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Health, Aging and Childhood Socio-Economic Conditions in Mexico

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Abstract

We investigate the long-term effect of childhood socio-economic conditions on the health of the elderly in Mexico. We utilize a panel of individuals aged 50 and above from the Mexican Health and Aging Survey and find that the conditions under which the individual lived at the age of 10 affect health in old age, even accounting for education and income. This paper contributes to the literature of the long-term effects of childhood socio-economic status by being the first, to our knowledge, to consider exclusively the case of the elderly in a developing country.

Keywords

Health; Aging; Mexico

1. Introduction

Recent research from both developed and developing countries documents the importance of childhood socio-economic status on both health and economic outcomes later in life. From their origins in the income gradient literature (Case et al., 2002; Currie and Stabile, 2003), studies of the effects of socio-economic status among the elderly (Case et al., 2005; Buckley et al., 2004) and those linking birth outcomes to health in adulthood (Currie and Hyson, 1999; Almond, 2006; Currie and Moretti, 2007; Oreopoulos et al., 2008; and Maccini and Yang, 2009) suggest that conditions in childhood lead to long lasting health effects, at least in developed countries.

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With increased living standards and higher life expectancy, developing countries face a dual challenge of managing an epidemiological and a demographic transition. This combination means that chronic degenerative diseases become more prominent among these countries' aging populations. The elderly are thus experiencing increasing rates of chronic, in addition to communicable, disease. Yet, despite the growing proportion of elderly persons in developing countries, the majority of analyses of the effects of socio-economic status on health in these countries continues to focus on children or on the economically active populations, and those that do consider the elderly do not investigate the long term effects of childhood conditions (Mete, 2005; Roy and Choudhuri, 2008).

We contribute to the literature on the long-run effects of childhood conditions on health by providing evidence from a developing country. While recent work in development economics by Maccini and Yang (2009) and Akresh et al. (forthcoming) focus on these effects for adults in general, we focus our lens on the elderly. We use panel data from the 2001/2003 Mexican Health and Aging Survey (MHAS) to assess the long run impact of childhood socio-economic conditions on health among individuals aged 50 and above. Faced with this dual demographic and epidemiological transition, Mexico provides a unique setting in which to study the role of childhood conditions on elderly health outcomes.

We begin in section 2 by highlighting the conceptual framework commonly adapted in the literature (Grossman, 1972; Maccini and Yang, 2009). We then turn to the MHAS and discuss the construction of the main variables of interest in section 3. We construct a measure of good health from self-reported status. Because such subjective measures are measured with error (Crossley and Kennedy, 2002; Baker et al., 2004), we provide evidence that this measure is nonetheless correlated with self-reported ailments.

In section 4, we present the results of a reduced form relationship between health status in old age and childhood socio-economic conditions. We find that conditions at age 10 indicating low socio-economic status have long lasting and negative impacts on an individual's health. We find that this effect persists, yet weakens, when we control for an individual's education and wealth. That the effect weakens points to the likelihood that childhood socio-economic conditions influence health via their role in the accumulation of human capital and thus future earnings potential. That the effect persists suggests that childhood circumstances have an effect beyond adult socio-economic conditions by directly influencing health in adulthood.

We check for the robustness of our results to several limitations of our empirical strategy. Because our dependent and independent variables may possibly be measured with error, we check for robustness of our results using alternative measures of health and socio-economic conditions. Our results are robust to these considerations.

Because our variables of childhood circumstances do not measure conditions *in utero* or early infancy (which the literature suggests is most important) we consider the possibility that our results are driven by endogeneity between health in old age and conditions before age 10. The data do not provide us with any plausible exogenous variation or strong instrument for childhood circumstances. However, we make an attempt by appealing to

parental background variables (such as parental education and the father's occupation) to eliminate one possible source of omitted variable bias. We still find persistent effects of childhood conditions, especially among women. In section 5, we conclude and discuss the policy implications of our results.

2. Conceptual Background

We seek to establish the long run impact of childhood socio-economic status (CSES) on health among the elderly. The literature has proposed several mechanisms through which this relationship may operate. First, life-course models (Kuh and Wadsworth, 1993; Ben-Shlomo and Kuh, 2002) emphasize the role that CSES can play on adult health through its effect on adult socio-economic status (SES). These models imply that good adult socio-economic outcomes can partially compensate for poor early childhood socio-economic conditions (Graham, 2002).

Life-course models operate in the following way. An individual's health at any given time is a function of her SES (income, education, etc...) at that time and of her health stock in previous periods. Similarly, socio-economic conditions at any point in time are themselves a function of health in previous periods: poor health in one period might negatively affect an individual's ability to acquire education and may also have a detrimental effect on her earnings capabilities. The determination of health among the elderly thus has a recursive nature since health stock at any given point in time is a function of health stock in the previous period. This recursive structure, both in terms of health stock and SES, implies that initial period health stock and initial SES (be it *in utero*, at birth or in early infancy) will carry through to adulthood and old age.¹

Note that this process can be reconciled with the Grossman (1972) model of gross investment if we consider that an individual's ability to invest in health from one period to the next is a function of her SES. Wealthier individuals are better able to afford health investments. Similarly, more educated individuals invest more in health partly because their education makes them wealthier and partly through the non-pecuniary benefits of an increased education (e.g. knowledge).

Second, the critical period programming (or foetal origins) model (Barker, 1998; Barker et al., 2002) links conditions *in utero*, at birth or in early infancy with lower health trajectories over the life-course. Specifically, there is growing biological evidence that conditions during such a critical period can change an individual's physiology causing a permanent and downward shift in her health trajectory.

According to Barker (1998), nutritional deficiencies *in utero* have been linked to chronic health problems in old age (such as coronary heart disease and stroke). Explanations based on biological research suggest that these effects are driven by "permanent changes" in the "body's structure, physiology and metabolism (p. 13)". Citing work by Reynaldo Martorell, Elo and Preston (1992) suggest that adult height is "essentially determined by events in the

¹For good discussions of the socio-economic determinants of health in developing economies, see Glewwe and Miguel, 2008; Mwabu, 2008; Strauss and Thomas, 2008.

first three years of life (p.193).” Thus, any effects of CSES on height can have long lasting impacts on health (Case et al., 2005; Almond, 2006; Thomas and Strauss, 2008; Maccini and Yang, 2009).

