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The Impact of SES on Health over the Life-Course

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People of lower socio-economic status (SES) have much worse health outcomes (Marmot (1999), Smith (1999)). But why this is so remains under considerable debate ((Adams et al. (2003), Deaton (2003)). A central question is whether these large differences in health by such SES indicators as income or wealth largely reflect causation from SES to health. But even if SES mainly affects health, what dimensions of SES actually matter—financial aspects such as income or wealth or non-financial dimensions like education?

This paper begins with the perspective that there is a distinct life-course component to the health SES 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 age fifty or age seventy as required by the widely used Health and Retirement data (HRS). To see this, Figure 1 displays the main contours of the SES health gradient 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.[1] At least until the end of life, at each age every movement down in income is associated with being in poorer health. Moreover, the health differences by income class are quite 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. Finally, there is a quite 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.

Figure 2 illustrates that a quite similar pattern emerges when our SES measure shifts to household wealth—an expanding health gradient into middle age followed by a gradual contraction.[2] Given these quite dramatic age patterns, the value of panels such as the PSID that

[1] The data in figure 1 are calculations by the author from the National Health Interview Survey.
[2] Figure 3 is calculated by the author from the PSID wealth modules.

span the entire life course in addressing these difficult questions about the meaning of the SES-health gradient would seem to be potentially quite high.

This paper is divided into three sections. Section 1 describes and then Section 2 evaluates the quality of the health data available in the Panel Study of Income Dynamics (PSID). In recent years, the PSID has added significant health content to its core module so that such an evaluation is timely. Section 3 examines the effect of SES on health by looking at whether the onset of new chronic conditions is related to household income, wealth, and education once one conditions of a set of pre-existing set of demographic and health conditions. This analysis attempts to take advantage of two of the unique features of the PSID for this type of analysis—its ability to track health onsets well into the future and to assess whether the predictive effects of the principal SES measures vary with age.

Section 1—Data

The Panel Study of Income Dynamics (PSID) 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 who live in those families. There are several potential advantages to using the PSID to study issues surrounding the SES-health gradient. In contrast to the other panel surveys such as the HRS and AHEAD that have been widely used to investigate the SES-health gradient, the PSID spans all age groups allowing one to examine behavior over the complete life-cycle. Figures 1 and 2 have already indicated that the nature of the SES-health gradient may differ significantly across age groups.

The PSID is rightly recognized as one of the premier general-purpose panel survey measuring several key dimensions of SES. For example, details on family income and its components have been gathered in each wave since the inception of PSID in 1968. Starting in

1984 and in five-year intervals until 1999, PSID asked a set of questions to measure household wealth. Starting in 1997, the PSID switched to a two year periodicity, and wealth modules are now included as part of the core interview. No other survey can match the long history of financial SES information now available in the PSID.

The PSID has not traditionally been known as a health survey but that situation may change. The PSID has been collecting information on self-reported general health status (the standard five-point scale from excellent to poor) since 1984 and has always collected good information on work-related disabilities. Starting in 1999 and for all subsequent waves, PSID has collected 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. Thus keeping in mind issues related to recall bias, the implications of which I discuss below, the timing of the onset of a health shock can potentially be identified and the impact of these new health events on labor supply, income, and wealth can be estimated.

The PSID offers several key potential additions to the research agenda. First, as the data provided in Figures 1 and 2 suggest, the nature of the SES-health gradient may vary considerably over the life cycle. For example, during the age span covered by the HRS, the income-health gradient was actually becoming considerably smaller. 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 relatively long periods of time—at this point measured in decades. It may well be, for example, that health responds to changes in financial measures of SES but only after a considerable lag. Third, the PSID also permits a unique long-term perspective in the other direction. Given that many panel members have been members of the PSID since 1967, the entire past sequence of financial situation can be exploited in the research.

Table 1 summarizes in more detail the relevant health variables available in the PSID.[3] The major revisions that impacted the entire sample took place in 1984 and in 1999. In 1984, the PSID introduced as part of its core module for respondents and spouses the 5-point scale on general health status (ranked from excellent to poor). In 1999, respondents were asked about specific chronic conditions (the specific list is contained in Table 1) including information about date of onset. These questions have been repeated in subsequent waves with some new health domains included, especially mental health and depression indexes added in 2001.[4]

While it doesn't directly measure respondents' health, Table 1 documents other health-related information available in the PSID that may be quite useful in analysis. This includes a short list of health behaviors (smoking, drinking, and exercise), which was then added in the core in 1999. Periodic surveys have also been conducted in the PSID about older respondents (ages 50+ or ages 55+).

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[3] Given its substantive focus on work and income, the PSID has always included some questions related to health limitations on work ability. For example, respondents have been asked since 1968 whether a physical or nervous condition limited their ability to work and the severity of any limitation. While quite useful for their intended purpose, these work-limitation questions focus on one aspect of health, have little to say about the health of those not currently working, and thus have not been used for studies of population health.

[4] ADL and IADL type questions have been part of the PSID since 1992, but until the 2003 wave these questions have been restricted to ages 55 and over so that they are of limited value for health analysis across the entire age distribution and will not be used in this paper.

Eventually, the repetition of this information on new health conditions in all future PSID waves will mean that longitudinal analysis of the impact of such onsets will be possible. But it will be some time in the future before enough waves are available to conduct such research. The question I investigate in this paper is whether this type of analysis can currently be usefully pursued using the contemporaneous and retrospective data collected in the 1999 wave.

Section 2—The Quality of the PSID Health Data

The two major elements of the expanded PSID health content that potentially would seem analytically useful are the prospectively collected general health status available yearly since 1984 and the 1999 retrospective health conditions information. Retrospective data have several well-documented issues surrounding the ability to recall.[5] The extensive literature on this subject indicates that a question about an event that occurred decades ago tends to yield a less reliable response than a query about a similar event taking place last week or last year. Similarly, the more salient an event, the more likely it will be recalled, particularly as time since the event increases. Some studies have shown that salience of an event is also associated with a tendency to report the event as having taken place more recently than it actually did (forward telescoping).

In addition to general recall issues, a practical concern also arises in the PSID that directly impacts the sample size available for any analysis. Since the 1999 chronic condition questions were only asked of heads and their spouses who were present in that year, the condition data are obviously limited to only such people. Respondents who had left the PSID for any reason before then—no matter how long they had been PSID members—would not be part of any research that required knowledge of chronic conditions. Similarly, going backwards from

[5] A large literature has developed on response errors in general, and the quality of recall data in particular. Sudman and Bradburn (1974), Sudman et al (1996), Bound et al. (2000) and Smith and Thomas (2003) provide insightful summaries.

1999, we would cumulatively start losing respondents the further back we went because they were not yet members of the PSID panel. For example, in the analyses described below the sample size for changes between 1994-1999 is 7,205; between 1989-1994 is 5,783 and between 1984-1989 is 4,820.

The key dimension of the quality of PSID health information, especially for the analyses in this paper, concerns the retrospective information on date of onset of disease. A common practice with date recall involves an over-reliance on focal responses, especially easily remembered digits. When asked about the date of onset, respondents may center their responses on 5, 10, 15, 20 years ago etc.[6] Figures 3 and 4 document the extent of this problem by plotting the frequency distribution of year of onset for each chronic condition. Figure 3 illustrates those four chronic conditions—arthritis, diabetes, high blood pressure, and diseases of the lung—where the problem appears to be most serious while Figure 4 includes the three diseases—cancer, stroke, and heart disease—where the use of focal values is less common. The tendency to concentrate responses on focal responses appears to be far greater the less serious the disease and the further back in time the health event occurred. For example, concentration on focal responses appears to be far less for cancer than hypertension and greater at the 10-year point than at the 5-year points.

