NBER WORKING PAPER SERIES

EXPLORING THE HEALTH-WEALTH NEXUS

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Working Paper 9554
http://www.nber.org/papers/w9554

NATIONAL BUREAU OF ECONOMIC RESEARCH
1050 Massachusetts Avenue
Cambridge, MA 02138
March 2003

We are grateful to Princeton's Center for Economic Policy Studies for support. Doug Miller acknowledges financial support from the Robert Wood Johnson Foundation. We thank Bo Honoré, Erica Field, Adriana Lleras-Muney, and participants in Princeton's Public Finance Working Group for useful comments, and Cynthia Cohen for assistance in the preparation of the manuscript. The views expressed herein are those of the authors and not necessarily those of the National Bureau of Economic Research.

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NBER Working Paper No. 9554
March 2003
JEL No. I12

ABSTRACT

The casual links between health and economic resources have long concerned social scientists. We use four waves of data from the Panel Study of Income Dynamics to analyze the impact of wealth upon an individual's health status. The difficulty in approaching this task that has bedeviled previous studies is that wealth may be endogenous; a priori, it is just as likely that changes in health affect wealth as vice versa. We argue that inheritance is a suitable instrument for the change in wealth, and implement a straightforward instrumental variables strategy to deal with this problem. Our results suggest that the causal relationship running from wealth to health may not be as strong as first appears. In the data, wealth exerts a positive and statistically significant effect on health status, but it is very small in magnitude. Instrumental variables estimation leaves the point estimate approximately the same, but renders it insignificantly different from zero. And even when the point estimate is increased by twice its standard error, the quantitative effect is small. We conclude that the wealth-health connection is not driven by short run changes in wealth.

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1. INTRODUCTION

The existence of a strong positive correlation between health and economic resources is well documented. However, the direction of causation remains an open issue. One possibility is that economic resources affect health (Ettner [1996], Smith [1999]); individuals with more wealth can afford better medical care, live in healthier environments, and so on. Another is that health affects economic resources. Healthier individuals may be able to work more than those who are ill, enabling them to accumulate more wealth (McClellan [1998], Levy [2000], Wu [2003]). Finally, third factors may determine both health and economic resources. For example, individuals with a low rate of time preference may undertake investments in human capital that enhance future earnings as well as engage in behaviors that improve future health (Barsky *et al.* [1997]).

As Attanasio and Hoynes [2000] note, the nature of this relationship is central to economists' understanding of life cycle wealth accumulation, and to the interpretation of cohort based age-wealth profiles. Further, it is directly relevant to the public policy debate over health care. In particular, it is widely assumed that there is a causal link running from wealth to health, and that, as a consequence, the key to improving health status is to transfer income to the poor. For example, in a report on a conference on income inequality and health entitled "Dollars Count More Than Doctors," Lefkowitz [2000] argues that "[s]mall differences in socioeconomic status matter to health. In other words, there are significant gains to be made with relatively moderate spending."

However, in light of the possible endogeneity of wealth, such inferences based on the correlation between changes in wealth and changes in health are not compelling. Establishing a causal link requires an appropriate instrumental variables strategy and, to our

knowledge, this has not been done. We propose to deal with endogeneity by using inheritance as an instrumental variable for changes in wealth. Receipt of an inheritance is clearly correlated with the change in an individual's wealth, but is plausibly unrelated to changes in his or her health, conditional on initial health status.

Section 2 examines some of the previous literature on the subject of the connection between economic resources and health status, with a focus on the problem of causal inference. Section 3 describes the data, which are drawn from four waves of the Panel Study of Income Dynamics (PSID) spanning the years 1984 to 1999. In Section 4, we specify the econometric model and present the results. To begin, we estimate the relationship between changes in health and changes in wealth without taking endogeneity into account, and replicate the previous findings that changes in wealth have a positive and statistically significant effect on changes in health. The point estimate, however, is minuscule. We next re-estimate the model using inheritance as an instrument, and no longer obtain a statistically significant result. Further, even when the point estimate is increased by twice its standard error, the implied quantitative effect is very small. Thus, to the extent there is a causal link running from short-term changes in wealth to changes in health, it essentially disappears once endogeneity is taken into account. Section 5 concludes the paper and provides suggestions for future research.

2. PREVIOUS LITERATURE

A critical issue in examining the relationship between economic resources and health status is how to measure economic resources. A number of studies have used income, and virtually all find that health improves with income, *ceteris paribus.* (See, for

example, Ettner [1996], McDonough, *et al.* [1997], Meara [2001]). Income data have the advantage of being relatively easy to obtain, but for some purposes, wealth is a superior measure of economic resources. As Smith and Kington [1997] note, "[i]ncome in a single year may not adequately measure the financial resources available to an individual over the lifetime in which decisions affecting health are made." McDonough *et al.* [1997], Feinstein [1993], and Smith [1999] similarly argue for the superiority of a wealth measure of economic capacity. Feinstein [1993] suggests that "the problem of reverse causality is less likely to afflict household wealth than household income measures, primarily because wealth accumulates over time and hence is less affected by a single episode of sickness." Two caveats apply to this comment. First, as noted by Feinstein, very serious negative health shocks could result in a decline in wealth. Indeed, Levy [2000] finds that the number of nights spent in a hospital is negatively correlated with changes in wealth for those individuals who do not have health insurance. Second, the relationship between wealth and health could still be driven by third factors, such as childhood environment, genetics, or the like.

