The Impact of Education on Health Status: Evidence from Longitudinal Survey Data

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Abstract

Using the NLSY79 panel data set from 1979-2006 for a cross-section of 12,686 individuals, this paper investigates the effect of educational attainment on the health status of an individual as measured by "the inability to work for health reasons." The present study bridges the gap in the literature by using the fixed-effects model, random-effects model, between-effects, and the Arellano-Bond dynamic model to analyze the impact of education on health status. We use these alternative models to control unobserved heterogeneity. Educational attainment has a statistically significant and positive effect on the quality of an individual's health status.

Keywords: Education, Health Status, Fixed-Effects, Random-Effects, Between-Effects, Arellano-Bond Model

JEL Classification: I12, I20

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1. Introduction

The positive association between education and health is widely studied, reported, and accepted by health economists (Adams, 2002). The remaining dispute, however, relates to one of causation versus association (Fuchs, 1982; Berger and Leigh, 1989; Silles, 2009). One body of literature contends that education causes better health as it improves the technology of the health production function (Grossman, 1972). Education also increases the lifetime earnings of individuals, making the opportunity cost of becoming ill high and thus discouraging them from engaging in health-reducing activities (Cowell, 2006).

Another strand of the literature, however, casts doubt on whether better education leads to better health. This strand argues that the causation may be reverse, or there may be no causal relationship between education and health based on the assumption that there is a third missing factor such as the rate of discount (Fuchs, 1982), heredity (Rosenzweig, 1995), or preferences that affect both education and health (Silles, 2009).

Furthermore, there are three main issues related to the education-health relationship. The first pertains to the definition of health status itself in that different variables are used as measures of health status. The second has to do with the endogeneity issue related to individual specific-effects (unobserved heterogeneity) which introduces bias in estimating the education parameter's effect on health. The third is the endogeneity problem associated with reverse causation from the determinants of health status. This paper re-examines the three issues using U.S. panel data from

the National Longitudinal Survey of Youth 1979 (NLSY79) for the years 1979-2006 for a cross-section of 12,686 individuals in the survey.

There are many definitions of health status, most of which are subjective in nature. Generally, health levels are measured by mortality and morbidity rates, work-days lost, self-evaluation of health status, and physiological measures (Berger and Leigh, 1989). Economists have also traditionally used the days of normal activity limited by illness as a measure of health status (Farrell and Fuchs, 1982; Berger and Leigh, 1989). In this paper, we adopt the definition of health status as measured by *the inability to work for health reasons,* based on the NLSY79 survey since such disabilities have important implications for household employment and earnings of men and women, especially over the business cycles (Burkhauser, et al., 2001). Using data developed from the 1978 Survey of Disability and Work, Butler, et al. (1987) have demonstrated that a tetrachoric correlation coefficient measure which focuses on the symptom of disease (such as arthritis) is a superior estimate in self-reported health status variables. Heiss (2010) is of the opinion that self-reported health status has been found to be a useful and powerful measure since it maps the high dimensional and complex concept of health into one dimension using individual perceptions and judgments. Using a battery of tests, Benitez-Silva et al. (2004) are also unable to reject the hypothesis that self-reported disability is an unbiased indicator of health status.

In previous studies, the endogeneity of education due to individual specific-effects was either not addressed (as in Grossman, 1975), or was taken into account by using instrumental variables (Berger and Leigh, 1989; Ardent, 2005; Lleras-Muney, 2005), or by using the sibling fixed-

effects model (Berhman and Wolfe, 1989). The use of parental relationship as an instrumental variable for dealing with the endogeneity problem in earlier studies was criticized on the ground that it was related to the dependent variable (Griliches, 1977; Bound and Solon, 1998).

