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The welfare cost of late-life depression

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ABSTRACT

We quantify the welfare cost of depression among older Americans by estimating a panel VAR model of mental and physical health, labor supply, and consumption using data from the Health and Retirement Study. We use the estimated model and age sixty joint distribution of outcomes to simulate life-cycle paths with and without prevalence of depressive symptoms after age sixty. We estimate that the prevalence of late-life depressive symptoms costs an average of between 0.85 and 2.1 years in quality-adjusted life expectancy per person. Moreover, depression may result in an average loss of labor supply of up to 1.1 months and lifetime consumption of up to $16,000. Combining into a single compensating variation welfare metric, we estimate a bound on the average welfare cost of depression of 8–15% of annual consumption after age sixty. On aggregate, this amounts to roughly $180–360 billion annually. We also project that while the average welfare cost of late-life depression is declining slightly over birth cohorts, the welfare burden is becoming significantly more unequal.

1. Introduction

Depression is one of the leading causes of emotional distress and lower quality of life among older adults (Blazer, 2003; Sivertsen et al., 2015). Depression and depressive symptoms are also highly correlated with other physical and psychiatric conditions in older populations (Moussavi et al., 2007; Vaughan et al., 2015; Soysal et al., 2017; Chu et al., 2019). Increasing depressive symptoms with age have also been shown to be predictive of an increased risk of mortality (Bruce et al., 1994; Chui et al., 2015). Nonetheless, under-treatment of depression remains prevalent in older populations despite the wide availability of effective treatments (Barry et al., 2012; Kok and Reynolds, 2017). Improving our understanding of the comprehensive costs of late-life depression may be a fruitful avenue for expanding uptake of effective antidepressants and treatment therapies.

While preventing and treating late-life depression is of major social importance in its own right, significant spillover benefits are also possible. Empirical studies have found depression to be related to increased risk of frailty, reduced mobility, functional limitations, and progression of chronic diseases (Stuck et al., 1999; Penninx et al., 1999; Ciechanowski et al., 2000; De Groot et al., 2001; Geerlings et al., 2001; Rubio-Guerra et al., 2013; Vaughan et al., 2015; Chiang et al., 2015; Soysal et al., 2017; Penninx, 2017; Lwin et al., 2020). This has led some researchers to hypothesize a causal link from depression to

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poor physical health in older adults. However, whether the association between depression and physical health is driven by reciprocal influences or common causes remains widely debated (Mayerl et al., 2020).

Many theoretical explanations for why depression would affect physical health have been proposed (Penninx et al., 1999; Bruce, 2001). It could be that depressive symptoms such as sleep disturbance or lost appetite have a direct effect on functional decline and disability. There could also be indirect effects through intermediate behaviors (Bruce, 2001). For example, depressive symptoms could reduce motivation and lead to reduced medical care or poor health behaviors (e.g., smoking, poor nutrition, reduced physical activity). Other proposed mechanisms include antidepressant use (Lakey et al., 2012), increased allostatic load (McEwen, 2003), or other neuronal, hormonal, and/or immunological alterations (Bruce, 2001).

Beyond health effects, late-life depression could also influence an individual's economic outcomes. For example, depression among older adults increases health service utilization and costs (Luppa et al., 2012). Standard consumer theory suggests this could have a negative contemporaneous effect on consumption expenditures. Consumption could also decrease with depression due to reduced productivity and earnings (Lerner and Henke, 2008) or even decreased utility from goods and services that are complements to good mental health. On the other hand, the life-cycle hypothesis suggests that an unexpected depressive episode could *increase* contemporaneous consumption if there is an associated decline in life expectancy. Moreover, there could be additional dynamic effects that persist over time, for example if depression leads to an early retirement (Doshi et al., 2008; Rice et al., 2011). In the presence of such dynamic effects, cross-sectional correlations between depression and other health and economic outcomes would only reveal part of the larger story.

In this paper, we adopt a life-cycle approach to better quantify the welfare cost of late-life depression when incorporating persistence and dynamic spillover effects. We extend the panel VAR model proposed by Miller and Bairoliya (2022) to simulate the joint evolution of health and economic outcomes, adapted to include the onset and persistence of late-life depression. We estimate the model using longitudinal data from the Health and Retirement Study (HRS) spanning more than twenty years. Using the observed joint distribution of outcomes at age sixty as initial conditions, we show that model simulations are able to closely match the empirically observed evolution of depressive symptoms, physical health, labor supply, and consumption.

Equipped with our simulation model, we next estimate the welfare cost of late-life depression. As the causal relationships between depressive symptoms and other health and economic outcomes remains unsettled, we take a bounds analysis approach. First, we estimate a lower bound on welfare costs by assuming there are no spillover effects on other model outcomes. More specifically, we leave all expected paths of comorbidities, mortality, and economic outcomes at their baseline levels and only remove the health utility penalty associated with depressive symptoms at age sixty-two and older. We follow this with an upper bound estimate calculated by running a second set of counterfactual simulations starting from the same initial conditions but removing any possibility of depressive symptoms after age sixty. We consider this an upper bound as it assumes all the statistical relationships estimated in the restricted VAR model are entirely causal.

These analyses provide bounds on the expected costs of late-life depression in terms of quality-adjusted life years (QALYs), labor years, and dollars of consumption. We combine these differing costs using standard expected utility theory by calculating an ex-ante compensating variation (CV) measure of welfare. The welfare concept is akin to asking how much an individual would be willing to pay at age sixty to avoid any possibility of depressive symptoms over their remaining life. As our measure integrates multiple health and economic outcomes, it gives a more comprehensive view of well-being loss than the direct utility cost of depression alone. As it incorporates individual expectations over the entirety of remaining life from age sixty, it also provides a useful single metric of the ex-ante welfare cost of late-life depression.

1.1. Contributions

This study makes several contributions to our understanding of the welfare or utility burden of depression in older adults. First, previous studies have focused on estimating lost quality of life in older populations using cross-sectional observation, clinical settings, and/or limited longitudinal data (Sivertsen et al., 2015). Our estimates capture both contemporaneous and dynamic spillover effects on the evolution of depression, health, and economic outcomes over the entirety of remaining life. This provides a more complete measure of the total welfare burden of depression as it incorporates the cumulative burden of disease over time. We also provide an estimate that combines the impact of depression on health-related quality-of-life, leisure, and consumption into a single measure grounded in economic and public health theory. Moreover, as our simulations are at the individual level within a larger representative sample, we are able to examine the entire distribution of welfare as opposed to only specific sub-samples or summary aggregates. This approach also allows us to examine how the level and distribution of welfare costs changed over birth cohorts, as opposed to cross-sectional changes over time.

Finally, we also contribute to the literature that has attempted to estimate the economic burden of depression to society. Studies have examined the impact of depression on direct medical costs and indirect workplace costs, including absenteeism from work and presenteeism while at work (Wang et al., 2003; Stewart et al., 2003; Lerner and Henke, 2008; Birnbaum et al., 2010; Luppa et al., 2012). Combined with suicide-related mortality costs, Greenberg et al. (2015) estimate the economic burden of major depression disorders in the U.S. was $210.5 billion in 2010. The total direct healthcare costs for treating depressive disorders among those aged 65 and older have been estimated at $9.8 billion in 2016. About $930 million out-of-pocket, $1.4 billion from private insurance, and the remaining $7.4 billion paid for by public insurance programs (Dieleman et al., 2020). While these costs center on the employer or healthcare side, we complement these studies

by incorporating economic costs to private individuals. We also focus on older adults and quantify effects from the full range of depressive symptoms as opposed to only major disorders.

2. Data and methods

2.1. Data

The HRS is an ongoing longitudinal survey of U.S. individuals over the age of fifty and their spouses. The survey began in 1992 and data has since been collected every two years with new birth cohorts added periodically. There are currently eight birth cohorts in the study—the early HRS cohort (born 1931–36), late HRS cohort (born 1937–41), AHEAD cohort (born before 1924), Children of Depression (born 1924–30), War Babies (born 1942–47), early Baby Boomers (born 1948–53), mid-Baby Boomers (born 1954–59), and late-Baby Boomers (born 1960–65). We use the publicly available RAND HRS Longitudinal File 2016 (V2) to obtain data on depression, health, mortality, and economic outcomes from 1992 to 2016. We also utilize other individual characteristics including age, education, gender, race, birth cohort, region, and occupation.

2.1.1. Depression

Depressive symptoms in the HRS were measured using the eight-item Center for Epidemiologic Studies Depression scale (CESD). The measure ranges from zero (no depressive symptoms) to eight, created by summing the respondent's number of "yes" answers across eight survey items (with positive items reverse-coded).[1] The CESD is a common measure of depressive symptoms in older adults (Lewinsohn et al., 1997; Turvey et al., 1999; Steffick, 2000; Karim et al., 2015). The CESD was designed to measure a continuum of psychological distress (symptoms of depression), rather than determining the presence or absence of specific psychiatric disorders. However, a longer form CESD scale has been broadly validated against diagnostic interviews for depression and other anxiety disorders (Fechner-Bates et al., 1994; Lewinsohn et al., 1997). The eight-item CESD has also been shown to be a valid and reliable instrument of depression in a large sample of older Europeans (Karim et al., 2015).

2.1.2. Additional health outcomes

In addition to depression, our model incorporates data on comorbidities. These include eight binary indicators for ever having been diagnosed by a doctor with the following health problems—(1) high blood pressure or hypertension; (2) diabetes or high blood sugar; (3) cancer or a malignant tumor of any kind except skin cancer; (4) chronic lung disease except asthma such as chronic bronchitis or emphysema; (5) heart attack, coronary heart disease, angina, congestive heart failure, or other heart problems; (6) stroke or transient ischemic attack (TIA); (7) emotional, nervous, or psychiatric problems; and (8) arthritis or rheumatism. We also include an indicator for ever reported difficulty with any activity of daily living (ADL) such as bathing, getting dressed, or walking across a room. ADL difficulties are a common health metric in older populations.

As a final health measure we use self-rated health status reported on a five-point scale from poor (one) to excellent (five). Self-rated health has been shown to be predictive of mortality in the HRS and other datasets, even after controlling for other health conditions, health behavior, and socioeconomic characteristics (Idler and Benyamini, 1997; Stenholm et al., 2014). This may reflect that people have private information about their health over and above disease diagnosis.

2.1.3. Economic outcomes

As our empirical focus is on individuals nearing the end of working life, we limit labor considerations to retirement. We treat retirement as an absorbing state in our model and define retired individuals as those reporting zero annual hours of paid work in the current or any previous survey wave.[2]

We use consumption data from the Consumption and Activities Mail Survey (CAMS), which was sent to a random subsample of HRS respondents in off years of the core survey. We use the RAND 2017 CAMS data file (V1), which contains a constructed estimate of total household consumption from 2001 to 2015 based on household spending on durables, nondurables, transportation, and housing. We create our measure of individual consumption by subtracting out-of-pocket health spending from household consumption and then dividing by the total number of household members.[3] As consumption data is only available between the core HRS waves, we merge each CAMS wave with the HRS core data from the previous wave.[4]

A challenge to our analysis is that CAMS data is only available for approximately 20% of HRS respondents for the years 2000–2014. We follow Miller and Bairoliya (2022) and use closely related available data such as wealth and income to

[1] About 12% of observations in our estimation sample were missing CESD score. These were imputed along with consumption and other missing data as detailed in the online appendix.

