Health and Wealth of Elderly Couples: Causality Tests Using Dynamic Panel Data Models*

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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 most industrialized countries with similar levels of health care technology and economic welfare. This study analyzes causality from health to wealth (health causation) and from wealth to health (wealth or social causation) for elderly couples in the US. Using six biennial waves of couples aged 51-61 in 1992 from the Health and Retirement Study, we compare the recently developed strategy using Granger causality tests of Adams et al. (2003, *Journal of Econometrics*) with tests for causality in dynamic panel data models incorporating unobserved heterogeneity. While Adams et al. tests reject the hypothesis of no causality from wealth to husband's or wife's health, the tests in the dynamic panel data model do not provide evidence of wealth-health causality. On the other hand, both methodologies lead to strong evidence of causal effects from both spouses' health on household wealth.

JEL Codes: C33, D31, I12, J14.
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1 Introduction

Explaining the health-wealth gradient, the observed association between wealth and health, has been a challenge for many economists as well as other social scientists. 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 of respondents had grown to 274%, with median wealth $127,900 for those who reported excellent health in 1984, and $34,700 for those in fair or poor health in 1984 (amounts in 1996$). The ratio in 1984 was largest for the age group 45-54, an impressive 176%, which increased to 264% in 1994. Although often less pronounced than in the United States, 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 systematically over the life-cycle. Adda (2003) finds similar results for Sweden, with a health-wealth correlation that peaks at about the same age. In the United Kingdom, one of the puzzles created by the widely cited Whitehall I (1967) and II (1985-1988) studies (Marmot, 1999) looking at the health of civil servants over three decades, 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). A similarly challenging finding is the evidence of Deaton and Paxson (1998) that, controlling for age, health assessments show no significant increases and even tend to decrease slightly for men and women born after 1945, even though, on average, these cohorts live longer and are wealthier than earlier cohorts.

Understanding the sources of the gradient is important in order to understand the sources of health inequalities and to design economic policy measures 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 bottom 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 income, in which 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. Health (measured from bad to good) is positively related to household savings, labor force participation,

and earnings, and negatively related to the social security retirement benefits replacement rate. Availability of Medicare at age 65 may explain the retirement peak at that age, where social security incentives no longer apply (Rust and Phelan, 1997; Blau and Gilleskie, 2001). 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). To understand the sources of the health-wealth or health-SES gradient, it is important to realize that health and wealth are dynamic processes that evolve over an individual's life-cycle. A large part of the life-cycle is subject to the history of a series of shocks and events on the health and wealth front. Some of these are under the individual's control and others are completely unpredictable.

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.[1] Health is worth investing in since it yields utility: it extends life and therefore the horizon over which gains from productivity can be used for consumption and provides consumption of healthy days that can be enjoyed through leisure (as opposed to sick days which do not yield utility). 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 as one progresses over the life-cycle, health is more and more predetermined by the complete past of the individual.

The relation between health and wealth can be explained in this framework. Health and expectations about future health can affect productivity and hourly wages as well as labor supply at the intensive and the extensive margin. It therefore drives the capacity to accumulate savings for retirement, and affects the retirement decision both in this way and through the direct effect of health on the marginal rate of substitution between leisure and work. Moreover, health affects expenditures directly, particularly in the United States where about 20% of workers below 65 are not covered by health insurance (Gruber, 1998), and where even those who are covered will often face copayments or additional expenditures such as prescription drugs not covered by Medicare. Consequently, health events can lead to considerable revisions of saving plans or other life-cycle decisions such as bequests (Smith, 2003). Causal effects from health to wealth are also referred to as health causation.[2]

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 is often named *social causation* which we will refer to as SES or wealth causation, the opposite of health causation. Theories explaining such a link have been put forward in various fields, such as biology, psychology, and economics. For example, one explanation is *risk behaviors*: the relation between behavior that is

[1] see Dustmann and Windmeijer (1999) for an empirical application of the Grossman model.

[2] This is often referred as health selection in the social science literature.

detrimental to health like smoking and drinking and socioeconomic status (Marmot, 1999).

The effect of health on wealth may be related to *access to health care*. If not all people are fully covered by the same 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 United Kingdom 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. Moreover, it is hard to reconcile this explanation with the evidence provided by the RAND health experiment (Newhouse, 1993), which, in an experiment with randomly assigned copayment rates, showed that those with lower copayment rates used on average more health care services but did not experience significantly different health outcomes. The variation in quality of care and treatments that one can obtain in different socioeconomic groups may be even more important to this issue 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 level (an indicator of socioeconomic status), 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 real differences in health knowledge.

Another potential causal effect of wealth on health through *wealth inequalities* comes from the stress associated with being at the bottom of the distribution (Wilkinson, 1996). In the Whitehall study, Marmot (1999) shows that there is some evidence 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. The observation that wealth inequalities have risen but that the average health level may have fallen (as found by Deaton and Paxson, 1998) would be in line with this effect, as is Wilkinson's (1996) finding that countries with higher wealth inequality tend to have higher mortality rates. 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 may 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, *allostatic loads*, a measure of the cumulative effect of stressful events on the health system (see, e.g., Seeman et al., 1997), will be different across SES groups .

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 were significant predictors of 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. (2003) found that macroeconomic conditions at birth affected mortality hazards of cohorts throughout the 19th and 20th century, highlighting the importance (the "reach" in terms of Smith, 1999) of early childhood environment. 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 due to the causal links from SES to health discussed above. 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 fifty 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 persistent unobserved heterogeneity terms may drive household wealth, and the unobserved heterogeneity terms in household wealth and in health of both spouses can be correlated.

The goal of this paper is to disentangle the sources of the health-wealth gradient: causal effects from health to wealth (health causation), causal effects from wealth to health (wealth or SES causation), observed exogenous factors that affect health and wealth in the same way, and correlated unobserved factors (unobserved heterogeneity) driving health as well as wealth. Panel data with extensive information on wealth and health offer a non-experimental setting in which causality can be addressed using common time series concepts of non-causality and conditional independence (Granger, 1969; Sims, 1972). If correlation between unobservables plays a role, these tests will only be valid if they control for such correlations (Chamberlain, 1984).

Using Granger causality to study the health-wealth gradient was proposed by Adams et al. (2003), who test for an effect of wealth on health in the AHEAD cohort of age 70 and older. They only have three waves, limiting the richness of the dynamic specifications they can use. Moreover, they do not control for unobserved heterogeneity. Their results indicate a clear health causation channel but they also find some 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 causation mechanisms are present. He does not discuss or control for 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 unobservables. 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, while Wu (2003) concludes 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, implying that the dynamics of health and SES causation are not explored.

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 and the dynamics of their model imply that wealth changes have a one shot 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 self-reported general health status and for mental health. The latter results for the U.S. are also found by Adda et al. (2003) for the U.K. and Sweden. Using roughly similar models as Adams et al. (2003), Hurd and Kapteyn (2003) find that changes in health are more related to income in the U.S. than in the Netherlands. In all these three studies, a test of non-causality is performed without controlling for unobserved heterogeneity. As argued above, correlated unobserved heterogeneity may be important, because of genetic transmissions and early childhood effects and other persistent shocks on health as well as wealth. Not allowing for unobserved heterogeneity may bias the estimates and the test results, possibly explaining why the null of no causality is often rejected.

In this paper, we develop a dynamic vector autoregressive panel data framework that makes it possible to test for health and wealth causation, controlling for unobserved heterogeneity. Alonso-Borrego and Arellano (1999) emphasize that dynamic vector autoregressive panel data models offer a rich environment for performing such tests. We apply the framework to the HRS cohort of elderly households born between 1931 and 1941 who are observed over six biennial waves from 1992 to 2002. We consider health for each spouse but wealth at the household level, analyzing the interplay of health and wealth for elderly couples (as in Wu, 2003). We use the instruments of Smith (2003), Wu (2003) and Meer et al. (2003) to identify the structural links between health and wealth, conditioning on initial conditions. We perform the tests and explore their sensitivity to different sets of assumptions, particularly concerning the types of dynamic feedback allowed for and the specification of the initial conditions (Ahn and Schmidt, 1995; Blundell and Bond, 1998). We also present some results where we separately look at mental and physical health, distinguish between couples that do and do not have access to health insurance, and look at liquid and non-liquid wealth.

