Genes and the Risk of Alzheimer's Disease: Cognitive Decline, Diagnosis, and Economic Outcomes*

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Abstract

The increasing availability of genetic data make it possible to study the evolution of outcomes and behaviors for individuals with different observed genetic markers. We examine the rich genetic architecture of Alzheimer's disease (AD). While genetic factors contribute substantially to AD risk, they are not deterministic, which allows us to examine people at genetic risk for AD who may not be diagnosed with a memory-related disease. This prompts us to ask whether such individuals are in some way *protected* from cognitive decline despite genetic risk or, alternatively, are *insulated* from its economic consequences, both of which could explain a lack of diagnosis. Using data from the Health and Retirement Study (HRS), we find evidence of the opposite. Genetic risk for AD predicts worse cognitive performance for both the full sample and among individuals who are never diagnosed. Individuals at higher risk also experience worse economic outcomes on a variety of dimensions, including work, income, and wealth. We also find that individuals at high genetic risk for AD are less likely to engage in planning activities that could mitigate the consequences of cognitive decline (e.g., assigning durable power of attorney), compared to individuals at lower AD risk, likely since they are unaware of their risk. Our findings suggest there is a large population of under-diagnosed and presumably under-treated and under-prepared people. They also raise concerns of selection bias in analyses of AD that focus solely on diagnosed individuals. Finally, they suggest there is clinically-valuable and policy-relevant information in AD genetic measures, which we demonstrate have predictive power beyond standard measures currently in use.

Keywords: Alzheimer's disease and related dementias, cognitive decline, genetic endowments, labor market outcomes, financial decisions, aging
JEL Codes: J14, I12, I14, J22, J26, D14, G51, G52

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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, burdens on informal caregivers, and the consequences of financial errors, among others (e.g., Hurd et al. 2013, Coe et al. 2018, Nicholas et al. 2021). The direct medical costs alone reached $321 billion in 2022 (Alzheimer's Association 2022). AD is a slowly progressive brain disease that includes three phases: pre-clinical AD, mild cognitive impairment, and Alzheimer's dementia. In the pre-clinical phase, cognitive symptoms such as memory loss are absent, but biological changes in the brain are present (e.g., beta-amyloid plaques, tau tangles). In the mild cognitive impairment phase, in addition to changes in the brain, problems with memory, language, and problem-solving emerge, but individuals can usually maintain independence in daily activities. The dementia phase involves severe cognitive impairment that increasingly limits an individual's ability to function independently (Alzheimer's Association 2022, Chandra et al. 2023). 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. Indeed, a large literature documents changes in economic and health outcomes that precede an eventual AD diagnosis. For example, Nicholas et al. (2021) find that those eventually diagnosed with Alzheimer's disease show evidence of financial mistakes well before diagnosis. Such analyses are important because they contribute to our understanding of the cumulative life-cycle costs of AD, and they can potentially suggest early-warning predictors to be used by individuals or clinicians. For example, information from such risk predictors could be useful for medical and estate planning, or for the targeting of early therapeutic interventions. While much is gained from this approach, we argue that it also faces two important limitations. First, case-control studies like these could be biased because they depend on an eventual diagnosis of AD to learn about earlier-life trajectories and predictors. Since an AD diagnosis is itself endogenous (e.g., it depends on the choice to seek medical attention), predictors of this outcome will reflect a mix of both early-warning signs for AD and factors affecting the propensity to seek medical attention. Second, it may be challenging to practically implement such

[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.

predictive systems if they require the simultaneous tracking of several streams of real-time financial, economic, and health data. These predictors may also have limited value if they only signal that an individual is at high risk after they already start exhibiting signs of financial or medical hardship.

In this paper, we study the relationship between observed genetic risk for late-onset Alzheimer's disease and trajectories of cognition, memory-related disease, economic outcomes, planning activities, and awareness of risk using the *Health and Retirement Study* (HRS). Focusing on genetic risk is natural in this context because genetic factors play a large role in the etiology of AD. Twin studies suggest that genetic factors explain 60–80% of the variation in the risk of AD (Gatz et al. 2006). Unlike eventual diagnosis, genetic factors are pre-determined at conception. Thus, molecular genetic data available in the HRS allow us to observe outcomes and trajectories for those with varying underlying genetic risk for AD—including, crucially, individuals who are not diagnosed with AD or do not exhibit signs of cognitive decline based on standard measures. Our study is guided by an overarching question: what drives variation in diagnosis with a memory-related illness among people with similar genetic risk for AD? Put another way, what does it mean if an individual with a high genetic risk of AD is not diagnosed? As genes are not deterministic, one possibility is that some individuals with high genetic risk are *protected*, i.e., they do not exhibit cognitive decline despite their genetic risk, obviating the need for a diagnosis. Another possibility is that some individuals are *insulated*, i.e., they do exhibit cognitive decline, but no evidence of its economic consequences, which means their illness may go undetected.

To address this question, the complex genetic architecture of AD leads us to examine three sets of genetic variables. The first measure 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. The second measure is 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). The third measure is a polygenic score for educational attainment (hereafter the EA score). Education has traditionally been central to the theory of "cognitive reserve," which posits that some individuals can withstand the brain changes and pathology associated with Alzheimer's disease, maintain function, and avoid cognitive decline better than others. Education is thought to be positively associated with one's cognitive reserve (Stern 2012), although it is not clear if this reflects a causal effect of education on cognition or operation of some brain features that both promote education and add to cognitive reserve later in life.

We present five main sets of results. First, we establish that increased genetic risk for AD, whether measured by the AD score, the EA score, or carrying an APOE allele, is indeed related to

lower cognitive function. These genetic factors are also significant predictors of being observed with extremely low cognitive scores consistent with dementia. This is not surprising. What is surprising is that these patterns hold even among those who are never diagnosed with a memory-related disease while observed in the HRS. High-risk individuals who were never diagnosed thus do not appear to be well protected from cognitive decline. This in turn suggests that individuals with observable genetic risk may be under-diagnosed, under-prepared, and under-treated.

Second, critically, we demonstrate that the predictive power of the genetic measures studied here may be of practical clinical value. Among those aged 50–64 without an existing diagnosis of a memory-related disease and who do not exhibit cognitive scores consistent with impairment or dementia, we find that the AD polygenic score and APOE carrier status significantly predict the probability of later cognitive decline as well as the probability of being diagnosed with a memory-related disease, even after controlling extensively for current and past cognitive test results. That is, the genetic measures contain information beyond what is likely available to an individuals' primary care physician.

People at higher genetic risk might avoid diagnosis if they are insulated from the consequences of decline, e.g., its effects on economic outcomes. Our third set of results provide evidence against this hypothesis, as we show a higher AD score is associated with negative economic outcomes, including a lower probability of working for pay, lower total income, and lower household wealth. These relationships survive after controlling for cognitive function and memory-related disease diagnosis and among those who are never diagnosed while observed in the HRS.

Given that the genetic measures predict economically meaningful differences in cognition and economic outcomes, it is reasonable to ask whether individuals at greater genetic risk are aware of their elevated risk and make medical or financial preparations to insulate themselves and their families from future economic losses and communicate their preferences about future health care. Our fourth set of results reveal that those with higher AD scores are less likely to engage in a variety of planning activities, including holding long-term care 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 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. However, we do find suggestive evidence that some APOE carriers are more likely to engage in planning activities.

Our fifth set of findings provide an explanation for the differences in planning behavior by AD genetic risk. APOE carriers appear to be more aware of their risk—they report higher probabilities

of needing nursing home care and developing AD or dementia in the future. They are also more likely to have parents who were diagnosed with memory-related disease and who received nursing home care. By contrast, those with higher AD scores do not appear aware of their elevated risk. Together, our findings highlight the complex genetic architecture of AD, and that those who are at elevated risk due to higher AD scores may be a particularly vulnerable group that is unaware of their increased prospects of cognitive decline.

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 AD diagnosis, confers costs in terms of worse economic outcomes, such as lower employment, income, and wealth, 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, Nicholas et al. 2021, Li et al. 2022, Mazzonna and Peracchi forthcoming). Chandra et al. (2023) 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).[2] Consistent with these studies, we show that genetic endowments for AD are associated with worse economic outcomes and decreased planning activities from ages 50–70 and even among those not diagnosed.

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,

[2]Our results also complement the work of Ameriks et al. (2023) which develops a theoretical model of cognitive decline that features limited awareness of such decline and a desire to maintain control of one's own finances while capable, which can lead to suboptimal timing of the transfer of financial control.

and save more in "hands off" assets.[3] Linnér and Koellinger (2022) study the extent to which 27 polygenic scores for common medical conditions predict mortality in the HRS, and find that the AD score had the second largest association with mortality. Other work has largely explored relationships between the polygenic score for educational attainment 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 measure AD risk using three separate *molecular* genetic variables. Human DNA consists of a sequence of roughly 3 billion nucleotide base-pair molecules spread out across 23 chromosomes. At each location in the genome, individuals can possess one of two possible base pairs: an adenine-thymine (AT) pair or a guanine-cytosine (GC) pair. At nearly all of these locations, every human being has exactly the same base-pair molecules. However, at a small number (less than 1%) of these sites, individuals can differ. Locations featuring such differences are referred to as "single nucleotide polymorphisms," or SNPs. Since an individual inherits one copy of a chromosome from their father and one from their mother, individuals can possess 0, 1, or 2 copies of a particular molecule (AT or GC) at each SNP.

Our first molecular genetic measure for late-onset AD (i.e., onset after age 65) is APOE-ε4 (hereafter APOE) carrier status.[4] An individual is a carrier of the APOE-ε4 variant if they have a specific combination of base-pair molecules at two SNPs. Being a carrier of the APOE genetic variant is the strongest single 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.

Given its devastating impact, there is a vast body of research on different aspects of APOE. However, APOE is not the only source of genetic risk, and indeed it does not contribute to genetic risk for the majority of people. While about 15–25% of individuals carry at least one copy of the variant, only 2–5% possess two copies and thus face the maximum genetic risk. There are many other variants in the genome that contribute to AD risk, although their individual associations with AD tend to be much smaller than that exhibited by APOE. We measure an individual's risk from

[3] In contrast to Shin et al. (2019), we use a more recent polygenic score for AD and distinguish between AD risk that manifests from a higher AD score (that excludes the APOE region) versus carrying the APOE allele.

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

these variants using a *polygenic score for AD* (henceforth: *AD score*). The AD score is a weighted index of 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. A polygenic score (PGS) is constructed as:

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

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.[5]

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.[6] 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.[7] We provide more details on the AD score and our measures of APOE in the next section.

Finally, our third genetic measure is a polygenic score for educational attainment (henceforth: *EA score*). The construction of the EA score is similar to that of the AD score, and is thus a weighted average of the genetic variants that predict years of completed education. We use the EA score based on the GWAS of Lee et al. (2018). The literature on cognitive reserve has emphasized the strong correlation between educational attainment and cognitive performance later in life. Individuals who are observed acquiring higher levels of human capital could have patterns of neuronal connectivity that promote cognitive resilience in the face of aging, or give the brain more resources to compensate for decline. This could reflect either a causal effect of educational attainment on cognitive health, or the fact that connectivity promotes both human capital accumulation earlier in life and slower cognitive decline later on. It is thus natural to consider genetic factors linked to educational attainment in a study of genetic endowments and AD. The EA score has been shown to predict many of the outcomes we study here, such as income, labor supply,

[5] 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).

[6] 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).

[7] 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.

and wealth at retirement (Belsky et al. 2016, Barth et al. 2020, Papageorge and Thom 2020). It is also associated with a host of variables related to cognition, such as measures of cognitive decline, biases in expectations, and time horizons for decision-making (Papageorge and Thom 2020).

The use of genetic data has a number of benefits and drawbacks, many of which have been thoroughly discussed in other papers. We provide a very brief summary here. First, the measures tend to be noisy in the sense that they only capture a fraction of the variance in outcomes attributable to genes. This means that there are likely to be other genetic variants that predict the outcome and which are omitted from the analysis. Second, genes are unlikely to be exogenous to family environments, which means it is difficult to claim that estimated coefficients capture a causal effect. In the case of the EA score, basic associations with economic behavior and outcomes tend to survive more demanding econometric specifications, such as those that rely on within-family variation. Third, the construction of polygenic scores is largely limited to large datasets with individuals of European descent and it is well understood that using such scores to make cross-ethnic-group comparisons can be misleading, which means we are limited to studying individuals of European descent. In general, these difficulties suggest caution when interpreting our estimated coefficients. Our analyses here tend to be descriptive, which can provide guidance for future analyses, including plausible interactions with environmental factors and more theoretical models that capture the structural relationships between genes, illness, behavior, and outcomes. Moreover, descriptive analyses can provide insights with clinical value if they show that certain genetic measures are able to predict outcomes, such as cognitive decline.

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 scores we use are based on the findings from GWAS where the discovery samples consisted only of those of European ancestry (i.e., non-Hispanic whites). About 12,000

genotyped individuals have genetic European ancestry. In what follows, we describe key variables used in our analysis, construction of the analytic sample, and summary statistics.

3.1 Key Variables

3.1.1 Genetic Variables: AD Score, APOE, and the EA Score

As described earlier, we consider two measures of genetic risk for late-onset AD—the polygenic score for AD and whether an individual carries the APOE allele. The polygenic score is based on the Kunkle et al. (2019) GWAS and includes all SNPs regardless of their p-value. The score excludes the APOE region based on the recommendations in Ware et al. (2020). The AD score is normalized to have mean zero and standard deviation of one. We create two APOE-related indicators. The first takes value one if the individual carries at least one copy of APOE-$\varepsilon 4$, and the second takes value one if the individual has exactly two copies of the allele.[8]

Also described earlier, we use a polygenic score for educational attainment, the EA score, which is based on the Lee et al. (2018) GWAS. Like the AD score, the EA score includes all SNPs and associated coefficients regardless of p-value and is normalized to have mean zero and standard deviation one. While research using the AD score in economic analysis is scant, a number of studies investigate how the EA score relates to lifecycle behavior and outcomes and interacts with various environmental factors.

3.1.2 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).[9] We rely on the score itself and create indicators for whether

[8]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.

[9]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

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).

3.1.3 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 analyze log total individual income, which includes income from earnings, pensions, annuities, Social Security, unemployment and workers' compensation, and other government transfers. We also examine log household wealth.[10] We winsorize both income and wealth at the 1st and 99th percentiles.

3.1.4 Planning Outcomes

We consider several measures related to later-life planning activities. 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 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.

3.1.5 Expectations and Awareness of Risk

We study whether genetic risk for AD correlates with expectations about mortality, long-term care utilization, and developing AD. The HRS asks respondents aged 65 and under about their expected

(Gianattasio et al. 2019). The three tests included in the L-W score are asked of individuals of all ages, whereas other 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.

[10] Household wealth is the the sum of the value of primary residence, value of secondary residence, net value of real estate (not primary residence), net value of vehicles, net value of businesses, net value of IRA, Keogh accounts, net value of stocks, mutual funds, and investment trusts, value of checking, savings, or money market accounts, value of CD, government savings bonds, and T-bills, net value of bonds and bond funds, and net value of all other savings less all debt, where debt is the sum of value of all mortgages/land contracts (primary residence), value of other home loans (primary residence), value of other debt, and value of all mortgages/land contracts (secondary residence).

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, which we consider as a measure of expected long-term care utilization. The question is only asked to those not currently in a nursing home. Each wave, about a 10% random sample of the core HRS respondents are asked questions from experimental modules. We pool together responses to questions from these modules in 2002, 2012, and 2016 that ask respondents about their probability of developing Alzheimer's disease or dementia in the future.[11] Given the experimental modules are fielded to a small subsample and we only consider responses among genotyped individuals, sample size is substantially smaller when we analyze expected probability of developing AD.

Individuals may also learn about their risk for AD via their parents' development of the disease or use of long-term care. We therefore examine how own genetic risk for AD correlates with whether the respondent's mother or father has ever been diagnosed with MRD as well as whether a parent ever received nursing home care. Parental nursing home care is determined via questions about where and with whom a parent currently resides (if they are alive) and whether a parent received nursing home care prior to death (if a parent passed away since the prior wave or was deceased at the respondent's initial interview).

3.2 Analysis Sample Construction

The main sample consists of person-year observations who are genotyped and aged 50–85 between 1998–2018. However, analysis sample sizes fluctuate across regressions, and the age range of our analysis samples vary depending on the outcome we consider. For example, when we consider employment-related outcomes, we limit the sample to those 50–70 years old since most people are retired by age 70. For planning outcomes, some questions are asked for only certain age groups or during specific periods. Moreover, for some analyses, we limit the sample to age 50–70, prior to widespread onset of AD, though results are robust to expanding the sample to those aged 50–85. Finally, for some outcomes which are "absorbing states," we drop observations after the individual first enters the state. For example, when we examine the relationship between genetic risk for AD and the probability of ever being cognitively impaired, we drop observations after the first wave in which they register an L-W score that is less than 12.

[11] There are additional experimental modules that ask about the development of Alzheimer's disease. However, the question wording and the scale of the responses in 2002, 2012, and 2016 are most comparable. In 2002, respondents are asked "Using a scale of 0–100 where 0 means no chance and 100 means absolutely certain, what are the chances that you will ever develop Alzheimer's Disease?" In 2012, respondents are asked "Using a scale of 0–100 where 0 means no chance and 100 means absolutely certain, what are the chances that you will develop Alzheimer's Disease sometime in the future?" In 2016, the question is "On a scale of 0–100, what is the percent chance that you will develop dementia sometime in the future?"

3.3 Summary Statistics of Key Variables

Summary statistics for key variables used in our analysis are found in Table 1. We show these moments for the maximum number of person-year observations for each variable, which is why the sample size varies. Average birth year is 1940, about 41.9% of the sample is male, the average age is 67.6, and the average years of completed education is 13.3. About 32% of observations have completed at least some college.

