HEALTH AND THE WAGE RATE: CAUSE, EFFECT, BOTH, OR NEITHER? NEW EVIDENCE ON AN OLD QUESTION

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Abstract

In this paper we use the wage rate—the most comprehensive measure of the stock of knowledge of investors who have completed formal schooling—and a number of measures of health to investigate two-way causality between health and the wage. We employ insights from the human capital and compensating wage differential models, a panel formed from the National Longitudinal Survey of Youth 1997, and dynamic panel estimation methods (for example, Arellano 2003, Baltagi 2008) in this investigation. We adopt plausible specifications in which a change in health induces a change in the wage with a lag and in which a change in the wage induces a change in health, also with a lag. We uncover a causal relationship between health and the wage in which a reduction in health leads to an increase in the wage rate in a panel of U.S. young adults who had completed their formal schooling by 2006 and were continuously employed from that year through 2011. There is no evidence of a causal relationship running from the wage rate to health in this panel. The former result is consistent with an extension of the compensating wage differential model in which a large amount of effort in one period is required to obtain promotions and the wage increases that accompany them in subsequent periods. That effort may cause reductions in health and to a negative effect of health in the previous period on the current period wage. The latter result may suggest that forces that go in opposite directions in the human capital and compensating wage differential models offset each other.

I. Introduction

Both the human capital model and the compensating wage differential model generate relationships between health and the wage rate. In Grossman's treatment of health as a form of human capital, he draws a sharp distinction between health capital and other forms of human capital, which he refers to as knowledge capital (Grossman 1972a, 1972b, 2000, 2017). Investments in knowledge capital raise wage rates, while investments in health capital raise the total amount of time available for market and household production in a given year and prolong length of life. That treatment suggests, however, important complementarities between the two forms of human capital. Returns to investments in knowledge are higher the more time that is available for market and household production, especially if productivity in both sectors is positively related to the stock of knowledge. Returns also are higher the longer are the number of periods that the investor lives. Returns to investments in health are larger the greater is the monetary value of the increase in healthy time and length of life. The wage rate of the investor is the most direct measure of this monetary value. These complementarities suggest causality from knowledge capital to health capital and from health capital to knowledge capital. [1]

The compensating wage differential model, which dates to Adam Smith (1937), was formalized in a seminal paper by Rosen (1974).[2] This model emphasizes that wage offers may

[1] Despite these complementarities and the two-way causality they suggest, Grossman (1972a, 1972b) treats knowledge capital as exogenous in his theoretical model of the demand for health. Galama and van Kippersluis (2016) construct a rich theoretical model in which both types of capital are endogenous. Their model allows for a complex and intriguing set of interactions and complementarities between health capital and knowledge capital. To date, it has not been used as the basis of empirical work. In our view, attempts to specify and estimate this model will be very rewarding.

[2] For comprehensive reviews of this literature, see Rosen (1986) and Viscusi (1993, 2014). We use the term compensating wage differentials to distinguish the negative health-wage relationships in a model with health-wage tradeoffs from positive health-wage relationships due to complementarities in the human capital model. Of course, the higher wages offered to the more educated be viewed as compensating them for the earnings they forego for attending school for more years than their less educated colleagues. So our terminology, is simply a "short-hand" way to distinguish between the two basic reasons for expecting relationships between health and the wage.

have to be adjusted upwards to compensate for health risks associated with specific jobs, occupations, and industries. Thus, in stark contrast to the human capital model, it generates negative relationships between health and the wage rate.

In this paper we use the wage rate—the most comprehensive measure of the stock of knowledge of investors who have completed formal schooling—and a number of measures of health to investigate two-way causality between health and the wage. We do this in a panel formed from National Longitudinal Survey of Youth 1997. We employ dynamic panel models (for example, Arellano 2003, Baltagi 2008) in this investigation. We employ plausible specifications in which a change in health induces a change in the wage with a lag and in which a change in the wage induces a change in health, also with a lag.

II. Background

A. Human Capital Model

Grossman and others who conducted empirical tests of his 1972 model of the demand for health have not explored complementarities between health capital and knowledge capital just outlined in detail (see Grossman 2000 for a discussion of empirical studies related to those in Grossman 1972b). Instead, the approach in this empirical literature has been to treat both schooling and wage rates as exogenous or more formally as predetermined variables that are not correlated with the disturbance term in the health outcome equation. Separate estimates of the effect of each have been obtained to explore the hypothesis that an increase in schooling can increase the demand for health, with the wage rate held constant, because it raises the efficiency with which health is produced or because it changes the mix of inputs selected by the investor. On the other hand, higher wage individuals demand more health, with schooling held constant,

because their monetary rates of return to investments in health exceed those of individuals with lower wage rates.[3]

In general, the research just mentioned finds positive effects of schooling and the wage rate on a variety of correlates of good health. The question of whether some portion of these effects reflects causality from health capital to knowledge capital is left open. In addition, the possibility that an unobserved "third variable" causes both types of capital to vary in the same direction is not addressed.

A rich empirical literature in health economics reviewed by Grossman (2006, 2015, 2017) addresses causality between correlates of knowledge capital and health capital in a thorough manner. Studies in this literature employ years of formal schooling completed as the measure of knowledge capital and a number of different correlates of better health. Much of the emphasis is on the estimation of causal effects of more schooling on the health of the investor or on her or his children. Some studies, however, seek to uncover causal effects of early investments in the health, cognitive, and noncognitive development of children on later outcomes.

