Genetic Predictors of Cognitive Decline and Labor Market Exit*

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Abstract

We analyze administrative and genetic data from over 200,000 Danes to study the effects of genetic risk for Alzheimer's Disease (AD) on labor market outcomes. Higher AD genetic risk increases dementia diagnoses and GP visits for both genders. Among women aged 45–65, it reduces labor participation and raises disability pension uptake, especially near retirement. These effects weaken for women with high polygenic scores for education. For men, AD genetic risk shows no employment impact. These gender differences align with evidence that AD genetic markers are more predictive in women.

Keywords: Alzheimer's Disease, labor supply, genoeconomics

JEL classification: I14, J14, J22

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1 Introduction

Alzheimer's Disease (AD) is a neurodegenerative disease and the most common form of dementia. Currently, the Alzheimer's Association (2024) estimates that seven million Americans are living with AD. This number is projected to double by 2050 due to population aging. Because of its prevalence, it is important to understand the costs of AD for both individuals and for society. At the individual level, AD is the fifth-leading cause of death in the United States. Its symptoms include: memory loss, cognitive difficulties, and personality changes (Alzheimer's Association, 2024). In the year 2024 alone, AD is estimated to cost the US 360 billion USD in health and long-term care expenditures. This is projected to increase to one trillion USD by 2050 (Alzheimer's Association, 2024).

In this paper, we investigate the relationship between genetic predisposition for Alzheimer's Disease and economic outcomes. We primarily focus on labor market outcomes but we also consider wealth accumulation. We concentrate on people who are close to but below the state pension retirement age. This is a point in the life-cycle at which the early symptoms of AD may be present but not yet salient. Doing this is important because the costs of AD might be underestimated if there are important effects of AD on critical economic outcomes at relative early stages in life that typically are not considered in standard cost calculations.

There are several reasons why higher genetic risk of AD would impact labor market outcomes. First, AD could cause cognitive decline before retirement, reducing an individual's work capacity. While it is true that few individuals are diagnosed with AD prior to the retirement age, research studies show that individuals experience some degree of cognitive decline prior to retirement (Chandra et al., 2023). For example, one out of nine Americans aged 45-64 reported subjective cognitive decline - one of the earliest noticeable symptoms of AD or other types of dementia (Centers for Disease Control and Prevention, 2019). Second, individuals with increased genetic risk of AD have a higher probability of having family members with AD. Hence, the individual might need to provide informal care to their affected family members, which could decrease their labor supply. In fact, Maestas et al. (2024) find that care-giving significantly reduces employment and earnings for women. Third, an individual might have private knowledge about their increased genetic risk of AD by having observed family members with the disease or from genetic testing. This knowledge might cause an individual to retire earlier to have time for leisure prior to the onset of more advanced stages of AD.

The economic literature on Alzheimer's Disease is relatively new but growing. We refer readers to Chandra et al. (2023) for an excellent review of the literature. The authors of that review highlight

that the relationship between AD and labor supply is relatively unexplored and, therefore, represents an important avenue for future economic research. Our contribution to this literature is that we quantify the effects of AD in its earliest stages, well before retirement, on critical labor market outcomes. We accomplish this by employing genetic markers that have been shown to be highly predictive of AD. Since these markers are immutable individual characteristics, we can use them to identify individuals, while they are still active in the labor market.

There are a few papers closely related to our own, albeit with important differences. First, Jeong et al. (2024) investigate the association between genetic measures of Alzheimer's Disease and related dementias (ADRD) and cognition, economic outcomes, and planning activities for individuals over the age of 50, using the Health and Retirement Study (HRS). Notably, they find that genetic measures of ADRD meaningfully predict cognition and the probability of being diagnosed with a memory-related disease. They also find that increased genetic risk of ADRD is associated with a lower probability of working for pay. While they do not study gender differences in the relationship between genetic endowments and labor supply, they do study this for the link with wealth accumulation; they find that females drive a negative association between genetic risk of AD and household wealth. The primary focus of their work is financial decision-making. The authors show that individuals with higher genetic risk are less likely to engage in planning activities. Next, Shin et al. (2020) study how genetic risk of AD relates to saving behavior, also using the HRS. They find an association between the genetic risk of AD and the composition of wealth holdings.

While we will discuss our findings in greater detail momentarily, we would like to highlight some key differences between our work and these previous studies. First, we focus on a substantially younger sample. For instance, the mean age in Jeong et al. (2024) is 68, whereas our mean age is 52 for women and 53 for men. This allows us to investigate the labor market consequences of AD at a much earlier stage than has previously been examined. Second, because we have access to high-quality administrative data with large sample sizes, we are able to carefully examine gender differences. This is important, as it is well established that certain genetic markers of AD exhibit greater penetrance in women than in men. Third, our focus is on labor supply, whereas Jeong et al. (2024) and Shin et al. (2020) concentrate on financial decision-making. All told, all three papers are highly complementary but retain important differences.

In this work, we employ Danish administrative data combined with iPSYCH genetic data. The iPSYCH project is one of the world's largest studies of the genetic and environmental causes of mental disorders. It contains genetic data on over 140,000 Danes, which we can merge with full population

administrative data. The individuals in the iPSYCH dataset are too young to be studied close to the retirement age. Therefore, we use a proxy-phenotype design to examine the relationship between children's genetic variants associated with AD and parents' labor market outcomes. Since this introduces measurement error that biases our results downwards, our findings should be interpreted as lower bounds of the true effects. We construct two measures of genetic risk of AD: (1) an indicator for the child's *APOE-e4* carrier status, and (2) the child's AD polygenic score (PGS). Crucially, we study genetic risk of AD rather than an AD diagnosis, as diagnosis is an endogenous outcome reflecting the decision to seek medical care. This decision may vary by socioeconomic status, gender, and other factors. Moreover, AD is often under-diagnosed (Alzheimer's Association, 2024). Specifically, we analyze the associations between genetic risk of AD and labor market outcomes separately for men and women, while controlling for a wide range of characteristics such as year fixed effects, age fixed effects, education, labor market experience, and genetic stratification.

We show that our genetic measure of AD predicts health and medical utilization. For women aged 45 to 65, having a child with *APOE-e4* carrier status increases the risk of a dementia diagnosis by 91%. For men, the risk of a dementia diagnosis increases by 44%. For both men and women, we find that having a child with carrier status has a statistically significant, positive association with GP visits. Likewise, we find that the AD PGS also positively predicts dementia diagnosis for men and women.

Next, we study the association between genetic predisposition for AD and labor market decline close to the retirement age. For women, we find that an increased genetic risk of AD is associated with lower employment and higher take-up of disability pensions. We find no robust relationship with receipt of unemployment benefits, other transfers, or pensions (excluding the disability pension). Women aged 45 to 65 with an *APOE-e4* carrier child have a lower probability of being employed of 0.3 percentage points and an increased probability of receiving disability pension of 0.4 percentage points compared to women with a non-carrier child. For women aged 55 to 65, having a carrier-child is associated with a 0.7 percentage point decrease in employment and a 0.8 percentage point increase in the likelihood of receiving disability pension. Using the AD PGS, we find similar results for women aged 55 to 65. Generally, our results show that the associations between labor market outcomes and genetic risk for AD are stronger closer to the state pension retirement age.

We attribute a significant portion of the negative association between genetic risk of AD and lower employment probability for women to early cognitive decline occurring before the state pension retirement age. This is due to the fact that disability pension is only granted when an individual's capacity to work is permanently reduced, and this is determined through a health assessment by the municipality.

Therefore, the take-up of disability pension, instead of employment, reflects that women with higher genetic risk for AD leave the labor market before reaching the retirement age due to health-related reasons. Combined with evidence of a positive link between genetic risk for AD, diagnosed dementia, and increased GP visits, this suggests that cognitive decline related to AD helps explain the negative relationship between genetic risk and employment for women.

We also investigate how genetic risk of AD relates to different income and wealth measures. We do not find a statistically significant relationship between the genetic measures of AD and our income and wealth measures for women. However, as we use a proxy-phenotype design, we again caution that our results are biased downwards.

We also find that while genetic risk for AD predicts a number of important health outcomes for men, we do not find statistically significant associations with any labor market outcomes. This suggests important heterogeneity across genders. Importantly, medical research has shown that the *APOE-e4* gene is a stronger predictor of AD in women than in men (Altmann et al., 2014). However, it is also possible that behavioral differences in response to higher genetic risks of AD vary by gender. Furthermore, men and women often work in different occupations (Cortes and Pan, 2018), which could influence the ability of individuals experiencing cognitive decline to remain employed. Additionally, women are more likely than men to utilize health services (Bertakis et al., 2000), which may also impact employment outcomes and disability pension take-up.

Finally, we explore the possibility that a genetic predisposition for higher educational attainment may mitigate the genetic risk for AD. To examine this, we incorporate the polygenic score for educational attainment (EA) and interact it with genetic markers for AD risk in our estimations. For women aged 55 to 65, a higher EA PGS appears to buffer the impact of AD genetic risk on disability pension receipt, aligning with the "cognitive reserve theory"—the notion that some individuals can tolerate greater brain changes without clinical impairment (Stern, 2012). Notably, including the EA PGS does not alter the coefficient for carrier status or the AD PGS. For both men and women, we find a strong positive association between the EA PGS and employment, and a negative association between the EA PGS and disability pension receipt, with these relationships becoming more pronounced with age.

All told, we make two key contributions to this emerging literature. First, we show that the costs of Alzheimer's Disease begin to manifest well before its more advanced stages, expanding the existing literature on the economic burden of AD (see, e.g., Meijer et al. (2022), Nandi et al. (2024)). While previous studies have primarily focused on the direct costs of healthcare and long-term care, or the indirect costs of unpaid caregiving by friends or family, we demonstrate that these costs are broader.

Specifically, they include reduced labor supply and increased government transfers during middle age. Second, we reveal significant gender differences in how genetic risk for AD is associated with labor market outcomes. While genetic risk for AD affects women's labor supply on the extensive margin, it does not appear to impact men's labor supply. These findings are particularly important as they contribute to our understanding of the early economic costs of AD. With rising labor force participation among older adults due to retirement reforms in many countries, understanding the costs of AD prior to the retirement age is becoming increasingly crucial.

For the balance of the paper, we proceed as follows. In Section 2, we provide some background information on Alzheimer's Disease and Danish institutions. In Section 3, we describe the data. In Section 4, we discuss the methods that we employ. In Section 5, we discuss our findings. Finally, in Section 6, we conclude.

2 Background

2.1 Some Basics on Alzheimer's Disease

Alzheimer's Disease (AD) is a progressive neurological disorder. It is the most common form of dementia accounting for 60-80% of all dementia diagnoses and is the fifth-leading cause of death in Americans aged 65 and older (Alzheimer's Association, 2024). AD is linked to a build-up of beta-amyloid (Aβ), a protein fragment, which has been shown (both in vitro and in vivo) to damage neurons in the brain adversely impacting memory, language, and cognition.[1] AD is progressive and so, over time, these symptoms worsen. While AD cannot be cured, there are some treatments that can alleviate some of its symptoms.[2]

There is a continuum of phases in AD progression including: (1) Preclinical AD; (2) Mild Cognitive Impairment (MDI); (3) Mild AD; (4) Moderate AD; and (5) Severe AD. When AD is in its preclinical phase, there are no symptoms. However, there are possible biological changes in the brain. AD typically begins more than 20 years prior to the manifestation of symptoms such as memory loss. Next, during the MDI stage, patients experience very mild symptoms that may not interfere with everyday activities. People typically are not diagnosed either in the MDI phase or in the preclinical phase. In the mild stage of AD, individuals often are still able to function independently and work. However, they might require assistance to perform certain tasks such as financial decision-making. In the moderate phase of AD,

[1] Even though its pathogenetic role in disease development may be complex, the Aβ hypothesis is still the dominant paradigm in the development of AD (Jagust et al., 2023).

[2] Cholinesterase inhibitors can reduce cognitive and behavioral symptoms in people with mild to severe AD. Memantine is often prescribed to individuals with moderate to severe AD (National Insitute on Ageing, 2023).

symptoms start to interfere with a larger number of everyday activities. In addition, individuals in this phase experience difficulty recognizing family and friends. They also experience behavioral changes. Finally, in the most severe phase of AD, individuals need assistance with self-care including bathing, eating, and taking medications. Advanced AD patients also have difficulty communicating with the most advanced patients often being non-verbal.

AD has a number of risk factors that are both non-modifiable and modifiable. The three primary non-modifiable risk factors for developing AD are age, genetics, and sex. Of these, the most predictive is age. For example, while 5% of Americans aged 65-74 have AD, 33% of Americans aged 85 or older have AD. There are also a number of other modifiable risk factors. These include poor cardiovascular health, low education, a history of brain injuries, an accumulation of lack of sleep, hearing loss, social isolation, and chronic exposure to air pollution (Alzheimer's Association, 2024).

2.2 Genetic Determinants of Alzheimer's Disease

There are two primary ways in which we assess genetic risk. The first is to identify individuals with specific genes that are known to be associated with AD. The second is to construct scores based on a large number of small genetic variants that are known to predict the onset of AD. These scores are called polygenic scores (PGS).

First, we focus on the *APOE-e4* allele. This is a variant of the APOE gene which has three common alleles: *APOE-e2*, *APOE-e3*, and *APOE-e4*. Possessing the *APOE-e4* allele greatly increases the risk of the onset of AD (Corder et al., 1993, Strittmatter et al., 1993). Compared to the other two alleles, *APOE-e4* is less efficient at clearing lipids from the bloodstream. People with this allele also have a more difficult time maintaining and repairing neurons. As a consequence, carriers of the *APOE-e4* allele are more likely to experience an accumulation of beta-amyloid plaques in their brains, as this particular allele is less efficient at promoting the clearance of these protein fragments.

People can either be homogeneous, heterogeneous, or non-carriers of the *APOE-e4* allele. Each child inherits one *APOE* gene from their mother and one from their father yielding a total of six possible combinations for the APOE gene: *APOE-e2*/*APOE-e2*, *APOE-e3*/*APOE-e3*, *APOE-e4*/*APOE-e4*, *APOE-e2*/*APOE-e3*, *APOE-e2*/*APOE-e4*, and *APOE-e3*/*APOE-e4*. Individuals who have the *APOE-e4*/*APOE-e4*-combination are called homogeneous carriers. They constitute approximately 3% of the Danish population. People who have either the *APOE-e2*/*APOE-e4*- or *APOE-e3*/*APOE-e4* -combination are called heterogeneous carriers. They constitute approximately 28% of the Danish population (Rasmussen et al., 2018). Heterogeneous carriers have a 4-fold increased risk of developing AD, whereas homogeneous

carriers have a 12-fold increased risk of developing AD (Spinney, 2014). While the genoeconomics literature usually advises against using a single gene when attempting to explain economic behaviors, the *APOE* gene is typically seen as an exception due to its high (albeit incomplete) penetrance (Benjamin et al., 2012).

Second, we use a PGS for AD as a proxy for AD risk. Polygenic scores (also sometimes called polygenic risk scores or polygenic indexes) summarizes the cumulative effect of many small genetic variants, known as single nucleotide polymorphisms (SNPs), on a particular phenotype (e.g. height, IQ, heart disease, diabetes, AD, etc.) (Benjamin et al., 2024). To compute a PGS, researchers determine which SNPs are most strongly associated with the phenotype in question. The PGS is then computed by summing the number of risk alleles that an individual possesses weighted by their respective effect size. The effect sizes are typically obtained from Genome-Wide Association Studies (GWAS). The PGS for AD is computed as any other score would be while using AD as the phenotype to be predicted.

2.3 Institutional Setting

In Denmark, the majority of healthcare services are provided free of charge. They are financed by general taxes.[3] Particularly, general practitioner (GP) visits are free of charge. GPs act as "gatekeepers" between primary care and specialized care as they refer patients to specialists or hospital admissions. Accordingly, a diagnosis of dementia first necessitates a visit to a GP, who then refers the individual to a structured assessment of cognitive functioning at a dementia or memory clinic if they show symptoms of dementia (DaneAge Association, 2024).

The Danish government partly subsidies prescription drug expenses above certain thresholds for most pharmaceuticals. If expenditures exceed 1075 DKK (≈151 USD)[4], reimbursements are 50% of the total cost. However, reimbursements rates increase with expenditures on pharmaceuticals. For drug expenses above 21,298 DKK (≈2982 USD), the reimbursement rate is 100%. Hence, the annual maximum out-of-pocket for an individual is 4575 DKK (≈641 USD) (Danish Medicines Agency, 2024).

While Denmark is a generous welfare state, it also has very flexible labor markets. This system of flexible hiring and firing rules, a generous social safety net, and active labor market programs is known as 'flexicurity' (Andersen and Svarer, 2007). As a consequence, Denmark has a high employment rate for both men and women. In addition, Denmark is known for its family-friendly policies, which enable women to have a high labor force participation (Datta Gupta et al., 2008).

[3] See also Danish Ministry of Health (2017) for a more detailed description of the Danish healthcare system.
[4] 1 DKK = 0.14 USD

There are multiple layers to the Danish safety net. First, individuals who are not employed are eligible for unemployment benefits provided that they are members of an unemployment insurance (UI) fund for at least one year, are actively seeking work, and have received income above a certain threshold for the last three years. Next, people who are not able to work due to health-related reasons are eligible for a disability pension (DP). To qualify for DP, the applicant must undergo a health assessment and participate in a rehabilitation plan. Finally, if an individual is not eligible for unemployment benefits or any other public transfers such as an age-dependent state pension (SP), they can qualify for means-tested cash benefits.

The retirement age in Denmark is currently 67 years old. At this age, Danes qualify for SP. The retirement age was gradually increased from 65 years old in 2019 as a consequence of the 2011 Retirement Reform. This law also gradually increased the early retirement age. Currently, Danes can retire early and receive Voluntary Early Retirement Pay (VERP, in Danish *efterløn*) if they have paid into the early retirement scheme for at least 30 years. The early retirement age was 64 years old in 2024. The 2011 reform gradually increased the early retirement age from 60 years in 2011.

3 Data

We employ Danish administrative data on labor market outcomes coupled with detailed genetic information from the 2015 iPSYCH study. The iPSYCH study is one of the world's largest studies of the genetic and environmental causes of mental disorders containing genetic data on over 140,000 Danes. We match the genetic data to full population Danish registers containing information on labor market outcomes, education, and key demographic variables. Attrition from the data is minimal as individuals leave the sample only if they die or move out of the country.

An important feature of the sample that we ultimately used is that the labor market information comes from a sample of older people, while the genetic data comes from their children. This choice was necessary given the data constraints that we faced. We used older people aged 45-65 because we required individuals close to retirement age. However, the genetic data we employed came from their children because the iPSYCH sample from which we obtained the genetic information included people born no earlier than 1981. Accordingly, we could not use labor market outcomes such as retirement behavior as the individuals in the genetic sample are currently too young. As some individuals have multiple children in the iPSYCH sample, we use genetic information on the first-born child for which there exists genetic data.

3.1 Genetic Data

The population for the 2015 iPSYCH sample is nested within all singleton births between May 1, 1981 to December 31, 2008 corresponding to a total of 1,657,449 individuals.[5] From this population, the iPSYCH 2015 study included 93,608 individuals who were diagnosed with schizophrenia, bipolar disorder, affective disorder, autism, or ADHD in the period 1994-2015. In addition, iPSYCH contains a control group consisting of 48,227 individuals chosen at random from the sample frame. In total, the 2015 iPSYCH sample contains 141,835 people. Because we will require labor market outcomes for people near retirement, our defined sample frame only includes children who could be matched to parents in the relevant age range in the registers.

For reasons that we have already discussed, we use parent outcomes and child genetic information in a proxy-phenotype design. The use of children's genetic information introduces measurement error in our main explanatory variable. This biases our estimates. We show in the Appendix that this bias is of the typical attenuation variation when APOE carrier status is the explanatory variable. We also provide a rule-of-thumb for correcting this bias.

The first measure of genetic predisposition for AD that we employ is an indicator for *APOE-e4* carrier status. We construct an indicator variable for *APOE-e4* carrier status that takes the value of zero if the child is a non-carrier and one if the child is either a homogeneous or a heterogeneous carrier. We combine heterogeneous and homogeneous carrier status to increase power. We use the Single Nucleotide Polymorphisms (SNPs) *'rs7412'* and *'rs429358'* to determine if the person carries the *APOE-e4* allele.

The second measures of genetic predisposition for AD that we employ are PGSs for AD. In general, there is a wide range of PGSs available for AD in the iPSYCH data. These are shown in Table 1. We employ 'phen19' as our primary PGS for AD as it was created using a sample of more than 450,000 individuals. This relatively large sample size should mitigate the effects of measurement error. However, we also use the other AD PGSs for robustness checks to ensure the validity of our results. In addition, to study whether educational attainment (EA) has a moderating effect on AD, we also use an EA PGS described in Table 1. Finally, we standardize all PGSs to have a mean equal to zero and SD equal to one to ease interpretation.[6]

[5] See also Pedersen et al. (2018) and Bybjerg-Grauholm et al. (2020) for a more detailed description of the iPSYCH study.

[6] The distribution of the AD PGS and EA PGS can be seen in Table A.2. We also show the correlation between our primary AD PGS and all possible AD PGSs in Figure A.3.

Table 1: Polygenic Scores

PGS	Description	*N*	GWAS
Panel A: Alzheimer's Disease			
phen19	Alzheimer's Disease (AD)	455,258	Jansen et al. (2019)
gcst007511	Late-onset AD	63,926	Kunkle et al. (2019)
gcst005921	Family history of AD	314,278	Marioni et al. (2018)
gcst005923	Maternal history of AD	288,676	Marioni et al. (2018)
gcst005920	Paternal history of AD	260,279	Marioni et al. (2018)
gcst007320	AD/family history of AD	455,258	Jansen et al. (2019)
ga3635	Illness of father: AD	355,137	Watanabe et al. (2019)
ga3646	Illness of mother: AD	367,939	Watanabe et al. (2019)
Panel B: Educational Attainment			
phen62	Educational Attainment (EA)	766,345	Lee et al. (2018)

Notes: This table describes the polygenic scores available in the iPSYCH sample for Alzheimer's Disease and educational attainment. *N* refers to the number of observations used in the Genome-Wide Association Studies (GWAS).

3.2 Health Outcomes

To investigate if our genetic measures of AD predict health, we construct two outcomes related to AD. First, we create a dummy for hospital contact related to dementia using the following ICD10 codes: dementia in Alzheimer's disease (F00); vascular dementia (F01); dementia in other diseases classified elsewhere (F02); unspecified dementia (F03); and Alzheimer's disease (G30). For that, we use data on hospital contacts from The National Patient Register (LPR) for the years 2005-2020. We also employ data on GP visits from The Health Insurance Registry (SSSY) for the years 2005-2020. We construct the GP visits measure as in Nielsen (2019). We only include in-person visits to the GP and exclude email or telephone consultations.

3.3 Economic Outcomes

We employ the following economic outcomes. We start by studying labor market attachment where we create five categories that partition the total population using The Integrated Database for Labour Market Research (IDA). Statistics Denmark determines an individual's primary labor market attachment using income, education, and unemployment information from the Danish registers and the guidelines recommended by the International Labour Market Organization (ILO) where employment takes priority over unemployment, and unemployment takes priority over being outside of the labor force.

Specifically, we define the following five indicator variables: (1) Employment, (2) Unemployment, (3) Disability pension, (4) Transfers, and (5) Pension which are explained in detail below. First, **employment** is an indicator variable for employment. Next, **unemployment** is an indicator variable for receiving unemployment benefits. Third, **disability pension** (DP) is an indicator variable for receiving

disability pension. Fourth, **transfers** is an indicator variable for receiving cash benefits, students grants, or other public transfers excluding unemployment benefits, disability pension, VERP, and SP. Finally, **pension** is an indicator variable for receiving VERP, SP, other pensions, or being self supporting, i.e., being outside of the labor force without receiving government transfers. These outcomes reflect attachment to the labor market as well as receipt of various welfare benefits.

Thereafter, we study different dimensions of income and wealth using The Income Register (IND). First, we study **earnings** which includes income primarily due to labor supply containing taxable earnings, fringe benefits, tax-free earnings, anniversary and severance pay, and the value of stock options. Second, we study **income from shares** which is defined as the sum of dividends and gains or losses on shares and certain equity-based investment fund certificates. Third, we study **disposable income** which is income after tax and interest expenses and including the calculated rental value of own home. Fourth, we look at **wealth** which is defined as net wealth excluding pension wealth. Due to a now abolished wealth tax, the Danish registers contains detailed information on individual's wealth. All monetary values are winsorized at the 1st and 99th percentile and are deflated to DKK, 2023-prices, using the Consumer Price Index (CPI) from Statistics Denmark.

Sample sizes vary across the dependent variables because of data availability. Labor market attachment is available from year 2008 and on while income variables are measured from year 2000 and on. Sample sizes are presented in the regression tables.

3.4 Sample Selection

Our sample consists of parents of individuals in the iPSYCH sample. We restrict our sample to parents who are between 45 and 65 years old. This allows us to study labor market decline near retirement age. We exclude individuals of non-European ancestry to ensure the PGSs are valid as the GWAS tend to come from European samples.

