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Private Insurance and Uninsured

The Effect of Private Insurance on the Health of Older, Working Age Adults: Evidence from the Health and Retirement Study

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Objective. Primarily, to determine if the presence of private insurance leads to improved health status, as measured by a survey-based health score. Secondarily, to explore sensitivity of estimates to adjustments for endogeneity. The study focuses on adults in late middle age who are nearing entry into Medicare.

Data Sources. The analysis file is drawn from the Health and Retirement Study, a national survey of relatively older adults in the labor force. The dependent variable, an index of 5 health outcome items, was obtained from the 1996 survey. Independent variables were obtained from the 1992 survey. State-level instrumental variables were obtained from the Area Resources File and the TAXSIM file. The final sample consists of 9,034 individuals of which 1,540 were uninsured.

Study Design. Estimation addresses endogeneity of the insurance participation decision in health score regressions. In addition to ordinary least squares (OLS), two models are tested: an instrumental variables (IV) model, and a model with endogenous treatment effects due to Heckman (1978). Insurance participation and health behaviors enter with a lag to allow their effects to dissipate over time. Separate regressions were run for groupings of chronic conditions.

Principal Findings. The OLS model results in statistically significant albeit small effects of insurance on the computed health score, but the results may be downward biased. Adjusting for endogeneity using state-level instrumental variables yields up to a six-fold increase in the insurance effect. Results are consistent across IV and treatment effects models, and for major groupings of medical conditions. The insurance effect appears to be in the range of about 2–11 percent. There appear to be no significant differences in the insurance effect for subgroups with and without major chronic conditions.

Conclusions. Extending insurance coverage to working age adults may result in improved health. By conjecture, policies aimed at expanding coverage to this population may lead to improved health at retirement and entry to Medicare, potentially leading to savings. However, further research is needed to determine whether similar results are found when alternative measures of overall health or health scores are used. Future

research should also explore the use of alternative instrumental variables. Preliminary results provide no justification for targeting certain subgroups with susceptibility to certain chronic conditions rather than broad policy interventions.

Key Words. Insurance, health scores, health status, endogeneity bias, instrumental variables

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According to the U.S. Census Bureau, 45 million Americans were without health insurance in 2003 and their numbers continues to grow. Health benefits in the U.S. remain heavily employer-based with about 60 percent of all insured individuals being enrolled through employer-sponsored plans. While the indigent can often qualify for public assistance programs such as Medicaid, many participants in the labor force may not have adequate access to coverage through their employer. Although it is possible to purchase individual plans privately, these may only be available at prohibitively high rates compared with the group rates available through large employers. As a consequence, 16 percent of full-time workers are uninsured (Fronstin 2003). Not surprisingly, much of the policy discussion focuses on ways of expanding coverage to all workers. In 2005, Congress enacted Health Savings Accounts to allow small employers and their employees to purchase high deductible insurance plans at low premiums. During the last presidential election campaign the Bush administration proposed a tax credit of up to $1,000 to help low income workers purchase insurance and health savings accounts, while the Kerry–Edwards campaign favored expanding employer-based coverage by allowing private buy-ins through the health insurance program for Federal government employees or through Medicare.

The potential impact of health insurance of the health of the population is one important issue that underlies the policy debate. Evidence to this effect may provide further support in favor of policies designed to expand health insurance coverage, irrespective of the policy mechanism ultimately chosen. In a recent review of the literature, Hadley (2003) found broad evidence of positive impacts in studies based on observational data. The magnitude of

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these effects, however, varied with the type of population sampled, type of illness and the particular measure of health outcomes chosen and thus merits further investigation. Most of the previous research focused on mortality as the outcome measure, rather than actual measures of health status. Moreover Hadley notes the paucity of research that adjusts for endogeneity of insurance participation in health equations, suggesting that previous studies may have underestimated the insurance effect (p. 43). In this paper, we attempt to fill both of these gaps. First, we employ a composite health score as our outcome measure, rather than the more commonly used mortality probabilty (e.g., Franks, Clancy, and Gold 1993; Sorlie et al. 1994). Second, we attempt to adjust for potential endogeneity. Our identification approach is similar to that of Goldman et al. (2001), who focused on the effect of Medicaid on declines in mortality probabilities for HIV-positive individuals. However, given our interest in employment related insurance we focus on working-age adults. While our findings are in agreement with Goldman et al. in that both studies yield greater insurance effects after adjusting for endogeneity, we are also careful to point out difficulties in statistical identification inherent in such models and we note the possible range of estimates for the insurance effect. With this caveat noted, our results provide deeper evidence that expanding insurance will have direct benefits in terms of improved health outcomes. The rest of the paper proceeds as follows: the second section summarizes the relevant literature on health scores, and on the effect of insurance on outcomes. The third section presents the methodological approach and estimation framework. The fourth section presents data and variable definitions. The fifth section presents results from the insurance participation equation and the health status equation for the full sample and a summary of number of tests for endogeneity bias. The sixth section replicates this analysis for subsets of survey respondents based on groupings of chronic conditions. This was done in order to test whether insurance effects are repeated across various settings in which symptoms of the underlying medical condition may not be equally observable to the individual. Finally, implications and limitations of the results are discussed in the last section.

Previous Literature

Previous literature on the impact of insurance on health has tended to focus on mortality as the outcome measure of choice. In this study we focus instead on a now commonly accepted health scoring methodology, similar in content to the physical component summary scale of the SF-36 and SF-12 (Ware and Kosinski 2001). Below we briefly review the literature on these measures. In

addition, we provide a brief review of the literature on insurance and mortality, including the issue of treating potential endogeneity bias.

Health Scores

There is a substantial body of literature on using survey-based measures of health status. These measures appear with similar wording in major household surveys such as the Medical Expenditures Panel Survey (MEPS), the Health and Retirement Survey, and the National Health Interview Survey. Indicators are generally classified into three types: Subjective measure, i.e., self rated overall health (poor, fair, good, very, excellent); objective measures based on a general criterion, especially physical limitation, defined as inability to perform certain tasks defined in the survey; and objective measures that pertain to self-reporting of specific diagnoses or medical conditions. In general, these measures have been shown to perform well. Perry and Rosen (2001, p. 19) find that "objective measures give exactly the same answer as subjective measures" when testing for differences in health status between wage earners and the self-employed. Specifically in the Health and Retirement Study, Hurd and McGarry (1995) find that subjective probabilities of survival vary with health predictors in the same way as actual outcomes.