[2] We could also consider the intensive margin, partial retirement, and/or reentry into the workforce but this comes with additional model complexity. Moreover, retirement is likely to be the largest labor market decision for this age group, but we find relatively small effects of depression on retirement in our empirical analysis. See also the appendix for robustness results where we use full-time employment instead of zero hours for our definition of retirement.

[3] Health spending includes health insurance, medication, health services, and medial supplies. We use the CPI-U to convert all waves to 2010 dollars. Household members include all residents but exclude spouses/parents living in nursing homes. We do not adjust for lower consumption of any resident children given the small number of children in HRS households.

[4] This is the recommended procedure for use of the RAND CAMS data file and is also consistent with the time structure of our simulation model.

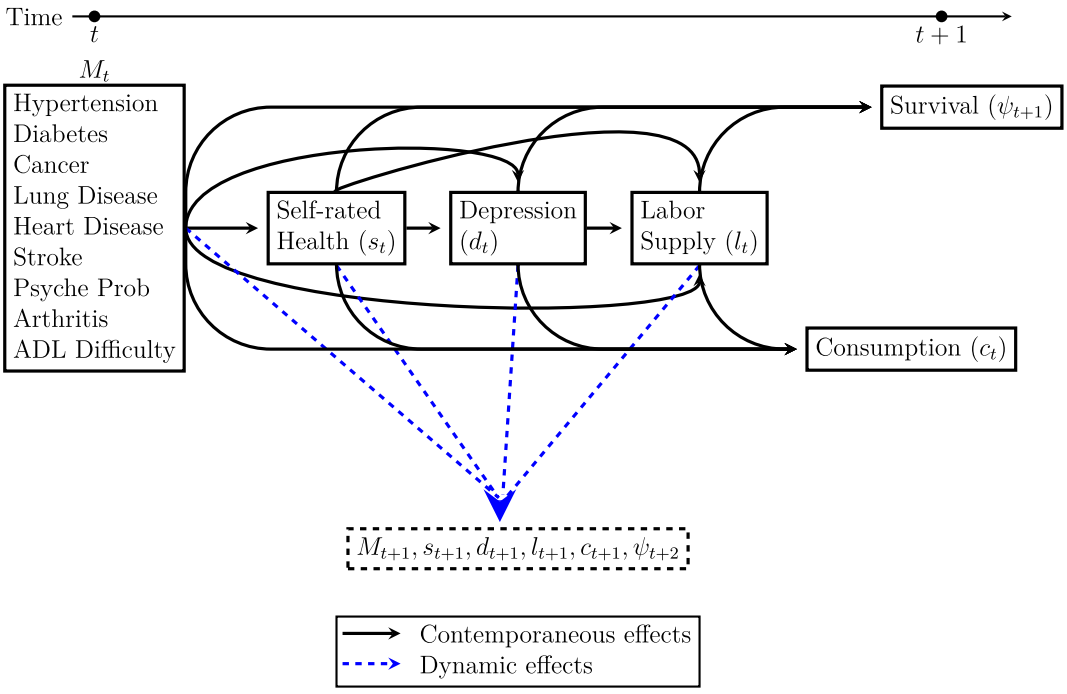

Fig. 1. Forecasting model with one period lag.

address missing consumption data by using the multiple imputation method proposed by Honaker and King (2010) for cross-sectional time-series data (see online appendix for details).

2.2. Simulation model

We extend and estimate the forecasting model proposed by Miller and Bairoliya (2022), adapted to include the onset and persistence of late-life depression. The model follows the structure of a panel vector autoregression (VAR) making it useful for microsimulations. Specifically, we use the model to repeatedly simulate potential outcome paths for each individual with and without the prevalence of late-life depression, given a set of initial (age sixty) conditions. Here we discuss the basic structure of the model and identifying assumptions. The online appendix provides additional details on sample selection, descriptive statistics, and model estimation procedures and results.

The general structure of the simulation model is illustrated in Fig. 1. At the beginning of each time period, morbidity status is updated based on (correlated) random shocks, which in turn influence an individual's self-rated health status. Morbidities and self-rated health then contemporaneously influence an individual's reported depression status.[5] We choose this outcome sequence because (1) it is consistent with evidence that general health affects depression (Moussavi et al., 2007; Ambresin et al., 2014); (2) it allows block identification of the system for estimation (details below); and (3) it provides a more conservative estimate of the welfare cost of depression. On the last point, there is quite plausibly some contemporaneous reverse causation between depression and general health (Rothermund and Brandtstädter, 2003; Moussavi et al., 2007). However, our later counterfactual simulations will assume that depression does *not* influence current period general health, yielding the more conservative estimate of its total welfare burden (see online appendix for robustness simulations where we relax this assumption). The simulations *will* allow current depression status to influence the evolution of health moving forward through general lagged effects. For example, mild depression today may result in a higher chance of stroke or lower self-rated health the following period. Moreover, higher order lagged effects allow, for example, the recent onset of depression to alter next period self-rated health more than if an individual has been living with depression for an extended period of time.

The latter part of the model allows morbidities, self-rated health, and depression to influence labor supply, consumption, and survival to following period of life. The assumed pathway from health to labor supply to consumption is consistent with evidence that health and depression affects the retirement decision (Currie and Madrian, 1999; Doshi et al., 2008; Rice

[5] Note that we posit each of the morbidity states to contemporaneously influence depression both directly and through changes in self-rated health. For example, a stroke may lower an individual's self-rated health status which in turn may worsen depression. However, a stroke may also influence depression beyond any changes in self-rated health.

et al., 2011), that consumption declines with retirement due to lower income and changing time constraints (Hall, 2009), and that health impacts economic outcomes, particularly at older ages (Smith, 1999).[6]

2.2.1. Panel VAR representation

While multiple lags are used in estimation of the simulation model, the following VAR(1) demonstrates the key features of the framework (see online appendix for extension to higher order lags). Let Y_{it} be a vector of outcomes for individual i at time t that includes depression d, log consumption c, retirement indicator r, self-rated health s, and our $n = 9$ morbidity states given by $n \times 1$ vector M. Outcomes are assumed to jointly evolve according to the structural VAR(1) model:

$$AY_{it} = BY_{it-1} + \epsilon_{it},$$

where ϵ is a vector of mean zero shocks that are normally distributed. The shocks are assumed to be independent and identically distributed (*iid*) across individuals and time and independent across outcomes. The main diagonal terms of matrix A are scaled to one and we assume that all parameters are homogeneous across individuals and time (e.g. $A_{it} = A \quad \forall i, t$). This implies that estimated relationships are assumed constant across individuals in our sample. For example, the marginal impact of depression on consumption does not change over time or across individuals.[7]

We estimate our model in five "blocks" of outcomes—the morbidity block consisting of n outcomes, and the self-rated health, depression, retirement, and consumption blocks, each consisting of one outcome. The unrestricted model can be written in block matrix form as:

$$\begin{array}{c} \overbrace{\phantom{-A_{11}}}^{n} \\ \left.\begin{array}{c} \\ \\ \\ \\ \end{array}\right\} n \\ \left.\begin{array}{c} \\ \\ \\ \\ \end{array}\right\} 4 \end{array} \overbrace{\left[\begin{array}{c|cccc} -A_{11} & -A_{12} & -A_{13} & -A_{14} & -A_{15} \\ \hline -A_{21} & 1 & -a_{23} & -a_{24} & -a_{25} \\ -A_{31} & -a_{32} & 1 & -a_{34} & -a_{35} \\ -A_{41} & -a_{42} & -a_{43} & 1 & -a_{45} \\ -A_{51} & -a_{52} & -a_{53} & -a_{54} & 1 \end{array}\right]}^{4} \left[\begin{array}{c} M_{it} \\ \hline s_{it} \\ d_{it} \\ r_{it} \\ c_{it} \end{array}\right] = \overbrace{\left[\begin{array}{c|cccc} B_{11} & B_{12} & B_{13} & B_{14} & B_{15} \\ \hline B_{21} & b_{22} & b_{23} & b_{24} & b_{25} \\ B_{31} & b_{32} & b_{33} & b_{34} & b_{35} \\ B_{41} & b_{42} & b_{43} & b_{44} & b_{45} \\ B_{51} & b_{52} & b_{53} & b_{54} & b_{55} \end{array}\right]}^{4} \left[\begin{array}{c} M_{it-1} \\ \hline s_{it-1} \\ d_{it-1} \\ r_{it-1} \\ c_{it-1} \end{array}\right] + \left[\begin{array}{c} \epsilon_{1,it} \\ \hline \epsilon_{2,it} \\ \epsilon_{3,it} \\ \epsilon_{4,it} \\ \epsilon_{5,it} \end{array}\right],$$

where $n \times n$ matrix A_{11} has main diagonal terms scaled to one.

The causal pathways we propose in Fig. 1 suggest a block recursive system. Specifically, we assume that $A_{12} = A_{13} = A_{14} = A_{15} = 0$ in the morbidity block, $a_{23} = a_{24} = a_{25} = 0$ in the self-rated health block, $a_{34} = a_{35} = 0$ in the depression block, and $a_{45} = 0$ in the retirement block. In other words, we assume the contemporaneous causal pathway runs from morbidities to self-rated health to depression to retirement to consumption. However, we allow health and retirement to affect future outcomes through lagged effects.[8] Block triangulation of the system eliminates simultaneity across blocks and allows for block-by-block estimation.[9]

2.2.2. Exogenous characteristics

We also allow the evolution of outcomes in the simulation model to depend on a set of exogenous individual characteristics. Denoting the $k \times 1$ vector of exogenous regressors X_{it}, the VAR(1) model may be written:

$$AY_{it} = BY_{it-1} + CX_{it} + \epsilon_{it}. \tag{1}$$

Exogenous characteristics include a linear calendar year trend and dummies for age, education, gender, race, census division, census occupation code, birth cohort, and a post-2008 recession indicator.[10] In order to replicate the observed variance in consumption in the data, we also include a time invariant individual fixed effect π in the consumption equation. The fixed

[6] In contrast, the effects of economic status on health appear concentrated during childhood and young adulthood when health trajectories are being established (Smith, 1999).

[7] As detailed below, all outcomes besides consumption are simulated using nonlinear models (e.g., ordered probit for depression). So while the coefficients are assumed homogeneous in the nonlinear models, clearly marginal effects differ across individuals. For example, the marginal impact of depression on retirement probability will depend on other modeled characteristics like age, calendar year, and co-morbidities.

