Intergenerational Effects of Genetic Risk for Dementia on Socio-Economic Outcomes

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Abstract

Dementia is a prevalent and costly disease, but its effects on individuals and families' economic outcomes are poorly understood. Existing papers have used the timing of disease diagnosis in an event study framework to estimate the effect of disease onset on work, income and wealth. One limitation of such approach is that dementia is difficult to diagnose and whether and when an individual gets diagnosed is endogenous. In this paper, we take a different route and focus on the genetic risk for dementia. Dementia has a strong genetic basis and we investigate how this genetic risk is related to early, mid, and late-life economic outcomes for individuals and their family members. To do that, we construct a novel data set that merges genetic and survey-based data from the Dutch Lifelines Biobank with dementia diagnosis records, longitudinal tax and health expenditure data from national-level administrative sources. We show that our constructed measure of genetic risk for dementia is predictive of dementia diagnosis from administrative data, but only after age 70, when dementia prevalence begins to increase sharply. Further, genetic risk for dementia is unrelated to socio-economic background in early and mid-life, contrary to what we find if we use dementia diagnosis. Preliminary results indicate that genetic risk for dementia has limited effects on the earnings, income and wealth of genotyped individuals, but that it has a substantial negative effect on the wealth of their offspring. As parents age, offspring of parents in the top tercile of genetic risk see a reduction in wealth of 15% when compared to those with parents in the lower tercile of genetic risk. In ongoing work we have been investigating the mechanisms behind such reductions in wealth, including the provision of informal care by offspring, payments related to long-term health care, and real estate decisions based on geographic distance between parents and offspring.

Keywords: ADRD, polygenic scores, socio-economic status, families.

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1 Introduction

In 2021, 57 million people were estimated to be living with Alzheimer disease and related dementia (ADRD) worldwide, with 9.9 million new cases expected per year until 2050(Organization, 2020). The sharp predicted increase in dementia prevalence has important consequences for affected individuals and their families, as well as for governments around the world. Cognitive impairment, even in the absence of a dementia diagnosis, is often associated with limitations in an individual's ability to work, manage finances, and deal with routine activities, with implications for not only affected individuals, but also their spouses and offspring[1]. It is crucial to have credible estimates of the costs of dementia, financial and otherwise, for individuals and their families, so we can appropriately predict and better prepare for such increasing costs.

A challenge of estimating the economic and health costs of dementia is determining ADRD diagnosis and progression. Dementia is an umbrella term that encompasses a range of mild to severe symptoms found in people living with ADRD. A detailed clinical assessment examining symptoms, risk factors, and laboratory results is necessary for diagnosis, which can be separated into different ADRD types such as Alzheimer's Disease (AD), vascular dementia, and Lewy body dementia, amongst others. Alzheimer's Disease, present in around 60% dementia cases, is a progressive neurodegenerative disorder with a preclinical or prodromal phase now detectable using biomarkers — referred to as Core 1 biomarkers — which capture biological changes in the brain, specifically amyloid β pathology, that appear years or even decades before cognitive symptoms emerge (Dubois et al., 2016). This preclinical phase is followed by a second stage of mild cognitive impairment, where symptoms such as memory lapses, reductions in language and visuospatial abilities, and difficulty solving problems are present, but individuals remain able to live independently. In a third stage, cognitive symptoms become severe and individuals lose the ability to live indepen-

[1]A substantial literature on the economic and health effects of ADRD exists, see (Hurd et al., 2013; Coe et al., 2018) for reviews on the effects of caregivers see (Pinquart & Sörensen, 2003; Neubert et al., 2021)

dently, representing clinical Alzheimer's disease dementia. Diagnosis across these stages can draw on standardised clinical assessments[2] alongside Core 2 biomarkers — capturing tau pathology — which together with Core 1 biomarkers substantially increase the probability of clinical dementia (Jack Jr et al., 2024). Dementia is typically diagnosed only in this final stage where symptoms are most visible. Even in a world with perfect measurement of preclinical biomarkers and symptoms, the question of when exactly AD dementia should be diagnosed remains unresolved — while Core 1 biomarkers represent a necessary condition for AD development, they are not sufficient for clinical diagnosis, as a significant proportion of amyloid positive older individuals will never develop dementia (Livingston et al., 2024). The current guidance is that biomarkers are sufficient for detecting AD pathology (Jack Jr et al., 2024), while clinical guidance recommends their use in conjunction with other information — including risk factors and standardised diagnostic tools — when assessing AD dementia (Livingston et al., 2024). Thus, properly accounting for the full consequences of ADRD requires covering individuals and their families across the entire disease continuum — including the preclinical phase spanning years or decades before symptom onset — and must account for the substantial proportion of those living with ADRD who remain undiagnosed.

