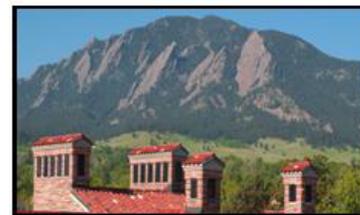


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WORKING PAPER

Unhealthy parents delay college graduation for their children

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Abstract

While the intergenerational relationship between SES and health has been a staple of sociological and epidemiological research, there have been few attempts to understand the potential impact of parents' health on the SES of children in later life. To address this gap, we use four waves of data from the National Longitudinal Study of Adolescent Health to examine the association between parent's health and the educational attainment of their children. We show that young adults whose parents were in poor health during their middle/high school years are significantly less likely to attend or graduate from college and significantly more likely to drop out of high school compared to those whose parents were in relatively good health. We test several different models to explain this association and we show that much of this association operates indirectly through the health and health behaviors of the child. However, after adjusting for a range of different intervening variables, we continue to show a fairly large and robust influence of parental health on the educational outcomes of their children. These findings partially support the health selection model and they illustrate both the complexity and significance of the health-SES association.

Introduction

The relationship between socioeconomic status (SES) and health is a central focus of epidemiology, public health, sociology, psychology, and demography. This large and diverse body of work has yielded consistent evidence that various aspects of SES including education, income, wealth, and occupational/employment status are all strongly associated with physical health and mortality (Rogers, Hummer, and Nam 2000; Montez and Hayward 2011). However there continue to be debates regarding the most appropriate operationalization of SES (Braveman et al. 2010), the functional form of the SES-health association (Adler et al. 1999), the level at which these associations are structured (Diez-Roux 2001), and the causal nature of this observed association (Smith 1999). To date, the bulk of this research has examined these associations within single generations; the health of an individual (whether cross sectional or longitudinal) is assessed as a function of their current or lifecourse socioeconomic position (Link and Phelan 1995; Hayward and Gorman 2004). Very little work has examined intergenerational processes by which health and SES are *transferred simultaneously*. More specifically, virtually no work has assessed the extent to which having an ill parent may compromise one's socioeconomic attainment in young adulthood. And no work to our knowledge has identified social, behavioral, or biological mechanisms that may account for this association. This paper is unique in its attempt to provide some evidence regarding these complex associations.

SES and Health

The existence of health disparities by SES has persisted into the 21st century despite universal social safety nets such as Medicaid and Medicare; those with higher education, income, wealth, and occupational status tend to enjoy healthier lives (Cutler & Lleras-Mavey 2008; Marmot 2004). Although this literature is quite large, there are two major competing explanations for the observed associations between SES and health. First, it is possible that SES *causes* health to improve

or decline. Mirowsky and Ross (2003) contend that different dimensions of education (e.g. problem solving skills, self-efficacy) coalesce to bring about a kind of “learned effectiveness” that promotes healthy lifestyle choices. This is supported by recent research by Stringhini et al. (2010) who show that the association between SES (measured as occupational status) and mortality is largely due to health behavior differences across the groups (see Jha et al. 2006 for similar results). This perspective is in line with the fundamental causes theory (Link and Phelan 1995) which emphasizes the increased rate at which more affluent persons adopt relatively healthy lifestyles in light of newer evidence that suggests adverse effects of certain behaviors.

Second, it is possible that SES does not cause health, rather health causes SES. The *health selection* model (Smith 1999) posits that individuals who struggle with health problems throughout their lives are less likely to acquire the requisite human capital necessary to acquire jobs with high income or social status (Haas 2006; Palloni 2009). Similarly, a health shock such as a sudden disability or major illness may occur in adulthood and affect earnings capacity while at the same time become a substantial and potentially insurmountable expense (Smith 2004). Currie and Stabile (2003) demonstrate that children from lower SES families experience a greater number of and more frequent health-shocks compared to other children. According to these authors, these health shocks interfere with the attainment of on-time academic goals and significantly reduced math and reading scores. Similar results are also reported by Case et al. (2005) who show that members of the 1958 British birth cohort who experienced poor health as children, were significantly more likely to work less, have lower occupational status, and passed less important qualifying exams. Importantly, these associations remained despite controlling for family background SES. Smith (2009) has produced comparable findings using data from the PSID.

