NBER WORKING PAPER SERIES

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Working Paper 32181
http://www.nber.org/papers/w32181

NATIONAL BUREAU OF ECONOMIC RESEARCH
1050 Massachusetts Avenue
Cambridge, MA 02138
March 2024

We gratefully acknowledge comments from Julie Bynam, Weiwei Chen, Norma B. Coe, Nicole Maestas, Lauren Nicholas, Daniel Polsky, Kosali Simon, Jon Skinner, and seminar and conference participants at the NBER Summer Institute-Aging, the CEPRA/NBER Workshop on Aging, Cognitive Ability, and Decision-Making, the CAPRA/NBER Scientific Meeting, the Southeastern Health Economics Study Group Conference, the ASSA Annual Meeting, the Hopkins Economics of Alzheimer's Disease and Services (HEADS) Center, Colorado State University, George Mason University, and the University of Georgia. Research reported in this paper was supported by the National Institute on Aging of the National Institutes of Health under Award Number P30AG012810. Kevin Thom acknowledges additional support from the National Institute on Aging of the National Institutes of Health (R01AG079554, RF1055654, and R56AG058726). The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health or the National Bureau of Economic Research.

Genetic Risk for Alzheimer's Disease and Related Dementias: Cognition, Economic Behavior, and Clinically Actionable Information

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NBER Working Paper No. 32181
March 2024
JEL No. D14,G51,G52,I12,I14,J14,J22,J26

ABSTRACT

Genetic factors play a major role in the development of Alzheimer's disease and related dementias (ADRD). Observable genetic factors could impact household planning and medical care if they contain actionable information, meaning that they i) are associated with significant harms, ii) reflect risks for which individuals are not already prepared, and iii) are informative above and beyond current knowledge or expectations. We examine these properties for existing genetic measures related to ADRD in the Health and Retirement Study (HRS). We replicate existing relationships between genetic factors and cognitive health. We also show that higher genetic risk is associated with worse economic outcomes on several dimensions including work, income, and wealth. Surprisingly, individuals at higher risk are less likely to engage in planning activities that could mitigate the consequences of cognitive decline (e.g. assigning durable power of attorney). In predictive exercises, existing genetic indices provide clinically valuable and policy-relevant information on the development of severe adverse cognitive outcomes in the future.

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1 Introduction

As of 2022, 6.5 million Americans aged 65 and over have been diagnosed with Alzheimer's disease and related dementias (ADRD), and this number is projected to reach 12.7 million by 2050 (Alzheimer's Association 2022). The costs associated with ADRD 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, Skira, and Larson 2018, Nicholas et al. 2021). The direct medical costs alone reached $321 billion in 2022 (Alzheimer's Association 2022). Alzheimer's disease (AD), the most common cause of dementia, is a slowly progressive brain disease that includes three phases: pre-clinical AD, mild cognitive impairment, and Alzheimer's dementia.[1] 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, Coile, and Mommaerts 2023). AD is typically clinically diagnosed during this third phase.[2] 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, early detection and long-run planning may be important for at-risk individuals to safeguard themselves and their families from the associated medical, financial, and legal burdens. In particular, it is important to understand when and how ADRD affects cognitive and economic outcomes. Indeed, a large literature documents changes in economic and health outcomes that precede an eventual ADRD diagnosis or the onset of dementia. For example, Nicholas et al. (2021) find that those eventually diagnosed with ADRD show evidence of financial mistakes well before diagnosis, and Li et al. (2023) show that household wealth declines faster among those with probable dementia than among those without in the decade before dementia onset. Such analyses are important because they contribute to our understanding of the

[1] ADRD refers to the set of debilitating neurodegenerative conditions that impair memory, thought processes, and functioning. AD is the most common cause of dementia with hallmark brain changes that include the accumulation of protein beta-amyloid (plaques) and tau tangles. Other forms of dementia include Lewy body dementia, frontotemporal dementia, and vascular dementia, among others, and they often co-occur with AD. These other forms of dementia share many cognitive and pathological features with AD and can be difficult to distinguish from AD.

[2] 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 (Jack Jr. et al. 2018). The National Institute on Aging and the AlzheimerâĂŹs Association spell out core clinical diagnostic criteria for AD in Albert et al. (2011), McKhann et al. (2011), and Sperling et al. (2011).

cumulative life-cycle costs of ADRD, and they potentially suggest early-warning predictors. 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 some important limitations. First, it may be challenging to practically implement predictive systems based on these insights if they require the simultaneous tracking of several streams of real-time financial, economic, and health data. Second, these predictors may have limited value if they only signal that an individual is at high risk after they start exhibiting signs of financial or medical hardship. Third, studies that ascertain risk factors from case-control approaches could be biased because they select on an outcome variable (i.e., eventual diagnosis or eventual dementia) to determine cases and controls. Since an ADRD diagnosis, for example, is itself endogenous (e.g., it depends on the choice to seek medical attention), predictors of this outcome will reflect both early-warning signs for ADRD and factors affecting the propensity to seek medical attention.

In this paper, we study the relationship between observed genetic risk for ADRD and trajectories of cognition, memory-related disease, economic outcomes, planning activities, and awareness of risk using the Health and Retirement Study (HRS). Focusing on ex-ante genetic risk is natural in this context because genetic factors play a large role in the etiology of ADRD. Twin studies suggest that genetic factors explain 60–80% of the variation in the risk of AD (Gatz et al. 2006). Unlike eventual diagnosis or eventual dementia, genetic factors are pre-determined at conception, and can be observed earlier in life—certainly well before any clinical evidence of ADRD. The molecular genetic data available in the HRS allow us to study how cognitive and economic outcomes and planning activities evolve across individuals with different levels of genetic risk for ADRD, including those who never receive a memory-related disease diagnosis or develop dementia.

We ask whether the molecular genetic measures of ADRD risk contain *actionable* information for financial, legal, and medical planning. Genetic measures—even those that predict significant cognitive decline—are not automatically valuable for planning purposes. From an economic perspective, we argue that personal genetic information will be actionable if it meets three important conditions. First, such measures must reflect a risk of real harm. It is important to know whether the cognitive decline predicted by genetic factors is actually associated with changes in outcomes that impact welfare (e.g., ability to perform in the labor market). Some individuals could be *insulated* from the harm of cognitive decline if, for example, their productivity at work or decision-making is not affected by the kind of impairment present in early- or mid-stage ADRD. Second, genetic information will be useful if individuals are not already *prepared* to deal with risks predicted by such information. Even if genetic measures predict a higher rate of cognitive decline, the results of genetic tests might not affect welfare if individuals already take the kinds of

financial and legal precautions that would help in the event of cognitive decline. Finally, in order to be actionable, genetic measures must be *informative* about future cognitive decline beyond what would already be predicted by demographics, family background, and existing health.

Given the complex genetic architecture of ADRD, we study three sets of genetic markers. 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 ADRD (hereafter the AD score), which is a linear index of genetic markers associated with ADRD (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). Indeed, genetic markers that predict educational attainment have been shown to also predict ADRD and cognitive decline (Ding et al. 2019, Anderson et al. 2020). 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 ADRD, 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 replicate established associations showing that increased genetic risk for ADRD, whether measured by the AD score, the EA score, or carrying an APOE-$\varepsilon 4$ 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. We show that these patterns hold even among those who are never diagnosed with a memory-related disease while observed in the HRS. One implication of this finding is that high-risk individuals who were never diagnosed 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.

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 second 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. These findings provide novel evidence that higher polygenic risk for ADRD is economically relevant even when clinical presentations of ADRD are absent.

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 third 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. They also hold among those who never exhibit cognitive function consistent with dementia while observed in the HRS. 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-ε4 carriers are more likely to engage in planning activities.

Our fourth set of findings provide an explanation for the differences in planning behavior by ADRD genetic risk. APOE-ε4 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 ADRD, 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.

Fifth, critically, we demonstrate that the genetic measures studied here are *informative* and may have 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-ε4 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 and family background. That is, the genetic measures contain information beyond what is likely available to an individual's primary care physician. Furthermore, the literature on revelation of ADRD genetic risk focuses on APOE carrier status, but our results underscore that total genetic risk for ADRD matters. We find that a non-trivial fraction of APOE non-carriers have overall risk for eventual severe adverse cognitive outcomes that is on par with or greater than many APOE carriers due to their polygenic score for ADRD.

Our work contributes to several literatures. First, we add to the literature on associations

between cognitive decline and household economic outcomes, which is surveyed by Chandra, Coile, and Mommaerts (2023). Several studies in this area show that the development of dementia is preceded by worsening financial outcomes (e.g., Triebel et al. 2009, Sudo and Laks 2017, Angrisani and Lee 2019, Martin et al. 2019b, Gresenz et al. 2020, Nicholas et al. 2021, Li et al. 2022, 2023, Mazzonna and Peracchi forthcoming).[3] Some evidence suggests that households respond to cognitive decline by making adjustments, including changing who is in control of (or at least who reports on) household finances (Hsu and Willis 2013). Studies in this literature generally measure dementia or impairment based on an eventual ADRD diagnosis or an eventual score on a diagnostic cognitive test. By contrast, our study examines associations between economic outcomes and ex-ante genetic risk for ADRD. This is important for two reasons. Since genetic risk is observable, our results highlight the potential usefulness of genetic measures as a clinical or personal tool for predicting future financial difficulties well in advance of actual cognitive decline. Additionally, the fact that gene-outcome associations survive even when controlling for current cognitive function further suggests that genetic risk for ADRD may operate through channels that start affecting household outcomes before cognitive difficulties appear.