The second difficulty is the complex relationship between the development of ADRD and socio-economic status. The most important risk factor for ADRD is age (Dubois et al., 2016), and people high socio-economic status live longer. However, other known risk factors for ADRD include physical inactivity, unbalanced diets, tobacco use, hypertension and cognitive inactivity which are typically associated with low socio-economic status. Indeed, low educational attainment is often indicated as the main risk factor for ADRD (Norton et al., 2014). The causal relationship between these risk factors is also hard to assess; the direction of causation can go both ways with ADRD causing, for example, reductions in physical activity, or physical activity increasing ADRD or both happening concurrently. Randomised control trials (RCTs) have been used to elucidate some of the causal pathways from these

[2]Such as the Mini-Mental State Examination (MMSE) and the Montreal Cognitive Assessment (MoCA), amongst others

risk factors to ADRD. A meta-analysis of 12 RCTs involving 96,158 participants with a mean follow-up of 4.1 years found that antihypertensive medications were associated with lower risk of dementia or cognitive impairment compared to controls (Hughes et al., 2020). An umbrella review of RCTs on the effects of physical exercise on cognition found consistent small cognitive benefits across multiple studies, suggesting exercise does confer measurable cognitive benefit even if its specific effect on dementia incidence remains less clearly established (Ciria et al., 2023). A 2023 Cochrane review of 25 RCT studies with 1,893 participants found that cognitive stimulation of people living with dementia resulted in improvements in cognitive test results (Woods et al., 2023). Multidomain Interventions that includes cognitive training, diet and exercise had positive effects on cognition, although they were too imprecise for dementia (Ngandu et al., 2015). In fact, in the latest Lancet Commission dementia(Livingston et al., 2024), using the evidence described above and others, now has a list of 14 risk factors, the majority of which are related to SES, for dementia.

In this article we leverage on individuals genetic risk of developing ADRD as a measure of ADRD burden and administrative data on income, wealth and health to analyse the economic and health effects of genetically driven dementia on individuals and their offspring. We argue that consequences of elevated genetic risk for ADRD are substantively important in their own right — individuals with high genetic predisposition may face a fundamentally different life course trajectory, making different decisions about labour supply, retirement, savings, and insurance and placing different demands on family members and caregivers. It is established that measures of genetic risk of ADRD are highly predictive of ADRD diagnosis and symptoms (Kunkle et al., 2019; Wehby et al., 2018; Shin et al., 2020; Escott-Price et al., 2015). ADRD genetic risks also predicts disease progression (Jack Jr et al., 2015; Tan et al., 2017), and there is now emerging evidence that genetic risk of ADRD effects can operate independently of clinical diagnosis on cognition and economic outcomes (Jeong et al., 2024). Moreover, although genetic effects are generally still confounded by SES, from the fact that genes your parents pass on to you also correlate with the environment they construct (Kong

et al., 2018)- measures of genetic risk to ADRD are found to have little SES confounding (Barcellos et al. 2025). Together these considerations motivate the use of genetic risk as the primary measure of ADRD burden in the present study. Further the use of admistrative data on wealth and income of individuals and families allows us to follow individuals from the preclinical AD phase and is not subject to selective attrition as survey data.

The literature on the economic and health impact of ADRD on individuals and families has typically observational cohors studies and compared mean outcomes of individuals who developed ADRD versus those who never did (Cribb et al., 2025). However, diagnosis data suffers from endogeneity- where individuals who are diagnosed with ADRD are systematically difference to those never diagnosed, for example, diagnosis is related to the ability of individuals to seek and receive adequate medical care. Recognising this problem a body of literature has used causal inference methods to estimate ADRD effects; most often, these studies have employed propensity score matching techniques (Aye et al., 2024; Frahm-Falkenberg et al., 2016; Altındağ et al., 2026). (Frahm-Falkenberg et al., 2016) using Danish data finds that ADRD is associated with lower incomes and higher morbidity rates.(Altındağ et al., 2026) uses PSM together with two-way fixed effect methods and administrative data from Denmark to find that having a parent with ADRD does not affect income and physical health of children with the exception of mental health for daughters. A methodological concern in studies examining the consequences of ADRD using propensity score matching (PSM) relates to the treatment of socioeconomic status as a control variable. Given the well-established bidirectional causal relationship between SES and ADRD — whereby lower SES increases dementia risk through reduced cognitive reserve, limited healthcare access, and greater exposure to modifiable risk factors, while ADRD simultaneously erodes SES through reduced earning capacity, wealth depletion, and early labour market exit — controlling for SES in PSM models may constitute term a bad controls problem, blocking legitimate causal pathways through which ADRD exerts its effects. Critically, the extended prodromal phase of ADRD, which may span more than a decade before any symptoms, means that measured

SES at or near the time of matching may already reflect the downstream consequences of subclinical cognitive decline rather than representing a true pre-exposure confounder. We contribute to this literature by using administrative datasets starting when individuals are age 50, ensuring that we can obtain estimates of effects decades before a potential diagnosis takes place, and avoid the problem of selective attrition due to cognitive loss. Administrative data also ensures we have the universe of children from affected individuals.

Similarly to this article, an emerging literature explores genetic risk of ADRD to estimate the economic and health costs of dementia (Shin et al., 2020; Jeong et al., 2024; Wehby et al., 2018). (Borgbjerg et al., 2025), following our strategy closely and using administrative data from Denmark, but focusing on relatively young individuals aged 45-65, finds that ADRD genetic risk is associated with lower labour market participation and pension uptake. Using data from the USA, (Jeong et al., 2024) shows that increased genetic risk for ADRD was associated with negative economic outcomes and lower cognitive function even in the absence of an ADRD diagnosis, and despite these outcomes, individuals with higher genetic risk were less likely to financially plan. We contribute to this literature by relying on large administrative datasets that spans ages from 50-85 and we go beyond individuals by also analysing the intergenerational effects of ADRD.

