Wealth and the two margins of dependency in old age: onset and duration in Europe and the US*

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Abstract

Using harmonized panel data from SHARE in Europe and the HRS in the United States, we examine two margins through which household wealth shapes dependency in old age, the risk of losing autonomy and survival after its loss. We estimate Cox models for both transitions and instrument baseline wealth with transfers received from outside the close family using a two-stage residual inclusion approach. Wealth substantially delays dependency onset. A ten-percentile increase in the within-country wealth distribution reduces the hazard of losing autonomy by approximately 10% to 17%. Yet wealth does not extend survival after dependency begins, and the conditional association reverses in the United States. We show that this reversal reflects index-event selection rather than an adverse effect of wealth. Estimates for the baseline population provide a clearer picture. Over an eight-year horizon, older adults in the bottom wealth tercile spend 0.22 more years dependent than those in the top tercile in Europe and 0.41 more years in the United States. Despite dying earlier, they spend more than twice as large a share of their remaining life dependent. Poorer older adults therefore face both an earlier loss of autonomy and a greater burden of dependency over their remaining lives.

Keywords: loss of autonomy; long-term care; household wealth; survival analysis; competing risks; aging.

JEL classification: D14, D31, G51, I12, I14, J14.

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1 Introduction

Life expectancy at older ages has risen by more than thirteen years in the OECD since 1960 (OECD, 2024). Those additional years are not distributed equally across social groups and they are not lived in the same condition. Cambois et al. (2011) showed for France that blue workers both die earlier than managers and spend more of their shorter lives with disability. It constitutes a "double penalty" that aggregate longevity statistics conceal. Economists have measured the first half of that penalty with increasing precision and the income gradient in mortality is now documented to the centile (Chetty et al., 2016; Milligan and Schirle, 2021; Jusot, 2006). However, the process by which people lose the ability to look after themselves remains far less well understood.

Most existing evidence either documents functional limitations at a given point in time (Freedman and Martin, 1999; Liu and Wang, 2022; Zaninotto et al., 2020; Connolly et al., 2025) or summarizes mortality and disability in measures such as disability-free life expectancy (Sullivan, 1971; Robine and Ritchie, 1991). While informative about the overall burden of disability, such measures do not identify separately the age at which dependency begins and survival once dependency has occurred. Yet dependency has two distinct margins: first when autonomy is lost and second how long individuals live after its onset. Cross-sectional prevalence cannot disentangle these margins because it reflects both the incidence and duration of dependency, together with mortality. Consequently, two groups may exhibit the same prevalence of dependency even if one experiences earlier onset followed by shorter survival, while the other experiences later onset but longer spells of dependency. These patterns have very different implications for individual welfare and for the demand for long-term care. This distinction is particularly important given that long-term care (LTC) expenditure already represents between 0.5% and 3.7% of GDP across OECD countries and is projected to increase substantially as populations age (Colombo et al., 2011; de la Maisonneuve and Oliveira Martins, 2013). The distributional and policy implications also depend on which margin drives socioeconomic inequalities. A socioeconomic gradient in onset implies that disadvantaged individuals lose autonomy earlier and therefore become eligible for LTC support at younger ages. A gradient in duration implies that socioeconomic groups differ in the number of years spent in dependency once it begins. Distinguishing between these two mechanisms is therefore essential for assessing future care needs and for designing eligibility rules, the mix of cash and in-kind benefits and the financing of public LTC insurance.

This paper estimates both margins jointly. We use harmonised longitudinal data from the Survey of Health, Ageing and Retirement in Europe (SHARE) and the U.S. Health and Retirement Study (HRS)

over 2010–2020 to follow some 34,000 individuals aged 50 and over through two successive transitions. The first runs from autonomy to dependency, defined as difficulty with at least two activities of daily living (ADL) and the second from dependency to death. We pair these two surveys because the two regions sit near opposite ends of the long-term care financing spectrum. In most of continental Europe long-term care is a collective risk while in the United States it is financed privately until assets are exhausted.[1] Socioeconomic position is measured by household net wealth at the first observed wave of survey, expressed in percentiles of the within-country distribution. We use wealth rather than income because it is likely the relevant resource at these ages. It summarises cumulative lifetime means and it is comparable across countries with very different pension systems. It has also been shown that it is what actually finances the lumpy expenditures that dependency generates such as home adaptation, domestic help and residential care (Smith, 1999; Banks et al., 2003; De Nardi et al., 2010).

However, estimating the relationship between wealth and dependency poses a challenge because wealth is potentially endogenous to health. Deteriorating health can deplete assets through medical spending, care purchases, and early labour-market exit (De Nardi et al., 2010; French and Jones, 2011; Poterba et al., 2017). A dependency hazard model estimated using contemporaneous wealth may therefore conflate the effect of wealth on health with the effect of actual or anticipated health decline on wealth. To address this concern, we measure wealth at baseline which is the first year of individual observation and condition on baseline physical and mental health. We further instrument baseline wealth using inheritances and inter vivos gifts received from donors outside the close family. This restriction is motivated by the concern that parental bequests convey information about parental longevity and childhood circumstances, both of which may predict the respondent's subsequent health trajectory. Our identifying assumption is that transfers from more distant donors do not convey comparable information. Finally, because we estimate dependency hazards using a nonlinear semiparametric Cox model, conventional two-stage least squares is not appropriate (Terza et al., 2008; Tchetgen Tchetgen et al., 2015). We instead use a two-stage residual inclusion control-function approach with bootstrap inference to account for uncertainty arising from the generated regressor.

A second issue concerns mortality selection. Death is not independent censoring for the dependency transition. Poorer individuals die earlier which implies that the risk set of "still alive and still autonomous" is progressively purged of its most disadvantaged members. Thus a cause-specific hazard

[1] For example, long-term care is provided through mandatory social insurance in Germany and the Netherlands, a universal allowance in France and tax-financed municipal services in the Nordic countries. Universal health insurance also insulates households from the medical spending shocks that precede dependency (Colombo et al., 2011; Bakx et al., 2020) In the US, Medicare does not cover custodial care and private long-term care insurance covers a small minority. Medicaid requires spending down assets to qualify (Brown and Finkelstein, 2009; Ameriks et al., 2020). Private wealth is thus simultaneously the means of financing care and the barrier to public support.

understates the population gradient. We therefore estimate cause-specific hazards for both exits from autonomy and a hazard for exit from the autonomous state through either channel.

Finally, it appears that analyzing survival conditional on having become dependent conditions on an intermediate outcome that wealth itself causes. Saying differently, dependency is a collider. Even with wealth randomly assigned, the dependent sub-population is not comparable across wealth groups because the wealthy who nonetheless become dependent must be drawn from further into the tail of the unobserved frailty distribution (Hernán et al., 2004; Dahabreh and Bibbins-Domingo, 2024). The direction of the induced bias is predictable and our results make it concrete.

Anticipating our results, we first show that the wealth gradient in the onset of dependency is large. A ten-percentile rise in the within-country wealth distribution lowers the hazard of losing autonomy by about 10%, and by about 17% once wealth is instrumented. Weak-instrument-robust confidence sets exclude a null effect in both surveys. Second, the duration margin behaves completely differently. In Europe, survival once dependent is unrelated to wealth but in the United States, wealthier dependent individuals face a higher mortality hazard. Although surprising, we do not claim against American long-term care but we argue it is instead an artefact of conditioning on dependency. Looking at the pattern across the two surveys, it appears that how large the collider bias is depends on how strongly wealth prevents the conditioning event. Where wealth does little to delay onset, the people who become dependent are no more selected among the rich than among the poor so that no spurious association arises. The bias should therefore be largest where the protective effect on onset is largest. That is the direction of what we find although the evidence is suggestive rather than decisive. The instrumented onset gradient is somewhat steeper in the United States than in Europe, at 0.982 against 0.985, and the mortality reversal appears in the United States and is absent in Europe. We then also report expected years lived autonomous and dependent by wealth tercile. Over an eight-year horizon the bottom wealth tercile spends 0.22 more years dependent than the top in Europe and 0.41 more in the United States. It also devotes roughly twice as large a share of its remaining life to dependency even though it dies earlier. Higher mortality among the poor truncates their dependent spells but nowhere near enough to offset their much earlier onset. The results also support the institutional prediction more strongly than we had anticipated. The burden of dependency is not uniformly higher in the United States. Outcomes in the highest wealth tercile are nearly identical on both sides of the Atlantic but the wealth gradient in dependency is approximately twice as steep in the United States. This gradient is also most pronounced among those in good physical health at baseline, for whom its magnitude is roughly twice that observed among those already impaired. Finally, it is about three times as steep among women as among men.

Our paper contributes to the literature in three main ways. First it extends the work on socioeconomic gradients in mortality (Chetty et al., 2016; Milligan and Schirle, 2021; Cutler and Lleras-Muney, 2006) to the morbidity process that precedes death. It is done using transition rates rather than prevalence. Second it complements the health–wealth nexus literature (Smith, 1999; Meer et al., 2003; Hurd and Kapteyn, 2003; Michaud and van Soest, 2008; Lefebvre et al., 2018; Connolly et al., 2025), and the smaller body of work estimating health transitions across countries (Banks et al., 2010; Jones et al., 2018; Hosseini et al., 2022), by treating wealth as endogenous and by taking the competing-risks and index-event problems seriously rather than as footnotes. Finally it speaks to the comparative LTC literature (Colombo et al., 2011; Barczyk and Kredler, 2018; Bakx et al., 2020) by asking not whether systems differ in generosity but whether they differ in how far private wealth determines who becomes dependent and when.

The rest of the paper is organized as follows. Section 2 describes the data, sample and variables. Section 3 sets out the empirical strategy. Section 4 presents the results for both transitions and combines them. Section 5 reports robustness and sensitivity analyses, Section 6 discusses the policy implications and Section 6 concludes.

2 Data and descriptive statistics

2.1 Sample selection

Our analysis draws on data from two major longitudinal ageing surveys, the *Survey of Health, Ageing and Retirement in Europe* (SHARE) and the U.S. *Health and Retirement Study* (HRS). SHARE is an international and interdisciplinary panel survey designed to collect microdata on individuals aged 50 and older across Europe. Conducted biennially since 2004, it gathers extensive information on respondents' physical and mental health, socioeconomic status, living conditions, and family networks. To date, more than 380,000 interviews have been carried out with approximately 140,000 individuals in 28 European countries and Israel. The *Health and Retirement Study* (HRS), launched in 1992, follows a nationally representative sample of about 20,000 Americans aged 50 and over, with comparable content on income, wealth, labour supply, pension coverage, health, functional limitations, cognition and medical spending. The two surveys were designed to be comparable (the HRS served as the template for SHARE) which makes them the standard basis for cross-national research on ageing (Börsch-Supan et al., 2013; Eibich et al., 2022). We use the RAND HRS Longitudinal File and the SHARE release harmonised on the same concepts so that health and limitattions items, wealth components and demographic variables are constructed identically on both sides of the Atlantic.

For the purpose of this study, we restrict attention to 2010–2020, corresponding to SHARE waves 4–8 and HRS waves 10–14. We keep the full age range of both surveys, that is respondents aged 50 and over, rather than restricting attention to the oldest group. Dependency before 65 is uncommon but excluding it would discard precisely the early transitions that we want to capture since low-wealth individuals are the ones who lose autonomy young. In our sample the median age at first observation is 66, and 45% of respondents in SHARE and 49% in the HRS are under 65 when first observed. A small number of respondents are younger than 50 (2.5% overall), because both surveys interview the partners of age-eligible respondents regardless of the partner's own age.Respondents must be non-dependent at their first observation. The risk set for the onset transition is by construction the set of individuals who have not yet experienced the event. We thus limit the sample to individuals for whom we have information on the dependency status from the first wave of observation and we keep individuals observed at least twice to ensure that transitions into dependency can be properly identified between survey waves and that both incidence and duration can be studied within a longitudinal framework.

This left-truncation-by-design is standard but has a cost. Individuals who are already dependent when first observed are excluded but they are likely to be disproportionately poor. Our estimates therefore describe the risk of incident dependency among those still autonomous at entry and, if anything, understate the population-level wealth gradient. A comparison of included and excluded respondents show that standardised differences are small for most characteristics but included respondents are on average two years older in SHARE and have somewhat better mental health, which is mechanically implied by the exclusion of the already-dependent.[2]

These restrictions leave an analysis cohort of 19,753 individuals in SHARE and 14,165 in the HRS, contributing 67,485 and 49,491 person-intervals to the onset analysis. Depending on the estimation and specifications, the estimated samples can be smaller (due to missing covariates or risk sets conditions), we therefore report the number of individuals, the number of events and the number of person-intervals in each table. We note in the text wherever a comparison between columns could be affected by the difference.

2.2 Variables of interest

Dependency. Both surveys elicit difficulties with six basic *Activities of Daily Living* (ADL). These are (i) dressing, including putting on shoes and socks; (ii) walking across a room; (iii) bathing or showering; (iv) eating, such as cutting up food; (v) getting in or out of bed; and (vi) using the toilet. Following

[2]Results are available upon request.

standard practice, we define an individual as dependent when she reports difficulty with at least two of the six items. This threshold is not arbitrary since two ADL limitations is the criterion used by most private LTC insurance contracts and by several public schemes to trigger benefit eligibility. It corresponds to a level of need that cannot generally be met without regular personal assistance.[3]

Two measurement issues deserve attention. First, ADL limitations are self-reported and may vary with socioeconomic status. However, reporting bias is generally stronger for global health measures than for specific functional limitations (Bound, 1991; Baker et al., 2004; Jürges, 2007). Furthermore studies correcting for differential reporting find that poorer individuals tend to under-report ill health. This means that adjustment typically widens socioeconomic health gaps (Bago d'Uva et al., 2008; Dowd and Todd, 2011; Rossouw et al., 2018). Moreover, our gradient becomes stronger at more severe disability thresholds, which is difficult to reconcile with reporting heterogeneity alone. Second, because SHARE is biennial, the timing of dependency onset is interval-censored. Following standard practice, we date onset at the interview when dependency is first reported (Laditka and Wolf, 1998; Crimmins et al., 2009; Banks et al., 2010). Section 5.4 shows that midpoint imputation and a discrete-time complementary log-log model for grouped duration data (Allison, 1982; Jenkins, 1995) yield similar results.