3.5 Descriptive Statistics

In Table 2 (women) and Table 3 (men), we report descriptive statistics for the parent sample. First, we see that 3% of parents have a child who is a homogeneous *APOE-e4* carrier, whereas 28% of parents have a child who is a heterogeneous carrier.[7] Next, we see that men are more likely to be employed than women; 81% of men are employed between the ages of 45-65 whereas 75% of women of these

[7] This is in line with Rasmussen et al. (2018) who also found that 3% of the Danish population are homogeneous carriers, and 28% are heterogeneous carriers.

ages are. Consistent with this, men have higher average earnings and wealth compared to women. In addition, for both men and women, we see that age has a negative effect on labor market participation. Specifically, we see less labor force participation in the restricted sample aged 55-65 than we do in the full sample aged 45-65. We also show descriptive statistics by whether an individual's child is in the iPSYCH control group in the Appendix as well as compare our sample to the full population (Table A.1 for women and Table A.2 for men).

Table 2: Descriptive Statistics for Women

	Women aged 45 to 65		Women aged 55 to 65	
	Mean	SD	Mean	SD
Homogeneous carrier	0.03	0.17	0.03	0.17
Heterogeneous carrier	0.28	0.45	0.28	0.45
Age	52.45	5.22	58.62	2.89
Married	0.61	0.49	0.62	0.49
Control group (iPSYCH)	0.34	0.47	0.33	0.47
Lower sec., primary, unknown	0.24	0.43	0.25	0.44
General upper secondary	0.04	0.20	0.04	0.18
Vocational education	0.37	0.48	0.33	0.47
Short cycle tertiary	0.04	0.19	0.04	0.19
Bachelor	0.25	0.43	0.27	0.45
Master, doctoral	0.06	0.25	0.07	0.25
Experience	20.03	9.53	22.74	10.68
Year	2013.75	4.32	2015.46	3.75
GP visits	4.33	4.70	4.35	4.74
Dementia	0.00	0.01	0.00	0.02
Employment	0.75	0.44	0.68	0.47
Unemployment	0.02	0.15	0.02	0.15
Disability Pension	0.12	0.33	0.16	0.36
Other Transfers	0.06	0.24	0.04	0.20
Pension	0.05	0.21	0.10	0.30
Earnings (DKK)	309,881.21	230,492.19	285,356.46	238,635.23
Income from Shares (DKK)	1,390.93	7,483.68	1,897.33	8,596.73
Disposable Income (DKK)	287,963.68	117,021.56	286,304.43	120,902.28
Net Wealth (DKK)	277,004.38	871,114.50	432,520.27	972,974.69
$N \times Years$	1,123,485		375,336	
N	106,374		61,992	

Notes: This table displays the means and standard deviations for the genetic measure, control variables, and dependent variables for women aged 45-65 and women aged 55-65 for the years 2005-2020 (2008-2020 for labor market attachment variables). All monetary values are measured in DKK (2023-prices). N refers to the number of individuals, and $N \times Years$ refers to the total number of individual-year observations.

Table 3: Descriptive Statistics for Men

	Men aged 45 to 65		Men aged 55 to 65	
	Mean	SD	Mean	SD
Homogeneous carrier	0.03	0.17	0.03	0.17
Heterogeneous carrier	0.28	0.45	0.28	0.45
Age	53.40	5.50	59.04	3.01
Married	0.66	0.47	0.68	0.47
Control group (iPSYCH)	0.35	0.48	0.34	0.47
Lower sec., primary, unknown	0.25	0.44	0.25	0.44
General upper secondary	0.04	0.20	0.04	0.20
Vocational education	0.44	0.50	0.42	0.49
Short cycle tertiary	0.05	0.22	0.04	0.20
Bachelor	0.13	0.34	0.14	0.35
Master, doctoral	0.09	0.29	0.10	0.30
Experience	23.24	9.65	25.05	10.78
Year	2013.23	4.42	2014.49	4.14
GP visits	3.03	4.23	3.43	4.51
Dementia	0.00	0.01	0.00	0.02
Employment	0.81	0.39	0.75	0.43
Unemployment	0.03	0.17	0.03	0.16
Disability Pension	0.07	0.26	0.09	0.29
Other Transfers	0.04	0.19	0.03	0.18
Pension	0.05	0.22	0.09	0.29
Earnings (DKK)	418,762.20	330,770.13	375,798.13	330,229.50
Income from Shares (DKK)	8,471.34	38,478.96	9,238.66	39,879.88
Disposable Income (DKK)	354,849.90	209,190.83	354,381.95	212,788.61
Net Wealth (DKK)	507,119.12	1,496,333.75	712,262.50	1,635,967.50
$N \times$ *Years*	1,084,496		440,274	
N	99,993		66,369	

Notes: This table displays the means and standard deviations for the genetic measure, control variables, and dependent variables for men aged 45-65 and men aged 55-65 for the years 2005-2020 (2008-2020 for labor market attachment variables). All monetary values are measured in DKK (2023-prices). N refers to the number of individuals, and $N \times$ *Years* refers to the total number of individual-year observations.

4 Empirical Strategy

We estimate a linear regression model relating genetic risk for AD to a number of economic and health outcomes while adjusting for a host of potential confounds. We subscript individuals with i, age with a, and year with t. Specifically, we estimate the model:

$$Y_{iat} = G_i^C \delta + X_{ia}\theta + \pi_a + \rho_t + \varepsilon_{iat} \tag{1}$$

where Y_{iat} is either a health or an economic outcome, G_i^C is a measure of the genetic risk for AD of individual i's child (*APOE-e4* carrier status or polygenic risk score for AD), X_{ia} is a vector of observable exogenous characteristics, π_a is an age fixed effect, ρ_t is a year fixed effect, and ε_{iat} is the residual error

term. To account for biological differences across genders, we estimate the model separately for women and men. The vector of observable characteristics includes: the first ten principal components from the underlying genotype matrix to control for population stratification[8], education indicator variables[9], a marriage indicator, a quadratic in experience, and an indicator for whether the child is in the control group in the iPSYCH sample.

4.1 Spurious Associations with Other Genes

A potential concern is that genetic predisposition for AD might be correlated with genetic predispositions to other phenotypes due to what is termed "population stratification" (Benjamin et al., 2024). One of the primary ways that we address this is by including the principal components in the model. This allows us to avoid spurious associations with other genes. On a related note, a more specific concern is that a high risk of AD might be correlated with a lower score for educational attainment (EA). If so, our results might be driven by the low score for EA rather than a high score for AD. This could be a concern despite controlling for education as it has been shown that the polygenic score for EA is strongly associated with labor earnings (Papageorge and Thom, 2020) and wealth (Barth et al., 2020), even after controlling for completed education. However, we find no clear relationship between the AD PGS and the EA PGS. While our primary AD PGS (phen19) is weakly negatively correlated with the EA PGS, another AD PGS (gcst005921) is weakly positively correlated with the EA PGS as shown in Figure A.3. Therefore, there is no systematic relationship between the two PGSs. Furthermore, we also investigate whether the genetic risk of AD differs by whether individuals belong to the iPSYCH control group or not in Figure A.4. We find that in both the iPSYCH control group and the non-control group, the share of homogeneous carriers is 3%, and the share of heterogeneous carriers is 28%. We also find that the distribution of the AD PGS is the same for both groups.

4.2 Bias from Proxy-phenotype Design in APOE Estimations

Because we employ a proxy-phenotype design in which we use the child's phenotype as a proxy for the parent's phenotype, OLS estimates will be biased due to this measurement error. Per the usual intuition, this bias will attenuate estimates towards zero. In Appendix A.1, we provide some simple calculations that shed light on the magnitude of this bias in the regressions that use carrier status as the independent

[8]It is standard procedure to include the first (usually four or more) principal components of the genotypes from the dense single-nucleotide polymorphism (SNP) data to account for population differences (Benjamin et al., 2012).

[9]We define six education groups as follows: 1) Lower secondary, primary or unknown education, 2) General upper secondary, 3) Vocational education, 4) Short cycle tertiary, 5) Bachelor, and 6) Master or Doctoral.

variable. Specifically, we show that

$$\text{True Effect} \approx \text{Estimated Effect} \times \frac{1 - P(D=1)}{P(D=1|D^*=1) - P(D=1)}$$

where $P(D=1)$ is the probability of a child (or parent) being either a homogeneous or a heterogeneous carrier and $P(D=1|D^*=1)$ is the probability of a child being a carrier conditional on the parent being a carrier. We conduct some simple calculations in the Appendix and we show that

$$\frac{1 - P(D=1)}{P(D=1|D^*=1) - P(D=1)} = 2.19$$

To account for the effects of measurement error on the estimations, readers can roughly double our estimated effects.

5 Results

We now present our results. We begin by showing how the genetic risk of Alzheimer's Disease relates to both health and economic outcomes, with a focus on labor market outcomes, for women and men aged 45-65 and 55-65. We then study whether educational attainment has a moderating effect on AD in line with the 'cognitive reserve theory'. Lastly, we conduct a few robustness exercises.

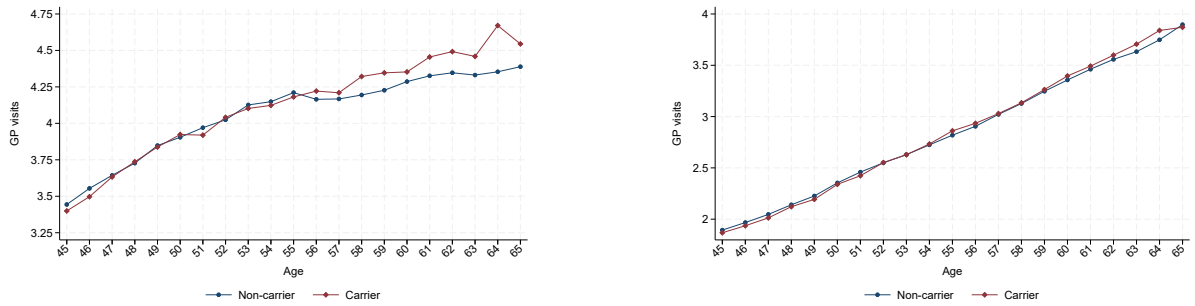
5.1 Motivation

Prior to presenting the regression estimation results, we begin with a visualization of the association between *APOE-e4* carrier status and labor supply over the life-course. In Figure 1, we plot the life-cycle profiles of GP visits and disability status by carrier status. We do so for both outcomes for women and men for a total of four plots. In panel (a), we see a clear positive association between carrier status and GP visits for women over the age of 55. In contrast, in panel (b), the association between carrier status and GP visits for men is not clear. Next, in panel (c), we also see a clear positive relationship between carrier status and share on disability pension for women. However, in panel (d), we do see that male carriers are slightly *less* likely to be on disability, although we will see in the regression results that there is no statistically significant association when we control for exogenous characteristics. Finally, in Figure A.5, we display similar figures for earnings, employment, and wealth. For the first two outcomes, we do not see stark differences. Notably, however, we do see that *APOE-e4* carriers do have slightly less wealth than non-carriers around age 65.

Figure 1: *APOE-e4* Carrier Status and Health and Labor Market Outcomes

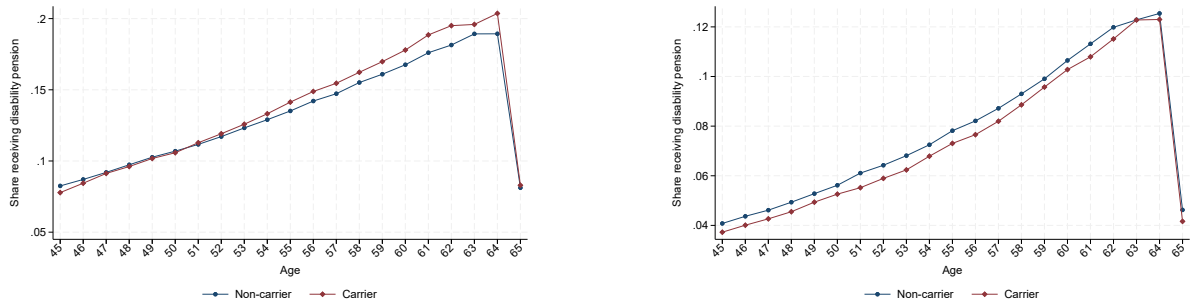
(a) Women: GP visits

(b) Men: GP visits



(c) Women: Disability Status

(d) Men: Disability Status



Notes: Displays the average number of GP visits or disability shares for women and men aged 45 to 65 by their child's *APOE-e4* carrier status.

5.2 Health Outcomes

We start by showing that our genetic measures of AD meaningfully predict health-related outcomes in Table 4 and Table 5. Both *APOE-4* carrier status and a higher AD PGS increase the risk of a dementia diagnosis for men and women. For women aged 45 to 65, having a carrier-child increases the risk of a dementia diagnosis by 91%, and for men aged 45 to 65, the risk increases by 44%. For this relatively young sample, a hospital contact related to a dementia diagnosis is a rare outcome, but we nevertheless see a large, statistically significant increase in the probability of a dementia diagnosis. A one SD increase in the AD PGS predicts an increased risk of dementia of around 43% for women and 24% for men aged 45 to 65. As contact with a GP is necessary for a dementia diagnosis in Denmark, we also investigate how our genetic measure for AD relates to visits to the GP. We find that having a carrier-child is positively associated with the number of GP visits for men and women aged 55 to 65. Hence, we conclude that our genetic markers for AD significantly predict AD-related outcomes. This validates the genetic proxies for AD.

Table 4: *APOE-e4* Carrier Status and Health Outcomes

	Dementia		GP visits	
	Age 45-65	Age 55-65	Age 45-65	Age 55-65
	(1)	(2)	(3)	(4)
Panel A: Women				
APOE-e4 Carrier	0.00016***	0.00035***	0.03496	0.09410***
	(0.00004)	(0.00010)	(0.02265)	(0.03399)
$N \times Years$	1,123,485	375,336	1,123,485	375,336
N	106,374	61,992	106,374	61,992
R^2	0.000	0.000	0.043	0.035
Mean	0.0002	0.0004	4.33	4.35
Pct. Change	91.40	93.91	0.81	2.16
Panel B: Men				
APOE-e4 Carrier	0.00008**	0.00018**	0.01978	0.05163*
	(0.00004)	(0.00008)	(0.02046)	(0.02981)
$N \times Years$	1,084,496	440,274	1,084,496	440,274
N	99,993	66,369	99,993	66,369
R^2	0.000	0.000	0.031	0.027
Mean	0.0002	0.0003	3.03	3.43
Pct. Change	44.44	52.93	0.65	1.51

Notes: This table reports estimates based on Equation 1 using child's *APOE-e4* carrier status as measure of genetic risk for women (Panel A) and men (Panel B). In columns (1) and (2), the outcome is a dummy for diagnosed dementia. In columns (3) and (4), the outcome variable is number of GP visits. Control variables included are age fixed effects, year fixed effects, the first 10 principal components, education dummies, marriage dummy, experience, experience squared, and dummy for whether child is in iPSYCH control group or not. N refers to the number of individuals, and $N \times Years$ refers to the total number of individual-year observations. Standard errors are clustered at the individual level and shown in parentheses. Significance levels are denoted as * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

5.3 Economic Outcomes for Women

We now turn to the relationship between genetic risk of AD and labor market outcomes for women. In Table 6, we do so using carrier status. In Table 7, we do so using the PGS.

In Table 6, we show that, for women aged 45-65, having a child with *APOE-e4* carrier status decreases the probability of employment by 0.3 percentage points and increases the probability of receiving disability pension by 0.4 percentage points. As 12% of women aged 45 to 65 are on disability pension, this corresponds to a large increase of 3%. We find no association between *APOE-e4* carrier status and the probability of receiving unemployment benefits, other transfers, or pension receipt.

The associations are larger when the sample is restricted to women aged 55 to 65. As age is the greatest risk factor of AD (Alzheimer's Association, 2024), we expect there to be larger consequences of genetic risk of AD for older individuals. For women aged 55 to 65, having a child with *APOE-e4* carrier status decreases the probability of employment by 0.7 percentage points and increases the probability of receiving disability pension by 0.8 percentage points - an increase of 5%. We still find no

Table 5: AD PGS and Health Outcomes

	Dementia		GP visits	
	Age 45-65	Age 55-65	Age 45-65	Age 55-65
	(1)	(2)	(3)	(4)
Panel A: Women				
AD PGS	0.00007***	0.00016***	0.01031	0.02040
	(0.00002)	(0.00005)	(0.01045)	(0.01583)
$N \times Years$	1,123,485	375,336	1,123,485	375,336
N	106,374	61,992	106,374	61,992
R^2	0.000	0.000	0.043	0.035
Mean	0.0002	0.0004	4.33	4.35
Pct. Change	43.31	43.54	0.24	0.47
Panel B: Men				
AD PGS	0.00004**	0.00009**	0.01164	0.03286**
	(0.00002)	(0.00004)	(0.00963)	(0.01385)
$N \times Years$	1,084,496	440,274	1,084,496	440,274
N	99,993	66,369	99,993	66,369
R^2	0.000	0.000	0.031	0.027
Mean	0.0002	0.0003	3.03	3.43
Pct. Change	23.87	27.08	0.38	0.96

Notes: This table reports estimates based on Equation 1 using child's AD PGS as measure of genetic risk for women (Panel A) and men (Panel B). In columns (1) and (2), the outcome is a dummy for diagnosed dementia. In columns (3) and (4), the outcome variable is number of GP visits. Control variables included are age fixed effects, year fixed effects, the first 10 principal components, education dummies, marriage dummy, experience, experience squared, and dummy for whether child is in iPSYCH control group or not. N refers to the number of individuals, and $N \times Years$ refers to the total number of individual-year observations. Standard errors are clustered at the individual level and shown in parentheses. Significance levels are denoted as * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

relationship with the probability of receiving unemployment benefits, others transfers, or being outside of the labor force (excluding disability pension).

Second, we show the relationship between increased genetic risk of AD and women's labor market attachment using the AD PGS in Table 7. We still find a negative relationship between increased genetic risk of AD and employment and a positive relationship with the probability of receiving disability pension for older women. For women aged 55 to 65, a one SD increase in the AD PGS decreases the probability of being employed by 0.2 percentage points, but increases the probability of receiving disability pension by 0.2 percentage points. We find no relationship for the probability of receiving unemployment benefits, other transfers, or pensions for women aged 55 to 65.

Next, we investigate the relationship between genetic risk of AD and earnings, income from shares, disposable income, and wealth for women in Table 8 using carrier status and Table 9 using the AD PGS. While we do not find any statistically significant effects, we do caution that measurement error means that our estimates should be interpreted as lower bounds. We also notice that for women aged

Table 6: *APOE-e4* Carrier Status and Labor Market Attachment for Women

	Employment (1)	Unemployment (2)	DP (3)	Transfers (4)	Pension (5)
Panel A: Age 45-65					
APOE-e4 Carrier	-0.00342*	-0.00031	0.00390**	0.00036	-0.00052
	(0.00183)	(0.00050)	(0.00176)	(0.00105)	(0.00087)
$N \times Years$	1,005,275	1,005,275	1,005,275	1,005,275	1,005,275
N	105,896	105,896	105,896	105,896	105,896
R^2	0.364	0.007	0.308	0.061	0.212
Mean	0.75	0.02	0.12	0.06	0.05
Pct. Change	-0.46	-1.31	3.15	0.58	-1.14
Panel B: Age 55-65					
APOE-e4 Carrier	-0.00716**	0.00037	0.00769***	0.00013	-0.00103
	(0.00290)	(0.00074)	(0.00272)	(0.00126)	(0.00179)
$N \times Years$	360,716	360,716	360,716	360,716	360,716
N	61,814	61,814	61,814	61,814	61,814
R^2	0.346	0.007	0.335	0.037	0.253
Mean	0.68	0.02	0.16	0.04	0.10
Pct. Change	-1.05	1.70	4.88	0.30	-1.06

Notes: This table reports estimates based on Equation 1 using child's *APOE-e4* carrier status as measure of genetic risk for women aged 45 to 65 (Panel A) and women aged 55 to 65 (Panel B). In column (1), the outcome is a dummy for being employed, in column (2), the outcome is a dummy for being unemployed, in column (3), the outcome is a dummy for receiving disability pension, in column (4), the outcome variable is a dummy for receiving transfers, and in column (5), the outcome variable is a dummy for pension. Control variables included are age fixed effects, year fixed effects, the first 10 principal components, education dummies, marriage dummy, experience, experience squared, and dummy for whether child is in iPSYCH control group or not. N refers to the number of individuals, and $N \times Years$ refers to the total number of individual-year observations. Standard errors are clustered at the individual level and shown in parentheses. Significance levels are denoted as * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

55 to 65, having a carrier-child is associated with a decrease in wealth of 13,500 DKK (≈1890 USD), corresponding to a decrease of 3%. However, the standard errors are large, and we cannot reject a null effect. It may seem surprising that while we find a decrease in employment, we do not find a decrease in earnings. However, when decomposing the effect of earnings further by age, we do find a statistically significant decrease associated with carrier status for women aged 56 to 57 years old (see Figure A.6).

5.4 Economic Outcomes for Men

We now repeat the same analysis for men. In Table 10, we present the association between having an *APOE-e4* carrier child and labor market outcomes. In Table 11, we present the relationship between the AD PGS and labor market outcomes. We find no statistically significant relationships between genetic risk of AD and labor market attachment, neither for *APOE-e4* carrier status nor for the AD PGS. These results highlight the important gender differences in genetic predictors of labor market decline.

Table 7: AD PGS and Labor Market Attachment for Women

	Employment	Unemployment	DP	Transfers	Pension
	(1)	(2)	(3)	(4)	(5)
Panel A: Age 45-65					
AD PGS	-0.00014	-0.00033	0.00111	-0.00051	-0.00013
	(0.00085)	(0.00023)	(0.00082)	(0.00049)	(0.00040)
$N \times Years$	1,005,275	1,005,275	1,005,275	1,005,275	1,005,275
N	105,896	105,896	105,896	105,896	105,896
R^2	0.364	0.007	0.308	0.061	0.212
Mean	0.75	0.02	0.12	0.06	0.05
Pct. Change	-0.02	-1.39	0.89	-0.84	-0.28
Panel B: Age 55-65					
AD PGS	-0.00240*	0.00021	0.00244*	-0.00018	-0.00007
	(0.00134)	(0.00034)	(0.00126)	(0.00060)	(0.00083)
$N \times Years$	360,716	360,716	360,716	360,716	360,716
N	61,814	61,814	61,814	61,814	61,814
R^2	0.346	0.007	0.335	0.037	0.253
Mean	0.68	0.02	0.16	0.04	0.10
Pct. Change	-0.35	0.97	1.55	-0.42	-0.08

Notes: This table reports estimates based on Equation 1 using child's AD PGS as measure of genetic risk for women aged 45 to 65 (Panel A) and women aged 55 to 65 (Panel B). In column (1), the outcome is a dummy for being employed, in column (2), the outcome is a dummy for being unemployed, in column (3), the outcome is a dummy for receiving disability pension, in column (4), the outcome variable is a dummy for receiving transfers, and in column (5), the outcome variable is a dummy for pension. Control variables included are age fixed effects, year fixed effects, the first 10 principal components, education dummies, marriage dummy, experience, experience squared, and dummy for whether child is in iPSYCH control group or not. N refers to the number of individuals, and $N \times Years$ refers to the total number of individual-year observations. Standard errors are clustered at the individual level and shown in parentheses. Significance levels are denoted as * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Altmann et al. (2014) found that *APOE-e4* confers greater AD risk for women than men. If it is the case that genetic predictors are more important for women, it would follow naturally that the associations between genetic risk of AD and labor market outcomes would be stronger for women than for men, which is precisely what we find.

We now look at the effects of genetic risk of AD on earnings, income from shares, disposable income, and wealth in Table 12 (using carrier status) and Table 13 (using the PGS). Again, we find no statistically significant relationships between genetic risk of AD and income and wealth measures. However, as was the case for women, we highlight that having a carrier-child is associated with a large and negative effect on income from shares for men aged 55 to 65 of 3%. We find similar effects for wealth. That said, we do offer the caveat that the standard errors associated with these estimates are large. Nevertheless, this negative impact of AD on income from shares and wealth would be in line with other evidence showing that AD affects cognitive abilities and financial planning, and hence investment and wealth.

