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Abstract

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 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 not only for detailed traditional controls and cognitive-noncognitive skills, but also for a large set of PGIs that proxy health, skills, and environment, all of which are major expected confounders. This result is suggestive of a causal effect of education on health.

Key words: health, health behaviors, polygenic index, polygenic score, environmental bottleneck, Scarr-Rowe hypothesis, educational attainment, parental socioeconomic status, child development, education, mediators, pleiotropy, Add Health data

JEL codes: I12, I14, I24, J24

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 weighted sums of a person's molecular genetic variants.[1] Weights put on genetic variants in a PGI depend on which particular life outcome (phenotype) 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 three contributions. First, we show that the EA PGI predicts health-related outcomes even after controlling for education, which the EA PGI is designed to predict. While this result is not surprising given the polygenic nature of the EA PGI and the methodology of its construction, the finding is useful for two reasons. (1) It provides *empirical evidence* consistent with additional pathways from the EA PGI to health that are separate from education, a case of a pleiotropy.[2] We are not the first to provide evidence consistent with pleiotropic effects of the EA PGI (e.g., Barcellos et al., 2018; Barth et al., 2020; Papageorge and Thom, 2020), but we are the first to demonstrate associations between the EA PGI and a variety of health-related outcomes after controlling for detailed thresholds of educational investment. (2) Our findings of a sizable pleiotropic effect motivates a study of the mechanisms linking the EA PGI with health-related outcomes, which we do in a companion paper (Savelyev and Bolyard, 2025).

Second, 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 the EA PGI and health-related variables is strong and positive for subjects with high parental SES but low or nonexistent for low-SES subjects.[3] We thus add new results to the growing

[2]Pleiotropy typically refers to a situation in which a single gene influences more than one phenotype (e.g., Solovieff et al., 2013). Since PGIs aggregate multiple genetic variants, they may demonstrate pleiotropic effects as well, defined as a single PGI affecting more than one phenotype.

[3]To avoid cumbersome language such as "direct marginal conditional association," the word "effect" in this paper does not necessarily imply "causal effect," unless explicitly stated or implied.

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., 2023; Scarr-Salapatek, 1971).

Similar gene-by-SES interactions have been established for the effect of the 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 question: whether the interaction between the EA PGI and SES in affecting health-related outcomes is fully driven by a similar interaction that has already been shown for education. We test and reject this hypothesis. 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 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).

Third, we contribute to understanding the confounders behind the education-health gradient. 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 possible confounders behind the relationship (Galama et al., 2018; Grossman, 2022).

Among the expected major confounding factors in education-health studies are genetic endowments (e.g., Boardman et al., 2015; Conti and Heckman, 2010). From twin studies, we know that the average heritability across traits is about 50%, with health measures being among highly heritable ones (Polderman et al., 2015). The heritability of education estimates typically range from 20% to 70%, with 40% being the average estimate across studies (Branigan et al., 2013).

Genetic confounders have historically been viewed as unobservables but recently be-

come 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 the endowments, which makes them good candidates for proxies. The proxy model not only contributes to efficiency by reducing residual variance, but also has the potential to perfectly control for the omitted variable bias (Wooldridge, 2010).

To the best of our knowledge, this is the first time molecular genetic measures are used as proxies of major expected confounders behind the education-health gradient. 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 to a model that already controls for traditional background variables and cognitive-noncognitive skills reduces the estimated association between education and health by about 11%. However, education still exhibits a large and statistically significant association with general health and all other health-related outcomes after controlling for genomic proxies of skill endowments, general health endowments, mental health endowments, and environment. This novel result is suggestive of a causal effect of education on health and 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.

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 environment, as we explain below.

Parental genotypes influence offspring outcomes not only directly through genetic

transmission, but also via environmental pathways, a phenomenon known as "genetic nurture" (e.g., Wang et al., 2021). Due to genetic nurture and the peculiarities in the PGI construction, the EA PGI captures not only the subjects' own genetic endowments but also their environment.[4]

Both inherited and noninherited parental genotypes contribute to the correlation between the PGI and the environment. The path though noninherited genotypes is well documented by Kong and Thorleifsson (2018), who show that the non-inherited parental endowments that are still passed down to children through the environment account for about 30% of the variation in education endowments explained by the EA PGI. As for the inherited genotypes, they also contribute to the correlation between PGIs and the environment, as some parental traits that affect the environment are genetically inherited by children. Overall, the confounding role of the environment is known to be large for the EA PGI. Based on a comparison between raw and within-sibship estimates, Howe et al. (2022) have shown that accounting for the environment reduces the association between the EA PGI and education by 50%.

The implications of these limitations of the EA PGI differ across our contributions. For our third 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 environment. For our first and second contributions on the association between the EA PGI and health as a function of SES, the correlation between the EA PGI and SES with unobserved family environment in the error term can be expected to lead to biased estimates. So does measurement error in the 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

[4]The 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).

confirmatory analysis (Savelyev and Bolyard, 2025).

2 Data

Add Health is a nationally representative panel dataset that follows roughly 20,000 individuals and contains detailed information on their family background, skills, education, and life outcomes in young adulthood (Harris, 2013). 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.

Our estimation sample size of 3,709 is constrained by the availability of genetic data and the reliability of the imputed EA PGI. First, we restrict our sample to those who participated in genotyping. Second, because the EA PGI that we use is constructed based on data collected from individuals with European ancestry, we restrict our sample to those who self-report as white. Here we rely on the established result that PGIs constructed using European-ancestry samples are both biased and less predictive when applied to populations with different ancestry (Martin et al., 2017).

In Table 1 we show descriptive statistics for the highest level of education, health, health behaviors, and potential mechanisms behind the effect of the EA PGI on health. We study health-related outcomes 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). Obesity and depression can also 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.

