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A Life Course Model of Self-Rated Health through Adolescence and Young Adulthood

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Abstract

This paper proposes and tests a life course model of self-rated health (SRH) extending from late childhood to young adulthood, drawing on three waves of panel data from the U.S. National Longitudinal Study of Adolescent Health (Add Health). Very little research has examined SRH during the early decades, or whether and how these self-assessments reflect experiences in the family of origin. Background characteristics (parental education, income, and family structure), parental health conditions (asthma, diabetes, obesity, migraines), and early health challenges (physical abuse, presence of a disability, and parental alcoholism and smoking) predict SRH from adolescence to young adulthood. These experiences in the family-of-origin are substantially mediated by the young person's health and health behaviors (as indicated by obesity, depression, smoking, drinking, and inactivity), although direct effects remain (especially for early health challenges). Associations between SRH and these mediators (especially obesity) strengthen with age. In turn, efforts to promote healthy behaviors in young adulthood, after the completion of secondary school, may be especially strategic in the promotion of health in later adulthood.

Keywords

life course; self-rated health; parent health; adolescent health; U.S.A

Introduction

A large body of research has established associations between childhood socioeconomic position (SEP) and adult health and mortality (Elo, 2009; Palloni, 2006), including self-

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assessed health (Haas, 2007; Luo & Waite, 2005; Warren, 2009). With few exceptions, such work has examined childhood SEP and health from mid- to late-adulthood and has not examined the mediational processes that might explain these observed associations. As reviews of this literature observe, however, identifying such mediators early in the life course is a strategic next step in understanding the emergence of health inequalities, and may inform effective efforts at prevention and intervention (Avison et al., 2010; Galobardes et al., 2008).

The present paper begins to "fill in the gap" between childhood SEP and health in mid- to late-adulthood in four ways. First, drawing on data from the National Longitudinal Study of Adolescent Health (Add Health), we examined health from adolescence into young adulthood, a time when differences in objective health are emerging and likely coalescing into "pre-disease pathways" that carry into later life (Harris, 2010; Singer & Ryff, 2001). We focus on self-rated health (SRH) because of its central place in the childhood SEP-adult health literature; because it is reliably associated with mortality and cause-specific mortality even with extensive controls (Benjamins et al., 2004; DeSalvo et al., 2006; Jylhä et al., 2006); and because it is considered especially appropriate for assessments of health in young adulthood, when clinical endpoints are relatively uncommon (Kestilä et al., 2009).

Second, we considered parental health and early challenges (such as physical abuse and having an alcoholic parent), which typically have not been examined. Such factors may partially account for the association between childhood SEP and later health but, in any event, their inclusion provides a more complete description of early circumstances (Gilman & McCormick, 2010; Taylor et al., 2011). Third, we examined the extent to which the effects of parental health and early health challenges are mediated by central indicators of young people's health and health behaviors. A few studies suggest that such factors mediate associations between education and health in adulthood (Dupre & George, 2011; van de Mheen et al., 1998), but almost no research has examined mediators for the childhood SEP-young adult health association (Kestilä et al., 2009).

Fourth, several life course models suggest dynamic processes by which early experiences are associated with SRH in young adulthood. Life course epidemiology recognizes a chain-of-risks model (Hayward & Gorman, 2004; Kuh et al., 2003) according to which early disadvantages increase the likelihood of subsequent risks, a mediational sequence that in turn diminishes health. A chain-of-risks model, however, may be complicated by other life course processes, particularly the accumulation and the timing of risks. We therefore tested a chain-of-risks model and then considered these additional nuances.

A Chain-of-Risks Model of Self-Rated Health in Young Adulthood

The overarching conceptual model is shown in Figure 1. Childhood SEP is likely the most investigated early source of adult health disparities (Galobardes et al., 2008), but parental health and several serious early challenges are also likely candidates. Poor parental health may influence young adult health through several mechanisms (Coneus & Spiess, 2012), although few studies have examined this possibility, especially among young adults. Parents with poor health may serve as role models for poor health-related behaviors, including poor

diets and eating patterns (Kral & Rauh, 2010), negativity (DiMarco et al., 2011), inactivity (Richards et al., 2009), and smoking and drinking (Göhlmann et al., 2010). Moreover, parental health problems often coincide with increased psychosocial stressors, which are associated with depressed mood and the quality of sleep in adulthood (Koskenvuo et al., 2010). These considerations suggest that parental health problems predict young adult health and that these associations will be mediated by health-related behaviors and depressive symptoms. Poor parental health may also indicate genetic predispositions for illnesses that in turn are present among the second generation, raising the possibility of direct, unmediated effects with later health status.

A growing body of evidence suggests that early health challenges may also have lasting impacts on health. Low birth weight may have long-term implications for health, perhaps including high blood pressure (Adair & Dahly, 2005), type 2 diabetes and osteoporosis (Gluckman et al., 2008), and altered kidney function and adiposity (Christian & Stewart, 2010). Exposure to smoking is also a known risk factor for later health problems both directly [e.g., by sensitizing airways to allergens (Lim & Kobzik, 2009)] and indirectly through the off-spring's increased likelihood of smoking (Beyer et al., 2009; Gilman et al., 2009). These considerations suggest that low birth weight and exposure to smoking in the household will influence young adult health and these effects will be partially mediated by the adolescent's health behaviors related to smoking; some of the effect, however, may not be mediated, reflecting physiological changes experienced by the child.

There is also mounting evidence that early psychosocial stressors alter stress reactivity, resulting in pro-inflammatory tendencies that persist into adulthood (Chen, 2010; Taylor, 2010). According to the biological embedding hypothesis (Coe & Laudenslager, 2007; Miller et al., 2009), chronic childhood stressors are associated with durable, heightened reactivity to stressors (both psychosocial and biological), which in turn predict diseases in adulthood. Jylhä (2009) suggests that such differences in stress reactivity create introceptive differences (i.e., in physical sensations, including pain and discomfort) that result in inter-individual differences in SRH. These considerations suggest that serious stressors early in life will have direct, unmediated effects on later health because of durable, heightened reactivity to stressors. Early stress is also known to increase the likelihood of depressive symptoms (and hence inactivity and obesity), suggesting that some of the effects of early stressors will be mediated by such factors. Add Health includes measures of serious physical abuse and parental alcoholism.

Further, although disabilities resulting in serious functional impairments are relatively uncommon among youth, they are often associated with long-lasting challenges to health because of compromised motor skills, inactivity, obesity, and depression (Cervantes & Porretta, 2010; Rimmer et al., 2010). Moreover, even disabilities and health challenges that are not markedly debilitating are known to increase chronic stressors in the household (Miodrag & Hodapp, 2010) and compromise social competence and skills at school and in peer groups (Cook & Oliver, 2011). These considerations suggest that the presence of an early disability will predict health in young adulthood and that this association will be mediated by health-related behaviors and negativity.