Parent’s health and their children’s socioeconomic attainment

The causation and selection SES-Health models tend to examine the economic resources available to an individual at different points in their lives and the bulk of the debates focus on the recursive and non-recursive nature of these relationships. An understudied distinction in this general conceptual model is the possibility that the health complications for members of the first generation (G1) have adverse effects the SES of their children (G2). This denotes a model akin to the health selection perspective but it is potentially more important because it anticipates the intergenerational transmission of both health and SES as well as a direct influence of parental health on the SES of their children. To our knowledge only one existing study has examined similar question empirically and none have explicitly looked at educational attainment. Andrew and Ruel (2010) use data from the Wisconsin Longitudinal Study to examine the influence of parental health shocks on the *inter vivos* (as opposed to inheritance) transfer of wealth. They show that parents who experience a health shock transfer significantly *more* wealth to their children while they are still living compared to those who do not experience this same shock. This association was only among those with incomes below the median but it still makes it clear that health needs to be considered as an important component of the hypothesized G1 health- G2 SES association.

In this paper, we explore several possible mechanisms that may link parental health to the educational attainment of their children. Parental health may indirectly influence their children's SES through observable factors such as G1 SES, G2 health, the intergenerational transmission of health behaviors, and the health-related capacity for parents to provide support to children through shared activities. Given the preponderance of evidence that SES and health are positively related, G1 SES, which represents the SES of both the parent and the adolescent during the time of the child's adolescence, should positively impact the health of the adolescent. Because childhood health has been shown to be positively related to adult SES (Smith 2009; Haas 2006), the indirect model predicts that G1 SES and adolescent self-rated health should attenuate the relationship between G1

health and G2 SES. G1 health may also affect G2 SES through shared health behaviors. If parents have poor nutritional habits and lead a sedentary lifestyle or smoke regularly then these factors will influence their health and their children may model these behaviors because of informal socialization (Presson et al. 1986). The inverse relationship between unhealthy behaviors and SES has long been demonstrated (Pampel 2010). Thus the indirect model predicts that G1 unhealthy behaviors will have a negative association with SES and attenuate the G1 health – G2 SES relationship.

It is also possible that health complications may influence the capacity of parents to be involved with their children on a regular basis. Research suggests that children who had parents that were involved with their school and social lives tend to have better educational and economic outcomes as adults (Auerbach 1989). Unhealthy parents may be less able or have less time to dedicate to shared activities with their children such as helping with homework, discussing school issues, or even going shopping (Stein et al. 1999). As such, children will have less time to dedicate to school-related activities such as homework compared to their peers with relatively healthy parents. If this process is cumulative in nature, then relatively small influences may become large enough to compromise the probability that a child will attain educational skills that lead to timely graduation, success in school, and ultimately transitions to post-secondary education. As skilled labor is critical to securing stable and rewarding employment, what may seem like a nascent parental health condition may have lasting influences on the economic well-being of their children. If part of the observed relationship between G1 health and G2 SES is due to factor, then we expect to see this relationship attenuated when time in shared activities is included as an intervening factor.

Finally, parental health may continue to influence the educational attainment of their children above and beyond adjustments for these mediating mechanisms. For example, parents with chronically poor health may be spend a greater share of their disposable income on health care and, as such may be less likely to accrue savings over several years (French 2005). Families with fewer

savings may be less likely to send their children to college because of financial limitations that are linked to the health of one or both parents.

In this paper, we use data from a large and nationally representative sample of US adolescents to examine the influence of their parent's during their adolescence on their subsequent educational attainment. The methods, measures, and models for these analyses are described below.