By construction, the polygenic scores have means that are near zero and standard deviations near one. Slight deviations arise because the scores were standardized for all individuals, some of whom were dropped from the analytic sample. The mean AD score is slightly below zero, reflecting that people at higher risk of AD exit the sample (through attrition and/or death). Relatedly, the mean EA score is slightly above zero, likely reflecting positive selection into the sample. Roughly 26% of the sample has at least one copy of APOE and 2% have two copies, which dramatically increases the likelihood of developing AD.

The distributions of the polygenic scores are found in Figures 1 and 2, for the AD score and the EA score, respectively. As the figures show, both scores are symmetric around the mean and seem to be normally distributed. Moreover, Figures 3 and 4 show the joint distribution between the two scores and do so in different ways. The key takeaway is that, while there is a correlation between the scores, it is very low (roughly 0.045). The interpretation of this relatively low correlation requires some care. One possibility is that there is little overlap between the SNPs that predict education and those that predict cognitive decline. An alternative possibility is that there is in fact greater overlap, but that the GWAS did not capture it. For example, suppose there is a SNP that is both highly predictive of lower education and of AD diagnosis, but that it is relatively rare. It is possible that it appears as important in the AD score, but that there are too few individuals in the GWAS sample who have the SNP for it to play a large role in the EA score. In short, the noisiness of the scores may obscure a stronger correlation between the two and therefore we cannot take the low correlation as definitive proof of near-independence.

Turning to direct outcomes related to cognition and memory-related disease among those aged 50–85, the average L-W score is 16.4. Slightly less than 3% of the sample ever has an L-W score low enough to be categorized as demented, while about 22% are ever impaired or demented. About 2% of our sample ever receives a memory-related disease diagnosis. Figure 5 examines how the relationship between genetic risk for AD and the L-W score evolves over the later life-cycle observed in the HRS. Panel A 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 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 allele exhibit increasingly lower L-W score averages compared to the least risky APOE group. In Panel C, we plot differences in the age-cognition profiles by above and below median values of the EA score. We see a fairly constant gap in unconditional average L-W scores between the two groups. These differences raise the possibility that while the AD score, EA score, and APOE represent genetic endowments linked to cognitive performance, they may operate through different mechanisms and capture different aspects of cognitive decline.

We also examine economic outcomes relevant to people at risk for cognitive decline. According to Table 1, 56% of those aged 50–70 work for pay while 32% is retired. The remaining observations are people who are not working for pay, but who do not describe themselves as being completely retired. Average total individual income is slightly above $22,000 and average household wealth is just above $240,000.

The HRS includes a number of later-life planning outcomes, which we also examine. Among those aged 50–70, about 13% hold long-term care insurance (LTCI), 71% hold life insurance policies, and about 56% report having a witnessed will. Among those aged 65–70, 48% have a living will, 46% have assigned someone a durable power of attorney for future health care, and 59% have discussed future medical care with anyone. Together, these means suggest that a majority of the sample reports engaging in some kind of later-life planning. Indeed, of the six planning activities we consider, on average, individuals engage in 2.7 of them.

Part of our analysis includes assessing to what degree individuals appear to be aware of their risk of cognitive decline. For example, individuals are asked to report the subjective probability that they live to 75, and on average, individuals believe they have a 66% chance. On average, individuals aged 65–70 report an 11% probability of moving to a nursing home in the next five years. On average, those aged 50–70 report a 36% chance of developing Alzheimer's disease in the future. These expectations could relate to incentives to purchase LTCI and engage in other financial and non-financial planning activities. Last, about 28% of individuals aged 50–70 have a parent who has ever been diagnosed with MRD, and almost 38% of respondents by age 70 have a parent who received nursing home care.

4 Empirical Strategy

We show a variety of results, including descriptive figures, but most of our results are estimates of the following regression via OLS:

$$\begin{aligned} Y_{it} = \beta_0 \; + \; \beta_1 ADScore_i + \beta_2 \mathbb{1}(APOE\,copies_i \geq 1) \; + \; \beta_3 \mathbb{1}(APOE\,copies_i = 2) \\ + \; \beta_4 EAScore_i \; + \; \beta_5 X_{it} \; + \; \varepsilon_{it} \end{aligned} \tag{2}$$

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. $EAScore$ is the polygenic score for educational attainment. Following Barth et al. (2020), X_{it} includes "standard controls"—birth year dummies, age dummies, survey wave dummies, a male dummy, and two-way interactions between the male dummy and the birth year dummies and age dummies. As is standard practice, X_{it} also includes the first 10 principal components of the genetic data to account for possible population stratification, and we allow those coefficients to vary by gender. We cluster standard errors at the individual level.

In some specifications, we include additional individual-level controls. In particular, we add dummy variables for each value of the current Langa-Weir score to flexibly control for cognitive function. We sometimes control for whether an individual has ever been diagnosed with MRD. We include these controls to learn whether the genetic endowments for AD have predictive power even after accounting for cognitive function and MRD diagnosis. Where indicated, we also control for completed education via a full set of degree dummies and indicators for different numbers of years of education, fully interacted with gender.

5 Main Results

Our main results estimate the relationship between genetic endowments and a series of outcomes: cognitive function and memory related-disease diagnosis, labor supply, household economic resources, planning activities, and awareness of one's risk for Alzheimer's disease.

5.1 Alzheimer's Disease-Related Outcomes

Our first set of results demonstrate that genetic endowments predict disease-related outcomes, such as cognitive impairment and dementia for different sets of individuals, including those who never receive a memory-related disease diagnosis. These relationships are estimated using the

model discussed in the previous section, and results are presented in Table 2. In Panel A, we show results from regressions of the L-W score onto different sets of controls. In all cases, the standard set of controls outlined in the previous section are included. The first four columns present results for the full sample. In column (1), we show estimates from a model that includes the AD score and dummy variables for at least one copy of APOE and two copies of APOE. All three variables have statistically significant and sizable associations with the L-W score. A one standard deviation increase in the AD score is associated with a decrease of 0.24 points in the L-W score from a mean of 16.4. Having at least one copy of APOE is associated with a L-W score decrease of 0.40 and having two copies is associated with a further decrease of 0.56.

The second column includes the EA score, which slightly lowers the magnitude of the coefficients on the AD score and APOE dummy variables. Moreover, a one standard deviation increase in the EA score is associated with an increase of 0.63 in the L-W score, meaning that having a one standard deviation higher EA score more than offsets having a one standard deviation higher AD score, underscoring the importance of education in understanding the genetic architecture of AD. The third column includes a full set of controls for completed education. While this diminishes the size of the coefficients on the AD and EA scores, all genetic predictors remain highly significant. At least part of the association between genes and cognitive performance can be explained by the relationship between genes and education.

It is useful to ask whether the cognitive risk measured by the genetic endowments is ultimately reflected in eventual diagnosis. That is, is the diminished cognition that comes with higher genetic risk fully reflected in later MRD diagnosis, or does it linger undiagnosed. To investigate, we consider whether these relationships are limited to individuals who are ever diagnosed with an MRD. We add two dummy variables: one for "ever MRD" (reports MRD in the current wave or any wave prior) and one for "eventual MRD" (reports MRD diagnosis at any point in the HRS sample period). Ever having an MRD diagnosis and eventual diagnosis both predict a lower L-W score (by 2.13 and 1.52 points, respectively). Adding these variables moderately lowers the size of the coefficients on the APOE variables, slightly attenuates the coefficient on the AD score, but has virtually no impact on the EA score coefficient. This suggests that holding diagnosis fixed, genetic variables predict cognitive function as measured by the L-W score. To explore further, we next focus on the subsample of individuals who are never diagnosed with an MRD while observed in the HRS (columns 5–7). In column (5), we return to a specification with only the three AD genetic variables. We find that among the undiagnosed, a one standard deviation increase in the AD score is associated with 0.23 point decline in the L-W score. Having at least one copy of APOE lowers the L-W score by 0.27 and having two copies further decreases it by 0.39, though the latter is not significant

at conventional levels. Adding the EA score (column 6) lowers the size of these coefficients, and a one standard deviation increase in the EA score is associated with a 0.61 increase in the L-W score. These relationships are somewhat diminished when we add a full set of education controls in column (7), but remain substantial and significant. According to the results of this specification, among the undiagnosed, a one standard deviation rise in the AD score predicts a decline of 0.12 points in the L-W score. A corresponding increase in the EA score predicts a 0.24 point increase.

To explore whether the associations between genetic risk and the L-W score are clinically meaningful, we replace the outcome variable with a dummy variable for ever having an L-W score low enough to be considered cognitively impaired or to suffer from dementia (i.e., less than 12). We exclude individuals from the sample after their first L-W score less than 12, as we treat cognitive impairment and dementia as absorbing states. Results are presented in Panel B of Table 2. Across all seven specifications, estimates are significant and generally stable. Among the undiagnosed and including the full set of controls, a one standard deviation rise in the AD score corresponds to a 0.4 percentage point increase in the probability of impairment or dementia from a baseline of 4.7%. A corresponding increase in the EA score predicts a 0.6 percentage point decline. Moreover, having at least one copy of APOE corresponds to a 0.7 percentage point increase in the probability of impairment or dementia, and those with two copies see an additional 1.7 percentage point increase.[12]

We also examine whether genetic endowments related to AD predict memory-related disease diagnosis. The outcome is an indicator for whether an individual is ever diagnosed, and we exclude individuals from the sample after their first report of an MRD diagnosis. Results are presented in Table 3. Each of the three columns progressively adds covariates in a manner similar to Table 2. Column (1) includes the genetic variables related to AD; column (2) adds the EA score; and column (3) adds controls for completed education. Across the specifications, we find economically meaningful and statistically significant associations between the AD genetic measures and diagnosis. A one standard deviation rise in the AD score increases the probability of MRD diagnosis by 0.1 percentage points, from a mean of 0.7%. Carrying the APOE allele increases this probability by 0.5 percentage points, and carrying two copies further increases this probability by 0.9–1.0 percentage points. Once educational attainment controls are included, we do not find a statistically significant relationship between the EA score and diagnosis. Overall, we find that genes that predict cognitive decline also predict diagnosis.

[12]We repeat this analysis and replace the outcome with an indicator for ever being demented (i.e., scoring less than 7 on the L-W score), which is a rare outcome. Results are shown in Appendix Table A1. Carrying the APOE allele increases the probability of dementia non-trivially. A higher EA score is associated with a lower probability of dementia. In some specifications we find a higher AD score predicts a higher dementia risk, but in specifications with educational attainment controls, the relationship attenuates and is no longer significant.

The results from Tables 2 and 3 demonstrate that genetic endowments predict lower cognitive performance, higher rates of impairment, and higher rates of MRD diagnosis, as would be expected.[13] The relationships between genetic endowments and cognitive performance hold even among individuals who are never diagnosed with an MRD, which runs counter to the idea that those without a diagnosis but a high genetic risk of AD are somehow protected from cognitive decline, or are able to avoid significant impairment. They are not. The strikingly stable coefficients suggest there is a potentially large population of individuals who suffer from impaired cognitive performance, but are not observed as such. This result not only raises concerns about under-diagnosis, but also opens questions about what we know about cognitive decline and MRD including AD. The lessons we learn about AD, for example, are mostly drawn from those with a diagnosis, which is not a concern if most people with the illness are diagnosed. However, if many go undiagnosed, this raises questions about potential selection bias and generalizability, i.e., that lessons we learn about AD apply solely to the population that is also diagnosed

5.2 Do Genes Have Clinical Value?

The results from the previous section demonstrate that the genetic endowments studied here are significantly related to cognitive performance and eventual MRD diagnosis. However, these results do not necessarily establish the genetic measures as useful clinical tools for predicting individual risk or targeting possible interventions. In order for these measures to be valuable for these tasks, they need to be able to predict risk for future AD above and beyond the kind of data and medical history that a physician would already have at their disposal. That is, it would be ideal to ask whether the genetic measures predict future AD given current cognitive health. To operationalize this in a blunt but transparent way, we designate ages 50–64 as an "early period" and ages 65–80 as a "late period." We ask whether the genetic measures predict eventual impairment and/or MRD diagnosis in the late period among individuals that otherwise appear to be healthy in the early period. We therefore restrict our sample to those individuals who are not observed with an L-W score less than 12 (impairment), and who are not diagnosed with MRD in the age range 50–64. We then calculate different summary statistics related to their cognitive performance during this period, including their average L-W score, minimum L-W score, and maximum L-W score. This approximates the infor-

[13] In Appendix Tables A2 and A3, we show associations between the genetic endowments and sample attrition, and find those with higher AD scores and APOE carriers are less likely to appear in the next survey wave, while higher EA scores associate with a higher probability of being observed next wave. In Appendix Tables A4 and A5, we do not find compelling evidence that the increased attrition for those with higher AD genetic risk is due to increased risk of mortality, though a higher EA score predicts lower mortality risk. Thus, those with higher AD genetic risk are less likely to consistently respond to the survey, perhaps due in part to their lower cognitive function. Our results therefore likely reflect those of a selected set of healthier individuals.

mation set that could be available to an individual, their family, or their doctor. We then estimate regressions that predict whether these individuals are eventually observed with impaired cognition or dementia, or as having an MRD diagnosis in the later age ranges 65–80. These regressions are different from our basic model in Equation 2 because they are fundamentally cross-sectional regressions. Thus our control set is different and features the first 10 genetic principal components, a full set of birth year dummies, a full set of dummies for years of schooling, a full set of dummies for highest degree, and a male dummy and its interactions with all of the previous regressors. Importantly, we control for the average L-W score (at ages 50–64), a full set of dummies for minimum L-W score (at ages 50–64), and a full set of dummies for maximum L-W score (at ages 50–64).

Table 4 reports the estimated coefficients on the genetic predictors from the specification described above. We find that the AD score significantly predicts future impairment, dementia, and diagnosis conditional on current cognition. We also generally find strong positive coefficients on the APOE dummy variables. However, we do not find any significant associations with the EA score. The sizes of the coefficient estimates on the genetic variables is striking. Unsurprisingly, being an APOE carrier (especially having two copies) dramatically increases the risk for major cognitive problems in the future. While about 4% of individuals in this sample are eventually diagnosed with MRD (at ages 65–80), carrying one copy of APOE is associated with an increased risk of about 4 percentage points, and carrying two copies is associated with an additional 9 percentage point increase. It is noteworthy, however, that variation in the AD score predicts meaningful differences in future MRD conditional on current cognition. A one standard deviation increase in the AD score is associated with a 1.3 percentage point higher chance of eventually having an MRD diagnosis, a 33% increase above baseline risk. Moreover, this relationship appears to be fairly linear. Figure 6 plots a local polynomial approximation of the relationship between the AD score and the MRD diagnosis outcome, after it has been residualized on all of the control variables from the specification in Table 4. There is a steady increase in the risk of future MRD diagnosis with higher and higher AD scores, generating large differences in the risks faced by those in either tail of the AD score distribution.

These results complement the findings from the prior subsection by showing that in addition to APOE carrier status, the AD score may also be a clinically relevant predictor for future AD. Even when controlling for information that individuals (or their doctors) are likely to have about their current cognition, the AD score predicts large and statistically significant differences in AD risk. Beyond the possible clinical use, this also means that the AD score represents a powerful *observable* measure of AD risk. This allows us to answer a series of questions about the effect of AD risk on important outcomes, the kinds of behaviors that individuals might undertake (or not) in response to their risk, and whether individuals are aware of their own genetic risk. We

turn to these questions in subsequent sections.

5.3 Economic Outcomes

The previous two sections provide evidence that genetic measures are predictive of diagnosis and, moreover, could be used to predict diagnosis earlier than traditional measures. The results also suggest that individuals who are never diagnosed, but are at high risk of AD, exhibit worse cognitive function. Their lack of diagnosis does not mean they are somehow protected from decline, which underscores the need for additional diagnostic measures. Nevertheless, it might be the case that people at high risk of AD and who exhibit cognitive decline do not suffer the consequences of cognitive decline. If they are somehow insulated from the negative consequences, this would not only explain a lack of diagnosis, but would mean that finding ways to diagnose them is perhaps not an urgent need.

To explore this question, we examine how genetic endowments related to AD predict economic outcomes. We start with whether or not an individual works for pay. Results are in Table 5 and are presented following a similar progression as we did in Table 2 in Section 5.1, but with some additions. Each column shows results from a separate regression where the outcome is an indicator for whether the respondent currently works for pay, and the sample includes those aged 50–70. In all specifications, we include the standard controls. In column (1) we include the AD score and APOE dummies and find that a one standard deviation rise in the AD score is associated with a statistically significant 1.7 percentage point decrease in the likelihood of working for pay, from a baseline of 56.3%. The coefficients on APOE carrier status are not significant. Column (2) adds the EA score, which lowers the AD score gradient to 1.5 percentage points, and the corresponding gradient on the EA score is 4.0 percentage points. In column (3), we add educational attainment controls, which lowers the AD score association to a 1.1 percentage point decline and that of the EA score by over half, to a 1.9 percentage point increase. In column (4), we add dummy variables for the contemporaneous L-W score, which slightly attenuates the coefficients on the AD and EA scores, but they remain statistically significant.

We next examine whether the relationship between genetic variables and working for pay holds when we control for ever and eventual MRD diagnosis, and find that they do (column 5). To further explore this point, we repeat the exercises from columns (1)–(4) among those never diagnosed with an MRD while observed in the HRS. Estimates are reported in columns (6)–(9). The final specification regresses an indicator for working for pay onto the four genetic variables and a full set of education and L-W controls. We find that a one standard deviation rise in the AD score is associated with a 0.9 percentage point decrease in working for pay, while a one standard deviation rise in the EA score is associated with a 1.4 percentage point increase.