All PSID questions including the 1999 health battery are answered by one respondent who reports for him/herself and the spouse, if any. It is reasonable to suspect that the accuracy of reports may be better when the information requested refers to the respondent than when a respondent is asked about the spouse. This may be especially true when data are requested on the date of onset of disease. Table 2 investigates this issue by listing the fraction of cases where

[6] This well-known problem has even more salience in the PSID because these focal dates are the same as the year the wealth modules were administered.

disease onset was reported five or ten years ago or fifteen or twenty years ago. This data are stratified by whether the onset was major or minor and whether the report was about the respondent or spouse. Major conditions were defined as cancer, heart condition, stroke, and diseases of the lung. All other onsets are defined as minor.[7]

For recent events, say around the five or ten year marker, where the event is likely to be remembered but the date is uncertain, one would anticipate more focal year responses when the onset refers to the spouse instead of the respondent. Moreover, this tendency should be greater for minor health onsets than for major ones. Table 2 supports both conjectures—27% of all minor onsets for the spouse are reported at a five-year or ten-year interval compared to 23% for the respondent. The difference for major onsets is much smaller—17.8% compared to 16.7%.

In contrast when the event took place in the distant past, say around the fifteen or twenty-year marker, remembering the event at all is at risk, especially for the spouse where the health event may have even preceded the marriage date. The greater tendency to not remember the event for spousal onsets could overwhelm the greater concentration at focal years. Once again, the data in Table 2 support this conjecture. For health events that took place more than ten years ago, there is actually a greater concentration at focal years for self-health onsets than there are for spousal onsets.

A useful comparison to assess data quality would involve the same type of information for people of the same age collected at the same time prospectively. Surveys like the HRS that have been collecting information prospectively for over a decade presumably should serve as a useful quality benchmark for the retrospective PSID onset data as the uncertainly over timing may be largely (but not completely) confined to a two-year window.

[7] In this table major trumps minor so that an individual who reports both a minor and major onset is included in the major category.

Table 3 documents the extent of onset of new health events that took place across the first five waves of the HRS—an eight-year time interval. In the left side of the panel, these onsets represent incidence rates for major and minor chronic conditions for respondents who were members of the original HRS cohort (those born between 1931 and 1941) in 1992 (approximately 51-61 years old).

The extent of the new health problems reported in HRS during these eight years is impressive. Independent of their baseline status, about half of all respondents experienced some type of onset during the first five HRS waves. Note that the conditional probability of a major onset is much higher if one had already reported some type of health problem at HRS baseline than if one was chronic condition free. This no doubt reflects the progressive nature of disease where having relatively minor medical problem (such as hypertension) heightens the odds of experiencing another more severe one (such as a heart attack).

Table 3 also attempts to replicate the same information retrospectively from the PSID in the right side panel. Since the chronic conditions information is first available in 1999, the sample I use to maintain comparability with HRS are those PSID respondents who were 59-69 years old in that year and who would have been 51-61 years old in 1991. Using the PSID prevalence and year of onset data available, I compute 1991 prevalence rates and the extent of new onset over the next eight years—the same time frame used for the HRS in Table 3.

When we examine incidence across these eight years, minor incidence is actually higher in the PSID compared to the HRS. This ranking may indicate some forward telescoping of minor conditions in terms of date of onset in the PSID, a not uncommon finding with less salient events. In contrast, the two surveys are almost identical in the reporting incidence of major

conditions. The data in Table 3 suggest that the timing data in the PSID may be more reliable for the major onset conditions with more serious recall issues for the minor onsets.

To this point, my discussion of the health information in the PSID has focused on chronic conditions, ignoring the prospectively collected self-reports of general health status since 1984. This information will serve as its own measure of health status below, but it also can also be used to help make the dating on onset more exact. For example, one might expect that at the time when the onset actually occurred, respondents may have been much more likely to downgrade their own assessment of their general health.

Table 4 examines this issue by listing the percent of cases in which a PSID respondent increased, decreased, or left the same his/her reported general health status using the standard five point scale. The data are provided separately for the experience of a major onset, a minor onset, and no onset during the period 1987-1994 inclusive. Based on the reported year of onset, the data are then provided separately for self-reports that are one year removed, two years removed, and three years removed in both directions from the reported onset.

The potential usefulness of these self-reports is illustrated by examining the fraction who downgraded their health assessment using the two-year interval. Forty-one percent of those who reporting experiencing a major onset downgraded their self-reported health compared to 23% among those experiencing no onset. However, the limitations of self-reports for precise timing are also apparent as a similar fraction of people upgraded their status no matter whether they reported a major, minor, or no onset within this interval. Self- reports of general health status are known to be quite noisy, and using the self-reports to more precisely time events is difficult. For example, when asked the self-reported health status questions twice in the same survey, 28% of respondents changed their answer (Crossley and Kennedy (2002)).

The likelihood that the reported interval contains the time of the true onset should increase with the length of the interval, which would suggest that the differences across those reporting onsets and those not reporting should also increase as the interval lengthens. This does happen for both the major and minor onsets as the fraction who down grade their health increases with the length of the interval. However, this effect stabilizes after the four-year interval suggesting that the actual major onset most likely took place within those four years. This should not be surprising given the salience of the major health onsets. In contrast, the fraction who downgrade continues to rise for those with a minor onset, also consistent with its lower salience and the dating difficulty documented above.[8]

The final set of rows provides another HRS reference standard by showing the same type of data for respondents between the first and second wave of HRS. While not certain, it is highly likely (compared to the PSID) that the actual onset occurred within this interval. Given the lack of uncertainty about the onset between the HRS waves, this data add weight to any concern about the noisy nature of self-reports—fifteen percent of HRS respondents who had a major onset reported that their health improved! If the onset is more likely to have taken place within this interval in HRS, we should also find a sharper differentiation in the changes in self-report between those who had an onset and those who did not. This is indeed what we do find especially for the minor onsets where I believe the dating reliability issues are more serious.

These problems related to recall bias suggest several points to keep in mind in the analyses presented below. First, any dating reporting problems appear to be much less important for major chronic conditions than for minor ones. Second, health onsets that took place close to

[8] The only difficulty with these comparisons is that I would have thought that there would have been little change in the no onset group from lengthening the reporting interval but it appears to also produce an increase in down reports between the two and four year intervals One explanation may be that the age difference between the before and after assessment must increase by two years across these reporting intervals. But this seems like too small an age difference to produce this effect.

1999 should be less affected by recall bias so that quality of estimates of the impact of health events may decay as we move the time interval back in time. Third, the bias may be less important for analysis where health onsets are the outcomes studied (as in this paper) then when we are attempting to measure their impact and place them on the RHS of the analysis. Fourth, the quality of dating on onsets appears to be more reliable when the reports refer to the respondent than when they are about the spouse. Finally, by allowing some relatively minor flexibility in the dating of events one may be able to mitigate the impact of any reporting bias. For example, by extending the year prior to and after the reported time of onset, the probability that the health onset occurred within the income or labor supply interval appears to be significantly increased. I will pursue that strategy in this paper.

Section 3—Predicting the Onset of Future Health Conditions

In this section, I explore the pathway from SES to health by examining whether the future onset of new chronic conditions is related to levels of household income, wealth, and education, once one conditions of a set of pre-existing set of demographic, economic, and health conditions.

