Income Volatility and Health

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Abstract

We investigate the impact of exogenous income fluctuations on health using twenty years of data from the Panel Study of Income Dynamics. To unravel the impact of income on health from unobserved heterogeneity and reverse causality, we employ techniques from the literature on the estimation of dynamic panel data models. Contrary to much of the previous literature on health and socio-economic status, we find that, on average, adverse income shocks lead to a deterioration of health. These effects are most pronounced for working-aged men and are dominated by transitions into the very bottom of the earnings distribution. We also provide suggestive evidence of an association between negative income shocks and higher mortality for working-aged men.

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1 Introduction

The relationship between economic circumstances and health or the gradient has been the subject of academic inquiry for quite some time. While these investigations have documented a strong positive correlation between socioeconomic status (SES) and health in a variety of contexts, they have failed to produce a consensus among scholars concerning the underlying causal pathways. Indeed, fierce debate has characterized the discussions among social scientists concerning the possible directions of causality with the dividing lines often being drawn between disciplines. Typically, on one side of the divide are the economists, who tend to champion the causal pathway from health to income (Smith 1999, Adams, Hurd, *et al.* 2002). On the other side of the divide are the public health experts and epidemiologists who tend to be advocates of the reverse causal pathway from SES to health (Marmot, *et al.* 1991, Marmot 2004). In this paper, we attempt to shed a new light on this debate by tackling the question of what happens to a person's health when they experience a shock to their income.

There are many possible pathways through which earnings fluctuations can impact health. The first and, perhaps most obvious, is that they might be accompanied by higher stress levels due to increased difficulty paying bills or providing for one's family. Within the context of a model of health investment *a la* Grossman (1972), this would be modeled by income directly entering the health production function. In addition, income fluctuations may also be related to a person's ability to finance medical care, for example, through a loss of employer-sponsored health insurance.

However, contrary to conventional wisdom, not all of these pathways suggest that an earnings shock should lead to a deterioration of health. For example, adverse shocks to employment might actually *improve* health since this would tend to relax time constraints and tighten budget constraints which would provide more leisure time that could be used to exercise and decrease the consumption of unhealthy vices (provided that they are normal goods). Indeed, Ruhm (2000; 2005) and Adda, Banks and von Gaudecker (2006) provide evidence for these "healthy living" mechanisms. In addition, while being unemployed might induce stress, working long hours and constantly being subject to the exigencies of the modern workplace is also a potential source of stress and stress-induced illnesses such as hypertension. Accordingly, the direction of the impact of an income shock on health is not *a priori* obvious and will largely depend on the relative magnitudes of these different effects. Moreover, these impacts may also depend on how the shock changes the individual's standing within the income distribution.

We employ data from the Panel Study of Income Dynamics (PSID) which offers a wealth of information which can be exploited to investigate these issues. To measure economic circumstance, we use data on labor income and county-level unemployment rates. Our health data are provided by measures of self-reported health status (SRHS) and the PSID's death file which provides a record of the deaths of all PSID respondents through 2003.

One primary advantage of the PSID is that its longitudinal structure allows us to use a rich literature on the estimation of dynamic panel data models. The estimation technique that we employ comes from Arellano and Bond (1991). It exploits moment conditions which allow health to impact labor supply in contemporaneous and future time periods. If valid, these conditions enable us to identify the causal impact of income shocks on health. One of the advantages of the PSID is that its length guarantees a large number of moment conditions which allow us to carry out specification tests that shed light on the validity of these restrictions. In addition, the procedure allows for individual-specific fixed-effects which can be arbitrarily correlated with the right-hand-side covariates which mitigates many concerns of omitted variables bias. While this and similar techniques have commonly been employed in labor economics (see Carrasco 2001, Hyslop 1999, Meghir and Pistaferri 2004, for just a few examples), these techniques are utilized with far less frequency in health economics. One notable exception, however, is Adda, Banks and von Gaudecker (2006) who employ panel data techniques and synthetic cohort data to investigate the impact of aggregate income shocks on mortality, morbidity and health behaviors.[1]

Using the Arellano-Bond estimator, we provide substantial evidence that, *on average*, an adverse income shock leads to a deterioration in health. These effects are largest for working-aged men, but we also find some weaker effects for working-aged women. For men, our estimated coefficient on income is large and is often equal and opposite the coefficient on age. These effects tend to be concentrated in the bottom part of the income distribution and appear to be dominated by transitions into a prolonged period of unemployment. In addition, despite finding that adverse income shocks lead to worse health outcomes *on average*, we also provide some evidence that movements from either the lower or the upper tail of the income distribution towards the middle are associated with *improvements* in health. This is suggestive of a story in which both unemployment and high earnings are associated with increased stress levels.

Using the PSID's mortality file, we provide suggestive evidence of an association between

[1]This study contrasts from their study in that we primarily focus on idyncratic income shocks whereas they focus on aggregate shocks.

negative income shocks and higher mortality for working-aged men. These mortality results are interesting since the bulk of the evidence that documents an association between economic booms and higher mortality relies on aggregate data, whereas our results rely on individual data. These aggregate studies may be biased due to recessions inducing out-migration and, thereby, lowering the measurement of mortality rates which is a point that we discuss at greater length in Section 4. Our individual level data, however, are not subject to such biases.

The balance of this paper is organized as follows. Section 2 discusses the data. In Section 3, we present our identification strategy and core results. In Section 4, we provide evidence on the relationship between income risk and mortality. Section 5 concludes.

