Genetic Endowments, Alzheimer's Disease, and Economic Outcomes *

Yeongmi Jeong
University of Georgia

Nicholas W. Papageorge
Johns Hopkins University

Meghan M. Skira
University of Georgia

Kevin Thom
University of Wisconsin - Milwaukee

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Abstract

Genetic factors contribute substantially to individual-level variation in the risk of developing Alzheimer's disease. The increasing availability of genetic data and the accumulation of results from genome-wide association studies now make it possible to study the evolution of outcomes and behaviors for individuals with different observed genetic markers. Using data from the Health and Retirement Study (HRS), we find that individuals at greater genetic risk for AD are observed to have lower levels of cognition, even after controlling for the self-reported diagnosis of a memory-related illness. Higher genetic risk is also associated with lower propensities to work and lower incomes. However, we also find that individuals at the greatest genetic risk do not have systematically different expectations about their own mortality and only a particular subset of high-risk individuals anticipate need for nursing home care. Furthermore, these individuals are no more likely (and in many specifications less likely) to engage in planning behaviors like the acquisition of long-term care insurance or the maintenance of a will. We also fail to find evidence of robust interactions between genetic risk and various measures of socioeconomic status (including completed education and childhood conditions) in predicting later-life outcomes. This combination of results suggests that efforts to encourage end-of-life planning might be particularly beneficial for those at greater risk for needing such services.

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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 are projected to reach $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. forthcoming). 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 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 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

[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.

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. 2019, Gresenz et al. 2020, Mazzonna and Peracchi 2020, Nicholas et al. 2021, Li et al. 2022). Chandra et al. (forthcoming) 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).

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:

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

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]

[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).

While several GWAS have implicated various genetic markers associated with late-onset AD, being a carrier of the APOE-ε4 (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 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 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.

[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.

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).

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.

[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 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.

3.4 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.

3.5 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.

3.6 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-ε4, and the second takes value one if the individual has exactly two copies of the allele.[7]

3.7 Descriptive Statistics

In Table 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 2. About 26 percent of the person-waves in our sample have at least one copy of the APOE-ε4 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.

4 Empirical Strategy

We estimate the following regression via OLS:

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

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-ε4 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

[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.

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.

5 Main Results

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 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 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 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 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 based on carrier status of the APOE-ε4 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-ε4 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 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 A1). 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, 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 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.

5.2 Economic Outcomes

We now present and discuss the estimated relationship between the genetic risk for AD and several economic outcomes. Table 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 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 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.

[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.

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 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 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.

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 7 and 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,

[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.

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.

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 9–14. 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 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.

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 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.

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 A2. 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

[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).

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 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 A3, 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 A4).

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 A5 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 A6). 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.

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 2 with years of education (Table A18) and an indicator for completing at least some college (Table A19) 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 A7–A17. The addition of education controls does not change our main conclusions regarding expectations. In particular, we still find a robust position 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.

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-ε4 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-ε4 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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Tables and Figures

Table 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
CD and Memory-Related Disease:			
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
EA Score	0.007	0.996	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:			
Work 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 (LI)	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
Discuss future medical care with anyone	0.553	0.497	10,059
Environmental factors:			
Ever had union jobs	0.151	0.358	88,191
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 parents' MRD diagnosis	0.295	0.456	37,766
Childhood SES (Family Welloff)	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).

Table 2: Relationship between Genetic Risk for AD and Cognition and Memory-Related Disease (MRD) Diagnosis

	L-W Score			Ever MRD diagnosis			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
AD Score	-0.236***	-0.130***	-0.111***	0.003**	0.003**	0.002	0.002
	(0.035)	(0.031)	(0.030)	(0.001)	(0.001)	(0.001)	(0.001)
APOE (At least 1 copy)	-0.403***	-0.401***	-0.291***	0.010***	0.009***	0.007***	0.005**
	(0.065)	(0.056)	(0.055)	(0.003)	(0.003)	(0.003)	(0.003)
APOE (2 copies)	-0.560**	-0.525***	-0.364**	0.020*	0.020*	0.015	0.013
	(0.217)	(0.193)	(0.186)	(0.011)	(0.011)	(0.011)	(0.011)
EA Score		0.255***	0.251***		-0.002*	-0.001	-0.001
		(0.025)	(0.025)		(0.001)	(0.001)	(0.001)
Ever demented (L-W)						0.125***	
						(0.013)	
Ever Impaired (L-W)						0.031***	
						(0.003)	
Ever MRD			-2.138***				
			(0.190)				
Eventually diagnosed			-1.516***				
			(0.093)				
L-W Score Dummies	No	No	No	No	No	No	Yes
Standard Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Principal Comp.	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Years	1998-2018	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	50-85
Sample	All	All	All	All	All	All	All
Mean	16.392	16.392	16.392	0.021	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	22,899
N w/ APOE (2 copies)	1,717	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	88,045
R^2	0.133	0.251	0.275	0.017	0.024	0.055	0.069