The paper is organized as follows. In section 2 we document the association between wealth and health and the way it evolves over time for the HRS cohort. In section 3, the econometric framework is presented and the identification, testing and estimation strategies are discussed. Section 4 presents the results of the Adams et al. (2003) tests and Section 5 presents the results for the dynamic panel data models. Section 6 concludes.

2 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. The project started in 1990 and was 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 elderly singles and couples. 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 even if they were not eligible according to their age.

We use the public release file from the RAND corporation that merged records from the six available waves (1992-2002).[3] Data is arranged by couples consisting of respondent and spouse. We select all couples present in 1992 with complete information on the relevant variables during their participation in the HRS. To avoid losing too many observations, we retain observations with missing information or bracket information on one or more components of wealth, using imputed values (see below).

We observe couples until one of the spouses dies, until the dissolution of the household because of divorce or separation, or until one member of the household is not interviewed. We do not analyze widows and widowers or divorced or separated spouses, since we focus on the interplay between wealth and the health of the two spouses in a couple.

Table 1 gives the frequencies at each wave along with the recorded exits from our sample. Overall, the average attrition rate for each wave is roughly 10% which gives an annual attrition rate of about 5%.[4] In 1992, there are 4,160 households of which 2,463 remain until the sixth wave in 2002.

[Table 1 about here]

Table 2 shows the demographic composition of the sample in 1992 according to the number of waves the respondents remain in the panel. Wives are on average four years younger than their husband. Both spouses have a similar average level of education. Approximately 6% of respondents are Hispanic and about 8% are blacks. These figures reflect the oversampling of those groups in the HRS. About 8% of husbands are immigrants, compared to 10% of the wives. One out of four respondent has been married at least once before their current relationship.

Those who exit before the end of the panel are on average older, which is an obvious consequence of decreasing survival probabilities. Attritors have slightly less education than respondents who remain in the panel for all six waves. Blacks and Hispanics seem more likely to exit than others.

[Table 2 about here]

[3] See http://www.rand.org/labor/aging/dataprod/. We used version D of the data released in January 2004.

[4] 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/).

2.1 Wealth Data

We summarize wealth in two broad categories: liquid and non-liquid wealth. Liquid wealth consists of individual retirement accounts, stocks, bonds, certificate deposits, T-bills/saving bonds, checking/saving accounts and other debts and savings. Non-liquid wealth includes the net value of the primary residence, other real estate, and vehicles. This definition is the same as the one used by Adams et al. (2003), except that we do not include business assets, which is nonzero for not many respondents but varies enormously over time for some respondents. It does not include the value of defined contribution pension plans but does include the value of life insurances and other annuities (in "other savings"). All amounts are expressed in US dollars of 2002 using the Consumer Price Index of the Bureau of Labor Statistics. In the analysis, we will use log transformations of the different wealth measures to reduce the effect of outliers.[5]

Table 3 gives the composition of wealth holdings for our sample. The first column gives the percentage of cases where imputation was used across all waves for each wealth component. RAND Imputations are used for open and closed bracket responses and for ownership of specific items (see Hoynes et al. (1998) for a comparison of imputation methods). The next two columns give the median of each component conditional on ownership (with 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, primarily consisting of the value of the primary residence. The share of non-liquid assets in total wealth falls over the decade.

Participation of the elderly in stocks and individual retirement accounts is far more important in the United States than in many other countries (Hurd, 2001). More than half of the respondents in the panel own Individual Retirement Accounts (IRAs), with a median value of $31,570 in 1992. Moreover, by 2002, 37.4% of households hold stocks for a median value of $50,000. Increases in IRA and stock holdings from 1992 to 2002 certainly reflect to some extent the high returns observed throughout the period. Participation went from 32.1% to 37.4% for stocks and from 45.1% to 47.2% for IRAs from 1992 to 2002. The median value of stocks and IRAs more than doubled over the 10 years.

[Table 3 about here]

2.2 Health Variables

Table 4 summarizes the health information for the 1992 and 2002 waves. The HRS age groups are subject to considerable health risks. In 1992, 16.7% (23.8%) of wives (husbands) have suffered from a condition that Smith (2003) labels a severe one: cancer, heart condition, lung disease or a stroke (or a combination of these). More than half the respondents have ever had an onset of a mild condition - diabetes, high blood

[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 of wealth, this is approximately log wealth.

pressure, arthritis, or mental problems (depression). This makes clear that these elderly respondents have a whole health history behind them, suggesting that much of the current association between health and wealth in the time period that the respondents are observed may stem from their past.

[Table 4 about here]

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 in the 10 years covered by the survey, about one in every five respondents experienced their first severe health condition. In 2002, 81.3% of husbands (79.9% of wives) had experienced the onset of a mild health condition, mostly arthritis (56.2% for husbands and 63.2% for wives) and high blood pressure (53.8% for husbands and 49.2% for wives).

Mental health problems are much more frequent for wives (18.4% in 2002) than for husbands (8.9% in 2002). Similar differences are found for the *CESD scores*, computed from a series of questions measuring mental health.[6] The Body-Mass Index (BMI) increases more over time for wives than for husbands. The percentage of respondents having difficulties with activities of daily living (ADL) also increases over time (doubles) and is always larger for wives than for husbands. Husbands are more pessimistic with respect to their chance of surviving up to 75 than their wives are in 1992 but this gap is eliminated by 2002. One fifth of all respondents report having health problems limiting work in 1992. This increases to one fourth in 2002.

Our analysis requires one summary measure of health. General indicators like self-reported health convey general information about health, while the indicators for onsets of health conditions or the CESD scores yield more specific information. Adams et al. (2003) consider each of these dimensions independently while they recognize that all indicators are interrelated. Hurd and Kapteyn (2003) consider self-reported health status and Smith (2003) studies serious health conditions.

We will work with a one-dimensional health indicator. Following Adda (2003), we build a "constructed health index" (CHI) from the indicators displayed in Table 4, using principal component analysis. This measure combines the many dimensions and indices of health outlined in Table 4. The index is normalized such that it has mean 0 and variance 1. Low values of the index refer to good health while high values refer to bad health. Most factors score highly, with self-reported health and health onsets scoring the highest.

[Table 5 about here]

In Table 5 we present the bivariate distribution of the husband's CHI and the wife's CHI in 1992. The table shows that health of husband and wife are correlated. For

[6]This score is based on the Center for Epidemiologic Studies Depression (CESD) scale. It gives the sum of six negative indicators minus two positive indicators. The negative indicators measure 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 measure whether the respondent felt happy and enjoyed life, all or most of the time.

example, 36% of wives of husbands in the best health quartile are also in the best health quartile themselves. On the other hand, only 13% of wives of respondents in the worst health quartile are in the best 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 behaviors).

2.3 Association between Wealth and Health

Table 6 reveals the health-wealth gradient in the 1992 and 2002 waves, in a similar way as Table 1 of Smith (1999). It presents median household wealth by 1992 health quartile (using the CHI to measure health). 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 worst health quartile. For the same households, the wealth difference was even larger in 2002. Median household wealth for wives in the worst health quartile in 1992 was only 40% of median household wealth for wives in the best health quartile. For the same households, the wealth differential increased even further in 2002. These differences are of similar magnitude as those found in Hurd and Kapteyn (2003) and Smith (1999) using similar data source but distinguishing health categories using self-reported general health, which has a high weight in the CHI-s. These remarkable differences do not only appear at the extremes of the distribution. Even among the households with relatively healthy wives in the second quartile in 1992, median wealth is 20 to 25% lower than in the top health quartile. Thus the association between health and wealth is not a simple dichotomy between "the rich and the poor" but rather a gradient that is observed everywhere in the SES ladder.

