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Health Inequality over the Life-Cycle

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Health Inequality over the Life-Cycle*

Timothy Halliday

Abstract

We estimate a dynamic model of health that is rooted in “stress models” from Epidemiology. Health is determined by time-invariant endowments, permanent shocks, and transitory shocks. We estimate that the variance in health at age 60 ranges between 2.5 and five times its variance at age 25 depending on which demographic group we consider. We show that the stress model performs better than a simple alternative, the random effects Probit, particularly for less educated people.

KEYWORDS: health, dynamic panel data models, variance decomposition

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1 Introduction

In this paper, we consider the evolution of the variance of health over the life-cycle. We do this using a model of health in which shocks can be either permanent or transitory. As individuals age, permanent shocks accumulate. On the other hand, temporary shocks affect health for a brief period, but then dissipate. This view of health as a non-stationary process in which the burden of past events persists until death has roots in “stress models” from Epidemiology, as discussed by McEwen and Stellar (1993) and Seeman, Singer, Rowe, Horwitz, and McEwen (1997).

During the past fifteen years, these models of health have started to penetrate Health Economics. The first instance of this that we are aware of is Deaton and Paxson (1998a). They point out that stress models have the desirable property that they imply that health inequality in the cross-section will increase as cohorts age which is a result that is consistent with their empirical evidence. In addition, these models bear a nice concordance with Deaton and Paxson (1994) who show a similar result for consumption inequality in a variety of contexts. More recently, stress models have formed the basis of the estimations of Adda, Banks, and von Gaudecker (2009).

This work contributes to a relatively new literature on the dynamics of health that is rooted in the earnings growth literature.¹ In one of the earliest studies, Shakotko (1980) investigates the formation of health and cognitive development in early childhood using factor structure models. More recently, Adda, Banks, and von Gaudecker (2009), Adams, Hurd, McFadden, Merrill, and Ribeiro (2003), and Borsch-Supan, Heiss, and Hurd (2003) investigate the joint dynamics of health and income using dynamic panel data techniques. These studies center largely on eliciting the causal pathways between health and socioeconomic status (SES). Other studies, such as Contoyannis, Jones, and Rice (2004a), Contoyannis, Jones, and Rice (2004b), Halliday (2008), and Carro and Traferri (2010), are more closely tied to the labor economics literature on income and employment dynamics (*e.g.* Hyslop (1999)). These studies focus exclusively on health status and emphasize the statistical properties of its dynamics by modeling it as a discrete variable and attempting to identify state dependence in the presence of unobserved heterogeneity. Despite recent progress, this is very much a fledgling field.

We further this field by exploring health inequality from a perspective that is largely ignored in the literature. Much of the extant literature on health inequality

¹Notable examples from the latter include Lillard and Willis (1978), Abowd and Card (1989), Baker (1997), Meghir and Pistaferri (2004), and Guvenen (2009).

focuses on disentangling causality between health and SES as in Adda, Banks, and von Gaudecker (2009) and Adams, Hurd, McFadden, Merrill, and Ribeiro (2003). However, the vast majority of the literature does not focus on the structural underpinnings of health inequality. This sentiment is echoed in Deaton and Paxson (1998a) where they note, “that much of the literature on health inequality is not concerned with inequality in years lived, but with the inequalities in health outcomes across socioeconomic groups.” In our view, this is a major omission since, as discussed by Blundell and Preston (1998), volatility in consumption can have a large impact on consumer welfare and we believe that the same can be said for volatility in health.

In this paper, we ask three questions. First, how does the variance of health evolve over the life-course? Second, how does this depend on SES? Third, how does the stress model of health compare to a simpler alternative with only time-invariant endowments and transitory shocks?

To help us to answer these questions, we model health as a continuous latent variable that forms the basis of a survey respondent’s self-reported health status (SRHS). Latent health depends on three factors: individual-specific endowments, transitory shocks, and permanent shocks. This exercise allows us to *quantify* how, given health inequality at age 25, the variance of health evolves with age. The existing descriptive evidence in Deaton and Paxson (1998a) and Deaton and Paxson (1998b) does not permit this as it relies on ordinal SRHS measures that can be arbitrarily modified by monotonic transformations. We employ the Panel Study of Income Dynamics (PSID) to estimate the model using a Simulated Method of Moments (SMM) estimator. One very important caveat to this exercise is that permanent shocks are the only means by which health inequality will increase with age in our model. In the conclusion, we discuss how this major limitation can be relaxed in future work.

Our main findings can be summarized as follows. First, we find that the variation in health at age 60 is substantially larger than the variation at age 25. We see the smallest disparity for college-educated women for whom the variance at age 60 is 2.5 times the variation at age 25. Men with some college experience but no degree exhibit the largest variation at age 60 which is about four to five times what it was at age 25. Second, we find that permanent shocks matter less for women and college-educated people. This finding complements Case and Deaton (2005) who conjecture that those with less education will exhibit a steeper decline in health with age because they tend to use their bodies more in their occupations. The wear-and-tear of manual labor, thus, can be viewed as a permanent shock. Third, when we compare the stress model to the random effects Probit model, we find that it performs better, particularly for less educated people.

The balance of this paper is organized as follows. Next, we set up our econometric model and discuss estimation of its parameters. After that, we describe our data. We then present our findings. Finally, we conclude.