To measure cognitive skills, we use participants' scores on the Add Health Picture Vocabulary Test, recent science grades, and recent math grades as dedicated measures

Table 1: Education, Health-Related Outcomes, 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 summarized. See measures listed in Table A-1. [m]Bands: 1(lowest)–12. [n]Bands: 1(lowest)–9. [o]Self-rating: 1(least)–4.

of a latent cognitive factor. To measure noncognitive skills 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.[5]

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 Table A-1 of the Web Appendix.

From the Table 1 we can see that high-SES subjects tend to have 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, graduating 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.

SES To study the interaction of respondents' genetic endowments with family SES in their childhood, we follow the literature on PGI-SES interaction (Bierut et al., 2023; Papageorge and Thom, 2020; Ronda et al., 2020), and construct measures of SES from relevant variables that are available in the Add Health data. 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

[5]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.

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. (2023) 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 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, which makes the histogram right-skewed.

Finally, Panel C shows a histogram of an SES factor score that is implied by the

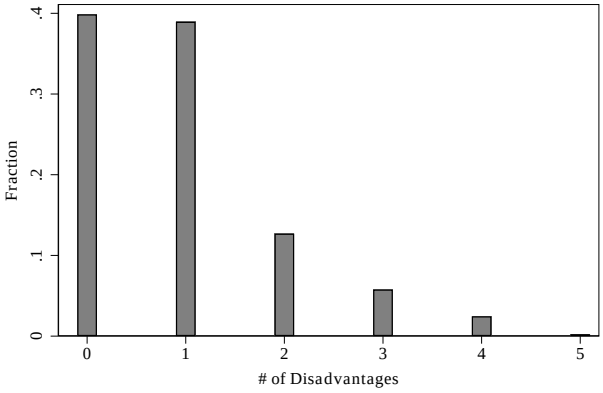
[6] "Parental college" is our only positive measure of SES, and so the corresponding "lack of parental college" is used for a count of disadvantages.

Figure 1: Description of SES

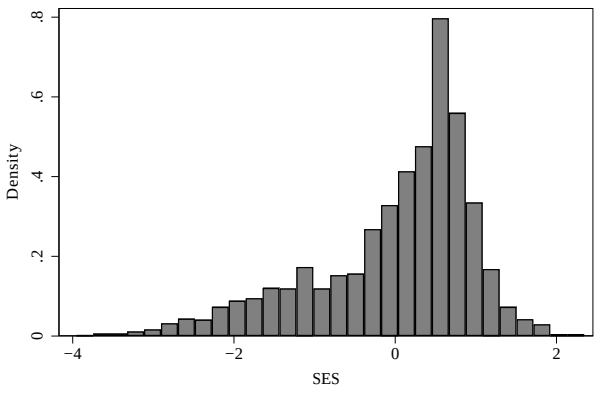
A. Measures of SES

	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, including 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 sizable mass of reported income exactly at the 20th percentile leads to the average of 0.19 rather than 0.20.)

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 in Panel B is a negative measure of SES. 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 area around zero and above in Panel C roughly corresponds to having at most one disadvantage. The long left tail in Panel C corresponds to having two or more disadvantages.

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 a weighted aggregate of multiple SNPs. PGIs demonstrate considerably stronger predictive power 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 the 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

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

and different numbers of aggregated SNPs, among other choices. In this paper we rely on the recent state-of-the-art the 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 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 (Beauchamp et al., 2011; Benjamin et al., 2012). For technical details behind PGIs used in this paper, see Braudt and Harris (2018) and Okbay et al. (2018).

In addition to modeling the effects of the EA PGI, which is our main variable of interest, we also take advantage of PGIs that proxy health endowments: nine PGIs that describe physical health endowments[8] and seven mental health PGIs.[9] The choice of these PGIs is determined by their availability in the AddHealth data. We demonstrate correlations between the 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 In addition to controlling for the 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 groups, US regions, 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. See Table A-4 in the Web Appendix for variable definitions and descriptive statistics by SES.

[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.

3 Methodology

Model of the EA PGI and Health-Related Outcomes For our study of the effect of the EA PGI on health-related outcomes and on potential mechanisms behind health formation, we employ a model that accounts for an interaction between the EA PGI and parental SES. This approach 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).

Our simple reduced form outcome model is comparable to models used in recent economic papers on gene-environment interactions (e.g., Barth et al., 2020; Bierut et al., 2023; Papageorge and Thom, 2020). The model is specified as follows:

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

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 a standardized 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[11], 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

[11]See Table A-4 in the Web Appendix.

viewed as a proxy for family investments in a child's human capital. 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).[12]

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.

Estimating the Association between the EA PGI and Health-Related Outcomes Conditional on Education We also estimate a model that is similar to model (1) but is conditional on education $\boldsymbol{D}$. This version of the model helps us establish the part of the association between the EA PGI and an outcome of interest is **not** explained by education. The causal analogue of this association is the direct effect of the 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.[13]

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 educa-

[12] We also tested and rejected a number of 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.

[13] 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.

tion and its interactions. Similarly to model (1), model (2) is estimated jointly with the measurement system (4).

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 strong correlation between environment and children's PGIs, these PGIs also proxy unobserved environment (Howe et al., 2022; Kong and Thorleifsson, 2018), which is another major expected confounder of the effect of education on health.

Proxies help eliminate or mitigate the omitted variable bias while also reducing the residual variance. PGIs make good proxies as they are highly predictive, a feature that makes the proxi model assumptions more plausible (Wooldridge, 2010). 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. 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 the 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 the 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 in addition to the EA PGI, resulting in a vector of 17 PGIs denoted as $\boldsymbol{PGI}$. We also control for early cognitive and noncognitive skills through 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 interaction 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.

Further, 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.[14] This comparison helps us explore how controlling for a large number of genomic controls affects associations $\boldsymbol{d}_{1k}$. We also explore how associations $\boldsymbol{d}_{1k}$ change when we omit all traditional controls, $\boldsymbol{X}$, all cognitive-noncognitive controls, $\boldsymbol{\theta}^{CN}$, or various combinations of these types of controls.

