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Abstract

We investigate an Educational Attainment Polygenic Score (EA PGS), an index that predicts years of formal education based on individual genetic data. In our analysis of the National Longitudinal Study of Adolescent to Adult Health we find that the EA PGS is associated with a number of health-related outcomes and interacts with parental socioeconomic status (SES) in childhood. We observe that the association between EA PGS and health-related outcomes is typically strong and health-beneficial for high-SES subjects, but small or nonexistent for low-SES ones. We provide suggestive evidence of the mechanisms behind estimated relationships including early health, skills, parental and child's own attitude towards education, education itself, as well as outcomes related to occupation and wealth. Finally, we show that a strong association between education and health-related outcomes survives controlling for genetic endowments for health and education, which adds evidence to the ongoing debate about the causal relationship between education and health.

Key words: Educational Attainment Polygenic Score, socioeconomic status, environmental bottleneck effect, Scarr-Rowe hypothesis, health, health behaviors, education, mechanisms, Add Health data

JEL codes: I12, I14, I24, J24

1 Introduction

Researchers are quickly gaining access to a wealth of genetic data, creating opportunities to answer new research questions and revisit old ones. There are many ways for economists to benefit from this development. The new availability of reliable measures of endowments allows economists to study topics such as interactions between endowments and socioeconomic environment, intrafamily resource allocation, and effects of one's own endowments on human capital investment in oneself. Other opportunities include using genetic data to construct instrumental variables (*Mendelian randomization*), or to directly control for genetic confounders in econometric models.

However, predictions based on single DNA building blocks, called SNPs,[1] lead to low statistical power and issues with replicability, as many life outcomes are affected by multiple SNPs. A well-established solution to this problem is using a polygenic score (PGS) instead of a SNP. A PGS is an optimally-weighted aggregate of multiple SNPs. PGSs demonstrate considerably stronger predictive power and more robust results across populations than a single SNP (e.g., Benjamin et al., 2011).

This paper is concerned with a specific PGS called Educational Attainment PGS (EA PGS). The EA PGS, which measures an individual's genetic predisposition for the total number of years of formal education, has particularly important socioeconomic implications. The EA PGS has been studied already in the literature. However, we would still benefit from more knowledge about the predictive power of the EA PGS outside of the life outcome it is constructed to predict, the interaction between the EA PGS and parental socioeconomic status, the mechanisms through which EA PGS affects life outcomes, and the consequences of adding EA PGS and other types of polygenic scores as additional controls to traditional models.

Such a study is useful because of the importance of understanding how and why hu-

[1] Single-nucleotide polymorphisms (SNPs, pronounced "snips") are nucleotides at a particular location on the genome that represent variation among humans.

man capital seems to be linked to better life outcomes, including health. A study of the mechanisms helps suggest policy implications—not because genetic factors should be manipulated by policy, but to provide a better understanding of why high EA PGS individuals have better health and how EA PGS interacts with parental SES. These relationships shed light on policies that can substitute for the sources of advantage associated with SES and higher levels of the EA PGS.

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 in 1996 through young adulthood. We study a variety of health outcomes related to general and mental health, substance use, exercise, and body weight. Our sample is restricted to white individuals due to data limitations and the well-established result that imputing a PGS outside the ethnicity for which it was constructed can lead to a bias and a loss of statistical power (Martin et al., 2017).

Our paper offers two main contributions. First, we demonstrate a novel result that conditional association between the EA PGS and health-related outcomes in young adulthood depends on parental socioeconomic status (SES). We observe that EA PGS is more beneficial for high-SES subjects. The interaction is so strong that a health-beneficial association between the EA PGS and a number of health-related outcomes is fully canceled when SES is low enough. Conversely, for high-SES subjects we observe associations that are both statistically and economically significant. Therefore, we add new results to the growing literature on environmental bottlenecks. We also provide suggestive evidence for the mechanisms behind the established relationships, which include early skills, early health, parental support of the child's education, the child's own motivation for education, education itself, occupation, household income, and household wealth in young adulthood.

Second, we contribute to a separate body of literature on the relationship between education and health, the causal status of which is still debated, as we discuss in Section

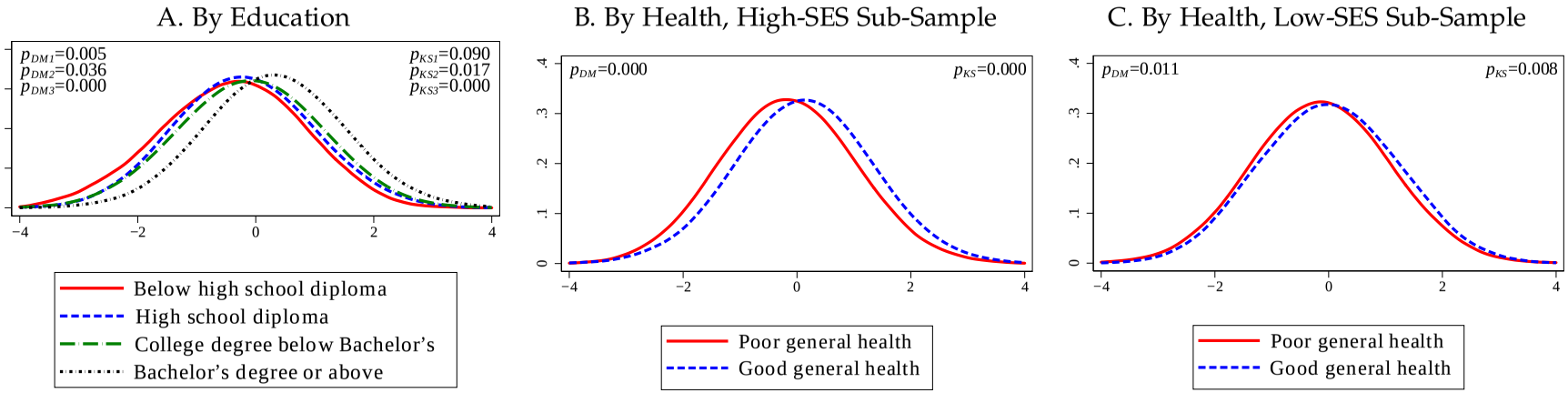
2. Among expected confounding factors in education-health studies are genetic endowments, which may be strongly predictive of both education and health (e.g., Boardman et al., 2015; Conti and Heckman, 2010a). We find, however, that education still exhibits large and statistically significant associations with a variety of health outcomes when, on top of cognitive skills, noncognitive skills, and detailed traditional background controls, we add a large set of PGSs that control for skill and health endowments. This novel result is consistent with a causal relationship between education and health.

Following a number of recent economic papers on polygenic scores, we use a reduced form approach (e.g., Barth et al., 2020; Bierut et al., 2018; Papageorge and Thom, 2020). However, there is a possible source of confounding behind the estimated associations between EA PGS and outcomes, which is family influence. The confounding arises from correlation between own and parental genetic endowments (e.g., Howe et al., 2022). We partly control for parental influences through background controls, but this cannot rule out a remaining confounding influence. To address this issue, we support this reduced-form general paper with a more technical companion paper (Savelyev and Bolyard, 2022), which explicitly models the contribution of the mechanisms to total effects using systems of simultaneous equations and shows that our qualitative reduced-form results are preserved after accounting for unobserved heterogeneity that includes parental genetic endowments.

We also perform an alternative estimation strategy using family fixed effects based on 200 families who have at least two children surveyed by Add Health. However, like Amin et al. (2019) and Ronda et al. (2020), who study similar models using comparable or larger sample sizes, we can see that the family fixed effects approach is too underpowered to be useful in processing our data.

As a motivation of our parametric model, we present a number of nonparametric estimates in Figure 1. As shown in Panel A, the EA PGS imputed for the Add Health data demonstrates statistically significant differences by education, in both means and

Figure 1: Kernel Densities of EA PGS by Education and Health in Young Adulthood



Notes: Calculations are based on the Add Health Data. EA PGS is standardized for the full estimation sample ($N = 3,709$). Epanechnikov Kernel Densities are used with bandwidth 0.7. We test the equality of differences in means (DM) to zero (see p-values p_{DM}) and the equality of distributions using the Kolmogorov-Smirnov (KS) test (see p-values p_{KS}). In Panel A, DM and KS tests compare, respectively: (1) those with education below a high school diploma (including GED) to high school graduates; (2) high school graduates to holders of a post-high school degree below bachelor's that takes at least 1 year to complete; and (3) a post-high school degree below bachelor's to bachelor's or above. In Panels B and C, DM and KS tests compare the EA PGS of those with poor health to those with good health. "Good health" is defined as "very good," or "excellent" health self-reported in young adulthood, "poor health" otherwise. For the purposes of descriptive analysis only (Tables 1, 2 and Figure 1), high SES is defined as having SES factor above its average; low SES otherwise.

distributions, even for close education levels (e.g. high school vs. college degree below bachelor's). This result adds to multiple existing validations of the predictive power of the EA PGS and shows for our specific sample that the EA PGS is highly predictive of the education levels that we use in our model.[2]

Panel B shows that for the high-SES subsample, a higher polygenic score is associated with better health in young adulthood ($p_{DM} = 0.000$ for the difference-in-means test and $p_{KS} = 0.000$ for the Kolmogorov-Smirnov test). Panel C shows that the same association is less strong and less precisely determined for the low-SES subsample, though still statistically significant ($p_{DM} = 0.011$, $p_{KS} = 0.008$). The nonparametric result from Panels B and C of Figure 1 and its confirmation based on our parametric model presented below are in line with the environmental bottleneck hypothesis. The hypothesis suggests that adverse environments can limit the benefits of productive genetic endowments (e.g., Fletcher, 2019).

2 Relating our Contributions to the Existing Literatures

In the introduction we have outlined our contributions and cited several key papers. In this section we elaborate on these contributions by linking them to related literatures.