The results from Table 5 provide evidence against the idea that lack of MRD diagnosis is

due to individuals somehow being protected from the economic consequences of cognitive decline. Instead, these results support the idea that there are people at high risk of cognitive decline who may in fact be suffering from cognitive decline and its economic consequences, but who never receive a diagnosis. This underscores the clinical value of alternative diagnostic measurements as well as concerns about our current knowledge of MRD, which is largely based on a selected sample of individuals who for one reason or another were diagnosed.

We examine a host of other economic outcomes, including retirement, log individual total income, and log household total wealth. Rather than present the full set of results for each outcome as in Table 5, we provide a summary, only showing results from the specifications that correspond to columns (4) and (9) in Table 5. The results are presented in Table 6, and the full set of results for each outcome are presented in Appendix Tables A6–A8. Across outcomes and specifications, we generally find further evidence that the AD score predicts worse economic outcomes and the EA score predicts better outcomes even after controlling for the L-W score, completed education, and the standard controls. Moreover, these relationships hold for the full sample and when we limit attention to the never-diagnosed sample.

We examine wealth more closely given it is measured at the household, rather than individual, level. That means for married couples in the sample, the husband and wife are assigned the same amount of wealth. To shed light on whether the relationship between genetic measures and wealth are driven by particular household members, we estimate associations between log household total wealth and genetic measures separately for males and females. Results corresponding to columns (4) and (9) of Table 5 are presented in Table 7.[14] The estimates suggest the negative association between the AD score and wealth is driven by females, particularly married women (including those never diagnosed). In particular, a one standard deviation rise in the AD score predicts a 4.3–4.8 log point decline in household wealth. We also find weak evidence that women who carry the APOE allele have higher wealth. We do not find much gender heterogeneity in the association between the EA score and household wealth.

5.4 Planning Activities

Given that the 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 and make clear how they want their future health care to be managed. We therefore examine how genetic risk for AD associates with a variety of later-life

[14]The full set of results are presented in Appendix Tables A9–A11.

planning activities, 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. We limit these analyses to those aged 50–70 (or 65–70 for questions only asked to those 65 and over), before the widespread onset of cognitive decline and diagnosis.[15] After age 70, cognitive decline becomes increasingly evident for some people, in which case a correlation between planning activities and genetic risk for AD might not reflect planning at all, but instead a reaction to illness. Results corresponding to columns (4) and (9) of Table 5 are presented in Table 8.[16]

We find an increase in the AD score is associated with statistically significant declines in the probability of engaging in most of these planning activities. A one standard deviation increase in the AD score is associated with a 0.9–1.0 percentage point decline in the probability of having LTCI (from a mean of 13%), a 1.2–1.3 percentage point decline in having a witnessed will (from a mean of 56%), a 3.8 percentage point decline in having a durable power of attorney (from a mean of 46%), and a 2.5–2.6 percentage point decline in the probability of discussing future medical care with anyone (from a mean of 59%). These associations are remarkably similar across the full and never-diagnosed samples. We generally find no significant association between being an APOE carrier and the planning activities, though we find a marginally significant increase in the probability of having a living will for APOE carriers. Generally, the coefficients on the EA score are positive, but only reach statistical significance in the case of having a witnessed will.

In Table 9, we hone in on the sample of individuals aged 65–70 who responded to questions about all six planning activities, and examine their probability of engaging in at least 1, 2, 3, 4, 5, or 6 planning activities as well as the total number of activities engaged in. We find that a higher AD score is associated with decreased probabilities of engaging in 3, 4, or 5 or more planning activities, and with 0.07–0.09 fewer activities. We find some evidence that APOE carriers are significantly more likely to engage in at least 3 planning activities, and the point estimates on the APOE dummies are generally positive across the specifications. For the never-diagnosed sample, APOE carriers engage in 0.13 more activities, on average. The point estimates on the EA score are generally positive but only reach statistical significance when we consider the probability of engaging in 2 or more planning activities.

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 as measured by the AD score, are no more likely to engage in these planning activities, and if anything, are less likely to do so. The picture is more nuanced for APOE carriers, as we find some evidence, albeit

[15] Results are robust to expanding the sample to those aged 50–85.

[16] The full set of results for each planning activity are presented in Appendix Tables A12–A17.

weak, that they are more likely to engage in planning activities. It is possible that APOE carriers are relatively more aware of their risk for the disease compared to those with higher AD scores, which may explain the difference in planning results by genetic risk type. We explore this idea next.

5.5 Expectations and Awareness

Individuals with higher AD scores face the prospects of diminished cognition and more challenging economic circumstances, but they do not seem to engage in medical or financial planning activities, and if anything, do so less. At the same time, APOE carriers also face a substantially higher risk of diminished cognition, and we find weak evidence that they are more likely to engage in some planning activities. These patterns could be explained by differences in the extent to which individuals understand their elevated risk status. To explore this idea, we examine how genetic risk for AD correlates with self-reported expectations about mortality, future nursing home use, and future development of AD or dementia. Given the strong heritability of AD, we also examine associations between genetic risk and parents' diagnosis of MRD as well as parents' receipt of nursing home care to shed light on the extent to which individuals receive signals of their risk via their parents. Results corresponding to columns (4) and (9) of Table 5 are presented in Table 10.[17]

In columns (1) and (2) of Table 10 we show results where the outcome is the self-reported probability of living to age 75, which is asked to those aged 50–65. The point estimate on the AD score is positive, but standard errors are large. APOE carriers report lower probabilities, but none of the coefficients on the APOE dummies reach statistical significance. A one standard deviation rise in the EA score is associated with a statistically significant 0.5 percentage point increase in the probability of living to age 75. In columns (3) and (4), we consider the self-reported probability of using a nursing home in the next five years, which is only asked to those aged 65 and older and not currently residing in a nursing home. We focus on those aged 65–70 to align with the samples used in our analyses of planning activities. The point estimate on the AD score is negative, but not estimated precisely. Those with at least one copy of the APOE allele report a statistically significant 0.7–0.8 percentage point increase in the probability of future nursing use, from a mean of 11%. The coefficient on the indicator for having exactly two copies of APOE is very imprecisely estimated, and negative for the never diagnosed. Given the question about future nursing home use is only asked to those not currently in a nursing home and not interviewed by proxy, 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, consistent

[17]The full set of results for each outcome are presented in Appendix Tables A18–A23.

with the idea that some individuals are aware of their genetic risk and incorporate that knowledge into their assessment of future long-term care needs.

We next combine responses to questions from various experimental modules and examine whether and how genes predict one's self-reported probability of developing AD or dementia in the future. This analysis is under-powered as experimental modules were fielded to small subsamples of the HRS and we require that these individuals be genotyped and aged 50–70. Results are presented in columns (5) and (6) of Table 10. Those who carry at least one copy of APOE report a statistically significant 7 percentage point increase in their self-reported probability of developing AD. The coefficient on carrying exactly two copies is negative and very imprecisely estimated. Across the specifications, the coefficient on the AD score is negative, but not precisely estimated. In two of these experimental modules, those who had been diagnosed with MRD or currently reside in a nursing home were not asked these questions, and we impose that sample restriction throughout this analysis. Thus, this sample is also positively selected on those who are not yet diagnosed and do not need institutional care, which may make these estimates conservative. We also find that a one standard deviation increase in the EA score is associated with a 3 percentage point increase in the self-reported probability of developing AD in the future.[18]

Taken together, these results suggest that some APOE carriers are aware of their elevated risk status, while those with higher AD scores are not. It is possible that APOE carriers receive stronger signals of their risk if their parents experience cognitive decline and are diagnosed with AD. We examine whether those aged 50–70 have had a parent diagnosed with MRD, and we drop individuals after their first report of a parent being diagnosed. The results are presented in columns (7) and (8) in Table 10. Carrying at least one copy of APOE associates with a 2 percentage point increase in the probability that a parent was ever diagnosed with an MRD. We also find that a one standard deviation increase in the AD score is associated with a 0.4–0.5 percentage point increase in the probability of parental MRD. The question about whether a parent has a memory-related disease was not asked until 1998 and is only asked if the parent is currently alive. Thus, we likely understate parental diagnosis of MRD as some individuals with parents who passed away before the 1998 wave may have had an MRD, which will make these estimates conservative.

We next examine how genetic risk correlates with parents' receipt of nursing home care. The sample includes individuals as of the last time we observe them in the 50–70 age range, and the outcome is an indicator for whether either of the respondent's parents used a nursing home by that point. We include only one observation per individual here because information about parental

[18] In Appendix Table A21, we replace the self-reported probability with an indicator for whether the individual reports a 50% or greater chance of developing AD or dementia in the future. We again find that those who carry at least one copy of the APOE allele are more likely to report a higher probability of developing AD.

nursing home use is gleaned from both a question about where parents who are currently alive reside as well as questions about whether parents who have passed away used a nursing home before death. It is not uncommon for at least one parent to be deceased at a respondent's first interview and to observe little within-person variation in this measure. The results in columns (9) and (10) in Table 10 suggest that those carrying two copies of APOE are 15 percentage points more likely to have had a parent use a nursing home. While individuals may use a nursing home for a variety of reasons, many individuals with advanced cognitive impairment or dementia are likely to live in a nursing home toward the end of their lives. Our estimates are therefore consistent with those at particularly high risk of developing AD being more likely to have had a parent whose cognitive impairment led to a nursing-home-level of care needs.

Taken together, these results imply that APOE carriers are more aware of their elevated risk, perhaps due to witnessing their own parents' decline. Those with higher AD scores do not seem to be aware of their elevated risk, which could explain why they do not engage in planning activities that would shield them and their families from subsequent economic losses and that would communicate their preferences about the management of their future health care.

6 Conclusion

We explore how genetic endowments related to Alzheimer's disease associate with cognitive function, diagnosis of a memory-related disease, economic outcomes, later-life planning activities, and awareness of one's risk of cognitive decline. An overarching question motivating and guiding our analysis is why individuals at similar risk of AD exhibit variation in diagnosis. Put another way, why do some people at high genetic risk of AD lack a diagnosis? Genetic risk is not deterministic, so perhaps such individuals are protected from cognitive decline, which would obviate the need for diagnosis. Alternatively, these individuals may exhibit cognitive decline, but are insulated from some of its consequences, perhaps because they have the resources and support to manage some of the worst outcomes. We find evidence of neither. Undiagnosed individuals at high genetic risk for AD are neither protected from cognitive decline, nor insulated from the economic consequences we measure (and surely many we cannot). Thus, it is likely that there is a large population of people who are under-diagnosed, under-treated, and under-resourced, which genetic data allow us to observe.

Because we examine genetic risk, we are able to address a number of questions. First, we provide evidence that there is potential clinical value in genetic variables since they predict cognitive decline even after controlling for a host of standard measures typically used to identify it. There are practical reasons to use genetic risk rather than, say, financial behavior as the latter would be difficult to track, and would also emerge once decline is underway and has begun to

have costly negative consequences. Second, we ask whether individuals at high AD genetic risk engage in activities that are protective, i.e., that could mitigate the consequences of eventual cognitive decline. A subset of high-risk individuals, namely those with higher AD polygenic scores, are less likely to do so compared to individuals who are not at high risk of cognitive decline, likely, as we demonstrate, because they are unaware of their risk. This finding also has practical implications. While a reasonable goal is to improve treatment and prevention of cognitive decline, an immediate policy step could be to improve access to straightforward, available, and scalable planning activities that limit the worst consequences, e.g., by promoting estate planning.

Our study raises a host of new questions and we highlight two that we believe are clear next steps for future research. First, our study suggests that there are many people who are undiagnosed, but could be based on their cognitive function. Yet, much of what we know about AD is based on populations of diagnosed individuals, both in the clinical setting, but also in the social sciences, which tends to compare "eventually diagnosed" individuals to people who are never diagnosed. Drawing conclusions in this way is problematic since such methods essentially use those who select into diagnosis as the "treatment" group and those who are never diagnosed as a "control" group. Selection into treatment can bias parameters used to draw conclusions as they could reflect myriad unobserved factors that are not directly due to illness; moreover, the individuals in the control group may suffer from illness that has simply gone undetected. Future research could assess whether lessons learned about AD using diagnosed (or eventually diagnosed) individuals are consistent with what we might learn by examining undiagnosed people with a high AD genetic risk. If not, a new focus on the undiagnosed may be a useful path forward in studies of AD. Second, and relatedly, are there environmental factors that help us understand why people with similar genetic risk for AD exhibit very different clinical and economic outcomes? If certain environmental factors systematically predict better or worse outcomes for people with similar genetic risk, they may prompt questions about potential future treatment or care. This is especially true if we consider differences in cognitive decline for a given level of genetic risk.[19] There may be people who are at risk of AD and might have exhibited cognitive decline under different circumstances or under different environments. We miss such lessons since they are unlikely to be diagnosed precisely because they do not need to be. Future research to further our understanding of the genetic architecture of AD, including its interactions with cognition and economic outcomes, can help to shed light on such possibilities.

[19] In results available upon request from the authors, we have conducted a series of simple gene-by-environment ($G \times E$) analyses to assess whether certain environments when interacted with certain genetic risks predict outcomes, ranging from decline and diagnosis to income and wealth. We have examined many environmental factors, including childhood SES and family structure. We generally find the interaction coefficients are noisily estimated. Thus, the project of detecting whether certain environments moderate or exacerbate how genes related to AD predict outcomes of interest has not yielded conclusive results.

References

Agarwal, S. and B. Mazumder (2013): "Cognitive abilities and household financial decision making," *American Economic Journal: Applied Economics*, 5, 193–207.

Alzheimer's Association (2022): "2022 Alzheimer's disease facts and figures," *Alzheimer's & Dementia*, 18, 700–789.

Ameriks, J., A. Caplin, M. Lee, M. D. Shapiro, and C. Tonetti (2023): "Cognitive Decline, Limited Awareness, Imperfect Agency, and Financial Well-Being," *American Economic Review: Insights*, 5, 125–140.

Barth, D., N. W. Papageorge, and K. Thom (2020): "Genetic endowments and wealth inequality," *Journal of Political Economy*, 128, 1474–1522.

Beauchamp, J. P., D. Cesarini, M. Johannesson, M. J. van der Loos, P. D. Koellinger, P. J. Groenen, J. H. Fowler, J. N. Rosenquist, A. R. Thurik, and N. A. Christakis (2011): "Molecular genetics and economics," *Journal of Economic Perspectives*, 25, 57–82.

Belsky, D. W., T. E. Moffitt, D. L. Corcoran, B. Domingue, H. Harrington, S. Hogan, R. Houts, S. Ramrakha, K. Sugden, B. S. Williams, et al. (2016): "The genetics of success: How single-nucleotide polymorphisms associated with educational attainment relate to life-course development," *Psychological science*, 27, 957–972.

Benjamin, D. J., D. Cesarini, C. F. Chabris, E. L. Glaeser, D. I. Laibson, V. Age, Gudnason, T. B. Harris, L. J. Launer, S. Purcell, et al. (2012): "The promises and pitfalls of genoeconomics," *Annual Review of Economics*, 4, 627–662.

Bolyard, A. and P. A. Savelyev (2021): "Understanding the educational attainment polygenic score and its interactions with SES in determining health in young adulthood," SSRN Working Paper 3397735.

Chandra, A., C. Coile, and C. Mommaerts (2023): "What Can Economics Say about Alzheimer' s Disease?" *Journal of Economic Literature*, 61, 428–470.

Christelis, D., T. Jappelli, and M. Padula (2010): "Cognitive abilities and portfolio choice," *European Economic Review*, 54, 18–38.

Coe, N. B., M. M. Skira, and E. B. Larson (2018): "A comprehensive measure of the costs of caring for a parent: Differences according to functional status," *Journal of the American Geriatrics Society*, 66, 2003–2008.

Crimmins, E. M., J. K. Kim, K. M. Langa, and D. R. Weir (2011): "Assessment of cognition using surveys and neuropsychological assessment: The Health and Retirement Study and the Aging, Demographics, and Memory Study," *Journals of Gerontology Series B: Psychological Sciences and Social Sciences*, 66, i162–i171.

Escott-Price, V. and J. Hardy (2022): "Genome-wide association studies for Alzheimer's disease: Bigger is not always better," *Brain Communications*, 4, fcac125.

Friedman, E. M., R. A. Shih, K. M. Langa, and M. D. Hurd (2015): "US prevalence and predictors of informal caregiving for dementia," *Health Affairs*, 34, 1637–1641.

Gatz, M., C. A. Reynolds, L. Fratiglioni, B. Johansson, J. A. Mortimer, S. Berg, A. Fiske, and N. L. Pedersen (2006): "Role of Genes and Environments for Explaining Alzheimer Disease," *Archives of General Psychiatry*, 63, 168–174.

Gianattasio, K. Z., Q. Wu, M. M. Glymour, and M. C. Power (2019): "Comparison of methods for algorithmic classification of dementia status in the Health and Retirement Study," *Epidemiology (Cambridge, Mass.)*, 30, 291.

Gresenz, C. R., J. M. Mitchell, J. Marrone, and H. J. Federoff (2020): "Effect of early-stage Alzheimer's disease on household financial outcomes," *Health Economics*, 29, 18–29.

Hsu, J. W. and R. Willis (2013): "Dementia risk and financial decision making by older households: The impact of information," *Journal of Human Capital*, 7, 340–377.

Hurd, M. D., P. Martorell, A. Delavande, K. J. Mullen, and K. M. Langa (2013): "Monetary costs of dementia in the United States," *New England Journal of Medicine*, 368, 1326–1334.

