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ISSN: 2365-9793

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ABSTRACT

Understanding the Educational Attainment Polygenic Index and Its Interactions with SES in Determining Health in Young Adulthood*

Based on the sample of The National Longitudinal Study of Adolescent to Adult Health (Add Health), we investigate the formation of health capital and the role played by genetic endowments, parental SES, and education. To measure genetic endowments we take advantage of the new availability of quality polygenic indexes (PGIs), which are optimally-weighted summaries of individual molecular genetic data. Our main focus is on the Educational Attainment Polygenic Index (EA PGI), which is designed to predict the highest level of education achieved in life. We find that the EA PGI demonstrates stronger effects on health and health behaviors for subjects with high parental socioeconomic status (SES). These effects are only partially explained by education as a mechanism. We provide suggestive evidence for the mechanisms behind estimated relationships, including early health, skills, and the parents' and child's own attitudes towards education, as well as outcomes related to occupation and wealth. We also show that a strong association between education and health survives controlling for a large set of PGIs that proxy health, skills, and home environment, with only a modest reduction in regression coefficients despite controlling for major expected confounders. This result informs the ongoing debate about the causal relationship between education and health and the confounders behind the education-health gradient.

JEL Classification: I12, I14, I24, J24
Keywords: health, health behaviors, Polygenic Index (PGI), Polygenic Score (PGS), Educational Attainment, parental socioeconomic status (SES), child development, education, mediators, Add Health data

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* A version of this paper was presented to Brown Bag Seminar of The Board of Governors of the Federal Reserve System; Economics Department Seminar at Virginia Commonwealth University; The 4th Annual Southeastern Micro Labor Workshop at The University of South Carolina in Columbia, USA; 21st IZA/SOLE Transatlantic Meeting of Labor Economists (TAM); The Economics Department Seminar of St. Lawrence University, USA; The Economics Department Seminar of Diego Portales University, Santiago, Chile; The Economics Department Seminar of the Rensselaer Polytechnic Institute, USA; The Health Economics Group of the 2020 NBER Summer Institute; The 28th European Workshop on Econometrics and Health Economics in Leuven, Belgium, and the 89th Annual Southern Economic Association Meeting in Fort Lauderdale, Florida, USA. The authors are grateful to the participants of these conferences and seminars for useful suggestions and stimulating discussions. We thank Govert Bijwaard, Gabriella Conti, Michael Darden, Matthew Harris, Ian M. Schmutte, two anonymous JHR referees for their productive comments. William Anderson, Jack Buckman, Owen Haas, Isabel Haber, Zehra Sahin Ilkorkor, Maxwell Sacher, Katia Savelyeva, and Nathan Troutman provided excellent proofreading. Bolyard and Savelyev benefited from the NSF 1460003 grant and research support from The College ofWilliam & Mary and Virginia Commonwealth University. 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 Eunice Kennedy Shriver National Institute of Child Health and Human Development (NICHD), with cooperative funding from 23 other federal agencies and foundations. Add Health GWAS data were funded by NICHD Grants R01 HD073342 (Harris) and R01 HD060726 (Harris, Boardman, and McQueen). Investigators thank the staff and participants of the Add Health Study for their important contributions. The views expressed in this paper are those of the authors and do not necessarily reflect the views of the funders.

1 Introduction

This paper is concerned with understanding the determinants of human capital formation, with a focus on health capital. We take advantage of modern advances in molecular genetic measurements and study how genetic endowments are related to health and health behaviors, how these relationships depend on parental socioeconomic status, and what the possible mechanisms behind these relationships are. In addition, we inform the ongoing debate about the relationship between education and health. Education and health are highly correlated, but education is endogenous, with a significant positive selection into education expected. However, little is known about the determinants of this selection and its magnitude. We rely on molecular genetic proxies of endowments and find novel results.

We measure genetic endowments using Polygenic Indexes (PGIs), which are optimally-weighted sums of a person's molecular genetic variants.[1] Weights put on genetic variants in a PGI depend on which particular life outcome a PGI is designed to predict. Our key PGI of interest is the Educational Attaintment PGI (EA PGI), which is designed to predict the total years of formal education. We also utilize a large number of PGIs that proxy genetic endowments for various aspects of general and mental health. PGIs are well-established and useful because they are highly predictive of life outcomes, and results based on them are typically replicable when tested using different datasets (Benjamin et al., 2012).

We use data from The National Longitudinal Study of Adolescent to Adult Health (Add Health), which follows a cohort of individuals from middle or high school through young adulthood. Add Health is considered nationally representative for the USA (Harris, 2013). We study a variety of *health outcomes*, which are self-reported general health,

[1]The term "Polygenic Index" (PGI) refers to exactly the same index as the earlier-established terms "Polygenic Score" (PGS) and "Polygenic Risk Score" (PRS). This new term is used increasingly often because it is less likely to give the impression of a value judgment where one is not intended (Becker et al., 2021).

depression, and obesity. We also study *health behaviors*, which are risky drinking of alcohol, marijuana use, smoking cigarettes, and doing no physical exercise. All these variables correspond to ages 24–32. We refer to them collectively as *health-related outcomes* in young adulthood.

We offer two contributions. First, motivated by the growing literature on gene-by-environment interactions, we investigate how parental socioeconomic status (SES) interacts with the endowments for education measured by the EA PGI. This allows us to better understand the process through which socio-economic environments can affect health capital formation. We demonstrate a novel interaction effect: the conditional association between EA PGI and health-related variables is strong and positive for subjects with high parental SES but low or nonexistent for low-SES subjects. We thus add new results to the growing literature on what Fletcher (2019) calls *environmental bottlenecks*: an adverse environment can limit the benefits of productive genetic endowments or the remediation of harmful ones (e.g., Bierut et al., 2018; Scarr-Salapatek, 1971).

Similar gene-by-SES interactions have been established for the effect of EA PGI on education (Fletcher, 2019; Papageorge and Thom, 2020; Ronda et al., 2020). In this paper we replicate this important result for the AddHealth data. This result suggests another hypothesis: whether all of our estimated conditional associations between EA PGI and health-related outcomes, including the interaction between AE PGI and SES, are fully driven by education as a mechanism. We test and reject this hypothesis. We find that our estimated associations between EA PGI and health, as well as the corresponding interactions with SES, are only partially explained by education. We also perform an exploratory study of potential behavioral mediators other than education that may link the EA PGI with health capital and health behaviors to better understand our findings and inform further research. We find a large set of such potential mediators: early skills, early health, parental support of the child's education, the child's self-motivation for education, and the child's own job market outcomes in young adulthood (occupation,

household income, and household wealth).

Second, we contribute to understanding why more educated individuals are healthier. While there is a large literature in economics concerned with estimating the effect of education on health, the conclusions drawn by these papers regarding the causal status of the relationship are contradictory, with little attention paid to establishing the mechanisms behind the estimated effects and possible confounders behind the relationship (Galama et al., 2018; Grossman, 2022).

Among the expected major confounding factors in education-health studies are genetic endowments that predict both education and health (e.g., Boardman et al., 2015; Conti and Heckman, 2010). Genetic confounders have historically been viewed as unobservables, but recently became measurable due to major advances in genotyping and PGI construction techniques. Modern quality PGIs are still imperfect measures of genetic endowments. However, they are highly correlated with respective endowments, which makes them good candidates for proxies. As we discuss below, the proxy model has the potential to perfectly control for the omitted variable bias or at least mitigate it.

To the best of our knowledge, this is the first time molecular genetic measures are used as proxies of major expected confounders behind the effect of education on health. The reduction in the estimates of the effects of education on general health in our proxy model is substantial: the incremental effect of adding 17 PGI controls on top of traditional and cognitive-nonconnective controls is a reduction in the estimated association by about 11%. However, education still exhibits large and statistically significant association with general health and all other health-related outcomes after controlling for genoeconomic proxies of skill endowments, general health endowments, mental health endowments, and home environment. This novel result is at odds with a sizeable fraction of papers claiming that there is no causal effect of education on health, discussed in Section 4.2.1.

The use of PGIs is characterized by both advantages and limitations. One advantage

of using PGIs is that genetic endowments are determined at conception, and so parental actions afterwards (during pregnancy, childhood, adolescence, and so on) do not affect the child's PGI. This distinguishes PGIs from traditional measures of endowments, such as IQ tests. This property of the PGI creates an exclusion restriction that is useful for structural modeling and regression coefficient interpretation (Papageorge and Thom, 2020). However, PGIs are known to be imputed with measurement error (Becker et al., 2021). They are also known to correlate with home environment, as we explain below.

Due to peculiarities in the PGI construction,[2] the EA PGI does not only capture subjects' genetic endowments but also the non-inherited genetic endowments of parents, grandparents, etc., a phenomenon called *genetic nurture*. According to Kong and Thorleifsson (2018), these non-inherited parental endowments are still passed down to children through the family environment and account for about 30% of the variation in education endowments explained by EA PGI. In addition to genetic nurture, heredity also contributes to the correlation between PGIs and home environment, as some parental traits that affect home environment are genetically inherited by children. Overall, the confounding role of environment is known to be large for EA PGI. Based on a comparison between raw and within-sibship estimates, Howe et al. (2022) have shown that controlling for environment reduces the association between EA PGI and education by 50%.

The implications of these limitations of EA PGI differ across our two contributions. For our second contribution on the association between education and health, we need to proxy for as many potential unobserved confounders as possible. Therefore, it is an advantage for the proxy model that the PGIs do not only capture subjects' genetic endowments but also their home environment. For our first contribution on the association between EA PGI and health as a function of SES, the correlation between EA PGI and

[2]EA PGI depends not only on subjects' molecular genetic data but also on weights imputed from associations between molecular genetic data and observed education outcomes (of people from an independent sample).

SES with unobserved family environment in the error term can be expected to lead to biased estimates. So does measurement error in EA PGI. Therefore, we stress that we estimate a number of novel associations, not causal effects. We support the results of this general and exploratory paper with a more technical companion paper (Savelyev and Bolyard, 2024).

2 Data

Add Health is a panel dataset that follows roughly 20,000 individuals and contains detailed information on their family background, skills, education, and life outcomes in young adulthood. The respondents were first surveyed in 1995–1996, when they were in grades 7–12, and were followed into young adulthood. The most recent data that are used in this paper, Wave IV, were collected when the participants were 24–32 years old.

Add Health participants were drawn from a sample of middle and high schools. High schools were randomly chosen by stratifying schools within the Quality Education Database according to several demographic factors and weighting the probability that they would be selected according to their enrollment. One feeder middle school was randomly selected for each high school, weighted proportionally to its size. Respondents were chosen randomly after being stratified according to grade, sex, and school. The sample is considered nationally representative (Harris, 2013).

Our sample size is constrained by the availability of genetic data and the reliability of the imputed EA PGI. We perform this analysis only for individuals who took part in genotyping and self-identify as white because of the data limitations described below. Our estimation sample is 3,709.

In Table 1 we show descriptive statistics for education, health outcomes, health behaviors, and potential mechanisms behind the effect of EA PGI on health. Below we discuss these groups of variables.

Table 1: The Highest Education Level, Health-Related Outcomes in Young Adulthood, and Potential Mechanisms of the EA PGI Effects on Health

	Full Sample ($N_f = 3,709$)		Low SES ($N_l = 1,404$)		High SES ($N_h = 2,305$)	
	Average	Standard Deviation	Average	Standard Deviation	Average	Standard Deviation
Highest Education Level						
Below high school[a]	0.048	0.214	0.080	0.272	0.029	0.167
High school diploma	0.415	0.493	0.516	0.500	0.353	0.478
College below Bachelor's[b]	0.174	0.380	0.180	0.384	0.171	0.377
Bachelor's or above	0.363	0.481	0.223	0.416	0.448	0.497
Health and Health Behaviors in Young Adulthood						
General health rating[c]	3.745	0.889	3.640	0.908	3.810	0.870
Good health[d]	0.625	0.484	0.565	0.496	0.662	0.473
Risky drinking of alcohol[e]	0.209	0.407	0.228	0.420	0.197	0.398
Marijuana use[f]	0.096	0.294	0.093	0.291	0.097	0.296
No exercise[g]	0.130	0.336	0.148	0.355	0.119	0.324
Smoking cigarettes[h]	0.261	0.439	0.300	0.459	0.237	0.425
Obesity[i]	0.343	0.475	0.387	0.487	0.317	0.465
Depression[j]	0.191	0.393	0.187	0.390	0.194	0.396
Potential Mechanisms						
Early health[k]	0.705	0.456	0.658	0.475	0.734	0.442
Cognitive skills[l]	0.000	1.000	-0.158	1.003	0.096	0.986
Conscientiousness[l]	0.000	1.000	-0.015	0.999	0.009	1.001
Extraversion[l]	0.000	1.000	-0.059	0.993	0.036	1.003
Emotional stability[l]	0.000	1.000	-0.053	1.013	0.032	0.991
Education support-self[l]	0.000	1.000	-0.258	1.058	0.157	0.929
Education support-parental[l]	0.000	1.000	-0.165	1.035	0.100	0.965
Household income[m]	8.398	2.354	8.004	2.465	8.633	2.252
Household assets[n]	3.834	1.902	3.593	1.861	3.980	1.911
Job satisfaction[o]	2.215	1.054	2.071	1.025	2.302	1.061
Job physicality[o]	2.073	1.071	2.197	1.074	2.000	1.063

Notes: Calculations based on the Add Health data. Estimation sample size reported. For the purposes of descriptive analysis only, high SES is defined as having the SES factor score above its average; low SES otherwise. [a]No high school diploma (including having a GED certificate). [b]Completed post-high school degree that takes at least one year to complete. [c]Self-evaluated on a scale from 1 (poor) to 5 (excellent). [d]General health ranked 4 or 5. [e]Typical number of drinks per occasion exceeds four. [f]Smoking marijuana once or more per week, on average, during the last year. [g]None of the following: playing sports, exercising outside, walking for exercise, or engaging in other physical activity during the past week. [h]Smoking at least one cigarette within the past 30 days. [i]BMI $\geq$ 30. [j]Had ever been told by a health care provider that they had depression. [k]Self-reported good health. [l]Standardized factor score. See measures listed in Table A-1. [m]Bands: 1(lowest)–12. [n]Bands: 1(lowest)–9. [o]Self-rating: 1(least)–4.

Education Because the effect of education on health-related outcomes might be nonlinear, we rely on educational categories rather than total years of education. We distinguish four categories of the highest degree completed by wave IV: (1) no high school diploma; (2) high school diploma; (3) a completed post-high school degree below bachelor's that takes at least one year to complete; and (4) a bachelor's degree or above.[3]

Health and Health Behaviors in Young Adulthood We study health and health behaviors from wave IV of AddHealth. Self-reported health is the key outcome of interest, because it has been shown to be predictive of mortality, and it is an essential measure of overall health (Idler and Benyamini, 1997). We use self-reported good health as an outcome. Obesity and depression can be viewed as measures of health. In addition, we study health behaviors: risky drinking of alcohol, smoking cigarettes, marijuana use, and lack of physical exercise. See Table 1 for variable definitions and descriptive statistics.

Potential Mechanisms We also study a number of potential mechanisms in order to suggest possible causal pathways from EA PGI to health and health behaviors in young adulthood. Those include characteristics of the parent (attitude towards child's education) and the child (general health, cognitive and noncognitive skills, and educational motivation.) We supplement these data from wave I with data on potential mechanisms later in life that are available in wave 4: "household income," "household assets," "job satisfaction," and "job physicality," the degree to which the job is physically demanding.

To measure cognitive skills, we use participants' scores on the Add Health Picture Vocabulary Test, recent science grades, and recent math grades. To measure noncognitive skills we use the well-established Big Five Personality taxonomy. Most noncognitive

[3]Given that the youngest participant is 24 by wave IV, we leave the study of more advanced degrees to future research. That said, we can expect the effects of more advanced degrees on health to be, at best, weak: there is evidence in the literature that advanced degrees do not further contribute to health on top of the health effect of the bachelor's (Savelyev, 2022). This evidence is based on a high-IQ sample, but completing advanced degrees is strongly associated with having a high IQ (Jensen, 1998).

skills map into the Big Five in some manner (e.g., Borghans et al., 2008). The Big Five skills are Openness, Conscientiousness, Extraversion, Agreeableness, and Emotional Stability. Openness is a propensity to be open to new experiences and ideas; Conscientiousness is a propensity to follow rules and plan the future; Extraversion is a propensity to be active and social; Agreeableness is a propensity to behave amicably towards others; and Emotional Stability is a propensity to control one's emotions. We follow a paper by psychologists Young and Beaujean (2011) who suggest measures of early Conscientiousness, Extraversion, and Emotional Stability based on available measures of personality in the first wave of the Add Health.[4]

We call the attitudes towards education variables "education support—self," and "education support—parental." Typical questions about parental support ask whether the father would be disappointed if the child did not graduate from high school. The same question is asked about graduation from college. The same questions are repeated about the mother's attitudes. Self-support is measured by questions about the student's own plans to go to college and their expectations about graduating from college. The full list of questions is available in the Web Appendix.[5]

From the Table 1 we can see that high-SES subjects tend to report better early health, superior early skills and education support, higher levels of education, more favorable job-related outcomes, better health, and healthier lifestyles in young adulthood. For instance, graduation from college is about twice as likely for high-SES subjects (0.45 for high-SES vs. 0.23 for low-SES). These differences present evidence that our SES measures described below capture important population differences that are relevant for socio-economic outcomes.

[4]See Table A-1 of the Web Appendix for the list of measures for all continuous latent factors. Due to data limitations, we are not able to study early Agreeableness and Openness.

[5]See Table A-1 of the Web Appendix.

SES To study the interaction of respondents' genetic endowment with family SES in their childhood, we follow the literature on PGI-SES interaction (Bierut et al., 2018; Papageorge and Thom, 2020; Ronda et al., 2020), and construct measures of SES from relevant variables that are available in the Add Health data. We also show the robustness of our results to a number of alternative definitions of SES.

The literature has proposed a number of SES measures. In particular, Ronda et al. (2020), who use the Integrative Psychiatric Research Study data from Denmark, utilize the following four binary measures of low SES: both parents lacking any post-secondary education; growing up in a family in the lowest quintile of disposable family income; either parent ever being diagnosed with a mental health condition; growing up in a broken family, with non-cohabiting parents, between the ages 0 and 10. Papageorge and Thom (2020) and Bierut et al. (2018) use Health and Retirement Study (HRS) data from the USA and also utilize binary measures of SES: father's income above the median; family is well-off; family never had to move or to ask for help; father never experienced any significant unemployment spell ("several months or more").

The Add Health data contain measures that either match measures used in the literature or describe related disadvantages. Like in the above literature, we proxy SES with binary measures. We use the following five measures of family SES in childhood for our main model specification: living in an unsafe neighborhood; receiving government assistance (such as welfare); having difficulty paying bills; at least one parent has a college degree; and parental income from the lowest quintile. These measures are summarized in Panel A of Figure 1. This particular set of five measures is characterized by the strong specification statistics of the corresponding factor model, as we discuss in Section 3. We also show the robustness of our results to using alternative sets of measures for the SES factor, as well as to alternative methods of their aggregation.

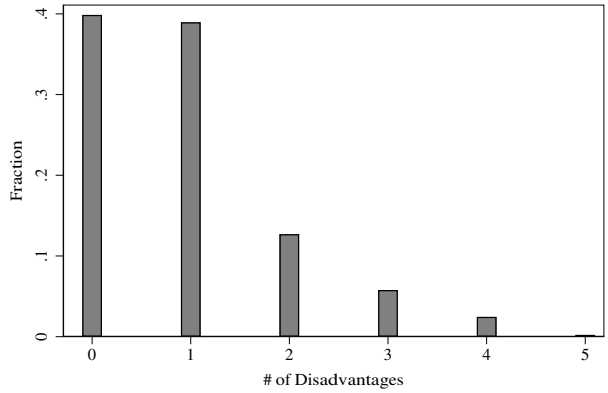
Panel B presents a histogram for the count of disadvantages based on variables listed

Figure 1: Description of SES

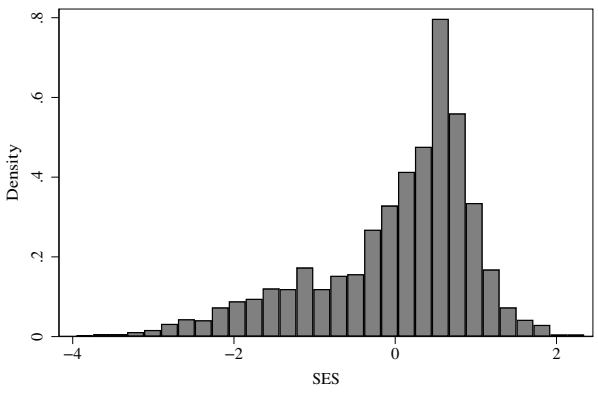
A. Measures of SES

	Average	Standard Deviation
Living in an unsafe neighborhood[(a)]	0.073	0.259
Household received assistance[(b)]	0.198	0.399
Trouble paying bills[(c)]	0.121	0.326
Parental college[(d)]	0.523	0.500
Income from the lowest quintile[(e)]	0.190	0.393

B. Number of Disadvantages



C. SES Factor Score



Note: Calculations are based on the Add Health data. Estimation sample size is 3,709. All SES measures are reported by either a parent or the subject (child, student) in wave I, with the exception of "unsafe neighbourhood," which was reported in wave 2. All variables are binary. [(a)]The subject indicates that they do not usually feel safe in their neighborhood. [(b)]Any member of the subject's family received any form of social assistance last month before the survey: Social Security or Railroad Retirement payments, Supplemental Security Income, Aid to Families with Dependent Children (AFDC), food stamps, unemployment or workers' compensation, housing subsidy, or public housing. [(c)]Based on a question to a parent: "do you have enough money to pay your bills?" [(d)]Subject reports that at least one of their parents graduated from a college or university. [(e)]Parent's reported income is below the 20th percentile in the sample. (A large mass of reported income exactly at the 20th percentile leads to the average of 0.19 rather than 0.20.)

in Panel A.[6] We can see that experiencing no disadvantages is the mode, which is characterized by a likelihood of about 0.40. Experiencing one disadvantage has a similar likelihood, 0.39. After that, likelihoods quickly drop to 0.13 for 2 disadvantages and keep declining: 0.06 for 3, 0.02 for 4, and 0.0023 for 5. This right tail makes the histogram right-skewed.