[Table 6 about here]

3 Econometric Methodology

3.1 The Evolution of Health and Wealth

We develop a model for three outcome variables for a given couple $i = m$(husband) and f(wife) in year t: $\mathbf{Y}_{it} = (h^m_{it}, h^f_{it}, y_{it})'$ where h^j_{it} is health of spouse j and y_{it} is the log transformed value of household wealth. As explained in section 1, a model explaining the evolution of wealth and health should have several features. First, it must allow for instantaneous causality as well as dynamic feedback from wealth to health and vice versa. This captures the most cited pathways, causal effects of wealth on health (wealth causation) and of health on wealth (health causation). Second, it should address whether or not health influences the health of the spouse, potentially trough mental health or other channels, as possible explanations for the association between CHI-s of both spouses, apparent from Table 5. Moreover, the model should take into

account potentially correlated unobserved heterogeneity in health and wealth, leading to a permanent correlation of wealth and health from the beginning of the observation window. We will use a panel data vector autoregressive model for $\mathbf{Y}_{it}$ that captures the features discussed above and allows for the various explanations of the gradient. The model is given by a Pth order vector autoregressive process:

$$\mathbf{\Gamma Y}_{it} = \mathbf{A x}_{it} + \sum_{p=1}^{P} \Phi_p \mathbf{Y}_{it-p} + \eta_i + \varepsilon_{it} \tag{1}$$

The matrices $\mathbf{\Gamma}, \mathbf{A}$ and Φ contain the parameters of the model. $\mathbf{x}_{it}$ is a vector of time invariant and time varying characteristics of the household (education, race, age, etc.). These characteristics can be correlated with a vector of time-invariant unobserved heterogeneity terms η_i, which, for example, capture unobserved traits at birth such as genes of members of the household, early childhood events (cf. Barker, 1997; Wadsworth and Kuh, 1997) or other intergenerational factors that affect health and wealth. We will allow that within each couple the three unobserved heterogeneity terms are correlated. The transitory shocks in ε_{it} are also potentially correlated.

The matrix Φ contains the parameters that reflect causal links that take some 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 parameters in the matrix $\mathbf{\Gamma}$, we also allow for instantaneous causality. This is particularly relevant in our case since observations are spaced by two years, and it seems 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. Such effects may point at direct mental or physical health links, but since our health variables also incorporate self-reported health and subjective life expectancy, they may also mean that respondents adjust their subjective beliefs following a deterioration in the health of their spouse.

Each component of $\mathbf{Y}_{it}$ has its own dynamics propagating the effect of shocks and potentially increasing their long-term impact. In order to estimate the dynamic interactions between health and wealth consistently, it is crucial to incorporate a dynamic structure that is flexible enough to describe the data. In particular, the order P of autoregression has to be chosen large enough. Specification tests as in Arellano and Bond (1991) will be used for this purpose.

Since the individual effects are allowed to be correlated with the regressors in $\mathbf{x}_{it}$, it will not be possible to estimate the influence of time-invariant regressors. For the same reason, it is not possible to disentangle the effects of age and a common time trend. For similar reasons, we will also not include variables on risk behavior (smoking, drinking). Persistent risk behavior over the life cycle can have a causal effect on health and also correlates negatively with socio-economic status. This, however, is captured by the individual effects. On the other hand, the variation of risk behavior over time in the elderly age group that we consider is likely to be endogenous: people stop smoking or

drinking due to health problems. Indeed, in the data, very few elderly individuals start smoking (about 1%), while more than 17% stop smoking. Incorporating risk behavior would require instrumenting it and this is beyond the goal of the paper. Instead, it should be kept in mind that some of the mechanisms that we find may be due to behavioral changes.

3.2 Estimation, Identification, and Causality Tests

In this panel data setting, it is possible to test for causality taking account of the presence of unobserved heterogeneity in η_i, avoiding the problem that the null of no causality between health and wealth can be rejected due to "spurious correlation." We first consider the reduced form model in which instantaneous causality is eliminated, explain how to estimate this model with GMM, and how to test for causality using a Wald test. We then turn to the structural model with instantaneous causality and instruments needed for identification, and discuss estimation and testing for causality in that model also.

Tests for Reduced-Form Vector Autoregressions

Consider the reduced-form VAR of (1),

$$\mathbf{Y}_{it} = \mathbf{B}\mathbf{x}_{it} + \sum_{p=1}^{P} \mathbf{C}_p \mathbf{Y}_{it-p} + \eta_i^* + \varepsilon_{it}^* \tag{2}$$

where $\mathbf{B} = \mathbf{\Gamma}^{-1}\mathbf{A}, \mathbf{C}_p = \mathbf{\Gamma}^{-1}\Phi_p$ for $p = 1, .., P$, $\eta_i^* = \mathbf{\Gamma}^{-1}\eta_i$ and $\varepsilon_{it}^* = \mathbf{\Gamma}^{-1}\varepsilon_{it}$. For a test for wealth causation in the sense of Granger (1969) causality, the null hypothesis of no causality can be written as

$$H_0 : E(\mathbf{h}_{it+1}|\mathbf{Y}_i^t, \mathbf{x}_{it}^t, \eta_i^*) = E(\mathbf{h}_{it+1}|\mathbf{h}_i^t, \mathbf{x}_i^t, \eta_i^*) \text{ for } t = 0, ..., T \tag{3}$$

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

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

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, etc.

The null hypothesis of no health causation is given by

$$H_0 : E(y_{it+1}|\mathbf{Y}_i^t, \mathbf{x}_i^t, \eta_i^*) = E(y_{it+1}|y_i^t, \mathbf{x}_i^t, \eta_i^*) \text{ for } t = 0, ..., T \tag{5}$$

In model 2, this takes the form

$$H_0 : \mathbf{C}_{1,ym} = \mathbf{C}_{1,yf} = ... = \mathbf{C}_{P,ym} = \mathbf{C}_{P,yf} = 0. \tag{6}$$

Chamberlain (1984) defines (3) and (5) as tests for "Granger causality conditional on unobservables."Adams et al. (2003) perform their tests for non-causality without conditioning on η_i^*, i.e., they test the null hypothesis

$$H_0 : E(\mathbf{h}_{it+1}|\mathbf{Y}_i^t, \mathbf{x}_i^t) = E(\mathbf{h}_{it+1}|\mathbf{h}_i^t, \mathbf{x}_i^t) \text{ for } t = 0, ..., T. \tag{7}$$

As Adams et al. (2003) emphasize, rejecting this null hypothesis only leads to the conclusion that y "Granger causes" $\mathbf{h}$ under the maintained hypothesis that there is no unobserved heterogeneity.

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

$$E(\Delta\varepsilon_{it}^*|\mathbf{Y}_i^{t-2}) = 0 \text{ for } t = 2, ..., T \tag{8}$$

using the reduced form VAR in (2). 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 $\Delta\varepsilon_{it}^* = (\varepsilon_{it}^* - \varepsilon_{it-1}^*)$, implying that $\mathbf{Y}_{it-1}$ will not be a valid instrument in the equation in first differences. This is why the history up to $t-2$, $\mathbf{Y}_i^{t-2}$, is used as instruments (following, for example, Arellano and Bond, 1991). This implies that estimation (and testing for health-wealth or wealth-health effects) in this framework requires at least three observations per household.