Wealth Household wealth is our main proxy for socioeconomic status. Among older individuals, it better captures long-term economic resources than current income, which may be affected by retirement, health shocks, or pension-system features. Wealth reflects accumulated earnings, savings, transfers, and assets and is therefore a more stable measure of permanent socioeconomic position (Smith, 1999; Banks et al., 2003). It is also directly relevant to the ability to finance health and long-term care (De Nardi et al., 2010) and is often more strongly associated with health at older ages than income (Hurd and Kapteyn, 2003; Meer et al., 2003; Eibich et al., 2022).

We measure household net wealth as total real, financial, business, and vehicle assets minus liabilities. We use wealth observed at the individual's first panel wave and treat it as predetermined with respect to subsequent health transitions. It limits reverse causality from dependency or care expenditures to asset depletion. Baseline wealth thus captures long-term economic position rather than short-run financial fluctuations. To ensure comparability across countries, we express wealth as percentiles of the within-country and within-wave distribution. This avoids problems related to currencies, inflation, and cross-country differences in asset prices and limits the influence of extreme wealth values and measurement error in the upper tail. The resulting coefficient captures the effect of moving up the national wealth

[3]We use ADL rather than Instrumental Activities of Daily Living (IADL) because ADL limitations capture more severe and clinically meaningful dependency, whereas IADL difficulties often reflect mild or early declines and are more sensitive to gender-specific task familiarity. Section 5.4 reports results for alternative definitions (one or more ADL, three or more ADL, and a combined ADL/IADL index) and shows that the wealth gradient is essentially unchanged across them.

distribution. Robustness checks in Section 5.2 show that results are robust to using log net worth, wealth quintiles, financial wealth excluding housing, and equivalized wealth.

Covariates All specifications control for gender, age and age squared, being single, having at least one living child and baseline indicators of poor physical and mental health. The quadratic age term captures the non-linear increase in functional decline. Marital status and children proxy control for access to informal care. Baseline health controls account for pre-existing vulnerability and help separate the effect of wealth on subsequent transitions from the contemporaneous wealth–health correlation. European specifications also include country fixed effects.

For the heterogeneity analysis we also group the European countries into four regions with broadly distinct long-term care regimes, namely North (Sweden, Denmark), Centre (Austria, Germany, the Netherlands, France, Switzerland, Belgium), South (Spain, Italy, Portugal, Greece) and East (Czechia, Poland, Slovenia, Estonia, Croatia and other Central and Eastern European countries), following the standard typology in the comparative LTC literature (Srakar and Nagode, 2018; Uccheddu et al., 2019; Lefebvre et al., 2025; De Donder et al., 2026).

2.3 Descriptive statistics

Table 1 reports baseline characteristics by wealth tercile and survey. In both SHARE and HRS, respondents in the bottom tercile are more likely to be female and far more likely to be single, reflecting both the wealth losses associated with widowhood and the joint accumulation of assets within couples. They are less likely to have children and they report markedly worse health. The age profile is non-monotonic and in the United States the bottom tercile is younger on average than the top. The youngest respondents are concentrated in the lower terciles, while the oldest are drawn towards the middle and top through a combination of accumulation and the selective mortality of the poor. It is a further reason to control for age flexibly as we do throughout.

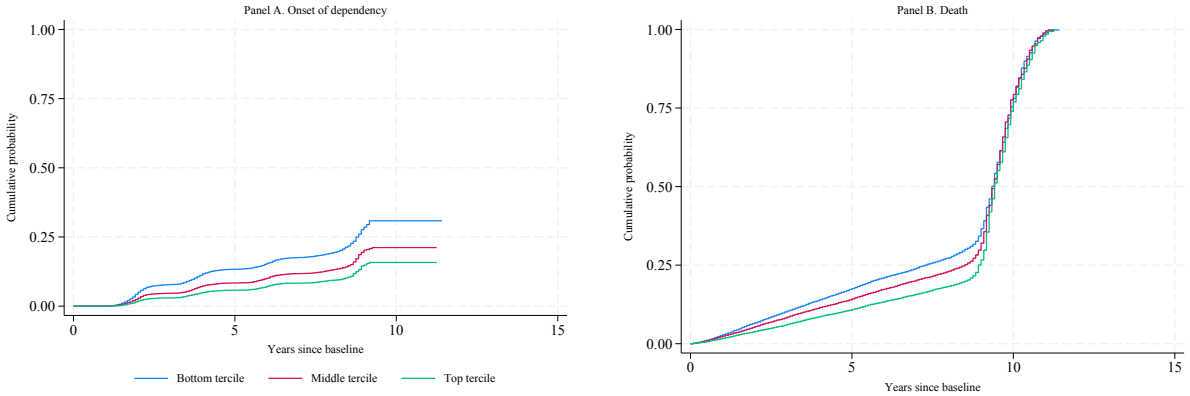
Figure 1 traces the two outcomes over follow-up. Cumulative incidence of dependency and cumulative mortality both decline monotonically with wealth tercile and the gaps open early and widen throughout. After four years, the cumulative probability of having entered dependency is roughly twice as high in the bottom tercile as in the top. The same ordering holds for mortality. It means that the group most at risk of dependency is also the group most likely to leave the risk set before dependency is observed. This is something we explore through additional analysis with competing-risks analysis (see below).

Table 1: Baseline characteristics of the estimation sample, by wealth tercile

	Bottom tercile		Middle tercile		Top tercile	
	SHARE	HRS	SHARE	HRS	SHARE	HRS
Age	68.87	62.57	67.23	66.83	65.77	67.86
	(10.97)	(11.62)	(9.98)	(11.54)	(9.21)	(10.63)
Female (%)	60.1	61.5	55.1	57.2	51.6	53.0
	(49.0)	(48.7)	(49.7)	(49.5)	(50.0)	(49.9)
Single (%)	48.7	49.3	30.3	31.5	20.9	20.6
	(50.0)	(50.0)	(46.0)	(46.4)	(40.6)	(40.5)
At least one child (%)	88.3	91.7	91.2	93.3	92.5	92.9
	(32.1)	(27.6)	(28.4)	(25.1)	(26.4)	(25.7)
Poor mental health (%)	32.5	27.8	25.3	16.5	21.9	10.9
	(46.8)	(44.8)	(43.5)	(37.1)	(41.4)	(31.1)
Poor physical health (%)	53.5	59.0	47.5	58.8	42.6	53.2
	(49.9)	(49.2)	(49.9)	(49.2)	(49.4)	(49.9)
Observations	6,590	4,731	6,579	4,724	6,572	4,727

Notes: Standard deviations in parentheses. Terciles are defined on baseline household net wealth within country and wave. The sample is restricted to respondents aged 50 and over who are alive and non-dependent at their first observation and observed at least twice.

Figure 1: Cumulative incidence of dependency and death by wealth tercile



Notes: Cumulative probabilities by years since baseline among respondents alive and non-dependent at baseline. Terciles are defined on baseline household net wealth within country. The left panel shows the first transition into dependency and the right panel shows death.

The tercile ordering in Figure 1 is not an artefact of one follow-up period. Figure A.1 in the Appendix reports the wave to wave transition rate into dependency separately for each interval, and the bottom tercile lies above the top in every one of them, so the cumulative gap builds steadily rather than opening at a single point in the panel.

3 Empirical strategy

Our analysis follows each individual through two successive transitions. The first is the passage from autonomy to dependency and the second, conditional on the first, is death after the loss of autonomy. Together these two transitions define the end-of-life trajectory that we seek to relate to socioeconomic status,

$$\texttt{Autonomous} \xrightarrow{\text{transition 1}} \texttt{Dependency} \xrightarrow{\text{transition 2}} \texttt{Death (once dependent)}$$

The risk set for the first transition contains, at each instant, every individual still autonomous and still under observation. As soon as an individual becomes dependent she leaves that risk set and becomes an entry point for the second transition. Individuals who die without a prior dependency episode, who leave the panel or who are still autonomous when follow-up ends are right-censored. We know they had not experienced the event by the time we stopped observing them but not what happened afterwards. Symmetrically, individuals still alive at the last available wave after entering dependency are right-censored in the second transition.[4]

3.1 Survival framework

Given the right-censoring of our data, we rely on survival analysis that let censored individuals contributing exposure up to their last observation and model the time dynamics of the transition directly.[5] The Cox partial likelihood does so while leaving the baseline hazard $\lambda_{0k}(t)$ completely unrestricted. This is valuable since we do not have prior on the shape of the age profile of functional decline.

Let T_i denote the duration until the relevant event. For transition $k \in \{\text{onset, post-dependency survival}\}$ we estimate a Cox proportional hazards model (Cox, 1972),

$$\lambda_k(t \mid W_{i,t_0}, X_{it}) = \lambda_{0k}(t) \exp\left(\beta_{1k} W_{i,t_0} + X_{it}' \beta_{2k}\right), \tag{1}$$

where $\lambda_{0k}(\cdot)$ is an unspecified baseline hazard, W_{i,t_0} is the baseline wealth percentile and X_{it} is a set of

[4] Recovery from dependency, the reverse of transition 1, is not modelled. It is not rare in our data, since about a third of the individuals ever observed dependent are observed to return to autonomy at least once, so this is a real restriction rather than a harmless simplification. It does not affect the onset transition, where first onset is first onset regardless of what follows. Section 5.6 recomputes the expected time in state of Section 4.4 without the restriction and shows that the conclusions are unchanged.

[5] A linear regression of the observed duration on wealth that dropped censored individuals would be estimated on the selected sub-population of those who experienced the event earliest and would therefore be biased in a direction that depends on wealth. A binary regression that coded censored individuals as non-occurrences would keep the full sample but discard the timing of the event, which is the object of interest.

covariates (as presented in Section 2), together with country fixed effects in the pooled and SHARE specifications. Time is measured in months since the relevant origin. Because W_{i,t_0} is a percentile, $\exp(10\,\beta_{1k})$ is the proportional change in the hazard associated with moving ten percentiles up the national wealth distribution. It is the quantity that we report in the text. The two transitions differ only in their risk sets and time origins. In all specification standard errors are clustered at the individual level.

Treating death before dependency as ordinary censoring is valid only if censoring is independent of the latent time to dependency. The mortality transition also requires caution because it conditions on dependency, an outcome itself affected by wealth. Dependency therefore acts as a collider, inducing an association between wealth and unobserved determinants of survival (Hernán et al., 2004; Dahabreh and Bibbins-Domingo, 2024). Intuitively, if frailty increases both dependency and mortality while wealth protects against dependency, wealthy individuals who nevertheless become dependent are likely to be more frail, on average, than poorer dependent individuals. Conditioning on dependency can therefore make wealth appear to increase subsequent mortality. A higher mortality rate among wealthy dependent individuals should thus not be interpreted as evidence that wealth is harmful, but may instead reflect selection induced by wealth's protective effect on dependency onset.

We therefore report, alongside the conditional hazards, the cumulative incidence of dependency from a competing-risks estimator defined on the full baseline population (Section 4.3). In addition, in Section 4.4, we present the expected time spent in each state, estimated on the baseline cohort. The latter is actually informative since it gives the expected number of dependent person-years generated by each wealth group.

3.2 Adressing endogeneity of wealth

A causal interpretation of β_{1k} in (1) requires baseline wealth to be conditionally independent of unobserved determinants of the hazard. This may fail if latent health, preferences or access to informal care affect both wealth accumulation and health outcomes (Smith, 1999; Meer et al., 2003). Reverse causality may also arise if health deterioration already under way at baseline depletes assets. Measurement error in self-reported wealth may also attenuate estimates.

We address these concerns using two-stage residual inclusion (2SRI), a control-function approach appropriate for nonlinear models such as the Cox model. Standard 2SLS is generally inconsistent in this setting because replacing an endogenous regressor with its predicted value does not preserve the nonlinear structure of the outcome model (Terza et al., 2008; Tchetgen Tchetgen et al., 2015). Instead, 2SRI includes the first-stage residual in the survival model to account for the component of wealth correlated

with unobserved determinants of the hazard. This procedure effectively adjusts for the unobserved confounding that induces endogeneity, assuming the residual captures the part of the endogenous variable correlated with the error term in the survival model.

In the first stage we regress baseline wealth on the instrument and the full set of covariates,

$$W_{i,t_0} = \gamma_0 + \gamma_1 I_{i,t_0} + X'_{it}\gamma_2 + \nu_i, \tag{2}$$

where I_{i,t_0} is the instrument in the beginning of the period, and ν_i is the residual term capturing unobserved variation in wealth. We estimate this stage using ordinary least square and retain the residuals $\hat{\nu}_i$ for the second stage in which we augment the Cox model,

$$\lambda_k(t \mid W_{i,t_0}, \hat{\nu}_i, X_{it}) = \lambda_{0k}(t) \exp\left(\beta_{1k} W_{i,t_0} + X'_{it}\beta_{2k} + \beta_{3k}\hat{\nu}_i\right). \tag{3}$$

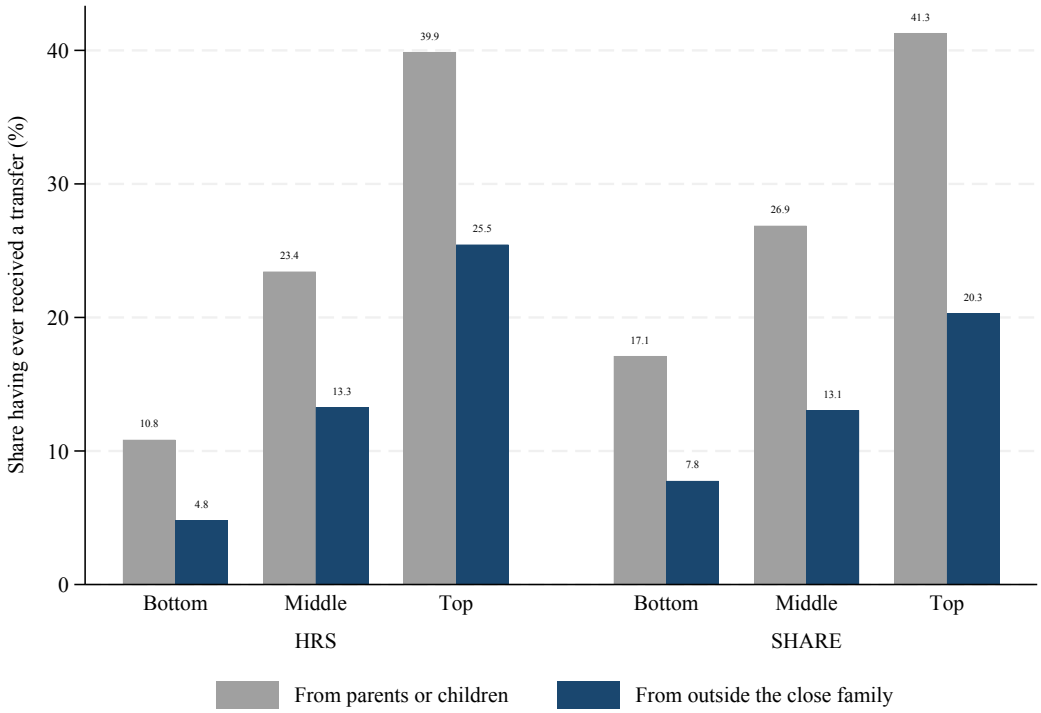
The residual proxies for the component of wealth correlated with the error term, so that β_{1k} recovers the structural effect. The coefficient β_{3k} is a direct test of exogeneity, since under the null of no endogeneity $\beta_{3k} = 0$. The conventional Cox standard errors are invalid because $\hat{\nu}_i$ is a generated regressor. We therefore obtain standard errors by non-parametric bootstrap, resampling individuals (not person-intervals) and re-estimating the complete two-stage procedure in 200 replications.