Grossman (2006, 2015, 2017) outlines the broad conceptual foundation employed in the literature on the relationship between health and schooling. Students in poor health are almost certain to miss more days of school due to illness than their healthy peers and may also learn less while they are in school. Both factors suggest negative effects of poor health in childhood on school achievement and ultimately on years of formal schooling completed. Furthermore, this causal path may have long-lasting effects if past health is an input into current health status. Thus, even for non-students, a positive relationship between health and schooling may reflect

[3]See below for qualifications of these predictions.

causality from health to schooling in the absence of controls for past health. Health also may cause schooling because reductions in morbidity and in mortality increase the amount of time in a period and the number of periods during which the returns from investments in knowledge can be collected.

Productive and allocative efficiency models generate causality from schooling to health. In the former model, the more educated are assumed to obtain more health output from given amounts of medical care and other inputs. In the latter model, the more educated are assumed to pick a different input mix to produce health than the less educated. That mix gives them more output than the mix selected by the less educated.[4] Schooling may also cause health because it changes tastes in favor of health.

In addition to mutual causality, an unobserved third variable may cause health and schooling to vary in the same direction. Fuchs (1982) identifies time preference as perhaps the key third variable. He argues that persons who are more future oriented (who have a high degree of time preference for the future or discount it at a modest rate) make larger investments in health and schooling. Hence, the effects of schooling on health or of health on schooling are biased if one fails to control for time preference.[5]

The studies in this literature employ one of three econometric procedures. The first one directly includes such hard-to-measure third variables as time preference, cognitive development, noncognitive development, and past health. The second procedure controls for unobserved

[4] Both productive and allocative efficiency effects are grounded in Becker's (1965) household production function model of consumer behavior. Becker draws a sharp distinction between fundamental objects of choice (called commodities) that enter the utility function and market goods and services. Consumers produce commodities with inputs of goods and services and their own time. That model is one of the key building blocks Grossman (1972a, 1972b) uses to construct a model of the demand for health.

[5] Becker and Mulligan (1997) develop a model in which time preference for the future is an endogenous variable that is caused by schooling. Hence, an increase in a future orientation becomes a causal mechanism via which more schooling causes better health. None of the empirical literature discussed below treats time preference as endogenous.

genetic and environmental factors by examining the effects of differences in schooling obtained by identical twins on differences in their health outcomes or on the effects of differences in health at birth of identical twins on differences in the amount of schooling they obtain. The third procedure employs the technique of instrumental variables.

In his summary of this literature, Grossman (2006, 2015, 2017) points out that the studies that include hard-to measure third variables all find positive and significant effects of completed schooling on at least some measures of adult health and health behaviors as well as positive effects of past health on completed schooling. The results of studies that focus on twin differences or employ instrumental variables are more mixed. Some find significant causal effects, while others do not. Grossman concludes that, while there is a good deal of support for complementarities between schooling and health, there is enough conflicting evidence to warrant more research.

Part of our contribution in this paper is grounded in the literature just discussed but takes a somewhat different approach to investigating complementarities between health capital and knowledge capital and the extent of the causal relationships generated by these complementarities. Instead of measuring knowledge capital by completed schooling, we measure it by the wage rate. And instead of estimating only a health equation or only a knowledge (proxied by schooling) equation, we estimate equations for both types of human capital.

Our approach is not an entirely new one. Grossman and Benham (1974), Grossman (1976), and Lee (1982) estimate simultaneous-equations health-wage models in cross-sectional data. These studies are motivated by considerations briefly mentioned in the introduction to this paper. In that section we pointed out that returns to investments in health are larger the greater is

the marginal benefit of the increase in healthy time and length of life. The wage rate of the investor is the most direct measure of these benefits. One necessary condition for the marginal rate of return on the investment to rise is that the own time of the investor is not the only input in the production function of investment in health. In that case, the percentage increase in the marginal benefit of an investment exceeds the percentage increase in its marginal cost. A second necessary condition that underlies the prediction of a positive wage coefficient in the health demand function is that health is what Grossman (1972a, 1972b) terms a "pure investment commodity" because it does not enter the utility function. The prediction becomes complicated in a model in which health is both an investment commodity and a "consumption commodity" (a commodity that enters the utility function directly) because one has to take account of how the marginal cost of investment changes relative to the marginal costs of other household commodities.[6] Even if the relative price of health rises, the resulting negative substitution effect may be offset or more than offset by a positive income or wealth effect.

Returns to investments in knowledge are higher the more time that is available for market and household production, especially if productivity in both sectors is related to the stock of knowledge. Returns also are higher the longer are the number of periods that the investor lives. While these factors typically are used to explain why better health may cause more schooling, they also are relevant to investments in knowledge made after the completion of school. Grossman (2006) summarizes evidence suggesting that students' health influences their productivity in school. Reasoning by analogy, should not adults' health influence their productivity in the labor market? For example, individuals who miss a significant amount of

[6] These necessary conditions also are required to justify the predications made by Grossman and Benham (1974), Grossman (1976), and Lee (1982) about the effects of an increase in years of formal schooling completed on health, with the wage rate held constant.

time at work due to illness may lose the opportunity to advance by undergoing intensive computer training. Ultimately, that may hinder their opportunities for promotion, result in lower wage rates, and less facility with the use of computers in home production. More generally, Mincer (1974) stresses that investment in on-the job training, measured by the total amount of time spent in such an activity, plays a major role in the wage function in his seminal treatment of that function. That variable is imperfectly measured in most datasets. To the extent that poor health reduces the amount of time spent in the labor market and opportunities to invest in on-the-job training, past health may affect the current wage via its impact on past investment in that training.

