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Dementia and Long-run Trajectories in Household Finances

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ABSTRACT

Existing evidence suggests that wealth may decline before dementia onset, but the mechanisms underlying these reductions are poorly understood. Using longitudinal data from the Health and Retirement Study, we compare household finance trajectories for individuals who later develop dementia and those who do not. We find that wealth divergence between the two groups is not explained by reduced earnings, higher healthcare spending, intentional “spend-down” to qualify for Medicaid coverage, state-dependent utility, or reverse causation by which wealth declines cause dementia. Instead, our results point to impaired financial decision-making beginning about six years prior to clinically recognizable dementia.

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1. Introduction

Dementia imposes substantial private and social costs through its effects on health, caregiving needs, and financial security (Alzheimer's Association, 2024; Hurd et al., 2013). Because cognitive decline often develops gradually and capabilities related to memory and risk perception are among those lost first, its economic consequences may begin years before symptoms onset or clinical diagnosis. Declining cognitive ability can impair households' capacity to make intertemporal financial choices, manage complex assets, and safeguard wealth against shocks (Ameriks et al., 2022; Angrisani & Lee, 2019; Nicholas et al., 2021). Even modest deterioration in financial decision-making may have compounding effects on savings, investment portfolios, and susceptibility to financial exploitation or fraud, ultimately altering the distribution of resources in later life.

Most existing research on dementia and financial behavior examines cross-sectional associations (Peterson et al., 2014; Sudo & Laks, 2017) or focuses on specific financial decisions—such as portfolio allocation or delegation of financial control within the household (Angrisani & Lee, 2019; Hsu & Willis, 2013). In prior work (Li et al., 2023), we documented substantial declines up to \$100,000 in median net wealth, or about 50% of median at baseline, among older adults who later developed dementia relative to matched controls. However, that study did not examine the mechanisms underlying these losses.

In this paper, we consider six hypotheses that could, separately or jointly, explain the divergence in wealth well before household members might develop dementia. First, cognitive decline may directly affect individual earnings through labor force participation owing to difficulty in carrying out job tasks; this may also affect longer-term pension benefits and Social Security. Second, cognitive decline itself or associated comorbidities could prompt higher out-of-pocket medical spending if the individual sought treatments (Hudomiet et al., 2019; Kelley et al., 2015), which could lead to a depletion of wealth.

Third, cognitively impaired individuals and their families could be aware of their risk of developing dementia and the likelihood of needing long-term care, prompting them to dissave intentionally, possibly in order to qualify for Medicaid coverage of nursing home residence

(“spend-down”).² ³ Fourth, individuals could also increase spending rationally because of state dependent utility: if an individual realizes they are declining cognitively (or physically) they may choose to spend more now under the presumption that they get more utility from a dollar spent now rather than when they are cognitively impaired, limited in what activities they can enjoy (e.g. no travel, theater) or just not remembering enough to enjoy things (Brown et al., 2016).

Fifth, cognitive decline could be associated with a decline in judgment in financial decision-making, leading to poor investment decisions, heightened vulnerability to scams or fraudulent offers, and difficulties managing money, resulting in lower returns or outright losses on financial assets (Ameriks et al., 2022; Christelis et al., 2010; Mazzonna & Peracchi, 2024). In this case in particular, one might expect larger effects of cognition on assets if the individual has not yet been formally diagnosed and fails to recognize the need for assistance with financial tasks. Finally, the empirical pattern may be the result of endogeneity or reverse causality where negative shocks in income or wealth lead to a decline in cognition (Meneton et al., 2015; Schwandt, 2018).

In this study, we use nearly two decades of nationally representative data from the Health and Retirement Study (HRS) and novel dementia probability estimates (Hudomiet et al., 2022) to test these hypotheses for why wealth should begin to decline so many years prior to likely dementia. We examine decisions in various domains, including earnings, investment, consumption, and planning for long-term care. Our classification of predictive dementia leverages the cognitive assessment information available in the HRS and does not rely on presence of a clinical diagnosis, thereby circumventing the concern with the well-documented variability in the timing and likelihood of diagnosis.

These six hypotheses yield distinct empirical predictions regarding income, spending, and the timing of cognitive change in relation to wealth decline, each of which we explore. We employ three main sets of analyses. First, we use propensity score reweighting to compare graphically the trajectories in financial outcomes of interest among those who eventually developed dementia and controls who were observably similar in age, sex, education, marital

² Note that Medicare, the health insurance program that covers nearly all elderly in the US, does not include coverage for most long-term care. While Medicaid does cover long-term care, it is means tested and available only to those with few other resources to pay for care.

³ An additional hypothesis is that people who later develop dementia exhibit lower rates of wealth accumulation all through life. However, this hypothesis was largely rejected by Li et al. (2023), described below.

status and race/ethnicity but whom we do not observe to develop dementia. Second, we rely instead on an event study design with person and calendar year fixed effects to examine relative changes in financial outcomes surrounding dementia onset that are robust to unobserved and time-invariant heterogeneity across individuals. Third, we use the panel vector autoregression (VAR) approach (Stock & Watson, 2001) that is agnostic about the direction of causality to test for Granger causality with regard to both cognitive function and wealth. We additionally employ a few robustness checks as described below.

As in our earlier study using a different dementia measure (Hurd et al., 2013), the cohort of people who ultimately develop dementia experience a decline in wealth that begins at least 6 years prior to “incident dementia”, defined as the survey wave in which the individual’s estimated probability of dementia rises above 50 percent for the first time (with no subsequent reversal).⁴ We find reduced earnings of relatively small magnitude (about \$1,000/year) only at the wave of dementia onset, making it unlikely to be a cause of declining wealth preceding dementia. Using the consumption data in the HRS, we do not find evidence that dementia individuals and their households increase spending, whether to enjoy consumption while they are still able (Brown et al., 2016), because of transferring more resources to children to “spend down” their wealth, or because of out-of-pocket health care expenses prior to the onset of dementia. Indeed, we find that people with cognition declines appear unaware of their risk of needing long-term care as measured by subjective probabilities of moving to a nursing home, so it seems unlikely that they fully anticipate the financial and medical risks associated with dementia. Instead, our results point towards poor financial management. The early wealth decline relative to controls occurs primarily in liquid assets, particularly stocks, bonds, mutual funds and investment accounts—assets that require some degree of cognitive engagement to manage.

One concern with our longitudinal study is reverse causation; that exogenous declines in wealth (for instance due to bad luck in investments) may accelerate declines in cognition (Schwandt, 2018; Wang et al., 2024). While our panel VAR estimates show the probability of cognitive impairment significantly predicts subsequent wealth, we do not find evidence that wealth changes affect cognition. This is consistent with recent evidence showing that

⁴ In our previous study (Li et al., 2023), we found the decline began to occur somewhat earlier; the differences likely arise because we are using the newer measure of dementia (Hudomiet et al., 2022) instead of Hurd et al. (2013).

acceleration in cognitive decline largely precedes rather than follows a large wealth decline (Cho et al., 2025). Taken together, these findings strengthen our confidence that the relationship between cognition and wealth changes arising from poor financial decisions is plausibly causal.

In sensitivity analyses, we leverage the timing of self-reported diagnosis of any memory-related conditions (including but not limited to dementia) to compare the trajectories in wealth for those with such a reported diagnosis preceding or coinciding with their predicted dementia onset versus those without a timely diagnosis. If financial mismanagement underlies the observed wealth losses, we would expect to see such losses mitigated (to an extent) among those reporting a timely diagnosis who are made aware of their cognitive impairment (Mazzonna & Peracchi, 2024). Indeed, we find that wealth difference relative to those without dementia flattens and even reverses after dementia onset among those with a timely diagnosis, whereas the gap widens further after predicted dementia onset among those with late or no diagnosis.

Finally, we show that these patterns are plausibly unique to dementia by conducting parallel analyses comparing individuals developing other major chronic medical conditions (cancer, heart disease, lung disease and arthritis)—conditions which by themselves are not linked with cognitive decline—and finding no evidence of declining wealth among those with these conditions relative to those without.

Our findings contribute to several strands of research on cognitive aging and household finance. A growing body of work links cognitive ability to financial outcomes such as portfolio allocation, credit use, and susceptibility to fraud (Ameriks et al., 2022; Angrisani & Lee, 2019; Boyle et al., 2019; Christelis et al., 2010; Hsu & Willis, 2013; Nicholas et al., 2021). Most recently, Mazzonna and Peracchi (2024) show that older adults are often unaware of their own cognitive decline, and that such misperception can lead to persistent financial mistakes. We extend this literature by showing that the economic consequences of cognitive decline appear well before the onset of dementia symptoms (or clinical diagnosis) and manifest in sustained losses in household wealth. By quantifying the timing, magnitude, and dynamic effects of dementia-related impairment on wealth, we identify behavioral mechanisms underlying early financial decline that are distinct from medical or labor-market channels.

Our results also bridge the literatures on health and household finance by identifying dementia as a distinct mechanism of wealth redistribution. Because losses from impaired decision-making largely represent transfers to counterparties or financial intermediaries (as in the

case of poorly timed market transactions) (Agarwal et al., 2007), the high prevalence of dementia at older age (Hudomiet et al., 2022) has macroeconomic implications for aggregate savings behavior and wealth inequality.

Our study shows that financial mismanagement can and often precedes overt memory symptoms, implying that preventive interventions cannot rely solely on early diagnosis. Rather, the risk of future cognitive decline should be treated as a broad, insurable risk faced by all households. Policies that promote structural safeguards—such as multiple authorized financial contacts, partial wealth protection through annuities and defined-benefit pensions (Brown et al., 2001), or default fiduciary oversight—may help preserve assets and limit inefficient wealth transfers as the population ages.

The rest of the paper proceeds as follows. Section 2 presents a simple theoretical framework to illustrate the hypotheses under consideration. Section 3 describes the HRS data and the analytic approaches. Section 4 presents the main results that either support or refute the hypotheses. Section 5 shows results from sensitivity analyses. Section 6 concludes.

2. Accounting for Wealth Changes: A Simple Framework

Our cognition measure (Hudomiet et al., 2022) translates responses to multiple cognitive assessment questions into a probability of having dementia, so it ranges nearly continuously between 0 and 1. Following (Willis et al., 2023), we consider a simple model of how cognition (and the cognition score) evolves over time. Let m_{it}^* be the true cognition index for individual i at time t (with larger values denoting worse cognition) with m_{it}^* assumed to be a first-order additive Markov process with a variable depreciation rate δ_{it} , so that $m_{it+1}^* = m_{it}^* + \delta_{it}$. The measured cognition index from the HRS, m_{it} , is equal to the true level of cognition plus an error term ε_{it} reflecting mismeasurement both at a point in time, and persistently for individual i ; thus

$$m_{it} = m_{it}^* + \varepsilon_{it} = m_{it-1}^* + \delta_{it} + (\varepsilon_{it} - \varepsilon_{it-1}) \quad (1)$$

Thus, the evolution of the HRS cognition score is a function both of the true depreciation rate δ_{it} and the first-difference of the measurement error term.⁵

⁵ That the individual mismeasurement is not reflected here in the evolution of measured cognition depends on the first-order Markov assumption; it would appear in an autoregressive framework.

Consider next the accounting identity for the change over time in wealth:

$$W_{it+1} = W_{it}(1 + r_{it}(m_{it}^*, d_{it})) + [w_{it}L_{it}(m_{it}^*) + \tau_{it}] - C_{it}(m_{it}^*, d_{it}) \quad (2)$$

where wealth W_{it} measures household wealth for individual i (including wealth held by members of individual i 's family), $r_{it}(m_{it}^*, d_{it})$ is the household rate of return at time t , which we allow to be a function of true cognition m_{it}^* as well as a binary measure of whether the individual has been diagnosed with memory deficits d_{it} ; as noted above when an individual has been diagnosed, it is more likely (though certainly not always) that financial decisions are supervised by a spouse or other family member, turned over to a financial planner, or other safeguards put in place, and thus less likely to be sub-optimal. The rate of return captures both conventional interest and dividend income, as well as capital gains or losses, for example from questionable or fraudulent investment mechanisms. Alternatively, if measurement error ε_{it} is negatively correlated with d_{it} because people with unmeasured cognition loss are more likely to be diagnosed, d_{it} could be negatively associated with wealth.