Table 8: *APOE-e4* Carrier Status, Income, and Wealth for Women

	Earnings	Income from Shares	Disposable Income	Wealth
	(1)	(2)	(3)	(4)
Panel A: Age 45-65				
APOE-e4 Carrier	-511.04	-25.84	5.86	-6,274.34
	(919.72)	(37.80)	(536.69)	(5,386.59)
$N \times Years$	1,229,258	1,229,258	1,229,258	1,229,258
N	106,735	106,735	106,735	106,735
R^2	0.507	0.024	0.391	0.099
Mean	308,707.80	1,313.37	285,150.85	274,620.24
Pct. Change	-0.17	-1.97	0.00	-2.28
Panel B: Age 55-65				
APOE-e4 Carrier	-831.49	-52.87	-81.52	-13,538.29
	(1,384.46)	(67.37)	(805.49)	(8,740.21)
$N \times Years$	382,779	382,779	382,779	382,779
N	62,063	62,063	62,063	62,063
R^2	0.499	0.025	0.389	0.090
Mean	284,489.22	1,876.72	285,440.72	432,197.07
Pct. Change	-0.29	-2.82	-0.03	-3.13

Notes: This table reports estimates based on Equation 1 using child's *APOE-e4* carrier status as measure of genetic risk for women aged 45 to 65 (Panel A) and women aged 55 to 65 (Panel B). In column (1), the outcome is earnings, in column (2), the outcome is income from shares, in column (3), the outcome is disposable income, and in column (4), the outcome is net wealth excluding pension wealth. All monetary values are in DKK, 2023-prices. Control variables included are age fixed effects, year fixed effects, the first 10 principal components, education dummies, marriage dummy, experience, experience squared, and dummy for whether child is in iPSYCH control group or not. N refers to the number of individuals, and $N \times Years$ refers to the total number of individual-year observations. Standard errors are clustered at the individual level and shown in parentheses. Significance levels are denoted as * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

5.5 Educational Attainment

We now investigate if a genetic propensity towards higher education levels can moderate the effects of genetic AD risk on labor market outcomes. Specifically, it may be the case that some individuals can withstand larger changes to the brain stemming from the build-up of beta-amyloid due to a greater cognitive reserve. This is referred to as the "cognitive reserve theory" (Stern, 2012). In fact, the model proposed by Grossman (1972) in which higher educational attainment causes better health is very much in this spirit; in his model, higher education mitigates the aging process. Likewise, Barcellos et al. (2025), using a compulsory schooling reform in the UK, find that increased education reduces the incidence of Alzheimer's Disease and related dementias. To test the cognitive reserve theory, we include the educational attainment (EA) PGS and interact it with carrier status in Table 14 using health outcomes and in Table 15 and Table 16 using economic outcomes. We also use the AD PGS in robustness checks in the Appendix in Table A.3, Table A.4 and Table A.5.

Table 9: AD PGS, Income, and Wealth for Women

	Earnings	Income from Shares	Disposable Income	Wealth
	(1)	(2)	(3)	(4)
Panel A: Age 45-65				
AD PGS	118.82	-1.11	62.07	-501.93
	(425.83)	(17.16)	(251.13)	(2,492.49)
$N \times Years$	1,229,258	1,229,258	1,229,258	1,229,258
N	106,735	106,735	106,735	106,735
R^2	0.507	0.024	0.391	0.099
Mean	308,707.80	1,313.37	285,150.85	274,620.24
Pct. Change	0.04	-0.08	0.02	-0.18
Panel B: Age 55-65				
AD PGS	-40.87	-10.95	-262.78	-2,345.32
	(642.05)	(30.59)	(377.88)	(4,066.31)
$N \times Years$	382,779	382,779	382,779	382,779
N	62,063	62,063	62,063	62,063
R^2	0.499	0.025	0.389	0.090
Mean	284,489.22	1,876.72	285,440.72	432,197.07
Pct. Change	-0.01	-0.58	-0.09	-0.54

Notes: This table reports estimates based on Equation 1 using child's AD PGS as measure of genetic risk for women aged 45 to 65 (Panel A) and women aged 55 to 65 (Panel B). In column (1), the outcome is earnings, in column (2), the outcome is income from shares, in column (3), the outcome is disposable income, and in column (4), the outcome is net wealth excluding pension wealth. All monetary values are in DKK, 2023-prices. Control variables included are age fixed effects, year fixed effects, the first 10 principal components, education dummies, marriage dummy, experience, experience squared, and dummy for whether child is in iPSYCH control group or not. N refers to the number of individuals, and $N \times Years$ refers to the total number of individual-year observations. Standard errors are clustered at the individual level and shown in parentheses. Significance levels are denoted as * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Looking at Table 14, we note that inclusion of the EA PGS and its interaction with the carrier indicator does not change the coefficient estimates for carrier status. For women, the EA PGS does not predict dementia diagnoses. A higher EA PGS is associated with fewer GP visits, but the interaction between carrier status and the EA PGS is not significant. Turning to men, we find that higher EA PGS predicts both fewer dementia diagnoses and GP visits. However, we also find a positive statistically significant interaction between carrier status and EA PGS for GP visits. This may reflect that more educated people have heightened awareness of their own health and so may seek out medical care at earlier stages in response to subtle health changes.

We now turn to economic outcomes in Table 15 (women) and Table 16 (men). For both women and men, we can clearly see that the EA PGS strongly predicts *all* economic outcomes. For women aged 55-65, a one SD increase in the EA PGS increases the probability of employment by 1.5 percentage points and decreases the probability of disability pension by 1.1 percentage points. For men aged 55-65,

Table 10: *APOE-e4* Carrier Status and Labor Market Attachment for Men

	Employment (1)	Unemployment (2)	DP (3)	Transfers (4)	Pension (5)
Panel A: Age 45-65					
APOE-e4 Carrier	0.00063	0.00050	-0.00122	-0.00028	0.00036
	(0.00183)	(0.00059)	(0.00150)	(0.00087)	(0.00090)
$N \times Years$	940,718	940,718	940,718	940,718	940,718
N	98,560	98,560	98,560	98,560	98,560
R^2	0.251	0.013	0.187	0.051	0.166
Mean	0.81	0.03	0.07	0.04	0.05
Pct. Change	0.08	1.78	-1.69	-0.72	0.71
Panel B: Age 55-65					
APOE-e4 Carrier	-0.00124	0.00076	-0.00054	0.00014	0.00088
	(0.00276)	(0.00081)	(0.00223)	(0.00105)	(0.00167)
$N \times Years$	407,980	407,980	407,980	407,980	407,980
N	65,436	65,436	65,436	65,436	65,436
R^2	0.240	0.011	0.196	0.038	0.189
Mean	0.75	0.03	0.09	0.03	0.09
Pct. Change	-0.16	2.76	-0.57	0.43	0.97

Notes: This table reports estimates based on Equation 1 using child's *APOE-e4* carrier status as measure of genetic risk for men aged 45 to 65 (Panel A) and men aged 55 to 65 (Panel B). In column (1), the outcome is a dummy for being employed, in column (2), the outcome is a dummy for being unemployed, in column (3), the outcome is a dummy for receiving disability pension, in column (4), the outcome variable is a dummy for receiving transfers, and in column (5), the outcome variable is a dummy for pension. Control variables included are age fixed effects, year fixed effects, the first 10 principal components, education dummies, marriage dummy, experience, experience squared, and dummy for whether child is in iPSYCH control group or not. N refers to the number of individuals, and $N \times Years$ refers to the total number of individual-year observations. Standard errors are clustered at the individual level and shown in parentheses. Significance levels are denoted as * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

a one SD increase in the EA PGS increases the probability of employment by 1.9 percentage points and decreases the probability of disability pension by 1.5 percentage points. These findings are generally consistent with Papageorge and Thom (2020) who find that the EA PGS has a large effect on labor market outcomes - even when controlling for education.

We now consider the evidence for or against the cognitive reserve theory. To do this, we will investigate if the point estimates on the interaction coefficients have the opposite signs of the coefficient estimates for AD risk for each outcome. As before, for women, carrier status is negatively associated with employment but positively associated with disability pension receipt. For women aged 55 to 65, the interaction between carrier status and the EA PGS is statistically significant and negative for disability pension receipt. For employment, we do not find a statistically significant interaction between carrier status and the EA PGS, but we note that the coefficient is positive. This is generally consistent with the theory. Turning to men, we see no statistically significant interactions effects for employment or

Table 11: AD PGS and Labor Market Attachment for Men

	Employment (1)	Unemployment (2)	DP (3)	Transfers (4)	Pension (5)
Panel A: Age 45-65					
AD PGS	-0.00012	-0.00001	0.00008	0.00042	-0.00037
	(0.00085)	(0.00027)	(0.00071)	(0.00040)	(0.00041)
$N \times Years$	940,718	940,718	940,718	940,718	940,718
N	98,560	98,560	98,560	98,560	98,560
R^2	0.251	0.013	0.187	0.051	0.166
Mean	0.81	0.03	0.07	0.04	0.05
Pct. Change	-0.01	-0.03	0.12	1.07	-0.73
Panel B: Age 55-65					
AD PGS	-0.00015	-0.00012	0.00062	0.00056	-0.00092
	(0.00129)	(0.00038)	(0.00105)	(0.00048)	(0.00076)
$N \times Years$	407,980	407,980	407,980	407,980	407,980
N	65,436	65,436	65,436	65,436	65,436
R^2	0.240	0.011	0.196	0.038	0.189
Mean	0.75	0.03	0.09	0.03	0.09
Pct. Change	-0.02	-0.43	0.65	1.72	-1.01

Notes: This table reports estimates based on Equation 1 using child's AD PGS as measure of genetic risk for men aged 45 to 65 (Panel A) and men aged 55 to 65 (Panel B). In column (1), the outcome is a dummy for being employed, in column (2), the outcome is a dummy for being unemployed, in column (3), the outcome is a dummy for receiving disability pension, in column (4), the outcome variable is a dummy for receiving transfers, and in column (5), the outcome variable is a dummy for pension. Control variables included are age fixed effects, year fixed effects, the first 10 principal components, education dummies, marriage dummy, experience, experience squared, and dummy for whether child is in iPSYCH control group or not. N refers to the number of individuals, and $N \times Years$ refers to the total number of individual-year observations. Standard errors are clustered at the individual level and shown in parentheses. Significance levels are denoted as * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

disability pension receipt. When using earnings as the outcome, we do not see evidence of statistically significant interactions for women and, while we do see a significant interaction estimate for men aged 45-65, the interaction is no longer significant when using the AD PGS in Table A.5. Finally, when employing wealth as the outcome, for both men and women, we see that the EA PGS is associated with an increase in wealth, but for men aged 55 to 65, the interaction between the EA PGS and carrier status is negative and significant too. Using the AD PGS, we also find a statistically significant, negative interaction. For women, we find no statistically significant negative association for the interaction between the EA PGS and carrier status in Table 15, but we do find a negative interaction between the EA PGS and the AD PGS for wealth in Table A.4. Prima facie, this seems at odds with the cognitive reserve theory. One possible explanation is that, because individuals with a high EA PGS have more wealth, genetic risk of AD might have larger effects because there is more wealth to lose.

Table 12: *APOE-e4* Carrier Status, Income, and Wealth for Men

	Earnings	Income from Shares	Disposable Income	Wealth
	(1)	(2)	(3)	(4)
Panel A: Age 45-65				
APOE-e4 Carrier	-463.50	-288.89	-587.38	-9,255.85
	(1,540.46)	(216.05)	(1,077.72)	(9,529.03)
$N \times$ *Years*	1,243,045	1,243,045	1,243,045	1,243,045
N	101,228	101,228	101,228	101,228
R^2	0.396	0.021	0.231	0.076
Mean	415,563.64	7,977.01	347,974.46	496,335.66
Pct. Change	-0.11	-3.62	-0.17	-1.86
Panel B: Age 55-65				
APOE-e4 Carrier	-925.62	-232.05	-1300.43	-20,391.22
	(2,032.03)	(307.18)	(1,474.17)	(14,038.20)
$N \times$ *Years*	465,012	465,012	465,012	465,012
N	67,010	67,010	67,010	67,010
R^2	0.403	0.021	0.235	0.073
Mean	373,107.11	9,046.00	351,391.72	710,473.37
Pct. Change	-0.25	-2.57	-0.37	-2.87

Notes: This table reports estimates based on Equation 1 using child's *APOE-e4* carrier status as measure of genetic risk for men aged 45 to 65 (Panel A) and men aged 55 to 65 (Panel B). In column (1), the outcome is earnings, in column (2), the outcome is income from shares, in column (3), the outcome is disposable income, and in column (4), the outcome is net wealth excluding pension wealth. All monetary values are in DKK, 2023-prices. Control variables included are age fixed effects, year fixed effects, the first 10 principal components, education dummies, marriage dummy, experience, experience squared, and dummy for whether child is in iPSYCH control group or not. N refers to the number of individuals, and $N \times$ *Years* refers to the total number of individual-year observations. Standard errors are clustered at the individual level and shown in parentheses. Significance levels are denoted as * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

5.6 Robustness Checks

We conduct additional robustness checks to ensure the validity of our results. First, we check whether the use of different AD PGSs (as mentioned in Table 1) significantly impacts our results. We present these results in Figures A.7 to A.12. Generally, our results are robust to the use of different AD PGSs. In fact, while our primary AD PGS does not predict a positive association between AD and GP visits for women aged 55-65, six other scores do. Likewise, we find that, while our primary AD PGS does not predict a decrease in employment for women aged 45-65, five of the other scores considered in the Appendix do indicate a negative association with employment. For men aged 55-65 and for gcst005920 and ga3646, we do find negative associations with income from shares and wealth.

While we combine heterogeneous and homogeneous carriers to increase power in our primary specification, in Table A.6-A.10, we include dummies for heterogeneous and homogeneous carriers separately. For women, having a homogeneous carrier child is more predictive of a dementia diagnosis than

Table 13: AD PGS, Income, and Wealth for Men

	Earnings	Income from Shares	Disposable Income	Wealth
	(1)	(2)	(3)	(4)
Panel A: Age 45-65				
AD PGS	-79.35	-164.73	-276.36	-3947.66
	(715.84)	(101.28)	(500.42)	(4451.13)
$N \times Years$	1,243,045	1,243,045	1,243,045	1,243,045
N	101,228	101,228	101,228	101,228
R^2	0.396	0.021	0.231	0.076
Mean	415,563.64	7,977.01	347,974.46	496,335.66
Pct. Change	-0.02	-2.07	-0.08	-0.80
Panel B: Age 55-65				
AD PGS	-581.42	-229.89	-334.18	-7776.15
	(939.83)	(142.64)	(679.55)	(6512.42)
$N \times Years$	465,012	465,012	465,012	465,012
N	67,010	67,010	67,010	67,010
R^2	0.403	0.021	0.235	0.073
Mean	373,107.11	9,046.00	351,391.72	710,473.37
Pct. Change	-0.16	-2.54	-0.10	-1.09

Notes: This table reports estimates based on Equation 1 using child's AD PGS as measure of genetic risk for men aged 45 to 65 (Panel A) and men aged 55 to 65 (Panel B). In column (1), the outcome is earnings, in column (2), the outcome is income from shares, in column (3), the outcome is disposable income, and in column (4), the outcome is net wealth excluding pension wealth. All monetary values are in DKK, 2023-prices. Control variables included are age fixed effects, year fixed effects, the first 10 principal components, education dummies, marriage dummy, experience, experience squared, and dummy for whether child is in iPSYCH control group or not. N refers to the number of individuals, and $N \times Years$ refers to the total number of individual-year observations. Standard errors are clustered at the individual level and shown in parentheses. Significance levels are denoted as * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

having a heterogeneous carrier, in line with our knowledge of genetic determinants of AD. Having a homogeneous carrier-child is associated with an increased risk of dementia by 340% for women aged 55 to 65, while having a heterogeneous carrier-child is associated with an increased risk of 68%. For men aged 55 to 65, we find that having a heterogeneous carrier-child is associated with an increased risk of dementia of 44% and having a homogeneous carrier-child is associated with a 139% increase. However, the association between dementia and having a homogeneous carrier-child is not statistically significant for men aged 55 to 65. We caution that dementia diagnosis is a rare outcome and by not combining homogeneous and heterogeneous carriers, we lose power (hence our motivation for combining the indicators for homo- and heterogeneous carries). From our results, it can clearly be seen that carrier status is more predictive for women than for men which again highlights important gender differences.

We also study the separate effects of homo- and heterogeneous carrier status on economic outcomes. For women, we only find statistically significant effects for heterogeneous carriers as one might expect.

Table 14: *APOE-e4* Carrier Status, Educational Attainment, and Health

	Dementia		GP visits	
	Age 45-65	Age 55-65	Age 45-65	Age 55-65
	(1)	(2)	(3)	(4)
Panel A: Women				
APOE-e4 Carrier	0.00016***	0.00035***	0.03555	0.09446***
	(0.00004)	(0.00011)	(0.02259)	(0.03419)
EA PGS	-0.00002	-0.00001	-0.14648***	-0.15161***
	(0.00002)	(0.00005)	(0.01313)	(0.01912)
APOE-e4 Carrier × EA PGS	0.00001	-0.00005	-0.00441	0.00832
	(0.00004)	(0.00010)	(0.02219)	(0.03331)
$N \times Years$	1,123,485	375,336	1,123,485	375,336
N	106,374	61,992	106,374	61,992
R^2	0.000	0.000	0.044	0.036
Mean	0.0002	0.0004	4.33	4.35
Pct. Change (Carrier)	91.52	94.70	0.82	2.17
Pct. Change (EA PGS)	-12.67	-2.77	-3.39	-3.48
Pct. Change (Carrier × EA PGS)	5.33	-13.74	-0.10	0.19
Panel B: Men				
APOE-e4 Carrier	0.00008**	0.00019**	0.01881	0.04568
	(0.00004)	(0.00008)	(0.02050)	(0.03009)
EA PGS	-0.00003	-0.00006*	-0.16044***	-0.18629***
	(0.00002)	(0.00004)	(0.01182)	(0.01707)
APOE-e4 Carrier × EA PGS	-0.00001	-0.00004	0.06353***	0.07941***
	(0.00004)	(0.00008)	(0.01931)	(0.02881)
$N \times Years$	1,084,496	440,274	1,084,496	440,274
N	99,993	66,369	99,993	66,369
R^2	0.000	0.000	0.032	0.029
Mean	0.0002	0.0003	3.03	3.43
Pct. Change (Carrier)	44.58	53.59	0.62	1.33
Pct. Change (EA PGS)	-16.45	-17.38	-5.30	-5.44
Pct. Change (Carrier × EA PGS)	-5.61	-11.27	2.10	2.32

Notes: This table reports estimates based on Equation 1 using child's *APOE-e4* carrier status as measure of genetic risk for women (Panel A) and men (Panel B), but additionally adding child's EA PGS and interactions of EA PGS and *APOE-e4* carrier status. In columns (1) and (2), the outcome is a dummy for diagnosed dementia. In columns (3) and (4), the outcome is number of GP visits. Control variables included are age fixed effects, year fixed effects, the first 10 principal components, education dummies, marriage dummy, experience, experience squared, and dummy for whether child is in iPSYCH control group or not. N refers to the number of individuals, and $N \times Years$ refers to the total number of individual-year observations. Standard errors are clustered at the individual level and shown in parentheses. Significance levels are denoted as * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

However, the point estimates are larger for homogeneous carriers. For example, for women aged 55 to 65, having a homogeneous carrier-child is associated with a 7% increase in disability pension-receipt, while having a heterogeneous carrier-child is associated with an increase of 5%. For men aged 45-65

Table 15: *APOE-e4* Carrier Status, Educational Attainment, and Economic Outcomes for Women

	Employment	DP	Earnings	Wealth
	(1)	(2)	(3)	(4)
Panel A: Age 45-65				
APOE-e4 Carrier	-0.00344*	0.00388**	-536.95	-6,597.88
	(0.00183)	(0.00175)	(919.45)	(5,366.82)
EA PGS	0.01219***	-0.00804***	6,000.36***	76,237.52***
	(0.00107)	(0.00102)	(538.18)	(3,191.51)
APOE-e4 Carrier × EA PGS	0.00128	-0.00245	-647.93	-5,120.52
	(0.00183)	(0.00177)	(940.50)	(5,526.98)
$N \times$ *Years*	1,005,275	1,005,275	1,229,258	1,229,258
N	105,896	105,896	106,735	106,735
R^2	0.365	0.308	0.507	0.106
Mean	0.75	0.12	308,707.80	274,620.24
Pct. Change (Carrier)	-0.46	3.13	-0.17	-2.40
Pct. Change (EA PGS)	1.63	-6.50	1.94	27.76
Pct. Change (Carrier × EA PGS)	0.17	-1.98	-0.21	-1.86
Panel B: Age 55-65				
APOE-e4 Carrier	-0.00742**	0.00800***	-879.31	-13,490.20
	(0.00290)	(0.00274)	(1,372.18)	(8,573.90)
EA PGS	0.01457***	-0.01099***	6,033.66***	93,725.60***
	(0.00169)	(0.00154)	(805.94)	(5,169.71)
APOE-e4 Carrier × EA PGS	0.00351	-0.00480*	323.59´	-8,303.47
	(0.00290)	(0.00274)	(1,405.29)	(8,918.78)
$N \times$ *Years*	360,716	360,716	382,779	382,779
N	61,814	61,814	62,063	62,063
R^2	0.347	0.336	0.500	0.098
Mean	0.68	0.16	284,489.22	432,197.07
Pct. Change (Carrier)	-1.09	5.08	-0.31	-3.12
Pct. Change (EA PGS)	2.14	-6.97	2.12	21.69
Pct. Change (Carrier × EA PGS)	0.51	-3.05	0.11	-1.92

Notes: This table reports estimates based on Equation 1 using child's *APOE-e4* carrier status as measure of genetic risk for women aged 45-65 (Panel A) and women aged 55-65 (Panel B), but additionally adding child's EA PGS and interactions of EA PGS and *APOE-e4* carrier status. In column (1), the outcome variable is a dummy for employment, in column (2), the outcome is a dummy for receiving disability pension, in column (3), the outcome is earnings, and in column (4), the outcome is net wealth excluding pension wealth. All monetary values are in DKK, 2023-prices. Control variables included are age fixed effects, year fixed effects, the first 10 principal components, education dummies, marriage dummy, experience, experience squared, and dummy for whether child is in iPSYCH control group or not. N refers to the number of individuals, and $N \times$ *Years* refers to the total number of individual-year observations. Standard errors are clustered at the individual level and shown in parentheses. Significance levels are denoted as * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

and 55-65, we do find a statistically significant negative association between having a homogeneous carrier-child and employment.

We also estimate a probit model for our binary outcomes, namely, the dementia diagnosis and the

Table 16: *APOE-e4* Carrier Status, Educational Attainment, and Economic Outcomes for Men

	Employment	DP	Earnings	Wealth
	(1)	(2)	(3)	(4)
Panel A: Age 45-65				
APOE-e4 Carrier	0.00058	-0.00120	-384.47	-8,938.16
	(0.00183)	(0.00150)	(1530.12)	(9,415.44)
EA PGS	0.01685***	-0.01180***	12,442.90***	13,1075.45***
	(0.00108)	(0.00092)	(887.49)	(5,598.27)
APOE-e4 Carrier × EA PGS	0.00128	0.00059	-3,050.42*	-14,295.14
	(0.00184)	(0.00154)	(1,572.67)	(9,777.01)
$N \times$ *Years*	940,718	940,718	1,243,045	1,243,045
N	98,560	98,560	101,228	101,228
R^2	0.252	0.189	0.397	0.082
Mean	0.81	0.07	415,563.64	496,335.66
Pct. Change (Carrier)	0.07	-1.66	-0.09	-1.80
Pct. Change (EA PGS)	2.08	-16.37	2.99	26.41
Pct. Change (Carrier × EA PGS)	0.16	0.81	-0.73	-2.88
Panel B: Age 55-65				
APOE-e4 Carrier	-0.00126	-0.00058	-698.45	-17,899.73
	(0.00277)	(0.00226)	(2,004.44)	(13,761.78)
EA PGS	0.01854***	-0.01464***	11,279.71***	16,1705.64***
	(0.00161)	(0.00133)	(11,65.34)	(8,194.66)
APOE-e4 Carrier × EA PGS	0.00128	-0.00005	-2,512.98	-25,004.38*
	(0.00276)	(0.00226)	(2,094.26)	(14,154.46)
$N \times$ *Years*	407,980	407,980	465,012	465,012
N	65,436	65,436	67,010	67,010
R^2	0.242	0.198	0.404	0.081
Mean	0.75	0.09	373,107.11	710,473.37
Pct. Change (Carrier)	-0.17	-0.61	-0.19	-2.52
Pct. Change (EA PGS)	2.46	-15.43	3.02	22.76
Pct. Change (Carrier × EA PGS)	0.17	-0.05	-0.67	-3.52

Notes: This table reports estimates based on Equation 1 using child's *APOE-e4* carrier status as measure of genetic risk for men aged 45-65 (Panel A) and men aged 55-65 (Panel B), but additionally adding child's EA PGS and interactions of EA PGS and *APOE-e4* carrier status. In column (1), the outcome variable is a dummy for employment, in column (2), the outcome is a dummy for receiving disability pension, in column (3), the outcome is earnings, and in column (4), the outcome is net wealth excluding pension wealth. All monetary values are in DKK, 2023-prices. Control variables included are age fixed effects, year fixed effects, the first 10 principal components, education dummies, marriage dummy, experience, experience squared, and dummy for whether child is in iPSYCH control group or not. N refers to the number of individuals, and $N \times$ *Years* refers to the total number of individual-year observations. Standard errors are clustered at the individual level and shown in parentheses. Significance levels are denoted as * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

labor market attachment outcomes. We report the results in Tables A.11-A.15. We find that our results remain robust.

Additionally, we repeat our regressions on earnings, income from shares, disposable income, and

wealth using log-specifications in Tables A.16-A.19. For women, we find no statistically significant results. For men, we find that our genetic measures are associated with a decrease in income from shares. A one SD increase in the AD PGS is associated with a 4% decrease in income from shares for men aged 55 to 65 given positive income from shares. This is in line with other evidence suggesting increased risk of AD and cognitive decline is associated with worse financial decision-making.