2 A Stress Model of Health

We now formalize a stress model of health. At the core of these models is the notion of *allostatic load* which, to paraphrase, is the cumulative physiologic toll exacted on the body through multiple attempts to cope with stressors. Specifically, Seeman, Singer, Rowe, Horwitz, and McEwen (1997) say that allostatic load can be viewed as, “an index of the relative degree of failure at a physiological level (*i.e.* a marker of the cumulative, physiologic costs of previous efforts to cope with life’s slings and arrows.)”

To fix ideas, we follow Adda, Banks, and von Gaudecker (2009) and postulate the following model for individual i ’s latent health at time t (defined as h_{it}^*):

$$h_{it}^* = \delta + \gamma_i + u_{it} + \varepsilon_{it}. \quad (1)$$

There are three key terms in the model: endowments (denoted by γ_i), and a permanent and a transitory shock to health (denoted by u_{it} and ε_{it} , respectively). The term δ is a constant. The permanent shock is modeled as a random walk with drift:

$$u_{it} = \eta + u_{i(t-1)} + e_{it}, \quad (2)$$

where the drift term is given by η .² The permanent shock implies that latent health at time t will depend on $\sum_{s=0}^{t-1} e_{i(t-s)}$. This term represents allostatic load in our model. Finally, we assume that the transitory shock follows a white noise process. This is a special case of Adda, Banks, and von Gaudecker (2009) who allow the transitory shock to follow an MA(q). The process is observed until T .

We treat the initial condition as follows. We assume that the process begins at $t = 1$ and that $u_{i0} = 0$. Similar to Halliday (2008), we assume that the process begins during a person's twenties after their childhood has ended. We assume that the initial condition is age 25, so that $t = 1$ corresponds to age 25. We do so because we suspect that the process guiding a child's health should be substantially different than the process governing the health of an adult.

Each component of equation (1) has an interpretation. The permanent shock, which is modeled as e_{it} , represents events that leave a residue on one's health, such as onset of chronic illness or accidents that have lasting effects. Endowments, modeled as γ_i , represent time-invariant personal characteristics formed early in life that affect a person's health throughout the life-course. The term ε_{it} , which models transitory shocks, could include mild bouts of illnesses, such as the flu or broken bones.

Stacking the permanent and transitory shocks as $u_i \equiv (u_{i1}, \dots, u_{iT})'$ and $\varepsilon_i \equiv (\varepsilon_{i1}, \dots, \varepsilon_{iT})'$, the covariance matrix is

$$\begin{pmatrix} \gamma_i \\ u_i \\ \varepsilon_i \end{pmatrix} \sim N \left(\mathbf{0}, \begin{bmatrix} \sigma_\gamma^2 & \mathbf{0}_{1,T} & \mathbf{0}_{1,T} \\ \mathbf{0}_{T,1} & \Sigma^{PS} & \mathbf{0}_{T,T} \\ \mathbf{0}_{T,1} & \mathbf{0}_{T,T} & \sigma_\varepsilon^2 \mathbf{I}_T \end{bmatrix} \right) \quad (3)$$

where

$$\Sigma^{PS} = \begin{bmatrix} \sigma_e^2 & \dots & \dots & \sigma_e^2 \\ \vdots & 2\sigma_e^2 & \dots & 2\sigma_e^2 \\ \vdots & \vdots & \ddots & \vdots \\ \sigma_e^2 & 2\sigma_e^2 & \dots & T\sigma_e^2 \end{bmatrix}.$$

This structure implies that latent health will be non-stationary, serially correlated at all leads and lags, and heteroskedastic.³ Finally, the latent variable structure requires a normalization, so we set $\sigma_\varepsilon^2 = 1$.

²We do not allow for an AR term in this process *i.e.* $u_{it} = \eta + \rho u_{i(t-1)} + e_{it}$. In our view, shocks should either be permanent or transitory. Allowing for both a (possibly) stationary AR process and an MA process seems odd since if the AR process is stationary, both processes are essentially modeling transitory shocks.

³While writing this paper, we also attempted to estimate some more general models. One

We can decompose the variance of the latent health variable as follows. Given our assumptions on the initial condition, we can write

$$h_{it}^* = \delta + \gamma_i + \eta t + \varepsilon_{it} + \sum_{s=0}^{t-1} e_{i(t-s)} \quad (4)$$

which, in turn, implies

$$\sigma_{h_{it}^*}^2 = \sigma_\gamma^2 + t\sigma_e^2 + 1. \quad (5)$$

At any point in time, health inequality depends on the variances of the endowments and both types of shocks.

This formula has several important implications. First, it tells us that inequality in latent health increases as the cohort ages. This result is consistent with empirical evidence both on health inequality (Deaton and Paxson (1998a) and Deaton and Paxson (1998b)) and consumption inequality (Deaton and Paxson (1994) and Primiceri and van Rens (2009)). Second, permanent shocks explain an increasing portion of health inequality within a cohort. Hence, as people age and adverse health events accumulate for some but not others, the disparity between the healthiest and unhealthiest widens.

The econometrician does not observe the individual's latent health stock. Rather, she observes the agent's SRHS, which we denote by h_{it} . The agent's SRHS is reported according to the rule

$$h_{it} = d \Leftrightarrow \alpha_{d-1} \geq h_{it}^* > \alpha_d \quad (6)$$

for $d \in \{1, \dots, 4\}$, where $\alpha_0 = \infty$, $\alpha_1 = 0$ and $\alpha_4 = -\infty$. The health states in (6) correspond to the different categorizations of SRHS. Once we account for the cut points, the parameter vector to be estimated is $\theta \equiv (\delta, \sigma_\gamma^2, \sigma_e^2, \eta, \alpha_2, \alpha_3)$.⁴

generalized the white noise process for the transitory shock to an MA(1) process. The other also allowed the permanent shock to be correlated with the endowments. When we optimized the SMM objective function from separate starting values, we found that the two resulting minimized objective functions were numerically very close, but the MA and correlation coefficients that optimized the functions were drastically different. This is an identification problem. Consequently, we decided against further exploration of these models in this paper.