To explore bi-directional causality between health and the wage, Grossman and Benham (1974) employ white males in the 1963 health interview survey conducted by the National Opinion Research Center (NORC) and the Center for Health Administration Studies of the University of Chicago. They measure health as an index formed from the first principal component of the number of symptoms reported from a checklist of twenty common symptoms and individuals' self-evaluation of their general health as excellent, good, fair, or poor. Grossman (1976) employs the National Bureau of Economic Research-Thorndike sample, which contains data on high ability white males in 1968 and additional data in 1971. He employs the self-rated health variable just defined and scaled so that it is proportional to the average number of work-loss weeks due to illness of men in poor health relative to the average number of work-loss weeks due to illness of men in each of the three other categories. Lee (1982) focuses on all males in the National Longitudinal Survey of Men 1966. He uses a multiple indicator model in which self-rated health and the presence of health conditions that affect the kind or amount of work are indicators of the unobserved stock of health. Grossman and Benham (1974) and

Grossman (1976) obtain estimates by conventional two-stage least squares methods. Lee (1982) applies maximum likelihood methods to a specification that incorporates a multiple indicator model with measurement error and endogenous right-hand-side variables in each of the two structural equations.

All three studies report positive and significant effects of increases in better health in the wage equation. Grossman (1976) and Lee (1982) also find positive and significant wage coefficients in the health equation, but Grossman and Benham's (1974) coefficient is not significant. All three studies treat potentially endogenous variables as exogenous and impose questionable exclusion restrictions. For example, years of formal schooling is exogenous in the health and wage equations in all three studies. Grossman (1976) assumes that a correlate of obesity and an indicator of job satisfaction are exogenous in the health equation and do not enter the wage equation. He also excludes region and city size indicators from the health equation while including them in the wage equation. Grossman and Benham (1974) and Lee (1982) also employ this exclusion restriction. In addition, Lee includes indicator variable for nonwhites in the wage equation, while excluding it from the health equation. Grossman and Benham assume that the length of time that has elapsed since the last physical examination is an exogenous regressor in the health equation, while Lee assumes that assets are an exogenous variable in that equation.

Subsequent studies in this literature have estimated the wage equation but not the health equation. For example, Schultz and Tansel (1997) report that the number of disability days, instrumented by local area food prices and health services availability, has negative and significant effects on wage rates in Ghana and Côte d'Ivoire. Since the data are cross-sectional,

area level fixed effects, which potentially are correlated with the wage rate and the instruments, cannot be included in the model.

B. Compensating Wage Differential Model

In the compensating wage differential model, consumers tradeoff an increase in job risk, reflected by the probability of a fatal or nonfatal accident or injury, in return for a higher wage rate. Since the production of safety is costly, firms are willing to reduce the accident probability only if they can offer a lower wage rate. Market equilibrium is determined by the point of tangency between a given worker's expected utility function and a given firm's isoprofit curve. Since firms and consumers differ, the connection of these points of tangency generates an upward sloping function termed the equilibrium market wage-risk function or the hedonic wage function. As discussed in detail by Rosen (1974, 1986) and Viscusi (1993, 2014), the complete model consists of this function, a demand function for safety by consumers, and a supply function of safety by firms.

For our purposes, the market equilibrium function is the most relevant one. Typically, the wage is measured at the individual level, but the risks are injury or death rates at the occupation or industry level. Extensive reviews of this literature are contained in Rosen (1986) and Viscusi (1993, 2014). The finding that an increase in job risk is associated with an increase in the wage is universal, once one controls for worker characteristics. Almost all of these studies treat job risk as exogenous because they are imbedded in a model that views the equation that is estimated as one that depicts market equilibrium.[7] The study by Kniesner, Viscusi, Woock, and

[7] Based on an estimation strategy proposed by Rosen (1974), a number of authors have estimated the demand function or the supply function that complete the model. These estimates first compute marginal prices from the market equilibrium equation and then treat it and safety as endogenous variables. For reviews of these studies and a new estimation procedure that has not yet been implemented at the empirical level, see Ekeland, Heckman, and Nesheim (2004) and Heckman, Matzkin, and Nesheim (2010).

Ziliak is one exception. They use panel data for men ages 18 through 65 in the Panel Study of Income Dynamics for the years 1993, 1995, 1997, 1999, and 2001. Since they employ a first-difference specification, most of the variation in fatality risk is due to job changes, which may be caused by wage changes. Hence, they instrument the first difference of the fatality risk with lagged levels and differences of that variable.

We modify the compensating wage differential framework because our health measures are specific to an individual rather than to an industry or occupation and because we examine the health-wage relationship in a relatively young panel in which job changes are rare. Hence, a person who accepts a relatively high wage offer in one period may incur a reduction in health in a subsequent period because the offer he or she accepted was associated with health risks. That suggests a negative effect of the lagged wage on current health.

On the other hand, the acquisition of a chronic condition that shortens life expectancy may lead to an increase in effort in an attempt by the head of a household to provide for his or her family, which may be associated with promotions and higher wages in future periods. Moreover, a large amount of effort in one period may be required to obtain promotions and the wage increases that accompany them in subsequent periods. That effort may cause reductions in health and to a negative effect of health in the previous period on the current wage. The metaphor of "working oneself to death" is relevant here. Indeed, Inoue and Specia (2017) describe the death of a thirty-one year old female journalist for Japan's state-run TV network from congestive heart failure in July 2013. She died after working 159 hours of overtime during the previous month in order to cover two local elections in Tokyo. Her death, termed karoshi or "death from overwork" by the Japanese, has been widely recognized since the late 1980s in

Japan. Cases first were reported among blue-collar workers, but they also have occurred among white-collar executives.

C. Summary

Both the human capital model and the compensating wage differential model generate relationships between health and the wage rate. In the pure investment component of the former model, an increase in the wage causes health to increase as long as the own time of the investor is not the only input in the health production function. This prediction becomes ambiguous in a mixed consumption-investment model because the relative price of health may increase substantially if its production is much more time intensive than the production of other household commodities. In addition, in the human capital model an increase in health should increase investments in knowledge and hence the wage after the completion of formal schooling because more time is available for market and nonmarket production and because the number of periods over which returns can be collected grows. On the other hand, the compensating differential model generates negative causal effects from health to the wage and from the wage to health. Current period reductions in health might have been avoided if a lower initial wage offer had been accepted. Moreover, large amounts of effort in previous periods might result in reductions in health in that period and in raises and promotions in the current period.