Before summarizing those results, it is useful to first outline the essential issues in estimating effects of SES on health. Following the treatment in Adams et al (2003), 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 equation:

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

where X_{t-1} represents a vector of other possibly non-overlapping time and non-time varying factors influencing health and SES and u_{1t} are stochastic shocks to health.

In this framework, we can estimate whether past values of SES predict health ($\alpha_2 \neq 0$).[9] Strictly speaking, α_2 does not estimate a causal impact and is better viewed, as it will be in this paper, simply as the ability of past values of SES to predict future health onsets. The reason is that there may well be unobserved factors correlated both with past SES and health, even after we have conditioned on the measured components. But even with this important caution, the estimated values of α_2 may be suggestive in the causality debate. Most of these unobserved factors would tend to produce a positive correlation between past SES and health biasing α_2 upwards. Not finding any impact of some components of SES on health would then indicate either no effect or a somewhat implausible negative one.

Another key parameter α_3 measures the effect of new innovations of SES ($\Delta\hat{Y}_t$) on health.[10] To estimate α_3, we require exogenous variation in SES ($\Delta\hat{Y}_t$) that is not induced by health . In particular, this implies that it is not appropriate to use the full between period changes SES (ΔY_t) to estimate these effects since such variation hopelessly confounds feedback effects. One opportunity for estimating the effects of SES not caused by health lies in the large wealth increases that were accumulated during the large stock market run-up during the late 1980s and 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.

[9] 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.

[10] The term ΔY_t represents that part of the change in SES that is an innovation.

While this empirical approach is followed in this paper, a legitimate criticism in my view is that—given the distribution of stock market capital gains in the population—we essentially end up studying the effect on 'exogenous' financial wealth changes on health for mostly the very well to do. Not only may we be missing the policy relevant part of the population, but the average health change induced by these innovations in the financial dimensions of SES are measured across a population whose health is very good to begin with and whose health is probably less likely to change.

The central implication of equation (1) is that it is necessary to condition on the full array of baseline health and SES status to estimate the relation between SES and health. Thus, the models I estimate include as covariates a vector of baseline health conditions of the respondent—self-reported general health status (excellent, very good, good, fair with poor the left out group) and the presence of chronic conditions (arthritis, cancer, diabetes, heart disease, hypertension, lung disease, and stroke) all measured at baseline. The models also include a standard set of demographic controls—age (dummies for whether one is 51-61 years old or age 62 and over), race (dummy for African-American), ethnicity (dummy for Hispanic), sex (dummy for female, marital status and transition between waves, and Census region of residence.[11]

The PSID only allows one to back cast one health behavior so that one can also condition on it at each of the baseline years. Fortunately, it is the most important health behavior- smoking- and is well known to be highly correlated with both education and income. Three smoking related variables are included in these models- whether one ever smoked before baseline, whether one currently smoked at baseline, and the number of cigarettes normally smoked.

[11] To preserve space and since they are not the primary variables of interest, the estimated coefficients of the marital and region variables are not included in Tables 5,6, and 7.

In this analyses, my main interest, however, lies in evaluating the importance of the SES measures that include household income, baseline levels of and changes in household wealth, and respondent's education. 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. In addition to years of schooling, three financial measures are used—baseline levels of household income and household wealth and the increase in stock wealth observed over the period covered by the health shock.

Tables 5, 6, and 7 summarize my principal results using the PSID to predict the future onset of major and minor chronic conditions.[12] Consistent with the time frame allowed by the wealth modules, which were collected at five-year intervals starting in 1984, three time periods are used with alternative baseline years—1984, 1989, and 1994. The occurrence of health events is then measured over five year intervals so that for the 1984 and 1989 baseline analyses we can look forward for three and two five year intervals respectively.

In all specifications, not surprisingly onsets of minor and major disease are strongly positively related to age and negatively related to baseline levels of self-assessed health status. Collectively, prior chronic conditions are also strong predictors of future new health onsets.[13]

[12] These models and those in the next section are restricted to survivors- those who neither attrited or died across the waves so this analysis ignores the relationship of SES with attrition and mortality. Given the age range of PSID respondents, mortality selection but not attrition 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).

[13] Negative coefficients on some prior chronic conditions are not an anomaly since having some of these conditions in the past precludes new onset of that condition.

Smoking, and especially its intensity as proxied by the number of cigarettes, continues to produce new incremental bad health outcomes, even after conditioning on an extensive set of measures of current health status. Since smoking has a strong negative gradient with education, its inclusion in these models reduces the predictive effects of education on new health onsets.

Whether one looks at the relatively short horizon of the next five years or a decade or more ahead, all three financial measures of SES are very poor predictors of future health outcomes whether they are major or minor onsets. The estimated coefficients are rarely statistically significant and they even vacillate in sign being more often positive than negative. To illustrate, of the 36 coefficients estimated on the three financial variables (income, wealth, and new stock wealth) in Tables 5-7 in only four cases is the estimated effect statistically significant and in three of those four the effect is actually positive.

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 including income. In this case, the conditioning variables are sometimes measured more than a decade before the disease onset. 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 15 years.[14] These results imply that whether interpreted as predictions or more boldly as causally, financial measures of SES have no discernable impact on future health outcomes.

All this does not imply that SES cannot predict future health events, as education is consistently a statistically significant predictor of major health events across both a short and long term horizon. Even after one controls for an extensive array of baseline health and financial economic conditions, those with less schooling appear to be more likely to experience a major

[14] For supporting evidence on these statements using the HRS, see Smith (2003).

negative health onset. Of the 12 education coefficients estimated in these three tables, all but two are negative and seven of them are statistically significant. This is true both over the short horizon of the next five years as well as over the longest horizon represented in the data—11-15 years out. Even after conditioning on all prior health events which are themselves related to education, years of schooling still has predictive effects on diminishing the likelihood of a new health onset.

A legitimate issue that can be raised about the models in Tables 5 through 7 is their reliance on single period Markovs in both income and health. For several reasons, of which measurement error in income or health (documented above with self-reported health status) is but one good example, one period Markovs seem quite limited. For example, current income may not be the appropriate concept for predicting future disease trajectories. Future onset of disease may be more influenced by longer-term measures of financial resources, which may well be one reason why education matters so much. Similarly, measures that rely only on last periods health ignore the duration dimension of any health problems that might exist, a dimension quite likely to be associated with the onset of new disease. With this in mind, the models in Tables 5, 6, 7 were re-estimated using up to four period income lags and four period lags in self-reported general health status. The baseline 1984 model only would allow a year one lag in general health status and a baseline 1994 would only permit a short window for a new onset to occur. Thus, models with these longer lag structures in income and health were estimated for the 1989 baseline. All other covariates in these models remain the same as those summarized in Tables 5, 6, 7.

Since the principal issue is whether these lags matter, Table 8 summarizes these results with a series of "F" tests and the associated probabilities for differences from zero for a one

period lag, the three additional lags beyond the last year, and the full four periods. The results obtained with lag structures on income are easy to summarize—in no case do any of the variants of the income lags matter. Since a four period year may serve as an approximation to longer run measures of income, this suggests that the reason current income doesn't matter does not lie in the familiar permanent transitory income distinction. Moreover, the prior summary that they also do not predict future onset for the other financial variables included in the model-wealth and the change in wealth- remains intact even with the full set of income and health lags in place. Finally, education still predicts future onset indicating that it is not simply serving as a proxy for permanent income, which should be captured in part by the lag structure in past incomes.

