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Educational inequalities in health behaviors at midlife: Is there a role for early-life cognition?

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Abstract

Educational attainment is a fundamental cause of social inequalities in health because it influences the distribution of resources, including money, knowledge, power, prestige, and beneficial social connections that can be used *in situ* to influence health. However, recent studies have highlighted early-life cognition as commonly indicating the propensity for education and determining health and mortality. A primary causal mechanism through which education and adolescent cognition plausibly impact health is through the modification of health behaviors. We integrate analyses using the Wisconsin Longitudinal Study, the National Survey of Health and Development, and the National Childhood Development Study to examine the role of adolescent cognition and education on smoking, heavy drinking, and physical inactivity at midlife. Results suggest that adolescent cognition is associated with the propensity for education, but that associations between cognition and health behaviors are attenuated by adjusting for education. In contrast, education was robustly associated with poor health behaviors. These results support the view that education is robustly associated with health behaviors.

Keywords

Education; Cognition; Physical activity; Smoking; Drinking; Health Behaviors; Counterfactual; Life course analysis; National Child Development Study; National Survey of Health and Development; Wisconsin Longitudinal Study

Education is an primary determinant of the distribution of resources including knowledge, money, power, prestige and beneficial social connections (Link and Phelan 1995). Many researchers have suggested that have noted that social inequalities have a generalized impact on health (Marmot 2005); however, due to a lasting relationship between adolescent cognition with education and later-life health, researchers have questioned the reliability and interpretation of educational inequalities in health research (Deary and Johnson 2010). Such arguments arise in part because of limited data available to examine the nature of causal mechanisms; life course analysis is unique in providing us with the tools to examine long-

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term causal mechanisms (Dannefer 2003). In this paper, we use life course data to examine the extent to which educational inequalities in health behaviors can be explained by selective and causal mechanisms relating to adolescent cognition.

Background

A variety of social factors, including education, are related to health, disease, and ultimately mortality (Huisman et al. 2013, Link and Phelan 1995, Mackenbach et al. 2004, Masters, Hummer and Powers 2012, Mirowsky and Ross 2003). For example, examining the role of education on occupation, educational attainment has been associated with higher status and lower risk occupations throughout life (Becker 1980, Keeley 2007). Yet, there is a social element as well, as individuals with higher education tend to have friends and acquaintances who have better health self-management and are healthier for it (Christakis and Fowler 2007, Christakis and Fowler 2008). Indeed, One theory holds that education is important to health across the life course because it works flexibly and persistently to influence a host of social, economic, and behavioral factors that ultimately influence disease and death (Link and Phelan 1995, Link and Phelan 2010, Phelan and Link 2013). Thus, education indicates or modifies the risk of a range of mechanisms and exposures that work to influence health at multiple stages: for example, education may at once influence health behaviors, the ability to interpret and to comply with health recommendations, the ability to effectively interface with healthcare professionals, and the social and physical environment that individuals inhabit. Since development, the theory has since been tested and expanded upon using a variety of different outcomes and measures, socioeconomic status has been associated with smoking (Link and Phelan 2009), reduced use of preventive medicine including vaccination (Clouston, Kidman and Palermo 2014, Polonijo and Carpiano 2013) or cancer screening (Link et al. 1998), colorectal cancer mortality (Saldana-Ruiz et al. 2013), lung cancer mortality (Rubin, Clouston and Link 2014), suicide (Clouston et al. In Press), cognitive capability (Clouston et al. 2012, Glymour et al. 2008), and all-cause mortality (Huisman et al. 2013).

While education could reflect a structural cause of health, many researchers have suggested that it sometimes acts as a signal for other underlying factors such as investment in human capital (Becker 1980). A recent challenge to a causal interpretation of educational has arisen within the cognitive epidemiological literature, and broadly suggests that educational attainment is an outcome of individual characteristics, such as childhood achievement or cognition, that are known to predict an individual's propensity for educational attainment (Deary and Johnson 2010, Gottfredson 2004), which in turn has little independent effect on health-related outcomes. Following this, a rapidly-increasing body of research suggests that adolescent cognition both influences socioeconomic inequalities and predicts health, illness and mortality (Batty, Deary and Gottfredson 2007, Batty et al. 2009, Calvin et al. 2010, Deary et al. 2005, Deary et al. 2006, Deary et al. 2007, Deary, Whalley and Starr 2009, Deary and Johnson 2010, Gottfredson and Deary 2004, Hart et al. 2004, Johnson, Brett and Deary 2010, Johnson, Deary and Iacono 2010, McGurn, Deary and Starr 2008).

Link et al. (2008) consider the support for such conclusions and provide detailed analyses examining the support for results linking adolescent cognition and socioeconomic status to

self-reported health, chronic conditions, and mortality. In it, they find that adjusting for cognition does little to predict health above measures of education and income. However, these analyses are limited because they do not consider the role of reciprocal effects. Moreover, during the last decades there have been substantial changes in the social structure and in our understanding of the determinants of health that may further force us to question the generalizability of their results. Policymakers, researchers, and the public alike are influenced by the causal attribution of disease and as a result highlighting these mechanisms is of primary importance to socio-medical researchers (Galea and Link 2013). Providing guidance on the issue of causation, Marmot and Kivimäki (2009) note that three possible roles that cognition may play in predicting health: 1) "causation," cognition is causally associated with risk factors for disease; 2) "selection," cognition influences educational attainment, a known predictor of health behaviors and health outcomes; or 3) "indication," both good health and cognition are commonly caused by another agent. Differentiating between these three mechanisms is important because it can help us to identify tests that can usefully propel research forward.