Kunkle, B. W., B. Grenier-Boley, R. Sims, J. C. Bis, V. Damotte, A. C. Naj, A. Boland, M. Vronskaya, S. J. Van Der Lee, A. Amlie-Wolf, et al. (2019): "Genetic meta-analysis of diagnosed Alzheimer's disease identifies new risk loci and implicates Aβ, tau, immunity and lipid processing," *Nature Genetics*, 51, 414–430.

Langa, K. M., M. E. Chernew, M. U. Kabeto, A. Regula Herzog, M. Beth Ofstedal, R. J. Willis, R. B. Wallace, L. M. Mucha, W. L. Straus, and A. M. Fendrick (2001): "National estimates of the quantity and cost of informal caregiving for the elderly with dementia," *Journal of general internal medicine*, 16, 770–778.

Lee, J. J., R. Wedow, A. Okbay, E. Kong, O. Maghzian, M. Zacher, T. A. Nguyen-Viet, P. Bowers, J. Sidorenko, R. Karlsson Linnér, et al. (2018): "Gene discovery and polygenic prediction from a genome-wide association study of educational attainment in 1.1 million individuals," *Nature Genetics*, 50, 1112–1121.

Li, J., A. Kelley, K. McGarry, L. H. Nicholas, and J. Skinner (2022): "Dementia and Long-run Trajectories in Household Finances," Working Paper.

Linnér, R. K. and P. D. Koellinger (2022): "Genetic risk scores in life insurance underwriting," *Journal of Health Economics*, 81, 102556.

Liu, C.-C., T. Kanekiyo, H. Xu, and G. Bu (2013): "Apolipoprotein E and Alzheimer disease: Risk, mechanisms and therapy," *Nature Reviews Neurology*, 9, 106–118.

Martin, R. C., A. Gerstenecker, K. L. Triebel, M. Falola, T. McPherson, G. Cutter, and D. C. Marson (2019): "Declining financial capacity in mild cognitive impairment: A six-year longitudinal study," *Archives of Clinical Neuropsychology*, 34, 152–161.

Mazzonna, F. and F. Peracchi (forthcoming): "Are Older People Aware of Their Cognitive Decline? Misperception and Financial Decision Making," *Journal of Political Economy*.

Michaelson, D. M. (2014): "APOE $\varepsilon 4$: The most prevalent yet understudied risk factor for Alzheimer's disease," *Alzheimer's & Dementia*, 10, 861–868.

Nicholas, L. H., K. M. Langa, J. P. Bynum, and J. W. Hsu (2021): "Financial presentation of Alzheimer disease and related dementias," *JAMA internal medicine*, 181, 220–227.

Papageorge, N. W. and K. Thom (2020): "Genes, education, and labor market outcomes: Evidence from the Health and Retirement Study," *Journal of the European Economic Association*, 18, 1351–1399.

Scharre, D. W. (2019): "Preclinical, prodromal and dementia stages of Alzheimer's disease," *Practical Neurology*, 19, L36–47.

Shin, S. H., D. R. Lillard, and J. Bhattacharya (2019): "Understanding the correlation between Alzheimer's Disease polygenic risk, wealth, and the composition of wealth holdings," *Biodemography and Social Biology*, 65, 323–350.

Stern, Y. (2012): "Cognitive reserve in ageing and Alzheimer's disease," *The Lancet Neurology*, 11, 1006–1012.

Sudo, F. K. and J. Laks (2017): "Financial capacity in dementia: A systematic review," *Aging & Mental Health*, 21, 677–683.

Triebel, K., R. Martin, H. Griffith, J. Marceaux, O. Okonkwo, L. Harrell, D. Clark, J. Brockington, A. Bartolucci, and D. C. Marson (2009): "Declining financial capacity in mild cognitive impairment: A 1-year longitudinal study," *Neurology*, 73, 928–934.

Villemagne, V. L., S. Burnham, P. Bourgeat, B. Brown, K. A. Ellis, O. Salvado, C. Szoeke, S. L. Macaulay, R. Martins, P. Maruff, et al. (2013): "Amyloid β deposition, neurodegeneration, and cognitive decline in sporadic Alzheimer's disease: A prospective cohort study," *The Lancet Neurology*, 12, 357–367.

Ware, E. B., J. D. Faul, C. M. Mitchell, and K. M. Bakulski (2020): "Considering the APOE locus in Alzheimer's disease polygenic scores in the Health and Retirement Study: A longitudinal panel study," *BMC Medical Genomics*, 13, 1–13.

Zissimopoulos, J., E. Crimmins, and P. S. Clair (2015): "The value of delaying Alzheimer's disease onset," in *Forum for Health Economics and Policy*, De Gruyter, vol. 18, 25–39.

Tables and Figures

Table 1: Summary Statistics

	Mean	SD	Ages	N
Demographics:				
Birth Year	1940.222	9.782	50–85	88,048
Male	0.419	0.493	50–85	88,048
Age	67.553	9.000	50–85	88,048
Years of Education	13.315	2.499	50–85	88,048
At Least Some College Degree	0.322	0.467	50–85	88,048
Cognition and Memory-Related Disease (MRD):				
Langa-Weir (L-W) Score	16.392	4.004	50–85	88,048
Ever Demented (L-W < 7)	0.028	0.165	50–85	88,048
Ever Impaired or Demented (L-W < 12)	0.217	0.412	50–85	88,048
Ever Diagnosed with MRD	0.021	0.144	50–85	88,048
Genetic Data:				
AD Score	-0.009	1.000	50–85	88,048
APOE (At least 1 copy)	0.260	0.439	50–85	88,048
APOE (2 copies)	0.020	0.138	50–85	88,048
EA Score	0.007	0.996	50–85	88,048
Economic Outcomes:				
Work for Pay	0.563	0.496	50–70	53,592
Retired	0.320	0.467	50–70	49,564
Log Individual Total Income	10.010	0.913	50–85	81,393
Log Household Total Wealth	12.400	1.599	50–85	84,548
Planning Outcomes:				
Holds Long-Term Care Insurance (LTCI)	0.126	0.332	50–70	52,747
Holds Life Insurance (LI)	0.714	0.452	50–70	53,322
Has a Witnessed Will	0.559	0.497	50–70	53,484
Has a Living Will	0.478	0.500	65–70	5,066
Has Assigned Someone Durable Power of Attorney for Health Care	0.462	0.499	65–70	5,067
Discuss Future Medical Care with Anyone	0.590	0.492	65–70	3,500
Awareness Outcomes:				
Probability of Living to Age 75	66.275	26.389	50–65	36,282
Probability of Moving to Nursing Home	10.982	17.207	65–70	16,795
Probability of Developing Alzheimer's Disease	36.303	26.128	50–70	660
Probability of Parent Ever Diagnosed with MRD	0.277	0.447	50–70	29,657
Probability of Parent Receiving Nursing Home Care	0.379	0.485	50–70	4,344

Note: The table presents summary statistics at the person-wave level from 1998–2018. Questions about living will, durable power of attorney, and discussing future medical care were asked of those ages 65 and older from 2012 on. Expectations about moving to a nursing home in the next 5 years were only asked to those ages 65 and older. Parental nursing home care use is measured as of the latest observation for a respondent between the ages 50–70.

Table 2: Relationship between Genetic Endowments and Cognition

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	Full Sample				Never Diagnosed		
	Panel A: L-W Score						
AD Score	-0.236***	-0.206***	-0.130***	-0.111***	-0.225***	-0.196***	-0.122***
	(0.035)	(0.034)	(0.031)	(0.030)	(0.036)	(0.035)	(0.031)
APOE (At least 1 copy)	-0.404***	-0.397***	-0.402***	-0.292***	-0.267***	-0.261***	-0.263***
	(0.065)	(0.063)	(0.056)	(0.055)	(0.066)	(0.065)	(0.058)
APOE (2 copies)	-0.558**	-0.509**	-0.523***	-0.350*	-0.391	-0.366	-0.292
	(0.218)	(0.213)	(0.193)	(0.186)	(0.239)	(0.233)	(0.201)
EA Score		0.630***	0.256***	0.252***		0.614***	0.243***
		(0.027)	(0.025)	(0.025)		(0.028)	(0.026)
Ever MRD				-2.130***			
				(0.191)			
Eventual MRD				-1.522***			
				(0.093)			
Education Controls	No	No	Yes	Yes	No	No	Yes
Mean	16.392	16.392	16.392	16.392	16.648	16.648	16.648
N	88,048	88,048	88,048	88,048	80,264	80,264	80,264
R^2	0.133	0.156	0.251	0.275	0.118	0.141	0.240
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
	Panel B: Ever Impaired or Demented (L-W Score < 12)						
AD Score	0.006***	0.006***	0.005***	0.004***	0.006***	0.005***	0.004***
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
APOE (At least 1 copy)	0.013***	0.013***	0.013***	0.009***	0.007***	0.007***	0.007***
	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)
APOE (2 copies)	0.022***	0.021***	0.023***	0.015**	0.017**	0.017**	0.017**
	(0.007)	(0.007)	(0.007)	(0.007)	(0.007)	(0.007)	(0.007)
EA Score		-0.014***	-0.007***	-0.007***		-0.013***	-0.006***
		(0.001)	(0.001)	(0.001)		(0.001)	(0.001)
Ever MRD				0.077***			
				(0.017)			
Eventual MRD				0.066***			
				(0.005)			
Education Controls	No	No	Yes	Yes	No	No	Yes
Mean	0.054	0.054	0.054	0.054	0.047	0.047	0.047
N	72,823	72,823	72,823	72,823	67,631	67,631	67,631
R^2	0.022	0.026	0.047	0.056	0.018	0.022	0.042
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

Note: Each column within a panel presents results from a separate regression. In Panel A, the outcome is the Langa-Weir (L-W) cognition score, which ranges from 0–27. In Panel B, the outcome is an indicator for an L-W score below 12 in the current wave, and the sample excludes individuals after their first observed L-W score below 12. 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 (6), we add the educational attainment (EA) polygenic score. In columns (3) and (7), we add controls for educational attainment. In column (4), we add controls for whether the respondent has ever been diagnosed with a memory-related disease (MRD) and whether they will eventually report diagnosis (in subsequent survey waves). Columns (5)-(7) are estimated only on those never diagnosed with an MRD during the period they are observed. 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 Endowments and Memory-Related Disease Diagnosis

	Ever MRD Diagnosis		
	(1)	(2)	(3)
AD Score	0.001***	0.001***	0.001***
	(0.000)	(0.000)	(0.000)
APOE (At least 1 copy)	0.005***	0.005***	0.005***
	(0.001)	(0.001)	(0.001)
APOE (2 copies)	0.010***	0.010***	0.009***
	(0.003)	(0.003)	(0.003)
EA Score		-0.001**	-0.000
		(0.000)	(0.000)
Education Controls	No	No	Yes
Mean	0.007	0.007	0.007
N	86,747	86,747	86,747
R^2	0.008	0.008	0.009
Years	1998-2018	1998-2018	1998-2018
Ages	50-85	50-85	50-85

Note: Each column presents results from a separate regression where the outcome is an indicator for whether the individual has ever been diagnosed with a memory-related disease (MRD). The sample excludes individuals after their first report of an MRD diagnosis. 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 the educational attainment (EA) polygenic score. In column (3), we add controls for educational attainment. Standard errors are clustered at the individual level. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table 4: Relationship between Genetic Endowments and Future (Age 65–80) Cognitive Outcomes

	(1) Ever Impaired	(2) Ever Demented	(3) Ever Diagnosed
AD Score	0.022***	0.007**	0.013***
	(0.007)	(0.003)	(0.004)
APOE (At least 1 copy)	0.062***	0.028***	0.042***
	(0.013)	(0.007)	(0.008)
APOE (2 copies)	0.126***	0.046	0.093**
	(0.043)	(0.029)	(0.037)
EA Score	-0.009	0.002	0.003
	(0.006)	(0.003)	(0.003)
Mean	0.203	0.028	0.040
N	4860	4860	4871
R^2	0.231	0.074	0.066

Note: Each column presents results from a separate regression with the outcome listed in the column header. In column (1) the outcome is an indicator for an individual being impaired (L-W score < 12) in the age range 65–80. In column (2) the outcome is an indicator for being demented (L-W score < 7) in this age range, and in column (3) the outcome is an indicator for ever being diagnosed with a memory-related disease in this age range. We restrict the sample to individuals who were not impaired, demented, or diagnosed in the age range 50–64. In all specifications, we control for the first 10 principal components of the genetic data, birth year, dummies for years of schooling, dummies for degree attained, and a complete set of interactions between these variables and a male indicator. We also control for the average L-W score observed for ages 50–64, as well as a complete set of dummies for the minimum L-W score and maximum L-W score observed over this age range. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table 5: Relationship between Genetic Endowments and Currently Working for Pay

	Currently Working for Pay								
	Full Sample					Never Diagnosed			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
AD Score	-0.017***	-0.015***	-0.011**	-0.010**	-0.009*	-0.016***	-0.014***	-0.010**	-0.009*
	(0.005)	(0.005)	(0.005)	(0.005)	(0.005)	(0.005)	(0.005)	(0.005)	(0.005)
APOE (At least 1 copy)	0.005	0.006	0.005	0.007	0.009	0.005	0.006	0.004	0.005
	(0.009)	(0.009)	(0.009)	(0.009)	(0.009)	(0.009)	(0.009)	(0.009)	(0.009)
APOE (2 copies)	-0.011	-0.009	-0.008	-0.004	0.004	0.014	0.015	0.022	0.024
	(0.026)	(0.026)	(0.027)	(0.026)	(0.025)	(0.027)	(0.028)	(0.028)	(0.028)
EA Score		0.040***	0.019***	0.016***	0.015***		0.038***	0.016***	0.014***
		(0.004)	(0.004)	(0.004)	(0.004)		(0.004)	(0.004)	(0.004)
Ever MRD					-0.282***				
					(0.030)				
Eventual MRD					-0.062***				
					(0.018)				
L-W Score Dummies	No	No	No	Yes	Yes	No	No	No	Yes
Education Controls	No	No	Yes	Yes	Yes	No	No	Yes	Yes
Mean	0.563	0.563	0.563	0.563	0.563	0.578	0.578	0.578	0.578
N	53,592	53,592	53,592	53,592	53,592	50,485	50,485	50,485	50,485
R^2	0.151	0.157	0.177	0.184	0.191	0.154	0.159	0.180	0.185
Years	1998-2018	1998-2018	1998-2018	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	50-70	50-70	50-70

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 columns (2) and (7), we add the educational attainment (EA) polygenic score. In columns (3) and (8), we add controls for educational attainment. In columns (4) and (9), we add dummy variables for each value of the most current L-W score. In column (5), we add controls for whether the respondent has ever been diagnosed with a memory-related disease (MRD) and whether they will eventually report diagnosis (in subsequent survey waves). Columns (6)-(9) are estimated only on those never diagnosed with an MRD during the period they are observed. Standard errors are clustered at the individual level. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table 6: Relationship between Genetic Endowments and Employment, Income, and Wealth

	Currently Working for Pay		Retirement		Log Individual Total Income		Log Household Total Wealth	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
AD Score	-0.010**	-0.009*	0.007	0.007	-0.014*	-0.008	-0.039**	-0.030*
	(0.005)	(0.005)	(0.004)	(0.004)	(0.007)	(0.008)	(0.016)	(0.016)
APOE (At least 1 copy)	0.007	0.005	-0.007	-0.005	-0.006	-0.004	0.002	-0.002
	(0.009)	(0.009)	(0.008)	(0.008)	(0.013)	(0.015)	(0.029)	(0.031)
APOE (2 copies)	-0.004	0.024	0.030	0.015	0.009	0.041	0.106	0.041
	(0.026)	(0.028)	(0.024)	(0.026)	(0.039)	(0.042)	(0.093)	(0.105)
EA Score	0.016***	0.014***	-0.014***	-0.013***	0.011*	0.008	0.096***	0.099***
	(0.004)	(0.004)	(0.004)	(0.004)	(0.006)	(0.006)	(0.013)	(0.014)
L-W Score Dummies	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Education Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Sample	All	Never MRD	All	Never MRD	All	Never MRD	All	Never MRD
Mean	0.563	0.578	0.320	0.309	10.010	10.036	12.400	12.415
N	53,592	50,485	49,564	46,907	81,393	73,999	84,548	77,110
R^2	0.184	0.185	0.221	0.224	0.274	0.273	0.180	0.177
Years	1998-2018	1998-2018	1998-2018	1998-2018	1998-2018	1998-2018	1998-2018	1998-2018
Ages	50-70	50-70	50-70	50-70	50-85	50-85	50-85	50-85

Note: Each column presents results from a separate regression. In columns (1) and (2), the outcome is an indicator for whether the respondent currently works for pay. In columns (3) and (4), 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 columns (5) and (6), the outcome is logged total individual income. In columns (7) and (8), the outcome is logged household wealth. 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 as well as the educational attainment (EA) polygenic score, dummy variables for educational attainment, and 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 7: Relationship between Genetic Endowments and Log Household Wealth by Gender

	Log Household Total Wealth					
	Males		Females		Married Females	
	(1)	(2)	(3)	(4)	(5)	(6)
AD Score	-0.024	-0.011	-0.049**	-0.044**	-0.048**	-0.043*
	(0.023)	(0.024)	(0.021)	(0.022)	(0.021)	(0.022)
APOE (At least 1 copy)	-0.018	-0.040	0.018	0.027	0.038	0.071*
	(0.042)	(0.044)	(0.039)	(0.042)	(0.039)	(0.040)
APOE (2 copies)	-0.043	-0.139	0.220*	0.171	0.136	0.098
	(0.143)	(0.161)	(0.119)	(0.135)	(0.130)	(0.145)
EA Score	0.085***	0.091***	0.104***	0.105***	0.080***	0.075***
	(0.019)	(0.020)	(0.019)	(0.019)	(0.019)	(0.020)
L-W Score Dummies	Yes	Yes	Yes	Yes	Yes	Yes
Education Controls	Yes	Yes	Yes	Yes	Yes	Yes
Sample	All	Never MRD	All	Never MRD	All	Never MRD
Mean	12.543	12.558	12.296	12.312	12.694	12.702
N	35,569	32,298	48,979	44,812	30,554	28,165
R^2	0.196	0.192	0.164	0.162	0.179	0.177
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