A significant coefficient β_3 provides evidence of endogeneity. Under standard assumptions (namely instrument relevance, exclusion restriction, and monotonicity) this procedure yields consistent estimates of the causal effect of wealth on both the hazard of becoming dependent and the hazard of death once dependent.

Our instrument I_{i,t_0} is an indicator for having ever received an inheritance or an inter vivos gift from a donor outside the close family; that is from someone other than a parent or a child. The exclusion restriction requires that, conditional on X_{it}, such transfers affect the hazard of dependency only through wealth. A bequest from a parent is informative about parental longevity and parental health, which are genetically and environmentally correlated with the respondent's own functional trajectory. It may also proxy for childhood circumstances, which Meer et al. (2003) identify as the principal threat to inheritance-based instruments. Transfers from more distant relatives carry far less of this information while still shifting resources materially.

Figure 2 shows the distribution of the instrument. The receipt of inheritances and gifts rises steeply with wealth in both surveys and the gradient is preserved when transfers from parents and children are removed.

Figure 2: Receipt of inheritances and gifts by wealth tercile and survey



Notes: Share of respondents having ever received a transfer, by baseline wealth tercile. "From family" denotes transfers from parents or children; "without close family members" denotes transfers from all other donors and is the variable used as instrument.

Table 2 gives the first-stage coefficient, its standard error and the F-statistic on the excluded instrument for the pooled sample and for every subsample used in Section 4. Having received a transfer from outside the close family is associated with a position 13.9 percentiles higher in the polled sample and 11.7 percentiles higher in the European wealth distribution and 15.5 percentiles higher in the American one. All are significnatly different than zero. The instrument is strong everywhere. The F-statistic is 456 in SHARE, 755 in the HRS and 1,216 pooled, and it never falls below 34 in any subsample. The remaining diagnostics, such as weak-instrument-robust inference and sensitivity to violations of the exclusion restriction, concern the second-stage estimate itself and are presented in Section 5.1.

Table 2: First-stage strength, by sample and subsample

	SHARE				HRS			
	Coef.	(S.E.)	First-stage F	N	Coef.	(S.E.)	First-stage F	N
Panel A. Main estimation samples								
Pooled (SHARE + HRS)	*Coef.=13.862*		*S.E.=0.398*		*First-stage F =1216*		*N =105,325*	
SHARE only	11.655	(0.546)	455.9	57,372				
HRS only					15.502	(0.564)	755.3	47,953
Panel B. Subsamples used in the heterogeneity analysis								
Women	12.174	(0.728)	279.7	32,457	16.059	(0.756)	451.6	27,980
Men	10.968	(0.824)	177.3	24,915	14.788	(0.845)	305.9	19,973
Single	13.562	(1.043)	169.0	19,218	18.928	(1.156)	268.2	16,785
Partnered	10.783	(0.634)	288.9	38,154	14.222	(0.625)	518.1	31,168
Aged under 80	11.609	(0.574)	409.5	49,664	15.640	(0.616)	644.3	41,009
Aged 80+	11.590	(1.468)	62.3	7,708	14.127	(1.168)	146.2	6,944
Good mental health	11.189	(0.589)	360.6	43,292	14.915	(0.582)	657.8	39,790
Poor mental health	13.434	(0.954)	198.2	14,080	18.845	(1.309)	207.3	8,163
Good physical health	11.112	(0.665)	279.6	29,774	15.517	(0.862)	324.1	18,840
Poor physical health	12.265	(0.737)	277.0	27,598	15.437	(0.708)	475.2	29,113
Panel C. European regions								
North	9.346	(1.228)	57.9	7,482				
Centre	12.494	(0.741)	284.4	23,780				
South	10.876	(1.872)	33.8	8,395				
East	11.709	(1.300)	81.1	17,715				

Notes: Each row is a separate first-stage OLS regression of the baseline household wealth percentile on an indicator for having ever received an inheritance or inter vivos gift from outside the close family, together with the full covariate set of Table 3 and country fixed effects (pooled and SHARE specifications), estimated on the risk set for the onset transition. Only the coefficient on the excluded instrument is reported. Standard errors are clustered on the individual, and N is the number of person-intervals. The first-stage F is the test that the coefficient on the excluded instrument is zero; with one excluded instrument it equals the square of the corresponding t-statistic. Regional subsamples are within SHARE.

4 Results

We first estimate the association between wealth and dependency onset, then examine survival after onset. Because wealth affects who enters the latter risk set, we discuss how this selection shapes the interpretation of both hazard ratios. We then account for death before dependency as a competing risk and estimate expected years lived autonomous and dependent over a fixed horizon for the baseline population.

Finally, we examine heterogeneity by region, gender, marital status, age and baseline health.

4.1 The onset of dependency

Table 3 reports the estimates for the first transition. Columns (1)–(3) present Cox models for the pooled sample, SHARE and the HRS; columns (4)–(6) the corresponding 2SRI specifications. All coefficients are exponentiated and read as hazard ratios.

Table 3: Hazard of becoming dependent

	(1) All	(2) SHARE	(3) HRS	(4) All 2SRI	(5) SHARE 2SRI	(6) HRS 2SRI
Household wealth percentile	0.989***	0.993***	0.987***	0.982***	0.985**	0.982***
	(-18.38)	(-8.21)	(-15.60)	(-5.04)	(-2.41)	(-3.94)
Female	0.871***	0.834***	0.934	0.871***	0.826***	0.939
	(-4.14)	(-3.85)	(-1.45)	(-4.14)	(-4.02)	(-1.33)
Age	0.863***	0.929**	0.867***	0.883***	0.949	0.877***
	(-9.59)	(-2.55)	(-7.92)	(-6.58)	(-1.58)	(-6.14)
Age squared	1.001***	1.001***	1.001***	1.001***	1.001***	1.001***
	(12.97)	(5.06)	(10.22)	(9.64)	(3.58)	(8.89)
Single	1.009	1.009	1.014	0.901	0.902	0.943
	(0.26)	(0.19)	(0.28)	(-1.60)	(-1.05)	(-0.66)
At least one child	0.921	0.966	0.896	0.911*	0.969	0.874
	(-1.49)	(-0.48)	(-1.28)	(-1.69)	(-0.44)	(-1.51)
Poor mental health	2.237***	2.194***	2.192***	2.136***	2.123***	2.118***
	(24.28)	(17.07)	(16.68)	(19.07)	(14.41)	(12.88)
Poor physical health	1.799***	1.600***	2.158***	1.734***	1.550***	2.093***
	(15.12)	(9.34)	(12.57)	(12.74)	(7.79)	(10.67)
First-stage residual				1.008**	1.008	1.005
				(2.08)	(1.32)	(0.99)
Individuals	33,253	19,365	13,888	33,253	19,365	13,888
Dependency events	4,124	2,067	2,057	4,124	2,067	2,057
Person-intervals	105,325	57,372	47,953	105,325	57,372	47,953

Notes: z-statistics in parentheses and standard errors clustered on the individual. Country fixed effects are included in the pooled and SHARE specifications. Columns (4)-(6) add the first-stage residual from a regression of the baseline wealth percentile on receipt of transfers from outside the close family and the full covariate set. Each individual contributes one person-interval per pair of consecutive interviews for as long as they remain autonomous. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

The wealth gradient is negative, highly significant and economically large. In the pooled Cox model the hazard ratio is 0.989, so ten additional wealth percentiles lower the instantaneous risk of losing autonomy by about 10%. Moving from the middle of the bottom tercile to the middle of the top (roughly 67 percentile points, using the tercile centroids) implies a hazard about half as large. Without instrumenting, the gradient is steeper in the United States (0.987, or 12.5% per ten percentiles) than in Europe (0.993, or 6.6%).

The 2SRI estimates are uniformly larger in absolute value than the previous estimates. The first-stage residual enters with a hazard ratio slightly above one and is marginally significant in the pooled and HRS

specifications so that the exogeneity of wealth is rejected. The naive estimate understates the protective effect of wealth which means there is likely attenuation from measurement error and confounding by unobserved ability; more than reverse causality.[6] Interestingly, our results with the instrument variable change the transatlantic comparison. The naive estimates put the American gradient ahead of the European one but the instrumented estimates are close. This could mean that measurement error and unobserved heterogeneity for which the instrument corrects are more severe in the European data. Once they are corrected, the two populations look more alike on this margin than the raw hazard ratios suggest. The transatlantic contrast that survives is therefore about precision and about the expected years results of Section 4.4, not about the onset gradient itself.

The covariates behave as expected. Baseline poor mental health nearly quadruples the hazard of dependency and poor physical health nearly triples it. The total effect of lifetime socioeconomic position is larger. Women face a hazard about 20% lower than men conditional on age and health, consistent with the well-documented male-female health-survival paradox operating in the opposite direction for severe ADL limitations at these ages. Marital status and the presence of children are not significant once health is controlled for.

4.2 Survival once dependent

Table 4 turns to the second transition and the results are much different than in Table 3. In the baseline Cox specifications the wealth coefficient is indistinguishable from one in every sample. Once dependency has occurred, the wealth gradient in survival has disappeared. In the 2SRI specifications the coefficient in the HRS sample becomes significant and is equal to 1.015, implying a 16.1% higher mortality hazard per ten wealth percentiles. The first-stage residual is significantly below one in the same column, again rejecting exogeneity. The raw survival curves tell the same story before any covariate is introduced. Figure A.3 in the Appendix plots Kaplan–Meier survival from the date of first observed dependency by wealth tercile, and the three curves are close together in both surveys, with no visible ordering in Europe and, if anything, the top tercile slightly below the others in the United States.

The apparent mortality disadvantage of wealth among dependent Americans may reflect selection rather than a harmful causal effect. As explained in Section 3.1, this specification conditions on becoming dependent, an event that wealth itself helps prevent. Wealthier individuals who nevertheless become

[6] This result differs from the usual concern that associations between wealth and health overstate the causal effect because poor health depletes assets (Adams et al., 2003; Poterba et al., 2017). Studies using inheritances or lottery wins generally find smaller effects or none (Meer et al., 2003; Kim and Ruhm, 2012; Cesarini et al., 2016; Michaud and van Soest, 2008). In our setting, wealth is measured before the observation window and we control for baseline health which limits this source of reverse causality. Measurement error in self-reported wealth percentiles may therefore be more consequential and attenuate the naive estimate.

Table 4: Hazard of death after the onset of dependency

	(1) All	(2) SHARE	(3) HRS	(4) All 2SRI	(5) SHARE 2SRI	(6) HRS 2SRI
Household wealth percentile	1.000	0.999	1.002	1.007	1.002	1.015**
	(0.28)	(-0.41)	(1.59)	(1.40)	(0.21)	(2.53)
Female	0.653***	0.583***	0.702***	0.668***	0.590***	0.724***
	(-6.99)	(-5.99)	(-4.25)	(-6.54)	(-4.81)	(-3.84)
Age	1.118***	1.084	1.102**	1.098**	1.080	1.084
	(3.10)	(1.29)	(2.15)	(2.34)	(1.29)	(1.28)
Age squared	1.000	1.000	1.000	1.000	1.000	1.000
	(-1.62)	(-0.47)	(-1.07)	(-1.12)	(-0.43)	(-0.69)
Single	1.115*	1.110	1.107	1.219**	1.151	1.303**
	(1.67)	(1.09)	(1.12)	(2.16)	(0.84)	(2.33)
At least one child	0.952	0.995	0.824	0.948	0.990	0.840
	(-0.51)	(-0.04)	(-1.33)	(-0.56)	(-0.07)	(-1.12)
Poor mental health	1.215***	1.276***	1.142*	1.246***	1.285**	1.182*
	(3.34)	(2.81)	(1.67)	(3.35)	(2.56)	(1.95)
Poor physical health	1.232**	1.057	1.709***	1.244**	1.069	1.675***
	(2.31)	(0.51)	(3.14)	(2.28)	(0.57)	(2.88)
First-stage residual				0.993	0.997	0.987**
				(-1.40)	(-0.27)	(-2.26)
Individuals	2,984	1,462	1,522	2,984	1,462	1,522
Deaths	1,303	640	663	1,303	640	663
Person-intervals	5,007	2,222	2,785	5,007	2,222	2,785

Notes: See Table 3. The risk set comprises individuals observed to become dependent during follow-up, and duration is measured from the date of first observed dependency, with delayed entry. As in Table 3, person-intervals count records rather than people, and the number of individuals and the number of deaths are the quantities that govern precision. Median observed duration in the dependent state is 2.5 years in SHARE and 3.2 years in the HRS. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

dependent may therefore be unusually frail, whereas poorer individuals can enter dependency with less underlying frailty. Restricting the sample to dependent individuals can thus induce a positive association between wealth and unobserved frailty, biasing the estimated effect of wealth on mortality upward. This mechanism should be stronger where wealth offers greater protection against dependency. The contrast between the HRS and SHARE is directionally consistent with this prediction, although the difference in their onset gradients cannot by itself explain the difference in mortality estimates. These results therefore cannot be interpreted as evidence that wealth increases mortality after dependency begins. To avoid this selection problem, Section 4.4 examines outcomes defined over the baseline population rather than conditional on having become dependent.