The story for lagged health however is quite different. A one period lag for health is clearly rejected and the full four period lag structure is needed to predict future health onset. As mentioned above, this probably reflects both measurement error in health, especially in the noisy self-reported general health status, as well as the impact of past duration of illness. However, even that four period structure does not alter the conclusion that even after conditioning on these measures of health over the last four years, education still predicts future onset and financial measures of SES do not.

One advantage of the PSID is that it allows us to investigate whether predictive effects of SES on health onset vary with age. Much of the current research on this topic is based either on the original HRS (Smith 1999) or the AHEAD cohort (Adams et al, 2003), which are restricted to those between the ages of 51-61 and 70+ plus respectively. To investigate this possibility, separate models were estimated over three age groups—those less than 40, those over 60, and those between those ages. While certainly less common than for those in the HRS and AHEAD age ranges, health episodes for PSID respondents less than 50 years old are not negligible. For

example, among those in their 40's in the 1999 wave, one in seven had previously experienced a major disease onset at some time in their lives and 40% have a minor chronic health condition. In the five years before 1999, 7% of these 40-year olds experienced a major disease onset while one-in-four reported a new minor onset.

Sample sizes are necessarily much smaller when the models are estimated separately into these three age groups. This reduction in sample size becomes especially problematic when onsets are rare- that is the younger the sample (especially for the major onsets) and the shorter the duration over which one allows an onset to occur. Because of these considerations, I estimated these age group models over ten year horizons for the1984 and 1989 baseline years specifications and over the full fifteen-year horizon for the 1984 baseline model. With the exception of age, which is the stratifying variable, these models include the same set of covariates as in Tables 5-7.

The principal results are summarized for all SES variables in Table 9. The column labeled 'financials' counts the number of statistically significant coefficients for the three financial variables-family income, wealth, and changes in stock market wealth- and then indicates the sign of the statistically significant coefficient if any. In these age specific models, once again, financial measures of SES did not matter in any of the age groups. Of the fifty-four estimated coefficients on these financial variables, in only three cases was the variable statistically significant and in each of those three cases the sign was positive. Apparently, the ability of financial variables to predict future onsets is not sensitive to the stage of the adult life cycle one happens to be in.[15]

15 In contrast family income as a child appears to matter a great deal. See Case, Lubotsky, and Paxson (2002). Case, Fertig, and Paxson (2004), and Currie and Hansen (1999).

The remaining columns in Table 9 list the estimated coefficient and associated z value for the education variable in each model. Data necessarily gets thinner with these age stratifications (especially in the oldest age group) and conclusions must be tempered. But for those in the two age groups ages less than 60 and under, the previous conclusion that education remains a strong predictor of future onset of disease apparently remains intact. This is far less apparent for the over age 60 age groups where the estimated effects of education are statistically significant. This is consistent with the findings of Adams et al. (2003) who used the AHEAD sample (ages 70+) and reported virtually no effects of any SES variables including education on subsequent health outcomes.

However, it may also reflect an inherent limitation of the PSID for research in this age group. The 1984 baseline estimates are especially problematic with this age stratification. The 60 plus population in 1984 would be ages 75 plus and living in 1999 when the questions on past disease onset were first asked. Very few people of that age who suffered a severe disease onset would still be alive in 1999 and they would be quite unrepresentative of the actual onsets that occurred. While somewhat less severe since they would have to be only ages 70 plus in 1999, the same type of problem exists with the 1989 baseline models. This raises a more general point that the earlier years of the PSID for this type of analysis are probably best restricted to younger age groups.

In the evaluation of the quality of the PSID health data above, the evidence indicated that the quality of the dating of past onset of disease was better for the respondent than it was for the spouse. There is a standard, and in this case real cost, of lost of power by dropping all spousal observations from the analysis, especially when one is concerned with the onset of sometimes relatively rare events such as a major onset at relatively young age over a duration as short as

five years. To test the sensitively of the main conclusions to a restriction to respondents only and to deal with the lost of sample, I combined major and minor onsets and examined onsets over a ten and fifteen year duration. These results are reported in Table 10 by listing the estimated education coefficient and its associated z value. While estimated education coefficients are slightly smaller in the respondents only sample, the overarching conclusion of this paper that years of schooling continues to predict future onsets of disease is maintained.

Conclusions

In this paper I evaluated the new health information that has recently become available in the PSID to assess whether or not it can serve a constructive role in the ongoing SES-health debate. There are two types of information that appear to be promising—the self-reports of general health status that were first introduced in 1984 and the prevalence and incidence of new chronic conditions that were first added in 1999. In this evaluation, I place particular emphasis on the possibility of using the retrospective information on incidence of chronic conditions. While nothing is perfect, the PSID passes this test with the principal issues being that researchers must take into account dating issues—especially the use of focal points. Analysis that allows for some relatively minor flexibility in the precise dating of events would be less subject to error. In addition, especially for older persons in the sample, the data become less useful the further one goes back in time due to issues of mortality and attrition associated with the onset of health events, in particular the more severe ones. The quality of the dating of onsets also appears to be more reliable when they refer to more severe events and when they refer to the PSID respondent than when they are about a spouse.

The paper also offers several substantive conclusions. First, across the life course SES impacts future health outcomes although the primary culprit appears to be education and not an

individual's financial resources in whatever form they might be received. That conclusion appears to be robust to whether the financial resources are income or wealth or to whether the financial resources represent new information such as the largely unanticipated wealth that was a consequence of the recent stock market boom, Finally, this conclusion appears to be robust across new health outcomes that take place across the short and intermediate time frames of up to fifteen years in the future.

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.

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Table 1. Summary of Selected Health Status and Health Behavior Measures in the PSID: 1968-2003

Measure of health status or health behavior	Family member and years available: H=head, W=wife, FUM=Family members
General health status, height, weight, cognition	
General health status (5-point scale)	H, W: 84-03
General health status when < 17 years old	H, W: 99-03
Height & weight	H, W: 86, 99-03
ADL/IADL-type measures	
Difficulty doing each of the following by self, without equipment?	
bathing or showering, dressing, walking, getting outside, in or out of chair, using toilet	H, W 55+: 92-96, 99-01; All FUM 55+: 92-96; H, W all ages: 03
if yes to any of the above, anyone help you do these activities?	H, W 55+: 92-96, 99-01; All FUM 55+: 92-96; H, W all ages: 03
Specific conditions	
Doctor ever told you that you have…	H, W: 99-03
stroke, high blood pressure, diabetes, cancer, chronic lung disease, heart attack,	H, W: 99-03
emotional, nervous, psychiatric problem, arthritis, asthma, memory loss	H, W: 99-03
How long had each condition?	H, W: 99-03
How much does each condition limit normal daily activities?	H, W: 99-03
Mental health	
30-day depression/anxiety (K6 short screening scale)	Respondent: 01-03
12-mo. major depression screener (from the Composite International Diagnostic Interview)	Respondent: 03
Health behaviors	
Currently smoke? How many packs?, ever smoke, when started, when stop	H, W: 86, 99-03
Anyone in family smoke? How many packs family smoke?	68-72
How often participate in light physical activity, heavy physical activity	H, W: 99-03
Ever drink alcohol? How much?	H, W: 99-03
Parents' health status	
Parents alive? Date of death?	H, W: 88
General health status of mother/father relative to people their age	H, W: 88
Does your father/mother require extra care because of health condition? In nursing home?	H, W: 88

Table 2. Focal Point Responding for Self and Spouse
(% Reporting at focal years)

	Focal Point Years	
	5, 10	11, 15
Minor Onset		
Spouse	27.2	12.8
Self	22.7	15.1
Major Onset		
Spouse	17.8	5.9
Self	16.7	8.5

Percent of PSID respondents with an onset who report at a focal point year, such as 5 years ago, ten years ago etc.

