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THE IMPACT OF CHILDHOOD HEALTH ON ADULT LABOR MARKET OUTCOMES*

Abstract:

This paper examines the impact of childhood health on a series of SES outcomes observed during adulthood. These outcomes include levels and trajectories of education, family income, household wealth, individual earnings and labor supply. The analysis is conducted using unique data that collects these SES measures starting with a panel who were originally children and who are now well into their adult years. Since all siblings are also included in the panel, one is able to control for all unmeasured family and neighborhood background effects. With the exception of education, I find that the poor childhood health has a quantitatively large effect on all these outcomes. Moreover, these estimated effects are much larger when unobserved family effects are controlled. (JEL codes; I_{10}, J_{00})

There is renewed scholarly and policy interest in why people of lower socio-economic status (SES) have worse health outcomes. No matter which measures of SES are used (income, wealth, or education), the evidence that this association is large is abundant (Michael Marmot (1999), James Smith (1999)). A central issue in this debate concerns the extent to which childhood health status affects levels and trajectories of some of the most central measures of SES including education, income and wealth during one's adult years. However, estimating this relationship is difficult, as it requires data that follow individuals from their childhood years into at least a significant component of their adulthood, all the while simultaneously measuring health and these key SES concepts. Since unmeasured background attributes of the family, neighborhood and environment in which people are raised are central determinants of their adult outcomes and trajectories, the data must permit some method of controlling for them, especially that undoubtedly large sub-component that is unmeasured in most surveys.

This paper uses some unique data derived from the Panel Survey of Income Dynamics that has followed groups of siblings and their parents for as long as thirty years. Throughout that period, information on education, income, wealth, and health were collected mostly prospectively on all parties. Most important, following siblings from the same family offers a very unique opportunity to control for unmeasured family and other background effects common to children raised in the same family. Using this data, I present estimates that indicate that health conditions during childhood have quantitatively large impacts on virtually all the key adult indicators of socioeconomic status that are used by economists.

This paper is divided into three sections. The first briefly summarizes some recent contributions by economists on this question of the origins and impact of childhood health on adult outcomes. Section 2 highlights the salient strengths and weaknesses of the data that will be

used in the analysis. The final section contains empirical models that investigate the impact of family background and childhood attributes on latter life health and several central SES outcomes and trajectories. In particular, these analyses will focus on the question of the impact of childhood health on latter adult outcomes, including completed levels of schooling and household and individual income and earnings, and household wealth.

I. Background

The recent revival of interest in the determinants and consequences of poor health during childhood can be traced in part to the impact of a series of studies by David J. Barker and his associates (1997). In that work, they provide evidence that even nutrition in utero impacts health outcomes much later during one's adulthood. Some of this research is derived from animal studies where purposely-deprived fetal growth has been shown to affect subsequent mortality. Data from some natural experiments also lend some support to this view. For example, Ravelli et al (1998) studied people born in Amsterdam who were exposed pre-natally to famine conditions in 1944-1945. When compared to those conceived a year before or after the famine, prenatal exposure to famine, especially during late gestation, was linked to decrease glucose tolerance in adults producing higher risks of diabetes.

In the last few years, economists have done some insightful work on this topic. For example, using data from two health surveys (NHANES III and the National Health Interview Survey) as well as the PSID child development supplement (PSID-CDS), Anne Case, Darren Lubotsky, and Christina Paxson (AER, 2002) demonstrated that the relationship between SES and health has it origins in early childhood and even expanded as children age. They report a strong relationship between parental income and several salient measures of child health including common childhood chronic health conditions, a relationship that accumulated as

children age. This relationship persisted even after controlling for other background characteristics, including parental education.

Janet Currie and Michael Stabile (AER, 2003) report similar findings about the importance and shape of the child health-SES gradient using Canadian data. They argue that the gradient emerges when people become adults not because lower SES children have a more difficult time recovering from a health shock but rather that low SES children receive more frequent health shocks during their childhood. They also report that poor childhood health disadvantages children in part to lower cognitive and academic achievement as indicated by higher probabilities of grade repetition and lower math and reading test scores among children in poorer health.

In a subsequent study, Case, Fertig, and Paxson (2004) investigated the persistent impacts of childhood health on adult health, employment, and some measures of SES using a 1958 British birth cohort who were followed prospectively into their adult years. They find that people who experienced poorer health outcomes either in terms of low birth weight or the presence of chronic conditions when they were children not only had worse health as adults, but they passed fewer O-level exams, worked less during their adult years, and had lower occupational status at age 42 even after some standard key measures of parental background such as education and income were controlled for.

A number of other studies also point to the potential long-lasting importance of childhood health for adult outcomes. For example, using data from the 1946 British cohort which following a sample of people born during one week in England, Pless et al. (1989) demonstrated a relationship between chronic illness in childhood and lower educational attainment by age 26 and a higher risk of unemployment by age 36. Similarly, Currie and Hyson (1999), using data

from the 1958 British cohort, found that lower birth weight had a negative impact on education and employment by age 33.

But despite the rich set of controls available especially in these prospective British cohort studies, these studies can not rule out the possibility that the role of child health in determining adult SES, are in fact driven by other, unobserved characteristics of the family or home environment that are correlated with child health and unmeasured family background effects. For example, Allison Currie, Michael Shields, and Stephen Price (2004) estimate that among a sample of English siblings 1997-2002, as much as 60% of the total variation in child health might be explained by unobserved family effects.[1] Some of the child health effects identified in the papers cited above could be due to similar unobserved heterogeneity in the cohort study data.

II. 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. For example, details on family income and its components and labor market activity 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. The PSID is rightly recognized as one of the premier general-purpose panel survey measuring several key dimensions of adult labor market outcomes.

The PSID has not been known as a health survey but that situation may change. It has been collecting information on self-reported general health status (the standard five-point scale

from excellent to poor) since 1984 and starting in 1999 and for all subsequent waves, information was gathered 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.