In recent studies, however, there is a wider use of institutional or policy characteristics such as compulsory education laws, or the availability of local colleges as instruments (Currie and Moretti, 2003; Park and Kang, 2008; Tenn et al., 2010). These instruments are criticized on the basis of exclusion restriction since they vary by birth cohort and gender, or by birth cohort and geographic location (Tenn et al., 2010). The difficulties in the selection of instrumental variables are well documented in the literature (Bound et al., 1995; Staiger and Stock, 1997). To overcome the hurdles associated with selecting instrumental variables to control for endogeneity, we use alternative approaches, namely, the individual fixed-effects, random-effects models, between-effects also known as between-estimator model, and the Arellano-Bond dynamic model. The application of the fixed-effects model and the Arellano-Bond model is the contribution of this paper to the growing literature on the impact of education on health status in that we are able to control for both unobserved individual heterogeneity and reverse causation, subject to the assumptions of Arellano-Bond. To the best of our knowledge, there is no published work on the effect of education on health which utilizes the fixed-effects and the Arellano-Bond model from which we obtain the evidence that the causation runs from education to health.

Our panel starts from 1979 where the cohort age ranges between 14 and 22 years of age and ends in 2006. For the year 2006, the age of the cohort ranges from 41 to 49. Thus, there may be the perception that college education takes place primarily between ages 17-25 and hence education

does not vary after the age of 25 years (de Walque, 2010). Recent data by the National Center for Education Statistics (NCES), however, suggest that the number of older students attending schools has been growing along with the younger students. Between 1990 and 1999, the enrollment of students under the age of twenty-five years increased by eight percent. During the same period, enrollment of persons 25 and over rose by seven percent. In 1997, the average age of community college students was 29, with 46 percent of the students being over the age of 25. From 1999 to 2010, the NCES projects an increase of nine percent in the number of students over the age of 25 (Miller, 2001). This fact is important to dispel the wrong perception that education does not vary after certain age. A closer inspection of the NLSY79 data reveals that older cohorts do get education beyond the presumed traditional age.

The paper is organized as follows. The next section gives a review of selected literature. Section 3 discusses the theoretical model and data used for the study, while Section 4 presents and interprets the results. The last section draws some conclusions based on the results.

2. Review of Selected Literature

Many studies have found the relation between the education of individuals and their health to be positive and statistically significant (Silles, 2009). In earlier literature, it was assumed that education has a positive relationship with income which in turn has positive effect on the health status (Antonovsky, 1967). Studies which emerged later, however, found not only a positive relation between education and income, but also a direct correlation between education and health, even after controlling for income (Auster et al., 1969; Newhouse and Friedlander, 1980; Taubman and Rosen, 1982). Grossman (1972) articulated the idea that education improves the

efficiency of the health production function which in turn improves the health status. Rosenzwieg (1995) is of opinion that education helps individuals to employ a more efficient mix of inputs in the health production function. Cowell (2006) also argues that education enhances the earnings potential such that individuals will avoid being involved in health-reducing activities since the opportunity cost of being ill in the future is high. In this context, the causation runs from education to health.

Some researchers are, however, skeptical of the fact that education causes the health status to improve because there is a missing variable that affects both education and health. According to Fuchs (1982), this missing variable is the rate of discount, whereas Rosenzweig and Schultz (1983) argue that the missing variable may be an endowment such as hereditary ability that affects both education and health. There may also be a case of reverse causation in the form of poor health that hinders attaining more education (Currie and Hyson, 1999). In his analysis, Grossman (1975) deals with the question of missing variables and reverse causation by using proxies such as parental education, test scores, and health at the high school level. However, his analysis does not deal with the unobserved heterogeneity (Arendt, 2005).

Wolfe and Behrman (1987) deal with endogeneity due to unobserved heterogeneity by applying the within-family correlation technique. They collect data for sisters in Nicaragua since they are expected to have the same childhood background and control for unobserved elements related to childhood health status. They find that the mother's education has no significant effect on her children's health status. In another study, however, Behrman and Wolfe (1989) find that the women's education appears to make them healthier.

The unobserved heterogeneity and endogeneity problems are also typically dealt with by using instrumental variables. Berger and Leigh (1989) use the per capita income and per capita expenditures on education in the state of birth as instrumental variables. The result of their study shows that education has positive and significant impact on the health status. The instruments may, however, be related to the per capita expenditures on health which might make them questionable (Arendt, 2005). Adams (2002) uses the quarter of birth as an instrument since it affects one's educational attainment and finds a positive, but marginally significant effect of educational attainment on health. Lleras-Muney (2005) uses compulsory school and child labor laws in thirty states from 1915 to 1939 as instruments for education and finds that they have a significant effect in reducing the mortality rate. Currie and Moretti (2003) employed data on the availability of colleges in the woman's county in her seventeenth year as an instrument for maternal education and find that maternal education improves infant health as measured by birth weight and gestational age. Using panel data of school reforms as an instrumental variable for education in Denmark, Arendt (2005) finds that the effect of education on the three alternative measures of health (including self-reported health, body mass index, and never been smoking) is inconclusive. In a recent study, Silles (2009) also uses changes in compulsory schooling laws in the United Kingdom as an instrumental variable and finds a positive and significant effect of education on health.