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 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 3: Relationship between Genetic Risk for AD and Currently Working for Pay

	Currently working for pay					
	(1)	(2)	(3)	(4)	(5)	(6)
AD Score	-0.017***	-0.011**	-0.009**	-0.010**	-0.009*	-0.008
	(0.005)	(0.005)	(0.005)	(0.005)	(0.005)	(0.006)
APOE (At least 1 copy)	0.005	0.005	0.007	0.005	0.007	0.002
	(0.009)	(0.009)	(0.009)	(0.009)	(0.009)	(0.011)
APOE (2 copies)	-0.011	-0.008	-0.004	-0.003	0.000	0.021
	(0.026)	(0.027)	(0.026)	(0.026)	(0.025)	(0.033)
EA Score		0.019***	0.016***	0.017***	0.015***	0.013***
		(0.004)	(0.004)	(0.004)	(0.004)	(0.005)
Ever MRD	No	No	No	Yes	Yes	No
L-W Score Dummies	No	No	Yes	No	Yes	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	50-70	50-70	50-70	50-70	50-70	50-70
Sample	All	All	All	All	All	Never diagnosed; never impaired
Mean	0.563	0.563	0.563	0.563	0.563	0.624
N w/ APOE (at least 1 copy)	14,159	14,159	14,159	14,159	14,159	9,065
N w/ APOE (2 copies)	1,180	1,180	1,180	1,180	1,180	646
N	53,598	53,598	53,598	53,598	53,598	36,622
R^2	0.151	0.177	0.184	0.185	0.191	0.177

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 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 4: Relationship between Genetic Risk for AD and Retirement

	Retirement					
	(1)	(2)	(3)	(4)	(5)	(6)
AD Score	0.012***	0.008*	0.007	0.007*	0.007	0.007
	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)	(0.005)
APOE (At least 1 copy)	-0.008	-0.006	-0.007	-0.007	-0.007	-0.000
	(0.008)	(0.008)	(0.008)	(0.008)	(0.008)	(0.009)
APOE (2 copies)	0.037	0.032	0.030	0.029	0.027	0.005
	(0.024)	(0.024)	(0.024)	(0.024)	(0.024)	(0.029)
EA Score		-0.017***	-0.015***	-0.016***	-0.014***	-0.010**
		(0.004)	(0.004)	(0.004)	(0.004)	(0.004)
Ever MRD	No	No	No	Yes	Yes	No
L-W Score Dummies	No	No	Yes	No	Yes	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	50-70	50-70	50-70	50-70	50-70	50-70
Sample	All	All	All	All	All	Never diagnosed; never impaired
Mean	0.320	0.320	0.320	0.320	0.320	0.274
N w/ APOE (at least 1 copy)	13,019	13,019	13,019	13,019	13,019	8,593
N w/ APOE (2 copies)	1,110	1,110	1,110	1,110	1,110	625
N	49,570	49,570	49,570	49,570	49,570	34,619
R^2	0.203	0.217	0.222	0.224	0.227	0.218

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

	Log total individual income					
	(1)	(2)	(3)	(4)	(5)	(6)
AD Score	-0.039***	-0.018**	-0.014*	-0.017**	-0.014*	-0.007
	(0.008)	(0.008)	(0.008)	(0.008)	(0.008)	(0.011)
APOE (At least 1 copy)	-0.015	-0.016	-0.006	-0.013	-0.004	0.022
	(0.016)	(0.014)	(0.014)	(0.014)	(0.014)	(0.020)
APOE (2 copies)	-0.011	-0.018	-0.004	-0.011	-0.001	0.032
	(0.048)	(0.043)	(0.042)	(0.042)	(0.041)	(0.058)
EA Score		0.016**	0.010	0.016**	0.010	0.006
		(0.006)	(0.006)	(0.006)	(0.006)	(0.009)
Ever MRD	No	No	No	Yes	Yes	No
L-W Score Dummies	No	No	Yes	No	Yes	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	Never diagnosed; never impaired
Mean	10.007	10.007	10.007	10.007	10.007	10.176
N w/ APOE (at least 1 copy)	21,101	21,101	21,101	21,101	21,101	10,894
N w/ APOE (2 copies)	1,541	1,541	1,541	1,541	1,541	674
N	81,392	81,392	81,392	81,392	81,392	46,604
R^2	0.169	0.245	0.253	0.247	0.254	0.243

