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Abstract

We investigate an Educational Attainment Polygenic Score (EA PGS), an index that predicts years of formal education based on genetic data. In our analysis of the National Longitudinal Study of Adolescent to Adult Health we find that the EA PGS affects a number of health-related outcomes. Moreover, the EA PGS interacts with parental socioeconomic status (SES) in childhood: for a number of health outcomes we observe that the effect of the EA PGS is more beneficial for high-SES subjects. We decompose the total effects of the EA PGS into the indirect effect (through education) and the direct effect. We also decompose both the direct and the total effect with respect to potential mechanisms. The mechanisms that partially explain the effects of EA PGS include early skills, early health, education support in the family, and education. As a result of our discovery of a strong direct effect we cast our doubts on the validity of the EA PGS used as an instrumental variable for education affecting health, a case of an increasingly utilized technique called Mendelian Randomization. Finally, after controlling for the EA PGS, genetic health endowments, and unobserved heterogeneity in addition to more traditional controls, we still find that education is associated with better health outcomes, which adds evidence to the ongoing debate about the causal link between education and health.

Key words: Educational Attainment Polygenic Score, socioeconomic status, mechanisms, Mendelian Randomization, gene-environment interaction, health, health-related outcomes, Add Health

JEL codes: I12, I14, I24, J24

1 Introduction

Researchers are quickly gaining access to a wealth of genetic data, creating opportunities for new research. However, predictions based on single units of genetic information called SNPs[1] lead to low statistical power and issues with replicability, as many life outcomes are affected by multiple SNPs. Polygenic scores (PGSs) provide a well-established solution to this problem. A PGS is an optimally-weighted aggregate of multiple SNPs that predict a specific life outcome. PGSs demonstrate stronger predictive power and more robust results across populations than single SNPs (e.g., Benjamin et al., 2011). Yet we would still benefit from superior knowledge about the predictive power of PGSs outside the life outcomes they are designed to predict, the interaction of PGSs with the environment, the mechanisms through which they affect outcomes, and the consequences of adding PGSs as additional controls to traditional models.

An Educational Attainment PGS (EA PGS), which measures an individual's genetic predisposition for the total number of years of formal education, has particularly important socioeconomic implications. This paper is focused on the EA PGS and offers five main contributions.

First, we confirm an already known association between EA PGS and health-related outcomes while adding superior controls, including controls for potential genetic confounders and unobserved heterogeneity.

Second, we demonstrate a novel result that the EA PGS exhibits differential effects on a number of health-related outcomes depending on parental socioeconomic status (SES) in childhood: we observe that the effect of the EA PGS is more beneficial for high-SES subjects.[2] The interaction effect is so strong that a beneficial effect of an EA PGS is fully canceled when SES is low enough. Therefore, we add new results to the growing

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

[2] As in the related literature, our SES factor is measured by variables that imply major socioeconomic hardship in childhood to examine the effect of an SES "bottleneck."

literature on environmental bottlenecks, though we do not confirm the result from the literature that the EA PGS interacts with SES in predicting education in our sample.

Third, our decompositions of the effects of the EA PGS on health-related outcomes reveal the mechanisms through which the EA PGS works, which allows us to better understand the EA PGS itself. We perform a novel decomposition of this effect into indirect effects (through education) and direct effects (not through education) and find that for a number of outcomes both effects are strong and statistically significant.[3] We further decompose both the total and the direct effect with respect to potential mechanisms. The total effect is explained not only by early skills, as is known from the literature, but also through early health, education support in the household, and through formal education itself.[4] We further decompose a specific part of the total effect called the direct effect. We find that the direct effect is partially explained by early skills and early health. This novel result not only enhances our understanding of the mechanisms, but also has an important methodological implication that we discuss in the next paragraph.

Fourth, our discovery of a strong direct effect, which is, moreover, explained by the mechanisms that we can theoretically expect, casts doubts on the validity assumption behind using an EA PGS as an instrument for education affecting health, a case of an increasingly popular Mendelian Randomization (MR) approach. Even though the validity assumption can generally be neither proven or disproven, various theoretical and empirical considerations can be brought to either support this assumption or question it. Our result holds under the assumptions of our model for a variety of health outcomes after controlling for a large set of family background variables, cognitive skills, noncognitive skills, genetic ancestry, general and mental health PGSs, and unobserved heterogeneity.

[3]Even the indirect effect of the EA PGS on health through education is not guaranteed ex ante given that the causal status of the relationship between education and health-related outcomes is controversial, as we discuss in Section 2.

[4]This particular contribution of education is conditional on earlier mechanisms that affect education, which are early health, early skills, and education support in the household.

Finally, we contribute to the debate on the causal effect of education on health by showing that the conditional association between education and health-related outcomes survives controlling for important genetic confounders.

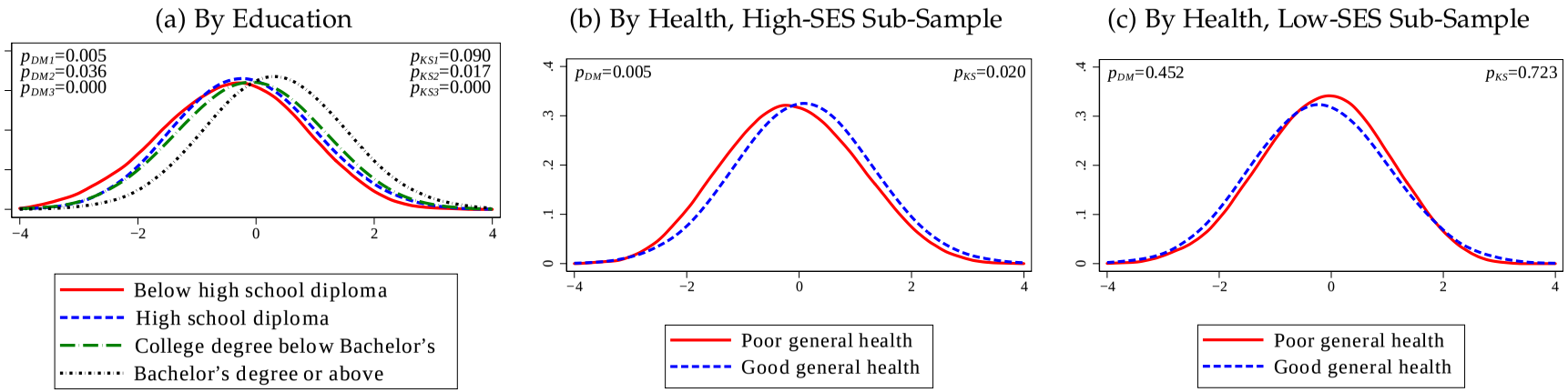
We use data from The National Longitudinal Study of Adolescent to Adult Health (Add Health), which follows a cohort of individuals who were in either middle or high school in 1996, and who are now young adults. We study a variety of health outcomes related to general and mental health, substance use, exercise, and body weight. As we discuss in more detail in the data section, we only study white individuals due to data limitations and the well-established result that imputing a PGSs outside the ethnicity for which it was constructed can lead to a bias and a loss of statistical power (Martin et al., 2017).

As a descriptive analysis that motivates our main model, we present a number of nonparametric estimates in Figure 1. The EA PGS that we use was constructed without the use of Add Health data.[5] As shown in Panel (a), the EA PGS imputed for the Add Health data demonstrates statistically significant differences, in both means and 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 that the EA PGS is highly predictive of the education levels that we use in our model.

Panel (b) shows that for the high-SES subsample, a higher polygenic score is associated with better health in young adulthood ($p_{DM} = 0.005$ for the difference-in-means test and $p_{KS} = 0.020$ for the Kolmogorov-Smirnov test). Panel (c) shows that the same association is not statistically significant for the low-SES subsample ($p_{DM} = 0.452$, $p_{KS} = 0.723$). Later, once we control for both observed and unobserved heterogeneity in our main semiparametic model, this unconditional and nonparametric result is preserved.

[5]EA PGS construction involves SNP selection and optimal weights calculation.

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. "Poor health" is defined as responding "poor" or "fair" when asked to evaluate ones own health. "Good health" is defined as "good," "very good," or "excellent" reported health. For the purposes of descriptive analysis only (Tables 1, 2 and Figure 1), low SES is defined as having an adverse SES factor score above its average; high SES otherwise.

Drawing on other models of gene-environment interaction in the literature (e.g., Bierut et al., 2018), we continue our empirical analysis by estimating a reduced form model with an interaction term between the EA PGS and SES. In this model we find associations between the EA PGS and health-related outcomes. We also find evidence of interactions between the EA PGS and childhood SES, which suggests that a high EA PGS may predict better health only for those with high childhood SES. Thus, a reduced-form parametric model conditional on a large set of controls shows the same qualitative results as unconditional nonparametric estimates from Panels (b) and (c) of Figure 1. This result is in line with the environmental bottleneck hypothesis, which suggests that adverse environments can limit the benefits of productive genetic endowment (e.g., Fletcher, 2019).

We take this framework a step further in our main model by accounting for unobserved heterogeneity and for the mechanisms behind these effects. We estimate a set of recursive equations, including potential mechanisms. The results of the main model confirm the bottleneck role of childhood SES for health development. This model also allows us to study the decompositions already discussed above: an indirect effect through education, the direct effect, and the mechanisms behind them.

The EA PGS is determined at conception, which greatly reduces the amount of possible confounders compared to traditional measures of endowment, such as IQ. However, it is a well-known concern that the gene-environment interaction effect is confounded due to unobserved heterogeneity, in particular the correlation between the genetic endowment of biological parents and the child's EA PGS. For instance, some aspects of intelligence, which is captured in the EA PGS, are heritable. Belsky et al. (2016) find that high-SES families tend to have children with higher EA PGS. However, Lee et al. (2018) find that the EA PGS still predicts educational attainment among siblings after controlling for family fixed effects. Papageorge and Thom (2020) use auxiliary models to argue that such endogeneity would not alter the sign of the SES-PGS interaction that

they estimate. In this paper, we address the endogeneity concern directly by explicitly modelling unobserved heterogeneity that affects child's EA PGS, parental SES, and child's life outcomes.

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.

Our model also contributes 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 along with other literatures that are relevant to our contributions. One possible confounding factor in education-health studies is genetic endowment, which may be strongly predictive of both education and health (e.g., Boardman et al., 2015). We find, however, that education still exhibits large and statistically significant associations with a variety of health outcomes when we control for EA PGS. This result is preserved when we add controls for general health PGS, mental health PGS, and unobserved heterogeneity.

