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Volume Title: Perspectives on the Economics of Aging

Volume Author/Editor: David A. Wise, editor

Volume Publisher: University of Chicago Press

Volume ISBN: 0-226-90305-2

Volume URL: http://www.nber.org/books/wise04-1

Conference Date: May 17-20, 2001

Publication Date: June 2004

Title: Healthy, Wealthy, and Wise? Tests for Direct Causal Paths between Health and Socioeconomic Status

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URL: http://www.nber.org/chapters/c10350

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Healthy, Wealthy, and Wise?
Tests for Direct Causal Paths between Health and Socioeconomic Status

Peter Adams, Michael D. Hurd, Daniel McFadden, Angela Merrill, and Tiago Ribeiro

This chapter consists of four components: (1) the paper *Healthy, Wealthy and Wise? Tests for Direct Causal Paths between Health and Socioeconomic Status* by Peter Adams, Michael D. Hurd, Daniel McFadden, Angela Merrill, and Tiago Ribeiro, which originally was presented at the conference and then appeared in the *Journal of Econometrics,* Vol. 112: (2003); (2) a new addendum that describes updates in data and analysis since its publication; (3) additional appendix tables; and (4) the authors' response to comments on the paper.

11.1 Introduction

11.1.1 The Issue

The links between health, wealth, and education have been studied in a number of populations, with the general finding that higher socioeconomic status (SES) is associated with better health and longer life.[1] In a survey of

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We gratefully acknowledge financial support from the National Institute on Aging through a grant to the NBER Program Project on the Economics of Aging. Tiago Ribeiro acknowledges support from scholarship PRAXIS XXI/BD/16014/98 from the Portuguese Fundação para a Ciência e a Tecnologia. We thank Laura Chioda, Victor Fuchs, Rosa Matzkin, James Poterba, and Jim Powell for useful comments.

1. See Backlund, Sorlie, and Johnson (1999); Barsky et al. (1997); Bosma et al. (1997); Chandola (1998, 2000); Davey-Smith, Blane, and Bartley (1994); Drever and Whitehead (1997); Ecob and Smith (1999); Elo and Preston (1996); Ettner (1996); Feinstein (1992);

this literature, Goldman (2001) notes that this association has been found in different eras, places, genders, and ages, and occurs over the whole range of SES levels, so that it is not linked solely to poverty. The association holds for a variety of health variables (most illnesses, mortality, self-rated health status, psychological well-being, and biomarkers such as allostatic load) and alternative measures of SES (wealth, education, occupation, income, level of social integration).[2] There has been considerable discussion of the causal mechanisms behind this association, but there have been relatively few natural experiments that permit causal paths to be definitively identified.[3] In this paper, we test for the *absence* of direct causal links in an elderly population by examining whether *innovations* in health and wealth in a panel are influenced by features of the historical state.

Figure 11.1 depicts possible causal paths for the health and SES innovations that occur over a short period. An individual's life history is built from these period-by-period transitions. First, low SES may lead to failures to seek medical care and delay in detection of conditions, reduced access to medical services, or less effective treatment.[4] Also, increased risk of health problems may result from increased stress or frustration, or increased exposure to environmental hazards, that are associated with low

Fitzpatrick et al. (1997); Fitzpatrick and Dollamore (1999); Fox, Goldblatt, and Jones (1985); Goldblatt (1990); Haynes (1991); Hertzman (1999); Humphries and van Doorslaer (2000); Hurd (1987); Hurd and Wise (1989); Kaplan and Manuck (1999); Karasek et al. (1988); Kitawaga and Hauser (1973); Lewis et al. (1998); Leigh and Dhir (1997); Luft (1978); Marmot et al. (1991); Marmot, Bobak, and Davey-Smith (1995); Marmot et al. (1997); Martin and Preston (1994); Martin and Soldo (1997); McDonough et al. (1997); Murray, Yang, and Qiao (1992); Power, Matthews, and Manor (1996); Power and Matthews (1998); Rodgers (1991); Ross and Mirowsky (2000); Schnall, Landsbergis, and Baker (1994); Seeman et al. (2002); Shorrocks (1975); Stern (1983); Wadsworth (1991); Whitehead (1988); Wilkinson (1998); and Woodward et al. (1992).

2. The associations can become more complex when multiple health conditions and multiple SES measures are studied. Competing risks may mask the hazard for late-onset diseases; for example, elevated mortality risk from cardiovascular diseases in low SES groups may induce an apparent reverse relationship between SES and later-onset cancer in the surviving population (Adler and Ostrove 1999). Longer-run measures of SES such as education, occupation, and wealth appear to have a stronger association with health status than current income (Fuchs 1993). Using carefully measured wealth, we find that it explains most of the association with health, and education conditioned on wealth is not systematically correlated with health.

3. Papers examining explicit causal mechanisms include Chapman and Hariharran (1994); Dohrenwend et al. (1992); Evans (1978); Felitti et al. (1998); Fox, Goldblatt, and Johnson (1985); Goldman (1994); Kelley, Hertzman, and Daniels (1997); and McEwen and Stellar (1993).

4. There may be an important distinction between direct causal mechanisms influencing mortality, conditioned on health status, and direct causal mechanisms influencing onset of health conditions. For mortality, an SES gradient could be due to differentially effective treatment of acute health conditions. For morbidity, an SES gradient could reflect differentials in prevention and detection of health conditions. These involve different parts of the health care delivery system, and differ substantially in the importance of individual awareness and discretion, and allocation of costs between Medicare and the individual.

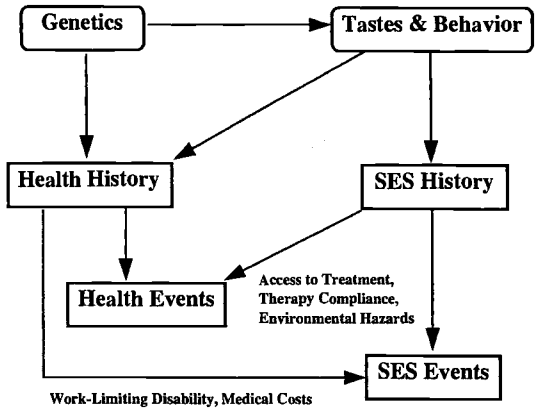


Fig. 11.1 Possible causal paths for SES and health

SES.[5] These factors could provide direct causal links from SES history to health events. Second, poor health may reduce the ability to work or look after oneself, and increase medical care expenditures, leading to reduced income and less opportunity to accumulate assets. This could provide a direct causal link from health to changes in SES.

There may also be hidden *common factors* that lead to ecological association of health and SES. For example, unobserved genetic heterogeneity may influence both resistance to disease and ability to work. Causal links may be reinforced or confounded by behavioral response. Behavioral factors such as childhood nutrition and stress, exercise, and smoking may influence both health and economic activity level. For example, tastes for work and for "clean living," whether genetic or learned, may influence both health and earnings. Finally, rational economic decision making may induce robust consumers to accumulate in order to finance consumption over a long-expected retirement, or unhealthy individuals to spend down assets.

Preston and Taubman (1994), Smith and Kington (1997a, b), and Smith (1998, 1999) give detailed discussions of the various causal mechanisms that may be at work and the role of behavioral response from economic consumers. The epidemiological literature (Goldman 2001) uses a different terminology for the causal paths: Links from SES to health innovations are termed *causal mechanisms,* while links from health to SES are termed *selection* or *reverse causation* mechanisms. Apparent association due to measurement errors, such as overstatement of the SES of the healthy or under-

5. For example, industrial and traffic pollution, and poor dwelling ventilation are risk factors for lung disease, and housing prices and household income are negatively correlated with air pollution levels in census data (Chay and Greenstone 1998).

detection of illnesses among the poor, are called *artifactual mechanisms.*[6] This literature classifies all common factors in terms of their implicit initial action as either causal or selection mechanisms.[7]

11.1.2 This Study

We study the population of elderly Americans aged seventy and older, and in this population test for the *absence* of direct causal paths from SES to innovations in health, and from health status to innovations in SES. These hypotheses will in general be accepted *only* if no causal link is present *and* there are no persistent hidden factors that influence both initial state and innovations. Rejection of one of these hypotheses does not demonstrate a direct causal link, because this may be the result of common hidden factors. However, in an elderly population persistent hidden factors will often be manifest in observed covariates, so that once these covariates are controlled, the residual impact of the hidden factors on innovations will be small. For example, genetic frailty that is causal to both health problems and low wages, leading to low wealth, may be expressed through a health condition such as diabetes. Then, onset of new health conditions that are also linked to genetic frailty may be only weakly associated with low wealth, once diabetic condition has been entered as a covariate. Thus, in this population, *rejection* of the hypotheses *may* provide useful diagnostics for likely causal paths.

The objectives and conclusions of this paper are limited. We study only elderly Americans, for whom Medicare provides relatively homogeneous and comprehensive health care at limited out-of-pocket cost to the individual. This population is retired, so new health problems do not impact earnings. Statements about the presence or absence of direct causal mechanisms in this population, given previous health and SES status, say nothing about the structure of these mechanisms in a younger population, where associations of health and SES emerge as a result of some pattern of causation and operation of common factors.

Our tests for the *absence* of causality do *not* address the question of how to identify invariant models and causal links when these tests fail. If a test

6. Consider phenomena such as underestimation of the hazard of a disease due to competing risks from other illnesses or death. In an unfortunate discrepancy in terminology, economists would call this a selection effect, while epidemiologists would classify it as an artifactual mechanism rather than a selection mechanism.

7. Usually, one can argue that observed association must originate from some initial causal action so that common factors originate from some initial direction of causation. However, there is no apparent initial causal action for genetically linked conditions such as Down syndrome, which increase mortality risk and preclude work. Further, as a practical matter, it is often impossible to make observations at the high frequencies that would be required to identify causal chains when feedbacks are nearly instantaneous. Then common factors will appear at feasible levels of detection to operate simultaneously, and their true causal structure will not be identified. For these reasons, there would be considerable merit in adding *common mechanisms* to the epidemiologist's classification.

for the absence of a direct causal path is rejected, it may be possible through natural or designed experiments to separate causal and ecological effects; see Angrist, Imbens, and Rubin (1996), Heckman (2000, 2001). Suppose a strictly exogenous variable is causal to SES, and clearly not itself directly causal to health or causal to common factors. Then, an association of this variable with innovations in health conditions can only be through a direct causal link from SES to health. A variable with these properties is termed a *proper instrument* or *control variable* for SES. Proper instruments are hard to find. They can be obtained through designed experiments, where random treatment assignment precludes the possibility of confounding by common factors, provided recruitment and retention of experimental subjects does not reintroduce confounding. For example, an experiment that randomly assigned co-payment rates and coverage within Medicare for prescription drugs or assisted living could provide evidence on direct causal links from SES to health conditions, provided attrition and compliance are not problems. Occasionally, natural experiments may provide random treatment assignment. Economic events that impact individuals differently and that are not related to their prior SES or health are potentially proper instruments. For example, a tax change that affects wealth differently in different states is arguably a proper instrument, as is a change in mandated Medicaid coverage that has a differential impact across states. Individual events such as receipt of inheritances may be proper instruments, although they would be confounded if they are anticipated, or if the probability of their occurrence is linked to health status; see Meer, Miller, and Rosen (2001). Weak association between SES and a proper instrument for it makes it difficult to obtain precise estimates of direct causal effects; see Staiger and Stock (1997).

Section 11.2 of this paper discusses the foundation for econometric causality tests, and sets out the models for the dynamics of health and SES that will be used for our analysis. Section 11.3 describes the panel study and data that we use. Section 11.4 describes the association of SES and prevalence of health conditions in the initial wave of the panel. Section 11.5 analyzes incidence of new health conditions, and presents tests for noncausality of SES for the incidence of health innovations. Section 11.6 tests for the absence of a causal link from health conditions to wealth accumulation and other SES indicators. Section 11.7 uses our estimated models for prevalence and incidence to simulate life histories for a current population aged seventy under counterfactual (and unrealistically simplistic) interventions that assume a major health hazard can be removed, or SES shifted for the entire population. This simulation accounts consistently for comorbidities and competing hazards over the life course. The purpose of this exercise is to demonstrate the feasibility of using our modeling approach for policy applications when the models pass the causality tests described in next section. Finally, section 11.8 gives conclusions and outlines

topics for future research. The appendix to this paper, containing tables 11A.1 to 11A.11 with detailed estimation results and the data and computer routines we use, are posted on the internet at http://elsa.berkeley.edu/wp/hww/hww202.html.

11.2 Association and Causality in Panel Data

11.2.1 Testing Causality

The primary purpose of this study is to test for direct causal links between SES and health. There is a large literature on the nature of causality and the interpretation of "causality tests."[8] Our analysis fits generally within the approach of Granger (1969), Sims (1972), and Hoover (2001), but our panel data structure permits some refinements that are not available in a pure time series setting.

Let Y_t denote a K-vector of demographic, health, and socioeconomic random variables for a household at date t and interpret a realization of these variables as an observation in one wave of a panel survey. Let $\mathbf{Y}_t$ be the information set containing the history of this vector through date t. Let

$$(1)\quad f(Y_t \mid \mathbf{Y}_{t-1}) \equiv f_1(Y_{1t} \mid \mathbf{Y}_{t-1}) \cdot f_2(Y_{2t} \mid Y_{1t}, \mathbf{Y}_{t-1}) \cdot \ldots \cdot f_K(Y_{Kt} \mid Y_{1t}, \ldots, Y_{K-1,t}, \mathbf{Y}_{t-1})$$

denote a model of the conditional distribution of Y_t given $\mathbf{Y}_{t-1}$. Without loss of generality, we have written this model as a product of one-dimensional conditional distributions, given history and given components of Y_t determined previously. Writing the model in this way does not imply that the components of Y_t form a causal chain, as they may be simultaneously determined, or determined in some causal sequence other than the specified sequence. However, the model structure simplifies if the current components of Y_t in the specified order do form a causal chain or are conditionally independent. If one takes Wold's view that causal action takes time, then for sufficiently brief time intervals, $f_K(Y_{Kt} \mid Y_{1t}, \ldots, Y_{K-1,t}, \mathbf{Y}_{t-1})$ will not depend on contemporaneous variables, and what Granger calls *instantaneous causality* is ruled out. In practice, time aggregation to observation intervals can introduce apparent simultaneous determination. Conversely, in applications where time aggregation is an issue, one can treat observed variables as indicators for some latent causal chain structure defined for very short time intervals.

8. See Dawid (2000); Freedman (1985, 2001); Granger (1969); Sims (1972); Zellner (1979); Swert (1979); Engle, Hendry, and Richard (1983); Geweke (1984); Gill and Robins (2001); Heckman (2000, 2001); Holland (1986, 1988); Pearl (2000); Robins (1999); Sobel (1997, 2000); Hendry and Mizon (1999); and Woodward (1999).

We shall focus on first-order Markov processes, specializations of (1) in which only the most recent history conveys information,

$$
\begin{aligned}
(2) \qquad f(Y_t \mid \boldsymbol{Y}_{t-1}) &\equiv f(Y_t \mid Y_{t-1}) \\
&\equiv f_1(Y_{1t} \mid Y_{t-1}) \cdot f_2(Y_{2t} \mid Y_{1t}, Y_{t-1}) \\
&\quad \cdot \ldots \cdot f_K(Y_{Kt} \mid Y_{1t}, \ldots, Y_{K-1,t}, Y_{t-1}).
\end{aligned}
$$

Note that if (1) is a higher-order Markov process, then (2) can be obtained by expanding the variables in Y_t to include higher-order lags. Greater generality could be achieved via a hidden Markov structure in which the observed Y_t are deterministic functions of a latent first-order Markov process.[9] We leave this extension for future research.

Model (2) is *valid* for a given history $\boldsymbol{Y}_{t-1}$ if it is the true conditional distribution of Y_t given this history. Term f a *structural* or *causal* model, or a (*probabilistic*) *law,* for Y_t *relative* to a family of histories if it has the *invariance* property that it is valid for each history in the family. Operationally, this means that within specified domains, f has the *transferability* property that it is valid in different populations where the marginal distribution of Y_{t-1} changes, and the *predictability* or *invariance under treatments* property that it remains valid following policy interventions that alter the marginal distribution of Y_{t-1}. By including temporal or spatial variables in Y, it is possible to weaken invariance requirements to fit almost any application. Done indiscriminately, this creates a substantial risk of producing an "overfitted" model that will be invalid for any "out-of-sample" policy interventions. Then, proposed models should be as generic as possible. However, it may be necessary in some applications to model "regime shifts" that account for factors that are causal for some populations or time periods, and not for others.

Suppose the vector $Y_t = (H_t, S_t, X_t)$ is composed of subvectors H_t, S_t, and X_t, which will later be interpreted as health conditions, SES status, and strictly exogenous variables, respectively. We say that S is *conditionally noncausal* for H, given X, if $f(H_t \mid H_{t-1}, X_{t-1})$ is a valid model; that is, given H_{t-1} and X_{t-1}, knowledge of S_{t-1} is *not needed* to achieve the invariance properties of a causal model. Conversely, if $f(H_t \mid H_{t-1}, S_{t-1}, X_{t-1}) \not\equiv f(H_t \mid H_{t-1}, X_{t-1})$, then knowledge of S_{t-1} *contributes to the predictability* of H_t. Note that either one or both conditional noncausality of S for H and conditional noncausality of H for S may hold. If either holds, then H and S can be arrayed in a (block) causal chain, and if both hold, then H and S are conditionally independent. Writing model (2) as a product of univariate conditional

9. Any discrete-time stationary stochastic process can be approximated (in distribution for restrictions to a finite number of periods) by a first-order hidden Markov model so there is no loss of generality in considering only models of this form; see Kunsch, Geman, and Kehagias (1995).

probabilities $f_i(H_{it} \mid H_{1t}, \ldots, H_{i-1,t}, H_{t-1}, S_{t-1}, X_{t-1})$, one can test for conditional noncausality of S for each component H_i. It is possible to have a causal chain in which S is conditionally causal to a previous component of H, and this component is in turn "instantaneously" causal to H_j, yet there is no direct causal link from S to H_i. Placing S after H in the vector Y, we have conditional probabilities $f(S_t \mid H_t, H_{t-1}, S_{t-1}, X_{t-1})$. There may be instantaneous conditional independence of H and S, with the conditional distribution of S_t not depending on H_t or conditional noncausality of H for S, with the conditional distribution not depending on H_{t-1}, or both. The statement that X is *strictly exogenous* in a valid model (2) is equivalent to the condition that H and S are conditionally noncausal for X in this model.[10]

The conventional definition of a *causal model* or *probabilistic law* requires that f be valid for the universe of possible histories (except possibly those in a set that occurs with probability zero); see Pearl (2000). It is possible to reject statistically a proposed causal model by showing that it is highly improbable that an observed sample with a given history was generated by this model. It is far more difficult using statistical analysis to conclude inductively that a proposed model is valid for the universe of possible histories. We have the far more limited objective of providing a foundation for policy analysis, where it is the invariance property under policy interventions that is crucial to predicting policy consequences. We have defined *validity* and *noncausality* as properties of a *model,* and of the outcomes of a *process* of statistical testing that could in principle be conducted on this model. Only within the domain where the model is valid, and invariance confirms that the model is accurately describing the true data generation process, can these limited positivistic model properties be related to the causal structure embedded in the true data generation process. Further, we can choose the domain over which invariance will be tested to make the definition operational and relevant for a specific analysis of policy interventions. Similarly, our definition of conditional noncausality is a positivistic construct in the spirit of the purely statistical treatment of "causality" by Granger (1969), and the test we will use is simply Granger's test for the absence of causality, augmented with invariance conditions. Thus, for example, if our analysis using this framework concludes that SES is not conditionally causal for new health events within the domain where the Medicare system finances and delivers health care, then this finding would support the conclusion that policy interventions in the Medicare system to increase access or reduce out-of-pocket medical expenses will not alter the conditional probabilities of new health events, given the health histories of enrollees in this system. It is unnecessary for this policy purpose to answer

10. Econometricians have traditionally used the term *strictly exogenous* to refer to properties of variables in the true data generation process, a stronger nonpositivistic version of this condition.

the question of whether the analysis has uncovered a causal structure in any deeper sense. Econometric analysis is better matched to the modest task of testing invariance and noncausality in limited domains than to the grander enterprise of discovering universal causal laws. However, our emphasis on invariance properties of the model, and on tests for Granger causality within invariant families, is consistent with the view of philosophers of science that causality is embedded in "laws" whose validity as a description of the true data generation process is characterized by their invariance properties; see Pearl (2000), Feigl (1953), and Nozick (2001).

11.2.2 Some Specific Formulations

Starting from class (2), we consider operational models of the linear latent variable form

$$Y^*_{it} = Y_{1,t}\alpha_{1i} + \ldots + Y_{i-1,t}\alpha_{i-1,i} + Y'_{t-1}\beta_i + \delta_i - \sigma_i\varepsilon_{it} \tag{3}$$

with

$$Y_{it} = \psi_i(Y^*_{it}, Y_{t-1}), \tag{4}$$

where Y^* is a latent variable, ε_{it} is an unobserved disturbance that is standard normal and independent across i and t, and ψ is a partial observability mapping that depends on the latent variable, and possibly on the lagged variables. For example, for a chronic health condition such as diabetes, Y_{it} will indicate whether there has ever been a diagnosis of the disease, with $Y_{it} = \max\{Y_{i,t-1}, \mathbf{1}(Y^*_{it} \geq 0)\}$. For an acute condition such as a heart attack, $Y_{it} = \mathbf{1}(Y^*_{it} \geq 0)$ indicates a new occurrence. Components of Y may be binomial or ordered discrete variables such as health status, or continuous variables such as household income. In this model, the α's, β, δ, and σ are parameters; restrictions are imposed as necessary for identification. In (3), the linearity in variables and parameters, the first-order Markov property, and the triangular dependence of Y^*_{it} on previous components of Y_t are not, in themselves, particularly restrictive, as one can approximate any continuous Markov model of form (2) by a form (3) in which Y_t is expanded to include transformations and interactions to sufficient order. The normality assumption is also not restrictive in principle. A latent random variable with conditional cumulative distribution function (CDF) $F(Y^*_{1t} \mid Y_{t-1})$ and the partial observability transformation (4) can be redefined using the standard normal CDF Φ as $Y^{**}_{1t} = \Phi^{-1}(F(Y^*_{1t} \mid Y_{t-1}))$ and $Y_{1t} = \psi_1(F^{-1}(\Phi(Y^{**}_{1t}) \mid Y_{t-1}), Y_{1,t-1})$; this gives a version of models (3) and (4) in which the disturbance is standard normal.[11] The same construction can be

11. For the CDF F of a random variable Y, define $F(y-) = \sup_{y'<y} F(y')$ and $F^{-1}(p) = \min\{x' \mid F(x') \geq p\}$. Define the random variable $Z \equiv h(Y) = \Phi^{-1}(F(Y-) + U[F(Y) - F(Y-)])$, where U is a uniform (0, 1) random variable. Then Z is a.s. standard normal. Define $Y^* = F^{-1}(\Phi(Z))$. Then, $Y^* = Y$ a.s., so that Y is given a.s. by a nondecreasing transformation of a standard normal random variable.

applied to the remaining components of Y_t. The causal chain assumption is innocuous when the time interval is too short for most causal actions to operate, and the components of Y_t are conditionally independent. However, the causal chain assumption is a more substantive restriction when the time interval is long enough for multiple events to occur, as it precludes even the feedbacks that would appear in multiple iterations of a true causal chain. Of course, the generally nonrestrictive approximation properties of models (3) and (4) do not imply that a particular specification chosen for an application is accurate, and failures of tests for invariance can also be interpreted as diagnostics for inadequate specifications.

In models (3) and (4), a binomial component i of Y_t with the partial observability mapping $\max\{Y_{i,t-1}, \mathbf{1}(Y^*_{it} \geq 0)\}$ and the identifying restriction $\sigma_i = 1$ satisfies $Y_{it} = 1$ if $Y_{i,t-1} = 1$, and otherwise is one with the probit probability

$$(5)\quad f_i(Y_{it} = 1 \mid Y_{1t}, \ldots, Y_{i-1,t}, Y_{t-1}) = \Phi(Y_{1,t}\alpha_{1i} + \ldots + Y_{i-1,t}\alpha_{i-1,i} + Y'_{t-1}\beta_i + \delta_i).$$

Analogous expressions can be developed for ordered or continuous components.

11.2.3 Measurement Issues

A feature of the panel we use is that the waves are separated by several years, and the interviews within a wave are spread over many months, with the months between waves differing across households. If model (2) applies to short intervals, say months, then the transition from one wave in month t to another in month $t + s$ is described by the probability model

$$(6)\qquad f(Y_{t+s} \mid Y_t) = \sum_{Y_{t+1},\ldots,Y_{t+s-1}} f(Y_{t+1} \mid Y_t) \cdot \ldots \cdot f(Y_{t+s} \mid Y_{t+s-1}).$$

Direct computation of these probabilities will generally be intractable, although analysis using simulation methods is possible.

A major additional complication in our panel is that interview timing appears to be related to health status, with household or proxy interviews delayed for individuals who have died or have serious health conditions. This introduces a spurious correlation between apparent time at risk and health status that will bias estimation of structural parameters. To study empirical approximations to (6) and corrections for spurious correlation, we consider a simple model of interview delay. Let $p = \Phi(\alpha + \beta x)$ denote the monthly survival probability for an individual who was alive at the previous wave interview, where x is a single time-invariant covariate that takes the value –1, 0, +1, each with probability 1/3. Counting from the time of the previous wave interview, let k denote the number of months this individual lives, and c denote the month that interviews begin for the current

wave.[12] There is a distribution of initial contact times; let q denote the probability of a month passing without being contacted, and let m denote the month of initial contact. Assume that an individual who is living at the time of initial contact is interviewed immediately, but for individuals who have died by the time of initial contact, there is an interview delay, with r denoting the probability of an additional month passing without a completed interview with a household member or proxy. Let n denote the number of months of delay in this event. Assume that m and n are not observed, but the actual interwave interval t, equal to m if the individual is alive at time of initial contact, and equal to $m + n$ otherwise, is observed. The density of k is $p^{k-1}(1 - p)$ for $k \geq 1$. The density of m is $q^{m-c}(1 - q)$ for $m \geq c$. Let d be an indicator for the event that the individual is dead at the time of initial contact. The probability of t and $d = 0$ is $h(0, t) = q^{t-c}(1 - q)p^t$, the product of the probability of contact at t and the probability of being alive at t. The probability of t and $d = 1$, denoted $h(1, t)$, is the sum of the probabilities that the individual is dead at an initial contact month m, with $c \leq m \leq t$, and the subsequent interview delay is $n = t - m$, or

$$h(1, t) = \sum_{m=c}^{t} (1 - p^m)q^{m-c}(1 - q)r^{t-m}(1 - r). \tag{7}$$

If $r < pq$, then $h(1, t) = (1 - q)(1 - r)\{(q^{t-c+1} - r^{t-c+1})/(q - r) - p^c((pq)^{t-c+1} - r^{t-c+1})/(pq - r)\}$. Then, the probability of an observed interwave interval t is $h(t) = h(0, t) + h(1, t)$, and the conditional probability of $d = 1$, given t, is $P(1 \mid t) = h(1, t)/h(t)$. Absent interview delay, the conditional probability of $d = 1$ given t would be simply $p^{t-1}(1 - p)$. The parameter values $\alpha = -2.47474$, $\beta = 0.3$, $c = 22$, $q = 0.85$, and $r = 0.5$ roughly match our panel. For these values, the median interwave interval is 25.5 months, and at $t = 34$, 87 percent of the interviews have been completed.

Figure 11.2 plots the inverse normal transformations of the true death rate $p^{t-1}(1 - p)$ and the apparent death rate $P(1 \mid t)$ against $\log(t)$ for each value of the covariate x. The *true* relationship is to a reasonable empirical approximation linear in x and in $\log(t)$. Then, in the absence of interview delay, one could approximate p, given x, with reasonable accuracy by estimating a probit model for death of the form $\Phi(\theta + \gamma x + \lambda \log(t))$, and then estimating p using the transformation $p = (1 - \Phi(\theta + \gamma x + \lambda \log(t)))^{1/t}$ for the observed interwave interval t.[13] However, the figure shows that interview delay induces a sharp gradient of apparent mortality hazard with interwave interval, so that an estimated model will not extrapolate to realistic mortality hazards over shorter periods. A simple imputation of time at

12. It does not matter for the example if the previous wave interview month is fixed or has a distribution, provided the *relative* interwave interval c is fixed, and current wave outcomes are independent of the timing of the previous wave interview.

13. A Box–Cox transformation of time at risk, $z = 4(t^{1/4} - 1)$, gives a somewhat better approximation in the probit model than $\log(t)$, but has no appreciable effect on the accuracy of estimated monthly transition probabilities.

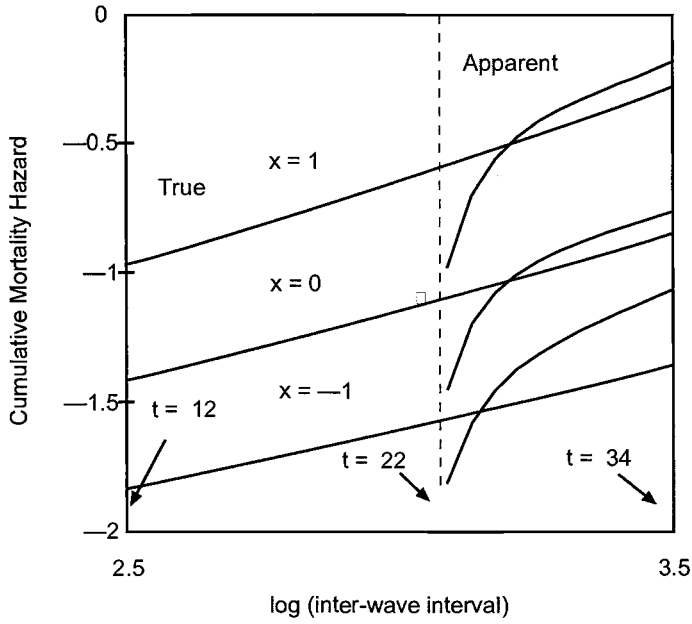


Fig. 11.2 Effect of interview delay (cumulative mortality hazard = Φ^{-1} [death probability])

risk up to initial contact leads again to models that work well with the procedure just outlined for estimation of p conditioned on x. A simple imputed time of initial contact for those who have died is the observed interwave interval less the difference in the mean interwave interview times for dead and living respondents. This imputation can be adjusted further so that the extrapolated annual death rate, $\Phi(\theta + \lambda \log(12) + \gamma x)$, matches the sample average mortality rate. We do the additional adjustment for our panel, with results that are almost identical to the simple imputation of time of initial contact.

We conducted a Monte Carlo calculation of the approximations above in a sample of 50,000. In this simulation, the empirical approximation to observed mortality in the absence of interview delay is $\Phi(-3.1053 + 0.5327x + 0.652 \log(t))$. With interview delay and the simple imputation described above, $\Phi(-2.9900 + 0.5349x + 0.6174 \log(t))$ is the empirical approximation.[14] Table 11.1 gives the annual mortality rates implied by these approximations. From these results, we conclude first that in the absence of interview delay, the probit model $\Phi(\theta + \lambda \log(t) + \gamma x)$ provides an adequate approximation to exact annual mortality rates as a function of time

14. The model estimated with interview delay and without imputation, is $\Phi(-4.6639 + 0.5349x + 1.1178 \log(t))$.

Table 11.1 Approximation Accuracy with Interview Delay (%)

x	Exact Annual Mortality Rate	Approximate Annual Mortality Rate without Interview Delay	Error	Approximate Annual Mortality Rate with Interview Delay and Imputed Contact Time	Error
0	7.71	7.90	2.41	7.93	2.79
–1	3.27	3.06	–6.55	3.07	–6.03
+1	16.41	16.71	1.80	16.75	2.05
Avg.	9.13	9.22	0.98	9.25	1.34

Note: The approximate annual mortality rate is given by $1 - (1 - \Phi(\theta + \gamma x + \lambda \log(t)))^{12/t}$, where t is the exact or imputed initial contact time and the model is estimated from the data generated by the Monte Carlo experiment.

at risk, across values of the covariate x that substantially change relative risk, and second that this remains true in the presence of interview delay when one imputes the initial contact time for dead subjects.

These conclusions on the accuracy of the approximation should extend to the exact interwave transition probabilities (6) in our Markov model, supporting use of the probit approximation

$$(8) \quad f_i(Y_{i,t+s} = 1 \mid Y_{1t+s}, \ldots, Y_{i-1,t+s}, Y_t) = \Phi(Y_{1,t+s}\alpha_{1i} + \ldots + Y_{i-1,t+s}\alpha_{i-1,i} + Y_t'\beta_i + \delta_i + \lambda_i \log(s)),$$

where $s = t_{i2} - t_{i1}$ is the imputed months between initial contact for a wave and previous wave interview, for estimation of incidence between waves. For simulation of yearly transitions, we use the approximation

$$(9) \quad \begin{aligned} f_i(Y_{i,t+12} = 1 \mid Y_{1t+12}, \ldots, Y_{i-1,t+12}, Y_t) &= 1 - (1 - \Phi(Y_{1,t+1}\alpha_{1i} + \ldots + Y_{i-1,t+1}\alpha_{i-1,i} + Y_t'\beta_i + \delta_i + \lambda_i \log(s)))^{12/s} \\ &\approx \Phi(Y_{1,t+1}\alpha_{1i} + \ldots + Y_{i-1,t+1}\alpha_{i-1,i} + Y_t'\beta_i + \delta_i + \lambda_i \log(s))12/s, \end{aligned}$$

where s is the median interwave interval, and the final approximation holds when the probability of a transition is small. Formula (9) generalizes to any probability of a transition from the status quo, with the probability of remaining at the initial state defined so that all the transition probabilities sum to one. We expect this formula to approximate well the probabilities of no new health conditions in a sample population over periods corresponding to the observed interwave intervals.

Estimation of models based on (2)–(8) is straightforward. Because of the independence assumption on the disturbances and the absence of common parameters across equations, the estimation separates into a probit, ordered probit, or ordinary least-squares regression for each component of Y, depending on whether the partial observability mapping is binary,

ordered, or linear. Conventional likelihood ratio tests can be used for the significance of explanatory variables.

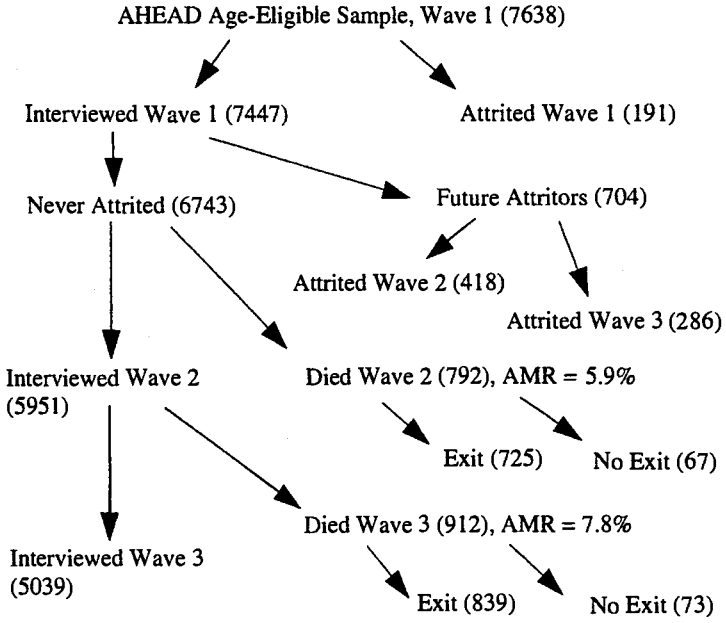
11.3 The AHEAD Panel Data

11.3.1 Sample Characteristics

Our data come from the Asset and Health Dynamics among the Oldest Old (AHEAD) study.[15] This is a panel of individuals born in 1923 or earlier and their spouses. At baseline in 1993 the AHEAD panel contained 8,222 individuals representative of the noninstitutionalized population, except for oversamples of blacks, Hispanics and Floridians. Of these subjects, 7,638 were over age sixty-nine; the remainder were younger spouses. There were 6,052 households, including individuals living alone or with others, in the sample. The wave 1 surveys took place between October 1993 and August 1994, with half the total completed interviews finished before December 1993. The wave 2 surveys took place approximately twenty-four months later, between November 1995 and June 1996, with half the total completed interviews finished by the beginning of February 1996. The wave 3 surveys took place approximately twenty-seven months after that, between January 1998 and December 1998, with half the total completed interviews finished near the beginning of March 1998. In each wave, there was a long but thin tail of late interviews, heavily weighted with subjects who had moved, or required proxy interviews due to death or institutionalization. Subjects never interviewed, directly or by proxy, are excluded from the calculation of the distribution of interview months. The AHEAD is a continuing panel, but it has now been absorbed into the larger Health and Retirement Study (HRS), which is being interviewed on a three-year cycle.