The term in the brackets represents non-capital income; earnings equal to wage w_{it} times labor supply $L_{it}(m_{it}^*)$, also written as a function of cognition, plus transfer income τ_{it} . Finally, consumption $C_{it}(m_{it}^*, d_{it})$ may be affected by cognition, whether because of higher out-of-pocket health care costs, state-dependent utility, or greater levels of transfers to family members (included in consumption) so as to spend down wealth prior to applying for Medicaid.

Our hypothesis testing is therefore whether the rate of return, labor supply, and consumption (defined broadly) are affected systematically by changes over time in the continuous measure of cognition. To allow for the hypothesis that cognition may itself be affected by wealth, we also allow for the transition term δ_{it} to be a function of W_{it} which we consider below using the panel VAR approach. Note that we are not testing explicitly for other causal pathways such as from labor supply (or consumption) to cognition.⁶

⁶ In theory all variables are endogenous (see Rohwedder and Willis, 2010 for a study of the possible causal effects of retirement on cognition); unfortunately, our data are too noisy to use the VAR approach to test for general Granger causality across a variety of measures. For this reason, we focus on causal pathways that are the most central to our research question and where the evidence suggests the largest effects, i.e. between cognition and wealth (and vice versa).

3. Methods and Data

We use data from the longitudinal Health and Retirement Study (HRS) which conducts biennial interviews of U.S. adults ages 50 and above, collecting comprehensive information on demographics, health, cognition and wealth. We construct a cohort of respondents with probable incident dementia (cases) based on predicted dementia probabilities estimated by Hudomiet et al. (2022) for waves from 2000-2016. They developed a longitudinal, latent-variable model of cognitive status that addressed the limitations of earlier models (particularly biases in dementia measurement for different racial groups) and estimated year-specific dementia probabilities for HRS respondents based on the cognitive assessment information available in the survey. This measure has been validated for a subset of HRS respondents for whom there was a detailed clinical assessment for dementia.⁷ The authors estimated three related probabilities for each HRS respondent in their study: (1) the probability of having dementia, (2) the probability of having mild cognitive impairment, and (3) the probability of being cognitively normal. These probabilities are available for all HRS respondents ages 65 or older; for years 2000 to 2016 there were 21,442 individuals. These individuals form the basis of our analytic sample. Following conventions in the literature, we classify an individual as having dementia in a given HRS wave if their predicted dementia probability is at least 0.5.

We construct a control cohort of individuals who were never classified as having probable dementia in HRS during our observation window and never had a spouse with dementia. For both cases and controls we impose the following inclusion restrictions: 1) between 65 and 100 years old, 2) having been observed in at least two consecutive waves between 2000 and 2016, and 3) continuously enrolled in Medicare (to ensure homogeneity in insurance coverage), and require that these restrictions hold in all the waves we include in the study. In addition, cases were required to have at least one “clean” wave before dementia wave to qualify as having incident dementia. We also exclude a small group of individuals whose dementia status reversed after onset (i.e. we require that the predicted dementia probability remains at or above 0.5 after onset).

In addition to cognitive health and functional status, the HRS collects rich information on various categories of household wealth and income, as well as information on healthcare use and

⁷ This information comes from the Aging, Demographics, and Memory Study (ADAMS) that is a supplement to the HRS.

out-of-pocket health care costs; all of which we use in our study.⁸ For the portions of our analyses relying on household consumption and expenditure, we use data from the Consumption and Activities Mail Surveys (CAMS) supplement to the HRS that collects information on household spending in a variety of categories over the previous 12 months.⁹ Our final analysis sample includes 2,312 cases (13,942 person-waves) and 8,431 controls (43,100 person-waves).

3.1. Inverse Probability Weighting

To compare trends in wealth, income and spending between cases and controls, we apply inverse probability weighting to all person-year observations among controls so they are observably similar to cases in terms of key demographics (described below) for each year relative to dementia onset (or event time) among cases (e.g. $t = -8, -6, -4, -2, 0, 2, 4$). To do so, we estimate a separate logistic regression for each t which includes all observations of controls across all waves and observations of cases from that event time only, with the dependent variable being an indicator of whether the observation belongs to the case group, and with age, gender, race/ethnicity, education and marital status as independent variables.¹⁰ This yields a distinct set of weights for all the control group observations specific to each t , defined as:

$$\hat{w}_{ijt} = \frac{\hat{p}_{ijt}}{1 - \hat{p}_{ijt}}$$

where \hat{p}_{ijt} is the estimated propensity of being in the case group for control individual i in HRS wave j from the logistic regression specific to year t . Then, for each year t relative to dementia onset among cases, we apply the re-weighting such that each control observation receives a weight of \hat{w}_{ijt} , each case observation in Year t receives a weight of 1, and each case observation in years other than t receives a weight of 0.¹¹

⁸ Whenever possible, we rely on the RAND version of the HRS which has imputed values for income and wealth as well as other variables.

⁹ CAMS is administered biennially to a random subsample of approximately one-half of all households participating in the HRS. Expenditure categories include large purchases such as appliances, regularly purchased items like food, household utilities and gasoline, health care costs, and leisure activities like dining out, travel, and hobbies. See <https://hrs.isr.umich.edu/documentation/data-descriptions> for descriptions of the CAMS on the HRS core surveys.

¹⁰ Re-weighted results are similar if race/ethnicity categories are excluded as additional independent variables in the logistic regressions to estimate the propensity scores.

¹¹ We do not apply the HRS official sampling weights as they are either specific to a survey-year or a short sequence of years, therefore not applicable in our setting.

Table 1 shows summary statistics for cases in the wave immediately before dementia onset ($t = -2$) and reweighted controls for the same event time. By construction, cases and controls look observably similar in terms of age, fraction female, education, race/ethnicity, and marital status, factors important for both dementia prevalence and financial outcomes (Panel A). Even after reweighting, there is a higher fraction of cases with certain comorbidities including heart conditions, stroke, diabetes, and psychiatric conditions. Compared to controls, cases also have more functional limitations in terms of activities of daily living (ADL) and instrumental ADL (Panel B). Finally, cases appear to have lower wealth by all measures shown.

3.2. Event study

To ensure the robustness of our comparisons, we further employ an event study approach to use only within-individual variation overtime to examine differential changes in wealth, income and spending measures between cases and controls. To account for the highly skewed nature of our dependent variables that include a large number of zeros, we estimate the following Poisson event study specification (Mullahy & Norton, 2024):

$$E(Y_{it}) = \exp(\alpha + \sum 1[R_{it} = r]\beta_r + X_{it}'\gamma + \rho_t + \eta_i) \quad (3)$$

where Y_{it} is a measure of financial outcome of interest for individual i in year t ; $1[R_{it} = r]$ is an indicator function if a given observation for the individual corresponds to a specific year relative to dementia¹². For controls, all the indicator functions evaluate to 0. X_{it} is a vector of time-varying individual specific characteristics, including marital status and comorbidities. ρ_t and η_i represent calendar and individual fixed effects. The models are estimated using pseudo-likelihood regression and the standard errors are clustered at the household level. To ensure that estimates are not sensitive to the Poisson model assumptions, we alternatively apply inverse hyperbolic sine transformations to our dependent variables and use a linear event study models as sensitivity analyses.

¹² Note that as shown below, instead of choosing the wave before dementia ($t = -2$) as the omitted year, we set $t = -6$ as the omitted year to show trajectories in wealth during the years leading up to dementia, as wealth among cases and controls begin to diverge about three waves before dementia onset based on the descriptive trends.

3.3. Panel vector autoregression

We further employ a panel vector autoregression (VAR) approach developed in analyzing time series data to investigate the temporal relationship between cognitive function and wealth, informing the likelihood of reverse causality. VAR is a statistical model that captures the reduced-form association between multiple variables in a dynamic framework. Formally for the panel VAR, let y denote a $K \times 1$ vector of endogenous variables of interest for individual i in time period t , then

$$y_{it} = A_1 y_{it-1} + A_2 y_{it-2} + \cdots + A_p y_{it-p} + u_{it} + e_{it} \quad (4)$$

where A_l ($l=1,2,\dots,p$) is a time-invariant $K \times K$ matrix of coefficients with p lags, u_{it} is a $K \times 1$ vector of dependent variable-specific panel fixed effects, and e_t is a $K \times 1$ vector of error terms that satisfy: (1) mean zero, (2) the contemporaneous covariance matrix of error terms is positive-semidefinite, and (3) no correlation in error across time.

In our application, y has two elements (hence $K=2$): the continuous cognitive function score and the continuous measure of household wealth. For the cognitive function variable, we use a score that equals the probability of being cognitively normal multiplied by -100 such that the coefficients may be interpreted as the response to a *one-percentage point decrease* in the probability of being cognitively normal (an increase in the probability of having *any* cognitive impairment including dementia and MCI). For the wealth variable, we use total household wealth in each period expressed as percentage of wealth at baseline (the first observation for each individual in our study). A one-unit change is thus equivalent to a one percent change in wealth from baseline. This measure is more sensitive to the relative decline in wealth over time than is a measure of the absolute change, a relationship that we would expect if the primary impact of dementia is on the rate of return to assets.

We use a three-lag model (or 6 years) to account for the long-run effects of cognitive function on wealth and vice versa, constrained by the limited number of lagged values available in the data. Note that the panel VAR coefficients do not have a causal interpretation without additional assumptions. Rather, they capture the extent to which values of an endogenous variable in the earlier periods are predictive of the same or another variable in later periods.

3.4. Heterogeneity by timing of self-reported diagnosis of memory conditions

As a sensitivity analysis, we examine heterogeneity in trajectories in household wealth by the timing of self-reported diagnosis of memory conditions. The HRS asks each respondent (or their proxy) in each wave whether they were told by their doctor that they had any memory-related conditions. We stratify cases by whether there is a self-reported diagnosis of any memory related condition prior to our measure of dementia onset: those with such a diagnosis at $t=0$ or earlier are in the “timely diagnosis” subgroup, and those with a diagnosis at $t=2$ or later, or who never report such a diagnosis, are in the “late or no diagnosis” group. Timely diagnosis may mitigate wealth decline if it prompts patients and families to take action to mitigate any financial impact of cognitive decline, for instance by transferring financial management responsibilities or changing asset composition (e.g. shifting to less risky assets). Alternatively, it could hasten wealth decline if utility is state dependent and cognitively impaired individuals prefer to consume assets while able to enjoy activities such as travel, or if individuals endeavor to “spend down” assets to secure Medicaid coverage of long-term care. Additionally, early diagnosis may also reflect unmeasured cognitive or behavioral deficits not captured in our primary cognition measure and thus be associated with a more rapid wealth decline, particularly if wealth declines causally affect cognition.

3.5. Placebo conditions as falsification test

Finally, to investigate whether any patterns we find are specific to dementia and cognitive impairment instead of serious illnesses in general, we construct alternative samples with (self-reported) cancer, heart disease, lung disease and arthritis respectively as the condition of interest rather than dementia. We apply similar inclusion and exclusion criteria to the case group and control group as we do in the main analysis sample, and we define the year of condition onset as the first year in which the HRS respondent reported having each of these four conditions. We conduct analogous event study analysis examining any differential trajectories in household finances between those who eventually developed one of the four placebo conditions and the condition-free control group. These results are reported in section 5.2.

4. Main Results

4.1. Overall wealth

Figure 1 shows the reweighted trends in mean total household wealth (and its 95% confidence interval) and its two components: financial and non-financial wealth. Financial wealth includes liquid assets: stocks, bonds, checking and savings accounts, CDs, other savings, individual retirement accounts (IRAs) and Keogh accounts (retirement accounts for self-employed and small businesses), net any financial debt. Non-financial wealth includes the net value of real estate, vehicles and businesses.