If the health and wealth variables are close to non-stationary, then the instruments in (8) may be weak since past levels will not be correlated with current changes (see, e.g., Arellano, 2003). This may well be the case for health since, for example, "onsets ever had" enter the constructed health index. Blundell and Bond (1998) suggest using an assumption of mean stationarity on errors and individual effects to add more moments and improve the efficiency of the estimator. Mean stationarity of (2) implies moments of the form[7]

$$E(\Delta\varepsilon_{it-1}^* \otimes (\eta_i^* + \varepsilon_{it}^*)) = 0 \tag{9}$$

which can be re-expressed as

$$E(\Delta\varepsilon_{it-1}^* \otimes \eta_i^*) + E(\Delta\varepsilon_{it-1}^* \otimes \varepsilon_{it}^*) = 0. \tag{10}$$

Sufficient for this assumption is that $E(\varepsilon_{it}^*\eta_i^{*\prime})$ does not depend on t (or is zero) and that there is no serial correlation in ε_{it}^*. The latter assumption was made already in (8) and is justified if all correlation over time is picked up by the AR(P) structure (the matrix $\mathbf{\Phi}$) and the unobserved heterogeneity terms. The former implies that heterogeneity can be related to health or wealth shocks, but only in a way that does not vary over time.

As discussed in Arellano (2003), the assumption (9) given above leads to moment conditions that are non-linear in the parameters. Under the additional assumption

$$E(\Delta\mathbf{x}_{it} \otimes \eta_i^*) = 0, \tag{11}$$

[7] $\otimes$ denotes the Kronecker product. For two matrices A, B, of size $M \times N$ and $P \times Q$, $A \otimes B$ denotes the $MP \times BQ$ matrix consisting of the scalar product of each element of A : A_{mn} by the matrix B.

(9) can be replaced by:

$$E(\Delta \mathbf{Y}_{it-1} \otimes (\eta_i^* + \varepsilon_{it}^*)) = 0 \text{ for } t = 2, ..., T \tag{12}$$

Using (2), this leads to the following moments that are linear in the reduced form parameters $\mathbf{B}$ and $\mathbf{C}_1, ..., \mathbf{C}_P$:

$$E(\Delta \mathbf{Y}_{it-1} \otimes (\mathbf{Y}_{it} - \mathbf{B}\mathbf{x}_{it} - \sum_{p=1}^{P} \mathbf{C}_p \mathbf{Y}_{it-p})) = 0 \text{ for } t = 2, ..., T \tag{13}$$

The additional assumption (11) seems 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).[8] We refer to (13) in addition to (8) as the reduced form VAR. 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 $\mathbf{\Gamma}$ and the lagged effects in $\mathbf{\Phi}$ similar to the restrictions in (4). To be precise, non-causality of wealth to husband's health and wife's health implies:

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

and

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

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 causality 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 $\mathbf{\Gamma}$ and $\mathbf{\Phi}$. Exclusion restrictions (i.e., instruments) are needed in order to identify the instantaneous causal mechanisms.

Our strategy for finding instruments for health and wealth is to look for shocks that do not have direct effects on the other outcome. This same strategy has also been used recently by Smith (2003), Wu (2003), and Meer et al. (2003). As instruments for health

[8] In principle, (13) would also identify the effects of time-invariant exogenous variables. Following Alonso-Borrego and Arellano (1999), however, we do not exploit this and do not include the time-invariant exogenous variables $\mathbf{x}_{it}$.

changes, we use onsets of critical health conditions. It was already documented in tables 3 and 4 that such onsets are abundant for this cohort. It seems plausible that these onsets have no direct effect of wealth (other than through the health change they induce). 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 it an appropriate instrument for wealth changes.

To identify the parameters of $\mathbf{\Gamma}$, we therefore use the following moments

$$E(\Delta\varepsilon^*_{yit}\mathbf{z}^h_{it}) = 0 \ , \ E(\Delta\varepsilon^*_{hit}\mathbf{z}^y_{it}) = 0 \tag{16}$$

Here $\mathbf{z}^h_{it} = (\mathbf{z}^{h\prime}_{mit}, \mathbf{z}^{h\prime}_{fit})'$ are indicators of onsets of health conditions for both spouses. We use separate dummies for severe and mild onsets. $\mathbf{z}^y_{it}$ is a vector with two elements: whether or not the couple received an inheritance in the last two years, and the size of that inheritance in dollars.

To identify the instantaneous effect of health of one spouse on health of the other spouse, we also use the onsets of health conditions. We thus make the plausible assumption that such an onset has no direct effect on the other spouse other than through the constructed health index. We will test the overidentifying restrictions it implies. The additional moments are given by:

$$E(\Delta\varepsilon^*_{mit}\mathbf{z}^h_{fit}) = 0 \ , \ E(\Delta\varepsilon^*_{fit}\mathbf{z}^h_{mit}) = 0. \tag{17}$$

To identify the structural VAR as defined in (1) we therefore use moments of the form $E(\Delta\varepsilon^*_{it} \otimes \mathbf{Y}_i^{t-2}) = 0$ along with (16), (17) and stationarity restrictions (13). We denote this model the structural VAR. Similarly to the reduced-form VAR, we can use the incremental Sargan test to test the stationarity restrictions. Tests for "lagged" causality essentially remain the same as (3) and (5) except that they involve the matrices $\mathbf{\Phi}_p$ instead of $\mathbf{C}_p$. Tests for contemporaneous causation that rely on orthogonality restrictions (16) and (17) involve testing whether elements of $\mathbf{\Gamma}$ are zero.

Both reduced form VARs and structural VARs are estimated by GMM using moments in levels and differences (Blundell and Bond, 1998). Since the cross-sectional dimension is quite large (compared to, e.g., Arellano and Bond, 1991), we use two-step GMM estimates constructing the optimal weighting matrix from first-step estimates.

4 Adams et al. (2003) Tests

We first follow the approach of Adams et al. (2003) to test for non-causality of wealth on health and health on wealth of couples in the HRS cohort without controlling for unobserved heterogeneity and using only first order lags. Comparing this with the results of causality tests conditioning on unobserved heterogeneity will show whether controlling for unobserved heterogeneity is important.

4.1 Wealth to Health

To test the null hypothesis that wealth does not cause health, Tables 7 and 8 present the results of models that explain each indicator of health of husband and wife from lagged husband's and wife's health, lagged log wealth, and additional controls (demographics and past risk behavior, as in Adams et al., 2003).[9] We model such variables as ADLs, CESD scores and Self-reported Health as ordered responses, onsets as binary outcomes, and constructed health indices and self-reported probabilities of dying before age 75 as continuous outcomes. As in Adams et al. (2003), normality of the errors is assumed for the binary and ordered response models and the invariance property (the causal effect is constant over time) is imposed.

The non-causality test is a t-test on the coefficient of lagged log wealth. For husbands, the null is rejected in seven out of eight cases. In six of these, the coefficient is significantly negative, implying that higher wealth leads to fewer health problems, as expected. The significantly positive effect of wealth on the probability of dying before reaching age 75 seems counter-intuitive. The effect of wealth on the probability of a severe onset is negative but not significant.

The results for the wife's health are presented in Table 8. The effect of lagged wealth is always negative and significant in four out of eight cases. Focusing on the constructed health index as a summary measure of all health variables, we find evidence of wealth causation for both husbands and wives, in line with the results of Adams et al. (2003) for the older cohorts. Although statistically significant, the magnitude of the effects is quite small. For example, having twice as much lagged wealth would reduce the probability of a severe onset for wives by about 0.4 percentage-points, keeping other variables constant.

Tables 7 and 8 can also be used to test for non-causality of the wife's health on the husband's health and vice versa (controlling for household wealth etc.). This is a t-test on the coefficient of the spouse's health. In four out of eight cases, we find a positive and significant effect of the wife's health on the husband's health. In the other four cases, the effect is insignificant. The effect of the husband's health on the wife's health is significantly positive in five out of eight cases. Focusing on the constructed health index, we find evidence of causality in both directions.