Next, we investigate the relationship between genetic risk of AD and mortality using precise information on deaths from The Cause of Death Register (DODSAARS/DODSAASG) for the years 2005-2020. First, we show descriptive statistics in Figure A.13, and second, we use mortality as dependent variable and estimate the association between genetic predictors of AD and mortality in Table A.20. For this relatively young sample, we find that mortality does not vary by genetic risk of AD.

We now turn to studying selection out of the labor market in Tables A.21-A.22. Some individuals could already be outside the labor market at age 45. For these individuals, genetic risk of AD might not matter as much as they are already outside the labor market - possibly due to reasons other than cognitive decline given their young age. We repeat our analysis on labor market outcomes using only individuals with positive labor market earnings at age 45.[10] We find similar results as for our main sample. For the restricted sample, for women aged 55 to 65, having a carrier status child is now associated with an 7% increase in disability pension compared to an estimate of 5% for our main specification.

We also study effects for other family members. First, we start by showing that having a carrier-*grandchild* is also associated with an increased risk of dementia diagnosis in Table A.23. We again see gender differences as genetic risk of AD is associated with a larger risk of dementia diagnosis for grandmothers than grandfathers. Second, we study household earnings and wealth for couples in Tables A.24-A.26. We find that for women, having a carrier-child is associated with a negative decrease in household wealth of 49,000 DKK (≈6,860 USD) corresponding to a 3% decrease. This effect is driven by couples where the woman is married to the parent of the child whose genetics are utilized. For men, we do not find a statistically significant effect. However, we do not find a statistically significant association when we restrict the sample to couples who are both in the age range of 45-65 and 55-65 for neither men nor women. This suggests that differential effects for men and women could possibly be due to women in general being younger than their male partner, and hence, different age distributions of couples could affect the results. However, we again caution that measurement error biases our results downwards, and the estimates for wealth are in general large and negative, although these standard errors are also large.

[10] We use data from 1988-2020 to create a dummy for positive labor market earnings.

As an additional robustness check, we re-balance the distributions of age, education, and time periods of women to match the distributions of men using entropy balancing. We discuss this methodology in Subsection A.4. Balancing does not change our results significantly. We can hence conclude that the difference in age, education, and time period cannot explain the total difference in genetic risk of AD associations for men and women.

Finally, we estimate IV regressions in Appendix A.5 where we instrument our AD PGS using another AD PGS which under certain assumptions can address measurement error bias. We find a strong first stage, and in general, we find that our estimates are approximately doubled in the IV regressions. We find that a one SD increase in the AD PGS is associated with an 83% increase in the risk of dementia for women aged 55-65 and 48% for men in the same age range. In comparison, in our main specification, we find that a one SD increase in the AD PGS is associated with 44% increase for women aged 55-65 and 27% for men aged 55-65.

6 Conclusion

Our results show that there is an association between genetic predisposition for Alzheimer's Disease and health and labor market outcomes before the state pension retirement age. First, we find that genetic risk of AD predicts diagnosed dementia and GP visits for men and women. Having an *APOE-e4* carrier child is associated with an increase in the probability of diagnosed dementia by 94% and 2% more GP visits for women aged 55 to 65. For men aged 55 to 65, carrier status is associated with a 53% increase in the risk of diagnosed dementia and a 2% increase in GP visits.

We find that for women, genetic risk of AD decreases the probability of employment and increases the probability of disability pension. These associations become stronger by age. Mothers aged 45-65 with an *APOE-e4* carrier child are 0.3 percentage points less likely to be employed and 0.4 percentage points more likely to receive disability pension than mothers with a non-*APOE-e4* carrier child. These effects are about half the size of the effect of educational attainment PGS on disability and one-quarter the size of the effect of educational attainment PGS on employment. Mothers aged 55-65 are 0.7 percentage points less likely to be employed and 0.8 percentage points more likely to receive disability pension. These effects are about three-quarters the size of the effect of educational attainment PGS on disability and one-half the size of the effect of educational attainment PGS on employment. As shown earlier, accounting for measurement error roughly doubles the estimated effects of carrier status.

Our hypothesis was that individuals with a higher genetic risk of AD would have a lower labor

supply due to either: (1) cognitive decline before retirement; (2) care-giving to family members with AD; or (3) increased desire for leisure due to private knowledge about increased AD risk. For women, the decrease in the probability of being employed with a higher genetic risk of AD is almost equal to the increase in disability pension take-up. The association between genetic risk for AD, medical care, and disability pension uptake suggests that health-related factors contribute to why women with a higher predisposition for AD leave the labor force.

For men, we find that a higher genetic risk for AD is not robustly associated with any labor market outcomes. This result underscores significant gender differences in how genetic predisposition for AD affects men and women. Additionally, our genetic measure of AD appears to be more predictive of women's health than men's health.

We then test the cognitive reserve theory by examining the relationship between the EA PGS and the AD PGS. Our findings show that the inclusion of the EA PGS does not affect the robustness of our results. Among women aged 55 to 65, a high EA PGS appears to moderate the impact of a high AD PGS on disability pension take-up, consistent with the cognitive reserve theory. For both men and women, a higher EA PGS is associated with greater employment, earnings, and wealth, as well as lower disability pension uptake near retirement age.

This paper aims to understand the broader costs associated with AD. While AD is among the most expensive diseases globally, most cost estimates focus on the direct costs of formal care and the indirect costs of unpaid caregiving by family and friends. According to Alzheimer's Association (2024), AD is projected to cost the U.S. government $360 billion in 2024, while unpaid care provided by family and friends was valued at $347 billion in 2023. These costs are expected to rise further. Our findings suggest that the economic impact of AD may be underestimated, as we show that women with a higher genetic risk for AD experience worse labor market outcomes well before reaching retirement age.

With rising longevity and declining fertility rates, many countries are raising retirement ages. This makes it increasingly important to understand how genetic risk for AD affects individuals' lives even before an official diagnosis. As more people are expected to work later in life—and given that age is the greatest risk factor for AD—cognitive decline may prevent a growing number of individuals from remaining in the workforce until retirement.

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A Appendix

A.1 Bias Correction for APOE Regressions

We derive our bias correction using a simple bivariate regression:

$$y = \alpha + \beta D^* + u$$

where $D^* \in \{0,1\}$ denotes the carrier status of the parent. Unity indicates that the parent is either a heterogeneous or a homogeneous carrier. We do not consider X's in the regression for simplicity and because the R^2 of the short regression of D^* onto X for any sensible set of controls is small. The econometrician only observes $D \in \{0,1\}$ which is an indicator for the carrier status of a child. As such, they estimate

$$y = \widetilde{\alpha} + \widetilde{\beta} D + \widetilde{u}$$

We can write the coefficient on D as

$$\widetilde{\beta} = \beta \frac{C(D,D^*)}{V(D)}$$

To derive the bias, we note that

$$\begin{aligned} C(D,D^*) &= E\left[DD^*\right] - \pi^2 \\ &= P(D=1|D^*=1)\pi - \pi^2 \\ &= \pi\left(P(D=1|D^*=1) - \pi\right) \end{aligned}$$

where $\pi = E[D] = E[D^*]$. We have implicitly assumed that the distribution of carrier status is stationary across generations. Putting this all together, we show that

$$\beta = \widetilde{\beta} \times \frac{1-\pi}{P(D=1|D^*=1) - \pi} \tag{2}$$

From the data, we know that $\pi = 0.31$. Therefore, all that remains is to compute $P(D=1|D^*=1)$.

In the absence of a full intergenerational sample containing the *APOE-e4* carrier status of two generations, we can compute $P(D=1|D^*=1)$ using known probabilities and a few mild assumptions concerning assortative mating. Before we proceed, we let $D_2 \in \{0,1\}$ denote an indicator variable for the individual being a homogeneous carrier. Likewise, we define D_1 and D_0 to be indicator variables for

being a heterogeneous carrier and a non-carrier. We know from the data that

$$P(D_0) = 0.69$$

$$P(D_1) = 0.28$$

$$P(D_2) = 0.03$$

As before, the $*$-superscript denotes the parent generation. Next, we note that

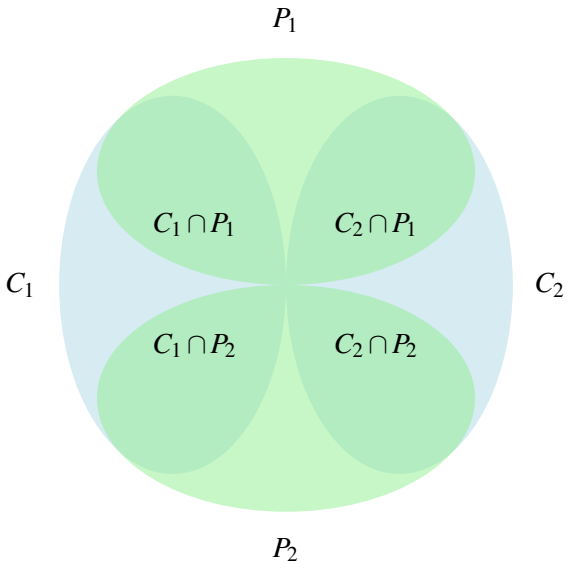
$$D = 1 \Leftrightarrow D_1 = 1 \cup D_2 = 1$$

and that $\{D_1 = 1\} \cap \{D_2 = 1\} = \emptyset$. We can write

$$P(D = 1|D^* = 1) = \frac{P((D_1 = 1 \cup D_2 = 1) \cap (D_1^* = 1 \cup D_2^* = 1))}{\pi} \tag{3}$$

To compute the probability in the numerator, we refer to the Venn diagram in Figure A.1 which shows the intersection of $C_1 \cup C_2$ with $P_1 \cup P_2$ where $C_1 \equiv \{D_1 = 1\}$, $C_2 \equiv \{D_2 = 1\}$, $P_1 \equiv \{D_1^* = 1\}$, and $P_2 \equiv \{D_2^* = 1\}$. Accordingly, we can write the conditional probability in equation (3) as

Figure A.1: Venn Diagram



$$P(D = 1|D^* = 1) = \frac{P(C_1 \cap P_1) + P(C_1 \cap P_2) + P(C_2 \cap P_1) + P(C_2 \cap P_2)}{\pi}$$

Now, we compute the four probabilities in the numerator. To illustrate, we carefully walk through the

logic for the first probability. We write

$$P(C_1 \cap P_1) = P(C_1|P_1)\,P(P_1)$$

To compute the conditional probability on the right, we introduce the notation that $\widetilde{P}_j$ for $j \in \{0,1,2\}$ is the carrier status of the parent's spouse. We will then have that

$$\begin{aligned} P(C_1|P_1) &= \sum_{j\in\{0,1,2\}} P\left(C_1|\widetilde{P}_j, P_1\right) P(\widetilde{P}_j) \\ &= 0.5 \times (P(\widetilde{P}_0) + P(\widetilde{P}_1) + P(\widetilde{P}_2)) \\ &= 0.5 \end{aligned}$$

Implicitly, we have assumed that there is no assortative mating based on carrier status. Therefore, we obtain that

$$P(C_1 \cap P_1) = 0.5 \times 0.28 = 0.14$$

We can proceed in a similar fashion for the three remaining conditional probabilities obtaining:

$$\begin{aligned} P(C_1 \mid P_2) &= P(\widetilde{P}_0) + 0.5 \times P(\widetilde{P}_1) = 0.69 + 0.5 \times 0.28 = 0.83 \\ P(C_2 \mid P_1) &= 0.25 \times P(\widetilde{P}_1) + 0.5 \times P(\widetilde{P}_2) = 0.25 \times 0.28 + 0.5 \times 0.03 = 0.085 \\ P(C_2 \mid P_2) &= 0.5 \times P(\widetilde{P}_1) + P(\widetilde{P}_2) = 0.5 \times 0.28 + 0.03 = 0.17 \end{aligned}$$

Therefore, we obtain that

$$P(D = 1|D^* = 1) = \frac{0.14 + 0.83 \times 0.03 + 0.085 \times 0.28 + 0.17 \times 0.03}{0.31} = 0.6252$$

Therefore, the bias correction in equation (2) can be computed as

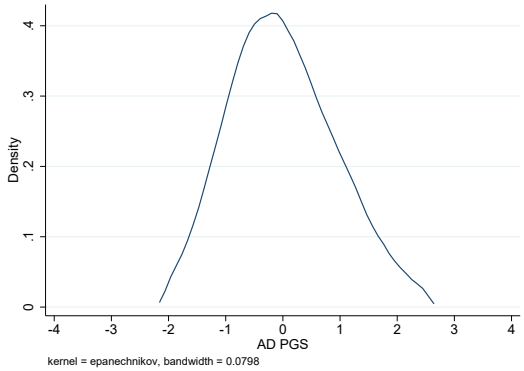
$$\frac{1-\pi}{P(D=1|D^*=1)-\pi} = \frac{1-0.31}{0.6252-0.31} = 2.19$$

This implies that we need to increase our coefficient estimates by a factor of 2.19 to account for measurement error in the *APOE-e4* carrier status.

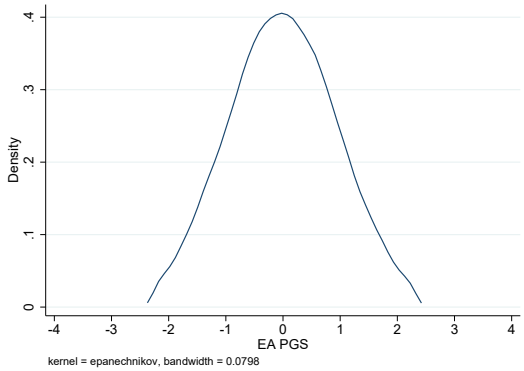
A.2 Figures

Figure A.2: Distribution of Polygenic Scores

(a) AD PGS



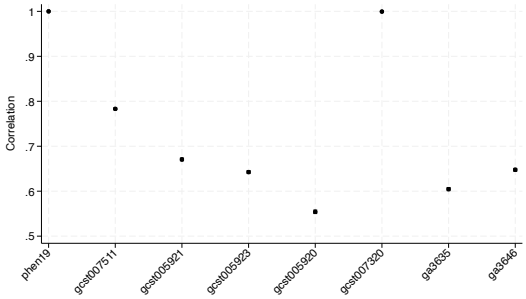
(b) EA PGS



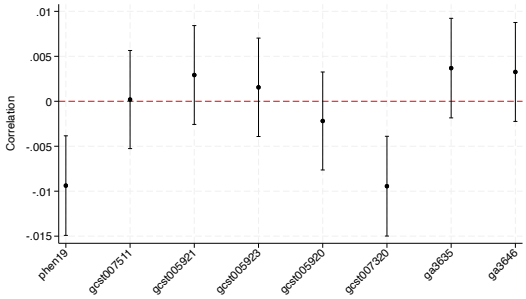
Notes: Displays the smoothed density of the AD PGS (phen19) and EA PGS (phen62). The 1^{st} and 99^{th} percentile of the polygenic scores are excluded in the figure.

Figure A.3: Polygenic Scores Correlation

(a) AD PGS



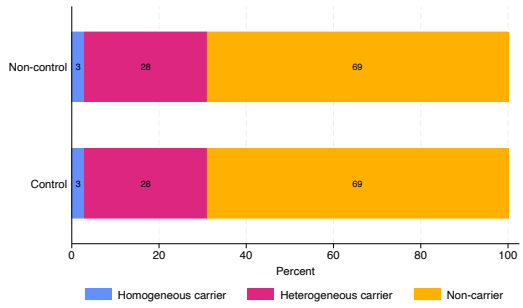
(b) EA PGS



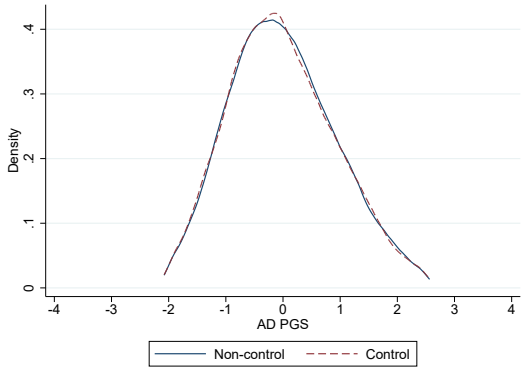
Notes: Panel (a) displays the correlation between the primary AD PGS (phen19) and all possible AD PGSs in the sample. Panel (b) displays the correlation between the EA PGS (phen62) and all possible AD PGSs in our sample. Bootstrapped standard errors using 500 replications, 95% confidence intervals are displayed.

Figure A.4: Correlation between Genetic Risk of AD and iPSYCH Control Group Indicator

(a) *APOE-e4* Carrier Status



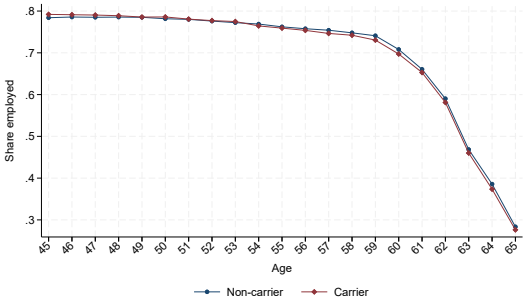
(b) AD PGS



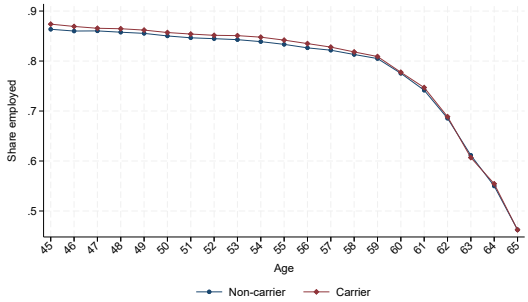
Notes: Panel (a) displays *APOE-e4* carrier status (homogeneous carrier, heterogeneous carrier, and non-carrier) by the iPSYCH control group indicator. Panel (b) displays the smoothed density of the AD PGS (phen19) by the iPSYCH control group indicator. Epanechnikov kernel is used. The 1^{st} and 99^{th} percentile of the polygenic score are excluded in the figure.

Figure A.5: *APOE-e4* Carrier Status and Labor Market Outcomes

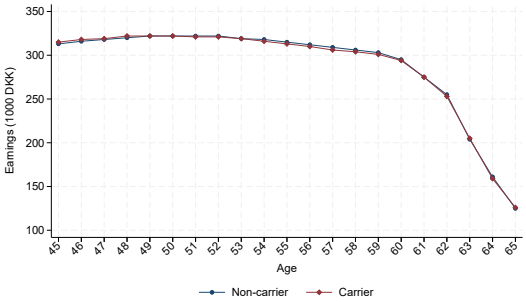
(a) Women: Employment



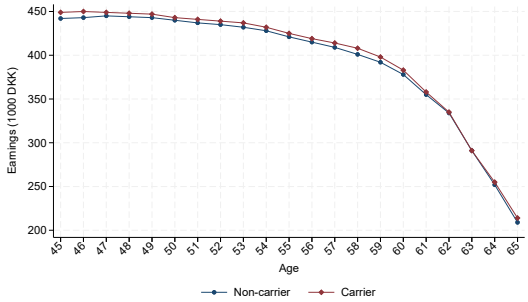
(b) Men: Employment



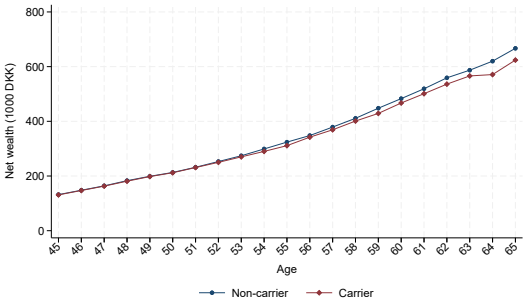
(c) Women: Earnings



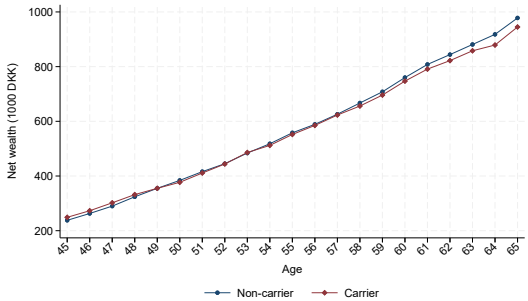
(d) Men: Earnings



(e) Women: Wealth



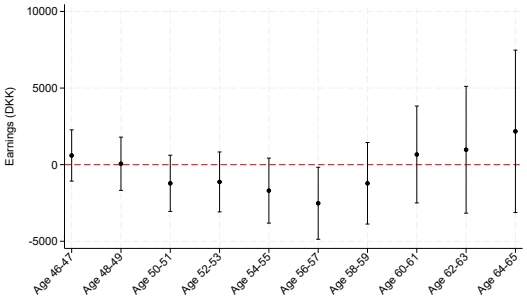
(f) Men: Wealth



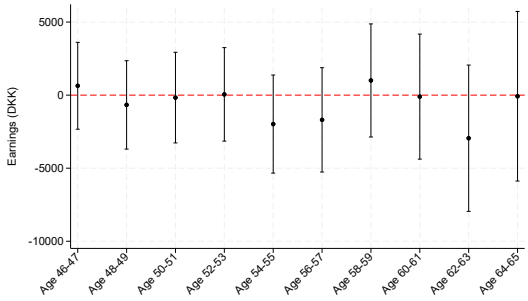
Notes: Displays share employed, earnings, and net wealth (excluding pension wealth) for women and men aged 45 to 65 by their child's *APOE-e4* carrier status. Earnings and wealth are measured in 1,000 DKK (2023-prices).

Figure A.6: *APOE-e4* Carrier Status and Earnings by Age

(a) Women



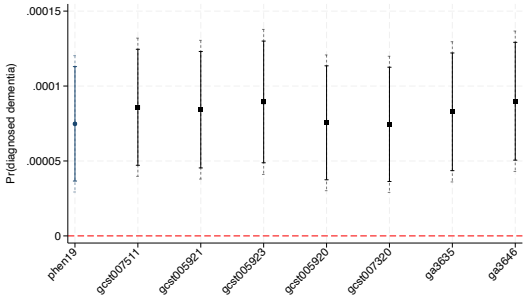
(b) Men



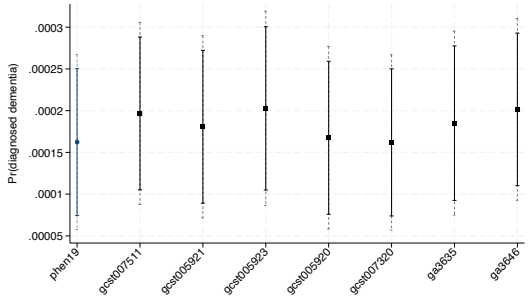
Notes: Displays estimated coefficients of child's *APOE-e4* carrier status on earnings by age for women in panel (a) and men in panel (b). Control variables included are age fixed effects, year fixed effects, the first 10 principal components, education dummies, marriage dummy, experience, experience squared, and dummy for whether child is in iPSYCH control group or not. Earnings are measured in DKK (2023-prices). Standard errors are clustered at the individual level, and 90% confidence intervals are displayed.

Figure A.7: AD PGSs and Health for Women

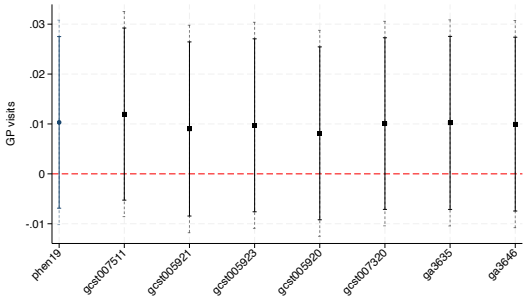
(a) Women aged 45-65: Dementia



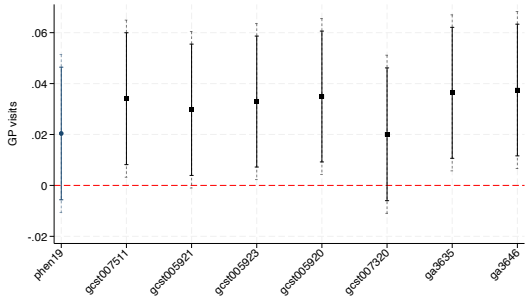
(b) Women aged 55-65: Dementia



(c) Women aged 45-65: GP visits



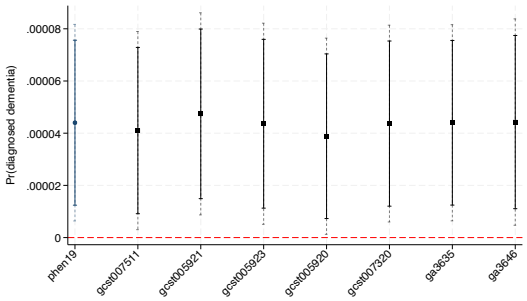
(d) Women aged 55-65: GP visits



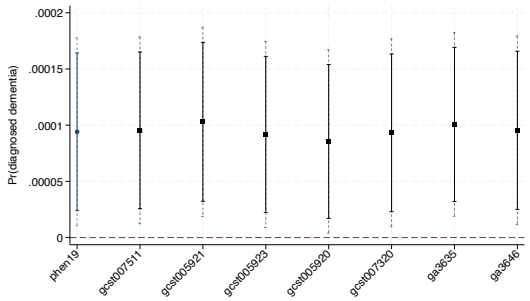
Notes: Panel (a) displays the coefficients of child's AD PGSs on the probability of diagnosed dementia for women aged 45 to 65, panel (b) displays the coefficients of child's AD PGSs on the probability of diagnosed dementia for women aged 55 to 65, panel (c) displays the coefficients of child's AD PGSs on GP visits for women aged 45 to 65, and panel (d) displays the coefficients of child's AD PGSs on GP visits for women aged 55 to 65. Control variables included are age fixed effects, year fixed effects, the first 10 principal components, education dummies, marriage dummy, experience, experience squared, and dummy for whether child is in iPSYCH control group or not. Standard errors are clustered at the individual level. Solid lines display 90% confidence intervals, and dashed lines display 95% confidence intervals.

