Three Essays in Health Economics

by

Yeongmi Jeong

(Under the Direction of Meghan Skira)

Abstract

In Chapter 1, I examine the effect of adolescent peers' genetic risk for depression on own mental health. I exploit variation in same-gender grademates' genetic risk within schools and across grades. I find that an increase in peers' genetic risk for depression has immediate negative impacts on own mental health, and the effects persist into adulthood for females. As potential mechanisms, I find that an increase in peers' genetic risk for depression in adolescence worsens friendship, increases substance use, and leads to lower socioeconomic status. Overall, the results suggest there are important social-genetic effects in the context of mental health. In Chapter 2, Meghan Skira and I examine whether genetic endowments linked to risky health behaviors explain variation in health. We find that among those aged 50–65, higher genetic predisposition for smoking is associated with worse health, even after controlling for individual smoking behavior and among those who have never smoked, suggesting the genetic endowments correlate with health through non-smoking channels. The genetic endowments for smoking correlate with longevity expectations, planning horizons, and measures of conscientiousness, but these channels do not fully explain the estimated relationship. Furthermore, we find that an increase in a spouse's genetic risk for smoking intensity has adverse spillovers for own health. Overall, our results suggest the genetic factors linked to smoking capture a complex array of traits that correlate with less engagement in health-promoting activities. In Chapter 3, Nicholas Papageorge, Meghan Skira, Kevin Thom, and I find that individuals with higher genetic risk for Alzheimer's Disease (AD) have lower levels of cognition, lower propensities to work, and lower incomes, and these relationships hold even after conditioning on diagnosis of a memory-related disease. However, we also find that individuals at the greatest genetic risk for AD do not have systematically different expectations about their own mortality. Moreover, these individuals are less likely to engage in planning behaviors such as the acquisition of long-term care insurance. These results imply that efforts to encourage end-of-life planning might be particularly beneficial for those at greater risk for needing such services.

Index words: Genetic Endowment, Mental Health, Smoking Behavior, Alzheimer's Disease, Polygenic Score

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Dedication

To my parents, Hee Man Jeong and Bu Rye Lim, who instilled in me a love of learning and a curiosity about the world. Thank you for always believing in me.

To my beloved sister Sang Mi Jeong and my brother-in-law Jong Hun Bak, who supported me through every step of this journey. Your unwavering love and encouragement kept me going when the going got tough.

To my niece Ji Hyeon Bak and Chae Ah Bak, my nephews Seong Ho Bak and Si Yeon Bak, and last but not least, to my most adorable nephew Seong Bin Bak, who lives forever in our hearts. You all inspire me every day to be a better person. Your laughter and smiles light up my life, and I am grateful for the joy you bring me. Your auntie loves you all.

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Contents

List of Figures

List of Tables

Chapter 1

The Effect of Peers' Genetic Predisposition to Depression on Own Mental Health

1.1 Introduction

Depression is one of the most common mental disorders, affecting 17% of adolescents and 8% of adults in the U.S. (NIMH, 2022). Adolescents with depression are about three times more likely to be depressed in adulthood compared to non-depressed adolescents (Johnson et al., 2018). Understanding the determinants of adolescent depression and the role of the adolescent period in shaping later-life mental health is key for informing policy interventions and treatments and curbing the sharp rise in both adolescent and adult depression observed over the last several decades. Motivated by a large body of evidence that suggests peer influence peaks during adolescence, I explore how adolescent peers' underlying risk for depression impacts own mental health.

Specifically, I examine whether peers' genetic predisposition to depression affects one's own mental health in the short- and long-run. Peers' genetic endowments for depression may influence own mental health via peers' depression as well as peers' behaviors (e.g., substance use, interpersonal conflict). I use data from the National Longitudinal Study of Adolescent to Adult Health (Add Health), which follows a nationally representative sample of US adolescents starting in the 1994-95 school year. The genetic data in Add Health include the polygenic risk score for major depressive disorder (hereafter, MDD score), a composite measure of genetic markers that are correlated with MDD. A higher MDD score means a higher genetic risk for depression. I define peers as same-gender grademates and exploit variation in peers' average MDD score within schools and across grades while controlling for own genetic risk for depression.

There are three well-known challenges in identifying the causal effects of peers—the reflection problem, endogenous peer group formation, and common environments. The reflection problem arises when estimating own behavior as a function of average group behaviors because it is impossible to disentangle the effects of average group behaviors (e.g., average peer depression) and average group characteristics (e.g.,

peers' average parental income) on individual behavior (e.g., own depression) since they move together in a linear-in-means model (Manski, 1993). This is not a concern in my case since genes are not affected by others' behaviors or characteristics. Concerns about endogenous peer group formation arise because individuals tend to befriend others who have similar observed and unobserved traits. To address this issue, I rely on cohort-to-cohort variation in the average MDD score within a school (Hoxby, 2000; Hanushek et al., 2003; Angrist and Lang, 2004; Lavy and Schlosser, 2011). While parents might select a school for their children based on observed characteristics, the assignment into each grade within a school is determined by age, making the formation of grademates as good as random. Another challenge arises from the fact that peers share common environments, which may result in similar behaviors and outcomes. While school-grade correlated effects cannot be completely ruled out, I control for school and grade fixed effects and include numerous school-grade level controls to alleviate these concerns.

I find that having same-gender grademates with higher genetic risk for depression during adolescence immediately deteriorates own mental health. A one standard deviation increase in peers' average MDD score significantly increases the likelihood of being depressed by 2.3 and 3 percentage points for adolescent girls and boys, respectively. These effects translate to 8.7% and 20% increases in depression relative to the sample means for female and males, respectively. The peer effects persist into adulthood, but only for females. A one standard deviation increase in peers' average MDD score during adolescence leads to a statistically significant 2.9 percentage point increase in the probability that a female is depressed in adulthood, a 14% increase. These findings suggest that depression in adolescence and adulthood is influenced not only by one's own genetic risk for depression, but also by the genes of those around us. In other words, there are important social-genetic effects in the context of mental health.

The results are robust to alternative measures of depression and specifications, and I provide various pieces of evidence in support of the main identifying assumption that within-school across-grade variation in peers' genetic predisposition to MDD is as good as random. I also explore non-linear effects of peers' genetic predisposition to depression. Both in the short- and long-run, having peer groups with relatively high average MDD scores increases the likelihood of experiencing depression for females, but not males.

Next, I explore mechanisms that could drive the link between peers' genes and own depression, including friendship, substance use, educational attainment, and labor market outcomes. Friendship is a natural mechanism to consider since peers with higher underlying risk for depression may reduce the quantity and quality of social ties. I find that having peers with high genetic risk for depression worsens friendship and social connectedness both in the short- and long-run. For both adolescent girls and boys, an increase in same-gender grademates' average MDD score decreases the probability of spending time with friends. The results also indicate that being exposed to peers with high genetic risk for depression during adolescence reduces the frequency of females hanging out or communicating with friends in adulthood, the number of high school friends females still have as adults, and the number of close friends for both genders in adulthood. Thus, weaker social ties may be an important channel that explains the baseline effects, especially for females.

Substance abuse is often associated with depression, and may be another channel linking peer genetic risk for depression and own depression. I find evidence that having peers with high genetic risk for de-

pression increases substance use, particularly for females. An increase in same-gender grademates' average MDD score increases the frequency of female binge drinking both in adolescence and adulthood, and also generates a slight increase in marijuana use. There is little to no impact on male substance use. Finally, I study effects on socioeconomic outcomes, including college attendance, employment, and labor income. Males who had same-gender grademates with higher genetic risk for depression during adolescence are less likely to attend college, while females are less likely to be employed. The findings indicate that substance abuse and lower socioeconomic status may be additional underlying channels that explain the persistence of the social-genetic effects into adulthood.

This paper contributes to the literature in several ways. First, I add to a growing literature studying peer effects on depression. While a significant correlation between peer and own mental health is well documented in the psychology and medical literatures, only a handful of studies explore causal peer effects on mental health (Eisenberg et al., 2013; Zhang, 2019; Giulietti et al., 2022).[1] Eisenberg et al. (2013) and Zhang (2019) find no significant short-term effects of peers' mental health on own mental health using variation generated from random assignment of college roommates in the US and random assignment of junior secondary school students to classrooms in China, respectively. Different than Eisenberg et al. (2013) and Zhang (2019), I focus on the role of adolescent peers in the US context. Most similar to my work is Giulietti et al. (2022), which examines the long-term effects of peers' depression on own depression using Add Health. They find that an increase in the share of same-gender grademates in adolescence who are depressed significantly increases females' likelihood of being depressed in adulthood. My analysis differs from that of Giulietti et al. (2022) in several ways. First, because I focus on peers' genetic risk for depression, I can identify the contemporaneous effects of adolescent peers on own mental health, whereas Giulietti et al. (2022) cannot identify such effects due to the reflection problem. Second, I explore a wider range of potential mechanisms, including friendship and socialization and substance use. My results complement those of Giulietti et al. (2022) and suggest that social-genetic effects may be an important factor underlying their findings.

This work also contributes to a growing literature on social-genetic effects. The importance of genetics in mental health has been well-recognized (Abkevich et al., 2003; Greene and Vostanis, 2007; NIMH, 2020), but little is known about the indirect effects of the genetic makeup of those around us (i.e., social-genetic effects) (Baud et al., 2017; Domingue and Belsky, 2017; Cawley et al., 2019; Sotoudeh et al., 2019; Cawley et al., 2023). Most studies on social-genetic effects focus on genetically-related groups such as families and relatives.[2] Recently, researchers have examined indirect genetic effects using genetically-unrelated groups such as friends and classmates (Domingue and Belsky, 2017). For instance, Sotoudeh et al. (2019) find a significant causal effect of peers' genetic risk for smoking on own smoking behavior in adolescence. Brunello et al. (2020) explore the short- and long-term effects of peers' genetic risk for body mass index

[1] See De Silva et al. (2005), McPherson et al. (2014), and Ehsan et al. (2019) for a systematic review of the relationship between social capital and mental health, and see Santini et al. (2015) for a literature review of the association between social relationships and depression. There is also a growing literature on social contagion of mental health (e.g., see Bearman and Moody 2004, Fowler and Christakis 2008, Rosenquist et al. 2011, Dishion and Tipsord 2011, and Schwartz-Mette and Smith 2018).

[2] For example, see Kong et al. (2018) and Cawley et al. (2019).

(BMI) on own BMI. They find significant short-term peer effects on BMI for females, with no effects for males. My analysis contributes to this small but growing literature on genomic effects beyond the family. The results imply that there are significant social-genetic effects in the context of mental health, which are stronger among females, consistent with the findings in Brunello et al. (2020).

The remainder of this paper is organized as follows. Section 1.2 includes background information on polygenic scores. Section 1.3 describes the data, sample construction procedure, mental health outcomes, and descriptive statistics. In Section 1.4, I discuss the empirical strategy and identifying assumptions. Section 1.5 reports baseline results, and in Section 1.6, I explore and discuss possible mechanisms underlying the baseline effects. Section 1.7 discusses robustness of the baseline results. I offer some concluding thoughts in Section 1.8.

1.2 Background on Polygenic Scores

As a measure of genetic risk for depression, I use a polygenic score (PGS). A PGS is a linear index of genetic markers that are linked to a particular observable trait or outcome. The calculation of a PGS is based on the results from genome-wide association studies (GWAS), where geneticists run millions of separate linear regressions of the outcome or trait of interest on genetic variants, called single nucleotide polymorphisms (SNPs), conditioning on a set of controls. The PGS is calculated as a weighted sum of the estimated coefficients on each SNP:

$$PGS_i = \sum_{j=1}^{K} \beta_j SNP_{ij} \tag{1.1}$$

where SNP_{ij} is the genotype for individual i at SNP j, and β_j is the effect size for SNP j estimated in the GWAS.[3,4] A higher PGS means that an individual possesses more of the genetic variants associated with that trait or outcome. For example, a higher depression PGS indicates that the individual has a higher genetic risk for depression.

I focus on the PGS related to major depressive disorder (hereafter, the MDD score). Major depressive disorder (MDD), also known as clinical depression, is a common mental disorder characterized by negative feelings such as sadness, emptiness, and hopelessness that can interfere with one's daily activities. The MDD score in the National Longitudinal Study of Adolescent to Adult Health (Add Health) is based on the GWAS by Howard et al. (2019), which identified 102 independent SNPs associated with MDD using a discovery sample of 807,553 individuals of European ancestry. Those 102 genetic markers accounted for 8.9% of the variation in MDD in the discovery sample.[5] In out-of-sample prediction exercises reported

[3] More concretely, SNP_{ij} is the number of instances of the reference allele (zero, one, or two) at SNP j.

[4] See Benjamin et al. (2011) and Beauchamp et al. (2011) for a detailed discussion of the human genome and Barth et al. (2020) and Papageorge and Thom (2020) for a detailed discussion of PGSs. See Braudt and Harris (2020) for details on PGS construction in the Add Health.

[5] In Howard et al. (2019), MDD cases are defined as those who were ever diagnosed with MDD or those with "broad depression" based on self-reported help-seeking behaviors for problems related with nerves, anxiety, tension, or depression.

in Howard et al. (2019), the MDD score explained 1-3% of the variation in depression.[6] In Section 1.3.3, I report the association between the MDD score and depression in the analysis sample.

1.3 Data

The Add Health study follows a nationally representative sample of individuals in the U.S. who were in grades 7-12 during the 1994-95 academic year. Respondents were drawn from a sample of 80 high schools and 52 middle schools stratified according to region, urbanicity, school size, school type (public, private, parochial), ethnic composition, and size. Wave I data was collected in 1994-1995 when respondents were aged 12-20 and contains an in-school questionnaire completed by over 90,000 students who were present at school on the interview day and an in-home questionnaire completed by 20,745 adolescents. The in-home survey respondents from Wave I were followed for four subsequent waves in 1996 (Wave II) when they were aged 13-21, in 2001-02 (Wave III) when they were aged 18-27, in 2008-09 (Wave IV) when they were aged 24-33, and in 2016-18 (Wave V) when they were aged 33-44. The in-home survey includes detailed information on individual characteristics, physical and mental health, parents, family, and school. I use data from the in-home surveys primarily from Waves I and IV.[7] Waves I is used to examine the short-term effects during the adolescent period, and Wave IV is used to explore the long-term effects in adulthood.

The Add Health collected genetic information from Wave IV in-home respondents who agreed to provide a saliva sample.[8] Among the consenting participants, approximately 12,200 respondents agreed to archive their genetic information for long-term use. After quality control procedures for genotyping, 9,974 individuals were eventually genotyped. The Add Health constructed and released a set of PGSs for various diseases and behavioral outcomes. I use the polygenic risk score for major depressive disorder (the MDD score), a composite measure of genetic markers that are correlated with MDD, from the second release of Add Health PGS data. The PGSs in the Add Health are standardized to have a mean of zero and a standard deviation of one within ancestry groups to control for between-group population stratification (Braudt and Harris, 2020).[9,10] A higher MDD score means that the individual has a higher genetic risk for MDD.

[6] Add Health data were not included in the GWAS discovery sample, and therefore not used in the estimation of the β_j coefficients.

[7] The in-school survey contains information on school context, peer networks, and school activities. It was conducted only in Wave I.

[8] Approximately 96% of Wave IV respondents agreed to provide their saliva sample (Braudt and Harris, 2020).

[9] Population stratification refers to differences in genetic variation arising from geographical separation. Geographic isolation leads to mating within the region which, in turn, results in high correlation between genetic variation and geography (Conley and Fletcher, 2017; Hellwege et al., 2017; Braudt and Harris, 2020).

[10] There are four genetic ancestry groups in the Add Health: (1) European ancestry, (2) African ancestry, (3) Hispanic ancestry, and (4) East Asian ancestry (Braudt and Harris, 2020). There is concern about using GWAS results to calculate PGSs for individuals from different ancestry groups, as most discovery samples used in GWAS only include those of European ancestry (Martin et al., 2017, 2019a; Ware et al., 2017a). To address this concern, I normalize the PGSs within each ethnic group and conduct sensitivity analysis separately for each ethnic group following Brunello et al. (2020).

1.3.1 Sample Construction

I construct the analysis sample as follows. First, I select Wave I in-home respondents with non-missing information on school, grade, and race. Second, I restrict the sample to individuals with valid genetic data. Third, I exclude individuals who attended grades with fewer than 10 genotyped same-gender grademates to ensure that there is enough variation in peers' average polygenic score. Then, I use this sample to construct the average MDD score for same-gender grademates in a given school-grade. Finally, I consider Wave I in-home respondents with non-missing information on depression, demographics, family and parental characteristics, and sample weights.[11,12] After the above procedures, 2,335 females and 1,682 males from 91 schools are left for analysis. The average number of same-gender genotyped grademates in one's peer group is 15.8 for females and 17.2 for males.

1.3.2 Mental Health Outcomes

The Add Health in-home survey includes rich information on individuals' mental health. The main outcome I focus on is a binary depression variable based on the Center for Epidemiologic Studies Depression Scale (CES-D scale). The CES-D scale is based on self-reported symptoms of depression and psychological distress, and is widely used in the economics and medical literatures. Waves I, II, and IV of the Add Health include the 10-item version of the CES-D score (hereafter, the CES-D-10 score).[13,14] Several studies have found that the CES-D-10 score is a good screening tool for depression in the adolescent and adult populations (Radloff, 1991; Andresen et al., 1994; Irwin et al., 1999; Bradley et al., 2010). The score ranges from 0 to 30, with higher scores representing more depressed mood. Andresen et al. (1994) recommends cutoffs of 8 and 10 for identifying individuals at risk of depression. Following Suglia et al. (2016) and Giulietti et al. (2022), I take a conservative approach and construct an indicator for experiencing depression that has a value of one if the respondent's CES-D-10 score is greater than or equal to 11, and zero otherwise.[15] Appendix Figure A1 shows histograms of the CES-D-10 score in Waves I and IV by gender. The figures indicate that females' CES-D-10 score distribution has more mass above the threshold of 11 in both Waves I and IV.

I also consider other mental health outcomes, including suicidal ideation and suicide attempts. Information on suicide risk is available in Waves I, II, IV, and V. I create an indicator for suicidal ideation that has a value of one if the respondent has ever seriously considered dying by suicide during the past 12 months, and zero otherwise. Similarly, I construct a suicide attempt indicator that takes on a value of one if the individual has ever actually attempted suicide during the past 12 months. The next two

[11] To avoid losing information when constructing same-gender grademates' average MDD score, I drop individuals with missing values on depression, demographics, family and parental characteristics, and sample weights at the end of sample construction.

[12] Variables included as controls are listed in Section 1.4.

[13] Appendix Table A1 contains the 10 items used to construct CES-D-10 score.

[14] The Wave I in-home survey includes items that also allow me to construct the CES-D-19 score. I conduct a robustness check using the CES-D-19 score as an outcome in column (1) of Table A5.

[15] In Section 1.7.1, I present results using different cutoffs.

mental health measures are available only in Waves III, IV, and V. I create an indicator based on whether respondents reported ever having been diagnosed with depression by a doctor, nurse, or other health care provider. I also construct an indicator that takes on a value of one if the respondent has used any type of antidepressant in the past four weeks and zero otherwise.

1.3.3 Descriptives

Table 1.1 reports summary statistics separately for females and males. In the main analysis, I focus on Wave I for short-term effects and Wave IV for long-term effects.[16] Average ages in Waves I and IV are 16 and 28 regardless of gender, respectively. In both the female and male samples, over 60% of individuals are white. In both samples, over 70% of students' mothers had at least a high school degree, and more than 30% of them had a blue-collar job in Wave I. The number of siblings and household income were also similar across gender. The proportion of individuals whose father was not present in the household was higher for the female than the male sample, but overall, family and parental characteristics in Wave I are similar across the two samples. Generally, depression, suicidal ideation, suicide attempts, depression diagnosis, and antidepressant use are more prevalent among females than males in both waves. This is consistent with clinical findings.

The analysis sample only includes respondents who agreed to provide their genetic data. In Appendix Table A2, I present summary statistics for the non-genotyped sample for comparison.[17] In general, there are more white and fewer Hispanic individuals in my analysis sample. Moreover, females in the genotyped sample exhibit slightly worse mental health than females in the non-genotyped sample, but in most cases the differences are not statistically significant.[18]

The individual MDD score is normalized to have a mean of zero and a standard deviation of one by race and gender. Figure 1.1 displays the distribution of the MDD score in the analysis sample by gender. Both distributions look approximately normal. In the analysis sample, the MDD score explains approximately 0.5% and 0.1% of the total variation in the CES-D-10 score of adult females and males, respectively, and approximately 0.4% and 0.6% of the total variation in diagnosis of depression for adult females and males, respectively.[19]

Same-gender grademates' average MDD score is also normalized to have a mean of zero and a standard deviation of one for each gender. Figure 1.2 shows the distribution of same-gender grademates' average

[16] The attrition from Waves I to IV in my analysis sample is minimal since the genetic data is collected for the Wave IV respondents. To be included in the analysis sample (even for Wave I outcomes) one must appear in Wave IV. I discuss potential attrition bias from Waves I to IV in Section 1.7.4.

[17] There are four types of non-genotyped individuals: those who appeared in Wave IV and refused to provide a saliva sample; those who appeared in Wave IV and provided a saliva sample, but did not agree to archive it for long-term use; those who appeared in Wave IV, provided a saliva sample, and agreed to long-term archiving of the saliva sample, but did not pass quality control; and those who did not appear in Wave IV. Descriptive statistics for the first three types are included in Appendix Table A2.

[18] p-values from a test of equality of means across genotyped and non-genotyped individuals are presented in the last two columns of Appendix Table A2 separately for females and males.

[19] The prediction results are presented in Appendix Tables A3. The MDD score explains approximately 0.3% of the total variation in the binary depression variable (i.e., CES-D-10 score $\geq$ 11) of adult females, but is not predictive of males.

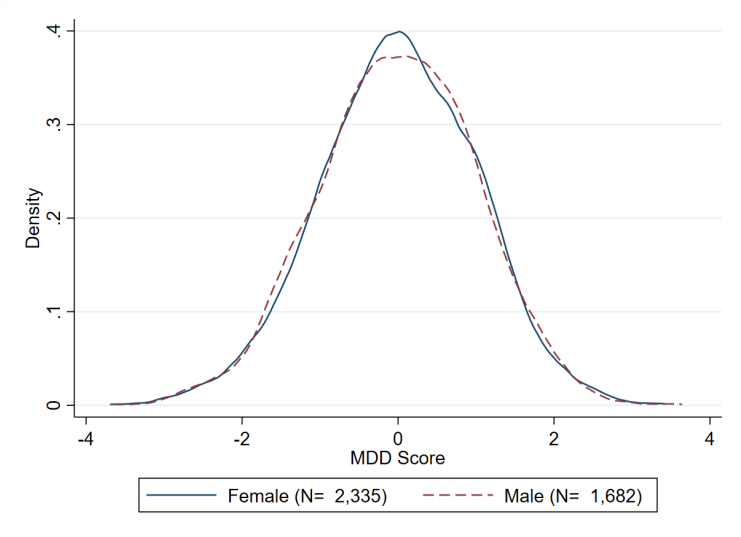
MDD score in the analysis sample by gender. Before the normalization, its standard deviation is one third of the standard deviation of the individual MDD score for both females and males. When interpreting the estimated coefficients, it is important to keep in mind that there is more variation in individual MDD scores than peers' average MDD score.

Table 1.1: Summary Statistics

	Females		Males		*p*-value
	Mean	N	Mean	N	
Demographics:					
Age in Wave I	15.51	2,335	15.80	1,682	0.00
	(1.66)		(1.59)		
Age in Wave II	16.13	1,786	16.45	1,267	0.00
	(1.54)		(1.52)		
Age in Wave III	21.84	2,040	22.16	1,384	0.00
	(1.68)		(1.60)		
Age in Wave IV	28.39	2,331	28.71	1,680	0.00
	(1.70)		(1.65)		
Age in Wave V	37.36	1,791	37.71	1,058	0.00
	(1.83)		(1.75)		
Race: White	0.63	2,335	0.64	1,682	0.41
Race: Black or African-American	0.24	2,335	0.18	1,682	0.00
Race: Asian or Pacific Islander	0.05	2,335	0.09	1,682	0.00
Race: Other	0.07	2,335	0.09	1,682	0.03
Ethnicity: Hispanic	0.10	2,335	0.14	1,682	0.00
Family and Parental Characteristics in Wave I:					
Mother's edu: Missing	0.08	2,335	0.10	1,682	0.18
Mother's edu: High school/some college	0.53	2,335	0.52	1,682	0.84
Mother's edu: College degree or above	0.23	2,335	0.25	1,682	0.11
Mother's occ: Missing	0.05	2,335	0.05	1,682	0.85
Mother's occ: Managerial/professional	0.23	2,335	0.24	1,682	0.45
Mother's occ: Technical/office/sales	0.25	2,335	0.26	1,682	0.25
Mother's occ: Blue collar	0.33	2,335	0.32	1,682	0.20
Father not present	0.31	2,335	0.25	1,682	0.00
Number of siblings	1.47	2,335	1.46	1,682	0.86
	(1.21)		(1.15)		
Household income (imputed, thousands dollars)	44.47	2,335	44.53	1,682	0.96
	(39.55)		(29.07)		
Depression Measures:					
Depressed (CESD-10 $\geq$ 11) in Wave I	0.26	2,335	0.15	1,682	0.00
Depressed (CESD-10 $\geq$ 11) in Wave II	0.25	1,786	0.15	1,267	0.00
Depressed (CESD-10 $\geq$ 11) in Wave IV	0.21	2,331	0.12	1,680	0.00
Suicidal ideation in Wave I	0.16	2,327	0.11	1,671	0.00
Suicidal ideation in Wave II	0.13	1,783	0.08	1,261	0.00
Suicidal ideation in Wave IV	0.08	2,327	0.07	1,665	0.24
Suicidal ideation in Wave V	0.08	1,750	0.07	1,036	0.52
Suicidal attempt in Wave I	0.05	2,327	0.02	1,671	0.00
Suicidal attempt in Wave II	0.05	1,783	0.02	1,261	0.00
Suicidal attempt in Wave IV	0.01	2,328	0.02	1,665	0.19
Suicidal attempt in Wave V	0.02	1,751	0.01	1,035	0.46
Ever diagnosed with depression in Wave III	0.14	2,039	0.07	1,383	0.00
Ever diagnosed with depression in Wave IV	0.21	2,331	0.10	1,680	0.00
Ever diagnosed with depression in Wave V	0.30	1,786	0.17	1,054	0.00
Antidepressant use in Wave III	0.07	2,039	0.02	1,382	0.00
Antidepressant use in Wave IV	0.08	2,323	0.04	1,675	0.00
Antidepressant use in Wave V	0.14	1,783	0.07	1,054	0.00

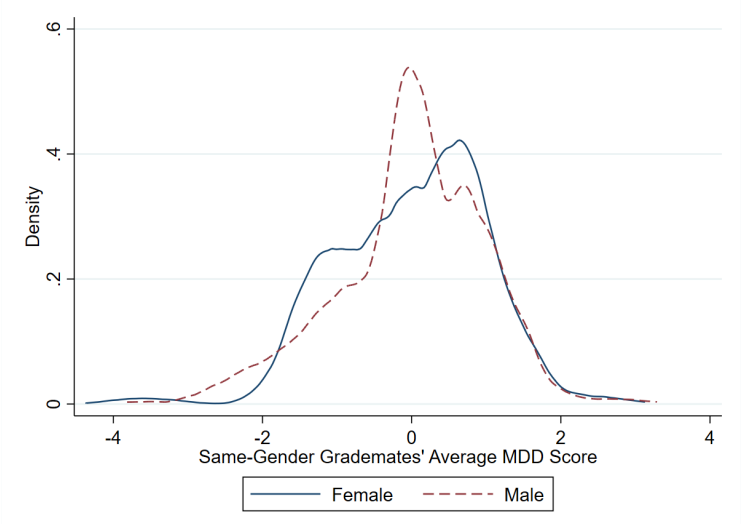
Note: This table shows summary statistics separately for the female and male samples. Demographics and family and parental characteristics are measured in Wave I. The last column includes *p*-values from the test of equality of means across the female and male samples.

Figure 1.1: Individual MDD Score Distribution



Note: This figure displays the distribution of the individual MDD score separately for males (dashed line) and females (solid line).

Figure 1.2: Same-Gender Grademates' Average MDD Score Distribution



Note: This figure displays the distribution of same-gender grademates' average MDD score separately for males (dashed line) and females (solid line).

1.4 Empirical Strategy

I estimate the following baseline specification via ordinary least squares (OLS) regression:

$$\begin{aligned} Y_{isgw} = \quad & \beta_0 + \beta_1 \overline{PGS}_{-isgI} + \beta_2 PGS_{isg} \\ & + \alpha_0 X_{isgI} + \alpha_1 G_{sgI} + \rho_s + \delta_g + \varepsilon_{igsw}, \end{aligned} \tag{1.2}$$

where Y_{isgw} is the measure of mental health of individual i at school s and grade g in wave w, $\overline{PGS}_{-isgI}$ is the average depression PGS of same-gender grademates, excluding individual i, attending the same grade g and school s of individual i in Wave I, PGS_{isg} is the depression PGS of individual i, and X_{isgI} is a set of individual and family characteristics measured in Wave I. G_{sgI} is a set of school-grade specific characteristics measured in Wave I. ρ_s and δ_g are school and grade fixed effects, respectively. Standard errors are clustered at the school level.

Specifically, X_{isgI} includes a set of individual and family characteristics as well as the first 10 principal components of the full SNP matrix. The individual characteristics are age in months dummies, race dummies, a Hispanic origin dummy, and dummies for the number of siblings the respondent has. The family characteristics include household income, mother's education dummies, mother's occupation dummies, and an indicator for father's presence in the household.[20] For mother's education and occupation controls, I include dummies for missing values. Since a nontrivial amount of data on father's education and occupation are missing, I do not include them in the model. Instead, I include an indicator for father's presence in the household following Giulietti et al. (2022). Lastly, I control for the first 10 principal components of the genetic data to minimize the potential bias that arises from within-group population stratification (Price et al., 2006; Belsky et al., 2016; Barth et al., 2020).

G_{sgI} is a set of school-grade specific characteristics measured using information from the wave I in-home survey, which contains average age in months, the proportion of females and proportion in each race category, the share in each category of mother's education and occupation, the share of individuals whose father is present in household, average household income, and grade size. I estimate the baseline specification (equation 1.2) via OLS separately for males and females.[21]

1.4.1 Challenges in Identifying Peer Effects

There are three well-known challenges in identifying peer effects—the reflection problem, endogenous peer group formation, and common environments. First, by using peers' genes, I avoid the reflection problem, which arises from the fact that we cannot separately identify the effects of average group behaviors (e.g., average peer depression) and average group characteristics (e.g., peers' average parental income)

[20] I impute household income with the average household income whenever it is missing and include a dummy variable to indicate that income was imputed.

[21] I do not use peers' average MDD score as an instrument for peers' actual depression (e.g., the share of same-gender grademates with CES-D-10 scores $\geq$ 11) because the exclusion restriction would not be satisfied due to pleiotropy. That is, genetic markers can associate with multiple traits. The MDD-associated SNPs identified in Howard et al. (2019) are also linked to schizophrenia, bipolar disorder, some cardio-metabolic traits, and earlier age of smoking initiation.

on individual behavior (e.g., own depression) in a linear model since they are a function of one another (Manski, 1993). That is, the reflection problem arises when we estimate own behavior as a function of average group behaviors. Since human DNA is determined at conception, does not change over time, and is not affected by others' behavior, my identification strategy does not suffer from this concern.[22]

Second, people tend to befriend others who have similar observed and unobserved traits, which leads to endogenous selection into a peer group. I rely on the widely used approach of defining grademates as one's peer group (Hoxby, 2000; Hanushek et al., 2003; Angrist and Lang, 2004; Lavy and Schlosser, 2011). Although parents might select a school for their children based on observed characteristics, the assignment into each grade within a school is primarily determined by age, which makes it reasonable to assume that the formation of grademates is as good as random.

Third, another challenge arises from exposure to similar environments. Grademates share common environments, which may result in similar behaviors and outcomes and make it difficult to isolate true peer effects. While school-grade correlated effects cannot be completely ruled out, these concerns can be alleviated by controlling for school and grade fixed effects as well as school-grade observables.

1.4.2 Identifying Assumptions

The empirical strategy relies on two main identifying assumptions. I conduct several experiments to examine whether there is support for the assumptions following previous studies (Bifulco et al., 2011; Lavy and Schlosser, 2011; Rodríguez-Planas et al., 2018; Brunello et al., 2020; Olivetti et al., 2020; Giulietti et al., 2022).

First, there needs to be sufficient variation in peers' average MDD score within school and across grades. Each row of Table 1.2 shows the residual standard deviation in same-gender grademates' average MDD score by gender after including various controls. After adding school and grade fixed effects as well as school-grade level controls, the residual standard deviation is about 70% of the raw standard deviation. Thus, there appears to be sufficient variation in the same-gender grademates' average MDD score.

The second identifying assumption is that students were quasi-randomly assigned to a grade within a school. To examine this assumption, I conduct balancing tests, Monte-Carlo simulations, and placebo tests.[23] I start by performing balancing tests. I regress each covariate (e.g., individual MDD score, individual and family characteristics, and 10 genetic principal components) on same-gender grademates' average MDD score conditional on school and grade fixed effects as well as school-grade controls. Table 1.3 reports the estimated coefficients on same-gender grademates' average MDD score, where each cell represents a separate regression. For both samples, the first row reports the estimated coefficients where the individual MDD score is the outcome. I omit oneself when constructing same-gender grademates' average MDD score, which may mechanically lead to a negative correlation between own MDD score and same-gender

[22] In general, individuals' alleles cannot be affected by environmental factors while gene expressions can be altered (i.e., epigenetics) (Cawley et al., 2019).

[23] The experiments are similar to those in Brunello et al. (2020) and Giulietti et al. (2022).

Table 1.2: Standard Deviation of Same-Gender Peers' Average MDD Score

	Females	Males
	SD	SD
Same-gender grademates' average MDD score	1.002	1.001
Same-gender grademates' average MDD score residualized after removing school FE and grade FE	0.740	0.765
Same-gender grademates' average MDD score residualized after removing school FE, grade FE, and school-grade	0.708	0.734
N	2,335	1,682

Note: The first row of this table reports the standard deviation of same-gender grademates' average MDD score by gender. The second row shows the standard deviation of residualized same-gender grademates' average MDD score after controlling for school and grade fixed effects. In row 3, I additionally control for school-grade level controls.

grademates' average MDD score (Guryan et al., 2009; Giulietti et al., 2022).[24,25] In the female and male samples, only 2 and 3 out of 27 regression coefficients are significant at the 5% level or better, which may happen by chance, and the significant characteristics do not overlap across gender except the own MDD score. This suggests no systematic relationship between same-gender grademates' average MDD score and the covariates.

I also perform Monte-Carlo simulations following Lavy and Schlosser (2011), Rodríguez-Planas et al. (2018), and Brunello et al. (2020). The idea is that if students are quasi-randomly assigned into a grade within a school, the variation in average MDD score across grades within a school in the actual sample should be similar to the variation in the average MDD score calculated from randomly assigning peers within a school. For each female (male) in each school, I randomly draw an MDD score using a normal distribution with the school-specific MDD score mean and standard deviation.[26] Then, I compute school-grade specific averages of the simulated female (male) MDD score. For each school, I compute the standard deviation of these averages using residuals from a regression of the simulated female school-grade average MDD score on school and grade fixed effects as well as school-grade level controls. I repeat this procedure

[24] For example, assume that there are four individuals with PGSs of 1, 2, 3, and 4 in a grade. For each of them, the grademates's average PGS (calculated excluding oneself) is 3, 2.667, 2.333, and 2. This means that individual who has the lowest (highest) PGS has the highest (lowest) peer group average PGS.

[25] To verify whether the negative correlation I find is mechanical, I run the balancing tests again after additionally controlling for same-gender schoolmates' average MDD score. Guryan et al. (2009) suggest controlling for the population mean, from where each individual is drawn, to correct this bias. These results are included in Appendix Table A4. The estimated coefficients in row (1) are smaller and no longer statistically significant for females and weakly significant for males after additionally controlling for same-gender schoolmates' average MDD score, which implies that the negative significant correlation that I initially find is mechanical.

[26] The distributions of the actual individual MDD scores by school are approximately normal. I also perform a version of the simulation exercise where I randomly draw an MDD score from the empirical MDD score distribution, and the results are nearly identical.

Table 1.3: Balancing Tests

	Females		Males	
MDD Score	-0.381***	(0.109)	-0.557***	(0.128)
Age in months	-0.247	(0.265)	-0.254	(0.481)
Race: White	0.003	(0.017)	-0.025	(0.022)
Race: Black	0.006	(0.013)	0.015	(0.015)
Race: Asian	0.009	(0.007)	0.009	(0.010)
Hispanic	-0.010	(0.010)	-0.003	(0.009)
Number of siblings	0.036	(0.030)	-0.013	(0.045)
Father not in household	-0.012	(0.020)	-0.004	(0.029)
Household income (imputed)	2.066	(2.142)	-0.460	(1.186)
Household income (missing)	0.026**	(0.011)	-0.018	(0.013)
Mother's edu: Missing	-0.017	(0.012)	-0.017	(0.017)
Mother's edu: High school graduate/some college	0.009	(0.022)	0.030	(0.039)
Mother's edu: College graduate and above	0.007	(0.018)	-0.004	(0.037)
Mother's occ: Missing	-0.015	(0.010)	-0.031**	(0.012)
Mother's occ: Managerial/professional	0.012	(0.023)	0.018	(0.023)
Mother's occ: Technical/office/sales	-0.006	(0.018)	0.023	(0.025)
Mother's occ: Blue collar	0.019	(0.026)	0.020	(0.030)
PC1	-0.001	(0.000)	-0.000	(0.001)
PC2	-0.001	(0.001)	0.001	(0.000)
PC3	-0.000	(0.001)	0.000	(0.001)
PC4	-0.000	(0.001)	0.000	(0.001)
PC5	0.000	(0.001)	-0.001	(0.001)
PC6	0.000	(0.001)	-0.000	(0.001)
PC7	0.000	(0.001)	0.000	(0.001)
PC8	-0.001	(0.001)	-0.001	(0.001)
PC9	-0.000	(0.000)	-0.001	(0.001)
PC10	0.001	(0.001)	0.001***	(0.000)
N	2,335		1,682	

Note: Each row includes coefficients from a separate regression of a covariate on same-gender grademates' average MDD score conditional on school and grade fixed effects, and school-grade level controls. Standard errors are clustered at the school level and included in parenthesis. All individual and family level characteristics are measured in Wave I, and all genetic information (i.e., MDD PGS and PC1-10) is collected in Wave IV. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

1,000 times and construct a 90% empirical confidence interval for the simulated within-school standard deviations.[27] I find that approximately 88% and 89% of the schools' actual standard deviation falls within

[27] I calculate the actual standard deviation of school-grade specific averages similarly. That is, for each school, I compute the standard deviations of the actual school-grade specific averages of MDD score using residuals from a regression of the actual female (male) school-grade average MDD score on school and grade fixed effects as well as school-grade level controls.

Table 1.4: Effect of Same-Gender Grademates' and Own MDD Score on Mental Health in Wave I

	Depressed (CES-D-10 $\geq$ 11)							
	Females				Males			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Same-Gender Grademates' Average MDD Score	-0.004	0.017*	0.025**	0.023**	0.001	0.024	0.031*	0.030**
	(0.009)	(0.009)	(0.011)	(0.010)	(0.009)	(0.015)	(0.016)	(0.015)
Own MDD Score	0.019**	0.023***	0.023**	0.018**	0.006	0.012	0.014	0.013
	(0.008)	(0.008)	(0.009)	(0.009)	(0.010)	(0.010)	(0.010)	(0.010)
Principal Components	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
School and Grade FEs	No	Yes	Yes	Yes	No	Yes	Yes	Yes
School-Grade Controls	No	Yes	Yes	Yes	No	Yes	Yes	Yes
Individual Controls	No	No	Yes	Yes	No	No	Yes	Yes
Family and Parental Controls	No	No	No	Yes	No	No	No	Yes
Mean	0.262	0.262	0.262	0.262	0.150	0.150	0.150	0.150
N	2,335	2,335	2,335	2,335	1,682	1,682	1,682	1,682
R^2	0.005	0.069	0.112	0.132	0.007	0.060	0.132	0.147

Note: The outcome is an indicator for being depressed (i.e., CES-D-10 $\geq$ 11) in Wave I and each column includes separate regression results from various specifications. Columns (1)-(4) and (5)-(8) present the point estimates for females and males, respectively. In columns (1) and (5), I show results after controlling for the 10 genetic principal components. Then, I add school and grade fixed effects and school-grade level controls (columns 2, 6). Next, I include a set of individual controls in columns (3) and (7). Then, I add a set of family and parental controls (columns 4, 8). Standard errors are clustered at the school level. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

the 90% empirical confidence interval for both the female and male samples.[28] These results support the assumption of random assignment.

1.5 Main Results

In this section, I present estimates of the effects of peers' genetic predisposition to depression on own mental health in the short- and long-term. The results suggest that there is an immediate adverse effect of peers' genetic predisposition to depression on own mental health, which persists into adulthood for females, but not males.

[28]Appendix Figure A2 displays distributions of the actual and simulated school-specific MDD score standard deviations separately for the female and male samples.

1.5.1 Short-Term Effects

I first examine the short-term effects of same-gender grademates' average MDD score on mental health as measured by an indicator for being depressed (i.e., CES-D-10 score $\geq$ 11). Data from Wave I is used to estimate these short-run effects.[29,30]

Each column of Table 1.4 contains separate regression results from various specifications. Columns (1)-(4) and (5)-(8) show point estimates for females and males, respectively. In columns (1) and (5), I control only for the genetic principal components. In columns (2) and (6), I add school and grade fixed effects and school-grade level controls. In columns (3) and (7), I additionally include individual-level controls. Columns (4) and (8) contain the results from the most exhaustive specification that additionally includes family and parental controls. This is my preferred specification going forward.

The estimate in column (4) indicates that a one standard deviation increase in peers' average MDD score significantly increases the likelihood of being depressed by 2.3 percentage points for adolescent girls, an 8.7% increase relative to the sample average. In column (8), I find a one standard deviation increase in same-gender grademates' average MDD score leads to a 3 percentage point increase in the probability of being depressed for adolescent boys, a 20% increase relative to the sample average.

Taken together, the estimates suggest there are immediate social-genetic effects on mental health. An increase in same-gender grademates' genetic risk for depression exerts short-run adverse impacts on own mental health for both females and males. The effect size is similar across gender in percentage point terms, though larger for males in percentage terms.[31] Notably, the effects of one's own MDD score on the probability of being depressed is positive for both genders but statistically significant only for girls.

The results are consistent with existing evidence on gender differences in peer effects on mental health. In particular, Giulietti et al. (2022) document significant peer effects on depression for females during adolescence, but no significant short-term effects for males.[32] They find that a one standard deviation increase in the share of same-gender grademates in adolescence who are depressed increases females' likelihood of being depressed the following 1-2 years (i.e., in Wave II) by 2.9 percentage points (an 11.8% increase). The social-genetic effects on females I find are about three-quarters the size of their estimated peer effects. The peer effects found in Giulietti et al. (2022) may, therefore, be partly explained by social-genetic effects.[33]

[29] In Waves I and II, most of the respondents are adolescents, and all of them are adults by Wave III.

[30] Most of the respondents who were in the 12^{th} grade in Wave I were not included in the in-home survey sample in Wave II. Therefore, I focus on the short-term results using Wave I data.

[31] I fail to reject equality of the coefficients on same-gender grademates' average MDD score across genders.

[32] They fail to reject equality of the coefficients of interest across genders.

[33] While Giulietti et al. (2022) did not find evidence of short-run peer effects on male mental health, my results suggest substantial evidence of social-genetic effects for males. The difference in results is likely explained by the difference in our main explanatory variables. I rely on peers' genetic predisposition to depression, while Giulietti et al. (2022) rely on peers' self-reported depressive symptoms (i.e., the CES-D-10 score).

Table 1.5: Effect of Same-Gender Grademates' and Own MDD Score on Mental Health in Wave IV

	Depressed (CES-D-10 $\geq$ 11)							
	Females				Males			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Same-Gender Grademates' Average MDD Score	0.007	0.029**	0.029**	0.029**	0.009	0.019**	0.018*	0.016
	(0.009)	(0.013)	(0.013)	(0.013)	(0.007)	(0.009)	(0.010)	(0.010)
Own MDD Score	0.022***	0.027***	0.026***	0.024***	0.006	0.008	0.010*	0.010
	(0.007)	(0.007)	(0.007)	(0.007)	(0.007)	(0.007)	(0.006)	(0.006)
Principal Components	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
School and Grade FEs	No	Yes	Yes	Yes	No	Yes	Yes	Yes
School-Grade Controls	No	Yes	Yes	Yes	No	Yes	Yes	Yes
Individual Controls	No	No	Yes	Yes	No	No	Yes	Yes
Family and Parental Controls	No	No	No	Yes	No	No	No	Yes
Mean	0.206	0.206	0.206	0.206	0.124	0.124	0.124	0.124
N	2,331	2,331	2,331	2,331	1,680	1,680	1,680	1,680
R^2	0.010	0.066	0.109	0.119	0.004	0.061	0.106	0.125

Note: The outcome is an indicator for being depressed (i.e., CES-D-10 $\geq$ 11) in Wave IV, and each column includes separate regression results from various specifications. Columns (1)-(4) and (5)-(8) present the point estimates for females and males, respectively. In columns (1) and (5), I show results after controlling for the 10 genetic principal components. Then, I add school and grade fixed effects and school-grade level controls (columns 2,6). Next, I include a set of individual controls in columns (3) and (7). Then, I add a set of family and parental controls (columns 4, 8). Standard errors are clustered at the school level. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

1.5.2 Long-Term Effects

Next, I explore whether the short-term peer effects found in the prior section persist into adulthood. I focus on mental health in Wave IV when respondents are aged 28-33 and consider the same indicator variable for depression (i.e., CES-D-10 score $\geq$ 11).[34]

In Table 1.5, I present estimated effects of same-gender grademates' average MDD score on the likelihood of experiencing depression in Wave IV. Columns (1)-(4) contain the results for females. Across most of the specifications, a one standard deviation increase in peers' average MDD score leads to a statistically significant 2.9 percentage point increase in the probability of being depressed, a 14% increase from the sample average. Columns (5)-(8) display the results for males. In the specifications with the most rigorous

[34]I focus on Wave IV for the long-term results for two reasons. First, the CES-D-10 score is not available in Waves III or V. Second, it is the first wave where all of the respondents are over 20 years old. In Wave III, the respondents are aged 18-27.

set of controls (column 8), I find a positive effect of same-gender grademates' average MDD score on the probability of being depressed, but it is not significant at conventional levels.[35]

Earlier I found that peers' genetic risk for depression has an immediate negative impact on own mental health for both females and males. The findings in this section imply that the social-genetic effects on mental health in adolescence carry over into adulthood for females, but not males. My findings are in line with the evidence on gender differences in depression, which documents that females exhibit earlier onset of and a higher rate of depression, and that the gender gap continues throughout life (Mirowsky, 1996; Piccinelli and Wilkinson, 2000; Patten et al., 2001; Lewis et al., 2015; Breslau et al., 2017; Salk et al., 2017; Bogren et al., 2018). Giulietti et al. (2022) find that a one standard deviation increase in the share of same-gender grademates who are depressed during adolescence increases females' likelihood of being depressed by 2.6 percentage points (an 11.7% increase) in adulthood. My results suggest socio-genetic effects may explain, in part, their long-term effects.

One explanation for the more persistent peer effects on depression of females relates to different response styles to stress by gender. The response styles theory (RST) says females are more likely to repeatedly think about negative feelings and problems (i.e., ruminate and internalize) while males tend to deal with them by problem-solving (i.e., externalize) (Nolen-Hoeksema, 1987, 1991; Hilt et al., 2010; Johnson and Whisman, 2013). Giulietti et al. (2022) show that co-rumination, which means having excessive discussion about problems or concerns with others, is prevalent among adolescent girls, and may explain the gender difference in peer effects on depression. Evidence from the psychopathology field suggests that gender differences in reactivity to stress leads to gender differences in vulnerability to anxiety and depression, which becomes noticeable during adolescence and continues through adulthood (Rudolph, 2002). If adolescent females with peers who are more genetically predisposed to depression tend to ruminate and and co-ruminate, which in turn makes them more vulnerable to depression, this may partially explain the effects I find.

