Genetic Risk for Alzheimer's Disease and Related Dementias: Cognition, Economic Behavior, and Long-Run Planning*

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Abstract

Alzheimer's disease and related dementias (ADRD) are widespread and costly. The severe under-diagnosis of ADRD impedes many people from preparing for its health and economic consequences. Existing work has identified several genetic predictors of ADRD, including APOE-ε4 and a polygenic score for ADRD, but genes do not yet figure prominently in ADRD screening. Using data from the Health and Retirement Study, we examine whether these genetic factors can significantly improve the prediction of future ADRD beyond what is possible using standard observables like past cognitive test scores and family history. We then test whether individuals with elevated genetic risk engage in behaviors that could help them plan for or respond to future ADRD. APOE-ε4 carriers face dramatically higher rates of future ADRD, and we find weak evidence that they are aware of their elevated risk and engage in some financial and legal preparations. Individuals with higher polygenic scores are also more likely to develop ADRD, but are significantly less likely to engage in planning activities, such as having long-term care insurance or a durable power of attorney. These results raise the possibility that genetic screening can help individuals at risk of ADRD, but who are unaware and unprepared and thus especially vulnerable to such risk.

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

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1 Introduction

People face a variety of idiosyncratic health risks. A core idea in health economics is that if individuals are aware of these risks, they will take costly actions to prevent them or mitigate their effects. Such actions could take many forms, from pursuing preventive care and lifestyle changes to securing health or disability insurance. Individuals can also take steps to prepare for the financial consequences of negative health shocks and safeguard their families, such as designating a power of attorney, preparing a will, or engaging in precautionary saving. When medical treatment options are limited, these forms of preparation can take on an especially prominent role. Understanding how people make these choices, and the frictions that complicate them, is important for understanding the demand for medical services and financial products (e.g., Finkelstein and McGarry 2006), the selection into program uptake (e.g., Fang, Keane, and Silverman 2008), and the stability of markets (e.g., Rothschild and Stiglitz 1978).

The possibility of developing Alzheimer's disease or related dementias (ADRD) is a particularly salient idiosyncratic health risk.[1] ADRD constitutes a worldwide healthcare crisis. As populations grow older in the United States and around the world, the costs associated with ADRD are likely to put tremendous strain on healthcare systems. Beyond direct medical expenditures, ADRD is linked to numerous other costs, including burdens on family caregivers and consequential financial mistakes. Two features of ADRD amplify the importance of understanding how individuals perceive and react to their risk. First, recent work in the social sciences finds that the economic and financial consequences of ADRD, such as missed payments on credit accounts and declines in wealth, precede an eventual ADRD diagnosis or the onset of dementia by as much as a decade (Nicholas et al. 2021, Li et al. 2023).[2] Second, ADRD is severely underdiagnosed, with some estimates suggesting that 60% of dementia cases remain undetected (Lang et al. 2017). This is particularly concerning given evidence that the financial losses associated with ADRD are larger for those who underestimate their own cognitive decline (Mazzonna and Peracchi 2024).

In this paper, we document the predictive power of genetic measures of ADRD risk and investigate the extent to which individuals with higher genetic risk engage in economic and long-run planning and activities that may mitigate the burdens of ADRD. Individuals vary considerably

[1] ADRD refers to the set of debilitating neurodegenerative conditions that impair memory, thought processes, and functioning. Alzheimer's disease (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, such as Lewy body, frontotemporal, and vascular, 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, hence our focus on ADRD.

[2] The progression of Alzheimer's disease 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 their idiosyncratic risk of developing ADRD, and genes play a large role. Twin studies suggest that genetic factors account for 60–80% of the variation in the risk of Alzheimer's disease (Gatz et al. 2006). Given the complex genetic architecture of ADRD, we study two sets of genetic markers. The first is an individual's status as a carrier of the $\varepsilon 4$ allele of the Apolipoprotein E (APOE) gene. Having one copy of the APOE-$\varepsilon 4$ allele triples the risk of developing Alzheimer's disease (AD), while having two copies increases this risk by a factor of 15 (Sims, Hill, and Williams 2020). 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). We examine the relationships between observed genetic risk for ADRD and trajectories of cognition and memory-related disease, economic outcomes, planning activities, and awareness of risk using the Health and Retirement Study (HRS). These analyses allow us to not only assess how genetic risk for ADRD relates to long-run consequences (e.g., cognitive decline), but also whether individuals are aware of their risk and take concrete steps to prepare. Importantly, working with genetic measures avoids using endogenous outcomes (like an eventual diagnosis) to measure ex-ante risk for ADRD.

We present three main sets of results. First, consistent with earlier work, we find that increased genetic risk for ADRD, whether measured by the AD score or APOE-$\varepsilon 4$ carrier status, predicts lower cognitive function, extremely low cognitive scores consistent with impairment and dementia, and faster rates of cognitive decline. Strikingly, these relationships hold even if we focus on a sample of individuals who are never diagnosed with a memory-related disease, such as AD. We also conduct novel empirical exercises that assess whether the genetic markers contain information that may be quantitatively useful to individuals or their healthcare providers for planning purposes. Among those aged 50–64 who are cognitively healthy, the AD score and APOE-$\varepsilon 4$ carrier status significantly predict the probability of later impairment and dementia after controlling extensively for factors that should reflect their information set at mid-life, including current and past cognitive performance and familial dementia history. About 8.6% of individuals in our sample eventually show signs of serious cognitive difficulty during ages 65–80. Unsurprisingly, carrying the APOE-$\varepsilon 4$ allele massively shifts this probability—by 4.6 percentage points for carriers of one copy and 16.6 percentage points for carriers of two copies. However, our estimates also suggest the AD score contains substantial information; a one standard deviation higher AD score predicts a 1.6 percentage point increase in this risk, even after conditioning on the age 50–64 information set.

Second, we estimate associations between the genetic factors and later-life economic outcomes. We find that higher AD scores are associated with negative economic outcomes. A one standard deviation higher AD score is associated with a 1 percentage point reduction in the probability

of working for pay, and with 3.8% lower household wealth. These relationships survive after controlling for cognitive function and among those who are never diagnosed with a memory-related disease while observed in the HRS. By contrast, we find little to no relationship between APOE-ε4 carrier status and economic outcomes. Thus, it does not appear that those at elevated genetic risk respond by working and earning more or accumulating more wealth (e.g., through precautionary savings). These findings also provide novel evidence that higher polygenic risk for ADRD is economically relevant, and point to a potential distinction between how APOE-ε4 carrier status and polygenic risk operate over the life-cycle.

Third, we find a striking set of associations between the genetic measures and a variety of planning activities. We find weak suggestive evidence that APOE-ε4 carriers are more likely to engage in planning activities, in line with theoretical expectations. However, those with higher AD scores are significantly less likely to engage in 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. For example, a one standard deviation higher AD score is associated with a 1 percentage point lower probability of having long-term care insurance (relative to a 12.6% mean), and a 4 percentage point lower probability of assigning someone as a durable power of attorney for healthcare (mean of 46.2%). These relationships likewise hold after controlling for observed cognitive function and among those who never experience diagnosis during the sample period. These findings may be surprising, as they imply that those who stand to gain the most from precautionary planning are not more likely to engage in such activity, and if anything, are less likely to do so.

The differences in planning behavior related to APOE and the polygenic score could arise for several reasons. First, given the large effects of APOE-ε4 on ADRD risk, carriers of this variant may be more likely to have observed dementia in their family and thus learn about their own idiosyncratic risk. Indeed, we find that APOE-ε4 carriers report higher subjective probabilities of developing ADRD and lower subjective probabilities of being able to live independently and being cognitively healthy 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. Second, APOE-ε4 and the genetic markers captured in the AD score may operate through different mechanisms. While APOE-ε4 appears to be strongly related to deterioration of cognition later in life, high polygenic risk is associated with lower cognition throughout the latter part of the life-cycle. This could explain why those with higher polygenic risk are less likely to engage in cognitively complex actions—like advanced planning—earlier in life. Such an explanation is also consistent with the associations we find between the AD

score and economic outcomes, and the lack of association between APOE and those outcomes.

Our results offer important policy implications. Genetic screening could be valuable for individuals and their healthcare providers as part of an early-warning system. Genetic factors are pre-determined at conception, and can be inexpensively observed earlier in life—well before any clinical evidence of ADRD. Thus, learning about genetic risk may help individuals make decisions about precautionary planning that could mitigate the financial and economic impacts of ADRD. While APOE-ε4 has been discussed extensively as a clinical risk factor, our results demonstrate that *polygenic risk* is also relevant, especially since it is negatively correlated with preparatory actions. Thus, it is important for individuals and healthcare providers to take both APOE and polygenic risk into account when interpreting genetic test results and discussing a patient's genetic predisposition. Given the literature's focus on APOE and its large effects on cognition, individuals who learn that they do not carry APOE might very well revise their beliefs about personal ADRD risk downwards after genetic testing, since they do not have "the gene" for AD. However, our predictive analyses suggest that when genetic factors are incorporated into models of future cognitive illness, about 15% of APOE *non-carriers* should objectively revise such beliefs upwards because their higher polygenic risk more than offsets the reduction in risk from their APOE status.

Genetic measures stand in sharp contrast to the early-warning predictors of ADRD emphasized in the literature, such as financial mistakes or measures of cognitive decline. Such outcome-based measures signal elevated risk only after individuals start exhibiting signs of financial or medical hardship. Put differently, information about genetic risk may allow for precautionary actions, rather than corrective actions after harms have been realized or a diagnosis has been determined. The practical value of genetic risk measures for ADRD is likely to rise as new treatments for ADRD emerge. Although ADRD is currently incurable, a series of new drugs have been approved by the Food and Drug Administration that show promise in treating disease progression. In a world with efficacious treatments, it will be even more important for individuals to be aware of their risk and begin treatment as soon as possible if medically appropriate. Indeed, new research suggests that novel ADRD treatments will be most effective when taken early to prevent (rather than reverse) cognitive decline (Reiman et al. 2024). Our finding that those at high polygenic risk for ADRD seem unaware of their elevated risk highlights an important concern—those that are genetically vulnerable may be the least likely to take advantage of new treatments. Genetic screening could help identify those at high risk and target additional testing and monitoring so that treatment can begin, if medically appropriate, as early as possible in the disease progression.

Our work contributes to several literatures. We add to existing work on associations between cognition, 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).[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). However, Mazzonna and Peracchi (2024) find that those who underestimate their own cognitive decline are particularly vulnerable to losses in household wealth. Thus, it is important to understand what observable measures—like the genetic markers we study—reveal about idiosyncratic ADRD risk, and whether individuals already recognize their risk and take preparatory actions.

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 based on APOE carrier status and 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, Barcellos et al. 2025). 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 risk. Exceptions include Wehby, Domingue, and Wolinsky (2018), Shin, Lillard, and Bhattacharya (2019), and Borgbjerg et al. (2025). Using the HRS, Wehby, Domingue, and Wolinsky (2018) report a negative association between a polygenic score for ADRD and household wealth, while Shin, Lillard, and Bhattacharya (2019) find that higher polygenic risk for ADRD is associated with less savings in active investments (e.g., IRAs) and more saving in passive investments as individuals age. Borgbjerg et al. (2025) estimate associations between children's genetic risk for ADRD and parents' economic outcomes, including employment, earnings, and wealth, using Danish data.

We offer a novel contribution to the literature on genetic predictors of ADRD. Our predictive model of future cognitive illness explicitly controls for observables that are likely in an individual's or healthcare provider's information set, including current and past cognition and family history. This more directly establishes the practical relevance of genetic measures for early-warning or screening purposes compared to most existing studies. However, even if genetic measures like the AD score

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

statistically predict cognitive decline, they will have limited usefulness if they convey information that is unrelated to economic outcomes, or which is already fully understood and accounted for in household preparations. To our knowledge, we offer the first associations between genetic risk for ADRD and a broad suite of financial and legal planning outcomes. The negative associations between the AD score and these activities suggest that there may indeed be scope for public policies or financial products to help at-risk households better understand and respond to their risk.

Our work is also related to the literature on individual beliefs about their 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 ADRD are significantly more likely to explore professional financial planning services and less likely to plan for an early retirement. Some studies implement randomized controlled trials and find that revealing 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 of genetic risk, especially polygenic risk, to influence household outcomes and planning activities. Furthermore, our results highlight a critical distinction between APOE status (studied in the information revelation literature) and polygenic risk for ADRD. Those at higher polygenic risk are systematically less likely to take specific actions to prepare for future ADRD and appear unaware of their increased risk—perhaps because of lower baseline cognitive performance. These findings offer important insights for the design of future randomized experiments that reveal genetic information. For example, if elevated genetic risk for ADRD is correlated with less sophisticated planning activity, it may be important to test interventions that not only reveal genetic test results, but simultaneously convey information about or offer assistance with related planning and preparatory activities.

Finally, we more broadly contribute to a growing body of work that incorporates molecular genetic data into empirical analyses of economic outcomes (Beauchamp et al. 2011, Benjamin et al. 2012). Much of this literature studies whether policies and interventions have heterogeneous treatment effects across individuals with different genotypes. Such heterogeneity constitutes one form of "gene-by-environment" or $G \times E$ interactions (Biroli et al. 2024). Particularly relevant for our context, Barcellos et al. (2025) demonstrate that higher compulsory schooling requirements in the United Kingdom reduced associations between molecular genetic factors and the incidence of ADRD. Other studies investigate how genetic factors influence the human capital development process, including through assortative mating and parental responses (Abdellaoui et al. 2022, Rustichini et al. 2023, Houmark, Ronda, and Rosholm 2024, Sanz-de-Galdeano and Terskaya Forthcoming).

Our work contributes to this literature by demonstrating another important application. Many questions in labor and health economics center on how individuals learn about, signal, or respond to idiosyncratic traits that may be difficult to observe. Genetic endowments—like those we study in the context of ADRD—constitute a potentially important source of heterogeneity that individuals themselves may not fully observe. Our analysis highlights how observed genetic data can be used to test hypotheses about the extent to which individuals learn about and respond to their endowments, and assess the scope for the revelation of genetic information to impact behavior.

The rest of this paper is organized as follows. Section 2 provides background on ADRD diagnosis and genetic variables that predict ADRD. Section 3 describes the data used in our analysis. Section 4 presents our empirical strategy. Section 5 presents our main results. Section 6 concludes.

2 Background: ADRD Diagnosis and Genetic Risk

Here we provide background on two topics essential to contextualize our empirical analyses. First, we discuss how ADRD is diagnosed along with evidence of substantial underdiagnosis. We then discuss genetics and the measures of genetic risk for ADRD we use in our study.

2.1 Diagnostic Context

When diagnosing ADRD, physicians routinely follow clinical criteria like those set out by the joint workgroup of the National Institute on Aging (NIA) and the Alzheimer's Association (AA). The NIA-AA's 2011 guidelines suggest first making an all-cause dementia diagnosis, and then determining whether Alzheimer's disease (AD) is likely to be present. All-cause dementia is marked by a decline in cognitive functioning that interferes with work or daily activities. Such impairment can be assessed by reports from the patient or a family member and from performance on a cognition test or exam. The NIA-AA guidelines suggest that a dementia diagnosis is appropriate when at least two of the following domains are affected: (1) memory and the acquisition of new information, (2) reasoning and judgment in complex tasks, (3) visuospatial abilities, (4) language abilities, and (5) personality and mood (McKhann et al. 2011). Since other conditions besides Alzheimer's disease may result in dementia (e.g., stroke or Lewy body dementia), the guidelines then suggest making a probable AD diagnosis if other diagnoses can be ruled out, and if the development of dementia has been gradual and clearly worsening.