Note: Each column presents results from a separate regression where the outcome is logged household wealth. In columns (1) and (2), the sample consists of men. In columns (3) and (4), the sample consists of women. In columns (5) and (6), the sample consists of married women. 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 as well as the educational attainment (EA) polygenic score, dummy variables for educational attainment, and dummy variables for each value of the most current L-W score. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table 8: Relationship between Genetic Endowments and Later-Life Planning

	(1)	(2)	(3)	(4)	(5)	(6)
Panel A:	Holds Long-Term Care Insurance		Holds Life Insurance		Has a Witnessed Will	
AD Score	-0.010***	-0.009**	-0.006	-0.003	-0.013**	-0.012**
	(0.004)	(0.004)	(0.005)	(0.005)	(0.006)	(0.006)
APOE (At least 1 copy)	0.006	0.007	-0.000	0.001	0.013	0.015
	(0.007)	(0.007)	(0.009)	(0.009)	(0.010)	(0.011)
APOE (2 copies)	-0.000	-0.006	-0.006	-0.027	-0.005	-0.009
	(0.019)	(0.020)	(0.026)	(0.028)	(0.031)	(0.033)
EA Score	0.001	-0.001	0.004	0.003	0.017***	0.017***
	(0.003)	(0.003)	(0.004)	(0.004)	(0.005)	(0.005)
L-W Score Dummies	Yes	Yes	Yes	Yes	Yes	Yes
Education Controls	Yes	Yes	Yes	Yes	Yes	Yes
Sample	All	Never MRD	All	Never MRD	All	Never MRD
Mean	0.126	0.126	0.714	0.716	0.559	0.558
N	52,747	49,706	53,322	50,233	53,484	50,389
R^2	0.045	0.046	0.055	0.057	0.134	0.133
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
Panel B:	Has a Living Will		Has Assigned Someone Durable Power of Attorney for Healthcare		Discussed Future Medical Care with Anyone	
AD Score	-0.018	-0.016	-0.038***	-0.038***	-0.025**	-0.026**
	(0.012)	(0.012)	(0.012)	(0.012)	(0.011)	(0.011)
APOE (At least 1 copy)	0.033	0.041*	0.015	0.017	-0.006	0.003
	(0.022)	(0.023)	(0.022)	(0.023)	(0.019)	(0.020)
APOE (2 copies)	0.063	0.060	0.057	0.092	-0.026	-0.014
	(0.062)	(0.064)	(0.063)	(0.064)	(0.061)	(0.065)
EA Score	0.013	0.013	0.013	0.012	-0.005	-0.005
	(0.010)	(0.010)	(0.010)	(0.010)	(0.009)	(0.009)
L-W Score Dummies	Yes	Yes	Yes	Yes	Yes	Yes
Education Controls	Yes	Yes	Yes	Yes	Yes	Yes
Sample	All	Never MRD	All	Never MRD	All	Never MRD
Mean	0.478	0.476	0.462	0.461	0.590	0.592
N	5,066	4,850	5,067	4,852	3,500	3,347
R^2	0.100	0.104	0.097	0.100	0.126	0.126
Years	2012-2018	2012-2018	2012-2018	2012-2018	2012-2018	2012-2018
Ages	65-70	65-70	65-70	65-70	65-70	65-70

Note: Each column within a panel presents results from a separate regression where the outcome is described in the column heading. 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 as well as the educational attainment (EA) polygenic score, dummy variables for educational attainment, and dummy variables for each value of the most current L-W score. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table 9: Relationship between Genetic Endowments and Number of Later-Life Planning Activities

	Probability of Number of Planning Activities													
	≥ 1		≥ 2		≥ 3		≥ 4		≥ 5		≥ 6		Count (0-6)	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)
AD Score	0.002	0.005	-0.006	-0.002	-0.037***	-0.033***	-0.024**	-0.022**	-0.015*	-0.015*	-0.008	-0.006	-0.088**	-0.072*
	(0.007)	(0.007)	(0.011)	(0.011)	(0.012)	(0.012)	(0.011)	(0.011)	(0.009)	(0.009)	(0.005)	(0.005)	(0.039)	(0.039)
APOE (At least 1 copy)	-0.001	0.004	0.016	0.023	0.041*	0.051**	0.022	0.028	0.005	0.011	0.011	0.014	0.094	0.132*
	(0.012)	(0.012)	(0.020)	(0.021)	(0.022)	(0.022)	(0.020)	(0.021)	(0.016)	(0.017)	(0.009)	(0.009)	(0.073)	(0.074)
APOE (2 copies)	0.040	0.032	0.027	0.018	0.041	0.009	0.055	0.066	-0.012	0.015	0.018	0.014	0.170	0.153
	(0.029)	(0.033)	(0.064)	(0.068)	(0.068)	(0.074)	(0.065)	(0.070)	(0.049)	(0.053)	(0.030)	(0.031)	(0.218)	(0.237)
EA Score	0.007	0.006	0.025**	0.025**	0.006	0.007	0.007	0.008	0.004	0.004	0.002	0.003	0.051	0.053
	(0.006)	(0.006)	(0.010)	(0.010)	(0.010)	(0.010)	(0.009)	(0.010)	(0.007)	(0.008)	(0.004)	(0.004)	(0.034)	(0.034)
L-W Score Dummies	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Education Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Sample	All	Never MRD	All	Never MRD	All	Never MRD	All	Never MRD	All	Never MRD	All	Never MRD	All	Never MRD
Mean	0.918	0.921	0.710	0.711	0.493	0.493	0.352	0.350	0.193	0.192	0.047	0.048	2.713	2.714
N	3,411	3,261	3,411	3,261	3,411	3,261	3,411	3,261	3,411	3,261	3,411	3,261	3,411	3,261
R^2	0.114	0.104	0.109	0.109	0.127	0.131	0.112	0.119	0.085	0.086	0.071	0.069	0.154	0.157
Years	2012-2018	2012-2018	2012-2018	2012-2018	2012-2018	2012-2018	2012-2018	2012-2018	2012-2018	2012-2018	2012-2018	2012-2018	2012-2018	2012-2018
Ages	65-70	65-70	65-70	65-70	65-70	65-70	65-70	65-70	65-70	65-70	65-70	65-70	65-70	65-70

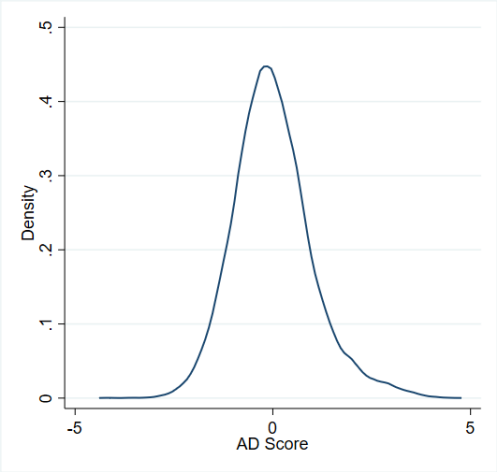
Note: In columns (1)–(12), each column presents results from a separate regression where the outcome is an indicator variable for engaging in at least a certain number of planning activities. In columns (13)–(14), the outcome is the total number of planning activities the respondent engages in. The sample is limited to those who provided an answer to all 6 planning activity questions. 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 as well as the educational attainment (EA) polygenic score, dummy variables for educational attainment, and dummy variables for each value of the most current L-W score. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table 10: Relationship between Genetic Endowments and Awareness of Risk

	Probability of Living to Age 75		Probability of Moving to Nursing Home in Next 5 Years		Probability of Developing Alzheimer's Disease		Parental Diagnosis of MRD		Parental Use of Nursing Home Care	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
AD Score	0.143	0.277	-0.060	-0.106	-0.288	-0.498	0.004*	0.005**	0.002	0.004
	(0.305)	(0.312)	(0.212)	(0.217)	(1.570)	(1.631)	(0.002)	(0.002)	(0.009)	(0.010)
APOE (At least 1 copy)	-0.665	-0.759	0.672*	0.785*	7.404***	7.297***	0.023***	0.022***	0.029	0.029
	(0.558)	(0.570)	(0.389)	(0.407)	(2.580)	(2.668)	(0.005)	(0.005)	(0.018)	(0.018)
APOE (2 copies)	-0.587	-0.356	0.034	-1.900	-5.681	-5.645	0.001	0.009	0.118**	0.117**
	(1.889)	(2.044)	(1.347)	(1.394)	(8.021)	(8.829)	(0.014)	(0.016)	(0.051)	(0.053)
EA Score	0.541**	0.542**	0.040	-0.014	3.036**	3.331***	-0.002	-0.001	0.014*	0.014
	(0.258)	(0.263)	(0.179)	(0.184)	(1.207)	(1.237)	(0.002)	(0.002)	(0.008)	(0.008)
L-W Score Dummies	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Education Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Sample	All	Never MRD	All	Never MRD	All	Never MRD	All	Never MRD	All	Never MRD
Mean	66.275	66.645	10.982	10.847	36.303	36.105	0.070	0.070	0.379	0.377
N	36,282	34,611	16,795	15,426	660	638	23,065	22,041	4,344	4,200
R^2	0.085	0.085	0.023	0.023	0.345	0.348	0.031	0.030	0.083	0.085
Years	1998-2018	1998-2018	1998-2018	1998-2018	2002, 2012, 2016	2002, 2012, 2016	1998-2018	1998-2018	2006-2018	2006-2018
Ages	50-65	50-65	65-70	65-70	50-70	50-70	50-70	50-70	50-70	50-70

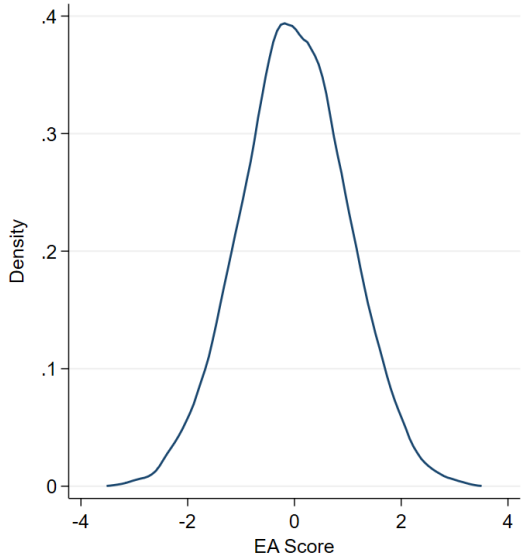
Note: Each column within a panel presents results from a separate regression where the outcome is described in the column heading. In all specifications, we control for the first 10 principal components of the genetic data as well as the standard controls for age, birth cohort, sex of respondent, and survey year as described in Section 4 as well as the educational attainment (EA) polygenic score, dummy variables for educational attainment, and dummy variables for each value of the most current L-W score. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Figure 1: Distribution of the Alzheimer's Disease Polygenic Score



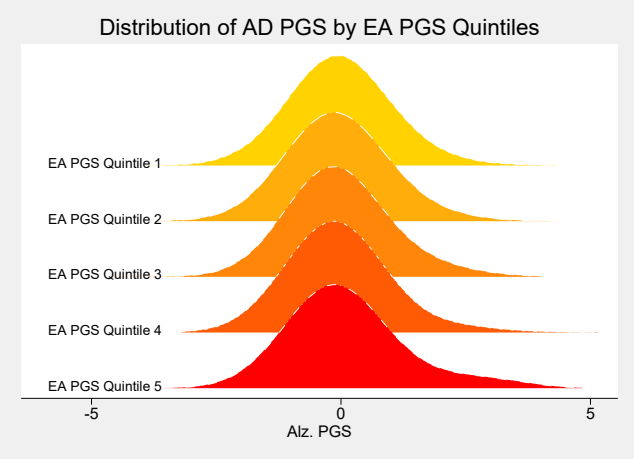
Note: The figure shows the smoothed density of the polygenic score for Alzheimer's disease in our sample.

Figure 2: Distribution of the Educational Attainment Polygenic Score



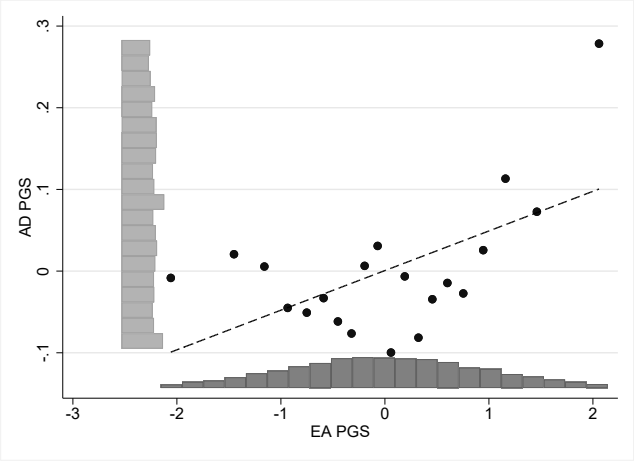
Note: The figure shows the smoothed density of the polygenic score for educational attainment in our sample.

Figure 3: Conditional Distribution of AD Score



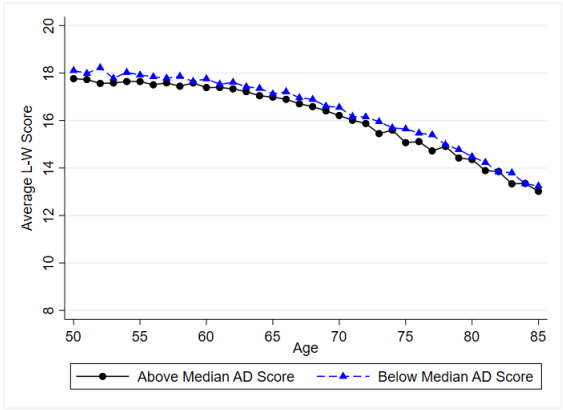
Note: The figure shows the distribution of the AD score conditional on different quintiles of the EA score.

Figure 4: Joint Distribution of the Polygenic Scores

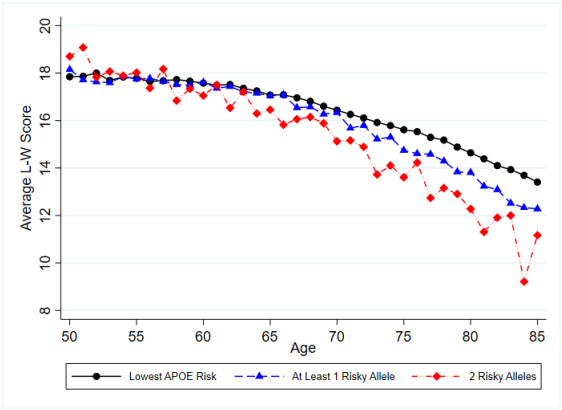


Note: The figure presents a bin-scatter plot for the AD score and EA score, and shows the marginal distributions of each score.

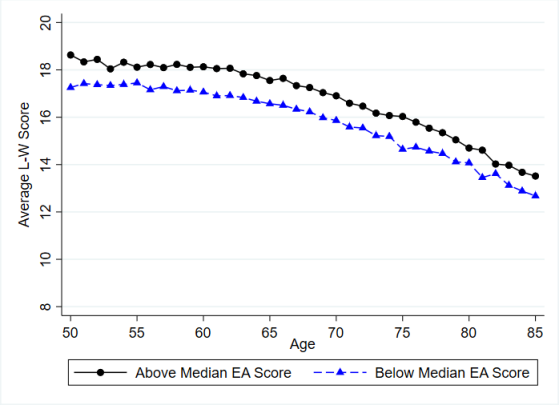
Figure 5: Age-Cognition Profiles by Genetic Risk Groups



(a) L-W Score by Above vs. Below Median AD Score



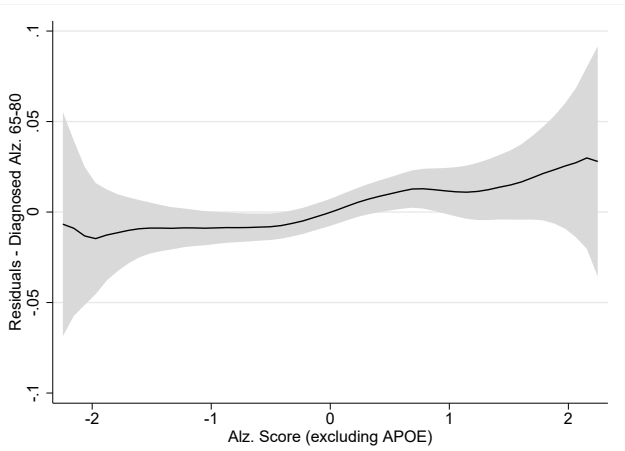
(b) L-W Score by APOE Status



(c) L-W Score by Above vs. Below Median EA Score

Note: The figure plots average values of the Langa-Weir cognition score by age for individuals in our main genotyped sample.

Figure 6: Relationship between AD Score and Residual Probability of MRD Diagnosis at Ages 65–80



Note: The figure plots a local polynomial approximation of the relationship between the AD score and an indicator for ever being diagnosed with MRD between the ages of 65–80 after it has been residualized on all of the control variables from the specification in Table 4. The sample is restricted to those who were not impaired, demented, or diagnosed with MRD in the age range 50–64.