In addition, the scarring effects for females of exposure to peers during adolescence with a higher underlying risk for depression is consistent with the evidence in economics and social psychology that females are more vulnerable to peer influence (Eagly, 1978; Minton and Schneider, 1980; Han and Li, 2009).[36] Moreover, the long-lasting importance of one's adolescent experience throughout the life course is well-recognized since adolescence is a period in which individuals develop social and noncognitive skills (Alwin and Krosnick, 1991; Gong et al., 2020). Thus, the effects of peer influence during this period may be especially salient and long-lasting.

[35] To examine across-gender peer effects, I reestimate the baseline specification additionally including other-gender grademates' average MDD score. For females, the coefficient on other-gender grademates' genetic risk is not statistically significant in the short- and long-term. For males, the short-term effect is not precisely estimated while the long-term effect is significant.

[36] Descriptive results using the Add Health also support the notion that females are more susceptible to peer influence. The Add Health asks Wave III respondents whether they agree or disagree that in social situations they tend not to follow the crowd. Approximately 10% of females disagreed or strongly disagreed whereas approximately 7% of males disagreed or strongly disagreed. I reject equality of the two at the 5% level.

1.6 Mechanisms

I find significant short-term social-genetic effects, which carry over into adulthood for females but not for males. I explore several possible mechanisms underlying these effects. Peers' genes may influence own short- and long-term depression through peers' depression as well as behavioral outcomes such as friendship, substance use, and socioeconomic status. I investigate the latter three in this section.

1.6.1 Friendship and Socialization

During adolescence, individuals seek social acceptance and show an increased sensitivity to peers' reactions (Brown and Larson, 2009; Andrews et al., 2020; Giulietti et al., 2022). Moreover, the importance of friendship (or peer support) for mental health during adolescence is widely studied and recognized (Reisman, 1985; Ueno, 2005; Sias and Bartoo, 2007; King and Terrance, 2008; King et al., 2016; Cleary et al., 2018; Narr et al., 2019). Peers with high genetic risk for depression may themselves have higher prevalence of depression, which may negatively influence their friendship. Worse friendships may, in turn, deteriorate own mental health. It could also be that individuals with high genetic risk for depression tend to emotionally and physically detach themselves from friends. Then, having peers with high genetic risk for depression may have a negative impact on the quality and/or quantity of friendships, which may lead to worse mental health. I investigate whether peers' genetic risk for depression impacts friendship.

Short-Term Effects on Friendship

In Wave I, the Add Health asks questions about interactions with friends, such as visiting friends' houses, hanging out with friends after school, and spending time with friends during the weekend.[37]

Table 1.6 reports estimated results where the outcome variables take on a value of one if respondents did the corresponding activities (i.e., visit a friend's home, hang out after school, spend time together during the weekend) with one or more of their nominated friends during the past week. The coefficients on same-gender grademates' average MDD score are all negative. For females, there is a statistically significant decline in the probability of visiting friends' homes and hanging out after school. For males, there is a significant decrease in the likelihood of spending time with friends during the weekend. While the above-mentioned outcomes capture physical detachment from friends, I also explore emotional connectedness to friends and one's school in Appendix Tables A9 and A10, respectively. Although the estimates are not precisely estimated, the results imply a decrease in emotional attachment to friends and school.[38,39]

[37] Specifically, respondents can nominate up to five best friends. They are then asked: "Did you go to [friend 1,...,5]'s house during the past seven days?", "Did you meet [friend 1,...,5] after school to hang out or go somewhere during the past seven days?", and "Did you spend time with [friend 1,...,5] during the past weekend?" Respondents answer yes or no.

[38] I consider sense of belonging at school since friendship plays an important role in developing adolescents' sense of belonging (Hamm and Faircloth, 2005).

[39] I use the following items to measure emotional connectedness to friends: "Did you talk to [friend 1,...,5] about a problem during the past seven days?", "Did you talk to [friend 1,...,5] on the telephone during the past seven days?", and "How much do you feel that your friends care about you?" For the first two items, respondents answer yes or no. For the last item, respondents choose from five categories: not at all; very little; somewhat; quite a bit; and very much, and I create two indicator variables

Table 1.6: Effect of Same-Gender Grademates' and Own MDD Score on Friendship in Wave I

	During the Past Week or Weekend					
	Visit Friends' Home		Hang out After School		Spend Time Together	
	(1) Females	(2) Males	(3) Females	(4) Males	(5) Females	(6) Males
Same-Gender Grademates' Average MDD Score	-0.022*	-0.011	-0.032**	-0.021	-0.004	-0.047***
	(0.013)	(0.017)	(0.015)	(0.013)	(0.019)	(0.016)
Own MDD Score	-0.012	-0.008	0.004	0.002	0.001	-0.010
	(0.011)	(0.013)	(0.009)	(0.010)	(0.010)	(0.011)
Mean	0.599	0.690	0.651	0.700	0.632	0.687
N	2,277	1,637	2,277	1,636	2,277	1,637
R^2	0.165	0.189	0.154	0.187	0.145	0.167

Note: The outcome variable in columns (1) and (2) is an indicator for whether respondents visit one or more of their nominated friends' houses during the past week. The outcome variable in columns (3) and (4) is an indicator for whether respondents hang out with one or more of their nominated friends during the past week. The outcome variable in columns (5) and (6) is an indicator variable for whether respondents spend time with one or more of their nominated friends during the past weekend. All regressions include the controls in my most preferred specification (columns 4 and 8 of Table 1.4). Standard errors are clustered at the school level. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Taken together, the findings indicate that an increase in same-gender grademates' average MDD score has an immediate negative impact on friendship. Thus, weaker friendships due to less interaction with friends may be an important channel through which the short-term effects operate.

Long-Term Effects on Friendship

I consider friendship-related measures in Waves III and IV to explore the enduring effects of peers' depression genes. The Add Health asks respondents "In the past seven days, how many times did you just hang out with friends, or talk on the telephone for more than five minutes?" and "Thinking back to all your friends from high school, how many are you still friends with?" in Wave III, and "How many close friends

for whether respondents feel that their friends care about them quite a bit or more and very much, respectively. As measures of sense of belonging at school, I use questions asking how strongly one agrees or disagrees with the following: "I feel close to people at this school", "I feel like I am part of this school", and "I am happy to be at this school." Respondents choose from five categories: strongly agree; agree; neither agree nor disagree; disagree; and strongly disagree. I construct three indicators for whether individuals strongly disagree or disagree with 1 or more, 2 or more, and 3 of those statements.

Table 1.7: Effect of Same-Gender Grademates' and Own MDD Score on Friendship in Waves III or IV

	Wave III						Wave IV			
			Number of High School Friends Still Friends With				Number of Close Friends			
	Times Hang Out During Past Week		Most or More		All		3 to 5 Friends or More		6 to 9 Friends or More	
	(1) Females	(2) Males	(3) Females	(4) Males	(5) Females	(6) Males	(7) Females	(8) Males	(9) Females	(10) Males
Same-Gender Grademates' Average MDD Score	-0.149**	-0.205*	-0.331	-0.658	-0.542**	0.750**	0.004	-0.009	-0.039**	-0.028*
	(0.068)	(0.104)	(0.512)	(0.946)	(0.258)	(0.332)	(0.017)	(0.021)	(0.016)	(0.015)
Own MDD Score	-0.146***	0.035	-0.094	-0.256	-0.139*	0.229**	-0.010	-0.006	-0.027***	-0.004
	(0.047)	(0.066)	(0.154)	(0.278)	(0.074)	(0.098)	(0.009)	(0.010)	(0.010)	(0.011)
Mean	4.322	4.214	0.283	0.474	0.046	0.052	0.714	0.745	0.235	0.316
N	2,034	1,380	389	232	389	232	2,298	1,651	2,298	1,651
R^2	0.150	0.212	0.284	0.463	0.266	0.577	0.169	0.150	0.142	0.123

Note: Columns (1)-(6) and (7)-(10) contain results from Waves III and IV, respectively. The outcome variable in columns (1)-(2) is the frequency of hanging out with friends or talking on on the phone during the past week. The outcome variables in columns (3)-(4) and (5)-(6) are indicator variables for whether the number of high school friends that respondents still have as adults is "most or more" and "all", respectively. The outcomes in columns (7)-(8) and (9)-(10) are indicator variables for whether the number of close friends is 3 to 5 friends or more and 6 to 9 friends or more, respectively. All regressions include the controls in my most preferred specification (columns 4 and 8 of Table 1.4). Standard errors are clustered at the school level. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

do you have?" in Wave IV.[40,41] For the number of high school friends one still has as an adult question, I create two dummies that have a value of one if the respondent answers that the corresponding number of friends is most or more, and all, respectively, and zero otherwise. For the number of close friends question, I create two indicator variables that take a value of one if the respondent answers that the number of close friends is three or more and six or more, respectively, and zero otherwise.

Table 1.7 includes estimated results for Waves III and IV. The results indicate that an increase in same-gender grademates' average genetic risk score for depression negatively affects the frequency of hanging out or talking on the phone, decreases the probability of still being friends with all of one's high school friends in adulthood, and decreases the probability of having many (i.e., 6 or more) close friends for adult females. For adult males, the point estimates are mostly negative, but not significant.

Overall, having peers with high genetic risk for depression during adolescence has negative impacts on friendship in the short- and long-term. The effects are more pronounced for females than males. Weaker social ties may be an important channel that explains the long-term baseline effects for females.

[40]When answering the number of high school friends the respondent still has as an adult question, they choose among none, one, a few, some, most, or all. This question was asked only to the Wave III respondents who were in 7^{th} and 8^{th} grades in Wave I.

[41]For the number of close friends question, respondents choose among none, one or two friends, three to five friends, six to nine friends, ten or more friends. According to the Add Health documentation, close friends include people whom we feel at ease with, can talk to about private matters, and can call on for help.

Another possible pathway could be that being exposed to peers with high genetic risk for depression during adolescence reduces the likelihood of females joining new peer groups in adulthood, which possibly leads to isolation and worse mental health. However, due to lack of detailed data on adulthood friendships, I cannot directly explore this path.

1.6.2 Substance Use

There is growing evidence regarding peer effects on substance use (Lundborg, 2006; Clark and Lohéac, 2007; Cawley and Ruhm, 2011; Eisenberg et al., 2014). Moreover, studies in epidemiology and economics suggest a negative correlation between substance use and mental health, although the causal link is unclear (Jane-Llopis and Matytsina, 2006; Swendsen et al., 2010; Van Ours and Williams, 2011, 2012; Lipari and Van Horn, 2017; Conway et al., 2018; Friedman, 2020). Some people may use substances such as alcohol, tobacco, and marijuana to relieve mental distress (Cornah, 2006; Stapinski et al., 2016; Friedman, 2020). On the other hand, substance use may lead to poor mental health by increasing anxiety and tension over time (Taylor et al., 2014; Plurphanswat et al., 2017; Taylor et al., 2021).

If peers with high genetic risk for depression are more likely to use substances, interacting with them during adolescence may increase own substance use, which may trigger or intensify a deterioration of own mental health. To test this hypothesis, I examine whether same-gender grademates' average MDD score affects own use of alcohol, tobacco, and marijuana.[42]

Alcohol

The Add Health asks respondents "During the past 12 months, on how many days did you drink alcohol?", "Over the past 12 months, on how many days did you drink five or more drinks in a row?", and "Over the past 12 months, on how many days have you gotten drunk or very, very high on alcohol?"[43] For each question, I create indicator variables that have a value of one if the respondent answers that the corresponding number of days is once a month or more, 2 to 3 days a month or more, or 1 to 2 days a week or more, respectively, and zero otherwise.

Table 1.8 reports results from Wave I. The results in Panels A and B imply that the effects of same-gender grademates' average MDD score on frequency of alcohol consumption, especially binge drinking, are positive for females. For males, there is some evidence of an increase in the frequency of binge drinking. In Table 1.9, I present results from Wave IV. The estimates generally indicate that having same-gender grademates' with higher genetic risk for depression during adolescence causes more frequent alcohol use among adult females. For adult males, I find the opposite—an increase in same-gender grademates' average MDD score during adolescence negatively affects alcohol use. Overall, the findings suggest that having

[42] According to the Centers for Disease Control and Prevention (CDC), alcohol, marijuana, and tobacco are the most commonly used substances by adolescents and they are known to be closely related to mental health problems (Gart and Kelly, 2015; Conway et al., 2018; Choudhury, 2021).

[43] For these questions, individuals choose among none, one or two days in the past 12 months, once a month or less, two to three days a month, one to two days a week, three to five days a week, everyday or almost everyday.

Table 1.8: Effect of Same-Gender Grademates' and Own MDD Score on Alcohol Use in Wave I

	During the Past 12 Months					
	Once a Month or More		2 or 3 Days a Month or More		1-2 Days a Week or More	
	Panel A: Days Drink Alcohol					
	(1) Females	(2) Males	(3) Females	(4) Males	(5) Females	(6) Males
Same-Gender Grademates' Average MDD Score	0.019	-0.002	0.025*	0.012	0.012**	0.001
	(0.018)	(0.012)	(0.013)	(0.018)	(0.006)	(0.014)
Own MDD Score	0.014	0.020*	0.010	0.010	0.006	0.009
	(0.010)	(0.010)	(0.009)	(0.010)	(0.006)	(0.008)
Mean	0.272	0.322	0.148	0.199	0.067	0.117
N	2,324	1,672	2,324	1,672	2,324	1,672
R^2	0.176	0.197	0.157	0.188	0.117	0.159
	Panel B: Days Drink 5 or More Drinks in a Row					
	(1) Females	(2) Males	(3) Females	(4) Males	(5) Females	(6) Males
Same-Gender Grademates' Average MDD Score	0.028**	0.014	0.024**	0.026**	0.011**	0.006
	(0.012)	(0.013)	(0.010)	(0.011)	(0.006)	(0.008)
Own MDD Score	0.005	0.005	0.004	0.004	0.002	-0.005
	(0.009)	(0.015)	(0.008)	(0.010)	(0.004)	(0.006)
Mean	0.130	0.213	0.076	0.144	0.039	0.084
N	2,321	1,667	2,321	1,667	2,321	1,667
R^2	0.150	0.193	0.129	0.179	0.110	0.149
	Panel C: Days Get Drunk					
	(1) Females	(2) Males	(3) Females	(4) Males	(5) Females	(6) Males
Same-Gender Grademates' Average MDD Score	0.018	0.009	0.004	0.012	-0.005	-0.000
	(0.013)	(0.013)	(0.008)	(0.011)	(0.005)	(0.009)
Own MDD Score	-0.000	0.010	-0.005	-0.005	-0.004	0.001
	(0.008)	(0.010)	(0.007)	(0.008)	(0.004)	(0.006)
Mean	0.125	0.188	0.071	0.118	0.029	0.064
N	2,322	1,667	2,322	1,667	2,322	1,667
R^2	0.177	0.174	0.124	0.174	0.092	0.153

Note: The outcome variable in columns (1) and (2) is an indicator variable for whether an individual used alcohol once a month or more during the past year in Wave I. The outcomes in columns (3)-(4) and (5)-(6) are created similarly for two or three days a month and one or two days a week, respectively. All regressions include the controls in my most preferred specification (columns 4 and 8 of Table 1.4). Standard errors are clustered at the school level. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table 1.9: Effect of Same-Gender Grademates' and Own MDD Score on Alcohol Use Wave IV

	During the Past 12 Months					
	Once a Month or More		2 or 3 Days a Month or More		1-2 Days a Week or More	
	Panel A: Days Drink Alcohol					
	(1)	(2)	(3)	(4)	(5)	(6)
	Females	Males	Females	Males	Females	Males
Same-Gender Grademates' Average MDD Score	0.045***	-0.041**	0.029*	-0.035*	0.024*	0.008
	(0.015)	(0.016)	(0.016)	(0.019)	(0.015)	(0.015)
Own MDD Score	-0.011	-0.009	0.001	-0.017	0.009	-0.019
	(0.010)	(0.011)	(0.011)	(0.012)	(0.008)	(0.012)
Mean	0.536	0.684	0.355	0.548	0.196	0.378
N	2,322	1,671	2,322	1,671	2,322	1,671
R^2	0.188	0.203	0.168	0.170	0.146	0.165
	Panel B: Days Drink 5 or More Drinks in a Row					
	(1)	(2)	(3)	(4)	(5)	(6)
	Females	Males	Females	Males	Females	Males
Same-Gender Grademates' Average MDD Score	0.030**	-0.042**	0.003	-0.026	0.010	0.007
	(0.013)	(0.018)	(0.009)	(0.022)	(0.008)	(0.013)
Own MDD Score	0.002	0.003	0.001	0.001	-0.002	0.012
	(0.009)	(0.013)	(0.008)	(0.015)	(0.006)	(0.010)
Mean	0.225	0.373	0.135	0.254	0.061	0.157
N	2,319	1,667	2,319	1,667	2,319	1,667
R^2	0.139	0.173	0.112	0.145	0.098	0.118
	Panel C: Days Det Drunk					
	(1)	(2)	(3)	(4)	(5)	(6)
	Females	Males	Females	Males	Females	Males
Same-Gender Grademates' Average MDD Score	0.029**	-0.061***	0.013*	-0.037**	0.008	-0.007
	(0.011)	(0.019)	(0.007)	(0.018)	(0.006)	(0.012)
Own MDD Score	0.004	-0.014	0.005	-0.011	0.005	-0.001
	(0.007)	(0.014)	(0.005)	(0.011)	(0.004)	(0.008)
Mean	0.174	0.333	0.082	0.190	0.035	0.099
N	2,320	1,669	2,320	1,669	2,320	1,669
R^2	0.156	0.187	0.123	0.158	0.091	0.132

Note: The outcome variable in columns (1) and (2) is an indicator variable for whether an individual used alcohol once a month or more during the past year in Wave IV. The outcomes in columns (3)-(4) and (5)-(6) are created similarly for two or three days a month and one or two days a week, respectively. All regressions include the controls in my most preferred specification (columns 4 and 8 of Table 1.4). Standard errors are clustered at the school level. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

peers with high genetic risk for depression during adolescence leads to increased use of alcohol for females both in adolescence and adulthood.

Tobacco

A strong correlation between smoking and mental illness is well-established although the causal link is unclear (Breslau et al., 1998; Prochaska, 2011; Burki, 2016; Lipari and Van Horn, 2017; Friedman, 2020; Choudhury, 2021). If socializing with peers who have a higher genetic risk for depression increases own tobacco use, it may contribute to worsening mental health.

I consider three smoking-related measures in the Add Health—whether one ever smoked, the number of cigarettes smoked per day (CPD) in the past 30 days, and quit attempts during the past 6 months. Results are reported in Table 1.10. Panel A shows results in the short-term (Wave I) and Panel B shows the long-term effects (Wave IV). In general, I do not find significant impacts of same-gender grademates' average MDD score on smoking in the short- or long-term. The one exception is a decline in the probability of attempting to quit among males in the short-run.

Table 1.10: Effect of Same-Gender Grademates' and Own MDD Score on Tobacco Use in Waves I and IV

			Conditional on Smoking			
	Have Ever Smoked Regularly		CPD During Past 30 Days		Quit Attempt During Past 6 Months	
	Panel A: Wave I					
	(1) Females	(2) Males	(3) Females	(4) Males	(5) Females	(6) Males
Same-Gender Grademates' Average MDD Score	-0.006	0.014	0.064	0.279	0.009	-0.195***
	(0.013)	(0.012)	(0.536)	(1.287)	(0.036)	(0.054)
Own MDD Score	0.008	0.007	-0.128	0.540	0.004	-0.030
	(0.008)	(0.009)	(0.288)	(0.372)	(0.023)	(0.024)
Mean	0.197	0.205	5.982	7.278	0.554	0.546
N	2,330	1,672	611	442	632	463
R^2	0.206	0.177	0.373	0.361	0.359	0.426
	Panel B: Wave IV					
	(1) Females	(2) Males	(3) Females	(4) Males	(5) Females	(6) Males
Same-Gender Grademates' Average MDD Score	0.002	0.006	0.170	0.797	0.028	-0.013
	(0.015)	(0.013)	(0.557)	(1.121)	(0.023)	(0.046)
Own MDD Score	-0.002	0.017	-0.361	-0.347	0.040**	-0.004
	(0.009)	(0.014)	(0.437)	(0.462)	(0.019)	(0.021)
Mean	0.425	0.497	10.320	12.078	0.827	0.743
N	2,326	1,673	779	682	713	482
R^2	0.202	0.168	0.334	0.378	0.308	0.380

Note: Panels A and B reports results from Waves I and IV, respectively. The outcome variable in columns (1) and (2) is an indicator variable for whether one has ever smoked. In columns (3) and (4), the outcome variable is the number of cigarettes smoked per day during the past month conditional on smoking. The outcome variable in columns (5) and (6) is an indicator variable for whether one attempts to quit smoking in the past 6 months conditional on smoking. All regressions include the controls in my most preferred specification (columns 4 and 8 of Table 1.4). Standard errors are clustered at the school level. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table 1.11: Effect of Same-Gender Grademates' and Own MDD Score on Marijuana Use

	During the Past 30 Days							
	Wave I		Wave IV: Days Used Marijuana					
	Times (≥ 0) Used Marijuana		Once a Month or More		Two or Three Days a Month or More		One or Two Days a Week or More	
	(1) Females	(2) Males	(3) Females	(4) Males	(5) Females	(6) Males	(7) Females	(8) Males
Same-Gender Grademates' Average MDD Score	0.212*	-0.797	0.008	-0.021	0.011	-0.011	0.011	-0.008
	(0.118)	(0.713)	(0.008)	(0.015)	(0.007)	(0.016)	(0.007)	(0.015)
Own MDD Score	0.241**	-0.004	0.008	-0.016	0.009*	-0.006	0.007	-0.008
	(0.093)	(0.370)	(0.005)	(0.010)	(0.005)	(0.009)	(0.005)	(0.008)
Mean	0.866	2.763	0.094	0.172	0.074	0.146	0.066	0.134
N	2,314	1,653	2,323	1,670	2,323	1,670	2,323	1,670
R^2	0.130	0.157	0.108	0.145	0.098	0.142	0.099	0.143

Note: The outcome variable in columns (1) and (2) is the number of times the respondent used marijuana during the past month in Wave I. In columns (3) and (4), the outcome variable is an indicator variable for whether an individual used marijuana once a month or more during the past 30 days in Wave IV. The outcomes in columns (5)-(6) and (7)-(8) are created similarly for two or three days a month and one or two days a week, respectively, in Wave IV. All regressions include the controls in my most preferred specification (columns 4 and 8 of Table 1.4). Standard errors are clustered at the school level. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Marijuana

Although there is no consensus on the causal link between marijuana use and mental health (Richardson et al., 2010; Serafini et al., 2013; Keith et al., 2015; NIDA, 2021), studies in economics and epidemiology have found causal evidence of marijuana use increasing the probability of mental illness, such as depression and suicidal ideation (Richardson et al., 2010; Van Ours and Williams, 2011, 2012, 2015; Van Ours et al., 2013; Pieniazek, 2022). If being around peers who have a higher genetic risk for depression during adolescence leads to an increase use of marijuana, it may result in worse mental health outcomes. I investigate whether peers' genetic risk for depression affects the frequency of marijuana use.

In both Waves I and IV, the Add Health asks respondents "During the past 30 days, how many times did you use marijuana?" In Wave I, respondents report the frequency of marijuana use while in Wave IV they choose from categories representing different frequencies. Table 1.11 presents the short- and the long-term effects of peers' genetic predisposition to depression on marijuana use. In columns (1) and (2), I find that an increase in same-gender grademates' average MDD score weakly increases the frequency of marijuana use for females in the short-run (Wave I), with no effects for males. In columns (3)-(8), I explore the long-term (Wave IV) effects of same-gender grademates' average MDD score on days used marijuana. The outcome variable in columns (3)-(4) is an indicator variable that takes on a value of one if days used marijuana is once a month or more. The outcome variables in columns (5)-(6) and (7)-(8) are created similarly for two or three days a month and one or two days a week, respectively. The point estimates for females are all positive and insignificant, but the estimates for males are all negative and insignificant.

Table 1.12: Effect of Same-Gender Grademates' and Own MDD Score on Socioeconomic Status (SES)

	At Least Some College		Currently Work for Pay		Log Labor Income	
	(1) Females	(2) Males	(3) Females	(4) Males	(5) Females	(6) Males
Same-Gender Grademates' Average MDD Score	-0.005	-0.045***	-0.027**	-0.013	-0.061	-0.013
	(0.012)	(0.012)	(0.011)	(0.021)	(0.072)	(0.074)
Own MDD Score	-0.003	-0.029***	-0.018	0.018*	-0.096	0.025
	(0.009)	(0.007)	(0.011)	(0.010)	(0.081)	(0.063)
Mean	0.788	0.699	0.737	0.841	9.021	9.912
N	2,331	1,680	1,942	1,374	2,248	1,618
R^2	0.220	0.273	0.150	0.172	0.159	0.180

Note: The outcome variable in columns (1) and (2) is an indicator variable for whether individuals have attended any type of higher level of training or education after high school regardless of completion, and zero otherwise. The outcome in columns (3)-(4) is an indicator variable that has a value of one if respondents currently work for pay at least 10 hours a week, and zero otherwise. The outcome in columns (5)-(6) is log labor income. All regressions include the controls in my most preferred specification (columns 4 and 8 of Table 1.4). Standard errors are clustered at the school level. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Taken together, I find that having peers with high genetic risk for depression increases alcohol and marijuana use for females. Thus, substance use may be a channel underlying the social-genetic effects.

1.6.3 Socioeconomic Status

Finally, I explore socioeconomic status (SES) as a mechanism that may explain the persistence of peer effects on mental health into adulthood. On the one hand, I show that having peers with higher genetic risk for depression increases adolescent depression, which may have a negative impact on SES in adulthood (Fletcher, 2010, 2013; Lundborg et al., 2014; Cornaglia et al., 2015; Mousteri et al., 2019). Worse SES may, in turn, result in worse mental health in the long-run (Chatterji et al., 2011; Layard, 2013; Salokangas, 2021). On the other hand, it could be that worse short-term mental health causes both worse mental health and lower SES in adulthood. I explore the effects of peers' genetic risk for depression on college attendance, employment, and log labor income.

In Wave IV (when all respondents are aged 24 and older), the Add Health asked respondents about the highest level of education achieved. I create a variable that takes on value one if an individual attended any type of higher level of training or education after high school regardless of completion, and zero otherwise. The Add Health also collected information on the respondents' current employment and income in Wave IV by asking the following questions, respectively: "Are you currently working for pay at least 10 hours a week?", and "Now think about your personal earnings. In (2006/2007/2008), how much income did

you receive from personal earnings before taxes, that is, wages or salaries, including tips, bonuses, and overtime pay, and income from self-employment?"

Table 1.12 presents the estimates. The results in column (2) indicate that a one standard deviation increase in same-gender grademates' average MDD score during adolescence significantly decreases the likelihood of pursing higher education after high school by 4.5 percentage points, a 6.4% decrease, for males, but has no effect for females. The results in column (3) suggest that a one standard deviation increase in same-gender grademates' average MDD score during adolescence leads to a 2.7 percentage point decrease, a 3.6% decrease, in the probability of working for pay in adulthood for females, with no effects for adult males. I do not find any significant effects on the log labor income for either gender.

Overall, there is some evidence that having peers with high genetic risk for depression during adolescence adversely affects various socioeconomic outcomes.

1.7 Robustness

I conduct several exercises to assess robustness of the baseline depression results. I explore sensitivity of the results to different definitions of depression as well as additional measures of mental health, non-linearities in peer effects, attrition bias, and bias that may arise from the absence of valid genetic data. I also perform placebo tests that provide support for the identification strategy.

1.7.1 Sensitivity to Different Depression Outcomes

In Appendix Table A5, I estimate my preferred specification (i.e., the specification with the most exhaustive set of controls) using different definitions of depression as outcomes. Panels A and B include the estimates for Waves I and IV, respectively. In columns (1) and (8), I create an indicator variable for experiencing depression based on the CES-D-19 score (i.e., CES-D-19 $\geq$ 16).[44] The results in Panel A are qualitatively consistent with the short-term effects in that the estimates are positive for both genders, but they are not significant. In columns (2) and (9), I use the continuous CES-D-10 score as an outcome. In Wave I, the coefficients are positive although they are not precisely estimated (Panel A). In Wave IV, the point estimate for females is positive and significant at the 5% level, but negative and statistically insignificant for males (Panel B).

In columns (3)-(7) and (10)-(14), I explore robustness of the baseline results to using different CES-D-10 cutoffs to define depression (i.e., $\geq$ 8, $\geq$ 9, $\geq$ 10, and $\geq$ 12). For comparison, I present the baseline results in columns (6) for females and (13) for males. The short-term effects (Panel A) for both females and males are robust to using cutoffs of 8 to 10, and if anything are larger in magnitude for females.[45] However, the results decrease in magnitude and become less precisely estimated with cutoffs of 12. Thus, the adverse effects of peers' genetic predisposition to depression are larger among those very close to the

[44] The CES-D-19 score is available only in Wave I. A cutoff of 16 is recommended for the CES-D-20 score when screening for depression (Radloff, 1977; Weissman et al., 1977). Instead of CES-D-20, the CES-D-19 score is available in Wave I. I use the CES-D-19 score with a cutoff of 16 following Giulietti et al. (2022).

[45] Andresen et al. (1994) identified CES-D-10 cutoffs of 8 and 10 as the optimal threshold for screening of depression.

clinically-defined threshold of depression (i.e., a cutoff of 11).[46] The long-term effects are presented in Panel B. I find that the increase in depression among females in the long-run due to peers' genetic risk is robust to perturbations of the cutoff, though the magnitude and statistical significance varies as the threshold changes.[47]

1.7.2 Nonlinear Effects of Peers' Average MDD Score

In the baseline model, I assume that the relationship between peers' genes for depression and own mental health is linear. But, the effect of peers' genetic risk for depression may differ across the distribution of peers' genetic risk. Moreover, evidence suggests that peer effects are often not linear (Betts and Shkolnik, 2000; Hoxby and Weingarth, 2005; Cooley, 2010; Sacerdote, 2011; Lavy et al., 2012; Imberman et al., 2012).

I re-estimate the baseline specification by replacing same-gender grademates' average MDD score with indicators for having same-gender grademates in different parts of the average MDD score distribution. Columns (1)-(4) and (5)-(8) in Appendix Table A6 contain results for the short- and long-term, respectively. In columns (1), (3), (5), and (7), I include an indicator for same-gender grademates' average MDD scores being above the median of the distribution. In columns (2), (4), (6), and (8), I use separate indicators for the first and third terciles of the same-gender grademates' average MDD score distribution.

I find nonlinear effects only for females. The results indicate that having same-gender peer groups with average MDD scores in the third tercile of the distribution increases the likelihood of females' own depression by 7.6 percentage points both in adolescence and adulthood (relative to having peers with average scores in the middle of the distribution). This implies that the baseline linear effects for females are driven by the upper tercile of the distribution.

Peer groups' average MDD score could be high if a handful of peers have very high MDD scores or if most individuals in the peer group have high MDD scores. In future work, I will explore these two possibilities to better understand the main source of the nonlinear effects, which has implications for how to structure peer groups to improve mental health for females.

1.7.3 Additional Measures of Mental Health

I next consider more severe mental health outcomes such as suicidal ideation and suicide attempts as well as depression diagnosis and antidepressant use.[48] The latter two measures are not available in Wave I, and all four measures are available in Wave IV.

In columns (1)-(2) and (3)-(4) of Appendix Table A7, I present the short-term (Wave I) effects of same-gender grademates' average MDD score on the probability of self-reported suicidal ideation and suicide

[46] For females, I fail to reject the null hypothesis of equality of coefficients of same-gender grademates' average MDD score when using the CES-D-10 score cutoffs of 10 and 11. However, I reject equality of coefficients across the results with cutoffs of 11 and 12 at the 5% level. For males, I fail to reject equality of coefficients in both tests.

[47] I fail to reject the null hypothesis of equality of coefficients of same-gender grademates' average MDD score across the columns for both genders.

[48] Information on suicidal ideation and suicide attempts are not available in Wave III, and the CES-D-10 score is not available in Wave V.

attempts, respectively. The results suggest that there is no statistically significant effect of same-gender grademates' average MDD score on these outcomes, and most of the point estimates are very close to zero.

In columns (5)-(12) of Appendix Table A7, I present the long-term (Wave IV) effects of same-gender grademates' average MDD score. Columns (5)-(6) and (7)-(8) report results for the likelihood of self-reported suicidal ideation and suicide attempts, respectively. For females, the estimate in column (5) indicates that a one standard deviation increase in same-gender grademates' average MDD score increases the probability of suicidal ideation by 2.2 percentage points (a 29.3% increase) and the probability of suicide attempts by 0.7 percentage points (a 53.8% increase). I find no statistically significant long-term peer effects on these outcomes among males.

Columns (9)-(10) and (11)-(12) show results for depression diagnosis and antidepressant use, respectively. For both genders, there is no evidence of peer effects on either outcome.

In sum, same-gender grademates' genetic risk for depression does not have immediate impacts on suicidal risk for either gender, but significantly affects suicidal ideation and weakly affects suicide attempts for females in the long-run.

1.7.4 Attrition and Absence of Valid Genetic Data

I explore two potential sources of bias—one arises from attrition and the other from the absence of valid genetic data.

First, I examine attrition from Waves I to IV. If respondents who had same-gender grademates' with a higher genetic risk score in adolescence are more likely to attrite from Waves I to IV, this may lead to biased results. In columns (1) and (2) of Appendix Table A8, I present results from a regression of an indicator for whether an individual dropped out of the sample from Wave I to IV on same-gender grademates' average MDD score conditional on the usual sets of controls in the most preferred specification except for the genetic principal components and own MDD score.[49] In all cases, I do not find evidence that same-gender grademates' average MDD score systematically affects sample attrition.

Then, I assess whether peers' average MDD score significantly affects an individual's decision to be genotyped. If respondents who had same-gender grademates' with higher genetic risk for depression in adolescence are less likely to be genotyped, this may result in biased results. In columns (3)-(4) of Appendix Table A8, I present results where I regress an indicator for not being genotyped on same-gender grademates' average MDD score controlling for the usual sets of controls in the most preferred specification except for the genetic principal components and own MDD score and conditional on appearing in Wave IV. I do not find evidence that same-gender grademates' average MDD score significantly influences the decision to be genotyped. Thus, it seems concerns about bias arising from attrition or absence of genetic data are minimal.

[49] In these analyses, the sample includes individuals who are not genotyped. Hence, their own genetic information is not available.

1.7.5 Verifying the Identification Strategy

To further assess the validity of the identification strategy, I perform placebo tests where I estimate the baseline specification after randomly re-assigning individuals to a different grade within the same school. I replace the actual same-gender grademates' average MDD score with a placebo same-gender grademates' average MDD score.[50] I then estimate my preferred baseline specification using the binary depression measure in Waves I and IV as the outcome. I repeat this procedure 1,000 times. In Appendix Figures A3 and A4, I plot the histogram of the coefficients on the placebo same-gender grademates' average MDD score from each regression against the coefficient on actual same-gender grademates' average MDD score from the baseline results. The distributions of the placebo estimates are centered around zero. I calculate p-values for a one-tailed test of the likelihood of observing a placebo coefficient being greater than or equal to the baseline estimate. For females, 1.3% and 0.1% of the placebo coefficients are greater than or equal to the actual coefficients in Waves I and IV, respectively. For males, 4.3% and 11% of the placebo coefficients are greater than the actual coefficients in Waves I and IV, respectively. With the exception of the effects for males in the long-run, these results provide strong support for the validity of the identification strategy.

1.8 Conclusion

I examine how peers' genetic risk for depression affects own mental health during adolescence and early adulthood using data from the Add Health survey. I find that a one standard deviation increase in peers' average MDD score significantly increases the probability of being depressed by 2.3 and 3 percentage points for adolescent girls (an 8.7% increase) and boys (a 20% increase), respectively. I also find that the short-term peer effects persist into adulthood for females. A one standard deviation increase in peers' average MDD score during adolescence leads to a statistically significant 2.9 percentage point increase in the probability of females being depressed in adulthood, a 14% increase. The findings suggest that depression in adolescence as well as adulthood is influenced not only by one's own genetic risk for depression but also by the genetic risk of one's peers. In other words, social-genetic effects are salient in the mental health context.

I explore several mechanisms underlying the effects, including friendship, substance use, educational attainment, and labor market outcomes. Interacting with peers with high genetic risk for depression worsens short- and long-term friendships. I find those with peers in adolescence with a higher genetic risk for depression interact less with friends during adolescence. Also, having same-gender grademates with higher genetic risk for depression decreases the frequency of hanging out with friends and the likelihood of having long-term and close friendships in adulthood. These effects are more pronounced for females than males. For females, being exposed to peers with high genetic risk for depression increases the frequency of binge drinking and marijuana use in both adolescence and adulthood. I also find that males and females who had peers with high genetic risk for depression in adolescence experience a lower SES in adulthood.

[50] Specifically, students are randomly re-assigned to a new grade (i.e., different grade) within the same school. They are then assigned their new grade's original same-gender grademates' average MDD score.

Males who had same-gender grademates with higher genetic risk for depression during adolescence are less likely to attend college, and females who used to have same-gender grademates with higher genetic risk for depression while they were in middle or high school are less likely to work for pay.

Overall, my findings imply that genes are an important part of the social environment, and mental health is a function of the genes of those around us. Hence, efforts to prevent and treat depression would be more effective by taking peers' genetic risk into account. However, this study is limited in that, by construction, the estimates do not tell us whether the effects of peers' genetic risk for depression operate mainly through peer depression. If the main mechanism is peer depression, my findings have implications for the design of interventions to curb adolescent depression (e.g., group-based vs. individual interventions) and suggest there are both short- and long-run social multiplier effects in schools in the context of mental health. In future work, I will explore whether there are circumstances or environments, such as childhood SES or relationships with parents, that mitigate the effects of peers' genetic risk for depression on mental health.

Chapter 2

The Complicated Links Between Health and the Genetic Endowments for Smoking with Meghan Skira

2.1 Introduction

Many chronic conditions and health outcomes, such as diabetes, cardiovascular disease, and body weight, have a strong genetic basis. For example, studies that compare identical and fraternal twins reveal that genetic differences explain 50–90 percent of the variation in body mass index (Wehby, 2016). Evidence on the heritability of health largely comes from these twin studies, which tell us how much genes collectively matter, but do not identify the particular genetic markers or pathways that influence health.

In this paper, we take advantage of recent advances in genetics that have led to the discovery of genetic markers that correlate with health behaviors. We explore the empirical relationship between health and genetic risk for smoking to understand whether and how genes linked to risky health behaviors explain variation in health outcomes. Understanding the factors underlying differences in health is important as the effects of public health policies will vary depending on the sources of health variation. Many policies directly restrict risky health behaviors, such as bans on smoking in public places or minimum legal purchase ages of cigarettes and alcohol. But, if the factors that predispose one toward risky behaviors also influence health even conditional on engagement in the risky behavior, then targeting these factors may be a more attractive policy option. For example, if genetic endowments linked to smoking are also tied to time preferences or returns on health investments, then commitment devices or monetary rewards for engaging in health-promoting behaviors may be more effective in modifying behavior and improving health.

We estimate associations between the genetic risk for smoking and health outcomes among those aged 50–65 using the Health and Retirement Study (HRS). We focus on two genetic measures—the polygenic score for smoking initiation (hereafter, the initiation score) and the polygenic score for smoking intensity

as measured by cigarettes smoked per day (hereafter, the CPD score). A polygenic score is a linear index of genetic markers that associate with the outcome or trait of interest; a higher score means an increased genetic risk for that outcome or trait. In our context, a higher initiation score means an individual is more likely to ever regularly smoke and a higher CPD score means they are more likely to smoke heavily.

Our estimates reveal a meaningful and statistically significant relationship between the smoking polygenic scores and several adverse health outcomes, and the associations are present even after flexibly controlling for individual smoking behavior. Higher initiation scores and CPD scores are associated with worse health on a variety of dimensions including self-reported health, diabetes, arthritis, body mass index, and obesity as well as an index that aggregates the individual health outcomes. Furthermore, a higher CPD score is robustly associated with worse health among both those who have ever smoked and never smokers, while never smokers drive the relationship between the initiation score and health. Taken together, our results suggest the genetic endowments for smoking associate with health through channels other than smoking.

As individuals inherit their genes from their parents, those with a higher genetic risk for smoking may grow up with parents who smoked or were in poor health. Variation in the smoking polygenic scores could, therefore, reflect different childhood environments, such as differential exposure to secondhand smoke. We add controls for parental smoking, parental mortality, and parental risky behaviors (problematic drug use or drinking), and find these controls associate significantly with an individual's health, but barely change the estimated relationship between the smoking polygenic scores and health. Given evidence that health outcomes and behaviors are correlated between spouses and that smokers are more likely to marry smokers, we also add controls for spousal smoking behavior and spousal smoking polygenic scores. The addition of these controls, particularly spousal smoking behavior, diminishes the estimated relationship between one's own CPD score and health by 15–25 percent. We find that the spousal CPD score has a statistically significant adverse relationship with own health (controlling for spousal smoking behavior). As couples likely engage in similar health behaviors, such as those related to diet, nutrition, and physical activity, these results further support the notion that genetic endowments for smoking reflect factors that influence health and health behaviors more generally beyond smoking.

Given the robust associations between the smoking polygenic scores and health that remain even after including parental and spousal controls, we then explore channels through which the genetic factors may operate, namely risk preferences, longevity expectations, time preferences, and impulsivity. Studies show smokers tend to be more risk-prone, impatient, and impulsive than non-smokers (e.g., Khwaja et al., 2006, 2007a). If the genetic endowments for smoking reflect these traits, this could explain their association with health. We find that increased genetic risk for smoking significantly associates with lower expectations of living to age 75, shorter financial planning horizons, and lower degrees of conscientiousness (more impulsiveness), but controlling for these mechanisms only moderately decreases the relationship between the polygenic scores and health. Collectively, these channels account for at most a quarter of the relationship between the CPD score and health, and about half of the relationship between the initiation score and health.

Our focus on genetic endowments for smoking rather than other risky behaviors is motivated in part by data availability. We want to understand whether genetic markers associated with risky health behaviors explain variation in health and whether these markers influence health through channels other than that particular behavior. As such, we need detailed information about one's engagement in the risky behavior as well as that of their parents and spouse. The HRS includes rich data on smoking that meets these requirements. While polygenic scores for other health behaviors like alcohol use and physical activity exist, these behaviors are not measured as comprehensively in the HRS and are likely more difficult to recall. Furthermore, several of the genetic variants linked to smoking relate to specific biological systems, which provides a lens through which to interpret our results. For example, variants near dopamine receptors strongly correlate with smoking intensity, and variants associated with ever smoking relate to systems that affect reward-based learning and addiction (Liu et al., 2019). Thus, the smoking polygenic scores reflect, in part, biological factors linked to addiction, which may be important for health even conditional on smoking behavior. We note, however, that the polygenic scores aggregate various traits and biological pathways that associate with smoking, and should not be interpreted narrowly as a predisposition to addiction.

Our work is tied to several strands of literature. One strand focuses on the role of genes in explaining variation in health. Twin studies, which compare identical and fraternal twins to calculate the fraction of the variance in health outcomes attributed to genes, find a number of chronic conditions, such as cardiovascular disease, obesity, diabetes, and arthritis, have a strong genetic component (see reviews in Polderman et al., 2015; Wehby, 2016). A more nascent related line of literature studies the role of genes in the intergenerational transmission of health by comparing adoptive parent-child pairs and biological parent-child pairs (Thompson, 2014; Classen and Thompson, 2016). These studies find genetic transmission accounts for an important share of the intergenerational correlation in chronic conditions (e.g., asthma, diabetes, obesity). Twin studies and adoptee-based designs tell us how much genes matter collectively for a given health outcome or for health transmission, but not the specific genes or genetic pathways.[1] As a result, our knowledge of the channels through which genes influence health is limited. We demonstrate that genetic endowments for smoking, a policy-relevant and not uncommon risky behavior, predict health even after accounting for smoking behavior. Our results are consistent with individuals with a higher genetic propensity to smoke being less likely to engage in health-promoting behaviors. Our findings also provide new insights on the high prevalence of comorbidities (i.e., having two or more chronic conditions) in the United States.

This study also relates to recent literature that shows the polygenic score for educational attainment (hereafter, the EA score) predicts economic and health outcomes even after accounting for educational attainment and sheds light on the channels through which the EA score operates (Belsky et al., 2016; Barth et al., 2020; Papageorge and Thom, 2020; Bolyard and Savelyev, 2021). Our paper is similar in that we show a polygenic score predicts outcomes it was not designed to predict and we explore the mechanisms

[1] Linnér and Koellinger (2022) is a recent exception. They investigate how 27 polygenic scores for common medical conditions and mortality risk, including the CPD and smoking initiation scores, predict differences in longevity in the HRS. They find several of the polygenic scores, including the CPD score, have a non-negligible association with mortality. Consistent with our results, the CPD score significantly predicts mortality even after accounting for observed smoking.

underlying that relationship, but our focus is distinct in that we consider smoking polygenic scores and health outcomes.

We also contribute to the economic literature on the genetic risk for smoking, which typically focuses on the role of gene-environment interactions in smoking behavior (Boardman et al., 2011; Fletcher, 2012; Meyers et al., 2013; Domingue et al., 2016; Schmitz and Conley, 2016; Bierut et al., 2018; Wedow et al., 2018; Biroli and Zwyssig, 2021). Our focus is not on gene-environment interactions or smoking decisions, but rather understanding whether the smoking polygenic scores are predictive of health and the channels that mediate the association. By providing a comprehensive picture of how genetic endowments for smoking function, our analysis may provide an interpretative framework for the gene-environment interactions found in prior studies.

Our paper also contributes to a large strand of literature that documents differences in the characteristics of smokers and non-smokers with respect to preferences and subjective beliefs (e.g., Barsky et al., 1997; Khwaja et al., 2006, 2007a; Anderson and Mellor, 2008; Chabris et al., 2008; Ida and Goto, 2009). These studies generally find smokers are more impatient, risk tolerant, and impulsive than non-smokers. Khwaja et al. (2006) find no significant change in these characteristics among those who change their smoking status, which they interpret as evidence that these characteristics are innate and not caused by smoking. We contribute to this literature by exploring whether individuals who are genetically predisposed to smoking initiation and heavy smoking differ along these margins. Our results show those with higher genetic risk for smoking expect earlier mortality, have shorter financial planning horizons, and exhibit more impulsiveness. We therefore provide a possible genetic microfoundation for the documented differences among smokers and non-smokers.

Finally, while our analysis is descriptive in nature, the results have implications for the literature that estimates causal effects of smoking on health, underscoring the importance of time-invariant unobserved heterogeneity. For example, Darden (2017) and Darden et al. (2018) uncover strong evidence of unobserved heterogeneity—individuals who are more likely to smoke are also at increased risk of poor chronic health and more likely to die independently of smoking. The sources of unobserved heterogeneity are not modeled, though the authors speculate genetics or differences in the likelihood of engaging in correlated risks may be at play. Our results shed light on the black box of unobserved heterogeneity, highlighting a non-trivial role for genetic endowments, and our findings are consistent with a genetic underpinning for engagement in risky and health-degrading behaviors. Our findings also have implications for interpreting instrumental variable estimates of smoking on health. In particular, if compliers systematically differ in their genetic endowments for smoking, substantial treatment effect heterogeneity is likely, limiting the generalizability of the local average treatment effect of smoking on health. In addition, our results underscore that the smoking polygenic scores should not be used as instruments for smoking as the exclusion restriction will be violated.[2]

The rest of the paper is organized as follows. Section 2.2 provides a brief overview of polygenic scores, including the ones used in our analyses. Section 2.3 describes the Health and Retirement Study data and

[2]We are not the first to caution against the use of polygenic scores as instruments for specific health conditions or behaviors. See for example Benjamin et al. (2011), Lehrer and Ding (2017), and Fletcher (2018).

sample construction. Section 2.4 describes the empirical strategy. Section 2.5 presents the estimates of the relationship between health outcomes and smoking polygenic scores, and Section 2.6 explores channels that may mediate the observed relationship. Section 2.7 presents the results of robustness exercises. Section 2.8 provides some final thoughts.