There is a statistically insignificant gap in mean household net worth (about \$30,000) (Panel A) and net financial wealth (around \$40,000) (Panel B) between cases and controls which trend roughly in parallel between eight and six years prior to dementia onset, but both widen further thereafter, with a difference of \$124,149 in net worth and \$78,664 in net financial wealth at dementia onset (time $t=0$). Mean net non-financial wealth is very similar between cases and controls up to 4 years before dementia onset (cases are slightly higher than controls by about \$5,000) and starts to diverge thereafter, with cases lagging controls by \$45,483 at dementia onset (Panel C).

Results from the event study analyses (Figure 2 and Table A1), which control for individual fixed effects, are consistent with the trends analysis, although the magnitude of the wealth declines is attenuated because only within-individual variation is used. Notably, the relative decline between cases and controls in financial wealth occurs earlier compared to that for non-financial wealth, accounting entirely for the decline in net worth between six and four years prior to onset (about -\$16,800). The decline in non-financial wealth catches up during the two years before dementia onset, and each component of net worth accounts for roughly half of the decline in net worth during that period (-\$26,164 for financial wealth and -\$33,033 for non-financial wealth compared to -\$51,166 for net worth).¹³ Both declines are further accelerated (especially non-financial wealth) after dementia onset.

If we break down the components of financial wealth (Figure 3), the largest decline in financial wealth occurs for stocks, bonds, mutual funds, and investment accounts (Figure 3A),

¹³ The decline in net value of real estate accounts entirely for the decline in non-financial wealth, a point we return to below.

with a more modest impact for checking and savings accounts (Figure 3B). The reweighted trends reflect the same pattern (Figure A1). We return to this point below.

4.2. Income and Financial Wealth Changes

To explain the patterns described above for overall household wealth, we begin by examining whether reduced earnings can explain the decline. In Figure 4 we show trends in total household income and income by type. The gap in total annual household income widens gradually during the six years prior to dementia onset (though the decline is noisy), reaching nearly \$2,000 by the wave in which the individual exhibits incident dementia. Close to half of the decline may be attributed to reduced earnings, although the absolute decline in earnings (at $t = 0$) in dollar terms is only \$947, or less than 3% of average annual income among cases at that point. The accumulated differential loss in earnings from year $t = -4$ (extrapolated across waves) to $t = 0$ is less than \$1,500, and thus (holding consumption constant) cannot explain more than a tiny fraction of the overall gap in wealth. Notably, about one-half of our sample of cases already has zero earnings (so are retired or out of the labor force) six years prior to dementia onset.

A few additional patterns are noteworthy. Pension/annuity income exhibits a consistent decline for both the case and control groups (Panel C in Appendix Figure A2), with a somewhat more rapid decline for the case group; this general decline is likely the consequence of pensions that are not indexed to inflation or the loss of benefits on the death of a spouse.¹⁴ Social Security retirement income declines mostly in parallel between cases and controls (Panel D in Figure A2).¹⁵ The relative decline among cases compared to controls is small for both income types and unlikely to account for the substantial decline in household net worth (Panel C and D in Figure 4). Finally, other income, which are mostly safety net benefit payments including unemployment/workers compensation, Social Security disability/supplemental security income

¹⁴ The default option for private defined benefit pension plans is to provide survivorship benefits to a spouse, but these survivor benefits are typically reduced relative to those for the married couple and may be opted out of altogether in exchange for higher annual payout as single life (i.e. no survivor benefit is elected). An earlier Urban Institute report finds 28 percent of married men and 69 percent of married women opting for single life pensions (Johnson et al., 2003). A more recent study using data on individuals with a pension administered by the PBGB found 58 percent of men selecting joint and survivorship benefits and just 38 percent of women with this option (Clark et al. 2023). Consistent with this evidence, we find that the case cohort experience a larger increase in the death of a spouse than relative to controls, by approximately 2-3 percentage points per year in the years leading up to dementia onset, a small but meaningful difference.

¹⁵ Death of a spouse can also affect household social security retirement income, however, the surviving spouse can typically claim the higher between own retirement benefit and survivor benefit, thereby mitigating the impact.

and other government transfer income, increase marginally after dementia onset, consistent with individuals or households claim more benefits after dementia onset. We discuss capital income below.

We next consider the more complicated question of how dementia affects the return to financial investment. We cannot observe individual investment returns directly but instead must infer changes in value of stocks and other investment accounts as well as capital income conditional on total income and consumption in the case/control propensity score analysis. Both Appendix Figure A1 and Figure 2 point to value of stocks, bonds, mutual funds and investment accounts as displaying the largest relative decline between cases and controls, at -\$9,090 at $t = -4$ (imprecisely estimated), -\$15,775 at $t = -2$ and -\$20,147 at $t = 0$ according to Figure 2 Panel A, while stabilizing somewhat after. This decline is all the more remarkable given that the mean value of this asset category is virtually *identical* between cases and controls at $t = -6$, at about \$92,000 (Panel A of Figure A1). This is the category where poor financial decisions, including susceptibility to fraud or even lack of attention, can have an immediate impact on asset value. Checking and savings accounts also exhibit smaller though non-trivial decline from -\$4,532 to -\$8,417 annually during this period. By contrast, individual retirement account (IRA) and Keogh account, which are more restrictive, show no relative change.

Capital income shows a general downward trend among cases relative to controls in the years leading up to dementia onset (Figure A2 Panel E), though the estimates are noisy (Figure 4 Panel E). The drop in capital income is large and significant at $t = 0$, however, at about \$1,600 (Figure 4 Panel E), or a 20% relative decline of the average capital income at $t = -6$ among cases, compared to roughly 10% relative decline in wealth during the same period (-\$51,116 from a mean of \$525,021), a finding that can be consistent with poor financial decisions.¹⁶

4.4. Household spending and nursing home status

It is still possible that individuals with cognitive decline and their families intentionally decumulate assets in order to qualify for Medicaid coverage of nursing homes, if they are conscious of the risk of needing long-term care. Alternatively, out-of-pocket medical expenses may be higher (e.g., hiring health aides) or people may just choose to spend more while they can

¹⁶ Capital income only reflects a small part of return on assets as it includes primarily dividend and interest income as well as income from business or farm, self-employment earnings, gross rent and trust funds or royalties.

still enjoy it (Brown et al., 2016). To examine these hypothesized mechanisms, we show household spending patterns (excluding transfer to family members) in Figure 5 and Figure A3, Panel A and B. We find no evidence that the “case” households overall spend more than controls during the 6 years leading up to dementia onset (Panel A in Figure A3 and Figure 5).¹⁷ This finding does not support the view that people increase spending before dementia onset because of state-dependent utility, perhaps by traveling or otherwise enjoying consumption while they are still cognitively able (Brown et al., 2016). Note that while we cannot rule out small increases in spending due to the large confidence intervals, they are unlikely to explain the large wealth decline prior to dementia onset even at the upper bound of the 95% CIs.

Nor is there evidence that older adults recognize cognitive changes and transfer assets to their children, either as a way of spending down to Medicaid or to be able to enjoy seeing the children use these resources during their lifetimes. As shown in Figure A5 in the appendix; transfers to children are actually marginally less likely among the ever dementia group. By contrast, we see that transfers made *to* those in the dementia group rise sharply, suggesting that they are in need of financial assistance.

Additionally, while out-of-pocket healthcare expenditures do increase prior to incident dementia relative to controls, the magnitude of the change is small relative to the relative decline in wealth, ranging between \$1,036 and 1,567 per year before dementia onset. The decline was higher at \$3,958 in the wave of dementia onset, when much of the financial wealth decline had already occurred. Not surprisingly, out-of-pocket healthcare spending continues to increase once dementia is pronounced, likely due to care needs as evidenced by the higher probability of nursing home residence. Taken together, the moderate increase in healthcare spending and insignificant change in total spending suggests that households with dementia patients may have actually *decreased* consumption outside of healthcare relative to controls.

Another important test for spend-down is whether households anticipate the increased likelihood of nursing home residence. As shown in Panel C of Figure 5, the self-reported probability of moving to a nursing home in the next five years shows little difference between the two groups (Panel C), although the proportion of living in a nursing home jumps up sharply (from 5% to over 20% in Figure A3 Panel D) among cases at dementia onset, or an estimated

¹⁷ Further, there is no evidence of spending down even among the subgroup in the bottom tertile of initial wealth who are closest to the Medicaid eligibility threshold.

relative increase of 17 percentage points (Figure 5 Panel D), with no discernible evolution in controls.¹⁸ Note that this increase in nursing home residence is highly consistent with decreases in ownership of primary residence (Figure A5), explaining most of the decline in non-financial wealth. These results confirm again that intentional spend-down is unlikely to explain the divergence in wealth between cases and controls.

4.5. Panel VAR results

Our results so far may still be consistent with reverse causality, i.e. shocks in wealth, such as fluctuations in the stock market, may lead to accelerated cognitive decline and dementia onset. To expand testing for reverse causality, we next consider the panel VAR analyses in two formats commonly shown for this approach. The first is a Granger-Causality table with results from Wald-tests for joint-significance of all lagged values of a given variable (e.g. cognitive function score) in predicting the other variable (e.g. normalized wealth). Table 2 shows that cognitive function score from the previous three periods significantly predicts normalized wealth in the current period ($p<0.001$), whereas normalized wealth in the previous three periods do not significantly predict cognitive function score ($p=0.805$). The full coefficients estimated for the two models are shown in Appendix Table A3.

In addition, Figure 8 shows graphs for cumulative impulse response functions (IRF) interpreted as the cumulative change (or “response”) in the dependent variable in subsequent periods to a one-unit increase (or “impulse”) in the independent variable in the initial period, for all four possible combinations of independent and dependent variables as predicted by the panel VAR equations. The top row shows IRFs for normalized wealth as the independent variable, and the bottom row shows IRFs for cognitive function score as the independent variable. While it is not surprising that a one-unit increase in each of these two variables yields a significant response in its own values one, two, and three periods later (top left and bottom right panels), the bottom left panel shows the IRF for (lagged) cognitive function score as the independent variable and normalized wealth as the dependent variable.

Based on the IRF point estimates, an increase in the cognitive function score (equivalent to a decrease in the probability of being cognitively normal) by one percentage point in Period 0

¹⁸ This lack of recognition of the coming need for long-term care is noteworthy and consistent with the failure of many to plan for long-term care needs.

is expected to evoke a cumulative (and statistically significant) decrease in normalized wealth by 0.68 percentage point three periods later and 1.26 percentage points four periods later (equivalent to 6 and 8 years in our data respectively). To put the results in context, the average case individual in our sample experiences a decrease in the probability of being cognitively normal by 28 percentage points between 8 and 6 years prior to dementia onset, which would translate to a wealth decline of 19 percent by the time of dementia onset compared to the baseline wealth level, and 35 percent by two years after dementia onset. These are roughly on the same order of magnitude with the observed decline in mean net worth among cases, by 29% at dementia onset (\$372,668) and 33% two years after onset (\$353,210) compared to six years before dementia (\$525,021).

By contrast, a one percent increase in normalized wealth relative to the baseline is not predicted to significantly change the cognitive function score in subsequent periods, as indicated by the 95% CI overlapping with zero in the top right panel. These results provide additional evidence that the causal pathway is from cognitive decline to a wealth decline.¹⁹

5. Sensitivity Analyses Results

5.1 *Heterogeneity in timing of diagnosis of memory conditions*

One way to check the robustness of impaired financial decision-making as an explanation for wealth loss is to compare the wealth trajectory of people who received a timely diagnosis – right when their cognition scores indicated likely dementia or earlier and thus became aware of their cognitive impairment – and those diagnosed later. Figure 6 provides this comparison for the group of “timely” diagnosis – those with a self-reported diagnosis with dementia at or prior to incident dementia – and the “late or no diagnosis” group without a timely diagnosis; 29% of the case sample is in the timely diagnosis group. While this group still experiences wealth loss by the time of dementia onset (Panel A), there is evidence of reversal of this loss in the years after dementia (i.e. after diagnosis), particularly for financial wealth with the gap closing almost completely by four years post dementia. By contrast, the decline in wealth among those without a timely diagnosis (Panel B) continues further on after dementia onset.

