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Abstract

A positive relationship between socio-economic status (SES) and health, the so-called "health-wealth gradient", is repeatedly found in many industrialized countries. This study analyzes competing explanations for this gradient: causal effects from health to wealth (health causation) and causal effects from wealth to health (wealth or social causation). Using six biennial waves of couples aged 51-61 in 1992 from the U.S. Health and Retirement Study, we test for causality in panel data models incorporating unobserved heterogeneity and rich dynamics. In contrast to tests relying on models with only first order dynamics or without unobserved heterogeneity, these tests do not provide any evidence of wealth-health causality. On the other hand, we find strong evidence of causal effects from both spouses' health on household wealth.

JEL Codes: C33, D31, I12, J14.
Keywords: health, inequality, aging, dynamic panel data models, causality.

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1 Introduction

One of the stylized facts in many industrialized countries is the positive association between health and wealth at the micro level, the "health-wealth gradient." Explaining this gradient has been a challenge for economists and other social scientists. Three types of explanations exist: causal effects from health to wealth, causal effects from wealth to health, and unobserved common factors that drive health and wealth in similar ways. It is important to distinguish these explanations to understand the sources of health inequalities and to design economic policy to improve welfare, health, and well-being.

Many empirical studies have studied the importance of causal effects from health to wealth and wealth to health, often using longitudinal data. The contribution of this paper is that dynamic linear panel data techniques are used to analyze these causal effects, allowing for unobserved heterogeneity and for a rich dynamic model structure that is supported by the data. For this purpose, we use six waves of the Health and Retirement Study (HRS), covering the period 1992 - 2002 of a representative sample from the US cohort born between 1931 and 1941.

The main innovations are as follows. First, our models have a richer structure than existing models. In particular, even though we work in first differences, our model is richer than existing models in shocks, since in our framework, changes also depend on lagged changes in health. Our dynamic panel data model not only allows for unobserved heterogeneity, but also for a more complete dynamic structure. A new finding compared to the existing literature is that the latter is quite important - specification tests demonstrate that two lags are needed to fit the data, and models with only one lag indeed lead to different conclusions.

We explore the sensitivity of the test results to different sets of assumptions, particularly concerning the nature of the dynamic feedback allowed for. Whereas existing studies use multiple data sources as well as many different specifications, an important methodological contribution of our study is that we show that for a given data set, conclusions depend crucially on the specification (i.e., incorporating rich dynamics and unobserved heterogeneity).

Our main finding concerns the causal effects of wealth on health. Existing studies often find causal effects of household wealth on indexes of health. We show that this result disappears if an appropriate dynamic structure and unobserved heterogeneity are allowed for. In our preferred model specification sustained by specification tests, we find a strong effect of health on wealth, but no effect of wealth on health.

Our model considers couples, looking at household wealth and at health of both spouses. It thus also incorporates several explanations why health indexes of the two spouses in a couple are correlated, an issue which, to the best of our knowledge, is not addressed in a panel data context in the existing literature. We find no evidence of a causal effect of health of one spouse on health of the other spouse.

The paper is organized as follows. In Section 2, we discuss the health-wealth gradient and its potential sources, explain why it is important to understand these, and discuss the existing studies on causal effects from health to wealth and wealth to health. In

section 3 we describe the association between wealth and health and the way it evolves over time in our data. In section 4, the econometric framework is presented, with the identification, testing, and estimation strategies. Section 5 presents the results for the dynamic panel data models. Section 6 compares these results with those of simpler models. Section 7 concludes.

2 The Health-Wealth Gradient and Its Sources

In the United States, respondents of the 1984 wave of the Panel Survey of Income Dynamics (PSID) who reported to be in excellent health had almost 75% higher median wealth than those who reported fair or poor health (Smith, 1999). Ten years later the ratio between median wealth of the same groups had grown to 274%. The ratio in 1984 was largest for the age group 45-54, 176%, which increased to 264% in 1994. Although often less pronounced than in the U.S., a similar relation between socioeconomic status (SES) and health (the "health-SES gradient") is found in most industrialized countries with similar levels of health care technology and economic welfare (Wilkinson, 1996).

Using data from the PSID, Deaton and Paxson (1998) show that the correlation between income and self-reported health increases over the life-cycle until about age 60, while the variance in self-reported health outcomes increases monotonically over the complete life-cycle. Adda (2003) finds similar results for Sweden. In the United Kingdom, one of the puzzles created by the widely cited Whitehall I (1967) and II (1985-1988) studies looking at the health of civil servants over three decades (Marmot, 1999), is that among these individuals of similar socioeconomic status, the health-SES gradient, which was already substantial in 1967, has further increased over time, despite rising real median wealth and increasing efforts to facilitate access to health care (Smith, 1999).

Understanding the sources of the health - wealth gradient is important in order to understand the sources of health inequalities and to design economic policy to improve welfare, health, and well-being. Curbing health inequalities may be desirable for many reasons. Deaton and Paxson (1998) argue that a mean-preserving spread in the health distribution leads to increasing mortality and reduced welfare under the plausible assumption that the marginal effect of health changes on mortality is higher at the lower end of the health distribution, where individuals are more fragile and exposed to risks. Pradhan et al. (2003) argue that a social welfare function should have health as an argument and should be concave in that argument if poor health is a stronger sign of deprivation of capabilities than low income. In that case health becomes intrinsically important as opposed to instrumentally significant.

Another reason why the gradient is important, is the relation between health, retirement, and incentives of social security benefits and health insurance. Better health is positively associated with household savings, labor force participation, and earnings, and negatively with the old age social security benefits replacement rate. Availability of Medicare at age 65 may explain the retirement peak in the U.S. at that age, where social

security incentives no longer apply (Rust and Phelan, 1997). Since the importance of public health insurance depends on health as well as SES, the health-SES relations are relevant for the debate on universal health care and the efficiency of proposed reforms.

Attempts to understand the different causal effects ("pathways") through which socioeconomic status and health affect each other have been numerous (see Smith, 1999 and Adler et al., 1994 for reviews). Causal pathways from health to wealth have been emphasized by economists, relying on the human capital theory by Grossman (1972), where health is seen as a stock that is built up through investment. Health is worth investing in since it yields utility: it extends life and thus the horizon over which gains from productivity can be used for consumption, and provides utility through consumption of leisure on healthy days. At a given point of the individual's life-cycle, the health stock is the result of investments and shocks from the individual's past, implying that over the life-cycle, health is more and more driven by health events from the past.

Causal effects from health to wealth (also referred to as health causation)[1] can be explained in this framework. Health and health expectations can affect productivity, hourly wages, and labor supply at the intensive and extensive margin. Health thus drives the capacity to accumulate savings for retirement, and affects retirement decisions both in this way and through the direct effect of health on the marginal rate of substitution between leisure and work. Moreover, health directly affects expenditures, particularly in the U.S. where about 20% of workers below 65 are not covered by health insurance, and where those who are covered often face copayments or other additional expenditures. Consequently, health events can lead to considerable revisions of saving plans or, for example, expected bequests (Smith, 2003).

Pathways from wealth or, more generally, from socioeconomic status to health have been studied extensively in other social sciences (Adler et al., 1994) and since recently also in economics (Adams et al., 2003; Adda, 2003; Hurd and Kapteyn, 2003; Meer et al., 2003; Smith, 2003). This causal link, often named *social causation* in other social sciences, will be referred to as SES or wealth causation, the opposite of health causation.

Theories explaining such pathways have been put forward in several fields, such as biology, psychology, and economics. One factor is *access to health care*. If not all people are covered by health insurance or if there are copayments or deductibles, those with low income or wealth will consume less health care services (in quantitative or qualitative terms) and thus invest less in their health. This cannot explain, however, why in the U.K. the wealth-health gradient has increased over a period in which general access to health care has increased, as shown by the two Whitehall studies referred to above. Moreover, it is hard to reconcile this explanation with the evidence provided by the RAND health experiment (Newhouse, 1993), which, in a set-up with randomly assigned copayment rates, showed that those with lower copayment rates on average used more health care services but did not have significantly different health outcomes. The variation in quality of care and treatments across socioeconomic groups may be even more important than access to health care services per se. Indeed, the Grossman health production model implies that the marginal benefits of investment in health care can rise with education

[1] This is often referred to as *health selection* in the social science literature.

level (an indicator of SES), explaining why the demand for health care quality increases with SES. Still, Kenkel (1991) finds that only part of the relationship between schooling and health is explained by differences in health knowledge.