2.2 Background on Polygenic Scores

Large-sample twin studies reveal that smoking is moderately to highly heritable (Li et al., 2003; Agrawal and Lynskey, 2008), and recent advances in genetics have led to the discovery of particular genetic markers that predict smoking behavior. We rely on polygenic scores, which aggregate these genetic markers. We provide a brief overview of polygenic scores here and refer the reader to Benjamin et al. (2011) and Beauchamp et al. (2011) for a detailed discussion of the human genome and Barth et al. (2020) and Papageorge and Thom (2020) for a detailed discussion of polygenic scores.

Broadly, a polygenic score is a linear index of genetic markers (or variants) associated with an observable trait or outcome, such as smoking behavior, educational attainment, body mass index, blood pressure, etc. A polygenic score is obtained by aggregating millions of individual genetic variants across the human genome and weighting those variants by the strength of their association with the outcome of interest. The estimated associations between genetic markers and the observable traits or outcomes come from genome-wide association studies (GWAS), where thousands or millions of linear regressions are estimated linking specific genetic variants called single nucleotide polymorphisms (SNPs) to the outcome of interest. The polygenic score is a linear combination of the GWAS coefficients and is calculated as follows:

$$PGS_i = \sum_{j=1}^{J} \beta_j SNP_{ij} \tag{2.1}$$

where SNP_{ij} is the genotype (i.e., the number of copies of the reference allele–0, 1, or 2) for individual i at SNP j and β_j is the effect size for SNP j estimated in the GWAS. A higher polygenic score means an individual possesses more of the genetic variants that associate with the trait or outcome. That is, a higher score means the individual has a higher genetic risk for that trait or outcome.

There are various ways to construct a polygenic score. Sometimes, researchers use only the variants with the strongest GWAS associations, namely SNPs that are considered genome-wide significant (e.g., p-value $< 5 \times 10^{-8}$). Other times, all SNPs are included regardless of genome-wide significance. In some cases, the GWAS coefficients (i.e., the β_j's) are adjusted to account for correlations between nearby SNPs using linkage disequilibrium. We use the polygenic scores constructed and released by the Health and Retirement Study. The scores include all available SNPs that overlap between the HRS genetic database and the relevant GWAS, without accounting for linkage disequilibrium or p-value thresholds, as this set of decisions led to the largest predictive power of the polygenic scores (Ware et al., 2017b).

In our analysis, we focus on two polygenic scores related to smoking behavior—one for smoking initiation (the initiation score) and one for cigarettes per day (the CPD score). The initiation score measures the

genetic endowments linked to whether an individual ever smoked regularly while the CPD score measures genetic endowments linked to heaviness of smoking. The scores are based on GWAS meta-analyses results from Liu et al. (2019), which used a discovery sample of 1.2 million individuals of European ancestry for the initiation score and a subsample of 337,334 current or former smokers for the CPD score.[3] The discovery sample did not include data from the HRS, but the HRS was used to assess out-of-sample predictive power. The out-of-sample prediction results in Liu et al. (2019) imply that the initiation score explains about 4 percent of the variation in smoking initiation and the CPD score likewise explains about 4 percent of the variation in smoking intensity (among current or former smokers) in the HRS. We document the relationship between the polygenic scores and smoking behavior in our sample in the next section.

It is worth noting that several of the genetic variants implicated in the smoking polygenic scores are linked to particular biological systems and functions. For example, variants near dopamine receptors are associated with smoking heaviness but not smoking initiation. Some variants associated with smoking initiation relate to systems that affect reward-based processing and addiction. Nicotine receptor and nicotine metabolizing genes are also associated with smoking behaviors (Liu et al., 2019). Importantly, although the polygenic scores contain variants related to addiction, nicotine response, and reward-related learning, they should not be interpreted narrowly as a predisposition to nicotine addiction given they aggregate many biological systems, pathways, and traits.

2.3 Data

The Health and Retirement Study (HRS) is a longitudinal survey of individuals aged 50 and over in the United States and their spouses. The survey started in 1992 and followup surveys are conducted biennially. We use data from 1994–2016 in our analysis.[4] The data contain rich information on an array of topics, such as health, family structure, employment, assets, and expectations about future events.

Genetic data was collected during four waves (2006, 2008, 2010, and 2012) from respondents who consented to provide DNA samples. The HRS has made several polygenic scores publicly available for the approximately 15,000 individuals who have been genotyped. We use the fourth release of the HRS's polygenic score data. Most polygenic scores are for diseases and clinical outcomes, such as Alzheimer's disease, high blood pressure, and body mass index. More recently, due in part to the increased availability of genetic data, polygenic scores linked to behavioral traits, such as smoking, have been calculated from GWAS and released by the HRS. As discussed above, we focus on two polygenic scores linked to smoking—one for smoking initiation (initiation score) and one for cigarettes smoked per day (CPD score). The HRS normalizes the scores to have mean zero and a standard deviation equal to one within ancestry groups (i.e., European and African). A higher score implies more genetic risk for the corresponding trait (e.g., to ever smoke or smoke heavily).

[3] Although the sample used to discover the genetic markers that relate to smoking intensity only included current or former smokers, the CPD score can be constructed for all individuals regardless of their smoking history.

[4] We exclude the 1992 survey wave due to survey question differences.

2.3.1 Sample Construction

Our sample includes HRS respondents aged 50–65 who have been genotyped. We focus on this age group to minimize survival bias concerns and avoid capturing potential impacts of retirement on health.[5] The HRS provides polygenic scores for about 12,000 respondents with European ancestry and 3,000 respondents with African ancestry. The scores we use were discovered based on the GWAS of individuals with European ancestry, and scores based on such GWAS tend to lack predictive power when applied to other ethnic groups. We follow the convention in this literature (e.g., Barth et al., 2020; Papageorge and Thom, 2020) and limit our sample to respondents classified as genetic Europeans and who self-identify as white.

2.3.2 Health Outcomes

The HRS provides rich information on various health outcomes. We consider both self-reported measures of physical health as well as doctor-diagnosed health conditions.

Respondents are asked to rate their general health on a 1–5 scale, where 1 is excellent, 2 is very good, 3 is good, 4 is fair, and 5 is poor. We create a "bad" self-reported health indicator that takes on a value of one if the individual reports their health as fair or poor, and zero otherwise. We create an indicator for whether an individual reports any overnight hospital stay in the last two years. We consider an individual's body mass index (BMI) based on self-reports of weight and height as well as an indicator for being obese (i.e., BMI greater than or equal to 30 kg/m^2).

We also create several indicators based on whether an individual reports that they have ever been diagnosed with certain conditions by a doctor, including high blood pressure; diabetes; cancer; chronic lung disease (e.g., chronic bronchitis, emphysema); heart problems (e.g., heart attacks coronary heart disease, angina, congestive heart failure); stroke; and arthritis. We construct an indicator for having difficulties with activities of daily living (ADLs) as well as an indicator for having difficulties with instrumental activities of daily living (IADLs). ADLs include walking across a room, dressing, bathing, eating, getting in and out of bed, and using the toilet. IADLs include using the phone, taking medications, managing money, shopping for groceries, preparing meals, using a map, using a calculator, using a microwave, and using a computer.

We construct a summary standardized index of these health measures in the spirit of Kling et al. (2007), Hoynes et al. (2016), and Bütikofer et al. (2021). To calculate this index, we create the z-score for each individual health measure and then take the average of the z-scores. The z-score is created by subtracting the mean and dividing by the standard deviation. Aggregating the measures in this way improves statistical power (Kling et al., 2007). We create the index using all the health measures described above (except the obesity indicator as BMI is already included). As all the components of the index are associated with poor health, an increase in the index suggests a deterioration in health. In our analysis, we largely focus on this

[5] See for example Coe and Zamarro (2011); Behncke (2012); Insler (2014); Eibich (2015); Godard (2016); Mazzonna and Peracchi (2017); Gorry et al. (2018); Shai (2018). Later, we verify the robustness of our results to only including those aged 50–61, i.e., below the early Social Security retirement age.

summary standardized index of health as it allows us to draw general conclusions about health and genetic endowments for smoking, but we also examine each health outcome separately.[6]

2.3.3 Own Smoking Behavior

To examine whether the smoking polygenic scores are associated with health even after accounting for smoking behavior, we control for an individual's current and past smoking behavior as flexibly as possible. We make use of a wealth of smoking data available in the HRS described below.

We create an indicator for current smoking that takes on a value of one if the individual reports that they currently smoke, and zero otherwise. Similarly, we create an indicator variable for ever smoking that takes on a value of one for individuals who currently smoke or ever smoked in their lifetime. Among those who currently smoke, the HRS asks about the average number of cigarettes smoked per day. We set this variable to zero for those who do not currently smoke. For those who ever smoked but are not current smokers, we have information on the maximum number of cigarettes smoked per day when they were smokers. We use information on the age when ever smokers started smoking and quit smoking (if they have quit). We also create an indicator for teenage smoking that takes on a value of one if the individual reports they regularly smoked cigarettes when they were in grade school or high school, and zero otherwise. When data on smoking variables are missing, we set the value to zero and include an indicator for missing values of that variable.

2.3.4 Parental Smoking, Risky Behaviors, and Mortality

In some analyses, we control for parental behaviors during the respondent's childhood as well as parental mortality to the extent the data allows. The survey asks respondents whether their parents smoked during their childhood and the responses include neither parent smoked, one of them smoked, or both smoked. We create indicators for each potential response. We also construct an indicator for whether either parent drank or used drugs so often that it caused family problems during the respondent's childhood. We construct various measures of parental mortality. For a small subset of our sample, we can create an indicator for whether a parent died before the individual turned age 16.[7] The HRS also contains information on whether parents are alive as of the current survey wave, and if not, the parent's age when they passed away. Using this information, we create a categorical variable for the respondent's mother's mortality status: alive, died before age 65, and died after age 65 as well as analogous measures for the respondent's father. When data on any of these parental variables are missing, we set the value to zero and include an indicator for missing values of that variable.

[6] We also show our baseline results are robust when we consider a summary standardized index based only on the doctor-diagnosed conditions.

[7] Parental mortality before the respondent reached age 16 was only collected in recent years via life history mail surveys that are distributed to a subsample of respondents and have far less than 100 percent response rates.

Table 2.1: Summary Statistics

	Full Sample			Ever Smoker Sample			Never Smoker Sample			
	Mean	SD	N	Mean	SD	N	Mean	SD	N	*p*-value
Demographics:										
Age	58.56	4.16	46,692	58.60	4.16	26,963	58.51	4.16	19,729	0.02
Birth Year	1945.24	7.64	46,692	1944.96	7.65	26,963	1945.63	7.61	19,729	0.00
Male	0.42	0.49	46,692	0.48	0.50	26,963	0.34	0.47	19,729	0.00
Own Smoking:										
# Cigs per Day (Among Current Smokers)	17.55	10.99	8,427	17.55	10.99	8,427				
Maximum # Cigs per Day (Among Former Smokers)	22.25	17.83	16,560	22.25	17.83	16,560				
Age Start Smoking (Among Ever Smokers)	17.62	5.00	25,785	17.62	5.00	25,785				
Age Quit Smoking (Among Quitters)	36.81	11.27	16,379	36.81	11.27	16,379				
Smoked as a Teenager	0.38	0.49	26,391	0.38	0.49	26,391				
Own Health:										
Poor Self-Reported Health	0.17	0.38	46,676	0.20	0.40	26,952	0.13	0.33	19,724	0.00
Any Hospital Stay	0.17	0.38	46,624	0.19	0.39	26,922	0.15	0.36	19,702	0.00
Ever Had High Blood Pressure	0.39	0.49	46,659	0.40	0.49	26,932	0.37	0.48	19,727	0.00
Ever Had Diabetes	0.11	0.32	46,663	0.12	0.32	26,949	0.11	0.31	19,714	0.18
Ever Had Cancer	0.08	0.28	46,637	0.08	0.28	26,919	0.09	0.28	19,718	0.47
Ever Had Lung Disease	0.06	0.25	46,671	0.09	0.29	26,956	0.03	0.17	19,715	0.00
Ever Had Heart Problem	0.13	0.34	46,667	0.15	0.36	26,951	0.11	0.31	19,716	0.00
Ever Had Stroke	0.03	0.17	46,669	0.04	0.19	26,947	0.02	0.14	19,722	0.00
Ever Had Arthritis	0.45	0.50	46,654	0.47	0.50	26,945	0.42	0.49	19,709	0.00
Any Limitations with ADLs	0.08	0.27	46,669	0.09	0.29	26,952	0.06	0.24	19,717	0.00
Any Limitations with IADLs	0.04	0.19	46,667	0.04	0.20	26,951	0.03	0.16	19,716	0.00
Body Mass Index (kg/m^2)	28.12	5.73	46,115	28.05	5.65	26,681	28.23	5.82	19,434	0.00
Obese	0.31	0.46	46,115	0.31	0.46	26,681	0.32	0.47	19,434	0.00
Parental Smoking:										
Neither Parent Smoked	0.18	0.38	46,692	0.15	0.36	26,963	0.21	0.41	19,729	0.00
One Parent Smoked	0.30	0.46	46,692	0.30	0.46	26,963	0.29	0.45	19,729	0.00
Both Parents Smoked	0.19	0.39	46,692	0.21	0.41	26,963	0.16	0.36	19,729	0.00
Missing	0.34	0.47	46,692	0.33	0.47	26,963	0.34	0.48	19,729	0.01
Parental Mortality:										
Neither Parent Died During Childhood	0.19	0.39	46,692	0.18	0.38	26,963	0.20	0.40	19,729	0.00
Parent(s) Died During Childhood	0.03	0.18	46,692	0.03	0.18	26,963	0.03	0.18	19,729	0.62
Parental Mortality During Childhood Missing	0.78	0.41	46,692	0.79	0.41	26,963	0.77	0.42	19,729	0.00
Mother Currently Alive	0.38	0.49	46,692	0.37	0.48	26,963	0.39	0.49	19,729	0.00
Mother Died Before Age 65	0.14	0.35	46,692	0.15	0.35	26,963	0.13	0.34	19,729	0.00
Mother Died Age 65+	0.47	0.50	46,692	0.47	0.50	26,963	0.46	0.50	19,729	0.02
Mother Mortality Information Missing	0.02	0.13	46,692	0.02	0.13	26,963	0.02	0.12	19,729	0.14
Father Currently Alive	0.17	0.37	46,692	0.15	0.36	26,963	0.19	0.39	19,729	0.00
Father Died Before Age 65	0.25	0.43	46,692	0.26	0.44	26,963	0.23	0.42	19,729	0.00
Father Died Age 65+	0.57	0.50	46,692	0.57	0.50	26,963	0.56	0.50	19,729	0.39
Father Mortality Information Missing	0.02	0.14	46,692	0.02	0.15	26,963	0.01	0.12	19,729	0.00
Parental Risky Behavior During Respondent's Childhood:										
Parent(s) Drank/Used Drugs Often	0.20	0.40	46,692	0.23	0.42	26,963	0.17	0.38	19,729	0.00
Neither Parent Drank/Used Drugs	0.73	0.44	46,692	0.70	0.46	26,963	0.77	0.42	19,729	0.00
Missing	0.07	0.25	46,692	0.07	0.26	26,963	0.06	0.23	19,729	0.00

Note: The table presents summary statistics for the full sample and separately for those who ever and never smoked in their life. The sample includes those aged 50–65 of European ancestry who self-identify as white and who have been genotyped. The last column presents p-values from a test of equality of means for ever and never smokers.

2.3.5 Descriptives

Summary statistics for our sample are provided in Table 2.1. In our empirical analyses, we estimate models on the full sample and then separately for ever and never smokers. Thus, we present statistics for the full sample and by whether an individual has ever smoked. Average age in the sample, regardless of ever smoking status, is 58. About half the ever smoker sample is male compared to a third of the never smoker group.

Among current smokers, the average number of cigarettes smoked per day is almost 18, and among those who do not currently smoke but have smoked in the past, their smoking peaked, on average, at 22 cigarettes per day. On average, ever smokers began smoking between ages 17–18, and among those who have quit, the average age of quitting is 36. Almost 40 percent of ever smokers smoked during their teenage years.

As expected, the health of the never smokers is better than that of the ever smokers along several dimensions, and the differences in health are statistically significant in almost all cases. Consistent with smoking being one of the leading causes of coronary and pulmonary diseases in the US (US Department of Health and Human Services, 2020), the proportion of individuals with diagnosed lung disease and heart problems is higher in the ever smoker sample. Parental smoking during the respondent's childhood is more common among the ever smokers. In addition, the ever smokers are more likely to have experienced a parental death before age 16 and to report that one or both parents drank or used drugs so often it caused problems during their childhood.

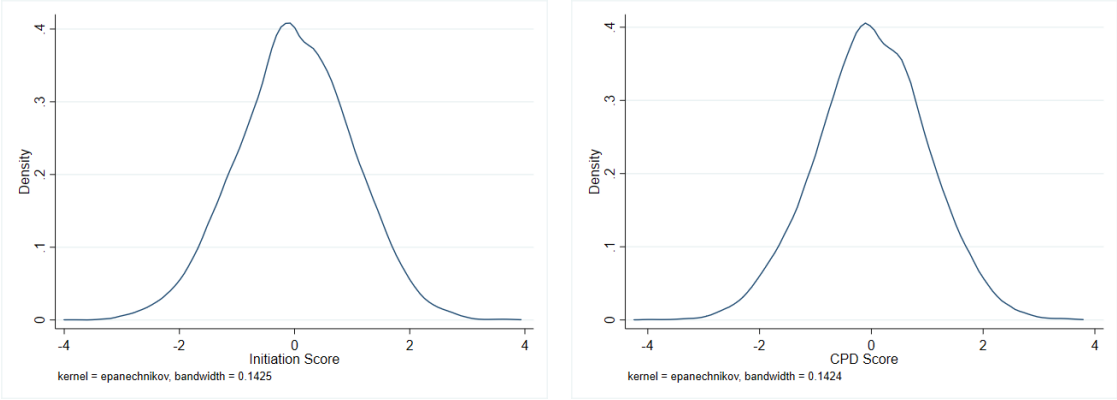
Only HRS respondents who consented to provide genetic data are included in our sample. For comparison, in Appendix Table B1, we provide summary statistics from 1994–2016 for non-genotyped HRS respondents aged 50–65 of European decent who identify as white. On average, the genotyped sample has more females and is healthier. If anything, the genotyped sample is positively selected on health, which will bias any negative association between health and the polygenic scores associated with smoking toward zero.

In Figure 2.1, we show the smoothed densities of the smoking polygenic scores for individuals in our sample. Next, we display the non-parametric (lowess) relationships between the smoking polygenic scores and their corresponding smoking outcomes. Panel (a) of Figure 2.2 shows the relationship between the initiation score and the probability of ever smoking, which is positive and approximately linear. Similarly, Panel (b) of Figure 2.2 shows the relationship between the CPD score and average cigarettes smoked per day among current smokers.[8] This relationship is also positive and approximately linear. For former smokers, we have information on the maximum number of cigarettes smoked per day when they smoked. Panel (c) of Figure 2.2 plots this measure of peak smoking among former smokers against their CPD score. We observe a positive relationship, which is particularly strong among those with relatively high scores.

We also quantify the relationship between the smoking polygenic scores and smoking behavior in our sample. Appendix Table B2 presents estimates from regressions of the maximum number of cigarettes smoked per day among ever smokers (columns 1–3) or an indicator for whether an individual ever smoked

[8] For each respondent, we calculate their average reported number of cigarettes smoked per day across periods when they report currently smoking and plot that average against their CPD score.

Figure 2.1: Distributions of the Smoking Polygenic Scores



(a) Initiation Score

(b) CPD Score

Note: Panel A shows the smoothed density of the smoking initiation score and Panel B shows the smoothed density of the CPD (smoking intensity) score.

(columns 4–6) on the CPD score only, the initiation score only, and both scores.[9] Unsurprisingly, the CPD score explains more of the variation in smoking intensity than the initiation score, with an incremental R^2 of 0.015 compared to 0.004 for the initiation score. A one standard deviation increase in the CPD score increases the number of cigarettes smoked by 2, while a one standard deviation increase in the initiation score is associated with a 1 cigarette increase. Similarly, the initiation score explains far more of the variation in the probability of ever smoking compared to the CPD score, with an incremental R^2 of 0.031 compared to 0.003 for the CPD score. A one standard deviation increase in the CPD score is associated with a 1.6–2.7 percentage point increase in the probability of ever smoking, whereas a one standard deviation increase in the initiation score is associated with about a 9 percentage point increase. In sum, the polygenic scores predict their respective smoking outcomes in our sample.

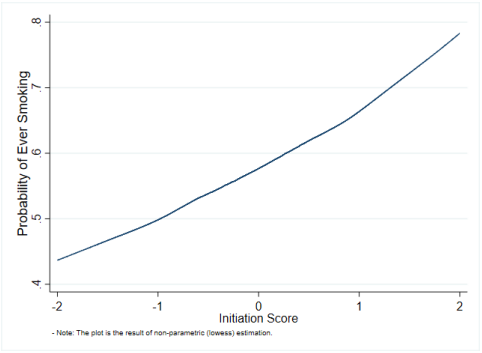
2.4 Empirical Strategy

To examine the link between health and genetic endowments for smoking, we estimate the following model via OLS:

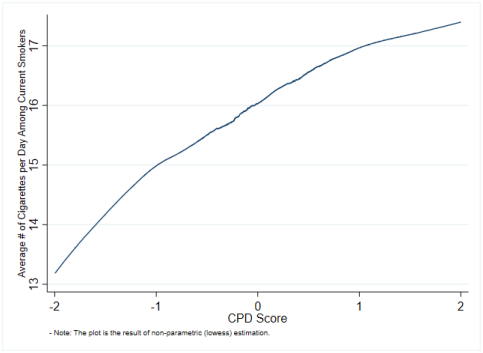
$$Y_{it} = \beta_0 + \beta_1 CPDScore_i + \beta_2 InitiationScore_i + \beta_3 X_{it} + \varepsilon_{it}, \tag{2.2}$$

[9] In all specifications we include the standard controls and genetic principal components described in Section 2.4.

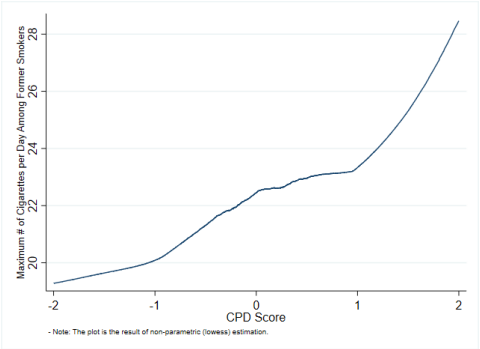
Figure 2.2: Relationship between Smoking Polygenic Scores and Various Smoking Measures



(a) Initiation Score and Probability of Ever Smoking



(b) CPD Score and Average # of Cigarettes per Day Among Current Smokers



(c) CPD Score and Maximum # of Cigarettes per Day Among Former Smokers

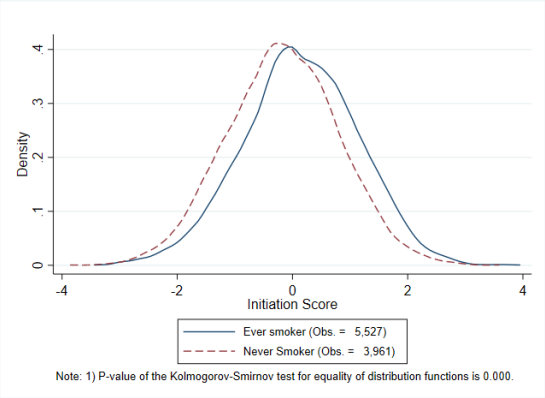
Note: Panel (a) shows the non-parametric (lowess) relationship between the smoking initiation score and the probability of ever smoking (as of the last time an individual is observed in our sample). Panel (b) shows the lowess relationship between the CPD score and average cigarettes smoked per day among current smokers. Panel (c) shows the lowess relationship between the CPD score and the maximum number of cigarettes smoker per day among former smokers.

where Y_{it} is the health outcome of individual i at survey wave t, $CPDScore_i$ is the polygenic score for the number of cigarettes per day, $InitiationScore_i$ is the polygenic score for smoking initiation, and X_{it} is a vector of controls.[10] Given we observe the same individual potentially many times, we cluster standard errors at the individual level.

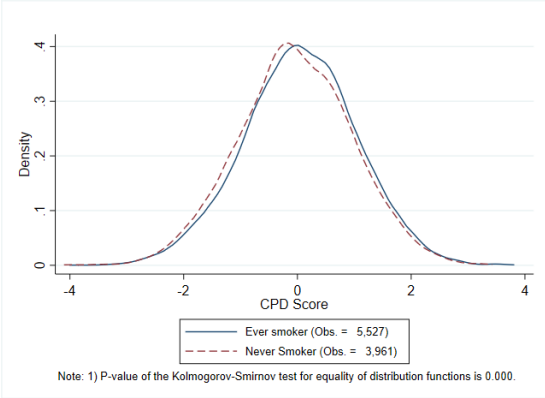
We categorize the variables in X_{it} into the following groups: standard controls, smoking controls, and genetic principal components. Following Barth et al. (2020) and Papageorge and Thom (2020),

[10]We have also allowed for an interaction between the initiation and CPD scores. The point estimate on the interaction is generally insignificant and very close to zero and its inclusion leaves the point estimates on the (non-interacted) initiation and CPD scores largely unchanged. We proceed without the interaction term for ease of exposition and interpretability of the estimates, but results with the interaction term are available by request.

Figure 2.3: Distribution of Smoking Polygenic Scores Among Ever and Never Smokers



(a) Initiation Score Among Ever and Never Smokers



(b) CPD Score Among Ever and Never Smokers

Note: Panel A shows the smoothed density of the smoking initiation score separately for ever and never smokers, and Panel B shows the smoothed density of the CPD (smoking intensity) score separately for ever and never smokers.

the standard controls include age dummies, a male dummy, birth year dummies, the interaction of age dummies and the male dummy, the interaction of birth year dummies and the male dummy, and survey year dummies. As we want to understand whether the smoking polygenic scores predict health outcomes even after accounting for smoking, we control for smoking behavior as flexibly as possible. We include dummies for ever smoked status, dummies for each reported number of cigarettes smoked per day, and dummies for each level of reported number of maximum cigarettes smoked per day among former smokers. We additionally include dummies for current smoking status, dummies for age started smoking, dummies for age quit smoking, and teenage smoking status. For all of these smoking controls, we include dummies for missing values. Finally, to account for potential bias that may arise from population stratification and genetic differences across ethnic groups (even within our sample of individuals with European ancestry who identify as white), we follow standard practice (e.g., Price et al., 2006; Belsky et al., 2016; Barth et al., 2020) and include the 10 principal components of the full matrix of SNP data as well as their interactions with the male dummy.

We estimate equation 2.2 on the full sample of individuals, pooling across ever and never smokers. We also estimate the model separately by ever smoking status.[11] By estimating equation 2.2 on never smokers, we directly examine whether the smoking polygenic scores correlate with health through avenues other than own smoking. Thus, any observed relationship between the smoking scores and health for this sample must be explained by non-smoking channels. In Panels (a) and (b) of Figure 2.3, we show that distributions

[11]We only observe 4 individuals who smoke for the first time during the sample period and hence change ever smoking status. Results are robust to excluding these individuals.

of the smoking initiation and CPD scores for ever and never smokers share common support, alleviating concerns about comparing estimates derived from these samples.[12]

2.5 Main Results

2.5.1 Health Outcomes and the Smoking Scores

We now present and discuss the estimated relationship between health and the genetic endowments for smoking. In Table 2.2, we show results for the summary standardized index of the 12 health measures described in Section 2.3.2.[13] Estimates for the full sample are shown in columns (1)–(3). Column (1) contains results not including any controls. The estimates imply a one standard deviation increase in the CPD score increases the health index by 0.042 standard deviations and a one standard deviation increase in the initiation score increases the health score by 0.026 standard deviations; both estimates are significant at the 1 percent level. Recalling that an increase in the index implies worse health, the results imply that higher genetic risk for smoking is associated with worse overall health. In Column (2), we add the standard controls and the 10 principal components of the genetic data. The coefficient on the initiation score grows in magnitude while the coefficient on the CPD score barely changes. In Column (3), we add the smoking controls, and unsurprisingly, the coefficients on the CPD and initiation scores attenuate. In this case, a one standard deviation increase in the CPD (initiation) score increases the health index by 0.035 (0.020) standard deviations. Thus, even when we flexibly control for current and past smoking behavior, higher genetic risk for smoking is significantly associated with worse health.

Columns (4)–(6) present results for the ever smoker sample. After including the standard controls, principal components, and smoking controls, we find a one standard deviation increase in the CPD score is correlated with a statistically significant 0.037 standard deviation increase in the summary index. A one standard deviation increase in the initiation score increases the health index by 0.011 standard deviations, but the estimate is not statistically significant. We present results for the never smoker sample in columns (7) and (8), a group for which there is no smoking behavior to control for. For this group, a one standard deviation increase in the CPD score or the initiation score is associated with a statistically significant 0.030 standard deviation increase in the health index.[14] We cannot reject equality of the CPD score coefficients across the ever and never smoker samples, while we reject equality of the initiation score coefficients at the 5 percent level.

[12] We note, however, that the p-values from Kolmogorov-Smirnov tests reject the null hypothesis of equality of the distributions by smoking status at the 5 percent level. This is unsurprising, particularly for the initiation score given it predicts ever smoking status.

[13] The outcomes include indicator variables for "bad" self-reported health status, having a hospital stay in the past two years, having doctor-diagnosed high blood pressure, diabetes, cancer, lung disease, heart problems, stroke, arthritis, having difficulties with ADLs, having difficulties with IADLs as well as BMI measured continuously.

[14] In Appendix Table B3, we present results for a summary standardized index that aggregates just the seven doctor-diagnosed conditions (high blood pressure, diabetes, cancer, lung disease, heart problems, stroke, and arthritis). The estimates are similar to those using the summary index that includes the broader set of health measures.

Table 2.2: Relationship between the Smoking Polygenic Scores and the Summary Standardized Index of Health

	Full Sample			Ever Smoker Sample			Never Smoker Sample	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
CPD Score	0.042***	0.043***	0.035***	0.048***	0.047***	0.037***	0.032***	0.032***
	(0.004)	(0.004)	(0.004)	(0.006)	(0.006)	(0.006)	(0.006)	(0.006)
Initiation Score	0.026***	0.035***	0.020***	0.019***	0.023***	0.011	0.021***	0.030***
	(0.004)	(0.005)	(0.005)	(0.006)	(0.007)	(0.007)	(0.006)	(0.007)
Full Smoking Controls	No	No	Yes	No	No	Yes	No	No
Standard Controls	No	Yes	Yes	No	Yes	Yes	No	Yes
Principal Components	No	Yes	Yes	No	Yes	Yes	No	Yes
Sample Mean	-0.000	-0.000	-0.000	0.038	0.038	0.038	-0.052	-0.052
N	45,850	45,850	45,850	26,509	26,509	26,509	19,341	19,341
R^2	0.014	0.070	0.117	0.013	0.081	0.143	0.010	0.080

Note: Each column presents results from a separate regression where the outcome is the summary standardized health index. Results are presented for the full sample and then separately for ever smokers and never smokers. For each sample, we first present results without controls (columns 1, 4, 7). Then we add the standard controls and the genetic principal components described in Section 2.4 (columns 2, 5, 8). Then for the full sample and ever smoker sample, we add the full set of smoking controls described in Section 2.4 (columns 3, 6). * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

In Table 2.3, we present estimates for the individual health measures that appear in the summary standardized index for the full sample (Panel A), ever smokers (Panel B), and never smokers (Panel C). All models include the standard controls, genetic principal components, and the controls for smoking behavior (except in Panel C where there is no smoking behavior to account for). For most of the measures, a higher genetic risk for smoking, particularly smoking intensity, is associated with worse health in the full sample. We find some differences in these relationships among the ever and never smokers, though in most cases, we cannot reject equality of the CPD score coefficients or initiation score coefficients across the two groups.[15] For example, the smoking initiation score has a stronger relationship with health outcomes among the never smokers, and the CPD score correlates significantly with more health outcomes among the ever smokers. We explore these patterns in detail later.[16] Notably, we observe qualitatively similar and precisely estimated point estimates on the CPD score across the ever and never smoker samples for the following outcomes: self-reported "bad health," diabetes diagnosis, arthritis diagnosis, BMI, and obesity. Diabetes and obesity are influenced by health behaviors such as diet and exercise, and being overweight is a common cause of arthritis. One interpretation of these results is that genetic endowments for smoking correlate with behaviors that are health degrading besides smoking.

[15]We reject equality of the CPD score coefficients for the following outcomes: recent hospital stay, diagnosed lung disease, and BMI. We reject equality of the initiation score coefficients for recent hospital stay, BMI, and obesity.

[16]These results are in line with those in Linnér and Koellinger (2022) who find larger associations between the CPD score and mortality among ever smokers relative to never smokers.

Table 2.3: Relationship between the Smoking Polygenic Scores and Individual Health Measures

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)
			Ever Diagnosed With:							Any Limitations With:			
	Poor Self-Reported Health	Any Hospitalization	High Blood Pressure	Diabetes	Cancer	Lung Disease	Heart Problem	Stroke	Arthritis	ADLs	IADLs	BMI	Obese
						Panel A: Full Sample							
CPD Score	0.019***	0.010***	0.017***	0.019***	0.000	0.011***	0.007**	-0.000	0.021***	0.010***	0.004***	0.502***	0.033***
	(0.003)	(0.002)	(0.005)	(0.003)	(0.003)	(0.002)	(0.003)	(0.002)	(0.005)	(0.002)	(0.001)	(0.061)	(0.005)
Initiation Score	0.018***	0.008***	0.005	0.003	-0.004	0.009***	0.005	0.004**	0.004	0.007***	0.005***	0.229***	0.016***
	(0.003)	(0.003)	(0.005)	(0.003)	(0.003)	(0.003)	(0.004)	(0.002)	(0.005)	(0.002)	(0.001)	(0.067)	(0.005)
Full Smoking Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Sample Mean	0.170	0.174	0.386	0.114	0.084	0.064	0.132	0.030	0.451	0.080	0.036	28.122	0.312
N	46,676	46,624	46,659	46,663	46,637	46,671	46,667	46,669	46,654	46,669	46,667	46,115	46,115
R^2	0.068	0.026	0.058	0.058	0.033	0.087	0.052	0.033	0.075	0.037	0.030	0.084	0.060
						Panel B: Ever Smoker Sample							
CPD Score	0.018***	0.013***	0.021***	0.016***	-0.001	0.018***	0.010**	-0.000	0.022***	0.010***	0.003*	0.387***	0.027***
	(0.004)	(0.003)	(0.007)	(0.004)	(0.004)	(0.004)	(0.005)	(0.003)	(0.007)	(0.003)	(0.002)	(0.078)	(0.006)
Initiation Score	0.015***	0.003	-0.002	-0.001	-0.006	0.010**	0.001	0.003	-0.002	0.004	0.006**	0.107	0.008
	(0.005)	(0.004)	(0.007)	(0.004)	(0.004)	(0.004)	(0.005)	(0.003)	(0.007)	(0.003)	(0.002)	(0.086)	(0.006)
Full Smoking Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Sample Mean	0.202	0.189	0.398	0.115	0.083	0.090	0.149	0.038	0.470	0.093	0.043	28.046	0.305
N	26,952	26,922	26,932	26,949	26,919	26,956	26,951	26,947	26,945	26,952	26,951	26,681	26,681
R^2	0.086	0.035	0.082	0.075	0.049	0.103	0.067	0.044	0.088	0.051	0.044	0.121	0.086
						Panel C: Never Smoker Sample							
CPD Score	0.021***	0.004	0.011	0.024***	0.002	0.004	0.002	-0.001	0.017**	0.009***	0.004**	0.641***	0.041***
	(0.004)	(0.004)	(0.008)	(0.005)	(0.004)	(0.003)	(0.005)	(0.002)	(0.008)	(0.003)	(0.002)	(0.097)	(0.007)
Initiation Score	0.019***	0.013***	0.014*	0.008	-0.004	0.007**	0.008	0.006***	0.012	0.011***	0.003*	0.373***	0.026***
	(0.005)	(0.004)	(0.008)	(0.005)	(0.005)	(0.003)	(0.005)	(0.002)	(0.008)	(0.003)	(0.002)	(0.104)	(0.008)
Sample Mean	0.126	0.154	0.370	0.111	0.085	0.029	0.109	0.020	0.424	0.063	0.026	28.227	0.321
N	19,724	19,702	19,727	19,714	19,718	19,715	19,716	19,722	19,709	19,717	19,716	19,434	19,434
R^2	0.032	0.017	0.050	0.053	0.032	0.031	0.039	0.026	0.070	0.024	0.014	0.062	0.049

Note: Each column within a panel presents results from a separate regression where the outcome is noted in the column heading. Results for the full sample are presented in Panel A, for the ever smoker sample in Panel B, and for the never smoker sample in Panel C. In all specifications, we include the standard controls and the genetic principal components described in Section 2.4. We also include the full set of smoking controls described in Section 2.4 for the full sample and ever smoker samples. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Overall, the results in Tables 2.2 and 2.3 imply higher genetic risk for smoking is associated with worse health even after flexibly controlling for smoking behaviors. This finding is robust across ever and never smokers. The fact that these associations are present and precisely estimated for never smokers is particularly compelling as there are no own smoking channels through which the scores can operate, suggesting non-smoking mechanisms are at play. Furthermore, if there is concern that the relationships between the scores and health in the full and ever smoker samples simply reflect smoking behavior that is not well-captured in our smoking controls, the fact that we find robust and meaningful associations that are fairly similar in the ever and never smoker samples should alleviate that concern.

The most notable difference in the results across the ever and never smoker samples is the point estimate on the initiation score. In the specification with the most rigorous set of controls, we find a positive but not precisely estimated relationship between the initiation score and the summary health index among ever smokers but a larger and precisely estimated relationship among never smokers. Earlier, we showed the initiation score distribution is, unsurprisingly, shifted left for never smokers compared to ever smokers. If the association between the initiation score and health is non-linear and the samples represent different parts of the distribution, this could explain the difference in the point estimates. Appendix Figure B1 shows lowess plots of the smoking polygenic scores and the summary health index separately for ever and never smokers. For never smokers, the genes-health gradient somewhat flattens as the polygenic scores, especially the initation score, increase, suggesting non-linearities may be important. Another possibility is that the initiation score interacts with ever smoking status. To explore these possibilities, we examine the relationship between different terciles of the CPD score and initiation score distributions and health.

The results for the summary standardized index and the individual health measures are presented in Tables 2.4 and 2.5, respectively, for the full sample, ever smokers, and never smokers. For brevity, we focus on the summary health index. The indicators for being in the second tercile of the CPD score and the initiation score are normalized. The estimates reveal some interesting patterns. In the full sample, those in the first tercile of the CPD score distribution experience a 0.034 standard deviation decrease (i.e., improvement) in the summary health index relative to those in the middle of the distribution, and this improvement is amplified for never smokers. In the full sample, those in the third tercile of the CPD score distribution experience a 0.046 standard deviation increase (i.e., deterioration) in the health index relative to those in the second tercile. In this case, the relationship is larger in magnitude for ever smokers. A similar but even more striking pattern is apparent for the initiation score. Ever smokers in the first tercile of the initiation score see no significant difference in health relative to those in the second tercile, whereas the third tercile is associated with worse health, but the association is not significant once smoking controls are included. Never smokers with initiation scores in the first tercile experience significantly better health relative to those in the middle tercile, but the third tercile does not significantly correlate with health. We are only able to reject the null hypothesis of equality of coefficients across the ever and never smoker samples for the first tercile of the initiation score ($p < 0.05$).

Table 2.4: Relationship between the Terciles of the Smoking Polygenic Scores and the Summary Standardized Index of Health

	Full Sample			Ever Smoker Sample			Never Smoker Sample	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
CPD Score Tercile 1	-0.044***	-0.045***	-0.034***	-0.040***	-0.041***	-0.026*	-0.042***	-0.048***
	(0.010)	(0.010)	(0.010)	(0.014)	(0.014)	(0.014)	(0.015)	(0.014)
CPD Score Tercile 3	0.052***	0.052***	0.046***	0.063***	0.060***	0.051***	0.036**	0.030*
	(0.011)	(0.011)	(0.011)	(0.015)	(0.015)	(0.015)	(0.016)	(0.016)
Initiation Score Tercile 1	-0.022**	-0.034***	-0.022**	0.001	-0.005	0.003	-0.036**	-0.046***
	(0.010)	(0.011)	(0.011)	(0.015)	(0.015)	(0.015)	(0.014)	(0.015)
Initiation Score Tercile 3	0.035***	0.039***	0.020*	0.041***	0.038***	0.023	0.010	0.015
	(0.011)	(0.011)	(0.011)	(0.015)	(0.015)	(0.014)	(0.017)	(0.017)
Full Smoking Controls	No	No	Yes	No	No	Yes	No	No
Standard Controls	No	Yes	Yes	No	Yes	Yes	No	Yes
Principal Components	No	Yes	Yes	No	Yes	Yes	No	Yes
Sample Mean	-0.000	-0.000	-0.000	0.038	0.038	0.038	-0.052	-0.052
N	45,850	45,850	45,850	26,509	26,509	26,509	19,341	19,341
R^2	0.011	0.066	0.115	0.010	0.079	0.141	0.010	0.079

Note: Each column presents results from a separate regression where the outcome is the summary standardized health index. Results are presented for the full sample and then separately for ever smokers and never smokers. For each sample, we first present results without controls (columns 1, 4, 7). Then we add the standard controls and the genetic principal components described in Section 2.4 (columns 2, 5, 8). Then for the full sample and ever smoker sample, we add the full set of smoking controls described in Section 2.4 (columns 3, 6). Indicators for being in tercile 2 of the CPD score and tercile 2 of the initiation score are omitted. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Taken together, these results suggest there are some non-linearities in the health-smoking genes relationship.[17] Moreover, the difference in the baseline point estimates on the initiation score for ever and never smokers is driven by those with low initiation scores. In particular, the genetic factors associated with a low initiation score combined with never smoking seem to be protective of health. There is a qualitatively similar but quantitatively smaller interaction effect between the CPD score and ever smoking status. The one exception is a large and statistically significant negative association between being in the lowest tercile of the CPD score distribution and BMI and obesity among never smokers.

[17]We note, however, that we cannot reject symmetry of the associations along the distribution of each score within each sample. For example, we cannot reject that the coefficients on being in the first tercile of the CPD score equals minus the coefficient of being in the third tercile.

Table 2.5: Relationship between the Terciles of the Smoking Polygenic Scores and Individual Health Measures

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)
			Ever Diagnosed With:							Any Limitations With:			
	Poor Self-Reported Health	Any Hospitalization	High Blood Pressure	Diabetes	Cancer	Lung Disease	Heart Problem	Stroke	Arthritis	ADLs	IADLs	BMI	Obese
Panel A: Full Sample													
CPD Score Tercile 1	-0.017**	-0.009	-0.008	-0.021***	-0.002	-0.010*	-0.001	0.001	-0.022*	-0.009*	-0.002	-0.629***	-0.044***
	(0.007)	(0.006)	(0.012)	(0.007)	(0.007)	(0.005)	(0.008)	(0.004)	(0.012)	(0.005)	(0.003)	(0.143)	(0.011)
CPD Score Tercile 3	0.027***	0.010*	0.028**	0.024***	-0.005	0.016***	0.017**	-0.001	0.030**	0.015***	0.004	0.545***	0.038***
	(0.008)	(0.006)	(0.012)	(0.008)	(0.007)	(0.006)	(0.008)	(0.004)	(0.012)	(0.006)	(0.003)	(0.148)	(0.011)
Initiation Score Tercile 1	-0.009	-0.008	-0.014	0.000	0.001	-0.002	-0.001	-0.006	-0.017	-0.006	-0.006**	-0.414***	-0.034***
	(0.008)	(0.006)	(0.012)	(0.008)	(0.007)	(0.005)	(0.008)	(0.004)	(0.012)	(0.005)	(0.003)	(0.147)	(0.011)
Initiation Score Tercile 3	0.028***	0.010*	0.000	0.001	-0.008	0.014**	0.002	0.002	0.002	0.011**	0.005	0.078	0.001
	(0.008)	(0.006)	(0.012)	(0.008)	(0.007)	(0.006)	(0.008)	(0.004)	(0.012)	(0.006)	(0.003)	(0.149)	(0.011)
Full Smoking Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Sample Mean	0.170	0.174	0.386	0.114	0.084	0.064	0.132	0.030	0.451	0.080	0.036	28.122	0.312
N	46,676	46,624	46,659	46,663	46,637	46,671	46,667	46,669	46,654	46,669	46,667	46,115	46,115
R^2	0.067	0.026	0.058	0.058	0.033	0.086	0.052	0.033	0.076	0.036	0.030	0.083	0.060
Panel B: Ever Smoker Sample													
CPD Score Tercile 1	-0.020*	-0.012	-0.000	-0.016	0.002	-0.016*	0.005	-0.002	-0.023	-0.005	-0.002	-0.257	-0.021
	(0.011)	(0.008)	(0.016)	(0.010)	(0.009)	(0.008)	(0.011)	(0.006)	(0.016)	(0.007)	(0.004)	(0.181)	(0.014)
CPD Score Tercile 3	0.018*	0.014*	0.043***	0.022**	-0.006	0.023**	0.034***	-0.004	0.035**	0.013*	0.003	0.513***	0.035**
	(0.011)	(0.008)	(0.015)	(0.010)	(0.009)	(0.009)	(0.011)	(0.006)	(0.016)	(0.008)	(0.005)	(0.182)	(0.014)
Initiation Score Tercile 1	-0.005	0.005	-0.001	0.018	0.000	-0.005	0.008	0.001	-0.005	0.003	-0.004	-0.262	-0.031**
	(0.011)	(0.008)	(0.017)	(0.011)	(0.009)	(0.009)	(0.012)	(0.006)	(0.017)	(0.007)	(0.005)	(0.194)	(0.015)
Initiation Score Tercile 3	0.028***	0.011	-0.004	0.005	-0.008	0.014	-0.004	0.005	0.005	0.015**	0.009*	0.054	-0.010
	(0.010)	(0.008)	(0.015)	(0.010)	(0.009)	(0.009)	(0.011)	(0.006)	(0.015)	(0.007)	(0.005)	(0.183)	(0.014)
Full Smoking Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Sample Mean	0.202	0.189	0.398	0.115	0.083	0.090	0.149	0.038	0.470	0.093	0.043	28.046	0.305
N	26,952	26,922	26,932	26,949	26,919	26,956	26,951	26,947	26,945	26,952	26,951	26,681	26,681
R^2	0.086	0.035	0.082	0.076	0.049	0.102	0.068	0.044	0.089	0.051	0.043	0.120	0.086
Panel C: Never Smoker Sample													
CPD Score Tercile 1	-0.015	-0.006	-0.021	-0.031***	-0.008	-0.006	-0.011	0.006	-0.024	-0.013*	-0.003	-1.154***	-0.077***
	(0.010)	(0.008)	(0.018)	(0.011)	(0.010)	(0.006)	(0.011)	(0.005)	(0.018)	(0.007)	(0.004)	(0.228)	(0.017)
CPD Score Tercile 3	0.036***	0.002	0.005	0.025**	-0.004	0.002	-0.007	0.002	0.013	0.015*	0.004	0.511**	0.036*
	(0.012)	(0.009)	(0.019)	(0.012)	(0.010)	(0.007)	(0.012)	(0.005)	(0.019)	(0.008)	(0.005)	(0.249)	(0.018)
Initiation Score Tercile 1	-0.012	-0.022***	-0.028	-0.017	0.003	0.003	-0.011	-0.015***	-0.025	-0.015**	-0.008**	-0.531**	-0.034**
	(0.010)	(0.008)	(0.018)	(0.011)	(0.011)	(0.006)	(0.012)	(0.005)	(0.018)	(0.007)	(0.004)	(0.224)	(0.017)
Initiation Score Tercile 3	0.024**	0.011	0.004	-0.002	-0.010	0.017**	0.011	-0.003	0.004	0.005	-0.001	0.085	0.015
	(0.012)	(0.009)	(0.020)	(0.013)	(0.011)	(0.008)	(0.013)	(0.006)	(0.020)	(0.009)	(0.005)	(0.257)	(0.019)
Sample Mean	0.126	0.154	0.370	0.111	0.085	0.029	0.109	0.020	0.424	0.063	0.026	28.227	0.321
N	19,724	19,702	19,727	19,714	19,718	19,715	19,716	19,722	19,709	19,717	19,716	19,434	19,434
R^2	0.031	0.017	0.050	0.053	0.032	0.031	0.040	0.027	0.070	0.024	0.014	0.063	0.051

Note: Each column within a panel presents results from a separate regression where the outcome is noted in the column heading. Results for the full sample are presented in Panel A, for the ever smoker sample in Panel B, and for the never smoker sample in Panel C. In all specifications, we include the standard controls and the genetic principal components described in Section 2.4. We also include the full set of smoking controls described in Section 2.4 for the full sample and ever smoker samples. Indicators for being in tercile 2 of the CPD score and tercile 2 of the initiation score are omitted. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

2.5.2 Parental Smoking, Risky Behaviors, and Mortality

One potential explanation for the relationship between health outcomes and the smoking polygenic scores is parental smoking, risky behaviors, and health and mortality. As one inherits their genes from their

parents, an individual's genetic risk of smoking is correlated with their parents' genetic risk of smoking. Parental genetic predisposition toward smoking could impact their child's health in at least four ways. First, it could impact the child's health directly through smoking and secondhand smoke exposure. Second, parents with higher smoking polygenic scores may have been in worse health during the respondent's childhood due to smoking and/or the influence of the smoking genes that operates outside of smoking, which may have led to a disadvantaged childhood. Third, if the smoking polygenic scores are correlated with poor or risky health behaviors more broadly, children may have learned those behaviors from their parents. Fourth, if parents with a genetic predisposition for smoking experience worse health or die at an earlier age and individuals view the health of their parents as foreshadowing their own health, they may adjust their own health-related behaviors in response.[18]

Table 2.6 presents the estimated relationship between the summary standardized index of health and the smoking polygenic scores after controlling for parental smoking, parental risky behaviors, and parental mortality during the respondent's childhood as well as parental mortality as of the current survey wave. We do not have parental genetic information, and hence cannot directly explore the role of parents' genetic endowments for smoking. We again present results for the full sample and separately by ever smoker status. Across the various samples, there is a slight decline in the magnitude of the associations between the summary index of health and the smoking polygenic scores after including the parental variables, but they are still statistically significant (except the initiation score among ever smokers). We still find a non-trivial and robust association between the CPD score and health. The correlation between the initiation score and health, like in the baseline results, is largely driven by the never smoker sample.