By implementing dynamic panel data models that allow lagged levels of health and the wage to be specified as instruments for differences of right-hand side measures of these variables on theoretical grounds, we relax the arbitrary identification assumptions in the studies by Grossman and Benham (1974), Grossman (1976), and Lee (1982). We improve on the subsequent literature by using comprehensive measures of health and obtaining health equations as well as wage equations. Finally, by incorporating insights from the compensating differential

literature, we make a stronger case as to why there may be relationships between health and the wage rate. At the same time, we justify why we are somewhat agnostic about the signs of these relationships.

III. Specification of Structural Equations

Consider a panel of n individuals, all of whom have completed their formal schooling. Let W_{it} be the wage rate or the log of that rate of the ith person in period or time t and H_{it} be a measure of the health of that person defined such that it is a positive correlate of better health. Omitting intercepts, period indicators, and period-varying observed exogenous variables, we specify the wage and health equations as follows:

$$W_{it} = \alpha_1 H_{it-1} + \alpha_2 W_{it-1} + f_i + u_{it} \quad (1)$$

$$H_{it} = \beta_1 W_{it-1} + \beta_2 H_{it-1} + g_i + v_{it}. \quad (2)$$

In these equations f_i and g_i are unobserved fixed effects (effects that do not vary by period) and u_{it} and v_{it} are unobserved effects that vary by period (unobserved time-varying effects).

We use equations (1) and (2) to examine the extent of two-way causality between health and the wage rate just outlined. They incorporate the plausible assumption that the causal effects (α_1 and β_1) occur with a lag. They also allow for lagged effects of the dependent variable in each equation because the presence of rising marginal costs of investment imply that the optimal stock of health and knowledge are reached gradually rather than instantaneously (Ehrlich and Chuma 1990; Galama and van Kippersluis 2016) as well as for reasons specified in Section II.[8] Inclusion of unobserved fixed effects (f_i and g_i) and with period-varying unobserved effects (u_{it} and v_{it}) that may be correlated with each other and with the observed variables incorporates biases due to such third variables as time preference.

[8] Technically, we follow the notions of causality developed by Granger (1969) by exploring, for example, whether past health predicts the current wage, with past health held constant.

We use standard dynamic panel methods (for example, Arellano 2003; Arellano and Bond 1991; Arellano and Bover 1995; Baltagi 2008; Blundell and Bond 1998) to address the issues just mentioned. First we take the first difference of each equation to eliminate the period-invariant fixed effects:

$$(W_{it} - W_{it-1}) \equiv \Delta W_{it} = \alpha_1(H_{it-1} - H_{it-2}) + \alpha_2(W_{it-1} - W_{it-2}) + (u_{it} - u_{it-1}) \quad (3)$$

$$(H_{it} - H_{it-1}) \equiv \Delta H_{it} = \beta_1(W_{i-1t} - W_{it-2}) + \beta_2(H_{it-1} - H_{it-2}) + (v_{it} - v_{it-1}). \quad (4)$$

Following standard practice in the dynamic panel methods literature, we assume that current values of the wage and health are correlated with current and past values of disturbance terms but not with future values. Given those assumptions, equations (3) and (4) cannot be estimated by ordinary least squares. One consideration is that the lag of the first difference of the dependent variable in each equation is correlated with the disturbance term in that equation. For example, $\Delta W_{it-1} \equiv (W_{it-1} - W_{it-2})$ is correlated with Δu_{it} because W_{it-1} is correlated with u_{it-1}. Along the same lines, the lagged first difference of health in equation (4) is correlated Δv_{it}. A second consideration is that the lagged first difference of health in equation (3) and the lagged first difference of the wage in equation (4) are correlated with the respective disturbance term in each equation because u_{it-1} and v_{it-1} are correlated.

Given these considerations, we use the second lag of health (H_{t-2}) and longer lags of that variable as instruments for the lag of the first difference of health at time t in estimating the two equations. Those variables are not correlated with Δu_{it} or with Δv_{it}. For the same reason, we use the second lag of the wage and longer lags of the wage as instruments for the lag of the first difference of that variable at time t.[9]

[9] Both H_{t-1} and W_{t-1} are predetermined variables in equations (1) and (2) because they are correlated with past values of the disturbance term in each equation but not with the current value of that term. Technically, since H_{it-1} is a predetermined variable, it is correlated with u_{it-2} and longer lags of the disturbance term but is not correlated with u_{it}

IV. Data and Measurement of Variables

Our panel data come from the National Longitudinal Survey of Youth 1997 (NLSY97). This is a sample of the United States population consisting of 8,984 individuals who were ages 12-16 as of December 31, 1996. Two subsamples make up the NLSY97 cohort. The first is a nationally representative sample of 6,748 respondents. The second consists of 2,236 oversampled black non-Hispanics and Hispanics. The cohort was interviewed each year from 1997 through 2011 and every other year starting in 2013. Since the maximum number of health measures are available in the period from 2007-2011, we limit our sample to individuals who reported data in all five of those years, had completed their formal schooling by 2006, and were in the labor force throughout that period. Our working data set contains 3,760 persons and approximately 18,800 person-years. A large majority of the sample range in age from 23 through 27 in 2007 and from 27 through 31 in 2011.