2 Background

2.1 The genetics of ADRD

Many complex diseases result from the consideration of small additive linear effects across the genome. For these traits, genetic susceptibility is well captured by Polygenic Indices (PGI), which consists of a sum of weighted genetic variants across an individual. However, for ADRD three main alleles of the APOE gene ($\varepsilon 4, \varepsilon 3$ and $\varepsilon 2$) carry differential non-linear risks[3]. We

[3]Interestingly, ADRD also has a deterministic gene, individuals who carry an autosomal dominantly inherited AD (ADAD) mutation in the amyloid precursor protein (APP), presenilin 1 (PSEN1) or presenilin 2 (PSEN2) will develop the disease with near 100% and time of onset in predictable (Johnson et al., 2023)

follow Ware et al. (2020) and construct both a polygenic score that excludes the APOE region and treat the APOE locus as an independent covariate when modeling dementia. Specifically, we use the publicly available weights of Kunkle et al. (2019), and excluded the we omitted all single nucletoid polyphormisms around Chromosome 19 in the region 44.4-46.5Mb where the APOE gene lies. [4] We model the APOE locus as described in Lumsden et al. (2020), categorising into 6 categories APOE genotype was $\varepsilon 3\varepsilon 3, \varepsilon 3\varepsilon 4, \varepsilon 2\varepsilon 3, \varepsilon 2\varepsilon 4, \varepsilon 4\varepsilon 4$. Other combination for the APOE genotype were excluded because they are are not observed in the Lifelines sample.

2.2 The Netherlands ADRD context

Long-term care (LTC) expenditure varies substantially across countries, reflecting differences in the generosity of public systems, eligibility thresholds, and the balance between formal and informal care provision. Drawing on cross-country comparisons across ten high-income countries, Gruber et al. (2023) document that in 2019 an average of 2.1% of GDP was spent on formal LTC, with a strong positive correlation between the public share of funding and total LTC spending as a share of GDP. The Netherlands stands out as a clear outlier: Bakx et al. (2023) show that no other country spends more per capita on publicly financed formal care, with LTC expenditure reaching 4.4% of GDP — more than mean.

[4]Following the standard practice in social genomics, the sample is restricted to participants with European genetic ancestries.The data providers flagged 249 genotyped respondents as being of non-European ancestry, as based on a combination of variables reported by the respondents and the genetic data, assessing ancestry using a principal components analysis. We do not include non-European ancestry individuals because of the *problem of portability:* due to differing *linkage disequlibrium* patterns among groups of different ancestry, constructing a PGI based on GWAS results estimated in a different ancestry group will result in large measurement error of this PGI and lower predictiveness as a result Bitarello & Mathieson (2020); Privé et al. (2022) We standardize the PGI using its cross-sectional standard deviation (as computed among all 78,038 for which the PGI could be constructed).

3 Data

To conduct our analysis, we merge genetic and survey data from the Dutch Lifelines Cohort Study with administrative tax records and dementia records made available by Statistics Netherlands (CBS). The tax records include detailed data on both income and wealth for the entire population of the Netherlands. The healthcare data provides information on dementia and related diseases diagnosis.

3.1 The Dutch Lifelines Biobank

Lifelines is a multi-disciplinary prospective population-based cohort study examining in a unique three-generation design the health and health-related behaviours of 167,729 persons living in the North of the Netherlands (i.e., in the provinces of Drenthe, Friesland and Groningen). It employs a broad range of investigative procedures in assessing the biomedical, socio-demographic, behavioural, physical and psychological factors which contribute to the health and disease of the general population, with a special focus on multi-morbidity and complex genetics. Lifelines' sample size corresponds to about 10% of this region's population. The first wave of data was collected during 2006 and 2013. Sampling occured according to a two-step procedure Scholtens et al. (2015). First, general practitioners recruited individuals of ages 25 to 49. These original participants were then encouraged to invite their family members to also enroll in the study (no age restrictions applied to these family members). Every 5 years since the first wave. For this study, we therefore had completed data of waves 1, 2, and 3 at our disposal.

3.1.1 Genetic Data in Lifelines

To date, abouts 80,000 Lifelines participants have been genotyped. Study participants were genotyped using blood samples drawn during the first assessment visit. We subjected the genetic data sets to standard quality control steps, in order to ensure that only well-measured

SNPs were included in the analysis (see, van Alten et al. (2025) for a more detailed discussion). These quality control checks imply that in total, we had ~ 6.4 million well-measured SNPs of 78,038 Lifelines respondents at our disposal.

3.1.2 Data on Cognitive ability in Lifelines

Most outcome variables, inlcuding dementia diagnosis, are assessed using administrative records (see section 3.2). However, to assess whether ADRD-related genetic risk already affects cognitive ability prior to, or independent of, receiving a dementia diagnosis, we complement our administrative outcomes with measures of cognitive ability collected in Lifelines. During sampling at baseline (2006-2013), GPs were asked to exclude those with severe mental illness or those unable to fill in questionnaires from participation. Further, few people were of old age at baseline, with only 602 respondents being above the age of 75. Because of these reasons, we focus on the effects of ADRD genetic risk on cognitive ability mesaures collected in wave 2 and 3. The following tests were used to collect measures of cognitive ability in Lifelines:

Cogstate: Cogstate assesses broad cognitive functioning. The test was conducted in wave 2 and wave 3 by all participants of those waves. Digital representations of playing cards were shown, and respondents had to answer questions about these cards as quickly as possible, without making mistakes, responding either "yes" or "no". We used the number of correct responses across all domains as our overall measure of cognition, and standardized this variable to have mean zero and standard deviation of one.