As noted above, researchers in this area are well aware of the possibility that economic resources and health status may be simultaneously determined. However, not much has been done to deal with this problem econometrically. One careful attempt, by Ettner [1996], examines the relationship between health and income in an ordered probit instrumental variables framework. Among the instruments she uses for an individual's income are the state unemployment rate, work experience, parental education, and spousal characteristics. She finds that the effect of income on health remains significant and even increases after instrumenting. Of course, the instrumental variables results are

dependent on the assumption that the instruments can be excluded from the main regression, and as Ettner notes, there may be some problems in this regard. The state unemployment rate is driven by regional variation, and so is a valid instrument only if the well-known regional variations in health are due solely to differences in income. Parental education is plausibly related to early human capital investments in health. Spousal characteristics may reflect assortative mating, and so may be directly related to health. And work experience is generally a function of age and education, each of which arguably may directly influence health.

Hurd, McFadden, and Merrill [1997] also analyze the causal relationship between health and wealth. They use data from the Asset and Health Dynamics among the Oldest-Old (AHEAD) study, and focus on Granger-causality between measures of socioeconomic status and health.[1] They find that self reported health status (SRHS) in 1993 is correlated with changes in wealth between 1993 and 1995, but that changes in health conditions, conditional on SRHS, are not correlated with changes in wealth. They also find that measures of socioeconomic status in 1993 are correlated with mortality rates between 1993 and 1995. In this paper we pursue an alternative strategy for determining the causal influence of wealth on health, namely using inheritance as an instrument.

3. DATA

Our estimation framework, which is described in detail in the next section, models the short-term change in health status of a household head over a five year period as a function of the contemporaneous change in household wealth, initial wealth, and other

[1] A similar approach is employed by Attanasio and Emmerson [2001] using the British Retirement Survey data set.

covariates. This strategy motivates our selection of data, which are drawn from the 1984, 1989, 1994, and 1999 waves of the Panel Study of Income Dynamics (PSID). The PSID is a longitudinal survey of a representative sample of the U.S. population. Data exist for individuals as well as for the family units in which they reside. We focus on these four waves, as they contain detailed information about household wealth as well as inheritance.

Our basic unit of observation is the household head. We form our dataset from individuals who are heads for three consecutive waves; either 1984, 1989, and 1994, or 1989, 1994, and 1999. For those individuals who were heads for all 4 waves, we disregard information from 1999 so as to maintain a 3-year balanced nature to our panel. We drop observations that indicate a change in age (over a 5-year period) of less than 4 or more than 6 years, or if the PSID indicated that the family moved or changed household heads. We also drop those observations with missing information on inheritance, regional location, health, and wealth, and those observations with zero individual-level weights. We dropped the observations on one individual with extreme outliers in the "change in wealth" variable (losses greater than $5 million or gains greater than $10 million).[2] We use the first wave of each individual to obtain lagged health and wealth information, and so each individual's 3-wave panel contributes 2 observations to our dataset. Our final dataset contains 3302 individuals and 6604 person-year observations.

Our measure of the individual's health status is based on the answer to the following question: "Would you say your health in general is excellent, very good, good, fair, or poor?" The answer to this question is coded on a 1 to 5 scale, with 1 being excellent, 2 as very good, 3 as good, 4 as fair, and 5 as poor. We create a dichotomous variable

HEALTHY, which takes a value of one if the individual rates his or her health as "excellent," "very good," or "good," and zero otherwise. Out of a total of 6604 person-year observations, there are 433 positive transitions in health (that is, movements from NOT HEALTHY to HEALTHY), and 643 have negative transitions. Of the 3302 individuals in our sample, 411 experience a decline in their health status (and no increase), 201 experience an improvement (and no decline), 232 experience a decline in one period and an improvement in the other period, and 2458 have no change in their health.

An important question is whether these self-reported measures provide meaningful indicators of health status. In a review of more than two dozen studies, Idler and Benjamini [1997] document that poor SRHS is strongly correlated with mortality. This is true across many populations, and after controlling for a variety of socio-demographic variables, the presence of health conditions, and even medical doctors' objective health assessments. Additional evidence along these lines is provided by Hurd *et al.* [1997], who find correlations in the AHEAD data between SRHS and both mortality and the onset of several serious health conditions, again controlling for socio-demographic conditions.[3] While the consensus thus appears to be that SRHS is a meaningful indicator of health, we also experimented with a more "objective" measure reported in the PSID, whether the individual reports having a physical or nervous disability. As noted below, this had no effect on our substantive results.

The PSID wealth variable is computed as the sum of main home value, net value of other real estate, net value of vehicles, net value of any farms or businesses, net value of stocks and other financial instruments, cash accounts, and the net value of other assets

[2] However, our substantive results were unchanged when we included the outlier back in the sample.

less outstanding mortgage principal and other debts.[4] We convert the wealth variable to thousands of 1999 dollars using the CPI deflator. Table 1 provides the definition and summary statistics for wealth as well as for the other key variables used in our analysis. All of the variables other than the change in wealth, initial wealth, and age are dichotomous.

The figures in Table 2 show how the change in wealth varies with initial health status and with the kind of health transition over the five-year periods. From the first column of figures, we see that those who are initially ill have lower increases in their wealth than those who are initially healthy. Further, from the second and third columns, those whose health improves during the sample period experience higher increases in wealth than those who remain ill, and those who become ill accumulate less wealth than those who stay healthy. These patterns are consistent with previous findings (see, for example, Smith [1999]).

4. ANALYSIS

4.1 Preliminary Issues

Our basic equation models the impact of changes in wealth on changes in health as follows:

$$\Pr(H_t = 1) = F[\beta_0 + \beta_1(\Delta W) + \beta_2 H_{t-5}) + \beta_3 X_t] \qquad (1)$$

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[3] For a discussion of the reliability of the PSID self-reported health status variable, see Gouskova and Schoeni [2002].