We employ six dependent variables: the log of the hourly wage rate and five alternative measures of health. The wage rate itself is the one reported by the respondent on his or her full- or part-time job. If a respondent has a full-time and a part-time job, then the wage from the full-time job is used. If he or she has multiple full-time jobs, the simple average of the full-time jobs is used. If a respondent has multiple part-time jobs and no full-time jobs the simple average of the part-time jobs is used.

The health measures are self-rated health, an indicator of whether health limits the kind of work a respondent can do, an indicator of whether health limits the amount of work a

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and u_{it-1}. Strictly speaking that assumes there would be no need to instrument for the lagged difference of health in equation (3). Since, however, it is unrealistic to assume that u_{it-1} and v_{it-1} are uncorrelated, we treat the lagged of difference of H_{it} in equation (3) in the same manner as we treat the lagged difference of W_{it} in that equation, and instrument it with the second lag of H_{it} and longer lags. Along the same lines, we instrument the lagged wage in equation (4) with the second lag of that variable and longer lags.

respondent can do, the number of times a respondent was injured or ill in the past year and not treated by a physician or a nurse, and the number of times a respondent was injured or ill and was treated by a physician or a nurse. The last two variables range from zero through four or more. We assign a value of four to the top-coded category. Except for self-rated health, all of these measures are negative correlates of better health.

Self-rated health has five categories: excellent, very good, good, fair, and poor. We scale this variable using a procedure developed by Grossman (1972b, 1976). He considers the number of healthy work days (D in a year as the output produced by health capital (H) and notes that this output has a finite upper limit of 250 days under the assumption that individuals work five days a week for fifty weeks. Then a plausible form of the production function of D is

$$D \equiv 250 - S = BH^{-C}, \tag{5}$$

where S is the number of work-loss days due to illness or injury and B and C are positive constants. Solve equation (5) for H to obtain

$$H = B^{1/C}S^{-1/C}. \tag{6}$$

Let S_P, S_F, S_G, S_V, and S_E be mean work-loss of people in poor, fair, good, very good, and excellent health, respectively. Grossman views health capital as a units-free measure in an index number form with $H_P = 1$. Hence, $H_F/H_P = (S_P/S_F)^{1/C}$, etcetera.

Work-loss days are not reported in NLSY97, but they are reported in the U.S. National Health Interview Survey. We pool survey years 2005 through 2013 to obtain $H_F/H_P = 2.01$, $H_G/H_P = 4.33$, $H_V/H_P = 6.72$, and $H_E/H_P = 9.57$. That series assumes that the unknown constant C in equation (6) is equal to one. To relax that assumption, the natural logarithm of the scaled self-rated health measure is the right-hand side variable in the estimation of equation of the wage equation and the left-hand side variable in the estimation of the health equation. In the former

case, the regression coefficient and its standard errors are divided by 1/C, and in the latter case, they are multiplied by C. Since C is an unknown constant, tests of significance are unaffected.

Means and standard deviations of the six dependent variables are contained in Table 1. These summary statistics are not weighted to correct for the oversampling of minorities because we estimate unweighted regressions in the next section. These estimates are very similar to weighted regressions. In addition, we fit separate regressions for whites, black non-Hispanics, and Hispanics after presenting our main results.

In addition to presenting the standard deviation of each variable in Table 1, we decompose that measure into between- and within-person components. That decomposition is important because our estimates would be biased if the within-person variation was small relative to the between person variation (Blundell and Bond 1998). According to the table, that decidedly is not the case. In almost all cases, the within component is at least as large as the between component.

Table 2 contains key characteristics of our sample, including potential independent variables. We do not control for observed time-invariant variables in the regressions because these variables drop out when first differences are taken. As, however, just indicated, we do fit separate models by race-ethnicity after we obtain our main results. We do the same for gender and education. We include survey year and age indicators (ten age indicators, with age 22 the omitted category) in the health equations and survey year and potential experience indicators (14 indicators with 0 the omitted category) in the wage equations. We omit the other time-varying variables in Table 2 (married, spouse present; number of kids; own income excluding earnings; and spouses' income) because these variables are endogenous and because we want to focus on

the two key endogenous variables in our model. We note, however, that our results are not affected when these variables are included and treated as exogenous.

V. Results

Arellano-Bond dynamic panel estimates of the generalized methods of moments (GMM) first-differenced health and wage equations are contained in Tables 3 and 4, respectively. Since there are five alternative health measures, five regressions are shown in each table.[10] Of the five observations for each person, the first one is lost because of the inclusion of the first lag of the wage and health in the specification of the structural level equations. The second one is lost by taking first differences. Hence, three observations remain: the first difference as of 2009, the first difference as of 2010, and the first difference as of 2011.

The estimates in each table employ up to three lags of health and up to three lags of the wage as instruments. In the first period (2009), the only available instruments are the second lags of these two variables—their values as of 2007. In the second period (2010), the second and third lags are available (values as of 2008 and 2007). In the third period (2011), all three lags are available (values as of 2009, 2008, and 2007).

Blundell and Bond (1998) point out that the performance of the GMM first-difference estimator has a large finite sample bias and poor precision when the autoregressive parameters are relatively large and the number of time series observations is small. Lagged levels of the series provide weak instruments for first differences in this case. They suggest using an extended GMM estimator that employs lagged differences as instruments for equations in level form, in addition to lagged levels as instruments for equations in difference form.

[10] Estimates were obtained using the xtabond2 routine is *Stata* (Roodman 2009).