A second interpretation, which is not exclusive of the first, is a compression of morbidity (Fries, 1980). If wealth buys support before the two ADL threshold is crossed, dependency among the wealthy begins later and therefore closer to the end of life. In that case, wealth would shorten the dependent phase as well as postpone it. Unlike the selection account, this one describes a real effect on duration rather than an artefact of conditioning and the two are observationally very similar in Table 4. Separating them

again calls for estimates defined on the baseline population, which is where we turn in Section 4.3 and Section 4.4.

Two further results in Table 4 are worth noting because they are robust across specifications. Women survive substantially longer once dependent. It is the mirror image of their lower onset hazard and it implies a considerably longer expected dependent spell for women. Being single raises the mortality hazard once dependent by 20-30% in the instrumented specifications, whereas it had no effect on onset. Informal care appears to matter for surviving dependency but not for avoiding it.

4.3 Mortality selection and competing risks

The onset analysis treats death before dependency as censoring. This is valid only if death is uninformative about the latent dependency time. Figure 1 tends to say the contrary since the bottom tercile has both the highest dependency incidence and the highest mortality. Figure A.2 in the Appendix shows what that does to a naive measure. It plots the share of the baseline cohort alive and dependent at each wave, keeping the deceased in the denominator, and the tercile gap in that prevalence measure is far narrower than the gap in incidence, because the poor who would have been dependent have died instead. Any statistic that conditions on being alive will understate the gradient for this reason. Table 5 therefore re-estimates the first transition under four alternative treatments of death. In our sample, individuals begin in the autonomous state and can leave by becoming dependent or by dying while still autonomous. This is a competing-risks structure and so cause-specific hazards and cumulative incidence functions should both be reported (Latouche et al., 2013; Austin et al., 2016; Andersen et al., 2012).

Column (1) in Table 5 reports the baseline Cox model of Section 4.1 but estimated on a sample where individuals who die beforehand are removed from the risk set at death and treated as censored. The estimand is the instantaneous rate of losing autonomy among those still alive and still autonomous. So doing, we can see if wealth affectx functional decline for a person who is still at risk but we cannot see how likely a person is to ever become dependent because the risk set has been selectively depleted by death. Column (2) then uses the same construction with the roles reversed and the event is death among the still-autonomous and those who become dependent are censored. It is helpful for diagnostic becasue if wealth predicts this hazard, then the censoring applied in column (1) is informative. The risk set there is progressively purged of its poorest members and the cause-specific estimate understates the population gradient. In column (3), we estimate the Fine–Gray subdistribution hazard. This model targets the cumulative incidence function directly by looking at the probability of having become dependent by time t but acknowledging that some people die first and can therefore never do so (Fine and Gray, 1999). The

difference from column (1) is that individuals who die without dependency are not removed from the risk set. Actually, they remain in the set after death with a declining weight so that their inability to become dependent is counted rather than discarded. A subdistribution hazard ratio below one therefore means a lower lifetime probability of becoming dependent. Finally in column (4), the event is whichever comes first: dependency or death. In that case, the model describes exit from the state "alive and autonomous". Because the two exits are pooled, no competing risk arises by construction and the estimate is free of the selection problem altogether. It allows to look at time spent alive and independent, something we also look at in the next Section 4.4.

Table 5: Mortality selection and competing risks

	(1) Cox, dependency		(2) Cox, death		(3) Fine–Gray		(4) Cox, either event	
	HRS	SHARE	HRS	SHARE	HRS	SHARE	HRS	SHARE
Wealth percentile	0.986***	0.993***	0.993***	0.995***	0.987***	0.994***	0.990***	0.994***
	(0.0009)	(0.0009)	(0.0008)	(0.0006)	(0.0009)	(0.0009)	(0.0006)	(0.0005)
Female	0.878**	0.827***	0.613***	0.504***	0.952	0.932	0.712***	0.591***
	(0.045)	(0.040)	(0.027)	(0.018)	(0.049)	(0.045)	(0.024)	(0.017)
Age	0.859***	0.930***	1.012	0.969*	0.919***	1.042	0.917***	0.957***
	(0.017)	(0.026)	(0.023)	(0.018)	(0.019)	(0.031)	(0.014)	(0.015)
Age squared	1.001***	1.001***	1.000***	1.001***	1.001***	1.000	1.001***	1.001***
	(0.0001)	(0.0002)	(0.0002)	(0.0001)	(0.0001)	(0.0002)	(0.0001)	(0.0001)
Single	1.025	0.949	1.157***	1.151***	1.041	0.935	1.091**	1.082**
	(0.058)	(0.049)	(0.059)	(0.044)	(0.058)	(0.049)	(0.041)	(0.034)
At least one child	0.886	0.932	0.719***	0.833***	0.954	0.955	0.787***	0.861***
	(0.083)	(0.070)	(0.053)	(0.043)	(0.091)	(0.073)	(0.045)	(0.036)
Poor mental health	2.155***	2.043***	1.377***	1.380***	1.995***	1.857***	1.695***	1.578***
	(0.115)	(0.097)	(0.075)	(0.052)	(0.106)	(0.089)	(0.066)	(0.047)
Poor physical health	2.093***	1.839***	1.773***	1.228***	1.961***	1.804***	1.903***	1.388***
	(0.125)	(0.096)	(0.088)	(0.041)	(0.119)	(0.094)	(0.071)	(0.038)
Individuals	13,713	19,112	13,713	19,112	13,713	19,112	13,713	19,112

Notes: standard errors in parentheses. Column (1) is a cause-specific Cox model for dependency onset, with death treated as censoring. Column (2) is a cause-specific Cox model for death without prior observed dependency, with dependency treated as censoring. Column (3) reports Fine–Gray subdistribution hazards for dependency with death as competing event. Column (4) is a Cox model for exit from the state "alive and independent", where the event is dependency or death, whichever comes first. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

In Table 5, we first see in columns (1) that mortality selection is empirically real. Columns (2) show that wealth significantly reduces the hazard of dying without having been observed dependent, in both surveys. The risk set for the onset transition is therefore progressively purged of its poorest members and a cause-specific hazard understates the population gradient. However selection does not explain all results. The Fine-Gray subdistribution hazard in columns (3), which by construction keeps individuals who die in the risk set and therefore targets the cumulative incidence of dependency in the presence of competing mortality, remain significantly below one. Moreover in the HRS, it is essentially identical to the cause-specific estimate (0.987 against 0.986). Wealthier individuals have a lower cumulative probability of ever becoming dependent but not merely a lower conditional hazard. Third, columns (4) provide

a single summary and shows that wealth reduces the hazard of exiting the state "alive and autonomous" through either channel. Saying it differently, poorer individuals spend less time alive and independent.

The comparison between columns (1) and (3) also illuminates the gender result. The cause-specific hazard for dependency is lower for women and their mortality hazard is far lower. Once competing mortality is accounted for, the Fine-Gray subhazard for women is much higher and insignificant. Almost all of women's apparent protection against dependency in the cause-specific model is attributable to their much longer survival.

4.4 Expected years of autonomous and dependent life

The previous results show that wealth strongly delays onset but seems unrelated to survival once dependent. We have explained why the second result cannot be read causally but it does not tell us what the two margins imply together. In particular it would be interesting to know how many years a person can expect to live autonomous and how many dependent.

We therefore estimate, over a horizon τ, the restricted mean time spent in each state,

$$\mathrm{RMT}_j(\tau) = \int_0^\tau P_j(u)\,\mathrm{d}u, \qquad j \in \{\text{autonomous, dependent, dead}\},$$

where $P_j(\cdot)$ are state occupation probabilities in the dependent-death model. Because recovery is not modelled, these follow from two Kaplan–Meier curves estimated on the baseline cohort. That is we measure the survival function for exit from autonomy (the first of dependency or death) and the survival function for death from any state. Expected years autonomous is the restricted mean of the first and expected years alive is the restricted mean of the second. Taking the difference between both gives the expected years dependent. Neither quantity conditions on dependency so that the index-event problem does not arise. Both keep individuals who die in the denominator which prevents the mortality selection to arise. We set $\tau = 8$ years, the median follow-up in both panels and obtain confidence intervals by resampling individuals 400 times. The eight year horizon runs from each individual's own baseline interview rather than from a common age, so the quantities below are averages over the baseline age distribution of the cell. Mean age at baseline is 67.3 years in SHARE and 65.7 in the HRS.

The results in Table 6 show that in Europe, an individual in the bottom wealth tercile can expect 6.32 of the next eight years in autonomy and 0.45 years dependent, against 7.09 and 0.23 years at the top. In the United States the corresponding figures are 6.45 and 0.65 years at the bottom, against 7.06 and 0.24 at the top. The bottom tercile thus spends both fewer years autonomous and more years dependent,

Table 6: Expected years lived autonomous and dependent over eight years, by wealth tercile

	SHARE (Europe)			HRS (United States)		
	Bottom	Middle	Top	Bottom	Middle	Top
Years lived autonomous	6.32	6.83	7.08	6.45	6.70	7.06
Years lived dependent	0.45	0.29	0.23	0.65	0.38	0.24
Years lived (any state)	6.77	7.11	7.31	7.09	7.08	7.30
Years of life lost	1.23	0.89	0.69	0.91	0.92	0.70
Share of years alive spent dependent (%)	6.6	4.0	3.1	9.1	5.4	3.3
Difference, bottom − top tercile (95% bootstrap interval)						
Years lived dependent	+0.220 [+0.182, +0.261]			+0.408 [+0.353, +0.458]		
Years lived autonomous	-0.768 [-0.844, -0.690]			-0.617 [-0.707, -0.527]		
Share dependent (pp)	+3.511 [+2.972, +4.089]			+5.843 [+5.101, +6.546]		
Individuals	6,525	6,525	6,526	4,722	4,721	4,722

Notes: Restricted mean time in state over a horizon of $\tau = 8$ years are computed from Kaplan–Meier estimates of exit from autonomy and of overall survival on the baseline cohort. "Years of life lost" is τ minus expected years lived. Brackets report 95% percentile intervals from 400 individual-level bootstrap resamples. The horizon starts at each individual's baseline interview, not at a common age, so each cell is an average over its own baseline age distribution. Table A.1 reports the same quantities within bands of baseline age.

despite dying earlier. The higher mortality of the poor truncates their dependent spells but not by enough to offset their much earlier onset.

The gap is economically large and statistically signicantly different than zero. Relative to the top tercile, the bottom spends 0.22 additional years dependent in SHARE and 0.41 additional years in the HRS. Expressed as a share of the years actually lived, the contrast is starker still. In Europe 6.6% of remaining life is spent dependent at the bottom of the European wealth distribution against 3.1% at the top. It is 9.1% against 3.3% in the United States. The bottom–top gap in that share is 3.5 percentage points in Europe and 5.8 in the United States. These results show that the bottom-top gap in years lived dependent is close to twice as large in the United States as in Europe and the gap in the share of remaining life spent dependent is 1.7 times as large. Both ratios are of the same order as the ratio of the onset gradients in Table 3. Wealth is not associated with a markedly different average burden of dependency on the two sides of the Atlantic but it sorts people into that burden far more steeply in the United States. This raises questions on the role of institutions as we argued in the introduction.

Because the horizon starts at baseline rather than at a common age, these estimates depend on age composition. The bottom wealth tercile is older than the top in SHARE but younger in the HRS, amplifying the apparent disadvantage in Europe while attenuating it in the United States. Table A.1 in the Appendix repeats the analysis within three baseline age bands. Although expected years autonomous declines substantially with age, the wealth gradient persists in every band and both surveys. The transat-

lantic contrast also becomes sharper, with the bottom-to-top gap in years dependent two to four times larger in the HRS than in SHARE within age bands, compared with roughly twice as large in the pooled estimates. Repeating the exercise holding age, gender, marital status, fertility and baseline physical and mental health constant, by standardisation over the observed covariate distribution, leaves the gradient intact and the transatlantic ratio implied by these adjusted figures, close to three to one, is if anything larger than the unadjusted one (Table A.2 in the Appendix).[7]

4.5 Heterogeneity

We estimate the two transitions separately by region, gender, marital status, age group and baseline mental and physical health. We only report the 2SRI estimations which is the specification the rest of the paper uses. The full tables, with every covariate reported, are in Appendix E, and Figures 3 and 4 summarise the wealth hazard ratios, expressed per ten percentiles of the wealth distribution so that the magnitudes are readable.

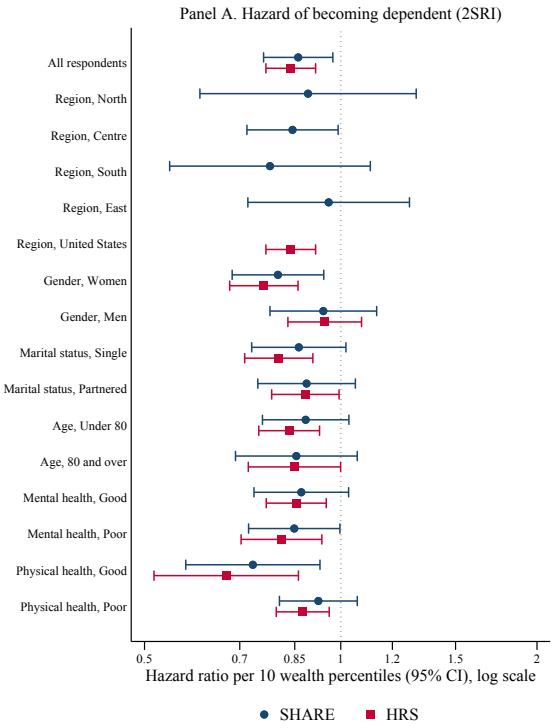
Region. The estimated protective effect of wealth against dependency does not differ significantly across European regions or between Europe and the United States. Differences concern the precision of the estimates rather than their magnitude, with only Central Europe showing a statistically significant effect. The regional pattern observed in the Cox models is therefore not robust to instrumentation. Among dependent individuals, higher wealth is significantly associated with higher mortality only in the United States, while the European estimates are too imprecise to draw firm conclusions.

Gender. Wealth is associated with a lower risk of becoming dependent among women in both Europe and the United States. The estimated relationship is weaker and not statistically significant among men. Among dependent Americans, higher wealth is associated with higher mortality only for women. This pattern is consistent with selection into dependency. If wealth delays dependency, wealthier women who nevertheless become dependent may be particularly frail which could explain their higher subsequent mortality. The European mortality estimates are too imprecise to draw clear conclusions. The stronger protective role of wealth among women may also reflect their greater reliance on paid care as they tend to spend longer in dependency and are more likely to be widowed and lack support from a spouse.