[8] Though we assume there is no such feedback from consumption and set $B_{15} = b_{25} = b_{35} = b_{45} = 0$.

[9] Note this produces the same results as the Cholesky decomposition of shocks from a reduced form VAR.

[10] The inclusion of age, cohort, and calendar year introduces some multicollinearity into the model, so interpreting point estimates on these variables should be done with caution. However, using the estimates for forecasting does not pose an issue (Holford, 1991).

effects acts as person specific drift in the autoregressive process. The modeled exogenous characteristics can be explicitly written as:

$$CX_{it} = \underset{n}{\left\{ \underbrace{\left[\begin{array}{cccccccccc} C_{11} & C_{12} & C_{13} & C_{14} & C_{15} & C_{16} & C_{17} & C_{18} & C_{19} & 0 \\ \hline c_{21} & c_{22} & c_{23} & c_{24} & c_{25} & c_{26} & c_{27} & c_{28} & c_{29} & 0 \\ c_{31} & c_{32} & c_{33} & c_{34} & c_{35} & c_{36} & c_{37} & c_{38} & c_{39} & 0 \\ c_{41} & c_{42} & c_{43} & c_{44} & c_{45} & c_{46} & c_{47} & c_{48} & c_{49} & 0 \\ c_{51} & 0 & 0 & 0 & 0 & 0 & 0 & c_{58} & c_{59} & c_{510} \end{array}\right]}_{(n+4)\times k} \right.} \underbrace{\left[\begin{array}{c} Age_{it} \\ Education_i \\ Gender_i \\ Race_i \\ Division_i \\ Occupation_i \\ Cohort_i \\ Year_t \\ Post_t \\ \pi_i \end{array}\right]}_{k\times 1}.$$

Note that we normalize $c_{510} = 1$ to allow identification of the unobserved fixed effects. We have also excluded time invariant exogenous characteristics from the consumption equation due to colinearity with the fixed effect. However, we include socioeconomic characteristics instead of additional fixed effects in the health and retirement equations because (1) morbidities and retirement are absorbing states and depression and self-rated health are ordinal, each of which poses difficulties in estimating dynamic panel models with fixed effects[11] and (2) the simpler model does well in replicating the observed dynamics of health and retirement in the data (see online appendix).

2.2.3. Morbidities

As there are multiple morbidities in the triangulated VAR system, we cannot identify the underlying structural parameters in the morbidity block. Instead we estimate the block as a reduced form VAR. We can premultiply the structural morbidity block by the inverse of matrix $-A_{11}$ to obtain the reduced form system:

$$M_{it} = \hat{B_1} M_{it-1} + \hat{B_2} s_{it-1} + \hat{B_3} d_{it-1} + \hat{B_4} r_{it-1} + \hat{C} X_{it} + e_{it},$$

where $\hat{B_j} = -A_{11}^{-1} B_{1j}$, $\hat{C} = -A_{11}^{-1}[C_{11}, \ldots, C_{19}]$ and $e_t = -A_{11}^{-1}\epsilon_{1,t}$. In this reduced form system all right hand side variables are predetermined at time t and morbidity states do not a have direct contemporaneous effect on each other. However, the error terms e_t are composites of morbidity specific structural shocks and thus are potentially correlated across morbidity states (i.e. $cov\left(e_{it}, e'_{it}\right) \neq 0$). This allows for contemporaneous correlation in the probability of morbidity states. For example, the onset of heart disease may be correlated with the onset of hypertension or stroke due to correlated contemporaneous shocks. Reduced form morbidity shocks are assumed to follow a standard multivariate normal distribution with an $n \times n$ covariance matrix given by Σ.

As morbidity outcomes are binary, forecasting of the measures is not a true linear VAR process. Instead, we assume a continuous latent variable m^* underlies each observed outcome such that:

$$m_{j,it} = 0 \; if \; m^*_{j,it} \leq 0$$
$$m_{j,it} = 1 \; if \; m^*_{j,it} > 0$$

for $j = 1 \ldots n$. The estimated reduced form VAR can then be written:

$$\begin{bmatrix} m^*_{1,it} \\ \vdots \\ m^*_{n,it} \end{bmatrix} = \begin{bmatrix} \hat{b}_{11} & \cdots & \hat{b}_{1n} \\ \vdots & \ddots & \vdots \\ \hat{b}_{n1} & \cdots & \hat{b}_{nn} \end{bmatrix} \begin{bmatrix} m_{1,it-1} \\ \vdots \\ m_{n,it-1} \end{bmatrix} + \hat{B_2} s_{it-1} + \hat{B_3} d_{it-1} + \hat{B_4} r_{it-1} + \hat{C} X_t + \begin{bmatrix} e_{1,it} \\ \vdots \\ e_{n,it} \end{bmatrix}. \tag{2}$$

Note that each latent morbidity variable is determined by lagged values of the other *observed* self-rated health, depression, and morbidity states. As we have assumed joint normality in the error term, this morbidity block of equations is in the form of a multivariate probit model.

2.2.4. Self-rated health

Self-rated health is measured on a five point scale so we assume a continuous latent variable s^* underlies the observed self-rated health state. The relevant equation from system (1) can then be explicitly written as:

$$s^*_{it} = A_{21} M_{it} + B_{21} M_{it-1} + b_{22} s_{it-1} + b_{23} d_{it-1} + b_{24} r_{it-1} + [c_{21}, \ldots, c_{29}] X_{it} + \epsilon_{2,it}, \tag{3}$$

[11] For example, it is not possible to estimate fixed effects for individuals that never enter an absorbing state in the data and estimated fixed effects would be needed for our simulations. This is one reason we exclude fixed effects from the health and retirement equations.

with the observed self-rated health state defined as:

$$s_{it} = \delta \;\; if \;\; \kappa_{\delta-1} < s^*_{it} < \kappa_\delta \;\; for \; \delta = 1, \ldots, 5$$

for cut-points $(\kappa_0, \ldots, \kappa_5)$. The worst health state (poor) is given by $\delta = 1$ and the best health state (excellent) by $\delta = 5$. We assume ϵ_2 is an *iid* shock with standard normal distribution so that the evolution of self-rated health follows an ordered probit structure. Unlike the morbidity block, block triangulation of the system allows this equation to be estimated independently of other outcome blocks with all structural parameters identified.

2.2.5. Depression

Similar to other health outcomes, we assume a continuous latent variable d^* underlies the observed depression state such that the forecasting equation given in system (1) can be written:

$$d^*_{it} = A_{31}M_{it} + B_{31}M_{it-1} + a_{32}s_{it} + b_{32}s_{it-1} + b_{33}d_{it-1} + b_{34}r_{it-1} + [c_{31}, \ldots, c_{39}]X_{it} + \epsilon_{3,it}, \tag{4}$$

with the observed depression state defined as:

$$d_{it} = \delta \;\; if \;\; \kappa_\delta < d^*_{it} < \kappa_{\delta+1} \;\; for \; \delta = 0, \ldots, 8$$

for cut-points $(\kappa_0, \ldots, \kappa_9)$ with $\delta = 0$ representing the no depressive symptoms state and $\delta = 8$ the worst depression state. Note that latent depression is assumed to depend on the lagged value of the *observed* depression category to incorporate the persistence in depression over time. We assume ϵ_3 is an *iid* shock with standard normal distribution yielding an ordered probit structure for the depression model. Given our block recursive system, this equation may also be estimated independently of other blocks with all structural parameters identified.

2.2.6. Retirement

As retirement is a binary outcome, we again assume a continuous latent variable r^* underlies the observed outcome such that:

$$r_{it} = 0 \; if \; r^*_{it} \leq 0$$
$$r_{it} = 1 \; if \; r^*_{it} > 0.$$

Conditional on working the previous period, the retirement block equation is given by:

$$r^*_{it} = A_{41}M_{it} + a_{42}s_{it} + a_{43}d_{it} + B_{41}M_{it-1} + b_{42}s_{it-1} + b_{43}d_{it-1} + [c_{41}, \ldots, c_{49}]X_{it} + \epsilon_{4,it}. \tag{5}$$

Note that as retirement is an absorbing state, we set $b_{44} = 0$. In addition to exogenous individual characteristics, retirement is influenced by current and lagged values of health (depression, self-rated health, and specific morbidities). We assume ϵ_4 is an *iid* shock with standard normal distribution implying the retirement model has a standard probit structure.

2.2.7. Consumption

The consumption forecasting equation given in system (1) can be explicitly written as:

$$\begin{aligned} c_{it} &= A_{51}M_{it} + a_{52}s_{it} + a_{53}d_{it} + a_{54}r_{it} + B_{51}M_{it-1} + s_{52}d_{it-1} + b_{53}d_{it-1} + b_{54}r_{it-1} \\ &\quad + b_{55}c_{it-1} + c_{51}Age_{it} + c_{58}Year_t + c_{59}Post_t + \pi_i + \epsilon_{5,it}. \end{aligned} \tag{6}$$

This equation is in the form of a standard linear dynamic panel data model with lagged dependent variable and individual level fixed effects. Block triangulation of the system also allows this equation to be estimated independently of other blocks with all structural parameters identified including the variance of ϵ_5.

2.2.8. Mortality

Mortality probabilities are estimated independently of the VAR system above as all other outcomes are conditional on survival. Survival from time period $t-1$ to time period t is modeled by:

$$\psi_{it} = I\left(\sum_{k=1}^{K}\left[\gamma^M_k M_{it-k} + \gamma^s_k s_{it-k} + \gamma^s_k d_{it-k} + \gamma^r_k r_{it-k}\right] + \delta X_{it} + u_{it} > 0\right), \tag{7}$$

where $I(.)$ is an indicator function and $\psi = 1$ indicates survival, X the vector of observed individual characteristics previously defined, and u_{it} an *iid* random shock with standard normal distribution. The model allows K lags of health, depression, and retirement to influence survival probability.

2.3. Welfare measure

We use an ex-ante consumption-compensating variation (CV) measure to quantify the welfare costs of late-life depression using simulations from our VAR model. We first define expected lifetime utility at age j for individual i as:

$$E\left[\sum_{a=j}^{J}\psi_{ia}\beta^{a-j}\phi(h_{ia})[\bar{u}+log(c_{ia})+\nu(l_{ia})]\right]$$

where c is consumption, l leisure, h health, and ψ is a survival indicator. Health measure h is a vector of modeled morbidities, self-rated health, and depressive symptoms. Expectations are taken over the uncertain path of all outcomes after age j. This simple formulation yields an additive decomposition of welfare allowing us to add cumulative corrections for the cost of depression on comorbidities, mortality, leisure, and consumption (see online appendix for derivation). We also check the robustness of our results to more general preferences (see robustness Section 3.5 for results). We model health in the utility function to map to the large literature on quality-adjusted life years (QALYs). Specifically, we assume utility from consumption and leisure each period is scaled by the health function $\phi(h)\in[0,1]$. Here, $\phi(h)=1$ represents utility in the "best" health state and $\phi(h)=0$ represents death. In this form, $\psi\phi(h)$ provides a measure of QALYs. For example, a year spent in the best health state is a single QALY and represented by $\psi\phi(h)=1$.