By combining the variety of self-reported conditions into a single index the Medical Outcomes Study produced a health status measure or *physical summary scale* that minimizes individual error (Ware and Sherbourne 1992; Ware et al. 1995). The index, also known as the Short-Form 36 (SF-36), utilizes the same indicators of physical health as those found in large household surveys. The index avoids the use of self-reports on specific diagnoses, which have been shown to be sensitive to false negative errors in Canadian data (Baker, Stabile, and Deri 2001). Variables in the Health and Retirement Study render themselves to an approximation of the Physical Component Summary subscale of the SF-36 since it includes the same basic groupings of health indicators. Differences are found in the wording used to describe certain physical limitations or conditions. In the fourth section, we provide further detail regarding construction of this variable in the HRS and its validation. Past research has demonstrated that the Physical Component Summary scale is strongly correlated with the presence of symptoms (e.g., shortness of breath, chest pain) and inability to work because of health problems (McHorney et al. 1994; Ware et al. 1995). Prior studies on the impact of insurance on physical health tended to focus on components of the physical summary scale rather than the combined score, with conflicting results. Mirowsky and Ross (1998)

find no significant effects of private or public health insurance on physical outcomes. On the other hand, Baker et al. (2001) find positive and significant effects of private insurance on self-reported health and physical functioning for working age adults, after allowing for appropriate lags. Again, the endogeneity issue was not explicitly addressed in any of these studies.

Effect of Insurance on Mortality

Several studies have used proportional hazard models to estimate the effect of private, employer-based insurance on mortality after adjustment for age, sex, income, and comorbidities. Using data from the National Health and Nutrition Survey, Franks, Clancy, and Gold (1993) found that between 1975 and 1987 the adjusted mortality rate for uninsured employed workers was about 25 percent higher compared with those with private employer-sponsored insurance. Sorelie et al. (1994) found similar effects using data from the National Longitudinal Mortality Study (NLMS). A similar study of women with breast cancer (Ayanian et al. 1993) found equally large insurance effects as the all-cause mortality studies. A number of prior studies on cause-related mortality uncovered much smaller effects, but these tended to be descriptive (see Hadley 2003) In the case of public insurance programs such as Medicare and Medicaid, only weak effects of insurance on adult or infant mortality were found (Sorlie et al. 1994; Kaestner, Joyce, and Racine 1999).

Goldman et al. (2001) found an anomaly when studying the impact of Medicaid on AIDS-related mortality, whereby Medicaid participation appeared to result in *higher* death rates. However after adjusting for endogeneity in a probit-IV model, they found that the sign of the Medicaid effect reversed, with Medicaid participation having an even larger impact on reduced mortality compared with private insurance in the earlier studies. Goldman et al. attribute this to the efficacy of newer prescription drugs not previously available even with insurance.

METHODOLOGICAL APPROACH

General Model

To address the potential endogeneity of insurance in the health equation, we estimate the following system of equations:

$$H_t = H(X_{t-k}, B_{t-k}, I_{t-k}, H_{t-k}, e) \tag{1}$$

$$I_{t-k} = I(F_{t-k}, v) \tag{2}$$

where H is the health index in period t, X denotes the socioeconomic characteristics, B denotes the health indicators and behavioral variables, I is the binary indicator of insurance, and F denotes the instrumental variables that predict the likelihood of insured but are uncorrelated with e, the error term in equation (1). Finally, k denotes the length of the lag. Equation (1) defines the health "production function" (Grossman 1972, 1976), while equation (2) is used to predict the likelihood of having insurance.[1] Equation (2) is the indicator function for purchasing insurance, which can be estimated as a probabilistic model. While interesting issues arise in conjunction with the insurance decision, our main interest is in creating a well-identified instrument. Thus the specification of the probability equation is incidental to our main research question.

The lag structure is used in equation (1) to allow for the fact that an adjustment to behavioral covariates does not occur instantaneously. For instance, smoking and alcohol abuse require some passage of time before causing adverse health effects.[2] The same can be assumed for health insurance. Including the lagged dependent variable, H_{t-k}, is consistent with the Grossman investment model, which states that current health depends on the initial level of health. For convenience, the lag is also applied to time invariant demographic characteristics such as gender, race, and marital status.

Although this model can be rewritten in the form of a change equation,[3] in our particular case we will stop short of interpreting it as such due to a definitional change in health variables that occurred between the 1992 and 1996 waves of the HRS. In 1996, HRS introduced a change in the phrasing of responses to a small subset of questions that make up the indicators of the health index. This caused a slight upward "creep" in the index. As a result, for some observations health status would appear to artificially increase over time. However, this does not affect the distribution of health status within each wave. Thus, the 1992 index provides a reasonable baseline measure of health status, with slightly altered scaling. Note that we are not interested in the magnitude of change in health status per se, but rather in the effect of insurance participation, holding everything else constant. More detail on the construction of the health index is provided in the fourth section.

Econometric Approach

The error terms associated with equations (1) and (2) may be correlated if there is some unobserved trait that makes people who purchase insurance more or less likely to be healthy in a future period. For instance, if insurance is positively correlated with an unobserved trait, say "awareness" and this trait also

leads a person to take better care of his health, then the error terms would be positively correlated. In this case the coefficient of insurance in the health equation would be *upward biased.* If on the other hand, insurance is positively correlated with an unobserved trait that also causes a person to neglect her health, e.g., reduce preventive effort, than error terms would be negatively correlated and the coefficient of insurance in the health equation would be *downward biased.* The final direction of the simultaneity bias cannot be ascertained a priori.

To address this issue, we use a two-step procedure, whereby we initially estimate equation (2) to obtain the predicted value of I_{t-k}, or some related transformation (see below) and then substitute this predicted value, or some related transformation (see discussion below) into equation (1). Note that the model is statistically identified since the vector of coefficients F_{t-k} is included in (2) but omitted from (1). The estimation procedure we use is essentially an OLS regression for equation (2) augmented by the hazard function from a probit regression for (1), i.e., the "treatment effects" model, due to Heckman (1978, 1979) and Maddala (1983). This model, often referred to as the *restricted control function* (RCF) method, is appropriate when the censoring of the nonparticipating group does not take place as it would in the standard Heckman selection model.[4] Moreover it is at least as efficient as its alternative, the instrumental variable estimator (Vella and Verbeek 1999).[5] The model can be summarized as follows:

$$H_i = \alpha + bI_i + \gamma Y_i + \sigma_{t\varepsilon} I\lambda_i + \sigma_{t\varepsilon}(1 - I)h_i + e_i \tag{3}$$

where H_i is health status or some other outcome measure such as expenditures for individual i, I_i is a binary indicator of being in the treatment group (in our case, = 1 if insured, = 0 if uninsured), Y_i is the vector of covariates, λ_i is the hazard rate of (having insurance) for individual in treatment group, h_i is the hazard (also of having insurance) for individuals in the nontreatment group (uninsured) obtained from the first stage probit on the treatment indicator (e.g., if individual is insured), and $\sigma_{t\varepsilon}$ is the covariance of the disturbance terms in the treatment function and the nonaugmented OLS equation. An algorithm by Maddala (1983, p. 122) provides adjusted standard errors.[6] The estimate of $\sigma_{t\varepsilon}$ provides a specification test for (3), with high statistical significance indicating that the null hypothesis (the nonaugmented OLS equation is true), should be rejected.[7]