[Insert Tables 6 and 7 about here]

4.2 Health to Wealth

Table 9 presents the regressions underlying tests for non-causality of health to wealth. Both log wealth and the hyperbolic transformation of wealth proposed by Adams et al. (2003) are used. The column "levels" presents the OLS results. To account for outliers in the log wealth distribution, we also present some robust regression results. Although this leads to somewhat lower t-values, the main conclusion remains the same: health

[9] We experimented with more specifications, more lagged health variables, etc. In general, the test results do not change much and qualitative conclusions remain the same.

problems of both husband and wife lead to a significant reduction in household wealth, so that non-causality from health to wealth is clearly rejected, in line with the results of Adams et al. (2003).

[Insert Table 9 about here]

4.3 Attrition and Invariance

We know from tables 2 and 4 that those who die or for other reasons leave the panel before 2002 have poorer health outcomes and on average lower schooling (specially husbands) – an indicator of lower socioeconomic status. This suggests that attrition may also affect the relation between health and wealth outcomes and the outcomes of the causality tests.

Comparing estimates of the equations explaining the health index for the unbalanced sample and for the balanced sample consisting of those who remain alive and in the panel until 2000 enables to test for non-random attrition (cf. Nijman and Verbeek, 1996). Since under the null of ignorable attrition the estimates from the unbalanced sample are efficient and those from the balanced panel are consistent, while both would be inconsistent under the alternative, a generalized Hausman test can be performed. Since we are particularly interested in the causal effect of wealth in this equation, we performed the Hausman test on the lagged wealth coefficient only. We find that the two estimates for males are similar (-0.003 (balanced) vs -0.005 (unbalanced)) and marginally reject the null (Chi-sq.[1] = 4.26, p-value = 0.039). Similarly for wives the coefficients are -0.0046 (balanced) and -0.0056 (unbalanced) and there is no evidence for selective attrition (Chi-sq.[1] = 0.72, p-value = 0.395).

These tests thus all suggest that attrition is mostly ignorable for the parameters of interest – the causal effects from wealth to health. In what follows, we will report estimates from the unbalanced samples. The results using the balanced samples are always qualitatively similar.

Adams et al. (2003) pay a lot of attention to testing whether causal effects are invariant over time, although Poterba (2003) casts some doubt on the importance of this issue. We tested whether the relationship between health and wealth was stable over time. For husbands, the coefficients on lagged wealth (in a test for non-causality to the CHI) varies from -0.0124 to 0.0014 in 2002. Some evidence is provided that the effects are not the same (Chi-square(4) = 12.83, p-value = 0.012) while the equality is not rejected for wives (Chi-square(4) = 4.18, p-value = 0.382). In fact, the parameters on lagged wealth for husbands appear to be decreasing over time, which could be a combined effect of attrition and invariance if differential mortality considerably reduces the variance of health and wealth outcomes (see Attanasio and Hoynes (2000) for evidence on differential mortality).

5 Causality Tests in Dynamic Panel Data models

To estimate the reduced form and structural VARs we use the generalized method of moments with robust two-step estimates (see for example Arellano and Bond, 1991). In order to incorporate mean stationarity restrictions, we use the combination of level and difference moments of Blundell and Bond (1998) discussed in Section 3.2. These additional moments in levels were not rejected by incremental Sargan tests. We include time dummies to pick-up unobserved trends in the components determining the gradient and, where necessary as indicated by specification tests rejecting invariance, interactions with time dummies to account for changing relationships over time. Efficiency gains can be realized by estimating all equations of the VAR system together if the optimal weighting matrix is not diagonal (Alonso-Borrego and Arellano, 1999). However, the finite-sample properties of the estimator may deteriorate. We therefore estimate them separately. We experimented with several lag structures and found that models with two lags were needed to pass the usual specification tests (the Sargan test on overidentifying restrictions and the test on second order autocorrelation in the differenced residual; see Arellano and Bond, 1991). The results for the selected models are presented in Tables 10, 11 and 12. In each case, we present a reduced form equation without instantaneous effects of wealth on health or vice versa, and a structural form equation in which the instruments in section 3.2 are used to identify instantaneous effects of endogenous variables.

5.1 Health to Wealth

Table 10 presents the results for equations explaining log household wealth. For these selected models, overidentifying restrictions are marginally 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 that the errors in levels are uncorrelated over time).

The reduced form estimates imply a significant negative effect of both lagged husband's health and lagged wife's health on log wealth. Joint tests indicate that lagged values of husband's health significantly affect log wealth, rejecting the hypothesis that husband's health causes does not cause wealth. This is the same conclusion as from the Adams et al. (2003) tests in the previous section, but now unobserved heterogeneity is controlled for and the lag structure is richer, chosen on the basis of specification tests. The wife's health also affects log wealth but this effect is significant only at the 3% level.

The structural estimates confirm the evidence of health causation. There is no evidence for an immediate effect of husband's health, and the effects of the lagged husband health variables are similar to those in the reduced form equation. The joint significance remains, confirming the conclusion that husband's health causes wealth. Current and lagged variables on the wife's health are jointly significant at any reasonable significance level. The immediate negative effect dominates, and the conclusion that health problems of the wife cause negative wealth changes is stronger than in the reduced form. Thus, overall, we can conclude that the results of the Adams et al. tests on health wealth causation were not just a consequence of unobserved heterogeneity - strong evidence

remains in a model that controls for this. 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 the wife's health and a lagged effect for husbands. This may also 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.

[Insert Table 10 about here]

5.2 Wealth to Health

The results for the equation explaining the husband's health are presented in Table 11. Adding the second order lags and the interaction of lagged health with time was necessary to obtain a model that passes the Sargan test on overidentifying restrictions and the test autocorrelation in the errors. The results provide no evidence whatsoever on wealth health causation for husbands. Both in the reduced form and in the structural equation, the wealth variables are jointly (and individually) insignificant. This result differs from what we found with the Adams et al. (2003) tests in the previous section. The plausible explanation is that rejecting non-causality there was due to the presence of permanent unobserved heterogeneity affecting health and household wealth. These terms are controlled for in Table 11.

Another difference with Table 7 is that we now 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. Unobserved factors that affect husband's and wife's health in the same way are the most plausible explanation for the difference in findings.

Table 12 presents the results for the equations explaining the wife's health. The results are essentially the same as for the husband's health. Other than in the previous section, the models controlling for fixed effects do not provide any evidence of causal effects from wealth on the wife's health or from the husband's health on the wife's health.

5.3 Some Disaggregated Results

We have found clear evidence of causal effects of both the husband's and the wife's health on household wealth, using the constructed health index which incorporates all features of health. Table 13 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 and work-related health and the self-reported probability of dying before age 75 are not included since they capture both mental and physical health features.

The results in Table 13 show evidence of causal household wealth effects of physical health for the husband and mental health for the wife. Mental health is not significant for the husband and physical health is insignificant for wives. 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 what we found earlier.

Further disaggregation is possible by partitioning wealth into liquid and non-liquid wealth, as in Table 3. This shows that for non-liquid wealth, both physical and mental health of both husband and wife are significant, albeit the significance probabilities for the wife are close to 5%. Only mental health of the wife has a significant instantaneous effect, the other causal mechanisms work with lags of two years. For liquid wealth, causal effects are found for mental health of both spouses but not for physical health. Again, the effect of mental health of the wife is instantaneous, that of the husband is not. Detailed results are available upon request.

One explanation for the strong effects of mental health might be the lack of insurance coverage for mental health problems. Indeed these are covered in a limited way by Medicare and Medicaid and therefore employer-provided insurance coverage or other insurance coverage is necessary to protect against those onsets (Adams et al., 2003). Disaggregating by health insurance coverage status does lead to a clear picture. Indeed those wives who do not have employer-provided health insurance tend to be those for which a health shock has a large immediate effect on wealth. Furthermore, the stronger effect of the wife's mental health status than of the husband's 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.