We also contribute to the literature on the use of genetic markers as predictors of cognitive decline and ADRD. A substantial literature predicts ADRD and related outcomes on the basis of APOE carrier status as well as measures of polygenic risk for ADRD (e.g., Escott-Price et al. 2015, Ajnakina, Cadar, and Steptoe 2020, Sims, Hill, and Williams 2020, Zhang et al. 2020, Leonenko et al. 2021, Stocker et al. 2021, Vacher et al. 2022, Gao et al. 2023). There is also evidence that polygenic indices predict worse cognitive outcomes and the presence of more ADRD-related biomarkers even among cognitively healthy adults (Mormino et al. 2016, Tan et al. 2019, Daunt et al. 2020, Kauppi et al. 2020, Kumar et al. 2021, Skoog et al. 2021) as well as faster transitions from mild cognitive impairment to late-onset AD (Chaudhury et al. 2019, Daunt et al. 2020). Less studied are the associations between genetic risk for ADRD and economic outcomes or planning activities that suggest whether individuals are aware of their genetic risk. Exceptions include Wehby, Domingue, and Wolinsky (2018), who report a negative association between a polygenic score for ADRD and household wealth, and Shin, Lillard, and Bhattacharya (2019), who find that higher polygenic risk for ADRD is associated with less savings in active investments (e.g., IRAs) and more saving in passive investments, and that these associations emerge as individuals age. Our results make two contributions to this literature. First, we provide a novel combination of results that establishes polygenic risk for ADRD as a potentially valuable source of information for households and clinicians. Poly-

[3] A broader literature also assesses the more general relationship between cognitive performance and financial outcomes and choices at all ages (Christelis, Jappelli, and Padula 2010, Agarwal and Mazumder 2013).

genic indices will have limited usefulness if they convey information about genetic risk that either does not affect cognition and economic outcomes or which is already fully understood and accounted for in household preparations. We find that not only is higher polygenic risk associated with lower cognitive performance and economic resources, it is also *negatively* associated with a broad suite of financial and legal planning outcomes not previously studied in this literature. This suggests that a better understanding of polygenic risk may be useful for households. Second, we demonstrate the practical clinical value of existing polygenic scores. In particular, we show that measures of polygenic risk predict future cognitive decline even after controlling for current and past cognitive performance. Controlling for an observable history of cognition is important for assessing clinical usefulness, since it comes closest to replicating the information sets likely available to households and physicians when making choices about financial and legal preparation or preventative action.

Finally, our work is related to the literature on individual beliefs about own risk for ADRD and the consequences of revealing biomarker or genetic test results indicative of this risk. Studies such as Zick, Smith, and Mayer (2016) demonstrate that individuals with family members diagnosed with AD are significantly more likely to explore professional financial planning services and less likely to plan for an early retirement. Several studies estimate the effects of randomized controlled trials that reveal APOE carrier status. These studies have found that communication of APOE carrier status is associated with changes in expectations and financial plans (Zick et al. 2005, Chao et al. 2008, Taylor et al. 2010, Bemelmans et al. 2016, Largent et al. 2021). Although we do not exploit random variation in information provision, our results contribute to this literature by assessing the scope for increased knowledge on polygenic risk to influence household outcomes. Furthermore, our results highlight a critical distinction between APOE status (studied in the information revelation literature) and polygenic risk for ADRD. Indeed, we find that APOE carriers show some evidence of greater planning activities and awareness of their own elevated risk. By contrast, those at higher polygenic risk are systematically less likely to take specific actions to prepare for future ADRD, which could arise if it is more difficult to infer higher polygenic risk versus APOE carrier status.

2 Background on Genetic Risk for Alzheimer's Disease and Related Dementias

We measure ADRD 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.[4] At each location in the genome, individuals can possess one of two possible base pairs: an adenine-

[4]The discussion in this section is similar to those in Barth, Papageorge, and Thom (2020) and Papageorge and Thom (2020).

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 each parent, individuals can possess 0, 1, or 2 copies of a particular molecule (AT or GC) at each SNP.

Our first molecular genetic measure for ADRD is APOE-ε4 (hereafter APOE) carrier status.[5] 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 risky APOE allele generally exhibit the brain changes and cognitive symptoms associated with AD earlier than non-carriers. The APOE allele is found in more than half of diagnosed AD patients (Michaelson 2014), but being a carrier is neither necessary nor sufficient to develop AD. A vast medical literature studies the function of APOE and its possible role in the etiology of AD. Significant evidence links APOE carrier status to an increased presence of AD-related biomarkers, including elevated levels of beta-amyloid plaques between brain cells (Fan et al. 2019).

While APOE has received a great deal of attention, it 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 these variants using a *polygenic score for AD* (the 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

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

genetic risk for the trait or outcome.[6]

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. We recognize that this score is a noisy measure of underlying genetic risk for AD. First, the coefficients used to the construct these scores are estimated from limited samples and thus contain estimation error. Second, even in the absence of estimation error, noise can be introduced into the score because of errors or inconsistencies in the classification of cases and controls. In their discovery sample, Kunkle et al. (2019) pool data from 46 different cohorts with AD cases determined by various clinical procedures depending on the cohort (e.g., MRI results, biomarkers, cognitive testing, autopsy findings, etc). Consequently, there may be measurement error in the AD phenotype used in the GWAS. Different diagnostic procedures can generate different rates of classification error with some studies finding that 10–30% of individuals clinically diagnosed with AD-related dementia while alive did not display AD pathology changes at autopsy. Thus, the AD score may capture genetic risk for cognitive decline and dementia not due specifically to AD, hence our emphasis throughout on genetic risk for ADRD. This is not a problem for our analysis as, fundamentally, we are interested in genetic risk for severe cognitive decline and its associations with cognitive performance, economic outcomes, planning activities, and awareness of risk of decline.[7] 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 ADRD.[8] 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 (the 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 acquire 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 ADRD. Indeed, studies show that SNPs and

[6]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, Papageorge, and Thom (2020) and Papageorge and Thom (2020).

[7]We caution, however, that using the AD score to target medical treatments that address the underlying brain changes of AD is unwise given the likely measurement error in AD status in the GWAS.

[8]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.

polygenic scores discovered to predict educational attainment also predict ADRD and cognitive decline (Ding et al. 2019, Anderson et al. 2020). Moreover, the EA score has been shown to predict many of the outcomes we study here, such as income, labor supply, and wealth (Belsky et al. 2016, Barth, Papageorge, and Thom 2020, Papageorge and Thom 2020).

The use of genetic data has a number of benefits and drawbacks, many of which have been thoroughly discussed elsewhere. We provide a brief summary here. First, the measures tend to be noisy in that they only capture a fraction of the variance in outcomes attributable to genes. This could be due to limited power in the GWAS to detect rare variants or variants with small association sizes. 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 (Martin et al. 2017, 2019a), which means we are limited to studying individuals of European descent.[9] In general, these difficulties suggest caution when interpreting our estimated coefficients. Our analyses are descriptive, which can provide guidance for future analyses, including plausible interactions with environmental factors and 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, beyond the variables that capture current information sets of clinicians.

3 Data

We use data from the Health and Retirement Study (HRS), which follows a nationally representative sample of adults age 51 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

[9] Applying a score constructed from one ethnic group to another group can vastly over- or under-estimate predicted likelihood of an outcome due to statistical artifacts, such as differences across groups in how SNPs correlate to one another. We thus refrain from doing so, as is common practice. Barth, Papageorge, and Thom (2020) and Papageorge and Thom (2020) provide further discussion of this issue in the context of the EA score.

(2006, 2008, 2010, 2012). Our sample only includes these genotyped individuals.[10] 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 various measures of genetic risk for ADRD. First, we use a polygenic score for AD based on the Kunkle et al. (2019) GWAS that 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. Second, we consider whether an individual carries the APOE allele and create two APOE-related indicators. The first takes value one if the individual carries at least one copy of APOE-ε4, and the second takes value one if the individual has exactly two copies of the allele.[11] Third, 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.

3.1.2 Direct Outcomes: Cognition and Memory-Related Disease Diagnosis

We examine how genetic risk for ADRD associates with directly-related outcomes, namely cognitive functioning and diagnosis of memory-related disease. We rely on a summary cognition 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 memory and cognition. Our primary measure of cognitive function is a 27-point score that includes the following tests: (1) immediate and delayed recall (0–20 points); (2) serial sevens subtraction (0–5 points); (3) counting backward (0–2 points). This measure is a modified version of the Telephone Interview for Cognitive Status (TICS), which we refer to as the TICS-M score. Previous work has shown performance on the TICS to be highly correlated with the Mini-Mental State Examination (MMSE), which is richer but more

[10] Genotyped individuals were not provided information about their genome, including their genetic risk for Alzheimer's disease. By contrast, the HRS began collecting biomarker information in 2006 and notified respondents about certain measures, such as blood pressure and total cholesterol.

[11] 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.

difficult to implement (Fong et al. 2009). Specific ranges of the 27-point TICS-M score in the HRS (the Langa-Weir classifications) have been shown to accurately identify cognitive impairment and dementia (Crimmins et al. 2011). 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.[12] We study the TICS-M score itself and create indicators for whether an individual has ever achieved a score that corresponds with the impaired or demented categories based on the Langa-Weir classifications, 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.[13] 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

[12] In our analysis, we only include person-wave observations of self-respondents 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 TICS-M score (Gianattasio et al. 2019). The three tests included in the 27-point TICS-M score are asked of individuals of all ages, whereas other tests are only asked to those aged 65 and older; hence, we prefer the 27-point TICS-M score as it is consistently measured across the ages we study. Additional information on the Langa-Weir classifications can be found here.

[13] Household wealth is 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).

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 ADRD correlates with expectations about mortality, long-term care utilization, and developing AD or dementia. The HRS asks respondents aged 65 and under about their expected probability of living to age 75 on a scale of 0–100. Starting in 1998, the HRS asks individuals aged 65 and older who do not currently reside in a nursing home 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. 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.[14] 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 or dementia.