METHODS

Data

All data in this analysis are drawn from the National Longitudinal Study of Adolescent Health (Add Health), a study that examines health and health-related behaviors among a nationally representative sample of adolescents who were initially in the seventh through twelfth grades. In 1994, roughly 90,000 adolescents from 134 schools completed questionnaires about their daily activities, health-related behaviors, and basic social and demographic characteristics. A subset of respondents and their parents were then followed up with for in-home interviews (Waves I-IV) with more detailed questions across a number of important domains. Wave IV was conducted in 2008 and early 2009 when the sampled individuals were in early adulthood. The data for this analysis is limited to those adolescents whose parents responded to the parental questionnaire and provided valid responses to questions related to socioeconomic status and health. Of the 15,702 young adult respondents remaining in Wave IV, 2,141 either did not have a parent questionnaire completed in Wave 1 or did not have valid data for parent self-rated health. From the remaining 13,560 cases, nearly 2,000 did not have complete non-missing data for every variable in the analysis. After listwise deletion, our final sample consists of 11,829 adults who are, on average, 28 years old.

Measures

Self-rated health. For the parental self-rated health measure, parents were asked, "In general, how is your health?" If the parents answered excellent, very good, or good, they are given a value of

1 to signify “good” parental health. If they answered fair or poor they are assigned a value of 0. The respondents of Add Health were asked the same question and were coded in an ordinal manner from 1 to 5 with 1 being poor and 5 being excellent. While self-rated health tends to capture more of a world view than simply health-related symptoms, self-rated health is an adequate and often used concept to represent health (see Idler [2006] and Boardman [2004]).

Socioeconomic status. Wave I socioeconomic status is a composite of three census tract variables (proportion of tract age 25+ without high school diploma or equivalent; proportion of tract age 15+ with at least a college degree; and median household income), household income, and highest education level of a parent ($\alpha=.81$).

Health behaviors. In this study, measures of health behaviors are somewhat limited by the availability of data on the health behaviors of parents. Perhaps the most influential behavior on health is smoking, and fortunately, data are available on smoking habits for both parents and children. With respect to diet and exercise, the best measure available is obesity for both parents and children. Both measures of regular smoking and obesity are operationalized as dummy variables. In Wave I, parents who answered yes to the question, “Do you a smoke?” and adolescents who said they had smoked regularly (every day for 30 days) were labeled “smokers”. If the biological mother or father was identified as “obese” by the parental respondent and lived in the household then the adolescent is considered to have had an obese parent. For the adolescents, BMI in Wave I is calculated from self-reported heights and weights and if BMI was 30 or greater, the individual is identified as obese. In Wave IV, respondents are identified as regular smokers if they reported smoking every day for the previous 30 days and obesity was assessed by measured height and weight using the same BMI (e.g. >30) threshold.

Shared activities. In Wave I, respondents were asked whether their resident parental figure participated in various activities with them in the previous four weeks. The possibilities included

shopping, playing a sport, attending a religious service, talking about dating or parties, attending events, talking about serious problems, talking about grades, working on a project for school, talking about other school issues. We define parental involvement as the sum of the number of items answered in the affirmative by the respondent ($\alpha = .95$).

Educational attainment. This variable was measured in Wave IV and captures the highest level of education to date for the young adult. Respondents were placed into one of four mutually exclusive categories: high school dropout, high school graduate only, some college, and college graduate and higher.

Control variables. Several variables were drawn from Wave IV and used as control variables in different areas of the analysis. These control variables include age, race (Non-Hispanic White, Non-Hispanic Black, Hispanic, Asian American, and other), marital status (married, not married), and number of children.

Analyses

Our primary aim is to test the relationship between G1 self-rated health and G2 educational attainment. Our secondary aim is to identify mechanisms that may explain this relationship. We use multinomial logistic regression to gauge the influence of parental health on the likelihood of achieving various levels of education (less than high school, high school graduate, some college, and college graduate). After establishing a main effect, we successively add blocks of variables that represent the intervening factors that are hypothesized to mediate the initial direct relationship. All data are weighted to reflect the complex sampling design of the Add Health study (Chantala and Tabor, 1999).