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), we need additional information provided by the measurement system (4).

[14] The first principal components of genetic data control for ethnic origin and serve as 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.

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:

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

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.

We follow the literature on factor model specification testing by calculating several established specification statistics, which are consistent with correct model specification. 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.[15]

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.[16] Finally, the

[15]See Web Appendix D for these specification and robustness checks.

[16]Our first SES measure is "living in an unsafe neighborhood," a negative measure

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).

Advantages of the Factor Model Over Its Alternatives By using a factor model rather than alternative methods of dimensionality reduction,[17] we gain several advantages: we explicitly control for measurement error, avoid arbitrarily equal weights,[18] and control for possible systematic determinants of peoples' perceptions that may affect answers. 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. See Web Appendix B for a more detailed discussion of the factor model advantages.

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. All reported standard errors account for the imputation error.

of SES, and so 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.

[17] For instance, relying on an equally-weighted index, a binary variable that aggregates all measures (e.g., "having at least two disadvantages"), or principal components.

[18] We test and reject the equality of weights in the Web Appendix B.

4 Results

Our empirical part is split in two sections, 4.1 and 4.2. In Section 4.1, which is devoted to our contributions 1 and 2, we first present a number of descriptive graphs to motivate our regression analysis. Then we study conditional associations between the EA PGI and health-related outcomes as a function of parental SES. Afterwards, we proceed to suggestive evidence regarding the mechanisms behind these relationships.

Section 4.2 is devoted to our contribution 3. Here we study the conditional association between education and health-related outcomes and establish the relative confounding role of of the traditional, cognitive-noncognitive, and genomic controls.

4.1 PGI, SES, and Health

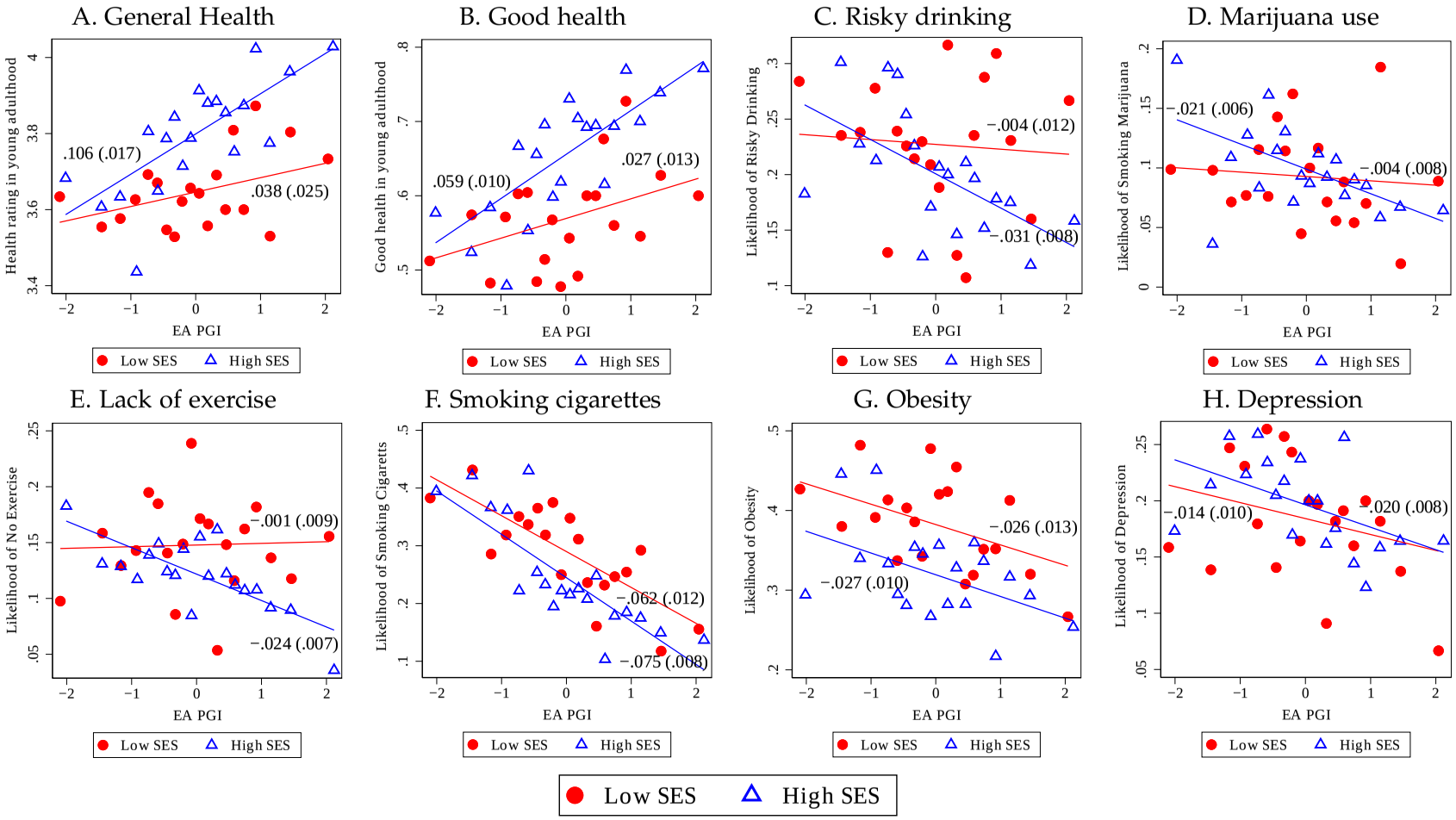
4.1.1 Descriptive Results

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 related results: (1) The bin scatter plot for the relationship between the EA PGI and a health-related outcome by SES. Each such scatter plot is a nonparametric estimate of the conditional expectation function; (2) A superimposed univariate linear regression line of a health-related outcome regressed on the 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: (1) For subjects with high parental SES, a higher EA PGI tends to correspond to better health or a smaller likelihood of an adverse health behavior. (2) For subjects with low parental SES, the relationship between the EA PGI and health-related outcomes tends to be weaker or statistically

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



Notes: Calculations are based on the AddHealth Data. [(a)]For 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.

insignificant.