Finally, Panel C shows a histogram of an SES factor score that is implied by the measurement system of our main factor model.[7] Our SES factor score in Panel C is normalized to be positive, so that higher levels of SES correspond to more advantaged families. In contrast, the count of disadvantages is a negative measure of SES, with higher values of disadvantages corresponding to less advantaged families. Keeping in mind the reversed signs of these two panels as well as the differences between discrete and continuous random variables, we can see that histograms in Panels B and C are similar in shape. The high-likelihood part around zero and above roughly corresponds to having at most one disadvantage. The long left tail in Panel C corresponds to having two or more disadvantages.

We leave the discussion of the advantages of latent factor models over alternative methods of data aggregation to Section 3.

PGIs The most basic DNA building blocks that vary among humans are called single-nucleotide polymorphisms (SNPs, pronounced "snips"). In principle, individual SNPs can be used as predictors of life outcomes. In practice, predictions based on individual SNPs lead to low statistical power and issues with replaceability, as life outcomes are typically affected by a large number of SNPs. A well-established solution to this problem is using a polygenic index (PGI) instead of a SNP. A PGI is an optimally-weighted aggregate of multiple SNPs. PGIs demonstrate considerably stronger predictive power

[6] "Parental college" is our only positive measure of SES, and so the corresponding "lack of parental college" is used for a count of disadvantages.

[7] Measurement system (4) is introduced and discussed in Section 3.

and more robust results across populations than a single SNP (Benjamin et al., 2012).

Modern quality PGIs are constructed using large independent samples by regressing an outcome (phenotype) of interest, on millions of SNPs obtained through genotyping, SNP-by-SNP. The coefficients are then adjusted to correct for known correlations among SNPs (linkage disequilibrium) to prevent double counting of genetic information. The adjusted coefficients are then used as weights to impute PGIs as a weighted sum of SNPs.

This paper is focused on a specific PGI called EA PGI, which is designed to capture individuals' genetic predisposition for the total number of years of formal education. PGIs are constructed by various groups of authors who rely on different samples, different total sample sizes, and different numbers of aggregated SNPs, among other choices. In this paper we rely on the recent state-of-the-art EA PGI constructed by Lee et al. (2018) based on a sample of over 1.1 million people of European descent and aggregating 10 million of measured SNPs. This EA PGI explains about 13% of variation in years of education in the Add Health data.

For technical details behind PGI construction in general, see reviews of genetic literature written for an economic audience (see Benjamin et al. (2012), Beauchamp et al. (2011). For technical details behind PGIs used in this paper, see Braudt and Harris (2018) and Okbay et al. (2018).

Because the EA PGI is constructed based on data collected from individuals with European ancestry, we restrict our sample to those who self-report as white. Here we follow the literature confirming that polygenic indices constructed using European-ancestry samples are both biased and less predictive when applied to populations with different ancestry (Martin et al., 2017).

In addition to modeling the effects of EA PGI, which is our main variable of interest, we also take advantage of PGIs that proxy health endowments. When we study the effect of education on health (contribution 2) we control for nine PGIs that describe

Table 2: Background Variables

	Full Sample ($N_f = 3,709$)		Low SES ($N_l = 1,404$)		High SES ($N_h = 2,305$)	
	Average	Standard Deviation	Average	Standard Deviation	Average	Standard Deviation
Educational Attainment Polygenic Index						
EA PGI[a]	0.000	1.000	-0.171	0.984	0.104	0.996
Background Controls						
Biological sex is male	0.464	0.499	0.468	0.499	0.462	0.499
Age 10-12 at wave I	0.084	0.277	0.085	0.279	0.083	0.276
Age 13-14 at wave I	0.300	0.458	0.294	0.456	0.303	0.460
Age 15-16 at wave I	0.394	0.489	0.399	0.490	0.391	0.488
Age 17-19 at wave I	0.222	0.416	0.222	0.416	0.222	0.416
US Region: West	0.144	0.351	0.152	0.359	0.139	0.346
US Region: Midwest	0.332	0.471	0.330	0.471	0.332	0.471
US Region: Northeast	0.156	0.363	0.159	0.366	0.154	0.361
US Region: South	0.368	0.482	0.359	0.480	0.374	0.484
Rural residence	0.361	0.480	0.351	0.478	0.367	0.482
Suburban residence	0.399	0.490	0.398	0.490	0.400	0.490
Urban residence	0.239	0.427	0.251	0.434	0.233	0.423
Low birth weight[b]	0.083	0.276	0.089	0.285	0.080	0.271
The only child	0.205	0.403	0.216	0.412	0.197	0.398
First-born	0.320	0.467	0.309	0.462	0.328	0.469
Second-born	0.308	0.462	0.311	0.463	0.306	0.461
Third-born	0.112	0.316	0.107	0.309	0.115	0.320
Number of siblings	2.549	1.950	2.623	2.011	2.504	1.911
Parents married	0.805	0.396	0.741	0.439	0.845	0.362
Cigarettes smoked at home	0.459	0.498	0.462	0.499	0.456	0.498
Meals with parents[c]	4.897	2.328	4.791	2.406	4.961	2.278
Hispanic origin	0.060	0.238	0.075	0.263	0.051	0.220
Genetic ancestry PC[d]	Yes		Yes		Yes	

Notes: Calculations based on the Add Health data; estimation sample size reported. For the purposes of descriptive analysis only, high SES is defined as an SES factor score above its average; low SES otherwise. [a]Standardized EA PGI (Lee et al., 2018). [b]Birthweight$\leq$2.5 kg. [c]Number of evening meals with parents per week. [d]10 principal components based on genetic data.

physical health endowments[8] and seven mental health PGIs.[9] The choice of these PGIs is determined by their availability in the AddHealth data. However, we do not use these additional controls for our contribution 1 so that we keep the estimated effects of EA PGI clearly interpretable and comparable to the literature. We demonstrate correlations between EA PGI and PGIs that describe general health and mental health in the Web Appendix. These correlations range from negligible to modest.[10]

Background Control Variables On top of controlling for EA PGI and SES, we control for a range of early-life controls from wave I that could influence education and health. Those include biological sex, age, US region, degree of urbanization of the family residence, low birth weight, number of siblings, the order of birth among siblings, having parents who are married, cigarettes smoked at home, and number of meals with parents per week. We also use 10 principal components of the full matrix of genetic data, which is a standard way to account for ethnic differences (intra-European in our case). See Table 2 for variable definitions and descriptive statistics by SES.

3 Methodology

Model of EA PGI and Health-Related Outcomes For our study of the association between EA PGI and health-related outcomes, as well as potential mechanisms behind health formation, we employ a reduced form model that accounts for an interaction between the EA PGI and parental SES. This model is comparable to models used in recent economic papers on gene-environment interactions (e.g., Barth et al., 2020; Bierut

[8]These include PGIs for coronary artery disease, myocardial infarction, low-density lipoprotein cholesterol, triglycerides, Type II diabetes, BMI, Waist-to-hip ratio, Height, and Smoking.

[9]These include PGIs for Depression, Neuroticism, Attention-deficit disorder, Bipolar disorder, Major depressive disorder, Schizophrenia, and Mental health cross disorder.

[10]See Tables A-2 and A-3 of the Web Appendix.

et al., 2018; Papageorge and Thom, 2020). The model is specified as follows:

$$Y_k^* = b_{1k}EAPGI + b_{2k}EAPGI \cdot \theta^{SES} + b_{3k}EAPGI^2 + b_{4k}\theta^{SES} + \boldsymbol{b}_{5k}\boldsymbol{X} + \eta_k, \quad (1)$$

where outcome Y_k^* denotes a latent propensity for an outcome Y_k of type k, $k = 1, ..., K_1$. Equation (1) summarizes several types of models depending on the type of outcome Y_k. For binary outcomes we use a logit model, so that $Y_k = 1$ if $Y_k^* > 0$ and $Y_k = 0$ otherwise. For ordered categorical outcomes we use an ordered logit model. For continuous outcomes $Y_k^* = Y_k$, resulting in a model that is linear in parameters. $EAPGI$ denotes EA PGI; θ^{SES} is a latent continuous factor that represents parental socioeconomic status at the time of the subject's childhood. Vector $\boldsymbol{X}$ represents a full set of background controls from Table 2, plus a constant to allow for an unrestricted intercept; ϵ_k is an error term.

We follow the analysis by Papageorge and Thom (2020), who argue that SES can be viewed as a proxy for family investments in a child's human capital. They also argue that EA PGI may affect the measurement error in SES, which is a measure of such investment. Based on a structural model, the authors demonstrate that if a reduced form model controls for PGI^2, we can properly interpret the sign of the interaction effect, b_{2k}, as the sign of interaction between genetic endowment and family investments, while without this quadratic control the sign of the interaction would be indeterminant. Therefore, all of our outcome models include a quadratic PGI term, similar to the main model by Papageorge and Thom (2020).[11]

For the identification of model (1), which involves a latent SES factor θ^{SES}, we jointly estimate model (1) with a measurement system (4) that we discuss below.

[11]We also explored other potential nonlinearities. Following Keller (2014), we tested the joint statistical significance of the following potential regressors: $\boldsymbol{X} * PGI$ and $\boldsymbol{X} * SES$. We failed to reject the test and found that both AIC and BIC increase when these regressors are added. Therefore, we keep these interactions out of our main model specification for the sake of superior parsimony and efficiency.

Estimating the Association between EA PGI and Health-Related Outcomes Conditional on Education We also estimate a model that is similar to (1) but conditional on education $\boldsymbol{D}$ to establish which part of the association between the EA PGI and an outcome of interest that is not explained by education. The causal analogue of this association is the direct effect of EA PGI on outcomes, with the indirect effect acting through education.

$$\begin{aligned} Y_k^* = & \boldsymbol{c}_{1k}\boldsymbol{D} + \boldsymbol{c}_{2k}\boldsymbol{D} \cdot \theta^{SES} + c_{3k}EAPGI + c_{4k}EAPGI \cdot \theta^{SES} \\ & + c_{5k}EAPGI^2 + c_{6k}\theta^{SES} + \boldsymbol{c}_{7k}\boldsymbol{X} + \lambda_k, \end{aligned} \tag{2}$$

where $\boldsymbol{D}$ denotes a vector of three binary variables representing the education levels.[12]

To make this direct association comparable to the total association estimated in model (1), model (2) is specified exactly the same way as (1) except for controlling for education and its interactions. Similarly to model (1), model (2) is estimated jointly with the measurement system (4).

We interpret the coefficients of models (1) and (2) as associations. To address the endogeneity issues, we support this exploratory general paper with a more technical companion paper (Savelyev and Bolyard, 2024).

Model of Education and Health We also estimate a third reduced form model that is designed to test whether well-known strong associations between education and health-related outcomes survive controlling for proxies of major expected confounders, which are endowments for skills, general health, and mental health proxied by PGIs. Moreover, due to the correlation between home environment and children's PGIs, these PGIs also proxy unobserved home environment (Howe et al., 2022; Kong and Thorleifsson, 2018), which is another major expected confounder of the effect of education on health.

[12]These binary variables include: education below high school, high school diploma, and college degree below bachelor's. Bachelor's degree or above serves as a comparison category.

Proxies help eliminate or mitigate the omitted variable bias while also reducing the residual variance. There is no need for a proxy variable to perfectly correlate with the omitted variable; however, it makes the proxy model assumptions (under which the bias is fully eliminated) more plausible when a proxy is strongly predictive of it (Wooldridge, 2010). Given the increasingly high predictive power of modern PGIs, they make good candidates for proxies. While the proxy model has been originally established for the linear regression, proxies proved to be effective for logistic regression as well (e.g., Rosenbaum et al., 2023), which is the preferred model for binary outcomes in this paper.

The model is the following:

$$\begin{aligned} Y_k^* = & \boldsymbol{d}_{1k}\boldsymbol{D} + \boldsymbol{d}_{2k}\boldsymbol{D}\cdot\theta^{SES} + \boldsymbol{d}_{3k}\boldsymbol{PGI} + \boldsymbol{d}_{4k}\boldsymbol{PGI}\cdot\theta^{SES} \\ & + \boldsymbol{d}_{5k}\boldsymbol{PGI}^2 + d_{6k}\theta^{SES} + \boldsymbol{d}_{7k}\boldsymbol{\theta}^{CN} + \boldsymbol{d}_{8k}\boldsymbol{X} + \xi_k, \end{aligned} \tag{3}$$

where variables $Y_k, k = 1, ..., K_2$, represent health-related outcomes in young adulthood. In a logit model, Y_k^* is a latent propensity for outcome Y_k. We follow the same notation as in models (1) and (2), but with a number of additional features described below.

The main difference between models (2) and (3) is that (2) is designed to to estimate the direct effect of EA PGI while keeping comparable specification to model (1). In contrast, model (3) is designed to estimate the total effect of education, described by coefficients $\boldsymbol{d}_{1k}$ and $\boldsymbol{d}_{2k}$, which downgrades the role of EA PGI from the main variable of interest to one of many proxy variables. To maximize the set of accounted-for confounders, we include 16 additional PGIs on top of the EA PGI, resulting in a vector of 17 PGIs denoted as $\boldsymbol{PGI}$. We also control for early cognitive and noncognitive skills though a vector of latent variables, $\boldsymbol{\theta}^{CN}$.

To better account for possible nonlinearities and to be consistent with the models above, we control for a vector of squared PGI indices, $\boldsymbol{PGI}^2$, and interaction terms, $\boldsymbol{PGI}\cdot\theta^{SES}$. However, to keep the model parsimonious, we do not control for the in-

teraction between the 17 PGIs and three levels of education. Coefficients for these 51 potential variables are not jointly statistically significant and other coefficients are robust to these potential controls. Nor do we control for the interaction of SES and $\boldsymbol{PGI}$ with $\boldsymbol{X}$ for the same reason.

Finally, we compare an unrestricted model (3) with its restricted version, in which we jointly set to zero the following coefficients: $\boldsymbol{d}_{3k}$, $\boldsymbol{d}_{4k}$, $\boldsymbol{d}_{5k}$ and a part of vector $\boldsymbol{d}_{8k}$ that corresponds to the 10 first principal components of genetic data.[13] This comparison helps us explore how controlling for a large number of genoeconomic controls affects associations $\boldsymbol{d}_{1k}$. We also explore how associations $\boldsymbol{d}_{1k}$ change when we omit all traditional controls, all cognitive-noncognitive controls, and various combinations of these restrictions.

As with models (1) and (2), model (3) is estimated jointly with the measurement system (4), which we now specify not only for latent SES, but also for latent cognitive and noncognitive skills.

Measurement system Following well-established factor model methodology (e.g., Anderson and Rubin, 1956; Conti and Heckman, 2010), to identify each of the models above (1, 2, and 3), which involve a latent SES factor, θ^{SES}, we need additional information provided by the measurement system (4). This system of equations relates latent factor θ^{SES} to its several observable dedicated measures M_j conditional on background controls $\boldsymbol{X}$, where $\boldsymbol{X}$ includes a constant, while accounting for measurement error ϵ_j:

$$M_j^* = a_{1j}\theta^{SES} + \boldsymbol{a}_{2j}\boldsymbol{X} + \epsilon_j, \quad j = 1, ..., J. \tag{4}$$

[13]The first principal components of genetic data control for ethnic origin and serve as a standard controls in regression analysis involving PGIs, because ethnic origin is a potential confounder of the effect of a PGI. Therefore, it is natural to test restrictions for PGIs together with restriction for principal components.

Here J is the total number of dedicated measures of θ^{SES}, and ϵ_j are error terms. All models in this system are logit models, and so variables M_j^* are latent variables, so that $M_j = 1$ if $M_j^* > 0$; $M_j = 0$ otherwise; a_{1j} and $\boldsymbol{a}_{2j}$ are unknown coefficients to be estimated.

We make assumptions and normalizations that are standard for a classical factor model with dedicated measures (e.g., Conti et al., 2014). Error terms are independent of each other and of covariates. Conditional on observable controls, latent factor θ^{SES} absorbs common variation across outcomes and measures, which helps us justify the assumption of independence of the error terms from each other. Therefore, conditional on controls, the latent factor is the only source of correlation among its dedicated measures. Our conditioning of the factor model on a substantial set of controls helps us to account for possible systematic influences.

We follow the literature on factor model specification testing by calculating several established specification statistics, which are consistent with correct model specification (as documented in Section 4 below). In addition, we show that simple equally-weighted indices and binary aggregations of SES measures lead to the same conclusions as our main factor model, which implies that our results are not driven by the peculiar factor model assumptions described above. Finally, we show the robustness of our results to using alternative sets of SES measures.

We follow an established approach to normalization that allows us to identify the model while keeping it easily interpretable: each latent variable is normalized to have mean zero and variance one, and for each factor we set a sign to the coefficient $a_{1,1}$ in such a way so that the resulting latent factor is interpreted positively.[14] Finally, the

[14]Our first SES measure is "living in an unsafe neighborhood," a negative measure of SES, and so we reversing the sign of the corresponding factor loading creates a positive latent SES. As we can see, an indeterminacy of factor sign that requires an arbitrary normalization creates no issues for interpretation: after all, we do need to choose whether we wish to define the SES as positive (a measure of advantage) or negative (a measure of disadvantage) and then interpret the results accordingly.

sufficient condition for model identification is satisfied for our factor model, as we have at least three dedicated measures M_j per latent factor, $J \geq 3$ (e.g., Conti et al., 2014). We model latent cognitive and noncognitive skills using models with dedicated measures using the same type of measurement systems as (4).

Possible Alternatives to the Factor Model We argue that the factor model is preferable to other established methods of aggregation: equally-weighted indices, binary aggregation, and principal components.

Using an equally-weighted average of measures is a common alternative approach to aggregation (e.g., Kaestner and Callison, 2011). This procedure has the benefit of calculational simplicity. However, it is based on an arbitrarily equal weighting of measures: all measures are assumed to be equally informative about the underlying factor that they proxy. This assumption is at odds with our data: we test and overwhelmingly reject the equality of factor loadings hypothesis.[15] Therefore, at least for the measures used in this paper, a simple sum is not an optimal representation of the latent factor.

Another approach is a binary aggregation (e.g., Ronda et al., 2020). For instance, we can define aggregate $SES = 0$ if at least one of its binary measures shows disadvantaged SES, and $SES = 1$ otherwise. As with the index approach, the main benefit of this aggregation is its calculational simplicity, while the cost is an already mentioned implicit unrealistic assumption that different SES measures are equally reliable. In addition, a binary aggregation leads to an information loss. For instance, in the example of binary SES aggregation given above, the aggregate is the same for those having only one disadvantage and those having several disadvantages.

Yet another common approach to dimensionality reduction is the method of principal components. However, unlike the factor model, this method does not account for measurement error, and so it is less desirable even though it is often used in the literature

[15] See Table A-4 of the Web Appendix.

as an alternative to the factor model. See Conti et al. (2014) for a discussion about the advantages of the factor model over principal components.

To summarize, by using a factor model rather than its alternatives, we gain several advantages: we explicitly control for measurement error, avoid arbitrarily equal weights, and control for possible systematic determinants of peoples' perceptions that may affect answers. This is done based on an internally consistent system of logit models, rather than approximations such as linear probability models. These advantages come at the cost of increased complexity and making factor model assumptions. However, we provide empirical evidence consistent with correct factor model specification and show the robustness of our qualitative results to simple alternatives to the factor model.

Imputation of Missing Values in Controls We impute missing values for a subset of background control variables $\boldsymbol{X}$ using the well-established MCMC multiple imputation procedure, which is known to preserve the variance-covariance matrix of variables (Schafer, 1999). This imputation allows us to control for more background variables without diminishing the estimation sample size.

4 Results

Our empirical part is split in two sections, 4.1 and 4.2, which are devoted to our contributions 1 and 2 respectively. Both sections start with the big picture questions regarding our contributions (what is done in the literature and how we contribute in general) and then proceed with the empirical results.

In Section 4.1 we present a number of descriptive graphs to motivate our regression analysis. Then we study conditional associations between EA PGI and health-related outcomes as a function of parental SES. Afterwards, we proceed to suggestive evidence regarding the mechanisms behind these relationships.

In Section 4.2 we study the association between education and health-related outcomes and establish the relative confounding role of of the traditional, cognitive-noncognitive, and genoeconomic controls. We find that the strong and statistically significant association between education and health survives not only controlling for traditional background variables and skills, but also for genoeconomic proxies of major expected confounders.

This paper is designed to be general and exploratory. We leave a more technical approach to a companion paper (Savelyev and Bolyard, 2024), one contribution of which is to support the results of this paper.

4.1 EA PGI, Parental SES, and Health

4.1.1 Motivation and Contributions to the Literature

Several studies provide evidence consistent with a positive relationship between a person's EA PGI and health. Marioni et al. (2016) use a child's EA PGI as a proxy for the unobserved parental EA PGI and find that the proxy is positively associated with parental longevity. Barcellos et al. (2018) find that EA PGI is negatively associated with blood pressure and poor health[16]. Further, Huibregtse et al. (2021) report a negative association between an EA PGI and frailty in old age. Selzam et al. (2019) find that an EA PGI is negatively associated with BMI and positively associated with self-reported health, though these associations do not survive controlling for dizygotic twin fixed effects.[17] Wedow et al. (2018) report that an EA PGI is negatively associated with smoking. Finally, Demange et al. (2020) find associations between an EA PGI and a number of health-related outcomes based on multiple datasets, including Add Health.

In this paper we replicate the health-beneficial associations of EA PGI with health

[16]Poor health is measured by an index, which is a weighted average of blood pressure, body size, and adverse lung function.

[17]However, a positive association between EA PGI and IQ, and a negative association between EA PGI and ADHD survive controlling for fixed effects.

and health behaviors, but the novel result of this paper is our discovery of a strong interaction between EA PGI and parental SES in affecting health and health behaviors and our study of the potential mechanisms behind these relationships. Below, we put this result in the context of the literature.