Figure A.8: AD PGSs and Health for Men

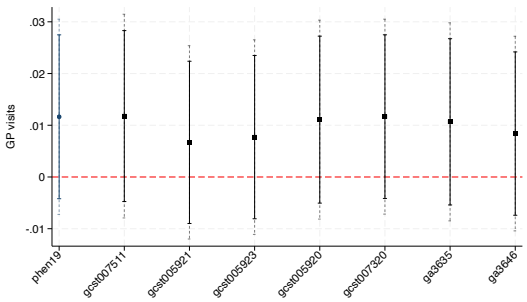
(a) Men aged 45-65: Dementia



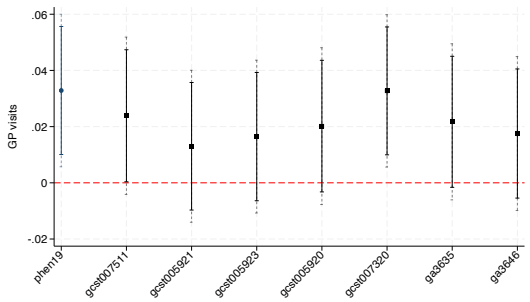
(b) Men aged 55-65: Dementia



(c) Men aged 45-65: GP visits



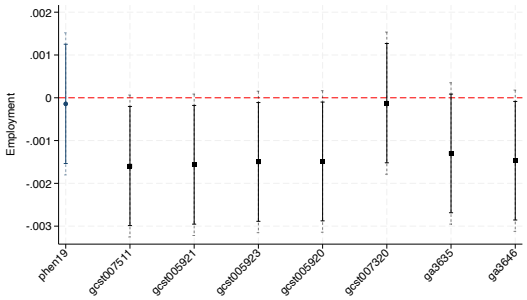
(d) Men aged 55-65: GP visits



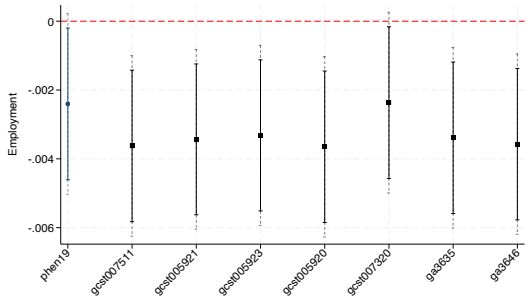
Notes: Panel (a) displays the coefficients of child's AD PGSs on the probability of diagnosed dementia for men aged 45 to 65, panel (b) displays the coefficients of child's AD PGSs on the probability of diagnosed dementia for men aged 55 to 65, panel (c) displays the coefficients of child's AD PGSs on GP visits for men aged 45 to 65, and panel (d) displays the coefficients of child's AD PGSs on GP visits for men aged 55 to 65. Control variables included are age fixed effects, year fixed effects, the first 10 principal components, education dummies, marriage dummy, experience, experience squared, and dummy for whether child is in iPSYCH control group or not. Standard errors are clustered at the individual level. Solid lines display 90% confidence intervals, and dashed lines display 95% confidence intervals.

Figure A.9: AD PGSs and Labor Market Attachment for Women

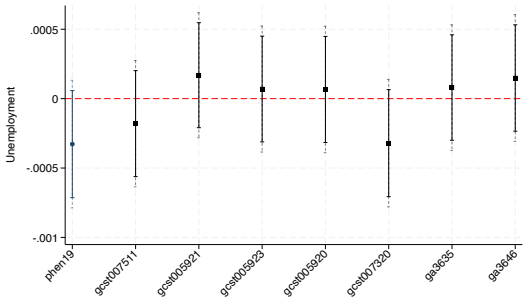
(a) Women aged 45-65: Employment



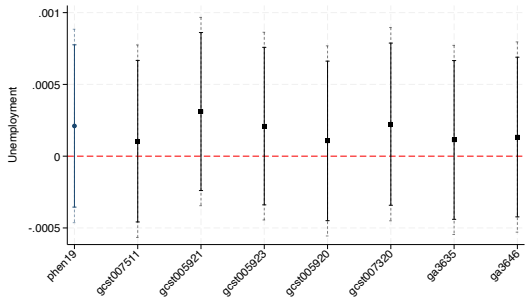
(b) Women aged 55-65: Employment



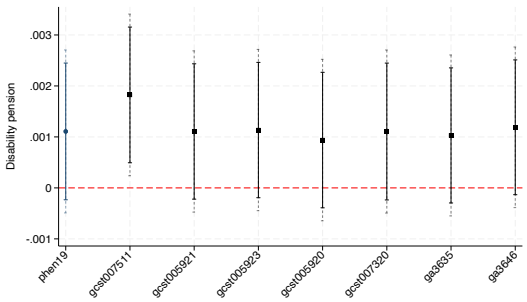
(c) Women aged 45-65: Unemployment



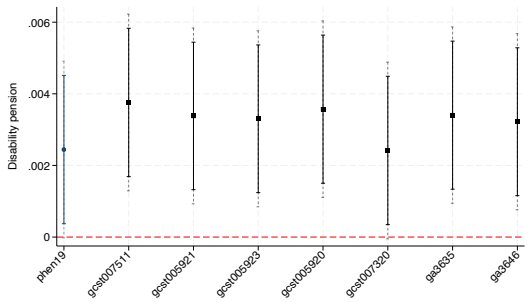
(d) Women aged 55-65: Unemployment



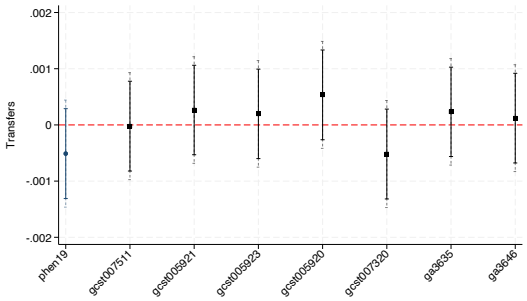
(e) Women aged 45-65: Disability pension



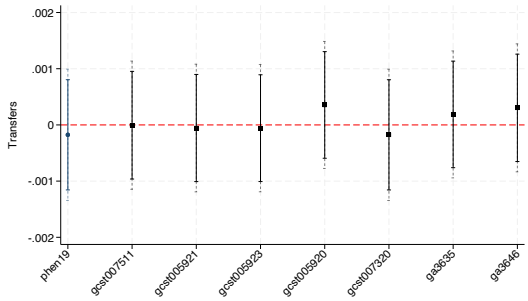
(f) Women aged 55-65: Disability pension



(g) Women aged 45-65: Transfers



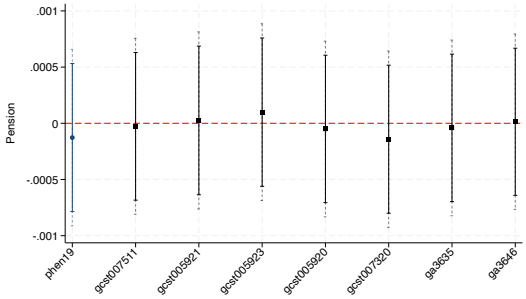
(h) Women aged 55-65: Transfers



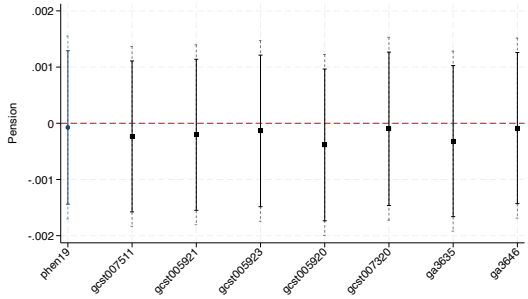
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Figure A.9: AD PGSs and Labor Market Attachment for Women cont'd

(i) Women aged 45-65: Pension



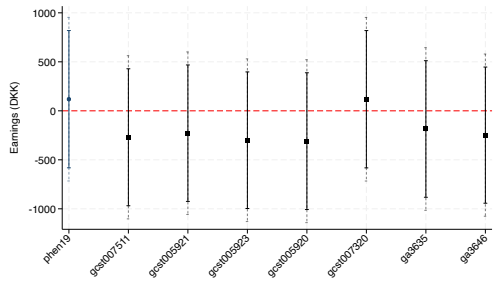
(j) Women aged 55-65: Pension



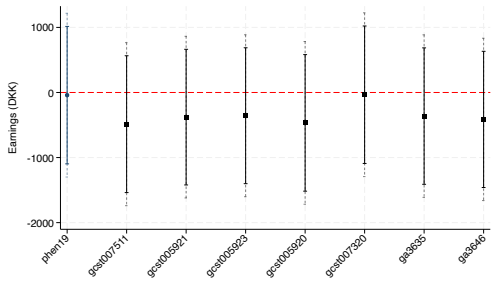
Notes: Displays the coefficients of child's AD PGSs on employment for women aged 45-65 in panel (a), employment for women aged 55-65 in panel (b), unemployment for women aged 45-65 in panel (c), unemployment for women aged 55-65 in panel (d), disability pension for women aged 45-65 in panel (e), disability pension for women aged 55-65 in panel (f), transfers for women aged 45-65 in panel (g), transfers for women aged 55-65 in panel (h), pension for women aged 45-65 in panel (i), and pension for women aged 55-65 in panel (j). Control variables included are age fixed effects, year fixed effects, the first 10 principal components, education dummies, marriage dummy, experience, experience squared, and dummy for whether child is in iPSYCH control group or not. Standard errors are clustered at the individual level. Solid lines display 90% confidence intervals, and dashed lines display 95% confidence intervals.

Figure A.10: AD PGSs, Income, and Wealth for Women

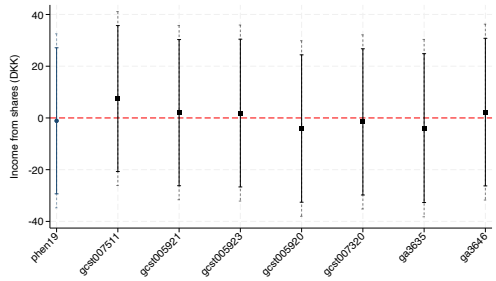
(a) Women aged 45-65: Earnings



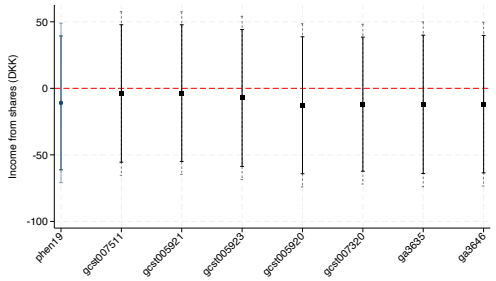
(b) Women aged 55-65: Earnings



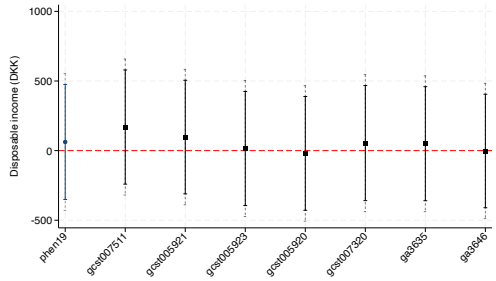
(c) Women aged 45-65: Income from shares



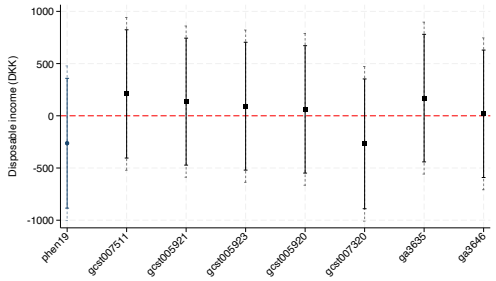
(d) Women aged 55-65: Income from shares



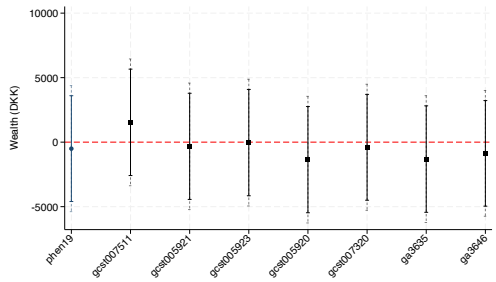
(e) Women aged 45-65: Disposable income



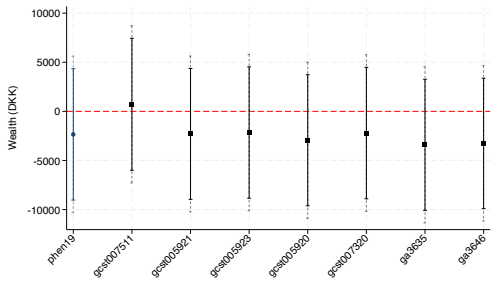
(f) Women aged 55-65: Disposable income



(g) Women aged 45-65: Wealth



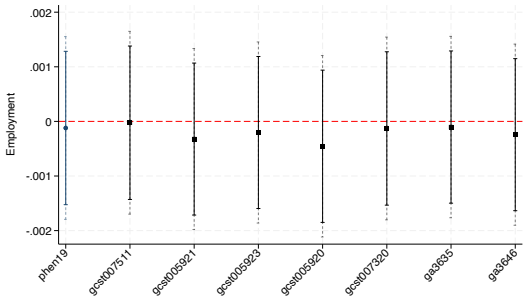
(h) Women aged 55-65: Wealth



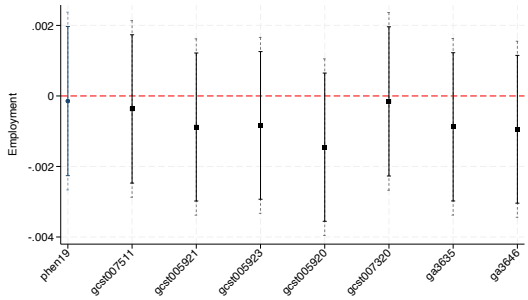
Notes: Displays the coefficients of child's AD PGSs on earnings for women aged 45-65 in panel (a), earnings for women aged 55-65 in panel (b), income from shares for women aged 45-65 in panel (c), income from shares for women aged 55-65 in panel (d), disposable income for women aged 45-65 in panel (e), disposable income for women aged 55-65 in panel (f), net wealth (excluding pension wealth) for women aged 45-65 in panel (g), and net wealth (excluding pension wealth) for women aged 55-65 in panel (h). Control variables included are age fixed effects, year fixed effects, the first 10 principal components, education dummies, marriage dummy, experience, experience squared, and dummy for whether child is in iPSYCH control group or not. All monetary values are measured in DKK (2023-prices). Standard errors are clustered at the individual level. Solid lines display 90% confidence intervals, and dashed lines display 95% confidence intervals.

Figure A.11: AD PGSs and Labor Market Attachment for Men

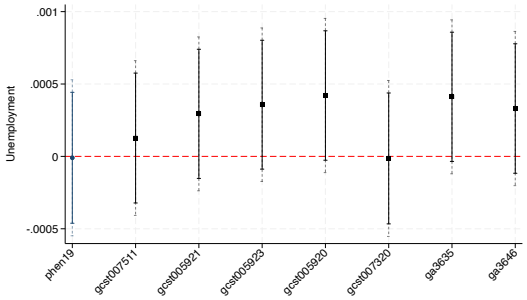
(a) Men aged 45-65: Employment



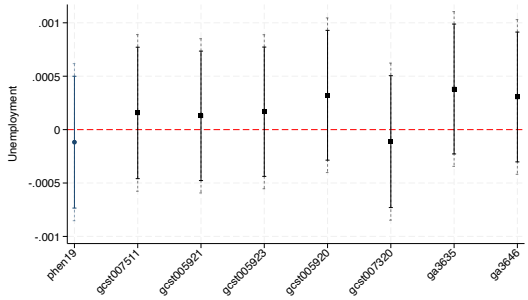
(b) Men aged 55-65: Employment



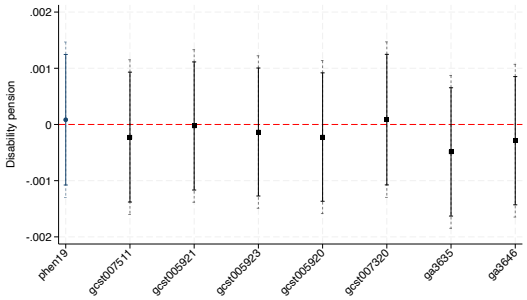
(c) Men aged 45-65: Unemployment



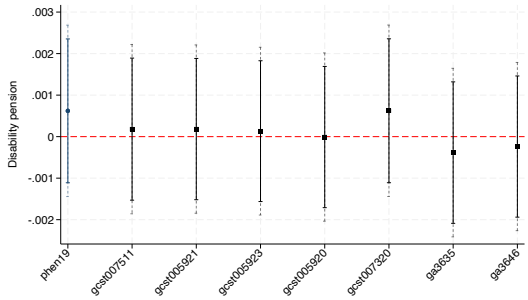
(d) Men aged 55-65: Unemployment



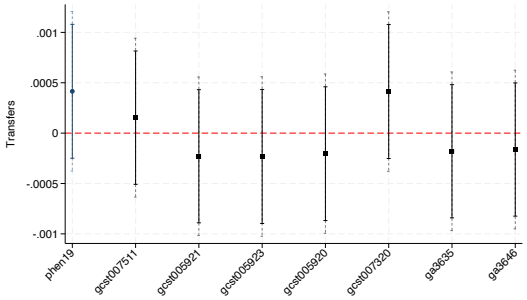
(e) Men aged 45-65: Disability pension



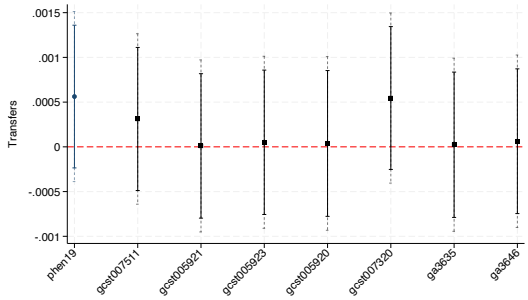
(f) Men aged 55-65: Disability pension



(g) Men aged 45-65: Transfers



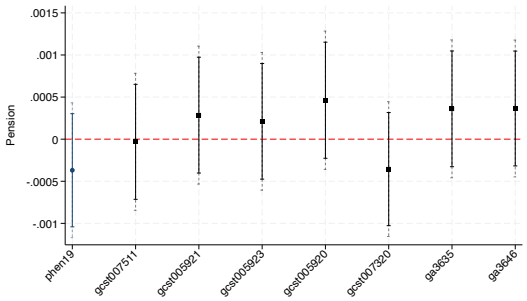
(h) Men aged 55-65: Transfers



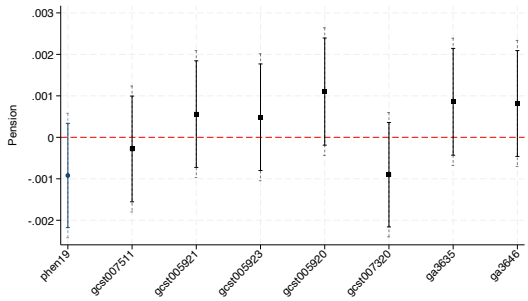
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Figure A.11: AD PGSs and Labor Market Attachment for Men cont'd

(i) Men aged 45-65: Pension



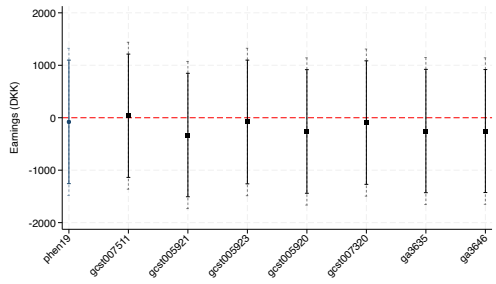
(j) Men aged 55-65: Pension



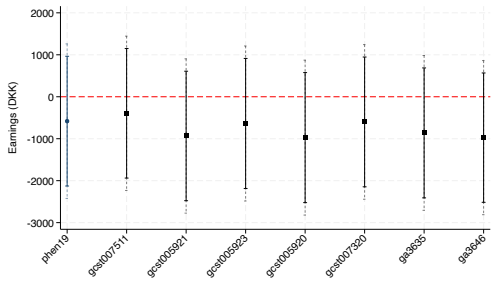
Notes: Displays the coefficients of child's AD PGSs on employment for men aged 45-65 in panel (a), employment for men aged 55-65 in panel (b), unemployment for men aged 45-65 in panel (c), unemployment for men aged 55-65 in panel (d), disability pension for men aged 45-65 in panel (e), disability pension for men aged 55-65 in panel (f), transfers for men aged 45-65 in panel (g), transfers for men aged 55-65 in panel (h), pension for men aged 45-65 in panel (i), and pension for men aged 55-65 in panel (j). Control variables included are age fixed effects, year fixed effects, the first 10 principal components, education dummies, marriage dummy, experience, experience squared, and dummy for whether child is in iPSYCH control group or not. Standard errors are clustered at the individual level. Solid lines display 90% confidence intervals, and dashed lines display 95% confidence intervals.

Figure A.12: AD PGSs, Income, and Wealth for Men

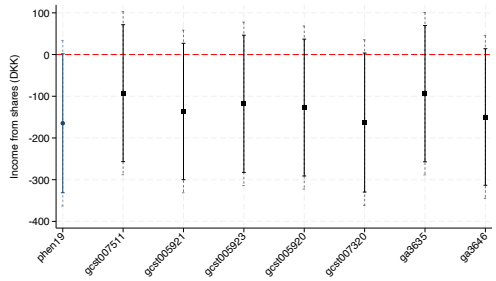
(a) Men aged 45-65: Earnings



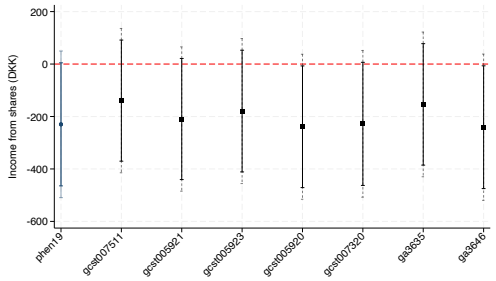
(b) Men aged 55-65: Earnings



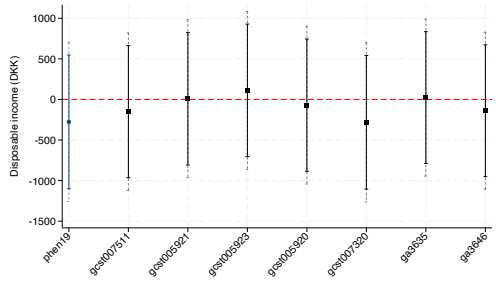
(c) Men aged 45-65: Income from shares



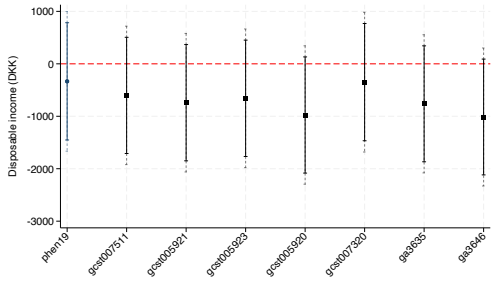
(d) Men aged 55-65: Income from shares



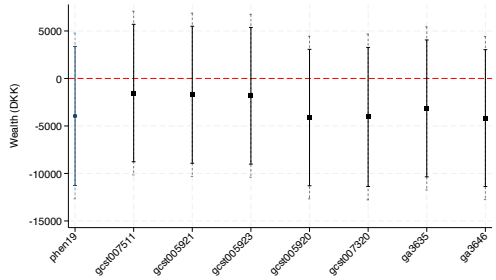
(e) Men aged 45-65: Disposable income



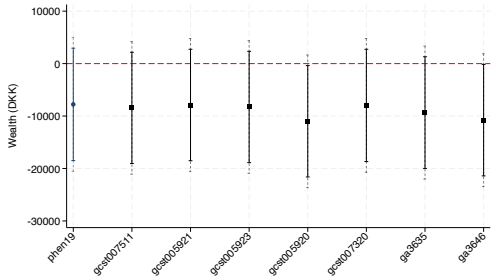
(f) Men aged 55-65: Disposable income



(g) Men aged 45-65: Wealth



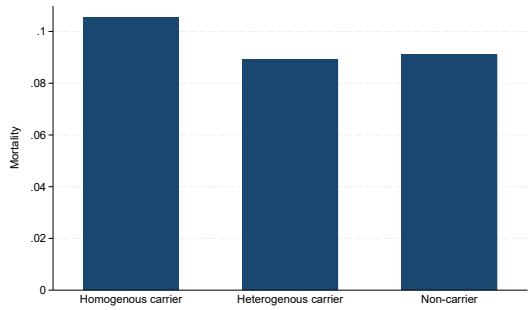
(h) Men aged 55-65: Wealth



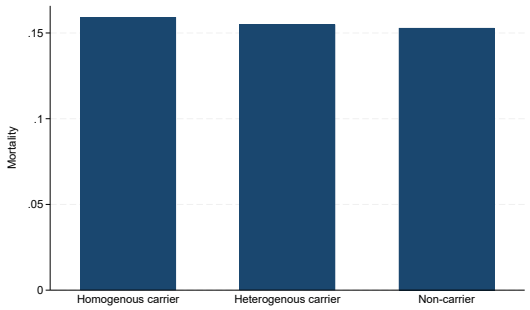
Notes: Displays the coefficients of child's AD PGSs on earnings for men aged 45-65 in panel (a), earnings for men aged 55-65 in panel (b), income from shares for men aged 45-65 in panel (c), income from shares for men aged 55-65 in panel (d), disposable income for men aged 45-65 in panel (e), disposable income for men aged 55-65 in panel (f), net wealth (excluding pension wealth) for men aged 45-65 in panel (g), and net wealth (excluding pension wealth) for men aged 55-65 in panel (h). Control variables included are age fixed effects, year fixed effects, the first 10 principal components, education dummies, marriage dummy, experience, experience squared, and dummy for whether child is in iPSYCH control group or not. All monetary values are measured in DKK (2023-prices). Standard errors are clustered at the individual level. Solid lines display 90% confidence intervals, and dashed lines display 95% confidence intervals.