In summary, SEP, parental health (migraines, allergies, asthma, diabetes, and obesity), and early challenges (low birth weight, physical abuse, parental alcoholism, smoking in the household) were expected to have direct associations with SRH in young adulthood (Hypothesis 1), and also to be mediated by way of health behaviors and indicators of health (BMI, depressive symptoms, inactivity, smoking, and drinking) (Hypothesis 2).

Age-Graded Patterns of the Chain-of-Risks Model

Research that examines chain-of-risk models rarely considers additional complexities regarding the timing of risks and their accumulation. Yet these possibilities are not mutually exclusive and may work in tandem. The data allowed us to test several prominent life course hypotheses, in addition to the base, chain-of-risks model. First, according to cumulative disadvantage and cumulative inequality theories, differences attributable to initial risks—particularly SES of the household-of-origin--may be magnified over time (Ferraro et al., 2009; O'Rand, 1996), akin to "compound returns" on risk factors (DiPrete & Eirich, 2006). Thus, the effect of SEP may be moderated by age.

Second, according to an age-of-exposure hypothesis, the salience of risk factors (including indicators of poor health and health behaviors) vary by age when the risk occurred, consistent with the sensitivity model in life course epidemiology. In the present case, it may be that as young people age, their bodies lose some capacity for recovery from insults, thereby making risk factors more consequential at later ages. And third, consistent with life course epidemiology's cumulative risk model (Ben-Shlomo & Kuh, 2002), a repeated-exposures hypothesis holds that the salience of risk factors increases as they persist through time due to wear-and-tear. Indeed, Stringhini et al. (2010) shows that repeated assessments of risk behaviors over time significantly attenuate the link between childhood SEP and mortality (beyond baseline risks).

In summary, the mediational patterns suggested by the chain-of-risks model may be subject to moderation in three respects: the effects of SEP may be moderated by age (Hypothesis 3); the effects of mediators may be moderated by age (Hypothesis 4); and the effects of mediators may become more pronounced as they persist (Hypothesis 5).

Methods

Data

The National Longitudinal Study of Adolescent Health (Add Health) was based initially on a nationally representative sample of youth in grades 7 through 12 in the United States. Four waves of data have been collected and the present study draws on Waves 1, 3, and 4, which were collected at roughly seven-year intervals spanning approximately 12 to 32 years of age. The National Quality Education Database provided the sampling frame with its list of all high schools in the United States (N=26,666). To qualify, a high school had to include an 11th grade and to have an enrollment of more than 30 students. From this frame, 80 schools were selected. The sample was stratified according to several distinctions, including region, suburban/urban/rural, school type (whether public, private, parochial), ethnic mix, and size. Fifty-two of the 80 schools agreed to participate, and 28 replacement schools were selected

based on the stratifying variables. Each of the 80 schools was paired with a middle school (based on its contribution to the high school student body). A total of 145 of the schools agreed to host a confidential in-school survey that focused on adolescent health and friends. This first wave yielded 90,118 students from grades 7 to 12 (in 1994).

From the rosters, students were randomly selected for a one and one-half hour interview, conducted in the home. Approximately 200 students were recruited from schools in each school pair, regardless of size. This procedure resulted in a self-weighting sample. A total of 20,745 adolescents in grades 7 through 12 (ages 11 through 19) were interviewed at home. This in-home wave of interviews with target child and parent was carried out in 1995, between April and December. A third wave was collected between August, 2001, and April, 2002 from Wave I participants, resulting in 15,197 18-26 year olds. Data also come from Wave IV, collected between April, 2007, and February, 2009, when participants ranged from 24 to 33 years old. Of the eligible respondents who had participated in the first in-home interview, 92.5% were re-located and 80.3% were re-interviewed, resulting in 15,701 adult in-home interviews collected between January, 2008, and February, 2009.

The present analyses include all respondents with at least one non-missing observation for self-rated health across Waves 1, 3, and 4 and no missing data on covariates, the latter criterion to facilitate the bootstrapping procedure for our assessment of mediation (discussed below). This approach rests on strong assumptions about the nature of the missing data. We thus conducted preliminary analyses by constructing 10 multiply imputed datasets, a procedure that relies on weaker assumptions about the missing data, and obtained the same substantive results (available on request) as those reported in this paper. Our analysis sample consists of 28,552 observations representing 10,375 unique respondents. This paper is part of a larger project (second author, PI) that has been reviewed by the University of North Carolina IRB.

Measures

The outcome measure, self-rated health (SRH), was assessed at Waves 1, 3, and 4 by asking “In general, how is your health” with possible responses ranging from excellent (5) to poor (1).

Parental health—Add Health includes a series of measures of parent health based on self-reports from a parent interview fielded at Wave I. The questions ask the respondent’s biological mother or biological father whether s/he currently has any of the following conditions: obesity, migraine headaches, allergies or hay fever, asthma or emphysema, and diabetes. Among the parents in our sample, allergies and migraines were somewhat common (55% and 33% respectively), a little under 25% of the respondents had an obese parent, and less than 15% of the parents suffered from asthma or diabetes (see Table 1).

Early health challenges—We considered a number of indicators of early health challenges. Parents reported on the birth weight of respondents. The child respondent was classified as low birth weight if the birth weight was less than or equal to 2500 g. Parents also reported whether the biological mother or biological father suffered from alcoholism and whether s/he or other people considered the child to have a disability. The parent

interview included a question about whether anyone in the household currently smoked, a measure of early exposure to cigarette smoke. We included a measure of physical abuse based on retrospective reports from respondents at Wave 3. Respondents were asked how often a parent or caregiver slapped, hit, or kicked them prior to starting 6th grade. The response categories ranged from "this have never happened" (0) to "more than ten times" (5). Adolescents who reported this happening more than 10 times were classified as having been physically abused. Among the respondents in our sample, 45% lived in a household with someone who smoked, 15% had an alcoholic parent, and 9% were classified as low birth weight (see Table 2). Serious physical abuse and having an early disability were rare, occurring among 6% and 1% of youth, respectively.

