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Dynamics of self-rated health and selective mortality

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Abstract

Self-rated health status (SRHS) is one of the most frequently used health measures in empirical health economics. This article analyzes the first seven waves of the Health and Retirement Study (HRS) and finds that (1) all available lags have decreasing but significant predictive power for current SRHS and (2) SRHS and future mortality are strongly related which leads to a specific selection problem known as survivorship bias. A parsimonious joint model with an autocorrelated latent health component in both the SRHS and the mortality equation is suggested. It is better able to capture the empirical facts than commonly used models including random effects and/or state dependence and better able to correct the survivorship bias than commonly used strategies such as inverse probability weighting.

Keywords

Self-rated health; Survivorship bias; Limited dependent variables models; Panel data

1 Introduction

Self-rated health status (SRHS) is one of the most frequently studied measures of individual health. While it is an obviously subjective health measure, it does have considerable objective content such as predictive power for mortality. Furthermore, it is very easy to collect and therefore available in many large-scale surveys.¹

Panel data permit a much richer analysis of the determinants of health than pure cross-sectional data and allow to account for unobserved heterogeneity and changes over time. Typical empirical studies of SHRS estimate a model with a simple specification for the correlation over time on a data set of the living respondents. This article discusses two issues regarding panel data models of SRHS and suggests a parsimonious model structure to overcome the problems of the commonly used approaches.

The first issue concerns the modeling of the intertemporal correlation structure. While it is obvious that individual SRHS is positively correlated over time conditional on observable characteristics, the strategies to explain and account for this fact vary in the literature. Contoyannis et al. (2004a) carefully discuss classical panel data models of SRHS. They distinguish state dependence from time-constant unobserved heterogeneity. State dependence refers to a causal effect of past *outcomes* on current outcomes. These kinds of models are common in labor economics, see for example Card and Sullivan (1988). While it

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¹Examples include the Panel Study of Income Dynamics (PSID), the Health and Retirement Study (HRS), the WHO World Health Survey, the British Household Panel Survey (BHPS), the English Longitudinal Study of Ageing (ELSA), the Survey of Health, Ageing and Retirement in Europe (SHARE), the German Socio-Economic Panel Study (GSOEP), the Centerpanel and many more.

is plausible to assume a causal link from yesterday's discrete outcome variable "labor market status" for example through human capital accumulation, the mechanism is different for the outcome variable SRHS: Where on the scale a respondent made a tick in a previous wave does most likely not causally affect the results of a later survey.² It is more natural to think about state dependence of the *actual health* for which the *reported health* is an imperfect proxy. For a related argument see Pudney (2008) who discusses the modeling of subjective wellbeing. Unobserved heterogeneity refers to individual differences not captured by observable characteristics. While the presence of unobserved heterogeneity in SRHS models is very plausible, it is less obvious why the idiosyncratic health differences should be constant over time.

A closer look at the first seven waves of the HRS³ reveals that the dynamic pattern of SRHS is incompatible with a combination of time-constant unobserved heterogeneity and (low-order) state dependence. This article suggests a somewhat different model specification which explains the intertemporal correlation pattern with state dependence of the underlying actual health y_{it}^* instead of SRHS y_{it} . The latter is modeled as a classical threshold crossing of y_{it}^* and is correlated over time since y_{it}^* is. The model can also be interpreted as unobserved heterogeneity which is correlated but not necessarily constant over time.

The second issue discussed in this article is selective mortality or survivorship bias (Jones et al. 2006). Health is obviously only observed for living respondents. Since SRHS is highly persistent and a strong predictor of mortality, cross-sectional age profiles necessarily confound individual profiles with an increasing selection of the relatively healthy survivors. The same holds for any potential determinant of health such as socio-economic status or policy interventions: Increases of health are potentially confounded by changes of the composition of survivors. To account for survivorship bias, this article proposes a joint dynamic model of SRHS and mortality and estimates it on the HRS sample. This allows to disentangle effects on health from selection effects through mortality. Unlike the approach of inverse probability weighting used by Contoyannis et al. (2004a) and Jones et al. (2006), the joint models allows for selection on unobservables which affect both SRHS and mortality. Simulations show that the model captures both the SRHS dynamics and mortality selection observed in the data well.

This article is structured as follows. Section 2 discusses SRHS, introduces the sample used for the empirical analyzes, and provides descriptive analyzes with a focus on SRHS and mortality. Section 3 discusses how to reconcile the findings with econometric panel data models. Based on this discussion, a joint model for SRHS and mortality is suggested. Results and simulations are presented in Section 4. Section 5 concludes.

2 SRHS: Background and descriptives

2.1 Self-rated health status

SRHS is collected in many surveys. The wording of the question used in the HRS is "Would you say your health is excellent, very good, good, fair, or poor?" Obviously, SRHS is a very subjective measure and prone to both random measurement error and potentially systematic reporting biases. Crossley and Kennedy (2002) find that many respondents report a different level of SRHS when answering the same question twice within one survey. Lindeboom and van Doorslaer (2004) discuss the effects of different scales of reference for self-reported

²SRHS could have additional explanatory power given actual health if respondents remember their previous answers and base their current responses on them.

³The HRS is sponsored by the National Institute of Aging (grant number NIA U01AG009740) and conducted by the University of Michigan.

measures. A different strand of literature studies the effect of health on labor supply and retirement decisions and finds effects of these decisions on the reporting of SRHS—an effect called justification bias, see for example Disney et al. (2006).

On the other hand, more objective health measures such as the prevalence of chronic conditions are error-ridden as well as long they are reported by the respondent, see Baker et al. (2004) and Butler et al. (1987). Despite its obvious drawbacks, SRHS has been found a useful and powerful measure. It maps the high-dimensional and complex concept of health into one dimension using individual perceptions and judgments. And it has been found to have real content, for example it is a strong predictor of other health measures (Gerdtham et al. 1999) and mortality (van Doorslaer and Gerdtham 2003).

With all its advantages and disadvantages, SRHS is one of the most widely studied measures of individual health in empirical health economics. Examples are the articles of Adams et al. (2003), Deaton and Paxson (1998), and Smith (1999).

2.2 Sample

The data used for the empirical example are from the HRS. For the analyzes presented here, the RAND HRS Data File (Version G) is used. It is a user-friendly data set produced by the RAND Center for the Study of Aging, with funding from the National Institute on Aging and the Social Security Administration.⁴

The HRS is a biannual panel survey and contains data on different cohorts of elderly Americans which entered the panel survey at different points in time. Table 1 gives an overview of these cohorts and shows the wave of the first interview, the number of individuals, and the number of observations and deaths in the working sample. It includes all individuals aged 50 or older in the initial interview and excludes individuals with missing information on one of the key variables used in this study. The distribution of these variables in the sample is shown in Table 2.