In Appendix Tables B4, B5, and B6, we present the estimates for the individual health measures for the full sample, ever smokers, and never smokers, respectively, controlling for the parental measures described above. The inclusion of parental controls does not meaningfully change the estimated relationships between the smoking polygenic scores and the individual health measures. Taken together, the results thus far suggest the relationship between genetic endowments for smoking and health largely operates through channels other than own smoking and parental behaviors and mortality.

2.5.3 Spousal Genetic Smoking Endowments and Smoking Behavior

Another potential avenue through which the smoking polygenic scores may relate to own health is spousal smoking and spousal genetic endowments for smoking. It is well-documented that health outcomes and health behaviors are correlated between spouses and smokers are more likely to marry smokers (Clark and Etilé, 2006; Oreffice and Quintana-Domeque, 2010; Chiappori et al., 2012, 2018). Thus, an individual's own smoking behavior may be correlated with that of their spouse. Less clear is whether genetic risk for smoking is correlated among spouses.

Following Charles et al. (2013) and Barth et al. (2020), in Appendix Table B7 we consider married couples where both members provided genetic data and are aged 50–65 and categorize them into quartiles

[18] Darden and Gilleskie (2016) find limited evidence that offspring smoking is sensitive to parental health. One exception is women decrease their smoking following a parent's smoking-related cardiovascular event and they also report worse subjective health after the smoking-related death of a parent.

Table 2.6: Relationship between the Smoking Polygenic Scores and the Summary Standardized Index of Health Accounting for Parental Smoking, Risky Behaviors, and Mortality

	Full Sample		Ever Smoker Sample		Never Smoker Sample	
	(1)	(2)	(3)	(4)	(5)	(6)
CPD Score	0.035***	0.033***	0.037***	0.034***	0.032***	0.029***
	(0.004)	(0.004)	(0.006)	(0.006)	(0.006)	(0.006)
Initiation Score	0.020***	0.017***	0.011	0.010	0.030***	0.025***
	(0.005)	(0.005)	(0.007)	(0.007)	(0.007)	(0.007)
Parental Controls	No	Yes	No	Yes	No	Yes
Full Smoking Controls	Yes	Yes	Yes	Yes	No	No
Standard Controls	Yes	Yes	Yes	Yes	Yes	Yes
Principal Components	Yes	Yes	Yes	Yes	Yes	Yes
Sample Mean	-0.000	-0.000	0.038	0.038	-0.052	-0.052
N	45,850	45,850	26,509	26,509	19,341	19,341
R^2	0.117	0.134	0.143	0.156	0.080	0.106

Note: Each column presents results from a separate regression where the outcome is the summary standardized health index. Results are presented for the full sample and then separately for ever smokers and never smokers. In all specifications, we include the standard controls and the genetic principal components described in Section 2.4. We also include the full set of smoking controls described in Section 2.4 for the full sample and ever smoker samples. In columns (2), (4), and (6), we add controls for parental smoking, parental risky behaviors, and parental mortality as described in Section 2.3.4. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

based on their CPD score (Panel A) and their initiation score (Panel B). We calculate the fraction of men in each quartile within a given female quartile. The diagonals of these matrices would be 100 percent and off-diagonal elements would be 0 percent with perfect assortative mating, while random assignment would imply 25 percent in each cell. For the CPD score, while we reject the random assignment null hypothesis (i.e., all cell entries equal to 25 percent) at the 10 percent level, the extent of assortative mating is small, with cell entries ranging from 20.9–30.4 percent. For the initiation score, we cannot reject the random assignment null hypothesis, and cell entries range from 22.2–28.3 percent. Furthermore, the within-couple correlation of the CPD scores is 0.03 as is the correlation of the initiation scores, while the correlation of ever smoking status is 0.24. Thus, we find little evidence of assortative mating on genetic endowments related to smoking, but smokers are more likely to marry smokers.

We next estimate the relationship between the summary standardized index of health and own smoking polygenic scores after controlling for spousal smoking polygenic scores and spousal smoking behaviors.[19] We first add spousal smoking controls only, then spousal smoking polygenic scores only, and then both sets of spousal measures. The sample is smaller than that of our main analyses as we only include married households where both members were genotyped and aged 50–65. The results are presented in Table 2.7 for the full sample, ever smokers, and never smokers. Across the samples, the addition of spousal controls moderately decreases the magnitude of the point estimate on the CPD score, but it is still positive and statistically significant. For ever smokers, the point estimate on the initiation score is never statistically significant, and for never smokers, the association between the initiation score and health declines in magnitude with the inclusion of spousal controls but remains statistically significant. The decline in the size of the association between the polygenic scores and health is mostly driven by the inclusion of the spousal smoking variables, not the spousal polygenic scores. This is not surprising given we found little evidence of assortative mating on smoking polygenic scores. Interestingly, we find a robust and significant relationship between the spouse's CPD score and own health. A one standard deviation increase in the spouse's CPD score generates a 0.2 standard deviation increase in the health index. Thus, spousal genetic risk for smoking generates negative spillovers for own health.[20] To further explore this idea, we focus on couples where neither person has ever smoked, eliminating any own or spousal smoking influences. The results are presented in Appendix Table B11. Focusing on the summary standardized index of health in column (1), the association between own genetic risk for smoking and health attenuates somewhat, but we still find a significant association between a spouse's CPD score and own health. These results are consistent with smoking polygenic scores reflecting poor health behaviors more generally besides smoking, as a lack of health-promoting behaviors by a spouse could adversely affect one's own health.

Overall, spousal smoking behavior plays a small mediating role in the relationship between own smoking genes and health. Spousal genetic endowments for smoking have a direct correlation with own health, but do not explain the association between own genetic endowments for smoking and own health. Even after including these spousal controls, we are still left with a robust and meaningful association between the smoking polygenic scores and own health. This conclusion also holds when including *both* the parental and spousal variables. The results in Appendix Table B12 show that the association between the CPD score and the health index decreases by less than 20 percent across all samples, and in the case of never smokers, the association between the initiation score and the health index declines by 38 percent when parental and spousal controls are added.

2.6 Additional Mechanisms

In Section 2.5, we found higher genetic risk for smoking associates with worse health even after flexibly controlling for respondent's own smoking behaviors. The estimated relationship is not explained by parental

[19] We control for spousal smoking identically to how we control for own smoking as described in Section 2.3.3.

[20] In Appendix Tables B8–B10, we present results for the individual health measures after controlling for spousal polygenic smoking scores and spousal smoking behavior. We again find modest declines in the associations between own smoking polygenic scores and health, as well as adverse spillover effects of the spouse's CPD score on some of the health measures.

Table 2.7: Relationship between the Smoking Polygenic Scores and the Summary Standardized Index of Health Accounting for Spouse's Smoking Behavior and Smoking Polygenic Scores

	Full Sample				Ever Smoker Sample				Never Smoker Sample			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
CPD Score	0.030***	0.028***	0.030***	0.028***	0.030***	0.029***	0.031***	0.030***	0.029***	0.024***	0.029***	0.025***
	(0.006)	(0.006)	(0.006)	(0.006)	(0.008)	(0.008)	(0.008)	(0.008)	(0.008)	(0.008)	(0.008)	(0.008)
Initiation Score	0.015**	0.011*	0.013**	0.009	0.005	0.001	0.002	-0.001	0.029***	0.025***	0.027***	0.024***
	(0.006)	(0.006)	(0.006)	(0.006)	(0.008)	(0.008)	(0.008)	(0.008)	(0.008)	(0.008)	(0.008)	(0.008)
Spouse's CPD Score			0.025***	0.022***			0.022***	0.020***			0.026***	0.023***
			(0.006)	(0.006)			(0.008)	(0.008)			(0.008)	(0.008)
Spouse's Initiation Score			0.004	0.000			0.007	0.006			0.001	-0.005
			(0.006)	(0.006)			(0.008)	(0.008)			(0.009)	(0.009)
Spouse's Full Smoking Controls	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes
Spouse's Principal Components	No	No	Yes	Yes	No	No	Yes	Yes	No	No	Yes	Yes
Full Smoking Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	No	No	No	No
Standard Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Principal Components	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Sample Mean	-0.035	-0.035	-0.035	-0.035	0.004	0.004	0.004	0.004	-0.085	-0.085	-0.085	-0.085
N	21,357	21,357	21,357	21,357	11,888	11,888	11,888	11,888	9,469	9,469	9,469	9,469
R^2	0.129	0.153	0.136	0.159	0.172	0.203	0.181	0.212	0.088	0.142	0.097	0.150

Note: Each column presents results from a separate regression where the outcome is the summary standardized health index. Results are presented for the full sample and then separately for ever smokers and never smokers, and we only include individuals in couples where both members are aged 50–65 and genotyped. In all specifications, we include the standard controls and the genetic principal components described in Section 2.4, and for the full sample and ever smoker sample, we always include the full set of smoking controls described in Section 2.4. In columns (2), (6), and (10), we include the full set of the spouse's smoking controls. In columns (3), (7), and (11), we include the spouse's smoking polygenic scores and their genetic principal components. In columns (4), (8), and (12), we include both the full set of the spouse's smoking controls and the spouse's smoking polygenic scores and their genetic principal components. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

smoking, parental risky behaviors, or parental mortality. Spousal smoking behavior plays a role, but does not fully explain the estimated relationship. In this section, we investigate other potential non-smoking channels through which the smoking polygenic scores may operate including risk preferences, longevity expectations, planning horizons, and impulsivity. Prior studies suggest smokers and non-smokers differ along these margins; we explore whether those with a higher genetic predisposition for smoking differ along these characteristics.[21]

[21]For the results presented in this section, we use the estimation sample for the summary standardized index of health outcome (i.e., those with non-missing variables for all of the individual health measures that compose the index). Results are robust if we instead use a larger sample consisting of anyone who has a non-missing value for any individual health measure we consider.

2.6.1 Risk Preferences

The first channel we investigate is risk preferences. Risk aversion is negatively and significantly associated with smoking, heavy drinking, and being overweight or obese (e.g., Barsky et al., 1997; Khwaja et al., 2006; Anderson and Mellor, 2008; Ida and Goto, 2009; Sutter et al., 2013). If individuals with higher genetic risk for smoking tend to be less risk averse (i.e., more risk prone), they may engage in fewer health-promoting behaviors and more risky behaviors (besides smoking), which adversely impact health. We explore whether there is a relationship between risk preferences and the smoking polygenic scores.

From 1998–2008, the HRS elicited information on risk tolerance based on hypothetical income gambles. Individuals make a choice between a guaranteed stream of income and a 50-50 gamble that doubles that amount of income or cuts it by various amounts (10, 20, 33, 50, and 75 percent). The questions are asked such that they separate individuals into six distinct risk aversion (RA) categories ordered from least (category 1) to most (category 6) risk averse.[22] We create an indicator variable equal to one if the individual always makes the most risk averse decision in the income gambles (i.e., RA category 6) as well as separate indicators for an RA category greater than or equal to 5, 4, 3, or 2. For example, those with an RA category greater than or equal to 4 compose the most, second most, and third most risk averse individuals.

We make use of other risk-related questions available in the 2014 and 2016 survey waves. The HRS asks "Are you generally a person who tries to avoid taking risks or one who is fully prepared to take risks?" Respondents answer on a 0–10 scale, where 0 means not willing to take any risks and 10 means fully willing to take risks. We create an indicator variable for whether respondents are generally *not* willing to take risk, corresponding to a response of 0 or 1 on the 0–10 scale, as well as an indicator variable for being very willing to take risk, corresponding to a response of 9 or 10. We also consider the 0–10 score itself as an outcome.[23]

Table 2.8 presents the estimated relationship between various measures of risk aversion and the smoking polygenic scores. Panels A, B, and C show the results for the full, ever smoker, and never smoker samples, respectively. Generally, we find little to no evidence that genetic risk for smoking correlates significantly with risk preferences. One exception is that the CPD score is associated with a higher likelihood of exhibiting the highest degree of risk aversion based on the income gambles, especially for never smokers. If anything, this result runs counter to the hypothesis above that the relationship between genetic endowments for smoking and health is explained by less risk aversion among those with higher polygenic smoking scores. Overall, the results do not reveal a systematic relationship between risk preferences and

[22] The least risk averse category corresponds to those who would take a job with even chances of doubling their income or cutting it by 75 percent, while the most risk averse category corresponds to always choosing the guaranteed stream of income. Risk categories 2, 3, 4, and 5 consist of those who would take a job with even chances of doubling income or cutting it by 50 percent, a third, 20 percent, and 10 percent, respectively.

[23] The HRS also asks "How willing are you to take risks [while driving/in financial matters/during leisure and sport/in your occupation/with your health]?" These questions were asked only in the 2014 and 2016 waves via a leave-behind questionnaire that participants complete and return by mail; thus, the sample for analyzing these measures is relatively small. We find no significant correlation between the smoking polygenic scores and measures of risk aversion for these specific activities. Results are available by request.

Table 2.8: Relationship between the Smoking Polygenic Scores and Risk Preferences

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Risk Aversion Category (Income Gamble)					General Willingness to Take Risk		
	$=6$	≥ 5	≥ 4	≥ 3	≥ 2	Very Unwilling	Very Willing	0-10 Score
	Panel A: Full Sample							
CPD Score	0.013**	0.008	0.006	0.004	-0.001	0.002	0.002	-0.016
	(0.006)	(0.006)	(0.005)	(0.004)	(0.003)	(0.003)	(0.004)	(0.037)
Initiation Score	0.008	-0.002	0.003	0.005	0.001	0.002	-0.001	0.000
	(0.006)	(0.006)	(0.005)	(0.004)	(0.003)	(0.004)	(0.005)	(0.040)
Full Smoking Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Sample Mean	0.379	0.602	0.772	0.872	0.948	0.045	0.067	5.878
N	9,211	9,211	9,211	9,211	9,211	5,518	5,518	5,518
R^2	0.046	0.046	0.043	0.048	0.045	0.061	0.057	0.073
	Panel B: Ever Smoker Sample							
CPD Score	0.009	0.004	0.008	0.006	-0.002	0.002	0.003	-0.043
	(0.008)	(0.008)	(0.007)	(0.005)	(0.003)	(0.005)	(0.006)	(0.051)
Initiation Score	0.008	0.002	0.002	0.006	0.004	-0.001	0.006	0.050
	(0.008)	(0.008)	(0.007)	(0.006)	(0.004)	(0.006)	(0.006)	(0.057)
Full Smoking Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Sample Mean	0.385	0.601	0.765	0.862	0.944	0.049	0.073	5.863
N	5,346	5,346	5,346	5,346	5,346	3,011	3,011	3,011
R^2	0.068	0.065	0.059	0.065	0.065	0.107	0.102	0.117
	Panel C: Never Smoker Sample							
CPD Score	0.020**	0.013	0.002	0.002	-0.001	0.001	0.004	0.036
	(0.009)	(0.009)	(0.007)	(0.006)	(0.004)	(0.005)	(0.006)	(0.054)
Initiation Score	0.005	-0.004	0.005	0.004	-0.004	0.006	-0.006	-0.060
	(0.010)	(0.010)	(0.008)	(0.007)	(0.005)	(0.005)	(0.007)	(0.058)
Sample Mean	0.371	0.604	0.783	0.885	0.954	0.040	0.061	5.896
N	3,865	3,865	3,865	3,865	3,865	2,507	2,507	2,507
R^2	0.040	0.049	0.048	0.046	0.042	0.041	0.036	0.057

Note: Each column within a panel presents results from a separate regression. The outcomes in columns (1)–(5) are based on respondents' choice between a guaranteed stream of income and a 50-50 gamble that doubles that amount of income or cuts it by various amounts. Individuals are grouped into six categories from least (category 1) to most (category 6) risk averse. Category 6 corresponds to always choosing the guaranteed stream of income, and categories 1, 2, 3, 4, and 5 consist of those who would take a job with even chances of doubling income or cutting it by 75%, 50%, a third, 20%, and 10%, respectively. The outcomes in columns (6)–(8) are based on respondents' answers on a 0–10 scale to whether they avoid taking risks or are fully prepared to take risks. In column (6), the outcome is an indicator for not willing to take risk (response of 0 or 1). In column (7), the outcome is an indicator for very willing to take risk (response of 9 or 10). In column (8), the outcome is the score itself. Results for the full sample are presented in Panel A, for the ever smoker sample in Panel B, and for the never smoker sample in Panel C. In all specifications, we include the standard controls and the genetic principal components described in Section 2.4, and for the full sample and ever smoker sample, we always include the full set of smoking controls described in Section 2.4. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

the polygenic scores; thus, risk tolerance does not appear to explain the estimated relationship between the smoking polygenic scores and health.

2.6.2 Longevity Expectations

Longevity expectations are another channel that may explain the correlation between the smoking polygenic scores and health. An increase in expected longevity should increase the incentive to invest in health, a mechanism referred to as the "Mickey Mantle effect" (Fang et al., 2007). As explained in Fang et al. (2007), if one expects to live longer, there is more incentive to lead a healthier lifestyle to improve quality of life in old age.[24] A priori, it is unclear how genetic risk for smoking correlates with longevity expectations, though the literature has documented systematic differences in such beliefs by smoking status (e.g., Khwaja et al., 2007b; Wang, 2014; Bissonnette et al., 2017). For example, Khwaja et al. (2007b) use the HRS and find smokers' longevity expectations are relatively optimistic (compared to objectively estimated probabilities) while never smokers are relatively pessimistic.[25] We explore whether the genetic risk for smoking correlates with longevity expectations (though not the accuracy of those beliefs). Given the "Mickey Mantle effect" above, those who expect to live longer may make larger investments in health and engage in more health-promoting activities, which would lead to better health outcomes.

In each survey wave, the HRS asks respondents younger than 65 about the probability of living to age 75, with answers on a 0–100 scale. We examine how these responses correlate with the genetic endowments for smoking. Results for the full sample, ever smokers, and never smokers are presented in Table 2.9. We find a one standard deviation increase in the CPD score decreases the self-reported probability of living to age 75 by about 1–1.8 percentage points and the estimates are significant at the 1 percent level. The magnitude of the decline is larger in the ever smoker sample. We do not find a statistically significant relationship between the initiation score and the self-reported probability of living to age 75 in any of the samples.

Since we find evidence of a correlation between longevity expectations and genetic risk for smoking, we reestimate the health specifications additionally including the respondent's self-reported probability of living to age 75. The results are presented in Table 2.10. An increase in the self-reported probability of living to age 75 is associated with an improvement in the summary health index, consistent with the "Mickey Mantle effect." Furthermore, the inclusion of longevity expectations decreases the magnitude of the point estimate on the CPD score across all samples by about 15–25 percent and leaves the initiation score point estimate largely unchanged. Thus, the negative association between longevity expectations and the CPD score explains part of the estimated relationship between the CPD score and health. However,

[24] In a similar vein, Picone et al. (2004) find individuals who expect to live longer are more likely to undergo screenings for early detection of breast and cervical cancer.

[25] Wang (2014) finds important differences when comparing adult smokers' own subjective longevity expectations to objective ones estimated using a rational expectations framework. In particular, they attach less weight to their health conditions and smoking choices and more weight to factors like age, race, and parents' longevity. Bissonnette et al. (2017) find at the median, smokers and non-smokers in the HRS correctly perceive their remaining life expectancy, but the distributions have heavy tails, such that on average, smokers are too optimistic.

Table 2.9: Relationship between the Smoking Polygenic Scores and Self-Reported Probability of Living to Age 75

	Full Sample	Ever Smoker Sample	Never Smoker Sample
	(1)	(2)	(3)
CPD Score	-1.421***	-1.752***	-1.018***
	(0.227)	(0.313)	(0.329)
Initiation Score	0.029	0.420	-0.429
	(0.248)	(0.339)	(0.361)
Full Smoking Controls	Yes	Yes	No
Sample Mean	66.672	64.362	69.806
N	42,001	24,182	17,819
R^2	0.082	0.109	0.028

Note: Each column presents results from a separate regression where the outcome is the self-reported probability of living to age 75. Results are presented for the full sample and then separately for ever smokers and never smokers. In all specifications, we include the standard controls and the genetic principal components described in Section 2.4, and for the full sample and ever smoker sample, we always include the full set of smoking controls described in Section 2.4. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

there still remains a non-trivial, positive, and significant link between both smoking scores and health after accounting for longevity expectations.

2.6.3 Planning Horizons

Another mechanism through which the smoking polygenic scores may operate is time preferences. Those with higher discount rates (i.e., more impatient, less future-oriented) derive less value from future health and are less likely to invest in their health and more likely to engage in risky behaviors.[26] Indeed, some studies have found a significant association between higher rates of time preference and smoking (e.g., Khwaja et al., 2006, 2007b; Scharff and Viscusi, 2011).[27] Using data from the Survey on Smoking, Khwaja et al. (2007b) find subjective rates of time discounting obtained via committed choice scenarios do not differ by smoking status, but more general measures of self-control, impulsivity, and financial planning do. In particular, those who are more impulsive and plan less for the future are more likely to smoke. They conclude that smoking "may be a marker for greater problems of self-control..."

[26] For example, Picone et al. (2004) find those with lower time preference, as measured by financial planning horizons, are more likely to undergo cancer screening.

[27] Scharff and Viscusi (2011) examine workers' fatality risk-wage decisions in the labor market and find smokers have significantly higher rates of time preference than non-smokers with respect to years of life. Khwaja et al. (2006) find smokers in the HRS are more impatient than non-smokers, measured indirectly in terms of their financial planning horizons.

Table 2.10: Relationship between the Smoking Polygenic Scores and the Summary Standardized Index of Health Accounting for Longevity Expectations

	Full Sample		Ever Smoker Sample		Never Smoker Sample	
	(1)	(2)	(3)	(4)	(5)	(6)
CPD Score	0.036***	0.029***	0.038***	0.030***	0.031***	0.027***
	(0.004)	(0.004)	(0.006)	(0.006)	(0.006)	(0.006)
Initiation Score	0.020***	0.020***	0.010	0.012**	0.030***	0.028***
	(0.005)	(0.005)	(0.007)	(0.006)	(0.007)	(0.006)
Probability of Living to 75		-0.005***		-0.005***		-0.005***
		(0.000)		(0.000)		(0.000)
Full Smoking Controls	Yes	Yes	Yes	Yes	No	No
Sample Mean	-0.009	-0.009	0.029	0.029	-0.061	-0.061
N	42,001	42,001	24,182	24,182	17,819	17,819
R^2	0.115	0.191	0.141	0.215	0.075	0.151

Note: Each column presents results from a separate regression where the outcome is the summary standardized health index. Results are presented for the full sample and then separately for ever smokers and never smokers. In all specifications, we include the standard controls and the genetic principal components described in Section 2.4. We also include the full set of smoking controls described in Section 2.4 for the full sample and ever smoker samples. In columns (2), (4), and (6), we add controls for the individual's self-reported probability of living to age 75. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

We explore whether and how the smoking polygenic scores associate with financial planning horizons, noting that these horizons likely capture a combination of present (versus future) orientation, planning abilities, and/or more general aspects of self-control. We consider personality measures related to conscientiousness in the next subsection.

The HRS asks individuals "In deciding how much of their (family) income to spend or save, people are likely to think about different financial planning periods. In planning your (family's) saving and spending, which of the following time periods is most important to you (and your husband/and your wife/and your partner), the next few months, the next year, the next few years, the next 5–10 years, or longer than 10 years?" Similar to Barth et al. (2020), we construct indicator variables that equal one if the respondent's financial planning horizon is longer than next year, next few years, next 5–10 years, or longer than 10 years, respectively, and zero otherwise.[28]

Table 2.11 presents the estimated relationship between planning horizons and the smoking polygenic scores for the full sample (Panel A), ever smokers (Panel B), and never smokers (Panel C). In the full sample, a one standard deviation increase in the CPD score or the initiation score is significantly associated with about a 0.8 percentage point decrease in the probability of reporting planning horizons greater than one

[28]The financial planning horizon question is not asked in all waves nor to all respondents. Khwaja et al. (2006) provide details on the availability of this information throughout the survey waves.

Table 2.11: Relationship between the Smoking Polygenic Scores and Financial Planning Horizon

	(1) PH $\geq$ Next Year	(2) PH $\geq$ Few Years	(3) PH $\geq$ 5-10 Years	(4) PH $\geq$ 10 Years
		Panel A: Full Sample		
CPD Score	-0.008***	-0.008**	-0.007*	0.001
	(0.003)	(0.004)	(0.004)	(0.003)
Initiation Score	-0.007**	-0.009**	-0.005	-0.002
	(0.003)	(0.004)	(0.005)	(0.003)
Full Smoking Controls	Yes	Yes	Yes	Yes
Sample Mean	0.880	0.777	0.503	0.140
N	18,878	18,878	18,878	18,878
R^2	0.034	0.031	0.033	0.021
		Panel B: Ever Smoker Sample		
CPD Score	-0.008**	-0.007	-0.011*	-0.003
	(0.004)	(0.005)	(0.006)	(0.004)
Initiation Score	-0.005	-0.008	-0.005	-0.002
	(0.004)	(0.005)	(0.006)	(0.004)
Full Smoking Controls	Yes	Yes	Yes	Yes
Sample Mean	0.869	0.761	0.492	0.131
N	10,810	10,810	10,810	10,810
R^2	0.053	0.044	0.046	0.034
		Panel C: Never Smoker Sample		
CPD Score	-0.006	-0.007	-0.004	0.005
	(0.004)	(0.006)	(0.007)	(0.005)
Initiation Score	-0.007	-0.008	-0.004	-0.003
	(0.005)	(0.006)	(0.007)	(0.005)
Sample Mean	0.895	0.798	0.519	0.151
N	8,068	8,068	8,068	8,068
R^2	0.020	0.022	0.031	0.021

Note: Each column presents results from a separate regression where the outcome is an indicator for financial planning horizon (PH) length. Results are presented for the full sample and then separately for ever smokers and never smokers. In all specifications, we include the standard controls and the genetic principal components described in Section 2.4, and for the full sample and ever smoker sample, we always include the full set of smoking controls described in Section 2.4. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

year or the next few years. The point estimates in the ever and never smoker samples are similar to those in the full sample but generally not statistically significant. The exception is a higher CPD score is associated with a significant decline in the probability of ever smokers having a planning horizon longer than one year. Thus, there is some evidence that a higher genetic risk for smoking correlates with shorter planning

Table 2.12: Relationship between the Smoking Polygenic Scores and the Summary Standardized Index of Health Accounting for Financial Planning Horizons

	Full Sample		Ever Smoker Sample		Never Smoker Sample	
	(1)	(2)	(3)	(4)	(5)	(6)
CPD Score	0.040***	0.038***	0.042***	0.041***	0.034***	0.033***
	(0.005)	(0.005)	(0.008)	(0.008)	(0.007)	(0.007)
Initiation Score	0.024***	0.022***	0.015*	0.014*	0.032***	0.031***
	(0.006)	(0.006)	(0.008)	(0.008)	(0.008)	(0.008)
PH = Next Year		-0.101***		-0.102***		-0.098***
		(0.017)		(0.023)		(0.025)
PH = Next Few Years		-0.157***		-0.162***		-0.144***
		(0.015)		(0.020)		(0.022)
PH = Next 5-10 Years		-0.188***		-0.192***		-0.175***
		(0.015)		(0.020)		(0.022)
PH > 10 years		-0.184***		-0.185***		-0.172***
		(0.017)		(0.023)		(0.025)
Full Smoking Controls	Yes	Yes	Yes	Yes	No	No
Sample Mean	0.017	0.017	0.064	0.064	-0.045	-0.045
N	18,878	18,878	10,810	10,810	8,068	8,068
R^2	0.123	0.140	0.150	0.166	0.077	0.094

Note: Each column presents results from a separate regression where the outcome is the summary standardized health index. Results are presented for the full sample and then separately for ever smokers and never smokers. In all specifications, we include the standard controls and the genetic principal components described in Section 2.4. We also include the full set of smoking controls described in Section 2.4 for the full sample and ever smoker samples. In columns (2), (4), and (6), we add dummy variables for the individual's financial planning horizon (PH). * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

horizons. We reestimate our baseline health specifications including mutually exclusive indicator variables for the individual's reported financial planning horizon. Results are presented in Table 2.12. Longer financial planning horizons are associated with better health (i.e., a lower summary health index), but the inclusion of these horizons decreases the magnitude of the CPD and initiation score coefficients only slightly. Thus, planning horizons play a very small role in explaining the link between the smoking polygenic scores and health.

Table 2.13: Relationship between the Smoking Polygenic Scores and Impulsivity and Conscientiousness

	Full Sample			Ever Smoker Sample			Never Smoker Sample		
	(1) Big 5 (1-4)	(2) MPQ Score (1-6)	(3) RCSG Score (1-6)	(4) Big 5 (1-4)	(5) MPQ Score (1-6)	(6) RCSG Score (1-6)	(7) Big 5 (1-4)	(8) MPQ Score (1-6)	(9) RCSG Score (1-6)
CPD Score	-0.019***	-0.046***	-0.036***	-0.021**	-0.016	-0.015	-0.017*	-0.080***	-0.062***
	(0.007)	(0.016)	(0.011)	(0.010)	(0.023)	(0.016)	(0.009)	(0.023)	(0.015)
Initiation Score	-0.015**	-0.041**	-0.020*	-0.001	-0.031	-0.030*	-0.030***	-0.048**	-0.001
	(0.007)	(0.017)	(0.011)	(0.011)	(0.025)	(0.016)	(0.010)	(0.023)	(0.016)
Full Smoking Controls	Yes	Yes	Yes	Yes	Yes	Yes	No	No	No
Sample Mean	3.280	4.318	4.737	3.235	4.259	4.655	3.333	4.389	4.833
N	5,565	3,255	3,157	2,999	1,763	1,707	2,566	1,492	1,450
R^2	0.112	0.090	0.137	0.148	0.150	0.186	0.079	0.081	0.116

Note: Each column presents results from a separate regression. The outcome in columns (1), (4), and (7) is the Big 5 conscientiousness score, which ranges from 1 (less conscientious)–4 (more conscientious). The outcome in columns (2), (5), and (8) is the conscientiousness score from the Multidimensional Personality Questionnaire (MPQ), which ranges from 1 (less conscientious)–6 (more conscientious). The outcome in columns (3), (6), and (9) is the conscientiousness score from Roberts et al. (2005) (the RCSG score) which ranges from 1 (less conscientious)–6 (more conscientious). More details on these outcomes are provided in Section 2.6.4. Results are presented for the full sample and then separately for ever smokers and never smokers. In all specifications, we include the standard controls and the genetic principal components described in Section 2.4, and for the full sample and ever smoker sample, we always include the full set of smoking controls described in Section 2.4. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

2.6.4 Impulsivity and Conscientiousness

The final channels we explore are impulsivity and conscientiousness. Khwaja et al. (2007a) find current and former smokers are significantly more impulsive than never smokers based on an index of impulsivity that measures an individual's ability to set goals and exercise self-control from the Survey on Smoking.[29] Studies have found conscientiousness, one of the "big five" personality traits, is a good proxy for self-control (Ameriks et al., 2007) and is negatively correlated with smoking (e.g., Terracciano and Costa Jr, 2004; Malouff et al., 2006). Conscientiousness is also positively correlated with a range of health-promoting behaviors like seeking preventative care and healthy eating (see reviews by Bogg and Roberts, 2004, 2013). If the genetic endowments for smoking reflect impulsiveness and conscientiousness, these characteristics may be important mediators of the relationship between the smoking polygenic scores and health.

In various waves, the HRS ask questions related to personality traits to subsets of the sample via leave-behind questionnaires that respondents mail back to the HRS. We construct a variety of measures based on these questions. From 2010–2016, 31 items assessed the "big five" personality traits of neuroticism, extraversion, openness to experience, agreeableness, and conscientiousness. The survey asked respondents to indicate how well various adjectives described them on a scale of 1 (a lot) to 4 (not at all). We focus on conscientiousness, which is based on the following adjectives: organized, responsible, hardworking, care-

[29] The index is based on the degree to which individuals agree with statements like "I make hasty decisions," "I do not control my temper," "I act on impulse."

Table 2.14: Relationship between the Smoking Polygenic Scores and the Summary Standardized Index of Health Accounting for Conscientiousness

	(1)	(2)	(3)	(4)	(5)	(6)
			Panel A: Full Sample			
CPD Score	0.034***	0.028***	0.036***	0.032***	0.039***	0.034***
	(0.008)	(0.008)	(0.009)	(0.009)	(0.008)	(0.008)
Initiation Score	0.023**	0.019**	0.027***	0.023**	0.032***	0.029***
	(0.009)	(0.009)	(0.009)	(0.009)	(0.009)	(0.009)
Big 5 Conscientiousness Score		-0.274***				
		(0.021)				
MPQ Conscientiousness Score				-0.096***		
				(0.010)		
RCSG Conscientiousness Score						-0.152***
						(0.016)
Full Smoking Controls	Yes	Yes	Yes	Yes	Yes	Yes
Sample Mean	0.066	0.066	0.056	0.056	0.056	0.056
N	5,565	5,565	3,255	3,255	3,157	3,157
R^2	0.137	0.181	0.148	0.174	0.161	0.191
			Panel B: Ever Smoker Sample			
CPD Score	0.036***	0.029**	0.034**	0.032**	0.036***	0.034**
	(0.012)	(0.012)	(0.013)	(0.013)	(0.013)	(0.013)
Initiation Score	0.016	0.016	0.026*	0.023	0.037***	0.033**
	(0.014)	(0.013)	(0.015)	(0.014)	(0.014)	(0.014)
Big 5 Conscientiousness Score		-0.300***				
		(0.030)				
MPQ Conscientiousness Score				-0.110***		
				(0.015)		
RCSG Conscientiousness Score						-0.134***
						(0.023)
Full Smoking Controls	Yes	Yes	Yes	Yes	Yes	Yes
Sample Mean	0.129	0.129	0.110	0.110	0.104	0.104
N	2,999	2,999	1,763	1,763	1,707	1,707
R^2	0.169	0.215	0.185	0.214	0.213	0.233
			Panel C: Never Smoker Sample			
CPD Score	0.031***	0.027**	0.038***	0.031***	0.043***	0.032***
	(0.011)	(0.010)	(0.011)	(0.011)	(0.011)	(0.011)
Initiation Score	0.029**	0.022*	0.027**	0.023*	0.023*	0.023*
	(0.012)	(0.012)	(0.012)	(0.012)	(0.012)	(0.012)
Big 5 Conscientiousness Score		-0.239***				
		(0.029)				
MPQ Conscientiousness Score				-0.083***		
				(0.014)		
RCSG Conscientiousness Score						-0.172***
						(0.023)
Sample Mean	-0.008	-0.008	-0.009	-0.009	-0.001	-0.001
N	2,566	2,566	1,492	1,492	1,450	1,450
R^2	0.092	0.133	0.116	0.139	0.122	0.166

Note: Each column within a panel presents results from a separate regression where the outcome is the summary standardized health index. Results for the full sample are presented in Panel A, for the ever smoker sample in Panel B, and for the never smoker sample in Panel C. In all specifications, we include the standard controls and the genetic principal components described in Section 2.4. We also include the full set of smoking controls described in Section 2.4 for the full sample and ever smoker samples. In columns (2), (4), and (6), we add controls for the Big 5 conscientiousness score (1–4), the MPQ conscientiousness score (1–6), and the RCSG conscientiousness score (1–6), respectively. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

less, thorough, reckless, self-disciplined, impulsive, cautious, and thrifty.[30] Scales of the original adjectives are reversed so that 1 corresponds to not at all and 4 corresponds to a lot except for the careless, reckless, and impulsive adjectives. We then average the scores across the conscientiousness adjectives and we refer to this measure as the big five conscientiousness score. The score ranges from 1–4, with a higher score indicating a higher degree of conscientiousness. We also create a score based on items the HRS included in 2010 and 2012 from the Multidimensional Personality Questionnaire (hereafter, the MPQ conscientiousness score) as well as a score based on an inventory developed in Roberts et al. (2005) (hereafter, RCSG conscientiousness score) that taps into six facets of conscientiousness (self-control, traditionalism, responsibility, industriousness, order, and virtue) included in the HRS in 2008 and 2010. Both the MPQ and RCSG conscientiousness scores range from 1–6 and a higher score indicates a higher degree of conscientiousness.[31]

In Table 2.13, we present estimated associations between the smoking polygenic scores and the various conscientiousness measures. For the full sample (columns 1–3), we find the CPD score is significantly and negatively correlated with the three measures we consider. That is, a higher genetic risk for heavy smoking is correlated with a lower degree of conscientiousness (i.e., more impulsiveness). We also find a negative relationship between the initiation score and the conscientiousness scores, though the point estimates are smaller and somewhat less precisely estimated. When we turn to the ever and never smoker samples, the picture is more complex. We always estimate a negative correlation between the polygenic scores and the conscientiousness measures, but the point estimates tend to be larger and more precisely estimated in the never smoker sample.

Given the negative correlation between the smoking polygenic scores and the conscientiousness scores, we reestimate our health specifications including the conscientiousness measures. Results are shown in Table 2.14. Notably, conscientiousness has a large and statistically significant negative correlation with the summary standardized index of health. A one unit increase in the conscientiousness measures is associated with a 0.1–0.3 standard deviation decline (i.e., improvement) in the health index. Generally, the inclusion of the conscientiousness scores decreases the magnitude of the smoking polygenic score point estimates. Thus, the observed relationship between the smoking genetic endowments and health is mediated, in part, by impulsiveness and conscientiousness as measured through the personality questions in the HRS. However, there still remains a statistically significant relationship between the smoking polygenic scores and health, and this relationship is particularly robust for the CPD score.

[30] In 2006 and 2008, the conscientiousness scale included only five adjectives (organized, responsible, hardworking, careless, thorough). The HRS added adjectives in 2010 to expand coverage of the sub-facets of conscientiousness. We prefer the scale based on the expanded set of items.

[31] The MPQ score is based on how strongly a respondent agrees with statements like "I often stop one thing before completing it and start another," "I often act without thinking," "I am often not as cautious as I should be." The RCSG score is based on how strongly a respondent agrees with statements like "I rarely jump into something without first thinking about it," "I hardly ever lose or misplace things," "I have high standards and work toward them." See Smith et al. (2017) for a detailed description of the various measures and their construction.

2.6.5 Synthesis

We next explore how much of the association between the genetic endowments for smoking and health remains after including controls for risk aversion, longevity expectations, planning horizons, and conscientiousness simultaneously. Results are shown in Table 2.15. The inclusion of these potential mechanisms dampens the associations between the summary health index and the CPD score, but by less than 25 percent. Similarly, for never smokers, over 75 percent of the association between the initiation score and the health index remains. We then control for these mechanisms as well as parental and spousal variables, and again find that about 75 percent of the association between the CPD score and health summary measure remains. For never smokers, the association between the initiation score shrinks by 45 percent and is no longer statistically significant. In sum, much of the relationship between the genetic endowments for smoking and health remains after controlling for an array of potential mediating variables.

2.7 Robustness Exercises

We next explore the robustness of our results to different specifications and samples. For the sake of brevity, we present results for the summary standardized health index. Results for the individual health outcomes are available by request.

We first examine whether the associations between the genetic endowments for smoking and health are robust to alternate polygenic scores. In particular, we consider the CPD and initiation scores built from a 2010 GWAS conducted by the Tobacco and Genetics Consortium (The Tobacco and Genetics Consortium, 2010) that used a much smaller discovery sample compared to Liu et al. (2019). The results are presented in Appendix Table B13 Panel A. The estimated associations are smaller in magnitude but qualitatively similar to those in Table 2.2, which use the scores built from Liu et al. (2019). We still find the CPD score is associated with worse health for both ever and never smokers, and a higher initiation score is also associated with worse health for never smokers.

In addition to smoking initiation and heaviness, Liu et al. (2019) identified genetic variants associated with smoking cessation and age of initiation of regular smoking. We do not consider these polygenic scores in our main analysis for several reasons. First, the older polygenic scores available in the HRS, which we used above to explore the robustness of our results, are only constructed for smoking initiation and smoking intensity. Second, in the Liu et al. (2019) GWAS, the scores related to smoking cessation and age of smoking initiation were substantially less predictive in- and out-of-sample than the smoking initiation and smoking intensity scores, explaining about 1 percent of the variation in their respective outcomes in the HRS (versus 4 percent for the initiation and CPD scores). For the sake of completeness, we present results including all four smoking-related polygenic scores based on the GWAS estimates in Liu et al. (2019). A higher cessation score implies a higher propensity for current smoking (rather than being a former smoker) and a higher initiation age score reflects a later age of initiation. The results are shown in Appendix Table B13 Panel B. Once we include the smoking controls, we find no significant association between the initiation age score and the summary health index. A higher cessation score (a

Table 2.15: Relationship between the Smoking Polygenic Scores and the Summary Standardized Index of Health Accounting for Risk Aversion, Longevity Expectations, Planning Horizons, and Conscientiousness Simultaneously

	Full Sample		Ever Smoker Sample		Never Smoker Sample	
	(1)	(2)	(3)	(4)	(5)	(6)
CPD Score	0.037***	0.029***	0.043***	0.033***	0.029***	0.024***
	(0.006)	(0.005)	(0.008)	(0.008)	(0.007)	(0.007)
Initiation Score	0.018***	0.013**	0.007	0.005	0.027***	0.021**
	(0.006)	(0.006)	(0.008)	(0.008)	(0.009)	(0.009)
Risk Aversion Controls	No	Yes	No	Yes	No	Yes
Longevity Controls	No	Yes	No	Yes	No	Yes
Planning horizon Controls	No	Yes	No	Yes	No	Yes
Self-control Controls	No	Yes	No	Yes	No	Yes
Full Smoking Controls	Yes	Yes	Yes	Yes	No	No
Standard Controls	Yes	Yes	Yes	Yes	Yes	Yes
Principal Components	Yes	Yes	Yes	Yes	Yes	Yes
Sample Mean	-0.027	-0.027	0.003	0.003	-0.065	-0.065
N	25,818	25,818	14,565	14,565	11,253	11,253
R^2	0.130	0.219	0.166	0.248	0.095	0.192

Note: Each column presents results from a separate regression where the outcome is the summary standardized health index. Results are presented for the full sample and then separately for ever smokers and never smokers. In all specifications, we include the standard controls and the genetic principal components described in Section 2.4. We also include the full set of smoking controls described in Section 2.4 for the full sample and ever smoker samples. In columns (2), (4), and (6), we account for risk aversion, longevity expectations, planning horizons, and conscientiousness simultaneously. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

lower propensity to quit) is significantly associated with worse health. The inclusion of the cessation and initiation age scores slightly dampens the point estimates on the CPD and initiation scores, but the main takeaways remain—the CPD score is correlated with worse health for both ever and never smokers, and the initiation score is correlated with worse health for the never smokers.

Our main estimation sample includes those aged 50–65. The full Social Security retirement age depends on one's birth year and some individuals in our sample have a full retirement age of 65. Furthermore, individuals may begin claiming Social Security retirement benefits as early as age 62, thus, our sample contains some individuals who are retired. Given the potential influence of retirement on health, we explore the robustness of our results to restricting the sample to those younger than the early retirement

age, i.e., those aged 50–61. The results are presented in Appendix Table B13 Panel C and they are nearly identical to the full sample results.

We also verified that our main estimates are robust to clustering standard errors at the household level rather than the individual level. We find little to no change in the estimated standard errors when clustering at the broader household level. Those results are available by request.

2.8 Discussion and Conclusion

We study how genetic endowments linked to smoking associate with health using rich data from the Health and Retirement Study. Among individuals aged 50–65, we find a higher genetic predisposition for smoking associates with worse health, even after flexibly controlling for individual smoking behavior and even among those who have never smoked. The results suggest the smoking polygenic scores correlate with health through non-smoking channels. Several of the health outcomes where we find robust and meaningful relationships (e.g., diabetes, arthritis, body mass index, obesity) have a strong link to health behaviors like diet and exercise. Our results, therefore, are consistent with the idea that the smoking polygenic scores capture a complex array of traits that correlate with less engagement in health-promoting activities. This hypothesis is further supported by our finding that a spouse's genetic predisposition toward heavy smoking adversely and independently correlates with one's own health. It is quite possible that a spouse's propensity toward health-degrading behaviors (e.g., poor diet, lack of exercise) could spillover negatively to own health.

An important advantage of the genetic data is we can learn who may be most affected by changes in behavior. While we find significant associations between the smoking polygenic scores and health for both ever and never smokers, the magnitudes vary across these groups and by one's place in the polygenic score distributions. In particular, those with initiation scores in the lowest tercile who never smoke exhibit better health relative to never smokers with scores in the middle of the distribution, while ever smokers with low initiation scores do not experience such benefits. The results also suggest that both ever smokers and never smokers with CPD scores in the top tercile experience worse health outcomes, but the deterioration in health is smaller for never smokers. Thus, efforts to prevent those with a high risk for heavy smoking from ever starting could have important health benefits beyond those directly associated with not smoking.

One concern with interpretation of our results involves pleiotropy. That is, genetic variants can have multiple functions and affect multiple outcomes. In our setting, one particular concern is that genetic markers that correlate with smoking also correlate with biological mechanisms that impact health outcomes (e.g., poor cholesterol metabolism or lipid transport). While we cannot dismiss this notion, we do not think it solely drives our results.[32] First, as noted above, several (though not all) of the genetic markers implicated in the Liu et al. (2019) GWAS are involved in systems related to neurotransmission, reward-related learning and memory, and stress response. Second, an increase in a spouse's CPD score is

[32] Liu et al. (2019) documents correlations between the smoking polygenic scores and polygenic scores for other outcomes and traits. Of note, the CPD and initiation scores are statistically significantly and positively correlated with polygenic scores for BMI and obesity, coronary artery disease, and proinsulin.

associated with declines in the other spouse's health, including among couples where neither member ever smoked. If the smoking polygenic scores operated solely through biological mechanisms, we should not observe spousal spillovers. Furthermore, to the extent that genetic variants that predict smoking overlap with those that predict other health outcomes, our analysis sheds light on the mechanisms through which those variants link to health outcomes.