The development of new biomarker tests for AD has opened the door for diagnosis before the presence of significant cognitive symptoms. The International Working Group for New Research Criteria for the Diagnosis of AD has argued for diagnostic criteria that are based exclusively on physical evidence of pathology, including the use of cerebrospinal fluid (CSF) tests for amyloid and

tau protein levels and positron emission tomography (PET) scans to test for amyloid deposition in the brain. These "in-vivo" tests for AD pathology may imply that there is "no longer a reason to wait until patients have developed full-blown dementia or to exclude from diagnosis and treatment a large number of patients who lack functional disability" (Dubois et al. 2010). The most recent revision to the NIA-AA diagnostic criteria accepts the presence of biomarkers as sufficient to establish an AD diagnosis (Jack Jr. et al. 2024). However, outside of research and clinical trials, biomarker tests are not routinely administered to asymptomatic individuals, as these tests are invasive, expensive, or require significant time to perform (Hansson et al. 2022, Schindler and Atri 2023).[4] In the United States, the Food and Drug Administration recently approved a new blood test for AD pathology which could significantly increase the ability to detect AD biomarkers early in the course of the disease (Hampel et al. 2023).

Regardless of the diagnostic standards used, the detection of ADRD begins with primary care physicians (PCPs), who are uniquely positioned to notice gradual changes in cognition and order subsequent testing or referrals to specialists. However, there are significant obstacles to timely diagnosis. Survey evidence from the US suggests that even though nearly all healthcare providers and 80% of older adults believe that brief cognitive testing of those 65 and older is beneficial, less than half are ever tested and less than 20% receive regular brief cognitive assessments (Alzheimer's Association 2019). PCPs cite numerous reasons for this shortfall, including the lack of time during visits, concerns about reimbursement, and the belief that there is no beneficial intervention that could be offered to those with early stages of cognitive decline (Galvin, Tolea, and Chrisphonte 2020, Liss et al. 2021). Federal policy in the US has sought to address this bottleneck. The Affordable Care Act (ACA) of 2010 requires that Medicare reimburse cognitive testing as part of its covered Annual Wellness Visits (AWV). However, uptake of this benefit is not widespread. Although 64% of those age 65 and older recall seeing a provider for a covered AWV, only 32% recall their provider asking about memory or cognition at such visits (Alzheimer's Association 2019).

Genetic factors play an important role in ADRD risk, as detailed next in Section 2.2. However, genes have not figured prominently in diagnostic guidelines for ADRD and certainly not as an early-warning tool. For example, the NIA-AA's guidelines only specifically mention genetic factors for early-onset Alzheimer's disease (distinct from the more common late-onset AD that we study) (McKhann et al. 2011, Jack Jr. et al. 2024). The NIA-AA working group took a cautious approach and included "very little about genetic aspects of AD" in their 2011 guidelines (Reiman et al. 2011). Their most recent guidelines mention the possibility of using genetic factors together with

[4]For example, the average cost of an amyloid PET scan is $4,000–$7,000, with very low rates of insurance reimbursement (Schindler and Atri 2023).

biomarker and observational data to create a "comprehensive system to stratify risk of onset and progression," but note that such a system "is aspirational at this point" (Jack Jr. et al. 2024).

With this diagnostic context in mind, ADRD appears to be severely underdiagnosed. The meta-analysis in Lang et al. (2017) suggests that about 60% of dementia cases in the US have not been diagnosed. Long gaps between signs of cognitive decline and an eventual diagnosis with a memory problem are evident in longitudinal data sets like the Health and Retirement Study. Using data on all 3,911 HRS respondents who report ever being diagnosed with a memory-related illness, we calculate the difference between the age when an individual first reports being diagnosed with such an illness and the age at which they first receive a score on an HRS-administered cognitive test consistent with cognitive impairment. Figure 1 plots a histogram of this measure and reveals evidence of considerable diagnostic delay. The average gap is approximately four years, with 25% of the eventually diagnosed waiting six or more years after the first evidence of cognitive impairment.

2.2 Genetic Risk for ADRD

We measure ADRD risk using two separate *molecular* genetic variables. Human DNA consists of a sequence of roughly 3 billion nucleotide base-pair molecules spread out across 23 chromosomes.[5] At each location in the genome, individuals possess one of two possible base pairs: an adenine-thymine (AT) pair or a guanine-cytosine (GC) pair. At nearly all of these locations, every human being has exactly the same base-pair molecules. However, at a small number of sites (less than 1%), 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.[6] An individual carries the APOE-ε4 variant if they have a specific combination of base-pair molecules at two SNPs. Carrying the APOE genetic variant is the strongest single predictor of AD. Having one copy triples AD risk, while two copies leads to a 15-fold increase in risk (Sims, Hill, and Williams 2020). 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. 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).

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

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

While APOE has received much attention, it is not the only source of genetic risk, and 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. There are many other genetic markers 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 genetic risk for the trait or outcome.[7]

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 follow the guidance of Ware et al. (2020) and use the AD score that excludes the APOE region and treat APOE as a separate measure of genetic risk.[8] We provide more details on the AD score and our measures of APOE in the next section.

The use of genetic data comes with several caveats, many of which have been discussed elsewhere. We provide a brief summary. First, because of estimation error in the GWAS coefficients, a polygenic score tends to be a noisy measure of any underlying genetic propensity. Finite-sample GWAS have particularly limited statistical power to detect the influence of rare variants or variants with small association sizes. The AD score will thus only capture a fraction of the variance in ADRD attributable to genes. 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).[9] Consequently, there may be measurement error in the

[7] 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).

[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.

[9] Until recently, the only way to confirm whether an individual indeed had AD was after death via autopsy.

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 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.[10]

Second, the construction of polygenic scores is largely based on GWAS estimates from 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). We are therefore limited to studying individuals of European descent.[11]

Finally, polygenic scores are unlikely to be exogenous with respect to family environments. Genetic factors that influence the outcomes of one generation tend to predict outcomes of the next generation not only because of direct biological effects, but also indirect effects operating through parental outcomes. It is therefore difficult to claim that estimated relationships involving polygenic scores capture causal effects. More demanding econometric specifications can yield causal effects by exploiting the fact that the genotypes of children are random conditional on the genotypes of their parents. In settings where genetic data are available for multiple family members, one can estimate causal effects using family fixed effects models, or by controlling for parental genes.

We cannot implement family-based designs to estimate causal effects because the HRS does not feature sibling or parental genetic data. Nevertheless, we argue that the associations that we estimate are valuable. In applications that use molecular genetic data, it is often critical to isolate exogenous variation in genes to answer questions about the mechanisms through which genes operate, or to evaluate whether certain policies moderate inequality arising from genetic factors. For our research questions, having strictly exogenous variation in genetic factors is far less important. Our results establish that polygenic scores contain information about future cognitive difficulties,

As detailed in Section 2.1, clinical diagnosis can now be accompanied by biomarker testing, such as PET scans, spinal taps to measure CSF, and blood tests.

[10] While our results suggest that information about genetic risk for ADRD is useful for financial, legal, and medical planning purposes, we caution that using the AD score alone to target medical treatments that address the underlying brain changes of AD is unwise given the likely measurement error in AD cases in the GWAS. Nevertheless, genetic risk measures could be used to target additional screening and monitoring, so that a patient can begin treatment as soon as possible if medically appropriate.

[11] Applying a score constructed from one ethnic group to another can vastly over- or under-estimate the predicted likelihood of an outcome due to statistical artifacts, such as differences across groups in how SNPs correlate with 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 educational attainment polygenic score.

and that many high-risk people are unaware of their elevated risk. Even if the associations we report reflect some degree of environmental confounding, the policy implications still stand—polygenic scores contain valuable information for planning purposes and have potential clinical use.

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 (Health and Retirement Study 2024). 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 use data from 1998–2018, as key measures regarding cognitive function and diagnosis of memory-related disease did not become available until the 1998 survey wave.

The HRS collected genetic samples from nearly 20,000 respondents over the course of four waves (2006, 2008, 2010, 2012). Our sample only includes these genotyped individuals.[12] 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 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 and APOE

As described earlier, we consider two 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 using all genotyped HRS respondents of European ancestry. Second, we consider whether an individual carries the APOE allele and create two indicators. The first takes value one if the individual carries at least one copy of APOE-$\varepsilon 4$, and the second takes value one if the individual has exactly two copies.[13]

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

[13] 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 regarding which imputed values to include in the analysis (Faul et al. 2021).

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 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 difficult to implement (Fong et al. 2009). Specific ranges of the 27-point TICS-M score 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 (Langa et al. 2020).[14] We study the TICS-M score and indicators for whether an individual ever achieved a score that corresponds with the impaired or demented categories, where "ever" means they registered such a score in the current survey wave or any prior wave.

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.[15] We

[14] 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 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.

[15] 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

winsorize both income and wealth at the 1st and 99th percentiles.

3.1.4 Planning Outcomes

We consider several measures related to later-life planning activities. We create an indicator for holding long-term care insurance (LTCI). We also create indicators for whether the respondent holds life insurance, has a witnessed will, has a living will (i.e., an advance healthcare directive), has assigned someone durable power of attorney for health care, 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 subjective expectations about mortality, long-term care utilization, ability to live independently, cognitive health, and developing ADRD. The HRS asks respondents younger than 65 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. In 2006 and 2008, respondents younger than 65 who did not report needing help with activities of daily living (e.g., walking, bathing, eating, transferring, toileting) or instrumental activities of daily living (e.g., meal preparation, shopping, medication management, using the phone) were asked about the chances their health would allow them to live independently (i.e., at home without help) at age 75 as well as the chances they will be free of serious problems in thinking, reasoning, or remembering things that would interfere with the ability to manage their own affairs at age 75. In addition, 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 ADRD in the future.[16]

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).

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

Given the experimental modules are fielded to a small subsample and we only include responses among genotyped individuals, sample size is substantially smaller for this outcome.

Individuals may also learn about their risk for ADRD via their parents. We examine how own genetic risk for ADRD correlates with whether the respondent's mother or father was ever 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 alive. Parental nursing home care is determined via questions about where and with whom a parent currently resides (if alive) and whether a parent received nursing home care prior to death (if the 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 genotyped individuals aged 50–85 between 1998–2018 who are self-respondents. For most analyses, we exclude person-year observations involving proxy responses since they are missing most of our outcomes of interest, including direct measurements of cognition.[17] Analysis sample sizes fluctuate across regressions, and the age range of our analysis samples vary depending on the outcome. For employment and retirement outcomes, we limit the sample to those 50–70 years old since most people retire by age 70. For the analyses of planning activities and awareness of risk, we also limit the sample to ages 50–70, prior to the typical age of onset of ADRD, though results are robust to using a sample aged 50–85. Moreover, for some planning and awareness outcomes, the questions were only asked to certain age groups within the 50–70 age range or during specific survey waves. 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. Sample sizes vary because the statistics are calculated using the maximum number of person-year observations that meet our sample inclusion criteria for that variable or outcome. Among those aged 50–85, about 42% are 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.

Polygenic scores were standardized using all genotyped HRS respondents to have mean zero and standard deviation equal to one. The mean AD score in our sample is slightly below zero,

[17] Proxy interviews arise when the respondent cannot complete an interview due to physical or cognitive limitations. In our sample, conditional on being a self-respondent, only 1% go on to have a proxy interview in the next wave.

reflecting that those at higher risk of ADRD exit the sample (through survey non-response, proxy interview, and/or death). We address concerns about attrition in Section 5.6.1. Roughly 26% of the sample has at least one copy of APOE and 2% have two copies. The distribution of the AD polygenic score is presented in Figure 2. The score is symmetric around the mean and approximately normally distributed. In Figure 3, we show the AD score distribution conditional on APOE carrier status. Those who do not carry APOE and those who carry one copy have similar distributions. For both groups, 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 lower polygenic risk for ADRD than the average person who carries zero or one copy.

Turning to outcomes related to cognition 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 4 shows how the average TICS-M score evolves over the later life-cycle for groups with different genetic risk in our sample. For all groups, there is a smooth downward trajectory in TICS-M scores, with the average score falling by approximately 0.14 points per year when pooling all observations. Panel (a) plots the unconditional average TICS-M score by age separately for those with above and below median values of the AD score. Modest differences in the average TICS-M score are observed at every age, with little change in this gap over the life-cycle. This contrasts with the results in Panel (b), which plots age profiles based on APOE carrier status. Each carrier group has a nearly identical age-cognition trajectory until the mid 60s, when individuals possessing the APOE allele exhibit increasingly lower TICS-M score averages compared to non-carriers. These differences raise the possibility that while both the AD score and APOE represent genetic endowments linked to cognitive performance, they may operate through different mechanisms that influence behavior and health at different times in the life-cycle.

We also examine economic outcomes. According to Table 1, 56% of those aged 50–70 work for pay while 32% are retired. The remaining observations are people who do not work for pay but do not describe themselves as being completely retired. Among those aged 50–85, average total individual income is about $33,000 and average household wealth is almost $590,000.

In terms of later-life planning outcomes, 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. On average, individuals engage in 2.7 of the six planning activities we consider.

Our analysis also assesses to what degree individuals are aware of their risk of cognitive decline. On average, individuals aged 50–65 believe they have a 66% chance of living to 75. On average, individuals aged 65–70 report an 11% probability of moving to a nursing home in the next five years. Individuals aged 50–65, on average, report a 70% probability of being able to live independently at age 75 and a 66% probability of being cognitively healthy at age 75. Those aged 50–70 report a 36% chance of developing ADRD in the future. Last, about 7% of individuals aged 50–70 have a parent who has ever been diagnosed with MRD, and by their last observation between ages 50–70, almost 38% 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. The genotyped sample is positively selected on education, cognition, economic outcomes, and later-life planning, which could attenuate the associations between the genetic measures of ADRD risk and outcomes we study.

4 Empirical Strategy

Most of our results take the form of OLS coefficient estimates from specifications like the following:

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

where Y_{it} denotes the outcome of individual i in survey wave t. $ADScore_i$ is the polygenic score for AD (that excludes the APOE region). We include an indicator for having at least one copy of the APOE-ε4 allele as well as a separate indicator for having two copies. In this way, we allow for non-linearities in the relationship between the number of APOE copies and the outcomes of interest. 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.[18]

In some specifications, we include additional individual-level controls. In particular, we add

[18]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.

measures of socioeconomic status (SES) during childhood—an indicator for whether the respondent reports that their family was well off financially or about average from birth to age 16 (versus being poor) as well as dummy variables for their mother's years of education. In cases where these variables are missing, we assign a value of zero and include indicators for missing values. Where indicated, we also control for own completed education via a full set of degree dummies and indicators for different numbers of years of education, fully interacted with gender.[19] The childhood SES and educational attainment variables are meant to control for the early-life environment. In some specifications, we add dummy variables for each value of the current TICS-M score to learn whether the genetic measures of ADRD risk have predictive power even after flexibly accounting for cognitive function.

5 Main Results

Our main results are estimates of the relationship between genetic risk for ADRD and a series of outcomes: cognitive function and memory related-disease diagnosis, labor supply, household economic resources, planning activities, and awareness of ADRD risk.

5.1 Replication: Cognition and Memory-Related Disease Diagnosis

We first demonstrate that known associations between the genetic measures and direct ADRD outcomes (e.g., cognitive impairment) replicate in our HRS sample. Panel A of Table 2 presents estimates from a regression of the TICS-M score on different sets of controls, all of which include the "standard controls" outlined in the previous section. Column (1) presents estimates for a specification that includes our three genetic variables of interest: 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 lower TICS-M score, compared to a mean of 16.4. This is equivalent to the difference associated with 1.7 extra years of cognitive aging based on the relationships in Figure 4. Having at least one copy of APOE is associated with a 0.40 lower TICS-M score and having two copies is associated with a further decrease of 0.56. Carrying one or two copies of APOE thus predicts differences in cognition equivalent to 2.9 and 6.9 years of average cognitive aging, respectively. In column (2), we include controls for childhood SES, and we additionally include controls for completed education in column (3). The addition of these controls decreases the magnitude of the coefficient on the AD score somewhat and modestly attenuates the coefficients on the APOE indicators, but all the genetic predictors remain highly significant.

[19] In a causal inference framework, controlling for some of these variables could be interpreted as conditioning on "bad controls." We do not aim to make causal statements, but instead show that the genetic measures predict behaviors and outcomes independently of these variables.