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 effects of an EA PGS on 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 an EA PGS also explains large amounts of variation in stock market returns and wealth inequality. They offer suggestive evidence that an 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 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 a relationship between an 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. However, this evidence is indirect and therefore inconclusive. A possible alternative explanation of the observed effect is that better education of children leads to better care of their elderly parents through children's superior skills and wealth.

Barcellos et al. (2018) focus on the role education plays in interacting with a genetic predisposition for poor health, including genetic endowments measured by an EA PGS. They find a negative association between an EA PGS and blood pressure, and between an EA PGS and a weighted average of blood pressure, body size, and adverse lung function. 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 do not survive controlling for dizygotic twin fixed effects.[6] 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.

Overall, there is substantial evidence in the literature on the association between EA PGS and health-related outcomes. Therefore, our contribution to this particular question is modest: we use a model that broadens the set of controls, including our

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

modeling of unobserved heterogeneity, and confirm that a relationship between EA PGS and health outcomes survives superior controlling for possible confounders. Our main contributions lie elsewhere and include the study of an interaction between EA PGS and SES affecting health, a number of decomposition results, our criticism of MR, and the additional evidence in favor of the effect of education on health. We describe these contributions below in the context of existing literature.

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 *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. Given that EA PGS is a strong predictor of IQ, this result is closely related to what is known in social science literatures as the Scarr-Rowe effect, which was discovered long before a PGS became available: lower socioeconomic status and greater exposure to social disadvantage during childhood leads to a decrease in the heritability of IQ (Scarr-Salapatek, 1971). However, having the option of using a PGS instead of IQ allows modern researchers to exclude many possible confounders that might be behind the original Scarr-Rowe effect estimation.

Similarly, 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. 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. 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.

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.[7] In addition to depression, we study six other health outcomes as well as education.

Our study contributes to this literature, as we use different data to study different outcomes caused by an EA PGS and its interaction with childhood SES, namely outcomes that are related to health and health behaviors. 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.

One result of our decomposition of the effect of the EA PGS on health is especially relevant to this subsection: for our sample of young adults in the US we do not confirm the result from the literature discussed above that disadvantaged SES diminishes the effect of an EA PGS on education, although we do find such interaction effects for health-related outcomes. Apart from differences in populations, one possible contributing reason for this difference is that we have more controls than other studies, including additional background controls, PGS controls, and latent controls for unobserved heterogeneity. We support this possible explanation in the results section.

The Mechanisms Linking EA PGS to Health Outcomes One of the advantages of our main model is that it allows us to investigate the mechanisms behind the effects of the EA PGS on health and behind the interaction with SES. Here we outline the literature on the mechanisms through which the EA PGS affects health and other life outcomes.

[7]However, we address individual SES mediation as well, as individual education can be viewed as a key measure of individual SES.

There is substantial evidence that genes predict cognitive and noncognitive skills. In particular, Lee et al. (2018) find that the EA PGS explains 7–10% of variation in cognitive performance. Belsky et al. (2016) find strong associations between EA PGS and cognitive and noncognitive skills. At the same time, there is growing evidence that cognitive and noncognitive skills predict health (e.g., Bijwaard et al., 2015; Conti et al., 2010; Cunha and Heckman, 2007; Savelyev and Tan, 2019). Therefore, skills are among the expected mechanisms linking EA PGS to health.

Other potential mechanisms that we model include early health, education support in the household, and formal education.[8] Education and health are known to be predicted by genes: this is why we are able to use established polygenic scores for education and health in this paper. At the same time, as we discuss below, education is a possible determinant of health. As for health, its earlier value directly affects its future value in a dynamic equation for health stock; we can also expect an indirect effect of earlier health on future health-related outcomes, as early health may affect educational and health investments (Galama and van Kippersluis, 2018; Grossman, 1972).

Finally, we suggest and empirically confirm that the EA PGS affects health outcomes through a factor that we call education support in the household. Here we hypothesize, following the literature on household resource allocation, that a child's genetic endowment affects parental support of that child's education (e.g., Almond and Mazumder, 2013). In addition, in line with a criticism by Savelyev et al. (2020) of a systematic bias in this literature, we take into account that it is not only parental support that matters, but also a child's own motivation, which is affected by the child's genetic endowments. To keep our model parsimonious and to account for possible measurement error in multi-

[8]The categorical highest level of education, which we use as an education measure, is realized later in life than early skills, early health, and early education support in the household. Therefore, we model education as a function of these earlier mechanisms. We also model how education contributes to our decomposition conditional on other mechanisms, thus estimating a direct effect of education on health conditional on early skills, health, and education support.

ple self-reported measures of education support by father, mother, and child, we use a factor model that aggregates these measures into one education support factor.

Our main contribution to the EA PGS mechanisms literature is our decomposition of the total effect of EA PGS on health-related outcomes into direct and indirect parts and our further decomposition of the direct part. This focus is different from that of Demange et al. (2020), who concentrate on comparing cognitive genetic predictors of education with noncognitive ones.

In addition to finding an indirect effect through education, which is an expected result as long as we expect education to affect health, we find a strong direct effect for a number of health-related outcomes. To better understand the mechanisms behind the direct effect, we further decompose the direct part and its interaction with SES with respect to early skills, early health, education support in the family, and residual channels. Our decomposition of the direct part shows a strong contribution of early health and skills, a result that not only allows to us to better understand the mechanisms behind an EA PGS, but also contributes to the criticism of the MR technique, as discussed in the next subsection. In contrast, for the PGS-SES interaction part of the direct effect, we do not find any statistically significant contributions of early skills or health, which suggests that the SES itself might affect the outcome directly in this channel. To the best of our knowledge, our decompositions of the direct effect and the interaction effect are novel.

Our study is related to that by Belsky et al. (2016), but with four important differences: (1) we study health, not socioeconomic success as an outcome; (2) we decompose not only the total effect but also the direct effect with respect to the mechanisms; (3) in addition to estimating contributions of cognitive and noncognitive skills, we estimate contributions of early health and education support in the family[9]; (4) we control for unobserved heterogeneity.

[9] As a related note, Belsky et al. (2016) report an association between an EA PGS and subjects' own educational aspirations. However, authors' mediation model does not include any education support variables.

Mendelian Randomization Our results also contribute to better understanding MR, a technique that uses genetic variables as instrumental variables (IVs) for the traits they predict. Studies in many fields, including economics, have already made use of MR. The MR case relevant to this paper is the use of EA PGS as an IV for estimating the effect of education on health outcomes (e.g., Böckerman et al., 2017; Davies et al., 2019; Tillmann et al., 2017).

The MR method is being criticized for its possible violation of the validity assumption: genes may predict outcomes not only indirectly through the variable being instrumented, but also directly. This is particularly likely to happen when a PGS rather than a single SNP is used as an IV due to the polygenic nature of a PGS (DiPrete et al., 2018). A PGS is constructed in order to be highly correlated with an outcome of interest, but the PGS construction process is not designed to satisfy the exclusion restriction that is needed for the validity of an IV.

According to von Hinke et al. (2016), MR is very controversial within economics, in part because we still have a very limited understanding about the function of specific genes: studies of specific genes are often underpowered. The authors argue that genetic variation need to be used as IVs with care.

Munafò et al. (2019) are optimistic about the use of MR in general, but point to its limitations. In particular, they caution that the direct effect (horizontal pleiotropy) that violates the validity assumption is especially likely to occur for complex outcomes (phenotypes), such as educational attainment, where genetic variants are likely to operate via a range of biological, behavioral, and social pathways. Kippersluis and Rietveld (2018) also cast doubt on the validity assumption in MR (pleiotropy), including the case where EA PGS is used as an instrument for health outcomes.

This paper contributes to the criticism of MR by establishing that the EA PGS affects health outcomes in adulthood not only through education, but directly through early life skills and health among other channels. This finding is consistent with certain SNPs be-

ing selected in the process of PGS construction because they predict education through early skills and health. However, because health is a stock, early health directly affects later health in a dynamic model (Grossman, 1972). Early skills are also expected to contribute to better health decisions and better resource allocation conditional on education (e.g., Savelyev, 2020; Savelyev and Tan, 2019). Our mediation analysis confirms these theoretical expectations.

Our results are consistent with Belsky et al. (2016), who perform a mediation analysis of the association between an EA PGS and measures of socioeconomic success in life based on the Dunedin study from New Zealand. The authors find that the EA PGS is associated with cognitive and noncognitive abilities in early life and that socioeconomic success in young adulthood is associated with EA PGS even conditional on education. However, Belsky et al. (2016) do not perform any direct effect decompositions and do not discuss any implications that their study may hold for Mendelian Randomization.

The validity assumption is known to be untestable (see von Hinke et al. (2016) and Munafò et al. (2019) for the special case of MR). However, additional assumptions and considerations can be used to either support the validity assumption or to question it. Just as a number of authors rhetorically support their validity assumption, we cast doubts on this assumption based on theoretical and empirical considerations: our model is consistent with a strong direct effect of EA PGS on a number of health outcomes, an effect that works through early health and skills among possible other channels.

Education and Health We also contribute to the important debate about the effect of education on health. For recent literature reviews on the topic see Grossman (2015) and Galama et al. (2018).

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.[10] 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, but the concern is whether all confounders are fully controlled for. This paper diminishes concerns about results from literature (3) by adding a powerful genetic confounder in addition to detailed observable controls, cognitive and noncognitive skills, and accounted for unobserved heterogeneity, and still finds strong associations between education and health. 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). Natural experiments have a well-defined source of variation, but they only identify the local treatment effect 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). Results of these papers differ greatly. For instance, some (e.g., Lleras-Muney, 2005; van Kippersluis et al., 2011) find a strong effect of education on health-related outcomes, while others (e.g., Albouy and Lequien, 2009; Clark and Royer, 2013; Mazumder, 2008; Meghir et al., 2018) find none. Barcellos et al. (2018) found a negative effect of education on body size, an adverse lung function index, and blood pressure. This effect is stronger by absolute value for people with less favorable genetic endowments; however, the authors do not account for possible parental genetic confounding of this interaction.

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

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

trends, and differences in effects by population, cohort, and sex (Galama et al., 2018).

Literature (2) relies on differencing out a large number of constant 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 differences among siblings or twins in their early life. Also, establishing external validity of results based on these methods might 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., 2020; van den Berg et al., 2015), while some others find little to no effects (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 tend to better preserve statistical power than methods (1) and (2), and, unlike (1), do not limit results to local average treatment effects. The biggest concern with this literature is its ability to account for possible remaining 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, 2010; Savelyev and Tan, 2019). Further, Savelyev (2020) and Hong, Savelyev, and Tan (2000) also account for latent unobserved heterogeneity in addition to latent human capabilities. The contribution of this paper is that in addition to controls that have been used in this literature, we account for the EA PGS, a strong genetic confounder, plus control for general health PGS and mental health PGS, and still find strong conditional associations between education and health-related outcomes.