We note some important limitations of our work. First, we are quick to caution that our results should be interpreted as descriptive, not causal, relationships. Second, we try to shed light on why those with higher smoking polygenic scores exhibit worse health, but there is much left to explore and understand. The health-smoking genes relationship is not explained by parental smoking, parental risky behaviors, or parental mortality. Spousal smoking behavior plays a modest role, and we find small to moderate roles for longevity expectations, planning horizons, and conscientiousness. A more complete understanding of the channels underlying the relationship between health and the smoking polygenic scores may allow policy-makers to better design policies aimed at improving health, particularly for those disproportionately endowed with a propensity to smoke. For example, to the extent the genetic endowments for smoking reflect differences in time preferences or impulsiveness, this has implications for the design of interventions like exercise incentives and workplace wellness programs.

Chapter 3

Genetic Endowments, Alzheimer's Disease, and Economic Outcomes with Nicholas Papageorge, Meghan Skira, and Kevin Thom

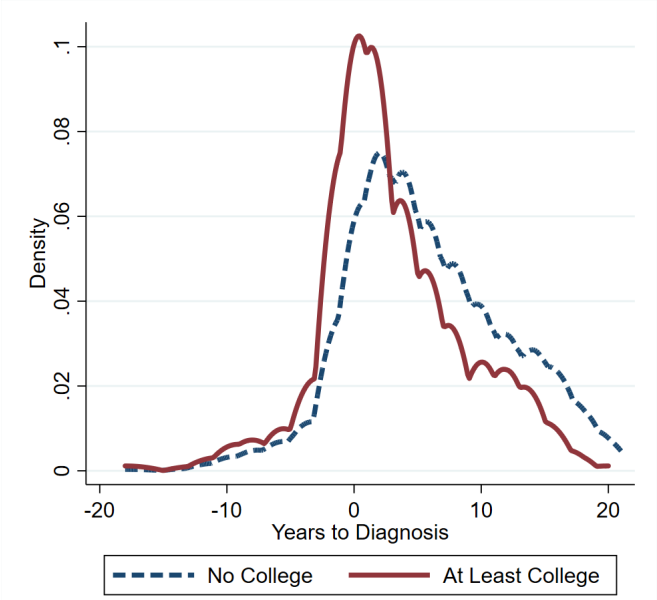
3.1 Introduction

As of 2022, 6.5 million Americans aged 65 and over have been diagnosed with Alzheimer's disease (AD), and this number is projected to reach 12.7 million by 2050 (Alzheimer's Association, 2022). The costs associated with AD are enormous and include direct medical expenditures, costs to informal caregivers, and financial errors, among others (e.g., Hurd et al., 2013; Coe et al., 2018; Nicholas et al., 2021). The direct medical costs alone reached $321 billion in 2022 (Alzheimer's Association, 2022). AD is a slowly progressive brain disease that includes three phases: pre-clinical AD, mild cognitive impairment, and Alzheimer's dementia. In the pre-clinical phase, cognitive symptoms such as memory loss are absent, but biological changes in the brain are present (e.g., beta-amyloid plaques, tau tangles). In the mild cognitive impairment phase, in addition to changes in the brain, problems with memory, language, and problem-solving emerge, but individuals can usually maintain independence in daily activities. The dementia phase involves severe cognitive impairment that increasingly limits an individual's ability to function independently (Alzheimer's Association, 2022; Chandra et al., ming). AD is typically clinically diagnosed during this third phase.[1] The progression of AD can take place over a long horizon, with studies finding that the brain changes can begin 20 years or more before cognitive symptoms emerge (Villemagne et al., 2013; Scharre, 2019).

In the absence of any proven cure for AD, it is important to understand when and how AD begins to affect cognitive and economic outcomes, and whether environmental or behavioral factors slow disease

[1] Until recently, the only way to confirm whether an individual had AD was after death via autopsy. Clinical diagnosis can now be accompanied by biomarker testing, such as brain scans, spinal taps to measure cerebrospinal fluid, and blood tests.

Figure 3.1: Years to Memory-Related Disease Diagnosis since First Observed Cognitive Impairment by Educational Attainment



Note: The figure shows a density plot of years to memory-related disease diagnosis since observed initial cognitive impairment by completed education among HRS respondents. Initial cognitive impairment is defined as the first wave where an HRS respondent registers a Langa-Weir score less than 12.

progression or insulate individuals from harm. A large literature pursues questions like these based on case-control designs that compare outcome trajectories of individuals eventually diagnosed with AD with those of people who are never diagnosed. A drawback to this approach is that the diagnosis of AD is itself an endogenous outcome. Figure 3.1 plots the distribution of years between the first signs of cognitive impairment and the diagnosis of a memory-related disease among diagnosed individuals in the Health and Retirement Study (HRS) separately by college attainment. Not only is there substantial dispersion in each distribution, but more educated individuals are diagnosed more quickly after the onset of cognitive decline. Observed trajectories of outcomes for individuals diagnosed with AD may therefore reflect the influence of factors that encourage diagnosis independent of disease severity.

We study the relationship between observed genetic risk for late-onset Alzheimer's disease and trajectories of cognition, memory-related disease, expectations, and economic outcomes in the HRS. Focusing on genetic risk is natural in this context because genetic factors play a large role in the etiology of AD. Twin studies suggest that genetic factors explain 60-80% of the variation in the risk of AD (Gatz et al., 2006). We examine two sets of genetic variables. The first is an individual's status as a carrier of the $\varepsilon 4$ allele of the Apolipoprotein E (APOE) gene. Having one copy of the APOE-$\varepsilon 4$ allele significantly increases the risk of developing AD, and having two copies increases this risk even further. We also study a polygenic score

for AD (hereafter the AD score), which is a linear index of genetic markers associated with AD (omitting those in the APOE gene) based on the genome-wide association study in Kunkle et al. (2019). An advantage of focusing on genetic endowments is that unlike eventual diagnosis, these are pre-determined at conception. Thus, we can observe outcomes and trajectories for those with varying underlying risk for AD, including individuals who are not diagnosed with AD or do not exhibit signs of cognitive decline based on standard measures.

We present three main sets of results. First, we establish the predictive power of our genetic measures in the HRS sample. Higher levels of the AD score and carrying the APOE-$\varepsilon 4$ allele both predicts lower cognitive functioning and a higher probability of being diagnosed with a memory-related disease. The AD genetic measures robustly predict cognitive functioning even after accounting for whether the individual has been or will be diagnosed with a memory-related disease, and carrying the APOE allele predicts diagnosis even after flexibly controlling for cognitive function. Thus, the genetic endowments for AD appear to contain important information about AD risk over and above standard measures of cognition and diagnosis. A crucial difference between APOE and the variants captured by the AD score emerges when looking at the timing of their associations with cognition. While differences in cognition by APOE genotype emerge late in the life-cycle, differences in cognition based on the AD score are present at all ages in our sample. This may suggest that these genetic risk factors operate through distinct mechanisms. Second, we find that a higher AD score is associated with worse economic outcomes, including a lower probability of working for pay, a higher probability of being retired, and lower total income. These relationships survive after controlling for cognitive function and diagnosis and among those who are never diagnosed or cognitively impaired while observed in the HRS.

Our third set of results centers on planning and expectations. Given that these genetic measures predict economically meaningful differences in cognition and economic outcomes, it is reasonable to ask whether individuals at greater genetic risk seem to be aware of their elevated risk and make medical or financial preparations to insulate themselves and their families from future economic losses. We find that those with higher AD scores are less likely to engage in a variety of planning activities, including holding long-term care insurance, life insurance, having a witnessed will, having assigned someone durable power of attorney, and having discussed future medical care with someone. These relationships likewise hold after controlling for cognitive function and diagnosis and among those who never experience cognitive impairment or diagnosis during the sample period. The planning outcome results are particularly worrisome, as they imply that those who have the most to gain from engaging in precautionary planning do not do so, and if anything, are less likely to do so. Furthermore, this lack of planning occurs even though some APOE carriers report a higher probability of future nursing home use.

A key advantage of the genetic data is that we can observe individuals with similar genetic propensity for AD but who differ in cognitive function or diagnosis status; therefore, we can explore factors that may protect high-risk individuals from cognitive impairment. Identifying such factors could inform interventions that slow cognitive decline and the development of AD. Beyond identifying protective factors, analyzing gene-environment interactions provides information about who is likely to be most affected by changes in behavior or the environment. We explore several interactions between the AD genetic measures

and various environmental influences, including educational attainment, childhood socioeconomic status, whether one's children live nearby, and whether one's parents have been diagnosed with a memory-related disease. Higher educational attainment and more advantaged childhood circumstances are associated with better cognitive function, in line with the "cognitive reserve" hypothesis (Roe et al., 2007; Meng and D'arcy, 2012), and we find modest evidence that they differentially protect those with more genetic risk for AD against cognitive decline. We find little evidence of significant interactions between the AD-related genes and the other environmental influences.

Our work contributes to several strands of literature. First, we contribute to the literature that documents substantial direct and indirect costs of AD on individuals and their families (e.g., Langa et al., 2001; Hurd et al., 2013; Zissimopoulos et al., 2015; Friedman et al., 2015; Coe et al., 2018). We show that genetic risk for AD, even among those without cognitive impairment or AD diagnosis, confers costs in terms of worse economic outcomes, such as lower employment and income, and lack of later-life planning. Second, we contribute to the literature on the impacts of AD and cognitive abilities on various economic outcomes (e.g., Triebel et al., 2009; Christelis et al., 2010; Agarwal and Mazumder, 2013; Hsu and Willis, 2013; Sudo and Laks, 2017; Martin et al., 2019b; Gresenz et al., 2020; Mazzonna and Peracchi, 2020; Nicholas et al., 2021; Li et al., 2022). Chandra et al. (ming) provides a comprehensive overview of this literature. These studies generally rely on observed diagnosis or cognitive function (e.g., memory performance), whereas we focus on genetic endowments for AD, avoiding concerns about selection into diagnosis or reliance on measures of cognition that may not reflect nuanced declines or changes in pathology. The fact that the associations between genetic risk for AD and various outcomes hold even after conditioning on cognitive function and diagnosis suggests that the genetic data contain useful information over and above these standard measures. Furthermore, most of these studies focus on financial outcomes and decisions (e.g., wealth, missed credit payments, credit scores, portfolio choices). We show that higher genetic risk for AD is negatively associated with a host of economic and planning measures. Third, our analysis relates to recent work that shows AD and dementia begin to have effects on financial decisions and wealth years before clinical diagnosis (e.g., Gresenz et al., 2020; Nicholas et al., 2021; Li et al., 2022). Consistent with these studies, we show that genetic endowments for AD are associated with worse economic outcomes and decreased planning activities from ages 50–85 and even among those not diagnosed and not experiencing cognitive impairment.

Last, our work relates to literature in economics that uses genetic data to understand AD as well as variation in complex social and economic behavior and outcomes. In a related study, Shin et al. (2019) finds HRS respondents with higher AD polygenic scores hold less wealth in and make smaller contributions to assets that require more active management, such as IRAs, and save more in "hands off" assets. Other work has largely explored relationships between the polygenic score for educational attainment (hereafter the EA score) and a host of behaviors such as educational attainment, earnings, health, and wealth (e.g., Belsky et al., 2016; Barth et al., 2020; Papageorge and Thom, 2020; Bolyard and Savelyev, 2021).

3.2 Background on Genetic Risk for Alzheimer's Disease

We rely on two measures of genetic risk for late-onset AD (i.e., onset after age 65)—the polygenic score for AD (the AD score) as well as whether an individual carries the Apolipoprotein E (APOE-$\varepsilon 4$) allele.[2] The AD score is a weighted index of genetic variants (single-nucleotide polymorphisms or SNPs) that are associated with AD. The weights come from genome-wide association studies (GWAS), where associations between individual SNPs and the outcome of interest (in our case, AD) are estimated via millions of regressions. The polygenic score (PGS) is given by:

$$PGS_i = \sum_{j=1}^{J} \tilde{\beta}_j SNP_i \tag{3.1}$$

where $\tilde{\beta}_j$ are the estimated coefficients from the GWAS and $SNP_i \in \{0, 1, 2\}$ measures the number of alleles individual i carries at SNP j. Intuitively, a PGS is a linear combination of SNPs and their association sizes with the outcome or trait of interest. The higher the PGS, the higher one's genetic risk for the trait or outcome.[3]

While several GWAS have implicated various genetic markers associated with late-onset AD, being a carrier of the APOE-$\varepsilon 4$ (hereafter APOE) is the strongest predictor of AD. Having one copy triples one's AD risk, while two copies leads to a 12–15-fold increase in risk (Liu et al., 2013; Michaelson, 2014). Those with the APOE allele generally exhibit the brain changes and cognitive symptoms associated with AD earlier than non-carriers. The APOE allele is expressed in more than half of diagnosed AD patients (Michaelson, 2014), but being a carrier is neither necessary nor sufficient to develop AD.

In our analysis, we rely on a late-onset AD polygenic score based on the GWAS of Kunkle et al. (2019) that includes all SNPs regardless of their p-values.[4] We follow the guidance of Ware et al. (2020) and use the AD score that excludes the APOE region and treat the APOE region as a separate measure of genetic risk for AD.[5] We provide more details on the AD score and our measures of APOE in the next section.

3.3 Data

We use data from the *Health and Retirement Study* (HRS), which follows a nationally representative sample of adults age 50 and over as well as their spouses in the United States. Individuals were first surveyed in 1992 and subsequent interviews have occurred biennially. The data include detailed information on

[2]The APOE gene provides instructions for making a protein that combines with fats and transports low-density lipids and removes cholesterol from the bloodstream.

[3]For more details on the human genome, we refer the reader to Beauchamp et al. (2011) and Benjamin et al. (2012), and for more details on polygenic scores, see Barth et al. (2020) and Papageorge and Thom (2020).

[4]In Kunkle et al. (2019), AD cases are those that were clinically-diagnosed or autopsy-documented. They do not use family history of Alzheimer's disease or dementia as a proxy for AD. Issues related to using "proxy" dementia cases are described in Escott-Price and Hardy (2022).

[5]As explained in Ware et al. (2020), including the APOE region in the AD polygenic score does not sufficiently account for the large risk attributed to the APOE region and it overstates the polygenic nature of AD.

demographics, health, employment, retirement, family structure, expectations, and financial and non-financial planning. We primarily use data from 1998–2018, as key measures regarding cognitive function and diagnosis of memory-related disease did not become available until the 1998 survey wave.

The HRS collected genetic samples from nearly 20,000 respondents over the course of four waves (2006, 2008, 2010, 2012). Our sample only includes these genotyped individuals. Furthermore, we only include individuals classified as genetic Europeans by the HRS and who self-identify as white because the polygenic score we use is based on the findings from a GWAS where the discovery sample consisted only of those of European ancestry (i.e., non-Hispanic whites). About 12,000 genotyped individuals have genetic European ancestry. The age range of our sample varies depending on the outcome we consider, but in many cases, we focus on those aged 50–85. A key exception is when we consider employment-related outcomes, in which case we limit the sample to those 50–70 years old.

3.3.1 Direct Outcomes: Cognition and Memory-Related Disease Diagnosis

We examine how genetic risk for AD associates with directly-related outcomes, namely cognitive functioning and diagnosis of memory-related disease. We rely on a summary cognition score, which we refer to as the Langa-Weir (L-W) score, and discrete classifications based on that score. Starting in the 1996 wave, the HRS includes a variety of tests and exercises to measure respondent cognition. We use a 27-point score that includes the following tests: (1) immediate and delayed recall test (0–20 points); (2) serial sevens subtraction test (0–5 points); (3) counting backward test (0–2 points). The L-W classifications are based on this 27-point score. Those with scores ranging from 12–27 are considered normal; those with scores from 7–11 are considered cognitively impaired but not demented; and those with scores from 0–6 are considered demented. More information on these categories can be found in Crimmins et al. (2011).[6] We rely on the score itself and create indicators for whether an individual has ever achieved an L-W score that corresponds with the impaired or demented categories, where "ever" means they registered such a score in the current survey wave or any prior wave. We create another indicator for whether the individual ever scored in the demented range.

Starting in 1998, HRS respondents were asked whether a doctor has ever told them they have a memory-related disease. In 2010, the question wording changed and respondents were asked whether a doctor has ever told them they have Alzheimer's disease or dementia. We create an indicator for being diagnosed with a memory-related disease (MRD) that is equal to one if individuals report a memory-related disease (prior to 2010) or Alzheimer's disease or dementia (in 2010 and after).

[6]We only include cognitive functioning measures of individuals who self-respond and exclude those who respond via proxy as proxy interviews do not include any direct assessment of cognition. While the measures used to classify HRS self-respondents as demented vary across studies, they generally rely on the tests included in the L-W score (Gianattasio et al., 2019). The three tests included in the L-W score are asked of individuals of all ages, whereas some tests are only asked to those aged 65 and older; hence, we prefer to rely on the L-W score as it is consistently measured across the ages we study. Additional information on the L-W classifications can be found here.

3.3.2 Economic Outcomes

We consider a variety of economic outcomes, including whether the individual currently works for pay as well as whether they are retired. An individual is retired if they currently do not work for pay and self-report they are completely retired. We also analyze log total individual income, which includes income from earnings, pensions, annuities, Social Security, unemployment and workers' compensation, and other government transfers.

3.3.3 Mortality and Attrition

We examine whether those with a genetic propensity for AD have higher mortality rates. Using information in the HRS on year of death, we create a mortality indicator equal to one if the individual dies in the next year and zero otherwise. We also consider attrition of individuals from one survey wave to the next (for any reason). We create an indicator that has value one if the respondent appears in the current wave and the following wave and zero if one appears in the current wave but not the following. Examining how genetic risk for AD associates with mortality and attrition is important for understanding the extent to which differential survival and survey response may impact our estimates. As we show, those with more genetic risk for AD have higher 1-year mortality rates and survey attrition rates. We therefore interpret our results as conservative, as they likely reflect those of a selected set of healthier individuals.

3.3.4 Expectations

We study whether genetic risk for AD correlates with expectations about mortality and long-term care utilization. In particular, the HRS asks respondents aged 65 and under about their expected probability of living to age 75 on a scale of 0–100. Starting in 1998, the HRS asks individuals aged 65 and older about their expected probability of moving to a nursing home in the next five years. The question is only asked to those not currently in a nursing home.

3.3.5 Planning Outcomes

We consider several measures related to later-life planning. We create an indicator for holding long-term care insurance (LTCI). We also create indicators for whether the respondent holds life insurance, has a witnessed will, has a living will (i.e., an advance healthcare directive), has assigned someone(s) durable power of attorney for healthcare, and whether they have ever discussed medical care if they were to become seriously ill in the future with anyone. The questions about living wills, durable power of attorney and discussing medical care are asked to those aged 65 and older, and are only available starting in the 2012 wave.

3.3.6 AD Score and APOE

As described earlier, we consider two measures of genetic risk for late-onset AD—the polygenic score for AD and whether an individual carries the APOE allele. The polygenic score is based on the Kunkle et al. (2019) GWAS and includes all SNPs regardless of their p-value. The score excludes the APOE region based on the recommendations in Ware et al. (2020). The AD score is normalized to have mean zero and standard deviation of one. We create two APOE-related indicators. The first takes value one if the individual carries at least one copy of APOE-$\varepsilon 4$, and the second takes value one if the individual has exactly two copies of the allele.[7]

3.3.7 Descriptive Statistics

In Table 3.1, we present summary statistics for our sample. For most measures, the statistics are shown for those aged 50–85. For employment-related outcomes, we only show statistics for those aged 50–70. The age ranges for some measures are dictated by who the HRS targeted certain questions to and are noted in the caption. About 60 percent of our sample is female and has over 13 years of education, on average. The majority of the sample has Langa-Weir scores in the normal range, with an average score of 16.4. Among those aged 50–85, 22 percent have ever registered a Langa-Weir score that corresponds to the impaired or demented categories, and about 3 percent have ever registered scores that correspond to being demented. Only about 2 percent of person-waves have ever been diagnosed with MRD.

By construction, the AD score has mean zero and standard deviation one. We also show the kernel-smoothed density of the AD score in our sample in Figure 3.2. About 26 percent of the person-waves in our sample have at least one copy of the APOE-$\varepsilon 4$ allele and 2 percent have exactly two copies, putting them at substantially increased risk for AD.

Those aged 65 and under report a 66 percent probability of living to age 75, on average, and those aged 65–85 report a 14 percent probability of moving to a nursing home in the next 5 years. A little over half the sample aged 50–70 works, and with the exception of long-term care insurance, about 60 percent of the sample engages in the planning activities we consider. Less than 15 percent of the sample holds LTCI.

3.4 Empirical Strategy

We estimate the following regression via OLS:

$$\begin{aligned} Y_{it} = \quad & \beta_0 + \beta_1 ADScore + \beta_2 \mathbb{1}(APOE\,copies \geq 1) \\ & + \beta_3 \mathbb{1}(APOE\,copies = 2) + \beta_4 X_{it} + \varepsilon_{it} \end{aligned} \tag{3.2}$$

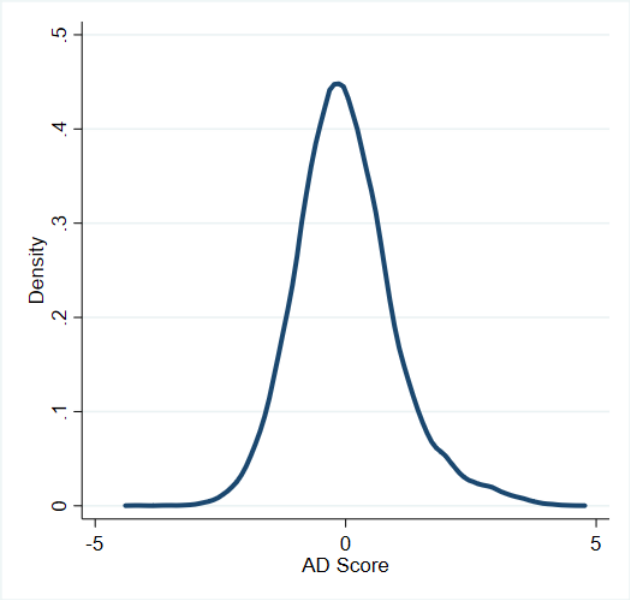
[7] The vast majority of our sample were directly genotyped for APOE. For a small fraction, their APOE status was imputed (either because there was insufficient DNA sample or their sample did not pass quality control for determining APOE). We follow the HRS's guidance here regarding which imputed values to include in the analysis.

Table 3.1: Summary Statistics

	Mean	SD	N
Demographics:			
Birth year	1940.245	9.789	88,191
Male	0.419	0.493	88,191
Age	67.537	9.001	88,191
Years of education	13.315	2.499	88,045
At least some college degree	0.322	0.467	88,045
Cognition and Memory-Related Disease (MRD):			
Langa-Weir (L-W) Score	16.393	4.004	88,191
Ever demented (L-W < 7)	0.028	0.165	88,191
Ever impaired or demented (L-W < 12)	0.217	0.412	88,191
Ever diagnosed with MRD	0.021	0.144	88,191
Genetic Data:			
AD Score	-0.009	1.000	88,191
APOE (At least 1 copy)	0.260	0.439	88,191
APOE (2 copies)	0.019	0.138	88,191
Expectations:			
Probability of living to age 75	66.294	26.385	36,415
Probability of moving to nursing home	14.449	20.302	49,350
Economic Outcomes:			
Currently working for pay	0.564	0.496	53,743
Retired	0.320	0.466	49,713
Log individual total income	10.007	0.964	81,520
Planning Outcomes:			
Holds long-term care insurance (LTCI)	0.148	0.356	86,864
Holds life insurance	0.677	0.468	87,576
Has a witnessed will	0.640	0.480	87,893
Has a living will	0.598	0.490	18,902
Has assigned durable power of attorney	0.596	0.491	18,942
Discussed future medical care with anyone	0.553	0.497	10,059
Environmental factors:			
Kids live within 10 miles	0.548	0.498	72,875
Mother's years of schooling	10.618	3.029	82,538
Mother's years of schooling ≥ 12	0.534	0.499	82,538
Either parent ever diagnosed with MRD	0.295	0.456	37,766
High childhood SES	0.740	0.439	86,962

Note: The table presents summary statistics at the person-wave level from 1998–2018. Except for the following outcomes, statistics are shown for those aged 50–85: probability of living to age 75 (ages 50–65); probability of moving to a nursing home (ages 65–85 and not currently residing in a nursing home); work for pay and retirement (ages 50–70); living will, durable power of attorney, and discussing future medical care (ages 65–85 from 2012 on).

Figure 3.2: Distribution of the Alzheimer's Disease Polygenic Score



Note: The figure shows the smoothed density of the polygenic score for Alzheimer's disease in our sample.

where Y_{it} denotes the outcome of individual i in survey wave t. $ADScore$ is the polygenic score for AD (that excludes the APOE region). We include an indicator for having at least one copy of the APOE-$\varepsilon 4$ allele as well as a separate indicator for having two copies. In this way, we allow for non-linear effects of the number of APOE copies an individual carries. Following Barth et al. (2020), X_{it} includes "standard controls"—birth year dummies, age dummies, survey wave dummies, a male dummy, and two-way interactions between the male dummy and the birth year dummies and age dummies. As is standard practice, X_{it} also includes the first 10 principal components of the genetic data to account for possible population stratification, and we allow those coefficients to vary by gender. We cluster standard errors at the individual level.

In some specifications, we include additional individual-level controls. In particular, we add dummy variables for each value of the current Langa-Weir score to flexibly control for cognitive function. We sometimes control for whether an individual has ever been diagnosed with MRD. We include these controls to learn whether the genetic endowments for AD have predictive power even after accounting for cognitive function and MRD diagnosis.

3.5 Main Results

3.5.1 Direct Outcomes

We first quantify the relationship between the AD genetic measures and direct outcomes like cognitive function and the development of AD. We measure cognitive function using the L-W score and we examine whether an individual reports ever being diagnosed with a memory-related disease (MRD). Results are presented in Table 3.2. All specifications include the standard controls and the first 10 principal components of the genetic data. The results in column (1) imply that both the AD score and the two APOE indicators are predictive of worse cognitive functioning. A one standard deviation increase in the AD score is associated with a statistically significant 0.24 decrease in the L-W score, a 1.4 percent decline relative to the sample mean. Having at least one copy of the APOE allele is associated with a 0.40 decline in the L-W score, and having two copies decreases the L-W score by another 0.56 units. Thus, having two copies of APOE is associated with an almost 6 percent decline in the L-W score. In column (2), we include indicators for whether one has ever been diagnosed with MRD and whether one is eventually diagnosed with MRD later in life, hence this control is post-determined. Both indicators are, unsurprisingly, significantly negatively associated with the L-W score. Their inclusion barely changes the coefficient on the AD score while somewhat weakening the magnitude of the association between carrying the APOE allele and cognitive decline. The results from column (2) suggest that even after accounting for current and eventual diagnosis of MRD, the AD genetic measures are predictive of cognitive function.

Columns (3)–(5) present results from regressing whether one has ever been diagnosed with MRD on the genetic endowments for AD. The estimates in column (3) of Table 3.2 imply that the AD score and two APOE indicators are predictive of diagnosis. A one standard deviation increase in the AD score is associated with a statistically significant 0.3 percentage point increase in the probability of being diagnosed, a 14 percent increase relative to the sample mean. Having at least one copy of APOE significantly increases the likelihood of being diagnosed with MRD by 1 percentage point, and having two copies further increases that probability by 2 percentage points. Therefore, having two copies of the APOE allele increases the probability of diagnosis by almost 150 percent. In column (4), we add two indicators for whether one has ever registered an L-W score in the impaired or demented ranges and for whether one ever registered a score in the demented range. In column (5), we flexibly control for each value of the L-W score an individual most recently received. The addition of cognition controls attenuates the relationship between the AD genetic measures and MRD diagnosis. Having at least one copy of the APOE allele is correlated with a 0.6–0.8 percentage point increase in the probability of diagnosis and is the only AD genetic measure that maintains statistical significance ($p < 0.05$).

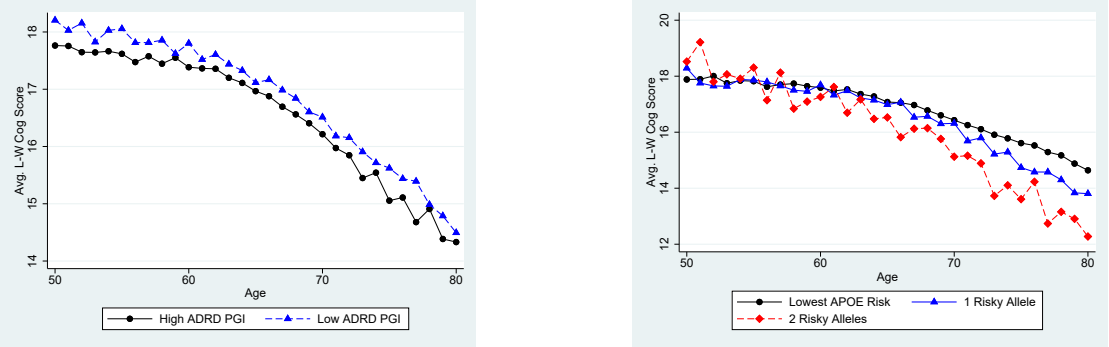
Figure 3.3 examines how the relationship between genetic risk and the L-W score evolves over the later life-cycle observed in the HRS. Panel A of Figure 3.3 plots the unconditional average L-W score by age separately for those with above and below median values of the AD score, respectively. Modest differences in the average L-W score are observed at every age across these groups, with little change in this gap over the life-cycle. This contrasts with the results in Panel B, which plots differences in these age profiles

Table 3.2: Relationship between Genetic Risk for AD and Cognition and Memory-Related Disease (MRD) Diagnosis

	Langa-Weir (LW) Score		Ever Diagnosed with Memory-Related Disease		
	(1)	(2)	(3)	(4)	(5)
AD Score	-0.236***	-0.214***	0.003**	0.002	0.002
	(0.035)	(0.034)	(0.001)	(0.001)	(0.001)
APOE (At least 1 copy)	-0.403***	-0.280***	0.010***	0.008***	0.006**
	(0.064)	(0.063)	(0.003)	(0.003)	(0.003)
APOE (2 copies)	-0.560**	-0.382*	0.020*	0.015	0.013
	(0.217)	(0.214)	(0.011)	(0.011)	(0.011)
Ever demented (L-W $<$ 7)				0.121***	
				(0.013)	
Ever impaired or demented (L-W $<$ 12)				0.030***	
				(0.003)	
Ever diagnosed with MRD		-2.354***			
		(0.200)			
Eventually diagnosed with MRD		-1.668***			
		(0.107)			
L-W Score Dummies	No	No	No	No	Yes
Standard Controls	Yes	Yes	Yes	Yes	Yes
Principal Components	Yes	Yes	Yes	Yes	Yes
Year	1998-2018	1998-2018	1998-2018	1998-2018	1998-2018
Ages	50-85	50-85	50-85	50-85	50-85
Sample	All	All	All	All	All
Mean	16.393	16.393	0.021	0.021	0.021
N w/ APOE (At least 1 copy)	22,943	22,943	22,943	22,943	22,943
N w/ APOE (2 copies)	1,717	1,717	1,717	1,717	1,717
N	88,191	88,191	88,191	88,191	88,191
R^2	0.133	0.163	0.018	0.049	0.065

Note: Each column presents results from a separate regression. In columns (1) and (2), the outcome is the Langa-Weir (L-W) cognition score, which can take on values from 0–27. In columns (3)–(5), the outcome is an indicator for whether the individual has ever been diagnosed with a memory-related disease (MRD). In all specifications, we control for the first 10 principal components of the genetic data as well as the standard controls for age, birth cohort, sex of respondent, and survey year as described in Section 3.4. In column (2), we add controls for whether the respondent has ever been diagnosed with MRD and whether they will eventually report diagnosis (in subsequent survey waves). In column (4), we add controls for whether the respondent ever registered an L-W score in the impaired or demented ranges (below 12) and in the demented range (below 7). In column (5), we instead add dummy variables for each value of the most current L-W score. Standard errors are clustered at the individual level. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Figure 3.3: Age-Cognition Profiles by Genetic Risk Groups



(a) Initiation Score

(b) CPD Score

Note: The figure plots average values of the Langa-Weir cognition score by age for individuals in our main genotyped sample. In Panel A, "high ADRD PGI" corresponds to above median values of the AD score, while "low ADRD PGI" corresponds to below median values. In Panel B, the lowest APOE risk group is those with zero copies of the APOE-$\varepsilon 4$ allele; the 1 risky allele group is those with one copy; and the 2 risky alleles group is those with two copies.

based on carrier status of the APOE-$\varepsilon 4$ allele. Here we see that each carrier group has a nearly identical age-cognition trajectory until the mid 60s, when individuals possessing copies of the APOE-$\varepsilon 4$ allele exhibit increasingly lower L-W score averages compared to the least risky APOE group. These differences raise the possibility that, while both the AD score and APOE represent genetic endowments linked to cognitive performance, they may operate through different mechanisms, or may interact differently with environmental and behavioral factors as individuals age.

We analyze these age patterns more rigorously by estimating a modified version of Equation 3.2 that includes interactions between the AD genetic measures and indicators for age in 5-year bins. In line with the clinical literature and the graphical patterns described above, carrying the APOE allele is associated with larger declines in cognition and increased probability of MRD diagnosis as individuals age (Appendix Table C1). The declines in the L-W score sharpen and grow in magnitude for those with one copy of APOE starting at age 70, while declines for those with two copies begin as early as age 55 and grow over time. For carriers of one or two copies of APOE, MRD diagnosis begins to significantly rise starting at age 70. Similar to patterns in Figure 3.3, we find little evidence of age heterogeneity with respect to the AD score. In particular, the negative robust association between the AD score and the L-W score appears relatively stable as individuals age.

Overall, the results in Table 3.2 imply that a higher genetic risk for AD is predictive of worse cognitive function and a higher probability of MRD diagnosis. AD-related genes are predictive of cognitive decline even after accounting for current and eventual MRD, and the relationship between carrying the APOE allele and MRD is robust to flexibly controlling for cognitive function. Taken together, the results

suggest genetic endowments for AD contain important information over and above standard measures of cognition and diagnosis of MRD.

3.5.2 Economic Outcomes

We now present and discuss the estimated relationship between the genetic risk for AD and several economic outcomes. Table 3.3 reports results where the outcome is an indicator for currently working for pay. The estimates imply that a one standard deviation increase in the AD score is associated with a statistically significant 1.7 percentage point decline in the probability of working for pay, a 3 percent decline relative to the sample mean. We do not find a significant association between carrying the APOE allele and working. When we add controls for cognitive function or when we condition the sample on those who never experience cognitive impairment or MRD diagnosis while we observe them in the HRS, the magnitude of the association between the AD score and working for pay attenuates modestly.

In Table 3.4, we consider retirement, defined as not currently working for pay and self-reporting oneself as completely retired. Consistent with our working for pay results, we find an increase in the AD score is associated with a statistically significant increase in the probability of being retired, and this relationship attenuates slightly with the addition of cognition controls. When we condition the sample on those never diagnosed with MRD and never cognitively impaired, the point estimate is similar to that of the full sample, but no longer statistically significant.[8] Overall, we find evidence that higher genetic risk for AD is associated with decreased labor market attachment.

We also consider log total individual annual income and find a one standard deviation increase in the AD score is associated with about a 4 percent decline in income (see Table 3.5). Adding controls for cognitive function or conditioning on the never diagnosed and never impaired sample somewhat mutes this relationship. The estimated coefficients on the APOE indicators never achieve statistical significance. In sum, AD-related genes, particularly the AD score, predict worse economic outcomes even after adjusting for cognitive function and MRD diagnosis.

3.5.3 Mortality and Attrition

We next shed light on the degree to which AD genetic risk predicts 1-year mortality and survey attrition. When we consider mortality, we exclude years before genotyping. For both outcomes, we include individuals aged 50–85. The coefficients in column (1) of Table 3.6 imply that a one standard deviation increase in the AD score significantly increases the probability of death in the next year by 0.1 percentage points. Having at least one copy of APOE increases mortality by 0.2 percentage points, but the coefficient is not statistically significant. Having two copies of APOE increases the mortality rate by another 0.6 percentage points ($p < 0.10$).[9] Column (2) suggests that a one standard deviation increase in the AD score decreases

[8] We considered alternative definitions of retirement based on whether one is partly or completely retired and whether one is currently working for pay. The coefficients are similar across these different definitions. Results are available by request.

[9] Our results are consistent with those of Linnér and Koellinger (2022) who find that out of 27 polygenic scores, the AD score had the second largest association with mortality.

Table 3.3: Relationship between Genetic Risk for AD and Currently Working for Pay

	Currently Working for Pay				
	(1)	(2)	(3)	(4)	(5)
AD Score	-0.017***	-0.013***	-0.016***	-0.013***	-0.011*
	(0.005)	(0.005)	(0.005)	(0.005)	(0.006)
APOE (At least 1 copy)	0.005	0.008	0.006	0.008	0.002
	(0.009)	(0.009)	(0.009)	(0.009)	(0.011)
APOE (2 copies)	-0.012	-0.005	-0.006	-0.001	0.020
	(0.026)	(0.026)	(0.025)	(0.025)	(0.032)
Ever diagnosed with MRD	No	No	Yes	Yes	No
L-W Score Dummies	No	Yes	No	Yes	Yes
Standard Controls	Yes	Yes	Yes	Yes	Yes
Principal Components	Yes	Yes	Yes	Yes	Yes
Years	1998-2018	1998-2018	1998-2018	1998-2018	1998-2018
Ages	50-70	50-70	50-70	50-70	50-70
Sample	All	All	All	All	Never diagnosed; never impaired
Mean	0.564	0.564	0.564	0.564	0.624
N w/ APOE (At least 1 copy)	14,203	14,203	14,203	14,203	9,101
N w/ APOE (2 copies)	1,180	1,180	1,180	1,180	646
N	53,743	53,743	53,743	53,743	36,741
R^2	0.151	0.169	0.161	0.176	0.162

Note: Each column presents results from a separate regression where the outcome is an indicator for whether the respondent currently works for pay. In all specifications, we control for the first 10 principal components of the genetic data as well as the standard controls for age, birth cohort, sex of respondent, and survey year as described in Section 3.4. In column (2), we add dummy variables for each value of the most current L-W score. In column (3), we control for whether the respondent has ever been diagnosed with memory-related disease (MRD). In column (4), we control for both the L-W dummies and MRD diagnosis. In column (5), we only include those who are never diagnosed with MRD and never cognitively impaired (as measured by the L-W score) while we observe them in the HRS, and we also control for the L-W dummies. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table 3.4: Relationship between Genetic Risk for AD and Retirement

	Whether Retired				
	(1)	(2)	(3)	(4)	(5)
AD Score	0.012***	0.009**	0.011**	0.009**	0.009
	(0.004)	(0.004)	(0.004)	(0.004)	(0.005)
APOE (At least 1 copy)	-0.007	-0.008	-0.008	-0.009	-0.001
	(0.008)	(0.008)	(0.008)	(0.008)	(0.009)
APOE (2 copies)	0.037	0.034	0.034	0.031	0.004
	(0.024)	(0.024)	(0.024)	(0.023)	(0.028)
Ever diagnosed with MRD	No	No	Yes	Yes	No
L-W Score Dummies	No	Yes	No	Yes	Yes
Standard Controls	Yes	Yes	Yes	Yes	Yes
Principal Components	Yes	Yes	Yes	Yes	Yes
Years	1998-2018	1998-2018	1998-2018	1998-2018	1998-2018
Ages	50-70	50-70	50-70	50-70	50-70
Sample	All	All	All	All	Never diagnosed; never impaired
Mean	0.320	0.320	0.320	0.320	0.274
N w/ APOE (At least 1 copy)	13,063	13,063	13,063	13,063	8,629
N w/ APOE (2 copies)	1,110	1,110	1,110	1,110	625
N	49,713	49,713	49,713	49,713	34,738
R^2	0.203	0.213	0.211	0.219	0.209

Note: Each column presents results from a separate regression where the outcome is an indicator for whether the respondent is retired, defined as currently not working for pay and self-reporting oneself as completely retired. In all specifications, we control for the first 10 principal components of the genetic data as well as the standard controls for age, birth cohort, sex of respondent, and survey year as described in Section 3.4. In column (2), we add dummy variables for each value of the most current L-W score. In column (3), we control for whether the respondent has ever been diagnosed with memory-related disease (MRD). In column (4), we control for both the L-W dummies and MRD diagnosis. In column (5), we only include those who are never diagnosed with MRD and never cognitively impaired (as measured by the L-W score) while we observe them in the HRS, and we also control for the L-W dummies. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table 3.5: Relationship between Genetic Risk for AD and Log Individual Total Income

	Log Total Individual Income				
	(1)	(2)	(3)	(4)	(5)
AD Score	-0.040***	-0.029***	-0.038***	-0.028***	-0.022*
	(0.008)	(0.008)	(0.008)	(0.008)	(0.011)
APOE (At least 1 copy)	-0.015	0.003	-0.012	0.005	0.026
	(0.016)	(0.015)	(0.016)	(0.015)	(0.021)
APOE (2 copies)	-0.011	0.015	-0.003	0.018	0.040
	(0.048)	(0.045)	(0.047)	(0.045)	(0.065)
Ever diagnosed with MRD	No	No	Yes	Yes	No
L-W Score Dummies	No	Yes	No	Yes	Yes
Standard Controls	Yes	Yes	Yes	Yes	Yes
Principal Components	Yes	Yes	Yes	Yes	Yes
Years	1998-2018	1998-2018	1998-2018	1998-2018	1998-2018
Ages	50-85	50-85	50-85	50-85	50-85
Sample	All	All	All	All	Never diagnosed; never impaired
Mean	10.007	10.007	10.007	10.007	10.176
N w/ APOE (At least 1 copy)	21,140	21,140	21,140	21,140	10,925
N w/ APOE (2 copies)	1,541	1,541	1,541	1,541	674
N	81,520	81,520	81,520	81,520	46,705
R^2	0.169	0.199	0.172	0.199	0.186

Note: Each column presents results from a separate regression where the outcome is logged total individual income. In all specifications, we control for the first 10 principal components of the genetic data as well as the standard controls for age, birth cohort, sex of respondent, and survey year as described in Section 3.4. In column (2), we add dummy variables for each value of the most current L-W score. In column (3), we control for whether the respondent has ever been diagnosed with memory-related disease (MRD). In column (4), we control for both the L-W dummies and MRD diagnosis. In column (5), we only include those who are never diagnosed with MRD and never cognitively impaired (as measured by the L-W score) while we observe them in the HRS, and we also control for the L-W dummies. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table 3.6: Relationship between Genetic Risk for AD and Mortality and Survey Attrition

	Observed Mortality	Appear in the Next Wave
	(1)	(2)
AD Score	0.001**	-0.004***
	(0.001)	(0.001)
APOE (At least 1 copy)	0.002	-0.005***
	(0.001)	(0.002)
APOE (2 copies)	0.006*	-0.005
	(0.004)	(0.007)
Standard Controls	Yes	Yes
Principal Components	Yes	Yes
Year	2006-2017	1998-2016
Age	50-85	50-85
Mean	0.023	0.941
N w/ APOE (At least 1 copy)	24,570	22,196
N w/ APOE (2 copies)	1,987	1,663
N	92,933	84,709
R^2	0.016	0.055

Note: Each column presents results from a separate regression. In column (1) the outcome is an indicator for whether the individual dies in the next year. In column (2), the outcome is an indicator for whether the individual appears in the next survey wave. In all specifications, we control for the first 10 principal components of the genetic data as well as the standard controls for age, birth cohort, sex of respondent, and survey year as described in Section 3.4. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

the probability an individual responds to the survey in the following wave by 0.4 percentage points, and having at least one copy of the APOE allele decreases that probability by 0.5 percentage points. While the point estimates for having two copies of the APOE allele imply further decreases in the probability of appearing in the next survey wave, the standard errors are large. Overall, the mortality and attrition results imply that those with higher genetic risk for AD are less likely to appear in subsequent survey waves. This is important to keep in mind as we proceed, as our results likely reflect those of a selected sample of relatively healthier individuals, leading our estimates to be conservative.

3.5.4 Expectations

Individuals at higher genetic risk for AD face the prospects of diminished cognition, more challenging economic circumstances, and a greater risk of mortality. If these individuals understood their elevated risk status, we would expect this to be reflected in their own expectations about mortality or future care needs, and in their propensity to make preparations to insulate themselves and their families from related medical

or financial burdens. In Tables 3.7 and 3.8, we display estimates that reflect the relationship between AD genetic risk and expectations about mortality and future nursing home utilization, respectively. The point estimates show that among individuals aged 65 and younger, those with a higher genetic propensity for AD report a lower probability of living to age 75, but none of the estimates reach statistical significance at conventional levels.

Among those aged 65–85, we do not find a significant relationship between the AD score and one's self-reported probability of using a nursing home. Those with at least one copy of the APOE allele report about an 0.8 percentage point increase in the probability of future nursing home use, about a 6 percent increase relative to the sample mean. The coefficient on the indicator for having exactly two copies of APOE is negative and very imprecisely estimated. Interestingly, the relationship between having at least one copy of APOE and expected nursing home use is very robust to the inclusion of controls for cognitive function and MRD diagnosis, and almost doubles in magnitude among those who are never cognitively impaired or diagnosed with MRD while observed in the HRS. One possible interpretation is that some individuals are aware of their genetic risk and incorporate that knowledge into their assessment of future long-term care needs.

The question about future nursing home use is only asked to those not currently in a nursing home. Thus, the sample may be positively selected on those who do not need institutional care, potentially leading these estimates to be conservative. Nevertheless, the results suggest that some APOE carriers who currently live in the community anticipate using a nursing home in the near future. It is possible these expectations affect individuals' economic and planning behavior. We examine these outcomes next.

3.5.5 Planning Outcomes

We next examine how genetic risk for AD associates with a variety of later-life planning outcomes, including having: LTCI, life insurance, a witnessed will, a living will (i.e., an advance care directive), a durable power of attorney for health care, and ever discussed future medical care with anyone. Results are presented in Tables 3.9–3.14. We find an increase in the AD score is associated with statistically significant declines in most of these outcomes. A one standard deviation increase in the AD score is associated with a 1.4 percentage point decline in the probability of having LTCI (a 9.5 percent decrease), a 0.9 percentage point decline in holding life insurance (a 1.3 percent decrease), a 2.2 percentage point decline in having a witnessed will (a 3.4 percent decrease), a 1.8 percentage point decline in having a durable power of attorney (a 3.0 percent decrease), and a 1.7 percentage point decline in the probability of discussing future medical care with anyone (a 3.1 percent decrease). These associations are generally robust to the inclusion of controls for cognitive function and to limiting the sample to those never diagnosed with MRD and never cognitively impaired, with the exception of holding life insurance. We generally find no significant association between being an APOE carrier and the planning outcomes, though in some specifications we find a marginally significant increase in the probability of holding LTCI among APOE carriers. The planning results are striking in that those who potentially have the most to gain by planning, namely those with increased risk of experiencing cognitive decline and developing AD, are no more likely to engage in these planning activities, and if anything, are less likely to do so. Furthermore, earlier, we found APOE

Table 3.7: Relationship between Genetic Risk for AD and Expected Mortality

	Probability of Living to Age 75				
	(1)	(2)	(3)	(4)	(5)
AD Score	-0.326	-0.112	-0.282	-0.082	0.127
	(0.316)	(0.310)	(0.314)	(0.309)	(0.357)
APOE (At least 1 copy)	-0.786	-0.694	-0.796	-0.703	-0.871
	(0.585)	(0.573)	(0.580)	(0.569)	(0.653)
APOE (2 copies)	-1.108	-0.885	-0.925	-0.742	-2.306
	(1.910)	(1.889)	(1.907)	(1.887)	(2.508)
Ever diagnosed with MRD	No	No	Yes	Yes	No
L-W Score Dummies	No	Yes	No	Yes	Yes
Standard Controls	Yes	Yes	Yes	Yes	Yes
Principal Components	Yes	Yes	Yes	Yes	Yes
Years	1998-2018	1998-2018	1998-2018	1998-2018	1998-2018
Ages	50-65	50-65	50-65	50-65	50-65
Sample	All	All	All	All	Never diagnosed; never impaired
Mean	66.294	66.294	66.294	66.294	68.272
N w/ APOE (At least 1 copy)	9,622	9,622	9,622	9,622	6,686
N w/ APOE (2 copies)	844	844	844	844	513
N	36,415	36,415	36,415	36,415	26,517
R^2	0.027	0.050	0.033	0.054	0.041

Note: Each column presents results from a separate regression where the outcome is the self-reported probability of living to age 75 (on a 0–100 scale). The question is only asked to those aged 65 and younger. In all specifications, we control for the first 10 principal components of the genetic data as well as the standard controls for age, birth cohort, sex of respondent, and survey year as described in Section 3.4. In column (2), we add dummy variables for each value of the most current L-W score. In column (3), we control for whether the respondent has ever been diagnosed with memory-related disease (MRD). In column (4), we control for both the L-W dummies and MRD diagnosis. In column (5), we only include those who are never diagnosed with MRD and never cognitively impaired (as measured by the L-W score) while we observe them in the HRS, and we also control for the L-W dummies. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

carriers reported a higher probability of using a nursing home in the future, suggesting they may be aware of their genetic risk; yet the estimates suggest they do not adjust their financial and non-financial later-life plans. We acknowledge, however, that in many cases the standard errors on the APOE carrier estimates, especially for having two copies of the allele, are large and we cannot rule out non-trivial associations.