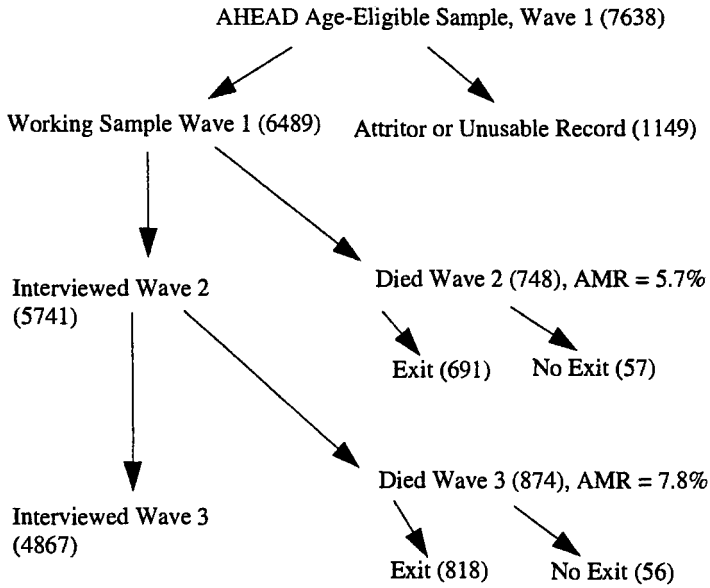
The AHEAD panel has substantial attrition, with death being the primary, but not the only, cause. A significant effort has been made to track attritors, and identify those who have died through the National Death Register. Figure 11.3 describes outcomes for the full age-eligible sample. For subjects where a proxy interview was possible, an "exit interview" gives information on whether decedents had a new occurrence of cancer, heart attack, or stroke since the previous wave. From the 6,743 age-eligible individuals who did not attrit prior to death, we formed a working sample for analysis consisting of 6,489 by excluding 254 additional individuals with critical missing information. Figure 11.4 describes their outcomes. In a few cases, attritors in wave 2 rejoined the sample in wave 3, but we treat these as permanent attritors because the missing interview makes the observation unusable.

15. The AHEAD survey is conducted by the University of Michigan Survey Research Center for the National Institute on Aging; see Soldo et al. (1997).



AMR = Crude annual mortality rate, Exit = Proxy interview following death

Fig. 11.3 Age-eligible sample outcomes



AMR = Crude annual mortality rate, Exit = Proxy interview following death

Fig. 11.4 Working sample outcomes

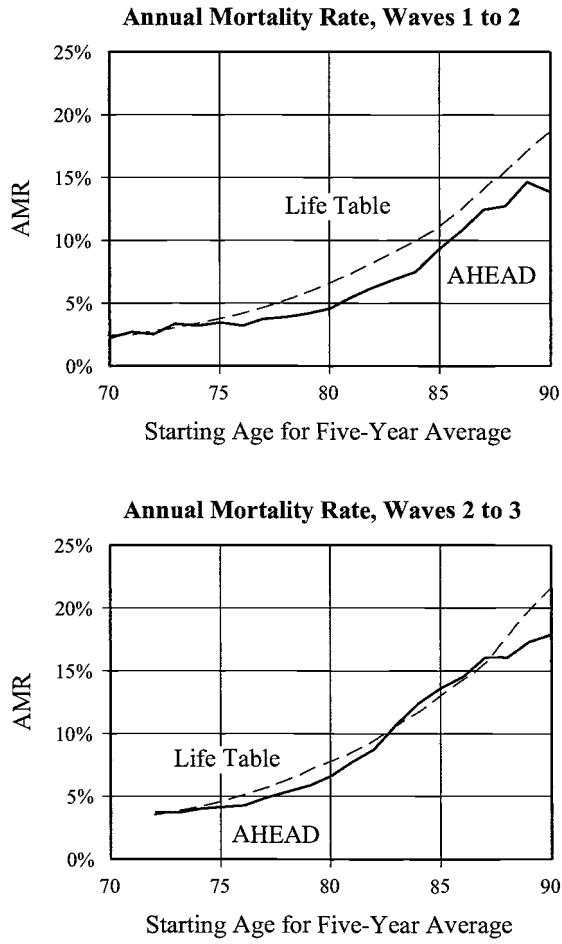


Fig. 11.5 Mortality hazard for AHEAD white females

The restriction of the AHEAD panel to the noninstitutionalized elderly in wave 1 selects against those with the highest mortality risk, particularly at the oldest ages, but the impact of this selection attenuates over time. For white females, figure 11.5 compares the observed annual mortality rate in the AHEAD panel with the expected annual mortality rate from the 1997 life tables for the United States (U.S. Census 1999).[16] Between waves 1 and 2, the AHEAD mortality risk is substantially below the life table for ages above seventy-five, reflecting the selection effect of noninstitutionalization.

16. The AMR for the AHEAD sample is the actual death rate between waves for each five-year segment of ages in the initial wave, annualized using the median 25.5 month interval between the waves. The AMR from the life tables is obtained by applying life table death rates by month to the actual months at risk for each individual in the five-year segment of ages in

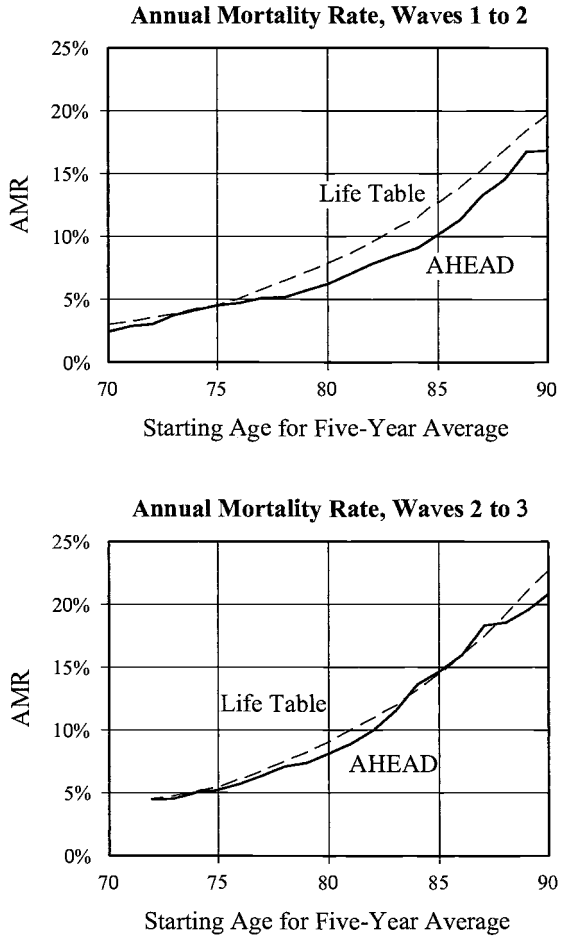


Fig. 11.6 Mortality hazard for AHEAD working sample

Between waves 2 and 3, this effect has essentially disappeared. There is a persistent divergence of the mortality risks above age ninety. In this range, the AHEAD data is sparse, so the curve is imprecisely determined. However, the life tables derived from historical mortality experience may overstate current mortality risk at advanced ages. Figure 11.6 makes the same mortality experience comparisons for the full AHEAD working sample and draws the same conclusions.[17]

the initial wave to calculate expected deaths between waves. This is annualized. For these calculations, the distribution of months at risk for decedents is assumed to be the same as that for survivors.

17. The construction mimics figure 11.5, with life table rates applied using the age, sex, and race of each subject.

11.3.2 Descriptive Statistics

The AHEAD survey provides data on health and socioeconomic status, as well as background demographics. A list of the health conditions we study, with summary statistics, is given in table 11.2. A list of the socioeconomic conditions and demographic variables we use is given in table 11.3. Table 11A.2 lists the variable transformations used in our statistical analysis.[18] In setting up causality tests, using the framework set out in section 11.2, we will use the health conditions, followed by the socioeconomic conditions, in the order given in these tables. We list cancer, heart disease, and stroke first because they may be instantaneously causal for death, and because we have information from decedent's exit interviews on new occurrence of these diseases. We group the remaining health conditions by degenerative and chronic conditions, then accidents, then mental conditions, because if there is any contemporaneous causality, it will plausibly flow in this order. Similarly, if there is contemporaneous causality between health and socioeconomic conditions, it plausibly flows from the former to the latter.

11.3.3 Constructed Variables

The collection and processing of some of the variables requires comment. The AHEAD has an extensive battery of questions about health conditions, including mental health. Most health conditions are asked for in the form "Has a doctor ever told you that you had . . . ?" However, for cancer, heart disease, and stroke, subjects are also asked if there was a new occurrence since the previous interview, and for some conditions such as arthritis, incontinence, and falls, the questions in wave 1 ask for an occurrence in the past twelve months. We note that there are some major groups of health conditions that were not investigated in AHEAD: degenerative neurological diseases, kidney and liver diseases, immunological disorders other than arthritis, sight and hearing problems, back problems, and accidents other than falls. The body mass index (BMI) index is calculated from self-reported height and weight. Information is collected on the number of limitations for six activities of daily living (ADL), and on the number of limitations for five instrumental activities of daily living (IADL). A high ADL limitation count indicates that the individual has difficulty with personal self-care, while a high IADL limitation count indicates difficulty in household management. The study collects data on self-assessed health status, where the subject is asked to rate his or her health as excellent, very good, good, fair or poor. We use an indicator for a poor/fair response. No reference is made to other groups such as "people your age." The study contains the Center for Epidemiologic Studies Depression scale (CESD)

18. All appendices can be found at http://elsa.berkeley.edu/wp/hww/hww202.html.

Table 11.2 Health Condition Variables in AHEAD

	Wave 1				Wave 2				Wave 3			
Label	Variable	*N*	Mean	SD	Variable	*N*	Mean	SD	Variable	*N*	Mean	SD
Health condition prevalence												
Doc ever told had cancer?	CANCER1	6,489	0.137	0.344	CANCER2	6,432	0.176	0.381	CANCER3	5,685	0.188	0.391
Doc ever told heart attack/disease?	HEART1	6,489	0.317	0.465	HEART2	6,432	0.364	0.481	HEART3	5,685	0.321	0.467
Doc ever told had stroke?	STROKE1	6,489	0.089	0.285	STROKE2	6,432	0.123	0.329	STROKE3	5,685	0.143	0.350
Doc ever told had lung disease?	LUNG1	6,489	0.115	0.318	LUNG2	6,432	0.124	0.330	LUNG3	5,685	0.127	0.333
Doc ever told had diabetes?	DIABET1	6,489	0.134	0.340	DIABET2	5,741	0.147	0.354	DIABET3	4,867	0.158	0.365
Doc ever told had high blood pressure?	HIGHBP1	6,489	0.502	0.500	HIGHBP2	5,741	0.525	0.499	HIGHBP3	4,867	0.553	0.497
Seen Doc for arthritis in last 12 m?	ARTHRT1	6,489	0.267	0.442	ARTHRT2	5,741	0.278	0.448	ARTHRT3	4,867	0.279	0.448
Incontinence last 12 m?	INCONT1	6,489	0.202	0.402	INCONT2	5,741	0.301	0.459	INCONT3	4,867	0.371	0.483
Fall in last 12 m require treatment?	FALL1	6,489	0.080	0.271	FALL2	6,432	0.180	0.384	FALL3	5,685	0.267	0.443
Ever fractured hip?	HIPFRC1	6,489	0.050	0.219	HIPFRC2	6,432	0.068	0.252	HIPFRC3	5,685	0.084	0.278
Proxy interview	PROXYW1	6,489	0.104	0.306	PROXYW2	5,741	0.132	0.339	PROXYW3	4,867	0.154	0.361
Age-educ. adjust cognitive impairment?	COGIM1	6,489	0.257	0.437	COGIM2	5,741	0.349	0.477	COGIM3	4,867	0.403	0.491
Ever seen Doc for psych prob?	PSYCH1	6,489	0.109	0.312	PSYCH2	5,741	0.128	0.334	PSYCH3	4,867	0.143	0.350
Depressed (cesd8 $>$ 4)	DEPRES1	6,488	0.099	0.299	DEPRES2	5,741	0.086	0.280	DEPRES3	4,862	0.108	0.310
Body mass index (Quetelet)	BMI1	6,475	25.4	4.5	BMI2	5,740	25.1	4.6	BMI3	4,866	25.0	4.6
Low BMI spline = max(0, 20 – bmi)	LOBMI1	6,475	0.155	0.654	LOBMI2	5,740	0.198	0.763	LOBMI3	4,866	0.223	0.810
High BMI spline = max(0, bmi – 25)	HIBMI1	6,475	1.889	3.164	HIBMI2	5,740	1.808	3.143	HIBMI3	4,866	1.740	3.037
Current smoker?	SMOKNOW1	6,489	0.102	0.303	SMOKNOW2	5,741	0.078	0.267	SMOKNOW3	4,867	0.066	0.249
No. of ADLs (needs help/difficult)	NUMADL1	6,489	0.725	1.391	NUMADL2	6,432	0.882	1.652	NUMADL3	5,444	1.035	1.774
No. of IADLs (needs help/difficult)	NUMIADL1	6,489	0.618	1.166	NUMIADL2	6,432	0.582	1.204	NUMIADL3	5,685	0.711	1.253
Poor/fair self-reported health	DHLTH1	6,483	0.373	0.484	DHLTH2	5,739	0.368	0.482	DHLTH3	4,859	0.434	0.496
Health condition incidence												
Cancer[a]					JCANCER2	6,432	0.050	0.218	JCANCER3	5,685	0.061	0.239
Heart attack/condition[a]					JHEART2	6,432	0.087	0.281	JHEART3	5,685	0.154	0.361
Stroke[a]					JSTROKE2	6,432	0.050	0.219	JSTROKE3	5,685	0.069	0.254
Died since last wave?					TDIED2	6,489	0.115	0.319	TDIED3	5,741	0.152	0.359

(*continued*)

Table 11.2 (continued)

	Wave 1				Wave 2				Wave 3			
Label	Variable	*N*	Mean	SD	Variable	*N*	Mean	SD	Variable	*N*	Mean	SD
Lung disease[a]					ILUNG2	6,432	0.024	0.154	ILUNG3	5,685	0.032	0.177
Diabetes[a]					IDIABET2	5,741	0.024	0.153	IDIABET3	4,867	0.025	0.156
High blood pressure (HBP)[a]					IHIGHBP2	5,741	0.053	0.225	IHIGHBP3	4,867	0.055	0.229
Arthritis[a]					IARTHRT2	5,741	0.112	0.316	IARTHRT3	4,867	0.117	0.321
Incontinence *in last 12 months*					JINCONT2	5,741	0.233	0.423	JINCONT3	4,867	0.258	0.438
Fall requiring treatment[a]					JFALL2	6,432	0.128	0.335	JFALL3	5,240	0.165	0.371
Hip fracture[a]					JHIPFRC2	6,432	0.02	0.150	JHIPFRC3	5,681	0.033	0.178
Proxy interview					PROXYW2	5,741	0.132	0.339	PROXYW3	4,867	0.154	0.361
Cognitive impairment					ICOGIM2	5,741	0.121	0.326	ICOGIM3	4,867	0.100	0.300
Psychiatric problems[a]					IPSYCH2	5,741	0.046	0.210	IPSYCH3	4,867	0.040	0.196
Depression					IDEPRES2	5,741	0.051	0.220	IDEPRES3	4,862	0.072	0.259
BMI better indicator					BMIBT2	5,740	0.197	0.397	BMIBT3	4,866	0.190	0.392
BMI worse indicator					BMIWS2	5,740	0.167	0.373	BMIWS3	4,866	0.188	0.391

Notes: *N* = number of observations; SD = standard deviation.

[a]AHEAD Waves 2 and 3: "Since the last interview . . . ?".

Table 11.3 SES and Demographic Variables in AHEAD

	Wave 1				Wave 2				Wave 3			
Label	Variable	*N*	Mean	SD	Variable	*N*	Mean	SD	Variable	*N*	Mean	SD
SES variables												
Wealth 97 Dol (V.2) (000)	C.2W1	6,489	176.6	297.8	C.2W2	5,741	237.5	527.3	C.2W3	4,867	247.7	736.2
Nonliquid wealth (000)	C.2N1	6,489	112.0	191.8	C.2N2	5,741	121.2	312.1	C.2N3	4,867	119.2	400.7
Liquid wealth (000)	C.2L1	6,489	64.7	185.2	C.2L2	5,741	116.3	309.6	C2.L3	4,867	128.6	560.6
Income 97 Dol (V.2) (000)	C.211	6,489	23.8	28.8	C.212	5,741	24.5	58.6				
1st quartile wealth indicator	Q1WB1	6,489	0.261	0.654	Q1WB2	5,741	0.228	0.419	Q1WB3	4,867	0.255	0.436
4th quartile wealth indicator	Q4WB1	6,489	0.216	0.412	Q4WB2	5,741	0.273	0.446	Q4WB3	4,867	0.268	0.443
1st quartile income indicator	Q11B1	6,489	0.260	0.439	Q11B2	5,741	0.246	0.431				
4th quartile income indicator	Q41B1	6,489	0.247	0.431	Q41B2	5,741	0.246	0.431				
Change residence?					NMOVED2	5,642	0.068	0.251	MOVED3	4,867	0.102	0.303
Own residence?	DNHOUS1	6,489	0.732	0.443	DNHOUS2	5,741	0.721	0.449	DNHOUS3	4,867	0.707	0.455
Neighborhood safety poor/fair	HOODPF1	6,489	0.147	0.354	HOODPF2	5,741	0.107	0.309	HOODPF3	4,867	0.101	0.301
House condition poor/fair	CONDPF1	6,489	0.146	0.354	CONDPF2	5,741	0.109	0.312	CONDPF3	4,867	0.123	0.328
Demographic variables												
Widow?	WIDOW1	6,489	0.416	0.493	WIDOW2	5,741	0.443	0.497	WIDOW3	4,867	0.434	0.496
Divorced/separated?	DIVSEP1	6,489	0.054	0.226	DIVSEP2	5,741	0.054	0.226	DIVSEP3	4,867	0.055	0.228
Married	MARRIED1	6,489	0.498	0.500	MARRIED2	5,741	0.465	0.499	MARRIED3	4,867	0.477	0.500
Never married?	NEVMARR1	6,489	0.032	0.176	NEVMARR2	5,741	0.035	0.172	NEVMARR3	4,867	0.031	0.172
Age at interview in months	AGEM1	6,489	939.2	72.0	AGEM2	6,432	962.9	71.9	AGEM3	5,741	984.8	68.7
Mother's death age	MAGEDIE1	6,489	73.9	17.4								
Father's death age	PAGEDIE1	6,489	71.5	15.1								
Ever smoke?	SMOKEV	6,489	0.526	0.499								
Education (years)	EDUC	6,489	10.7	3.8								
Educ > 10 years indicator	HS	6,489	0.605	0.489								
Educ > 14 years indicator	COLL	6,489	0.143	0.350								

Notes: *N* = number of observations; SD = standard deviation.

battery of questions measuring general mood, and from this we form an indicator for depression.

The AHEAD is linked to Medicare records. There is insufficient detail to permit reconciliation of self-reports on objective health conditions against diagnoses in the medical records, but errors in self-reports are an issue. We find small, but significant, inconsistencies across waves of AHEAD in reported chronic conditions. A study of Canadian data for a younger population finds substantial discrepancies between self-reported conditions and diagnoses from medical records, particularly for chronic conditions such as arthritis; see Baker, Stabile, and Deri (2001). These authors also find support for a "self-justification" hypothesis that nonworkers are more likely to make false positive claims for health conditions. If this reporting behavior carries into old age, then the reduction in SES as a consequence of spotty employment would induce an artifactual association of self-reported health conditions and SES.

The study measures cognition using a battery of questions which test several domains (Herzog and Wallace 1997): Learning and memory are assessed by immediate and delayed recall from a list of ten words that were read to the subject; reasoning, orientation, and attention are assessed from serial 7s, counting backwards by 1, and the naming of public figures, dates, and objects.[19] This score reflects both long-term native ability and health-related impairments due to health events. We carry out the following statistical analysis to reduce the effect of native ability so that we can concentrate on health-related loss of cognitive function. First, we analyze a "nonimpaired" sample of younger individuals, born between 1942 and 1947, who were administered the same cognitive battery in the 1998 Health and Retirement Study (HRS) as were the AHEAD subjects. For these younger individuals, where health-related impairment of cognitive function is rare, we carry out a least absolute deviation (LAD) regression of the cognitive score on education level, sex, and race. We use this fitted regression to predict a "baseline" nonimpaired cognitive score for each member of the AHEAD sample. An additional adjustment is required because average education levels were rising rapidly early in the twentieth century, due to changes in child labor laws and introduction of compulsory education. We assign each AHEAD subject a "1923 cohort equivalent" education level by first regressing education on sex, race, and birth cohort, using a specification search to find interactions and nonlinearities, and then adding to their actual years of education the difference in the mean years of education for their sex–race cohort and the corresponding 1923 sex–race cohort. We then calculate for each AHEAD sample member the deviation of their cognitive score from this adjusted baseline. As a normaliza-

19. Serial 7s asks the subject to subtract 7 from 100, and then to continue subtracting from each successive difference for a total of five subtractions.

tion, we assign a threshold such that 15 percent of AHEAD subjects aged seventy–seventy-four in wave 1 fall below the threshold. We then use the same threshold in other age groups and other waves to define an indicator for cognitive impairment.[20]

11.3.4 Measurement of Wealth

The AHEAD individuals and couples are asked for a complete inventory of assets and debts, and about income sources. Subjects are asked first if they have any assets in a specified category, and, if so, they are asked for the amount. A nonresponse to the amount is followed by unfolding bracket questions to bound the quantity in question, and this may result in complete or incomplete bracket responses. Through the use of unfolding brackets, full nonresponse to asset values was reduced to levels usually less than 5 percent, much lower than would be found in a typical household survey. Generally, median responses among full respondents for an asset category are comparable to other economic surveys, such as the Survey of Consumer Finance (SCF). However, changes in reported assets between waves contain outliers that suggest significant response errors between waves. For couples, where both members are asked the questions on assets, there is also substantial intersubject response variation. It is possible that these repeated reports could be used to control statistically for response error in couples. However, there are systematic differences between respondents, and we use the asset responses only from the individual that a couple says manages the household finances. There may also be an issue of bias in responses recovered by unfolding brackets. Hurd et al. (1998) used experimental variation in the bracket sequences for two financial questions on wave 2 of AHEAD, and found that anchoring to the bracket quantities was significant.

For complete or incomplete bracket responses in an asset category, we impute continuous quantities using hot deck methods. In wave 1, if information on ownership of an asset is missing for a subject, but this subject does give ownership status in wave 2, then we impute wave 1 ownership by drawing from the conditional empirical distribution of those who have the same response in wave 2 and give a response in wave 1. For subjects missing ownership in both waves 1 and 2, we draw an ownership pair from the empirical distribution of ownership pairs for those giving responses. Given ownership and complete or incomplete bracket information, we draw from the empirical distribution of wave 1 continuous responses that are consistent with the subject's bracket. In later waves, we have adopted a first-order

20. When an interview was done with a proxy, the cognitive battery was not given, but the interviewee was asked if the respondent was cognitively impaired. In our analysis, we treat proxy interview status as a component of the state that appears as a contemporaneous explanation of cognitive impairment; the coefficient on this variable compensates for differences in the definitions of cognitive impairment.

Markov cross-wave hot deck imputation procedure that assigns a continuous quantity within the given response bracket. First, missing ownership is imputed by choosing randomly from respondents, conditional on ownership in the previous wave. Then, given ownership, we impute a quantitative *change* in the item from the previous wave by drawing from the empirical distribution of subjects with complete responses that fall in the corresponding brackets in the current and the previous wave. This assures that imputed changes will have the same empirical distribution as observed changes, given the conditioning information available. This procedure does not revise previous wave imputations, so analyses based on earlier waves are not affected. We have experimented with cross-item imputation methods, where bracket information on some asset categories would be used to refine the conditioning used in the imputation of other asset categories. We have found that this has very little effect on the imputed variables or on the results obtained from analyses that use these variables. Therefore, we carry out all imputations one item at a time.

Measured wealth is accumulated over eleven asset categories, including imputed items. We distinguish *liquid wealth,* composed of IRA balances, stocks, bonds, checking accounts, and certificates of deposit, less debt; and *nonliquid wealth,* composed of net homeowner equity, other real estate, vehicles and other transportation equipment, businesses, and other assets. The variation in reported wealth of AHEAD households by asset category is substantial from wave to wave, suggesting that in addition to real volatility and reallocation of wealth portfolios, there are serious reporting problems with assets. Values of businesses owned and real estate are problematic items, because current market valuations may be unavailable to respondents, and subjective valuations may be unreliable. Suppose that W_t is measured wealth of household in wave t, in 1997 dollars, and that $W_t = W_t^* + \eta_t$, where W_t^* is true wealth and η_t is reporting error. To minimize the impact of extreme outliers in wealth and wealth changes, which we believe are a particular problem due to gross reporting errors, our statistical analysis will use bounded transformations of measured wealth.

The equations of motion for real wealth satisfy $\mathrm{d}W_i^*/\mathrm{d}t = rW_i^* + S_i$, where $i = T, N, L$ indexes total wealth or its nonliquid and liquid components, r is the instantaneous real rate of return, including unrealized capital gains, and S_i is the flow of savings to the wealth component. Make the logistic transformation $Z_i = 1/(1 + \exp(-c_i W_i + d_i))$, where c_i and d_i are chosen so that in AHEAD wave 1 the median and the semi-interquartile range of Z_i are one-half. Then, Z_i is a monotone transformation of measured wealth that is less sensitive to extremes. The equation of motion for Z_i is $\mathrm{d}Z_i/\mathrm{d}t - rZ_i(1 - Z_i)(\log(Z_i/(1 - Z_i)) + d_i) = c_i Z_i(1 - Z_i)S_i + c_i Z_i(1 - Z_i)(\mathrm{d}\eta/\mathrm{d}t - r\eta)$. We assume that over an inter-wave interval, this equation of motion can be approximated by

$$(10)\qquad \frac{Z_{it} - Z_{i,t-1} - R_{t-1}Z_{i,t-1}(1 - Z_{i,t-1})(\log(Z_{i,t-1}/(1 - Z_{i,t-1})) + d_t)}{m_t}$$

$$= S^{\#}_{it} + \sigma\nu_{it},$$

where t indexes the wave, m_t is the interval in months between waves $t - 1$ and t, R_t is the S&P real rate of return over the given interval, $S^{\#}_{it}$ is the measured part of $c_i Z_{i,t-1}(1 - Z_{i,t-1})S_{i,t-1}$, attenuated at extreme values of $Z_{i,t-1}$, and the disturbance ν_{it} includes the measurement error $c_i Z_{i,t-1}(1 - Z_{i,t-1})((\eta_{it} - \eta_{i,t-1})/m_{t-1} - r\eta_{t-1})$ and the unmeasured part of $c_i Z_{i,t-1}(1 - Z_{i,t-1})S_{i,t-1}$. We assume ν is homoskedastic. This is consistent with a measurement error in observed wealth that is heteroskedastic, with gross measurement errors more likely when true wealth is near its extremes.[21] The disturbance in (10) may be serially correlated; however, we have not incorporated this into our analysis. The effect of the transformation is to substantially reduce the influence of outliers in the distribution of changes in measured wealth. In application, we specify $S^{\#}_{it}$ to be a linear function of transformed nonliquid and liquid wealth, $Z_{T,t-1}(1 - Z_{T,t-1})\ \log(Z_{N,t-1}/(1 - Z_{N,t-1}))$ and $Z_{T,t-1}(1 - Z_{T,t-1})\ \log(Z_{L,t-1}/(1 - Z_{L,t-1}))$, and of other SES, demographic, and health variables, scaled by $Z_{T,t-1}(1 - Z_{T,t-1})$.

11.3.5 Mortality and Observed Wealth Change

A problem with the analysis of wealth changes is that terminal wealth is not observed following the death of a single, or the death of both members of a household, introducing a selection effect. A second problem is that a household death may have a direct impact on the wealth of a survivor, due to the expenses associated with a death and the disposition of the estate. There are also severe wealth measurement problems following a household death, because a death typically requires a valuation of assets, and in many cases changes the financially responsible respondent. For this reason, we will analyze separately wealth changes for singles and for couples, allow a regime shift following the death of one member of a couple, and account for the selection that occurs when there are no survivors.

For a single female, we adopt a bivariate selection model,

$$(11)\quad Y^{*}_{ft} = Y_{f,t-1}\beta_f + \varepsilon,\quad y_{ft} = \mathbf{1}(Y^{*}_{ft} > 0),\quad Y_{wt} = Y_{w,t-1}\beta_w + Y'_{w,t}\gamma_w + \lambda\varepsilon + \kappa\eta,$$
$$Y_{wt} \text{ observed if } y_{ft} = 1,$$

21. Heteroskedasticity in (10) will arise from selection effects, described later, as well as possibly from a failure of the transformation to fully control the effects of gross reporting errors. When working with this model, we use standard error estimates that are robust with respect to heteroskedasticity of unknown form and do not attempt direct tests of the implicit error specification underlying transformation (10).

where the first latent equation determines survival, $y_{ft} = 1$, the second equation corresponds to the transformed wealth change equation (10) with dependence on the previous state $Y_{w,t-1}$ and the previously determined components $Y'_{w,t}$ of the current state, with the wealth change observed for survivors. The disturbance ε_f has mean zero, variance one, and a density $f(\varepsilon)$. The disturbance η is independent of ε, and has mean zero and variance one. The correlation of the disturbances in the selection and wealth change equations is $\rho = \lambda/(\lambda^2 + \kappa^2)^{1/2}$, and the unconditional variance of the wealth change equation is $\sigma^2 = \lambda^2 + \kappa^2$. When ε and η are standard normal, this is the conventional bivariate normal selection model. However, specification tests for normality fail, and for robustness we adopt a more flexible specification, approximating the density $f(\varepsilon)$ by an Edgeworth expansion,

$$f(\varepsilon) = \sum_{j=0}^{J} \gamma_j H_j(\varepsilon)\phi(\varepsilon), \tag{12}$$

where the γ_j are parameters and $H_j(\varepsilon)$ are Hermite orthogonal polynomials; see Newey, Powell, and Walker (1990). Let $\Psi_{jk}(a) = \int_a^\infty \varepsilon^k H_j(\varepsilon)\phi(\varepsilon)d\varepsilon$. Then, the polynomials $H_j(\varepsilon)$ and the partial moment functions $\Psi_{jk}(a)$ can be constructed using the recursions

$$\begin{aligned}
&(13)\quad H_0(\varepsilon) = 1,\ H_1(\varepsilon) = \varepsilon,\ \text{ and }\ H_j(\varepsilon) = \varepsilon H_{j-1}(\varepsilon) - (j-1)H_{j-2}(\varepsilon)\ \text{ for } j > 1,\\
&\Psi_{00}(a) = \Phi(-a),\quad \Psi_{01}(a) = \phi(a),\quad \text{and}\\
&\Psi_{0k}(a) = a^{k-1}\phi(a) + (k-1)\Psi_{0,k-2}(a) \text{ for } k > 1,\\
&\Psi_{j0}(a) = H_{j-1}(a)\phi(a)\quad \text{and}\quad \Psi_{jk}(a) = a^k H_{j-1}(a)\phi(a) + k\Psi_{j-1,k-1}(a)\\
&\qquad \text{for } k > 0, \text{ for } j > 0.
\end{aligned}$$

Table 11A.1 derives these results and gives the leading terms for H_j and Ψ_{jk}: We require that f integrate to one and have unconditional mean zero and variance one; this forces $\gamma_0 = 1$, $\gamma_1 = 0$, and $\gamma_2 = 0$. The free parameters γ_j for $j > 2$ determine higher-order moments of ε. For example, skewness and kurtosis are determined by $E\varepsilon^3 = 6\gamma_3$ and $E\varepsilon^4 = 3 + 24\gamma_4$. With these restrictions, we have, finally

$$E(\varepsilon \mid \varepsilon > a) = \frac{\phi(a) + \sum_{j=3}^{j} \gamma_j \Psi_{j1}(a)}{\Phi(-a) + \sum_{j=3}^{J} \gamma_j \Psi_{j0}(a)} \quad \text{and} \tag{14}$$

$$E(\varepsilon^2 \mid \varepsilon > a) = \frac{\Phi(-a) + a\phi(a) + \sum_{j=3}^{j} \gamma_j \Psi_{j2}(a)}{\Phi(-a) + \sum_{j=3}^{j} \gamma_j \Psi_{j0}(a)}.$$

We use the Edgeworth approximation and these conditional expectations with $J = 4$. We then have

$$(15)\quad \mathrm{E}(Y^*_{wt} \mid \varepsilon > -Y_{f,t-1}\beta_f) = Y_{w,t-1}\beta_w + Y'_{w,t}\gamma_w + \lambda \frac{\phi(Y_{f,t-1}\beta_f) + \sum_{j=3}^{4} \gamma_j \Psi_{j1}(-Y_{f,t-1}\beta_f)}{\Phi(Y_{f,t-1}\beta_f) + \sum_{j=3}^{4} \gamma_j \Psi_{j0}(-Y_{f,t-1}\beta_f)}.$$

We estimate this conditional expectation in a two-step procedure. First, the parameters β_f of the selection equation are estimated by maximum likelihood, and substituted into expression (15) for the expectation of the wealth change equation.[22] Then, the parameters in this conditional expectation are estimated using nonlinear least squares. The disturbance $\zeta = \lambda\varepsilon + \kappa\eta - \mathrm{E}(\varepsilon \mid \varepsilon > -Y_{f,t-1}\beta_f)$ has mean zero and variance:

$$(16)\quad \mathrm{E}(\zeta^2 \mid \varepsilon > -Y_{f,t-1}\beta_f) = \kappa^2 + \lambda^2 \left(\frac{\sum_{j=0}^{J} \gamma_j \Psi_{j2}(-Y_{f,t-1}\beta_f)}{\sum_{j=0}^{J} \gamma_j \Psi_{j0}(-Y_{f,t-1}\beta_f)} - \left[\frac{\sum_{j=0}^{J} \gamma_j \Psi_{j1}(-Y_{f,t-1}\beta_f)}{\sum_{j=0}^{J} \gamma_j \Psi_{j0}(-Y_{f,t-1}\beta_f)} \right]^2 \right).$$

We regress the squared residuals from the estimation of (15) on the right-hand-side variables in (16) to obtain an estimate of κ^2. Finally, we estimate the covariance matrix for the parameter estimates using the generalized method of moments "sandwich" formula, with the Eicker-White procedure used for robustness against heteroskedasticity of unknown form, and the delta method used to incorporate the effects of variance in the first-stage selection parameter estimates.

Next consider the effects of death and selection on couples. We adopt a trivariate selection model with selection equations

$$(17)\quad Y^*_{ft} = Y_{f,t-1}\beta_f + \varepsilon_f,\ Y^*_{mt} = Y_{m,t-1}\beta_m + \varepsilon_m,\quad y_{ft} = \mathbf{1}(Y^*_{ft} > 0),\quad y_{mt} = \mathbf{1}(Y^*_{mt} > 0)$$

for the female and male members of the couple, respectively, where ε_f and ε_m are assumed to be independent with zero mean and unit variance, and densities $g_f(\varepsilon_f)$ and $g_m(\varepsilon_m)$. The independence assumption could fail if there are hidden common factors in mortality risk for both household members; for example, indirect effects of smoking. However, the frequency of multiple deaths in a household between waves is sufficiently rare in the AHEAD data so that mortality risk interactions are empirically not identified. We distinguish three regimes (y_f, y_m) in which wealth change is observed: intact couples where both members survive (1,1), the female survives the death of her spouse (1,0), and the male survives the death of his

22. We find that the coefficients of the index in the mortality model are not sensitive to the Edgeworth generalization, and in estimation of the model for wealth change use the first-stage probit models for mortality estimated earlier, with invariance imposed, to obtain the indices that appear in the selection effects.

spouse (0,1). We will let $Y_t^0 = [Y_{ht-1}\, Y_{mt-1}\, y_{ft} \cdot\ Y'_{ft} y_{mt} \cdot\ Y'_{mt}]$ denote the vector of variables that explain wealth change, where Y_{ht-1}, Y_{ft-1}, and Y_{mt-1} are, respectively, previous wave common, female, and male variables, Y'_{ft} are previously determined components of the current state for females, observed only for survivors and hence zeroed out for nonsurvivors, and Y'_{mt} are the analogous previously determined components of the current state for males, again observed only for survivors. We assume that in an observed regime *jk* the wealth change model takes the form

$$(18)\quad Y_{wt} = Y_t^0 \beta_{wjk} + (\lambda_f - \theta_f \mathbf{1}(j+k=1))\varepsilon_f + (\lambda_m - \theta_m \mathbf{1}(j+k=1))\varepsilon_m + \kappa_{jk}\eta,$$

where η is independent of ε_f and ε_m, with zero mean and unit variance. For intact couples, unobserved dependence of wealth change on selection is reflected in the parameters λ_f and λ_m, a direct extension of the bivariate selection model to the trivariate case with two independent selection effects. For intact couples, the correlations of the disturbances in the selection and wealth change equations are $\rho_f = \lambda_f/(\kappa_{11}^2 + \lambda_f^2 + \lambda_m^2)^{1/2}$ and $\rho_m = \lambda_m/(\kappa_{11}^2 + \lambda_f^2 + \lambda_m^2)^{1/2}$. The coefficients β_{jk} and the standard deviation κ_{jk} are allowed to vary by regime, to capture the observed and unobserved economic effects of a death on valuation and reporting of assets. In addition, the selection effects are allowed to shift, from λ_f to $\lambda_f - \theta_f$ for the female selection disturbance, and from λ_m to $\lambda_m - \theta_m$ for the male selection disturbance. We incorporate these effects to accommodate an apparent interaction in which unexpected survival of a couple (for example, ε_f and ε_m large positive) increases dissaving, perhaps due to additional medical and living expenses linked to overcoming high mortality hazards, but the unexpected death of a spouse (for example, ε_f large negative) also increases dissaving, perhaps because revaluations of assets tend to be more drastic in circumstances where mortality hazard is low.