To explore whether the ADRD genetic measures predict especially poor cognitive function, we consider as an outcome 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 after their first TICS-M score less than 12, as we treat impairment and dementia as absorbing states. Results are presented in columns (1)–(3) in Panel B of Table 2. Across the specifications, estimates are significant and generally stable. In the main sample including the full set of controls (column 3), 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 5.4%. Moreover, having at least one copy of APOE corresponds to a 1.1 percentage point increase in this probability, and those with two copies see an additional 2.3 percentage point increase.[20] We also estimated the specifications in Table 2 separately for those aged 50–65 and 66–85. The results (available by request) confirm the patterns observed in Figure 4. A higher AD score predicts statistically significant lower cognitive function across both age groups. Carrying APOE robustly predicts worse cognitive health among those aged 66–85, but the coefficients on the APOE indicators are much smaller and not statistically significant for the sample aged 50–65.

Our results thus far demonstrate that genetic risk for ADRD associates with lower cognitive health in levels. We next investigate *changes* in cognitive performance. In Table 3, we report results where the outcomes are the change in the TICS-M scores between survey wave t and $t-1$, t and $t-2$, and t and $t-3$, and we incrementally add covariates as in Table 2. We only include those observed continuously from $t-3$ to t to ensure that compositional changes do not influence the results. Generally, higher AD scores and carrying an APOE allele are associated with significant declines in cognitive performance. For example, including all controls (column 3 in Panel C), a one standard deviation higher AD score predicts a 0.04 decline in the TICS-M score from wave $t-3$ to t, where the average change is -0.91. Carrying one copy of APOE predicts a 0.30 decline in the TICS-M score, and holding two copies predicts a further decline of 0.63.

We next examine whether genetic endowments 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 MRD diagnosis. Results where we progressively add covariates are presented in Table 4. Across the specifications, we find economically meaningful and statistically significant associations between the genetic measures and diagnosis. A one standard deviation rise in the AD score is associated with a 0.1 percentage point increase in the probability of MRD

[20]We repeat this analysis with an indicator for ever being demented (i.e., TICS-M score less than 7) as the outcome. Results are shown in Appendix Table A2. Carrying the APOE allele increases the probability of dementia non-trivially, and a one standard deviation increase in the AD score is associated with a 0.1 percentage point increase in this probability.

diagnosis, from a mean of 0.7%. Carrying the APOE allele increases this probability by 0.4–0.5 percentage points, and carrying two copies further increases this probability by 1.0 percentage point. Thus, genes that predict cognitive function and decline also predict diagnosis.

Finally, we investigate whether the genetic risks revealed in Tables 2 and 3 are fully reflected in eventual MRD diagnosis. In columns (4)–(6) of both of these tables, we repeat our analyses but focus on the subsample of individuals who are never diagnosed with MRD while observed in the HRS.[21] For example, in columns (4)–(6) in Panel B of Table 2, we examine whether the genetic variables predict cognitive impairment or dementia among those who are never diagnosed with MRD. Among this group in a specification with full controls (column 6), a one standard deviation increase in the AD score is associated with a 0.4 percentage point increase in the risk of impairment or dementia (mean of 4.7%). Having at least one copy of APOE is associated with an increase in this risk of 0.6 percentage points, and having two copies with an additional increase of 1.8 percentage points. In columns (4)–(6) of Table 3, the associations between the genetic measures and changes in cognition are similar to (if modestly smaller than) those found in the main sample. These results highlight how genetic predictors may contain valuable information about cognitive health—even significant impairment and dementia—that may not be efficiently detected and diagnosed in existing healthcare institutions.

5.2 Prediction Exercise

The results in Tables 2–4 suggest that observable genetic factors can predict cognitive difficulties that are not reliably diagnosed in this sample. These results, however, do not necessarily establish the genetic measures as useful clinical tools for predicting individual risk or targeting interventions (e.g., additional screening or monitoring). For these measures to be valuable for such tasks, they need to predict risk for future ADRD above and beyond the data and medical history that a physician would have at their disposal. We therefore examine 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 healthy in the early period. We therefore restrict our sample to 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 then estimate regressions that predict outcomes related to dementia in the later age range of 65–80. These regressions are different from our model in Equation 2 because they

[21] 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.

are 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, birth year dummies, dummies for years of schooling, dummies for highest degree, and a male dummy, along with its interactions with all of the previous regressors. Importantly, we flexibly control for the individual's cognitive performance during the early period by including the average TICS-M score, the minimum TICS-M score, and the maximum TICS-M score. As healthcare providers usually consider family history of ADRD when assessing risk, we include an indicator for whether a parent was diagnosed with MRD during the early period.[22] While it is unclear the extent to which providers consider the early-life environment when evaluating risk, we additionally control for the childhood SES variables. Given the serious barriers to detecting ADRD outlined in Section 2.1, this specification almost certainly overstates the information currently used by providers and families, and thus understates the potential usefulness of genetic predictors.

We examine 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 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 a comprehensive assessment of acute cognitive difficulties. 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 65–80 are observed with a cognitive test score consistent with dementia during the late period.

Table 5 reports the estimated coefficients on the genetic predictors from the specification described above. We find strikingly large coefficients on the APOE variables. 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 eventually exhibit at least one of these more severe signs of decline. Carrying one copy of the risky APOE allele is associated with a 4.6 percentage point higher probability of this event, while carrying two alleles is associated with an *additional* increase of 12 percentage points.

[22] Because questions about parental MRD were not asked until 1998 and only asked if a parent is alive, parental diagnosis is missing for about half the sample (e.g., respondents without living parents as of the 1998 wave or their initial survey if after 1998). We set parental diagnosis to zero for those with missing information and separately control for an indicator for missing parental information.

The large differences in future cognitive health by APOE status justify the attention that APOE receives as a focal point for genetic testing and personalized medicine. However, 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 0.6 percentage point higher chance of demented cognitive performance (2.8% sample prevalence) and a 1.6 percentage point increase in the probability of a severe adverse cognitive outcome (8.6% sample prevalence). The associations between the outcomes in Table 5 and a one standard deviation higher AD score are roughly 0.20 to 0.40 times the size of the association between these outcomes and carrying one risky APOE allele.[23]

To assess the possible clinical relevance of the genetic predictors of ADRD, we compare estimates of the risk for a severe cognitive outcome (at ages 65–80) across two predictive models. First, we consider individual-level estimated risk from the model in column (5) of Table 5, 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 the AD score and APOE dummies. We refer to the fitted values from this specification as $EstCogRisk_i^{NoGenes}$.[24] We assume that $EstCogRisk_i^{NoGenes}$ measures the objective clinical risk that one should expect in the absence of genetic data, but given knowledge of demographics (including education and early-life SES), 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 5 presents a kernel density estimate of $\Delta Risk_i$. The distribution appears to be trimodal, which results from the large differences in predicted risk due to the three APOE genotypes. Mechanically, this distribution has zero mean. Incorporating genetic factors increases

[23]Livingston et al. (2024) identifies 14 modifiable risk factors for dementia, including less education, hearing loss, high LDL cholesterol, depression, traumatic brain injury, physical inactivity, diabetes, smoking, hypertension, obesity, excessive alcohol, social isolation, air pollution, and vision loss. We reestimate the prediction exercise above adding in "early period" controls for a subset of these risk factors that can be straightforwardly obtained from the HRS (i.e., ever smoked, ever have hypertension, ever have diabetes, ever have a heart condition, ever wear a hearing aid, ever register a Center for Epidemiologic Studies Depression (CES-D) score consistent with depression, ever obese) and find the point estimates on the genetic measures barely change (Appendix Table A3).

[24]We retain the principal components, as they capture population stratification which may reflect observable demographics.

estimated risk for some and decreases it for others. Panel (b) of Figure 5 separately plots the distribution of $\Delta Risk_i$ for APOE carriers and non-carriers. 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 APOE carriers 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 5 suggest that APOE carrier status is associated with an increased risk of a severe cognitive outcome of at least 4.6 percentage points, more than 24% 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.

While polygenic factors can substantially offset the risk associated with APOE for some, it can also add significantly to risk—even for those who do not carry APOE. As revealed in Panel (b) of Figure 5, 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 15% of APOE non-carriers would actually see their estimated risk *increase*. This is striking, since the literature on the clinical or planning value of genetic markers for ADRD has largely centered on the disclosure of APOE carrier status. A large number of individuals 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. For example, almost 4% of APOE non-carriers would see their estimated risk of severe cognitive difficulties rise by more than the 10th percentile of the distribution of $\Delta Risk_i$ for APOE carriers (1.1 percentage points).

The results above imply that polygenic indices can identify meaningful differences in predicted dementia risk conditional on current cognitive health, demographic factors, and reported family history, and can productively supplement or contextualize counseling on APOE genotype status. Using polygenic information to enhance predictive models can identify those who are APOE non-carriers but nevertheless face elevated genetic risk. Conversely, polygenic information can also identify individuals who are APOE carriers but face only modestly higher genetic risk. Providing information on total ADRD genetic risk (i.e., genetic risk beyond the APOE genotype) 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 suggest 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).

5.3 Economic Outcomes

The previous section provides evidence that genetic measures related to ADRD are predictive of future cognitive function and diagnosis. In this section and the next, we ask whether these genetic factors predict behaviors and outcomes indicative of preparation for or response to these cognitive risks. We first turn to economic outcomes, including working for pay, retirement, log individual total income, and log household total wealth. In anticipation of future medical and long-term care costs, individuals at a higher risk of ADRD may work more, or engage in precautionary saving (leading to higher current wealth). We provide a summary of the results in Table 6. In all specifications, we include the standard controls, childhood SES controls, own educational attainment controls, as well as dummy variables for the contemporaneous TICS-M score. In Appendix Tables A4–A7, we present results where we incrementally add these control variables. For each outcome, we present estimates from the full sample and for those never diagnosed with MRD while observed in the HRS.

We find that a one standard deviation rise in the AD score is associated with a statistically significant 1.0 percentage point *decrease* in the likelihood of working for pay among those aged 50–70, from a baseline of about 57%, and a 0.9 percentage point decline among the undiagnosed.[25] An increase in the AD score is also associated with an increased probability of retirement and lower individual income, though the associations are not significant at conventional levels once controls for educational attainment and current cognition are included. A higher AD score predicts statistically significant lower household wealth for the full sample (3.8% lower) and the never-diagnosed sample (3% lower). There is no notable pattern in the coefficient estimates on the APOE indicators and they are not precisely estimated.

We thus find no evidence that individuals respond to elevated genetic risk by working and earning more or accumulating more wealth. If anything, higher polygenic risk is associated with less attachment to the labor market and lower wealth. This could reflect that higher genetic risk for ADRD is associated with greater economic vulnerability—even when clinical presentations of ADRD are absent. The qualitative difference in results between the AD score and APOE carrier status is also noteworthy, and mirrors the different life-cycle associations between these genetic factors and cognition from Figure 4. While APOE carriers show little if any difference in economic or cognitive outcomes before age 70, those at higher polygenic risk show significant differences in both cognition and economic outcomes earlier in life.

[25] We repeat the analysis of 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 aged 70–75.

5.4 Planning Activities

We next ask whether individuals at greater genetic risk for ADRD are 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 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 typical age of onset of cognitive decline and diagnosis.[26] 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 but a reaction to illness. Results from specifications that include the standard controls, childhood SES controls, own educational attainment controls, as well as dummy variables for the contemporaneous TICS-M score are presented in Table 7.[27]

Higher AD scores are associated with significantly lower probabilities of engaging in planning activities. A one standard deviation increase in the AD score is associated with a 0.8–0.9 percentage point decline in the probability of having LTCI (from a mean of 13%), about a 1 percentage point decline in having a witnessed will (from a mean of 56%), a 3.7–3.9 percentage point decline in having a durable power of attorney (from a mean of 46%), and a 2.3 percentage point decline in the probability of discussing future medical care with anyone (from a mean of 59%). These associations are remarkably similar across the full and never-diagnosed samples. We generally find no significant association between being an APOE carrier and the planning activities, though the majority of the estimated coefficients on the indicator for carrying one copy are positive.

In Table 8, we restrict the sample to 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. For the full sample, 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 0.08 fewer total activities. The point estimates are similar for the undiagnosed but often not precisely estimated. There is weak suggestive evidence of increased likelihood of engaging in planning activities among APOE carriers as the vast majority of coefficient estimates on the APOE variables are positive though they never reach statistical significance at conventional levels.

One possible explanation for the decreased engagement in planning activities among those

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

[27] Results for each planning activity with controls incrementally added are presented in Appendix Tables A8–A13.

with higher AD scores that is consistent with our results so far is that these individuals have fewer financial resources to protect. For example, for individuals without wealth, there is less incentive to have a witnessed will or LTCI. To shed light on this idea, we re-estimated 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 lacking financial resources to shield.

The planning results are striking in that those who stand to gain from precautionary planning, namely those with increased risk of experiencing cognitive decline and developing ADRD, are no more likely to engage in these planning activities. If anything, those with higher polygenic risk for ADRD are *less* likely to plan. It is possible that APOE carriers are relatively more aware of their risk for ADRD compared to those with higher AD scores, which may explain the difference in planning by genetic risk type. We explore this idea next.

5.5 Expectations and Awareness

Individuals with higher AD scores face the prospects of diminished cognition and more challenging economic circumstances, but they do not seem to engage in medical or financial planning activities, and if anything, are less likely to do so. At the same time, APOE carriers also face a substantially higher risk of diminished cognition, and if anything, we find weak suggestive evidence that they are more likely to plan. These patterns could be explained by differences in the extent to which individuals understand their elevated risk. To explore this idea, we examine how genetic risk for ADRD correlates with subjective expectations about mortality, nursing home use, ability to live independently, cognitive health, and development of ADRD. 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 from specifications that include the standard controls, childhood SES controls, own educational attainment controls, as well as dummy variables for the contemporaneous TICS-M score are presented in Table 9.[28]

In columns (1) and (2), we show results where the outcome is the self-reported probability of living to age 75, which is asked to those younger than 65. The point estimate on the AD score is positive, but standard errors are large. APOE carriers report lower probabilities, but none of the coefficients reach statistical significance. 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

[28] Results for each outcome with controls incrementally added are presented in Appendix Tables A14–A20.

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 statistically significant. Three of the four APOE coefficients are positive, though none are precisely estimated. 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 cognitive function, potentially leading these estimates to be conservative. In columns (5) and (6), we focus on the self-reported probability of living independently at age 75, and in columns (7) and (8), we consider the self-reported probability of being cognitively healthy at age 75 (i.e., not having problems in thinking, reasoning, or remembering that interfere with the ability to manage one's own affairs), which are asked to respondents younger than 65. The coefficients on the AD score are positive but not statistically significant. For both outcomes, those who carry one copy of APOE report a statistically significant 2 percentage point lower probability. The sum of the APOE coefficients implies that those who carry two copies of APOE also report lower probabilities, but the association is not precisely estimated. These estimates may also be conservative given these questions were only asked to those without any activity of daily living or instrumental activity of daily living limitations.

We next combine responses to questions from experimental modules and examine whether genes predict one's self-reported probability of developing AD or dementia in the future. Sample size decreases dramatically as experimental modules were fielded to a very small share of respondents and we require that these individuals be genotyped and aged 50–70. Results are presented in columns (9) and (10) of Table 9. Those who carry one copy of APOE report a statistically significant 6.5–6.9 percentage point higher probability of developing ADRD. The association between carrying two copies of APOE and expectations of developing ADRD, obtained by taking the sum of the APOE coefficients, is negative with very large standard errors. 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 likely positively selected on cognitive function, which may make these estimates conservative.

Taken together, these results suggest that APOE carriers are aware of their elevated risk, while, on average, those with higher AD scores are not. APOE carriers may receive stronger signals of their risk if their parents experienced cognitive decline or were 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 (11) and (12) in Table 9. Carrying at least one copy of APOE associates with a 2 percentage point increase in the probability of parental MRD diagnosis. A one standard deviation increase in the AD score is associated with a 0.5 percentage point increase in that probability. Questions about parental

MRD were not asked until 1998 and only if the parent is alive. Thus, our estimates are likely conservative, as parental MRD is understated for respondents who have one living cognitively healthy parent but the other had MRD and died before the 1998 survey wave (or 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 parent used a nursing home by that point. We include only one observation per individual because information about parental nursing home use is gleaned from both a question about where parents who are 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 are shown in columns (13) and (14) in Table 9. The coefficients on all the ADRD genetic risk variables are positive, but only the coefficients on carrying two copies of the APOE allele are statistically significant. The estimates suggest that those carrying two copies of APOE are 14 percentage points ($p < 0.01$) 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 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.