¹⁹ One eigenvalue in the VAR lies just barely outside the unit circle (1.02); this makes sense as people rarely if ever recover from dementia prior to death.

One possibility is that the subgroup of cases with timely diagnosis are those with higher socioeconomic status who have better access to medical care and treatment and are better at financial decision-making even with cognitive decline (or have financial planners to manage their net worth) compared to their peers who also develop dementia. However, Table A2 suggests that this is not the case: those with timely diagnosis are broadly similar to those without timely diagnosis in terms of education, race/ethnic composition and comorbidities. The former group are somewhat more likely to be married or partnered than the latter group, indicating the potential importance of the support of family members in facilitating a timely diagnosis.²⁰

5.2 Results from placebo conditions

Finally, we examine whether the patterns observed so far apply to populations with serious medical conditions other than dementia, including cancer, heart, lung and arthritis. These conditions all differ from dementia in that they do not lead to decline of cognitive functioning unless the patient is approaching end-of-life (though they often lead to declines in physical functioning). If we observe similar patterns of decline in household wealth among the cancer group compared to the controls, it would suggest that factors other than cognitive impairment influence these trends, such as reduced earnings or increased consumption. Appendix Figure A6 show analogous event study results on wealth comparing individuals who reported having each of these conditions and those who did not. For most of the measures examined, we see very little change throughout the period spanning between six years prior to reported condition onset and four years after. If anything, several wealth measures actually experience a relative *increase* among the case group compared to the control group. One possible reason for this is selection in reporting—those who report having these conditions and remain in HRS are likely those who have adequate healthcare access to be able to detect them.²¹

²⁰ In additional analysis, we find no evidence that being married or partnered by itself helps mitigate wealth loss, supporting that it is diagnosis rather than family composition per se that matters.

²¹ Note that while this could also apply to those with timely dementia diagnosis, we still observe relative decline in wealth prior to dementia onset for that group, suggesting that factors unique to dementia are largely responsible for wealth decline prior to symptoms onset.

6. Discussion

Using nearly two decades of longitudinal HRS data, we examine several candidate explanations for the widening divergence in household wealth between individuals who eventually develop dementia and those who do not. We find little evidence that reduced earnings, higher out-of-pocket medical spending, intentional asset “spend-down,” state-dependent utility, or reverse causation from wealth shocks account for these patterns. Instead, the results point to declining cognitive function impairing the quality of financial decision-making, particularly in the management of liquid and complex financial assets.

Two additional findings reinforce the interpretation that these effects are driven by cognitive decline rather than correlated shocks. First, individuals with other serious medical conditions—such as cancer or heart disease—do not exhibit similar pre-onset wealth declines. Second, respondents who report a timely memory-related diagnosis experience some attenuation of wealth losses after onset, particularly in financial assets, consistent with households taking corrective actions once cognitive impairment becomes salient. However, because financial mismanagement often precedes noticeable memory symptoms by several years, timely diagnosis may reduce—but not fully prevent—wealth loss.

This study highlights an important but under-recognized dimension of the economic burden of dementia: the deterioration of household finances during the pre-dementia period. These early losses can compound the already substantial medical and long-term care costs associated with dementia, affecting not only the individuals experiencing cognitive decline but also their spouses, families, and caregivers.

More broadly, our findings underscore that dementia-related financial risk should be viewed as a universal and forward-looking household risk, not just a concern for those who are already symptomatic or diagnosed. Because financial mistakes often appear before individuals or families become aware of any cognitive decline, relying solely on diagnosis or subjective awareness is unlikely to be sufficient. Safeguards that can be implemented well in advance—such as designating trusted financial contacts, simplifying financial arrangements, or allocating part of retirement wealth to products with guaranteed payouts—may help reduce exposure to losses from impaired financial decision-making.

Addressing this risk requires coordination across multiple sectors, including health care, financial institutions, legal services, and policymakers. As populations age, failing to recognize

and insure against the financial risks posed by early cognitive decline will place a growing burden on affected households. Broader institutional and policy measures that help households protect assets well before symptoms emerge may play a key role in mitigating the long-run financial consequences of dementia.

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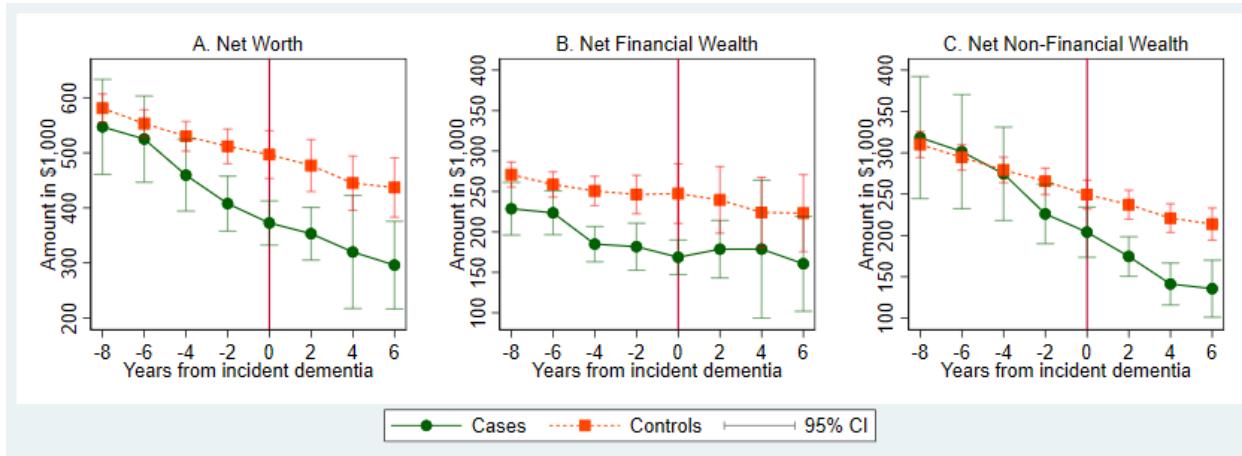
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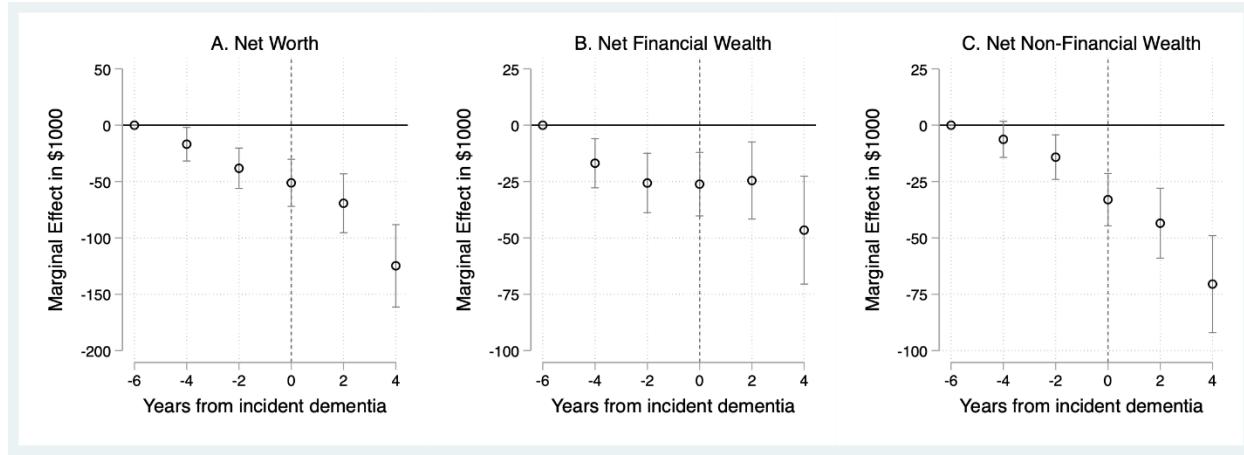
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Figure 1. Trends in mean household wealth among dementia cases and weighted controls



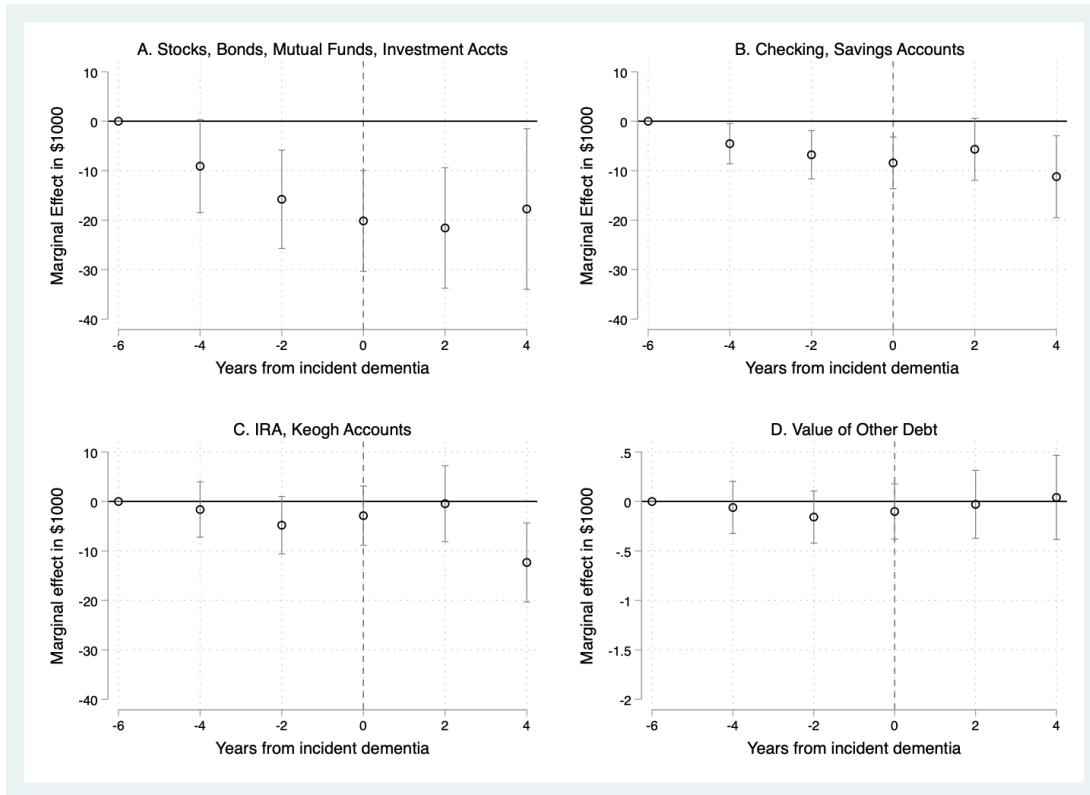
Notes: All wealth variables are in 2018 dollars. Total wealth is the sum of financial wealth and non-financial wealth. Financial wealth includes the net total value of (1) stocks, bonds, mutual funds and investment accounts, (2) checking and savings, (3) individual retirement accounts, and (4) other debts. Non-financial wealth includes the net value of vehicles, real estate (including primary and secondary residences) and businesses. Values for controls were re-weighted to represent cases using propensity score estimated from a logistic function using age, gender, education (no high school degree, high school degree, some college or above), race/ethnicity (Black, Hispanic, White and Other) and marital status (married or partnered, divorced or separated, widowed, never married) as covariates. Separate logistic regressions are estimated for each event time (year relative to incident dementia: -8, -6... +6), and each regression is estimated among case observations at that event time and control observations across all waves. Each regression yields a event time-specific set of weights which are used to re-weight control observations from all years and calculate means for controls in each event time.