6 Conclusion

In this paper, we compare two ways of testing for causal pathways between health and socioeconomic status using panel data on an elderly US cohort. One follows the methodology of Adams et al. (2003) based upon Granger causality. The second is an extension of this using a dynamic panel data framework. The main difference is that this allows us to control for unobserved heterogeneity, avoiding the problem that rejecting non-causality might be due to ignoring unobserved heterogeneity terms. We use biennial five waves of elderly couples in the HRS, following the 1931-1941 birth cohort over the time period 1992-2000.

While the Adams et al. (2003) suggest causal effects in both directions, from health to wealth and from wealth to health, our dynamic panel data model based tests also provide clear evidence of causal effects from health to wealth, but hardly any evidence of causal effects from wealth to either the husband's or the wife's health. An analysis of the residuals suggests that this difference is not due to unobserved heterogeneity in

health, but to unobserved heterogeneity in wealth or the richer dynamic specification of the dynamic panel data model. Disaggregating health into mental and physical health suggests that both have causal effects on wealth, but 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). Interestingly insurance coverage appears to play a role as suggested by the evidence that uninsured wives who experience onsets of mental conditions tend to spend down household assets more importantly.

We would like to stress that the absence of an active causal link does not mean that it has not operated in the past. Here, we only consider households with at least one spouse 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.

The finding that health - wealth causation (health selection, in the social science literature) is the main driving force for the development of the gradient in this age group confirms evidence of Smith (1999,2003), Adda (2003), Hurd and Kapteyn (2003) and Wu (2003). Particularly for husbands, the long-run effect of a health shock is considerable. This raises an interesting welfare and policy question: Is this drop in wealth planned or is it the consequence of inadequate health insurance? Smith (2003) finds that out-of-pocket medical expenditures can be considerable in the HRS cohort. Therefore, even for individuals with health insurance, there remains considerable risk to insure.

Further research could also incorporate the role of labor force participation and earnings. Other than the AHEAD cohort studied by Adams et al. (2003), the HRS cohort that we consider is typically at work in the first wave that we observe them and has 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.

References

[1] Adams, P., Hurd, M.D., McFadden, D., Merrill, A. and T. Ribiero (2003): Healthy, Wealthy and Wise? Tests for Direct Causal Paths between Health and Socioeconomic Status, *Journal of Econometrics*, 112, pp. 3-56.

[2] Adda J., T. Chandola and M. Marmot (2003): Socioeconomic Status and Health: Causality and Pathways, *Journal of Econometrics*, 112, pp. 57-63.

[3] Adda, J. (2003): Income and Health over the Life-Cycle: A Dynamic Approach to Evaluate the Importance of Selection and Social Causation, mimeo University College London.

[4] Adler, N.E., Boyce, T., Cohen, S., Folkman, S., Kahn, R.L. and S.L. Syme (1994): Socioeconomic Status and Health: The Challenge of the Gradient, *American Psychologist*, 49(1), pp. 15-24.

[5] Alonso-Borrego, C. and M. Arellano (1999): Symmetrically Normalized Instrumental-Variable Estimation using Panel Data, *Journal of Business and Economic Statistics*, 17, pp. 36-49.

[6] Arellano, M. (2003): *Panel Data Econometrics*, Oxford University Press, Oxford.

[7] Arellano, M. and S. Bond (1991): Some Tests of Specification for Panel Data: Monte Carlo Evidence and an Application to Employment Equations, *Review of Economic Studies*, 58, pp. 277-297.

[8] Attanasio, O.P. and H. Williamson Hoynes (2000): Differential Mortality and Wealth Accumulation, *Journal of Human Resources*, 35:1, pp. 1-29.

[9] Barker, D.J.P (1997): Maternal Nutrition, Fetal Nutrition and Diseases in Later Life, *Nutrition*, 13:9, pp.807-13.

[10] Blau, D.M. and D. Gilleskie (2001): Health Insurance and Retirement of Married Couples, Mimeo, University of North Carolina at Chapel Hill, October.

[11] Blundell, R.and S. Bond (1998): Initial Conditions and Moment Restrictions in Dynamic Panel Data Models, *Journal of Econometrics*, 87, pp. 115-143.

[12] Chamberlain, G. (1984): Panel Data, in *Handbook of Econometrics*, Vol 2, eds. Z. Griliches and M.D. Intriligator, Elsevier Science, Amsterdam, chapter 22.

[13] Deaton, A.S. and C. Paxson (1998): Aging and Inequality in Income and Health, *American Economic Review Papers and Proceedings of the 110th AEA Annual Meeting*, 88:2, pp. 248-253.

[14] Dustmann and Windmeijer (1999): Wages and the Demand for Health: A Life-Cycle Analysis, IFS Discussion paper W99/20.

[15] Granger, C.W.J. (1969): Investigating Causal Relations by Econometric Models and Cross-Spectral Methods, *Econometrica*, 37, pp. 424-438.

[16] Grossman, M. (1972): On the Concept of Health Capital and the Demand for Health, *Journal of Political Economy*, 80:2, 223-255.

[17] Gruber, J. (1998): Health Insurance and the Labor Market, NBER Working Paper 6762.

[18] Hoynes, H., Hurd, M. and H. Chand (1998): Household Wealth of the Elderly under Alternative Imputation Procedures, in *Inquiries in the Economics of Aging*, David Wise, ed., University of Chicago Press, Cicago, pp. 229-257.

[19] Hurd, M.D. (2001): Portfolio Holdings of the Elderly, in Household Portfolios chapter 11, eds L.Guiso, M. Haliassos and T. Jappelli, pp. 431-472.

[20] Hurd, M.D. and A. Kapteyn (2003): Health, Wealth and the Role of Institutions, *Journal of Human Resources* 38:2, pp. 386-415.

[21] Kenkel, D.S. (1991): Health Behavior, Health Knowledge and Schooling, *Journal of Political Economy*, 99:2, pp.287-305.

[22] Lindeboom, M., Portrait, F. and G. van den Berg (2003): Individual Mortality and Macro-Economic Conditions from Birth to Death, Discussion Paper Tinbergen Institute 03-072/3, Amsterdam.

[23] Marmot, M. (1999): Multi-Level Approaches to Understanding Social Determinants, in *Social Epidemiology.* Berkman, L. and I. Kawachi eds, Oxford University Press, Oxford.

[24] Meer, J., Miller, D.L. and H.S. Rosen (2003): Exploring the Health-Wealth Nexus, *Journal of Health Economics*, 22, pp. 713-730.

[25] Mundlak, Y. (1961): Empirical Production Function Free of Management Bias, *Journal of Farm Economics*, 43, 44-46.

[26] Newhouse, J. (1993): *Free for All*, Harvard University Press, Cambridge (Mass.).

[27] T.E. Nijman and M. Verbeek (1996): Incomplete Panels and Selection Bias, in *The Econometrics of Panel Data: Handbook of Theory and Applications* (Second Revised Edition), L. Mátyás and P. Sevestre, (eds.), Kluwer Academic Publishers, Dordrecht, 449-490.

[28] Pradhan, M., Sahn, D. and S.D. Younger (2003): Decomposing World Health Inequality, *Journal of Health Economics*, 22, pp. 271-293.

[29] Ravelli, A.C.J., van der Meulen J.H.P., Michels, R.P.J., Osmond, C., Barket D.J.P., Hales C.N. and O.P. Bleker (1998): Glucose Tolerance in Adults after Prenatal Exposure to Famine, *Lancet*, 351, pp. 173-76.

[30] Rust, J. and C. Phelan (1997): How Social Security and Medicare affect Retirement Behavior in a World of Incomplete Markets, *Econometrica*, 64:4, pp. 781-831.