A second mechanism is *risk behaviors*: low SES leads to behavior like smoking and drinking that is detrimental to health (Marmot, 1999). A third potential causal effect of wealth on health through *wealth inequalities* comes from the stress associated with being at the bottom of the distribution. Marmot (1999) finds that civil servants in higher ranks have lower level of cholesterol than those in lower ranks, suggesting that a low wealth position may create additional stress. Wilkinson (1996) finds that countries with higher wealth inequality have higher mortality rates. In contrast, Deaton (2003) finds that the evidence of a causal effect of wealth inequalities on health in more developed countries is weak. A way to think of the effect of stress is to consider the adaptation of the health system to a series of stressful events. The immune system can adapt by functioning at a more intensive level, which may in the long run be detrimental to blood pressure and the health system. Episodes of stress such as the loss of a job may then in the long run lead to higher incidence of cardiovascular disease or high blood pressure. Since the frequency of stressful events differs across SES groups, the amount of *allostatic loads*, a measure of the cumulative effect of stressful events on the health system (e.g., Seeman et al., 1997), will vary with SES.

A final set of explanations of the health-wealth gradient refers to *early childhood*. Small health events at the beginning of life may affect an individual's complete health trajectory over the life-cycle (Barker, 1997). Following a sample of the March-1946 birth cohort in the UK over nearly 50 years, Wadsworth and Kuh (1997) found that early childhood events such as poor living conditions significantly predicted many diseases later in life. Moreover, they showed that children of age two from this 1946 cohort had a higher risk of developing bronchitis if their parents had a similar childhood condition or smoked as adults, implying that health is partly transmitted from the previous generation. Lindeboom et al. (2006) found that macroeconomic conditions at birth affected mortality hazards of cohorts throughout the 19th and 20th century. Ravelli et al. (1998) showed that children born during the 1944-45 famine in Amsterdam were more likely to develop diabetes later in life. These examples show that health is partly determined by health of the parents or health in early childhood, which will be related to the parents' SES. Since there is also a strong intergenerational effect of SES, this can explain part of the health-SES gradient later in life. In our analysis of people aged 50 and over, such effects arise as permanent health shifts throughout the observation window. We will model them as individual specific health effects reflecting *unobserved heterogeneity*. Similar unobserved heterogeneity terms may drive household wealth, and the unobserved heterogeneity in household wealth and in health of both spouses can be correlated.

The goal of many studies including this paper is to disentangle these sources of the health-wealth gradient and to determine the importance of causal effects in both directions. Longitudinal data with extensive information on wealth and health offer a non-experimental setting in which causality can be addressed using the time series

concept *non-causality* (Granger, 1969). Adams et al. (2003) used Granger causality to study the health-wealth gradient, testing for causal effects of wealth on health and health on wealth in the AHEAD cohort of age 70 and older in the US. They used only three waves, limiting the richness of the dynamic specifications they could use. Moreover, they control for a large set of observed covariates, but not for unobserved heterogeneity. Their results provide clear evidence of health causation but they also find evidence of wealth/SES causation. They point out that rejecting their hypothesis of no Granger causality could also be an indication of correlated unobserved heterogeneity in health and wealth. Adda (2003) uses Swedish panel data for individuals over the whole life-cycle and implements a test for health and SES causation. He concludes that both health wealth and wealth health causation mechanisms are present. He does not address the issue of unobserved heterogeneity.

On the other hand, Smith (2003) and Wu (2003) perform tests of health causation conditional on initial conditions. Since the initial values are correlated to the unobservable heterogeneity terms, this goes in the direction of controlling for unobserved heterogeneity. They estimate the impact of onsets of critical health conditions such as cancer or lung disease on changes in wealth and other SES indicators, conditioning on initial health status. Smith (2003) looks at changes between the first and the fifth wave of the HRS, while Wu (2003) looks at changes over the first two waves. Using onsets as exogenous health shocks that are not affected by wealth changes seems a plausible identification strategy. Smith (2003) estimates that the cumulative effect of the onset of a critical disease after eight years is about $40,000, and Wu (2003) finds that household wealth responds more strongly to the onset of a serious condition for the wife than for the husband. Neither Smith (2003) nor Wu (2003) exploit the full panel nature of the HRS or explore the dynamics of health and SES causation.

Using a similar strategy to test for causal wealth-health effects, Meer et al. (2003) use three 5-year spaced observations from the PSID, using bequests as instruments that directly affect wealth but not health. Their test looks at the effect of wealth changes on self-reported health. The dynamics of their model imply that wealth changes have an immediate effect on health after which health returns to a stationary value. They find small and insignificant wealth-health effects. Adams et al. (2003) reject the hypothesis that wealth changes do not cause health changes for three of the four main causes of death among older men, as well as for mental health and self-reported general health. Similar results are found by Adda et al. (2003) for the U.K. and Sweden. Hurd and Kapteyn (2003) find that changes in health are more related to income in the U.S. than in the Netherlands. In these studies, non-causality is tested without controlling for unobserved heterogeneity. Hausman (2003) as well as Mealli and Rubin (2003) argue that this may bias the estimates and the test results, possibly explaining why the null of no causality is often rejected.

In this paper, we develop dynamic panel data models that make it possible to test for health and wealth causation, controlling for unobserved heterogeneity, using the econometric framework of Arellano and Bond (1991). Alonso-Borrego and Arellano (1999) emphasize that dynamic vector autoregressive panel data models offer a rich

environment for performing such tests. We apply the models to the HRS cohort of couples with at least one spouse born between 1931 and 1941 who are observed over six biennial waves from 1992 to 2002. We consider health for each spouse and wealth at the household level, as Wu (2003). We consider two types of models - models without instantaneous effects of wealth on health and vice versa, in which no instruments are needed, and structural models with instantaneous links between health and wealth. In the latter models, we use the instruments of Smith (2003), Wu (2003) and Meer et al. (2003). In addition, we impose mean stationarity to increase efficiency of the estimates, following Blundell and Bond (1998).

3 Wealth and Health in the HRS cohort

The Health and Retirement Study is a longitudinal survey of individuals aged 51-61 in 1992 in the United States, funded by the National Institute on Aging and other partners such as the Social Security Administration. Data were collected every two years and cover a wide range of aspects of the life of the 50+ population. For the first wave of 1992, 12,652 interviews were conducted for a random sample of individuals born 1931-1941. Spouses of these individuals were also included in the sample, irrespective of their age.

We use the public release file from the RAND corporation that merged records from the six available waves (1992-2002).[2] We selected all couples in the 1992 wave with complete information on the relevant variables. We retained observations with missing or bracket information on one or more components of wealth, using imputed values (see below). We observe couples until one spouse dies, until divorce or separation, or until at least one member of the household could not (or refused to) be interviewed. We do not analyze widows and widowers or divorced or separated spouses, since our models focus on the relation between wealth and health of both spouses.

In 1992, there are 4,160 households, of which 2,463 remain until 2002. The average attrition rate for each wave is about 10%, implying an annual attrition rate of about 5%.[3] See Appendix A for sample size, attrition rates, and some descriptive statistics. In the sample, wives are on average four years younger than their husbands. Both spouses have a similar average level of education. About 6% of respondents are Hispanic and 8% are black. About 8% of husbands and 10% of wives are immigrants. One out of four respondents has been married at least once prior to their current relationship. Those who exit before the end of the panel are on average older, because survival probabilities fall with age. Attritors have slightly less education than respondents who remain in the panel for all six waves, while Blacks and Hispanics are more likely to exit than others.

[2] See http://www.rand.org/labor/aging/dataprod/.

[3] From life-table figures, yearly death rates for this cohort vary from 0.5% to 2.6% over the decade considered (Berkeley Mortality Database: http://www.demog.berkeley.edu/wilmoth/mortality/).

3.1 Wealth Data

We use two broad wealth categories: liquid and non-liquid. Liquid wealth consists of individual retirement accounts, stocks, bonds, certificate deposits, T-bills/saving bonds, checking/saving accounts and other savings, net of financial debts (excluding mortgages). Non-liquid wealth includes the net value of the primary residence, other real estate, and vehicles.[4] Wealth includes the value of life insurances and other annuities (in "other savings") but not the value of defined contribution pension plans. All amounts are expressed in 2002 U.S. dollars using the Consumer Price Index of the Bureau of Labor Statistics. We will use log transformed wealth to reduce the effect of outliers.[5]

Table 1 describes the sample composition of wealth.[6] It gives the median of each component conditional on ownership (and holding a positive amount) and the ownership rates for the 1992 and 2002 waves. In 1992, respondents held more than two thirds of their wealth in non-liquid assets, mainly the primary residence. The share of non-liquid assets in total wealth falls over the decade. Participation in stocks and individual retirement accounts (IRAs) is much more important in the U.S. than in many other countries (Hurd, 2001). More than half of the respondents own IRAs, with a median value of $31,570 in 1992. Moreover, by 2002, 37.4% of households held stocks, with a median value among holders of $50,000. The median value of stocks and IRAs more than doubled over the 10 years, partly reflecting the high returns on these holdings throughout this period.