We adopt the Edgeworth approximation (12) for each of the densities $g_f(\varepsilon_f)$ and $g_m(\varepsilon_m)$. In regime *jk* with $j + k > 0$, letting $s_f = 2y_f - 1$ and $s_m = 2y_m - 1$, one has

$$\begin{aligned}(19)\qquad \mathrm{E}(Y_{wt} \mid jk) \;=\; & Y_t^0 \beta_{wjk} + s_f(\lambda_f - y_f\theta_f) \\ & \cdot \frac{\phi(Y_{f,t-1}\beta_f) + \sum_{j=3}^{4} \gamma_j \Psi_{j1}(-Y_{ft-1}\beta_f)}{\Phi(s_f Y_{f,t-1}\beta_f) + s_f \sum_{j=3}^{4} \gamma_j \Psi_{j0}(-Y_{f,t-1}\beta_f)} \\ & + s_m(\lambda_m - Y_m\theta_m) \\ & \cdot \frac{\phi(Y_{m,t-1}\beta_m) + \sum_{j=3}^{4} \gamma_j \Psi_{j1}(-Y_{m,t-1}\beta_m)}{\Phi(s_m Y_{m,t-1}\beta_m) + s_m \sum_{j=3}^{4} \gamma_j \Psi_{j0}(-Y_{m,t-1}\beta_m)}.\end{aligned}$$

As in the case of singles, we estimate the conditional expectation (19) by nonlinear least squares after plugging in estimates of β_f and β_m from the earlier mortality models. A final stage, analogous to (16), regresses the

squared residuals from (19) for each regime on an intercept, $E(\varepsilon_f^2 \mid s_f) - (E(\varepsilon_f \mid s_f))^2$, and $E(\varepsilon_m^2 \mid s_m) - (E(\varepsilon_m \mid s_m))^2$; the coefficient on the intercept is an estimate of κ_{jk}^2. Because the number of deaths among couples is relatively small, we impose the constraints $\beta_{w10} = \beta_{w01}$ for empirical identification.

Household income in AHEAD is also susceptible to measurement error, and to minimize its effect, we use income quartiles as explanatory variables. These are obtained by converting all incomes to 1997 dollars, determining the quartiles for the pooled incomes of all subjects in all waves, and using the thresholds thus established to classify each observed subject income. AHEAD respondents rate the safety of their neighborhood and the condition of their dwelling on a five-point scale, from poor to excellent; we use indicators for poor or fair responses.

11.4 Socioeconomic Status and Prevalence of Health Conditions

11.4.1 Descriptive Statistics

We first give some descriptive statistics on the prevalence of health conditions in the AHEAD population. Table 11.4 shows prevalence rates in the AHEAD sample in wave 1, classified by age and sex, for the health conditions listed in table 11.2. Generally, prevalence of health conditions does not show a strong age gradient, indicating broadly that morbidity rates among survivors do not increase much with age. Selection effects from initial noninstitutionalization and from mortality may be responsible. The major exception is cognitive impairment, which rises as age increases. The prevalence of acute and degenerative conditions among survivors fall after about age eighty, reflecting the effect of selection due to deaths from these conditions. Males have higher prevalence of acute and degenerative diseases than do females, but females have higher prevalence of mental and chronic conditions, and accidents.

Figure 11.7 shows the age gradients of wealth, income, and education in wave 1 of the AHEAD sample. These gradients reflect substantial cohort effects, as well as life-cycle and composition effects. Work, income, and asset accumulation patterns of the AHEAD population were impacted by World War II, and those over age eighty experienced the Great Depression during their prime working years. The United States was substantially rural when the AHEAD population was born, and education was truncated for work for many members of this population. In addition to cohort effects, the curve for assets reflects life-cycle decumulation of assets through the retirement years, and the curve for income reflects the rising proportion of widows in the survivors to older ages. There is an additional compositional effect from the association of SES and mortality: Higher SES is selected preferentially among survivors. However, in aggregate cross section, the life-cycle and cohort effects dominate the compositional effects.

Table 11.4 **Prevalence of Health Conditions, by Age and Sex**

	White Females					White Males				
Condition	70–74	75–79	80–84	85–89	90+	70–74	75–79	80–84	85–89	90+
Cancer[a]	0.122	0.137	0.168	0.141	0.127	0.132	0.185	0.202	0.187	0.093
Heart disease[a]	0.244	0.275	0.361	0.339	0.370	0.361	0.390	0.423	0.368	0.296
Stroke[a]	0.051	0.069	0.104	0.121	0.133	0.066	0.12	0.102	0.130	0.148
Lung disease[a]	0.109	0.123	0.108	0.082	0.055	0.151	0.171	0.173	0.093	0.056
Diabetes[a]	0.121	0.097	0.107	0.088	0.055	0.133	0.145	0.112	0.104	0.074
High blood pressure[a]	0.481	0.510	0.537	0.582	0.448	0.433	0.477	0.418	0.321	0.167
Arthritis[b]	0.222	0.285	0.267	0.328	0.254	0.167	0.205	0.196	0.161	0.167
Incontinence[b]	0.228	0.263	0.266	0.331	0.365	0.085	0.138	0.145	0.161	0.259
Fall[b]	0.082	0.096	0.116	0.138	0.133	0.044	0.046	0.048	0.088	0.056
Hip fracture[a]	0.032	0.052	0.079	0.127	0.138	0.020	0.031	0.041	0.067	0.056
Cognitive impairment	0.120	0.149	0.338	0.452	0.635	0.137	0.173	0.291	0.446	0.500
Psychiatric disease[a]	0.149	0.137	0.105	0.082	0.044	0.094	0.092	0.071	0.036	0.056
Depression	0.070	0.117	0.131	0.099	0.116	0.048	0.050	0.071	0.083	0.093
Smoker	0.133	0.095	0.058	0.040	0.017	0.142	0.123	0.082	0.041	0.093
ADL impairment[c]	0.375	0.544	0.829	1.266	2.16	0.285	0.499	0.691	1.114	1.444
IADL impairment[c]	0.293	0.340	0.662	1.099	2.011	0.304	0.422	0.658	0.964	1.426
Self-reported health	0.273	0.348	0.387	0.393	0.42	0.263	0.364	0.437	0.332	0.296

[a]AHEAD 1993 (wave 1) question: "Has a doctor ever told you . . . /Do you have . . . /Have you ever . . . ?."

[b]AHEAD 1993 (wave 1) question: "During the last 12 months, have you . . . ?".

[c]Average number; max(ADL) = 6, max(IADL) = 5.

11.4.2 Models of Association

To examine the association of SES and health conditions, we estimate a series of binomial probit models of the form

$$P(Y_{it} = 1 \mid Y_{1t}, \ldots, Y_{i-1,t}, W_t, X_t), \tag{20}$$

where the Y_{it} are indicators for the prevalence of various health conditions, W_t denotes a vector of SES conditions, and X_t denotes other demographic variables. The health conditions appear in the same sequence as in table 11.2, with previous conditions in (20) providing information on association among health conditions. Included in W_t are indicators for the top and bottom quartiles of wealth and income, indicators for ten or more years of education (high school) and for fourteen or more years of education (college), and indicators for poor or fair neighborhood safety and dwelling condition.

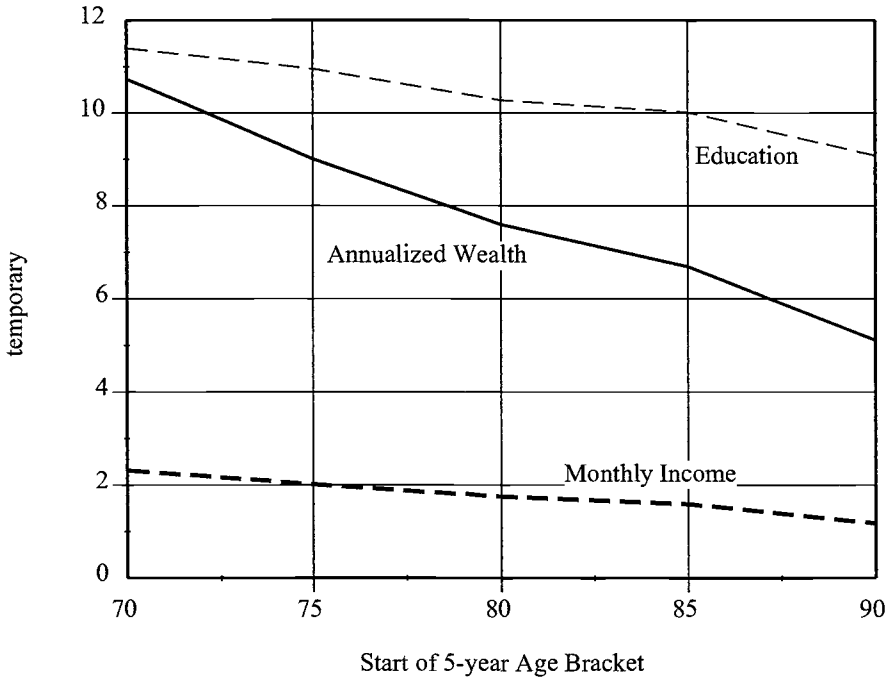


Fig. 11.7 Cohort gradients (education, wealth, and income)

The detailed estimation results can be found at http://elsa.berkeley.edu/wp/hww/hww202.html. Sample sizes are not adequate for comparable models for non-whites. We find the expected patterns of comorbidity, with a strong association of heart disease, stroke, lung disease, and arthritis, and a strong association of diabetes, heart disease, and high blood pressure. Incontinence is associated with cancer and stroke, and for women with diabetes, high blood pressure, and arthritis. Falls, hip fractures, and strokes are associated. Psychiatric diseases and depression are associated with arthritis and falls. BMI is positively associated with diabetes, high blood pressure, and arthritis, and negatively associated with lung disease. Current smokers have lower BMI, are less likely to have diabetes, and are more likely to be depressed. Numbers of ADLs and IADLs are positively associated with most acute diseases, arthritis, falls, hip fractures, cognitive impairment, and psychiatric disease. A poor or fair self-reported health status is associated with most acute and chronic diseases, with ADLs and IADLs, and with depression.

Some covariates are associated with health conditions, and may be risk factors for these conditions. Widowhood is associated with increased cancer and heart disease for women, increased psychiatric disease for men, and increased lung disease and depression for both men and women. For

Table 11.5 Summary of Associations of SES and Health Conditions AHEAD Wave 1

SES Association	White Females	White Males
1 percent level	Heart disease, stroke, lung disease, diabetes, cognitive impairment, depression, BMI, IADL impairment, self-reported health status	Lung disease, cognitive impairment, BMI, current smoker, ADL impairment, IADL impairment, self-reported health status
5 percent level	Cancer, HBP, Arthritis	HBP, depression
Not significant	Incontinence, fall, hip fracture, psychiatric condition, current smoker, ADL impairment	Cancer, heart disease, stroke, diabetes, arthritis, incontinence, fall, hip fracture, psychiatric condition

Note: HBP = high blood pressure; BMI = body mass index (high or low); ADL = activities of daily living (impairment requiring assistance with personal care); IADL = instrumental activities of daily living (impairment requiring assistance with household management).

women, father's age at death is associated with heart disease, and mother's age at death is associated with high blood pressure. For men, father's age at death is associated with high blood pressure and arthritis.

We generally find a statistically significant association of SES and prevalence of health conditions, as summarized in table 11.5. It is noteworthy that for males the prevalence of the acute diseases, cancer, heart disease, and stroke are not strongly associated with SES, contrary to literature findings for younger populations. This may be the result of early onset of these diseases, particularly among the poor and among smokers, that selects out of the AHEAD population those males most at risk for these diseases. Overall, wealth is the SES component most commonly associated with health conditions. Education, neighborhood rating, and dwelling rating are occasionally significant, and income is almost never significant. Table 11.6 summarizes the SES components that are individually significant in their association with various health conditions and indicates the sign of the correlation. For a number of these conditions, prevalence rates are insufficient to detect the effects of SES components with satisfactory power. However, for heart disease, high blood pressure, arthritis, cognitive impairment, and self-rated health status, sample sizes should guarantee reliable indicators of association.

11.4.3 Relative Risk

To provide an indication of the direction and magnitude of the association of health conditions and SES, we calculate *relative risk* for low SES versus high SES, where the definition of *low SES* is bottom quartiles for income and wealth, less than a high school education, and a poor/fair neighborhood and dwelling, and the definition of *high SES* is top quartiles for income and wealth, a college education, and a good or better neighborhood and dwelling. Relative risk is defined as the AHEAD sample average of the ratio of the two probabilities, all other variables remaining at the ob-

Table 11.6 **Significant Associations of SES Components and Health Conditions, by Gender**

	Wealth		Income		Education		Unsafe Neighborhood		Dwelling Poor/Fair	
Condition	F	M	F	M	F	M	F	M	F	M
Cancer						↗	↘			
Heart disease	↘↘								↗↗	
Stroke	↘↘				↘↘					
Lung disease		↘								
Diabetes	↘	↘		↘						
HBP										↗↗
Arthritis	↘	↘								
Incontinence							↗		↗	↗↗
Fall	↘						↗			
Hip fracture										
Cognitive impairment	↘↘		↘↘		↘↘	↘↘		↗		↗↗
Psychiatric condition					↘					
Depression				↘	↘				↗↗	
BMI	↘↘				↘↘	↘↘				
Current smoker		↘↘								↗
ADL impairment		↘↘								
IADL impairment	↘↘				↘↘					
Poor/Fair self-rated health	↘↘				↘↘	↘		↗	↗↗	↗↗

Notes: The table summarizes 180 two-tailed significance tests, so that if the tests were independent, one would expect about 9 of the 41 significant coefficients at the 5 percent level by chance, and about 2 of the 23 significant coefficients at the 1 percent level by chance.

↗ = positive at 5 percent level; ↗↗ = positive at 1 percent level; ↘ = negative at 5 percent level; ↘↘ = negative at 1 percent level. See table 11.5 notes for explanations of abbreviations.

Table 11.7 **Relative Risk of High vs. Low SES for Various Health Conditions, by Gender**

	Relative Risk			Relative Risk			Relative Risk	
Condition	F	M	Condition	F	M	Condition	F	M
Cancer	1.97	0.98	HBP	0.76**	0.65**	Cognitive impairment	0.53**	0.17**
Heart disease	0.46**	0.75**	Arthritis	0.80	0.60**	Psychiatric	1.14	0.64
Stroke	0.61	0.43**	Incontinence	0.83	0.71	Depression	0.34**	0.21**
Lung disease	0.30**	0.33**	Fall	0.68	0.44**	Smoke now	0.27**	0.23**
Diabetes	0.19**	0.65	Hip fracture	0.82	0.83	Health poor/fair	0.31**	0.34**

Notes: High SES is defined as top quartile in wealth and income, college education, and good neighborhood and welling; low SES is defined as bottom quartile in wealth and income, less than a high school education, and poor neighborhood and welling. Associations in AHEAD wave 1. See table 11A.3 for an pdated version of these results.

**Relative risks that are significantly different from one at the 5 percent level.

served levels for the subjects. Table 11.7 summarizes the relative risks for the various health conditions. Note that the prevalence models are describing only association, not causation, so that relative risk numbers cannot be interpreted causally. With the statistically insignificant exception of cancer and psychiatric conditions for females, high SES is associated with lower prevalence. Thus, we confirm in the AHEAD population the literature findings of a systematic association of SES with mortality and morbidity risk and show that this association extends across a variety of acute, degenerative, chronic, and mental health impairments.

11.5 Incidence of Health Conditions and Tests for Causality in the AHEAD Panel

11.5.1 Models of Incidence

Following the format described in section 11.2, we use the incidence of new health problems (or recurrence of cancer, heart disease, stroke, incontinence, falls, and hip fractures), conditioned on initial demographic, health, and SES status, to test for the absence of direct causal pathways. We define incidence for a group of health conditions to be the occurrence of a condition that was not previously reported, or a recorded reoccurrence in the case of an acute condition (cancer, heart disease, stroke). The descriptive statistics in table 11.2 provide information on rates of incidence of these conditions between waves.[23]

We estimate models for incidence of each health condition, conditioned on previously considered incidences of health conditions, the prevalence of health conditions in the previous wave of the panel, and on SES and demographic variables in the previous wave. The models are binomial probit except for BMI, which is fitted with a linear model using ordinary least squares (OLS), and numbers of ADL and IADL impairments, which are fitted as ordered probits. Detailed parameter estimates can be found at http://elsa.berkeley.edu/wp/hww/hww202.html. Again, the data do not permit the same analysis of nonwhites. The models are estimated by stacking the data for wave 1 to wave 2 transitions above the data for wave 2 to wave 3 transitions. Table 11.8 summarizes the health conditions and covariates that are significant risk factors for the incidence of health conditions. The associations reflect a number of known comorbidities, but show relatively few associations of SES components and incidence of health conditions.

11.5.2 Causality Tests

Figure 11.8 gives the structure of the invariance and causality tests we report. We test only whether the model parameters are invariant between the

23. The incidence rates in table 11.2 can be converted to crude annual rates via the formula $0.4706 \cdot \log(1 + \text{rate})$. These rates are uncorrected for population composition effects.

Table 11.8 **Statistically Significant Risk Factors for Incidence of Health Conditions**

	Health Conditions and Comorbidities		SES Covariates	
Incidence	Female	Male	Female	Male
Cancer	Cancer (↗), BMI (↘),		Ever smoke (↗)	
Heart disease	Lung disease (↗), diabetes (↗), poor/fair self-rated health (↗)	Diabetes (↗), poor/fair self-rated health (↗)		
Stroke	Stroke (↗), new heart (↗)	Stroke (↗), HBP (↗), psychiatric (↗), new heart (↗)	Income (↗)	
Mortality	Cancer (↗), HBP (↗), arthritis (↘), cognitive impairment (↗), BMI (↘), ADL (↗), poor/fair self-rated health (↗), new cancer (↗), new heart (↗), new stroke (↗)	Cancer (↗), lung (↗), diabetes (↗), incontinence (↘), cognitive impairment (↗), BMI[a], ADL (↗), poor/fair self-rated health (↗), new cancer (↗), new heart (↗), new stroke (↗)		Mother's death age (↘)
Lung disease	Heart (↗), current smoker (↗), poor/fair self-rated health (↗)	Poor/fair self-rated health (↗)		Wealth (↘)
Diabetes	BMI (↗)	Lung disease (↘), BMI (↗)	Ever smoke (↘)	Education (↘)
HBP	BMI (↗), new heart (↗), new stroke (↗), new diabetes (↗)	Heart (↗), arthritis (↗), new heart (↗), new stroke (↗)		
Arthritis	Lung (↗), depression (↗), BMI (↗), ADL (↗), poor/fair self-rated health (↗)	HBP (↗), BMI (↗)	Education (↘), mother's death age (↗)	
Incontinence	Arthritis (↗), incontinence (↗), fall (↗), BMI (↗), ADL (↗), IADL (↗), new heart (↗), new stroke (↗), new arthritis (↗)	Lung (↗), incontinence (↗), IADL (↗), new stroke (↗)		

(*continued*)

Table 11.8 (continued)

	Health Conditions and Comorbidities		SES Covariates	
Incidence	Female	Male	Female	Male
Fall	Fall (↗), hip fracture (↗), cognitive impairment (↗), psychiatric (↗), new incontinence (↗)	Fall (↗), new stroke (↗), new incontinence (↗)		
Hip fracture	Fall (↗), hip fracture (↗), new fall (↗)	Hip fracture (↗), new incontinence (↗)		
Cognitive impairment	New stroke (↗), new hip fracture (↗)	HBP (↘), IADL (↗)	Education (↘)	
Psychiatric	Heart (↗), hip fracture (↘), cognitive impairment (↗), depression (↗), new incontinence (↗), new cognitive impairment (↗),	Arthritis (↗), BMI (↘), new HBP (↗), new incontinence (↗)	Income (↘), widow (↘), divorced/separated (↘)	
Depression	Cancer (↘), heart (↗), poor/fair self-rated health (↗), new arthritis (↗), new incontinence (↗), new psychiatric (↗)	Lung (↗), new incontinence (↗)	Ever smoke (↗)	
Self-rated health	Heart (↗), lung (↗), diabetes (↗), arthritis (↗), cognitive impairment (↗), depression (↗), poor/fair self-rated health (↗), new cancer (↗), new heart (↗), new stroke (↗), new lung (↗), new arthritis (↗), new depression (↗), new ADL (↗)	Heart (↗), lung (↗), HBP (↗), poor/fair self-rated health (↗), new cancer (↗), new heart (↗), new stroke (↗), new lung (↗), new cognitive impairment (↗), new psychiatric (↗), new depression (↗), new BMI [a], new ADL (↗)	Dwelling poor/fair (↗)	

Notes: One percent significance level. new = incidence since last wave. The table summarizes approximately 1000 tests of individual coefficients, so that if they were independent, one would expect approximately 10 of the listed associations reflect type 1 errors. See table 11.5 for explanations of abbreviations.

[a]Any change in BMI (body mass index), up or down, lowers self-rated health.

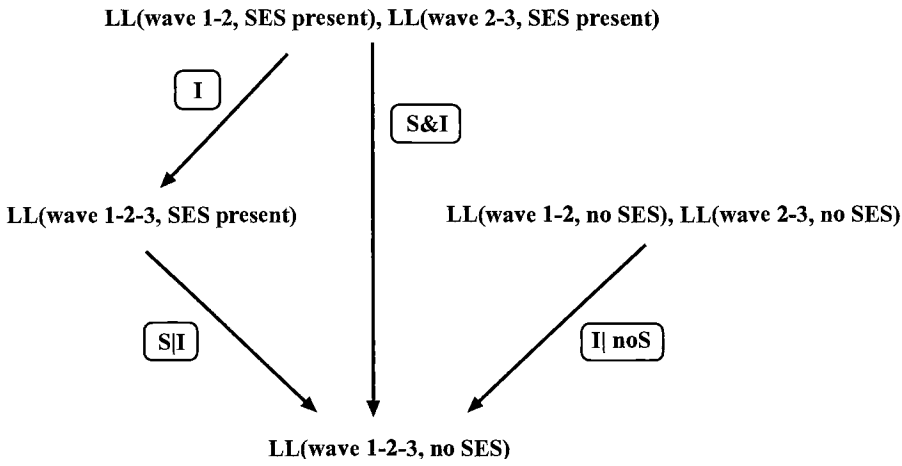


Fig. 11.8 Invariance and causality tests

Note: LL denotes log likelihood for specified condition, an unconditional invariance test is denoted by "I," a conditional test for no direct SES causality, given invariance, is denoted by "S | I" and a joint test of invariance and no direct SES causality is denoted by "S&I."

wave 1 to 2 transitions and the wave 2 to 3 transitions. We exclude intercepts, age splines, and log of time at risk terms from the invariance test. The reason for doing so is that these terms will capture variations in survey recontact procedure across waves. However, we find in most cases that there is no significant difference in the age spline coefficients across the different transitions. The models are estimated unconstrained, and with the imposition of invariance, noncausality of SES, or both. Likelihood ratio tests are conducted for invariance, with and without noncausality imposed, and for noncausality conditioned on invariance. Because the invariance test without noncausality of SES imposed and the noncausality test conditioned on invariance are nested, they should give the same conclusion, at compatible significance levels, and as a joint test of invariance and noncausality. In accordance with section 11.2, we take acceptance of the joint hypothesis as evidence that there is *not* a direct causal path from SES to incidence of the given health condition and take this as support for the proposition that differential access to medical care and SES-linked environmental hazards are not causing incidence rates to vary with SES.

The test results are given in table 11.9. The columns of numbers in these tables are, respectively, significance levels for the invariance test with SES variables included, the invariance test with SES variables excluded, the noncausality test conditioned on invariance, and the joint test of invariance and noncausality. The final columns in the table give the relative risk for high versus low SES (see section 11.4.3), and the significance level of a *T*-test of the null hypothesis that the relative risk is one.

Table 11.9 **Health Innovations, Tests for Invariance and Causality**

Condition	Condition occurred previously?	Sex	Significance Levels: Invariance with SES Variables	Significance Levels: Invariance w/o SES Variables	Significance Levels: No SES Causality, Given Invariance	Significance Levels: Joint Invariance and No SES Causality	Relative Risk: High vs. Low SES	Relative Risk: Significance
Cancer	All	F	0.056	0.023	0.311	0.057	1.260	0.568
	No	F	0.856	0.865	0.315	0.777	1.234	0.633
	Yes	F	0.133	0.335	0.949	0.313	3.729	0.608
	All	M	0.000	0.000	0.225	0.000	0.569	0.045
	No	M	0.122	0.071	0.059	0.044	0.467	0.008
	Yes	M	0.002	0.046	0.021	0.000	14.731	0.645
Heart	All	F	0.000	0.000	0.812	0.000	0.889	0.617
	No	F	0.214	0.075	0.447	0.240	0.814	0.480
	Yes	F	0.690	0.832	0.623	0.744	0.963	0.927
	All	M	0.017	0.020	0.290	0.018	1.321	0.412
	No	M	0.802	0.571	0.007	0.240	1.698	0.350
	Yes	M	0.472	0.601	0.250	0.382	1.225	0.710
Stroke	All	F	0.068	0.034	0.056	0.023	0.765	0.410
	No	F	0.371	0.239	0.104	0.205	0.748	0.413
	Yes	F	NC	0.529	0.597	NC	1.999	0.755
	All	M	0.086	0.042	0.290	0.080	1.179	0.717
	No	M	0.248	0.162	0.641	0.336	0.874	0.756
	Yes	M	NC	0.002	0.200	NC	8.300	0.584
Mortality		F	0.010	0.006	0.812	0.030	0.680	0.122
		M	0.021	0.032	0.228	0.018	1.196	0.664
Lung disease		F	0.493	0.479	0.381	0.470	0.341	0.000
		M	0.603	0.620	0.013	0.174	0.225	0.000
Diabetes		F	0.110	0.177	0.246	0.091	0.847	0.778
		M	0.243	0.199	0.043	0.085	2.649	0.941

HBP	F	0.004	0.009	0.777	0.012	1.109	0.779
	M	0.220	0.121	0.668	0.310	0.886	0.809
Arthritis	F	0.041	0.017	0.042	0.012	1.126	0.623
	M	0.187	0.276	0.145	0.116	0.415	0.000
Incontinence	F	0.357	0.329	0.080	0.183	0.867	0.324
	M	0.161	0.486	0.237	0.129	0.980	0.943
Fall	F	0.864	0.763	0.515	0.861	1.016	0.940
	M	0.402	0.383	0.507	0.438	0.944	0.871
Hip fracture	F	0.604	0.470	0.275	0.520	0.413	0.048
	M	0.056	0.051	0.305	0.055	0.198	0.003
Proxy	F	0.345	0.442	0.250	0.283	0.414	0.000
	M	0.424	0.326	0.019	0.131	0.447	0.001
Cognitive	F	0.020	0.007	0.001	0.000	0.759	0.162
	M	0.429	0.245	0.022	0.140	0.522	0.003
Psychiatric	F	0.075	0.031	0.012	0.012	0.342	0.000
	M	0.194	0.546	0.110	0.108	0.102	0.000
Depression	F	0.299	0.347	0.078	0.151	0.411	0.000
	M	0.767	0.856	0.302	0.695	0.352	0.001
BMI	F	0.419	0.330	0.738	0.531		
	M	0.010	0.002	0.249	0.009		
Smoke now	F	0.509	0.242	0.838	0.650	0.649	0.530
	M	0.366	0.146	0.064	0.182	6.448	0.841
ADL	F	0.010	0.018	0.818	0.027		
	M	0.004	0.044	0.370	0.005		
IADL	F	0.673	0.514	0.368	0.636		
	M	0.016	0.003	0.006	0.002		
Self-rated health	F	0.151	0.111	0.001	0.009	0.670	0.000
	M	0.581	0.570	0.034	0.282	0.656	0.000

Note: See table 11.5 for explanations of abbreviations.

In a majority of cases, our test for invariance is accepted. For cancer and heart disease incidence, it is necessary to separate models for those with and without a previous occurrence of the condition. For females, exceptions where invariance is rejected at the 1 percent level are mortality and ADL count. Exceptions for males are cancer with a previous occurrence, mortality, ADL count, BMI, and IADL count. The failure of the mortality models to satisfy invariance may be related to the initial selection of a noninstitutionalized population in wave 1 of the AHEAD panel. Of course, in addition to the question of the power of our test to detect invariance failures, our single test of invariance across waves falls considerably short of the battery of invariance tests that would be desirable to establish that the model system has the stability and sensitivity required for policy applications.

For females, the tests for noncausality of the SES variables, conditioned on a maintained hypothesis of invariance, are rejected for arthritis and psychiatric disease at the 5 percent level and for cognitive impairment and self-rated health at the 1 percent level. Notably, these are all chronic or mental conditions where Medicare coverage is limited and the cost of drugs or assistance may be substantial. For males, this test for noncausality is rejected for cancer with a previous occurrence, heart disease with no previous occurrence, lung disease, diabetes, cognitive impairment, and self-rated health at the 5 percent level, and for IADL count at the 1 percent level. The results of the joint test for invariance and noncausality are roughly consistent with the separate tests. For conditions such as cancer with a previous occurrence and IADL count for males, cognitive impairment for females, and mortality for both females and males, the noncausality test results may be confounded by the failure of invariance.

The relative risks in table 11.9 should *not* be interpreted causally, since again the cases where noncausality is rejected and the relative risks are substantially different from one may be due to a common unobserved effect rather than a direct causal link. The pattern of fifteen relative risks exceeding one and eighteen less than one suggests no broad linkage between SES and health changes, *given prior health,* and the direct links that may be indicated from the significance levels (lung disease and hip fractures for males and females, some cancers and arthritis for males) appear to be related to specific features of poverty, such as smoking history and poor dwelling environment. There are a few cases where the relative risk for high versus low SES is substantially less than one, irrespective of statistical significance, indicating an unproven link of sufficient magnitude to warrant further investigation: lung disease, hip fracture, and the mental diseases for females, and lung disease, diabetes, arthritis, and the mental diseases for males. Large deviations in relative risk from one that are not statistically significant suggest that acceptance of the joint hypothesis of invariance and noncausality could be due to low power. Notably, death shows no re-

lation to SES, once previous health state is controlled, and the relative risks are insignificantly different from one. This indicates that there are no strong direct causal links from SES to mortality, which at the level of resolution of this study rules out differential access to medical treatment for life-threatening illness. Thus, the association of SES and mortality among the elderly appears to come primarily from variation in the prevalence of health conditions with SES, and more weakly from indirect causal links from SES to incidence of health conditions that increase mortality risk.

The pattern of failures of the noncausality test for mental diseases suggests the possibility of a direct causal link related to differential access. Medicare limits the scope of care for mental conditions, so ability to pay may indeed be an important factor in efficacy of treatments that prevent or control these conditions.

11.6 Tests for Noncausality from Health Status to Asset Accumulation

11.6.1 Models of Incidence

Health may influence asset accumulation of elderly households because of the cost of medical treatment and related services. Medicare covers acute conditions with limited copayments, but there is the possibility of direct effects from uncovered costs of drugs and living assistance. Also, health conditions may limit the consumption of other goods, and because health status is an indicator of longevity, an individual planning consumption and precautionary reserves over remaining life may adjust target wealth based on altered perceptions of longevity and anticipated medical costs; see Alessie, Lusardi, and Kapteyn (2000), Attanasio and Hoynes (2000), Hurd (1987), Hurd and Wise (1989), Hurd and McGarry (1997), Hurd, McFadden, and Gan (1998). These effects could induce an association of SES and health status even if there were no causal links from SES to health. In the elderly AHEAD population, we will not observe the most likely direct causal link from health status to accumulation among workers, the effect of health on current labor market participation and productivity.

We analyze transitions in wealth from wave to wave using the framework of section 11.2 and model (11) for singles and (17) and (18) for couples, with demographics, previous wave health conditions, and current wave incidence of new health conditions as explanatory variables. Statistically significant selection coefficients are consistent with a direct causal link from death to a change in household wealth, but also consistent with ecological factors that induce an association of mortality risk and SES. Total, nonliquid, and liquid wealth are analyzed separately, with transformation (10) applied to each component.

Tables 11A.5, 11A.6, and 11A.7 give the detailed incidence models for

total, nonliquid, and liquid wealth change. As in previous studies of savings, we find that most of the variance in wealth changes over the population is not explained by observed economic variables. This remains true after introduction of health conditions. We find dissaving rates out of liquid wealth, before realization of returns calculated from the S&P 500, that are 5.3 percent for couples, 4.8 percent for singles, and 6.0 percent for survivors whose spouses have died. The dissaving rates from nonliquid wealth, again before realization of returns, are, respectively, 6.9 percent, 6.3 percent, and 8.0 percent for intact couples, singles, and survivors. The higher dissaving rates from nonliquid assets indicates that the wealth portfolios of the elderly are rebalanced to become more liquid as they age. These dissaving rates can be compared to an average rate of dissaving of 8.3 percent of remaining wealth in an age seventy-plus population with life-table survival probabilities who consume the expected annuitized value of their wealth.[24] Then, observed dissaving rates out of wealth are not grossly lower than would be expected with pure life-cycle consumption averaging over retirement and full pooling of mortality risk. We find that low income couples and individuals have significantly higher dissaving rates than their high income counterparts, but the differences are not quantitatively large. Home ownership is associated with significantly less dissaving for intact couples.

The models for both singles and couples show significant departures from normality in the selection equations. The Edgeworth expansion parameters show positive skewness and smaller than normal kurtosis for female singles, negative skewness and insignificantly different from normal kurtosis for male singles. For couples, both males and females have negative skewness and larger than normal kurtosis. We also find significant selection effects, with $\rho_f = -0.49$ for couples and -0.21 for singles, and $\rho_m = -0.51$ for couples and -0.89 for singles. These imply that households that survive despite unfavorable mortality risks have increased dissaving, either because of increased cost of overcoming health problems or because households at elevated risk spend down more rapidly. Equation (18) includes shift parameters that modify the dependence of the wealth change disturbance on the unobserved selection effects in regimes where a spouse dies. These are statistically significant, and sufficiently large to reverse the direction of the intact couple selection effects.

Table 11.10 summarizes the health conditions and other covariates that are individually significant in explaining changes in wealth. For intact couples, we find some acute conditions *increase* saving, perhaps because they restrict consumption, or perhaps because couples conserve assets for a potential surviving spouse. For the conditions that are associated with in-

24. This calculation is made from the 1996 life tables and assumes the historical S&P rate of return from 1993 to 1997, and a 7 percent real rate of return on assets after 1997.

Table 11.10 Statistically Significant Risk Factors for Wealth Changes

Component	Health Conditions	Covariates
Intact couple		
Total wealth	M self-rated health poor/fair (↗), M new heart (↗), M new hip fracture (↘), F new cancer (↗)	Nonliquid wealth (↘), liquid wealth (↘), income (↗), homeowner (↗)
Nonliquid wealth		Nonliquid wealth (↘), liquid wealth (↗), homeowner (↗)
Liquid wealth	M new heart (↗), M BMI worse (↘)	Nonliquid wealth (↗), liquid wealth (↘), income (↗), dwelling poor/fair (↘)
Spouse died		
Total wealth		Nonliquid wealth (↘), liquid wealth (↘), income (↗)
Nonliquid wealth		Nonliquid wealth (↘)
Liquid wealth	F new stroke (↘)	Nonliquid wealth (↗), liquid wealth (↘), income (↗)
Single		
Total wealth	M new cancer (↗), F new cancer (↘), F new depression (↘)	M,F nonliquid wealth (↘), M,F liquid wealth (↘), M,F income (↗)
Nonliquid wealth	M hip fracture (↗), M new cancer (↗), M new heart (↗)	M,F nonliquid wealth (↘), M liquid wealth (↗), F income (↗), F homeowner (↗)
Liquid wealth	F cognitive impairment (↘), F new stroke (↘), F new cognitive impairment (↘)	M,F nonliquid wealth (↗), M,F liquid wealth (↘), M,F income (↗), F dwelling poor/fair (↘)

Notes: One percent significance level. M = male; F = female; new = incidence since last wave; ↗ indicates increased saving; ↘ indicates decreased saving.

creased dissaving (cognitive impairment and stroke for single females), costs of maintenance associated with these conditions may be directly causal to wealth changes.

11.6.2 Causality Tests

Table 11.11 summarizes our tests for invariance and absence of direct causal links. We test for common parameters in the wealth change models between waves 1–2 and waves 2–3, excepting intercepts and age effects to allow for the effects of sample timing. Invariance is convincingly rejected for each demographic group and wealth category, indicating that our model fails to capture the structural determinants of wealth change. As a consequence, our noncausality tests to follow may produce rejections due to model misspecification, confounding the detection of direct causal

Table 11.11 Wealth Changes, Tests for Invariance and Causality

	Significance Levels			
		Noncausality		
Demographic Group Wealth Component	Invariance	Previous Health Conditions	Current Health Conditions	All Health Conditions
Intact couple				
Total wealth	0.000	0.036	0.001	0.000
Nonliquid wealth	0.000	0.014	0.028	0.001
Liquid wealth	0.000	0.006	0.012	0.000
Spouse died				
Total wealth	0.000	0.811	0.776	0.074
Nonliquid wealth	0.000	0.071	0.814	0.037
Liquid wealth	0.000	0.676	0.456	0.023
Single				
Total wealth	0.000	0.004	0.022	0.001
Nonliquid wealth	0.000	0.100	0.347	0.235
Liquid wealth	0.000	0.000	0.028	0.000

links. A deconstruction of the invariance failures, detailed in appendix table 11A.8, shows that for nonliquid and liquid wealth, invariance passes for demographic and health prevalence and incidence variables, but fails for female SES variables, and for all male variables including SES taken together. Thus, there was an unexplained regime shift before and after wave 2 of AHEAD. Possible explanations for this are an interaction between the criterion of noninstitutionalization in the initial panel recruitment and economic behavioral response; a wealth-linked interaction in panel retention; problems in the measurement of wealth in the AHEAD population, which exhibits unexplained mean reversion; or a true behavioral shift with age in a single cohort that is not captured accurately by a model that pools wealth change observations across cohorts.

