symptoms in adulthood (relative to father's education) does suggest that this effect may be related to the child's environment while growing up (and especially childrearing practices). As such, measures of birth weight (see Vasiliadis et al., 2010) and parenting skills (see Power et al., 2002) would allow a better test of this hypothesis, but were unfortunately not available in these data. Alternatively, these results may also indicate that childhood is indeed a sensitive period to the effects of SEP, but that the strength of this effect wanes as individuals progress through the life course and more proximate effects of adult SEP take precedence. This alternative explanation is in line with recent research on U.S. mobility over the life course (Warren et al., 2002).

In this paper, we only show household income measures, but we did conduct numerous sensitivity analyses to ensure that our conclusions were not unduly affected by parallel processes. In order to replicate analyses with an alternate measure of financial resources and control for family size, we ran these models using an income to needs ratio (models not shown), which yielded substantively similar results but a poorer model fit. We also split the sample by race and found no substantive differences in the effects of adult SEP reported here. All trajectories were tested with alternate specifications, particularly the income and

depressive symptoms trajectories since the last wave of reporting differed in timing for individuals.

Finally, while these results are telling, we must point out that a number of questions remain unanswered. First, we consider that the next logical step is to disaggregate these processes by gender. Although we did validate our model with both subgroups, the line of research on age and depressive symptoms (e.g. Mirowsky, 1996), suggests fruitful avenues for exploring gender variations in other predictors of mental health known to be salient for individuals in midlife, such as marriage, childbearing, physical health, stressors and social support. More specifically, much of the research on mental health in the medical sociology literature suggests that marital history, and especially specific transitions, are salient to mental health in adulthood (e.g. Barrett, 2000). It is possible that our measure of household income acted as a proxy for some of these processes, but testing this specific hypothesis proved to be beyond the scope of our study.

In sum, we found in this study clear evidence that depressive symptoms trajectories in adulthood are part of life course pathways of status attainment that take root in parents' education, but that may be mitigated by intervening circumstances, and particularly, the offspring's own educational attainment.

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Research Highlights

- Parents' education had an inverse relationship with respondents' depressive symptoms in adulthood
- This relationship was fully explained by respondents' education
- In turn, the effect of respondent's education was also largely mediated by their household income
- Adult depressive symptoms are the outcome of life course pathways of social attainment rooted in parents' education
- Increasing educational opportunities may break the intergenerational transmission of low status and poor mental health

Figure 1. Graphical representation of Equation 1 for depressive symptoms

Note: Oval shapes represent latent variables, while rectangular boxes represent observed variables. Loadings on the intercept are all fixed to 1, while loadings on the slope are fixed to the lambdas shown on the figure, representing a linear trajectory of depressive symptoms. This figure can be generalised to represent other unconditional models, such as the one estimated for income.

Figure 2. Graphical representation of the analytic design

Note: Oval shapes represent latent variables, while rectangular boxes represent observed variables. For ease of comprehension, direct relationships between control variables and all other variables in the model have been omitted from the figure, although they have been estimated in the model. Similarly, while there are only single arrows between each model box, all the paths between each observed or latent variable contained in any two given boxes estimated.

Table 1

Unweighted Descriptive Statistics (Means and Standard Deviations)

	Mean/%	Std. Dev.	N
Black	30.3%	(0.460)	9348
Hispanic	19.7%	(0.379)	9348
Male	49.9%	(0.500)	9348
Father's Depression	1.2%	(0.110)	9348
Father's Education	10.862	(3.720)	9348
Father's Education Missing	15.1%	(0.358)	9348
Mother's Depression	0.9%	(0.095)	9348
Mother's Education	10.788	(3.167)	9348
Mother's Education Missing	6.7%	(0.249)	9348
Early Depression	0.2%	(0.046)	9348
Education Max 24–26	12.672	(2.284)	9211
CESD Mean 28–34	4.050	(3.361)	8184
CESD Mean 35–39	3.869	(3.985)	2986
CESD Mean 40–51	3.477	(4.160)	8374
Income Median 28–34	1.490	(0.583)	9127
Income Median 35–39	1.581	(0.674)	8307
Income Median 40–51	1.642	(0.723)	8369

Table 2

Means and Variances of the Random Intercepts and Slopes and Overall Fit Measures for Models 1a and 1b

	Model 1a. Depressive Symptoms N=9348		Model 1b. Household Income N=9248	
	Intercept (α)	Slope (β)	Intercept (α)	Slope (β)
Mean (s.d.)	3.840 *** (0.047)	-0.303 *** (0.029)	1.601 *** (0.008)	0.103 *** (0.004)
Variance (s.d.)	5.770 *** (0.231)	0.000 (0.000)	0.261 *** (0.010)	0.057 *** (0.005)
Cov(α, β) (s.d.)	0.000 (0.000)	---	0.001 (0.005)	---
χ^2 (df)	4.523(3)	---	0.027(1)	---
<i>prob.</i>	0.210	---	0.870	---
TLI	0.998	---	1.001	---
CFI	0.998	---	1.000	---
RMSEA	0.007	---	0.000	---