EA PGS and Health We are not the first to study the associations between EA PGS and outcomes other than education. Belsky et al. (2016) find that EA PGS is associated with a number of measures of socioeconomic success, as well as cognitive and noncogitive skills. Barth et al. (2020) find that EA PGS also explains large amounts of variation in stock market returns and wealth inequality. They offer suggestive evidence that EA PGS may capture aspects of individual information-processing that affect decisions, such as conceptions of probability and risk aversion. The results of this paper are consistent with

[2]Add Health data were excluded as data source for effect size estimation in a genome-wide association study (GWAS). Hence, weights used for EA PGS imputation are independent of Add Health data as well.

these earlier results because socioeconomic success in life and superior skills are known to be complementary with health (e.g., Becker, 2007).

Several studies provide evidence about the relationship between EA PGS and health outcomes. Marioni et al. (2016) use data on European cohorts and show that a child's EA PGS is predictive of parental longevity. They suggest that some genetic mechanism captured in a child's EA PGS might be relevant to determining parental health, since parents and children share some of their genetic traits. Barcellos et al. (2018) study whether genetic endowments moderate the effects of education on health. As part of this research they find a negative association between EA PGS and blood pressure, and between an EA PGS and a weighted average of blood pressure, body size, and adverse lung function.

Further, Huibregtse et al. (2021) report an association between an EA PGS and frailty in old age. Selzam et al. (2019) find than an EA PGS is associated with BMI and self-reported health, though these associations do not survive controlling for dizygotic twin fixed effects.[3] Wedow et al. (2018) report that an EA PGS is associated with smoking. Ding et al. (2019) find an association between an EA PGS and cognitive decline in old age. Finally, Demange et al. (2020) show associations between an EA PGS and a number of health-related outcomes based on multiple datasets, including Add Health.

However, as we explain in the next section, we focus on the interaction between EA PGS and parental SES rather than on simply providing additional evidence for the association between the EA PGS and health-related outcomes.

Interaction Between EA PGS and SES Economic theory suggests that SES may contribute to health differences through interaction effects (e.g., Galama and van Kippersluis, 2018; Grossman, 1972), while a number of empirical papers have shown that SES interacts with a number of polygenic scores. In particular, Fletcher (2019) supports the

[3]However, associations of an EA PGS with IQ and ADHD survive controlling for fixed effects.

environmental bottleneck hypothesis by providing evidence that adverse family environments may reduce the effect of a child's genetic endowment on that child's educational attainment. The environmental bottleneck hypothesis is related to what is known in social science literatures as the Scarr-Rowe hypothesis (Scarr-Salapatek, 1971). The Scarr-Rowe hypothesis states that lower socioeconomic status and greater exposure to social disadvantage during childhood leads to a decrease in the heritability of IQ.

As part of testing the environmental bottleneck hypothesis, we confirm in this paper that a positive relationship between EA PGS and cognitive skills gets stronger with SES. This result is in line with the Scarr-Rowe hypothesis: since a child's EA PGS is genetically related to parental EA PGS, a stronger relationship between a child's EA PGS and a child's cognitive skills is consistent with a greater heritability of IQ.

While the introduction of the Scarr-Rowe hypothesis was a productive insight back in 1970s, the recent environmental bottleneck literature based on newly available polygenic scores represents a step forward. Having the option of using a PGS, which is determined at conception, instead of measures of capabilities in childhood or adulthood, such as IQ, allows modern researchers to better account for environmental confounders.

Further, in line with the environmental bottleneck hypothesis, Bierut et al. (2018) show that advantaged childhood SES provides a major protective effect against a genetic predisposition to smoke, as measured by a smoking PGS. Papageorge and Thom (2020) find that an EA PGS is associated with higher education gains when children have high SES. Ronda et al. (2020) find that hardship in childhood, as measured by low childhood SES, diminishes the effect of EA PGS on education and skill capital. We confirm these results for education for our data as part of our study of the mechanisms behind the effects on EA PGS on health.

Schmitz and Conley (2017) find that reductions in educational attainment as a result of Vietnam-era conscription are larger for individuals with lower EA PGS, providing evidence that a combination of experiencing severe environmental conditions and having an

unfavorable genetic endowment is particularly harmful. Avinun (2019) finds that an EA PGS interacts with a subject's own SES in affecting depression. Our paper has a different focus, as we study the interaction of the PGS 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. In addition to depression, we study six other health outcomes, as well as a number of potential mechanisms.

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, caused by an EA PGS and its interaction with childhood SES. To the best of our knowledge, we are the first to study the interaction between an EA PGS and childhood SES in predicting health outcomes.

Education and Health We also contribute to the important 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) the use of randomized or natural experiments as sources of exogenous variation; (2) the use of family fixed effects or twin fixed effects; (3) the explicit modeling of unobserved heterogeneity.[4]

All these methods have their advantages and disadvantages, as discussed below. Literatures (1) and (2) are both characterized by contradictory results. Literature (3) tends to find effects of education on health; however, there is a concern that certain confounders might be not fully controlled for. This paper diminishes concerns about results from literature (3) by controlling for major genetic confounders measured by PGSs on top of cognitive skills, noncognitive skills, and detailed observable controls and still finds strong associations between education and health. These results are further

[4]This approach is also referred to as "structural," as it assumes a specific structure linking observed and latent variables.

supported by our companion paper (Savelyev and Bolyard, 2022), which shows that this result is robust to controlling for unobserved heterogeneity. Below we briefly explain results of literatures (1–3).

In literature (1), the use of randomized controlled experiments in education is usually limited to early childhood education (Conti et al., 2016). As for natural experiments, they have a well-defined source of variation. However, they only identify the Local Average Treatment Effect (LATE) and may suffer from lack of validity, lack of monotonicity, and weakness of instruments (e.g., Heckman and Vytlacil, 2007). Literature (1) 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).

To conclude, papers in this literature show very different results. 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 (2) 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 external validity of twin-based results could be challenging. Just as for literature (1), there are contradictory results for literature (2). 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 results could be partly related to different

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

Literature (3) explicitly models relationships between observed and unobserved confounders, education, and health-related outcomes. These methods preserve statistical power better than methods (1) and (2). Also, unlike literature (1), literature (3) attempts to estimate the Average Treatment Effect (ATE) rather than Local Average Treatment Effect (LATE). The biggest concern with this literature 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, include major human capabilities: health, cognitive skills, and noncognitive skills (Bijwaard et al., 2015; Conti and Heckman, 2010b; Savelyev and Tan, 2019). Further, Savelyev (2022) and Hong, Savelyev, and Tan (2000) also account for latent unobserved heterogeneity on top of latent human capabilities. The contribution of this paper is that we account for essential genetic confounders for skills and health through PGSs and still find strong conditional associations between education and health-related outcomes.

3 Data

Add Health is a panel dataset, which 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 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 PGS. About 9,000 Add Health study participants took part in genotyping, 5,728 of whom are white. We perform this analysis only for individuals who self-identify as white because of the data limitations described below.

Education and Health Outcomes Because the effect of additional education on health-related outcomes might be nonlinear in total years of education, 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.[5]

All of the health outcomes in young adulthood in this study are measured in Wave IV. 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). In addition, we study three variables that measure substance use: risky drinking of alcohol, smoking cigarettes, and frequent marijuana use. We also have two variables that proxy lifestyles: *obesity* and *no exercise*. Finally, we measure mental health using our *depression* variable. See Table 1 for variable definitions and descriptive statistics.

[5]Given that the youngest participant is 24 by wave IV, we leave the study of more advanced degrees to future research based on future waves. 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).

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

	Full Sample (N=3,709)		Low SES (N=1,388)		High SES (N=2,321)	
	Average	Standard Deviation	Average	Standard Deviation	Average	Standard Deviation
Highest Education Level						
Below high school[a]	0.048	0.214	0.083	0.276	0.028	0.164
High school diploma	0.415	0.493	0.511	0.500	0.357	0.479
College below Bachelor's[b]	0.174	0.380	0.182	0.386	0.170	0.376
Bachelor's or above	0.363	0.481	0.225	0.418	0.445	0.497
Health-Related Outcomes in Young Adulthood						
General health[c]	3.745	0.889	3.641	0.910	3.808	0.870
Excellent or good health[d]	0.625	0.484	0.563	0.496	0.662	0.473
Risky drinking of alcohol[e]	0.209	0.407	0.225	0.418	0.200	0.400
Smoking cigarettes[f]	0.261	0.439	0.296	0.457	0.240	0.427
Marijuana use[g]	0.096	0.294	0.092	0.289	0.098	0.297
Obesity[h]	0.343	0.475	0.389	0.488	0.316	0.465
No exercise[i]	0.130	0.336	0.149	0.356	0.119	0.324
Depression[j]	0.191	0.393	0.189	0.392	0.193	0.394
Potential Mechanisms						
Early health[k]	0.705	0.456	0.654	0.476	0.736	0.441
Cognitive skills[l]	0.000	1.000	-0.144	1.005	0.086	0.987
Conscientiousness[l]	0.000	1.000	-0.011	1.002	0.006	0.999
Extraversion[l]	0.000	1.000	-0.061	1.003	0.036	0.997
Emotional stability[l]	0.000	1.000	-0.058	1.016	0.035	0.989
Education support—self[l]	0.000	1.000	-0.252	1.061	0.151	0.930
Education support—parental[l]	0.000	1.000	-0.166	1.035	0.099	0.965
Household income[m]	8.398	2.354	8.001	2.470	8.633	2.250
Household assets[n]	3.834	1.902	3.611	1.857	3.967	1.916
Job satisfaction[o]	2.215	1.054	2.094	1.038	2.287	1.056
Job physicality[o]	2.073	1.071	2.186	1.074	2.008	1.064

Notes: Calculations based on the Add Health data; estimation sample size, N, 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 cigarettes at least once within the past 30 days. [g]Smoking marijuana once or more per week, on average, during the last year. [h]BMI $\geq$ 30. [i]None of the following: playing sports, exercising outside, walking for exercise, or engaging in other physical activity during the past week. [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.

Potential Mechanisms We also study a number of potential mechanisms in order to suggest possible causal pathways from EA PGS to health-related outcomes. 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 available in wave 4: household income, household assets, job satisfaction, and job physicality.