RESULTS

[Table 1 about here]

Table 1 presents descriptive statistics for the entire sample and by health status of the adolescents' parents in Wave I. Eighty-six percent (95% CI: [.84, .88]) of parents rated themselves as being in good health (excellent, very good, or good). Importantly, we show the parental health status is strongly and positively associated with educational attainment of children by Wave IV of the study. Whereas only 7.8% of the young adults of healthy parents dropped out of high school, 17% of the children of unhealthy parents had not completed high school by their late 20s. Similarly, children of healthy parents (thirty-three percent) were more than twice as likely as those with unhealthy parents (fifteen percent) to complete college by their mid to late 20s. Importantly, healthy parents also report participating in more activities with their adolescence than less healthy parents. Over 31 percent of the adolescents had at least one parent in the household who smoked and about 20 percent had at least one parent in the household who was obese and both factors were strongly associated with parental self-rated health.

[Table 2 about here]

To determine to extent to which the observed relationship between G1 health and G2 educational attainment is mediated by adolescent health, G2 health behaviors, shared activities, and adult characteristics (health and health behaviors), we ran sequential multinomial logistic regressions on educational attainment. The results are presented in Tables 2 and 3. The reference educational attainment category is high-school graduate, and the comparison groups are less than high-school (LT HS), some college (SC), and at least a college graduate (CG). The cell entries represent odds-ratios (compared to high school graduation). Without controlling for possible mediating factors, parent health has a very strong and positive relationship with educational attainment. Having a parent in good health, reduced the risk of dropping out of high-school by nearly 40%, increased the relative risk of having some college compared to high school only by 30%. Importantly, those with healthy parents in Wave 1 were nearly 2.7 times more likely to graduate from college by Wave 4

compared to those with unhealthy parents. These associations are all above and beyond demographic controls for age, race, and gender. These estimates (Model 1) denote the baseline risk estimates and subsequent changes in the coefficients are linked to the statistical controls entered into each model. Parental SES at Wave 1 is entered first. As shown, the G1 health – G2 SES association for some college is explained by Wave 1 SES and nearly one-half of the college graduate association is attenuated with this control. Model 3 enters controls for the adolescent’s self-rated health at Wave 1 which is one of the primary independent pathways through which this association should be operating. Healthy parents are more likely to have healthy children and their health may predict their likelihood of matriculating through various school programs on time. The associations operate in the expected direction and this control further attenuates the main effect on college graduation by an additional fourteen percent. Model 4 adjusts for parental health behaviors at Wave 1 and smoking status is strongly linked to educational attainment and this control further reduces the link between parental health and the educational attainment of their children but we continue to observe statistically significant associations for both highschool dropout and for college graduation.

Table 3 continues this line of inquiry and further adjusts for shared activities, self-rated health of the respondent in Wave 4, and smoking Wave 4, and obesity Wave 4. These controls are all strongly associated with educational attainment however none of these mechanisms completely explains the association between parental health and the educational attainment of their children. In total, we observe a direct effect for both high school dropout and college graduation. Adolescents are nearly 30% less likely to drop out of high school compared to graduating high school only if their parent is in good health at Wave 1. Similarly, adolescents are over 40% more likely to graduate from college compared to have only a high school degree if their parent is in good health at Wave 1.

[Table 3 about here]

DISCUSSION

Parental health has a direct influence on children's educational advancement, which remains substantial after controlling potential confounding and mediating influences. A major potential confounder is parental socioeconomic status. To the extent that poor parental health is concentrated in the lower social strata, children of unhealthy parents may advance less far in school because of their lower socioeconomic background more so than the influence of poor parental health *per se*. Taking this possibility into account, we find that parental socioeconomic status reduced the association of parental health and children's educational attainment, but the association remained substantial. In the broader theoretical debate of whether poor health leads to lower SES (the 'selection' hypothesis) or vice-versa (the 'causation' hypothesis), these study results provide unique support for an understudied aspect of selection. Poor health seems to impair the educational attainment of individuals (a key component of SES), albeit the poor health of individuals' *parents*. To the extent that poor health is transmitted across a lineage, it would be expected that the influence of poor health on the educational attainment on each successive generation would cumulate over time. Such a process would lead to a substantial concentration of poor health in the lower socioeconomic strata, a process would be exceedingly difficult to detect with standard, intragenerational analyses.