Table 3. Prospective Incidence in the Original HRS Cohort and Retrospective Incidence in the PSID Cohort

	Original HRS		PSID Cohort	
	Cond. Incidence	**Incidence**	**Cond. Incidence**	**Incidence**
NONE		49.9		43.8
None	47.8		43.8	
Minor	36.4		40.4	
Major	15.5		15.8	
MINOR		28.9		35.8
None	51.6		43.7	
Minor	22.1		28.8	
Major	26.5		27.5	
MAJOR		21.4		20.4
None	50.3		44.4	
Minor	28.9		31.8	
Major	21.9		23.8	

Source: Calculations by author from first five waves of HRS—sample born between 1931-1941.
*HRS cohort was 51-61 years old in 1991. The PSID cohort was 59-69 years old in 1999 and therefore the same ages as the HRS over the interval in which the incidence and prevalence is measured.

Table 4. Changes in Self-Reported Health Status Around a Health Event (% of cases)

	Down	Same	Up
Two-year interval			
Major onset	40.6	41.8	17.7
Minor onset	26.9	54.7	18.4
No onset	23.2	55.0	21.9
Four-year interval			
Major onset	47.9	40.9	11.2
Minor onset	33.3	49.3	17.4
No onset	28.9	51.5	19.5
Six-year interval			
Major onset	47.4	38.5	14.1
Minor onset	37.8	43.4	18.8
No onset	28.5	49.9	21.5
HRS—wave1-wave2			
Major onset	47.3	38.0	14.8
Minor onset	36.1	45.0	19.0
No onset	25.4	53.8	20.8

First three panels based on the PSID with variation in the widow over which a new onset is measured. Final panel based on the first two waves of the original HRS cohort ages 51-61 years old.

Table 5
Probits Predicting Onsets of New Health Conditions- Baseline 1994

	Major Onset 95-99		Minor Onset 95-99	
	Coef	z	Coef.	z
Age 51-61	.402	(5.59)	.371	(6.27)
Age 62	.835	(12.38)	.349	(5.75)
Excellent health	-.331	(3.65)	-.443	(5.99)
Very good health	-.291	(3.71)	-.306	(4.65)
Good health	-.237	(3.23)	-.083	(1.32)
Arthritis	.187	(2.61)	-.457	(6.21)
Cancer	.067	(0.51)	-.336	(2.42)
Diabetes	.226	(2.14)	-.184	(1.77)
Heart disease	-.265	(2.17)	.232	(2.43)
High blood pressure	.1063	(1.53)	-.252	(3.95)
Lung disease	.090	(0.68)	.175	(1.50)
Stroke	.135	(0.72)	-.306	(1.62)
Family income	-.0087	(1.83)	-.0051	(1.38)
Wealth	.0006	(1.48)	.0007	(1.58)
Education	-.014	(1.26)	-.017	(2.06)
Ever Smoke	.030	(0.37)	.001	(0.02)
Currently Smoke	.225	(3.20)	-.084	(1.51)
Number of cigarettes	.006	(2.79)	.007	(3.61)
Female	.051	(0.99)	.058	(1.48)
Black	-.024	(0.38)	.150	(3.21)
Hispanic	-.417	(1.84)	.172	(1.53)
Change in stock wealth	.0004	(1.53)	- .0012	(1.83)
Constant	-1.455	(9.04)	-.632	(4.86)

Models also control for Region of Residence and marital status and change in marital status between the waves. Z values are based on robust standard errors. Financial variables measured in $10,000 units.

Table 6
Probits Predicting Onsets of New Health Conditions-Baseline 1989

	Major Onset 90-94		Minor Onset 90-94	
	Coef	z	Coef.	z
Age 51-61	.453	(5.39)	.602	(8.77)
Age 62	.581	(6.20)	.473	(5.99)
Excellent health	-.306	(2.63)	-.534	(5.72)
Very good health	-.214	(2.03)	-.334	(3.97)
Good health	-.056	(0.58)	-.218	(2.71)
Arthritis	.180	(1.67)	-.409	(3.85)
Cancer	.301	(1.58)	.337	(1.87)
Diabetes	.273	(1.81)	-.056	(0.38)
Heart disease	-.063	(0.37)	.016	(0.11)
High blood pressure	.109	(1.12)	-.220	(2.49)
Lung disease	.295	(1.51)	.259	(1.48)
Stroke	-.220	(0.64)	.101	(0.40)
Family income	.0032	(0.52)	.0133	(2.53)
Wealth	-.0006	(0.72)	-.0029	(3.05)
Education	-.030	(2.41)	.015	(1.44)
Ever Smoke	-.026	(0.26)	.184	(2.43)
Currently Smoke	.165	(1.93)	-.211	(3.01)
Number of Cigarettes	.009	(3.47)	.001	(0.41)
Female	-.004	(0.06)	-.024	(0.49)
Black	-.388	(4.52)	.078	(1.30)
Hispanic	-.199	(0.76)	.032	(0.19)
Change in stock wealth	.0015	(0.56)	-.0009	(0.58)
Constant	-1.376	(7.03)	-.919	(5.83)
	Major Onset 95-99		Minor Onset 95-99	
	Coef	z	Coef.	z
Age 51-61	.564	(7.90)	.223	(3.56)
Age 62	.840	(10.84)	.298	(4.27)
Excellent health	-.325	(3.35)	-.291	(3.57)
Very good health	-.324	(3.61)	-.207	(2.66)
Good health	-.100	(1.19)	-.035	(0.48)
Arthritis	.121	(1.29)	-.572	(5.64)
Cancer	-.070	(0.34)	-.313	(1.59)
Diabetes	.157	(1.16)	-.166	(1.21)
Heart disease	-.211	(1.30)	.044	(0.33)
High blood pressure	.233	(2.86)	-.199	(2.57)
Lung disease	-.100	(0.51)	.257	(1.64)
Stroke	-.059	(0.22)	-.215	(0.84)
Family income	-.0025	(0.58)	.0035	(0.96)
Wealth	.0005	(1.28)	-.0006	(0.02)
Education	-.027	(2.40)	-.025	(2.80)
Ever Smoke	.006	(0.06)	-.073	(1.06)
Currently Smoke	.175	(2.35)	-.029	(0.47)
Number of Cigarettes	.006	(2.69)	.008	(3.87)
Female	.005	(0.10)	.025	(0.59)
Black	-.054	(0.78)	.138	(2.69)
Hispanic	-.456	(0.37)	.184	(1.468)
Change in stock wealth	.0006	(2.47)	.0009	(1.28)
Cons	-1.239	(7.46)	-.548	(3.93)

Models also control for Region of Residence and marital status and change in marital status between the waves. Z values are based on robust standard errors. Financial variables measured in $10,000 units.

