

# Health Shocks and the Evolution of Earnings over the Life-Cycle\*

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## Abstract

We study the contribution of health shocks to earnings inequality and uncertainty in labor market outcomes. We calibrate a life-cycle model with idiosyncratic health, earnings, employment and survival risk, where individuals make labor supply and savings decisions, adding two novel features. First, we model health as a complex multi-dimensional concept. We differentiate between functional health and latent health risk, and between temporary/persistent and predictable/unpredictable health shocks. Second, we model interactions between health and human capital accumulation. We find that, in an environment with both costly health shocks and means-tested transfers, low-skill workers find it optimal to reduce their labor supply in order to maintain eligibility for transfers that protect them from potentially high health care costs. Thus, means-tested transfers generate a moral hazard effect that causes agents (especially those with low productivity) to invest less in human capital. Provision of public insurance can alleviate this problem and enhance labor supply.

*Keywords:* Health, Health Shocks, Human Capital, Income Risk, Precautionary Saving, Earnings Inequality, Health Insurance, Welfare

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# 1 Introduction

In this paper we study the contribution of health shocks to earnings inequality and uncertainty in labor market outcomes. Our work can be viewed as a structural extension of [Smith \(2004\)](#), who studied effects of major health shocks on employment and earnings in the Health and Retirement Study (HRS), finding large negative effects over horizons of two to ten years. We embed health shocks in a life-cycle labor supply/consumption framework that enables us to investigate both their direct effects and indirect effects operating through behavioral responses. We extend prior work by incorporating both health shocks and endogenous human capital formation in one model, and we find they interact in important ways over the life-cycle.

A key contribution of our work is the detailed data on health shocks that we construct using the Medical Expenditure Panel Survey (MEPS). In our framework people are subject to health shocks that may be temporary or persistent, and predictable or unpredictable. We categorize all health shocks in MEPS by these criteria. This allows us to capture how health shocks contribute to transitory vs. persistent and anticipated vs. unanticipated variation in wages and employment. Importantly, our model allows us to distinguish between exogenous earnings risk generated directly by health shocks, and endogenous labor supply and human capital responses to health shocks. We find these behavioral responses considerably amplify the contribution of health shocks to inequality.

In our model health shocks are specific events, whose probability of occurrence is influenced by one's health state. We model health as a multi-dimensional concept. We distinguish between "functional health" ( $H$ ) and asymptomatic "risk factors" ( $R$ ). The state variable  $H$  includes aspects of health that directly affect labor productivity. The state variable  $R$  captures underlying risk factors that have no immediate effect on productivity, but that affect the evolution of functional health ( $H$ ) and the probabilities of adverse health shocks in the future. The most important risk factors in  $R$  are hypertension and high cholesterol.

Our stochastic process for the arrival of health shocks incorporates the fact that some are predictable based on  $H$  and  $R$ , while others are idiosyncratic. The nature of health shocks - i.e., their predictability and persistence - has an important impact on the nature of fluctuations in both productivity and medical expenses. The extent to which health-induced shocks to productivity and medical costs are mainly transitory/predictable vs. persistent/unpredictable is important to how well individuals can self-insure against these risks and for the value of health insurance. The degree to which health shocks can be anticipated is important for precautionary savings and the degree of pass-through to consumption.

The second major contribution is the study of human capital accumulation in the presence of health risk.<sup>1</sup> Individuals accumulate human capital via learning-by-doing, as in [Keane and Wolpin \(1997\)](#) and [Imai and Keane \(2004\)](#). But in our model health affects human capital accumulation in several ways.<sup>2</sup> For instance, returns to current investments in human capital

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<sup>1</sup>These two features have not been combined in a life-cycle framework, with the notable exceptions of [Hokayem and Ziliak \(2014\)](#) and [Hai and Heckman \(2015\)](#). But the way we model health risk is very different.

<sup>2</sup>We define "human capital," as comprising skill generated by education and work experience, which we distinguish from the stock of health. Both impact on worker productivity in our model. The distinction between health and human capital was first made by [Grossman \(1972\)](#). While our modeling is different, we also stress the ways in which human capital and health interact.

depend on an agent's future ability to work, which can be diminished by adverse health shocks. Thus, if one anticipates poor health and reduced work in the future, the incentive to work today and invest in human capital is lower. Our framework allows us to predict the dynamic effect of a health shock on future earnings, incorporating the evolution of both health and human capital after the shock.

Aside from our approach to building health and health shocks into the life-cycle model, three other features of our framework are notable. First, we model job offers that may or may not include employer sponsored health insurance (ESHI), where probabilities depend on education. This is a key aspect of the US environment and an important aspect of risk, as the vast majority of insurance for those under 65 is employer linked.

Second, following [De Nardi et al. \(2010\)](#), [French and Jones \(2011\)](#) and [Capatina \(2015\)](#), we take the view that if a health shock occurs the realized cost of treatment (net of insurance) *must* be borne by the agent.<sup>3</sup> Thus, medical expenditures are not a *choice*, but rather an exogenous realization from an expenditure distribution. This distribution specifies the level of medical expenditures associated with each possible combination of functional health and health shocks. Within each health state, we also allow for the possibility of "normal" vs. "catastrophic" expenditures based on the observed distribution of costs.

Third, we model the US tax and social insurance system in some detail. In particular, if a person has sufficiently low financial resources he may qualify for a transfer that guarantees a minimum level of consumption, calibrated to approximate social insurance benefits. We capture disability insurance (DI) benefits by allowing for a positive probability that the transfer amount is increased for those in poor health (for discussions of the probabilistic nature of DI eligibility see [Benítez-Silva et al. \(1999\)](#) or [Low and Pistaferri \(2015\)](#)).

A brief overview of our model is as follows. Individuals begin every period with stocks of functional health ( $H$ ), risk factors ( $R$ ), assets and human capital. Working age individuals receive employment offers (part or full-time) which they accept or reject. A fraction of offers include employer provided health insurance. Wage offers depend on human capital and functional health, and are subject to transitory and persistent shocks. The disutility of working also depends on health. After the employment decision is made, health shocks occur with probabilities that depend on  $H$  and  $R$ . These health shocks, along with functional health and age, determine medical expenditures and sick days suffered by workers. Finally, individuals make consumption/savings decisions. At the start of the next period, new stocks of health and human capital are revealed (based on their laws of motion).

To summarize, the direct pathways through which health and health shocks affect labor market outcomes include effects on wages, sick days, and the disutility of work. The indirect pathways include effects on incentives for human capital accumulation, demand for employer health insurance and reliance on means tested social insurance programs.

We calibrate our model to the U.S. male population using the Medical Expenditure Panel Survey (MEPS).<sup>4</sup> The MEPS contains detailed information on respondents' medical conditions, coded according to the International Classification of Diseases (ICD). Based on expert medical advice, we categorized medical conditions according to (i) whether they affect productivity, (ii) whether they are risk factors for other health problems, (iii) predictability

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<sup>3</sup>Our view is that patients have little ability to know the cost of their treatment *ex-ante*, or to make informed choices whether to bear that cost. Hence, they pay for whatever treatment is prescribed.

<sup>4</sup>We also use the CEX, CPS and PSID to estimate various moments that are used in the calibration.

and (iv) persistence.<sup>5</sup> The MEPS also contains detailed measures of total and out-of-pocket medical expenditures. Using this information, we estimate stochastic processes for health, health shocks, and medical costs. These are critical inputs to our model.

Our main results are as follows: To begin, we use our model to estimate the fraction of variance (across people) in the present value of lifetime earnings (PVE) that can be explained by initial conditions vs. other factors. Following [Keane and Wolpin \(1997\)](#), we run regressions of the PVE (for simulated individuals) on initial conditions. Similar to their results, we find that 86.8% of the variance in the PVE can be explained by initial conditions at age 25, primarily education and a fixed productivity type. There is only a small contribution of initial health, which varies little across people at age 25. When we add measures of realized health shocks to this regression, the  $R^2$  increases to 92.4%, implying an incremental contribution of health shocks of 5.6% to the variance of the PVE.

Next, we simulate lifetime earnings in a counterfactual world without health shocks. In this world the variance of the PVE is reduced by 11.2% relative to the baseline. This figure is twice as great as the contribution of health shocks implied by the [Keane and Wolpin \(1997\)](#)-style regression analysis. The reason for the difference is that the regression analysis assesses the “direct” impact of differential incidence of health shocks in a world with fixed decision rules for labor supply and consumption.<sup>6</sup> In contrast, if we simulate elimination of health shocks, the decision rules for labor supply and consumption change in important ways, generating a “behavioral” response. Much of our analysis is devoted to understanding the direct and behavioral channels through which health shocks affect earnings inequality:

First, consider how health shocks contribute to earnings inequality in a fixed environment. Differential exposure to health shocks over the life-cycle generates increasing inequality with age in functional health, which directly affects productivity. Lost work time due to health shocks also generates increasing inequality with age in accumulated work experience and human capital. These are the two main “direct” channels.

Now consider the behavioral responses: Health shocks have significant negative effects on labor supply for low-skill workers. Our model implies that uninsured medical expenses push a large fraction of low-skill workers onto means tested social insurance (SI) programs. Low skill workers have an incentive to reduce their earnings to maintain eligibility for social insurance. This, in turn, reduces human capital accumulation, amplifying the negative effect of health shocks on productivity.<sup>7</sup> Conversely, health shocks have positive effects on labor supply and savings of high-skill workers who wish to save more in order to self-insure against expenses from health shocks. Via these two behavioral mechanisms, the elimination of health shocks causes low-skill workers to work and earn more, and high-skill workers to work and earn less, thus reducing lifetime earnings inequality.

Finally, we analyze the impact of providing mandatory public health insurance to people who lack employer provided insurance. Publicly provided insurance against medical costs

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<sup>5</sup>We thank Dr Philip Haywood for his assistance in classifying health shocks based on the ICD codes.

<sup>6</sup>In other words, the regression analysis assesses the role of heterogeneity across agents in the incidence of health shocks on earnings inequality, holding the environment - including the risk of health shocks - fixed.

<sup>7</sup>Low-skill workers have lower labor supply from early on in the life-cycle due to low wages and the option to use means tested insurance. Non-employment and low incomes lead to poorer functional health and a higher probability of health shocks as they age. This creates a vicious cycle, as worse health feeds back to further lower wage offers and labor supply, amplifying earnings inequality.

eliminates the perverse incentive for low skill workers to reduce their labor supply to maintain eligibility for means-tested social insurance. It has the opposite effect on high skill workers, as it reduces their incentive to work and save for self-insurance purposes. Our simulations imply that positive work effect for low skill workers is much larger. Public health insurance raises additional tax revenue and saves on means-tested social insurance costs through this positive labor supply mechanism, counteracting a large share of the cost of provision. To our knowledge this benefit of public health insurance has not been noted previously.

The outline of the paper is as follows. Section 2 reviews the literature and Section 3 presents our model. Section 4 describes our MEPS data. Section 5 describes the calibration, and section 6 discusses model fit. Section 7 presents results and Section 8 concludes.

## 2 Relation to Literature

Our paper contributes to the large literature on earnings inequality by assessing the importance of health risk as a contributing factor. The large literature on earnings dynamics (e.g., [MaCurdy \(1982\)](#), [Gottschalk and Moffitt \(1994\)](#), [Geweke and Keane \(2000\)](#), [Moffitt and Gottschalk \(2002\)](#), [Meghir and Pistaferri \(2004\)](#), [Meghir and Pistaferri \(2011\)](#), [Guvonen \(2009\)](#), [Blundell et al. \(2013\)](#)) does not attempt to disentangle the sources of shocks to the earnings process. Attempts to open the “black box” of earnings shocks include [Abowd and Card \(1989\)](#), who considered joint fluctuations in hours and earnings, and later [Low et al. \(2010\)](#) and [Altonji et al. \(2013\)](#), who extended the income process to allow for endogenous fluctuations in employment, hours and wages.

We also contribute to the rapidly growing literature on life-cycle models with health uncertainty (e.g., [Palumbo \(1999\)](#), [French \(2005\)](#), [Jeske and Kitao \(2009\)](#), [Khwaja \(2010\)](#), [Attanasio et al. \(2010\)](#), [De Nardi et al. \(2010\)](#), [French and Jones \(2011\)](#), [Kitao \(2014\)](#), [Capatina \(2015\)](#), [Pashchenko and Porapakarm \(2016\)](#), [Jung and Tran \(2016\)](#), [De Nardi et al. \(2017\)](#), [Cole et al. \(2018\)](#), and [Hosseini et al. \(2018\)](#)). We extend this work by using a richer model of the health process, and by incorporating endogenous human capital.

Our work is closely related to the reduced form literature on effects of health shocks on employment and earnings. Much of that work defines health shocks as changes in the stock of self-reported or objective health ([Au et al. \(2005\)](#), [García Gómez and López Nicolás \(2006\)](#), [Lenhart \(2019\)](#)). These papers find declining health reduces earnings and employment.

Because the stock of health and employment/earnings are jointly determined over the life-cycle, [Smith \(1999, 2004\)](#) argues the best way to identify the effect of health on labor market outcomes is to control for baseline health and human capital and estimate effects of the onset of specific health shocks. Adopting this approach, he finds that onset of cancer, heart and lung disease have substantial negative effects on employment and earnings. For example, in the HRS he estimates a cumulative income loss of \$37k over ten years (1994-2003) following a major health shock. Using a similar approach, [Pelkowski and Berger \(2004\)](#) find that onset of permanent health conditions reduces wages and hours.

Our work can be viewed as a structural extension of this type of analysis, where we build health shocks into a life-cycle labor supply model. As we emphasized in the introduction, our model distinguishes several mechanisms through which health shocks affect labor market outcomes. As we also discussed in the introduction, we classify health shocks as persistent vs.

transitory and predictable vs. unpredictable, as these types of shocks should have different impacts on earnings, labor supply and consumption. To our knowledge, the only prior work that estimates effects of persistent vs. transitory health shocks on employment and earnings is [Blundell et al. \(2016\)](#), who find much larger effects of persistent shocks.

We also contribute to the literature on life-cycle models of human capital accumulation (e.g., [Shaw \(1989\)](#), [Eckstein and Wolpin \(1989\)](#), [Keane and Wolpin \(1997, 2001\)](#), [Imai and Keane \(2004\)](#)) by incorporating health and health shocks into such models. The interaction between health and human capital has received little attention in that literature until relatively recently. [Hai and Heckman \(2015\)](#) and [Hokayem and Ziliak \(2014\)](#) estimate structural models where health and human capital formation interact over the life-cycle. Those papers view medical spending as an investment, while we treat it primarily as a cost of treatment shock induced by health shocks.

Our paper is also closely related to the literature studying how public insurance programs interact with health risk in determining labor supply and earnings. [French \(2005\)](#) studies the pattern of job exits at old ages and the roles of Social Security benefit rules. [French and Jones \(2011\)](#) study the roles of employer-based health insurance, Medicare and Social Security on labor supply and retirement behavior. [Benitez-Silva et al. \(2010\)](#), [Low and Pistaferri \(2015\)](#), and [Kitao \(2014\)](#) study the impact of Disability Insurance policies on employment decisions. [Pashchenko and Porapakarm \(2016\)](#) show that means-tested Medicaid significantly distorts labor supply decisions, leading to a large fraction of Medicaid enrollees not working in order to qualify. We contribute to this literature by showing that insuring health risk through means tested social insurance has the unintended negative consequences of reducing labor supply and human capital accumulation, and increasing earnings inequality.

Finally, there is a large literature that studies the impact of various aspects of socioeconomic status (education, income, wealth) on health. For example, see [Adams et al. \(2003\)](#), [Stowasser et al. \(2011\)](#), [Currie and Madrian \(1999\)](#), [Hall and Jones \(2007\)](#) and [Galama and Van Kippersluis \(2018\)](#). As [Smith \(1999, 2004\)](#) discusses, this literature faces difficult issues of disentangling causality. He recommends analyzing effects of employment and earnings shocks on health status while controlling for lagged health and human capital. Along these lines, several papers have examined the effect of exogenous job separations on health ([Eliason and Storrie \(2009\)](#), [Black et al. \(2015\)](#), [Schaller and Stevens \(2015\)](#)). They find that job loss leads to worse health behaviors, worse self-reported health, and worse mental health. However, they do not find short-run effects on chronic conditions or frequency of health shocks. Similarly, [Adda et al. \(2009\)](#) look at effects of permanent and transitory income shocks on health using cohort level data. They find no effects on health over a 3-year horizon, but they do find effects on mortality and health related behaviors.

We argue that in order to estimate the effects of income and employment on health, the health production should also control for health shocks. Health shocks may affect contemporaneous earnings and labor supply, so a failure to control for health shocks may cause one to falsely assign the effects of health shocks to earnings and employment. In other words, it could be that lower employment is associated with worse health transitions in part because unobserved innovations to health affect both variables negatively. This leads to an upward bias in the estimated effect of employment on health. We contribute to this literature by estimating the effect of employment (and income) on health transitions using a model that explicitly controls for the onset of health shocks, eliminating this potential bias.

### 3 Model

In our life-cycle model agents face idiosyncratic risk to wages, employment, earnings, health and survival. They enter the economy at age 25 and face survival risk every period. The model period is one year, and the maximum lifespan is 100. Retirement is exogenous at age 65. From age 25 to 64, agents receive employment offers probabilistically each year, and decide on whether to accept or reject them. They also make a continuous consumption/savings decision, but borrowing is not allowed. Workers accumulate human capital through work experience. The model is solved in partial equilibrium, assuming a fixed interest rate and a fixed rental rate on skill.

Education is taken as given at age 25 when agents enter the model. We assume three education groups: high school (HS) or less, some college (1-3 years), and college graduates.<sup>8</sup> We allow most model parameters, including the health and human capital production functions, tastes for leisure and job offer probabilities, to differ by education group. Consistent with prior work, we find the health production function differs in important ways by education, but a limitation of our analysis is that we do not attempt to explain why.<sup>9</sup>

#### 3.1 The Timing of Decisions and Shocks



Agents begin each period ( $t$ ) with stocks of assets  $A_t$ , human capital  $HC_t$ , functional health  $H_t$ , and asymptomatic risk factors  $R_t$ . Working age individuals in poor functional health also know their disability insurance (DI) status  $I_t^{DI}$ , which affects the level of government transfers received. Immediately after the start of the period, working age individuals receive an employment offer, which can be either full or part-time, and with or without employer health insurance. Wage offers are determined by health and human capital and are subject to temporary and persistent shocks. Agents decide whether to accept or reject the tied wage/hours/insurance offer. Then health shocks are realized. These, together with functional health, determine mortality, medical expenditures and sick days. Sick days reduce work hours and reduce the accumulation of human capital. Next, agents make a continuous

<sup>8</sup>These three education groups make up 40%, 27% and 33%, respectively, of the working age population in the CPS from 2000-2010. The fraction of HS dropouts is relatively small (11%), so we combine them with the HS graduates (29%).

<sup>9</sup>Grossman (2006) provides a survey of the literature on the relationship between health and education. The literature suggests that educational attainment leads to greater efficiency in health production (e.g., Lleras-Muney (2006), Oreopoulos (2007), Grossman (2000, 2006)). We abstract from modeling the effect of health on formal education choices, an issue that is analyzed in Hai and Heckman (2015).

consumption/saving decision. Finally, next period state variables become known, and the next period begins. We let  $t$  denote both the time period and the age of the individual.

### 3.2 Health and Health Shocks

An important feature of our model is a detailed specification of the processes for health and health shocks over the life-cycle. There are two stocks of health: functional health ( $H_t$ ) and underlying asymptomatic health risk ( $R_t$ ). And in each period agents can experience three types of health shocks: predictable and persistent ( $d_t^p$ ), unpredictable and persistent ( $d_t^u$ ), and unpredictable and transitory ( $s_t$ ). Details are as follows:

Functional health status  $H_t$  measures the ability to perform daily activities and function in a work environment. Thus, it impacts on productivity. It is discrete and can take three values: poor, fair or good ( $H_t \in \{P, F, G\}$ ). In contrast, the stock of underlying health risk  $R_t$  has no impact on current productivity.  $R_t$  captures asymptomatic risk factors whose only effect is to increase the probability of predictable health shocks ( $d_t^p$ ) in the future. Examples are obesity and high cholesterol, which increase the probability of heart disease.  $R_t$  is also discrete with three values: low, medium or high ( $R_t \in \{L, M, H\}$ ).  $H_t$  and  $R_t$  evolve from year-to-year with transition probabilities that we describe below.

Let  $\Upsilon_t = (d_t^p, d_t^u, s_t)$  be a vector of indicator functions for occurrence of health shocks. All three types of health shock affect ability to function in the *current* period. The persistence of shocks is categorized as short or long-term. For example, a broken is a short-term shock that affects the individual *only* in the current period. Long-term shocks, such as damage to the spinal column, have effects that last for multiple periods. Our model captures this by letting the transition probabilities for  $H_t$  and  $R_t$  depend on persistent shocks ( $d_t^p, d_t^u$ ).<sup>10</sup>

Persistent shocks are classified as predictable ( $d_t^p$ ), or unpredictable ( $d_t^u$ ). We assume all transitory shocks ( $s_t$ ) are unpredictable.<sup>11</sup> The “predictable” shocks ( $d_t^p$ ) have a probability of occurrence that depends on  $H_t$  and  $R_t$ , along with age and education. Examples are stroke and lung cancer. Probabilities of “unpredictable” shocks  $d_t^u$  and  $s_t$  depend only on age.