2.3 SRHS dynamics and mortality

The key issue studied in this article is the dynamics of the SRHS. Table 3 shows the transitions between two adjacent waves in the HRS data. SRHS is highly persistent— 47% of the respondents report the same level of health and more than 86% do not change their report by more than one “unit”. At the same time, SRHS is a strong predictor of mortality. A respondent who reported poor SRHS in one wave has ten times the risk to die before the next wave compared to someone who reported excellent SRHS.

Current SRHS has predictive power not only for the next wave, but also for the longer future. Figure 1a plots the share of the deceased during the next waves by SRHS in wave 1. While only 6.75% of the respondents who reported excellent SRHS in wave 1 have died by wave 7, this is true for 43.65% who reported poor SRHS in wave 1. The mortality risk differs in all future waves, so the lines keep diverging instead of becoming parallel at some point.

Figure 1b shows the share of respondents in poor or fair health among those who survive all seven waves by initial SRHS. The predictive power of SRHS in wave 1 decreases slowly over time but is still strong even for SRHS in wave 7.

⁴See <http://www.rand.org/labor/aging/dataproduct> for details.

The age profile of the share of respondents who report poor or fair SRHS is plotted as the solid line in Fig. 2. It rises with age except for the very old. As shown above, SRHS is highly correlated over time and a strong predictor of mortality. The age profile obviously only represents the living respondents, so it is a compound effect of individual development and selection due to differential mortality. The shorter lines in Fig. 2 illustrate this effect. They show the age profile only for those respondents who are observed living at least at age 60, 70, 80, 90, and 95. This takes out the selection effect from the first appearance in the sample through the respective age. These lines are much steeper and represent the pure effect of aging, whereas the difference to the solid line represents the selective mortality.

As already shown, SRHS in wave 1 has explanatory power for both SRHS and mortality in wave 7. The question Table 4 addresses is: does this explanatory power vanish once shorter lags and other variables are controlled for? It shows results from a reduced-form regression of SRHS and mortality on a set of sociodemographic variables and different numbers of lags of SRHS. The striking result is that even if six lags of SRHS are included so that only the seventh wave for the original HRS cohort is analyzed, the magnitude of the coefficients decreases with the lag length but all lags have significant explanatory power on their own. For mortality, the picture looks similar, but there are only 235 deaths observed between waves 6 and 7, so hardly any coefficients are estimated significantly different from zero. This is especially the case for the highly correlated lagged values of SRHS. With three lags, all previous SRHS states have a highly significant predictive power for mortality which decreases in magnitude with increasing lag length.⁵

In order to assess the quantitative relevance of these results, consider the SRHS specification with six lags (third column of Table 4) again. The results imply that a *ceteris paribus* change of SRHS lagged by 1 (2/3/4/5/6) waves from “excellent” to “poor” increases the risk of reporting “fair” or “poor” health by 55.6 (38.8/15.5/5.8/1.1/1.4) percentage points.

These results are very robust to different specifications. If instead of the linear specification of the SRHS lags four dummy variables are included for each lag, the results are harder to read and display in one table, but lead to the same qualitative findings. When collapsing the 5-point scale into a binary variable and analyzing it accordingly, the results are again unaffected. The same holds if the regressions are run separately by gender instead of including a gender dummy variable and if additional regressors such as marital status, individual earning and household income are added. These additional results can be requested from the author.

The next section discusses how all these findings can be explained by a parsimonious model.

3 Models of SRHS dynamics and mortality

This section discusses how the dynamics of SRHS and mortality can be modeled in a way which is consistent with the descriptive findings of Sect. 2. Section 3.1 ignores mortality for a moment and looks at SRHS of the survivors alone and Sect. 3.2 adds mortality to the preferred model.

⁵With a 5% significance level, the null hypothesis that all coefficients of the lags are equal is clearly rejected by a Wald test in all four regressions that include higher lags. In separate tests, any of the coefficients of higher lags is significantly different from the corresponding coefficient of the first lag in all these regressions. The equality of the coefficient of any *SRHS*, lag l and *SRHS*, lag $l - 1$ is rejected for the SRHS regression with three lags for all $l = 2, 3$ and for the regression with six lags for all $l = 2, 3, 5$. This suggests that the decrease of the coefficients is largely statistically significant. In the mortality regressions, this equality of coefficients is rejected for $l = 2$, but not for higher lags.

3.1 SRHS dynamics

To use a consistent notation, assume that SRHS is determined by a standard ordered response model. Individual $i = 1, \dots, N$ reports a SRHS $y_{it} \in \{1, \dots, 5\}$ at time $t = 1, \dots, T$ if a latent continuous variable y_{it}^* is between two thresholds:

$$y_{it}=j \iff \alpha_{j-1} \leq y_{it}^* < \alpha_j \text{ with } 1 \leq j \leq 5, \quad (1)$$

where $\alpha_0 = -\infty$, $\alpha_5 = \infty$, and $\alpha_1, \dots, \alpha_4$ are unknown model parameters.⁶

Let $\mathbf{x}_{it}$ denote a vector of strictly exogenous covariates and u_{it} a random error term which is independent over time and individuals and independent of $\mathbf{x}_{it}$. Consider four common types of models for y_{it}^* as discussed by Contoyannis et al. (2004a):

- (1) Independent $y_{it}^* = \mathbf{x}_{it}\beta + u_{it}$
- (2) State dependence $y_{it}^* = \mathbf{x}_{it}\beta + y_{i,t-1}\gamma + u_{it}$
- (3) Random effects (RE) $y_{it}^* = \mathbf{x}_{it}\beta + a_i + u_{it}$
- (4) State dependence and RE $y_{it}^* = \mathbf{x}_{it}\beta + y_{i,t-1}\gamma + a_i + u_{it}$

The independent model (1) implies that conditional on $\mathbf{x}_{it}$, there is no explanatory power of past values of SRHS which clearly violates the findings in Table 4, at least with the set of covariates used there.

Model (2) with state dependence assumes that the latent variable y_{it}^* is affected by the previous outcome $y_{i,t-1}$.⁷ This implies that the first lag of SRHS has explanatory power for today's SRHS, but not the longer lags. The findings in Table 4 disagree with that. Of course, this model can be changed to include higher-order lags, but the results show that one would need at least six lags and there is no indication that the seventh lag would be insignificant if it were available. A more structural interpretation of this model is not very plausible either. If the dependent variable were labor force participation, previous outcomes could affect today's outcomes causally by signaling or human capital depreciation. With SRHS, it is more likely that past actual health y_{it}^* instead of the reports y_{it} affects current health and therefore SRHS. The lags of y_{it} can be interpreted as proxies for y_{it}^* , but then γ does not have a structural interpretation and the explanatory power of such a model can be worse than that of a more parsimonious model including past actual health.