3 Data

We use the National Longitudinal Study of Adolescent to Adult Health (Add Health). This panel dataset follows roughly 20,000 individuals and contains detailed information on their family background, health outcomes, skills, and education. 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 high and middle 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 selected 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. 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 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 essential 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 (category 2 otherwise); and (4) a Bachelor's degree or above.[11]

[11]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. Also, we can expect the effects of more advanced degrees on health to be, at best, weak: there is evidence in

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). We also study three variables that measure substance use: frequent marijuana use, smoking tobacco, and risky drinking. We also have two variables related to lifestyle: "obesity" and "no exercise." Finally, we measure mental health using our "depression" variable. See Table 1 for variable definitions and descriptive statistics.

Potential Mechanisms We include a number of variables in the model in order to explain how the EA PGS translates to health outcomes in early adulthood. These variables include cognitive and noncognitive skills, the degree of education support in the household, and general health, all measured in the first wave. To reduce dimensionality and account for measurement error in multiple measures of the mechanisms, we use a factor model described in Section 4. See Table A-1 for measures of each factor discussed below.

To measure cognitive skills, we use participants' scores on the Add Health Picture Vocabulary Test, recent science grades, and recent math grades.[12] 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. Since noncognitive skills are malleable in young adulthood (e.g., Borghans et al., 2008; Fletcher and Schurer,

the literature that advanced degrees do not further contribute to health on top of the health effect of Bachelor's (Savelyev, 2020). This evidence is based on a high-IQ sample, but completing advanced degrees is strongly associated with having a high IQ (Jensen, 1998).

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

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

	Full Sample (N=3,709)		Low SES (N=1,030)		High SES (N=2,679)	
	Average	Standard Deviation	Average	Standard Deviation	Average	Standard Deviation
Highest Education Level						
Below high school[a]	0.048	0.214	0.097	0.296	0.029	0.169
High school diploma	0.415	0.493	0.517	0.500	0.375	0.484
College below Bachelor's[b]	0.174	0.380	0.181	0.385	0.172	0.378
Bachelor's or above	0.363	0.481	0.205	0.404	0.423	0.494
Health Outcomes						
General health[c]	3.745	0.889	3.580	0.912	3.809	0.871
Risky drinking of alcohol[d]	0.301	0.459	0.319	0.466	0.294	0.456
Smoking[e]	0.261	0.439	0.324	0.468	0.237	0.425
Marijuana use[f]	0.096	0.294	0.111	0.314	0.090	0.286
Obesity[g]	0.343	0.475	0.398	0.490	0.322	0.467
No exercise[h]	0.130	0.336	0.146	0.353	0.124	0.330
Depression[i]	0.191	0.393	0.205	0.404	0.186	0.389
Potential Mechanisms						
Cognitive skills[j]	0.000	1.000	-0.215	0.989	0.082	0.992
Conscientiousness[j]	0.000	1.000	0.039	1.015	-0.015	0.994
Extraversion[j]	0.000	1.000	0.125	1.013	-0.048	0.991
Neuroticism[j]	0.000	1.000	-0.126	1.014	0.049	0.991
Education support[j]	0.000	1.000	-0.195	1.013	0.075	0.985
Early health[k]	0.705	0.456	0.645	0.479	0.729	0.445

Notes: Calculations based on the Add Health data; estimation sample size, N, reported. For the purposes of descriptive analysis only (Tables 1, 2 and Figure 1), low SES is defined as having an adverse SES factor score above its average; high SES otherwise. [a]Having no high school diploma (including having a GED certificate). [b]Completed post-high school degree that takes 1–3 years. [c]General health self-evaluated on a scale from 1 (poor) to 5 (excellent). [d]Typical number of drinks per occasion exceeds three. [e]Smoking cigarettes at least once within the past 30 days. [f]Smoking marijuana once or more per week, on average, during the last year. [g]BMI $\geq$ 30. [h]None of the following: playing sports, exercising outside, walking for exercise, or engaging in other physical activity during the past week. [i]Had ever been told by a health care provider that they had depression. [j]Standardized factor score. See measures listed in Table A-1. [k]Self-reported good health.

2017), we model skills measured during the first wave of the Add Health study to avoid capturing reverse causality, which is more likely to appear in noncognitive skills that are measured in later waves. Due to data limitations, we are not able to study early Agreeableness and Openness, but we account for early Conscientiousness, Extraversion, and Emotional Stability using measures obtained when the Add Health cohort was in grades 7–12.[13] The measures of Big Five that we use were suggested by psychologists (Young and Beaujean, 2011a).

The education support in the household factor is based on measures of positive attitude towards education by the mother, father, and self. Since the educational motivation of a student and parental support both contribute to the choice of the highest degree completed in the education range from high school dropout to Bachelor's, this factor score is a parsimonious way to account for the contribution of aggregated household values regarding education to actual education choice. Finally, we account for self-reported good health in wave I.

In Table 1 we show descriptive statistics by SES level for all potential mechanisms. All factor scores are standardized for the full estimation sample.

SES To study the interaction of respondents' genetic endowment with low family SES in childhood we follow the literature (Bierut et al., 2018; Papageorge and Thom, 2020) and use multiple available measures of family hardship: (1) living in an unsafe neighborhood; (2) receiving government assistance (such as welfare); and (3) having difficulty paying bills. However, we deviate from the literature by using a factor score rather than an equally-weighted index, a summation of SES measures. We elaborate on this choice in section 4.

[13] We are unable to account for all possible mechanisms, but we allow for the direct effect that is unexplained by observed mechanisms.

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 that are based on well-established methodologies and fit the needs of this paper, we do not construct any PGSs ourselves. However, we still briefly outline the general principles of PGS construction as a way to make this key variable clear to all readers.

First, one needs to select relevant SNPs and estimate the weights that define a PGS. Then, the weights are used to impute the PGS. To select SNPs and estimate the weights, a researcher needs access to a large dataset (e.g., $N = 1,000,000$) that would contain both data on SNPs and data on a life outcome of interest. For instance, to calculate the EA PGS one needs data on the total years of formal schooling. By regressing a life outcome on SNPs taken one-by-one and selecting SNPs that generate regression coefficients with p-values that are low enough to account for the familywise error rate, a researcher identifies a set of SNPs that are highly predictive of that life outcome. The regression coefficients are then used to weight the contribution of each single SNP to the overall PGS, while adjusting for double-counting of correlated genetic effects (Domingue et al., 2015).[14] Once relevant SNPs are selected and related weights are calculated based on a large-sample dataset, a PGS can be imputed for any other dataset, such as Add Health, no matter the sample size, as long as relevant measurements of SNPs are available in that dataset. We use an EA PGS available in Add Health data that was constructed by Lee et al. (2018) using information from 1,271 SNPs that are predictive of education and based on a sample of 1.1 million individuals of European descent. This EA PGS predicts about 11% of variation in years of education completed among 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 that we use.

Because the EA PGS is constructed based on a sample of individuals with European

[14]The linkage disequilibrium regression method is used to avoid double counting.

Table 2: Polygenic Scores and Background Variables

	Full Sample (N=3,709)		Low SES (N=1,030)		High SES (N=2,679)	
	Average	Standard Deviation	Average	Standard Deviation	Average	Standard Deviation
Polygenic Scores						
EA PGS[a]	0.000	1.000	-0.177	0.988	0.068	0.996
Physical Health PGS[b,c]	0.000	1.000	-0.018	1.022	0.007	0.992
Mental Health PGS[b,d]	0.000	1.000	0.002	1.013	-0.001	0.995
Background Controls						
Biological sex is male	0.464	0.499	0.450	0.498	0.470	0.499
Age 10-12 at wave I	0.084	0.277	0.085	0.280	0.083	0.276
Age 13-14 at wave I	0.300	0.458	0.320	0.467	0.292	0.455
Age 15-16 at wave I	0.394	0.489	0.407	0.491	0.389	0.488
Age 17-19 at wave I	0.222	0.416	0.187	0.390	0.236	0.424
US Region: West	0.144	0.351	0.128	0.334	0.150	0.357
US Region: Mid-West	0.332	0.471	0.318	0.466	0.337	0.473
US Region: North-East	0.156	0.363	0.162	0.369	0.154	0.361
US Region: South	0.368	0.482	0.391	0.488	0.359	0.480
Rural residence	0.361	0.480	0.364	0.481	0.360	0.480
Suburban residence	0.399	0.490	0.353	0.478	0.417	0.493
Urban residence	0.239	0.427	0.283	0.451	0.223	0.416
Crime rate[e,f]	4,770	2,590	4,810	2,810	4,760	2,500
Poverty rate[f]	0.113	0.091	0.139	0.105	0.103	0.082
Low birthweight[g]	0.083	0.276	0.095	0.293	0.078	0.269
The only child	0.205	0.403	0.235	0.424	0.193	0.395
First-born	0.320	0.467	0.284	0.451	0.334	0.472
Second-born	0.308	0.462	0.287	0.453	0.316	0.465
Third-born	0.112	0.316	0.124	0.329	0.108	0.310
Number of siblings	2.549	1.950	2.952	2.265	2.394	1.791
College-educated parent[h]	0.523	0.500	0.435	0.496	0.549	0.498
Parents married	0.805	0.396	0.652	0.477	0.871	0.335
Cigarettes smoked at home	0.459	0.498	0.575	0.495	0.408	0.492
Meals with parents[i]	4.897	2.328	4.715	2.472	4.967	2.266
Genetic ancestry PC[j]	Yes		Yes		Yes	

Notes: Calculations based on the Add Health data; estimation sample size, N, reported. For the purposes of descriptive analysis only (Tables 1, 2 and Figure 1), low SES is defined as adverse SES factor score above its average; high SES otherwise. [a]Standardized EA PGS (Lee et al., 2018). [b]Standardized factor score. [c]Based on 7 PGS scores that predict physical health (see Table A-1). [d]Based on 6 PGS scores that predict mental health (see Table A-1). [e]Per population of 100,000. [f]Census Regional Variables. [g]Birthweight$\leq$5.5 lbs. [h]At least one parent has a 4-year college degree. [i]Number of evening meals with parents per week. [j]Principal components based on genetic data describing ethnic origin.

ancestry, we restrict our sample to those who self-report as white.[15] Here we follow the literature confirming that polygenic scores constructed using European-ancestry samples can be both biased and less predictive when applied to different populations (Martin et al., 2017; Price et al., 2006). There are several reasons why a PGS constructed from one population's genetic data exhibits different statistical and predictive properties in a population with a different genetic ancestry. First, there could be differences between the two groups in the prevalence of SNPs used in the score's construction. Second, it may also be that SNPs identified in one population as predictive of a particular outcome (phenotype) are not predictive of that outcome in other populations. Finally, the extent to which particular genetic differences are correlated with other types of genetic differences could vary across ethnic groups (linkage disequilibrium). See Martin et al. (2017) for more details about the difficulties in applying polygenic scores to different populations.