The following table lists the state variables that enter the transition probabilities for  $H_t$  and  $R_t$  and the probabilities of health shocks  $d_t^p$ ,  $d_t^u$  and  $s_t$ . For example, functional health  $H_t$  evolves according to a transition matrix that depends on the current level of  $H$ , age, long-term health shocks ( $d_t^p, d_t^u$ ), employment and health insurance status (summarized by the categorical variable  $O$ ), education, and income group ( $inc$ ). We assume the probabilities of initial levels of  $H$  and  $R$  at age 25 depend only on education.

Variable	Transition Probability Matrix / Probability
$H_t$	$\Lambda_H(H', H, t, d^p, d^u, O, educ, inc)$
$R_t$	$\Lambda_R(R', R, t, d^p, d^u, H, O, educ, inc)$
$d_t^p$	$\Gamma^{dp}(R, H, t, educ)$
$d_t^u$	$\Gamma^{du}(t)$
$s_t$	$\Gamma^s(t)$

<sup>10</sup>Thus, lagged shocks are not state variables. Effects of all lagged shocks are embedded in  $H_t$  and  $R_t$ .

<sup>11</sup>In the data section, we show there are very few medical conditions that are predictable but short lasting.



Finally, the survival probability (year-to-year) depends on functional health, age, and long-term health shocks, and is given by  $\varphi(H_t, t, d_t^p, d_t^u)$ . The risk factors  $R$  affect the survival probability indirectly, by altering the probability of adverse health shocks  $d_t^p$ .

### 3.3 Medical Expenditures

We treat medical expenditures as exogenous cost shocks. They are a function of health, health shocks, age, and a stochastic term  $\varepsilon^{ME}$ , and are given by  $ME(H_t, \Upsilon_t, t, \varepsilon^{ME})$ . The shock  $\varepsilon^{ME}$  determines whether the person must bear the normal level of treatment cost associated with their state ( $\varepsilon^{ME} = 0$ ), or a higher "catastrophic" level of cost ( $\varepsilon^{ME} = 1$ ). The catastrophic level of costs varies by health state  $(H_t, \Upsilon_t, t)$ , but we assume the probability of a catastrophic shock is uniform across health states  $(H_t, \Upsilon_t, t)$ , and is given by  $\delta$ .

We assume that all individuals must bear the cost of treatment associated with their medical condition (as drawn from  $ME(\cdot)$ ). In reality people may have choices about their course of treatment, and thus have some control over costs. But we abstract from this, in effect assuming people lack the medical knowledge to make such decisions.<sup>12</sup>

Our model directly captures the costs of health shocks  $d_t^p$ ,  $d_t^u$  and  $s_t$  only in the year in which they occur. However, persistent health shocks  $d_t^p$  and  $d_t^u$  lead to higher probabilities of poor health in future periods, and hence higher expected future medical expenditures.

### 3.4 Health Insurance

Health insurance is of three types: (1) employer provided, (2) Medicare, and (3) all other forms of public insurance, including Medicaid and disability. Employer provided insurance is available to a fraction of workers, as described in the next section. Workers whose employers provide health insurance pay an out-of-pocket premium  $p^{EI}$ .<sup>13</sup> Employer insurance pays for a fraction  $q^{EI}$  of workers total medical costs. Medicare is available to those 65 and older, and it covers a fraction  $q^{Med}$  of medical expenditures. The Medicare premium is  $p^{Med}$  is paid by those 65 and over, and a payroll tax  $\tau^{Med}$  is paid by workers.

Given their resources, some individuals may be unable to afford the level of medical costs they draw from  $ME(\cdot)$ , at least not while also maintaining a minimum level of consumption. In such cases, we assume the government provides a guaranteed consumption floor, described in section 3.6. This is meant to capture programs like Medicaid that cover medical expenses of the poor, as well as other social programs like Food stamps. It also captures the possibilities of simply not paying hospital bills or declaring medical bankruptcy.<sup>14</sup>

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<sup>12</sup>Thus medical expenditures are non-discretionary, and they do not *directly* affect health in our model. De Nardi et al. (2010) argue that medical spending that supplements Medicaid, Medicare, and private insurance has very small effects (if any) on health of the U.S. elderly. Finkelstein and McKnight (2008) find that Medicare did not significantly increase life expectancy in the first 10 years after its introduction. There are a few studies that find expenditures are positively related to survival, but these consider only a subset of conditions, mainly related to emergency room visits (see Card et al. (2009) and Doyle (2011)).

<sup>13</sup>Employers pay on average 81% of health insurance premiums for singles (Kaiser Family Foundation (2010)). We only model the part paid for by the employee. The premium for employer provided health insurance does not vary with health status, age or other personal characteristics.

<sup>14</sup>As we rule out borrowing, we cannot explicitly model bankruptcy decisions. Himmelstein et al. (2009), Livshits et al. (2010) and Gross and Notowidigdo (2011) specifically study medical bankruptcies.

Finally, to capture disability insurance benefits in a simple way, we assume working age people in poor functional health are probabilistically eligible for a higher consumption floor (see Benítez-Silva et al. (1999), Low and Pistaferri (2015)). This is meant to approximate benefits from the SSI and SSDI programs. See Section 3.6 for details.

## 3.5 Employment

### 3.5.1 Employment Offers

At the start of each period, and before health shocks are realized, individuals aged 25 to 64 receive employment offers probabilistically. If an offer is received, an individual decides whether to accept or reject it. Employment offers are characterized by a wage, number of hours, and the provision of employer health insurance. Letting  $*$  superscripts denote offers, we have:  $\{W^*, h^*, ins^*\}$ . Wage offers are continuous, and are described in detail in section 3.5.4. The number of hours  $h^*$  takes one of three values, 0 (no offer),  $hrs^{PT}$  (part-time) or  $hrs^{FT}$  (full-time),  $h^* \in \{0, hrs^{PT}, hrs^{FT}\}$ . Insurance  $ins^* \in \{0, 1\}$  is an indicator for whether the offer includes health insurance. We let the categorical variable  $O^*$  summarize employment offers based on the five possible combinations of hours and insurance.<sup>15</sup>

The probability of receiving each type of offer  $O^*$  depends on education and age, and is given by  $\Pi(O^*, educ, t)$ . To help capture the decline in hours at older ages observed in the data, we allow for a positive probability of receiving no offer at ages 54+. This may be interpreted as a simple way to capture various reasons that employers are reluctant to hire older workers. At younger ages, all non-employment is voluntary.

When employment offers are accepted or rejected, medical expenditures are not yet known, as health shocks occur after the decision is made. However, individuals know  $H_t$  and  $R_t$ , so they can calculate *expected* medical expenditures.

After individuals accept/reject their employment offer(s), employment and health insurance status are summarized by the categorical variable  $O = \{W, h, ins\}$ .

### 3.5.2 Hours Worked and Sick Days

When an individual accepts an employment offer, he commits to working  $h^*$  hours at wage  $W^*$ . This commitment is fulfilled unless the worker experiences sick days. Sick days  $sd(educ, H_t, \Upsilon_t)$  are a function of education, health and health shocks. The actual number of hours worked by those who participate in the labor force is given by  $h_t = h^* - sd(educ, H_t, \Upsilon_t)$ .

We assume health shocks do not affect wages within a period. Employers cannot lower wages immediately if an employee receives a negative health shock. However, health shocks may force workers to reduce work hours so as to attend doctor appointments, undergo treatment, or simply rest. Thus, the model captures the fact that a worker may have high human capital and high wages, yet, have little earning capacity for health reasons. We allow sick days to vary by education level to capture the fact that ability to work after health shocks differs by occupation. We assume all sick days are unpaid.<sup>16</sup>

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<sup>15</sup>Specifically, the five possibilities are: no offer ( $h = 0$ ), part-time offer with and without insurance, and full-time offer with and without insurance.

<sup>16</sup>In reality, workers have on average 7 paid sick days per year (BLS Statistics).

### 3.5.3 Human Capital Accumulation

Human capital  $HC$  is defined as a function of education and work experience. It evolves probabilistically according to the law of motion:

$$HC_{t+1} = (HC_t + h_t)\varepsilon_{t+1}^{HC} \quad (3.1)$$

where initial human capital  $HC_1$  differs by education, and  $\varepsilon^{HC}$  is a shock governed by:

$$\varepsilon_{t+1}^{HC} = \begin{cases} 1 + \nu & \text{with probability } p^1(educ, I_w) \\ 1 & \text{with probability } 1 - p^1(educ, I_w) - p^2(educ, I_w) \\ 1 - \nu & \text{with probability } p^2(educ, I_w) \end{cases} \quad (3.2)$$

Probabilities of human capital “shocks” (i.e., increments) depend on education and an indicator  $I_w$  equal to 1 if an agent is employed and 0 otherwise. We expect more educated employed workers are more likely to receive positive shocks, given the evidence they have faster wage growth with experience (Imai and Keane (2004)). We expect unemployed workers are more likely to receive negative shocks, due to skill depreciation during unemployment (e.g., Low et al. (2010), Rogerson and Schindler (2002), Ljungqvist and Sargent (1998)).

### 3.5.4 The Wage Offer Function

The wage offer function is given by:

$$\ln W^* = w(educ, HC, H, h^*) + \kappa_j(educ) + \varepsilon^W \quad (3.3)$$

$$w(educ, HC, H, h^*) = \beta_0 + \beta_1 HC + \beta_2 HC^2 + \beta_3 HC^3 + \beta_4 I_{H \in \{F, G\}} + \beta_5 I_{H=G} + \beta_6 I_{h^* = hrs^{PT}} \quad (3.4)$$

Wage offers  $W^*$  depend on (1) a function  $w(educ, HC_t, H, h^*)$  that depends on education, human capital, health and hours, (2) the agent’s productivity type  $\kappa_j$ , and (3) transitory shocks  $\varepsilon_t^W$ . The latent type  $\kappa$  is age invariant and discrete, with  $j$  indexing types (see Section 5.2.4 for details). Transitory wage shocks are distributed as  $\varepsilon^W \sim N(0, \sigma_{\varepsilon^W}^2(educ))$ . We assume *observed* log wages include additive measurement error  $\varepsilon^N \sim N(0, \sigma_{\varepsilon^N}^2(educ))$ .

The function  $w(educ, HC, H, h^*)$ , that combines human capital  $HC$  and functional health  $H$  to determine the mean of the (log) wage offer distribution, is given in equation 3.4. Here  $I_{H \in \{F, G\}}$  is an indicator equal to 1 for people in fair or good health and 0 for those in poor health, while  $I_{H=G}$  is an indicator equal to 1 for those in good health and zero otherwise.

We also let the mean of the wage offer distribution depend on  $I_{h^* = PT}$ , and indicator equal to one for part-time offers, to capture the observation that part time-wages tend to be lower than full-time wages - see Moffitt (1984), Lundberg (1985), and Aaronson and French (2004).

The parameters  $\beta_0 - \beta_6$  of 3.4 are all allowed to be education-level specific.

Persistent shocks to wages arise from three distinct sources: 1) the persistent shocks  $\varepsilon_{t+1}^{HC}$  to the human capital process in 3.1, 2) the persistent health shocks that affect wages through persistent effects on  $H$ , and 3) long-term effects that arise endogenously through workers’ responses to all current period shocks - including transitory shocks - as these responses are embedded in the next period’s human capital and assets.

Transitory health shocks affect current earnings through sick days that reduce current work hours. This generates persistence as next period human capital and assets are reduced. Similarly, employment offer risk (i.e., the possibility of no offer) and labor force participation decisions also generate persistence through their effects on  $HC$  and  $A$ .

### 3.6 Taxes, Social Security and Social Insurance

We assume all workers retire at age 65, at which point they start to receive Social Security payments. Social Security rules are complex, and our focus is on sources of earnings uncertainty for working age men, so we abstract from the details of the rules. We simply assume the Social Security benefit is a constant  $SS(educ)$  that depends only on education.<sup>17</sup>

For individuals aged 25 to 64, taxable income  $y_t$  equals the sum of labor and capital income, minus the employee health insurance premium  $p^{EI}$  for those with insurance, and minus the tax deductible part of out-of-pocket medical expenditures (i.e., expenses in excess of 7.5% of income). The taxable income for retirees is similar, except Social Security income replaces labor income. Letting  $I_w$  be an indicator for employment, we have:

$$\begin{aligned} y_{t < 65} &= \max[0, rA + I_w(W^*h - p^{EI}ins^*) - \max(0, ME(1 - q^{EI}I_wins^*) - 0.075(rA + I_wW^*h))] \\ y_{t \geq 65} &= \max[0, rA + SS - \max(0, ME(1 - q^{Med}) - 0.075(rA + SS))] \end{aligned} \quad (3.5)$$

We follow [Jeske and Kitao \(2009\)](#) and [Pashchenko and Porapakkarm \(2016\)](#) in modeling income taxes. All individuals pay an income tax  $T(y_t)$  that consists of a progressive and a proportional tax. The function  $T(y)$  includes non-linear and linear components:

$$T(y) = a_0[y - (y^{-a_1} + a_2)^{-1/a_1}] + \tau_y y. \quad (3.6)$$

The non-linear component approximates the progressive US federal tax schedule, following [Gouveia and Strauss \(1994\)](#). The linear component captures other taxes, such as State taxes.

Workers also face payroll taxes. They pay a Medicare tax  $\tau^{Med}$  (on earnings minus the premium  $p^{EI}$ ) and a Social Security tax  $\tau^{SS}$  (on earnings minus the premium  $p^{EI}$ , up to the income threshold  $\bar{y}_{ss}$ ). Total income and payroll taxes are given by:

$$Tax = T(y) + I_w[\tau^{SS} \min(W^*h - p^{EI}ins^*, \bar{y}_{ss}) + \tau^{Med}(W^*h - p^{EI}ins^*)] \quad (3.7)$$

Consumption is taxed at the rate  $\tau^c$ , which captures sales taxes.

We assume there exists a public social welfare program that guarantees a minimum level of consumption  $\bar{c}(educ, I^{DI})$  to every individual. This consumption floor approximates a range of benefits we do not explicitly model, such as Medicaid, Food stamps, unemployment benefits, workers' compensation, Social Security Disability Insurance (SSDI), and Supplemental Security Income (SSI).  $I^{DI}$  is a 1/0 indicator for disability insurance (DI) eligibility.

As we noted in Section 3.4, our model incorporates a simple form of disability insurance. Individuals are eligible for disability with a probability  $\eta(educ, H, t)$  that depends on education, functional health, and age. Only working age individuals in poor health have positive

<sup>17</sup>This is a common assumption in the macro-health literature that focuses on working age individuals. See for example [Jung and Tran \(2016\)](#), [Pashchenko and Porapakkarm \(2016\)](#) and [De Nardi et al. \(2017\)](#) who also assume that Social Security payments depend only on fixed types.

probability of DI eligibility. Those eligible for DI have a higher level of the consumption floor  $\bar{c}(educ, I^{DI})$ . We calibrate  $\bar{c}(educ, I^{DI})$  to match benefits observed in the data.

When disposable income (net of medical costs) falls below  $\bar{c}$ , the person receives a transfer  $tr$  that compensates for the difference. Thus the transfer is given by:

$$\begin{aligned} tr_{t < 65} &= \max\{0, (1 + \tau^c)\bar{c} + ME(1 - q^{EI}I_w ins^*) - (1 + r)A - I_w(W^*h - p^{EI}ins^*) + Tax\} \\ tr_{t \geq 65} &= \max\{0, (1 + \tau^c)\bar{c} + ME(1 - q^{Med}) + p^{Med} - (1 + r)A - SS + Tax\} \end{aligned} \quad (3.8)$$

### 3.7 Preferences

In each period, agents derive utility from consumption ( $c$ ) and leisure ( $l$ ). The within-period utility function is given by:

$$u(c, l) = \frac{1}{1 - \sigma} [c^\alpha l^{(1-\alpha)}]^{(1-\sigma)} + \zeta I_{death}. \quad (3.9)$$

Leisure is equal to the total time endowment (normalized to one) minus the dis-utility of work expressed in units of leisure time, given by  $\phi(educ, H, h^*)$ . We have:

$$l = 1 - I_w \phi(educ, H, h^*). \quad (3.10)$$

The time cost of work depends on education, health and hours of work (part-time or full-time). Workers in poor health must expend more effort to work any agreed number of hours  $h^*$ , so they have greater dis-utility of work (expressed in leisure units). Also, the dis-utility of work  $\phi$  depends on  $h^*$ , not on the actual number of hours worked after sick days are realized  $h$ . This embeds an assumption that sick days provide no additional leisure to workers. For retirees, leisure is equal to 1, so utility is only a function of consumption.

The utility function in 3.9 creates an incentive for individuals to smooth the consumption/leisure aggregate  $c^\alpha l^{(1-\alpha)}$  over time. This causes consumption to drop at retirement. Also, given that poor health reduces effective leisure time of workers in 3.10, consumption will tend to increase if workers are in poor health (*ceteris paribus*).

We assume a utility cost of death  $\zeta$  that is incurred only in the period when the individual dies, in which case the indicator  $I_{death} = 1$ . We introduce this feature because the first term of 3.9 can be negative. This is not a problem in life-cycle models without health, but here it could have the perverse effect of causing individuals to value behaviors that lower  $H$  so as to reduce the survival probability. Introducing a dis-utility of death avoids this problem.

### 3.8 Individual's Problem

#### 3.8.1 Working Age Individuals

At the beginning of every period, an agent's state includes his age, education, fixed productivity type, functional health, health risk, human capital, assets, DI eligibility, and the employment offer. Letting  $\chi$  denote the state vector we have:

$$\chi = (t, educ, \kappa, H_t, R_t, HC_t, A_t, I_t^{DI}, (W_t^*, h_t^*, ins_t^*)) \quad (3.11)$$

Given  $\chi$ , an agent decides whether to accept or reject the employment offer, so as to maximize the expected present value of lifetime utility. This decision is summarized by the indicator function  $I_w$ . After the labor supply decision is made, health shocks are realized. Then the agent draws medical expenses, including the shock  $\varepsilon^{ME}$  that determines if expenses are “catastrophic.” The agent experiences sick days given by the function  $sd(educ, H_t, \Upsilon_t)$ . At this stage, the state of the agent is summarized by  $\chi$ ,  $I_w$ , the vector of health shocks  $\Upsilon = (d^p, d^u, s)$ , and  $\varepsilon^{ME}$ . Finally, he makes the consumption/savings decision.

The agent solves the problems in two stages. First, he solves for the policy function for consumption conditional on  $\chi$  and all possible realizations of  $\Upsilon$ , and  $\varepsilon^{ME}$ , for both  $I_w = 0$  and  $I_w = 1$ . This policy function  $c(\chi, I_w, \Upsilon, \varepsilon^{ME})$  is the solution to the problem:

$$G(\chi, I_w, \Upsilon, \varepsilon^{ME}) = \max_c \{u(c, l) + \beta E_{\Psi} V(\chi')\} \quad (3.12)$$

where the expected value of the next period’s state is calculated over the probabilities of all possible realizations of  $\Psi \equiv (O^*, H', R', I^{DI'}, \varepsilon^{HC'}, \varepsilon^{W'})$ , which uniquely determine  $W^*$ , and where the maximization is subject to equations 3.5 to 3.10 and:

$$\begin{aligned} A' &= (1+r)A + I_w(W^*h - p^{EI}ins^*) + tr - (1+\tau^c)c \\ &\quad - ME(H, \Upsilon, t, \varepsilon^{ME})(1 - q^{EI}I_w ins^*) - Tax \end{aligned} \quad (3.13)$$

$$\begin{aligned} c &\leq \frac{1}{1+\tau^c} [(1+r)A + I_w(W^*h - p^{EI}ins^*) + tr \\ &\quad - ME(H, \Upsilon, t, \varepsilon^{ME})(1 - q^{EI}I_w) - Tax] \end{aligned} \quad (3.14)$$

Equation 3.14 is the no-borrowing constraint. After solving for the policy functions, the agent chooses whether to accept or reject the employment offer by solving:

$$V(\chi) = \max_{I_w} E_{(\Upsilon, \varepsilon^{ME})} \{ \varphi G(\chi, I_w, \Upsilon, \varepsilon^{ME}) \}. \quad (3.15)$$

Here the expectation is taken over the probabilities of all possible  $\Upsilon$  and  $\varepsilon^{ME}$ . The survival probability  $\varphi = \varphi(H_t, t, d_t^p, d_t^u)$  was defined in Section 3.2.