The REs model (3) is more plausible in this sense. It assumes that there is unobserved heterogeneity represented by the random variable a_i which is usually assumed to be independent of $\mathbf{x}_{it}$ as well as u_{it} . Individuals differ by factors beyond the ones captured by the covariates. This creates an additional correlation of the outcomes. In reduced-form regressions like those in Table 4, lagged outcomes are significant predictors since they contain information on a_i . The i.i.d. error term u_{it} might reflect temporary health shocks like a cold or just random influences on the response process like the current mood. They make the signal of lagged SRHS noisy so that all the lags have additional explanatory power. But

⁶The model could be generalized by allowing these thresholds to depend on observed characteristics of the respondents (Lindeboom and van Doorslaer 2004).

⁷Typically, one would actually include dummy variables for the different lagged outcomes of $y_{i,t-1}$ instead of a linear term.

since the REs are assumed to be constant over time, each lag tends to have the same amount of information and so the predictive power of all lags should not systematically vary. This contradicts the finding in Table 4 that the explanatory power decreases with the lag length.

Specification (4) is the preferred model of Contoyannis et al. (2004a). The combination of state dependence and a RE has the advantage that it introduces additional flexibility in the correlation structure over time. Compared to the RE model, the first lag is allowed to have additional predictive power due to the state dependence. While this fits the observed pattern in Table 4 better than the previous models, it still cannot capture the decreasing explanatory power of the higher lags.

Instead of the models discussed so far, consider a model with an AR(1) error component instead of the RE:

$$\begin{aligned} y_{it}^* &= \mathbf{x}_{it}\beta + a_{it} + u_{it} \\ a_{it} &= \rho a_{i,t-1} + e_{it}. \end{aligned} \quad (2)$$

For the random process a_{it} to be stationary, assume that the i.i.d. shocks e_{it} have a variance of $(1 - \rho^2)\sigma_a^2$ where σ_a^2 is the variance of a_{it} . In the special case $\rho = 1$, the a_{it} are constant over time and the model is equivalent to a RE model. This unobserved stochastic process can be interpreted as a latent health component. Similar to the RE model, this model implies that lagged SRHS values $y_{i,t-s}$ predict outcomes because they contain information on latent health in $t - s$, but the information becomes noisier with an increasing lag length because of the accumulated shocks $e_{i,t-s}$ through e_{it} . This can explain the decreasing explanatory power found in Table 4. Note that the REs model emerges as a special case of this model with $\rho = 1$. For a binary SRHS measure, Contoyannis et al. (2004b) discuss similar models with autocorrelated error terms. Pudney (2008) presents a related model of subjective wellbeing.

Stern (1994) discusses a method of simulated moments estimator for a similar model structure in which y_{it}^* depends on lagged values $y_{i,t-1}^*$ and gives SRHS as an example for which it is appropriate. For maximum likelihood estimation, the unobserved health component has to be integrated out of the conditional outcome probability numerically just as for nonlinear RE models. While a_i and therefore the integral is univariate for RE models, the sequence $a_{i1}, \dots, a_{iT}$ in this model is T -dimensional. Define $\mathbf{x}_i = [\mathbf{x}_{i1}, \dots, \mathbf{x}_{iT}]$, $\mathbf{y}_i = [y_{i1}, \dots, y_{iT}]$, and $\mathbf{a}_i = [a_{i1}, \dots, a_{iT}]$. Conditional on $\mathbf{x}_i$ and $\mathbf{a}_i$, the individual outcomes are independent over time. The conditional outcome probabilities are

$$\Pr(\mathbf{y}_i | \mathbf{x}_i, \mathbf{a}_i) = \prod_{t=1}^T \Pr(y_{it} | \mathbf{x}_{it}, a_{it}) \quad (3)$$

with $\Pr(y_{it} | \mathbf{x}_{it}, a_{it})$ representing time-specific outcome probabilities. With the assumption that u_{it} are i.i.d. logistic error terms, the probabilities are standard ordered logit probabilities for y_{it} with a_{it} as additional regressors. The evaluation of the likelihood function requires integrating the sequence of latent states out of this expression:

$$\Pr(\mathbf{y}_i | \mathbf{x}_i) = \int \dots \int \Pr(\mathbf{y}_i | \mathbf{x}_i, \mathbf{a}_i) f(\mathbf{a}_i | \mathbf{x}_i) da_{i1} \dots da_{iT}, \quad (4)$$

where $f(\mathbf{a}_i|\mathbf{x}_i)$ is the joint p.d.f. of $\mathbf{a}_i$. The easiest approach to approximate this integral is simulation: draw a number of vectors $\mathbf{a}_i^1, \dots, \mathbf{a}_i^R$ from this joint distribution, calculate $\Pr(y_i|\mathbf{x}_i, \mathbf{a}_i^r)$ for $r = 1, \dots, R$ and average the results. For a thorough discussion of estimation using approximation by simulation, see Hajivassiliou and Ruud (1994). An alternative is to use multivariate numerical integration algorithms such as Gaussian quadrature on sparse grids (Heiss and Winschel 2008).

Nonlinear Kalman filters represent an alternative method for approximation the likelihood function of models like the one at hand. Heiss (2008) discusses such an algorithm which is suitable for our purpose. It expresses the joint probability as the product of conditional time-specific probabilities

$$\Pr(y_i|\mathbf{x}_i) = \Pr(y_{i1}|\mathbf{x}_i) \Pr(y_{i2}|\mathbf{x}_i, y_{i1}) \cdots \Pr(y_{iT}|\mathbf{x}_i, y_{i1}, y_{i2}, \dots, y_{i,T-1}), \quad (5)$$

and approximates each of these terms in a sequential fashion using reweighted Gaussian quadrature. This algorithm can be a very accurate alternative to simulation (Heiss 2008).

Table 5 shows maximum likelihood results for the independent, REs, and latent AR(1) models for the HRS sample, separately by gender. The models are sequentially nested: The independent model follows from the RE and AR(1) model with $\sigma_a = 0$ and the RE is a special case of the AR(1) model with $\rho = 1$. Both restrictions are clearly rejected by Wald and likelihood ratio tests, so the AR(1) model fits the data significantly better than the other two models.⁸

In the AR(1) model, the idiosyncratic unobserved health component a_{it} has an estimated standard deviation of 3.0 and 2.9 for females and males, respectively. These are large numbers when compared to the i.i.d. error u_{it} which by normalization of the underlying

ordered logit model has a standard deviation of $\frac{\pi}{\sqrt{3}} \approx 1.8$. The estimated values of ρ are quite high with values of 0.941 and 0.946 for females and males, respectively, but significantly smaller than 1. This can be easily seen from likelihood ratio tests: The REs model is clearly rejected with LR test statistics of 949.8 and 779.8 for females and males, respectively. While the unobserved idiosyncratic health component is highly correlated over time, it is not constant as the RE model assumes.

