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**The Effect of Female and Male Health on Economic Growth:
Cross-Country Evidence within a Production Function Framework**

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Abstract

Adopting a production function based approach, we model the role of health as a regular factor of production on economic growth, and use disaggregate measures of male and female health capital using principal components analysis. Allowing for the dynamics of TFP to be embedded in the production function, we estimate it both in levels and in growth rates to distinguish between long- and short-run effects. We use appropriate panel cointegration methodology to control for endogeneity, cross-sectional dependence and heterogeneity. Our main finding is that while male and female health capital stock has a significantly positive effect on level of output in the long-run, changes in gender disaggregated health capital has a negative or insignificant effect on output growth in the short-run.

Key Words

health and economic development
economic growth
endogeneity
panel data
TFP convergence
economics of gender

JEL Classification

I15; O47; J16

1. Introduction

The importance of health for a country's economic growth has been well documented in the literature: Barro (1996), Bloom *et al.* (2003), Bloom *et al.* (2000), Bhargava *et al.* (2001), McDonald and Roberts (2002), Knowles and Owen (1995). Health, as a measure of human capital, has been included in many cross-country regression studies. These studies in general, find a positive contribution of health on growth. A healthy population is able to contribute directly to economic growth through its influence on increased productivity and income (Bloom *et al.* 2004), and indirectly through its influence on investment in education (Barro 1996), increased savings (Ashraf *et al.* 2008), and reduced fertility (Barro 1996). Although basic economic intuition suggests that health should matter for growth, the relationship is not conclusive. For example, in most cross-country growth regressions which include health, it is not clear whether health directly influences economic growth or whether it acts as a proxy for omitted variables (Barro and Sala-i-Martin, 1995).

Health may also appear to have a negative impact on growth. Recently, Acemoglu and Johnson (2007) (henceforth AJ), by modelling the health-growth relationship in a neo-classical framework where health is treated as a regular factor of production, addressed the issue of health, measured by life expectancy and economic growth. By constructing an instrument which is dependent on exogenous shocks to national health generated by improvements in health technology using the pre-intervention distribution of mortality from 15 diseases, AJ showed that increases in life expectancy are not significantly positively correlated with economic growth. They showed that instrumented changes in life expectancy have a large effect on population: a 1 percent increase in life expectancy leads to an increase in population of about 1.5 – 2 percent, but a much smaller effect on total GDP both initially and in the long-run. Accordingly, the impact of an increase in life expectancy on per capita income is insignificant or negative. The interpretation of the insignificant or negative impact of life expectancy on GDP per capita is that, in the face of population growth, the other factors in the production function, capital and land in particular, did not adjust. As population increases, the capital-labour ratio decreases, which eventually lead to a decrease in the per capita income.

The findings in AJ appear to contradict much of the preceding literature, surveyed in Weil (2007) and Bloom *et al.* (2004), and Lorenzen *et al.* (2008) (henceforth LMW) which generally find that countries with better health achieve higher rates of economic growth. LMW used an endogenous growth framework where a higher stock of health spurs growth by facilitating technological innovation and/or technological adoption allowing productivity growth to be positively correlated with the level of health. LMW used seventeen instruments based on a malaria ecology index, climate variables and geographic features and found that high adult mortality, as a measure of health, reduces economic growth by shortening time horizons, because a greater risk of death during the prime productive years is associated with higher levels of risky behaviour, higher fertility, and lower investment in physical capital.

Aghion *et al.* (2010) adopted a unified framework combining both neoclassical and endogenous type growth models and showed that the level and the accumulation of health, measured as life expectancy, have significant positive effects on growth of per capita GDP, even when they had used the LMW instruments for the level and accumulation of life expectancy. The key to the difference in their findings compared with AJ is convergence. While AJ showed that countries whose life expectancy grew rapidly did not tend to experience more rapid income growth; Aghion *et al.* (2010) showed that it was only because these same countries also typically started with lower initial levels of health, which offset the positive growth effect of their rapid improvement in health.

In a similar effort, Bloom *et al.*(2009) highlighted the issue of adjustment of national income to changes in health conditions and argued that the dynamic relationship between health and income as formulated in AJ where current income is modelled as a function of, and adjusts instantaneously to, current health may be incorrectly specified. This is because there exist both theoretical and empirical reasons to expect income to adjust non-instantaneously to changes in population health. The conditional convergence hypothesis in the modern economic growth literature suggests a slow convergence of income to its steady-state level: a convergence rate of about two percent per year is a common estimate (Barro and Sala-i-Martin 2004; Durlauf, Johnson *et al.* 2005). It is likely that when health improves, income adjusts slowly to the new steady state. The effect of initial conditions on economic growth was not controlled for in AJ's study, but there is a strong convergence in health, particularly in the countries which start with relatively good initial health conditions have relatively smaller subsequent health gains (Deaton *et al.*2006). Bloom *et al.*(2009) showed that once they had controlled for initial health conditions, the estimated effect of changes in life expectancy on economic growth became positive.

In relation to the discussions in the preceding paragraphs, this paper models differences across cross-countries growth in output as a function of traditional factor inputs, health conditions and technological progress. Our main contribution to the health-growth debate is three-fold. First, we offer an additional insight to the literature in terms of how the health-growth effects responds to a disaggregate measures of female and male health. Second, while it is almost customary to use life expectancy at birth as the measure of health capital, (see; Table 1), we measure female and male health capital using the largest principal component of the three available indicators of health: life expectancy, adult mortality and survival rate. Third, we use a panel methodology based on panel cointegration and error correction modelling (ECM) to distinguish between the short- and long-run effects of health. In doing so, we take account of cross-sectional dependence in the residuals and the presence of heterogeneity in the data.

While the role of gender equality in the economic-demographic transition has received considerable attention over the past years, less attention has been devoted to understand the role of female health on economic growth (Knowles *et al.*2002; Knowles and Owen 1995). In general, it may be expected that better health of women increases the return to educational investments via lower morbidity and lower mortality and therefore enhances their capacity to

participate productively in the labour market with direct consequences for effective labour supply and hence level and growth of economic output (Jayachandran and Lleras-Muney 2009; Albanesi and Olivetti, 2013). In addition, the better health of mothers directly affects the health of children through *in utero* effects and the mothers' ability to breastfeed and nourish their children in other ways (Field *et al.* 2009). In addition, better female health may also lead to lower fertility as a result of not only improved reproductive health but also indirectly as a response to female opportunity costs of child-rearing and changes in the return to education (see Bailey 2006, Galor and Weil 2000, Soares and Falcao 2008).

Many developing countries face gender inequality in health and education. Gender inequality in health could have adverse consequences on the development goals of many countries. Gender inequality in health and access to health services, may slow down the goal of attaining lower levels of child mortality, fertility and better health of future generations (Klasen 2008). This in turn, slows down economic growth. It is plausible therefore that the female and male health capital have differential impacts on economic growth.

The sample of our study covers 83 countries from across the regions of East Asia and the Pacific, Europe and Central Asia, Latin America and the Caribbean, the Middle East and North Africa, South Asia and Sub-Saharan Africa over the 1975-2009 period. In most studies the health status of the economy is measured by life expectancy at birth (see Barro 1996, Bloom *et al.* 2000 and Bloom *et al.* 2004), which reflects the overall mortality level of a population summarising the mortality pattern that prevails across all age groups - children and adolescents, adults and the elderly. However, to circumvent the econometric problem associated with high collinearity between female and male life expectancies at birth, we are motivated to search for alternative measures of gender based health capital to carry out the analysis in this paper. With the aid of principal-component analysis, we have employed the largest principal component based on the following three widely used indicators of health: life expectancy, adult mortality and survival rate. While life expectancy is defined above, adult mortality rate is the probability of dying between the ages of 15 and 60, that is, the probability of a 15-year-old dying before reaching age 60, if subject to current age-specific mortality rates between those ages, and survival rate (survival to age 65) refers to the percentage of a cohort of newborn infants that would survive to age 65, if subject to current age specific mortality rates.

Our econometric methodology, which enables us to explain much of the variation in cross country income in our sample of countries, is based on a production function approach, where a gender disaggregated health capital is treated as additional input to production along with capital and labour. Our framework also explicitly models convergence which has been crucial to reconciling the results of Aghion *et al.* (2010) and Bloom *et al.* (2009) with those of AJ. But convergence in our model do not appear in life expectancies rather through technology, that is, total factor productivity (TFP). This approach makes sense because productivity growth should be positively correlated with the level of health capital. The way our empirical strategies differs from those in the literature (see papers summarised in Table 1) are that we estimate and distinguish between a long- and short-run relationship between

income and gender disaggregated health measures by using a panel cointegration equation and a panel ECM which emphasises variable non-stationarity, cross-sectional dependence as well as parameter heterogeneity.

