UNRAVELING THE SES HEALTH CONNECTION

James P. Smith

THE INSTITUTE FOR FISCAL STUDIES
WP04/02

January 2004

Unraveling the SES-Health Connection

James P. Smith

James P. Smith is a Senior Economist at RAND. This research was generously supported by grants from the National Institute on Aging.

People of lower socio-economic status (SES) appear to always have much worse health outcomes[1]. No matter which measures of SES are used or how health is measured, the evidence that this association is large and pervasive across time and space is abundant (Marmot 1999; Smith 1999). To document its principal features, Figure 1 displays the main contours of the SES health gradient in America by plotting at each age the fraction of people who self-report themselves in excellent or very good health by age-specific household income quartiles. Figure 2 has the same structure but the outcomes now represent those in poor or fair health.

At least until the end of life, at each age every movement down in income is associated with being in poorer health. Moreover, these health differences by income class can only be described as dramatically large. The fraction in excellent or very good health in the top income quartile is often 40 percentage points larger than the fraction in those health groups in the lowest income quartile. In both Figures 1 and 2, there also exists a strong non-linearity in the relation between income and health, with the largest health differences taking place between the lowest income quartile and all the others. Since this non-linearity will turn out to be important in resolving some of the key issues surrounding the SES health gradient, I shall return to it below. Finally, there is a distinct age pattern to the SES-health gradient with health disparities by income class expanding up to around age 50, after which the health gradient slowly fades away. This age pattern will also be critical later in this paper.

There actually is a reasonably broad consensus about the "facts" and about what the key scientific and policy questions surrounding the SES health gradient are—only the answers are controversial. Do these large differences in health by SES indicators such as income largely reflect causation from SES to health, as most non-economists appear to believe? Medical

[1] Socioeconomic Status (SES) is defined as any one of several composite measures of social rank, usually including income, education, and occupational prestige.

scientists are often convinced that the dominant pathway is that variation in socioeconomic status produces large health disparities; their main debate is about why low economic status leads to poor health (Marmot 1999). Important recent and often insightful contributions by these scholars has involved investigations of the influence of other factors besides access to high-quality health care or deleterious personal behaviors, both of which are believed to offer incomplete explanations. These contributions have emphasized instead long-term impacts of early childhood or even inter-uterine environmental factors (Barker 1997), the cumulative effects of prolonged exposures to individual stressful events (Seeman et al. 1997), or reactions to macro-societal factors such as rising levels of income inequality (Wilkinson 1996), and discrimination (Krieger 1999).

While that debate rages on about competing reasons why SES may affect health, there is little recognition that the so-called reverse causation from health to economic status may be pretty fundamental as well. Even if the direction of causation is that SES mainly affects health, what dimensions of SES actually matter—the financial aspects such as income or wealth or non-financial dimensions like education? Finally, is there a life course component to the health gradient so that we may be mislead in trying to answer these questions by only looking at people of a certain age—say those past 50.

This paper, which is divided into four sections, provides my answers to these questions. The first section examines the issue of reverse causation or whether a new health event has a significant impact on four dimensions of SES—out-of-pocket medical expenses, labor supply, household income, and household wealth. The next section switches the perspective by asking whether the so-called direct causation from SES to health really matters all that much. If the answer is yes and it will be, a sub-theme in this section concerns which dimensions of SES—

income, wealth, or education—matter for individual health. Since the answer to that question turns out to be education, Section 3 deals with the very much more difficult issue of why education matters so much. The evidence in these first three sections relies on data for people above age 50.

Figures 1 and 2 suggest that the nature of the SES-health gradient may be quite different after than before 50. In the final section of the paper, I test the robustness of my answers to these basic questions of the meaning of the SES-health gradient using data that span the entire life-course.

Section 2—Does Health Affect SES?

The primary focus among epidemiologists and those in health research community more generally has been on understanding and disentangling the multiple ways in which socio-economic status may influence a variety of health outcomes. Consequently, much less is known about the possible impacts health may have on SES. But for many individuals, especially those middle aged, health feedbacks to labor supply, household income, or wealth may be quantitatively important. In this section, I will explore this question by estimating the effect of new health events on a series of subsequent outcomes that are both directly and indirectly related to SES. The outcomes investigated will include out-of-pocket medical expenses, labor supply, household income, and wealth.

Before summarizing those results, it is useful to first outline the essential issues in estimating effects of SES on health as well as the effects of health on SES. Current realizations of both economic status and health reflect a dynamic history in which both health ($H_{t)}$ and SES (Y_t) are mutually affected by each other as well as by other relevant forces. Most of the relevant ideas can be summarized by the following two equations:

(1) $H_t = \alpha_0 + \alpha_1 H_{t-1} + \alpha_2 Y_{t-1} + \alpha_3 \Delta Y_t + \alpha_4 X_{t-1} + u_{1t}$

(2) $Y_t = \beta_0 + \beta_1 H_{t-1} + \beta_2 Y_{t-1} + \beta_3 \Delta Ht + \beta_4 X_{t-1} + u_{2t}$

where X_{t-1} represents a vector of other possibly non-overlapping time and non-time varying factors influencing health and SES u_{1t} and u_{2t} are possibly correlated stochastic shocks to health and SES. The key parameters α_3 and β_3 measure the effects of new innovations of SES on health and health on SES respectively. In this framework, we can also estimate whether past values of SES predict health ($\alpha_2 \neq 0$) or past values of health predict SES ($\beta_1 \neq 0$).[2]

While cross-sectional data can shed light on these issues (and I utilize such data below), there are advantages to examining questions of causation with panel data. To estimate the "effect" of either on the other (α_3 and β_3), we require exogenous variation in health (or SES) that is not induced by SES (health). In an earlier paper (Smith 1999), I proposed one research strategy for isolating new health events—the onset of new chronic conditions. While to some extent people may anticipate onset, much of the actual realization and especially its timing may be unanticipated. While new onsets may provide the best chance of isolating health shocks, not all new onset is a surprise. A set of behavioral risk factors and prior health or economic conditions may make some people more susceptible than others to this risk. Thus, predictors of new onsets should be included in models to increase one's confidence that the remaining statistical variation in new onsets is "news." I will make a similar point in the next section in discussing the impact of SES on health.

A new health event in one year may affect medical expenditure, labor supply and income not only in the year in which the event occurred but in future years as well. For example, the onset of a new condition may induce only single period changes in labor supply after which labor

[2] For an insightful debate about the conditions under whether coefficients are zero or stationary also reveals something about causality, see the paper by Adams et al. (2003) and the comments on that paper in the same volume.

supply may stabilize. But it is possible that spillover effects of a health shock may further depress work effort in future years or alternatively some recovery to original levels may take place. One way of estimating such patterns is to estimate a series of four equations for each of HRS waves 2-5 summarizing changes in each outcome between adjacent waves (say labor supply L_t) as

$$\Delta \boldsymbol{L}_t = \alpha \boldsymbol{X} + \sum_{t-2}^{5} \beta_t \Delta \boldsymbol{H}_t$$

where L_t is the between-wave change in labor supply and H_t the within-period health event from period t to t-1. Similar equations would apply for household income, out-of-pocket medical expenses, and other outcomes. If there is only contemporaneous one period effect of health events, all lagged values of changes in health will be zero.

The research I summarize here uses the first five waves of data on health status and transitions, medical expenses, labor supply, income, and wealth accumulation from the Health and Retirement Survey (HRS). HRS is a national sample of about 7,600 households (12,654 individuals) with at least one person in the household 51-61 years old originally interviewed in the fall of 1992 and winter of 1993. The principal objective of HRS is to monitor economic transitions in work, income, and wealth, as well as changes in many dimensions of health status. Follow-ups of HRS respondents were fielded at two-year intervals. HRS instruments span the spectrum of behaviors of interest; on the economic side, work, income and wealth: on the functional side, health and functional status, disability, and medical expenditures.