5.6 Robustness and Sensitivity

5.6.1 Sample Attrition

The estimated associations between genetic risk for ADRD and the 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 with proxy interviews 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 overall sample attrition and attrition due to proxy interviews

and death. We create an indicator that has value one if a self-respondent is a self-respondent in the following wave and zero otherwise. If an individual does not self-respond next wave, they either responded via proxy, passed away, or did not respond 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. In the full sample, APOE carriers and those with higher AD scores are less likely to self-respond in the next survey wave. These relationships attenuate and lose statistical significance when controls for current cognition are included, and the coefficients on the APOE indicators 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. Results are presented in Appendix Table A22. Among the full sample aged 50–85, APOE carriers have a higher probability of a proxy interview next wave. These estimates 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 among those at higher ADRD genetic risk but largely for those over age 70. Using information from HRS exit interviews, we create an indicator equal to one if the individual dies before the next survey wave and zero otherwise. The point estimates on the AD score and indicator for carrying at least one copy of APOE presented in Appendix Table A23 are generally close to zero and not statistically significant. The coefficients on the indicator for carrying two copies of APOE are all positive, larger in magnitude, and reach the 10% significance level in a few cases. Thus, increased mortality explains some of the sample attrition among those carrying two copies of APOE. 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, which would tend to make the estimated relationships between genetic risk and the outcomes we consider conservative.

5.6.2 Relationships Among the Never Demented

Our results suggest that those at elevated polygenic risk for ADRD are a particularly vulnerable group as they have worse economic outcomes, are less likely to engage in later-life planning activities, and are unaware 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 early-life SES and current cognitive performance. We further test 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 (never register a 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 6, 7, and 9 and replace the

"never MRD" columns with results from the "never demented" sample. The results are shown in Tables A24–A26. Overall, the results from the never-demented sample are very similar to those from the full and never-diagnosed samples. These findings underscore that elevated risk for ADRD, especially polygenic risk, is relevant even in the absence of clinical presentations of ADRD.

5.6.3 Including the Educational Attainment Polygenic Score

We have also re-estimated our models additionally controlling for the polygenic score for educational attainment (the EA score) based on the GWAS of Lee et al. (2018). Genetic markers that predict educational attainment have been shown to also predict ADRD and cognitive decline (Ding et al. 2019, Anderson et al. 2020). Furthermore, education has 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 cognitive reserve (Stern 2012), although it is not clear if this reflects a causal effect of education on cognition or operation of brain features that both promote education and add to cognitive reserve later in life. Throughout our analyses we have flexibly controlled for educational attainment, but genetic factors linked to educational attainment may predict the outcomes we consider over and above their influence on completed education (Belsky et al. 2016, Barth, Papageorge, and Thom 2020, Papageorge and Thom 2020). Indeed, we find a higher EA score is associated with better cognitive function, better economic outcomes (e.g., higher probabilities of working for pay and household wealth), and in some cases more engagement in planning activities even when we control for educational attainment (and our usual set of controls). This suggests that individuals with higher EA scores may have genetic endowments that can offset risk related to APOE carrier status or the genetic factors measured by the AD scores. Importantly, however, adding the EA score to our regression models has little to no impact on coefficient estimates for the AD score or APOE indicator variables, and we find no evidence of interactions between these genetic measures. Results that include the EA score are available by request.

6 Conclusion

We explore whether individuals with higher observed genetic risk for ADRD engage in economic and long-run planning that may mitigate the consequences of ADRD. We first show that higher genetic risk for ADRD, as measured by a polygenic score for ADRD and APOE carrier status, predicts worse cognitive function in levels and changes and an increased probability of being diagnosed with a memory-related disease, consistent with prior work. We also demonstrate that the genetic measures of ADRD risk contain information that is quantitatively substantial and of clinical value—that is, they predict future cognitive decline among those who are cognitively

healthy even after controlling for current and past cognitive performance, demographics, and familial dementia history. Furthermore, higher polygenic risk for ADRD associates with a lower probability of working and less wealth. These results imply those at higher genetic risk for ADRD, especially polygenic risk, stand to gain from precautionary planning. Yet, those with higher polygenic risk for ADRD are less likely to engage in planning activities, and they appear to be unaware of their increased prospects of diminished cognition.

The collection of results suggests that observable genetic measures of ADRD risk associate with significant harms, reflect health risks for which individuals are relatively unprepared, and contain information above and beyond current knowledge or expectations. Genetic screening could, therefore, be valuable for individuals and their healthcare providers as part of an early-warning system. Importantly, because genetic factors can be observed earlier in life, information about genetic risk may allow individuals to take steps to safeguard themselves and their families before harms, such as financial mistakes or declines in wealth, have been realized. Genetic screening may also help identify high-risk individuals for increased monitoring and screening. Although ADRD is currently incurable, as the treatment landscape improves, it becomes even more important to identify high-risk individuals so treatment can begin early in the disease progression if medically appropriate.

Our results also point to an interesting distinction between the genetic risks related to APOE carrier status and the AD score. Carrying the risky APOE allele is associated with dramatic declines in cognition later in life, but not with cognition or economic outcomes earlier in the life-cycle. There is also some weak evidence that APOE carriers are aware of their elevated risk and may engage in some planning activities, consistent with standard theoretical predictions. On the other hand, higher polygenic scores for ADRD are associated with lower cognitive performance earlier in life, less advantageous economic outcomes, and significantly less planning for long-run health risks. A potential explanation is that these correlations reflect the same biological process. Genetic factors that reduce cognitive performance earlier in life can increase the chance of cognition falling into impaired or demented states later in life. Precisely because these factors impact cognition early in life, they may also diminish an individual's capacity for the kinds of sophisticated economic and legal planning that could insulate themselves and their families from harms related to future ADRD. Regardless of the specific mechanism, it may be particularly important to screen individuals for elevated polygenic risk if they are also less likely to be prepared for the possibility of future ADRD.

Our study raises new questions. Our proposed explanation for the differences between APOE carriers and those at higher polygenic risk for ADRD is speculative, and future work can explore if these differences are systematic and if they reflect distinct biological pathways. A very clear next step is to try to randomize genetic screening for both APOE and polygenic ADRD risk, and

to estimate whether such treatments affect household preparation and long-run outcomes. Our results highlight some important considerations that should shape how such information revelation exercises are performed. First, it may be important to clearly communicate the polygenic nature of genetic risk for ADRD, and to reveal total genetic risk and not just APOE status. Second, individuals at higher risk for future ADRD might be constrained in how they react to such information because of different capacities for complex legal or financial planning. Simply revealing genetic information may be insufficient, and the value of genetic screening might only be apparent if it is paired with counseling or advice about the range of planning options. Finally, future research should also take into account the interplay between genetic endowments and environmental risk factors for ADRD. Better understanding the policies or economic environments that moderate or exacerbate genetic risk for ADRD can shed light on the pathways through which genetic factors operate and the frictions that may prevent people from recognizing or responding to such risk.

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Tables and Figures

Table 1: Summary Statistics

	Mean	SD	N
Demographics:			
Birth Year	1940.222	9.782	88,048
Male	0.419	0.493	88,048
Age	67.553	9.000	88,048
Years of Education	13.315	2.499	88,048
At Least Some College	0.322	0.467	88,048
Parental Education:			
Mother's Years of Education	10.584	3.056	82,405
Missing	0.064	0.245	88,048
Self-Reported Family SES During Childhood:			
Well-Off or Average	0.729	0.444	88,048
Missing	0.014	0.117	88,048
Genetic Data:			
AD Score	-0.009	1.000	88,048
APOE (At least 1 copy)	0.260	0.439	88,048
APOE (2 copies)	0.020	0.138	88,048
Cognition and Memory-Related Disease (MRD):			
TICS-M Score	16.392	4.004	88,048
Ever Demented (TICS-M < 7)	0.028	0.165	88,048
Ever Impaired or Demented (TICS-M < 12)	0.217	0.412	88,048
Ever Diagnosed with MRD	0.021	0.144	88,048
Economic Outcomes:			
Work for Pay	0.563	0.496	53,592
Retired	0.320	0.467	49,564
Individual Total Income ($)	33,010	32,290	81,393
Household Total Wealth ($)	588,604	843,313	84,548
Planning Outcomes:			
Holds Long-Term Care Insurance (LTCI)	0.126	0.332	52,747
Holds Life Insurance	0.714	0.452	53,322
Has a Witnessed Will	0.559	0.497	53,484
Has a Living Will	0.478	0.500	5,066
Has Assigned Someone Durable Power of Attorney for Health Care	0.462	0.499	5,067
Discuss Future Medical Care with Anyone	0.590	0.492	3,500
Awareness Outcomes:			
Self-Reported Probability of Living to Age 75	66.275	26.389	36,282
Self-Reported Probability of Moving to Nursing Home	10.982	17.207	16,795
Self-Reported Probability of Living Independently at Age 75	69.999	23.087	4,178
Self-Reported Probability of Being Cognitively Healthy at Age 75	65.779	23.413	4,168
Self-Reported Probability of Developing ADRD	36.303	26.128	660
Parent Ever Diagnosed with MRD	0.070	0.255	23,065
Parent Received Nursing Home Care	0.379	0.485	4,344

Note: The table presents summary statistics at the person-wave level from 1998–2018. We calculate them using the maximum number of person-year observations that meet our sample inclusion criteria for that variable or outcome.

Table 2: Relationship between Genetic Endowments and Cognition

	(1)	(2)	(3)	(4)	(5)	(6)
	Full Sample			Never Diagnosed		
	Panel A: TICS-M Score					
AD Score	-0.236***	-0.188***	-0.134***	-0.225***	-0.175***	-0.122***
	(0.035)	(0.034)	(0.031)	(0.036)	(0.035)	(0.032)
APOE (At least 1 copy)	-0.404***	-0.356***	-0.381***	-0.267***	-0.235***	-0.248***
	(0.065)	(0.065)	(0.059)	(0.066)	(0.067)	(0.060)
APOE (2 copies)	-0.558**	-0.605***	-0.526***	-0.391	-0.477**	-0.291
	(0.218)	(0.216)	(0.200)	(0.239)	(0.236)	(0.206)
Childhood SES Controls	No	Yes	Yes	No	Yes	Yes
Education Controls	No	No	Yes	No	No	Yes
Mean	16.392	16.392	16.392	16.648	16.648	16.648
N	88,048	88,048	88,048	80,264	80,264	80,264
R^2	0.133	0.170	0.252	0.118	0.155	0.242
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
	Panel B: Ever Impaired or Demented (TICS-M Score $<$ 12)					
AD Score	0.006***	0.005***	0.004***	0.006***	0.005***	0.004***
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
APOE (At least 1 copy)	0.013***	0.011***	0.011***	0.007***	0.006***	0.006***
	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)
APOE (2 copies)	0.022***	0.023***	0.023***	0.017**	0.020***	0.018**
	(0.007)	(0.007)	(0.007)	(0.007)	(0.007)	(0.007)
Childhood SES Controls	No	Yes	Yes	No	Yes	Yes
Education Controls	No	No	Yes	No	No	Yes
Mean	0.054	0.054	0.054	0.047	0.047	0.047
N	72,823	72,823	72,823	67,631	67,631	67,631
R^2	0.022	0.029	0.048	0.018	0.025	0.043
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 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 (5), we control for childhood socioeconomic status via an indicator for whether the respondent reports that their family was well off financially or about average from birth to age 16 (versus being poor) as well as dummy variables for their mother's years of education. In columns (3) and (6), we add controls for own educational attainment. Columns (4)-(6) are estimated only on those never diagnosed with a memory-related disease 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 Cognition Changes

	(1)	(2)	(3)	(4)	(5)	(6)
	Full Sample			Never Diagnosed		
	Panel A: Change in TICS-M Score between t and $t-1$					
AD Score	-0.023**	-0.022**	-0.022**	-0.020**	-0.018*	-0.018*
	(0.010)	(0.010)	(0.010)	(0.010)	(0.010)	(0.010)
APOE (At least 1 copy)	-0.151***	-0.146***	-0.148***	-0.093***	-0.085***	-0.086***
	(0.019)	(0.019)	(0.019)	(0.019)	(0.019)	(0.019)
APOE (2 copies)	-0.209***	-0.218***	-0.216***	-0.140*	-0.157**	-0.154**
	(0.068)	(0.069)	(0.069)	(0.072)	(0.072)	(0.072)
Childhood SES Controls	No	Yes	Yes	No	Yes	Yes
Education Controls	No	No	Yes	No	No	Yes
Mean	-0.348	-0.348	-0.348	-0.288	-0.288	-0.288
N	56,741	56,741	56,741	52,039	52,039	52,039
R^2	0.010	0.010	0.010	0.009	0.009	0.009
Years	2002-2018	2002-2018	2002-2018	2002-2018	2002-2018	2002-2018
Ages	50-85	50-85	50-85	50-85	50-85	50-85
	Panel B: Change in TICS-M Score between t and $t-2$					
AD Score	-0.043***	-0.039***	-0.038***	-0.037***	-0.031**	-0.030**
	(0.014)	(0.015)	(0.015)	(0.014)	(0.014)	(0.014)
APOE (At least 1 copy)	-0.237***	-0.229***	-0.234***	-0.139***	-0.130***	-0.133***
	(0.028)	(0.029)	(0.029)	(0.027)	(0.028)	(0.028)
APOE (2 copies)	-0.426***	-0.432***	-0.432***	-0.307***	-0.326***	-0.324***
	(0.099)	(0.100)	(0.100)	(0.101)	(0.102)	(0.101)
Childhood SES Controls	No	Yes	Yes	No	Yes	Yes
Education Controls	No	No	Yes	No	No	Yes
Mean	-0.668	-0.668	-0.668	-0.570	-0.570	-0.570
N	56,741	56,741	56,741	52,039	52,039	52,039
R^2	0.020	0.021	0.021	0.016	0.017	0.017
Years	2002-2018	2002-2018	2002-2018	2002-2018	2002-2018	2002-2018
Ages	50-85	50-85	50-85	50-85	50-85	50-85
	Panel C: Change in TICS-M Score between t and $t-3$					
AD Score	-0.049***	-0.045***	-0.043**	-0.044***	-0.039**	-0.036**
	(0.017)	(0.018)	(0.018)	(0.017)	(0.017)	(0.017)
APOE (At least 1 copy)	-0.304***	-0.295***	-0.299***	-0.172***	-0.159***	-0.162***
	(0.035)	(0.036)	(0.036)	(0.034)	(0.035)	(0.035)
APOE (2 copies)	-0.638***	-0.635***	-0.630***	-0.549***	-0.558***	-0.547***
	(0.120)	(0.122)	(0.122)	(0.122)	(0.124)	(0.123)
Childhood SES Controls	No	Yes	Yes	No	Yes	Yes
Education Controls	No	No	Yes	No	No	Yes
Mean	-0.910	-0.910	-0.910	-0.781	-0.781	-0.781
N	56,741	56,741	56,741	52,039	52,039	52,039
R^2	0.030	0.030	0.031	0.024	0.024	0.025
Years	2002-2018	2002-2018	2002-2018	2002-2018	2002-2018	2002-2018
Ages	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 Panels A, B, and C, the outcome is the change in the TICS-M cognition score from wave $t-1$ to t, $t-2$ to t, and $t-3$ to t, respectively. We only include respondents observed continuously from $t-3$ to t. In all specifications, we control for the first 10 principal components of the genetic data as well as the standard controls for age, birth cohort, sex of respondent, and survey year as described in Section 4. In columns (2) and (5), we control for childhood socioeconomic status via an indicator for whether the respondent reports that their family was well off financially or about average from birth to age 16 (versus being poor) as well as dummy variables for their mother's years of education. In columns (3) and (6), we add controls for own educational attainment. Columns (4)-(6) are estimated only on those never diagnosed with a memory-related disease 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 4: Relationship between Genetic Endowments and Memory-Related Disease Diagnosis