Figure 2. Regression adjusted differences in household wealth among cases with dementia relative to controls



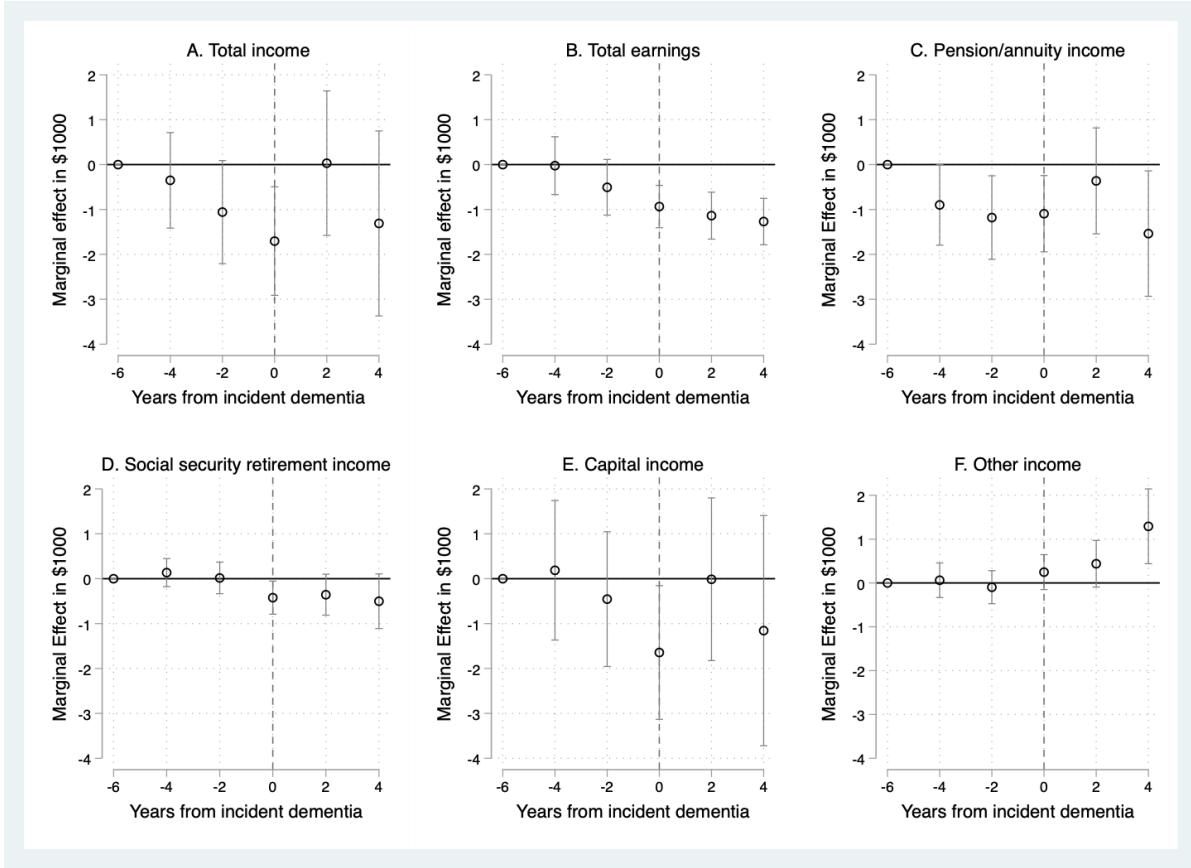
Notes: All financial outcomes have the same definitions as Figure 1. Marginal effects and 95% confidence intervals are shown for each financial outcome representing differential changes among cases in a specific year from incident dementia relative to 6 years prior to incident dementia compared to controls. Results are estimated from Poisson event study regressions including all person-wave observations, with financial outcomes in 2018 dollars left censored at zero and top coded at the 95th percentile as dependent variables. All regressions control for individual fixed effects, calendar year fixed effects and marital status. Standard errors are clustered at the household level.

Figure 3. Regression results for subcategories of net financial wealth



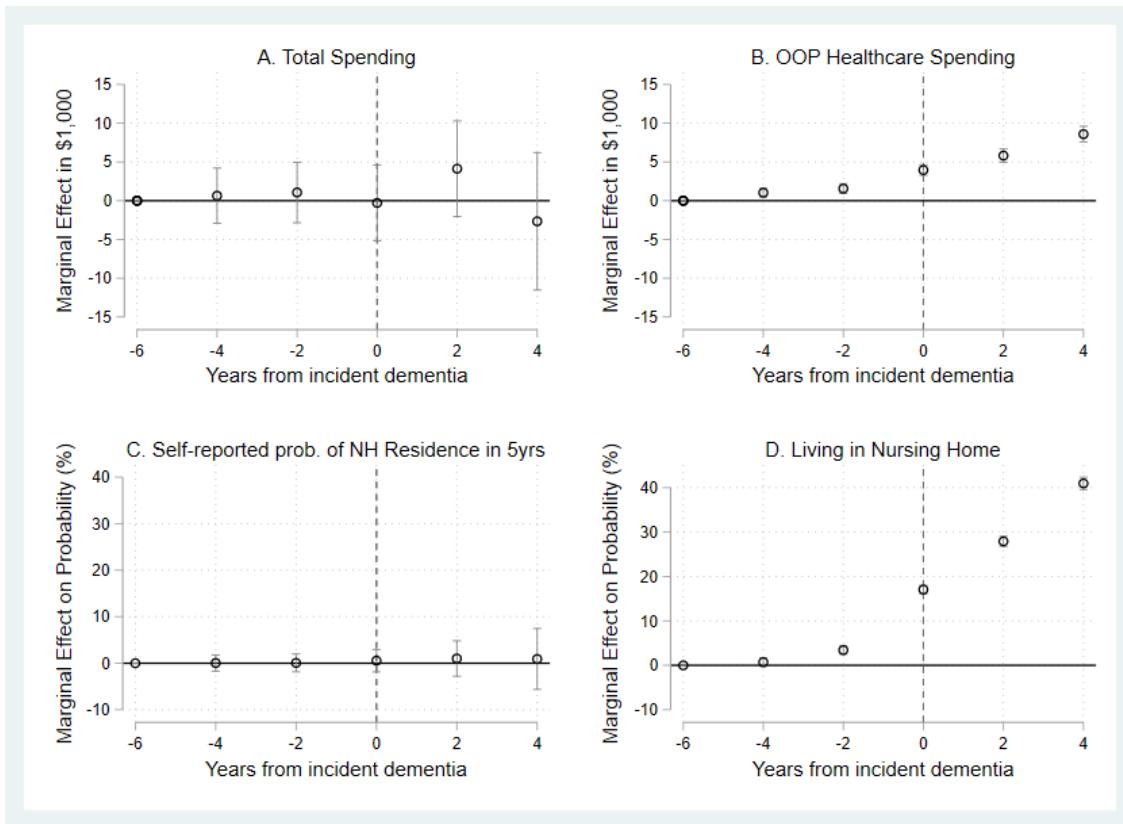
Notes: All financial outcomes are mutually exclusive and collectively exhaustive subcategories of net financial wealth in Figures 1-2. Marginal effects and 95% confidence intervals are shown for each financial outcome representing differential changes among cases in a specific year from incident dementia relative to 6 years prior to incident dementia compared to controls. Results are estimated from Poisson event study regressions including all person-wave observations, with financial outcomes in 2018 dollars left censored at zero and top coded at the 95th percentile as dependent variables. All regressions control for individual fixed effects, calendar year fixed effects and marital status. Standard errors are clustered at the household level.

Figure 4. Regression results for household income, total and by subcategory



Notes: Financial outcomes in Panel (B)-(F) are mutually exclusive and collective exhaustive subcategories of total income in Panel (A). Marginal effects and 95% confidence intervals are shown for each financial outcome representing differential changes among cases in a specific year from incident dementia relative to 6 years prior to incident dementia compared to controls. Results are estimated from Poisson event study regressions including all person-wave observations, with financial outcomes in 2018 dollars left censored at zero and top coded at the 95th percentile as dependent variables. All regressions control for individual fixed effects, calendar year fixed effects and marital status. Standard errors are clustered at the household level.

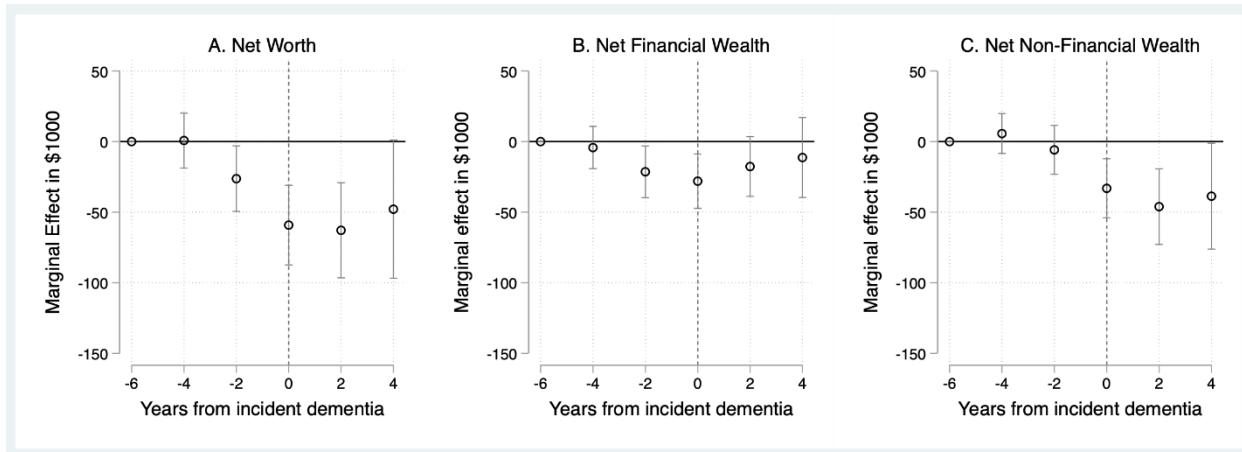
Figure 5. Regression results for household spending and individual nursing home status



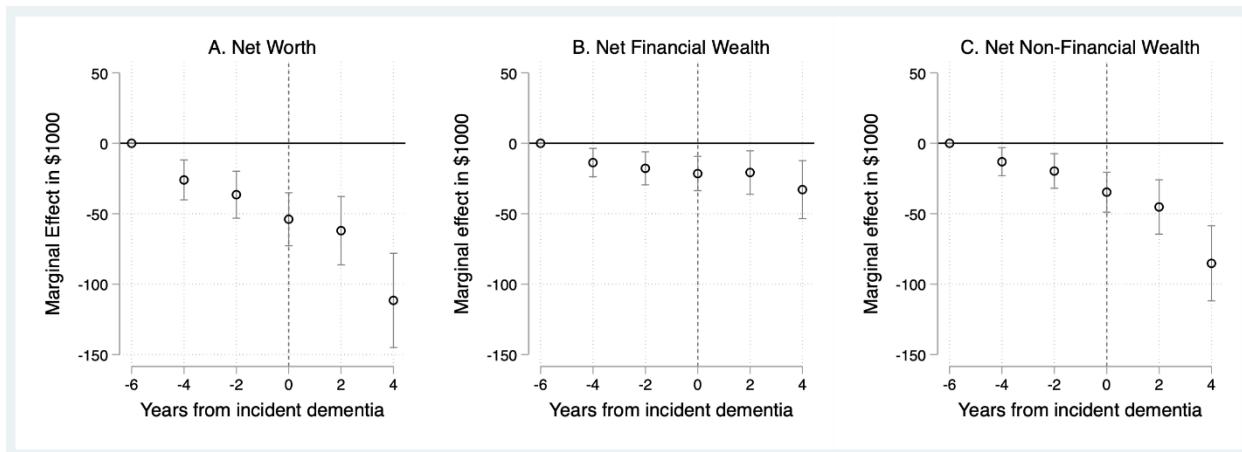
Notes: OOP: out-of-pocket. NH: nursing home. The dependent variable in Panel (A) is total household spending among the subset of HRS respondents in our sample who participated in the Consumption and Activities Mail Survey. The dependent variables in Panel (B) is household out-of-pocket healthcare spending among the larger sample of all HRS respondents in our analytic sample. The dependent variable in Panel (C) is self-predicted probability of residing in a NH in five years from the interview date for all HRS respondents not already in NH. The dependent variable in Panel (D) is an indicator for actually residing in NH at the time of interview for all respondents. Marginal effects and 95% confidence intervals are shown for each outcome representing differential changes among cases in a specific year from incident dementia relative to 6 years prior to incident dementia compared to controls. For Panels (A) and (B), results are estimated from Poisson event study regressions. For Panels (C) and (D), results are estimated from linear event study regressions. All regressions control for individual fixed effects, calendar year fixed effects and marital status. Standard errors are clustered at the household level.