Health and health-related behaviors from adolescence to adulthood—We examined five measures of health and health behaviors that were each repeated across the three waves of data covering the transitions from adolescence to adulthood (at mean ages of about 16, 22, and 28). Body mass index (BMI) is weight in kilograms divided by height in meters squared. We classified respondents into four categories at each wave: underweight (BMI < 18.5), normal weight (18.5 ≤ BMI < 25), overweight (25 ≤ BMI < 30), and obese (BMI ≥ 30) (NHLBI, 1998). Depressive symptoms consists of the average of five items asking respondents during the past seven days how often they were (1) "bothered by things that usually don't bother you," (2) "could not shake off the blues, even with help from your friends," (3) "felt depressed," (4) "felt that you were too tired to do things," and (5) "felt sad." Possible responses ranged from "never or rarely" (0) to "most of the time or all of the time" (3). Cronbach's alphas for the scales were 0.78, 0.75, and 0.78 for Waves 1, 3, and 4 respectively.

Add Health includes a series of questions asking respondents how many days per week they engage in a variety of physical activities. The possibilities range from outdoor activities and organized sports to various forms of exercise.[i] Respondents were coded as physically inactive if they reported spending less than three days per week engaged in any physical activity. Respondents were also classified as either never having smoked at least a cigarette a day for a month, ever having smoked a cigarette a day for a month, and currently smoking one cigarette per day over the last month at the time of the survey. We also classified respondents as non-drinkers, light drinkers (drink less than 3 per week), or moderate to heavy drinkers (drink at least 3 days per week).

Table 2 reveals a number of notable changes in health behaviors as the respondents make the transition from adolescence to young adulthood. The percentage of obese members of the sample increased from 7% to 36%; the percentage of moderate to heavy drinkers rose from 3 to 11%; and the percentage of current smokers increased from 8 to 21% over this period in the life course. Concurrent with the increases in these health risk behaviors, there was a similar increase in the percentage of respondents who were not physically active, from 55% to 72%. In contrast to the trends in health behaviors, mean depressive symptoms was relatively stable. Thus, this phase of life is a strategic time to study health patterns given notable changes in health behaviors.

Demographic and socioeconomic measures include age, sex, race/ethnicity (non-Hispanic white, non-Hispanic black, Hispanic, and other races), family structure at Wave 1, and parent education. Hispanics include any respondent self-identifying as Hispanic, even if they also identify as white, black, or another race. Family structure is a five category variable that differentiates respondents living with two biological parents, two parents with at least one non-biological, a single mother, a single father, or some other arrangement (Harris, 1999). Parent education ranges from 8th grade or less (0) to professional training beyond a four-year college or university (5) with scores reflecting the highest level reported for either parent. The childhood SEP literature draws on diverse measures of SEP (Galobardes et al., 2008), but parental education is considered especially salient for later health in contemporary American cohorts (Miech & Hauser, 2001).

Auxiliary analyses also examined parent report of household income at Wave I. Because the inclusion of income results in a substantial loss of cases, the tabled findings focus on parental education and household structure as indicators of SEP. In auxiliary analyses, we included a measure of logged family income in the models (available on request). Based on 10 complete data sets constructed using multiple imputation, we found that the effects of family income followed a similar pattern as the effects of parent education across the models and that the effect of parent education remained statistically significant, albeit somewhat reduced, when family income was included in the models. In contrast to much prior research, these indicators of SEP are not retrospectively recalled and thus their associations with health will be comparatively larger than those observed by studies using recalled information (Galobardes et al., 2008).

We included a measure of the child's educational attainment (Galobardes et al., 2008): the number of years of education completed by each wave, which captures the dynamic nature of educational attainment during the transition from adolescence to young adulthood. At wave 1, respondents had completed an average of 8.54 years of education with variation largely reflecting the different ages when respondents were first interviewed (see Table 2). By the time the respondents were in their late 20s and early 30s, they had completed an average of 14.58 years of education.

Statistical Analyses

The analyses tested the mediational, chain-of-risks model (Hypotheses 1 and 2), and then evaluated the moderation of parental SES (Hypothesis 3) and the mediators (Hypothesis 4) by age, and the effects of the mediators as they persisted (Hypothesis 5). Random intercept models were used to test our hypotheses and account for the panel structure of the data:

$$SRH_{it}=\alpha+\beta^{'}\mathrm{x}_{i}+\gamma^{'}\mathrm{z}_{it}+v_{i}+s_{it}, \quad (1)$$

where *i* indexes 1, …, N individuals and *t* indexes the three waves of data that correspond to three life-course periods (adolescence, transition to adulthood, and young adulthood). The vector $\mathbf{x}_{\mathrm{i}}$ includes the time-invariant variables (background characteristics, parent health, and early disadvantages), the vector $\mathbf{z}_{\mathrm{it}}$ consists of time-varying variables (age, health behaviors and mental health), ν_{i} is a between-individual error term, and $\varepsilon_{\mathrm{it}}$ is a within-individual error term. *SRH* is a time-varying continuous variable (based on Waves I, III, and

IV).[ii] A series of regression models are estimated that enter sequential blocks of variables: age and background characteristics only (Model 1), Model 1 + measures of parent health (Model 2), Model 2 + measures of early disadvantage (Model 3), Model 3 + educational attainment (Model 4) and, finally, Model 4 + health behaviors and depressive symptoms (Model 5).

The structure of the panel data allowed for other reasonable model specifications. In preliminary analyses we included a random slope for age, which resulted in a multilevel growth model. The model revealed little variation in the slope (age $\beta = -0.02$, $\sigma_\beta = 0.03$), supporting the adequacy of the more parsimonious specification that just included a random intercept and is consistent with research demonstrating the stability of SRH during adolescence (Boardman, 2006) We also examined a model that estimated ν_i as a vector of person-specific intercepts, which resulted in a fixed effects model. The fixed effects models focused the analysis on within-person change in SRH. This specification necessarily dropped time invariant variables (i.e., the background, parental health, and early health challenges measures). In many cases, however, fixed effects models are preferred because they rely on fewer assumptions. In preliminary analyses we compared the estimates for the time-varying variables across random effects and fixed effects models and found the same substantive pattern of effects, though the fixed effects estimates were generally smaller than the random effects estimates (results available on request). In addition, as a diagnostic, we examined histograms of the level-1 residuals and empirical Bayes estimates of the random effects and found them to be approximately normally distributed, suggesting that the assumptions underlying the random effects models were not notably violated.

Within the framework of this random effects model, the mediational and moderational hypotheses were tested. Prior studies relied on methods to test for mediation that can produce misleading results (MacKinnon, 2002). One way to test whether the effect of a variable *x* is mediated by another variable *m* is to check for a statistically significant decline in the coefficients for *x* with and without *m* in the model. This procedure is particularly useful when one considers a vector of potential mediators, **m**, rather than just one mediator (Preacher & Hayes, 2008). The estimated coefficients from model 3 were compared with the estimated coefficients in model 4 to determine how much respondent's education mediated the effects of background characteristics, parent health, and early health challenges. Then, the estimated coefficients from model 4 were compared with those from model 5 to determine how much the measures of health behaviors and depressive symptoms mediated the effects of background characteristics, parent health, early health challenges, and respondent education. This process involves numerous statistical tests, so a Bonferroni correction was applied when assessing statistical significance.