Table 3.8: Relationship between Genetic Risk for AD and Expected Nursing Home Use

	Probability of Moving to Nursing Home in Next 5 Years				
	(1)	(2)	(3)	(4)	(5)
AD Score	-0.142	-0.166	-0.157	-0.173	-0.187
	(0.190)	(0.189)	(0.190)	(0.189)	(0.261)
APOE (At least 1 copy)	0.851**	0.818**	0.772**	0.766**	1.401***
	(0.357)	(0.356)	(0.357)	(0.357)	(0.512)
APOE (2 copies)	-0.505	-0.555	-0.639	-0.629	-0.069
	(1.137)	(1.133)	(1.134)	(1.131)	(2.023)
Ever diagnosed with MRD	No	No	Yes	Yes	No
L-W Score Dummies	No	Yes	No	Yes	Yes
Standard Controls	Yes	Yes	Yes	Yes	Yes
Principal Components	Yes	Yes	Yes	Yes	Yes
Years	1998-2018	1998-2018	1998-2018	1998-2018	1998-2018
Ages	65-85	65-85	65-85	65-85	65-85
Sample	All	All	All	All	Never diagnosed; never impaired
Mean	14.449	14.449	14.449	14.449	13.926
N w/ APOE (At least 1 copy)	12,616	12,616	12,616	12,616	5,261
N w/ APOE (2 copies)	823	823	823	823	247
N	49,350	49,350	49,350	49,350	24,290
R^2	0.041	0.042	0.042	0.043	0.059

Note: Each column presents results from a separate regression where the outcome is the self-reported probability of moving to a nursing home in the next 5 years (on a 0–100 scale). The question is only asked to those aged 65 and older who do not currently reside in a nursing home. In all specifications, we control for the first 10 principal components of the genetic data as well as the standard controls for age, birth cohort, sex of respondent, and survey year as described in Section 3.4. In column (2), we add dummy variables for each value of the most current L-W score. In column (3), we control for whether the respondent has ever been diagnosed with memory-related disease (MRD). In column (4), we control for both the L-W dummies and MRD diagnosis. In column (5), we only include those who are never diagnosed with MRD and never cognitively impaired (as measured by the L-W score) while we observe them in the HRS, and we also control for the L-W dummies. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table 3.9: Relationship between Genetic Risk for AD and Holding Long-Term Care Insurance

	Holds Long-Term Care Insurance				
	(1)	(2)	(3)	(4)	(5)
AD Score	-0.014***	-0.012***	-0.014***	-0.012***	-0.011**
	(0.004)	(0.004)	(0.004)	(0.004)	(0.005)
APOE (At least 1 copy)	0.009	0.012*	0.009	0.012*	0.002
	(0.007)	(0.007)	(0.007)	(0.007)	(0.010)
APOE (2 copies)	-0.005	-0.001	-0.005	-0.001	0.014
	(0.021)	(0.021)	(0.021)	(0.021)	(0.031)
Ever diagnosed with MRD	No	No	Yes	Yes	No
L-W Score Dummies	No	Yes	No	Yes	Yes
Standard Controls	Yes	Yes	Yes	Yes	Yes
Principal Components	Yes	Yes	Yes	Yes	Yes
Years	1998-2018	1998-2018	1998-2018	1998-2018	1998-2018
Ages	50-85	50-85	50-85	50-85	50-85
Sample	All	All	All	All	Never diagnosed; never impaired
Mean	0.148	0.148	0.148	0.148	0.157
N w/ APOE (At least 1 copy)	22,532	22,532	22,532	22,532	11,947
N w/ APOE (2 copies)	1,685	1,685	1,685	1,685	763
N	86,864	86,864	86,864	86,864	50,764
R^2	0.023	0.029	0.023	0.029	0.037

Note: Each column presents results from a separate regression where the outcome is an indicator for whether the respondent holds long-term care insurance. In all specifications, we control for the first 10 principal components of the genetic data as well as the standard controls for age, birth cohort, sex of respondent, and survey year as described in Section 3.4. In column (2), we add dummy variables for each value of the most current L-W score. In column (3), we control for whether the respondent has ever been diagnosed with memory-related disease (MRD). In column (4), we control for both the L-W dummies and MRD diagnosis. In column (5), we only include those who are never diagnosed with MRD and never cognitively impaired (as measured by the L-W score) while we observe them in the HRS, and we also control for the L-W dummies. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table 3.10: Relationship between Genetic Risk for AD and Holding Life Insurance

	Holds Life Insurance				
	(1)	(2)	(3)	(4)	(5)
AD Score	-0.009*	-0.007	-0.008*	-0.007	-0.003
	(0.004)	(0.004)	(0.004)	(0.004)	(0.006)
APOE (At least 1 copy)	-0.005	-0.001	-0.004	-0.001	0.003
	(0.008)	(0.008)	(0.008)	(0.008)	(0.011)
APOE (2 copies)	-0.010	-0.005	-0.009	-0.005	-0.049
	(0.025)	(0.025)	(0.025)	(0.025)	(0.035)
Ever diagnosed with MRD	No	No	Yes	Yes	No
L-W Score Dummies	No	Yes	No	Yes	Yes
Standard Controls	Yes	Yes	Yes	Yes	Yes
Principal Components	Yes	Yes	Yes	Yes	Yes
Years	1998-2018	1998-2018	1998-2018	1998-2018	1998-2018
Ages	50-85	50-85	50-85	50-85	50-85
Sample	All	All	All	All	Never diagnosed; never impaired
Mean	0.677	0.677	0.677	0.677	0.703
N w/ APOE (At least 1 copy)	22,741	22,741	22,741	22,741	12,058
N w/ APOE (2 copies)	1,700	1,700	1,700	1,700	772
N	87,576	87,576	87,576	87,576	51,157
R^2	0.048	0.053	0.048	0.053	0.061

Note: Each column presents results from a separate regression where the outcome is an indicator for whether the respondent holds life insurance. In all specifications, we control for the first 10 principal components of the genetic data as well as the standard controls for age, birth cohort, sex of respondent, and survey year as described in Section 3.4. In column (2), we add dummy variables for each value of the most current L-W score. In column (3), we control for whether the respondent has ever been diagnosed with memory-related disease (MRD). In column (4), we control for both the L-W dummies and MRD diagnosis. In column (5), we only include those who are never diagnosed with MRD and never cognitively impaired (as measured by the L-W score) while we observe them in the HRS, and we also control for the L-W dummies. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table 3.11: Relationship between Genetic Risk for AD and Having a Witnessed Will

	Has a Witnessed Will				
	(1)	(2)	(3)	(4)	(5)
AD Score	-0.022***	-0.017***	-0.021***	-0.017***	-0.014**
	(0.005)	(0.005)	(0.005)	(0.005)	(0.006)
APOE (At least 1 copy)	0.005	0.011	0.005	0.011	0.007
	(0.009)	(0.009)	(0.009)	(0.009)	(0.012)
APOE (2 copies)	0.006	0.016	0.007	0.016	0.011
	(0.028)	(0.027)	(0.028)	(0.027)	(0.042)
Ever diagnosed with MRD	No	No	Yes	Yes	No
L-W Score Dummies	No	Yes	No	Yes	Yes
Standard Controls	Yes	Yes	Yes	Yes	Yes
Principal Components	Yes	Yes	Yes	Yes	Yes
Years	1998-2018	1998-2018	1998-2018	1998-2018	1998-2018
Ages	50-85	50-85	50-85	50-85	50-85
Sample	All	All	All	All	Never diagnosed; never impaired
Mean	0.640	0.640	0.640	0.640	0.642
N w/ APOE (At least 1 copy)	22,842	22,842	22,842	22,842	12,104
N w/ APOE (2 copies)	1,708	1,708	1,708	1,708	772
N	87,893	87,893	87,893	87,893	51,310
R^2	0.094	0.112	0.095	0.112	0.113

Note: Each column presents results from a separate regression where the outcome is an indicator for whether the respondent has a witnessed will. In all specifications, we control for the first 10 principal components of the genetic data as well as the standard controls for age, birth cohort, sex of respondent, and survey year as described in Section 3.4. In column (2), we add dummy variables for each value of the most current L-W score. In column (3), we control for whether the respondent has ever been diagnosed with memory-related disease (MRD). In column (4), we control for both the L-W dummies and MRD diagnosis. In column (5), we only include those who are never diagnosed with MRD and never cognitively impaired (as measured by the L-W score) while we observe them in the HRS, and we also control for the L-W dummies. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table 3.12: Relationship between Genetic Risk for AD and Having a Living Will

	Has a Living Will (Advance Healthcare Directive)				
	(1)	(2)	(3)	(4)	(5)
AD Score	-0.006	-0.004	-0.007	-0.004	-0.003
	(0.007)	(0.007)	(0.007)	(0.007)	(0.009)
APOE (At least 1 copy)	-0.007	0.001	-0.007	0.000	-0.004
	(0.013)	(0.013)	(0.013)	(0.013)	(0.018)
APOE (2 copies)	0.025	0.035	0.025	0.033	0.037
	(0.043)	(0.042)	(0.043)	(0.042)	(0.066)
Ever diagnosed with MRD	No	No	Yes	Yes	No
L-W Score Dummies	No	Yes	No	Yes	Yes
Standard Controls	Yes	Yes	Yes	Yes	Yes
Principal Components	Yes	Yes	Yes	Yes	Yes
Years	2012-2018	2012-2018	2012-2018	2012-2018	2012-2018
Ages	65-85	65-85	65-85	65-85	65-85
Sample	All	All	All	All	Never diagnosed; never impaired
Mean	0.598	0.598	0.598	0.598	0.615
N w/ APOE (At least 1 copy)	4,844	4,844	4,844	4,844	2,647
N w/ APOE (2 copies)	339	339	339	339	151
N	18,902	18,902	18,902	18,902	11,413
R^2	0.057	0.066	0.057	0.066	0.075

Note: Each column presents results from a separate regression where the outcome is an indicator for whether the respondent has a living will. In all specifications, we control for the first 10 principal components of the genetic data as well as the standard controls for age, birth cohort, sex of respondent, and survey year as described in Section 3.4. In column (2), we add dummy variables for each value of the most current L-W score. In column (3), we control for whether the respondent has ever been diagnosed with memory-related disease (MRD). In column (4), we control for both the L-W dummies and MRD diagnosis. In column (5), we only include those who are never diagnosed with MRD and never cognitively impaired (as measured by the L-W score) while we observe them in the HRS, and we also control for the L-W dummies. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table 3.13: Relationship between Genetic Risk for AD and Having Assigned Someone Durable Power of Attorney

	Has Assigned Someone Durable Power of Attorney for Healthcare				
	(1)	(2)	(3)	(4)	(5)
AD Score	-0.018***	-0.016**	-0.018***	-0.016**	-0.017*
	(0.007)	(0.007)	(0.007)	(0.007)	(0.009)
APOE (At least 1 copy)	0.003	0.009	0.003	0.008	0.006
	(0.013)	(0.013)	(0.013)	(0.013)	(0.017)
APOE (2 copies)	0.000	0.006	-0.000	0.005	0.045
	(0.044)	(0.043)	(0.044)	(0.043)	(0.065)
Ever diagnosed with MRD	No	No	Yes	Yes	No
L-W Score Dummies	No	Yes	No	Yes	Yes
Standard Controls	Yes	Yes	Yes	Yes	Yes
Principal Components	Yes	Yes	Yes	Yes	Yes
Years	2012-2018	2012-2018	2012-2018	2012-2018	2012-2018
Ages	65-85	65-85	65-85	65-85	65-85
Sample	All	All	All	All	Never diagnosed; never impaired
Mean	0.596	0.596	0.596	0.596	0.610
N w/ APOE (At least 1 copy)	4,865	4,865	4,865	4,865	2,653
N w/ APOE (2 copies)	342	342	342	342	151
N	18,942	18,942	18,942	18,942	11,416
R^2	0.066	0.072	0.066	0.073	0.087

Note: Each column presents results from a separate regression where the outcome is an indicator for whether the respondent has assigned someone durable power of attorney. In all specifications, we control for the first 10 principal components of the genetic data as well as the standard controls for age, birth cohort, sex of respondent, and survey year as described in Section 3.4. In column (2), we add dummy variables for each value of the most current L-W score. In column (3), we control for whether the respondent has ever been diagnosed with memory-related disease (MRD). In column (4), we control for both the L-W dummies and MRD diagnosis. In column (5), we only include those who are never diagnosed with MRD and never cognitively impaired (as measured by the L-W score) while we observe them in the HRS, and we also control for the L-W dummies. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table 3.14: Relationship between Genetic Risk for AD and Having Discussed Future Medical Care with Someone

	Discussed Future Medical Care with Anyone				
	(1)	(2)	(3)	(4)	(5)
AD Score	-0.017***	-0.013**	-0.017***	-0.013**	-0.019**
	(0.006)	(0.006)	(0.006)	(0.006)	(0.008)
APOE (At least 1 copy)	-0.019	-0.011	-0.020*	-0.013	-0.013
	(0.012)	(0.012)	(0.012)	(0.012)	(0.016)
APOE (2 copies)	-0.028	-0.014	-0.029	-0.015	-0.081
	(0.039)	(0.039)	(0.039)	(0.039)	(0.056)
Ever diagnosed with MRD	No	No	Yes	Yes	No
L-W Score Dummies	No	Yes	No	Yes	Yes
Standard Controls	Yes	Yes	Yes	Yes	Yes
Principal Components	Yes	Yes	Yes	Yes	Yes
Years	2012-2018	2012-2018	2012-2018	2012-2018	2012-2018
Ages	65-85	65-85	65-85	65-85	65-85
Sample	All	All	All	All	Never diagnosed; never impaired
Mean	0.553	0.553	0.553	0.553	0.588
N w/ APOE (At least 1 copy)	2,603	2,603	2,603	2,603	1,355
N w/ APOE (2 copies)	179	179	179	179	81
N	10,059	10,059	10,059	10,059	5,734
R^2	0.102	0.117	0.102	0.118	0.139

Note: Each column presents results from a separate regression where the outcome is an indicator for whether the respondent has discussed future medical care with someone. In all specifications, we control for the first 10 principal components of the genetic data as well as the standard controls for age, birth cohort, sex of respondent, and survey year as described in Section 3.4. In column (2), we add dummy variables for each value of the most current L-W score. In column (3), we control for whether the respondent has ever been diagnosed with memory-related disease (MRD). In column (4), we control for both the L-W dummies and MRD diagnosis. In column (5), we only include those who are never diagnosed with MRD and never cognitively impaired (as measured by the L-W score) while we observe them in the HRS, and we also control for the L-W dummies. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

3.5.6 Heterogeneity Analysis

We explored whether the above results differed for men and women. In general, we found no significant heterogeneity by gender, with one exception which we display in Table 3.15. Men who carry at least one copy of the APOE allele are about 2.5 percentage points more likely to have LTCI, whereas there is no statistically significant relationship between carrying the APOE allele and LTCI holding for women. As women typically outlive men and wives are usually younger than their husbands, the increase in LTCI holding among male APOE carriers may reflect an effort to protect family assets for the wife. While we do not know whether individuals with higher genetic risk for AD are aware of their increased risk, the LTCI results for men are consistent with findings in Zick et al. (2005) and Taylor et al. (2010). In those studies, individuals who learn they carry the APOE-$\varepsilon 4$ allele were significantly more likely to purchase LTCI or to report that they planned to purchase LTCI. The full set of results from the gender heterogeneity analysis are available by request.

3.5.7 Gene-Environment Interactions

An advantage of the genetic data is we can examine whether there are protective factors that characterize individuals who do not exhibit cognitive decline and/or who are not diagnosed with MRD, yet they have a genetic propensity for AD. Identifying protective factors could inform interventions that slow cognitive decline or reduce the risk of AD. More generally, analyzing gene-environment interactions provides important information about who is likely to be most impacted by changes in behavior or the environment. We explore a variety of environmental factors below.

One leading candidate is educational attainment, which is strongly associated with a lower risk of developing AD, a phenomena often referred to as the "cognitive reserve" hypothesis.[10] We examine the extent to which years of education associates with cognitive decline and MRD diagnosis and whether there are significant gene-education interactions. The results are presented in Appendix Table C2. We first present our baseline estimates that only include the AD genetic variables, standard controls, and genetic principal components. We then control for years of education and interact it with the AD genetic measures. We find more education is associated with a higher L-W score and weak evidence that education is protective against cognitive decline for those with more genetic risk for AD—only the coefficient on the interaction with the AD score is statistically significant, but marginally so and only when we do not control for current or eventual MRD diagnosis. Turning to MRD, more education is associated with a statistically significant lower probability of diagnosis. However, once we flexibly control for cognitive functioning, more education is associated with a marginally significant increase in diagnosis. Thus, on net, education

[10]The AD literature often distinguishes between cognitive reserve and brain reserve. Cognitive reserve refers to the ability to maintain cognitive function even in the presence of the neuropathologic changes associated with AD via recruitment of alternative neural networks or more efficient utilization of existing networks, sometimes referred to as cognitive flexibility. Educational attainment is the most commonly used proxy for cognitive reserve. Brain reserve refers to quantifiable brain resources (e.g., intracranial volume, number of neurons) that enhance or maintain cognitive function as pathological changes emerge. The line between the two has become more blurry as recent evidence shows that stimulating environments can boost brain reserve by fostering the growth of new neurons and neural plasticity (Stern, 2012; Stern et al., 2020).

Table 3.15: Relationship between Genetic Risk for AD and Holding Long-Term Care Insurance by Gender

	Holds Long-Term Care Insurance (LTCI)			
	(1)	(2)	(3)	(4)
AD Score	-0.015***	-0.014***	-0.011**	-0.010*
	(0.005)	(0.005)	(0.006)	(0.006)
APOE (At least 1 copy)	-0.002	0.002	0.024**	0.026**
	(0.009)	(0.009)	(0.011)	(0.011)
APOE (2 copies)	0.005	0.011	-0.020	-0.017
	(0.029)	(0.029)	(0.030)	(0.030)
Ever diagnosed with MRD	No	Yes	No	Yes
L-W Score Dummies	No	Yes	No	Yes
Standard Controls	Yes	Yes	Yes	Yes
Principal Components	Yes	Yes	Yes	Yes
Years	1998-2018	1998-2018	1998-2018	1998-2018
Ages	50-85	50-85	50-85	50-85
Sample	Females	Females	Males	Males
Mean	0.150	0.150	0.146	0.146
N w/ APOE (At least 1 copy)	13,228	13,228	9,304	9,304
N w/ APOE (2 copies)	956	956	729	729
N	50,417	50,417	36,447	36,447
R^2	0.024	0.032	0.022	0.027

Note: Each column presents results from a separate regression where the outcome is an indicator for whether the respondent holds long-term care insurance. Results for women are in columns (1) and (2) and for men in columns (3) and (4). In all specifications, we control for the first 10 principal components of the genetic data as well as the standard controls for age, birth cohort, sex of respondent, and survey year as described in Section 3.4. In columns (2) and (4), we add dummy variables for each value of the most current Langa-Weir (L-W) score and whether the respondent has ever been diagnosed with memory-related disease (MRD). * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

appears to reduce diagnosis by improving cognitive functioning, but conditional on cognitive function, it hastens diagnosis. There is no evidence of significant interactions between educational attainment and the AD genetic measures when we consider MRD diagnosis.

We consider a host of other potential environmental influences, including one's own early childhood environment. The literature has found more advantaged childhood socioeconomic status (SES) is associated with higher levels of cognitive functioning later in life, which may be due in part to increased cognitive reserve. However, the literature is mixed on whether childhood SES slows or hastens the speed of cognitive decline (see for example Lyu and Burr, 2016; Aartsen et al., 2019; Tsang et al., 2022). We create a high childhood SES indicator for whether the respondent describes their family's financial situation growing up (from birth to age 16) as "very well off" or "average" (versus "poor"). In Appendix Table

C3, we indeed find that high childhood SES is associated with better cognitive performance, and there is some evidence that high childhood SES protects against cognitive decline for those with higher AD scores and lowers the probability of MRD diagnosis among those who carry the APOE allele. In fact, for those with exactly one copy of APOE, more advantaged childhood SES can almost completely offset the increased probability of MRD diagnosis associated with carrying this allele. We also examine the extent to which the AD genetic measures interact with whether the respondent's mother completed at least 12 years of education, which serves as a proxy for a potentially more advantageous childhood environment and parental transfer of resources (Barth et al., 2020). We find no significant interactions between maternal education and genetic propensity for AD (Appendix Table C4).

We also examine whether an individual has children that live nearby (i.e., within 10 miles). Children who live close may notice even mild symptoms of cognitive impairment and perhaps encourage their parent to seek out a diagnosis. The results are shown in Appendix Table C5 and we only include individuals who have living children. None of the interactions between the AD genetic measures and having children nearby achieve statistical significance.

We next explore whether the relationships between AD genetic risk and impairment and diagnosis vary with whether the respondent's parents have ever been diagnosed with MRD. Individuals with a parental history of MRD may update their expectations about their own genetic risk for AD and adjust their behavior, perhaps engaging in activities to maintain cognitive function, and/or seek out a diagnosis. Starting in 1998, respondents were asked whether their mother or father was ever told by a doctor that they have MRD. Starting in 2010, respondents were asked whether their parents were ever told by a doctor that they have Alzheimer's disease or dementia. These questions are only asked if the respondent's mother or father is still alive; thus, our sample size shrinks dramatically, limiting statistical power. We find no statistically significant interactions between the genetic measures and a parent ever being diagnosed with MRD when we consider the L-W score as an outcome (Appendix Table C6). However, individuals who carry two copies of APOE and have a parental history of MRD are 6.5 percentage points less likely to be diagnosed themselves with MRD than those who carry two copies but have no parental history of MRD.

3.6 Genetic Risk for AD, Educational Attainment, and the EA Score

The results in the prior section suggest that educational attainment protects against cognitive decline, but we found weak evidence that it differentially protects those with more genetic risk for AD. We further characterize the role of education by first examining the relationship between educational attainment and genetic risk for AD and then exploring the robustness of our results to flexibly controlling for educational attainment as well as the polygenic score for educational attainment.

We analyze whether genetic endowments for AD correlate with educational attainment by estimating equation 3.2 with years of education (Table C18) and an indicator for completing at least some college (Table C19) as the outcomes. In column (1), we find a statistically significant negative association between the AD score and education—a one standard deviation increase in the AD score correlates with 0.16 fewer

years of education and a 1.9 percentage point decrease in the probability of attending at least some college. Carrying the APOE allele is not significantly associated with education. We then add the EA polygenic score from the GWAS of Lee et al. (2018) in column (2). Not surprisingly, the EA score is very predictive of education. Somewhat surprisingly, despite the addition of the EA score, which should capture the genetic endowments for education, we still find a precisely estimated negative point estimate on the AD score.

There are a few potential explanations for the above finding. The first we believe is extremely unlikely—that low cognitive functioning and manifestations of AD begin prior to completing one's education. We have found no research to support this notion. While studies have found cognitive and pathological changes associated with AD may begin 15–20 years before diagnosis, the literature has not established a precise age when symptoms begin. A recent study using an older AD polygenic score based on the GWAS of Lambert et al. (2013) found that those with higher AD scores began to exhibit modestly worse performance on three cognitive tests (pairs matching, symbol digit substitution, and numeric memory) by early middle age (i.e., age 47) (Zimmerman et al., 2022). When they exclude the APOE region from their AD score, the divergence in cognitive performance begins slightly later. Given the subtlety in differences in cognitive performance at midlife in Zimmerman et al. (2022), we find it very unlikely that genetic risk for AD manifests in differences in educational attainment. Another explanation is that some of the biological factors that protect against AD also promote educational attainment and there is measurement error in the EA score. That is, if some genetic markers predict both educational attainment and AD but are not fully captured in the EA score, the AD score may explain some variation in education. We explore this idea by replacing the EA score based on Lee et al. (2018) with an older one from the GWAS of Okbay et al. (2016), which had a smaller discovery sample (N = 1.1 mil versus N = 293,723), in column (3). Comparing columns (2) and (3), the coefficient on the AD score attenuates when we use the more recent EA score that captures more genetic variation in education. Thus, measurement error in the EA score likely explains some of the AD score-education relationship. Another explanation, especially in light of our findings that AD genetic risk is associated with worse economic and planning outcomes combined with the fact that genes are inherited from one's parents, is that the AD score captures indirect factors (e.g., childhood socioeconomic status) related to education. In column (4), we add controls for childhood SES and dummies for the respondent's mother's years of completed education. The inclusion of these controls nearly halves the coefficient on the AD score relative to that in column (2), and in the case of completing at least some college, the coefficient is no longer statistically significant. Thus, the AD score-education relationship is explained in part by aspects of early life environment.

Given the observed correlation between the AD score and educational attainment, even conditioning on the EA score, we reestimate our expectation, economic outcome, and planning outcome specifications that control for cognitive function and MRD diagnosis and add flexible controls for education (i.e., a complete set of dummy variables for each year of schooling and dummies for the highest-completed degree as well as interactions with the male dummy). In addition, we sometimes control for the EA polygenic score. The results are presented in Appendix Tables C7–C17. The addition of education controls does not change our main conclusions regarding expectations. In particular, we still find a robust position

Table 3.16: Relationship between Genetic Risk for AD and Years of Education

	Years of Education			
	(1)	(2)	(3)	(4)
AD Score	-0.139***	-0.109***	-0.126***	-0.062**
	(0.029)	(0.028)	(0.028)	(0.027)
APOE (At least 1 copy)	-0.001	0.002	-0.012	0.020
	(0.054)	(0.052)	(0.053)	(0.049)
APOE (2 copies)	-0.037	-0.019	-0.036	-0.132
	(0.165)	(0.159)	(0.158)	(0.144)
EA Score		0.661***		0.531***
		(0.023)		(0.022)
EA Score (Old)			0.575***	
			(0.023)	
Childhood SES	No	No	No	Yes
Mother's Years of Education	No	No	No	Yes
Standard Controls	Yes	Yes	Yes	Yes
Principal Components	Yes	Yes	Yes	Yes
Years	1994-2018	1994-2018	1994-2018	1994-2018
Ages	50-85	50-85	50-85	50-85
Sample	All	All	All	All
Mean	13.388	13.388	13.388	13.388
N w/ APOE (At least 1 copy)	2,835	2,835	2,835	2,835
N w/ APOE (2 copies)	221	221	221	221
N	10,985	10,985	10,985	10,985
R^2	0.090	0.155	0.139	0.259

Note: Each column presents results from a separate regression where the outcome is completed years of education. In all specifications, we control for the first 10 principal components of the genetic data, the standard controls for age, birth cohort, sex of respondent, and survey year as described in Section 3.4, and Langa-Weir score dummies and MRD diagnosis. In column (2), we add the EA score from Lee et al. (2018). In column (3), we instead add the EA score from Okbay et al. (2016). In column (4), we control for the EA score from Lee et al. (2018) as well dummies for childhood SES and completed years of maternal education. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

association between having at least one copy of the APOE allele and the probability of future nursing home use. In general, controlling for educational attainment slightly dampens the association between the AD score and economic and planning outcomes, but in most cases the point estimate remains significant at the 10 percent level or better. We also find some evidence that a higher EA score is associated with more planning activities, such as having a witnessed will, having a living will, and assigning durable power of attorney, even after flexibly controlling for educational attainment. These results are consistent with the evidence in Barth et al. (2020) that those with higher EA scores have a facility with complex decision-making and probabilistic thinking.

Table 3.17: Relationship between Genetic Risk for AD and At Least Some College Education

	At Least Some College			
	(1)	(2)	(3)	(4)
AD Score	-0.019***	-0.014***	-0.017***	-0.008
	(0.006)	(0.005)	(0.006)	(0.005)
APOE (At least 1 copy)	-0.000	0.000	-0.002	0.003
	(0.010)	(0.010)	(0.010)	(0.010)
APOE (2 copies)	-0.003	0.000	-0.002	-0.020
	(0.033)	(0.032)	(0.032)	(0.030)
EA Score		0.107***		0.087***
		(0.004)		(0.004)
EA Score (Old)			0.090***	
			(0.004)	
Childhood SES	No	No	No	Yes
Mother's Years of Education	No	No	No	Yes
Standard Controls	Yes	Yes	Yes	Yes
Principal Components	Yes	Yes	Yes	Yes
Years	1994-2018	1994-2018	1994-2018	1994-2018
Ages	50-85	50-85	50-85	50-85
Sample	All	All	All	All
Mean	0.334	0.334	0.334	0.334
N w/ APOE (At least 1 copy)	2,835	2,835	2,835	2,835
N w/ APOE (2 copies)	221	221	221	221
N	10,985	10,985	10,985	10,985
R^2	0.072	0.118	0.104	0.189

Note: Each column presents results from a separate regression where the outcome is a dummy variable for whether the respondent has at least some college education. In all specifications, we control for the first 10 principal components of the genetic data, the standard controls for age, birth cohort, sex of respondent, and survey year as described in Section 3.4, and Langa-Weir score dummies and MRD diagnosis. In column (2), we add the EA score from Lee et al. (2018). In column (3), we instead add the EA score from Okbay et al. (2016). In column (4), we control for the EA score from Lee et al. (2018) as well dummies for childhood SES and completed years of maternal education. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

3.7 Conclusion

We study the relationship between genetic risk for Alzheimer's disease and a range of cognitive and economic outcomes in the Health and Retirement Study. Observed genetic factors—specifically an AD polygenic score and the APOE-$\varepsilon 4$ allele—predict cognition and the risk of diagnosis for a memory-related disease. Relationships between these genetic factors and cognition remain even after controlling for the self-reported diagnosis of a memory disease, suggesting that these measures contain useful information about cognitive health beyond standard observables. Those at higher genetic risk for AD (higher AD scores) are less likely to work for pay and are observed with lower levels of total income. Despite the economically meaningful differences in cognitive and economic outcomes based on these measures, those at higher genetic risk do not appear to expect greater mortality risk, or a greater likelihood of living in a nursing home (except for carriers of the APOE-$\varepsilon 4$ allele). Moreover, those at greater genetic risk are not more likely to engage in potentially beneficial planning activities (like will-making or purchasing long-term care insurance). Indeed, those with higher values of the AD score are consistently found to be significantly less likely to engage in such behaviors. Taken together, these results highlight a concerning pattern—the individuals at the greatest genetic risk for developing AD seem to have systematically fewer resources for insulating themselves and their families from long-term financial or medical costs.

One advantage of using observed molecular genetic variation is that it permits the analysis of interactions between environmental or behavioral factors and latent risk for AD in the development of cognitive impairment. We fail to find evidence that education, childhood conditions, or the nearby presence of adult children play significant roles in moderating genetic risks for AD. The other critical advantage of molecular genetic data is that it defines risk groups for AD that are observable much earlier in life. This naturally sets the stage for a series of questions for future research on whether and how learning about one's personal genetic risks might change behavior. If individuals are unaware of their genotypes, how much would the provision of genetic information have to shift subjective beliefs in order to alter planning behaviors? Future research can better explore the possible feasibility and welfare consequences of greater individual-level access to personal genetic information in this context.

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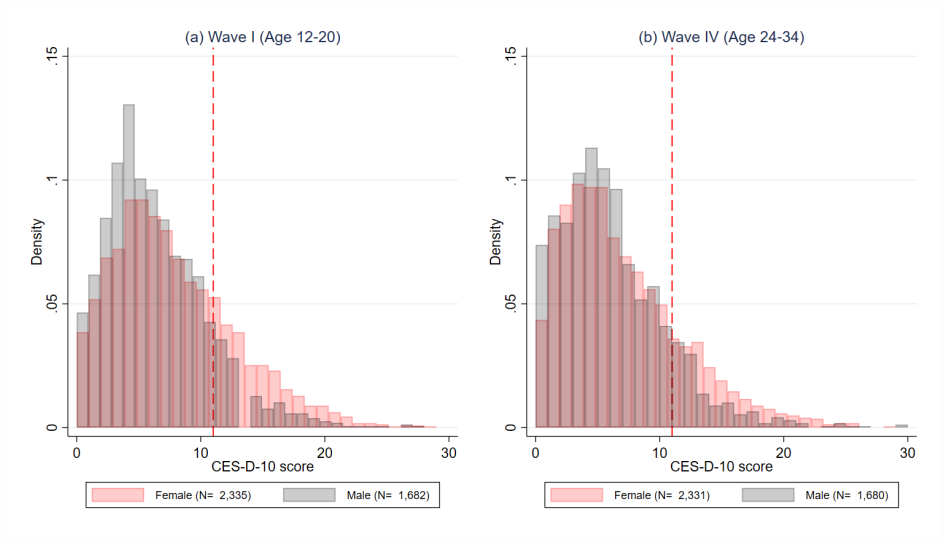
Appendix A

The Effect of Peers' Genetic Predisposition to Depression on Own Mental Health

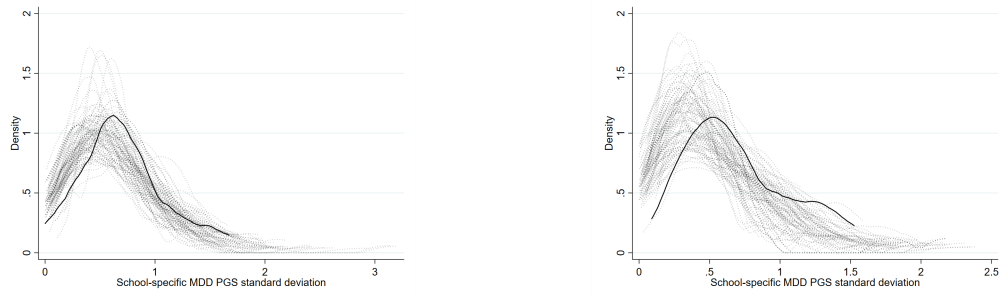
A.1 Figures

Figure A1: CES-D-10 Score Distribution



Note: Panels (a) and (b) show the histogram of the CES-D-10 score for the female (red) and male (gray) samples in Waves I and IV, respectively, along with the CES-D-10 score cutoff of 11 (red dotted line).

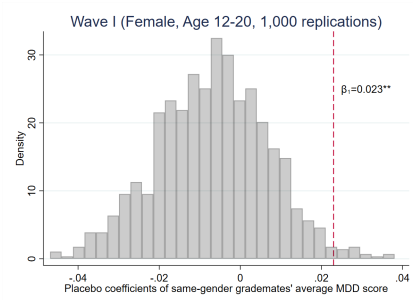
Figure A2: Distributions of Actual vs. Simulated School-Specific MDD Score Standard Deviations



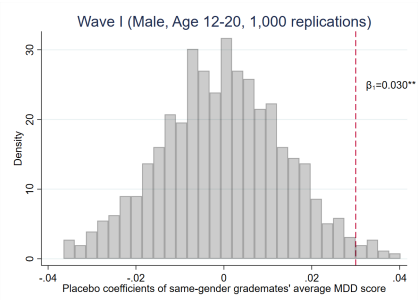
(a) Females (b) Males

Note: The figures in Panels (a) and (b) display distributions of actual (solid line) and simulated (dotted line) school-specific MDD PGS standard deviations separately for the female and male samples, respectively. I show 100 randomly chosen simulated (dotted) school-specific MDD PGS standard deviations along with the actual (solid) standard deviation.

Figure A3: Placebo Coefficients on Same-Gender Grademates' Average MDD Score (Wave I)



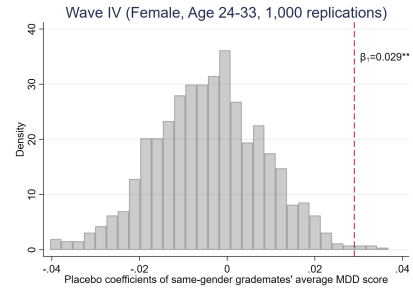
(a) Females



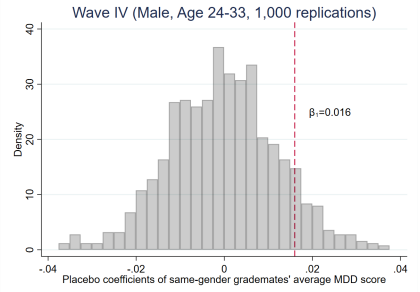
(b) Males

Note: The dotted red line indicates the estimated coefficient from the baseline model. Panels (a) and (b) show the histograms of placebo coefficients for females and males, respectively. In the female and male samples, 1.3% and 4.3% of the placebo coefficients are larger than the actual coefficients, respectively. The number of replications is 1,000.

Figure A4: Placebo Coefficients on Same-Gender Grademates' Average MDD Score (Wave IV)



(a) Females



(b) Males

Note: The dotted red line indicates the estimated coefficient from the baseline model. Panels (a) and (b) show the histograms of placebo coefficients for females and males, respectively. In the female and male samples, 0.1% and 11% of the placebo coefficients are larger than the actual coefficients, respectively. The number of replications is 1,000.

A.2 Tables

Table A1: 10-Item Center for Epidemiologic Studies Depression Scale (CESD-10)

1.	You were bothered by things that don't usually bother you.
2.	You felt that you could not shake off the blues, even with help from your family and your friends.
3.	You felt you were just as good as other people.
4.	You had trouble keeping your mind on what you were doing.
5.	You felt depressed.
6.	You felt that you were too tired to do things.
7.	You were happy.
8.	You enjoyed life.
9.	You felt sad.
10.	You felt that people disliked you.

Note: Scores for each question range from 0 to 3 where 0 means never or rarely and 3 means most or all of the time. Scores for questions 3, 7, and 8 are reverse coded.

Table A2: Summary Statistics of Non-Genotyped Individuals

	Female		Male		p-value	p-value (f)	p-value (m)
	Mean	N	Mean	N			
Demographics:							
Age in Wave I	15.69	5,602	15.83	5,874	0.00	0.00	0.61
	(1.75)		(1.74)				
Age in Wave II	16.20	3,854	16.32	3,842	0.00	0.10	0.02
	(1.64)		(1.65)				
Age in Wave III	21.97	3,800	22.17	3,581	0.00	0.00	0.82
	(1.78)		(1.79)				
Age in Wave IV	28.47	3,486	28.68	2,992	0.00	0.08	0.49
	(1.77)		(1.81)				
Age in Wave V	37.57	3,215	37.79	2,510	0.00	0.00	0.22
	(1.88)		(1.90)				
Race: White	0.50	5,602	0.52	5,874	0.49	0.00	0.00
Race: Black or African-American	0.23	5,602	0.22	5,874	0.01	0.33	0.00
Race: Asian or Pacific Islander	0.10	5,602	0.09	5,874	0.05	0.00	0.44
Race: Other	0.17	5,602	0.16	5,874	0.44	0.00	0.00
Ethnicity: Hispanic	0.22	5,602	0.22	5,874	0.23	0.00	0.00
Family and Parental Characteristics in Wave I:							
Mother's edu: Missing	0.11	5,602	0.13	5,874	0.00	0.00	0.00
Mother's edu: High school/some college	0.47	5,602	0.47	5,874	0.13	0.00	0.00
Mother's edu: College degree or above	0.24	5,602	0.24	5,874	0.05	0.40	0.43
Mother's occ: Missing	0.06	5,602	0.07	5,874	0.02	0.00	0.00
Mother's occ: Managerial/professional	0.23	5,602	0.23	5,874	0.34	0.76	0.31
Mother's occ: Technical/office/sales	0.23	5,602	0.24	5,874	0.23	0.03	0.04
Mother's occ: Blue collar	0.33	5,602	0.30	5,874	0.00	0.84	0.31
Father not present	0.34	5,602	0.29	5,874	0.00	0.04	0.00
Number of siblings	1.47	5,602	1.48	5,874	0.91	0.90	0.74
	(1.26)		(1.28)				
Household income (imputed, thousands dollars)	45.18	5,602	45.43	5,874	0.93	0.52	0.49
	(44.39)		(48.24)				
Depression Measures:							
Depressed (CESD-10 $\geq$ 11) in Wave I	0.26	5,602	0.16	5,874	0.00	0.97	0.33
Depressed (CESD-10 $\geq$ 11) in Wave II	0.26	3,854	0.16	3,842	0.00	0.78	0.32
Depressed (CESD-10 $\geq$ 11) in Wave IV	0.17	3,486	0.13	2,992	0.00	0.00	0.46
Suicidal ideation in Wave I	0.16	5,557	0.10	5,796	0.00	0.95	0.13
Suicidal ideation in Wave II	0.13	3,847	0.08	3,820	0.00	0.79	0.91
Suicidal ideation in Wave IV	0.06	3,482	0.05	2,962	0.00	0.05	0.01
Suicidal ideation in Wave V	0.06	3,147	0.06	2,471	0.23	0.03	0.11
Suicidal attempt in Wave I	0.16	5,557	0.10	5,796	0.00	0.95	0.13
Suicidal attempt in Wave II	0.13	3,847	0.08	3,820	0.00	0.79	0.91
Suicidal attempt in Wave IV	0.06	3,482	0.05	2,962	0.00	0.05	0.01
Suicidal attempt in Wave V	0.06	3,147	0.06	2,471	0.23	0.03	0.11
Ever diagnosed with depression in Wave III	0.13	3,800	0.06	3,581	0.00	0.06	0.11
Ever diagnosed with depression in Wave IV	0.18	3,492	0.08	2,999	0.00	0.01	0.01
Ever diagnosed with depression in Wave V	0.28	3,216	0.15	2,511	0.00	0.05	0.18
Antidepressant use in Wave III	0.06	3,796	0.02	3,585	0.00	0.28	0.99
Antidepressant use in Wave IV	0.06	3,484	0.03	2,996	0.00	0.00	0.37
Antidepressant use in Wave V	0.11	3,215	0.07	2,511	0.00	0.01	0.50

Note: This table shows summary statistics separately for females and males who have *not* been genotyped. Demographics and family and parental characteristics are measured in Wave I. The third from the last column includes p-values from a test of equality of means across the non-genotyped female and male samples. The last two columns include p-values from a test of equality of means across genotyped and non-genotyped individuals separately for females and males.