[Table 1 here]

3.2 Health Variables

Table 2 summarizes the health information for the 1992 and 2002 waves. In 1992, 16.7% (23.8%) of wives (husbands) had suffered from a condition that Smith (2003) labels as severe: cancer, heart condition, lung disease or a stroke (or a combination of these). More than half of the respondents had ever had an onset of a mild condition - diabetes, high blood pressure, arthritis, or depression. Thus many respondents have faced considerable health shocks before 1992, suggesting that much of the association between health and wealth may stem from before they were observed in the HRS.

By the end of 2002, 44.9% of husbands and 31.7% of wives had reported the onset of a severe health condition, implying that between 1992 and 2002, about one in every five respondents experienced their first severe health condition. In 2002, 81.3% of husbands

[4] Unlike Adams et al. (2003), we do not include business assets. These are nonzero for few respondents but vary enormously over time for some respondents.

[5] To deal with zero wealth (0.5-1% of the observations per wave) and negative wealth (2-3% of the observations per wave), we use the following log transformation: $\log(y) = 1(y \geq 0)\log(1+y) - (1-1(y \geq 0))(1-\log(-y))$; for positive values, this is virtually identical to log wealth.

[6] Where necessary, imputed values are used, often based upon bracket information on missing amounts. The percentage of imputed values varies from 3.4 (bonds) to 21.6 (IRAs). The imputations are provided in the RAND version of the HRS data; the procedure is described in Hoynes et al. (1998).

(79.9% of wives) had experienced the onset of a mild health condition, mostly arthritis or high blood pressure. Emotional and psychological health problems are much more frequent for wives than for husbands, in line with the differences in *CESD scores* for mental health.[7] The Body-Mass Index (BMI) increased more over time for wives than for husbands. The percentage having difficulties with activities of daily living (ADL) also increased over time and was always larger for wives than for husbands.

Indicators like self-reported health convey more general information about health than indicators for specific onsets, CESD scores, etc. Adams et al. (2003) consider general and specific health variables separately; Hurd and Kapteyn (2003) consider self-reported health status and Smith (2003) studies onsets of serious health conditions. In our panel data models, we combine health variables into a one-dimensional health indicator, following Adda (2003). A "constructed health index" (CHI) is built from the indicators presented in Table 2 using principal component analysis to combine them (see Appendix B for details on construction of the index). The index is normalized such that it has mean 0 and variance 1. Low values of the index refer to good health and high values to bad health. Most health variables contribute substantially to the CHI, with the highest score for self-reported health. To check the robustness of our findings, the empirical analysis will also considers some alternative health measures, e.g., not including self-reported health.

[Table 2 here]

Table 3 presents the bivariate distribution of the husband's and the wife's CHI in 1992, showing that health of husband and wife are correlated. For example, 38% of wives with husbands in the best health quartile are in the best health quartile themselves, compared to only 16% of wives with husbands in the worst health quartile. A chi-square test confirms that CHI-s of both spouses are not independent (p-value < 0.001). This can be due to causal mechanisms (e.g. stress due to a health problem of the spouse), assortative matching, or common factors affecting both spouses' health in the same way (e.g. environment, socio-economic position, risk behavior).

[Table 3 here]

3.3 Association between Wealth and Health

Table 4 reveals the health-wealth gradient in the 1992 and 2002 waves (cf. Smith, 1999, Table 1). It presents median household wealth by 1992 health quartile (using CHI as the health measure). In 1992, median household wealth of husbands in the best health quartile was more than twice as high as median household wealth of husbands in the

[7] This score is based on the Center for Epidemiologic Studies Depression (CESD) scale. It gives the sum of six negative yes/no indicators for mental health minus two positive indicators. The negative indicators say whether the respondent experienced the following sentiments all or most of the time: depression, everything is an effort, sleep is restless, felt alone, felt sad, and could not get going. The positive indicators are whether the respondent felt happy and enjoyed life, all or most of the time.

worst health quartile. The same health differential is found between health quartiles of wives in 1992. The wealth differential increased further in 2002. These differences are of similar magnitude as those found by Hurd and Kapteyn (2003) and Smith (1999), who used self-reported general health instead of CHI. The large differences do not only appear in the tails of the distribution: even among the households with relatively healthy wives in the second quartile of the husbands' CHI distribution in 1992, median wealth is 16.2 to 26.5% lower than in the top health quartile.

[Table 4 here]

4 Models and Tests

4.1 Model for the Evolution of Health and Wealth

We develop a model for three outcome variables for a given couple i in year t: $\mathbf{Y}_{it} = (h_{it}^m, h_{it}^f, y_{it})'$, where h_{it}^m is health of the husband, h_{it}^f is health of the wife, and y_{it} is log household wealth. As explained in section 1 and emphasized by, e.g., Hausman (2003), a model explaining the evolution of wealth and health should have several features. First, it must capture immediate and lagged causal effects of wealth on health (wealth causation) and of health on wealth (health causation). Second, it should allow for an effect of each spouse's health on health of the other spouse, as a possible explanation for the association between CHIs of both spouses, apparent from Table 3. Moreover, the model should take into account potentially correlated unobserved heterogeneity in health and wealth, leading to a permanent correlation between wealth and health from the beginning of the observation window. We will use the following panel data vector autoregressive model of order P for $\mathbf{Y}_{it}$, capturing all these features and allowing for the various explanations of the health - wealth gradient:

$$\mathbf{\Gamma Y}_{it} = \mathbf{A x}_{it} + \sum_{p=1}^{P} \Phi_p \mathbf{Y}_{it-p} + \boldsymbol{\eta}_i + \boldsymbol{\varepsilon}_{it} \tag{1}$$

The matrices $\mathbf{\Gamma}, \mathbf{A}$ and $\mathbf{Phi}_p, p = 1, \ldots, P$ contain the parameters of the model. $\mathbf{x}_{it}$ is a vector of time invariant and time varying characteristics (education, race, age, etc.). These characteristics can be correlated with a vector of time-invariant unobserved heterogeneity terms $\boldsymbol{\eta}_i$, which, for example, capture unobserved traits at birth such as intergenerational factors, early childhood events (cf. section 1), etc. We will allow for correlation among the three components of $\boldsymbol{\eta}_i$ within a couple, as well as for correlation among the three transitory shocks in $\boldsymbol{\varepsilon}_{it}$. The matrices $\mathbf{\Phi}_1, \ldots, \mathbf{\Phi}_P$ reflect causal links that take time to become effective. The parameters on the effect of lagged wealth on health can be seen as transmission channels for wealth causation while the parameters on the effect of lagged health on wealth are indications of health causation (Adda, 2003).

Through the matrix $\mathbf{\Gamma}$, we also allow for instantaneous causality. In our case, this is particularly relevant since observations are spaced by two years, and it is unlikely

that all causal links will take two years or more to become effective. We also allow for instantaneous effects of the health of one spouse on the other spouse's health. Since our health indicators also incorporate self-reported health and subjective life expectancy, such effects also arise if respondents adjust their subjective beliefs if the health of their spouse deteriorates.

To estimate the dynamic interactions between health and wealth consistently, it is crucial to incorporate a dynamic structure that is flexible enough to fit the data. In particular, the order of autoregression P has to be large enough. Specification tests as in Arellano and Bond (1991) will be used to select a model that satisfies this.

Since individual effects can be correlated with the regressors in $\mathbf{x}_{it}$, it is not possible to estimate the influence of time-invariant regressors or to disentangle the effects of age and a common time trend. For similar reasons, we will not include variables on risk behavior (smoking, drinking, body-mass index). Persistent risk behavior over the life cycle can have a causal effect on health and also correlates negatively with socio-economic status. Most of this, however, refers to events earlier in life and will be captured by the individual effects. The variation of risk behavior over time in the age group that we consider is likely to be endogenous: people stop smoking or drinking due to health problems. Indeed, in the data, only about 1% of all individuals start smoking, while more than 17% stop smoking. Incorporating risk behavior explicitly would require instrumenting it and this is beyond the goal of the paper. Instead, it should be kept in mind that some of the causal mechanisms that we find may be due to changes in health behavior.

We first consider the reduced form model from which instantaneous causality is eliminated, explain how to estimate this model with GMM, and how to test for causal effects using a Wald test. We then turn to the structural model with instantaneous causality.