[4] As is well-known, wealth data in surveys are subject to substantial measurement error. The standard errors-in-variables argument suggests that non-instrumental variables estimates should be attenuated. We find below that the IV estimates of the coefficient on the change in wealth are in fact larger than their non-IV counterparts. Our data contain no information on pension wealth. In their analysis of British data, Attanasio and Emmerson [2001] show that the inclusion of pension wealth has no material effect on the estimate of the impact of wealth rank on health status. While encouraging, of course one cannot know whether this result would hold in US data.

where H_t is an indicator for whether the individual is healthy (HEALTHY = 1) in year t; ΔW is the change in wealth from year (t-5) to year t; and F[●] is the cumulative normal distribution. We augment this basic regression with a set of covariates X_t, which includes the individual's current age, household wealth at the beginning of the 5-year period, and time invariant indicators for education, sex, race and region.[5]

Throughout the paper, we present results for three specifications for X_t. The first includes only age (as well as change in wealth and initial health). The second adds the initial level of wealth (wealth in year (t-5)) as a control variable. While we are concerned about the possible endogeneity of this variable, it may be informative to determine whether the impact of the change in wealth is sensitive to its initial level. The third specification has initial health, the two wealth variables, as well as the remaining covariates discussed above. In all the specifications we include time effects. In computing confidence intervals for our parameter estimates, we wish to account for possible within-individual correlation of the errors and right hand side variables. To do this, we estimate robust standard errors within a clustered framework, with all years of an individual serving as the cluster.

4.2 Basic Probit Results

To begin, we estimate the model without correcting for the endogeneity of the change in wealth. The results are reported in Table 3. In the basic specification in the first column, the coefficient on the change in wealth is positive, and exceeds its standard error by more than a factor of two. This finding demonstrates that we can reproduce the standard result in the literature – there is a positive and statistically significant relation-

[5] We enter the values of education, sex, race and region as of 1984. It is well documented that education is an important correlate of health (Ross and Mirowsky [2000]), as are race (Smith and Kington [1997]), re-

ship in the data between changes in health status and changes in economic resources. Quantitatively, the coefficient indicates that increasing the change in wealth over a five-year period by a million dollars increases the probability of being in good health at the end of the period by 4.8 percentage points. We return below to the issue of how to assess whether this is a "large" or "small" magnitude. As we move across the columns in Table 3, we see that augmenting the equation with initial wealth increases the magnitude of the coefficient on change in wealth, but adding in the other covariates brings it back to the same level as in the basic equation. Note also that including additional covariates increases the relative size of the coefficient's standard error so that one cannot reject the hypothesis that it is zero.

4.3 Accounting for Endogeneity of the Change in Wealth

As emphasized above, a major concern in the estimation of this model is the possible endogeneity of change in wealth. Our instrument for the change in wealth between year (t-5) and year t is the amount of gifts and inheritances received during that time interval, as reported in the answer to the following question:

> "Some people's assets come from gifts and inheritances. During the last five years, have you (or anyone in your family living there) received any large gifts or inheritances of money or property worth $10,000 or more?"

Those who answered "yes" were then asked to specify the amount; this is our *Total Inheritance* variable. Its summary statistics for the entire sample and stratified by initial health are in Tables 1 and 2, and respectively. On average, the 297 nonzero responses in the sample received $92,149 in inheritances over an average 5-year period. The 273 recipients who were healthy at the beginning of the period received $91,014 on average,

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gion (Preston and Taubman, [1994]), and sex (Verbrugge, [1985]).

and the 24 who were not healthy received $104,888 (the difference is not statistically significant).

We first test for the predictive power of our instrument in the first stage.[6] To do so we estimate a series of ordinary least squares regressions of change in wealth on inheritance. The results, reported in Table 4, show that inheritances are strongly correlated with changes in wealth for all three models. These results suggest that we have suitable first-stage power in our instruments.

We next estimate a reduced form regression, substituting inheritance received for change in wealth. The motivation for this exercise is very simple: if change in wealth is correlated with inheritance, then if we substitute inheritance into the health status equation, the significance and magnitude of its coefficient indicate the potential role for the causal impact of changes in wealth on health. The results are in Table 5. The inheritance variable is insignificant, both with and without other covariates. We take this as evidence that the change in wealth is not a significant determinant of health when endogeneity is taken into account.

While informative, this result does not tell us about the size of the coefficient on change in wealth. We are interested in inference about the possible magnitudes of the impact of wealth on health, and so require information on the distribution of this coefficient. To obtain such information, we estimate equation (1) with a two-stage probit. Specifically, we first regress our endogenous variable (change in wealth) on our instrument (inheritance) and the other exogenous variables, and generate the predicted values and residuals from this first stage regression. We next estimate a probit equation for the

[6] For discussions of IV problems in the presence of weak instruments, see Bound *et al.* [1993] and Staiger and Stock [1997].

probability of being in good health, including on the right hand side the predicted values and residuals from the first stage, as well as the other exogenous variables.[7] Our focus is on the coefficient on predicted change in wealth. We use the bootstrap with 1000 repetitions to compute consistent standard errors.

Table 6 reports the marginal effects for the bootstrapped two-stage probit model.[8] The figures in column 3 of the table indicate that the marginal effect of a $1,000,000 change in the change in wealth is 0.09505, with a standard error of 0.1460. The coefficient on initial wealth remains significant, both with and without the other covariates.