In addition to modeling the effects of EA PGS, which is our main variable of interest, we also control for general and mental health endowments as possible confounders. However, there are multiple types of correlated PGSs that describe general and mental health. To avoid multicollinearity and to keep our model parsimonious, we construct factor scores for general and mental health (see Table A-1 for measures of the factors).

We standardize all polygenic scores for the estimation sample prior to main model estimation (see Table 2 for descriptive statistics of PGS by SES).

Background Control Variables 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, crime and poverty rates in the neighborhood, low birth weight, number of siblings, the order of birth among siblings unless the only child, having a college-educated parent, having parents who are married, cigarettes smoked at home, number of meals with parents per week, and genetic ancestry. See

[15] A reliable PGS construction requires large quantities of genetic data, which are not yet available for non-whites.

Table 2 for variable definitions and descriptive statistics by SES.

4 Methods

4.1 Reduced Form Model

We first estimate a reduced form model of the PGS-SES interaction, comparable to models from the economic literature on gene-environment interactions (e.g., Bierut et al., 2018; Fletcher, 2019; Schmitz and Conley, 2017). The model is specified as follows:

$$Y_{2k}^{*} = \alpha_{0k} + \alpha_{1k}\boldsymbol{X} + \boldsymbol{\alpha}_{2k}\boldsymbol{PGS} + \alpha_{3k}\Theta^{SES} + \boldsymbol{\alpha}_{4k}\boldsymbol{PGS}\cdot\Theta^{SES} + \zeta_k, \quad k = 1, ..., 8, \qquad (1)$$

where Y_{2k}^{*} denotes a latent propensity for a health-related outcome Y_{2k} in a logit model, so that $Y_{2k} = 1$ if $Y_{2k}^{*} > 0$ and $Y_{2k} = 0$ otherwise. For categorical general health and education outcomes we use an ordered logit model. $\boldsymbol{PGS}$ denotes a vector of PGSs: EA PGS, General Health PGS, and Mental Health PGS; Θ^{SES} denotes latent parental SES, defined so that larger values imply a greater degree of disadvantage.[16] ζ_k is an idiosyncratic error term. $\boldsymbol{X}$ is a vector of controls that are shown in Table 2.

4.2 Main Model

In addition to the reduced form model (1), we also estimate our main model, which has two key advantages: (1) it controls for unobserved heterogeneity; (2) it allows us to study the mechanisms behind the estimated effects.

We simultaneously estimate a system of equations accounting for: (1) common unobserved determinants of parental SES, subject's PGS, and subject's life outcomes, (2)

[16] To avoid repetition of equations, we define the factor model for SES, General Health PGS, and Mental Health PGS as part of our more general main model description (equation 7), as both reduced and main models share the same way of modeling these three factors.

formation of initial skills, health, and education support in the household, (3) education choices, and (4) health outcomes in young adulthood.

We use a full maximum likelihood estimation and take advantage of the sequential nature of human capital formation. Genetic endowments measured by PGS scores are determined at conception. The possible mechanisms, such as early skills and health, are affected by subject's genetic endowment and parental SES and are formed before decisions about post-compulsory schooling are made. Schooling occurs before adult health outcomes are realized. Therefore, it is a plausible assumption that later outcomes do not affect early outcomes conditional on observable controls and approximated unobserved heterogeneity, an assumption that leads to essential identifying exclusion restrictions.

These exclusion restrictions imply recursive equations. Simultaneously estimating a system of such equations both provides gains in efficiency and allows us to control for unobserved heterogeneity, $\boldsymbol{\mu}$, using the semiparametric heterogenity model (Heckman and Singer, 1984).[17] Mroz (1999) shows that such control for unobserved heterogeneity is effective for solving the endogeneity problem in a recursive nonlinear system of equations. We interpret this control for unobserved heterogeneity as capturing important personal, family, or genetic characteristics outside of what is explicitly controlled for.

The recursive sub-model that relates causes to outcomes through potential mecha-

[17]The model is also known as latent class analysis (Aitken and Rubin, 1985), discrete factor approximation (Mroz, 1999), discrete factor random-effects model (Gilleskie, 2014), and finite mixture modeling of unobserved heterogeneity (Cameron and Trivedi, 2005).

nisms is the following:

$$\boldsymbol{PGS} = \boldsymbol{a}_1 \tilde{\boldsymbol{X}} + \boldsymbol{\mu}^{PGS} + \boldsymbol{\epsilon}^{PGS} \tag{2}$$

$$\Theta^{SES} = \boldsymbol{b}_1 \tilde{\boldsymbol{X}} + \mu^{SES} + \epsilon^{SES} \tag{3}$$

$$\boldsymbol{\Theta}^{Y_1} = \boldsymbol{c}_1 \boldsymbol{X} + \boldsymbol{c}_2 \boldsymbol{PGS} + \boldsymbol{c}_3 \Theta^{SES} + \boldsymbol{c}_4 \boldsymbol{PGS} \cdot \Theta^{SES} + \boldsymbol{\mu}^{Y_1} + \boldsymbol{\epsilon}^{Y_1} \tag{4}$$

$$\begin{aligned} D^* = {} & \boldsymbol{d}_1 \boldsymbol{X} + \boldsymbol{d}_2 \boldsymbol{PGS} + d_3 \Theta^{SES} + \boldsymbol{d}_4 \boldsymbol{PGS} \cdot \Theta^{SES} + \boldsymbol{d}_5 \boldsymbol{\Theta}^{Y_1} \\ & + \boldsymbol{d}_6 \boldsymbol{\Theta}^{Y_1} \cdot \Theta^{SES} + \mu^D + \epsilon^D \end{aligned} \tag{5}$$

$$\begin{aligned} Y_{2k}^* = {} & \boldsymbol{e}_{1k} \boldsymbol{X} + \boldsymbol{e}_{2k} \boldsymbol{PGS} + e_{3k} \Theta^{SES} + \boldsymbol{e}_{4k} \boldsymbol{PGS} \cdot \Theta^{SES} + \boldsymbol{e}_{5k} \boldsymbol{\Theta}^{Y_1} \\ & + \boldsymbol{e}_{6k} \boldsymbol{\Theta}^{Y_1} \cdot \Theta^{SES} + \boldsymbol{e}_{7k} \boldsymbol{D} + \boldsymbol{e}_{8k} \boldsymbol{D} \cdot \Theta^{SES} + \mu^{Y_{2k}} + \epsilon^{Y_{2k}}, \quad k = 1, ..., 7, \end{aligned} \tag{6}$$

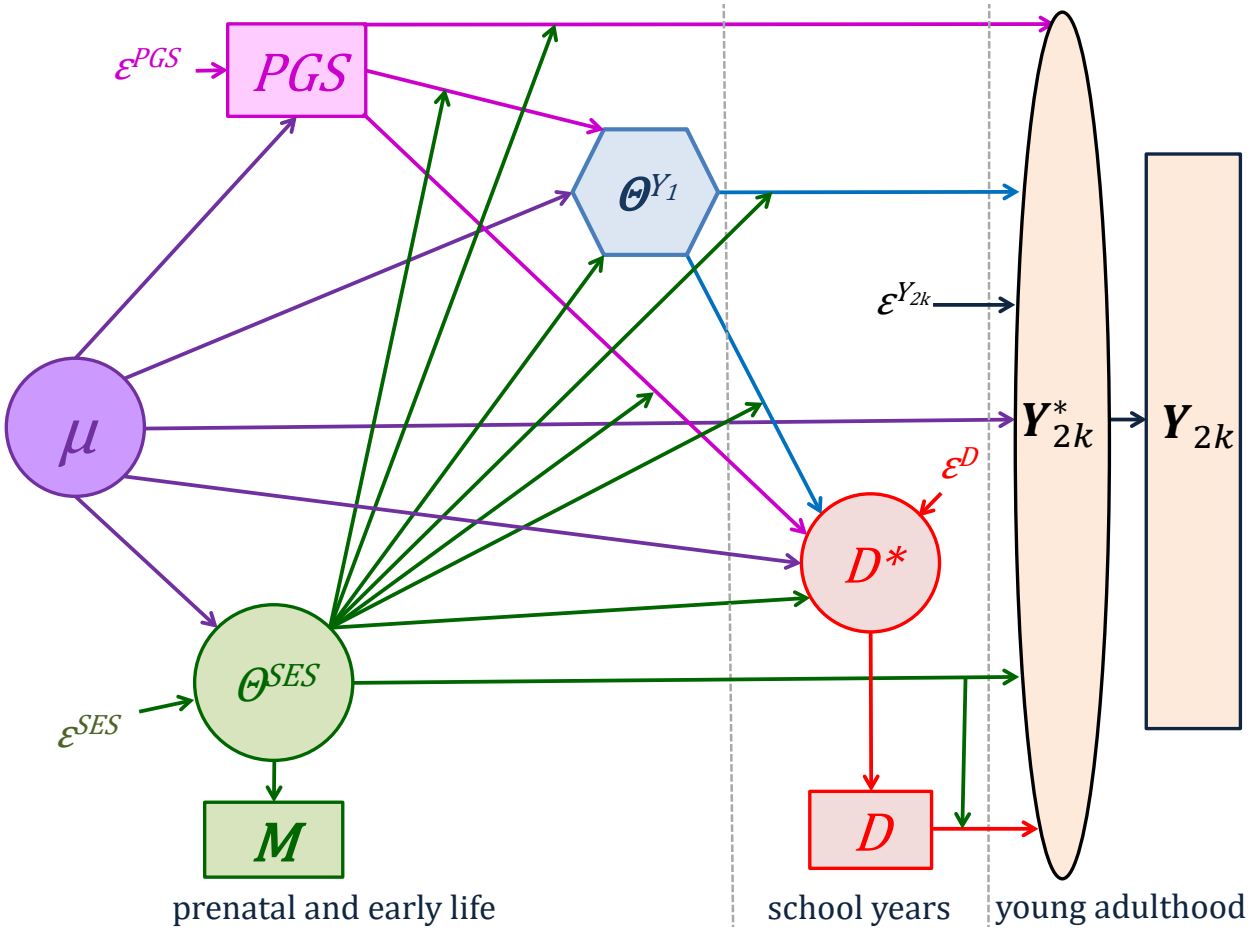
where we use the same notation as in model (1), as well as: $\boldsymbol{\Theta}^{Y_1}$, the vector of potential mechanisms; D^*, the latent propensity for education in an ordered logit model; $\boldsymbol{D}$, the vector of binary schooling choice variables. Vector $\tilde{\boldsymbol{X}}$ represents a reduced set of controls that could have affected early outcomes. Vector $\boldsymbol{X}$ represents the complete set of available background controls (see Table 2). We estimate the model separately for each health-related outcome, Y_{2k}, due to computational limitations. Symbols ϵ^{PGS}, ϵ^{SES}, ϵ^{Y_1}, ϵ^D, and $\epsilon^{Y_{2k}}$ represent idiosyncratic error terms.