Health behaviors

If either cognition or education causally influence health and mortality, the modification of health behaviors are likely to act as a primary causal mechanism for a number of reasons. First, health behaviors including smoking, physical inactivity, and heavy alcohol intake influence some of the most prolific causes of death from lung cancer (Doll and Hill 1956, Doll and Hill 1954, Ernster 1996, National Library of Medicine 1964, Office on Smoking and Health 2001, Pierce et al. 1998, Preston and Wang 2006) to cardiovascular disease (Alberg and Samet 2003, Ambrose and Barua 2004, Blair et al. 1996) and diabetes (Hu et al. 2001, Magliano et al. 2008), and are also associated with higher all-cause mortality (Ford et al. 2012), as well as reductions in physical functioning (Paterson and Warburton 2010) and cognitive reserve (Lee et al. 2010). Indeed, as much as 25% of the risk of mortality at midlife may be directly associated with smoking behaviors (Jha et al. 2013).

Second, while morbidity or mortality may reasonably be caused by prior individual differences in personality or genetic factors that also influence adolescent cognition (hypothesis 3 above), health behaviors are modifiable and not likely to be as strongly influenced by such factors. Indeed, health behaviors are routinely modified in whole populations as a result of policy changes to substantial gain. For example, smoking cessation has been made easier through the use of multiple forms of interventions (Centers for Disease Control 2011), and ceasing drastically reduces the risk of all-cause mortality (Jha et al. 2013).

Finally, cognitive or socioeconomic resources could both influence health through the active and intentional modification of health behaviors by social actors (e.g. individuals, families, or communities), who use resources to align health behaviors with their current understanding of disease risk and causation (Cockerham 2005). Indeed, one of the most convincing explanations provided for the association between socioeconomic factors and health is that those with more resources actively modify health-related behaviors in order to improve health and reduce the risk of disease. Interrogating why such might be the case,

Freese & Lutfey (2011) posit that those who are better educated have more control over their own actions, and have greater influence over the behavior of others in their social networks. Such internal and external control could reasonably give rise to a socioeconomic gradient in "health lifestyles" (Cockerham 2005), whereby individuals with different socioeconomic statuses engage in lifestyles that are at once derived from individual choices but also carry social signals identifying status. Indeed, those with more education tend to change some behaviors while maintaining others in order to adhere to healthier lifestyles (Margolis 2013). If cognition and/or education act to influence health behaviors, it is reasonable to think that those who are more highly educated, and among them those with the most capability, will more rapidly modify their behaviors to comply with new health information and the lifestyles of their social circles.

Counterfactual hypotheses

We hypothesize that both adolescent cognition and educational attainment may influence health behaviors in later life, but that educational attainment may be concurrently predicted by adolescent cognition (Figure 1). We suggest that the adolescent cognition is a main determinant, in conjunction with gender and parental social class, of the propensity for education. This is made evident on figure 1 by the shifting of cognitive distribution between educational outcomes, which may not entirely overlap. Nevertheless, we suggest that there will be an overlapping region (highlighted using a light-gray vertical bar) in which adolescent cognition does not determine educational attainment. In the overlapping region, we may find an association between adolescent cognition and health behaviors ($slopes_{1,2} \neq 0$), supporting the view that adolescent cognition causes behavioral change. Similarly, differences in health behaviors by education in this region similarly indicate that education is robustly associated with health behaviors absent the influence of cognitive selection. [Figure 1]

Methods

Data

We propose to jointly test the role of adolescent cognition and education on late-life health behaviors. Data adequate to answer such a question are rare and require substantial societal investments in sustained support for life course data (Welshman 2011). Uniquely, for this study we use three internationally comparable studies: the Wisconsin Longitudinal Study (US 1939), the Medical Research Council's National Survey of Health and Development (GB 1946), and the National Child Development Study (GB 1958). Each of these datasets is unique and incorporates information from a variety of individuals and over a long period of time, and thus provide a robust platform on which to examine such longitudinal hypotheses.

The US 1939 cohort began in 1957 when 10,317 Wisconsin high-school graduates, born on average in 1939, were recruited (Herd, Carr and Roan 2014). Health outcomes were measured at age 53, and educational attainment was measured at age 36. Data were linked to state-administered cognitive testing, measured at age 16. The GB 1946 cohort follows 5,362 singleton births during one week in March 1946 in Great Britain (including England, Scotland and Wales) and has been followed up regularly until most recently in 2006–2010

(Kuh et al. 2011). Adolescent cognition was measured at age 15, educational attainment at age 26, and health outcomes were measured at age 53. The GB 1958 cohort follows 16,782 individuals living in Great Britain and born during one week in March 1958 (Power and Elliott 2006). Adolescent cognition was measured at age 16, educational attainment at ages 33 and 42, and adult health behaviors at age 50. We limited analysis to those who had at least secondary-level qualifications and information on health behaviors at midlife, leaving sample sizes of 3,685, 1,555, and 5,218 for the 1939, 1946, and 1958 cohorts respectively.

Measures

Highest educational attainment was measured at ages 36, 26, and 33 in the US 1939, GB 1946, and GB 1958 cohorts respectively. Some education is mandatory in most countries; we therefore focus on the difference between university education as an educational experience that is both variable and relatively comparable between countries. For clarity, we exclude those with mixed qualifications, who received secondary qualifications but then received further sub-university education such as vocational schooling or attended university but did not graduate. Because the US 1939 cohort is limited to those who graduated from high school, we further exclude those without formal educational qualifications from analysis. While the US 1939 cohort excludes individuals who did not graduate with secondary (high school) qualifications, information is available on those who did not continue with secondary qualifications.