The PSID permits a unique long-term perspective on the impact of childhood health. Given that many panel members have been in the PSID since 1967, the entire past sequence of financial situation can be exploited. This is especially important for those who were children in the original panel or born thereafter. When these children become panel members on their own, we have SES histories yearly for them and their parents. Since the same applies to their siblings, the PSID offers a very unique opportunity to model and control for family background effects.

The PSID has both advantages and disadvantages for studying the impact of childhood health on latter life outcomes. There are two main disadvantages. The most obvious is that variables that measure health conditions during the childhood years are quite limited and are available only for those PSID respondents who were adults in the 1999 panel wave. In particular, one must rely on a single index—a retrospective self-evaluation using the standard five-point scale (excellent, very good, good, fair, or poor) of the general state of one's health when one was less than 17 years old. This question was asked in the 1999 wave of the PSID for the respondent and spouse.[2] A counterpoint to the obvious limitations of using a single health index is that this one attempts to obtain a summary measure of the complete childhood health experience, a useful concept to have in mind.[3] Some potential biases in using this childhood health question are discussed below.

The second disadvantage is that to have the most comprehensive measures of family background one must select children of the original PSID respondents which implies that adult

outcomes are necessarily limited to early and mid adulthood. For example, a 16 year old living in the parental home in 1968 would only be approaching age 50 by the critical 1999 wave. If we take age 25 as the minimum cutoff for monitoring adult outcomes, we can only include all children 16 and younger in 1968 and all children subsequently born up to 1974.

Fortunately, the advantages are also unique. First, the PSID is the premiere American panel data source for the two most important financial SES measures (income and wealth) for a sample that spans the complete age distribution. Most important, it also offers a rare opportunity to examine generational and family effects. As part of its panel structure, the PSID follows all family members of the 1967 panel of respondents as well as any new family members who arrive subsequently. Thus any children of the original PSID panel members as well as their siblings are also considered members of the panel.

For these PSID panel children and their siblings, the full array of SES measures potentially exist each year not only for both themselves and their parental households from which they came but also for their siblings when they become independent households. For this sample we have their own and their siblings income histories up to the 1999 wave as well as that of their parents (the original PSID sample members). We would also have for all siblings and parents the type of health data described above for all PSID respondents. In particular, for those now adult households with surviving parents and adult siblings in 1999, we potentially have the available information for both on general health status since 1984 (since the time they were independent households) and the 1999 wave battery on the onset of chronic conditions.

III. Empirical Models of the Impact of Family Background and Childhood Health on Adult SES

This paper rests on three substantive points. The first is that outcomes that we observe throughout one's adult years often are the legacy of events that took place in one's childhood and that health is one of the best examples of that link. Figure 1 provides evidence of this link by plotting contemporaneous self-reports of general health status by age against self-reports about general health status during childhood. For both childhood and adult health, good health is defined as a report of excellent or very good and all other categories are labeled bad. Individuals who were in better health during their childhood years have health levels and health trajectories that are much above those whose childhood health was not good.

The second and third points are that this link between childhood and adult health outcomes are the consequence of many observable and unobservable attributes of one's family background during childhood. The importance of the impact of unmeasured family effects is the principal topic of this paper and these are discussed in detail in subsections A and B below. To document this point for measured family effects, Table 1 summarizes results from a simple model of the generational transmission of health through disease prevalence. In addition to standard demographic attributes, these models include controls for respondent's age (entered as a quadratic), mother's and father's education, and the ln of the average parental income computed during all of a respondent's childhood years that were available (from birth to age 18 across the years present in the PSID panel). In this paper, all financial variables are measured in 2001 dollars. The childhood health variable is a dummy that indicates whether respondents' health during the childhood years up to age 16 was either excellent or very good.

Since there are not sufficient data to conduct a disease-by-disease analysis, two adult health outcomes are modeled—whether the respondent has a chronic condition rated severe or one rated minor. Severe conditions were defined as cancer, heart condition, stroke, and diseases

of the lung. All other onsets (hypertension, arthritis, diabetes) are defined as minor. To isolate the possibility of inter-generational transmission of disease, two variables are added to the model indicating the at least one parent has a major chronic condition and that at least one parent had a minor chronic condition.[4] This analysis cannot tell us whether these generational health transmissions are the consequence of shared biology or shared environment, as any estimated linkage is some combination of these mechanisms.[5]

The estimates contained in Table 1 suggest an important role for family background in adult health. To use just one example, higher incomes of parents when one was a child does appear to lower the probability of having a chronic conditions during adulthood although this effect is only statistically significant for the more severe diseases. Similarly whether through biology or through the common impacts of a shared family environment or shared health behaviors, the diseases of the parents also appear to be associated with those of their adult children. Since the ages of the adult PSID children in the sample used in Table 1 may still be too young to have shared severe common conditions with their parents, these forms of transmission are more readily transparent for minor chronic conditions. Finally, there is evidence of the long legacy effects that health problems have spanning from childhood into mid-adulthood. For both minor and severe adult chronic conditions, the odds of having health problems as adults are correlated with general health status during childhood.

A. Estimates on 1999 SES levels

The primary goal involves estimating the impact of childhood health on several salient SES outcomes measured during the adult years. This inquiry begins with four key SES outcomes—completed years of schooling, ln family income, household wealth and individual

earnings, all of which are measured in the 1999 PSID. In addition to a standard list of demographic controls of the respondent (an age quadratic to capture a combination of life-cycle and possible cohort effects), race (=1 if Black), Hispanic ethnicity (= 1 if Latino), and gender =1 if female)), all models include measures of the background of the family in which the PSID respondent was raised (education of both the mother and father, the average ln parental income during the years when the child was less than 17 years old[6], and the number of siblings).

The sample on which these models are based are those PSID respondents present in interview wave 1999 who were no more than 16 years old in 1968 or who were subsequently born into the PSID between the years 1968 and 1974. The results obtained from these models are listed in Table 2 (education), Table 3 (ln family income and household wealth) and Table 4 (individual earnings). The baseline model in each table provides estimates of the magnitude of mean differences across demographic groups, the coefficients and t values listed in the extended model columns add the set of family background variables, while those in the within sibling columns are fixed effects estimates using differences amongst the siblings of these PSID families. The 't' statistics in parenthesis are based on robust standard errors.