Individuals may also learn about their risk for ADRD via their parents' development of the disease or use of long-term care. We therefore examine how own genetic risk for ADRD 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. Questions about parental MRD were not asked until 1998 and are only asked if that parent is currently alive. Parental nursing home care is determined via questions about where and with whom a parent currently resides (if he/she is 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 for individuals aged 50–85 between 1998–2018 who are genotyped and self-respondents (as opposed to proxy respondents). We exclude proxy

[14] Additional experimental modules 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 AlzheimerâĂŹs 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?"

respondents as there is no direct assessment of the individual's cognition, and we do not observe many of the outcomes we consider when the interview occurs via proxy.[15] Analysis sample sizes fluctuate across regressions, and the age range of our analysis samples vary depending on the outcome we consider. For example, for employment and retirement 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 to only certain age groups or during specific periods. Moreover, for some analyses, we limit the sample to ages 50–70, prior to widespread onset of ADRD, 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 ADRD and the probability of ever being cognitively impaired, we drop observations after the first wave in which they register a TICS-M score less than 12.

3.3 Summary Statistics of Key Variables

Summary statistics for key variables are found in Table 1. We calculate them using the maximum number of person-year observations available for each variable, which is why the sample size varies. Average birth year is 1940, about 42% 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 genotyped HRS respondents, some of whom were dropped from the analytic sample. The mean AD score is slightly below zero, reflecting that people at higher risk of ADRD exit the sample (through survey attrition, proxy interview, and/or death). We address concerns about attrition in Section 5.5.1. 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 ADRD.

The distributions of the AD and EA polygenic scores are found in Figures 1 and 2, respectively. Both scores are symmetric around the mean and seem to be normally distributed. Moreover, Figure 3 Panel (a) and Figure 4 show the joint distribution between the AD and EA scores in different ways. Panel (a) of Figure 3 plots the densities of the AD score conditional on quintiles of the EA score, while Figure 4 presents a bin-scatter plot of the AD and EA scores. The key takeaway is that, while there is a correlation between the scores, it is very low (roughly 0.045).

[15] Proxy interviews usually arise when the HRS respondent is unable to complete an interview due to physical or cognitive limitations. Conditional on being a self-respondent in our sample in a given wave, only 1 percent go on to have a proxy respondent in the next survey wave.

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 ADRD. An alternative possibility is that there is in fact greater overlap, but the GWAS did not capture it. For example, suppose there is a SNP that is both highly predictive of lower education and of ADRD diagnosis, but it is relatively rare. It may appear as important in the AD score, but there could be 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.

In Panels (b) and (c) of Figure 3, we show the AD and EA score distributions conditional on APOE carrier status. Those who do not carry APOE and those who carry one copy have similar AD and EA score distributions. For both groups for both scores, the mean is approximately zero with a standard deviation close to one. Among individuals who carry two copies of APOE, the AD score has mean -0.18 and standard deviation 0.91. In other words, the average person who carries two copies of APOE has a lower AD score than those who carry zero or one copy. Those who carry two copies of APOE also have slightly lower average EA scores, with mean -0.05 and standard deviation of one.

Turning to direct outcomes related to cognition and diagnosis among those aged 50–85, the average TICS-M score is 16.4. Slightly less than 3% of the sample has or has had a TICS-M score low enough to be categorized as demented, while about 22% are impaired or demented. About 2% of the sample has received a memory-related disease diagnosis. Those statistics are measured at the person-wave level. At the individual level (not shown in the table), slightly less than 10% of individuals in our sample are ever diagnosed with MRD, about 40% ever register a TICS-M score consistent with impairment or dementia, and almost 10% ever register a score in the dementia range. Figure 5 examines how the relationship between genetic risk for AD and the TICS-M score evolves over the later life-cycle observed in the HRS. Panel (a) plots the unconditional average TICS-M score by age separately for those with above and below median values of the AD score, respectively. Modest differences in the average TICS-M 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 TICS-M 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 TICS-M 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% are 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 about $33,000 and average household wealth is almost $590,000.

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, on average, individuals engage in 2.7 of the six planning activities we consider.

Part of our analysis includes assessing to what degree individuals appear to be aware of their risk of cognitive decline. 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 AD or dementia 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 by age 70, almost 38% of respondents have a parent who received nursing home care.

Appendix Table A1 provides summary statistics for the non-genotyped HRS respondents that otherwise meet our sample selection criteria. On average, the genotyped sample is more educated, more cognitively healthy, has better economic outcomes, and is more likely to engage in planning activities. Thus, the genotyped sample is positively selected on human capital, cognition, economic resources, and later-life planning, which could attenuate negative associations between genetic measures of ADRD risk (namely the AD score and APOE carrier status) and the outcomes we study.

4 Empirical Strategy

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-$\varepsilon 4$ allele as well as a separate indicator for having two copies. In this way, we allow for non-linearities in the relationship between the number of APOE copies an individual carries and the outcomes of interest. $EAScore$ is the polygenic score for educational attainment. As in Barth, Papageorge, and Thom (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 (Price et al. 2006, Benjamin et al. 2012), and we allow those coefficients to vary by gender. We cluster standard errors at the individual level.[16]

In some specifications, we include additional individual-level controls. In particular, we add dummy variables for each value of the current TICS-M 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.[17]

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 TICS-M 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

[16] For binary outcomes, estimated marginal effects from probit models are nearly identical to the estimated coefficients from the linear probability models we present here. The probit results are available by request.

[17] In a causal inference framework, controlling for some of these variables could be interpreted as conditioning on "bad controls." Our aim is to show that the AD score predicts certain behavior and outcomes independently of these variables.

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 TICS-M score. A one standard deviation increase in the AD score is associated with a 0.24 point decrease in the TICS-M score from a mean of 16.4. Having at least one copy of APOE is associated with a TICS-M 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 TICS-M 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 ADRD. 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. Thus, 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 eventual 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 TICS-M score (by 2.13 and 1.52 points, respectively). Adding these variables moderately lowers the size of the coefficients on the APOE variables and 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 TICS-M score.

To explore further, we next focus on the subsample of individuals who are never diagnosed with MRD while observed in the HRS (columns 5–7).[18] In column (5), we return to a specification with only the three ADRD genetic variables. We find that among the undiagnosed, a one standard deviation increase in the AD score is associated with a 0.23 point decline in the TICS-M score. Having at least one copy of APOE lowers the TICS-M 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)

[18]While we limit the analysis sample to those aged 50–85, we use information beyond age 85 to determine whether an individual is diagnosed with MRD during the HRS sample period.

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 TICS-M score. These relationships are somewhat diminished when we add a full set of education controls in column (7) but remain substantial and significant—among the undiagnosed, a one standard deviation rise in the AD score predicts a decline of 0.12 points in the TICS-M score and a corresponding increase in the EA score predicts a 0.24 point increase.

To explore whether the genetic associations with the TICS-M score reflect substantial differences in clinical outcomes, we replace the outcome variable with a dummy variable for ever having a TICS-M 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 TICS-M 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.[19]

We also examine whether genetic endowments related to ADRD predict memory-related disease diagnosis. The outcome is an indicator for whether an individual is ever diagnosed, and we exclude individuals 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 ADRD, 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 ADRD 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.

The results from Tables 2 and 3 replicate established associations demonstrating that increased genetic risk for ADRD, whether measured by the AD score, the EA score, or carrying an APOE-ε4

[19]We repeat this analysis with an indicator for ever being demented (i.e., scoring less than 7 on the TICS-M score) as the outcome, which is rare. Results are shown in Appendix Table A2. Carrying the APOE allele increases the probability of dementia non-trivially. A higher EA score is associated with lower risk. In some specifications, a higher AD score predicts higher dementia risk, but in specifications with educational attainment controls, the relationship attenuates and is no longer significant.

allele, predicts lower cognitive performance, higher rates of impairment, and higher rates of MRD diagnosis. The relationships between genetic endowments and cognitive performance hold even among individuals who are never diagnosed with MRD, which runs counter to the idea that those without a diagnosis but a high genetic risk of ADRD are somehow protected from cognitive decline or are able to avoid significant impairment. 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 ADRD. The lessons we learn about ADRD are often 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 ADRD apply solely to the population that is also diagnosed.

5.2 Economic Outcomes

The previous section provides evidence that genetic measures are predictive of diagnosis. The results also suggest that individuals who are never diagnosed but are at high risk of ADRD exhibit worse cognitive function. Their lack of diagnosis does not mean they are somehow protected from decline. Nevertheless, it might be the case that people at high risk of ADRD who exhibit cognitive decline are somehow insulated from the negative consequences, which would perhaps explain a lack of diagnosis.

To explore this question, we examine how genetic endowments related to ADRD predict economic outcomes. We start with whether or not an individual works for pay. Results are in Table 4 and are presented following a similar progression as in Table 2 in Section 5.1 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 TICS-M 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 TICS-M 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. Overall, the results from Table 4 provide evidence against the idea that lack of MRD diagnosis is due to individuals somehow being insulated 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 lower cognitive performance and its economic consequences but who never receive a diagnosis.

We examine a host of other economic outcomes, including retirement, log individual total income, and log household total wealth. We provide a summary in Table 5, only showing results from the specifications that correspond to columns (4) and (9) in Table 4. The full set of results for each outcome are presented in Appendix Tables A3–A5. 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 TICS-M 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.[20] These findings provide novel evidence that higher polygenic risk for ADRD is economically relevant even when clinical presentations of ADRD are absent.

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 4 are presented in Table 6.[21] 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. Exploring the mechanisms underlying the wealth results is a promising avenue for future work. We do not find evidence of gender heterogeneity in the association between the EA score and household wealth.