We present a visualization of our main model (2–6) in Figure 2, which shows causal links between variables that are determined before birth, at early life, during school years, and later in young adulthood, relationships between observed and latent variables, and interaction effects.

Measurement System for the Factor Model In order to reduce dimensionality, account for measurement error in variables, and avoid multicollinearity issues, we use a factor model, which is a well-established methodology for achieving these goals.

For an identification of the factor model it is essential to specify a measurement system (7) in addition to equations (2–6). The measurement system relates each latent

Figure 2: Diagram of the Main Model Conditional on Background Variables



Notes: The diagram represents the main model (2–7) conditional on background variables, $\boldsymbol{X}$ (not shown to avoid clutter). Rounds and ellipses represent latent variables. Rectangles represent observables. A hexagon represents a vector of potential mechanisms, which are multiple types of variables, latent (socioemotional skills, cognitive skills, and a degree of education support by parents and self) and observed (early health). Arrows ending in a geometric shape denote causal links. Arrows ending in the middle of another arrow denote interactions. Notation of variables matches the notation of the main model: $\boldsymbol{\mu}$ is unobserved heterogeneity; $\boldsymbol{M}$ is a vector of adverse SES measures. D^* is a latent propensity for education; D is chosen highest education level; Y^*_{2k} is a latent propensity to have health-related outcome Y_{2k}; ϵ^{PGS}, ϵ^{SES}, ϵ^{D}, $\epsilon^{Y_{2k}}$ represent idiosyncratic error terms.

factor of type s, Θ^s, to its observable measures, M_j^s:

$$M_j^s = \beta_{0,j}^s + \boldsymbol{\beta}_{1,j}^s \tilde{\boldsymbol{X}} + \beta_{1,j}^s \Theta^s + \eta_j^s, \quad s = 1, ..., 7, \quad j = 1, ..., J^s, \tag{7}$$

where $\beta_{0,j}^s$ and $\boldsymbol{\beta}_{1,j}^s$ are coefficients, $\beta_{1,j}^s$ is a factor loading, and η_j^s is the error term. We make the standard assumption that $\eta_j^{s_1} \perp\!\!\!\perp \eta_l^{s_2}$ for any $s_1, s_2 = 1, ..., 7$ and any pair of j and l, $j = 1, ..., J^s$; $l = 1, ..., J^{s_2}$, except for the case where $s_1 = s_2$ and $j = l$, and that error terms are independent of latent factors. Each latent variable is normalized to have mean zero and variance one. For each factor of type s, we make another standard normalization: $\beta_{1,1}^s > 0$ so that skills have the usual interpretation without a loss of generality.[18]

We use a similar measurement system for the reduced form model (1), with the only difference being that the reduced form model utilizes only three factors: parental SES, general health PGS, and mental health PGS, a subset of seven factors that are used in the main model.

Factors can be used in the model as latent variables or they can be replaced by factor scores, the unbiased estimates of latent factors (e.g., Anderson and Rubin, 1956; Yung and Yuan, 2013). While it is more efficient to estimate all sub-models simultaneously, this choice is impractical given our computational limitations. We have as many as eight continuous latent factors that appear both on the left-hand side and on the right-hand side of equations, plus a discrete latent factor, $\boldsymbol{\mu}$, with points of support that differ across a system of 11 equations of the recursive system. Moreover, we estimate this model 400 times (and then 800 times again to be sure) from different random starting points to rule out local maxima of the maximum likelihood function. Therefore, as a compromise, we keep the discrete unobserved heterogeneity factor as latent, but use factor scores to account for continuous latent factors.

[18]For instance, more emotionally-stable people have a higher (not lower) score for "Emotional Stability."

We follow a paper by psychologists Young and Beaujean (2011a) who propose measures of early Conscientiousness (C), Extraversion (E), and Emotional Stability (ES) based on available measures of personality in the first wave of Add Health. We also estimate a factor of early Cognition (G) based on a cognitive test and school achievements, a factor of education support in the household based on its many proxies, and factors for genetic endowment in physical and mental health based on multiple PGSs related to these two factors.[19]

Using a factor score for SES has three advantages: 1) We avoid using an index that has arbitrarily equal weights (a sum of measures); 2) We avoid using a binary SES variable that is generated with information loss and also creates issues with numerical stability of our main model; 3) We explicitly account for measurement error in proxies of SES through a factor model.

Identification This model consists of standard components, the identification of which is well-established. The factor model (7) is standard and has at least three dedicated measures of each factor, which is a sufficient identification condition as long as standard normalizations and assumptions of the factor model are made (Anderson and Rubin, 1956). Equations (2–6) have a recursive structure, which contributes to identification conditional on idiosyncratic error terms being uncorrelated among recursive parts (Maddala, 1983). Unobserved heterogeneity terms $\boldsymbol{\mu}$ help to satisfy this condition by controlling for common correlation across outcomes conditional on observables.

Decomposition Our main model allows us to decompose the effect of EA PGS on conditional expectation of Y_{2k} for each outcome of type k with respect to the underlying mechanisms. In particular, by taking a derivative of the conditional expectation of Y_{2k} with respect to the EA PGS and applying the chain rule, we can get two parts of the

[19]See Table A-1 of the Appendix for the list of measures for all continuous latent factors.

effect of the EA PGS on health-related outcome Y_{2k}: an indirect effect (IE_k) that works through education and the direct effect (DE_k), which works through other channels.

$$
\begin{aligned}
IE_k = & \underbrace{\sum_{q=1}^{3}(\tilde{e}_{7k}^{q} + \tilde{e}_{8k}^{q}\Theta^{SES})(\tilde{\boldsymbol{d}}_{5}^{q} + \tilde{\boldsymbol{d}}_{6}^{q}\Theta^{SES})(\tilde{c}_{2}^{E} + \tilde{c}_{4}^{E}\Theta^{SES})}_{\text{effect through the mechanisms}} \\
& + \underbrace{(\sum_{q=1}^{3} \tilde{e}_{8k}^{qE}\tilde{d}_{2}^{q}\Theta^{SES} + \tilde{e}_{7k}^{q}\tilde{d}_{4}^{qE}\Theta^{SES} + \tilde{e}_{8k}^{q}\tilde{d}_{4}^{qE}(\Theta^{SES})^2)}_{\text{SES directly}} + \underbrace{\sum_{q=1}^{3}(\tilde{e}_{7k}^{q}\tilde{d}_{2}^{qE})}_{\text{other channels}} \qquad (8) \\
DE_k = & \underbrace{(\tilde{\boldsymbol{e}}_{5k} + \tilde{\boldsymbol{e}}_{6k}\Theta^{SES})(\tilde{\boldsymbol{c}}_{2}^{E} + \tilde{\boldsymbol{c}}_{4}^{E}\Theta^{SES})}_{\text{effect through the mechanisms}} \\
& + \underbrace{\tilde{e}_{4k}^{E}\Theta^{SES}}_{\text{SES directly}} + \underbrace{\tilde{e}_{2k}^{E}}_{\text{other channels}}. \qquad (9)
\end{aligned}
$$

Here tildes above coefficients denote marginal coefficients in logit models, which are proportional to corresponding model coefficients.

Components of Equations (8–9) can be re-arranged in various ways depending on the research question at hand. In particular, in addition to our concentration on the direct and indirect effects through education, we can concentrate on the total contribution of each specific mechanism to the total effect: terms labeled "effects through the mechanisms," that are present in both equations, can be put together and studied as the total contribution through the mechanisms.

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 control variables without diminishing our sample size.

5 Results

Below we first explore the interaction of the education PGS and SES using a reduced-form model that is comparable with the literature. Then we analyze the results of the more advanced main model. The qualitative results of the main model are consistent with those of the reduced-form model.

5.1 Reduced Form Model for Health-Related Outcomes

We first estimate a reduced-form model (1). As we argue in Section 3, our approach to defining SES is similar to the one used in economic literature, with the difference being that we use a factor score instead of a sum of equally-weighted measures of disadvantage or a binary variable. Our reduced form model is conditional on a large set of background controls from Table 2, the same set that we control for in the main model, including possible genetic confounders: general health PGS and mental health PGS and their interactions with SES.

In Table 3, the EA PGS shows statistically significant and strong associations with a number of health-beneficial outcomes: superior general health, no smoking, no frequent marijuana use, and no depression. We also see borderline statistically significant associations of EA PGS with risky drinking, obesity, and lack of exercise, with signs of the estimates all pointing in a health-beneficial direction.

A statistically significant interaction with the SES score is present for general health, marijuana use, lack of exercise, and depression. For risky drinking this interaction is borderline statistically significant (it will become statistically significant in the main model, which reduces unexplained variation). All estimated interaction effects act in the opposite direction of the effects of EA PGS. The interpretation of this result is that EA PGS is less health-beneficial for those with low SES status (low SES status implies a high value of our SES factor score). This result is consistent with the bottleneck hypothesis (Fletcher,

Table 3: Conditional Reduced-Form Associations Between EA PGS, Gene-SES Interaction, and Health-Related Outcomes, Logit Regression Results

	General health (1)	Risky drinking (2)	Smoking tobacco (3)	Marijuana use (4)	Obesity (5)	Lack of exercise (6)	Depression (7)
Education PGS	0.111 ***	-0.010	-0.056 ***	-0.012 **	-0.013	-0.008	-0.014 **
	(0.032)	*(0.008)*	*(0.008)*	*(0.005)*	*(0.008)*	*(0.006)*	*(0.007)*
Education PGS x SES score	-0.068 **	0.013	0.002	0.009 *	0.006	0.010 **	0.015 **
	(0.032)	*(0.008)*	*(0.007)*	*(0.005)*	*(0.008)*	*(0.005)*	*(0.007)*

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.

2019): low SES is a good proxy for severely constrained conditions in childhood.

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 tobacco). 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.

We also explore a corresponding model employing family fixed effects, documented in Table A-2 of the Appendix, but find that it suffers from low statistical power. Expectedly, the statistical power is greatly diminished compared to our main model for two reasons: (1) a major reduction of identifying variation 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, both our reduced-form model and our main model use 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 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 having a sample of siblings more than three times larger in their study of a Danish population than we have in the Add Health data.