Let $U_{ij}(1-\lambda)$ denote the expected lifetime utility at age j from the outcome bundles of individual i if consumption is multiplied by a factor $(1-\lambda)$ at each age and realization of the world:

$$U_{ij}(1-\lambda)=E\left[\sum_{a=j}^{J}\psi_{ia}\beta^{a-j}\phi(h_{ia})[\bar{u}+log((1-\lambda)c_{ia})+\nu(l_{ia})]\right].$$

The consumption-compensating variation measure of welfare for individual i, λ_{ij}, is derived through the condition:

$$U_{mj}\left(1-\lambda_{ij}\right)=U_{ij}(1), \tag{8}$$

where U_{mj} refers to the expected lifetime utility from the outcome bundles in the absence of any possible depression after age j. In words, λ_{ij} is the proportion of the individual's (depression-free) consumption they would be willing to give up at every age starting from j (in all possible realizations of the world and holding health and leisure fixed) to eliminate all possibility of depression after age j. For example, if person i expects depression to be a serious problem in late life, they may have a welfare measure $\lambda_{ij}=0.3$. This implies they would be ex-ante willing to give up to 30% of their consumption in every period from age j to avoid any possibility of depression. Note that this does not imply that depression must have any direct effect on consumption. It could be the entire 30% is due to lost health utility. The welfare measure simply provides a convenient way to combine multiple types of utility costs (e.g., health, mortality, retirement) into a common metric—dollars of consumption. Moreover, as this measure is based on potential outcomes over remaining life, it encompasses the likelihood of persistence and emergence of depression over remaining life.

In order to gain a sense of the aggregate cost of depression, we also calculate the product of an individual's expected remaining lifetime consumption at age sixty (ELC) and our CV measure: $\lambda\times ELC$. This is a similar concept (but not the same) as an individual's willingness-to-pay at age sixty to eliminate all possibility of depression after age sixty. Effectively, it is an individual's *expected* willingness-to-pay or the expected value of consumption they are willing to forgo.

2.3.1. Health utility weights

Analysis using our welfare model requires calibration of preference parameters. This includes parameters of the function $\phi(h)$ mapping health states into flow utility. We assume health utility depends linearly on our health state vector: $\phi(h_t)=\omega h_t$. Our health utility weights ω are derived from the Health Utilities Index Mark 3 (HUI3) instrument which was collected from approximately 1200 respondents in the HRS in the year 2000. The HUI3 was developed to produce cardinal utility scores on the conventional utility scale ranging from zero (death) to one (best health) and has been extensively used in the literature on health utilities (Furlong et al., 1998; Feeny et al., 2002; Horsman et al., 2003).[12] We use the HUI multi-attribute utility score (*hui3ou*).

The HUI3 was conceptualized such that $u(h_i)=HUI3_i\times u(h_{best})$ for individual i and general utility function $u(.)$. For example, a year in the best health state is equal in utility to two years with $HUI3=0.5$. As another example, three years of life spent with an $HUI3=0.33$ is equal to the same utility as about one year in perfect health, or a single QALY. In the context of our model, we assume that the HUI3 measures the relative utility across health states *holding consumption and leisure fixed*:

$$\omega h_i[\bar{u}+log(c_i)+\nu(l_i)]=HUI3_i\times h_{best}[\bar{u}+log(c_i)+\nu(l_i)].$$

This approach is consistent with the HUI3 instrument where the interview script reads: "*when imagining yourself in these health states please remember that where you live, your income, your friends, and family would be the same as now.*" With this

[12] HUI3 scores less than zero are possible and represent current health states worse than death, but this is very rare in our simulations and do not alter our results.

assumption, the above equation simplifies to $\omega h_i = HUI3_i$ when $h_{best} = 1$. The utility weights ω can then be estimated by regressing the HUI3 utility score on depression score, self-rated health, and all morbidity indicators. Results are also robust to relaxing the assumption of holding consumption and leisure fixed (see online appendix).

2.3.2. Calibration of other parameters

Leisure is normalized to one for retired individuals. Leisure for working individuals is set to $0.66 = 1 - (2000/5,840)$, based on an annual time endowment of 5840 h (16 h a day $\times$ 365 days) and 2000 h of work. Preferences over leisure are defined by $v(l) = -\frac{\theta\epsilon}{1+\epsilon}(1-l)^{\frac{1+\epsilon}{\epsilon}}$, where ϵ is a constant Frisch elasticity of labor supply. Note that in addition to retirement, we are assuming that the cost of depression on leisure is captured through the health utility function $\phi(h)$. In other words, depression is assumed to make leisure time less valuable in terms of utility. It is feasible there could be an additional direct time cost of depression, for example due to additional time needed for treatment or personal care. By not explicitly attempting to include a direct time cost, our model is again pushed towards a conservative estimate of the costs of depression. We follow Miller and Bairoliya (2022) and set the disutility weight θ such that the marginal cost of leisure equals the marginal benefit for the median individual in our sample. This gives us a benchmark $\theta = 7.8$. We use a benchmark value of $\epsilon = 1$ and a discount factor $\beta = 0.98$ implying an annual discount rate of one percent (with additional discounting implicit due to mortality risk). We examine robustness of results to each of these parameter values.

Finally, note that with our benchmark preferences, as long as flow intercept $\bar{u}$ plus log consumption is positive (and health utility is positive), a retired individual will prefer life to death in the current period.[13] After normalizing consumption to thousands of 2010 dollars, we set $\bar{u} = -log(3)$, implying that \$3000 of consumption is needed for a retiree to maintain positive flow utility. This is approximately 10% of mean annual consumption in our sample. Although there is not much evidence on this value, Murphy and Topel (2006) argue 10% as a reasonable parameterization. This value also yields a mean and median value of remaining life for sixty year olds in our simulation sample of \$78,000 and \$47,000 per QALY.[14] In a review of the literature, Ryen and Svensson (2015) estimate mean and median values of life across studies of approximately \$98,000 and \$32,000.[15] Traditional values in the U.S. often range from \$50,000 to \$100,000 (Kaplan and Bush, 1982). In some robustness exercises, we show that using log consumption and a relatively low value of life in our benchmark likely yields conservative estimates of welfare costs.

2.4. Estimation and simulations

Equipped with our simulation model and welfare concept, our empirical analysis involves four steps.

1. We use data from the HRS to estimate the parameters of the simulation model. Here we use data on all individuals aged fifty and older from all available waves of the HRS from 1992 to 2016 (40,708 unique individuals and 238,091 total individual-year observations). See the online appendix for details on the model estimation sample and procedures.
2. We repeatedly simulate remaining life-cycle paths for all outcomes for a sub-sample of the HRS respondents using the parameter estimates and age sixty data as initial conditions. This simulation sample includes all individuals with age sixty data and requisite lagged data for simulations. This yields representative results over four birth cohorts—early HRS (EHRS), late HRS (LHRS), War Babies, and (early) Baby Boomers. See the online appendix for details on initial condition descriptives, sampling weights and representativeness, and simulation procedure.
3. We estimate a lower bound of the welfare costs of depression after age sixty for each individual in our simulation sample by assuming there are no spillover effects on other model outcomes. More specifically, we leave all expected paths of morbidities, self-rated health, mortality, labor supply, and consumption at their baseline levels and only remove the health utility penalty associated with depression at age sixty-two and older (i.e., we set all the CESD weights to zero in Table 1). In doing so, we are assuming that all statistical associations estimated in our VAR model do not reflect any causal impact going from depression to other modeled outcomes (hence, no spillover).
4. We estimate an upper bound of the welfare costs of depression by running a new set of simulations starting from the same initial conditions but removing any possibility of depressive symptoms after age sixty. We consider this an upper bound as it assumes all the coefficients estimated in the simulation model are purely causal. We embed the baseline and counterfactual simulated data within our expected utility framework to construct a measure of the ex-ante welfare cost of future depression at age sixty for each individual in our simulation sample.

[13] A current period utility less than zero does not necessarily imply an individual would rather die than continue living, as it does not guarantee an expected remaining lifetime utility less than zero. For example, consumption may be expected to increase from next period onward.

[14] The value of life per QALY at age j is given by $VOL_j/E\left[\sum_{a=j}^{J}\psi_a\beta^{a-j}\phi(h_a)\right]$ where $VOL_j = U_{ij}(1)c_j/\phi\left(h_j\right)$.

[15] Ryen and Svensson (2015) document substantial variation across estimates of willingness-to-pay for a QALY, most notably with conversions based on revealed preferences of the value of statistical life (VSL) averaging 5–7 times higher than those based directly on stated preferences. The VSL studies reviewed are by definition measuring value of length of life, while stated preference studies elicited willingness-to-pay for pure quality of life improvements, pure length of life, or a mixture of both.

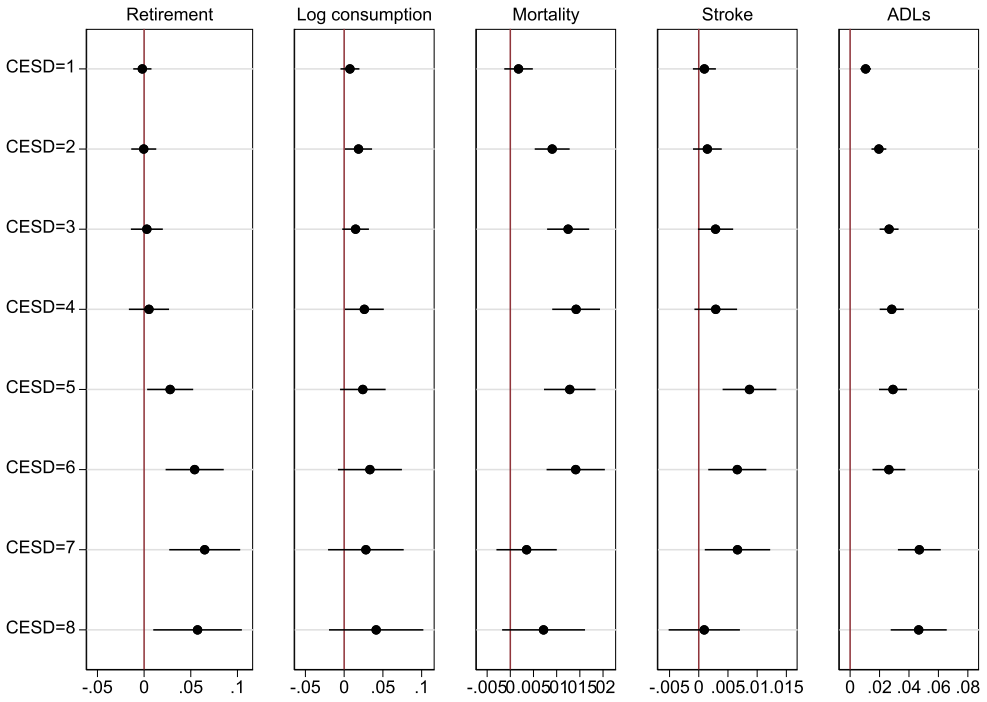

Fig. 2. Selected estimation results. *Notes*: Dependent variables across columns. Average marginal effects on the probability of an outcome reported for probit results—retirement, mortality, stroke, and ADLs. Contemporaneous associations reported for retirement and log consumption as dependent variables. Lagged associations reported for mortality, stroke, and ADLs. CESD=0 (no depression) is the reference group. Spikes indicate 95% confidence intervals.