Before obtaining the estimates in Tables 3 and 4, we explored this issue by estimating an exactly identified version of our model. In this version, there are only two instruments: the second lag of health and the second lag of the wage. Since the equations are exactly identified, the Arellano-Bond first difference procedure is identical to conventional two-stage least squares. Following Blundell and Bond (1998), our evaluation is based on the first stage of the exactly identified model. Consider the structural equations for W_{it-1} and H_{it-1}. Subtract W_{it-1} from each side of the first equation, and subtract H_{it-1} from each side of the second equation to obtain

$$W_{it-1} - W_{it-2} = \alpha_1 H_{it-2} + (\alpha_2 - 1)W_{it-2} + f_i + u_{it-1} \quad (8)$$

$$H_{it-1} - H_{it-2} = \beta_1 W_{it-2} + (\beta_2 - 1) H_{it-2} + g_i + v_{it-1}. \quad (9)$$

Equations (8) and (9) constitute the first stage. Note that the fixed effects appear in the equations, and the second lag of the wage and the second lag of the health are correlated with those effects. But those two variables are not correlated with the disturbance terms in equations (3) and (4), which are $u_{it} - u_{it-1}$ and $v_{it} - v_{it-1}$, respectively. In this sense, the second lags are valid instruments.

Blundell and Bond show that parameter estimates of α_2 -1 and $\beta_2 - 1$ are inconsistent because the second lags of the wage and health are correlated with the fixed effects. The biases or inconsistencies are upwards towards zero and are particularly severe as α_2 and β_2 approach one and/or if the variance in the fixed effect relative to the variance in the time-varying disturbance term approaches infinity. In either case, an F-test of the significance of the two instruments as a set should have a small value.

The relevant F-ratios are reported in the last two rows in Tables 3 and 4. In the former table they range from 93.67 to 96.05 in the first-stage wage equation and from 163.50 to 851.2 in the first-stage health equation. In Table 4 the corresponding range is from 81.12 to 84.06 in the

first-stage wage equation and from 154.30 to 749.50 in the first-stage health equation. Clearly, these F-ratios are very large, and they reject the hypothesis that the instruments are weak. Moreover, the coefficients of α_2 -1 in the wage equation and $\beta_2 - 1$ in the health equation are nowhere near 0. The values of α_2 -1 are all -0. 40 in the first-stage wage equation ($\alpha_2 = 0.60$) and range from -0.45 to -0.71 in the first-stage health equation (β_2 ranges from 0.29 to 0.55) that correspond to the F-ratios in Table 3. These values are almost the same in the first stage wage regression and health regressions that correspond to the F-ratios in Table 4. In addition, as shown in Table 1, the within-person variation in our outcomes is at least as large as the between-person variation in almost all cases. In short, there is little evidence that our estimates would be improved by estimating the level equation. Since that procedure would greatly increase the number of instruments, we do not pursue it. Finally, we note that the key coefficients of interest—the cross effect of past health on the current wage (α_1) and the cross effect of the past wage on current health (β_1) are very similar in the two-stage least squares model and in the Arellano-Bond mode with up to three lags of the wage and health as instruments.

Turning to the estimated health equations in Tables 3, one sees that the coefficients of β_1 never are significant. In words, we have no evidence that a change in last period's wage has an effect on health this period. Why might that be the case? One explanation is that the reasons for causality that flow from the human capital and the compensation wage differential model simply are not relevant. Another explanation, and one that is perhaps more sympathetic to these models, is that they suggest effects that go in opposite directions and that could offset each other. For example, in the human capital model, the positive effect of the wage on health could be small or nonexistent if the share of the money price of medical care and other goods purchased in the market in the marginal cost of health production is small. In that case the marginal rate of return

to an investment in health would be insignificant and might be swamped by substitution effects towards goods-intensive commodities in a mixed investment-consumption framework. Alternatively, positive effects in the human capital model might be offset by negative realizations of current period reductions in health by persons who accepted a relatively high wage in a previous period to accept a job with a higher than average expected health risk.

In Table 4, one sees that two of the five coefficients of α_1 are statistically significant. These are the ones associated with self-rated health and with the number of times a respondent was injured or ill and not treated. Keep in mind that the self-rated health measure is a positive correlate of better health so that both the negative coefficient of that variable and the positive coefficient of the frequency of untreated injury or illness indicate that a reduction in past-year health is associated with an increase in the current-year wage. This result is not consistent with the notion that an increase in health after the completion of formal schooling should increase further investments in knowledge via on-the-job training. It is consistent with one of our extensions of the compensating wage differential model in which a large amount of effort in one period is required to obtain promotions and the wage increases that accompany them in subsequent periods. That effort may cause reductions in health and to a negative effect of health in the previous period on the current period wage.[11]

One explanation for why we find the results for injured or ill and not treated and not for injured or ill and treated is that those treated contain two groups of individuals. The first group has serious injuries that force them to miss work for some period of time. This would cause lower wage rates, possibility because they invest less in on-the-job training or have lower levels

[11] In both Tables 3 and 4, the overidentification restrictions are accepted, and there is no evidence of second-order serial correlation in the residuals. First-order serial correlation in the difference equation is to expected if, for example, u_{it} and u_{is} ($s \neq t$) are uncorrelated since the disturbance in the first difference equation at times t and t-1 share a common term ($u_{it-1)}$.

of effort. The second group does not miss work and like the untreated group has higher levels of effort that lead to higher wages.

In Tables 5 and 6, we examine how the negative cross effects of past health on the current wage vary by groups classified by education, gender, and ethnicity. We focus on the two significant effects for the population at large because there are no cross effects of the lagged wage on current health for these groups. In addition, the only significant lagged health effects pertain to those involving self-rated health and the frequency of untreated illness or injury.[12]

In Table 5, we compare individuals with a high school education or less to those with some college or more and males versus females. While the self-rated health status effect is not significant for either education group, it is bigger for the more educated group, bigger for that group than for than for the full sample, and significant at $p < 0.16$. On the other hand, the coefficient of the number of times injured or ill and not treated, while positive and significant for both groups, is larger for the less educated than for the more educated. One explanation of these results is that the less educated tend to be in employed in more risky and physically demanding blue-collar occupations, while the more educated tend to be found in mentally demanding white-collar occupations. Hence, higher levels of effort in a prior period require more physical labor on the part of the less educated and result in untreated injuries or accidents in that group. On the other hand, these higher levels make more mental demands on the more educated and result in reductions in their self-rated health. In either case, compensating wage increases are realized in subsequent periods.