Adolescent cognition was measured using a general test of cognitive ability between ages 15–16. In the US 1939 cohort, cognition was measured using the Henmon-Nelson test; in the GB 1946 cohort, adolescent cognition was measured using the Alice Heim-4 test of cognitive ability, and in the GB 1958 cohort we used a scale combining reading and mathematics scores (α=0.90). In the GB 1946 cohort, general ability is strongly correlated with factor scores calculated using similar tests of reading and mathematical ability (r=0.80 at age 15). Father's social class was measured in adolescence and has been dichotomized (manual versus non-manual) to maximize comparability between countries. When missing, earlier measures of parental social class were used, if still missing we assume that this is due to a missing father and that the household was disadvantaged in a way similar to being from a lower social class; analyzing these individuals separately supports this decision.

We examine three poor health behaviors measured between ages 50–53. *Smoking* identifies whether individuals report being current smokers. *Heavy drinking* identifies individuals who reported drinking more than 3 drinks per day in men and 2 drinks among women, following the average guidance for alcohol intake in both countries (International Center for Alcohol Policies 2013)). In both GB cohorts, alcohol intake was measured by asking individuals how many glasses of beer, wine, spirits, and in the GB 1958 cohort, Sherry, over the past week; in the US 1939 cohort, we used the number of alcoholic drinks imbibed over the past month. *Physical inactivity* identifies whether individuals reported being physically active less than once per month on average in the past year.

For sensitivity analyses, we examine the possibility that incorporation of non-cognitive skills modifies these findings. Specifically, we examined the impact of incorporating measures of personality (measured at age 53 using a 29-item subscale of the big five

inventory (BFI-54) in the US 1939 cohort, at age 50 using five items from the International Personality Item Pool (extraversion, agreeableness, conscientiousness, emotional stability, and intellect) in the GB 1958 cohort, and at age 13 using the Pintner scale in the GB 1946 cohort), teacher's ratings of student excellence (at age 18 in the US 1939 cohort, teachers noted excellent students while respondents noted whether their teachers had encouraged them to attend university), high school class ranking (measured in US 1939), behavioral scores (measured by teachers and parents at age 16 using Rutter behavioral scores in the GB 1958 cohort), illness as a child (indicated by parents in the GB 1946 cohort), and birth-weight (measured directly in the GB 1946 and GB 1958 cohorts).

Analyses

We use logistic regression to estimate the effect of education (E) on each health behavior (Y), while adjusting for sex (F), parental social class (S) and adolescent cognition (C) following equation 1 below:

$$\ln(Y)=\ln\left(\frac{\pi(x)}{1-\pi(x)}\right)=\beta_0+\beta_1 E+\beta_2 S+\beta_3 F+\beta_4 C+\varepsilon \quad (1)$$

We provide odds ratios (OR), 95% confidence intervals (95% CI), and exact p-values. We use Huber-White robust standard errors. Sensitivity analyses are used to examine whether β_4 differs by educational attainment. To test the robustness of our results to model specification, we replicated analyses using propensity scores adjustment on balanced samples, which necessitated that we limit our analyses to individuals whose adolescent cognition was observed in the region of overlap. Propensity score methods provide a more robust treatment estimate, especially when selection is a potential explanation for effects (Rubin 1978), though in practice such effects rarely diverge from linear regression (Shah et al. 2005). These models estimate the propensity for educational attainment using sex, parental social class, and adolescent cognition and adjust for the propensity score using multilevel logistic modeling (Clouston 2014). We further examined the robustness of these results to subsample structures by stratifying the sample by sex, cognitive tertile, and by incorporating interactions between adolescent cognition and sex or parental social class.

Results

We begin by providing the percentage of each cohort at different educational attainment levels (Table 1), wherein we see a secular increase in the likelihood of higher education across the three cohorts. Firstly, in the US 1939 and GB 1946 cohorts we find that women, those from manual social classes, and those with lower adolescent cognition were less likely to go to university. In the GB 1946 cohort, the likelihood of going to university was much improved for women with high adolescent cognition. However, in the GB 1958 cohort we see substantial differences: those with higher adolescent cognition are much more likely to go on to university than they were previously, while the impact of parental social class and sex are substantially reduced.

Figure 2 provides the average number of respondents, expressed as a percent of the sample who were smokers, heavy drinkers, and physically inactive. During this period, people smoke less and are also less physically inactive. However, heavy drinking *increases* over time between cohorts, from very low in the US 1939 to higher in the GB 1958 cohort.

Histograms (Figure 3) show that the range and distribution of adolescent cognitive scores among university graduates differed from those with secondary qualifications. The average respondent with secondary qualifications had an adolescent cognition near sample average (−0.04), −0.85 standard deviations (SDs) lower ($p<0.001$) than those with university degrees. Potentially more importantly, those with adolescent cognitive scores 1 SDs below sample average were unlikely to earn a university degree while those with scores 1.5 SDs above sample average were very likely to receive one.

Next we estimate the association between a university degree and the likelihood of being a current smoker (Table 2). Model 1 shows a significant bivariate association between education and reduced smoking. Model 2 shows similar results for higher adolescent cognition. Assessing model fit we note that education explains more variation than adolescent cognition. Model 3 estimates the impact of education on smoking adjusting for sex and parental social class. Finally, Model 4 accounts for adolescent cognition, showing a robust effect remains for educational attainment, but further highlights an attenuation of the independent association between adolescent cognition and smoking compared to model 2. The relationship between adolescent cognition and smoking does not vary by education (average results shown in Figure 4 below).

