HHS Public Access

Author manuscript

Biodemography Soc Biol. Author manuscript; available in PMC 2021 November 22.

Published in final edited form as:

Biodemography Soc Biol. 2021 ; 66(2): 132–144. doi:10.1080/19485565.2020.1869919.

Achieved educational attainment, inherited genetic endowment for education, and obesity

Yi Li[1], **Tianji Cai**[1], **Hongyu Wang**[1], **Guang Guo**[2,3,4]

[1]Department of Sociology, University of Macau, Macau, China

[2]Department of Sociology, University of North Carolina, Chapel Hill, USA

[3]Carolina Population Center, University of North Carolina, Chapel Hill, USA

[4]Carolina Center for Genome Sciences, University of North Carolina, Chapel Hill, USA

Abstract

This study investigates two sources of education effects on obesity—achieved educational attainment and inherited genetic endowment for education. In doing so, we accomplish two goals. First, we assess the role of genetic confounding in the association between education and health. Second, we consider the heterogeneity in the extent to which genetic potential for education is realized and examine its impact on obesity. Data come from the National Longitudinal Study of Adolescent to Adult Health. Using a polygenic score approach, we find that, net of genetic confounding, holding a college degree is associated with a lower likelihood of obesity. Moreover, we find that among individuals who hold a college degree, those with a high education polygenic score (a greater genetic propensity to succeed in education) are less likely to be obese than those with a relatively low education polygenic score. However, when individuals with a high education polygenic score do not have a college degree, their risk of obesity is similar to that of non-college-educated individuals with a low education polygenic score, suggesting that the effect of genetic endowment for education on obesity is conditional on college education.

Introduction

The association between educational attainment and health has been well-documented (Mirowsky and Ross 2003). A large body of research shows that individuals with higher levels of education report fewer health risk factors (e.g., Baker et al. 2017) and lower incidence of diseases (e.g., Hayward et al. 2000), and have lower mortality (e.g., Hummer and Lariscy 2011). Education is therefore often referred to as one of the "fundamental causes" of health (Link and Phelan 1995).

Obesity is one of the most significant contributors to ill health (Kopelman 2000). In the United States, the prevalence of adult obesity has more than tripled from 12% in 1991 to

Corresponding author: Yi Li (yili@um.edu.mo) Department of Sociology, Humanities and Social Sciences Building (E21), University of Macau, Macau.

Declaration of interest statement

No conflict of interest.

42% in 2018 (Mokdad et al. 1999, Hales et al. 2020). This growing epidemic poses a grave threat to population health. Education has been shown to be associated with obesity (Cohen et al. 2013, Newton, Braithwaite, and Akinyemiju 2017). In this article, we distinguish two sources of education effects on obesity—achieved educational attainment and inherited genetic endowment for education. Separating the two distinct, yet related, education effects enables us to achieve two goals.

First, we obtain a more accurate estimate of the direct effect of educational attainment by addressing bias caused by genetic confounding. This education-obesity link can be explained by causal theories that higher educational attainment leads to better health (Cutler and Lleras-Muney 2006, Masters, Hummer, and Powers 2012, Smith 2004). This link, however, can also be explained by the theory that common confounders are responsible for causation as well as reverse causation (i.e., education is the result of health) (Fuchs 1982). Researchers argue that genetic influences are a critical confounder that can bias the estimate of the effects of achieved educational attainment on health (Lundborg, Lyttkens, and Nystedt 2016, Amin, Behrman, and Kohler 2015, Behrman et al. 2011).

Drawing on genetic data from the National Longitudinal Study of Adolescent to Adult Health, we evaluate the impact of genetic confounding with a polygenic score (PGS) approach. Using findings from genome-wide association studies (GWASs) of educational attainment (Lee et al. 2018), we construct the education PGS. The education PGS combines genome-wide information summarized by the GWASs to indicate genetic propensity to succeed in education, a potentially important source of genetic confounding.

Our second goal is to examine the interactive effect between achieved educational attainment and the education PGS on obesity. A rapidly growing body of research provides evidence that health and many other outcomes may depend on gene-environment interaction—environmental influences on an outcome can be contingent on genetic effects and vice versa (e.g., Gaydosh et al. 2018, Perry 2016, Conley et al. 2015, Simons et al. 2011, Mitchell et al. 2015, Boardman et al. 2014, Herd et al. 2019, Belsky et al. 2018, Domingue et al. 2018, Liu 2018, Wedow et al. 2018, Shanahan et al. 2008, Guo et al. 2015). By separating achieved educational attainment and the education PGS, our study brings to light the heterogeneity in educational level attained and genetic potential, while the existing literature typically compares health between different educational levels and ignores this heterogeneity.

1.1 Genetic confounding and the education polygenic score

The importance of identifying genetic confounding for causal inference has long been recognized (McGue, Osler, and Christensen 2010, Pingault et al. 2018). However, genotypes are indirectly observed in past studies. For example, twin studies exploit the fact that monozygotic twins share an identical genome and face similar conditions, such as parents and food (e.g., Lundborg 2013, Böckerman and Maczulskij 2016, Webbink, Martin, and Visscher 2010, Amin et al. 2015), but they do not use direct measures of genotype.

A contribution of this study is that we directly address genetic confounding using molecular genetic data to construct the education PGS. Complex behaviors such as education and health are affected by a very large number of social and biological factors. The effect

of a single genetic variant is small (Chabris et al. 2015). Genome-wide association studies (GWASs) identify associations between typically millions of genetic variants and a behavior. PGSs summarize the effects of genetic variants discovered by GWASs to estimate individual genetic propensities (Krapohl et al. 2018). The education PGS captures a genetic predisposition for educational attainment. The education PGS may reflect the genetics that influence the development of traits and behaviors (e.g., cognitive abilities) that lead to different achievement in school (Plomin and von Stumm 2018, Belsky et al. 2018). The education PGS is also predictive of college completion in entirely independent samples (Lee et al. 2018).