Reduced-Form Vector Autoregressions

Consider the reduced-form Vector Autoregression (VAR) of (1):

$$\mathbf{Y}_{it} = \mathbf{B}\mathbf{x}_{it} + \sum_{p=1}^{P} \mathbf{C}_p \mathbf{Y}_{it-p} + \boldsymbol{\eta}_i^* + \boldsymbol{\varepsilon}_{it}^* \tag{2}$$

where $\mathbf{B} = \boldsymbol{\Gamma}^{-1}\mathbf{A}, \mathbf{C}_p = \boldsymbol{\Gamma}^{-1}\boldsymbol{\Phi}_p$ for $p = 1, .., P$, $\boldsymbol{\eta}_i^* = \boldsymbol{\Gamma}^{-1}\boldsymbol{\eta}_i$ and $\boldsymbol{\varepsilon}_{it}^* = \boldsymbol{\Gamma}^{-1}\boldsymbol{\varepsilon}_{it}$. The null hypothesis of no causal effects from wealth to husband's health can be written as

$$H_0 : E(h_{i,t+1}^m | \mathbf{Y}_i^t, \mathbf{x}_i^t, \boldsymbol{\eta}_i^*) = E(h_{i,t+1}^m | \mathbf{h}_i^t, \mathbf{x}_i^t, \boldsymbol{\eta}_i^*) \text{ for } t = 0, ..., T \tag{3}$$

where $\mathbf{h}_{it} = (h_{it}^m, h_{it}^f)'$, $\mathbf{Y}_i^t = (\mathbf{Y}_{i0}, ..., \mathbf{Y}_{it})$, $\mathbf{h}_i^t = (\mathbf{h}_{i0}, ..., \mathbf{h}_{it})$, and $\mathbf{x}_i^t = (\mathbf{x}_{i0}, ..., \mathbf{x}_{it})$. In model (2), this takes the form

$$H_0 : \mathbf{C}_{1,my} = ... = \mathbf{C}_{P,my} = 0 \tag{4}$$

where $\mathbf{C}_{p,my}$ is the m, y element of the matrix $\mathbf{C}_p$, the effect of p-periods lagged log wealth on the husband's health. Similarly, the null hypothesis of no causal effects from wealth to the wife's health takes the form:

$$H_0 : \mathbf{C}_{1,fy} = ... = \mathbf{C}_{P,fy} = 0 \tag{5}$$

The null hypothesis of no causal effects from the husband's health to household wealth is given by

$$H_0 : E(y_{it+1}|\mathbf{Y}_i^t, \mathbf{x}_i^t, \boldsymbol{\eta}_i^*) = E(y_{it+1}|y_i^t, h_i^{ft}, \mathbf{x}_i^t, \boldsymbol{\eta}_i^*) \text{ for } t = 0, ..., T \tag{6}$$

In model (2), this takes the form

$$H_0 : \mathbf{C}_{1,ym} = ... = \mathbf{C}_{P,ym} = 0. \tag{7}$$

Similarly, the null hypothesis of no causal effects of the wife's health on household wealth takes the form

$$H_0 : \mathbf{C}_{1,yf} = ... = \mathbf{C}_{P,yf} = 0. \tag{8}$$

Chamberlain (1984) defines (3) and (6) as tests for "Granger causality conditional on unobservables." Adams et al. (2003) look at individuals (in the older AHEAD cohort) instead of couples. Their tests are conditional on a covariates $\mathbf{x}_{it}$ but not on unobserved heterogeneity $\boldsymbol{\eta}_i^*$, and they only consider first order models. As Adams et al. (2003) emphasize, rejecting their null hypothesis leads to the conclusion that y "Granger causes" $\mathbf{h}$ under the maintained hypothesis that there is no unobserved heterogeneity. They expect that this is not a major problem since they use a rich set of covariates $\mathbf{x}_{it}$.

The reduced form model (2) can be estimated using GMM, based upon moments in first differences:

$$E(\Delta\boldsymbol{\varepsilon}_{it}^*|\mathbf{Y}_i^{t-2}, \mathbf{x}_\mathbf{i}^\mathbf{t}) = 0 \text{ for } t = 2, ..., T \tag{9}$$

First-differencing gets rid of the unobserved heterogeneity terms, but also introduces (negative) correlation between $\Delta\mathbf{Y}_{it-1} = (\mathbf{Y}_{it-1} - \mathbf{Y}_{it-2})$ and $\Delta\boldsymbol{\varepsilon}_{it}^* = (\boldsymbol{\varepsilon}_{it}^* - \boldsymbol{\varepsilon}_{it-1}^*)$. This is why the history up to $t-2$, $\mathbf{Y}_i^{t-2}$, and not $\mathbf{Y}_{i,t-1}$, is used to construct moments (cf., e.g., Arellano and Bond, 1991). It implies that estimation (and testing for causal health-wealth or wealth-health effects) requires at least three observations per household.

If the health and wealth variables are close to non-stationary, then the instruments obtained from (9) may be weak since changes will be weakly correlated with past levels (see, e.g., Arellano, 2003). Blundell and Bond (1998) suggest to solve this problem by assuming mean stationarity of errors and individual effects, leading to more moments and improving the efficiency of the estimator. Following Blundell and Bond (1998) and Arellano (2003), we impose the following additional moments:

$$E(\Delta Y_{it-1}^j(\mathbf{Y}_{it} - \mathbf{B}\mathbf{x}_{it} - \sum_{p=1}^{P} \mathbf{C}_p\mathbf{Y}_{it-p})) = 0 \text{ for } t = 3, ..., T; j = 1, 2, 3 \tag{10}$$

where $Y_{it-1}^{j}, j = 1, 2, 3$ are the components of the vector $\mathbf{Y}_{it-1}$. These moments are valid under the following additional assumptions:

$$E(\boldsymbol{\varepsilon}_{it}^{*}\boldsymbol{\eta}_{i}^{*\prime}) \text{ does not depend on } t \tag{11}$$

$$\text{No serial correlation in } \boldsymbol{\varepsilon}_{it}^{*} \tag{12}$$

$$E(\Delta\mathbf{x}_{it}\boldsymbol{\eta}_{i}^{j*}) = 0, j = 1, 2, 3 \tag{13}$$

The first of these assumptions implies that heterogeneity can be related to health or wealth shocks, but only in a way that does not vary over time. The second assumption was already made above and is the basis for (9). It is justified if all correlation over time is picked up by the auto-regressive structure (the matrices $\boldsymbol{\Phi}_p, p = 1, \ldots, P$) and the unobserved heterogeneity terms. The third additional assumption (13) is innocuous in our case, since $\Delta\mathbf{x}_{it}$ only contains time dummies (with age differences linear in time and other exogenous variables invariant over time). As Blundell and Bond (1998) emphasize, imposing these mean stationarity restrictions or not is a trade-off between robustness and efficiency. Hence it is important to test the additional restrictions. We will do this using the increment in the Sargan test statistic (cf. Arellano and Bond, 1991).

Tests for Structural Vector Autoregressions

In the structural form (1), the hypothesis of non-causality implies restrictions on both the instantaneous effects in $\boldsymbol{\Gamma}$ and the lagged effects in $\boldsymbol{\Phi}_p$, similar to the restrictions in (4). For example, non-causality of wealth to husband's health implies:

$$H_0 : \boldsymbol{\Phi}_{1,my} =, ..., = \boldsymbol{\Phi}_{P,my} == 0 \tag{14}$$

and

$$H_0 : \boldsymbol{\Gamma}_{my} = 0. \tag{15}$$

Note that these restrictions are stronger than those for the reduced form, since the reduced from parameters are linear combinations of the structural form parameters that are restricted to zero under the null. Thus the test on the reduced form will not have power for some violations of non-causality in the structural form.

Without imposing additional identifying assumptions, we can estimate the reduced form parameters in (2) but not the structural parameters in $\boldsymbol{\Gamma}$ and $\boldsymbol{\Phi}_p$. Exclusion restrictions (i.e., instruments) are needed in order to identify the instantaneous causal mechanisms (cf. Hausman, 2003). Our instruments for health and wealth relate to shocks that do not have direct effects on the other outcome, following Smith (2003), Wu (2003), and Meer et al. (2003). As instruments for health changes, we use onsets of critical health conditions. Such onsets are quite frequent for our sample (Table 2).

It seems plausible that these onsets have no direct effect on wealth and thus can only affect wealth through the change in overall health that they induce. We use separate dummies for severe and mild onsets.

To instrument changes in wealth, we use inheritances. Many of the households in the sample receive inheritances from the death of a parent or sibling (approximately 5% each wave; the median inheritance is 29,000$ and the mean is 64,100$). While the death of a family member might be correlated to the level of health due to genetic background or early childhood events etc., it seems reasonable to assume that it is not directly related to current health changes, making the inheritance an appropriate instrument for wealth changes. We use two instruments here: a dummy whether or not the couple received an inheritance in the last two years, and the size of that inheritance in dollars (with value 0 if no inheritance was received).

To identify the instantaneous effect of health of one spouse on health of the other spouse, we also use the onsets of health conditions. Here we make the plausible assumption that such onsets have no direct effect on the other spouse other than through the constructed health index. We will test the overidentifying restrictions this implies.