Results from Table 3 show the impact of a university degree on heavy drinking. Models 1 and 2 show no significant bivariate associations between heavy drinking and either education or adolescent cognition, except in the GB 1946 cohort where higher educational attainment and cognition were associated with more drinking. Assessing model fit, we note that neither education nor adolescent cognition significantly predicts variation in heavy drinking, except in the 1946 cohort. Accounting for sex and parental social class (Model 3) increases the positive association from education in GB 1958, but attenuates the association between education and heavy drinking in the GB 1946 cohort. Finally, adjusting for adolescent cognition (Model 4), education begins to have an inverse association with heavy drinking in both the GB 1958 and US 1939 cohorts, but higher adolescent cognition in the 1946 cohort remains predictive of more drinking.

We next provide associations between education and physical inactivity (Table 4). Models 1 and 2 show significant protective bivariate associations between physical inactivity and both education and adolescent cognition (respectively); however, assessing model fit we note that educational attainment provides explains more variability on average than adolescent cognition. Adjusting for sex and parental social class (Model 3) does not reduce associations between education and physical inactivity. Further adjusting for adolescent cognition (Model 4) does not modify the relationship between education and physical inactivity. However, the association between adolescent cognition and physical inactivity is attenuated in all three cohorts.

Examining the overall relationship (Figure 4), allowing the association between adolescent cognition and health behaviors to vary between educational groups while noting that the region of overlap is defined between 1 SD below and 1.5 SDs above sample average, we find that there is a reduction in poor health behaviors related to educational attainment. However, in that region of overlap, there is no association between adolescent cognition and the chances of poor health behaviors. There is, however, a robust relationship between education and both smoking and physical inactivity, though there is no relationship between education and heavy drinking on average.

Sensitivity Analyses

Sensitivity analyses used adolescent cognition, gender, and parental social class to estimate the educational propensity score and to attain balance in groups we limited analysis to the region of overlap defined here. Propensity score methodology showed similar results to those shown above, specifically showing a robust link between education and smoking (OR=0.53 [0.45, 0.63], p<0.001), heavy drinking (OR=0.84 [0.70, 1.00], n.s), and physical inactivity (OR=0.54 [0.47, 0.61], p<0.001). Models including different estimates of childhood and adolescent cognition measured at ages 7/8 and age 11 in both GB cohorts find that the year that cognitive performance is measured does not generally affect the educational or cognitive estimates, however it does limit comparability with the US model. Modeling an interaction between education and adolescent cognition does not generally modify these results, with one exception: in the GB 1958 sample, university education was less predictive of physical inactivity among those with higher adolescent cognition (p=0.043); however, adjusting for such an interaction *increases* the estimated association between a university degree and physical inactivity (OR=0.48 [0.33, 0.70], p<0.001). We assessed the relative impact of incorporating information from those who with mixed qualifications or no qualifications. Increasing the breadth of the sample increased overall specificity, but did not change the conclusions. We assessed the influence of non-cognitive skills where available by using indicators of personality measured in early life, teacher's ratings of student excellence after accounting for adolescent cognition, high school ranking, behavioral scores, illness as a child, death of a parent during childhood, and birth-weight. These analyses, though not comparable between datasets, were sometimes relevant to the outcomes but did not attenuate the impact of education. Indeed, in the GB 1958 cohort the impact of education on smoking *increased* upon the inclusion of respondent's behavioral and personality scores.

Discussion

In this study, we fill gaps in the literature by examining the impact of having a university degree over secondary qualifications on three poor health behaviors (smoking, heavy drinking, and physical inactivity) at midlife (50–53 years) after adjusting for covariates, including adolescent cognition and parental social class, that predict educational attainment. We examined the association between adolescent cognition and educational attainment, and explicitly tested the hypothesis that adolescent cognition relates to health through a direct relationship with health behaviors. We used life course data from three cohorts in the United States and Great Britain. We used comparable measures and methods to find that while there

is cognitive selection into educational attainment, educational attainment independently predicted better health behaviors while adolescent cognition did not. Results support a wealth of prior research that suggests that social inequalities in health behaviors arise out of structural processes.

Limitations

Our study has a number of limitations that temper our results. First, because we harmonized the data, we were limited to using those who had at least secondary qualifications. This resulted in a substantial gain in comparability, but a loss in power and in effect size as those who left prior to gaining secondary qualifications are the most likely to engage in poorer health behaviors (Lynch, Kaplan and Salonen 1997). To ensure comparability across the cohorts, we also limited our analysis, and thus our generalizability, in the GB 1958 cohort to respondents born in Britain.

While we made substantial efforts to ensure that measures were as comparable as possible between cohorts, analyses ultimately uses information derived from variations in measurement. For example, the U.S. 1939 cohort uniquely measured the number of drinks over the past month rather than the past week. The GB 1958 cohort measured math and reading scores rather than more general measures of adolescent cognition. Such measurement problems may have introduced some variability into these results. Furthermore, the social context around drinking is fundamentally different in the U.S. and G.B., with substantial contextual differences in heavy drinking. Such results do not generally equate to uniquely higher disease rates in GB over the US (World Health Organization 2013b), suggesting that further research is needed to examine the extent to which drinking-related poor health outcomes are related to divergent drinking practices rather than to intake *per se*.

Non-cognitive skills have a substantial impact on educational attainment, later life success, and are likely to impact health (Heckman, Stixrud and Urzua 2006). We could not incorporate analyses in a comparable manner between cohorts. Sensitivity analyses incorporating such measures in non-comparable ways resulted in similar estimates for the impact of education on health behaviors, with the following exception: accounting for personality increased the effect of education on smoking and physical inactivity, though not drinking. For example, accounting for adolescent behavioral traits and personality increased in the impact of education on smoking in the GB 1958 cohort (B=−0.397, p=0.020).

