College Completion and Adult Health

Childhood (Mis)fortune, Educational Attainment, and Adult Health: Contingent Benefits of a College Degree?

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College-educated adults are healthier than other people in the United States, but selection bias complicates our understanding of how education influences health. This article focuses on the possibility that the health benefits of college may vary according to childhood (mis)fortune and people's propensity to attain a college degree in the first place. Several perspectives from life course sociology offer competing hypotheses as to whether the most or the least advantaged see the greatest return of a college education. The authors use a national survey of middle-age American adults to assess risk of two cardiovascular health problems and mortality. Results from propensity score and hierarchical regression analysis indicate that the protective effect of college attainment is indeed heterogeneous. Further, the greatest returns are among those least likely to experience this life course transition (i.e., compensatory leveling). Explanations for this selection effect are offered, along with several directions for future research on the health benefits of completing college.

College completion is an important milestone in the life course of many American adults. In the last century, the expansion of higher education has coincided with a long-standing interest in how college education shapes occupational attainment, as well as how the achievement of a college degree influences life chances relative to more ascriptive statuses (Hout 2012). Extending this area of inquiry, life course sociologists and demographers have documented the health and social advantages conferred by college completion among adults in middle and later life (Dupre 2007; Mirowsky and Ross 2003). College-educated adults fare better than those with lower levels of education on a broad range of outcomes: These include mental health, sense of personal control, self-rated

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health, disability, chronic conditions and mortality (Hummer and Lariscy 2011; Mirowsky and Ross 2003; Schnittker 2004).

A plausible interpretation of these findings is that college attainment leverages a diffuse set of health-related social advantages. As Mirowsky and Ross (2003) maintain, "Education enables people to coalesce health-producing behaviors into a coherent lifestyle that improves health."(52) Prior findings indicate that although education has indirect benefits for health through socioeconomic status (SES), health behavior and social psychological pathways, it also maintains a direct protective effect over the life course—an effect irreducible to any specific causal mechanism (Hummer and Lariscy 2011; Ross and Wu 1995). There lurks, however, a potential complication within this line of thought. As life course theory has elucidated, transitions such as college degree completion shape future life trajectories but are also influenced by antecedent conditions (Elder, Johnson and Crosnoe 2003). The endogeneity of these life course pathways implies that the health benefits provided by a college degree may be contingent upon the very factors that shaped college attainment in the first place.

Specifically, early life course factors lead to differential selection into key social institutions such as higher education. Low household SES, limited parental education, disorganization in family structure, traumatic experiences and health problems can stifle educational attainment (Duncan et al. 1998; Sandefur and Wells 1999). That being said, careful observational studies that include childhood measures still report a robust effect of education on adult health and mortality (Hayward and Gorman 2004; Link et al. 2008). This suggests that despite the nonrandomness of educational pathways, selection bias does not obscure an actual effect that educational attainment holds for health. This conclusion is also largely upheld by U.S. econometric research using quasi-experimental instrumental variables (e.g., variation in compulsory education laws) and fixed-effects twin models (Lleras-Muney 2005; Lundborg 2008, but see also Eide and Showalter 2011).

Nevertheless, an important second form of heterogeneity remains: People with different propensities to receive a college education may see different returns on their college degree. This is referred to as the *heterogeneity of treatment effects* and represents the focus of our study. Different perspectives in life course sociology imply opposing expectations in this regard—*does the benefit of education enhance the health advantage of the most privileged, or does it serve as a compensatory mechanism for the least advantaged?* An emerging body of literature has begun to address the selection processes that link childhood (mis)fortune, education and health (Haas 2006; Palloni 2006), but the issue of who gains the most from a college degree—treatment heterogeneity—has not yet received widespread and systematic attention.

Brand and Xie (2010) recently undertook a careful examination of the heterogeneous treatment effect issue in an innovative study of the income return of a college degree. The authors articulated two competing hypotheses: *positive selection* and *negative selection*. The former assumes that the advantage of a college degree is most pronounced among those whose background characteristics predict a high probability of college completion. Negative selection, on the

other hand, implies that the benefits of a degree are smallest among those most likely to graduate from college. To adjudicate between these rival hypotheses, the authors developed an innovative technique to parsimoniously evaluate the returns to education across a range of degree-completion propensity.

This article adopts Brand and Xie's (2010) heterogeneous treatment effect (HTE) approach to assess the relationship between college attainment and health. In the absence of experimental data, definitive conclusions about education's causal influence on adult health are hard to establish. Nevertheless, counterfactual approaches to causal analysis (e.g., propensity scores) can be useful in asking whether the benefits of education—whether strictly causal or not—accrue differently according to early life course conditions. Using national data from the Midlife Development in the United States (MIDUS) study, we consider two self-reported outcomes related to cardiovascular health: hypertension and general heart problems. Cardiovascular conditions are relevant as health outcomes because they represent the leading causes of death in the United States for men and women. In addition, we make use of recent data linking MIDUS to the National Death Index (CDC/National Center for Health Statistics 2012), allowing us to observe whether the college advantage for mortality is conditional on the propensity to attain a college degree.

Health, Education and Causality: The Problem of Selection Bias

Much of the sociological literature on education and health comprises population-based surveys, an understandable scenario given the rare circumstances permitting a natural experiment approach. As past observers have noted, social scientists using observational data to assess the causal role of a "treatment" (e.g., college completion) must confront two forms of selection bias (Morgan and Winship 2007). The first is *heterogeneity in preexisting conditions*, meaning that people select into treatment effects on the basis of temporally prior traits, events or statuses. This form of selection bias complicates the task of explicating causal processes because the preexisting conditions may be the underlying cause of both treatment and outcome. Returning to the example of college attainment and health, the widespread assumption that higher education protects against disease and mortality potentially confounds two plausible explanations for the association: (1 the social background or personality factors that predispose a person to attain a college degree in the first place and (2 any actual benefit of a college degree.

Granting that people are nonrandomly selected into "treatments" such as college, a second form of selection bias complicates matters further. *Heterogeneity of treatment effects* means that the effect of a particular treatment differs across a population. Earlier studies on educational gradients and health make an implicit assumption that the benefits of educational attainment are equal for individuals who differ greatly in social background (e.g., Dupre 2007; Ross and Wu 1995). Sometimes subsample analyses are used to assess differential education-based returns on health according to race or gender (e.g., Farmer and Ferraro 2005),

but this approach does not acknowledge variability in the matrix of factors that predict college completion and are spread across these demographic groups. Similar to Brand and Xie's (2010) recent study on the wage returns of a college degree, we will address heterogeneity in preexisting conditions by using a propensity score strategy and turn the majority of our attention to this second form of selection bias—the *heterogeneous treatment effects of a college degree*. By first identifying people according to their propensity to obtain a degree, we can observe whether the highest returns from college are among those most likely to have completed a college education versus those least likely to complete college on the basis of advantage or disadvantage early in the life course. Rather than viewing heterogeneous treatment effects as a nuisance to the endeavor of causal inquiry, we see the issue as an apt opportunity for testing competing hypotheses drawn from life course scholarship.

Added Protection

The first hypothesis, what we call *added protection*, suggests that those of greater early advantage would see the biggest health boost associated with a college degree. Studies of health, for instance, often reference the so-called "Matthew Effect" coined by Robert Merton (1968). In this model, early advantage increases opportunity for future advantage, thereby accentuating early variation and generating widespread inequality in the health of a cohort over time (Dannefer 2003). A cumulative advantage perspective would presume, then, that college would be most protective of the ones with the greatest propensity to complete college if these individuals are better equipped to capitalize on the advantages that educational attainment can provide. This is plausible under several scenarios. If the most advantaged people are most prepared for college (academically, socially or financially), they may reap a disproportionate share of education's rewards. Also, if lifestyle preferences and basic dispositions (often described as *habitus*) of the most advantaged tend to emphasize the pursuit of (a educational attainment and (b good health, it follows that each of these factors may reinforce the other (cf., Freese and Lutfey 2011:73-4). A host of compelling class-based cultural factors influence whether college is an expected life course event, and the same normative influences that emphasize educational attainment and social mobility also likely inform and underlie people's lifelong health choices and behaviors (Cockerham 2005).

Supplementing the cumulative advantage perspective, social epidemiologists have articulated a life course "critical period" model in which early events define crucial pathways of disease risk (Kuh and Ben-Shlomo 2004). In its strictest sense, this view follows the fetal origins thesis, which maintains that in-utero conditions have an enduring lifelong influence on cardiovascular and metabolic health (Barker 1994). Applying the idea of a "critical period" to the present study would suggest that people hampered by early disadvantage, such as family instability, experience of trauma or abuse or low socioeconomic standing start on an adverse life course trajectory and may be less capable of offsetting these initial insults. Preston, Hill and Drevenstedt (1998) refer to this process as

scarring if the effect is direct—stress exposure produces indelible physiological insults—and as *correlated environments* when disadvantage indirectly leads to health problems in later life via adulthood conditions. In all, the *added protection hypothesis* anticipates that those who are best poised to receive health benefits from education are those with the most auspicious start in life.