The analysis also identified indirect, mediating factors that partially explain how parental health exerts an influence on children's educational attainment. One mechanism through which parental health has an indirect influence on children's education is through health behaviors. Healthy parents have healthy children, and healthy adolescents are more likely to complete a college education because of factors such as fewer school absences and less impaired cognitive development (Haas 2006). An additional indirect mechanism at work is the number of shared activities between parents and their children. As expected, healthy as compared to unhealthy parents engage in more

activities with their children, such as homework and school issues. Taking into account these mediators reduced somewhat the association of parental health and children's educational attainment, but, again, the association remained substantial. Specifically, after taking into account major confounders and mediators children with healthy as compared to unhealthy parents were 36% more likely to go to college and 39% more likely to advance beyond a high school degree.

What accounts for this influence of parental health on children's educational advancement? A least three factors warrant future research. On the one hand, an unhealthy parent may reduce the financial resources available to an adolescent to pursue a college degree. Given two families at the same socioeconomic level, the one with an unhealthy parent will likely have less money to send a child to college. In light of the fact that family income is a major determinant of college attendance and college persistence (Turley, Santos, and Ceja 2006), it is likely that family finances may explain much of the association between parental health and children's educational attainment. Social support may also play a role, both in terms of the social support provided by parents and caregiving required by an unhealthy parent. Social support and encouragement provided by parents is required not only for a child to attend college but also for the child to see it through and earn a degree. Analysis of peer, teacher, and parent influence on adolescent's decisions to continue in college indicate that it is the parents who are most influential (Bank, Slavings, and Biddle 1990), and the college persistence literature points to parental support and involvement as a key factor in children's college success (reviewed in Seymour and Hewitt 1997). A negative impact of poor health on parental support of their children's college efforts would partly explain the influence of parental health on children's educational attainment. In addition, caregiving responsibilities may also play a role. An unhealthy parent places children at risk of taking on the role of parental caregiver, a role that for youth is stressful and associated with increased levels of anxiety and depression (Armistead, Klein, and Forehand 1995; Worsham, Compas, Ey 1997; Pederson and Reverson 2005). Such

additional stressors may have both a direct influence on college degree attainment, and may also, as well, exacerbate other risk factors for lower educational attainment.

Finally, it is also possible that the association between SES and health is due to an unobserved mechanism that causes both SES and health. For instance, low intelligence could prevent individuals from learning about or implementing healthy lifestyle choices and could also prevent individuals from succeeding economically (Fuchs 1982). Link et al. (2008) addressed this concern by adjusting for differences in cognitive ability and still provide evidence supporting the fundamental cause theory. Because the source of the covariance between health and SES may be genetically oriented, researchers have used twin and sibling studies to examine the extent to which genetic factors associated with physical health are the same as those related to SES. With regards to this study, there is consistent evidence that genetic factors influence physical health (Johnson and Krueger 2005), health related behaviors (Boardman 2009), and SES (Nielsen 2006). Each phenotype has evidenced heritability estimates that range from .4-.7 suggesting that a large proportion of the total variance is due to additive genetic factors. Because these factors are highly intercorrelated and because each is highly heritable, some have tested the possibility that the genes linked to one phenotype are common to all; that is their association in the population may be due to a common genetic source rather than one causing the other. In one of the largest studies done on this issue, Silventoinen et al. (2004: 544) use two large twin registries in Minnesota and Finland and show that the correlation between body height and educational attainment is “overwhelmingly due to the correlation of the shared *environmental* factors affecting these two traits.” (emphasis added).