Mini Mental State Examination (MMSE): The MMSE was collected in wave 3 amongst all participants aged 65 and older at the time of the visit. It is designed specifically to assess cognitive impairments, and is typically used to screen for dementia. Participants were asked to respond to basic questions such as "What year is it", or asked to perform simple tasks, such as repeating a sentence or copying a figure. A maximum of 30 points could be scored on all tasks combined. We used the number of points as our overall measure

of cognition, and standardized this variable to have mean zero and standard deviation of one.

Ruff Figural Fluency Test (RFFT): The Ruff Figural Fluency Test was collected in wave 3 amongst all adult participants. It measures nonverbal executive functioning. Participants were given sheets with dot patterns, and were asked to draw as many unique designs as possible within a time limit. We used the number of unique designs as our overall measure of cognition, and standardized this variable to have mean zero and standard deviation of one.

3.2 Dutch administrative records

3.2.1 Data on Dementia diagnosis

To assess which Lifelines respondents have ever received a AD-related diagnosis, we use a database of individuals diagnosed with dementia, collected by Vektis. Vektis is a Dutch organization that collects and centralizes health care data in the Netherlands. Vektis has organised and maintained a database of people living with dementia van den Pol (2020). The dataset is constructed using data from health claims involving medication use, hospital care, mental health care, nursing home care and home care. The data contains those diagnosed with dementia starting 2004 until 2025. However, the data set is characterized by undercounting of dementia cases up until 2016. From 2017 onwards, its prevalence of dementia is equal to the expected prevelence in the Dutch population van den Pol (2020). The Vektis gives information on date of first dementia diagnosis, meaning the year in which a person first received a dementia-related care or activity, and current dementia-related care and activities. The data also provide the type of care or indication received.

3.2.2 Data on income and wealth

We use data on annual income (before taxes) and on wealth for the period between 2015 and 2024.[5] Data on annual income are available both at the individual and the household level.[6] Earnings and income from state and private pensions are also available at the individual level. Data on wealth, including its subcomponents, are available at the household level. Net wealth was measured as the total amount of assets of a household, minus its debt. Assets could further be broken down into financial assets, housing assets, business assets, and other assets. Debt could be broken down into mortgage debt or other debt. Financial assets could further be broken down into (i) checkings and savings balances and (ii) bonds and shares.[7]

We convert our measures of income and wealth into rank percentiles calculated within calendar year and year of birth, as compared to the full Dutch population. We also estimate effects in terms of the level of these variables, where income and wealth measures were deflated using the consumer price index, provided by Statistics Netherlands, with 2015 as the base year. For wealth and income data, to reduce the effect of outliers, such levels were winsorized at the value of the 1st and the 99th percentile, as compared to the full population.

3.3 Selection of the main sample and summary statistics

Our final sample consists of Lifelines genotyped individuals, born before 1967, for whom we observe at least one measure of wealth or income between 2015 and 2024, and who have children aged 30 or over during this period. This gives a sample of 24,357 individuals, and their 30,318 children. Summary statistics of these Lifelines respondents are shown in table 3. Summary statistics for the children are shown in table A1

[5] Wealth is measured as of January 1st of each year.

[6] Household income is calculated by aggregating the individual incomes of everyone living in the household. CBS provides a household-specific identifier.

[7] Note that all our measures of wealth do not include pension funds. In the Netherlands, pension funds are not freely transferable and are therefore not considered an asset.

4 Methods

4.1 Predicting Dementia

The first step of our empirical strategy is to construct a measure of genetic susceptibility to Alzheimer's and related diseases (AD).

We decided to estimate two versions of the above regression (i) using own-individual genetic markers on own dementia as an outcome, and (ii) using average children's genetic markers to predict parental dementia. The advantages of (ii) are that we have a sample of individuals who are on average much older and the sample size becomes larger, as we can focus on finding parents of genotyped Lifelines respondents that were born before 1967. The sample size (i) is 35 thousand individuals, roughly born on average in the year of 1955, while the sample size of (ii) is 95 thousand individuals born on average in the year of 1968, with dementia prevalence of around 7.6 per 100 compared to 2.7 in the first sample.

Once we have computed both the AD PGI that excludes the APOE region, and the six categories of APOE risk we estimate the following equation:

$$Y_i(t) = \alpha_0 + \alpha_1 PGI_i + \sum_{m=1}^{5} \delta_m APOE_m + \epsilon_i(t) \tag{1}$$

where i indexes the individual. $Y_i(t)$ is the individual's dementia in year t and PGI_i is their AD PGI. The five APOE categories are included to model for the differential risk of this locus and the base (excluded) category is APOE $\varepsilon 3\varepsilon 3$.

5 Preliminary Results

We show that our constructed measure of genetic risk for dementia is highly predictive of dementia diagnosis from administrative data, but only after age 70, when dementia prevalence begins to increase sharply (Figures 1 and 2, Table 2). By age 80, dementia prevalence is about 50 percent higher in the top tercile risk group versus the two bottom tercile groups.