### 3.8.2 Retired Individuals

After age 65, when retirement occurs exogenously, an individual makes decisions on consumption only. At the time these decisions are made, the state of the individual is given by age, education, health, health risk factors, assets, health shocks and medical expense shocks. The agent maximizes the expected present value of lifetime utility by solving the problem:

$$V(t, educ, H, R, A, \Upsilon, \varepsilon^{ME}) = \max_c \{u(c) + \beta E \varphi V(t+1, educ, H', R', A', \Upsilon', \varepsilon^{ME'})\} \quad (3.16)$$

subject to equations 3.5 to 3.10 and:

$$A' = (1+r)A + SS + tr - (1+\tau^c)c - ME(H, \Upsilon, t, \varepsilon^{ME})(1 - q^{Med}) - p^{Med} - T(y) \quad (3.17)$$

$$c \leq \frac{1}{1+\tau^c} [(1+r)A + SS + tr - ME(H, \Upsilon, t, \varepsilon^{ME})(1 - q^{Med}) - p^{Med} - T(y)] \quad (3.18)$$

The solution algorithm is described in Appendix A.

## 4 Data and Variable Construction

Our main data set is the Medical Expenditure Panel Survey (MEPS), a rotating panel in which each household is interviewed 5 times over two and a half years. A new panel is sampled every year. We use panels 5 to 16 covering years 2000 to 2012. Panels 1-4 are not used because some key variables are not available before 2000. Our sample consists of males 25 years of age and older as of the beginning of the survey. We also use the CPS, HRS, PSID and CEX to construct other statistics used in the analysis.

### 4.1 Constructing Health Shocks ( $d^p$ , $d^u$ , and $s$ )

An important advantage of MEPS over other panel surveys is that it contains information on respondents' detailed medical conditions. The medical conditions and procedures reported by respondents were recorded by interviewers as verbatim text which was then coded by professional coders into three digit ICD-9 codes.<sup>18</sup> The high level of detail in the classification of conditions allows us to distinguish the different types of health shocks in our model.

We categorize each of the 989 3-digit ICD-9 medical conditions based on four criteria: 1) short-term productivity loss, 2) long-term productivity loss, 3) predictive power, and 4) predictability.<sup>19</sup> Productivity loss includes both productivity at work and limitations in daily functioning. We define a *short-term productivity loss* as one that lasts for at least 2 weeks per year but for less than two years.<sup>20</sup> A *long-term productivity loss* occurs if a medical condition has an impact for at least 2 weeks per year for more than two years. A medical condition is classified as a *predictor* if it increases the probability of other medical conditions arising in the future. Finally, a condition is classified as *predictable* if health related behavior and prior health conditions are together implicated in at least 50% of its occurrences.<sup>21</sup>

Table 2 shows how we map ICD-9 conditions that satisfy different combinations of these four criteria into the  $d^p$ ,  $d^u$ , and  $s$  shocks. Conditions with no effect on current productivity are not classified as health shocks, but they may be risk factors (see below). Conditions with both current and long-term effects are classified as  $d^p$  shocks if predictable, and  $d^u$  shocks if not. Conditions with only short-term effects are labeled  $s$  shocks. We define  $d_t^p$ ,  $d_t^u$ , and  $s_t$  as 1/0 indicators of whether a respondent has one or more conditions of each type. They are constructed at the annual level, based on the the two years of interviews in each panel.

Table 2 also reports the number of ICD-9 codes in each category. A total of 65 conditions are classified  $d^p$ , while 290 are  $d^u$  and 315 are  $s$ . Note that only 9 short-term conditions are classified as predictable, and in our sample their combined prevalence is only 0.5%. Rather than have a separate category for such rare shocks, we include them as part of  $s$ . We also include the “unknown” conditions as part of  $s$ , because the Appendix shows they have characteristics similar to the short-term unpredictable health shocks.

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<sup>18</sup>The International Statistical Classification of Diseases and Related Health Problems (abbreviated ICD) is published by the World Health Organization and is used world-wide for morbidity and mortality statistics, reimbursement systems and automated decision support in medicine.

<sup>19</sup>We are grateful to Dr. Phil Haywood, a clinician and research fellow at the Centre of Health Economic Research and Evaluation at University of Technology Sydney, who classified ICD codes based on our criteria.

<sup>20</sup>The two week minimum is meant to rule out short-run minor illnesses like the common cold.

<sup>21</sup>Some ICD codes have different characteristics by age. We split these into separate conditions by age.

## 4.2 Constructing Health ( $H$ )

Our functional health measure ( $H$ ) combines self-reports and objective measures. Specifically, it is constructed from the following MEPS variables: 1) self-reported health, 2) self-reported mental health, 3) activities of daily living (ADL) limitations, 4) instrumental activities of daily living (IADL) limitations, and 5) a set of eight physical functioning limitations.<sup>22</sup>

Self-reported health and mental health take values from 1 to 5 indicating poor, fair, good, very good and excellent. The ADL and IADL variables are binary indicators for the presence of any limitations. We construct a score for physical functioning limitations from the eight categorical variables. All five variables are standardized using data on all men 25 and over.

We conduct factor analysis on these five standardized variables. The results are reported in Appendix B. All five variables load highly on the first factor, which we interpret as functional health. We use the factor scores to construct functional health for all individuals in interviews 1, 3, and 5. These correspond to initial health, and health 1 and 2 years later.

Finally, as this health measure is continuous, we discretize it into three categories corresponding to poor, fair and good functional health (as in the model).<sup>23</sup> Figure 1 presents the distribution of  $H$  by age. Of course, the fraction of people in good health declines with age. The figure also reveals a strong positive correlation between education and good health even at young ages. At age 25 over 80% of college types are in good health, compared to about 60% of high school types. By age 65 the divergence swells to about 60% vs. 35%.

## 4.3 Constructing Asymptomatic Health Risk ( $R$ )

Table 2 also lists the criteria a medical condition must satisfy to be categorized as an asymptomatic risk factor. These conditions do not affect current (short-term) productivity but they predict future health conditions and/or long-term productivity. There are 41 ICD-9 conditions that meet our criteria. Of these, only 28 are present in our sample. In addition, we use 8 items in the ICD-9 classification that measure family history of disease. The 36 ICD-9 codes used in the construction of  $R$  are listed in Appendix B.

We first construct three variables that summarize these 36 conditions: 1) an indicator for essential hypertension, which has no identifiable cause, 2) an indicator for disorders of lipid metabolism, e.g., high cholesterol, and 3) the count of all other ICD-9 conditions used to construct  $R$ . Hypertension and high cholesterol are by far the most common risk factors, which is why we group all others together. We construct a measure of excessive BMI and a measure of low BMI. All five variables are standardized using data on all men 25 and over.

We take a weighted sum of these five variables to form a scalar measure. The weights are based on the relative importance of each variable for predicting the health shocks  $d_t^p$  (see Appendix B for details).<sup>24</sup> We do this to construct measures of  $R$  for all individuals in

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<sup>22</sup>These measure difficulty with 1) lifting 10 pounds, 2) walking up 10 steps, 3) walking 3 blocks, 4) walking a mile, 5) standing 20 minutes, 6) bending/stooping, 7) reaching overhead, and 8) using fingers to grasp.

<sup>23</sup>Our discretization is based on the distribution of the continuous health factor among all males aged 25 and over. Good health corresponds to values of the health factor above the median. Poor health corresponds to values at least one standard deviation below the mean. Fair health corresponds to the interval in between.

<sup>24</sup>The astute reader may notice an asymmetry: We form  $H$  by combining conditions using factor scores, while we form  $R$  by taking a weighted sum based on predictive ability. We think this makes sense, given that  $H$  is meant to be a scalar measure of overall health, which is the type of measure factor analysis is designed to construct, while  $R$  plays a very different role as a best predictor of future medical conditions.



interviews 1, 3, and 5. These correspond to initial  $R$ , as well as 1 and 2 years later.

Finally, we discretize the health risk variable into three categories corresponding to low, medium, and high risk, as in the model.<sup>25</sup> This is done separately by education, so a fixed fraction of individuals falls into each risk class *within* each education group.<sup>26</sup> Figure 1 shows the distribution of the final health risk variable by age. The fraction of high risk individuals is almost zero at age 25, but grows to approximately 30% at age 65.

## 4.4 Reduced Form Regressions

Table 1 presents regressions of labor market outcomes on health shocks, along with controls for lagged health and human capital, using data from the MEPS. These specifications are consistent with Smith (1999)’s approach to estimating effects of health shocks. We find no significant effects of health shocks on *current* wages, consistent with our assumption that wages do not respond immediately. But we see significant declines in work hours and annual earnings following all three types of shocks ( $d^p$ ,  $d^u$ ,  $s$ ). The finding that health has greater short-run effects on hours than wages is consistent with our modeling assumptions.

## 5 Calibration

Our benchmark model is calibrated to features of the US economy for the period 2000 to 2010, for civilian, non-institutionalized 25+ year old males who are not in school. We estimate some parameters directly from the MEPS data, while calibrating others (i) to match moments of the data, or (ii) based on prior work. Most parameters are calibrated separately for the three education groups (high school, some college, college).

### 5.1 Parameters Estimated from the MEPS Data

#### 5.1.1 Transition Probabilities: Functional Health and Health Risk

As  $H$  and  $R$  are discretized into 3 levels, we specify their laws of motion as multinomial logits. Recall we have  $\Lambda_H(H', H, t, d^p, d^u, O, educ, inc)$  and  $\Lambda_R(R', R, t, d^p, d^u, H, O, educ, inc)$ . We estimate separate models for the 25-64 and 65+ populations. This is because  $O$  and  $inc$  are irrelevant for the latter, as we assume everyone retires at 65 and is covered by Medicare. The estimates are reported in Appendix B.<sup>27</sup>

Our logit specification for health transitions implies the existence of “idiosyncratic” health shocks that cause  $H$  and  $R$  to change from  $t$  to  $t + 1$  for reasons not captured by the observed health shocks or other state variables that enter  $\Lambda_H(\cdot)$  and  $\Lambda_R(\cdot)$ . This “idiosyncratic” health risk is accounted for by agents when they solve the problem in Section 3.8. However, as these logit errors are not revealed until time  $t + 1$ , they cannot directly affect time  $t$  decisions.

It is internally consistent to estimate the law of motion for health separately from our structural labor supply model if the errors in the  $H$  equation are independent of other sources of error in the structural model. That is true, given our assumption that the errors in the

<sup>25</sup>We discretize continuous  $R$  analogously to how we discretized  $H$ . That is,  $R$  is “Low” if its value is below the median, “High” if it is above the mean plus one standard deviation, and “Medium” if it falls in between.

<sup>26</sup>We do this because the role of  $R$  in the model is to predict  $d^p$  shocks, and education is also a predictor.

<sup>27</sup>We find that income and employment do not significantly affect the transitions for  $R$ , so in practice the  $R$  transitions entered in the model are independent of income and employment.

logit model  $\Lambda_H(\cdot)$  capture purely “idiosyncratic” health shocks, revealed after time  $t$  decisions are made. Then, the covariates in the  $H$  equation are exogenous. We argue this assumption is plausible given our rich controls for current health shocks and lagged health. In contrast, a failure to adequately control for time  $t$  health shocks may render  $O$  and  $inc$  endogenous in the  $H$  equation, as the omitted current health shocks could affect time  $t$  labor supply as well as  $H$  transitions. This illustrates why it is important to use the MEPS data to construct rich measures of health and health shocks. (A similar argument applies to the  $R$  equation, but we exclude  $O$  and  $inc$  from that equation as they were not significant).

Figure 1 shows distributions of predicted vs. actual  $H$  and  $R$  by age. Our models capture well the pattern that the prevalence of fair/poor health  $H$  both starts higher (at age 25) and increases much more quickly with age for less educated workers. In contrast, the rate of increase in risk factors  $R$  with age is similar for all education groups.

The left panel of Figure 2 shows how transition rates from fair-to-poor health vary by age and employment status for high school types with a  $d^u$  shock. The right panel shows the transition rate is small but positive even with no observed health shocks. This reflects the purely “idiosyncratic” health risk captured by the logit errors, as well as natural effects of aging. As expected, we see that the fair-to-poor transition rate increases substantially if a  $d^u$  shock occurs. Clearly, our measures of persistent health shocks are strong predictors of health transitions. Both panels show the transition rate increases if a person is not employed. In contrast, we find that  $R$  transitions are strongly predicted by lagged  $R$ ,  $d^u$  and  $d^p$  shocks, but not by employment status or income.

### 5.1.2 Probabilities of Health Shocks ( $d^p$ , $d^u$ , and $s$ )

The stochastic processes for the three types of health shock  $\Gamma^{dp}(R, H, t, educ)$ ,  $\Gamma^{du}(t)$ , and  $\Gamma^s(t)$  are specified as logits. The estimation results are reported in Appendix B.

Figure 3 shows that the frequency of unpredictable health shocks ( $d^u$ ,  $s$ ) increases rapidly with age, but it does not differ by education level (see Appendix B). In contrast, predictable health shocks ( $d^p$ ) are more likely for men with less education, particularly at older ages.

### 5.1.3 Survival Probabilities

We specify the annual survival probability  $\varphi(H_t, t, d_t^p, d_t^u)$  as a logit (see Appendix B). Consistent with Pijoan-Mas and Ríos-Rull (2014), we find that mortality does not depend on income or education once we condition on health. Nor is it significantly affected by temporary shocks  $s_t$  or health risk  $R_t$ .

### 5.1.4 Medical Expenditures

We use MEPS data on total annual medical expenditures to construct the expenditure function  $ME(H_t, \Upsilon_t, t, \varepsilon^{ME})$ , where  $\Upsilon = (d^p, d^u, s)$ .<sup>28</sup> For each  $(H_t, \Upsilon_t, t)$  cell, we take the 95th percentile as the cutoff between regular and catastrophic expenditures. We then calculate mean medical expenditures for men below and above the 95th percentile in each cell. In order to obtain smooth age profiles, we run regressions of these mean values on age and age

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<sup>28</sup>Total medical expenditures in MEPS are defined as the sum of direct payments for health care services provided during the year, including out-of-pocket payments and payments by private insurance, Medicaid, Medicare, and other sources. Payments for over-the-counter drugs are not included.

squared (see Appendix B) and use the fitted values to construct  $ME(\cdot)$ . Consistent with this, we set the probability of catastrophic expenditures in each  $(H_t, \Upsilon_t, t)$  state,  $\delta$ , to 5.0%.

It is well known that MEPS tends to underestimate aggregate medical expenditures (Pashchenko and Porapakkarm (2016), De Nardi et al. (2017)). Therefore, we follow De Nardi et al. (2017) and multiply the estimated medical expenses by 1.60 for men under 65, and by 1.90 for men 65 or older. This brings aggregate medical expenses computed from the MEPS in line with statistics in the National Health Expenditure Account (NHEA).

### 5.1.5 Hours Worked and Sick Days

We set hours in full and part-time employment offers,  $hrs^{FT}$  and  $hrs^{PT}$ , to 40 and 20 per week, respectively. These values are equal to median full and part-time hours of workers in good health with no health shocks in the MEPS.<sup>29</sup>

Next, we estimate sick days as the difference in annual hours worked between workers with no health shocks and those with various combinations of health shocks. Specifically, to estimate the function for sick days  $sd(educ, H_t, \Upsilon_t)$  we run regressions of *weekly* hours worked on age, age<sup>2</sup>, and all possible combinations of health shocks  $\Upsilon = (d^p, d^u, s)$ , separately by health  $H$  and education group. We report the results in Table 3.

Table 3 reveals that the long-term shocks  $d^p$  and  $d^u$  generate substantial losses of work hours. For example, for workers in fair health, and with college or some college education, a  $d^p$  shock reduces work hours by about 2.6 hours per week (or about 135 annually). Hours lost are much greater if multiple shocks occur together. For example, for workers in fair health, and with college or some college education, the joint occurrence of  $d^p$  and  $d^u$  shocks reduces work hours by about 7.3 hours per week (or about 380 annually).

## 5.2 Calibration of Remaining Parameters

We take several parameters from prior literature. These include utility function parameters, and parameters related to taxes, social security and health insurance. The values are listed in Table 4. The coefficient of relative risk aversion  $\sigma$  is set to 2.0, a widely used value. We take the progressive tax function parameters  $a_0$  and  $a_1$  from Gouveia and Strauss (1994). We take mean SS benefit levels (by education) from the HRS. The ESHI and Medicare coverage rates are set to 70% and 50% of medical expenditures, respectively, consistent with Attanasio et al. (2010) and Pashchenko and Porapakkarm (2016).

Table 5 lists all calibrated parameters and key moments we target for each. Of course, all calibrated model parameters affect all moments, but some parameters are relatively more important for particular moments. We now discuss identification of each parameter:

### 5.2.1 Time Discounting

We calibrate the discount factor  $\beta(educ)$  to match the average asset to income ratio observed in the PSID data for working age individuals aged 30 to 55, by education. As we see in the first row of Table 5, the college types are more patient.

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<sup>29</sup>According to our definitions, “not employed” means annual hours worked less than 520, “part-time” means annual hours between 520 and 1,500, and “full-time” means annual hours of 1,500+.

### 5.2.2 Dis-utility of Work

The leisure cost of work  $\phi(educ, H, h^*)$  is calibrated by targeting the shares of 30-50 year old men working full and part-time in the MEPS, by age, education, and health ( $H$ ). To eliminate the effect of sick days on hours we look at these statistics only for those without health shocks. The calibrated taste for work parameters are near the top of Table 5. Interestingly, they differ modestly by education/health, implying that differences in employment by education/health are mostly explained by differences in productivity and offer probabilities.

### 5.2.3 Employment Offer Probabilities

We calibrate job offer probabilities  $\Pi(O^*, educ, t)$  to target the shares of men employed full and part-time in the CPS, with and without insurance, conditional on education. The calibrated job offer probabilities are presented in Table 6. Clearly, the probability of receiving a full-time offer with health insurance is strongly increasing in education.

We assume men aged 25-53 always get a job offer, so all unemployment is voluntary.<sup>30</sup> At ages 54+, we allow for the possibility of receiving no offer, to better match the decline in labor force participation at later ages. We let the no-offer probability follow a linear trend in age, with a notch at 60, and parameters that differ by education.<sup>31</sup> In Table 5 we see the probability of receiving no offer increases more rapidly with age for the less educated.

### 5.2.4 The Offer Wage Function and Other Wage Parameters

In the data, we only observe earnings of those who *choose* to work. So the coefficients of the wage offer function would be subject to selection bias if estimated directly from observed wage data in a first stage.<sup>32</sup> Instead, we follow the internally consistent procedure of simulating data from the model, calculating the distribution of accepted wages among men who choose to work, and iterating on the wage parameters until the mean of simulated accepted wages matches as closely the means of accepted wages in the data (conditional on age, health status, full and part-time status, and education).

We also calibrate parameters that determine higher moments of wages. These are: (1) the levels of the latent productivity types  $\kappa_j(educ)$ , (2) the variance of the transitory shocks  $\sigma_{\varepsilon_w}^2(educ)$ , (3) the variance of measurement error in log wages,  $\sigma_N^2(educ)$ , and (4) the parameters that characterize the human capital shocks ( $\nu$ ,  $p^1(educ, I_{h_t > 0})$ , and  $p^2(educ, I_{h_t > 0})$ ).

To identify these parameters, we target: (i) the structure wage residuals, (ii) the variance of log wages by education, and (iii) transition rates from employment to non-employment for individuals in good health in consecutive periods.<sup>33</sup> We can separately identify measurement error from true wage shocks because measurement error affects (i)-(ii) but does not affect transition rates (iii).

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<sup>30</sup>Of course, some transitions from employment to non-employment before age 54 are due to involuntary separations. Our model captures this implicitly through the possibility of poor wage draws.

<sup>31</sup>Specifically,  $\Pi(O^*, educ, t) = \delta^O(educ, t)(t - 29)$  for  $t > 29$  and  $O^* = 1$  (no offer). Note that  $t = 30$  corresponds to age 54. We let  $\delta^O(educ, t)$  increase at  $t=35$ , which is age 60.

<sup>32</sup>It is useful to compare the logic here, as to why the wage equation must be estimated simultaneously with the full structural model, with our previous argument in Section 5.1.1 that it *is* consistent with the internal logic of our model to estimate the health transition process separately in a first stage.

<sup>33</sup>Transitions are also induced by health shocks, so to identify effects of wage shocks we target only transition rates for workers in good health in consecutive periods.

To obtain residual wages, we regress wages on a cubic in age, separately by education. Then, as in indirect inference, we use residual wages from both the simulated and real data to estimate a random effects plus AR(1) process, which we view as a descriptive model of the wage process. We target the following estimates from this descriptive model: the variance of the individual random effect, the variance of the transitory component, the AR(1) parameter and variance of the innovation in the AR(1) process.<sup>34</sup>

To calibrate the parameters of equation 3.2 that characterize the shocks to human capital, we set the increment  $\nu$  to 0.3 and calibrate the probabilities of positive and negative shocks. For employed workers, we assume the probabilities of positive and negative shocks are equal ( $p = p^1 = p^2$ ). Higher values of  $p$  generate more dispersion in wages and higher transition rates between employment and non-employment. For the unemployed, we assume the probability of a positive human capital shock is zero ( $p^1 = 0$ ), so we only calibrate the probability of a negative shock ( $p^2$ ). A higher  $p^2$  during non-employment periods implies more wage depreciation and a lower transition rate from non-employment to employment.