Marital status. Wealth appears more protective against dependency among single individuals than among those with a partner. This suggests that financial resources may partly compensate for the absence of spousal support. This pattern is clearer in the United States. Among dependent Americans, the

[7] We present the unadjusted estimates in the main text because they require no modelling assumptions beyond the two survival curves. In addition, holding baseline health constant conditions on a variable that is itself partly a consequence of lifetime wealth, so the adjusted figures answer the narrower question of what wealth adds among people who enter the panel in the same health.

Figure 3: Wealth gradient in the onset of dependency, by subgroup



Notes: 2SRI estimates of the hazard ratio on the baseline wealth percentile, from a separate estimation on each indicated subsample, with the full covariate set of Table 3 and country fixed effects (SHARE only). When the sample is split on a covariate, that covariate is dropped from both stages. Hazard ratios are expressed per ten percentiles of the within-country wealth distribution, so that a value of 0.90 means a 10% lower hazard for someone ten percentiles higher up. Horizontal lines are 95% confidence intervals from analytic standard errors clustered on the individual, as in Table 3.

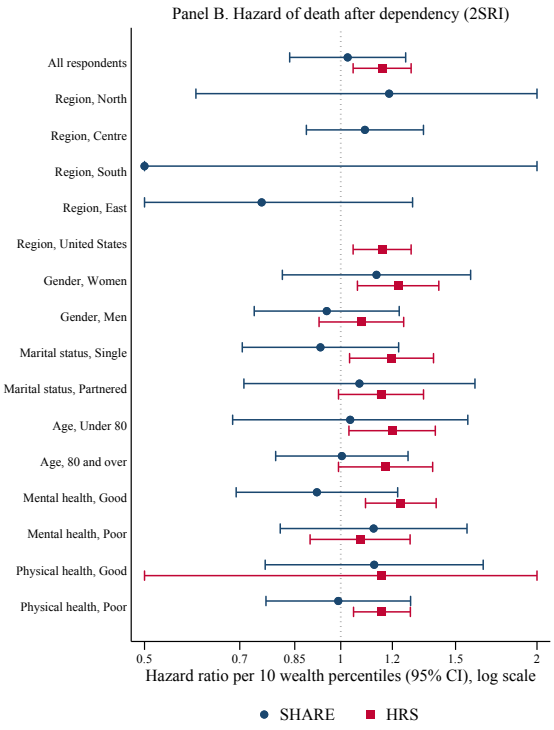
positive association between wealth and mortality is also slightly stronger for single individuals although the difference is small. The European mortality estimates do not support firm conclusions.

Age. The estimates are similar for individuals below age 80 and those aged 80 and over in both surveys. There is no clear age differences in the association of wealth with either dependency onset or subsequent mortality.

Baseline physical health. In both Europe and the United States, wealth is more protective among individuals initially in good physical health than among those already in poor health. This pattern supports a preventive interpretation, suggesting that financial resources may be particularly important before substantial physical deterioration occurs. The mortality results cannot meaningfully assess the corresponding selection mechanism. Too few deaths make the estimates for initially healthy Americans highly uncertain. The positive association between wealth and mortality is observed among those initially in poor health, who account for most deaths.

Baseline mental health. Mental health provides the only subgroup comparison that runs against the pattern predicted by selection into dependency. Wealth is slightly more protective against onset among

Figure 4: Wealth gradient in mortality after the onset of dependency, by subgroup



Notes: See Figure 3. The risk set comprises individuals observed to become dependent during follow-up, with duration measured from the date of first observed dependency. The axis range is the same as in Figure 3, so the two panels are directly comparable. Four intervals extend beyond the range and are drawn to the edge of the plot. All of them belong to cells with a weak first stage, and the widest, the European South, has an interval running from 0.001 to 150 and should be read as carrying no information at all.

individuals in poor mental health. Yet its positive association with mortality among dependent Americans is stronger among those in good mental health. Poor baseline mental health may also play a distinct role because it strongly predicts mortality after dependency, potentially making the contribution of wealth less apparent.

5 Robustness and sensitivity

5.1 Instrument validity

Section 3.2 established that the instrument is strong. Two further questions concern the estimate it delivers. The first is whether conventional inference on that estimate is reliable, and the second is how much of a violation of the exclusion restriction it would take to overturn it.

Weak-instrument-robust inference. Conventional Wald inference on the 2SRI coefficient relies on a first-order approximation that can be poor when the instrument is only moderately informative. We therefore invert an Anderson–Rubin test, which is valid regardless of instrument strength and does not depend

on the 2SRI estimator at all. Under the null $\beta_1 = b$, imposing bW_{i,t_0} as an offset in the hazard should leave the instrument with no residual explanatory power. We fit that model over a grid of b, test the coefficient on the instrument and collect the values of b that are not rejected at the 5% level. Panel B of Table 7 reports the resulting confidence sets. Both exclude a hazard ratio of one. The European set is wider but it too lies entirely below one. This is a stronger statement than the Wald-based result in Table 3 supports, and it changes the reading of the European estimate. The imprecision of the SHARE 2SRI coefficient reflects the limits of Wald inference in this setting rather than an absence of effect.

Sensitivity to violations of the exclusion restriction. Even distant-kin transfers may favour individuals from advantaged backgrounds, potentially overstating the protective effect of wealth. To assess sensitivity to this violation of the exclusion restriction, we conduct a bounding exercise in the spirit of Conley et al. (2012). We allow a share δ of the instrument's reduced-form association with dependency to operate directly rather than through wealth. We then the confidence intervals across the permitted range of direct effects. Here, $\delta = 0$ imposes the exclusion restriction while $\delta = 1$ allows the entire association to bypass wealth. Panel C shows that the estimated wealth effect remains significantly protective when this share reaches 0.5 in the HRS and 0.3 in SHARE. The results therefore withstand meaningful departures from the exclusion restriction although larger violations leave greater uncertainty about the causal effect.

Table 7: Weak-instrument-robust inference and sensitivity to the exclusion restriction

	SHARE		HRS	
	Per percentile	Per 10 percentiles	Per percentile	Per 10 percentiles
Panel A. Point estimate and conventional interval				
2SRI hazard ratio	0.9802	0.819	0.9773	0.795
Wald 95% interval	[0.9685, 0.9921]	[0.726, 0.923]	[0.9689, 0.9859]	[0.729, 0.867]
Panel B. Anderson–Rubin 95% confidence set (weak-instrument robust)				
AR set	[0.9622, 0.9920]	[0.680, 0.922]	[0.9688, 0.9859]	[0.729, 0.868]
Panel C. Conley et al. (2012) union of confidence intervals, by δ				
$\delta = 0$	[0.9685, 0.9921]	[0.726, 0.923]	[0.9689, 0.9859]	[0.729, 0.867]
$\delta = 0.1$	[0.9685, 0.9921]	[0.726, 0.923]	[0.9689, 0.9859]	[0.729, 0.867]
$\delta = 0.2$	[0.9685, 0.9945]	[0.726, 0.946]	[0.9689, 0.9888]	[0.729, 0.893]
$\delta = 0.3$	[0.9685, 0.9969]	[0.726, 0.970]	[0.9689, 0.9917]	[0.729, 0.920]
$\delta = 0.5$	[0.9685, 1.0018]†	[0.726, 1.018]	[0.9689, 0.9975]	[0.729, 0.975]
$\delta = 1$	[0.9685, 1.0116]†	[0.726, 1.123]	[0.9689, 1.0093]†	[0.729, 1.097]
Reduced-form coef. of instrument	-0.2277		-0.3636	

Notes: The table presents hazard of the onset of dependency. All specifications include the full covariate set of Table 3 and country fixed effects (SHARE only). Panel A reports the 2SRI point estimate with a conventional Wald interval based on model standard errors. Panel B reports the Anderson–Rubin 95% confidence set, obtained by imposing each candidate value of the wealth coefficient as an offset and testing the residual coefficient on the instrument; it is robust to weak identification and does not use the 2SRI estimator. Panel C reports the Conley et al. (2012) union of confidence intervals when the instrument is allowed a direct effect on the hazard of up to δ times its reduced-form coefficient, which is reported in the final row. † marks intervals that include a hazard ratio of one.

5.2 Alternative measures of wealth

Tables A.3 and A.4 in the Appendix re-estimate the two transitions with four alternative measures, namely the log of positive net worth, wealth quintiles, financial wealth excluding housing, and wealth adjusted for household size. The onset results are confirmed in the HRS for all four measures, with log net worth entering at 0.789 and a quintile step at 0.627, both highly significant, while in SHARE the point estimates lie below one throughout but are not individually significant, mirroring the main specification. For the second transition, the positive HRS coefficient is reproduced by every alternative measure (1.162 for log net worth, 1.365 per quintile), while SHARE remains flat, which confirms that the sign reversal is not an artefact of the percentile transformation.

5.3 The proportional hazards assumption and functional form

Proportional hazards. Appendix Table A.5 reports tests of the proportional-hazards assumption based on scaled Schoenfeld residuals. The tests provide no evidence of violations for wealth in either transition or survey. Rejections concern only baseline mental health in the European onset model and baseline physical health in the American onset and post-dependency models. All other covariates satisfy the assumption. As a robustness check, stratifying the baseline hazard by baseline health leaves the wealth coefficient essentially unchanged. This suggests that these violations do not materially affect our main findings.

Wealth rank linearity. Our specification assumes that each additional wealth percentile has the same proportional effect on dependency risk. Figure 5 relaxes that assumption with a restricted cubic spline in the wealth rank, estimated separately on each survey. In both panels the fitted log hazard stays close to the linear-in-rank line, and a joint test of the non-linear basis terms comes nowhere near rejecting, with $\chi^2(3) = 2.88$ and $p = 0.41$ in SHARE and $\chi^2(3) = 0.96$ and $p = 0.81$ in the HRS. The protective association of wealth is therefore spread across the distribution rather than concentrated at either end. In particular there is no threshold below which dependency risk rises disproportionately, which offers little support for a narrow eligibility cutoff based on this relationship alone.

Parametric alternatives. The Cox model leaves the baseline hazard unrestricted, which is a strength for estimating the gradient but means we never test whether a parametric shape would change it. Table 8 re-estimates the onset transition with Weibull and log-logistic baselines alongside the Cox specification. The Weibull results are hazard ratios and are directly comparable with the Cox column. The log-logistic model has no proportional hazards representation, so it is reported in the accelerated failure time metric, where a value above one means a longer time to dependency and is therefore the mirror image of a

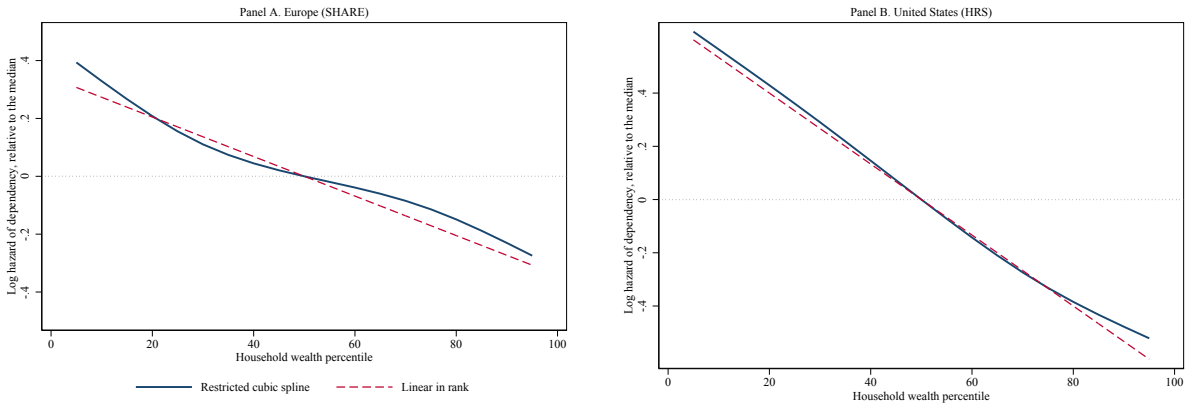


Figure 5: The wealth gradient in the hazard of dependency, flexibly estimated

Notes: Log hazard of the onset of dependency relative to the median of the wealth distribution, from a Cox model with a restricted cubic spline in the wealth percentile and the full covariate set, with country fixed effects in SHARE. The five knots sit at percentiles 5, 29, 51, 74 and 96, which are the quantiles Stata selects for a five-knot spline. The dashed line is the linear-in-rank specification used throughout the paper, centred on the median so that the two coincide there by construction. The left panel is SHARE and the right panel is the HRS, drawn on a common vertical scale. The legend applies to both panels.

hazard ratio below one. The Weibull hazard ratio per ten percentiles is 1.052 in SHARE and 1.099 in the HRS which corresponds closely to the Cox hazard ratios of 0.933 and 0.872. The log-logistic time ratios of 1.055 and 1.111 tell the same story. The transatlantic ordering is unchanged and every estimate is significant at better than the 1% level.

Table 8: Parametric alternatives to the Cox baseline hazard

	SHARE			HRS		
	Cox hazard ratio	Weibull hazard ratio	Log-logistic time ratio	Cox hazard ratio	Weibull hazard ratio	Log-logistic time ratio
Wealth percentile	0.933***	1.052***	1.055***	0.872***	1.099***	1.111***
	(-8.37)	(8.86)	(9.13)	(-15.62)	(14.86)	(15.43)
Person-intervals	57,372			47,953		

Notes: Coefficients on the baseline wealth percentile, expressed per ten percentiles, from the onset specification of Table 3 with the full covariate set and country fixed effects (SHARE only); z-statistics in parentheses. The Cox and Weibull columns report hazard ratios, so values below one indicate a lower risk of losing autonomy. The log-logistic column reports an accelerated failure time ratio, so values above one indicate a longer time to dependency. Analysis time is measured from entry into the panel. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

5.4 Alternative dependency thresholds and timing

Dependency status. Appendix Table A.6 examines alternative definitions of dependency, using thresholds of one, two and three ADL limitations and a broader measure covering any ADL or IADL difficulty. The wealth gradient remains similar across definitions in both surveys, including when milder limitations substantially increase the number of observed events. Although we expected a stronger gradient at more severe thresholds, where reporting differences might matter less, the estimated changes are small rela-

tive to their uncertainty. These findings show that the gradient is not driven by the chosen dependency threshold. They also provide some reassurance against reporting heterogeneity as the main explanation, although stability across thresholds cannot rule it out.