Compensatory Leveling

The alternative pattern of heterogeneous treatment effects can be described by a *compensation* process. This hypothesis expects that the health benefits of a college degree would be *least* pronounced among the most advantaged—those with the greatest propensity to obtain a college education. Alternatively, this could be stated as the most disadvantaged stand to gain the most from a college degree.

The attainment of a college degree may serve as a critical life course transition that narrows, rather than exacerbates social inequality. As Ferraro and Kelly-Moore (2003) suggest, "it is important to identify whether there are compensatory mechanisms that can eliminate, or at least reduce, the effects of earlier disadvantages" (p. 710). Compensatory mechanisms may take a variety of forms. For instance, education enhances the subjective sense of personal control, an important predictor of good health across the life course (Mirowsky and Ross 1998). Gains in personal control endowed by higher education may be most important among those with initially lower levels. Furthermore, the social capital generated during one's matriculation through an educational institution may also be more consequential for those whose social network is initially more limited. Taken together, these countervailing mechanisms may help reduce—rather than exacerbate—the health advantage associated with a college degree. Ross and Mirowsky (2010) find some evidence of this pattern—which they identify as *resource substitution*—in a recent study of parental education, personal education and physical impairment.

Another rationale for the *compensatory leveling hypothesis* is that those attaining a college degree against considerable odds may represent a resilient subset of people who are likewise most resistant to health problems. Individuals most likely to attend college, on the other hand, may comprise an aggregate more diverse in their susceptibility to disease and mortality. This is a plausible scenario, given the strong association between parental and child educational attainment (Hout 2012). In essence, a nontrivial proportion of those with privileged childhoods have simply followed the expected course of events in attaining a college degree, whereas those who graduate college despite adverse social origins may represent an especially hardy set of people. In both the "selectivity" scenario and the compensatory mechanism explanation, the health benefit of a college degree should be most clearly discernible for those least likely to attain the degree.

In weighing the added protection hypothesis against the compensatory leveling hypothesis, it is crucial to first demonstrate that people have *unequal propensities* to receive a college education. Indeed, many potential factors increase the likelihood of attaining a college degree, or, alternatively, may stand in the way of obtaining one. For incisive literature reviews on this topic, see Haveman

and Wolfe (1995) and Kao and Thompson (2003). The approach of this article is not to identify particular causal pathways from childhood to a college degree and compare their relative influence. Rather, the goal is to predict an overarching propensity for college completion based on a large vector of observed variables on childhood social conditions and to examine the corresponding implications for health. These variables and their measurement are elaborated upon in the Methods section. An overview of the statistical model that will aid in adjudicating between the competing hypotheses (what Brand and Xie [2010] call positive vs. negative selection) is provided in the Appendix.

Data and Measurement

Evaluating the competing hypotheses proposed in this study requires a dataset of individuals old enough to be considered "at risk" for significant diseases that emerge in adulthood and increase the risk of death. Furthermore, a rich array of variables related to childhood background is needed to adequately construct propensity scores of college completion. We identified the MIDUS study as an ideal data source for examining the heterogeneous treatment associated with a college degree.

The MIDUS study was initiated in 1995 by the MacArthur Foundation's Network on Successful Midlife Development. The survey was composed of two parts. First, the investigators used random-digit-dialing to obtain a sampling frame of all English-speaking, noninstitutionalized adults aged 25-74 years in the contiguous 48 states, oversampling older males. The response rate from these initial telephone interviews was 70 percent. Second, the survey included a two-part follow-up questionnaire mailed to those who participated in the telephone interview, yielding an 86.6 percent response rate. Thus, the overall response rate was 61 percent (.70 x .87 = .61), producing a total sample of 3,032 participants who completed both the telephone and mail interview. The final sample includes 2,923 respondents with complete data on the predictor variables[1]; modest missing data on the variables for hypertension and heart problems reduces the analytic sample to 2,909 (hypertension outcome) and 2,915 (heart problems outcome). Data for key study variables were obtained from both the phone interviews and the mailed questionnaires. In addition to these modes of data collection, an investigation to track mortality of the initial MIDUS respondents was undertaken during a second wave of data collection. The investigators used the National Death Index (NDI) to obtain the date of death for 245 deceased respondents who completed the first wave of the study. Mortality data are available for deaths that occurred between March 1995 and April 2006.

Health Outcomes

We consider two health conditions that are among, or directly contribute to, the leading causes of death in the United States: hypertension and heart problems. Both of these diseases are commonly analyzed as dependent variables in the study of socioeconomic gradients of adult health and account for 25 percent of all deaths among American men and women.

During the phone interview component of the MIDUS survey, respondents were asked, "Have you ever had heart trouble suspected or confirmed by a doctor?" Hypertension was assessed in the mailed questionnaire in which respondents were asked to indicate whether in the past year they had "experienced or been treated for" a host of health conditions, including "high blood pressure or hypertension." We recognize several limitations in these measures, including the reliance on participant self-reports and the necessity of interaction with medical professionals to obtain a disease diagnosis. Unfortunately, few comprehensive national studies with information on childhood background provide access to objective clinical records concerning cardiovascular health. Objective mortality records, therefore, will serve as an important supplement to the self-reported health measures. Respondents were assigned a binary value for mortality: "1" if NDI records indicated that they died anytime from the outset of the MIDUS study through April 2006 and "0" otherwise.

Childhood Background

We selected a number of variables related to childhood life chances that were measured in the MIDUS survey. The purpose of these childhood background variables was to generate a propensity score for college completion based specifically on advantage/disadvantage from early life context. Accordingly, childhood background variables include factors related to family structure, childhood socioeconomic status, trauma or mistreatment.[2] Numerous studies link low childhood SES (often operationalized by parental education, working conditions, receipt of government aid or overall assessment of household finances) with adult cardiovascular health problems and mortality (e.g., Gruenewald et al. 2012). Studies have also suggested a link between family processes and structure during childhood (e.g., family violence, family disruption) with a broad range of health outcomes and mortality (Felitti et al. 1998; Tucker et al. 1997). In all, we include a wide set of the factors that have been considered in previous studies and that predict both the treatment (college completion) and the outcome (health).

Household stability during childhood is an important predictor of educational attainment (Sandefur and Wells 1999) and is related to health and longevity (Tucker et al. 1997), so we include a dummy variable assessing parental divorce prior to 16 years of age (1 = yes, 0 = no). Trauma during childhood tends to limit education and is detrimental to health (Malinosky-Rummell and Hansen 1993), and so we utilize information adapted from the Conflict Tactics Scale that was incorporated in the MIDUS survey. Respondents were first asked how often either parent had perpetrated multiple forms of physical (e.g., "slapped you") or emotional abuse (e.g., "threatened to hit you."). Response categories included never, rarely, sometimes or often. We collapsed the latter two responses in the "often" category.

As one proxy for childhood SES, we used a MIDUS question that asked: "During your childhood and adolescence, was there ever a period of six months or more when your family was on welfare or ADC" (Aid to Dependent Children). This item was dichotomized as either 1 = yes or 0 = otherwise. Parental

educational attainment is an important dimension of social mobility (Hout 2012) and an additional indicator of childhood SES; accordingly, we generated several categorical variables to assess the highest level of education received by each parent (less than high school; high school; college), as reported by the study respondent. In cases where parents' education level was not known (425 respondents missing on father's education, 207 respondents missing on mother's education), we used a binary variable for missing to retain the respondents within the models. Besides financial hardship and parental education, prestige or other aspects of a parent's occupational life may also be influential in shaping one's likelihood of attending and completing college. We therefore incorporate a dummy variable to indicate the status of each parent's occupational sector (coded as "0" for upper-white-collar and "1" equal to lower-white-collar or blue-collar occupations). Separate indicators were included for mothers and fathers, along with binary variables for missing data (372 respondents missing on father's occupation, 1,542 respondents missing on mother's occupation). In addition, we included a binary variable for whether the father supervised others in the workplace to indicate whether they served in a managerial workplace role (1 = supervised others, 0 = otherwise).

Finally, we include an indicator of childhood health. Early health problems may disrupt educational attainment as well as increase the risk for health problems in later life such as cardiovascular disease. MIDUS respondents were asked to retrospectively evaluate their overall health at 16 years of age (excellent, very good, good, fair, poor), and we collapsed the latter two responses as "poor health."