We see a similar pattern with survey-based measures of cognition, with differences by genetic risk arising as early as age 60 and growing with age (Table 1, Figure 2). We then show that this measure is orthogonal to socio-economic background in early and mid-life (see Figure 3 and Table 3). Being in the top tercile of AD genetic risk is uncorrelated with education, birthweight, midlife measures of cognition, parental health and number of siblings. Genetic risk is associated with parental dementia diagnosis, as expected: those in the top tercile of risk are 27 percent more likely to report a paternal dementia diagnosis and 44 percent more likely to report a maternal diagnosis. Genetic risk is negatively associated with BMI, but the magnitude is small: 0.4 percent of the sample mean. Consistent with the finding that AD genetic risk is orthogonal to early-life factors, we find that, for a subsample of siblings, direct genetic effects are not statistically different than population associations. This is in stark contrast with dementia diagnosis: those ever diagnosed have lower education, income and wealth starting early in life compared to those who were never diagnosed. Results indicate that genetic risk for dementia has limited effects on the earnings, income and wealth of affected individuals (see Figures 4 and 5, Table 3), but that it has a substantial negative effect on the wealth of offspring (see Figures 6 and 7, Table 4). As parents age, offspring of parents in the top tercile of genetic risk (including APOE4 carriers) have average wealth 15 percent (or 20 thousand Euros) lower than those with parents with low genetic risk. Assuming that the effect of genetic risk on offspring wealth works exclusively through dementia diagnosis implies that parental dementia diagnosis reduces offspring wealth by 46K or 30% on average. Most of the reduction in offspring wealth comes from real estate assets (Table 5).

6 Discussion

Our findings that income and wealth of individuals exposed to Alzheimer's disease genetic risk remain unaffected later in life indicate that late-life insurance mechanisms and institutional protections in the Netherlands are sufficient to protect against direct financial losses

of individuals suffering from Alzheimer's and dementia-related diseases. By contrast, substantial negative effects on offspring's wealth suggest that the economic burden of dementia is partially shifted to the next generation, indicating that insurance mechanisms and institutional protections do not fully protect against financial losses in the next generation.

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Table 1: Summary Statistics

Variable	N	Mean	SD	Median
Panel A1: Observations at the person-year level ($N \times T$)				
Gross Income (euro)	236653	34559.4	27082.2	27741
Gross household Income	243100	88045.9	47689.4	81731.7
Net wealth (euro)	236376	289913	343019	202545
Earnings (euro)	236653	28911.4	21283.2	24238.7
State pension (euro)	236653	4514.16	5988.48	0
Household size	236731	2.16	0.83	2
Panel A2: Observations at the individual level (N)				
Dementia	24357	0.03	0.17	0
Age in 2024	24357	68.41	7.62	67
Male	24243	0.37	0.48	0
Education (years)	23717	12.46	2.46	13
Number of Children	24357	2.53	0.91	2
Born in NL	24174	0.99	0.08	1
APOE (e2 alleles)	24357	0.16	0.38	0
APOE (e3 alleles)	24357	1.63	0.56	2
APOE (e4 alleles)	24357	0.22	0.45	0
ADRD PGI (No APOE)	24357	-0.01	0.99	-0.09
Died before 2025	24357	0.06	0.23	0
Age at death	1361	71.14	9.75	72

Notes: The table shows summary statistics for all parents in the main sample. Parents were genotyped Lifelines respondents for whom we could identify at least one child in Dutch administrative data, with the additional requirement that this child was at least 30 years old in 2024. Panel A1 reports variables measured at the person-year level, where N denotes $N \times T$ observations. Panel A2 reports variables measured at the individual level, where N denotes the number of individuals.

Table 2: Predictiveness of the ADRD PGI (excluding APOE) and APOE score on having ever received a dementia diagnosis

	(1) Dementia	(2) Dementia
ADRD PGI (No APOE)	0.698***	0.745***
	(0.115)	(0.143)
Apoe Score	0.633***	0.773***
	(0.207)	(0.261)
Constant	3.131***	2.982***
	(0.0979)	(0.119)
Observations	37,835	24,357
R-squared	0.003	0.004
Outcome Prevalence	3.256	3.137
Mean age 2024	67.52	68.41

Notes: *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$. Robust standard errors in parentheses. The first column includes all Lifelines respondents with genetic data who were born after 1967. The second column includes the subset of those respondents who also have a child older than 30 (the parents in our main sample).

Table 3: Effects of AD Genetic Risk on Own Outcomes

	Dementia	Cognition	Net Wealth	Personal Income	Hhld. Income
Individuals aged 55–74					
Panel A1: outcomes in levels					
Top Tercile Risk	0.00546***	-0.032***	5,310	272.1	-50.03
	(0.00183)	(0.009)	(4,907)	(250.8)	(664.9)
Panel A2: outcomes in percentiles					
Top Tercile Risk			0.283	0.213	-0.257
			(0.343)	(0.333)	(0.330)
Observations	18,741		173,801	174,061	177,500
Individuals at least age 75					
Panel B1: outcomes in levels					
Top Tercile Risk	0.0592***	-0.111***	-6,617	-287.9	-1,414*
	(0.00894)	(0.033)	(10,354)	(445.4)	(840.3)
Panel B2: outcomes in percentiles					
Top Tercile Risk			-0.991	0.412	-0.716
			(0.806)	(0.886)	(0.841)
Observations	5,068	6,021	23,680	23,696	25,932

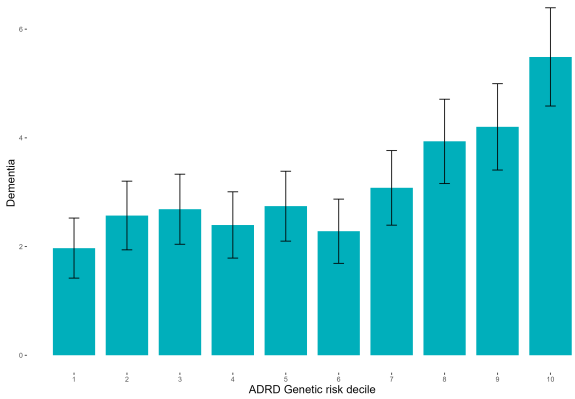
Notes: *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$. The tables show the effect of highest tercile of genetic ADRD risk. Robust standard errors in brackets, clustered on parent ID. All specifications additionally control for parent's sex and the first 10 PCs of the genetic data, year of measurement fixed effects, and age (linearly).