Table 6 shows results for the models (2) and (4) which include lagged-dependent variables as regressors. Because no lagged values are known for the first observation of each individual, the estimates are based on the likelihood contributions conditional on y_{i1} for all the individuals. It is well known that this together with the RE included in model (4) leads to inconsistent parameter estimates due to the initial conditions problem (Heckman 1981). The distribution of the RE depends on y_{i1} and standard REs software ignores this dependence. As the results reported in Contoyannis et al. (2004a), the results for model (4) are based on the approach of Wooldridge (2005): instead of the usual RE assumption on the distribution of the RE, assume that the RE a_i conditional on $y_{i1} = j$ is normally distributed with mean μ_j and variance σ_a^2 so the conditional mean depends on the first observation. While this assumption is somewhat ad hoc, it is very convenient: the model is equivalent to a RE model with dummy variables for the initial value of the dependent variable as additional regressors.

⁸Note that $\sigma = 0$ and $\rho = 1$ are on the boundary of the parameter space. Therefore, usual LR tests are conservative, see Self and Liang (1987).

In order to allow a comparison, Table 6 also shows results of the same latent AR(1) model as discussed above, but where the estimation is also conditional on the initial observations. To see how this conditioning is implemented, consider the joint outcome probability conditional on y_{i1}

$$\Pr(\mathbf{y}_i|\mathbf{x}_i, y_{i1}) = \int \cdots \int \Pr(y_{i2}, \dots, y_{iT} | \mathbf{x}_i, \mathbf{a}_i) f(\mathbf{a}_i | \mathbf{x}_i, y_{i1}) da_{i1} \cdots da_{iT} \quad (6)$$

because conditional on the latent health states, the dependent variables are independent over time. Note that the conditional density of the latent health process is

$$f(\mathbf{a}_i | \mathbf{x}_i, y_{i1}) = f(a_{i1} | \mathbf{x}_i, y_{i1}) f(a_{i2} \dots a_{iT} | a_{i1}). \quad (7)$$

The joint density conditional on the initial value a_{i1} is implied by the AR(1) process assumed for latent health. The conditional distribution of a_{i1} can be expressed by Bayes' rule as

$$f(a_{i1} | \mathbf{x}_i, y_{i1}) = f(a_{i1} | \mathbf{x}_i) \frac{\Pr(y_{i1} | \mathbf{x}_i, a_{i1})}{\Pr(y_{i1} | \mathbf{x}_i)}. \quad (8)$$

This suggests to approximate the likelihood contribution using importance sampling: Given a number of R draws $a_i^1, \dots, a_i^R$ from the joint distribution characterized by $f(\mathbf{a}_i)$, calculate

$w_i^r = \frac{\Pr(y_{i1} | \mathbf{x}_i, a_{i1}^r)}{\sum_{s=1}^R \Pr(y_{i1} | \mathbf{x}_i, a_{i1}^s)}$ for all $r = 1, \dots, R$. The simulated likelihood contribution is then

$$\tilde{\Pr}(\mathbf{y}_i | \mathbf{x}_i, y_{i1}) = \sum_{r=1}^R w_i^r \Pr(y_{i2}, \dots, y_{iT} | \mathbf{x}_i, \mathbf{a}_i^r). \quad (9)$$

The same reweighting approach works accordingly if sequential Gaussian quadrature is used instead of simulation.

As the results in Table 6 show, the pure state dependency model (2) is clearly rejected by an LR test in favor of model (4) involving both state dependence and a RE. These models are not nested within the latent AR(1) model, so a straightforward test is infeasible. However, the latter model has both a better fit as measured by the log likelihood value and fewer parameters than the other models, so it would be favored by any likelihood-based criterion such as AIC or BIC.

The model parameters are not easy to interpret directly except for the signs and relative magnitudes. Before turning to substantive interpretations and simulations in Sect. 4, the next section discusses how to add selective mortality to the latent AR(1) model.

3.2 SRHS and mortality

The fact that healthy individuals have a higher life expectancy creates a selection effect which tends to attenuate true health differentials if it is ignored. This effect was

demonstrated for the age profile of SRHS in Sect. 2, but it works also for other effects. For example, the impact of a chronic disease on SRHS is underestimated if the selection through mortality is stronger for the chronically ill. And the impact of policy interventions on health is underestimated if its effect on mortality is ignored.

Contoyannis et al. (2004a) and Jones et al. (2006) attempt to correct for survivorship bias in a SRHS model using an inverse probability weighting estimator for a pooled ordered probit model. This approach has the disadvantages that it cannot account for unobserved heterogeneity in the SRHS model and that selection is assumed to be random conditional on covariates. Mortality is a special source of sample selection. The HRS allows to follow respondents over time and observe many of them die. Therefore, a joint model of SRHS and mortality can be estimated which corrects for survivorship bias based on all available information in a model-consistent way. The model presented in this section allows for the unobserved health component as discussed in the previous section. In addition to covariates, mortality is also allowed to be driven by this latent health variable.

Let d_{it} denote an indicator which has the value 1 if individual i dies at time t and 0 otherwise. Consider the following model which adds mortality to the AR(1) model of SRHS in Eqs. 1 and 2.

$$\begin{aligned}
 y_{it}^* &= \mathbf{x}_{it}\beta^y + \sigma^y a_{it} + u_{it}^y \\
 d_{it}^* &= \mathbf{x}_{it}\beta^d + \sigma^d a_{it} + u_{it}^d \\
 y_{it} = j &\iff \alpha_{j-1} \leq y_{it}^* < \alpha_j \text{ with } 1 \leq j \leq 5 \\
 d_{it} &= \mathbf{1}[d_{it}^* > \alpha^d] \\
 a_{it} &= \rho a_{i,t-1} + e_{it} \\
 a_{it} &\sim N(0, 1), \quad e_{it} \sim N(0, 1 - \rho^2)
 \end{aligned} \tag{10}$$

The SRHS part of this model corresponds to the AR(1) model discussed in Sect. 3.1. Mortality is modeled as a binary outcome which depends not only on the same set of covariates as SRHS but also on the same latent health process as SRHS. This generates a correlation of SRHS and mortality conditional on covariates and allows to capture selective mortality beyond the observed differences. While even extremely healthy individuals (in terms of covariates $\mathbf{x}_{it}\beta^d$ and the latent health component a_{it}) can die at any time if they draw an extreme shock u_{it}^d (say a car accident), unhealthy individuals can die from much less severe shocks.