Our model allows us to test a number of hypotheses. One important question is, with unobserved technology, how much impact health capital has on the level and growth of income after having controlled for factor inputs. This has important policy implications on the allocation of resources to the health sector. If health capital is effective in generating further economic growth, then policy makers may decide to allocate more resources for health services and aim at improving the efficiency of the health sector. The other important question is whether the effect of female and male health capital is uniform across the economy. For example, whether increasing an additional unit of female health capital has the same or different impact on economic growth than that of a male counterpart. This also has important implications for policy makers because if, for example, the impact of female health is transmitted more onto the economy than their male counterpart, then it makes more sense to perhaps allocate more resources to female health services.

Finally, we wish to answer the question through our model whether the health effects for both genders vary or remain same as the dynamics within economies move from the short-run to long-run. We believe this is an important aspect of our paper which offers a reconciliation to previously conflicting findings in the literature. Our results do suggest that, in our sample, while both female and male health impact on economic growth are positive in the long-run thus conforming to the findings of Bloom *et al.* (2004, 2009), Weil (2007), LMW, and Aghion *et al.* (2010), amongst others, these effects can be negative or insignificant in the short-run, thus also lending some support to the findings of AJ. Intuitively, changes in health capital can take place fast in the short-run due to medical advances or proliferations of vaccines but in response to this the time required for the economy to adjust from a particular steady state to another may be long such that the full adjustment only takes place in the long-run.

The rest of this study is structured as follows. Section 2 presents the summary of previous studies and their methodologies in the literature. Section 3 presents the model. Section 4 describes the data. Section 5 presents the empirical results and section 6 concludes.

2. Summary of Previous Studies

Many studies that investigate the effects of human capital on economic growth use education as a proxy for human capital. In this study, we use health capital to measure human capital. Table 1 summarises a few influential studies carried out on the influence of health capital on economic growth. Note that most of these studies use life expectancy to measure health capital (Bloom and Canning 2000, 2003, Bloom *et al.* 1998, 2000) while few studies use survival rates (Bhargava *et al.* 2001, Weil 2007). Weil (2007) employs microeconomic data to investigate the macroeconomic effects of health on GDP per capita. The majority of studies find that health capital has a positive effect on economic growth with the exception of Caselli *et al.* (1996) who attribute this mainly to the use of GMM estimation.

Table 1. Summary of Previous Studies

| Author(s) | Health Capital Measure | Sample | Estimation Technique | Coefficient Estimate | Conclusions |
|---------------------------------------|--|--|---|--|---|
| Barro (1996) | Life expectancy at birth | Panel data over 1960-1990. | Three Stage Least Squares | Life expectancy = 0.042 (Table 1, p.48) | An increase in life expectancy leads to an increase economic growth. |
| Bloom, Canning and Malaney (2000) | Life expectancy in initial year | Cross country and panel data for 70 countries 1965-1990. | OLS, IV | Life expectancy in initial year = 3.28 – 2.64 (Table 1, p.267 cross sectional results) | Life expectancy is found to have a robust effect on economic growth. |
| Bhargava, Jamison, Lau, Murray (2001) | Survival rate | Panel data in five year intervals 1965-1990. | Random Effects | Survival rate = 0.181 and 0.358 (Table 1, pg 431) | Human capital as proxied by the adult survival rate has a significant effect on economic growth particularly in the poorer countries. |
| Bloom, Canning and Sevilla (2004) | Life Expectancy | Panel data in ten year intervals 1960-1990. | Non-linear two stage least squares estimation | Life expectancy = 0.040 (Table 4, p.10) | Health capital has a positive and statistically significant impact on economic growth. |
| Caselli, Esquivel and Lefort (1996) | Life expectancy | Panel data in five year intervals 1960-1985. | Arellano-Bond GMM estimation method | Life expectancy = -0.001 (Table 4, p.37) | Life expectancy does not have a significant effect on economic growth with the use of GMM. |
| Knowles and Owen (1995) | The shortfall in average life expectancy at birth from 80 years. | Cross sectional data on a group of developed and developing countries 1960-1985. | OLS. | Life expectancy for the full sample unrestricted regressions in the range of = 0.342-0.381. (Table 1. P.103) | The existence of a robust relationship between life expectancy and income per capita. |

| Author(s) | Health Capital Measure | Sample | Estimation Technique | Coefficient Estimate | Conclusions |
|------------------------------------|---|---|----------------------|---|--|
| McDonald and Roberts (2002) | The shortfall in life expectancy relative to a benchmark. | Five yearly panels 1970-1984 for 77 OECD and developing countries. | Pooled OLS | Life expectancy for the full sample = 0.120. (Table 1 p. 274) | The coefficient on health capital is significant for the full sample. When the sample is disaggregated by LDCs and OECD countries, health capital has a positive and significant effect on economic growth in the LDCs but not the OECD group. |
| Bloom, Sachs, Collier, Udry (1998) | Life expectancy in initial year | Cross country data over 1965-1990 for 18 African and 59 non-African countries. | OLS | Life expectancy in initial period = 4.25 for Africa and 3.06 for non-Africa (Table 6, p.257) | Life expectancy is found to be one of the main reasons for the gap in growth between Africa and the non-African countries. |
| Sachs and Warner (1997) | Life expectancy in 1970. | Cross country data 1965-1990 for 79 countries (Africa, other fast growing developing and all other developing economies). | OLS | Life expectancy in 1970 = 45.38-47.85 (Table 2, p.345) | The effect on growth of an additional year of life expectancy is higher at lower levels of life expectancy, and almost zero at higher levels of life expectancy. |
| Bloom and Williamson (1998) | Life expectancy in 1960. | 78 Asian and non-Asian countries over 1965-1990. | OLS and IV | Life expectancy in 1960 = 5.81. (Table 2, p.434) | When life expectancy is added to the estimation, population has a significant effect on economic growth. |
| Acemoglu and Johnson (2007) | Life expectancy at birth in 1940, 1980, 1990 | 120 countries | OLS, IV | Life expectancy at birth = 1.17 (OLS estimation, Table 3, p.994) | There is no evidence that an increase in life expectancy leads to faster growth. in income per capita. |
| Lorenzen <i>et al.</i> (2008) | Adult Mortality Rate | 110 countries, 27 Indian states | FE, RE, IV | Adult mortality rate (age 15 – 60) = -7.46 (IV estimation, Table 7, p.102) | Mortality level reduces economic growth. |

A number of studies investigate the reason for the low growth rates experienced by Africa. The lower life expectancy at birth faced by this region due to higher levels of disease and lower quality of health institutions is cited as one factor among others (see Sachs and Warner 1997). Sachs and Warner (1997) find that the effect on growth of an additional year of life expectancy is higher at lower levels of life expectancy, and almost zero at higher levels of life expectancy. The view that poorer countries benefit more from increases in health capital is further supported by Bhargava *et al.* (2001), McDonald and Roberts (2002). Low life expectancy is attributed to be a factor contributing to the lower rates of growth in Africa compared to other regions also in Bloom *et al.* (1998). Similarly, in an investigation of demographic change and economic growth in Asia, Bloom *et al.* (2000) argue that a large part of East Asia's rapid economic growth and South Asia's low progress are due to the influence of differences in demographic factors. They show that during a period of rapid economic progress, life expectancy in East Asia increased substantially between 1965 and 1990. Similarly, Collins and Bosworth (1996) in a study of the reasons for East Asia's rapid growth, show that higher education and life expectancy account for about a 0.75 percentage point per year of increased growth. This is supported by Bloom and Williamson (1998) who show that demographic factors play an important role in East Asia's rapid growth.

A general consensus which emerges in these studies, for example see Sachs and Warner (1997), is that at lower levels of life expectancy, improvements in life expectancy come from developments in public health and eradication of disease, which have a larger effect on economic growth compared to improvements in survival rates experienced at higher levels of life expectancy. These conclusions actually hold true only in the long-run and a failure of these studies have been to not make a distinction between the long- and short-run impacts of health in generating economic growth. Our contribution in this paper is to show that enhancements in health outcome through such the provisions of public health and eradication of diseases may have larger growth effects only in the long run, while in the short-run, as the health improves relatively faster and the economic parameters takes time to adjust to these new health conditions, the effect can be relatively smaller or even negative.

3. The Empirical Model

Following Bloom *et al.* (2004), we adopt a production function based approach to analyse the effect of health on growth. We adapt to their model by including the gender disaggregated effect of health. The production function based approach decomposes sources of growth into two parts: growth in the level of input and growth in TFP. Our inputs include physical capital, labour and human capital as measured by health disaggregated by gender. Our production function thus models output as a function of inputs and technology which is represented for a country i at time t as follows:

$$Y = AK^\alpha L^\beta e^{\phi_1 FH + \phi_2 MH}, \quad (1)$$

where Y is output or real gross domestic product (GDP); A represents TFP; K is physical capital; L is labour force; and human capital consists of two aggregate components of health. We disaggregate this human capital factor into two components by gender: MH , i.e. a measure of male health and FH , a measure of female health. Note that the effect of human capital on output is expressed in exponential form. The main advantage of such a functional form is that it allows the log of Y to be dependent on health status, which is much similar to the specification of the Mincerian regression estimating returns to human capital (Mincer, 1974) where the log of wages depends on level of schooling and health status. Thus a production function specified this way is more compatible with the relationship estimated in microeconomic studies.