Table A3: Predictive Power of the MDD Score in the Analysis Sample

	Wave IV					
	CESD-10 Score		Depressed (CESD-10 $\geq$ 11)		Ever Diagnosed with Depression	
	(1) Female	(2) Male	(3) Female	(4) Male	(5) Female	(6) Male
MDD Score	0.353***	0.152	0.023***	0.007	0.027***	0.023***
	(0.104)	(0.104)	(0.008)	(0.008)	(0.008)	(0.007)
Mean	6.771	5.636	0.206	0.124	0.210	0.100
N	2,331	1,680	2,331	1,680	2,331	1,680
R^2	0.025	0.018	0.018	0.010	0.026	0.026
Incremental R^2	.005	.001	.003	0	.004	.006

Note: The outcome variables in columns (1)-(2), (3)-(4), and (5)-(6) are the continuous CES-D-10 score, a binary depression variable (i.e., CES-D-10$\geq$11), and a dummy variable for whether respondents reported ever being diagnosed with depression, respectively. All regressions include age dummies, a female dummy, and race dummies. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$

Table A4: Balancing Tests Controlling for Same-Gender Schoolmates' Average MDD Score

	Females		Males	
MDD Score	-0.022	(0.031)	-0.073*	(0.037)
Age in months	-0.115	(0.294)	-0.190	(0.570)
Race: White	-0.000	(0.019)	-0.028	(0.026)
Race: Black	0.007	(0.013)	0.017	(0.018)
Race: Asian	0.010	(0.008)	0.010	(0.012)
Hispanic	-0.013	(0.011)	-0.002	(0.008)
Number of siblings	0.034	(0.028)	0.050	(0.039)
Father not in household	-0.010	(0.021)	-0.012	(0.035)
Household income (imputed)	2.371	(2.318)	-0.745	(1.345)
Household income (missing)	0.027**	(0.011)	-0.014	(0.013)
Mother's edu: Missing	-0.013	(0.013)	-0.021	(0.018)
Mother's edu: High school graduate/some college	0.004	(0.024)	0.044	(0.045)
Mother's edu: College graduate and above	0.001	(0.018)	-0.021	(0.043)
Mother's occ: Missing	-0.015	(0.010)	-0.038***	(0.014)
Mother's occ: Managerial/professional	0.009	(0.024)	0.005	(0.026)
Mother's occ: Technical/office/sales	-0.017	(0.019)	0.038	(0.027)
Mother's occ: Blue collar	0.029	(0.028)	0.017	(0.034)
PC1	-0.001*	(0.001)	-0.000	(0.001)
PC2	-0.001	(0.001)	0.001**	(0.000)
PC3	-0.001*	(0.000)	-0.000	(0.001)
PC4	-0.000	(0.001)	0.000	(0.001)
PC5	-0.000	(0.001)	-0.000	(0.001)
PC6	0.000	(0.001)	-0.000	(0.001)
PC7	0.000	(0.001)	-0.000	(0.001)
PC8	-0.001*	(0.001)	-0.002	(0.001)
PC9	-0.000	(0.000)	-0.000	(0.001)
PC10	0.000	(0.000)	0.001***	(0.000)
N	2,335		1,682	

Note: Each row includes coefficients from a separate regression of a covariate on same-gender grademates' average MDD score conditional on school and grade fixed effects, school-grade level controls, and same-gender schoolmates' average MDD score. Standard errors are clustered at the school level and included in the parenthesis. All individual and family level characteristics are measured in Wave I, and all genetic information (i.e., MDD PGS and PC1-10) is collected in Wave IV. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table A5: Robustness of the Effect of Same-Gender Grademates' and Own MDD Score Depression in Waves I and IV to Different Definitions of Depression

	Females							Males						
	CESD-19		CESD-10					CESD-19		CESD-10				
	≥ 16	CESD-10	≥ 8	≥ 9	≥ 10	≥ 11	≥ 12	≥ 16	CESD-10	≥ 8	≥ 9	≥ 10	≥ 11	≥ 12
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)
Panel A: Wave I														
Same-Gender Grademates' Average MDD Score	0.015	0.125	0.037***	0.031***	0.029***	0.023**	0.007	0.014	0.254	0.024	0.013	0.035**	0.030**	0.020
	(0.011)	(0.109)	(0.012)	(0.010)	(0.009)	(0.010)	(0.010)	(0.015)	(0.196)	(0.026)	(0.020)	(0.017)	(0.015)	(0.014)
Own MDD Score	0.022**	0.286**	0.022**	0.024**	0.018*	0.018**	0.018**	0.020	0.175	0.024	0.012	0.010	0.013	0.008
	(0.010)	(0.116)	(0.011)	(0.010)	(0.009)	(0.009)	(0.008)	(0.015)	(0.138)	(0.018)	(0.018)	(0.013)	(0.010)	(0.010)
Mean	0.299	7.618	0.439	0.373	0.316	0.262	0.211	0.201	6.271	0.336	0.271	0.207	0.150	0.111
N	2,334	2,335	2,335	2,335	2,335	2,335	2,335	1,679	1,682	1,682	1,682	1,682	1,682	1,682
R^2	0.153	0.159	0.142	0.142	0.141	0.132	0.127	0.155	0.179	0.169	0.148	0.153	0.147	0.135
Panel B: Wave IV														
		(1)	(2)	(3)	(4)	(5)	(6)		(7)	(8)	(9)	(10)	(11)	(12)
Same-Gender Grademates' Average MDD Score		0.377**	0.029**	0.026*	0.030**	0.029**	0.024*		-0.065	0.015	0.026**	0.024**	0.016	0.015*
		(0.143)	(0.014)	(0.013)	(0.013)	(0.013)	(0.013)		(0.141)	(0.014)	(0.012)	(0.011)	(0.010)	(0.008)
Own MDD Score		0.344***	0.027***	0.030***	0.022***	0.024***	0.021***		0.112	0.022*	0.014	0.010	0.010	0.004
		(0.089)	(0.010)	(0.009)	(0.008)	(0.007)	(0.007)		(0.095)	(0.012)	(0.010)	(0.008)	(0.006)	(0.007)
Mean		6.771	0.369	0.308	0.254	0.206	0.171		5.636	0.274	0.223	0.165	0.124	0.090
N		2,331	2,331	2,331	2,331	2,331	2,331		1,680	1,680	1,680	1,680	1,680	1,680
R^2		0.141	0.140	0.127	0.117	0.119	0.117		0.141	0.127	0.131	0.118	0.125	0.118

Note: In columns (1) and (8), the outcome is the continuous CES-D-19 score. The outcome in columns (2) and (9) is the continuous CES-D-10 score. In columns (3)-(7) and (10)-(14), the outcomes are indicator variables for being depressed based on CES-D-10 cutoffs of 8, 9, 10, 11, and 12. Each column includes a separate regression result. All regressions include the controls in my most preferred specification (columns 4 and 8 of Table 1.4). Standard errors are clustered at the school level. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table A6: Nonlinear Effects of Same-Gender Grademates' and Own MDD Score on Mental Health in Waves I and IV

	Nonlinearities: Depressed (CES-D-10 $\geq$ 11)							
	Wave I				Wave IV			
	Females		Males		Females		Males	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Above Median Peer Avg MDD Score	0.025		-0.008		0.066**		0.018	
	(0.026)		(0.026)		(0.028)		(0.021)	
Tercile 1 of Peer Avg MDD Score		0.029		-0.016		-0.017		-0.042
		(0.028)		(0.031)		(0.028)		(0.030)
Tercile 3 of Peer Avg MDD Score		0.076***		0.011		0.076**		0.002
		(0.026)		(0.030)		(0.031)		(0.026)
Own MDD Score	0.016*	0.018*	0.008	0.010	0.023***	0.024***	0.009	0.011*
	(0.009)	(0.009)	(0.010)	(0.011)	(0.007)	(0.007)	(0.007)	(0.006)
Mean	0.262	0.262	0.150	0.150	0.206	0.206	0.124	0.124
N	2,335	2,335	1,682	1,682	2,331	2,331	1,680	1,680
R^2	0.131	0.134	0.146	0.146	0.120	0.121	0.125	0.126

Note: The outcome is an indicator for being depressed (i.e., CES-D-10 $\geq$ 11) in Waves I and IV, and each column includes separate regression results. Columns (1)-(4) and (5)-(8) present the point estimates for Waves I and IV, respectively. The variable of interest in columns (1), (3), (5), and (7) is an indicator variable for whether same-gender grademates' average MDD score is above the median and zero otherwise. The variables of interest in columns (2), (4), (6), and (8) are indicator variables representing the first and the third terciles of same-gender grademates' average MDD score. All regressions include the controls in my most preferred specification (columns 4 and 8 of Table 1.4). Standard errors are clustered at the school level. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table A7: Effect of Same-Gender Grademates' and Own MDD Score on Other Mental Health Outcomes in Waves I and IV

	Wave I				Wave IV							
	Suicidal Ideation		Suicide Attempts		Suicidal Ideation		Suicide Attempts		Depression Diagnosis		Antidepressant Use	
	(1) Female	(2) Male	(3) Female	(4) Male	(5) Female	(6) Male	(7) Female	(8) Male	(9) Female	(10) Male	(11) Female	(12) Male
Same-Gender Grademates' Average MDD Score	0.005	-0.005	0.004	0.003	0.022***	-0.004	0.007*	0.005	0.018	0.007	0.001	-0.004
	(0.015)	(0.013)	(0.009)	(0.007)	(0.007)	(0.007)	(0.004)	(0.005)	(0.012)	(0.009)	(0.008)	(0.009)
Own MDD Score	0.014**	-0.011	0.007	0.001	0.002	-0.001	0.003	-0.001	0.030***	0.026***	0.016***	0.009*
	(0.006)	(0.007)	(0.005)	(0.005)	(0.005)	(0.006)	(0.002)	(0.002)	(0.009)	(0.007)	(0.005)	(0.005)
Mean	0.161	0.111	0.053	0.023	0.075	0.065	0.013	0.018	0.210	0.100	0.081	0.035
N	2,327	1,671	2,327	1,671	2,327	1,665	2,328	1,665	2,331	1,680	2,323	1,675
R^2	0.099	0.121	0.108	0.115	0.108	0.112	0.086	0.165	0.129	0.157	0.107	0.165

Note: The outcomes in columns (1)-(2) and (3)-(4) are indicators for suicidal ideation and suicide attempts, respectively, in Wave I. The outcomes in columns (5)-(6), (7)-(8), (9)-(10), and (11)-(12) are indicators for suicidal ideation, suicide attempts, depression diagnosis, and antidepressant use, respectively, in Wave IV. Each column includes a separate regression result. All regressions include the controls in my most preferred specification (columns 4 and 8 of Table 1.4). Standard errors are clustered at the school level. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table A8: Effect of Same-Gender Grademates' MDD Score on Attrition

	Attrition from Wave I to IV		Absence of Valid Genetic Data	
	(1) Females	(2) Males	(3) Females	(4) Males
Same-Gender Grademates' Average MDD Score	-0.023	0.053	-0.012	0.131
	(0.028)	(0.035)	(0.074)	(0.098)
Mean	0.159	0.247	0.431	0.413
N	3,224	2,512	2,711	1,891
R^2	0.083	0.106	0.164	0.186

Note: The outcome in columns (1) and (2) is an indicator for whether the respondent drops out of the survey from Waves I to IV. The outcome in columns (3) and (4) is an indicator for whether the respondent decided not to be genotyped conditional on being interviewed at Wave IV. Each column includes a separate regression result. All regressions include the controls in my most preferred specification (columns 4 and 8 of Table 1.4) except for the 10 principal components and own MDD score. Standard errors are clustered at the schoosl level. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table A9: Effect of Same-Gender Grademates' and Own MDD Score on Additional Friendship Measures in Wave I

	During the Past Week or Weekend				How Much Friends Care About You			
	Talk About Problems		Talk on Phone		Quite a Bit or More		Very Much	
	(1) Females	(2) Males	(3) Females	(4) Males	(5) Females	(6) Males	(7) Females	(8) Males
Same-Gender Grademates' Average MDD Score	-0.018	0.011	-0.015	-0.010	-0.020	0.001	-0.023	-0.003
	(0.013)	(0.023)	(0.011)	(0.013)	(0.012)	(0.015)	(0.014)	(0.018)
Own MDD Score	-0.007	0.021	-0.008	0.015	-0.012*	-0.006	-0.029***	0.002
	(0.009)	(0.014)	(0.007)	(0.011)	(0.007)	(0.009)	(0.010)	(0.012)
Mean	0.766	0.497	0.863	0.795	0.881	0.802	0.507	0.343
N	2,277	1,637	2,277	1,637	2,331	1,678	2,331	1,678
R^2	0.190	0.191	0.138	0.170	0.129	0.132	0.113	0.145

Note: The outcome variable in columns (1) and (2) is an indicator for whether respondents talk to one or more nominated friends on the phone during the past week. The outcome variable in columns (3) and (4) is an indicator variable for whether respondents talk to one or more nominated friends about a problem during the past week. The outcomes in columns (5)-(6) and (7)-(8) are indicator variables for whether respondents feel that their friends care about them quite a bit or more and very much, respectively. All regressions include the controls in my most preferred specification (columns 4 and 8 of Table 1.4). Standard errors are clustered at the school level. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table A10: Effect of Same-Gender Grademates' and Own MDD Score on Sense of Belonging at School in Wave I

	Number of Sense of Belonging Statements Strongly Disagreed or Disagreed With					
	One or More		Two or More		All Three	
	(1) Females	(2) Males	(3) Females	(4) Males	(5) Females	(6) Males
Same-Gender Grademates' Average MDD Score	0.015	-0.008	0.019*	-0.011	0.001	-0.006
	(0.015)	(0.020)	(0.011)	(0.014)	(0.009)	(0.008)
Own MDD Score	0.017*	0.021**	0.020**	0.018**	0.004	0.008
	(0.009)	(0.009)	(0.008)	(0.007)	(0.005)	(0.007)
Mean	0.263	0.245	0.129	0.105	0.053	0.042
N	2,334	1,682	2,334	1,682	2,334	1,682
R^2	0.130	0.133	0.109	0.150	0.129	0.123

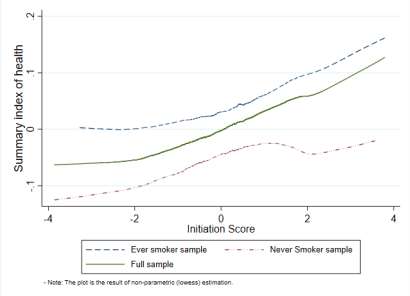
Note: As measures of sense of belonging at school, I use questions asking how strongly one disagrees or disagrees with the following: "I feel close to people at this school", "I feel like I am part of this school", and "I am happy to be at this school." The outcome variables in columns (1)-(2), (3)-(4), and (5)-(6) are indicator variables for whether individuals strongly disagree or disagree with 1 or more, 2 or more, and 3 of those statements, respectively. All regressions include the controls in my most preferred specification (columns 4 and 8 of Table 1.4). Standard errors are clustered at the school level. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Appendix B

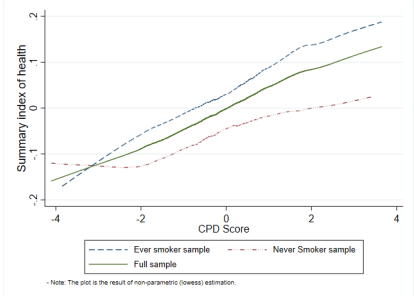
The Complicated Links Between Health and the Genetic Endowments for Smoking with Meghan Skira

B.1 Figures

Figure B1: Relationship between smoking polygenic scores and the summary health index separately for full, ever, and never smokers samples



(a) Initiation Score and Summary Health Index



(b) CPD Score and Summary Health Index

Note: Panel A shows the lowess plot of the initiation score and the summary health index separately for full, ever, and never smokers samples, and Panel B shows the lowess plot of the CPD score and the summary health index separately for full, ever, and never smokers samples.

B.2 Tables

Table B1: Summary Statistics for Non-Genotyped Individuals

	Full Sample			Ever Smoker Sample			Never Smoker Sample			*p*-value
	Mean	SD	N	Mean	SD	N	Mean	SD	N	
Demographics:										
Age	58.20	4.18	31,015	58.38	4.15	18,877	57.93	4.21	12,138	0.00
Birth Year	1945.38	9.14	31,015	1944.59	9.01	18,877	1946.59	9.22	12,138	0.00
Male	0.46	0.50	31,015	0.53	0.50	18,877	0.35	0.48	12,138	0.00
Own Smoking:										
# Cigs per Day (Among Current Smokers)	17.76	12.30	7,076	17.76	12.30	7,076				
Maximum # Cigs per Day (Among Former Smokers)	19.43	17.76	8,183	19.43	17.76	8,183				
Age Start Smoking (Among Ever Smokers)	18.04	5.56	13,753	18.04	5.56	13,753				
Age Quit Smoking (Among Quitters)	39.09	11.59	10,105	39.09	11.59	10,105				
Smoked as a Teenager	0.36	0.48	11,722	0.36	0.48	11,722				
Own Health:										
Poor Self-Reported Healt	0.29	0.46	30,995	0.32	0.47	18,867	0.26	0.44	12,128	0.00
Any Hospital Stay	0.20	0.40	30,813	0.22	0.42	18,775	0.17	0.37	12,038	0.00
Ever Had High Blood Pressure	0.42	0.49	30,986	0.42	0.49	18,861	0.42	0.49	12,125	0.53
Ever Had Diabetes	0.16	0.37	30,967	0.16	0.37	18,843	0.16	0.37	12,124	0.76
Ever Had Cancer	0.08	0.28	30,951	0.09	0.29	18,837	0.07	0.26	12,114	0.00
Ever Had Lung Disease	0.08	0.26	30,997	0.11	0.31	18,867	0.03	0.17	12,130	0.00
Ever Had Heart Problem	0.14	0.35	30,986	0.17	0.37	18,857	0.10	0.30	12,129	0.00
Ever Had Stroke	0.04	0.20	30,998	0.05	0.23	18,869	0.02	0.16	12,129	0.00
Ever Had Arthritis	0.42	0.49	30,980	0.45	0.50	18,861	0.38	0.49	12,119	0.00
Any Limitations with ADLs	0.13	0.34	30,925	0.15	0.36	18,834	0.11	0.31	12,091	0.00
Any Limitations with IADLs	0.08	0.26	30,917	0.09	0.28	18,831	0.06	0.24	12,086	0.00
Body Mass Index (kg/m^2)	28.17	5.85	30,286	27.87	5.79	18,520	28.65	5.93	11,766	0.00
Obese	0.32	0.47	30,414	0.30	0.46	18,554	0.35	0.48	11,860	0.00
Parental Smoking:										
Neither Parent Smoked	0.18	0.39	31,015	0.14	0.35	18,877	0.25	0.43	12,138	0.00
One Parent Smoked	0.25	0.43	31,015	0.24	0.43	18,877	0.27	0.44	12,138	0.00
Both Parents Smoked	0.13	0.34	31,015	0.15	0.35	18,877	0.11	0.32	12,138	0.00
Missing	0.43	0.49	31,015	0.47	0.50	18,877	0.37	0.48	12,138	0.00
Parental Mortality:										
Neither Parent Died During Childhood	0.07	0.25	31,015	0.06	0.24	18,877	0.08	0.27	12,138	0.00
Parent(s) Died During Childhood	0.02	0.15	31,015	0.02	0.15	18,877	0.02	0.15	12,138	0.63
Parental Mortality During Childhood Missing	0.91	0.29	31,015	0.91	0.28	18,877	0.90	0.30	12,138	0.00
Mother Currently Alive	0.37	0.48	31,015	0.35	0.48	18,877	0.41	0.49	12,138	0.00
Mother Died Before Age 65	0.17	0.37	31,015	0.18	0.38	18,877	0.15	0.36	12,138	0.00
Mother Died Age 65+	0.44	0.50	31,015	0.45	0.50	18,877	0.41	0.49	12,138	0.00
Mother Mortality Information Missing	0.02	0.15	31,015	0.03	0.16	18,877	0.02	0.14	12,138	0.00
Father Currently Alive	0.16	0.37	31,015	0.15	0.35	18,877	0.19	0.40	12,138	0.00
Father Died Before Age 65	0.25	0.43	31,015	0.27	0.44	18,877	0.23	0.42	12,138	0.00
Father Died Age 65+	0.55	0.50	31,015	0.55	0.50	18,877	0.55	0.50	12,138	0.80
Father Mortality Information Missing	0.03	0.18	31,015	0.04	0.19	18,877	0.03	0.17	12,138	0.02
Parental Risky Behavior During Respondent's Childhood:										
Parent(s) Drank/Used Drugs Often	0.10	0.30	31,015	0.11	0.31	18,877	0.09	0.28	12,138	0.00
Neither Parent Drank/Used Drugs	0.40	0.49	31,015	0.37	0.48	18,877	0.45	0.50	12,138	0.00
Missing	0.50	0.50	31,015	0.53	0.50	18,877	0.46	0.50	12,138	0.00

Note: The table presents summary statistics for the full sample and separately for those who ever and never smoked. The sample includes those aged 50–65 of European ancestry who self-identify as white and who have *not* been genotyped. The last column presents *p*-values from a test of equality of means for ever and never smokers.

Table B2: Relationship between the Smoking Polygenic Scores and Smoking Behavior

	Max # of Cigarettes Smoked per Day			Ever Smoked		
	(1)	(2)	(3)	(4)	(5)	(6)
CPD Score	2.044***		1.949***	0.027***		0.016***
	(0.218)		(0.219)	(0.005)		(0.005)
Initiation Score		1.155***	0.924***		0.095***	0.093***
		(0.235)	(0.234)		(0.005)	(0.005)
Standard Controls	Yes	Yes	Yes	Yes	Yes	Yes
Principal Components	Yes	Yes	Yes	Yes	Yes	Yes
Sample Mean	22.468	22.468	22.468	0.583	0.583	0.583
N	5,392	5,392	5,392	9,488	9,488	9,488
R^2	0.103	0.092	0.105	0.049	0.078	0.079
Incremental R^2	0.015	0.004	0.018	0.003	0.031	0.032

Note: Each column presents results from a separate regression. In columns (1)–(3), the outcome is the maximum number of cigarettes smoked per day among current and former smokers. In columns (4)–(6), the outcome is an indicator for whether the individual has ever smoked as of the last time we observe them in our sample. In all specifications, we include the standard controls and the genetic principal components described in Section 2.4. In columns (1) and (4), we report the incremental R^2 of the CPD score; in columns (2) and (5), we report the incremental R^2 of the initiation score; and in columns (3) and (6), we report the incremental R^2 of both scores. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table B3: Relationship between the Smoking Polygenic Scores and Summary Standardized Index of Doctor-Diagnosed Conditions

	Full Sample			Ever Smoker Sample			Never Smoker Sample	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
CPD Score	0.035***	0.036***	0.029***	0.044***	0.044***	0.034***	0.021***	0.022***
	(0.005)	(0.005)	(0.005)	(0.007)	(0.007)	(0.007)	(0.006)	(0.007)
Initiation Score	0.020***	0.027***	0.013**	0.012*	0.016**	0.004	0.017**	0.023***
	(0.005)	(0.005)	(0.005)	(0.007)	(0.007)	(0.007)	(0.007)	(0.007)
Full Smoking Controls	No	No	Yes	No	No	Yes	No	No
Standard Controls	No	Yes	Yes	No	Yes	Yes	No	Yes
Principal Components	No	Yes	Yes	No	Yes	Yes	No	Yes
Sample Mean	-0.000	-0.000	-0.000	0.037	0.037	0.037	-0.051	-0.051
N	45,850	45,850	45,850	26,509	26,509	26,509	19,341	19,341
R^2	0.008	0.083	0.123	0.009	0.093	0.145	0.004	0.092

Note: Each column presents results from a separate regression where the outcome is a summary standardized index of the doctor-diagnosed conditions. Results are presented for the full sample and then separately for ever smokers and never smokers. For each sample, we first present results without controls (columns 1, 4, 7). Then we add the standard controls and the genetic principal components described in Section 2.4 (columns 2, 5, 8). Then for the full sample and ever smoker sample, we add the full set of smoking controls described in Section 2.4 (columns 3, 6). * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table B4: Relationship between the Smoking Polygenic Scores and Individual Health Measures Accounting for Parental Smoking, Risky Behaviors, and Mortality (Full Sample)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)
			Ever Diagnosed With:							Any Limitations With:			
	Poor Self-Reported Health	Any Hospitalization	High Blood Pressure	Diabetes	Cancer	Lung Disease	Heart Problem	Stroke	Arthritis	ADLs	IADLs	BMI	Obese
Panel A: Full Sample without Parental Controls													
CPD Score	0.019***	0.010***	0.017***	0.019***	0.000	0.011***	0.007**	-0.000	0.021***	0.010***	0.004***	0.502***	0.033***
	(0.003)	(0.002)	(0.005)	(0.003)	(0.003)	(0.002)	(0.003)	(0.002)	(0.005)	(0.002)	(0.001)	(0.061)	(0.005)
Initiation Score	0.018***	0.008***	0.005	0.003	-0.004	0.009***	0.005	0.004**	0.004	0.007***	0.005***	0.229***	0.016***
	(0.003)	(0.003)	(0.005)	(0.003)	(0.003)	(0.003)	(0.004)	(0.002)	(0.005)	(0.002)	(0.001)	(0.067)	(0.005)
Full Smoking Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Sample Mean	0.170	0.174	0.386	0.114	0.084	0.064	0.132	0.030	0.451	0.080	0.036	28.122	0.312
N	46,676	46,624	46,659	46,663	46,637	46,671	46,667	46,669	46,654	46,669	46,667	46,115	46,115
R^2	0.068	0.026	0.058	0.058	0.033	0.087	0.052	0.033	0.075	0.037	0.030	0.084	0.060
Panel B: Full Sample with Parental Controls													
CPD Score	0.017***	0.009***	0.015***	0.018***	0.000	0.011***	0.006*	-0.001	0.020***	0.009***	0.003**	0.488***	0.032***
	(0.003)	(0.002)	(0.005)	(0.003)	(0.003)	(0.002)	(0.003)	(0.002)	(0.005)	(0.002)	(0.001)	(0.060)	(0.005)
Initiation Score	0.016***	0.007**	0.003	0.003	-0.005	0.009***	0.003	0.004**	0.003	0.006**	0.004***	0.206***	0.015***
	(0.003)	(0.003)	(0.005)	(0.003)	(0.003)	(0.003)	(0.004)	(0.002)	(0.005)	(0.002)	(0.001)	(0.066)	(0.005)
Parental Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Full Smoking Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Sample Mean	0.170	0.174	0.386	0.114	0.084	0.064	0.132	0.030	0.451	0.080	0.036	28.122	0.312
N	46,676	46,624	46,659	46,663	46,637	46,671	46,667	46,669	46,654	46,669	46,667	46,115	46,115
R^2	0.080	0.030	0.064	0.063	0.034	0.090	0.057	0.035	0.078	0.045	0.035	0.089	0.064

Note: Each column within a panel presents results from a separate regression where the outcome is noted in the column heading. Results for the full sample without parental controls are presented in Panel A and with parental controls in Panel B. In all specifications, we include the standard controls and the genetic principal components described in Section 2.4. We also include the full set of smoking controls described in Section 2.4. In Panel B, we add controls for parental smoking, parental risky behaviors, and parental mortality as described in Section 2.3.4.

Table B5: Relationship between the Smoking Polygenic Scores and Individual Health Measures Accounting for Parental Smoking, Risky Behaviors, and Mortality (Ever Smoker Sample)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)
			Ever Diagnosed With:							Any Limitations With:			
	Poor Self-Reported Health	Any Hospitalization	High Blood Pressure	Diabetes	Cancer	Lung Disease	Heart Problem	Stroke	Arthritis	ADLs	IADLs	BMI	Obese
Panel A: Ever Smoker Sample without Parental Controls													
CPD Score	0.018***	0.013***	0.021***	0.016***	-0.001	0.018***	0.010**	-0.000	0.022***	0.010***	0.003*	0.387***	0.027***
	(0.004)	(0.003)	(0.007)	(0.004)	(0.004)	(0.004)	(0.005)	(0.003)	(0.007)	(0.003)	(0.002)	(0.078)	(0.006)
Initiation Score	0.015***	0.003	-0.002	-0.001	-0.006	0.010**	0.001	0.003	-0.002	0.004	0.006**	0.107	0.008
	(0.005)	(0.004)	(0.007)	(0.004)	(0.004)	(0.004)	(0.005)	(0.003)	(0.007)	(0.003)	(0.002)	(0.086)	(0.006)
Full Smoking Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Sample Mean	0.202	0.189	0.398	0.115	0.083	0.090	0.149	0.038	0.470	0.093	0.043	28.046	0.305
N	26,952	26,922	26,932	26,949	26,919	26,956	26,951	26,947	26,945	26,952	26,951	26,681	26,681
R^2	0.086	0.035	0.082	0.075	0.049	0.103	0.067	0.044	0.088	0.051	0.044	0.121	0.086
Panel B: Ever Smoker Sample with Parental Controls													
CPD Score	0.016***	0.012***	0.020***	0.014***	-0.001	0.017***	0.010**	-0.001	0.021***	0.009***	0.003	0.382***	0.027***
	(0.004)	(0.003)	(0.007)	(0.004)	(0.004)	(0.004)	(0.005)	(0.003)	(0.007)	(0.003)	(0.002)	(0.078)	(0.006)
Initiation Score	0.015***	0.003	-0.003	-0.000	-0.006	0.010**	0.000	0.003	-0.002	0.003	0.005**	0.105	0.008
	(0.005)	(0.004)	(0.007)	(0.004)	(0.004)	(0.004)	(0.005)	(0.003)	(0.007)	(0.003)	(0.002)	(0.086)	(0.006)
Parental Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Full Smoking Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Sample Mean	0.202	0.189	0.398	0.115	0.083	0.090	0.149	0.038	0.470	0.093	0.043	28.046	0.305
N	26,952	26,922	26,932	26,949	26,919	26,956	26,951	26,947	26,945	26,952	26,951	26,681	26,681
R^2	0.097	0.039	0.087	0.081	0.051	0.107	0.073	0.046	0.090	0.059	0.049	0.124	0.088

Note: Each column within a panel presents results from a separate regression where the outcome is noted in the column heading. Results for the ever smoker sample without parental controls are presented in Panel A and with parental controls in Panel B. In all specifications, we include the standard controls and the genetic principal components described in Section 2.4. We also include the full set of smoking controls described in Section 2.4. In Panel B, we add controls for parental smoking, parental risky behaviors, and parental mortality as described in Section 2.3.4.

Table B6: Relationship between the Smoking Polygenic Scores and Individual Health Measures Accounting for Parental Smoking, Risky Behaviors, and Mortality (Never Smoker Sample)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)
			Ever Diagnosed With:							Any Limitations With:			
	Poor Self-Reported Health	Any Hospitalization	High Blood Pressure	Diabetes	Cancer	Lung Disease	Heart Problem	Stroke	Arthritis	ADLs	IADLs	BMI	Obese
Panel A: Never Smoker Sample without Parental Controls													
CPD Score	0.021***	0.004	0.011	0.024***	0.002	0.004	0.002	-0.001	0.017**	0.009***	0.004**	0.641***	0.041***
	(0.004)	(0.004)	(0.008)	(0.005)	(0.004)	(0.003)	(0.005)	(0.002)	(0.008)	(0.003)	(0.002)	(0.097)	(0.007)
Initiation Score	0.019***	0.013***	0.014*	0.008	-0.004	0.007**	0.008	0.006***	0.012	0.011***	0.003*	0.373***	0.026***
	(0.005)	(0.004)	(0.008)	(0.005)	(0.005)	(0.003)	(0.005)	(0.002)	(0.008)	(0.003)	(0.002)	(0.104)	(0.008)
Sample Mean	0.126	0.154	0.370	0.111	0.085	0.029	0.109	0.020	0.424	0.063	0.026	28.227	0.321
N	19,724	19,702	19,727	19,714	19,718	19,715	19,716	19,722	19,709	19,717	19,716	19,434	19,434
R^2	0.032	0.017	0.050	0.053	0.032	0.031	0.039	0.026	0.070	0.024	0.014	0.062	0.049
Panel B: Never Smoker Sample with Parental Controls													
CPD Score	0.019***	0.003	0.009	0.023***	0.001	0.003	0.001	-0.001	0.015**	0.008***	0.003*	0.615***	0.040***
	(0.004)	(0.004)	(0.007)	(0.005)	(0.004)	(0.003)	(0.005)	(0.002)	(0.008)	(0.003)	(0.002)	(0.096)	(0.007)
Initiation Score	0.015***	0.011***	0.010	0.006	-0.004	0.007**	0.006	0.006***	0.009	0.008**	0.002	0.313***	0.022***
	(0.005)	(0.004)	(0.008)	(0.006)	(0.005)	(0.003)	(0.005)	(0.002)	(0.008)	(0.003)	(0.002)	(0.103)	(0.008)
Parental Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Sample Mean	0.126	0.154	0.370	0.111	0.085	0.029	0.109	0.020	0.424	0.063	0.026	28.227	0.321
N	19,724	19,702	19,727	19,714	19,718	19,715	19,716	19,722	19,709	19,717	19,716	19,434	19,434
R^2	0.048	0.022	0.062	0.059	0.034	0.034	0.047	0.030	0.076	0.035	0.018	0.079	0.062

Note: Each column within a panel presents results from a separate regression where the outcome is noted in the column heading. Results for the never smoker sample without parental controls are presented in Panel A and with parental controls in Panel B. In all specifications, we include the standard controls and the genetic principal components described in Section 2.4. In Panel B, we add controls for parental smoking, parental risky behaviors, and parental mortality as described in Section 2.3.4.

Table B7: Smoking Polygenic Scores and Assortative Mating

Husband's CPD Score Quartile	**Panel A:** CPD Score (N=2,663)			
	Wife's CPD Score Quartile			
	Q1	Q2	Q3	Q4
Q1	30.4	25.4	25.5	24.7
Q2	22.3	25.7	25.0	26.2
Q3	22.6	23.2	25.0	28.2
Q4	24.7	25.7	24.4	20.9
Husband's Initiation Score Quartile	**Panel B:** Initiation Score (N=2,663)			
	Wife's Initiation Score Quartile			
	Q1	Q2	Q3	Q4
Q1	25.0	28.3	23.4	24.0
Q2	25.3	24.5	26.8	27.4
Q3	26.8	24.2	27.0	22.0
Q4	22.8	23.1	22.8	26.6

Note: Panel A (B) shows the distribution of the husband's CPD (initiation) score conditional on the quartile of the wife's CPD (initiation) score for all individuals in couples where both members were genotyped and aged 50–65. The column probabilities sum to 100.

Table B8: Relationship between the Smoking Polygenic Scores and Individual Health Measures Accounting for Spouse's Smoking Behavior and Smoking Polygenic Scores (Full Sample)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)
			Ever Diagnosed With:							Any Limitations With:			
	Poor Self-Reported Health	Any Hospitalization	High Blood Pressure	Diabetes	Cancer	Lung Disease	Heart Problem	Stroke	Arthritis	ADLs	IADLs	BMI	Obese
Panel A: Full Sample without Spousal Controls													
CPD Score	0.016***	0.006*	0.007	0.023***	0.003	0.010***	0.001	-0.000	0.015**	0.009***	0.001	0.462***	0.033***
	(0.004)	(0.003)	(0.007)	(0.005)	(0.004)	(0.003)	(0.005)	(0.002)	(0.007)	(0.003)	(0.002)	(0.079)	(0.006)
Initiation Score	0.016***	0.008**	0.007	-0.003	-0.007	0.006*	-0.003	0.003	0.016**	0.006**	0.002	0.225**	0.019***
	(0.004)	(0.004)	(0.008)	(0.005)	(0.004)	(0.003)	(0.005)	(0.002)	(0.008)	(0.003)	(0.002)	(0.088)	(0.007)
Full Smoking Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Sample Mean	0.139	0.164	0.376	0.108	0.076	0.048	0.128	0.027	0.426	0.066	0.028	28.064	0.307
N	21,682	21,667	21,675	21,679	21,676	21,679	21,681	21,687	21,671	21,682	21,681	21,448	21,448
R^2	0.075	0.035	0.072	0.074	0.049	0.088	0.066	0.067	0.078	0.049	0.041	0.105	0.078
Panel B: Full Sample with Spousal Controls													
CPD Score	0.014***	0.006*	0.005	0.022***	0.004	0.010***	0.000	-0.001	0.013*	0.008***	0.001	0.445***	0.031***
	(0.004)	(0.004)	(0.007)	(0.004)	(0.004)	(0.003)	(0.005)	(0.002)	(0.007)	(0.003)	(0.002)	(0.079)	(0.006)
Initiation Score	0.013***	0.006*	0.003	-0.004	-0.007*	0.005	-0.005	0.002	0.016**	0.004	0.001	0.183**	0.016**
	(0.004)	(0.004)	(0.008)	(0.005)	(0.004)	(0.003)	(0.005)	(0.002)	(0.008)	(0.003)	(0.002)	(0.086)	(0.007)
Spouse's CPD Score	0.016***	0.009***	0.014**	0.009**	0.000	0.005*	0.003	0.001	0.009	0.007***	0.003*	0.217***	0.016**
	(0.004)	(0.003)	(0.007)	(0.004)	(0.004)	(0.003)	(0.005)	(0.002)	(0.007)	(0.003)	(0.002)	(0.077)	(0.006)
Spouse's Initiation Score	0.010**	0.009**	0.001	-0.005	-0.002	-0.007**	0.003	-0.001	-0.001	-0.002	0.000	-0.024	-0.005
	(0.005)	(0.004)	(0.007)	(0.005)	(0.004)	(0.003)	(0.005)	(0.002)	(0.008)	(0.003)	(0.002)	(0.088)	(0.007)
Spouse's Full Smoking Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Spouse's Principal Components	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Full Smoking Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Sample Mean	0.139	0.164	0.376	0.108	0.076	0.048	0.128	0.027	0.426	0.066	0.028	28.064	0.307
N	21,682	21,667	21,675	21,679	21,676	21,679	21,681	21,687	21,671	21,682	21,681	21,448	21,448
R^2	0.099	0.049	0.102	0.097	0.082	0.116	0.091	0.097	0.107	0.065	0.062	0.141	0.108

Note: Each column within a panel presents results from a separate regression where the outcome is noted in the column heading. Results for the full sample without spousal controls are presented in Panel A and with spousal controls in Panel B. In all specifications, we include the standard controls and the genetic principal components described in Section 2.4. We also include the full set of smoking controls described in Section 2.4. In Panel B, we add the full set of the spouse's smoking controls and the spouse's smoking polygenic scores and their genetic principal components. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table B9: Relationship between the Smoking Polygenic Scores and Individual Health Measures Accounting for Spouse's Smoking Behavior and Smoking Polygenic Scores (Ever Smoker Sample)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)
			Ever Diagnosed With:							Any Limitations With:			
	Poor Self-Reported Health	Any Hospitalization	High Blood Pressure	Diabetes	Cancer	Lung Disease	Heart Problem	Stroke	Arthritis	ADLs	IADLs	BMI	Obese
Panel A: Ever Smoker Sample without Spousal Controls													
CPD Score	0.014**	0.002	0.015	0.021***	0.005	0.015***	0.005	-0.001	0.016	0.008**	0.001	0.298***	0.028***
	(0.006)	(0.005)	(0.010)	(0.006)	(0.005)	(0.005)	(0.007)	(0.003)	(0.010)	(0.004)	(0.003)	(0.103)	(0.009)
Initiation Score	0.010	0.007	-0.000	-0.008	-0.006	0.009	-0.002	0.003	0.010	0.000	0.002	0.009	0.005
	(0.006)	(0.005)	(0.010)	(0.006)	(0.006)	(0.006)	(0.007)	(0.004)	(0.010)	(0.004)	(0.003)	(0.115)	(0.009)
Full Smoking Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Sample Mean	0.166	0.179	0.397	0.111	0.073	0.071	0.148	0.035	0.451	0.076	0.034	28.092	0.304
N	12,042	12,037	12,033	12,042	12,035	12,043	12,046	12,045	12,040	12,044	12,043	11,937	11,937
R^2	0.108	0.053	0.111	0.106	0.088	0.108	0.093	0.092	0.111	0.074	0.065	0.159	0.117
Panel B: Ever Smoker Sample with Spousal Controls													
CPD Score	0.014**	0.004	0.012	0.023***	0.005	0.015***	0.005	-0.001	0.015	0.007*	0.000	0.293***	0.028***
	(0.006)	(0.005)	(0.010)	(0.006)	(0.005)	(0.005)	(0.007)	(0.003)	(0.010)	(0.004)	(0.002)	(0.102)	(0.009)
Initiation Score	0.006	0.003	-0.004	-0.008	-0.007	0.007	-0.005	0.002	0.007	-0.001	0.001	-0.001	0.006
	(0.006)	(0.005)	(0.010)	(0.006)	(0.005)	(0.006)	(0.007)	(0.004)	(0.010)	(0.004)	(0.002)	(0.113)	(0.009)
Spouse's CPD Score	0.021***	0.009*	0.009	0.011*	-0.003	0.004	-0.001	0.000	-0.004	0.010***	0.004**	0.254**	0.017**
	(0.006)	(0.005)	(0.009)	(0.006)	(0.005)	(0.005)	(0.007)	(0.003)	(0.009)	(0.004)	(0.002)	(0.099)	(0.008)
Spouse's Initiation Score	0.013**	0.011**	0.011	-0.005	-0.003	-0.008	0.003	0.002	0.007	0.002	-0.001	0.036	0.002
	(0.006)	(0.005)	(0.010)	(0.006)	(0.006)	(0.005)	(0.007)	(0.003)	(0.010)	(0.004)	(0.003)	(0.108)	(0.009)
Spouse's Full Smoking Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Spouse's Principal Components	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Full Smoking Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Sample Mean	0.166	0.179	0.397	0.111	0.073	0.071	0.148	0.035	0.451	0.076	0.034	28.092	0.304
N	12,042	12,037	12,033	12,042	12,035	12,043	12,046	12,045	12,040	12,044	12,043	11,937	11,937
R^2	0.137	0.077	0.161	0.143	0.140	0.149	0.133	0.135	0.159	0.098	0.097	0.209	0.160

Note: Each column within a panel presents results from a separate regression where the outcome is noted in the column heading. Results for the ever smoker sample without spousal controls are presented in Panel A and with spousal controls in Panel B. In all specifications, we include the standard controls and the genetic principal components described in Section 2.4. We also include the full set of smoking controls described in Section 2.4. In Panel B, we add the full set of the spouse's smoking controls and the spouse's smoking polygenic scores and their genetic principal components. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table B10: Relationship between the Smoking Polygenic Scores and Individual Health Measures Accounting for Spouse's Smoking Behavior and Smoking Polygenic Scores (Never Smoker Sample)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)
			Ever Diagnosed With:							Any Limitations With:			
	Poor Self-Reported Health	Any Hospitalization	High Blood Pressure	Diabetes	Cancer	Lung Disease	Heart Problem	Stroke	Arthritis	ADLs	IADLs	BMI	Obese
Panel A: Never Smoker Sample without Spousal Controls													
CPD Score	0.018***	0.008*	0.000	0.027***	0.000	0.003	-0.003	-0.001	0.013	0.008**	0.001	0.650***	0.038***
	(0.006)	(0.005)	(0.010)	(0.007)	(0.005)	(0.003)	(0.006)	(0.003)	(0.010)	(0.004)	(0.002)	(0.123)	(0.010)
Initiation Score	0.023***	0.009*	0.018	0.003	-0.006	0.004	-0.003	0.004	0.028**	0.012***	0.002	0.497***	0.035***
	(0.006)	(0.005)	(0.012)	(0.008)	(0.006)	(0.003)	(0.007)	(0.003)	(0.011)	(0.004)	(0.002)	(0.135)	(0.011)
Sample Mean	0.105	0.146	0.350	0.105	0.080	0.020	0.104	0.017	0.394	0.053	0.019	28.029	0.312
N	9,640	9,630	9,642	9,637	9,641	9,636	9,635	9,642	9,631	9,638	9,638	9,511	9,511
R^2	0.041	0.028	0.059	0.063	0.049	0.047	0.059	0.062	0.067	0.038	0.021	0.091	0.073
Panel B: Never Smoker Sample with Spousal Controls													
CPD Score	0.015***	0.007	0.001	0.025***	0.000	0.003	-0.004	-0.002	0.013	0.008**	0.000	0.618***	0.035***
	(0.005)	(0.005)	(0.010)	(0.007)	(0.005)	(0.003)	(0.006)	(0.003)	(0.011)	(0.004)	(0.002)	(0.124)	(0.010)
Initiation Score	0.018***	0.008	0.011	0.002	-0.005	0.002	-0.005	0.004	0.030***	0.011***	0.001	0.465***	0.036***
	(0.006)	(0.005)	(0.011)	(0.008)	(0.006)	(0.003)	(0.007)	(0.003)	(0.011)	(0.004)	(0.002)	(0.134)	(0.011)
Spouse's CPD Score	0.010*	0.011**	0.017*	0.006	0.004	0.007**	0.011	0.003	0.022**	0.004	0.001	0.091	0.010
	(0.006)	(0.005)	(0.010)	(0.007)	(0.006)	(0.003)	(0.007)	(0.003)	(0.011)	(0.003)	(0.002)	(0.120)	(0.010)
Spouse's Initiation Score	0.006	0.005	-0.009	-0.005	-0.003	-0.003	-0.001	-0.003	-0.004	-0.003	0.002	-0.086	-0.011
	(0.007)	(0.006)	(0.011)	(0.008)	(0.006)	(0.004)	(0.007)	(0.003)	(0.011)	(0.005)	(0.002)	(0.144)	(0.011)
Spouse's Full Smoking Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Spouse's Principal Components	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Sample Mean	0.105	0.146	0.350	0.105	0.080	0.020	0.104	0.017	0.394	0.053	0.019	28.029	0.312
N	9,640	9,630	9,642	9,637	9,641	9,636	9,635	9,642	9,631	9,638	9,638	9,511	9,511
R^2	0.093	0.049	0.125	0.115	0.112	0.106	0.110	0.109	0.123	0.073	0.050	0.154	0.130

Note: Each column within a panel presents results from a separate regression where the outcome is noted in the column heading. Results for the never smoker sample without spousal controls are presented in Panel A and with spousal controls in Panel B. In all specifications, we include the standard controls and the genetic principal components described in Section 2.4. In Panel B, we add the full set of the spouse's smoking controls and the spouse's smoking polygenic scores and their genetic principal components. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table B11: Relationship between the Smoking Polygenic Scores and Health Measures Accounting for Spouse's Smoking Polygenic Scores where Neither Member of Couple Ever Smoked

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)
			Ever Diagnosed With:							Any Limitations With:				
	Summary Index	Poor Self-Reported Health	Any Hospitalization	High Blood Pressure	Diabetes	Cancer	Lung Disease	Heart Problem	Stroke	Arthritis	ADLs	IADLs	BMI	Obese
CPD Score	0.019*	0.009	0.002	-0.016	0.023**	-0.006	0.007*	0.001	-0.002	0.013	0.005	0.002	0.572***	0.030**
	(0.010)	(0.007)	(0.006)	(0.014)	(0.009)	(0.007)	(0.004)	(0.009)	(0.003)	(0.014)	(0.005)	(0.003)	(0.169)	(0.013)
Initiation Score	0.009	0.007	-0.002	0.005	-0.009	0.007	0.004	-0.013	0.000	0.029*	0.004	-0.002	0.253	0.017
	(0.011)	(0.008)	(0.007)	(0.015)	(0.010)	(0.008)	(0.005)	(0.010)	(0.003)	(0.015)	(0.005)	(0.002)	(0.178)	(0.015)
Spouse's CPD Score	0.026**	0.011	0.016**	0.025*	0.009	-0.001	0.008*	0.023***	0.006	0.014	0.002	0.000	-0.059	0.003
	(0.011)	(0.007)	(0.007)	(0.014)	(0.009)	(0.008)	(0.004)	(0.009)	(0.005)	(0.014)	(0.004)	(0.003)	(0.162)	(0.013)
Spouse's Initiation Score	-0.007	0.005	0.006	-0.015	-0.018*	-0.001	0.000	-0.006	-0.011***	0.013	0.000	0.001	0.130	-0.001
	(0.012)	(0.009)	(0.008)	(0.015)	(0.010)	(0.008)	(0.003)	(0.009)	(0.004)	(0.015)	(0.006)	(0.003)	(0.199)	(0.015)
Spouse's Principal Components	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Standard Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Principal Components	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Sample Mean	-0.099	0.093	0.137	0.350	0.103	0.073	0.019	0.104	0.016	0.361	0.049	0.018	28.095	0.312
N	5,304	5,396	5,391	5,396	5,393	5,396	5,390	5,393	5,396	5,390	5,394	5,394	5,327	5,327
R^2	0.107	0.066	0.043	0.110	0.107	0.095	0.090	0.107	0.122	0.104	0.054	0.049	0.135	0.105

Note: Each column presents results from a separate regression where the outcome is noted in the column heading. The sample includes individuals in couples where both members were genotyped, aged 50–65, and neither member has ever smoked. In all specifications, we include the standard controls and the genetic principal components described in Section 2.4. We also include the spouse's smoking polygenic scores and their genetic principal components. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table B12: Relationship between the Smoking Polygenic Scores and the Summary Standardized Index of Health Accounting for Parental and Spousal Variables

	Full Sample				Ever Smoker Sample				Never Smoker Sample			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
CPD Score	0.030***	0.027***	0.028***	0.025***	0.030***	0.027***	0.030***	0.025***	0.029***	0.028***	0.025***	0.024***
	(0.006)	(0.006)	(0.006)	(0.006)	(0.008)	(0.008)	(0.008)	(0.008)	(0.008)	(0.008)	(0.008)	(0.008)
Initiation Score	0.015**	0.012**	0.009	0.007	0.005	0.006	-0.001	0.001	0.029***	0.022***	0.024***	0.018**
	(0.006)	(0.006)	(0.006)	(0.006)	(0.008)	(0.008)	(0.008)	(0.008)	(0.008)	(0.008)	(0.008)	(0.008)
Spouse's CPD Score			0.022***	0.021***			0.020***	0.018**			0.023***	0.022***
			(0.006)	(0.006)			(0.008)	(0.008)			(0.008)	(0.008)
Spouse's Initiation Score			0.001	-0.002			0.006	0.004			-0.005	-0.009
			(0.006)	(0.006)			(0.008)	(0.008)			(0.009)	(0.009)
Parental Controls	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes
Spouse's Full Smoking Controls	No	No	Yes	Yes	No	No	Yes	Yes	No	No	Yes	Yes
Spouse's Principal Components	No	No	Yes	Yes	No	No	Yes	Yes	No	No	Yes	Yes
Full Smoking Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	No	No	No	No
Standard Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Principal Components	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Sample Mean	-0.035	-0.035	-0.035	-0.035	0.004	0.004	0.004	0.004	-0.085	-0.085	-0.085	-0.085
N	21,357	21,357	21,357	21,357	11,888	11,888	11,888	11,888	9,469	9,469	9,469	9,469
R^2	0.129	0.144	0.159	0.174	0.172	0.187	0.212	0.227	0.088	0.108	0.150	0.168

Note: Each column presents results from a separate regression where the outcome is the summary standardized health index. Results are presented for the full sample and then separately for ever smokers and never smokers, and we only include individuals in couples where both members are aged 50–65 and genotyped. In all specifications, we include the standard controls and the genetic principal components described in Section 2.4, and for the full sample and ever smoker sample, we always include the full set of smoking controls described in Section 2.4. In columns (2), (6), and (10), we add controls for parental smoking, parental risky behaviors, and parental mortality as described in Section 2.3.4. In columns (3), (7), and (11), we include the full set of the spouse's smoking controls and the spouse's smoking polygenic scores and their genetic principal components. In columns (4), (8), and (12) we include both sets of parental and spousal variables. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table B13: Robustness of the Relationship between the Smoking Polygenic Scores and the Summary Standardized Index of Health