It is useful to begin with adult education, which is the most common outcome investigated in previous research. The first set of columns in Table 2 simply documents the age adjusted average difference in mean schooling among the principal demographic sub-groups in the PSID. On average African-Americans trial whites by 1.2 years of schooling, Hispanics lag about half a year beyond whites, and women's education exceeds that of men by about one-tenth of a year. All family background variables in the extended model listed in the middle columns behave as expected and are consistent with the prior literature—higher education of either parent and greater levels of parental income all significantly increase adult schooling levels while a

larger number of siblings depresses adult schooling achievements. In fact, these measured family background effects are sufficiently strong that they more than fully explain the racial difference in schooling in the PSID. Controlling for these measured family background effects, the only statistically significant difference among the demographic groups involves women, who complete about .2 of a year more schooling than men do.

The central variable of interest concerns the estimated impact of respondent's self-reported health during the childhood years. The estimates contain in the extended model indicate that respondents whose self-reported that their health was excellent or very good achieved a third of a year more schooling than those whose health was worse than that, a result that is both statistically significant and consistent with the prior literature summarized earlier. While it is often argued that one advantage of using schooling as a SES measure is that it eliminates the possibility of reverse causality from health to SES, these results indicate that this argument may be overstated since early life health (which is correlated with latter life health) may alter adult educational achievements.[7]

However, one problem in assigning these effects to childhood health is that there remains the strong possibility that there remain other as yet unmeasured dimensions of family background that may be correlated both with adult education with childhood health. Fortunately, PSID offers an unusual opportunity to control for some of these unmeasured family traits since all siblings from the original PSID families are potentially included within its sampling frame. The final two columns take advantage of this aspect of the PSID design by presenting across sibling within family fixed effects estimates. The standard expectation is that due to common unobserved family effects, which are correlated both with childhood health and adult SES outcomes, that within family estimates of the impact of childhood health would be smaller. This

indeed is what was found for education where the magnitude of the impact of childhood health on adult education was cut more than in half and is no longer statistically significant.[8]

A distinct advantage of the PSID is that one can also investigate the impact of early childhood health on financial economic outcomes such as income and wealth. Table 3 provides the first such test by using ln household income from the 1999 wave as the dependent variable.[9] The two most important demographic controls in terms of the magnitude of impacts are race and currently married. Even with background family variables included in the middle columns, African-Americans family incomes are about 8 percent less than the white majority and, not surprisingly given the combination of incomes, married individuals have much higher household incomes than individuals who were not married in 1999. Once again measures family background variables behave as expected—individuals enjoy higher family incomes when they come from families whose incomes were also high when they were children, when their parents have higher levels of education, and when they have fewer siblings.

Even after controlling for the influence of childhood health on subsequent education as an adult, childhood health is strongly related to adult household incomes. Moreover, the size of this effect is decidedly non-trivial. To illustrate, even when we condition on adult education, which is negatively affected by poorer childhood health, household income is 13 percent higher amongst those whose childhood health was excellent or very good. When this model is estimated not controlling for completed adult education, the magnitude of this effect rises to 17 percent (results not shown).

One reason why childhood health may impact later life financial outcomes is that childhood health may be correlated with parental health, which as we have seen from Table 1 is itself correlated with adult health of children. A strength of the PSID is that dimensions of

parental health are recorded since the parents were or still are PSID panel members as well. To check this possibility, I added measures of parental health (whether alive, age of death if not, prevalence of major and minor illness of each parent) to the extended model estimated in Table 3. The estimated coefficient on childhood health with these parental variables included was 0.126 compared to the 0.130 estimate listed in the extended model contained in Table 3. Thus, it seems unlikely that the impact of childhood health on adult outcomes is simply picking up an indirect effect of the health of the parents that we can measure transmitted to their children.

However, there are many other aspects of unobserved family background that are not measured and that brings us to the within sibling estimates listed in the next two columns of Table 3. Unlike education, within sibling estimated impacts of childhood health on ln adult family income are now actually higher and non-trivially so than those contained in the extended model. The within sibling models indicate that better health during childhood increase adult family incomes by 24% compared to the 13% estimate when only measured family background variables are included. I will offer some explanations for the larger estimated effect from within sibling models below. A comparison of within sibling models in Tables 2 and 3 does indicate that much of the impact of poor health during childhood on financial success as an adult apparently is not mediated through education.

Another financial measure available from the PSID is total household wealth.[10] The last four columns of Table 3 present models estimating the impacts of background attributes including childhood health on total household wealth in 1999. Coefficient estimates for the covariates largely mimic those from the existing literature. The key addition relates to the summary of childhood health. Being in good or excellent health as a child has a positive impact on wealth at all these quantiles, but the estimated impact increases significantly as we move

above the median. I estimate an impact at the mean of about $2,000 but once again this estimated impact is five times larger in within sibling models.

The differences that emerge in family income and wealth associated with childhood health could reflect several underlying reasons. Some may show up in individual earnings either through skill (wages) or work effort. Not only may poor health during childhood affect the amount of work effort as an adult, more generally poor childhood health may limit energy levels most commonly associated with higher levels of financial success. One form in which this may take place is lower level of human capital investment and lower wage growth. Since the financial outcome in Table 3 is family income, poorer childhood health may also have effects that operate through marriage markets. A history of poor health during childhood, especially that component which has lasting health consequences, is probably not a positive trait in attracting a life partner so that one may end up marrying a lower income mate.

To examine these mechanisms, Table 4 models the impact of childhood health on both individual arithmetic and ln earnings. Since the sample used for arithmetic earnings includes respondents with zero earnings while the ln model is limited to respondents with positive earnings, one difference between them is that ln models are confined to labor market participants. Given that earnings includes some labor market effort dimension, it is unsurprising that earnings differences using either functional form are much larger for women than for men. Parental income during childhood is the most important predictor amongst all family background measures, a reflection of the well-established inter-generational correlation in incomes.