Finally, while our methods provide a robust comparison across multiple datasets, we cannot fully assess the independent effects of education outside of the restrictions placed on this sample. Such limitations may lead to substantively different conclusions if those who were excluded from analysis are qualitatively different from others: such divergence may occur if abnormally high or low cognition impacts behavior differently, or if adolescent cognition is more influential when formal qualifications are lacking. Indeed, these results differ somewhat from those presented by Richards, Stephens and Mishra (2010), who find that cognition measured at age 11 is robustly associated with physical exercise at age 53 (OR=1.22, p=0.002) in the GB 1946 cohort upon adjusting for a range of early- to mid-life measures of socioeconomic status. Sensitivity analyses incorporating those with mixed

qualifications or those at extreme ends of the cognitive distribution do not change results shown. Divergent results may therefore suggest that cognition may play an increased role among those without formal educational qualifications.

Social Inequalities and Health

It is unlikely that cognition acts alone in predicting social inequalities in health. Fundamental cause theory provides a useful and practical guide to explaining how education might predict better health in a broad range of outcomes (Link and Phelan 1995, Phelan and Link 2005, Phelan, Link and Tehranifar 2010). Fundamental causes of health are causes that consistently determine who is "at risk of risks." For instance, Phelan et al. (2004) argue that when prevention is as important to disease avoidance, individuals will use *all of their resources* to avoid disease, further highlighting that resources are unevenly distributed. Supporting prior work (Link et al. 2008, Richards, Stephens and Mishra 2010), our analyses do not find that individuals "use" cognitive capabilities as a fungible resource towards improving health. Instead, our results showed that within educational groupings, adolescent cognition was generally not associated with poor health behaviors. This leads us to conclude that education is integral in promoting good health behaviors and reducing the risk of poor health behaviors in a way that is not dependent on cognitive capital (Mirowsky and Ross 2003, Reynolds and Ross 1998).

Link et al. (2008) highlighted a gap in the literature examining the association between adolescent cognition, in conjunction with educational attainment, on health behaviors. We help to fill this gap, and found that adolescent cognition is a strong predictor of the propensity for educational attainment, along with sex and parental social class (Deary et al. 2007, Deary and Johnson 2010). However, we further note that adolescent cognition was not robustly associated with health behaviors after adjusting for educational attainment. Future analyses should work to broaden these results to incorporate research into the impact of concentrated poverty and segregation, which both lead to reduced capacity in early life (Carpiano, Lloyd and Hertzman 2009, Sampson, Sharkey and Raudenbush 2008), lower educational attainment (Wodtke, Harding and Elwert 2011), and increased mortality (Hunt 2013)).

Marmot and Kivimäki (2009) provide three reasons that cognition may be related to health: causation, selection, and indication. In these analyses, we used health behaviors and sensitivity analyses to reduce the likelihood that "indication" was at play and have instead focused analysis on examining the selective and causal hypotheses. We used data adequate to the task and methodology that explicitly allows for the joint examination of these associations. We found support for the view that adolescent cognition predicted the propensity for educational attainment, and for the view that educational attainment robustly influenced health behaviors, supporting a selective interpretation of the role of adolescent cognition in determining health behaviors. However, our results showed no support for a causal interpretation of adolescent cognition in relation to health behaviors. Further research is needed that examines whether other factors, including perhaps early-life developmental factors, are associated with both improved health and higher cognition in adolescence.

Health behaviors

Alcohol intake is the third-highest risk-factor for premature death worldwide, largely because of its influence on accidental and intentional injury (World Health Organization 2013a). Results suggest that higher educational attainment and adolescent cognition had no consistent beneficial effect on the likelihood of heavy drinking, though cognition appears to have a small negative effect in one cohort and education a beneficial effect in another. Heightened use is undoubtedly associated with increased access to money to buy recreational goods. However, taking cocaine use as one example, higher adolescent cognition has emerged as a robust predictor of elevated use (Kanazawa and Hellberg 2010, White and Batty 2012), even as protective educational gradients have emerged (Miech 2008). Future analyses are needed that examine when and where alcohol intake is susceptible to educational inequalities, while explaining where it is not.

Our results may provide preliminary evidence that contextual factors, including both time and place, influence the specific types of behaviors in which people engaged and the role of educational attainment in influencing them. Specifically, there was a substantial decrease over time in the number of respondents who smoked and who were physically inactive; however, there was an increase over time in the number of people who drank. Coincident to these changes, the influence of educational attainment on smoking generally decreased over time. The number of respondents reporting regular alcohol intake was much higher in Great Britain over the U.S. Educational attainment was more influential on individual physical activity in the U.S. Contextual factors are often believed to influence the number of people who engage, these results suggest that contextual factors also influence the extent to which socioeconomic inequalities influence health behaviors.

Population health is shaped through the creative use of social, economic and educational policies that influence health and wellbeing (Clouston and Quesnel-Vallée 2012, Glymour et al. 2008, Kaplan and Lynch 2001, Link 2008). Modification of health behaviors provide the backbone of the U.S. government's *Healthy People 2020* policy (US Department of Health and Human Services 2011), the *American Heart Association's* guidelines to improve cardiovascular health (Pearson et al. 2003), and the *American Cancer Society's* guidelines for cancer prevention (American Cancer Society 2013). As we increase in our ability to prevent disease and improve health, it is likely that social inequalities will similarly increase (Phelan and Link 2005). Causal attribution of changing inequalities is of primary importance for identifying the best approaches to reduce poor health behaviors while attacking social inequalities. Our results support prior research suggesting that education plays a fundamental role in the unequal distribution of health behaviors (Masters, Hummer and Powers 2012).