4.1.2 Main Model Estimates

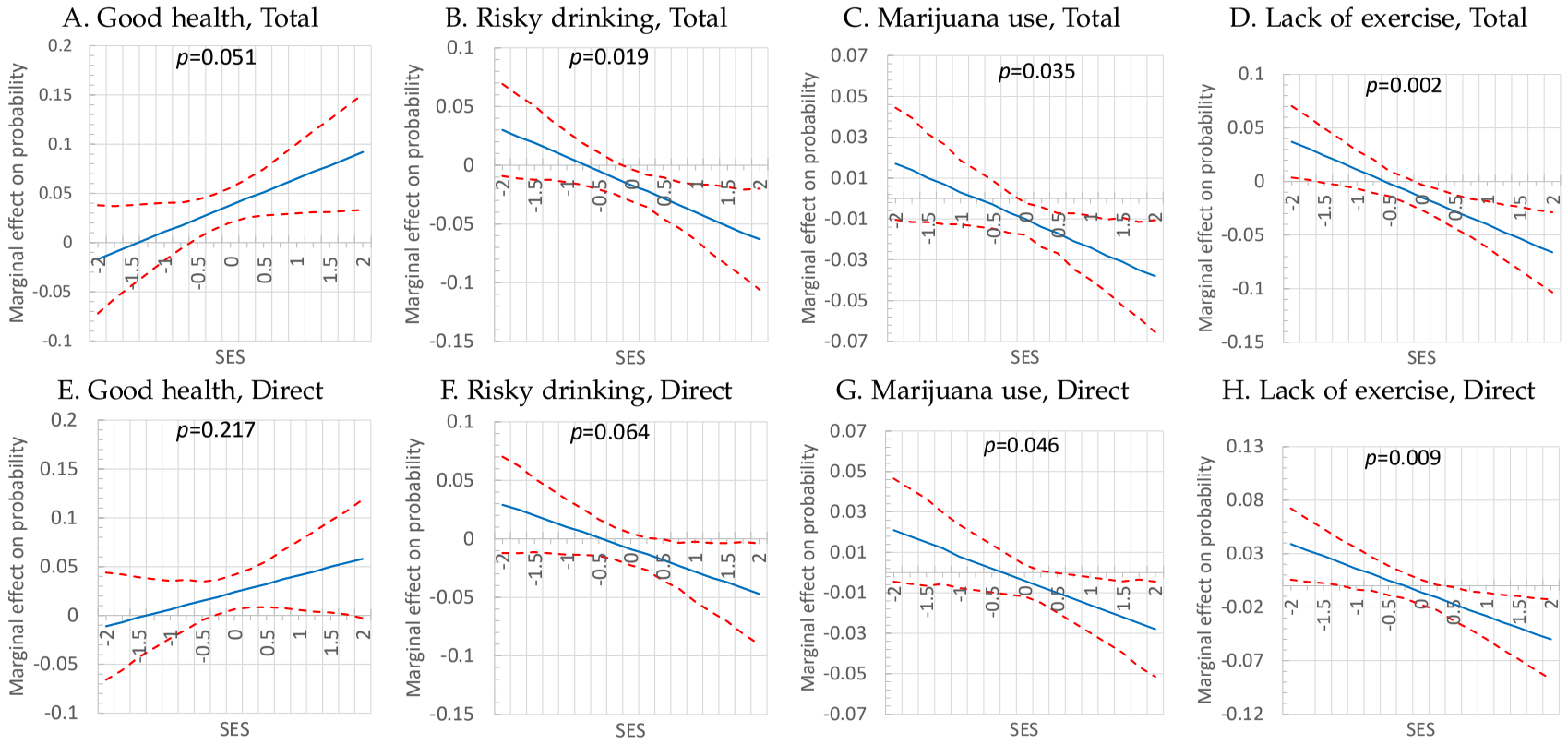
We first estimate an association between the EA PGI and health-related outcomes while allowing for an interaction between the EA PGI and parental SES. Figure 3 visualizes estimated relationships by showing marginal effects of the EA PGI on health-related outcomes as a function of standardized parental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

The upper panels of Figure 3 show the total effects of the 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 the 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.

Panel A of Figure 3 shows a marginal effect of the 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.