	(1)	(2)	(3)
	Ever MRD Diagnosis		
AD Score	0.001***	0.001***	0.001***
	(0.000)	(0.000)	(0.000)
APOE (At least 1 copy)	0.005***	0.004***	0.004***
	(0.001)	(0.001)	(0.001)
APOE (2 copies)	0.010***	0.010***	0.010***
	(0.003)	(0.003)	(0.003)
Childhood SES Controls	No	Yes	Yes
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 control for childhood socioeconomic status via an indicator for whether the respondent reports that their family was well off financially or about average from birth to age 16 (versus being poor) as well as dummy variables for their mother's years of education. In column (3), we add controls for own educational attainment. 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 Future (Age 65–80) Cognitive Outcomes

	(1) Ever Impaired	(2) Ever Demented	(3) Ever Diagnosed	(4) Ever Proxy	(5) Ever Demented, Diagnosed, or Proxy
AD Score	0.022***	0.006**	0.012***	0.006	0.016***
	(0.007)	(0.003)	(0.004)	(0.004)	(0.005)
APOE (At least 1 copy)	0.060***	0.030***	0.042***	0.015**	0.046***
	(0.013)	(0.007)	(0.008)	(0.008)	(0.010)
APOE (2 copies)	0.117***	0.049	0.090**	0.055*	0.120***
	(0.044)	(0.030)	(0.038)	(0.032)	(0.042)
Childhood SES Controls	Yes	Yes	Yes	Yes	Yes
Education Controls	Yes	Yes	Yes	Yes	Yes
Parental MRD Controls	Yes	Yes	Yes	Yes	Yes
Early-Period Cognition Controls	Yes	Yes	Yes	Yes	Yes
Mean	0.203	0.028	0.040	0.047	0.086
N	4,860	4,860	4,871	4,871	4,871
R^2	0.216	0.074	0.065	0.057	0.095

Note: Each column presents results from a separate regression. In column (1), the outcome is an indicator for an observation of impairment (TICS-M score < 12) in the age range 65–80. In column (2), the outcome is an indicator for an observation of dementia (TICS-M score < 7) in this age range, and in column (3), the outcome is an indicator for being diagnosed with a memory-related disease (MRD) in this age range. In column (4), the outcome is an indicator for having an interview wave completed by a proxy respondent during ages 65–80. In column (5), the outcome is an indicator for at least one of the following events during ages 65–80: an observation of dementia, MRD diagnosis, or a proxy interview. We restrict the sample to individuals who were not impaired, demented, or diagnosed with MRD 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 an indicator for whether the respondent reports that their family was well off financially or about average from birth to age 16 (versus being poor), dummy variables for their mother's years of education, the average, minimum, and maximum TICS-M scores observed for ages 50–64 as well as an indicator for whether a parent was diagnosed with MRD while the respondent was aged 50–64 and an indicator for missing parental MRD information. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

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

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Currently Working for Pay		Retirement		Log Individual Total Income		Log Household Total Wealth	
AD Score	-0.010**	-0.009*	0.007	0.007	-0.012	-0.005	-0.038**	-0.030*
	(0.005)	(0.005)	(0.004)	(0.005)	(0.008)	(0.008)	(0.016)	(0.017)
APOE (At least 1 copy)	0.010	0.008	-0.011	-0.008	-0.003	0.000	0.000	-0.005
	(0.009)	(0.009)	(0.008)	(0.008)	(0.014)	(0.015)	(0.030)	(0.032)
APOE (2 copies)	-0.019	0.009	0.043*	0.027	0.001	0.034	0.061	0.018
	(0.027)	(0.029)	(0.025)	(0.026)	(0.040)	(0.042)	(0.097)	(0.108)
Childhood SES Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Education Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
TICS-M Score Dummies	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	0.186	0.188	0.223	0.226	0.276	0.276	0.185	0.181
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 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 an indicator for whether the respondent reports that their family was well off financially or about average from birth to age 16 (versus being poor), dummy variables for their mother's years of education, dummy variables for own 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 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	-0.009**	-0.008**	-0.005	-0.003	-0.011*	-0.009
	(0.004)	(0.004)	(0.005)	(0.005)	(0.006)	(0.006)
APOE (At least 1 copy)	0.007	0.009	0.002	0.004	0.011	0.011
	(0.007)	(0.007)	(0.009)	(0.010)	(0.011)	(0.011)
APOE (2 copies)	-0.004	-0.008	-0.008	-0.032	-0.008	-0.008
	(0.020)	(0.020)	(0.027)	(0.028)	(0.032)	(0.035)
Childhood SES Controls	Yes	Yes	Yes	Yes	Yes	Yes
Education Controls	Yes	Yes	Yes	Yes	Yes	Yes
TICS-M Score Dummies	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	0.046	0.047	0.057	0.060	0.137	0.137
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	-0.016	-0.013	-0.039***	-0.037***	-0.023**	-0.023**
	(0.012)	(0.012)	(0.012)	(0.012)	(0.011)	(0.011)
APOE (At least 1 copy)	0.024	0.029	0.009	0.009	-0.009	-0.001
	(0.023)	(0.024)	(0.023)	(0.023)	(0.020)	(0.021)
APOE (2 copies)	0.086	0.086	0.078	0.118*	-0.033	-0.020
	(0.063)	(0.067)	(0.065)	(0.066)	(0.064)	(0.069)
Childhood SES Controls	Yes	Yes	Yes	Yes	Yes	Yes
Education Controls	Yes	Yes	Yes	Yes	Yes	Yes
TICS-M Score Dummies	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	0.111	0.115	0.108	0.112	0.132	0.133
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 an indicator for whether the respondent reports that their family was well off financially or about average from birth to age 16 (versus being poor), dummy variables for their mother's years of education, dummy variables for own 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 8: Relationship between Genetic Endowments and Number of Later-Life Planning Activities

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)
	Probability of Number of Planning Activities													
	≥ 1		≥ 2		≥ 3		≥ 4		≥ 5		≥ 6		Count (0-6)	
AD Score	0.004	0.007	-0.002	0.003	-0.033***	-0.028**	-0.022**	-0.018	-0.016*	-0.014	-0.009*	-0.007	-0.078**	-0.057
	(0.007)	(0.007)	(0.012)	(0.012)	(0.012)	(0.012)	(0.011)	(0.011)	(0.009)	(0.009)	(0.005)	(0.005)	(0.040)	(0.041)
APOE (At least 1 copy)	-0.005	-0.000	0.010	0.015	0.028	0.036	0.014	0.018	0.000	0.005	0.013	0.015	0.060	0.088
	(0.013)	(0.013)	(0.021)	(0.022)	(0.023)	(0.023)	(0.021)	(0.022)	(0.017)	(0.018)	(0.010)	(0.010)	(0.076)	(0.077)
APOE (2 copies)	0.036	0.029	0.019	0.010	0.040	0.008	0.067	0.080	-0.009	0.019	0.011	0.008	0.163	0.154
	(0.031)	(0.035)	(0.067)	(0.072)	(0.071)	(0.076)	(0.067)	(0.072)	(0.051)	(0.055)	(0.032)	(0.032)	(0.228)	(0.247)
Childhood SES Controls	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
TICS-M Score Dummies	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	0.128	0.119	0.120	0.122	0.139	0.144	0.120	0.127	0.092	0.094	0.076	0.074	0.163	0.168
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

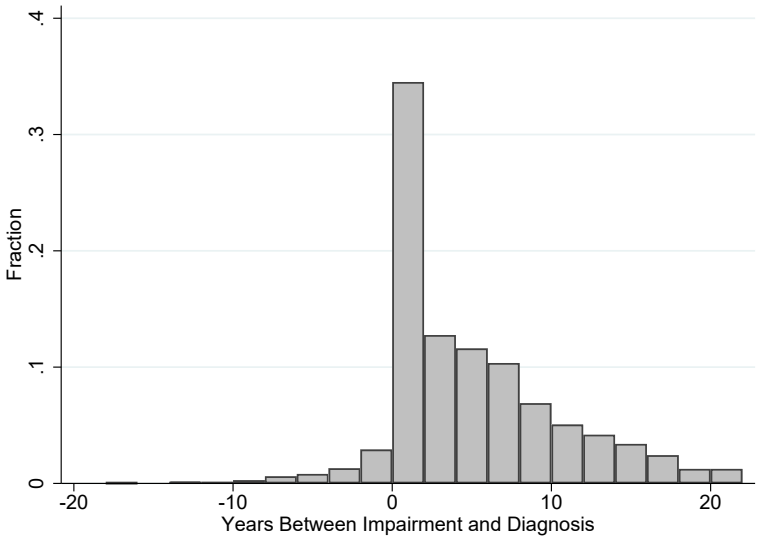
Note: In columns (1)–(12), each column presents results from a separate regression where the outcome is an indicator variable for engaging in at least a certain number of planning activities. In columns (13)–(14), the outcome is the total number of planning activities the respondent engages in. The sample 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 an indicator for whether the respondent reports that their family was well off financially or about average from birth to age 16 (versus being poor), dummy variables for their mother's years of education, dummy variables for own 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 9: Relationship between Genetic Endowments and Awareness of Risk

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)
	Probability of Living to Age 75		Probability of Moving to Nursing Home in Next 5 Years		Probability of Living Independently at Age 75		Probability of Cognitively Healthy at Age 75		Probability of Developing ADRD		Parental Diagnosis of MRD		Parental Use of Nursing Home Care	
AD Score	0.384	0.455	-0.057	-0.083	0.287	0.226	0.375	0.257	-1.162	-1.285	0.005**	0.005**	0.004	0.005
	(0.309)	(0.315)	(0.216)	(0.221)	(0.492)	(0.496)	(0.513)	(0.520)	(1.703)	(1.755)	(0.002)	(0.002)	(0.010)	(0.010)
APOE (At least 1 copy)	-0.536	-0.740	0.541	0.649	-1.985**	-2.185**	-2.182**	-2.335**	6.866**	6.518**	0.023***	0.021***	0.027	0.026
	(0.571)	(0.582)	(0.398)	(0.414)	(0.931)	(0.934)	(0.917)	(0.918)	(2.756)	(2.877)	(0.005)	(0.005)	(0.018)	(0.019)
APOE (2 copies)	-1.687	-1.526	0.117	-1.659	0.121	0.531	0.881	1.475	-8.404	-9.149	-0.001	0.007	0.116**	0.118**
	(1.927)	(2.076)	(1.377)	(1.425)	(2.561)	(2.699)	(2.753)	(2.921)	(8.154)	(8.763)	(0.014)	(0.016)	(0.053)	(0.055)
Childhood SES	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
TICS-M Score Dummies	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	66.275	66.645	10.982	10.847	69.999	70.451	65.779	66.298	36.303	36.105	0.070	0.070	0.379	0.377
N	36,282	34,611	16,795	15,426	4,178	4,039	4,168	4,031	660	638	23,065	22,041	4,344	4,200
R^2	0.094	0.093	0.030	0.029	0.143	0.139	0.151	0.147	0.373	0.373	0.033	0.032	0.089	0.090
Years	1998-2018	1998-2018	1998-2018	1998-2018	2006-2008	2006-2008	2006-2008	2006-2008	2002, 2012, 2016	2002, 2012, 2016	1998-2018	1998-2018	2006-2018	2006-2018
Ages	50-65	50-65	65-70	65-70	50-65	50-65	50-65	50-65	50-70	50-70	50-70	50-70	50-70	50-70

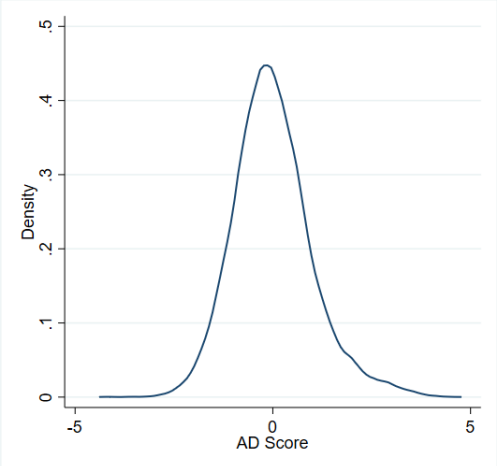
Note: Each column within a panel presents results from a separate regression where the outcome is described in the column heading. In all specifications, we control for the first 10 principal components of the genetic data as well as the standard controls for age, birth cohort, sex of respondent, and survey year as described in Section 4 as well as an indicator for whether the respondent reports that their family was well off financially or about average from birth to age 16 (versus being poor), dummy variables for their mother's years of education, dummy variables for own 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 $p < 0.01$.

Figure 1: Distribution of Gap Between First Evidence of Cognitive Impairment and Diagnosis (Years)



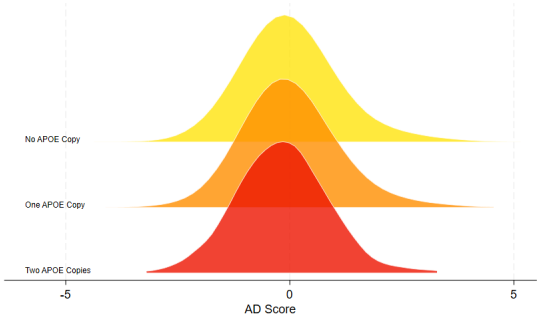
Note: The figure shows a histogram of the gap (in years) between the age at which an individual was first diagnosed with a memory-related disease and the age at which they first showed evidence of cognitive impairment on the TICS-M cognitive test administered as part of the HRS (i.e., TICS-M < 12). The sample includes all 3,911 individuals who eventually report being diagnosed with a memory-related disease. For individuals who are never observed with a cognitive test score meeting the criteria for cognitive impairment, we set the age of first impairment equal to the age of first diagnosis, which will tend to understate the severity of underdiagnosis in the sample.

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



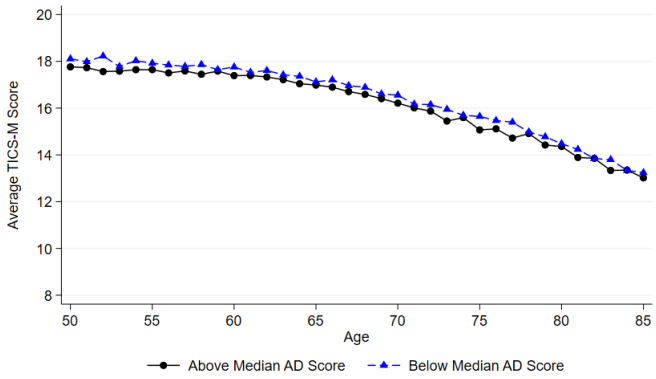
Note: The figure shows the smoothed density of the polygenic score for Alzheimer's disease in our sample.

Figure 3: Conditional Distribution of the AD Score by APOE Carrier Status

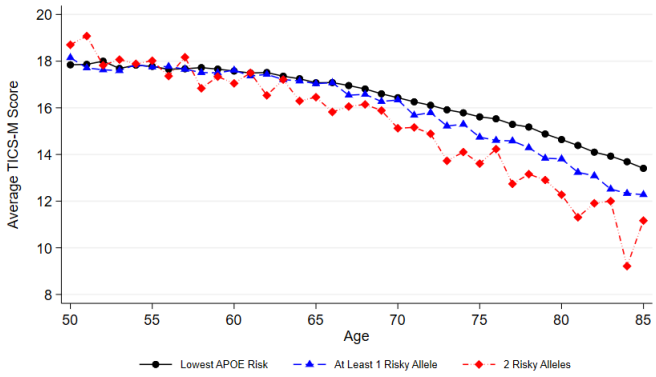


Note: The figure shows the distribution of the AD score conditional on APOE carrier status.