In addition to its excellent array of economic variables, HRS measured many different aspects of respondents' health. These included self-reports of general health status, the prevalence and incidence of many chronic conditions, the extent of functional limitations, and out-of-pocket

and total health care expenditures. The chronic diseases asked about include hypertension, diabetes, cancer, chronic lung, heart problems (e.g., heart attack, angina, coronary heart disease, congestive heart failure), stroke, and arthritis. In addition, risk behaviors include current and past smoking, current and past drinking, self-reported height and weight (BMI), and exercise.

To calculate the impact of the onset of new health events, I estimated a parallel set of models predicting out-of-pocket medical expenses, changes in labor supply, and changes in household income. A vector of baseline attributes is included in all models, including baseline measures of birth cohort (or age), marital status, race, ethnicity, education, region of residence, quintiles of family income and most importantly an extensive vector of measures of baseline health. These health measures include dummies for four of the five categories of self-reported health status, the presence at baseline of each chronic condition, a set of behavioral risk factors (smoking, exercise, BMI, drinking) and a scaled index of functional limitations based on the answers to the ADL questions.

I divided new health shocks in two categories—major (cancer, heart disease, and diseases of the lung) and minor (all the rest). My results for health shocks that took place between the first and second wave of HRS are summarized in Table 1.[3] The columns represent the principal outcomes of interest (medical expenses, labor supply, and household income) while the rows trace the evolving impact of this health shock across the HRS waves. The final row summarizes the full impact of the health event across all five waves.

A severe health shock that occurred between waves 1 and 2 of HRS initially increased mean OOP medical expenses by $1,720 during the two-year interval when it happened. This same health event also produced future increases in health costs that were of progressively

[3] Health shocks that took place between the other HRS waves had similar types of effects and thus are not repeated here. See Smith (2003) for more details on the full set of impacts.

smaller amounts. By the fifth wave, the mean total cost was a little over $4,000 so that less than half of the incremental costs were borne around the time of the event. All of these impacts on OOP medical expenses are much smaller when the health event was minor.[4]

Similar to the time pattern of effects documented for OOP medical expenses, a new severe health onset has an immediate and large impact of reducing the probability of working, followed by diminishing ripple like effects in subsequent waves. To illustrate, a severe health event between the first and second wave of HRS reduced the probability of work by 15 percentage points between the same two waves. Since the average labor force participation rate at baseline among those who were about to experience this major health event was .55, the impact on work is decidedly not trivial. Once again, estimated incremental effects in subsequent years cascade downward so that by the end of HRS wave five, the probability of work had declined by about 27 percentage points due to a major health shock between waves 1 and 2. Just as was reported for medical costs, estimated effects are considerably smaller if the health events come under the minor label.

Not surprisingly given the labor force results just described, new health events also depress household income with the reduction larger when the shock is major. There is no evidence of any household income recovery in subsequent years so that the initial income losses persist. In fact, consistent with the labor force participation effects, there are additional diminishing income losses in subsequent waves. The final row in Table 1 presents the total change in household incomes loss associated with the health event. On average, by the end of wave 5 total household income is about $6,300 lower when a major health event was

[4] The estimates in Table 1 summarize mean impacts. Effects of new health shocks on the tails of the out-of-pocket medical expense distribution were much larger (see Smith 2003).

experienced between the first and second wave of HRS—the comparable estimate for a minor health event was about $1,700.

Income losses that persist over time can eventually accumulate into large sums indeed. The first rows in Table 2 (for major health events) and Table 3 (for minor health events) contain my estimates of the cumulative income loss associated with the onset of health events occurring between the HRS waves. To illustrate, by wave 5 a health event that took place between the first two waves of HRS led to a total loss in household income of almost $37,000. It is important to note that these losses in household income are typically far larger than any cumulative out-of-pocket medical expenses associated with the health event. For example, for the wave 1-2 major health shock, the OOP medical expenses of about $4,000 are only one-ninth of the total household income loss. While less dramatic for the severe health shocks that took place between the other waves of HRS, cumulative income losses typically exceed cumulative medical expenses by a large single digit integer. Once again, cumulative household income losses are much smaller when the health event is minor, but even in this case income losses far exceed the additional medical expenses.

Table 2 also includes the same summary measures of household income loss and cumulative medical expenses obtained from the same models estimated using the original AHEAD sample, a sample of respondents who were at least age 70 at baseline. Given the predominance of retirement and virtually universal coverage by Medicare in the AHEAD sample, not surprisingly changes in household income and OOP medical expenses triggered by a new health event whether major or minor are considerably smaller in the AHEAD sample. There is much less possibility of any income loss since most AHEAD respondents' income is either annutized through social security or private pensions and thus is not contingent on changes in

health status (Smith and Kington 1997). These much smaller feedbacks from health to several SES measures in AHEAD serve as a warning that the magnitude of any casual effects from health to SES may vary a good deal over the life course. I return to that issue in Section 4.

The lifetime budget constraint linking consumption, income, wealth, and savings implies that this sum of period-by-period income loss plus medical expenses (adjusted by the foregone interest on this money) represents one way of measuring the wealth change or dis-savings that took place across the first five waves of HRS due to the health shocks.[5] This measure of lost wealth due to a health event is listed in the second rows of Tables 2 and 3. My estimates of the reduction in wealth due to a new health event are not trivial—for a new major health shock between the first and second HRS wave it is almost $50,000. Given the much smaller income losses involved, estimated wealth losses are considerably smaller when the health events are minor and when estimated over the older AHEAD sample.

What then have we learned? First, at least among people in their fifties, pathways from health to the financial measures of SES are decidedly not trivial. Especially as time unfolds, new health events have a quantitatively large impact on work, income, and wealth. This pathway should not be viewed as a sideshow to the main event and dismissing or ignoring it misses a good deal of the relevant action. Second, the principal risk people face when poor health arrives is not the medical expenses they must pay but rather the currently not fully insured loss of work and income. Finally, not all health events are unlike. I estimate quantitatively quite different effects of the health events labeled major compared to the minor ones. More research is needed on how to best combine and differentiate new health events.

[5] The only component not included in this wealth loss measure is any change in household consumption other than medical expenses. Smith (1999) outlines the conditions by which other total household consumption increases or decreases due to a new medical event.

Section 3—Does SES Affect Health?

Finding evidence of significant feedbacks from new health events to several key SES measures does not negate the likelihood that the probability of experiencing an onset of a new health event may not be uniform across several SES dimensions. The pathway from SES to health will be explored by examining whether the future onset of new chronic conditions is related to levels of household income, wealth, education, once one conditions of a set of pre-existing set of demographic and health conditions.[6] I will also explore the extent to which innovations in economic status "cause" health.

These models once again include as covariates a vector of baseline health conditions of the respondent—self-reported general health status, the presence of a chronic condition at baseline, and the extent of functional limitations scale. The models also include a standard set of behavioral risk factors (currently a smoker, number of cigarettes smoked), whether one engaged in vigorous exercise, and BMI and a standard set of demographic controls—birth cohort, race, ethnicity, sex, and region of residence. My main interest, however, lies in the SES measures that include household income, baseline levels of and changes in household wealth, and respondent's education. [7]

Just as one needed innovations in health that were not caused by SES to estimate the impact of health on SES, it is also necessary to isolate innovations in SES that were not caused by health to estimate the impact of SES on health. One opportunity for doing so lies in the large

[6] An important controversy that has occupied a substantial part of the recent literature has investigated the hypothesis attributed to Wilkinson (1996) that societal levels measures such as income inequality affect individual-level health. For an excellent review and critique of the theoretical and empirical literature on this hypothesis see Deaton (2003). Deaton concludes that at least in the U.S. and UK there is little empirical support for this view, at least as when it is confined to income inequality per se.