In ancillary analyses (results presented in Appendices 1 and 2), we estimated a Bivariate Cholesky¹ model (similar technique used in the Silvertainen et al. (2004) paper) for educational attainment and self-rated health using data from the sibling and twin pairs sample of the Add Health study. If the transmission of the SES-health association is due to a common genetic source then a

within generation association should provide an *upper-bound* for the estimate of genetic influence. We find that both traits are only moderately heritable ($h^2_{\text{srh}} = .28$; $h^2_{\text{educ}} = .12$) and we show a fairly modest correlation between the two traits ($r = .23$). According to our results, roughly 45% of the observed correlation between self-rated health and educational attainment within this generation may be due to common genetic influences. If this is taken as a maximum value then it is fair to conclude that the bulk of the intergenerational association between parental health and children's educational attainment is independent of common genetic influences. Further, evidence for a common genetic source does not necessarily mean that genetic factors that *cause* health are also those that *cause* SES rather it suggests that subtle genetic influences may place persons within contexts that make them more vulnerable to educational limits and poor health. This is critical because it does not challenge the importance of the social factors that link health and SES it simply provides another perspective on one additional source of this association. Thus, in some ways, the social environment remains fundamental because it provides the context in which this form of selection actually materializes. Absent this existing social environment in which health and SES are generally comorbid, it is unclear if the genetic etiology of this comorbidity would remain as large.

In terms of policy these results indicate support for the notion that parental health should be included in the assessment of individual progress within educational settings. Thus, although individualized assessments are generally limited to students with diagnosed learning disabilities, parental health information could be obtained at enrollment time that may provide teachers and administrators with a more nuanced understanding of all student's home lives. Our results also suggest that improving the health of parents may also have important educational returns for their children. As such, one spillover of policies to improve the health of the population is the very real possibility that educational attainment of the population will increase as well. This is particularly

important because large efforts to decrease health disparities may go a long way towards reducing educational disparities by race, ethnicity, and class (Farkas 2003).

Who gets ahead in the U.S. has long been of central interest to sociologists (Jencks 1980). While ambition and hard work certainly play a key role in socioeconomic advancement, so too do factors outside of the individual's control, such as parent's education and income. The results of this study indicate that parental health also independently affects individual status attainment, an influence that has received little recognition to date. We encourage researchers to further explore this association in an effort to identify the social, behavioral, or even biological mechanisms that may support this association. Further, it is likely that this association varies considerably by race and gender. For example, does maternal health have the same influence on the educational attainment of boys and girls from the same family? This would be an important extension of the health disparities literature and the results from this paper provide a useful point of departure for future studies in this important area of research.

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Table 1. Descriptive Statistics by Parental Health Status

	Total	Healthy	Unhealthy	Pr. <
Parent Variables (Wave I Data)				
At least one parent smokes (=1)	0.311	0.294	0.418	.001
At least one parent obese (=1)	0.195	0.182	0.278	.001
Shared activities (0-9)	3.880	3.916	3.600	.001
Adolescent Variables (Wave I Data)				
Self-rated health (1-5)	3.887	3.922	3.665	.001
Regular smoker (=1)	0.217	0.214	0.242	.080
Obese (=1)	0.089	0.080	0.149	.001
Young Adult Variables (Wave IV Data)				
Health and health behaviors				
Self-rated health	3.657	3.693	3.453	.000
Regular smoker (=1)	0.260	0.255	0.295	.058
Obese (=1)	0.358	0.343	0.451	.000
Education				
Less than high school	0.090	0.078	0.170	.000
High school degree only	0.177	0.165	0.257	.000
Some college	0.432	0.431	0.438	.703
College degree or more	0.306	0.330	0.154	.000
Control Variables				
Age (Wave IV)	28.200	28.202	28.416	.032
Socioeconomic status (Wave I)	-0.012	0.052	-0.417	.000
Race and ethnicity				
Non-Hispanic White	0.698	0.720	0.562	.000
Non-Hispanic Black	0.144	0.134	0.205	.000
Hispanic	0.114	0.104	0.179	.001
Other race	0.017	0.014	0.031	.175
Family characteristics				
Married (=1)	0.395	0.395	0.394	.480
Number of children	0.842	0.809	1.052	.000
Number of observations	13560	11632	1928	

Table 2. Multinomial logistic regression estimates: the effect of healthy parental status on educational attainment of their children.