Our EA PGI-SES interaction model is grounded in theory. As we know from epigenetic research, environment shapes gene expression. This means the traditional nature versus nurture distinction is outdated: gene-environment interaction is important and should be accounted for (e.g., Heckman, 2007). In addition, economic theory also suggests that SES may contribute to health differences through interaction effects (e.g., Galama and van Kippersluis, 2018). In particular, as discussed above, we can expect that severe economic disadvantage may limit the effect of a child's genetic potential, thus creating an environmental bottleneck effect (Fletcher, 2019).[18]

Related to environmental bottlenecks is the Scarr-Rowe hypothesis: an exposure to socioeconomic disadvantage leads to lower association between the IQ of parents and their children (Scarr-Salapatek, 1971). The Scarr-Rowe effect can be interpreted as gene-by-environment interaction: low parental SES may prevent children from taking full advantage of their genetic endowments. Therefore, we can see that EA PGI-based studies, including ours, are consistent with the same type of environmental bottleneck effect that has been established earlier based on IQ scores, even though EA PGI and IQ are very different in terms of their construction and limitations.

Similar interactions have been found between the environment and genetic endowment. Bierut et al. (2018) show that an advantaged childhood SES provides a major protective effect against a genetic predisposition to smoke, as measured by a smoking PGI. Papageorge and Thom (2020) find that an EA PGI is associated with higher education gains when children have high SES. Ronda et al. (2020) find that hardship in

[18] An *environmental bottleneck* should not be confused with a *genetic bottleneck*, as both terms are related to genes and adverse environment, but very differently: genetic bottleneck is a term for a catastrophic shrinkage in a population that reduces genetic variation.

childhood, as measured by low childhood SES, diminishes the effect of EA PGI on education and skill capital. We confirm these results for education for different dataset (Add Health) as part of our study of the mechanisms behind the effects on EA PGI on health.

Related results are reported by Schmitz and Conley (2017) and Avinun (2019). Schmitz and Conley (2017) find that reductions in educational attainment as a result of Vietnam-era conscription are larger for individuals with lower EA PGI, providing evidence that a combination of severe environmental conditions and an unfavorable genetic endowment is particularly harmful. Avinun (2019) finds that an EA PGI interacts with a subject's own SES in affecting depression. Our paper has a different focus than these studies, as we study the interaction of the PGI with childhood SES (which is parental SES in the subject's childhood) as a measure of a child's developmental bottleneck rather than mediation through a person's own SES later in life.

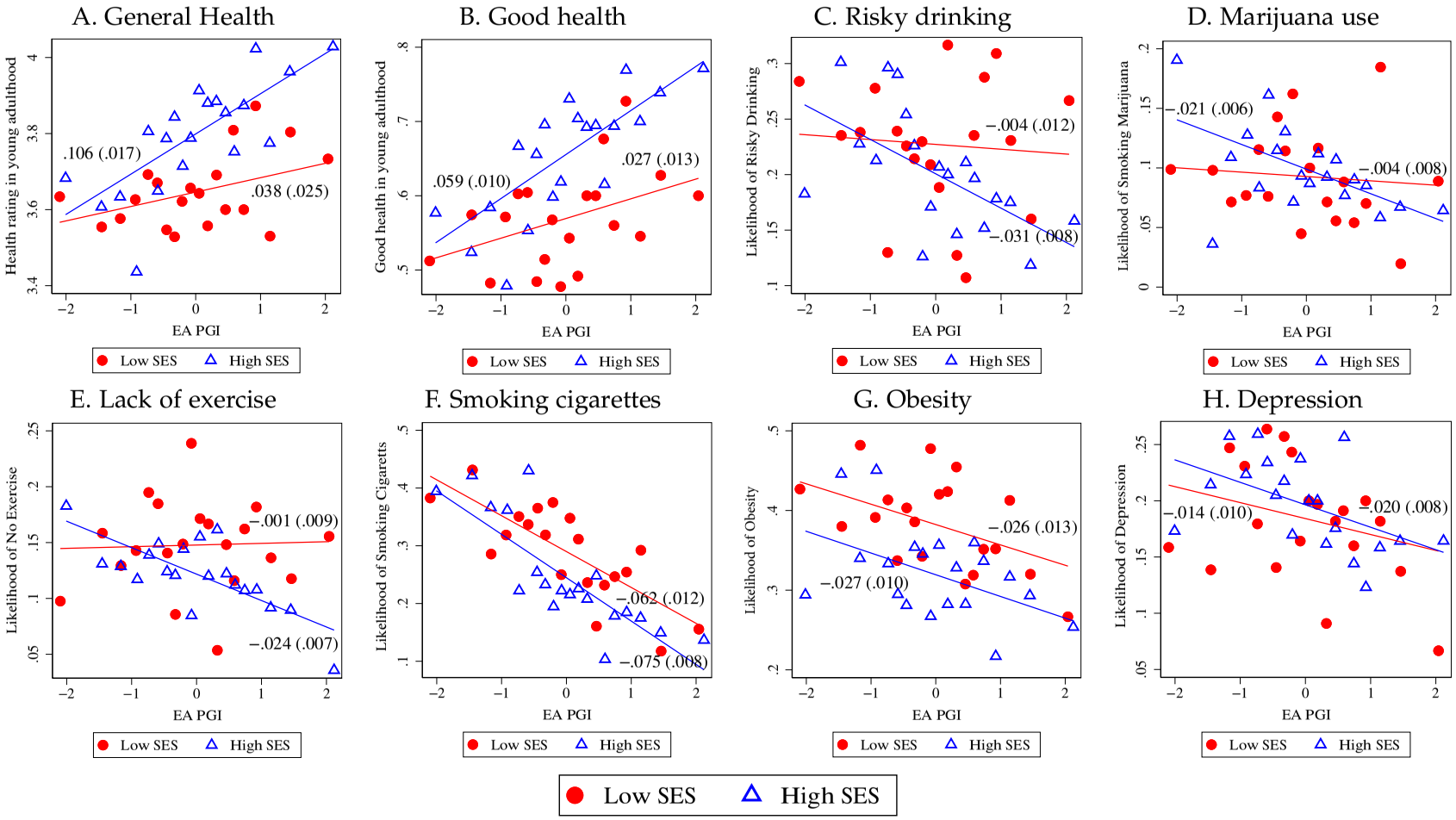
Our study contributes to this literature, as we use different data to study different outcomes, namely outcomes that are related to health and health behaviors. We study how these outcomes are related to EA PGI and how this relationship depends on parental SES. To the best of our knowledge, we are the first to study the interaction between an EA PGI and childhood SES in predicting health and health behaviors. In addition, we contribute with studying pathways that connect EA PGI and health. These findings allow us to better understand the health capital formation and inform policy debates on the costs and benefits of anti-poverty measures.

4.1.2 Descriptive Results

In Figure 2 we provide a descriptive preview of our contribution 1: the relationship between the EA PGI and health-related outcomes by parental SES. For the purpose of descriptive analysis only, high SES is defined as SES factor score above its average; low SES otherwise.

Each panel of Figure 2 shows two results: (1) The bin scatter plot for the relation-

Figure 2: Bin Scatter Plots and Univariate Linear Regressions by Parental SES[(a)]



Notes: Calculations are based on the AddHealth Data. [(a)]For the purposes of descriptive analysis only, high SES is defined as having a parental SES factor score above its average; low SES otherwise. For each SES group, the x-axis variable is grouped into 20 equal-sized bins. For each bin of each SES group, averages of x and y are computed for a nonparametric data visualization In addition, for each SES level, an estimated linear univariate regression of y on x is plotted using the original data (no bins). Slope coefficients are shown for these regressions, with standard errors presented in parentheses.

ship between EA PGI and a health-related outcome by SES. Each such scatter plot is a nonparametric estimate of conditional expectation function;[19] (2) A superimposed univariate linear regression line of a health-related outcome regressed on EA PGI by SES (using actual data, not bins). Slope coefficients from these regressions are shown in the graph, with corresponding standard errors in parentheses.

The descriptive analysis in Figure 2 shows the following tendency: (1) For subjects with high parental SES, higher EA PGI always corresponds to better health or a smaller likelihood of an adverse health behavior (see Panels A–H). (2) For subjects with low parental SES, the relationship between EA PGI and health-related outcomes tends to be weaker or statistically insignificant (see Panels A, B, C, E, and G).

Our parametric analysis below shows similar results after conditioning on a large number of potential confounders and using latent continuous SES rather than a binary SES.

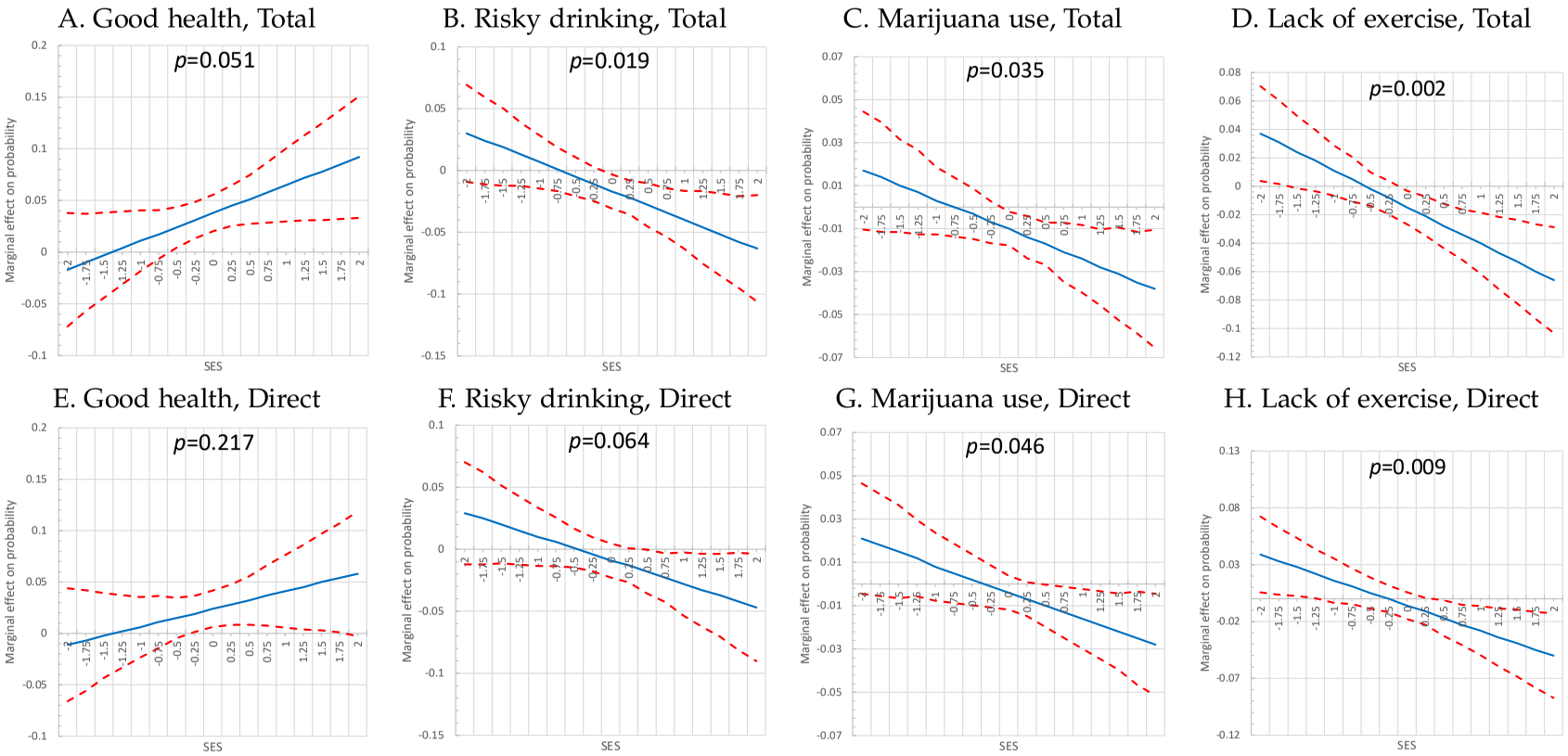
4.1.3 Main Model Estimates

We first estimate an association between EA PGI and health-related outcomes while allowing for an interaction between EA PGI and parental SES. Figure 3 visualizes estimated relationships by showing marginal effects of EA PGI on health-related outcomes as a function of standardized parental SES.

The upper panels of Figure 3 show the total effects of EA PGI as a function of standardized parental SES based on model (1). For the purpose of pairwise comparisons, the bottom panels show the corresponding direct effects, which are effects of EA PGI conditional on education, as defined by outcome model (2). The direct effect can be viewed as a part of the total effect that works through all possible mechanisms other than education.[20]

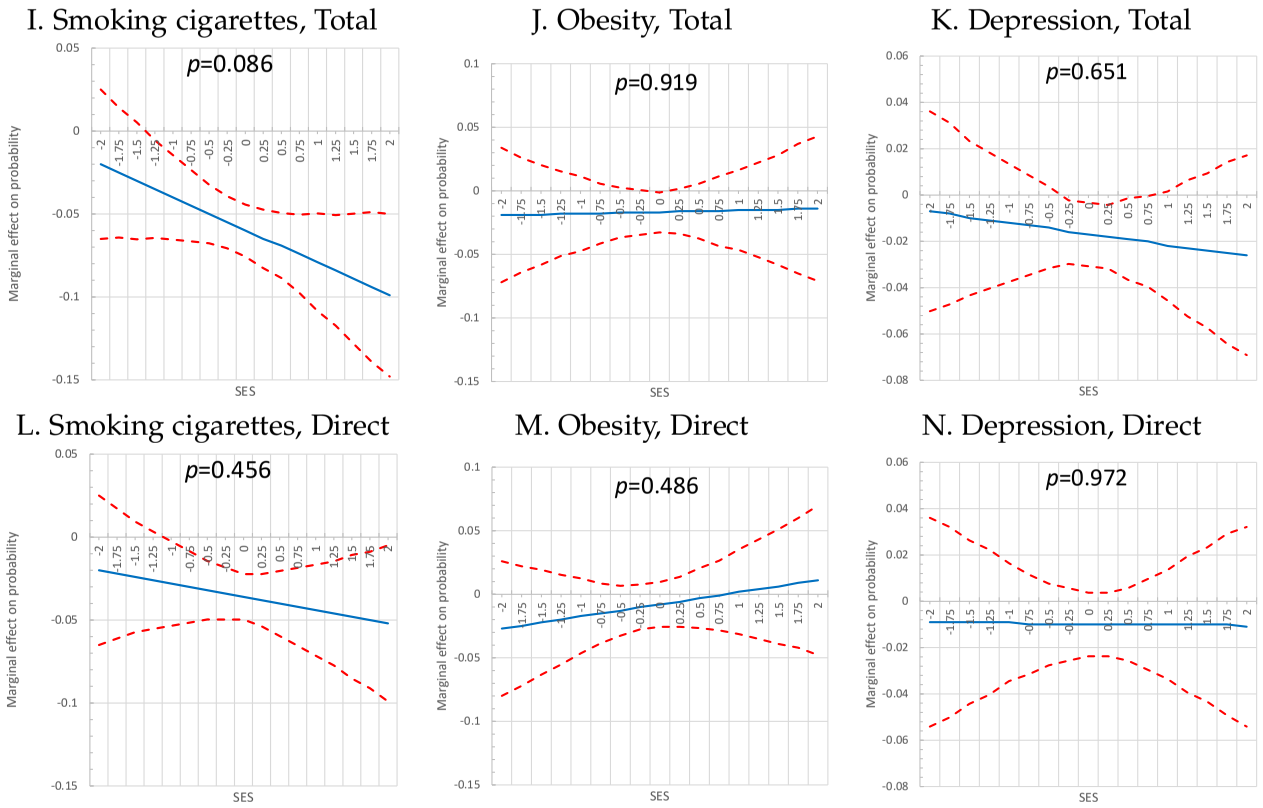
[19] See Note to Figure 2 for a definition of a bin scatter plot.
[20] See notes to Figure 3 for the exact definition of the estimated relationship.

Figure 3: Total and Direct Marginal Effects of EA PGI on Health-Related Outcomes in Young Adulthood as a Function of Parental SES



Notes: All panels present the results of logit models. Panels A–D present the results of the model (1). For them, the total effect of EA PGI is defined as $\hat{b}^*_{1k} + \hat{b}^*_{2k} \cdot SES$, where coefficients $\hat{b}^*_{1k}$ and $\hat{b}^*_{2k}$ are estimates of marginal effects from model (1); Superimposed p-values are for the two-tailed test H_0: $b^*_{2k} = 0$. Panels E–H present the corresponding results of model (2). The direct effect is defined as $\hat{c}^*_{3k} + \hat{c}^*_{4k} \cdot SES$, with p-values are shown for the two-tailed test H_0: $c^*_{4k} = 0$. Outcome variable for panels A and E shortly referred to as "good health" is defined as self-reported "excellent or very good health." Dashed lines represent the 95% Huber-White confidence intervals calculated using the delta method. Corresponding regression coefficients are documented in Tables A-5 and A-6 of the Web Appendix. See also Table A-4 for parameters of the measurement system (4). Calculations are based on the AddHealth Data.

Figure 3: Total and Direct Marginal Effects of EA PGI on Health-Related Outcomes in Young Adulthood as a Function of Parental SES (continued)



Notes: All panels present the results of logit models. Panels I–K present the results of outcome model (1). For them, the total effect of EA PGI is defined as $\hat{b}^*_{1k} + \hat{b}^*_{2k} \cdot SES$, where coefficients $\hat{b}^*_{1k}$ and $\hat{b}^*_{2k}$ are estimates of marginal effects from model (1); Superimposed p-values are for the two-tailed test H_0: $b^*_{2k} = 0$. Panels L–N present the corresponding results of outcome model (2). The direct effect is defined as $\hat{c}^*_{3k} + \hat{c}^*_{4k} \cdot SES$, with p-values are shown for the two-tailed test H_0: $c^*_{4k} = 0$. Dashed lines represent the 95% Huber-White confidence intervals calculated using the delta method. Corresponding regression coefficients are documented in Tables A-5 and A-6 of the Web Appendix. See also Table A-4 for parameters of the measurement system (4). Calculations are based on the AddHealth Data.

The comparison between total and direct effects is of interest because education is the most expected potential mechanism relating EA PGI to health and health behaviors. The interaction effects that we find for health and health behaviors have been found earlier for education as an outcome (and also confirmed by us below), as we discuss in Section 4.1.1. Therefore, it is useful to verify whether the effect that we find is fully driven by education or whether there is anything in this effect that is above and beyond the effects implied by education as a likely mechanism. As we argue below, even though education is an important mechanism, it explains our results only partially. There is a substantial direct effect for a number of outcomes that shows similar patterns.

Panel A of Figure 3 shows a marginal effect of EA PGI on having good health. From Panel A we can see that the effect of PGI on health increases with the level of parental SES. The p-values superimposed in each panel are for the test of the interaction between PGI and SES. For good health, this p-value is 0.051, which is borderline statistically significant at the 5% level.

Apart from p-values that allow us to test for the interaction effect directly, we observe results that are consistent with the interaction effect: a small and statistically insignificant effect of EA PGI at low levels of SES, as opposed to a large and statistically significant effect at high SES levels. For instance, an increase in EA PGI by one standard deviation is associated with about a 3.8 percentage points (PP) higher likelihood of having excellent or very good health at the average level of SES (SES=0), as we can see in Panel A. This association is stronger for those with SES=1 (6.5%), and weaker for SES below the average. For SES around -1 and below the effect of PGI is no longer statistically significant. Given that the probability of having excellent or very good health for this population is 0.625, these estimates imply strong effect sizes: at SES=0 the effect size is 6.1% (0.038/0.625), while at SES=1 the effect size is 10.4% (0.065/0.625). These strong effect size estimates should be interpreted with caution throughout this paper, though: the effect sizes are based on conditional associations, not causal effects.

Panel E shows the direct effect corresponding to total effect in Panels A. The superimposed p-value suggests that the direct effect's interaction term loses its statistical significance for the general health outcome. However, the effect of EA PGI is statistically significant in Panel E at the average level of SES and above and is not statistically significant at low level of SES, which is consistent with an interaction effect. Numerically, the direct effects are 3.8% (0.024/0.625), at SES=0, and 6.6% (0.041/0.625) at SES=1.

Panel B shows the effect of EA PGI on the probability of risky drinking of alcohol. The effect of EA PGI is statistically significant at the 5% level when SES level is in the vicinity of $SES = 0$ (the SES average) and above. At $SES = 0$ the effect is -1.7 PP. The effect decreases with SES level, and at $SES = 1$ the effect reaches -4.0 PP. Given that risky drinking is characteristic for 45.7% of young adults in our sample, the corresponding effect size is -3.7% at $SES = 0$ (-0.017/0.457) and -9.0% at $SES = 1$ (-0.040/0.457). The total interaction effect is also statistically significant (p=0.019).

The pattern of the corresponding direct effect in Panel F is similar to the total effect in Panel B but smaller in magnitude, something that we observe for a number of other health-related outcomes. The effect becomes statistically insignificant at $SES = 0$, but at $SES = 1$ the effect size is statistically significant and sizable (-6.1%=-0.028/0.457). The p-value for the direct interaction effect is 0.064.

We observe similar direct effects patterns for Panels C, D, and I, for which the effects are strong and statistically significant at high levels of SES, but significance is lost at the low levels. These patterns are in line with low p-values for the interaction effects.

For marijuana use in Panel C effect sizes are 10% at $SES = 0$ (0.010/0.096) and 25% at $SES = 1$ (0.024/0.096). The corresponding direct effect in Panel G at $SES = 0$ loses its statical significance, but the effect at $SES = 1$ declines to 16% (0.016/0.096) while remaining statistically significant (see Panel N). The direct interaction effect is statistically significant at the 5% level (p=0.046).

For the lack of physical exercise in Panel D, we estimate the effect size to be 12% at

$SES = 0$ (0.015/0.13) and 31% at $SES = 1$ (0.040/0.13). The corresponding direct effects in Panel H are the following: at $SES = 0$ the effect is no longer precisely determined. At $SES = 1$ the effect declines to -22% (-0.028/0.13). The direct interaction effect is statistically significant at the 1% level (p=0.009).

The total effect size for smoking cigarettes in Panel I is -23% at $SES = 0$ (0.060/0.261) and -30% at $SES = 1$ (0.079/0.261). Those remain statistically significant for the direct effect but decline to -14% (0.036/0.261) and -17% (0.044/0.261) correspondingly, which is consistent with interaction effect even though the interaction coefficient is not precisely determined for this case.