5.2 The Main Model

Our main model (2–7) has the following advantages over the reduced-form model (1): (1) it controls for unobserved heterogeneity $\boldsymbol{\mu}$, and (2) it accounts for potential mechanisms $\boldsymbol{\Theta}^{Y_1}$, which allows us to study decompositions, such as (8,9). Under the assumptions of the model, the effects can be viewed as causal. A conservative interpretation of the results would be as associations conditional on a large number of essential observables and approximated unobservables.

Effects of EA PGS on Health-Related Outcomes and PGS-SES Interactions Table 4 shows the total effect of the EA PGS on health-related outcomes, both the effect evaluated at the average level of SES and the interaction effect, which is the effect of increasing the SES factor by one standard deviation. Here a higher value of the SES variable implies a higher degree of socioeconomic disadvantage. Additionally, we decompose both effects with respect to direct and indirect components using formulas (8,9), where the indirect effect works through education, and the direct effect works through all mechanisms that are unrelated to education.

The results of Table 4 qualitatively confirm the results of the reduced form model from Table 3, as we again see beneficial total effects of the EA PGS on many health-related outcomes (see Panel A), plus total PGS-SES interaction effects of the opposite

Table 4: Direct, Indirect, and Total Effects of EA PGS on Health-Related Outcomes

	General health (1)	Risky Drinking (2)	Smoking Tobacco (3)	Marijuana Use (4)	Obesity (5)	Lack of exercise (6)	Depression (7)
A. At $\overline{SES}$[(a)]							
Total effect	0.139 ***	-0.010	-0.058 ***	-0.010 **	-0.018 **	-0.009	-0.017 **
	(0.035)	*(0.008)*	*(0.008)*	*(0.004)*	*(0.009)*	*(0.006)*	*(0.007)*
Effect size	-	-0.033	-0.222	-0.105	-0.052	-0.069	-0.089
Indirect effect	0.051 ***	-0.009 ***	-0.021 ***	-0.006 ***	-0.005 **	-0.007 ***	-0.005 ***
	(0.009)	*(0.002)*	*(0.003)*	*(0.001)*	*(0.002)*	*(0.002)*	*(0.002)*
Direct effect	0.088 **	-0.002	-0.038 ***	-0.004	-0.013	-0.002	-0.012 *
	(0.035)	*(0.008)*	*(0.008)*	*(0.004)*	*(0.009)*	*(0.005)*	*(0.007)*
Indirect-Direct Difference[(c)]	-0.037	-0.007	0.017 **	-0.002	0.008	-0.004	0.007
	(0.037)	*(0.009)*	*(0.008)*	*(0.004)*	*(0.009)*	*(0.006)*	*(0.007)*
B. Interaction[(b)]							
Total effect	-0.090 ***	0.014 *	0.003	0.008 **	0.007	0.011 **	0.016 ***
	(0.033)	*(0.008)*	*(0.007)*	*(0.004)*	*(0.008)*	*(0.005)*	*(0.006)*
Effect size	-	0.047	0.011	0.084	0.020	0.085	0.084
Indirect effect	-0.004	0.002	0.002	0.000	0.000	-0.001	0.002
	(0.009)	*(0.002)*	*(0.003)*	*(0.001)*	*(0.002)*	*(0.002)*	*(0.002)*
Direct effect	-0.086 **	0.012	0.001	0.008 **	0.007	0.011 **	0.014 **
	(0.034)	*(0.008)*	*(0.007)*	*(0.004)*	*(0.009)*	*(0.005)*	*(0.006)*
Indirect-Direct Difference[(c)]	0.082 **	-0.010	0.001	-0.008 *	-0.007	-0.012 **	-0.013 *
	(0.036)	*(0.009)*	*(0.008)*	*(0.004)*	*(0.009)*	*(0.006)*	*(0.007)*

Notes: Asterisks indicate statistical significance level: ***, 1 % level; **, 5 % level; *, 10 % level. Calculations are based on the main model (2–7), decompositions (8–9), and the Add Health data. Sample size is 3709. [(a)]Effect estimated at the average level of SES. [(b)]Effect of increasing the low SES score by one standard deviation. [(c)]The difference between indirect and direct effects.

sign (see Panel B). Moreover, perhaps through decreasing residual variation, we gained statistical significance for two effects that are borderline statistically significant in the reduced form model: the negative effect of EA PGS on the probability of obesity, and the positive interaction effect for risky drinking. Expectedly, the estimates somewhat changed numerically in our main model compared to the reduced form model. Statistically significant estimates of the effects of EA PGS changed in absolute value by up to 25%, while estimates of statistically significant interactions changed in absolute value up to about 30%.

Table 4 also shows the effect sizes, which suggest that the estimated statistically significant effects are also economically significant. The effect size is 22% on smoking tobacco, 11% on marijuana use, 5% on obesity, and 9% on depression. The interaction effect sizes are also substantial: 5% for risky drinking, 8% for marijuana use, 9% for lack of exercise, and 8% for depression.

Direct and Indirect Effects From the analysis of the direct and indirect effects of the EA PGS on health-related outcomes (see Panel A of Table 4) we can see that education is not the only channel through which EA PGS affects health. As expected, we can see large and statistically significant indirect effects, the effects explained by education. The direct effects tend to have larger standard errors than the indirect effects, but still we observe strong and statistically significant direct effects for general health, smoking tobacco, and depression.

Notably, the indirect effect is statistically significant for all seven outcomes including risky drinking and lack of exercise, for which the total effect is statistically insignificant, which is clearly driven by large standard errors of the direct effects.

The direct and indirect effects have comparable contributions to the total effect for these outcomes: we test and do not reject the hypothesis that the direct effect is the same as the indirect effect for all outcomes except for smoking tobacco. Moreover, for smoking

tobacco the direct effect is statistically larger by absolute value than the indirect effect.

Among the outcomes that demonstrate a strong direct effect, the general health outcome is particularly important, as it relies on a subjective evaluation of health to summarize all health issues and behaviors that affect quality of life, including health determinants that we do not observe. For general health, the indirect effect is estimated as about 5 PP ($p = 0.000$), and the direct effect as about 9 PP ($p = 0.013$). The discovery of the direct effect is important for our criticism of the MR methodology discussed in the literature review. Below in this section we further support the existence of the direct effect by demonstrating that it works through early health and skills.

Additionally, in Panel B of Table 4, we decompose the EA PGS-SES interaction effect into direct and indirect components. Here we can see that all statistically significant total interaction effects are explained by the direct effects rather than the indirect effects, and the corresponding differences between the direct and indirect effects tend to be statistically significant. Therefore, we can conclude that disadvantaged SES diminishes the effect of EA PGS not through education but, possibly, through other channels and also directly (through SES itself). Perhaps, apart from direct educational benefits, such as an ability to quickly understand new ideas and solve new problems, having a high education PGS leads children and young adults to make better use of available family resources for their development (parental knowledge and networks, family wealth, access to health care, access to certain compulsory schools). Because high-SES children have access to superior parental resources, they are better able to leverage their genetic endowment.

EA PGS, SES, and 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 surveyed in Section 2. This interaction is not the focus of this paper. However, because this interaction is a part of the mechanisms linking EA PGS and health, it is important for us

to understand it for the population that we study.

Table 5 shows results both for the reduced form estimation of model (1), with categorical education used as an outcome (see Panel A), and comparable results based on the main model estimation (Panel B). As before, results of the reduced form and the main model give us similar qualitative results, with some differences in estimated effects.

Table 5: Total Marginal Effect of EA PGS on Education Levels

	Below High School (1)	High School Diploma (2)	College below Bachelor's (3)	Bachelor's or above (4)
A. Reduced form[a]				
At $\overline{SES}$[b]	-0.009 ***	-0.081 ***	-0.001	0.091 ***
	(0.001)	*(0.008)*	*(0.002)*	*(0.008)*
Effect size[c]	-0.188	-0.195	-0.006	0.251
Interaction[d]	0.000	0.004	0.000	-0.004
	(0.001)	*(0.007)*	*(0.000)*	*(0.008)*
B. Main model[e]				
At $\overline{SES}$[b]	-0.012 ***	-0.094 ***	0.028 ***	0.079 ***
	(0.002)	*(0.009)*	*(0.004)*	*(0.008)*
Effect size[c]	-0.250	-0.227	0.161	0.218
Interaction[d]	0.001	0.008	-0.002	-0.006
	(0.001)	*(0.009)*	*(0.003)*	*(0.007)*

Notes: Asterisks indicate statistical significance level: ***, 1 % level; **, 5 % level; *, 10 % level. Calculations are based on the Add Health data. Sample size is 3709. [a]Accounts for observed controls presented in Table 2 but does not account for unobserved heterogeneity. [b]Effect of one standard deviation increase in EA PGS at the average level of SES. [c]Ratio of the estimated effect to the sample average of the outcome. [d]The effect of EA PGS $\times$ SES. [e]Based on the main model (2–7), which accounts for unobserved heterogeneity.

Because the EA PGS is designed to predict years of formal education and is known to be externally valid, it is not surprising that both reduced form and the main models show a strong positive relationship between the EA PGS and the highest education level (the effect is evaluated at the average SES level). We can see that the EA PGS makes

lower levels of education less likely and higher levels of education more likely. The only qualitative difference between the reduced form and the main model is at which level of education the effect changes its sign (obviously, effects across a full set of education levels sum up to zero). For the reduced form model, the change of effect sign happens at about the college degree below Bachelor's level (column 3). For the main model, the sign change happens between the high school diploma (column 2) and the college degree below Bachelor's level (column 3), as there is a negative effect on getting a high school diploma but a positive effect on the college degree below Bachelor's level. Otherwise, the estimated effects are comparable across models and the estimates are statistically significant at the 1% level.

Unlike the effects of the EA PGS, the interaction effects are statistically insignificant for both the reduced form and main models, which is a departure from the results of the previous literature (Fletcher, 2019; Papageorge and Thom, 2020; Ronda et al., 2020). This lack of statistical significance is not driven by high standard errors (they are not any larger than for the main effect), but by small estimates of the interaction coefficients.

The lack of interaction effects for our sample is robust to omitting our detailed controls either fully or partially, though using a smaller set of controls makes the estimates closer to being statistically significant, as the estimates of the interaction tend to increase in absolute value and the standard error stays similar (See Table A-3 of the Appendix). This feature could partially explain the difference with other papers, which tend to use a smaller set of controls.[20]

Other possible sources of differences with the literature may include the following two: (1) differences in general populations, and (2) alternative definitions of SES. While we cannot test hypothesis (1) based on our sample, we can shed light on hypothesis (2). We find that our no-interaction result is robust to a number of alternative definitions of SES that can be constructed based on the Add Health data (see Table A-4 of the

[20]Conditional on having superior statistical power to ours, these changes in controls could possibly make a difference between accepting rejecting of the null hypothesis.