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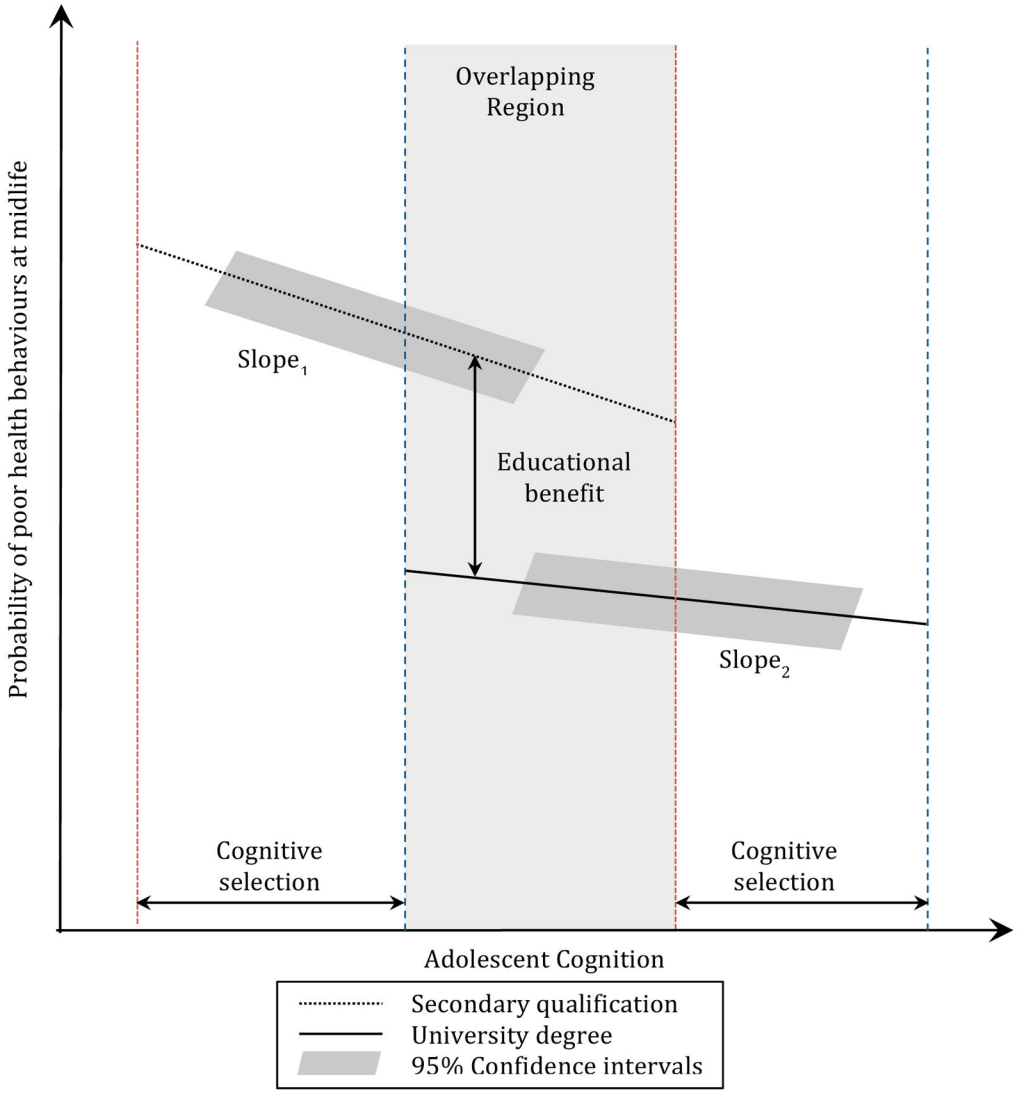

Figure 1.
Counterfactual graphical comparisons linking educational selection and educational causation