RCF is appropriate when the distribution of the dependent variable is fully observed, but assignment to treatment groups is nonrandom. Unlike the standard Heckman selectivity bias model, where the hazard enters in lieu of

the treatment indicator, the RCF method includes the binary indicator in addition to the hazard rate. Thus, RCF allows for direct comparisons of α with the coefficient of the treatment indicator in a simple OLS model. Note that model (3) is very similar to a class of instrumental variable (IV) estimators in which the residuals from a linear probability regression on the indicator function are included in second stage equation in lieu of the hazard rate.[8]

Choosing one model over the other entails a tradeoff between making distributional assumptions about the errors and attaining consistency of the structural parameters of interest. The RCF assumes a bivariate normal distribution of the errors in the first and second stage equation, but yields consistent and efficient structural parameters. In comparison, the IV model is free of distributional assumptions, but the estimates may be inconsistent. Vella and Verbeek (1999) show that if the normality assumption is satisfied, IV and RCF are identical, and they recommend comparing estimates from both models. Our own comparison (e.g., Table 3) indicated that the two models yielded virtually identical estimates. We opted to present regression results from RCF since it allows for a more intuitive interpretation of the participation parameter as a "treatment" or intervention and because it provided corrected standard errors. Henceforth we refer to this model as the "treatment" model.

Instrumental Variables

Addressing the endogeneity issue requires creating an instrument for insurance participation in the health status equation. To satisfy identification requirements, Joyce (1999) used interaction between health districts and year (pre–post Medicaid expansions) to instrument for a Medicaid prenatal care program participation. Goldman et al. (2001) used area-level policy variables as predictors of insurance coverage for HIV-infected persons; they note that such variables are correlated with insurance availability via Medicaid for HIV patients, while they are uncorrelated with the individual's state of health. In this paper, we adopt the latter approach, noting that only state-level information could be matched to our data.[9] The validity of instruments was ascertained using Wald tests as described in Goldman et al. (2001). However since no definitive tests exists in the literature we caution that the instrumental variable approach provides an upper-bound to a range of estimates (Jaeger et al. 1995).

There is substantial literature to suggest that state-level marginal tax rates are uncorrelated with health but correlated with insurance participation (e.g., Royalty 2000; Gruber 2001). Therefore we obtained marginal tax rates from the TAXSIM program maintained by the National Bureau of Economic

Research. From the area resources file, we obtained two state-level variables, percent of all workers belonging to a union (unionization rate), and the unemployment rate. Unionization rate is correlated with the likelihood of being offered insurance coverage in the state; Lo Sasso and Meyer (2003) suggest unemployment rate as a negative predictor of private health insurance coverage in the market.

HRS Database and Variables

Our main analysis file was draw from the Health and Retirement Study (HRS), which is a household survey of mostly working age adults. As part of this survey, follow-up interviews were conducted every 2 years. For purposes of our research we focused on wave 1 and wave 3, corresponding to the years 1992 and 1996. In 1992, face-to-face interviews were conducted for 7,702 households, yielding a total of 12,652 individuals for wave 1 (1992). We focused our sample on adults 45–64 years old; few people below age 45 participated, and almost all participants age 65 and older at baseline were insured through Medicare. Because we were interested in the insurance choices available to participants in the labor force, we also excluded a small number of Medicaid and Medicare beneficiaries, and those enrolled through other federal health insurance programs (e.g., Veteran's Administration, CHAMPUS). Due to the lagged variable structure of our estimation procedure, we further considered only individuals who participated in both waves. This and a small number of omitted observations due to missing data resulted in a final sample size of 9,034 individuals. Of these 1,537 lacked insurance.[10]

Our main dependent variable is the *health index wave 3*, which is a summary measure of self-reported overall health, two measures of physical limitations (mobility and agility), and a measure of pain. All four components of this health index are coded so that higher values indicate better health.[11] The same structure is used to generate an independent *health index wave 1*, which provides a measure of baseline health status. The health index used in our analysis closely mirrors the construction of a widely used summary measure of physical health known as the SF-36. The health status domains measured and the weightings are similar to the Physical Component Summary scale of the SF-36. Past research has demonstrated that the Physical Summary Scale is strongly correlated with the presence of symptoms (e.g., shortness of breath, chest pain with activity) and inability to work because of health problems (McHornery et al. 1994; Ware et al. 1995). The association between the health

status scale and the number of self-reported chronic conditions in our own data supports the validity of the scale. The mean health status scores for people with 0, 1, 2, 3, and 4 or more chronic conditions were 87.3, 79.1, 69.8, 59.7, and 46.0, respectively. The health status scale was also strongly associated with all-cause mortality from 1992 to 1998. Crude mortality rates for individuals with health status scores of 0–25, 26–50, 51–75, and 76–100 were 6.3, 2.5, 2.3, and 1.4 percent, respectively.

All independent variables in the health equation were taken from *wave 1*, i.e., as lagged values. These include socioeconomic variables such as age, gender, race, marital status, years of school completed, and the household income-to-needs ratio (measured as the ratio of total household income to the official U.S. poverty line in 1991, adjusted for family size[12]). In addition, we include variables that reflect past health behaviors, such as smoking, number of alcoholic drinks of per day, and measures of initial health stock such as the body mass index (BMI) (weight in kilograms divided by height in meters squared), and the lagged health index. Finally, we use the number of chronic conditions reported in HRS (including hypertension, diabetes, heart disease, chronic lung disease, cancer, arthritis, stroke, and visual difficulties) to develop categories for stratified anaysis by groupings of medical condtions. Not shown in the Table 1 are the ranges of the BMI: quintile 1: 16.7–23.0; quintile 2: 23.0–25.2; quintile 3: 25.3–27.4; quintile 4: 27.4–30.5; quintile 5: 30.6-50.5. Persons are considered "overweight" if their BMI is between 25 and 30, and "obese" if BMI $>$ 30. Thus the third and fourth quintiles correspond to the overweight category, and the fifth quintile corresponds to the obesity category (see Averett and Korenman 1996, for instance).