Roughly speaking, taking Tables 3 and 6 together suggests that within each model, taking into account endogeneity increases the coefficient on the change in wealth, but increases its standard error by an even greater proportion. A natural question is whether the point estimate is in some sense large. To be specific, consider the coefficient of 0.09505 in the third column of Table 6, which is our preferred specification. It tells us that for each $1,000,000 wealth increase over a five-year period, the probability of being healthy (*ceteris paribus*) increases by 9.5 percentage points. Is this a "large" or "small" effect? A natural way to approach this question is to compute the elasticity of the probability of being healthy with respect to changes in wealth. But in our context, this is not a viable strategy because the median value of the change in wealth is near zero, and computing percentage changes around a value of zero can lead to meaningless results.

Instead, we take a different tack. We begin by using our IV estimate of the coefficient on the change in wealth to simulate the impact on health status of an enormous

[7] For further details, see Rivers and Vuong [1988]. .

[8] Two-stage probit coefficients were calculated using a Stata module developed by Jonah Gelbach of the University of Maryland. As noted above, we perform a clustered procedure to allow for the possibility of within-individual correlation of the errors. To implement this in a bootstrapping context, we first create a

difference in the change in wealth--from the 10th percentile (-$81,454) to the 90th percentile ($176,422). Our preferred IV specification in the third column of Table 6 implies that this roughly quarter of a million dollar increase in the change in wealth leads to only a 2.2 percentage point increase in the probability of being healthy (off a baseline probability of about 0.81). When we increase the coefficient by 1.96 times its standard error, the analogous figure is about 6.7 percentage points.

Given the huge difference in the change in wealth needed to generate a 6.7 percentage point increase in the probability of being healthy even at the upper end of the 95-percent confidence interval, we feel quite comfortable in characterizing the effect as small. To gain further perspective on this issue, we compute the cross-sectional effects of a number of variables (race, education, and age) that previous literature has shown to exert large effects on health status. Specifically, we consider the impact on the probability of being healthy (conditional on age only) of being white rather than black, of completing high school rather than not completing high school, and of being 20 years younger. We find that these increase the probability of being in good health by 16, 18, and 12 percentage points, respectively. All of these effects substantially exceed the effect of a quarter million dollar increase in the change in wealth, evaluated with a coefficient at the upper end of the 95-percent interval. In short, the wealth effect is not in the same league as other effects that have previously been viewed as large.

4.4 Evaluating the IV Strategy

In this section, we discuss several possible concerns with our instrumentation strategy. These concerns fall into two classes. The first is potential violations of the IV

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list of individuals. For each bootstrap iteration, we then draw a set of individuals from this list, and use all years of data from the selected individuals to construct that iteration's dataset.

exclusion restrictions. The second relates to the possibility that the use of inheritance as an instrumental variable results in coefficients that do not measure the true wealth effect of health. We now discuss each set of concerns in turn.

4.4.1 Does inheritance fail the exclusion restrictions?

Two reasons that inheritance might violate the exclusion restrictions are that inheritances give a signal about one's health, or that there are third variables driving both inheritances and health:

A family member dying might signal something about one's own health. This could induce a correlation between the inheritance variable and the error term in the health status equation. One way to investigate this possibility would be to augment the equation with an interaction between the inheritance and the age of the donor. If there is anything to the story that inheritance is a signal about health, then individuals who receive inheritances from younger donors should have higher probabilities of being in poor health, *ceteris paribus*. Unfortunately, the PSID reports no information about the donor. However, we note in passing some other research that may cast some light on this issue. Holtz-Eakin, Joulfaian, and Rosen [2001] studied the impact of inheritance on retirement decisions using a data set that did include age of donor. They found that it had no effect on the probability of a donee retiring from the labor force. We know that retirement decisions are strongly affected by health (McClellan [1998]). Therefore, the fact that donor's age had no impact on retirement in Holtz-Eakin *et al.*'s study does not lend support to the idea that receiving inheritance is a signal that an individual is in poor health. We

interpret this as rough evidence that the signal of one's health associated with inheritance is of secondary importance to the direct wealth impact.[9]

Some unobserved variable might drive both inheritance and health status. Suppose, for example, that individuals with "privileged" backgrounds are particularly likely to receive inheritances, and are also likely to have benefited from the childhood care that leads to good health as adults. In effect, then, the coefficient on inheritance in the first stage regression partly reflects the impact of "privilege," biasing its coefficient upward. This in turn leads to an upward bias in the coefficient on the fitted value of change in wealth in the second stage equation. For example, wealthier individuals tend to have healthier children. Therefore, one might expect children who receive inheritances to be on a better lifetime health trajectory, possibly even conditional on initial health. Such a mechanism would tend to render inheritance a poor instrument for examining the causal impact of wealth on health. However, three considerations suggest that this is not an important problem in our context.

First, as noted above, the coefficient on the change in wealth in the second stage is close to zero. Hence, to the extent that such bias is present, it does not seem very powerful. Second, observable variables like wealth and education might be serving the same role as "privilege," yet when they are included, there is very little impact on the results. Third, the PSID contains retrospective information about the economic status of the parents of the respondents, enabling us to examine this issue directly. Respondents are asked the following question: Were your parents poor when you were growing up, pretty

[9] A related possibility is that when someone dies, it produces stress and illness for the relatives. We would expect this effect to be greatest for close relatives of the deceased. However, Holtz-Eakin, Joulfaian and Rosen [1993] found that the effect of inheritance on the probability that an individual left the labor force was independent of whether or not he or she was a close relative of the deceased. It appears, then, that

well off, or what? Based on the response, we create the dichotomous variables *Parents-Poor* (equals one if the parents were poor); *Parents-Average* (equals one if the parents were average), and *Parents-Well* (equals one if the parents were well-off). We then augment the basic model from the third columns of Tables 3, 4, 5, and 6 with these variables (the omitted category being those who did not grow up with their parents). The basic finding is that the parental background variables are statistically insignificant, and their inclusion leaves the coefficient on the change in wealth small and statistically insignificant. For example, the coefficient from the specification of the third column of Table 6 is 0.08762 with a standard error of 0.1356. In short, to the extent that the data allow us to investigate this issue, our results do not appear to be driven by the joint impact of family background on both health and inheritance.