Timing. Because interviews occur every two years, dependency onset is known only to fall between interviews. Our baseline dates onset at its first report. Appendix Table A.7 considers two alternatives, assigning onset to the midpoint between interviews and estimating a discrete-time complementary log-log model that does not require an exact onset date. The latter produces estimates nearly identical to the baseline Cox results. Midpoint imputation leaves the Cox estimates unchanged and modestly weakens the instrumented effects, which remain statistically significant in both surveys. The findings are therefore robust to alternative treatments of onset timing, while the common biennial design limits concerns about differences in interview frequency across surveys.

5.5 Socioeconomic position

Wealth may capture broader socioeconomic advantages rather than financial resources alone. Table 9 examines this concern by estimating dependency onset with education alone, wealth alone and both together. Education is generally determined early in life and is therefore less exposed to reverse causality from later health deterioration. Both measures show substantial gradients that weaken but remain statistically significant when included jointly, with a larger attenuation of the wealth gradient in SHARE than in the HRS. Wealth therefore captures an association with dependency beyond that explained by education, consistent with a role for access to care, home adaptations and early treatment, although these results do not identify the underlying mechanisms. Education also retains an independent association, suggesting that a fuller account of socioeconomic inequalities in dependency should consider both dimensions. We regard that as beyond the scope of this paper and flag it as the obvious extension.

Table 9: Hazard of dependency with wealth and education as measures of socioeconomic status

	SHARE			HRS		
	(1)	(2)	(3)	(4)	(5)	(6)
	Educ.	Wealth	Both	Educ.	Wealth	Both
Low	1		1	1		1
	(.)		(.)	(.)		(.)
Medium	0.724***		0.758***	0.597***		0.685***
	(-5.62)		(-4.75)	(-9.12)		(-6.56)
High	0.590***		0.647***	0.544***		0.702***
	(-7.96)		(-6.40)	(-11.31)		(-6.19)
Household wealth percentile		0.993***	0.995***		0.987***	0.988***
		(-8.21)	(-6.28)		(-15.60)	(-12.84)
Observations	56880	57372	56709	47953	47953	47953

Notes: Cox models for the onset of dependency with the full covariate set of Table 3 and country fixed effects (SHARE only); t-statistics in parentheses. Education enters as the three-level classification recorded in each survey, with the least educated group as the reference. The sample is restricted to respondents with valid education data. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

5.6 Recovery and sample selection

Recovery. The baseline analysis treats dependency as an absorbing state, yet Table 10 shows that around a third of respondents ever observed dependent subsequently recover autonomy in both surveys. The question is then to see if allowing for recovery from dependency can affect our main findings. Actually this does not affect the analysis of first onset but it may overstate expected years dependent by counting periods of recovered autonomy as dependent time. We therefore recalculate this outcome from the share of the baseline cohort observed dependent at each wave. We allow repeated transitions between autonomy and dependency and count deceased individuals as not dependent. The resulting estimates remain close to the baseline values. The gap between the bottom and top wealth terciles is virtually unchanged in SHARE and falls by about a sixth in the HRS. The gap remains roughly one and a half times larger in the United States than in Europe. Thus, both the wealth gradient and the transatlantic contrast persist when recovery is allowed. We retain the baseline estimates in the main text, where bootstrap confidence intervals are available, and report this exercise as evidence that the absorbing-state assumption does not drive our conclusions.

Table 10: Recovery from dependency and its consequences for expected years dependent

	SHARE	HRS
Status at the following wave, among waves observed dependent		
Returned to autonomy	30.7%	30.4%
Still dependent	34.2%	40.9%
Died	35.2%	28.7%
Transitions observed	2,673	2,530
Individuals ever observed dependent		
Ever observed to return to autonomy	32.6%	33.3%
Individuals	2,476	2,243
Expected years dependent over eight years, by wealth tercile		
Bottom tercile, dependency absorbing	0.45	0.65
Bottom tercile, allowing recovery	0.443	0.609
Middle tercile, dependency absorbing	0.29	0.38
Middle tercile, allowing recovery	0.292	0.392
Top tercile, dependency absorbing	0.23	0.24
Top tercile, allowing recovery	0.221	0.263
Bottom minus top, allowing recovery	0.222	0.346

Notes: The upper panels use every pair of consecutive waves in which the respondent is observed dependent at the first of the two. The lower panel compares expected years dependent over eight years computed under the absorbing assumption, as in Table 6, with the same quantity computed as the integral of the share of the baseline cohort observed dependent at each wave, which places no restriction on transitions back to autonomy. Respondents who have died count as not dependent in both calculations.

Health deterioration before baseline. One concern is that if health was already declining when wealth was measured, it depletes assets and so drives the gradient. Because the cohort is defined to start at its first observed wave, there is no earlier wave from which to construct a pre-baseline health change. To address the concern, one can exclude the respondents for whom it is most acute. Table 11 reports two such exclusions. First, we see that dropping everyone who becomes dependent by the second wave, that is within roughly two years of the wealth measurement, removes 1,575 individuals and around 40% of the events. Second, dropping everyone reporting poor self-rated health at baseline removes 11,317 individuals. Neither materially changes the gradient. Under 2SRI the hazard ratio per ten percentiles

moves from 0.823 to 0.831 and then 0.811 in SHARE, and from 0.797 to 0.824 and then 0.830 in the HRS. The estimates lose precision because of the smaller samples but the point estimates are stable.

Table 11: Excluding respondents whose health may already have been deteriorating at baseline

	SHARE		HRS	
	Cox	2SRI	Cox	2SRI
Full sample	0.933***	0.823***	0.872***	0.797***
	(-8.37)	(-3.17)	(-15.62)	(-5.12)
Events	2,067	2,067	2,057	2,057
Excluding onset by wave 2	0.945***	0.831**	0.879***	0.824***
	(-5.36)	(-2.48)	(-12.38)	(-3.79)
Events	1,261	1,261	1,409	1,409
Excluding poor self-rated health at baseline	0.925***	0.811*	0.886***	0.830***
	(-5.23)	(-1.90)	(-10.46)	(-3.06)
Events	633	633	1,117	1,117

Notes: Hazard ratios on the baseline wealth percentile, expressed per ten percentiles, from the onset specification of Table 3; z-statistics in parentheses. The second panel excludes individuals first observed dependent at wave 2, and the third excludes individuals reporting poor self-rated health at their first observed wave. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

6 Discussion and conclusion

Using harmonised longitudinal data from SHARE and the HRS, we have separated the two margins along which wealth shapes dependency in old age, and the two behave very differently. Wealth delays the loss of autonomy. A ten percentile rise in the within-country wealth distribution lowers the hazard of becoming dependent by about 10% in a conventional Cox specification and by about 17% once the endogeneity of wealth is addressed with a control function. It does not prolong survival once dependency has begun, and in the United States the conditional association even reverses sign. We show that the reversal is the expected consequence of conditioning on an event that wealth itself causes, not evidence that wealth harms the dependent, and the heterogeneity results support that reading, since the reversal appears among American women, for whom wealth strongly delays onset, and is absent among American men, for whom it does not. Combining the two margins on the baseline population, the conclusion is unambiguous. Wealth lowers the cumulative incidence of dependency and raises the expected number of

years lived autonomously.

The asymmetry points to a compression rather than a translation of the dependent period. Wealth pushes onset later without extending the time spent dependent, so the dependent phase occupies a smaller share of the lives of the wealthy. Two further features of the data compound the disadvantage at the bottom. Those in the bottom tercile lose autonomy earlier and spend more of their remaining life dependent, so they need care for longer. And they are more often single and childless (Table 1), which is precisely the group in which our heterogeneity results say wealth matters most for onset, so the informal care that might substitute for purchased care is least available exactly where purchasing power is lowest. We observe wealth, transitions and household composition, not care arrangements or expenditure, so we can document that conjunction but not measure how the resulting need is financed or by whom. How much of it falls on public schemes and how much on families is a question our data cannot answer and one that the institutional contrast below makes pressing.

Two methodological points generalise beyond this application. In any study of sequential health transitions, mortality is a competing risk rather than independent censoring, and the resulting bias is systematic in that it understates gradients for groups with high mortality. And any analysis conditional on an intermediate health event is exposed to index-event selection whose sign is predictable from the first transition. Both have the same remedy, which is to report quantities defined on the baseline population, and both are, we suspect, more common in the health inequality literature than is generally acknowledged.

The transatlantic contrast is sharper in expected years than in hazard ratios, and that is where we rest it. The onset gradients are close, at 0.84 per ten percentiles in the HRS against 0.86 in SHARE, but the bottom to top gap in years lived dependent over eight years is 0.41 in the United States against 0.22 in Europe, and the gap in the share of remaining life spent dependent is 5.8 percentage points against 3.5. Holding age, gender, marital status, fertility and baseline health constant, the transatlantic ratio is closer to three than to two. Wealth is not associated with a markedly different average burden of dependency on the two sides of the Atlantic, but it sorts people into that burden far more steeply in the United States. The mechanism is straightforward. Where health care is universal and long-term care is publicly financed, private resources have less scope to determine who receives the preventive care, the home adaptations and the early support that delay decline. Where care must be purchased until assets are exhausted, they determine a great deal.

We state this carefully for two reasons. The comparison is not experimental, and Europe and the United States differ in much besides long-term care financing, including inequality itself, the composition of wealth portfolios and the age structure of the sample. And the within-Europe regional ordering

does not track long-term care generosity, which a clean institutional story would predict. A convincing test would exploit within-country variation in generosity over time, such as the German long-term care insurance reform, the French APA reforms, or state-level variation in Medicaid home and community based services waivers. We flag that as the natural next paper rather than claiming it here.

Three policy implications follow. Because the gradient operates through onset rather than duration, policies that delay functional decline carry larger distributional returns than policies that improve care once dependency has begun, and the fact that the gradient is steepest among people in good baseline health points the same way. Prevention aimed at the not-yet-frail poor, through fall prevention, housing adaptation, hearing and vision care and the management of chronic conditions, is where the equalising potential lies. Next, wealth matters most for those without a partner, and the share of older adults living alone is rising everywhere in our sample, so the wealth gradient in dependency will steepen mechanically unless public provision expands to substitute for the informal care that is disappearing. Finally, because the gradient is concentrated on onset, the fiscal cost of low socioeconomic status is incurred through longer eligibility spells rather than more intensive care per year. Means-tested schemes that key eligibility to assets are therefore doubly regressive here, since they withdraw support from the group whose earlier onset makes it most needed, and they do so at the point in the life cycle when asset depletion is irreversible.

Four limitations deserve emphasis. Identification rests on a single binary instrument, and while the exclusion restriction is more defensible for transfers from outside the close family than for inheritances in general, it is not testable, so the 2SRI estimates are better read as an upper bound on the causal effect. The observation window is five waves, with a median follow-up of 6.0 years in SHARE and 7.6 in the HRS, which is short relative to the process we study and makes the restricted mean quantities horizon-dependent. Self-reported ADL limitations may be reported differently across socioeconomic groups, though the stability of the gradient across severity thresholds argues against this driving the results. And the excluded population, those already dependent when first observed, is disproportionately poor, so the population-level gradient exceeds what we estimate.

Inequality in old age is not only about how long people live but about the condition in which those years are lived, and the disadvantage compounds. Poorer older adults lose their autonomy earlier, spend a larger share of their remaining life dependent, and less often have a partner or a child at hand. As the baby-boom cohorts reach the ages at which dependency becomes common, the design of long-term care systems will decide whether this gradient widens or narrows. Our results suggest the largest returns lie upstream, in delaying the onset of dependency among those who cannot buy that delay themselves.

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Appendix

A Covariate-adjusted expected years in state

Table A.1: Expected years autonomous and dependent over eight years, by baseline age band and wealth tercile

		SHARE			HRS		
Age at baseline	Tercile	Autonomous	Dependent	Alive	Autonomous	Dependent	Alive
50–64	Bottom	7.20	0.25	7.44	6.94	0.63	7.57
	Middle	7.48	0.14	7.62	7.48	0.29	7.77
	Top	7.56	0.13	7.68	7.70	0.12	7.82
	Bottom minus top	−0.36	*0.12*		−0.76	*0.51*	
65–74	Bottom	6.62	0.43	7.05	6.20	0.61	6.81
	Middle	6.90	0.27	7.18	6.90	0.32	7.21
	Top	7.19	0.20	7.39	7.32	0.19	7.51
	Bottom minus top	−0.57	*0.23*		−1.12	*0.42*	
75 and over	Bottom	4.92	0.72	5.64	4.77	0.83	5.60
	Middle	5.42	0.58	5.99	5.27	0.55	5.81
	Top	5.46	0.58	6.04	5.86	0.46	6.32
	Bottom minus top	−0.54	*0.14*		−1.09	*0.37*	
All ages	Bottom	6.33	0.45	6.78	6.44	0.65	7.09
	Middle	6.83	0.28	7.11	6.71	0.37	7.08
	Top	7.09	0.23	7.32	7.06	0.24	7.30
	Bottom minus top	−0.76	*0.22*		−0.62	*0.41*	

Notes: Restricted mean times in state over $\tau = 8$ years from baseline, computed exactly as in Table 6 but separately within bands of age at the baseline interview. "Alive" is expected years lived and "Dependent" is the difference between expected years lived and expected years autonomous. The "All ages" panel reproduces Table 6.

Table A.2: Expected years autonomous and dependent by wealth percentile, adjusted for covariates

Wealth percentile	10	30	50	70	90
Panel A. SHARE (Europe)					
Years autonomous	6.568	6.693	6.809	6.917	7.017
Years dependent	0.380	0.341	0.305	0.271	0.240
Share dependent (%)	5.466	4.847	4.281	3.768	3.303
Panel B. HRS (United States)					
Years autonomous	6.388	6.614	6.816	6.994	7.150
Years dependent	0.620	0.496	0.388	0.295	0.216
Share dependent (%)	8.845	6.974	5.380	4.042	2.935

Notes: Restricted mean times over $\tau = 8$ years obtained by standardisation (G-formula). Cox models for exit from autonomy and for overall survival are estimated with the full covariate set of Table 3, the wealth percentile is then set counterfactually to the value in the column heading for every individual, and the resulting survival curves are averaged over the observed distribution of the remaining covariates before integration.