As is evident from the above discussions, there is no definitive answer as to whether the instruments are weak, or in some cases the results have very low precision. Previous studies of the relation between education and health are either based on a cross-sectional data framework

(Berger and Leigh, 1989; Adams, 2002), or synthetic cohort analysis (Lleras-Muney, 2005). The Arendt (2005) study uses panel data, but does not apply the fixed-effects model to control for unobserved heterogeneity. In the next section, we specify the theoretical model, describe the determinants of health status, and discuss the survey data used for our analysis.

3. The Theoretical Model and Data

Our basic equation for estimating the impact of education on health status is based on the standard formulation in most of the previous studies (Grossman, 1972; Berger and Leigh, 1989; Arendt, 2005; Lleras-Muney; 2005; and Silles, 2009) as given below.

$$HST_{it} = \alpha_i + \gamma\, EDU_{it} + X_{it}\,\beta + \varepsilon_{it} \qquad (1)$$

where α_i is the unobserved heterogeneity (also known as individual-specific effects). HST_{it} is a measure of the health status of individual i at time t, set as 1 when *the individual is not limited to work now for health reasons*, zero otherwise. EDU_{it} is the educational attainment of individual i at time t measured as years of schooling completed. X_{it} denotes a vector of the control variables such as income, gender, race, marital status, family size, residence in metropolitan area, and region of residence of individual i at time t. ε_{it} is the disturbance term which accounts for omitted factors and other random errors. The OLS estimates of the above equation can also be obtained by assuming α_i to be constant.

Among the control variables used in this study, the effect of income on the health status has been addressed in a number of studies. Generally, income is expected to have a positive effect on health since a higher level of income permits more access to consumption of higher quality of goods and services, better housing, and medical services which are expected to positively affect

the health status (Fayissa and Gutema, 2005). Other researchers, however, argue that higher income may not necessarily result in better health beyond some threshold level of affluence and may, in fact, lead to stressful and unhealthy lifestyles (Auster et al., 1969; Rodger, 1979; Fuchs, 1994). Thus, the impact of income on health status is ambiguous.

Similarly, the impact of family size on the health status cannot be predicted *a priori*. Economists have formulated a variety of models linking family size with child health outcomes, beginning with the quantity/quality trade-offs described by Becker (1960). It is difficult to measure the causal impact of an increase in the number of children on child health outcomes because households select into larger or smaller families, and a family's optimal trade-off between the quantity and quality of children may be simultaneously determined (Filmer, et al., 2009). Based on the above arguments, the impact of family size on the health status of individuals in our sample is ambiguous.

Marital status is considered to have a positive effect on the health status of individuals since married individuals receive better home care and place a higher value on health relative to other market goods and risky activities than unmarried persons, on average (Thornton, 2002). While it is possible that individuals in poorer health are less likely to get married so the causation runs from health to marital status, Taubman and Rosen (1982), however, find no empirical evidence for such reverse causation hypothesis.

We include race as a control variable to estimate if race has a positive or negative effect on the health status. There are some studies which argue the minority groups that have less access to

health care services to be negatively impacted relative to other groups (Weinick et al., 2001).

Therefore, we expect a negative relationship between minority race and the health status.

The impact of gender on the health status of individuals has been neglected in previous studies (American Academy of Pediatrics, 2000). A recent study by Mitchell (2004) found that women and vulnerable groups of women were more likely to experience significant health and financial problems which affect their health status, different from men. Therefore, we hypothesize that there is a negative relationship between being a female and the measure of health status.

Residence in urban areas and in different regions of the U.S. is also included in our model to capture the impact of environmental and other factors. These variables are proxies for potential negative or positive health related factors as pollution, congestion, and access to better medical care (Thornton, 2002). Consequently, we cannot predict the relationship between the place or region of residence and the health status. We first estimate equation (1) above using the OLS, logit, between estimator, fixed-effects, and random-effects models.