2 Data

The data that we employ come from the PSID. Our sample includes variables on age, race, education, self-reported health status (SRHS), the unemployment rate in the respondent's county of residence, labor income and mortality. Because we are interested in income and employment shocks, we restrict our analysis to working-aged people which we define to be between 30 (inclusive) and 60 (exclusive) years old. Table 1 reports the summary statistics for all of the variables in our sample except for the mortality data.[2] The SRHS data that we employ span the years 1984 to 1997. The SRHS data are not available prior to 1984. The data on county level unemployment rates span the years 1984 to 1993. These data are not publicly available past 1993. The labor income data span the years 1978 to 1997. The reason for going back to 1978

[2]Note that because we include the Survey of Economic Opportunities in our sample, which we discuss in more detail later, these summary statistics may not be representative of the US population.

with these data is that it allows us to have more instruments when we employ the Arellano-Bond estimator later on in the paper. We did not employ data beyond 1997 because after this year the PSID sampled people every other year which creates complications when using dynamic panel data techniques. In addition, the PSID contains a sample of economically disadvantaged people called the Survey of Economic Opportunities (SEO). We include the SEO in our analysis.[3] Finally, we further restrict our analysis to heads of household and their spouses (provided that they are married) as the SRHS data are only available for these people.

Our primary measure of health is SRHS which is a categorical variable that takes on integer values between one and five and measures the respondent's assessment of their own health. A one represents the highest category and a five represents the lowest category. These measures, while subjective, do correlate extremely well with more objective measures of health. Numerous studies have shown that SRHS is informative of specific morbidities and subsequent mortality (Mossey and Shapiro 1982; Kaplan and Camacho 1983; Idler and Kasl 1995). In addition, Smith (2004) has used retrospective health measures from the PSID and shown that there is a tendency for people to downgrade their self-assessment of their own health when a new condition manifests.[4] For much of this analysis, we map the SRHS measure into two dummy variables: good health, which is turned on when SRHS is either a one or a two, and bad health, which is turned on when SRHS is either a four or a five. The omitted category is SRHS equal to three.

[3] There is little consensus within the profession about how one should deal with the SEO. Because it is selected on income and, thus, endogenous, conventional weighting schemes will not work. Accordingly, some people such as Lillard and Willis (1977) simply recommend dropping the SEO due to endogenous selection. Nevertheless, there are others such as Hyslop (1999) and Meghir and Pistaferri (2004) who include the SEO. The latter justify its inclusion on the claim that purging the model of the heterogeneity addresses the endogenous selection into the SEO. We follow these authors and include the SEO as well. Our reasons for doing so are twofold. First, like Meghir and Pistaferri (2004), we also purge fixed-effects from most of our estimations. Second, we primarily employ semi-parametric techniques which require a lot of data.

[4] We do not believe that the retrospective health measures would be well-suited for this paper due to problems associated with recall bias.

However, at times, we will also employ the full five point categorical variable.

We also employ mortality data from the PSID's death file which is considered sensitive and, thus, not publicly available. The death file contains mortality information on all individuals in the PSID from 1968 to 2003 who were known to have died prior to 2004.[5] However, because it is essential for our purposes to control for the individual's morbidity and because SRHS is not available prior to 1984, we only use death dates from 1984 to 2003. Figure 1 plots survivor functions from the PSID for men and women between the ages of 30 and 60. Both panels of the figure contains ten graphs which correspond to a year between 1984 and 1993. Each of these graphs takes all of the people in the sample of a certain age from a given wave of the survey and plots the percentage of these people who survived to each subsequent year through 2003. For example, the bottom graph in each panel corresponds to the base year 1984 and plots the percentage of people who survived until 1985, 1986, 1987, *etc.*[6]

Table 2 shows the results from estimation of Cox-Proportional hazard models to illustrate the relationship between SRHS and mortality in the PSID. We estimate the models using the 1984 wave of the PSID with the number of years that the individual survived subsequent to 1984 as the dependent variable. Our estimations use a sample of working-aged people. This table shows that SRHS is a strong predictor of mortality in the PSID and, thus, provides further evidence that these SRHS variables are very good measures of the respondent's health.

[5] Mortality information first comes from interviews with PSID families. PSID then corroborates this information with the National Death Index.

[6] It is important to note that our data show the stylized fact that women have lower mortality, as shown in Figure 1, and higher morbidity, as shown in Table 1. However, this does *not* suggest that the SRHS are of poor quality. Rather, it merely reflects that women tend to suffer from a different distribution of chronic ailments than men (Case and Paxson, 2005).

3 Income Volatility and Morbidity

To identify the impact of income shocks on health, we work with the dynamic model:

$$h_{i,t} = \alpha_i + \gamma h_{i,t-1} + y'_{i,t}\lambda + a_{i,t}\delta + \upsilon_{i,t}. \tag{1}$$

We let $h_{i,t}$ denote an indicator for bad health (*i.e.* SRHS is either four or five), $y_{i,t}$ denote a vector which includes labor income or functions of labor income and $a_{i,t}$ denote age.[7] We assume that the residual is mean zero and serially uncorrelated so that $E[\upsilon_{i,t}] = E[\upsilon_{i,t}\upsilon_{i,s}] = 0$ for $s \neq t$.[8] To purge the model of fixed-effects, we work with a first-differenced version of (1):

$$\Delta h_{i,t} = \gamma \Delta h_{i,t-1} + \Delta y'_{i,t}\lambda + \Delta a_{i,t}\delta + \Delta\upsilon_{i,t}. \tag{2}$$

Note that $\Delta a_{i,t}$ should be unity for most people and so δ can be interpreted as a measure of the depreciation of a person's health stock.