The computation of standard errors that allow for statistical tests of mediation is complicated in panel models due to the clustering and the possibility of cross-level mediation (Krull & MacKinnon, 2001). In the present case, time-invariant variables are mediated by time-varying variables. A bootstrap procedure with 1000 replications was used to obtain standard errors for the difference in coefficients across models (Preacher & Hayes, 2008). This procedure does not require assumptions about the distribution of the difference in

coefficients and has been shown in simulation studies to perform better than alternative approaches (Williams & MacKinnon, 2008).

Finally, we examined the moderation hypotheses. We examined whether the effects of parental education and health behaviors on SRH varied as respondents grew older by introducing a series of interaction terms with age. To facilitate the interpretation of the results, the predicted value of SRH for each age was computed for a given value of a health behavior (e.g., being obese) by treating all of the cases as if they had that value of a health behavior while averaging over all of the other covariates in the model. This approach, sometimes referred to as microsimulations or adjusted predictions, allows one to construct predicted values based on the model for subgroups of interest while otherwise maintaining the sample distribution for the remaining covariates (as opposed to the more common practice of substituting the mean values for the other covariates). Standard errors for the predicted values of SRH were obtained by the delta method (Casella & Berger, 2002; StataCorp, 2009). We plotted the adjusted predicted values along with 95% confidence intervals (95% CI) to illustrate the changing effects across ages on SRH.

Results

Chain-of-Risks Model of SRH

Table 3 reports the estimates from random intercept models regressing self-rated health on demographic and background characteristics, parent health, early health challenges, respondent education, health behaviors and depressive symptoms. Model 1 includes background characteristics and shows that, consistent with past research, females had worse SRH than males (b = −0.118), other races had lower SRH than whites (b = −0.138), higher parent education was associated with better SRH (b = 0.075), and living with two biological parents was associated with higher SRH than all other family structures. We also noted a slight downward trend in SRH as participants aged (b = −0.016). The estimates for the variance components and the intraclass correlation indicated that about 30% of the variance was between individuals and 70% was within individuals over time. The between-variance in a baseline model with no covariates was 0.508, so the background characteristics accounted for about 4% of the between-person variance ([0.508 − 0.490]/0.508 = 0.035).

Model 2 adds the measures of parental health. Consistent with expectations, four out of the five measures, namely allergies (b = −0.030), diabetes (b = −0.114), migraines (b = −0.042), and obesity (b = −0.137) had significant negative associations with SRH net of respondent background characteristics. The estimates for respondent background remained virtually unchanged, with the exception that Hispanics now showed significantly worse SRH than whites (b = −0.046). Combined, these measures accounted for an additional 1% of the between-person variance in SRH ([0.490 − 0.485]/0.485). The fact that the background effects barely changed provides evidence that these effects cannot be explained by parental health.

Model 3 includes the measures of early health challenges. Three out of the five measures, namely early disability (b = −0.362), smoker in household (b = −0.150), and physical abuse (b = −0.141) had significant negative associations with SRH. Taken together, these

measures account for an additional 2% ([0.485 − 0.476]/0.485) of the between-person variance. As with the parent health measures, there is little indication that including these variables substantially changed the effects of respondent background measures or parent health. This suggests that the respondent characteristics (including SEP), parent health, and early health challenges all had largely independent effects on the SRH of young adults.

We added time-varying years of education completed in Model 4. As anticipated, completed years of education had a significant positive effect on SRH (b = 0.057). There was some indication that the effects of background characteristics, parental health, and early health challenges on health partially operated through educational attainment. The effect of parent education, for instance, fell from 0.060 to 0.038 (a 37% decline), but most of the declines were modest and virtually all of the significant predictors from the Model 3 remained significant in Model 4.

Finally, we added the time-varying measures of health behaviors and depressive symptoms in Model 5. BMI, drinking, smoking, inactivity, and depressive symptoms had strong negative associations with SRH over time. Respondents who were overweight (b = −0.131), and obese (b = −0.455) all had significantly lower SRH than normal weight respondents; light drinkers (b = −0.036) and moderate to heavy drinkers (b = −0.086) reported lower SRH than non-drinkers; past smokers (b = −0.108) and current smokers (b = −0.271) reported lower SRH than non-smokers; inactive respondents (b = −0.129) and respondents who had more depressive symptoms (b = −0.289) had lower SRH. These measures of health behaviors and negativity accounted for an additional 15% ([0.467 − 0.398]/0.467) of the between-person variance and 2% ([0.733 − 0.717]/0.733) of the within-person variance in SRH. Overall, the final model accounted for 22% of the between-person variance in SRH and 2% of the within-person variance. Although many of the individual measures did not account for more than a percent or two of the between-person variance, the combined ability to account for about a fifth of the between-person variance in SRH is impressive, particularly among adolescents and young adults. It is much more difficult to account for the within-person variance in SRH in this age group as is reflected in the notable unexplained variance. This lack of explanatory power may reflect properties of SRH at this age and/or the spacing of observations (roughly six years apart).

Table 4 reports the results of our analysis of mediation for variables that had a statistically significant association with SRH in the first model in which they appeared. Including time-varying respondent's education in the model led to a reduction in the effects of parent education and family structure by about a third. Respondent's education played less of a mediating role for the measures of parent health and early health challenges which generally attenuated by around 5 to 10%.

Adding health behaviors and depressive symptoms to the model resulted in more significant attenuation. The effects of parent education and family structure reduced an additional 50% to 100%. In addition, the health behaviors and depressive symptoms led to large percentage declines in the effects of most of the parent health measures: diabetes (48%), migraines (41%), and obesity (63%). Finally, controlling for health behaviors and depressive

symptoms also led to moderate declines in the effects of living in a household with a smoker (51%), early disability (20%), and physical abuse (33%).

This pattern of results supports Hypothesis 2, that the indicators of health and health behaviors play a significant role in mediating the effects of childhood SEP, parental health, and early health challenges on SRH during the transition from adolescent to adulthood. Furthermore, the indicators of health and health behaviors play a more significant mediational role than respondent's education. Hypothesis 1 was also supported: despite controls and the inclusion of central candidates for mediation, parental allergies, diabetes, obesity, early disability, smoker in the household, and physical abuse had significant, direct associations with SRH.