To measure cognitive skills we use participants' scores on the Add Health Picture Vocabulary Test, recent science grades, and recent math grades.[6] To measure noncognitive skills we use the well-established Big Five Personality taxonomy. Most noncognitive 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 Add Health.[7]

We also study attitudes towards education, both child's and parental, which we call "education support—self," and "education support—parental." Typical questions about parental support ask whether the father would be disappointed if the child would not graduate from high school. The same question is asked about college. The same questions are repeated about 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

[6]The Add Health Picture Vocabulary test is a shortened version of the Peabody Picture Vocabulary Test.

[7]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.

college.

In Table 1 we show descriptive statistics for all health-related outcomes and potential mechanisms that we study. From the table we can see that high-SES subjects report better early health, superior early skills and education support, higher levels of education, more favorable job-related outcomes, and better health and healthier lifestyles in young adulthood. For instance, graduation from college is about twice as likely for high-SES subjects (45 for high-SES vs. 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 endowment with family SES in childhood, we follow the literature on PGS-SES interaction (Bierut et al., 2018; Papageorge and Thom, 2020; Ronda et al., 2020), and construct measures of SES from relevant variables available in the Add Health data. We also show the robustness of our results to a number 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 Danmark, 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 closely-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.[8] This particular set of five measures is characterized by the strong specification statistics of the corresponding factor model, as we discuss in Section 4. 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.

PGS We capture genetic endowments using a PGS. Constructing a PGS from scratch can be a major research project by itself. Because the Add Health dataset offers a large set of already constructed PGSs based on state-of-the-art methodologies (Braudt and Harris, 2018; Okbay et al., 2018), we do not construct any PGSs ourselves. Below we provide a brief general characterization of a PGS, which we expect to be enough for most readers given that this paper focuses on economic implications of models that include PGSs as variables rather than on PGS construction from raw genetic data.

As mentioned earlier, to construct a PGS one needs to focus on SNPs, which are nucleotides at a particular location on the genome that represent variation among humans.[9] Associated with each SNP are alleles, which are specific SNP versions at a given location. Typically, there are only two alleles per SNP and we need to count just one of them, as the other is the only alternative and thus carries no additional information for outcome prediction. The value of a specific allele count can be at most 2, which happens when each of two inherited chromatids has a copy of this allele. The count is 1 if only one chromatid has the allele and 0 if no such allele was inherited.

A PGS is a weighted average of allele counts throughout the genome. The weights are

[8] These measures are summarized and explained in Table A-2 of the Web Appendix.

[9] A variation should be distinguished from mutation, a low-likelihood random mistake in genetic coding. A 1% threshold is typically used to distinguish genetic variation from mutation.

based on the results of genome-wide association studies (GWASs), which are performed based on large independent samples. These weights are adjusted associations between every given allele count and the outcome of interest, such as education. The adjustment is needed to avoid underprediction or overpredication of the PGS due to correlations among alleles in the genome. For technical details behind PGS construction in general, we recommend reviews of genetic literature written for an economic audience (see Benjamin et al. (2011), Beauchamp et al. (2011), and the Web Appendix to Papageorge and Thom (2020)). For technical details behind PGSs used in this paper, see Braudt and Harris (2018) and Okbay et al. (2018).

Because the EA PGS is constructed based on data collected from individuals with European ancestry, we restrict our sample to those who self-report as white.[10] Here we follow the literature confirming that polygenic scores constructed using European-ancestry samples can be both biased and less predictive when applied to populations with different ancestry (Martin et al., 2017).

Our key PGS variable of interest is an EA PGS that was constructed by Lee et al. (2018) based on a sample of 1.1 million individuals of European descent. This EA PGS predicts about 11% of variation in years of education for a validation sample that includes Add Health data (Lee et al., 2018). Panel A of Figure 1 is a further demonstration of the external validity and high predictiveness of the EA PGS for a specific sample that we use.

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

[10] A reliable PGS construction requires giant quantities of genetic data, which are already available for white individuals and takes time to be accumulated for nonwhite individuals.

Table 2: Background Variables

	Full Sample (N=3,709)		Low SES (N=1,388)		High SES (N=2,321)	
	Average	Standard Deviation	Average	Standard Deviation	Average	Standard Deviation
Educational Attainment Polygenic Score						
EA PGS[(a)]	0.000	1.000	-0.051	1.000	0.031	0.999
Background Controls						
Biological sex is male	0.464	0.499	0.463	0.499	0.465	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	0.292	0.455	0.305	0.460
Age 15-16 at wave I	0.394	0.489	0.397	0.489	0.393	0.488
Age 17-19 at wave I	0.222	0.416	0.226	0.419	0.220	0.414
US Region: West	0.144	0.351	0.151	0.358	0.140	0.347
US Region: Midwest	0.332	0.471	0.333	0.471	0.331	0.471
US Region: Northeast	0.156	0.363	0.154	0.361	0.157	0.364
US Region: South	0.368	0.482	0.362	0.481	0.372	0.483
Rural residence	0.361	0.480	0.349	0.477	0.369	0.483
Suburban residence	0.399	0.490	0.405	0.491	0.396	0.489
Urban residence	0.239	0.427	0.246	0.431	0.235	0.424
Low birth weight[(b)]	0.083	0.276	0.082	0.275	0.084	0.277
The only child	0.205	0.403	0.213	0.410	0.200	0.400
First-born	0.320	0.467	0.313	0.464	0.325	0.468
Second-born	0.308	0.462	0.307	0.461	0.308	0.462
Third-born	0.112	0.316	0.108	0.311	0.115	0.319
Number of siblings	2.549	1.950	2.665	2.047	2.480	1.887
Parents married	0.805	0.396	0.738	0.440	0.846	0.361
Cigarettes smoked at home	0.459	0.498	0.470	0.499	0.452	0.498
Meals with parents[(c)]	4.897	2.328	4.788	2.418	4.962	2.271
Hispanic origin	0.060	0.238	0.071	0.257	0.053	0.225
Genetic ancestry PC[(d)]	Yes		Yes		Yes	

Notes: Calculations based on the Add Health data; estimation sample size, N, 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 PGS (Lee et al., 2018). [(b)]Birthweight$\leq$2.5 kg. [(c)]Number of evening meals with parents per week. [(d)]Principal components based on genetic data describing ethnic origin.

physical health endowments[11] and seven mental health PGSs.[12] However, we do not use these additional controls for our contribution 1 so that we keep the estimated effects of EA PGS clearly interpretable and comparable to the literature. We demonstrate correlations between EA PGS and PGSs that describe general health and mental health in the Web Appendix. These correlations range from negligible to modest.[13]

Background Control Variables On top of controlling for EA PGS 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, number of meals with parents per week, and genetic ancestry. See Table 2 for variable definitions and descriptive statistics by SES.

4 Methodology

Model of EA PGS and Health-Related Outcomes For our study of the effect of the EA PGS on health-related outcomes and potential mechanisms behind health formation, we employ a reduced form model that accounts for an interaction between the EA PGS 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 et al., 2018; Papageorge and Thom, 2020). The model is specified as follows:

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

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

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

[13] See Tables A-3 and A-4 of the Web Appendix.

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.

PGS is short for EA PGS; θ^{SES} is a latent continuous factor that represents parental socioeconomic status at the time of subject's childhood. For identification of model (1) involving this latent factor we simultaneously estimate model (1) with a measurement system (3) that we discuss below. 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 PGS 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 PGS^2, we can properly interpret the sign of the interaction effect, β_{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 our equations include a quadratic PGS term, similar to the main model by Papageorge and Thom (2020).

Model of Education and Health Another reduced form model is designed to test whether strong conditional associations γ_{1k} between education levels dummies $\boldsymbol{D}$ and health-related outcomes Y_k survive controlling for a vector of polygenic scores $\boldsymbol{PGS}$ that includes the EA PGS and 16 other types of PGS that measure general and mental health endowments. To better account for possible nonlinearities, we control for a vector of squared PGS scores, $\boldsymbol{PGS}^2$, and interaction terms $\boldsymbol{PGS} \cdot \theta^{SES}$ The model also explores a potential interaction γ_{2k} between SES and education.

The model is the following:

$$Y_k^* = \gamma_{1k}\boldsymbol{D} + \gamma_{2k}\boldsymbol{D}\cdot\theta^{SES} + \gamma_{3k}\boldsymbol{PGS} + \gamma_{4k}\boldsymbol{PGS}^2 + \gamma_{5k}\boldsymbol{PGS}\cdot\theta^{SES} + \gamma_{6k}\theta^{SES} + \gamma_{7k}\boldsymbol{S} + \gamma_{8k}\boldsymbol{X} + \xi_k, \quad (2)$$

where variables $Y_k, k = 1, ..., K_2$, represent health-related outcomes in adulthood and $\boldsymbol{D}$ denotes a vector of three binary variables representing the education levels.[14] A vector of coefficients γ_{2k} is informative of potential interaction between education and SES. We also allow for interactions between PGS and SES to account for possible nonlinearities and add other controls already described in our description of equation (1). The model is conditional on a set of early cognitive and noncognitive skills $\boldsymbol{S}$ and on background controls $\boldsymbol{X}$. Model 2 is estimated simultaneously with the measurement system (3). We compare an unrestricted model (2) with its restricted version, in which we set $\gamma_{3k} = \gamma_{4k} = \gamma_{5k} = 0$ and conclude that the omitted variable bias due to missing major genetic endowments for general health, mental health, and skill is modest in expectation.

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

$$M_j^* = \alpha_{1j}\theta^{SES} + \alpha_{2j}PGS + \alpha_{3j}PGS^2 + \boldsymbol{\alpha}_{4j}\boldsymbol{X} + \epsilon_j, \quad j = 1, ..., J, \quad (3)$$

[14] 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.

where 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; α_{kj}, $k = 1, 2, 3, 4$, are 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 common influences including differences in people's perceptions of own disadvantage.