Equations (1) and (2) account for two important aspects of the theory of health investment. First, because equation (2) is purged of the fixed-effect, it allows for all time-invariant individual characteristics to be correlated with both health and earnings. This is important in light of the "Fuchs' Hypothesis" which states that heterogeneity in preferences and discount factors will generate a correlation between earnings and health even in the absence of any underlying causal relationships (Fuchs 1982). Accordingly, it is essential that the model is purged of these unobserved individual characteristics. Second, because we control for an individual's health

[7] We also expiremented with quadtratic functions of age. We found little evidence of non-linear age effects nor were our results affected. Accordingly, we stuck with the linear function of age.

[8] We will provide tests of the plausibility of the lack of serial correlation in $\upsilon_{i,t}$ later in the paper.

yesterday, we rule out any omitted variable biases that would result from a person's health yesterday feeding-back and impacting labor supply today. This is particularly important in light of Grossman's original health investment model in which sickness reduces a person's stock of "healthy time" which, in turn, constrains their ability to earn. In fact, the estimation procedure that we employ, which is discussed in the next sub-section, can be generalized to allow for, not only health yesterday, but also health today, to impact today's earnings.

Some readers may be inquiring why we are not employing a non-linear model. The first reason is that the linear model allows us to employ the estimation procedure discussed in Arellano and Bond (1991) which provides us with a tractable means of addressing both unobserved heterogeneity and the predeterminedness (or endogeneity) of income while requiring minimal assumptions on unobservables and no assumptions on the initial condition. The second reason is that this procedure comes with nice specification tests whose properties have been well-explored. The third is that (to our knowledge) the only procedure for the estimation of a non-linear discrete choice model with unobserved heterogeneity and predetermined regressors is Arelleno and Carrasco (2003). This procedure would be inappropriate for our purposes as it requires us to observe the complete history of outcomes for all individuals in our data which we do not. Failure to observe complete histories may result in an egregious mis-specification of the distribution of unobservables. For example, in the case of discrete regressors, a mixture of normal distributions would be mis-specified as a normal distribution.

3.1 Identification and Estimation

Identification of the parameters in equations (1) and (2) comes from two sets of moment conditions which exploit the time dimension of the data. Adopting the notation that $x_i^t = (x_{i,1}', ..., x_{i,t}')$, the strongest set of moment conditions that we employ is

$$E^*[\upsilon_{i,t}|h_i^{t-1}, y_i^t] = 0 \tag{P}$$

where $E^*[y|x]$ denotes the linear-projection of y onto x. We call this Assumption P because these moment conditions suppose that income and labor supply are predetermined variables. This condition assumes that health shocks today are uncorrelated with the history of health outcomes through yesterday and labor market outcomes through today. However, it allows for feedback in the sense that health today can impact labor market outcomes tomorrow. The weaker set of moment conditions that we work with is

$$E^*[\upsilon_{i,t}|h_i^{t-1}, y_i^{t-1}] = 0. \tag{E}$$

We call this Assumption E because, in contrast to Assumption P, it allows for a contemporaneous relationship between health and labor supply and, thus, treats income as an endogenous variable. Assumption E has the advantage that it imposes weaker assumptions on the data, but comes at the expense of reduced efficiency.[9]

To estimate the model, we use the GMM estimator outlined in Arellano and Bond (1991).

[9] For an excellent discussion of using these types of moment restrictions to identify dynamic linear panel data models, see Arellano and Honoré (2001).

The Arellano and Bond (AB) estimator applies Assumptions P and E to the first-differenced model in equation (2) and, thus, uses

$$E^*[\Delta \upsilon_{i,t} | h_i^{t-2}, y_i^{t-1}] = 0 \tag{3}$$

and

$$E^*[\Delta \upsilon_{i,t} | h_i^{t-2}, y_i^{t-2}] = 0 \tag{4}$$

as moment conditions. Equation (3) applies Assumption P to the first-differenced model and, thus, uses y_i^{t-1} and h_i^{t-2} as instruments for $\Delta y_{i,t}$ and $\Delta h_{i,t-1}$. Analogously, equation (4), which is implied by Assumption E, uses y_i^{t-2} and h_i^{t-2} as instruments for $\Delta y_{i,t}$ and $\Delta h_{i,t-1}$. We follow the recommendations of AB and report the parameter estimates from the one-step procedure. As we discussed in the data section, the SRHS data are not available prior to 1984 and, consequently, we can only use health as an instrument through that year. However, because data on labor income are available for the entire duration of the PSID, we employ data on income through 1978. We did not use data prior to 1978 because we did not expect income from 1977 or earlier to have much explanatory power for the first-difference in health for 1985 or later.[10]

3.2 The "Justification" Bias

[10] We investigated the possibility that these instruments are weak. Recent research has shown that when instrumental variables do not have sufficient explanatory power in the first-stage regressions, the finite sample distribution of the estimator can differ substantially from its asymptotic distribution (see Staiger and Stock (1994) and Bound, Jaeger and Baker (1995), for example). To look into this issue, we regressed $\Delta h_{i,t}$ and each element of $\Delta y_{i,t}$ on the vector, $(h_{i,t-2}, ..., h_{i,t-4}, y'_{i,t-2}, ..., y'_{i,t-4})$. The F-tests of joint significance of the regressors all had extremely low p-values and, thus, there was no indication that weak instruments was a problem. The results are not reported, but are available upon request.

At this point, a few words need to be mentioned about the "justification" bias in which people justify being jobless by claiming that they are in worse health than they actually are (Baker, Stabile and Deri 2004). This bias would generate a systematic correlation between the residual in our equation and our income measurement. We claim that Assumption E will mitigate (but not eliminate) problems with this bias.