3. Results

3.1. Model estimates

We begin with estimation results from our simulation model to demonstrate the association between depression and other outcomes in the data. Selected results are provided in Fig. 2 while the full set of results are available in the online appendix. The first panel shows the average marginal effects of depressive symptoms on the contemporaneous probability of retirement (controlling for other health outcomes as shown in model (1)). Results indicate that low and mild depression (CESD = 0,1,2,3,4) do not have a significant association with the probability of retirement for older adults. However, as the severity of depression increases, a significant association emerges. For example, there is an increase in the probability of retirement of around 2 percentage points (pp) for a CESD score of five, rising to a point estimate of over 5 pp at higher levels of depression. The second panel of Fig. 2 shows a small *positive* relationship between the severity of depression and contemporaneous log consumption. For example, a CESD score of five is associated with an increase in consumption of about 0.03 log points. Expectations about the longevity and quality of life may be a plausible explanation for this positive association among older adults. For example, lower expectations about a long and healthy life might push older depressed adults to discount their future utility and hence consume more in the near term. Moreover, there is an additional indirect effect operating on consumption due to any changes in retirement induced by depression. For example, we saw that a CESD score above five is associated with an increased probability of retirement. Our estimates also show that retirement is associated with an immediate decrease in consumption of about 0.04 log points (see appendix Table 5).

Panel three of Fig. 2 shows a generally positive association between depressive symptoms and mortality. For example, an individual with a CESD score of three has about a 1 pp lower probability of surviving to the next model period compared to if they had a CESD score of zero. Note that at the highest CESD scores the association with mortality is moderately diminished, although results are somewhat noisy given that relatively few individuals have very high levels of depression (about 4% of our sample). In addition to a comparatively small sample with very high depression score, this pattern is also explained by indirect associations between high CESD score and mortality. For example, CESD scores higher than six have

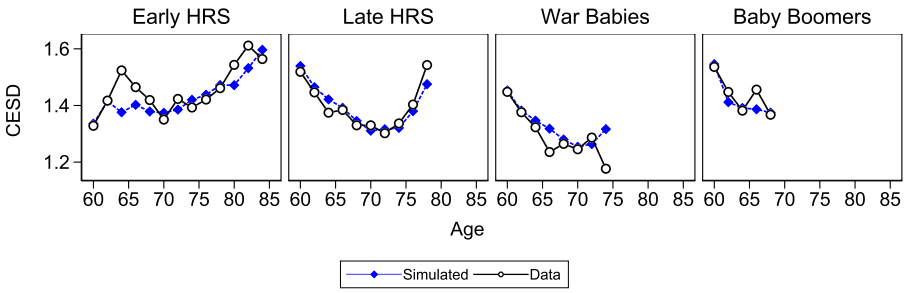

Fig. 3. Mean of life-cycle CESD profiles by cohort. *Notes*: "Data" plots mean of all available data (inclusive of imputed missing values) in HRS by two-year age interval and cohort. "Simulated" plots mean of expected simulated outcome for each observation in the data (i.e. the expected outcome for each person-year observation in the data).

a strong correlation with several worse health outcomes like difficulties with ADLs (see the last panel of Fig. 2). So part of the association between high CESD score and mortality is captured through poorer health.[16]

The final two panels help illustrate the dynamics of the system by showing the average marginal effects of current depression state on the probability of having a stroke or ADL difficulty the *following* model period. For example, a CESD score of five increases the probability of a stroke the following period by nearly 1 pp and the probability of having difficulty with ADLs by more than 2 pp. Moreover, these relationships continue to propagate dynamically throughout the system influencing the evolution of other comorbidities and self-rated health along with future retirement and consumption decisions.

3.1.1. Simulation fit

A comparison between mean simulated CESD scores and those based on available data is shown by age and cohort in Fig. 3. Additional comparisons for each outcome by cohort are provided in the online appendix. In both the data and simulations, mean CESD score tends to rise with age in the EHRS cohort.[17] For the LHRS cohort the relationship is U shaped, CESD scores decline until the age of 72, then begin to increase. For the younger War Baby and Baby Boomers cohorts there is less available data, but both cohorts have falling CESD scores over the sixties. Note that by construction, the data and simulations are the same at age sixty. However, using only age sixty data and the estimated model parameters, the simulations continue to match the data reasonably well even up to 24 years later (when the EHRS cohort is age 84). Overall, the simulations match the available aggregated data well suggesting our life-cycle dynamics model provides a good approximation of the underlying data generating processes.

3.1.2. Health utility weights

Table 1 provides our health utility weights ω estimated via a linear regression of HUI3 utility score on health outcomes. Depressive symptoms measured by the CESD scale have a strong and highly significant negative association with utility. For example, moving from no depressive symptoms (the base category) to a CESD score of three lowers flow health utility by 7.9 pp. Moving all the way to a score of eight lowers health utility by 29.3 pp. In addition to depression, self-rated health also has a strong association with health utility. For example, moving from poor health (the base category) to good health improves flow health utility by 25.0 pp. Conditions such as hypertension, diabetes, and cancer have little independent effect on health utility after controlling for their association with self-rated health, depression, and other comorbidities. Other morbidities such as stroke and arthritis have larger (and statistically significant) independent negative effects. The most influential morbidity indicator is difficulty with ADLs, which lowers health utility by an estimated 14.2 pp.

While the eight-point CESD does not map directly into clinical diagnosis of depression disorder, Turvey et al. (1999) propose a CESD score of six or higher to approximate cases of clinical depression. Our weights then imply that a clinically depressed individual in good self-rated health and without other comorbidities would have a health utility score between 0.56-0.68. A clinically depressed individual with poor self-rated health would have a score of 0.31–0.43. In a systematic review, Mohiuddin and Payne (2014) examine results from studies using indirect valuation methods to estimate health utility scores in alternate depressive states. They calculate pooled mean utilities across studies of 0.56 for mild, 0.45 for moderate, and 0.25 for severe depression. By comparison, our results are likely conservative in attributing health utility penalties to depressive states.

[16] For example, when we exclude other health outcomes from our mortality equation, CESD scores higher than six show slightly *stronger* associations with mortality than lower CESD scores.

[17] Conditioning data on survival to the end of the simulation period to eliminate mortality bias yields lower CESD scores but similar dynamics for each cohort (see figure in online appendix).

Table 1
Estimated health utility weights (ω).

Measure	Weight	SE
Depression		
CESD=1	-0.021	0.015
CESD=2	-0.087	0.018
CESD=3	-0.079	0.023
CESD=4	-0.094	0.028
CESD=5	-0.138	0.030
CESD=6	-0.172	0.039
CESD=7	-0.225	0.046
CESD=8	-0.293	0.056
Hypertension	0.004	0.012
Diabetes	-0.000	0.017
Cancer	0.007	0.017
Lung disease	-0.024	0.021
Heart disease	-0.034	0.015
Stroke	-0.073	0.022
Psych problem	-0.041	0.020
Arthritis	-0.053	0.012
Difficulty with ADL	-0.142	0.016
Self-rated health		
Fair	0.179	0.026
Good	0.250	0.027
Very good	0.331	0.028
Excellent	0.338	0.032
Constant	0.610	0.030

Notes: Results from regression of HUI3 score on CESD score, self-rated health, and morbidities. SE denotes standard error. $R^2 = 0.48$. N = 1,089.

Table 2
Mean costs of depression after age sixty.

		Cumulative corrections			
	Depression	Comorbidities	Mortality	Leisure	Consumption
Expected loss					
QALYs	0.853	1.241	2.064	2.064	2.064
Labor supply (yrs)				0.095	0.095
Consumption (annual)					-0.631
CV (λ)	0.084	0.108	0.158	0.156	0.148
$\lambda \times$ELC	45.933	63.030	94.128	93.459	89.405

Notes: Estimates use base year respondent analysis weights. ELC denotes expected lifetime consumption. Consumption in $1000s.

3.2. Cost of depression

We start with a detailed examination of the cost of depression in the EHRS cohort as it is the oldest of the four cohorts and contains the longest panel of available data. Table 2 shows the mean cost of depression after age sixty for the EHRS. The first column provides our lower bound estimate where we simply remove the health utility penalty of depressive symptoms but leave all simulated outcomes at their baseline levels. On average, removing the health utility penalty of depression increases quality-adjusted life expectancy by 0.85 years. The mean associated CV welfare measure is 0.084, implying a willingness-to-pay up to 8.4% of annual consumption over remaining life to avoid any possibility of depression. As shown in the final row, this amounts to an expected loss of $45,933 of lifetime consumption.

The final four columns of Table 2 provide results from re-simulating outcomes for each individual after removing any possibility of depression after age sixty. Each column cumulatively adjusts welfare for an additional outcome with our "fully adjusted" upper bound provided in the last column. For example, when accounting for the possible spillover effects of depression on comorbidities (column two), eliminating depression increases quality-adjusted life expectancy by 1.24 years. The mean associated CV implies a willingness-to-pay up to 10.8% of annual consumption to avoid depression when accounting for these spillovers, or an expected loss of $63,030 of lifetime consumption. Further adjusting for the effect of depression on mortality rates yields an increase in quality-adjusted life expectancy of 2.06 years, a willingness-to-pay of 15.8% of annual consumption, or an expected loss of $94,128 in lifetime consumption.

Moving to leisure time, our simulations suggest that eliminating depression after age sixty could increase labor supply only by an average of about 1.1 months (0.095 years). This relatively small impact is likely due to the fact that many in-

dividuals in the simulation sample are already retired by age sixty and the direct effects of depression on retirement are minimal except with quite severe symptoms (recall Fig. 2). As increased labor supply alone results in a loss in welfare due to less leisure time, the mean CV falls very slightly to 15.6% of annual consumption. Finally, simulations suggest eliminating depression could lower consumption by up to $631 annually. This is consistent with the positive contemporaneous association between depression and annual consumption shown in Fig. 2. The mean associated CV implies the willingness-to-pay falls to 14.8% of annual consumption to avoid depression when accounting for these consumption losses. However, as we demonstrate in the next section, a fall in annual consumption does *not* imply a fall in lifetime consumption.