B First-stage and instrument diagnostics

	SHARE				HRS			
	Coef.	(S.E.)	First-stage *F*	*N*	Coef.	(S.E.)	First-stage *F*	*N*
Panel A. Main estimation samples								
Pooled (SHARE + HRS)	*Coef.=13.862*		*S.E.=0.398*		*First-stage F =1216*		*N =105,325*	
SHARE only	11.655	(0.546)	455.9	57,372				
HRS only					15.502	(0.564)	755.3	47,953
Panel B. Subsamples used in the heterogeneity analysis								
Women	12.174	(0.728)	279.7	32,457	16.059	(0.756)	451.6	27,980
Men	10.968	(0.824)	177.3	24,915	14.788	(0.845)	305.9	19,973
Single	13.562	(1.043)	169.0	19,218	18.928	(1.156)	268.2	16,785
Partnered	10.783	(0.634)	288.9	38,154	14.222	(0.625)	518.1	31,168
Aged under 80	11.609	(0.574)	409.5	49,664	15.640	(0.616)	644.3	41,009
Aged 80+	11.590	(1.468)	62.3	7,708	14.127	(1.168)	146.2	6,944
Good mental health	11.189	(0.589)	360.6	43,292	14.915	(0.582)	657.8	39,790
Poor mental health	13.434	(0.954)	198.2	14,080	18.845	(1.309)	207.3	8,163
Good physical health	11.112	(0.665)	279.6	29,774	15.517	(0.862)	324.1	18,840
Poor physical health	12.265	(0.737)	277.0	27,598	15.437	(0.708)	475.2	29,113
Panel C. European regions								
North	9.346	(1.228)	57.9	7,482				
Centre	12.494	(0.741)	284.4	23,780				
South	10.876	(1.872)	33.8	8,395				
East	11.709	(1.300)	81.1	17,715				

C Additional descriptive evidence

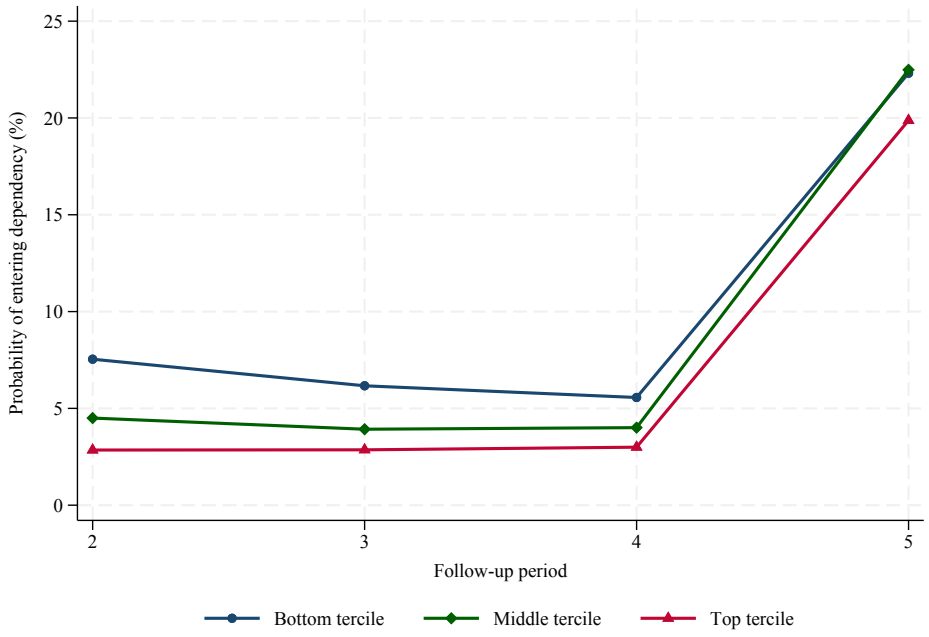

Figure A.1: Dependency incidence by wealth tercile and follow-up period

Notes: Transition into dependency between wave t and wave $t+1$ among respondents non-dependent at t and alive at $t+1$.

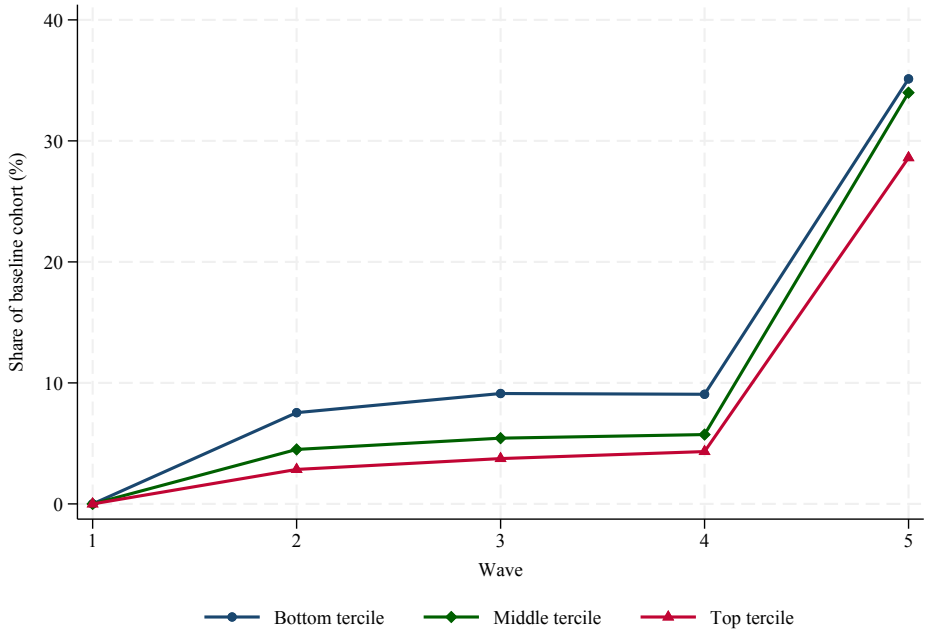

Figure A.2: Dependency prevalence by wealth tercile and wave

Notes: Share of the baseline cohort alive and dependent at each wave. Deceased respondents remain in the denominator.

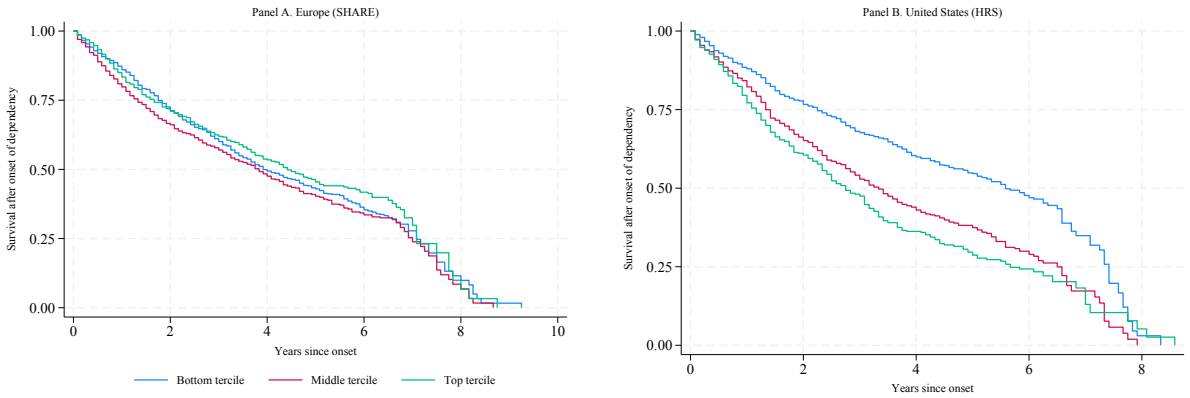

Figure A.3: Kaplan–Meier survival following first observed dependency, by wealth tercile

D Additional robustness tables

This appendix collects the robustness tables discussed in Section 5. Table A.3 and Table A.4 vary the measure of wealth, Table A.5 tests the proportional hazards assumption, Table A.6 varies the definition of dependency and Table A.7 varies the treatment of the interval-censored onset date.

Table A.3: Hazard of becoming dependent under alternative wealth measures (2SRI)

	Log net worth		Wealth quintiles		Financial wealth		Equivalised wealth	
	SHARE	HRS	SHARE	HRS	SHARE	HRS	SHARE	HRS
Wealth measure	0.889	0.789***	0.836	0.627***	1.000	1.000***	1.000	1.000***
	(-0.71)	(-4.33)	(-1.19)	(-3.56)	(-1.31)	(-3.55)	(-1.21)	(-3.84)
First-stage residual	1.058	1.174***	1.037	1.238	1.000	1.000***	1.000	1.000***
	(0.34)	(2.88)	(0.23)	(1.51)	(0.82)	(3.13)	(0.51)	(2.75)
Person-intervals	57,815	45,621	58,949	49,460	58,949	49,460	58,949	49,460

Notes: Each column is a separate 2SRI Cox model with the full covariate set of Table 3, reported here in abbreviated form. Financial and equivalised wealth enter in levels, so hazard ratios are reported to three decimals and are close to one by construction; the t-statistics are the informative quantity. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table A.4: Hazard of death after dependency under alternative wealth measures (2SRI)

	Log net worth		Wealth quintiles		Financial wealth		Equivalised wealth	
	SHARE	HRS	SHARE	HRS	SHARE	HRS	SHARE	HRS
Wealth measure	1.035	1.162**	1.052	1.365**	1.000	1.000***	1.000	1.000**
	(0.29)	(2.31)	(0.26)	(2.42)	(0.25)	(2.60)	(0.27)	(2.33)
First-stage residual	0.965	0.878*	0.927	0.756**	1.000	1.000**	1.000	1.000**
	(-0.31)	(-1.92)	(-0.39)	(-2.26)	(-0.26)	(-2.45)	(-0.28)	(-2.18)
Person-intervals	2,165	2,407	2,222	2,785	2,222	2,785	2,222	2,785

Notes: See Table A.3. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table A.5: Tests of the proportional hazards assumption (scaled Schoenfeld residuals)

	Onset of dependency				Death after dependency			
	SHARE		HRS		SHARE		HRS	
	χ^2	p	χ^2	p	χ^2	p	χ^2	p
Household wealth percentile	0.53	0.466	2.56	0.110	0.03	0.859	0.00	0.985
Female	0.94	0.331	1.04	0.309	0.17	0.679	0.94	0.331
Age	0.05	0.822	1.35	0.245	0.02	0.890	1.95	0.163
Age squared	0.22	0.636	0.35	0.555	0.20	0.658	1.95	0.163
Single	0.05	0.824	0.11	0.738	0.00	0.963	0.08	0.772
At least one child	2.40	0.122	0.15	0.696	1.13	0.288	0.85	0.358
Poor mental health	9.14	0.002	3.28	0.070	0.09	0.762	3.69	0.055
Poor physical health	2.49	0.115	9.87	0.002	3.35	0.067	5.52	0.019

Notes: Tests of a zero slope of the scaled Schoenfeld residuals on log time, computed on the baseline cohort with covariates measured at entry. A small p-value indicates a time-varying effect and hence a violation of proportionality for that covariate.

Table A.6: Hazard of onset under alternative definitions of dependency

	1+ ADL	2+ ADL (baseline)	3+ ADL	1+ ADL or IADL
SHARE				
Wealth percentile	0.9938***	0.9927***	0.9923***	0.9922***
	(-9.27)	(-8.37)	(-6.68)	(-12.60)
Events	3,076	1,857	1,068	3,581
Person-intervals	51,584	57,372	58,493	49,293
HRS				
Wealth percentile	0.9890***	0.9868***	0.9869***	0.9889***
	(-14.24)	(-13.96)	(-10.58)	(-14.81)
Events	2,561	1,745	1,022	2,681
Person-intervals	41,793	47,953	49,329	40,836

Notes: Hazard ratios on the baseline wealth percentile from Cox models with the full covariate set of Table 3 and country fixed effects (SHARE only); z-statistics in parentheses. Each column redefines the event, and hence both the risk set and the number of events. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table A.7: Alternative treatments of the interval-censored onset date

	SHARE		HRS	
	Cox	2SRI	Cox	2SRI
Interview date (baseline)	0.933***	0.823***	0.872***	0.797***
	(-8.37)	(-3.17)	(-15.62)	(-5.12)
Midpoint imputation	0.940***	0.877**	0.871***	0.812***
	(-7.46)	(-2.14)	(-15.80)	(-4.70)
Discrete-time cloglog	0.936***		0.870***	
	(-7.65)		(-14.74)	
Events	2,067	2,067	2,057	2,057

Notes: Hazard ratios on the baseline wealth percentile expressed per ten percentiles, with z-statistics in parentheses. The first two rows use the onset specification of Table 3, dating onset at the interview at which dependency is first reported and at the midpoint between that interview and the preceding one. The third row reports a discrete-time complementary log-log model of the probability of becoming dependent between consecutive waves, with wave dummies absorbing the baseline hazard, estimated on person-wave observations at which the respondent is autonomous and observed again. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

E Full heterogeneity tables

These tables report the full coefficient vector behind Figures 3 and 4. Every column is a separate 2SRI Cox model estimated on the indicated subsample, with hazard ratios and t statistics from standard errors clustered on the individual. When the sample is split on a covariate, that covariate is dropped from both stages and therefore does not appear in the table. Country fixed effects are included in all SHARE columns and in none of the HRS columns. "First-stage residual" is the control function, whose coefficient is the test of exogeneity of the wealth percentile.