To better see this, suppose that $h_{i,t}$ is the "true" health status and that $h^*_{i,t} = h_{i,t} + e_{i,t}$ is the reported health status. Clearly, the reporting error, which is denoted by $e_{i,t}$, is either equal to unity, zero or minus unity. If unemployed people justify their unemployment by claiming to be ill when they are not, then we have that $P\left(e_{i,t} = 1 | h_{i,t} = 0, y_{i,t}\right)$ is higher for unemployed people than employed people. Substitution into equation (2) then gives us that

$$\Delta h^*_{i,t} = \gamma \Delta h^*_{i,t-1} + \Delta y'_{i,t}\lambda + \Delta a_{i,t}\delta + \Delta v_{i,t} + \Delta e_{i,t} - \gamma \Delta e_{i,t-1}. \tag{5}$$

If the justification bias is large, then both Assumptions P and E will be violated, although the violation is more egregious with predetermined variables. The reason for this is that Assumption P will require orthogonality between the vector y_i^{t-1} and $\Delta e_{i,t} - \gamma \Delta e_{i,t-1}$. Assuming that the errors are serially uncorrelated, in the presence of the justification bias, the orthogonality conditions in Assumption P will not be met because $y_{i,t-1}$ and $y_{i,t-2}$ will be correlated with both $e_{i,t-1}$ and $\gamma \Delta e_{i,t-1}$. In contrast, Assumption E will use y_i^{t-2} as instruments. In the presence of serially uncorrelated errors and the justification bias, the conditions in Assumption E will not be met because $y_{i,t-2}$ will be correlated with $\gamma e_{i,t-2}$. This correlation should be smaller, but still not zero. This suggests that, relative to Assumption P, Assumption E will mitigate, but not eliminate, the justification bias and, thus, if this bias matters, we would expect our estimates to be smaller with Assumption E than with Assumption P.

3.3 Specification Tests

One of the primary advantages of the AB procedure is that the model's assumptions yield many moment restrictions which can be used to construct specification tests which shed light on the plausibility of the identifying assumptions of the model. AB propose two specification tests. The first test centers on the fact that when $\upsilon_{i,t}$ exhibits no serial correlation, we will have that $E[\Delta\upsilon_{i,t}\Delta\upsilon_{i,t-1}] \neq 0$ and $E[\Delta\upsilon_{i,t}\Delta\upsilon_{i,t-2}] = 0$. This specification test calculates the sample analogues of $E[\Delta\upsilon_{i,t}\Delta\upsilon_{i,t-1}]$ and $E[\Delta\upsilon_{i,t}\Delta\upsilon_{i,t-2}]$ to construct statistics that converge to a standard normal distribution. We follow the notation in AB and let m_1 denote the statistic that is based on $E[\Delta\upsilon_{i,t}\Delta\upsilon_{i,t-1}]$ and let m_2 denote the statistic that is based on $E[\Delta\upsilon_{i,t}\Delta\upsilon_{i,t-2}]$.[11] Calculation of m_1 is very important because if $\upsilon_{i,t}$ follows a random walk then we will have that $E[\Delta\upsilon_{i,t}\Delta\upsilon_{i,t-1}] = E[\Delta\upsilon_{i,t}\Delta\upsilon_{i,t-2}] = 0$. Consequently, it is possible for m_2 to be small even if $\upsilon_{i,t}$ exhibits a large degree of persistence. So, if the model is correctly specified and there is no serial correlation in $\upsilon_{i,t}$ then m_1 should be big and m_2 should be small. Further detail on the calculation of m_1 and m_2 can be found in AB (pp. 281 - 282).

The second specification test that we work with is the Sargan test of over-identifying restrictions (Sargan 1958; Hansen 1982). We use the two-step Sargan Statistic which is robust to heteroskedasticity.[12] We chose the two-step statistic over the one-step statistic because Monte Carlo experiments in AB suggest that there is a tendency for the non-robust test to over-reject

[11] In fact, AB can accommodate serial correlation in $\upsilon_{i,t}$ of the form MA(q) via weaker moment conditions. However, as it turns out, our calculations of m_2 suggests that such accommodation is not necessary.

[12] Unlike the Sargan Statistics, the specification test that uses m_1 and m_2 is defined in terms of any consistent estimator. In other words, the statistics m_1 and m_2 do not necessarily require the efficient two-step estimator.

and, thus, AB recommend placing more weight on the two-step statistic. The statistic is asymptotically chi-squared with degrees of freedom equal to the number of over-identifying restrictions in the model.

These tests will shed some light on the importance of the justification bias. First, if the moment conditions are valid then the tests will pass with a probability equal to one minus the size of the test (*e.g.* 95%). Thus, low p-values on these specification tests are a necessary (but not sufficient) condition for the validity of our moment conditions which crucially hinges on the relevance of the justification bias in our data. Second, the statistic m_2 will blow up if there are many reporting errors in health status. The reason is that, in the presence of reporting errors, this statistic will depend not on the correlation between $\Delta\upsilon_{i,t}$ and $\Delta\upsilon_{i,t-2}$ but, instead, on the correlation between $\Delta\upsilon_{i,t} + \Delta e_{i,t} - \gamma\Delta e_{i,t-1}$ and $\Delta\upsilon_{i,t-2} + \Delta e_{i,t-2} - \gamma\Delta e_{i,t-3}$ which is not zero even if $\upsilon_{i,t}$ and $e_{i,t}$ are not serially correlated.

3.4 Results

We estimated these models using twenty years of data which spanned the years 1978 to 1997. The income data spanned 1978 to 1997. The SRHS data spanned 1984 to 1997.[13] It is important to emphasize that, although the data for our main regression equation only date back to 1984, we still use additional income data from the years 1978 to 1983 as instruments.