Figure 6. Regression results for household wealth among cases relative to controls, by timing of memory-related diagnosis

Panel A: Timely memory diagnosis

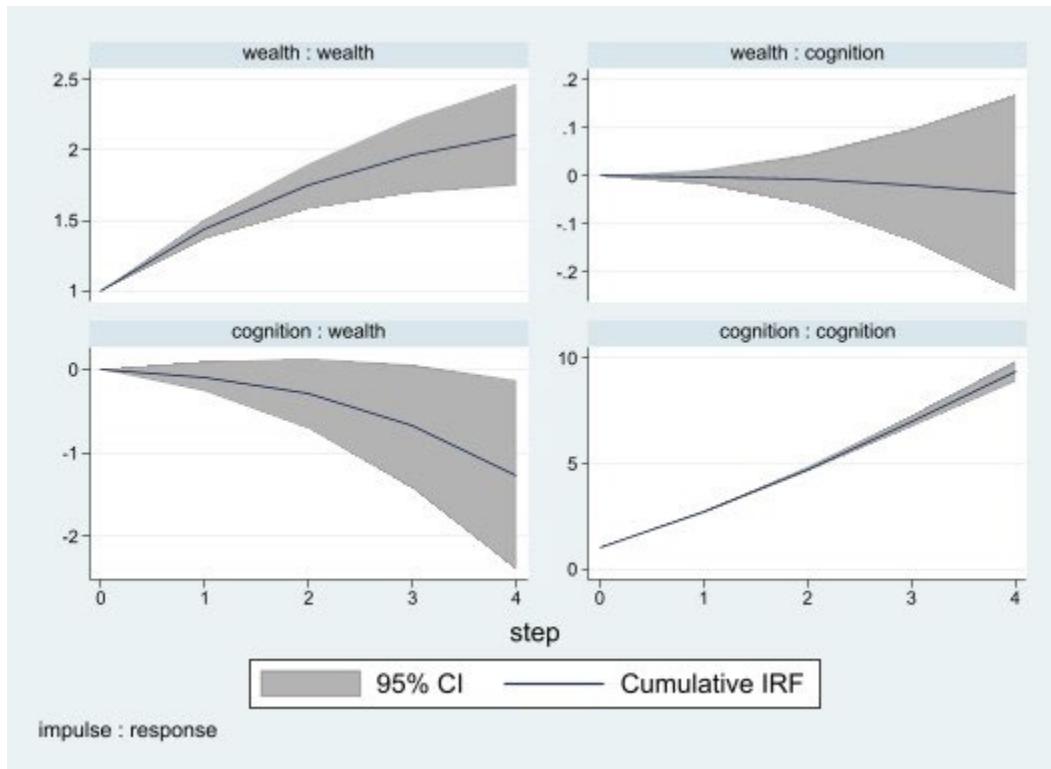


Panel B: Late or no memory diagnosis



Notes: Marginal effects and 95% confidence intervals are shown for Poisson event study regressions identical to those in Figure 2, stratified by timing of memory-related diagnosis. “Timely diagnosis” refers to case individuals with a self-reported diagnosis of memory conditions during or before the wave of incident dementia. “Late or no diagnosis” refers to case individuals without a self-reported memory condition diagnosis up to the wave of dementia onset. The control groups are the same between Panel (A) and Panel (B) and identical to those in Figure 2.

Figure 7. Impulse response functions of cognitive impairment score and relative household wealth from panel vector autoregression predictions



Notes: The graphs show cumulative responses in the dependent variable in subsequent periods to a one-unit increase in the independent variable in the initial period as predicted by the panel VAR with year fixed effects. The first variable (before the colon) in each panel title indicates the independent variable, and the second variable (after the colon) indicates the dependent variable. “cognition” is short for cognitive function score that equals to the probability of being cognitively normal multiplied by -100. “wealth” is short for relative household net worth that equals to the ratio of current household net worth to its value in the first wave in our sample multiplied by 100.

Table 1. Summary statistics of cases and weighted controls, two years prior to dementia onset among cases

	Cases	Controls, weighted
Panel A. Covariates in propensity score estimation		
Age (SD)	81.81 (7.16)	82.00 (7.91)
Female	0.62	0.62
<i>Education:</i>	0.35	0.35
Less than HS education level		
HS degree or GED education level	0.34	0.34
Some college or above education level	0.31	0.31
<i>Marital Status</i>	0.46	0.45
Married or partnered		
Divorced or separated	0.07	0.07
Widowed	0.45	0.46
Never married	0.02	0.02
<i>Race/ethnicity:</i>		
Non-Hispanic Black	0.15	0.15
Non-Hispanic White	0.76	0.76
Non-Hispanic Other	0.02	0.02
Hispanic	0.08	0.08
Panel B. Other characteristics		
Cancer	0.21	0.23
Lung Disease	0.13	0.12
Heart Condition	0.42	0.38
Stroke / TIA - core	0.17	0.10
Hypertension	0.68	0.68
Diabetes	0.26	0.22
Psychiatric Condition	0.20	0.12
Arthritis	0.73	0.72
Index of ADLs	0.94	0.50
Index of IADLs	0.95	0.35
Prob. of being cognitively normal	0.01	0.70
Prob. of having dementia	0.23	0.02
Panel C. Dependent variables		
Total household wealth (\$)	407,813	529,054
Non-financial wealth (\$)	189,964	223,513
Financial wealth (\$)	147,766	204,912
Net value of stocks, bonds, mutual funds and other investment accounts (\$)	89,434	112,627
Net value of checking, savings and money market accounts (\$)	59,998	85,984
Net value of IRA, Keogh accounts (\$)	33,997	49,407
Net value of other debt (\$)	1,666	1,743
Total household income (\$)	43,506	47,711
Total household earnings (\$)	2,132	2,703
Total household pension/annuity income (\$)	7,886	10,796
Total household social security retirement income (\$)	17,880	18,928
Total household capital income (\$)	11,788	12,118
Total household other income (\$)	3,820	3,165
Total household spending (\$)	36,685	38,906
Total out-of-pocket healthcare spending (\$)	7,885	6,217
Self-reported probability of living in nursing home in the next five years (%)	18.9	18.4
Proportion living in a nursing home (%)	4.5	2.2
N	2,312	2,335

Notes: Means are reported, except noted otherwise. Column 2 shows values for controls weighted to represent cases two years before dementia onset. All financial variables are in 2018 dollars.

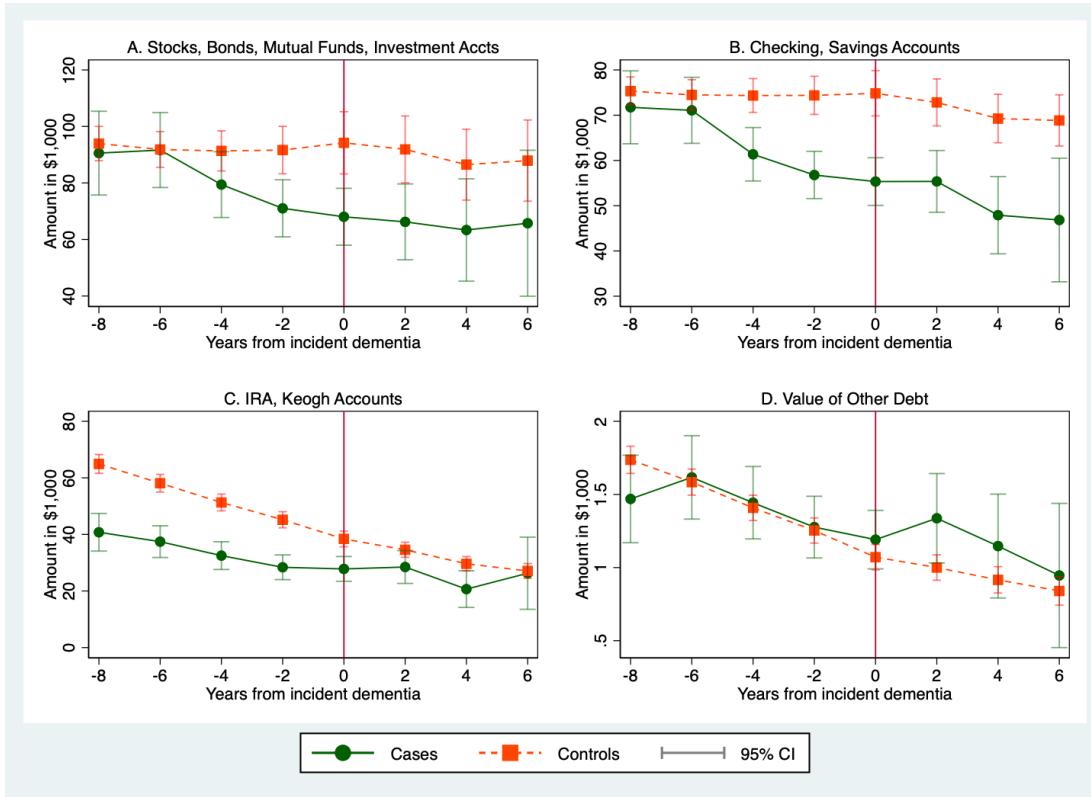
Table 2. Granger-causality Wald test results from panel VAR

Dependent variable	Excluded lagged independent variable	Chi-squared	Degrees of freedom	<i>p</i> -value
Cognitive function score	Relative wealth	5.983	3	0.805
Relative wealth	Cognitive score	23.675	3	<0.001

Notes: Results from Granger-causality Wald-tests are shown which test the joint significance of the excluded lagged independent variable in predicting the dependent variable of the equation. Each row shows results for a separate test for a separate equation. The cognitive function score equals the probability of being cognitively normal multiplied by -100. Normalized household wealth equals the ratio of total household wealth to its value in the first wave in our sample multiplied by 100.

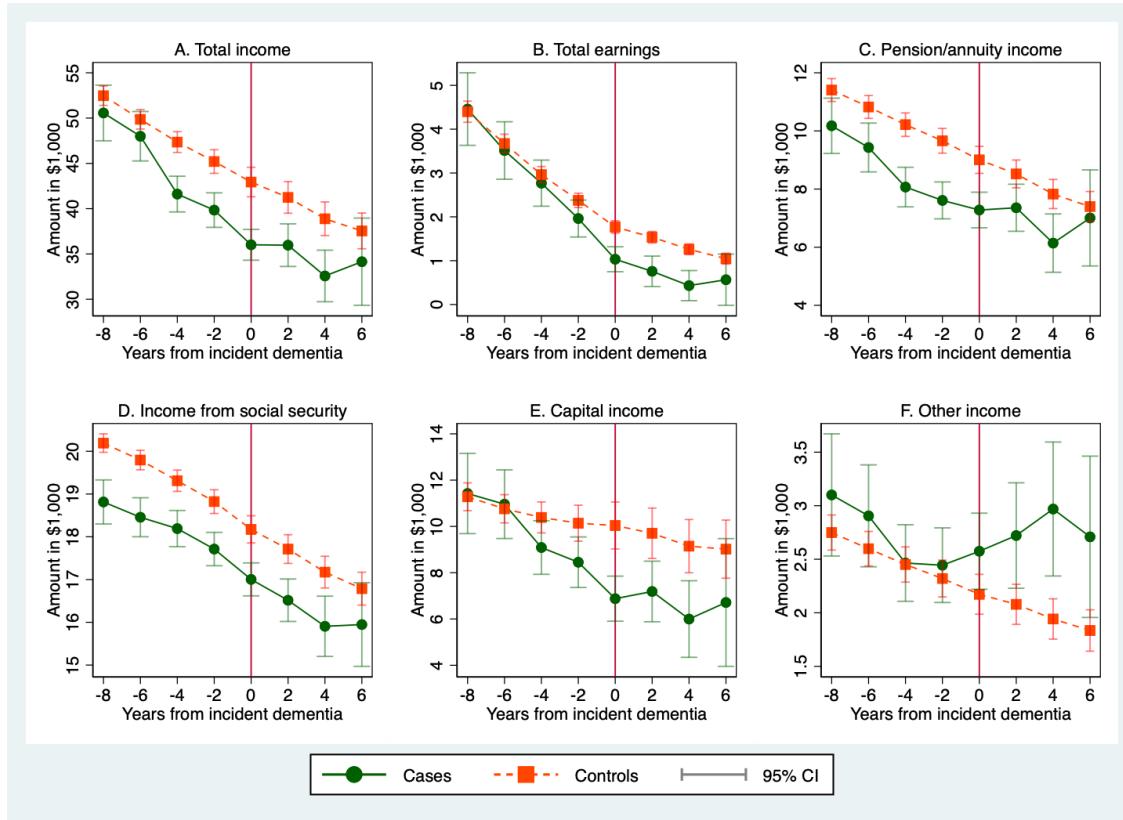
Appendix

Figure A1. Trends in means of subcategories of net financial wealth among dementia cases and weighted controls