Appendix

Table A1: Relationship between Genetic Endowments and Probability of Being Demented

	Ever Demented (L-W Score < 7)						
	Full Sample				Never Diagnosed		
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
AD Score	0.001***	0.001**	0.001	0.000	0.001**	0.001**	0.000
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
APOE (At least 1 copy)	0.006***	0.006***	0.006***	0.005***	0.002***	0.002***	0.002***
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
APOE (2 copies)	0.010***	0.009***	0.009***	0.006*	0.008**	0.008**	0.008**
	(0.003)	(0.003)	(0.003)	(0.004)	(0.003)	(0.003)	(0.003)
EA Score		-0.003***	-0.001***	-0.001***		-0.002***	-0.001**
		(0.000)	(0.000)	(0.000)		(0.000)	(0.000)
Ever MRD				0.062***			
				(0.008)			
Eventual MRD				0.023***			
				(0.002)			
Education Controls	No	No	Yes	Yes	No	No	Yes
Mean	0.009	0.009	0.009	0.009	0.006	0.006	0.006
N	86,412	86,412	86,412	86,412	79,119	79,119	79,119
R^2	0.013	0.014	0.025	0.042	0.009	0.010	0.022
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

Note: Each column presents results from a separate regression where the outcome is an indicator for an L-W score below 7 in the current wave or any prior 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. In columns (2) and (6), we add the educational attainment (EA) polygenic score. In columns (3) and (7), we add controls for educational attainment. In column (4), we add controls for whether the respondent has ever been diagnosed with a memory-related disease (MRD) and whether they will eventually report diagnosis (in subsequent survey waves). Columns (5)-(7) are estimated only on those never diagnosed with an MRD during the period they are observed. Standard errors are clustered at the individual level. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table A2: Relationship between Genetic Endowments and Survey Attrition

	Appear in the Next Wave								
	Full Sample					Never Diagnosed			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
AD Score	-0.004***	-0.004***	-0.003***	-0.003**	-0.003**	-0.004***	-0.004***	-0.003***	-0.002**
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
APOE (At least 1 copy)	-0.004*	-0.004*	-0.004*	-0.001	-0.003*	-0.005**	-0.005**	-0.005**	-0.003
	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)
APOE (2 copies)	-0.002	-0.001	-0.002	0.002	-0.000	-0.004	-0.003	-0.003	-0.001
	(0.006)	(0.006)	(0.006)	(0.006)	(0.006)	(0.007)	(0.007)	(0.007)	(0.007)
EA Score		0.006***	0.003***	0.002**	0.002*		0.006***	0.003***	0.002**
		(0.001)	(0.001)	(0.001)	(0.001)		(0.001)	(0.001)	(0.001)
Ever MRD					-0.087***				
					(0.008)				
Eventual MRD					0.039***				
					(0.002)				
L-W Score Dummies	No	No	No	Yes	Yes	No	No	No	Yes
Education Controls	No	No	Yes	Yes	Yes	No	No	Yes	Yes
Mean	0.944	0.944	0.944	0.944	0.944	0.943	0.943	0.943	0.943
N	82,461	82,461	82,461	82,461	82,461	74,852	74,852	74,852	74,852
R^2	0.049	0.050	0.052	0.059	0.061	0.048	0.048	0.051	0.058
Years	1998-2016	1998-2016	1998-2016	1998-2016	1998-2016	1998-2016	1998-2016	1998-2016	1998-2016
Ages	50-85	50-85	50-85	50-85	50-85	50-85	50-85	50-85	50-85

Note: Each column presents results from a separate regression where 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. In columns (2) and (7), we add the educational attainment (EA) polygenic score. In columns (3) and (8), we add controls for educational attainment. In columns (4) and (9), we add dummy variables for each value of the most current L-W score. In column (5), we add controls for whether the respondent has ever been diagnosed with a memory-related disease (MRD) and whether they will eventually report diagnosis (in subsequent survey waves). Columns (6)-(9) are estimated only on those never diagnosed with an MRD during the period they are observed. Standard errors are clustered at the individual level. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table A3: Relationship between Genetic Endowments and Survey Attrition (Ages 50–70)

	Appear in the Next Wave								
	Full Sample					Never Diagnosed			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
AD Score	-0.004***	-0.004***	-0.003***	-0.003**	-0.003**	-0.004***	-0.004***	-0.003**	-0.003**
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
APOE (At least 1 copy)	-0.002	-0.002	-0.002	-0.002	-0.003	-0.003	-0.003	-0.002	-0.002
	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)
APOE (2 copies)	0.001	0.001	0.001	0.001	0.000	-0.001	-0.001	-0.001	-0.001
	(0.007)	(0.007)	(0.007)	(0.007)	(0.007)	(0.008)	(0.008)	(0.008)	(0.008)
EA Score		0.004***	0.002*	0.001	0.001		0.005***	0.002*	0.001
		(0.001)	(0.001)	(0.001)	(0.001)		(0.001)	(0.001)	(0.001)
Ever MRD					-0.031***				
					(0.010)				
Eventual MRD					0.016***				
					(0.003)				
L-W Score Dummies	No	No	No	Yes	Yes	No	No	No	Yes
Education Controls	No	No	Yes	Yes	Yes	No	No	Yes	Yes
Mean	0.957	0.957	0.957	0.957	0.957	0.956	0.956	0.956	0.956
N	50,963	50,963	50,963	50,963	50,963	47,912	47,912	47,912	47,912
R^2	0.040	0.040	0.043	0.046	0.046	0.039	0.039	0.042	0.045
Years	1998-2016	1998-2016	1998-2016	1998-2016	1998-2016	1998-2016	1998-2016	1998-2016	1998-2016
Ages	50-70	50-70	50-70	50-70	50-70	50-70	50-70	50-70	50-70

Note: Each column presents results from a separate regression where 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. In columns (2) and (7), we add the educational attainment (EA) polygenic score. In columns (3) and (8), we add controls for educational attainment. In columns (4) and (9), we add dummy variables for each value of the most current L-W score. In column (5), we add controls for whether the respondent has ever been diagnosed with a memory-related disease (MRD) and whether they will eventually report diagnosis (in subsequent survey waves). Columns (6)-(9) are estimated only on those never diagnosed with an MRD during the period they are observed. Standard errors are clustered at the individual level. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table A4: Relationship between Genetic Endowments and Mortality

	Observed Mortality								
	Full Sample					Never Diagnosed			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
AD Score	0.002	0.002	0.001	0.000	0.000	0.002*	0.002	0.001	0.001
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
APOE (At least 1 copy)	0.001	0.001	0.001	-0.002	-0.000	0.002	0.002	0.002	-0.000
	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)
APOE (2 copies)	0.005	0.005	0.004	-0.000	0.003	0.011	0.011	0.009	0.004
	(0.007)	(0.007)	(0.007)	(0.007)	(0.007)	(0.008)	(0.008)	(0.008)	(0.008)
EA Score		-0.007***	-0.004***	-0.003**	-0.002**		-0.007***	-0.004***	-0.003**
		(0.001)	(0.001)	(0.001)	(0.001)		(0.001)	(0.001)	(0.001)
Ever MRD					0.118***				
					(0.009)				
Eventual MRD					-0.086***				
					(0.003)				
L-W Score Dummies	No	No	No	Yes	Yes	No	No	No	Yes
Education Controls	No	No	Yes	Yes	Yes	No	No	Yes	Yes
Mean	0.043	0.043	0.043	0.043	0.043	0.042	0.042	0.042	0.042
N	44,363	44,363	44,363	44,363	44,363	41,171	41,171	41,171	41,171
R^2	0.032	0.033	0.038	0.049	0.057	0.034	0.035	0.040	0.053
Years	2006-2016	2006-2016	2006-2016	2006-2016	2006-2016	2006-2016	2006-2016	2006-2016	2006-2016
Ages	50-85	50-85	50-85	50-85	50-85	50-85	50-85	50-85	50-85

Note: Each column presents results from a separate regression where the outcome is an indicator for whether the individual dies before 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. In columns (2) and (7), we add the educational attainment (EA) polygenic score. In columns (3) and (8), we add controls for educational attainment. In columns (4) and (9), we add dummy variables for each value of the most current L-W score. In column (5), we add controls for whether the respondent has ever been diagnosed with a memory-related disease (MRD) and whether they will eventually report diagnosis (in subsequent survey waves). Columns (6)-(9) are estimated only on those never diagnosed with an MRD during the period they are observed. Standard errors are clustered at the individual level. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table A5: Relationship between Genetic Endowments and Mortality (Ages 50–70)

	Observed Mortality								
	Full Sample					Never Diagnosed			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
AD Score	0.001	0.001	0.001	0.000	0.000	0.001	0.001	0.001	0.000
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
APOE (At least 1 copy)	-0.002	-0.003	-0.003	-0.003	-0.002	-0.002	-0.002	-0.002	-0.002
	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)
APOE (2 copies)	0.006	0.006	0.005	0.003	0.005	0.010	0.010	0.008	0.007
	(0.007)	(0.007)	(0.007)	(0.007)	(0.007)	(0.008)	(0.007)	(0.008)	(0.008)
EA Score		-0.005***	-0.003**	-0.002*	-0.002*		-0.005***	-0.002**	-0.002*
		(0.001)	(0.001)	(0.001)	(0.001)		(0.001)	(0.001)	(0.001)
Ever MRD					0.045***				
					(0.009)				
Eventual MRD					-0.038***				
					(0.003)				
L-W Score Dummies	No	No	No	Yes	Yes	No	No	No	Yes
Education Controls	No	No	Yes	Yes	Yes	No	No	Yes	Yes
Mean	0.020	0.020	0.020	0.020	0.020	0.020	0.020	0.020	0.020
N	24,314	24,314	24,314	24,314	24,314	23,339	23,339	23,339	23,339
R^2	0.010	0.012	0.019	0.029	0.031	0.011	0.013	0.021	0.032
Years	2006-2016	2006-2016	2006-2016	2006-2016	2006-2016	2006-2016	2006-2016	2006-2016	2006-2016
Ages	50-70	50-70	50-70	50-70	50-70	50-70	50-70	50-70	50-70

Note: Each column presents results from a separate regression where the outcome is an indicator for whether the individual dies before 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. In columns (2) and (7), we add the educational attainment (EA) polygenic score. In columns (3) and (8), we add controls for educational attainment. In columns (4) and (9), we add dummy variables for each value of the most current L-W score. In column (5), we add controls for whether the respondent has ever been diagnosed with a memory-related disease (MRD) and whether they will eventually report diagnosis (in subsequent survey waves). Columns (6)-(9) are estimated only on those never diagnosed with an MRD during the period they are observed. Standard errors are clustered at the individual level. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table A6: Relationship between Genetic Endowments and Retirement

	Retirement								
	Full Sample					Never Diagnosed			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
AD Score	0.012***	0.010**	0.008*	0.007	0.007	0.011**	0.010**	0.008*	0.007
	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)	(0.005)	(0.005)	(0.004)	(0.004)
APOE (At least 1 copy)	-0.008	-0.008	-0.006	-0.007	-0.009	-0.007	-0.007	-0.004	-0.005
	(0.008)	(0.008)	(0.008)	(0.008)	(0.008)	(0.008)	(0.008)	(0.008)	(0.008)
APOE (2 copies)	0.037	0.035	0.032	0.030	0.024	0.024	0.023	0.016	0.015
	(0.024)	(0.024)	(0.024)	(0.024)	(0.024)	(0.025)	(0.026)	(0.026)	(0.026)
EA Score		-0.029***	-0.016***	-0.014***	-0.014***		-0.027***	-0.015***	-0.013***
		(0.003)	(0.004)	(0.004)	(0.004)		(0.004)	(0.004)	(0.004)
Ever MRD					0.270***				
					(0.033)				
Eventual MRD					0.043**				
					(0.018)				
L-W Score Dummies	No	No	No	Yes	Yes	No	No	No	Yes
Education Controls	No	No	Yes	Yes	Yes	No	No	Yes	Yes
Mean	0.320	0.320	0.320	0.320	0.320	0.309	0.309	0.309	0.309
N	49,564	49,564	49,564	49,564	49,564	46,907	46,907	46,907	46,907
R^2	0.203	0.206	0.217	0.221	0.228	0.206	0.209	0.220	0.224
Years	1998-2018	1998-2018	1998-2018	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	50-70	50-70	50-70

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 columns (2) and (7), we add the educational attainment (EA) polygenic score. In columns (3) and (8), we add controls for educational attainment. In columns (4) and (9), we add dummy variables for each value of the most current L-W score. In column (5), we add controls for whether the respondent has ever been diagnosed with a memory-related disease (MRD) and whether they will eventually report diagnosis (in subsequent survey waves). Columns (6)-(9) are estimated only on those never diagnosed with an MRD during the period they are observed. 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 Endowments and Log Individual Total Income

	Log Individual Total Income								
	Full Sample					Never Diagnosed			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
AD Score	-0.037***	-0.033***	-0.017**	-0.014*	-0.013*	-0.032***	-0.028***	-0.011	-0.008
	(0.008)	(0.008)	(0.007)	(0.007)	(0.007)	(0.009)	(0.008)	(0.008)	(0.008)
APOE (At least 1 copy)	-0.015	-0.014	-0.016	-0.006	-0.003	-0.011	-0.010	-0.011	-0.004
	(0.015)	(0.015)	(0.014)	(0.013)	(0.014)	(0.016)	(0.016)	(0.015)	(0.015)
APOE (2 copies)	0.002	0.010	-0.004	0.009	0.015	0.020	0.024	0.032	0.041
	(0.046)	(0.044)	(0.040)	(0.039)	(0.039)	(0.051)	(0.049)	(0.043)	(0.042)
EA Score		0.092***	0.016***	0.011*	0.011*		0.091***	0.014**	0.008
		(0.006)	(0.006)	(0.006)	(0.006)		(0.007)	(0.006)	(0.006)
Ever MRD					-0.187***				
					(0.036)				
Eventual MRD					-0.037*				
					(0.020)				
L-W Score Dummies	No	No	No	Yes	Yes	No	No	No	Yes
Education Controls	No	No	Yes	Yes	Yes	No	No	Yes	Yes
Mean	10.010	10.010	10.010	10.010	10.010	10.036	10.036	10.036	10.036
N	81,393	81,393	81,393	81,393	81,393	73,999	73,999	73,999	73,999
R^2	0.186	0.195	0.266	0.274	0.275	0.184	0.193	0.266	0.273
Years	1998-2018	1998-2018	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	50-85	50-85

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 columns (2) and (7), we add the educational attainment (EA) polygenic score. In columns (3) and (8), we add controls for educational attainment. In columns (4) and (9), we add dummy variables for each value of the most current L-W score. In column (5), we add controls for whether the respondent has ever been diagnosed with a memory-related disease (MRD) and whether they will eventually report diagnosis (in subsequent survey waves). Columns (6)-(9) are estimated only on those never diagnosed with an MRD during the period they are observed. Standard errors are clustered at the individual level. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table A8: Relationship between Genetic Endowments and Log Household Wealth

	Log Household Total Wealth								
	Full Sample					Never Diagnosed			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
AD Score	-0.091***	-0.079***	-0.046***	-0.039**	-0.038**	-0.080***	-0.068***	-0.037**	-0.030*
	(0.017)	(0.017)	(0.016)	(0.016)	(0.016)	(0.018)	(0.018)	(0.016)	(0.016)
APOE (At least 1 copy)	-0.016	-0.013	-0.019	0.002	0.004	-0.013	-0.011	-0.015	-0.002
	(0.032)	(0.031)	(0.029)	(0.029)	(0.029)	(0.034)	(0.033)	(0.031)	(0.031)
APOE (2 copies)	0.058	0.080	0.077	0.106	0.108	-0.022	-0.011	0.021	0.041
	(0.107)	(0.105)	(0.094)	(0.093)	(0.093)	(0.120)	(0.117)	(0.106)	(0.105)
EA Score		0.267***	0.108***	0.096***	0.096***		0.265***	0.111***	0.099***
		(0.014)	(0.014)	(0.013)	(0.013)		(0.014)	(0.014)	(0.014)
Ever MRD					-0.249***				
					(0.085)				
Eventual MRD					0.003				
					(0.047)				
L-W Score Dummies	No	No	No	Yes	Yes	No	No	No	Yes
Education Controls	No	No	Yes	Yes	Yes	No	No	Yes	Yes
Mean	12.400	12.400	12.400	12.400	12.400	12.415	12.415	12.415	12.415
N	84,548	84,548	84,548	84,548	84,548	77,110	77,110	77,110	77,110
R^2	0.039	0.065	0.168	0.180	0.181	0.039	0.065	0.165	0.177
Years	1998-2018	1998-2018	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	50-85	50-85

Note: Each column presents results from a separate regression where the outcome is logged household wealth. 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 (7), we add the educational attainment (EA) polygenic score. In columns (3) and (8), we add controls for educational attainment. In columns (4) and (9), we add dummy variables for each value of the most current L-W score. In column (5), we add controls for whether the respondent has ever been diagnosed with a memory-related disease (MRD) and whether they will eventually report diagnosis (in subsequent survey waves). Columns (6)-(9) are estimated only on those never diagnosed with an MRD during the period they are observed. Standard errors are clustered at the individual level. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table A9: Relationship between Genetic Endowments and Log Household Wealth (Males)