Figure 4: Age-Cognition Profiles by Genetic Risk Groups



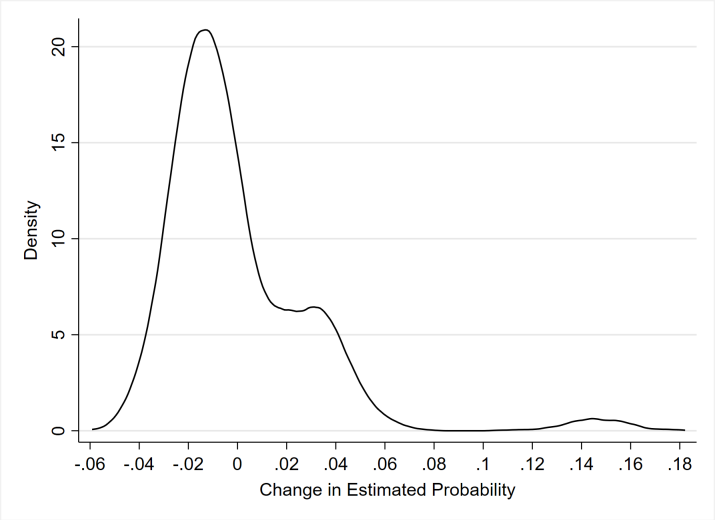
(a) TICS-M Score by Above vs. Below Median AD Score



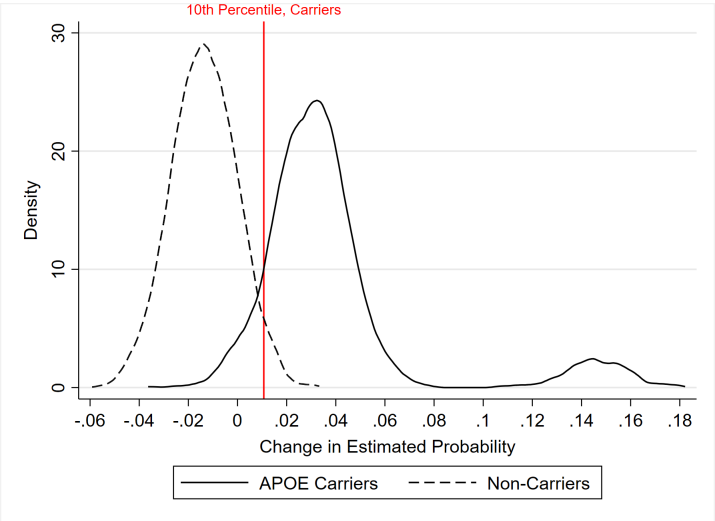
(b) TICS-M Score by APOE Carrier Status

Note: Each subfigure plots average values of the 27-point TICS-M cognition score by age for different genetic risk groups.

Figure 5: 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 and APOE measures into a predictive linear probability model. Panel (a) presents the distribution of such changes in the full sample. Panel (b) presents the distribution of changes separately for APOE carriers and non-carriers.

Appendix

Table A1: Summary Statistics for 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	0.235	0.424	48,541
Parental Education:			
Mother's Years of Education	8.992	4.073	43,623
Missing	0.101	0.302	48,541
Self-Reported Family SES During Childhood:			
Well-Off or Average	0.684	0.465	48,541
Missing	0.012	0.110	48,541
Cognition and Memory-Related Disease (MRD):			
TICS-M Score	15.100	4.636	48,541
Ever Demented (TICS-M < 7)	0.072	0.259	48,541
Ever Impaired or Demented (TICS-M < 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 ($)	25,874	30,513	48,541
Household Total Wealth ($)	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:			
Self-Reported Probability of Living to Age 75	59.922	30.647	21,570
Self-Reported Probability of Moving to Nursing Home	11.127	19.621	7,474
Self-Reported Probability of Living Independently at Age 75	65.948	25.725	1,297
Self-Reported Probability of Being Cognitively Healthy at Age 75	61.739	25.811	1,285
Self-Reported Probability of Developing ADRD	32.037	26.821	461
Parent Ever Diagnosed with MRD	0.245	0.430	16,099
Parent Received Nursing Home Care	0.332	0.471	30,581

Note: The table presents summary statistics at the person-wave level from 1998–2018. We calculate them using the maximum number of non-genotyped person-year observations that otherwise meet our sample inclusion criteria for that variable or outcome.

Table A2: Relationship between Genetic Endowments and Probability of Being Demented

	(1)	(2)	(3)	(4)	(5)	(6)
	Ever Demented (TICS-M Score < 7)					
	Full Sample			Never Diagnosed		
AD Score	0.001***	0.001**	0.001**	0.001**	0.001**	0.001*
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
APOE (At least 1 copy)	0.006***	0.006***	0.006***	0.002***	0.002***	0.002***
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
APOE (2 copies)	0.010***	0.010***	0.009**	0.008**	0.009***	0.008**
	(0.003)	(0.003)	(0.004)	(0.003)	(0.003)	(0.003)
Childhood SES Controls	No	Yes	Yes	No	Yes	Yes
Education Controls	No	No	Yes	No	No	Yes
Mean	0.009	0.009	0.009	0.006	0.006	0.006
N	86,412	86,412	86,412	79,119	79,119	79,119
R^2	0.013	0.015	0.026	0.009	0.010	0.023
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 an indicator for a TICS-M score below 7 in the current wave, and the sample excludes individuals after their first observed TICS-M score below 7. In all specifications, we control for the first 10 principal components of the genetic data as well as the standard controls for age, birth cohort, sex of respondent, and survey year as described in Section 4. In columns (2) and (5), we control for childhood socioeconomic status via an indicator for whether the respondent reports that their family was well off financially or about average from birth to age 16 (versus being poor) as well as dummy variables for their mother's years of education. In columns (3) and (6), we add controls for own educational attainment. Columns (4)-(6) are estimated only on those never diagnosed with a memory-related disease 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 Future (Age 65–80) Cognitive Outcomes Including Controls for Modifiable Risk Factors

	(1) Ever Impaired	(2) Ever Demented	(3) Ever Diagnosed	(4) Ever Proxy	(5) Ever Demented, Diagnosed, or Proxy
AD Score	0.021***	0.006**	0.011***	0.006	0.016***
	(0.007)	(0.003)	(0.004)	(0.004)	(0.005)
APOE (At least 1 copy)	0.061***	0.031***	0.041***	0.016**	0.047***
	(0.014)	(0.007)	(0.008)	(0.008)	(0.010)
APOE (2 copies)	0.117***	0.048	0.092**	0.054*	0.120***
	(0.044)	(0.030)	(0.038)	(0.032)	(0.043)
Childhood SES Controls	Yes	Yes	Yes	Yes	Yes
Education Controls	Yes	Yes	Yes	Yes	Yes
Early-Period Cognition Controls	Yes	Yes	Yes	Yes	Yes
Early-Period Risk Factor Controls	Yes	Yes	Yes	Yes	Yes
Mean	0.203	0.028	0.040	0.047	0.085
N	4,831	4,831	4,842	4,842	4,842
R^2	0.218	0.076	0.067	0.058	0.097

Note: Each column presents results from a separate regression. In column (1), the outcome is an indicator for an observation of impairment (TICS-M score < 12) in the age range 65–80. In column (2), the outcome is an indicator for an observation of dementia (TICS-M score < 7) in this age range, and in column (3), the outcome is an indicator for being diagnosed with a memory-related disease (MRD) in this age range. In column (4), the outcome is an indicator for having an interview wave completed by a proxy respondent during ages 65–80. In column (5), the outcome is an indicator for at least one of the following events during ages 65–80: an observation of dementia, MRD diagnosis, or a proxy interview. We restrict the sample to individuals who were not impaired, demented, or diagnosed with MRD 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 an indicator for whether the respondent reports that their family was well off financially or about average from birth to age 16 (versus being poor), dummy variables for their mother's years of education, the average, minimum, and maximum TICS-M scores observed for ages 50–64 as well as an indicator for whether a parent was diagnosed with MRD while the respondent was aged 50–64 and an indicator for missing parental MRD information. Finally, we control for modifiable risk factors including indicators for ever smoked, ever have hypertension, ever have diabetes, ever have a heart condition, ever wear a hearing aid, ever register a Center for Epidemiologic Studies Depression (CES-D) score consistent with depression, and ever obese in the age range 50–64. * for $p < 0.10$, ** for $p < 0.05$, and *** for $p < 0.01$.

Table A4: Relationship between Genetic Endowments and Currently Working for Pay

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Currently Working for Pay							
	Full Sample				Never Diagnosed			
AD Score	-0.017***	-0.014***	-0.011**	-0.010**	-0.016***	-0.013**	-0.010*	-0.009*
	(0.005)	(0.005)	(0.005)	(0.005)	(0.005)	(0.005)	(0.005)	(0.005)
APOE (At least 1 copy)	0.005	0.011	0.009	0.010	0.005	0.010	0.007	0.008
	(0.009)	(0.009)	(0.009)	(0.009)	(0.009)	(0.009)	(0.009)	(0.009)
APOE (2 copies)	-0.011	-0.031	-0.023	-0.019	0.014	-0.007	0.008	0.009
	(0.026)	(0.027)	(0.028)	(0.027)	(0.027)	(0.028)	(0.029)	(0.029)
Childhood SES Controls	No	Yes	Yes	Yes	No	Yes	Yes	Yes
Education Controls	No	No	Yes	Yes	No	No	Yes	Yes
TICS-M Score Dummies	No	No	No	Yes	No	No	No	Yes
Mean	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	50,485	50,485	50,485	50,485
R^2	0.151	0.164	0.179	0.186	0.154	0.166	0.182	0.188
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	50-70	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 (6), we control for childhood socioeconomic status via an indicator for whether the respondent reports that their family was well off financially or about average from birth to age 16 (versus being poor) as well as dummy variables for their mother's years of education. In columns (3) and (7), we add controls for own educational attainment. In columns (4) and (8), we add dummy variables for each value of the most current TICS-M score. Columns (5)-(8) are estimated only on those never diagnosed with a memory-related disease 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 Retirement

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Retirement							
	Full Sample				Never Diagnosed			
AD Score	0.012***	0.009**	0.007	0.007	0.011**	0.009**	0.007	0.007
	(0.004)	(0.005)	(0.004)	(0.004)	(0.005)	(0.005)	(0.005)	(0.005)
APOE (At least 1 copy)	-0.008	-0.013	-0.011	-0.011	-0.007	-0.011	-0.008	-0.008
	(0.008)	(0.008)	(0.008)	(0.008)	(0.008)	(0.008)	(0.008)	(0.008)
APOE (2 copies)	0.037	0.051**	0.045*	0.043*	0.024	0.038	0.028	0.027
	(0.024)	(0.025)	(0.025)	(0.025)	(0.025)	(0.026)	(0.026)	(0.026)
Childhood SES Controls	No	Yes	Yes	Yes	No	Yes	Yes	Yes
Education Controls	No	No	Yes	Yes	No	No	Yes	Yes
TICS-M Score Dummies	No	No	No	Yes	No	No	No	Yes
Mean	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	46,907	46,907	46,907	46,907
R^2	0.203	0.210	0.219	0.223	0.206	0.213	0.222	0.226
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	50-70	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 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 (6), we control for childhood socioeconomic status via an indicator for whether the respondent reports that their family was well off financially or about average from birth to age 16 (versus being poor) as well as dummy variables for their mother's years of education. In columns (3) and (7), we add controls for own educational attainment. In columns (4) and (8), we add dummy variables for each value of the most current TICS-M score. Columns (5)-(8) are estimated only on those never diagnosed with a memory-related disease 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 Individual Total Income

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Log Individual Total Income							
	Full Sample				Never Diagnosed			
AD Score	-0.037***	-0.027***	-0.015**	-0.012	-0.032***	-0.022**	-0.008	-0.005
	(0.008)	(0.008)	(0.008)	(0.008)	(0.009)	(0.009)	(0.008)	(0.008)
APOE (At least 1 copy)	-0.015	-0.007	-0.013	-0.003	-0.011	-0.004	-0.006	0.000
	(0.015)	(0.015)	(0.014)	(0.014)	(0.016)	(0.017)	(0.015)	(0.015)
APOE (2 copies)	0.002	-0.019	-0.012	0.001	0.020	-0.002	0.026	0.034
	(0.046)	(0.045)	(0.041)	(0.040)	(0.051)	(0.049)	(0.043)	(0.042)
Childhood SES Controls	No	Yes	Yes	Yes	No	Yes	Yes	Yes
Education Controls	No	No	Yes	Yes	No	No	Yes	Yes
TICS-M Score Dummies	No	No	No	Yes	No	No	No	Yes
Mean	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	73,999	73,999	73,999	73,999
R^2	0.186	0.206	0.268	0.276	0.184	0.204	0.269	0.276
Years	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

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 (6), we control for childhood socioeconomic status via an indicator for whether the respondent reports that their family was well off financially or about average from birth to age 16 (versus being poor) as well as dummy variables for their mother's years of education. In columns (3) and (7), we add controls for own educational attainment. In columns (4) and (8), we add dummy variables for each value of the most current TICS-M score. Columns (5)-(8) are estimated only on those never diagnosed with a memory-related disease 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

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Log Household Total Wealth							
	Full Sample				Never Diagnosed			
AD Score	-0.091***	-0.070***	-0.046***	-0.038**	-0.080***	-0.060***	-0.037**	-0.030*
	(0.017)	(0.017)	(0.016)	(0.016)	(0.018)	(0.018)	(0.017)	(0.017)
APOE (At least 1 copy)	-0.016	-0.009	-0.020	0.000	-0.013	-0.012	-0.018	-0.005
	(0.032)	(0.032)	(0.030)	(0.030)	(0.034)	(0.034)	(0.032)	(0.032)
APOE (2 copies)	0.058	-0.002	0.032	0.061	-0.022	-0.074	-0.002	0.018
	(0.107)	(0.109)	(0.098)	(0.097)	(0.120)	(0.121)	(0.110)	(0.108)
Childhood SES Controls	No	Yes	Yes	Yes	No	Yes	Yes	Yes
Education Controls	No	No	Yes	Yes	No	No	Yes	Yes
TICS-M Score Dummies	No	No	No	Yes	No	No	No	Yes
Mean	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	77,110	77,110	77,110	77,110
R^2	0.039	0.085	0.172	0.185	0.039	0.084	0.170	0.181
Years	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

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 (6), we control for childhood socioeconomic status via an indicator for whether the respondent reports that their family was well off financially or about average from birth to age 16 (versus being poor) as well as dummy variables for their mother's years of education. In columns (3) and (7), we add controls for own educational attainment. In columns (4) and (8), we add dummy variables for each value of the most current TICS-M score. Columns (5)-(8) are estimated only on those never diagnosed with a memory-related disease 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 Holding Long-Term Care Insurance

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Holds Long-Term Care Insurance							
	Full Sample				Never Diagnosed			
AD Score	-0.013***	-0.011***	-0.009**	-0.009**	-0.012***	-0.010***	-0.008**	-0.008**
	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)
APOE (At least 1 copy)	0.005	0.007	0.007	0.007	0.007	0.009	0.009	0.009
	(0.007)	(0.007)	(0.007)	(0.007)	(0.007)	(0.008)	(0.007)	(0.007)
APOE (2 copies)	-0.001	-0.010	-0.005	-0.004	-0.010	-0.017	-0.008	-0.008
	(0.020)	(0.020)	(0.020)	(0.020)	(0.021)	(0.021)	(0.020)	(0.020)
Childhood SES Controls	No	Yes	Yes	Yes	No	Yes	Yes	Yes
Education Controls	No	No	Yes	Yes	No	No	Yes	Yes
TICS-M Score Dummies	No	No	No	Yes	No	No	No	Yes
Mean	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	49,706	49,706	49,706	49,706
R^2	0.018	0.025	0.046	0.046	0.018	0.025	0.047	0.047
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	50-70	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 (6), we control for childhood socioeconomic status via an indicator for whether the respondent reports that their family was well off financially or about average from birth to age 16 (versus being poor) as well as dummy variables for their mother's years of education. In columns (3) and (7), we add controls for own educational attainment. In columns (4) and (8), we add dummy variables for each value of the most current TICS-M score. Columns (5)-(8) are estimated only on those never diagnosed with a memory-related disease 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 Life Insurance