Figure A.13: Mortality by *APOE-e4* Carrier Status

(a) Women



(b) Men



Notes: Displays the proportion of deceased by age 65 for women in panel (a) and men in panel (b) for years 2005-2020 by child's *APOE-e4* carrier status.

A.3 Tables

Table A.1: External Validity for Women aged 45 to 65

	Non-control iPSYCH		Control iPSYCH		Full sample	
	Mean	SD	Mean	SD	Mean	SD
Homogeneous carrier	0.03	0.17	0.03	0.17		
Heterogeneous carrier	0.28	0.45	0.28	0.45		
Age	52.49	5.21	52.36	5.23	54.70	6.04
Married	0.58	0.49	0.68	0.47	0.63	0.48
Lower sec., primary, unknown	0.26	0.44	0.20	0.40	0.27	0.44
General upper secondary	0.04	0.19	0.05	0.21	0.04	0.20
Vocational education	0.37	0.48	0.37	0.48	0.36	0.48
Short cycle tertiary	0.04	0.19	0.04	0.20	0.04	0.19
Bachelor	0.24	0.43	0.26	0.44	0.22	0.42
Master, doctoral	0.06	0.23	0.08	0.27	0.07	0.25
Experience	19.34	9.67	21.39	9.10	22.32	11.21
Year	2013.70	4.32	2013.84	4.32	2012.61	4.60
GP visits	4.57	4.90	3.84	4.23		
Dementia	0.00	0.01	0.00	0.01		
Employment	0.71	0.45	0.81	0.39	0.69	0.46
Unemployment	0.03	0.16	0.02	0.14	0.02	0.15
Disability pension	0.15	0.35	0.08	0.27	0.11	0.32
Other transfers	0.07	0.26	0.04	0.20	0.05	0.21
Pension	0.04	0.21	0.05	0.21	0.12	0.33
Earnings (DKK)	292,010.68	229,977.88	344,701.54	227,494.00	285,410.06	245,303.08
Income from shares (DKK)	1,195.47	6,904.01	1,771.76	8,487.45	2,381.00	14,367.88
Disp. income (DKK)	279,159.42	112,882.48	305,118.59	122,893.90	278,360.80	133,767.02
Wealth (DKK)	231,752.21	827,754.50	365,177.22	943,720.81	447,951.82	1,101,523.63
$N \times$ *Years*	742,446		381,039		12,455,780	
N	69,428		36,946		1,368,065	

Notes: Displays means and standard deviations for the genetic measure, control variables, and dependent variables for women aged 45 to 65 whose child is in the iPSYCH non-control group (columns (1)-(2)), women aged 45 to 65 whose child is in the iPSYCH control group (columns (3)-(4)), and all women aged 45 to 65 (columns (5)-(6)) for the years 2005-2020 (2008-2020 for labor market attachment). All monetary values are measured in DKK (2023-prices). *N* refers to the number of individuals, and $N \times$ *Years* refers to the total number of individual-year observations.

Table A.2: External Validity for Men aged 45 to 65

	Non-control iPSYCH		Control iPSYCH		Full sample	
	Mean	SD	Mean	SD	Mean	SD
Homogeneous carrier	0.03	0.17	0.03	0.17		
Heterogeneous carrier	0.28	0.45	0.28	0.45		
Age	53.49	5.49	53.23	5.50	54.62	6.03
Married	0.63	0.48	0.73	0.45	0.63	0.48
Lower sec., primary, unknown	0.28	0.45	0.21	0.41	0.26	0.44
General upper secondary	0.04	0.20	0.04	0.21	0.05	0.21
Vocational education	0.43	0.50	0.44	0.50	0.43	0.49
Short cycle tertiary	0.05	0.21	0.05	0.22	0.05	0.21
Bachelor	0.12	0.33	0.15	0.35	0.13	0.33
Master, doctoral	0.08	0.27	0.10	0.30	0.09	0.29
Experience	22.97	9.72	23.75	9.50	24.70	11.86
Year	2013.15	4.42	2013.37	4.41	2012.59	4.60
GP visits	3.17	4.38	2.76	3.91		
Dementia	0.00	0.01	0.00	0.01		
Employment	0.79	0.41	0.86	0.35	0.75	0.43
Unemployment	0.03	0.17	0.02	0.15	0.03	0.17
Disability pension	0.09	0.28	0.05	0.21	0.09	0.28
Other transfers	0.04	0.21	0.03	0.16	0.04	0.19
Pension	0.05	0.22	0.05	0.21	0.09	0.29
Earnings (DKK)	398,334.58	321,121.69	456,741.95	344,797.06	372,644.54	322,957.84
Income from shares (DKK)	7,184.10	35,351.97	10,864.63	43,601.21	7,027.76	27,564.09
Disp. income (DKK)	340,030.22	196,127.33	382,403.17	228,993.08	337,889.93	193,176.94
Wealth (DKK)	431,286.82	1,391,349.50	648,109.24	1,664,962.38	695,922.51	1,526,708.13
$N \times$ *Years*	705,205		379,291		12,498,357	
N	64,307		35,686		1,395,471	

Notes: Displays means and standard deviations for the genetic measure, control variables, and dependent variables for men aged 45 to 65 whose child is in the iPSYCH non-control group (columns (1)-(2)), men aged 45 to 65 whose child is in the iPSYCH control group (columns (3)-(4)), and all men aged 45 to 65 (columns (5)-(6)) for the years 2005-2020 (2008-2020 for labor market attachment). All monetary values are measured in DKK (2023-prices). *N* refers to the number of individuals, and $N \times$ *Years* refers to the total number of individual-year observations.

Table A.3: AD PGS, Educational Attainment, and Health

	Dementia		GP visits	
	Age 45-65	Age 55-65	Age 45-65	Age 55-65
	(1)	(2)	(3)	(4)
Panel A: Women				
AD PGS	0.00007***	0.00016***	0.00948	0.01999
	(0.00002)	(0.00005)	(0.01042)	(0.01594)
EA PGS	-0.00002	-0.00002	-0.14778***	-0.14878***
	(0.00002)	(0.00004)	(0.01104)	(0.01631)
AD PGS × EA PGS	0.00001	0.00001	-0.01097	-0.00637
	(0.00002)	(0.00004)	(0.01036)	(0.01522)
$N \times Years$	1,123,485	375,336	1,123,485	375,336
N	106,374	61,992	106,374	61,992
R^2	0.000	0.000	0.044	0.036
Mean	0.0002	0.0004	4.33	4.35
Pct. Change (AD)	43.35	43.41	0.22	0.46
Pct. Change (EA)	-10.68	-6.70	-3.42	-3.42
Pct. Change (AD × EA)	5.92	1.75	-0.25	-0.15
Panel B: Men				
AD PGS	0.00004**	0.00009**	0.01055	0.03048**
	(0.00002)	(0.00004)	(0.00966)	(0.01400)
EA PGS	-0.00003*	-0.00007**	-0.14048***	-0.16128***
	(0.00002)	(0.00004)	(0.00971)	(0.01424)
AD PGS × EA PGS	0.00000	-0.00000	0.01846*	0.01201
	(0.00002)	(0.00004)	(0.00956)	(0.01323)
$N \times Years$	1,084,496	440,274	1,084,496	440,274
N	99,993	66,369	99,993	66,369
R^2	0.000	0.000	0.032	0.029
Mean	0.0002	0.0003	3.03	3.43
Pct. Change (AD)	23.75	26.95	0.35	0.89
Pct. Change (EA)	-18.04	-20.65	-4.64	-4.71
Pct. Change (AD × EA)	1.17	-1.08	0.61	0.35

Notes: This table reports estimates based on Equation 1 using child's AD PGS as measure of genetic risk for women (Panel A) and men (Panel B), but additionally adding child's EA PGS and interactions of EA PGS and AD PGS. In columns (1) and (2), the outcome is a dummy for diagnosed dementia. In columns (3) and (4), the outcome variable is number of GP visits. Control variables included are age fixed effects, year fixed effects, the first 10 principal components, education dummies, marriage dummy, experience, experience squared, and dummy for whether child is in iPSYCH control group or not. N refers to the number of individuals, and $N \times Years$ refers to the total number of individual-year observations. Standard errors are clustered at the individual level and shown in parentheses. Significance levels are denoted as * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table A.4: AD PGS, Educational Attainment, and Economic Outcomes for Women

	Employment	DP	Earnings	Wealth
	(1)	(2)	(3)	(4)
Panel A: Age 45-65				
AD PGS	-0.00007	0.00105	145.11	-172.29
	(0.00085)	(0.00081)	(425.78)	(2485.53)
EA PGS	0.01258***	-0.00880***	5797.61***	74625.07***
	(0.00090)	(0.00086)	(451.70)	(2657.97)
AD PGS × EA PGS	0.00074	-0.00094	-260.52	-5077.50**
	(0.00084)	(0.00082)	(430.58)	(2520.29)
$N \times$ *Years*	1,005,275	1,005,275	1,229,258	1,229,258
N	105,896	105,896	106,735	106,735
R^2	0.365	0.308	0.507	0.106
Mean	0.75	0.12	308,707.80	274,620.24
Pct. Change (AD)	-0.01	0.85	0.05	-0.06
Pct. Change (EA)	1.69	-7.11	1.88	27.17
Pct. Change (AD × EA)	0.10	-0.76	-0.08	-1.85
Panel B: Age 55-65				
AD PGS	-0.00242*	0.00251**	19.11	-1397.70
	(0.00134)	(0.00127)	(635.63)	(3994.09)
EA PGS	0.01565***	-0.01247***	6132.96***	91073.47***
	(0.00143)	(0.00132)	(678.21)	(4320.07)
AD PGS × EA PGS	0.00172	-0.00259**	-439.16	-7529.19*
	(0.00132)	(0.00125)	(651.11)	(4061.54)
$N \times$ *Years*	360,716	360,716	382,779	382,779
N	61,814	61,814	62,063	62,063
R^2	0.347	0.336	0.500	0.098
Mean	0.68	0.16	284,489.22	432,197.07
Pct. Change (AD)	-0.35	1.59	0.01	-0.32
Pct. Change (EA)	2.29	-7.92	2.16	21.07
Pct. Change (AD × EA)	0.25	-1.65	-0.15	-1.74

Notes: This table reports estimates based on Equation 1 using child's AD PGS as measure of genetic risk for women aged 45-65 (Panel A) and women aged 55-65 (Panel B), but additionally adding child's EA PGS and interactions of EA PGS and AD PGS. In column (1), the outcome is a dummy for employment, in column (2), the outcome is a dummy for receiving disability pension, in column (3), the outcome is earnings, and in column (4), the outcome is net wealth excluding pension wealth. All monetary values are in DKK (2023-prices). Control variables included are age fixed effects, year fixed effects, the first 10 principal components, education dummies, marriage dummy, experience, experience squared, and dummy for whether child is in iPSYCH control group or not. N refers to the number of individuals, and $N \times$ *Years* refers to the total number of individual-year observations. Standard errors are clustered at the individual level and shown in parentheses. Significance levels are denoted as * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table A.5: AD PGS, Educational Attainment, and Economic Outcomes for Men

	Employment	DP	Earnings	Wealth
	(1)	(2)	(3)	(4)
Panel A: Age 45-65				
AD PGS	-0.00004	0.00003	4.89	-2904.04
	(0.00085)	(0.00071)	(711.41)	(4405.20)
EA PGS	0.01725***	-0.01161***	11489.61***	126576.65***
	(0.00091)	(0.00076)	(750.47)	(4745.25)
AD PGS × EA PGS	0.00084	-0.00026	-488.67	-9863.85**
	(0.00086)	(0.00074)	(734.47)	(4530.52)
$N \times$ *Years*	940,718	940,718	1,243,045	1,243,045
N	98,560	98,560	101,228	101,228
R^2	0.252	0.189	0.397	0.082
Mean	0.81	0.07	415,563.64	496,335.66
Pct. Change (AD)	-0.01	0.04	0.00	-0.59
Pct. Change (EA)	2.13	-16.11	2.76	25.50
Pct. Change (AD × EA)	0.10	-0.36	-0.12	-1.99
Panel B: Age 55-65				
AD PGS	0.00001	0.00046	-428.16	-5367.64
	(0.00129)	(0.00107)	(927.20)	(6400.58)
EA PGS	0.01894***	-0.01464***	10491.56***	153826.88***
	(0.00136)	(0.00111)	(990.24)	(6911.36)
AD PGS × EA PGS	0.00070	0.00016	-840.69	-14593.51**
	(0.00129)	(0.00108)	(959.49)	(6486.44)
$N \times$ *Years*	407,980	407,980	465,012	465,012
N	65,436	65,436	67,010	67,010
R^2	0.242	0.198	0.404	0.081
Mean	0.75	0.09	373,107.11	710,473.37
Pct. Change (AD)	0.00	0.49	-0.11	-0.76
Pct. Change (EA)	2.51	-15.44	2.81	21.65
Pct. Change (AD × EA)	0.09	0.17	-0.23	-2.05

Notes: This table reports estimates based on Equation 1 using child's AD PGS as measure of genetic risk for men aged 45-65 (Panel A) and men aged 55-65 (Panel B), but additionally adding child's EA PGS and interactions of EA PGS and AD PGS. In column (1), the outcome is a dummy for employment, in column (2), the outcome is a dummy for receiving disability pension, in column (3), the outcome is earnings, and in column (4), the outcome is net wealth excluding pension wealth. All monetary values are in DKK, 2023-prices. Control variables included are age fixed effects, year fixed effects, the first 10 principal components, education dummies, marriage dummy, experience, experience squared, and dummy for whether child is in iPSYCH control group or not. N refers to the number of individuals, and $N \times$ *Years* refers to the total number of individual-year observations. Standard errors are clustered at the individual level and shown in parentheses. Significance levels are denoted as * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table A.6: *APOE-e4* Carrier Status and Health Outcomes

	Dementia		GP visits	
	Age 45-65	Age 55-65	Age 45-65	Age 55-65
	(1)	(2)	(3)	(4)
Panel A: Women				
Homogeneous carrier	0.00054**	0.00127**	-0.01435	0.01884
	(0.00025)	(0.00057)	(0.06080)	(0.09216)
Heterogeneous carrier	0.00012***	0.00025***	0.04017*	0.10202***
	(0.00004)	(0.00010)	(0.02350)	(0.03528)
$N \times Years$	1,123,485	375,336	1,123,485	375,336
N	106,374	61,992	106,374	61,992
R^2	0.000	0.000	0.043	0.035
Mean	0.0002	0.0004	4.33	4.35
Pct. Change (Homo)	311.41	339.67	-0.33	0.43
Pct. Change (Hetero)	68.15	68.04	0.93	2.34
Panel B: Men				
Homogeneous carrier	0.00028*	0.00048	0.09893	0.11776
	(0.00015)	(0.00033)	(0.06254)	(0.09047)
Heterogeneous carrier	0.00006	0.00015*	0.01142	0.04469
	(0.00004)	(0.00008)	(0.02099)	(0.03059)
$N \times Years$	1,084,496	440,274	1,084,496	440,274
N	99,993	66,369	99,993	66,369
R^2	0.000	0.000	0.031	0.027
Mean	0.0002	0.0003	3.03	3.43
Pct. Change (Homo)	152.02	139.45	3.27	3.44
Pct. Change (Hetero)	33.06	43.84	0.38	1.30

Notes: This table reports estimates based on Equation 1 using child's *APOE-e4* carrier status (homogeneous or heterogeneous carrier) as measure of genetic risk for women (Panel A) and men (Panel B). In columns (1) and (2), the outcome is a dummy for diagnosed dementia. In columns (3) and (4), the outcome variable is number of GP visits. Control variables included are age fixed effects, year fixed effects, the first 10 principal components, education dummies, marriage dummy, experience, experience squared, and dummy for whether child is in iPSYCH control group or not. N refers to the number of individuals, and $N \times Years$ refers to the total number of individual-year observations. Standard errors are clustered at the individual level and shown in parentheses. Significance levels are denoted as * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table A.7: *APOE-e4* Carrier Status and Labor Market Attachment for Women

	Employment	Unemployment	DP	Transfers	Pension
	(1)	(2)	(3)	(4)	(5)
Panel A: Age 45-65					
Homogeneous carrier	-0.00384	0.00013	0.00191	0.00062	0.00118
	(0.00499)	(0.00140)	(0.00476)	(0.00281)	(0.00239)
Heterogeneous carrier	-0.00338*	-0.00036	0.00411**	0.00033	-0.00070
	(0.00190)	(0.00052)	(0.00183)	(0.00109)	(0.00090)
$N \times Years$	1,005,275	1,005,275	1,005,275	1,005,275	1,005,275
N	105,896	105,896	105,896	105,896	105,896
R^2	0.364	0.007	0.308	0.061	0.212
Mean	0.75	0.02	0.12	0.06	0.05
Pct. Change (Homo)	-0.51	0.54	1.54	1.01	2.58
Pct. Change (Hetero)	-0.45	-1.50	3.32	0.54	-1.54
Panel B: Age 55-65					
Homogeneous carrier	-0.00844	-0.00041	0.01155	-0.00027	-0.00242
	(0.00794)	(0.00196)	(0.00766)	(0.00349)	(0.00467)
Heterogeneous carrier	-0.00702**	0.00045	0.00728***	0.00017	-0.00088
	(0.00301)	(0.00077)	(0.00281)	(0.00130)	(0.00186)
$N \times Years$	360,716	360,716	360,716	360,716	360,716
N	61,814	61,814	61,814	61,814	61,814
R^2	0.346	0.007	0.335	0.037	0.253
Mean	0.68	0.02	0.16	0.04	0.10
Pct. Change (Homo)	-1.24	-1.89	7.33	-0.65	-2.51
Pct. Change (Hetero)	-1.03	2.08	4.62	0.40	-0.91

Notes: This table reports estimates based on Equation 1 using child's *APOE-e4* carrier status (homogeneous or heterogeneous carrier) as measure of genetic risk for women aged 45 to 65 (Panel A) and women aged 55 to 65 (Panel B). In column (1), the outcome is a dummy for being employed, in column (2), the outcome is a dummy for being unemployed, in column (3), the outcome is a dummy for receiving disability pension, in column (4), the outcome variable is a dummy for receiving transfers, and in column (5), the outcome variable is a dummy for pension. Control variables included are age fixed effects, year fixed effects, the first 10 principal components, education dummies, marriage dummy, experience, experience squared, and dummy for whether child is in iPSYCH control group or not. N refers to the number of individuals, and $N \times Years$ refers to the total number of individual-year observations. Standard errors are clustered at the individual level and shown in parentheses. Significance levels are denoted as * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table A.8: *APOE-e4* Carrier Status, Income, and Wealth for Women

	Earnings	Income from Shares	Disposable Income	Wealth
	(1)	(2)	(3)	(4)
Panel A: Age 45-65				
Homogeneous carrier	461.08	-9.31	944.31	15947.78
	(2491.32)	(98.42)	(1479.03)	(15250.93)
Heterogeneous carrier	-613.71	-27.58	-93.25	-8621.33
	(953.22)	(39.24)	(555.26)	(5558.24)
$N \times$ *Years*	1,229,258	1,229,258	1,229,258	1,229,258
N	106,735	106,735	106,735	106,735
R^2	0.507	0.024	0.391	0.099
Mean	308,707.80	1,313.37	285,150.85	274,620.24
Pct. Change (Homo)	0.15	-0.71	0.33	5.81
Pct. Change (Hetero)	-0.20	-2.10	-0.03	-3.14
Panel B: Age 55-65				
Homogeneous carrier	1,172.48	-63.19	2,142.81	13,305.45
	(3,738.90)	(179.53)	(2,154.38)	(24,669.63)
Heterogeneous carrier	-1,042.15	-51.79	-315.33	-16,360.04*
	(1,435.21)	(69.87)	(834.90)	(9,013.53)
$N \times$ *Years*	382,779	382,779	382,779	382,779
N	62,063	62,063	62,063	62,063
R^2	0.499	0.025	0.389	0.090
Mean	284,489.22	1,876.72	285,440.72	432,197.07
Pct. Change (Homo)	0.41	-3.37	0.75	3.08
Pct. Change (Hetero)	-0.37	-2.76	-0.11	-3.79

Notes: This table reports estimates based on Equation 1 using child's *APOE-e4* carrier status (homogeneous or heterogeneous carrier) as measure of genetic risk for women aged 45 to 65 (Panel A) and women aged 55 to 65 (Panel B). In column (1), the outcome is earnings, in column (2), the outcome is income from shares, in column (3), the outcome is disposable income, and in column (4), the outcome is net wealth excluding pension wealth. All monetary values are in DKK, 2023-prices. Control variables included are age fixed effects, year fixed effects, the first 10 principal components, education dummies, marriage dummy, experience, experience squared, and dummy for whether child is in iPSYCH control group or not. N refers to the number of individuals, and $N \times$ *Years* refers to the total number of individual-year observations. Standard errors are clustered at the individual level and shown in parentheses. Significance levels are denoted as * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table A.9: *APOE-e4* Carrier Status and Labor Market Attachment for Men

	Employment (1)	Unemployment (2)	DP (3)	Transfers (4)	Pension (5)
Panel A: Age 45-65					
Homogeneous carrier	-0.00920*	0.00251	0.00161	0.00108	0.00399
	(0.00505)	(0.00162)	(0.00409)	(0.00241)	(0.00251)
Heterogeneous carrier	0.00167	0.00029	-0.00151	-0.00042	-0.00003
	(0.00189)	(0.00061)	(0.00155)	(0.00090)	(0.00093)
$N \times Years$	940,718	940,718	940,718	940,718	940,718
N	98,560	98,560	98,560	98,560	98,560
R^2	0.251	0.013	0.187	0.051	0.166
Mean	0.81	0.03	0.07	0.04	0.05
Pct. Change (Homo)	-1.13	8.88	2.23	2.80	7.93
Pct. Change (Hetero)	0.21	1.03	-2.10	-1.09	-0.06
Panel B: Age 55-65					
Homogeneous carrier	-0.01475*	0.00077	0.00458	0.00237	0.00703
	(0.00768)	(0.00209)	(0.00620)	(0.00313)	(0.00478)
Heterogeneous carrier	0.00018	0.00076	-0.00108	-0.00009	0.00024
	(0.00285)	(0.00085)	(0.00231)	(0.00108)	(0.00173)
$N \times Years$	407,980	407,980	407,980	407,980	407,980
N	65,436	65,436	65,436	65,436	65,436
R^2	0.240	0.011	0.196	0.038	0.189
Mean	0.75	0.03	0.09	0.03	0.09
Pct. Change (Homo)	-1.96	2.79	4.83	7.25	7.72
Pct. Change (Hetero)	0.02	2.75	-1.14	-0.28	0.26

Notes: This table reports estimates based on Equation 1 using child's *APOE-e4* carrier status (homogeneous or heterogeneous carrier) as measure of genetic risk for men aged 45 to 65 (Panel A) and men aged 55 to 65 (Panel B). In column (1), the outcome is a dummy for being employed, in column (2), the outcome is a dummy for being unemployed, in column (3), the outcome is a dummy for receiving disability pension, in column (4), the outcome variable is a dummy for receiving transfers, and in column (5), the outcome variable is a dummy for pension. Control variables included are age fixed effects, year fixed effects, the first 10 principal components, education dummies, marriage dummy, experience, experience squared, and dummy for whether child is in iPSYCH control group or not. N refers to the number of individuals, and $N \times Years$ refers to the total number of individual-year observations. Standard errors are clustered at the individual level and shown in parentheses. Significance levels are denoted as * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table A.10: *APOE-e4* Carrier Status, Income, and Wealth for Men