We expect that terminal medical and burial expenses, estate taxes and other estate settlement costs, insurance payments, and bequests will have a substantial impact on the size of a decedent's estate and surviving spouse's reported wealth. We easily reject the hypothesis that the model coefficients for intact couples and for surviving spouses are the same, but note that measurement problems associated with a change in financially responsible respondent could also produce this rejection.

The hypothesis of no direct causality of health conditions for total wealth changes is rejected at the 1 percent level for intact couples and singles. For nonliquid wealth, the hypothesis is rejected for intact couples, and for liquid wealth, the hypothesis is rejected for all demographic groups.

The failure of the invariance tests, and the possibility of confounding by persistent common factors and selection suggest that conclusions on the health to wealth link be interpreted with caution. Table 11A.9 deconstructs the causality tests and identifies the health conditions and genders responsible for rejections. The pattern of results suggest that if there is indeed a direct causal link, then it is most likely to involve liquid wealth and health conditions that require assisted living.

Table 11A.10 estimates models of income change, given health conditions and other covariates. One would not expect health status to have a significant impact on the incomes of retirees, conditioned on previous wealth, and the empirical results are generally consistent with this expectation. We have not done formal tests of invariance or causality for income. Also included in the state vector Y_t that describes individuals are changes in home ownership status, and dwelling and neighborhood conditions. We estimate incidence models for these components; results are in table 11A.11.

11.7 Simulation of Life Courses under Counterfactual Conditions

11.7.1 The Simulation Experiment

For policy analysis of interventions that alter delivery or cost of medical services, or retirement financing, one would like to trace through the direct and indirect causal links between wealth, health, and mortality. If Markov models of the sort developed in sections 11.2–11.6 satisfy the required invariance properties, then they can be used to simulate the impacts of these interventions on the life courses of a synthetic population. In this section, we develop such a simulation analysis and apply it to illustrative interventions. Because we have not uniformly accepted invariance, and in a number of cases find associations that may be either direct causal links or hidden common factors, this simulation analysis assumes more than our estimates support. It should be interpreted only as an exercise that shows how a model of this general structure might be used in a policy application to unravel the dynamics of comorbidities and forecast condition-specific morbidity and mortality and life expectancy.

We simulate the life courses of a synthetic population in which heads of household are initially aged seventy–seventy-four. To synthesize this population, we start from the 1,612 households in AHEAD wave 1 whose heads are white and aged seventy–seventy-four. Using the SES and demographic variables for each household in this subsample, we make ten Monte Carlo draws from the prevalence models in section 11.4 to create synthetic initial health profiles for the household head, and spouse if present. This gives an initial synthetic sample of 16,120 households. We then

create life courses for the members of each household by drawing recursively from the Markov incidence models in sections 11.5 and 11.6, adjusted to annual transitions using (9) to approximate the probabilities of moving to new states.

We first consider a base scenario (S0) in which initial prevalence and incidence transitions are given by our models estimated on the AHEAD data. We note that the simulation outcomes can be expected to differ to some degree from the cross-cohort patterns found in AHEAD, because the distribution of conditions at ages seventy–seventy-four will differ from the distributions of prevalence that actually prevailed for older individuals in AHEAD when they were aged seventy–seventy-four. They should also differ to some degree from the actual experience that the aged seventy–seventy-four cohort in AHEAD will have through the remainder of their lives, because the simulation cannot anticipate the realized future distribution of exogenous variables and because our models do not allow for drift-in disease incidence or condition-specific mortality hazards that will result from changes in medical care. Historically, these drifts have been very significant, reducing morbidities and increasing life expectancies. If these trends continue, then the baseline simulations will underestimate actual survival experience.

We next consider two stylized policy interventions. The first alternative scenario (S1) examines the impact on life courses of the introduction of a hypothetical medical treatment that cures diabetes, for example, by stem cell and immune system therapy that rejuvenates the pancreas for both type I and type II diabetics. In this scenario, we assume that prevalence of diabetes at the start of the simulated panel drops to zero, and that there is zero incidence of this condition as the cohort ages. We do not alter the historical prevalence of conditions associated with diabetes. Thus, we assume that the historical impact on individuals of type I diabetes, notably increased prevalence of heart disease and stroke at ages seventy–seventy-four, is not altered. The second alternative scenario (S2) examines the impact on life courses of reducing the entire population from their current SES to our definition of a low-SES individual: bottom quartile for wealth and income, less than a high school education, and a poor or fair neighborhood and dwelling. This alternative is obviously hypothetical and is not even a stylized approximation to any real policy alternative. However, it provides an extreme in which the interactions of health and SES are permitted maximum play, giving an upper bound on the effect that SES could possibly have on health outcomes, and providing a finger exercise that tests the plausibility of our model system.

11.7.2 Baseline Simulation

Table 11.12 summarizes the survival probabilities and prevalence of health conditions among survivors in the simulated cohort as it ages, under

Table 11.12 Simulation Outcomes

		Age					
	Scenario	70	75	80	85	90	95
White females							
Survival probability[a]	S0	1.000	0.905	0.743	0.500	0.240	0.079
	S1	1.000	0.906	0.757	0.517	0.257	0.087
	S2	1.000	0.884	0.662	0.359	0.125	0.027
Cancer[b]	S0	0.112	0.174	0.219	0.245	0.257	0.262
	S1	0.112	0.169	0.217	0.243	0.258	0.252
	S2	0.112	0.208	0.273	0.298	0.302	0.336
Heart disease[b]	S0	0.224	0.322	0.442	0.538	0.588	0.594
	S1	0.224	0.321	0.429	0.515	0.569	0.608
	S2	0.224	0.344	0.495	0.608	0.663	0.679
Stroke[b]	S0	0.042	0.105	0.191	0.272	0.326	0.341
	S1	0.042	0.104	0.186	0.260	0.317	0.351
	S2	0.042	0.105	0.196	0.274	0.320	0.366
Lung disease[b]	S0	0.105	0.151	0.179	0.190	0.168	0.143
	S1	0.105	0.150	0.183	0.190	0.175	0.154
	S2	0.105	0.193	0.258	0.278	0.276	0.286
Diabetes[b]	S0	0.114	0.161	0.198	0.199	0.170	0.157
	S1	0.114	0.000	0.000	0.000	0.000	0.000
	S2	0.114	0.181	0.231	0.233	0.207	0.231
High Blood Pressure[b]	S0	0.467	0.583	0.678	0.738	0.761	0.778
	S1	0.467	0.585	0.678	0.742	0.783	0.804
	S2	0.467	0.597	0.701	0.764	0.786	0.810
Arthritis[b]	S0	0.232	0.501	0.677	0.791	0.857	0.901
	S1	0.232	0.501	0.670	0.784	0.856	0.905
	S2	0.232	0.567	0.769	0.876	0.925	0.965
Incontinence[b]	S0	0.232	0.416	0.601	0.751	0.843	0.891
	S1	0.232	0.427	0.611	0.749	0.844	0.906
	S2	0.232	0.444	0.664	0.814	0.891	0.917
Fall[b]	S0	0.071	0.271	0.464	0.635	0.759	0.837
	S1	0.071	0.268	0.456	0.628	0.754	0.846
	S2	0.071	0.260	0.460	0.640	0.759	0.822
Hip fracture[b]	S0	0.030	0.046	0.070	0.109	0.153	0.215
	S1	0.030	0.043	0.069	0.109	0.156	0.190
	S2	0.030	0.056	0.104	0.160	0.224	0.258
Proxy interview	S0	0.038	0.056	0.103	0.163	0.230	0.294
	S1	0.038	0.063	0.095	0.155	0.222	0.271
	S2	0.038	0.103	0.211	0.316	0.409	0.521
Cognitive impairment[b]	S0	0.104	0.253	0.437	0.609	0.736	0.818
	S1	0.104	0.251	0.435	0.602	0.722	0.803
	S2	0.104	0.405	0.669	0.838	0.910	0.952
Psychiatric disease[b]	S0	0.154	0.215	0.307	0.376	0.421	0.434
	S1	0.154	0.219	0.305	0.379	0.416	0.426
	S2	0.154	0.316	0.508	0.634	0.704	0.762
Depression[b]	S0	0.068	0.183	0.305	0.429	0.515	0.575
	S1	0.068	0.179	0.305	0.424	0.521	0.596
	S2	0.068	0.247	0.434	0.574	0.665	0.749

(continued)

Table 11.12 (continued)

		Age					
	Scenario	70	75	80	85	90	95
Body Mass Index[c]	S0	25.7	24.7	23.6	22.3	21.0	20.5
	S1	25.7	24.7	23.6	22.3	21.1	20.3
	S2	25.7	24.7	23.4	22.2	21.2	20.8
Current smoker[b]	S0	0.128	0.088	0.063	0.043	0.027	0.017
	S1	0.128	0.091	0.063	0.048	0.031	0.016
	S2	0.128	0.109	0.084	0.056	0.039	0.020
ADL limits[c]	S0	0.31	0.27	0.66	1.18	1.68	2.14
	S1	0.31	0.26	0.59	1.08	1.63	1.99
	S2	0.31	0.45	1.21	2.08	2.77	3.33
IADL limits[c]	S0	0.25	0.11	0.29	0.58	0.85	1.14
	S1	0.25	0.10	0.27	0.53	0.81	1.04
	S2	0.25	0.25	0.73	1.34	1.84	2.30
Poor/fair self-rated health[b]	S0	0.254	0.305	0.441	0.540	0.585	0.613
	S1	0.254	0.293	0.416	0.501	0.574	0.582
	S2	0.254	0.507	0.711	0.811	0.841	0.860
White males							
Survival probability[a]	S0	1.000	0.828	0.558	0.279	0.091	0.019
	S1	1.000	0.844	0.586	0.304	0.105	0.025
	S2	1.000	0.827	0.485	0.170	0.032	0.004
Cancer[b]	S0	0.145	0.242	0.320	0.400	0.478	0.514
	S1	0.145	0.248	0.321	0.395	0.482	0.546
	S2	0.145	0.281	0.391	0.479	0.555	0.596
Heart[b]	S0	0.352	0.490	0.611	0.709	0.774	0.733
	S1	0.352	0.488	0.626	0.701	0.752	0.766
	S2	0.352	0.443	0.554	0.627	0.632	0.681
Stroke[b]	S0	0.075	0.140	0.213	0.282	0.317	0.310
	S1	0.075	0.142	0.213	0.287	0.323	0.351
	S2	0.075	0.190	0.317	0.423	0.517	0.596
Lung disease[b]	S0	0.150	0.185	0.205	0.203	0.183	0.152
	S1	0.150	0.184	0.206	0.212	0.200	0.184
	S2	0.150	0.183	0.201	0.195	0.190	0.085
Diabetes[b]	S0	0.135	0.171	0.183	0.190	0.177	0.171
	S1	0.135	0.000	0.000	0.000	0.000	0.000
	S2	0.135	0.135	0.119	0.095	0.085	0.128
High Blood Pressure[b]	S0	0.443	0.553	0.636	0.699	0.719	0.743
	S1	0.443	0.551	0.637	0.702	0.740	0.784
	S2	0.443	0.565	0.643	0.689	0.692	0.638
Arthritis[b]	S0	0.175	0.405	0.582	0.718	0.801	0.848
	S1	0.175	0.419	0.602	0.719	0.792	0.865
	S2	0.175	0.581	0.806	0.913	0.948	0.915
Incontinence[b]	S0	0.097	0.259	0.433	0.603	0.748	0.852
	S1	0.097	0.252	0.414	0.582	0.707	0.840
	S2	0.097	0.309	0.528	0.703	0.849	0.872
Fall[b]	S0	0.041	0.162	0.300	0.435	0.547	0.667
	S1	0.041	0.163	0.292	0.440	0.559	0.681
	S2	0.041	0.214	0.410	0.588	0.728	0.809

Table 11.12 (continued)

		Age					
	Scenario	70	75	80	85	90	95
Hip fracture[b]	S0	0.025	0.032	0.044	0.066	0.119	0.167
	S1	0.025	0.030	0.044	0.068	0.108	0.138
	S2	0.025	0.076	0.147	0.247	0.357	0.447
Proxy Interview	S0	0.111	0.110	0.114	0.125	0.155	0.157
	S1	0.111	0.109	0.120	0.147	0.170	0.170
	S2	0.111	0.223	0.303	0.382	0.429	0.468
Cognitive impairment[b]	S0	0.146	0.326	0.505	0.662	0.768	0.857
	S1	0.146	0.329	0.509	0.657	0.772	0.897
	S2	0.146	0.484	0.743	0.881	0.934	0.936
Psychiatric disease[b]	S0	0.085	0.133	0.186	0.240	0.268	0.291
	S1	0.085	0.135	0.187	0.240	0.263	0.319
	S2	0.085	0.237	0.430	0.557	0.621	0.660
Depression[b]	S0	0.038	0.105	0.184	0.233	0.262	0.281
	S1	0.038	0.110	0.196	0.259	0.263	0.344
	S2	0.038	0.199	0.344	0.421	0.445	0.404
Body Mass Index[c]	S0	26.1	25.5	24.9	24.6	24.4	24.4
	S1	26.1	25.6	25.0	24.7	24.6	24.1
	S2	26.1	24.6	23.6	23.2	23.2	22.9
Current smoker[b]	S0	0.131	0.063	0.035	0.015	0.009	0.005
	S1	0.131	0.061	0.041	0.014	0.003	0.000
	S2	0.131	0.075	0.038	0.013	0.000	0.000
ADL limits[c]	S0	0.33	0.36	0.75	1.33	1.84	2.45
	S1	0.33	0.38	0.79	1.37	1.88	2.46
	S2	0.33	0.94	2.11	3.24	4.15	4.51
IADL limits[c]	S0	0.34	0.16	0.33	0.63	0.94	1.24
	S1	0.34	0.15	0.34	0.64	0.95	1.23
	S2	0.34	0.52	1.22	2.00	2.65	2.85
Poor/fair self-rated health[b]	S0	0.282	0.355	0.445	0.487	0.514	0.543
	S1	0.282	0.352	0.448	0.488	0.482	0.500
	S2	0.282	0.586	0.760	0.818	0.871	0.830

Notes: S0 = baseline; S1 = no diabetes; S2 = all low SES. See table 11.5 for explanation of abbreviations.

[a]Proportion of age 70 population surviving to the specified age.

[b]Proportion of surviving population at specified age who ever had condition.

[c]Mean in surviving population.

the baseline scenario (S0), the no-diabetes scenario (S1), and the low-SES scenario (S2). Keeping in mind that we expect the simulation model to differ from the historical cross-cohort record in AHEAD, the success of this model in plausibly mimicking observed conditions in the AHEAD population can be judged by comparing the results for scenario S0 with life table survival probabilities. Life expectancy for a cohort of white females aged seventy–seventy-four is 13.15 years from the 1996 life tables and 14.36 years in our baseline simulation. The life table probability of survival for

fifteen years for the aged seventy–seventy-four cohort is 0.535, while the corresponding survival probability in the simulation model is 0.500. For white males, the life expectancy at age seventy is 11.31 years from the life tables and 10.81 years from the S0 simulation. The fifteen-year survival probability is 0.381 from the life tables and 0.279 from the simulation model. Thus, relative to the life tables, the simulation model underpredicts female mortality and overpredicts male mortality. The comparison of annual mortality rates for white females given in figure 11.5 indicates that actual AHEAD mortality experience was more favorable than the life tables between waves 1 and 2, presumably due to selection in panel recruitment, and very close to the life tables between waves 2 and 3. Then, the baseline simulation appears to reproduce relatively accurately the cross-cohort survival experience in AHEAD. This provides a reality check for the simulation model, but also suggests that if the survival experience of a current cohort differs from the historical cross-cohort pattern, then the simulation model will miss the drift in mortality hazards that a single cohort will face in the future.

A comparison of prevalence rates for various health conditions among survivors of various ages can be made between AHEAD at wave 1, given in table 11.4, and the baseline simulation in table 11.12. There are issues of comparability in the definition of prevalence for some conditions, but the pattern that emerges is that the simulated prevalences are systematically higher than the historical prevalences and increasingly so at older ages. For example, for white females aged eighty–eighty-four, the historical and simulated prevalence rates are 0.168 versus 0.219 for cancer, 0.361 versus 0.442 for heart disease, and 0.338 versus 0.437 for cognitive impairment. One possible explanation for this is that the links from morbidity to mortality are stronger than the mortality model detects, perhaps because of underreporting of health conditions that arise prior to death, so that the simulation model underestimates the selection effect of mortality that reduces prevalence among survivors. A second possible explanation is that there is strong unobserved heterogeneity in susceptibility to various health conditions, so that cumulative prevalence is overestimated by our first-order Markov models which describe prevalence for most conditions as the result of one or more positives in a series of Bernoulli trials. It is possible to test for persistent unobserved heterogeneity by asking whether the frequency of a negative for a condition between waves 1 and 3 of AHEAD is the product of the frequencies of a negative between successive waves. When we do this, we do not find persistent unobserved heterogeneity. However, the power of the test is modest, and it is possible that even a limited degree of persistent unobserved heterogeneity is enough to explain the differences in AHEAD and the simulation.

A comparison is given in table 11.13 between median wealth and income by age in the AHEAD panel and in the baseline simulation. The historical

Table 11.13 Wealth and Income in AHEAD and in the Baseline Scenario, by Age

	70–74	75–79	80–84	85–89	90+
AHEAD cross-cohort data					
Total wealth (000)					
1st quartile	64.38	46.92	32.76	14.05	2.17
Median	144.04	112.76	94.35	71.34	40.13
3rd quartile	314.27	242.68	203.20	185.91	117.93
Nonliquid wealth (000)					
1st quartile	47.72	28.20	10.81	0.86	0.00
Median	94.94	80.48	62.01	44.19	5.44
3rd quartile	178.96	151.68	113.88	109.12	65.47
Liquid wealth (000)					
1st quartile	2.18	1.31	0.54	0.33	0.00
Median	26.19	15.97	11.89	9.73	5.40
3rd quartile	98.21	70.93	70.93	55.31	37.83
Income (000)					
1st quartile	14.15	11.97	9.76	8.68	6.74
Median	22.78	19.59	15.26	13.77	9.76
3rd quartile	34.92	31.65	28.06	25.10	15.29
Baseline simulation data					
Total wealth (000)					
1st quartile	56.67	65.69	37.07	16.08	8.22
Median	136.20	121.43	79.94	51.86	41.59
3rd quartile	299.13	206.63	136.38	91.16	74.29
Nonliquid wealth (000)					
1st quartile	42.72	37.39	17.56	2.97	–3.77
Median	91.53	74.34	47.13	29.17	21.21
3rd quartile	171.34	118.09	78.90	55.44	45.07
Liquid wealth (000)					
1st quartile	1.64	19.16	10.91	4.96	3.61
Median	22.79	43.77	31.16	22.01	19.38
3rd quartile	95.90	80.05	58.58	42.39	36.69
Income (000)					
1st quartile	13.56	13.57	11.25	8.62	7.92
Median	21.82	18.19	15.89	13.58	11.92
3rd quartile	34.70	29.58	23.12	17.81	15.85

cross-cohort data shows sharply declining wealth and income with age, and a less liquid portfolio with age, reflecting strong cohort effects as well as life-cycle and selection effects. The simulation results, which exclude cohort effects, nevertheless show even more sharply declining wealth with age and a strong shift toward a more liquid portfolio mix. If the simulation is correctly describing portfolio balance of a single cohort over its life course, then there is a strong cross-cohort effect, with older cohorts starting from retirement portfolios that are more heavily invested in housing equity and other nonliquid forms. The simulated semi-interquartile range is narrower than its historical counterpart, particularly for older households. In the

simulation, variability (defined as the ratio of the semi-interquartile range divided by the median) falls with age, whereas in the historical cross-cohort data variability rises with age. This suggests that in addition to cross-cohort effects, that there may be persistent heterogeneity in savings behavior that is not captured in our model.

11.7.3 Alternative Scenarios

Table 11.12 gives the survival probabilities and prevalences of various health conditions under our alternative no-diabetes scenario (S1) and the low-SES scenario (S2). In the no-diabetes simulation, the direct mortality risk from diabetes, and the incidence of comorbidities with diabetes are eliminated, although our simulated population will display elevated prevalence of heart disease and stroke at age seventy among former diabetics. Life expectancy at age seventy under this scenario increases from 14.36 to 14.69 years for females, and from 10.81 to 11.26 years for males. These rates imply in turn that a former diabetic's life expectancy increases by 2.72 years for females and 3.32 years for males. Other health condition prevalences that fall in the absence of diabetes are heart disease, stroke, cognitive impairment, ADL and IADL impairment, and poor/fair self-rated health. While reduction in mortality risk from one source must as a matter of accounting eventually lead to more deaths from competing risks, there are no substantial movements in prevalences of the remaining health conditions.

The alternative low-SES scenario reduces our entire aged seventy simulated population to the bottom quartile for wealth and income, gives them less than a high school education and places them in a dwelling in poor condition in an unsafe neighborhood. They are kept in this low-SES status for the remainder of their lives; that is, there is no opportunity in this simulation for households to escape low SES by a lucky change in wealth or income. However, our population displays the patterns of prevalence of health conditions established in their earlier lives with their historical SES status. This scenario is quite artificial, but it demonstrates the holistic effect on the broad spectrum of health conditions of low SES. Life expectancies at age seventy in this scenario drop dramatically, from 14.36 to 12.27 years for females, and from 10.81 to 9.56 years for males. Prevalences of cancer, heart disease, lung disease, diabetes, arthritis, incontinence, hip fractures, cognitive impairment, psychiatric disease, and depression all increase sharply, as do the number of ADL and IADL impairments. Conditions whose prevalence is not affected substantially by low SES are stroke, high blood pressure, and falls. These results indicate that *if* the associations of SES and incidence of health conditions that we find in AHEAD *were entirely* the result of direct causal links from wealth to health, then the protective effect of the prevailing pattern of higher SES is 1.26–2.08 years of

added life expectancy. Thus, our findings that for most health conditions the evidence is against direct causal links from SES to incidence do not appear to rule out a substantial cumulative effect of SES over conditions and time that induce a noticeable SES gradient in mortality. Given our specific findings against direct causal links from SES to incidence of acute conditions and mortality, the most obvious possible source for this gradient are SES-linked differences in genetic susceptibility and behavior.

We have emphasized that our stylized, hypothetical policy intervention and the changes in health they produce over the life course are strictly illustrative and should be interpreted with great caution. These finger exercises *cannot* be used to draw conclusions about any real policy initiatives. This is particularly true since we have included within our model system components that fail the invariance tests that we have emphasized must be met by a valid policy model, and because in many cases our models display *some* wealth or income gradients for incidence that we cannot with our statistical methods identify as the sole result of direct causal links. While most of these effects are not statistically significant, it is possible that in a larger or longer panel with greater statistical power, they will prove to be significant. Then, it is essential to turn to the more advanced statistical methods of Heckman (2001) and others to identify the direct causal components in these incidence associations and improve the models to achieve invariance. Only after this is done, and realistically detailed policy scenarios are considered, could policy makers take our model system seriously as a policy tool. However, we believe that our results do demonstrate that it would be useful for health policy analysis to utilize a system of invariant models with a causal chain structure to simulate policy impacts, in a framework that takes into account indirect impacts, competing hazards, and direct causal links between SES and health. We believe that analysis of the broad sweep of comorbidities and wealth effects over the life course is an important complement to the diseasecentric orientation of many medical and epidemiological studies of health outcomes.

11.8 Summary and Speculations for Further Research

This paper has used innovations in health conditions and in wealth in the AHEAD panel to carry out tests for the absence of direct causal links from SES to health, and from health conditions to wealth. By advancing beyond the detection of association to a framework in which there is some possibility of detecting the absence of causal links, this paper provides a methodology that may be useful in winnowing the list of possible direct causal mechanisms, or delimiting their domain of action. For the AHEAD sample, a panel of U.S. elderly aged seventy and older in 1993, we conclude generally that for mortality and for acute, sudden-onset diseases, the hy-

pothesis of *no* causal link from SES is accepted, and for incidence of mental problems the hypothesis is rejected. The results for chronic and degenerative diseases are mixed. We generally reject the hypothesis of no direct causal link from health conditions to total wealth changes but cannot rule out confounding of the test by invariance failures.

The pattern of results suggests that incidence of acute, sudden onset health conditions, conditioned on existing health conditions, does not exhibit a significant SES gradient, while incidence of some mental, chronic, and degenerative conditions appear to have an association to SES due to some combination of direct causal links and common unobserved behavioral or genetic factors. The results suggest that there may be an SES gradient in seeking treatment for the second class of conditions that may influence detection, or for maintaining preventative regimens that may maintain some conditions below the reporting thresholds. Our findings are not inconsistent with the possibility that for mental and chronic illnesses where the acute care procedures covered by Medicare are often inapplicable, ability to pay may be a causal factor in seeking and receiving treatment. We do not find systematic persuasive associations of health conditions and changes in total wealth, except for surviving spouses. Problems in measuring and modeling wealth changes suggest caution in concluding from these results that there is generally no direct causal link from health conditions to wealth changes.

We emphasize that our results apply only to elderly individuals in the United States, where Medicare and Medicaid programs limit out-of-pocket medical costs, particularly for acute care, and where retired status eliminates a possible direct causal link from health status to ability to work. Further, in an elderly population, common factors may be manifest in prior health conditions and economic status, so they have little impact once incidence is conditioned on prior state. Our results provide no evidence on the nature of the causal links at younger ages, during the stages of life where association of health and wealth is emerging as a consequence of some causal structure.

Future waves of the AHEAD (HRS) panel will allow the hypotheses of invariance and noncausality to be tested with greater power. This will particularly be the case when full tracking of decedents, and determination of cause of death from medical records, become part of the data. It seems likely that some of the associations we have found between changes in health and wealth will survive more detailed analysis, and that suitably defined natural or designed experiments are likely to be needed to fully unravel the causal structure underlying these associations.

The modeling structure used in this paper is parametric, and the high dimensionality of the vector of possible explanatory variables and the relatively limited information contained in binomial outcomes in the AHEAD panel make it difficult to move to a more robust nonparametric analysis.

However, we have been flexible in specifying the variable transformations that appear in our models, and we interpret our analysis as conforming in spirit, if hardly in fact, to a method of sieves approach to nonparametric analysis. One of the major limitations of our models, which would be likely to lead them to fail invariance tests in situations where a sharp test is possible, is that they do not account adequately for the multiple risk structure of health conditions and its implications for the duration patterns that can emerge, particularly over the relatively long intervals between waves. Some outcomes, such as mortality and nonfatal heart disease, are competing risks, while others, like diabetes and heart conditions, are complementary risks. For future research, we are investigating hidden Markov models in which a latent vector of propensities for all health and SES conditions follows a first-order Markov process, conditioned on demographic state, and all possible causal links across the components of this latent vector appear in the model. Given thresholds that trigger observed states, this model provides a consistent but computationally demanding data generation process for the vector of Markov states month-by-month. Within this model, it is possible to carry out joint tests for the absence of classes of causal links. The next wave of this research, incorporating wave 4 of AHEAD, will include full development of flexible multiple-risk duration models.

Appendix

Table 11A.1 Functions Related to Partial Moments of an Edgeworth Expansion

j/k	(0)	(1)	(2)	(3)	(4)
0	$\Phi(-a)$	ϕ	$a\phi + \Phi(-a)$	$(a^2 + 2)\phi$	$(a^3 + 3a)\phi + 3\Phi(-a)$
1	ϕ	$a\phi + \Phi(-a)$	$(a^2 + 2)\phi$	$(a^3 + 3a)\phi + 3\Phi(-a)$	$(a^4 + 4a^2 + 4)\phi$
2	$H_1\phi$	$(aH_1 + 1)\phi$	$(a^2H_1 + 2a)\phi + 2\Phi(-a)$	$(a^3H_1 + 3a^2 + 6)\phi$	$(a^4H_1 + 4a^3 + 12a)\phi + 12\Phi(-a)$
3	$H_2\phi$	$(aH_2 + H_1)\phi$	$(a^2H_2 + 2aH_1 + 2)\phi$	$(a^3H_2 + 3a^2H_1 + 6a)\phi + 6\Phi(-a)$	$(a^4H_2 + 4a^3H_1 + 12a^2 + 24)\phi$
4	$H_3\phi$	$(aH_3 + H_2)\phi$	$(a^2H_3 + 2aH_2 + 2H_1)\phi$	$(a^3H_3 + 3a^2H_2 + 6aH_1 + 6)\phi$	$(a^4H_3 + 4a^3H_2 + 12a^2H_1 + 24a)\phi + 24\Phi(-a)$
5	$H_4\phi$	$(aH_4 + H_3)\phi$	$(a^2H_4 + 2aH_3 + 2H_2)\phi$	$(a^3H_4 + 3a^2H_3 + 6aH_2 + 6H_1)\phi$	$(a^4H_4 + 4a^3H_3 + 24a^2H_2 + 24H_1)\phi$
6	$H_5\phi$	$(aH_5 + H_4)\phi$	$(a^2H_5 + 2aH_4 + 2H_3)\phi$	$(a^3H_5 + 3a^2H_4 + 6aH_3 + 6H_2)\phi$	$(a^4H_5 + 4a^3H_4 + 24a^2H_3 + 24H_2)\phi$

Note: Let $H_j(\varepsilon)$ denote Hermite polynomials, defined by $H_j(\varepsilon)\phi(\varepsilon) = (-1)^j\phi^{(j)}(\varepsilon)$, where $\phi^{(j)} \equiv d^j\phi/d\varepsilon^j$. This definition implies the recursion $H_j(\varepsilon) = \varepsilon H_{j-1}(\varepsilon) - dH_{j-1}(\varepsilon)/d\varepsilon$ with $H_0(\varepsilon) = 1$ and $H_1(\varepsilon) = \varepsilon$. Other leading polynomials are $H_2(\varepsilon) = \varepsilon^2 - 1$, $H_3(\varepsilon) = \varepsilon^3 - 3\varepsilon$, $H_4(\varepsilon) = \varepsilon^4 - 6\varepsilon^2 + 3$, $H_5(\varepsilon) = \varepsilon^5 - 10\varepsilon^3 + 15\varepsilon$, and $H_6(\varepsilon) = \varepsilon^6 - 15\varepsilon^4 + 45\varepsilon^2 - 15$. A useful recursion for computation is $H_j(\varepsilon) = \varepsilon H_{j-1}(\varepsilon) - (j-1)H_{j-2}(\varepsilon)$. For $j \le k$, repeated integration by parts yields

$$\int_{-\infty}^{\infty} H_j(\varepsilon)H_k(\varepsilon)\phi(\varepsilon)d\varepsilon \equiv (-1)^k \int_{-\infty}^{\infty} H_j(\varepsilon)\phi^{(k)}(\varepsilon)d\varepsilon = (-1)^{k-j} \int_{-\infty}^{\infty} H_j^{(j)}(\varepsilon)\phi^{(k-j)}(\varepsilon)d\varepsilon = (j!) \int_{-\infty}^{\infty} H_{k-j}(\varepsilon)\phi(\varepsilon)d\varepsilon.$$

Then, $\int_{-\infty}^{\infty} H_j(\varepsilon)^2\phi(\varepsilon)d\varepsilon = j!$, and for $j < k$, $\int_{-\infty}^{\infty} H_j(\varepsilon)H_k(\varepsilon)\phi(\varepsilon)d\varepsilon = 0$, since $\int_{-\infty}^{\infty} H_{k-j}(\varepsilon)\phi(\varepsilon)d\varepsilon = (-1)^{k-j}\int_{-\infty}^{\infty} \phi^{(k-j)}(\varepsilon)d\varepsilon = 0$. Define $\Psi_{jk}(a) = \int_{-\infty}^{\infty} \varepsilon^k H_j(\varepsilon)\phi(\varepsilon)d\varepsilon$. One has $\Psi_{00}(a) = \Phi(-a)$, $\Psi_{01}(a) = \phi(a)$, and integrating by parts, $\Psi_{0k}(a) = a^{k-1}\phi(a) + (k-1)\Psi_{0,k-2}(a)$. For $j > 0$, one has $\Psi_{j0}(a) = \int_{-\infty}^{\infty} H_j(\varepsilon)\phi(\varepsilon)d\varepsilon = \int_{-\infty}^{\infty}(-1)^j\phi^{(j)}(\varepsilon)d\varepsilon = -(-1)^j\phi^{(j-1)}(a) = H_{j-1}(a)\phi(a)$. Integration by parts for $k > 0$ gives $\Psi_{jk}(a) = \int_{-\infty}^{\infty} \varepsilon^k(-1)^j\phi^{(j)}(\varepsilon)d\varepsilon = -a^k(-1)^j\phi^{(j-1)}(a) - k\int_{-\infty}^{\infty} \varepsilon^{k-1}(-1)^j\phi^{(j-1)}(\varepsilon)d\varepsilon = a^kH_{j-1}(a)\phi(a) + k\Psi_{j-1,k-1}(a)$. The table below gives $\Psi_{jk}(a)$ for $k \le 4$ and $j \le 6$. An expansion $f(\varepsilon) = \Sigma_{j=0}^{J} \gamma_j H_j(\varepsilon)\phi(\varepsilon)$ satisfies $\int_{-\infty}^{\infty} \varepsilon^k f(\varepsilon)d\varepsilon = \Sigma_{j=0}^{J} \gamma_j\Psi_{jk}(a)$. Then, $\int_{-\infty}^{\infty} f_j(\varepsilon)d\varepsilon = \gamma_0$, $\int_{-\infty}^{\infty} \varepsilon f_j(\varepsilon)d\varepsilon = \gamma_1$, $\int_{-\infty}^{\infty} \varepsilon^2 f_j(\varepsilon)d\varepsilon = \gamma_0 + 2\gamma_2$, $\int_{-\infty}^{\infty} \varepsilon^3 f_j(\varepsilon)d\varepsilon = 3\gamma_1 + 6\gamma_3$, and $\int_{-\infty}^{\infty} \varepsilon^4 f_j(\varepsilon)d\varepsilon = 3\gamma_0 + 12\gamma_2 + 24\gamma_4$. When $J = 4$, $\gamma_0 = 1$, and $\gamma_1 = \gamma_2 = \gamma_3 = 0$, a sufficient condition for f to be positive is $0 \le \gamma_4 < 1/6$.

The appendix tables published in this chapter are an *updated* version of those originally cited in the 2003 *Journal of Econometrics* article. As such, some of the numerical results reported in the paper do not directly match those presented in the appendix as published in this chapter. The original appendix containing detailed parameter estimates can be found at http://elsa.berkeley.edu/wp/hww/hww202.html.