Table 7
Probits Predicting Onsets of New Health Conditions- Baseline 1984

	Major Onset 85-89		Minor Onset 85-89	
	Coef.	z	Coef.	z
Age 51-61	.484	(4.56)	.599	(8.13)
Age 62	.538	(3.94)	.672	(7.17)
Excellent health	-.513	(3.30)	-.500	(4.84)
Very good health	-.352	(2.68)	-.193	(2.14)
Good health	-.169	(1.40)	-.196	(2.26)
Arthritis	.362	(2.79)	-.422	(3.30)
Cancer	.025	(0.08)	-.439	(1.50)
Diabetes	.290	(1.34)	-.149	(0.77)
Heart disease	-.253	(1.04)	.122	(0.76)
High blood pressure	.205	(1.59)	-.154	(1.50)
Lung disease	-.159	(0.51)	-.276	(1.19)
Stroke	NA	NA	.249	(0.70)
Family income	.0084	(0.84)	.0033	(0.40)
Wealth	.0004	(0.88)	.0003	(0.75)
Education	.005	(0.34)	-.024	(2.17)
Ever smoke	.226	(1.84)	.036	(0.41)
Currently smoke	.064	(0.61)	-.154	(1.92)
Number of cigarettes	.008	(2.55)	.011	(4.49)
Female	-.110	(1.38)	.114	(1.96)
Black	-.140	(1.25)	.139	(1.95)
Hispanic	NA	NA	-.366	(1.59)
Change in stock wealth	.0022	(0.82)	-.0017	(0.82)
Cons	-2.063	(8.47)	-1.110	(6.46)
	Major Onset 90-94		Minor Onset 90-94	
	Coef	z	Coef.	z
Age 51-61	.557	(6.71)	.433	(5.98)
Age 62	.413	(3.60)	.306	(3.16)
Excellent health	-.533	(4.49)	-.458	(4.79)
Very good health	-.276	(2.67)	-.224	(2.61)
Good health	-.090	(0.95)	-.171	(2.08)
Arthritis	.122	(1.00)	-.522	(4.15)
Cancer	.272	(1.15)	.344	(1.56)
Diabetes	.360	(2.10)	.116	(0.66)
Heart disease	.072	(0.40)	.1343	(0.80)
High blood pressure	.044	(0.39)	-.262	(2.60)
Lung disease	-.131	(0.53)	.304	(1.58)
Stroke	.216	(0.47)	.149	(0.43)
Family income	.0018	(0.19)	.0094	(1.50)
Wealth	.0002	(0.51)	.0003	(1.11)
Education	-.024	(1.80)	-.020	(1.83)
Ever smoke	-.054	(0.49)	.189	(2.30)
Currently smoke	.179	(1.90)	-.191	(2.53)
Number of cigarettes	.009	(3.71)	.002	(1.05)
Female	-.070	(1.05)	.002	(0.03)
Black	-.364	(4.08)	.107	(1.67)
Hispanic	0.160	(0.59)	-.015	(0.08)
Change in stock wealth	.0002	(0.16)	-.0017	(1.91)
Cons	-1.301	(6.53)	-.839	(5.16)

	Major Onset 95-99		Minor Onset 95-99	
	Coef	z	Coef.	z
Age 51-61	.612	(8.20)	.144	(2.17)
Age 62	.847	(9.11)	.159	(1.84)
Excellent health	-.397	(3.91)	-.231	(2.77)
Very good health	-.311	(3.39)	-.185	(2.36)
Good health	-.110	(1.28)	-.005	(0.07)
Arthritis	-.010	(0.09)	-.542	(4.71)
Cancer	-.263	(0.95)	-.396	(1.56)
Diabetes	.250	(1.50)	-.276	(1.54)
Heart disease	-.163	(0.89)	.046	(0.29)
High blood pressure	.227	(2.48)	-.233	(2.67)
Lung disease	-.162	(0.70)	.281	(1.63)
Stroke	-.143	(0.09)	-.565	(1.50)
Family income	-.0068	(0.96)	.0072	(1.30)
Wealth	.0004	(1.24)	.0003	(1.12)
Education	-.029	(2.57)	-.032	(3.34)
Ever Smoke	-.056	(0.59)	-.111	(1.47)
Currently Smoke	.227	(2.71)	-.008	(0.12)
Number of Cigarettes	.007	(2.70)	.008	(3.61)
Female	-.063	(1.21)	.048	(1.05)
Black	-.098	(1.45)	.186	(3.32)
Hispanic	.043	(0.37)	.267	(1.98)
Change in stock wealth	.0006	(2.47)	.0005	(1.47)
Cons	-1.019	(5.83)	-.460	(3.16)

Models also control for Region of Residence and marital status and change in marital status between the waves. Z values are based on robust standard errors. Financial variables measured in $10,000 units. NA means no coefficient estimated for that variable.

Table 8. Does SES Predict Future Major Onset
F-tests (Probability)
1989 baseline – 10 year forecast

	Major onset		Minor onset	
Income lag1	0.14	(0.71)	0.00	(0.95)
Income lag 2,3,4	4.24	(0.24)	4.45	(0.22)
Income lag 1,2,3,4	4.24	(0.37)	6.05	(0.20)
Health lag1	6.00	(0.11)	17.67	(0.01)
Health lag 2,3,4	24.19	(0.01)	33.73	(0.00)
Health lag 1,2,3,4	52.49	(0.00)	77.90	(0.00)
Wealth	0.01	(0.90)	1.70	(0.19)
Change in Stock Wealth	7.36	(0.01)	2.39	(0.12)
Ed	5.23	(0.02)	5.49	(0.02)

Based on probit models with the same set of variables as in Tables 5-7 with the addition of these lags on income and self-reported health status. Based on robust standard errors.

Table 9 Does SES Predict Future Onsets by Age?

	Financials*	Education Coefficient	Education Z Score
1984 Baseline Models			
Age Group <40			
Major 85-94	0	-.037	-1.61
Minor 85-94	0	-.042	-2.61
Major 85-99	0	-.024	-1.32
Minor 85-99	0	-.038	-2.87
Age Group 40-60			
Major 85-94	0	-.020	-1.21
Minor 85-94	0	-.006	-0.37
Major 85-99	0	-.049	-3.14
Minor 85-99	1+	-.035	-2.41
Age Group > 60			
Major 85-94	0	.006	0.22
Minor 85-94	0	-.009	-0.41
Major 85-99	0	.012	0.53
Minor 85-99	0	-.012	-0.56
1989 Baseline Models			
Age Group <40			
Major 90-99	1+	-.033	-1.60
Minor 90-99	0	-.035	-2.41
Age Group 40-60			
Major 90-99	0	-.053	-3.27
Minor 90-99	0	-.026	-1.97
Age Group >60			
Major 90-99	1+	-.027	-1.59
Minor 90-99	0	-.014	-0.85

* This column counts the number of statistically significant coefficients for the three financial variables-income, wealth, and changes in stock market wealth- and indicates the signs of the significant coefficient. Models include all variables included in models in Tables 5-7.

Table 10- Estimated Effects of Education on Future Onsets

	All		Respondents Only	
	Coefficient	Z value	Coefficient	Z value
1984 Baseline				
Onset 1-10 years	-.024	(2.62)	-.020	(1.81)
Onset 1-15 years	-0.38	(4.43)	-.028	(2.70)
1989 Baseline				
Onset 1-10 years	-.029	(3.54)	-0.18	(1.86)