⁴Three issues require some clarification. First, there is an inverse relationship between h_{it} and h_{it}^* , so that higher values of h_{it}^* and lower values of h_{it} denote better health. Our reason for doing this is that SRHS measures are such that lower values correspond better health, but when health is incorporated in economic models as a continuous variable (most notably by Grossman (1972)), higher values correspond to better health. Hence, we maintain consistency with both the standard way of measuring SRHS and also conventions in the literature on health investment. Second, there are only three finite cut points because we consolidate the "fair" and "poor" states into a single

Identification of θ is as follows. The error structure in our model has a one-factor representation, so the linear index can be written as $\kappa_t \alpha_i + v_{it}$ where the v_{it} are serially uncorrelated. Subject to a normalization, the parameters in this model are easily identified. These parameters can then be mapped into θ . In other words, θ can be backed out of the parameters from the one-factor model. For a more detailed treatment, we refer the reader to Heckman (1981). Note that the one-factor representation imposes non-stationarity and so precludes stationary AR processes.

3 Estimation

We propose a tractable SMM procedure that matches the probabilities of simulated sequences of h_{it} with their counterparts in the data to estimate θ . First, we simulate the model for 250,000 individuals.⁵ For each individual, we simulate the model from age 25 to 60. We then take "snapshots" of sub-sequences of the 36-period sequence of health outcomes. The specific sub-sequences used are in the Appendix. We collect all of these probabilities in the vector $P^M(\theta)$. Next, we collect the analogues of $P^M(\theta)$ from the data in P^D . These probabilities are estimated non-parametrically. Our moments are then defined as $m(\theta) \equiv P^M(\theta) - P^D$. For reasons discussed in Altonji and Segal (1996), we follow Meghir and Pistaferri (2004) and employ Equally Weighted Minimum Distance, which minimizes the objective function:

$$Q(\theta) = m(\theta)' m(\theta).$$

Once again following Meghir and Pistaferri (2004), we use the block bootstrap in which we re-sample individuals (not individual-time observations) to compute standard errors.⁶ This procedure accounts for correlations in observations within individuals and across time. Further details are in the Appendix.

4 Data

We use a sample of Caucasian women and men ages 25 to 60 from PSID waves 1984 to 1997. We do not use data before 1984 because there is no information on SRHS

category. We do this because there are often too few observations of poor health in certain age cells which created difficulties pinning down the bottom cut point with all five categories. Third, as in Carro and Traferri (2010), we normalize α_1 to zero because we include a constant in equation (1).

⁵We use the same simulations for all of our estimations.

⁶We re-sampled the data 50 times.

prior to this year. We do not go beyond 1997 as the PSID was collected every other year beyond 1997.

Our main health measure is SRHS, a categorical variable by which the respondent classifies their health into one of five categories: Excellent (SRHS equal to 1), Very Good (SRHS equal to 2), Good (SRHS equal to 3), Fair (SRHS equal to 4), and Poor (SRHS equal to 5). For the main analysis, we also use data on age and educational attainment. A detailed discussion of our sample selection is provided in the Appendix.⁷

We estimate the model for six demographic sub-groups corresponding to three educational categories separated by gender. The first educational category is "College" and corresponds to people who hold college degrees. The second is "HS Grad" and corresponds to people who have more than twelve years of schooling but no college degree. The third is "HS" and corresponds to people with twelve years of schooling or less. This partition of the data closely mimics Meghir and Pistaferri (2004).

To demonstrate the degree of health inequality in the PSID, we present Figures 1 through 6 where each figure corresponds to a separate gender-education category. We plot the evolution of the 5th, 50th and 95th percentiles of SRHS over the life-course as in Deaton and Paxson (1998b). The figures show that the discrepancy between the 95th and the 5th quantiles fans out with age for all demographic groups. However, because the SRHS measures are ordinal, they do not allow for statements about how much the variation in health increases with age. This motivates the need for our latent variable model which does allow such statements subject to a normalization.

⁷While SRHS can be criticized as being subjective, it does have some merit. First, it has long been documented that these measures of health correlate well with more objective health measures. Second, many alternative health measures are not without flaws. For example, Baker, Stabile, and Deri (2004) investigated the possibility of measurement errors in self-reported, objective measures of health (such as those from the Health and Retirement Survey) by comparing them with medical records. They concluded that these measurement errors were often quite large and regrettably correlated with labor market activity.