Finally, for two outcomes, obesity and depression, we observe statistically significant total effects of EA PGI at $SES = 0$ but see no evidence of the total interaction effects (see Panels J and K). Effect sizes at $SES = 0$ are 4.9% for obesity (0.017/0.343) and 8.9% for depression (0.017/0.089). We lose the statistical significance of the effect of EA PGI for both high and low SES levels. However, the result cannot be explained by an interaction with SES. Instead, the result is related to the decreased precision of our estimation away from $SES = 0$, a feature that is characteristic of all panels in Figure 3. For both obesity and depression, we observe no direct effects at any levels of SES and no interaction effect (see Panels J and L).

Discussion of the Results on the Association between EA PGI and Health-Related Outcomes as a Function of SES Overall, we can see that all of the estimated interaction effects that are statistically significant at least at the 10% level have the same sign as effects of EA PGI: positive for general health (Panel A), and negative for adverse health-related outcomes (Panels B, C, D, and I). Therefore, we can conclude that EA PGI tends to be more health-beneficial for those with higher SES. This result is consistent with the bottleneck hypothesis (Fletcher, 2019): low SES is a good proxy for severely constrained conditions in childhood. Large total effect sizes for all seven health-related outcomes in

Figure 3 imply the economic significance of the results reported in this paper.

The overall conclusion from the comparison between total and direct effects is that there are mechanisms above and beyond education that explain the PGI effect and its interaction with SES for a number of them. Even after controlling for education, we still find effects of EA PGI on health-related outcomes, as well as evidence of the interaction effect with SES.[21]

Our results complement those found by Bierut et al. (2018), as we find similar interaction effects but for a different type of PGI (we use EA PGI, not smoking PGI) and different outcomes (we use a variety of health-related outcomes, not only smoking cigarettes). This study also complements the results of Papageorge and Thom (2020), who use an interaction between EA PGI and childhood SES to study the determinants of education.

Limitations As discussed in the introduction, EA PGI correlates with family environment that we only partially control for, which likely creates an upward bias (by absolute value) in the estimated effect of EA PGI at $SES = 0$ (see coefficient b_{1k} in model (1)) relative to the true causal effect of genetic endowment. In addition, measurement error in EA PGI is expected to create an attenuation bias for this coefficient. While there is a benefit of these two biases canceling each other, this comes at the cost that the direction of the resulting bias is indeterminant.

For the interaction term (see coefficient b_{2k} in model (1)), no bias is created by the endogeneity as long as unobserved heterogeneity contributes to the error term in a linear way, as we demonstrate in the Web Appendix.[22] While we account for measurement error in SES in our factor model, measurement error in EA PGI leads to measurement error in SES-PGI interaction. Therefore, we can expect an attenuation bias in the estimated interaction due to this reason. Our statistically significant estimates of the interaction

[21] We also explore the role of controls that are correlated with SES and show that their role is quite small: the results barely change when we restrict the model to a smaller set of controls. See Figure A-1 of the Web Appendix.

[22] See Appendix B.

are found despite the attenuation bias.

Overall, estimates should be treated as associations that might be informative of the qualitative causal relationships.

Robustness of Our Results to Alternative Measures of SES and to Alternative Methods of Measure Aggregation We have surveyed measures of SES from the literature and have introduced comparable types of SES measures that are available in our data (see Section 2). In this section we show that our results are robust to using alternative sets of SES measures and to alternative methods of SES aggregation. This estimation is useful for justifying our model specification and for showing that our results are not driven by peculiarities of factor modeling.

For each health-related outcome we estimate the interaction between EA PGI and SES based on 18 alternative specifications regarding modeling SES (18 = 6 SES sets $\times$ 3 aggregation methods). While the results are similar across outcomes, the example of physical exercise is especially useful because for this outcome both coefficients for EA PGI and the interaction are estimated with high precision, which allows us to reliably study differences across models for both coefficients. We present the robustness check for physical exercise below, with the results for other outcomes available in the Web Appendix.[23]

Table 3 documents marginal associations between EA PGI and physical exercise for alternative sets 1–6 of SES measures. Panels A–C represent three alternative methods of SES aggregation: (A) a standardized latent factor, (B) a standardized equally-weighted index of measures, and (C) a binary aggregation: no single disadvantage (SES=1) vs. at least one disadvantage (SES=0) from the list of disadvantages that are marked in Panel E.

Our most preferred model specification corresponds to Set 1 of SES measures (three

[23]See Tables A-7–A-12.

Table 3: Robustness of Our Main Model to Alternative Specifications Involving Various Sets of SES Measures and Various Measure Aggregation Methods: Marginal Effects of EA PGI on Lack of Physical Exercise in Young Adulthood, Logit Model Results

	Set 1	Set 2	Set 3	Set 4	Set 5	Set 6
A: Aggregation Based on Latent Standardized SES Factor						
EA PGI	-0.015 **	-0.017 ***	-0.014 **	-0.020 ***	-0.013 **	-0.014 **
as SES=0	*(0.006)*	*(0.006)*	*(0.006)*	*(0.007)*	*(0.006)*	*(0.006)*
EA PGI	-0.026 ***	-0.033 ***	-0.017 *	-0.035 ***	-0.016 **	-0.024 ***
× SES Factor	*(0.008)*	*(0.009)*	*(0.009)*	*(0.013)*	*(0.008)*	*(0.008)*
B. Aggregation Based on Equally-Weighted Standardized Index of SES Measures						
EA PGI	-0.015 **	-0.015 **	-0.014 **	-0.014 **	-0.014 **	-0.015 **
as SES=0	*(0.006)*	*(0.006)*	*(0.006)*	*(0.006)*	*(0.006)*	*(0.006)*
EA PGI	-0.021 ***	-0.020 ***	-0.015 **	-0.012 **	-0.013 **	-0.020 ***
× SES Index	*(0.006)*	*(0.006)*	*(0.006)*	*(0.006)*	*(0.006)*	*(0.006)*
C. Aggregation Using a Binary SES: No Single Disadvantage from the List vs. At Least One						
EA PGI	-0.033 ***	-0.029 ***	-0.027 ***	-0.022 ***	-0.022 ***	-0.030 ***
as SES=0	*(0.009)*	*(0.008)*	*(0.008)*	*(0.007)*	*(0.007)*	*(0.008)*
EA PGI	-0.030 ***	-0.029 ***	-0.026 **	-0.026 **	-0.024 **	-0.029 ***
× Binary SES	*(0.011)*	*(0.010)*	*(0.011)*	*(0.012)*	*(0.012)*	*(0.010)*
D. Factor Model Goodness of Fit						
RMSEA	0.009	0.000	0.019	0.022	0.022	0.020
Prob(RMSEA $\leq$ .05)	1.000	1.000	0.989	1.000	0.981	1.000
CFI	0.999	1	0.998	0.854	0.997	0.996
TLI	0.971	1	0.857	0.445	0.800	0.861
E. Alternative Sets of Parental SES Measures						
Self-reported issues[(a)]	✓	✓	✓	✓	✓	✓
Parental college[(b)]	✓	✓				✓
Lowest income quintile[(c)]	✓		✓			
Income below median[(c)]					✓	✓

Notes: Set 1 represents the set of SES measures chosen for the main specification of the measurement system (4). Sets 2–6 are alternatives to Set 1. Panel A shows our main aggregation method, the factor model. Panels B and C offer alternative methods of measure aggregation. Asterisks indicate statistical significance level: ***, 1 % level; **, 5 % level; *, 10 % level. Calculations are based on the Add Health data. [(a)]Living in an unsafe neighborhood; having difficulties with paying bills; and household receiving government assistance. [(b)]At least one parent graduated from college. [(c)]Household income in subject's childhood. Similar tables for other outcomes that we study are available in the Web Appendix, A-7–A-12.

self-reported poverty issues + parental college + lowest income quintile) that are aggregated using method A (the factor model). Therefore, the upper left estimated coefficients in the table (-0.015* for the effect and -0.026*** for the interaction) correspond to Panel D of Figure 3 that we discuss above.

We chose our most preferred model based on well-established statistics: the Root Mean Square Error of Approximation (RMSEA), the Comparative Fit Index (CFI), and the Tucker-Lewis Index (TLI). For a well-specified model, the literature suggests the following thresholds that are based on simulations: $RMSEA \leq 0.05$, $CFA \geq 0.9$, and $TLI \geq 0.9$ (e.g., Bollen and Long, 1993; Kline, 2011).

Statistics in Panel D of Table 3 help us choose our most preferred model specification.[24] From Panel D we can see that condition $RMSEA \leq 0.05$ holds for all alternative model specifications. This result is supported by the estimated probability $Prob(RMSEA \leq 0.05)$, which varies for Models 1–6 from 0.981 to 1.000. Therefore, in terms of $RMSEA$, all six alternative factor models are satisfactory. In terms of CFI, all models are satisfactory but model 4, for which CFI is somewhat below the threshold. Finally, only Models 1 and 2 satisfy the condition $TLI \leq 0.9$. Therefore, only models 1 and 2 satisfy all three specification criteria, while other models deviate from at least one of the established thresholds. Choosing among models 1 and 2, we select model 1 for two reasons: (1) it informs our factor model with an additional important measure of SES, the family income; (2) it makes our model specification more comparable to the literature due to our use of this income measure (Ronda et al., 2020).

The differences in the estimates in Table 3 are explained not only by six different sets of measures but also by different methods of measure aggregation across Panels A, B, and C. The index in Panel B is based on equal weighting of measures, whereas factor models in Panel A make no assumption that all measures are equally informative. Despite these differences, the results in Panels A and B are hardly distinguishable given

[24]These statistics test the measurement system (4) estimated separately from the outcome equation, and so they are not specific to any particular health outcome.

the standard errors.

As for Panel C, even though its numerical results show a stronger quantitative deviation from Panel A, the qualitative results are the same. The numerical deviation from Panels A is an expected one because estimates in Panels A and C have different interpretations. The PGI coefficient in Panel C is the marginal effect not *at the average SES level*, as in Panels A, but *for the low-SES group*, which, moreover, is defined in a specific way (at least one disadvantage). Also, the interaction coefficients in Panel C show the effects of moving from a group with disadvantaged SES to a group with advantaged SES, not by one standard deviation of a continuous SES.

Despite the differences across models 1–6 and panels A–C, all 18 alternative cases show a strong negative effect of EA PGI and a strong negative interaction between SES and EA PGI. Some numerical variation in estimates is observed. However, as discussed above, this variation can be expected given somewhat different interpretations of these alternative estimates.

Sibling Fixed Effects It would be ideal to rely on the sibling fixed effect to establish the causal effect of EA PGI because Mendel's laws imply that genetic differences between siblings are uncorrelated with the environment (Morris et al., 2020). Therefore, within-sibship estimates of PGIs could be interpreted as the effects of one's own genetic endowments. However, we show that the sample size that we have (200 sibling pairs who are not identical twins) is by far insufficient to follow this route because of low statistical power. The dramatic fall in statistical power for the sibling fixed effects estimator compared to OLS and its generalizations is not surprising: estimation sample size sharply declines (in our case, from 3700 to 200) and variation of variables gets restricted to within-sibship one only.[25]

[25]We calculated the sample size to achieve the desired statistical power of 0.8 for a two-sided test with a significance level of 5%. Our calculation takes into account the dramatically diminished identifying variation due to using within-family variation only. Under the assumption that estimated conditional associations are informative of causal

The impractically low statistical power of family fixed effects when estimating the effect of a PGI is in line with the literature based on datasets of comparable and or larger sample sizes. For example, Amin et al. (2019) report insufficient statistical power for family fixed effects when using the same Add Health dataset, but a different PGI index and a different outcome. Ronda et al. (2020) also lack sufficient statistical power for the family fixed effect of the EA PGI on education and skill capital, despite using a sample of siblings that is more than three times larger than ours in their study of a Danish population. In line with our power calculations, we find that all sibling fixed effects estimates that we calculate for AddHealth are not precisely determined.[26]

4.1.4 The Mechanisms

To better understand the effects of EA PGI on health and health behaviors in young adulthood, the effects we have discussed above, we provide suggestive evidence for the mechanisms behind the estimated effects.[27]

We explore potential mechanisms from two time periods: early life and young adulthood. The early life potential mechanisms have the advantage of being observed long before health-related outcomes in young adulthood, which minimizes the likelihood of capturing the reverse causal effect. The young adulthood measurements supplement the early life ones by adding previously unavailable information. However, because they are measured simultaneously with health-related outcomes that we attempt to explain, this implies that these suggested mechanisms should be interpreted with extra caution.

effects, we would need sample sizes N=700–2,800 to identify effects of EA PGI, depending on the type of health-related outcomes. To identify the EA PGI-SES interaction effect, we would need N=2,800–47,000 observations. However, if the causal effects are smaller by absolute value than the estimated conditional associations, even larger sample sizes would be needed to identify the effects.

[26]These estimates are uninformative given low statistical power, but we follow the literature and still document them for readers' reference in the Web Appendix (see Table A-13).

[27]In a more technical companion paper, we explicitly incorporate the mechanisms into the model of health formation (Savelyev and Bolyard, 2024).

Overall, our aim in this exploratory study is to identify as many potential mechanisms as possible, with testing for their possible causal status left for future research.

Health Behaviors The partition between health-related outcomes (health and health behaviors in young adulthood) and the mechanisms of health formation is somewhat blurred. For instance, risky drinking of alcohol, a health behavior, could be viewed both as a health-related outcome in young adulthood and as a mechanism behind the formation of general health in young adulthood.

This observation implies that we already have several results on potential mechanisms behind the positive effect of EA PGI on general health, all documented in Figure 3, which we have discussed above. Specifically, the results for the positive effect of EA PGI on general health (in Panel A the effect is above zero at the average level of SES (SES=0)) can be explained by the negative effects of EA PGI on risky drinking, marijuana use, lack of physical exercise, smoking cigarettes, and depression (see negative effects at SES=0 in Panels B, C, D, I, and K).

The results in Figure 3 also offer suggestive pathways for the positive interaction between SES and EA PGI in general health formation that we can see in Panel A (see the positively-sloped line, $p = 0.051$). One possible reason for this positive interaction could be the negative interactions between SES and EA PGI for risky drinking of alcohol, marijuana use, lack of exercise, and smoking cigarettes (see the negatively-sloped lines in Panels B, C, D, and I).

However, the possible effects of health behaviors on health stock suggested above might be small or negligible given that we study health stock in early adulthood, ages 24–32.

Early Life Mechanisms Figure 4 presents estimates of model (1), with early life potential mechanisms serving as outcomes, Y_k. We can see that at the average SES level ($SES = 0$), EA PGI is positively associated with cognitive skills (Panel A), early general

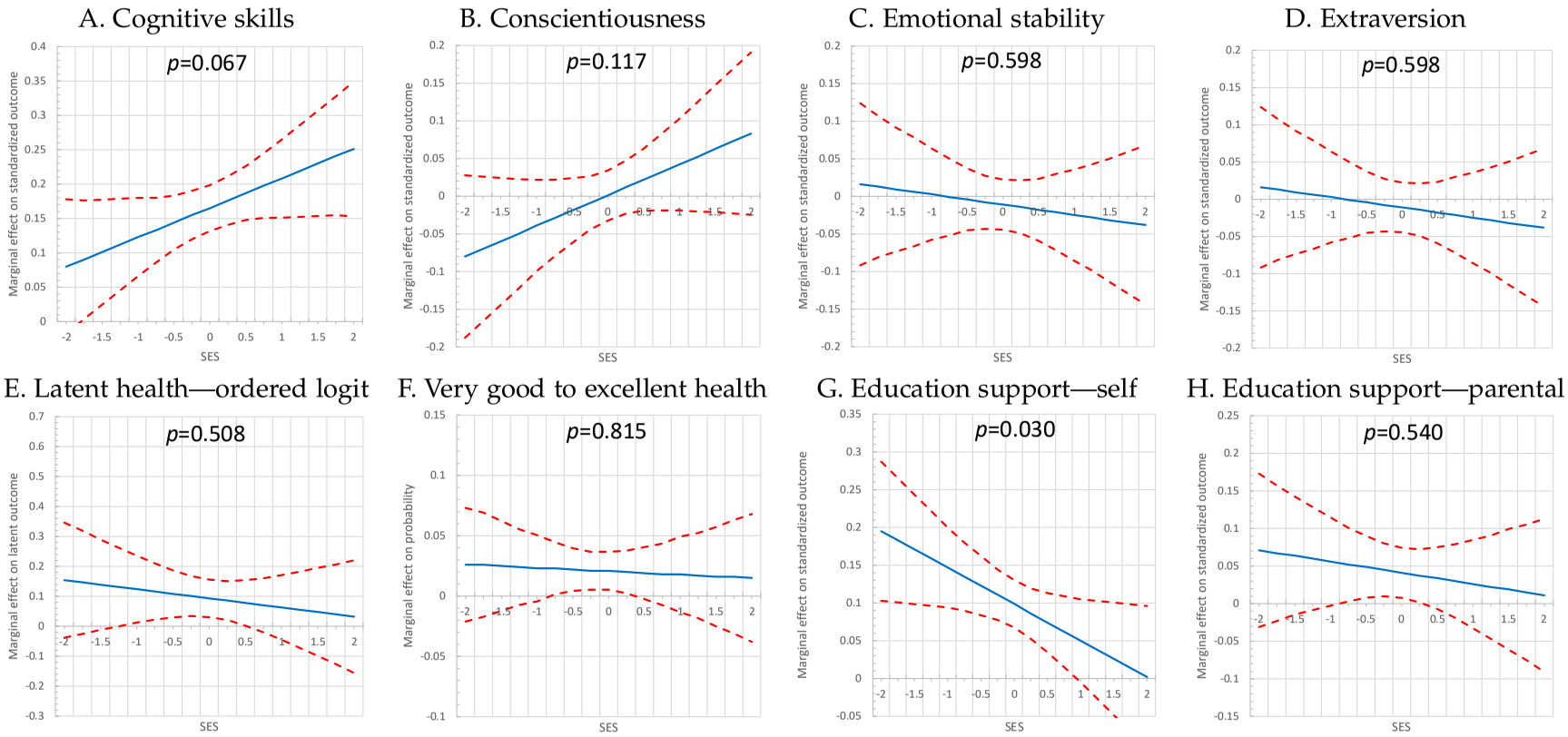
health (Panels E and F), the child's positive attitude towards their own education (Panel G), and parental support of the child's education (Panel H). These suggested mechanisms are possible explanations behind the positive effect of EA PGI on health in young adulthood.

It should be noted that our estimates might be biased due to genetic nurture, as discussed above. This especially applies to parental support of education. We offer two interpretations of the observed association, one genetic casual and another spurious. The genetic causal explanation of the positive relationship between EA PGI and the parental support of the child's education is that parents observe early outcomes of the child's genetic endowment for education, such as good performance at school, which makes them more supportive of the child's further education. The spurious interpretation is that EA PGI captures non-inherited parental traits that correlate with parental propensity to support their child's education. These two explanations are not mutually exclusive, which means that we cannot rule out a genetic causal component behind the estimated associations.

Apart from explaining the mechanisms behind the effect of EA PGI on health-related outcomes at the average SES level, we seek to explain the mechanisms behind the interaction between EA PGI and SES to better understand the origins of the interaction effect. However, among early mechanisms that we sturdy, only the results for cognitive skills (see Panel A) show a positive and statistically significant interaction that could explain the main results.

In Panel G of Figure 4, we can see that while EA PGI is associated with self-motivation for own education at the average level of SES, this association is not increasing with SES but declining. This interaction sign is the opposite of the one that would explain the positive interaction for general health. We provide the following potential interpretation of this result: high-SES children expect to get a high level of education regardless of whether their genetic endowment is low or high because of social expectations in their

Figure 4: Marginal Conditional Associations Between EA PGI and Potential Early Life Health Mechanisms as a Function of Parental SES: Cognitive Skills, Noncognitive Skills, Health, and Education Support



Notes: Marginal effects on outcomes are shown as a function of standardized latent SES factor. The results are based on estimating the system of equations (1,4). Panels correspond to the following type of outcome model (1): A–D, linear-in-parameters; E, ordered logit; F–H, logit. Dashed lines represent the 95% Huber-White confidence intervals calculated using the delta method. Corresponding regression coefficients are documented in Table A-14 of the Web Appendix. See also Table A-4 for parameters of the measurement system (4). Calculations are based on the AddHealth Data.

SES-group and available parental resources. For low-SES students, social expectations for education and available resources are smaller, so genetic endowments for education, which allow them to reduce education costs and overcome obstacles, play a larger part in their educational motivation.

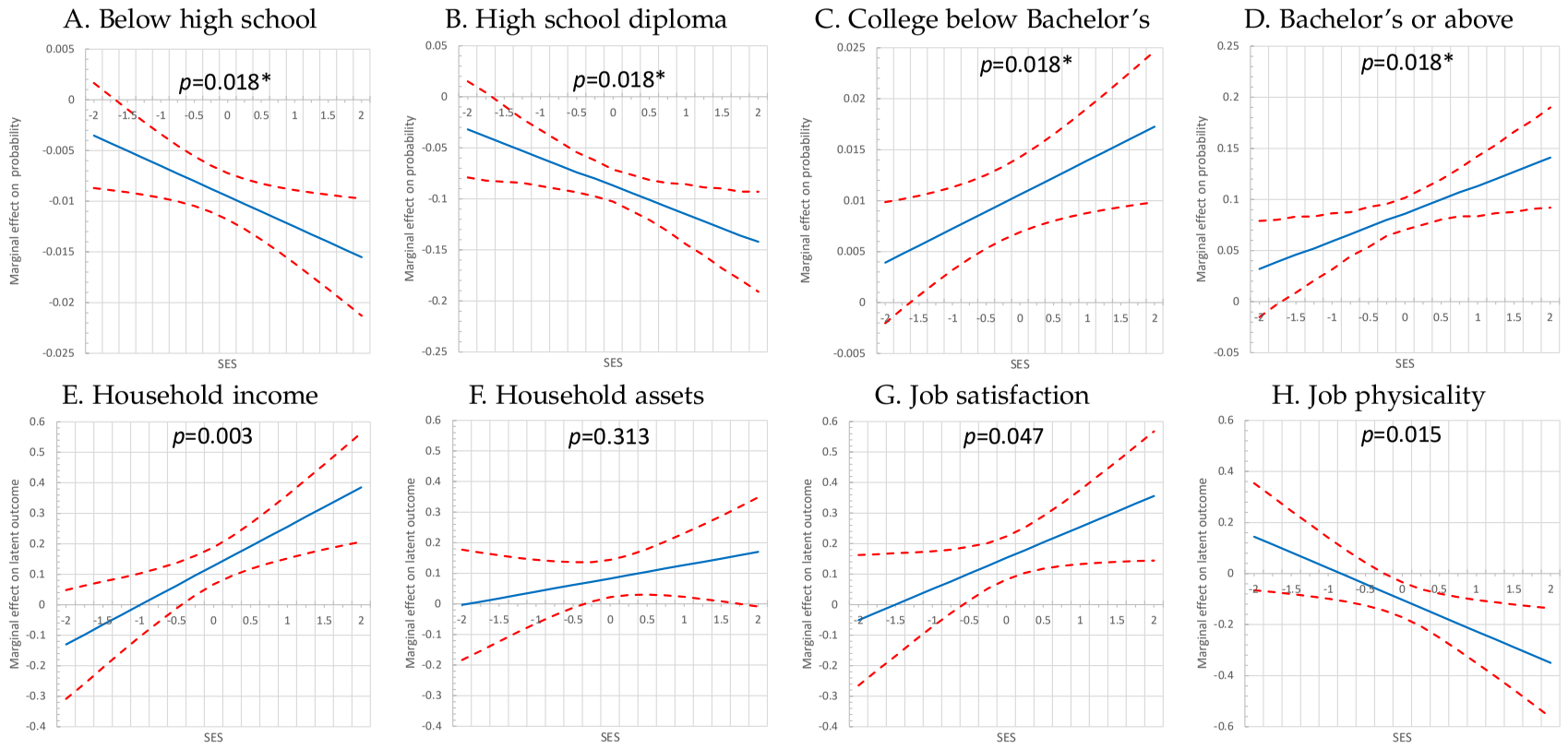
For all other outcomes, which include noncognitive skills and early health, we do not observe the interaction effect.