Table 11A.2 Transformed Variables

	Wave 1–2				Wave 2–3			
	Variable	*N*	Mean	SD	Variable	*N*	Mean	SD
				Females				
Demographic/SES variables								
1st quartile income indicator	XQ1I1	3,992	0.0655	0.1068	XQ1I2	3,580	0.0613	0.1043
4th quartile income indicator	XQ4I1	3,992	0.0385	0.0825	XQ4I2	3,578	0.0339	0.0771
Neighborhood safety poor/fair	XHOODPF1	3,992	0.0366	0.0859	XHOODPF2	3,580	0.0324	0.0814
House condition poor/fair	XCONDPF1	3,992	0.0361	0.0858	XCONDPF2	3,580	0.0334	0.0827
Own residence?	XDNHOUS1	3,992	0.1306	0.1150	XDNHOUS2	3,578	0.1209	0.1139
Educ > 10 yrs indicator	XHS1	3,992	0.1270	0.1147	XHS2	3,578	0.1218	0.1135
Educ > 14 yrs indicator	XCOLL1	3,992	0.0222	0.0665	XCOLL2	3,580	0.0201	0.0626
	XAS701S	3,992	23.2281	18.2049	XAS702S	3,578	26.2357	18.1556
	XAS801S	3,992	5.4289	10.3034	XAS802S	3,579	6.6345	11.1556
Never married?	XNEVMARR1	3,992	0.0082	0.0434	XNEVMARR2	3,580	0.0076	0.0413
Widow?	XWIDOW1	3,992	0.1297	0.1180	XWIDOW2	3,579	0.1309	0.1168
Divorced/Separated?	XDIVSEP1	3,992	0.0135	0.0550	XDIVSEP2	3,580	0.0133	0.0547
Mother's death age	XMAGEDI1	3,992	16.0466	5.7117	XMAGEDI2	3,578	15.4259	6.2546
Father's death age	XPAGEDI1	3,992	15.6112	5.3007	XPAGEDI2	3,578	15.0117	5.8754
Ever smoke?	XSMOKEV1	3,992	0.0814	0.1115	XSMOKEV2	3,579	0.0778	0.1096
Health condition prevalence								
Cancer[a]	XCANCER1	3,992	0.0274	0.0754	XCANCER2	3,579	0.0284	0.0760
Heart attack/Condition[a]	XHEART1	3,992	0.0657	0.1057	XHEART2	3,579	0.0698	0.1072
Stroke[a]	XSTROKE1	3,992	0.0186	0.0636	XSTROKE2	3,580	0.0209	0.0667
Lung disease[a]	XLUNG1	3,992	0.0225	0.0694	XLUNG2	3,580	0.0211	0.0672
Diabetes[a]	XDIABET1	3,992	0.0300	0.0787	XDIABET2	3,580	0.0331	0.0819
HBP[a]	XHIGHBP1	3,992	0.1209	0.1177	XHIGHBP2	3,580	0.1195	0.1167

(*continued*)

Table 11A.2 (continued)

	Wave 1–2				Wave 2–3			
	Variable	*N*	Mean	SD	Variable	*N*	Mean	SD
Arthritis[a]	XARTHRT1	3,992	0.0672	0.1064	XARTHRT2	3,580	0.0690	0.1067
Incontinence *in last 12 months*	XINCONT1	3,992	0.0536	0.0984	XINCONT2	3,579	0.0729	0.1073
Fall requiring treatment[a]	XFALL1	3,992	0.0210	0.0669	XFALL2	3,580	0.0434	0.0903
Hip fracture[a]	XHIPFRC1	3,992	0.0133	0.0545	XHIPFRC2	3,580	0.0149	0.0570
Proxy interview	XPROXYW1	3,992	0.0197	0.0652	XPROXYW2	3,580	0.0274	0.0755
Cognitive impairment	XCOGIM1	3,992	0.0604	0.1033	XCOGIM2	3,580	0.0785	0.1109
Psychiatric problems[a]	XPSYCH1	3,992	0.0275	0.0755	XPSYCH2	3,580	0.0320	0.0806
Depression	XDEPRES1	3,992	0.0263	0.0745	XDEPRES2	3,580	0.0226	0.0693
Low BMI	XLOBMI1	3,983	0.0445	0.1704	XLOBMI2	3,579	0.0557	0.2012
High BMI	XHIBMI1	3,983	0.4588	0.7938	XHIBMI2	3,578	0.4180	0.7682
Current smoker	XSMOKNOW1	3,992	0.0186	0.0635	XSMOKNOW2	3,580	0.0154	0.0583
No. of ADLs	XNUMADL1	3,992	0.1836	0.3375	XNUMADL2	3,580	0.1884	0.3572
No. of ADLs	XNUMIADL1	3,992	0.1473	0.2755	XNUMIADL2	3,580	0.1202	0.2619
Fair/Poor self-reported health	XDHLTH1	3,992	0.0865	0.1143	XDHLTH2	3,580	0.0832	0.1125
Health condition incidence								
Cancer[a]	XJCANCER2	3,951	0.0094	0.0459	XJCANCER3	3,547	0.0092	0.0449
Heart attack/Condition[a]	XJHEART2	3,951	0.0292	0.0780	XJHEART3	3,548	0.0316	0.0801
Stroke[a]	XJSTROKE2	3,951	0.0126	0.0533	XJSTROKE3	3,548	0.0154	0.0578
Lung disease[a]	XILUNG2	3,951	0.0050	0.0339	XILUNG3	3,548	0.0068	0.0393
Diabetes[a]	XIDIABET2	3,580	0.0054	0.0352	XIDIABET3	3,090	0.0052	0.0345
HBP[a]	XIHIGHBP2	3,580	0.0120	0.0513	XIHIGHBP3	3,090	0.0119	0.0512
Arthritis[a]	XIARTHRT2	3,580	0.0274	0.0758	XIARTHRT3	3,090	0.0244	0.0708

Incontinence *in last 12 months*	XJINCONT2	3,951	0.0588	0.1015	XJINCONT3	3,547	0.0631	0.1029
Fall requiring treatment[a]	XJFALL2	3,951	0.0340	0.0826	XJFALL3	3,548	0.0362	0.0845
Hip fracture[a]	XJHIPFRC2	3,951	0.0066	0.0389	XJHIPFRC3	3,548	0.0074	0.0409
Proxy interview	XPROXYW2	3,580	0.0279	0.0762	XPROXYW3	3,090	0.0331	0.0819
Cognitive impairment	XICOGIM2	3,992	0.0233	0.0705	XICOGIM3	3,580	0.0174	0.0611
Psychiatric problems[a]	XIPSYCH2	3,580	0.0114	0.0506	XIPSYCH3	3,090	0.0096	0.0464
Depression	XIDEPRES2	3,580	0.0131	0.0539	XIDEPRES3	3,090	0.0151	0.0575
BMI better Indicator	XBMIBT2	3,992	0.0402	0.0889	XBMIBT3	3,580	0.0378	0.0863
BMI worse Indicator	XBMIWS2	3,992	0.0341	0.0829	XBMIWS3	3,579	0.0360	0.0844
Current smoker	XSMOKNOW2	3,580	0.0154	0.0582	XSMOKNOW3	3,090	0.0127	0.0531
No. of ADLs	XNUMADL2	3,951	0.2544	0.4351	XNUMADL3	3,548	0.2994	0.4731
No. of ADLs	XNUMIADL2	3,951	0.1760	0.3337	XNUMIADL3	3,548	0.2038	0.3522
Fair/Poor self-reported health	XDHLTH2	3,580	0.0846	0.1135	XDHLTH3	3,089	0.0948	0.1149
			Males					
Demographic/SES variables								
1st quartile income indicator	XQ1I1	2,497	0.0525	0.0988	XQ1I2	2,161	0.0529	0.0992
4th quartile income indicator	XQ4I1	2,497	0.0419	0.0830	XQ4I2	2,158	0.0355	0.0764
Neighborhood safety poor/fair	XHOODPF1	2,497	0.0310	0.0797	XHOODPF2	2,161	0.0294	0.0777
House condition poor/fair	XCONDPF1	2,497	0.0318	0.0810	XCONDPF2	2,161	0.0297	0.0783
Own residence?	XDNHOUS1	2,497	0.1430	0.1087	XDNHOUS2	2,158	0.1323	0.1089
Educ > 10 yrs indicator	XHS1	2,497	0.1126	0.1111	XHS2	2,158	0.1065	0.1091
Educ > 14 yrs indicator	XCOLL1	2,497	0.0267	0.0695	XCOLL2	2,158	0.0233	0.0642
	XAS701S	2,497	19.4711	16.6014	XAS702S	2,158	21.5620	16.5232
	XAS801S	2,497	3.8552	8.2739	XAS802S	2,160	4.5202	8.8800
Never married?	XNEVMARR1	2,497	0.0055	0.0349	XNEVMARR2	2,161	0.0048	0.0330
Widow?	XWIDOW1	2,497	0.0399	0.0882	XWIDOW2	2,161	0.0426	0.0895

(*continued*)

Table 11A.2 (continued)

	Wave 1–2				Wave 2–3			
	Variable	*N*	Mean	SD	Variable	*N*	Mean	SD
Divorced/Separated?	XDIVSEP1	2,497	0.0104	0.0486	XDIVSEP2	2,159	0.0102	0.0478
Mother's death age	XMAGEDI1	2,497	15.1925	6.3133	XMAGEDI2	2,158	14.4231	6.8565
Father's death age	XPAGEDI1	2,497	14.7368	6.0174	XPAGEDI2	2,158	13.9436	6.5630
Ever smoke?	XSMOKEV1	2,497	0.1589	0.1064	XSMOKEV2	2,159	0.1496	0.1077
Health condition prevalence								
Cancer[a]	XCANCER1	2,497	0.0306	0.0779	XCANCER2	2,161	0.0333	0.0801
Heart attack/Condition[a]	XHEART1	2,497	0.0740	0.1072	XHEART2	2,160	0.0748	0.1068
Stroke[a]	XSTROKE1	2,497	0.0214	0.0674	XSTROKE2	2,161	0.0231	0.0692
Lung disease[a]	XLUNG1	2,497	0.0308	0.0792	XLUNG2	2,161	0.0284	0.0759
Diabetes[a]	XDIABET1	2,497	0.0290	0.0765	XDIABET2	2,161	0.0298	0.0772
HBP[a]	XHIGHBP1	2,497	0.0916	0.1127	XHIGHBP2	2,160	0.0927	0.1117
Arthritis[a]	XARTHRT1	2,497	0.0457	0.0927	XARTHRT2	2,161	0.0442	0.0911
Incontinence *in last 12 months*	XINCONT1	2,497	0.0273	0.0744	XINCONT2	2,159	0.0433	0.0891
Fall requiring treatment[a]	XFALL1	2,497	0.0119	0.0510	XFALL2	2,160	0.0218	0.0671
Hip fracture[a]	XHIPFRC1	2,497	0.0079	0.0424	XHIPFRC2	2,161	0.0090	0.0450
Proxy interview	XPROXYW1	2,497	0.0293	0.0778	XPROXYW2	2,161	0.0325	0.0810
Cognitive impairment	XCOGIM1	2,497	0.0545	0.0995	XCOGIM2	2,160	0.0705	0.1069
Psychiatric problems[a]	XPSYCH1	2,497	0.0179	0.0622	XPSYCH2	2,161	0.0210	0.0668
Depression	XDEPRES1	2,497	0.0168	0.0609	XDEPRES2	2,161	0.0136	0.0548
Low BMI	XLOBMI1	2,492	0.0179	0.1042	XLOBMI2	2,161	0.0214	0.1199
High BMI	XHIBMI1	2,492	0.3524	0.6063	XHIBMI2	2,160	0.3318	0.6056
Current smoker	XSMOKNOW1	2,497	0.0283	0.0762	XSMOKNOW2	2,161	0.0196	0.0645

No. of ADLs	XNUMADL1	2,497	0.1310	0.2918	XNUMADL	2,160	0.1323	0.3130
No. of ADLs	XNUMIADL1	2,497	0.1253	0.2654	XNUMIADL2	2,161	0.0860	0.2264
Fair/Poor self-reported health	XDHLTH1	2,497	0.0804	0.1113	XDHLTH2	2,161	0.0768	0.1091
Health condition incidence								
Cancer[a]	XJCANCER2	2,481	0.0162	0.0591	XJCANCER3	2,137	0.0160	0.0588
Heart attack/Condition[a]	XJHEART2	2,481	0.0291	0.0769	XJHEART3	2,137	0.0347	0.0821
Stroke[a]	XJSTROKE2	2,481	0.0125	0.0524	XJSTROKE3	2,137	0.0141	0.0552
Lung disease[a]	XILUNG2	2,481	0.0061	0.0373	XILUNG3	2,137	0.0074	0.0408
Diabetes[a]	XIDIABET2	2,162	0.0047	0.0323	XIDIABET3	1,781	0.0053	0.0341
HBP[a]	XIHIGHBP2	2,162	0.0103	0.0470	XIHIGHBP3	1,781	0.0099	0.0463
Arthritis[a]	XIARTHRT2	2,162	0.0202	0.0655	XIARTHRT3	1,780	0.0211	0.0649
Incontinence *in last 12 months*	XJINCONT2	2,481	0.0378	0.0854	XJINCONT3	2,135	0.0398	0.0865
Fall requiring treatment[a]	XJFALL2	2,481	0.0185	0.0629	XJFALL3	2,135	0.0191	0.0629
Hip fracture[a]	XJHIPFRC2	2,481	0.0033	0.0280	XJHIPFRC3	2,137	0.0029	0.0256
Proxy interview	XPROXYW2	2,162	0.0329	0.0817	XPROXYW3	1,781	0.0342	0.0825
Cognitive impairment	XICOGIM2	2,497	0.0228	0.0690	XICOGIM3	2,161	0.0171	0.0596
Psychiatric problems[a]	XIPSYCH2	2,162	0.0087	0.0444	XIPSYCH3	1,781	0.0065	0.0383
Depression	XIDEPRES2	2,162	0.0086	0.0442	XIDEPRES3	1,780	0.0111	0.0492
BMI better Indicator	XBMIBT2	2,497	0.0355	0.0833	XBMIBT3	2,161	0.0316	0.0788
BMI worse Indicator	XBMIWS2	2,497	0.0300	0.0781	XBMIWS3	2,161	0.0282	0.0748
Current smoker	XSMOKNOW2	2,162	0.0204	0.0660	XSMOKNOW3	1,780	0.0169	0.0603
No. of ADLs	XNUMADL2	2,481	0.2103	0.4204	XNUMADL3	2,136	0.2252	0.4362
No. of ADLs	XNUMIADL2	2,481	0.1463	0.3154	XNUMIADL3	2,137	0.1594	0.3159
Fair/Poor self-reported health	XDHLTH2	2,162	0.0790	0.1106	XDHLTH3	1,780	0.0883	0.1115

Notes: N = number of observations; SD = standard deviation. See table 11.5 for explanations of abbreviations.

Table 11A.3 Prevalence Regressions—Causality Tests and Relative Odds

Variable	No. of Observations		Noncausality		Relative Odds (high vs. low SES)			
					Female		Male	
	Female	Male	Female (p-value)	Male (p-value)	Odds	SE	Odds	SE
CANCER	3,153	2,028	0.0148	0.1963	1.92	0.59	0.97	0.28
HEART	3,153	2,028	0.0000	0.0971	0.46**	0.07	0.74**	0.10
STROKE	3,153	2,028	0.0061	0.4981	0.70	0.23	0.41**	0.15
LUNG	3,153	2,028	0.0005	0.0007	0.31**	0.10	0.42**	0.12
DIABETES	3,153	2,028	0.0000	0.4123	0.19**	0.07	0.61	0.21
HIGH BLOOD PRESSURE	3,153	2,028	0.1024	0.0153	0.82**	0.08	0.63**	0.08
ARTHRITIS	3,153	2,028	0.0076	0.0770	0.76**	0.12	0.61**	0.15
INCONTINENCE	3,153	2,028	0.0114	0.2886	0.87	0.14	0.77	0.24
FALL	3,153	2,028	0.0034	0.1508	0.60**	0.17	0.43**	0.25
HIP FRACTURE	3,153	2,028	0.7840	0.5262	0.70	0.28	0.81	0.63
PROXY INTERVIEW	3,153	2,028	0.0000	0.0000	0.90	0.44	0.16**	0.06
COGNITIVE IMPAIRMENT	3,153	2,028	0.0000	0.0000	0.55**	0.09	0.14**	0.04
PSYCHIATRIC	3,153	2,028	0.2938	0.3554	1.16	0.31	0.56	0.23
DEPRESSION	3,153	2,028	0.0000	0.0098	0.36**	0.11	0.19**	0.10
BODY MASS INDEX	—	—	0.0000	0.0122	—	—	—	—
CURRENT SMOKER	3,145	2,027	0.0591	0.0001	0.21**	0.27	0.23**	0.09
ACTIVITIES DAILY LIVING	—	—	0.4505	0.0008	—	—	—	—
INSTRUMENTAL ACTIVITIES DAILY LIVING	—	—	0.0000	0.0092	—	—	—	—
SELF REPORTED HEALTH	3,147	2,027	0.0000	0.0000	0.32**	0.05	0.37**	0.06

Note: SE = standard error.

**Relative risks that are significantly different from one at the 5 percent level.

Table 11A.4 Incidence Regressions—Tests (*p*-values)

	Invariance			
	With SES		Without SES	
Variable	Female	Male	Female	Male
CANCER	0.5275	0.0155	0.3088	0.0071
CANCER—no previous	0.8573	0.1291	0.8116	0.0639
CANCER—previous	0.0682	0.0443	0.0789	0.0835
HEART	0.0854	0.6901	0.0517	0.6188
HEART—no previous	0.1704	0.8228	0.0604	0.4982
HEART—previous	0.5468	0.6925	0.4918	0.7248
STROKE	0.3842	0.2036	0.2663	0.1351
STROKE—no previous	0.1719	0.2584	0.1603	0.1799
STROKE—previous	0.9073	0.0448	0.7838	0.0866
MORTALITY	0.2212	0.3777	0.3153	0.4915
LUNG	0.5518	0.6892	0.4825	0.5021
DIABETES	0.1893	0.2340	0.2810	0.1282
HIGH BLOOD PRESSURE	0.0066	0.3934	0.0244	0.1718
ARTHRITIS	0.0461	0.0709	0.0319	0.1665
INCONTINENCE	0.7809	0.3505	0.5968	0.6817
INCONTINENCE—no previous	0.7057	0.1019	0.4261	0.5548
INCONTINENCE—previous	0.9620	0.7931	0.8921	0.7303
FALL	0.9035	0.2626	0.7904	0.2072
FALL—no previous	0.7918	0.1401	0.6119	0.1332
FALL—previous	0.3649	0.0000	0.3576	0.0000
HIP FRACTURE	0.4915	0.1261	0.2803	0.0530
HIP FRACTURE—no previous	0.9297	0.2997	0.7237	0.1227
PROXY INTERVIEW	0.3008	0.5908	0.4646	0.3777
PROXY INTERVIEW—no previous	0.1275	0.7948	0.4442	0.6064
COGNITIVE IMPAIRMENT	0.0053	0.2878	0.0083	0.1425
PSYCHIATRIC	0.1266	0.2949	0.0516	0.3647
DEPRESSION	0.2109	0.9437	0.2989	0.9343
BODY MASS INDEX	0.2599	0.0089	0.2511	0.0020
CURRENT SMOKER	0.4203	0.7097	0.2140	0.5042
CURRENT SMOKER—no previous	0.1331	0.0328	0.0783	0.0401
CURRENT SMOKER—previous	0.7966		0.7332	
ACTIVITIES DAILY LIVING	0.0010	0.0211	0.0069	0.0377
INSTRUMENTAL ACTIVITIES DAILY LIVING	0.6031	0.0077	0.3883	0.0027
SELF RATED HEALTH	0.3761	0.6342	0.3408	0.6065
SELF RATED HEALTH—no previous	0.5144	0.2515	0.5578	0.2151
SELF RATED HEALTH—previous	0.2395	0.1652	0.1763	0.1886

(*continued*)

Table 11A.4 (continued)

	Noncausality					
	Wave 1–2		Wave 2–3		Wave 1–3	
	Female	Male	Female	Male	Female	Male
CANCER	0.9874	0.5883	0.4080	0.1653	0.6133	0.1980
CANCER—no previous	0.7038	0.6584	0.2886	0.1730	0.3130	0.1658
CANCER—previous	0.1428	0.2277	0.4719	0.0168	0.2989	0.0359
HEART	0.5398	0.0538	0.3963	0.9964	0.3978	0.2430
HEART—no previous	0.8722	0.3444	0.7495	0.4118	0.7360	0.0393
HEART—previous	0.1040	0.0707	0.1533	0.7372	0.0192	0.1716
STROKE	0.7567	0.8367	0.5845	0.0278	0.6573	0.0593
STROKE—no previous	0.4147	0.9546	0.6708	0.0639	0.7128	0.2035
STROKE—previous	0.8239	0.3012	0.8819	0.1548	0.7986	0.3827
MORTALITY	0.0980	0.2753	0.8830	0.3056	0.6516	0.3636
LUNG	0.1657	0.1739	0.8663	0.1224	0.3431	0.0097
DIABETES	0.6152	0.1413	0.0197	0.2361	0.1096	0.0250
HIGH BLOOD PRESSURE	0.3422	0.9956	0.0831	0.9409	0.5339	0.9897
ARTHRITIS	0.1786	0.0285	0.2423	0.7129	0.0848	0.3947
INCONTINENCE	0.7627	0.0191	0.2536	0.7999	0.1628	0.4632
INCONTINENCE—no previous	0.9140	0.0369	0.6018	0.1970	0.4530	0.6408
INCONTINENCE—previous	0.8996	0.2471	0.1675	0.6643	0.1490	0.2440
FALL	0.5325	0.6875	0.953	0.4539	0.6003	0.5918
FALL—no previous	0.5015	0.6973	0.9435	0.2509	0.5576	0.5321
FALL—previous	0.3286	0.0000	0.8034	0.0000	0.6959	0.0529
HIP FRACTURE	0.6125	0.5974	0.3700	0.6656	0.1586	0.4304
HIP FRACTURE—no previous	0.4606	0.4610	0.7058	0.7249	0.1277	0.2344
PROXY INTERVIEW	0.0172	0.4856	0.8700	0.1267	0.2540	0.0317
PROXY INTERVIEW—no previous	0.0016	0.7935	0.5408	0.1207	0.0874	0.0732
COGNITIVE IMPAIRMENT	0.0007	0.2587	0.2591	0.1995	0.0016	0.0262
PSYCHIATRIC	0.0428	0.2376	0.2138	0.0685	0.0036	0.0649
DEPRESSION	0.0071	0.1433	0.2605	0.3770	0.0113	0.0653
BODY MASS INDEX	0.1876	0.8388	0.9308	0.7108	0.6729	0.6640
CURRENT SMOKER	0.6031	0.2821	0.9741	0.6927	0.6251	0.1286
CURRENT SMOKER—no previous	0.3355	0.1965	0.8744	0.9802	0.5148	0.9615
CURRENT SMOKER—previous	0.3750	0.3248	0.5473	0.3248	0.2736	0.0249
ACTIVITIES DAILY LIVING	0.0771	0.0137	0.3460	0.9714	0.8278	0.3577
INSTRUMENTAL ACTIVITIES DAILY LIVING	0.3738	0.0510	0.8166	0.2426	0.2408	0.0085
SELF RATED HEALTH	0.0133	0.0476	0.0996	0.3006	0.0013	0.0204
SELF RATED HEALTH—no previous	0.0149	0.1874	0.2022	0.1210	0.0071	0.0337
SELF RATED HEALTH—previous	0.2250	0.1955	0.5565	0.2555	0.1934	0.1752

Table 11A.4 (continued)

	Joint (Invariance + Noncausality)		Relative Odds (high vs. low SES)			
			Female		Male	
	Female	Male	Odds	SE	Odds	SE
CANCER	0.5999	0.0124	1.3099	0.4606	0.7316	0.2699
CANCER—no previous	0.7778	0.0844	1.2423	0.4835	0.6733	0.2934
CANCER—previous	0.0666	0.0110	5.8494	7.7533	2.6866	3.4206
HEART	0.0986	0.5708	0.9739	0.2107	1.0196	0.2379
HEART—no previous	0.2780	0.4464	0.8190	0.2525	1.3003	0.5022
HEART—previous	0.1686	0.5161	1.1323	0.3180	0.8778	0.2629
STROKE	0.4812	0.0800	0.7308	0.2537	0.9363	0.3517
STROKE—no previous	0.2711	0.1893	0.6787	0.2666	0.8182	0.3603
STROKE—previous	0.9477	0.0537	1.1538	0.9298	1.6050	1.2472
MORTALITY	0.3076	0.3596	0.6881	0.2069	1.1677	0.4066
LUNG	0.5039	0.2032	0.3272	0.1731	0.5043	0.4315
DIABETES	0.1013	0.0622	0.6932	0.4119	1.4837	3.1948
HIGH BLOOD PRESSURE	0.0127	0.6660	0.8959	0.2884	0.7425	0.3568
ARTHRITIS	0.0204	0.0816	1.0309	0.2177	0.5433	0.1596
INCONTINENCE	0.6148	0.3733	0.8501	0.1268	1.1093	0.3031
INCONTINENCE—no previous	0.6954	0.1583	0.7738	0.1983	1.0196	0.3574
INCONTINENCE—previous	0.8574	0.6836	0.9398	0.0903	1.1258	0.2871
FALL	0.9133	0.3285	0.9845	0.1947	0.9228	0.3132
FALL—no previous	0.8079	0.1809	1.0259	0.2536	0.8638	0.3220
FALL—previous	0.4700	0.0000	1.0032	0.2718	89.6130	267.8210
HIP FRACTURE	0.3475	0.1434	0.3270	0.2205	0.2096	0.2770
HIP FRACTURE—no previous	0.7845	0.2381	0.2289	0.1690	0.1397	0.1924
PROXY INTERVIEW	0.2482	0.2610	0.4587	0.1610	0.4756	0.1750
PROXY INTERVIEW—no previous	0.0610	0.5310	0.2666	0.1094	0.3992	0.2009
COGNITIVE IMPAIRMENT	0.0002	0.0888	0.7795	0.1721	0.5920	0.1852
PSYCHIATRIC	0.0123	0.1372	0.3942	0.1529	0.1589	0.1243
DEPRESSION	0.0422	0.7534	0.3753	0.1453	0.2856	0.1530
BODY MASS INDEX	0.3452	0.0190				
CURRENT SMOKER	0.4918	0.5276	0.4609	0.3702	0.9187	1.1083
CURRENT SMOKER—no previous	0.1652	0.0970	0.1055	0.1581	19.9184	132.7460
CURRENT SMOKER—previous	0.7141		0.9262	0.1595	0.9499	0.1758
ACTIVITIES DAILY LIVING	0.0036	0.0240				
INSTRUMENTAL ACTIVITIES DAILY LIVING	0.5089	0.0008				
SELF RATED HEALTH	0.0444	0.2784	0.6749	0.0780	0.6445	0.0919
SELF RATED HEALTH—no previous	0.1380	0.0907	0.5892	0.1224	0.5066	0.1290
SELF RATED HEALTH—previous	0.1773	0.1159	0.8500	0.0754	0.9227	0.0824

Notes: SE = standard error. NC = nonconvergence.

Table 11A.5 Total Wealth Regressions—(*t*-statistics)

	Couples		Spouse Died		Single	
Variable	Estimate	*t*-statistic	Estimate	*t*-statistic	Estimate	*t*-statistic
XONE1	0.0610	12.72	0.0917	5.35	0.0552	4.70
XONE2	0.0487	9.94	0.0755	3.46	0.0430	3.45
XLWLTH12	–0.0528	–10.98	–0.530	–4.23	–0.0503	–7.93
XNWLTH12	–0.0673	–8.23	–0.1033	–5.61	–0.0629	–5.52
XQ1I12	–0.0037	–3.88	–0.0052	–1.58	–0.0014	–0.53
XQ4I12	0.0098	6.98	0.0040	0.58	0.0066	2.68
XHOODPF12	–0.0001	–0.05	0.0066	1.59	0.0030	1.02
XCONDPF12	–0.0031	–2.48	–0.0027	–0.52	0.0000	0.01
XDNHOUS12	0.0085	5.29	0.0124	2.74	0.0015	0.66
M_XHS12	0.0007	0.79	0.0019	0.51	0.0035	1.67
M_XCOLL12	0.0008	0.57	0.0034	0.62	0.0034	0.86
M_XAS701S	0.0000	1.69	0.0001	0.67	0.0001	1.94
M_XAS801S	0.0000	–0.80	0.0000	0.05	–0.0001	–1.29
M_XAS702S	0.0000	1.22	0.0000	–0.37	0.0001	1.82
M_XAS802S	0.0000	–0.58	0.0001	0.47	–0.0001	–0.76
M_XNEVMARR12					0.0131	1.62
M_XWIDOW12					0.0123	1.64
M_XDIVSEP12					0.0138	1.73
M_XMAGEDI12	0.0000	1.24	–0.0001	–1.06	0.0000	–0.89
M_XPAGEDI12	0.0000	–0.10	0.0001	0.86	–0.0001	–1.40
M_XSMOKEV12	–0.0012	–1.17	–0.0046	–1.40	0.0012	0.44
M_XCANCER12	0.0003	0.24	0.0025	0.76	0.0004	0.15
M_XHEART12	–0.0004	–0.45	0.0009	0.28	–0.0007	–0.33
M_XSTROKE12	–0.0003	–0.16	–0.0024	–0.47	–0.0015	–0.49
M_XLUNG12	0.0039	2.15	–0.0057	–1.51	–0.0009	–0.33
M_XDIABET12	–0.0013	–1.13	–0.0004	–0.11	0.0024	0.91
M_XHIGHBP12	–0.0003	–0.36	0.0035	1.19	–0.0002	–0.09
M_XARTHRT12	–0.0007	–0.56	0.0015	0.41	–0.0011	–0.54
M_XINCONT12	0.0012	0.87	–0.0061	–1.77	–0.0008	–0.30
M_XFALL12	–0.0018	–1.26	–0.0021	–0.39	–0.0051	–0.151
M_XHIPFRC12	–0.0029	–0.84	0.0042	0.57	0.0059	1.49
M_XPROXYW12	–0.0004	–0.20	–0.0027	–0.69	0.0055	1.04
M_XCOGIM12	0.0011	0.88	–0.0048	–1.69	–0.0024	–1.10
M_XPSYCH12	–0.0012	–0.61	0.0055	1.25	0.0015	0.43
M_XDEPRES12	–0.0001	–0.02	0.0033	0.69	–0.0015	–0.54
M_XLOBMI12	–0.0009	–0.20	–0.0002	–0.09	0.0020	0.99
M_XHIBMI12	0.0000	0.25	0.0002	0.44	–0.0003	–0.88
M_XSMOKNOW12	–0.0029	–1.71	–0.0030	–0.54	–0.0045	–1.59
M_XNUMADL12	0.0001	0.17	–0.0001	–0.03	–0.0010	–1.05
M_XNUMIADL12	–0.0010	–0.93	0.0002	0.05	0.0014	1.16
M_XDHLTH12	0.0000	–0.04	0.0022	0.53	0.0039	1.62
M_XJCANCER23	0.0007	0.22	0.0011	0.10	0.0091	2.21
M_XJHEART23	0.0007	0.29	–0.0023	–0.19	0.0029	1.00
M_XJSTROKE23	0.0043	1.22	–0.0024	–0.22	0.0051	1.33
M_XILUNG23	–0.0021	–1.08	–0.0051	–0.35	–0.0033	–1.22
M_XIDIABET23	–0.0012	–0.48	0.0250	1.94	–0.0015	–0.35
M_XIHIGHBP23	–0.0006	–0.18	–0.0174	–1.15	0.0009	0.34
M_XIARTHRT23	–0.0008	–0.53	–0.0016	–0.25	–0.0020	–0.67

Table 11A.5 (continued)

	Couples		Spouse Died		Single	
Variable	Estimate	*t*-statistic	Estimate	*t*-statistic	Estimate	*t*-statistic
M_XJINCONT23	–0.0006	–0.39	0.0015	0.17	0.0007	0.31
M_XJFALL23	0.0003	0.24	0.0048	0.34	–0.0029	–1.30
M_XJHIPFRC23	–0.0044	–0.57	–0.0006	–0.02	–0.0009	–0.14
M_XPROXYW23	–0.0005	–0.29	0.0017	0.20	–0.0010	–0.27
M_XICOGIM23	0.0001	0.06	–0.0057	–0.48	0.0020	0.84
M_XIPSYCH23	–0.0003	–0.18	0.0008	0.06	0.0046	1.54
M_XIDEPRES23	–0.0016	–0.72	0.0009	0.11	–0.0032	–1.07
M_XBMIBT23	0.0004	0.36	–0.0020	–0.26	0.0022	1.14
M_XBMIWS23	–0.0013	–1.00	–0.0031	–0.34	0.0008	0.39
M_XSMOKNOW23	0.0040	2.08	–0.0069	–0.38	–0.0006	–0.24
M_XNUMADL23	–0.0001	–0.12	–0.0055	–1.63	–0.0003	–0.56
M_XNUMIADL23	0.0004	0.41	0.0006	0.20	–0.0008	–1.06
M_XDHLTH23	–0.0007	–0.67	–0.0118	–1.32	0.0015	0.90
F_XONE1					0.0753	12.84
F_XONE2					0.0656	10.87
F_XLWLTH12					–0.0503	–16.12
F_XNWLTH12					–0.0640	–11.29
F_XQ1I12					–0.0006	–0.81
F_XQ4I12					0.0089	7.79
F_XHOODPF12					–0.0010	–1.12
F_XCONDPF12					–0.0012	–1.36
F_XDNHOUS12					0.0035	3.54
F_XHS12	0.0008	0.75	0.0069	1.66	0.0016	2.16
F_XCOLL12	0.0019	1.34	0.0029	0.49	0.0026	1.80
F_XAS701S	0.0000	1.14	–0.0001	–0.83	0.0000	–0.09
F_XAS801S	–0.0001	–0.75	0.0000	0.04	0.0000	0.04
F_XAS702S	0.0000	–1.88	0.0000	0.66	0.0000	–0.35
F_XAS802S	0.0001	1.96	–0.0001	–0.37	0.0000	0.51
F_XNEVMARR12					0.0023	0.50
F_XWIDOW12					0.0010	0.23
F_XDIVSEP12					0.0001	0.01
F_XMAGEDI12	0.0000	1.08	0.0000	–0.02	0.0000	0.44
F_XPAGEDI12	0.0000	–0.79	0.0000	–0.40	0.0000	–0.47
F_XSMOKEV12	0.0024	2.58	0.0007	0.17	0.0007	0.88
F_XCANCER12	–0.0012	–0.87	–0.0049	–0.86	–0.0007	–0.69
F_XHEART12	0.0002	0.21	–0.0025	–0.77	–0.0010	–1.42
F_XSTROKE12	–0.0010	–0.52	0.0021	0.29	–0.0002	–0.20
F_XLUNG12	0.0002	0.10	–0.0033	–0.71	–0.0015	–1.48
F_XDIABET12	–0.0027	–1.99	0.0041	0.86	–0.0018	–1.89
F_XHIGHBP12	0.0006	0.61	0.0047	1.55	–0.0010	–1.35
F_XARTHRT12	0.0008	0.73	–0.0018	–0.40	0.0004	0.44
F_XINCONT12	–0.0013	–1.01	0.0001	0.03	0.0006	0.66
F_XFALL12	0.0001	0.09	–0.0002	–0.05	0.0010	0.99
F_XHIPFRC12	0.0011	0.33	0.0018	0.28	–0.0005	–0.40
F_XPROXYW12	–0.0013	–0.40	–0.0013	–0.20	–0.0004	–0.25
F_XCOGIM12	–0.0003	–0.17	–0.0050	–1.11	–0.0023	–2.70

(*continued*)

Table 11A.5 (continued)

	Couples		Spouse Died		Single	
Variable	Estimate	*t*-statistic	Estimate	*t*-statistic	Estimate	*t*-statistic
F_XPSYCH12	–0.0005	–0.44	0.0007	0.14	–0.0010	–0.97
F_XDEPRES12	–0.0016	–0.91	–0.0012	–0.24	–0.0007	–0.61
F_XLOBMI12	0.0007	0.34	0.0013	0.58	0.0001	0.10
F_XHIBMI12	–0.0003	–1.79	–0.0011	–2.61	–0.0002	–1.47
F_XSMOKNOW12	–0.0014	–0.60	–0.0013	–0.20	–0.0026	–1.48
F_XNUMADL12	0.0013	1.58	0.0015	0.63	–0.0005	–1.64
F_XNUMIADL12	0.0002	0.20	–0.0016	–1.00	–0.0006	–1.37
F_XDHLTH12	0.0002	0.15	–0.0012	–0.21	–0.0004	–0.44
F_XJCANCER23	0.0019	0.71	–0.0002	–0.01	–0.0077	–3.19
F_XJHEART23	0.0033	1.69	–0.0045	–0.80	–0.0023	–2.00
F_XJSTROKE23	0.0041	1.77	–0.0185	–2.60	–0.0034	–1.98
F_XILUNG23	0.0044	1.15	–0.0039	–0.49	–0.0004	–0.26
F_XIDIABET23	–0.0011	–0.37	–0.0002	–0.03	0.0000	0.02
F_XIHIGHBP23	0.0023	1.09	0.0010	0.15	0.0001	0.06
F_XIARTHRT23	0.0012	0.81	0.0029	0.44	0.0010	1.09
F_XJINCONT23	0.0024	2.50	–0.0009	–0.24	0.0011	1.37
F_XJFALL23	–0.0011	–0.60	–0.0036	–0.75	0.0003	0.35
F_XJHIPFRC23	0.0010	0.27	0.0067	0.82	0.0006	0.036
F_XPROXYW23	–0.0017	–0.67	–0.0036	–0.52	–0.0011	–0.82
F_XICOGIM23	–0.0001	–0.05	–0.0023	–0.48	–0.0024	–2.42
F_XIPSYCH23	–0.0014	–0.70	–0.0158	–2.49	0.0012	0.93
F_XIDEPRES23	–0.0026	–1.52	0.0005	0.07	–0.0011	–0.78
F_XBMIBT23	0.0006	0.39	0.0017	0.34	–0.0001	–0.19
F_XBMIWS23	–0.0001	–0.10	0.0013	0.22	0.0001	0.11
F_XSMOKNOW23	–0.0038	–1.56	0.0013	0.13	0.0012	0.70
F_XNUMADL23	–0.0002	–0.33	0.0020	1.22	–0.0002	–0.93
F_XNUMIADL23	0.0006	0.73	–0.0014	–0.65	–0.0003	–0.84
F_XDHLTH23	–0.0032	–2.68	–0.0028	–0.60	–0.0003	–0.33
CF4	–0.2411	–0.18	–0.2411	–0.18	–0.0128	–0.27
CF3	–1.1433	–1.38	–1.1433	–1.38	0.5102	6.01
CM4	0.1903	1.40	0.1903	1.40	–0.1306	–1.97
CM3	–0.3582	–2.24	–0.3582	–2.24	–0.4368	–3.62
AM	–0.0025	–1.87	0.0012	2.80	–0.0076	–4.39
AF	–0.0008	–0.30	0.0004	0.45	–0.0019	–10.04
C	0.0000	21.10	0.0000	7.74	0.0000	6.11
VARM	0.0000	–0.88	0.0000	–0.89	0.0000	–0.81
VARF	0.0000	–2.53	0.0000	1.20	0.0000	0.48
SIGMA	0.0043	42.20	0.0000	7.74	0.0088	6.12
THETA	2.4733	19.10				
RHOM	–0.4882	–6.31			–0.8641	–17.28
RHOF	–0.1606	–3.11			–0.2103	–5.32

Notes: Prefixes M_ and F_ refer to males and females, respectively. For couples and spouse died regressions household-level variables are common for males and females. We report the estimates under the male section. CF3, CF4 = third/fourth order Edgeworth expansion terms for females; CM3, CM4 = third/fourth order Edgeworth expansion terms for males; AF, AM = estimates of λ in equations (15) and (19); C = estimate of k^2 in equation (16); VARM, VARF = estimates of λ^2 in equation (16) for males and females; SIGMA = estimate of σ as defined in page 440 of paper; THETA = estimate of θ in equation (19); RHOF, RHOM = estimate of ρ as defined in page 442 of paper.