Table 4: Effects of AD Genetic Risk on Children's Outcomes

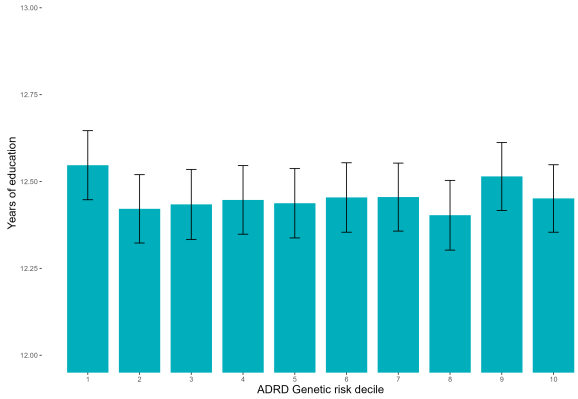
	Net Wealth	Personal Income	Hhld. Income	Financial Assets	Real Estate	Total Assets
Parents aged 55–74						
Panel A: outcomes in levels						
Top Tercile Risk	4,141	-196.7	-278.6		965.4	
	(2,542)	(277.6)	(483.9)		(1,813)	
Panel B: outcomes in percentiles						
Top Tercile Risk	0.438*	-0.183	-0.166	0.341	0.105	0.217
	(0.266)	(0.260)	(0.264)	(0.262)	(0.293)	(0.252)
Observations	270,895	271,501	271,831	270,895	270,895	208,178
Parents at least age 75						
Panel A: outcomes in levels						
Top Tercile Risk	-20,296***	-954	-3,035***		-16,297***	
	(7,736)	(649)	(1,172)		(4,504)	
Panel B: outcomes in percentiles						
Top Tercile Risk	-1.510***	-0.509	-1.387**	-0.691	-1.818***	-1.737***
	(0.549)	(0.539)	(0.557)	(0.541)	(0.592)	(0.542)
Observations	56,866	57,076	57,144	56,866	56,866	56,866

Notes: *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$. The table shows the effect of highest tercile of genetic ADRD risk. Robust standard errors in parentheses, clustered on parent ID. All specifications additionally control for parent's sex, child's sex, first 10 PCs of the genetic data, year of measurement fixed effects, and age (linearly).

Figure 1: Relation Between AD Genetic Risk, Dementia Diagnosis and Years of Education