Between the 1992 and 1996 surveys, there was a change in wording of questions and response options for items in the physical difficulties subscale.[13] Health is expected to decline with time; thus the small increase in mean health index from 76.9 to 77.9 (Table 1) is probably an artifact of a slight change in scaling previously described rather than a reflection of a true increase in health status. However, our initial exploration of the the data showed a similar distribution in both waves, with sample moments (variance, skewness, kurtosis) for both the insured and uninsured are virtually equal for both periods (see Dor, Sudano, and Baker 2003). Thus the 1992 index provides a reasonable baseline measure for the 1996 regressions. Detailed definitions of HRS and instrumental variables, along with summary statistics are presented in Table 1. Summary statistics for binary indicators are the percent in the sample belonging to group. Means and standard deviations are reported for continuous variables. Results from the first stage regressions on insurance

Table 1: Variable Definitions and Descriptive Statistics

Variable	*Description*	*Range and/or Values*	*Mean (SD) or %*
Dependent variable			
Health index 1996	0–100 index of self-reported health status, in 1996. Based on rankings of overall health, number of physical limitations, and pain	0–100	77.90 (22.49)
Insurance status	Insurance status as of 1992, dichotomized as privately insured or uninsured	0 = uninsured	83.00
		1 = private	17.00
Health status equation			
Independent variables (1992)			
Age	Age in years	45–64	55.34 (4.17)
Race/ethnicity	Binary indicators	1 = white/other	77.77
		2 = black	14.34
		3 = Hispanic	7.89
Female	Binary indicator	0 = male	44.53
		1 = female	55.47
Marital status	Binary indicator of marital status in 1992	0 = not married	17.52
		1 = married	82.48
Education	Education in years categorized as follows: less than 9, 9–11, high school graduate or equivalent (GED), some college (more than 12 years)	1 = 0–8	10.57
		2 = 9–11	14.95
		3 = 12 or GED	36.31
	Binary indicators	4 = $\geq$ 12	38.17
Income-to-needs ratio	Ratio of 1992 total household income to 1991 DHHS poverty guidelines, adjusted for family size.	1 = $\leq$ 1.00	5.26
		2 = 1.00–1.49	4.54
		3 = 1.50–1.99	5.72
		4 = 2.00–2.99	13.71
	Binary indicators	5 = 3.00–4.99	27.44
		6 = $\geq$ or = 5.00	43.33

Continued

Table 1: *Continued*

Variable	*Description*	*Range and/or Values*	*Mean (SD) or %*
Smoking status	Self-reported smoking behavior as of 1992 categorized as never smoked, past smoker, and current smoker	1 = never	37.94
		2 = past smoker	36.07
		3 = current	25.99
Alcohol consumption	Self-reported drinking behavior categorized by the number of drinks per day. "Moderate" drinking is defined as $\leq$ 2 drinks; "Heavy" drinking is defined as $\geq$ 3 drinks	1 = abstainer	37.58
		2 = moderate	57.56
		3 = heavy	4.86
Body mass index quintile	Body mass index (BMI) as of 1992 categorized by quintile. This the classic calculation of body weight for height, where BMI = kg/m^2.	Quintile 1	21.15 (1.45)
		Quintile 2	24.18 (0.67)
		Quintile 3	26.37 (0.62)
		Quintile 4	28.90 (0.91)
	Binary indicators, quintile means reported	Quintile 5	34.69 (4.41)
Health index 1992	Control variable for baseline health status in 1992	0–100	76.89 (21.58)
Participation equation			
Additional instruments (1992)			
Unionization	State unionization rates, percent (area resource file)		15.30 (6.86)
Unemployment	State unemployment rate, percent (area resource file)		7.57 (1.42)
State tax	Average state+federal tax rate on wages or earned income, percent (TAXSIM data file, National Bureau of Economic Research).		26.56 (3.03)

participation, and second stage regressions on health status are described below.

Other regressions replicated the analysis by type of major chronic condition available in the Health and Retirement Survey. We designate these as follows: Population 1, persons with no major chronic condition at all; Population 2, persons with any major chronic conditions, i.e., heart disease, stroke, cancer, arthritis, asthma or other chronic lung disease, diabetes, and hypertension; Population 3, the subset of persons with asymptomatic conditions, i.e., conditions which do not have visible symptoms in early stages of the disease, namely hypertension and diabetes; and finally, Population 4, persons with hypertension, the single most common chronic condition in the population. Sample sizes for these grouping were 3,203, 5,831, 3,591, and 3,283, respectively. Further detail on definitions of medical conditions and their groupings are provided in the sixth section.

Results: The Full Sample

First Stage Estimates: Probability of Insurance Participation

Table 2 presents probit estimates on insurance participation, with the dependent variables = 1 if the individual has insurance and = 0 if the individual does *not* have insurance. Two versions of the model are shown. In either case we are not interested in the insurance participation decision per se; rather, we aim to create an instrument that can be incorporated into the second stage estimates of health status. The only explanatory variables included in Model 1 are the state-level variables previously described. Model 2 adds all variables that are also included in the health equation. The two models reflect two levels of identification: Model 1 meets identification criteria, but is more parsimonious; Model 2 adds all regressors from the second stage and is akin to two stage least squares (2SLS) in a fully linearized model. The choice between the two models entails a familiar trade-off between IV and 2SLS: Model 1 might allow a higher correlation of the instruments with treatment variable, while Model 2 should provide a better fit of for the predictive equation. For each model we report the usual goodness of fit statistics, and Wald statistic for the joint significance of the instruments in the insurance participation equations. The results suggest a slight preference in favor of Model 2.