The education PGS represents a potential major source of genetic confounding in the context of educational attainment and health outcomes like obesity. Here, a confounder is a variable that affects both education and obesity. Pleiotropy can explain how the education PGS may influence obesity and education. Pleiotropy occurs when a single gene creates effects for multiple phenotypic traits. Pleiotropy is a common feature of the human genome (Sivakumaran et al. 2011). There are two forms of pleiotropy—biological and mediated (Solovieff et al. 2013). Biological pleiotropy occurs when the effects of a gene are due to biological influences. Educational attainment and obesity could be affected by common genetic influences related to traits such as cognitive performance (Lee et al. 2018, Okbay et al. 2016). Mediated pleiotropy occurs when a gene influences a second phenotype by influencing a first phenotype that affects the second phenotype. Genetic correlation between education and obesity could also result from the possibility that educational attainment affects obesity (Mirowsky and Ross 2003).

1.2 Previous gene-environment interaction research

Null or weak average effects of education may conceal large differential effects across social environments. In other words, ignoring potential interaction effects could underestimate the education effects. Gene-environment interaction research is often guided by two models. The diathesis-stress model posits that unfavorable conditions trigger or magnify genetic propensities for risky behaviors, and favorable conditions protect against genetic risk (Belsky et al. 2009). The differential susceptibility model contends that some genotypes are more sensitive to environmental influences; individuals with more sensitive genotypes have more positive outcomes in favorable conditions and in unfavorable conditions they have more negative outcomes (Ellis et al. 2011). Both models suggest interaction effects that individuals with “risky” genes and individuals without “risky” genes act differently, depending on the environment in which they live.

Recently, Barcellos et al. (2018) investigate whether the effect of education on body weight depends on the education PGS and BMI PGS. The authors use a compulsory schooling age reform that raises the minimum school-leaving age from 15 to 16 as an instrumental variable (IV). The authors find no evidence that the education PGS interacts with the IV to affect body weight. They find that for individuals with higher BMI PGSs, staying in school until age 16 leads to a reduction in body weight.

The advantage of using compulsory schooling laws is that it is an exogenous variable, but a limitation of this IV is that its impact usually concerns the effect of one or two additional

years of education in secondary school on a small, select group of students (Hamad et al. 2018, Galama, Lleras-Muney, and van Kippersluis 2018). For example, if a reform requires students to stay one more year at 9th grade, only students who intend to leave at 9th grade is affected. Students who intend to, for example, finish high school or go to college are not likely to be affected because these students would stay after 9th grade regardless of the reform. Moreover, the effect of staying one additional year in secondary school for health outcomes many years after graduation might be limited.

In this article, we focus on college education, an arguably more significant achievement than one or two additional years of education in secondary school. College education is viewed as a salient indicator of achieved status over the life course (Hout 2012). Past research has documented the health and social advantages among college-educated individuals (Dupre 2007, Schafer, Wilkinson, and Ferraro 2013, Backlund, Sorlie, and Johnson 1999, Zheng 2017, Montez, Hummer, and Hayward 2012, Schnittker 2004, Hummer and Lariscy 2011), and found that having a college degree is a stronger predictor for body weight than other degrees such as high school diploma (Benson, von Hippel, and Lynch 2018, von Hippel and Lynch 2014, Amin, Behrman, and Kohler 2015, Baum 2017). By controlling for genetic confounding with genetic data, we aim to come closer to the goal of estimating the causal effect of college education on obesity.

Materials and methods

2.1 Data

Data come from the National Longitudinal Study of Adolescent to Adult Health (Add Health). Add Health is a nationally representative survey of U.S. adolescents enrolled in grades 7 through 12 during the 1994 to 1995 school year (Harris et al. 2019). More than 20,000 respondents participated in the in-home phase of the survey at Wave I (1994–95). Respondents were then followed. Wave II, III, and IV data were collected in 1996, 2001–02, and 2008–09, respectively.

Add Health collected saliva samples for genotyping via Oragene saliva collection at Wave IV. A total of 15,072 respondents provided saliva samples and 12,058 provided consent for genotyping. After quality control, 9,975 respondents retained. We use the genotype dataset posted to dbGaP (Study Accession phs001367.v1.p1). After genotyping quality control and excluding respondents with missing data, our final sample consists of 5,105 non-Hispanic whites. We restrict the final sample to non-Hispanic whites because the GWAS results we use focus on individuals of European ancestry (Lee et al. 2018, Locke et al. 2015), and because there are known problems associated with applying PGSs in diverse ancestry groups (Martin et al. 2017). This approach is consistent with other PGS studies that limit analysis to non-Hispanic whites (e.g., Herd et al. 2019).

2.2 Measures

During Wave IV in-home survey, interviewers measured participant's weight to the nearest 1/2 pound and height to the nearest 1/8 inch. BMI is calculated as measured weight in kilograms divided by measured height in meters squared. A respondent is classified as obese

if this respondent's BMI is 30 or greater (CDC 2017). If a participant held a bachelor's degree or higher by Wave IV, this participant's educational level is coded as with a college degree. Otherwise, we code it as without a college degree.

The most recent education GWAS of 1.1 million individuals (Lee et al. 2018) identifies single nucleotide polymorphisms (SNPs) associated with educational attainment. The GWAS estimates an effect, β, for every SNP. A PGS is calculated as the weighted sum of risk alleles (the allele positively associated with educational attainment) of SNPs:

$$\mathrm{PGS}_i = \Sigma_{j=1}^{J} \beta_j G_{ij}$$

where i denotes individual and j denotes SNP, β is the coefficient for SNP j estimated by the GWAS, and G_{ij} is the number of risk alleles j for individual i.