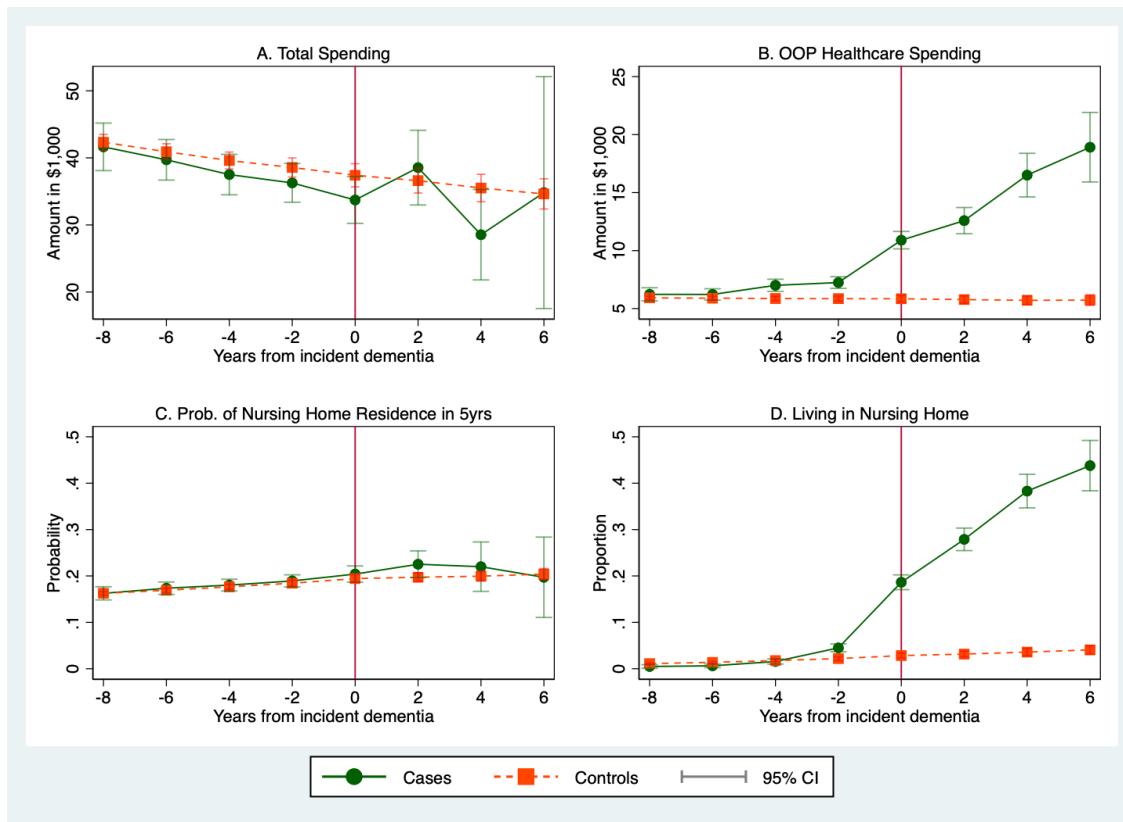
Notes: All financial variables have the same definitions as in Figure 3. Values for controls were re-weighted to represent cases using propensity score estimated from a logistic function using age, gender, education (no high school degree, high school degree, some college or above) and marital status (married or partnered, divorced or separated, widowed, never married) as covariates. Separate logistic regressions are estimated for each event time (year relative to incident dementia: -8, -6... +6), and each regression is estimated among case observations at that event time and control observations across all waves. Each regression yields an event time-specific set of weights which are used to reweight control observations from all years and calculate means for controls in each event time.

Figure A2. Trends in means of total and subcategories of household income among dementia cases and weighted controls



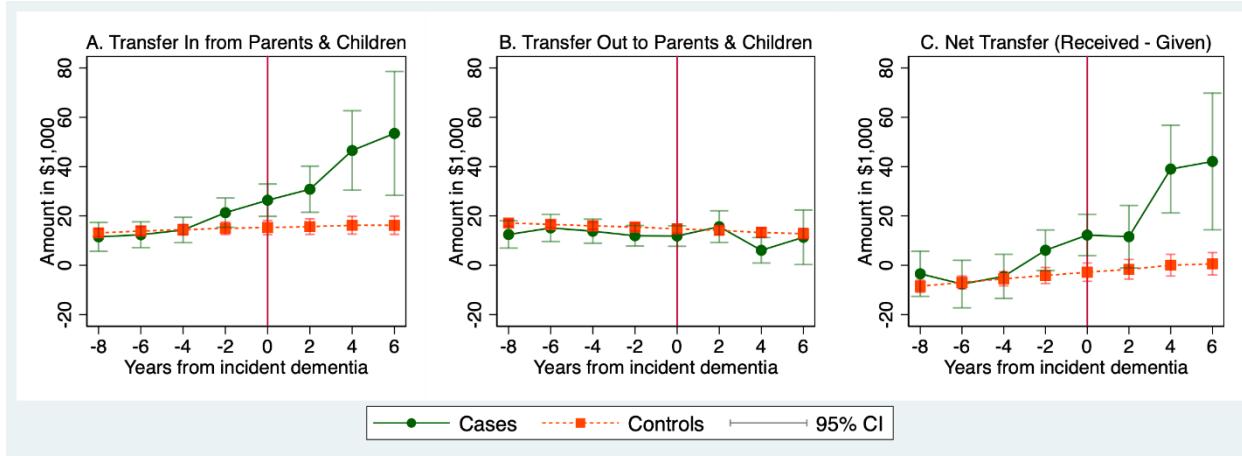
Notes: All financial variables have the same definitions as in Figure 4. Values for controls were re-weighted to represent cases using propensity score estimated from a logistic function using age, gender, education (no high school degree, high school degree, some college or above) and marital status (married or partnered, divorced or separated, widowed, never married) as covariates. Separate logistic regressions are estimated for each event time (year relative to incident dementia: -8, -6...+6), and each regression is estimated among case observations at that event time and control observations across all waves. Each regression yields an event time-specific set of weights which are used to reweight control observations from all years and calculate means for controls in each event time.

Figure A3. Trends in means of household spending and individual nursing home status among dementia cases and weighted controls



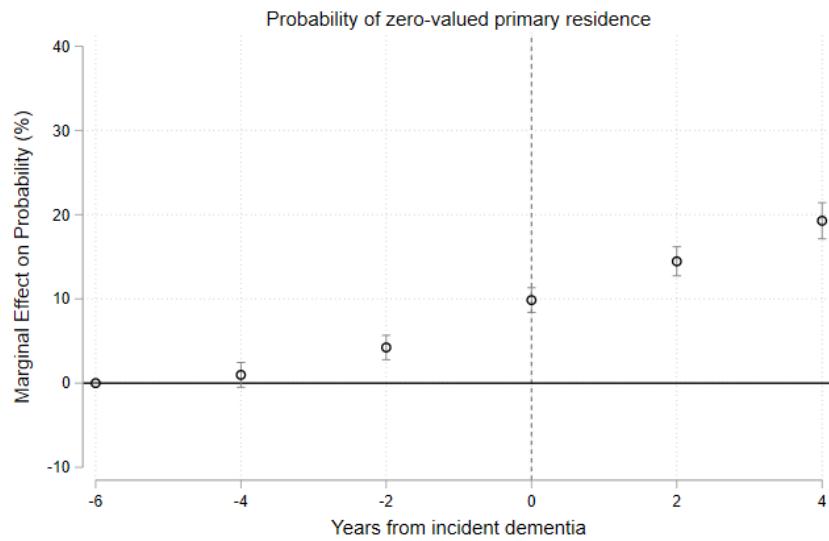
Notes: All variables have the same definitions as in Figure 5. Values for controls were re-weighted to represent cases using propensity score estimated from a logistic function using age, gender, education (no high school degree, high school degree, some college or above) and marital status (married or partnered, divorced or separated, widowed, never married) as covariates. Separate logistic regressions are estimated for each event time (year relative to incident dementia: -8, -6... +6), and each regression is estimated among case observations at that event time and control observations across all waves. Each regression yields an event time-specific set of weights which are used to reweight control observations from all years and calculate means for controls in each event time.

Figure A4. Trends in means of monetary transfers from and to parents and children



Notes: All transfer variables are in 2018 dollars. Values for controls were re-weighted to represent cases using propensity score estimated from a logistic function using age, gender, education (no high school degree, high school degree, some college or above) and marital status (married or partnered, divorced or separated, widowed, never married) as covariates. Separate logistic regressions are estimated for each event time (year relative to incident dementia: -8, -6...+6), and each regression is estimated among case observations at that event time and control observations across all waves. Each regression yields an event time-specific set of weights which are used to reweight control observations from all years and calculate means for controls in each event time.

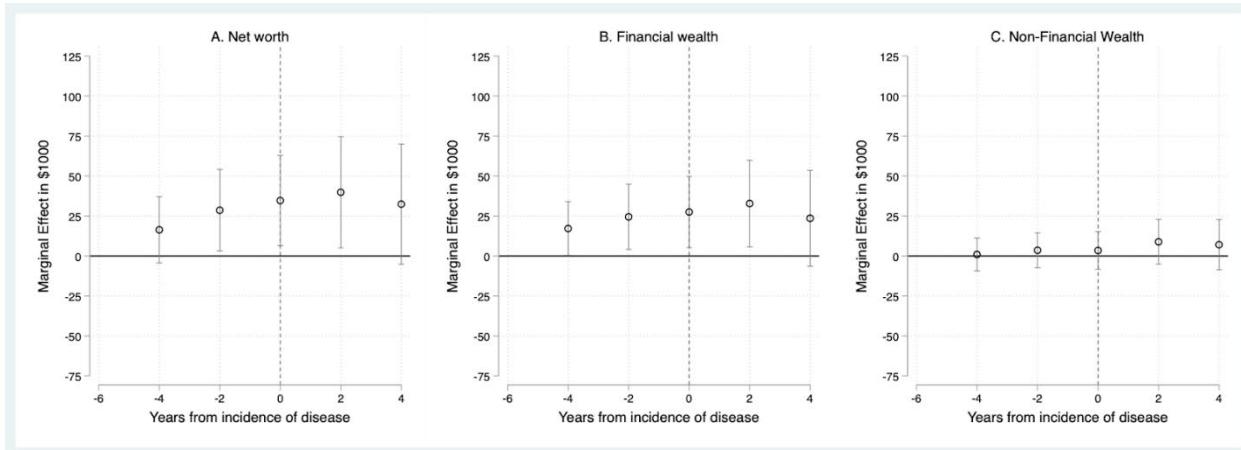
Figure A5. Regression results for probability of zero-valued primary residence