In order to gain a better sense of how depression influences the dynamics of other outcomes in the system, Fig. 4 plots the average percentage change in expected outcomes with the exogenous elimination of all prevalence of depression after age sixty. The first two plots show that the elimination of depression after age sixty is associated with a significant decline in psychiatric problems, difficulty with ADLs, and to some extent lung disease. For example, the elimination of depression is associated with nearly a 30% decrease in the probability of diagnosed psychiatric problems by the late-seventies. Associated effects were fairly small for the other morbidities.[18] These patterns are consistent with previous literature that has also found depressive symptoms to be predictive of increased difficulties with ADLs, such as eating, dressing, and bathing among the elderly (Penninx et al., 1999; Cronin-Stubbs et al., 2000; Kivelá and Pahkala, 2001; Kazama et al., 2011; Sodhi and Al Snih, 2020). A number of studies have also established a strong correlation between depression and chronic lung disease such as Chronic obstructive pulmonary disease (COPD) (Dowson et al., 2001; Van Manen et al., 2002; Mikkelsen et al., 2004; Kunik et al., 2005; Wilson, 2006; Ryerson et al., 2011). Hypothesized mechanisms linking depression to the decline in ADLs and increased lung disease include increased lifestyle risk factors (e.g., smoking, poor nutrition, low physical activity) and less social integration. For example, depression has been shown to significantly reduce the effectiveness of smoking cessation programs (Kinnunen et al., 1996; Cinciripini et al., 2003).

The third plot of Fig. 4 shows the upper bound effect of eliminating depression on health utility, labor supply, and mortality. The age-specific mortality rate is estimated to be over 10% lower by the early-seventies and remains more than 5% lower even into the nineties. In contrast, the probability of being retired falls only very slightly. This is consistent with the small association of depression with average labor supply discussed above. There is an immediate increase of about 8% in health utility at age sixty-two, which climbs to nearly 15% by age eighty. The final plot shows the response of consumption (unconditional and conditional on survival) to the elimination of depression. There is about a 1–2% decline in annual consumption conditional on survival throughout the remaining life-cycle. In contrast, when examining expected unconditional consumption (i.e. imputing zero consumption for the dead state), there is a large rise over time, reaching differences of more than 50% by the early nineties. These plots again highlight the small loss in annual consumption but potential gains in lifetime consumption due to an increase in life expectancy.

3.3. Distribution of cost

Another advantage of our approach is that we have individual level data and simulations so we are able to examine the entire distribution of estimated costs. Fig. 5 shows the distribution of estimated QALYs lost due to late-life depression. The “depression only” curve plots our lower bound estimate with a mean of 0.853 as shown in Table 2. The distribution demonstrates substantial inequality in the expected direct utility cost of late-life depression—the worst off individuals expect a loss of nearly three QALYs. When adding the estimated spillover effects on comorbidities, the mean shifts to 1.241 and the distribution flattens. This implies the health utility cost of depression becomes even more unequal when accounting for spillovers on other comorbidities. When further adjusting for increased mortality rates associated with depression, the mean reaches 2.064 and inequality in the distribution continues to rise.

Turning to economic outcomes, Fig. 6 shows the distribution of the estimated upper bound on the expected loss in consumption and labor supply associated with late-life depression. The first panel shows the change in expected annual consumption (conditional on survival). The negative values again demonstrate the small expected *gain* in annual consumption from late-life depression. For most individuals this gain is less a $1,000, though a small share expect to gain more than $3000 in annual consumption. Despite this rise in annual consumption with depression, the second panel of Fig. 6 shows there is an expected fall in total lifetime consumption for all individuals due to decreased life expectancy. The expected loss in lifetime consumption averages around $16,000, but ranges from almost zero to over $100,000. Finally, the third panel shows the distribution of lost labor supply. The potential loss due to early retirement is quite small—less than a year for all individuals.

Fig. 7 shows the distribution of the expected welfare cost of late-life depression (compensating variation) and the approximate expected monetary equivalent. As shown in Table 2, ignoring all spillovers (depression only model) yields a lower bound on the average welfare cost of 8.4% of annual consumption. The first panel of Fig. 7 shows that most of the lower bound distribution falls under 10%, though there is a thin right tail suggesting substantially higher costs for a select few. Likewise, the second panel shows this lower bound translates into an expected loss in lifetime consumption of under $100,000 for most individuals in the sample. When adjusting for potential spillover effects of depression on comorbidities,

[18] Cancer has relatively little association with depression or other morbidities. However, as eliminating depression improves chances of survival even when sick, there is actually a small increase in the prevalence of cancer (conditional on survival) starting around age eighty.

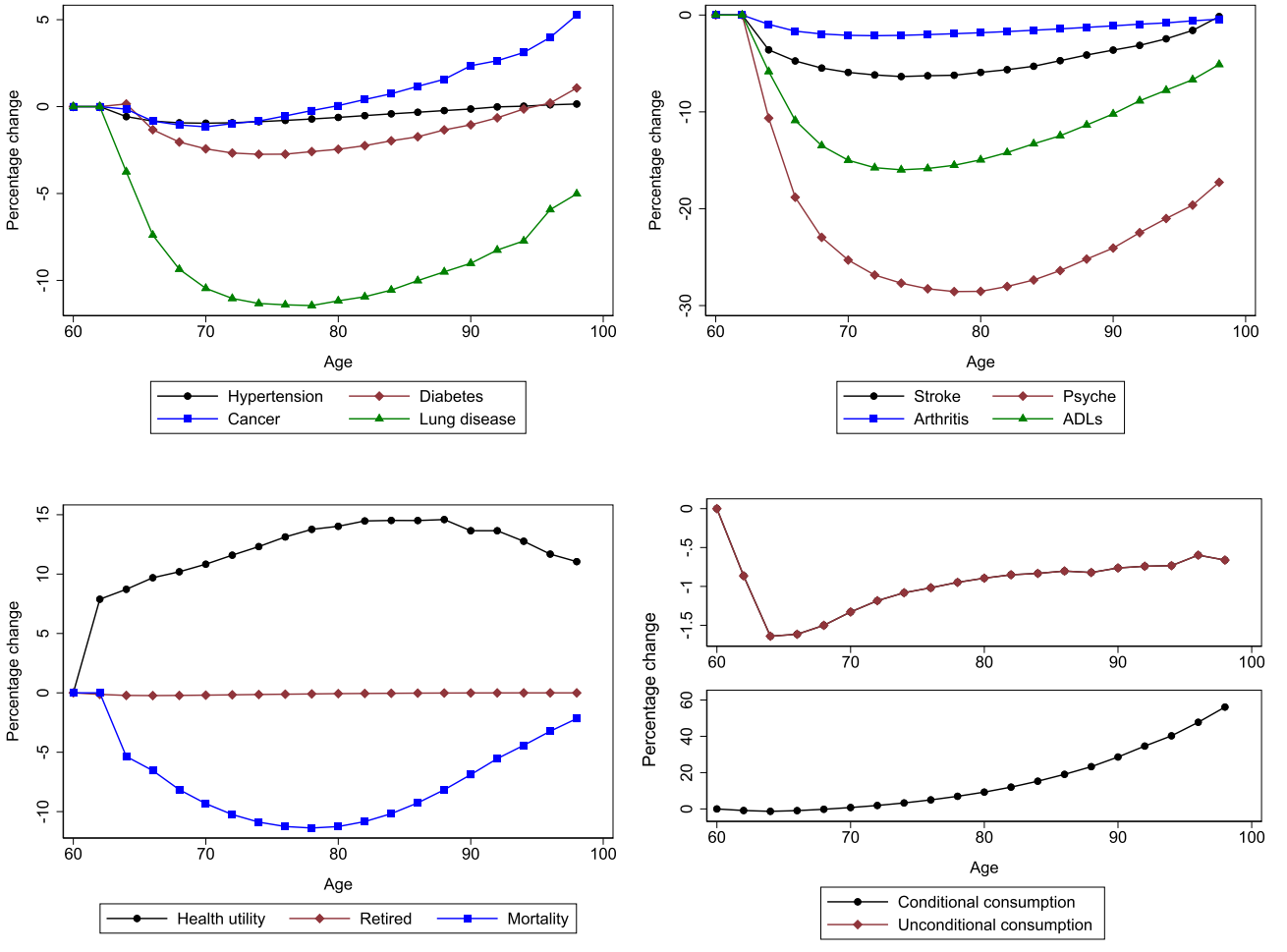

Fig. 4. Expected cost of depression by age. *Notes*: Results plot percentage difference in expected outcomes with the exogenous elimination of all prevalence of depression after age sixty. Sample includes all individuals in the simulation sample. Expected outcomes in first three panels are conditional on survival.

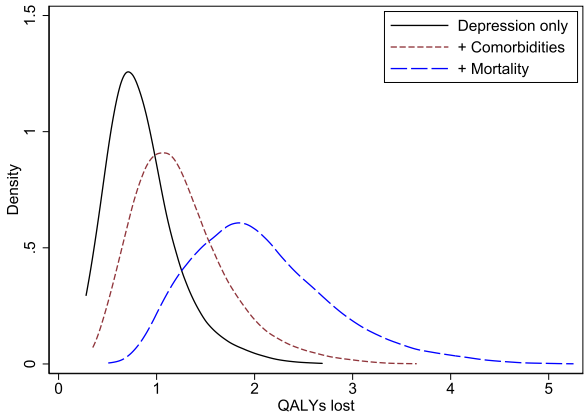

Fig. 5. Expected lost quality-adjusted life years (QALYs) after age sixty.

Table 3
Mean costs of depression after age sixty by birth cohort.

	Depression only				Full model			
	EHRS	LHRS	WB	BB	EHRS	LHRS	WB	BB
Expected loss								
QALYs	0.853	0.825	0.791	0.813	2.064	1.975	1.889	1.929
Labor supply (yrs)					0.095	0.097	0.097	0.112
Consumption (annual)					-0.631	-0.615	-0.558	-0.497
CV (λ)	0.084	0.083	0.077	0.075	0.148	0.144	0.134	0.128
$\lambda \times$ELC	45.933	46.630	44.370	40.131	89.405	90.406	85.584	77.338
CV Gini	0.337	0.359	0.381	0.419	0.280	0.293	0.316	0.347
CV P90	0.154	0.161	0.155	0.163	0.250	0.252	0.246	0.247

Notes: Estimates use base year respondent analysis weights. ELC denotes expected lifetime consumption. Consumption in $1000s.

mortality, leisure, and consumption, there is a substantial increase in the mean and inequality of welfare costs. For example, there is now a substantial portion of the distribution willing to pay over 20% of their annual consumption to avoid the possibility of late-life depression.