It is still possible that there is some combination of a true wealth effect and unobserved bias that exactly cancel in our data to produce a coefficient of zero. Suppose, for instance, that wealth from inheritances leads to improved health, but that inheritances also signal poor health in the family. If these two effects were equal in magnitude, we might find a zero result. While we cannot rule this story out entirely, we believe that the robustness of our results to a number of alternative specifications as well as the evidence in Holtz-Eakin *et al.* reduce the likelihood that this is what is going on.

4.4.2 Is inheritance related to the relevant wealth effect?

The interpretation of our estimates becomes less clear if inheritances are anticipated. To the extent this is true, forward looking people may base consumption and saving decisions on anticipated receipts rather than actual dollars in hand (provided that they

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whatever health effects might accompany receipt of an inheritance, they are not sufficient to generate a change in labor force behavior.

can dissave based on the expected inheritance). If so, then a reduced form regression of health status on inheritance produces a small and imprecisely estimated coefficient on inheritance, but it tells us nothing about the underlying relationship between health and wealth. The PSID provides information that is of substantial interest in this context. In the 1984 wave of our sample, the head of household was asked,

> "What about future inheritances--are you fairly sure that you (or someone in your family living there) will inherit some money or property in the next ten years?"

Summary statistics for this anticipated inheritance variable are reported toward the bottom of Table 1. When we augment the first stage equation with this variable, we find it is miniscule and insignificant (-0.05477 with a standard error of 0.07126), while the actual inheritance coefficient remains unchanged. That is to say, the expectation of inheritance does not affect wealth accumulation behavior when inheritances actually received are taken into account. In short, perhaps because of capital market constraints, individuals do not appear to change their wealth accumulation patterns in anticipation of receiving inheritances. This conclusion is consistent with the work of Holtz-Eakin, Joulfaian, and Rosen [1993], who analyzed the labor force behavior of individuals before and after the receipt of inheritances. If individuals fully anticipated inheritances and could borrow on them, then we would expect the receipt of an inheritance *per se* to have no effect on their labor supply. However, in contrast, Holtz-Eakin, Joulfaian and Rosen found that there were substantial effects on labor force participation rates.[10]

[10] It is possible that the children of a decedent are more likely to anticipate their inheritances than other relations. Hence, comparing the labor supply response of children with other recipients provides an additional way to shed some light on this issue. Holtz-Eakin, Joulfaian, and Rosen find that there are no statistically significant differences in the responses between the children of decedents and other donees.

Another possible problem is that a concave relationship between economic resources and health builds in a bias against finding a relationship between inheritance and health status. The argument here is that individuals who receive inheritances tend to be wealthier to start with and already in the flat region of the health status-wealth curve. Therefore, the fact that their health status does not change when they receive an inheritance is uninteresting. It is indeed true that individuals who receive inheritances have higher average wealth than those who do not. Mean initial wealth for the sample of those who do not receive an inheritance is $167,766, while initial wealth for those who did receive an inheritance was $277,652. A test for equality of means is rejected, with a t-statistic of 3.75. We note, however, that inheritances do not go only to individuals at the top of the wealth distribution: 76 individuals with below-median wealth received inheritances, representing about a quarter of all those who received inheritances.

4.5 Alternative specifications

We estimate a number of variations on our basic model in order to assess the robustness of the results.

Alternative measures of health status. As noted above, our self-reported health status variable is one if the individual reports himself to be in "good," "very good," or "excellent" health, and a zero if his health is "fair" or "poor." There is clearly some arbitrariness to this procedure; to see if it made a substantive difference, we grouped "good" with "fair" and "poor," and re-estimated the model with the new left-hand side variable. There was essentially no change in our results. For example, in the version of the model with all covariates (i.e., column (3) of Table 6), the coefficient on change in wealth is 0.07521 with a standard error of 0.2324. An alternative approach is to eschew any ag-

gregation of the health categories at all, and use an ordered probit statistical model to estimate the probability that the individual is in any given category. This exercise also leads to an imprecisely estimated impact of the change in wealth on health status.[11]

A related question is whether the results would change if an alternative health measure was used. The PSID asks respondents if they had a physical or nervous disability that limits their ability to work. We create a dichotomous variable based on the answer to this question, and use it on the left hand side in our basic model. Again, we find a statistically insignificant coefficient on the change in wealth (-0.3229 with a standard error of 0.2233).