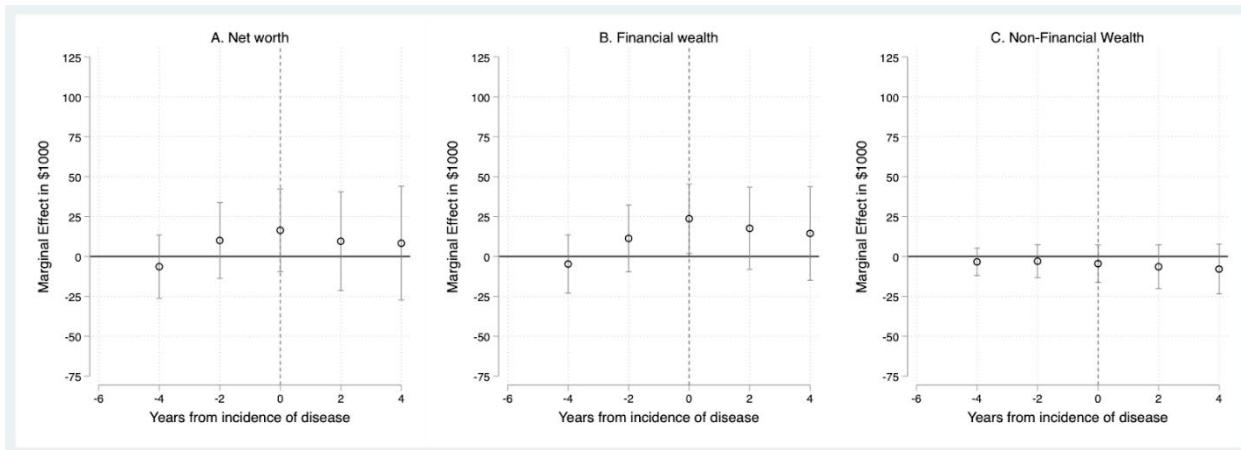
Notes: Marginal effects and 95% confidence intervals are shown for linear probability event study regression with the dependent variable being an indicator for whether the household reported zero value for primary residence in non-financial wealth. The control groups are identical to those in Figure 2.

Figure A6. Regression results for self-reported placebo conditions

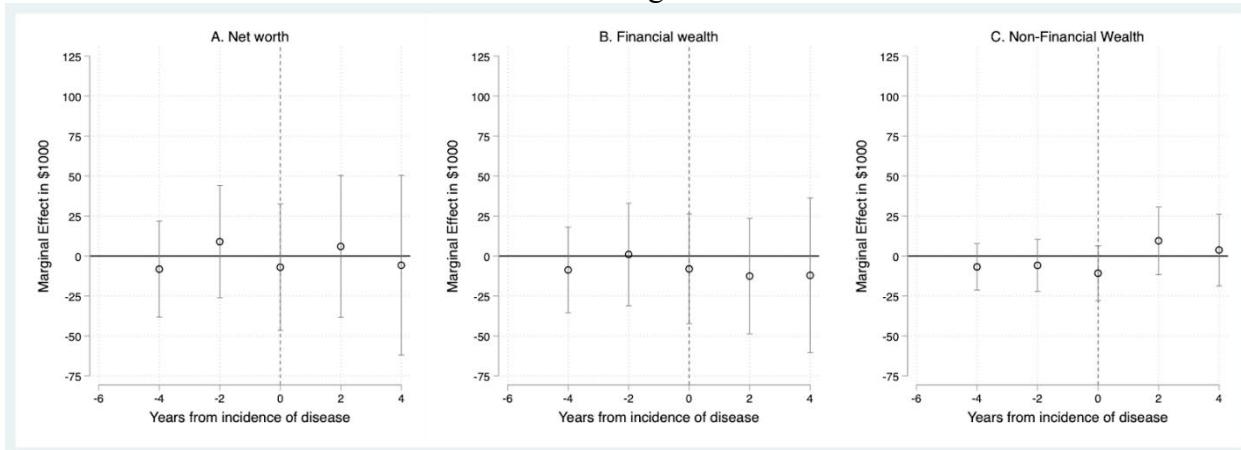
Panel A. Cancer



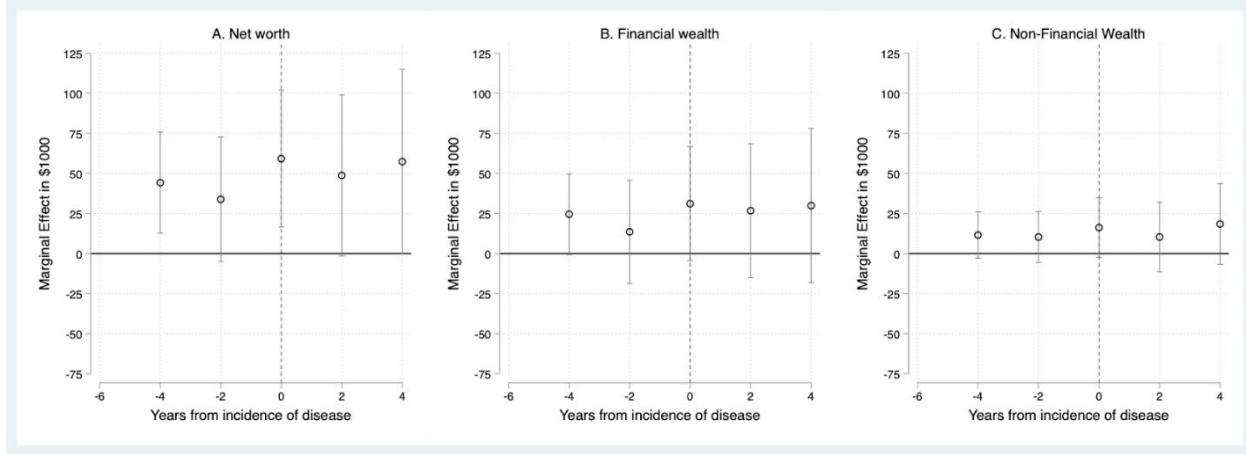
Panel B. Heart diseases



Panel C. Lung diseases



Panel D. Arthritis



Notes: All financial outcomes have the same definitions as Figure 1. Marginal effects and 95% confidence intervals are shown for each financial outcome representing differential changes among cases for each condition in a specific year from incidence of disease diagnosis relative to 6 years prior to diagnosis compared to controls. Results are estimated from Poisson event study regressions including all person-wave observations, with financial outcomes in 2018 dollars left censored at zero and top coded at the 95th percentile as dependent variables. All regressions control for individual fixed effects, calendar year fixed effects and marital status. Standard errors are clustered at the household level.

Table A1. Marginal effect estimates from event study regressions for household wealth

	Net worth (in \$1000s) (1)	Net financial wealth (in \$1000s) (2)	Net non-financial wealth (in \$1000s) (3)
Years from dementia onset			
$t = -6$ (omitted)	-	-	-
$t = -4$	-16.8 [-31.8, -1.9]	-16.9 [-27.8, -5.98]	-6.3 [-14.3, 1.7]
$t = -2$	-38.2 [-56.2, -20.2]	-25.7 [-38.8, -12.5]	-14.2 [-24.1, -4.3]
$t = 0$	-51.2 [-72.1, -30.2]	-26.2 [-40.2, -12.1]	-33.0 [-44.6, -21.4]
$t = 2$	-69.3 [-95.5, -43.1]	-24.6 [-41.7, -7.47]	-43.5 [-59.0, -28.0]
$t = 4$	-124.8 [-161.3, -88.2]	-46.6 [-70.5, -22.6]	-70.5 [-92.0, -49.0]
Observations	57,042	57,042	57,042
Mean Dep Var	441.4	196.2	223.0

Notes: Each column shows results from a separate regression. Marginal effects and 95% confidence intervals (in brackets) shown are identical to those shown in Figure 2.

Table A2. Summary statistics of cases by timing of self-reported memory diagnosis and controls, two years prior to dementia onset among cases

	Cases with late or no memory diagnosis	Cases with timely memory diagnosis	Controls, weighted
Panel A. Covariates used in propensity score estimation			
Age (SD)	82.21 (7.19)	80.86 (6.98)	82.00 (7.91)
Female	0.63	0.59	0.62
<i>Education:</i>			
Less than HS education level	0.36	0.32	0.35
HS degree or GED education level	0.35	0.34	0.34
Some college or above education level	0.30	0.34	0.31
<i>Marital Status</i>			
Married or partnered	0.43	0.53	0.45
Divorced or separated	0.07	0.07	0.07
Widowed	0.48	0.39	0.46
Never married	0.02	0.01	0.02
<i>Race/Ethnicity:</i>			
Non-Hispanic Black	0.15	0.14	0.15
Non-Hispanic White	0.76	0.76	0.76
Non-Hispanic Other	0.02	0.02	0.02
Hispanic	0.08	0.08	0.08
Panel B. Other characteristics			
Cancer	0.21	0.20	0.23
Lung Disease	0.13	0.12	0.12
Heart Condition	0.44	0.40	0.37
Stroke / TIA - core	0.17	0.16	0.10
Hypertension	0.69	0.65	0.68
Diabetes	0.26	0.25	0.22
Psychiatric Condition	0.19	0.20	0.11
Arthritis	0.75	0.69	0.72
Index of ADLs	1.03	0.71	0.51
Index of IADLs	0.96	0.91	0.36
Prob. of being cognitively normal	0.01	0.01	0.70
Prob of dementia	0.23	0.24	0.02
Panel C. Dependent variables			
Total wealth (\$)	385,918	460,361	511,780
Non-financial wealth (\$)	215,731	250,816	265,505
Financial wealth (\$)	170,187	209,544	246,275
Net value of stocks, mutual funds, bonds and other investment accounts (\$)	81,341	108,859	112,627
Net value of checking, savings and money market accounts (\$)	58,549	63,475	85,984
Net value of IRA, Keogh accounts (\$)	31,693	39,523	49,407
Net value of other debt (\$)	1,396	2,312	1,743
Total household income (\$)	42,905	44,945	47,711
Total household earnings (\$)	1,860	2,781	2,703
Total household pension/annuity income (\$)	7,364	9,137	10,796

Total household social security retirement income (\$)	17,351	19,149	18,928
Total household capital income (\$)	12,465	10,160	12,118
Total household other income (\$)	3,864	3,716	3,165
Total household spending (\$)	35,369	40,318	38,906
Total out-of-pocket healthcare spending (\$)	7,814	8,054	6,217
Self-reported prob. of living in nursing home in the next five years (%)	18.7	19.4	18.4
<u>Proportion living in a nursing home (%)</u>	<u>4.5</u>	<u>4.6</u>	<u>2.2</u>
<u>N</u>	<u>1,632</u>	<u>680</u>	<u>2,335</u>

Notes: Means are reported, except noted otherwise. Column 1 shows values for case individuals without a self-reported memory condition diagnosis by the wave of dementia onset. Column 2 shows values for case individuals with a self-reported diagnosis of memory conditions before or during the wave of dementia onset. Column 3 shows values for controls weighted to represent cases two years before dementia onset. All financial variables are in 2018 dollars.

Table A3. Coefficients on lagged values from panel VAR model

	Cognition (1)	Wealth (2)
Cognition one-period lag	1.69 (0.02)	-0.08 (0.09)
Cognition two-period lag	-0.79 (0.03)	-0.02 (0.08)
Cognition three-period lag	0.11 (0.01)	-0.08 (0.05)
Wealth one-period lag	-0.001 (0.007)	0.44 (0.03)
Wealth two-period lag	-0.003 (0.003)	0.11 (0.02)
Wealth three-period lag	-0.002 (0.003)	0.03 (0.02)
Observations	17,858	17,858
Panels	5,998	5,998

Notes: Each column shows coefficients and standard errors in parentheses from an equation in the panel VAR system with two endogenous variables. “Cognition” refers to the cognitive function score that equals the probability of being cognitively normal multiplied by -100. “Wealth” refers to normalized household wealth that equals the ratio of total household wealth to its value in the first wave in sample multiplied by 100. Cross-sectional means are removed from each dependent variable to account for panel-specific fixed effects.