[20] We repeat the analysis for the working for pay and retirement outcomes for those aged 50–75. The point estimates are nearly identical, and if anything, precision improves when we additionally include those between ages 70–75.

[21] The full set of results are presented in Appendix Tables A6–A8.

5.3 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 ADRD associates with a variety of later-life planning activities, including having LTCI, life insurance, a witnessed will, a living will (i.e., an advance care directive), or a durable power of attorney for health care and having 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.[22] After age 70, cognitive decline becomes increasingly evident for some people, in which case a correlation between planning activities and genetic risk for ADRD might not reflect planning at all but instead a reaction to illness. Results corresponding to columns (4) and (9) of Table 4 are presented in Table 7.[23]

We find that an increase in the AD score is associated with statistically significant declines in the probability of engaging in most of the 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, although 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 8, 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 in which they engaged. A one standard deviation 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

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

[23] The full set of results for each planning activity are presented in Appendix Tables A9–A14.

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.

One possible explanation for the decreased engagement in planning activities among those with higher AD scores that is consistent with our collection of results so far is that these individuals have fewer or no financial resources to protect. For example, for individuals without wealth, there is less incentive to have a witnessed will or long-term care insurance. To shed light on this idea, we re-estimate the planning activity specifications only on those with positive housing wealth or positive overall wealth. The results, which are available by request, are nearly identical to those estimated on the full sample. Thus, the negative association between planning activities and the AD score does not seem to be driven by high-risk individuals not having financial resources to protect.

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 ADRD 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 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.

5.4 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 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 ADRD correlates with self-reported expectations about mortality, future nursing home use, and future development of AD or dementia. Given the strong heritability of ADRD, 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 4 are presented in Table 9.[24]

In columns (1) and (2) of Table 9 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

[24]The full set of results for each outcome are presented in Appendix Tables A15–A20.

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 with the idea that they 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 9. Those who carry at least one copy of APOE report a statistically significant 7 percentage point increase in their self-reported probability of developing ADRD. 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 particular 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 ADRD in the future.[25]

Taken together, these results suggest that some APOE carriers are aware of their elevated risk status, while, on average, those with higher AD scores are not. It is possible that APOE carriers receive stronger signals of their risk if their parents experienced cognitive decline or were

[25]In Appendix Table A18, 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 ADRD.

diagnosed with ADRD. 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 9. 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. Questions about parental MRD were not asked until 1998 and are only asked if a parent is currently alive. Thus, our estimates are likely conservative, as parental MRD is understated for respondents who have one living cognitively healthy parent but the other parent had MRD and died before the 1998 survey wave (or before the respondent's initial interview). We do not observe parental MRD at all for respondents without living parents as of the 1998 wave (or their initial survey).

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 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 9 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 ADRD 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.

5.5 Robustness and Sensitivity

5.5.1 Sample Attrition

The estimated associations between genetic risk for ADRD and the various outcomes we consider are likely conservative. For a number of reasons, the analysis sample is positively selected on health and cognition. First, individuals must survive until at least 2006 in order to be genotyped. Second, we exclude person-wave observations when the interview occurred via proxy as there is no direct assessment of the individual's cognition, and many of the outcomes we consider are not observed. We explore how genetic risk for ADRD associates with sample attrition overall and attrition due to proxy interviews as well as death. We create an indicator that has value one if a self-respondent appears in the following wave as a self-respondent and zero otherwise. If an individual does not self-respond next wave, they either responded via proxy, passed away, or did not respond to the survey for other reasons. In Appendix Table A21, we show associations between the genetic endowments and sample attrition for the full sample and among those aged 50–70. Among individuals aged 50–85, APOE carriers and those with higher AD scores are less likely to self-respond in the next survey wave, while higher EA scores associate with a higher probability of being observed next wave. These relationships somewhat attenuate when controls for current cognition are included, and they dampen and are less precisely estimated among those aged 50–70.

We next consider whether the relatively higher sample attrition among those at higher genetic risk for ADRD is due to having a proxy respondent next wave. Results are presented in Appendix Table A22. Among those aged 50–85, APOE carriers have a higher probability of having a proxy interview next wave, while higher EA scores associate with a lower probability of a proxy interview. These patterns again attenuate when controls for current cognition are included and weaken substantially among the never diagnosed sample and among those aged 50–70, groups for which proxy interviews are especially rare. Thus, proxy interviews play some role in explaining higher attrition from the analysis sample among those at higher AD genetic risk but largely for those over age 70. Using information from HRS exit interviews, we create a mortality indicator equal to one if the individual dies before the next survey wave and zero otherwise. The point estimates presented in Appendix Table A23 are not statistically significant, but the overall pattern weakly suggests that some of the increased sample attrition for those with higher ADRD genetic risk is due to increased mortality. The remaining explanation for the sample attrition is survey non-response. Overall, the results suggest those with higher ADRD genetic risk are less likely to consistently respond to the survey. If anything, this would tend to make the estimated relationships between genetic risk and the outcomes we consider conservative.

5.5.2 Relationships Among the Never Demented

We have shown that those with higher AD scores have worse economic outcomes, are less likely to engage in later-life planning activities, and seem to be less aware of their elevated risk for ADRD. The genetic measures have predictive power even among those who are never diagnosed with MRD and when controlling for current cognitive performance. Next, we perform a more stringent test of the predictive power of the genetic measures and examine whether these relationships hold among a relatively cognitively healthy group. To do so, we limit the sample to those who are never demented (TICS-M score below 7) and never have a proxy interview while observed in the HRS. We simply refer to this group as "never demented." We show tables analogous to Tables 5, 7, and 9 and replace the "never MRD" columns with results from the "never demented" sample. The results are shown in Tables A24–A26. While precision weakens in a few cases, overall, the results among the never demented sample are very similar to those among the full sample and the never diagnosed. These findings further underscore that higher genetic risk for ADRD is relevant even in the absence of clinical presentations of ADRD.

6 Assessing the Clinical Value of Molecular Genetic Measures

The literature on the clinical or planning value of genetic markers for ADRD has largely centered on the disclosure of APOE carrier status. This focus makes sense given the large differences in AD risk across APOE genotypes. However, with the development and refinement of polygenic measures of ADRD risk (excluding APOE), it is natural to ask what such measures might add to screening and counseling activities. Here we ask whether polygenic indices can identify meaningful differences in predicted dementia risk (compared to APOE status, for example) and whether they can productively supplement or contextualize counseling on APOE genotype status. Providing information on total ADRD genetic risk (i.e., risk due to all genetic factors and not just APOE) may be important given evidence that the communication of genetic risk can have complicated effects on behavior and mental health. For example, results from randomized information experiments provide evidence that individuals change their expectations and planning activities after learning their APOE type (Zick et al. 2005, Chao et al. 2008, Taylor et al. 2010, Bemelmans et al. 2016, Largent et al. 2021).

The results from Section 5.1 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. For these measures to be valuable for these tasks, they

need to predict risk for future ADRD above and beyond the kind of data and medical history that a physician would already have at his or her disposal. That is, it would be ideal to ask whether the genetic measures predict future ADRD 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 investigate whether the genetic measures predict outcomes related to dementia 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 a TICS-M score less than 12 (impairment or dementia) and who are not diagnosed with MRD in the age range 50–64. We flexibly control for the individual's cognitive performance during this period as well as indicators for whether or not their parents were ever diagnosed with a memory-related illness.[26] This approximates the information set likely available to an individual, their family, or their doctor. We then estimate regressions that predict outcomes related to dementia in the later age range of 65–80. These regressions are different from our basic model in Equation 2 because they are essentially cross-sectional models of "late period" outcomes with explanatory variables restricted to only include information from the "early period." 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, along with its interactions with all of the previous regressors. Importantly, we add controls for the average TICS-M score, the minimum TICS-M score, and the maximum TICS-M score as well as parental memory-related disease diagnosis during the "early period."

We examine a set of five binary outcomes in the "late period": 1) at least one period of impairment, 2) at least one period of dementia, 3) a self-reported diagnosis of MRD, 4) having at least one interview wave completed by a proxy, and 5) having at least one severe cognitive outcome (dementia, MRD diagnosis, or a proxy interview). We examine proxy response as an outcome because individuals who are sufficiently impacted by cognitive decline may be unable to participate in the survey, electing instead to have a spouse or caretaker provide answers. The "severe cognitive outcome" binary offers the most comprehensive assessment of acute cognitive difficulties in the late period. Considering such a composite is important because many individuals do not progress slowly or smoothly through impairment, dementia, and diagnosis. Some may be diagnosed without ever registering a TICS-M score consistent with dementia, either because of sampling variation, missing data, or because a diagnosis was made on the basis of a more thorough cognitive assessment. For example, only 32% of individuals who are eventually diagnosed at ages

[26]For reasons mentioned earlier, information on parental diagnosis is missing for about half of the sample. We therefore set parental diagnosis to zero for those with missing parental information and separately control for an indicator for missing parental information.

65–80 are observed with a cognitive test score consistent with dementia during the late period.

Table 10 reports the estimated coefficients on the genetic predictors from the specification described above. We find that the AD score significantly predicts future impairment, dementia, diagnosis, and proxy status conditional on early period 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, with the exception of a marginally significant negative association when future impairment is the outcome of interest. The sizes of the coefficient estimates on the genetic variables are striking. Unsurprisingly, being an APOE carrier (especially having two copies) dramatically increases the risk for major cognitive problems in the future. While 2.8% of individuals in this sample are eventually observed with cognitive scores consistent with dementia (at ages 65–80), carrying one copy of APOE is associated with an increased risk of 2.8 percentage points (doubling mean risk), and carrying two copies is associated with an *additional* 4.4 percentage point increase. Alternately, consider whether an individual is ever observed to be demented, diagnosed, or have a proxy interview in the late period. About 8.6% of respondents in our prediction sample will eventually exhibit at least one of these more severe signs of decline in the late period. Carrying one copy of the risky APOE allele is associated with a 4.4 percentage point higher probability of this event, while carrying two risky alleles is associated with an *additional* increase of 11.8 percentage points.