The study is further extended to address the issue of the factors which explain the health status being either predetermined, or endogenous. Since current-period heath status depends on its value in the past, a dynamic variant of equation (1) above known as the Arellano-Bond (1991) model (Fayissa et al., 2008) is specified as follows:

$$\Delta HST_{it} = \delta\, \Delta HST_{it-1} + \gamma\, \Delta EDU_{it-1} + \Delta\, X_{it}\, \beta + \alpha_i + \varepsilon_{it} \qquad (2)$$

where ΔHST_{it} is the first difference of the health status of individual i during period t; ΔHST_{it-1} is the lagged difference of the dependent variable, ΔEDU_{it-1} is the lagged level and assumed to be a

predetermined endogeneous variable, and ΔX_{it} is vector of exogenous variables. α_i and ε_{it} are assumed to be independent over all time periods for individual i. The term α_i represents individual-specific effects that are distributed independently and identically over the individuals and ε_{it} is the noise stochastic disturbance term and is also assumed to be distributed independently. This model controls for endogeneity of education as long as the endogeneity is fixed over time, not changing within each period.

The empirical analysis of the present paper is based on data from the National Longitudinal Survey of Youth 1979 (NLSY79) which is a nationally representative sample of 12,686 young men and women who were between 14 and 22 years of age when they were first surveyed. These individuals were interviewed annually from 1979 through 1994 and have been interviewed on a biannual basis since 1994 (i.e., 1996, 1998, 2000, 2002, 2004, and 2006).

Although the original data set started with 12,686 respondents, the number of respondents decreased to 7,764 by the end of 2006 due to attrition which amounts to less than 2 percent per year on average. These observations are treated as missing information such that they do not create a systematic bias. Unanswered questions are also treated as missing data.

One of the survey questions asks, *"Would your health limit the kind of work you do now?"* We use this variable as a measure of the health status (our dependent variable), following Berger and Leigh (1989). The wording and structure of the questions regarding the variables used in this study remain the same over the survey and are exactly comparable.

<<Table 1 here>>

Table 1 gives the definitions of the variables while Table 2 presents the descriptive statistics of the variables used in the study. It is important to note that only 0.7 percent of the survey population consider that "*their health limits the kind of work they do now*" in 1979 which eventually decreases to only 0.4 percent in 2006. This implies that there is a marginal improvement in health status from 0.983 in 1979 to 0.986 in 2006. Intuitively, it appears that the health status of individuals has improved with age, but this may be due to their consciousness of the importance of maintaining active working-life to control stresses and diseases such as diabetes and high blood pressure. The educational attainment increases from 10.4 years of schooling in 1979 to 13.6 years of schooling in 2006 (see, Table 2) whereas the nominal annual income of individuals in the sample rises from $15,556 to $46,548 on average, or from $36897 to $39548 in constant dollars using the 2000 CPI as a base. The demographic composition of our sample consists of 51 percent females, 27 percent Blacks, and 16 percent Hispanics. Family size also decreases from an average of 4 in 1979 to 3 in 2006.

<<Table 2 here>>

4. Results

The estimation of the pooled time series cross-section data yields the OLS within estimates assuming α is constant in equation (1) above. Generally, when the dependent variable is a binary variable which assumes a value of 1 or 0, nonlinear models such as the logit or probit models are preferable based on the range of the predicted values being 0 to 1 and the model being based on a propensity to have a disability. The OLS model can always be considered a linear approximation. The results of both the OLS and logit models suggest that education and income

have positive and statistically significant impact on the health status (see, Table 3). For example, the coefficient of education of 0.0017 suggests that each year of schooling improves the health status of individuals by 0.17 percent, which is quite large relative to the observed percentage of the sample with work-limiting disability. The logit model results also show similar trend of the impact of education and income on the health status.