Demographic Covariates

Several demographic variables were also included in the analysis. Age was coded as a continuous variable, and sex was coded as binary variable (1 = female, 0 = male). In addition, race was treated as a binary variable (1 = nonwhite, 0 = white).

Analysis

The general strategy for our analysis was to differentiate respondents on the basis of their propensity to complete college and to assess whether a college degree is associated with a lower probability of health risks, granting the possibility of heterogeneous treatment effects. Undertaking this analysis proceeded in five steps.

First, we used a binary logit regression with college degree as the dependent variable. Coefficients from this equation are shown in the Appendix (Table A1). Second, predicted probabilities for college completion were generated from the logit model and each respondent was assigned a propensity score. In essence, a single index—the created propensity score—represents the contribution of 21 different terms. Third, after the estimation of property scores, respondents were grouped into balanced propensity score strata on the basis of their means and variances. The balancing property is considered satisfied when the distribution

of the propensity score and each covariate does not differ for the treated and nontreated within each propensity score stratum. Thus, within each stratum, exposure to treatment is considered random. Fourth, we applied the common support restriction to the balancing algorithm to ensure optimal propensity score matching. Using the condition of common support, only those propensity score regions that contain both treated and nontreated cases are included in the analysis.

After respondents were assigned to propensity score strata, the fifth step of the analysis was to estimate the average treatment effect on health and mortality across the strata and fit a linear regression line over these data points. To undertake these analyses, we used Jann, Brand and Xie's (2008) "hte" Stata module. HTE produces a variance-weighted least squares regression estimate for the intercept and slope of the average treatment effects. This is a hierarchical linear model in the sense that each stratum's treatment equation is nested within the second-level regression that summarizes the linear trend in treatment effects across propensity strata. The two-level approach—treatment effects nested within propensity strata—using a variance-weighted least squares estimator is a preferable method to a single-level interaction term (i.e., college degree completion times propensity score) because the former strategy accounts for heteroskedasticity in the effects of college across the range of propensity scores. The latter approach—single-level regression—would be less efficient and has problematic distributional assumptions because it supposes homogeneity of treatment variance. Recent empirical applications of the HTE analytic model include college's association with earnings (Brand and Xie 2010) and civic participation (Brand 2010).

Related to the current study, evidence for the *added protection hypothesis* would be demonstrated by a downward level-two slope: Risk for a given health problem would be decreased among college graduates, but by a growing amount for those most likely to have graduated college. Conversely, the *compensatory leveling hypothesis* would be supported by a positive, level-two slope: The average treatment effects decrease health risk but the relative benefit shrinks from the lowest propensity stratum to the highest.

Results

Table 1 presents the means of each variable used in this study, differentiated by college completion status. Consistent with the expectation that college attainment endows a health benefit, college graduates have lower rates of hypertension, heart problems and mortality (all differences significant at $p < .001$). These differences represent the focal inquiry of this study, as the health benefits of a college degree may differ according to the background factors preceding this life course transition. Indeed, college graduates in the MIDUS survey display the marks of early life advantage; this is demonstrated by lower levels of divorce, less likelihood of frequent physical or emotional abuse, lower likelihood of being on welfare during childhood, higher levels of parental education and parents represented in higher status occupations. Ignoring this vector of childhood

Table 1. Descriptive Statistics of Variables from the MIDUS (N = 2,909), Displayed by College Completion Status (30.22% College Graduates)

	Non-College Graduate	College Graduate
Hypertension	0.202	0.145***[a]
Heart problems	0.140	0.084***
Mortality	0.092	0.052***
Parental divorce	0.139	0.100**
Poor health at age 16	0.040	0.028
Physical abuse[b]		
Rare	0.309	0.366**
Often	0.302	0.197***
Emotional abuse[b]		
Rare	0.251	0.317***
Often	0.381	0.309***
Welfare	0.075	0.023***
Father's education[b]		
High school	0.336	0.407***
College	0.080	0.299***
Missing on education	0.160	0.076***
Mother's education[b]		
High school	0.461	0.567***
College	0.056	0.218***
Missing on education	0.079	0.022***
Father supervised	0.418	0.604***
Father's occupation[b]		
Upper-white-collar	0.142	0.374***
Missing on occupation	0.131	0.091**
Mother's occupation[b]		
Upper-white-collar	0.076	0.171***
Missing on occupation	0.512	0.490
Age	47.660 (13.369)[c]	45.643 (12.426)***
Non-white	0.127	0.107
Female	0.550	0.430***

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

Note: All dichotomous variables are scored 0 and 1 (0 = no or otherwise). [a]p-value from χ^2 tests for binary variables and *t* tests for continuous variables; [b]reference group is no abuse, less than high school, and blue-collar/lower-white-collar; [c]standard deviation.

background variables, therefore, complicates the interpretation of how educational attainment shapes adult health.

A simple model that neglects childhood background and assumes homogenous treatment effects of a college degree is presented in Table 2. The purpose of this logistic regression analysis is to demonstrate that there is indeed the expected association between college completion and health conditions without the complicating consideration of unequal "treatment" (i.e., college) propensities. In line with the descriptive statistics in Table 1, college graduates have lower prevalence of disease and lower mortality risk, an association that is robust upon including several demographic covariates also related to adult health. These findings are also consistent with prior observational studies that report that college completion has a long-term direct effect on health (e.g., Ross and Wu 1995).

But the assumption of equal treatment effects may be unfounded. Following the initial steps toward testing for heterogeneous treatment effects, the childhood background variables shown in Table 1 were used to calculate propensity scores for college completion. We used a binary logit regression with college degree as the dependent variable. Coefficients from this equation are presented in the appendix (Table A1). Probabilities for college completion were then generated from the logit model and each respondent was assigned a propensity score. In addition, the condition of common support was applied to improve the propensity score matching.[3] For the analysis of hypertension and heart problems, respondents were grouped into seven strata in order to satisfy the balancing property; for mortality, respondents were classified by six strata. Given the lower number of deaths (n = 233) compared with cases of hypertension (n = 534) or heart problems (n = 357), there was insufficient mortality to distribute over seven strata, especially at the high range of college completion propensity. Therefore, mortality is presented separately from the two disease outcomes.

Results of generating the strata are displayed in Table 3, and demographic data for each stratum are presented in Table 4 (six strata for mortality follow the seven strata for hypertension and heart problems).

Hypertension and Heart Disease

As expected, people with low propensity for college completion tend to be concentrated most heavily among the nontreated ($d = 0$), whereas people with higher propensity scores are more heavily represented among the college graduates ($d = 1$; see Table 3). Nevertheless, there are sufficient cases falling into the counterfactual condition of completing college though being in the lowest range of propensity scores (i.e., 18 individuals in the .02-.06 strata). The uppermost propensity stratum includes scores from the 0.75-1.0 range.

After respondents were assigned to propensity score strata, we used the HTE module to assess the competing selection hypotheses (added protection vs. compensatory leveling). Results of estimating the average treatment effects for each propensity stratum and then fitting a level-two regression line to capture the linear trend are each summarized in Figures 1 and 2. To investigate the effects

Table 2. Homogeneous Effects of College Completion and Control Variables on Health

	Hypertension		Heart Problems		Mortality	
College	-0.444**[a]	0.642[c]	-0.514**	0.598	-0.572**	0.565
	(0.130)[b]		(0.157)		(0.200)	
Age	0.057***	1.059	0.040***	1.041	0.097***	1.102
	(0.005)		(0.006)		(0.009)	
Non-white	0.774***	2.168	-0.072	0.930	0.395	1.484
	(0.188)		(0.238)		(0.309)	
Female	-0.039	0.962	-0.217	0.805	-0.220	0.803
	(0.126)		(0.138)		(0.185)	
Constant	-4.305***		-3.701***		-7.533***	
N	2,895		2,901		2,909	

*p < .05 **p < .01 ***p < .001
[a]Coefficient
[b]Standard error
[c]Odds ratio

Table 3. Respondents by Propensity Score Strata

Hypertension and Heart Problems		
P-Score	$d = 0$	$d = 1$
[.02, .06)	224	18
[.06, .12)	646	102
[.12, .19)	394	100
[.19, .25)	262	111
[.25, .50)	379	293
[.50, .75)	105	197
[.75, .88)	20	58
Mortality		
P-Score	$d = 0$	$d = 1$
[.02, .08)	455	41
[.08, .17)	709	145
[.17, .25)	362	145
[.25, .33)	190	136
[.33, .67)	269	283
[.67, .88)	45	129

Note: d = treatment condition. P-scores range from .02-.88 due to the common support restriction.