Finally, we allow for two fixed productivity types ( $\kappa$ ) in the “college” and “some college” groups, and three in the “high school” group (as it also includes dropouts). Within education groups, all productivity types are equal in size. They cover a range of roughly  $\pm 0.30$  sd. All calibrated wage process parameters are presented in the second panel of Table 5.

### 5.2.5 Consumption Floor, Disability Benefits, Dis-utility of Death, Taxes

We calibrate the consumption floor for non-disability recipients to match the percent of working age men who receive non-DI government transfers (conditional on education). We calibrate disability benefits to match average DI benefits in the CPS. We estimate DI benefits to be \$10,400 for the HS type, \$14,040 for the Some College type, and \$17,160 for the College type. These DI benefit levels are roughly double the basic (non-DI) floors.

Working age men in poor health are eligible for DI benefits with a positive probability  $\eta(educ, H, t)$ . Because we model DI as a higher consumption floor, only those who qualify for the floor  $\bar{c}(educ, I^{DI})$  get positive DI benefits. We calibrate  $\eta$  so the model matches the fraction of working age men who are DI recipients in the CPS.<sup>35</sup> We define DI benefits as including SSDI, SSI, and workers’ compensation.

We set the utility cost of death  $\zeta$  to equal the present value at age 25 of discounted future utility evaluated at the minimum consumption floor and a level of leisure associated with full-time employment in poor health, for those with high school or less. This ensures that all individuals prefer to live in all possible states. The final parameter value of  $\zeta = -30$  is set after calibrating the minimum consumption floor and dis-utility of work.

Finally, we calibrate the tax parameters  $a_2$  and  $\tau_y$  in eqn. 3.6 to match effective tax rates by income level. Table 4 and the bottom panel of Table 5 lists calibrated values of the tax/transfer rule parameters discussed in this section.

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<sup>34</sup>It is well known that wage data contains measurement error (Bound and Krueger (1991), Bound et al. (2001), Gottschalk (2005)). Therefore, we add noise to simulated wage data before constructing moments. The variance  $\sigma_N^2(educ)$  of this measurement error term clearly has an effect on the overall variance of log wages, but it has no effect on the rates of transition between employment and non-employment. This enables us to identify the amount of actual wage risk without confounding it with measurement error in wages.

<sup>35</sup>Assuming all DI recipients are in poor functional health, we can back out the percent of working age men who receive DI conditional on poor  $H$ .

## 6 Model Fit

A novel feature of our model is that workers receive tied wage/hours/insurance offers. Table 6 reports on the model fit to the proportions of workers employed full and part-time with and without employer sponsored health insurance. The model captures well the pattern that more educated workers are more likely to receive full-time offers that include health insurance. For example, at ages 35-44, the model predicts that 82.4% of college types have full-time jobs with insurance, compared to only 56.8% of high school types, and these fractions align well with the data frequencies (82.3% and 59.7%).

Table 6 also shows that the model captures well the rapid declines in employment as workers approach age 65.<sup>36</sup> Less educated workers tend to stop working sooner, both in the model and the data. An important consequence is that only one-third of high school types have full-time jobs with insurance at ages 55-64.

Our model fits patterns of full and part-time employment by age and education very well, as we see in Figure 4. An exception is that part-time employment rises a bit as workers approach age 65, but the model does not generate this.

Figure 5 shows life-cycle paths of full-time employment, conditional on education and health. Clearly, both higher education and better health generate more full-time employment, and our model captures these features of the data well. The low full-time employment rate of workers in poor health is striking. It hovers around 40% regardless of age/education. As we saw in Table 5, tastes for work only differ modestly by health in our calibration, and offer probabilities do not depend on health. So our model implies the low employment rate of workers in poor health is mostly due to low wage offers. This interacts in an important way with the consumption floor and disability insurance, as we will show in Section 7.<sup>37</sup>

Next, in Table 8, we show the model's fit to many of the key data moments that we listed in Table 5. The model gives a very good fit to asset/income ratios, which are higher for the college types. The second panel of Table 8 shows how the model fits full and part-time employment rates, conditional on education and health. The fit is generally very good. We are less accurate in the case of men in poor health with no health shocks, but the data moments are very noisy in those cells.

The third panel of Table 8 shows our fit to targeted moments of involving mean full-time wages, conditional on education, health and age. Again the fit is quite good. Figure 6 reports on how we fit the age profiles of wages more generally. It is evident that poor health shifts wage profiles downward, and the model captures this well. The model also captures the facts that wages start higher and grow faster over the life-cycle for more educated workers. The one area where the model fails is that it systematically overestimates wages at ages 55-64.

The fourth panel of Table 8 focuses on moments involving wage variability. The model matches moments of the stochastic process for residual wages fairly well, except that, for high school types, it understates the variance of the permanent error component and exaggerates that of the transitory component. Table 9 reports on how we fit quantiles of the distribution

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<sup>36</sup>Recall that, starting at age 54, we assume a positive probability of receiving no job offer. This captures a number of reasons firms may be reluctant to hire older workers. For example, a match with an older worker is less valuable as it is likely to last for a shorter period of time. There may also be age discrimination.

<sup>37</sup>As we see in Table 5, our calibration implies full-time work reduces leisure by about 52 to 55% for those in good or fair health. This only increases substantially with poor health for the some college type.

of wages, conditional on age and education. The model’s fit to the quantiles of the wage distribution is very impressive, except at the 99th percentile for college types.

Regarding transition rates between employment states, the model slightly understates the transition rate from employment to unemployment, while slightly exaggerating the transition rate from unemployment to employment (for non-college types). In reality, unemployed workers without a college degree may not always have job offers, a possibility our model does not capture at ages younger than 54.

The bottom panel of Table 8 shows our fit to moments that involve the consumption floor and disability benefits. We slightly under-predict the (very high) fraction of men in poor health who receive disability benefits. For instance, for high school types, this fraction is 80% in the data vs. 74% in the model. We capture fairly accurately the (much smaller) fraction of working age men who receive non-DI transfers, which is about 4% to 9% depending on education. In our model this means these men are at the consumption floor.

Figure 8 describes the distribution of medical spending in our model vs. the MEPS data. The model does a reasonably good job matching the extreme skewness of the expenditure distribution (i.e., the top 1% of spenders account for 25% of total costs).

Finally, Figure 7 shows how our model fits the Gini coefficient for income by age, where income is defined as labor earnings plus asset income. This is an untargeted moment in estimation, yet we fit it quite well. This is critically important, as much of the next section focuses on how health shocks (and other factors) contribute to income inequality.

## 7 Results

### 7.1 Effects of Health Shocks on Key Outcomes

We begin by examining the impact of health shocks ( $s$ ,  $d^u$ ,  $d^p$ ) on some key outcomes in our baseline model. To this end, we compare simulated life-cycle histories from the baseline model with alternative simulations in which agents are “lucky” and do not experience health shocks. We hold the perceived risk of health shocks unchanged.

In these experiments, agents’ decision rules are unchanged, and they still behave *as if* they expect to draw health shocks from the distributions  $\Gamma^{dp}(R, H, t, educ)$ ,  $\Gamma^{du}(t)$ , and/or  $\Gamma^s(t)$ . This allows us to examine what we call “direct” effects of health shocks. Later, in Section 7.3, we run counterfactuals where we shut down health risk, and let agents’ decision rules adapt. That will allow us to also study “behavioral” responses to health risk.

To proceed, we run several experiments in which agents never receive  $s$ ,  $d^u$  or  $d^p$  shocks.<sup>38</sup> Table 10 presents results for working age individuals (age 25-64), emphasizing effects on medical costs, health, labor supply, wages and transfers. First consider the effect of eliminating all three types of observed health shocks ( $s$ ,  $d^u$ ,  $d^p$ ). Our model predicts this would reduce average annual medical expenditures from \$4465 to \$1041. Note that even people with no health shocks have some medical expenses, due to minor illnesses that we do not classify as shocks, preventive care, etc. According to our model, elimination of all health shocks

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<sup>38</sup>As we discussed in Section 5.1.1, our logit model for health transitions implies the existence of “idiosyncratic” health shocks that cause  $H$  and  $R$  to change from one year to the next for reasons not attributable to the observed health shocks ( $s$ ,  $d^u$ ,  $d^p$ ) or other state variables. Here we focus entirely on the effects of the observed health shocks ( $s$ ,  $d^u$ ,  $d^p$ ) that we can identify and categorize.

at ages 25-64 would raise the probability of survival to age 65 from 85% to 92%, increase lifetime labor supply from 29.8 years to 32.1 years, increase the mean hourly wage offer from \$22.88 to \$23.25, and reduce the fraction of men who receive government transfers (including disability) from 12.9% to 8.9%. (Appendix A presents these results by education.)

It is also interesting to compare the impact of different types of health shocks. Our model implies that among working age men, unpredictable shocks ( $s$ ,  $d^u$ ) have larger effects than predictable shocks ( $d^p$ ). Together, eliminating  $s$  and  $d^u$  shocks reduces medical expenditures by 65% and sick days by 86%. Eliminating the predictable shocks ( $d^p$ ) reduces them by only 14% and 25%, respectively. This is not because unpredictable shocks are more severe, but because they are much more prevalent.<sup>39</sup> As we see in Table 10, life expectancy, labor supply and wage offers all increase more in the absence of unpredictable shocks.

We also assess the importance of asymptomatic health risk  $R$ . Specifically, we run an experiment where we give all agents a low risk level initially (at age 25), and shut down transitions to higher levels of  $R$ . Agents' decision rules are again held fixed. Thus, all changes in outcomes arise solely due to "luck" rather than changes in decision rules.

We find that giving all individuals low health risk has fairly small effects. The probability of having a  $d^p$  shock falls by 41%. However, as only about 11% of working age men experience  $d^p$  shocks, the overall benefit of reducing  $R$  is modest. In the bottom row of Table 10, we see that average medical expenditures decrease by only 5.6% and the fraction of those relying on social insurance decreases by only 5.8%. The fraction of men in good functional health would increase by only 1.2 percentage points. In general, most health shocks that occur at working ages are unrelated to  $R$ , so reducing health risk has fairly small effects.

These findings suggest a limited potential impact of policies aimed at reducing risk factors like high blood pressure, cholesterol and obesity, as they are not likely to have large effects on health or labor market outcomes for the working age population. Of course, the potential benefits of reducing health risk are greater at ages over 65, when predictable shocks such as heart attack become more prevalent.

## 7.2 Decomposing Sources of Earnings Inequality

Next we use our model to estimate the fraction of variance (across people) in the present value of lifetime earnings (PVE) that is explained by initial conditions and health shocks. We generate simulated life-cycle histories from the benchmark model, and calculate the PVE discounted to age 25 for each simulated agent. Then, similar to Keane and Wolpin (1997), we run regressions of the PVEs on initial conditions (i.e., education, skill type, initial health). But we also include measures of health shocks that occur at ages 25-64.

Table 11 presents the  $R^2$  values from alternative specifications of these regressions, both run separately by education and for all groups combined. First we focus on the combined results. Similar to results in Keane and Wolpin (1997), we find that a substantial 86.8% of the variance in the PVE across agents can be explained by initial conditions at age 25, primarily education and a fixed productivity type. There is only a small contribution of initial health  $H$  and the initial risk level  $R$ , which vary little across people.

Next, we add a set of variables designed to capture flexibly the impact of health shocks

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<sup>39</sup>The most prevalent shocks are transitory  $s$  shocks (39% of working age individuals experience these each year), followed by  $d^u$  (21%) and lastly by  $d^p$  (13% for HS, 12% for Some College, and 8% for College).



throughout working life. We include the number of times the agent experienced each of the eight possible combinations of the three health shocks ( $s$ ,  $d^u$ ,  $d^p$ ). We enter these as separate variables to allow the health shocks to have different effects when they occur in combination. We also enter as separate variables the counts of health shocks that occurred when the agent was in poor, fair, or good health. This captures the fact that health shocks may have a larger effect if the person was in worse health to begin with. We also include the number of years the person spent in good, fair or poor health, primarily to pick up the effects of the “idiosyncratic” health shocks (i.e., the logit errors in the health transitions). Finally, to control for mortality shocks, we include the number of years prior to age 65 when the individual died, if positive. We were not able to find additional health variables that significantly improved the fit of our PVE regression.

When we include this array of health shock measures, the  $R^2$  of our PVE regression increases to 92.4%. Thus, initial conditions (at age 25) and health shocks together can “explain” (or predict) 92.4% of the variance of lifetime earnings. The independent contribution of health shocks to explaining the variance of the PVE, beyond what can be predicted based solely on initial conditions, is 5.6%.<sup>40, 41</sup>

Finally, Table 11 row three presents regressions that only control for initial health and the array of health shock variables, while omitting education and the skill endowment. Here, we find that initial health and health shocks explain 40.0% of the variance in the PVE across all agents. Almost all of this is due to the health shock variables because, as we noted earlier, initial health at age 25 does not vary much across people.

Combining these results, we see that initial conditions independently explain 52.5% of the variance of the PVE, while health shocks independently explain 5.6%. A substantial 34.4% of the variance is “explained” by the covariance between initial conditions and health shocks. The covariance term is so large because of the strong negative correlation between education/productivity and the incidence of health shocks.<sup>42</sup>

There are three basic explanations for this correlation: First, causality may run from education to health, perhaps because more educated people have a better understanding of health risks and good nutrition, are better at utilizing health improving technologies/treatments, and so on. Second, there may be some omitted factor that causes people to get more education and to take better care of their health. This might be a personality trait like “good judgment” or “self control.” Third, it is possible that fore-knowledge of one’s health transition function impacts one’s human capital investment decisions. Thus, we cannot rule out that causality runs from health outcomes to education, despite the fact the education decision is temporally prior to those outcomes.

This discussion highlights the limitation of using a regression decomposition of variance to assess the importance of health shocks for earnings inequality. What we can say is that,

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<sup>40</sup>We run similar regressions for the present values of utility and consumption. We find that initial conditions explain 82% of the variance of the present value utility and 87% of the variance of the present value of consumption. Both these figures increase to 93% when health shocks are included.

<sup>41</sup>If we look within education types, the results are very similar, except that health shocks are somewhat more important, particularly for the less educated. Within education types, the initial conditions (primarily the latent skill endowment) explain 79% to 86% of the variance of the PVE. The incremental contribution of health shocks ranges from 7.0% for the some college type to 10.1% for the high school type.

<sup>42</sup>The source of the positive correlation between education and health, often called the “SES gradient,” is of course one of the great open questions in the social sciences. See [Smith \(2004\)](#) for a discussion.

for all workers, health shocks “explain” roughly 40.0% of the variance of the PVE, but 34.4% of that variation is predictable based on one’s initial education and skill type. Thus, it is not clear how much of that 34.4% is actually caused by health shocks, and, indeed, our prior is that most of it reflects causality running from education to health, or from some omitted third factor to both education and health. What is clear, however, is that 5.6% of the variance of lifetime earnings is directly attributable to “luck” whereby agents with the *same initial conditions* experience different incidence of health shocks.

### 7.3 The Role of Health Shocks in Generating Earnings Inequality

Next, we use our model to conduct counterfactual experiments that clarify how health shocks contribute to earnings inequality. Specifically, we eliminate health shocks from the baseline model and simulate life-cycle histories for agents in the new environment. Shutting down health shocks affects earnings inequality for three reasons: (1) it eliminates the “luck of the draw” whereby agents with the same initial conditions (education/productivity/initial health) experience different health shock realizations, (2) it eliminates the advantage of better-educated workers that arises because they face more favorable probability distributions of predictable health shocks, and (3) it induces a behavioral response as agents update their decision rules in response to the new health risk environment.<sup>43</sup> We call effects that arise given fixed decision rules the “direct” effects of eliminating health shocks, and effects that arise from changing decision rules the “behavioral” response to reduced health risk.

An advantage of the counterfactual simulation approach is that we can run simulations where we hold decision rules fixed (i.e., the same as the baseline model), just as we did in Section 7.1. Comparing the results of such simulations with ones that also allow decision rules to adapt enables us to isolate both the direct effect of health shocks and the behavioral response to reducing health risk.

To proceed, Table 12 reports both means and measures of dispersion for the present value of lifetime earnings (PVE), both in the baseline model and in counterfactuals where we eliminate health shocks for working-age men.<sup>44</sup> In the baseline, the mean PVE is \$762k, with a standard deviation of \$422k, implying a coefficient of variation of 0.555. The great heterogeneity of the PVE across education/productivity types, already apparent from the regressions of Section 7.2, is clearly evident. The mean PVE ranges from only \$294k for low-skill high school types to \$1,522k for high-skill college types.

The middle columns of Table 12 show how the distribution of the PVE is altered when we eliminate health shocks for working-age men, while holding their decision rules fixed. The mean PVE increases by 5.6% to \$805k. The coefficient of variation (CV) of the PVE decreases 4.9% from 0.555 in the baseline to 0.528 in the experiment. And the Gini inequality measure also decreases 4.9% from 0.304 to 0.289.

The right columns of Table 12 show how the distribution of the PVE is altered when we also allow agents’ decision rules to adapt to the lower health risk environment. Compared to

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<sup>43</sup>These counterfactuals differ in important ways from the regression decompositions of variance reported in Section 7.2. The regressions do not capture channel (3), the behavioral response to reduced risk. They only capture the impact of different incidence of health shocks (due to “luck”) in a fixed risk environment.

<sup>44</sup>Eliminated health shocks for men aged 65+ leads to an increase in average lifespans of 10 years, drastically changing the savings needs for retirement, and affecting savings and labor supply decisions. On the other hand, eliminating shocks only at working ages leads to an increase in average lifespans of only 1.5 years.

the baseline, the mean PVE increases by 9.3% to \$833k. The coefficient of variation (CV) of the PVE decreases by 13.7% from 0.555 in the baseline to 0.479 in the experiment, and the Gini inequality measure decreases by 15.1% from 0.304 to 0.258.

Thus, health shocks generate about 15% of inequality in present value of lifetime earnings for men. Notably, direct effects of health shocks on health/productivity account for only about 1/3 of their impact on inequality, while behavioral responses account for 2/3.

The reason behavioral responses to health risk contribute substantially to inequality becomes apparent if we examine how mean PVE changes for different education and productivity types when health shocks are eliminated. We report this in the bottom panel of Table 12. For the low-skill high school type the direct effect of eliminating health shocks is to increase mean PVE by 12.9% (from \$294k to \$331k). But when we factor in their behavioral response, mean PVE increases by 37.5% (to \$404k).

The large behavioral effect of health risk on earnings arises because, in the baseline model, low-skill high school types have a strong incentive to hold down their labor supply and human capital accumulation so as to maintain eligibility for social insurance that cushions against high medical costs. In fact, as we see in Table 14, eliminating health shocks increases the employment rate for low-skill high school types from 57.1% to 84.3%, and reduces the fraction who receive social transfers from 42% to 9%. As we report in Appendix Table A4, only ten points of that decline is due to health shocks *per se*, while 24 points is due to the behavioral response. Thus, in an environment with costly health shocks, social insurance creates a type of “moral hazard” that reduces labor supply and human capital investment (analogous to how health insurance generates moral hazard by reducing the incentive to invest in health).

Next, consider the effects of health shocks on the medium and high productivity types within the high school group. For them, the direct effects of eliminating health shocks are to increase mean PVE by 7.1% and 5.5% respectively, but the additional behavioral effects are trivial. Thus, among the medium and high skill types, social insurance has no significant moral hazard effect on labor supply and human capital investment.

The same pattern holds within the some college and college groups: For low skill types there is a large behavioral effect of health shocks on mean PVE, while for high skill types the behavioral effects are very small. In fact, within all three education groups, the behavioral effect of eliminating health shocks is to slightly *reduce* PVE for the high skill type. These agents are unlikely to use transfers to help pay medical costs, and they instead self-insure. Removing health shock risk reduces the need for precautionary savings, slightly reducing the incentive to supply labor. For instance, in Table 14 we see the employment rate of high skill college types declines slightly from 93.7% to 92.6% when health shocks are eliminated.

### 7.3.1 The Role of Medical Cost Shocks

Next, we consider simulations where, instead of eliminating health shocks, we eliminate only the medical expenses created by those shocks.<sup>45</sup> This allows us to disentangle effects of health shocks operating through their impact on health and productivity vs. effects operating through their impact on the lifetime budget constraint.

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<sup>45</sup>This is a partial equilibrium experiment where we insure all health care costs, but we do not finance the program by raising taxes. It is only meant to clarify how health care costs affect behavior. Later, in Section 7.6 we consider experiments where we introduce health insurance financed by premiums and/or taxes.

Table 13 reports our results. The middle columns show how the distribution of the PVE is altered when we eliminate the medical costs of health shocks for working-age men, while holding their decision rules fixed. Notice that the effects on both mean PVE and measures of inequality are trivial, and this is true for all education/productivity types.

The right columns of Table 13 show how the distribution of the PVE is altered when we also allow agents' decision rules to adapt to the lower medical cost risk environment. Compared to the baseline, across all agents, the mean PVE increases by 2.5% to \$781k, and the Gini measure of inequality drops by 8.6% to 0.278. This masks substantially heterogeneity across types: The mean PVEs of low-skill types within the high school, some college and college types increase by 16.3%, 8.9% and 9.2% respectively. And inequality measures drop by about 1/4 to 1/3 within the low productivity types. In contrast, the behavioral responses among high-productivity types are trivial within all education groups.