[31] Seeman, T., Singer, B., Rowe, J., Horwitz, R. and B. McEwen (1997): Price of Adaption-Allostatic Load and its Health Consequences, *Archives of Internal Medicine*, 157:19, pp.2259-2268.

[32] Sims, C.A. (1972): "Money, Income, and Causality", *American Economic Review*, 62: pp. 540-552.

[33] Smith, J.P. (1999): Healthy Bodies and Thick Wallets: The Dual Relation between Health and Economic Status, *Journal of Economic Perspectives*, 13:2, pp. 145-166.

[34] Smith, J.P. (2003): Consequences and Predictors of New Health Events, mimeo, RAND, Santa Monica.

[35] Wadsworth, M.E.J. and D.J.L. Kuh (1997): Childhood Influences on Adult Health: A Review of Recent Work from the British 1946 National Birth Cohort Study, the MRC National Survey of Health and Development, *Pediatric and Perinatal Epidemiology*, 11, pp. 2-20.

[36] Wilkinson, R.G. (1996): *Unhealthy Societies: The Afflictions of Inequality*, Routledge, London.

[37] Wu, S. (2003): The Effects of Health Events on the Economic Status of Married Couples, *Journal of Human Resources*, 38:1, 219-230.

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

Table 1: Sample Composition : Observations per wave along with exits that occur in between this wave and the following wave are reported. The percentage of those present in a wave exiting prior to the next interview is also reported in the second column.

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 Not first 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 Not first marriage (%)	0.243	0.353	0.256	0.281	0.273	0.312
Number of couples	2463	419	355	335	278	310
Total number of observations	14 778	419	710	1005	1112	1550

Table 2: Demographic Characteristics: Characteristics in 1992 by period of exit from the panel.

median (participation %¿0)	% imputed	1992	2002
Liquid wealth		38.81 (74.1)	80.0 (47.8)
IRAs	21.6	31.57 (45.1)	68.24 (47.2)
Stocks	11.2	25.24 (32.1)	50.0 (37.4)
Bonds	3.4	12.62 (6.9)	35.0 (8.6)
Checking/savings account	18.8	6.56 (86.2)	10.0 (89.1)
Cert./T.bills/Sav.Bds	7.3	10.10 (29.5)	20.0 (26.9)
Debt (substracted)		3.78 (41.0)	5.0 (28.2)
Non-liquid wealth		103.51 (97.2)	136.0 (99.0)
Primary residence	4.3	100.98 (87.9)	130.0 (92.3)
Mortgage (subtracted)	3.5	37.87 (50.6)	56.0 (35.5)
Other real estates	5.1	56.80 (27.4)	70.0 (21.3)
Business assets	4.6	88.36 (15.2)	150.0 (13.4)
Trans./vehicules assets	10.2	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

Table 3: Composition of Household Wealth. All wealth figures are in thousands USD 2002 using the BLS CPI index. The percentage imputed is reported. Ownership (participation) is in brackets. Other debts or loans are not shown in the table but enter negatively the calculation of liquid wealth. Business assets are not included when computing non-liquid and total wealth. Note that the highest 1affect the calculation of the median

Health Indicators	Husbands		Wives	
mean/fraction reported	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
ADLs (¿0)	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
P(death before 75)	0.365	0.326	0.334	0.329
Health limits work	0.213	0.263	0.179	0.239
Number of respondents	4160	2463	4160	2463

Table 4: Health of Husbands and Wives. Cells report mean or pct of each health variable. The first column for each spouse (1992 all) refers to the whole sample in 1992 while the second column reports the same statistics for respondents in 2002. ADLs refer to limitations in performing activities of daily life while CESD scores are on a scale of 0 to 8 and record affirmative questions to a set of questions on mental health (10 bads and 2 goods which are substracted).

Health 1992	Wife (from good to bad health)				
Husband	1st qtile	2nd qtile	3rd qtile	4th qtile	Total
1st qtile	392	296	205	163	1056
	(0.09,0.36)	(0.06,0.25)	(0.06,0.25)	(0.03,0.13)	
2nd qtile	290	295	236	207	1028
	(0.07,0.29)	(0.07,0.28)	(0.06,0.24)	(0.05,0.20)	
3rd qtile	222	268	255	291	1036
	(0.05,0.21)	(0.06,0.26)	(0.06,0.24)	(0.07,0.30)	
4th qtile	140	234	294	372	1040
	(0.04,0.14)	(0.05,0.21)	(0.07,0.27)	(0.10,0.38)	
Total	1044	1093	990	1033	4160
Chi-square = 254.77 (df=15)					

Table 5: Distribution of Household Health Indices in 1992. 1st row for each quartile report actual frequencies with underneath the cell relative frequency and the conditional relative frequency within each quartiles. The constructed health indices of both spouses in 1992 are used. Individuals in high quartiles are in worse health compared to those in the lower quartiles. The chi-square test of independence has 15 degrees of freedom.

	Median wealth	
Husband Health Index	1992	2002
best quartile	176.7	285.4
2nd quartile	151.2	225.4
%Δ w.r.t. 1st	-14.4%	-21.0%
3rd quartile	127.5	185.6
%Δ w.r.t. 1st	-27.8%	-35.0%
worst quartile	86.3	113.5
%Δ w.r.t. 1st	-51.2%	-60.2%
Wife Health Index	1992	2002
best quartile	203.9	326.3
2nd quartile	162.6	248.0
%Δ w.r.t. 1st	-20.3%	-24.0%
3rd quartile	111.8	176.3
%Δ w.r.t. 1st	-45.2%	-46.0%
worst quartile	83.3	108.5
%Δ w.r.t. 1st	-59.1%	-66.7%

Table 6: The Wealth-Health Gradient . Cells give median wealth in thousands 2002 USD, while the second row gives the percentage difference with respect to the 1st quartile of the health distribution of the spouse in 1992. Total household wealth and constructed health indices (CHI) are used

Husbands (over all waves)				
Covariates	Health Index	Severe Onsets	Mild Onsets	Self-Report
y_{t-1}	-0.005	-0.006	-0.008	-0.008
	(-3.33)	(-1.54)	(-2.57)	(-2.59)
h^m_{t-1}	0.867	0.068	-0.019	0.893
	(129.9)	(4.08)	(-1.31)	(52.16)
h^f_{t-1}	0.021	0.023	0.018	0.035
	(3.85)	(1.31)	(1.27)	(2.61)
Invariance	12.83**	Attrition	4.26**	
Covariates	CESD score	ADLs	Hazard 75	Hlth limits work
y_{t-1}	-0.014	-0.011	0.196	-0.010
	(-4.78)	(-2.64)	(2.39)	(-2.66)
h^m_{t-1}	0.476	0.578	12.845	0.789
	(32.82)	(30.86)	(32.68)	(36.97)
h^f_{t-1}	0.058	0.052	-0.114	-0.006
	(3.84)	(2.65)	(-0.29)	(-0.35)

Table 7: Non-Causality: Wealth to Husband Health: Point estimates of lagged health and wealth are reported with t-values in parenthesis. Models for the health index, self-reported hazard are estimated by OLS, for Self-reported health, CESD scores and ADL count, ordered probits. and for the remaining variables, probits where errors are clustered at the couple level. Controls are included for demographics and lagged risk factors as well as dummy variables. The complete results are available upon requests

Wives (over all waves)				
Covariates	Health Index	Severe Onsets	Mild Onsets	Self-Report
y_{t-1}	-0.006	-0.012	-0.006	-0.006
	(-3.52)	(-3.03)	(-2.04)	(-1.80)
h^m_{t-1}	0.026	0.017	0.021	0.049
	(4.74)	(0.88)	(1.47)	(3.67)
h^f_{t-1}	0.860	0.166	-0.037	0.917
	(129.69)	(9.81)	(2.46)	(49.4)
Invariance	4.18	Attrition	0.72	
Covariates	CESD score	ADLs	Hazard 75	Hlth limits work
y_{t-1}	-0.005	-0.004	-0.040	-0.007
	(-2.00)	(-1.12)	(-0.47)	(-1.55)
h^m_{t-1}	0.067	0.021	0.944	0.041
	(4.93)	(1.02)	(2.48)	(2.15)
h^f_{t-1}	0.480	0.659	11.71	0.765
	(34.34)	(35.07)	(26.73)	(34.63)