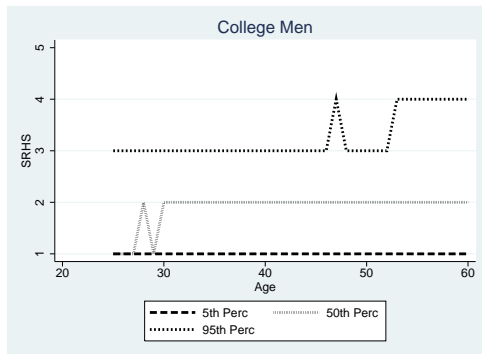


Figure 1

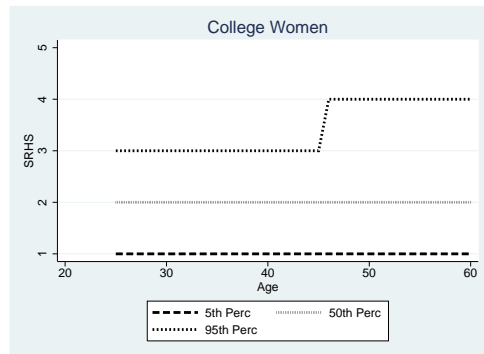


Figure 4

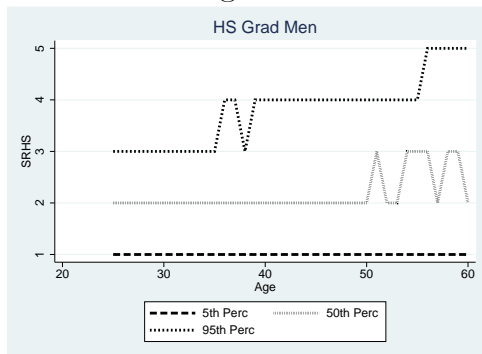


Figure 2

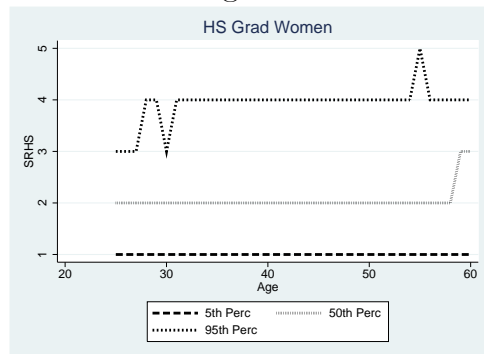


Figure 5

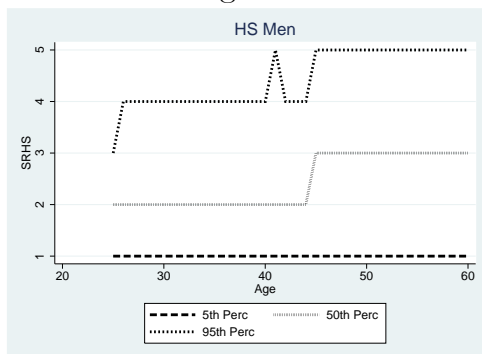


Figure 3

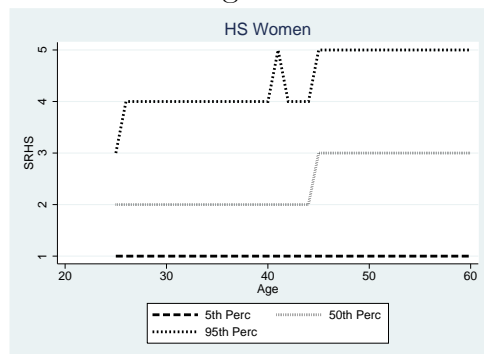


Figure 6

Halliday: Health Inequality

	College	HS Grad	HS
	(1)	(2)	(3)
α_2	-2.3643 (0.1694)	-2.5106 (0.2517)	-2.4251 (0.2128)
α_3	-4.7677 (0.4238)	-4.8669 (0.5333)	-4.9349 (0.3742)
δ	0.1205 (0.1453)	-0.2921 (0.2142)	-1.0649 (0.1908)
η	-0.0367 (0.0066)	-0.0688 (0.0148)	-0.0664 (0.0099)
σ_e^2	0.2374 (0.1400)	0.3407 (0.1013)	0.3063 (0.0703)
σ_γ^2	1.8518 (0.2131)	1.7137 (0.2824)	2.0237 (0.2267)
$Q_{UNRE}(\hat{\theta})$	0.0615	0.1155	0.0514
$Q_{\sigma_e^2=0}(\hat{\theta})$	0.0695	0.1598	0.0756
$\frac{Q_{UNRE}(\hat{\theta})}{Q_{\sigma_e^2=0}(\hat{\theta})}$	88.5%	72.3%	67.8%
N	1588	1091	2870

For each parameter, we report the point-estimate and the bootstrapped standard error in parentheses. The value for $Q_{UNRE}(\hat{\theta})$ corresponds to the objective function maximized subject to no restrictions. The value for $Q_{\sigma_e^2=0}(\hat{\theta})$ corresponds to the objective function maximized subject to the restriction that $\sigma_e^2 = 0$.

Table 1: Parameter Estimates Men

	College	HS Grad	HS
	(1)	(2)	(3)
α_2	-2.3147 (0.1962)	-2.2275 (0.2486)	-2.3087 (0.1464)
α_3	-4.4209 (0.4995)	-4.5117 (0.4787)	-4.7276 (0.2990)
δ	-0.2442 (0.1636)	-0.7033 (0.2114)	-1.3534 (0.1736)
η	-0.0343 (0.0080)	-0.0402 (0.0117)	-0.0540 (0.0089)
σ_e^2	0.1320 (0.1594)	0.2591 (0.1007)	0.2916 (0.0628)
σ_γ^2	1.8276 (0.2451)	1.7417 (0.2483)	1.8035 (0.1666)
$Q_{UNRE}(\hat{\theta})$	0.1089	0.1093	0.0544
$Q_{\sigma_e^2=0}(\hat{\theta})$	0.1092	0.1316	0.0788
$\frac{Q_{UNRE}(\hat{\theta})}{Q_{\sigma_e^2=0}(\hat{\theta})}$	99.7%	83.1%	69.0%
N	1321	1040	2901

Per Table 1.