Our conceptual framework follows from Grossman (1972) and especially Maccini and Yang (2009). Specifically, we consider that an individual’s health in old age (H_t) is a function of health states over her lifetime including initial health ($H_0, H_1, \dots, H_{t-1}$), health inputs purchased and consumed over her life-time ($N_1, \dots, N_t$), characteristics of her location over her life-time ($C_0, \dots, C_t$) which includes community infrastructure and disease environment, and her time-invariant demographic characteristics (X). These inputs generate a health production function that can be summarized as follows:

$$H_t = H(H_0, H_1, \dots, H_{t-1}, N_1, \dots, N_t, C_0, \dots, C_t, X)$$

Because this process is recursive in health stock, sequentially replacing H_τ with its expression for each time period from $\tau=0$ to $\tau=t-1$ leads to the following reduced form expression for health at time t :

$$H_t = h(H_0, N_1, \dots, N_t, C_0, \dots, C_t, X) \quad (1)$$

which is precisely the approach in Maccini and Yang (2009), an approach we co-opt here. Specifically, we focus on the effect of CSES on health in old age and so we turn our attention to initial health, H_0 , which relates to “critical-period programming” where conditions *in utero* or in infancy have measurable implications for long-run health. In the same vein, we take CSES to be a determinant of initial health. Initial health is also influenced by unobserved genetic factors (G) and initial community-level characteristics:

$$H_0 = g(CSES, G, C_0) \quad (2)$$

Both the life-course and critical period programming models imply that health in ‘old age’ is influenced by CSES conditions. The difference between the two classes of models is that the critical period programming model allows for CSES to have an effect on health independently of their effect via adult SES.

The questions we explore are first whether CSES affects health in old age and second whether this effect persists when controlling for adult SES. We expect that life-course mechanisms are present, and so our empirical strategy will consider the influence of adult SES. However, we seek to uncover evidence of the critical period programming hypothesis. The exercise we conduct summarizes as follows. If critical period programming is not an issue, then CSES should affect adult health only via its effect on adult SES. This would imply that we could consider CSES an instrumental variable (IV) for adult SES which are thought to be endogenous (Thomas and Strauss, 2008). However, if we find that the adult health effects of CSES persist even when controlling for adult SES, then we have two candidate explanations. The first explanation would be that there is evidence that critical period programming is an important channel and we would not reject the critical period programming hypothesis that early life circumstances have permanent effects on an

individual's health. In other words, both life-course and critical period programming mechanisms operate and CSES cannot be used as IV for adult SES. The second explanation is that we are not doing a good job at capturing CSES or adult SES. If instead CSES are picking up a component of adult SES which are measured with error, then it would be misleading to conclude that both life-course and critical period programming mechanisms operate. Our research strategy will be to conduct a series of robustness checks to try to rule out this second explanation.

3. Data

Mexican Health and Aging Study (MHAS)

The MHAS is a two-year panel study modeled after the U.S. Health and Retirement Study.² At its baseline in 2001, it was nationally representative of Mexicans born prior to 1951 (i.e. age 50 and older in 2000). The survey includes information on health measures (self-reports of conditions and functional status), background (education and childhood living conditions), family demographics, and economic measures (wage and non-wage income and assets). In our analysis we keep non-proxy interview observations for individuals who responded both in 2001 and in 2003. In addition, we keep individuals with complete information on the key variables. Namely we drop respondents with missing information on childhood health, CSES, or education. These restrictions yield a sample of 3818 men and 4392 women born before 1951. Descriptive statistics are presented in Table 1, and below we describe the construction of the key dependent and independent variables.

Dependent Variables – Health Outcomes

To construct a measure of good health, we utilize MHAS data on self-reported quality of health, based on the question “Would you say your health is excellent, very good, good, fair, or poor?” From these responses, we define a binary indicator of “Good Health” that equals 1 if the response is ‘excellent’, ‘very good’, or ‘good’, and 0 otherwise. While this definition is arbitrary, later in this section, we compare this to a more stringent definition of ‘good health’ as ‘excellent’ or ‘very good’. We find a stronger correlation between the former definition (‘excellent’, ‘very good’, or ‘good’) and several self-reported chronic conditions, lending support for the former.

We observe in Table 1 that 38% of men and 30% of women report being in good health in 2003. This contrasts with higher rates of 44% and 32% for men and women respectively in 2001. We attribute this difference to the fact that respondents have aged by two years between rounds and consequently report somewhat worse health.

This measure of health status is often used in predicting health outcomes such as health care utilization and mortality (Buckley et al., 2004; Roy and Chaudhuri, 2008) and is used widely in this literature (Case et al, 2005; Maccini and Yang, 2009). Yet, self-reported health measures are prone to measurement error (Crossley and Kennedy, 2002; Baker et al., 2004). Because of the subjectivity of our measure, we utilize supplementary information contained in the MHAS by comparing this measure of health to more objective, albeit self-reported,

²Data and information from MHAS can be obtained from their website, <http://www.mhas.pop.upenn.edu/>.

indicators. While we cannot entirely eliminate the problem of measurement error, we try to reduce the likelihood that our results are driven by the subjectivity of the health measure. Though our health measure is imprecisely measured, since it is the dependent variable, the error is absorbed in the regression error and is thus not of great concern.

The MHAS asked respondents whether they suffered from particular ailments (hypertension, diabetes, cancer, heart disease, respiratory illness, stroke and arthritis) in the last two years or ever (see Table 2 for prevalence). Table 2 provides the correlations between our subjective measure of good health and having experienced each of the individual ailments. In all cases, the direction of the correlation confirms the validity of the subjective measure: having suffered from hypertension, diabetes, cancer, respiratory problems, heart problems, strokes or arthritis is negatively correlated with being in good health. These correlations are always (save for cancer in 2001) very strongly significantly different from zero. We also construct an aggregate ailment measure as those who report having ever or in the last two years suffered from any one of these seven ailments. We find that 45 to 47% of men and 61 to 64% of women report having experienced at least one of the seven ailments.

However, the magnitudes of these correlations only range from -0.02 to -0.41 for each individual ailment, and the aggregate (in the last row) has a correlation over -0.43 . This could be due to the fact that respondents in ill-health are suffering from ailments that are not listed in the above. This could also be a result of measurement error: given the subjectivity of the good health measure, it is possible that respondents who suffer from an ailment actually report being in 'good' health. This is in fact the case, for example, for 25% of respondents who report hypertension in 2001 and, surprisingly, 29% of respondents who report in 2001 having been diagnosed with cancer. Thus, we check for the robustness of our results by considering 'good' health responses to the quality of health variable as being in 'bad' health as an alternative measure: our constructed measure may be biased because of the presence of false negatives (the ill who report 'good' health). The correlations of this alternative constructed measure of good health are also reported in Table 2. We immediately observe that the magnitudes and significance of the correlations fall drastically (except in the case of cancer in 2003, where cell size is very small). While this alternative measure might pick up those individuals who reported being in 'good' health despite suffering from an ailment, it would seem that the possibility of a false positive (the healthy who report being in 'good' health but considered to be in 'bad' health in the alternative measure) is more problematic.