These results highlight the strong impact of health care costs on the behavior of the low productivity types. According to our model, they have strong incentives to reduce labor supply and invest less in human capital so as to maintain eligibility for social insurance that protects them from high medical costs. In fact, as we see in Table 14, the employment rate of the low-skill high school type increases from 57.1% to 71.7% when the cost of health shocks is eliminated. The increases in employment for the low productivity types within the college and some college groups are substantial as well. Reliance on social insurance declines dramatically for almost all groups, but the largest absolute decline is observed for the low productivity high school type, for whom receipt of transfers drop from 42% to 22%.

### 7.3.2 Effects of Health Shocks on Income Inequality over the Life Cycle

Next we examine how income inequality varies over the life-cycle. Figure 9 plots the Gini coefficient for cross sections of agents at each age from 25 to 64. Recall from Figure 7 that our model fits the life cycle pattern of income inequality very well. In both the model and the data, cross-sectional income inequality increases as people age. The increase is very gradual in the 40s, but accelerates for agents in their 50s and 60s. Much of the increase at later ages is driven by retirement behavior, but much is also due to health shocks.<sup>46</sup>

Consider the experiment where we eliminate health shocks, and allow agents to update decision rules. As we see in Figure 9, this causes income inequality to drop at all ages, but the drop is much greater for workers in their 50s and 60s. For example, at age 55 the Gini drops substantially by .11 points (from .46 to .35), while at age 40 it only drops by .03 (from .34 to .31). Half the drop (even more at younger ages) arises from the behavioral effect.

It is interesting to contrast these figures with the drop of .046 (from .304 to .258) that we saw in Table 12 for the present value of lifetime earnings evaluated at age 25. The drop in PVE is relatively modest because later ages, where health shocks are more influential, are discounted in the present value calculation. There is no inconsistency in finding that health shocks can explain about a quarter of income inequality for people in their 50s and 60s and our earlier finding that health shocks only explain about 15% of PVE inequality at age 25.

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<sup>46</sup>The model generates a jump in income inequality at age 60 because the probability of receiving no job offer jumps at 60. Figure 4 shows how the model also generates a drop in full-time employment at 60.

## 7.4 Direct and Behavioral Effects of Health Shocks

In the previous section we explored how health shocks contribute to earnings inequality. In this section we explore how health shocks affect a range of behaviors and outcomes including health itself, work experience, wage offers, and reliance on social transfers and disability benefits. This clarifies the channels through which health shocks affect earnings.

To disentangle direct and behavioral effects of health shocks, we compare results from three experiments: (1) eliminate health shocks but hold labor supply and savings fixed, (2) eliminate health shocks but hold decision rules fixed (allowing labor supply and savings to change according to the optimal policy functions of the benchmark environment), and (3) eliminate health shocks and allow agents to update their optimal decision rules. As we explained in Section 7.3, the first two simulations capture the direct effects of health shocks, while the latter experiment incorporates the behavioral response to reduced health risk.<sup>47</sup>

### 7.4.1 Effects of Health Shocks on Health

Figure 10 shows how the evolution of health itself ( $H$ ) is altered in counterfactuals where we shut down health shocks for working age men. The figure reports the fraction of men in fair and poor health in the baseline model and in the three counterfactual simulations described above. We label these "No Shocks 1," "No Shocks 2," and "No Shocks 3."

Not surprisingly, the direct effect of eliminating health shocks (without any decisions changing) is to improve health substantially (as health shocks are key drivers of  $H$  and  $R$  transitions). For example, the fraction of men in poor or fair health at age 64 drops from .56 in the baseline to only .42 in the absence of health shocks. This improvement in health leads to higher wage offers, higher employment and higher incomes. These in turn have an additional positive reinforcement effect on  $H$  as seen in experiments (2) and (3). However, Figure 10 reveals that these effects are relatively minor: the fraction of men in poor or fair health at age 64 drops by only an additional .01 when decision rules remain fixed and an additional .01 when decision rules are allowed to adapt. Thus, the bulk of the inequality in  $H$  generated by health shocks is accounted for by the immediate effect of health shocks on  $H$ , not the reinforcement effect operating through employment and income.

### 7.4.2 Effects on Employment, Human Capital and Wage Offers

Figure 11 plots the mean and coefficient of variation of experience, human capital, and wage offers from the same set of three experiments. In the first experiment experience and human capital remain unchanged from the benchmark, because we hold labor supply fixed. But offer wages can change, because they depend on  $H$ . Thus, the first experiment shows only the direct effect of health shocks on wages through their direct impact on  $H$ .

As we see in the third panel of Figure 11, the direct effect of health shocks on wages (operating through  $H$  itself) is very modest. Only when workers reach their 50s and 60s does it start to become a non-negligible factor. For example, the mean offer wage of 50 year old workers only increases from \$25.0 to \$25.2 per hour if health shocks are eliminated, but that of 60 year old workers increases from \$25.5 to \$25.9.

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<sup>47</sup>Experiments (1) and (2) can be interpreted as a situation where all agents are "lucky" and experience no health shocks, but where the perceived probabilities of health shocks are unchanged (at baseline levels).

In the second experiment we let elimination of health shocks alter labor supply decisions and employment. We still call this a “direct” effect because decision rules are held fixed, but it includes the reinforcement effect that arises because increased employment and income further improve health and increase human capital.<sup>48</sup> In this experiment work hours increase both because sick days are eliminated and because improved health leads to higher wage offers, which increases labor supply. As we saw earlier in Table 10, the elimination of health shocks causes lifetime work experience to increase by 2.3 full-time equivalent years. However, as we now see in the top two panels of Figure 11, impacts on accumulated work experience and human capital are very modest until workers are in their 50s and 60s.

When we account for how eliminating health shocks alters the accumulation of work experience and human capital, the implied effect on offer wages roughly doubles. Now, as we see in the third panel of Figure 11, the mean offer wage of 50 year old workers increases from \$25.0 to \$25.4 per hour, while that of 60 year olds increases from \$25.5 to \$26.3.

Finally, in the third experiment we let agents’ decision rules for labor supply and saving adapt to the reduced risk environment. As we see in Table 15, elimination of health shocks causes lifetime full-time equivalent work to increase by 4.5 years (or 15%). This is almost double the increase of 2.3 years that we found from the direct effects of health shocks (holding decision rules fixed). Once all three channels of effects are factored in, the mean offer wage of 50 year old workers increases from \$25.0 to \$25.9 per hour when health shocks are eliminated, while that of 60 year olds increases from \$25.5 to \$26.8.

It is worth emphasizing that the reduced form studies reviewed in Section 2 do not attempt to capture behavioral effects of health risk on employment and wages. They estimate only what we call the “direct” effects of differential incidence of health shocks (i.e., “luck”) within a given risk environment (with fixed decision rules). But we find that the behavioral effects on employment and wages are as large as the direct effects.

Inequality in work experience drops very sharply when we allow decision rules to adapt to the lower risk environment (see the top right panel of Figure 11). This is primarily because labor supply of low-skill workers increases sharply when health shocks are eliminated, as they no longer have an incentive to constrain their labor supply and human capital accumulation to maintain eligibility for social insurance that protects them from high medical costs. For example, in Table 15, note that lifetime full-time equivalent work increases from 19.9 to 31.1 years for low-skill high school types. As we see in the bottom panel of Figure 11, the drop in inequality in work experience translates into a sharp drop in inequality in wage offers.

Eliminating health shocks only has large *direct* effects on hours at older ages, but it has a substantial positive *behavioral* effect on hours of low-skill workers even at young ages. As a result, the behavioral response generates noticeable increases in mean offer wages, and declines in wage inequality, at much younger ages than implied by direct effects alone.

As a summary of how health shocks affect wage inequality through the three channels, note that the coefficient of variation of wage offers at ages 50 (60) declines by 0.9% (1.7%) in the first experiment due to changes in  $H$ , by an additional 1.6% (2.0%) in the second experiment due to less dispersion in human capital, and by an additional 3.8% (4.3%) in the third experiment due to the behavioral response to reduced health risk. Thus, the behavioral

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<sup>48</sup>In contrast to our model De Nardi et al. (2017) introduce heterogeneity by having different health types within each education group. Then, the poor health types tend to spend longer periods in poor health states and non-employment, but there is no feedback effect of employment or income on health.

response to health risk accounts for the bulk of the effect of health shocks on wage inequality.

Next, we examine how health shocks affect wage offers for different education groups. Figure 12 plots age profiles of the mean and coefficient of variation of offer wages, separately by education. The mean offer wage at age 25 is normalized to 1.0, so one can read wage growth off the graphs. In the benchmark, wage growth from age 25 to 55 is 27% for the high school and some college types, and 74% for the college type. When we shut down health shocks, and allow decision rules to adapt, wage growth increases to 35% for the high school type, 34% for some college types, and 81% for college types.

In the baseline model, the coefficient of variation (CV) of offer wages grows substantially from age 25 to 55, from .37 to .45 for the high school type, .40 to .48 for the some college type, and .42 to .58 for the college type. Thus, for the more educated, the CV starts higher and grows more with age. When we shut down health shocks and allow decision rules to adapt, the growth of the CV declines by 2/3 within the high school and some college types. But for the college type the figure is only 23%. Thus, health shocks account for only a modest fraction of the growth in offer wage inequality over the life-cycle for college workers, but for a very large share within the high school and some college groups. And, as we see in Figure 12, most of that large share is due to the behavioral response to health risk.

Table 15 reports how eliminating health shocks at ages 25-64 affects mean offer wages across all ages. Averaged over all types, the mean offer wage increases from \$22.88 in the baseline to \$23.56 in the counterfactual, which is only 3%. However, Table 15 also reveals that the growth in mean offer wages is very concentrated among the low-skill types. Within the high school, some college and college types the mean offer wage of the low-skill types grows by 11.1%, 7.5% and 7.1%, respectively. The growth for higher skill types is much smaller. This largely reflects the increased labor supply and human capital accumulation of low skill workers in the absence of health shocks.

Finally, the results in Table 15 show that eliminating health shocks at ages 25-64 causes mean lifetime work hours to increase by 15.1% while the mean offer wage increases by only 3%. Thus, health shocks reduce work hours far more than they reduce offer wages. We saw in Table 12 that elimination of health shocks increases PVE by 9.3%, which is much less than the roughly 18% increase in undiscounted earnings. This is because most of the increases in hours and wages are concentrated at older ages.

### 7.4.3 Health Shocks and Social Insurance

As we saw in Table 8, our baseline model accurately predicts the fraction of working age men who receive social transfers or disability benefits. In Table 14, our model predicts the elimination of health shocks would cause the fraction who receive social transfers to drop from 12.9% to 2.0%. This is a much larger than the drop to 8.9% that we saw in Table 10 in the exercise where we held decision rules fixed. Thus, roughly 2/3 of the drop in social transfer receipt is due to the behavioral response to reduced health risk.

The behavioral response is a substantial increase in labor supply, concentrated among low-skill workers. In Table 10 we saw the direct effect of eliminating health shocks is to increase lifetime work from 29.8 full-time equivalent years to 32.1 years, while in Table 15 we see that the behavioral effect leads to an additional increase to 34.3 years. Among low-skill high school types the increase in work is 19.9 to 31.1 years, the drop in all social transfer receipt is 41.6% to 8.6%, and the drop in disability receipt is 8.4% to 1.5%.

The behavioral response of reduced health risk generating increased labor supply arises because, once health risk is reduced, agents have less incentive to constrain labor supply so as to maintain eligibility for social insurance. Similarly, [Pashchenko and Porapakarm \(2016\)](#) find that means-tested Medicaid discourages labor supply, as individuals would rather exit the labor force and receive Medicaid than work and pay medical costs out-of-pocket.<sup>49</sup>

New from prior literature, in our model health risk and social insurance also interact to reduce incentives for human capital investment. Agents anticipate that future health shocks and the possibility of qualifying for means tested transfers will reduce future employment. As human capital generates zero returns in periods of non-employment, this reduces the incentive for human capital investment today, further reducing current labor supply.

## 7.5 The Effect of Health Risk on Earnings Inequality

Next we ask how heterogeneity in health *risk* contributes to earnings inequality. In our model, the probability distribution of predictable health shocks ( $d_p$ ) and the laws of motion for health ( $H$ ) and risk factors ( $R$ ) differ by education. Less educated workers face a higher probability of predictable health shocks, and they face higher probabilities of transition to inferior health states (see Figures 1 to 3).<sup>50</sup> To what extent do these differences in health risk by education generate differences in labor supply, human capital investment and earnings?

To address this question, we conduct counterfactuals where we equalize health risk across different types of agents. Specifically, we give all agents, regardless of education, the health transition functions and health shock distribution of the some college type.<sup>51</sup> The results are reported in Table 16. Our key finding is that the mean present value of earnings (PVE) of high school types only increases by 2.8% in this experiment. In contrast, in Table 12, when we eliminated health shocks entirely, the PVE of high school types increased by 11.8%. Furthermore, because the earnings of high school types increases so modestly, the overall Gini coefficient actually increases from .304 to .319 when we equalize health risk.<sup>52</sup>

Do these results imply that heterogeneity (by education) in the risk of health shocks is not an important source of earnings inequality? That would be an incorrect interpretation.

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<sup>49</sup>[Hubbard et al. \(1995\)](#) quantify the degree to which social insurance, that cushions against idiosyncratic *income* shocks, discourages labor supply and saving for self-insurance purposes. Our results extend theirs by quantifying how means-tested health insurance programs (like Medicaid) discourage labor supply in an environment with health shocks that generate health care costs.

<sup>50</sup>It is well known that education is positively associated with health (e.g., [Grossman and Kaestner \(1997\)](#), [Grossman \(2000\)](#), [Smith \(2004, 2007\)](#), [Cutler and Lleras-Muney \(2008, 2010\)](#)). Smith calls the reason for the “education/health gradient” one of the greatest open questions in the social sciences. We discuss possible reasons for the correlation in Section 7.2, but it is beyond the scope of our analysis to explain what ultimately drives the positive education/health association. Instead, we focus on using our model to examine how differences in health risk by education level - taken as given - affect earnings inequality, labor supply and other behaviors.

<sup>51</sup>We do this because the level of health risk faced by the some college type is intermediate between that faced by the high school and college types. Alternatively, we can also re-estimate the  $H$ ,  $R$  and  $d^p$  functions with education omitted, and simulate the behavior of all agents when they face these common equations describing health risk. That approach generates very similar results.

<sup>52</sup>What drives the increase in the Gini is that low-skill college types work quite a bit less when their health risk is increased to the level of the some college type. With lower level they stay eligible for transfers in the event of expensive health shocks. Thus, inequality increases substantially within the college type.



Rather, what drives these results is that, even in the lower risk environment, high school types still have a strong incentive to constrain their labor supply so as to maintain eligibility for transfers that protect them from high medical costs. Reducing the health risk they face to the some college level does little to change that fact. In contrast, the larger risk reduction generated by removing health shocks entirely does change their incentives fundamentally.

## 7.6 Providing Public Health Insurance to the Uninsured

Finally, we use our model to simulate the impact of providing government funded health insurance to uninsured workers. In the baseline environment 35% of working age men lack employer provided health insurance (ESHI), and 12.9% resort to social insurance (including disability benefits) to pay health care costs. As we saw in Table 7, rates of coverage by ESHI vary greatly by age, education and full or part-time employment status, and our model provides a good fit to these patterns.<sup>53</sup> The fractions of high school, some college and college types with ESHI are 54%, 67% and 77%, respectively.

In our counterfactual experiments we leave ESHI as in the benchmark, but assume that all uninsured individuals participate in a mandatory government funded health insurance program.<sup>54</sup> Participants in the public plan pay an annual premium equal to the employee's share of the ESHI premium in the benchmark (\$652/year), and this is tax deductible. They face a co-insurance rate of 30%, which is comparable to the typical ESHI plan.

Tables 17 and 18 present the results. The mandatory public insurance program spends on average \$4,031/year per privately uninsured individual. A large fraction of this is accounted for by expenditures on those who were previously covered by Medicaid/DI. The average Medicaid/DI expenditures per uninsured person decline from \$2,886 in the baseline to \$599. The average out-of-pocket medical expenses of the uninsured decline from \$2,858 to \$1,130.

The employment rate increases from 83.1% to 85.3% when the public plan is introduced. Not surprisingly, this is primarily driven by an increase in the fraction of job offers without ESHI that are accepted, from 84.8% in the baseline to 89.8%. Lifetime labor supply increases from a mean of 29.8 years in the baseline to 30.6 years in the experiment (a 2.7% increase). Because labor supply increases, human capital accumulation, wage offers and lifetime earnings increase as well. The mean offer wage increases from \$22.55 per hour to \$22.68 per hour, and the present value of lifetime earnings increases from \$762k to \$772k, a 1.3% increase. Increases in labor supply and earnings are greater among the low-skill types.

In addition to increasing labor supply, introduction of public insurance also reduces the fraction of working age men who rely on social insurance (including Medicaid and disability), from 12.9% in the benchmark to 8.8% in the experiment. As we see in Table 17 government expenditures on social insurance decline by \$762 per capita (36%) from the benchmark, a substantial cost savings.

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<sup>53</sup>A limitation of our model is we assume all unemployed workers lack ESHI. In reality, 10% (17%) of unemployed men aged 26-44 (45-64) were covered by their previous employer's plan in 2010 (Janicki (2013)). Accounting for this would significantly complicate the model, as we would need to add a state variable for whether an agent had access to health insurance through a previous employer.

<sup>54</sup>If we were to introduce a universal health insurance plan that *replaced* employer provided health insurance we would need to account for how wage/job offer distributions and government revenues change when firms no longer receive tax benefits for providing ESHI. But this is beyond the capacity of our partial equilibrium model. For this reason, we only present results from experiments where ESHI remains unchanged.

Together, the declines in social insurance payments slightly outweigh the \$689 per capita cost of the new public insurance plan. After taking into account all changes in expenditures on other programs, as well as the increase in government revenue stemming from the increase in labor supply, we find that the introduction of the public insurance plan actually saves the government \$64 per capita.<sup>55</sup>

There are two important caveats to this finding: First, we abstract from moral hazard effects of enhanced insurance coverage on total medical expenditures. Second, provision of public insurance could increase demand for medical services, which could increase their price. Thus, our experiment is likely to understate the cost of providing public insurance.<sup>56</sup> Given these limitations, our aim is simply to quantify the extent to which provision of public health insurance would (i) increase labor supply and tax revenues, and (ii) reduce reliance on Medicaid and other social transfers, reducing government spending. These two channels are important enough to outweigh the direct cost of the program, so that any cost increase arises through the secondary effects of moral hazard and increased prices.

Finally, assuming it would be self-financing, we find that the consumption equivalent variation (CEV) of introducing the public insurance plan is 1.44% of baseline consumption.<sup>57</sup> In Table 18 we see that the low educated groups experience larger welfare gains: 2.0% in CEV for the HS, 1.1% for some college and 0.9% for college graduates.<sup>58</sup>

## 8 Conclusion

In this paper, we provide a detailed study of how health contributes to earnings inequality. We construct a rich life-cycle framework of labor supply and asset accumulation decisions, with two novel features: (1) a detailed health process over the life cycle that includes several dimensions of health: functional health, underlying health risk, and health shocks that are predictable/unpredictable and temporary/persistent, and (2) interactions between health risk and human capital accumulation (learning-by-doing). We show that both of these fea-

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<sup>55</sup>In this calculation, we factor in all expenditures on social insurance, social security, and medical expenses covered by Medicare and the public insurance program, as well as all taxes and premiums collected.

<sup>56</sup>We may also understate the benefit of public insurance if it improves health. We do not know how  $H$  and  $R$  transitions would change for the uninsured who obtain public insurance, so we take a conservative approach and assume they have the same  $H$  and  $R$  transitions as the uninsured in the benchmark economy. If public insurance leads to better health transitions, our experiments will underestimate its value.

<sup>57</sup>The consumption equivalent variation (CEV) is given by:  $CEV = \left[ \frac{V(c^*, l^*) - D(c_0, l_0)}{V(c_0, l_0) - D(c_0, l_0)} \right]^{\frac{1}{\alpha(1-\sigma)}} - 1$ , where  $(c_0, l_0)$  and  $(c^*, l^*)$  are the consumption-labor allocations in the benchmark and in the counterfactual,  $V$  is the expected discounted value at age 25, and  $D(c_0, l_0)$  is the expected discounted sum of death costs at age 25 in the benchmark.  $D$  depends on  $(c, l)$  up to age 65 since these affect employment decisions and income, which in turn affect  $H$  and  $R$  transitions and thus the probability of death. The CEV takes into account changes in welfare arising from different life expectancies in the counterfactuals.

<sup>58</sup>Within education groups, gains are larger for those with high productivity. For example, for those with high school or less, the welfare gains are 1.0%, 2.4%, and 2.7% for the low, medium and high productivity groups. The higher productivity types benefit more because fewer of them are at the consumption floor, and hence fewer of them can rely on government transfers to cover medical costs. Note that the new public insurance plan covers 70% of medical expenditures, which are on average \$4,500/year. Given the premium is \$652/year, those not at the consumption floor get approximately \$2,500 in additional disposable income, which translates into a 2-3% increase in consumption.

tures are important in allowing the model to capture the degree to which and the pathways through which health impacts earning inequality.