The large differences in future cognitive health outcomes by APOE status certainly justify the attention that APOE receives as a focal point for genetic testing and personalized medicine. However, it is noteworthy that variation in the AD score also predicts meaningful differences in future cognitive health conditional on current cognition. A one standard deviation increase in the AD score is associated with a 0.7 percentage point higher chance of demented cognitive performance (2.8% sample prevalence) and a 1.8 percentage point increase (8.6% sample prevalence) in the probability of a severe cognitive outcome. Across the outcomes studied in Table 10, we find associations between a one standard deviation higher AD score and cognitive outcomes that are roughly 0.25 to 0.50 times the size of the association between these outcomes and carrying one risky APOE allele.

To assess the possible clinical relevance of molecular genetic predictors of ADRD, we compare estimates of the risk for a severe cognitive outcome (at ages 65–80) across two separate predictive models. First, we consider individual-level estimated risk for any severe cognitive outcome from the linear model in column (5) of Table 10, which incorporates molecular genetic information. We refer to the predicted value from this specification as $EstCogRisk_i^{Genes}$. We also re-estimate this model, but exclude measures of the AD score, the EA score, and the APOE dummies. We refer to the fitted values from this specification as $EstCogRisk_i^{NoGenes}$.[27] We assume that $EstCogRisk_i^{NoGenes}$

[27]Note that we retain the principal components, as they are meant to capture population stratification which

measures the objective clinical risk that one should expect in the absence of genetic data, but given knowledge of demographics (including education), current and past measures of cognitive performance, and parental MRD. That is, $EstCogRisk_i^{NoGenes}$ replicates objective risk of cognitive decline based on predictors that would be available to individuals, their families, and their physicians. We assess the possible impact of revealing genetic information by constructing the difference between the estimated risk from the model that incorporates genetic risk factors and the one that does not:

$$\Delta Risk_i = EstCogRisk_i^{Genes} - EstCogRisk_i^{NoGenes} \tag{3}$$

Panel (a) of Figure 6 presents a kernel density estimate of $\Delta Risk_i$ given our predictive model. The distribution appears to be trimodal, which results from the large differences in predicted risk of a severe cognitive outcome due to the three APOE genotypes. Mechanically, this distribution has zero mean. Incorporating genetic factors increases estimated risk for some individuals and decreases it for others. Panel (b) of Figure 6 separately plots the distribution of $\Delta Risk_i$ for APOE carriers and non-carriers. Naturally, these distributions are quite different, owing to the large effect of APOE. The dispersion and overlap of these distributions highlight the value of incorporating polygenic risk measures. Even within APOE status, there can be tremendous variation in the change in estimated risk stemming from genetic factors. Indeed, there are many individuals who are APOE carriers but whose total genetic risk is far more modest than what is suggested by unconditional average differences across APOE genotypes. For example, while regression estimates in column (5) of Table 10 suggest that APOE carrier status is associated with an increased risk of a severe cognitive outcome of at least 4.4 percentage points, more than 25% of APOE carriers would see a change in risk of *less* than 2.0 percentage points due to more protective (or less deleterious) polygenic factors. This offers an example of how the communication of total genetic risk may be helpful to contextualize results on APOE. Given previous findings that the communication of genetic risk, particularly APOE carrier status, can cause distress, adding information on other genetic factors may significantly change how a non-trivial number of APOE carriers react to genetic screening results.

While polygenic factors can substantially offset the extra risk associated with APOE for a non-trivial set of people, it is also true that they can add significantly to severe cognitive risk—even for those who do not carry APOE. As revealed in Panel (b) of Figure 6, the majority of APOE non-carriers would see their expected risk of a severe cognitive outcome decline if individual genetic risk factors were used in predictive models. However, over 17% of APOE non-carriers would actually see their estimated risk *increase* with the use of genetic data. This is striking, since so much of the literature on genetic risk for ADRD has focused on the communication of APOE

may reflect observable demographics.

carrier status. A large number of APOE non-carriers could learn that they do not carry APOE but should nevertheless expect higher risk for ADRD based on other genetic factors. Even more concerning, a non-trivial fraction of these non-carriers would actually see their estimated risk rise to levels squarely within the distribution of estimated changes for APOE carriers. Almost 5% of APOE non-carriers would see their estimated severe cognitive outcome risk rise by more than the 10th percentile of the distribution of $\Delta Risk_i$ for APOE carriers (1.1 percentage points).

The results presented here suggest that polygenic risk of ADRD can predict sizable differences in the risk of future serious cognitive events conditional on current cognitive health, demographic factors, and reported family history. Differences in cognitive risk associated with large changes in the AD polygenic score are comparable to changes in risk associated with APOE carrier status, which has received significant attention as a target for personalized risk reports in the ADRD domain. Polygenic risk therefore might have clinical relevance not only as a significant predictor in its own right but also as information that can provide individuals with a broader picture of their genetic risk beyond the APOE genotype. Using polygenic information to enhance predictive models can identify groups who are APOE non-carriers but nevertheless still face elevated genetic risk. Conversely, polygenic information can also identify individuals who are APOE carriers but face only modestly higher genetic risk.

7 Conclusion

We explore how genetic endowments related to ADRD 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. We find higher genetic risk for ADRD predicts worse cognitive function and an increased probability of being diagnosed with a memory-related disease, consistent with prior work that estimates relationships between genetic factors and cognitive health. Higher polygenic risk for ADRD associates with worse economic outcomes, and this relationship holds even in the absence of clinical presentations of ADRD. Those with higher polygenic risk for ADRD are less likely to engage in planning activities that could mitigate the consequences of cognitive decline, and they appear to be unaware of their increased prospects of diminished cognition. In predictive exercises, we show that the genetic measures of ADRD risk provide clinically valuable and policy-relevant information on the development of severe adverse cognitive outcomes in the future.

The collection of results suggests that observable genetic measures of ADRD risk contain actionable information. That is, they are associated with significant harms, reflect risks for which individuals are relatively unprepared, and contain information above and beyond current knowledge or expectations. Put differently, genetic measures of ADRD risk could inform household

financial, legal, and medical care planning, which is especially important given our findings imply there is a large population of people who are under-diagnosed, under-treated, and under-prepared.

Our study raises a host of new questions, and we highlight one that we believe is a clear next step for future research. Are there environmental factors that explain why people with similar genetic risk for ADRD 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.[28] There may be people who are at risk of ADRD and might have exhibited cognitive decline under different circumstances or environments. Future research can shed light on such possibilities.

[28]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 ADRD predict outcomes of interest has not yielded conclusive results.

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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):				
TICS-M Score	16.392	4.004	50–85	88,048
Ever Demented (TICS-M < 7)	0.028	0.165	50–85	88,048
Ever Impaired or Demented (TICS-M < 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
Individual Total Income ($s)	33,010	32,290	50–85	81,393
Household Total Wealth ($s)	588,604	843,313	50–85	84,548
Planning Outcomes:				
Holds Long-Term Care Insurance (LTCI)	0.126	0.332	50–70	52,747
Holds Life Insurance	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: TICS-M 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 (TICS-M 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 TICS-M cognition score, which ranges from 0–27. In Panel B, the outcome is an indicator for a TICS-M score below 12 in the current wave, and the sample excludes individuals after their first observed TICS-M 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 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)				
TICS-M 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 TICS-M 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 5: 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)
TICS-M 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 TICS-M score. 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 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)
TICS-M 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 TICS-M score. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table 7: 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)
TICS-M 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)
TICS-M 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 TICS-M score. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table 8: 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)
TICS-M 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 TICS-M score. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table 9: 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)
TICS-M 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

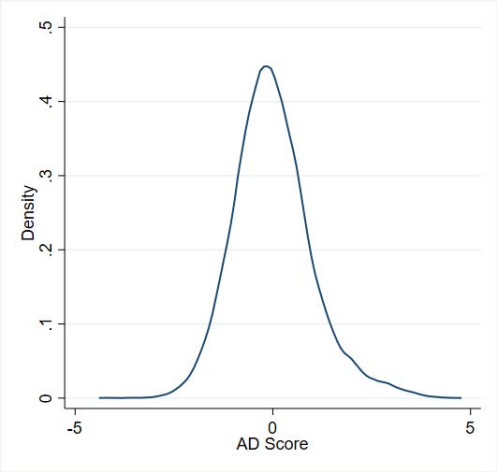
Note: Each column within a panel presents results from a separate regression where the outcome is described in the column heading. In all specifications, we control for the first 10 principal components of the genetic data as well as the standard controls for age, birth cohort, sex of respondent, and 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 TICS-M score. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table 10: Relationship between Genetic Endowments and Future (Age 65–80) Cognitive Outcomes

	(1)	(2)	(3)	(4)	(5)
	Ever Impaired	Ever Demented	Ever Diagnosed	Ever Proxy	Ever Demented, Diagnosed, or Proxy
AD Score	0.023***	0.007**	0.012***	0.007*	0.018***
	(0.007)	(0.003)	(0.004)	(0.004)	(0.005)
APOE (At least 1 copy)	0.061***	0.028***	0.042***	0.014*	0.044***
	(0.013)	(0.007)	(0.008)	(0.008)	(0.010)
APOE (2 copies)	0.120***	0.044	0.093**	0.051*	0.118***
	(0.043)	(0.029)	(0.037)	(0.031)	(0.042)
EA Score	-0.010*	0.001	0.002	-0.002	-0.003
	(0.006)	(0.003)	(0.003)	(0.003)	(0.004)
Mean	0.203	0.028	0.040	0.047	0.086
N	4,860	4,860	4,871	4,871	4,871
R^2	0.211	0.065	0.060	0.050	0.087