	Full Sample			Ever Smoker Sample			Never Smoker Sample	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Panel A: Using Older Polygenic Scores							
CPD Score	0.034***	0.029***	0.024***	0.036***	0.030***	0.023***	0.031***	0.024***
	(0.005)	(0.005)	(0.005)	(0.006)	(0.007)	(0.007)	(0.006)	(0.007)
Initiation Score	0.015***	0.019***	0.011**	0.003	0.007	0.002	0.020***	0.023***
	(0.004)	(0.005)	(0.005)	(0.006)	(0.007)	(0.006)	(0.006)	(0.007)
Full Smoking Controls	No	No	Yes	No	No	Yes	No	No
Standard Controls	No	Yes	Yes	No	Yes	Yes	No	Yes
Principal Components	No	Yes	Yes	No	Yes	Yes	No	Yes
Sample Mean	-0.000	-0.000	-0.000	0.038	0.038	0.038	-0.052	-0.052
N	45,850	45,850	45,850	26,509	26,509	26,509	19,341	19,341
R^2	0.007	0.059	0.111	0.006	0.073	0.138	0.008	0.074
	Panel B: Including Additional Smoking Polygenic Scores							
CPD Score	0.037***	0.038***	0.032***	0.043***	0.043***	0.034***	0.026***	0.028***
	(0.005)	(0.005)	(0.005)	(0.006)	(0.006)	(0.006)	(0.006)	(0.006)
Initiation Score	0.018***	0.028***	0.016***	0.011*	0.016**	0.007	0.014**	0.025***
	(0.005)	(0.005)	(0.005)	(0.007)	(0.007)	(0.007)	(0.006)	(0.007)
Cessation Score	0.017***	0.025***	0.022***	0.018***	0.022***	0.016**	0.019***	0.029***
	(0.005)	(0.005)	(0.005)	(0.006)	(0.007)	(0.007)	(0.006)	(0.007)
Initiation Age Score	-0.025***	-0.011**	-0.007	-0.022***	-0.011	-0.007	-0.025***	-0.006
	(0.005)	(0.005)	(0.005)	(0.006)	(0.007)	(0.007)	(0.007)	(0.007)
Full Smoking Controls	No	No	Yes	No	No	Yes	No	No
Standard Controls	No	Yes	Yes	No	Yes	Yes	No	Yes
Principal Components	No	Yes	Yes	No	Yes	Yes	No	Yes
Sample Mean	-0.000	-0.000	-0.000	0.038	0.038	0.038	-0.052	-0.052
N	45,850	45,850	45,850	26,509	26,509	26,509	19,341	19,341
R^2	0.017	0.072	0.118	0.015	0.083	0.143	0.015	0.083
	Panel C: Only Those Aged 50–61							
CPD Score	0.043***	0.043***	0.036***	0.049***	0.048***	0.038***	0.031***	0.032***
	(0.005)	(0.005)	(0.005)	(0.007)	(0.007)	(0.006)	(0.006)	(0.006)
Initiation Score	0.026***	0.034***	0.019***	0.020***	0.023***	0.010	0.020***	0.028***
	(0.005)	(0.005)	(0.005)	(0.007)	(0.007)	(0.007)	(0.006)	(0.007)
Full Smoking Controls	No	No	Yes	No	No	Yes	No	No
Standard Controls	No	Yes	Yes	No	Yes	Yes	No	Yes
Principal Components	No	Yes	Yes	No	Yes	Yes	No	Yes
Sample Mean	-0.029	-0.029	-0.029	0.010	0.010	0.010	-0.081	-0.081
N	32,413	32,413	32,413	18,667	18,667	18,667	13,746	13,746
R^2	0.014	0.062	0.111	0.014	0.078	0.140	0.010	0.069

Note: Each column within a panel presents results from a separate regression where the outcome is the summary standardized health index. Results are presented for the full sample and then separately for ever smokers and never smokers. In Panel A, we use the smoking polygenic scores from The Tobacco and Genetics Consortium (2010). In Panel B, we include additional smoking polygenic scores from Liu et al. (2019). In Panel C, we present results for those aged 50–61 (i.e., below the early Social Security retirement age). * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Appendix C

Genetic Endowments, Alzheimer's Disease, and Economic Outcomes with Nicholas Papageorge, Meghan Skira, and Kevin Thom

C.1 Tables

Table C1: Heterogeneity in the Relationship between Genetic Risk for AD and Cognition and Memory-Related Disease (MRD) Diagnosis by Age

	Langa-Weir (L-W) Score		Ever Diagnosed with Memory-Related Disease		
	(1)	(2)	(3)	(4)	(5)
AD Score	-0.261***	-0.240***	0.003**	0.002	0.002
	(0.057)	(0.057)	(0.002)	(0.002)	(0.002)
APOE (At least 1 copy)	-0.121	-0.133	-0.004	-0.004	-0.005
	(0.122)	(0.121)	(0.004)	(0.004)	(0.004)
APOE (2 copies)	0.423	0.443	-0.002	-0.001	0.000
	(0.291)	(0.289)	(0.007)	(0.007)	(0.007)
Ever demented (L-W $<$ 7)				0.120***	
				(0.013)	
Ever impaired or demented (L-W $<$ 12)				0.030***	
				(0.003)	
Ever diagnosed with MRD		-2.329***			
		(0.198)			
Eventually diagnosed with MRD		-1.648***			
		(0.107)			
AD score × Age 55-59	0.020	0.019	-0.001	-0.001	-0.001
	(0.051)	(0.051)	(0.001)	(0.001)	(0.001)
AD score × Age 60-64	0.044	0.043	0.000	0.000	0.000
	(0.056)	(0.056)	(0.002)	(0.002)	(0.002)
AD score × Age 65-69	0.051	0.047	-0.002	-0.002	-0.002
	(0.061)	(0.061)	(0.002)	(0.002)	(0.002)
AD score × Age 70-74	0.041	0.041	-0.002	-0.002	-0.002
	(0.066)	(0.065)	(0.002)	(0.002)	(0.002)
AD score × Age 75-79	-0.056	-0.046	0.001	-0.000	-0.000
	(0.070)	(0.069)	(0.002)	(0.002)	(0.002)
AD score × Age 80-85	0.022	0.038	0.003	0.002	0.003
	(0.075)	(0.073)	(0.003)	(0.003)	(0.003)
APOE (At least 1 copy) × Age 55-59	0.019	0.059	0.005	0.005	0.005
	(0.115)	(0.114)	(0.003)	(0.003)	(0.003)
APOE (At least 1 copy) × Age 60-64	0.035	0.102	0.004	0.004	0.004
	(0.128)	(0.127)	(0.004)	(0.004)	(0.004)
APOE (At least 1 copy) × Age 65-69	-0.090	0.043	0.010*	0.009*	0.009
	(0.142)	(0.140)	(0.005)	(0.005)	(0.005)
APOE (At least 1 copy) × Age 70-74	-0.325**	-0.133	0.017***	0.014**	0.013**
	(0.153)	(0.151)	(0.006)	(0.006)	(0.006)
APOE (At least 1 copy) × Age 75-79	-0.774***	-0.543***	0.028***	0.023***	0.020***

	(0.164)	(0.160)	(0.007)	(0.007)	(0.006)
APOE (At least 1 copy) × Age 80-85	-0.993***	-0.750***	0.038***	0.029***	0.023***
	(0.183)	(0.178)	(0.009)	(0.008)	(0.008)
APOE (2 copies) × Age 55-59	-0.506*	-0.411	0.019	0.018	0.017
	(0.306)	(0.309)	(0.014)	(0.014)	(0.014)
APOE (2 copies) × Age 60-64	-0.847**	-0.697**	0.012	0.009	0.008
	(0.348)	(0.346)	(0.013)	(0.014)	(0.014)
APOE (2 copies) × Age 65-69	-1.071***	-0.908**	0.012	0.010	0.007
	(0.378)	(0.383)	(0.016)	(0.016)	(0.016)
APOE (2 copies) × Age 70-74	-1.572***	-1.246***	0.054**	0.045*	0.042*
	(0.458)	(0.450)	(0.023)	(0.023)	(0.023)
APOE (2 copies) × Age 75-79	-1.576***	-1.350**	0.036	0.021	0.011
	(0.567)	(0.544)	(0.025)	(0.024)	(0.024)
APOE (2 copies) × Age 80-85	-2.117***	-1.927***	0.040	0.017	0.004
	(0.702)	(0.657)	(0.036)	(0.035)	(0.034)
L-W Score Dummies	No	No	No	No	Yes
Standard Controls	Yes	Yes	Yes	Yes	Yes
Principal Components	Yes	Yes	Yes	Yes	Yes
Years	1998-2018	1998-2018	1998-2018	1998-2018	1998-2018
Ages	50-85	50-85	50-85	50-85	50-85
Sample	All	All	All	All	All
Mean	16.393	16.393	0.021	0.021	0.021
N w/ APOE (At least 1 copy)	22,943	22,943	22,943	22,943	22,943
N w/ APOE (2 copies)	1,717	1,717	1,717	1,717	1,717
N	88,191	88,191	88,191	88,191	88,191
R^2	0.136	0.164	0.019	0.050	0.065

Note: Each column presents results from a separate regression. In columns (1) and (2), the outcome is the Langa-Weir (L-W) cognition score, which can take on values from 0–27. In columns (3)–(5), the outcome is an indicator for whether the individual has ever been diagnosed with a memory-related disease (MRD). In all specifications, we control for the first 10 principal components of the genetic data as well as the standard controls for age, birth cohort, sex of respondent, and survey year as described in Section 3.4. In column (2), we add controls for whether the respondent has ever been diagnosed with MRD and whether they will eventually report diagnosis (in subsequent survey waves). In column (4), we add controls for whether the respondent ever registered an L-W score in the impaired or demented ranges (below 12) and in the demented range (below 7). In column (5), we instead add dummy variables for each value of the most current L-W score. Standard errors are clustered at the individual level. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table C2: Heterogeneity in the Relationship between Genetic Risk for AD and Cognition and Memory-Related Disease (MRD) Diagnosis by Educational Attainment

	Langa-Weir (LW) Score			Ever Diagnosed with Memory-Related Disease		
	(1)	(2)	(3)	(4)	(5)	(6)
AD Score	-0.236***	-0.396***	-0.362**	0.003**	0.005	0.001
	(0.035)	(0.152)	(0.151)	(0.001)	(0.006)	(0.006)
APOE (At least 1 copy)	-0.403***	-0.948***	-0.790**	0.010***	0.025	0.010
	(0.065)	(0.348)	(0.339)	(0.003)	(0.017)	(0.017)
APOE (2 copies)	-0.560**	-1.348	-1.733*	0.020*	-0.081	-0.098
	(0.217)	(1.019)	(1.025)	(0.011)	(0.064)	(0.064)
Ever diagnosed with MRD			-2.184***			
			(0.192)			
Eventually diagnosed with MRD			-1.531***			
			(0.095)			
Years of education		0.521***	0.510***		-0.002***	0.001*
		(0.013)	(0.012)		(0.001)	(0.001)
AD score × Years of education		0.019*	0.018		-0.000	0.000
		(0.011)	(0.011)		(0.000)	(0.000)
APOE (At least 1 copy) × Years of education		0.041	0.038		-0.001	-0.000
		(0.025)	(0.025)		(0.001)	(0.001)
APOE (2 copies) × Years of education		0.058	0.099		0.007	0.008
		(0.073)	(0.072)		(0.005)	(0.005)
L-W Score Dummies	No	No	No	No	No	Yes
Standard Controls	Yes	Yes	Yes	Yes	Yes	Yes
Principal Components	Yes	Yes	Yes	Yes	Yes	Yes
Years	1998-2018	1998-2018	1998-2018	1998-2018	1998-2018	1998-2018
Ages	50-85	50-85	50-85	50-85	50-85	50-85
Sample	All	All	All	All	All	All
Mean	16.392	16.392	16.392	0.021	0.021	0.021
N w/ APOE (At least 1 copy)	22,899	22,899	22,899	22,899	22,899	22,899
N w/ APOE (2 copies)	1,717	1,717	1,717	1,717	1,717	1,717
N	88,045	88,045	88,045	88,045	88,045	88,045
R^2	0.133	0.237	0.262	0.017	0.019	0.065

Note: Each column presents results from a separate regression. In columns (1)–(3), the outcome is the Langa-Weir (L-W) cognition score, which can take on values from 0–27. In columns (4)–(6), the outcome is an indicator for whether the individual has ever been diagnosed with a memory-related disease (MRD). In all specifications, we control for the first 10 principal components of the genetic data as well as the standard controls for age, birth cohort, sex of respondent, and survey year as described in Section 3.4. In columns (2) and (5), we add controls for years of education and interactions between the AD genetic measures and years of education. Column (3) adds controls for whether the respondent has ever been diagnosed with MRD and whether they will eventually report diagnosis (in subsequent survey waves). Column (6) adds dummy variables for each value of the most current L-W score. Standard errors are clustered at the individual level. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table C3: Heterogeneity in the Relationship between Genetic Risk for AD and Cognition and Memory-Related Disease (MRD) Diagnosis by Childhood Socioeconomic Status

	Langa-Weir (L-W) Score			Ever Diagnosed with Memory-Related Disease		
	(1)	(2)	(3)	(4)	(5)	(6)
AD Score	-0.237***	-0.320***	-0.296***	0.003**	0.005*	0.003
	(0.035)	(0.062)	(0.061)	(0.001)	(0.003)	(0.003)
APOE (At least 1 copy)	-0.410***	-0.387***	-0.201	0.009***	0.022***	0.017***
	(0.065)	(0.135)	(0.130)	(0.003)	(0.007)	(0.006)
APOE (2 copies)	-0.540**	-0.808	-0.562	0.020*	0.032	0.020
	(0.219)	(0.496)	(0.502)	(0.011)	(0.026)	(0.026)
Ever diagnosed with MRD			-2.314***			
			(0.202)			
Eventually diagnosed with MRD			-1.646***			
			(0.108)			
High childhood SES		0.583***	0.563***		-0.003	-0.000
		(0.073)	(0.072)		(0.003)	(0.003)
AD score × High childhood SES		0.120*	0.117*		-0.003	-0.002
		(0.067)	(0.066)		(0.003)	(0.003)
APOE (At least 1 copy) × High childhood SES		-0.039	-0.127		-0.017**	-0.016**
		(0.154)	(0.149)		(0.007)	(0.007)
APOE (2 copies) × High childhood SES		0.366	0.264		-0.016	-0.008
		(0.550)	(0.552)		(0.028)	(0.029)
L-W Score Dummies	No	No	No	No	No	Yes
Standard Controls	Yes	Yes	Yes	Yes	Yes	Yes
Principal Components	Yes	Yes	Yes	Yes	Yes	Yes
Years	1998-2018	1998-2018	1998-2018	1998-2018	1998-2018	1998-2018
Ages	50-85	50-85	50-85	50-85	50-85	50-85
Sample	All	All	All	All	All	All
Mean	16.391	16.391	16.391	0.021	0.021	0.021
N w/ APOE (At least 1 copy)	22,622	22,622	22,622	22,622	22,622	22,622
N w/ APOE (2 copies)	1,701	1,701	1,701	1,701	1,701	1,701
N	86,962	86,962	86,962	86,962	86,962	86,962
R^2	0.134	0.138	0.166	0.018	0.019	0.064

Note: Each column presents results from a separate regression. In columns (1)–(3), the outcome is the Langa-Weir (L-W) cognition score, which can take on values from 0–27. In columns (4)–(6), the outcome is an indicator for whether the individual has ever been diagnosed with a memory-related disease (MRD). In all specifications, we control for the first 10 principal components of the genetic data as well as the standard controls for age, birth cohort, sex of respondent, and survey year as described in Section 3.4. In columns (2) and (5), we add an indicator for high childhood socioeconomic status (SES) and interactions between the AD genetic measures and high childhood SES. Column (3) adds controls for whether the respondent has ever been diagnosed with MRD and whether they will eventually report diagnosis (in subsequent survey waves). Column (6) adds dummy variables for each value of the most current L-W score. Standard errors are clustered at the individual level. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table C4: Heterogeneity in the Relationship between Genetic Risk for AD and Cognition and Memory-Related Disease (MRD) Diagnosis by Maternal Education

	Langa Weir (L-W) Score			Ever Diagnosed with Memory-Related Disease		
	(1)	(2)	(3)	(4)	(5)	(6)
AD Score	-0.216***	-0.208***	-0.181***	0.003**	0.003*	0.002
	(0.036)	(0.047)	(0.046)	(0.001)	(0.002)	(0.002)
APOE (At least 1 copy)	-0.362***	-0.381***	-0.227**	0.008***	0.012***	0.007*
	(0.066)	(0.099)	(0.096)	(0.003)	(0.004)	(0.004)
APOE (2 copies)	-0.519**	-0.898**	-0.750**	0.022**	0.015	0.002
	(0.223)	(0.355)	(0.349)	(0.011)	(0.015)	(0.015)
Ever diagnosed with MRD			-2.423***			
			(0.203)			
Eventually diagnosed with MRD			-1.601***			
			(0.108)			
Mother's education $\geq$ 12 years		1.029***	1.014***		-0.003	0.002
		(0.069)	(0.067)		(0.003)	(0.003)
AD score $\times$ Mother's education $\geq$ 12 years		0.036	0.022		-0.000	-0.000
		(0.057)	(0.055)		(0.002)	(0.002)
APOE (At least 1 copy) $\times$ Mother's education $\geq$ 12 years		0.028	-0.055		-0.007	-0.006
		(0.131)	(0.127)		(0.005)	(0.005)
APOE (2 copies) $\times$ Mother's education $\geq$ 12 years		0.613	0.665		0.013	0.024
		(0.447)	(0.439)		(0.022)	(0.022)
L-W Score Dummies	No	No	No	No	No	Yes
Standard Controls	Yes	Yes	Yes	Yes	Yes	Yes
Principal Components	Yes	Yes	Yes	Yes	Yes	Yes
Years	1998-2018	1998-2018	1998-2018	1998-2018	1998-2018	1998-2018
Ages	50-85	50-85	50-85	50-85	50-85	50-85
Sample	All	All	All	All	All	All
Mean	16.519	16.519	16.519	0.020	0.020	0.020
N w/ APOE (At least 1 copy)	21,437	21,437	21,437	21,437	21,437	21,437
N w/ APOE (2 copies)	1,623	1,623	1,623	1,623	1,623	1,623
N	82,538	82,538	82,538	82,538	82,538	82,538
R^2	0.132	0.147	0.175	0.017	0.017	0.064

Note: Each column presents results from a separate regression. In columns (1)–(3), the outcome is the Langa-Weir (L-W) cognition score, which can take on values from 0–27. In columns (4)–(6), the outcome is an indicator for whether the individual has ever been diagnosed with a memory-related disease (MRD). In all specifications, we control for the first 10 principal components of the genetic data as well as the standard controls for age, birth cohort, sex of respondent, and survey year as described in Section 3.4. In columns (2) and (5), we add an indicator for the respondent's mother completing at least 12 years of education and interactions between the AD genetic measures and maternal education. Column (3) adds controls for whether the respondent has ever been diagnosed with MRD and whether they will eventually report diagnosis (in subsequent survey waves). Column (6) adds dummy variables for each value of the most current L-W score. Standard errors are clustered at the individual level. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table C5: Heterogeneity in the Relationship between Genetic Risk for AD and Cognition and Memory-Related Disease (MRD) Diagnosis by Having Children Nearby

	Langa Weir (L-W) Score			Ever Diagnosed with Memory-Related Disease		
	(1)	(2)	(3)	(4)	(5)	(6)
AD Score	-0.214***	-0.222***	-0.197***	0.003**	0.003*	0.002
	(0.037)	(0.046)	(0.045)	(0.001)	(0.002)	(0.002)
APOE (At least 1 copy)	-0.389***	-0.397***	-0.280***	0.009***	0.007*	0.004
	(0.068)	(0.093)	(0.091)	(0.003)	(0.004)	(0.004)
APOE (2 copies)	-0.672***	-0.625**	-0.464	0.015	0.017	0.010
	(0.244)	(0.290)	(0.287)	(0.010)	(0.014)	(0.014)
Ever diagnosed with MRD			-2.254***			
			(0.221)			
Eventually diagnosed with MRD			-1.633***			
			(0.111)			
Kids within 10 miles		-0.524***	-0.521***		0.001	-0.001
		(0.057)	(0.056)		(0.002)	(0.002)
AD score × Kids within 10 miles		0.025	0.023		-0.000	-0.001
		(0.050)	(0.049)		(0.002)	(0.002)
APOE (At least 1 copy) × Kids within 10 miles		0.027	0.060		0.005	0.004
		(0.116)	(0.113)		(0.005)	(0.005)
APOE (2 copies) × Kids within 10 miles		-0.137	-0.137		-0.003	-0.006
		(0.372)	(0.355)		(0.017)	(0.016)
L-W Score Dummies	No	No	No	No	No	Yes
Standard Controls	Yes	Yes	Yes	Yes	Yes	Yes
Principal Components	Yes	Yes	Yes	Yes	Yes	Yes
Years	1998-2014	1998-2014	1998-2014	1998-2014	1998-2014	1998-2014
Ages	50-85	50-85	50-85	50-85	50-85	50-85
Sample	All	All	All	All	All	All
Mean	16.356	16.356	16.356	0.020	0.020	0.020
N w/ APOE (At least 1 copy)	17,199	17,199	17,199	17,199	17,199	17,199
N w/ APOE (2 copies)	1,265	1,265	1,265	1,265	1,265	1,265
N	65,612	65,612	65,612	65,612	65,612	65,612
R^2	0.133	0.138	0.167	0.019	0.020	0.065

Note: Each column presents results from a separate regression where the sample only includes those with living children. In columns (1)–(3), the outcome is the Langa-Weir (L-W) cognition score, which can take on values from 0–27. In columns (4)–(6), the outcome is an indicator for whether the individual has ever been diagnosed with a memory-related disease (MRD). In all specifications, we control for the first 10 principal components of the genetic data as well as the standard controls for age, birth cohort, sex of respondent, and survey year as described in Section 3.4. In columns (2) and (5), we add an indicator for whether any of the respondent's children live within 10 miles and interactions between the AD genetic measures and having children nearby. Column (3) adds controls for whether the respondent has ever been diagnosed with MRD and whether they will eventually report diagnosis (in subsequent survey waves). Column (6) adds dummy variables for each value of the most current L-W score. Standard errors are clustered at the individual level. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table C6: Heterogeneity in the Relationship between Genetic Risk for AD and Cognition and Memory-Related Disease (MRD) Diagnosis by Parental History of MRD

	Langa Weir (L-W) Score			Ever Diagnosed with Memory-Related Disease		
	(1)	(2)	(3)	(4)	(5)	(6)
AD Score	-0.207***	-0.220***	-0.204***	0.001	0.002	0.001
	(0.054)	(0.060)	(0.059)	(0.002)	(0.002)	(0.002)
APOE (At least 1 copy)	-0.242**	-0.146	-0.079	0.007	0.006	0.004
	(0.099)	(0.120)	(0.119)	(0.005)	(0.005)	(0.005)
APOE (2 copies)	0.093	0.065	0.293	0.017	0.041	0.040
	(0.293)	(0.367)	(0.355)	(0.018)	(0.028)	(0.028)
Ever diagnosed with MRD			-1.624***			
			(0.364)			
Eventually diagnosed with MRD			-1.794***			
			(0.229)			
Parent ever diagnosed with MRD		0.043	0.059		0.008	0.007
		(0.103)	(0.101)		(0.005)	(0.005)
AD score × Parent ever diagnosed with MRD		0.039	0.022		-0.004	-0.003
		(0.083)	(0.081)		(0.004)	(0.003)
APOE (At least 1 copy) × Parent ever diagnosed with MRD		-0.276	-0.247		0.000	0.001
		(0.194)	(0.191)		(0.009)	(0.009)
APOE (2 copies) × Parent ever diagnosed with MRD		0.080	-0.316		-0.067**	-0.065**
		(0.570)	(0.553)		(0.029)	(0.029)
L-W Score Dummies	No	No	No	No	No	Yes
Standard Controls	Yes	Yes	Yes	Yes	Yes	Yes
Principal Components	Yes	Yes	Yes	Yes	Yes	Yes
Years	1998-2018	1998-2018	1998-2018	1998-2018	1998-2018	1998-2018
Ages	50-85	50-85	50-85	50-85	50-85	50-85
Sample	All	All	All	All	All	All
Mean	17.094	17.094	17.094	0.020	0.020	0.020
N w/ APOE (At least 1 copy)	9,369	9,369	9,369	9,369	9,369	9,369
N w/ APOE (2 copies)	774	774	774	774	774	774
N	37,766	37,766	37,766	37,766	37,766	37,766
R^2	0.097	0.097	0.118	0.024	0.026	0.058

Note: Each column presents results from a separate regression where the sample includes only those who had at least one parent alive in 1998 or after. In columns (1)–(3), the outcome is the Langa-Weir (L-W) cognition score, which can take on values from 0–27. In columns (4)–(6), the outcome is an indicator for whether the individual has ever been diagnosed with a memory-related disease (MRD). In all specifications, we control for the first 10 principal components of the genetic data as well as the standard controls for age, birth cohort, sex of respondent, and survey year as described in Section 3.4. In columns (2) and (5), we add an indicator for whether the respondent's parent(s) were ever diagnosed with MRD and interactions between the AD genetic measures and parental history of MRD. Column (3) adds controls for whether the respondent has ever been diagnosed with MRD and whether they will eventually report diagnosis (in subsequent survey waves). Column (6) adds dummy variables for each value of the most current L-W score. Standard errors are clustered at the individual level. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table C7: Relationship between Genetic Risk for AD and Expected Mortality Controlling for Education

	Probability of Living to Age 75			
	(1)	(2)	(3)	(4)
AD Score	-0.090	0.181	-0.027	0.190
	(0.310)	(0.304)	(0.310)	(0.305)
APOE (At least 1 copy)	-0.675	-0.696	-0.636	-0.683
	(0.571)	(0.555)	(0.568)	(0.554)
APOE (2 copies)	-0.731	-0.458	-0.689	-0.450
	(1.887)	(1.877)	(1.896)	(1.880)
EA Score			1.763***	0.497*
			(0.250)	(0.257)
Ever diagnosed with MRD	Yes	Yes	Yes	Yes
L-W Score Dummies	Yes	Yes	Yes	Yes
Educational Attainment Controls	No	Yes	No	Yes
Standard Controls	Yes	Yes	Yes	Yes
Principal Components	Yes	Yes	Yes	Yes
Years	1998-2018	1998-2018	1998-2018	1998-2018
Ages	50-65	50-65	50-65	50-65
Sample	All	All	All	All
Mean	66.280	66.280	66.280	66.280
N w/ APOE (at least 1 copy)	9,583	9,583	9,583	9,583
N w/ APOE (2 copies)	844	844	844	844
N	36,291	36,291	36,291	36,291
R^2	0.054	0.089	0.058	0.089

Note: Each column presents results from a separate regression where the outcome is the self-reported probability of living to age 75 (on a 0–100 scale). The question is only asked to those aged 65 and younger. In all specifications, we control for the first 10 principal components of the genetic data, the standard controls for age, birth cohort, sex of respondent, and survey year as described in Section 3.4, and Langa-Weir score dummies and MRD diagnosis. In column (2), we include a complete set of dummy variables for each year of schooling and dummies for the highest-completed degree as well as interactions with the male dummy. In column (3), we control the EA score. In column (4), we control for educational attainment and the EA score. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table C8: Relationship between Genetic Risk for AD and Expected Nursing Home Use Controlling for Education

	Probability of Moving to Nursing Home in Next 5 Years			
	(1)	(2)	(3)	(4)
AD Score	-0.181	-0.155	-0.167	-0.148
	(0.189)	(0.190)	(0.189)	(0.190)
APOE (At least 1 copy)	0.757**	0.730**	0.750**	0.728**
	(0.357)	(0.357)	(0.357)	(0.357)
APOE (2 copies)	-0.620	-0.776	-0.589	-0.745
	(1.131)	(1.121)	(1.126)	(1.119)
EA Score			0.399**	0.287*
			(0.155)	(0.162)
Ever diagnosed with MRD	Yes	Yes	Yes	Yes
L-W Score Dummies	Yes	Yes	Yes	Yes
Educational Attainment Controls	No	Yes	No	Yes
Standard Controls	Yes	Yes	Yes	Yes
Principal Components	Yes	Yes	Yes	Yes
Years	1998-2018	1998-2018	1998-2018	1998-2018
Ages	65-85	65-85	65-85	65-85
Sample	All	All	All	All
Mean	14.448	14.448	14.448	14.448
N w/ APOE (at least 1 copy)	12,611	12,611	12,611	12,611
N w/ APOE (2 copies)	823	823	823	823
N	49,328	49,328	49,328	49,328
R^2	0.043	0.046	0.044	0.046

Note: Each column presents results from a separate regression where the outcome is the self-reported probability of moving to a nursing home in the next 5 years (on a 0–100 scale). The question is only asked to those aged 65 and older who do not currently reside in a nursing home. In all specifications, we control for the first 10 principal components of the genetic data, the standard controls for age, birth cohort, sex of respondent, and survey year as described in Section 3.4, and Langa-Weir score dummies and MRD diagnosis. In column (2), we include a complete set of dummy variables for each year of schooling and dummies for the highest-completed degree as well as interactions with the male dummy. In column (3), we control the EA score. In column (4), we control for educational attainment and the EA score. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table C9: Relationship between Genetic Risk for AD and Currently Working for Pay Controlling for Education

	Currently Working for Pay			
	(1)	(2)	(3)	(4)
AD Score	-0.013***	-0.009**	-0.012**	-0.009*
	(0.005)	(0.005)	(0.005)	(0.005)
APOE (At least 1 copy)	0.008	0.006	0.008	0.007
	(0.009)	(0.009)	(0.009)	(0.009)
APOE (2 copies)	-0.001	-0.001	0.001	0.000
	(0.025)	(0.025)	(0.025)	(0.025)
EA Score			0.028***	0.015***
			(0.004)	(0.004)
Ever diagnosed with MRD	Yes	Yes	Yes	Yes
L-W Score Dummies	Yes	Yes	Yes	Yes
Educational Attainment Controls	No	Yes	No	Yes
Standard Controls	Yes	Yes	Yes	Yes
Principal Components	Yes	Yes	Yes	Yes
Years	1998-2018	1998-2018	1998-2018	1998-2018
Ages	50-70	50-70	50-70	50-70
Sample	All	All	All	All
Mean	0.563	0.563	0.563	0.563
N w/ APOE (At least 1 copy)	14,159	14,159	14,159	14,159
N w/ APOE (2 copies)	1,180	1,180	1,180	1,180
N	53,598	53,598	53,598	53,598
R^2	0.176	0.190	0.179	0.191

Note: Each column presents results from a separate regression where the outcome is an indicator for whether the respondent currently works for pay. In all specifications, we control for the first 10 principal components of the genetic data, the standard controls for age, birth cohort, sex of respondent, and survey year as described in Section 3.4, and Langa-Weir score dummies and MRD diagnosis. In column (2), we include a complete set of dummy variables for each year of schooling and dummies for the highest-completed degree as well as interactions with the male dummy. In column (3), we control the EA score. In column (4), we control for educational attainment and the EA score. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table C10: Relationship between Genetic Risk for AD and Retirement Controlling for Education

	Whether Retired			
	(1)	(2)	(3)	(4)
AD Score	0.009**	0.007	0.008*	0.007
	(0.004)	(0.004)	(0.004)	(0.004)
APOE (At least 1 copy)	-0.009	-0.007	-0.009	-0.007
	(0.008)	(0.008)	(0.008)	(0.008)
APOE (2 copies)	0.031	0.028	0.030	0.027
	(0.023)	(0.024)	(0.024)	(0.024)
EA Score			-0.021***	-0.014***
			(0.003)	(0.004)
Ever diagnosed with MRD	Yes	Yes	Yes	Yes
L-W Score Dummies	Yes	Yes	Yes	Yes
Educational Attainment Controls	No	Yes	No	Yes
Standard Controls	Yes	Yes	Yes	Yes
Principal Components	Yes	Yes	Yes	Yes
Years	1998-2018	1998-2018	1998-2018	1998-2018
Ages	50-70	50-70	50-70	50-70
Sample	All	All	All	All
Mean	0.320	0.320	0.320	0.320
N w/ APOE (At least 1 copy)	13,019	13,019	13,019	13,019
N w/ APOE (2 copies)	1,110	1,110	1,110	1,110
N	49,570	49,570	49,570	49,570
R^2	0.219	0.227	0.221	0.227

Note: Each column presents results from a separate regression where the outcome is an indicator for whether the respondent is retired, defined as currently not working for pay and self-reporting oneself as completely retired. In all specifications, we control for the first 10 principal components of the genetic data, the standard controls for age, birth cohort, sex of respondent, and survey year as described in Section 3.4, and Langa-Weir score dummies and MRD diagnosis. In column (2), we include a complete set of dummy variables for each year of schooling and dummies for the highest-completed degree as well as interactions with the male dummy. In column (3), we control the EA score. In column (4), we control for educational attainment and the EA score. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table C11: Relationship between Genetic Risk for AD and Log Individual Total Income Controlling for Education

	Log Total Individual Income			
	(1)	(2)	(3)	(4)
AD Score	-0.028***	-0.014*	-0.025***	-0.014*
	(0.008)	(0.008)	(0.008)	(0.008)
APOE (At least 1 copy)	0.005	-0.004	0.004	-0.004
	(0.015)	(0.014)	(0.015)	(0.014)
APOE (2 copies)	0.018	-0.002	0.022	-0.001
	(0.045)	(0.041)	(0.044)	(0.041)
EA Score			0.068***	0.010
			(0.006)	(0.006)
Ever diagnosed with MRD	Yes	Yes	Yes	Yes
L-W Score Dummies	Yes	Yes	Yes	Yes
Educational Attainment Controls	No	Yes	No	Yes
Standard Controls	Yes	Yes	Yes	Yes
Principal Components	Yes	Yes	Yes	Yes
Years	1998-2018	1998-2018	1998-2018	1998-2018
Ages	50-85	50-85	50-85	50-85
Sample	All	All	All	All
Mean	10.007	10.007	10.007	10.007
N w/ APOE (at least 1 copy)	21,101	21,101	21,101	21,101
N w/ APOE (2 copies)	1,541	1,541	1,541	1,541
N	81,392	81,392	81,392	81,392
R^2	0.200	0.254	0.204	0.254

Note: Each column presents results from a separate regression where the outcome is logged total individual income. In all specifications, we control for the first 10 principal components of the genetic data, the standard controls for age, birth cohort, sex of respondent, and survey year as described in Section 3.4, and Langa-Weir score dummies and MRD diagnosis. In column (2), we include a complete set of dummy variables for each year of schooling and dummies for the highest-completed degree as well as interactions with the male dummy. In column (3), we control the EA score. In column (4), we control for educational attainment and the EA score. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table C12: Relationship between Genetic Risk for AD and Holding Long-Term Care Insurance Controlling for Education

	Holds Long-Term Care Insurance			
	(1)	(2)	(3)	(4)
AD Score	-0.012***	-0.009***	-0.011***	-0.009***
	(0.004)	(0.004)	(0.004)	(0.004)
APOE (At least 1 copy)	0.011*	0.010	0.011	0.010
	(0.007)	(0.007)	(0.007)	(0.007)
APOE (2 copies)	-0.001	-0.004	-0.000	-0.004
	(0.021)	(0.020)	(0.021)	(0.020)
EA Score			0.014***	0.001
			(0.003)	(0.003)
Ever diagnosed with MRD	Yes	Yes	Yes	Yes
L-W Score Dummies	Yes	Yes	Yes	Yes
Educational Attainment Controls	No	Yes	No	Yes
Standard Controls	Yes	Yes	Yes	Yes
Principal Components	Yes	Yes	Yes	Yes
Years	1998-2018	1998-2018	1998-2018	1998-2018
Ages	50-85	50-85	50-85	50-85
Sample	All	All	All	All
Mean	0.149	0.149	0.149	0.149
N w/ APOE (at least 1 copy)	22,488	22,488	22,488	22,488
N w/ APOE (2 copies)	1,685	1,685	1,685	1,685
N	86,720	86,720	86,720	86,720
R^2	0.029	0.053	0.031	0.053

Note: Each column presents results from a separate regression where the outcome is an indicator for whether the respondent holds long-term care insurance. In all specifications, we control for the first 10 principal components of the genetic data, the standard controls for age, birth cohort, sex of respondent, and survey year as described in Section 3.4, and Langa-Weir score dummies and MRD diagnosis. In column (2), we include a complete set of dummy variables for each year of schooling and dummies for the highest-completed degree as well as interactions with the male dummy. In column (3), we control the EA score. In column (4), we control for educational attainment and the EA score. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table C13: Relationship between Genetic Risk for AD and Holding Life Insurance Controlling for Education

	Holds Life Insurance			
	(1)	(2)	(3)	(4)
AD Score	-0.006	-0.006	-0.006	-0.006
	(0.004)	(0.004)	(0.004)	(0.004)
APOE (At least 1 copy)	-0.001	-0.002	-0.001	-0.002
	(0.008)	(0.008)	(0.008)	(0.008)
APOE (2 copies)	-0.005	-0.004	-0.004	-0.004
	(0.025)	(0.024)	(0.025)	(0.024)
EA Score			0.005	0.004
			(0.004)	(0.004)
Ever diagnosed with MRD	Yes	Yes	Yes	Yes
L-W Score Dummies	Yes	Yes	Yes	Yes
Educational Attainment Controls	No	Yes	No	Yes
Standard Controls	Yes	Yes	Yes	Yes
Principal Components	Yes	Yes	Yes	Yes
Years	1998-2018	1998-2018	1998-2018	1998-2018
Ages	50-85	50-85	50-85	50-85
Sample	All	All	All	All
Mean	0.677	0.677	0.677	0.677
N w/ APOE (at least 1 copy)	22,697	22,697	22,697	22,697
N w/ APOE (2 copies)	1,700	1,700	1,700	1,700
N	87,430	87,430	87,430	87,430
R^2	0.053	0.057	0.053	0.057

Note: Each column presents results from a separate regression where the outcome is an indicator for whether the respondent holds life insurance. In all specifications, we control for the first 10 principal components of the genetic data, the standard controls for age, birth cohort, sex of respondent, and survey year as described in Section 3.4, and Langa-Weir score dummies and MRD diagnosis. In column (2), we include a complete set of dummy variables for each year of schooling and dummies for the highest-completed degree as well as interactions with the male dummy. In column (3), we control the EA score. In column (4), we control for educational attainment and the EA score. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table C14: Relationship between Genetic Risk for AD and Having a Witnessed Will Controlling for Education

	Has a Witnessed Will			
	(1)	(2)	(3)	(4)
AD Score	-0.017***	-0.012**	-0.015***	-0.011**
	(0.005)	(0.005)	(0.005)	(0.005)
APOE (At least 1 copy)	0.011	0.007	0.011	0.007
	(0.009)	(0.009)	(0.009)	(0.008)
APOE (2 copies)	0.016	0.013	0.018	0.014
	(0.027)	(0.026)	(0.027)	(0.026)
EA Score			0.038***	0.015***
			(0.004)	(0.004)
Ever diagnosed with MRD	Yes	Yes	Yes	Yes
L-W Score Dummies	Yes	Yes	Yes	Yes
Educational Attainment Controls	No	Yes	No	Yes
Standard Controls	Yes	Yes	Yes	Yes
Principal Components	Yes	Yes	Yes	Yes
Years	1998-2018	1998-2018	1998-2018	1998-2018
Ages	50-85	50-85	50-85	50-85
Sample	All	All	All	All
Mean	0.641	0.641	0.641	0.641
N w/ APOE (at least 1 copy)	22,798	22,798	22,798	22,798
N w/ APOE (2 copies)	1,708	1,708	1,708	1,708
N	87,747	87,747	87,747	87,747
R^2	0.111	0.149	0.117	0.150

Note: Each column presents results from a separate regression where the outcome is an indicator for whether the respondent has a witnessed will. In all specifications, we control for the first 10 principal components of the genetic data, the standard controls for age, birth cohort, sex of respondent, and survey year as described in Section 3.4, and Langa-Weir score dummies and MRD diagnosis. In column (2), we include a complete set of dummy variables for each year of schooling and dummies for the highest-completed degree as well as interactions with the male dummy. In column (3), we control the EA score. In column (4), we control for educational attainment and the EA score. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table C15: Relationship between Genetic Risk for AD and Having a Living Will Controlling for Education

	Has a Living Will			
	(1)	(2)	(3)	(4)
AD Score	-0.004	0.001	-0.003	0.001
	(0.007)	(0.007)	(0.007)	(0.007)
APOE (At least 1 copy)	0.000	-0.006	-0.001	-0.006
	(0.013)	(0.013)	(0.013)	(0.013)
APOE (2 copies)	0.033	0.022	0.038	0.024
	(0.042)	(0.039)	(0.041)	(0.039)
EA Score			0.032***	0.011*
			(0.006)	(0.006)
Ever diagnosed with MRD	Yes	Yes	Yes	Yes
L-W Score Dummies	Yes	Yes	Yes	Yes
Educational Attainment Controls	No	Yes	No	Yes
Standard Controls	Yes	Yes	Yes	Yes
Principal Components	Yes	Yes	Yes	Yes
Years	2012-2018	2012-2018	2012-2018	2012-2018
Ages	65-85	65-85	65-85	65-85
Sample	All	All	All	All
Mean	0.598	0.598	0.598	0.598
N w/ APOE (at least 1 copy)	4,839	4,839	4,839	4,839
N w/ APOE (2 copies)	339	339	339	339
N	18,882	18,882	18,882	18,882
R^2	0.066	0.097	0.070	0.097

Note: Each column presents results from a separate regression where the outcome is an indicator for whether the respondent has a living will. In all specifications, we control for the first 10 principal components of the genetic data, the standard controls for age, birth cohort, sex of respondent, and survey year as described in Section 3.4, and Langa-Weir score dummies and MRD diagnosis. In column (2), we include a complete set of dummy variables for each year of schooling and dummies for the highest-completed degree as well as interactions with the male dummy. In column (3), we control the EA score. In column (4), we control for educational attainment and the EA score. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table C16: Relationship between Genetic Risk for AD and Having Assigned Someone Durable Power of Attorney Controlling for Education

	Has Assigned Someone Durable Power of Attorney for Healthcare			
	(1)	(2)	(3)	(4)
AD Score	-0.016**	-0.012*	-0.015**	-0.012*
	(0.007)	(0.007)	(0.007)	(0.007)
APOE (At least 1 copy)	0.008	0.004	0.007	0.003
	(0.013)	(0.013)	(0.013)	(0.013)
APOE (2 copies)	0.005	-0.009	0.009	-0.007
	(0.043)	(0.041)	(0.043)	(0.041)
EA Score			0.030***	0.010*
			(0.006)	(0.006)
Ever diagnosed with MRD	Yes	Yes	Yes	Yes
L-W Score Dummies	Yes	Yes	Yes	Yes
Educational Attainment Controls	No	Yes	No	Yes
Standard Controls	Yes	Yes	Yes	Yes
Principal Components	Yes	Yes	Yes	Yes
Years	2012-2018	2012-2018	2012-2018	2012-2018
Ages	65-85	65-85	65-85	65-85
Sample	All	All	All	All
Mean	0.596	0.596	0.596	0.596
N w/ APOE (at least 1 copy)	4,860	4,860	4,860	4,860
N w/ APOE (2 copies)	342	342	342	342
N	18,922	18,922	18,922	18,922
R^2	0.072	0.099	0.076	0.100

Note: Each column presents results from a separate regression where the outcome is an indicator for whether the respondent has assigned someone durable power of attorney. In all specifications, we control for the first 10 principal components of the genetic data, the standard controls for age, birth cohort, sex of respondent, and survey year as described in Section 3.4, and Langa-Weir score dummies and MRD diagnosis. In column (2), we include a complete set of dummy variables for each year of schooling and dummies for the highest-completed degree as well as interactions with the male dummy. In column (3), we control the EA score. In column (4), we control for educational attainment and the EA score. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table C17: Relationship between Genetic Risk for AD and Having Discussed Future Medical Care with Someone Controlling for Education

	Discussed Future Medical Care with Anyone			
	(1)	(2)	(3)	(4)
AD Score	-0.013**	-0.009	-0.012*	-0.009
	(0.006)	(0.006)	(0.006)	(0.006)
APOE (At least 1 copy)	-0.013	-0.016	-0.012	-0.016
	(0.012)	(0.012)	(0.012)	(0.012)
APOE (2 copies)	-0.015	-0.025	-0.011	-0.024
	(0.039)	(0.037)	(0.039)	(0.037)
EA Score			0.021***	0.006
			(0.005)	(0.005)
Ever diagnosed with MRD	Yes	Yes	Yes	Yes
L-W Score Dummies	Yes	Yes	Yes	Yes
Educational Attainment Controls	No	Yes	No	Yes
Standard Controls	Yes	Yes	Yes	Yes
Principal Components	Yes	Yes	Yes	Yes
Years	2012-2018	2012-2018	2012-2018	2012-2018
Ages	65-85	65-85	65-85	65-85
Sample	All	All	All	All
Mean	0.553	0.553	0.553	0.553
N w/ APOE (at least 1 copy)	2,599	2,599	2,599	2,599
N w/ APOE (2 copies)	179	179	179	179
N	10,046	10,046	10,046	10,046
R^2	0.118	0.137	0.119	0.137

Note: Each column presents results from a separate regression where the outcome is an indicator for whether the respondent has discussed future medical care with someone. In all specifications, we control for the first 10 principal components of the genetic data, the standard controls for age, birth cohort, sex of respondent, and survey year as described in Section 3.4, and Langa-Weir score dummies and MRD diagnosis. In column (2), we include a complete set of dummy variables for each year of schooling and dummies for the highest-completed degree as well as interactions with the male dummy. In column (3), we control the EA score. In column (4), we control for educational attainment and the EA score. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table C18: Relationship between Genetic Risk for AD and Years of Education

	Years of Education			
	(1)	(2)	(3)	(4)
AD Score	-0.139***	-0.109***	-0.126***	-0.062**
	(0.029)	(0.028)	(0.028)	(0.027)
APOE (At least 1 copy)	-0.001	0.002	-0.012	0.020
	(0.054)	(0.052)	(0.053)	(0.049)
APOE (2 copies)	-0.037	-0.019	-0.036	-0.132
	(0.165)	(0.159)	(0.158)	(0.144)
EA Score		0.661***		0.531***
		(0.023)		(0.022)
EA Score (Old)			0.575***	
			(0.023)	
Childhood SES	No	No	No	Yes
Mother's Years of Education	No	No	No	Yes
Standard Controls	Yes	Yes	Yes	Yes
Principal Components	Yes	Yes	Yes	Yes
Years	1994-2018	1994-2018	1994-2018	1994-2018
Ages	50-85	50-85	50-85	50-85
Sample	All	All	All	All
Mean	13.388	13.388	13.388	13.388
N w/ APOE (At least 1 copy)	2,835	2,835	2,835	2,835
N w/ APOE (2 copies)	221	221	221	221
N	10,985	10,985	10,985	10,985
R^2	0.090	0.155	0.139	0.259

Note: Each column presents results from a separate regression where the outcome is completed years of education. In all specifications, we control for the first 10 principal components of the genetic data, the standard controls for age, birth cohort, sex of respondent, and survey year as described in Section 3.4, and Langa-Weir score dummies and MRD diagnosis. In column (2), we add the EA score from Lee et al. (2018). In column (3), we instead add the EA score from Okbay et al. (2016). In column (4), we control for the EA score from Lee et al. (2018) as well dummies for childhood SES and completed years of maternal education. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table C19: Relationship between Genetic Risk for AD and At Least Some College Education

	At Least Some College			
	(1)	(2)	(3)	(4)
AD Score	-0.019***	-0.014***	-0.017***	-0.008
	(0.006)	(0.005)	(0.006)	(0.005)
APOE (At least 1 copy)	-0.000	0.000	-0.002	0.003
	(0.010)	(0.010)	(0.010)	(0.010)
APOE (2 copies)	-0.003	0.000	-0.002	-0.020
	(0.033)	(0.032)	(0.032)	(0.030)
EA Score		0.107***		0.087***
		(0.004)		(0.004)
EA Score (Old)			0.090***	
			(0.004)	
Childhood SES	No	No	No	Yes
Mother's Years of Education	No	No	No	Yes
Standard Controls	Yes	Yes	Yes	Yes
Principal Components	Yes	Yes	Yes	Yes
Years	1994-2018	1994-2018	1994-2018	1994-2018
Ages	50-85	50-85	50-85	50-85
Sample	All	All	All	All
Mean	0.334	0.334	0.334	0.334
N w/ APOE (At least 1 copy)	2,835	2,835	2,835	2,835
N w/ APOE (2 copies)	221	221	221	221
N	10,985	10,985	10,985	10,985
R^2	0.072	0.118	0.104	0.189

Note: Each column presents results from a separate regression where the outcome is a dummy variable for whether the respondent has at least some college education. In all specifications, we control for the first 10 principal components of the genetic data, the standard controls for age, birth cohort, sex of respondent, and survey year as described in Section 3.4, and Langa-Weir score dummies and MRD diagnosis. In column (2), we add the EA score from Lee et al. (2018). In column (3), we instead add the EA score from Okbay et al. (2016). In column (4), we control for the EA score from Lee et al. (2018) as well dummies for childhood SES and completed years of maternal education. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.