We find health shocks explain roughly 15% of the inequality in present value of lifetime earnings (PVE) across all workers. Within education groups, they explain roughly 25% of inequality within high school workers, 21% within some college workers, and 17% within college workers. However, analogous with results in [Keane and Wolpin \(1997\)](#), we find that, even in a life-cycle model extended to include health and health shocks, worker's education and skill endowment still explain over 80% of the heterogeneity in the PVE.

Importantly, health shocks mostly affect earnings of older workers, which are discounted when taking present values at age 25. We find that health shocks can explain about a quarter of income inequality for people in their 50s and 60s.

We decompose the effect of health shocks on earnings into “direct” effects that hold decision rules fixed, and “behavioral” effects that arise because health risk changes decision rules for labor supply and savings. Notably, direct effects of health shocks account for only about 1/3 of their impact on earnings inequality, while behavioral responses account for 2/3.

The large behavioral effect of health risk on earnings inequality arises because low-skill workers have a strong incentive to hold down their labor supply so as to maintain eligibility for social insurance that cushions against high potential medical costs. This reduces their rate of human capital accumulation, leading to slower wage growth over the life-cycle, and lower earnings. In contrast, among high skill workers, social insurance has no significant effect on labor supply. This asymmetry in responses generates the positive behavioral effect of health risk on earnings inequality.

Thus, in an environment with costly health shocks, social insurance creates a type of “moral hazard” that reduces labor supply and human capital investment of low skill workers (analogous to how health insurance may generate moral hazard by reducing the incentives to invest in health). Quantitatively, we find this moral hazard effect on labor supply is substantial. Providing public health insurance to the uninsured counteracts it, leading to both (i) substantial government cost saving on Medicare, disability and other social insurance programs, and (ii) increased tax revenues due to increased labor supply. Hence, our model predicts that a program providing heavily subsidized mandatory public health insurance to the uninsured would be self-financing and welfare improving.

A limitation of our analysis is we ignore any effect of public insurance on (i) health care spending of the newly insured (via the *ex-post* moral hazard effect), or (ii) the price of medical care (via increased demand for services). So we likely understate the cost of the program. Nevertheless, our key point is that any cost evaluation of public health insurance should factor in the positive effect on labor supply and the savings on social transfers.

Previous literature has considered the potential for public health insurance to generate an *ex-ante* moral hazard effect that reduces the incentive to invest in health. In contrast, we find that public health insurance alleviates the moral hazard problem that arises because means-tested social insurance discourages human capital investment in the presence of health risk. Health insurance has very different effects in our framework depending on whether it is means-tested. For example, increasing the income threshold for means-tested Medicaid would worsen the moral hazard effect on human capital accumulation, while provision of public health insurance alleviates it.

# Tables

**Table 1:** Wages, Hours and Earnings Regression Results, MEPS

Dependent Var	Log Wage	Weekly Hours	Annual Earnings
Mean	3.056	35.098	82.888
SD	0.563	19.283	38.564
$s$	0.003 (0.004)	-0.397*** (0.174)	-0.717*** (0.305)
$d^p$	0.003 (0.006)	-1.216*** (0.284)	-2.543*** (0.522)
$d^u$	0.005 (0.005)	-1.375*** (0.227)	-2.123*** (0.405)
Lagged Dep. Var.	0.878*** (0.006)	0.679*** (0.007)	0.734*** (0.006)
Education			
Some College	0.031*** (0.005)	1.040*** (0.211)	2.588*** (0.368)
College	0.085*** (0.006)	2.166*** (0.192)	7.005*** (0.377)
Initial Health			
Fair	0.020 (0.020)	5.151*** (0.405)	8.916*** (0.761)
Good	0.036* (0.020)	6.543*** (0.429)	12.020*** (0.796)
R2	0.836	0.552	0.643
Observations	22,875	37,004	38,065

Standard errors in parentheses

\*  $p < 0.1$ , \*\*  $p < 0.05$ , \*\*\*  $p < 0.01$

Notes:  $s$ ,  $d^p$  and  $d^u$  are health shock indicators defined in the text. All regressions include year dummies, and a cubic in age. The wage regression is estimated using only workers employed in both interviews 1 and 5. The Weekly Hours regression is estimated on all workers, including those with zero hours. For the 79.2% of the sample with positive hours, mean hours are 43.083 with a standard of 10.620. The earnings regression also includes non-employed workers, and incorporates a Box-Cox transform of annual earnings, with  $\lambda = 0.326$ . If we drop controls for health and health shocks, the R-squared of the three regressions decline to 0.836, 0.543 and 0.635, respectively.

**Table 2:** Classifying Medical Conditions

Assignment	Short-Term Productivity	Long-Term Productivity	Predictor	Predictable	Number of ICD codes
$d^p$	YES	YES	YES	YES	27
$d^u$	YES	YES	YES	NO	18
$d^p$	YES	YES	NO	YES	38
$d^u$	YES	YES	NO	NO	272
$s$	YES	NO	YES	YES	3
$s$	YES	NO	YES	NO	8
$s$	YES	NO	NO	YES	6
$s$	YES	NO	NO	NO	298
$s$	Unknown condition or condition details missing				1
$R$	NO	YES	YES	YES	5
$R$	NO	YES	YES	NO	6
$R$	NO	YES	NO	YES	1
$R$	NO	YES	NO	NO	0
$R$	NO	NO	YES	YES	6
$R$	NO	NO	YES	NO	23
Not used	NO	NO	NO	YES	9
Not used	NO	NO	NO	NO	269

**Table 3:** Work Hours Lost Due to Health Shocks (Sick Days)

Health Shocks	HS or Less			Some College and College		
	H=Poor	H=Fair	H=Good	H=Poor	H=Fair	H=Good
$du = 0, dp = 1, s = 0$	0.0	4.0	0.0	0.0	2.6	1.4
$du = 0, dp = 0, s = 1$	0.0	2.5	0.0	1.3	1.5	0.6
$du = 0, dp = 1, s = 1$	4.2	6.0	5.5	7.6	6.1	3.6
$du = 1, dp = 0, s = 0$	5.0	7.5	1.4	1.7	1.6	1.0
$du = 1, dp = 1, s = 0$	8.5	9.6	0.0	8.3	7.3	3.9
$du = 1, dp = 0, s = 1$	7.1	9.1	4.0	7.1	5.8	3.6
$du = 1, dp = 1, s = 1$	6.1	13.4	9.5	19.7	14.3	12.1

Note: We estimate weekly lost work hours using a regression of weekly hours worked on health risk, age,  $age^2$  and all health shock combinations (no health shocks is the base group). Regressions are run separately by functional health and education. Coefficients that are not statistically significant at the 10% level are set to zero.

**Table 4:** Model Parameters

Parameter	Value
<b>Preferences</b>	
CRRA parameter $\alpha$	0.4
Intertemporal substitution parameter $\sigma$	2.0
Cost of death $\zeta$	-30.0
<b>Interest rate</b>	
$r$	0.04
<b>Tax Parameters</b>	
Consumption tax $\tau^c$	5.70%
Social Security tax $\tau^{SS}$	6.20%
Medicare tax $\tau^{Med}$	1.45%
Income threshold $\bar{y}_{ss}$	\$98,000
Tax function parameter $a_0$	0.258
Tax function parameter $a_1$	0.768
<b>Social Security Income</b>	
HS or Less	\$13,655
Some College	\$14,678
College	\$15,883
<b>Health Insurance</b>	
Fraction of $ME$ paid by Medicare $q^{Med}$	50%
Fraction of $ME$ paid by Employer Insurance $q^{EI}$	70%
Medicare premium $p^{Med}$	\$854
Employer Insurance Premium (Employee's Share) $p^{EI}$	\$652

Note: Average Social Security income is calculated from the HRS. The tax function parameters  $a_0$  and  $a_1$  are taken from [Gouveia and Strauss \(1994\)](#). The co-insurance rates  $q^{EI}$  and  $q^{Med}$  are taken from [Attanasio et al. \(2010\)](#). The Medicare premium  $p^{Med}$  is set to the average annual Medicare Part B premium over the sample period, adjusting for the CPI. The average employee's share of the ESHI premium  $p^{EI}$  for a single's plan is calculated using the MEPSnet/IC Trend Query tool available at [https://www.meps.ahrq.gov/mepsweb/data\\_stats/MEPSnetIC.jsp](https://www.meps.ahrq.gov/mepsweb/data_stats/MEPSnetIC.jsp).

Table 5: Parameters Calibrated by Matching Moments

Parameter	Parameter Description	Target	Parameter Values	
			≤HS	<College
$\beta$	Time discount factor	Asset to inc ratio, ages 30-55	0.962	0.968
$\phi(educ, H = P, h^* = h^{FT})$	Leisure cost of FT work - Poor $H$	% FT, no health shocks, 30-55 - Poor $H$	0.525	0.700
$\phi(educ, H = F, h^* = h^{FT})$	- Fair $H$	- Fair $H$	0.525	0.552
$\phi(educ, H = G, h^* = h^{FT})$	- Good $H$	- Good $H$	0.520	0.545
$\phi(:, :, h^{PT})/\phi(:, :, h^{FT})$	Leisure cost of PT work rel. to FT	% PT, by $H$ , relative to FT, 30-55	0.500	0.500
$\Pi(O^*, educ, t)$	Employment offer probabilities	Emp shares by age, by PT/FT and ESHI	See Table 6	
$\delta^O(educ)$ , ages 54-59	Age trend of prob of no emp offer	Emp shares, ages 54-59	0.030	0.014
$\delta^O(educ)$ , ages 60-64	Age trend of prob of no emp offer	Emp shares, ages 60-64	0.053	0.035
$\beta_0(educ)$	Offer wage func - Intercept	Avg FT wages by age group if $H = G$	2.08	2.30
$\beta_1(educ)$	- $HC$ coeff	Avg FT wages by age group if $H = G$	0.0425	0.0425
$\beta_2(educ)$	- $HC^2$ coeff	Avg FT wages by age group if $H = G$	-0.0014	-0.00145
$\beta_3(educ)$	- $HC^3$ coeff	Avg FT wages by age group if $H = G$	0.000016	0.000017
$\beta_4(educ)$	- $I_{H \in \{F, G\}}$ coeff	Avg FT wages, 30-55, penalty if $H = P$	0.423	0.420
$\beta_5(educ)$	- $I_{H=G}$ coeff	Avg FT wages, 30-55, penalty if $H = F$	0.490	0.500
$\beta_6(educ)$	- $I_{h^* = hr^s PT}$ coeff	Average PT wages /FT wages, ages 30-55	0.920	0.930
$\kappa_1(educ)$	Fixed productivity types	Var fixed type, residual wage reg.	+/-0.283; 0	+/-0.311
$\sigma_{\varepsilon_w}^2(educ)$	Variance transitory wage shocks	Var trans shock, residual wage reg.	0.125	0.097
$\sigma_N^2(educ)$	Variance wage noise	Var log wages, FT, ages 30-35	0.360	0.260
$\nu$	Multiplicative shock to $HC$	Assumed	+/-0.3	
$p^1(educ, I_{h>0} = 1)$	Prob of +/-ve $HC$ shock, working	AR(1) process param, residual wage reg.	0.330	0.330
$p^2(educ, I_{h>0} = 0)$	Prob of -ve $HC$ shock, not working	% transition from Non-Emp to Emp, 30-55	0.950	0.950
$\eta(educ, H = P, t < 65)$	Prob of DI benefits if Poor $H$	% of Poor health receiving DI, 25-64	0.950	0.980
$\bar{c}(educ, I^{DI} = 0)$	Consumption floor, non-DI	% non-DI individuals getting TR, 30-55	4,696	5,408
$\bar{c}(educ, I^{DI} = 1)$	Consumption floor, DI recipients	Average DI benefits	10,400	14,040
$a_2$	Tax function parameter	Effective tax rate by inc group	0.08	
$\tau_y$	Tax function parameter	Effective tax rate by inc group	0.0	

Notes: 1. The leisure cost of work is the fraction of total leisure time lost if working. 2. The probability of positive and negative human capital shocks are equal if employed ( $p^2(educ, I_{h>0} = 1) = p^1(educ, I_{h>0} = 1)$ ). If not employed the probability of a good shock is set to zero ( $p^1(educ, I_{h>0} = 0) = 0$ ). 3. The consumption floor is expressed in dollars per year. 4. Within the High School type we allow for 3 latent productivity types (i.e., three levels of  $\kappa$ ), with type proportions 1/3 each. Within the Some College and College types we allow for two latent types, with proportions 1/2 each.

**Table 6:** Calibrated Employment Offer Probabilities, ages 25-53

	<b>HS or Less</b>	<b>Some College</b>	<b>College</b>
PT, no ESHI	0.050	0.031	0.013
PT, ESHI	0.050	0.030	0.032
FT, no ESHI	0.288	0.167	0.100
FT, ESHI	0.613	0.772	0.855

Notes: At ages 54-64, we allow for a positive probability of having no employment offer. The probabilities of PT and FT offers are scaled down appropriately.

**Table 7:** The Distribution of Employment, Model and Data

	<b>&lt;=HS</b>		<b>Some College</b>		<b>College</b>	
	<b>Model</b>	<b>Data</b>	<b>Model</b>	<b>Data</b>	<b>Model</b>	<b>Data</b>
<b>Ages 25-34</b>						
NE	7.5	8.2	8.4	6.0	8.0	4.9
PT, no ESHI	4.6	5.7	2.9	4.2	1.1	2.4
PT, ESHI	4.5	2.8	2.7	3.4	2.6	4.1
FT, no ESHI	26.5	31.1	15.3	17.5	9.1	9.6
FT, ESHI	56.9	52.2	70.7	68.9	79.1	78.9
<b>Ages 35-44</b>						
NE	8.1	10.2	6.3	6.5	3.8	3.5
PT, no ESHI	4.6	4.4	2.9	2.6	1.3	1.3
PT, ESHI	4.5	2.7	2.8	2.9	3.0	2.7
FT, no ESHI	26.0	23.1	15.3	14.0	9.6	10.2
FT, ESHI	56.8	59.7	72.7	74.1	82.4	82.3
<b>Ages 45-54</b>						
NE	16.8	18.0	15.7	12.3	8.0	6.3
PT, no ESHI	4.0	3.8	2.6	2.6	1.2	1.4
PT, ESHI	4.0	3.2	2.4	3.6	2.9	3.1
FT, no ESHI	22.8	17.8	13.3	12.9	9.1	10.2
FT, ESHI	52.4	57.2	66.0	68.6	78.8	78.9
<b>Ages 55-64</b>						
NE	50.0	43.7	41.4	36.3	34.0	26.1
PT, no ESHI	2.5	4.1	1.9	3.1	1.0	2.3
PT, ESHI	2.3	4.9	1.6	6.3	2.1	6.8
FT, no ESHI	12.9	11.4	8.8	10.0	6.1	9.3
FT, ESHI	32.4	35.8	46.3	44.3	56.7	55.5



**Table 8:** Calibration Moments, Model and Data

	HS or Less		Some College		College	
	Model	Data	Model	Data	Model	Data
<b>1. Identifying <math>\beta</math></b>						
Assets/income ratio, ages 30-55	1.22	1.21	1.27	1.32	1.85	1.88
<b>2. Identifying the leisure cost of work</b>						
% emp FT, no shocks, ages 30-50: $H = Poor$	26.11	31.01	17.53	11.11	34.68	57.14
$H = Fair$	78.43	78.00	80.64	82.22	86.54	88.26
$H = Good$	85.97	85.22	90.25	89.21	92.47	91.42
% emp PT, no shocks, ages 30-50: $H = Poor$	3.08	11.63	1.23	0.00	1.50	0.00
$H = Fair$	8.31	6.13	5.14	6.43	3.98	6.33
$H = Good$	9.30	5.13	5.84	4.30	4.22	3.82
<b>3. Identifying deterministic wages</b>						
Wages, FT, $H = Good$ : ages 25-34	16.57	15.30	20.66	19.47	27.60	29.94
ages 45-54	20.53	18.98	25.70	25.44	39.38	37.95
Wages, FT, ages 30-55: $H = Poor$	13.88	15.59	18.92	21.52	25.06	29.32
$H = Fair$	18.18	16.14	22.21	22.32	32.68	32.65
$H = Good$	19.01	17.68	23.71	23.77	35.37	35.81
Wages PT/ Wages FT, ages 30-55	0.93	0.93	0.94	0.94	0.90	0.90
<b>4. Identifying wage risk and wage noise</b>						
Var fixed effect	0.03	0.11	0.06	0.08	0.09	0.08
Var transitory shock	0.20	0.07	0.12	0.07	0.11	0.08
Permanent shock persistence	0.85	0.94	0.86	0.84	0.89	0.93
Var of innovation	0.01	0.02	0.01	0.04	0.01	0.03
Var log wages, FT, ages 30-55	0.26	0.26	0.23	0.24	0.27	0.28
% Emp to Non-Emp trans. rate, ages 30-55, Good H	2.61	3.02	2.29	2.86	1.51	2.29
% Non-Emp to Emp trans. rate, ages 30-55, Good H	63.08	42.51	66.20	46.59	48.57	48.72
<b>5. Identifying Consumption floor and DI (%)</b>						
% non-DI individuals getting transfers, ages 30-55	8.46	9.26	7.22	7.53	5.02	3.68
Average DI benefits	10,268	9,920	14,175	11,941	17,653	16,839
% receiving DI if H=Poor	73.87	80.33	78.41	83.56	55.85	64.30

**Table 9:** Wage Distribution, Model and Data (CPS)

Distribution of Wages Percentiles, Ages 30-35	HS or Less		Some College		College	
	Model	Data	Model	Data	Model	Data
5	6.5	7.1	8.6	9.1	11.3	12.2
25	10.5	11.7	13.3	15.6	17.8	21.0
50	14.8	16.5	18.6	21.1	25.6	29.3
75	20.9	22.6	26.2	28.3	37.2	41.4
90	28.4	29.7	34.9	37.4	50.4	54.8
95	34.0	36.1	40.6	44.3	58.8	66.8
99	46.9	50.8	51.9	65.1	76.7	114.0
<b>Percentiles, Ages 50-55</b>						
5	8.0	7.7	10.9	9.7	14.7	12.0
25	12.8	13.5	16.6	17.1	23.9	23.4
50	18.0	19.3	23.1	24.4	34.9	34.3
75	25.4	26.4	32.3	33.1	50.7	48.9
90	34.6	35.3	42.5	43.5	69.7	68.1
95	41.5	41.7	49.5	52.0	83.3	89.2
99	57.4	59.2	63.9	76.2	120.5	168.1

Notes: Hourly wages expressed in constant 2010 CPI adjusted dollars. The data is from the CPS, screening out workers in the top 1% of the wage distribution, or with wages below \$3.50/hour.

**Table 10:** The Importance of Health Shocks and  $R$  in the Benchmark Model

	$ME$	Sick days	Surv to 65 (%)	Emp (%)	Yrs Worked	SI (%)	Wage Offer
Benchmark	4,465	8.26	85.18	83.13	29.82	12.86	22.88
No $s$ shocks	2,894	4.35	85.74	83.87	30.60	11.42	22.98
No $d^u$ shocks	3,050	3.85	90.09	84.66	31.24	10.48	23.13
No $d^p$ shocks	3,858	6.23	89.64	83.55	30.47	12.08	22.95
No $s$ and $d^u$	1,571	1.15	90.10	85.08	31.74	9.53	23.18
No $s$ , $d^u$ , $d^p$	1,041	0.00	92.38	85.41	32.14	8.92	23.25
Low $R$	4,211	7.47	87.22	83.73	30.25	12.11	22.92

Notes: Data are simulated from the Benchmark model, with the indicated health shocks shut down at ages 25-64, but with decision rules unchanged.  $ME$  is total annual medical expenditure in dollars. Sick days are expressed in number of lost full time work days per year. “Yrs Worked” is lifetime labor supply in full time equivalent years (max = 40 years). Statistics are for ages 25-64 only, and we combine all education groups.

**Table 11:** Explaining the Variance of the Present Value of Lifetime Earnings

Independent Variables Included	$R^2$ from PV Earnings Regressions			
	$\leq$ HS	Some College	College	All
1. Initial conditions*	0.797	0.855	0.787	0.868
2. Health, health shocks + Initial conditions	0.898	0.928	0.866	0.924
3. Health, health shocks only	0.358	0.337	0.230	0.400

Notes: The table reports  $R^2$  from regressions of the present value of lifetime earnings on initial conditions and/or health measures, using simulated data from the benchmark model. Initial conditions are the latent skill type ( $\kappa$ ) and  $H$  and  $R$  at age 25. In the “All” column that combines education groups, we also include education and its interactions with  $\kappa$ ,  $H_{25}$  and  $R_{25}$ . In Rows 2 and 3, “health, health shocks” are  $H$  and  $R$  at ages 25 and 64, age of death if less than 65, ages that  $d^u$  and  $d^p$  shocks first occur, total years the agent was in Poor/Fair/Good health, and the total number of times each possible combination of health shocks occurred between the ages of 24 and 64, entered separately by health status at the time of occurrence.