To control for genetic confounding, we also construct the BMI PGS, derived from a large GWAS of BMI (Locke et al. 2015). The BMI PGS represents another potential major source of genetic confounding for the education-obesity link. Recall that a confounder in the present case is a variable that influences both obesity and educational attainment. The BMI PGS is associated with some causal genetic variants for body weight (Locke et al. 2015). As a result, the BMI PGS could influence obesity. We discuss above how the education PGS could influence educational attainment and obesity. For similar reasons, the BMI PGS may influence education and obesity through biological effects (e.g., genetic cognitive potential) and mediated effects (i.e., pleiotropic effects).

The education PGS and BMI PGS are constructed with PRSice software (Euesden, Lewis, and O'Reilly 2015), after removing highly correlated SNPs (SNPs having $r^2 > 0.1$ within 250k base pairs of the index SNP). The GWAS of education (Lee et al. 2018) includes the Add Health data. We use the effect, β, derived from data that exclude the Add Health data to construct the education PGS. The PGSs are standardized to have a mean of 0 and a standard deviation (SD) of 1. Table 2 shows that a 1 SD higher education PGS increases the odds of holding a college degree by 81%; a 1 SD higher BMI PGS increases the odds of being obese by 46%.

In our analysis, if an individual's education PGS is above the 50th percentile of the PGS distribution, this individual is assigned to the high education PGS group. If the score is at or below the 50th percentile, the individual is assigned to the low education PGS group. We also try the 40th and 60th percentiles as cut-off points. Results are similar. Control variables are reported in Table 1. A sense of self-control is a key benefit provided by education (Mirowsky and Ross 2003). Self-control at earlier stages of life is important for health (Moffitt et al. 2011). To measure self-control, we select items similar to previous research (Perrone et al. 2004). Physical activity and the inactivity score are constructed following previous research (Gordon-Larsen, McMurray, and Popkin 2000, Graff et al. 2016).

2.3 Analytical strategy

Given that the dependent variable is binary (obese or not), we estimate a logistic regression. First, we estimate the main effects of educational attainment and the education PGS on obesity in Equation (1).

$$\log\left(\frac{Probabilty\ of\ obesity}{1-probability\ of\ obesity}\right)=\alpha+\beta_1\text{Edu}+\beta_2\text{EduPGS}+\beta_3\text{Controls}+\beta_4\text{BMIPGS}+\beta_5\text{PCs} \quad (1)$$

where PCs represent the top 10 principal components (PCs) of genetic ancestry. PCs are included to control for population stratification (Price et al. 2006).

Next, we examine the interactive effect between college education and the education PGS in Equation (2). Because the coefficient of the interaction term, β_3 in Equation (2), cannot be readily evaluated due to the nonlinearity of logistic regression (Ai and Norton 2003), we will plot the predicted probability of being obese to better understand the interaction effect.

$$\begin{aligned}\log\left(\frac{Probabilty\ of\ obesity}{1-p\tau obability\ of\ obesity}\right)&=\alpha+\beta_1\text{Edu}+\beta_2\text{EduPGS}+\beta_3\text{Edu}\\&\times\text{EduPGS}+\beta_4\text{Controls}+\\&\beta_5\text{BMIPGS}+\beta_6\text{PCs}\end{aligned} \quad (2)$$

Results

Models 1 and 2 in Table 3 report odds ratios from the main effects models. The difference between the two models is that Model 1 excludes the education PGS and the BMI PGS whereas Model 2 includes both. We can examine the impact of the two PGSs as confounders by comparing Models 1 and 2. In Table 3 an odds ratio greater than 1 indicates a higher likelihood of being obese.

Model 1 shows that the odds of obesity are about 39% (= 1 − 0.61) lower for individuals who hold a college degree than for individuals who do not hold a college degree. The coefficient of holding a college degree is similar in Mode 2—the odds are 37% lower. Hence, evidence suggests that adding the education PGS and BMI PGS in the model may not affect the effect of college education on obesity. In Model 2, the coefficient for the education PGS is not statistically significant. A 1 SD higher BMI PGS increases the odds by about 46% (= 1.46 − 1). The coefficients of the control variables are similar between Models 1 and 2. When parental educational level is above high school, the odds of obesity are 23% lower for their offspring than for those whose parents do not finish high school. Individuals who score higher on the self-control scale, live in a smaller household, or are in the low inactivity group are generally less likely to be obese.

We now turn to the interaction effect results. Model 3 shows that holding a college degree decreases the odds of obesity by 25% (odds ratio 0.75). The coefficient for the education PGS is insignificant and the interaction term is significant. To interpret this interaction

effect, we plot it in Figure 1 below. Model 3 also shows that the coefficients of the control variables are similar to those of the main effects models.

We find evidence that the influence of the education PGS is contingent on college education. Figure 1 shows that individuals with no college degree are generally more likely to be obesity than individuals with college degree. Among the former group, individuals with a high education PGS and those with a low education PGS have a similar probability of obesity, as the two 95% confidence intervals overlap. This finding suggests that in an unfavorable environment (no college degree) the genes (education PGS) are not likely to make a difference in the risk of being obese. However, among those who hold a college degree, the high education PGS group is associated a lower probability of obesity than the low education PGS group (the two 95% confidence intervals do not overlap). The predicted probability of obesity for individuals with a high education PGS is about 0.25, whereas the predicted probability is 0.32 for individuals with a low education PGS. Therefore, in a favorable environment (college degree) individuals without “risky” genes (individuals with a high education PGS) tend to have a more positive outcome (lower risk of obesity) than individuals with “risky” genes (individuals with a low education PGS).