As was true for family income, better childhood health is associated with higher levels of own earnings as an adult. For example, in the ln specification, earnings are 12 percent higher amongst those in excellent or very good health during childhood, a difference that is $3,348

when the arithmetic metric is used. As I found for family income, these estimated impacts are much larger using the within sibling estimates—25% and $8,107 in the ln and arithmetic specifications respectively. Once again, I will offer some explanations for this below.

One reason these income impacts are so large is that childhood health has effects on the ability to work. Table 5 lists the estimates from a parallel set of models using annual weeks worked in 1999 as the outcome. Those whose childhood health was rated excellent or very good worked more than two weeks more than those whose childhood health was not as good, an effect that is 4.3 weeks in the within sibling model.

While these effects on own earnings are non-trivial in magnitude, they remain somewhat smaller than those estimated above for family income. A larger effect of poorer childhood health on family income is consistent with part of the impact operating through marriage markets as a childhood history of poor health results in marriage to a lower income mate. An indication that this is part of the explanation is that the correlation in our measure of childhood health across spouses is 0.34. To test this further, I estimated a parallel set of models among those who were married where the outcome was spousal earnings. In addition to the characteristics of the spouse (education, age, race, ethnicity and gender), these models included the full set of background attributes of the respondent included in Table 4 and the education of the respondent. Being in excellent or very good health as a child is associated with a $2,367 increase in spousal earnings ("t" statistic = 1.9). Assortative mating in the marriage market through the adult manifestations of any problems due to poor health during childhood is apparently one reason for these family income impacts of childhood health. [11]

B. Estimates on initial SES levels and subsequent growth rates

An important question not addressed by calendar year 1999 models is whether poor health during childhood only affects where one starts as a young adult or whether there are incremental effects latter. This question is examined in two complimentary ways in Tables 6 and 7. The first two columns of these tables are based on models with the same explanatory variables used in the calendar year 1999 models but the outcomes examined now are the ln of household income and the ln of individual earnings when the respondent was age 25.[12] The models in the two middle columns of Tables 6 and 7 examine more directly percent change in incomes between age 25 and calendar year 1999. In these models, the age 25 level of the outcome is included as a co-variate. The final columns in these two tables list the within sibling variants of these change models. As before, all financial variables are in real 2001 dollars.

If one compares estimated coefficients obtained for the demographic and family background variables for predictions of ln household income and ln individual income at age 25 to their year 1999 level counterparts contained in Tables 3 and 4, the magnitudes are by and large quite similar.[13] This implies that much (but as we shall see in a moment not all) of what happens to people in terms of the systematic component of their percent income trajectories as influenced by family background were right there at the beginning.

Given the focus of this paper, estimates of the effect of child health are of most interest. In this case, the magnitudes of estimated effects while strong at age 25 are somewhat below what were estimated in the earlier year 1999 estimates. The models that estimate changes between age 25 and calendar year 1999 provide more direct evidence of the process at work. For both household and individual incomes, better health during childhood not only is associated with higher income levels at age 25 but it also raises subsequent income paths after age 25 during adulthood. For example, Table 6 indicates that about two thirds of the overall impact on poorer

childhood health on adult family income is present at age 25 while the remaining one-third is the consequence of slower post age 25 family income growth. The division for individual earnings between initial and subsequent individual earnings captured in Table 7 is about fifty-fifty.[14]

As was the case with all previous models, within sibling estimates in Tables 6 and 7 indicate that the size of the predicted impact of child health is larger when within sibling models are estimated.[15] Why might all these estimated effects be larger in within sibling models, with the sole exception of the estimated impact of childhood health on own education?

There are several possible explanations. One reason is behavioral—children's education is much more in control of parents so their ability to engage in compensatory behavior in offsetting the impacts of poor health on one of their children should be greater for adult education than for the incomes of children when they become adults. By itself, however, this could only explain a smaller family effect bias on income than on education. It does not explain why the bias is in the opposite direction for income.

Another possibility involves the measurement of childhood health. There is growing evidence that when answering questions about whether or not their health is excellent, very good, good, fair or poor individuals use different standards or thresholds in placing their health with these thresholds (see Arie Kapteyn, James Smith and Arthur VanSoest (2005)). A similar set of issues would no doubt apply to our measure of childhood health, which relies on the same type of subjective ordinal scale. Thus, some of the differences in childhood health across people will reflect differences in these thresholds (which should have no effect) rather than real differences in their underlying health as children. Our estimate of the impact of childhood health would reflect a combination of these two sources of variation.

Since attitudes about what constitutes good and bad health are partly formed by families, these thresholds are likely to be more similar within than across families. If so, differences in self-reported childhood health among siblings will signal a bigger difference in true childhood health than health differences in the population as a whole which may contain a good deal of family differences in reporting thresholds. If siblings in a family shared some family component of reporting threshold on self-reported childhood health, eliminating that difference in the reporting threshold with within sibling models would show up as a larger real health difference.

A third reason is also behavioral. In within sibling models, all families with only one adult child in the PSID are excluded. If the impact of childhood health interacts positively with the number of siblings, the estimated mean impact of childhood health must be larger in within sibling models. To check this possibility, all models were re-estimated adding an interaction between childhood health and the number of siblings. These results obtained for both the main effect of childhood health and the interactions of childhood health with number of siblings are summarized in Table 8.[16] In general, the intercept terms on childhood health are not statistically different from zero but the interactions of good childhood health with numbers of siblings are all positive and statistically significant.

These estimates imply that the negative lifetime impacts of having a childhood characterized by poor health is most severe when there were a larger number of siblings in the family, the rest of whom were in relatively good health. The ability of families to use their resources to compensate a sickly child may be more constrained when there are many competitors for the limited resources.