<< Table 3 here>>

Individual-specific unobserved heterogeneity may be fixed or random over time. Broadly speaking, time-related random unobserved heterogeneity or individual-specific-effect heterogeneity is controlled by the random-effects model and between-effects estimator. The individual unobserved heterogeneity which is fixed over time is controlled by the fixed-effects model. The results from the logit with fixed-effects, fixed-effects, random-effects, and between-effects models also indicate that education has positive and statistically significant effect on the health status as reported in Table 4. In this context, our study makes a contribution to the literature by the using the between estimator to control for the random deviation of the variables from the long-run means. However, the between-effects model does not control for the individual unobserved heterogeneity which is typically captured by the fixed-effects model. To determine whether the fixed-effects or random effects model is appropriate, we employ the Hausman–test of difference between the fixed and random effects models.

<<Table 4 here >>

The Hausman-test rejects the random-effects model in favor of the fixed-effects model. The random-effects and between-effects estimators show significant and larger coefficients for

education and income as compared to the fixed-effects model because they fail to take into account the correlation of omitted individual-specific effects with explanatory variables.

Another interesting issue with respect to the impact of education on the health status relates to whether the relationship between health status and education is linear, or non-linear. Although the results are not reported here, we find that the coefficient of education squared in all the above models is insignificant, suggesting no evidence of non-linear relationship between the health status and education. To check the robustness of the results, we adopt an alternative definition of the health status which is slightly different from the one employed here (*Does your health limit the kind of work you do?*) and find very similar impact of education on the health status.

Our results for the different models we used also show that income has a positive and statistically significant impact on the health status. These results confirm the findings of McDonough et al. (1997) who found that income level was a strong predictor of mortality, especially for persons under the age of 65 years. As we have discussed earlier, there is no consistent relationship between some of the demographic variables (such as family size, gender, race, and marital status), residence in urban areas, and in different regions of the U.S. which we included in our model to capture the impact of environmental and other factors. We note that the race and gender variables drop out from the fixed-effects model in Table 4, that is, they are included in the fixed effects.

Furthermore, the coefficients of equation (2) are estimated using the Arellano-Bond (1991) GMM estimator as reported in Table 5. The results corroborate with the findings of the alternative models we previously employed to control for endogeneity and unobserved

heterogeneity, i.e. educational attainment has a positive and statistically significant effect on health. We recognize that the simple Arellano-Bond dynamic model may not capture the endogeneity problem in its entirety. As a specification test of the relationships between education and health, we tested education as a function of health status with the same explanatory varables, which did not reveal reverse causation running from health status to education (i.e. the impact of health status on education was not been found to be significant (These findings are not reported here, but can be obtained from the authors).

<<Table 5 here>>

5. Conclusion

This paper has examined the question of causation and association between education and the health status of individuals while controlling for demographic and environmental variables. Previous studies of the relation between education and the health status have been the subject of criticism for using weak instruments and not fulfilling the requirements of exclusive restriction in the selection of instruments (Arendt, 2005; Tenn, et al., 2010). The Arellano-Bond estimator controls for endogeneity which is fixed over time, which is not perfect, because endogenous changes in the effect can still be present, but offers an approach different from the literature, with similar positive findings. As discussed earlier, we find no evidence of reverse causation from health status to education (i.e. the positive effect of education on health status which our results show is not confounded by endogeneity.

All the alternative models we employed in our study suggest that education has a positive and significant effect on the health status of individuals, confirming the results of previous studies by

Grossman (1975), Berger and Leigh (1989), Adams (2002), Lleras-Muney (2005), and Cowell, (2006). An important contribution of our study to the literature is that we demonstrate the significant role education plays in improving the health capital of individuals in terms of enhanced work effort and increased productivity, although our methodologies differ from those used by previous researchers. Our study also suggests that investment in human capital might prove to be a cost-effective means of realizing better health since the measured effects of education on health are large (Auster, et al. 1969; Lleras-Muney, 2005). One policy implication which can be drawn from the study is that investment in education (in the form of financial aid, scholarships, or instructional resources) may yield larger long-run private and social returns.