Alternative samples. As noted above, we chose our sample having in mind the two goals of maintaining a balanced panel and maximizing the number of observations. One alternative strategy is to use a four-year balanced panel. This gives us more observations per person but at the cost of a smaller number of people (for a total number of person-year observations of 4452). Another possibility is to treat the data set as a series of two-year panels: 1984-89 (N = 3527), 1989-94 (N = 3886), and 1994-99 (N = 3057). This maintains a balanced panel for each pair of years, and uses (between the three panels) all of the data possible. Finally, a strategy that maximizes the number of observations in a single regression without regard to whether the panel is balanced is simply to include all the observations in adjacent years (N = 10470). We tried all three of these strategies to see if our substantive results were sensitive to the way our sample was constructed. As one might expect, there is some movement in the estimated coefficient on

[11] In the ordered probit model, we include on the right hand side a series of dichotomous variables for health status in the previous period. Some caution must be exercised in interpreting our result because the mechanics for computing correct standard errors in an IV ordered probit model are not well understood.

the change in wealth depending on the sample used. For example, using the largest sample we found a larger coefficient (0.18 with a standard error of 0.154), while using the four-year balanced panel yields a smaller coefficient (-0.012 with a standard error of 0.070). In all cases, though, the coefficient is small and imprecisely estimated. Our findings are robust to the way in which the sample is put together.

Attrition. The manner in which our sample is constructed gives rise to another issue: some people disappear from the sample due to death (and other nonrandom reasons). There is no straightforward way to deal with this problem because, by definition, one cannot compute the change in wealth from one period to the next for people who are not present in the latter period. To get some sense of whether or not nonrandom attrition would be a problem, we began by taking the sample of people who were present in 1984 and 1989, and dividing them into two groups, those who remain in the sample until 1999 and those who disappear. We then estimate our canonical specification for each subsample.

In the specification without instrumental variables, we find that the coefficients on the change in wealth in the two subsamples were very close to one another, small and positive (.070 in the main sample, .045 in the "selected out" sample), and statistically indistinguishable. In the IV specification, while we could not reject the hypothesis that the coefficients are the same, the point estimate for the "selected out" sample was rather large (2.3), with an extremely large standard error (4.1). We believe that this is due to the weak power in this sample: there are only 867 observations, with 31 inheritances, in this sample. Note that the results from the “selected out” sample do not drive the 1984-89

Our estimates are generated simply by taking the fitted value of the change in wealth from the first stage regression and substituting them for the actual value, without any correction of the standard errors.

data as a whole. When we estimate the IV specification using the pooled sample, the results are very similar to those found in the main sample (the coefficient is 0.040, with a t-statistic of 0.17). Hence, to the extent that we can examine the issue, we find no evidence that accounting for attrition changes our conclusions, although admittedly this is not a definitive conclusion.

Spouses. The PSID also asks about the health status of the head of household's spouse. As a check on our main results, we re-estimated our model with the health of the spouse on the left-hand-side, and the spouse- and household-specific variables on the right-hand-side. Since not every household has a spouse, our sample is smaller, with 3424 observations. The results for spouses indicate an even smaller effect of changes in wealth on health status. For example, in the specification with the full set of covariates, the coefficient on (instrumented) change in wealth is -.003429 (s.e. = 0.1069). While the spousal estimates are not statistically independent of the main results (the observations come from the same set of households, and have some identical household-level covariates), we view this as supportive evidence for our main findings.

Interactions with change in wealth. We augment our model with an interaction between wealth and the change in wealth and find that the interaction was statistically insignificant (-0.09201with a standard error of 0.1149). Hence, our results are not an artifact of underlying interactions between the level of wealth and the change in wealth. We also examine whether statistically significant interactions between age and change in wealth are present, and do not find any.

Timing of inheritance. Our instrument for the change in wealth over a five-year period is the value of inheritances received over that period. One can imagine that the

timing of inheritance may matter, with inheritances received early in the 5-year period influencing health more than those received later in the period. The PSID asks people when they received their inheritances, so we were able to investigate this possibility. To do so, we created a variable, TIME, which equals the number of years since the inheritance was received, and interacted TIME with the inheritance variable. We include the interaction term in the reduced form equation for health status (as in Table 5). In this specification, the interaction term has the expected (positive) sign, but both the interaction term and the inheritance terms are small and insignificant. Examining the point estimates, we find that an inheritance received four years prior has the approximate impact of the coefficients reported in Table 5. Second, we incorporate the interaction term into the IV probit estimates by including it as an instrument in the first stage. The second stage estimates of the coefficient on the change in wealth are uniformly statistically insignificant, and smaller than their Table 6 counterparts. Again, the timing of the inheritance within the five-year window does not affect our results.

Econometric specification. A possible problem with our results is that they rely on the normality assumptions underlying the two-stage probit model. We re-estimate our models using a linear probability model as a robustness check. The results, available upon request, are very similar to those obtained using the probit. In particular, the marginal effect of the change in wealth remains statistically insignificant and small - a million dollar increase in wealth changes the probability by a mere 6.4 percentage points. This suggests that our results are not an artifact of the assumptions behind the probit model.

5. CONCLUSION

We have used the Panel Study of Income Dynamics to examine the relationship between health and wealth. We confirmed the result from earlier studies that changes in wealth have a statistically strong correlation with changes in health. However, the use of an instrumental variables procedure to take into account the endogeneity of the change in wealth rendered the effect statistically insignificant. Further, both the uncorrected and instrumental variables point estimates suggested that changes in wealth have a minuscule impact on health status. We conclude that one of the three possible pathways in the health-wealth nexus, namely that wealth affects health in the short-run, is in fact illusory.[12]

This finding does not rule out the possibility of a long-term impact of wealth on health. Because we analyze changes in health and wealth only over 5-year intervals, we are unable to assess the long-term causal impact.[13] That said, we should again note that the strong (non-instrumented) short-term correlation between wealth and health is typical of the type of evidence that many have adduced for the existence of a causal impact of economic resources on health. In our setting, a reasonable instrumentation strategy makes the correlation disappear. We therefore believe that a stronger burden of proof must be met in claiming causal relationships between these variables.