Appendix).

The Mechanisms Table 6 presents decompositions of the total effect of EA PGS on health-related outcomes with respect to possible mechanisms. It also reports similar decompositions for the PGS-SES interaction effect.

The total effect of the EA PGS is decomposed in Panel A at the average level of SES. As before, we pay special attention to results for general health (Column 1), because this outcome is a quality-of-life-relevant summary of health that includes health issues unobserved by the econometrician. We can see sizable and statistically significant contributions to general health from such mechanisms as early health, skills, education support in the household, and the direct effect of education. The latter is a part of the total effect that is explained by education but not explained by early health, early skills, or college support in the household. The last term in the decomposition, called "other channels"— which is a part of the total effect that is not explained by the observed mechanisms that we model—is not precisely determined. The bottom of Panel A shows the total effect, which is a sum of all contributions from the decomposition, the same number that we have seen in Table 4.

Panel B has the same structure as Panel A, but different results for the general health outcome: the contributions of early health, skills, college support, and education to the total interaction effect are not precisely determined. The residual part of the decomposition, called "SES directly," is statistically significant and comparable in size to the total effect. We can interpret this result in the following way: the observed potential mechanisms play at best a weak role, while disadvantaged SES itself has a strong direct effect on the interaction. Perhaps parental resources help leverage more productive genetic endowments and help compensate for less productive ones. The results of these parental investments may change health outcomes in young adulthood, but not through the mechanisms that we observe in early life. Therefore, SES can be a powerful mecha-

Table 6: Decomposition of the Total Effect of EA PGS on Health-Related Outcomes with Respect to Possible Mechanisms

	General health (1)	Risky drinking (2)	Smoking tobacco (3)	Marijuana use (4)	Obesity (5)	Lack of exercise (6)	Depression (7)
A. At $\overline{SES}$[(a)]							
Early health	0.021 ***	0.000	-0.003 ***	-0.001 *	-0.004 ***	-0.001 **	-0.002 **
	(0.007)	*(0.001)*	*(0.001)*	*(0.000)*	*(0.001)*	*(0.001)*	*(0.001)*
Skills	0.033 ***	-0.006 ***	-0.010 ***	-0.003 ***	-0.002	-0.004 ***	-0.001
	(0.009)	*(0.002)*	*(0.002)*	*(0.001)*	*(0.002)*	*(0.001)*	*(0.002)*
College support	0.008 **	0.000	-0.002 **	-0.001 **	0.000	-0.001	-0.001
	(0.004)	*(0.001)*	*(0.001)*	*(0.000)*	*(0.001)*	*(0.001)*	*(0.001)*
Education directly	0.032 ***	-0.005 ***	-0.013 ***	-0.004 ***	-0.003 **	-0.004 ***	-0.003 ***
	(0.007)	*(0.002)*	*(0.002)*	*(0.001)*	*(0.001)*	*(0.001)*	*(0.001)*
Other channels	0.044	0.002	-0.031 ***	-0.002	-0.009	0.001	-0.010
	(0.036)	*(0.009)*	*(0.008)*	*(0.004)*	*(0.009)*	*(0.006)*	*(0.007)*
Total effect	0.139 ***	-0.01	-0.058 ***	-0.01 **	-0.018 **	-0.009	-0.017 **
	(0.035)	*(0.008)*	*(0.008)*	*(0.004)*	*(0.009)*	*(0.006)*	*(0.007)*
B. Interaction[(b)]							
Early health	-0.006	0.000	0.000	0.000	0.001	0.000	0.000
	(0.005)	*(0.001)*	*(0.001)*	*(0.000)*	*(0.001)*	*(0.000)*	*(0.001)*
Skills	-0.008	0.003 *	0.004 **	0.000	-0.001	0.001	-0.001
	(0.008)	*(0.002)*	*(0.002)*	*(0.001)*	*(0.002)*	*(0.001)*	*(0.001)*
College support	0.002	0.000	0.001	0.000	0.001	0.000	0.001 **
	(0.003)	*(0.001)*	*(0.001)*	*(0.000)*	*(0.001)*	*(0.000)*	*(0.001)*
Education directly	-0.001	0.001	0.001	0.000	0.000	-0.001	0.001
	(0.007)	*(0.001)*	*(0.002)*	*(0.001)*	*(0.001)*	*(0.001)*	*(0.001)*
SES directly	-0.077 **	0.010	-0.002	0.008 **	0.007	0.011 **	0.015 **
	(0.034)	*(0.008)*	*(0.007)*	*(0.004)*	*(0.009)*	*(0.005)*	*(0.006)*
Total effect	-0.09 ***	0.014 *	0.003	0.008 **	0.007	0.011 **	0.016 ***
	(0.033)	*(0.008)*	*(0.007)*	*(0.004)*	*(0.008)*	*(0.005)*	*(0.006)*

Notes: Asterisks indicate statistical significance level: ***, 1 % level; **, 5 % level; *, 10 % level. Calculations are based on the main model (2–7) using 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)]Effects are calculated at the average level of SES. [(b)]The change of the effect when the adverse SES score increases by one standard deviation.

nism in its own right.

Decompositions for other outcomes that show a statistically significant total effect of the EA PGS are similar (see columns 3, 4, 5, and 7 of Panel A) in that they all explain the total through 2–4 types of statistically significant mechanisms. Moreover, even for the totals that are not statistically significant (see Columns 2 and 6 of Panel A), we still see 2–3 statistically significant mechanisms for each, suggesting a beneficial effect through these mechanisms (a reduction of health-adverse outcomes). It is mainly the noise from the unobserved channel that leads to a statistically insignificant total in columns (2) and (6).

The decompositions of the interaction effects of the other outcomes that show statistically significant total interactions (see Columns 2, 4, 6, and 7 of Panel B) are also similar to the decomposition of general health (Column 1) in the sense that the unexplained part tends to be comparable to the total. We can see occasional contributions from skills and college support but they tend to be relatively small and statistically weak, and they show no consistent pattern across outcomes.

A Limitation of the Decomposition Other mechanisms may contribute to the small "other channels" part in Panel A and large "SES directly" part in Panel B. These channels might be partly due to access to family resources as discussed above, and partly due to incomplete controls for health and skills because of data limitations.

There are several unobserved skills that might add to the estimated contribution of "other channels." First, the EA PGS and its interaction with SES may affect not only early health and early skills, but also risk conceptions in early life, which may not be fully captured by available measures of skills. Recent evidence suggests that a large portion of an individual's risk tolerance is heritable (Linnér, Biroli et al., 2019) and associated with noncognitive skills and risky health behaviors. Second, it is possible that the EA PGS may not only affect risk tolerance itself, but may also affect how individuals evaluate risk

in the first place. Barth et al. (2020) show that an EA PGS predicts whether individuals distinguish the probability of scenarios. Individuals with higher polygenic scores may better understand risks associated with health behaviors. Finally, early Agreeableness and Openness were not available in the data set and could possibly explain a part of the effect through other channels.

Decomposition of the Direct Effect In addition to decomposing the total effect of EA PGS on health-related outcomes, we also decompose the direct effect, the effect that is not explained by education. This estimation is motivated by our criticism of the MR, which we discuss in detail in Section 2. The key innovation here is that we not only find a direct effect of EA PGS on health, such as was found by Belsky et al. (2016) for an effect of an EA PGS on socioeconomic success, but we further explain the direct effect through early skills and health.

This finding makes us more confident about the existence of the direct effect, especially given that these channels can be expected theoretically. When the EA PGS is constructed, some SNPs are selected because they strongly predict early health and skills, which, in turn, strongly predict education. However, early health and skills also predict adult health directly, as we should expect on the basis of theory that we discuss in Section 2. Our decomposition of the direct effect is documented in Table A-5 of the Appendix.

Effects of EA PGS on Cognitive and Noncognitive Skills Our research has a different focus than that by Demange et al. (2020), but we can see that our results related to cognitive and noncognitive skills are consistent with theirs.

The authors construct and study two about equally predictive components of an EA PGS, cognitive and residual noncognitive. Among other results, they find that these two components have the opposite-sign association with Conscientiousness and Extraversion, but both have a negative association with Neuroticism.

We confirm a strong association of the overall EA PGS with cognitive skill, which the authors represent as the cognitive component of an EA PGS. We find that one standard deviation increase in EA PGS is associated with 0.18 standard deviations of additional cognitive skill ($p = 0.000$). We find no effect of the overall EA PGS on Big Five Conscientiousness and Extraversion, which is consistent with canceling effects through cognitive and noncognitive channels. The positive effect on Emotional Stability, which is negative Neuroticism, is consistent with adding positive effects through both cognitive and noncognitive channels. Specifically, one standard deviation increase in EA PGS is associated with a 0.05 standard deviation increase in Emotional Stability (p=0.026) (see Table A-6 of the Appendix).

Evidence for the Effect of Education on Health Table 7, Panel A, shows the marginal effects of educational categories on health-related behaviors implied by the main model (2–7). The signs of estimated effects suggest a health-beneficial role of education for all outcomes. We jointly test whether all effects of educational categories are zero and do not reject the joint hypothesis for each outcome.

Another result of Table 7 is a lack of interaction effects between education and childhood SES in affecting health outcomes. We jointly test interactions between SES and each education level and cannot reject the interaction for each outcome.[21] Therefore, we can conclude that the strong EA PGS-SES interaction that we observe is not generated by stronger effects of education on health for those with higher SES.

A further contribution of Table 7 is that associations between education and health survive conditioning on major genetic confounders: the education PGS, general health PGS factor, and mental health PGS factor. Historically, data on SNPs were part of unobservables in studies of education-health relationships, and controlling for variables that summarize hundreds of relevant SNPs was unimaginable until recently. These results

[21] Because the joint test cannot be rejected for each outcome we do not show individual coefficients and tests for the interactions to save space.