Note that our model gives a representation on how output depends on factors of production and TFP. Though we do not explicitly model TFP, i.e. A in our specification, it will account for variables not mentioned on the right hand side of the equation. Moreover, it is also possible that some of these human capital variables actually work through TFP. In this case, estimating the effect of human capital variables will become more complicated as we will have to specify another equation for TFP to model the dynamics of human capital factors and then estimate this with the equation for the production function as a system. The other alternative would be to estimate the production function in reduced form after substituting an expression for A which captures the evolution of human capital. While these are viable approaches, we do not pursue these here but rather model the term A in our production function as a two-way error component disturbances following Wallace and Hussain (1969) and Amemiya (1971). This is explained below.

Taking the logs of our aggregate production function, the following long-run equation for log of output in country i at time t is derived:

$$y_{it} = a_{it} + \alpha k_{it} + \beta l_{it} + \phi_1 FH_{it} + \phi_2 MH_{it}, \quad (2)$$

where y_{it} , k_{it} , and l_{it} are logs of Y , K and L respectively from the aggregate production function (1). The term a_{it} which is the unobservable TFP of country i at time t , is modelled as a two-way error component as follows:

$$a_{it} = a_i^* + a_t^* + v_{it}, \quad (3)$$

where a_i^* represents the unobserved country specific time invariant level of TFP and a_t^* denotes the unobserved time effect represented by the worldwide technology frontier. The combined effect of $(a_i^* + a_t^*)$ gives an account of the steady-state level of TFP in each country. Each country's actual TFP a_{it} deviates from the steady state level by the difference

v_{it} . Note that this difference is not stochastic, but is assumed to follow an autoregressive process of order one AR(1) as in Lillard and Willis (1978). This is a reasonable assumption where a deviation of actual TFP from its steady-state value may be persistent because an unobserved shock to TFP in this period can carry forward to the next or even more periods. We restrict our analysis to the case where v_{it} is a AR(1) process as follows:

$$v_{it} = \rho v_{i,t-1} + \varepsilon_{it}, \quad (4)$$

where $0 < \rho < 1$ can be treated as a convergence coefficient. As time passes, any deviation from long-run steady-state TFP for any country is reduced at a rate $-(1-\rho)$. ε_{it} is a random shock having classical properties.

Estimating the production function, with the error term specified to hold the above characteristics would involve the generalised error component model to the serially correlated case. For example, Baltagi and Wu (1999) propose a feasible generalised least squares procedure which is simple and provide natural estimates of the serial correlation and variance component parameters.

However, rather than estimating a model with a serially correlated error structure, we find it useful to transform our production function into a growth equation. Differencing equation (2) gives us:

$$\Delta y_{it} = \Delta a_t + \alpha \Delta k_{it} + \beta \Delta l_{it} + \phi_1 \Delta FH_{it} + \phi_2 \Delta MH_{it} + \Delta v_{it} \quad (5)$$

Substituting out the error term Δv_{it} using equation (4) and noting that the lagged productivity gap $\Delta v_{i,t-1}$ is the difference between actual output and output at the average world TFP level at time $t-1$ gives us:

$$\Delta y_{it} = \Delta a_t + \alpha \Delta k_{it} + \beta \Delta l_{it} + \phi_1 \Delta FH_{it} + \phi_2 \Delta MH_{it} + (\rho - 1)(y_{i,t-1} - (a_{i,t-1} + \alpha k_{i,t-1} + \beta l_{i,t-1} + \phi_1 FH_{i,t-1} + \phi_2 MH_{i,t-1})) + \varepsilon_{it} \quad (6)$$

Equation (6) enables us to estimate both the long-run and short-run growth impacts of the factor inputs including health. This equation decomposes growth in output into four factors: growth in world TFP; growth in inputs; a catch-up term as the TFP gap is reduced in some countries to enable them to converge to steady-state level TFP at a rate $(\rho-1)$ and an idiosyncratic shock to country's TFP $\varepsilon_{it} \sim IID(0, \sigma_\varepsilon^2)$.

There are several issues worth noting in equation (6). First, focusing on the right hand side variables, it is safe to argue that male and female health are expected to be endogenous with respect to growth rate of output which stem from the covariance of the determinants of

health to the level and growth of income in the economy. Secondly, looking at the health capital variables, it is also easy to see that they do not appear additively in the equation, as a result the resulting equation represents a nonlinear-in-parameter model. Third, the unobserved time-varying heterogeneity induced by common shocks, such as global technology shocks to production function that influence all countries to a different degrees perhaps, will introduce cross-sectional dependence or correlation in the error term rendering conventional estimators inconsistent if these shocks are correlated with the regressors (Coakley *et al.* 2006). We address these issues in our empirical section.

4. Data

Because our methodology involves estimating cross-country production function, we need reliable estimate of capital stock data. Therefore, we use data from Bosworth and Collins (2003) (Henceforth BC) to estimate our growth model. The disaggregated human capital data on male and female health are all taken from the WDI (2011). BC have compiled data on real GDP, capital stock, and the labour force for 83 countries across all regions for the period 1960 – 2003. We have updated these data to 2009. Descriptive statistics on some of the main variables of interest and data sources are provided in Table 2.

Table 2: Descriptive Statistics and Data Sources for Selected Variables: 1975-2009

| Variable | Obs | Mean | Standard Deviation | Minimum | Maximum | Source |
|--|------|--------|--------------------|---------|---------|--------|
| Change in Log Per Capita Income (Constant 2000 \$US) | 2822 | 0.033 | 0.044 | -0.69 | 0.30 | WDI |
| Life Expectancy Female (Years) | 2905 | 68.65 | 11.55 | 28.36 | 86.44 | WDI |
| Life Expectancy Male (Years) | 2905 | 63.97 | 10.25 | 25.23 | 87.70 | WDI |
| Adult Mortality Rate Female (per 1000 female adults) | 2905 | 174.82 | 123.30 | 35.17 | 708.86 | WDI |
| Adult Mortality Rate Male (per 1000 male adults) | 2905 | 239.96 | 116.87 | 63.41 | 718.92 | WDI |
| Survival to Age 65 Female (% of cohort) | 2870 | 71.85 | 17.42 | 12.50 | 94.02 | WDI |
| Survival to Age 65, Male (% of cohort) | 2870 | 63.24 | 15.85 | 8.57 | 88.72 | WDI |
| Female Health (<i>FH</i>) (Principal Component) | 2870 | 0.339 | 1.64 | -4.25 | 3.99 | AC |
| Male Health (<i>MH</i>) (Principal Component) | 2870 | 0.346 | 1.65 | -4.55 | 4.65 | AC |

Note

WDI = World Development Indicators; AC=Authors' calculation

Table 3. Correlation Among Variables

| | Log capital | Log labour force | Life Expt Female | Life Expt Male | Adult Mortality Female | Adult Mortality Male | Survival to 65, Female | Survival to 65, Male | Female Health | Male Health |
|------------------------------|-------------|------------------|------------------|----------------|------------------------|----------------------|------------------------|----------------------|---------------|-------------|
| Log capital | 1 | | | | | | | | | |
| Log labour force | 0.449 | 1 | | | | | | | | |
| Life Expectancy Female | 0.163 | -0.064 | 1 | | | | | | | |
| Life Expectancy Male | 0.163 | -0.043 | 0.986 | 1 | | | | | | |
| Adult Mortality Rate, Female | -0.154 | 0.046 | -0.952 | -0.940 | 1 | | | | | |
| Adult Mortality Rate, Male | -0.174 | 0.025 | -0.915 | -0.947 | 0.955 | 1 | | | | |
| Survival to Age 65, Female | 0.164 | -0.065 | 0.993 | 0.981 | -0.976 | -0.938 | 1 | | | |
| Survival to Age 65, Male | 0.179 | -0.045 | 0.966 | 0.989 | -0.947 | -0.975 | 0.973 | 1 | | |
| Female Health | 0.200 | -0.072 | 0.981 | 0.975 | -0.939 | -0.924 | 0.981 | 0.972 | 1 | |
| Male Health | 0.186 | -0.060 | 0.945 | 0.975 | -0.910 | -0.955 | 0.946 | 0.988 | 0.971 | 1 |

The correlation matrix of some of the health indicators and input variables are presented in Table 3. It can be seen that indeed the gender disaggregation of the human capital based on health poses some serious problems, because all three measures of the male and female health indicators, viz. life expectancy, adult mortality and survival rate, are highly collinear. In fact, the correlations coefficients between these indicators all exceed 0.90. In order to address the issue of high collinearity in the data we used principal components analysis to group together the variables which are collinear to form a composite index capable of representing this group of variables by itself. Therefore, we grouped female life expectancy, mortality and survival rate in one group and constructed female health (*FH*) as the variable to represent this group

based on the largest principal component¹. Similarly, we constructed the male health (*MH*) variable. However, the collinearity between *FH* and *MH* still remains excessively high as their correlation coefficient is 0.97. This is actually not surprising, because health developments are often shared widely across overall population. Therefore, to avoid the econometric problem in our analysis arising from such a high correlation, we make female health orthogonal to male health by regressing *FH* on *MH* using the mean group estimator and obtain the residuals which represents our measure of female health that is uncorrelated to *MH*. Therefore in our econometric analysis, *FH* represents the largest principal component measure of female health orthogonal to *MH*. A significant coefficient on *FH* will indicate that female health is making a relative contribution (positive or negative) to explaining output compared to male health.