Note that y_{it} is only observed if $d_{it} = 0$. Therefore, the correlation between the remaining transitory errors u_{it}^y and u_{it}^d is not identified and is assumed to be zero which is plausible since in this setup, they represent nonhealth-related shocks (such as car accidents and the mood during the interview). Since these shocks are transitory, this does not affect the intertemporal correlations.

This model allows a straightforward calculation of outcome probabilities given values of a_{it} . The conditional mortality risk is given by its binary choice probability. The joint probability of survival and SRHS of j is the product of the binary and the ordered choice probabilities. Similar models have proved to be useful for the study the trajectories of health and disability in old age (Heiss et al. 2007) and for the modeling of the dynamics of prescription drug use (Heiss et al. 2009).

The likelihood function can be approximated in the same fashion as for the SRHS model of Sect. 3.1. But the mortality selection has worked before the initial sampling. If a respondent

appears in the sample for the first time at age 90, she belongs to a relatively healthy part of the population. Even if the *initial* idiosyncratic health (say at birth or age 50) is independent of socioeconomic variables, differential mortality leads to a dependence of age and these characteristics with latent health *in the living population* (and therefore in the initial sample) at older ages. This has to be accounted for when calculating the likelihood function. Assuming no mortality selection before age 50, we have to condition on the outcome of no mortality until the age of initial sampling. This is the same problem as conditioning on the initial SRHS outcome as it was discussed above and the same strategy can be used. Implementations of this type of model in the software package Stata can be requested from the author.

4 Results and simulations

Parameter estimates for the joint model of SRHS and mortality presented in Sect. 3.2 are shown in Table 7. The latent health state a_{it} enters both the SRHS and the mortality equation highly significantly for females and males. This captures the fact that even when controlling for observable characteristics, these outcomes are correlated due to the common influence.

Because the estimated parameters have no direct quantitative interpretation, Table 8 presents estimated partial effects of the explanatory variables on the probability of poor or fair SRHS. As in all nonlinear models, these partial effects differ by the values of all explanatory variables. Therefore, these effects are evaluated for each sample member. The table shows averages over everybody. The first two columns present the results of the independent and AR(1) SRHS models without mortality correction. The numbers are quite similar except that the effects of age are larger in the AR(1) specification. This is because it partially captures individual changes over time and not only cross-sectional differences by age but also it is not as prone to survivorship bias as the i.i.d. model.

Columns 3–6 present results from the joint model and disentangle direct effects on SRHS and selection effects. The third are the partial effects on the mortality risk over the next 2 years and on poor or fair SRHS, respectively. Variables which increase the mortality risk like nonwhite, low education, and age implicitly also increase the survivorship bias since the respective populations are more positively selected. The pure effects on SRHS shown in the fourth column are therefore higher for these variables relative to the isolated SRHS model in column 2 in which they are confounded by the survivorship bias.

The fifth column presents the estimated survivorship bias implied by the estimated model which is shown to facilitate the comparison of the results of different models. The pure effect on SRHS and the selection effect are combined in the last column which now presents the estimated difference of the share poor or fair health among the survivors. As can be expected, this is pretty much in line with the results from the models without mortality correction.

For example, the fact that *ceteris paribus* fewer females are in poor or fair SRHS is pretty much offset by the lower mortality of women so that in the surviving population, SRHS hardly differs by gender. Selective mortality attenuates the SRHS differences by education since the low educated have both worse SRHS and a higher mortality risk. The highest differences can be seen in the age effects. Since both poor SRHS and mortality selection increase steeply with age, poor or fair SRHS increases much slower if only the surviving are analyzed and the survivorship bias is ignored as has already been observed in the data in Fig. 2.

The joint model of SRHS and mortality is very parsimonious—in addition to the slope parameters of the explanatory variables, only three parameters capture the dynamics of

SRHS and its relation to mortality. The following simulation exercises allow to check whether it is able to explain the full dynamics of the seven SRHS waves. The simulations are based on 1,000 artificial data sets. Each of those consists of individuals with the same socio-economics as the initial HRS sample. For each individual, a latent health, SRHS, and mortality process is simulated through wave 7 according to the model with the estimated parameters.

Table 9 repeats the descriptive regression of Table 4 (third column) of wave 7 SRHS on socio-demographics and six lags of SRHS. Besides the results for the HRS sample, these regressions were performed on each of the 1,000 artificial data sets generated according to the model with a latent AR (1) health component. The table shows means, standard deviations, the first, and the 99th percentile of these 1,000 point estimates. All HRS coefficients are well within these intervals except for the coefficient of the first lag which is slightly higher in the HRS sample than for the artificial data.

Table 10 shows results of a similar exercise. It repeats the regression of mortality on socio-demographics and three lags of SRHS from Table 4 (fifth column). The coefficients of the socio-demographic variables agree very well using the HRS and the artificial samples. The coefficients of lagged SRHS only agree qualitatively. The explanatory power of the first lag is higher and then decreases faster in the HRS sample when compared to the data set generated by the estimated model. But the model is able to reproduce the qualitative structure of significant but decreasing predictive power of lagged SRHS.

One possible explanation would be that there are actually two latent processes: one encompasses deadly health problems and affects both SRHS and mortality risk. A second one could be chronic but not deadly health problems and/or response styles which only affect SRHS but not mortality. Such a generalized model would allow to more flexibly explain the observed data.

Another way to compare the HRS and simulated samples is to reproduce Fig. 2 for the latter. Figure 3 shows the age profile of the respondents who (are simulated to) survive to the corresponding age. The profiles look very similar. Notably, the model is able to reproduce the decreasing share of living individuals in poor or fair SRHS beyond age 90 which it explains by survivorship bias. The level in the very high ages is somewhat lower in the simulated sample, but sample sizes are very low in this area.

The interesting question is whether the model can also explain the decomposition of the pure age and the selection effect depicted in Fig. 2. Figure 4 repeats these results and adds the simulated counterparts. To facilitate the comparison, the simulated SRHS levels are adjusted for the differences seen in Fig. 3. The results agree strikingly well except for a few outliers where sample sizes are small in the original HRS sample.

Overall the simulation results suggest that even the very simple and parsimonious model discussed in the previous section can explain most of the dynamics of SRHS and mortality seen in the first seven waves of the HRS.

The joint model of SRHS and mortality is very parsimonious and could be refined in many dimensions. In this article, the variance of the idiosyncratic health state as well as its correlation over time are constant parameters. It is likely that specifying it as a function of observed characteristics would improve the model fit. For example, at higher ages, health might be subject to larger shocks. Also, the model in its current form does not account for differences in reporting styles. To add these effects, similar approaches as discussed in the literature such as vignettes (Kapteyn et al. 2007) could be combined with the model. A more refined response model such as one in which the response threshold varies by

sociodemographics or past responses can easily be incorporated, see Lindeboom and van Doorslaer (2004).