Age-Graded Patterns of the Chain-of-Risk Model

The next set of models examined age interactions with SEP (Hypothesis 3) and health behaviors (Hypothesis 4). First, did the effect of parental education on SRH vary by age? The interaction between parent education and age was statistically significant but exceptionally small in magnitude such that the main effect of parental education dominated the interaction. Thus, there was no evidence for a substantively meaningful cumulation of the effect of SEP.

Second, were the effects of the mediators moderated by age? In support of H3, statistically significant effects were found for BMI, inactivity, smoking, and depressive symptoms. Table 5 shows that the estimates of the interactions were an order of magnitude below the estimates of the main effects for all of the measures, except for BMI. The largest age-graded effect was observed among obese respondents ($b = -0.020$). The negative sign for the interaction term coupled with the non-significant main effect for obesity and the significant negative effect for age indicated that the effect of obesity was increasingly associated with poor health across the transition from adolescence to young adulthood. Thus, four out of five mediators were age-graded, but the pattern was noteworthy for BMI only.

Figure 2 shows the results of the microsimulation for BMI. The predicted value for SRH was largely stable from age 12 to age 34 for normal-weight individuals, with a minor drop-off at the end of the age range. For obese individuals, however, predicted SRH steadily declined from around 3.8 to 3.1 by age 34, a substantial decline given the general stability of SRH among this age group. As such, the gap in predicted SRH between normal-weight and obese individuals increased over time. The non-linear pattern shown in Figure 2 is not generated by the model's parameters (i.e., age $b = -0.018$, obese $b = 0.012$ ns, obese × age $b = -0.020$) but rather by the observed values of BMI and the other covariates in the dataset. This non-linear pattern likely reflects the fact that the distribution of BMI shifts over time and is associated with other factors, such as inactivity, related to self-rated health.

Finally, we examined whether the effects of the mediators became more pronounced as they persisted (Hypothesis 5). We constructed cumulative measures for each of the health behaviors to test the repeated exposures hypotheses. These cumulative measures were highly correlated with the point-in-time measures, which is unsurprising given three waves of data. Because of the high correlations, we could not assess the effects of the cumulative measures

and the point-in-time measures in the same model. Instead, separate models for the cumulative measures and the point-in-time measures were estimated (with all of the other covariates included in the model) and AIC and BIC fit statistics compared. The point-in-time measures fit the data better than the cumulative measures (as indicated by lower AIC and BIC statistics) (results available on request), thus providing no support for the cumulation of mediating risks, Hypothesis 5.

Discussion

The substantial literature that examines associations between childhood SEP and health in mid- to late-adulthood raises the issue of the extent to which, and why, childhood SEP is associated with health at earlier points in the life course. The present study addresses this gap, developing a chain-of-risks model according to which parental education, parental health, and early health challenges predict SRH in adolescence and young adulthood, relationships mediated by the youth's health and health behaviors. We further examined several extensions, reflecting prominent life course models of health, to assess the extent to which risks were age-graded. Several conclusions are warranted.

First, experiences in the family-of-origin such as parental health and early challenges were significantly associated with SRH in adolescence and young adulthood. These include parental allergies, diabetes, migraines, obesity, early disability, smoker in household, and physical abuse. Interestingly, the effects of background characteristics, including childhood SEP, were largely not attributable to differences in parental health conditions or early health challenges. Estimates for the effects of background characteristics did not change appreciably when measures of parental health and early health challenges were added to the model. Similarly, the effects of parental health conditions did not change much when measures of early health were added to the model. These results suggest that childhood SEP, parental health, and early health conditions all have largely independent effects on SRH among adolescents that persist into young adulthood.

Second, background characteristics and these early experiences were substantially mediated by a combination of educational attainment, health, and health behaviors from adolescence onwards. Past research has focused on the extent to which health behaviors mediate the effects of childhood (and adult) SEP on adult health. Van de Mheen and colleagues' (1998) study of an adult sample in the Netherlands (between 25 and 74 years of age) found that BMI, inactivity, smoking, and drinking accounted for about 10% of the link between childhood SEP and perceived general health in adulthood. On the other hand, one cross-sectional study suggested that health behaviors may account for 15% or more of the association between childhood SEP and SRH in young adulthood (Kestilä et al., 2009).

The present study found substantially larger mediational patterns (Hypothesis 2). The indicators of health and health behaviors substantially reduced the effects of parental education and family structure by 50 to 100%. The effects of the parental health variables and early challenges also attenuated substantially, ranging from 20% (for early disability) to 63% (for obesity). If these same factors accounted for about 10% of the link between SEP and adult health, then the present findings provide a compelling reason for targeting

interventions to young adulthood, when health disparities are being much more strongly generated by these same mechanisms. Even with the inclusion of these mediators, many indicators of parental health and early challenges continue to have direct associations with SRH (Hypothesis 1).

Third, several extensions of the chain-of-risk model examined possible sources of age-grading of risks. Cumulative disadvantage and cumulative inequality theories posit that the effects of early socioeconomic disadvantages increase with age (Hypothesis 3). The results were not consistent with this expectation, although such a pattern may emerge as the panel ages. However, the mediating factors--BMI, inactivity, smoking, and depressive symptoms —were moderated by age such that their associations with SRH strengthened with age (Hypothesis 4). These patterns were generally weak, excepting BMI. Obesity in particular appears to have an increasingly large, negative effect on SRH as people transition from adolescence to early adulthood, suggesting a wear-and-tear process even at this early point in the life course. On the other hand, there was no evidence that these same mediators were more predictive of SRH as they persisted (Hypothesis 5). That is, the cumulative measures of health and health behaviors did not explain SRH more than the point-in-time measures. Thus, the findings provide some support for the age-of-exposure hypothesis over the repeated-exposures hypothesis.

The overall pattern of results suggests the value of examining “hybrid” life course models of health. The standard models of health recognized by life course epidemiology and sociology —cumulation, sensitive period, chain-of-risk, cumulative disadvantage, etc.—are rarely considered in combination, although few of these models are mutually exclusive. In the present case, the results are consistent with a chain-of-risk model that is also characterized by age-of-exposure with respect to the mediational risk factors. Other research suggests a sensitive period with respect to very early SEP and a range of indicators of health (Miller et al., 2009), as well as cumulating effects of SEP in adulthood (Willson et al., 2007). Thus, the observed chain-of-risk and cumulation of mediating risks pattern may be further complicated by sensitive period and cumulation of childhood SEP.