[7] Since the sample is restricted to those who were in the HRS for all five waves, this analysis ignores the relationship of SES with attrition and mortality. Given the age range of HRS and PSID respondents, mortality selection is unlikely to be that critical. That is clearly not the case in the AHEAD sample. For a model that incorporates mortality selection see Adams et al. (2003).

wealth increases that were accumulated during the large stock market run-up during the 1990s. Given the unusually large run-up in the stock market during these decades, it is reasonable to posit that a good deal of this surge was unanticipated and thus captures unanticipated exogenous wealth increases that were not caused by a person's health. If financial measures of SES do improve health, such increases in stock market wealth should be associated with better subsequent health outcomes at least with a lag.[8]

Knowing which aspect of SES affects health is key to the policy debate that surrounds the issue of the SES-health gradient. For example, consider the extreme where all pathways from SES to health operate through education and none through the primary financial measures of SES income or wealth. If that were so, then policies directed at income redistribution while perhaps desirable on their own terms could not be justified in terms of any beneficial impact on health. Combining all dimensions of SES into a single construct basically precludes discussion of most of the policy relevant options.

The results from these models are reported in Table 4 and are provided for onsets of major and minor conditions and for each chronic disease separately. A consistent generalization can be made for household income—it never predicts future onsets of either minor or major health conditions. In no single case is the estimated coefficient on household income (which vacillates in sign) statistically significant. While the coefficients on wealth lean towards negative values (5 out of 7), in only one case (stroke) is a statistically significant negative result obtained for household wealth. Finally, my best measure of an exogenous wealth change—the wealth increase from the stock market—is only statistically significant in one instance (arthritis) and there it has the incorrect sign so that an increase in stock market wealth makes the onset of

[8] One limitation of using increases is stock market wealth is that these increases are concentrated at the top of the income distribution (see Smith 2000). Obtaining other believable measures of exogenous changes in financial resources that more evenly span the entire income distribution would be very useful.

arthritis more likely. In sum then, SES variables that directly measure or proxy for financial resources of a family are either not related or at best only weakly related to the future onset of disease over the time span of eight years.

This largely negative conclusion is in sharp contrast to the results obtained for the final SES measure—education. Additional schooling is strongly and statistically significantly predictive of the new onset of both major as well as minor disease over the first five waves of the HRS. In all cases except cancer (which looks very much like an equal opportunity disease), the effects of schooling are preventative against disease onset.

This moves us to the most perplexing question of all—why does education matter so much in the promotion of good health? To provide some insight on this question, I ran expanded versions of these models that included proxies for some of the most frequently mentioned reasons about why education might matter. The proxies available in the HRS included measures of cognition and memory, past health behaviors such as smoking and drinking, job-related environmental hazards, early life health outcomes and economic environments, parental education, and parental health.[9]

Within this list of expanded variables, the only ones that really mattered in terms of their own statistical significance and in reducing the size of the effects of education were the current self-evaluation of childhood health and economic status and parental health as measured by age of death of each parent. These results are summarized in Table 5. For the major health onsets, a (currently) self-assessed better health status and better economic status during childhood both

[9] HRS data on some concepts is limited but it does record whether one smoked in the past and whether one was exposed on the job to a health hazard (and the number of years of exposure), the education of parents, whether or not each parent is alive, and if deceased the age of death, self-accessed general health status as a child (the same five point scale) and an assessment of the economic environment in which one lived during childhood. The specific question for health was "Consider your health while you were growing up, from birth to age 16. Would you say that your health during that time was excellent, very good, good, fair, or poor?" The specific question for economic circumstances was "Now think about your family when you were growing up, from birth to age 16. Would you say your family during that time was pretty well off financially, about average, or poor?"

reduce the risk of incurring a serious health onset in one's fifties and early sixties even after controlling for current health and economic status. In its support for the delayed health impact of early childhood exposures, these results are consistent with the spirit of the research reported by Barker (1997), although his specific hypotheses related to the intra-uterine environments. In the minor onset specification in Table 5, measures of parental health make a difference. Having a living parent or having a parent being older when they died tend to reduce the likelihood of an onset on new chronic conditions at these ages. Whether this association between parental health and health during one's fifties reflects genetic factors, shared household economic and health environments during childhood, or something else would be speculative at the stage of our knowledge. Since the impact of education still remains after including these variables in Table 5, my overall conclusion would be that collectively these additional factors explain some but by no means all education's ability to predict future health onsets.

Another clue to why education may be so critical concerns the role education plays in self-management of disease (Goldman and Smith 2002). A positive trend in recent decades has been the development of many new effective therapies for disease management. While clearly beneficial, these therapies can often be quite complicated and difficult for patients to fully adhere to and consequently for many diseases adherence rates are often alarmingly low. The question Goldman and I asked was what role education played in self-management.

I will illustrate our findings with one of the diseases we investigated—diabetes.[10] New treatments for diabetes are known to be efficacious but the treatment is complicated and places great demands on a patient's ability to self-monitor his condition. One study we did was based on an important clinical trial—the Diabetes Control and Complications Trial (DCCT). In the DCCT patients with type 1 diabetes were randomized into treatment and control groups. The

[10] We found similar results for patients with HIV.

treatment arm involved a quite intensive regimen where there was very close self and external monitoring of blood glucose levels and encouragement of strict adherence. In particular, patients in the treatment arm were seen weekly until a stable treatment program was achieved. While not insignificant, the treatment in the control arm consisted of a more standard regimen and far less intrusive external monitoring of patients.

Table 6 shows that before the intervention there were large differences across education groups in several measures of good behaviors at the study's baseline. Whether it involved checking blood, following insulin regimens, exercise, or smoking, those with less education were not doing as well. Given these initial but unsurprising baseline differences by education in adherence to good practice, we hypothesized that imposing a good behavior regimen—which is essentially what the rigorous treatment regimen did—would impart more benefits to the less educated who were having more problems with treatment to begin with.

We used an objective health outcome measure in the DCCT—glycosolated hemoglobin, which measures the amount of sugar binding to the blood. Higher levels indicated worse control. The impact of enforcing a common treatment regime can be obtained by subtracting what normally would occur (the control sample) with what took place under an enforced treatment regimen (the treatment sample). The data in Table 7 demonstrate that while those in all education groups benefited from being in the treatment arm, the benefits from enforced better adherence relative to the control group were largest for the least educated (see the final row in Table 7). Thus, a differential ability to adhere to beneficial albeit complicated medical regimens appears to be one reason for the association between education and health outcomes for the chronically ill.

In our study, Goldman and I were also able to provide some evidence on why education might matter for adherence. Once again, two factors that did not matter in promoting better adherence were household income or having a better memory. Alternatively, it does appear to be related to higher-level aspects of abstract reasoning, part of which included the ability to internalize the future consequences of current decisions.

Additional research on why education matters so much should receive high priority. One possibility is that the education experience itself has little to do with it, but it is simply a marker for personal traits (reasoning ability, rates of time preference, etc.) that may lead people to acquire more education and to be healthier. But education may not be that passive. It may help train people in decision-making, problem solving, and adaptive skills, all of which have pretty direct applications to a healthier life. Education may well have biological effects on the brain, which result in improved cognitive function and problem-solving ability some of which may impart benefits to choices made regarding one's health. This is similar to the argument that more active brain functioning when younger pushes off the onset of dementia.

Section 4—The SES-Health Gradient and the Life-Course

The steady negative progression in health and disease as we age is well established. Long before age 51—the minimum age entry point for the HRS samples on which the previous analyses are based, there has taken place a slow but accelerating decline in average health status. What is less well established is the shape of the SES-health gradient across age groups. Imagine for a moment that all we knew about the SES-health gradient is what the AHEAD sample (originally those over age 70) or the HRS sample (originally those ages 51-61) was able to tell us. In Figures 1 and 2, in the AHEAD sample we would only observe that portion of the graph above age 70, which is demarcated by the vertical solid line at that age. While we would begin

with an income-health gradient among the youngest AHEAD respondents, what we really would be monitoring should more properly be called a story of the demise of the gradient. Indeed, among the oldest AHEAD respondents, there is hardly any income gradient to health at all.

Since we are mostly in a world of disappearing health differences with income, perhaps it should not be much of a surprise in this sample that income does not impact health. When we add the age groups contained in the other HRS cohorts so that the data would consist of the gradient past age 50 (indicated by another vertical solid line), the income gradient with health certainly stands out more clearly. But all we might really have done is to add additional ages to our tale of the demise of the health-income gradient.