Discussion

In this article, we find that, net of the education PGS and BMI PGS, holding a college is associated with a lower probability of being obese. Genetic influences do not appear to confound the effect of college education. This finding supports the idea that educational attainment is a cause of health. We also find an interaction effect between college education and the education PGS. The education PGS may indicate intrinsic academic and cognitive abilities (Okbay et al. 2016), and be related to the development of brain and central nervous system (Lee et al. 2018). A relatively high education PGS might indicate stronger deliberative abilities, and enable individuals to adopt a healthy lifestyle through calculations about the consequences of life choices. Consequently, in the favorable environment (college education) individuals with high education PGSs might enjoy more benefits including good health provided by “learned effectiveness” (Mirowsky and Ross 2005) than those with relatively low education PGSs. However, in the unfavorable environment (no college education) it is plausible that individuals with high education PGSs face limited opportunities to gain knowledge about healthy lifestyles or develop skills and abilities that would help them be rational about life choices. Without much “learned effectiveness,” the high education PGS group might exhibit levels of knowledge and capability similar to those of the low education PGS group in the unfavorable environment. Future research might test this possibility and explore other explanations.

The PGS approach has been extensively used in the social and biological sciences, but some caveats apply. The associations between genetic variants and education discovered in the GWASs reflect direct genetic influences on education, but they also reflect indirect influences such as genetic nurturing (Kong et al. 2018). In addition, within-family association between the education PGS and education is weaker than that of general population (Lee et al. 2018). This finding suggests that the GWAS education effects may be overestimated because educational attainment is also influenced by family environments.

Most GWASs analyze samples of individuals of predominantly European genetic ancestry (e.g., Locke et al. 2015, Lee et al. 2018, Okbay et al. 2016, Rietveld et al. 2013). When GWASs include more non-European ancestry groups in the future, the PGS approach can be expanded.

Our estimates are obtained after controlling for genetic confounding, but another caveat—for the present study, twin and fixed-effects models, the IV strategy, and observational studies more broadly—is that unobserved confounders may affect results. For example, our finding that the education PGS predicts a lower probability of obesity in the college education group may be caused by a confounder that makes some individuals with a high education PGS go to college, and, at the same time, less likely to be obese than those who have a low education PGS. In twin studies, monozygotic twins have different non-shared environments (e.g., different experiences in adult life). Non-shared environments can be a confounder (McGue, Osler, and Christensen 2010). For the IV strategy, there could be confounders like large secular improvements in education over the years (Galama, Lleras-Muney, and van Kippersluis 2018).

Estimating the causal effects of education on health is a challenging task. This article is an example of how genetic data can be a useful tool to improve our understanding of education's role in population health. As we learn more about the genetics of educational attainment and obesity, we can explore new ways to unpack the dynamic and complicated processes that generate the health disparity.

Acknowledgments

This research uses data from Add Health, a program project directed by Kathleen Mullan Harris and designed by J. Richard Udry, Peter S. Bearman, and Kathleen Mullan Harris at the University of North Carolina at Chapel Hill, and funded by grant P01-HD31921 from the Eunice Kennedy Shriver National Institute of Child Health and Human Development, with cooperative funding from 23 other federal agencies and foundations. Information on how to obtain the Add Health data files is available on the Add Health website (http://www.cpc.unc.edu/addhealth). No direct support was received from grant P01-HD31921 for this analysis. We gratefully acknowledge support to Yi Li from the University of Macau (SRG2017-00108-FSS). We thank Brandt Levitt and Aysu Okbay for their help with genotype data preparation.

References

Ai, Chunrong, and Norton Edward C. 2003. "Interaction terms in logit and probit models." Economics letters 80 (1):123–129.

Amin, Vikesh, Behrman Jere R, and Kohler Hans-Peter. 2015. "Schooling has smaller or insignificant effects on adult health in the US than suggested by cross-sectional associations: new estimates using relatively large samples of identical twins." Social science & medicine 127:181–189. [PubMed: 25110343]

Amin, Vikesh, Behrman Jere R, Kohler Hans-Peter, Xiong Yanyan, and Zhang Junsen. 2015. "Causal inferences: Identical twins help and clarity about necessary assumptions is critical." Social science & medicine 127 (20):1e202. [PubMed: 25557617]

Backlund, Eric, Sorlie Paul D, and Johnson Norman J. 1999. "A comparison of the relationships of education and income with mortality: the National Longitudinal Mortality Study." Social science & medicine 49 (10):1373–1384. [PubMed: 10509827]

Baker, David P, Smith William C, Muñz Ismael G, Jeon Haram, Fu Tian, Leon Juan, Salinas Daniel, and Horvatek Renata. 2017. "The population education transition curve: Education gradients across population exposure to new health risks." Demography 54 (5):1873–1895. [PubMed: 28875332]

Barcellos, Silvia H, Carvalho Leandro S, and Turley Patrick. 2018. "Education can reduce health differences related to genetic risk of obesity." Proceedings of the National Academy of Sciences 115 (42):E9765–E9772.

Baum Charles L. 2017. "The effects of college on weight: examining the “freshman 15” myth and other effects of college over the life cycle." Demography 54 (1):311–336. [PubMed: 27928734]

Behrman, Jere R, Kohler Hans-Peter, Jensen Vibeke Myrup, Pedersen Dorthe, Petersen Inge, Bingley Paul, and Christensen Kaare. 2011. "Does more schooling reduce hospitalization and delay mortality? New evidence based on Danish twins." Demography 48 (4):1347–1375. [PubMed: 21842327]

Belsky, Daniel W, Domingue Benjamin W, Wedow Robbee, Arseneault Louise, Boardman Jason D, Caspi Avshalom, Conley Dalton, Fletcher Jason M, Freese Jeremy, and Herd Pamela. 2018. "Genetic analysis of social-class mobility in five longitudinal studies." Proceedings of the National Academy of Sciences 115 (31):E7275–E7284.