Onset of dependency

Table A.8: Onset of dependency by region

	North	Centre	South	East	United States
Household wealth percentile	0.989	0.983**	0.975	0.996	0.982***
	(-0.59)	(-2.07)	(-1.38)	(-0.29)	(-3.94)
Female	1.066	0.737***	1.080	0.763***	0.939
	(0.38)	(-3.78)	(0.73)	(-3.43)	(-1.33)
Age	1.034	0.921*	0.983	0.964	0.877***
	(0.24)	(-1.65)	(-0.22)	(-0.62)	(-6.14)
Age squared	1.000	1.001***	1.001	1.001	1.001***
	(0.35)	(3.09)	(1.27)	(1.52)	(8.89)
Single	0.816	0.949	0.770	1.030	0.943
	(-0.56)	(-0.36)	(-1.13)	(0.15)	(-0.66)
At least one child	0.733	1.064	1.000	0.852	0.874
	(-1.25)	(0.55)	(-0.00)	(-1.25)	(-1.51)
Poor mental health	1.741***	2.037***	2.113***	2.311***	2.118***
	(3.19)	(7.83)	(6.30)	(9.13)	(12.88)
Poor physical health	1.871***	1.530***	1.260**	1.679***	2.093***
	(2.93)	(4.47)	(2.00)	(5.65)	(10.67)
First-stage residual	1.003	1.008	1.019	0.999	1.005
	(0.16)	(0.98)	(1.04)	(-0.06)	(0.99)
Country fixed effects	Yes	Yes	Yes	Yes	No
Person-intervals	7482	23780	8395	17715	47953

Table A.9: Onset of dependency by gender

	SHARE Women	SHARE Men	HRS Women	HRS Men
Household wealth percentile	0.978***	0.994	0.973***	0.994
	(-2.69)	(-0.64)	(-4.41)	(-0.85)
Age	0.975	0.916	0.918***	0.816***
	(-0.58)	(-1.64)	(-3.14)	(-5.68)
Age squared	1.001**	1.001***	1.001***	1.002***
	(2.13)	(2.82)	(5.51)	(7.06)
Single	0.810	0.997	0.816	1.057
	(-1.53)	(-0.02)	(-1.63)	(0.45)
At least one child	0.886	1.051	0.760**	1.021
	(-1.29)	(0.41)	(-2.35)	(0.15)
Poor mental health	2.020***	2.266***	1.863***	2.580***
	(10.36)	(9.92)	(8.42)	(9.97)
Poor physical health	1.466***	1.636***	1.992***	2.142***
	(4.88)	(6.12)	(7.14)	(7.64)
First-stage residual	1.016*	0.999	1.013**	0.994
	(1.90)	(-0.11)	(2.11)	(-0.94)
Country fixed effects	Yes	Yes	No	No
Person-intervals	32457	24915	27980	19973

Table A.10: Onset of dependency by marital status

	SHARE Single	SHARE Partnered	HRS Single	HRS Partnered
Household wealth percentile	0.985*	0.988	0.978***	0.988**
	(-1.74)	(-1.37)	(-3.56)	(-2.06)
Female	0.881	0.811***	0.976	0.901
	(-1.59)	(-3.39)	(-0.33)	(-1.59)
Age	0.926*	0.935	0.906***	0.829***
	(-1.67)	(-1.36)	(-2.99)	(-6.38)
Age squared	1.001***	1.001***	1.001***	1.002***
	(3.11)	(2.71)	(4.74)	(8.33)
At least one child	0.904	1.236	0.897	0.839
	(-1.20)	(1.38)	(-1.04)	(-1.03)
Poor mental health	1.933***	2.323***	1.759***	2.662***
	(8.75)	(11.78)	(7.74)	(11.11)
Poor physical health	1.460***	1.639***	1.944***	2.191***
	(4.55)	(6.53)	(6.71)	(8.30)
First-stage residual	1.010	1.004	1.010*	0.998
	(1.11)	(0.48)	(1.65)	(-0.29)
Country fixed effects	Yes	Yes	No	No
Person-intervals	19218	38154	16785	31168

Table A.11: Onset of dependency by age group

	SHARE Under 80	SHARE 80 and over	HRS Under 80	HRS 80 and over
Household wealth percentile	0.988	0.984	0.982***	0.984**
	(-1.59)	(-1.43)	(-3.33)	(-1.97)
Female	0.741***	1.006	0.925	0.975
	(-5.04)	(0.07)	(-1.37)	(-0.29)
Age	0.776***	1.992**	0.835***	2.716***
	(-4.58)	(2.10)	(-5.70)	(3.46)
Age squared	1.002***	0.996*	1.002***	0.995***
	(5.53)	(-1.89)	(6.65)	(-3.23)
Single	0.970	0.809	0.999	0.836
	(-0.24)	(-1.32)	(-0.01)	(-1.25)
At least one child	0.972	0.944	0.904	0.847
	(-0.28)	(-0.57)	(-0.96)	(-1.00)
Poor mental health	2.395***	1.778***	2.431***	1.500***
	(12.92)	(7.71)	(12.55)	(4.05)
Poor physical health	1.737***	1.286***	2.343***	1.366***
	(7.56)	(3.26)	(10.26)	(2.74)
First-stage residual	1.005	1.011	1.003	1.008
	(0.58)	(1.04)	(0.47)	(0.97)
Country fixed effects	Yes	Yes	No	No
Person-intervals	49664	7708	41009	6944

Table A.12: Onset of dependency by baseline mental health

	SHARE Good MH	SHARE Poor MH	HRS Good MH	HRS Poor MH
Household wealth percentile	0.986	0.984**	0.984***	0.979***
	(-1.63)	(-2.00)	(-2.91)	(-2.88)
Female	0.820***	0.820***	0.970	0.851**
	(-3.00)	(-2.91)	(-0.51)	(-2.09)
Age	0.907**	0.996	0.885***	0.895***
	(-2.21)	(-0.09)	(-4.18)	(-3.87)
Age squared	1.001***	1.000	1.001***	1.001***
	(3.85)	(1.36)	(6.46)	(5.27)
Single	0.958	0.845	1.013	0.835
	(-0.31)	(-1.30)	(0.12)	(-1.44)
At least one child	1.011	0.932	0.754**	1.174
	(0.11)	(-0.71)	(-2.53)	(1.10)
Poor physical health	1.530***	1.553***	2.016***	2.101***
	(5.78)	(5.17)	(8.26)	(6.45)
First-stage residual	1.006	1.012	1.002	1.010
	(0.65)	(1.42)	(0.29)	(1.32)
Country fixed effects	Yes	Yes	No	No
Person-intervals	43292	14080	39790	8163

Table A.13: Onset of dependency by baseline physical health

	SHARE Good PH	SHARE Poor PH	HRS Good PH	HRS Poor PH
Household wealth percentile	0.969**	0.992	0.960***	0.987***
	(-2.57)	(-1.13)	(-3.11)	(-2.82)
Female	0.837**	0.830***	0.943	0.951
	(-2.04)	(-3.27)	(-0.48)	(-0.97)
Age	1.020	0.901**	0.921	0.858***
	(0.35)	(-2.52)	(-1.58)	(-6.19)
Age squared	1.000	1.001***	1.001***	1.001***
	(0.82)	(4.08)	(3.28)	(8.37)
Single	0.776	0.968	0.625*	1.008
	(-1.38)	(-0.29)	(-1.82)	(0.09)
At least one child	0.940	0.992	0.693*	0.925
	(-0.47)	(-0.09)	(-1.75)	(-0.78)
Poor mental health	1.936***	2.199***	1.917***	2.128***
	(6.56)	(13.04)	(3.65)	(12.52)
First-stage residual	1.023*	1.002	1.026*	1.001
	(1.87)	(0.29)	(1.93)	(0.19)
Country fixed effects	Yes	Yes	No	No
Person-intervals	29774	27598	18840	29113

Death after dependency

Table A.14: Death after dependency by region

	North	Centre	South	East	United States
Household wealth percentile	1.017	1.009	0.899	0.972	1.015***
	(0.49)	(0.81)	(-0.34)	(-1.03)	(2.80)
Female	0.336***	0.602***	0.381	0.553***	0.724***
	(-3.27)	(-3.14)	(-0.82)	(-3.06)	(-3.84)
Age	1.468*	1.444***	0.860	0.881	1.084
	(1.65)	(3.12)	(-0.83)	(-1.34)	(1.49)
Age squared	0.998	0.998***	1.001	1.001*	1.000
	(-1.28)	(-2.65)	(0.88)	(1.72)	(-0.79)
Single	2.298*	1.267	0.402	0.795	1.303**
	(1.75)	(1.05)	(-0.33)	(-0.66)	(2.28)
At least one child	0.653	1.062	1.010	0.979	0.840
	(-0.99)	(0.28)	(0.02)	(-0.10)	(-1.25)
Poor mental health	1.032	1.408**	1.064	1.276	1.182**
	(0.11)	(2.31)	(0.23)	(1.33)	(2.04)
Poor physical health	0.908	1.216	1.013	0.821	1.675***
	(-0.25)	(1.00)	(0.02)	(-1.06)	(2.93)
First-stage residual	0.985	0.990	1.111	1.028	0.987**
	(-0.43)	(-0.89)	(0.34)	(1.02)	(-2.36)
Country fixed effects	Yes	Yes	Yes	Yes	No
Person-intervals	189	725	474	834	2785

Table A.15: Death after dependency by gender

	SHARE Women	SHARE Men	HRS Women	HRS Men
Household wealth percentile	1.013	0.995	1.020***	1.007
	(0.74)	(-0.38)	(2.76)	(0.96)
Age	1.163	1.100	1.084	1.070
	(1.62)	(1.14)	(1.31)	(0.67)
Age squared	0.999	1.000	1.000	1.000
	(-0.95)	(-0.60)	(-0.87)	(-0.16)
Single	1.163	1.062	1.481**	1.141
	(0.62)	(0.35)	(2.22)	(0.92)
At least one child	1.083	0.935	0.832	0.938
	(0.45)	(-0.31)	(-0.94)	(-0.29)
Poor mental health	1.212	1.387***	1.002	1.401***
	(1.39)	(2.77)	(0.02)	(2.79)
Poor physical health	1.057	1.128	1.449*	1.996**
	(0.36)	(0.67)	(1.68)	(2.28)
First-stage residual	0.989	1.003	0.982**	0.994
	(-0.64)	(0.21)	(-2.40)	(-0.78)
Country fixed effects	Yes	Yes	No	No
Person-intervals	1332	890	1674	1111

Table A.16: Death after dependency by marital status

	SHARE Single	SHARE Partnered	HRS Single	HRS Partnered
Household wealth percentile	0.993	1.007	1.018**	1.014*
	(-0.51)	(0.32)	(2.37)	(1.86)
Female	0.557***	0.553***	0.746***	0.748**
	(-4.05)	(-4.12)	(-2.59)	(-2.21)
Age	1.114	1.099	1.055	1.021
	(1.28)	(0.97)	(0.86)	(0.20)
Age squared	1.000	1.000	1.000	1.000
	(-0.66)	(-0.47)	(-0.43)	(0.31)
At least one child	1.257	0.444***	0.833	0.938
	(1.45)	(-2.94)	(-1.19)	(-0.22)
Poor mental health	1.010	1.630***	1.016	1.461***
	(0.09)	(3.14)	(0.15)	(2.84)
Poor physical health	1.134	1.051	1.772**	1.405
	(0.83)	(0.21)	(2.44)	(1.34)
First-stage residual	1.008	0.992	0.984**	0.986*
	(0.56)	(-0.40)	(-2.01)	(-1.69)
Country fixed effects	Yes	Yes	No	No
Person-intervals	1099	1123	1450	1335

Table A.17: Death after dependency by age group

	SHARE Under 80	SHARE 80 and over	HRS Under 80	HRS 80 and over
Household wealth percentile	1.003	1.000	1.018**	1.016*
	(0.16)	(0.03)	(2.33)	(1.87)
Female	0.440***	0.695***	0.802*	0.716***
	(-5.53)	(-2.58)	(-1.77)	(-2.77)
Age	1.620*	1.508	1.042	1.448
	(1.90)	(1.20)	(0.21)	(1.00)
Age squared	0.997*	0.998	1.000	0.998
	(-1.70)	(-1.03)	(-0.03)	(-0.87)
Single	1.234	1.012	1.700***	1.105
	(0.64)	(0.07)	(3.11)	(0.59)
At least one child	0.884	1.046	0.907	0.864
	(-0.55)	(0.29)	(-0.43)	(-0.74)
Poor mental health	1.584***	1.129	1.346**	1.084
	(2.64)	(1.16)	(2.32)	(0.78)
Poor physical health	0.919	1.093	1.889**	1.417
	(-0.38)	(0.66)	(2.18)	(1.46)
First-stage residual	0.994	1.000	0.983**	0.985
	(-0.27)	(-0.03)	(-2.02)	(-1.63)
Country fixed effects	Yes	Yes	No	No
Person-intervals	1238	984	1940	845

Table A.18: Death after dependency by baseline mental health

	SHARE Good MH	SHARE Poor MH	HRS Good MH	HRS Poor MH
Household wealth percentile	0.992	1.012	1.021***	1.007
	(-0.58)	(0.69)	(3.33)	(0.76)
Female	0.562***	0.623***	0.861	0.615***
	(-3.56)	(-3.33)	(-1.20)	(-4.38)
Age	1.223*	0.985	1.229***	1.046
	(1.75)	(-0.20)	(2.79)	(0.61)
Age squared	0.999	1.000	0.999**	1.000
	(-1.24)	(0.84)	(-2.25)	(-0.06)
Single	1.502	1.057	1.549**	1.094
	(1.60)	(0.28)	(2.54)	(0.59)
At least one child	1.512*	0.888	0.753	0.932
	(1.68)	(-0.75)	(-1.58)	(-0.31)
Poor physical health	1.053	1.093	1.877***	1.428
	(0.27)	(0.56)	(2.72)	(1.44)
First-stage residual	1.006	0.988	0.978***	0.998
	(0.43)	(-0.69)	(-3.21)	(-0.22)
Country fixed effects	Yes	Yes	No	No
Person-intervals	847	1375	1375	1410

Table A.19: Death after dependency by baseline physical health

	SHARE Good PH	SHARE Poor PH	HRS Good PH	HRS Poor PH
Household wealth percentile	1.012	0.999	1.014	1.015***
	(0.60)	(-0.07)	(0.23)	(2.84)
Female	0.646*	0.564***	0.907	0.707***
	(-1.81)	(-5.37)	(-0.19)	(-4.04)
Age	1.133	1.056	1.573**	1.049
	(0.86)	(0.83)	(2.28)	(0.87)
Age squared	1.000	1.000	0.997*	1.000
	(-0.48)	(-0.05)	(-1.71)	(-0.21)
Single	1.342	1.113	0.999	1.320**
	(0.60)	(0.65)	(-0.00)	(2.38)
At least one child	0.842	1.000	1.013	0.823
	(-0.48)	(-0.00)	(0.02)	(-1.35)
Poor mental health	1.319	1.272**	1.571	1.147*
	(1.32)	(2.35)	(0.71)	(1.65)
First-stage residual	0.982	1.001	0.977	0.988**
	(-0.91)	(0.10)	(-0.37)	(-2.24)
Country fixed effects	Yes	Yes	No	No
Person-intervals	397	1825	310	2475