Table 11A.6 Nonliquid Wealth Regressions—(*t*-statistics)

	Couples		Spouse Died		Single	
Variable	Estimate	*t*-statistic	Estimate	*t*-statistic	Estimate	*t*-statistic
XONE1	0.0582	14.64	0.0752	4.68	0.0482	4.74
XONE2	0.0459	11.33	0.0655	3.13	0.0438	3.94
XLWLTH12	0.0046	1.03	0.0041	0.40	0.0128	2.56
XNWLTH12	–0.1311	–15.98	–0.1646	–8.65	–0.1138	–8.91
XQ1I12	–0.0010	–1.19	–0.0011	–0.37	–0.0012	–0.50
XQ4I12	0.0021	2.17	0.0032	0.53	0.0015	0.66
XHOODPF12	–0.0017	–1.47	0.0050	1.27	0.0002	0.07
XCONDPF12	–0.0014	–1.19	–0.0008	–0.17	–0.0001	–0.03
XDNHOUS12	0.0068	4.45	0.0074	1.80	0.0013	0.50
M_XHS12	0.0000	0.04	0.0005	0.14	0.0010	0.47
M_XCOLL12	0.0000	–0.01	0.0021	0.44	–0.0008	–0.25
M_XAS701S	0.0000	0.22	0.0001	0.82	0.0001	2.71
M_XAS801S	–0.0001	–1.73	–0.0001	–0.43	–0.0001	–1.88
M_XAS702S	0.0000	0.48	0.0000	0.10	0.0001	2.01
M_XAS802S	0.0000	–1.16	0.0000	–0.19	–0.0001	–0.91
M_XNEVMARR12					0.0078	1.13
M_XWIDOW12					0.0078	1.26
M_XDIVSEP12					0.0101	1.45
M_XMAGEDI12	0.0000	0.33	–0.0001	–1.63	–0.0001	–1.64
M_XPAGEDI12	0.0000	0.23	0.0000	0.37	–0.0001	–1.20
M_XSMOKEV12	–0.0011	–1.33	–0.0044	–1.36	–0.0016	–0.67
M_XCANCER12	–0.0004	–0.35	0.0037	1.44	0.0032	1.20
M_XHEART12	0.0003	0.36	–0.0011	–0.42	0.0002	0.10
M_XSTROKE12	0.0015	0.83	–0.0013	–0.29	0.0012	0.44
M_XLUNG12	0.0022	1.30	–0.0018	–0.50	0.0001	0.05
M_XDIABET12	–0.0019	–1.89	0.0024	0.83	0.0044	1.87
M_XHIGHBP12	–0.0005	–0.71	0.0041	1.52	0.0007	0.37
M_XARTHRT12	0.0019	1.62	0.0052	1.52	–0.0012	–0.60
M_XINCONT12	0.0004	0.33	–0.0040	–1.48	–0.0034	–1.29
M_XFALL12	–0.0002	–0.14	–0.0025	–0.50	–0.0059	–1.90
M_XHIPFRC12	–0.0015	–0.48	0.0068	0.91	0.0100	2.32
M_XPROXYW12	0.0025	1.43	–0.0049	–1.35	0.0057	1.14
M_XCOGIM12	0.0024	2.07	–0.0018	–0.71	0.0017	0.81
M_XPSYCH12	–0.0007	–0.41	–0.0058	–1.62	0.0009	0.35
M_XDEPRES12	0.0018	0.48	–0.0027	–0.65	0.0018	0.57
M_XLOBMI12	0.0010	0.21	–0.0025	–1.25	0.0061	2.21
M_XHIBMI12	0.0003	2.04	0.0002	0.32	–0.0003	–0.70
M_XSMOKNOW12	–0.0006	–0.44	0.0006	0.10	–0.0050	–1.98
M_XNUMADL12	–0.0002	–0.24	0.0010	0.49	0.0004	0.51
M_XNUMIADL12	–0.0013	–1.31	0.0007	0.22	0.0008	0.76
M_XDHLTH12	–0.0017	–1.53	–0.0029	–0.81	0.0033	1.37
M_XJCANCER23	0.0012	0.42	0.0022	0.22	0.0170	3.07
M_XJHEART23	0.0020	0.93	0.0046	0.40	0.0097	2.51
M_XJSTROKE23	0.0024	0.69	–0.0077	–0.65	0.0066	1.62
M_XILUNG23	–0.0025	–1.54	–0.0010	–0.07	–0.0062	–2.39
M_XIDIABET23	0.0001	0.06	0.0212	2.97	–0.0003	–0.07

(*continued*)

Table 11A.6 (continued)

	Couples		Spouse Died		Single	
Variable	Estimate	*t*-statistic	Estimate	*t*-statistic	Estimate	*t*-statistic
M_XIHIGHBP23	–0.0025	–0.80	–0.0254	–1.90	0.0004	0.16
M_XIARTHRT23	–0.0010	–0.80	0.0058	0.96	–0.0023	–0.82
M_XJINCONT23	0.0000	0.00	–0.0069	–0.97	0.0014	0.78
M_XJFALL23	0.0000	0.01	–0.0034	–0.23	–0.0009	–0.49
M_XJHIPFRC23	–0.0038	–0.50	0.0221	0.91	–0.0040	–1.09
M_XPROXYW23	0.0002	0.11	–0.0017	–0.23	0.0012	0.34
M_XICOGIM23	0.0008	0.59	–0.0026	–0.20	–0.0013	–0.70
M_XIPSYCH23	–0.0004	–0.27	0.0027	0.28	0.0036	1.37
M_XIDEPRES23	–0.0007	–0.36	–0.0088	–1.15	–0.0001	–0.04
M_XBMIBT23	–0.0001	–0.15	–0.0057	–0.86	0.0005	0.31
M_XBMIWS23	–0.0004	–0.29	–0.0078	–0.97	0.0014	0.78
M_XSMOKNOW23	0.0015	0.97	–0.0128	–0.64	0.0038	1.72
M_XNUMADL23	0.0004	0.53	–0.0070	–2.30	–0.0005	–0.87
M_XNUMIADL23	–0.0002	–0.15	0.0037	1.36	–0.0008	–1.17
M_XDHLTH23	–0.0005	–0.55	–0.0044	–0.52	0.0015	0.92
F_XONE1					0.0688	12.20
F_XONE2					0.0619	10.64
F_XLWLTH12					0.0033	1.42
F_XNWLTH12					–0.1257	–20.66
F_XQ1I12					0.0004	0.59
F_XQ4I12					0.0030	3.13
F_XHOODPF12					–0.0010	–1.21
F_XCONDPF12					0.0002	0.26
F_XDNHOUS12					0.0045	4.66
F_XHS12	0.0004	0.41	0.0034	0.89	0.0001	0.21
F_XCOLL12	0.0005	0.44	0.0095	1.88	0.0016	1.36
F_XAS701S	0.0000	–0.51	0.0000	–0.32	0.0000	–1.70
F_XAS801S	0.0000	0.69	–0.0001	–0.81	0.0000	0.28
F_XAS702S	0.0000	–0.45	0.0000	0.45	0.0000	–1.09
F_XAS802S	0.0001	1.26	0.0000	0.03	0.0000	0.54
F_XNEVMARR12					0.0001	0.02
F_XWIDOW12					0.0000	0.01
F_XDIVSEP12					–0.0001	–0.01
F_XMAGEDI12	0.0000	0.64	0.0000	0.73	0.0000	0.74
F_XPAGEDI12	0.0000	–0.31	0.0000	0.32	0.0000	0.47
F_XSMOKEV12	–0.0003	–0.40	–0.0025	–0.63	0.0001	0.20
F_XCANCER12	0.0007	0.57	–0.0052	–1.00	0.0001	0.07
F_XHEART12	–0.0009	–0.89	–0.0046	–1.51	–0.0006	–0.96
F_XSTROKE12	0.0029	1.83	0.0010	0.13	–0.0005	–0.44
F_XLUNG12	0.0000	–0.01	–0.0008	–0.20	–0.0012	–1.45
F_XDIABET12	–0.0002	–0.13	0.0064	1.36	–0.0012	–1.38
F_XHIGHBP12	0.0001	0.15	0.0046	1.82	–0.0014	–2.16
F_XARTHRT12	–0.0002	–0.18	0.0029	0.68	0.0004	0.59
F_XINCONT12	0.0000	–0.02	–0.0014	–0.42	0.0000	0.02
F_XFALL12	0.0005	0.33	0.0012	0.31	0.0010	1.12
F_XHIPFRC12	0.0039	1.29	–0.0018	–0.33	–0.0008	–0.69
F_XPROXYW12	–0.0007	–0.24	–0.0011	–0.19	–0.0005	–0.32

Table 11A.6 (continued)

	Couples		Spouse Died		Single	
Variable	Estimate	*t*-statistic	Estimate	*t*-statistic	Estimate	*t*-statistic
F_XCOGIM12	0.0005	0.33	–0.0043	–1.04	–0.0014	–1.70
F_XPSYCH12	0.0000	–0.01	0.0033	0.66	–0.0013	–1.48
F_XDEPRES12	0.0006	0.40	–0.0025	–0.51	–0.0008	–0.83
F_XLOBMI12	0.0017	0.90	0.0004	0.20	–0.0004	–1.16
F_XHIBMI12	0.0000	0.17	–0.0012	–3.19	–0.0001	–0.87
F_XSMOKNOW12	–0.0004	–0.18	0.0038	0.61	–0.0033	–2.14
F_XNUMADL12	0.0001	0.18	0.0011	0.49	–0.0004	–1.28
F_XNUMIADL12	0.0008	0.89	0.0000	0.00	0.0003	0.89
F_XDHLTH12	0.0006	0.47	–0.0011	–0.21	–0.0008	–1.00
F_XJCANCER23	–0.0010	–0.46	0.0028	0.33	–0.0063	–2.60
F_XJHEART23	0.0007	0.38	–0.0039	–0.79	–0.0019	–1.76
F_XJSTROKE23	0.0015	0.83	–0.0032	–0.44	–0.0022	–1.33
F_XILUNG23	0.0060	1.74	–0.0070	–1.11	–0.0019	–1.41
F_XIDIABET23	–0.0028	–1.41	–0.0050	–0.58	–0.0005	–0.31
F_XIHIGHBP23	0.0002	0.09	–0.0009	–0.15	0.0002	0.14
F_XIARTHRT23	0.0004	0.27	0.0001	0.01	0.0010	1.14
F_XJINCONT23	0.0007	0.86	–0.0006	–0.17	0.0005	0.78
F_XJFALL23	–0.0006	–0.32	–0.0042	–1.04	–0.0005	–0.73
F_XJHIPFRC23	–0.0045	–1.86	–0.0025	–0.39	–0.0003	–0.23
F_XPROXYW23	0.0006	0.23	–0.0043	–0.61	–0.0008	–0.61
F_XICOGIM23	–0.0007	–0.41	–0.0016	–0.34	–0.0016	–1.98
F_XIPSYCH23	–0.0005	–0.28	–0.0047	–0.65	–0.0001	–0.08
F_XIDEPRES23	0.0007	0.42	–0.0041	–0.65	–0.0010	–0.88
F_XBMIBT23	0.0033	0.24	0.0044	1.00	0.0024	0.61
F_XBMIWS23	0.0001	0.06	0.0026	0.44	–0.0004	–0.62
F_XSMOKNOW23	–0.0013	–0.56	–0.0075	–0.88	0.0033	2.22
F_XNUMADL23	–0.0006	–0.91	0.0019	1.06	–0.0003	–1.45
F_XNUMIADL23	0.0002	0.22	–0.0021	–1.02	–0.0007	–2.34
F_XDHLTH23	–0.0027	–2.52	0.0032	0.76	0.0005	0.65
CF4	0.1230	0.09	0.1230	0.09	0.0191	0.36
CF3	–0.2322	–0.28	–0.2322	–0.28	0.4650	4.71
CM4	0.1776	1.30	0.1776	1.30	–0.1248	–2.74
CM3	–0.2742	–1.71	–0.2742	–1.71	–0.3308	–3.87
AM	–0.0015	–1.16	0.0006	2.09	–0.0118	–3.52
AF	–0.0004	–0.15	0.0001	0.30	–0.0015	–7.26
C	0.0000	23.59	0.0000	0.00	0.0000	5.84
VARM	0.0000	1.34	0.0000	0.00	0.0000	–0.56
VARF	0.0000	–0.12	0.0000	0.00	0.0000	–0.23
SIGMA	0.0041	47.19	0.0000	0.00	0.0125	4.00
THETA	2.3623	11.32				
RHOM	–0.3532	–3.29			–0.9434	–31.26
RHOF	–0.0939	–1.65			–0.1164	–3.53

Notes: See table 11A.5.

Table 11A.7 Liquid Wealth Regressions—(*t*-statistics)

	Couples		Spouse Died		Single	
Variable	Estimate	*t*-statistic	Estimate	*t*-statistic	Estimate	*t*-statistic
XONE1	0.0819	12.94	0.1150	5.73	0.0802	5.66
XONE2	0.0705	10.88	0.1021	4.35	0.0652	4.39
XLWLTH12	–0.1474	–22.15	–0.1581	–9.33	–0.1583	–13.68
XNWLTH12	0.0222	2.68	0.0058	0.42	0.0272	2.55
XQ1I12	–0.0077	–6.24	–0.0100	–2.50	–0.0012	–0.43
XQ4I12	0.0103	5.38	0.0032	0.40	0.0108	3.44
XHOODPF12	–0.0014	–0.86	0.0069	1.31	0.0032	0.91
XCONDPF12	–0.0038	–2.58	–0.0066	–1.20	0.0010	0.30
XDNHOUS12	0.0008	0.47	0.0104	2.36	–0.0007	–0.25
M_XHS12	0.0003	0.28	0.0044	1.00	0.0037	1.46
M_XCOLL12	0.0016	0.83	0.0061	0.88	0.0080	1.53
M_XAS701S	0.0000	0.85	–0.0001	–1.13	0.0000	0.67
M_XAS801S	0.0000	0.90	0.0003	1.62	0.0000	0.42
M_XAS702S	0.0000	0.12	–0.0001	–0.90	0.0001	1.12
M_XAS802S	0.0001	1.90	0.0003	1.69	0.0000	–0.28
M_XNEVMARR12					0.0078	0.90
M_XWIDOW12					0.0083	1.04
M_XDIVSEP12					0.0069	0.81
M_XMAGEDI12	0.0000	0.86	0.0000	–0.36	0.0000	–0.03
M_XPAGEDI12	0.0000	–0.34	0.0000	–0.16	–0.0001	–1.02
M_XSMOKEV12	0.0007	0.49	–0.0026	–0.68	0.0016	0.52
M_XCANCER12	0.0027	1.72	0.0011	0.28	–0.0002	–0.08
M_XHEART12	–0.0007	–0.63	–0.0042	–1.11	–0.0007	–0.28
M_XSTROKE12	0.0013	0.55	–0.0051	–0.92	–0.0036	–1.06
M_XLUNG12	0.0045	2.16	–0.0056	–1.22	–0.0047	–1.76
M_XDIABET12	–0.0023	–1.47	–0.0059	–1.39	–0.0015	–0.46
M_XHIGHBP12	0.0011	0.98	0.0021	0.66	–0.0017	–0.71
M_XARTHRT12	–0.0039	–2.48	0.0024	0.56	0.0002	0.09
M_XINCONT12	–0.0003	–0.15	–0.0015	–0.35	0.0005	0.19
M_XFALL12	–0.0025	–1.12	0.0076	1.22	–0.0007	–0.22
M_XHIPFRC12	–0.0021	–0.57	–0.0069	–0.81	0.0005	0.11
M_XPROXYW12	–0.0022	–1.00	0.0030	0.63	0.0006	0.13
M_XCOGIM12	–0.0002	–0.11	–0.0089	–2.49	–0.0070	–2.77
M_XPSYCH12	0.0004	0.17	0.0087	1.47	0.0044	1.07
M_XDEPRES12	–0.0064	–1.55	0.0109	1.99	–0.0034	–1.05
M_XLOBMI12	–0.0003	–0.05	0.0008	0.34	0.0024	0.95
M_XHIBMI12	–0.0003	–1.56	0.0012	1.76	0.0001	0.27
M_XSMOKNOW12	–0.0021	–0.85	–0.0057	–0.96	0.0002	0.05
M_XNUMADL12	0.0014	1.57	–0.0020	–0.89	–0.0011	–1.02
M_XNUMIADL12	–0.0022	–1.90	0.0005	0.14	0.0001	0.09
M_XDHLTH12	0.0031	2.01	0.0052	1.15	0.0030	1.10
M_XJCANCER23	0.0011	0.32	0.0015	0.11	–0.0010	–0.24
M_XJHEART23	0.0032	1.30	–0.0052	–0.45	0.0003	0.10
M_XJSTROKE23	0.0012	0.30	0.0079	0.71	0.0045	1.09
M_XILUNG23	0.0004	0.15	–0.0257	–1.76	–0.0024	–0.61
M_XIDIABET23	–0.0030	–0.90	0.0105	0.84	–0.0007	–0.11
M_XIHIGHBP23	0.0026	0.68	0.0003	0.02	–0.0064	–1.59

Table 11A.7 (continued)

	Couples		Spouse Died		Single	
Variable	Estimate	*t*-statistic	Estimate	*t*-statistic	Estimate	*t*-statistic
M_XIARTHRT23	–0.0011	–0.59	–0.0086	–1.40	–0.0046	–1.53
M_XJINCONT23	–0.0005	–0.25	0.0043	0.50	–0.0023	–0.92
M_XJFALL23	0.0021	1.05	0.0224	1.55	–0.0033	–1.05
M_XJHIPFRC23	–0.0075	–0.94	–0.0264	–1.00	0.0093	0.97
M_XPROXYW23	–0.0018	–0.83	0.0069	0.86	–0.0038	–1.12
M_XICOGIM23	–0.0008	–0.47	–0.0046	–0.41	0.0008	0.28
M_XIPSYCH23	0.0010	0.51	–0.0156	–0.74	0.0071	1.86
M_XIDEPRES23	0.0009	0.32	0.0041	0.55	–0.0053	–1.24
M_XBMIBT23	–0.0027	–2.02	–0.0038	–0.50	0.0004	0.15
M_XBMIWS23	–0.0038	–2.47	–0.0055	–0.53	–0.0014	–0.55
M_XSMOKNOW23	0.0020	0.68	0.0047	0.25	–0.0082	–2.11
M_XNUMADL23	–0.0004	–0.46	–0.0011	–0.37	–0.0006	–0.86
M_XNUMIADL23	0.0003	0.28	–0.0077	–2.41	0.0004	0.42
M_XDHLTH23	0.0000	0.01	–0.0046	–0.51	0.0000	–0.02
F_XONE1					0.0680	10.83
F_XONE2					0.0547	8.51
F_XLWLTH12					–0.1280	–20.62
F_XNWLTH12					0.0203	4.26
F_XQ1I12					–0.0020	–2.31
F_XQ4I12					0.0118	7.73
F_XHOODPF12					–0.0008	–0.69
F_XCONDPF12					–0.0031	–2.93
F_XDNHOUS12					0.0014	1.31
F_XHS12	0.0022	1.55	0.0103	2.16	0.0025	2.84
F_XCOLL12	0.0027	1.35	–0.0007	–0.09	0.0036	1.96
F_XAS701S	0.0001	2.35	0.0000	0.33	0.0000	0.55
F_XAS801S	–0.0002	–2.04	–0.0002	–0.82	0.0000	0.15
F_XAS702S	0.0000	–1.16	–0.0001	–0.79	0.0000	1.63
F_XAS802S	0.0000	0.30	0.0000	0.24	0.0000	–0.81
F_XNEVMARR12					0.0114	2.38
F_XWIDOW12					0.0087	1.97
F_XDIVSEP12					0.0061	1.33
F_XMAGEDI12	0.0000	0.71	0.0000	–0.39	0.0000	0.66
F_XPAGEDI12	0.0000	–1.31	–0.0001	–1.07	0.0000	0.36
F_XSMOKEV12	0.0034	2.76	0.0063	1.38	0.0011	1.10
F_XCANCER12	–0.0019	–1.10	–0.0024	–0.38	–0.0010	–0.82
F_XHEART12	–0.0006	–0.41	0.0038	1.01	0.0001	0.15
F_XSTROKE12	–0.0013	–0.52	0.0032	0.43	–0.0009	–0.67
F_XLUNG12	–0.0001	–0.03	–0.0068	–1.28	–0.0011	–0.85
F_XDIABET12	–0.0028	–1.66	–0.0027	–0.51	–0.0015	–1.27
F_XHIGHBP12	–0.0010	–0.82	0.0094	2.55	–0.0006	–0.63
F_XARTHRT12	0.0019	1.43	–0.0053	–1.13	–0.0005	–0.55
F_XINCONT12	0.0004	0.22	0.0012	0.26	–0.0003	–0.29
F_XFALL12	0.0026	1.28	0.0000	0.01	0.0003	0.27
F_XHIPFRC12	0.0034	0.89	–0.0001	–0.01	0.0030	1.80

(*continued*)

Table 11A.7 (continued)

	Couples		Spouse Died		Single	
Variable	Estimate	*t*-statistic	Estimate	*t*-statistic	Estimate	*t*-statistic
F_XPROXYW12	–0.0023	–0.62	–0.0017	–0.25	–0.0032	–1.54
F_XCOGIM12	–0.0011	–0.56	–0.0064	–1.24	–0.0034	–3.27
F_XPSYCH12	–0.0015	–0.99	–0.0009	–0.15	0.0017	1.31
F_XDEPRES12	–0.0016	–0.74	0.0031	0.56	–0.0020	–1.59
F_XLOBMI12	–0.0006	–0.31	0.0021	0.96	0.0002	0.32
F_XHIBMI12	–0.0005	–2.31	–0.0005	–0.88	–0.0003	–2.14
F_XSMOKNOW12	–0.0016	–0.43	–0.0136	–1.83	–0.0035	–1.71
F_XNUMADL12	0.0023	2.35	0.0005	0.20	–0.0006	–1.44
F_XNUMIADL12	0.0006	0.48	–0.0046	–2.31	–0.0011	–2.45
F_XDHLTH12	–0.0019	–1.13	0.0003	0.05	–0.0006	–0.60
F_XJCANCER23	0.0073	2.04	–0.0026	–0.21	–0.0066	–2.41
F_XJHEART23	0.0029	1.36	–0.0038	–0.57	–0.0024	–1.77
F_XJSTROKE23	0.0012	0.45	–0.0235	–2.53	–0.0041	–2.04
F_XILUNG23	–0.0052	–1.48	–0.0040	–0.32	0.0012	0.62
F_XIDIABET23	–0.0050	–1.26	0.0031	0.28	0.0012	0.54
F_XIHIGHBP23	–0.0024	–0.93	0.0024	0.32	–0.0007	–0.44
F_XIARTHRT23	0.0015	0.79	0.0077	1.14	0.0006	0.46
F_XJINCONT23	0.0034	2.59	0.0041	0.86	0.0017	1.71
F_XJFALL23	–0.0010	–0.48	–0.0074	–1.16	0.0017	1.56
F_XJHIPFRC23	0.0034	0.69	0.0198	1.68	–0.0005	–0.26
F_XPROXYW23	–0.0015	–0.50	–0.0001	–0.01	–0.0001	–0.04
F_XICOGIM23	–0.0017	–0.67	0.0007	0.10	–0.0036	–2.86
F_XIPSYCH23	–0.0015	–0.60	–0.0192	–2.81	0.0014	0.81
F_XIDEPRES23	–0.0027	–1.22	0.0030	0.41	–0.0023	–1.46
F_XBMIBT23	0.0007	0.41	–0.0001	–0.02	–0.0001	–0.11
F_XBMIWS23	–0.0010	–0.67	0.0018	0.25	0.0005	0.56
F_XSMOKNOW23	–0.0060	–1.59	0.0168	1.60	0.0013	0.64
F_XNUMADL23	–0.0017	–2.52	0.0037	1.84	–0.0002	–0.46
F_XNUMIADL23	0.0022	2.35	–0.0035	–1.29	0.0006	1.42
F_XDHLTH23	–0.0002	–0.16	–0.0094	–1.81	0.0007	0.75
CF4	0.0801	0.06	0.0801	0.06	–0.0320	–0.50
CF3	–0.1813	–0.22	–0.1813	–0.22	0.5701	5.63
CM4	0.1603	1.18	0.1603	1.18	0.0101	0.07
CM3	–0.3138	–1.97	–0.3138	–1.97	–0.5973	–2.35
AM	–0.0027	–2.09	0.0014	3.13	–0.0038	–2.89
AF	–0.0018	–0.66	0.0009	0.93	–0.0016	–10.38
C	0.0000	24.84	0.0000	4.23	0.0000	6.13
VARM	0.0000	–0.51	0.0000	0.72	0.0000	–1.45
VARF	0.0000	–0.38	0.0000	–0.67	0.0000	1.75
SIGMA	0.0055	49.68	0.0000	4.23	0.0061	7.47
THETA	2.5207	18.63				
RHOM	–0.4243	–7.48			–0.6292	–5.06
RHOF	–0.2826	–5.64			–0.2579	–6.27

Notes: See table 11A.5.

Table 11A.8 Wealth Regressions—Invariance Tests

	Wealth (DF)	Total		Nonliquid		Liquid	
		CHISQ	*p*-value	CHISQ	*p*-value	CHISQ	*p*-value
Couples							
All	97	468.6	0.000	355.1	0.000	377.2	0.000
All less SES	90	219.4	0.0000	168.1	0.0000	243.9	0.0000
All male	52	279.7	0.0000	232.5	0.0000	215.9	0.0000
All male less SES	45	109.1	0.0000	80.0	0.0010	117.4	0.0000
All female	52	277.2	0.0000	218.9	0.0000	200.1	0.0000
All female less SES	45	90.0	0.0001	76.9	0.0021	104.9	0.0000
All less prevalence	57	301.2	0.0000	224.6	0.0000	223.8	0.0000
All less incidence	57	358.4	0.0000	259.8	0.0000	266.1	0.0000
Only SES	7	123.4	0.0000	119.6	0.0000	69.8	0.0000
Only male demographic	5	6.9	0.2292	4.1	0.5382	8.9	0.1129
Only female demographic	5	5.2	0.3971	10.6	0.0590	4.9	0.4261
Only male prevalence	20	58.8	0.0000	34.2	0.0246	69.9	0.0000
Only female prevalence	20	57.2	0.0000	33.3	0.0310	45.8	0.0009
Only male incidence	20	38.9	0.0070	33.2	0.0324	44.0	0.0015
Only female incidence	20	31.1	0.0540	22.7	0.3060	53.5	0.0001
Spouse died							
All	97	672.2	0.0000	682.7	0.0000	700.2	0.0000
All less SES	90	524.0	0.0000	645.3	0.0000	576.9	0.0000
All male	52	268.2	0.0000	370.0	0.0000	224.6	0.0000
All male less SES	45	244.1	0.0000	350.1	0.0000	161.4	0.0000
All female	52	250.0	0.0000	181.5	0.0000	358.8	0.0000
All female less SES	45	164.7	0.0000	142.7	0.0000	256.6	0.0000
All less prevalence	57	223.4	0.0000	269.2	0.0000	361.1	0.0000
All less incidence	57	240.4	0.0000	234.9	0.0000	277.3	0.0000
Only SES	7	60.0	0.0000	34.2	0.0000	37.0	0.0000
Only male demographic	5	7.8	0.1692	8.2	0.1473	12.7	0.0259
Only female demographic	5	7.3	0.2020	5.6	0.3479	16.2	0.0063
Only male prevalence	20	69.5	0.0000	65.8	0.0000	66.3	0.0000
Only female prevalence	20	56.0	0.0000	49.9	0.0002	38.2	0.0085
Only male incidence	20	71.7	0.0000	105.5	0.0000	41.5	0.0021
Only female incidence	20	74.6	0.0000	65.0	0.0000	115.2	0.0000
Singles							
All	110	384.8	0.0000	353.6	0.0000	301.4	0.0000
All less SES	96	118.5	0.0594	139.9	0.0023	129.8	0.0124
All male	55	129.2	0.0000	139.4	0.0000	91.2	0.0015
All male less SES	48	43.3	0.6639	60.3	0.1094	59.4	0.1261
All female	55	255.6	0.0000	214.1	0.0000	210.1	0.0000
All female less SES	48	75.2	0.0073	79.6	0.0028	70.4	0.0192
All less prevalence	70	307.3	0.0000	296.7	0.0000	201.4	0.0000
All less incidence	70	287.4	0.0000	275.7	0.0000	222.0	0.0000
Only Male SES	7	30.2	0.0001	47.0	0.0000	16.4	0.0215
Only female SES	7	153.3	0.0000	129.3	0.0000	89.8	0.0000
Only male demographic	8	4.1	0.8446	6.5	0.5875	5.8	0.6742
Only female demographic	8	13.9	0.0842	14.9	0.0603	16.3	0.0379
Only male prevalence	20	19.0	0.5251	30.6	0.0613	30.8	0.0580
Only female prevalence	20	26.4	0.1536	30.6	0.0601	26.3	0.1559
Only male incidence	20	19.4	0.4955	23.3	0.2747	19.7	0.4759
Only female incidence	20	41.0	0.0037	40.7	0.0040	30.2	0.0663

Table 11A.9 Wealth Regressions—Causality Tests

	Wealth (DF)	Total		Nonliquid		Liquid	
		CHISQ	*p*-value	CHISQ	*p*-value	CHISQ	*p*-value
Incidence							
Couple							
Male	20	20.01	0.4575	16.03	0.7146	18.04	0.5847
Female	20	36.65	0.0129	33.87	0.0270	37.47	0.0103
Male & female	40	59.47	0.0243	51.44	0.1061	57.55	0.0356
Spouse died							
Male	20	22.65	0.3061	51.36	0.0001	50.99	0.0002
Female	20	23.02	0.2878	16.99	0.6533	30.71	0.0591
Male & female	40	62.86	0.0120	75.29	0.0006	88.09	0.0000
Single							
Male	20	18.78	0.5362	30.01	0.0696	24.38	0.2263
Female	20	32.65	0.0369	53.88	0.0001	33.03	0.0335
Male & female	40	51.43	0.1064	83.89	0.0001	57.41	0.0366
Prevalence							
Couple							
Male	20	27.85	0.1130	47.88	0.0004	45.58	0.0009
Female	20	24.73	0.2120	14.50	0.8041	38.30	0.0081
Male & female	40	58.87	0.0275	72.18	0.0014	111.42	0.0000
Spouse died							
Male	20	20.72	0.4137	29.71	0.0746	32.32	0.0401
Female	20	13.58	0.8511	16.95	0.6559	34.18	0.0249
Male & female	40	49.79	0.1607	74.75	0.0007	76.24	0.0005
Single							
Male	20	17.67	0.6093	18.07	0.5830	19.62	0.4818
Female	20	35.84	0.0161	27.75	0.1155	53.83	0.0001
Male & female	40	53.51	0.0749	45.82	0.2436	73.45	0.0010
Incidence and prevalence							
Couple							
Male	40	50.91	0.1157	70.14	0.0022	78.51	0.0003
Female	40	72.00	0.0014	54.30	0.0651	88.95	0.0000
Male & female	80	126.80	0.0007	124.70	0.0010	191.82	0.0000
Spouse died							
Male	40	61.81	0.0150	104.07	0.0000	100.39	0.0000
Female	40	51.95	0.0977	37.35	0.5904	70.08	0.0023
Male & female	80	122.23	0.0017	174.89	0.0000	190.94	0.0000
Single							
Male	40	35.35	0.6796	40.58	0.4448	49.87	0.1362
Female	40	80.77	0.0001	84.39	0.0001	79.31	0.0002
Male & female	80	116.11	0.0052	124.96	0.0010	129.18	0.0004

Table 11A.10 Income Regressions—(*t*-statistics)

	Couples		Spouse Died		Single	
Variable	Estimate	*t*-statistic	Estimate	*t*-statistic	Estimate	*t*-statistic
ONE	–0.1417	–5.75	–0.0864	–0.79	–0.1386	–2.23
WLTH12	0.1077	6.82	0.1249	1.41	0.1061	2.57
Q1I12	0.0799	12.32	0.1009	3.42	0.0388	2.15
Q4I12	–0.0882	–14.25	–0.1171	–3.05	–0.0908	–6.83
HOODPF12	0.0019	0.23	–0.0054	–0.13	0.0261	1.55
CONDPF12	–0.0028	–0.34	–0.0904	–2.31	–0.0304	–1.79
DNHOUS12	0.0019	0.29	0.0191	0.60	0.0051	0.39
M_HS12	–0.0013	–0.22	0.0210	0.70	0.0053	0.41
M_COLL12	0.0293	4.59	0.0177	0.46	0.0040	0.23
M_AS70S	0.0000	0.49	0.0000	0.09	–0.0001	–0.56
M_AS80S	0.0001	0.52	–0.0004	–0.56	–0.0002	–0.65
M_NEVMARR12					0.0925	2.18
M_WIDOW12					0.1141	2.85
M_DIVSEP12					0.1032	2.47
M_MAGEDI12	0.0002	1.27	–0.0010	–1.56	0.0000	–0.13
M_PAGEDI12	0.0001	0.49	–0.0002	–0.26	0.0003	0.94
M_SMOKEV12	0.0087	1.60	0.0223	0.75	–0.0190	–1.47
M_CANCER12	0.0073	1.19	0.0109	0.36	0.0331	2.18
M_HEART12	–0.0008	–0.17	–0.0304	–1.16	0.0014	0.12
M_STROKE12	–0.0107	–1.20	–0.0404	–1.08	–0.0122	–0.62
M_LUNG12	–0.0005	–0.07	–0.0307	–0.99	–0.0072	–0.49
M_DIABET12	0.0032	0.44	–0.0709	–2.28	–0.0145	–0.88
M_HIGHBP12	0.0134	2.82	0.0456	1.89	0.0010	0.08
M_ARTHRT12	0.0045	0.73	–0.0212	–0.65	0.0211	1.52
M_INCONT12	–0.0017	–0.20	–0.0273	–0.82	–0.0006	–0.03
M_FALL12	–0.0061	–0.52	0.0800	1.75	–0.0021	–0.09
M_HIPFRC12	–0.0015	–0.11	–0.0192	–0.30	–0.0121	–0.41
M_PROXYW12	–0.0193	–2.19	–0.0173	–0.57	–0.0052	–0.14
M_COGIM12	0.0047	0.66	0.0041	0.15	0.0083	0.54
M_PSYCH12	0.0056	0.66	–0.0334	–0.77	–0.0060	–0.33
M_DEPRES12	–0.0245	–1.77	0.0050	0.12	–0.0187	–1.02
M_LOBMI12	–0.0063	–0.70	–0.0262	–1.50	–0.0244	–1.55
M_HIBMI12	0.0006	0.67	0.0031	0.67	–0.0016	–0.69
M_SMOKNOW12	0.0139	1.20	0.0429	1.08	0.0316	1.37
M_NUMADL12	–0.0054	–1.67	–0.0037	–0.34	0.0013	0.19
M_NUMIADL12	0.0000	–0.01	0.0103	0.86	0.0059	0.66
M_DHLTH12	–0.0090	–1.47	0.0394	1.30	–0.0117	–0.81
M_JCANCER23	–0.0018	–0.18	–0.0330	–0.21	–0.0077	–0.35
M_JHEART23	–0.0042	–0.56	0.1318	1.59	0.0118	0.64
M_JSTROKE23	–0.0072	–0.59	0.0482	0.25	–0.0149	–0.59
M_JLUNG23	–0.0041	–0.30	0.0001	0.00	0.0210	0.62
M_IDIABET23	–0.0216	–1.50	–0.3218	–1.49	–0.0335	–0.93
M_IHIGHBP23	–0.0123	–1.17	0.1396	0.83	0.0081	0.36
M_IARTHRT23	–0.0161	–2.10	–0.2789	–3.54	0.0138	0.69
M_JINCONT23	–0.0043	–0.58	0.0622	0.84	0.0032	0.20

(*continued*)

Table 11A.10 (continued)

	Couples		Spouse Died		Single	
Variable	Estimate	*t*-statistic	Estimate	*t*-statistic	Estimate	*t*-statistic
M_JFALL23	–0.0018	–0.21	0.1214	0.98	–0.0080	–0.40
M_JHIPFRC23	–0.0315	–1.44	–0.0491	–0.34	0.0231	0.51
M_PROXYW23	–0.0028	–0.30	0.1074	1.70	0.0230	0.88
M_ICOGIM23	0.0042	0.58	0.0835	1.09	0.0199	1.17
M_IPSYCH23	0.0048	0.56	0.0548	0.47	–0.0164	–0.40
M_IDEPRES23	–0.0119	–0.91	–0.0135	–0.23	0.0261	0.96
M_BMIBT23	0.0068	1.09	–0.0380	–0.86	0.0219	1.53
M_BMIWS23	–0.0026	–0.39	0.0709	0.87	–0.0108	–0.77
M_SMOKNOW23	–0.0117	–0.85	–0.1375	–1.68	–0.0327	–1.32
M_NUMADL23	–0.0036	–1.18	–0.0188	–0.61	–0.0154	–2.58
M_NUMIADL23	0.0081	2.05	–0.0286	–0.97	0.0131	1.76
M_DHLTH23	0.0015	0.25	0.0011	0.02	–0.0138	–1.02
F_ONE					–0.1196	–3.13
F_WLTH12					0.0869	4.05
F_Q1I12					0.0395	6.34
F_Q4I12					–0.0967	–14.66
F_HOODPF12					0.0012	0.16
F_CONDPF12					–0.0037	–0.49
F_DNHOUS12					0.0056	0.98
F_HS12	0.0044	0.69	0.0250	0.88	0.0184	3.33
F_COLL12	0.0126	1.89	0.0601	1.43	0.0275	3.54
F_AS70S	0.0000	0.08	–0.0002	–0.47	0.0000	0.14
F_AS80S	0.0000	–0.19	0.0007	0.78	0.0001	0.68
F_NEVMARR12					0.0724	2.16
F_WIDOW12					0.0589	1.84
F_DIVSEP12					0.0552	1.67
F_MAGEDI12	0.0002	1.17	0.0004	0.72	0.0002	1.14
F_PAGEDI12	0.0002	1.26	0.0002	0.26	–0.0001	–0.47
F_SMOKEV12	0.0059	1.17	–0.0297	–1.10	0.0079	1.44
F_CANCER12	0.0104	1.47	–0.0399	–1.08	0.0105	1.57
F_HEART12	0.0070	1.24	–0.0061	–0.22	–0.0016	–0.29
F_STROKE12	0.0271	2.57	–0.0160	–0.37	–0.0008	–0.09
F_LUNG12	–0.0058	–0.70	0.0561	1.41	0.0031	0.39
F_DIABET12	0.0144	1.83	0.0003	0.01	0.0021	0.26
F_HIGHBP12	0.0093	1.98	–0.0234	–0.96	0.0003	0.07
F_ARTHRT12	0.0063	1.11	–0.0663	–2.31	0.0006	0.10
F_INCONT12	0.0042	0.70	0.0280	0.84	–0.0005	–0.08
F_FALL12	0.0117	1.40	–0.0098	–0.22	0.0008	0.10
F_HIPFRC12	0.0435	3.59	–0.0526	–0.97	–0.0031	–0.32
F_PROXYW12	0.0007	0.06	–0.0636	–1.14	–0.0005	–0.04
F_COGIM12	0.0183	2.32	–0.0273	–0.93	0.0010	0.15
F_PSYCH12	0.0027	0.41	–0.0275	–0.68	0.0082	1.07
F_DEPRES12	–0.0043	–0.46	–0.0404	–0.99	–0.0010	–0.13
F_LOBMI12	0.0039	0.99	–0.0079	–0.58	0.0017	0.46
F_HIBMI12	–0.0006	–0.89	–0.0017	–0.28	0.0006	0.66
F_SMOKNOW12	0.0079	0.44	–0.0286	–0.49	–0.0013	–0.11
F_NUMADL12	0.0036	1.10	0.0289	1.73	–0.0026	–0.94