5 Summary

SRHS is a frequently used measure of individual health in survey data. Despite its subjectiveness, it has considerable real content. For example, it is a strong predictor of mortality. This leads to a potential survivorship bias—effects of all kinds of determinants of health are confounded by their effect on selective mortality.

This article documents the dynamics of SRHS and mortality in the first seven waves of the HRS. It suggests that the findings are inconsistent with typically applied econometric models and suggests a parsimonious alternative. Simulations show that this model succeeds much better in capturing the dynamic structure of the data. They agree with all correlation patterns qualitatively and in most cases also quantitatively.

The strongest survivorship bias is found for the age profile, but also the SRHS differences by gender and education are attenuated by selective mortality.

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Fig. 1. Mortality and SRHS paths by initial SRHS. (data source: original HRS cohort with complete data on mortality status over all seven waves; 11,624 individuals)

Fig. 2. SRHS by age and survivor status at different ages

Fig. 3. Age profiles of poor/fair SRHS: HRS versus simulation

Fig. 4. Simulation: SRHS by age and survivor status at different ages

Sample: cohorts

Table 1

Cohort	Birth year	First wave	Indiv.	Obs. per indiv.	Deaths (%)	Total number of obs.	
						Alive	Deaths
AHEAD	Before 1924	2	8,294	4.0	54.06	33,073	4,484
CODA	1924–1930	4	2,380	3.4	17.35	8,115	413
HRS	1931–1941	1	13,131	5.4	14.71	71,261	1,932
WB	1942–1947	4	2,531	3.4	3.63	8,641	92
EBB	1948–1953	7	2,777	1.0	0.00	2,777	0
Total			29,113	4.3	23.77	123,867	6,921

The HRS row includes 113 “HRS/AHEAD overlap cases” who were born before 1924 but included in the initial HRS interview (wave 1)

AHEAD The Study of Assets and Health Dynamics Among the Oldest Old, CODA Children of Depression Age, HRS Initial Health and Retirement Study cohort, WB War Babies, EBB Early Baby Boomers

Table 2
Sample: descriptive statistics

	Observations	Percent
Total	123,867	100.0
Self-rated health		
Excellent	16,672	13.5
Very good	33,681	27.2
Good	37,541	30.3
Fair	23,908	19.3
Poor	12,065	9.7
Gender		
Male	53,526	43.2
Female	70,341	56.8
Age		
$50 \leq \text{age} \leq 60$	40,031	32.3
$60 < \text{age} \leq 70$	38,731	31.3
$70 < \text{age} \leq 80$	28,354	22.9
$80 < \text{age} \leq 90$	14,270	11.5
$90 < \text{age}$	2,481	2.0
Ethnicity		
Caucasian	93,744	75.7
Hispanic	9,947	8.0
Nonwhite, nonhispanic	20,176	16.3
Education		
Less than high school	40,724	32.9
High school	38,661	31.2
More than high school	44,482	35.9

Table 3

Wave-by-wave transitions: SRHS and mortality

SRHS	Observations	Next wave				
		1. Excellent	2. Very good	3. Good	4. Fair	5. Poor
1. Excellent	13,685	48.4	33.4	12.2	3.1	1.0
2. Very good	27,036	12.5	49.9	27.0	6.5	1.6
3. Good	29,728	3.7	20.9	48.0	18.4	4.2
4. Fair	18,868	1.4	6.3	23.4	43.7	14.9
5. Poor	9,752	0.6	1.8	7.1	23.7	44.2
Total	99,069	11.5	25.9	28.6	18.4	9.0
						6.6

Table 4
SRHS and mortality: reduced-form regressions on lagged SRHS

	SRHS, ordered logit			Mortality, binary logit		
Female	-0.024 (0.012)	-0.087 ** (0.017)	-0.100 * (0.048)	-0.464 ** (0.028)	-0.464 ** (0.038)	-0.779 ** (0.150)
Age spline, 50–59	0.016 ** (0.004)	-0.057 ** (0.013)		0.014 (0.016)	-0.059 (0.050)	
Age spline, 60–69	0.010 ** (0.002)	0.009 ** (0.003)	0.016 (0.010)	0.076 ** (0.007)	0.060 ** (0.010)	0.024 (0.034)
Age spline, 70–79	0.031 ** (0.003)	0.026 ** (0.004)	0.038 * (0.015)	0.077 ** (0.006)	0.088 ** (0.007)	0.050 (0.035)
Age spline, 80–89	0.017 ** (0.004)	0.018 ** (0.006)	-0.013 (0.071)	0.090 ** (0.006)	0.088 ** (0.007)	0.158 (0.095)
Age spline, 90+	-0.011 (0.013)	-0.008 (0.020)	-0.907 ** (0.145)	0.120 ** (0.011)	0.123 ** (0.014)	
Nonwhite	0.253 ** (0.017)	0.140 ** (0.024)	0.039 (0.067)	0.064 (0.038)	0.010 (0.053)	-0.072 (0.175)
Hispanic	0.310 ** (0.023)	0.184 ** (0.034)	0.203 * (0.095)	-0.371 ** (0.056)	-0.428 ** (0.078)	-0.534 * (0.270)
Education < HS	0.294 ** (0.016)	0.165 ** (0.022)	0.185 ** (0.065)	0.038 (0.034)	0.069 (0.047)	-0.014 (0.175)
Education > HS	-0.210 ** (0.015)	-0.100 ** (0.020)	0.004 (0.055)	-0.037 (0.037)	0.023 (0.050)	0.044 (0.180)
SRHS, lag 1	1.448 ** (0.010)	1.001 ** (0.014)	1.029 ** (0.042)	0.685 ** (0.015)	0.577 ** (0.026)	0.927 ** (0.119)
SRHS, lag 2		0.574 ** (0.014)	0.532 ** (0.040)		0.126 ** (0.024)	0.042 (0.103)
SRHS, lag 3		0.411 ** (0.012)	0.352 ** (0.039)		0.074 ** (0.023)	0.065 (0.095)
SRHS, lag 4			0.263 ** (0.038)			-0.185 * (0.092)
SRHS, lag 5			0.083 * (0.036)			0.004 (0.088)
SRHS, lag 6			0.130 ** (0.033)			0.147 (0.082)
Individuals	23,694	17,818	6,719	25,988	20,008	7,142
Observations	92,413	47,238	6,719	104,190	52,845	7,142
Deaths				6,525	3,603	235
Log likelihood	-111,015.8	-53,289.5	-7,110.9	-19,837.9	-10,651.6	-885.4