Since results on socioeconomic and demographic characteristics and health behaviors are incidental to our main research question, we present them only briefly. The coefficients indicate the likelihood of purchasing

Table 2: Probit Model for Insurance Participation (First Stage Estimates)

	Model 1		*Model 2*	
Variable	*Coefficient*	*SE*	*Coefficient*	*SE*
Instruments				
Unionization rate	.037	(.001)***	.021	(.003)***
Unemployment rate	.087	(.012)***	−.037	(.064)***
State tax	.004	(.006)	.002	(.006)
Common variables				
Age			.019	(.004)***
Black			−.002	(.050)
Hispanic			−.536	(.067)***
Female			.012	(.046)
Married			.118	(.046)**
Income–needs ratio—1.00–1.49			.332	(.089)***
Income–needs ratio—1.50–1.99			.687	(.085)***
Income–needs ratio—2.00–2.99			1.047	(.075)***
Income–needs ratio—3.00–4.99			1.377	(.072)***
Income–needs ratio—≥ 5.00			.553	(.078)***
Education—9–11 years			.391	(.063)***
Education—high school/GED			.535	(.062)***
Education college			.578	(.062)***
Past smoker			.058	(.043)
Current smoker			−.162	(.048)***
Alcohol abstainer			−.024	(.038)
Alcohol—heavy drinker			−.526	(.083)
Body mass index—2			.058	(.056)
Body mass index—3			.046	(.058)
Body mass index—4			.030	(.057)
Body mass index—5			.031	(.058)
Health index 1992			.002	(.001)***
Constant	.956	(.187)***	−.989	(.351)***
Pseudo R^2	0.033		.216	
Likelihood ratio χ^2	248.64		1,776.88	
Wald test on joint significance of instruments				
χ^2	991.46		888.92	
Degrees of freedom	9		9	
p-value	<0.001		<0.001	
N	9,034		9,034	

Notes: Standard error in parentheses.

* ≥ 90%, <95% significance.

** ≥ 95%, <99% significance.

*** ≥ 99% significance.

insurance increases significantly with age, adjusted household income level (i.e., income-to-needs quintiles) or education level. Black were less likely to have insurance than other races, whereas married persons are more likely to have insurance. Categories of education levels were significantly associated with increased likelihood of having insurance. Of the behavioral variables only current smoking was significantly related to having insurance, in the anticipated negative direction. Hazard rates generated from the probit model are included as independent variables in the health status regressions summarized in Table 4. The estimated coefficients of the hazard are denoted as ρ_{tc}.

Second Stage Estimates: Health Status Equations

Models 1 and 2 in Table 3 represent second stage estimates of "treatment effect" models, corresponding to first stage estimates of Models 1 and 2 from the previous table. With some minor rounding off, the two regressions on health status yield virtually identical results. Thus, the discussion below applies equally to both specifications.

Age had the expected negative effect on the health score, but was probably not significant due to the narrow range of ages in our sample. On the other hand there is a strong gender effect, with females having lower scores, while being married tended to increase the score. The race effect for black may have been mitigated by holding socioeconomic status and health behaviors constant. Smoking, heavy drinking (as opposed to the default category of moderate alcohol consumption), and increasing body-mass categories were all highly significant in the expected direction. As expected, there is a positive association between education and health, with diminishing incremental gains from one level of education to another. This result is consistent with the Grossman investment model, which predicts that an individual's marginal efficiency in deploying medical care and other health inputs increases with the level of education. Increases in relative income operate in a similar way as increases in educational attainment. There is a positive association between income (relative to need) and health. Again the incremental gain of this effect diminishes as income level rises, and becomes negligible at the highest quintile. Finally, the lagged health index, which indexes initial health stock has a significantly positive effect. The simple interpretation of this variable is that better health in the past leads to better health in the future. A further interpretation of this variable, based on rearranging the model to form a change equation, also suggests that it reflects the rate of depreciation of health stock.[14]

Table 3: Treatment Effects Model: Health Status Equations (Second Stage Estimates)

	Model 1		*Model 2*	
Variable	*Coefficient*	*SE*	*Coefficient*	*SE*
Age	−.046	(.042)	−.076	(.044)
Black	−.390	(.504)	−.373	(.510)
Hispanic	.603	(.691)	1.49	(.719)*
Female	−1.514	(.377)***	−1.502	(.381)***
Married	.918	(.468)**	.702	(.470)
Education—9–11 years	1.311	(.710)*	.411	(.737)
Education—high school/GED	3.379	(.658)***	2.244	(.850)***
Education—college	4.623	(.682)***	3.427	(.871)***
Income–needs ratio—1.00–1.49	.989	(1.074)	.115	(1.158)
Income–needs ratio—1.50–1.99	9.047	(1.027)	.701	(1.010)
Income–needs ratio—2.00–2.99	.887	(1.026)	.840	(1.054)
Income–needs ratio—3.00–4.99	2.025	(.860)**	1.571	(1.092)
Income–needs ratio—>or = 5.00	2.336	(.862)***	1.801	(1.121)
Past smoker	−.954	(.403)**	−1.033	(.410)**
Current smoker	−3.293	(.447)***	−1.034	(.450)***
Alcohol abstainer	−.389	(.372)	−.408	(.376)
Alcohol—heavy drinker	−1.560	(.807)*	−1.484	(.819)*
Body mass index—2	−1.227	(.538)**	−1.290	(.546)**
Body mass index—3	−2.339	(.550)***	−2.296	(.557)***
Body mass index—4	−2.339	(.550)***	−2.367	(.550)***
Body mass index—5	−5.142	(.566)***	−5.170	(.574)***
Health index 1992	0.656	(.008)***	.652	(.008)***
Insurance status	7.629	(2.678)***	8.828	(1.550)***
Constant	23.662	(3.004)***	27.222	(2.805)***
ρ	−0.220		−0.257	
σ_{ε}	16.104		16.126	
$\sigma_{u\varepsilon}$	−3.537		−4.138	
Wald (χ^2)	8,434.88		9,971.60	
N	9,034		9,034	

Notes: Standard error in parentheses.

* $\geq$ 90%, <95% significance.

** $\geq$ 95%, <99% significance.

*** $\geq$ 99% significance.

Since our main research question deals with the consequences of the lack of insurance, all of the above variables effects, while interesting, are treated here merely as controls. Nevertheless, it is noteworthy that their impact of health measures are as expected based on common knowledge and related literature on determinants of health, further indicating the validity of the health index as a measure of overall healthiness. This increases our confidence

Table 4: Instrumental Variable (IV) versus Treatment Model: Summary of Main Coefficients

		IV Estimator		*Treatment Model*	
Variable	*OLS*	*Model 1*	*Model 2*	*Model 1*	*Model 2*
Lagged health	0.656 (77.14)	0.656 (77.14)	0.655 (76.32)	0.656 (77.32)	0.652 (77.06)
Insurance participation	1.385 (2.73)	7.714 (2.30)	8.867 (1.97)	7.629 (2.85)	8.828 (2.80)
Residual	—	− 6.429 (− 2.30)	− 6.81 (− 1.69)	—	—
λ	—	—	—	− 3.537 (− 2.38)	− 4.138 (− 2.39)
Adjusted R^2	0.492	0.494	0.493	—	—
Wald χ^2	—	—	—	8,434.9(d.f. = 23)	9,971.6(d.f. = 45)

Notes: *t*-values in parentheses; the coefficient of λ is given by σ_{te}.

in the main finding, namely that lack of insurance has a significant negative impact on health. Because a number of measurement issues arose, we defer to the summary of related results as presented in Tables 4 and 5.