Table 11A.10 (continued)

	Couples		Spouse Died		Single	
Variable	Estimate	*t*-statistic	Estimate	*t*-statistic	Estimate	*t*-statistic
F_NUMIADL12	–0.0074	–1.73	–0.0039	–0.25	0.0001	0.04
F_DHLTH12	–0.0050	–0.77	0.0368	1.21	0.0027	0.42
F_JCANCER23	0.0064	0.47	–0.0632	–0.89	–0.0025	–0.18
F_JHEART23	–0.0165	–1.92	–0.0496	–1.10	–0.0163	–2.08
F_JSTROKE23	–0.0189	–1.46	–0.0688	–0.95	0.0083	0.64
F_JLUNG23	–0.0075	–0.43	0.0216	0.18	–0.0003	–0.02
F_IDIABET23	–0.0025	–0.14	0.0516	0.58	–0.0036	–0.23
F_IHIGHBP23	0.0221	1.98	–0.0730	–1.32	0.0091	0.86
F_IARTHRT23	0.0095	1.24	0.0417	0.80	–0.0062	–0.85
F_JINCONT23	–0.0037	–0.64	–0.0334	–0.91	0.0056	0.89
F_JFALL23	0.0009	0.13	0.0384	0.87	–0.0040	–0.58
F_JHIPFRC23	–0.0325	–1.46	–0.0973	–0.95	0.0070	0.49
F_PROXYW23	0.0005	0.05	0.1906	3.18	–0.0135	–1.29
F_ICOGIM23	–0.0009	–0.10	–0.0727	–1.75	–0.0039	–0.53
F_IPSYCH23	–0.0023	–0.25	–0.0264	–0.66	–0.0016	–0.13
F_IDEPRES23	–0.0082	–0.71	0.0403	0.95	0.0091	0.85
F_BMIBT23	–0.0172	–2.74	0.0032	0.09	0.0008	0.11
F_BMIWS23	–0.0084	–1.33	0.0236	0.63	–0.0035	–0.53
F_SMOKNOW23	–0.0056	–0.30	0.0571	0.73	–0.0128	–0.97
F_NUMADL23	–0.0025	–0.87	–0.0088	–0.64	0.0006	0.24
F_NUMIADL23	–0.0010	–0.26	–0.0165	–0.93	0.0049	1.61
F_DHLTH23	–0.0016	–0.25	–0.0665	–1.86	–0.0124	–1.95

Table 11A.11 **Mobility, Ownership, Neighborhood, and Dwelling Condition Regressions—(*t*-statistics)**

	Females		Males	
Variable	Coefficient	*t*-statistic	Coefficient	*t*-statistic
	Changed Residence Regressions			
one1	–8.0824	–5.1883	–8.4228	–5.5322
one2	–3.5406	–3.2190	–3.3344	–3.0886
logm1	2.0472	4.1790	2.1521	4.4979
logm2	0.5848	1.7574	0.5224	1.6023
q1wb12	0.2458	4.1263	0.2213	3.7980
q4wb12	–0.1640	–2.5800	–0.1952	–3.1669
q1ib12	0.0881	1.5198	0.0965	1.7090
q4ib12	0.1086	1.8830	0.1300	2.3259
hoodpf12	0.0351	0.4949	0.0246	0.3560
condpf12	–0.0950	–1.3343	–0.0855	–1.2402
cwlth23	–0.5547	–2.9274	–0.5864	–3.1662
male12	–0.0706	–1.0036	–0.0728	–1.0355
single12	0.3399	4.9781	0.3471	4.1435
spdied23	0.4072	3.1407	0.4890	3.3585
mspdie23	0.2545	1.4573	0.2889	1.6484
adl12	–0.0159	–0.6746	–0.0410	–1.7200
iadl12	0.0643	2.2692	0.0382	1.3670
dhlth12	–0.0307	–0.5561	0.0888	1.6706
adl23	0.0454	2.3124	0.0549	2.8570
iadl23	0.0429	1.8005	0.0539	2.2981
dhlth23	0.0003	0.0062	–0.0551	–1.0606
hmown23				
hoodpf23				
sadl12	0.0097	0.2181	0.0437	1.0901
siadl12	–0.1377	–2.4472	–0.0614	–1.1688
sdhlth12	0.1103	1.1330	–0.2603	–2.6730
sadl23	0.0389	0.7989	0.0780	1.7261
siadl23	0.0597	0.9476	–0.0663	–1.0395
sdhlth23	–0.0846	–0.7577	0.1738	1.6609
Likelihood	–2,080.6		–2,187.5	
Observations				
Negative	5,395 (88.47)		6,019 (89.21)	
Positive	703 (11.53)		728 (10.79)	
	Home Ownership—Regressions			
one1	7.6815	1.7802	5.1103	1.1990
one2	–2.1898	–0.7730	–1.2400	–0.4581
logm1	–2.3714	–1.7483	–1.5396	–1.1488
logm2	0.6749	0.7907	0.4041	0.4973
q1wb12	–1.0613	–5.2262	–1.1041	–5.4193
q4wb12	1.1765	7.0422	1.0881	6.7074
q1ib12	–0.0290	–0.1684	–0.0662	–0.3924
q4ib12	0.0937	0.6125	0.1012	0.6812
hoodpf12	–0.1658	–0.7884	–0.1677	–0.8208
condpf12	0.2148	1.0574	0.2051	1.0496
cwlth23	2.3604	5.2032	2.1317	4.8227

Table 11A.11 (continued)

	Females		Males	
Variable	Coefficient	*t*-statistic	Coefficient	*t*-statistic
male12	0.0218	0.1130	–0.0011	–.0058
single12	–0.8657	–5.5954	–0.8716	–4.0723
spdied23	–0.0252	–0.0885	–0.0496	–0.1491
mspdie23	–0.5329	–1.2507	–0.5408	–1.2679
adl12	–0.0116	–0.1682	–0.0333	–0.4443
iadl12	–0.1099	–1.2344	–0.1499	–1.5601
dhlth12	–0.0646	–0.4219	0.0686	0.4644
adl23	0.0338	0.6406	0.0291	0.5583
iadl23	–0.1331	–1.9536	–0.1406	–2.0826
dhlth23	–0.1977	–1.3659	–0.2537	–1.7724
hmown23				
hoodpf23				
sadl12				
siadl12				
sdhlth12				
sadl23				
siadl23				
sdhlth23				
Likelihood	–297.19		–312.63	
Observations				
Negative	510 (72.55)		521 (71.57)	
Positive	193 (27.45)		207 (28.43)	
	Neighborhood—Regressions			
one1	–5.1638	–1.0797	–4.5901	–0.9643
one2	0.2757	0.0596	2.9740	0.6733
logm1	0.8645	0.5791	0.7609	0.5103
logm2	–0.8698	–0.6188	–1.6126	–1.1976
q1wb12	0.1013	0.5063	0.0948	0.4845
q4wb12	–0.2939	–0.9384	–0.3117	–1.0448
q1ib12	0.1348	0.7182	0.1480	0.8100
q4ib12	–0.1193	–0.4647	–0.1579	–0.6348
hoodpf12	0.8861	4.6151	0.8985	4.8188
condpf12	–0.2322	–0.9571	–0.2772	–1.1564
cwlth23	–0.7506	–0.9264	–0.7756	–0.9988
male12	0.0804	0.3313	0.0758	0.3122
single12	0.6373	2.0210	0.4454	1.3979
spdied23	0.4967	1.1126	0.3041	0.6549
mspdie23	–0.2941	–0.4464	–0.2635	–0.3990
adl12	0.0176	0.2351	–0.0175	–0.2305
iadl12	–0.0031	–0.0374	0.0065	0.0787
dhlth12	0.0773	0.4279	0.0041	0.0231
adl23	0.0088	0.1523	0.0196	0.3490
iadl23	0.0110	0.1594	–0.0004	–0.0062
dhlth23	0.3240	1.7316	0.3610	1.9610
hmown23	0.2023	0.8696	0.1511	0.6839
hoodpf23				

(continued)

Table 11A.11 (continued)

	Females		Males	
Variable	Coefficient	*t*-statistic	Coefficient	*t*-statistic
sadl12				
siadl12				
sdhlth12				
sadl23				
siadl23				
sdhlth23				
Likelihood	–151.63		–161.16	
Observations				
Negative	655 (93.17)		677 (92.99)	
Positive	48 (6.83)		51 (7.01)	
	Dwelling Condition—Regressions			
one1	–5.1743	–1.0939	–6.9663	–1.4421
one2	–3.5792	–0.8303	–4.2874	–0.9590
logm1	0.8286	0.5609	1.1817	0.7866
logm2	0.2552	0.1961	0.2502	0.1862
q1wb12	0.4419	2.2384	0.3250	1.5940
q4wb12	0.1381	0.5095	0.1553	0.5661
q1ib12	0.2242	1.2029	0.3016	1.5771
q4ib12	0.0068	0.0291	0.0496	0.2087
hoodpf12	–0.1940	–0.8174	–0.1418	–0.5920
condpf12	1.2251	6.3478	1.2447	6.3933
cwlth23	0.3107	0.4315	0.2390	0.3295
male12	0.0589	0.2455	0.0409	0.1712
single12	0.5441	1.9046	1.2591	2.8052
spdied23	0.3015	0.7117	1.0219	1.8470
mspdie23	0.2957	0.5383	0.3331	0.6089
adl12	0.0211	0.2795	–0.0030	–0.0375
iadl12	0.0308	0.3609	0.0119	0.1350
dhlth12	0.1629	0.8842	0.0696	0.3692
adl23	–0.0349	–0.5898	–0.0153	–0.2606
iadl23	–0.0493	–0.6871	–0.0471	–0.6488
dhlth23	0.1229	0.6586	0.1519	0.7975
hmown23	0.4304	2.0561	0.4232	1.9598
hoodpf23	0.7158	2.8877	0.7217	2.8960
sadl12				
siadl12				
sdhlth12				
sadl23				
siadl23				
sdhlth23				
Likelihood	–160.73		–153.66	
Observations				
Negative	644 (91.61)		671 (92.17)	
Positive	59 (8.39)		57 (7.83)	

Note: Numbers in parentheses are percentages.

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Addendum

This addendum describes updates in data and analysis since the publication of this paper in the *Journal of Econometrics* (2003), 112, 3–53. The differences result from correcting some coding errors, producing a new dataset with revised imputations, and a new treatment of the simulation of wealth evolution. We also include in this addendum results from Lagrange multiplier (LM) tests of the Wold causal chain assumption that we impose on the

system of innovation equations. We comment on each of these points in turn.

We have corrected a health condition coding problem in the AHEAD data, described in "Data alert: Correction to F1156 [B7. Heart Condition]," from the HRS webpage. This coding problem produced a significant undercount of heart attack prevalence in wave 3. We have also corrected a problem in our count of new incidences, given existing previous condition, for the variables heart attack, cancer, and stroke, which was producing undercounts for the incidence variables. Updated tables 11.2R and 11.3R give summary statistics for the revised variables. These changes do not produce significant alterations to our overall results, but there are some differences in the coefficients of the incidence regressions. Table 11.8 in the published paper is updated in table 11.8R, which summarizes the coefficients that are now significant. A notable change is that, with the exception of cancer for males, we now obtain invariance of the models over time for the first three health conditions: cancer, heart disease, and stroke. The updated invariance and noncausality tests are given in table 11.9R. The wealth revisions have some impact on simulation results, which are updated in tables 11.12R and 11.13R.

In revising our code, we fixed the seed for the random number generator and produced a data set in which all imputations can be replicated. However, numerical imputations have changed, particularly imputed asset values. The tables listed above reflect these changes. The numerical differences from the previous published results are minor, and all the same results are obtained.

The simulations of wealth paths were producing, in some instances, unreasonable results in the first few years of simulation, with excessive increases in wealth observed in some cases. Our simulations start from a baseline population of seventy–seventy-five-year-olds in the first wave of the AHEAD, and these changes in wealth in the initial years of simulation are inconsistent with changes for the same population observed in subsequent waves. The observed pattern of wealth decrease is reproduced in the simulation after a few years, leading us to postulate, in loose terms, that our imputations and estimates capture the correct dynamics, but the wrong initial state of the baseline population. This is consistent with the findings of other researchers that wealth appears to be undercounted in AHEAD wave 1, due to undercounting of the categories in which assets are held. We therefore attempted to adjust to the correct initial state. We did this by calibrating the constant term of the wealth regressions using a cross-validation procedure. We calibrated the intercept so that the first year of simulated wealth changes (in terms of rate of change) matched, at the median, the observed transition for our baseline population. The changes in the intercept required to obtain this match are minor, as can be seen from table 11.14. Although we effectively force the prediction to match the

Table 11.2R Health Condition Variables in AHEAD

	Wave 1				Wave 2				Wave 3			
Label	Variable	*N*	Mean	SD	Variable	*N*	Mean	SD	Variable	*N*	Mean	SD
Health condition prevalence												
Doc ever told had cancer?	cancer1	6,489	0.137	0.344	cancer2	6,432	0.176	0.381	cancer3	5,709	0.190	0.392
Doc ever told heart attack/disease?	heart1	6,489	0.317	0.465	heart2	6,432	0.364	0.481	heart3	5,709	0.401	0.490
Doc ever told had stroke?	stroke1	6,489	0.089	0.285	stroke2	6,432	0.123	0.329	stroke3	5,709	0.143	0.350
Doc ever told had lung disease?	lung1	6,489	0.115	0.319	lung2	6,432	0.124	0.329	lung3	5,709	0.129	0.335
Doc ever told had diabetes?	diabet1	6,489	0.134	0.340	diabet2	5,741	0.147	0.354	diabet3	4,867	0.159	0.365
Doc ever told had high blood pressure?	highbp1	6,489	0.502	0.500	highbp2	5,741	0.525	0.499	highbp3	4,867	0.554	0.497
Seen Doc for arthritic in last 12 m?	arthrt1	6,489	0.267	0.442	arthrt2	5,741	0.277	0.448	arthrt3	4,867	0.279	0.449
Incontinence last 12 m?	incont1	6,489	0.203	0.402	incont2	6,489	0.318	0.466	incont3	6,489	0.405	0.491
Fall in last 12 m require treatment?	fall1	6,489	0.080	0.271	fall2	6,489	0.180	0.384	fall3	6,489	0.266	0.442
Ever fractured hip?	hipfrc1	6,489	0.050	0.218	hipfrc2	6,489	0.069	0.253	hipfrc3	6,489	0.088	0.283
Proxy Interview	proxyw1	6,489	0.104	0.306	proxyw2	5,741	0.132	0.339	proxyw3	4,867	0.154	0.361
Age-educ adjust cognitive impairment?	cogim1	6,489	0.256	0.436	cogim2	6,489	0.363	0.481	cogim3	6,487	0.438	0.496
Ever seen Doc for psych prob?	psych1	6,489	0.109	0.312	psych2	5,741	0.128	0.334	psych3	4,867	0.143	0.351
Depressed (cesd8>4)	depres1	6,489	0.099	0.299	depres2	5,741	0.086	0.280	depres3	4,867	0.100	0.300
Body Mass Index (Quetelet)	bmi1	6,475	25.369	4.529	bmi2	6,475	25.000	4.654	bmi3	6,475	24.679	4.680
Low BMI spline = max(0,20-bmi)	lobmi1	6,475	0.155	0.653	lobmi2	6,475	0.221	0.816	lobmi3	6,475	0.279	0.927
High BMI spline = max(0,bmi-25)	hibmi1	6,475	1.890	3.165	hibmi2	6,475	1.766	3.116	hibmi3	6,475	1.646	2.999
Current smoker?	smoknow1	6,489	0.103	0.304	smoknow2	5,741	0.079	0.270	smoknow3	4,867	0.066	0.249
No. of ADLs (needs help/difficult)	numadl1	6,489	0.725	1.391	numadl2	6,432	1.058	1.855	numadl3	5,709	1.254	2.016
No. of IADLs (needs help/difficult)	numiadl1	6,489	0.618	1.166	numiadl2	6,432	0.728	1.408	numiadl3	5,709	0.867	1.493
Poor/Fair self-reported health	dhlth1	6,489	0.373	0.484	dhlth2	5,741	0.368	0.482	dhlth3	4,867	0.434	0.496

(*continued*)

Table 11.2R (continued)

	Wave 1				Wave 2				Wave 3			
Label	Variable	*N*	Mean	SD	Variable	*N*	Mean	SD	Variable	*N*	Mean	SD
Health condition incidence												
Cancer[a]					jcancer2	6,432	0.057	0.231	jcancer3	5,685	0.058	0.234
Heart Attack/Condition[a]					jheart2	6,432	0.132	0.338	jheart3	5,685	0.154	0.361
Stroke[a]					jstroke2	6,432	0.058	0.233	jstroke3	5,685	0.070	0.255
Died Since Last Wave?					tdied2	6,489	0.115	0.319	tdied3	6,489	0.135	0.341
Lung Disease[a]					ilung2	6,432	0.024	0.153	ilung3	5,685	0.033	0.178
Diabetes[a]					idiabet2	5,741	0.024	0.154	idiabet3	4,867	0.025	0.156
High Blood Pressure (HBP)[a]					ihighbp2	5,741	0.054	0.226	ihighbp3	4,867	0.056	0.229
Arthritis[a]					iarthrt2	5,741	0.112	0.316	iarthrt3	4,867	0.117	0.322
Incontinence in last 12 months					jincont2	6,432	0.236	0.425	jincont3	5,709	0.263	0.440
Fall requiring treatment[a]					jfall2	6,432	0.129	0.335	jfall3	5,709	0.144	0.351
Hip Fracture[a]					jhipfrc2	6,432	0.023	0.151	jhipfrc3	5,709	0.027	0.163
Proxy Interview					proxyw2	5,741	0.132	0.339	proxyw3	4,867	0.154	0.361
Cognitive Impairment					icogim2	6,489	0.107	0.310	icogim3	6,487	0.075	0.263
Psychiatric Problems[a]					ipsych2	5,741	0.046	0.210	ipsych3	4,867	0.040	0.197
Depression					idepres2	5,741	0.051	0.220	idepres3	4,867	0.064	0.245
BMI better Indicator					bmibt2	6,489	0.174	0.379	bmibt3	6,489	0.149	0.356
BMI worse Indicator					bmiws2	6,489	0.150	0.357	bmiws3	6,489	0.143	0.350

Note: N = number of observations; SD = standard deviation.

[a]AHEAD Waves 2 and 3: "Since the last interview . . . ?"

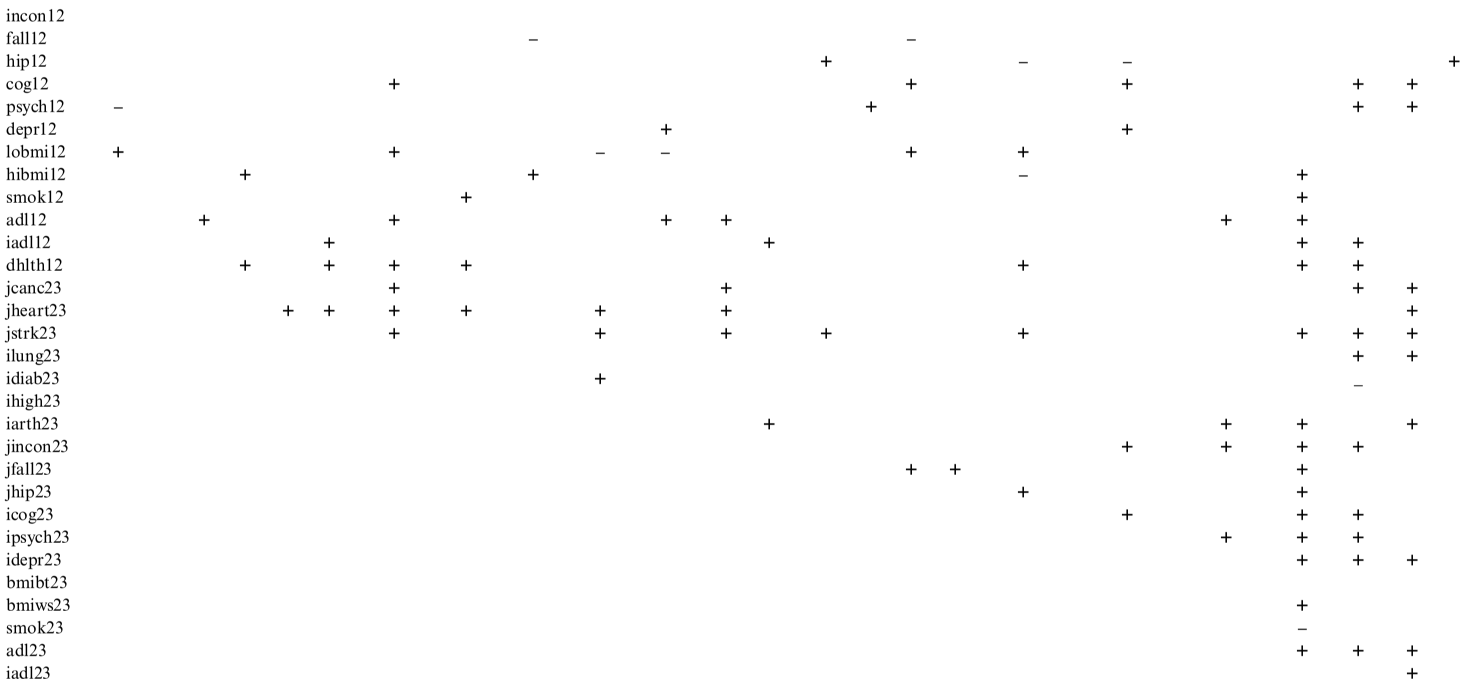
Table 11.3R SES and Demographic Variables in AHEAD

	Wave 1				Wave 2				Wave 3			
Label	Variable	*N*	Mean	SD	Variable	*N*	Mean	SD	Variable	*N*	Mean	SD
Wealth 97 Dol (V.2) (000)	c_2w1	6,489	0.643	0.075	c_2w2	5,736	2.473	1.122	c_2w3	4,862	2.508	1.112
Nonliquid wealth (000)	c_2n1	6,489	0.643	0.075	c_2n2	5,741	2.473	1.122	c_2n3	4,866	2.508	1.112
Liquid wealth (000)	c_2l1	6,489	0.643	0.075	c_2l2	5,737	2.473	1.122	c_2l3	4,864	2.508	1.112
Income 97 Dol (V.2) (000)	c_2i1	6,489	0.643	0.075	c_2i2	5,741	2.473	1.122				
1st quartile wealth indicator	q1wb1	6,489	0.258	0.438	q1wb2	6,489	0.208	0.406	q1wb3	6,489	0.185	0.388
4th quartile wealth indicator	q4wb1	6,489	0.213	0.409	q4wb2	6,489	0.241	0.428	q4wb3	6,489	0.203	0.402
1st quartile income indicator	q1wb1	6,489	0.256	0.436	q1ib2	6,489	0.219	0.414				
4th quartile income indicator	q4ib1	6,489	0.245	0.430	q4ib2	6,489	0.219	0.414				
Change residence?					moved2	5,741	0.096	0.295	moved3	4,867	0.102	0.303
Own residence?	dnhous1	6,489	0.667	0.472	dnhous2	5,741	0.660	0.474	dnhous3	4,867	0.640	0.480
Neighborhood safety poor/fair	hoodpf1	6,489	0.150	0.357	hoodpf2	5,741	0.139	0.346	hoodpf3	4,867	0.110	0.312
House condition poor/fair	condpf1	6,489	0.148	0.356	condpf2	5,741	0.139	0.346	condpf3	4,867	0.131	0.337
Widow?	widow1	6,489	0.416	0.493	widow2	6,432	0.453	0.498	widow3	5,706	0.496	0.500
Divorced/Separated?	divsep1	6,489	0.054	0.226	divsep2	6,432	0.053	0.225	divsep3	5,706	0.055	0.227
Married	married1	6,489	0.498	0.500	married2	6,432	0.459	0.498	married3	5,706	0.417	0.493
Never married?	nevmarr1	6,489	0.032	0.176	nevmarr2	6,432	0.031	0.174	nevmarr3	5,706	0.030	0.171
Age at interview (months)	agem1	6,489	27.379	2.662	agem2	6,489	26.958	2.544	agem3	5,775	26.958	2.544
Mother's death age	magedie1	6,489	73.833	17.386								
Father's death age	pagedie1	6,489	71.537	15.121								
Ever smoke?	smokev	6,489	0.526	0.499								
Education (years)	educ	6,489	0.605	0.489								
Educ > 10 yrs indicator	hs	6,489	0.605	0.489								
Educ > 14 yrs indicator	coll	6,489	0.143	0.35								

Note: *N* = number of observations; SD = standard deviation.

Table 11.8R **Statistically Significant Risk Factors for Incidence of Health Conditions (two percent significance level)**

	Cancer		Heart		Stroke		Mortality	Lung	Diabetes	HBP	Arthritis	Incontinence		Fall		Hip Fracture		Cognitive Impairment	Psychiatric Problem	Depression	ADL	IADL	SRHS	
	No	Yes	No	Yes	No	Yes	(All)	(All)	(All)	(All)	(All)	No	Yes	No	Yes	No	Yes	(All)	(All)	(All)	(All)	(All)	No	Yes
Female (pre-existing risk factor)																								
as70m12			+	+														+			+	+		
as80m12				–													–				–	–		
q1wb12																								
q4wb12																								
q1ib12																			+	+		+		
q4ib12																								
hs12	–										–													
coll12				+																				
hoodpf12																		+						
condpf12																								
nevmar12																								
widow12																			–					
divsep12																			–					
magedi12											+										–			
pagedi12																								
smokev12	+			+				+	–															
canc12							+													–				
heart12								+											+	+			+	
strk12				+								+												
lung12			+	+							+										+	+		+
diab12			+	+																	+		+	
high12					+																			
arth12							–					+									+		+	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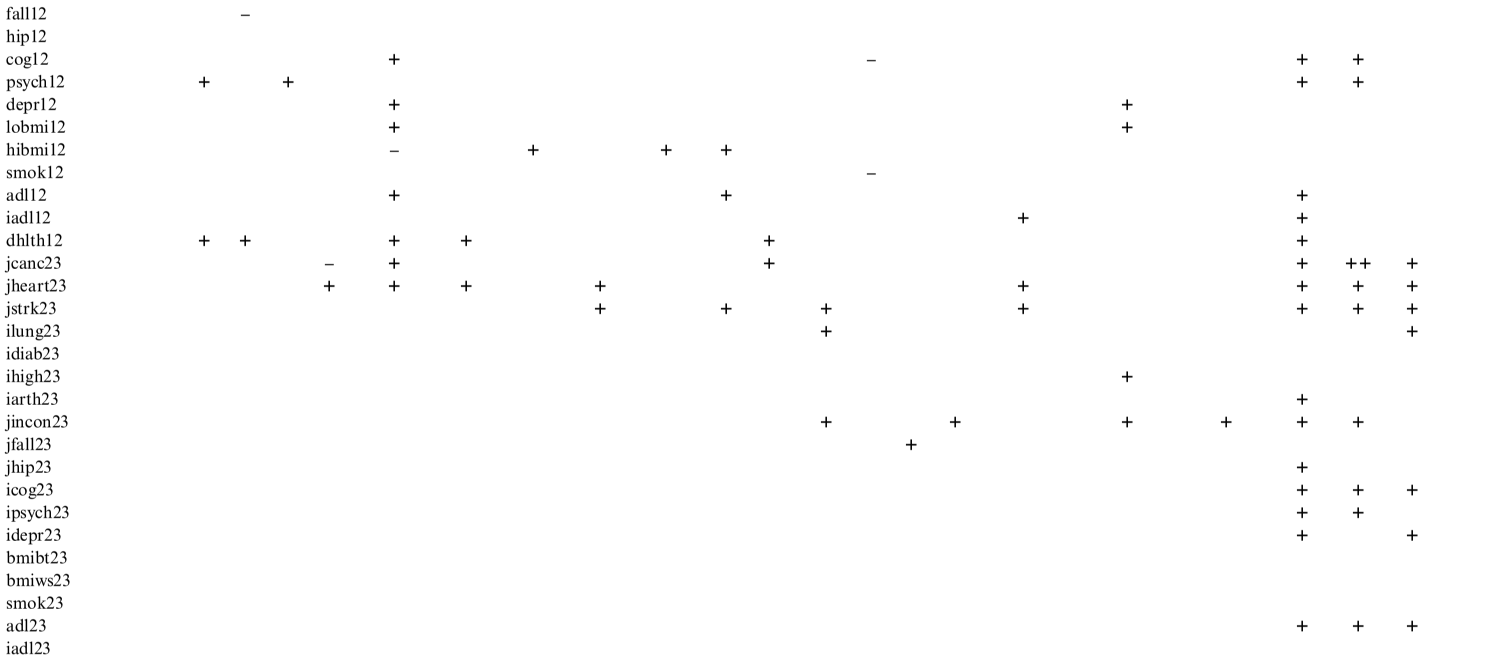
icog23
ipsych23
idepr23
bmibt23
bmiws23
smok23
adl23
iadl23

(*continued*)

Table 11.8R (continued)

	Cancer		Heart		Stroke		Mortality	Lung	Diabetes	HBP	Arthritis	Incontinence		Fall		Hip Fracture		Cognitive Impairment	Psychiatric Problem	Depression	ADL	IADL	SRHS	
	No	Yes	No	Yes	No	Yes	(All)	(All)	(All)	(All)	(All)	No	Yes	No	Yes	No	Yes	(All)	(All)	(All)	(All)	(All)	No	Yes
Male (pre-existing risk factor)																								
as70m12						+	+							+				+						
as80m12																								
q1wb12																								
q4wb12			–					–																
q1ib12																								
q4ib12																								
hs12																								
coll12									–															
hoodpf12		+																						
condpf12		–		–																		+		
nevmar12																								
widow12	+																							–
divsep12														+										
magedi12		–					–																	
pagedi12			–											–										+
smokev12																								
canc12							+																	
heart12										+												+		+
strk12																								
lung12							+					+										+		+
diab12		+	+	+			+		–															+
high12					+						+							–						+
arth12										+									+					
incon12																								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idepr23
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bmiws23
smok23
adl23
iadl23

Table 11.9R Health Innovations, Tests for Invariance and Causality (*p*-values)

	Invariance			
	With SES		Without SES	
Variable	Female	Male	Female	Male
CANCER	0.528	0.016	0.309	0.007
CANCER—no previous	0.867	0.129	0.812	0.064
CANCER—previous	0.068	0.044	0.079	0.083
HEART	0.085	0.690	0.052	0.619
HEART—no previous	0.170	0.823	0.060	0.498
HEART—previous	0.547	0.692	0.492	0.725
STROKE	0.384	0.204	0.266	0.135
STROKE—no previous	0.172	0.258	0.160	0.180
STROKE—previous	0.907	0.045	0.784	0.087
MORTALITY	0.221	0.378	0.315	0.491
LUNG	0.552	0.689	0.483	0.502
DIABETES	0.189	0.234	0.281	0.128
HIGH BLOOD PRESSURE	0.007	0.393	0.024	0.172
ARTHRITIS	0.046	0.071	0.032	0.167
INCONTINENCE	0.781	0.351	0.597	0.682
INCONTINENCE—no previous	0.706	0.102	0.426	0.555
INCONTINENCE—previous	0.962	0.793	0.892	0.730
FALL	0.904	0.263	0.790	0.207
FALL—no previous	0.792	0.140	0.612	0.133
FALL—previous	0.365	0.000	0.358	0.000
HIP FRACTURE	0.491	0.126	0.280	0.053
HIP FRACTURE—no previous	0.930	0.300	0.724	0.123
HIP FRACTURE—previous				
PROXY INTERVIEW	0.301	0.591	0.465	0.378
PROXY INTERVIEW—no previous	0.127	0.795	0.444	0.606
PROXY INTERVIEW—previous				
COGNITIVE IMPAIRMENT	0.005	0.288	0.008	0.143
PSYCHIATRIC	0.127	0.295	0.052	0.365
DEPRESSION	0.211	0.944	0.299	0.934
BODY MASS INDEX	0.260	0.009	0.251	0.002
CURRENT SMOKER	0.420	0.710	0.214	0.504
CURRENT SMOKER—no previous	0.133	0.033	0.078	0.040
CURRENT SMOKER—previous	0.797		0.733	
ADL	0.001	0.021	0.007	0.038
IADL	0.603	0.008	0.388	0.003
SELF RATED HEALTH	0.376	0.634	0.341	0.607
SELF RATED HEALTH—no previous	0.514	0.251	0.558	0.215
SELF RATED HEALTH—previous	0.239	0.165	0.176	0.189

Table 11.9R (continued)

	Noncausality					
	Wave 1–2		Wave 2–3		Wave 1–3	
	Female	Male	Female	Male	Female	Male
CANCER	0.987	0.588	0.408	0.165	0.613	0.198
CANCER—no previous	0.704	0.658	0.289	0.173	0.313	0.166
CANCER—previous	0.143	0.228	0.472	0.017	0.299	0.036
HEART	0.540	0.054	0.396	0.996	0.398	0.243
HEART—no previous	0.872	0.344	0.750	0.412	0.736	0.039
HEART—previous	0.104	0.071	0.153	0.737	0.019	0.172
STROKE	0.757	0.837	0.584	0.028	0.657	0.059
STROKE—no previous	0.415	0.955	0.671	0.064	0.713	0.203
STROKE—previous	0.824	0.301	0.882	0.155	0.799	0.383
MORTALITY	0.098	0.275	0.883	0.306	0.652	0.364
LUNG	0.166	0.174	0.866	0.122	0.343	0.010
DIABETES	0.615	0.141	0.020	0.236	0.110	0.025
HIGH BLOOD PRESSURE	0.342	0.996	0.083	0.941	0.534	0.990
ARTHRITIS	0.179	0.029	0.242	0.713	0.085	0.395
INCONTINENCE	0.763	0.019	0.254	0.800	0.163	0.463
INCONTINENCE—no previous	0.914	0.037	0.602	0.197	0.453	0.641
INCONTINENCE—previous	0.900	0.247	0.168	0.664	0.149	0.244
FALL	0.532	0.687	0.945	0.454	0.600	0.592
FALL—no previous	0.501	0.697	0.944	0.251	0.558	0.532
FALL—previous	0.329	0.000	0.803	0.000	0.696	0.053
HIP FRACTURE	0.613	0.597	0.370	0.666	0.159	0.430
HIP FRACTURE—no previous	0.461	0.461	0.706	0.725	0.128	0.234
HIP FRACTURE—previous						
PROXY INTERVIEW	0.017	0.486	0.870	0.127	0.254	0.032
PROXY INTERVIEW—no previous	0.002	0.793	0.541	0.121	0.087	0.073
PROXY INTERVIEW—previous						
COGNITIVE IMPAIRMENT	0.001	0.259	0.259	0.200	0.002	0.026
PSYCHIATRIC	0.043	0.238	0.214	0.069	0.004	0.065
DEPRESSION	0.007	0.143	0.261	0.377	0.011	0.065
BODY MASS INDEX	0.188	0.839	0.931	0.711	0.673	0.684
CURRENT SMOKER	0.603	0.282	0.974	0.693	0.625	0.129
CURRENT SMOKER—no previous	0.335	0.196	0.874	0.980	0.515	0.962
CURRENT SMOKER—previous	0.375	0.325	0.547	0.325	0.274	0.025
ADL	0.077	0.014	0.346	0.971	0.828	0.358
IADL	0.374	0.051	0.817	0.243	0.241	0.009
SELF RATED HEALTH	0.013	0.048	0.100	0.301	0.001	0.020
SELF RATED HEALTH—no previous	0.015	0.187	0.202	0.121	0.007	0.034
SELF RATED HEALTH—previous	0.225	0.195	0.557	0.256	0.193	0.175

(*continued*)

Table 11.9R (continued)

	Joint (Invariance + Noncausality)		Relative Odds (high vs. low SES)			
			Female		Male	
	Female	Male	Odds	SE	Odds	SE
CANCER	0.600	0.012	1.31	0.46	0.73	0.27
CANCER—no previous	0.778	0.084	1.24	0.48	0.67	0.29
CANCER—previous	0.067	0.011	5.85	7.75	2.69	3.42
HEART	0.099	0.571	0.97	0.21	1.02	0.24
HEART—no previous	0.278	0.446	0.82	0.25	1.30	0.50
HEART—previous	0.169	0.516	1.13	0.32	0.88	0.26
STROKE	0.481	0.080	0.73	0.25	0.94	0.35
STROKE—no previous	0.271	0.189	0.68	0.27	0.82	0.36
STROKE—previous	0.948	0.054	1.15	0.93	1.61	1.25
MORTALITY	0.308	0.360	0.69	0.21	1.17	0.41
LUNG	0.504	0.203	0.33	0.17	0.50	0.43
DIABETES	0.101	0.062	0.69	0.41	1.48	3.19
HIGH BLOOD PRESSURE	0.013	0.666	0.90	0.29	0.74	0.36
ARTHRITIS	0.020	0.082	1.03	0.22	0.54	0.16
INCONTINENCE	0.615	0.373	0.85	0.13	1.11	0.30
INCONTINENCE—no previous	0.695	0.158	0.77	0.20	1.02	0.36
INCONTINENCE—previous	0.858	0.684	0.94	0.09	1.13	0.29
FALL	0.913	0.328	0.98	0.19	0.92	0.31
FALL—no previous	0.808	0.181	1.03	0.25	0.86	0.32
FALL—previous	0.470	0.000	1.00	0.27	89.61	267.82
HIP FRACTURE	0.347	0.143	0.33	0.22	0.21	0.28
HIP FRACTURE—no previous	0.785	0.238	0.23	0.17	0.14	0.19
HIP FRACTURE—previous						
PROXY INTERVIEW	0.248	0.261	0.46	0.16	0.48	0.17
PROXY INTERVIEW—no previous	0.061	0.531	0.27	0.11	0.40	0.20
PROXY INTERVIEW—previous						
COGNITIVE IMPAIRMENT	0.000	0.089	0.78	0.17	0.59	0.19
PSYCHIATRIC	0.012	0.137	0.39	0.15	0.16	0.12
DEPRESSION	0.042	0.753	0.38	0.15	0.29	0.15
BODY MASS INDEX	0.345	0.019				
CURRENT SMOKER	0.492	0.528	0.46	0.37	0.92	1.11
CURRENT SMOKER—no previous	0.165	0.097	0.11	0.16	19.92	132.75
CURRENT SMOKER—previous	0.714		0.93	0.16	0.95	0.18
ADL	0.004	0.024				
IADL	0.509	0.001				
SELF RATED HEALTH	0.044	0.278	0.67	0.08	0.64	0.09
SELF RATED HEALTH—no previous	0.138	0.091	0.59	0.12	0.51	0.13
SELF RATED HEALTH—previous	0.177	0.116				

Notes: SE = standard error.