As in the reduced form model, we also exploit the moments based upon mean stationarity of the errors and test these using the incremental Sargan test. Tests for "lagged" causality essentially remain the same as (3) and (6), except that they involve the matrices $\mathbf{\Phi}_p$ instead of $\mathbf{C}_p$. Tests for contemporaneous causation test whether elements of $\mathbf{\Gamma}$ are zero.

5 Results

To estimate the models we used the generalized method of moments (GMM; cf., e.g., Arellano and Bond, 1991).[8] The additional mean stationarity moments in levels (see Section 3) were not rejected by incremental Sargan tests.[9] We include time dummies to pick-up unobserved trends and, where necessary as indicated by specification tests rejecting invariance of coefficients over time, we also include interactions with a time trend.

We estimate the three equations separately. We experimented with several lag structures and found that specifications with two lags (for wealth dynamics) and three lags (for health dynamics) were needed according to the usual specification tests (the Sargan test on overidentifying restrictions and the test on second order autocorrelation in the differenced residuals; see Arellano and Bond, 1991).[10] The results for the selected

[8] Since the cross-sectional dimension is quite large, we are not concerned about finite sample biases of two-step GMM, and use two-step GMM estimates constructing the optimal weighting matrix from first-step estimates.

[9] The test statistics are 13.87 (p=0.459), 4.99 (p=0.892) and 10.47 (p=0.401) in the wealth, husband's health and wife's health equation respectively.

[10] See Michaud and van Soest (2004) for more details of these specification checks. With one lag, the Sargan test statistic of the reduced-form model is 58.21 for the wealth equation. With two lags, it is 49.2 and 54.4 in the husband and wife's health equation respectively. Therefore, these specification are

models are presented in Tables 5, 6 and 7. Each table presents a reduced form equation without instantaneous effects of wealth on health etc., and a structural form equation in which the instruments proposed in Section 3 are used for identification.

5.1 Effects of Health on Wealth

Table 5 presents the results for equations explaining log household wealth. For the selected models, overidentifying restrictions are rejected at the 5% level but not at the 4% level. There is no evidence of second order serial correlation in the differenced errors, supporting the hypothesis that the errors in levels are uncorrelated over time. The reduced form estimates imply significant negative effects of health of both spouses on log wealth. Joint tests indicate that lagged values of husband's health significantly affect log wealth, so that the hypothesis that husband's health does not cause wealth is rejected. The same result is found for the wife's health.

[Table 5 here]

The structural estimates provide no evidence for an immediate effect of husband's health on wealth, and the effects of the lagged husband's health indicator are similar to those in the reduced form equation. The joint significance of all husband's health variables remains. Current and lagged CHI of the wife are also jointly significant at any reasonable significance level, but here, the immediate negative effect dominates the lagged effects. Thus, overall, we find strong evidence of causal effects of both spouses' health on household wealth. Moreover, the results of the structural model suggest differences in the time lags with which husband's and wife's health changes affect household wealth, with an instantaneous effect for wives and a lagged effect for husbands. This may explain the difference with Wu (2003), who uses only two waves of the HRS and finds that the wealth of households tends to respond more to health events of the wife than to health events of the husband. A longer time span is needed to find the effect of the husband's health.

5.2 Effects on Health of Wealth and Spouse's Health

The results for the equation explaining the husband's health are presented in Table 6. Adding the second order lags and the interaction of lagged health with time was necessary to obtain a model that passes the tests on overidentifying restrictions and autocorrelation in the errors. The results provide no evidence whatsoever of an effect of wealth on husband's health. Both in the reduced form and in the structural equation, the wealth variables are jointly (and individually) insignificant. Moreover, we find no evidence of a causal effect of the wife's health on the husband's health: in both the reduced form and the structural form equation, the wife's health variables are insignificant. Similarly, Table 7 presents the results for the equations explaining the wife's health. They provide

strongly rejected.

no evidence of causal effects from household wealth on the wife's health or from the husband's health on the wife's health.

[Tables 6 and 7 here]

6 Unobserved Heterogeneity, Lag Structure, and Other Sensitivity Checks

In this section we analyze the role of unobserved heterogeneity and whether the complete dynamic panel model leads to different conclusions than simpler alternative models. First, we consider the model of the previous section and investigate the nature of the unobserved heterogeneity terms. Second, we compare the results of the complete model with those of simpler models. Third, we consider alternative definitions of the health and wealth variables.

6.1 Unobserved Heterogeneity

The GMM residuals in each of the three equations are estimates of the sum of the error term and the unobserved heterogeneity term. Taking individual means over time gives an estimate of the unobserved heterogeneity term only. These estimates can be used to estimate the covariance matrix of the unobserved heterogeneity terms, accounting for the small number of time periods (3) used in estimation. Table 8 presents the estimated covariance matrix. For the wealth equation the unobserved heterogeneity term explains a substantial share of the total unexplained variance (1.119, about 82% of the total unexplained variance, 1.373). For the husband's and wife's health equations, the estimated shares are much smaller – 8.9% and 6.9%, respectively. The correlations between the individual effects are also small.

[Table 8 here]

Table 9 shows how the estimated individual effects (i.e., the household specific time means of the GMM residuals) in the three equations correlate with background variables measured at wave 1. The relations are mostly as expected - keeping other background variables constant, fixed effects in the wealth equation are higher for husbands with more education, for households where both spouses are white compared to households with at least one black or Hispanic spouse, and for households where the wife is more active in financial planning. We find no relation with health behavior or survival status of parents in the wealth equation. For the fixed effects in the health equations, we find that health behaviors (both obesity and smoking) play a significant role. For wives, a negative correlation between the number of living parents and bad health is found as well as a positive correlation between manual occupation and health.

[Table 9 here]

6.2 Models with no Unobserved Heterogeneity or Fewer Lags

We estimated some models that do not allow for unobserved heterogeneity and are more similar to the models analyzed by others. Tests for the hypothesis that wealth does not cause husband's and wife's health are presented in Table 10, based upon models that explain health of husband and wife from lagged health of husband and wife, lagged log wealth, and additional controls (demographics and past risk behavior). To increase comparability with Adams et al. (2003), we not only do this for the constructed health index (CHI, first column) but also for the separate health variables that were used to construct the CHI. We model (number of) ADLs, CESD score and Self-reported general health as ordered probits, onsets as binary probits (for at least one mild or severe onset), and CHI as a continuous outcome. Standard errors are corrected for clustering due to within individual and household correlations over time. We vary the number of lags of the dependent variable, keeping fix the number of lags of the right-hand side variables. Thus we can check if allowing for more lags in levels leads to different conclusions.

[Table 10 here]

In the first order models, the non-causality test is a joint test on the coefficient of $(y_{t-1}, y_{t-2}, y_{t-1} \times t)$. For husbands, the null is rejected in two out of six cases. For CHI, the null of no causality is rejected at the 10% level but not at the 5% level. For wives, the null of no causality from wealth to health is rejected in three out of six cases, including the case of CHI. This result differs from that in the complete model, where we found no significant effect of household wealth on the wife's health. If more lags are added, the results provide much weaker evidence of causal wealth to health effects. Only for one of the husband's health indicators (CESD) and one of the wife's health indicators (severe onset) a significant causal effect is found. Thus particularly for wives, allowing for a richer dynamic structure makes a substantial difference. To check if unobserved heterogeneity plays a role in these last two cases (CESD and severe onset), we also estimated the dynamic models (with specifications as in Tables 6 and 7) using the CESD index and the onset of a severe condition as the dependent variable. The result show that the rejections of no causality in Table 10 are due to residual unobserved heterogeneity, since none of the effects remain significant at any conventional level in the fixed effects models (see Appendix C).

Table 10 also provides tests for causal effects of the wife's health on the husband's health and vice versa (controlling for wealth etc.). The test for non-causality from the wife's health to the husband's health tests that coefficients for $(h^f_{t-1}, h^f_{t-2}$ and $h^f_{t-1} \times t)$ are all zero. First-order models provide evidence against non-causality. In four out of six cases, a significant (and positive) effect of the wife's health on the husband's health is found. On the other hand, the effect of the husband's health on the wife's health is significantly positive in three cases. The significance levels of the effects on the spouse's mental health (CESD scores) are the highest. The results for CHI would suggest causal effects in both directions. With the higher order models, the only significant effect is an effect of husband's health on the wife's mental health. Interestingly, this effect

does not disappear if we estimate a dynamic model with the wife's CESD index as the dependent variable (see Appendix C). Hence, there seems to be a genuine causal effect of the husband's health on the wive's mental health as measured by the CESD index. This effect gets swamped with the negative results for other health dimensions such that cannot be detected using the CHI.

Comparing the results for CHI with those in Tables 6 and 7 thus suggests that incorporating an appropriate lag structure makes the largest difference. Once this is done, controlling for fixed effects does not change the conclusions about causality, at least for CHI. This is in line with the conclusion from Table 8 that unobserved heterogeneity terms in the health equations are not so large. For the wealth equation, all models lead to the same conclusion - there are significant causal effects of both spouses' health indexes on household wealth.