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Holds Life Insurance							
	Full Sample				Never Diagnosed			
AD Score	-0.010**	-0.009*	-0.006	-0.005	-0.007	-0.006	-0.003	-0.003
	(0.005)	(0.005)	(0.005)	(0.005)	(0.005)	(0.005)	(0.005)	(0.005)
APOE (At least 1 copy)	-0.001	0.002	0.001	0.002	0.002	0.004	0.003	0.004
	(0.009)	(0.009)	(0.009)	(0.009)	(0.009)	(0.010)	(0.010)	(0.010)
APOE (2 copies)	-0.013	-0.017	-0.011	-0.008	-0.036	-0.045	-0.034	-0.032
	(0.026)	(0.027)	(0.027)	(0.027)	(0.028)	(0.029)	(0.029)	(0.028)
Childhood SES Controls	No	Yes	Yes	Yes	No	Yes	Yes	Yes
Education Controls	No	No	Yes	Yes	No	No	Yes	Yes
TICS-M Score Dummies	No	No	No	Yes	No	No	No	Yes
Mean	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	50,233	50,233	50,233	50,233
R^2	0.037	0.042	0.053	0.057	0.038	0.043	0.056	0.060
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	50-70	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 (6), we control for childhood socioeconomic status via an indicator for whether the respondent reports that their family was well off financially or about average from birth to age 16 (versus being poor) as well as dummy variables for their mother's years of education. In columns (3) and (7), we add controls for own educational attainment. In columns (4) and (8), we add dummy variables for each value of the most current TICS-M score. Columns (5)-(8) are estimated only on those never diagnosed with a memory-related disease 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 Having a Witnessed Will

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Has a Witnessed Will							
	Full Sample				Never Diagnosed			
AD Score	-0.024***	-0.017***	-0.012**	-0.011*	-0.022***	-0.015**	-0.010*	-0.009
	(0.006)	(0.006)	(0.006)	(0.006)	(0.006)	(0.006)	(0.006)	(0.006)
APOE (At least 1 copy)	0.013	0.012	0.010	0.011	0.015	0.013	0.010	0.011
	(0.011)	(0.011)	(0.011)	(0.011)	(0.011)	(0.012)	(0.011)	(0.011)
APOE (2 copies)	-0.013	-0.024	-0.010	-0.008	-0.025	-0.033	-0.010	-0.008
	(0.033)	(0.034)	(0.032)	(0.032)	(0.036)	(0.037)	(0.035)	(0.035)
Childhood SES	No	Yes	Yes	Yes	No	Yes	Yes	Yes
Education Controls	No	No	Yes	Yes	No	No	Yes	Yes
TICS-M Score Dummies	No	No	No	Yes	No	No	No	Yes
Mean	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	50,389	50,389	50,389	50,389
R^2	0.065	0.089	0.134	0.137	0.066	0.090	0.134	0.137
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	50-70	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 (6), we control for childhood socioeconomic status via an indicator for whether the respondent reports that their family was well off financially or about average from birth to age 16 (versus being poor) as well as dummy variables for their mother's years of education. In columns (3) and (7), we add controls for own educational attainment. In columns (4) and (8), we add dummy variables for each value of the most current TICS-M score. Columns (5)-(8) are estimated only on those never diagnosed with a memory-related disease 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 Living Will

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Has a Living Will (Advance Healthcare Directive)							
	Full Sample				Never Diagnosed			
AD Score	-0.023*	-0.020	-0.017	-0.016	-0.020	-0.016	-0.013	-0.013
	(0.012)	(0.012)	(0.012)	(0.012)	(0.012)	(0.013)	(0.012)	(0.012)
APOE (At least 1 copy)	0.032	0.024	0.025	0.024	0.040*	0.029	0.031	0.029
	(0.023)	(0.024)	(0.023)	(0.023)	(0.023)	(0.024)	(0.024)	(0.024)
APOE (2 copies)	0.045	0.066	0.088	0.086	0.030	0.056	0.086	0.086
	(0.066)	(0.067)	(0.063)	(0.063)	(0.069)	(0.070)	(0.067)	(0.067)
Childhood SES Controls	No	Yes	Yes	Yes	No	Yes	Yes	Yes
Education Controls	No	No	Yes	Yes	No	No	Yes	Yes
TICS-M Score Dummies	No	No	No	Yes	No	No	No	Yes
Mean	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	4,850	4,850	4,850	4,850
R^2	0.038	0.059	0.108	0.111	0.040	0.062	0.112	0.115
Years	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

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 (6), we control for childhood socioeconomic status via an indicator for whether the respondent reports that their family was well off financially or about average from birth to age 16 (versus being poor) as well as dummy variables for their mother's years of education. In columns (3) and (7), we add controls for own educational attainment. In columns (4) and (8), we add dummy variables for each value of the most current TICS-M score. Columns (5)-(8) are estimated only on those never diagnosed with a memory-related disease 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 Assigned Someone Durable Power of Attorney

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Has Assigned Someone Durable Power of Attorney for Healthcare							
	Full Sample				Never Diagnosed			
AD Score	-0.045***	-0.045***	-0.040***	-0.039***	-0.043***	-0.041***	-0.037***	-0.037***
	(0.012)	(0.012)	(0.012)	(0.012)	(0.012)	(0.012)	(0.012)	(0.012)
APOE (At least 1 copy)	0.014	0.007	0.010	0.009	0.016	0.008	0.011	0.009
	(0.022)	(0.023)	(0.023)	(0.023)	(0.023)	(0.024)	(0.023)	(0.023)
APOE (2 copies)	0.042	0.059	0.080	0.078	0.067	0.089	0.118*	0.118*
	(0.066)	(0.067)	(0.064)	(0.065)	(0.068)	(0.068)	(0.065)	(0.066)
Childhood SES Controls	No	Yes	Yes	Yes	No	Yes	Yes	Yes
Education Controls	No	No	Yes	Yes	No	No	Yes	Yes
TICS-M Score Dummies	No	No	No	Yes	No	No	No	Yes
Mean	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	4,852	4,852	4,852	4,852
R^2	0.035	0.055	0.105	0.108	0.036	0.057	0.108	0.112
Years	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

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 (6), we control for childhood socioeconomic status via an indicator for whether the respondent reports that their family was well off financially or about average from birth to age 16 (versus being poor) as well as dummy variables for their mother's years of education. In columns (3) and (7), we add controls for own educational attainment. In columns (4) and (8), we add dummy variables for each value of the most current TICS-M score. Columns (5)-(8) are estimated only on those never diagnosed with a memory-related disease 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 Discussed Future Medical Care with Someone

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Discussed Future Medical Care with Anyone							
	Full Sample				Never Diagnosed			
AD Score	-0.031***	-0.028**	-0.023**	-0.023**	-0.031***	-0.028**	-0.024**	-0.023**
	(0.011)	(0.011)	(0.011)	(0.011)	(0.011)	(0.011)	(0.011)	(0.011)
APOE (At least 1 copy)	-0.011	-0.012	-0.010	-0.009	-0.001	-0.004	-0.002	-0.001
	(0.020)	(0.020)	(0.020)	(0.020)	(0.020)	(0.021)	(0.021)	(0.021)
APOE (2 copies)	-0.032	-0.051	-0.042	-0.033	-0.030	-0.045	-0.030	-0.020
	(0.064)	(0.066)	(0.064)	(0.064)	(0.068)	(0.070)	(0.069)	(0.069)
Childhood SES Controls	No	Yes	Yes	Yes	No	Yes	Yes	Yes
Education Controls	No	No	Yes	Yes	No	No	Yes	Yes
TICS-M Score Dummies	No	No	No	Yes	No	No	No	Yes
Mean	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	3,347	3,347	3,347	3,347
R^2	0.071	0.084	0.124	0.132	0.073	0.087	0.127	0.133
Years	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

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 (6), we control for childhood socioeconomic status via an indicator for whether the respondent reports that their family was well off financially or about average from birth to age 16 (versus being poor) as well as dummy variables for their mother's years of education. In columns (3) and (7), we add controls for own educational attainment. In columns (4) and (8), we add dummy variables for each value of the most current TICS-M score. Columns (5)-(8) are estimated only on those never diagnosed with a memory-related disease 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 Expected Mortality

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Probability of Living to Age 75							
	Full Sample				Never Diagnosed			
AD Score	-0.350	0.107	0.333	0.384	-0.196	0.180	0.406	0.455
	(0.317)	(0.315)	(0.310)	(0.309)	(0.323)	(0.320)	(0.316)	(0.315)
APOE (At least 1 copy)	-0.749	-0.497	-0.579	-0.536	-0.807	-0.700	-0.787	-0.740
	(0.587)	(0.587)	(0.573)	(0.571)	(0.597)	(0.598)	(0.584)	(0.582)
APOE (2 copies)	-1.094	-2.550	-1.782	-1.687	-1.072	-2.636	-1.537	-1.526
	(1.910)	(1.949)	(1.937)	(1.927)	(2.070)	(2.109)	(2.087)	(2.076)
Childhood SES Controls	No	Yes	Yes	Yes	No	Yes	Yes	Yes
Education Controls	No	No	Yes	Yes	No	No	Yes	Yes
TICS-M Score Dummies	No	No	No	Yes	No	No	No	Yes
Mean	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	34,611	34,611	34,611	34,611
R^2	0.027	0.056	0.088	0.094	0.027	0.055	0.088	0.093
Years	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

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 younger than 65. 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 control for childhood socioeconomic status via an indicator for whether the respondent reports that their family was well off financially or about average from birth to age 16 (versus being poor) as well as dummy variables for their mother's years of education. In columns (3) and (7), we add controls for own educational attainment. In columns (4) and (8), we add dummy variables for each value of the most current TICS-M score. Columns (5)-(8) are estimated only on those never diagnosed with a memory-related disease 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 Nursing Home Use

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Probability of Moving to Nursing Home in Next 5 Years							
	Full Sample				Never Diagnosed			
AD Score	-0.055	-0.077	-0.059	-0.057	-0.085	-0.084	-0.075	-0.083
	(0.211)	(0.215)	(0.216)	(0.216)	(0.215)	(0.220)	(0.221)	(0.221)
APOE (At least 1 copy)	0.721*	0.584	0.572	0.541	0.809**	0.687*	0.656	0.649
	(0.390)	(0.399)	(0.400)	(0.398)	(0.407)	(0.413)	(0.414)	(0.414)
APOE (2 copies)	0.140	0.176	0.187	0.117	-1.830	-1.656	-1.663	-1.659
	(1.360)	(1.382)	(1.382)	(1.377)	(1.383)	(1.417)	(1.418)	(1.425)
Childhood SES Controls	No	Yes	Yes	Yes	No	Yes	Yes	Yes
Education Controls	No	No	Yes	Yes	No	No	Yes	Yes
TICS-M Score Dummies	No	No	No	Yes	No	No	No	Yes
Mean	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	15,426	15,426	15,426	15,426
R^2	0.014	0.022	0.026	0.030	0.015	0.022	0.026	0.029
Years	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

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 (6), we control for childhood socioeconomic status via an indicator for whether the respondent reports that their family was well off financially or about average from birth to age 16 (versus being poor) as well as dummy variables for their mother's years of education. In columns (3) and (7), we add controls for own educational attainment. In columns (4) and (8), we add dummy variables for each value of the most current TICS-M score. Columns (5)-(8) are estimated only on those never diagnosed with a memory-related disease 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 Probability of Living Independently at Age 75

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Probability of Living Independently at Age 75							
	Full Sample				Never Diagnosed			
AD Score	-0.322	-0.069	0.162	0.287	-0.285	-0.078	0.132	0.226
	(0.490)	(0.491)	(0.491)	(0.492)	(0.492)	(0.493)	(0.494)	(0.496)
APOE (At least 1 copy)	-1.570*	-1.853**	-2.095**	-1.985**	-1.733*	-2.062**	-2.332**	-2.185**
	(0.942)	(0.941)	(0.935)	(0.931)	(0.945)	(0.941)	(0.935)	(0.934)
APOE (2 copies)	0.678	-0.703	0.182	0.121	1.067	-0.376	0.721	0.531
	(2.505)	(2.515)	(2.549)	(2.561)	(2.663)	(2.663)	(2.682)	(2.699)
Childhood SES	No	Yes	Yes	Yes	No	Yes	Yes	Yes
Education Controls	No	No	Yes	Yes	No	No	Yes	Yes
TICS-M Score Dummies	No	No	No	Yes	No	No	No	Yes
Mean	69.999	69.999	69.999	69.999	70.451	70.451	70.451	70.451
N	4,178	4,178	4,178	4,178	4,039	4,039	4,039	4,039
R^2	0.045	0.092	0.130	0.143	0.045	0.092	0.131	0.139
Years	2006-2008	2006-2008	2006-2008	2006-2008	2006-2008	2006-2008	2006-2008	2006-2008
Ages	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 being able to live independently at age 75 (on a 0–100 scale). The question is only asked to those younger than 65 who do not report needing help with activities of daily living or instrumental activities of daily living. 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 control for childhood socioeconomic status via an indicator for whether the respondent reports that their family was well off financially or about average from birth to age 16 (versus being poor) as well as dummy variables for their mother's years of education. In columns (3) and (7), we add controls for own educational attainment. In columns (4) and (8), we add dummy variables for each value of the most current TICS-M score. Columns (5)-(8) are estimated only on those never diagnosed with a memory-related disease 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 Being Cognitively Healthy at Age 75

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Probability of Being Cognitively Healthy at Age 75							
	Full Sample				Never Diagnosed			
AD Score	-0.354	-0.024	0.265	0.375	-0.415	-0.112	0.173	0.257
	(0.510)	(0.513)	(0.513)	(0.513)	(0.517)	(0.518)	(0.519)	(0.520)
APOE (At least 1 copy)	-2.008**	-1.990**	-2.210**	-2.182**	-2.143**	-2.150**	-2.416***	-2.335**
	(0.946)	(0.940)	(0.923)	(0.917)	(0.948)	(0.939)	(0.922)	(0.918)
APOE (2 copies)	0.281	-0.314	0.826	0.881	0.972	0.186	1.529	1.475
	(2.831)	(2.831)	(2.813)	(2.753)	(3.007)	(3.004)	(2.970)	(2.921)
Childhood SES	No	Yes	Yes	Yes	No	Yes	Yes	Yes
Education Controls	No	No	Yes	Yes	No	No	Yes	Yes
TICS-M Score Dummies	No	No	No	Yes	No	No	No	Yes
Mean	65.779	65.779	65.779	65.779	66.298	66.298	66.298	66.298
N	4,168	4,168	4,168	4,168	4,031	4,031	4,031	4,031
R^2	0.046	0.091	0.136	0.151	0.044	0.090	0.138	0.147
Years	2006-2008	2006-2008	2006-2008	2006-2008	2006-2008	2006-2008	2006-2008	2006-2008
Ages	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 being free of serious problems in thinking, reasoning or remembering things that would interfere with the ability to manage their affairs at age 75 (on a 0–100 scale). The question is only asked to those younger than 65 who do not report needing help with activities of daily living or instrumental activities of daily living. 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 control for childhood socioeconomic status via an indicator for whether the respondent reports that their family was well off financially or about average from birth to age 16 (versus being poor) as well as dummy variables for their mother's years of education. In columns (3) and (7), we add controls for own educational attainment. In columns (4) and (8), we add dummy variables for each value of the most current TICS-M score. Columns (5)-(8) are estimated only on those never diagnosed with a memory-related disease 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 ADRD