Table 4. Descriptive Statistics of Controls from the MIDUS (N = 2,909), Displayed by Propensity Score Strata

Strata for Hypertension and Heart Problem Analysis																					
	Stratum 1 [.02, .06)			Stratum 2 [.06, .12)			Stratum 3 [.12, .19)			Stratum 4 [.19, .25)			Stratum 5 [.25, .50)			Stratum 6 [.50, .75)			Stratum 7 [.75, .88)		
Variables	$d=0$	$d=1$	B	$d=0$	$d=1$	B	$d=0$	$d=1$	B	$d=0$	$d=1$	B	$d=0$	$d=1$	B	$d=0$	$d=1$	B	$d=0$	$d=1$	B
Age	49.263	44.000	48.872	50.621	50.225	50.567	47.525	49.910	48.008	45.115	45.991	45.375	45.129	45.676	45.368	43.467	42.487	42.828	40.000	40.621	40.462
Non-white	0.156	0.222	0.161	0.156	0.147	0.155	0.135	0.210	0.150	0.065	0.108	0.078	0.106	0.096	0.101	0.086	0.061	0.070	0.150	0.034	0.064
Female	0.804	0.722	0.798	0.615	0.588	0.611	0.533	0.430	0.512	0.492	0.441	0.477	0.401	0.392	0.397	0.438	0.447	0.444	0.150	0.172	0.167
Sample Size	224	18	242	646	102	748	394	100	494	262	111	373	379	293	672	105	197	302	20	58	78
Strata for Mortality Analysis																					
	Stratum 1 [.02, .08)			Stratum 2 [.08, .17)			Stratum 3 [.17, .25)			Stratum 4 [.25, .33)			Stratum 5 [.33, .67)			Stratum 6 [.67, .88)					
Variables	$d=0$	$d=1$	B	$d=0$	$d=1$	B	$d=0$	$d=1$	B	$d=0$	$d=1$	B	$d=0$	$d=1$	B	$d=0$	$d=1$	B			
Age	50.593	47.049	50.300	49.261	51.103	49.574	45.124	46.014	45.379	45.305	46.103	45.638	44.472	44.035	44.248	42.156	41.682	41.805			
Non-white	0.145	0.195	0.149	0.150	0.172	0.153	0.094	0.131	0.105	0.116	0.118	0.117	0.086	0.078	0.082	0.156	0.031	0.063			
Female	0.749	0.610	0.738	0.551	0.538	0.549	0.508	0.428	0.485	0.426	0.346	0.393	0.390	0.452	0.422	0.333	0.295	0.305			
Sample Size	455	41	496	709	145	854	362	145	507	190	136	326	269	283	552	45	129	174			

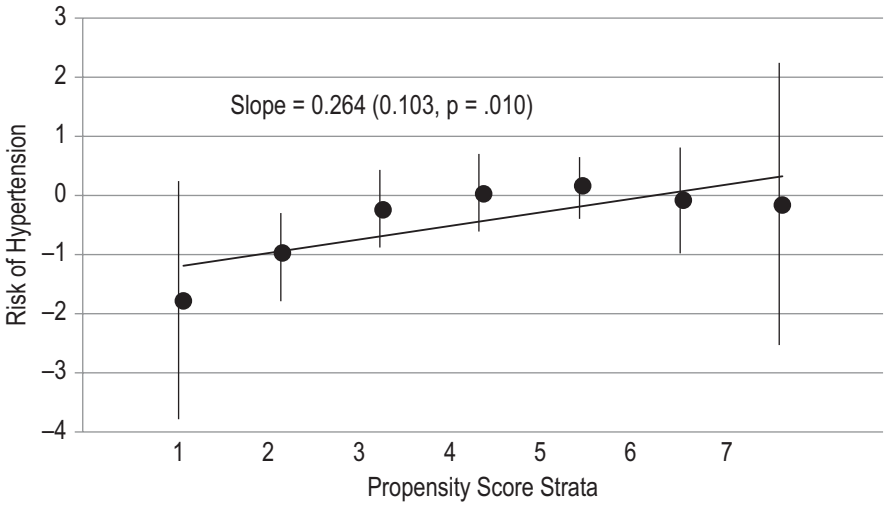
Note: Means are presented, where d indicates the treatment condition and B represents the overall mean for each propensity score stratum. t tests indicate that mean propensity scores did not differ between non-treated ($d = 0$) and treated ($d = 1$) respondents for any of the strata in Table 4 ($p < .001$; see Brand and Xie 2010:281).

across hypertension and heart problems, we incorporate a total of 14 separate logistic regression models (seven strata for two outcomes), the coefficients of which are shown in Table 5. Each equation models the response to the health condition at a single level of college completion propensity (e.g., hypertension at strata .02-.06; .06-.12; .12-.19; .19-.25; .25-.50; .50-.75; .75-.88), and the pooled treatment effects across the various strata represent level-one units. For a parsimonious representation of the treatment effects across propensity strata, the two figures show standard errors around each estimated treatment effect and provide a p value to assess the significance of the level-two slopes. Analysis of both health outcomes clearly supports the *compensatory leveling hypothesis*. In each case, the health benefit of a college degree is most pronounced among MIDUS respondents with the lowest propensity for attaining such a degree.

Though the level-two slopes vary in their magnitude, both reaching statistical significance in a two-tailed test ($p < .10$). This is noteworthy because, as Brand and Xie (2010) point out, the level-two coefficients are based on very few data points. Whereas the above authors reported small and nonsignificant level-two slopes (with absolute values ranging from .01 to .14 across a range of model specifications), the effects observed for the HLM regression lines in hypertension and heart problems are notable in their magnitude relative to their standard errors. In essence, the number of observations on which these regression lines are fitted is merely 7 (the number of propensity strata), so the expectations for observing "statistical significance" in this analysis are much lower than in the typical large random sample drawn from a population. For an overall one degree-of-freedom representation of heterogeneous treatment effects, this is a clear and compelling pattern of compensatory leveling selection. Those least likely to attain a degree are the ones most likely to gain health benefits from it.

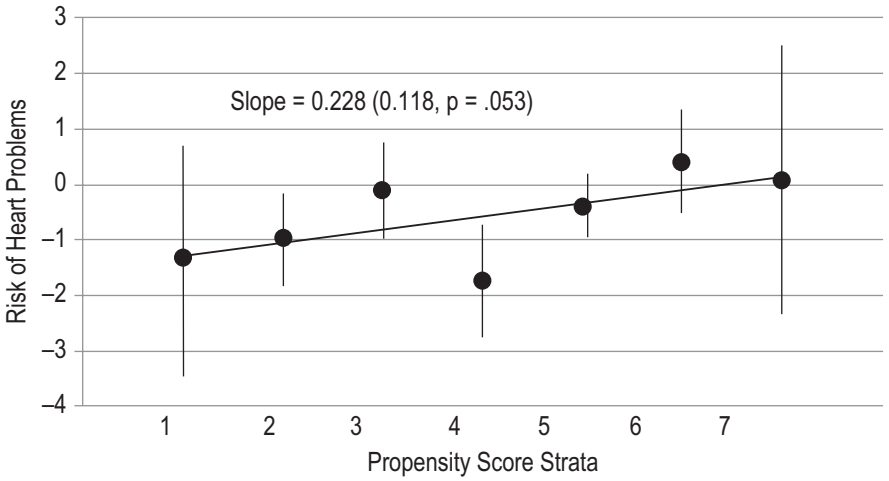
Interestingly, however, the treatment effects themselves (level-one data) do not consistently predict clear health benefits. Because the points in Figures 1 and 2 represent logit coefficients, values below the zero on the Y-axis indicate lowered risk of disease. Standard error bars are also provided to evaluate the precision of the estimate. The point estimates and standard error bars in Figures 1 and 2 indicate that when college protects health, it does so only among respondents in lower levels of propensity strata (stratum .06-.12 for hypertension and heart problems). In the highest propensity strata (e.g., .75-.88), the treatment effect of college plainly straddles both vertical sides of the null effect line. Translating the unstandardized logit coefficients into more interpretable predicted probabilities shows that respondents in the lowest propensity stratum who nevertheless completed college had a 0.057 probability of hypertension, whereas those who did not earn a college degree had a 0.264 probability.[4] By contrast, respondents in the highest propensity stratum who completed college had a 0.029 probability of hypertension, whereas those who did not earn a college degree had a 0.034 probability. In predicting heart problems, the differential effects are most pronounced among those who did not obtain a college degree: The probability of heart problems was 0.186 for those in the lowest propensity stratum, compared with a probability of 0.034 for those in the highest propensity stratum.

Figure 1. Heterogeneous Treatment Effects of College on Hypertension



Note: Standard error in parentheses.

Figure 2. Heterogeneous Treatment Effects of College on Heart Problems



Note: Standard error in parentheses.