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 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 6: Relationship between Genetic Risk for AD and Mortality and Survey Attrition

	Observed Mortality			Attrition (Appear in the next wave)		
	(1) All	(2) Male	(3) Female	(4) All	(5) Male	(6) Female
AD Score	0.001	-0.000	0.002*	-0.003***	-0.002	-0.004***
	(0.001)	(0.001)	(0.001)	(0.001)	(0.002)	(0.001)
APOE (At least 1 copy)	0.002	0.004*	-0.000	-0.005***	-0.009***	-0.002
	(0.001)	(0.002)	(0.001)	(0.002)	(0.003)	(0.002)
APOE (2 copies)	0.006	-0.000	0.011**	-0.006	0.006	-0.014*
	(0.004)	(0.005)	(0.005)	(0.007)	(0.011)	(0.008)
EA Score	-0.002***	-0.001	-0.003***	0.003***	0.002	0.004***
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Years	2006-2017	2006-2017	2006-2017	1998-2016	1998-2016	1998-2016
Ages	50-85	50-85	50-85	50-85	50-85	50-85
Mean	0.023	0.029	0.020	0.941	0.935	0.945
N w/ APOE (At least 1 copy)	24,570	10,142	14,428	22156	9293	12863
N w/ APOE (2 copies)	1,987	895	1,092	1663	729	934
N	92,933	39,275	53,658	84,578	36,053	48,525
R^2	0.021	0.023	0.018	0.058	0.060	0.057

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 4. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table 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 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 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 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 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 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 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 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 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 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 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 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 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 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 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

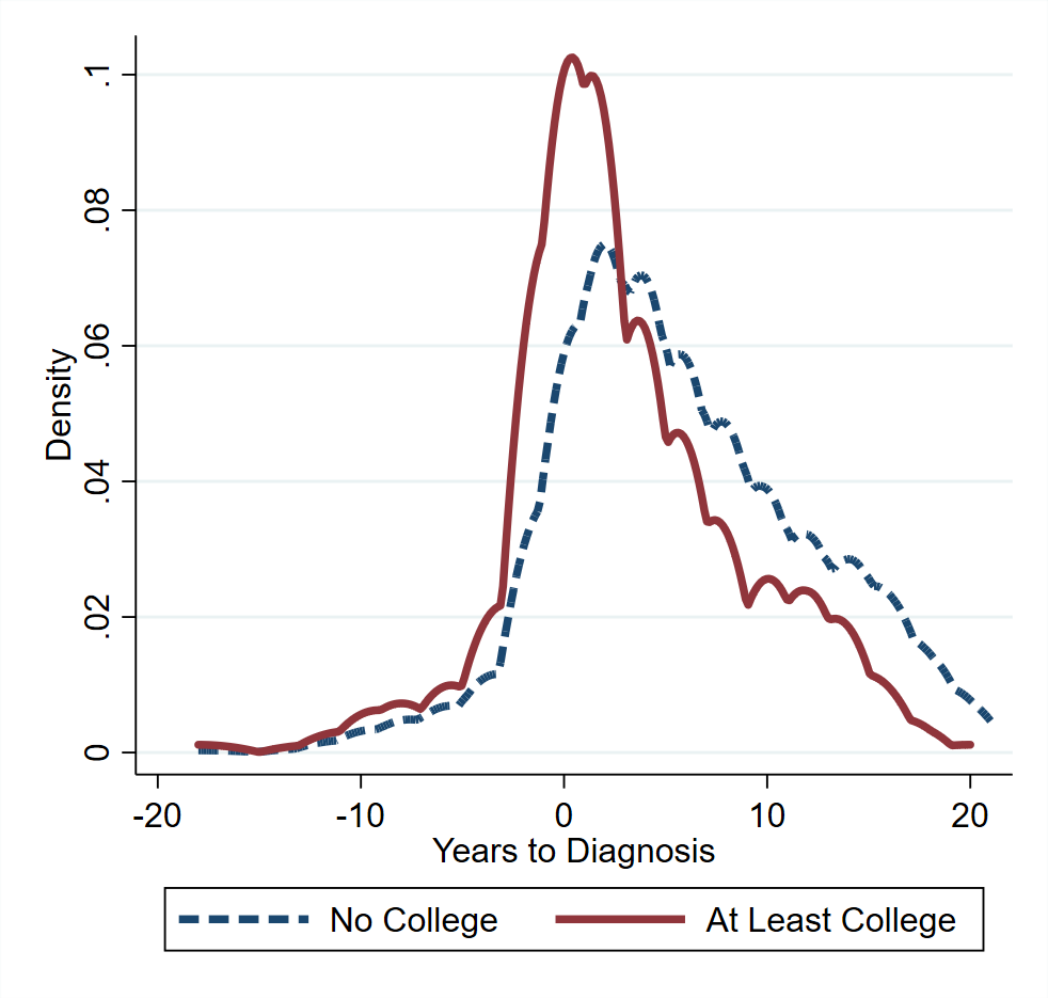
Note: Each column presents results from a separate regression where the outcome is an indicator for whether the respondent has discussed future medical care with someone. In all specifications, we control for the first 10 principal components of the genetic data as well as the standard controls for age, birth cohort, sex of respondent, and survey year as described in Section 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 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