Early Addictive Behaviors Given that some health behaviors, such as smoking, are addictive, we also explore the role of early health behaviors as possible mechanisms of later health behaviors. We first regress early health behaviors from wave I on EA PGI, SES, and EA PGI×SES conditional on other controls and find that most early measures of drinking alcohol, smoking cigarettes, and being overweight in adolescence are predicted by EA PGI. However, the interaction with SES is not precisely determined.[28] Secondly, we regress health behaviors in adulthood on EA PGI, SES, and EA PGI×SES conditional on corresponding early behaviors and other controls, and compare these results with our main model, which does not condition on early behaviors.[29]

We find that early behaviors are predictive of later behaviors, and that associations between EA PGI and health-related outcomes in young adulthood tend to slightly decline when controls for early health behaviors are added. These results imply that early behaviors represent one channel that partly explains the association between EA PGI on later behaviors. However, there is a substantial part of the association that can be expected to work through other channels. Also, early health behaviors do not explain the interaction with SES that we observe for health behaviors in young adulthood, which implies that the interaction works through channels other than early addictive behaviors.

[28]See Table A-15 of the Web Appendix.
[29]See Table A-16 of the Web Appendix.

Figure 5: Marginal Conditional Associations Between EA PGI and Potential Health Mechanisms in Young Adulthood as a Function of Parental SES: Education, Occupation, and Wealth



Notes: Marginal effects on outcomes are shown as a function of standardized latent SES factor. The results are based on the factor model (1,4). Panels A–D are all based on the same ordered logit model of education (1), for which coefficients are reported in Table A-17 of the Web Appendix. Panels E–H are based on ordered logit models documented in Table A-18. See also Table A-4 for parameters of the measurement system (4). Dashed lines represent the 95% Huber-White confidence intervals calculated using the delta method. Calculations are based on the AddHealth Data. (*)In Panels A–D, we report a p-value for the PGI-SES interaction coefficient from the underlying ordered logit model of education choice. Therefore, the same p-value is shared across all educational thresholds.

Education Panels A–D of Figure 5 show the marginal effects of EA PGI on the probabilities of achieving different highest education levels. As in previous figures, the marginal effects are plotted as functions of standardized SES. These four graphs are based on the same underlying ordered logit model of education (1), estimated simultaneously with the measurement system (4).

As we can see from the figure, the EA PGI makes lower levels of education—education below high school and high school diploma—less likely (see Panels A and B), and higher levels of education—college below bachelor's and bachelor's or above—more likely. For all four outcomes, the interaction with SES makes the education-enhancing effects of EA PGI stronger. All results are precisely determined and effect sizes are large. At the average SES, effect sizes of EA PGI are the following: 20% decline for education below high school ($= -0.0095/0.048$), 21% decline for high school diploma ($= -0.087/0.415$), 6% increase for college below bachelor's ($= 0.0106/0.174$), and 24% increase for bachelor's or above ($= 0.086/0.363$).[30]

These results for education are expected because EA PGI is specifically designed to predict years of formal education and because positive interaction with SES is documented in the literature, as we discuss in Section 4.1.1 (Fletcher, 2019; Papageorge and Thom, 2020; Ronda et al., 2020). Therefore, results in Panels A—D of Figure 5 serve two purposes: (1) to support the existing literature on EA PGI-SES interaction with additional evidence; (2) to confirm that these expected relationships are true for a specific population that we study and, therefore, can help us explain the mechanisms behind the effects of EA PGI on health.

Occupation and Wealth Finally, in Panels E–H of Figure 5 we explore the role of outcomes related to occupation and wealth as potential mechanisms of the health effects. We can see that for medium and high SES levels EA PGI is positively related to house-

[30]See Table A-17 of the Web Appendix for effect sizes and estimates behind Figure A-17.

hold income, household assets, and job satisfaction. Job physicality is affected negatively. However, none of these effects take place at low SES levels.

These findings are consistent with our results in Figure 3, as they suggest the mechanisms behind the relationship between EA PGI and health-related outcomes and its interaction with SES. The effects on income (a flow) and assets (a related stock) are consistent with Case and Deaton (2005), who argue that there is a direct protective effect of income on health, and with a number of other authors who make similar claims.[31]

Job satisfaction, which is related to overall life satisfaction and the individual's perception of the value of their own life, is another potential mechanism of health formation (Savelyev, 2022). Finally, job physicality is known to be related to worse health levels and quicker heath declines despite health-related selection effects that are typical for physical jobs (Case and Deaton, 2005; DeLeire and Levy, 2004; Fletcher et al., 2011; Ravesteijn et al., 2018).

4.2 Education and Health

4.2.1 Motivation and Contributions to the Literature

This section explains our contribution to the debate about the effect of education on health (see Galama et al. (2018) and Grossman (2022) for recent reviews). In this literature, apart from regressions conditional on observable controls and propensity score methods, there are three major methods that attempt to identify the effect of education on health-related outcomes: (1) randomized controlled trials (RTCs) (2) natural experiments; (3) family fixed effects or twin fixed effects; (4) the explicit modeling of unobserved heterogeneity.

[31] There is no consensus in the literature regarding the causal status of the relationship between wealth and health. A number of papers claim a positive effect of wealth on health-related outcomes (Frijters et al., 2005; Gardner and Oswald, 2007; Lindahl, 2005; Schwandt, 2018), a number of others find negative effects (Kippersluis and Galama, 2014; Snyder and Evans, 2006), and there are several papers that find either no effects or minor effects (Apouey and Clark, 2015; Cesarini et al., 2016; Kim and Ruhm, 2012).

Below we briefly explain the results of literatures (1–4) to motivate our study and highlight our contributions. In literature 1, the use of RTCs in education is usually limited to early childhood education (Conti et al., 2016).

As for natural experiments used in literature 2, they have the important benefit of a well-defined source of variation. However, this benefit comes with a cost, as these methods only identify the Local Average Treatment Effect (LATE) and may suffer from lack of validity, lack of monotonicity, low statistical power, and weakness of instruments (e.g., Heckman and Vytlacil, 2007).

Literature 2 mostly relies on changes in compulsory schooling laws as a source of exogenous variation, though rare exceptions exist, like the use of military draft avoidance (Buckles et al., 2016). The results of these papers differ greatly. For instance, some find a strong effect of education on health-related outcomes (e.g., Barcellos et al., 2018; Lleras-Muney, 2005; van Kippersluis et al., 2011), while others find none (e.g., Albouy and Lequien, 2009; Clark and Royer, 2013; Mazumder, 2008; Meghir et al., 2018). Likely reasons for these differences include the weakness of compulsory schooling laws as an instrument for a number of countries including the US, confounding influences of other reforms and trends, and differences in effects by population, cohort, and sex (Galama et al., 2018).

Literature 3 relies on differencing out a large number of unobserved confounders that are shared by twins or siblings. However, estimates based on these methods are highly sensitive to measurement error in education (e.g., Ashenfelter and Krueger, 1994) and could be confounded by unobserved health shocks among siblings or twins in their early life. Finally, establishing the external validity of twin-based results could be challenging. Just as for literature 1, the results of literature 2 are contradictory. Some papers find substantial effects (e.g., Lundborg et al., 2016; Savelyev et al., 2022; van den Berg et al., 2015), while others find little to no effect (e.g., Amin et al., 2015; Behrman et al., 2011; Madsen et al., 2010). Differences in the results could be partly related to different model

specifications and partly due to differences by population, cohort, and sex.

Literature 4 explicitly models relationships between observed and unobserved confounders, education, and health-related outcomes. These methods preserve statistical power better than methods 2 and 3. Also, unlike literature 2, literature 4 attempts to estimate the Average Treatment Effect (ATE) rather than the Local Average Treatment Effect (LATE). The biggest concern with literature 4 is its ability to adequately account for possible remaining unobserved confounders. Important confounders that are explicitly accounted for in literature (3), often through latent factor modeling, including major human capabilities in early life: health, cognitive skills, and noncognitive skills (Bijwaard et al., 2015; Conti and Heckman, 2010; Savelyev and Tan, 2019). Further, Savelyev (2022) and Hong, Savelyev, and Tan (2020) also account for latent unobserved heterogeneity on top of latent human capabilities.

To summarise, the alternative literatures discussed above have their advantages and disadvantages. Literature (1) has the most persuasive source of exogenous variation, but it is quite limited due to ethical considerations and high costs of RTCs. Literatures (2) and (3) are both characterized by a number of econometric issues and contradictory results. The results of literature (3) are more consistent, as authors tend to find positive effects of education on health, however, there is a concern that certain unobserved confounders are still not fully controlled for. This paper diminishes concerns about the results from literature (3) by controlling for a large number of PGIs, which we use to proxy genetic endowments for skills, physical health, mental health, and home environment. We establish that the association between education and health survives controlling for such proxies.

The current discussion on whether education causally affects health resembles a historical discussion on whether education causally affects wages (Griliches, 1977). In both discussions, one side of the debate argues that there is no causal effect of education on the outcome despite a strong association between the two. As Gronau (2005) reports in

his detailed survey of Grilliches' major contribution to human capital theory, Grilliches played an instrumental role in repelling the "revisionists'" claim that the correlation between education and wages was only an artifact of ability and family background and in showing that the bias in education coefficient in earnings regression was downward, not upward, as confirmed by future research. Back in the 1970s, Grilliches lacked access to quality measures of endowments. However, he provided an influential critique of his opponents' methods, which suffered from previously unrecognized major endogeneity issues. In contrast, our paper takes advantage of quality molecular genetic measures of endowments to help resolve a similar controversy.

4.2.2 Empirical Results on Education and Health

The well-known strong association between education and health can possibly be explained by uncontrolled confounders, or "third variables," that may include physical and mental health earlier in life (e.g., Grossman, 2000). Relatedly, several authors emphasize the importance of genetic confounders of this relationship (e.g., Boardman et al., 2015; Conti and Heckman, 2010).

In this section, we explore the confounding role of genetic endowments for skills, general health, and mental health, which we proxy using 17 PGIs: an EA PGI as a proxy for skills, plus nine PGIs proxying outcomes that are related to physical health, and seven PGIs proxying different mental health measures. As discussed in the introduction, PGIs do not only proxy skill endowments but also family environment, which allows us to proxy confounding variation even better.

The PGIs that we use typically show some pairwise correlation, but we control for all of them to maximize the potentially confounding variation that we control for.[32]

[32]The general health-related PGIs proxy the following nine outcomes: (1) coronary artery disease; (2) myocardial infarction; (3) low-density lipoprotein cholesterol; (4) triglycerides; (5) type II diabetes; (6) BMI; (7) waist-to-hip ratio; (8) height; and (9) smoking. The mental health-related PGIs proxy the following seven measures of mental health: (1) depression; (2) neuroticism; (3) attention-deficit disorder; (4) bipolar disorder;

Table 4: Marginal Associations Between Education Categories and Health-Related Outcomes in Young Adulthood

	Excellent or very good health	Risky drinking	Marijuana use	Lack of exercise	Smoking cigaretts	Obesity	Depression	Joint test[a]
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
A. Education[b]								
Below High School	-0.260***	0.095***	0.068***	0.063**	0.342***	0.020	0.079**	83.9
	(0.055)	*(0.036)*	*(0.020)*	*(0.030)*	*(0.041)*	*(0.054)*	*(0.038)*	*[0.000]*
High School Diploma	-0.132***	0.083***	0.060***	0.079***	0.248***	0.069***	0.069***	212.4
	(0.024)	*(0.018)*	*(0.011)*	*(0.014)*	*(0.021)*	*(0.023)*	*(0.018)*	*[0.000]*
College below Bachelor's	-0.095***	0.070***	0.037**	0.054***	0.241***	0.072***	0.054***	135.5
	(0.028)	*(0.021)*	*(0.015)*	*(0.017)*	*(0.023)*	*(0.027)*	*(0.021)*	*[0.000]*
Joint test[c]								
Wald stat.	39.0	19.3	29.8	29.0	143.1	9.7	14.0	
p-value	*[0.000]*	*[0.000]*	*[0.000]*	*[0.000]*	*[0.000]*	*[0.022]*	*[0.003]*	
B. Education × SES								
Joint test[d]								
Wald stat.	3.09	6.95	9.42	0.16	5.49	1.06	0.40	
p-value	*[0.378]*	*[0.074]*	*[0.024]*	*[0.983]*	*[0.140]*	*[0.786]*	*[0.940]*	

Notes: Marginal associations between education levels and probabilities of corresponding health-related outcomes are reported based on the logit factor model (3,4). The omitted education category is "Bachelor's degree or above." Asterisks indicate statistical significance level: ***, 1 % level; **, 5 % level; *, 10 % level. Add Health data are used. Sample size is 3709. The results are conditional on: (1) 17 polygenic indices that measure genetic endowments for education, general health, and mental health; squares of these indices and their interactions with SES; (2) Early cognitive and noncognitive skills; (3) A full set of observable controls presented in Table 2. [a]Wald test whether all seven coefficients in the corresponding row are jointly zero from estimating models (1-7) simultaneously. Wald test statistic is shown, with p-values reported in square brackets. [b]Effects of education are calculated at the average level of SES and other controls. [c]Testing jointly across the corresponding column whether all educational effects are zero. [d]Testing jointly whether all interaction effects are zero (three education binary variables, each multiplied by SES).

As discussed above, proxies are useful as they help either eliminate or mitigate the omitted variable bias. In addition, they reduce the residual variance. There is no need for proxies to be perfect measures of the unobserved variables. We show that the conditional association between education and health remains strong upon controlling for 17 proxies of the expected important confounders.

Associations Conditional on Multiple PGIs Table 4, Panel A, shows the marginal effects of educational categories on health-related outcomes that are estimated based on model (3). The presented effects are relative to the effect of "bachelor's degree or above," which is the omitted category. The novelty of these results is that they are conditional on proxies of genetic confounders that historically have been viewed as unobservables, but recently their measurement has became available due to major advances in genotyping and PGI construction techniques. These confounders include EA PGI, nine types of PGIs related to aspects of physical health, and seven types of mental health PGIs.[33]

All signs of estimated associations are consistent with the health-beneficial role of education. Among 21 individual t-tests in Panel A, only one cannot be rejected at the 5% level.[34] The results based on individual tests are supported by joint tests, all of which are rejected at the 1% level of significance. Those include Wald tests of two types: (1) Joint tests across all seven health-related outcomes, which are performed for each of the three education levels (see column (8)); (2) Joint tests across all three education levels, which are performed for each of the seven health-related outcomes (see Wald tests statistics in the bottom of Panel A);

Another result of Table 4 is a joint test for the interaction between educational cate-

(5) major depressive disorder; (6) schizophrenia; (7) mental health cross disorder.

[33]See Tables A-2 and A-3 of the Web Appendix for measures of PGI that describe general and mental health and for correlations among them.

[34]The test that we fail to reject is for the lowest education level category, "below high school", which is characterized by a small population (about 5% of the sample) and, therefore, the reduced precision of estimation (see Row 1 of Panel A for outcome (5)).

gories and parental SES, presented in Panel B. This interaction appears at best weak.[35] Therefore, while the effect of EA PGI on education depends on SES, as we have seen above, the effect of education on health is not dependent on SES. Therefore, the effect of education on health cannot explain the strong EA PGI-SES interaction that we observe for health-related outcomes in Figure 3.

Our failure to establish an SES interaction with education can be explained in at least two ways. First, this result is consistent with the prime importance of early development. Early development plays a key role in human development over the life cycle because of reasons such as critical and sensitive periods in childhood, dynamic complementarity, and self-productivity (Heckman, 2007). As we have shown earlier, EA PGI, which is a strong proxy of early life skills, strongly interacts with family SES in predicting education, health, and health behaviors. In contrast, an interaction between postcompulsory education and family SES conditional on EA PGI is an example of a skill-SES interaction in young adulthood. Second, in young adulthood, parental SES is a feature from the past that becomes increasingly less relevant with age, as the subject's own SES may gradually deviate from parental one. Any of these reasons or a combination of them might be behind the lack of education-SES interaction.

Relative Confounding Roles of Various Types of Controls We also contribute to understanding the relative confounding role of various sets of controls, with a special emphasis on the role of PGIs that predict education and health. These sets of controls are defined in Panel D of Table 5. We explore the following groups of controls to be defined below: traditional controls, skills, and genoeconomic controls.

By "traditional controls" we denote observable controls that have been used in economic literature for decades, such as biological sex, geographic location, and family background (see background controls that are documented in Table 2, excluding genetic

[35]Because of weak joint test results, we show neither individual coefficients nor the t-tests in Panel B to save space.

Table 5: Marginal Effects of Education on Self-Reported Health Depending on the Set of Controls, Logit Model Estimates

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
A. Education							
Below High School	-0.260 ***	-0.257 ***	-0.302 ***	-0.297 ***	-0.295 ***	-0.342 ***	-0.368 ***
	(0.055)	-0.154	*(0.051)*	*(0.035)*	*(0.041)*	*(0.036)*	*(0.027)*
High School Diploma	-0.132 ***	-0.154 ***	-0.161 ***	-0.188 ***	-0.189 ***	-0.224 ***	-0.245 ***
	(0.024)	*(0.017)*	*(0.023)*	*(0.016)*	*(0.020)*	*(0.020)*	*(0.014)*
College below Bachelor's	-0.095 ***	-0.119 ***	-0.116 ***	-0.146 ***	-0.144 ***	-0.160 ***	-0.190 ***
	(0.028)	*(0.020)*	*(0.027)*	*(0.019)*	*(0.025)*	*(0.024)*	*(0.018)*
B. Average change in education coefficients presented above relative to...							
Column 7	*42%*	*35%*	*30%*	*22%*	*22%*	*10%*	*0%*
Column 4	*26%*	*17%*	*11%*	*0%*	-	-	-
Column 2	*11%*	*0%*	-	-	-	-	-
C. Average change in education coefficients for all seven outcomes[a] relative to...							
Column 7	*28%*	*23%*	*18%*	*12%*	*13%*	*6%*	*0%*
Column 4	*10%*	*14%*	*8%*	*0%*	-	-	-
Column 2	*7%*	*0%*	-	-	-	-	-
D. Controls							
Traditional[b]	✓	✓	✓	✓			
Skills[c]	✓	✓			✓		
Genoeconomic[d]	✓		✓			✓	

Notes: The binary outcome is "Excellent or Very Good Health." Column 1 corresponds to the unrestricted model (3). All other columns are restricted versions of the same model, with certain sets of controls omitted, as shown by checkmarks in the bottom of the table. Column 7 corresponds to a regression of the outcome on education dummies only. [a]The percentages that are being averaged are documented in the following Tables for health-related outcomes other than general health: Tables A-19–A-24 of the Web Appendix. [b]Background controls that are documented in Table 2 including SES factor, but excluding genetic ancestry PCs. [c]Traditionally-measured cognitive and noncognitive skills, $\boldsymbol{S}$; [d]Data based on genotyping: 17 PGIs and 10 genetic ancestry PCs.

ancestry PCs). Plus, we include the SES factor in a set of traditional controls, at SES factor is identified of traditional observed measures of parental disadvantage.

A set of controls denoted as "skills" includes early cognitive and noncognitive skills. These controls are emphasized by a new field called the economics of human development, in which latent cognitive and noncognitive skills are modelled jointly using factor analysis to recognize the importance of multidimensional human capabilities and to account for measurement error in measures of skills (e.g., Heckman et al., 2013, 2006).[36]

Our final group of controls, labeled as "genoeconomic," are those recently introduced to economic research by genoeconomists (Benjamin et al., 2012). In our paper, this group includes 17 PGIs that proxy genetic education and health and the first 10 principal components of genetic data, which are standard controls for ethnic differences.[37] Those controls are based on genotyping combined with new techniques of processing genomic measurements.

Panel A of Table 5 shows the marginal effects of education categories on self-reported good health by type of controls. Panel B summarizes the differences in panel A coefficients relative to various baseline models.

Column 1 in Table 5 shows the results of the unrestricted model (3), the same estimates as in Panel 1 of Table 4, while columns 2–7 display the results of restricted models, with restrictions as defined in Panel D.

The most basic model, a regression of outcomes on education dummies only, is shown in column 7. As we can see in Panel B, controls decrease the absolute value of regression coefficients (on average) relative to the no-controls model in column 7, the following

[36] Arguably, cognitive skills can be also classified as "traditional controls," because IQ has been used by economists as a proxy for ability for a long time. However, the literature in economics of human development makes a step forward in its study of cognitive skills. While recognizing this classification challenge, we group cognitive and noncognitive skills together primarily to learn the overall confounding contribution of multidimensional early skills that can be measured using traditional data collection methods, not genotyping.