A graphical representation gives us a preliminary idea of the relationship between these variable for the sample period. These are shown in Figures 1 and 2. The scatter plots only show the simple relationship between health and growth without controlling for other factors, which is slightly positive for both female and male health.

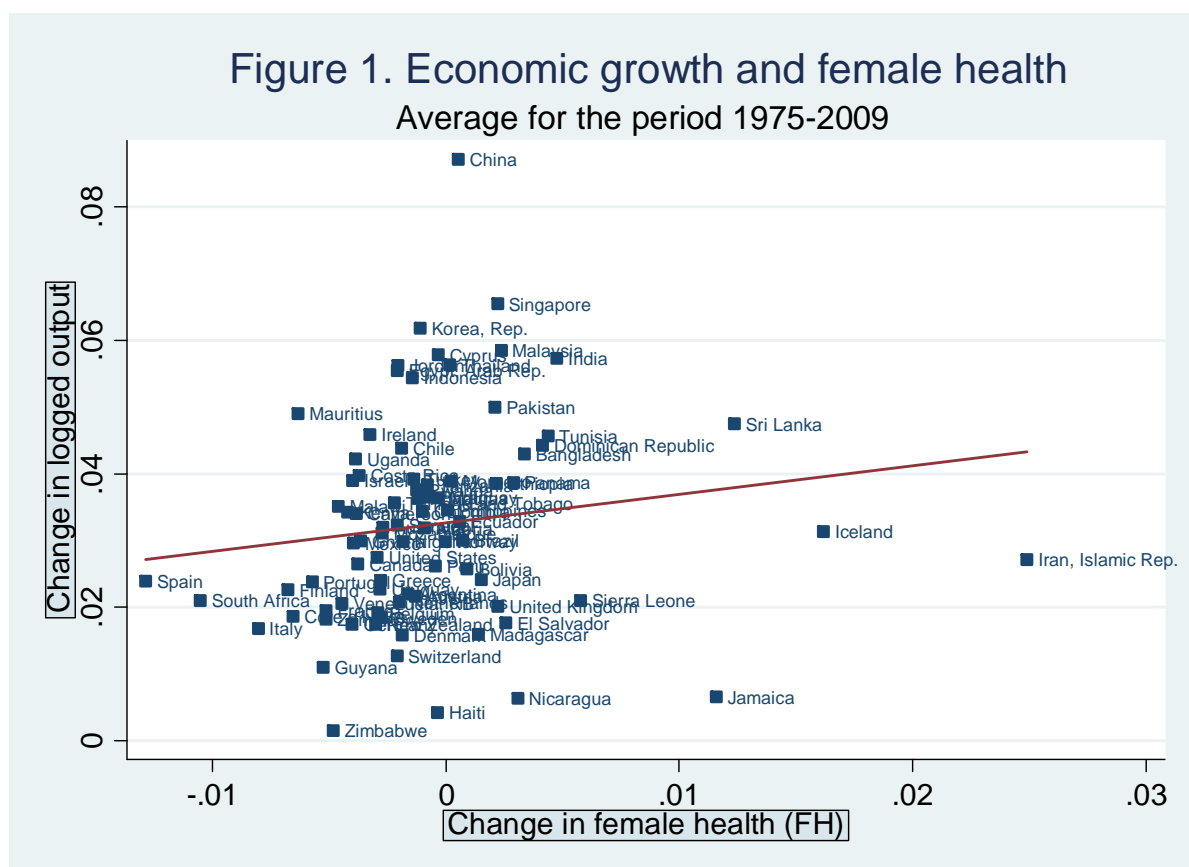
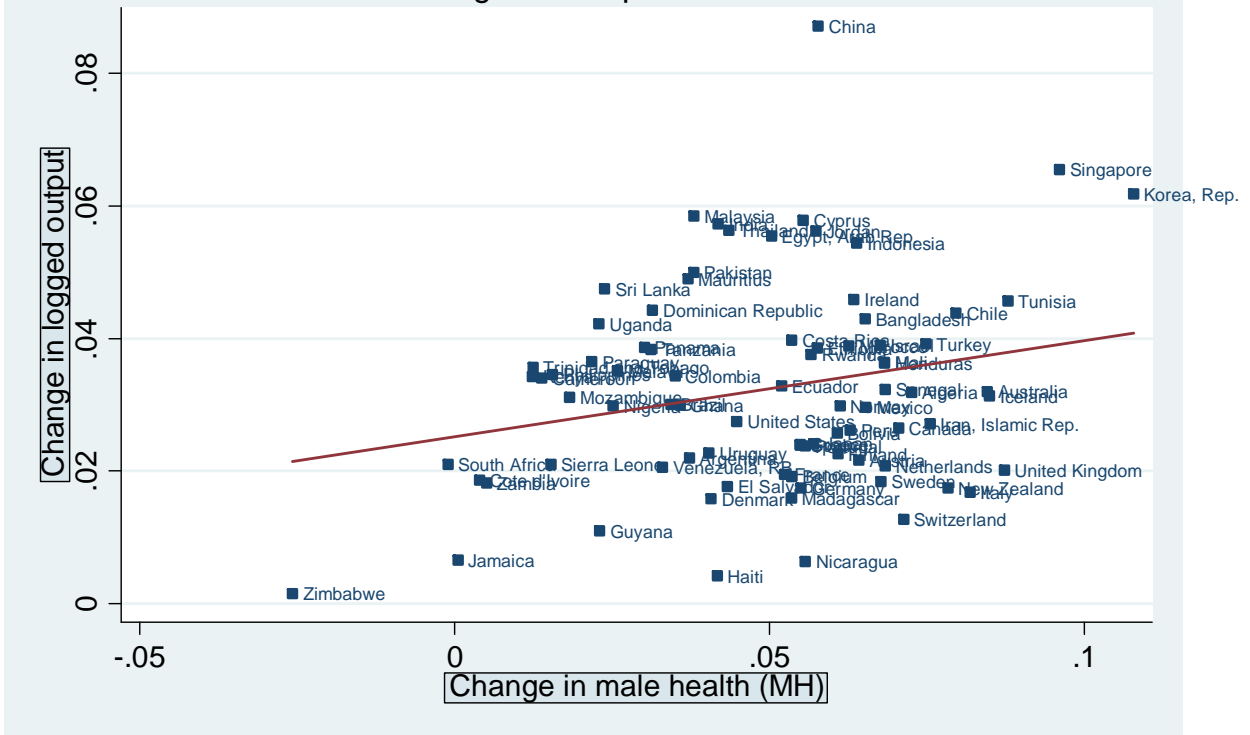


Figure 2. Economic growth and male health
Average for the period 1975-2009



These figures point to some interesting cases. Focusing attention towards the middle of the Figure 1's scatter plot we can see that for roughly similar changes in female health capital during the sample period under consideration, there are various magnitudes of economic growth rates that were achieved. For example, for almost the same unit change to female health, China's growth was 6 percent higher than that of Japan or Sri-Lanka's growth exceeded that of Jamaica's by 4 percent. Similarly looking from Figure 2, we can see that for an equivalent unit change in male health, Bangladesh achieved much faster growth rate compared to Switzerland or Pakistan's growth rate has much exceeded than that of Denmark's.

Such variability in the data is representative of a heterogeneous panel stemming from country specific unobserved factors or common regional as well as global effects. If we take a look at aggregate health status in our sample measured by one of the widely accepted health indicators such as the average life expectancy over the 1975-2009 period for the different regions (see; Table 4), it can be easily seen that life expectancy at birth in Europe and Central Asia is 76 while, in Africa it is 51 years and in South Asia 62 years, reflecting significant regional heterogeneity in life expectancy at birth. Similarly, such heterogeneity can also be observed at the country level (see; Table 5): Sierra Leone's average life expectancy at birth is as low as 39 years, while average life expectancy for both male and female in Bangladesh is 60 years and the same in Japan is 79 years which is the highest.

Table 4. Life Expectancy at Birth by Region, Average 1975-2009

| Region | Years |
|------------------------------|-------|
| East Asia and Pacific | 69 |
| Europe and Central Asia | 76 |
| Latin America and Caribbean | 67 |
| Middle East and North Africa | 69 |
| South Asia | 62 |
| Sub Saharan Africa | 51 |

Source: World Bank 2011.