In Section 5 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 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 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 negative sign to coefficient $\beta_{1,1}$, which effectively assigns the sign of the factor opposite to the sign of the first measure.[15]

[15]Our first measure is "living in unsafe neighborhood," a negative measure of SES, and so reversing the sign 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 our latent SES is positive or negative and then interpret the results accordingly. Standardizing the latent factor is another natural normalization. SES can be viewed as a representation of a person's rank in society. Measures of SES contain information about that rank. For different populations, the same

Finally, we can claim that the sufficient condition for model identification is satisfied for our factor model conditional on all our assumptions and normalizations: we need at least three dedicated measures M_j per factor, so that $J \geq 3$ (e.g., Conti et al., 2014).

Possible Alternatives to the Factor Model It is a natural question whether it would be productive to replace a factor model with another method of aggregation, possibly a simpler one. One common alternative approach to aggregation is to use equally-weighted averages of measures (e.g., Kaestner and Callison, 2011). This procedure has the benefit of calculational simplicity. However, it is based on arbitrarily equal weighting of measures, which implies that all measures are 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.[16] Therefore, at least for 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. Like 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. Plus, binary aggregation occurs with a loss of information. For instance, in the example of binary SES aggregation described 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

absolute value of a measure may imply different SES. For instance, a college education in today's USA and a high school education back in the first part of the 20th century imply a comparable SES because the distribution of education has greatly changed (Savelyev, 2022). Therefore, standardizing a continuous latent SES factor is a reasonable choice. This standardizing helps solve the problem of latent factor scale indeterminacy.

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

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

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, not 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. Plus, we show robustness of our qualitative results to calculationally simple alternatives to the factor model. The robustness checks imply that our results are not driven by assumptions that are specific 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.

5 Results

We first study the relationships between EA PGS and health-related outcomes, including the interaction between EA PGS and parental SES. Then we provide suggestive evidence about the mechanisms behind these relationships. Finally, we study the association between education and health-related outcomes and find that it survives controlling for genetic confounders that predict health and skills. We leave a more structural approach to a companion paper (Savelyev and Bolyard, 2022), one contribution of which is to show

the robustness of the qualitative results of this section to controlling for unobserved heterogeneity.

5.1 EA PGS, Parental SES, and Health-Related Outcomes

We first estimate the relationship between EA PGS and health-related outcomes while allowing for an interaction between EA PGS and parental SES. As explained in Section 4, our model is similar to the one used in recent economic papers on PGS and gene-environment interaction. Figure 2 visualizes estimated relationships by showing marginal conditional associations between EA PGS and health-related outcomes as a function of standardized SES.[17]

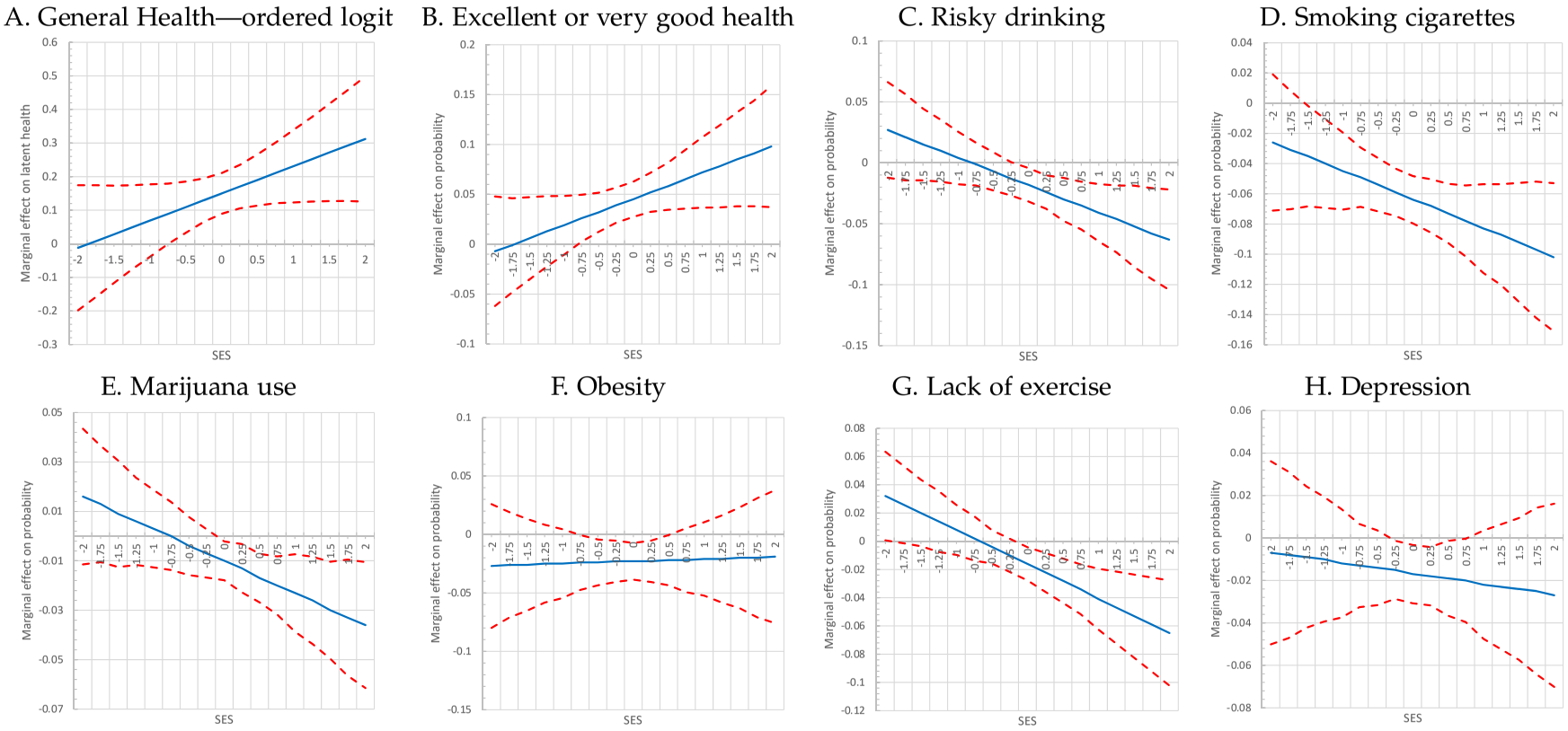
Panels A and B of Figure 2 show results for self-reported general health in adulthood, taking values from 1 (poor) to 5 (excellent). Panel A shows a conditional association between EA PGS and latent health based on the ordered logit model. Panel B reports an association between EA PGS and the probability of having either excellent or very good health based on the same general health measure.[18] We can see that the results based on ordered logit in Panel A are qualitatively similar to the results in Panel B based on a logit model for a related binary outcome. Panels A and B can be viewed as robustness checks to each other, as they use different models and different variable definitions.

While the results in Panel A have the advantage of using all available information about self-reported general health, the similar results in panel B are directly interpretable as the marginal conditional association with probability of a binary health outcome. From Panel B we can see that, for people with an average SES (SES=0), one standard deviation increase in EA PGS leads to about a 4.5 percentage points (PP) higher likelihood of having excellent or very good health, a statistically significant marginal association.

[17] These results are also available in Table A-6 of the Web Appendix, which shows corresponding regression coefficients, standard errors, and effect sizes.

[18] A dummy variable based on this specific threshold shows the highest variation in this population of young adults.

Figure 2: Marginal Conditional Associations Between EA PGS and Health-Related Outcomes in Young Adulthood as a Function of Parental SES, Ordered Logit and Binary Logit Results



Notes: Marginal effects on outcomes are shown as a function of standardized latent SES factor. Results are based on the factor model (1,3). Panels correspond to the following type of outcome model (1): A, ordered logit; B–H, logit. Dashed lines represent the 95% Huber-White confidence intervals calculated using the delta method. Corresponding regression coefficients are documented in Table A-6 of the Web Appendix. See also Table A-5 for parameters of the measurement system (3). Calculations are based on the AddHealth Data.

This association is stronger for those with SES=1 (7.2%), and weaker for SES below the average. For SES around -1 and below the effect of PGS is no longer statistically significant. Given that the probability of having excellent or very good health is 0.625, these effects imply strong effect sizes: at SES=0 the effect size is 7.2% (0.045/0.625), while at SES=1 the effect size is 11.5% (0.072/0.625).

Like Panel B, Panels C–H are also interpretable as marginal conditional associations between EA PGS and the probability of an outcome as a function of SES. Panel C shows such an association with the risky drinking of alcohol. Below SES=0 the effect of PGS quickly becomes statistically insignificant at the 5% level. At SES=0 the effect of PGS is a statistically significant -1.8 PP. At SES=1 the effect becomes -4.1 PP. Given that risky drinking is characteristic for 45.7% of young adults in our sample, the corresponding effect sizes are 3.9% at SES=0 (0.018/0.457) and 9.0% at SES=1 (0.041/0.457).

We observe similar patterns for Panels D, E, and G, for which the effects are statistically insignificant at low levels of SES but strong and statistically significant at its high levels. The effect size for smoking cigarettes in Panel D is 25% at SES=0 (0.064/0.261) and 32% at SES=1 (0.083/0.261). For marijuana use in Panel E effect sizes are 10% at SES=0 (0.010/0.096) and 24% at SES=1 (0.023/0.096). Finally, for lack of physical exercise in Panel G we estimate the effect size to be 12% at SES=0 (0.016/0.13) and 32% at SES=1 (0.041/0.13).

For two outcomes, obesity and depression, we cannot reject the hypothesis of no interaction with SES. However, we find statistically significant effects of EA PGS at SES=0 (see Panels F and H). For obesity, the effect size is at SES=0 is 6.7% (0.023/0.343). For depression, the effect size at SES=0 is 8.9% (0.017/0.089). For these two outcomes we lose statistical significance of the effect of EA PGS for both high and low SES levels. However, this result cannot be explained by an interaction with SES. Instead, the effect is related to decreased precision of our estimation away from $SES = 0$, a feature that is characteristic for all panels in Figure 2, not only Panels F and H.

We can see that all the estimated interaction effects that are statistically significant have the same sign as effects of EA PGS: positive for general health (Panels A and B), and negative for adverse health behaviors (Panels C, D, E and G). The interpretation of this result is that EA PGS is more health-beneficial for those with higher SES status. This result is consistent with the bottleneck hypothesis (Fletcher, 2019): low SES is a good proxy for severely constrained conditions in childhood. Large effect sizes for all seven binary health-related outcomes in Figure 2 imply the economic significance of the results reported in this paper.