	MODEL 1			MODEL 2			MODEL 3			MODEL 4		
	LT HS	SC	CG	LT HS	SC	CG	LT HS	SC	CG	LT HS	SC	CG
Par. Health (W1)	0.62***	1.29**	2.68***	0.69**	1.06	1.66***	0.70**	1.04	1.56***	0.72*	1.05	1.45**
Parent SES (W1)				0.64***	1.94***	4.48***	0.64***	1.92***	4.35***	0.67***	1.87***	4.03***
Ad. Health (W1)							0.94	1.09*	1.51***	0.95	1.09*	1.48***
Parent Smokes (W1)										1.73***	0.78***	0.48***
Parent Obese (W1)										1.05	1.20*	0.89
Log pseudo likelihood	-15344.42			-14424.12			-14306.66			-14151.133		

LT HS = less than high school; SC = some college; CG = at least college degree. The comparison group is high school graduation only.

Control variables include male (=1), age, non-Hispanic Black (=1), Hispanic (=1), other race (=1)

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

Table 3. Multinomial logistic regression estimates: the effect of healthy parental status on educational attainment of their children.

	MODEL 5			MODEL 6			MODEL 7		
	LT HS	SC	CG	LT HS	SC	CG	LT HS	SC	CG
Parent in Good Health (W1)	0.73*	1.04	1.42**	0.73*	1.03	1.38**	0.72*	1.03	1.36*
Parent SES (W1)	0.67***	1.84***	3.94***	0.68***	1.84***	3.88***	0.66***	1.84***	3.75***
Adolescent SR Health (W1)	0.95	1.08*	1.46***	1.01	1.06	1.30***	1.03	1.05	1.25***
Parent Smokes (W1)	1.73***	0.78***	0.48***	1.70***	0.79***	0.50***	1.61***	0.80**	0.57***
Parent Obese (W1)	1.07	1.18*	0.87	1.05	1.19*	0.91	1.14	1.17	0.94
Shared Activities (W1)	0.95	1.06***	1.15***	0.95	1.06***	1.15***	0.95	1.06**	1.15***
Young Adult SR Health (W4)				0.80***	1.10*	1.60***	0.79***	1.10*	1.44***
Young Adult Smokes (W4)							1.84***	0.79**	0.22***
Young Adult Obese (W4)							0.77*	1.04	0.74**
log pseudo likelihood		-14077.90			-13911.54			-13562.90	

LT HS = less than high school; SC = some college; CG = at least college degree. The comparison group is high school graduation only.

Control variables include male (=1), age, non-Hispanic Black (=1), Hispanic (=1), other race (=1)

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

NOTES

1. Factor loadings for the Bivariate Cholesky model are estimated by maximum likelihood methods using the OpenMx package for R 13.3 (Boker et al., in press) and are presented in Appendix 1. This model builds off of the various structural equation models available in *Mx* (Neale, 1993). The parameter tests from these models provide the following information: (1) the best fitting model indicates whether genetic or environmental influences or both are important sources of variation for each trait; (2) the models decompose environmental influences into shared and nonshared sources and indicate whether both sources are needed to fully characterize environmental influences; and (3) most importantly, they describe the extent to which the observed covariation between the two traits is due to environmental or genetic covariation. Parameter estimates are derived from the cross-twin cross-trait correlations provided in Appendix 2. The path coefficients described in Appendix 1, in combination with information about the variance of each trait and their respective covariances can be used to identify the source of the variation that is unique to each trait and that which is shared. This information is used to calculate the genetic correlation coefficient (r_g), which is given as