The subjectivity of health status suggests that these measures may be picking up perceptions of health rather than health itself. We address this concern in three ways. First, the last three columns of Table 2 consider only those individuals who report good health status in 2001, and we compare their ailment outcomes across their reported health status in 2003. We see that conditioning on self-reporting good health in 2001 those reporting bad health two years later have significantly worse ailment outcomes.

Second, we exploit the MHAS's module on "Functionality and Help" and consider respondents' reported difficulties with Activities of Daily Living (ADLs). We constructed a binary variable taking the value 1 if respondents reported difficulty with any of 10 activities

(walking, sitting, getting up, climbing stairs, stooping, reaching arm over shoulder, pulling, lifting, picking and dressing). Table 1 shows that between 43% and 45% of men report difficulties with at least one ADL, while between 59% and 62% of women report difficulties. This variable is inversely correlated with our subjective measure. Indeed, the correlation between this ADL measure and our preferred good health measure is -0.3034 and -0.2894 for 2001 and 2003 (significant at 1%).

Finally, according to the literature reviewed in section 2, one of the most likely health indicators of poor socio-economic outcomes in early life is height. The empirical advantage of height is that it is more objective than the self-reported health status, ailments or difficulties with ADLs. The MHAS reports measured height of all individuals. Correlations between health and the subjective health measure are in the magnitude of 0.11 for both years (significant at 1%).

Childhood SES

We construct measures of CSES from questions pertaining to the household when the individual was a child. For instance, the survey asked respondents about clothing, crowding, hunger and access to a toilet when they were 10 years old. There are many ways in which these conditions can affect health. For instance, not wearing shoes regularly may lead to parasitic infections, which has implications for nutrition. Crowding has implications for the spread of communicable disease and sleeping in the same room used for cooking can cause respiratory disease due to indoor air pollution. Going to bed hungry on a regular basis can indicate poor nutrition. Either way, these conditions may be picking up both socio-economic conditions (income) and related environmental (indoor air pollution) conditions which can directly affect health outcomes, though it is impossible to distinguish between these two. That said, these conditions, taken together, give a sense of the conditions in which these individuals lived at a young age. One concern for our analysis is that these four conditions are highly correlated and so to avoid problems of multi-collinearity, we combine them into a CSES index, where each component is equally weighted.³ Specifically, our index is increasing with poor outcomes. A CSES index value equal to zero indicates that the individual had a toilet in the residence, did not generally go to bed hungry, wore shoes regularly and no household member slept in the same room used for cooking. Thus, the CSES index should negatively predict health.

The literature cited in section 2 considers initial conditions to be either *in utero* or during early infancy. Our measures of CSES pertain to conditions at age 10. Though we are unable to make a one-to-one mapping between conditions at birth or in early infancy and CSES, we believe that the two are highly correlated because of the persistence of poverty that existed in Mexico in the 1950's. Ninety-nine percent of individuals in our sample are born before between 1914 and 1950, a period in which Mexico was a very poor country, and where the large majority of the population resided in rural areas (see López-Alonso (2007), López-

³We also considered principal components to reduce the dimensionality of the CSES information. The factor loadings on the first component are almost equal, so it is not clear that a principal components analysis would outperform the equally weighted index we use here. However, we ran the entire analysis in the paper with principal components instead of the equally-weighted index, and our results are almost identical (results available from the authors upon request).

Alonso and Porras Condey (2003) and Astorga et al (2005) for historical accounts of living standards in Mexico during this period). López-Alonso and Porras Condey (2003) and López-Alonso (2007) find that the health status of the Mexican population (measured by height) did not respond much to economic fluctuations from 1914 to 1940. However, their historical analysis of the period associated with the ‘Mexican miracle’ (1940s and 1950s) does suggest that stature did improve for these cohorts.

Using data on Mexican macro-economic crises during the first half of the Twentieth Century, CSES conditions at age 10 generally correlate well with time-series on per capita consumption. The correlation coefficients (ρ) between Mexican per capita consumption at birth year (Barro and Ursua, 2008) with the MHAS CSES variables at age 10 are statistically significant and always in directions consistent with intuition: the correlation is positive for residing in a home with a toilet ($\rho=0.6663$) and regularly wearing shoes ($\rho=0.4143$), and negative for regularly going to bed hungry ($\rho=-0.4085$) and living in a home where someone slept in the room used for cooking ($\rho=-0.2732$).

Descriptive statistics for CSES variables are found in Table 1. Only 27% of men and 30% of women reported living in a residence with a toilet inside the home at age 10, 34% of men and 29% of women reported going to bed hungry, 78% of men and women wore shoes regularly and 22% and 20% of women reported that at least one household member would sleep in the same room that was used for cooking. The general pattern revealed by these childhood socio-economic variables is that a large proportion of respondents likely come from a relatively poor background. Because recall and measurement error are possible, we include dummies in the analysis based on the degree to which respondents needed help to answer the questions in this section (as noted by the interviewers).

Other Independent Variables

Our other primary independent variables measure adult SES; the two most important dimensions are income and education. Because of the predominance of self-employment earnings in a country like Mexico, income may be poorly reported and measured. There are also retired individuals and pensioners who do not earn labour income. Furthermore, health outcomes may be more responsive to permanent income than to current (or transitory) income (see Van Ourti (2003) for a discussion). Because of these concerns, and since the effect of income on health is expected to be of a more cumulative nature, we utilize the log of 2001 “per capita assets” as a measure of wealth as it also reflects accumulated income. The MHAS reports household assets, which are constructed from the self-reported values and debt associated with real estate, businesses, capital assets, vehicles, and other. Missing values for each component were imputed by the MHAS using a multiple imputation multivariate regression technique.⁴ Per capita assets are derived by dividing household assets by this variable. Log per capita household assets is 11.66 on average, corresponding to mean per capita household assets valued at 255,981 Mexican Pesos (or about 24,000 US\$ in 2003).

⁴For detailed description of the variable and imputation methodology: <http://www.mhas.pop.upenn.edu/english/documents/Imputation/Imputation-2001-v21.pdf>.