Table 7: Marginal Effects of Education and Health-Related Outcomes in Young Adulthood

	General health (1)	Risky drinking (2)	Smoking tobacco (3)	Marijuana use (4)	Obesity (5)	Lack of exercise (6)	Depression (7)
A. At $\overline{SES}$[(a)]							
Below High School	-0.892 ***	0.068	0.355 ***	0.080 ***	0.031	0.053 *	0.066 *
	(0.175)	*(0.048)*	*(0.039)*	*(0.022)*	*(0.049)*	*(0.030)*	*(0.037)*
High School Diploma	-0.552 ***	0.117 ***	0.238 ***	0.069 ***	0.075 ***	0.080 ***	0.058 ***
	(0.085)	*(0.021)*	*(0.021)*	*(0.012)*	*(0.023)*	*(0.015)*	*(0.018)*
College below Bachelor's	-0.435 ***	0.119 ***	0.227 ***	0.054 ***	0.081 ***	0.057 ***	0.043 **
	(0.099)	*(0.025)*	*(0.023)*	*(0.014)*	*(0.026)*	*(0.017)*	*(0.020)*
College or above	omitted	omitted	omitted	omitted	omitted	omitted	omitted
Joint test[(b)]							
Wald stat.	48.2 ***	33.6 ***	133.2 ***	32.7 ***	13.9 ***	28.1 ***	10.9 **
p-value	0.000	0.000	0.000	0.000	0.003	0.000	0.012
B. Interaction[(c)]							
Joint test[(b)]							
Wald stat.	2.38	5.39	1.15	4.13	1.22	1.90	0.84
p-value	0.497	0.145	0.764	0.248	0.748	0.593	0.840

Notes: Asterisks indicate statistical significance level: ***, 1 % level; **, 5 % level; *, 10 % level. Calculations are based on the main model (2–7) using 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)]Effects of education are calculated at the average level of SES. [(b)]Testing whether all effects are jointly zero, 3 degrees of freedom. [(c)]Three education binary variables, each multiplied by SES. All individual interaction effects are statistically insignificant. We show joint tests only to save space.

contribute to resolving the controversy about the causal effect of education on health.

6 Conclusions

We find that the EA PGS exhibits economically relevant effects on a variety of health outcomes both indirectly through education and directly. However, these effects substantially interact with SES: individuals who grew up in low-SES households do not experience the full health benefits of the EA PGS.

Genes are unlikely to be a policy variable in the foreseeable future due to ethical, political, and practical considerations, but we provide evidence that targeting the SES bottleneck may allow individuals to achieve better health based on their genetic endowment. Based on the evidence here, targeting individuals at the low end of the SES spectrum appears to be an efficient way of boosting health outcomes.

We contribute to an understanding of the mechanisms through which the EA PGS works and provide a model of the mechanisms that can be used to guide future research. We also provide additional evidence that educational attainment has a causal effect on health.

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A Appendix

Table A-1: Measures of Continuous Latent Factors

Conscientiousness	Education support
Gathers facts when solving problems	Parent expects disappointment if the child does not go to college
Thinks of alternative ways to solve problems	Child expects father's disappointment if he/she does not graduate from college
Uses systematic methods when solving problems	Child expects mother's disappointment if he/she does not graduate from college
Analyzes outcome of solutions to problems	Child expects father's disappointment if he/she does not graduate from high school
	Child expects mother's disappointment if he/she does not graduate from high school
Extraversion	Child's own expectation of the likelihood of going to college.
Feels close to people at school	Child's own willingness to go to college
Feels like a part of the school	Child's expectations to graduate from college
Feels socially accepted	
	Physical health PGS
Emotional Stability	Height PGS
Has good qualities	BMI PGS
Has a lot to be proud of	Waist-to-hip ratio PGS
Likes oneself	Coronary artery disease PGS
Feels like doing things right	Myocardial infarction PGS
Feels socially accepted	Low-density lipoprotein cholesterol PGS
Feels loved and wanted	Triglycerides PGS
	Type II diabetes PGS
Cognition	
Add Health Picture Vocabulary Test	Mental health PGS
Recent math grade	Depression PGS
Recent science grade	Neuroticism PGS
	Bipolar disorder PGS
Family SES	Major depressive disorder PGS
Have Billing Problems	Schizophrenia PGS
Use Government Assistance, such as welfare	Mental health cross disorder PGS
Live in Unsafe Neighbourhood	

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, 2011b). 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 a parent and self in wave I.

Table A-2: 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	Obesity	No exercise	Depres-sion
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
EA PGS	-0.052	-0.014	-0.062	-0.012	-0.021	-0.030	0.032
	0.142	0.054	0.050	0.042	0.064	0.060	0.052
EA PGS × SES	-0.186	0.032	-0.022	0.002	-0.030	-0.086	-0.009
	0.115	0.051	0.053	0.038	0.058	0.058	0.042
Number of families	200	200	200	200	200	200	200

Notes: A lack of asterisks in the table corresponds to no statistically significant effects at the 10% level. We exclude identical twins, as they share the same genes 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-3: Robustness Check: The Effect of EA PGS on Education and Its Interaction with SES for Different Sets of Controls, Coefficients of the Ordered Logit Model. The Lack of Interaction Effect is Robust to Different Sets of Controls.

	(1)	(2)	(3)	(4)
EA PGS	0.379 ***	0.374 ***	0.437 ***	0.426 ***
standard error	*(0.035)*	*(0.034)*	*(0.033)*	*(0.032)*
p-value	0.000	0.000	0.000	0.000
SES × EA PGS	-0.018	-0.027	-0.040	-0.036
standard error	*(0.035)*	*(0.034)*	*(0.033)*	*(0.033)*
p-value	0.616	0.428	0.223	0.270
Controls				
SES	✓	✓	✓	✓
Genetic Ancestry, Region, Age, and Sex	✓	✓	✓	
Family Background Controls	✓	✓		
Mental and General Health PGS	✓			

Notes: Model (1) represents the main specification of the reduced-form model (see Equation 1). Models (2-3) are similar models but with different sets of controls denoted by check marks. Asterisks indicate statistical significance level: ***, 1 % level; **, 5 % level; *, 10 % level. Calculations are based on the Add Health data.

Table A-4: Robustness Check: The Effect of EA PGS on Education and Its Interaction with SES for Different Measures of SES, Coefficients of the Ordered Logit Model. The Lack of Interaction Effect is Robust to Different SES Measures.

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
EA PGS	0.379 ***	0.410 ***	0.401 ***	0.502 ***	0.388 ***	0.367 ***	0.414 ***
	(0.035)	*(0.050)*	*(0.049)*	*(0.185)*	*(0.039)*	*(0.040)*	*(0.041)*
Factor score SES × EA PGS	-0.018						
	(0.035)						
Binary SES × EA PGS		-0.048					
		(0.083)					
Index SES × EA PGS			-0.039				
			(0.066)				
Log family income × EA PGS				-0.037			
				(0.049)			
Billing problems × EA PGS					-0.062		
					(0.107)		
Gov. assistance × EA PGS						0.048	
						(0.095)	
Unsafe neighborhood × EA PGS							-0.071
							(0.154)
Controls							
SES measure used for interaction	✓	✓	✓	✓	✓	✓	✓
Full set of controls	✓	✓	✓	✓	✓	✓	✓

Notes: Model (1) represents the main specification of the reduced-form model (see Equation 1). Models (2-7) are similar models but with different measures of SES. Asterisks indicate statistical significance level: ***, 1 % level; **, 5 % level; *, 10 % level. Calculations are based on the Add Health data.

Table A-5: Decomposition of the Direct Effect (Not Through Education) of EA PGS on Health-Related Outcomes with Respect to Possible Mechanisms

	General health (1)	Risky drinking (2)	Smoking tobacco (3)	Marijuana use (4)	Obesity (5)	Lack of exercise (6)	Depression (7)
A. At $\overline{SES}$[(a)]							
Early health	0.019 ***	0.000	-0.002 **	0.000	-0.004 ***	-0.001 **	-0.001 **
	(0.006)	*(0.001)*	*(0.001)*	*(0.000)*	*(0.001)*	*(0.000)*	*(0.001)*
Skills	0.020 **	-0.004 *	-0.005 **	-0.001	-0.001	-0.002	0.000
	(0.009)	*(0.002)*	*(0.002)*	*(0.001)*	*(0.002)*	*(0.001)*	*(0.002)*
College support	0.004	0.000	0.000	0.000	0.001	0.000	0.000
	(0.003)	*(0.001)*	*(0.001)*	*(0.000)*	*(0.001)*	*(0.000)*	*(0.001)*
Other channels	0.044	0.002	-0.031 ***	-0.002	-0.009	0.001	-0.010
	(0.036)	*(0.009)*	*(0.008)*	*(0.004)*	*(0.009)*	*(0.006)*	*(0.007)*
Total direct effect	0.088 **	-0.002	-0.038 ***	-0.004	-0.013	-0.002	-0.012 *
	(0.035)	*(0.008)*	*(0.008)*	*(0.004)*	*(0.009)*	*(0.005)*	*(0.007)*
B. Interaction[(b)]							
Early health	-0.006	0.000	0.000	0.000	0.001	0.000	0.000
	(0.005)	*(0.001)*	*(0.001)*	*(0.000)*	*(0.001)*	*(0.000)*	*(0.001)*
Skills	-0.005	0.002	0.002	0.000	-0.001	0.000	-0.002
	(0.007)	*(0.002)*	*(0.002)*	*(0.001)*	*(0.002)*	*(0.001)*	*(0.001)*
College support	0.002	0.000	0.001	0.000	0.001	0.000	0.001 **
	(0.003)	*(0.001)*	*(0.001)*	*(0.000)*	*(0.001)*	*(0.000)*	*(0.001)*
SES directly	-0.077 **	0.010	-0.002	0.008 **	0.007	0.011 **	0.015 **
	(0.034)	*(0.008)*	*(0.007)*	*(0.004)*	*(0.009)*	*(0.005)*	*(0.006)*
Total direct effect	-0.086 **	0.012	0.001	0.008 **	0.007	0.011 **	0.014 **
	(0.034)	*(0.008)*	*(0.007)*	*(0.004)*	*(0.009)*	*(0.005)*	*(0.006)*

Notes: Asterisks indicate statistical significance level: ***, 1 % level; **, 5 % level; *, 10 % level. Calculations are based on the main model (2–7) using 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)]Effects are calculated at the average level of SES. [(b)]The change of the effect when the adverse SES score increases by one standard deviation.

Table A-6: Effects of EA PGS on Potential Mechanism

	Cognition	Conscien-tiousness	Extraver-sion	Neuroti-cism	Health	College support
	(1)	(2)	(3)	(4)	(5)	(6)
EA PGS	0.176 ***	-0.030	-0.023	0.046 **	0.161 ***	0.081 ***
	(0.017)	*(0.019)*	*(0.019)*	*(0.021)*	*(0.047)*	*(0.018)*
EA PGS × SES	-0.023	0.019	0.000	-0.022	0.003	0.004
	(0.016)	*(0.018)*	*(0.018)*	*(0.018)*	*(0.040)*	*(0.017)*

Notes: Asterisks indicate statistical significance level: ***, 1 % level; **, 5 % level; *, 10 % level. Calculations are based on the main model (2–7) using the Add Health data. Sample size is 3709.