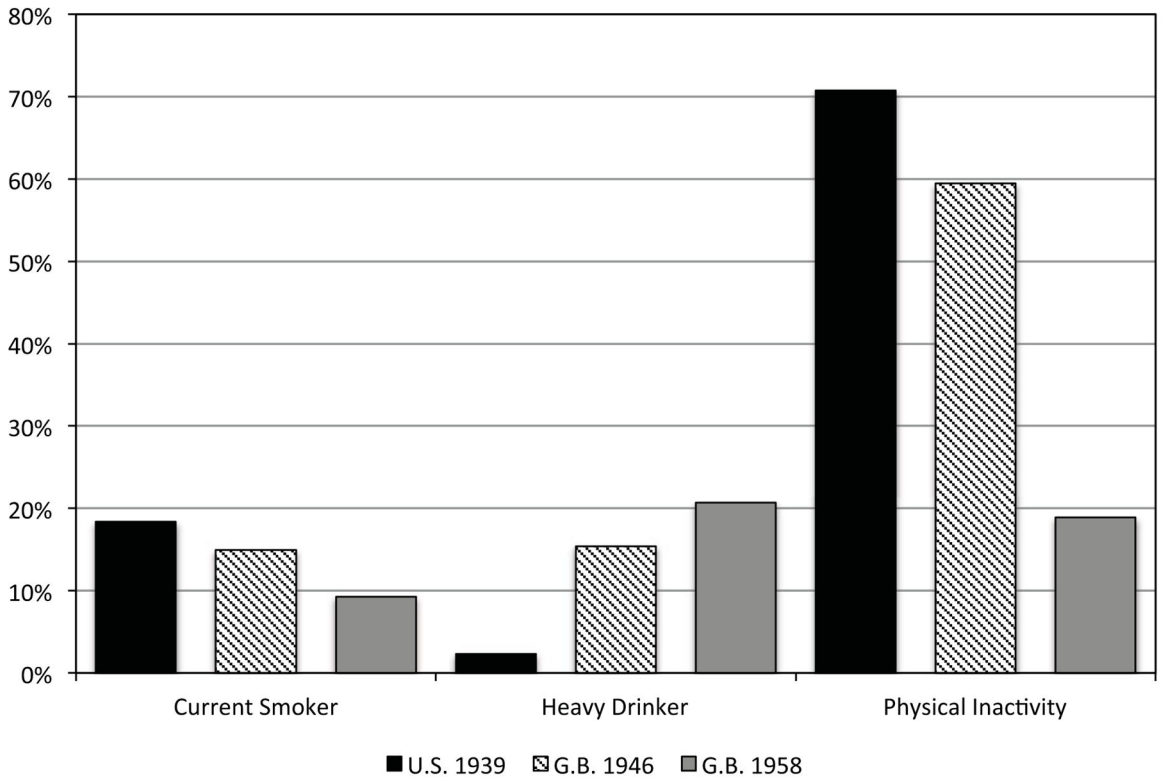

Figure 2.
Sample-specific likelihood of smoking, drinking, and physical inactivity, US 1939, GB 1946, and GB 1958

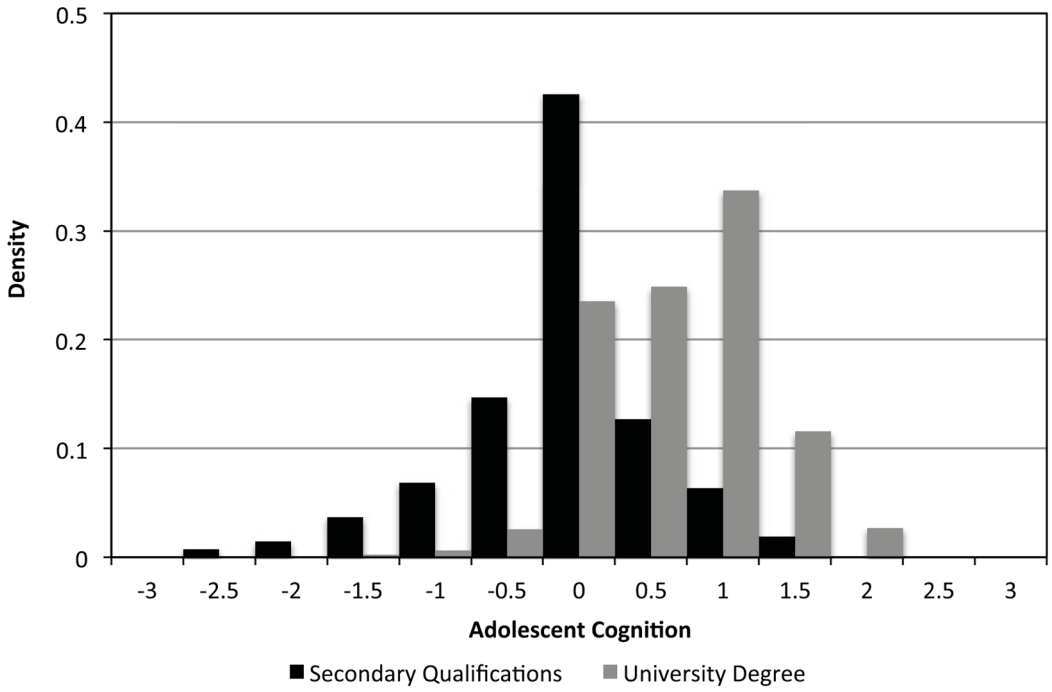

Figure 3.

Histogram of the density of adolescent cognition scores for those with a university degree in comparison to those with secondary qualifications

Source: Pooled data from US 1939, GB 1946, and GB 1958

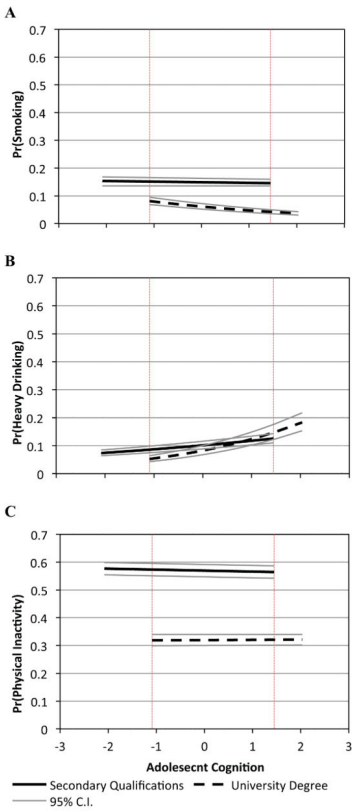

Figure 4.

Average association between adolescent cognition and the probability of **A)** smoking; **B**) heavy drinking, and **C**) physical inactivity by educational attainment with overlap in adolescent cognition marked with vertical dotted lines

Table 1

The percentage of respondents with sequential qualifications, US 1939, GB 1946, and GB 1958

	No Qualifications	Secondary Qualifications	Mixed Qualifications	University Degree
US 1939	25% *t*	53%	15%	7%
GB 1946	48%	19%	23%	9%
GB 1958	36%	35%	13%	17%

Note:

t indicates that this number is derived from external analysis of sampling structure rather than from available data (Sewell et al. 2003).