Tables 3 and 4 report the AB estimates for working-aged men and women, respectively. The top panel uses Assumption P and the bottom panel uses Assumption E. The first two columns use bad health as the dependent variable. The last two columns use the five point categorical

[13] We did not employ data beyond 1997 because PSID started to survey households every other year after 1997. This would have created substantial complications when working with the AB estimator.

SRHS variable as the dependent variable. We concede that the linear model that we estimate does not allow for the ordinal nature of the five-point SRHS variable. However, it does have the advantage that it has more variation in the time-series than bad health which only changes when people move in or out of the bottom two SRHS categories. This longitudinal variation is extremely useful with fixed-effects estimation.

Table 3 provides evidence of a causal effect of income shocks on health outcomes for working-aged men. In the top panel, we see that the coefficient on labor income is negative and highly significant in columns 1 and 3. This indicates that positive income shocks tend to improve health outcomes, at least, on average. In columns 2 and 4 of the top panel, we see that the indicator for having zero labor income is positive and significant which suggests that movements into unemployment are bad for one's health. In the bottom panel, where we allow for endogenous regressors, we see that all of the income variables are still highly significant. However, what is interesting is that the magnitudes of the coefficients rise once we allow for a contemporaneous relationship between income and health. This may be a consequence of classical measurement error attenuating the estimates in the top panel. Also, as pointed out in Section 3.2, this is not what we would expect to happen if the justification bias was an important factor.

The specification tests in the bottom of both panels suggest that our moment restrictions hold up reasonably well in the data when the dependent variable is the binary indicator for having fair or poor health. Looking at the calculations of m_2 in the first two columns of the top and bottom panels, we see that we cannot reject the null that $E[\Delta \upsilon_{i,t} \Delta \upsilon_{i,t-2}] = 0$ at the 5% level in all four specifications. However, when we use the 5-point SRHS variable, the specification tests which are based on m_2 perform considerably worse. Next, in all eight specifications in the table, the

p-values on m_1 are all extremely low and, thus, always reject the null that $E[\Delta \upsilon_{i,t} \Delta \upsilon_{i,t-1}] = 0$ which rules out a unit root in the process for $\upsilon_{i,t}$. The two-step Sargan Statistic, which AB recommend, is not significant at the 1% level in columns 1 and 2 of the top panel and column 1 of the bottom panel and it is not significant at the 5% level in column 3 of the bottom panel. The Sargan statistic only has an extremely low p-value in the third and fourth columns of both panels. However, this is not shocking since the linear model is probably not the best way to deal with the five-point SRHS variable. Finally, it is important to mention that later on in this section we estimate some other models in which the specification tests perform better than in this table.

Table 4, which reports the results for working-aged women, is somewhat of a contrast to the previous table. Most of the specifications suggest that there is no relationship running from income to health. However, in columns 3 and 4 of the top panel and in column 3 of the bottom panel, there is some evidence that adverse income shocks are associated with worse health, although these coefficients are only marginally significant. The specification tests in the table perform pretty well when the dependent variable is the binary indicator for bad health. Overall, the table only provides weak evidence that adverse income shocks negatively impact the health outcomes of working-aged women.

We now investigate how transitions into and out of different parts of the income distribution affect health outcomes. To do this, we construct three dummies for belonging to particular quartiles of the income distribution. The first equals one when the respondent has a positive income, but falls below the 25th percentile. The second equals one when the respondent earns between the 25th and 50th percentiles. The third equals one when the respondent earns between the 50th and 75th percentiles. In addition, we use the dummy indicating that the respondent earned no income during the survey year. The omitted category is having an income above the 75th percentile. We estimate a variant of equation (1) which includes these four dummy variables. We employ Assumption E and, thus, treat each of these dummy variables as endogenous. The quartiles that were used to construct the dummies were calculated separately for men and women. Finally, because the 25th percentile of income for working-aged women was zero, we did not include it when estimating the models for women.

The results are reported in Table 5. What we now see is a far more complicated picture than what we saw in the previous two tables. In the first three columns, we observe that the zero income dummy is always positive, whereas the other dummies are always negative. This indicates that transitions from positive earnings to zero earnings are associated with a deterioration in health. This is consistent with the previous results in this section. However, a careful look at the table suggests that, among people with positive earnings, transitions to higher quantiles are actually associated with *worse* health outcomes. Indeed, the fact that the dummies for the 25th, 50th and 75th percentile dummies all have negative coefficient estimates suggests that transitions from having a positive income that falls below the 75th percentile to having an income that falls above the 75th percentile is actually bad for a person's health. It is also important to emphasize that the specification tests in this table perform exceptionally well when the binary indicator for bad health is the dependent variable. Finally, in contrast to Table 4, this table provides stronger evidence that income fluctuations impact women's health.

We conclude this section with a few cautionary notes on the proper interpretation of these results. First, the results in Table 5 show us how movements in and out of various parts of the income distribution affect health. However, the coefficients on the quartile dummies are not informative of the level of health in that quartile. For example, the fact that the 75th percentile dummy is negative indicates that moving from the 75th percentile to the top percentile is associated with a deterioration in health. It does not indicate that people with income in the highest quartile of the distribution have worse health than those in the second highest quartile. Second, there is no contradiction between the results in Table 5 and the results in Tables 3 and 4. Tables 3 and 4 indicate that an adverse income shock has a negative impact on health outcomes *on average*. Table 5 indicates that these average effects in the previous tables mask some more subtle effects. In particular, Table 5 suggests that most of the adverse impact of a negative income shock on health is dominated by transitions into unemployment, as opposed to transitions to lower, but positive incomes. Finally, Table 5 does provide some evidence that negative income shocks can be good for your health, although Tables 3 and 4 suggest that, on average, they are not.