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Table 1. Definitions of Variables

Variables	Variables Definition
HST	Inability to work due to health now (Health Status)
EDU	Years of education attainment
WAG	Wages $*10^{-4}$
FSZ	Family size of the individual$*10^{-2}$
URB	Individual lives in urban area
MST	Marital Status
BLK	Black
HSP	Hispanic
GEN	Gender
SMSA	Standard Metropolitan Statistical Area
REG1	Region 1 (Northeast)
REG2	Region 2 (North Central)
REG3	Region 3 (South)

Table 3. OLS and Logit Estimates of Health Status

Variables	OLS Model			Logit Model		
	Coefficients		Standard errors	Coefficients		Standard errors
EDU	0.0017	***	0.0002	0.0365	**	0.0116
INC	0.0005	***	0.0001	0.7178	***	0.0336
FSZ	-0.1543		0.0285	0.0674		1.3737
URB	0.0007		0.0015	0.1394	*	0.0799
MST	0.0054	***	0.0010	-0.0895		0.0552
BLK	-0.0025	**	0.0011	-0.1425	**	0.0620
HIS	-0.0064	***	0.0014	-0.3087	***	0.0686
GEN	0.0002		0.0010	0.0185		0.0522
SMSA	0.0036	**	0.0015	-0.0134		0.0794
REG1	0.0012		0.0016	0.0095		0.0860
REG2	0.0022		0.0013	0.0636		0.0750
REG3	0.0021		0.0014	0.0874		0.0721
Intercept	0.9543	***	0.0034	2.7300	***	0.1770

*significant at 10%, **significant at 5% ***significant at 1%

Table 4. Effect on Health Status Controlling Heterogeneity

	Logit Fixed-Effect			Fixed-Effect			Random-Effects			Between-Effect		
	Coef.		Std. Err.	Coef.		Std. Err.	Coef.		Std. Err.	Coef.		Std. Err.
EDU	0.0382	**	0.0188	0.0006	**	0.0002	0.0010	***	0.0002	0.0023	***	0.0005
INC	0.0818	**	0.0260	0.0001	**	0.0001	0.0002	**	0.0001	0.0042	***	0.0005
FSZ	3.5800	**	1.727	0.0569	*	0.0314	0.0018		0.0300	-0.8439	***	0.1047
URB	0.2080		0.1079	0.0023		0.0017	0.0019		0.0016	-0.0053		0.0050
MST	-0.3008	***	0.0779	-0.0037	**	0.0012	-0.0010		0.0011	0.0296	***	0.0034
BLK							-0.0034		0.0022	0.0009		0.0023
HIS							-0.0076	*	0.0026	-0.0019		0.0027
GEN							0.0002		0.0019	0.0000		0.0019
SMSA	0.0398		0.1165	0.0010		0.0019	0.0018		0.0017	0.0098	**	0.0049
REG1	0.1030		0.1888	0.0019		0.0031	0.0017		0.0023	-0.0002		0.0037
REG2	0.2284		0.2284	0.0032		0.0023	0.0027		0.0018	0.0016		0.0031
REG3	0.2037		0.2037	0.0031		0.0024	0.0025		0.0019	0.0037		0.0031
Intercept				0.965	***	0.0046	0.9632	***	0.0043	0.9515	***	0.0088

Hausman test: rejects Random Effect Model in favor of Fixed Effect model
$\chi^2_{(8)}$= 89.93, p=0.000

*significant at 10% **significant at 5% ***significant at 1%

Table 5. Arellano-Bond dynamic panel data estimation results

Variables	Coefficient Estimates	Standard Errors	
HST(LD)	0.2387	0.0050	***
EDU(D(1))	0.0024	0.0007	**
INC(D(1))	0.0001	0.0001	
FSZ(D(1))	0.0700	0.0053	
URB(D(1))	0.0005	0.0029	
MST(D(1))	0.0018	0.0023	
SMSA(D(1))	0.0004	0.0031	
REG(D(1))	0.0003	0.0056	
REG(D(2))	0.0069	0.0069	*
REG(D(3))	0.0075	0.0075	*

Sargan test of over-identifying restrictions: $\chi^2_{(20)}=25586$ p> $\chi^2_{(20)}=0.00$

Arellano-Bond test of the null of AR(1) residual errors z = -189.84***

Arellano-Bond test of the null of AR(2) residual errors z = 15.05***

***Significant at 1% **significant at 5% * significant at10%.While the suffix *D(1)* after each variable denotes the number of time each variable was differenced, *LD* denotes the lagged difference . The *HST* is treated predetermined, while *EDU* is treated as an endogenous variable.