Standard errors (clustered by respondent) in *parentheses*

Different from 0 at * 5% and ** 1% significance level

Table 5

SRHS models: parameter estimates

	Females			Males		
	(1) Indep.	(3) RE	AR(1)	(1) Indep.	(3) RE	AR(1)
Age spline, 50–59	0.032 ^{**} (0.003)	0.081 ^{**} (0.005)	0.091 ^{**} (0.006)	0.041 ^{**} (0.004)	0.096 ^{**} (0.006)	0.106 ^{**} (0.007)
Age spline, 60–69	0.021 ^{**} (0.003)	0.046 ^{**} (0.004)	0.046 [*] (0.005)	0.019 ^{**} (0.003)	0.059 ^{**} (0.004)	0.059 ^{**} (0.006)
Age spline, 70–79	0.040 ^{**} (0.003)	0.086 ^{**} (0.005)	0.098 ^{**} (0.006)	0.048 ^{**} (0.004)	0.095 ^{**} (0.006)	0.108 ^{**} (0.007)
Age spline, 80–89	0.036 ^{**} (0.005)	0.109 ^{**} (0.006)	0.115 ^{**} (0.008)	0.023 ^{**} (0.006)	0.102 ^{**} (0.009)	0.106 ^{**} (0.011)
Age spline, 90+	–0.036 ^{**} (0.012)	0.041 ^{**} (0.014)	0.045 [*] (0.018)	–0.093 ^{**} (0.020)	–0.066 [*] (0.028)	–0.080 [*] (0.034)
Nonwhite	0.656 ^{**} (0.019)	1.191 ^{**} (0.057)	1.351 ^{**} (0.065)	0.476 ^{**} (0.023)	0.825 ^{**} (0.063)	0.936 ^{**} (0.072)
Hispanic	0.764 ^{**} (0.026)	1.341 ^{**} (0.077)	1.518 ^{**} (0.088)	0.478 ^{**} (0.030)	0.837 ^{**} (0.083)	0.935 ^{**} (0.095)
Education < HS	0.740 ^{**} (0.018)	1.131 ^{**} (0.053)	1.327 ^{**} (0.061)	0.544 ^{**} (0.021)	0.830 ^{**} (0.060)	0.992 ^{**} (0.069)
Education > HS	–0.446 ^{**} (0.017)	–0.760 ^{**} (0.052)	–0.877 ^{**} (0.060)	–0.507 ^{**} (0.019)	–0.844 ^{**} (0.059)	–0.980 ^{**} (0.068)
σ		2.453 ^{**} (0.019)	2.978 ^{**} (0.028)		2.365 ^{**} (0.021)	2.901 ^{**} (0.032)
ρ			0.946 ^{**} (0.002)			0.941 ^{**} (0.002)
Individuals	16,273	16,273	16,273	12,840	12,840	12,840
Observations	70,341	70,341	70,341	53,526	53,526	53,526
Log likelihood	–102,265.5	–86,756.3	–86,281.4	–78,601.7	–67,827.5	–67,437.6

Standard errors (clustered by respondent) in *parentheses*

Different from 0 at * 5% and ** 1% significance level

Table 6

SRHS models with state dependence

Females				Males			
(2) State	(4) State	Latent		(2) State	State	Latent	
Depend.	Dep. + RE	AR(1)		Depend.	Dep. + RE	AR(1)	
Age spline, 50–59	0.009 (0.005)	0.048** (0.006)	0.111** (0.011)	0.032** (0.006)	0.079** (0.008)	0.162** (0.013)	
Age spline, 60–69	0.012** (0.003)	0.021** (0.004)	0.063** (0.006)	0.008* (0.003)	0.026** (0.004)	0.081** (0.007)	
Age spline, 70–79	0.029** (0.003)	0.038** (0.005)	0.115** (0.007)	0.036** (0.004)	0.044** (0.006)	0.126** (0.008)	
Age spline, 80–89	0.019** (0.005)	0.055** (0.008)	0.124** (0.009)	0.015* (0.007)	0.049** (0.010)	0.129** (0.012)	
Age spline, 90+	0.002 (0.015)	−0.010 (0.023)	0.061** (0.019)	−0.048* (0.022)	−0.064 (0.035)	−0.052 (0.036)	
Nonwhite	0.279** (0.021)	0.317** (0.040)	0.878** (0.117)	0.213** (0.027)	0.263** (0.049)	0.704** (0.127)	
Hispanic	0.335** (0.030)	0.425** (0.058)	1.169** (0.162)	0.281** (0.035)	0.443** (0.067)	1.230** (0.165)	
Education <HS	0.326** (0.021)	0.349** (0.038)	1.162** (0.110)	0.234** (0.024)	0.236** (0.043)	0.833** (0.121)	
Education > HS	−0.207** (0.019)	−0.260** (0.034)	−0.761** (0.110)	−0.239** (0.023)	−0.295** (0.039)	−0.815** (0.119)	
SRHS (lag) exc.	−1.570** (0.038)	−0.610** (0.041)		−1.379** (0.040)	−0.546** (0.044)		
SRHS (lag) good	1.369** (0.025)	0.518** (0.030)		1.251** (0.028)	0.457** (0.034)		
SRHS (lag) fair	2.848** (0.034)	1.122** (0.046)		2.660** (0.039)	1.075** (0.052)		
SRHS (lag) poor	4.443** (0.051)	1.865** (0.070)		4.369** (0.061)	1.968** (0.084)		
SRHS (1st) exc.		−1.257** (0.051)			−1.065** (0.054)		
SRHS (1st) good		1.003** (0.041)			0.917** (0.045)		
SRHS (1st) fair		2.126** (0.058)			1.914** (0.066)		
SRHS (1st) poor		3.319** (0.090)			3.057** (0.103)		
Cut point 1	−1.539** (0.043)	−1.771** (0.059)	−2.588** (0.163)	−1.294** (0.056)	−1.354** (0.077)	−1.797** (0.189)	
Cut point 2	0.738** (0.041)	1.016** (0.059)	0.730** (0.162)	0.789** (0.055)	1.164** (0.077)	1.217** (0.189)	
Cut point 3	2.770** (0.044)	3.484** (0.063)	3.641** (0.163)	2.790** (0.058)	3.569** (0.082)	4.080** (0.191)	
Cut point 4	4.807** (0.050)	5.916** (0.071)	6.479** (0.166)	4.788** (0.063)	5.922** (0.091)	6.838** (0.195)	
σ		1.246** (0.026)	2.888** (0.030)		1.196** (0.030)	2.768** (0.033)	