We measure education using years of completed schooling. In Table 1, we immediately note the low educational attainment of our sample: the average man only has 5.18 years of schooling and the average woman only 4.37 years. These trends reflect that these individuals were of school age when Mexico was still a relatively poor country and where access to schools was not as pervasive as it is today.

The descriptive statistics also include a number of demographic characteristics. The average respondent in our sample is about 61 years of age, is most likely (around 60%) living in a large urban area (location has more than 100,000 inhabitants). The major difference across gender in the descriptive statistics is whether the individual is currently married or in a consensual union. Men are far more likely to have a spouse present than women. Only 18% of men and 14% of women reside in a location with less than 2,500 inhabitants, which would be largely rural. This is significantly less than the 25% of Mexicans who live in rural areas according to the UN.⁵ Ideally, our analysis would also control for current and childhood geographical location. Unfortunately, the MHAS public use files do not include them. While geographic location may be important determinants of health, our empirical approach minimizes the degree to which this lack of information leads to bias. That said, we observe whether the individual currently resides in a large city or in a rural environment, so we can control for the degree of urbanization.

4. Results

4.1. Main Results

We begin with a reduced form relationship, similar to Maccini and Yang (2009). Specifically, we consider an individual's health during their old age (H_i) to be a function of her childhood circumstances ($CSES_{i0}$) and demographic controls such as age, marital status and dummies for current location size (X_i). The 2001 and 2003 samples are pooled and we include a year fixed effect:⁶

$$H_i = \beta \cdot CSES_{i0} + X_i' \gamma + t + \varepsilon_i \quad (3)$$

Equation (3) is a reduced form relationship that assumes childhood circumstances to be a sufficient statistic for health in adulthood. Given the life-course model discussed above, and the important income gradient documented in Case et al. (2002) and Currie and Stabile (2003), as well as the findings in Case et al. (2005) that childhood outcomes affect adult SES which in turn affect adult health, this assumption is likely quite strong. In other words, the effect of CSES, captured by β , might be driven upwards by omitted variables bias. To assess whether the effect of CSES on elderly health is driven by its effects on adult

⁵Population Division of the Department of Economic and Social Affairs of the United Nations Secretariat. 2004. World Urbanization Prospects: The 2003 Revision. Urban and Rural Areas Dataset (POP/DB/WUP/Rev.2003/Table A.7). This discrepancy is likely due to the MHAS's over-sampling of high out-migration states. Weighting the means using the appropriate expansion factors yields a result much closer to the 25% reported by the UN. Our empirical analysis will use expansion-factor weights.

⁶We analyze a pooled regression at the suggestion of a referee. However, running the regression for the two repeated cross-sections generates very similar conclusions about the long-term effect of CSES on elderly health. Results available from the authors upon request.

outcomes such as education and income, we sequentially estimate equation (3) augmented with education (S_i) and our proxy for household income (Y_i), household assets, as follows:

$$H_i = \beta \cdot CSES_{i0} + X_i' \gamma + \delta S_i + t + \varepsilon_i \quad (4)$$

$$H_i = \beta \cdot CSES_{i0} + X_i' \gamma + \delta S_i + \lambda Y_i + t + \varepsilon_i \quad (5)$$

Equations (3), (4) and (5) are estimated for men and women separately. The probit marginal effects are presented in Panel A of Table 3. Results on the CSES index indicate that poor CSES at age 10 reduces the likelihood of reporting good health. Controlling for education in equation (4) reduces the strength of CSES in predicting health outcomes: the magnitude and significance of the CSES variables fall although the index remains statistically significant at 10%.

Adding our proxy for income in equation (5) does little to change the results from equation (4). In fact, per capita assets are not statistically significant in determining health outcomes, despite the well-documented income-gradient (Case et al., 2002; Currie and Stabile, 2003). One concern could be measurement error – per capita assets are mis-measured and a poor proxy for permanent income (which we discuss below). Another concern is that education and assets are so highly correlated that adding assets in equation (5) does little once we account for education. Indeed, the two variables are strongly correlated (0.3056 and 0.2892 for men and women respectively), and removing education from estimating equation (5) leads to more statistical significance (positively) of the effect of log per capita assets on health outcomes.

The results in Panel A suggest that a large part of CSES's effect on health can be explained by one adult SES factor: education. The coefficient on the CSES index drops by almost half when we add education. This result points to evidence of the life-cycle hypothesis. Nevertheless, the fact that the CSES index still has explanatory power once we control for education and assets suggests that we cannot reject the critical period programming model either. Since this is only circumstantial evidence at this point, we must explore numerous reasons to expect that these reduced form results are biased.

First, if socio-economic conditions in childhood are so bad that they lead to premature death, then these individuals would not appear in the 2001 or 2003 surveys. In other words, individuals surveyed by the MHAS are 'self-selected' in that they have survived. If this survivor bias is important, then whatever estimates we find here are in fact lower bounds of the true effects (see Strauss et al. (1993) and Maccini and Yang (2009) for a discussion of this effect). Since these would be lower bounds, the fact that we find some persistence of CSES suggests that the effects are important.

Second, due to the self-reported nature of the dependent variable, our results might be driven by our inability to properly measure an individual's health. Given the possibility that persons in poor health stoically report 'good health' status, section 4.2 checks for the

robustness of our results to using the more stringent definition of ‘good health’ status described in section 3.

Third, because we observe CSES at age 10, and not conditions *in utero* or in infancy, we explore the link between CSES and economic conditions at birth. Given the historical background relevant to the individuals in our sample, we believe that conditions at age 10 are a good proxy for conditions at birth. Even were this not the case, the fact that they have long lasting impacts on the health of the elderly, even controlling for adult SES, suggests that even childhood conditions beyond the ‘critical-period’ matter in the long-run.

Fourth, it is possible that our measures of education and wealth do not adequately capture adult SES. For instance, they may be measured with error or education may have important non-linear effects on elderly health. We explore this issue in section 4.3.

Finally, while we believe that CSES at age 10 is strongly correlated to initial conditions for the reasons discussed above, it is possible that this creates an endogeneity bias. Though we have no plausible source of exogenous variation in the determination of initial health outcomes, we appeal in section 4.4 to parental background.