A similar picture emerges when females are compared to males. The four health coefficients are negative and significant for both groups. But the self-rated health effect is larger

[12] The estimates in Tables 5 and 6 include the lag of the first difference of the wage, but its coefficients are not shown.

for females than for males, while the untreated injury or illness effect is larger for males. This is consistent with greater prevalence of males in blue collar occupations.

In Table 6, we compare white non-Hispanics, black non-Hispanics, and Hispanics.[13] The self-rated health coefficient is negative and significant for whites but not for the other two groups. Moreover, the magnitude of this coefficient is almost two and a half times as large as it is in the pooled sample. While all three coefficients of untreated illness or injury measure are positive and significant, they are bigger for black non-Hispanics and Hispanics than for whites. While the black coefficient is estimated imprecisely, the Hispanic coefficient is significant, twice as large as the white coefficient, and almost 70 percent larger than the black coefficient. Again, these results are consistent with occupational patterns in which whites are more likely to have white-collar jobs than minorities. The larger untreated illness and injury effect for Hispanics compared to blacks also is consistent with surveys in which Hispanics are more likely to believe in the efficacy of hard work than the population at large (for example, Taylor, Lopez, Martínez, and Velasco 2012).

VI. Discussion

We have uncovered a causal relationship between health and the wage in which a reduction in health leads to an increase in the wage rate in a panel of U.S. young adults who had completed their formal schooling by 2006 and were continuously employed from that year through 2011. There is no evidence of a causal relationship running from the wage rate to health in this panel. The former result is consistent with an extension of the compensating wage differential model in which a large amount of effort in one period is required to obtain promotions and the wage increases that accompany them in subsequent periods. That effort may

[13] We include the very small percentage of the sample who are in the other race, non-Hispanic category with whites. Exclusion of them from the white sample does not alter our results.

cause reductions in health and to a negative effect of health in the previous period on the current period wage. The latter result may suggest that forces that go in opposite directions in the human capital and compensating wage differential models may offset each other.

A good deal of caution is required in interpreting our finds and in extrapolating them to the population at large. Clearly, the results pertain to a selected sample. The panel members were at the early stages of their working careers during the period at issue and were continuously employed during the Great Recession. The realization that many of their colleagues were unemployed or could not find jobs may have caused them to increase their work effort, which resulted in reductions in health but led to subsequent wage increases. This may be one subtle factor in the findings in some studies that positive correlates of health rise in a recession (for example, Ruhm 2000, 2003, 2005; Cutler, Huang, and Lleras-Muney 2016). Another consideration is that the interpretation of our main result—that reductions in health cause increases in the wage because these increases are associated with high levels of past effort—needs to be investigated in detail.

We view the last item as challenging one on an agenda for future research. Work effort surely is related to hours of work, but the latter is not a perfect correlate of the former. In addition, effort may have different effects in different industries and occupations and on different measures of health as our results suggest. Finally, effort and hours of work surely are endogenous variables. Hence, in the short and intermediate runs, priority should be given to obtaining estimates in a longer panel than the one we have employed. That panel should contain persons of all ages and should have the ability to examine how estimates differ in recessions compared to expansions.

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Table 1: Means and Within and Between S.D. of Endogenously Treated Variables

Dependent Variable	Mean	S.D. Overall	S.D. Between	S.D. Within	Observations	Respondents
Log Wage	2.61	0.65	0.59	0.37	13630	3423
Self-Rated Health	1.68	0.49	0.39	0.31	18795	3760
Limited in the Kind of Work	0.07	0.25	0.19	0.17	18756	3760
Limited in the Amount of Work	0.07	0.25	0.18	0.17	18762	3760
# of Time Injured or Ill and not Treated by a Doctor or Nurse	0.72	1.21	0.81	0.90	18737	3760
# of Times Injured or Ill and Treated by a Doctor or Nurse	0.55	1.04	0.72	0.76	18771	3760

Note: Sample includes respondents from 2007 to 2011 who have completed education and who are not missing a wave. Wages are based on current fulltime or part-time jobs. Self-rated health is based on the question "In general, how is your health? Excellent, Very Good, Good, Fair, or Poor?" scaled by the predicted work-loss days from NHIS which has the same question. Specifically, each category is scaled as the natural log of each category's predicted work-loss days relative to the poor category [e.g. ln(predicted poor work-loss days/predicted Excellent work-loss days)). # of times injured or ill and not treated, and # of time injured or ill and treated are both range from 0 to 4+, where 4+ is a top code and is set equal to 4.

Table 2: Sample Characteristic Mean and S.D.