Table 5. Life Expectancy at Birth by Country, Average 1975-2009

| Country | Years | Country | Years |
|--------------------|-------|---------------------|-------|
| Algeria | 65 | Kenya | 57 |
| Argentina | 72 | Korea, Rep. | 72 |
| Australia | 77 | Madagascar | 54 |
| Austria | 76 | Malawi | 47 |
| Bangladesh | 61 | Malaysia | 71 |
| Belgium | 76 | Mali | 46 |
| Bolivia | 59 | Mauritius | 70 |
| Brazil | 67 | Mexico | 71 |
| Cameroon | 52 | Morocco | 64 |
| Canada | 78 | Mozambique | 45 |
| Chile | 74 | Netherlands | 77 |
| China | 70 | New Zealand | 76 |
| Colombia | 69 | Nicaragua | 65 |
| Costa Rica | 76 | Nigeria | 47 |
| Cote d'Ivoire | 50 | Norway | 78 |
| Cyprus | 77 | Pakistan | 62 |
| Denmark | 76 | Panama | 73 |
| Dominican Republic | 68 | Paraguay | 69 |
| Ecuador | 69 | Peru | 66 |
| Egypt, Arab Rep. | 64 | Philippines | 65 |
| El Salvador | 65 | Portugal | 74 |
| Ethiopia | 49 | Rwanda | 45 |
| Finland | 76 | Senegal | 55 |
| France | 77 | Sierra Leone | 39 |
| Germany | 76 | Singapore | 76 |
| Ghana | 56 | South Africa | 58 |
| Greece | 77 | Spain | 78 |
| Guatemala | 66 | Sri Lanka | 70 |
| Guyana | 63 | Sweden | 78 |
| Haiti | 55 | Switzerland | 78 |
| Honduras | 66 | Tanzania | 51 |
| Iceland | 79 | Thailand | 69 |
| India | 59 | Trinidad and Tobago | 68 |
| Indonesia | 64 | Tunisia | 69 |
| Iran, Islamic Rep. | 64 | Turkey | 65 |
| Ireland | 75 | Uganda | 49 |
| Israel | 77 | United Kingdom | 72 |
| Italy | 77 | United States | 76 |
| Jamaica | 71 | Uruguay | 73 |
| Japan | 79 | Venezuela, RB | 71 |
| Jordan | 70 | Zambia | 46 |
| | | Zimbabwe | 53 |

5. Estimation and Results

Our methodology involves estimating a long-run relationship between log of output, log of factor inputs and gender disaggregated health capital using a panel cointegration estimators based on the variables in the production function outlined in equation (2) as follows:

$$y_{it} = f(k_{it}, l_{it}, FH_{it}, MH_{it}) \quad (7)$$

where $i=1, \dots, N$, refers to the countries in our sample and $t=1, \dots, T$, refers to time over the study period 1975 – 2009. Panel cointegration estimators are robust under cointegration to a variety of estimation problems that often plague empirical work, including omitted variables, endogeneity, and measurement errors (see; Baltagi and Kao, 2000 and Pedroni, 2007). Because output depends on the stock of input variables in the long-run, equation (7) is therefore used as a basis for understanding the long-run behaviour among these variables. Although in theory, the presence of five variables leaves open the possibility that there is more than one panel cointegrating vector present, our prime concern is with the overall long-run relationship between y and MH, FH encapsulated in the single equation.

Our empirical procedure is divided into two parts. The first part includes the panel cointegration analysis which embodies unit root testing, panel tests for joint non-cointegration and analysis of the long-run equation (7). The second part includes panel-type ECM based on the lagged residuals from long-run equation and substituted into the transformed production function in growth form given by equation (6).

Our analysis requires estimating the production function outlined in equation (2) keeping technology unobserved, obliging us to address the issue of potential cross-sectional dependence in our data. Panel-data models are likely to exhibit substantial cross-sectional dependence in the errors, stemming from the presence of common shocks and unobserved components that ultimately become part of the error term, spatial dependence, and idiosyncratic pairwise dependence in the disturbances without showing any particular pattern of common components or spatial dependence (see Pesaran 2004, Baltagi 2005 and Chudik *et al.* 2011). A reason that could be causing such dependence is the increasing order of the global integration leading to strong interdependencies among the cross-sectional units. If the unobserved components that create interdependencies across cross-sectional units are correlated with the included regressors, fixed effects and random effects estimators will be biased and inconsistent (see; Pesaran 2006); even dynamic panel data models, such as those by Anderson and Hsiao (1981), Arellano and Bond (1991), and Blundell and Bond (1998) are inconsistent as the cross-sectional dimension (N) grows large, for a fixed panel's time dimension (T) (see; Sarafidis and Robertson 2006).

To test for the presence of cross-sectional dependence in our data, we implement the Pesaran (2004) CD-test that employs the correlation-coefficients between the time series for each panel member, on all five variables in equation (7) as well as to the residual from the estimated production function using the FE estimator. The results are presented in Table 6. Under the null of cross-sectional independence, the CD statistic is distributed as standard normal, which leads us to the rejection of null for all of these variables. As a result, we conclude that both the dependent and explanatory variables, as well as the residuals, have cross-sectional dependence in our model. As a consequence, we shall require the use of appropriate estimators to carry our empirical investigation which will be discussed later.

Table 6. Pesaran (2004) Cross-Section Dependence (CD) Test

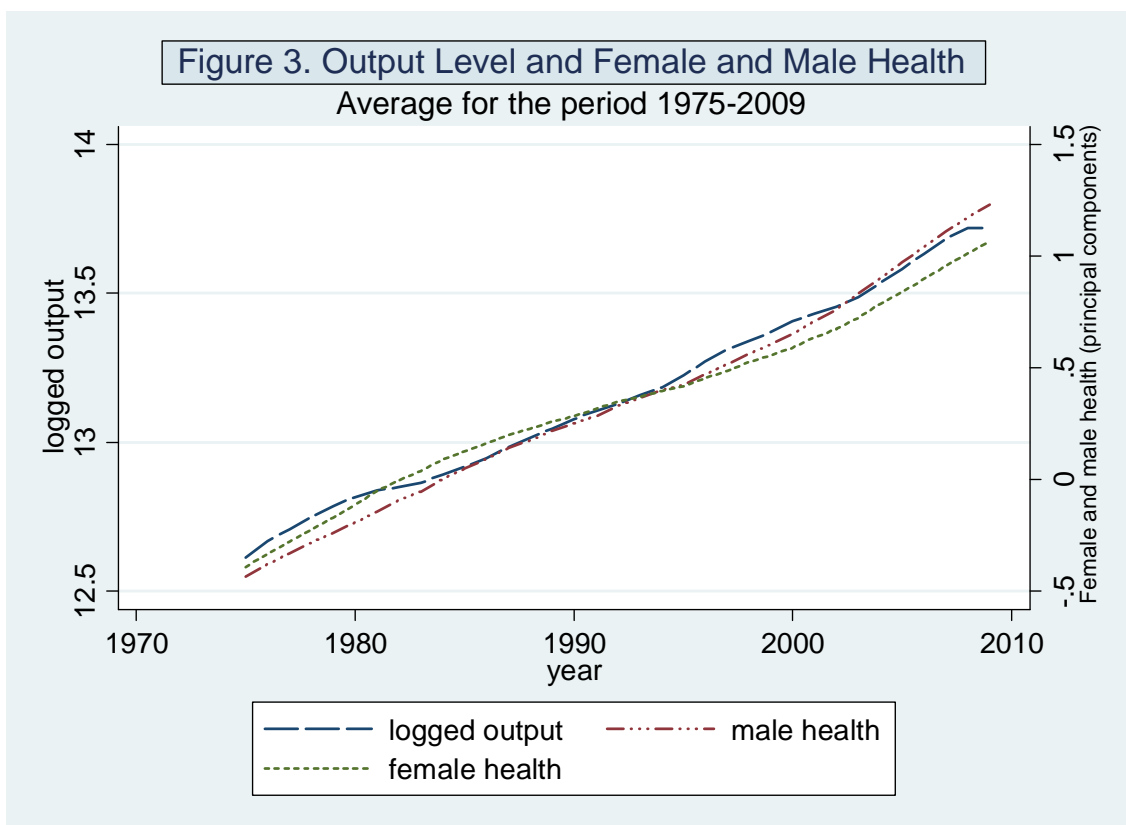
| Variable | CD-test | p-value | correlation | Decision |
|-----------------|---------|---------|-------------|----------|
| <i>y</i> | 294.16 | 0.00 | 0.86 | CSD |
| <i>k</i> | 284.47 | 0.00 | 0.83 | CSD |
| <i>l</i> | 312.75 | 0.00 | 0.92 | CSD |
| <i>FH</i> | 46.31 | 0.00 | 0.14 | CSD |
| <i>MH</i> | 221.60 | 0.00 | 0.65 | CSD |
| <i>Residual</i> | 16.64 | 0.00 | 0.05 | CSD |

Notes

Under the null hypothesis of cross-section independence $CD \sim N(0,1)$

Figure 3 shows the plot of the average logged level of output and average female and male health based on the largest principal components across all the countries from 1975-2009. As is expected from long time series data, the three series trend upwards. All three series show a stable increase over time, showing a particular pattern of rising more rapidly at the beginning and at the end of the sample. Such exploratory data analysis suggest that level of output and health stocks may be non-stationarity, which is further tested.