[37] PCs are standard controls that accompany PGIs, as ethnic differences are expected confounders of genetic effects.

way: genoeconomic controls only, 10% (see column 6); skill controls only, 22% (see column 5); traditional controls only, 22% (see column 4). Using all these controls together gives us a 42% change (see column 1), which is smaller than the sum of the above percentages ($42 < 54 = 10 + 22 + 22$) because different types of controls listed in Panel D are correlated.

The next interesting point of comparison is a model with traditional controls shown in column (4). Conditional on traditional controls, we study the contribution of controls introduced by new literatures that brought childhood skills and genotyping techniques into the picture. As seen in Panel B, relative to a model that has traditional controls only, other sets of controls decrease the regression coefficients of education as follows: skill controls, 17%; genoeconomic controls, 11%, and 26% if both are used. Again, for the same reason as above, using both types of controls creates a smaller change than the sum of changes from each type ($26 < 28 = 17 + 11$). Finally, relative to a model that controls for both traditional controls and skills (column 2), controls based on genotyping change the estimates by 11%.

In addition, Panel C shows a summary of similar results for all seven health-related outcomes.[38] Therefore, in Panel C we observe a central tendency for health-related outcomes. We can see that, while numbers in Panels B and C are somewhat different numerically, they are qualitatively similar. We see that controls based on genotyping decrease the estimates of the effect of education by 7% on average.

To summarize, after controlling for traditional background variables and skills, the incremental change in associations due to missing genoeconomic proxies for health endowments, skill endowments, and home environment is 11% for general health and 7% on average for health-related outcomes. While these biases are sizable, they are at odds with the hypothesis that the strong association between education and health is entirely

[38]The average is taken over 21 regression coefficients, which are coefficients for three education dummies over seven health-related outcomes. See Tables A-19–A-24 of the Web Appendix for the results that are summarized in Panel C.

driven by unobserved confounders.

Our results are in line with a related paper by Heckman et al. (2018) (HHV), which focuses on dynamic aspects of schooling choice. We complement their discussion of confounding factors of the effect of education on health. We are in agreement with HHV that education affects health and smoking even after accounting for confounders in various ways. In particular, we confirm that multidimensional skills are major confounders and that accounting for them preserves a strong and statistically significant association between education and health.

Another closely related paper is by Cutler and Lleras-Muney (2010) (CLM), who summarize the decrease in the association between education and health behaviors when various factors are controlled, including those that are simultaneously determined with health behaviors, such as current income. They conclude that income, health insurance, and family background can account for about 30% of the education-health gradient, whereas health knowledge and cognition explain an additional 30%. However, they do not find that personality measures contribute to closing this gap. Conti and Hansman (2013) use different data and alternative measures of child personality, and argue that the contribution of personality is nearly as large as that of cognition.

Our contribution relative to HHV and CLM is showing the selection bias correction due to molecular genetic proxies of health, ability, and home environment for a set of health-related outcomes.

5 Conclusions

We find that the EA PGI exhibits strong and health-beneficial conditional associations with a variety of life outcomes in young adulthood. Moreover, these associations strongly interact with SES: individuals who grew up in disadvantaged households do not experience the health benefits of the EA PGI the way their more advantaged peers do. We also

contribute to our understanding of the potential mechanisms through which the EA PGI may affect health. These mechanisms include early health, cognitive skills, positive attitude toward education by parents and self, education, occupations, wealth, and health behaviors. Finally, we provide evidence that is consistent with a causal relationship between education and health-related outcomes.

Major disadvantages that we capture using our SES measure can be dealt with through politically feasible anti-poverty policies. The benefits and costs of various anti-poverty policies are well documented in the literature. The first contribution of this paper provides evidence regarding an additional major benefit of such policies. In particular, we suggest that anti-poverty policies complement the effect of productive genetic endowments on essential life outcomes on top of the already known effect of enhancing human capital and life outcomes on their own. We show that poverty reduction can complement the productive influence of own genetic endowments on health and health behaviors in young adulthood. As part of our study of the mechanisms, we also show a number of other positive complementing effects of SES on skills, education, earnings, wealth, and job satisfaction. Our second contribution in this paper supports education as a health policy variable in cases when education happens to be at sub-optimal levels due to market failure.

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[FOR ONLINE PUBLICATION]

Web Appendix to "Understanding the Educational Attainment Polygenic Index and its Interactions with SES in Determining Health in Young Adulthood"

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June 5, 2024

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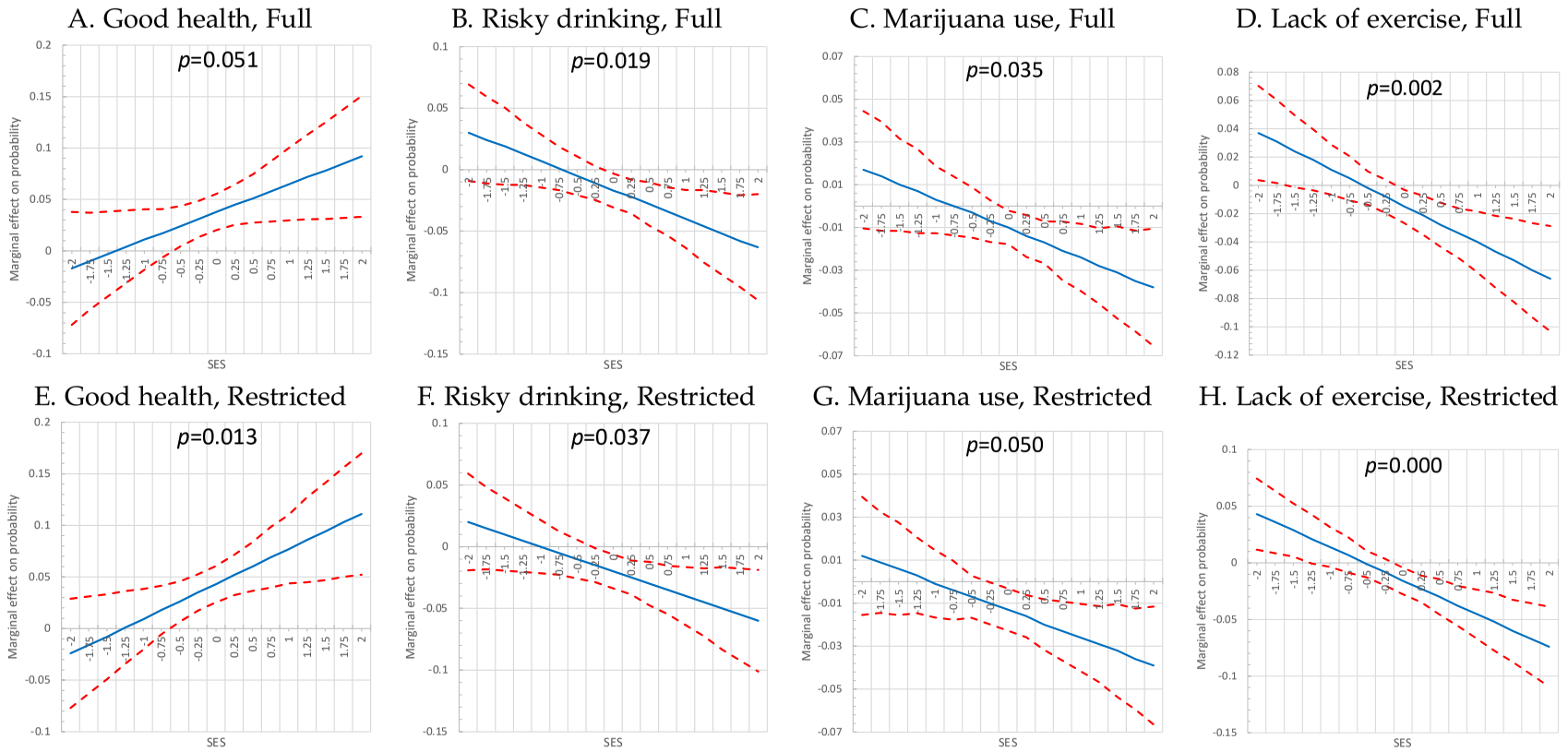
A Supplementary Figures and Tables

Table A-1: Measures of Continuous Latent Factors

Conscientiousness	Education support-self
Gathers facts when solving problems	Child's own expectation of the likelihood of going to college
Thinks of alternative ways to solve problems	Child's own willingness to go to college
Uses systematic methods when solving problems	Child's expectations of graduating from college
Analyzes outcome of solutions to problems	Education support-parental
	Child expects father's disappointment if he/she does not graduate from college
Extraversion	Child expects mother's disappointment if he/she does not graduate from college
Feels close to people at school	Child expects father's disappointment if he/she does not graduate from high school
Feels like a part of the school	Child expects mother's disappointment if he/she does not graduate from high school
Feels socially accepted	
Emotional Stability	
Has good qualities	
Has a lot to be proud of	Cognitive skills
Likes oneself	Add Health Picture Vocabulary Test
Feels like doing things right	Recent math grade
Feels socially accepted	Recent science grade
Feels loved and wanted	

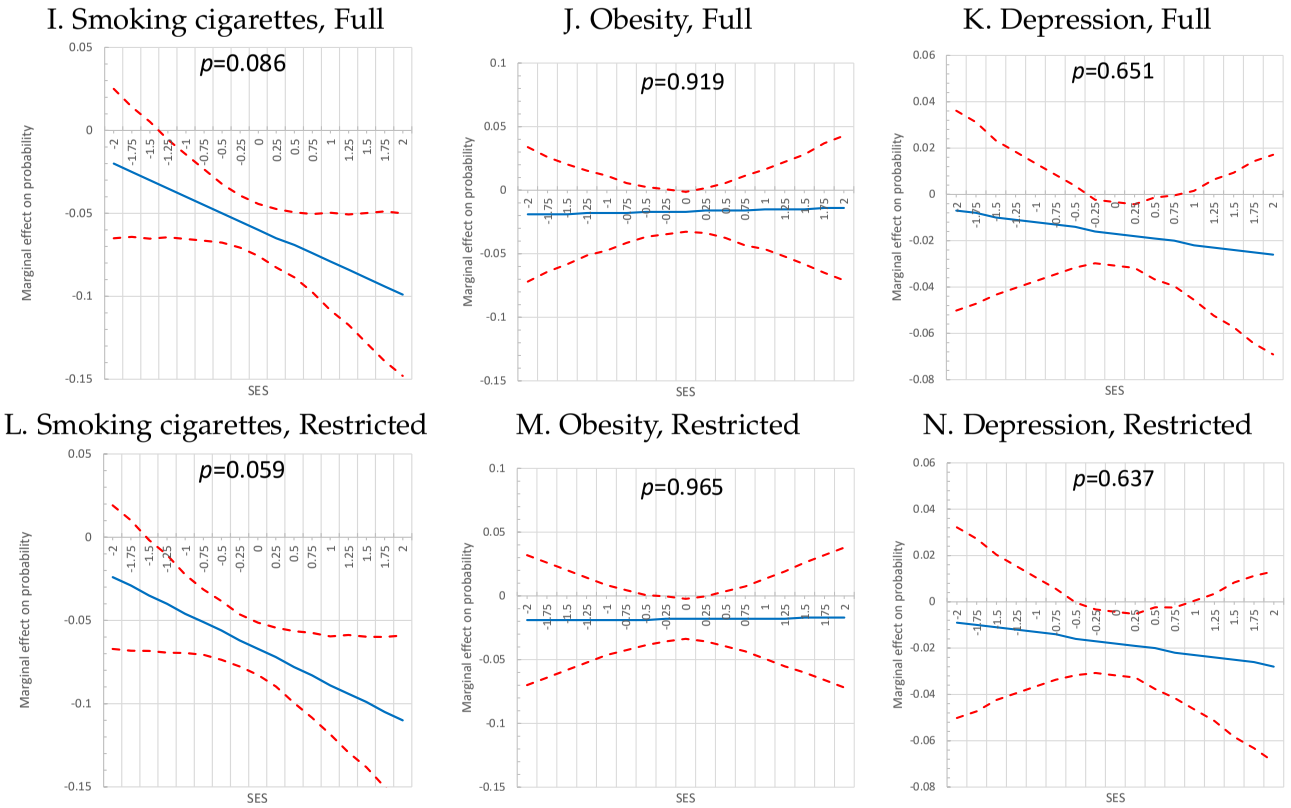
Note: All listed variables are part of the Add Health data. Sets of Add Health-specific measures of Conscientiousness, Extraversion, and Emotional Stability are based on analysis by psychologists (Young and Beaujean, 2011). Personality measures are self-reported in wave I. Indices for math and science are imputed from letter grades from wave I. Education support measures are reported by subjects in wave I.

Figure A-1: Total Marginal Effects of EA PGI on Health-Related Outcomes in Young Adulthood as a Function of Parental SES, Full vs. Restricted Set of Controls



Notes: Panels correspond to the following type of outcome models: A and E, ordered logit; logit otherwise. The effect of EA PGI is defined as $\hat{b}^*_{1k} + \hat{b}^*_{2k} \cdot SES$, where coefficients $\hat{b}^*_{1k}$ and $\hat{b}^*_{2k}$ are estimates of marginal effects from model (1); with p-values are shown for the test H_0: $b^*{}_{2k} = 0$. Effects are evaluated at average levels of covariates and plotted as a function of standardized SES factor. The full model utilized the full set of background controls. The restricted model omits background controls that are correlated with SES: meals with parents, low birth weight, cigarette smoking at home, rural, suburban, urban, the only child, number of siblings. Outcome variable for panels B and F shortly referred to as "good health' is defined as self-reported "excellent or very good health." Dashed lines represent the 95% Huber-White confidence intervals calculated using the delta method. Calculations are based on the AddHealth Data.

Figure A-1: Total Marginal Effects of EA PGI on Health-Related Outcomes in Young Adulthood as a Function of Parental SES, Full vs. Restricted Set of Controls (continued)



Notes: All panels estimated using the logit-type of outcome model. The effect of EA PGI is defined as $\hat{b}^*_{1k} + \hat{b}^*_{2k} \cdot SES$, where coefficients $\hat{b}^*_{1k}$ and $\hat{b}^*_{2k}$ are estimates of marginal effects from model (1); with p-values are shown for the test H_0: $b^*_{2k} = 0$. The full model utilized the full set of background controls. The restricted model omits background controls that are correlated with SES: meals with parents, low birth weight, cigarette smoking at home, rural, suburban, urban, the only child, number of siblings. Effects are evaluated at average levels of covariates and plotted as a function of standardized SES factor. Dashed lines represent the 95% Huber-White confidence intervals calculated using the delta method. Calculations are based on the AddHealth Data.

Table A-2: Correlations Among EA PGI and PGIs that Measure Physical Health Endowments

	EA PGI	Coronary artery disease	Myocardial infarction	Low-density lipoprotein cholesterol	Trigly-cerides	Type II diabetes	BMI	Waist-to-hip ratio	Smoking
Coronary artery disease	-0.094 *(0.000)*	1							
Myocardial infarction	-0.113 *(0.000)*	0.414 *(0.000)*	1						
Low-density lipoprotein cholesterol	-0.051 *(0.002)*	0.100 *(0.000)*	0.055 *(0.000)*	1					
Triglycerides	-0.120 *(0.000)*	0.137 *(0.000)*	0.092 *(0.000)*	0.229 *(0.000)*	1				
Type II diabetes	-0.109 *(0.000)*	0.102 *(0.000)*	0.117 *(0.000)*	0.125 *(0.000)*	0.142 *(0.000)*	1			
BMI	-0.155 *(0.000)*	-0.006 *(0.683)*	0.088 *(0.000)*	0.035 *(0.022)*	-0.037 *(0.017)*	0.182 *(0.000)*	1		
Waist-to-hip ratio	-0.141 *(0.000)*	0.104 *(0.000)*	0.113 *(0.000)*	0.068 *(0.000)*	0.313 *(0.000)*	0.096 *(0.000)*	-0.138 *(0.000)*	1	
Height	0.140 *(0.000)*	-0.116 *(0.000)*	-0.065 *(0.000)*	-0.303 *(0.000)*	-0.150 *(0.000)*	-0.327 *(0.000)*	-0.117 *(0.000)*	0.006 *(0.721)*	1
Smoking	-0.119 *(0.000)*	0.023 *(0.130)*	0.063 *(0.000)*	-0.014 *(0.370)*	-0.040 *(0.009)*	0.009 *(0.554)*	0.122 *(0.000)*	-0.036 *(0.019)*	-0.091 *(0.000)*

Note: Calculations based on the Add Health data for the main model estimation sample. Pairwise correlations reported with corresponding p-values shown in parentheses.

Table A-3: Correlations Among EA PGI and PGIs that Measure Mental Health Endowments

	EA PGI	Depression	Neuroticism	Attention-deficit disorder	Bipolar disorder	Major depressive disorder	Schizophrenia
Depression	0.106	1					
	(0.000)						
Neuroticism	0.154	0.459	1				
	(0.000)	*(0.000)*					
Attention-deficit disorder	-0.221	-0.144	-0.063	1			
	(0.000)	*(0.000)*	*(0.000)*				
Bipolar disorder	-0.004	-0.104	-0.068	-0.054	1		
	(0.823)	*(0.000)*	*(0.000)*	*(0.000)*			
Major depressive disorder	-0.144	-0.323	-0.234	0.184	0.169	1	
	(0.000)	*(0.000)*	*(0.000)*	*(0.000)*	*(0.000)*		
Schizophrenia	-0.031	-0.112	-0.074	0.028	0.275	0.240	1
	(0.061)	*(0.000)*	*(0.000)*	*(0.069)*	*(0.000)*	*(0.000)*	
Mental health cross disorder	-0.013	-0.155	-0.145	-0.065	0.623	0.311	0.414
	(0.414)	*(0.000)*	*(0.000)*	*(0.000)*	*(0.000)*	*(0.000)*	*(0.000)*

Note: Calculations based on the Add Health data for the main model estimation sample. Pairwise correlations reported with corresponding p-values shown in parentheses.

Table A-4: Factor Loadings of the Measurement System

	Factor loading	Standard error	p-value
Measures of SES			
Living in an unsafe neighbourhood	0.556	0.116	0.000
Having difficulties with paying bills	0.889	0.099	0.000
Household on government assistance	1.643	0.167	0.000
At least one parent has a college education	-1.001	0.098	0.000
Household income from the lowest quintile	1.892	0.216	0.000
Wald test of equality of factor loadings (with parental college loading reversed)			
Test statistic	47.74		
Degrees of freedom	4		
p-value	0.0000		

Note: Calculations are based on the Add Health data. See notes to Figure 1 of the main paper for variable definitions.

Table A-5: Reduced-Form Associations Between EA PGI, Gene-SES Interaction and Health-Related Outcomes, Not Conditional on Education ("Total Effect")

	Excellent or very good health	Risky drinking	Marijuana use	Lack of exercise	Smoking cigarettes	Obese	Depression
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
EA PGI	0.038 ***	-0.016 **	-0.010 **	-0.014 **	-0.060 ***	-0.016 **	-0.016 **
	(0.009)	*(0.007)*	*(0.004)*	*(0.006)*	*(0.008)*	*(0.008)*	*(0.007)*
Effect size[(a)]	0.061	-0.077	-0.104	-0.108	-0.230	-0.047	-0.084
EA PGI	0.027 *	-0.023 **	-0.014 **	-0.026 ***	-0.020 *	0.001	-0.005
× SES	*(0.014)*	*(0.010)*	*(0.007)*	*(0.008)*	*(0.011)*	*(0.013)*	*(0.011)*
Effect size[(a)]	0.043	-0.110	-0.146	-0.200	-0.077	0.003	-0.026
SES	0.086 ***	-0.020 **	0.002	-0.022 ***	-0.049 ***	-0.068 ***	0.001
	(0.014)	0.010	0.007	0.008	0.011	0.013	0.010
$(\text{EA PGS})^2$	0.003	-0.002	-0.001	-0.007 *	0.000	-0.008	-0.014 ***
	(0.006)	0.005	0.003	0.004	0.005	0.006	0.005
Sample Size	3709	3694	3705	3708	3699	3664	3709

Notes: The results are based on model (1) and conditional on the full set of observable controls presented in Table 2. Panel (1) shows a regression coefficient for the ordered logit model with five health categories. Panels (2–7) report estimated marginal associations based on logit models. Asterisks indicate statistical significance level: ***, 1 % level; **, 5 % level; *, 10 % level. Calculations are based on the Add Health data. Wald test statistics are shown, with p-values reported in square brackets. [(a)]Effect size is the ratio of the estimated marginal effect to the average level of the outcome.

Table A-6: Reduced-Form Associations Between EA PGI, Gene-SES Interaction and Health-Related Outcomes, Conditional on Education ("Direct Effect")

	Excellent or very good health	Risky drinking	Marijuana use	Lack of exercise	Smoking cigarettes	Obese	Depression
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
EA PGI	0.023 **	-0.009	-0.004	-0.006	-0.036 ***	-0.007	-0.009
	(0.009)	*(0.007)*	*(0.004)*	*(0.006)*	*(0.007)*	*(0.009)*	*(0.007)*
Effect size[(a)]	0.037	-0.043	-0.042	-0.046	-0.138	-0.020	-0.047
EA PGI	0.017	-0.019 *	-0.012 **	-0.022 ***	-0.008	0.009	0.000
× SES	*(0.014)*	*(0.010)*	*(0.006)*	*(0.008)*	*(0.011)*	*(0.014)*	*(0.011)*
Effect size[(a)]	0.027	-0.091	-0.125	-0.169	-0.031	0.026	0.000
SES	0.082 ***	-0.016	0.007	-0.005	-0.041	-0.071 **	0.014
	(0.030)	0.024	0.018	0.021	0.031	0.028	0.023
$(\text{EA PGI})^2$	0.002	-0.002	0.000	-0.006	0.001	-0.008	-0.014 ***
	(0.006)	0.005	0.003	0.004	0.005	0.006	0.005
Sample Size	3709	3694	3705	3708	3699	3664	3709

Notes: The results are based on model (2) and conditional on education and full set of observable controls presented in Table 2. Panel (1) shows a regression coefficient for the ordered logit model with five health categories. Panels (2–7) report estimated marginal associations based on logit models. Asterisks indicate statistical significance level: ***, 1 % level; **, 5 % level; *, 10 % level. Calculations are based on the Add Health data. Wald test statistics are shown, with p-values reported in square brackets. [(a)]Effect size is the ratio of the estimated marginal effect to the average level of the outcome.