Predicted probabilities of each health condition by college completion status across the seven propensity strata can be viewed in Figure A1 in the Appendix.

Drawing strict inferences from each of the 14 logistic regressions individually, however, becomes somewhat problematic because splitting the overall sample into propensity strata reduces statistical power. This becomes an acute dilemma when the condition is relatively rare; for instance, the fact that college attainment does not appear to have a statistically significant protective role for the

privileged .75-.88 propensity stratum (as demonstrated by the wide error bar in Figure 1) is likely influenced by the relatively low prevalence of hypertension in this subsample of the MIDUS (only four cases of hypertension observed). That being said, the relevant result from Figures 1 and 2 (and Table 5) is the *linear trend across propensity strata*, not the single point estimate for each separate stratum.

Mortality

The overall pattern of propensity score strata assignment was similar in the analysis of morality as it was for hypertension and heart problems. As mentioned above, the relatively small number of deaths required us to use one less stratum.

The HTE model was then applied to the mortality outcome, this time summarizing a level-two slope obtained from regression estimates within six level-one propensity strata (as there was one fewer stratum than was used for the disease outcomes). As with hypertension and heart problems, the results favor the compensatory leveling hypothesis. The level-two slope is positive (0.256, $p = .088$), indicating that the treatment effect is most pronounced (i.e., lowest probability of death) in lower strata of college propensity and approaches zero at higher strata of propensity. Indeed, significant within-strata treatment effects are observable only among respondents who were not likely to complete college—see the unstandardized coefficient of -1.78 ($p < .001$) in Table 5 and note the error bars below the zero threshold in strata 2 in Figure 3. As with the two disease outcomes, predicted probabilities for mortality by college completion status are shown across all propensity strata (see appendix). In the two lowest strata, college completion versus no completion is associated with a 0.065 vs. a 0.107 probability of death (strata 1) and a 0.017 vs. a 0.095 probability of death (strata 2). In the highest strata, however, the probabilities of death were more consistent between college completers and non-completers (.036 vs. .029).

Taken together, these patterns for hypertension, heart disease and mortality suggest that the health-protective benefits of college education taper to the point of nonexistence among people of the most privileged background. While the benefits of education found in prior studies and reported in Table 2 seem somewhat straightforward, the endogeneity of childhood background with subsequent life course pathways imply that education's benefits are conditional rather than homogenous.

Discussion

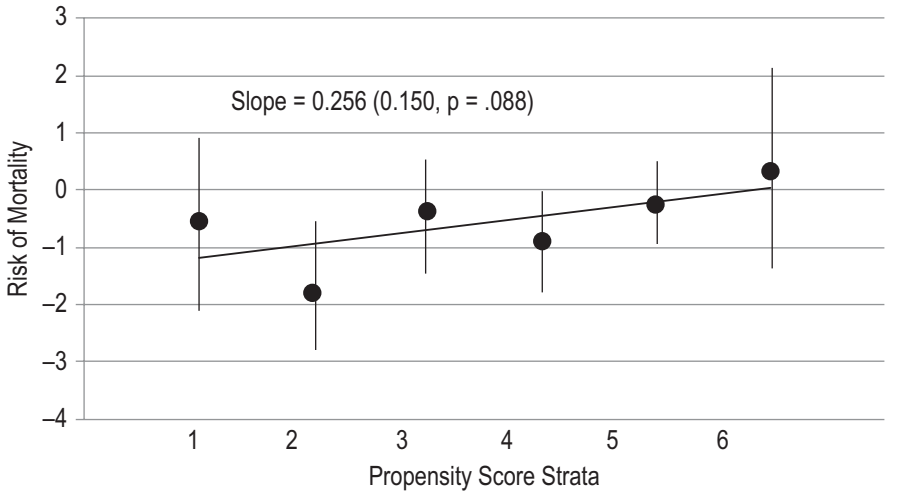
The health advantage of college-educated adults is one of the most consistent empirical findings in the sociology of health and the life course. Though few cast serious doubt that education improves life chances, a simple corresponding fact has not often been explicitly acknowledged when assessing the "benefits" of higher education: Completion of college does not occur haphazardly in a population. Indeed, the same mix of advantages and disadvantages that help or hinder educational attainment are implicated in health disparities over the life

Table 5. Heterogeneous Effects of College Completion on Health

	Hypertension		Heart Problems			Mortality	
Level-1 Slopes					**Level-1 Slopes**		
Stratum 1	-1.778*[a]	-1.025***[c]	-1.328	-1.475***[c]	*Stratum 1*	-0.547	-2.120***[c]
[.02, .06)	(1.056)[b]		(1.060)		[.02, .08)	(0.764)	
Stratum 2	-0.975***	-1.118***	-0.952**	-1.774***	*Stratum 2*	-1.784***	-2.258***
[.06, .12)	(0.346)		(0.432)		[.08, .17)	(0.550)	
Stratum 3	-0.239	-1.236***	-0.114	-2.176***	*Stratum 3*	-0.369	-2.793***
[.12, .19)	(0.293)		(0.443)		[.17, .25)	(0.433)	
Stratum 4	0.028	-1.687***	-1.728***	-1.766***	*Stratum 4*	-0.896*	-2.442***
[.19, .25)	(0.314)		(0.516)		[.25, .33)	(0.460)	
Stratum 5	0.155	-1.892***	-0.406	-2.011***	*Stratum 5*	-0.241	-2.939***
[.25, .50)	(0.243)		(0.277)		[.33, .67)	(0.369)	
Stratum 6	-0.080	-2.547***	0.414	-2.525***	*Stratum 6*	0.345	-3.678***
[.50, .75)	(0.468)		(0.448)		[.67, .88)	(0.885)	
Stratum 7	-0.156	-3.362***	0.070	-3.362***	**Level-2 Slope**	0.256*	
[.75, .88)	(1.205)		(1.227)			(0.150)	
Level-2 Slope	0.264**		0.228*				
	(0.103)		(0.118)				

*p < .1 **p < .05 ***p < .01
[a]Coefficient; [b]standard error; [c]intercept

Figure 3. Heterogeneous Treatment Effects of College on Mortality



Note: Standard error in parentheses.

course. From these basic insights, it follows that *factors from early in the life course may condition the benefits of college education on adult health*.

This article sought to answer whether the "treatment" effects of a college degree on health are truly homogenous, an effect that many prior studies have often implicitly taken for granted. Results from a national sample of American adults suggest that the homogeneity assumption is unwarranted. Analyzing self-reported hypertension and heart problems, as well as mortality records traced by the NDI, we found that *the benefits of college degree completion were highest among those least likely to attain a degree as a consequence of early misfortune*. This is consistent with the "compensatory leveling" hypothesis—a heterogeneous treatment effect favoring people who are most likely to be selected out of the treatment in the first place. For heart disease and mortality, obtaining a college degree leveled the risk enough that those with the lowest propensity scores were no more likely to experience the health outcome than those at the highest propensity of degree completion (see Figure A1 in the Appendix). For instance, college graduates from the lowest propensity group had an approximately identical predicted probability of heart disease (5.7%) to those in the two highest propensity groups that did not attend college (3.4% and 7.4%, respectively). Predicted probability of hypertension was decreased by 78 percent for college graduates versus noncollege graduates in the low-propensity group (.264 vs. .057; (.264-.057/.264)x100) = -.78), though the prevalence of this condition was somewhat higher overall.

Our analyses incorporated insights from experimental design for observational data; using a vector of childhood background variables, we estimated the probability that an individual would complete college. We then examined the protective effect of higher education across different levels of college degree propensity, summarizing the overall trend with a hierarchical linear model

(Brand and Xie 2010). Although people from households marked by divorce, low SES and maltreatment were less likely to gain a college education relative to people of a more advantaged background, the individuals who completed college despite an inauspicious start stood to gain the most. The consistency of this finding across two serious, life-threatening health problems and mortality provides some evidence of the robustness of this heterogeneous selection effect finding.

What makes the selection findings perhaps more intriguing is the degree of similarity with which they resemble other recent findings on the benefits of education. Brand and Xie (2010) investigated the benefits of a college degree for earnings across early adulthood to 40 years of age. The authors report that while education boosts wage earnings, the return is greatest among those least likely to attain a college degree. The correspondence of two key processes that unfold throughout adulthood—earning potential and risk of serious health problems—may signify a more generalizeable finding for life course sociology. That is, educational attainment in early adulthood appears to serve as a unique transition for helping to level the playing field and reduce the effects of early life misfortune—conditional, of course, on individuals actually completing college. A runaway cumulative advantage/disadvantage perspective may assume that college completion only widens inequality in wages or health by systematically benefitting those most likely to attain and advance through this life course transition. The current study suggests that health, like wages, does not fit such an assertion.