Table 8: Non-Causality: Wealth to Wife Health: Point estimates of lagged health and wealth are reported with t-values in parenthesis. Models for the health index, self-reported hazard are estimated by OLS, for Self-reported health, CESD scores and ADL count, ordered probits. and for the remaining variables, probits where errors are clustered at the couple level. Controls are included for demographics and lagged risk factors as well as dummy variables. The complete results are available upon requests

	log total wealth		hyper. total wealth
Covariates	levels	robust	levels
y_{t-1}	0.413	0.913	0.787
	(18.63)	(704.36)	(103.79)
husband			
h^m_{t-1}	-0.143	-0.012	-0.004
	(-4.02)	(-2.31)	(-4.74)
Wife			
h^f_{t-1}	-0.258	-0.024	-0.007
	(-6.56)	(-4.39)	(-8.24)

Table 9: Non-Causality: Health to Wealth: Point estimates and t-values are reported for regressions in levels (errors clustered at household level), robust regressions applying Huber weights and within (fixed-effects) regressions.The same set of controls as for the wealth non-causation tests are included. The complete results are available upon requests for total, non-liquid and liquid wealth.

	Unrestricted		Structural	
Covariates	Pe	T-stat	Pe	T-stat
y_{t-1}	0.157	2.86	0.154	2.86
y_{t-2}	0.034	1.48	0.029	1.29
$y_{t-1}\times t$	-0.017	-1.00	-0.016	-0.94
h_t^m	-		-0.163	-1.05
h_{t-1}^m	-0.636	-4.35	-0.517	-3.05
h_{t-2}^m	-0.275	-3.38	-0.236	-2.62
$h_{t-1}^m\times t$	0.081	2.60	0.081	2.63
h_t^f	-		-0.499	-2.54
h_{t-1}^f	-0.388	-2.63	-0.129	-0.70
h_{t-2}^f	0.007	0.01	0.108	1.31
$h_{t-1}^f\times t$	0.025	0.76	0.034	1.01
	χ^2	p-val	χ^2	p-val
Sargan test	45.61	0.04	46.71	0.04
Ar(2) test on residuals	-0.091	0.99	-0.02	0.99
Causality tests				
$h_{t-1}^m, t\times h_{t-1}^m, h_{t-2}^m$	9.88	0.00		
$h_{t-1}^f, t\times h_{t-1}^f, h_{t-2}^f$	5.19	0.00		
$h_t^m, h_{t-1}^m, t\times h_{t-1}^m, h_{t-2}^m$			7.31	0.00
$h_t^f, h_{t-1}^f, t\times h_{t-1}^f, h_{t-2}^f$			6.12	0.00
h_t^m, h_t^f			4.37	0.01
N	3386		3386	

Table 10: Dynamic Model for Health Causation: Two-step point estimates and t-values are reported. All estimation is done by system GMM making use of moments in levels.

	Unrestricted		Structural	
Covariates	Pe	t-stat	Pe	t-stat
h^m_{t-1}	0.596	12.91	0.602	12.56
h^m_{t-2}	0.279	12.29	0.282	12.25
h^m_{t-3}	0.106	4.74	0.106	4.73
$h^m_{t-1}\times t$	0.006	0.54	0.005	0.45
h^f_t			0.044	0.86
h^f_{t-1}	0.037	1.03	0.018	0.41
h^f_{t-2}	-0.002	-0.06	-0.012	-0.51
$h^f_{t-1}\times t$	-0.006	-0.81	-0.008	-0.97
y_t			0.007	0.28
y_{t-1}	-0.002	-0.22	-0.002	-0.20
y_{t-2}	-0.003	-0.76	-0.004	-0.89
$y_{t-1}\times t$	-0.001	-0.17	-0.001	-0.24
	χ^2	p-val	χ^2	p-val
Sargan test	18.16	0.58	20.94	0.524
Causality tests				
$h^f_{t-1}, t\times h^f_{t-1}, h^f_{t-2}$	0.41	0.58		
$y_{t-1}, t\times y_{t-1}, y_{t-2}$	0.27	0.84		
$h^f_t, h^f_{t-1}, t\times h^f_{t-1}, h^f_{t-2}$	.	.	0.52	0.71
$y_t, y_{t-1}, t\times y_{t-1}, y_{t-2}$	.	.	0.26	0.90
N	3051		3051	

Table 11: Dynamic Model for Wealth Causation on Husband Health: Two-step point estimates and t-values are reported. All estimation is done by system GMM making use of moments in levels.

	Unrestricted		Structural	
Covariates	Pe	t-stat	Pe	t-stat
h^f_{t-1}	0.546	34.82	0.541	12.13
h^f_{t-2}	0.266	12.55	0.265	12.41
h^f_{t-3}	0.081	3.66	0.082	3.56
$h^f_{t-1}\times t$	0.009	0.97	0.009	0.89
h^m_t	.	.	0.024	0.58
h^m_{t-1}	0.060	1.54	0.026	0.51
h^m_{t-2}	0.002	0.12	-0.015	-0.67
$h^m_{t-1}\times t$	-0.011	-1.29	-0.009	-1.06
y_t	.	.	-0.034	-1.25
y_{t-1}	0.008	0.83	0.007	0.59
y_{t-2}	-0.002	0.47	-0.001	-0.16
$y_{t-1}\times t$	-0.001	-0.48	-0.001	-0.08
	χ^2	p-val	χ^2	p-val
Sargan test	29.89	0.07	30.09	0.12
Causality tests				
$h^m_{t-1}, t\times h^m_{t-1}, h^m_{t-2}$	0.86	0.46		
$y_{t-1}, t\times y_{t-1}, y_{t-2}$	0.78	0.51		
$h^m_t, h^m_{t-1}, t\times h^m_{t-1}, h^m_{t-2}$	.	.	0.51	0.72
$y_t, y_{t-1}, t\times y_{t-1}, y_{t-2}$	.	.	0.98	0.41
N	3051		3051	

Table 12: Dynamic Model for Wealth Causation on Wife Health: Two-step point estimates and t-values are reported. All estimation is done by system GMM making use of moments in levels.

GMM/sys AR2	Estimates		Causality test	
Covariates	Pe	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_t^m	-0.616	-1.42		
h_{t-1}^m	-0.031	-0.13		
h_{t-2}^m	0.051	0.41		
$h_{t-1}^m\times t$	0.062	1.64	2.18	0.082
h_t^f	-1.125	-2.95		
h_{t-1}^f	0.298	1.55		
h_{t-2}^f	0.229	2.33		
$h_{t-1}^f\times t$	0.053	1.54	3.21	0.022
Physical Health				
h_t^m	0.139	0.91		
h_{t-1}^m	-0.484	-2.81		
h_{t-2}^m	-0.127	-1.27		
$h_{t-1}^m\times t$	0.093	2.90	3.09	0.026
h_t^f	0.051	0.27		
h_{t-1}^f	-0.378	-1.93		
h_{t-2}^f	-0.060	-0.56		
$h_{t-1}^f\times t$	0.001	0.04	1.83	0.139

Table 13: Wealth responses to Mental and Physical Healh: Two-step point estimates and t-values are reported. All estimation is done by system GMM making use of moments in levels. The mental health. index is composed of CESD scores and onsets of psychic or mental health condition while the physical health index includes onsets of severe and mild conditions as well as ADLs