3.4. Cost over birth cohorts

Our analysis so far has focused on results only in the EHRS birth cohort. We now compare our estimated welfare costs across the four cohorts in our simulation sample. We begin by examining the predicted mean CESD depression score by age and cohort (see Fig. 8). In contrast to each of the younger cohorts, simulations suggest that the EHRS experienced a rising mean CESD score during their sixties. However, based on currently available data, our model predicts that all cohorts have realized or will realize a rising mean CESD score over much of their seventies and eighties. In general, these trends are consistent with the U-shaped pattern over age found in previous studies (Mirowsky and Ross, 1992; Sutin et al., 2013; Tampubolon and Maharani, 2017; Abrams and Mehta, 2019). After the late-eighties, CESD scores are predicted to fall for all cohorts. In terms of levels, the model predicts that after their mid-sixties, mean depression scores will be lower among War Babies and Baby Boomers than the early or late HRS cohorts.

Table 3 reports the estimated costs of late-life depression by cohort. When only considering the direct health utility penalty of depression (depression only model), the expected loss in QALYs is slightly smaller for the younger cohorts. For example, the average expected QALYs lost falls from 0.85 for the EHRS cohort to 0.81 for Baby Boomers. Likewise, the willingness-to-pay to avoid depression falls from 8.4% to 7.5% of annual consumption. A similar general pattern of falling average costs of depression over cohorts remains when adjusting for spillover effects on other modeled outcomes (full model). For example, the expected QALYs lost falls from 2.06 to 1.93 between the EHRS and Baby Boomer cohorts. Similarly, the expected annual consumption gain from depression falls from $631 to $497. There is also a slightly higher gain in labor supply for younger cohorts. In terms of our CV welfare metric, fully-adjusted willingness-to-pay falls from 14.8% to 12.8% of annual consumption. This amounts to a fall in the average expected loss of lifetime consumption from $89,405 in the EHRS cohort to $77,338 among Baby Boomers.

While we estimate falling *average* costs of depression over cohorts, the final two row of Table 3 reveals another important trend. When looking at the distribution within a cohort, we see that the inequality of depression costs is rising. For example, the Gini coefficient on our fully-adjusted CV measure of welfare has increased from 0.28 in the EHRS cohort to 0.34 for

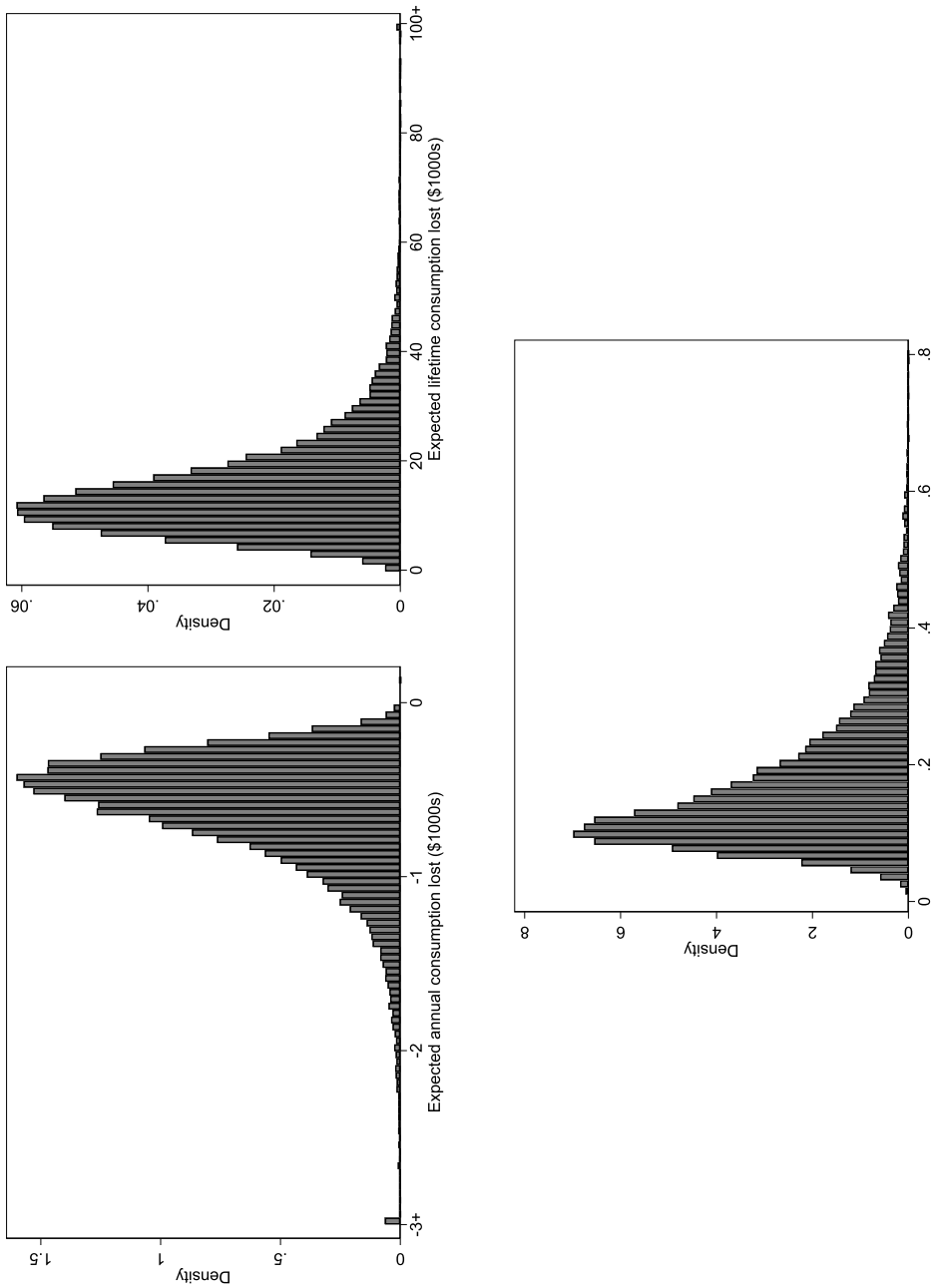

Fig. 6. Expected consumption and labor supply loss after age sixty (full model). *Notes*: Estimates use base year respondent analysis weights. Labor supply lost conditional on working at age sixty.

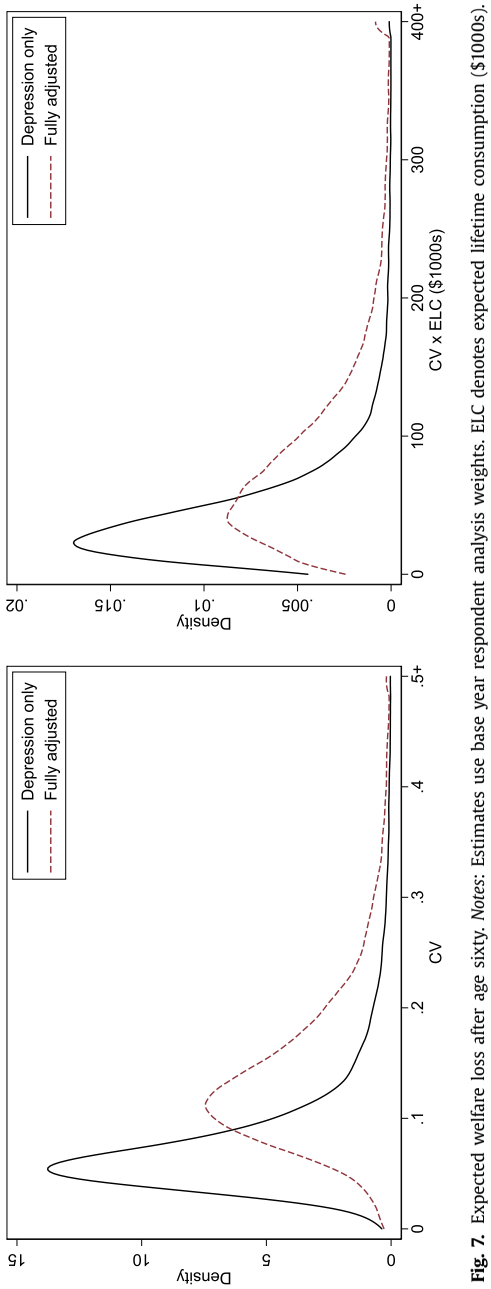

Fig. 7. Expected welfare loss after age sixty. *Notes*: Estimates use base year respondent analysis weights. ELC denotes expected lifetime consumption ($1000s).

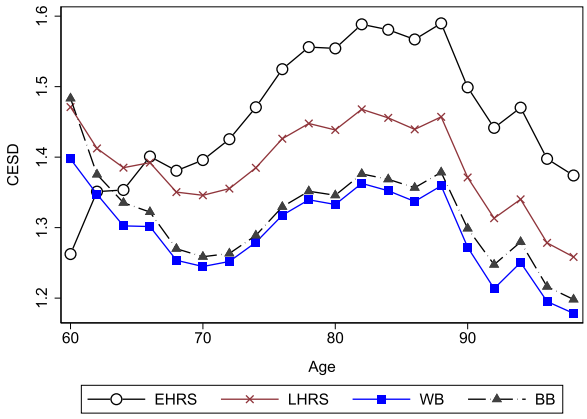

Fig. 8. Expected CESD by age and cohort. *Notes*: Expected CESD score (0-8 scale) conditional on survival.

Baby Boomers. Thus, while the average cost has decreased slightly across cohorts, the welfare burden of depression has become significantly more unequal *within* cohorts. The final row sheds further light on these changes by showing that the CV measure at the ninetieth percentile of the distribution has largely held steady across cohorts. In other words, falling costs of depression over cohorts have not been realized by the most severely affected, even though some improvements appear to have accrued overall.

One potential explanation for the falling average cost of depression over cohorts is the general rise in the use of depression treatments. For example, the share of the U.S. adult population using antidepressants increased from 7.7% in 1999 to 12.7% in 2014 (Pratt et al., 2017). This rise in treatment usage (or the introduction of more effective treatments) may have improved symptoms associated with moderate depression while remaining ineffective against more severe depression, explaining the patterns observed across cohorts in our estimates. Alternatively, it could be that antidepressant treatments are helping reduce severe depression as well, but prevalence of severe depression is simultaneously increasing at a comparable rate, hence leaving the welfare costs relatively constant across cohorts at the top end of the distribution.

3.5. Robustness

We examined the robustness of our key welfare numbers under several alternate modeling assumption. The online appendix provides robustness results for preference parameters $(\beta, \epsilon, \theta)$ and using alternate health utility weights. Here we examine the robustness of results to a more general form of flow utility for consumption and leisure given by:

$$u(c,l,h) = \phi(h)\left[\frac{c^{1-\gamma}}{1-\gamma}\left(1-(1-\gamma)\frac{\theta\epsilon}{1+\epsilon}(1-l)^{\frac{1+\epsilon}{\epsilon}}\right)^{\gamma} - \frac{\bar{c}^{1-\gamma}}{1-\gamma}\right] \tag{9}$$

which reduces to our benchmark case with $\gamma = 1$ and $\bar{c} = 3$.[19] With $\gamma > 1$ there is more curvature over consumption. These preferences follow those proposed by Trabandt and Uhlig (2011) and Jones and Klenow (2016) which maintain a constant Frisch elasticity of labor supply. We check robustness of results for curvatures up to $\gamma = 3.5$ as there have been a wide range of empirical estimates, with large curvatures arguably more plausible at older ages. Mean welfare results are reported in Table 4.