We know from Figures 1 and 2 that ages before 50 are very much the mirror image (now expanding with age) of what happens subsequently. It is a legitimate question to ask whether conclusions drawn about the meaning of the SES-health gradient over ages during which the gradient is withering away will generalize to the whole life course, especially to those ages during which it is emerging.

To address this question, I will first use the Panel Study of Income Dynamics (PSID), which has gathered almost 30 years of extensive economic and demographic data on a nationally representative sample of approximately 5000 (original) families and 35,000 individuals living in those families. The PSID is recognized as the premier general-purpose survey measuring several key aspects of SES. Details on family income and its components have been gathered in each wave since the inception of PSID in 1967. Starting in 1984 and in five-year intervals until 1999, PSID has asked a set of questions to measure household wealth.

The PSID has not traditionally been known as a health survey, but it has been collecting information on self-reported general health status (the standard five point scale from excellent to

poor) since 1984. Starting in 1999 and for all subsequent waves, PSID has obtained information on the prevalence and incidence of a list of chronic conditions for the respondent and spouse—heart disease, stroke, heart attack, hypertension, cancer, diabetes, chronic lung disease, asthma, arthritis, and emotional, nervous, or psychiatric problems. In addition to the prevalence in 1999, individuals were asked the date of onset of the condition as well as whether it limited their normal daily activities. Keeping in mind issues related to recall bias, the time of the onset of a health shock can be identified and the impact of these new health events on labor supplies, income, and wealth can be estimated.

The PSID offers several key additions to the research agenda. First, as the data provided in Figure 1 suggest, the nature of the SES health gradient may vary considerably over the life cycle. In contrast to HRS, PSID spans all age groups allowing us to examine behavior over the complete life cycle. Labor supply effects induced by new health events may be particularly sensitive to life-cycle stage as for shocks that take place in the late mid-50s or earlier 60s individuals have the option of selecting an option that they would have chosen in a few years anyway—retirement. Second, the long-term nature of the PSID allows one to estimate the impact of health and SES innovations over very long periods of time—even decades. It may well be that health responds to changes in financial measures of SES but only after a considerable lag.

Table 8 displays information on onsets of major and minor chronic conditions in four age groups—those less than 40, ages 41-50, ages 51-61, and those over 61 years old. Onsets during the previous 15 years are placed into three five-year windows-1994-99, 1989-93, and 1984-88. Both in cross-section (reading across a row) and within cohort (reading down a column) disease onset increases rapidly with age. While less common than for those in the HRS age ranges,

health episodes for PSID respondents less than 50 years old are not negligible. Among those in their 40's in the 1999 wave, one in seven had previously had a major disease onset at some time in their lives and 40 percent have a minor chronic health condition. In the five years before 1999, 7 percent of these 40-year olds experienced a major disease onset while one in four reported a new minor onset.

Table 9 lists the estimated impacts of a new major health onset that took place between 1995-1999 on three outcomes—the probability of continuing to work, the change in household income, and the change in household net worth. To detect the possibility of an age pattern, the impacts of the major health events are presented for three age groups—those less than 51, those 51-61, and those over 61 all measured in 1994.[11] The most unambiguous results apply for labor supply where the largest impact of a new severe health shock takes place amongst those in the 50's or early 60's. This may not be surprising since people in the pre-retirement years may be simply quickening the inevitable—the movement into retirement. While there are legitimate questions about robustness of results since income and household wealth are much harder to measure, it also appears that the largest impact on family income and wealth also takes place amongst those 51-61 years old. The offsetting factor to this ranking may be that diseases onsets that begin at a younger age are with you longer so that their impacts, while smaller when measured in a set time interval, have the potential to grow over longer periods of time.

The PSID can also be used to investigate the effect of SES on health across the full life-course. Table 10 summarizes my PSID results predicting future onsets of major chronic conditions. Following the HRS format, three financial measures of SES are used—baseline levels of household income and household wealth and the increase in stock wealth observed over

[11] Similar to the HRS findings, new minor health events had no detectible effects on any of these outcomes.

the period covered by the health shock. Consistent with the time frame allowed by the wealth modules, three time periods are used with alternative baseline years—1984, 1989, and 1994. The occurrence of major health events is measured over five-year intervals.

These results closely parallel those obtained for the older populations represented by the HRS and AHEAD. Whether one looks at the relatively short horizon of the next five years or more than a decade ahead, all three financial measures of SES are very poor predictors of future health outcomes. These longer horizon PSID results on financial measures of SES are quite powerful in that they partly respond to the objection that one may have controlled for most of the indirect effects of SES by conditioning on baseline attributes. In this case, the conditioning variables are sometimes measured more than a decade before.

Once again, all this does not imply that SES cannot predict future health events, as education is a statistically significant predictor across both a short and long horizon. Even after one controls for an extensive array of current health conditions, those with less schooling are much more likely to experience a major negative health onset.

The basic question is whether our main conclusion about the dominance of education over financial measures of SES is sustained when we consider the complete life-course. To place the issue in some perspective, Figure 3 plots the education gradient for those in poor or fair health in the same manner as Figure 2 did for income. In several key dimensions, the income and education health gradients are quite similar. Whether stratified by income or education, higher SES is associated with better health, a relationship that first expands with age up to around age 50 and then contracts, and one that is highly non-linear with the lowest SES group in much worse health than all the others. But there are some differences as well. Most important,

unlike income the education health gradient is more persistent and never fully disappears at either very old or very young ages.

Given the strong correlation of income and education, the question of whether the SES health gradient is due to income or education requires examining them jointly. Before doing so, a few preliminaries are in order. Those in lower SES groups are more likely not to be married, which alone produce lower family incomes. To control for this confounding factor, I limit samples in what follows to married individuals. For this sample, Figure 4 displays the health gradient by income quartile among those with 0-11 years of schooling. Now the strong income effects that were present especially at younger ages—say below age 50—virtually disappear with one key exception—those in the lowest income quartile remain in much worse health. While not shown here, the same story applies to the other two education groups—those with 12-15 or 16 plus years of schooling.

Why is the bottom income quartile so distinct as a signal of poor health even after controlling for education? A clue to the potential answer is contained in Figure 5, which plots for those with 0-11 years of schooling the fraction who are working within each income quartile. The basic age pattern is not surprising with labor force participation rates declining rapidly during ages 50-65 as retirement looms. Comparing Figures 4 and 5, the patterns across income quartiles are remarkably similar. There is not much difference among the top three income quartiles, but the bottom income quartile stands apart. Even at relatively young ages—30's and 40's—a large fraction of those in the bottom income quartile are not working, strongly suggesting that their low incomes are a consequence of not working.

To push the question one step further, why are so many people in the lowest income quartile not in the labor force even in the prime of their lives? Figure 6 completes the circle and

provides my answer. This graph plots within-education groups the fraction in poor or fair health by their labor force status. Those who are not working are much more likely to report very high fractions in poor or fair health. For example, at age 50, 70 percent of those not working report themselves in either poor or fair health—a fraction 40 percentage points larger than among those who are working.

Conclusion

In this paper, I re-examined one of the most important but mysterious social science issues of the day—the substantial gradient of health with SES. My midterm report based on my personal voyage of discovery is this. First, causal pathways from health to financial measures of SES are very important as new serious health events have a quantitatively large impact on work, income, and wealth. The current literature tends to downplay this pathway and it should not. Second, SES also impacts future health outcomes, although the primary culprit here is years of schooling and not an individual's financial resources.

Contrary to widespread and deeply held beliefs within the policy and research community, I present here a body of empirical evidence that the principal financial measures of SES—household income and household wealth—do not seem to be related to individual health outcomes. But in research one finding always begets another puzzle. There is growing evidence, including some presented in this paper, that measures of economic circumstances during childhood do matter for later life health outcomes. Parental incomes appear to be central correlates of the onset of some critical childhood diseases, which then set the stage for the adult SES-health gradient (Case, Lubotsky, and Paxson 2002). In a more historical vein, certain months of birth that coincide with the nutritional benefits of the agricultural cycle are associated with added years of life even at older ages (Doblhammer and Vaupel 2001). Why is health

apparently so sensitive to financial resources in the early years of life, an influence which then disappears as we age? While the influence of money may dissipate, the impact of how we are stratified by other aspects of SES decidedly does not. Whatever the origins of this stratification, it has profound implications for population health, where the consequences are serious and where the core reasons remain a mystery.