The estimates presented in this paper clearly depend on the ability of 1999 PSID respondents to accurately remember and report their health during their childhood years. There

are several legitimate reasons for some concern. First, a feature of the PSID is that one member of the family responds for both spouses. In about one-quarter of the cases, a spouse is responding for an original PSID child who is now a married adult. It is reasonable to wonder how well this person recalls the childhood health of their spouse. As a check of the robustness of the results, Table 9 presents side-by-side listings of the estimated impacts of childhood health for all respondents and for those respondents reporting about themselves only. As expected if childhood health is more accurately reported for oneself, the general tendency is for the estimated coefficients on children health on adult outcomes to actually become larger in models estimated across reporting respondents only.

Another potential issue is that individuals used current or recent outcomes either in terms of their financial position or their health and then project those realizations backward into their memory when asked in 1999 of how their health was as a child. If that was the dominant effect, then poor financial outcomes or poor health conditions in 1999 or in the years immediately proceeding 1999 would actually be affecting the 1999 reporting of what one's health was as a child—the reverse of the interpretation given in this paper.

While some of this may well be going on, there are two tests that suggest that it is not the dominant pathway. The first test used PSID measures of financial outcomes (family income or individual earnings) in years earlier than 1999 as the dependent variable. The results obtained when other years are substituted are quite similar to those reported in this paper. The second test takes advantage of the fact that PSID respondents were reporting their general health status contemporaneously in earlier years well before their 1999 description of their childhood health. These earlier reports should not have been influenced by subsequent health shocks including those close to calendar year 1999. For example, respondents who were between 35-39 years old

in 1999 reported their general health ten years earlier in the PSID waves of the late 1980s. The faction of those who said their health was excellent or very good when they were in their late 20s was 67% among those who said in 1999 that their general health status as a child was good compared to 50% who said their general health status as a child was not good.

IV. Conclusions

In this paper I use some unique PSID data that has followed prospectively a group of children as well as their parents from their childhood years well into their adulthood. These data allow one to estimate the impact of poor health as a child on a series of adult outcomes including education, income, and wealth. More importantly, by estimating differences amongst the siblings these data allow one to control for unobserved family level heterogeneity, a problem that has plagued previous research on this topic.

An individual's general health status during childhood appears to have significant direct and indirect effects on several salient adult SES financial outcomes, including one's ability to earn in the labor market as well as total family income. Some of this negative financial impact of poorer childhood health is felt immediately in lower levels of financial resources at the beginning of the adult years while another component is realized through lower growth rates with age in these financial resources. Part of these financial impacts appear to reflect a negative impact on adult labor supply as the lingering effects of poor childhood health are transmitted into poorer health as an adult which in turn reduces work effort. Some of the impacts on family income also appear to be the result of poor childhood health being associated with marrying a partner who also has lower earnings. Finally, I also produce evidence that poor heath as a child leads to lower levels of household wealth as an adult.

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Endnotes

[1] Their set of observable characteristics includes family income, parental education and employment status, birth weight of the child, and parental health measures.

[2] The question 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?"

[3] For example, Case, Fertig, and Paxson (2004) use the 1958 British cohort study which has much superior measures of childhood health including birth weight and childhood health conditions measured prospectively at various points in childhood and adulthood starting at birth. The tradeoff is that the measures of economic variables are not as strong as those in the PSID.

[4] I found no evidence that the transmission differed by whether the disease was the mother's or the father's so they are combined in these models.

[5] Since these measures only exist if the parent also survived to 1999, I also control for whether each parent is alive in that year and whether each parent has missing data for other reasons.

[6] This is measured over the years the child was observed in the PSID.

[7] Similar findings for adult years of schooling are reported by Case, Fertig, and Paxson (2004) and Case, Lubotsky, and Paxson (2002).

[8] All variables common to siblings drop out of the across sibling models. However, parental remains since these adult siblings were children were children at different points in time. However, the remaining variation in parental income is quite limited and is not a basis for estimating income effects. Therefore they are not discussed in the text.

[9] Cases with zero household income were excluded, but there are very few such cases.

[10] The definition of personal net worth in the PSID inclues housing equity, other real estate, autos, farm or business ownership; stocks, checking or savings accounts, C.D.'s, savings bonds

and IRA's; bonds, trusts, life insurance; and other debts. No attempts were made in the PSID to measure social security or pension wealth.

[11] The full set of these spousal estimates are available upon request.

[12] Other ages beyond age 25 were also explored but this had no substantive impact on the results summarized in the text.

[13] One difference is the age variable, but age serves a very different purpose in the age 25 models. Since everyone is evaluated at age 25 in these models, age in 1999 is a negative cohort or year index (the year one became twenty-five years old). Another difference is that the samples are somewhat different as the models in tables 6 and 7 place the additional restriction that income was observed at two points in time.

[14] I also estimated weeks worked variants of these change models. These estimates show no statistically significant impact of childhood health on weeks worked at age 25 but a depressing impact of poorer childhood health on the n the change in weeks worked between ages 25 and calendar year 1999.

[15] Similarly, the within sibling models of weeks worked indicate a larger impact on weeks worked of better childhood health

[16] The estimated effects of all other variables in the models were not affected by the addition of the interaction of childhood health and number of siblings so they are not included in this table.

Table 1. Probits Predicting Adult Disease Prevalence in 1999—PSID

	Severe Condition		Minor Condition	
Age 1999	-0.005	(0.07)	-0.021	(0.39)
Age squared 1999	0.003	(0.36)	0.001	(1.07)
Black	-0.260	(2.20)	0.048	(0.66)
Hispanic	-0.370	(1.61)	-0.103	(0.65)
Female	0.327	(4.38)	0.001	(0.02)
Health 16 EX-VG	-0.329	(4.13)	-0.195	(3.02)
Ln Parental Income Ages1-16	-0.168	(2.16)	-0.090	(1.58)
Mother Ed 12-15	-0.091	(1.05)	-0.165	(2.44)
Mother Ed College	0.012	(0.09)	-0.252	(2.28)
Father Ed 12-15	0.131	(1.45)	0.077	(1.14)
Father Ed College	0.048	(0.37)	-0.041	(0.42)
A Parent has Severe Condition	0.141	(1.62)	0.148	(2.26)
A Parent has Minor Condition	-0.019	(0.21)	0.152	(2.03)
Constant	0.098	(0.07)	-0.252	(0.24)

The sample consists of about 3053 PSID respondents present during the 1999 PSID wave who were also children of the original PSID respondents. These respondents were at most 16 in 1968 or subsequently born by 1984. Model also contains indicators for missing information on each parent, mother died and age of her death, father died and age of his death. The first column for each model lists the estimated coefficients from the probits. T statistics in parenthesis are based on robust standard errors.