Belsky Jay, Jonassaint Charles, Pluess Michael, Stanton Michael, Brummett Beverly, and Williams Redford. 2009. "Vulnerability genes or plasticity genes?" Molecular psychiatry 14 (8):746–754. [PubMed: 19455150]

Benson, Rebecca, von Hippel Paul T, and Lynch Jamie L. 2018. "Does more education cause lower BMI, or do lower-BMI individuals become more educated? Evidence from the National Longitudinal Survey of Youth 1979." Social science & medicine 211:370–377. [PubMed: 28366246]

Boardman, Jason D, Domingue Benjamin W, Blalock Casey L, Haberstick Brett C, Harris Kathleen Mullan, and McQueen Matthew B. 2014. "Is the gene-environment interaction paradigm relevant to genome-wide studies? The case of education and body mass index." Demography 51 (1):119–139. [PubMed: 24281739]

Böckerman, Petri, and Maczulskij Terhi. 2016. "The education-health nexus: fact and fiction." Social science & medicine 150:112–116. [PubMed: 26745865]

CDC. 2017. "Overweight and Obesity - Adult Overweight and Obesity." https://www.cdc.gov/obesity/adult/defining.html.

Chabris, Christopher F, Lee James J, Cesarini David, Benjamin Daniel J, and Laibson David I. 2015. "The fourth law of behavior genetics." Current directions in psychological science 24 (4):304–312. [PubMed: 26556960]

Cohen, Alison K, Rai Manisha, Rehkopf David H, and Abrams Barbara. 2013. "Educational attainment and obesity: a systematic review." Obesity reviews 14 (12):989–1005. [PubMed: 23889851]

Conley, Dalton, Domingue Benjamin W, Cesarini David, Dawes Christopher, Rietveld Cornelius A, and Boardman Jason D. 2015. "Is the effect of parental education on offspring biased or moderated by genotype?" Sociological Science 2:82. [PubMed: 29051911]

Cutler, David M, and Lleras-Muney Adriana. 2006. Education and health: evaluating theories and evidence. National bureau of economic research.

Domingue, Benjamin W, Belsky Daniel W, Fletcher Jason M, Conley Dalton, Boardman Jason D, and Harris Kathleen Mullan. 2018. "The social genome of friends and schoolmates in the National Longitudinal Study of Adolescent to Adult Health." Proceedings of the National Academy of Sciences 115 (4):702–707.

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):1–15. [PubMed: 17476920]

Ellis, Bruce J, Boyce W Thomas, Belsky Jay, Bakermans-Kranenburg Marian J, and Van IJzendoorn Marinus H. 2011. "Differential susceptibility to the environment: An evolutionary—neurodevelopmental theory." Development and psychopathology 23 (1):7–28. [PubMed: 21262036]

Euesden, Jack, Lewis Cathryn M, and O’Reilly Paul F. 2015. "PRSice: polygenic risk score software." Bioinformatics 31 (9):1466–1468. [PubMed: 25550326]

Fuchs, Victor R 1982. "Time Preference and Health: An Exploratory Study." In Economic Aspects of Health, 93–120. University of Chicago Press.

Galama, Titus J, Lleras-Muney Adriana, and van Kippersluis Hans. 2018. The Effect of Education on Health and Mortality: A Review of Experimental and Quasi-Experimental Evidence. National Bureau of Economic Research.

Gaydosh Lauren, Belsky Daniel W, Domingue Benjamin W, Boardman Jason D, and Harris Kathleen Mullan. 2018. "Father absence and accelerated reproductive development in non-Hispanic White women in the United States." Demography 55 (4):1245–1267. [PubMed: 29978338]

Gordon-Larsen, Penny, McMurray Robert G, and Popkin Barry M. 2000. "Determinants of adolescent physical activity and inactivity patterns." Pediatrics 105 (6):e83–e83. [PubMed: 10835096]

Graff, Misa, Richardson AS, Young Kristin L, Mazul AL, Highland Heather, North Kari E, Mohlke Karen L, Lange Leslie A, Lange Ethan M, and Harris Katherine M. 2016. "The interaction between physical activity and obesity gene variants in association with BMI: Does the obesogenic environment matter?" Health & place 42:159–165. [PubMed: 27771443]

Guo, Guang, Li Yi, Wang Hongyu, Cai Tianji, and Duncan Greg J. 2015. "Peer influence, genetic propensity, and binge drinking: a natural experiment and a replication." American journal of sociology 121 (3):914–954.

Hales, Craig M, Carroll Margaret D, Fryar Cheryl D, and Ogden Cynthia L. 2020. "Prevalence of obesity and severe obesity among adults: United States, 2017–2018."

Hamad, Rita, Elser Holly, Tran Duy C, Rehkopf David H, and Goodman Steven N. 2018. "How and why studies disagree about the effects of education on health: A systematic review and meta-analysis of studies of compulsory schooling laws." Social science & medicine 212:168–178. [PubMed: 30036767]

Harris, Mullan Kathleen, Halpern Carolyn Tucker, Whitsel Eric A, Hussey Jon M, Killeya-Jones Ley A, Tabor Joyce, and Dean Sarah C. 2019. "Cohort profile: The national longitudinal study of adolescent to adult health (add health)." International journal of epidemiology 48 (5):1415–1415k. [PubMed: 31257425]

Hayward, Mark D, Miles Toni P, Crimmins Eileen M, and Yang Yu. 2000. "The significance of socioeconomic status in explaining the racial gap in chronic health conditions." American sociological review:910–930.