Table 2

Beta coefficients estimated using logistic regression showing the association between smoking at midlife and both education and adolescent cognition

		Model 1			Model 2			Model 3			Model 4		
Sample	**Variable Name**	**OR**	**95% CI**	**P**	**OR**	**95% CI**	**P**	**OR**	**95% CI**	**P**	**OR**	**95% CI**	**P**
US 1939	University Degree	0.40	0.31, 0.52	<0.001				0.38	0.29, 0.49	<0.001	0.38	0.29, 0.49	<0.001
	Adolescent Cognition				0.90	0.84, 0.97	0.003				1.00	0.93, 1.08	0.973
	Female							0.89	0.77, 1.02	0.094	0.89	0.77, 1.02	0.094
	Parental social class							1.09	0.92, 1.28	0.312	1.09	0.92, 1.28	0.313
	Pseudo-R^2	0.012		<0.001	0.002		0.003	0.013		<0.001	0.013		<0.001
GB 1946	University Degree	0.52	0.40, 0.68	<0.001				0.52	0.39, 0.69	<0.001	0.56	0.42, 0.76	<0.001
	Adolescent Cognition				0.72	0.60, 0.86	<0.001				0.83	0.68, 1.00	0.054
	Female							0.80	0.60, 1.05	0.105	0.80	0.61, 1.06	0.118
	Parental social class							0.75	0.57, 0.99	0.042	0.79	0.59, 1.04	0.089
	Pseudo-R^2	0.016		<0.001	0.010		0.000	0.022		<0.001	0.024		<0.001
GB 1958	University Degree	0.69	0.53, 0.88	0.003				0.66	0.51, 0.86	0.002	0.72	0.54, 0.98	0.036
	Adolescent Cognition				0.81	0.69, 0.96	0.015				0.88	0.71, 1.09	0.233
	Female							0.95	0.76, 1.19	0.658	0.93	0.75, 1.17	0.543
	Parental social class							1.10	0.87, 1.40	0.424	1.14	0.89, 1.45	0.308
	Pseudo-R^2	0.004		0.003	0.003		0.015	0.004		0.0235	0.005		0.0228

Table 3

Beta coefficients estimated using logistic regression showing the association between heavy drinking at midlife and both education and adolescent cognition

		Model 1			Model 2			Model 3			Model 4		
Sample	**Variable Name**	**OR**	**95% CI**	**P**	**OR**	**95% CI**	**P**	**OR**	**95% CI**	**P**	**OR**	**95% CI**	**P**
US 1939	University Degree	0.76	0.42, 1.36	0.353				0.57	0.29, 1.10	0.095	0.52	0.26, 1.04	0.064
	Adolescent Cognition				1.03	0.88, 1.22	0.686				1.09	0.91, 1.31	0.352
	Female							0.60	0.41, 0.89	0.010	0.60	0.41, 0.87	0.008
	Parental social class							1.48	0.96, 2.28	0.073	1.46	0.95, 2.25	0.084
	Pseudo-R^2	0.001		0.353	0.000		0.686	0.010		0.025	0.011		0.036
GB 1946	University Degree	1.50	1.11, 2.02	0.009				1.20	0.87, 1.66	0.268	1.08	0.78, 1.51	0.632
	Adolescent Cognition				1.34	1.1, 1.62	0.004				1.28	1.04, 1.58	0.017
	Female							0.41	0.30, 0.57	<0.001	0.41	0.29, 0.57	<0.001
	Parental social class							1.10	0.81, 1.48	0.553	1.03	0.75, 1.40	0.867
	Pseudo-R^2	0.006		0.009	0.007		0.004	0.032		<0.001	0.036		<0.001
GB 1958	University Degree	0.94	0.78, 1.12	0.475				0.89	0.74, 1.08	0.230	0.80	0.64, 1.00	0.046
	Adolescent Cognition				1.14	1, 1.31	0.053				1.17	0.99, 1.39	0.060
	Female							0.50	0.42, 0.60	<0.001	0.51	0.43, 0.61	<0.001
	Parental social class							0.96	0.80, 1.16	0.667	0.93	0.77, 1.12	0.444
	Pseudo-R^2	0.000		0.475	0.001		0.053	0.019		<0.001	0.020		<0.001

Table 4

Beta coefficients estimated using logistic regression showing the association between physical inactivity at midlife and both education and adolescent cognition

		Model 1			Model 2			Model 3			Model 4		
Sample	**Variable Name**	**OR**	**95% CI**	**P**	**OR**	**95% CI**	**P**	**OR**	**95% CI**	**P**	**OR**	**95% CI**	**P**
US 1939	University Degree	0.42	0.36, 0.5	<0.001				0.47	0.4, 0.55	<0.001	0.45	0.38, 0.54	<0.001
	Adolescent Cognition				0.91	0.85, 0.96	0.002				1.03	0.97, 1.11	0.335
	Female							1.27	1.12, 1.44	<0.001	1.26	1.11, 1.43	<0.001
	Parental social class							0.90	0.78, 1.03	0.128	0.89	0.78, 1.03	0.110
	Pseudo-R^2	0.018		<0.001	0.002		0.002	0.021		<0.001	0.021		<0.001
GB 1946	University Degree	0.60	0.48, 0.73	<0.001				0.62	0.5, 0.78	<0.001	0.65	0.52, 0.82	<0.001
	Adolescent Cognition				0.78	0.68, 0.9	<0.001				0.89	0.77, 1.03	0.126
	Female							0.87	0.71, 1.08	0.218	0.88	0.71, 1.09	0.229
	Parental social class							0.67	0.54, 0.83	<0.001	0.69	0.55, 0.85	0.001
	Pseudo-R^2	0.012		<0.001	0.006		0.001	0.019		<0.001	0.021		<0.001
GB 1958	University Degree	0.49	0.41, 0.6	<0.001				0.55	0.45, 0.67	<0.001	0.59	0.48, 0.74	<0.001
	Adolescent Cognition				0.69	0.6, 0.78	<0.001				0.88	0.76, 1.03	0.120
	Female							1.23	1.04, 1.45	0.016	1.21	1.02, 1.43	0.029
	Parental social class							0.73	0.6, 0.88	0.001	0.75	0.62, 0.91	0.003
	Pseudo-R^2	0.015		<0.001	0.010		<0.001	0.020		<0.001	0.021		<0.001