Table 2. Predicting Adult Education in 1999 PSID

	Education in 1999					
	Baseline		**Extended**		**Within Sibling**	
Age 1999	-0.093	(1.27)	-0.012	(0.19)	-0.134	(1.44)
Age squared 1999	0.014	(1.42)	0.001	(0.66)	0.002	(1.55)
Black	-1.178	(14.3)	0.232	(2.57)		
Hispanic	-0.412	(1.88)	-0.206	(1.11)		
Female	0.100	(1.25)	0.196	(2.92)	0.303	(3.83)
Health 16 EX-VG			0.353	(4.28)	0.114	(1.15)
Ln Parental Income 1-16			0.779	(10.8)	0.363	(1.14)
Mother Ed 12-15			0.646	(7.45)		
Mother Ed College			1.216	(8.97)		
Father Ed 12-15			0.246	(2.89)		
Father Ed College			1.406	(11.9)		
Number of Siblings			-0.127	(6.52)		
Constant	14.99	(11.7)	3.429	(2.65)	11.24	(2.92)
N	3069		3055		2248	

The sample consists of PSID respondents present during the 1999 PSID wave who were also children of the original PSID respondents. These respondents were at most 16 in 1968 or subsequently born by 1984. "t" statistics based on robust standard errors. The fixed effects estimates place an additional restriction that there were at least two siblings who were PSID respondents in 1999.

Table 3. Predicting Adult Ln Household Income in 1999 PSID

	Ln Household Income 1999						Wealth			
	Baseline		Extended		Within Sibling		Extended		Within Sibling	
Age 1999	0.099	(4.98)	0.093	(4.62)	0.081	(1.80)	1,796	(2.56)	-1,299	(0.31)
Age squared 1999	-0.001	(3.93)	-0.001	(3.54)	-0.001	(1.41)	-19.4	(2.00)	44.8	(0.81)
Black	-0.289	(12.1)	-0.083	(2.87)			-2,605	(2.51)		
Hispanic	0.021	(0.35)	0.023	(0.40)			19.1	(0.01)		
Female	-0.070	(3.26)	-0.057	(2.72)	0.028	(0.73)	1.5	(0.00)	-1,000	(0.28)
Married in 1999	0.664	(28.7)	0.666	(29.5)	0.758	(18.2)	8,589	(10.9)	23,756	(6.24)
Health 16 EX-VG			0.130	(5.03)	0.240	(4.98)	1,847	(2.05)	10,005	(2.29)
Ln parental Income 1-16			0.227	(9.79)	0.011	(0.07)	4,283	(5.18)	11,647	(0.83)
Mother Ed 12-15			0.038	(1.37)			325	(0.34)		
Mother Ed College			0.044	(1.03)			-879	(0.58)		
Father Ed 12-15			0.060	(2.24)			-1,489	(1.60)		
Father Ed College			0.030	(0.79)			-1,017	(0.76)		
Number of Siblings			-0.017	(2.81)			-245.7	(1.15)		
Ed 1999	0.123	(23.3)	0.097	(16.7)	0.099	(7.68)	1,652	(7.98)	5,866	(4.96)
Constant	6.753	(18.9)	4.501	(11.1)	6.795	(3.62)	-96,531	(6.75)	-196,389	(1.16)
N	3047		3033		2222		2,750		2024	

The sample consists of about PSID respondents present during the 1999 PSID wave who were also children of the original PSID respondents. These respondents were at most 16 in 1968 or subsequently born by 1984. "t" statistics based on robust standard errors. The fixed effects estimates place an additional restriction that there were at least two siblings who were PSID respondents in 1999. When entered in the ln form, income had to be positive.

Table 4. Predicting Adult Earnings in 1999 PSID

	Individual Earnings 1999							
	Arithmetic Earnings		Within Sibling		Ln Earnings		Within Sibling	
Age 1999	2,448	(3.61)	1,762	(0.94)	0.102	(4.21)	0.058	(0.93)
Age squared 1999	-29.0	(3.10)	-15.33	(0.61)	-0.001	(3.55)	-0.001	(0.75)
Black	-885	(0.97)			-0.008	(0.23)		
Hispanic	-2,635	(1.35)			-0.042	(0.58)		
Female	-16,153	(22.7)	-19,461	(12.1)	-0.500	(19.8)	-0.551	(10.3)
Married in 1999	293	(0.39)	323	(0.19)	0.074	(2.75)	0.057	(0.99)
Health 16 EX-VG	3,348	(3.84)	8,107	(4.05)	0.123	(3.90)	0.248	(3.66)
Ln parental Income 1-16*	0.067	(5.73)	0.153	(1.91)	0.215	(7.67)	-0.023	(0.11)
Mother Ed 12-15	789	(1.02)			0.006	(0.17)		
Mother Ed College	-370	(0.15)			-0.016	(1.44)		
Father Ed 12-15	-443	(0.31)			0.049	(1.53)		
Father Ed College	677	(0.75)			0.050	(1.12)		
Number of Siblings	184	(0.40)			-0.015	(1.99)		
Ed 1999	3,189	(16.2)	4,058	(7.62)	0.111	(15.7)	0.123	(6.94)
Constant	-60,814	(5.04)	-69,041	(1.91)	4.502	(9.22)	7.549	(2.96)
N	3055		2248		2674		1862	

The sample consists of PSID respondents present during the 1999 PSID wave who were also children of the original PSID respondents. These respondents were at most 16 in 1968 or subsequently born by 1984. "t" statistics based on robust standard errors. The fixed effects estimates place an additional restriction that there were at least two siblings who were PSID respondents in 1999. When entered in the ln form, income had to be positive. * parents income is entered in arithmetic form in the arithmetic models.