Herd, Pamela, Freese Jeremy, Sicinski Kamil, Domingue Benjamin W, Harris Kathleen Mullan, Wei Caiping, and Hauser Robert M. 2019. "Genes, Gender Inequality, and Educational Attainment." American sociological review 84 (6):1069–1098.

Hout, Michael. 2012. "Social and economic returns to college education in the United States." Annual review of sociology 38:379–400.

Hummer, Robert A, and Lariscy Joseph T. 2011. "Educational attainment and adult mortality." In International handbook of adult mortality, 241–261. Springer.

Kong, Augustine, Thorleifsson Gudmar, Frigge Michael L, Vilhjalmsson Bjarni J, Young Alexander I, Thorgeirsson Thorgeir E, Benonisdottir Stefania, Oddsson Asmundur, Halldorsson Bjarni V, and Masson Gisli. 2018. "The nature of nurture: Effects of parental genotypes." science 359 (6374):424–428. [PubMed: 29371463]

Kopelman, Peter G 2000. "Obesity as a medical problem." Nature 404 (6778):635–643. [PubMed: 10766250]

Krapohl, Eva, Patel Hamel, Newhouse Stephen, Curtis Charles J, von Stumm Sophie, Dale Philip S, Zabaneh Delilah, Breen Gerome, O'Reilly Paul F, and Plomin Robert. 2018. "Multi-polygenic score approach to trait prediction." Molecular psychiatry 23 (5):1368–1374. [PubMed: 28785111]

Lee, James J, Wedow Robbee, Okbay Aysu, Kong Edward, Maghzian Omeed, Zacher Meghan, Nguyen-Viet Tuan Anh, Bowers Peter, Sidorenko Julia, and Linnér Richard Karlsson. 2018. "Gene discovery and polygenic prediction from a 1.1-million-person GWAS of educational attainment." Nature Genetics 50 (8):1112. [PubMed: 30038396]

Link, Bruce G, and Phelan Jo. 1995. "Social conditions as fundamental causes of disease." Journal of health and social behavior:80–94. [PubMed: 7560851]

Liu, Hexuan. 2018. "Social and genetic pathways in multigenerational transmission of educational attainment." American sociological review 83 (2):278–304.

Locke, Adam E, Kahali Bratati, Berndt Sonja I, Justice Anne E, Pers Tune H, Day Felix R, Powell Corey, Vedantam Sailaja, Buchkovich Martin L, and Yang Jian. 2015. "Genetic studies of body

mass index yield new insights for obesity biology." Nature 518 (7538):197–206. [PubMed: 25673413]

Lundborg, Petter. 2013. "The health returns to schooling—what can we learn from twins?" Journal of population economics 26 (2):673–701.

Lundborg, Petter, Hampus Lyttkens Carl, and Nystedt Paul. 2016. "The effect of schooling on mortality: New evidence from 50,000 Swedish twins." Demography 53 (4):1135–1168. [PubMed: 27393233]

Martin, Alicia R, Gignoux Christopher R, Walters Raymond K, Wojcik Genevieve L, Neale Benjamin M, Gravel Simon, Daly Mark J, Bustamante Carlos D, and Kenny Eimear E. 2017. "Human demographic history impacts genetic risk prediction across diverse populations." The American Journal of Human Genetics 100 (4):635–649. [PubMed: 28366442]

Masters, Ryan K, Hummer Robert A, and Powers Daniel A. 2012. "Educational differences in US adult mortality: A cohort perspective." American sociological review 77 (4):548–572. [PubMed: 25346542]

McGue, Matt, Osler Merete, and Christensen Kaare. 2010. "Causal inference and observational research: The utility of twins." Perspectives on Psychological Science 5 (5):546–556. [PubMed: 21593989]

Mirowsky, John, and Ross Catherine E. 2003. Education, Social Status, and Health: Transaction Publishers.

Mirowsky, John, and Ross Catherine E. 2005. "Education, learned effectiveness and health." London Review of Education 3 (3):205–220.

Mitchell, Colter, McLanahan Sara, Notterman Daniel, Hobcraft John, Brooks-Gunn Jeanne, and Garfinkel Irwin. 2015. "Family structure instability, genetic sensitivity, and child well-being." American journal of sociology 120 (4):1195–1225.

Moffitt, Terrie E, Arseneault Louise, Belsky Daniel, Dickson Nigel, Hancox Robert J, Harrington HonaLee, Houts Renate, Poulton Richie, Roberts Brent W, and Ross Stephen. 2011. "A gradient of childhood self-control predicts health, wealth, and public safety." Proceedings of the National Academy of Sciences 108 (7):2693–2698.

Mokdad, Ali H, Serdula Mary K, Dietz William H, Bowman Barbara A, Marks James S, and Koplan Jeffrey P. 1999. "The spread of the obesity epidemic in the United States, 1991–1998." JAMA 282 (16):1519–1522. [PubMed: 10546690]

Montez, Karas Jennifer, Hummer Robert A, and Hayward Mark D. 2012. "Educational attainment and adult mortality in the United States: A systematic analysis of functional form." Demography 49 (1):315–336. [PubMed: 22246797]

Newton, Suzy, Braithwaite Dejana, and Akinyemiju Tomi F. 2017. "Socio-economic status over the life course and obesity: Systematic review and meta-analysis." PloS one 12 (5).