4.2. Measurement Error in the Dependent Variable

Recall from section 3 that respondents were asked to rate their health on a scale from poor to excellent. The results in Panel A of Table 3 considered ‘good’, ‘very good’ and ‘excellent’ responses as “Good Health”. Yet, because of the possible false positives, a stricter definition of “Good Health” which only considers ‘very good’ and ‘excellent’ responses might lead to different results. Panel B of Table 3 presents the results from estimating equations (3), (4) and (5) using this alternative, more stringent, definition of good health. The results from Panel B are generally robust to this concern. The CSES index still reduces the probability that the individual reports “Good Health”. However, the magnitudes of these effects are lower, and in the case of men become insignificant when we control for adult SES. The differences we find across gender might partly be explained by differential mortality (Strauss et al., 1993). Specifically, the longer lasting effects of CSES among women could be driven by the fact that, since men tend to die at younger ages, we observe only the ‘high-health-type’ men which are then compared to both ‘high-health’ and ‘low-health’ type women.

In Panel C of Table 3, we consider difficulties with ADL as indicator of health. Here we find that poor CSES in childhood is associated with a higher probability that individuals have some difficulty with ADLs, and that this persists when controlling for education and assets. There are nonetheless two important differences in using this measure of health compared to the subjective self-reported measures in Panels A and B. First, the results are insignificant for women, but remain statistically significant for men at the 5% level. Second, adult SES (education and income) do not have much predictive power in these regressions.

Finally, in Panel D of Table 3, we consider (log) height as the dependent variable. The motivations for using height to proxy for health are discussed in sections 2 and 3. First, height is a more objective measure as it is precisely measurable. Second, since research has

demonstrated that height is determined by age 3, any long lasting impacts of CSES should be ‘visible’ here. Results show that the effects of CSES on (log) height are strongly statistically significant. Low CSES predicts lower heights, an effect which falls in magnitude but remains highly significant when controlling for adult SES. Education and assets are now very strongly significant, though if height is predetermined at a young age, then these coefficients are likely picking up a reverse causal effect. This is plausible given the large body of evidence that height is positively associated with earnings (see Strauss and Thomas (2008) and Mwabu (2008)). Since the statistical association between the dependent variable and our measures of education and assets is so strong, we suspect that these measures are indeed doing a fairly good job at capturing adult SES, a concern we turn to next.

4.3. Measurement error in adult socio-economic conditions

There remains the possibility that log per capita assets and years of schooling do a poor job at capturing adult SES. First, while many elderly do not generate income because they have left the work force, it is worthwhile either considering income as an alternative measure of wealth or as an IV for wealth. To the extent that log per capita assets are a mis-measured proxy of wealth, an IV approach may provide less biased results. Our results are robust to including income or using income as an IV for assets.

Second, years of schooling may be an inadequate measure of education. We check our results using the highest level of education attained and include years of education squared. Both of these techniques allow for the non-linearity of the effect of education which is ignored by simply using years of schooling. Our results are robust to these considerations.

Finally, it is possible that income and education alone do not fully account for adult socio-economic conditions. We utilize a module in the MHAS to capture an individual’s relative social position. Specifically, we consider the respondents’ position on the social ladder in their neighbourhood and country.⁷ In addition to being a proxy for socioeconomic status, there is scientific evidence that relative rank can impact health by leading to both physical and physiological stressors (Sapolsky, 2004; Marmot et al. 1997; and Cohen, 1999). Including either of these measures in the regression does little to change the fact that CSES has persistent effects on elderly health.^{8,9}

4.4. Endogeneity of CSES

The results in Table 3 are based on the assumption that the reduced form relationship between health during old age and CSES is free from bias. Many recent papers use such a reduced form, most of which relate to the critical period programming hypothesis (Maccini

⁷The question reads: “Here is a ladder. There are 10 steps in total from bottom to top. Look at this ladder. At the very top you’ll find people who have the most weight on decisions, or on how things are going to be done. At the very bottom are people with the least weight in decisions, or on how things are going to be done. If you consider your present situation and compare it with that of all other members of [your neighbourhood/Mexico], where would you place yourself?”

⁸All of our results for this section are available from the authors upon request.

⁹We believe that our adult SES measures are doing a good job at capturing health investment decisions throughout the life-cycle. The MHAS provides one more variable that would allow us to capture such decisions: whether the individual has ever smoked. We re-ran our main tables including whether the individual is currently smoking or has once smoked but has since quit. Our results (available from the authors upon request) are robust to including the smoking variables.

and Yang, 2009; Oreopoulos et al., 2007; Currie and Moretti, 2007; Almond et al., 2005). These papers have credible sources of exogenous variation at birth that support such a specification – they use either exogenous variation in weather to explain birth or early infancy outcomes, or exploit twin and sibling differences in birth weight. Almond et al. (2005) warn against interpreting birth outcomes on later health outcomes as causal without controlling for potential common third factors such as genetics or parental SES.

This line of research calls into question the results found in section 4.1 since we are not able to find plausible exogenous variations as the literature cited above. While in principle it would be feasible to link MHAS individuals to weather variations at birth as in Maccini and Yang (2009), the public release files do not include current geographic location, let alone geographic location at birth. Furthermore, the MHAS is not a twin or siblings study, so we are unable to control for third factors such as genetics in this way.

However, the structure of the MHAS allows us to investigate this causal issue using the SES of the respondents' parents. Respondents were asked about their father's (or guardian's) occupation at age 10 (e.g. agriculture, construction, service, ...), both parents' education, and whether the parental residence at age 10 was primarily urban. Including parental SES should reduce an omitted variables bias.

Table 4 presents the results of estimating equation (5) augmented with the parental SES variables, using all four dependent variables for health.¹⁰ Doing so does not change our conclusions for women that low CSES is associated with lower health outcomes: the coefficients in Table 4 are within a standard deviation of those in Table 3. The parental SES variables are jointly insignificant for three of the four dependent variables. The picture is different for men. Including parental SES reduces the coefficients on the CSES index by one half compared to Table 3 with the exception of the ADL regressions. The parental SES variables are jointly significant for the regressions on good health status and height and they are more strongly significant than in the women's sample. Men's CSES seem partly determined by parental SES and thus including these variables in the regressions reduces omitted variable bias. Men's parental SES and CSES may thus be highly related and both have critical period programming and life-course implications

Excluding parental SES (Table 3) provided support for the critical period programming hypothesis for both men and women, and coefficients did not vary much by gender. However, including them produces different results (Table 4): while the critical period programming hypothesis still holds for women, the effects of CSES seem to operate mostly through the life-course mechanism for men. Thus, once we control for omitted variables, the critical period programming effect for men found in Table 3 is reduced. This is in accordance with the oft-documented gender bias that results in persistent long run effects for women more than for men (e.g. Maccini and Yang, 2009).