Our results complement those found by Bierut et al. (2018), as we find similar interaction effects but for a different type of PGS (we use EA PGS, not smoking PGS) 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 of EA PGS with childhood SES to study education as an outcome.

Robustness to Alternative Measures of SES and to Alternative Methods of Measure Aggregation In Section 3 we survey measures of SES from the literature and introduce comparable types of SES measures that are available in our data. In this section we show that our results are robust to using alternative sets of SES measures and to alternative methods of SES aggregation.

Table 3 documents marginal associations between EA PGS and physical exercise, and the interaction between EA PGS and SES based on 18 alternative definitions of SES.[19] Models 1–6 differ by sets of SES measures shown in Panel E by check marks. Panels A–C represent three alternative methods of SES aggregation: (A) latent factor, (B) an equally-weighted index of residualized measures, and (C) a binary aggregation: no

[19]While results are similar for other health-related outcomes, the example of physical exercise is especially useful because for this outcome both coefficients for EA PGS and the interaction are estimated with high precision, which allows us to reliably study differences across models for both coefficients.

Table 3: Robustness of Our Main Model to Various Combinations of SES Measures: Marginal Effects of EA PGS on the Probability of Doing Physical Exercise in Young Adulthood, Logit Model Results

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
A. Latent SES factor						
EA PGS	-0.016 ***	-0.019 ***	-0.014 **	-0.020 ***	-0.015 **	-0.017 ***
	(0.006)	*(0.006)*	*(0.006)*	*(0.007)*	*(0.006)*	*(0.006)*
EA PGS	-0.024 ***	-0.032 ***	-0.016 *	-0.035 ***	-0.016 **	-0.023 ***
× Factor score SES	*(0.008)*	*(0.009)*	*(0.009)*	*(0.013)*	*(0.008)*	*(0.008)*
B. Standardized equally-weighted index of residualized SES measures						
EA PGS	-0.016 ***	-0.016 ***	-0.015 **	-0.014 **	-0.014 **	-0.016 ***
	(0.006)	*(0.006)*	*(0.006)*	*(0.006)*	*(0.006)*	*(0.006)*
EA PGS	-0.016 ***	-0.017 ***	-0.013 ***	-0.012 **	-0.013 ***	-0.017 ***
× SES Index	*(0.005)*	*(0.005)*	*(0.005)*	*(0.006)*	*(0.005)*	*(0.005)*
C. Binary SES: no single disadvantage from the list (SES=1) vs. at least one (SES=0)						
EA PGS	-0.032 ***	-0.028 ***	-0.026 ***	-0.021 ***	-0.022 ***	-0.029 ***
	(0.009)	*(0.008)*	*(0.008)*	*(0.007)*	*(0.007)*	*(0.008)*
EA PGS	-0.029 ***	-0.028 ***	-0.025 **	-0.026 **	-0.024 **	-0.028 ***
× Binary SES	*(0.011)*	*(0.010)*	*(0.011)*	*(0.012)*	*(0.012)*	*(0.010)*
D. Factor model specification statistics						
RMSEA	0.010	0.000	0.020	0.026	0.023	0.020
Prob(RMSEA $\leq$ .05)	1.000	1.000	0.987	1.000	0.978	1.000
CFI	0.999	1	0.997	0.828	0.997	0.995
TLI	0.969	1	0.859	0.347	0.809	0.868
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 (1,3). Models 2–6 are similar models but with different measures of SES used in the measurement system (3). 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.

single disadvantage (SES=1) vs. at least one disadvantage (SES=0).[20]

Model 1 in Panel A is our most preferred model, and so it corresponds to the main results presented in Figure 2. We choose the most preferred model based on well-established statistics: the Root Mean Square Error of Approximation (RMSEA),[21] the Comparative Fit Index (CFI), and the Tucker-Lewis Index (TLI).[22] 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 factor model 1.[23] 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 0.987 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 $TLI \leq 0.9$. Overall, models 1 and 2 satisfy all three specification criteria, while other models deviate from at least one of established thresholds. Choosing among models 1 and 2, we choose model 1 because it uses an additional key measure of SES

[20]Measures in Panel B are residualized to make the index more comparable to the latent factor in Panel A, which is conditional on $\mathbf{X}$ (see model 3).

[21]RMSEA is an index that is proportional to the square root of the likelihood ratio from the Likelihood Ratio (LR) test, which compares two models: the constrained model, which is the factor model that we test, and the unconstrained model, in which the model can exactly predict each observed covariance. Therefore, a lower number implies a better fit because it is consistent with well-specified factor model restrictions. RMSEA is also inversely proportional to the square root of the number of model restrictions, which is effectively a penalty for using a non-parsimonious model. See, e.g., Kolenikov (2009) and Web Appendix A to Savelyev (2022) for more details.

[22]CFI and TLI are comparative indices based on a comparison of two likelihood ratios: (1) Target (constrained) model relative to unconstrained (saturated) model; (2) Independence model (diagonal variance-covariance matrix) relative to the target (constrained) model. Since measures in a well-specified factor model are highly correlated, we expect our target factor model to have much better fit than the independence model, which makes the likelihood ratio (2) much larger than (1) and drives both indices upwards (away from zero and towards 1). See, e.g., Kolenikov (2009) and Web Appendix A to Savelyev (2022) for more details.

[23]These statistics test measurement system (3) only, and so they are not specific to any particular health outcome.

that is used in the related literature, family income from the lowest quintile (Ronda et al., 2020).

The differences in the estimates in Table 3 are explained not only by different sets of measures across models 1–6 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. As for Panel C, its results differ from Panels A and B because its estimates have different interpretations. The PGS coefficient in Panel C is the marginal effect for the low SES group, not for the average SES level, as in Panels A and B. 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 SES.

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

Sibling Fixed Effects We also explore a corresponding model employing sibling fixed effects, documented in the Web Appendix.[24] We find that even though this method is theoretically desirable, in practice it suffers from low statistical power given our sample size. As expected, the statistical power is greatly diminished compared to our main model for two reasons: (1) a major reduction of identifying variation by restricting it to within-family variation only; and (2) a dramatic decrease in the sample size: only about 200 families who have at least two children surveyed by Add Health contribute to the estimation. In contrast, our main model is estimated based on 3,709 observations.

The impractically low statistical power of family fixed effects when measuring the effect of a PGS is in line with the literature based on datasets of comparable and even

[24]See Table A-7.

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 PGS score and a different outcome. Ronda et al. (2020) also lack sufficient statistical power for the family fixed effect of the EA PGS 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.

5.2 The Mechanisms

To better understand the effects of EA PGS on the health-related outcomes that are described above, we provide suggestive evidence of the mechanisms behind the estimated effects.[25]

We explore potential mechanisms from two time periods: (1) early life; (2) early adulthood. Early life measurements have the advantage of being observed long before health-related outcomes in young adulthood, which helps minimize the likelihood of capturing the reverse causal effect. Early adulthood measurements supplement the early life ones by adding previously unavailable information. However, their simultaneity with health outcomes that we attempt to explain implies that these suggested mechanisms should be interpreted with extra caution.

Health Behaviors The partition between health-related outcomes and the mechanisms is somewhat blurred. For instance, smoking, a health behavior, could be viewed both as a health-related outcome and as a mechanism behind the formation of general health, another health-related outcome that we study. This observation implies that we already have several results on potential mechanisms behind the effect of EA PGS on general health, all documented in Figure 2, which we have discussed in the previous section. Specifically, results for the positive effect of EA PGS on general health are consistent

[25] In a more technical companion paper we explicitly incorporate the mechanisms into the model of health formation (Savelyev and Bolyard, 2022).

with negative effects of EA PGS on risky drinking, smoking cigarettes, marijuana use, obesity, lack of exercise, and depression.

Results in Figure 2 also offer suggestive pathways for the strong interaction between SES and EA PGS that we see for the general health outcome in Panels A and B. This interaction effect is consistent with similar interactions that we observe for risky drinking, smoking cigarettes, marijuana use, and lack of exercise.