Okbay, Aysu, Beauchamp Jonathan P, Fontana Mark Alan, Lee James J, Pers Tune H, Rietveld Cornelius A, Turley Patrick, Chen Guo-Bo, Emilsson Valur, and Fleur S, Meddens W. 2016. "Genome-wide association study identifies 74 loci associated with educational attainment." Nature 533 (7604):539–542. [PubMed: 27225129]

Perrone, Dina, Sullivan Christopher J, Pratt Travis C, and Margaryan Satenik. 2004. "Parental efficacy, self-control, and delinquency: A test of a general theory of crime on a nationally representative sample of youth." International Journal of Offender Therapy and Comparative Criminology 48 (3):298–312. [PubMed: 15142309]

Perry, Brea L 2016. "Gendering genetics: biological contingencies in the protective effects of social integration for men and women." American journal of sociology 121 (6):1655–1696.

Pingault, Jean-Baptiste, O'reilly Paul F, Schoeler Tabea, Ploubidis George B, Rijsdijk Frühling, and Dudbridge Frank. 2018. "Using genetic data to strengthen causal inference in observational research." Nature Reviews Genetics 19 (9):566–580.

Plomin, Robert, and Stumm Sophie von. 2018. "The new genetics of intelligence." Nature Reviews Genetics 19 (3):148.

Price, Alkes L, Patterson Nick J, Plenge Robert M, Weinblatt Michael E, Shadick Nancy A, and Reich David. 2006. "Principal components analysis corrects for stratification in genome-wide association studies." Nature Genetics 38 (8):904–909. [PubMed: 16862161]

Rietveld, Cornelius A, Medland Sarah E, Derringer Jaime, Yang Jian, Esko Tõnu, Martin Nicolas W, Westra Harm-Jan, Shakhbazov Konstantin, Abdellaoui Abdel, and Agrawal Arpana. 2013. "GWAS of 126,559 individuals identifies genetic variants associated with educational attainment." science 340 (6139):1467–1471. [PubMed: 23722424]

Schafer, Markus H, Wilkinson Lindsay R, and Ferraro Kenneth F. 2013. "Childhood (mis) fortune, educational attainment, and adult health: Contingent benefits of a college degree?" Social forces 91 (3):1007–1034.

Schnittker, Jason. 2004. "Education and the changing shape of the income gradient in health." Journal of health and social behavior 45 (3):286–305. [PubMed: 15595508]

Shanahan, Michael J, Vaisey Stephen, Erickson Lance D, and Smolen Andrew. 2008. "Environmental contingencies and genetic propensities: Social capital, educational continuation, and dopamine receptor gene DRD2." American journal of sociology 114 (S1):S260–S286.

Simons, Ronald L, Lei Man Kit, Beach Steven R H, Brody Gene H, Philibert Robert A, and Gibbons Frederick X. 2011. "Social environment, genes, and aggression: Evidence supporting the differential susceptibility perspective." American sociological review 76 (6):883–912.

Sivakumaran, Shanya, Agakov Felix, Theodoratou Evropi, Prendergast James G, Zgaga Lina, Manolio Teri, Rudan Igor, McKeigue Paul, Wilson James F, and Campbell Harry. 2011. "Abundant pleiotropy in human complex diseases and traits." The American Journal of Human Genetics 89 (5):607–618. [PubMed: 22077970]

Smith, James P 2004. "Unraveling the SES: health connection." Population and development review 30:108–132.

Solovieff, Nadia, Cotsapas Chris, Lee Phil H, Purcell Shaun M, and Smoller Jordan W. 2013. "Pleiotropy in complex traits: challenges and strategies." Nature Reviews Genetics 14 (7):483–495.

Hippel von, Paul T, and Lynch Jamie L. 2014. "Why are educated adults slim—Causation or selection?" Social science & medicine 105:131–139. [PubMed: 24524908]

Webbink, Dinand, Martin Nicholas G, and Visscher Peter M. 2010. "Does education reduce the probability of being overweight?" Journal of health economics 29 (1):29–38. [PubMed: 20116872]

Wedow, Robbee, Zacher Meghan, Huibregtse Brooke M, Harris Kathleen Mullan, Domingue Benjamin W, and Boardman Jason D. 2018. "Education, smoking, and cohort change: Forwarding a multidimensional theory of the environmental moderation of genetic effects." American sociological review 83 (4):802–832. [PubMed: 31534265]

Zheng, Hui. 2017. "Why does college education matter? Unveiling the contributions of selection factors." Social science research 68:59–73. [PubMed: 29108600]