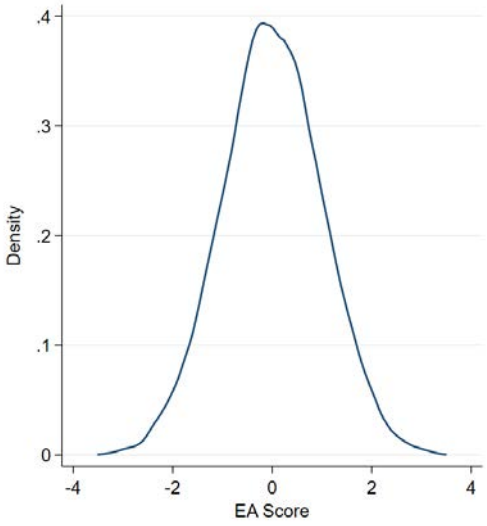
Note: Each column presents results from a separate regression with the outcome listed in the column header. In column (1) the outcome is an indicator for an individual being impaired (TICS-M score < 12) in the age range 65–80. In column (2) the outcome is an indicator for being demented (TICS-M score < 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. In column (4), the outcome is an indicator for ever having an interview wave completed by a proxy respondent. Column (5) presents a specification where the outcome is an indicator for at least one of the following events being observed in ages 65–80: an observation of dementia, a memory-related disease diagnosis, or a proxy interview. 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 TICS-M score observed for ages 50–64, the minimum and maximum TICS-M scores observed over this age range as well as an indicator for whether a parent was diagnosed with memory-related disease and an indicator for missing parental information. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

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



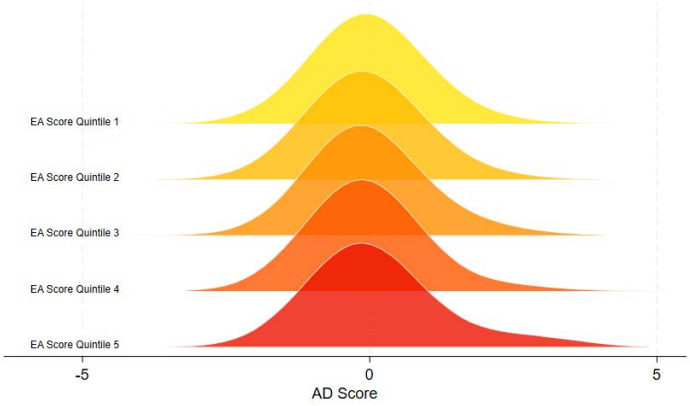
Note: The figure shows the smoothed density of the polygenic score for Alzheimer's disease in our sample.

Figure 2: Distribution of the Educational Attainment Polygenic Score

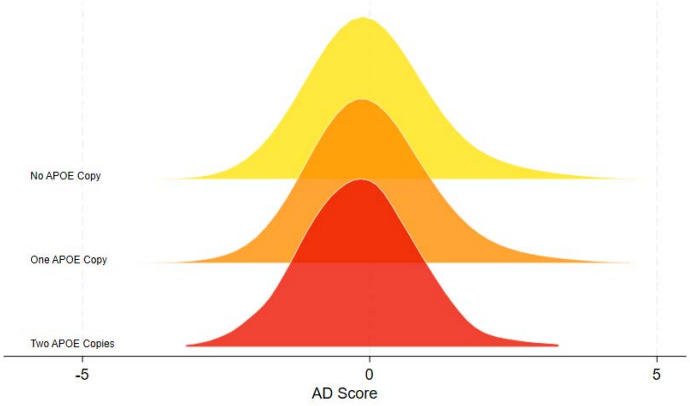


Note: The figure shows the smoothed density of the polygenic score for educational attainment in our sample.

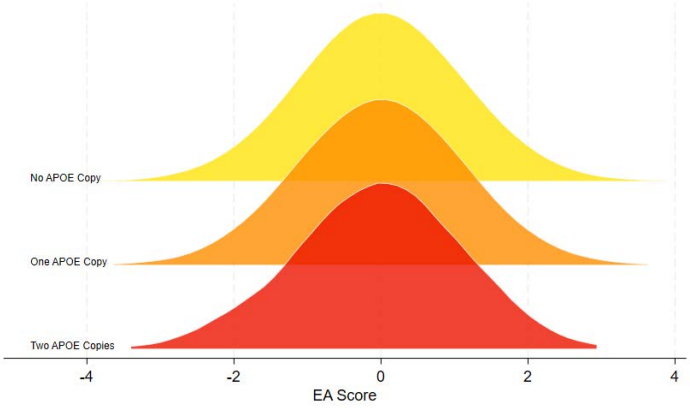
Figure 3: Conditional Distributions of the AD and EA Scores



(a) AD Score by EA Score Quintile



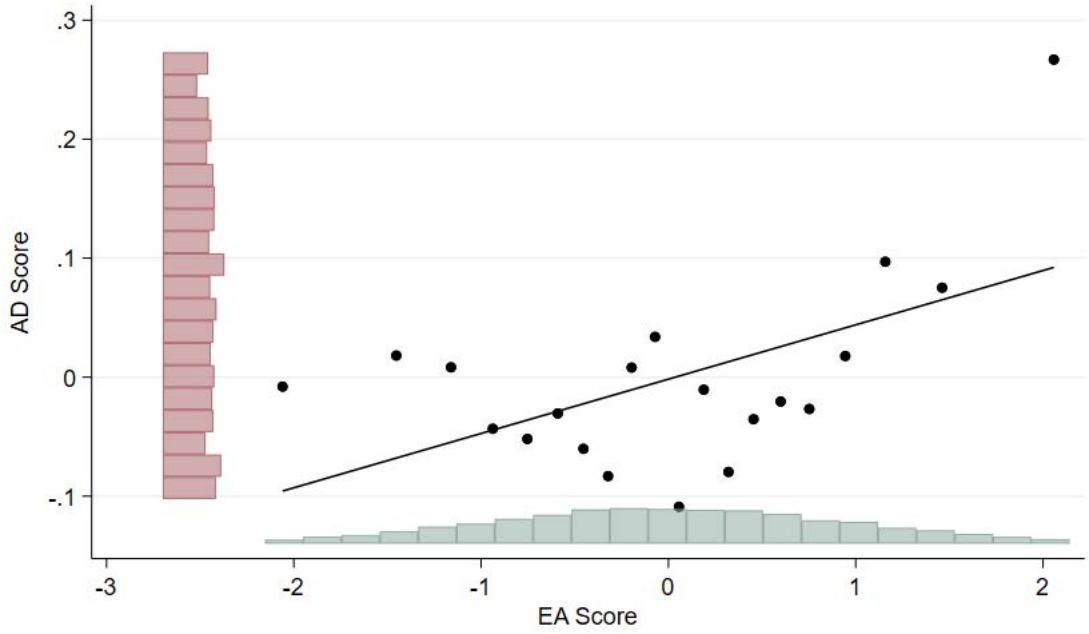
(b) AD Score by APOE Carrier Status



(c) EA Score by APOE Carrier Status

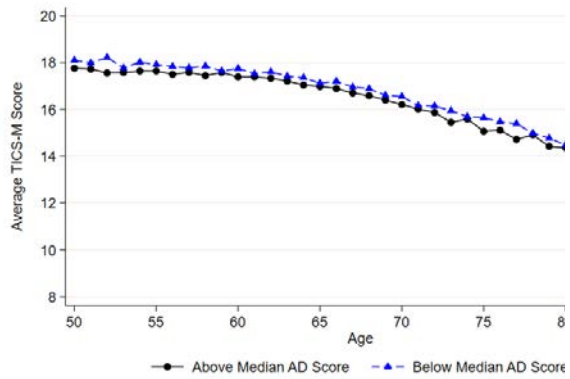
Note: Panel (a) shows the distribution of the AD score conditional EA score quintile. Panel (b) shows the distribution of the AD score conditional on APOE carrier status. Panel (c) shows the distribution of the EA score conditional on APOE carrier status.

Figure 4: Joint Distribution of the Polygenic Scores

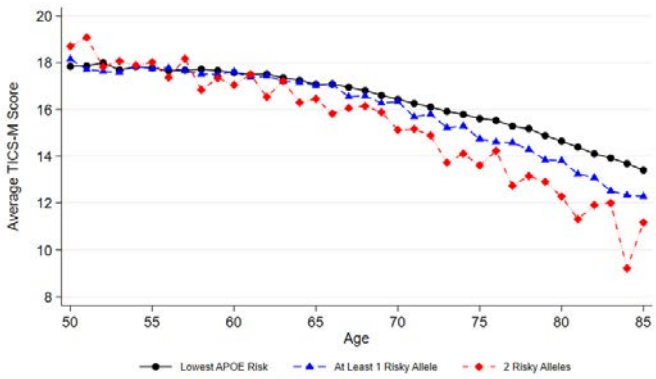


Note: The figure presents a bin-scatter plot for the AD score and EA score, and shows the marginal distributions of each score.