Variable	Mean	S.D.	Minimum	Maximum
White	0.47	0.50	0.00	1
Black, Non-Hispanic	0.27	0.44	0.00	1
Hispanic	0.23	0.42	0.00	1
Other Race, Non-Hispanic	0.03	0.17	0.00	1
Female	0.47	0.50	0.00	1
High School or Less	0.65	0.48	0.00	1
Some College, Less than Bachelor's Degree	0.20	0.40	0.00	1
Bachelor's Degree or Higher	0.16	0.36	0.00	1
Age	26.99	1.99	22	32
Potential Experience in Years	6.53	2.91	0.00	14.00
Wave=2008	0.25	0.43	0.00	1.00
Wave=2009	0.25	0.43	0.00	1.00
Wave=2010	0.25	0.43	0.00	1.00
Wave=2011	0.25	0.43	0.00	1.00
Married and Spouse Present	0.31	0.46	0.00	1.00
Own Income Excluding Earnings	1031.15	7312.62	-6900	198533
Spouse's Income	13197.05	22924.34	0.00	250000
Number of Kids	1.09	1.23	0.00	8

Table 3: Arellano-Bond Estimates of the Effects of Last Year's Health and Wage on This Year's Health

	Self-Rated Health	Health Limits Kind of Work	Health Limits Amount of work	Number of Times injured or Ill and not Treated	Number of Times Injured or Ill and Treated
Lag Health	0.0347	0.0519	-0.00650	0.0709***	0.142***
	(0.0280)	(0.0508)	(0.0517)	(0.0223)	(0.0251)
Lag Wage	-0.00994	-0.00217	0.0113	0.0148	0.000404
	(0.0207)	(0.0104)	(0.00975)	(0.0498)	(0.0454)
N	7121	7105	7108	7092	7115
Hansen Overidentification P-Value	0.148	0.303	0.393	0.0950	0.838
AB test for AR(2) P-Value	0.223	0.637	0.490	0.733	0.344
First Stage Wage Excluded Instruments F-Stat	96.05	94.49	93.67	95.41	94.45
First Stage Heath Excluded Instrument F-Stat	531.2	197.6	163.5	851.2	663.6

Note: * p<.1, ** p<.05, *** p<.01. Estimates based on Arellano-Bond using difference equations only. S.E. is clustered at the individual level. We use a maximum lag length of 3, but results are not sensitive to lag length. Reported first stage F-statistics are based on first stage regressions that are exactly identified with the second lag of the level of health and log wages used to predict the lag difference of health and log wages. Exogenous controls include age and wave dummies, although results are not sensitive to inclusion or exclusion of age. They are also not sensitive to inclusion or exclusion of marital status, number of kids, own income excluding earnings, and spouses' income, treated exogenously.

Table 4: Arellano-Bond Estimates of the Effects of Last Year's Health and Wage on This Year's Wage

	Self-Rated Health	Health Limits Kind of Work	Health Limits Amount of Work	Number of Times Injured or Ill and not Treated	Number of Times Injured or Ill and Treated
Lag Wage	0.0955***	0.0955***	0.0928***	0.0931***	0.0959***
	(0.0355)	(0.0351)	(0.0358)	(0.0350)	(0.0351)
Lag Health	-0.0479*	-0.00231	-0.0276	0.0209**	0.000995
	(0.0289)	(0.0576)	(0.0723)	(0.00818)	(0.00959)
N	6331	6323	6324	6318	6331
Hansen Overidentification P-Value	0.727	0.793	0.554	0.514	0.736
AB test for AR(2) P-Value	0.383	0.385	0.378	0.354	0.386
First Stage Wage Excluded Instrument F-Stat	84.06	82.02	81.12	83.04	82.36
First Stage Heath Excluded Instrument F-Stat	481.0	179.5	154.3	749.5	605.8

Note: * p<.1, ** p<.05, *** p<.01. Estimates based on Arellano-Bond using difference equations only. S.E. is clustered at the individual level. We use a maximum lag length of 3, but results are not sensitive to lag length. Reported first stage wage F-statistics are based on first stage regressions that are exactly identified with the second lag of the level of health and log wages used to predict the lag difference of health and log wages. Exogenous controls include potential experience and wave dummies, although results are not sensitive to inclusion or exclusion of potential experience. They are also not sensitive to inclusion or exclusion of marital status, number of kids, own income excluding earnings', and spouses' income, treated exogenously.

Table 5: Arellano-Bond Estimates of the effect of Last Year's Health on this Year's Wage by Education and Gender

	Self-Rated Health				Number of Times Injured or Ill and not Treated			
	High School or Less	Some College or More	Male	Female	High School or Less	Some College or More	Male	Female
Lag Health	-0.0239	-0.0569	-0.0264	-0.0815**	0.0253**	0.0176*	0.0238*	0.0182*
	(0.0388)	(0.0400)	(0.0419)	(0.0389)	(0.0121)	(0.0101)	(0.0122)	(0.0105)
N	3468	2863	3657	2674	3459	2859	3650	2668

Note: * p<.1, ** p<.05, *** p<.01. Estimates based on Arellano-Bond using difference equations only. Coefficients of lag wage not shown. S.E. is clustered at the individual level. We use a maximum lag length of 3, but results are not sensitive to lag length. Exogenous controls include potential experience and wave dummies, although results are not sensitive to inclusion or exclusion of potential experience. They are also not sensitive to inclusion or exclusion of marital status, number of kids, own income excluding earnings, and spouses' income, treated exogenously.

Table 6: Arellano-Bond Estimates of the Effect of Last year's Health on This Year's Wage by Race-Ethnicity

	Self-Rated Health			Number of Times Injured or Ill and not Treated		
	White	Black	Hispanic	White	Black	Hispanic
Lag Health	-0.116***	-0.0292	0.0474	0.0170*	0.0210	0.0352*
	(0.0422)	(0.0572)	(0.0484)	(0.00985)	(0.0224)	(0.0197)
N	3546	1336	1449	3459	1334	1448

Note: * p<.1, ** p<.05, *** p<.01. Estimates based on Arellano-Bond using difference equations only. Coefficients of lag wage not shown. S.E. is clustered at the individual level. We use a maximum lag length of 3, but results are not sensitive to lag length. Exogenous controls include potential experience and wave dummies, although results are not sensitive to inclusion or exclusion of potential experience. They are also not sensitive to inclusion or exclusion of marital status, number of kids, own income excluding earnings, and spouses' income, treated exogenously.