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[12] For research that examines the causal link from health to wealth, see Hurd *et al*. [1998], Levy [2000] and Wu [2003].

[13] Note, however, that in their study of the relationship between income and mortality in the United States and Britain from the 1950s through the 1990s, Deaton and Paxson [2001] find that income trends do not do a good job of explaining mortality trends. That is, over a decades long time period, the link between economic resources and health status appears to be weak.

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Table 1
Summary Statistics

Variable	Description	Mean	Standard Deviation
Health	Self-rated health: 1 if healthy and 0 otherwise.	0.8094	0.3928
ΔWealth	Difference between household wealth in year t and t-5 in millions of 1999 dollars.	0.04286	0.3330
Initial Wealth	Sum of main home value, net value of other real estate, net value of vehicles, net value of any farms or businesses, net value of stocks and other financial instruments, cash accounts, and the net value of other assets less outstanding mortgage principal and other debts in millions of 1999 dollars.	0.1744	0.4347
Total Inheritance	Dollar amount of all inheritances received between year t and t-5 in millions of 1999 dollars. Mean and standard deviation are reported for the 297 nonzero responses.	0.09214	0.2180
Expected Inheritance	Dollar amount of the inheritances the head expects to receive between year t and t-5, in millions of 1999 dollars. Mean and standard deviation are reported for 225 nonzero responses. This variable is only available in 1984.	0.1276	0.3922
Age	Head's age.	49.16	15.82
No Degree	Head does not have a high school degree during the first year head is present in the panel.	0.2358	0.4245
HS Degree	Head has a high school degree and possibly some further technical training during the first year head is present in the panel.	0.3487	0.4766
Some College	Head attended but did not complete college during the first year head is present in the panel.	0.1933	0.3949
College Degree	Head has a college degree during the first year head is present in the panel.	0.1565	0.3634
Male	Head is male.	0.7438	0.4366
Black	Head is black.	0.1157	0.3199
White	Head is white.	0.8582	0.3488
North-Central	Head lives in the north-central region during the first year head is present in the panel.	0.2867	0.4523
North-East	Head lives in the north-east region during the first year head is present in the panel.	0.2092	0.4068
South	Head lives in the southern region during the first year head is present in the panel.	0.3141	0.4642
Married	Head is married during the first year head is present in the panel.	0.5988	0.4902

Widowed	Head is widowed during the first year head is present in the panel.	0.1026	0.3034
Divorced	Head is divorced during the first year head is present in the panel.	0.1328	0.3394
Whether Children	Head has at least one child during the first year head is present in the panel.	0.4140	0.4926

Means are computed using PSID sample weights, and taken over 6604 person-year observations.

Table 2
Health Transitions and Change in Wealth

		Whole Sample	Sick in Year T	Healthy in Year T
Sick in Year T-5	Mean of ΔWealth	0.008847	0.007255	0.01187
	Standard Deviation	0.2950	0.3419	0.1741
	Sample Size	1274	841	433
Healthy in Year T-5	Mean of ΔWealth	0.04923	0.001815	0.05471
	Standard Deviation	0.3393	0.1725	0.3531
	Sample Size	5330	643	4687

Means are computed using PSID sample weights..

Table 3
Probit Estimates of Health Status (Marginal Effects)

	ΔWealth	*Wealth Variables*	*With Other Covariates*
ΔWealth	*0.04840* *(0.01932)*	0.07986 (0.05066)	0.04918 (0.03969)
Initial Wealth	-	*0.1647* *(0.03961)*	0.08370 (0.03435)
Initial Health	*0.4781* *(0.02152)*	*0.4498* *(0.02334)*	*0.4043* *(0.02420)*
Age	*-0.004555* *(0.0003304)*	*-0.005025* *(0.0003383)*	*-0.004809* *(0.0004615)*
No Degree	-	-	*-0.1149* *(0.03371)*
HS Degree	-	-	*-0.05531* *(0.02773)*
Some College	-	-	-0.01549 (0.02918)
College Degree	-	-	0.01864 (0.02831)
Male	-	-	-0.02831 (0.01720)
Black	-	-	-0.07396 (0.04527)
White	-	-	-0.02906 (0.03049)
North-Central	-	-	-0.01603 (0.01662)
North-East	-	-	0.0001899 (0.01786)
South	-	-	*-0.03329* (0.01688)
Married	-	-	0.03691 (0.02243)
Divorced	-	-	-0.01999 (0.02132)
Widowed	-	-	-0.008474 (0.02606)
Whether Children	-	-	*-0.02741* *(0.01356)*
1989 Time Effect	-0.01988 (0.02194)	0.01812 (0.02133)	-0.01719 (0.02188)
1994 Time Effect	-0.03931 (0.02135)	-0.03908 (0.02063)	-0.03862 (0.02099)

The left hand side variable is one if an individual is healthy in year t and zero otherwise. Coefficients are marginal probability effects of the respective variables, which are defined in Table 1. For dichotomous variables, the coefficient is the effect of a discrete change from 0 to 1. The sample size in all models is 6604 person-year observations. Standard errors are in parentheses. Those significant at the 5% level are italicized.