4 Income Volatility and Mortality

In this section, we investigate the relationship between income and mortality in the PSID. Unfortunately, however, while a person's health status may change at numerous points during their life, a person's mortality only changes once. Accordingly, we can no longer rely on time-series variation and appropriate moment restrictions for identification. However, we can document some interesting correlations which may, at least to some extent, reflect an underlying causal relationship.

The model that we focus on is:

$$d_{i,t}^{j}=1\left(u_{i,t}\beta^{j}+x_{i,t}\theta^{j}+\alpha_{i}^{j}+\varepsilon_{i,t}^{j}\geq 0\right)\text{ for }j=1,2,3. \tag{6}$$

The dependent variables, $d_{i,t}^{1}, d_{i,t}^{2}$ and $d_{i,t}^{3}$, are dummy variables which indicate that the person has died within a one, three or five year window of the survey year. We employ different windows to account for the possibility that it might take varying lengths of time for the consequences of income shocks to manifest. The unemployment rate in the individual's county of residence is $u_{i,t}$. We focus on these unemployment rates rather than income as we find the former to be more plausibly exogenous than the latter.[14] To illustrate that movements in unemployment rates translate into income shocks, we report the results of fixed-effects regressions of income measures on the unemployment rate in Table 6. Not surprisingly, we see that increases in the unemployment rate have negative and significant impacts on income. The vector $x_{i,t}$ contains additional controls including controls for good (SRHS equal to one or two) and bad (SRHS equal to four or five) health. This is important as it mitigates (but does not eliminate) selection concerns that areas with high unemployment might be inhabited by unhealthy people due to the fact that healthier people are more likely to migrate out of depressed areas.[15] Time-invariant individual effects are modelled as α_{i}^{j} and individual-period specific effect are modeled as $\varepsilon_{i,t}^{j}$. We estimate the model with a random effects probit estimator.

The results are reported in Table 7 and are broadly in line with the rest of the results that we have presented. In the top panel, we report the results for working-aged men. We see that high

[14] We concede that there are also reasons to believe that macroeconomic conditions would be endogenous as well. One reason would be that people often migrate in response to the business cycle. Despite these concerns, we find the potential endogoneity issues with labor income to be far greater than with the unemployment rates.

[15] For a discussion of this, see Halliday (2007).

unemployment is positively associated with dying within one year of the survey year. However, there is no relationship between unemployment rates and dying within three or five years of the survey. In the bottom panel, we report the results for working-aged women and the story is very different. In the first and last columns, there is no relationship between unemployment rates and mortality. However, in the second column, we see that there is a *negative* and significant relationship between unemployment and mortality for women.

It is important to note that the results in the top panel of Table 7 contrast remarkably with many of the existing studies on mortality and recessions which utilize aggregate data. One possible reason for this may be measurement errors in mortality rates which are systematically correlated with measures of aggregate income shocks which would induce biases in the estimated relationship between mortality and recessions. Such biases would result because recessions are often accompanied by large out-migrations of people (see Blanchard and Katz, 1992, for example) and because mortality rates are measured with the population of the region *at baseline* as the denominator and the number of deaths that occur *during the time period* as the numerator. As a result, during a recession the number of deaths documented in a region may fall simply because there are fewer people within the region who could possibly die. This would, in turn, create a negative bias in the estimated relationship between unemployment and mortality rates in macro-level data.

5 Conclusions and Caveats

Employing twenty years of data from the PSID and the Arellano-Bond estimator, we provided evidence that, on average, adverse income shocks lead to a deterioration in health. This relationship was strongest for working-aged men. These effects appeared to be dominated by transitions into unemployment. In addition, we provided evidence that movements from the bottom and top tails of the income distribution towards the middle of the distribution lead to improvements in health. Finally, we provided some suggestive evidence that negative income shocks *might* lead to higher mortality for working-aged men.

It is important to place these findings within the context of some of the literature which has investigated causal pathways between SES and health. One of the most important papers on this topic is Adams, Hurd, *at al.* (2003) who investigate causality between wealth and health in a population of older Americans. They find no evidence of a causal link from SES to mortality and many morbidities, but they do reject the hypothesis of non-causality for some primary causes of death of older men such as cancer and heart disease.[16] In a related piece, Meer, Miller and Rosen (2003) use inheritance as an instrument for changes in wealth and find no evidence that health improves with exogenous increases in wealth. While it may be tempting to say that our research is at loggerheads with this earlier work, we do not believe that this is the case. It is true that we do provide some evidence that income shocks may have sizable impacts on the health of working-aged men at the bottom of the income distribution. However, this is, by no means, in contradiction with the assertion that exogenous changes in wealth (not income) do not influence health in a population of older people.

Some caveats on the limitations of this work deserve to be mentioned. First, it is not clear to what extent our estimates of the impact of labor income on self-reported health status translate into an impact on mortality. Given the results of Section 4, we believe that there may be

[16] For an interesting comment on this paper, see Adda, Chandola and Marmot (2003).

some effect on mortality, but the magnitude of this effect is hard to infer from this analysis. Second, due to the constraints of the PSID, the health measures that we employ are somewhat limited. However, one of the primary advantages of these measures is that they exhibit significant variation across time which enables the use of panel data methods such as the AB estimator. Without substantial time variation, as would be the case with measures of specific conditions such as diabetes and heart disease, these methods cannot be used.