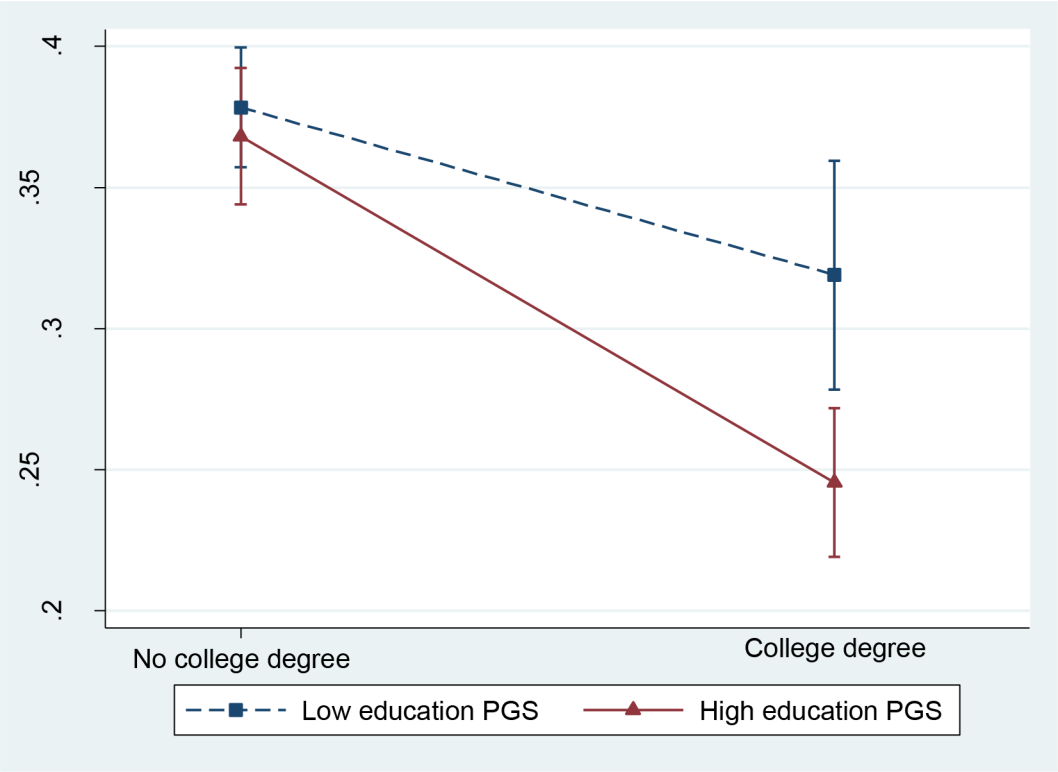

Figure 1.
Predicted probability of obesity by college education and the education polygenic score (PGS), with 95% confidence interval
Note: Results are derived from Model 3 of Table 3.

Table 1.

Sample characteristics

Variable	Description and coding	Percentage/ mean (S.D.)
Obesity	BMI > 30, Wave IV	34.12%
College degree	Holding a bachelor's degree or higher by Wave IV	32.43%
Age	Age, Wave IV	28.37
		(1.75)
Sex	Female = 1, male = 0	53.42%
Parental education	Highest degree attained by parents, Wave I	
	More than high school	60.80%
	High school	31.26%
	Below high school	7.93%
Biological parents	Lived with biological father and mother, Wave I	58.77%
PVT score	Verbal IQ score, Wave I	105.09 (11.82)
Self-control	Sum of responses to the following questions, Wave I. Higher scores indícate	8.25
	higher levels of self-control. Score ranges from 1 to 19. 1. had trouble getting along with teachers; 2. had trouble paying attention in school; 3. had trouble getting homework done; 4. had trouble keeping your mind on what you were doing; and 5. felt like doing everything just about right.	(2.25)
Household size	The number of individuals living with the respondent, Wave IV	3.07 (1.42)
Employment	Employed: worked for pay at least 10 hours a week, Wave IV	81.52%
Marital status	Married = 1, not married = 0, Wave IV	50.40%
Physical activity[a]	Sum of frequency occurrences (bouts) of the following moderate to vigorous physical activities (MVPA) in the past seven days, Wave IV bicycle, skateboard, dance, hike, hunt, yard work, roller blade, roller skate, downhill ski, snow board, racquet sports, aerobics, football, soccer, basketball, lacrosse, rugby, field hockey, ice hockey, run, wrestle, swim, cross-country ski, cycle race, martial arts, gymnastics, weight lifting, strength training, golf, fish, bowl, softball, baseball, walk for exercise	
	0 bouts	13.40%
	1 to 4 bouts	32.20%
	5 bouts or more	54.40%
Inactivity score[a]	In the past seven days, hours spent on watching television/videos, playing video/computer games, and using a computer, Wave IV	
	Low: 10 hours or below	46.97%
	Mediumml: 11 to 24 hours	34.67%
	High: 25 hours or more	18.35%
N		5,105

Table 2.

Polygenic scores (PGSs) predicting respective phenotypes, logistic regressions

	Dependent variable	Independent variable	Odds ratio	N
Model 1	College enrollment	Education PGS	1.81***	5,105
Model 2	Obesity	BMI PGS	1 46***	5,105

Note: All models control for age, sex, and the first 10 principal components to address population stratification.

* $p < .05$

** $p < .01$

*** $p < .001$ (two-tailed tests).

Table 3.

College education and the education polygenic score (PGS) predicting obesity: Odds ratios from logistic regressions

	Model 1	Model 2	Model 3
Independent variable			
College degree vs. no college degree	0.61***	0.62***	0.75*
High education PGS vs. low education PGS	------	0.95	1.04
College degree × High education PGS	------	------	0.71*
Age	1.03	1.03	1.03
Female vs. male	0.97	0.98	0.98
Parental education			
High school vs. below high school	0.97	0.96	0.95
More than high school vs. below high school	0.77*	0.76*	0.75*
Lived with two biological parents vs. no	1.03	1.05	1.04
Verbal IQ (PVT) score	1.00	1.00	1.00
Self-control	0.94***	0.94***	0.94***
Employed vs. unemployed	1.04	1.06	1.06
Married vs. unmarried	1.13	1.13	1.12
Household size	1.07**	1.07**	1.07**
Weekly physical activity[a]			
1 to 4 bouts vs. 0 bouts	0.88	0.88	0.88
5 bouts or more vs. 0 bouts	1.02	1.04	1.03
Composite inactivity score[a]			
Medium vs. low	1.40***	1.43***	1.43***
High vs. low	1.50***	1.54***	1.54***
BMI PGS	------	1.46***	1.47***
−2 Log-likelihood	6553.29	6553.29	6553.29
LR Chi-square	194.99***	378.85***	377.06***
N	5,105	5,105	5,105

Note: An odds ratio greater than 1 indicates a higher likelihood of being obese.

* $p < .05$

** $p < .01$

*** $p < .001$ (two-tailed tests)