*
p<.05,**
p<.01,***
p<.001.*Note:* Factor loadings for both trajectories set at 0,1,1.85

Parameter Estimates of Models 2–4

Table 3

Outcomes	Model 2: Early SEP			Model 3: Respondent's education			Model 4: Household Income Trajectory			
	CES-D α	CES-D β	R's Educ.	CES-D α	CES-D β	Income α	Income β	R's Educ.	CES-D α	CES-D β
Black	0.602 *** (0.107)	−0.079 (0.070)	0.010 (0.061)	0.605 *** (0.106)	−0.079 (0.070)	−0.323 *** (0.015)	−0.043 *** (0.009)	0.012 (0.061)	−0.050 (0.117)	−0.098 (0.078)
Hispanic	0.182 (0.140)	−0.251 *** (0.092)	0.301 *** (0.092)	0.261 (0.137)	−0.246 *** (0.092)	−0.058 *** (0.020)	−0.016 (0.012)	0.294 *** (0.091)	0.149 (0.137)	−0.267 *** (0.093)
Male	−1.078 *** (0.090)	0.011 (0.058)	−0.108 *** (0.048)	−1.108 *** (0.090)	0.011 (0.058)	0.008 (0.012)	0.020 *** (0.008)	−0.108 *** (0.048)	−1.093 *** (0.089)	0.049 (0.059)
Father's Depression	0.778 (0.438)	0.213 (0.232)	---	0.744 (0.431)	0.200 (0.232)	---	---	---	0.708 (0.391)	0.162 (0.236)
Mother's Depression	0.306 (0.444)	0.611 (0.338)	---	0.281 (0.436)	0.604 (0.337)	---	---	---	0.205 (0.432)	0.573 (0.326)
Early Depression	7.147 *** (1.454)	−1.645 (1.035)	---	6.984 *** (1.469)	−1.665 (1.044)	−0.432 *** (0.115)	−0.118 (0.077)	---	6.021 *** (1.414)	−1.731 (1.044)
Father's Education	−0.048 *** (0.016)	0.005 (0.010)	0.175 *** (0.011)	0.000 (0.017)	0.008 (0.011)	0.007 *** (0.003)	0.003 * (0.001)	0.174 *** (0.011)	0.013 (0.017)	0.012 (0.011)
Mother's Education	−0.070 *** (0.021)	−0.025 (0.013)	0.235 *** (0.015)	−0.007 (0.021)	−0.019 (0.013)	0.010 *** (0.003)	−0.001 (0.002)	0.235 *** (0.015)	0.015 (0.021)	−0.023 (0.014)
Respondent's Education	---	---	---	−0.275 *** (0.024)	−0.017 (0.015)	0.088 *** (0.004)	0.022 *** (0.002)	---	−0.097 *** (0.026)	0.005 (0.017)
Income α	---	---	---	---	---	---	---	---	−2.016 *** (0.140)	---
Income β	---	---	---	---	---	---	---	---	---	−1.643 *** (0.214)
Intercept	5.630 *** (0.272)	−0.011 (0.164)	8.322 *** (0.172)	7.931 *** (0.341)	0.112 (0.211)	0.345 *** (0.046)	−0.199 *** (0.028)	8.319 *** (0.172)	8.618 *** (0.342)	−0.248 (0.222)
Var.	5.142 *** (0.220)	0.000 (0.000)	3.779 *** (0.086)	4.824 *** (0.211)	0.000 (0.000)	0.164 *** (0.005)	0.039 *** (0.003)	3.780 *** (0.086)	4.183 *** (0.191)	0.000 (0.000)
χ^2 (df)	21.845 (14)	28.925 (18)	108.350 (42)							
prob.	0.082	0.049	0.000							
TLI	0.988	0.992	0.990							
CFI	0.995	0.997	0.996							
RMSEA	0.008	0.008	0.013							

* p<.05,
** p<.01,
*** p<.001.

Table 4

Direct, indirect and total effects of main SEP variables on the intercept of depressive symptoms, estimates from model 4

	Mother's education	Father's education	Respondent's education
Direct effect	0.015	0.013	−0.097
Indirect effects			
via respondent's education	−0.023	−0.017	
via income α	−0.020	−0.015	−0.177
via respondent's education and income α	−0.042	−0.031	
Total effect	−0.069	−0.049	−0.274

Note: Indirect effects are calculated by taking the product of all paths leading from the predictor to the outcome (for example for the indirect effect from Mother's education to depressive symptoms intercept going through the respondent's education: 0.235×-0.097), and total effects are the sum of all direct and indirect effects.