Table 5. Predicting Adult Weeks Worked 1999 PSID

	Weeks Worked 1999					
	Baseline		**Extended**		**Within Sibling**	
Age 1999	0.559	(1.06)	0.769	(1.42)	0.831	(0.83)
Age squared 1999	-0.008	(1.06)	-0. 011	(1.42)	-0.011	(0.84)
Black	-1.436	(2.34)	-0. 884	(1.15)		
Hispanic	-1.627	(1.03)	-1.461	(0.93)		
Female	-6.896	(12.1)	-7.036	(12.5)	-7.309	(8.63)
Married in 1999	-0.765	(1.26)	-1.265	(2.10)	-1.652	(1.80)
Health 16 EX-VG			2.330	(3.36)	4.299	(4.05)
Ln Parental Income 1-16			0.801	(1.30)	4.615	(1.37)
Mother Ed 12-15			0.534	(0.73)		
Mother Ed College			-0.402	(0.35)		
Father Ed 12-15			0.516	(0.72)		
Father Ed College			-1.674	(1.67)		
Number of Siblings			-0.192	(1.17)		
Ed 1999			1.170	(7.51)	1.251	(4.42)
Constant	37.34	(4.05)	7.495	(0.69)	-37.56	(0.91)
N	3010		2993		2192	

The sample consists of about PSID respondents present during the 1999 PSID wave who were also children of the original PSID respondents. These respondents were at most 16 in 1968 or subsequently born by 1984. The fixed effects estimates place an additional restriction that there were at least two siblings who were PSID respondents in 1999.

Table 6. Predicting Adult Ln Household Income at age 25 and Change in Ln Household Income between Age 25 and 1999

	Ln Household Income				Within Sibling	
	At Age 25		% Change to 1999		Estimates of %	
Change to 1999						
Age 1999	-0.009	(0.29)	0.105	(3.79)	0.147	(2.23)
Age squared 1999	-0.000	(0.48)	-0.001	(3.14)	-0.002	(2.23)
Black	-0.119	(3.38)	-0.037	(1.10)		
Hispanic	0.058	(0.86)	0.003	(0.05)		
Female	-0.056	(2.19)	-0.026	(1.08)	0.078	(2.00)
Married at Age 25	0.677	(26.2)				
Married-Married			0.498	(14.6)	0.582	(8.05)
Married–Single			-0.139	(2.96)	-0.264	(2.76)
Single–Married			0.612	(16.8)	0.610	(8.03)
Health 16 EX-VG	0.104	(3.35)	0.052	(1.78)	0.245	(4.07)
Ln Parental Income 1-16	0.216	(7.68)	0.195	(7.30)	0.336	(1.58)
Mother Ed 12-15	0.113	(3.49)	0.029	(0.94)		
Mother Ed College	0.082	(1.59)	0.031	(0.65)		
Father Ed 12-15	0.043	(1.35)	0.015	(0.50)		
Father Ed College	-0.043	(0.97)	-0.026	(0.62)		
Number of Siblings	-0.022	(3.02)	-0.011	(1.61)		
Ed 1999	0.068	(9.59)	0.082	(12.1)	0.092	(5.47)
Ln Income at Age 25			-0.713	(49.8)	-0.691	(23.2)
Constant	6.760	(11.4)	2.074	(3.64)	-0.857	(0.33)
N	2058		2045		1358	

The original sample consists of PSID respondents present during the 1999 PSID wave who were also children of the original PSID respondents. These respondents were at most 16 in 1968 or subsequently born by 1984. "t" statistics based on robust standard errors. The fixed effects estimates place an additional restriction that there were at least two siblings who were PSID respondents in 1999. When entered in the ln form, income had to be positive.

Table 7. Predicting Adult Ln Individual Income at age 25 and Change in Ln Individual Income between Age 25 and 1999

	Ln Income					
	At age 25		% Change to 1999		Within Sibling Estimates of % Change	
Age 1999	-0.069	(2.07)	0.126	(3.79)	-0.054	(0.56)
Age squared 1999	0.001	(2.22)	-0.002	(3.40)	0.001	(0.64)
Black	-0.071	(1.77)	0.053	(1.33)		
Hispanic	-0.084	(1.10)	-0.097	(1.23)		
Female	-0.389	(13.4)	-0.358	(12.1)	-0.411	(5.55)
Married at Age 25	-0.011	(0.39)				
Married-Married			0.038	(1.01)	0.045	(0.45)
Married–Single			-0.009	(0.20)	0.050	(0.38)
Single–Married			0.010	(0.16)	0.145	(1.28)
Health 16 EX-VG	0.105	(2.88)	0.110	(3.07)	0.276	(3.03)
Parental Income 1-16	0.203	(6.30)	0.182	(5.66)	0.266	(0.84)
Mother Ed 12-15	0.054	(0.90)	0.008	(0.22)		
Mother Ed College	0.053	(0.90)	0.006	(0.10)		
Father Ed 12-15	0.075	(2.06)	-0.002	(0.06)		
Father Ed College	0.023	(0.44)	0.036	(0.72)		
Ed 1999	0.051	(6.23)	0.085	(10.4)	0.084	(3.50)
Ln Income at Age 25			-0.769	(50.2)	-0.795	(20.5)
Constant	8.303	(12.3)	2.539	(3.72)	5.016	(1.32)
N	1824		1652		981	

The original sample consists of PSID respondents present during the 1999 PSID wave who were also children of the original PSID respondents. These respondents were at most 16 in 1968 or subsequently born by 1984. The fixed effects estimates place an additional restriction that there were at least two siblings who were PSID respondents in 1999. When entered in the ln form, income had to be positive.