Table A-7: Robustness of Our Main Model to Various Combinations of SES Measures: Marginal Effects of EA PGI on the Probability of Having Good Health in Young Adulthood, Logit Model Results

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
A. Standardized latent SES factor						
EA PGI	0.038 ***	0.040 ***	0.039 ***	0.043 ***	0.037 ***	0.036 ***
as SES=0	*(0.009)*	*(0.009)*	*(0.009)*	*(0.009)*	*(0.009)*	*(0.009)*
EA PGI	0.027 *	0.038 **	0.015	0.033	0.013	0.024 *
× SES Factor	*(0.014)*	*(0.016)*	*(0.014)*	*(0.023)*	*(0.013)*	*(0.013)*
B. Standardized equally-weighted index of SES measures						
EA PGI	0.036 ***	0.037 ***	0.035 ***	0.037 ***	0.036 ***	0.037 ***
as SES=0	*(0.009)*	*(0.009)*	*(0.009)*	*(0.009)*	*(0.009)*	*(0.009)*
EA PGI	0.025 ***	0.029 ***	0.015 *	0.020 **	0.017 **	0.027 ***
× SES Index	*(0.009)*	*(0.009)*	*(0.008)*	*(0.008)*	*(0.008)*	*(0.009)*
C. Binary SES: No single disadvantage from the list (SES=1) vs. At least one (SES=0)						
EA PGI	0.058 ***	0.057 ***	0.045 ***	0.047 ***	0.047 ***	0.061 ***
as SES=0	*(0.014)*	*(0.012)*	*(0.012)*	*(0.010)*	*(0.010)*	*(0.013)*
EA PGI	0.032 *	0.036 **	0.017	0.034 *	0.030 *	0.040 **
× Binary SES	*(0.017)*	*(0.016)*	*(0.017)*	*(0.018)*	*(0.017)*	*(0.016)*
D. Factor model goodness of fit						
RMSEA	0.009	0.000	0.019	0.022	0.022	0.020
Prob(RMSEA $\leq$ .05)	1.000	1.000	0.989	1.000	0.981	1.000
CFI	0.999	1	0.998	0.854	0.997	0.996
TLI	0.971	1	0.857	0.445	0.800	0.861
E. Sets of parental SES measures that define differences between models 1–6						
Self-reported issues[(a)]	✓	✓	✓	✓	✓	✓
Parental college[(b)]	✓	✓				✓
Lowest income quintile[(c)]	✓		✓			
Income below median(c)					✓	✓

Notes: Model 1 represents the main specification of the reduced-form model (1,4). Models 2–6 are similar models but with different measures of SES used in the measurement system (4). Panels B and C offer alternative methods of measure aggregation. Asterisks indicate statistical significance level: ***, 1 % level; **, 5 % level; *, 10 % level. Calculations are based on the Add Health data. [(a)]Living in an unsafe neighbourhood; having difficulties with paying bills; and household receiving government assistance; [(b)]At least one parent graduated from college; [(c)]Household income in subject's childhood.

Table A-8: Robustness of Our Main Model to Various Combinations of SES Measures: Marginal Effects of EA PGI on the Probability of Risky Drinking in Young Adulthood, Logit Model Results

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
A. Standardized latent SES factor						
EA PGI	-0.017 **	-0.016 **	-0.017 **	-0.017 **	-0.016 **	-0.016 **
as SES=0	*(0.007)*	*(0.007)*	*(0.007)*	*(0.007)*	*(0.007)*	*(0.007)*
EA PGI	-0.023 **	-0.016	-0.026 ***	-0.025 *	-0.014	-0.015 *
× SES Factor	*(0.010)*	*(0.011)*	*(0.010)*	*(0.013)*	*(0.009)*	*(0.009)*
B. Standardized equally-weighted index of SES measures						
EA PGI	-0.016 **	-0.016 **	-0.016 **	-0.016 **	-0.016 **	-0.016 **
as SES=0	*(0.007)*	*(0.007)*	*(0.007)*	*(0.007)*	*(0.007)*	*(0.007)*
EA PGI	-0.013 *	-0.012 *	-0.010	-0.009	-0.012 *	-0.014 **
× SES Index	*(0.007)*	*(0.007)*	*(0.007)*	*(0.007)*	*(0.007)*	*(0.007)*
C. Binary SES: No single disadvantage from the list (SES=1) vs. At least one (SES=0)						
EA PGI	-0.023 **	-0.018 **	-0.027 ***	-0.022 ***	-0.023 ***	-0.019 *
as SES=0	*(0.011)*	*(0.009)*	*(0.010)*	*(0.008)*	*(0.008)*	*(0.010)*
EA PGI	-0.012	-0.006	-0.022 *	-0.025 *	-0.024 *	-0.008
× Binary SES	*(0.013)*	*(0.012)*	*(0.013)*	*(0.014)*	*(0.013)*	*(0.012)*
D. Factor model goodness of fit						
RMSEA	0.009	0.000	0.019	0.022	0.022	0.020
Prob(RMSEA $\leq$ .05)	1.000	1.000	0.989	1.000	0.981	1.000
CFI	0.999	1	0.998	0.854	0.997	0.996
TLI	0.971	1	0.857	0.445	0.800	0.861
E. Sets of parental SES measures that define differences between models 1–6						
Self-reported issues[(a)]	✓	✓	✓	✓	✓	✓
Parental college[(b)]	✓	✓				✓
Lowest income quintile[(c)]	✓		✓			
Income below median(c)					✓	✓

Notes: Model 1 represents the main specification of the reduced-form model (1,4). Models 2–6 are similar models but with different measures of SES used in the measurement system (4). Panels B and C offer alternative methods of measure aggregation. Asterisks indicate statistical significance level: ***, 1 % level; **, 5 % level; *, 10 % level. Calculations are based on the Add Health data. [(a)]Living in an unsafe neighbourhood; having difficulties with paying bills; and household receiving government assistance; [(b)]At least one parent graduated from college; [(c)]Household income in subject's childhood.

Table A-9: Robustness of Our Main Model to Various Combinations of SES Measures: Marginal Effects of EA PGI on the Probability of Consuming Marijuana Regularly in Young Adulthood, Logit Model Results

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
A. Standardized latent SES factor						
EA PGI	-0.010 **	-0.010 **	-0.010 **	-0.011 **	-0.010 **	-0.010 **
as SES=0	*(0.004)*	*(0.004)*	*(0.004)*	*(0.005)*	*(0.004)*	*(0.004)*
EA PGI	-0.014 **	-0.014 **	-0.013 *	-0.021 **	-0.014 **	-0.015 **
× SES Factor	*(0.007)*	*(0.007)*	*(0.007)*	*(0.010)*	*(0.006)*	*(0.006)*
B. Standardized equally-weighted index of SES measures						
EA PGI	-0.012 **	-0.011 **	-0.012 **	-0.011 **	-0.011 **	-0.011 **
as SES=0	*(0.005)*	*(0.005)*	*(0.005)*	*(0.005)*	*(0.005)*	*(0.005)*
EA PGI	-0.009 *	-0.007	-0.011 **	-0.009 *	-0.007	-0.006
× SES Index	*(0.005)*	*(0.005)*	*(0.005)*	*(0.005)*	*(0.005)*	*(0.005)*
C. Binary SES: No single disadvantage from the list (SES=1) vs. At least one (SES=0)						
EA PGI	-0.019 **	-0.017 ***	-0.020 ***	-0.015 ***	-0.016 ***	-0.017 **
as SES=0	*(0.008)*	*(0.006)*	*(0.006)*	*(0.005)*	*(0.006)*	*(0.007)*
EA PGI	-0.014	-0.013	-0.020 **	-0.015 *	-0.015 *	-0.011
× Binary SES	*(0.009)*	*(0.009)*	*(0.009)*	*(0.009)*	*(0.009)*	*(0.009)*
D. Factor model goodness of fit						
RMSEA	0.009	0.000	0.019	0.022	0.022	0.020
Prob(RMSEA $\leq$ .05)	1.000	1.000	0.989	1.000	0.981	1.000
CFI	0.999	1	0.998	0.854	0.997	0.996
TLI	0.971	1	0.857	0.445	0.800	0.861
E. Sets of parental SES measures that define differences between models 1–6						
Self-reported issues[(a)]	✓	✓	✓	✓	✓	✓
Parental college[(b)]	✓	✓				✓
Lowest income quintile[(c)]	✓		✓			
Income below median(c)					✓	✓

Notes: Model 1 represents the main specification of the reduced-form model (1,4). Models 2–6 are similar models but with different measures of SES used in the measurement system (4). Panels B and C offer alternative methods of measure aggregation. Asterisks indicate statistical significance level: ***, 1 % level; **, 5 % level; *, 10 % level. Calculations are based on the Add Health data. [(a)]Living in an unsafe neighbourhood; having difficulties with paying bills; and household receiving government assistance; [(b)]At least one parent graduated from college; [(c)]Household income in subject's childhood.

Table A-10: Robustness of Our Main Model to Various Combinations of SES Measures: Marginal Effects of EA PGI on the Probability of Smoking in Young Adulthood, Logit Model Results

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
A. Standardized latent SES factor						
EA PGI	-0.060 ***	-0.060 ***	-0.061 ***	-0.061 ***	-0.060 ***	-0.059 ***
as SES=0	*(0.008)*	*(0.008)*	*(0.008)*	*(0.008)*	*(0.008)*	*(0.008)*
EA PGI	-0.020 *	-0.020	-0.014	-0.003	-0.015	-0.019 *
× SES Factor	*(0.011)*	*(0.013)*	*(0.012)*	*(0.016)*	*(0.011)*	*(0.011)*
B. Standardized equally-weighted index of SES measures						
EA PGI	-0.061 ***	-0.061 ***	-0.061 ***	-0.061 ***	-0.061 ***	-0.061 ***
as SES=0	*(0.008)*	*(0.008)*	*(0.008)*	*(0.008)*	*(0.008)*	*(0.008)*
EA PGI	-0.015 *	-0.014 *	-0.005	-0.002	-0.005	-0.015 *
× SES Index	*(0.008)*	*(0.008)*	*(0.007)*	*(0.007)*	*(0.007)*	*(0.008)*
C. Binary SES: No single disadvantage from the list (SES=1) vs. At least one (SES=0)						
EA PGI	-0.057 ***	-0.059 ***	-0.059 ***	-0.062 ***	-0.060 ***	-0.056 ***
as SES=0	*(0.013)*	*(0.011)*	*(0.011)*	*(0.009)*	*(0.009)*	*(0.011)*
EA PGI	-0.004	-0.006	-0.001	-0.005	-0.002	-0.002
× Binary SES	*(0.015)*	*(0.014)*	*(0.015)*	*(0.016)*	*(0.016)*	*(0.014)*
D. Factor model goodness of fit						
RMSEA	0.009	0.000	0.019	0.022	0.022	0.020
Prob(RMSEA $\leq$ .05)	1.000	1.000	0.989	1.000	0.981	1.000
CFI	0.999	1	0.998	0.854	0.997	0.996
TLI	0.971	1	0.857	0.445	0.800	0.861
E. Sets of parental SES measures that define differences between models 1–6						
Self-reported issues[(a)]	✓	✓	✓	✓	✓	✓
Parental college[(b)]	✓	✓				✓
Lowest income quintile[(c)]	✓		✓			
Income below median(c)					✓	✓

Notes: Model 1 represents the main specification of the reduced-form model (1,4). Models 2–6 are similar models but with different measures of SES used in the measurement system (4). Panels B and C offer alternative methods of measure aggregation. Asterisks indicate statistical significance level: ***, 1 % level; **, 5 % level; *, 10 % level. Calculations are based on the Add Health data. [(a)]Living in an unsafe neighbourhood; having difficulties with paying bills; and household receiving government assistance; [(b)]At least one parent graduated from college; [(c)]Household income in subject's childhood.

Table A-11: Robustness of Our Main Model to Various Combinations of SES Measures: Marginal Effects of EA PGI on the Probability of Obesity in Young Adulthood, Logit Model Results

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
A. Standardized latent SES factor						
EA PGI	-0.017 **	-0.017 **	-0.019 **	-0.021 ***	-0.018 **	-0.016 **
as SES=0	*(0.008)*	*(0.008)*	*(0.008)*	*(0.008)*	*(0.008)*	*(0.008)*
EA PGI	0.001	0.001	0.004	-0.001	0.002	0.002
× SES Factor	*(0.013)*	*(0.015)*	*(0.013)*	*(0.020)*	*(0.012)*	*(0.012)*
B. Standardized equally-weighted index of SES measures						
EA PGI	-0.018 **	-0.018 **	-0.019 **	-0.020 **	-0.019 **	-0.018 **
as SES=0	*(0.008)*	*(0.008)*	*(0.008)*	*(0.008)*	*(0.008)*	*(0.008)*
EA PGI	-0.009	-0.009	-0.004	-0.004	-0.004	-0.009
× SES Index	*(0.008)*	*(0.008)*	*(0.008)*	*(0.008)*	*(0.008)*	*(0.008)*
C. Binary SES: No single disadvantage from the list (SES=1) vs. At least one (SES=0)						
EA PGI	-0.007	-0.011	-0.013	-0.020 **	-0.017 *	-0.011
as SES=0	*(0.014)*	*(0.012)*	*(0.012)*	*(0.010)*	*(0.010)*	*(0.012)*
EA PGI	0.014	0.014	0.010	0.003	0.007	0.011
× Binary SES	*(0.017)*	*(0.016)*	*(0.016)*	*(0.018)*	*(0.017)*	*(0.016)*
D. Factor model goodness of fit						
RMSEA	0.009	0.000	0.019	0.022	0.022	0.020
Prob(RMSEA $\leq$ .05)	1.000	1.000	0.989	1.000	0.981	1.000
CFI	0.999	1	0.998	0.854	0.997	0.996
TLI	0.971	1	0.857	0.445	0.800	0.861
E. Sets of parental SES measures that define differences between models 1–6						
Self-reported issues[(a)]	✓	✓	✓	✓	✓	✓
Parental college[(b)]	✓	✓				✓
Lowest income quintile[(c)]	✓		✓			
Income below median(c)					✓	✓

Notes: Model 1 represents the main specification of the reduced-form model (1,4). Models 2–6 are similar models but with different measures of SES used in the measurement system (4). Panels B and C offer alternative methods of measure aggregation. Asterisks indicate statistical significance level: ***, 1 % level; **, 5 % level; *, 10 % level. Calculations are based on the Add Health data. [(a)]Living in an unsafe neighbourhood; having difficulties with paying bills; and household receiving government assistance; [(b)]At least one parent graduated from college; [(c)]Household income in subject's childhood.

Table A-12: Robustness of Our Main Model to Various Combinations of SES Measures: Marginal Effects of EA PGI on the Probability of Depression in Young Adulthood, Logit Model Results

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
A. Standardized latent SES factor						
EA PGI	-0.017 **	-0.017 **	-0.017 **	-0.018 **	-0.017 **	-0.017 **
as SES=0	*(0.007)*	*(0.007)*	*(0.007)*	*(0.007)*	*(0.007)*	*(0.007)*
EA PGI	-0.005	-0.016	0.002	-0.035 *	-0.015	-0.015
× SES Factor	*(0.011)*	*(0.012)*	*(0.011)*	*(0.021)*	*(0.010)*	*(0.010)*
B. Standardized equally-weighted index of SES measures						
EA PGI	-0.017 **	-0.017 **	-0.017 **	-0.018 **	-0.018 **	-0.017 **
as SES=0	*(0.007)*	*(0.007)*	*(0.007)*	*(0.007)*	*(0.007)*	*(0.007)*
EA PGI	-0.010	-0.012 *	-0.014 **	-0.017 **	-0.013 *	-0.010
× SES Index	*(0.007)*	*(0.007)*	*(0.007)*	*(0.007)*	*(0.007)*	*(0.007)*
C. Binary SES: No single disadvantage from the list (SES=1) vs. At least one (SES=0)						
EA PGI	-0.021 *	-0.021 **	-0.021 **	-0.024 ***	-0.021 ***	-0.020 *
as SES=0	*(0.012)*	*(0.010)*	*(0.010)*	*(0.008)*	*(0.008)*	*(0.011)*
EA PGI	-0.006	-0.008	-0.011	-0.027 *	-0.010	-0.005
× Binary SES	*(0.015)*	*(0.013)*	*(0.013)*	*(0.014)*	*(0.014)*	*(0.014)*
D. Factor model goodness of fit						
RMSEA	0.009	0.000	0.019	0.022	0.022	0.020
Prob(RMSEA $\leq$ .05)	1.000	1.000	0.989	1.000	0.981	1.000
CFI	0.999	1	0.998	0.854	0.997	0.996
TLI	0.971	1	0.857	0.445	0.800	0.861
E. Sets of parental SES measures that define differences between models 1–6						
Self-reported issues[(a)]	✓	✓	✓	✓	✓	✓
Parental college[(b)]	✓	✓				✓
Lowest income quintile[(c)]	✓		✓			
Income below median(c)					✓	✓

Notes: Model 1 represents the main specification of the reduced-form model (1,4). Models 2–6 are similar models but with different measures of SES used in the measurement system (4). Panels B and C offer alternative methods of measure aggregation. Asterisks indicate statistical significance level: ***, 1 % level; **, 5 % level; *, 10 % level. Calculations are based on the Add Health data. [(a)]Living in an unsafe neighbourhood; having difficulties with paying bills; and household receiving government assistance; [(b)]At least one parent graduated from college; [(c)]Household income in subject's childhood.

Table A-13: An Alternative Sibling Fixed Effect Estimation of the Effect of EA PGI and EA PGI-SES Interaction on Health-Related Outcomes

	General health	Risky Drinking	Marijuana use	No exercise	Smoking tobacco	Obese	Depres-sion
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
EA PGS	-0.055	0.002	-0.003	-0.019	-0.050	-0.023	0.021
	0.128	0.053	0.043	0.061	0.047	0.060	0.053
EA PGS × SES	0.078	0.021	0.041	-0.030	0.028	-0.067	-0.018
	0.092	0.044	0.034	0.050	0.045	0.042	0.038
Number of families	200	200	200	200	200	200	200

Notes: A lack of asterisks in the table indicates no statistically significant effects at the 10% level. We exclude identical twins, as they share the same genetic background and the same family SES. All regressions are conditional on the following regressors that may differ across children from the same family: 1^{st}, 2^{d}, and 3^{d}-born, meals with parents, low birth weight, genetic ancestry principal components, age, and sex. Calculations are based on the Add Health data.

Table A-14: Conditional Reduced-Form Associations Between EA PGI, Gene-SES Interaction, and Potential Mechanisms Behind the Effects of EA PGI

	Cognitive skills	Conscienti-ousness	Emotional stability	Extra-version	Early general health	Very good or excellent health	Education support-parents	Education support-self
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
EA PGI	0.166 ***	0.002	0.010	-0.012	0.091 ***	0.020 **	0.042 **	0.099 ***
	(0.017)	*(0.017)*	*(0.017)*	*(0.017)*	*(0.032)*	*(0.008)*	*(0.017)*	*(0.016)*
EA PGI	0.043 *	0.041	0.014	0.003	-0.030	-0.003	-0.015	-0.048 **
× SES	*(0.023)*	*(0.026)*	*(0.026)*	*(0.026)*	*(0.046)*	*(0.012)*	*(0.025)*	*(0.022)*
SES	0.206 ***	0.043 *	-0.084 ***	-0.081 ***	0.172 ***	0.061 ***	0.217 ***	0.338 ***
	0.026	0.026	0.026	0.026	*(0.046)*	*(0.012)*	0.026	0.025
$(\text{EA PGI})^2$	-0.009	-0.009	0.009	0.002	0.036	0.007	-0.008	-0.006
	0.012	0.012	0.012	0.012	*(0.022)*	*(0.006)*	0.012	0.011
Sample Size	3694	3699	3664	3705	3709	3709	3708	3709

Notes: The results are based on the reduced-form model (1) and conditional on the full set of observable controls presented in Table 2. Panel (1) shows marginal conditional association based on a logit model. Panels (1-7) show marginal conditional associations based on OLS, with standardized factor scores used as outcomes. Asterisks indicate statistical significance level: ***, 1 % level; **, 5 % level; *, 10 % level. Calculations are based on the Add Health data. [(a)]Testing whether all seven coefficients in the corresponding row are jointly zero from models (1-7) estimated simultaneously. Wald test statistics are shown, with p-values reported in square brackets. [(b)]The SES score is a standardized factor score that represents the degree of parental socioeconomic advantage.

Table A-15: Conditional Reduced Form Associations Between EA PGI, Gene-SES Interaction, and Early Health Behaviors

	Drinking Alcohol			Smoking cigarettes			Overweight	
	(A) at least once over the year	(B) at least 2 times per month	(C) at least 3 times per week	(A) at least once over the year	(B) at least 2 times per month	(C) at least 3 times per week	(A) slightly or very overweight	(B) very overweight
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
EA PGI	-0.023 **	-0.002	0.001	-0.035 ***	-0.026 ***	-0.020 ***	-0.013	0.001
	(0.009)	*(0.007)*	*(0.002)*	*(0.009)*	*(0.007)*	*(0.005)*	*(0.008)*	*(0.003)*
EA PGI × SES	0.021	0.002	-0.002	-0.002	0.005	-0.009	0.001	0.000
	(0.014)	*(0.010)*	*(0.003)*	*(0.014)*	*(0.011)*	*(0.008)*	*(0.013)*	*(0.004)*
Sample Size	3604	3604	3604	3611	3611	3611	3707	3707

Notes: The results are based on the reduced-form model (1) and conditional on the full set of observable controls presented in Table 2. Panel (1) shows a regression coefficient for the ordered logit model with five health categories. Panels (2–7) report estimated marginal effects based on logit models. The SES score is a factor score that represents the degree of socioeconomic problems faced by the household: the higher the score, the lower the SES. Asterisks indicate statistical significance level: ***, 1 % level; **, 5 % level; *, 10 % level. Calculations are based on the Add Health data. Sample size is 3709.