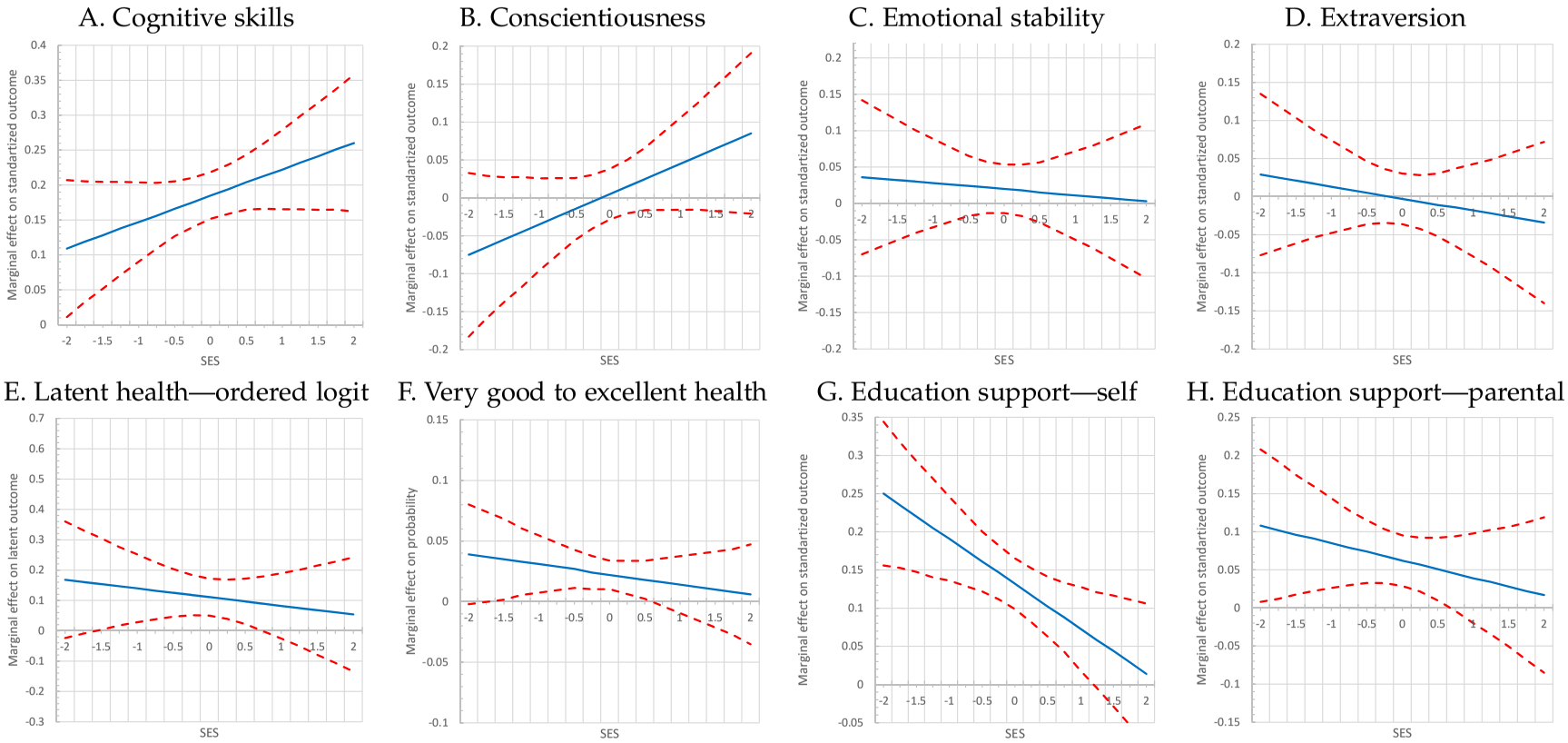
Early Life Mechanisms Figure 3 presents estimates of model (1), with early life potential mechanisms serving as outcomes. As we can see from the figure, EA PGS is positively associated with cognitive skills (Panel A), early general health (Panels E and F), the child's positive attitude towards own education (Panel G), and parental support of the child's education (Panel H). These suggested mechanisms add to explaining the effect of EA PGS on health in young adulthood. However, we fail to find statistical evidence that early noncognitive skills explain the effect of EA PGS (Panels B–D).

Apart from explaining the effect of EA PGS, we try to explain the interaction between EA PGS and SES. Unlike Figure 2, which provides several suggestive channels for this interaction through health behaviors, Figure 3 provides neither statistically strong nor uniform support of the interaction sign that we seek to explain. Namely, for outcomes presented in Figure 3, the interaction between EA PGS and SES is positive (and borderline statistically significant) only for cognitive skills and Conscientiousness (Panels A and B). [26] For other noncognitive skills and health, the interaction effect is not precisely determined.[27]

In contrast to cognitive skills and Conscientiousness, the own motivation for education shows the opposite interaction effect from the one that would explain the interaction effect that we observe for general health in young adulthood in Figure 2. In Panel G of Figure 3, we can see that while EA PGS increases self-motivation for own education at

[26] Corresponding p-values for these interactions are 0.108 and 0.123 correspondingly
[27] p-values for these interactions never fall below 0.5.

Figure 3: Marginal Conditional Associations Between EA PGS 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. Results are based on the factor model (1,3). 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 (3). Calculations are based on the AddHealth Data.

the average level of SES, this effect is not increasing with SES but declining. We interpret this result the following way: 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.

Results for parental support of education in Panel H are similar: the effect of EA PGS at low SES is statistically significant, unlike that at high SES, the same pattern as in Panel G. Our explanation for this result is similar to the result for self-support: high-SES parents tend to support child's education even if the child has low EA PGS, while low-SES parents, who face greater financial constrains and less peer pressure for their children's high education level, may need additional reasons for such support, such as good achievements at school driven by strong genetic endowment. A weaker interaction effect for parental support compared to own support could be explained by indirect observation of child's endowments by parents (e.g. through occasional signals from teachers), while children go to school and observe their own endowments directly and daily.

Overall, the strong positive interaction that we observe for health in young adulthood is partly explained by similar interactions for early cognitive skills and Conscientiousness. However, the effect is contrary to the negative interaction that we observe for college support.

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 PGS, SES, and EA PGS$\times$SES conditional on other controls and find that most measures of drinking alcohol, smoking cigarettes, and overweight in adolescence are predicted by EA PGS. However, the inter-

action with SES is not precisely determined.[28] Secondly, we regress health behaviors in adulthood on EA PGS, SES, and EA PGS×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 PGS 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 PGS 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.

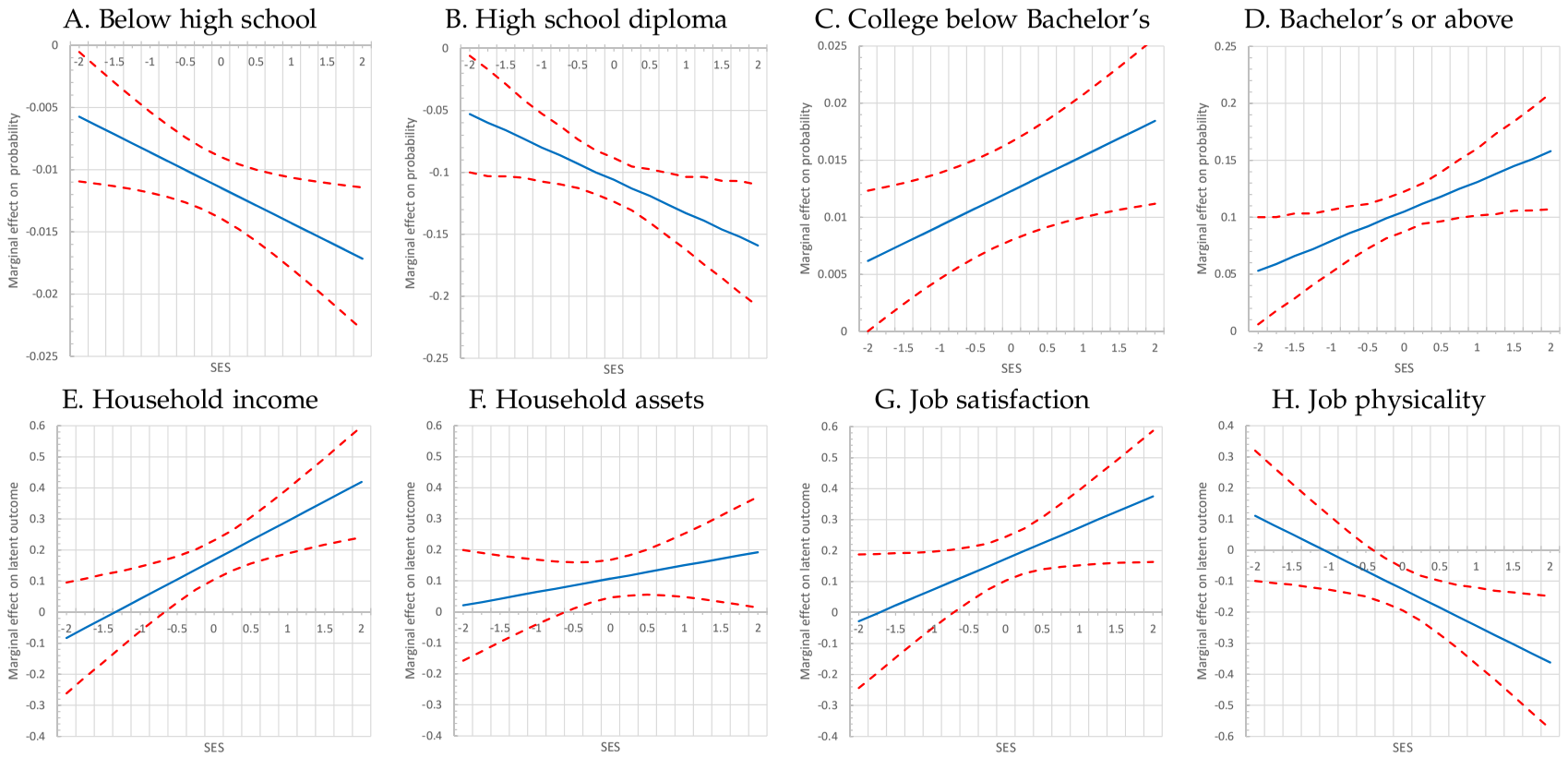
Education The effect of EA PGS on education and the possible interaction between EA PGS and SES have been studied in a number of papers that we survey in Section 2. Even though education itself is not the focus of this paper, we investigate the role of education as part of our exploration of potential mechanisms linking EA PGS, SES, and health.

Panels A–D of Figure 4 show marginal effects of EA PGS on the probabilities of achieving different highest education levels as a function of SES. These graphs are based on the ordered logit model of education (1), estimated simultaneously with the measurement system (3).

As we can see from the figure, the EA PGS 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 PGS stronger. All results are precisely determined and effect sizes are large. At the average SES, effect sizes of EA PGS are the following: 23% decline for below high

[28] See Table A-9 of the Web Appendix.
[29] See Table A-10 of the Web Appendix.

Figure 4: Marginal Conditional Associations Between EA PGS 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. Results are based on the factor model (1,3). 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 (3). Dashed lines represent the 95% Huber-White confidence intervals calculated using the delta method. Calculations are based on the AddHealth Data.

school, 26% decline for high school diploma, 7% increase for college below bachelor, and 29% increase for bachelor's or above.[30]

All these results are expected because EA PGS 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 2 (Fletcher, 2019; Papageorge and Thom, 2020; Ronda et al., 2020). Therefore, results in Panels A—D of Figure 4 serve two purposes: (1) to support the existing literature on EA PGS-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 PGS on health.

Occupation and Wealth Finally, in Panels E–H of Figure 4 we explore the role of outcomes related to occupation and wealth as potential mechanisms. We can see that for medium and high SES levels EA PGS is positively related to household 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 2, as they suggest the mechanisms behind the relationship between EA PGS 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 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 own life, is another potential mechanism of health formation

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

[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).