Table 2: Parameter Estimates Women

5 Empirical Results

We report point estimates and their bootstrapped standard errors in Table 1 for men and Table 2 for women. The tables indicate that for certain demographic groups there is a pronounced increase in inequality with age. For men, we see estimates of σ_e^2 of 0.3407 (HS Grad) and 0.3063 (HS). Both of these are greater than two standard errors above zero. In contrast, the estimate for college-educated men is 0.2374. This finding is reflected in Figures 1 through 3 which show that the increase in inequality appears to be greater for people without college degrees. A similar pattern exists for women. The estimates of σ_e^2 are 0.2591 (HS Grad) and 0.2916 (HS). Both are greater than two standard errors above zero. On the other hand,

the estimate for college-educated women is 0.1320. As for men, we see in Figures 4 through 6 that the increase in inequality is smallest for college-educated women.

In Figures 7 and 8, we display the life-cycle profile of the total variance in health normalized by its variance at age 25. For men, we see that the total variation at age 60 ranges between 3.5 (College) and five (HS Grad) times what it was at age 25. This suggests that men exhibit a steep rise in health inequality with age. We also see a similar pattern for women, although the increase over the life-cycle is less pronounced. The variation at age 60 ranges from 2.5 (College) to just over four (HS). One interesting pattern in these figures is that permanent shocks matter less for people with college degrees and for women. In particular, for women, there is a monotonic relationship between educational attainment and the importance of permanent shocks.

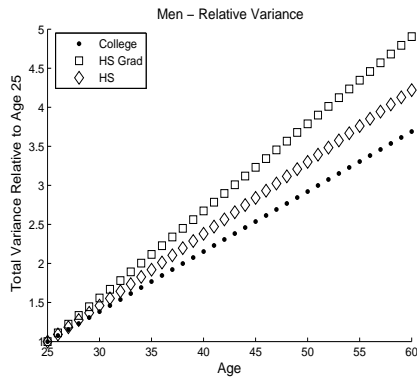


Figure 7

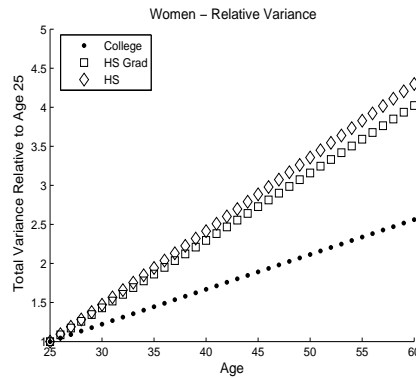


Figure 8

Finally, to demonstrate how the stress model compares to a simple random effects probit model, we compute something akin to a likelihood ratio statistic. To do this, we re-estimate the model subject to the restriction that $\sigma_e^2 = 0$ using the same moments and simulations. We report the value of the GMM objective function with this restriction as $Q_{\sigma_e^2=0}(\hat{\theta})$. We then compare this to the objective function from the unrestricted model which we call $Q_{UNRE}(\hat{\theta})$. The statistic that we report is

$$\frac{Q_{UNRE}(\hat{\theta})}{Q_{\sigma_e^2=0}(\hat{\theta})} \quad 8$$

We see that the model tends to perform better for people with less education. For men, the statistics are 88.5% (College), 72.3% (HS Grad) and 67.8% (HS). For

⁸While the "Holy Trinity" of likelihood based tests can be extended to GMM estimators as discussed in Newey and McFadden (1994), they cannot be applied in our case. The reason is that for the asymptotic theory of these tests to be applicable the parameter value under the null cannot be at the edge of a compact set. If it is, one cannot apply simple asymptotic results such as the

women, the statistics are 99.7% (College), 83.1% (HS Grad), and 69.0% (HS). For both genders, the statistics increase monotonically with educational attainment consistent with the notion that permanent shocks matter less for more educated people. Similarly, we also find that within educational groups the statistics are higher for women than for men suggesting that permanent shocks matter more for men.

6 Discussion

An important avenue for future work is to incorporate state dependence into the existing framework.⁹ First, one could allow the endowment to be correlated with the permanent and transitory shocks. This would allow events that occurred in childhood, which should be captured in the endowment, to impact what happens in adulthood. For example, malnourishment in childhood would impact the individual's endowment and would, in turn, affect the distribution of events faced by the person as an adult. Second, researchers could allow the permanent innovation to be correlated across time. This would allow people with a high disease burden to experience additional disease with higher probability.

In addition, researchers may also explore a stationary AR model with heterogeneous trends, a common alternative to the permanent-transitory model in the earnings dynamics literature (*e.g.* Baker (1997) and Guvenen (2009)). However, this exercise, while very important, would entail some challenges. In linear models, autocovariances of earnings growth yield a clean way of differentiating between the two models since the permanent-transitory model implies that these autocovariances should go to zero at long lags, whereas the other model does not. Unfortunately, we have a latent variable framework in this paper and, so we cannot do this. As such, we would need to rely on non-nested hypothesis testing which may have poor finite sample performance given our data.

However, there are two other findings in the literature that challenge the validity of the AR model with heterogeneous trends. First, in Halliday (2008), we explore a model with heterogeneous age trends and state dependence. The data reject the presence of heterogeneous trends in this related model. Nevertheless, researchers may want to see if replacing state dependence with stationary AR residuals changes this. Second, results in Deaton and Paxson (1998a) and Deaton and Paxson (1998b) as well this paper show that there is a tendency for within-cohort inequality in numer-

Central Limit Theorem to a first-order Taylor expansion. One way to rectify this would be to use a model selection criterion such as the AIC or BIC. However, these will not work either as these methods are likelihood based.