**Table 12:** Inequality in the Present Value of Earnings, Evaluated at Age 25

	Benchmark			No Health Shocks Decision Rules Fixed			No Health Shocks Decision Rules Change		
	Mean	CV	Gini	Mean	CV	Gini	Mean	CV	Gini
<b>All</b>	762,177	0.555	0.304	+5.56%	0.528	0.289	+9.26%	0.479	0.258
<b>By Education</b>									
$\leq$ High School	523,423	0.376	0.216	+7.41%	0.350	0.200	+11.83%	0.286	0.163
Some College	711,746	0.435	0.245	+5.72%	0.411	0.231	+9.94%	0.350	0.194
College	1,091,345	0.445	0.253	+4.42%	0.425	0.241	+7.41%	0.375	0.210
<b>By Productivity</b>									
$\leq$ High School									
Low Productivity	293,730	0.300	0.170	+12.85%	0.273	0.155	+37.49%	0.169	0.089
Med Productivity	539,185	0.150	0.077	+7.14%	0.130	0.063	+7.43%	0.125	0.060
High Productivity	734,667	0.134	0.065	+5.47%	0.122	0.059	+5.36%	0.124	0.059
<b>Some College</b>									
Low Productivity	425,701	0.256	0.144	+9.18%	0.233	0.130	+23.80%	0.140	0.072
High Productivity	997,662	0.127	0.059	+4.24%	0.114	0.053	+4.04%	0.114	0.053
<b>College</b>									
Low Productivity	661,093	0.312	0.172	+7.09%	0.279	0.149	+17.34%	0.166	0.086
High Productivity	1,521,622	0.158	0.080	+3.26%	0.152	0.077	+3.10%	0.150	0.076

Note: The mean (across simulated agents) of the present value of earnings (PVE) is expressed in 2010 dollars. CV denotes the coefficient of variation.

**Table 13:** Inequality in the Present Value of Earnings, Evaluated at Age 25

	Benchmark			No ME of Health Shocks			No ME of Health Shocks		
	Mean	CV	Gini	Decision Rules Fixed			Decision Rules Change		
	Mean	CV	Gini	Mean	CV	Gini	Mean	CV	Gini
<b>All</b>	762,177	0.555	0.304	+0.23%	0.551	0.302	+2.49%	0.511	0.278
<b>By Education</b>									
<b>≤High School</b>	523,423	0.376	0.216	+0.37%	0.372	0.213	+3.28%	0.329	0.188
<b>Some College</b>	711,746	0.435	0.245	+0.15%	0.434	0.245	+2.73%	0.395	0.221
<b>College</b>	1,091,345	0.445	0.253	+0.18%	0.440	0.250	+1.93%	0.395	0.223
<b>By Productivity</b>									
<b>≤High School</b>									
Low Productivity	293,730	0.300	0.170	+1.08%	0.302	0.172	+16.32%	0.232	0.129
Med Productivity	539,185	0.150	0.077	+0.63%	0.148	0.075	+1.04%	0.141	0.070
High Productivity	734,667	0.134	0.065	-0.12%	0.132	0.064	-0.01%	0.134	0.064
<b>Some College</b>									
Low Productivity	425,701	0.256	0.144	+0.20%	0.260	0.146	+8.87%	0.198	0.107
High Productivity	997,662	0.127	0.059	+0.14%	0.125	0.059	+0.11%	0.123	0.059
<b>College</b>									
Low Productivity	661,093	0.312	0.172	+0.90%	0.303	0.165	+9.21%	0.206	0.108
High Productivity	1,521,622	0.158	0.080	-0.13%	0.158	0.080	-1.24%	0.162	0.082

Notes: The mean (across simulated agents) of the present value of earnings (PVE) is expressed in 2010 dollars. CV denotes the coefficient of variation.

**Table 14:** Counterfactual Experiments: Employment and Social Insurance

	Employment (%)			Social Insurance (%)		
	Bench	No HS	No ME-HS	Bench	No HS	No ME-HS
<b>All</b>	83.1	91.2	87.6	12.9	2.0	5.6
<b>≤High School</b>	80.2	89.6	85.6	15.9	2.9	7.6
<b>Some College</b>	82.7	92.5	87.7	14.1	1.9	6.6
<b>College</b>	86.9	92.2	89.9	8.2	0.9	2.4
<b>≤High School</b>						
Low Productivity	57.1	84.3	71.7	41.6	8.6	22.1
Med Productivity	89.0	91.8	91.1	7.3	0.4	1.6
High Productivity	92.7	92.2	92.7	0.7	0.1	0.2
<b>Some College</b>						
Low Productivity	69.9	89.9	79.9	28.1	3.7	13.2
High Productivity	95.4	95.2	95.3	0.2	0.1	0.1
<b>College</b>						
Low Productivity	80.1	92.0	88.5	16.4	1.8	4.9
High Productivity	93.7	92.5	91.2	0.0	0.0	0.0

Notes: In the “No HS” counterfactual we eliminate health shocks at working ages. In “No ME-HS” we remove (only) the medical expenditures associated with health shocks at working ages. In each counterfactual, agents update their decision rules (for labor supply and saving) to reflect the new environment. The full-time employment rate and the rate of receiving government transfers are both calculated in the cross-section of working age men.

**Table 15:** Counterfactual Experiment: Effects of Eliminating Health Shocks

	FT Yrs Worked		Mean Wage Offers		DI (%)	
	Bench	No HS	Bench	No HS	Bench	No HS
<b>All</b>	29.83	34.33	22.88	23.56	2.46	0.38
<b>≤High School</b>	27.96	33.11	16.07	16.64	3.62	0.61
<b>Some College</b>	29.94	35.01	20.78	21.41	2.79	0.47
<b>College</b>	32.01	35.26	32.03	32.90	0.82	0.05
<b>≤High School</b>						
Low Productivity	19.93	31.13	10.76	11.95	8.40	1.53
Med Productivity	31.04	33.92	15.95	16.31	2.42	0.25
High Productivity	32.38	34.14	21.44	21.71	0.31	0.10
<b>Some College</b>						
Low Productivity	25.32	33.99	13.70	14.73	5.43	0.83
High Productivity	34.56	36.03	27.81	28.09	0.17	0.11
<b>College</b>						
Low Productivity	29.44	35.16	20.53	21.98	1.61	0.08
High Productivity	34.58	35.35	43.47	43.81	0.03	0.02

Notes: In the counterfactual we eliminate health shocks at ages 25-64, and let agents re-optimize their decision rules to the new environment. Full-time equivalent years worked over the life-cycle is an average over all simulated agents. All other statistics are calculated in the cross-section of working age men. Mean offer wages are calculated using only individuals with full-time employment offers.

**Table 16:** Inequality in the Present Value of Earnings, Evaluated at Age 25

	Benchmark			Some College $\Lambda_H, \Lambda_R$ and $\Gamma^{dp}$		
	Mean	CV	Gini	Mean	CV	Gini
<b>All</b>	762,177	0.555	0.304	-3.14%	0.581	0.319
<b>By Education</b>						
<b>High School or Less</b>	523,423	0.376	0.216	+2.81%	0.353	0.203
<b>Some College</b>	711,746	0.435	0.245	+0.00%	0.435	0.245
<b>College</b>	1,091,345	0.445	0.253	-8.23%	0.560	0.319
<b>By Productivity</b>						
<b>≤High School</b>						
Low Productivity	293,730	0.300	0.170	+8.85%	0.275	0.156
Med Productivity	539,185	0.150	0.077	+2.08%	0.145	0.072
High Productivity	734,667	0.134	0.065	+1.00%	0.135	0.065
<b>College</b>						
Low Productivity	661,093	0.312	0.172	-24.34%	0.516	0.297
High Productivity	1,521,622	0.158	0.080	-1.22%	0.161	0.081

Notes: The counterfactual sets the distribution of health shocks, and  $(H, R)$  transition rates, for all education types, to the Some College levels. The mean (across simulated agents) of the present value of earnings (PVE) is expressed in 2010 dollars. CV denotes the coefficient of variation.

**Table 17: Mandatory Public Health Insurance**

	<b>Benchmark</b>	<b>Public HI</b>
Average Medical Expenses, ages 25-64		
- If covered by ESHI	3,775	3,770
- If no ESHI:		
-Out-of-pocket	2,858	1,130
-Public HI	-	4,032
-SI	2,886	599
-Total	5,743	5,761
Wage Offers	22.55	22.68
FT Yrs of Work	29.83	30.61
Mean Government Expenditures per capita		
-Public HI	-	689
-SI	2,098	1,336
Government Deficit per capita	2,694	2,630

Notes: Mean government expenditures per capita are calculated as the total expenditures across all ages, divided by the total number of agents in the economy.

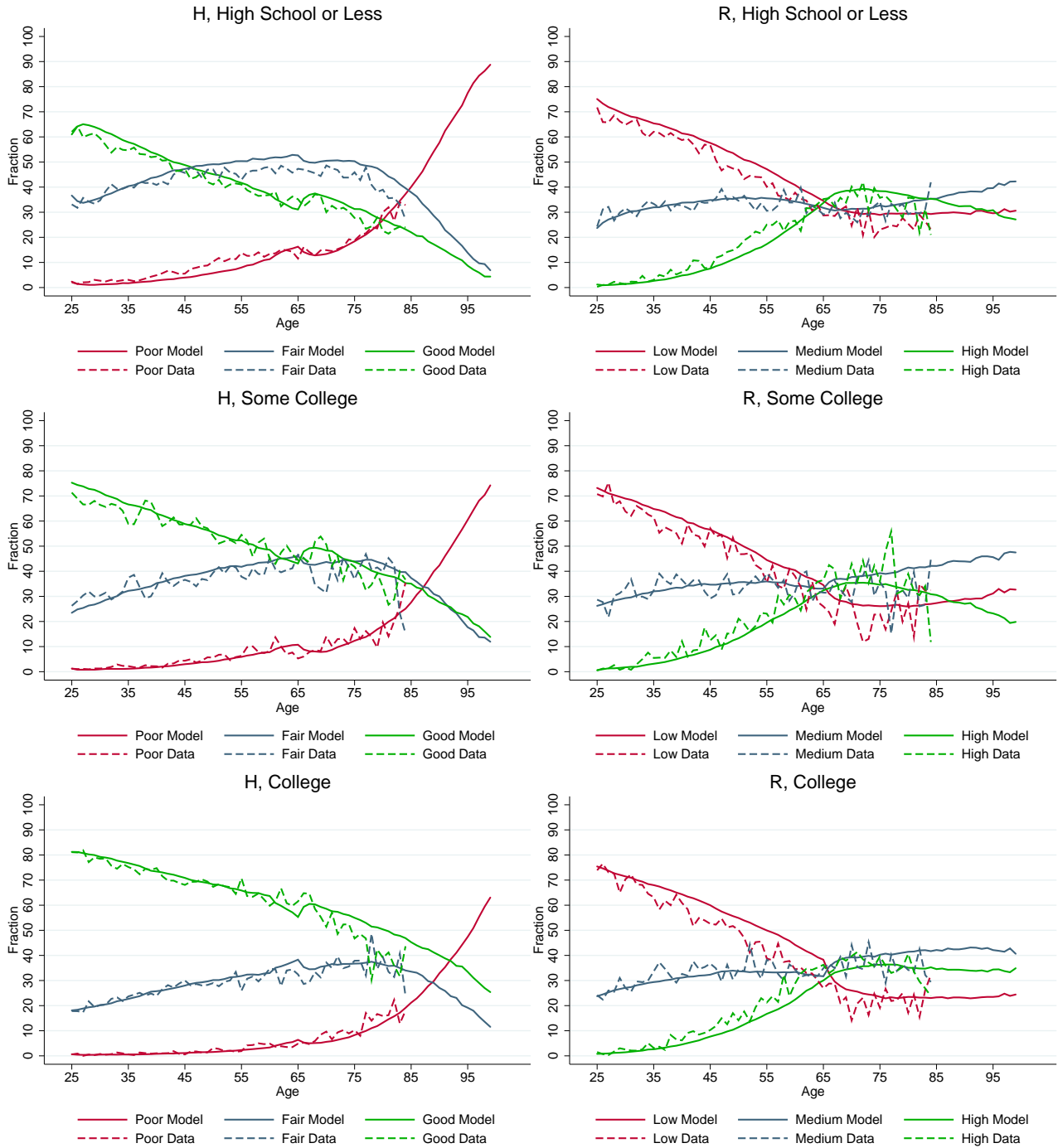
**Table 18: Mandatory Public Health Insurance Covering 70% of Medical Expenditures**

	<b>EMP (%)</b>		<b>SI (%)</b>		<b>PV Earnings</b>		<b>CEV (%)</b>
	<b>Bench</b>	<b>Public</b>	<b>Bench</b>	<b>Public</b>	<b>Bench</b>	<b>Public</b>	<b>Public</b>
<b>All</b>	83.1	85.3	12.9	8.8	762,177	+1.3%	1.44
<b>≤High School</b>	80.2	83.3	15.9	11.0	523,423	+1.9%	2.00
<b>Some College</b>	82.7	85.0	14.1	10.4	711,746	+1.3%	1.12
<b>College</b>	86.9	87.9	8.2	4.9	1,091,345	+0.9%	0.86
<b>≤High School</b>							
Low Productivity	57.1	65.1	41.6	31.6	293,730	+9.4%	1.03
Med Productivity	89.0	90.6	7.3	2.8	539,185	+0.9%	2.37
High Productivity	92.7	92.7	0.7	0.3	734,667	-0.1%	2.70
<b>Some College</b>							
Low Productivity	69.9	74.6	28.1	20.8	425,914	+4.5%	0.83
High Productivity	95.4	95.3	0.2	0.2	997,566	+0.0%	1.49
<b>College</b>							
Low Productivity	80.1	84.2	16.4	9.8	661,093	+4.8%	0.72
High Productivity	93.7	91.5	0.0	0.0	1,521,622	-0.8%	1.01

Notes: "EMP (%)" is the percentage of individuals employed either part or full time. All statistics are calculated in the cross-section of individuals 25-64 years of age.

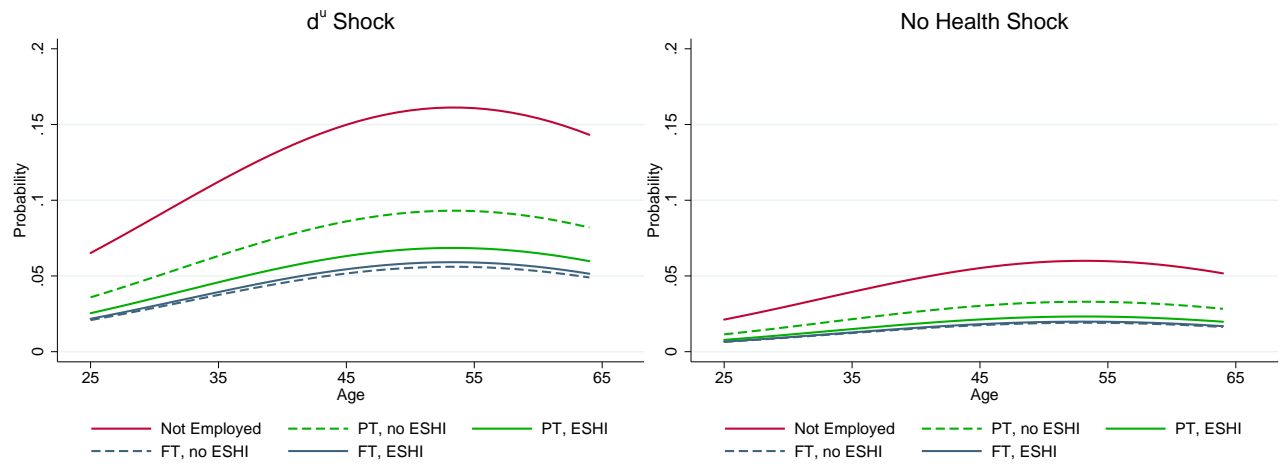
# Figures

**Figure 1:** Distribution of  $H$  and  $R$ , Model and Data



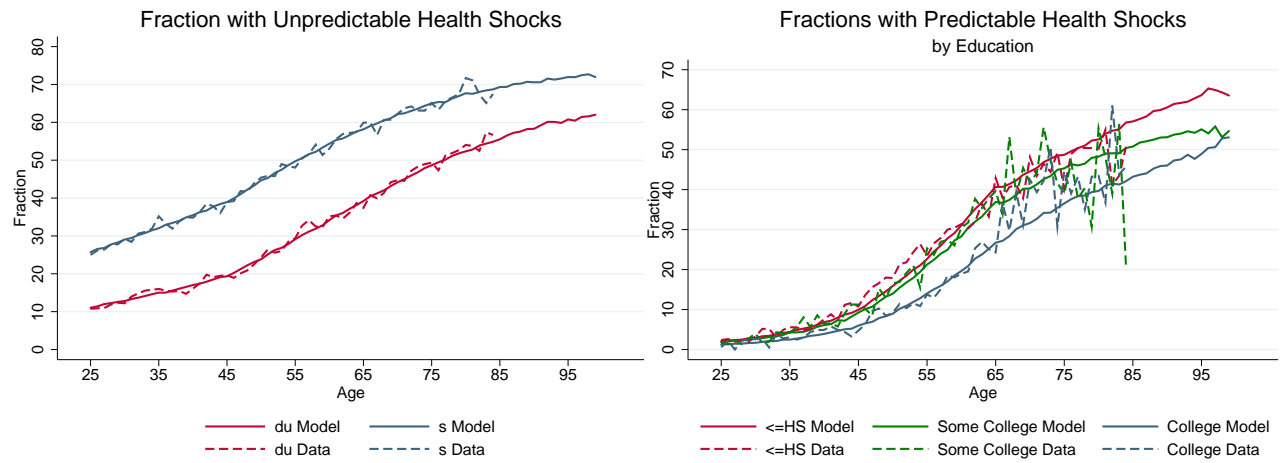


**Figure 2:** Selected Probabilities of Transitions from Fair to Poor Health ( $H$ ), High School or Less

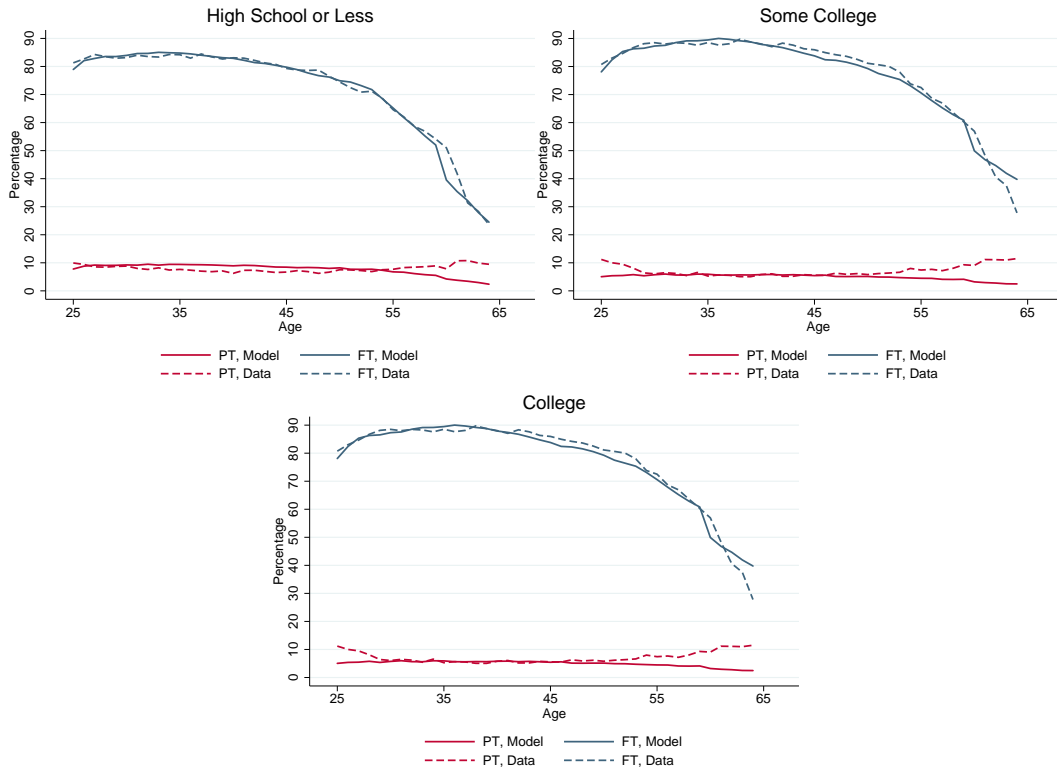


Note: All transitions are conditional on income quintile equal to 3.