	Log Household Total Wealth								
	Male Sample					Never Diagnosed			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
AD Score	-0.079***	-0.068***	-0.030	-0.024	-0.024	-0.063**	-0.050*	-0.017	-0.011
	(0.026)	(0.025)	(0.023)	(0.023)	(0.023)	(0.027)	(0.026)	(0.024)	(0.024)
APOE (At least 1 copy)	-0.027	-0.032	-0.034	-0.018	-0.015	-0.041	-0.044	-0.050	-0.040
	(0.047)	(0.046)	(0.042)	(0.042)	(0.042)	(0.049)	(0.049)	(0.045)	(0.044)
APOE (2 copies)	-0.097	-0.072	-0.059	-0.043	-0.031	-0.184	-0.177	-0.152	-0.139
	(0.162)	(0.158)	(0.146)	(0.143)	(0.144)	(0.186)	(0.181)	(0.165)	(0.161)
EA Score		0.250***	0.098***	0.085***	0.085***		0.249***	0.103***	0.091***
		(0.020)	(0.019)	(0.019)	(0.019)		(0.021)	(0.020)	(0.020)
Ever MRD					-0.400***				
					(0.126)				
Eventual MRD					-0.022				
					(0.066)				
L-W Score Dummies	No	No	No	Yes	Yes	No	No	No	Yes
Education Controls	No	No	Yes	Yes	Yes	No	No	Yes	Yes
Mean	12.543	12.543	12.543	12.543	12.543	12.558	12.558	12.558	12.558
N	35,569	35,569	35,569	35,569	35,569	32,298	32,298	32,298	32,298
R^2	0.042	0.067	0.183	0.196	0.198	0.043	0.068	0.180	0.192
Years	1998-2018	1998-2018	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	50-85	50-85

Note: Each column presents results from a separate regression where the outcome is logged household wealth and the sample is only males. 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, and survey year as described in Section 4. In columns (2) and (7), we add the educational attainment (EA) polygenic score. In columns (3) and (8), we add controls for educational attainment. In columns (4) and (9), we add dummy variables for each value of the most current L-W score. In column (5), we add controls for whether the respondent has ever been diagnosed with a memory-related disease (MRD) and whether they will eventually report diagnosis (in subsequent survey waves). Columns (6)-(9) are estimated only on those never diagnosed with an MRD during the period they are observed. Standard errors are clustered at the individual level. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table A10: Relationship between Genetic Endowments and Log Household Wealth (Females)

	Log Household Total Wealth								
	Female Sample					Never Diagnosed			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
AD Score	-0.100***	-0.087***	-0.057***	-0.049**	-0.049**	-0.092***	-0.080***	-0.051**	-0.044**
	(0.023)	(0.023)	(0.021)	(0.021)	(0.021)	(0.024)	(0.024)	(0.022)	(0.022)
APOE (At least 1 copy)	-0.007	-0.000	-0.008	0.018	0.018	0.007	0.013	0.010	0.027
	(0.043)	(0.042)	(0.040)	(0.039)	(0.039)	(0.046)	(0.045)	(0.042)	(0.042)
APOE (2 copies)	0.177	0.197	0.182	0.220*	0.219*	0.097	0.111	0.149	0.171
	(0.140)	(0.138)	(0.120)	(0.119)	(0.119)	(0.155)	(0.152)	(0.135)	(0.135)
EA Score		0.279***	0.116***	0.104***	0.104***		0.277***	0.117***	0.105***
		(0.019)	(0.019)	(0.019)	(0.019)		(0.020)	(0.020)	(0.019)
Ever MRD					-0.087				
					(0.111)				
Eventual MRD					0.021				
					(0.065)				
L-W Score Dummies	No	No	No	Yes	Yes	No	No	No	Yes
Education Controls	No	No	Yes	Yes	Yes	No	No	Yes	Yes
Mean	12.296	12.296	12.296	12.296	12.296	12.312	12.312	12.312	12.312
N	48,979	48,979	48,979	48,979	48,979	44,812	44,812	44,812	44,812
R^2	0.029	0.055	0.151	0.164	0.164	0.029	0.055	0.149	0.162
Years	1998-2018	1998-2018	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	50-85	50-85

Note: Each column presents results from a separate regression where the outcome is logged household wealth and the sample is only females. 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, and survey year as described in Section 4. In columns (2) and (7), we add the educational attainment (EA) polygenic score. In columns (3) and (8), we add controls for educational attainment. In columns (4) and (9), we add dummy variables for each value of the most current L-W score. In column (5), we add controls for whether the respondent has ever been diagnosed with a memory-related disease (MRD) and whether they will eventually report diagnosis (in subsequent survey waves). Columns (6)-(9) are estimated only on those never diagnosed with an MRD during the period they are observed. Standard errors are clustered at the individual level. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table A11: Relationship between Genetic Endowments and Log Household Wealth (Married Females)

	Log Household Total Wealth								
	Female Sample					Never Diagnosed			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
AD Score	-0.097***	-0.086***	-0.052**	-0.048**	-0.048**	-0.090***	-0.080***	-0.047**	-0.043*
	(0.023)	(0.023)	(0.022)	(0.021)	(0.021)	(0.024)	(0.024)	(0.023)	(0.022)
APOE (At least 1 copy)	0.024	0.023	0.021	0.038	0.038	0.061	0.059	0.059	0.071*
	(0.043)	(0.042)	(0.039)	(0.039)	(0.039)	(0.045)	(0.044)	(0.041)	(0.040)
APOE (2 copies)	0.117	0.137	0.106	0.136	0.136	0.063	0.073	0.075	0.098
	(0.152)	(0.150)	(0.134)	(0.130)	(0.130)	(0.168)	(0.166)	(0.149)	(0.145)
EA Score		0.220***	0.091***	0.080***	0.080***		0.212***	0.085***	0.075***
		(0.019)	(0.019)	(0.019)	(0.019)		(0.020)	(0.020)	(0.020)
Ever MRD					-0.069				
					(0.114)				
Eventual MD					0.001				
					(0.072)				
L-W Score Dummies	No	No	No	Yes	Yes	No	No	No	Yes
Education Controls	No	No	Yes	Yes	Yes	No	No	Yes	Yes
Mean	12.694	12.694	12.694	12.694	12.694	12.702	12.702	12.702	12.702
N	30,554	30,554	30,554	30,554	30,554	28,165	28,165	28,165	28,165
R^2	0.041	0.066	0.167	0.179	0.179	0.042	0.065	0.165	0.177
Years	1998-2018	1998-2018	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	50-85	50-85

Note: Each column presents results from a separate regression where the outcome is logged household wealth and the sample is only married females. 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, and survey year as described in Section 4. In columns (2) and (7), we add the educational attainment (EA) polygenic score. In columns (3) and (8), we add controls for educational attainment. In columns (4) and (9), we add dummy variables for each value of the most current L-W score. In column (5), we add controls for whether the respondent has ever been diagnosed with a memory-related disease (MRD) and whether they will eventually report diagnosis (in subsequent survey waves). Columns (6)-(9) are estimated only on those never diagnosed with an MRD during the period they are observed. Standard errors are clustered at the individual level. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table A12: Relationship between Genetic Endowments and Holding Long-Term Care Insurance

	Holds Long-Term Care Insurance								
	Full Sample					Never Diagnosed			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
AD Score	-0.013***	-0.012***	-0.010***	-0.010***	-0.010***	-0.012***	-0.012***	-0.009**	-0.009**
	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)
APOE (At least 1 copy)	0.005	0.006	0.006	0.006	0.005	0.007	0.007	0.007	0.007
	(0.007)	(0.007)	(0.007)	(0.007)	(0.007)	(0.007)	(0.007)	(0.007)	(0.007)
APOE (2 copies)	-0.001	-0.000	-0.001	-0.000	-0.002	-0.010	-0.010	-0.006	-0.006
	(0.020)	(0.020)	(0.019)	(0.019)	(0.019)	(0.021)	(0.020)	(0.020)	(0.020)
EA Score		0.017***	0.001	0.001	0.001		0.015***	-0.000	-0.001
		(0.003)	(0.003)	(0.003)	(0.003)		(0.003)	(0.003)	(0.003)
Ever MRD					-0.019				
					(0.022)				
Eventual MRD					0.021				
					(0.013)				
L-W Score Dummies	No	No	No	Yes	Yes	No	No	No	Yes
Education Controls	No	No	Yes	Yes	Yes	No	No	Yes	Yes
Mean	0.126	0.126	0.126	0.126	0.126	0.126	0.126	0.126	0.126
N	52,747	52,747	52,747	52,747	52,747	49,706	49,706	49,706	49,706
R^2	0.018	0.021	0.044	0.045	0.045	0.018	0.020	0.045	0.046
Years	1998-2018	1998-2018	1998-2018	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	50-70	50-70	50-70

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 columns (2) and (7), we add the educational attainment (EA) polygenic score. In columns (3) and (8), we add controls for educational attainment. In columns (4) and (9), we add dummy variables for each value of the most current L-W score. In column (5), we add controls for whether the respondent has ever been diagnosed with a memory-related disease (MRD) and whether they will eventually report diagnosis (in subsequent survey waves). Columns (6)-(9) are estimated only on those never diagnosed with an MRD during the period they are observed. Standard errors are clustered at the individual level. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table A13: Relationship between Genetic Endowments and Holding Life Insurance

	Holds Life Insurance								
	Full Sample					Never Diagnosed			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
AD Score	-0.010**	-0.009*	-0.006	-0.006	-0.006	-0.007	-0.006	-0.003	-0.003
	(0.005)	(0.005)	(0.005)	(0.005)	(0.005)	(0.005)	(0.005)	(0.005)	(0.005)
APOE (At least 1 copy)	-0.001	-0.001	-0.001	-0.000	-0.001	0.002	0.002	0.001	0.001
	(0.009)	(0.009)	(0.009)	(0.009)	(0.009)	(0.009)	(0.009)	(0.009)	(0.009)
APOE (2 copies)	-0.013	-0.012	-0.009	-0.006	-0.006	-0.036	-0.036	-0.029	-0.027
	(0.026)	(0.026)	(0.026)	(0.026)	(0.026)	(0.028)	(0.028)	(0.028)	(0.028)
EA Score		0.016***	0.006	0.004	0.004		0.015***	0.005	0.003
		(0.004)	(0.004)	(0.004)	(0.004)		(0.004)	(0.004)	(0.004)
Ever MRD					-0.049				
					(0.034)				
Eventual MRD					0.005				
					(0.017)				
L-W Score Dummies	No	No	No	Yes	Yes	No	No	No	Yes
Education Controls	No	No	Yes	Yes	Yes	No	No	Yes	Yes
Mean	0.714	0.714	0.714	0.714	0.714	0.716	0.716	0.716	0.716
N	53,322	53,322	53,322	53,322	53,322	50,233	50,233	50,233	50,233
R^2	0.037	0.038	0.050	0.055	0.055	0.038	0.039	0.052	0.057
Years	1998-2018	1998-2018	1998-2018	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	50-70	50-70	50-70

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 columns (2) and (7), we add the educational attainment (EA) polygenic score. In columns (3) and (8), we add controls for educational attainment. In columns (4) and (9), we add dummy variables for each value of the most current L-W score. In column (5), we add controls for whether the respondent has ever been diagnosed with a memory-related disease (MRD) and whether they will eventually report diagnosis (in subsequent survey waves). Columns (6)-(9) are estimated only on those never diagnosed with an MRD during the period they are observed. Standard errors are clustered at the individual level. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table A14: Relationship between Genetic Endowments and Having a Witnessed Will

	Has a Witnessed Will								
	Full Sample					Never Diagnosed			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
AD Score	-0.024***	-0.021***	-0.014**	-0.013**	-0.013**	-0.022***	-0.019***	-0.012**	-0.012**
	(0.006)	(0.006)	(0.006)	(0.006)	(0.006)	(0.006)	(0.006)	(0.006)	(0.006)
APOE (At least 1 copy)	0.013	0.014	0.012	0.013	0.013	0.015	0.016	0.014	0.015
	(0.011)	(0.011)	(0.010)	(0.010)	(0.010)	(0.011)	(0.011)	(0.011)	(0.011)
APOE (2 copies)	-0.013	-0.009	-0.007	-0.005	-0.005	-0.025	-0.024	-0.011	-0.009
	(0.033)	(0.033)	(0.031)	(0.031)	(0.031)	(0.036)	(0.036)	(0.034)	(0.033)
EA Score		0.057***	0.019***	0.017***	0.017***		0.056***	0.019***	0.017***
		(0.005)	(0.005)	(0.005)	(0.005)		(0.005)	(0.005)	(0.005)
Ever MRD					-0.041				
					(0.037)				
Eventual MRD					0.005				
					(0.019)				
L-W Score Dummies	No	No	No	Yes	Yes	No	No	No	Yes
Education Controls	No	No	Yes	Yes	Yes	No	No	Yes	Yes
Mean	0.559	0.559	0.559	0.559	0.559	0.558	0.558	0.558	0.558
N	53,484	53,484	53,484	53,484	53,484	50,389	50,389	50,389	50,389
R^2	0.065	0.077	0.131	0.134	0.134	0.066	0.078	0.130	0.133
Years	1998-2018	1998-2018	1998-2018	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	50-70	50-70	50-70

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 columns (2) and (7), we add the educational attainment (EA) polygenic score. In columns (3) and (8), we add controls for educational attainment. In columns (4) and (9), we add dummy variables for each value of the most current L-W score. In column (5), we add controls for whether the respondent has ever been diagnosed with a memory-related disease (MRD) and whether they will eventually report diagnosis (in subsequent survey waves). Columns (6)-(9) are estimated only on those never diagnosed with an MRD during the period they are observed. Standard errors are clustered at the individual level. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table A15: Relationship between Genetic Endowments and Having a Living Will

	Has a Living Will (Advance Healthcare Directive)								
	Full Sample					Never Diagnosed			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
AD Score	-0.023*	-0.021*	-0.019	-0.018	-0.018	-0.020	-0.019	-0.017	-0.016
	(0.012)	(0.012)	(0.012)	(0.012)	(0.012)	(0.012)	(0.012)	(0.012)	(0.012)
APOE (At least 1 copy)	0.032	0.033	0.034	0.033	0.032	0.040*	0.041*	0.042*	0.041*
	(0.023)	(0.023)	(0.022)	(0.022)	(0.022)	(0.023)	(0.023)	(0.023)	(0.023)
APOE (2 copies)	0.045	0.056	0.063	0.063	0.060	0.030	0.040	0.059	0.060
	(0.066)	(0.064)	(0.061)	(0.062)	(0.062)	(0.069)	(0.067)	(0.064)	(0.064)
EA Score		0.045***	0.013	0.013	0.013		0.045***	0.013	0.013
		(0.010)	(0.010)	(0.010)	(0.010)		(0.010)	(0.010)	(0.010)
Ever MRD					0.006				
					(0.095)				
Eventual MRD					0.048				
					(0.084)				
L-W Score Dummies	No	No	No	Yes	Yes	No	No	No	Yes
Education Controls	No	No	Yes	Yes	Yes	No	No	Yes	Yes
Mean	0.478	0.478	0.478	0.478	0.478	0.476	0.476	0.476	0.476
N	5,066	5,066	5,066	5,066	5,066	4,850	4,850	4,850	4,850
R^2	0.038	0.045	0.098	0.100	0.101	0.040	0.048	0.101	0.104
Years	2012-2018	2012-2018	2012-2018	2012-2018	2012-2018	2012-2018	2012-2018	2012-2018	2012-2018
Ages	65-70	65-70	65-70	65-70	65-70	65-70	65-70	65-70	65-70

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 columns (2) and (7), we add the educational attainment (EA) polygenic score. In columns (3) and (8), we add controls for educational attainment. In columns (4) and (9), we add dummy variables for each value of the most current L-W score. In column (5), we add controls for whether the respondent has ever been diagnosed with a memory-related disease (MRD) and whether they will eventually report diagnosis (in subsequent survey waves). Columns (6)-(9) are estimated only on those never diagnosed with an MRD during the period they are observed. Standard errors are clustered at the individual level. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table A16: Relationship between Genetic Endowments and Having Assigned Someone Durable Power of Attorney

	Has Assigned Someone Durable Power of Attorney for Healthcare								
	Full Sample					Never Diagnosed			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
AD Score	-0.045***	-0.043***	-0.039***	-0.038***	-0.038***	-0.043***	-0.041***	-0.038***	-0.038***
	(0.012)	(0.012)	(0.012)	(0.012)	(0.012)	(0.012)	(0.012)	(0.012)	(0.012)
APOE (At least 1 copy)	0.014	0.016	0.017	0.015	0.014	0.016	0.017	0.019	0.017
	(0.022)	(0.022)	(0.022)	(0.022)	(0.022)	(0.023)	(0.023)	(0.023)	(0.023)
APOE (2 copies)	0.042	0.053	0.057	0.057	0.053	0.067	0.076	0.092	0.092
	(0.066)	(0.065)	(0.063)	(0.063)	(0.064)	(0.068)	(0.067)	(0.064)	(0.064)
EA Score		0.045***	0.012	0.013	0.013		0.044***	0.012	0.012
		(0.009)	(0.010)	(0.010)	(0.010)		(0.010)	(0.010)	(0.010)
Ever MRD					-0.090				
					(0.092)				
Eventual MRD					0.107				
					(0.081)				
L-W Score Dummies	No	No	No	Yes	Yes	No	No	No	Yes
Education Controls	No	No	Yes	Yes	Yes	No	No	Yes	Yes
Mean	0.462	0.462	0.462	0.462	0.462	0.461	0.461	0.461	0.461
N	5,067	5,067	5,067	5,067	5,067	4,852	4,852	4,852	4,852
R^2	0.035	0.042	0.094	0.097	0.098	0.036	0.043	0.096	0.100
Years	2012-2018	2012-2018	2012-2018	2012-2018	2012-2018	2012-2018	2012-2018	2012-2018	2012-2018
Ages	65-70	65-70	65-70	65-70	65-70	65-70	65-70	65-70	65-70