References

Adams, Peter, Michael Hurd, Daniel McFadden, Angela Merrill, and Tiago Ribeiro. 2003. "Healthy, Wealthy, and Wise? Tests for Direct Causal Paths Between Health and Socioeconomic Status," *Journal of Econometrics* 112:3-56.

Barker, David J. P. 1997. "Maternal Nutrition, Fetal Nutrition and Diseases in Later Life," *Nutrition* 13(9):807-813.

Case, Anne, Darren Lubotsky, and Chris Paxson. 2002. "Economic Status and Health in Childhood: The Origins of the Gradient, "*American Economic Review* 92(5):1308-1334.

Doblhammer, Gabriele, and James Vaupel. 2001. "Lifespan Depends on Month of Birth," *Proceedings of the National Academy of Sciences* 98:2934-2939.

Goldman, Dana, and Smith James P. 2002. "Can Patient Self-Management Help Explain the SES Health Gradient?" *Proceedings of the National Academy of Sciences; USA (PNAS),* August 6, 99(16):10929-10934.

Krieger, Nancy. 1999. "Embodying Inequality: A review of Concepts, Measures, and Methods for Studying Health Consequences of Discrimination," *International Journal of Health Services* 29(2):295-352.

Marmot, Michael. 1999. "Multi-Level Approaches to Understanding Social Determinants," in Lisa Berkman and Ichiro Kawachi (eds.), *Social Epidemiology,* Oxford University Press, Oxford.

Seeman, Teresa, Burt Singer, John Rowe, Ralph Horwitz, and Bruce McEwen. 1997. "Price of Adaption-Allostatic Load and its Health Consequences," *Archives of Internal Medicine*, October 27, p 157.

Smith James P. 1999. "Healthy Bodies and Thick Wallets," *Journal of Economic Perspectives* 13(2):145-166.

Smith, James P. 2000. "Why is Wealth Inequality Rising?" in Finis Welch (ed.), *Increasing Income Inequality in America: The Facts, Causes, and Consequences*, University of Chicago Press, pp. 83-116.

Smith, James P. 2003. "Consequences and Predictors of New Health Events," paper presented at Economics of Aging Conference, Boulders, Arizona, April 2003 (to be published in the *Economics of Aging* Series, University of Chicago Press, forthcoming).

Smith James P., and Raynard Kington. 1997. "Demographic and Economic Correlates of Health in Old Age," *Demography* 34(1):159-170.

Wilkinson, Richard G. 1996. *Unhealthy Societies: The Afflictions of Inequality*, Routledge, London.

Table 1
Impacts of a New Health Shock Between Waves 1 and 2

Major Health Shock			
Wave	OOP medical expenses	Work Probability	Household Income
2	1,720	-.148	-4,033
3	1,037	-.054	-1,258
4	893	-.030	-658
5	503	-.036	-269
Total	4,153	-.268	-6,258
Minor Health Shock			
	OOP medical expenses	Work Probability	Household Income
2	175	-.041	-498
3	313	-.036	-988
4	160	-.017	-44
5	567	-.013	-169
Total	1,215	-.107	-1,699

Table 2
Cumulative Impact of New Major Health Event Taking Place Between

	W1-W2	W2-W3	W3-W4	W4-W5
HRS-sample				
Cumulative Income Loss	-36,884	-13,828	-6,856	-3,601
Cumulative Income Loss + OOP Expenses + Loss Interest	-48,941	-19,388	-9,805	-5,901
AHEAD–sample				
Cumulative Income Loss + OOP Expenses Lost Interest	-11,347	- 3,553	-3,005	

Table 3
Cumulative Impact of New Minor Health Event Taking Place Between

	W1-W2	W2-W3	W3-W4	W4-W5
HRS-sample				
Cumulative Income Loss	-8,727	-8,811	-6,949	351
Cumulative Income Loss + Increase Expenses + Loss Interest	-11,544	-11,584	- 8,610	-316
AHEAD-sample				
Cumulative Income Loss + Increase Expenses +Loss Interest		5,926	-6,838	-702

Table 4
Probits for Future Onset of Chronic Condition

	Any Major		Any Minor	
	Estimate	Chi Square	Estimate	Chi Square
Income	0.0111	0.06	-0.0063	0.03
Wealth	-0.0046	2.26	-0.0005	0.05
Ed 12-15	-0.1108	7.78	-0.0912	5.96
Ed College or more	-0.0844	2.43	-0.1588	10.26
Change in Stock Wealth	-0.0004	0.44	0.0004	0.88

	Cancer		Hypertension	
	Estimate	Chi Square	Estimate	Chi Square
Income	0.0130	0.05	0.0153	0.11
Wealth	-0.0030	0.53	-0.0032	1.01
Ed 12-15	0.0008	0.00	-0.0675	2.45
Ed College or more	0.0567	0.61	-0.0623	1.17
Change in Stock Wealth	0.0003	0.32	-0.0001	0.11

	Diseases of the Lung		Diabetes	
	Estimate	Chi Square	Estimate	Chi Square
Income	-0.0271	0.12	0.0382	0.40
Wealth	-0.0067	1.13	-0.0023	0.29
Ed 12-15	-0.1920	10.32	-0.1153	4.82
Ed College or more	-0.1432	2.67	-0.0777	1.11
Change in Stock Wealth	0.0006	1.13	-0.0023	1.37

	Heart Disease		Arthritis	
	Estimate	Chi Square	Estimate	Chi Square
Income	-0.0447	0.64	-0.0069	0.03
Wealth	0.0015	0.19	0.0000	0.00
Ed 12-15	-0.1086	5.10	-0.0819	4.29
Ed College or more	-0.0519	0.62	-0.1857	12.14
Change in Stock Wealth	-0.0012	1.36	0.0006	2.41

	Stroke	
	Estimate	Chi Square
Income	0.0683	0.70
Wealth	-0.0175	3.83
Ed 12-15	-0.0390	0.36
Ed College or more	-0.0746	0.59
Change in Stock Wealth	-0.0017	0.57

Models also control for presence of baseline health (self-reported health status, functional limitations, and the existence of specific chronic condition), and a standard set of health risk factors (smoking, drinking, and BMI). In addition, sex, race, and ethnicity, and region of residence are included. Income and wealth measured in $100,000 of dollars. Income and wealth measured in $100,000 of dollars.

Table 5
Probits for Future Onset of Chronic Conditions

	Major		Minor	
	Estimate	Chi Square	Estimate	Chi Square
Income	0.0456	0.93	-0.0044	0.00
Wealth	-0.0040	1.60	-0.0001	0.00
Change in Stk Wealth	-0.0008	1.06	0.0003	0.75
Ed 12-15	-0.0783	2.66	-0.0527	1.38
Ed College or more	-0.0483	0.52	-0.0927	2.33
Health Ex or VG as child	-0.0949	4.68	0.0042	0.01
Not poor during childhood	-0.0949	6.31	0.0155	0.20
Mother's ed	0.0028	0.18	0.0004	0.00
Father's ed	-0.0018	0.09	-0.0046	0.72
Father alive	-0.1362	1.34	-0.2001	3.32
Age of father's death	0.0001	0.00	-0.0014	0.88
Mother alive	-0.0743	0.49	-0.2465	6.51
Age of Mother's death	-.00002	0.09	-.00028	4.60

Models also control for presence of baseline health (self-reported health status, functional limitations, and the existence of specific chronic condition), and a standard set of health risk factors (smoking, drinking, and BMI). In addition, sex, race, and ethnicity, and region of residence are included. Income and wealth measured in $100,000 of dollars. Income and wealth measured in $100,000 of dollars.