Table 11.12R Simulation Outcomes

	Scenario	70	75	80	85	90	95
		White Females					
Survival probability[a]	S0	1.000	0.907	0.747	0.502	0.247	0.082
	S1	1.000	0.912	0.761	0.526	0.268	0.092
	S2	1.000	0.887	0.664	0.354	0.117	0.024
Cancer[b]	S0	0.117	0.180	0.229	0.264	0.280	0.267
	S1	0.117	0.174	0.222	0.257	0.275	0.259
	S2	0.117	0.213	0.277	0.309	0.335	0.301
Heart disease[b]	S0	0.225	0.329	0.457	0.561	0.630	0.667
	S1	0.225	0.323	0.438	0.538	0.604	0.625
	S2	0.225	0.361	0.521	0.635	0.701	0.733
Stroke[b]	S0	0.044	0.108	0.197	0.274	0.344	0.392
	S1	0.044	0.107	0.184	0.261	0.324	0.358
	S2	0.044	0.119	0.223	0.311	0.393	0.458
Lung disease[b]	S0	0.104	0.153	0.183	0.193	0.177	0.159
	S1	0.104	0.147	0.174	0.183	0.174	0.142
	S2	0.104	0.196	0.266	0.289	0.275	0.211
Diabetes[b]	S0	0.114	0.163	0.189	0.196	0.195	0.173
	S1	0.114	0.000	0.000	0.000	0.000	0.000
	S2	0.114	0.202	0.252	0.266	0.253	0.213
High blood pressure[b]	S0	0.467	0.584	0.675	0.733	0.763	0.787
	S1	0.467	0.581	0.672	0.740	0.769	0.809
	S2	0.467	0.622	0.729	0.799	0.842	0.890
Arthritis[b]	S0	0.227	0.495	0.677	0.794	0.871	0.916
	S1	0.227	0.506	0.676	0.800	0.874	0.935
	S2	0.227	0.574	0.775	0.885	0.944	0.961
Incontinence[b]	S0	0.231	0.410	0.591	0.732	0.831	0.892
	S1	0.231	0.422	0.601	0.747	0.842	0.901
	S2	0.231	0.438	0.646	0.796	0.879	0.938
Fall[b]	S0	0.072	0.276	0.462	0.628	0.762	0.860
	S1	0.072	0.276	0.462	0.624	0.754	0.854
	S2	0.072	0.273	0.473	0.652	0.776	0.854
Hip fracture[b]	S0	0.029	0.045	0.069	0.104	0.137	0.180
	S1	0.029	0.044	0.070	0.102	0.138	0.157
	S2	0.029	0.064	0.112	0.183	0.240	0.298
Proxy interview	S0	0.036	0.057	0.090	0.149	0.215	0.282
	S1	0.036	0.062	0.089	0.144	0.209	0.279
	S2	0.036	0.102	0.185	0.295	0.405	0.469
Cognitive impairment[b]	S0	0.108	0.254	0.440	0.619	0.741	0.812
	S1	0.108	0.261	0.434	0.612	0.747	0.813
	S2	0.108	0.403	0.678	0.845	0.921	0.963
Psychiatric disease[b]	S0	0.153	0.223	0.310	0.382	0.425	0.459
	S1	0.153	0.225	0.317	0.390	0.421	0.451
	S2	0.153	0.320	0.506	0.635	0.726	0.772
Depression[b]	S0	0.067	0.168	0.280	0.380	0.465	0.519
	S1	0.067	0.178	0.290	0.399	0.488	0.572
	S2	0.067	0.253	0.429	0.573	0.664	0.711
Body mass index[c]	S0	25.657	24.791	23.673	22.435	21.389	20.716
	S1	25.657	24.781	23.640	22.413	21.375	20.384
	S2	25.657	24.424	23.018	21.782	20.852	20.483

(*continued*)

Table 11.12R (continued)

	Scenario	70	75	80	85	90	95
Current smoker[b]	S0	0.130	0.088	0.059	0.038	0.021	0.015
	S1	0.130	0.086	0.057	0.037	0.025	0.016
	S2	0.130	0.120	0.100	0.071	0.045	0.039
ADL limits[c]	S0	0.307	0.272	0.607	1.075	1.581	2.071
	S1	0.307	0.245	0.558	1.029	1.529	2.013
	S2	0.307	0.478	1.183	2.047	2.721	3.365
IADL limits[c]	S0	0.241	0.113	0.263	0.534	0.790	1.110
	S1	0.241	0.106	0.257	0.503	0.770	1.096
	S2	0.241	0.249	0.688	1.311	1.751	2.045
Poor/fair self-rated health[b]	S0	0.249	0.309	0.433	0.530	0.583	0.605
	S1	0.249	0.288	0.416	0.504	0.565	0.594
	S2	0.249	0.515	0.717	0.815	0.846	0.882
		White Males					
Survival probability[a]	S0	1.000	0.822	0.555	0.285	0.090	0.018
	S1	1.000	0.838	0.592	0.313	0.116	0.026
	S2	1.000	0.834	0.513	0.201	0.040	0.005
Cancer[b]	S0	0.138	0.235	0.319	0.402	0.491	0.631
	S1	0.138	0.242	0.326	0.411	0.493	0.578
	S2	0.138	0.233	0.309	0.391	0.480	0.660
Heart[b]	S0	0.361	0.504	0.639	0.734	0.790	0.862
	S1	0.361	0.507	0.633	0.730	0.828	0.861
	S2	0.361	0.486	0.612	0.706	0.748	0.868
Stroke[b]	S0	0.071	0.140	0.216	0.278	0.341	0.379
	S1	0.071	0.134	0.212	0.282	0.311	0.366
	S2	0.071	0.186	0.308	0.400	0.502	0.528
Lung disease[b]	S0	0.153	0.185	0.218	0.254	0.241	0.222
	S1	0.153	0.184	0.224	0.242	0.241	0.272
	S2	0.153	0.167	0.181	0.211	0.232	0.189
Diabetes[b]	S0	0.133	0.165	0.186	0.197	0.188	0.138
	S1	0.133	0.000	0.000	0.000	0.000	0.000
	S2	0.133	0.136	0.134	0.125	0.083	0.038
High blood pressure[b]	S0	0.440	0.547	0.626	0.686	0.709	0.690
	S1	0.440	0.548	0.631	0.687	0.740	0.833
	S2	0.440	0.585	0.681	0.759	0.804	0.887
Arthritis[b]	S0	0.175	0.398	0.576	0.706	0.782	0.803
	S1	0.175	0.412	0.587	0.707	0.775	0.826
	S2	0.175	0.512	0.732	0.857	0.944	1.000
Incontinence[b]	S0	0.099	0.250	0.416	0.584	0.724	0.808
	S1	0.099	0.247	0.402	0.580	0.726	0.808
	S2	0.099	0.294	0.501	0.684	0.846	0.962
Fall[b]	S0	0.041	0.157	0.288	0.419	0.536	0.601
	S1	0.041	0.151	0.284	0.411	0.500	0.592
	S2	0.041	0.226	0.430	0.608	0.739	0.887
Hip fracture[b]	S0	0.024	0.032	0.042	0.067	0.094	0.148
	S1	0.024	0.030	0.045	0.064	0.091	0.139
	S2	0.024	0.067	0.131	0.207	0.295	0.472

Table 11.12R (continued)

	Scenario	70	75	80	85	90	95
Proxy Interview	S0	0.110	0.106	0.119	0.149	0.166	0.163
	S1	0.110	0.119	0.129	0.144	0.184	0.171
	S2	0.110	0.181	0.256	0.313	0.353	0.340
Cognitive impairment[b]	S0	0.144	0.323	0.505	0.660	0.768	0.837
	S1	0.144	0.326	0.508	0.668	0.778	0.829
	S2	0.144	0.435	0.686	0.835	0.920	0.868
Psychiatric disease[b]	S0	0.085	0.136	0.188	0.225	0.272	0.310
	S1	0.085	0.133	0.183	0.237	0.268	0.265
	S2	0.085	0.188	0.323	0.429	0.498	0.604
Depression[b]	S0	0.039	0.098	0.169	0.240	0.263	0.291
	S1	0.039	0.097	0.171	0.239	0.295	0.338
	S2	0.039	0.206	0.350	0.473	0.538	0.623
Body mass index[c]	S0	26.113	25.608	25.125	24.654	24.542	24.734
	S1	26.113	25.682	25.207	24.786	24.503	24.650
	S2	26.113	24.879	23.968	23.508	23.506	25.136
Current smoker[b]	S0	0.125	0.057	0.034	0.017	0.004	0.000
	S1	0.125	0.060	0.039	0.019	0.007	0.000
	S2	0.125	0.085	0.060	0.031	0.011	0.000
ADL limits[c]	S0	0.324	0.290	0.643	1.121	1.753	2.025
	S1	0.324	0.318	0.651	1.152	1.716	2.258
	S2	0.324	0.734	1.741	2.773	3.938	4.340
IADL limits[c]	S0	0.333	0.133	0.304	0.580	0.867	1.143
	S1	0.333	0.135	0.311	0.570	0.959	1.303
	S2	0.333	0.392	0.894	1.676	2.444	2.415
Poor/fair self-rated health[b]	S0	0.286	0.370	0.461	0.507	0.511	0.483
	S1	0.286	0.360	0.454	0.484	0.510	0.512
	S2	0.286	0.598	0.745	0.812	0.819	0.849

Notes: S0 = baseline; S1 = no diabetes; S2 = all low SES.

sample values for the first year, this is not so for subsequent years of the simulation, which produces no anomalous results in subsequent waves.

As mentioned in a note added in proof which appeared with our reply to published comments, LM tests for the triangular Wold causal chain structure of our model have been calculated. The results support that assumption in most cases. Table 11.15 reports the *t*-statistics of pairwise independence tests. The notable exceptions to acceptance of the causal chain structure are the mortality, ADL, and IADL equations, whose pairwise independence fails with respect to most of the remaining conditions. The joint test for independences across equations is not rejected for the case of males and is rejected for the case of females. The rejection for females is eliminated by removing the equations for mortality, BMI, ADL, and IADL.

Table 11.13R Wealth and Income in AHEAD and in the Baseline Scenario, by Age

	70–74	75–79	80–84	85–89	90+
AHEAD cross-cohort data					
Total wealth (000)					
1st quartile	63.51	44.07	29.28	15.13	1.64
Median	144.04	110.76	90.62	71.19	38.37
3rd quartile	307.29	239.41	190.23	180.84	113.49
Liquid wealth (000)					
1st quartile	2.18	1.09	0.54	0.32	0.00
Median	27.28	15.24	10.91	8.64	4.36
3rd quartile	100.39	70.93	64.85	54.15	34.21
Nonliquid wealth (000)					
1st quartile	38.09	15.24	5.40	0.11	0.00
Median	91.66	77.00	60.02	43.38	1.63
3rd quartile	176.84	147.31	110.25	108.45	63.29
Income (000)					
1st quartile	14.19	12.00	9.76	8.68	6.87
Median	24.01	19.64	15.55	13.56	10.37
3rd quartile	37.96	32.54	28.37	23.94	15.71
Baseline simulation data					
Total wealth (000)					
1st quartile	56.61	71.59	57.48	34.76	20.62
Median	136.41	121.52	98.82	69.50	52.88
3rd quartile	292.45	202.94	148.19	107.54	86.91
Liquid wealth (000)					
1st quartile	1.64	10.24	6.68	0.98	–1.74
Median	25.10	29.24	22.28	14.39	10.63
3rd quartile	96.35	60.16	41.96	29.10	23.25
Nonliquid wealth (000)					
1st quartile	34.10	53.69	43.52	26.45	15.76
Median	87.84	87.54	73.89	53.80	42.45
3rd quartile	170.85	130.09	106.62	81.51	68.95
Income (000)					
1st quartile	13.69	13.36	11.98	10.00	8.68
Median	22.22	22.99	21.59	18.89	17.01
3rd quartile	36.23	35.35	34.17	30.19	26.17

Table 11.14 Calibration of Wave 1 Wealth Levels

Model	Calibrated Constant	Estimated Constant	Standard Deviation
Liquid			
Couples	0.0744	0.0751	0.0078
Spouse died	0.0876	0.0839	0.0210
Singles			
Males	0.0567	0.0708	0.0156
Females	0.0635	0.0794	0.0061
Nonliquid			
Couples	0.0626	0.0592	0.0046
Spouse died	0.1062	0.0954	0.0154
Singles			
Males	0.0554	0.0632	0.0104
Females	0.0721	0.0824	0.0067

Table 11.15 **LM Tests of Wold Causal Chain Triangular Structure (*t*-statistics)**

	JCANCER2	JHEART2	JSTROKE2	TDIED2	ILUNG2	IDIABET2	IHIGHBP2	IARTHRT2	JINCONT2	JFALL2	IHIPFRC2	PROXYW2	ICOGIM2	IPSYCH2	IDEPRES2	BMI2	SMOKNOW2	NUMADL2	NUMIADL2
Pairwise Tests: Female																			
JCANCER2																			
JHEART2	1.49																		
JSTROKE2	0.37	1.86																	
TDIED2	1.80	4.90	1.83																
ILUNG2	0.31	–1.09	0.31	–0.37															
IDIABET2	0.43	–0.72	–0.12	1.68	0.15														
IHIGHBP2	–0.05	–0.07	0.43	–0.06	0.39	0.25													
IARTHRT2	–0.09	–0.84	–0.25	1.51	–0.09	–0.32	0.11												
JINCONT2	–0.64	0.33	0.40	–3.34	0.18	–0.08	0.29	–0.25											
JFALL2	0.16	0.15	0.24	–1.16	0.18	–0.34	0.11	0.21	0.41										
JHIPFRC2	–0.30	0.88	–0.37	–2.25	0.28	–0.34	–0.29	0.11	1.59	2.89									
PROXYW2	0.25	0.68	0.61	–4.87	0.06	–0.63	–1.22	–0.77	2.40	1.82	1.91								
ICOGIM2	–0.47	1.95	0.98	–4.30	–0.65	–1.03	–0.22	–1.17	0.16	0.87	1.76	4.06							
IPSYCH2	–1.03	–0.19	0.75	–0.13	0.31	–0.01	–0.11	0.05	0.45	–0.01	–0.14	0.40	–0.17						
IDEPRES2	–1.87	0.82	0.77	–1.22	0.36	–0.19	–0.28	0.33	0.53	–0.74	0.66	–0.54	1.10	–0.30					
BMI2	–0.17	0.55	–0.56	2.73	–0.32	0.62	–0.34	0.53	–0.46	–0.47	–1.08	–1.41	–0.84	0.08	–0.76				
SMOKNOW2	0.29	0.47	–0.41	1.91	–0.39	0.43	0.12	0.01	0.16	–0.64	–0.90	–1.53	–0.99	–0.01	–1.18	–2.02			
NUMADL2	–0.38	2.62	1.07	–5.77	–0.07	–0.97	0.35	–0.27	2.07	1.54	2.45	2.60	2.20	0.27	2.23	–4.78	–1.15		
NUMIADL2	–1.34	2.20	0.64	–3.71	0.20	–1.03	0.13	–0.60	1.49	1.10	1.76	2.71	2.02	0.28	0.49	–4.37	–0.25	6.88	
DHLTH2	0.85	–0.25	–0.38	–0.38	0.01	0.43	0.34	–0.05	0.33	–0.01	0.11	0.71	–0.62	0.39	–0.50	–3.99	–0.40	1.65	2.88
Pairwise Tests: Male																			
JCANCER2																			
JHEART2	1.03																		
JSTROKE2	0.46	1.80																	

(*continued*)

Table 11.15 (continued)

	JCANCER2	JHEART2	JSTROKE2	TDIED2	ILUNG2	IDIABET2	IHIGHBP2	IARTHRT2	JINCONT2	JFALL2	IHIPFRC2	PROXYW2	ICOGIM2	IPSYCH2	IDEPRES2	BMI2	SMOKNOW2	NUMADL2	NUMIADL2
TDIED2	2.04	3.83	1.49																
ILUNG2	–0.75	–0.03	0.38	–0.54															
IDIABET2	–0.13	–0.79	–0.16	0.04	–0.27														
IHIGHBP2	–0.13	0.12	–0.05	–0.56	0.36	0.02													
IARTHRT2	0.20	–0.71	–0.19	0.26	0.08	0.16	–0.15												
JINCONT2	–0.09	0.28	0.08	–0.81	0.19	–0.11	–0.06	–0.01											
JFALL2	–0.13	1.14	0.82	–3.04	0.48	–0.05	–0.25	0.34	0.55										
JHIPFRC2	–1.32	–0.11	0.60	–0.62	0.34	–0.13	0.81	–1.58	1.00	2.19									
PROXYW2	–0.57	–0.09	0.90	–1.72	0.17	0.55	–0.05	–0.25	0.92	0.46	0.69								
ICOGIM2	–0.64	0.12	1.04	–2.91	0.57	0.11	0.09	0.13	0.45	0.96	–0.31	1.60							
IPSYCH2	–0.52	–0.63	0.00	0.60	–0.12	0.08	–0.11	0.08	0.17	–0.16	0.62	–0.37	–0.84						
IDEPRES2	–1.34	–0.58	0.50	–2.17	–0.32	0.04	–0.11	–0.08	1.33	0.92	–0.52	–2.12	1.74	–0.87					
BMI2	0.40	–0.27	–0.40	2.78	–0.02	0.13	0.25	0.16	0.08	–0.52	0.23	–0.04	–1.43	0.14	–0.44				
SMOKNOW2	0.20	0.06	–0.04	0.35	0.02	–0.58	0.71	–0.15	–0.77	0.12	–0.65	–0.28	–0.41	–0.32	–0.19	–1.85			
NUMADL2	–0.64	0.32	0.86	–2.24	0.91	0.11	–0.11	0.20	0.81	1.31	0.40	0.94	2.08	–0.24	1.86	–4.27	0.50		
NUMIADL2	–1.49	–0.45	0.77	–1.28	0.45	0.26	0.10	0.43	0.45	1.01	0.65	1.31	1.23	–0.19	1.46	–1.84	–0.17	2.85	
DHLTH2	–0.49	–0.23	0.20	–0.18	0.18	–0.32	0.49	0.11	0.26	0.90	0.71	–0.06	0.55	–0.46	1.15	–2.32	0.59	2.24	2.20
									Cumulative Tests										
Female																			
LM	2.2	5.5	37.4	39.9	44.5	45.2	48.2	63.2	65.4	77.8	105.3	136.5	139.0	151.7	211.9	230.8	273.5	330.5	370.5
p-value	0.138	0.138	0.000	0.000	0.000	0.002	0.010	0.003	0.025	0.023	0.002	0.000	0.001	0.002	0.000	0.000	0.000	0.000	0.000
Male																			
LM	1.1	4.3	26.0	27.0	27.7	28.6	29.3	30.0	42.4	51.8	58.0	71.3	74.5	89.2	151.6	165.6	184.3	204.9	222.2
p-value	0.303	0.231	0.000	0.003	0.024	0.124	0.399	0.751	0.581	0.599	0.747	0.692	0.896	0.866	0.027	0.043	0.043	0.039	0.055
DF	1	3	6	10	15	21	28	36	45	55	66	78	91	105	120	136	153	171	190

Comment

James M. Poterba

This paper presents a wealth of interesting new information on the relationship between various measures of socioeconomic status (SES) and the health of elderly individuals. While most previous studies of the correlation between SES and health status have relied on cross-sectional data, this paper exploits panel data. Repeated observations on the same individuals make it possible to develop more refined tests than those in past studies. In particular, the authors are able to study how income, wealth, and other socioeconomic conditions are associated with the onset of various health conditions, rather than simply the point-in-time correlation between SES and health status.

The core of the study consists of two related empirical projects. The first explores how SES affects changes in health status, while the second explores the relationship between changes in health status and changes in financial circumstances.

For the first project, the authors develop some "central tendency measures" of socioeconomic status. They find that some of the components of socioeconomic status have greater impact on health than others. Wealth, for example, has a stronger link to health than some of the other components of SES. Many of their findings suggest only modest links between SES and the onset of adverse health conditions. One worry is that by using many different variables to construct a measure of socioeconomic status, the authors have increased the chance of finding weak links between the SES variables and health outcomes. It is always possible to find weak effects by including many marginally influential variables in an empirical analysis. Focusing the analysis on a few key variables relating to socioeconomic status, such as wealth, income, and education, would seem like a natural direction for further analysis. It may ultimately be possible to rank different measures of income, wealth, and other aspects of socioeconomic status in terms of their predictive power for various health events.

One by-product of the study of how SES affects the onset of health conditions is a comparison between measures of the incidence of chronic conditions in the AHEAD sample and the aggregate population. In some cases, there appear to be disparities between the two data sets; this seems like a natural subject for future study. Another related suggestion for further work involves distinguishing between long-term and short-term measures of socioeconomic status. It is possible that transitory shocks to socioeconomic status, such as a temporary decline in income, may have a less important effect on health status than persistent differences due to wealth.

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One of the most important advances in this study is the disaggregate analysis of various measures of health status. By studying acute conditions separately from chronic conditions, and distinguishing mental conditions from acute physical conditions due to accidents and other factors, the paper is able to show that there are important differences in the link between SES and the onset of different long-term health limitations. This is a key insight if one tries to forecast how changes in future economic circumstances will affect the health of the future elderly population.

The second component of this study explores the extent to which changes in socioeconomic status can be explained by changes in health status. Because some changes in health status may require expensive treatment or movement to a nursing home or other costly facility, there is a presumption that health changes might account for some changes in financial circumstances. The results on the link from health status changes to financial status changes are weak. This is one aspect of the paper that could benefit from further data analysis. Disaggregating survey respondents by health insurance status, and focusing on relatively expensive health changes, might reveal a stronger relationship between some types of health status change and subsequent financial changes. For many AHEAD participants, income flows—pensions, Social Security, and related flows—are not affected by health status. It is therefore possible that the links from health changes to income changes for this age group are more muted than the changes for younger elderly.

In addition to its two significant empirical components, the paper also proposes a conceptual framework for analyzing how wealth and other measures of socioeconomic status are related to health. The "invariance" condition proposed here requires that the relationship between SES components and health status must be stable over time. Invariance is stipulated as a logical precondition for a causal relationship between SES and health status. It is not clear that this is a reasonable restriction, however. There are many reasons to suspect that the relationship might change over time, even if there is a true underlying link. For example, medical treatment technology may change. A procedure that was expensive at one point in time may become less expensive and widely available at a later date, thereby changing the relationship between SES and observed health status. Even if income and wealth are positively correlated with access to this procedure at all points in time, a decline in the price of the technology might alter the slope of this relationship. Similarly, there could be changes over time in behavior or other factors that affect health status. These could lead to changes in the measured relationship between health status and socioeconomic status. Once again, the difficulty is that even if higher socioeconomic status is associated with a lower likelihood of chronic conditions at all points during the sample period, a test for the stability of coefficients over time might reject this restriction. The diffusion of exercise or other per-

sonal behaviors across the socioeconomic spectrum could lead to time-varying coefficients even in the presence of a causal link.

This paper opens a broad new field of inquiry directly at the detailed mapping of links from income, wealth, and other aspects of socioeconomic status to the level of, and changes in, health status. As panel data sets become more common in the study of elderly populations and researchers have increased access to information on both medical conditions and economic circumstances, this research is likely to reveal more and more subtle aspects of these relationships. This paper represents a very important step in this research program.

Response

Peter Adams, Michael D. Hurd, Daniel McFadden, Angela Merrill, and Tiago Ribeiro

The problem of how to describe, detect, and measure causal effects has an importance in economic and social policy analysis that transcends the specifics of our paper, and our discussants provide valuable perspectives on possible approaches. We thank them for their comments. In this response, we identify points in the discussion that we find particularly useful, and try to clarify some issues where there appears to be disagreement.

The comment by Jérôme Adda, Tarani Chandola, and Michael Marmot focuses on the association of health and wealth in panel data. They replicate our statistical analysis on two data sets, the Whitehall II panel in Great Britain and the Swedish Survey of Living Conditions (ULF) and conclude that these replications give similar results. This is extremely valuable, providing a powerful cross-population/cross-institutions test for model invariance. However, their general assessment that our model is transferable to these data sets may be overly generous; a detailed comparison reveals some significant differences whose exploration would be a good starting point for further research. Their finding that mental diseases in the British and Swedish data also fail the test for no direct causality suggests strongly that the sources of this rejection are behavioral factors, rather than our proposal of a possible gradient in affordability of preventative mental health services within Medicare. James Poterba points out that a detailed look at disease-specific therapies and Medicare reimbursement rules may permit a sharper test for a causal link from affordability of preventative care to health outcomes, and changes in medical insurance coverage over

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time or with recipient age may induce invariance failures unless they are accounted for explicitly.

Our discussants include central contributors to three major "schools" of causal analysis: time-series prediction criteria, or *G-causality,* grounded in the empirical tests proposed by Granger and Sims (Clive Granger, John Geweke); the structural or functional approach grounded in econometric simultaneous equations models (Jerry Hausman, Kevin Hoover); and the potential outcomes or counterfactual approach grounded in the statistical analysis of experimental treatments (Jean-Pierre Florens, James Heckman, Fabrizia Mealli, Donald Rubin, and James Robins). As a shorthand, we will refer to the structural approach as *S-causality* and the prospective outcomes approach as *P-causality.* These schools differ substantially in terminology, perspective, and prescription for applications. Nevertheless, there are strong links between them. Pearl (2000, chapter 7) demonstrates a formal equivalence of S-causality and P-causality, and Heckman's comment demonstrates the utility of interweaving the S and P formulations. Both the S and P schools are critical of G-causality, arguing that its rather sparse characterization of causal properties is not sufficient to predict the effect of interventions. In the testing scheme we adopt in our paper, we start from G-causality and add invariance tests as a way to addressing this problem. The questions then are whether our invariance requirements are consistent with the more complete S or P specifications for causal modeling; whether they are sufficient for the limited causality claims that we make; and, more broadly, whether on the road to a complete causal analysis our approach is a helpful way station or a dangerous diversion.

In linear econometrics, it is common to conduct exogeneity tests for model specification, which can be interpreted as invariance tests for model coefficients when instrumental variables are used, and conditional on acceptance of exogeneity tests, to use simple exclusion tests for the existence of direct effects of explanatory variables. Features of this setup are that the alternatives to exogeneity are vague, an exogeneity test may be rejected for a variety of model misspecification reasons, and the procedure may have zero power against some alternatives in which direct exogeneity failures are confounded by other model failures. Nevertheless, the procedure is a useful diagnostic whose robustness weighs against its lack of optimality against specifically focused alternative structures. The scheme in our paper for testing the absence of direct causal paths has a similar structure, and we argue that it has similar properties.

First, a universally valid causal model in the S or P framework will predict successfully given any history, defined broadly to include any changes in geographical or temporal frame and any policy interventions. Each successful prediction constitutes, in our terminology, a model invariance. When one requires only that a more restricted class of invariance conditions be met, there will be a *family* of S or P models that are not necessar-

ily universally valid, but which are valid and observationally equivalent for the specified class; see Florens and Heckman (2001). For example, the two directed acyclic graphs (DAGs) in in figure 11.9, only one of which has a direct causal path from S_{t-1} to H_t, may nevertheless both be valid for a class of invariance conditions that does not include interventions in which the conditional distribution of S_{t-1} given H_{t-1} changes. The fact that a broader class of invariance conditions could distinguish these models is not necessarily important if one is interested only in policy interventions in which the conditional distribution of S_{t-1} given H_{t-1} is itself always invariant. James Heckman makes a related, and even more important point, stating that "Evidence for invariance with respect to one class of manipulations does not necessarily carry over to other classes of manipulations" (2003, 77). The class of invariances tested should be precisely those needed for a targeted policy intervention, and these may or may not include transferability of the model across time periods or locations.

An empirical rejection of a model invariance is evidence against the validity of the family of S or P models that imply this invariance. Our proposed invariance tests are nothing more than the empirical counterpart of the logical relationships common to families of DAGs defining families of S or P models. We claim that our setup, with suitable articulation, thus provides a language for characterizing the empirical implications of causal analysis in an S or P framework. We strongly support the suggestion that DAGs and a full S or P analysis be used to map out the invariances that need to be satisfied by a valid model for predicting the effects of policy interventions in an application. However, we think there is methodological merit in developing "bottom up" approaches to causality that seek the broadest families of full causal models consistent with a particular policy application, to compliment "top down" approaches that start from full causal models embodying all prior information, and we then map out the policy applications for which they are valid. We recognize that the invariance test we actually conduct, for invariance of selected Markov transition probability parameters, falls far short of the battery of tests necessary to exhaust the empirical implications of a full causal model, or the relevant invariances for many policy applications, and note further that there are classes of causal models in which stationarity is not required.

Second, we claim in our paper that if our invariance and no direct causality (hereafter, NDC) tests are accepted, this is indeed evidence against the existence of a direct causal path. There are obvious limits to our claim in finite samples, particularly given the dimensions of invariance that remain untested. In light of the comments and the last paragraph, a more precise, and limited, statement is that acceptance of these tests is evidence against the existence of a direct causal path that is *active* (or, in genetic terms, *expressed*) within the class of invariances under consideration. In addition, there are an abundance of possible sources of model misspecifi-

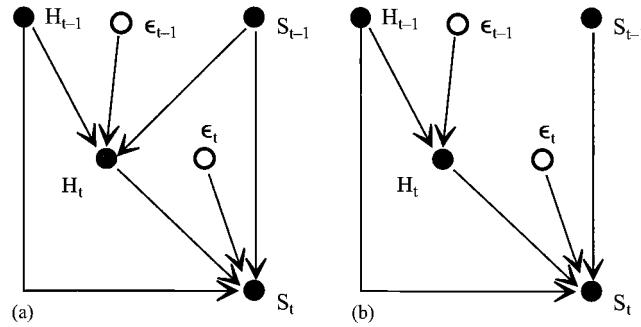

Fig. 11.9 Direct acyclic graph

cation that may confound our analysis and limit its power, particularly its linear single-index structure, untested treatment of "instantaneous causality," and first-order Markov dynamics. The power of the tests against plausible alternatives appears to be low; the observation that simulation using the models "as is" generates strong wealth–health links despite the acceptance of our tests for most conditions suggests this to be the case.

Several discussants, and also David Bloom in a personal communication, claim that our setup is statistically inconsistent so that there are circumstances where NDC will be accepted even when a direct causal link is present in a valid fully specified causal model. Kevin Hoover states that "A finding of invariance in the conditional model does not indicate a true causal relationship *unless* there is simultaneously a failure of invariance in the marginal model for the conditioning variable" (2003, 124). This is a succinct way of stating that our tests have power only against active direct causal links. In particular, we agree that one can devise examples where a direct causal effect is exactly offset by an unobserved common factor, or an ignored common factor exhibits no variation across populations that allow its effect to be identified, and our tests will have zero power against these alternatives, but we believe it improbable that these will occur and go undetected when the class of invariance tests is sufficiently broad. Speaking loosely, in the space of full causal models we expect manifolds on which effects are unvarying or exactly offsetting and thus not separately identifiable to have a priori probability zero, and if alternatively the effects can be identified under some circumstance, say by comparing populations with different initial conditions, then there will be a corresponding invariance test that should be rejected.

Finally, consider the broad question of whether our testing setup is a useful way station or a distraction. There are some legitimate concerns. Our testing procedure is generally insufficient for a full S or P causal analysis. Our treatment can be criticized as limited in applicability, because it is silent in the likely circumstance that direct causal effects are present but are

confounded by unobserved common factors, and we offer no way of describing and identifying direct causal effects when they are in fact present. We agree with the discussants' suggestion that drawing conclusions on causality within an incomplete framework for causal modeling invites mischief. We have ourselves used our model in a simulation mode with hypothetical policy alternatives, with an untested assumption that "instantaneous causality" has a specific causal chain structure, and it is somewhat disingenious for us to argue that the results can be used only for model evaluation, not for policy analysis. On the positive side, we believe that for policy applications, framing causality analysis in terms of the minimal conditions needed to support evaluation of specific policy alternatives is a useful counterpoint to a full S or P analysis, and for this reason it is scientifically useful to provide "pullouts" on the road to causal modeling.

We conclude with a few specific responses to points raised by individual discussants. First, Jerry Hausman notes that if the first-order Markov assumption for our specified state vector is not satisfied, then our estimation procedure is not statistically consistent. We agree; a test for this assumption will be practical when the panel is a little longer, and is important to do. He observes correctly that our invariance tests will have low power if there is limited variability in explanatory variables. For this reason, the transferability invariance tested by Adda, Chandola, and Marmot is particularly helpful.

Several discussants, including John Geweke, Jean-Pierre Florens, and James Heckman, observe that our treatment of "instantaneous causality" is untested and possibly disputable. The order we have selected for health conditions is based, roughly, on the etiology of the various diseases, but we agree that this is a potential source of serious model misspecification. If our hypothesized causal chain is not valid, then instantaneous causality induces a simultaneous equations problem that causes our model estimates to be statistically inconsistent. We have emphasized time aggregation between panel waves as a source of simultaneous causality, but agree that across some health conditions there could be true mutual instantaneous causality, perhaps in the form of the multivariate jump processes discussed by Florens. If our hypothesized causal chain is incorrect, then this should be detectable via a relatively straightforward invariance test using a joint probit structure for the various health conditions. It is possible to conduct such a test in a LM form that does not require estimation of the joint probit model.

John Geweke comments that portfolio management under life-cycle consumer theory may produce discontinuous shifts in portfolio mix, as well as discrete changes in measured wealth due to transfers and tax-motivated reorganizations. In our models for wealth component changes, this would produce outliers that are linked across asset categories. This may explain some of the noise we observe in wealth components. A good

solution will have to await further research. We note, however, that outliers in total wealth are sufficiently large and frequent to suggest that much of the noise we see is measurement error rather than true variation in the underlying economic variables.

The AHEAD study has notified users that incidence of new heart conditions in wave 3 was not collected and coded properly for subjects who had no previous history of a heart condition. In our working sample, the recorded 310 wave 3 incidences are then an undercount, and we estimate that approximately 122 additional incidences were not recorded. Because the intercepts for wave 2 to wave 3 incidence of heart conditions were allowed to vary freely in our models, this data problem will not affect our substantive conclusions if the undercount occurs at random, but will have a direct impact on our models for incidence of heart conditions between waves 2 and 3, and on the invariance tests based on comparing these transitions to the extent that the undercount does not occur at random. Because remaining models for health condition incidence and wealth change are conditioned on heart condition incidence, any direct impact of this data problem will have some effect on all the models in our paper.

In response to reviewer comments, we suggest that it would be possible to conduct a LM test for correlation of disturbances in our chain of incidence models. We have now calculated these tests and accept the hypothesis of no correlation of these disturbances in the equations for various health conditions. We note, however, that these tests are likely to have low power.