¹⁰Alternatively, we might include the first principal components of the parental background and CSES variables. We found that we would need the first 9 components if we wanted to capture over 85% of the variance in these measures. Doing so did not change the conclusions in our analysis (results available from the authors upon request).

Alternatively, parental SES might be candidate instrumental variables (IV) for CSES, especially for women since they rarely predict health outcomes. We thus investigate an IV strategy and instrument the CSES index with parental SES. Since we are dealing with binary endogenous variables, we use a 2SLS procedure (see Angrist, 1999).

The 2SLS-IV results (Table 5) differ from the non-instrumented results (Tables 3 and 4). While the CSES index still significantly predicts health when using our preferred measure of self-reported health status or height, there are now differences across gender. When controlling for adult SES, the CSES index remains statistically significant (and within a standard deviation in magnitude) for men, but becomes insignificant for women. Controlling for possible endogeneity in CSES, the main results hold for men but the effects of CSES now seem to mostly operate through the life-course mechanism for women. That is, the critical period programming effect for women found in Tables 3 and 4 disappears.

The validity of the IV results rests upon the validity of the IVs. The first stage joint F-tests of the instruments are statistically significant, suggesting that the parental SES variables do predict childhood conditions at age 10, and the over-identification tests are rarely rejected. While the joint F-tests of parental SES variables on health were insignificant for women (Table 4), the first-stage F-tests in Table 5 are generally low, pointing to possible weak instruments. It is thus difficult to offer a definite judgement on the validity of the instruments for women. The story is different for men. While the first stage statistics in Table 5 suggest that the instruments are more strongly related to the CSES index than for women when controlling for adult SES, the overidentification test is rejected in the height regressions. Given the joint significance of the parental SES variables in Table 4 for the main health measure and for height, it seems that these variables fail on statistical criteria. Thus, it is not clear to what extent the IV procedure is statistically valid for men in the case of our preferred health measure or for height, even though the overidentification test is not rejected for the former. For the alternative health measure and ADLs, the instruments satisfy the statistical criteria (though remain weak when controlling for adult SES) and the IV results for the alternative health measure are qualitatively similar to the non-instrumented results. Meanwhile, the IV results on ADL are both qualitatively and quantitatively similar to the non-instrumented results.

Statistically, the IV results do not offer conclusive evidence in favour or against the relative importance of the critical period programming versus life-course mechanisms. However, the differences across gender do suggest some differences in the channels through which current health is affected by CSES and parental SES. Specifically, parental SES predict health above and beyond CSES for men but not for women and they are weaker instruments for CSES for women than for men. Several explanations may be offered. First, men and women may have different recollections about parental SES, and so measurement error may vary across gender in ways that confound the IV analysis. Second, the relationship between parental SES and CSES might somehow be different for girls and boys. Third, the relationship between health and parental SES might also vary by gender. If gender bias was an issue when the individuals in our sample were born, son preferring rules might explain some of these differences. Finally, there could be other unobservable (e.g. genetic) differences across men and women that come to play in the relationship between parental SES, CSES and adult

health. Either way, the MHAS does not provide sufficient information to explore these differences more fully.

Regardless of the statistical criteria, these instruments may also fail on economic grounds: it is impossible to reject the theoretical possibility that parental background has a direct effect on health in old age. For instance, parental SES, CSES and health could also be jointly determined by a ‘third factor’ such as genetics (G in equation (2)). The error term of equation (5) would then contain G and the estimated coefficients in our tables would suffer from omitted variable bias to the extent that parental SES are related to G . Unfortunately, although we have tried to minimize this bias, the available data do not allow us to fully reject the possibility that a factor such as genetics may explain the significant effect of childhood circumstances on adult health.¹¹

5. Discussion

We find long-lasting effects of childhood SES on health among the elderly in Mexico. The effect is particularly persistent for women. While these results are consistent with growing evidence from developed and developing countries, our results contribute to this body of knowledge by finding evidence among a previously under-researched yet growing demographic: the elderly in a developing country.

We check for robustness of our results to several limitations of our empirical strategy. First, we take into account measurement error in the dependent variable by alternative proxies for good health. Second, we consider the possibility that our measures of adult socio-economic conditions are measured with error. Third, while the data do not allow us to formally control for endogeneity in childhood circumstances, we consider a two stage least squares approach to deal with endogeneity using parental background information. In all cases, we find persistent effects of childhood circumstances on health of the elderly. While it is still possible that our results are due to measurement error in adult socio-economic conditions (which would favor the life-course model only) or that they are due to an unobserved factor such as genetics (which would then invalidate both life course and critical programming hypotheses), it would appear that the critical programming hypothesis should not be dismissed as a factor explaining these effects.

If so, our results have several implications for policy. First, the fact that childhood circumstances have a long-lasting effect on health of the elderly, which persists when controlling for education and wealth, suggests that cash transfers to the elderly may not completely counter the negative effects from poverty a life-time ago. Second, because conditions during childhood are so long lived and independent of adult SES such as education and wealth (lending support to the critical period programming model), the long-term benefits such as improved health in old age should be factored into the costbenefit analysis of child welfare policy. For instance, our results imply that conditional cash transfers such as Mexico’s *Oportunidades* can have effects on recipients’ health in old age

¹¹It should be noted that other existing studies (Maccini and Yang, 2009; Oreopoulos et al., 2007; Currie and Moretti, 2007; Almond et al., 2005) cannot fully dismiss that possibility either.

even beyond the intended effects on schooling and child health outcomes. Third, as developing countries face their dual demographic and epidemiological transitions, and as childhood poverty rates fall, policy makers should be forward-looking in allocating resources to a growing, aging cohort, whose morbidity tends to be associated with relatively more expensive chronic diseases.