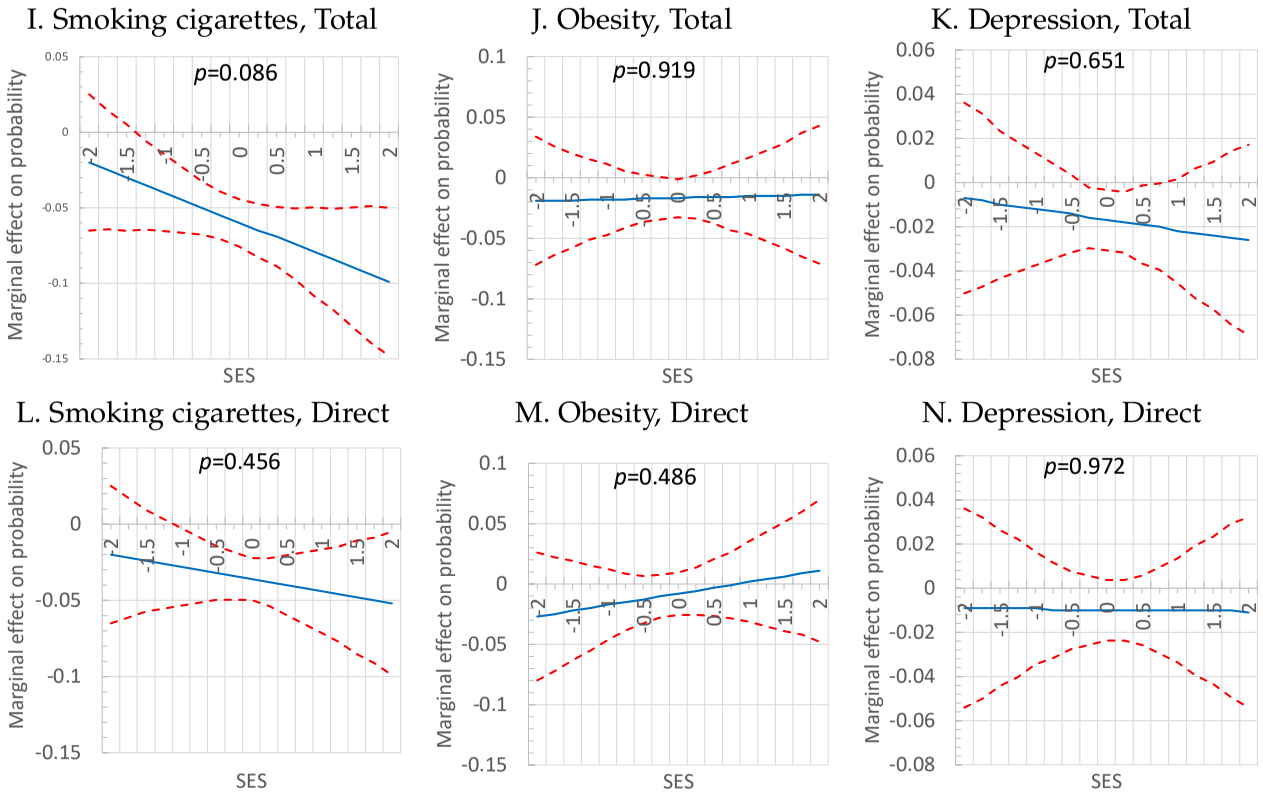
Apart from p-values that allow us to test for the interaction effect directly, we observe results that are consistent with the interaction effect: a small and statistically insignificant effect of the EA PGI at the low levels of SES, as opposed to a large and statistically significant effect at the high SES levels. For instance, an increase in the 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

Figure 3: Total and Direct Marginal Effects of the EA PGI on Health-Related Outcomes 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 the 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-6 and A-7 of the Web Appendix. See also Table A-5 for parameters of the measurement system (4). Calculations are based on the AddHealth Data.

Figure 3: Total and Direct Marginal Effects of the EA PGI on Health-Related Outcomes 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 the 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-6 and A-7 of the Web Appendix. See also Table A-5 for parameters of the measurement system (4). Calculations are based on the AddHealth Data.

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 the total effect in Panel 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 the EA PGI is statistically significant in Panel E at the average level of SES and above and is not statistically significant at the low level of SES. Numerically, the direct effects are 3.8% (0.024/0.625), at SES=0, and 6.6% (0.041/0.625) at SES=1.

All other panels are constructed in a similar way and a number of them show similar self-explanatory results, with the difference that the effect on adverse health-related outcomes and the associated slope tend to be negative, not positive. We discuss these results below.

Discussion Overall, we can see that all of the estimated total interaction effects that are statistically significant at least at the 10% level have the same sign as effects of the 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 the 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.[19]

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

[19]Effects sizes at $SES = 0$ are based on the associations, all of which are statistically significant at the 5% level. Numerically, the total effect sizes are: general health, 6.1%; risky drinking, -3.7%; marijuana use, -10%, lack of physical exercise, -12%, smoking cigarettes, -23%; obesity, -4.9%; depression, -8.9%.

interaction with SES for a number of them. Even after controlling for education, we still find effects of the EA PGI on health-related outcomes, as well as evidence of the interaction effect with SES.[20]

Our results complement those found by Bierut et al. (2023), as we find similar interaction effects but for a different type of PGI (we use the EA PGI, not the 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 the EA PGI and childhood SES to study the determinants of education.

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 a lower EA PGI, providing evidence that a combination of severe environmental conditions and an unfavorable genetic endowment is particularly harmful. Avinun (2019) finds that the 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.

Related to environmental bottlenecks is the Scarr-Rowe hypothesis: an exposure to socioeconomic disadvantage leads to a 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 a related type of environmental bottleneck effect that has been established earlier based on IQ scores, even though the EA PGI and the IQ

[20] 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.

are very different in terms of their construction and limitations.

Limitations As discussed in the introduction, the EA PGI correlates with the environment that we only partially control for, which likely creates an upward bias (by absolute value) in the estimated effect of the EA PGI at $SES = 0$ relative to the true causal effect of genetic endowment. In addition, measurement error in the EA PGI is expected to create an attenuation bias in the same estimate. While there is a benefit of these two biases partially canceling each other, the direction of the resulting bias is indeterminant.[21]

For the interaction term, we argue that if unobserved parental endowments contribute to the error term of the outcome equation in a linear way, no omitted variable bias is created under a set of assumptions (see Web Appendix C). While we account for the measurement error in SES in our factor model, measurement error in the EA PGI leads to measurement error in the SES-EA PGI interaction. Our strong and statistically significant estimates of the interaction are found despite this expected attenuation bias.

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

Low Statistical Power of Sibling Fixed Effects It would be ideal to rely on the sibling fixed effect to establish the causal effect of the 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 PGI effects could be interpreted as causal effects of one's own genetic endowments. However, we find 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.

[21] See also Biroli et al. (2022) for a detailed analysis of possible biases in models involving a gene-by-environment interaction.