Several limitations should be acknowledged. More fine-grained longitudinal data that extend further into adulthood would be useful in several respects. Future research could use such data to determine whether the SEP-health associations actually strengthen as the panel ages (Hypothesis 3). In addition, although our measures explain a little over 20% of the between-person variance in SRH, they explain little of the within-person variance. It may be that within-person changes in SRH at this stage of the life course are difficult to predict, but it is possible that longitudinal data with more time points and additional measures of time-varying health related behaviors and also potentially time-varying measures of SEP, could predict within-person patterns with greater accuracy. Finally, although the data allow us to test major hypotheses suggested by diverse life course models, we were not able to test all aspects of these models. For example, Ferraro and his colleagues (2009) derived a series of propositions that describe how the effects of early inequality accumulate across many years. This model served as an impetus for testing the age interaction involving parental education, but we were not able to test all of that model’s propositions.

Nevertheless, the present paper draws on nationally representative data to elucidate connections between childhood SEP and other relevant experiences in the family-of-origin and health in early adulthood. Childhood SEP, parental illnesses, and early challenges in the family-of-origin have relatively independent effects on SRH in early adulthood, which are strongly mediated by health and health behaviors, mediators that may become increasingly salient with age. Indeed, health and health behaviors appear to be much stronger mediators of childhood SEP and young adulthood SRH than childhood SEP and perceived health in adulthood (van de Mheen et al., 1998). Fostering healthy behaviors after secondary school, as many young adults are starting independent households and occupational careers, may be particularly effective in promoting positive health trajectories into later adulthood.

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Highlights

- Paper tests the effects of background characteristics, parent health, and early health challenges on young adult SRH.
- Effects of parent health and early health challenges on SRH are substantially mediated by health-related behaviors.
- Associations between self-rated health and obesity strengthen with age.

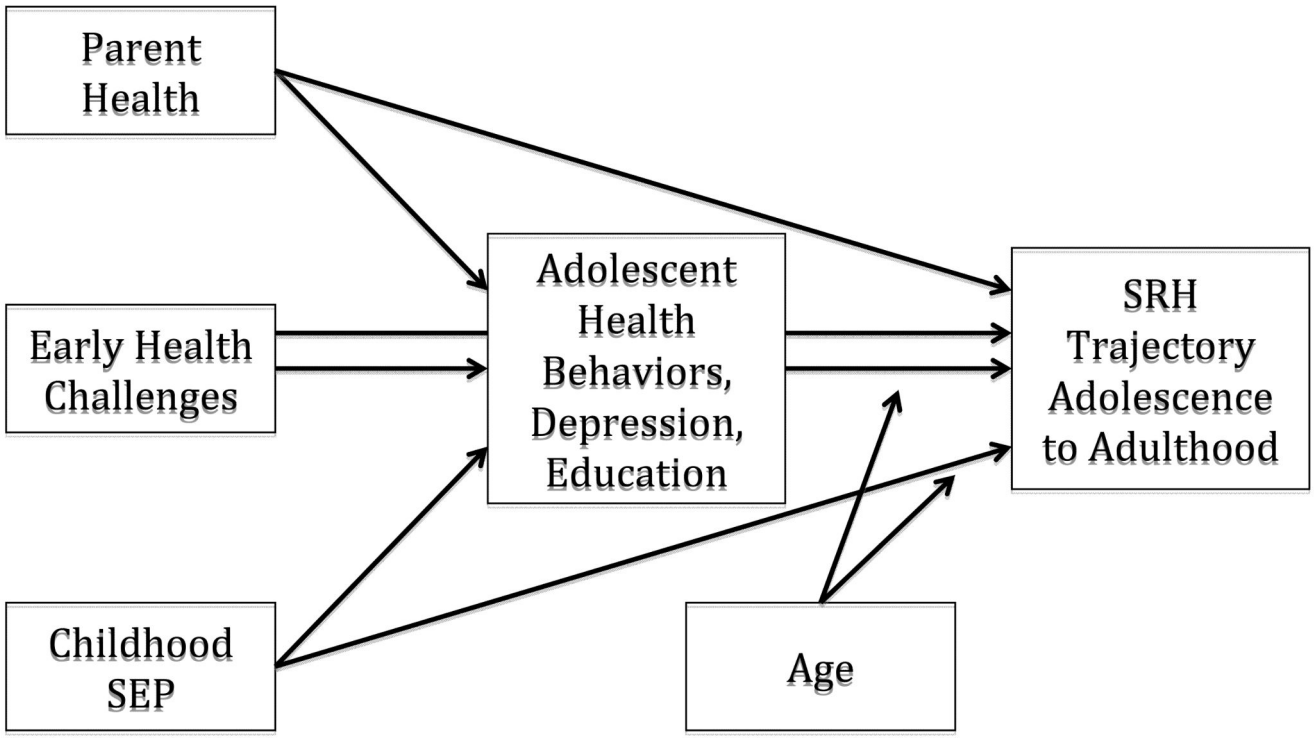

Figure 1.

Conceptual Model Illustrating Mediating and Moderating Effects for Parent Health, Early Health Challenges, and Childhood SEP on SRH.

Notes: Measures of parent health include allergies, asthma, diabetes, migraines, and obesity. Measures of early health challenges include early disability, household smoker, low birth weight parental alcoholism, and physical abuse. Measures of childhood SEP include built environment, family income, family structure, and parent education. Health behaviors include BMI, drinking, physical activity, and smoking.