Finally, while this work provides evidence that adverse income shocks lead to worse health outcomes, it is uninformative of the mechanisms by which this occurs. One possible mechanism that we discussed earlier is that negative income shocks are accompanied by increases in stress which, in turn, causes health to deteriorate. However, another potential mechanism is that negative shocks lead to a lower consumption of inputs in the production of health such as medical care. Indeed, the fact that the most dominant effects of income shocks on health that we uncovered occurred when people moved into unemployment and the fact that employer sponsored health insurance is the most common form of health coverage in the US suggests that this mechanism is worthy of serious consideration. Unfortunately, until recently, there has not been sufficient information in the PSID on medical insurance which (at least at this point) makes it immensely difficult to use the PSID to investigate this issue using dynamic panel data techniques.

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Table 1: Summary Statistics

	Definition	Mean (Standard Deviation) Men	Mean (Standard Deviation) Women
Age[1]	Individual's Age	40.95 (7.95)	41.14 (8.18)
White	= 1 if White	0.68 (0.46)	0.63 (0.48)
No College	= 1 if Individual Never Went to College	0.56 (0.50)	0.63 (0.48)
SRHS[1]	Self-Rated Health Status	2.30 (1.06)	2.47 (1.07)
Good Health[1]	= 1 if SRHS <= 2	0.60 (0.49)	0.52 (0.50)
Bad Health[1]	= 1 if SRHS >= 4	0.13 (0.33)	0.16 (0.37)
Unemployment Rate	County Level Unemployment Rate	6.28 (2.52)	6.39 (2.53)
Labor Income[2,3]	Individual's Labor Income	21932.41 (21819.00)	8999.45 (10106.46)
Zero Labor Income[2]	= 1 if Labor Income = 0	0.08 (0.27)	0.25 (0.43)

*All summary statistics correspond to the years 1984 - 1993 unless noted otherwise.
**Summary statistics are for people older than 30.
[1]These summary statistics correspond to 1984 - 1997.
[2]These summary statistics correspond to 1978 - 1997.
[3]Labor Income is in 1982 dollars.

Table 2: SRHS and Mortality in the PSID

	Men	Women
Age	1.075	1.087
	(10.32)	(10.85)
Good Health	0.637	0.747
	(-3.17)	(-1.64)
Bad Health	2.114	1.944
	(5.93)	(4.73)
Likelihood	-2170.32	-1896.37
N	2522	2875

*This table contains results from the Cox-Proportional Hazard model.

**Each cell reports the hazard ratio for an incremental change in a given variable.

***t-ratios correspond to the unreported coefficients for each variable.

****All estimations used a sample of people between 30 and 60.

Table 3: Arellano-Bond Estimates - Men Between Ages 30 and 60

	(1)	(2)	(3)	(4)
		Predetermined Variables		
Dependent Var	Bad[3]	Bad[3]	SRHS[4]	SRHS[4]
Lagged Health	0.101 (12.38)	0.098 (12.01)	0.101 (12.33)	0.097 (11.54)
Age	0.007 (4.89)	0.007 (5.01)	0.024 (5.91)	0.024 (5.83)
Zero Labor Income?[1]	-	0.038 (3.42)	-	0.137 (4.21)
Labor Income	-0.005 (-3.88)	-	-0.015 (-3.83)	-
m_1^2	-79.50 (0.000)	-78.76 (0.000)	-80.21 (0.000)	-76.82 (0.000)
m_2^2	0.47 (0.639)	0.28 (0.779)	3.07 (0.002)	2.86 (0.004)
Two-Step Sargan[2]	288.80 (0.014)	276.74 (0.043)	320.24 (0.000)	328.18 (0.000)
O.I. Restrictions	238	238	238	238
		Endogenous Variables		
Lagged Health	0.103 (12.52)	0.100 (12.05)	0.101 (12.39)	0.098 (11.65)
Age	0.007 (4.67)	0.007 (4.66)	0.023 (5.69)	0.022 (5.39)
Zero Labor Income?[1]	-	0.073 (2.15)	-	0.287 (3.01)
Labor Income	-0.009 (-2.24)	-	-0.024 (-2.15)	-
m_1^2	-76.50 (0.000)	-75.19 (0.000)	-77.17 (0.000)	-73.54 (0.000)
m_2^2	0.62 (0.535)	0.43 (0.668)	3.13 (0.002)	2.99 (0.003)
Two-Step Sargan[2]	270.81 (0.022)	254.27 (0.095)	306.84 (0.000)	319.74 (0.000)
O.I. Restrictions	226	226	226	226
N	6507	6507	6507	6507

[*]t-statistics reported below each coefficient estimate.

[1]Zero Labor Income? is an indicator which is turned on if labor income is zero.

[2]p-values in parentheses.

[3]The dependent variable is this column is an indicator that equals one when the person's health is either fair or poor.

[4]The dependent variable in this column is the 5-point SRHS variable.

Table 4: Arellano-Bond Estimates - Women Between Ages 30 and 60

	(1)	(2)	(3)	(4)
	Predetermined Variables			
Dependent Var	Bad[3]	Bad[3]	SRHS[4]	SRHS[4]
Lagged Health	0.080	0.081	0.068	0.066
	(10.65)	(10.83)	(8.96)	(8.69)
Age	0.010	0.010	0.023	0.023
	(6.97)	(6.99)	(6.30)	(6.31)
Zero Labor Income?[1]	-	0.001	-	0.031
		(0.19)		(1.51)
Labor Income	-0.000	-	-0.004	-
	(-0.03)		(-1.43)	
m_1^2	-85.11	-85.48	-82.06	-80.48
	(0.000)	(0.000)	(0.000)	(0.000)
m_2^2	2.12	2.18	3.18	3.10
	(0.034)	(0.029)	(0.002)	(0.002)
Two-Step Sargan[2]	273.40	245.42	348.33	315.95
	(0.057)	(0.357)	(0.000)	(0.001)
O.I. Restrictions	238	238	238	238
	Endogenous Variables			
Lagged Health	0.080	0.081	0.068	0.066
	(10.59)	(10.78)	(9.01)	(8.65)
Age	0.010	0.010	0.022	0.023
	(6.92)	(6.91)	(6.09)	(6.27)
Zero Labor Income?[1]	-	0.006	-	0.026
		(0.33)		(0.52)
Labor Income	0.000	-	-0.011	-
	(0.06)		(-1.70)	
m_1^2	-84.13	-84.69	-80.56	-79.85
	(0.000)	(0.000)	(0.000)	(0.000)
m_2^2	2.12	2.20	3.21	3.09
	(0.034)	(0.028)	(0.001)	(0.002)
Two-Step Sargan[2]	266.35	236.10	334.31	305.73
	(0.034)	(0.309)	(0.000)	(0.000)
O.I. Restrictions	226	226	226	226
N	7265	7265	7265	7265