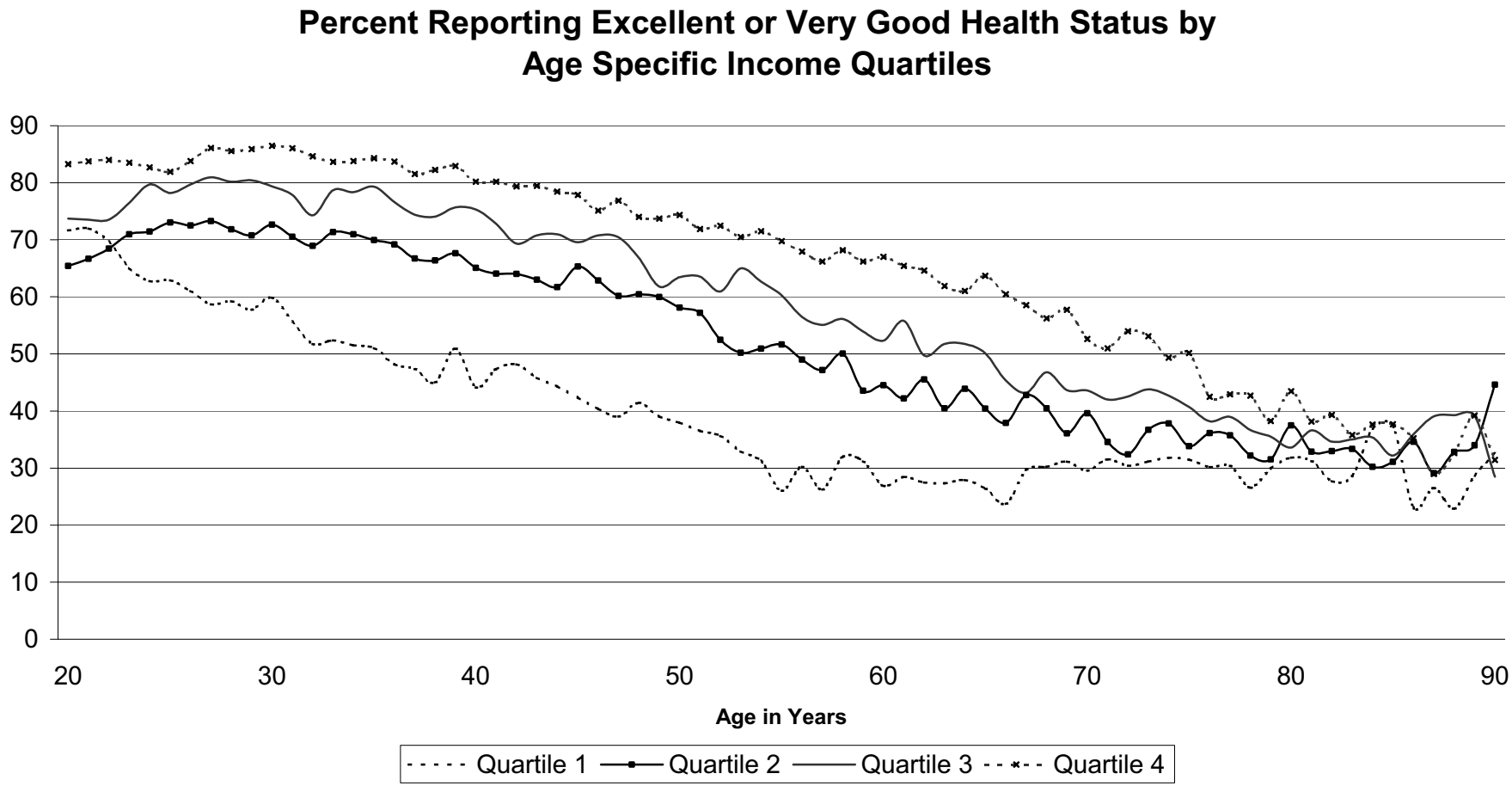
Percent Reporting Excellent or Very Good Health Status by
Age Specific Income Quartiles
90
80
70
60
50
40
30
20
10
0
20
30
40
50
60
70
80
90
Age in Years
Quartile 1
Quartile 2
Quartile 3
Quartile 4

Figure 1

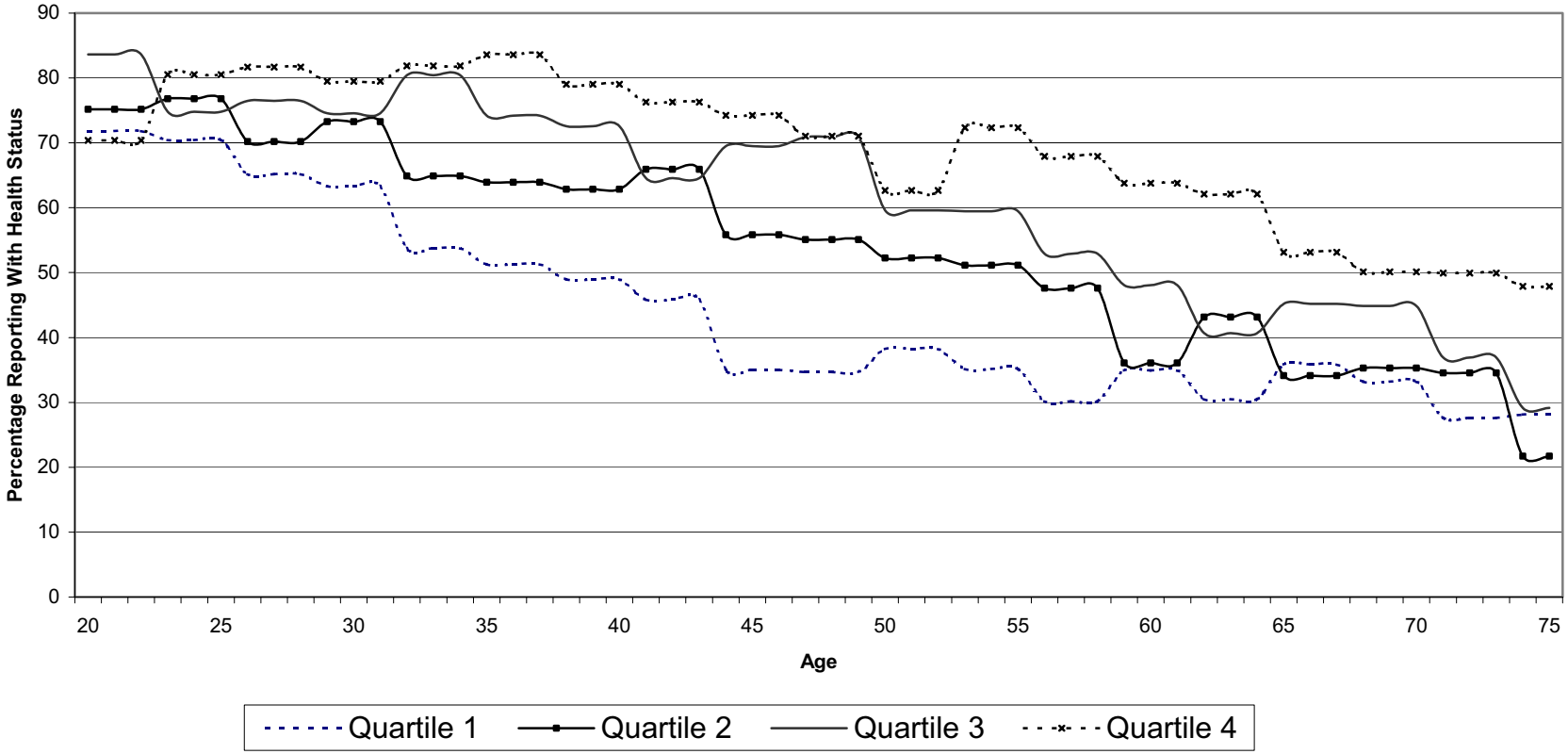

Figure 2. Percent Reporting Excellent or Very Good Health by Age-Specific Wealth Quartiles