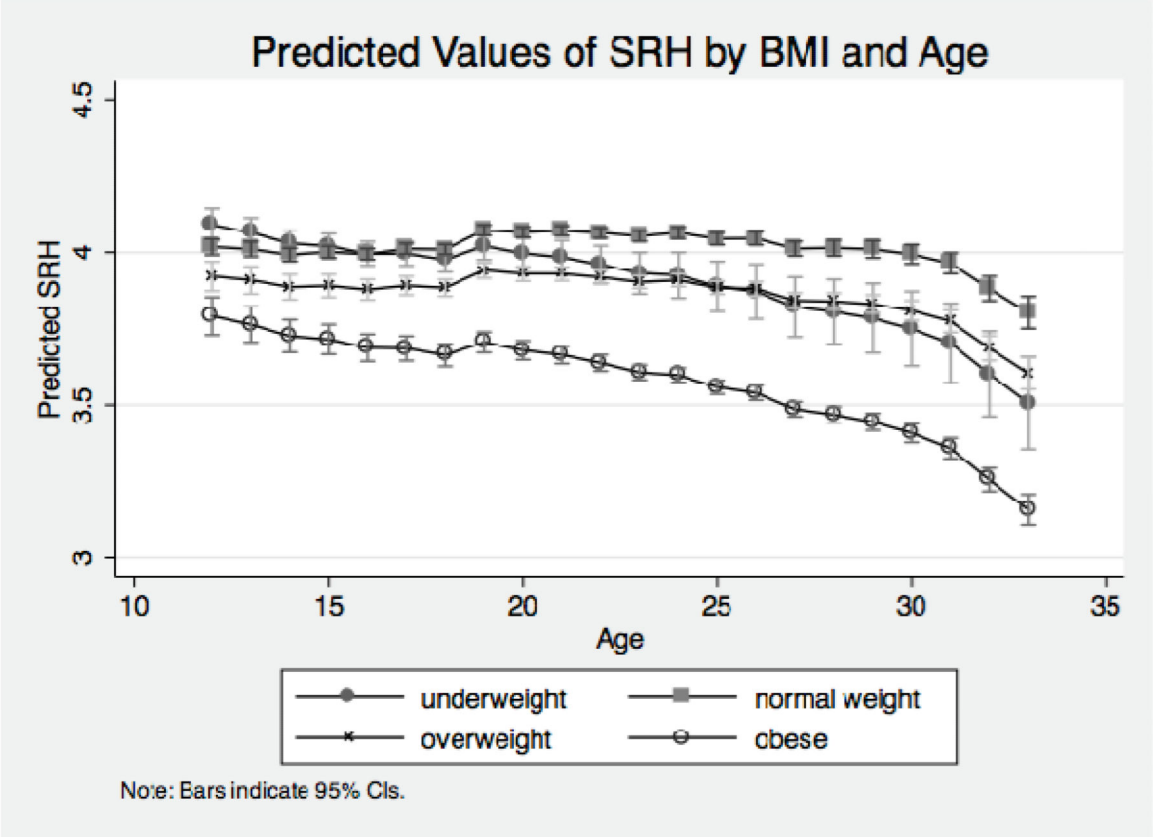

Figure 2.

[i] Wave 1 activities included: (a) roller-blading, roller-skating, skate-boarding, bicycling, (b) active sports such as baseball, softball, basketball, soccer, swimming, or football, (c) exercise such as jogging, walking, karate, jumping rope, gymnastics, or dancing. Waves 3 and 4 activities included: (a) bicycle, skateboard, dance, hike, hunt, or do yard work, (b) roller blade, roller skate, downhill ski, snow board, play racquet sports, or do aerobics, (c) team sports such as football, soccer, basketball, lacrosse, rugby, field hockey, or ice hockey, (d) individual sports such as running, wrestling, swimming, cross-country skiing, cycle racing, or martial arts, (e) gymnastics, weight lifting, or strength training, (f) golf, fishing, bowling, softball, or baseball, (g) walk for exercise.

[ii] In preliminary analyses we also estimated random intercept logit models predicting two dichotomous versions of self-rated health: (1) excellent or very good versus good, average, or poor and (2) excellent, very good, or good health versus average or poor health (see e.g., Salomon et al., 2009). We found substantively similar results with those reported below. The present analyses treat *SRH* as a continuous variable in order to facilitate our mediation analysis, which involves comparing coefficients across models.

Table 1

Descriptive Statistics for Respondent Background, Parent Health Measures, and Early Challenges; N = 10,375

	%/Mean
Male	47%
Female	53%
White	58%
Black	18%
Hispanic	14%
Other race	10%
HH: two bio parents	59%
HH: two parents	16%
HH: single mother	20%
HH: single father	2%
HH: other	3%
Parent education	3.01
Par Health: allergies	55%
Par Health: asthma	14%
Par Health: diabetes	8%
Par Health: migraine	33%
Par Health: obesity	23%
Early disability	1%
Smoker in household	45%
Low birth weight	9%
Parent alcoholism	15%
Physical abuse	6%

Table 2

Means (SD) or % for Age, Health, and Health Behaviors by Life Course Stage

	Adolescence N = 9,889		Transition to Adulthood N = 9,788		Young Adult N = 8,875	
	Mean/%	**SD**	**Mean/%**	**SD**	**Mean/%**	**SD**
Age	15.47	1.69	21.80	1.74	28.35	1.76
SRH	3.92	0.89	4.03	0.85	3.68	0.91
Very good or excel. SRH	70%		74%		59%	
Years of education	8.54	1.62	13.28	1.95	14.58	2.22
Underweight	13%		3%		1%	
Normal weight	65%		47%		32%	
Overweight	14%		28%		30%	
Obese	7%		22%		36%	
Drink: never	53%		25%		25%	
Drink: Light	44%		66%		63%	
Drink: moderate +	3%		9%		11%	
Smoke: never	81%		57%		52%	
Smoke: past	10%		21%		28%	
Smoke: current	8%		21%		21%	
Not physically active	55%		75%		72%	
Depressive symptoms	0.53	0.51	0.52	0.48	0.52	0.50

Notes: 10,375 individuals represented across the three waves of data.

Table 3

Estimates (SE) from Random Intercept Models for Self-Rated Health Regressed on Background Characteristics, Parent Health, Early Health Challenges, Education, and Health Behaviors and Depressive Symptoms

	Model 1		Model 2		Model 3		Model 4		Model 5	
	Est	SE	Est	SE	Est	SE	Est	SE	Est	SE
Age	−0.016	(0.001) ***	−0.016	(0.001) ***	−0.016	(0.001) ***	−0.043	(0.001) ***	−0.025	(0.002) ***
Female	−0.118	(0.013) ***	−0.116	(0.013) ***	−0.117	(0.013) ***	−0.139	(0.013) ***	−0.109	(0.012) ***
Black	0.008	(0.018)	0.004	(0.018)	−0.008	(0.018)	−0.004	(0.018)	−0.011	(0.017)
Hispanic	−0.036	(0.020)	−0.046	(0.020) *	−0.070	(0.020) ***	−0.071	(0.020) ***	−0.070	(0.018) ***
Other race	−0.138	(0.023) ***	−0.141	(0.023) ***	−0.150	(0.023) ***	−0.153	(0.022) ***	−0.141	(0.021) ***
Parent education	0.075	(0.005) ***	0.074	(0.005) ***	0.060	(0.006) ***	0.038	(0.006) ***	0.019	(0.005) ***
HH: two parents	−0.105	(0.018) ***	−0.113	(0.018) ***	−0.076	(0.019) ***	−0.051	(0.019) **	−0.017	(0.017)
HH: single mother	−0.085	(0.018) ***	−0.082	(0.017) ***	−0.064	(0.018) ***	−0.048	(0.018) **	−0.006	(0.016)
HH: single father	−0.187	(0.047) ***	−0.194	(0.047) ***	−0.160	(0.047) ***	−0.139	(0.046) **	−0.097	(0.042) *
HH: other	−0.103	(0.039) **	−0.113	(0.039) **	−0.090	(0.039) *	−0.056	(0.038)	0.001	(0.035)
Parents: allergies			−0.030	(0.014) *	−0.036	(0.014) **	−0.038	(0.013) **	−0.036	(0.012) **
Parents: asthma			−0.034	(0.019)	−0.024	(0.019)	−0.018	(0.019)	−0.012	(0.017)
Parents: diabetes			−0.114	(0.025) ***	−0.108	(0.025) ***	−0.101	(0.024) ***	−0.053	(0.022) *
Parents: migraine			−0.042	(0.014) **	−0.031	(0.014) *	−0.025	(0.014)	−0.015	(0.013)
Parents: obesity			−0.137	(0.016) ***	−0.136	(0.015) ***	−0.130	(0.015) ***	−0.049	(0.014) ***
Early disability					−0.362	(0.069) ***	−0.325	(0.068) ***	−0.260	(0.063) ***
Smoker in HH					−0.150	(0.014) ***	−0.129	(0.013) ***	−0.063	(0.012) ***
Low birth weight					−0.023	(0.023)	−0.022	(0.022)	−0.034	(0.021)
Parent alcoholism					−0.032	(0.019)	−0.018	(0.019)	−0.002	(0.017)
Physical abuse					−0.141	(0.027) ***	−0.145	(0.026) ***	−0.097	(0.024) ***
Years of education							0.057	(0.003) ***	0.058	(0.003) ***
Underweight									−0.027	(0.021)
Overweight									−0.131	(0.012) ***