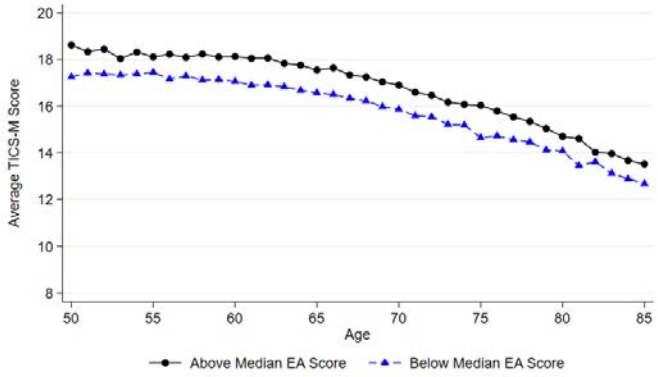
Figure 5: Age-Cognition Profiles by Genetic Risk Groups



(a) TICS-M Score by Above vs. Below Median AD Score



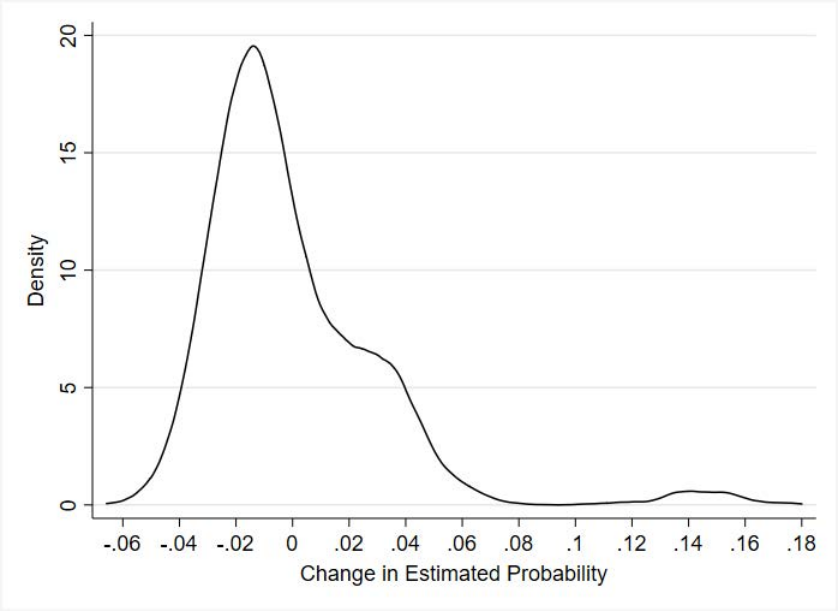
(b) TICS-M Score by APOE Status



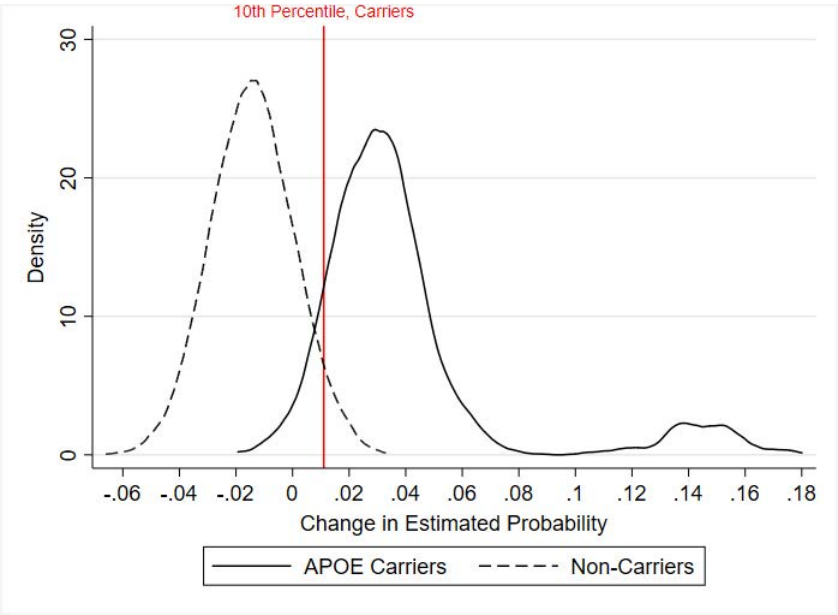
(c) TICS-M Score by Above vs. Below Median EA Score

Note: The figure plots average values of the 27-point TICS-M cognition score by age for individuals in our main genotyped sample.

Figure 6: Density of Change in Estimated Severe Cognitive Outcome Risk Using Genetic Data



(a) Full Sample



(b) Conditional on APOE Genotype

Note: Each panel presents kernel density estimates of the distribution of changes in individual-level predictions of severe cognitive outcome risk (i.e., predicted probability of displaying dementia, being diagnosed with a memory-related disease, or completing an interview by proxy at least once during ages 65–80) that arise from incorporating the AD score, EA score, and APOE measures into a predictive linear probability model. Panel (a) presents the distribution of such changes in the complete sample. Panel (b) presents the distribution of changes separately for individuals who are APOE carriers, and those who are APOE non-carriers.

Appendix

Table A1: Summary Statistics in the Non-Genotyped Sample

	Mean	SD	N
Demographics:			
Birth Year	1940.338	13.008	48,541
Male	0.420	0.494	48,541
Age	66.571	9.671	48,541
Years of Education	11.957	3.674	48,541
At Least Some College Degree	0.235	0.424	48,541
CD and Memory-Related Disease:			
Langa-Weir (L-W) Score	15.100	4.636	48,541
Ever Demented (L-W < 7)	0.072	0.259	48,541
Ever Impaired or Demented (L-W < 12)	0.328	0.469	48,541
Ever Diagnosed with MRD	0.030	0.170	48,541
Economic Outcomes:			
Work for Pay	0.526	0.499	30,699
Retired	0.297	0.457	27,173
Individual Total Income ($s)	25,874	30,513	48,541
Household Total Wealth ($s)	383,290	666,121	48,541
Planning Outcomes:			
Holds Long-Term Care Insurance (LTCI)	0.077	0.267	30,046
Holds Life Insurance	0.577	0.494	30,380
Has a Witnessed Will	0.380	0.486	30,518
Has a Living Will	0.320	0.466	2,028
Has Assigned Someone Durable Power of Attorney for Health Care	0.336	0.472	2,032
Discuss Future Medical Care with Anyone	0.436	0.496	1,554
Awareness Outcomes:			
Probability of Living to Age 75	59.922	30.647	21,570
Probability of Moving to Nursing Home	11.127	19.621	7,474
Probability of Developing Alzheimer's Disease	32.037	26.821	461
Probability of Parent Ever Diagnosed with MRD	0.245	0.430	16,099
Probability of Parent Receiving Nursing Home Care	0.332	0.471	30,581

Note: The table presents summary statistics at the person-wave level from 1998–2018 among HRS respondents who are not genotyped but otherwise meet our sample selection criteria. 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 A2: Relationship between Genetic Endowments and Probability of Being Demented

	Ever Demented (TICS-M 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 a TICS-M 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 A3: 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)				
TICS-M 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 TICS-M 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 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)				
TICS-M 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 TICS-M 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 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)				
TICS-M 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 TICS-M 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 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)				
TICS-M 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 TICS-M 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 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)				
TICS-M 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 TICS-M 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 (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)				
TICS-M 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 TICS-M 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 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)				
TICS-M 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 TICS-M 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 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)				
TICS-M 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 TICS-M 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 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)				
TICS-M 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 TICS-M 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 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)				
TICS-M 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 TICS-M 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 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)				
TICS-M 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 TICS-M 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 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)				
TICS-M 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 TICS-M 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 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)				
TICS-M 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 TICS-M 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 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)				
TICS-M 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 TICS-M 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 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)				
TICS-M 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 TICS-M 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 A18: 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)				
TICS-M 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 TICS-M 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 A19: 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)				
TICS-M 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 TICS-M 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 A20: 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)				
TICS-M 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 TICS-M 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 Sample Attrition

	Appear in the Next Wave as Self-Respondent								
	Full Sample					Never Diagnosed			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
	Panel A: Aged 50–85								
AD Score	-0.004***	-0.004***	-0.003**	-0.002	-0.002	-0.004***	-0.003***	-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)	-0.009***	-0.009***	-0.009***	-0.005**	-0.005**	-0.005**	-0.005**	-0.005**	-0.004*
	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)
APOE (2 copies)	-0.010	-0.009	-0.010	-0.003	-0.002	-0.006	-0.006	-0.006	-0.002
	(0.007)	(0.007)	(0.007)	(0.007)	(0.007)	(0.008)	(0.008)	(0.008)	(0.008)
EA Score		0.008***	0.004***	0.002**	0.002**		0.007***	0.003***	0.002**
		(0.001)	(0.001)	(0.001)	(0.001)		(0.001)	(0.001)	(0.001)
Ever MRD					-0.102***				
					(0.011)				
Eventual MRD					0.004				
					(0.003)				
TICS-M Score Dummies	No	No	No	Yes	Yes	No	No	No	Yes
Education Controls	No	No	Yes	Yes	Yes	No	No	Yes	Yes
Mean	0.933	0.933	0.933	0.933	0.933	0.936	0.936	0.936	0.936
N	82,461	82,461	82,461	82,461	82,461	74,852	74,852	74,852	74,852
R^2	0.051	0.052	0.056	0.076	0.079	0.044	0.045	0.048	0.057
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
	Panel B: Aged 50–70								
AD Score	-0.004***	-0.004***	-0.003**	-0.003**	-0.003**	-0.004***	-0.003**	-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)	-0.003	-0.003	-0.003	-0.003	-0.003	-0.003	-0.002	-0.002	-0.002
	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.003)	(0.003)	(0.002)	(0.002)
APOE (2 copies)	-0.001	-0.000	-0.001	0.000	-0.000	-0.004	-0.003	-0.003	-0.003
	(0.008)	(0.008)	(0.008)	(0.008)	(0.008)	(0.009)	(0.009)	(0.009)	(0.009)
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					-0.039***				
					(0.012)				
Eventual MRD					0.007*				
					(0.004)				
TICS-M Score Dummies	No	No	No	Yes	Yes	No	No	No	Yes
Education Controls	No	No	Yes	Yes	Yes	No	No	Yes	Yes
Mean	0.949	0.949	0.949	0.949	0.949	0.950	0.950	0.950	0.950
N	50,963	50,963	50,963	50,963	50,963	47,912	47,912	47,912	47,912
R^2	0.033	0.034	0.038	0.044	0.044	0.033	0.033	0.037	0.041
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 within a panel presents results from a separate regression where the outcome is an indicator for whether the individual appears in the next survey wave as a self-respondent. 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 TICS-M 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 A22: Relationship between Genetic Endowments and Proxy Interview Next Wave