Endogeneity of the Insurance Indicator

The Wald tests for parameter restrictions that we report in Table 2 indicate that our instruments are jointly significant. As Goldman et al. (2001) indicate, the hypothesis that an instrumental variable is uncorrelated with unobservable characteristics, and hence with the outcome measure itself, cannot be tested directly. Instead they propose a "weak test"—by regressing the full set of variables *including* the proposed instrumental variables on outcomes in second stage (Goldman et al. 2001). We therefore regressed the health index on the full set of variables, including the three instruments from the first stage. We ran these regressions separately for the subsample of the uninsured, the subsample of the insured, as well as for the pooled sample. In all of these runs, none of the instrumental variables were significant at the 5 percent significance level. While we cannot assert with certainty that these variables are uncorrelated with unobservable health status, the results from these additional regressions are strongly suggestive of this, at least for the particular population in our data. Insurance effects of the OLS estimates (Table 4) provide a lower bound.

IV Model and Treatment Model Comparisons

Table 4 provides a comparison of the three general types of econometric models: ordinary least squares, the IV estimator, and the treatment model. The treatment model was previously described, but the main coefficients are included here for expositional convenience. The specification of the IV model is analogous to the treatment model. Thus the same set of exogenous variable is included in both types of model, and insurance nonparticipation is instrumented two ways, i.e., using the strict identification criterion (Model 1), with insurance offer variables only, and then using a fuller specification with the full vector of health status covariates (Model 2). Only coefficients of the test variables and test scores, along with their levels of significance are included in Table 4.[15] The most important set of results pertains to the insurance participation variable. The OLS model serves as a baseline case. As was suspected, OLS underestimates the true effect of having insurance on health. The IV and treatment models yield much larger estimates, but are similar to each other (respectively, 4.06 and 5.05 in Model 1; 4.07 and 4.70 in Model 2).

Table 5: OLS versus Treatment Models for Specific Chronic Disease Subpopulations

	OLS				*Treatment Model*			
Variable	*Population 1, No Chronic*	*Population 2, All Chronic*	*Population 3, Asymp.*	*Population 4, Hypertension*	*Population 1, No Chronic*	*Population 2, All Chronic*	*Population 3, Asymp.*	*Population 4, Hypertension*
Lagged health	0.523	0.645	0.655	0.659	0.523	0.640	0.650	0.653
	(29.99)	(60.05)	(47.73)	(45.37)	(29.57)	(55.75)	(45.82)	(43.37)
Insurance participation	2.410	1.060	1.265	0.675	9.228	6.570	6.682	8.494
	(3.44)	(1.55)	(1.45)	(0.73)	(2.10)	(1.64)	(1.44)	(1.76)
λ	—	—	—	—	−3.795	−3.066	−3.044	−4.397
					(−1.57)	(−1.37)	(−1.19)	(−1.65)
Adjusted R^2	0.283	0.484	0.500	0.498	—	—	—	—
Wald χ^2	—	—	—	—	1,677.9	6,418.1	4,233.3	3,829.2
					(d.f. = 45)	(d.f. = 45)	(d.f. = 45)	(d.f. = 45)
N	3,203	5,831	3,591	3,283	3,203	5,831	3,591	3,283

Notes: Population 1, persons with no chronic conditions; Population 2, persons with any of the following chronic diseases: hypertension, diabetes, chronic obstructive pulmonary disease (COPD) or asthma, heart disease, stroke, cancer, arthritis, visual difficulty; Population 3, persons with asymptomatic diseases: hypertension and diabetes; Population 4, persons with hypertension.

The coefficients of the first stage residuals in the IV model and the coefficients of λ in the treatment models are negative, implying that OLS underestimates the effect of insurance. Since these coefficients are statistically significant in the first IV model and in both treatment models we reject the null hypotheses that the error terms in the two stages of the estimation are uncorrelated. However, estimates from IV estimators are always less efficient (Vella and Verbeek 1999). Therefore we refer to the treatment model henceforth. Note that we made no a priori conjectures about the direction of the bias. The results suggest that the former is the dominant explanation. An alternative explanation is related to an unobservable trait, say a propensity to exhibit behaviors consistent with moral hazard. This would lead people to choose insurance while reducing preventive effort, with adverse effects on health. Converting the insurance coefficient to percent terms yields a wide range of estimates for the effect of insurance on the health score, equivalent to 1.8 percent in OLS to 9.8–11 percent in the treatment effects models. A simple calculation showed that the combined effect of all health behaviors observed in our data (Model 1), e.g., converting from "current" smoking to past smoking, from heavy drinking to moderate drinking (the default state), and from the obese state to the overweight state yields a combined improvement in the health score of about 8.7 percent. While the definitions of these variables are somewhat ambiguous, it is interesting to note that the estimate of the insurance effect adjusted for endogeneity is roughly in the same order of magnitude as the combined behavioral effects.

Results: Chronic Conditions

It has been suggested that the effect of insurance may be particularly pronounced for specific chronic conditions such as HIV/AIDS (Goldman et al. 2001) and cancer (Ayanian et al. 1993) for which life-prolonging therapies are available to the uninsured only at prohibitively high costs. Others have emphasized the importance of insurance in accessing diagnostic and preventive services prior to the emergence of chronic illness or complications (Bednarek and Schone 2003; Sudano and Baker 2003; Nelson et al. 2000) suggesting that the impact of insurance may be larger in the presence of marker conditions such as diabetes and hypertension where early diagnosis and monitoring is critical in maintaining health (Nelson et al. 2000). Following, Baker, Shapiro, and Schur (2000) we characterize these particular conditions as asymptomatic. Focusing on a clinically heterogeneous population may mask important

differences between key subgroups, possibly leading to inappropriate policy conclusions regarding the need to extend coverage. While our main interest is in the overall effect, thereby allowing us to make comparisons between insurance effects in the health score and insurance effects found in the prior literature on all-cause mortality, we also implemented an exploratory analysis for major groupings of chronic conditions.