	Model 1		Model 2		Model 3		Model 4		Model 5	
	Est	**SE**	**Est**	**SE**	**Est**	**SE**	**Est**	**SE**	**Est**	**SE**
Obese									−0.455	(0.015) ***
Drink: Light									−0.036	(0.011) ***
Drink: moderate +									−0.086	(0.020) ***
Smoke: past									−0.108	(0.014) ***
Smoke: current									−0.271	(0.015) ***
Not active									−0.129	(0.010) ***
Dep. symptoms									−0.289	(0.010) ***
_constant	4.132	(0.028) ***	4.210	(0.029) ***	4.331	(0.031) ***	4.278	(0.030) ***	4.312	(0.030) ***
between variance	0.490	(0.007)	0.485	(0.007)	0.476	(0.007)	0.467	(0.007)	0.398	(0.007)
within variance	0.733	(0.004)	0.733	(0.004)	0.732	(0.004)	0.728	(0.004)	0.717	(0.004)
ICC	0.309	(0.007)	0.304	(0.007)	0.297	(0.007)	0.291	(0.007)	0.235	(0.007)

Notes: N = 28,552 observations for 10,375 individuals. The reference category for race is white, for family structure is two biological parents, for drinking is never, and for smoking is never.

* p<.05

** p<.01

*** p<.001

Table 4

Bootstrapped Tests of Mediation for Estimates from Random Intercept Models Regressing Self-Rated Health on Background Characteristics, Parental Health, Early Health Challenges, Education, and Health Behaviors and Depressive Symptoms

	Model 3 - Model 4			Model 4 - Model 5		
	% Change	Est Diff	SE Diff	% Change	Est Diff	SE Diff
Age	−165%	0.027	(0.001) ***	41%	−0.017	(0.001) ***
Female	−19%	0.022	(0.001) ***	21%	−0.030	(0.003) ***
Other race	−2%	0.003	(0.002)	8%	−0.012	(0.004) *
Parent education	36%	0.021	(0.001) ***	50%	0.019	(0.001) ***
HH: two parents	34%	−0.026	(0.002) ***	66%	−0.033	(0.003) ***
HH: single mother	24%	−0.015	(0.002) ***	88%	−0.042	(0.003) ***
HH: single father	13%	−0.021	(0.004) ***	30%	−0.042	(0.009) ***
HH: other	37%	−0.033	(0.003) ***	102%	−0.057	(0.007) ***
Parents: allergies	−8%	0.003	(0.001)	6%	−0.002	(0.002)
Parents: diabetes	6%	−0.007	(0.002) **	48%	−0.049	(0.005) ***
Parents: migraine	19%	−0.006	(0.001) ***	41%	−0.010	(0.002) ***
Parents: obesity	5%	−0.007	(0.001) ***	63%	−0.081	(0.004) ***
Early disability	10%	−0.037	(0.006) ***	20%	−0.065	(0.012) ***
Smoker in household	14%	−0.021	(0.001) ***	51%	−0.066	(0.003) ***
Physical abuse	−3%	0.004	(0.002)	33%	−0.048	(0.005) ***
Years of education				−2%	−0.001	(0.001)

Notes: Only variables with significant effects in the original models are included in the table. P-values adjusted for multiple testing using a Bonferroni correction.

* p < .002 (.05)

** p < .0003 (.01)

*** p < .00003 (.001)

Table 5

Estimates (SE) for Changing Effects of BMI During the Transition from Adolescence to Young Adulthood.

	Model 1	
	Est	**SE**
Age	−0.018	(0.002) ***
Female	−0.108	(0.012) ***
Black	−0.012	(0.017)
Hispanic	−0.070	(0.018) ***
Other race	−0.143	(0.021) ***
Parent education	0.020	(0.005) ***
HH: two parents	−0.019	(0.017)
HH: single mother	−0.007	(0.016)
HH: single father	−0.099	(0.043) *
HH: other	0.000	(0.035)
Parents: allergies	−0.036	(0.012) **
Parents: asthma	−0.012	(0.017)
Parents: diabetes	−0.053	(0.022) *
Parents: migraine	−0.015	(0.013)
Parents: obesity	−0.050	(0.014) ***
Early disability	−0.267	(0.063) ***
Smoker in household	−0.064	(0.012) ***
Low birth weight	−0.035	(0.021)
Parent alcoholism	−0.004	(0.018)
Physical abuse	−0.096	(0.024) ***
Years of education	0.054	(0.003) ***
Underweight	0.282	(0.078) ***
Overweight	−0.038	(0.052)
Obese	0.012	(0.061)
Underweight × Age	−0.018	(0.004) ***
Overweight × Age	−0.005	(0.002) *
Obese × Age	−0.020	(0.003) ***
Drink: Light	−0.036	(0.011) ***
Drink: moderate +	−0.091	(0.020) ***
Smoke: past	−0.112	(0.014) ***
Smoke: current	−0.275	(0.015) ***
Not physically active	−0.130	(0.010) ***
Depressive symptoms	−0.288	(0.010) ***
_constant	4.204	(0.035) ***

	Model 1	
	Est	**SE**
between variance	0.400	(0.007)
within variance	0.715	(0.004)
ICC	0.238	(0.007)

Notes: N = 28,552 observations for 10,375 individuals.

* p < .05

*** p<.001