Though this article has aimed to give nuanced attention to a core issue in health and life course sociology, the findings should be seen as primarily preliminary and descriptive accounts of a population pattern. The analyses illuminate no decisive causal mechanisms to explain why people with a college degree despite a low propensity should receive a better health benefit from education. Several accounts are plausible. For one, people least likely to go to college have the most ground to recover from the most advantaged. Prior work, for instance, demonstrates that children from disadvantaged backgrounds have the greatest academic gain from being in school, (Downey, von Hippel and Broh 2004). Related to the adults examined in this study, educational attainment could narrow the gap between these people in domains such as health literacy, health behaviors and lifestyle choices, sense of personal mastery and control or breadth of social networks. People from the most advantaged backgrounds may have already received these benefits by virtue of their social class and apart from their college experience itself, and so higher education would offer them a diminishing rate of return for adult health. Those who are disadvantaged early, however, reap a stronger health benefit, which may be viewed as a *compensatory mechanism* (Ferraro and Kelly-Moore 2003). Supplementary analyses (not shown) explored this premise by introducing controls for adult obesity, personal control and smoking, but these factors accounted for a very modest share of the heterogeneous treatment effects pattern. The inability to completely account for this finding may be due, in part, to the fact that the MIDUS data did not contain an exhaustive battery of all potential compensatory mechanisms.

Another explanation of this study's findings is a *compositional variability* argument. The population of privileged individuals (for whom college education is an expected norm) comprises people quite diverse in their personal abilities, intelligence and motivation. For this set of individuals, college education may have been driven in large part by already established life course gridlines guiding the transition to adulthood. In contrast, people with very low college completion propensities that defy their statistical odds may represent individuals of unusual resilience possessing a disproportionate reserve of valuable personal traits. These characteristics, in turn, should manifest in better health and lower susceptibility to disease and death over the life course. Again, this possibility remains speculative, as "exceptional resilience" is a theoretic characteristic not measured in our models.

An additional possibility is derived from behavioral economic reasoning. Some initial results suggest that a contributing reason for the heterogeneous selection effects in wages is due to low-propensity college students attempting to maximize their educational opportunities by selecting majors with immediate and maximal economic payoff (e.g., business), while high-propensity graduates are relatively less motivated by economic concerns and more likely to major in academic fields that offer less compensation (Brand and Xie 2010). The role of *personal choice* in educational options could conceivably shape adult health if it sorts people into different vocational tracks, but there is currently insufficient theory to articulate how these processes should unfold. For instance, it is not clear whether different vocational pathways following a college degree (e.g., management, law, engineering, education) lead to divergent health practices in the same way that these career choices influence wage growth.

Taken together, an important area of future study will be to differentiate between the compensatory mechanism, compositional variability and personal choice arguments in accounting for the unequal benefits of college attainment on adult health. It is probable that none of these factors completely accounts for the patterns in isolation. Demographic, survey-based research like the current article can be helpful in identifying broad trends, but ethnographic investigation may be more fruitful in explicating the crucial mechanisms in detailed precision. In-depth, qualitative work is needed to understand the lived experience of "low-propensity" college students (e.g., first-generation students), including their postcollege adulthood transition and the concomitant cultivation and crystallization of their health behaviors.

Though the present findings offer multiple avenues for future research, several limitations of this study must be kept in mind. First, our overall conclusions about heterogeneous treatment effects of college are derived from a multilevel analysis assessing trends across strata-specific regression estimates, many of which, individually, were not statistically significant. That is, when we divide MIDUS respondents according to their probability of completing college and then estimate the influence of college among these separate subsamples, we are unable to find consistent evidence of health-protective effects (though the coefficients for college completion are clearly significant in the simple homogenous effects model in Table 2). The fact that

the unique effects of college trend toward "0" at the high propensity levels support the view that health payoffs decline among those most advantaged, but this conclusion may be premature because statistically significant protective effects are not observed among people at the lowest end of the propensity distribution (e.g., stratum "1" in Figures 2 and 3). The wide confidence intervals for the regression estimates of the lowest and highest strata may be the reason, as reflected in Figures 1 to 3 and Table 3. Although our main interest was in the overall (level-two) slopes, we recognize that the concerns above necessitate further research on this topic before we can make conclusive statements about the heterogeneous effects of college on adult health. We urge other scholars to replicate our findings with alternative data from larger samples and with additional outcomes; especially useful would be to study health conditions that are distributed more continuously and not as vulnerable to the problem of rare occurrence in a given study sample.

As a second limitation, the MIDUS data used for our analyses were collected among adults; therefore, all documentation of childhood conditions is entirely retrospective. All reports of childhood conditions are assumed to be accurate despite the potential for recall bias or recollection problems. Another concern about the retrospective data is that no measures of childhood intelligence, motivation or plans to attend to college are available. Interviews with MIDUS respondents during childhood would have been useful variables to assess additional personal resources predicting educational attainment. That being said, the propensity scores used in this article were generated by a vector of variables representing a broad mix of childhood background factors—family structure, household SES, exposure to abuse—and so our concern has been more in line with early social advantage/disadvantage than with the myriad intellectual or behavioral traits that predict educational attainment. If there is an omitted variable bias, then it may result in an underestimate of the true effects. Brand and Xie (2010) demonstrate that the negative selection effect disappears in their wage data (i.e., slope of level-two effects change from -.02 to .03) when they remove measures such as high school achievement and academic aspirations. The robust and statistically significant level-two slopes reported in this study (0.26 for hypertension, 0.23 for heart problems, and 0.26 for mortality) indicate that selection effects are not masked when health is the dependent variable of interest, though perhaps they are somewhat underestimated.

A third limitation concerns the fact that the current study focused on the contingent health benefits of education, while bracketing the issue of whether childhood misfortune itself has direct or indirect effects on health and mortality (Hayward and Gorman 2004). The straightforward consequences of childhood misfortune are a pressing issue—and the focus of many other studies—but the emphasis of our models is on how early life conditions relate to the education-health association. Nevertheless, we acknowledge that viewing early life misfortune solely as a prelude to subsequent educational attainment passes over the wide range of other status changes and transitions that comprise life trajectories (Elder, Johnson and Crosnoe 2003).

Conclusion

The findings from this study suggest that inattention to childhood experiences may lead to an oversimplified conception of the benefits of education on health. Sociology has long identified the benefits of education on health and well-being (Hummer and Lariscy 2011; Mirowsky and Ross 2003; Schnittker 2004), but this analysis highlights the endogeneity of college completion itself as related to the relationship between education and health. It also emphasizes a longer view of the life course origins of adult health. Education is often seen as an arena for the fundamental cause of health inequality to play out (Freese and Lutfey 2011; Link et al. 2008), but the present analysis points toward how childhood experiences are part of the process by which one completes college and derives a health benefit from higher education.

Finally, while colleges and universities may offer a unique opportunity to help reduce health disparities, these institutions will likely have trouble meeting the challenge. Current state and federal fiscal problems may reduce available college scholarships and re-establish barriers for disadvantaged youths. This possibility—a slowing or even a reversal of the momentum toward wider college accessibility generated in recent decades—poses genuine threats to population health over the long term. Recent evidence suggests that health gradients in education have increased during the contemporary era of college expansion (Hummer and Lariscy 2011). With reducing social disparities in health and mortality, a signature goal of the Centers for Disease Control (2011) and other national health agencies, much remains to be seen as to the role that colleges and universities can play in this endeavor. Whatever the case, the conclusion from this analysis suggests that the health benefits of a college education are greatest for those least likely to complete it.

Notes

1. Father's and mother's education had 14.02 percent and 6.83 percent missing data, respectively, and this rate was 12.27 percent and 50.86 percent for occupational category (including those who were not in the workforce). All other indicator variables had less than 5 percent missing data (.03% for parental divorce; .69% for physical abuse; 1.02% for emotional abuse; 2.90% for nonwhite; and .07% for age).
2. Though our focus for the estimation of college propensity is on childhood conditions and events that represent advantage/disadvantage, additional factors clearly predict college completion. Demographic traits available in the MIDUS data are therefore also included in the propensity score models. Preliminary analyses conducted the propensity score analysis without the use of demographic variables and instead controlled for them in heterogeneous treatment effect models estimating the health consequences of college (as level-one variables in logistic regression estimation). The results of these analyses were substantively identical to the ones presented in the current study.
3. To meet the condition of support, 14 cases were dropped from the analysis. This explains why there are 2,923 observations used to calculate propensity scores for college completion (Table A1), yet only 2,909 respondents are assigned to a propensity score stratum and used in the analysis.
4. Predicted probabilities shown in Figure A1 and discussed in the text were calculated in Stata after each of the 14 logistic regressions. The formula is as follows: exp(a + bx)/

(1 + exp(a + bx)), where "a" is the intercept and "b" is the unstandardized coefficient given for college completion (value of 1 or 0). For example, the predicted probability for hypertension among those in the lowest propensity stratum who completed college can be computed by substituting coefficients from Table 5 into the above equation as follows: exp(-1.025 + (1)*-1.778)/(1 + exp(-1.025 + (1)*-1.778)) = .057. This indicates that the model predicts a 6 percent probability of hypertension for individuals fitting the assigned characteristics. In addition, 95 percent confidence intervals were calculated by converting the regression standard error to a probability. The width of the predicted probability confidence intervals averaged 11 percentage points for hypertension analyses and 9 percentage points for heart problem analyses (averaged across the 7 strata for each outcome). Confidence intervals were widest for lowest-stratum respondents, so care must be used when interpreting the specific predicted probabilities presented in the text. We caution against deriving firm conclusions from any single predicted probability point estimate.