Table 6
Educational Differences in Treatment Adherence at DCCT Baseline

Measure of Adherence	Postgrad degree	College grad/ Some college	HS degree/ Some secondary
Number of times self-monitored blood glucose per week	8.8	7.7	6.7
Missed insulin injection at least once in past month (%)	4.3	6.0	9.2
Did not follow insulin regimen at least once in past month (%)	15.7	25.2	26.6
Did not self-test blood or urine at least one day in past month (%)	66.1	74.1	77.2
Minutes of very hard exercise per week	58.1	49.6	19.7
Currently smoking cigarettes (%)	16.5	19.2	40.8

Source: Goldman and Smith (2002).

Table 7
Educational Differences in Treatment Impact for Diabetics

Group	Glycosolated Hemoglobin:		
	Postgraduate Degree	College grad/ Some college	HS degree/ Some secondary
Conventional Therapy Only (n=495)			
Baseline	8.42	8.76	8.96
End-of-study	8.88	9.08	9.59
Difference	0.46	0.32	0.63
Intensive Treatment Only (n=490)			
Baseline	8.04	8.86	8.93
End-of-study	7.18	7.30	7.43
Difference	-0.85	-1.56	1.51
Treatment Effect[#]	-1.31	-1.88*	2.14**

*p<.10; **p<.05

[#]Treatment effect is the improvement in glycemic control among the intensive treatment group relative to conventional therapy. Significance levels are for a test of equivalence with the postgraduate category and control for duration in study, gender, marital status, and age.

Table 8
Onsets of Major and Minor Conditions by Age
(% Experiencing an onset)

	Age Group			
	Less than 41	41-50	51-61	over 61
Major Onset				
1994-1999	3.9	7.2	12.9	26.0
1989-1993	1.5	3.4	6.9	12.0
1984-1988	0.6	1.4	4.2	6.0
1999 Major Prevalence	7.0	13.3	26.1	46.0
Minor Onset				
1994-1999	12.2	23.1	28.8	30.3
1989-1993	3.9	10.4	16.7	23.7
1984-1988	1.7	3.9	8.0	12.6
1999 Minor Prevalence	17.9	38.6	54.7	72.6

Source: Calculations by author from 1999 PSID.

Table 9
Impacts of New Major Health Shock — 1995-1999

	Ages		
	Less than 51	51-61	Over 61
Change in Employment	-0.084	-0.307	-0.202
Change in Family Income	-488	-2,731	-107
Change in Net Worth	-2,889	-8,789	-1,507

Table 6
Does SES Predict Future Major Disease Onsets? (ages 21 and over—PSID)

	First 1-5 Years		6-10 Years		11-15 Years	
			1984 Baseline			
Income	0.0013	(1.39)	0.0010	(0.11)	-0.0080	(1.14)
Wealth	0.0002	(0.54)	0.0001	(0.32)	0.0003	(0.97)
Change in Stock Wealth	0.0020	(0.74)	0.0001	(0.10)	0.0006	(2.40)
Ed 12-15	-0.1217	(1.25)	-0.2160	(2.82)	-0.1312	(1.94)
Ed 16+	-0.2834	(2.14)	-0.3238	(3.02)	-0.2888	(3.09)
			1989 Baseline			
Income	0.0016	(0.25)	-0.0030	(0.71)		
Wealth	-0.0007	(0.76)	0.0004	(1.11)		
Change in Stock Wealth	0.0010	(0.51)	0.0006	(2.30)		
Ed 12-15	-0.1971	(2.73)	-0.1489	(2.30)		
Ed 16+	-0.3170	(3.22)	-0.2743	(3.09)		
			1994 Baseline			
Income	-0.0089	(1.91)				
Wealth	0.0005	(1.21)				
Change in Stock Wealth	0.0004	(1.28)				
Ed 12-15	-0.1387	(2.27)				
Ed 16+	-0.1844	(2.18)				

Financial variables expressed in $10,000. *z* statistics based on robust standard errors.

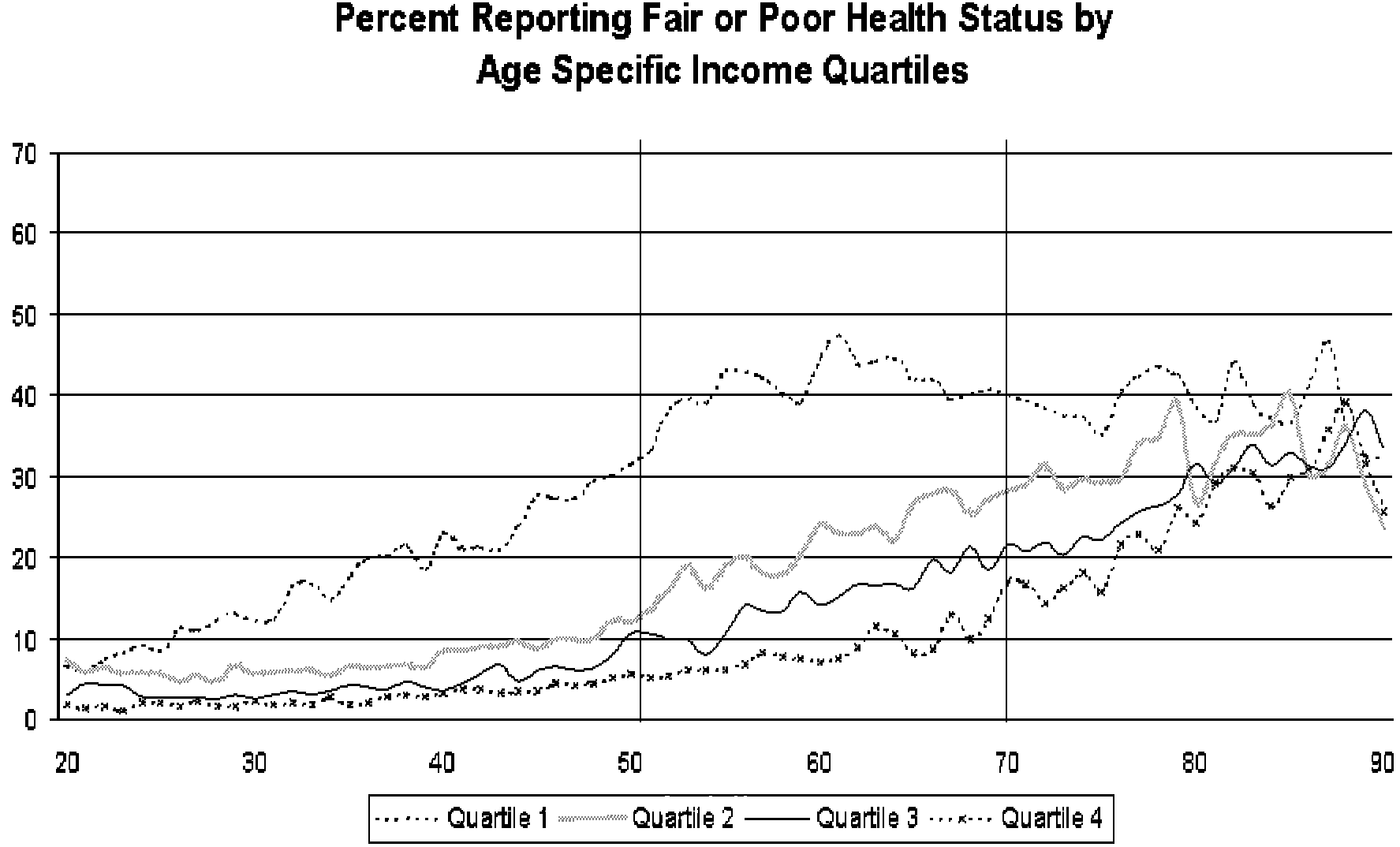
Percent Reporting Fair or Poor Health Status by
Age Specific Income Quartiles
70
60
50
40
30
20
10
0
20
30
40
50
60
70
80
90
Quartile 1
Quartile 2
Quartile 3
Quartile 4