Given the low power of country by country tests, we are motivated to conduct panel unit root tests on our data. In the presence of cross-section dependence, “first generation” panel unit root tests tend to reject the null hypothesis of a unit root excessively. Therefore we apply the CIPS test suggested by Pesaran (2007). Table 7 reports the CIPS panel unit root tests on our variables which are outlined in equation (7). The CIPS test allows for heterogeneity in the autoregressive coefficient of the Dickey-Fuller regression and allows for the presence of a single unobserved common factor with heterogeneous factor loadings in the data. The statistic is constructed from the results of panel-member-specific ADF regressions where cross-section averages of the dependent and independent variables (including the lagged differences to account for serial correlation) are included in the model (referred to as CADF regressions). Under the null of joint non-stationarity the test statistic has a non-standard distribution.



The output in Table 7 provides evidence of non-stationarity of all the five variables, both when an intercept only is included in the specification and when an intercept and a trend are included². The results are obtained including 3 lags in the ADF regressions³. Non-stationarity of the female and male health stock are hardly surprising as they can be easily justified by the declining mortality pattern for the elderly. For example, according to the UN-World Bank Population database, life expectancy on average for men at age 65 is likely to be 18.1 years in 2040 in the OECD countries (see also Hendricks and Graves, 2009). The level of integration in the variables is uniform because all variables are stationary in first-difference form, but do not reject the null hypothesis in levels. This indicates all panel units are I(1) and are appropriate for panel cointegration analysis.

Before we can estimate the production given by equation (7) with unobserved TFP, we first need to ensure that the variables involved are cointegrated. The length of the time series in the dataset allows an econometric model to estimate the long-run relationship that exists between the variables (cointegration), and the width (number of countries) enriches the information that is available to the model to estimate the relationship.

² The null of non-stationarity is rejected at 5 percent level of significance for FH, but when a trend is added to the constant in the ADF regression, the null cannot be rejected. For all five variables our preferred CIPS results are the ones which include both a constant and trend.

³ Some robustness checks show that the results reported do not change when varying the number of lags included in the ADF regression.

Table 7. Pesaran (2007) CIPS Panel Unit Root Tests

| Variable | Intercept only | Intercept & trend | Variable | Intercept only |
|----------|----------------|-------------------|-------------|----------------|
| y | -0.262 | -0.498 | Δy | -6.296*** |
| k | 2.500 | 3.908 | Δk | -1.589* |
| l | 3.241 | 8.700 | Δl | -4.446*** |
| FH | -1.944** | 2.014 | ΔFH | -3.499*** |
| MH | -0.132 | 1.084 | ΔMH | -1.425* |

Notes

The Pesaran (2007) CIPS test is a cross-section augmented unit root test which allows for heterogeneity in the autoregressive coefficient of the Dickey-Fuller regression and allows for the presence of a single unobserved common factor with heterogeneous factor loadings in the data. The averaging of the group-specific results follows the procedure in the Im, Pesaran and Shin (2003) test. Under the null of nonstationarity the test statistic has a non-standard distribution. ***, ** & *: Significant at 1, 5 & 10 percent level respectively.

We test for co-integration among the I(1) variables using the test developed by Pedroni (1997, 1999, 2004). The procedure for computing the test statistics for panel data non-cointegration involves estimating the hypothesized cointegration regression described in equation (7) and using the residuals to estimate the appropriate autoregression. Pedroni provides seven test statistics that can be used to test the null of no cointegration in the multivariate case. These test statistics are grouped into two categories: ‘group mean’ statistics and ‘panel’ statistics. The ‘group mean’ statistics are referred to as *between-dimension* statistics that average the estimated autoregressive coefficients for each country and are called *Group PP* which is a non-parametric and analogous to the Phillips-Perron t statistic and *Group ADF* which is a parametric statistic and analogous to the ADF t statistic⁴. The ‘panel’ statistics are referred to as *within-dimension* statistics and are called *Panel ν* , *Panel ρ* , *Panel t* and *Panel ADF*, that effectively pool the autoregressive coefficients across different countries during the unit root tests. In these tests, a common value for the autoregressive coefficient is specified under the alternative hypothesis of cointegration.

Under the alternative hypothesis of cointegration, the autoregressive coefficient is allowed to vary across countries. This allows one to model an additional source of potential heterogeneity across countries. Following an appropriate standardization, both of these statistics tend to a standard normal distribution as $N, T \rightarrow \infty$ diverging to negative infinity under the alternative hypothesis and consequently, the left tail of the normal distribution is used to reject the null hypothesis of non-cointegration (Pedroni (1999, p.668)). Baltagi (2005, p.255) provides the formal interpretation of a rejection of the null: ‘Rejection of the null hypothesis means that enough of the individual cross-sections have statistics “far away” from the means predicted by theory were they to be generated under the null’.

⁴ This latter statistic is analogous to the Im, Pesaran and Shin (2003) test for a panel unit root applied to the estimated residuals of a cointegrating regression.

In practice, the different test statistics can give contradictory results, but as reported in Pedroni (2004), the *Group ADF* and *Panel ADF* statistics have the best power properties when $T < 100$, whereas the *Panel ν* and *Group ρ* statistic perform comparatively worse. Table 8 presents the results of Pedroni (1999, 2004) panel cointegration test based on *Group ADF* and *Panel ADF* statistics for the full model between each y , k , l , FM and MH . As a procedure to correct for the cross sectional dependency in our panel data that could have been generated via global health technological shocks, we have subtracted out the common time effects from the five variables in our model using the time-demeaning method prescribed in Pedroni (1997, 2004). Our results strongly advocate that a long-run cointegrating relationship exists between y , k , l , FM and MH , both when only a constant is included and a constant and a trend is included as deterministic regressors, because the null of non-cointegration is strongly rejected at the 1% significance level.

Table 8. Panel Data Cointegration Tests

| A: Pedroni (1999, 2004) Panel Cointegration Test | | |
|--|----------------|---------------------|
| | Intercept only | Intercept and trend |
| Panel ADF | -3.736*** | -5.927*** |
| Group ADF | -7.067*** | -9.195*** |
| B: Westerlund (2007) ECM panel cointegration test | | |
| | Intercept only | Intercept and trend |
| Gt | -3.053*** | -3.657*** |
| Pt | -20.134*** | -24.050** |
| C: Kao (1999) Panel Cointegration Test | | |
| | Intercept only | |
| Kao-ADF | -6.729*** | |

Notes

These are the Pedroni tests for panel cointegration (discussed in Pedroni 1999, 2004) between each y , k , l , FM and MH . *Panel* and *Group ADF* is a parametric statistic and analogous to the ADF t statistic. These estimates include common time dummies. Individual lag lengths are based on the Schwarz's information criterion. These statistics tend to a standard normal distribution as $N, T \rightarrow \infty$. ***, ** and * denote rejection of the null of non-cointegration at the 1, 5 and 10% significance levels critical values of -2.33, -1.64 and -1.28 respectively.

Westerlund (2007) is ECM based panel cointegration test between each y , k , FM and MH . The *Gt* test statistic is based on a weighted average of the individually estimated short-run coefficients and their t -ratio's. The *Pt* test statistics pool information over all the cross-sectional units to test the null of non-cointegration for all cross-section entity. Individual lag and lead lengths are based on the Schwarz's information criterion. ***, ** and * denote rejection of the null of non-cointegration at the 1, 5 and 10% significance. Kao (1999) test, which is based on the Engle-Granger two-step procedure, and imposes homogeneity on the members in the panel. The null hypothesis of no cointegration is tested using an ADF-type test. Individual lag lengths are based on the Schwarz's information criterion, and ***, ** and * denote rejection of the null of non-cointegration at the 1, 5 and 10% significance levels.

In addition to this, we also check for cointegration between y and k , FM , MH by implementing the four panel cointegration tests developed by Westerlund (2007) which is based on structural rather than residual dynamics and, therefore, do not impose any common-factor restriction. The underlying idea is to test for the absence of cointegration by determining whether the individual panel members are error correcting. Westerlund develops four tests for cointegration that are very flexible and allow for an almost completely heterogeneous specification that are all normally distributed and are general enough to accommodate unit-specific short-run dynamics, unit-specific trend and slope parameters, and cross-sectional dependence⁵. Our results found that two of Westerlund (2007) statistics was rejecting the null of no error-correcting relationship which is reported in the panel B of Table 8. Finally, we also report the Kao (1999) test for panel cointegration. The Kao test is residual based which runs a static fixed-effects model on variables assumed cointegrated and then a pooled ADF regression, analogous to the Engle-Granger procedure in time-series, is applied to the residual. Under a rather restrictive assumption of a common cointegrating vector and common dynamics, the null hypothesis of non-cointegration is rejected if the residuals are $I(0)$. We also report the Kao (1999) residual cointegration test in panel C of Table 8. It can be seen that the joint null of non-cointegration is strongly rejected.