Appendix

Model to Evaluate Unequal Health Benefits of College

The counterfactual approach to causal inference provides a framework for thinking about observational data in experimental terms (Morgan and Winship 2007). Under the "fundamental problem of causal inference," it is impossible to observe the true effect of treatment d on a dependent variable y from observational data (Holland 1986). For each person *i*, if $d = 0$ (no treatment), we are unable to assess what happens when $d = 1$ (treatment) for that same person i. And of course, the reverse is also true. Causal inference in social science thus relies on *group-level* variability, an aggregated difference produced by d across a population of people (Holland 1986).

Using a vector of observed covariates, it is possible to "assign" *i* to a treatment or control condition on the empirical basis of which variables increase the probability that he or she was treated ($d = 1$). This enables us to explicitly obtain the "treatment effect on the treated," not simply the average treatment effect scattered across the entire population. Let y_i^1 be the value of some response variable under the conditions of treatment (college completion) and y_i^0 be the value of the dependent variable under the condition of no treatment (no college completion). Again, only one of these *y* values is actually observed with data.

$$E\left(y^0 \mid X, d = 1\right) = E\left(y^0 \mid X, d = 0\right) \tag{1a}$$

$$E(y^1 \mid X, d = 0) = E(y^1 \mid X, d = 1) \tag{1b}$$

The first equation specifies that had they not achieved a college degree, the average health value of college graduates would be the same as the average health value for non-college-graduates, conditional on observed covariates. Similarly, Equation 1b indicates that conditional on observed covariates, health values would be equal between college graduates and non-college-graduates, had they

obtained such a degree. Employing the conditional independence assumption (Gangl 2010), this gives us propensity scores as a strategy for satisfying equations 1a and 1b.

$$P = p\left(d_i = 1 \mid X\right) \tag{2}$$

The propensity score defined in equation 2 represents the probability of *i* being placed in the treatment group on the basis of *i*'s values on an observed vector of covariates. The covariates of interest in our study are childhood background variables, and *d* corresponds to college completion. Using this propensity score approach to confront pretreatment heterogeneity, we then follow Brand and Xie's (2010) lead to address heterogeneity of the treatment effect. Brand and Xie provide equation 3 for that purpose:

$$y_i = \alpha_i + \delta_i d_i + \beta' X_i + U_i \tag{3}$$

In equation 3, α_i and δ_i denote pre-treatment heterogeneity and treatment effect heterogeneity, respectively. Using propensity scores alleviates the need to estimate α_i, as heterogeneity in assignment to treatment is a function of observed covariates in a data set. Further, propensity scores provide a means for decomposing δ_i into subgroups on the basis of college-completion propensity. Again applying Brand and Xie's (2010) innovative design, we rely on a hierarchical linear model to examine potential treatment effect heterogeneity across aggregated propensity-based groups. Mean treatment effects and associated standard errors are estimated for each group, and a slope and intercept characterizing these effects are calculated as level-two parameters.

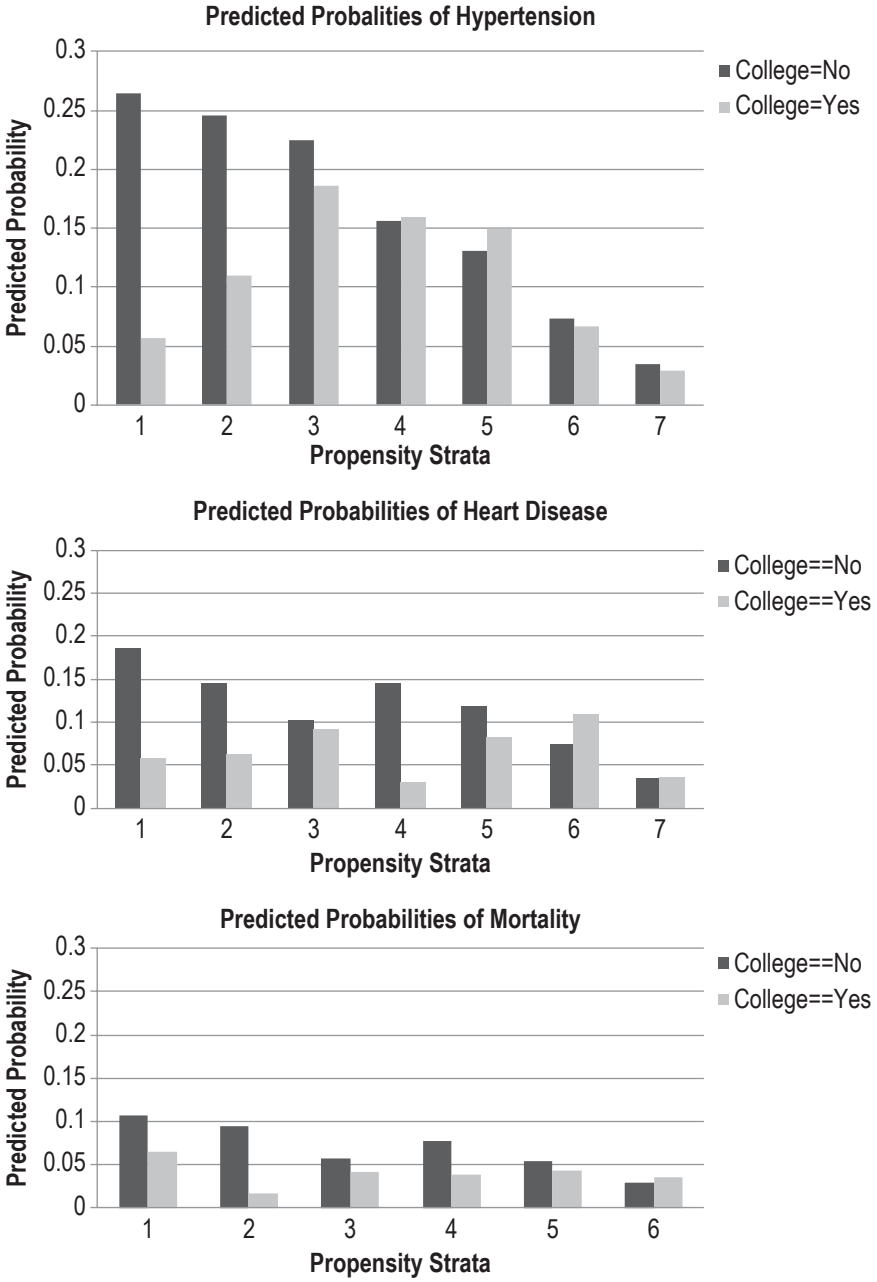
This statistical model is the grounds for testing between the rival hypotheses explicated above. The *added protection hypothesis* will be supported if having an advantaged background is associated with the greatest college-related reduction in cardiovascular disease and mortality risk. The *compensatory leveling hypothesis* posits the opposite—that college-related health gains are most pronounced among those facing the greatest levels of childhood misfortune.

Table A1. Binary Logit Model Predicting College Completion

	Coefficient	Standard Error
Parental divorce	-0.349*	0.177
Poor health at age 16	-0.305	0.290
Physical abuse[a]		
Rare	0.039	0.124
Often	-0.431**	0.161
Emotional abuse[a]		
Rare	0.185	0.130
Often	0.140	0.151
Welfare	-0.738*	0.302
Father's education[b]		
High school	0.609***	0.128
College	1.491***	0.182
Missing on education	-0.069	0.211
Mother's education[b]		
High school	0.636***	0.128
College	1.275***	0.204
Missing on education	-0.451	0.292
Father supervised	0.175	0.112
Father's occupation[c]		
Upper-white-collar	0.544***	0.136
Missing on occupation	0.414*	0.183
Mother's occupation[c]		
Upper-white-collar	0.312	0.189
Missing on occupation	-0.016	0.111
Age	0.010*	0.004
Non-white	0.203	0.169
Female	-0.409***	0.101
Log Likelihood	-1296.246	
N	2,923	

*p < .05; **p < .01; ***p < .001
[a]Reference group = no abuse
[b]Reference group = less than high school
[c]Reference group = blue-collar/lower-white-collar

Figure A1. Predicted Probabilities of Health Conditions by Propensity Strata and College Completion



Note: Predicted probabilities were calculated from the results in Table 5.