To allow for differential effects we replicated the previous analysis for the following groupings: no chronic condition, all major chronic conditions (mainly cancer, stroke, heart disease asthma, other upper respiratory illnesses), a subset of marker conditions, referred to as "*asymptomatic*"; see Baker, Shapiro, and Schur 2000), i.e., diabetes or hypertension, and finally, hypertension only.[16] We did not perform the analysis for other specific medical conditions due to small sample sizes available in the HRS. Main results are summarized in Table 5.[17] As is the case with the pooled analysis the coefficient of insurance participation is consistently higher in the treatment model compared with OLS. Moreover, the insurance coefficients change from being nonsignificant in the OLS models (populations 2–4), to being significant at the 10 percent level in the treatment models (with the exception of population 3). Converting the coefficient of insurance in the treatment model into percents indicates that the effect of insurance is about the same across all of the groupings (10.6, 8.9, 9.1, and 11.5 percent, respectively. Note the health score for the "no chronic disease" group, was highest at 87.5; it was approximately 73 for each of the other groupings). These results suggest that there are no substantial differentials between chronic, nonchronic conditions, and asymptomatic conditions. Rather, the finding of a large insurance effect on health applies about equally across the board, regardless of the underlying medical condition. However, because the coefficient of lambda (σ_{ts}) is nonsignificant in these models, we *cannot* reject the null hypotheses that the error terms in the two stages of the estimation are uncorrelated. By extension we cannot reject OLS unambiguously. The low standard error on the coefficient of the hazard may be due to smaller sample size, suggesting the need for analysis using sample sizes that are larger than currently available in the HRS.

Implications and Future Research

The finding of substantial insurance effects on mortality has been well documented in the previous literature. This study complements the literature by focusing on a health score measure akin to the physical component subscale of

the SF-36, as the measure of interest. Our study further extends the literature by implementing an adjustment for endogeneity bias and suggesting a range of adjusted and unadjusted estimates for the insurance effect. While results using different outcome measures are not strictly comparable, there appears to be a general agreement that insurance effects are positive, may be substantial, and persist under a variety of settings. We also present preliminary results, which suggest that the positive effects of insurance apply across the board, that is, for all major groupings based on underlying medical conditions.

The rising number of the uninsured has lead to many proposals to expand options for insurance coverage, but to date only incremental steps have been implemented. Our results provide additional support for the notion that health insurance, and in particular private insurance have beneficial effects, thereby providing impetus for undertaking further steps to expand coverage. Our focus on older, working age adults, ages 45–64, most of whom obtain coverage through employer-sponsored and other private insurance underscores the need for extending *private* insurance coverage in particular. Focusing on this age group bears other important implications, since it is the cohort approaching Medicare eligibility at age 65. Specifically, we are concerned with potential spillover effects, whereby preventable illness and morbidity translates into higher future costs for Medicare. To the extent that declines in health can be mitigated with the presence of insurance, potential savings can be accrued to Medicare due to expansion of private coverage which will offset some of the cost of providing subsidies to the private sector.

A related policy issue that has been debated is whether policies aimed at extending insurance coverage or other safety net measures should be targeted to particular groupings in the population that are most vulnerable to chronic disease and to those actually diagnosed with certain marker conditions such as diabetes or hypertension (IOM 2000). In 2002, however, the Institute of Medicine's (IOM) Committee on the Consequences of Uninsurance (IOM 2002, p. 103) reached the conclusion that "broad-based health insurance strategies across the entire uninsured population would be more likely to produce the benefits of enhanced health and life expectancy than would 'rescue' programs aimed only at the seriously ill." Our preliminary results for subgroup analysis tend to support this conclusion.

Another important area for investigation relates to the tradeoff between insurance and programs designed to induce behavioral changes to improve health. Our regression results yielded an insurance effect, adjusted for endogeneity, which is somewhat larger than the combined effect of behavioral changes observed in the HRS. At least two caveats apply. First, due to

limitations inherent in survey data, we can provide general orders of magnitude only. With the exception of obesity, which can be measured fairly accurately thanks to the BMI, the definitions of the other behavioral factors identified (smoking, drinking) are somewhat ambiguous in the HRS; moreover behavioral change can take a long time to work through, having a cumulative impact on future health; thus survey data may understate their value. Second, provision of insurance probably implies early detection, diagnosis, and treatment of disease through a variety of means, and therefore should not be viewed as an alternative to public health intervention designed to improve health behaviors. While the role of behavioral factors falls outside the scope of our research, there is a need to examine this issue more deeply in future research.

Despite the support implied in our results for policies that extend insurance coverage, important limitations should be noted. First, we use a relatively short time series of four years. Future research should consider longer study periods of a decade or more found in the seminal mortality studies (e.g., Franks, Clancy, and Gold 1993). This can be done by examining future waves of the Health and Retirement Survey. Second, there is a need to probe the stratified analysis of chronic condition groupings more deeply, given the relatively small sample sizes available for this purpose. The Health and Retirement Study remains the most detailed household survey for assessing physical health score measures, with the added feature of repeated interviewing of the same subjects at regular intervals. The Medical Expenditures Panel Survey (MEPS) presents an alternative with substantially larger sample sizes, but for now it provides a shorter panel of two to three years. Until this data set is extended with repeated interviews, research on condition-specific outcomes will continue to involve the tradeoff between analyses of longer time-series with relatively limited explanatory power versus analyses using larger sample sizes but with tracking periods which may be too short for observing meaningful changes. It should be noted that longer time series are expected to yield larger cumulative effects of insurance on health.

The choice of instrumental variables used to identify the insurance participation equation is another area that merits further exploration. In this study, our use of state-level variables led to dramatically higher estimates of the insurance effect on health outcomes compared with estimates in unadjusted models. We found similar results when we experimented with firm-level instruments (Dor, Sudano, and Baker 2003). Since no definitive test for instrument validity currently exists, studies with alternative instruments may lead to greater confidence in our findings if similar results are found. Variables

that better describe state and local government policies towards private insurance offerings are of special interest.

Ultimately policy choices for expanding coverage would depend on programmatic costs in addition to the value of the benefits noted. While preliminary estimates of the cost of the uninsured are now available from the Institute of Medicine Study (Miller, Vigdor, and Manning 2004) less is known about the direct and indirect costs of the various options proposed. Unfortunately, the Health and Retirement Study does not contain information on the type of plan or the generosity of benefits available to respondents. This issue should be examined in future research.