*t-statistics reported below each coefficient estimate.

[1]Zero Labor Income? is an indicator which is turned on if labor income is zero.

[2]p-values in parentheses.

[3]The dependent variable is this column is an indicator that equals one when the person's health is either fair or poor.

[4]The dependent variable in this column is the 5-point SRHS variable.

Table 5: Arellano-Bond Estimates - Income by Quartile, People Between 30 and 60

	(1)	(2)	(3)	(4)
	Men		*Women*	
	Bad[3]	SRHS[4]	Bad[3]	SRHS[4]
Lagged Health	0.097	0.097	0.098	0.060
	(12.01)	(11.76)	(12.09)	(7.99)
Age	0.006	0.022	0.007	0.022
	(4.45)	(5.32)	(4.62)	(6.24)
Zero Labor Income?[1]	0.062	0.021	0.074	−0.049
	(1.97)	(0.24)	(2.58)	(−0.92)
Income > 0 and <= 25th Percentile	−0.025	−0.220	-	-
	(−1.36)	(−4.59)		
Income > 25th Percentile and <= 50th Percentile	−0.023	−0.266	−0.009	−0.114
	(−0.96)	(−4.20)	(−0.90)	(−3.05)
Income > 50th Percentile and <= 75th Percentile	−0.024	−0.152	−0.008	0.009
	(−1.11)	(−2.62)	(−0.54)	(0.23)
m_1^2	−78.08	−77.25	−77.15	−80.04
	(0.000)	(0.000)	(0.000)	(0.000)
m_2^2	0.24	2.84	0.35	2.85
	(0.811)	(0.005)	(0.729)	(0.004)
Two-Step Sargan[2]	672.64	739.51	500.34	641.85
	(0.497)	(0.038)	(0.765)	(0.000)
O.I. Restrictions	673	673	524	524
N	6507	6507	7265	7265

*This table assumes that all income and labor supply variables are endogenous.
**t-statistics reported below each coefficient estimate.
[1]Zero Labor Income? is an indicator which is turned on if labor income is zero.
[2]p-values in parentheses.
[3]The dependent variable is this column is an indicator that equals one when the person's health is either fair or poor.
[4]The dependent variable in this column is the 5-point SRHS variable.

Table 6: Macroeconomic Shocks and Labor Market Outcomes

	Labor Income	Zero Labor Income?[1]
1% Increase in Unemployment	−0.030	0.002
Men	(−5.05)	(3.34)
1% Increase in Unemployment	−0.028	0.003
Women	(−3.55)	(3.00)

*This table reports the coefficient on unemployment from fixed-effects regressions where the dependent variables are labor income and labor supply. All regressions contain a polynomial in age. The regressions where estimated using people between the ages of 30 and 60.
**t-statistics in parentheses.
***Each cell reports the effects of a 1 percentage point increase in unemployment on labor income and labor force participation.
[1]Zero Labor Income? is an indicator which is turned on if labor income is zero.

Table 7: Random Effects Estimates - Mortality

	(1)	(2)	(3)
	Men Between 30 and 60		
Death Occurred	$\leq$ 1 Year After	$\leq$ 3 Years After	$\leq$ 5 Years After
Age	0.026	0.122	0.176
	(8.31)	(8.26)	(15.23)
White	−0.183	−1.054	−1.126
	(−3.26)	(−7.02)	(−7.32)
No College	0.044	0.083	0.794
	(0.72)	(0.61)	(5.24)
Good Health	−0.089	−0.220	−0.536
	(−1.26)	(−1.80)	(−3.94)
Bad Health	0.568	0.801	0.544
	(8.25)	(6.34)	(4.61)
Unemployment Rate	0.037	0.002	0.022
	(4.19)	(0.12)	(1.13)
Likelihood	-1296.04	-1459.66	-1690.95
N	6315	6315	6315
	Women Between 30 and 60		
Death Occurred	$\leq$ 1 Year After	$\leq$ 3 Years After	$\leq$ 5 Years After
Age	0.094	0.187	0.200
	(5.83)	(13.28)	(17.03)
White	−1.001	−1.943	−2.345
	(−3.76)	(−10.14)	(−13.49)
No College	−0.203	0.155	0.119
	(−0.97)	(0.84)	(0.74)
Good Health	−0.275	−0.510	−0.496
	(−1.46)	(−2.84)	(−3.33)
Bad Health	0.836	0.977	0.824
	(4.96)	(6.02)	(6.01)
Unemployment Rate	−0.040	−0.059	−0.020
	(−1.25)	(−2.03)	(−0.87)
Likelihood	-735.27	-1026.04	-1228.14
N	6923	6923	6923

*This table contains results from random effects probits where the dependent variables are indicators for dying between the survey year and one, three and five years after.

**t-ratios correspond to the unreported coefficients for each variable.

Figure 1: Survivor Functions in the PSID