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Table 1

Descriptive Statistics of Main Variables

Variable	Men (N=3818)	Women (N=4392)
Good Health in 2003	0.380	0.295
Good Health in 2001	0.443	0.320
Alternative health measure (2003)	0.059	0.039
Alternative health measure (2001)	0.086	0.049
Difficulty with at least one Activity of Daily Living (2003)	0.434	0.587
Difficulty with at least one Activity of Daily Living (2001)	0.451	0.620
Height in 2003 (cm)	166.348	155.718
	8.729	8.280
Height in 2001 (cm)	166.634	156.126
	8.540	7.788
Before age 10, residence had toilet inside house	0.269	0.302
Before age 10, generally go to bed hungry	0.344	0.291
Before age 10, wore shoes regularly	0.779	0.778
Before age 10, someone slept in same room used for cooking	0.220	0.198
CSES index (0=best CSES, 1=worst CSES)	0.379	0.352
	0.291	0.292
Years of Education	5.180	4.370
	4.787	4.022
Age	61.537	60.588
	8.780	8.311
Respondent married or in consensual union in 2001	0.854	0.617
Respondent married or in consensual union in 2003	0.839	0.586
Log per capita assets	11.587	11.724
	1.462	1.460
Location 100,000+	0.574	0.619
Location 15,000 – 99,999	0.153	0.146
Location 2,500 – 14,999	0.092	0.090
Location - 2,500	0.182	0.145
Respondent required help with the demographics questions	0.052	0.049
<i>Parental Background Variables</i>	<i>(N=3254)</i>	<i>(N=3710)</i>
Mother had completed primary school	0.129	0.148
Father had completed primary school	0.161	0.177
Parental residence was primarily urban	0.400	0.418
Father's main occupation was agriculture	0.584	0.563
Father's main occupation was construction	0.141	0.139
Father's main occupation was in the service sector	0.097	0.096
Father had his own business	0.070	0.083
Father worked in an office	0.032	0.034
Father had some other occupation	0.055	0.058

Table 2

Incidence of ailments

Ailment	Number of Non-Missing Observations		% with Ailment		Tetrachoric Correlation with Subjective Health Measure		Tetrachoric Correlation with Alternative Subjective Measure		% with ailment conditional on good health in 2001		
	2001	2003	2001	2003	2001	2003	2001	2003	Good health in 2003	Bad health in 2003	t-test
Men											
Hypertension	3679	3806	29.63	28.27	-0.2819*	-0.3086*	-0.1960*	-0.2033*	16.02	30.32	7.1044***
Diabetes	3677	3805	13.68	16.98	-0.4004*	-0.3123*	-0.2632*	-0.2650*	6.17	13.93	5.4245***
Cancer	3682	3809	0.92	0.68	-0.1096*	-0.3691*	0.0106	-1.0000*	0.00	0.10	3.0301***
Respiratory Problems	3684	3812	5.97	4.38	-0.3233*	-0.3140*	-0.1272*	-0.3691*	1.46	3.40	2.6342***
Heart Problems	3683	3812	3.96	2.22	-0.3219*	-0.3144*	-0.2748*	-1.0000*	0.94	1.76	1.4850
Stroke	3682	3815	2.30	0.89	-0.2907*	-0.3000*	-0.1188*	-1.0000*	0.31	0.67	1.0864
Arthritis	3685	3811	14.97	13.46	-0.3672*	-0.3791*	-0.3284*	-0.3295*	4.60	11.29	5.2127***
Any of the above	3818	3818	47.01	45.68	-0.4425*	-0.4318*	-0.3098*	-0.3242*	24.37	45.45	9.3349***
Women											
Hypertension	4311	4383	45.70	42.76	-0.3284*	-0.3806*	-0.3487*	-0.3343*	20.89	41.99	8.7756***
Diabetes	4309	4378	17.34	18.27	-0.4137*	-0.3664*	-0.2314*	-0.2800*	4.87	13.02	5.4835***
Cancer	4318	4385	2.61	0.82	-0.0206	-0.2898*	-0.0156	-1.0000*	0.26	0.93	1.6564*
Respiratory Problems	4317	4381	6.46	4.31	-0.2764*	-0.1906*	-0.3362*	-0.2269*	1.58	3.26	2.0753***
Heart Problems	4315	4392	2.64	2.21	-0.3293*	-0.3054*	-0.1122*	-0.2122*	0.13	1.70	3.2035***
Stroke	4315	4392	1.97	0.91	-0.2358*	-0.1788*	-0.1255*	-0.0724	0.39	0.93	1.2532
Arthritis	4311	4383	24.89	22.92	-0.3207*	-0.3605*	-0.1940*	-0.2603*	9.34	22.29	6.8291***
Any of the above	4392	4392	64.16	61.32	-0.4652*	-0.4837*	-0.3892*	-0.3745*	31.66	58.82	10.6167***

Notes:

* significant at 5%.

The "alternative" health measure considers a person to be of good health only if they respond being in "Excellent" or "Very Good" health in the quality of health question. "Good" answers to this question will instead be considered as being in "Bad health" (see section 3.4. for a discussion).

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Table 3

Effects of CSES on Adult Health

Men (N=7654)			Women (N=8789)			
Equation:	(3)	(4)	(5)	(3)	(4)	(5)
Panel A - Dependent Variable: Good Health Status. (probit marginal effects)						
CSES Index	-0.162 (0.040) ***	-0.080 (0.042) *	-0.079 (0.042) *	-0.163 (0.038) ***	-0.078 (0.042) *	-0.073 (0.037) *
Respondent's years of education		0.018 (0.003) ***	0.018 (0.003) ***		0.016 (0.003) ***	0.015 (0.004) ***
Log per capita assets			0.003 (0.011)			0.010 (0.009)
Regression Wald Chi-Squared	126.39 ***	175.26 ***	175.98 ***	72.55 ***	94.71 ***	95.72 ***
Pseudo R-Squared	0.0425	0.0575	0.0577	0.0363	0.0468	0.0475
Panel B - Dependent Variable: Alternative Measure. (probit marginal effects)						
CSES Index	-0.079 (0.022) ***	-0.031 (0.020) ***	-0.029 (0.019) ***	-0.059 (0.015) ***	-0.024 (0.013) *	-0.026 (0.013) **
Respondent's years of education		0.007 (0.001)	0.007 (0.001) ***		0.005 (0.001) ***	0.005 (0.001) ***
Log per capita assets			0.005 (0.004)			-0.004 (0.002)
Regression Wald Chi-Squared	58.56 ***	101.83 ***	103.07 ***	83.43 ***	99.70 ***	114.06 ***
Pseudo R-Squared	0.051	0.0912	0.0936	0.0728	0.1082	0.1110
Panel C - Dependent Variable: Difficulty with at least one Activity of Daily Living. (probit marginal effects)						
CSES Index	0.118 (0.041) ***	0.095 (0.042) **	0.095 (0.042) **	0.035 (0.038)	0.001 (0.043)	0.001 (0.042)
Respondent's years of education		-0.005 (0.003) *	-0.005 (0.003)		-0.007 (0.004) *	-0.007 (0.004) *
Log per capita assets			-0.006 (0.008)			0.001 (0.008)
Regression Wald Chi-Squared	135.53 ***	136.00 ***	147.05 ***	80.08 ***	91.79 ***	91.85 ***
Pseudo R-Squared	0.0480	0.0491	0.0493	0.0316	0.0330	0.0330
Panel D - Dependent Variable: log of height (OLS regression results)						
CSES Index	-0.021 (0.005) ***	-0.012 (0.005) **	-0.012 (0.005) **	-0.023 (0.005) ***	-0.014 (0.005) ***	-0.012 (0.005) **
Respondent's years of education		0.002 (0.000) ***	0.002 (0.000) ***		0.002 (0.000) ***	0.001 (0.000) ***
Log per capita assets			0.003 (0.001) ***			0.004 (0.001) ***
Regression F-test	4.90 ***	7.78 ***	9.82 ***	7.38 ***	8.80 ***	9.07 ***