$\frac{a_{21}a_{11}}{\sqrt{a_{11}^2(a_{21}^2 + a_{22}^2)}}$. The product of this the genetic correlation coefficient and the square root of each

univariate heritability ($\sqrt{h_x^2} * r_g * \sqrt{h_y^2}$) provides a value that when compared to the overall phenotypic correlation, provides the proportion of the phenotypic correlation that is due to shared genetic influences. This is a fairly conservative approach and it will provide an estimate of the maximum extent to which genes affect the covariation between the traits. Specifically, the additive genetic variance contains gene-environment interaction effects which may be affecting both sources of variance. However, the remaining correlation in this model is due to exclusively environmental influences. The genetic correlation coefficient ($r_g = .55$)

$$\left(\frac{a_{21}a_{11}}{\sqrt{a_{11}^2(a_{21}^2 + a_{22}^2)}} = \frac{(.45)(.49)}{\sqrt{.49^2(.45^2 + .68^2)}} = .55 \right) \text{ suggests a common genetic etiology in the}$$

association between education and self-rated health among young adults. When structured as a function of the two heritabilities the genetic correlation suggests that roughly 45% of the correlation

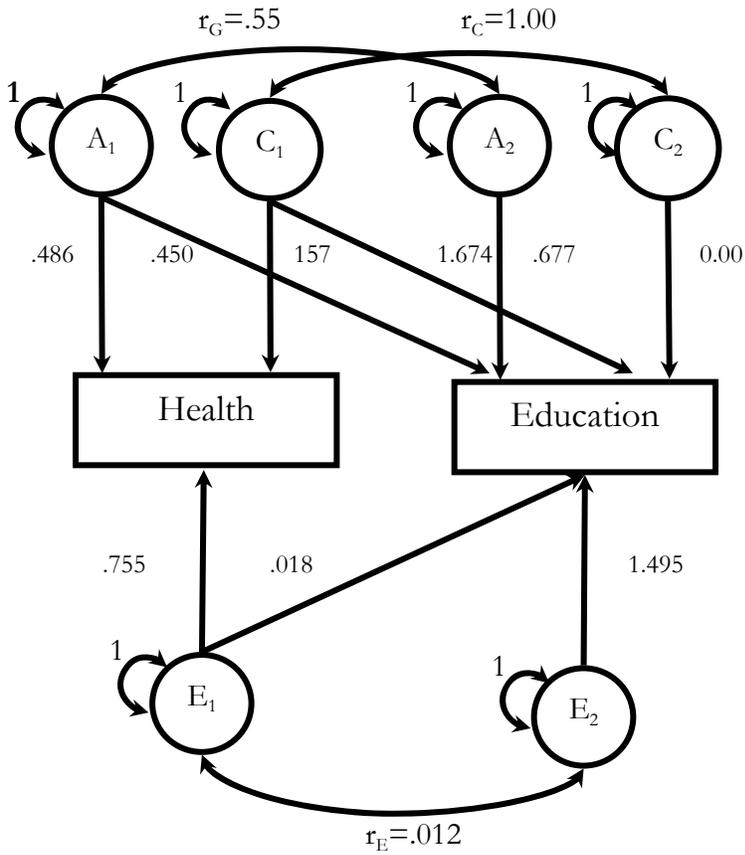
between health and education is due to common genetic influences; $\frac{\sqrt{h_x^2} * r_g * \sqrt{h_y^2}}{r_{x,y}}$ describes the

proportion of phenotypic correlation that is due to common genetic influences. This estimate

$$\left(\frac{\sqrt{.28} * .55 * \sqrt{.12}}{.23} \right) = .45 \text{ suggests that 45\% of the total correlation between health and education (r}$$

= .23) is due to common genetic influences.

Appendix 1. Bivariate Cholesky decomposition estimates: genetic and environmental influences on the covariance between education and health.



Appendix 2. Cross-twin, cross-trait correlation matrix: education and self-rated health at Wave IV

Identical (MZ) twins	Health 1	Education 1	Health 2	Education 2
Health 1	1.000			
Education 1	0.287	1.000		
Health 2	0.369	0.285	1.000	
Education 2	0.220	0.623	0.259	1.000

Fraternal (DZ) twins	Health 1	Education 1	Health 2	Education 2
Health 1	1.000			
Education 1	0.194	1.000		
Health 2	0.113	0.171	1.000	
Education 2	0.143	0.544	0.211	1.000

Note: Data obtained from the twin sample of the Add Health study.