The first row of Table 4 provides our benchmark results for easy comparison. The second row examines the impact of assuming a higher value for the flow intercept $\bar{c}$. Specifically, we set $\bar{c} = 1.5$, implying that $1,500 of consumption is needed for a retiree to prefer life to death. This increases the median value of life to about $66,000 per QALY in the EHRS cohort. It also increases the estimated mean welfare cost of late-life depression in the EHRS from 8–15% to 11–21% of annual consumption. There are similar increases in welfare estimates for later cohorts and we continue to see a small decline in mean welfare costs across cohorts.

The remaining rows of Table 4 provides results with higher curvature values over consumption. As discussed by Murphy and Topel (2006), one problem that arises with higher curvature in this framework is that as γ rises, the implied value of life grows rapidly. In order to gain a sense of this issue, the first column in Table 4 shows the median value of life per QALY with higher curvatures. With $\gamma = 2$ and $\bar{c}$ held at its benchmark value, the median value of life is high but not completely implausible at $104,000 per QALY. The bound on the estimated mean welfare cost of late-life depression in the EHRS rises to 20–34% of annual consumption. When $\gamma = 3.5$, the value of life reaches about $300,000 per QALY and the welfare bound reaches 38–48%. Only three out of 23 value of life studies surveyed by Ryen and Svensson (2015) estimated a mean value of life over $150,000. The likely overstated value of life at higher curvatures suggests caution should be taken

[19] $\bar{c} = 3$ is equivalent to $\bar{u} = -log(3)$ as in our benchmark when $\gamma = 1$.

Table 4
Selected robustness results.

γ	$\bar{c}$	VOL	Depression only				Full model			
			EHRS	LHRS	WB	BB	EHRS	LHRS	WB	BB
1.0	3.00	46.81	0.084	0.083	0.077	0.075	0.148	0.144	0.134	0.128
1.0	1.50	66.06	0.119	0.117	0.110	0.110	0.212	0.207	0.194	0.190
1.5	3.00	72.87	0.139	0.140	0.132	0.122	0.254	0.253	0.238	0.220
2.0	3.00	104.10	0.209	0.212	0.199	0.180	0.341	0.342	0.324	0.295
3.0	3.00	211.91	0.344	0.354	0.336	0.297	0.457	0.466	0.447	0.403
3.5	3.00	299.34	0.384	0.398	0.380	0.335	0.482	0.494	0.476	0.428
1.5	4.84	46.81	0.094	0.095	0.089	0.080	0.178	0.177	0.167	0.149
2.0	5.49	46.81	0.115	0.118	0.110	0.095	0.204	0.206	0.193	0.168
3.0	5.97	46.81	0.165	0.174	0.162	0.135	0.248	0.257	0.242	0.204
3.5	6.01	46.81	0.186	0.198	0.185	0.152	0.260	0.273	0.257	0.216

Notes: Median value of life per QALY (in thousands of dollars) for EHRS cohort denoted by VOL. All other columns report mean CV (λ). Estimates use base year respondent analysis weights. War Babies denoted by WB and Baby Boomers by BB.

Table 5
Mean welfare costs of depression: additional counterfactuals.

	Depression only				Full model			
	EHRS	LHRS	WB	BB	EHRS	LHRS	WB	BB
Benchmark	0.084	0.083	0.077	0.075	0.148	0.144	0.134	0.128
Age sixty only	0.065	0.063	0.059	0.057	0.122	0.116	0.108	0.103
Clinical only	0.047	0.046	0.042	0.043	0.078	0.076	0.069	0.067

Notes: Estimates use base year respondent analysis weights. All results are mean CV (λ). Age sixty only experiment eliminates depression at sxity but allows depression to emerge after age sixty. Clinical only experiment estimates costs of a CESD score over three.

when interpreting robustness results with high (but empirically plausible) curvature values. In light of this concern, the final four rows show results at the same curvature values but with intercept $\bar{c}$ adjusted to maintain the same median value of life as our benchmark estimates. These results give us a sense of how sensitive welfare results are to higher values of life versus risk aversion to consumption fluctuations. For example, with $\gamma = 3.5$, the intercept rises to about $6000 and the welfare cost of late-life depression in the EHRS is about 18–26%. This is still larger than the benchmark but significantly smaller than welfare estimates with higher curvature and constant intercept. Overall, the higher curvature values provide a sense of the robustness of key results and the conservative nature of our benchmark welfare estimates.

3.6. Policy simulations

Finally, in this section we conduct several additional counterfactual simulations to gain a sense of policy lessons we might learn from our results. First, we estimate the impact of setting the CESD score to zero at age sixty but allowing depression to emerge after age sixty. This gives a sense of how results are driven by initial (age sixty) conditions compared to the development of depressive symptoms after age sixty. Table 5 provides welfare results over cohorts for this experiment. As expected, the welfare cost of depression at age sixty is lower than our benchmark which captures the cost from age sixty onward. In the EHRS cohort, for example, the fully-adjusted willingness-to-pay is 12.2% compared to 14.8% in the benchmark. So while the costs are lower, depressive symptoms at age sixty still explain a large share of the total costs. Comparing to the benchmark numbers across cohorts, age sixty depression explains roughly 75–80% of the total welfare costs of late-life depression. This suggests that targeting depression interventions earlier in the life-cycle may have substantial benefits later in life. Of course, this does not imply that depression cannot be addressed at older ages, only that early interventions could have significant dynamic benefits later in life.

It is also useful to consider how far policy could realistically go towards mitigating the costs of late-life depression through promoting diagnosis and treatment. Our results so far have concerned the costs associated with reporting any symptoms on the CESD depression scale. In practice, low level symptoms may be very hard to diagnose and/or treat and could to some extent even reflect noise in the data. It could be argued that broader population level policies could potentially address low level depressive symptoms, for example through promoting general health and social interactions among older populations. Nonetheless, narrowing in on clinically-relevant depressive symptoms may give a better sense of the costs of depression that could realistically be addressed with direct diagnosis and treatment policy interventions. The final row of Table 5 provides results when we ignore the costs of low level symptoms by setting the utility penalty to zero for CESD scores under four. We chose a cutoff of four as it roughly corresponds to clinically-relevant symptoms of depression or “caseness” in the HRS (Steffick, 2000). In all cohorts, clinically-relevant depressive symptoms account for 50–60% of the total cost of late-life depression. For example, based on the full model, the EHRS cohort would be willing to pay up to 7.6%

of annual consumption over remaining life to avoid any possibility of clinically-relevant depression (compared to 14.4% to avoid all depressive symptoms). So while low level symptoms are clearly important, the costs associated with clinically-relevant depression suggests substantial scope for direct policy intervention. Of course, it is worth noting that treatment inventions do not guarantee the mitigation of depression. For example, although anti-depressant medications can be effective in treating major depressive disorder, they fail to achieve remission in roughly a third of patients (Souery et al., 2006; Voineskos et al., 2020). Optimal strategies to deal with such treatment resistant depression is an active area of research, and our results suggest substantial potential welfare gains through continuing to improve treatment outcomes.

4. Conclusion

We estimated a panel VAR model of mental and physical health, labor supply, and consumption using longitudinal data from the Health and Retirement Study. We used the estimated model to repeatedly simulate life–cycle paths for older Americans, with and without the prevalence of late–life depressive symptoms, given a set of initial age sixty conditions. We estimated an average loss of labor supply of up to 1.1 months, lifetime consumption of up to $16,000, and quality–adjusted life expectancy of between 0.85 and 2.1 years per person in the EHRS birth cohort. Combining into a single welfare metric, we estimated a bound on the expected welfare loss of depression of 8–15% of annual consumption after age sixty. This amounts to the utility equivalent of an expected loss in lifetime consumption (or expected willingness-to-pay to avoid late-life depression) of approximately $46,000-$91,000 per person. In a hypothetical world populated by identical cohorts of size four million at age sixty, this produces a back-of-the-envelope estimate of aggregate welfare loss on the order of $180–360 billion annually. We also found substantial heterogeneity in the estimated cost with some individuals willing to give up well over 20% of annual consumption to avoid late-life depression. Moreover, while we found a small general decline in average costs over birth cohorts, the welfare burden of depression appears to have become significantly more unequal within cohorts.

From a policy perspective, our results suggest there are substantial potential benefits from reducing depression among older Americans. If enacted early enough, such policies could be workplace based. For example, consistent with broader recommendations of the CDC, there could be customized and intensive employee assistance programs (EAPs) for pre-retirees that include employee classes or seminars. A stronger focus could be put on information and referral services for employees with symptoms of depression, and the EAPs could be responsive to events, stressors, and changes in the lives of employees. Work or community based programs to promote health and social interactions for older adults could also be used as adjunct therapies in preventing and treating clinical depression or even low level depressive symptoms (Elmer and Stadtfeld, 2020). From the standpoint of the state, there could be more active steps to address depression and major depressive disorders among older adults by promoting public awareness, prescriptions and therapies to treat depression, and treatment centers. States could legislate and execute mental health crisis intervention training and other evidence-based programs for people most likely to encounter older adults on the job. In addition, states could subsidize the cost of mental health screenings, promote “telepsychology” (providing mental health services via remote technology), and offer incentives for providers to screen retired individuals. States could also review programs such as Medicaid to ensure that older adults living on fixed incomes have access to the drugs they need to treat their depression in the most effective manner. States could also take a lead from the federal government and require depression and other mental health conditions to be treated and covered at the same level as physical health conditions as is the case with the Affordable Care Act.

This study is not without limitations. Our estimates only include private costs of depression and do not capture public expenses (e.g., Medicare costs) or general equilibrium effects. We also do not capture potential spillover effects. For example, depression could have substantial costs for a partner or children. On the other hand, we have included lost lifetime consumption in our upper bound cost estimate. But if some of this consumption is shifted to children or charities through higher bequests, some of the cost may be recovered by society at large. Moreover, while our statistical model does well in replicating the observed patterns in the data, point estimates cannot be viewed as necessarily causal nor as adhering to any particular unobserved mechanism. This leaves us with only an estimated bound on feasible costs. Our compensating variation measure is also quantitatively sensitive to the choice of curvature in the utility function. Nonetheless, this study’s novelty is in estimation of a more comprehensive measure that incorporates life-cycle dynamics to improve our understanding of the welfare costs of late-life depression.

Supplementary material

Supplementary material associated with this article can be found, in the online version, at 10.1016/j.jebo.2022.10.001

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