(Savelyev, 2022). Finally, job physicality is known to be related to worse health levels and quicker heath decline 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).

5.3 Education and Health

The association between education and health can possibly be explained by uncontrolled confounders, or "third variables," that may include factors such as physical and mental ability (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, 2010a). In this section we explore the confounding role of genetic endowments and show that the strong association between education and health survives controlling for most expected genetic confounders.

Table 4, Panel A, shows marginal associations between educational categories and health-related outcomes that are estimated based on model (2). The novelty of these results is that they are conditional on genetic confounders that historically have been viewed as unobservables but recently became available due to major advances in genome sequencing and PGS construction techniques. These confounders include EA PGS, nine types of PGSs related to aspects of physical health, and seven types of mental health PGSs.[32]

All signs of estimated associations are consistent with the health-beneficial role of education. Among 21 individual t-tests in Panel A, only two cannot be rejected at the 5% level.[33] Both tests are for the lowest education level (below high school), which is characterized by a small population (about 5% of the sample) and, therefore, reduced statistical power (see Row 1 of Panel A).

[32]See Tables A-3 and A-4 of the Web Appendix for measures of PGS that describe general and mental health and for correlations among them.

[33]Moreover, among these two, one can be rejected at the 10% level.

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

	Excellent or very good health	Risky drinking	Smoking tobacco	Consuming marijuana	Obesity	Lack of exercise	Depression	Joint test[a]
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
A. Education[b]								
Below High School	-0.258*** *(0.052)*	0.079* *(0.045)*	0.356*** *(0.039)*	0.075*** *(0.019)*	0.015 *(0.051)*	0.067** *(0.027)*	0.077** *(0.036)*	108.9 *[0.000]*
High School Diploma	-0.136*** *(0.023)*	0.117*** *(0.022)*	0.255*** *(0.021)*	0.062*** *(0.011)*	0.075*** *(0.023)*	0.079*** *(0.014)*	0.069*** *(0.018)*	234.0 *[0.000]*
College below Bachelor's	-0.095*** *(0.027)*	0.112*** *(0.026)*	0.245*** *(0.024)*	0.039*** *(0.015)*	0.073*** *(0.027)*	0.053*** *(0.016)*	0.054*** *(0.021)*	146.8 *[0.000]*
College or above	omitted	omitted	omitted	omitted	omitted	omitted	omitted	
Joint test[c]								
Wald statistic	42.7	28.7	147.7	32.9	12.1	30.9	14.7	208.7
p-value	*[0.000]*	*[0.000]*	*[0.000]*	*[0.000]*	*[0.007]*	*[0.000]*	*[0.002]*	*[0.000]*
B. Education × SES[d]								
Joint test								
Wald statistic	2.95	3.90	1.66	7.62	0.63	0.03	0.23	29.0
p-value	*[0.399]*	*[0.273]*	*[0.645]*	*[0.055]*	*[0.889]*	*[0.999]*	*[0.973]*	*[0.113]*

(table continues next page)

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

	Excellent or very good health	Risky drinking	Smoking tobacco	Consuming marijuana	Obesity	Lack of exercise	Depression	Joint test[a]
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
C. PGS[e]								
Joint test								
Wald statistic	43.03	12.51	42.57	11.38	181.91	19.03	35.59	338.2
p-value	*[0.001]*	*[0.768]*	*[0.001]*	*[0.836]*	*[0.000]*	*[0.327]*	*[0.005]*	*[0.000]*
D. PGS × SES[f]								
Joint test								
Wald statistic	18.88	33.53	17.74	20.57	20.50	18.66	13.97	214.1
p-value	*[0.335]*	*[0.010]*	*[0.405]*	*[0.246]*	*[0.249]*	*[0.348]*	*[0.669]*	*[0.000]*

Notes: Marginal associations between education levels and probabilities of corresponding health-related outcomes are reported based on the logit factor model (2,3). Asterisks indicate statistical significance level: ***, 1 % level; **, 5 % level; *, 10 % level. Add Health data are used. Sample size is 3709. Results are conditional on: (1) 17 polygenic scores that measure genetic endowments for education, general health, and mental health. Squares of these scores and their interactions with SES are also controlled for; (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). [e]Testing jointly whether direct effects of all PGSs are zero. [f]Testing jointly whether all direct interaction effects are zero.

Results based on individual tests are supported by joint tests, all of which are overwhelmingly rejected. Those include Wald tests of three types: (1) Joint tests for each of three education levels across all health-related outcomes (see column (8)); (2) Joint tests for each of the seven health outcomes across all education levels (see test statistics in the bottom of Panel A); (3) One joint test across all education levels **and** all outcomes (see the intersection of the row in the bottom of Panel A with column (8)).

Another result of Table 4 is our failure to reject at the 5% significance level the joint test of no interaction between education and childhood SES for any of seven outcomes. A joint test across all seven outcomes cannot be rejected either (see Panel B).[34] Therefore, this interaction is at best weak and cannot explain the strong EA PGS-SES interaction that we observe for health outcomes in Figure 2.

We also jointly test the direct effects of our 17 PGS controls on health outcomes and reject this test for four outcomes out of seven: (1) general health; (2) smoking tobacco; (3) obesity; and (4) depression (see Panel C of Table 4). The overall joint test across all seven outcomes is overwhelmingly rejected ($p = 0.000$). This result is consistent with genetic endowments being associated with health-related outcomes not only through education, but also directly.

Finally, for the direct effect of PGS, we test the interaction between PGS and SES (see Panel D) and can reject it for only one out of seven outcomes, risky drinking ($p = 0.010$). Overall, the weak results of Panel D, combined with similarly weak results in Panel B, are consistent with the at best weak role of PGS-SES and education-SES interactions in explaining health outcomes through the education channel.

We also contribute to understanding the size of the omitted variable bias associated with missing genetic endowments for health and skill. We estimate model 2 with the following restriction: $\gamma_{3k} = \gamma_{4k} = \gamma_{5k} = 0$. This restriction implies the omission of 17 polygenic scores, squares of these scores, and their interactions with SES. The 17 omit-

[34] Because the joint test cannot be rejected for any of the seven outcomes, we show neither individual coefficients nor the t-tests in Panel B to save space.

ted polygenic scores control for endowments that are predictive for education, general health, and mental health.

The omission of these potential confounders is expected to cause an omitted variable bias. We compare results of the restricted model, which is documented in the Web Appendix, with results of our main unrestricted model presented in Table 4. For this comparison we calculate relative differences between education coefficients in these models. We find that the bias for education regression coefficients varies from as small as 2.6% on average (for the risky drinking outcome) to as large as 26% on average (for the depression outcome). Those are average deviations calculated for each outcome across coefficients for different education levels. The average deviation across all seven outcomes is 9.5%.[35]

Therefore, the bias due to missing genetic confounders that predict general health, mental health, and education varies from small (about 3%) to sizable (about 30%) depending on the type of health outcome and is modest in expectation (about 10%). If major potential confounders, which are genetic skills and health, can be expected to explain only 10% of the effect, it is unclear which other potential confounders can be expected to explain the remaining 90% of the estimated effects, especially that we already condition on a large number of background controls, early cognitive and noncognitive skills, and SES.

This result resembles a classic Zvi Grilliches' result: the ability bias in the earnings regression is relatively minor, and so strong estimated associations between wages and education are not driven by unobserved ability (Griliches, 1977). 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

[35]Calculation of the difference is done for all estimates that are statistically significant, which are 19 estimates out of 21. See Table A-13 of the Web Appendix.

downward, not upward, as confirmed by future research. Our contribution is separate, however: we obtain our result for the health production function rather than the earnings function and we use previously unavailable data on genetic endowments for this aim.

6 Conclusions

We find that the EA PGS exhibits strong and health-beneficial conditional associations with a variety of life outcomes in young adulthood. Moreover, these associations substantially interact with SES: individuals who grew up in disadvantaged households do not experience health benefits of the EA PGS unlike their more advantaged peers. We also contribute to our understanding of the potential mechanisms through which the EA PGS 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.

Genetic variants are unlikely to become policy variables in the foreseeable future (if ever) due to ethical, political, and practical considerations. In contrast, major disadvantages that are reflected in low SES can be dealt with through a number of politically feasible anti-poverty policies. Benefits and costs of various anti-poverty policies are well documented in the literature. However, 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 either confirmed or demonstrated a number of

other positive complementing effects of SES on skills, education, earnings, wealth, and job satisfaction. Our second 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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[FOR ONLINE PUBLICATION]

Web Appendix to "Understanding the Educational Attainment Polygenic Score and Its Interactions with SES in Determining Health in Young Adulthood"*

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A Supplementary 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 to graduate 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. Scores for math and science are imputed from letter grades from wave I. Education support measures are reported by subjects in wave I.

Table A-2: Main 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

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.)

Table A-3: Correlations Among EA PGS and PGSs that Measure Physical Health Endowments

	EA PGS	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-4: Correlations Among EA PGS and PGSs that Measure Mental Health Endowments

	EA PGS	Depres-sion	Neuro-ticism	Attention -deficit disorder	Bipolar disorder	Major depressive disorder	Schizo-phrenia
Depression	0.106 *(0.000)*	1					
Neuroticism	0.154 *(0.000)*	0.459 *(0.000)*	1				
Attention-deficit disorder	-0.221 *(0.000)*	-0.144 *(0.000)*	-0.063 *(0.000)*	1			
Bipolar disorder	-0.004 *(0.823)*	-0.104 *(0.000)*	-0.068 *(0.000)*	-0.054 *(0.000)*	1		
Major depressive disorder	-0.144 *(0.000)*	-0.323 *(0.000)*	-0.234 *(0.000)*	0.184 *(0.000)*	0.169 *(0.000)*	1	
Schizophrenia	-0.031 *(0.061)*	-0.112 *(0.000)*	-0.074 *(0.000)*	0.028 *(0.069)*	0.275 *(0.000)*	0.240 *(0.000)*	1
Mental health cross disorder	-0.013 *(0.414)*	-0.155 *(0.000)*	-0.145 *(0.000)*	-0.065 *(0.000)*	0.623 *(0.000)*	0.311 *(0.000)*	0.414 *(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-5: Factor Loadings of the Measurement System

	Factor loading	Standard error	*p*-value
Measures of SES			
Living in an unsafe neighbourhood	0.533	0.116	0.000
Having difficulties with paying bills	0.873	0.100	0.000
Household on government assistance	1.646	0.174	0.000
At least one parent has a college education	-0.970	0.097	0.000
Household income from the lowest quintile	1.825	0.209	0.000
Wald test of the equality of factor loadings (sign of 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 Table A-2 for variable definitions.