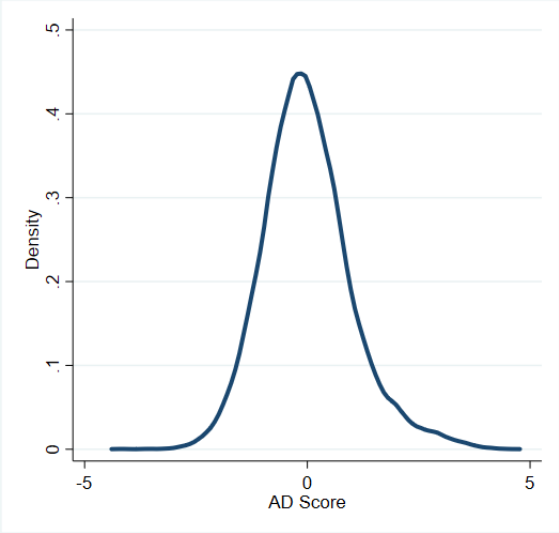
Note: Each column presents results from a separate regression where the outcome is an indicator for whether the respondent holds long-term care insurance. Results for women are in columns (1) and (2) and for men in columns (3) and (4). In all specifications, we control for the first 10 principal components of the genetic data as well as the standard controls for age, birth cohort, sex of respondent, and survey year as described in Section 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 $p < 0.01$.

Figure 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.

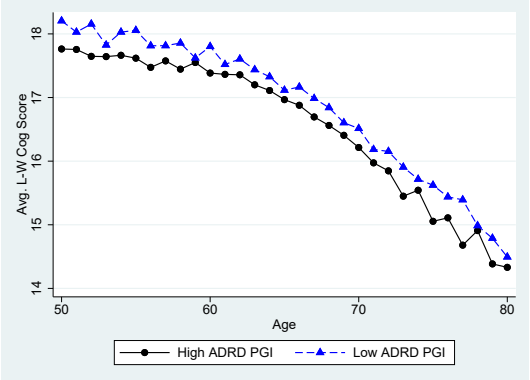
Figure 2: Distribution of the Alzheimer's Disease Polygenic Score



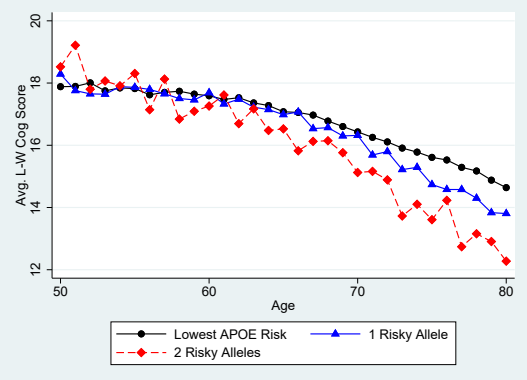
Note: The figure shows the smoothed density of the polygenic score for Alzheimer's disease in our sample.

Figure 3: Age-Cognition Profiles by Genetic Risk Groups

Panel A



Panel B



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.

Appendix

Table A1: 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 $\times$ 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 $\times$ 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 $\times$ 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 $\times$ 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 $\times$ 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 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 A2: 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 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 A3: 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 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 A4: 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 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 A5: 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 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 A6: 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 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 A7: 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 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 A8: 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 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 A9: 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 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 A10: 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 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 A11: 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 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 A12: 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 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 A13: 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 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 A14: 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 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 A15: 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 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 A16: 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 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 A17: 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 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 A18: 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 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 A19: 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 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 $p < 0.01$.