Figure 2

% Reporting Fair or Poor Health Status by Education

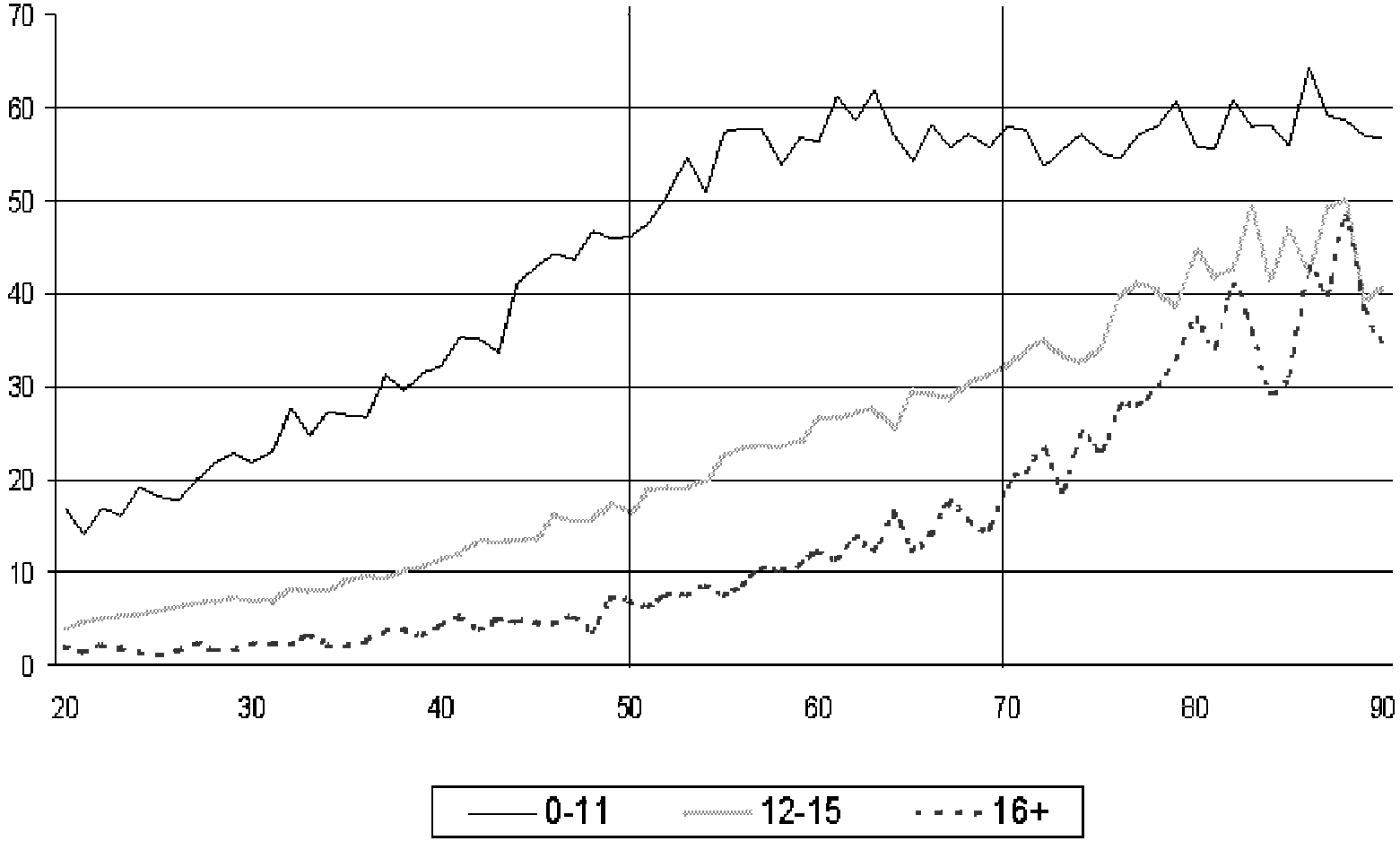

Figure 3

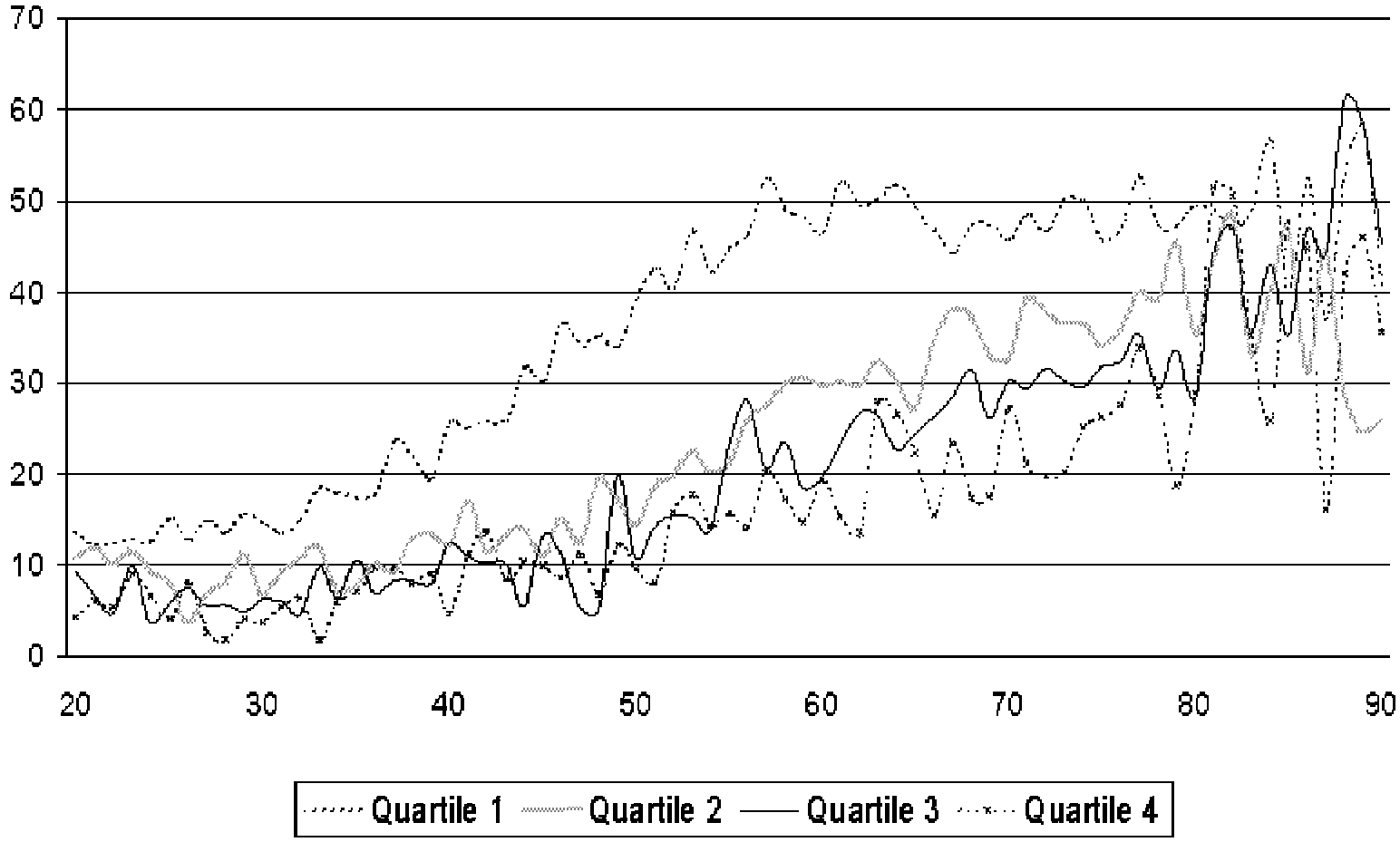
Percent of Married Male Respondents with
0-11 Years of Education Reporting
Fair or Poor Health Status by Income Quartiles
70
60
50
40
30
20
10
0
20
30
40
50
60
70
80
90
Quartile 1
Quartile 2
Quartile 3
Quartile 4