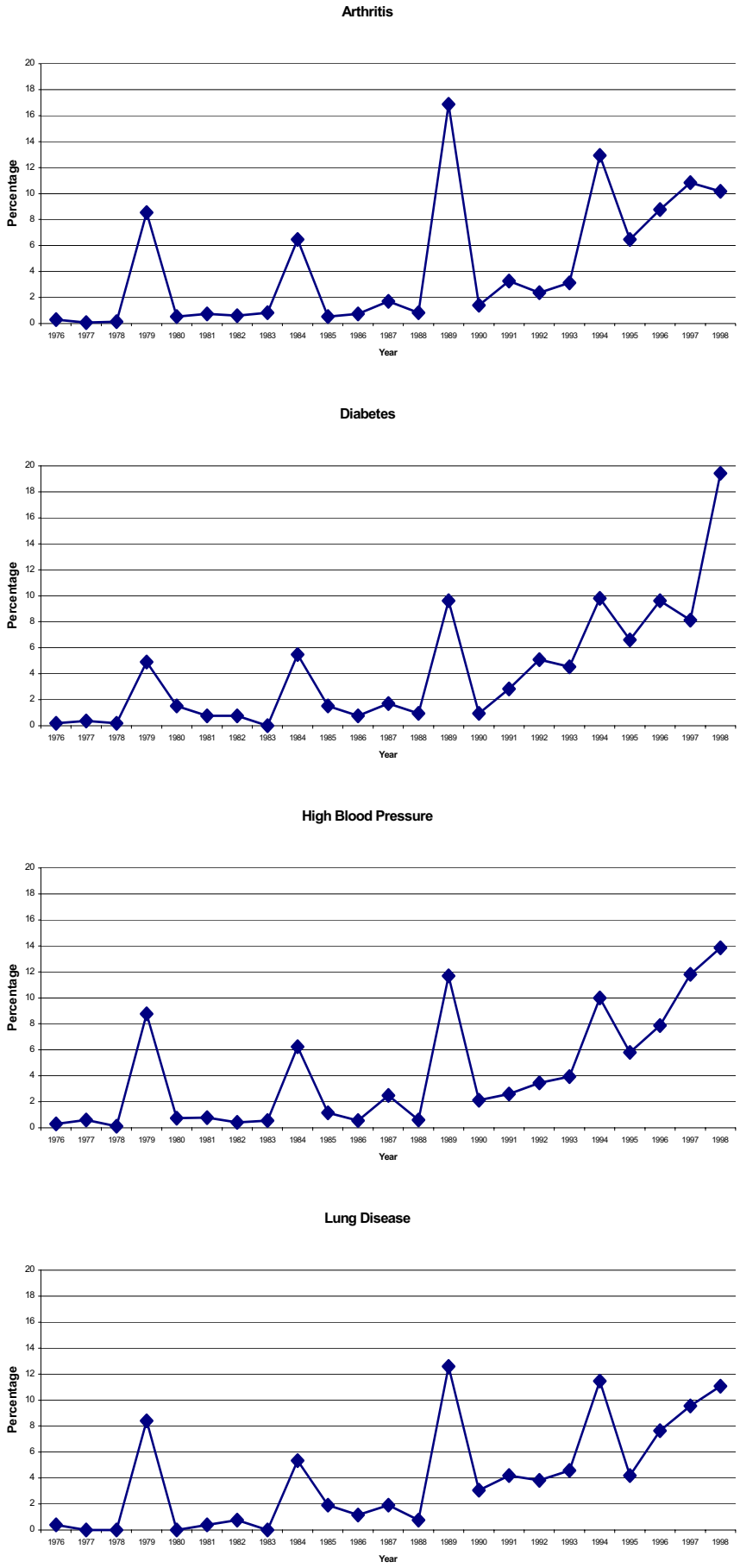

Figure 3. Date of Onset of Chronic Condition (1)

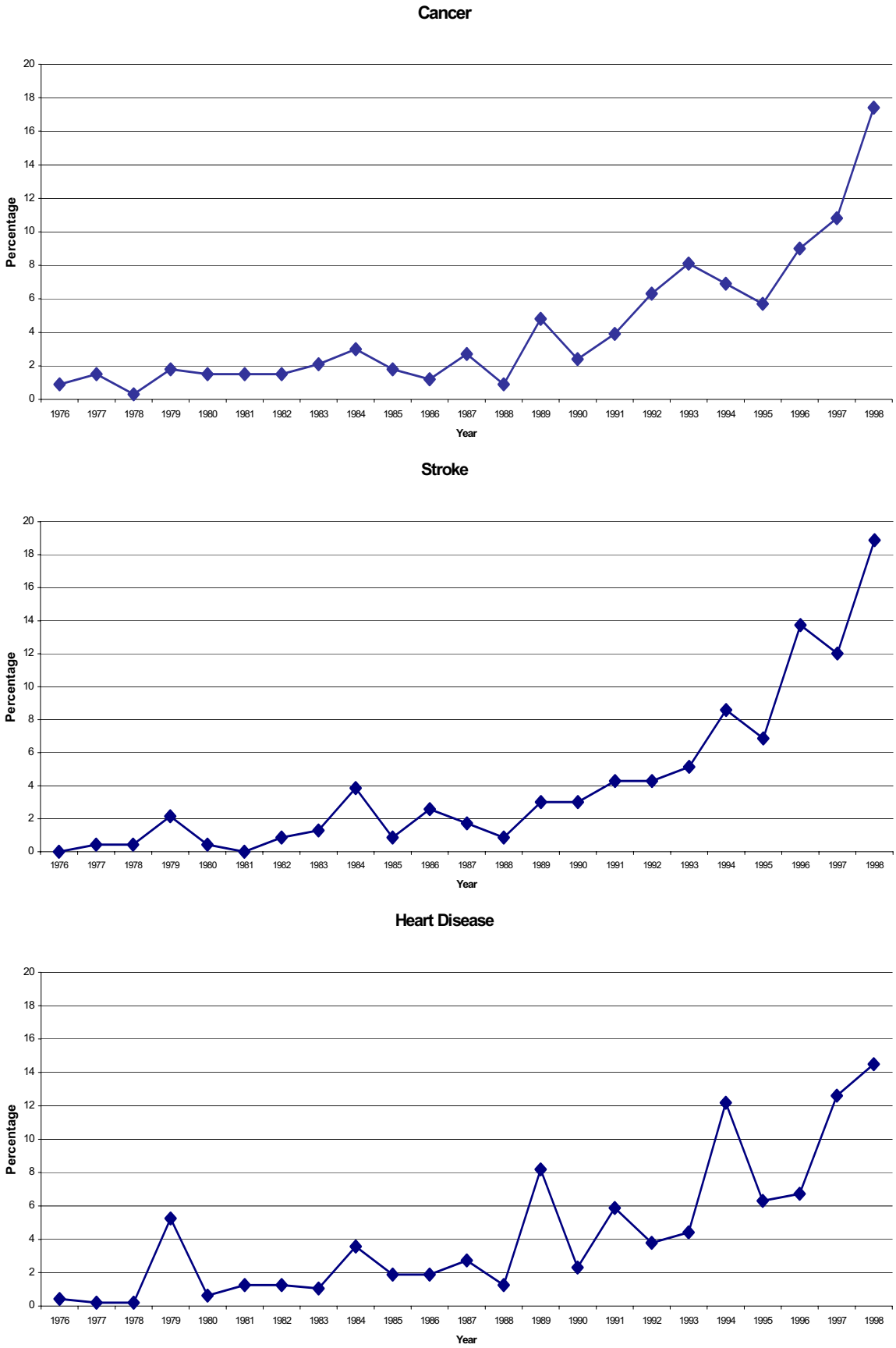

Figure 4. Date of Onset of Chronic Condition (2)