	Females			Males		
	(2) State	(4) State	Latent	(2) State	State	Latent
	Depend.	Dep. + RE	AR(1)	Depend.	Dep. + RE	AR(1)
ρ			0.945 ^{**} (0.002)			0.939 ^{**} (0.003)
Individuals	13,634	13,634	13,634	10,316	10,316	10,316
Observations	54,068	54,068	54,068	40,686	40,686	40,686
Parameters	17	22	15	17	22	15
Log likelihood	-64, 453.4	-62, 325.6	-62, 228.7	-49, 756.4	-48, 293.8	-48, 180.5

Standard errors (clustered by respondent) in *parentheses*
Different from 0 at * 5% and ** 1% significance level

Table 7
Joint SRHS and mortality model: parameter estimates

	Females		Males	
	SRHS	Mortality	SRHS	Mortality
Age spline, 50–59	0.101** (0.006)	0.049* (0.022)	0.122** (0.008)	0.035 (0.022)
Age spline, 60–69	0.065** (0.006)	0.109** (0.010)	0.082** (0.006)	0.097** (0.009)
Age spline, 70–79	0.136** (0.006)	0.103** (0.008)	0.159** (0.008)	0.115** (0.008)
Age spline, 80–89	0.168** (0.008)	0.140** (0.008)	0.175** (0.011)	0.136** (0.009)
Age spline, 90+	0.155** (0.018)	0.167** (0.013)	0.051 (0.034)	0.155** (0.021)
Nonwhite	1.477** (0.074)	0.457** (0.057)	1.040** (0.084)	0.316** (0.061)
Hispanic	1.459** (0.099)	−0.012 (0.089)	0.858** (0.109)	−0.105 (0.091)
Education <HS	1.575** (0.070)	0.458** (0.053)	1.215** (0.082)	0.267** (0.057)
Education > HS	−0.941** (0.066)	−0.236** (0.058)	−1.109** (0.078)	−0.346** (0.060)
Latent health (σ)	3.172** (0.031)	0.942** (0.028)	3.152** (0.036)	0.929** (0.029)
ρ	0.949** (0.002)		0.944** (0.002)	
Individuals	16,337		12,857	
Observations (mortality)	79,460		61,695	
Observations (SRHS)	70,341		53,526	
Log likelihood	−97, 129.5		−77, 676.6	

Table 8

Average partial effects (percentage points)

SRHS models		SRHS and mortality model				
Indep.	AR(1)	Effect on		Selection effect	SRHS and selection	
		Mortality	SRHS			
Gender: reference = male						
Female	0.58	-0.57	-2.07	-2.64	2.55	-0.08
Race: reference = nonhispanic white						
Nonwhite	11.51	11.53	1.43	13.26	-1.93	11.33
Hispanic	12.86	12.53	-0.19	10.36	2.18	12.54
Education: reference= high school degree						
Education < HS	14.04	12.28	1.37	15.49	-1.82	13.67
Education > HS	-8.06	-8.26	-0.93	-10.82	2.56	-8.26
Age: increase by 2 years, from current age						
50-59	1.10	1.50	0.13	1.98	-0.79	1.18
60-69	0.80	1.06	0.53	2.91	-2.28	0.63
70-79	1.75	2.16	1.18	7.94	-5.87	2.07
80-89	1.20	2.33	2.59	13.95	-12.28	1.67
90+	-2.32	0.18	2.14	24.55	-28.43	-3.87
Total	1.09	1.58	0.82	5.46	-4.30	1.17

The *numbers* show the ceteris paribus change of the probability of poor or fair health, averaged over the sample. They are obtained by counterfactual simulation

Table 9
Reduced-form SRHS regression: HRS versus simulated samples

	HRS sample		Simulated data		
	Est.	SE	Mean	SD	[1%; 99%]
Female	-0.099	(0.048)	-0.060	(0.046)	[-0.172; 0.054]
Age spline, 60–69	0.016	(0.010)	0.002	(0.010)	[-0.022; 0.026]
Age spline, 70–79	0.040	(0.015)	0.042	(0.015)	[0.007; 0.078]
Age spline, 80–89	-0.052	(0.080)	-0.005	(0.063)	[-0.153; 0.158]
Nonwhite	0.040	(0.067)	0.129	(0.066)	[-0.019; 0.285]
Hispanic	0.197	(0.095)	0.133	(0.091)	[-0.061; 0.364]
Education < HS	0.184	(0.065)	0.139	(0.062)	[-0.006; 0.283]
Education > HS	0.004	(0.055)	-0.111	(0.057)	[-0.237; 0.012]
SRHS, lag 1	1.029	(0.042)	0.822	(0.031)	[0.752; 0.892] ^a
SRHS, lag 2	0.531	(0.040)	0.493	(0.031)	[0.421; 0.562]
SRHS, lag 3	0.351	(0.039)	0.300	(0.031)	[0.230; 0.375]
SRHS, lag 4	0.263	(0.038)	0.190	(0.033)	[0.116; 0.268]
SRHS, lag 5	0.084	(0.036)	0.125	(0.031)	[0.050; 0.195]
SRHS, lag 6	0.129	(0.033)	0.096	(0.030)	[0.026; 0.170]

^aEst. parameter for HRS $\nsubseteq$ simulated [1%; 99%] interval

Table 10
Reduced-form mortality regression: HRS versus simulated samples

	HRS sample		Simulated data		
	Est.	SE	Mean	SD	[1%; 99%]
Female	-0.464	(0.038)	-0.453	(0.036)	[-0.535; 0.370]
Age spline, 50–59	-0.059	(0.050)	0.020	(0.050)	[-0.093; 0.136]
Age spline, 60–69	0.060	(0.010)	0.081	(0.010)	[0.059; 0.106]
Age spline, 70–79	0.088	(0.007)	0.076	(0.007)	[0.058; 0.093]
Age spline, 80–89	0.088	(0.007)	0.091	(0.008)	[0.074; 0.110]
Age spline, 90+	0.123	(0.014)	0.118	(0.013)	[0.090; 0.147]
Nonwhite	0.010	(0.053)	0.083	(0.048)	[-0.025; 0.199]
Hispanic	-0.428	(0.078)	-0.307	(0.072)	[-0.468; -0.141]
Education < HS	0.069	(0.047)	0.025	(0.044)	[-0.088; 0.120]
Education > HS	0.023	(0.050)	-0.036	(0.048)	[-0.151; 0.067]
SRHS, lag 1	0.577	(0.026)	0.344	(0.021)	[0.289; 0.389] ^a
SRHS, lag 2	0.126	(0.024)	0.224	(0.021)	[0.175; 0.274] ^a
SRHS, lag 3	0.074	(0.023)	0.169	(0.022)	[0.116; 0.217] ^a

^aEst. parameter for HRS $\nsubseteq$ simulated [1%; 99%] interval