Having established that the variables in our model given by equation (7) are cointegrated, we can now proceed to estimate the production function in equation (2) in level which gives us long-run estimates of the growth effects of female and male health stocks. The problems associated with the model and within the data will decide which the most appropriate estimator is. The main concerns that we want to address are, the endogeneity of the female and male health variables, cross-sectional dependence and potential heterogeneity in the data and parameter non-linearity of the health variables in the production function outlined in equation (2). Given these concerns, we shall use three estimators that are capable of estimating the long-run relationship among the panel variables correcting for one or more of the problems but not all. They are Panel Dynamic OLS (PDOLS) (Pedroni, 2001), Common Correlated Effects Mean Group (CCEMG) (Pesaran, 2006) and Pooled Mean Group (PMG) (Pesaran *et al.* 1999).

The standard fixed-effects panel OLS regressions are not entirely appropriate for a panel that is long T and small to medium N due to the presence of a second-order bias (leading to a size distortion) in the case of endogenous variables. This bias is not necessarily eliminated when N grows large. Also a key assumption of this estimator is that all parameters are homogenous for all countries, which is restrictive. In reality, the existence of a possible heterogeneity in the relationship between health and income across cross-section units creates a further econometric issue. For example, countries in our sample differ widely in terms of size, income level, economic structure, government policies, and other characteristics. It

⁵ The Ga and Gt test statistics are based on a weighted average of the individually estimated short-run coefficients and their t -ratio's, respectively. The Pa and Pt test statistics pool information over all the cross-sectional units to test the null of no-cointegration for all cross-section entity.

would therefore be inappropriate to pool observations across cross-sectional units and thus to assume that the slope coefficients, or effects, of female and male health are the same for all countries. For this reason, we prefer a heterogeneous (between-dimension) estimators based on the group mean approach, which accounts for heterogeneous panels by estimating separate OLS regressions to obtain the individual slope estimates for each cross-sectional units. In addition, under cointegration, heterogeneous panel cointegration estimators are robust to a variety of estimation problems that often plague empirical work, including omitted variables, slope heterogeneity, and endogenous regressors (Pedroni, 2007).

The PDOLS estimator is a panel extension of the single time series Dynamic OLS (DOLS) estimator that was proposed by Stock and Watson (1993). Under DOLS, lags and leads of the differences of the explanatory variables are added to a regression which account for possible serial correlation and endogeneity of the regressors. Thus, one important feature of DOLS estimator is that it is asymptotically unbiased and normally distributed even in the presence of endogenous regressors. Consequently, in contrast to cross-section and conventional panel approaches, the PDOLS does not require exogeneity assumptions nor does it require the use of instruments. In addition, the group-mean PDOLS estimator is super-consistent under cointegration, and is robust to the omission of variables that do not form part of the cointegrating relationship. To account for certain forms of cross-sectional dependence, the PDOLS procedure allows time-demeaning the data.

Relaxing the parameter homogeneity assumption, we can also find other heterogeneous (between-dimension) estimators based on the mean group approach, which estimate individual country coefficients separately and then report average country coefficients. The mean Group (MG) estimators of Pesaran and Smith (1995) were the first to allow complete parameter heterogeneity across cross-sections. However, our data shows considerable cross-sectional dependence due to common business cycle and other common factors, such as institutional, political, social or health technology shocks, which affect all cross-sectional units in the panel. The CCEMG estimator, which is also a Mean Group type, allows for cross-sectional dependencies that potentially arise from multiple unobserved common factors and permits the individual responses to these factors to differ across cross-sectional units (Pesaran, 2006). Another advantage of the CCEMG approach is that it yields consistent estimates even when the regressors are correlated with the common factors. In addition, Kapetanios *et al.* (2009) have shown that the CCEMG estimator is consistent regardless of whether the common factors are stationary or non-stationary.

One drawback of CCEMG estimator relative to PDOLS is that it is intended for the case in which the regressors are exogenous. Also, it does not take into account that some economic conditions tend to be common across countries in the long-run. The efficiency gains of assuming common long-run relationships while at the same time allowing for heterogeneous short-run dynamics are captured by the Pooled Mean Group (PMG) estimator (Pesaran *et al.* 1999). PMG uses a maximum likelihood procedure which is suitable for

estimating coefficients that are non-linear in parameters. The Newton–Raphson algorithm is used to estimate the long-run coefficient values through an iterative procedure that is constantly updated with information provided by the short-run regressions until convergence is achieved. The long-run parameters are hence non-linear functions of the short-run parameters. PMG estimators are consistent and efficient even in the presence of endogenous as well as non-stationary regressors (Pesaran *et al.* 1999).

We now turn to the estimation of our production function keeping technology unobserved as outlined in equation (7). The results to be discussed are, first, the relationship between level of output and health capital stock while controlling for level of factor inputs in the long-run, and second, the relationship between growth of output and change in health capital stock while controlling for growth of input in the short-run as outlined in equation (6). As discussed previously, we shall use three different estimators to ensure there is consistency in the results.

Table 9. Estimation of Long-Run Relationship for the Variables in Equation (7)

| Variables (Coefficients) | Dependent Variable: y | | |
|-----------------------------|-------------------------|--------------------|---------------------|
| | PDOLS | CCEMG | PMG |
| $k (\alpha)$ | 0.551 (23.41)*** | 0.291 (2.95)*** | 0.323 (15.65)*** |
| $l (\beta)$ | 0.293 (3.54)*** | 0.504 (2.87)*** | 0.596 (19.48)*** |
| $FH (\phi_1)$ | 0.138 (2.67)*** | 0.146 (1.70)* | 0.335 (16.36)*** |
| $MH (\phi_2)$ | 0.071 (3.46)*** | 0.113 (2.39)** | 0.156 (14.36)*** |
| $H_0: \phi_1 = \phi_2$ | 0.192 | 0.742 | 0.633 |
| Prob > Chi-square | | | |
| Observations | 2617 | 2870 | 2788 |
| Number of Countries | 82 | 82 | 82 |

Notes

***, **, * indicate significance at the 1 per cent, 5 per cent and 10 per cent level respectively. t statistics are in brackets. Time dummies have been added to PDOLS, and lags and leads in the PDOLS estimation have been chosen according to Schwarz's information criterion. PMG estimation includes an error correction term which is reported in Table 10.

Table 9 reports the results based the long-run co-integrating vector which strongly suggest that after controlling for the contribution of factor inputs, the female and male health stock have a significant positive effect on the level of output in the long-run and the result thus obtained is consistent across all three estimators. In column (1) the PDOLS results are reported first. The estimated coefficient of FH is 0.14 which is significant at 1 percent level, and the coefficient of MH is 0.07, also significant at 1 percent level. We also test the hypothesis whether the coefficients of female and male health are equal. The P-value of the Chi-square test statistic is found to be 0.19, lending no support to reject the hypothesis. The share of physical capital (k) and labour (l) are different from the stylized value of one-third and two-thirds, respectively (Mankiw et al, 1992). In particular, the share of capital seems

overestimated and that of labour underestimated from the stylized value. An explanation for this could be that developing countries have higher marginal productivity of capital and thus higher share of capital than developed countries and our sample includes both developed and developing countries.⁶ The share of labour similarly could be lower than the stylized value because of the inclusion of labour in a raw as well as in a human capital intense form through health.

In column (2) we report on the estimation of equation (7) by the CCEMG estimator. The estimated coefficient of *FH* is 0.15 and significant at 10 percent, which is very close to that of PDOLS the counterpart. The estimated coefficient of *MH* is 0.11 and significant at 5 percent level, but its magnitude is slightly higher than the one estimated in the PDOLS estimator. However, the null hypothesis that the male and female health coefficients are equal cannot be rejected. The share of capital and labour are closer to their stylised values than in the PDOLS estimations.

Since the worldwide frontier technology is same for each countries in the sample, it is a reasonable assumption that the long-run the steady state level of TFP is also identical for all countries. We account for this by using the PMG estimator which constraints the long-run parameters to be same across all countries. The results are reported in column (3) where we find that the estimated coefficients on *FH* and *MH* are 0.34 and 0.16 respectively and both are significant at 1 percent level. A test of the hypothesis that female and female coefficients are equal cannot be rejected. Although the results are qualitatively same, the magnitude of estimation via the PMG is higher than both PDOLS and CCEMG. An explanation for this could be that, since it is assumed every country has reached the frontier technology in the long-run, countries across the sample have now access to best and identical state of medical technology, thus boosting the marginal effects of health capital.