⁹By "state dependence," we mean that previous health outcomes condition what happens in the future.

ous health measures to fan out as the cohort ages. In the absence of heterogeneous trends, this finding would necessitate permanent shocks.

Understanding which of these models is more appropriate is important as they have very different implications for consumer behavior. To illustrate, we refer to Deaton (1992), who discusses the implications that different income processes have on consumption behavior in a stripped down life-cycle model with quadratic preferences and a discount factor set to the inverse of the real rate of return. He shows that modeling income either as trend stationary or as difference stationary has radically different implications for consumer behavior. The former (which many believe to be less plausible) implies that consumption is smoother than income, whereas the latter implies the opposite. Analogously, many health economists who have investigated the dynamics of health, have essentially modelled health as trend stationary (*e.g.* Contoyannis, Jones, and Rice (2004a), Contoyannis, Jones, and Rice (2004b), Halliday (2008), and Carro and Traferri (2010)). We contend that more work is needed using different estimators, modeling assumptions, and data sources if we wish to get a better handle on the stochastic process governing health. As illustrated by Deaton (1992), the consequences of different health processes for consumer behavior may be non-trivial.¹⁰

Finally, our investigation into the volatility of health suggests another dimension of the association between health and SES. As pointed out by Blundell and Preston (1998), “risk averse households with more uncertain incomes than others need to be considered worse off.” For example, if there is a precautionary savings motive, then this will result in lower consumption expenditures. A similar argument can be made for the volatility of health. First, to the extent that health shocks impact income, more volatile health implies more volatile income. Second, since health also has consumption value, then a mean-preserving spread in health will lower expected utility for risk averse people. So, our paper suggests that college-educated people are better off not only because their health is better but also because it is less uncertain. This is another dimension of the gradient that has yet to be mentioned in the literature.

7 Appendix: Sample Selection

We first extract all individuals from the 1984 to 1997 waves of the PSID who are either heads of household or the spouse of a household head. The initial sample size was 24,167 individuals. Next, we drop people with incomplete health data bringing

¹⁰For example, if health is modelled as an exogenous and continuous variable (as in this paper) and is allowed to impact income, the model is essentially the same as in Deaton (1992).

the sample size to 24,054. After this, we keep only Caucasians resulting in a sample size of 14,783. We then drop people who are not in the panel continuously. This lowers the sample size to 13,805. Next, we keep only people between ages 25 and 60, inclusive. This brings the sample size to 11,018. Note that there are an additional 207 people with missing educational information and, so if we add them to the six samples sizes from the table we obtain 11,018. Finally, we include the Survey of Economic Opportunity.¹¹

8 Appendix: Estimation Details

Optimization To obtain our parameter estimates, we optimize the GMM objective function using simulated annealing (SA). For each demographic sub-group, we run the procedure once and then we use the maximizer of that procedure as the starting value for a second run of SA. To be certain that we had the true maximand, we use the maximizer of the second run of SA as the starting value of a final run of Nelder-Mead (NM). The primary advantage of SA over NM is that it is able to go both uphill and downhill which makes it less vulnerable to getting stuck in local minima. The cost is that it is substantially slower than NM. Finally, to be entirely sure that we do not have any issues with local minima or weak identification, we run this whole routine twice using two different starting values. We did not encounter any problems with local minima or weak identification.

Moments We use two sets of moments in the estimation. The first set contains the unconditional probabilities of the four health states that we consider (*i.e.* excellent, very good, good, fair/poor). Here, we compute the unconditional probability of these four health states for a total of 36 ages (*i.e.* ages 25 to 60). This yields a total of $3 \times 36 = 108$ moments. These moments are informative of the drift parameter, constant and the cuts. In addition, as shown in Heckman (1981), they are also informative of the variance of the permanent shock. The second set of moments we use are sequences of length four of health states. We use the three most common sequences for each demographic sub-group at ages 25, 30, 35, 40, 45, 50, 55, and 60. The sequences that we use can be found in Tables 3 and 4.¹² This yields a total of $3 \times 7 = 21$ additional moments. These moments add additional information about the variance of the endowments and permanent shocks. In total, we use 129 moments.

¹¹Our estimation method places heavy demands on the data. Including the SEO prevents the sample sizes from becoming too small.

¹²The table reports the three most common sequences at each age. In the reported fraction, the numerator is the number of occurrences of each sequence and the denominator is the number of people who are present at that age and who remain for at least four periods.