References

Barker, David J. P. 1994. "The Fetal Origins of Adult Disease." *Fetal and Maternal Medicine Review* 6:71-80.

Brand, Jennie E. 2010. "Civic Returns to Higher Education: A Note on Heterogeneous Effects." *Social Forces* 89(2):417-33.

Brand, Jennie E., and Yu Xie. 2010. "Who Benefits Most from College?" *American Sociological Review* 75:273-302.

Centers for Disease Control and Prevention (CDC). 2011. "CDC Health Disparities and Inequalities Report—United States, 2011." *Morbidity and Mortality Weekly Report*. U.S. Health and Human Services.

CDC/National Center for Health Statistics. "National Death Index." Last modified July 19, 2012. http://www.cdc.gov/nchs/ndi.htm.

Cockerham, William. 2005. "Health Lifestyle Theory and the Convergence of Agency and Structure." *Journal of Health and Social Behavior* 46:51-67.

Dannefer, Dale. 2003. "Cumulative Advantage/Disadvantage and the Life Course: Cross-Fertilizing Age and Social Science Theory." *Journal of Gerontology: Social Sciences* 58B:S327-37.

Downey, Douglas B., Paul T. von Hippel, and Beckett A. Broh. 2004. "Are Schools the Great Equalizer? Cognitive Inequality during the Summer Months and the School Year." *American Sociological Review* 69:613-35.

Duncan, Greg J., W. Jean Yeung, Jeanne Brooks-Gunn and Judith R. Smith. 1998. "How Much Does Childhood Poverty Affect the Life Chances of Children?" *American Sociological Review* 63:406-23.

Dupre, Matthew E. 2007. "Educational Differences in Age-Related Patterns of Disease: Reconsidering the Cumulative Disadvantage and Age-As-Leveler Hypotheses." *Journal of Health and Social Behavior* 48:1-15.

Eide, Eric R., and Mark H. Showalter. 2011. "Estimating the Relation between Health and Education: What do We Know and What do We Need to Know?" *Economics of Education Review* 30:778-91.

Elder, Glen H., Monica Kirkpatrick Johnson, and Robert Crosnoe. 2003. "The Emergence and Development of Life Course Theory." Pp. 3-19 in *Handbook of the Life Course*, *Handbooks of Sociology and Social Research*, edited by Jeylan T. Mortimer, and Michael J. Shanahan. Springer.

Farmer, Melissa M., and Kenneth F. Ferraro. 2005. "Are Racial Disparities in Health Conditional on Socioeconomic Status?" *Social Science & Medicine* 60:191-204.

Felitti, Vincent J., Robert F. Anda, Dale Nordenberg, David F. Williamson, Alison M. Spitz, Valerie Edwards, Mary P. Koss, and James S. Marks. 1998. "Relationship of Childhood Abuse and Household Dysfunction to Many of the Leading Causes of Death in Adults: The Adverse Childhood Experiences (ACE) Study." *American Journal of Preventive Medicine* 14:245-58.

Ferraro, Kenneth F., and Jessica A. Kelley-Moore. 2003. "Cumulative Disadvantage and Health: Long-Term Consequences of Obesity?" *American Sociological Review* 68:707-29.

Freese, Jeremy, and Karen Lutfey. 2011. "Fundamental Causality: Challenges of an Animating Concept for Medical Sociology." Pp. 67-81 in *Handbook of the Sociology of Health, Illness, and Healing: A Blueprint for the 21st Century*, edited by Bernice A. Pescosolido, Jack K. Martin, Jane D. McLeod, and Anne Rogers. Springer.

Gangl, Markus. 2010. "Causal Inference in Sociological Research." *Annual Review of Sociology* 36:21-47.

Gruenewald, Tara L., Arun S. Karlamangla, Perry Hu, Sharon Stein-Merkin, Carolyn Crandall, Brandon Koretz, and Teresa E. Seeman. 2012. "History of Socioeconomic Disadvantage and Allostatic Load in Later Life." *Social Science & Medicine* 74:75-83.

Haas, Steven A. 2006. "Health Selection and the Process of Social Stratification: The Effect of Childhood Health on Socioeconomic Attainment." *Journal of Health and Social Behavior* 47:339-54.

Haveman, Robert, and Barbara Wolfe. 1995. "The Determinants of Children's Attainments: A Review of Methods and Findings." *Journal of Economic Literature* 33:1829-78.

Hayward, Mark D., and Bridget K. Gorman. 2004. "The Long Arm of Childhood: The Influence of Early-Life Social Conditions on Men's Mortality." *Demography* 41:87-107.

Holland, Paul W. 1986. "Statistics and Causal Inference." *Journal of the American Statistical Association* 81:945-60.

Hout, Michael. 2012. "Social and Economic Returns to College Education in the United States." *Annual Review of Sociology* 38:327-400.

Hummer, Robert A., and Joseph T. Lariscy. 2011. "Educational Attainment and Adult Mortality." Pp. 241-61 in *International Handbook of Adult Mortality*, vol. 2, *International Handbooks of Population*, edited by Richard G. Rogers and Eileen M. Crimmins. Springer.

Jann, Ben, Jennie E. Brand, and Yu Xie. 2008. HTE: Stata module to perform heterogeneous treatment effect analysis. Available at http://ideas.repec.org/.

Kao, Grace, and Jennifer S. Thompson. 2003. "Racial and Ethnic Stratification in Educational Achievement and Attainment." *Annual Review of Sociology* 29:417-42.

Kuh, Diana, and Yoav Ben-Shlomo. 2004. *A Life Course Approach to Chronic Disease Epidemiology*. Oxford University Press.

Link, Bruce G., Jo C. Phelan, Richard Miech, and Emily Leckman Westin. 2008. "The Resources That Matter: Fundamental Social Causes of Health Disparities and the Challenge of Intelligence." *Journal of Health and Social Behavior* 49:72-91.

Lleras-Muney, Adriana. 2005. "The Relationshps between Education and Adult Mortality in the United States." *Review of Economic Studies* 72:189-221.

Lundborg, Petter. 2008. "The Health Returns to Education—What Can We Learn from Twins?" IZA Discussion Paper No. 3399:1-32.

Malinosky-Rummell, Robin, and David J. Hansen. 1993. "Long-Term Consequences of Childhood Physical Abuse." *Psychological Bulletin* 114:68-79.

Merton, Robert K. 1968. "The Matthew Effect in Science." *Science* 159:56-63.

Mirowsky, John, and Catherine E. Ross. 1998. "Education, Personal Control, Lifestyle and Health." *Research on Aging* 20:415-49.

Mirowsky, John, and Catherine E. Ross. 2003. *Education, Social Status, and Health*. Hawthorne, NY: Aldine de Gruyter.

Morgan, Stephen L., and Christopher Winship. 2007. *Counterfactuals and Causal Inference: Methods and Principles for Social Research*. Cambridge University Press.

Palloni, Alberto. 2006. "Reproducing Inequalities: Luck, Wallets, and the Enduring Effects of Childhood Health." *Demography* 43:587-615.

Preston, Samuel H., Mark E. Hill, and Greg L. Drevenstedt. 1998. "Childhood Conditions That Predict Survival to Advanced Ages among African-Americans." *Social Science & Medicine* 47:1231-46.

Ross, Catherine E., and John Mirowksy. 2010. "The Interaction of Personal and Parental Education on Health" *Social Science & Medicine* 72:591-99

Ross, Catherine E., and Chia-Ling Wu. 1995. "The Links Between Education and Health." *American Sociological Review* 60:719-45.

Sandefur, Gary D., and Thomas Wells. 1999. "Does Family Structure Really Influence Educational Attainment?" *Social Science Research* 28:331-57.

Schnittker, Jason. 2004. "Education and the Changing Shape of the Income Gradient in Health." *Journal of Health and Social Behavior* 45:286-305.

Tucker, Joan S., Howard S. Friedman, Joseph E. Schwartz, Michael H. Criqui, Carol Tomlinson-Keasey, Deborah L. Wingard, and Leslie R. Martin. 1997. "Parental Divorce: Effects on Individual Behavior and Longevity." *Journal of Personality and Social Psychology* 73:381-91.