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Probability of Developing ADRD							
	Full Sample				Never Diagnosed			
AD Score	-0.395	-1.183	-1.149	-1.162	-0.333	-1.094	-1.300	-1.285
	(1.504)	(1.575)	(1.617)	(1.703)	(1.559)	(1.632)	(1.664)	(1.755)
APOE (At least 1 copy)	6.949**	6.658**	7.059**	6.866**	6.448**	6.143**	6.714**	6.518**
	(2.694)	(2.789)	(2.733)	(2.756)	(2.776)	(2.905)	(2.858)	(2.877)
APOE (2 copies)	-6.562	-6.747	-9.061	-8.404	-5.927	-6.965	-10.023	-9.149
	(7.850)	(8.274)	(8.211)	(8.154)	(8.741)	(9.026)	(8.866)	(8.763)
Childhood SES Controls	No	Yes	Yes	Yes	No	Yes	Yes	Yes
Education Controls	No	No	Yes	Yes	No	No	Yes	Yes
TICS-M Score Dummies	No	No	No	Yes	No	No	No	Yes
Mean	36.303	36.303	36.303	36.303	36.105	36.105	36.105	36.105
N	660	660	660	660	638	638	638	638
R^2	0.222	0.259	0.334	0.373	0.214	0.250	0.331	0.373
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
Ages	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 (on a 0–100 scale). The sample excludes those diagnosed with memory-related disease (MRD) or currently residing 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 (6), we control for childhood socioeconomic status via an indicator for whether the respondent reports that their family was well off financially or about average from birth to age 16 (versus being poor) as well as dummy variables for their mother's years of education. In columns (3) and (7), we add controls for own educational attainment. In columns (4) and (8), we add dummy variables for each value of the most current TICS-M score. Columns (5)-(8) 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

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Probability of Parent Ever Diagnosed with MRD							
	Full Sample				Never Diagnosed			
AD Score	0.003	0.004*	0.005**	0.005**	0.004	0.004*	0.005**	0.005**
	(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***	0.023***	0.023***	0.021***	0.021***	0.021***	0.021***
	(0.005)	(0.005)	(0.005)	(0.005)	(0.005)	(0.005)	(0.005)	(0.005)
APOE (2 copies)	0.001	-0.002	-0.002	-0.001	0.010	0.007	0.006	0.007
	(0.014)	(0.014)	(0.014)	(0.014)	(0.016)	(0.016)	(0.016)	(0.016)
Childhood SES Controls	No	Yes	Yes	Yes	No	Yes	Yes	Yes
Education Controls	No	No	Yes	Yes	No	No	Yes	Yes
TICS-M Score Dummies	No	No	No	Yes	No	No	No	Yes
Mean	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	22,041	22,041	22,041	22,041
R^2	0.026	0.027	0.031	0.033	0.025	0.026	0.030	0.032
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	50-70	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 (6), we control for childhood socioeconomic status via an indicator for whether the respondent reports that their family was well off financially or about average from birth to age 16 (versus being poor) as well as dummy variables for their mother's years of education. In columns (3) and (7), we add controls for own educational attainment. In columns (4) and (8), we add dummy variables for each value of the most current TICS-M score. Columns (5)-(8) 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

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Probability of Parent Receiving Nursing Home Care							
	Full Sample				Never Diagnosed			
AD Score	0.000	0.003	0.005	0.004	0.003	0.006	0.007	0.005
	(0.009)	(0.010)	(0.010)	(0.010)	(0.010)	(0.010)	(0.010)	(0.010)
APOE (At least 1 copy)	0.028	0.026	0.026	0.027	0.028	0.025	0.026	0.026
	(0.017)	(0.018)	(0.018)	(0.018)	(0.018)	(0.018)	(0.019)	(0.019)
APOE (2 copies)	0.112**	0.108**	0.108**	0.116**	0.114**	0.113**	0.114**	0.118**
	(0.052)	(0.053)	(0.053)	(0.053)	(0.054)	(0.055)	(0.055)	(0.055)
Childhood SES Controls	No	Yes	Yes	Yes	No	Yes	Yes	Yes
Education Controls	No	No	Yes	Yes	No	No	Yes	Yes
TICS-M Score Dummies	No	No	No	Yes	No	No	No	Yes
Mean	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	4,200	4,200	4,200	4,200
R^2	0.061	0.066	0.080	0.089	0.061	0.066	0.080	0.090
Years	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

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 from the latest wave they are 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 (6), we control for childhood socioeconomic status via an indicator for whether the respondent reports that their family was well off financially or about average from birth to age 16 (versus being poor) as well as dummy variables for their mother's years of education. In columns (3) and (7), we add controls for own educational attainment. In columns (4) and (8), we add dummy variables for each value of the most current TICS-M score. Columns (5)-(8) are estimated only on those never diagnosed with a memory-related disease 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

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Appear in the Next Wave as Self-Respondent							
	Full Sample				Never Diagnosed			
	Panel A: Aged 50-85							
AD Score	-0.004***	-0.003**	-0.002**	-0.001	-0.004***	-0.003**	-0.002*	-0.001
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
APOE (At least 1 copy)	-0.009***	-0.007***	-0.007***	-0.003	-0.005**	-0.003	-0.003	-0.002
	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)
APOE (2 copies)	-0.010	-0.010	-0.010	-0.003	-0.006	-0.010	-0.009	-0.006
	(0.007)	(0.007)	(0.007)	(0.007)	(0.008)	(0.008)	(0.008)	(0.008)
Childhood SES Controls	No	Yes	Yes	Yes	No	Yes	Yes	Yes
Education Controls	No	No	Yes	Yes	No	No	Yes	Yes
TICS-M Score Dummies	No	No	No	Yes	No	No	No	Yes
Mean	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	74,852	74,852	74,852	74,852
R^2	0.051	0.053	0.056	0.077	0.044	0.046	0.049	0.058
Years	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
	Panel B: Aged 50-70							
AD Score	-0.004***	-0.003**	-0.003**	-0.003*	-0.004***	-0.003**	-0.003*	-0.002
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
APOE (At least 1 copy)	-0.003	-0.002	-0.002	-0.002	-0.003	-0.001	-0.002	-0.002
	(0.002)	(0.002)	(0.002)	(0.002)	(0.003)	(0.003)	(0.003)	(0.003)
APOE (2 copies)	-0.001	-0.003	-0.003	-0.002	-0.004	-0.007	-0.006	-0.005
	(0.008)	(0.008)	(0.008)	(0.008)	(0.009)	(0.009)	(0.009)	(0.009)
Childhood SES Controls	No	Yes	Yes	Yes	No	Yes	Yes	Yes
Education Controls	No	No	Yes	Yes	No	No	Yes	Yes
TICS-M Score Dummies	No	No	No	Yes	No	No	No	Yes
Mean	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	47,912	47,912	47,912	47,912
R^2	0.033	0.035	0.038	0.044	0.033	0.034	0.037	0.041
Years	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

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 (6), we control for childhood socioeconomic status via an indicator for whether the respondent reports that their family was well off financially or about average from birth to age 16 (versus being poor) as well as dummy variables for their mother's years of education. In columns (3) and (7), we add controls for own educational attainment. In columns (4) and (8), we add dummy variables for each value of the most current TICS-M score. Columns (5)-(8) are estimated only on those never diagnosed with a memory-related disease 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

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Proxy Response in the Next Wave							
	Full Sample				Never Diagnosed			
	Panel A: Aged 50-85							
AD Score	0.000	-0.000	-0.000	-0.001	-0.000	-0.000	-0.000	-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***	0.001	0.001	0.001	0.000
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
APOE (2 copies)	0.009**	0.007*	0.007*	0.004	0.003	0.003	0.003	0.003
	(0.004)	(0.004)	(0.004)	(0.004)	(0.003)	(0.004)	(0.004)	(0.004)
Childhood SES Controls	No	Yes	Yes	Yes	No	Yes	Yes	Yes
Education Controls	No	No	Yes	Yes	No	No	Yes	Yes
TICS-M Score Dummies	No	No	No	Yes	No	No	No	Yes
Mean	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	70,604	70,604	70,604	70,604
R^2	0.015	0.016	0.020	0.062	0.008	0.008	0.012	0.016
Years	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
	Panel B: Aged 50-70							
AD Score	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)	(0.001)
APOE (At least 1 copy)	0.001	0.001	0.001	0.001	-0.000	0.000	0.000	0.000
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
APOE (2 copies)	0.002	0.003	0.002	0.002	0.003	0.003	0.003	0.003
	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)
Childhood SES Controls	No	Yes	Yes	Yes	No	Yes	Yes	Yes
Education Controls	No	No	Yes	Yes	No	No	Yes	Yes
TICS-M Score Dummies	No	No	No	Yes	No	No	No	Yes
Mean	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	45,821	45,821	45,821	45,821
R^2	0.008	0.009	0.014	0.026	0.009	0.010	0.014	0.017
Years	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

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 (6), we control for childhood socioeconomic status via an indicator for whether the respondent reports that their family was well off financially or about average from birth to age 16 (versus being poor) as well as dummy variables for their mother's years of education. In columns (3) and (7), we add controls for own educational attainment. In columns (4) and (8), we add dummy variables for each value of the most current TICS-M score. Columns (5)-(8) are estimated only on those never diagnosed with a memory-related disease 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

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Observed Mortality							
	Full Sample				Never Diagnosed			
	Panel A: Aged 50-85							
AD Score	0.002	0.001	0.001	-0.000	0.002*	0.001	0.001	0.000
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
APOE (At least 1 copy)	0.001	-0.000	-0.000	-0.004	0.002	0.001	0.001	-0.001
	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)
APOE (2 copies)	0.005	0.009	0.007	0.003	0.011	0.014*	0.012	0.008
	(0.007)	(0.007)	(0.007)	(0.007)	(0.008)	(0.008)	(0.008)	(0.008)
Childhood SES Controls	No	Yes	Yes	Yes	No	Yes	Yes	Yes
Education Controls	No	No	Yes	Yes	No	No	Yes	Yes
TICS-M Score Dummies	No	No	No	Yes	No	No	No	Yes
Mean	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	41,171	41,171	41,171	41,171
R^2	0.032	0.034	0.039	0.050	0.034	0.036	0.041	0.054
Years	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
	Panel B: Aged 50-70							
AD Score	0.001	0.001	0.001	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)
APOE (At least 1 copy)	-0.002	-0.003	-0.003	-0.003	-0.002	-0.002	-0.002	-0.003
	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)
APOE (2 copies)	0.006	0.009	0.007	0.005	0.010	0.013*	0.011	0.009
	(0.007)	(0.007)	(0.007)	(0.007)	(0.008)	(0.008)	(0.008)	(0.008)
Childhood SES Controls	No	Yes	Yes	Yes	No	Yes	Yes	Yes
Education Controls	No	No	Yes	Yes	No	No	Yes	Yes
TICS-M Score Dummies	No	No	No	Yes	No	No	No	Yes
Mean	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	23,339	23,339	23,339	23,339
R^2	0.010	0.014	0.021	0.031	0.011	0.015	0.023	0.034
Years	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

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 (6), we control for childhood socioeconomic status via an indicator for whether the respondent reports that their family was well off financially or about average from birth to age 16 (versus being poor) as well as dummy variables for their mother's years of education. In columns (3) and (7), we add controls for own educational attainment. In columns (4) and (8), we add dummy variables for each value of the most current TICS-M score. Columns (5)-(8) are estimated only on those never diagnosed with a memory-related disease 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

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Currently Working for Pay		Retirement		Log Individual Total Income		Log Household Total Wealth	
AD Score	-0.010**	-0.009*	0.007	0.007	-0.012	-0.002	-0.038**	-0.032*
	(0.005)	(0.005)	(0.004)	(0.005)	(0.008)	(0.009)	(0.016)	(0.017)
APOE (At least 1 copy)	0.010	0.009	-0.011	-0.011	-0.003	0.002	0.000	-0.010
	(0.009)	(0.010)	(0.008)	(0.009)	(0.014)	(0.016)	(0.030)	(0.033)
APOE (2 copies)	-0.019	-0.020	0.043*	0.040	0.001	-0.028	0.061	-0.001
	(0.027)	(0.031)	(0.025)	(0.027)	(0.040)	(0.048)	(0.097)	(0.118)
Childhood SES Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Education Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
TICS-M Score Dummies	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	0.186	0.181	0.223	0.223	0.276	0.268	0.185	0.176
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 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 an indicator for whether the respondent reports that their family was well off financially or about average from birth to age 16 (versus being poor), dummy variables for their mother's years of education, dummy variables for own 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	-0.009**	-0.008**	-0.005	-0.006	-0.011*	-0.011*
	(0.004)	(0.004)	(0.005)	(0.005)	(0.006)	(0.006)
APOE (At least 1 copy)	0.007	0.005	0.002	0.003	0.011	0.012
	(0.007)	(0.008)	(0.009)	(0.010)	(0.011)	(0.012)
APOE (2 copies)	-0.004	0.001	-0.008	-0.028	-0.008	-0.015
	(0.020)	(0.022)	(0.027)	(0.031)	(0.032)	(0.037)
Childhood SES Controls	Yes	Yes	Yes	Yes	Yes	Yes
Education Controls	Yes	Yes	Yes	Yes	Yes	Yes
TICS-M Score Dummies	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	0.046	0.046	0.057	0.058	0.137	0.135
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	-0.016	-0.023*	-0.039***	-0.042***	-0.023**	-0.030***
	(0.012)	(0.013)	(0.012)	(0.013)	(0.011)	(0.011)
APOE (At least 1 copy)	0.024	0.016	0.009	-0.002	-0.009	-0.014
	(0.023)	(0.025)	(0.023)	(0.024)	(0.020)	(0.021)
APOE (2 copies)	0.086	0.098	0.078	0.089	-0.033	-0.006
	(0.063)	(0.067)	(0.065)	(0.068)	(0.064)	(0.068)
Childhood SES	Yes	Yes	Yes	Yes	Yes	Yes
Education Controls	Yes	Yes	Yes	Yes	Yes	Yes
TICS-M Score Dummies	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	0.111	0.111	0.108	0.109	0.132	0.134
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 an indicator for whether the respondent reports that their family was well off financially or about average from birth to age 16 (versus being poor), dummy variables for their mother's years of education, dummy variables for own 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 A26: Relationship between Genetic Endowments and Awareness of Risk Among the Never Demented

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)
	Probability of Living to Age 75		Probability of Moving to Nursing Home in Next 5 Years		Probability of Living Independently At Age 75		Probability of Cognitively Healthy At Age 75		Probability of Developing ADRD		Parental Diagnosis of MRD		Parental Use of Nursing Home Care	
AD Score	0.384	0.345	-0.057	-0.072	0.287	0.221	0.375	0.258	-1.162	-0.877	0.005**	0.005*	0.004	0.004
	(0.309)	(0.328)	(0.216)	(0.233)	(0.492)	(0.514)	(0.513)	(0.536)	(1.703)	(1.818)	(0.002)	(0.003)	(0.010)	(0.010)
APOE (At least 1 copy)	-0.536	-0.711	0.541	0.343	-1.985**	-2.235**	-2.182**	-2.184**	6.866**	6.997**	0.023***	0.023***	0.027	0.023
	(0.571)	(0.607)	(0.398)	(0.436)	(0.931)	(0.976)	(0.917)	(0.969)	(2.756)	(3.038)	(0.005)	(0.005)	(0.018)	(0.019)
APOE (2 copies)	-1.687	-1.920	0.117	0.862	0.121	1.280	0.881	2.233	-8.404	-9.260	-0.001	-0.010	0.116**	0.093*
	(1.927)	(2.148)	(1.377)	(1.667)	(2.561)	(2.623)	(2.753)	(2.980)	(8.154)	(8.380)	(0.014)	(0.014)	(0.053)	(0.056)
Childhood SES	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
TICS-M Score Dummies	Yes	Yes	Yes	Yes	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	All	Never Demented	All	Never Demented
Mean	66.275	66.534	10.982	11.037	69.999	70.508	65.779	66.378	36.303	36.203	0.070	0.070	0.379	0.379
N	36,282	32,790	16,795	14,014	4,178	3,774	4,168	3,766	660	602	23,065	20,723	4,344	3,925
R^2	0.094	0.098	0.030	0.030	0.143	0.145	0.151	0.152	0.373	0.384	0.033	0.032	0.089	0.091
Years	1998-2018	1998-2018	1998-2018	1998-2018	2006-2008	2006-2008	2006-2008	2006-2008	2002, 2012, 2016	2002, 2012, 2016	1998-2018	1998-2018	2006-2018	2006-2018
Ages	50-65	50-65	65-70	65-70	50-65	50-65	50-65	50-65	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), (10), (12), and (14) 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 an indicator for whether the respondent reports that their family was well off financially or about average from birth to age 16 (versus being poor), dummy variables for their mother's years of education, dummy variables for own 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 $p < 0.01$.