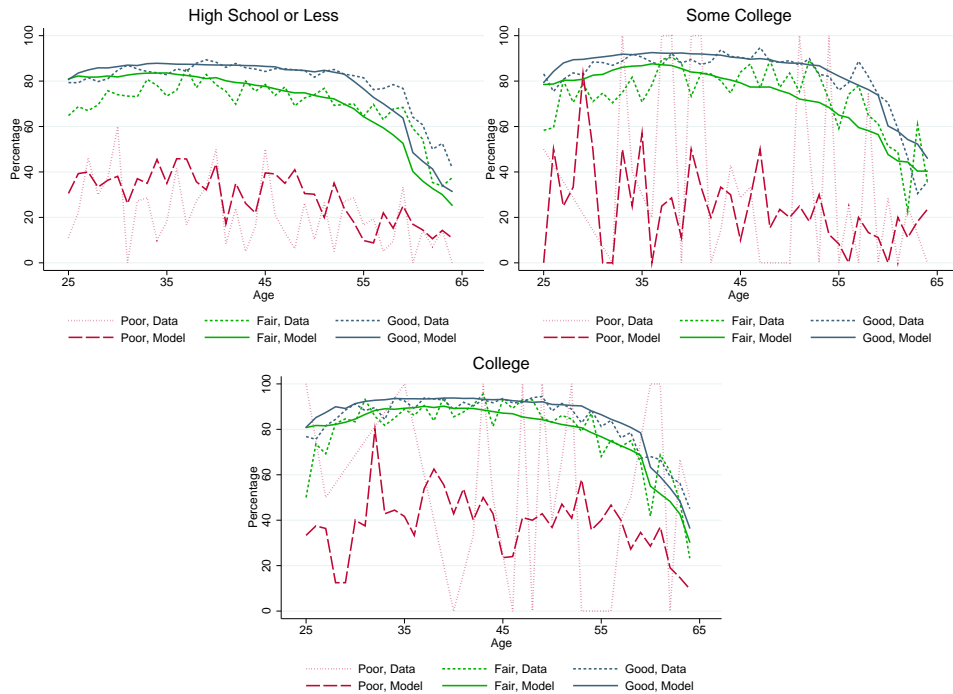
**Figure 3:** Fractions with  $d^u$ ,  $s$  and  $d^p$  Shocks by Age (Model and Data)



**Figure 4:** Distribution of Employment, Model and Data (CPS)

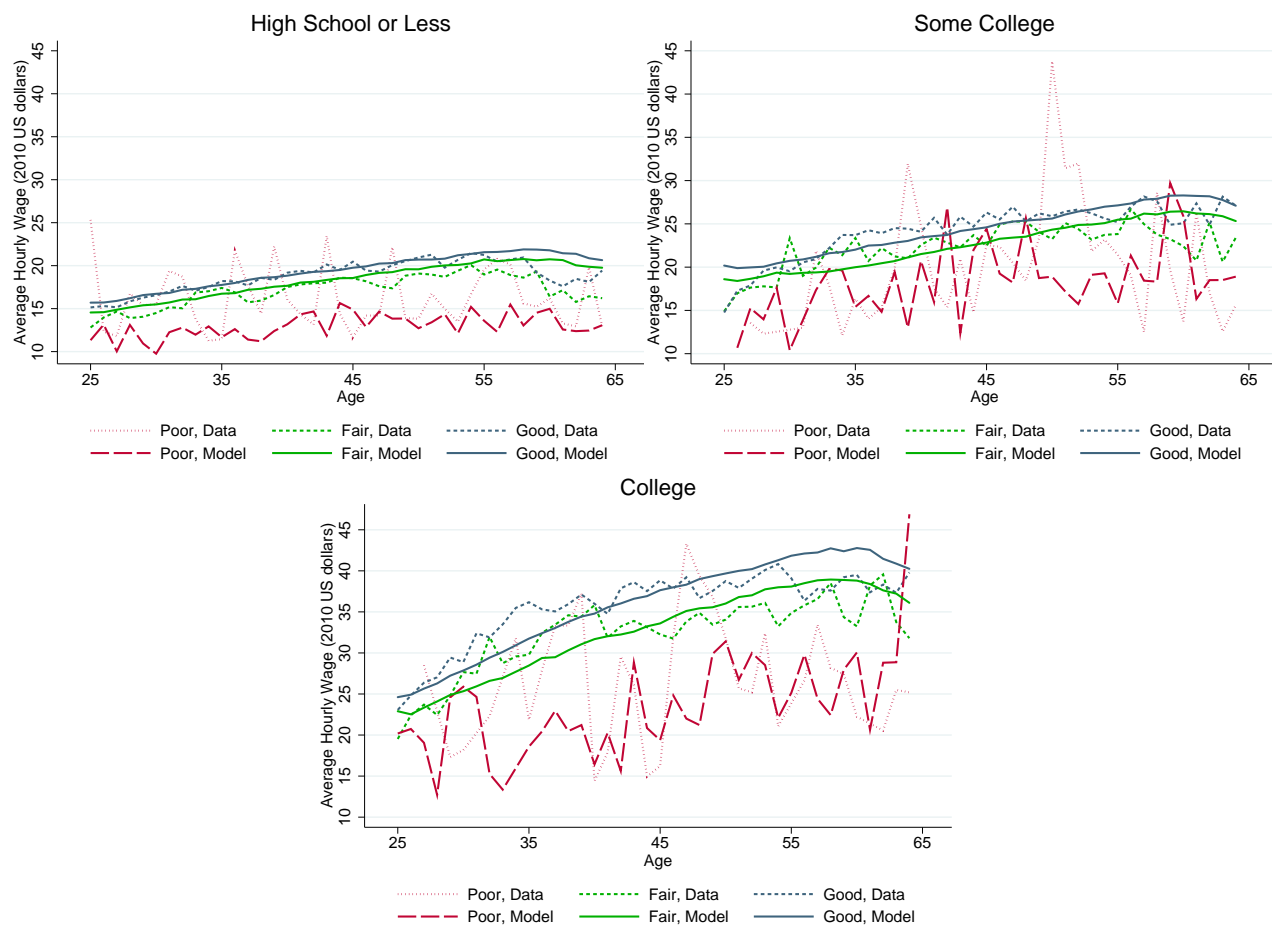


**Figure 5:** Distribution of FT Employment by Health and Age, Model and Data (MEPS)

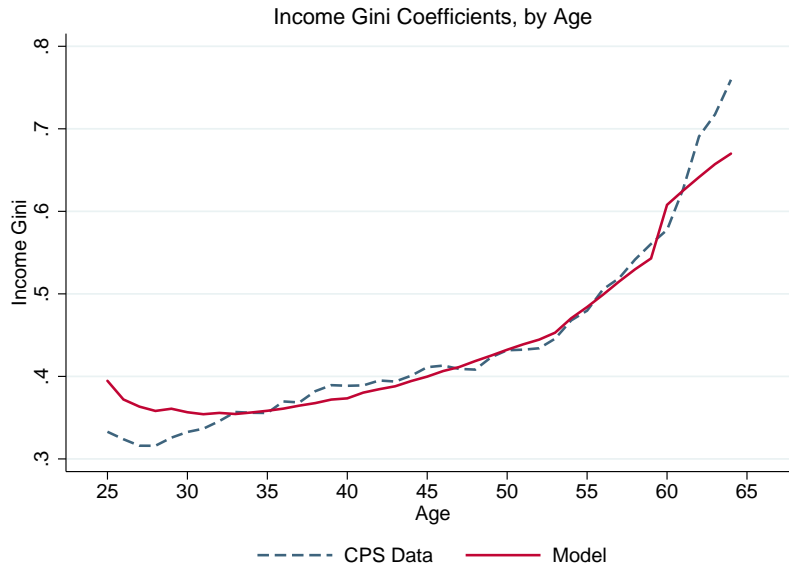


Note: The figure is constructed for workers with no persistent health shocks ( $d^u$  or  $d^p$ ).

**Figure 6:** Wage Profiles of Full Time Workers by Health, Model and Data (MEPS)

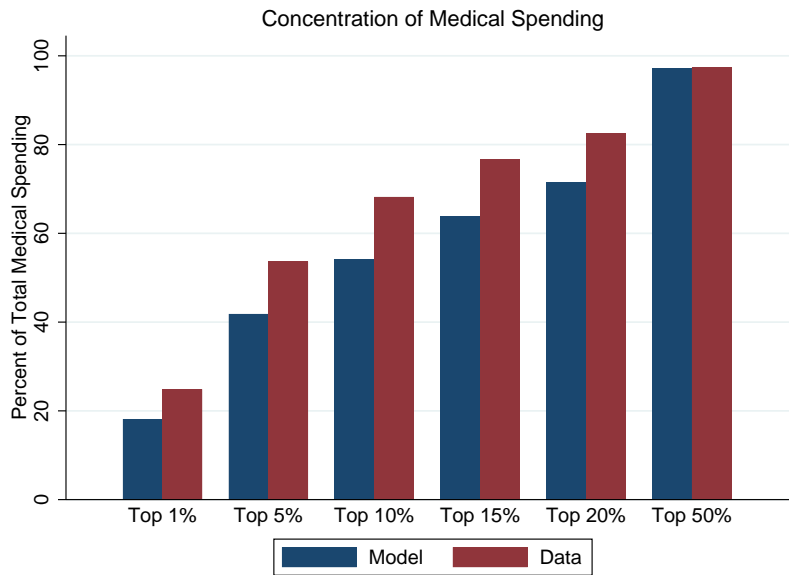


**Figure 7:** Income Inequality over the Life-cycle, Model and Data (CPS)

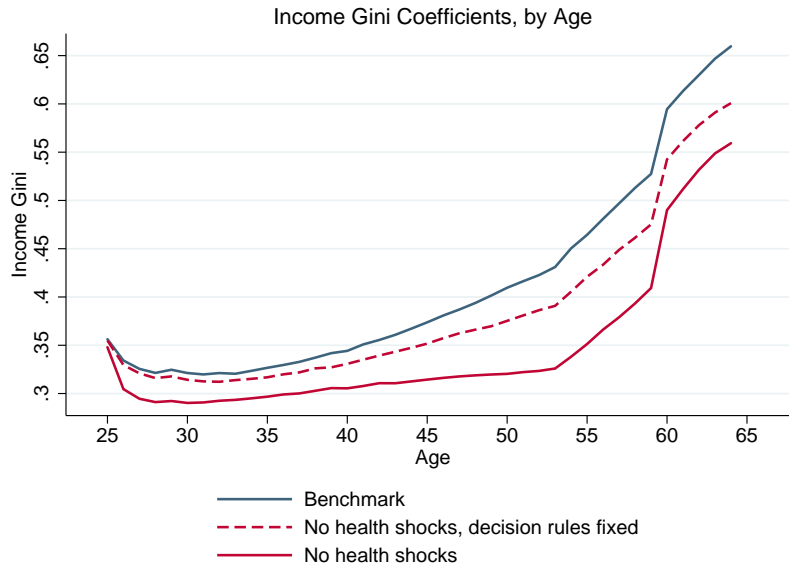


Note: Income equals earnings plus interest (both pre-tax). In the CPS, income inequality is calculated using men who are not in school or the armed forces. To reduce sensitivity of the Gini to outliers, we drop the top 2% of income observations at each age, as well as observations on employed workers with reported wage rates below the minimum wage. In the model, income is constructed using wages that include simulated measurement error.

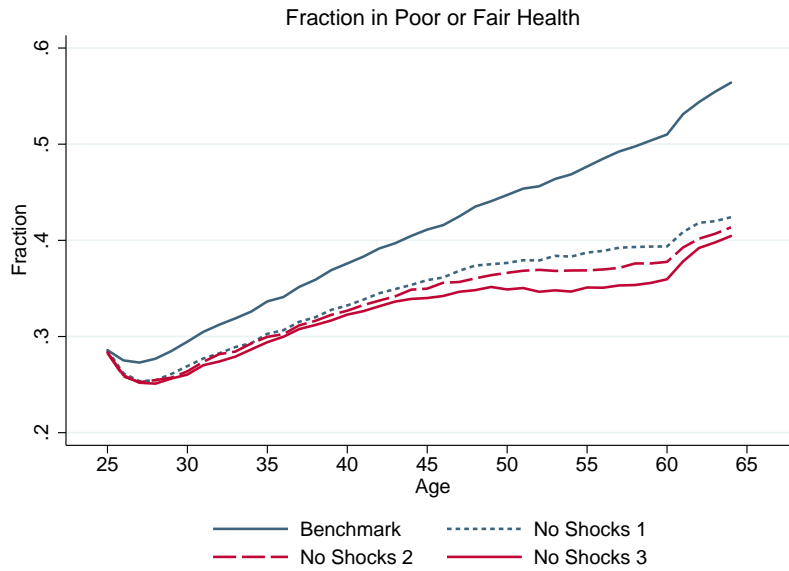
**Figure 8:** Distribution of Medical Spending, Ages 25-64, Model and Data (MEPS)



**Figure 9: Income Inequality over the Life-cycle**

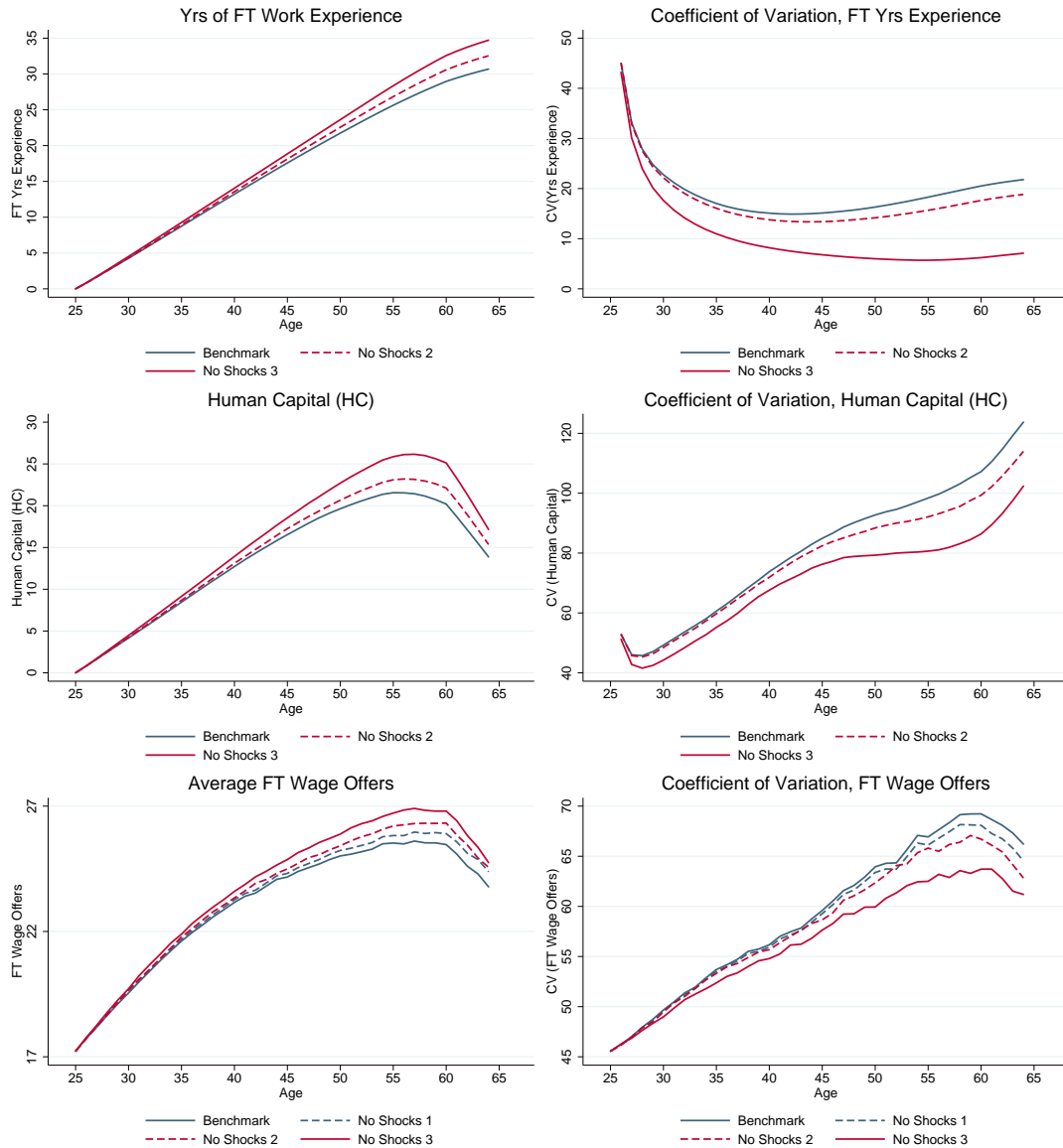


**Figure 10: Effects of Health Shocks on the Distribution of  $H$**



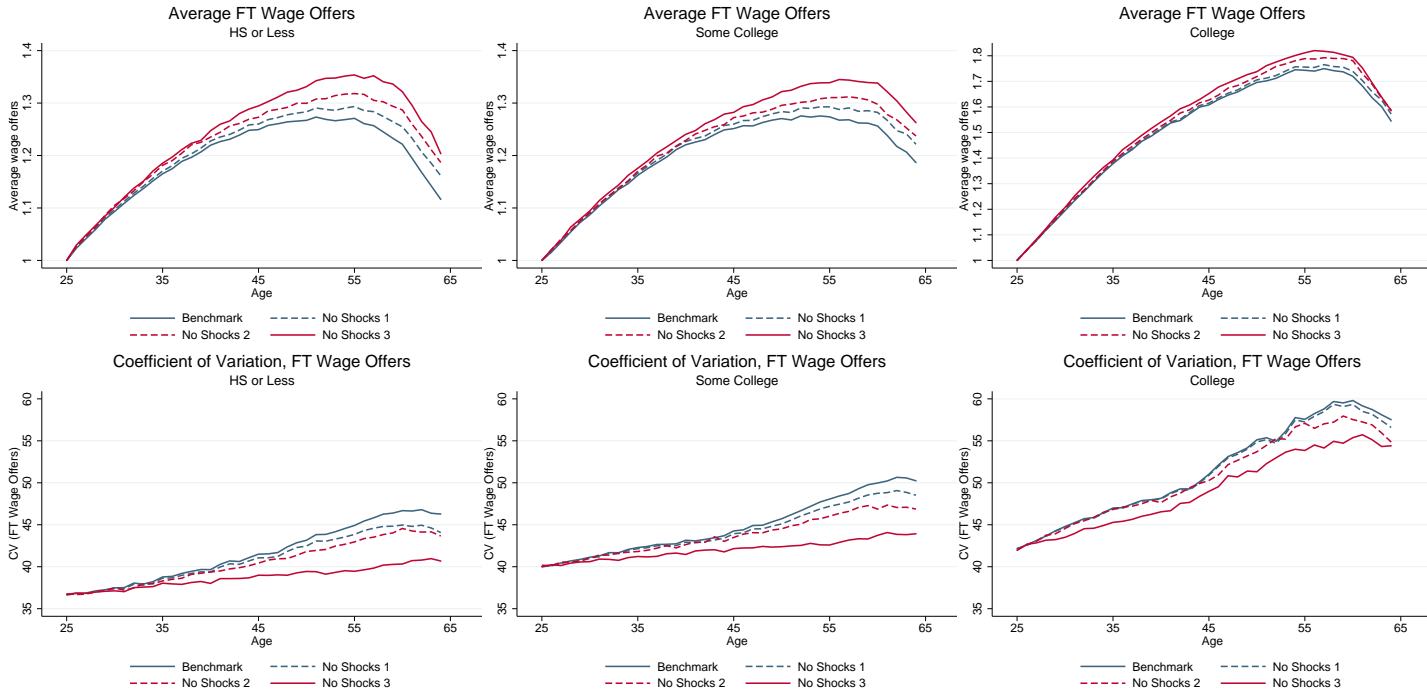
Note: In all three experiments we shut down health shocks at ages 25-64. (1) In “No Shocks 1” we hold employment, income and savings fixed at baseline values, so they cannot feedback and affect health. (2) In “No Shocks 2” we let agents adjust their labor supply and savings according to the optimal decision rules of the baseline model. (3) In “No Shocks 3” we let agents update their decision rules for labor supply and savings to reflect the new environment.

**Figure 11:** Effects of Health Shocks on Experience, Human Capital and Wage Offers



Note: In all three experiments we shut down health shocks at ages 25-64. (1) In “No Shocks 1” we hold employment, income and savings fixed at baseline values, so they cannot feedback and affect health. (2) In “No Shocks 2” we let agents adjust their labor supply and savings according to the optimal decision rules of the baseline model. (3) In “No Shocks 3” we let agents update their decision rules for labor supply and savings to reflect the new environment.

**Figure 12: Effects of Health Shocks on Wage Offers, by Education**



Note: In all three experiments we shut down health shocks at ages 25-64. (1) In “No Shocks 1” we hold employment, income and savings fixed at baseline values, so they cannot feedback and affect health. (2) In “No Shocks 2” we let agents adjust their labor supply and savings according to the optimal decision rules of the baseline model. (3) In “No Shocks 3” we let agents update their decision rules for labor supply and savings to reflect the new environment. In the figures, the mean full-time offer wage is normalized to 1.0 at age 25, within each education group. The actual means at age 25 are \$13.5, \$17.4 and \$21.4 for the High School, Some College and College groups, respectively.

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