Table 8. Interactions of Childhood Health with # of Siblings

	Childhood health		Childhood Health * Num of Sib	
Ln Household Income	0.021	(0.43)	0.057	(3.59)
Ln Household Income Fixed	0.043	(0.46)	0.079	(2.56)
Ln Earnings–Levels	0.079	(1.69)	0.021	(1.35)
Ln Earnings–Fixed	-0.102	(0.77)	0.145	(3.09)
Delta Ln Family Income	-0.002	(0.05)	0.024	(1.64)
Delta Ln Fam.Inc–Fixed	-0.051	(0.46)	0.132	(3.18)
Delta Ln Earnings	0.024	(0.44)	0.039	(2.14)
Delta Ln Earnings–Fixed	-0.115	(0.70)	0.191	(2.85)

Table 9. Estimated Effects of Childhood Health

	All Respondents		Self- Reporters Only	
Education-Levels	0.353	(4.28)	0.307	(3.33)
Education-Fixed	0.114	(1.15)	0.035	(0.59)
Ln Household Income	0.130	(5.03)	0.148	(5.05)
Ln Household Income Fixed	0.240	(4.98)	0.284	(4.87)
Ln Earnings–Levels	0.123	(3.90)	0.109	(3.04)
Ln Earnings–Fixed	0.248	(3.66)	0.279	(3.48)
Delta Ln Family Income	0.052	(1.78)	0.072	(2.13)
Delta Ln Family Income–Fixed	0.245	(4.07)	0.357	(4.80)
Delta Ln Earnings	0.110	(3.07)	0.099	(2.42)
Delta Ln Earnings–Fixed	0.276	(3.03)	0.359	(3.59)

Estimates for all respondents are contained in Tables 12- 15. Estimated for self-reporters are for the reporting respondents in the PSID. Fixed refers to within sibling models.

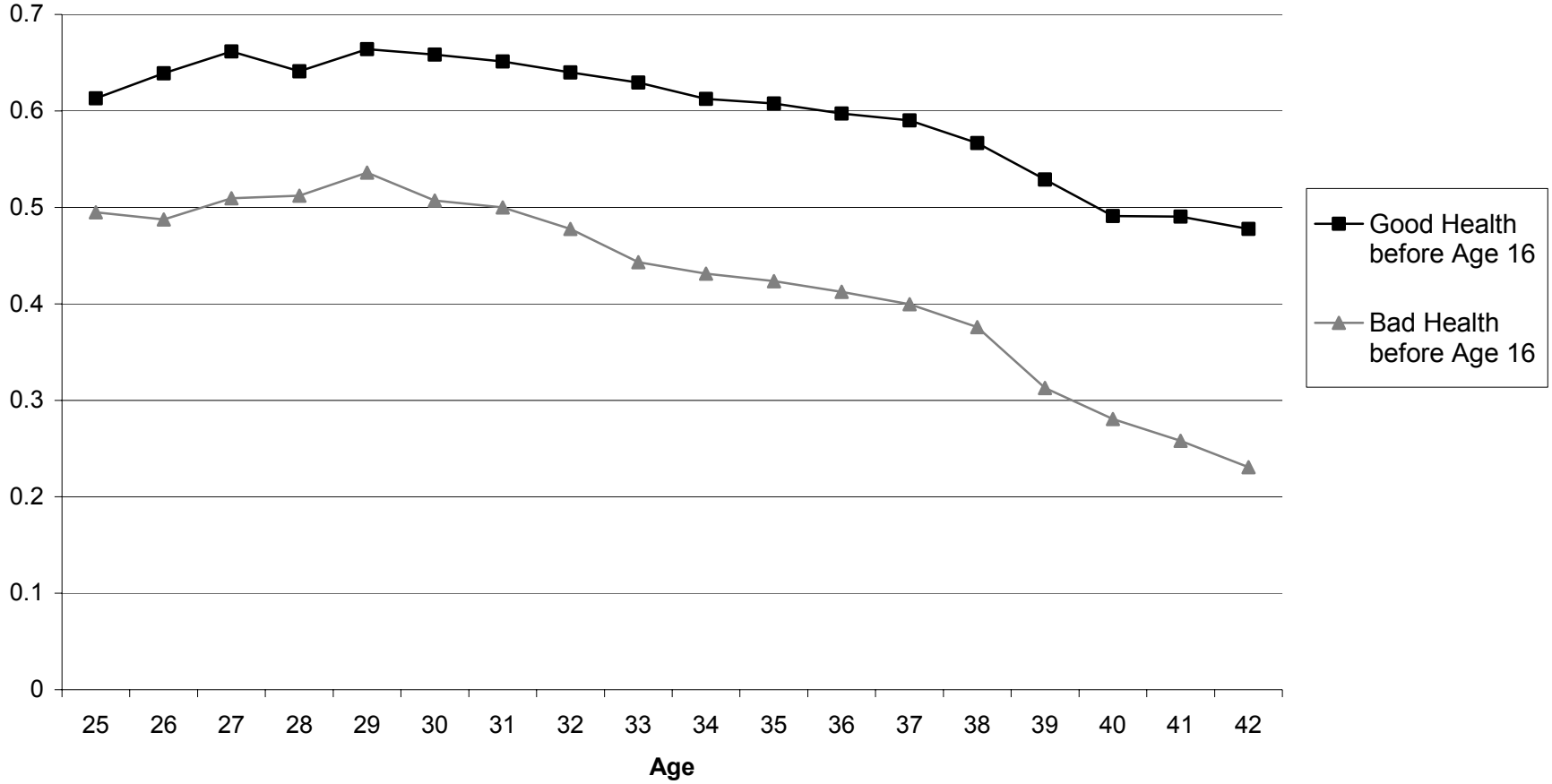

Fig. 1. Self-reported Health Status Excellent or Very Goood by Health Status before Age 16