	Earnings	Income from Shares	Disposable Income	Wealth
	(1)	(2)	(3)	(4)
Panel A: Age 45-65				
Homogeneous carrier	1,469.78	-83.87	966.32	300.99
	(4,407.00)	(578.29)	(2,926.70)	(26,219.17)
Heterogeneous carrier	-667.55	-310.53	-751.37	-10,264.53
	(1,590.27)	(224.02)	(1,116.65)	(9,864.07)
$N \times$ *Years*	1,243,045	1,243,045	1,243,045	1,243,045
N	101,228	101,228	101,228	101,228
R^2	0.396	0.021	0.231	0.076
Mean	415,563.64	7,977.01	347,974.46	496,335.66
Pct. Change (Homo)	0.35	-1.05	0.28	0.06
Pct. Change (Hetero)	-0.16	-3.89	-0.22	-2.07
Panel B: Age 55-65				
Homogeneous carrier	-4,400.15	-838.47	-4,574.65	-8,205.47
	(5,739.17)	(801.51)	(3,910.74)	(38,895.54)
Heterogeneous carrier	-559.69	-168.18	-955.60	-21,674.60
	(2,101.09)	(319.26)	(1,530.92)	(14,525.41)
$N \times$ *Years*	465,012	465,012	465,012	465,012
N	67,010	67,010	67,010	67,010
R^2	0.403	0.021	0.235	0.073
Mean	373,107.11	9,046.00	351,391.72	710,473.37
Pct. Change (Homo)	-1.18	-9.27	-1.30	-1.15
Pct. Change (Hetero)	-0.15	-1.86	-0.27	-3.05

Notes: This table reports estimates based on Equation 1 using child's *APOE-e4* carrier status (homogeneous or heterogeneous carrier) as measure of genetic risk for men aged 45 to 65 (Panel A) and men aged 55 to 65 (Panel B). In column (1), the outcome is earnings, in column (2), the outcome is income from shares, in column (3), the outcome is disposable income, and in column (4), the outcome is net wealth excluding pension wealth. All monetary values are in DKK, 2023-prices. Control variables included are age fixed effects, year fixed effects, the first 10 principal components, education dummies, marriage dummy, experience, experience squared, and dummy for whether child is in iPSYCH control group or not. N refers to the number of individuals, and $N \times$ *Years* refers to the total number of individual-year observations. Standard errors are clustered at the individual level and shown in parentheses. Significance levels are denoted as * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table A.11: Probit Regressions: Genetic Risk of AD and Dementia

	Women		Men	
	Age 45-65	Age 55-65	Age 45-65	Age 55-65
	(1)	(2)	(3)	(4)
Panel A: *APOE-e4* Carrier Status				
APOE-e4 Carrier	0.00015***	0.00031***	0.00008**	0.00017**
	(0.00004)	(0.00009)	(0.00003)	(0.00007)
$N \times Years$	1,047,052	371,752	1,084,496	440,274
N	103,355	61,957	99,993	66,369
Panel B: AD PGS				
AD PGS	0.00007***	0.00015***	0.00004**	0.00009**
	(0.00002)	(0.00005)	(0.00002)	(0.00004)
$N \times Years$	1,047,052	371,752	1,084,496	440,274
N	103,355	61,957	99,993	66,369

Notes: This table reports marginal effects based on a probit regression using child's genetic risk of AD (*APOE-e4* carrier status in Panel A and AD PGS in Panel B) for women and men on the probability of being diagnosed with dementia. Control variables included are age fixed effects, year fixed effects, the first 10 principal components, education dummies, marriage dummy, experience, experience squared, and dummy for whether child is in iPSYCH control group or not. N refers to the number of individuals, and $N \times Years$ refers to the total number of individual-year observations. Standard errors are clustered at the individual level and shown in parentheses. Significance levels are denoted as * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table A.12: Probit Regressions: *APOE-e4* Carrier Status and Labor Market Attachment for Women

	Employment	Unemployment	DP	Transfers	Pension
	(1)	(2)	(3)	(4)	(5)
Panel A: Age 45-65					
APOE-e4 carrier	-0.00354**	-0.00032	0.00435***	0.00056	-0.00056
	(0.00176)	(0.00050)	(0.00167)	(0.00099)	(0.00084)
$N \times Years$	1,005,275	1,005,275	1,005,275	1,005,275	1,005,275
N	105,896	105,896	105,896	105,896	105,896
Panel B: Age 55-65					
APOE-e4 carrier	-0.00715**	0.00037	0.00823***	0.00037	-0.00117
	(0.00283)	(0.00073)	(0.00260)	(0.00122)	(0.00176)
$N \times Years$	360,716	360,716	360,716	360,716	360,716
N	61,814	61,814	61,814	61,814	61,814

Notes: This table reports marginal effects based on a probit regression using child's *APOE-e4* carrier status as measure of genetic risk for women aged 45 to 65 (Panel A) and women aged 55 to 65 (Panel B). In column (1), the outcome is a dummy for being employed, in column (2), the outcome is a dummy for being unemployed, in column (3), the outcome is a dummy for receiving disability pension, in column (4), the outcome variable is a dummy for receiving transfers, and in column (5), the outcome variable is a dummy for pension. Control variables included are age fixed effects, year fixed effects, the first 10 principal components, education dummies, marriage dummy, experience, experience squared, and dummy for whether child is in iPSYCH control group or not. N refers to the number of individuals, and $N \times Years$ refers to the total number of individual-year observations. Standard errors are clustered at the individual level and shown in parentheses. Significance levels are denoted as * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table A.13: Probit Regressions: AD PGS and Labor Market Attachment for Women

	Employment	Unemployment	DP	Transfers	Pension
	(1)	(2)	(3)	(4)	(5)
Panel A: Age 45-65					
AD PGS	-0.00033	-0.00032	0.00137*	-0.00031	-0.00024
	(0.00082)	(0.00023)	(0.00078)	(0.00046)	(0.00039)
$N \times Years$	1,005,275	1,005,275	1,005,275	1,005,275	1,005,275
N	105,896	105,896	105,896	105,896	105,896
Panel B: Age 55-65					
AD PGS	-0.00245*	0.00025	0.00283**	0.00012	-0.00025
	(0.00131)	(0.00034)	(0.00121)	(0.00058)	(0.00081)
$N \times Years$	360,716	360,716	360,716	360,716	360,716
N	61,814	61,814	61,814	61,814	61,814

Notes: This table reports marginal effects based on a probit regression using child's AD PGS as measure of genetic risk for women aged 45 to 65 (Panel A) and women aged 55 to 65 (Panel B). In column (1), the outcome is a dummy for being employed, in column (2), the outcome is a dummy for being unemployed, in column (3), the outcome is a dummy for receiving disability pension, in column (4), the outcome variable is a dummy for receiving transfers, and in column (5), the outcome variable is a dummy for pension. Control variables included are age fixed effects, year fixed effects, the first 10 principal components, education dummies, marriage dummy, experience, experience squared, and dummy for whether child is in iPSYCH control group or not. N refers to the number of individuals, and $N \times Years$ refers to the total number of individual-year observations. Standard errors are clustered at the individual level and shown in parentheses. Significance levels are denoted as * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table A.14: Probit Regressions: *APOE-e4* Carrier Status and Labor Market Attachment for Men

	Employment	Unemployment	DP	Transfers	Pension
	(1)	(2)	(3)	(4)	(5)
Panel A: Age 45-65					
APOE-e4 carrier	0.00015	0.00049	-0.00100	-0.00004	0.00046
	(0.00173)	(0.00058)	(0.00143)	(0.00082)	(0.00088)
$N \times Years$	940,718	940,718	940,718	940,718	940,718
N	98,560	98,560	98,560	98,560	98,560
Panel B: Age 55-65					
APOE-e4 carrier	-0.00170	0.00073	0.00008	0.00040	0.00111
	(0.00264)	(0.00080)	(0.00216)	(0.00101)	(0.00164)
$N \times Years$	407,980	407,980	407,980	407,980	407,980
N	65,436	65,436	65,436	65,436	65,436

Notes: This table reports marginal effects based on a probit regression using child's *APOE-e4* carrier status as measure of genetic risk for men aged 45 to 65 (Panel A) and men aged 55 to 65 (Panel B). In column (1), the outcome is a dummy for being employed, in column (2), the outcome is a dummy for being unemployed, in column (3), the outcome is a dummy for receiving disability pension, in column (4), the outcome variable is a dummy for receiving transfers, and in column (5), the outcome variable is a dummy for pension. Control variables included are age fixed effects, year fixed effects, the first 10 principal components, education dummies, marriage dummy, experience, experience squared, and dummy for whether child is in iPSYCH control group or not. N refers to the number of individuals, and $N \times Years$ refers to the total number of individual-year observations. Standard errors are clustered at the individual level and shown in parentheses. Significance levels are denoted as * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table A.15: Probit Regressions: AD PGS and Labor Market Attachment for Men

	Employment	Unemployment	DP	Transfers	Pension
	(1)	(2)	(3)	(4)	(5)
Panel A: Age 45-65					
AD PGS	-0.00037	-0.00002	0.00026	0.00054	-0.00026
	(0.00080)	(0.00027)	(0.00066)	(0.00038)	(0.00040)
$N \times Years$	940,718	940,718	940,718	940,718	940,718
N	98,560	98,560	98,560	98,560	98,560
Panel B: Age 55-65					
AD PGS	-0.00034	-0.00015	0.00095	0.00072	-0.00086
	(0.00123)	(0.00037)	(0.00100)	(0.00046)	(0.00075)
$N \times Years$	407,980	407,980	407,980	407,980	407,980
N	65,436	65,436	65,436	65,436	65,436

Notes: This table reports marginal effects based on a probit regression using child's AD PGS as measure of genetic risk for men aged 45 to 65 (Panel A) and men aged 55 to 65 (Panel B). In column (1), the outcome is a dummy for being employed, in column (2), the outcome is a dummy for being unemployed, in column (3), the outcome is a dummy for receiving disability pension, in column (4), the outcome variable is a dummy for receiving transfers, and in column (5), the outcome variable is a dummy for pension. Control variables included are age fixed effects, year fixed effects, the first 10 principal components, education dummies, marriage dummy, experience, experience squared, and dummy for whether child is in iPSYCH control group or not. N refers to the number of individuals, and $N \times Years$ refers to the total number of individual-year observations. Standard errors are clustered at the individual level and shown in parentheses. Significance levels are denoted as * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table A.16: *APOE-e4* Carrier Status and Log-Specifications for Women

	Log(Earnings)	Log(Income from Shares)	Log(Disposable) Income)	Log(Wealth)
	(1)	(2)	(3)	(4)
Panel A: Age 45-65				
APOE-e4 carrier	-0.00366	0.00448	-0.00088	-0.00683
	(0.00505)	(0.02998)	(0.00242)	(0.01209)
$N \times$ *Years*	970,725	185,848	1,224,151	702,477
N	95,089	28,554	106,712	80,001
R^2	0.215	0.078	0.286	0.148
Panel B: Age 55-65				
APOE-e4 carrier	-0.00312	-0.02144	-0.00165	-0.01722
	(0.00912)	(0.04133)	(0.00405)	(0.01652)
$N \times$ *Years*	279,023	70,338	381,168	251,911
N	50,833	15,900	62,011	46,712
R^2	0.204	0.060	0.267	0.141

Notes: This table reports estimates based on Equation 1 using child's *APOE-e4* carrier status as measure of genetic risk for women aged 45 to 65 (Panel A) and women aged 55 to 65 (Panel B). In column (1), the outcome is the logarithm of earnings, in column (2), the outcome is the logarithm of income from shares, in column (3), the outcome is the logarithm of disposable income, and in column (4), the outcome is the logarithm of net wealth excluding pension wealth. All monetary values are in DKK, 2023-prices. Control variables included are age fixed effects, year fixed effects, the first 10 principal components, education dummies, marriage dummy, experience, experience squared, and dummy for whether child is in iPSYCH control group or not. N refers to the number of individuals, and $N \times$ *Years* refers to the total number of individual-year observations. Standard errors are clustered at the individual level and shown in parentheses. Significance levels are denoted as * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table A.17: AD PGS and and Log-Specifications for Women

	Log(Earnings)	Log(Income from Shares)	Log(Disposable) Income)	Log(Wealth)
	(1)	(2)	(3)	(4)
Panel A: Age 45-65				
AD PGS	0.00008	-0.00384	-0.00083	-0.00195
	(0.00231)	(0.01380)	(0.00111)	(0.00557)
$N \times$ *Years*	970,725	185,848	1,224,151	702,477
N	95,089	28,554	106,712	80,001
R^2	0.215	0.078	0.286	0.148
Panel B: Age 55-65				
AD PGS	0.00093	-0.00930	-0.00287	-0.00312
	(0.00419)	(0.01903)	(0.00185)	(0.00762)
$N \times$ *Years*	279,023	70,338	381,168	251,911
N	50,833	15,900	62,011	46,712
R^2	0.204	0.060	0.267	0.141

Notes: This table reports estimates based on Equation 1 using child's AD PGS as measure of genetic risk for women aged 45 to 65 (Panel A) and women aged 55 to 65 (Panel B). In column (1), the outcome is the logarithm of earnings, in column (2), the outcome is the logarithm of income from shares, in column (3), the outcome is the logarithm of disposable income, and in column (4), the outcome is the logarithm of net wealth excluding pension wealth. All monetary values are in DKK, 2023-prices. Control variables included are age fixed effects, year fixed effects, the first 10 principal components, education dummies, marriage dummy, experience, experience squared, and dummy for whether child is in iPSYCH control group or not.N refers to the number of individuals, and $N \times$ *Years* refers to the total number of individual-year observations. Standard errors are clustered at the individual level and shown in parentheses. Significance levels are denoted as * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table A.18: *APOE-e4* Carrier Status and Log-Specifications for Men

	Log(Earnings)	Log(Income from Shares)	Log(Disposable Income)	Log(Wealth)
	(1)	(2)	(3)	(4)
Panel A: Age 45-65				
APOE-e4 carrier	-0.00066	-0.05691*	-0.00163	-0.01041
	(0.00560)	(0.03189)	(0.00283)	(0.01173)
$N \times Years$	1,014,943	270,212	1,227,369	727,372
N	93,191	38,602	101,157	76,982
R^2	0.197	0.068	0.237	0.098
Panel B: Age 55-65				
APOE-4 carrier	-0.00048	-0.05541	-0.00582	-0.01404
	(0.00876)	(0.04056)	(0.00402)	(0.01519)
$N \times Years$	354,632	112,529	459,960	304,764
N	57,884	23,327	66,905	50,551
R^2	0.190	0.056	0.238	0.097

Notes: This table reports estimates based on Equation 1 using child's *APOE-e4* carrier status as measure of genetic risk for men aged 45 to 65 (Panel A) and men aged 55 to 65 (Panel B). In column (1), the outcome is the logarithm of earnings, in column (2), the outcome is the logarithm of income from shares, in column (3), the outcome is the logarithm of disposable income, and in column (4), the outcome is the logarithm of net wealth excluding pension wealth. All monetary values are in DKK, 2023-prices. Control variables included are age fixed effects, year fixed effects, the first 10 principal components, education dummies, marriage dummy, experience, experience squared, and dummy for whether child is in iPSYCH control group or not. N refers to the number of individuals, and $N \times Years$ refers to the total number of individual-year observations. Standard errors are clustered at the individual level and shown in parentheses. Significance levels are denoted as * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table A.19: AD PGS and Log-Specifications for Men

	Log(Earnings)	Log(Income from Shares)	Log(Disposable Income)	Log(Wealth)
	(1)	(2)	(3)	(4)
Panel A: Age 45-65				
AD PGS	0.00017	-0.03072**	-0.00144	-0.00498
	(0.00261)	(0.01481)	(0.00131)	(0.00547)
$N \times Years$	1,014,943	270,212	1,227,369	727372
N	93,191	38,602	101,157	76,982
R^2	0.197	0.068	0.237	0.098
Panel B: Age 55-65				
AD PGS	-0.00158	-0.03740**	-0.00171	-0.00435
	(0.00403)	(0.01882)	(0.00182)	(0.00705)
$N \times Years$	354,632	112,529	459,960	304,764
N	57,884	23,327	66,905	50,551
R^2	0.190	0.056	0.238	0.097

Notes: This table reports estimates based on Equation 1 using child's AD PGS as measure of genetic risk for men aged 45 to 65 (Panel A) and men aged 55 to 65 (Panel B). In column (1), the outcome is the logarithm of earnings, in column (2), the outcome is the logarithm of income from shares, in column (3), the outcome is the logarithm of disposable income, and in column (4), the outcome is the logarithm of net wealth excluding pension wealth. All monetary values are in DKK, 2023-prices. Control variables included are age fixed effects, year fixed effects, the first 10 principal components, education dummies, marriage dummy, experience, experience squared, and dummy for whether child is in iPSYCH control group or not. N refers to the number of individuals, and $N \times Years$ refers to the total number of individual-year observations. Standard errors are clustered at the individual level and shown in parentheses. Significance levels are denoted as * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table A.20: Genetic Risk of AD and Mortality

	Women		Men	
	(1)	(2)	(3)	(4)
APOE-e4 carrier	-0.00227		0.00362	
	(0.00524)		(0.00499)	
AD PGS		-0.00049		0.00016
		(0.00246)		(0.00231)
N	13,495	13,495	22,897	22,897
R^2	0.049	0.049	0.069	0.069
Mean	0.091	0.091	0.153	0.153
Pct. Change	-2.499	-0.544	2.365	0.105

Notes: This table reports estimates based on Equation 1 using child's *APOE-e4* carrier status (columns (1) and (3)) and AD PGS (columns (2) and (4)) as measure of genetic risk. In columns (1)-(2), the outcome is mortality at age 65 for women, and in columns (3)-(4), the outcome is mortality at age 65 for men. Control variables are measured the first year an individual is present in the sample period 2005-2020 and include cohort fixed effects, year fixed effects, the first 10 principal components, education dummies, marriage dummy, experience, experience squared, and dummy for whether child is in iPSYCH control group or not. N refers to the number of individuals. Robust standard errors are shown in parentheses. Significance levels are denoted as * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table A.21: *APOE-e4* Carrier Status and Labor Market Attachment for Women in Labor Market at Age 45

	Employment	Unemployment	DP	Transfers	Pension
	(1)	(2)	(3)	(4)	(5)
Panel A: Age 46-65					
APOE-e4 carrier	-0.00422**	-0.00001	0.00383***	0.00049	-0.00009
	(0.00176)	(0.00055)	(0.00141)	(0.00091)	(0.00083)
$N \times Years$	841,251	841,251	841,251	841,251	841,251
R^2	0.239	0.011	0.136	0.052	0.257
N	87,569	87,569	87,569	87,569	87,569
Mean	0.84	0.02	0.05	0.04	0.04
Pct. Change	-0.50	-0.06	7.68	1.09	-0.20
Panel B: Age 55-65					
APOE-e4 carrier	-0.00700**	0.00077	0.00588**	0.00105	-0.00071
	(0.00301)	(0.00082)	(0.00258)	(0.00125)	(0.00187)
$N \times Years$	305,034	305,034	305,034	305,034	305,034
R^2	0.273	0.007	0.182	0.040	0.281
N	51,948	51,948	51,948	51,948	51,948
Mean	0.75	0.02	0.09	0.04	0.10
Pct. Change	-0.93	3.35	6.63	2.75	-0.73

Notes: This table reports estimates based on Equation 1 using child's *APOE-e4* carrier status as measure of genetic risk for women aged 46 to 65 (Panel A) and women aged 55 to 65 (Panel B). The sample is restricted to women who have positive labor market earnings at age 45. In column (1), the outcome is a dummy for being employed, in column (2), the outcome is a dummy for being unemployed, in column (3), the outcome is a dummy for receiving disability pension, in column (4), the outcome variable is a dummy for receiving transfers, and in column (5), the outcome variable is a dummy for pension. Control variables included are age fixed effects, year fixed effects, the first 10 principal components, education dummies, marriage dummy, experience, experience squared, and dummy for whether child is in iPSYCH control group or not. N refers to the number of individuals, and $N \times Years$ refers to the total number of individual-year observations. Standard errors are clustered at the individual level and shown in parentheses. Significance levels are denoted as * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table A.22: *APOE-e4* Carrier Status and Labor Market Attachment for Men in Labor Market at Age 45

	Employment	Unemployment	DP	Transfers	Pension
	(1)	(2)	(3)	(4)	(5)
Panel A: Age 46-65					
APOE-e4 carrier	-0.00066	0.00078	-0.00018	0.00005	0.00002
	(0.00168)	(0.00062)	(0.00120)	(0.00076)	(0.00090)
$N \times$ *Years*	809,377	809,377	809,377	809,377	809,377
R^2	0.218	0.017	0.112	0.043	0.194
N	84,127	84,127	84,127	84,127	84,127
Mean	0.86	0.03	0.04	0.03	0.05
Pct. Change	-0.08	2.71	-0.49	0.16	0.04
Panel B: Age 55-65					
APOE-e4 carrier	-0.00092	0.00112	-0.00062	0.00017	0.00026
	(0.00273)	(0.00089)	(0.00206)	(0.00102)	(0.00178)
$N \times$ *Years*	350,837	350,837	350,837	350,837	350,837
R^2	0.236	0.013	0.136	0.039	0.208
N	55,938	55,938	55,938	55,938	55,938
Mean	0.79	0.03	0.06	0.03	0.09
Pct. Change	-0.12	3.90	-0.99	0.57	0.28

Notes: This table reports estimates based on Equation 1 using child's *APOE-e4* carrier status as measure of genetic risk for men aged 46 to 65 (Panel A) and men aged 55 to 65 (Panel B). The sample is restricted to men who have positive labor market earnings at age 45. In column (1), the outcome is a dummy for being employed, in column (2), the outcome is a dummy for being unemployed, in column (3), the outcome is a dummy for receiving disability pension, in column (4), the outcome variable is a dummy for receiving transfers, and in column (5), the outcome variable is a dummy for pension. Control variables included are age fixed effects, year fixed effects, the first 10 principal components, education dummies, marriage dummy, experience, experience squared, and dummy for whether child is in iPSYCH control group or not. N refers to the number of individuals, and $N \times$ *Years* refers to the total number of individual-year observations. Standard errors are clustered at the individual level and shown in parentheses. Significance levels are denoted as * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table A.23: Genetic Risk of AD and Dementia for Grandparents

	Grandmother (1)	Grandfather (2)	Grandmother (3)	Grandfather (4)
Panel A: Maternal Grandparents				
APOE-e4 Carrier	0.00194***	0.00131***		
	(0.00036)	(0.00044)		
AD PGS			0.00058***	0.00060***
			(0.00016)	(0.00020)
$N \times$ *Years*	205,801	158,018	205,801	158,018
N	32,438	26,447	32,438	26,447
R^2	0.005	0.005	0.005	0.005
Mean	0.0037	0.0043	0.0037	0.0043
Pct. Change	52.97	30.24	15.84	13.76
Panel B: Paternal Grandparents				
APOE-e4 Carrier	0.00215***	0.00131***		
	(0.00041)	(0.00049)		
AD PGS			0.00085***	0.00071***
			(0.00019)	(0.00023)
$N \times$ *Years*	166,934	127,397	166,934	127,397
N	24,975	20,493	24,975	20,493
R^2	0.006	0.005	0.006	0.005
Mean	0.0038	0.0046	0.0038	0.0046
Pct. Change	56.35	28.78	22.21	15.54

Notes: This table reports estimates based on Equation 1 using grandchild's *APOE-e4* carrier status (columns (1)-(2)) and AD PGS (columns (3)-(4)) as measure of genetic risk for grandmothers (columns (1) and (3)) and grandfathers (columns (2) and (4)) for maternal grandparents (Panel A) and paternal grandparents (Panel B) whose child is aged 45 to 65. In columns (1)-(4), the outcome is diagnosed dementia. Control variables included are cohort fixed effects, age fixed effects for child, year fixed effects, the first 10 principal components, education dummies for child, marriage dummy for child, experience for child, experience squared for child, and dummy for whether grandchild is in iPSYCH control group or not. N refers to the number of individuals, and $N \times$ *Years* refers to the total number of individual-year observations. Standard errors are clustered at the individual level and shown in parentheses. Significance levels are denoted as * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table A.24: Household Earnings and Wealth

	Household Earnings		Household Wealth	
	Age 45-65	Age 55-65	Age 45-65	Age 55-65
	(1)	(2)	(3)	(4)
Panel A: Women				
APOE-e4 Carrier	482.85	-1,837.96	-19,939.78	-49,185.18*
	(2,508.40)	(3,681.95)	(17,188.05)	(27,131.02)
$N \times$ *Years*	737,027	227,744	737,027	227,744
N	72,417	38,153	72,417	38,153
R^2	0.452	0.457	0.124	0.121
Mean	779,979	681,642	1,025,337	1,462,668
Pct. Change	0.06	-0.27	-1.94	-3.36
Panel B: Men				
APOE-e4 Carrier	613.39	-1,042.17	-14,752.01	-14,545.21
	(2,397.04)	(3,234.37)	(15,480.89)	(22,399.54)
$N \times$ *Years*	811,166	305,786	811,166	305,786
N	74,691	45,821	74,691	45,821
R^2	0.446	0.452	0.120	0.119
Mean	798,197	741,587	915,440	1,260,256
Pct. Change	0.08	-0.14	-1.61	-1.15