4.1.3 The Mechanisms

To better understand the effects of the EA PGI on health and health behaviors in young adulthood, the effects we have discussed in Section 4.1.2, we provide suggestive evidence for the mechanisms behind the estimated effects.

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 makes the likelihood of capturing the reverse causal effect small. 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, these suggested mechanisms should be interpreted with extra caution. Overall, our aim in this exploratory study is to identify multiple potential mechanisms, with testing for their possible causal status left for future research.

Health Behaviors The partition between health-related outcomes 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 the EA PGI on general health, all documented in Figure 3, which we have discussed above. Specifically, the results for the positive effect of the 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 the 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 the 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 the 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).

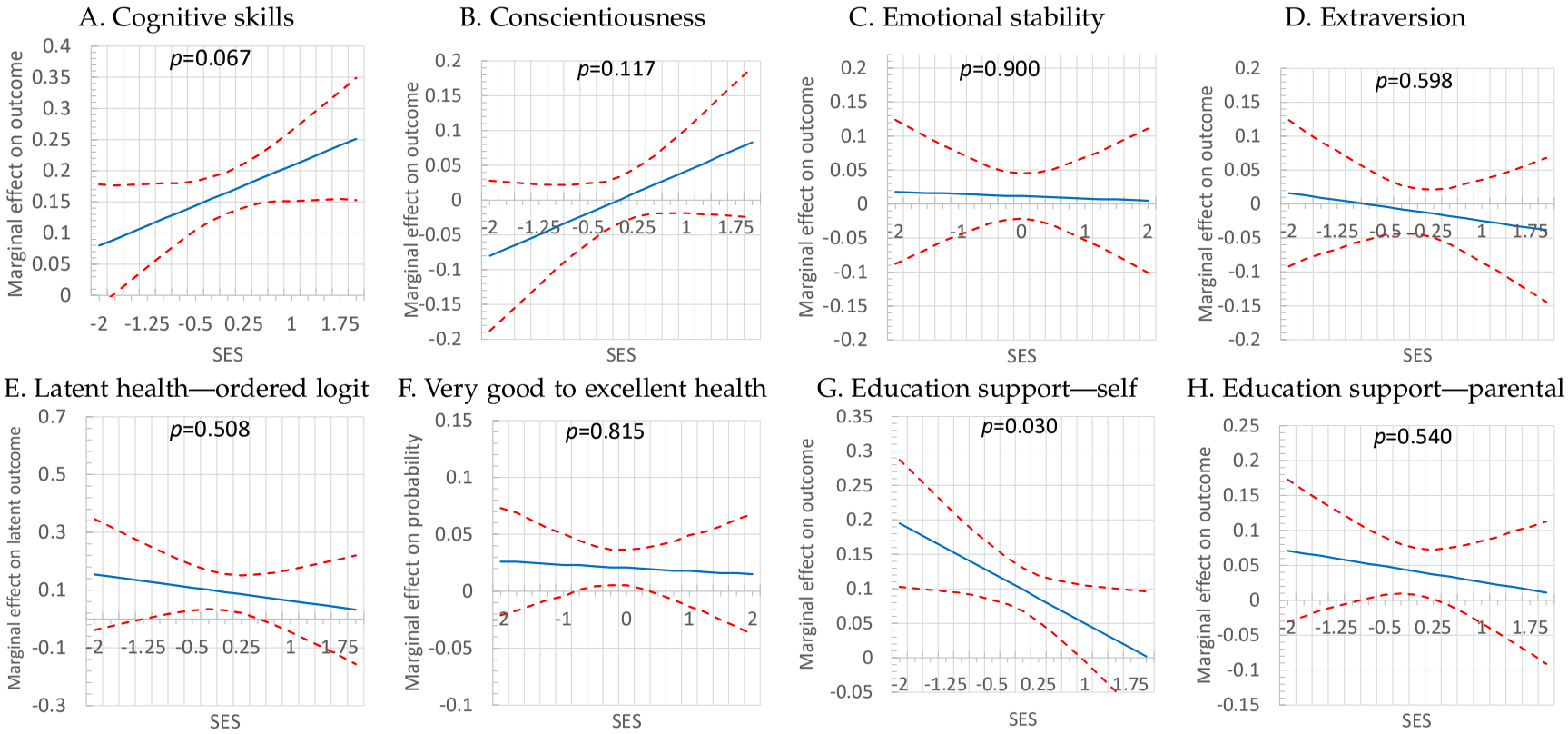
However, the possible effects of health behaviors on health stock suggested above might be small or negligible given that we study health stock in early adulthood, ages 24–32.

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 ($SES = 0$), the 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 the 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 causal and another spurious. The causal explanation of the positive relationship between the 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 the 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 the estimated associations may capture both causal and spurious components.

Apart from explaining the mechanisms behind the effect of the EA PGI on health-

Figure 4: Total Marginal Effects of the EA PGI on Potential Early Life Health Mechanisms as a Function of Parental SES



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-8 of the Web Appendix. See also Table A-5 for parameters of the measurement system (4). Calculations are based on the AddHealth Data.

related outcomes at the average SES level, we seek to explain the mechanisms behind the interaction between the EA PGI and SES to better understand the origins of the interaction effect. However, among early mechanisms that we study, 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 the 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 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.

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 the EA PGI, SES, and EA PGI$\times$SES conditional on other controls and find that most early measures of drinking alcohol, smoking cigarettes, and being overweight in adolescence are predicted by the EA PGI. However, the interaction with SES is not precisely determined.[22] Secondly, we regress health behaviors in adulthood on EA PGI, SES, and EA PGI$\times$SES conditional on corresponding early behaviors and other controls, and compare these results with our main model, which does not condition on early behaviors.[23]

We find that early behaviors are predictive of later behaviors, and that associations

[22]See Table A-9 of the Web Appendix.
[23]See Table A-10 of the Web Appendix.

between the 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 the EA PGI on later behaviors. However, there is a substantial part of the association that appears 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.