Results reported in Table 9 strongly suggest that after controlling for the contribution of factor inputs, the female and male health stock have a significant positive effect on the level of output in the long-run. Specifically, given that our model is log-linear in health variables, a one unit increase in female health indicator (*FH*) can induce a rise of 14 – 33 percent in level of long-run output; similarly a one unit increase in male health indicator (*MH*) can lead to an increase of 7 – 16 percent in level of long-run output. However, the hypothesis that female

⁶ By definition the share of profits is:

$$\alpha = \frac{\frac{\partial \ln(Y)}{\partial \ln(K)} \times K}{Y} \approx \frac{\Delta Y}{\Delta K} \left(\frac{K}{Y} \right)$$

The numerator is the remuneration for capital which is the marginal product of capital (*MPK*) multiplied by capital stock and (*K/Y*) is the capital-output ratio (*KYRAT*). It is to be expected that *MPK* will be higher in developing countries because of their lower capital stocks and α higher. This effect will be partly offset by lower *KYRATs* in the developing countries. But in proportionate terms the differences in *MPKs* are likely to be higher than *KYRATs*.

health contributes more to the level of long-run output than male health, cannot be supported through our data even though it is possible to lend support to such general equilibrium effects of female health theoretically.

Table 10. Estimation of Short-Run Relationship for the Variables in Equation (6)

| Variables (Coefficients) | Dependent Variable: Δy | | |
|---|--------------------------------|-----------------------|----------------------|
| | FE-IV | CCEMG | PMG |
| $\Delta k (\alpha)$ | 0.473 (2.64)*** | 0.253 (2.58)*** | 0.724 (5.91)*** |
| $\Delta l (\beta)$ | 1.017 (4.65)*** | 0.326 (2.23)** | -0.217 (-0.58) |
| $\Delta FH (\phi_1)$ | -1.325 (-2.15)** | -0.055 (-0.52) | -0.655 (-1.73)* |
| $\Delta MH (\phi_2)$ | -0.712 (-2.32)** | -0.101 (-2.17)** | -0.381 (-1.89)* |
| EC Speed of Adjustment ($\rho-1$) | -0.745 (-5.87)*** | -0.392 (-10.43)*** | -0.247 (-8.44)*** |
| $H_0: \phi_1 = \phi_2$ Prob > Chi-square | 0.218 | 0.506 | 0.744 |
| Observations | 809 | 2706 | 2788 |
| Number of Countries | 72 | 82 | 82 |
| Sargan test (p-value) | 0.273 | | |

Note

***, **, *indicate significance at the 1 per cent, 5 per cent and 10 per cent level respectively. t statistics are in brackets. For FE-IV lags of male and female primary, secondary and tertiary enrolments are used.

Having established the existence of cointegrating long-run relationship, we now turn to estimation of the short-run error correction model (ECM) represented by equation (6). The coefficient $(\rho - 1)$ measures speed of adjustment of the error correction (EC) term as in the adjustment of y to a deviation from long-run equilibrium relation between the dependent variable and the regressors. The term in the parenthesis multiplied to the speed of adjustment coefficient of EC is the previous period cointegrating relation which we obtain from the residual in the CCEMG estimator in column (2) of Table 9.

For estimation of the short-run relationship, we adopt the CCEMG and PMG estimators as before, but as a consistency check we start by estimating with a FE-IV estimator where the instruments for female and male health are given by lags of male and female primary, secondary and tertiary enrolments. Results are reported in Table 10. The FE-IV results can be seen in column (1) of Table 10. The female and male health variables are negative and significant at the 5 percent level, suggesting that short-run adjustments in health inputs have a negative impact on output growth. But the difference in the short-run female and male coefficients is not statistically significant. The speed of adjustment term is negative and significant at the 1 percent level, suggesting that 75 percent of the disequilibrium is corrected

within a year. In column (2), the CCEMG results are presented. The coefficient on female health is insignificant and that of male is negative and significant at the 5 percent level, but the hypothesis that they are equal cannot be rejected. The speed of adjustment term is -0.39 and significant at 1 percent level, suggesting around 40 percent of the deviation from the long run is corrected every year. Finally the PMG results are reported in column (3). The conclusion does not alter much, the female and male health variables are negative and significant at 10 percent level, but their impacts on growth are not unequal and 25 percent of the error is corrected each year, suggesting that the half-life of a deviation from a long-run relation is just over two years.

The conclusion from our short-run analysis is that there is difference in the long-run and short-run behaviour of the impact of health capital on output. Whereas the effect of female and male health stock on level of output is significantly positive in the long-run, the effect of changes in female and male health on growth of output in the short-run is negative or insignificant. Our explanation for this is as follows. Changes in the health capital stock, both female and male, can take place at a faster pace in the short-run. This is due to medical advances or proliferations of vaccines or diffusion in medical technologies such as percutaneous coronary interventions to treat coronary artery disease or availability of pharmaceuticals such as statins dispensed for prevention, proving to be effective in reabsorbing atherosclerotic plaques and hence reducing the need for angioplasty.

In response to these short-run changes in health conditions, the economy must adjust from a particular steady state to another, which takes time. For example, for improved health conditions to lead to higher growth, it must come through enhanced labour force participation by both females and males. But that may not happen in the short-run because such participation may depend on the fertility rate to decline thereby enabling youth dependency ratio to fall so that the parents can join labour market. Therefore, as it may be relatively less time involving to catch up with frontier health technology and improve the health conditions in the short-run, achieving a decline in fertility is only possible in the long-run. As the required full adjustment for the economy from one steady state to another due to a short term health technology shock may only take place in the long-run, the short-run relationship between health and growth can be negative or insignificant.

6. Conclusions

It is widely believed by development economists that the role of human capital is one of the most fundamental determinants of economic growth. Sustained growth depends on the level of human capital whose stocks increase due to better education, higher levels of health, learning and on-the-job-training. The intuition that good health raises the level of human capital and has a positive effect on productivity and economic growth has been modelled by endogenous growth theorists. But empirically ascertaining the causal relationship between health and growth has proved to be difficult on account of the possible existence of

endogeneity between these two variables. Using an approach based on panel cointegration and error correction, our paper models the differences across countries where the growth in output is a function of traditional factor inputs, health conditions, technological progress. Our key contribution to the health-growth debate is as follows. First, there is a significantly positive long-run impact from both female and male health capital stocks on the level of output after controlling for traditional factor inputs and also considering the endogeneity in the regressors, cross-sectional dependence in the residual and parameter heterogeneity. However, it is not possible to confirm if the gender differences in the health impacts are statistically variable, in other words we find no evidence to conclude that growth impact of female health is higher than that of male although women's health may be associated with some additional benefits through general equilibrium effects, but this is not captured in our model.

Secondly, in an attempt to diminish the high degree of collinearity between gender disaggregated health capital, we have made our measure of female health orthogonal to male health so that the derived female health variable has had the common health factor with male health stripped out. Whilst this is certainly arguable that female health variable only picks up idiosyncratic health measurement specific to females, the fact that female health is significantly different from zero implies that it certainly matters when compared to male health. We find that our measure of female and male health are non-stationary along with output and factor input variables, enabling a cointegration analysis.

Thirdly, we use panel time-series methodology, panel cointegration and error correction modelling to distinguish the long- and short-run health effects for both genders, that is, if they vary or remain same. Our short-run analysis confirm that the gender-disaggregated health capital has a negative growth effect for both females and males but in the long-run there is a positive level effect. Intuitively, changes in health capital stock can take place relatively faster, but time required for the economy to adjust from a particular steady state to another may take place in the long-run.

Finally, in terms of policy our paper has some implications. Our estimates indicate that a unit change in combined female and male health capital stock could account for at the least 20 percent of the rise in the level of income in the long-run that is over the 36 years in our sample period. Such contribution is equivalent to 0.64 percentage point of the 3.2 percent average growth of output in our whole sample. Therefore, the health sector is one of the major sources of growth and governments should allocate more budgets for expansion of this sector as well as to improve its efficiency. Also, based on our model, we do not advocate at this stage that the female health sector needs a substantially higher resource allocation than the male counterparts, although we recognise that understanding of the proper impact of female health on the economy may require a more complicated general equilibrium analysis.

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Data Appendix

| Variable | Source |
|---|---|
| Per capita income (constant 2000 US\$) (Y) | World Development Indicators 2011 |
| Capital stock dollars (K) | Bosworth and Collins (2003) from 2004 to 2009 World Development Indicators 2010 using perpetual inventory method. |
| Labour force number (L) | Bosworth and Collins (2003) from 2004 to 2009 World Development Indicators 2010. |
| Life Expectancy, Female (Years) | World Development Indicators 2011 |
| Life Expectancy, Male (Years) | World Development Indicators 2011 |
| Adult Mortality Rate, Female (per 1000 female adults) | World Development Indicators 2011 |
| Adult Mortality Rate, Male (per 1000 male adults) | World Development Indicators 2011 |
| Survival to Age 65, Female (% of cohort) | World Development Indicators 2011 |
| Survival to Age 65, Male (% of cohort) | World Development Indicators 2011 |
| Female Health (<i>FH</i>) (Principal Component) | Computed by authors |
| Male Health (<i>MH</i>) (Principal Component) | Computed by authors |