	Proxy Response in the Next Wave								
	Full Sample					Never Diagnosed			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
	Panel A: Aged 50–85								
AD Score	0.000	-0.000	-0.000	-0.001	-0.001*	-0.000	-0.000	-0.000	-0.001
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.000)	(0.000)	(0.000)	(0.000)
APOE (At least 1 copy)	0.006***	0.006***	0.006***	0.004***	0.002	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 (2 copies)	0.009**	0.009**	0.009**	0.005	0.002	0.003	0.003	0.003	0.002
	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)	(0.003)	(0.003)	(0.003)	(0.003)
EA Score		-0.002***	-0.001*	-0.000	-0.000		-0.001***	-0.001	-0.000
		(0.000)	(0.000)	(0.000)	(0.000)		(0.000)	(0.000)	(0.000)
Ever MRD					0.029***				
					(0.009)				
Eventual MRD					0.034***				
					(0.003)				
TICS-M Score Dummies	No	No	No	Yes	Yes	No	No	No	Yes
Education Controls	No	No	Yes	Yes	Yes	No	No	Yes	Yes
Mean	0.012	0.012	0.012	0.012	0.012	0.007	0.007	0.007	0.007
N	77,857	77,857	77,857	77,857	77,857	70,604	70,604	70,604	70,604
R^2	0.015	0.015	0.020	0.061	0.072	0.008	0.008	0.011	0.015
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
	Panel B: Aged 50–70								
AD Score	0.000	0.000	-0.000	-0.000	-0.000	-0.000	-0.000	-0.000	-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)	0.001	0.001	0.001	0.001	0.000	-0.000	-0.000	0.000	0.000
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
APOE (2 copies)	0.002	0.002	0.002	0.001	0.001	0.003	0.003	0.003	0.002
	(0.004)	(0.004)	(0.003)	(0.003)	(0.003)	(0.004)	(0.004)	(0.004)	(0.004)
EA Score		-0.002***	-0.001***	-0.001**	-0.001**		-0.002***	-0.001**	-0.001**
		(0.000)	(0.001)	(0.001)	(0.000)		(0.000)	(0.000)	(0.000)
Ever MRD					0.010				
					(0.008)				
Eventual MRD					0.008***				
					(0.003)				
TICS-M Score Dummies	No	No	No	Yes	Yes	No	No	No	Yes
Education Controls	No	No	Yes	Yes	Yes	No	No	Yes	Yes
Mean	0.008	0.008	0.008	0.008	0.008	0.007	0.007	0.007	0.007
N	48,775	48,775	48,775	48,775	48,775	45,821	45,821	45,821	45,821
R^2	0.008	0.009	0.014	0.025	0.026	0.009	0.009	0.013	0.017
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 within a panel presents results from a separate regression where the outcome is an indicator for whether the individual has a proxy interview next 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 TICS-M 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 A23: Relationship between Genetic Endowments and Mortality

	Observed Mortality								
	Full Sample					Never Diagnosed			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
	Panel A: Aged 50–85								
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)				
TICS-M 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
	Panel B: Aged 50–70								
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)				
TICS-M 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 TICS-M 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 A24: Relationship between Genetic Endowments and Employment, Income, and Wealth Among the Never Demented

	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**	-0.008	0.007	0.006	-0.014*	-0.005	-0.039**	-0.034**
	(0.005)	(0.005)	(0.004)	(0.005)	(0.007)	(0.008)	(0.016)	(0.017)
APOE (At least 1 copy)	0.007	0.006	-0.007	-0.006	-0.006	-0.000	0.002	-0.012
	(0.009)	(0.009)	(0.008)	(0.008)	(0.013)	(0.016)	(0.029)	(0.032)
APOE (2 copies)	-0.004	0.001	0.030	0.023	0.009	-0.009	0.106	0.017
	(0.026)	(0.030)	(0.024)	(0.026)	(0.039)	(0.047)	(0.093)	(0.114)
EA Score	0.016***	0.017***	-0.014***	-0.016***	0.011*	0.009	0.096***	0.096***
	(0.004)	(0.004)	(0.004)	(0.004)	(0.006)	(0.007)	(0.013)	(0.015)
TICS-M Score Dummies	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Education Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Sample	All	Never Demented	All	Never Demented	All	Never Demented	All	Never Demented
Mean	0.563	0.581	0.320	0.305	10.010	10.057	12.400	12.441
N	53,592	47,147	49,564	43,894	81,393	67,037	84,548	70,053
R^2	0.184	0.179	0.221	0.222	0.274	0.266	0.180	0.171
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 columns (2), (4), (6), and (8), never demented is defined as never having a TICS-M score below 7 and never having a proxy interview while observed in the HRS. 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 TICS-M score. Standard errors are clustered at the individual level. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table A25: Relationship between Genetic Endowments and Later-Life Planning Among the Never Demented

	(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	-0.006	-0.013**	-0.013**
	(0.004)	(0.004)	(0.005)	(0.005)	(0.006)	(0.006)
APOE (At least 1 copy)	0.006	0.004	-0.000	-0.000	0.013	0.015
	(0.007)	(0.007)	(0.009)	(0.010)	(0.010)	(0.011)
APOE (2 copies)	-0.000	0.007	-0.006	-0.016	-0.005	-0.014
	(0.019)	(0.022)	(0.026)	(0.030)	(0.031)	(0.035)
EA Score	0.001	-0.000	0.004	0.002	0.017***	0.015***
	(0.003)	(0.003)	(0.004)	(0.005)	(0.005)	(0.005)
TICS-M Score Dummies	Yes	Yes	Yes	Yes	Yes	Yes
Education Controls	Yes	Yes	Yes	Yes	Yes	Yes
Sample	All	Never Demented	All	Never Demented	All	Never Demented
Mean	0.126	0.127	0.714	0.720	0.559	0.561
N	52,747	46,419	53,322	46,940	53,484	47,059
R^2	0.045	0.044	0.055	0.055	0.134	0.131
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	-0.025**	-0.038***	-0.041***	-0.025**	-0.033***
	(0.012)	(0.013)	(0.012)	(0.012)	(0.011)	(0.011)
APOE (At least 1 copy)	0.033	0.026	0.015	0.006	-0.006	-0.008
	(0.022)	(0.024)	(0.022)	(0.023)	(0.019)	(0.021)
APOE (2 copies)	0.063	0.067	0.057	0.056	-0.026	0.000
	(0.062)	(0.064)	(0.063)	(0.067)	(0.061)	(0.064)
EA Score	0.013	0.014	0.013	0.014	-0.005	-0.001
	(0.010)	(0.011)	(0.010)	(0.011)	(0.009)	(0.010)
TICS-M Score Dummies	Yes	Yes	Yes	Yes	Yes	Yes
Education Controls	Yes	Yes	Yes	Yes	Yes	Yes
Sample	All	Never Demented	All	Never Demented	All	Never Demented
Mean	0.478	0.483	0.462	0.466	0.590	0.598
N	5,066	4,607	5,067	4,609	3,500	3,156
R^2	0.100	0.096	0.097	0.095	0.126	0.123
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 columns (2), (4), and (6) of each panel, never demented is defined as never having a TICS-M score below 7 and never having a proxy interview while observed in the HRS. 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 TICS-M score. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table A26: Relationship between Genetic Endowments and Awareness of Risk Among the Never Demented

	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	0.139	-0.060	-0.075	-0.288	-0.228	0.004*	0.004*	0.002	0.002
	(0.305)	(0.324)	(0.212)	(0.230)	(1.570)	(1.698)	(0.002)	(0.002)	(0.009)	(0.010)
APOE (At least 1 copy)	-0.665	-0.833	0.672*	0.452	7.404***	7.616***	0.023***	0.024***	0.029	0.024
	(0.558)	(0.594)	(0.389)	(0.431)	(2.580)	(2.786)	(0.005)	(0.005)	(0.018)	(0.019)
APOE (2 copies)	-0.587	-0.844	0.034	0.575	-5.681	-5.164	0.001	-0.008	0.118**	0.093*
	(1.889)	(2.100)	(1.347)	(1.649)	(8.021)	(8.112)	(0.014)	(0.014)	(0.051)	(0.055)
EA Score	0.541**	0.605**	0.040	-0.123	3.036**	3.245**	-0.002	-0.001	0.014*	0.013
	(0.258)	(0.273)	(0.179)	(0.198)	(1.207)	(1.279)	(0.002)	(0.002)	(0.008)	(0.009)
TICS-M 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 Demented	All	Never Demented	All	Never Demented	All	Never Demented	All	Never Demented
Mean	66.275	66.534	10.982	11.037	36.303	36.203	0.070	0.070	0.379	0.379
N	36,282	32,790	16,795	14,014	660	602	23,065	20,723	4,344	3,925
R^2	0.085	0.089	0.023	0.023	0.345	0.359	0.031	0.031	0.083	0.086
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 columns (2), (4), (6), (8), and (10), never demented is defined as never having a TICS-M score below 7 and never having a proxy interview while observed in the HRS. 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 TICS-M score. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.