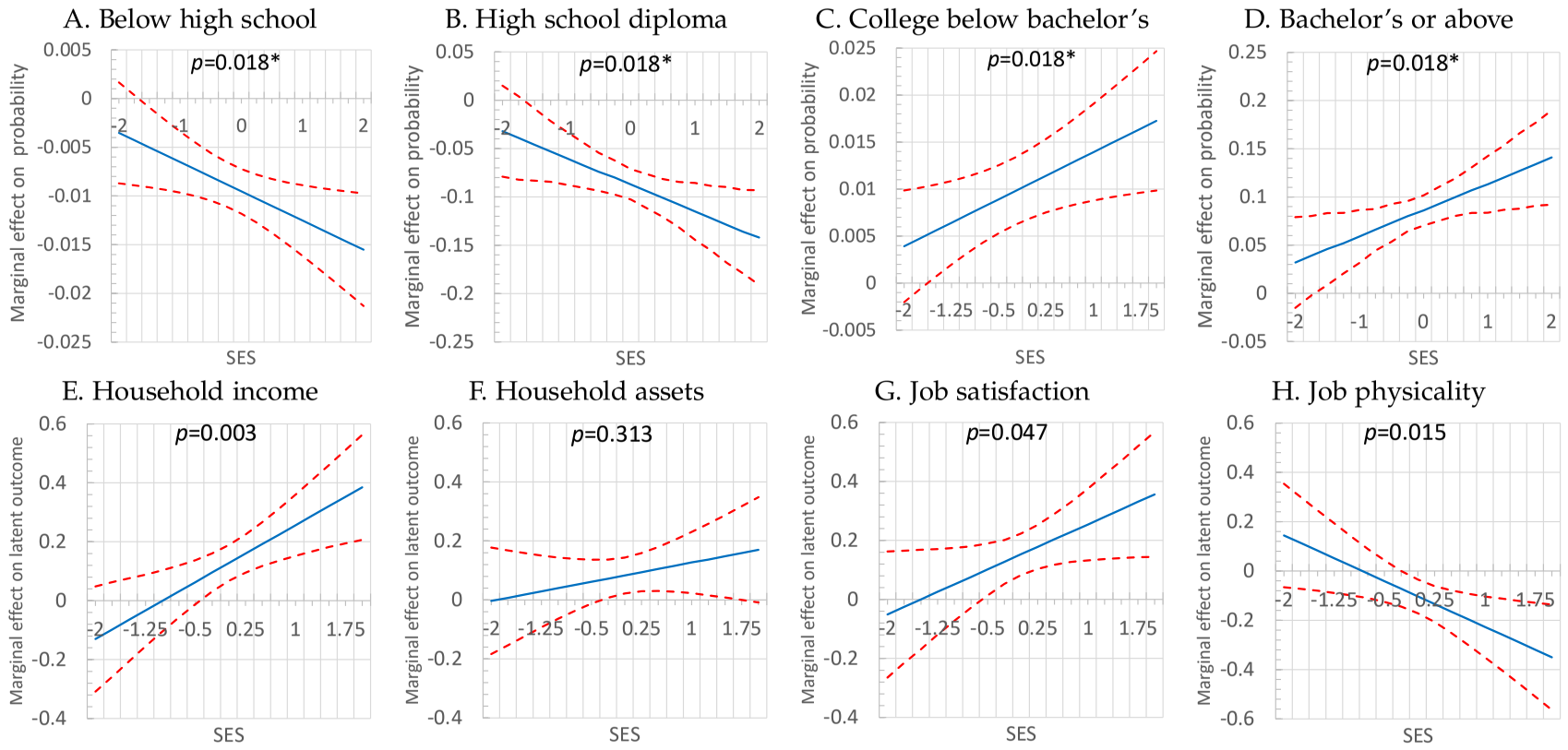
Education Panels A–D of Figure 5 show marginal effects of the EA PGI on the probabilities of achieving different highest education levels as functions of standardized SES. These four graphs are based on the same underlying ordered logit model of education (1), estimated simultaneously with the measurement 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 the EA PGI stronger. All results are precisely determined and effect sizes are large. At the average SES, effect sizes of the 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 ($= 0.086/0.363$).[24]

These results for education are expected because the EA PGI is specifically designed to predict years of formal education and because positive interaction with SES is documented in the literature (Fletcher, 2019; Papageorge and Thom, 2020; Ronda et al., 2020). Therefore, results in Panels A–D of Figure 5 serve two purposes: (1) to verify the existing results on the EA PGI-SES interaction using a different dataset; (2) to test whether these

[24]See Table A-11 of the Web Appendix for effect sizes and estimates behind Figure 4.

Figure 5: Marginal Conditional Associations Between the 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-11 of the Web Appendix. Panels E–H are based on ordered logit models documented in Table A-12. See also Table A-5 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.

expected relationships can help us explain the mechanisms behind the effects of the EA PGI on health for a specific population that we study.

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, the EA PGI is positively related to household income, household assets, and job satisfaction. Job physicality is affected negatively. However, none of these effects take place at the low SES levels.

These findings are consistent with our results in Figure 3, as they suggest the mechanisms that drive the relationship between the EA PGI and health-related outcomes and its interaction with SES. Viewing income (a flow) and assets (a related stock) as potential mechanisms is 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.[25]

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 faster heath declines despite positive health selection that is typical for physical jobs (Case and Deaton, 2005; DeLeire and Levy, 2004; Fletcher et al., 2011; Ravesteijn et al., 2018).

[25]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).

4.2 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. Those are proxied by 17 different types of PGIs. As discussed above, PGIs do not only proxy own genetic endowments but also the environment, which allows us to proxy the confounding variation even better.