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NOTES

1. Two possible omitted variables are need and ability. A person's innate ability may increase her likelihood of obtaining health insurance by searching, accessing information, etc., while simultaneously making her better equipped to act independently to maintain health. If ability matters the insurance effect in the unadjusted OLS is actually biased upwards. Conversely, a person's *perceived* need for health improvement is likely to be correlated negatively with health, since people in poorer health are more likely to actively pursue insurance options (in our sample of working age adults, mostly through employer offers). Thus if need matters the insurance effect in the health equation would be downward bias. However, given the mostly employer-based provision of private health insurance in the U.S., both ability and need to obtain insurance must be related to characteristics of labor market (e.g. unionization and employment rates) as well as individual incentive (e.g., implicit subsidy for health insurance in the tax code).
2. Lags considered in related seminal medical studies on the effects of smoking, physical activity, and alcohol consumption drinking range from one year to a decade (e.g., Frank et al. 1966, Lacroix et al. 1991, and Thun et al. 1997).

3. To this, we first write the health equation in the following form:

$$H_t = b_0 + b_1 Y_{t-k} + (1 - \delta)H_{t-k},$$

where Y is a vector of insurance and other variables related to health behaviors and δ is the rate of depreciation of health stock. Rewriting we get:

$$H_t - H_{t-k} = b_0 + b_1 Y_{t-k} - \delta H_{t-k}, \text{ where } \delta \leq 0.$$

The coefficients can be alternatively interpreted as being generated by distributed lag, adaptive expectations, or partial adjustment processes (Maddala 1983), but these are difficult to distinguish and not relevant to our discussion.

4. Though related, the treatment effects model differs from the standard Heckman selection model in several important respects: the selection model assumes a conditional sample (e.g., those having insurance only) whereas the treatment model applies to problems where all observations are pooled; the selection model uses a selection term (hazard rate) generated for the particular subsample (e.g., insured sample). The treatment model generates different hazard rates for two "groups"—those treated and those untreated (e.g., insured and uninsured)—but assigns the appropriate value for the hazard rate to each individual in the pooled sample (see *Stata* 7 manual, Su-Z, p. 207); the selection model usually applies to situations with *censoring*, e.g., wage rates are observed only for those participating in the labor force. The treatment effect applies where there is no censoring, but only some of the observations receive an "intervention," e.g., some workers receive training while others do not, but wages are observed for all; the selection model includes only the hazard (acting as a control for the probability of being in the sample). The treatment model, having a pooled sample, includes a dummy variable for the intervention (receiving training, having insurance, etc.) *in addition to* the hazard.
5. The advantage of the treatment model is that it yields a regression coefficient for the treatment dummy itself which is directly comparable to the coefficient of the treatment dummy from OLS. Note that this procedure provides us with the population effect (also referred to as "average treatment effect" by Imbens and Angrist 1994), which is further comparable to the IV estimation used in Table 3. A detailed explanation is given in Vella and Verbeek (1999).
6. This is available in the treatreg option in *Stata*, version 7.
7. Stated differently, a high correlation between regression errors indicates that endogeneity is present. The covariance term can be expressed in terms of the correlation: $\sigma_{t\hat{a}} = \rho\sigma_{\varepsilon}$. All relevant terms are reported in the regression tables.
8. The use of residuals to account for endogeneity is commonly encountered in models with censored endogenous regressors. For example, Heckman (1978, 1979) adopted this approach to account for sample-selection bias and endogeneity bias in models in which the treatment is captured through an indicator function. Vella (1993) employed the same approach for a range of models involving selection bias or censored endogenous regressors.
9. The public use files of our main data source, the Health and Retirement Study (HRS), do not provide any geographic information, including state of residence in the U.S., due to strict confidentiality requirements. We therefore submitted an application with the HRS staff and review committee for use of an HRS "restricted

data set" containing state-level identifiers. State identifiers were then merged with other state variables for use in our instrumental variable analysis.

10. Individuals who said they had only minimal coverage, e.g., coverage for special conditions such as mental health, dental insurance, or long-term care insurance, were classified as uninsured because these policies do not enable access to routine health care services. In the full sample, there were only 360 such cases.
11. The component items of the index are as follows: *Self-reported overall health*, with the options excellent, very good, good, fair, or poor (coded 5 = excellent to 1 = poor). The physical *mobility* component which measures ability to perform activities requiring large muscle strength using 4 items: walking several blocks; walking one block; climbing several flights of stairs; climbing one flight of stairs without resting. The *agility* component measures physical activities required to perform instrumental activities of daily living using 6 items: sitting for about 2 hours; getting up from a chair after sitting for long periods; lifting weights over 10 pounds; stooping, kneeling, or crouching; pulling/pushing a large object; and reaching/extending arms above shoulder level. Items were coded 1 if the respondent reported no difficulty with the activity, 0 otherwise, then summed for each component. The *pain level* measure is taken from several items in the questionnaire that ask respondents to characterize their pain as none, very mild, mild, moderate, and severe (coded from 5 to 1, respectively). Finally, all four measures were summed and scaled to form the 100-point *health index*. A test of the correlation of rankings across the various items indicated a very high degree of internal consistency (Cronbach's $\alpha = 0.82$).
12. The income-to-needs ratio has been found to perform well at predicting overall household resource availability. See Mayer and Jencks (1989) and Danziger and Gottschalk (1986).
13. In 1992, the options for the physical difficulties in the HRS were "not at all difficult," "a little difficult," "somewhat difficult," and "very difficult/cannot do." In 1996, the question was rephrased to "Do you have any difficulty?" with the responses: no, yes, cannot do. To have consistency between the questions and response options in 1992 and 1996, we used a similar approach to that used in previous studies (Baker, Stabile, and Deri 2001; Fillenbaum et al. 1993; Clark, Stump, and Wolinsky 1998). All questions for 1992 and 1996 were dichotomized into no difficulty (= 0) versus some difficulty (= 1). The absence of the option "a little difficult" in 1996 may have prompted certain respondents to report "no difficulty," for instance.
14. More explicitly, the complement of the lagged health coefficient, $1 - 0.65 = 0.35$ is the rate of depreciation, yielding a cumulative average annual rate of 0.6 percent over the observed four-year interval. Converted to elasticity terms, a 8 percent increase in past health contributes to approximately 5 percent increase in health in the later period, all things being equal.
15. In other specifications we also included individual employment or job type indicator variables for union status and job type status (e.g., professional managerial versus worker) as explanatory variables (see Dor, Sudano, and Baker 2003). Their coefficients were not significant and did not affect other results. However, we opted not to include these results due to potential endogeneity of work and effort in the health equation. This issue should be explored in future research.

16. Respiratory illnesses include chronic obstructive pulmonary disease (COPD) or emphysema. Cancer may be diagnosed prior to appearance of symptoms, but is classified is a symptomatic disease because symptoms would typically appear a short time later. Another common asymptomatic condition, high cholesterol count, was not available from the HRS survey.
17. To conserve space we did not report the full set of regressions, which are available from the authors upon request.

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