Men (N=7654)			Women (N=8789)		
Equation:					
	(3)	(4)	(5)	(3)	(4)
R-Squared	0.0295	0.0507	0.0600	0.0439	0.0552
					0.0645

*, **, *** significant at 10%, 5% and 1%. Robust (clustered) standard errors in parentheses.
All regressions include controls for age, marital status and location dummies, survey year and whether the respondent required help to answer the demographic questions.

Table 4

Effects of CSES on Adult Health, Controlling for Parental Background

	Men					Women				
	Main measure (probit marginal effect)	Alt. health measure (probit marginal effect)	Difficulty with at least one ADL (probit marginal effect)	log height (OLS)	Main measure (probit marginal effect)	Alt. health measure (probit marginal effect)	Difficulty with at least one ADL (probit marginal effect)	log height (OLS)		
CSES Index	-0.038 (0.047)	-0.013 (0.018)	0.092 (0.046)**	-0.007 (0.005)	-0.075 (0.045)*	-0.022 (0.014)*	0.005 (0.047)	-0.014 (0.005)***		
Respondent's years of education	0.013 (0.004)***	0.004 (0.002)***	-0.005 (0.004)	0.001 (0.000)**	0.014 (0.004)***	0.004 (0.001)***	-0.008 (0.004)*	0.001 (0.000)**		
Log per capita assets	0.004 (0.009)	0.005 (0.004)	-0.007 (0.009)	0.003 (0.001)***	0.010 (0.009)	-0.004 (0.002)*	-0.001 (0.009)	0.004 (0.001)***		
Mother had completed primary school	0.046 (0.058)	0.006 (0.018)	-0.014 (0.052)	0.014 (0.005)***	-0.016 (0.040)	-0.001 (0.010)	-0.014 (0.044)	0.008 (0.005)		
Father had completed primary school	0.056 (0.051)	0.017 (0.021)	0.049 (0.046)	0.004 (0.005)	0.079 (0.041)**	0.023 (0.012)**	-0.057 (0.042)	-0.001 (0.005)		
Parental residence was primarily urban	0.037 (0.030)	0.008 (0.012)	-0.028 (0.032)	-0.003 (0.004)	-0.043 (0.027)	-0.007 (0.006)	0.021 (0.030)	-0.002 (0.003)		
N	6517*	6518	6514	5634	7425	7428	7424	5283		
Regression Wald Chi Squared test [regression F-test]	164.11**	117.79***	137.99***	[6.80***]	123.90***	118.34***	108.07***	[5.33***]		
Pseudo R-Squared [R-Squared]	0.0631	0.0999	0.0514	[0.0743]	0.0568	0.1311	0.0408	[0.0728]		
Joint f-test of significance of parental background variables	20.86**	13.17	4.27	3.30***	11.16	15.33*	13.78	0.99		

*, **, *** significant at 10%, 5% and 1%. Robust (clustered) standard errors in parentheses.

All regressions include controls for age, marital status and location dummies, survey year and whether the respondent required help to answer the demographic questions, and dummies for father's occupation.

Table 5

2SLS results

Men (N=6517)					Women (N=7425)				
Panel A - Dependent Variable: Good Health Status.									
CSES Index	-0.581 (0.104)***	-0.434 (0.141)***	-0.430 (0.138)***	-0.402 (0.101)***	-0.107 (0.159)	-0.091 (0.161)			
First stage F-test of joint significance of instruments	28.12***	14.98***	14.95***	50.70***	11.68***	11.55***			
Overidentification test [p-value]	[0.1358]	[0.1736]	[0.1861]	[0.1750]	[0.1922]	[0.2194]			
	N	6517			7425				
Panel B - Dependent Variable: Alternative Measure									
CSES Index	-0.262 (0.076)***	-0.165 (0.108)	-0.157 (0.107)	-0.229 (0.076)***	-0.107 (0.159)	-0.070 (0.060)			
First stage F-test of joint significance of instruments	28.09***	14.98***	14.94***	50.75***	11.73***	11.60***			
Overidentification test [p-value]	[0.2844]	[0.3166]	[0.3612]	[0.2066]	[0.1129]	[0.0962]			
	N	6518			7428				
Panel C - Dependent Variable: Difficulty with at least one Activity of Daily Living									
CSES Index	0.173 (0.096)*	0.097 (0.124)	0.090 (0.124)	0.081 (0.097)	-0.108 (0.160)	-0.121 (0.162)			
First stage F-test of joint significance of instruments	27.97***	14.95***	14.91***	50.68***	11.73***	11.60***			
Overidentification test [p-value]	[0.8843]	[0.8735]	[0.8628]	[0.1808]	[0.1513]	[0.1507]			
	N	6514			7424				
Panel D - Dependent Variable: log of height (OLS regression results)									
CSES Index	-0.063 (0.011)***	-0.043 (0.014)***	-0.039 (0.014)***	-0.046 (0.010)***	-0.017 (0.017)	-0.013 (0.017)			
First stage F-test of joint significance of instruments	26.62***	14.89***	15.09***	43.00***	11.15***	10.91***			
Overidentification test [p-value]	[0.0011]	[0.0048]	[0.0046]	[0.1808]	[0.3937]	[0.4235]			
	N	5633			5283				
Includes control for education	N	Y	Y	N	Y	Y			Y
Includes control for assets	N	N	Y	N	N	Y			Y

*, **, *** significant at 10%, 5% and 1%. Robust (clustered) standard errors in parentheses.

All regressions include controls for age, marital status and location dummies, survey year and whether the respondent required help to answer the demographic questions. Each estimate relates to a separate regression.