Notes: This table reports estimates based on Equation 1 using child's *APOE-e4* carrier status as measure of genetic risk for women aged 45-65 and 55-65 (Panel A) and men aged 45-65 and 55-65 (Panel B). In columns (1)-(2), the outcome is household earnings, and in columns (3)-(4), the outcome is household net wealth excluding pension wealth. The sample is restricted to married individuals. All monetary values are in DKK, 2023-prices. Control variables included are age fixed effects, year fixed effects, the first 10 principal components, education dummies, experience, experience squared, and dummy for whether child is in iPSYCH control group or not. N refers to the number of individuals, and $N \times$ *Years* refers to the total number of individual-year observations. Standard errors are clustered at the household level and shown in parentheses. Significance levels are denoted as * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table A.25: Household Earnings and Wealth by Parenthood

	Household Earnings		Household Wealth	
	Non-parent	Parent	Non-parent	Parent
	(1)	(2)	(3)	(4)
Panel A: Women				
APOE-e4 Carrier	-7,443.94	-55.91	-27,760.41	-54,098.85*
	(8,166.34)	(4,109.12)	(47,452.10)	(31,278.44)
$N \times$ *Years*	41,378	186,366	41,378	186,366
N	8,664	29,553	8,664	29,553
R^2	0.443	0.463	0.140	0.110
Mean	667,383	684,808	767,004	1,617,123
Pct. Change	-1.12	-0.01	-3.62	-3.35
Panel B: Men				
APOE-e4 Carrier	-5,940.30	-58.51	-5,383.77	-13,723.99
	(7,504.14)	(3,570.17)	(39,629.48)	(25,678.56)
$N \times$ *Years*	55,986	249,800	55,986	249,800
N	10,257	35,882	10,257	35,882
R^2	0.468	0.450	0.095	0.117
Mean	725,240	745,251	578,015	1,413,162
Pct. Change	-0.82	-0.01	-0.93	-0.97

Notes: This table reports estimates based on Equation 1 using child's *APOE-e4* carrier status as measure of genetic risk for women aged 55-65 (Panel A) and men aged 55-65 (Panel B) by whether spouse is parent or not to child whose genetic risk is utilized. In columns (1)-(2), the outcome is household earnings, and in columns (3)-(4), the outcome is household net wealth excluding pension wealth. The sample is restricted to married individuals. All monetary values are in DKK, 2023-prices. Control variables included are age fixed effects, year fixed effects, the first 10 principal components, education dummies, experience, experience squared, and dummy for whether child is in iPSYCH control group or not. N refers to the number of individuals, and $N \times$ *Years* refers to the total number of individual-year observations. Standard errors are clustered at the household level and shown in parentheses. Significance levels are denoted as * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table A.26: Household Earnings and Wealth: Partner's Age Restricted

	Household Earnings		Household Wealth	
	Age 45-65	Age 55-65	Age 45-65	Age 55-65
	(1)	(2)	(3)	(4)
Panel A: Women				
APOE-e4 Carrier	13.84	-3,535.01	-12,939.34	-25451.94
	(2,611.31)	(4,069.15)	(17,611.08)	(29,067.84)
$N \times$ *Years*	658,549	169,809	658,549	169,809
N	69,768	33,749	69,768	33,749
R^2	0.439	0.434	0.115	0.114
Mean	806,170	736,552	1,027,854	1,488,353
Pct. Change	0.00	-0.48	-1.26	-1.71
Panel B: Men				
APOE-e4 Carrier	289.20	-3,270.47	-13,445.58	-24,336.58
	(2,630.02)	(4,037.15)	(17,338.11)	(27,962.50)
$N \times$ *Years*	638,424	168,758	638,424	168,758
N	66,585	32,963	66,585	32,963
R^2	0.445	0.444	0.115	0.115
Mean	807,738	733,297	1,027,685	1,450,881
Pct. Change	0.04	-0.45	-1.31	-1.68

Notes: This table reports estimates based on Equation 1 using child's *APOE-e4* carrier status as measure of genetic risk for women aged 45-65 (whose spouse is also 45-65) and women aged 55-65 (whose spouse is also 55-65) (Panel A) and men aged 45-65 (whose spouse is also 45-65) and men aged 55-65 (whose spouse is also 55-65) (Panel B). In columns (1)-(2), the outcome is household earnings, and in columns (3)-(4), the outcome is household net wealth excluding pension wealth. The sample is restricted to married individuals. All monetary values are in DKK, 2023-prices. Control variables included are age fixed effects, year fixed effects, the first 10 principal components, education dummies, experience, experience squared, and dummy for whether child is in iPSYCH control group or not. N refers to the number of individuals, and $N \times$ *Years* refers to the total number of individual-year observations. Standard errors are clustered at the household level and shown in parentheses. Significance levels are denoted as * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

A.4 Entropy Balancing

From descriptive statistics, we observe that men and women differ in age, education, and the time period, they are observed. Table A.27 presents descriptive statistics before and after entropy balancing for women and men aged 45 to 65 for year 2000–2020. Women in our sample tend to be younger on average than men (by 0.84 years) and are observed in slightly later periods (the mean observation year is 2013 for women compared to 2012 for men). This reflects the tendency for women to have children at a younger age than men. To address these differences, we apply entropy balancing, adjusting the weights for women so that their age, education, and time period distributions match the mean, standard deviation, and skewness of the corresponding distributions for men. For a detailed explanation of entropy balancing, see Hainmueller and Xu (2013). From Table A.27, it can clearly be seen that entropy balancing is successful in making women match the distribution of men for the selected covariates. We then show how that affects our results in Tables A.28-A.30.

Table A.27: Entropy Balancing

	Women			Men		
	Mean	SD	Skewness	Mean	SD	Skewness
Before entropy balancing						
Age	52.12	5.19	0.54	52.96	5.48	0.39
General upper secondary	0.04	0.20	4.64	0.04	0.20	4.58
Vocational education	0.36	0.48	0.58	0.43	0.50	0.28
Short cycle tertiary	0.04	0.19	4.83	0.05	0.21	4.27
Bachelor	0.25	0.43	1.14	0.13	0.34	2.18
Master, doctoral	0.06	0.25	3.55	0.09	0.29	2.85
Experience	19.54	9.48	-0.31	22.36	9.66	-0.57
Year	2012.77	5.23	-0.52	2011.83	5.54	-0.35
After entropy balancing						
Age	52.96	5.48	0.39	52.96	5.48	0.39
General upper secondary	0.04	0.20	4.59	0.04	0.20	4.58
Vocational education	0.43	0.50	0.28	0.43	0.50	0.28
Short cycle tertiary	0.05	0.21	4.27	0.05	0.21	4.27
Bachelor	0.13	0.34	2.18	0.13	0.34	2.18
Master, doctoral	0.09	0.29	2.85	0.09	0.29	2.85
Experience	22.36	9.66	-0.57	22.36	9.66	-0.57
Year	2011.83	5.54	-0.35	2011.83	5.54	-0.35
$N \times Years$		1,229,258			1,243,045	
N		106,735			101,228	

Notes: The table displays means, standard deviations, and skewnesses for men and women aged 45 to 65 for age, education dummies, and time period before and after entropy balancing on these covariates.

Table A.28: *APOE-e4* Carrier Status and Health Outcomes for Women

	Dementia		GP visits	
	Age 45-65	Age 55-65	Age 45-65	Age 55-65
	(1)	(2)	(3)	(4)
APOE-e4 Carrier	0.00012***	0.00023***	0.04192*	0.09402**
	(0.00004)	(0.00008)	(0.02459)	(0.03656)
$N \times Years$	1,123,485	375,336	1,123,485	375,336
N	106,374	61,992	106,374	61,992
R^2	0.000	0.000	0.041	0.035
Mean	0.0002	0.0003	4.13	4.09
Pct. Change	77.22	81.23	1.01	2.30

Notes: This table reports estimates based on Equation 1 using child's *APOE-e4* carrier status as measure of genetic risk for the re-weighted sample of women after entropy balancing. In columns (1)-(2), the outcome is a dummy for diagnosed dementia. In columns (3)-(4), the outcome variable is number of GP visits. Control variables included are age fixed effects, year fixed effects, the first 10 principal components, education dummies, marriage dummy, experience, experience squared, and dummy for whether child is in iPSYCH control group or not. N refers to the number of individuals, and $N \times Years$ refers to the total number of individual-year observations. Standard errors are clustered at the individual level and shown in parentheses. Significance levels are denoted as * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table A.29: *APOE-e4* Carrier Status and Labor Market Attachment for Women

	Employment	Unemployment	DP	Transfers	Pension
	(1)	(2)	(3)	(4)	(5)
Panel A: Age 45-65					
APOE-e4 Carrier	-0.00279	-0.00020	0.00309**	-0.00004	-0.00007
	(0.00170)	(0.00049)	(0.00132)	(0.00077)	(0.00105)
$N \times Years$	1,005,275	1,005,275	1,005,275	1,005,275	1,005,275
N	105,896	105,896	105,896	105,896	105,896
R^2	0.355	0.007	0.317	0.057	0.267
Mean	0.80	0.02	0.08	0.04	0.06
Pct. Change	-0.35	-0.93	3.73	-0.09	-0.12
Panel B: Age 55-65					
APOE-e4 Carrier	-0.00475*	0.00026	0.00427**	-0.00009	0.00031
	(0.00276)	(0.00075)	(0.00192)	(0.00092)	(0.00210)
$N \times Years$	360,716	360,716	360,716	360,716	360,716
N	61,814	61,814	61,814	61,814	61,814
R^2	0.337	0.007	0.337	0.032	0.279
Mean	0.75	0.02	0.09	0.03	0.11
Pct. Change	-0.63	1.34	4.67	-0.34	0.29

Notes: This table reports estimates based on Equation 1 using child's *APOE-e4* carrier status as measure of genetic risk for women aged 45 to 65 (Panel A) and women aged 55 to 65 (Panel B) for the re-weighted sample of women after entropy balancing. In column (1), the outcome is a dummy for being employed, in column (2), the outcome is a dummy for being unemployed, in column (3), the outcome is a dummy for receiving disability pension, in column (4), the outcome variable is a dummy for receiving transfers, and in column (5), the outcome variable is a dummy for pension. Control variables included are age fixed effects, year fixed effects, the first 10 principal components, education dummies, marriage dummy, experience, experience squared, and dummy for whether child is in iPSYCH control group or not. N refers to the number of individuals, and $N \times Years$ refers to the total number of individual-year observations. Standard errors are clustered at the individual level and shown in parentheses. Significance levels are denoted as * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table A.30: *APOE-e4* Carrier Status, Income, and Wealth for Women

	Earnings	Income from Shares	Disposable Income	Wealth
	(1)	(2)	(3)	(4)
Panel A: Age 45-65				
APOE-e4 Carrier	-531.81	2.96	-426.10	-11,454.49
	(1,098.98)	(51.95)	(670.11)	(7,189.80)
$N \times Years$	1,229,258	1,229,258	1,229,258	1,229,258
N	106,735	106,735	106,735	106,735
R^2	0.502	0.028	0.415	0.115
Mean	341,635.15	1,500.24	294,977.78	349,770.36
Pct. Change	-0.16	0.20	-0.14	-3.27
Panel B: Age 55-65				
APOE-e4 Carrier	170.68	36.50	-314.89	-18,047.51
	(1,688.00)	(93.83)	(1,017.15)	(11,481.90)
$N \times Years$	382,779	382,779	382,779	382,779
N	62,063	62,063	62,063	62,063
R^2	0.491	0.028	0.418	0.102
Mean	335,231.65	2,166.78	304,857.23	509,731.11
Pct. Change	0.05	1.68	-0.10	-3.54

Notes: This table reports estimates based on Equation 1 using child's *APOE-e4* carrier status as measure of genetic risk for women aged 45 to 65 (Panel A) and women aged 55 to 65 (Panel B) for the re-weighted sample of women after entropy balancing. In column (1), the outcome is earnings, in column (2), the outcome is income from shares, in column (3), the outcome is disposable income, and in column (4), the outcome is net wealth excluding pension wealth. All monetary values are in DKK, 2023-prices. Control variables included are age fixed effects, year fixed effects, the first 10 principal components, education dummies, marriage dummy, experience, experience squared, and dummy for whether child is in iPSYCH control group or not. N refers to the number of individuals, and $N \times Years$ refers to the total number of individual-year observations. Standard errors are clustered at the individual level and shown in parentheses. Significance levels are denoted as * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

A.5 Instrumental Variables

Our proxy-phenotype design introduces measurement error. Using another AD PGS to instrument for our primary AD PGS (phen19) could potentially address this measurement error given the following assumptions hold:

1. Relevance
2. Exclusion restriction

While the first assumption requires that the second PGS (our instrument) must be sufficiently strongly correlated with our primary AD PGS (our endogenous variable) and is testable, the second assumption requires the instrument to only affect outcomes exclusively via its effect on the endogenous variable. This means that the measurement error of the second PGS cannot be correlated with the measurement error of our primary PGS. For women, we instrument our primary AD PGS using ga3646 (AD PGS: illness of mother). For men, we use ga3635 (AD PGS: illness of father). As our primary AD PGS and these PGSs are derived from two separate GWASs using different large samples (Table 1), it is plausible that the exclusion restriction holds. Hence, we estimate the following:

$$X_i = \pi_0 + \pi_1 Z_i + \pi_2 W_i + \varepsilon_i \qquad \text{(First Stage)}$$

$$Y_i = \beta_0 + \beta_1 \widehat{X}_i + \beta_2 W_i + u_i \qquad \text{(IV)}$$

where Y_i is health and economic outcomes, X_i is our endogenous variable (AD PGS), Z_i is the instrumental variable (ga3646 for women, ga3635 for men), W_i is our control variables (as explained in Section 4), and ε_i, u_i are the error terms.

We empirically test the relevance assumption in Table A.31. Motivated by a strong first stage, we then estimate our IV regressions in Tables A.32-A.36.

Table A.31: First Stage

	2000-2020		2005-2020		2008-2020	
	Age 45-65	Age 55-65	Age 45-65	Age 55-65	Age 45-65	Age 55-65
	(1)	(2)	(3)	(4)	(5)	(6)
Panel A: Women						
ga3646	0.647***	0.648***	0.647***	0.648***	0.647***	0.648***
	(0.003)	(0.003)	(0.003)	(0.003)	(0.002)	(0.003)
$N \times$ *Years*	1,229,258	382,779	1,123,485	375,336	1,005,275	360,716
N	106,735	62,063	106,374	61,992	105,896	61,814
R^2	0.427	0.429	0.426	0.429	0.426	0.428
F	1,097.82	729.89	1,227.16	812.97	1,326.74	871.04
Panel B: Men						
ga3635	0.604***	0.605***	0.604***	0.604***	0.604***	0.604***
	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)
$N \times$ *Years*	1,243,045	465,012	1,084,496	440,274	940,718	407,980
N	101,228	67,010	99,993	66,369	98,560	65,436
R^2	0.373	0.374	0.373	0.373	0.373	0.373
F	847.06	651.93	929.39	709.76	985.71	741.19

Notes: This table reports first stage estimates for ga3646 (AD PGS: illness of mother) for women (Panel A) and ga3635 (AD PGS: illness of father) for men (Panel B). In columns (1)-(2), the time period is 2000-2020. In columns (3)-(4), the time period is 2005-2020. In columns (5)-(6), the time period is 2008-2020. Control variables included are age fixed effects, year fixed effects, the first 10 principal components, education dummies, marriage dummy, experience, experience squared, and dummy for whether child is in iPSYCH control group or not. N refers to the number of individuals, and $N \times$ *Years* refers to the total number of individual-year observations. Standard errors are clustered at the individual level and shown in parentheses. Significance levels are denoted as * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table A.32: IV Regressions: AD PGS and Health Outcomes

	Dementia		GP visits	
	Age 45-65	Age 55-65	Age 45-65	Age 55-65
	(1)	(2)	(3)	(4)
Panel A: Women				
AD PGS	0.00014***	0.00031***	0.01541	0.05784**
	(0.00004)	(0.00009)	(0.01637)	(0.02428)
$N \times$ *Years*	1,123,485	375,336	1,123,485	375,336
N	106,374	61,992	106,374	61,992
R^2	0.000	0.000	0.043	0.035
Mean	0.0002	0.0004	4.33	4.35
Pct. Change	80.44	83.40	0.36	1.33
Panel B: Men				
AD PGS	0.00007**	0.00017**	0.01765	0.03594
	(0.00003)	(0.00007)	(0.01617)	(0.02346)
$N \times$ *Years*	1,084,496	440,274	1,084,496	440,274
N	99,993	66,369	99,993	66,369
R^2	0.000	0.000	0.031	0.027
Mean	0.0002	0.0003	3.03	3.43
Pct. Change	39.49	47.93	0.58	1.05

Notes: This table reports estimates based on IV regressions instrumenting child's AD PGS using ga3646 for women (Panel A) and ga3635 for men (Panel B). In columns (1)-(2), the outcome is a dummy for diagnosed dementia. In columns (3)-(4), the outcome variable is number of GP visits. Control variables included are age fixed effects, year fixed effects, the first 10 principal components, education dummies, marriage dummy, experience, experience squared, and dummy for whether child is in iPSYCH control group or not. N refers to the number of individuals, and $N \times$ *Years* refers to the total number of individual-year observations. Standard errors are clustered at the individual level and shown in parentheses. Significance levels are denoted as * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table A.33: IV Regressions: AD PGS and Labor Market Attachment for Women

	Employment	Unemployment	DP	Transfers	Pension
	(1)	(2)	(3)	(4)	(5)
Panel A: Age 45-65					
AD PGS	-0.00227*	0.00023	0.00184	0.00019	0.00002
	(0.00130)	(0.00036)	(0.00124)	(0.00075)	(0.00061)
$N \times Years$	1,005,275	1,005,275	1,005,275	1,005,275	1,005,275
N	105,896	105,896	105,896	105,896	105,896
R^2	0.364	0.007	0.308	0.061	0.212
Mean	0.75	0.02	0.12	0.06	0.05
Pct. Change	-0.30	0.97	1.48	0.31	0.05
Panel B: Age 55-65					
AD PGS	-0.00552***	0.00021	0.00497**	0.00047	-0.00013
	(0.00207)	(0.00052)	(0.00194)	(0.00090)	(0.00126)
$N \times Years$	360,716	360,716	360,716	360,716	360,716
N	61,814	61,814	61,814	61,814	61,814
R^2	0.346	0.007	0.335	0.037	0.253
Mean	0.68	0.02	0.16	0.04	0.10
Pct. Change	-0.81	0.95	3.16	1.11	-0.14

Notes: This table reports estimates based on IV regressions instrumenting child's AD PGS using ga3646 for women aged 45 to 65 (Panel A) and women aged 55 to 65 (Panel B). In column (1), the outcome is a dummy for being employed, in column (2), the outcome is a dummy for being unemployed, in column (3), the outcome is a dummy for receiving disability pension, in column (4), the outcome variable is a dummy for receiving transfers, and in column (5), the outcome variable is a dummy for pension. Control variables included are age fixed effects, year fixed effects, the first 10 principal components, education dummies, marriage dummy, experience, experience squared, and dummy for whether child is in iPSYCH control group or not. N refers to the number of individuals, and $N \times Years$ refers to the total number of individual-year observations. Standard errors are clustered at the individual level and shown in parentheses. Significance levels are denoted as * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table A.34: IV Regressions: AD PGS, Income, and Wealth for Women

	Earnings	Income from Shares	Disposable Income	Wealth
	(1)	(2)	(3)	(4)
Panel A: Age 45-65				
AD PGS	-383.26	3.49	-4.14	-1,343.83
	(652.69)	(26.81)	(382.95)	(3,844.18)
$N \times$ *Years*	1,229,258	1,229,258	1,229,258	1,229,258
N	106,735	106,735	106,735	106,735
R^2	0.507	0.024	0.391	0.099
Mean	308,707.80	1,313.37	285,150.85	274,620.24
Pct. Change	-0.12	0.27	-0.00	-0.49
Panel B: Age 55-65				
AD PGS	-638.413	-18.336	29.519	-5,040.880
	(981.935)	(48.455)	(572.523)	(6,230.197)
$N \times$ *Years*	382,779	382,779	382,779	382,779
N	62,063	62,063	62,063	62,063
R^2	0.499	0.025	0.389	0.090
Mean	284,489.22	1,876.72	285,440.72	432,197.07
Pct. Change	-0.22	-0.98	0.01	-1.17

Notes: This table reports estimates based on IV regressions instrumenting child's AD PGS using ga3646 for women aged 45 to 65 (Panel A) and women aged 55 to 65 (Panel B). In column (1), the outcome is earnings, in column (2), the outcome is income from shares, in column (3), the outcome is disposable income, and in column (4), the outcome is net wealth excluding pension wealth. All monetary values are in DKK, 2023-prices. Control variables included are age fixed effects, year fixed effects, the first 10 principal components, education dummies, marriage dummy, experience, experience squared, and dummy for whether child is in iPSYCH control group or not. N refers to the number of individuals, and $N \times$ *Years* refers to the total number of individual-year observations. Standard errors are clustered at the individual level and shown in parentheses. Significance levels are denoted as * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table A.35: IV Regressions: AD PGS and Labor Market Attachment for Men

	Employment	Unemployment	DP	Transfers	Pension
	(1)	(2)	(3)	(4)	(5)
Panel A: Age 45-65					
AD PGS	-0.00017	0.00068	-0.00081	-0.00030	0.00060
	(0.00140)	(0.00045)	(0.00115)	(0.00067)	(0.00069)
$N \times Years$	940,718	940,718	940,718	940,718	940,718
N	98,560	98,560	98,560	98,560	98,560
R^2	0.251	0.013	0.187	0.051	0.166
Mean	0.81	0.03	0.07	0.04	0.05
Pct. Change	-0.02	2.41	-1.12	-0.77	1.18
Panel B: Age 55-65					
AD PGS	-0.00145	0.00063	-0.00064	0.00004	0.00142
	(0.00212)	(0.00061)	(0.00171)	(0.00082)	(0.00130)
$N \times Years$	407,980	407,980	407,980	407,980	407,980
N	65,436	65,436	65,436	65,436	65,436
R^2	0.240	0.011	0.196	0.038	0.189
Mean	0.75	0.03	0.09	0.03	0.09
Pct. Change	-0.19	2.29	-0.67	0.12	1.56

Notes: This table reports estimates based on IV regressions instrumenting child's AD PGS using ga3635 for men aged 45 to 65 (Panel A) and men aged 55 to 65 (Panel B). In column (1), the outcome is a dummy for being employed, in column (2), the outcome is a dummy for being unemployed, in column (3), the outcome is a dummy for receiving disability pension, in column (4), the outcome variable is a dummy for receiving transfers, and in column (5), the outcome variable is a dummy for pension. Control variables included are age fixed effects, year fixed effects, the first 10 principal components, education dummies, marriage dummy, experience, experience squared, and dummy for whether child is in iPSYCH control group or not. N refers to the number of individuals, and $N \times Years$ refers to the total number of individual-year observations. Standard errors are clustered at the individual level and shown in parentheses. Significance levels are denoted as * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table A.36: IV Regressions: AD PGS, Income, and Wealth for Men

	Earnings	Income from Shares	Disposable Income	Wealth
	(1)	(2)	(3)	(4)
Panel A: Age 45-65				
AD PGS	-419.65	-155.30	37.75	-5,214.72
	(1,183.11)	(164.32)	(817.25)	(7,248.41)
$N \times$ *Years*	1,243,045	1,243,045	1,243,045	1,243,045
N	101,228	101,228	101,228	101,228
R^2	0.396	0.021	0.231	0.076
Mean	415,563.64	7,977.01	347,974.46	496,335.66
Pct. Change	-0.10	-1.95	0.01	-1.05
Panel B: Age 55-65				
AD PGS	-1,424.96	-254.43	-1,261.27	-15,447.14
	(1,556.84)	(232.79)	(1,111.51)	(10,704.30)
$N \times$ *Years*	465,012	465,012	465,012	465,012
N	67,010	67,010	67,010	67,010
R^2	0.403	0.021	0.235	0.073
Mean	373,107.11	9,046.00	351,391.72	710,473.37
Pct. Change	-0.38	-2.81	-0.36	-2.17

Notes: This table reports estimates based on IV regressions instrumenting child's AD PGS using ga3635 for men aged 45 to 65 (Panel A) and men aged 55 to 65 (Panel B). In column (1), the outcome is earnings, in column (2), the outcome is income from shares, in column (3), the outcome is disposable income, and in column (4), the outcome is net wealth excluding pension wealth. All monetary values are in DKK, 2023-prices. Control variables included are age fixed effects, year fixed effects, the first 10 principal components, education dummies, marriage dummy, experience, experience squared, and dummy for whether child is in iPSYCH control group or not. N refers to the number of individuals, and $N \times$ *Years* refers to the total number of individual-year observations. Standard errors are clustered at the individual level and shown in parentheses. Significance levels are denoted as * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.