(a) First figure



(b) Second figure

Figure 2: Difference in Dementia Diagnosis by Age and Genetic Risk Tercile

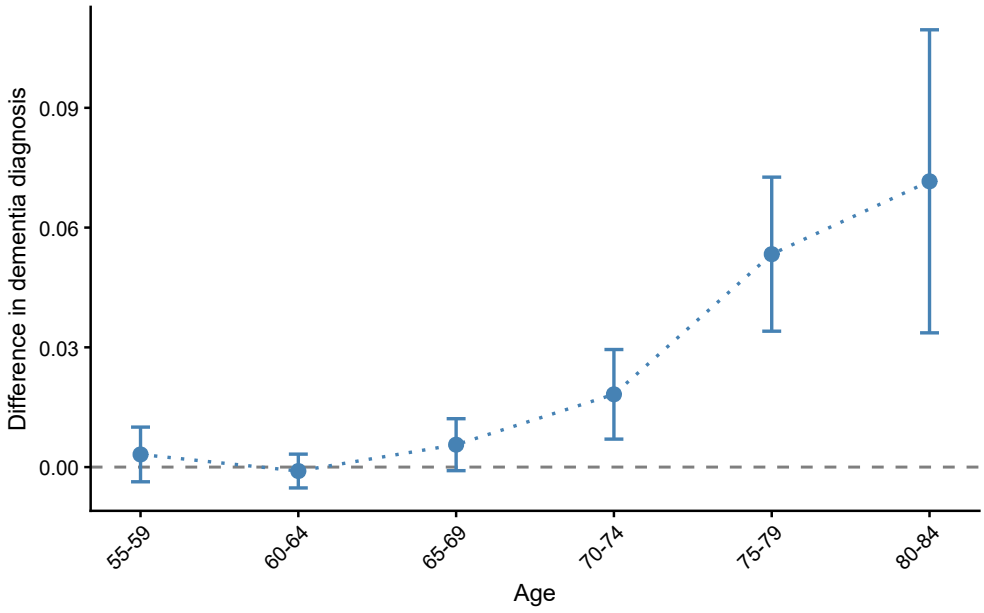


Figure 3: Difference in Cognition by Age and Genetic Risk Tercile

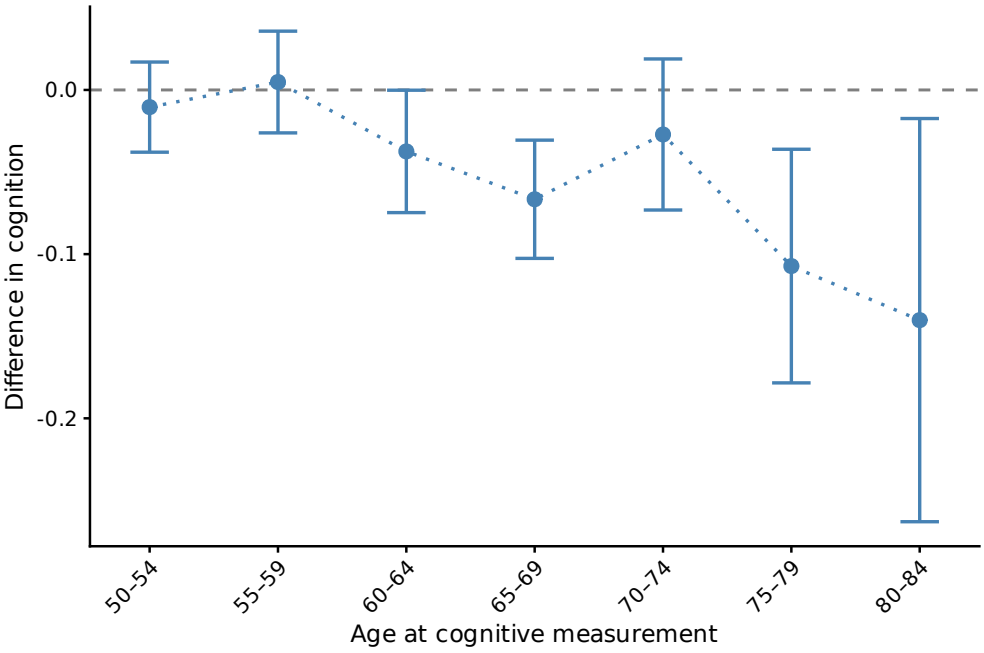


Figure 4: Difference in Household Income by Age and Genetic Risk Tercile

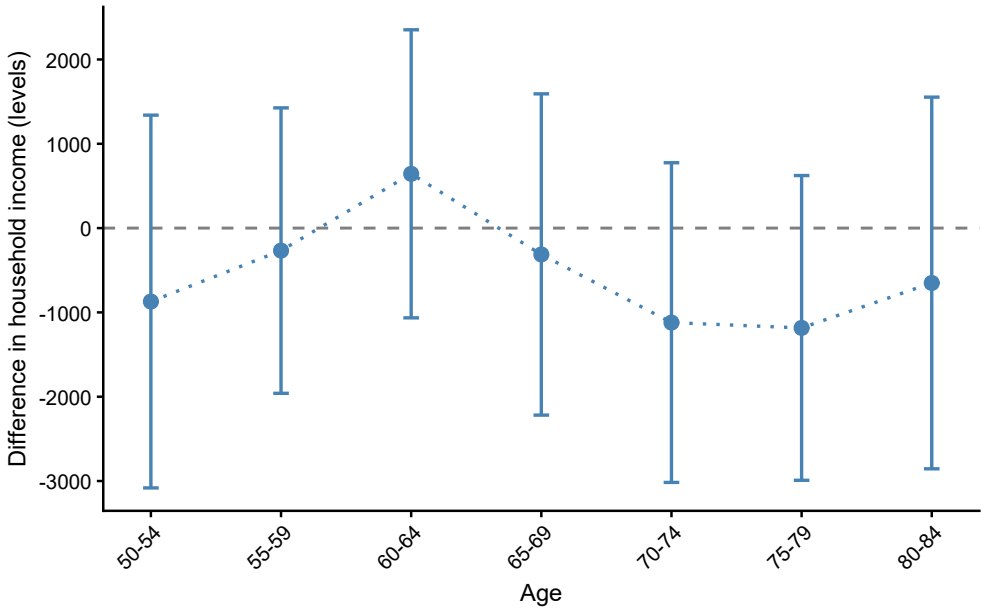


Figure 5: Difference in Net Wealth by Age and Genetic Risk Tercile

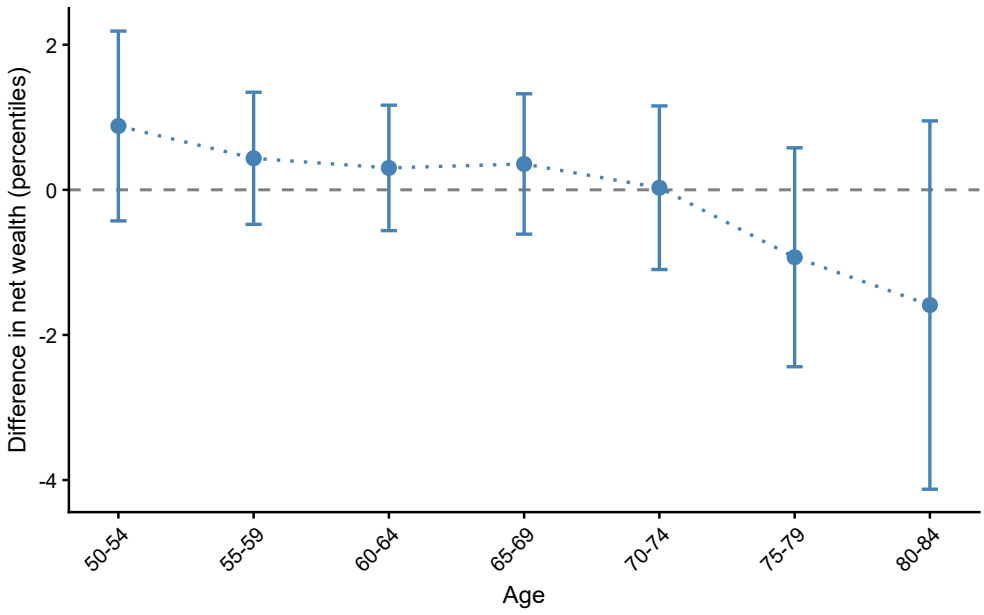


Figure 6: Difference in Children's Household Income by Parental Age and Genetic Risk Tercile