Table A-16: Conditional Reduced-Form Associations Between EA PGI, Gene-SES Interaction, and Health-Related Outcomes With and Without Controlling for Early Behaviors, Logit Regression Results

	Risky drinking		Smoking cigarettes		Obese	
	(1)	(2)	(3)	(4)	(5)	(6)
EA PGI	-0.016 **	-0.015 **	-0.060 ***	-0.050 ***	-0.016 **	-0.012
	(0.007)	*(0.007)*	*(0.008)*	*(0.008)*	*(0.008)*	*(0.009)*
EA PGI	-0.023 **	-0.027 ***	-0.020 *	-0.018	0.001	0.000
× SES	*(0.010)*	*(0.010)*	*(0.011)*	*(0.011)*	*(0.013)*	*(0.014)*
Corresponding	-	0.080 ***	-	0.150 ***	-	0.397 ***
early behavior (A)	-	*(0.015)*	-	*(0.019)*	-	*(0.018)*
Corresponding	-	0.028 *	-	0.039	-	0.305 ***
early behavior (B)	-	*(0.017)*	-	*(0.029)*	-	*(0.055)*
Corresponding	-	0.034	-	0.162 ***	-	-
early behavior (C)	-	*(0.034)*	-	*(0.029)*	-	-
Sample Size	3709	3604	3699	3603	3664	3662

Notes: Corresponding behaviors A, B, and C are used to make the table compact. See Table A-15 for definitions of corresponding behaviors A, B, and C. The corresponding behavior is taken from the same narrow behavioral type: for risky drinking, the corresponding behavior is drinking alcohol in early life (all variables A, B, and C describe drinking). It is early smoking measures for adult smoking; it is early life overweight measures for adult obesity. The results are based on the reduced-form model (1) and conditional on the full set of observable controls presented in Table 2. Panel (1) shows a regression coefficient for the ordered logit model with five health categories. Panels (2–7) report estimated marginal effects based on logit models. The SES score is a factor score that represents the degree of socioeconomic problems faced by the household: the higher the score, the lower the SES. Asterisks indicate statistical significance level: ***, 1 % level; **, 5 % level; *, 10 % level. Calculations are based on the Add Health data. Sample size is 3709.

Table A-17: Marginal Conditional Associations Between EA PGI and Education Categories, Ordered Logit Estimates

	Below high school	High school diploma	College below Bachelor's	Bachelor's or above	Underlying ordered logit coefficient
	(1)	(2)	(3)	(4)	(5)
EA PGI	-0.010 ***	-0.087 ***	0.011 ***	0.086 ***	0.391 ***
	(0.001)	*(0.008)*	*(0.002)*	*(0.008)*	*(0.036)*
Effect size[(a)]	-0.208	-0.210	0.063	0.237	-
EA PGI	-0.003 ***	-0.028 **	0.003 ***	0.027 **	0.124 **
$\times$ SES[(b)]	*(0.001)*	*(0.012)*	*(0.001)*	*(0.012)*	*(0.052)*
SES	-0.021 ***	-0.188 ***	0.023 ***	0.186 ***	0.845 ***
	(0.002)	*(0.015)*	*(0.004)*	*(0.014)*	*(0.063)*
$(\text{EA PGI})^2$	0.000	-0.003	0.000	0.003	0.013
	(0.001)	*(0.006)*	*(0.001)*	*(0.006)*	*(0.026)*

Coefficients in panels (1–4) are marginal conditional associations between a right-hand-side variable, such as EA PGI, and the probability of the corresponding educational level. Estimates (1–4) are based on the ordered logit model and sum up to zero across columns 1–4 by construction (up to a rounding error). Column (5) reports coefficients of the underlying logit model based on which marginal associations (1–4) are calculated. Asterisks indicate statistical significance level: ***, 1 % level; **, 5 % level; *, 10 % level. Calculations are based on the Add Health data. Sample size is 3709. [(a)]Ratio of the estimated effect to the sample average of the outcome. [(b)]The SES score is a standardized factor score that represents the degree of parental socioeconomic advantage.

Table A-18: Conditional Reduced-Form Associations Between EA PGI, Gene-SES Interaction, and Outcomes Related to Employment and Wealth

	Household income[a] (1)	Household assets[b] (2)	Job satisfaction[b] (3)	Job physicality[c] (4)
EA PGI	0.130 ***	0.085 ***	0.153 ***	-0.101 ***
	(0.031)	*(0.031)*	*(0.036)*	*(0.035)*
EA PGI	0.129 ***	0.043	0.102 **	-0.124 **
× SES	*(0.043)*	*(0.043)*	*(0.051)*	*(0.051)*
SES	0.432 ***	0.247 ***	0.231 ***	-0.244 ***
	(0.046)	0.045	0.050	0.051
$(\text{EA PGI})^2$	-0.041 *	-0.030	-0.005	-0.025
	(0.022)	0.022	0.025	0.025
Sample Size	3709	3709	3709	3709

Notes: The results are based on the reduced-form model (1) and conditional on the full set of observable controls presented in Table 2. Panels (1-4) show estimated ordered logit model coefficients. Asterisks indicate statistical significance level: ***, 1 % level; **, 5 % level; *, 10 % level. Calculations are based on the Add Health data. [a]The original data household income bands ranged from 1 (the lowest) to 12 (the highest). For the presented model, two low-probability categories, 1 and 12, are merged with categories 2 and 11 correspondingly to archive the numerical stability of the ordered logit model estimation procedure. [b]Bands 1 (the lowest)–9 (the highest). [c]Self-rating 1 (the least)–4 (the most).

Table A-19: Conditional Marginal Association Between Education and Risky Drinking Depending on the Set of Controls

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Education							
Below High School	0.095 *** *(0.036)*	0.069 ** *(0.028)*	0.108 *** *(0.034)*	0.085 *** *(0.027)*	0.109 *** *(0.023)*	0.117 *** *(0.028)*	0.128 *** *(0.022)*
High School Diploma	0.083 *** *(0.018)*	0.091 *** *(0.013)*	0.096 *** *(0.017)*	0.106 *** *(0.013)*	0.122 *** *(0.012)*	0.112 *** *(0.016)*	0.132 *** *(0.012)*
College below Bachelor's	0.070 *** *(0.021)*	0.075 *** *(0.016)*	0.082 *** *(0.021)*	0.088 *** *(0.016)*	0.087 *** *(0.015)*	0.095 *** *(0.020)*	0.095 *** *(0.015)*
Average change relative to...							
Column 7	*30%*	*33%*	*19%*	*20%*	*10%*	*8%*	*0%*
Column 4	*10%*	*16%*	*-4%*	*0%*	-	-	-
Column 2	*-7%*	*0%*	-	-	-	-	-
Controls							
Traditional[(a)]	✓	✓	✓	✓			
Skills[(b)]	✓	✓			✓		
Genotyping[(c)]	✓		✓			✓	

Notes: Marginal associations between education levels and probabilities of corresponding health-related outcomes are reported based on the logit factor model (3,4). The omitted education category is "Bachelor's degree or above." Effects of education are calculated at the average level of SES and other controls. Asterisks indicate statistical significance level: ***, 1 % level; **, 5 % level; *, 10 % level. Add Health data are used. Sample size is 3709. Percentages are average percentage changes of the three education coefficients reported. Column 1 corresponds to the unrestricted model (3). All other columns are restricted versions of the same model, with certain sets of controls omitted. Column 7 corresponds to a regression of outcomes on education dummies only. Other sets of controls are defined as follows: [(a)]Background controls that are documented in Table 2 including SES factor, but excluding genetic ancestry PCs. [(b)]Traditionally-measured cognitive and noncognitive skills, $\boldsymbol{S}$; [(c)]Data based on genotyping: 17 PGIs and 10 genetic ancestry PCs.

Table A-20: Conditional Marginal Association Between Education and Marijuana Consumption Depending on the Set of Controls

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Education							
Below High School	0.068 ***	0.081 ***	0.078 ***	0.090 ***	0.121 ***	0.118 ***	0.125 ***
	(0.020)	*(0.017)*	*(0.019)*	*(0.015)*	*(0.013)*	*(0.015)*	*(0.013)*
High School Diploma	0.060 ***	0.061 ***	0.066 ***	0.066 ***	0.081 ***	0.084 ***	0.084 ***
	(0.011)	*(0.009)*	*(0.011)*	*(0.009)*	*(0.009)*	*(0.010)*	*(0.008)*
College below Bachelor's	0.037 **	0.041 ***	0.040 ***	0.044 ***	0.057 ***	0.056 ***	0.058 ***
	(0.015)	*(0.012)*	*(0.015)*	*(0.012)*	*(0.011)*	*(0.013)*	*(0.011)*
Average change relative to...							
Column 7	*37%*	*31%*	*30%*	*25%*	*3%*	*3%*	*0%*
Column 4	*16%*	*8%*	*7%*	*0%*	-	-	-
Column 2	*9%*	*0%*	-	-	-	-	-
Controls							
Traditional[(a)]	✓	✓	✓	✓			
Skills[(b)]	✓	✓			✓		
Genotyping[(c)]	✓		✓			✓	

Notes: Marginal associations between education levels and probabilities of corresponding health-related outcomes are reported based on the logit factor model (3,4). The omitted education category is "Bachelor's degree or above." Effects of education are calculated at the average level of SES and other controls. Asterisks indicate statistical significance level: ***, 1 % level; **, 5 % level; *, 10 % level. Add Health data are used. Sample size is 3709. Percentages are average percentage changes of the three education coefficients reported. Column 1 corresponds to the unrestricted model (3). All other columns are restricted versions of the same model, with certain sets of controls omitted. Column 7 corresponds to a regression of outcomes on education dummies only. Other sets of controls are defined as follows: [(a)]Background controls that are documented in Table 2 including SES factor, but excluding genetic ancestry PCs. [(b)]Traditionally-measured cognitive and noncognitive skills, $\boldsymbol{S}$; [(c)]Data based on genotyping: 17 PGIs and 10 genetic ancestry PCs.

Table A-21: Conditional Marginal Association Between Education and Lack of Exercise Depending on the Set of Controls

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Education							
Below High School	0.063 ** *(0.030)*	0.055 ** *(0.027)*	0.076 *** *(0.028)*	0.074 *** *(0.024)*	0.072 *** *(0.019)*	0.084 *** *(0.022)*	0.091 *** *(0.018)*
High School Diploma	0.079 *** *(0.014)*	0.073 *** *(0.012)*	0.087 *** *(0.014)*	0.084 *** *(0.011)*	0.075 *** *(0.010)*	0.091 *** *(0.013)*	0.086 *** *(0.010)*
College below Bachelor's	0.054 *** *(0.017)*	0.040 *** *(0.014)*	0.058 *** *(0.016)*	0.047 *** *(0.014)*	0.047 *** *(0.013)*	0.065 *** *(0.016)*	0.055 *** *(0.013)*
Average change relative to...							
Column 7	*14%*	*27%*	*3%*	*12%*	*16%*	*-5%*	*0%*
Column 4	*2%*	*18%*	*-10%*	*0%*	-	-	-
Column 2	*-19%*	*0%*	-	-	-	-	-
Controls							
Traditional[(a)]	✓	✓	✓	✓			
Skills[(b)]	✓	✓			✓		
Genotyping[(c)]	✓		✓			✓	

Notes: Marginal associations between education levels and probabilities of corresponding health-related outcomes are reported based on the logit factor model (3,4). The omitted education category is "Bachelor's degree or above." Effects of education are calculated at the average level of SES and other controls. Asterisks indicate statistical significance level: ***, 1 % level; **, 5 % level; *, 10 % level. Add Health data are used. Sample size is 3709. Percentages are average percentage changes of the three education coefficients reported. Column 1 corresponds to the unrestricted model (3). All other columns are restricted versions of the same model, with certain sets of controls omitted. Column 7 corresponds to a regression of outcomes on education dummies only. Other sets of controls are defined as follows: [(a)]Background controls that are documented in Table 2 including SES factor, but excluding genetic ancestry PCs. [(b)]Traditionally-measured cognitive and noncognitive skills, $\boldsymbol{S}$; [(c)]Data based on genotyping: 17 PGIs and 10 genetic ancestry PCs.

Table A-22: Conditional Marginal Association Between Education and Smoking Cigarettes Depending on the Set of Controls

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Education							
Below High School	0.342 *** *(0.041)*	0.371 *** *(0.029)*	0.377 *** *(0.038)*	0.400 *** *(0.027)*	0.444 *** *(0.023)*	0.469 *** *(0.029)*	0.472 *** *(0.021)*
High School Diploma	0.248 *** *(0.021)*	0.264 *** *(0.015)*	0.269 *** *(0.020)*	0.284 *** *(0.014)*	0.296 *** *(0.014)*	0.302 *** *(0.018)*	0.312 *** *(0.013)*
College below Bachelor's	0.241 *** *(0.023)*	0.228 *** *(0.017)*	0.255 *** *(0.023)*	0.242 *** *(0.017)*	0.250 *** *(0.016)*	0.276 *** *(0.021)*	0.262 *** *(0.016)*
Average change relative to...							
Column 7	*19%*	*17%*	*12%*	*11%*	*5%*	*-1%*	*0%*
Column 4	*9%*	*7%*	*2%*	*0%*	-	-	-
Column 2	*3%*	*0%*	-	-	-	-	-
Controls							
Traditional[(a)]	✓	✓	✓	✓			
Skills[(b)]	✓	✓			✓		
Genotyping[(c)]	✓		✓			✓	

Notes: Marginal associations between education levels and probabilities of corresponding health-related outcomes are reported based on the logit factor model (3,4). The omitted education category is "Bachelor's degree or above." Effects of education are calculated at the average level of SES and other controls. Asterisks indicate statistical significance level: ***, 1 % level; **, 5 % level; *, 10 % level. Add Health data are used. Sample size is 3709. Percentages are average percentage changes of the three education coefficients reported. Column 1 corresponds to the unrestricted model (3). All other columns are restricted versions of the same model, with certain sets of controls omitted. Column 7 corresponds to a regression of outcomes on education dummies only. Other sets of controls are defined as follows: [(a)]Background controls that are documented in Table 2 including SES factor, but excluding genetic ancestry PCs. [(b)]Traditionally-measured cognitive and noncognitive skills, $\boldsymbol{S}$; [(c)]Data based on genotyping: 17 PGIs and 10 genetic ancestry PCs.

Table A-23: Conditional Marginal Association Between Education and Obesity Depending on the Set of Controls

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Education							
Below High School	0.020 *(0.054)*	0.038 *(0.040)*	0.038 *(0.050)*	0.066 * *(0.036)*	0.086 *** *(0.028)*	0.080 ** *(0.036)*	0.119 *** *(0.027)*
High School Diploma	0.069 *** *(0.023)*	0.104 *** *(0.017)*	0.084 *** *(0.022)*	0.121 *** *(0.016)*	0.143 *** *(0.015)*	0.125 *** *(0.019)*	0.162 *** *(0.014)*
College below Bachelor's	0.072 *** *(0.027)*	0.106 *** *(0.019)*	0.081 *** *(0.026)*	0.121 *** *(0.019)*	0.131 *** *(0.018)*	0.097 *** *(0.023)*	0.147 *** *(0.017)*
Average change relative to...							
Column 7	*54%*	*32%*	*47%*	*21%*	*17%*	*30%*	*0%*
Column 4	*51%*	*23%*	*35%*	*0%*	-	-	-
Column 2	*38%*	*0%*	-	-	-	-	-
Controls							
Traditional[(a)]	✓	✓	✓	✓			
Skills[(b)]	✓	✓			✓		
Genotyping[(c)]	✓		✓			✓	

Notes: Marginal associations between education levels and probabilities of corresponding health-related outcomes are reported based on the logit factor model (3,4). The omitted education category is "Bachelor's degree or above." Effects of education are calculated at the average level of SES and other controls. Asterisks indicate statistical significance level: ***, 1 % level; **, 5 % level; *, 10 % level. Add Health data are used. Sample size is 3709. Percentages are average percentage changes of the three education coefficients reported. Column 1 corresponds to the unrestricted model (3). All other columns are restricted versions of the same model, with certain sets of controls omitted. Column 7 corresponds to a regression of outcomes on education dummies only. Other sets of controls are defined as follows: [(a)]Background controls that are documented in Table 2 including SES factor, but excluding genetic ancestry PCs. [(b)]Traditionally-measured cognitive and noncognitive skills, $\boldsymbol{S}$; [(c)]Data based on genotyping: 17 PGIs and 10 genetic ancestry PCs.

Table A-24: Conditional Marginal Association Between Education and Depression Depending on the Set of Controls

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Education							
Below High School	0.079 **	0.102 ***	0.091 **	0.113 ***	0.084 ***	0.099 ***	0.101 ***
	(0.038)	*(0.029)*	*(0.035)*	*(0.026)*	*(0.022)*	*(0.027)*	*(0.020)*
High School Diploma	0.069 ***	0.069 ***	0.081 ***	0.080 ***	0.062 ***	0.078 ***	0.073 ***
	(0.018)	*(0.014)*	*(0.018)*	*(0.013)*	*(0.012)*	*(0.015)*	*(0.011)*
College below Bachelor's	0.054 ***	0.061 ***	0.060 ***	0.067 ***	0.060 ***	0.057 ***	0.066 ***
	(0.021)	*(0.016)*	*(0.021)*	*(0.015)*	*(0.014)*	*(0.019)*	*(0.014)*
Average change relative to...							
Column 7	*15%*	*4%*	*3%*	*-8%*	*14%*	*3%*	*0%*
Column 4	*21%*	*11%*	*10%*	*0%*	-	-	-
Column 2	*11%*	*0%*	-	-	-	-	-
Controls							
Traditional[(a)]	✓	✓	✓	✓			
Skills[(b)]	✓	✓			✓		
Genotyping[(c)]	✓		✓			✓	

Notes: Marginal associations between education levels and probabilities of corresponding health-related outcomes are reported based on the logit factor model (3,4). The omitted education category is "Bachelor's degree or above." Effects of education are calculated at the average level of SES and other controls. Asterisks indicate statistical significance level: ***, 1 % level; **, 5 % level; *, 10 % level. Add Health data are used. Sample size is 3709. Percentages are average percentage changes of the three education coefficients reported. Column 1 corresponds to the unrestricted model (3). All other columns are restricted versions of the same model, with certain sets of controls omitted. Column 7 corresponds to a regression of outcomes on education dummies only. Other sets of controls are defined as follows: [(a)]Background controls that are documented in Table 2 including SES factor, but excluding genetic ancestry PCs. [(b)]Traditionally-measured cognitive and noncognitive skills, $\boldsymbol{S}$; [(c)]Data based on genotyping: 17 PGIs and 10 genetic ancestry PCs.

B Omitted Variable Bias in Presence of an Interaction

Below we apply simple model considerations to understand the implications of omitted variables in a model with an interaction between two endogenous variables.

Suppose that there are unobserved parental traits PT_1 and PT_2 that affect the child's $EAPGI$ and parental SES respectively. Both traits also affect the child's outcome Y through home environment thus creating endogeneity. Traits PT_1 and PT_2 may correlate with each other.

Consider the following model in the population:

$$EAPGI = \beta_1 PT_1 + v_1 \tag{B.1}$$

$$SES = \beta_2 PT_2 + v_2 \tag{B.2}$$

$$\begin{aligned} Y = \gamma_0 \quad & +\gamma_1 \cdot PGI + \gamma_2 \cdot PGI \cdot SES + \gamma_3 SES \\ & +\boldsymbol{\gamma_4 X} + \gamma_5 PT_1 + \gamma_6 PT_2 + u \end{aligned} \tag{B.3}$$

Here, v_1 and v_2 represent a part of the total variation in $EAPGI$ and SES that creates no endogeneity problem in the outcome equation. Variable $\boldsymbol{X}$ represents background controls.

Suppose that equations (B.1), (B.2), and (B.3) make a perfectly specified regression model. In particular,

$$E[u|PGI, SES, \boldsymbol{X}, PT_1, PT_2] = 0.$$

To observe what we actually estimate when we omit the unobserved variables PT_1 and PT_2, let us substitute expressions for PT_1 and PT_2 from equations (B.1) and (B.2) into the outcome equation. Then we will get an equation that is similar to (B.3), but with no controls PT_1 and PT_2, and with some of the parameters changed:

$$Y = \gamma_0 + \tilde{\gamma}_1 \cdot PGI + \gamma_2 \cdot PGI \cdot SES + \tilde{\gamma}_3 SES + \boldsymbol{\gamma_4 X} + \tilde{u}, \tag{B.4}$$

where

$$\begin{aligned} \tilde{\gamma}_1 &= \gamma_1 + \tfrac{\gamma_5}{\beta_1} \\ \tilde{\gamma}_2 &= \gamma_2 + \tfrac{\gamma_6}{\beta_2} \\ \tilde{u} &= u - \tfrac{\gamma_5}{\beta_1} \cdot v_1 - \tfrac{\gamma_6}{\beta_2} \cdot v_2 \end{aligned} \tag{B.5}$$

Here $\tilde{u}$ is a new error term. We can see that estimates of $\tilde{\gamma}_1$ and $\tilde{\gamma}_2$ will be biased estimates of γ_1 and γ_2. However, coefficient γ_2 remains unchanged, and so we can expect no bias due to omission of PT_1 and PT_2.

To analyse the direction biases in $\tilde{\gamma}_1$ and $\tilde{\gamma}_2$, consider (without any loss of generality) PT_1 and PT_2 as productive parental traits, and consider Y as a positive outcome, such as good general health. Then we can expect PT_1 and PT_2 to positively affect $EAPGI$, SES, and Y, and so we can expect that $\beta_1 > 0$, $\beta_2 > 0$, $\gamma_5 > 0$, and $\gamma_6 > 0$. In line with the literature, we can also expect that $\gamma_1 > 0$ and $\gamma_2 > 0$. Therefore, we can see that $\tilde{\gamma}_1 > \gamma_1$ and $\tilde{\gamma}_2 > \gamma_2$, and so we can expect an upward bias in the estimated effect of PGI and SES.

The result that the estimate of γ_2 is unbiased depends on assumptions of our model. In particular, the model assumes that PT_1 and PT_2 enter the model without any interactions between them. If model (B.3) had an additional term $\gamma_7 \cdot PT_1 \cdot PT_2$, the estimated interaction would have been biased as well, with direction of the bias depending on the unknown sign of γ_7.

References

Young, J. K. and A. A. Beaujean (2011). Measuring personality in wave I of the National Longitudinal Study of Adolescent Health. *Frontiers in Psychology 2*.