6.3 Attrition and Outliers

The latter conclusion – both spouses' health indexes cause wealth – is also robust to other specification changes, such as robust regressions to reduce the influence of outliers, or regressions using a hyperbolic rather than a logarithmic transformation of wealth as the dependent variable.

Since panel attrition is associated with health (cf. Appendix A, Table 14), it might also affect the results of the causality tests. Following Nijman and Verbeek (1996), we checked this by comparing estimates for the complete unbalanced sample (Tables 5-7) with those for the the balanced sample consisting of those who remain (alive and) in the panel until 2002. The results show that attrition is largely ignorable for the parameters of interest and the tests for causality – the results using the balanced panels are always qualitatively similar to the presented results for the unbalanced panel (see Appendix C).

6.4 Alternative Measures of Health

Using the CHI incorporating all features of health, we have found clear evidence of causal effects of both the husband's and the wife's health on household wealth. Table 11 shows the results of a similar dynamic panel data model using separate indicators for physical and mental health. The physical health index combines onsets of physical disorders (all onsets except depression) and ADL-s, the mental health index combines the CESD score with the onset of depression. Self-reported general health is not included since it captures features of both mental and physical health. The table shows evidence of causal effects on household wealth of the husband's physical health and the wife's physical and mental health, while the husband's mental health is insignificant. A mental health problem of the wife has an instantaneous effect on household wealth, while the effect of the husband's physical health is not instantaneous, in line with the difference between husbands and wives found earlier for overall health. The stronger wealth effect of the wife's mental health status than of the husband's status is in line with Wu's (2003) argument that household expenditures increase if the wife is no longer able to perform

household tasks such as cooking and cleaning. The stronger effect of the husband's physical health might relate to his role as breadwinner. A model that simultaneously considers labor force participation and earnings would be needed to investigate this further.

[Table 11 here]

6.5 Disaggregation

An explanation for the strong effects of mental health may be the lack of insurance coverage for mental health problems. Since their coverage by Medicare and Medicaid is limited, employer-provided or other additional insurance coverage is necessary to protect against mental disease onsets (Adams et al., 2003). To investigate this, we disaggregated by health insurance coverage status estimating separate models for couples where none had insurance coverage in their first wave and couples where both had coverage. Table 12 shows the results.

[Table 12 here]

Rejection of non-causality from health to wealth is more frequent for couples who lack health insurance. Immediate effects of mental health problems are stronger among non-insured couples (direct effect is -0.541 for husbands and -0.886 for wives compared to -0.238 and -0.239 for those with insurance coverage). This means that an important channel for the effect of health on wealth in this age group is likely to be health expenditures, more so important for the uninsured.

As for the link between wealth to health, we have re-estimated the models on those who lack insurance as of the first wave (see Appendix C). Results are virtually the same, i.e. no effects from wealth to health. Since the effect may be non-linear in wealth, we have also estimated the models on the sub-sample of couples in the lowest wealth quartile in their first wave (693 couples). Again, the same conclusions emerge.

7 Conclusion

This paper uses dynamic panel data models to test for causal effects of health on socioeconomic status and vice versa for elderly couples in the US. The two main difference with earlier approaches is that this allows to control for unobserved heterogeneity and to select an appropriate lag structure using specification tests. Five biennial waves of couples in the HRS were used, following the 1931-1941 birth cohort from 1992 until 2002.

Our dynamic panel data model based tests provide clear evidence of causal effects from health to wealth, but no evidence of causal effects from wealth to either the husband's or the wife's health, or from one spouse's health on the health of the other spouse. Sensitivity analysis shows that simpler models without unobserved heterogeneity or with too few lags give biased results – they suggest causal effects of wealth on the health of

both spouses. The covariance structure of the residuals suggests that unobserved heterogeneity in wealth plays a more important role than in both husband's and wife's health (where state dependence can explain almost all correlation over time).

Disaggregating health into mental and physical health show that mental health is more important for wives while only physical health matters for husbands. While the mental health effects are instantaneous, the physical health effects take more time and are visible only in the next wave (two years later). Insurance coverage also appears to play a role here: it is mainly if wives without employer-provided insurance experience an onset of mental conditions that household assets decline.

The fact that we find no causal links from wealth to health for the age groups considered does not mean that such a causal link never operated earlier in life – we only consider households with one or both spouses in their fifties. It would be interesting to apply the same approach to younger households. It would also be interesting to look at different countries, and see whether the institutional setting makes a difference (Deaton, 2003; Hurd and Kapteyn, 2003).

Further research could also explicitly incorporate the role of labor force participation and earnings. The respondents in the HRS cohort that we consider are typically at work in the first wave and have retired before the last wave. One of the potential channels of health-wealth causality is through labor supply and earnings, making it worthwhile to extend the model with labor supply (and the decision to retire) and earnings.

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Table 1: Composition of Household Wealth

Wealth components	1992	2002
	median (part.)	median (part.)
Liquid wealth	38.81 (74.1)	80.0 (47.8)
IRAs	31.57 (45.1)	68.24 (47.2)
Stocks	25.24 (32.1)	50.0 (37.4)
Bonds	12.62 (6.9)	35.0 (8.6)
Checking/savings account	6.56 (86.2)	10.0 (89.1)
Cert./T.bills/Sav.Bds	10.10 (29.5)	20.0 (26.9)
Debt (subtracted)	3.78 (41.0)	5.0 (28.2)
Non-liquid wealth	103.51 (97.2)	136.0 (99.0)
Primary residence	100.98 (87.9)	130.0 (92.3)
Mortgage (subtracted)	37.87 (50.6)	56.0 (35.5)
Other real estates	56.80 (27.4)	70.0 (21.3)
Business assets	88.36 (15.2)	150.0 (13.4)
Trans./vehicules assets	12.62 (95.9)	15.0 (95.7)
Total wealth (bus. excluded)	142.64 (95.5)	211.0 (99.0)
Number of households	4150	2468

NOTES: Wealth in thousands of 2002 US dollars. Ownership rates in parentheses. Other debts or loans not shown in the table but enter negatively in the calculation of liquid wealth. Business assets not included.

Table 2: Health of Husbands and Wives

Health Indicators	Husbands		Wives	
Mean/Fraction reporting condition	1992	2002	1992	2002
Self-Reported Health	2.577	2.754	2.420	2.594
Severe Condition (ever had)	0.238	0.449	0.167	0.317
Cancer	0.039	0.132	0.059	0.119
Heart condition	0.148	0.286	0.072	0.153
Lung Disease	0.062	0.091	0.049	0.087
Stroke	0.038	0.075	0.017	0.045
Mild Condition (ever had)	0.565	0.813	0.554	0.799
Diabetes	0.101	0.208	0.076	0.138
High blood pressure	0.359	0.538	0.285	0.492
Arthritis	0.299	0.562	0.358	0.632
Emotional/psyc. problems	0.044	0.089	0.082	0.184
At least one ADL	0.045	0.105	0.034	0.102
CESD score	0.585	0.939	0.706	1.290
Body-Mass Index	27.31	27.98	26.76	27.82
Number of respondents	4160	2463	4160	2463

NOTES: Fractions with given conditions and means of quantitative health indicators. 1992 columns: all couples in 1992; 2002 columns: couples still in the panel in 2002. Self-reported health: scale from 1 (excellent) to 5 (poor); ADLs: limitations in performing activities of daily life; CESD scores: based upon eight questions on mental health; a higher score implies worse mental health.

Table 3: Distribution of Constructed Health Index for Husbands and Wives in 1992

Health 1992	Wife (from good to bad health)				
Husband	1st qtile	2nd qtile	3rd qtile	4th qtile	Total
1st quartile	419	286	219	176	1100
	(0.11,0.38)	(0.07,0.26)	(0.05,0.20)	(0.04,0.16)	
2nd quartile	285	255	250	200	990
	(0.07,0.29)	(0.06,0.26)	(0.06,0.25)	(0.05,0.20)	
3rd quartile	253	242	271	270	1036
	(0.06,0.25)	(0.06,0.23)	(0.06,0.26)	(0.06,0.26)	
4th quartile	164	185	310	375	1034
	(0.04,0.16)	(0.05,0.18)	(0.07,0.30)	(0.0,0.36)	
Total	1121	968	1050	1021	4160
Chi-square = 241.7 (df=15)					

NOTES: Higher quartile means worse health. First row for each quartile reports actual frequencies. Second row has relative cell frequency and conditional relative frequency within each quartile.