Figure 4

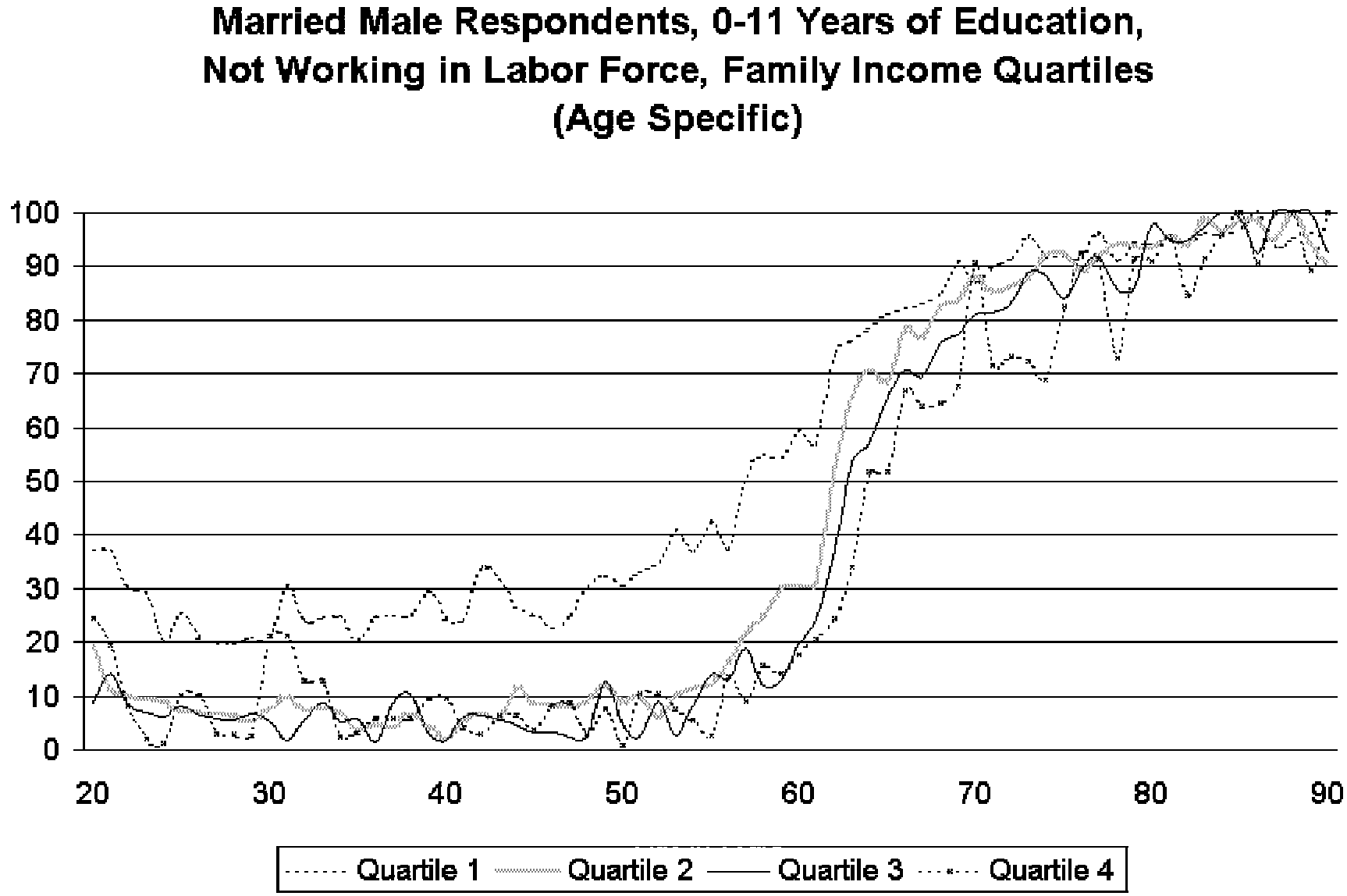
Married Male Respondents, 0-11 Years of Education,
Not Working in Labor Force, Family Income Quartiles
(Age Specific)
100
90
80
70
60
50
40
30
20
10
0
20
30
40
50
60
70
80
90
Quartile 1
Quartile 2
Quartile 3
Quartile 4

Figure 5

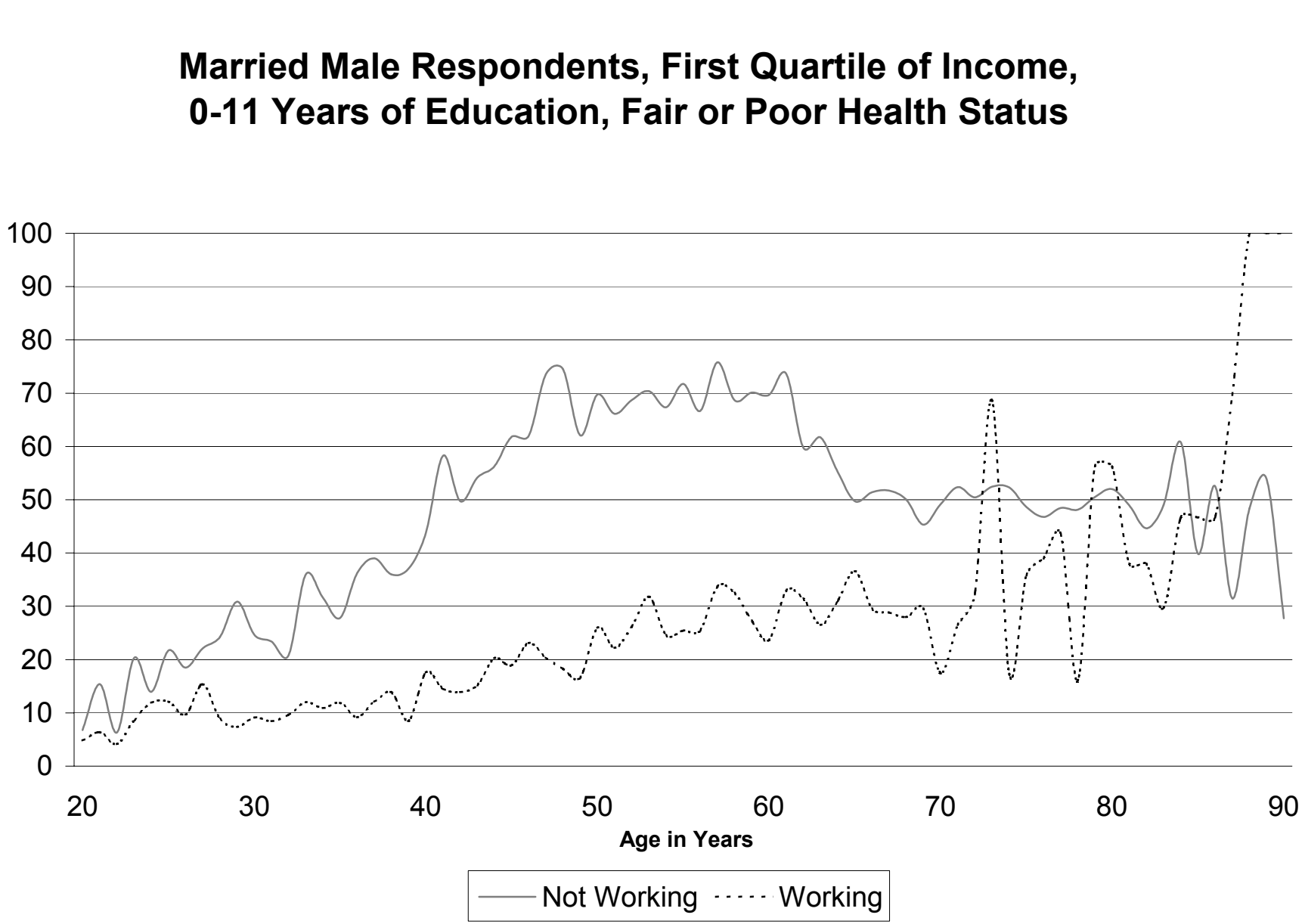
Married Male Respondents, First Quartile of Income,
0-11 Years of Education, Fair or Poor Health Status
100
90
80
70
60
50
40
30
20
10
0
20
30
40
50
60
70
80
90
Age in Years
Not Working
Working

Figure 6