Associations Conditional on Multiple PGIs Table 2, 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 the EA PGI, nine types of PGIs related to aspects of physical health, and seven types of mental health PGIs.[26]

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.[27] The results based on individual tests are supported by joint tests, all of which are rejected at the 5% level of significance. Those include Wald tests of two types: (1) Joint tests across all seven health-related outcomes, which are performed for each

[26] See Section 2 for more details about these PGIs.

[27] 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)).

Table 2: Marginal Effects of Education Categories on Health-Related Outcomes in Young Adulthood

	Excellent or very good health	Risky drinking	Marijuana use	Lack of exercise	Smoking cigarettes	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 A-4. [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).

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 2 is a joint test for the interaction between educational categories and parental SES, presented in Panel B. This interaction appears at best weak.[28] Therefore, while the effect of the EA PGI on education strongly depends on SES, as we have seen in the previous section, there is no robust evidence for an interaction with SES in the effect of education on health. 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.

We offer two explanations for our failure to establish an SES interaction with education. 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 for reasons such as critical and sensitive periods in childhood, dynamic complementarity, and self-productivity (Heckman, 2007). As we have shown earlier, the 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 the 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. We explore the following groups of controls to be defined

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

below: traditional controls, skills, and genomic 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 A-4, excluding genetic ancestry PCs). Plus, we include the SES factor in a set of traditional controls, as SES factor is identified from 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 typically modelled jointly using factor analysis to recognize the importance of multidimensional human capabilities and to account for measurement error (e.g., Heckman et al., 2013, 2006).[29]

Our final type of controls, labeled as "genomic," have been recently introduced to economic research by genoeconomists (Benjamin et al., 2012). In our paper, this group includes 17 PGIs that proxy genetic endowments for education and health and the first 10 principal components of genetic data, which are standard genomic controls for ethnic differences.[30] Those controls are based on genotyping combined with new techniques of processing genomic measurements.

Column 1 in Table 3 shows the results of the unrestricted model (3), while columns 2–7 display the results of various restricted models, with restrictions defined in Panel C. Panel A 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.

The most basic model, a regression of outcomes on education dummies only, is shown

[29] 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. 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.

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

Table 3: Marginal Effects of Education on Self-Reported Health: Comparing Models that Use Different Sets of Controls, Logit Model Estimates

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
A. Education							
Below High	-0.260 ***	-0.257 ***	-0.302 ***	-0.297 ***	-0.295 ***	-0.342 ***	-0.368 ***
School	*(0.055)*	-0.154	*(0.051)*	*(0.035)*	*(0.041)*	*(0.036)*	*(0.027)*
High School	-0.132 ***	-0.154 ***	-0.161 ***	-0.188 ***	-0.189 ***	-0.224 ***	-0.245 ***
Diploma	*(0.024)*	*(0.017)*	*(0.023)*	*(0.016)*	*(0.020)*	*(0.020)*	*(0.014)*
College below	-0.095 ***	-0.119 ***	-0.116 ***	-0.146 ***	-0.144 ***	-0.160 ***	-0.190 ***
Bachelor's	*(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. Controls							
Traditional[(a)]	✓	✓	✓	✓			
Skills[(b)]	✓	✓			✓		
Genomic[(c)]	✓		✓			✓	

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)]Background controls that are documented in Table A-4 including SES factor, but excluding genetic ancestry PCs. [(b)]Cognitive and noncognitive skills; [(c)]Data based on genotyping: 17 PGIs and 10 genetic ancestry PCs.

in column 7. This basic model makes a useful benchmark for comparisons. 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 way: genomic 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 C 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 multidimentional 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%; genomic 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%.

To summarize, after controlling for traditional background variables and skills, the incremental change in associations due to missing genomic proxies for health endowments, skill endowments, and environment is 11%. While this bias is sizable, it is at odds with the hypothesis that the strong association between education and health is entirely driven by unobserved confounders, of which skill endowments, health endowments, and environment are the most expected ones.

Discussion Our result contributes to the literature on the effect of education on health and the confounders behind this relationship. In this literature, apart from regressions conditional on observable controls and propensity score methods, there are four major

approaches that attempt to identify the effect of education on health-related outcomes: (1) randomized controlled trials (RCTs) (2) natural experiments; (3) family/twin fixed effects; (4) the explicit modeling of unobserved heterogeneity.

These approaches have their advantages and disadvantages. Approach 1 has the most persuasive source of exogenous variation, but it is limited to early childhood education due to ethical considerations (Conti et al., 2016). Approach 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 causal 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).

Approach 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 approach 2, the results based on approach 3 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.

Approach 4 explicitly models the relationships between observed and unobserved confounders, education, and health-related outcomes. These methods preserve statistical

power better than approaches 2 and 3. Also, unlike approach 2, approach 4 attempts to estimate the Average Treatment Effect (ATE) rather than the Local Average Treatment Effect (LATE). The results of approach 4 are more consistent than 2 and 3, as authors tend to find positive effects of education on health (e.g., Bijwaard et al., 2015; Conti and Heckman, 2010; Hong et al., 2020; Savelyev, 2022; Savelyev and Tan, 2019). The biggest concern with approach 4 that relies on the conditional independence assumption and its generalizations is its ability to adequately account for possible remaining unobserved confounders. This paper diminishes concerns about the results based on approach (4) by controlling for a large number of PGIs, which we use to proxy genetic endowments for skills, physical health, mental health, and environment, and establishing that the association between education and health survives controlling for such proxies.

We also contribute to discussions of the confounders behind the education-health gradient. 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 for, 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) (CH) 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, CLM, and CH is showing the selection bias correction due to molecular genetic proxies of health, ability, and environment.

5 Conclusions

We find that the EA PGI exhibits strong and health-beneficial conditional associations with a variety of life outcomes in young adulthood and that these associations are not fully driven by education as a mechanism. 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 second contribution of this paper provides novel evidence regarding an additional major benefit of such policies. 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 third contribution 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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