Table A-6: Conditional Reduced-Form Associations Between EA PGS, Gene-SES Interaction, and Health-Related Outcomes

	General health	Excellent or very good health	Risky drinking	Smoking cigarettes	Marijuana use	Obese	Lack of exercise	Depression
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
EA PGS	0.149 ***	0.045 ***	-0.012	-0.064 ***	-0.010 **	-0.023 ***	-0.016 ***	-0.016 **
	(0.032)	*(0.009)*	*(0.008)*	*(0.008)*	*(0.004)*	*(0.008)*	*(0.006)*	*(0.007)*
Effect size[(a)]	-	0.072	-0.057	-0.245	-0.104	-0.067	-0.123	-0.084
EA PGS	0.081 *	0.026 *	-0.036 ***	-0.019 *	-0.013 **	0.002	-0.024 ***	-0.005
× SES	*(0.045)*	*(0.014)*	*(0.012)*	*(0.011)*	*(0.006)*	*(0.013)*	*(0.008)*	*(0.011)*
Effect size[(a)]	-	0.042	-0.172	-0.073	-0.135	0.006	-0.185	-0.026
SES	0.253 ***	0.085 ***	-0.016	-0.049 ***	0.003	-0.068 ***	-0.022 ***	0.000
	(0.046)	*(0.013)*	0.012	0.011	0.007	0.013	0.008	0.010
$(\text{EA PGS})^2$	0.013	0.004	-0.012 **	-0.001	-0.002	-0.007	-0.009 **	-0.015 ***
	(0.022)	*(0.006)*	0.006	0.005	0.003	0.006	0.004	0.005
Sample Size	3709	3709	3694	3699	3705	3664	3708	3709

Notes: 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 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: An Alternative Sibling Fixed Effect Estimation of the Effect of EA PGS and EA PGS-SES Interaction on Health-Related Outcomes

	General health	Risky Drinking	Smoking tobacco	Marijuana use	Obese	No exercise	Depres-sion
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
EA PGS	-0.055	0.002	-0.050	-0.003	-0.023	-0.019	0.021
	0.128	0.053	0.047	0.043	0.060	0.061	0.053
EA PGS × SES	0.078	0.021	0.028	0.041	-0.067	-0.030	-0.018
	0.092	0.044	0.045	0.034	0.042	0.050	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-8: Conditional Reduced-Form Associations Between EA PGS, Gene-SES Interaction, and Potential Mechanisms Behind the Effects of EA PGS

	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 PGS	0.185 ***	0.006	0.020	-0.002	0.109 ***	0.026 ***	0.063 ***	0.133 ***
	(0.017)	*(0.017)*	*(0.017)*	*(0.017)*	*(0.032)*	*(0.008)*	*(0.017)*	*(0.017)*
EA PGS	0.038 *	0.040	-0.008	-0.016	-0.029	-0.003	-0.023	-0.059 ***
× SES	*(0.023)*	*(0.026)*	*(0.026)*	*(0.026)*	*(0.046)*	*(0.012)*	*(0.024)*	*(0.022)*
SES	0.205 ***	0.041	0.079 ***	0.083 ***	0.167 ***	0.060 ***	0.218 ***	0.340 ***
	0.026	0.026	0.026	0.026	*(0.045)*	*(0.012)*	0.026	0.025
$(\text{EA PGS})^2$	-0.007	-0.006	-0.003	-0.010	0.031	0.007	-0.010	-0.014
	0.012	0.012	0.012	0.012	*(0.022)*	*(0.006)*	0.012	0.012
Sample Size	3694	3699	3664	3705	3709	3709	3708	3709

Notes: 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 uses 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-9: Conditional Reduced Form Associations Between EA PGS, 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 PGS	-0.022 **	-0.003	0.001	-0.035 ***	-0.027 ***	-0.022 ***	-0.019 **	-0.001
	(0.009)	*(0.006)*	*(0.002)*	*(0.009)*	*(0.007)*	*(0.005)*	*(0.008)*	*(0.003)*
EA PGS	0.020	0.002	-0.002	0.000	0.006	-0.009	0.002	0.000
× SES	*(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: 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-10: Conditional Reduced-Form Associations Between EA PGS, 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 PGS	-0.012	-0.011	-0.064 ***	-0.054 ***	-0.023 ***	-0.017 *
	(0.008)	*(0.008)*	*(0.008)*	*(0.008)*	*(0.008)*	*(0.009)*
EA PGS	-0.036 ***	-0.041 ***	-0.019 *	-0.017	0.002	0.000
× SES	*(0.012)*	*(0.013)*	*(0.011)*	*(0.011)*	*(0.013)*	*(0.014)*
Corrsponding	-	0.116 ***	-	0.149 ***	-	0.398 ***
early beahvior (A)	-	*(0.018)*	-	*(0.019)*	-	*(0.018)*
Corrsponding	-	0.042 **	-	0.039	-	0.304 ***
early beahvior (B)	-	*(0.021)*	-	*(0.028)*	-	*(0.055)*
Corrsponding	-	0.042	-	0.161 ***	-	-
early beahvior (C)	-	*(0.045)*	-	*(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-9 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. 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-11: Marginal Conditional Associations Between EA PGS 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 PGS	-0.011 ***	-0.106 ***	0.012 ***	0.105 ***	0.475 ***
	(0.001)	*(0.009)*	*(0.002)*	*(0.009)*	*(0.038)*
Effect size[(a)]	-0.229	-0.255	0.069	0.289	-
EA PGS	-0.003 ***	-0.026 **	0.003 ***	0.026 **	0.119 **
$\times$ SES	*(0.001)*	*(0.012)*	*(0.001)*	*(0.012)*	*(0.052)*
SES	-0.020 ***	-0.188 ***	0.022 ***	0.187 ***	0.846 ***
	(0.002)	*(0.015)*	*(0.004)*	*(0.014)*	*(0.064)*
$(\text{EA PGS})^2$	0.000	-0.004	0.000	0.004	0.019
	(0.001)	*(0.006)*	*(0.001)*	*(0.006)*	*(0.027)*

Notes: Coefficients in panels (1–4) are marginal conditional associations between a right-hand-side variable, such as EA PGS, 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-12: Conditional Reduced-Form Associations Between EA PGS, 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 PGS	0.170 ***	0.108 ***	0.174 ***	-0.124 ***
	(0.032)	*(0.031)*	*(0.036)*	*(0.035)*
EA PGS	0.125 ***	0.043	0.101 **	-0.118 **
× SES	*(0.043)*	*(0.043)*	*(0.051)*	*(0.051)*
SES	0.435 ***	0.247 ***	0.228 ***	-0.244 ***
	(0.046)	0.044	0.050	0.051
$(\text{EA PGS})^2$	-0.031	-0.029	0.000	-0.036
	(0.022)	0.021	0.025	0.025
Sample Size	3709	3709	3709	3709

Notes: 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 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-13: Marginal Associations Between Education Categories and Health-Related Outcomes in Young Adulthood, the Case with Genetic Controls Excluded

	Excellent or very good health (1)	Risky drinking (2)	Smoking tobacco (3)	Marijuana use (4)	Obesity (5)	Lack of exercise (6)	Depression (7)	Average deviation[a] (8)
A. At average SES[b]								
Below High School	-0.242***	0.057	0.367***	0.082***	0.043	0.072**	0.087**	
	(0.048)	*(0.044)*	*(0.036)*	*(0.021)*	*(0.046)*	*(0.029)*	*(0.035)*	
% change[c]	*6.2%*	-	*3.1%*	*9.3%*	-	*7.5%*	*13.0%*	*7.8%*
High School Diploma	-0.142***	0.112***	0.265***	0.070***	0.094***	0.087***	0.079***	
	(0.022)	*(0.021)*	*(0.019)*	*(0.012)*	*(0.021)*	*(0.014)*	*(0.017)*	
% change[c]	*4.4%*	*4.3%*	*3.9%*	*12.9%*	*25.3%*	*10.1%*	*14.5%*	*10.8%*
College below Bachelor's	-0.103***	0.111***	0.240***	0.046***	0.093***	0.058***	0.056***	
	(0.026)	*(0.024)*	*(0.022)*	*(0.016)*	*(0.025)*	*(0.017)*	*(0.020)*	
% change[c]	*8.4%*	*0.9%*	*2.0%*	*17.9%*	*27.4%*	*9.4%*	*3.7%*	*10.0%*
College or above	omitted	omitted	omitted	omitted	omitted	omitted	omitted	
Av. % change(c)	*6.3%*	*2.6%*	*3.0%*	*13.4%*	*26.4%*	*9.0%*	*10.4%*	*9.5%*

Notes: Results are based on a modification of model (2) with the following restriction: $\gamma_{3k} = \gamma_{4k} = \gamma_{5k} = 0$. This restriction implies the omission of 17 polygenic scores $\boldsymbol{PGS}$, squares of these scores, $\boldsymbol{PGS}^2$, and their interactions with SES, $\boldsymbol{PGS} \cdot SES$. Asterisks indicate statistical significance level: ***, 1 % level; **, 5 % level; *, 10 % level. Calculations are based on the Add Health data. Sample size is 3709. Numbers in Panel (1) are in the same scale as ordered logit coefficients. Numbers in Panels (2–7) are estimated marginal effects on the probabilities of corresponding outcomes. [a]Average across the corresponding row. [b]Effects of education are calculated at the average level of SES ($SES = 0$). [c]Absolute values of deviations from main results in Table 4 of the main paper.