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 columns (2) and (7), we add the educational attainment (EA) polygenic score. In columns (3) and (8), we add controls for educational attainment. In columns (4) and (9), we add dummy variables for each value of the most current L-W score. In column (5), we add controls for whether the respondent has ever been diagnosed with a memory-related disease (MRD) and whether they will eventually report diagnosis (in subsequent survey waves). Columns (6)-(9) are estimated only on those never diagnosed with an MRD during the period they are observed. Standard errors are clustered at the individual level. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table A17: Relationship between Genetic Endowments and Having Discussed Future Medical Care with Someone

	Discussed Future Medical Care with Anyone								
	Full Sample					Never Diagnosed			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
AD Score	-0.031***	-0.030***	-0.026**	-0.025**	-0.025**	-0.031***	-0.030***	-0.026**	-0.026**
	(0.011)	(0.011)	(0.011)	(0.011)	(0.011)	(0.011)	(0.011)	(0.011)	(0.011)
APOE (At least 1 copy)	-0.011	-0.008	-0.007	-0.006	-0.005	-0.001	0.001	0.003	0.003
	(0.020)	(0.020)	(0.019)	(0.019)	(0.019)	(0.020)	(0.020)	(0.020)	(0.020)
APOE (2 copies)	-0.032	-0.026	-0.037	-0.026	-0.024	-0.030	-0.025	-0.024	-0.014
	(0.064)	(0.063)	(0.061)	(0.061)	(0.060)	(0.068)	(0.067)	(0.066)	(0.065)
EA Score		0.025***	-0.002	-0.005	-0.004		0.024***	-0.003	-0.005
		(0.008)	(0.009)	(0.009)	(0.009)		(0.009)	(0.009)	(0.009)
Ever MRD					0.092				
					(0.081)				
Eventual MRD					-0.072				
					(0.065)				
L-W Score Dummies	No	No	No	Yes	Yes	No	No	No	Yes
Education Controls	No	No	Yes	Yes	Yes	No	No	Yes	Yes
Mean	0.590	0.590	0.590	0.590	0.590	0.592	0.592	0.592	0.592
N	3,500	3,500	3,500	3,500	3,500	3,347	3,347	3,347	3,347
R^2	0.071	0.073	0.117	0.126	0.126	0.073	0.075	0.119	0.126
Years	2012-2018	2012-2018	2012-2018	2012-2018	2012-2018	2012-2018	2012-2018	2012-2018	2012-2018
Ages	65-70	65-70	65-70	65-70	65-70	65-70	65-70	65-70	65-70

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 columns (2) and (7), we add the educational attainment (EA) polygenic score. In columns (3) and (8), we add controls for educational attainment. In columns (4) and (9), we add dummy variables for each value of the most current L-W score. In column (5), we add controls for whether the respondent has ever been diagnosed with a memory-related disease (MRD) and whether they will eventually report diagnosis (in subsequent survey waves). Columns (6)-(9) are estimated only on those never diagnosed with an MRD during the period they are observed. Standard errors are clustered at the individual level. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table A18: Relationship between Genetic Endowments and Expected Mortality

	Probability of Living to Age 75								
	Full Sample					Never Diagnosed			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
AD Score	-0.350	-0.240	0.097	0.143	0.184	-0.196	-0.094	0.232	0.277
	(0.317)	(0.316)	(0.307)	(0.305)	(0.305)	(0.323)	(0.322)	(0.313)	(0.312)
APOE (At least 1 copy)	-0.749	-0.684	-0.706	-0.665	-0.625	-0.807	-0.750	-0.807	-0.759
	(0.587)	(0.582)	(0.560)	(0.558)	(0.556)	(0.597)	(0.593)	(0.572)	(0.570)
APOE (2 copies)	-1.094	-1.010	-0.678	-0.587	-0.290	-1.072	-1.085	-0.365	-0.356
	(1.910)	(1.922)	(1.898)	(1.889)	(1.883)	(2.070)	(2.076)	(2.052)	(2.044)
EA Score		2.442***	0.666**	0.541**	0.495*		2.381***	0.647**	0.542**
		(0.253)	(0.259)	(0.258)	(0.257)		(0.258)	(0.264)	(0.263)
Ever MRD					-11.910***				
					(2.249)				
Eventual MRD					-2.693**				
					(1.256)				
L-W Score Dummies	No	No	No	Yes	Yes	No	No	No	Yes
Education Controls	No	No	Yes	Yes	Yes	No	No	Yes	Yes
Mean	66.275	66.275	66.275	66.275	66.275	66.645	66.645	66.645	66.645
N	36,282	36,282	36,282	36,282	36,282	34,611	34,611	34,611	34,611
R^2	0.027	0.035	0.079	0.085	0.090	0.027	0.035	0.079	0.085
Years	1998-2018	1998-2018	1998-2018	1998-2018	1998-2018	1998-2018	1998-2018	1998-2018	1998-2018
Ages	50-65	50-65	50-65	50-65	50-65	50-65	50-65	50-65	50-65

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 columns (2) and (7), we add the educational attainment (EA) polygenic score. In columns (3) and (8), we add controls for educational attainment. In columns (4) and (9), we add dummy variables for each value of the most current L-W score. In column (5), we add controls for whether the respondent has ever been diagnosed with a memory-related disease (MRD) and whether they will eventually report diagnosis (in subsequent survey waves). Columns (6)-(9) are estimated only on those never diagnosed with an MRD during the period they are observed. Standard errors are clustered at the individual level. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table A19: Relationship between Genetic Endowments and Expected Nursing Home Use

	Probability of Moving to Nursing Home in Next 5 Years								
	Full Sample					Never Diagnosed			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
AD Score	-0.055	-0.060	-0.062	-0.060	-0.061	-0.085	-0.092	-0.098	-0.106
	(0.211)	(0.211)	(0.212)	(0.212)	(0.211)	(0.215)	(0.216)	(0.217)	(0.217)
APOE (At least 1 copy)	0.721*	0.721*	0.701*	0.672*	0.637	0.809**	0.809**	0.790*	0.785*
	(0.390)	(0.390)	(0.390)	(0.389)	(0.391)	(0.407)	(0.407)	(0.407)	(0.407)
APOE (2 copies)	0.140	0.122	0.136	0.034	-0.076	-1.830	-1.848	-1.880	-1.900
	(1.360)	(1.363)	(1.362)	(1.347)	(1.344)	(1.383)	(1.388)	(1.386)	(1.394)
EA Score		-0.115	-0.013	0.040	0.043		-0.153	-0.053	-0.014
		(0.168)	(0.178)	(0.179)	(0.179)		(0.174)	(0.184)	(0.184)
Ever MRD					5.165***				
					(1.813)				
Eventual MRD					0.245				
					(0.726)				
L-W Score Dummies	No	No	No	Yes	Yes	No	No	No	Yes
Education Controls	No	No	Yes	Yes	Yes	No	No	Yes	Yes
Mean	10.982	10.982	10.982	10.982	10.982	10.847	10.847	10.847	10.847
N	16,795	16,795	16,795	16,795	16,795	15,426	15,426	15,426	15,426
R^2	0.014	0.014	0.019	0.023	0.024	0.015	0.015	0.020	0.023
Years	1998-2018	1998-2018	1998-2018	1998-2018	1998-2018	1998-2018	1998-2018	1998-2018	1998-2018
Ages	65-70	65-70	65-70	65-70	65-70	65-70	65-70	65-70	65-70

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 columns (2) and (7), we add the educational attainment (EA) polygenic score. In columns (3) and (8), we add controls for educational attainment. In columns (4) and (9), we add dummy variables for each value of the most current L-W score. In column (5), we add controls for whether the respondent has ever been diagnosed with a memory-related disease (MRD) and whether they will eventually report diagnosis (in subsequent survey waves). Columns (6)-(9) are estimated only on those never diagnosed with an MRD during the period they are observed. Standard errors are clustered at the individual level. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table A20: Relationship between Genetic Endowments and Expected Probability of Developing Alzheimer's Disease

	Probability of Developing Alzheimer's Disease								
	Full Sample					Never Diagnosed			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
AD Score	-0.395	-0.457	-0.456	-0.288	-0.281	-0.333	-0.448	-0.723	-0.498
	(1.504)	(1.503)	(1.509)	(1.570)	(1.572)	(1.559)	(1.557)	(1.567)	(1.631)
APOE (At least 1 copy)	6.949**	7.442***	7.909***	7.404***	7.424***	6.448**	6.921**	7.789***	7.297***
	(2.694)	(2.683)	(2.590)	(2.580)	(2.591)	(2.776)	(2.756)	(2.679)	(2.668)
APOE (2 copies)	-6.562	-6.302	-7.451	-5.681	-5.660	-5.927	-5.763	-7.579	-5.645
	(7.850)	(7.729)	(7.848)	(8.021)	(8.035)	(8.741)	(8.638)	(8.650)	(8.829)
EA Score		2.534**	3.094***	3.036**	3.038**		2.658**	3.376***	3.331***
		(1.132)	(1.165)	(1.207)	(1.208)		(1.155)	(1.199)	(1.237)
Eventual MRD					-0.639				
					(6.608)				
L-W Score Dummies	No	No	No	Yes	Yes	No	No	No	Yes
Education Controls	No	No	Yes	Yes	Yes	No	No	Yes	Yes
Mean	36.303	36.303	36.303	36.303	36.303	36.105	36.105	36.105	36.105
N	660	660	660	660	660	638	638	638	638
R^2	0.222	0.229	0.308	0.345	0.345	0.214	0.222	0.307	0.348
Years	2002, 2012, 2016	2002, 2012, 2016	2002, 2012, 2016	2002, 2012, 2016	2002, 2012, 2016	2002, 2012, 2016	2002, 2012, 2016	2002, 2012, 2016	2002, 2012, 2016
Ages	50-70	50-70	50-70	50-70	50-70	50-70	50-70	50-70	50-70

Note: Each column presents results from a separate regression where the outcome is the self-reported probability of developing Alzheimer's disease or dementia. 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 (7), we add the educational attainment (EA) polygenic score. In columns (3) and (8), we add controls for educational attainment. In columns (4) and (9), we add dummy variables for each value of the most current L-W score. In column (5), we add controls for whether the respondent has ever been diagnosed with an MRD and whether they will eventually report diagnosis (in subsequent survey waves). Columns (6)-(9) are estimated only on those never diagnosed with an MRD during the period they are observed. Standard errors are clustered at the individual level. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table A21: Relationship between Genetic Endowments and Expected Probability of Developing Alzheimer's Disease Greater than 50%

	Probability of Developing Alzheimer's Disease $\geq$ 50								
	Full Sample					Never Diagnosed			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
AD Score	-0.017	-0.017	-0.019	-0.019	-0.019	-0.018	-0.020	-0.024	-0.023
	(0.028)	(0.028)	(0.029)	(0.030)	(0.030)	(0.028)	(0.028)	(0.030)	(0.031)
APOE (At least 1 copy)	0.099*	0.104**	0.122**	0.112**	0.112**	0.085	0.090*	0.111**	0.098*
	(0.051)	(0.051)	(0.050)	(0.051)	(0.051)	(0.052)	(0.052)	(0.051)	(0.052)
APOE (2 copies)	-0.145	-0.142	-0.160	-0.130	-0.130	-0.128	-0.127	-0.163	-0.128
	(0.148)	(0.148)	(0.153)	(0.157)	(0.157)	(0.164)	(0.164)	(0.166)	(0.169)
EA Score		0.025	0.042*	0.040	0.040		0.027	0.046*	0.046*
		(0.023)	(0.024)	(0.025)	(0.025)		(0.024)	(0.024)	(0.025)
Eventual MRD					-0.000				
					(0.138)				
L-W Score Dummies	No	No	No	Yes	Yes	No	No	No	Yes
Education Controls	No	No	Yes	Yes	Yes	No	No	Yes	Yes
Mean	0.436	0.436	0.436	0.436	0.436	0.433	0.433	0.433	0.433
N	660	660	660	660	660	638	638	638	638
R^2	0.200	0.202	0.275	0.304	0.304	0.203	0.205	0.282	0.315
Years	2002, 2012, 2016	2002, 2012, 2016	2002, 2012, 2016	2002, 2012, 2016	2002, 2012, 2016	2002, 2012, 2016	2002, 2012, 2016	2002, 2012, 2016	2002, 2012, 2016
Ages	50-70	50-70	50-70	50-70	50-70	50-70	50-70	50-70	50-70

Note: Each column presents results from a separate regression where the outcome is an indicator for whether the respondent's self-reported probability of developing Alzheimer's disease or dementia is greater than or equal to 50%. 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 (7), we add the educational attainment (EA) polygenic score. In columns (3) and (8), we add controls for educational attainment. In columns (4) and (9), we add dummy variables for each value of the most current L-W score. In column (5), we add controls for whether the respondent has ever been diagnosed with an MRD and whether they will eventually report diagnosis (in subsequent survey waves). Columns (6)-(9) are estimated only on those never diagnosed with an MRD during the period they are observed. Standard errors are clustered at the individual level. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table A22: Relationship between Genetic Endowments and Parental Diagnosis of Memory-Related Disease

	Probability of Parent Ever Diagnosed with MRD								
	Full Sample					Never Diagnosed			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
AD Score	0.003	0.003	0.004*	0.004*	0.004*	0.004	0.004	0.005**	0.005**
	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)
APOE (At least 1 copy)	0.023***	0.023***	0.022***	0.023***	0.023***	0.021***	0.021***	0.022***	0.022***
	(0.005)	(0.005)	(0.005)	(0.005)	(0.005)	(0.005)	(0.005)	(0.005)	(0.005)
APOE (2 copies)	0.001	0.001	0.001	0.001	0.001	0.010	0.010	0.009	0.009
	(0.014)	(0.014)	(0.014)	(0.014)	(0.014)	(0.016)	(0.016)	(0.016)	(0.016)
EA Score		0.001	-0.002	-0.002	-0.002		0.001	-0.001	-0.001
		(0.002)	(0.002)	(0.002)	(0.002)		(0.002)	(0.002)	(0.002)
Ever MRD					0.010				
					(0.018)				
Eventual MRD					0.002				
					(0.011)				
L-W Score Dummies	No	No	No	Yes	Yes	No	No	No	Yes
Education Controls	No	No	Yes	Yes	Yes	No	No	Yes	Yes
Mean	0.070	0.070	0.070	0.070	0.070	0.070	0.070	0.070	0.070
N	23,065	23,065	23,065	23,065	23,065	22,041	22,041	22,041	22,041
R^2	0.026	0.026	0.030	0.031	0.031	0.025	0.025	0.029	0.030
Years	1998-2018	1998-2018	1998-2018	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	50-70	50-70	50-70

Note: Each column presents results from a separate regression where the outcome is an indicator for whether the respondent's mother or father has ever been diagnosed with a memory-related disease (MRD). The sample excludes individuals after they first report their parent having an 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 (7), we add the educational attainment (EA) polygenic score. In columns (3) and (8), we add controls for educational attainment. In columns (4) and (9), we add dummy variables for each value of the most current L-W score. In column (5), we add controls for whether the respondent has ever been diagnosed with an MRD and whether they will eventually report diagnosis (in subsequent survey waves). Columns (6)-(9) are estimated only on those never diagnosed with an MRD during the period they are observed. Standard errors are clustered at the individual level. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table A23: Relationship between Genetic Endowments and Parental Use of Nursing Home Care

	Probability of Parent Receiving Nursing Home Care								
	Full Sample					Never Diagnosed			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
AD Score	0.000	0.002	0.003	0.002	0.001	0.003	0.004	0.005	0.004
	(0.009)	(0.009)	(0.009)	(0.009)	(0.009)	(0.010)	(0.010)	(0.010)	(0.010)
APOE (At least 1 copy)	0.028	0.029*	0.028	0.029	0.029	0.028	0.029	0.029	0.029
	(0.017)	(0.017)	(0.018)	(0.018)	(0.018)	(0.018)	(0.018)	(0.018)	(0.018)
APOE (2 copies)	0.112**	0.111**	0.111**	0.118**	0.117**	0.114**	0.113**	0.113**	0.117**
	(0.052)	(0.051)	(0.051)	(0.051)	(0.051)	(0.054)	(0.053)	(0.053)	(0.053)
EA Score		0.023***	0.015*	0.014*	0.015*		0.023***	0.014*	0.014
		(0.008)	(0.008)	(0.008)	(0.008)		(0.008)	(0.008)	(0.008)
Ever MRD					-0.077				
					(0.126)				
Eventual MRD					0.118				
					(0.120)				
L-W Score Dummies	No	No	No	Yes	Yes	No	No	No	Yes
Education Controls	No	No	Yes	Yes	Yes	No	No	Yes	Yes
Mean	0.379	0.379	0.379	0.379	0.379	0.377	0.377	0.377	0.377
N	4,344	4,344	4,344	4,344	4,344	4,200	4,200	4,200	4,200
R^2	0.061	0.063	0.075	0.083	0.084	0.061	0.063	0.075	0.085
Years	2006-2018	2006-2018	2006-2018	2006-2018	2006-2018	2006-2018	2006-2018	2006-2018	2006-2018
Ages	50-70	50-70	50-70	50-70	50-70	50-70	50-70	50-70	50-70

Note: Each column presents results from a separate regression where the outcome is an indicator for whether the respondent's mother or father has ever received nursing home care. The sample includes individuals in the latest wave observed between ages 50-70. 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 (7), we add the educational attainment (EA) polygenic score. In columns (3) and (8), we add controls for educational attainment. In columns (4) and (9), we add dummy variables for each value of the most current L-W score. In column (5), we add controls for whether the respondent has ever been diagnosed with an MRD and whether they will eventually report diagnosis (in subsequent survey waves). Columns (6)-(9) are estimated only on those never diagnosed with an MRD during the period they are observed. Standard errors are clustered at the individual level. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.