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College		HS Grad		HS	
Age 25					
1111	$\frac{47}{185} = 0.254$	1111	$\frac{28}{156} = 0.179$	2222	$\frac{52}{374} = 0.139$
2222	$\frac{24}{185} = 0.130$	2222	$\frac{18}{156} = 0.115$	1111	$\frac{43}{374} = 0.115$
2111	$\frac{11}{185} = 0.059$	3222	$\frac{8}{156} = 0.051$	3333	$\frac{21}{374} = 0.056$
Age 30					
1111	$\frac{81}{343} = 0.236$	1111	$\frac{36}{240} = 0.150$	2222	$\frac{71}{532} = 0.133$
2222	$\frac{31}{343} = 0.090$	2222	$\frac{31}{240} = 0.129$	1111	$\frac{58}{532} = 0.109$
1112	$\frac{18}{343} = 0.052$	2221	$\frac{9}{240} = 0.0375$	3333	$\frac{27}{532} = 0.051$
Age 35					
1111	$\frac{99}{406} = 0.244$	2222	$\frac{33}{242} = 0.136$	2222	$\frac{51}{474} = 0.108$
2222	$\frac{45}{406} = 0.111$	1111	$\frac{33}{242} = 0.136$	3333	$\frac{37}{474} = 0.078$
1112	$\frac{16}{406} = 0.039$	3333	$\frac{10}{242} = 0.041$	1111	$\frac{33}{474} = 0.069$
Age 40					
1111	$\frac{98}{404} = 0.243$	1111	$\frac{40}{206} = 0.194$	2222	$\frac{32}{349} = 0.092$
2222	$\frac{38}{404} = 0.094$	2222	$\frac{17}{206} = 0.083$	1111	$\frac{29}{349} = 0.083$
3333	$\frac{18}{404} = 0.045$	3333	$\frac{12}{206} = 0.058$	3333	$\frac{349}{24} = 0.069$
Age 45					
1111	$\frac{71}{305} = 0.233$	2222	$\frac{14}{115} = 0.121$	3333	$\frac{27}{287} = 0.094$
2222	$\frac{44}{305} = 0.144$	3333	$\frac{9}{115} = 0.078$	4444	$\frac{26}{287} = 0.091$
2232	$\frac{10}{305} = 0.033$	1111	$\frac{9}{115} = 0.078$	2222	$\frac{287}{24} = 0.084$
Age 50					
1111	$\frac{26}{141} = 0.184$	1111	$\frac{7}{74} = 0.095$	4444	$\frac{31}{228} = 0.136$
2222	$\frac{17}{141} = 0.121$	2222	$\frac{7}{74} = 0.095$	3333	$\frac{17}{228} = 0.075$
2111	$\frac{7}{141} = 0.050$	3333	$\frac{5}{74} = 0.068$	3332	$\frac{11}{228} = 0.048$
Age 55					
1111	$\frac{21}{107} = 0.196$	4444	$\frac{12}{75} = 0.160$	4444	$\frac{26}{231} = 0.113$
2222	$\frac{8}{107} = 0.075$	2222	$\frac{7}{75} = 0.093$	3333	$\frac{22}{231} = 0.095$
3332	$\frac{5}{107} = 0.047$	3232	$\frac{3}{75} = 0.053$	2222	$\frac{18}{231} = 0.078$

See footnote 12 for details.

Table 3: Sequences, Men

College		HS Grad		HS	
Age 25					
1111	$\frac{38}{215} = 0.177$	1111	$\frac{27}{205} = 0.131$	2222	$\frac{37}{354} = 0.104$
2222	$\frac{12}{215} = 0.056$	2222	$\frac{24}{205} = 0.117$	1111	$\frac{24}{354} = 0.068$
2232	$\frac{10}{215} = 0.051$	3333	$\frac{11}{205} = 0.054$	3333	$\frac{23}{354} = 0.065$
Age 30					
1111	$\frac{74}{354} = 0.209$	1111	$\frac{31}{266} = 0.117$	2222	$\frac{51}{514} = 0.099$
2222	$\frac{36}{354} = 0.102$	2222	$\frac{22}{266} = 0.083$	3333	$\frac{36}{514} = 0.070$
2212	$\frac{12}{354} = 0.034$	3333	$\frac{11}{266} = 0.041$	1111	$\frac{27}{514} = 0.053$
Age 35					
1111	$\frac{83}{381} = 0.218$	1111	$\frac{33}{236} = 0.140$	2222	$\frac{42}{494} = 0.085$
2222	$\frac{43}{381} = 0.113$	2222	$\frac{25}{236} = 0.106$	3333	$\frac{42}{494} = 0.085$
1112	$\frac{16}{381} = 0.042$	3333	$\frac{16}{236} = 0.068$	1111	$\frac{28}{494} = 0.057$
Age 40					
1111	$\frac{60}{295} = 0.203$	1111	$\frac{25}{197} = 0.127$	2222	$\frac{38}{413} = 0.092$
2222	$\frac{27}{295} = 0.092$	2222	$\frac{16}{197} = 0.081$	1111	$\frac{28}{413} = 0.068$
3333	$\frac{13}{295} = 0.044$	3333	$\frac{8}{197} = 0.041$	3333	$\frac{28}{413} = 0.068$
Age 45					
1111	$\frac{30}{188} = 0.160$	2222	$\frac{14}{124} = 0.113$	4444	$\frac{26}{323} = 0.080$
2222	$\frac{17}{188} = 0.090$	1111	$\frac{11}{124} = 0.089$	1111	$\frac{22}{323} = 0.068$
3333	$\frac{11}{188} = 0.059$	4444	$\frac{8}{124} = 0.065$	2222	$\frac{21}{323} = 0.065$
Age 50					
3333	$\frac{11}{88} = 0.125$	1111	$\frac{12}{85} = 0.141$	3333	$\frac{35}{302} = 0.116$
2222	$\frac{8}{88} = 0.091$	2222	$\frac{6}{85} = 0.071$	4444	$\frac{26}{302} = 0.086$
1111	$\frac{7}{88} = 0.080$	4444	$\frac{5}{85} = 0.059$	2222	$\frac{21}{302} = 0.070$
Age 55					
2222	$\frac{13}{77} = 0.169$	3323	$\frac{6}{69} = 0.087$	4444	$\frac{47}{309} = 0.152$
1111	$\frac{8}{77} = 0.104$	1111	$\frac{5}{69} = 0.072$	3333	$\frac{36}{309} = 0.117$
4444	$\frac{6}{77} = 0.078$	2222	$\frac{5}{69} = 0.072$	2222	$\frac{22}{309} = 0.071$

See footnote 12 for details.