Table 4: The Health-Wealth Gradient

	Median wealth	
Husband's Health Index	1992	2002
best (1st) quartile	172	283
2nd quartile	144	208
%Δ 2nd – 1st	-16.2%	-26.5%
3rd quartile	141	191
%Δ 3rd – 1st	-18%	-32.5%
worst (4th) quartile	84	119
%Δ 4th – 1st	-51.2%	-57.9%
Wife's Health Index	1992	2002
best (1st) quartile	186	294
2nd quartile	172	265
%Δ 2nd – 1st	-7.3%	-9.8%
3rd quartile	116	177
%Δ 3rd – 1st	-37.6%	-39.8%
worst (4th) quartile	82	106
%Δ 4th – 1st	-55.9%	-63.9%

NOTES: Median total household wealth in thousands 2002 US dollars by constructed health index quartile in 1992, and percentage difference with first quartile.

Table 5: Dynamic Model for Household Wealth

	Reduced form		Structural	
Covariates	Par.	t-stat.	Par.	t-stat.
y_{t-1}	0.157	2.86	0.154	2.84
y_{t-2}	0.027	1.2	0.021	0.92
$y_{t-1}\times t$	-0.022	-1.28	-0.021	-1.22
h_t^m	-		-0.218	-1.48
h_{t-1}^m	-0.720	-4.95	-0.586	-3.53
h_{t-2}^m	-0.308	-3.70	-0.249	-2.75
$h_{t-1}^m\times t$	0.109	3.42	0.112	3.57
h_t^f	-		-0.434	-2.40
h_{t-1}^f	-0.402	-2.67	-0.155	-0.86
h_{t-2}^f	0.038	0.48	0.123	1.51
$h_{t-1}^f\times t$	0.020	0.6	0.022	0.68
	χ^2	p-val	χ^2	p-val
Sargan p-value	45.64	0.04	46.75	0.04
AR(2) test on residuals	-0.096	0.95	-0.025	0.96
Causality tests (p-values)				
$h_{t-1}^m, t\times h_{t-1}^m, h_{t-2}^m$	<0.001			
$h_{t-1}^f, t\times h_{t-1}^f, h_{t-2}^f$	<0.001			
$h_t^m, h_{t-1}^m, t\times h_{t-1}^m, h_{t-2}^m$			<0.001	
$h_t^f, h_{t-1}^f, t\times h_{t-1}^f, h_{t-2}^f$			<0.001	
h_t^m, h_t^f			0.01	
N	3386		3386	

NOTES: Two-step system GMM estimates imposing mean stationarity.

Table 6: Dynamic Model for the Husband's Health

	Reduced Form		Structural	
Covariates	Par.	t-stat.	Par.	t-stat.
h^m_{t-1}	0.571	12.63	0.571	11.75
h^m_{t-2}	0.272	15.12	0.272	14.18
h^m_{t-3}	0.102	4.76	0.100	4.65
$h^m_{t-1}\times t$	0.009	0.85	0.009	0.82
h^f_t			0.037	0.78
h^f_{t-1}	0.052	1.45	0.033	0.74
h^f_{t-2}	-0.003	-0.21	-0.014	-0.7
$h^f_{t-1}\times t$	-0.009	-1.07	-0.010	-1.21
y_t			-0.003	-0.13
y_{t-1}	0.001	0.04	0.001	0.10
y_{t-2}	-0.002	-0.41	-0.002	-0.43
$y_{t-1}\times t$	-0.001	-0.24	-0.001	-0.27
	p-val		p-val	
Sargan test	0.585		0.546	
Causality tests				
$h^f_{t-1}, t\times h^f_{t-1}, h^f_{t-2}$	0.41			
$y_{t-1}, t\times y_{t-1}, y_{t-2}$	0.71			
$h^f_t, h^f_{t-1}, t\times h^f_{t-1}, h^f_{t-2}$	.	.	0.547	
$y_t, y_{t-1}, t\times y_{t-1}, y_{t-2}$	.	.	0.961	
N	3051		3051	

NOTES: Two-step system GMM estimates imposing mean stationarity.

Table 7: Dynamic Model for the Wife's Health

	Reduced form		Structural	
Covariates	Par.	t-stat.	Par.	t-stat.
h^f_{t-1}	0.574	13.25	0.570	13.16
h^f_{t-2}	0.254	13.1	0.253	13.11
h^f_{t-3}	0.080	3.73	0.082	3.82
$h^f_{t-1}\times t$	0.004	0.37	0.003	0.29
h^m_t	.	.	0.031	0.80
h^m_{t-1}	0.045	1.21	0.003	0.06
h^m_{t-2}	0.002	0.17	-0.018	-0.96
$h^m_{t-1}\times t$	-0.007	-0.76	-0.004	-0.45
y_t	.	.	-0.034	-1.33
y_{t-1}	0.007	0.73	0.007	0.61
y_{t-2}	-0.002	-0.54	-0.001	-0.21
$y_{t-1}\times t$	-0.001	-0.35	0.001	-0.12
	p-val		p-val	
Sargan test	0.412		0.325	
Causality tests				
$h^m_{t-1}, t\times h^m_{t-1}, h^m_{t-2}$	0.551			
$y_{t-1}, t\times y_{t-1}, y_{t-2}$	0.693			
$h^m_t, h^m_{t-1}, t\times h^m_{t-1}, h^m_{t-2}$	.	.	0.552	
$y_t, y_{t-1}, t\times y_{t-1}, y_{t-2}$	.	.	0.988	
N	3051		3051	

NOTES: Two-step system GMM estimates imposing mean stationarity.

Table 8: Covariance Structure of Individual Effects

covariance matrix	wealth	husband's health	wife's health
wealth	1.119 (81.5%)		
husband's health	0.019	0.019 (8.9%)	
wife's health	0.012	-0.003	0.016 (6.9%)

NOTES: Estimated using residuals from structural models in Tables 5, 6 and 7. Using observations in the balanced panel only, the covariance matrix can be constructed as follows: Denote the GMM residuals by $\hat{u}_{it}^j$, $j = 1, 2, 3$, $i = 1, ..., n$, $t = P + 1(= 4), ..., T(= 6)$. Let $\hat{\eta}_i^j = (T - P)^{-1} \sum_{t=P+1}^{T} \hat{u}_{it}^j$ (a point estimate for the individual effect). Under the assumptions of stationarity and independence of individual effects and error terms, it is easy to show that a consistent estimator (with T fixed and $n \to \infty$) for $Cov(u_{it}^j, u_{it}^k)$ is given by $(T-P-1)^{-1}[(T-P)\hat{Cov}(\hat{\eta}_i^j, \hat{\eta}_i^k,) - \hat{Cov}(\hat{u}_{it}^j, \hat{u}_{it}^k)]$, where $\hat{Cov}$ denotes the sample covariance. Similar consistent estimators can be constructed using observations that are not in the balanced panel, particularly households observed 5 consecutive times for which we have 3 (2 health) GMM residuals. The reported numbers are weighted averages of the covariance matrix estimates using households with 6 and 5 panel observations, weighted with the numbers of such households.

Table 9: Correlates of the Estimated Individual Effects

	Estimates			Estimates	
Covariates	Wealth	Health	Covariates	Wealth	Health
age m	0.193*	-0.049**	age f	0.125	0.015
age^2 m	-0.014	0.004**	age^2 f	-0.008	-0.001
high school m	0.670**	-0.023	high school f	0.564**	-0.026
college & above m	0.908**	-0.022	college & above f	0.653**	-0.029
high school (m,father)	0.185	0.016	high school (f, father)	0.106	-0.027
college & above (m,father)	0.425**	-0.023	college & above (f,father)	0.139	-0.041*
living parents (m)	-0.021	-0.013	living parents (f)	0.035	-0.024**
BMI [30,35] (m)	-0.011	0.053**	BMI [30,35] (f)	0.141	0.032*
BMI [35,+] (m)	0.091	0.062*	BMI [35,+] (f)	0.015	0.141**
ever smoked (m)	-0.007	0.031**	ever smoked (f)	-0.046	0.047**
manual occup. (m)	0.045	-0.003	manual occup. (f)	0.259	0.038*
occup. missing (m)	-0.123	-0.034	occup. missing (f)	-0.131	0.034
risk averse (m)	0.192	-0.019	risk averse (f)	0.113	0.002
aversion missing (m)	-0.301	-0.023	aversion missing (f)	-0.302	0.092
plan few years ahead (m)	0.161	0.032	plan few years ahead (f)	0.161	0.013
plean 5> years ahead (m)	0.190	0.034*	plean 5> years ahead (f)	0.225**	-0.002
planning missing	0.352	0.028	planning missing	-0.173	0.056
African-American	-0.908	0.023	African-American	-0.908	0.003
Hispanic	-0.565	0.002	Hispanic	-0.565	-0.043
constant	-12.513	1.196	Constant	-12.513	-0.458
N	3051			3051	

NOTES: parameter estimates from linear regression with robust standard errors. The equation for wealth includes all characteristics of both spouse. The equations for individual health effects are spouse-specific.Two starts denotes statistical significance at level lower than 5 pct, One star at 10 pct level.