Table 4
First Stage of Instrumental Variables Regressions

	ΔWealth	*Wealth Variables*	*With Other Covariates*
Total Inheritance	*0.9946* *(0.4493)*	*0.9910* *(0.4411)*	*0.9425* *(0.4374)*
Initial Wealth	-	0.008299 (0.08638)	-0.01710 (0.08871)
Initial Health	*0.03032* *(0.01153)*	*0.02912* *(0.01458)*	0.004821 (0.01318)
Age	*-0.0005182* *(0.0003296)*	-0.0005612 (0.0004792)	-0.0004685 (0.0005538)
No Degree	-	-	*-0.1244* *(0.03378)*
HS Degree	-	-	*-0.1211* *(0.03163)*
Some College	-	-	*-0.1115* *(0.03314)*
College Degree	-	-	-0.03052 (0.04003)
Male	-	-	0.01101 (0.01366)
Black	-	-	0.02043 (0.04983)
White	-	-	0.01899 (0.05096)
North-Central	-	-	-0.02362 (0.01518)
North-East	-	-	-0.009385 (0.02136)
South	-	-	*-0.03719* *(0.01472)*
Married	-	-	0.02739 (0.01663)
Divorced	-	-	0.007210 (0.01124)
Widowed	-	-	0.01331 (0.01903)
Whether Children	-	-	-0.006870 (0.01242)
1984-1989 Panel	-0.01015 (0.02636)	-0.01024 (0.02639)	-0.01384 (0.02671)
1989-1994 Panel	-0.03955 (0.02632)	-0.03983 (0.02633)	-0.04243 (0.02617)
F-statistic	*6.62*	*5.52*	*6.07*
Number of Observations	6604	6604	6604
R-squared	0.0350	0.0352	0.0533

The left hand side variable is ΔWealth. Estimation is by ordinary least squares. Standard errors in parentheses. Those significant at the 5% level are italicized. The sample size in all models is 6604 person-year observations.

Table 5
Reduced Form Probit Estimates of Health Status (Marginal Effects)

	No Wealth Variables	*Wealth Variables*	*With Other Covariates*
Total Inheritance	0.2405 (0.1426)	0.1482 (0.1405)	0.07243 (0.1037)
Initial Wealth	-	*0.1549* *(0.04079)*	*0.07405* *(0.03330)*
Initial Health	*0.4792* *(0.02148)*	*0.4539* *(0.02293)*	*0.4055* *(0.4056)*
Age	*-0.004582* *(0.0003275)*	*-0.005037* *(0.0003366)*	*-0.004796* *(0.0004658)*
No Degree	-	-	*-0.1196* *(0.03323)*
HS Degree	-	-	*-0.05845* *(0.02740)*
Some College	-	-	-0.01739 (0.01739)
College Degree	-	-	0.02115 (0.02835)
Male	-	-	-0.02740 (0.01752)
Black	-	-	-0.07622 (0.04530)
White	-	-	-0.03068 (0.03015)
North-Central	-	-	0.01621 (0.01672)
North-East	-	-	0.001159 (0.01793)
South	-	-	-0.03399 (0.01699)
Married	-	-	0.03824 (0.02263)
Divorced	-	-	-0.01959 (0.02142)
Widowed	-	-	-0.007623 (0.02617)
Whether Children	-	-	*-0.02795* *(0.01367)*
1984-1989 Panel	-0.02107 (0.02202)	0.02203 (0.02149)	0.01924 (0.02202)
1989-1994 Panel	-0.04173 (0.02140)	*-0.04430* *(0.02078)*	*-0.04142* *(0.02110)*

The left hand side variable is one if an individual is healthy in year t and zero otherwise. Coefficients are marginal probability effects of the respective variables, which are defined in Table 1. For dichotomous variables, the coefficient is the effect of a discrete change from 0 to 1. The sample size in all models is 6604 person-year observations. Standard errors are in parentheses. Those significant at the 5% level are italicized.

Table 6
Two Stage Probit Estimates of Health Status (Marginal Effects)

	ΔWealth	*Wealth Variables*	*With Other Covariates*
ΔWealth	0.2404 (0.1764)	0.1704 (0.1568)	0.09505 (0.1460)
Initial Wealth	-	*0.1625* *(0.04476)*	*0.08395* *(0.03915)*
Initial Health	*0.4684* *(0.02316)*	*0.4454* *(0.02473)*	*0.4039* *(0.02515)*
Age	*-0.004452* *(0.0003509)*	*-0.004967* *(0.0003626)*	*-0.004783* *(0.0004771)*
No Degree	-	-	*-0.1069* *(0.04468)*
HS Degree	-	-	-0.04877 (0.03588)
Some College	-	-	-0.009578 (0.03550)
College Degree	-	-	0.02041 (0.03006)
Male	-	-	-0.02850 (0.01661)
Black	-	-	-0.07522 (0.04853)
White	-	-	-0.03001 (0.03119)
North-Central	-	-	-0.01474 (0.01700)
North-East	-	-	0.0007501 (0.01776)
South	-	-	-0.03139 (0.01756)
Married	-	-	0.03530 (0.02286)
Divorced	-	-	-0.02038 (0.02133)
Widowed	-	-	-0.008980 (0.02583)
Whether Children	-	-	-0.02696 (0.01401)
1984-1989 Panel	-0.01796 (0.02247)	-0.01720 (0.02158)	-0.01646 (0.02255)
1989-1994 Panel	-0.03170 (0.02355)	-0.03545 (0.02189)	-0.03658 (0.02208)

The left hand side variable is one if an individual is healthy in year t and zero otherwise. Coefficients are marginal probability effects of the respective variables, which are defined in Table 1. For dichotomous variables, the coefficient is the effect of a discrete change from 0 to 1. The sample size in all models is 6604 person-year observations. Standard errors, which are obtained from a bootstrap procedure with 1000 replicatons, are in parentheses. Those significant at the 5% level are italicized.