Table 4: Sequences, Women

References

- ABOWD, J., AND D. CARD (1989): "On the Covariance Structure of Earnings and Hours Changes," *Econometrica*, 57(2), 411–45.
- ADAMS, H., M. HURD, D. MCFADDEN, A. MERRILL, AND T. RIBEIRO (2003): "Healthy, Wealthy and Wise? Tests for Direct Causal Paths between Health and Socioeconomic Status," *Journal of Econometrics*, 112(1), 3–56.
- ADDA, J., J. BANKS, AND H. VON GAUDECKER (2009): "The Impact of Income Shocks on Health: Evidence from Cohort Data," *Journal of the European Economics Association*, 7(6), 1361–99.
- ALTONJI, J., AND L. SEGAL (1996): "Small-Sample Bias in GMM Estimation of Covariance Structures," *Journal of Business and Economic Statistics*, 14(3), 353–366.
- BAKER, M. (1997): "Growth-Rate Heterogeneity and the Covariance Structure of Life-Cycle Earnings," *Journal of Labor Economics*, 15(2), 338–75.
- BAKER, M., M. STABILE, AND C. DERI (2004): "What Do Self-Reported, Objective Measures of Health Measure," *Journal of Human Resources*, 39(4), 1067–93.
- BLUNDELL, R., AND I. PRESTON (1998): "Consumption Inequality and Income Uncertainty," *Quarterly Journal of Economics*, 113(2), 603–640.
- BORSCH-SUPAN, A., F. HEISS, AND M. HURD (2003): "Healthy, Wealthy, and Knowing Where to Live: Predicted Trajectories of Health, Wealth, and Living Arrangements among the Oldest Old," NBER Working Paper.
- CARRO, J., AND A. TRAFERRI (2010): "Correcting the Bias in the Estimation of a Dynamic Ordered Probit with Fixed Effects of Self-Assessed Health," Unpublished Mimeo.
- CASE, A., AND A. DEATON (2005): "Broken Down by Work and Sex: How Our Health Declines," in *Analyses in the Economics of Aging*, ed. by D. Wise. University of Chicago Press, Chicago, IL.

- CONTOYANNIS, P., A. JONES, AND N. RICE (2004a): "The Dynamics of Health in the British Panel Survey," *Journal of Applied Econometrics*, 19(4), 473–503.
- (2004b): "Simulation-Based Inference in Dynamic Panel Probit Models: An Application to Health," *Empirical Economics*, 29(1), 49–77.
- DEATON, A. (1992): *Understanding Consumption*. Oxford University Press, Oxford.
- DEATON, A., AND C. PAXSON (1994): "Intertemporal Choice and Inequality," *Journal of Political Economy*, 102(3), 437–67.
- (1998a): "Aging and Inequality in Income and Health," *American Economic Review: Papers and Proceedings*, 88(2), 248–253.
- (1998b): "Health, Income and Inequality over the Life-Cycle," in *Frontiers in the Economics of Aging*, ed. by D. Wise. University of Chicago Press, Chicago, IL.
- GROSSMAN, M. (1972): "On the Concept of Health Capital and the Demand for Health," *Journal of Political Economy*, 80(2), 223–55.
- GUVENEN, F. (2009): "An Empirical Investigation of Labor Income Processes," *Review of Economic Dynamics*, 12(1), 58–79.
- HALLIDAY, T. (2008): "Heterogeneity, State Dependence, and Health," *Econometrics Journal*, 11(3), 499–516.
- HECKMAN, J. (1981): "Statistical Models for Discrete Panel Data," in *A Structural Analysis of Discrete Data with Econometric Applications*, ed. by D. McFadden, and C. Manski. MIT Press, Cambridge, MA.
- HYSLOP, D. (1999): "State Dependence, Serial Correlation and Heterogeneity in Intertemporal Labor Force Participation," *Econometrica*, 67(6), 1255–94.
- LILLARD, L., AND R. WILLIS (1978): "Dynamic Aspects of Earnings Mobility," *Econometrica*, 46(5), 985–1012.
- MCEWEN, B., AND E. STELLAR (1993): "Stress and the Individual: Mechanisms Leading to Disease," *Archives of Internal Medicine*, 153(18), 2093–101.
- MEGHIR, C., AND L. PISTAFERRI (2004): "Income Variance Dynamics and Heterogeneity," *Econometrica*, 72(1), 1–32.

- NEWWEY, W., AND D. MCFADDEN (1994): "Large Sample Estimation and Hypothesis Testing," in *Handbook of Econometrics, Volume IV*, ed. by R. F. Engle, and D. L. McFadden. North-Holland, Amsterdam.
- PRIMICERI, G., AND T. VAN RENS (2009): "Heterogeneous Life-Cycle Profiles, Income Risk and Consumption Inequality," *Journal of Monetary Economics*, 56(1), 20–39.
- SEEMAN, T., B. SINGER, J. ROWE, R. HORWITZ, AND B. MCEWEN (1997): "Price of Adaptation - Allostatic Load and Its Health Consequences," *Archives of Internal Medicine*, 157(19), 2259–2268.
- SHAKOTKO, R. (1980): "Dynamic Aspects of Children's Health, Intellectual Development, and Family Economic Status," NBER Working Paper.