Table 10: Levels Test for Health

Husbands (over all waves)						
Test (Chi-sq & p-value)	Health Index	Severe Onset	Mild Onset	Self-Report	CESD	ADL
Joint Test Wealth (df=3)						
One Lag of health	2.22	1.45	9.41	1.15	22.76	3.33
	0.083	0.693	0.023	0.0764	<0.001	0.348
Three Lags of health	0.07	0.86	6.30	1.93	14.3	1.39
	0.978	0.836	0.098	0.586	0.002	0.704
Joint Test Health (df=3)						
One Lag of health	7.3	7.09	1.15	13.41	19.54	7.91
	<0.001	0.069	0.764	0.003	<0.001	0.048
Three Lags of health	0.81	4.96	0.84	4.27	6.08	3.78
	0.486	0.180	0.838	0.234	0.108	0.287
Wives (over all waves)						
Covariates	Health Index	Severe Onset	Mild Onset	Self-Report	CESD	ADL
Joint Test Wealth (df=3)						
One Lag of health	6.37	8.76	11.17	4.54	1.43	5.87
	<0.001	0.032	0.011	0.201	0.69	0.118
Three Lags of health	1.76	9.31	7.79	2.04	2.75	2.1
	0.153	0.025	0.051	0.565	0.433	0.558
Joint Test Health (df=3)						
One Lag of health	5.19	1.81	1.77	13.3	17.58	2.33
	0.001	0.612	0.622	0.004	<0.001	0.507
Three Lag of health	0.97	1.03	0.67	9.69	12.31	0.84
	0.408	0.793	0.879	0.021	0.006	0.83

NOTES: Specification of Non-causality tests as in Table 6.7. The chi-square tests have 3 degrees of freedom. Model for health index is estimated by OLS; models for self-reported health, CESD scores and ADL count are ordered probits; models for the remaining variables are probits. Errors are clustered at the household level. Controls for demographics and lagged risk factors included. Detailed results available upon request.

Table 11: Wealth Responses to Mental and Physical Health

	Estimates		Causality test	
Covariates	Par.	t-stat.	test	p-value
y_{t-1}	0.146	2.66		
y_{t-2}	0.036	1.55		
$y_{t-1}\times t$	-0.013	-0.76		
Mental Health				
h^m_t	-0.616	-1.42		
h^m_{t-1}	-0.031	-0.13		
h^m_{t-2}	0.051	0.41		
$h^m_{t-1}\times t$	0.062	1.64	2.04	0.110
h^f_t	-1.125	-2.95		
h^f_{t-1}	0.298	1.55		
h^f_{t-2}	0.229	2.33		
$h^f_{t-1}\times t$	0.053	1.54	3.24	0.047
Physical Health				
h^m_t	0.139	0.91		
h^m_{t-1}	-0.484	-2.81		
h^m_{t-2}	-0.127	-1.27		
$h^m_{t-1}\times t$	0.093	2.90	3.02	0.033
h^f_t	0.051	0.27		
h^f_{t-1}	-0.378	-1.93		
h^f_{t-2}	-0.060	-0.56		
$h^f_{t-1}\times t$	0.001	0.04	4.12	0.003
N	3386		3386	

NOTES: Two-step system GMM-estimates imposing mean stationarity. The mental health index is composed of CESD scores and onsets of mental health conditions; the physical health index is based upon onsets of other severe and mild conditions and ADLs. Self-reported health excluded.

Table 12: Results by Health Insurance Status

	Insurance Coverage	
Joint Test (p-value)	None	Both
Immediate mental	0.006	0.437
Immediate physical	0.199	0.620
Husband		
mental	<0.001	0.332
physical	0.088	0.652
Wife		
mental	0.269	0.598
physical	0.055	0.001
N	417	867

NOTES: Specification of Non-causality tests as in Table 5.

A Sample Selection and Descritive Statistics

Table 13: Sample Composition

	Couples	Exit rate (%)
Sample 1992	4160	
Exits 1993-1994	419	10.07
Sample 1994	3741	
Exits 1995-1996	355	9.48
Sample 1996	3386	
Exits 1997-1998	335	9.89
Sample 1998	3051	
Exits 1999-2000	278	9.11
Sample 2000	2773	
Exits 2001-2002	310	11.18
Sample 2002	2463	

NOTES: Observations per wave, exits between waves, and exits as a percentage of observations per wave.

Table 14: Demographic characteristics in 1992 by period of exit from the panel

Demographics 1992	No-Exit	Ex 1993	Ex 1995	Ex 1997	Ex 1999	Ex 2001
Husband						
Age (years)	56.70	56.96	57.67	58.06	58.71	57.80
Years of schooling	12.44	11.75	11.36	11.91	11.77	11.74
Dummy Hispanic	0.071	0.093	0.079	0.071	0.075	0.096
Dummy Black	0.097	0.186	0.146	0.164	0.111	0.106
Dummy Not born in U.S.	0.089	0.093	0.104	0.101	0.089	0.087
Dummy >1 marriage	0.279	0.372	0.313	0.319	0.291	0.316
Wife						
Age (years)	52.84	52.90	53.40	54.30	53.91	53.76
Years of schooling	12.39	12.03	11.73	12.08	11.86	11.74
Dummy Hispanic	0.073	0.105	0.079	0.080	0.064	0.119
Dummy Black	0.095	0.169	0.149	0.167	0.111	0.103
Dummy Not born in U.S	0.099	0.107	0.101	0.119	0.061	0.112
Dummy >1 marriage	0.243	0.353	0.256	0.281	0.273	0.312
Number of couples	2463	419	355	335	278	310

NOTES: Ex 1993 refers to the subsample exiting the panel between the 1992 and 1994 waves, etc.

B Constructed Health Indices (CHI)

The health index is constructed by principle component analysis using 6 covariates: self-reported health, prevalence of severe and mild health conditions, CESD scores and the number of ADLs. This is performed for husbands and wives separately, pooling the data across all waves. One factor is retained from the analysis. Factor scores are presented in Table 14 along with Eigenvalues. The variable that score highest is self-reported health, but other variables also contribute. We also experimented with alternative health indices using fewer health variables; this gave similar results of the tests for causal effects. The constructed health index is normalized to have mean 0 and variance 1; it varies between -3 and 5 and the distribution tends to be skewed toward zero.

Table 15: Results of Principle Component Analysis

Health Indicators (husbands)	scoring	1st factor	Uniqueness	Factors	Eigenvalues
Self-Reported Health	0.37	0.79	0.37	1st	2.14
Severe Condition (ever had)	0.30	0.64	0.59	2nd	0.89
Mild Condition (ever had)	0.30	0.65	0.58	3rd	0.75
CESD score	0.27	0.57	0.67	4th	0.70
ADL	0.28	0.60	0.64	5th	0.52
Retained Factor 1	N	20373	LR (p-val)	<0.001	
Health Indicators (wives)	scoring	1st factor	Uniqueness	Factors	Eigenvalues
Self-Reported Health	0.35	0.79	0.37	1st	2.24
Severe Condition (ever had)	0.26	0.58	0.66	2nd	0.85
Mild Condition (ever had)	0.31	0.70	0.51	3rd	0.74
CESD score	0.28	0.62	0.62	4th	0.68
ADL	0.28	0.63	0.60	5th	0.50
Retained Factor 1	N	20373	LR (p-val)	<0.001	

C Additional Results for Health Equations

Table 16: Additional Results for Husband's Health

Husbands	Reduced Form		
Joint Test p-values	All	Balanced	Dep.=CESD
Spouse health	0.405	0.361	0.476
Wealth	0.713	0.642	0.444
	Structural		
Joint Test p-values	All	Dep.= CESD	Low SES
Spouse health	0.544	0.723	0.701
Wealth	0.961	0.481	0.847

NOTES: The first column refers to the specification in Table 6, Balanced refers to estimation on the balanced sample. The third column of the reduced form panel refers to the same specification as the first but using the CESD score as the dependent variable instead of the index. Finally, the third column of the second panel refers to estimation restricting to individuals in the lowest quartile of wealth in 1992.

Table 17: Additional Results for Wive's Health

Wives	Reduced Form			
Joint Test p-values	All	Balanced	Dep.=CESD	Severe
Spouse health	0.551	0.501	0.016	0.726
Wealth	0.691	0.623	0.541	0.766
	Structural			
Joint Test p-values	All	Dep.= CESD	Dep.=Severe	Low SES
Spouse health	0.552	0.078	0.943	0.745
Wealth	0.988	0.798	0.844	0.847

NOTES: The column titled severe estimates the same specification as in Table 7 but making use of the onset of a severe health shock as the dependent variable.