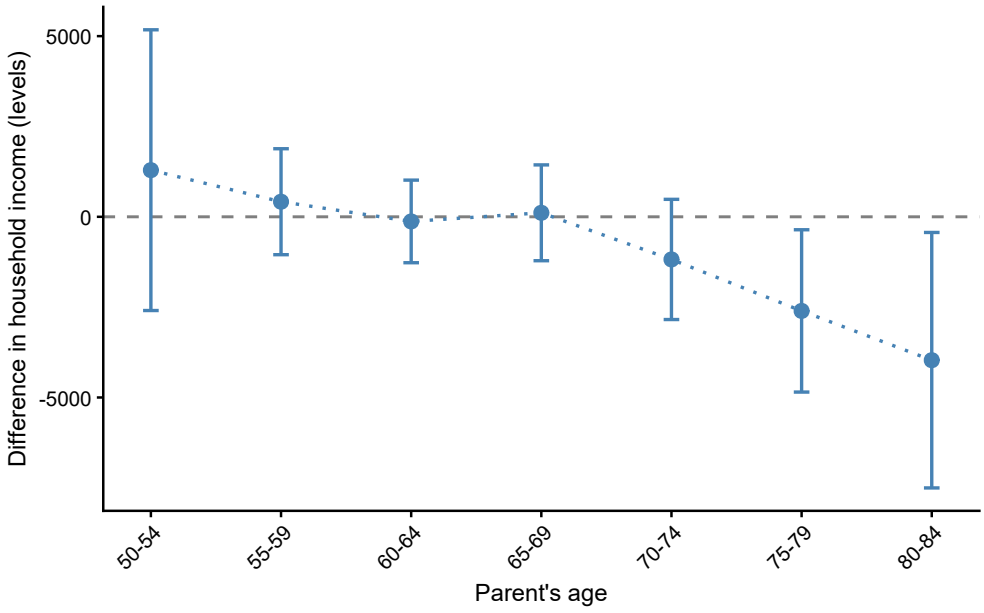
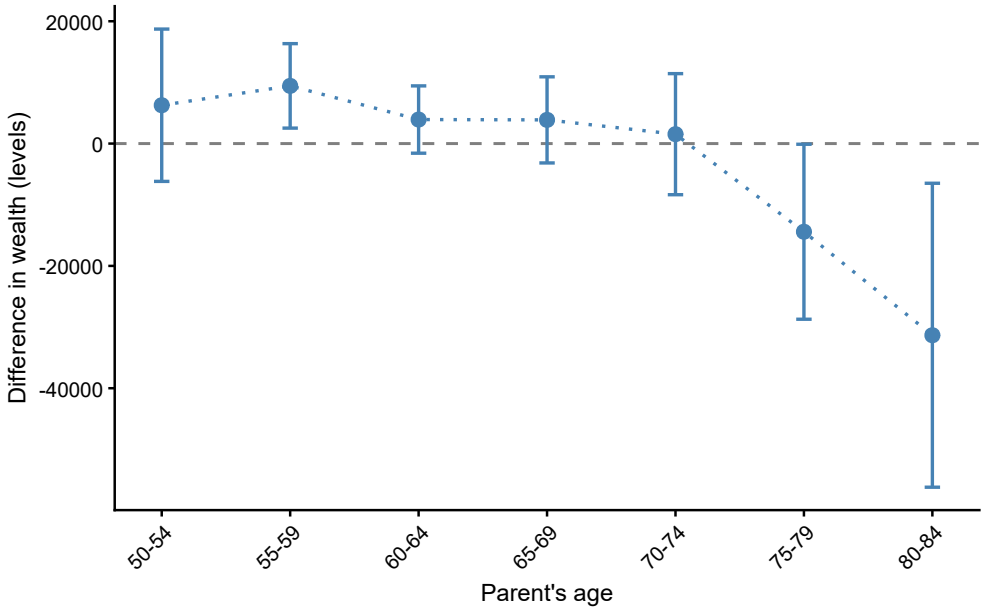


Figure 7: Difference in Children's Net Wealth by Parental Age and Genetic Risk Tercile



Appendix

Table A1: Summary statistics for the children in the main sample

Variable	N	Mean	SD	Median
Gross Income (euro)	395856	50265.5	28854.4	46669
Gross household Income	396309	96659.2	49810.6	90752.7
Net wealth (euro)	394895	163276	284462	82007.2
Earnings (euro)	395856	36360.2	21915.1	32577.2
State pension (euro)	395856	8.97	321.16	0
Household size	396309	3.11	1.36	3
Education (years)	23413	14.65	2.27	16
Male	30318	0.52	0.5	1
Age in 2024	30318	47.57	6.37	47
Number of siblings	29821	1.8	1.09	2

Notes: The table shows summary statistics for children of parents in the main sample. The first panel reports variables measured at the person-year level, where N denotes $N \times T$ observations. The second panel reports variables measured at the individual level, where N denotes the number of individuals.