

**From Physical to Human Capital Accumulation:
Effects of Mortality Changes**

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From Physical to Human Capital Accumulation: Effects of Mortality Changes

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Abstract

This paper develops a growth model à la Galor and Moav (Review of Economic Studies 71(4):1001–1026, 2004) that captures the replacement of physical capital accumulation by human capital accumulation as the prime engine of growth. We show that (i) decreased mortality promotes this replacement, and (ii) the effect of a decrease in mortality on per-capita income differs across the phases of the development process. A notable prediction of our theory is that the higher the level of education in an economy, the more likely it is that a decrease in mortality will increase income per capita. Using finite mixture models, we show that this prediction is supported by the data.

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1 Introduction

Does a decrease in mortality raise income per capita? An influential paper by Acemoglu and Johnson (2007) provoked a controversy over this question. In contrast to studies based on cross-country variation (e.g., Lorentzen et al., 2008), Acemoglu and Johnson (2007) use instruments for mortality changes to solve the endogeneity problem, and obtain a surprising result: improved life expectancy has a positive effect on population growth, but a negative effect on GDP per capita. This result is somewhat shocking because it implies a trade-off between health improvement and economic growth. Theoretically, their result is justified by the dilution effect in traditional growth theories: population growth decreases land per capita (the Malthus channel) and physical capital per capita (the Solow channel), thus reducing income per capita. On the other hand, recent growth literature suggests the increasing importance of human capital over land and physical capital as an engine of growth, particularly in developed countries (e.g., Jones and Romer, 2010). Here, we develop a growth model in which the replacement of physical capital accumulation by human capital accumulation is endogenous and mortality changes affect the replacement process. Furthermore, we theoretically and empirically investigate how a decrease in mortality affects income per capita.

We extend the model of Galor and Moav (2004) that captures the replacement of physical capital accumulation by human capital accumulation as a prime engine of growth to consider the effect of mortality changes on the development process. Our model derives two key results: (i) decreased mortality promotes this replacement, and (ii) the effect of a decrease in mortality on per-capita income differs across the phases of the development process. As in Galor and Moav (2004), we shed light on the portfolio choice between physical and human capital. However, our focus differs from theirs. We focus on an asymmetry between physical and human capital: human capital is inherently embodied in people and thus human capital yields a return only when the investor survives, whereas physical capital returns belong to a spouse and offspring even if the investor dies. This asymmetry implies that in environments with higher mortality, it might

be riskier for households to concentrate their assets in human capital. A decrease in mortality stimulates the incentive to invest in human capital and promotes the replacement of physical capital accumulation with human capital accumulation.¹

Many studies already establish the theoretical result that a decrease in mortality promotes human capital investment (e.g., de la Croix and Licandro, 1999; Kalemli-Ozcan et al., 2000; Boucekkine et al., 2002, 2003; Soares, 2005; Cervellati and Sunde, 2005).² The difference from prior studies is that we shed light on the cooperative behaviour among family members in asset formation, rather than the behaviour of a single agent.³ Most prior studies focus on a property of human capital that human capital continues to yield a return as long as agents are able-bodied workers once they form human capital, while physical capital investment yields a one-time-only return. Thus, a higher mortality shortens the working life over which investments in human capital pay off, thereby negatively affecting human capital investment. In contrast, we focus on the property that human capital cannot be carried over to surviving family members if the investor dies because human capital is embodied in the original investor. On the other hand, physical capital can be left to surviving family members. Thus, a higher mortality induces agents to substitute physical capital for human capital in case of death.⁴

In our model, an economy switches from a regime where only physical capital is accumu-

¹Galor and Moav (2004) focus on a different asymmetry between physical and human capital: in contrast to physical capital, human capital is inherently embodied in people, and human capital investment exhibits diminishing returns at the individual level. Based on this asymmetry, they show that income inequality, through capital-market imperfection, affects economic growth differently in the various stages of the development process.

²Jayachandran and Lleras-Muney (2009) empirically show that longer life expectancy increases educational attainment.

³It is generally agreed that the family institution serves as an insurance device for individuals (e.g., Weiss, 1997). For example, Kotlikoff and Spivak (1981) find that consumption and bequest-sharing arrangements in marriages and larger families can significantly substitute for complete and fair annuity markets, suggesting the importance of cooperation among family members in asset formation. An advantage of our approach focusing on family cooperation is that it does not contradict a finding of Hazan (2009). Hazan (2009) finds that the expected total hours worked over a lifetime for males has decreased because of a decrease in hours worked per year and early retirement, insisting that an increase in the expected length of working life should not be responsible for increasing education. On the other hand, the total lifetime hours worked for married couples has not decreased owing to increased female labour force participation (Ramey and Francis, 2009).

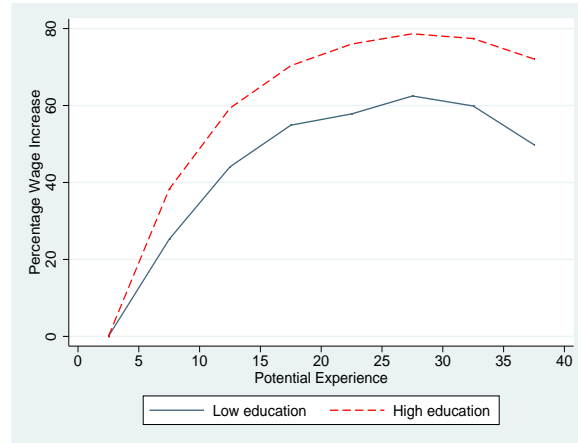
⁴Our mechanism might be justified in terms of family member altruism (e.g., a spouse, siblings, or offspring). The presence of altruism has been shown by many studies and is assumed in a large number of growth theories (e.g., Loury, 1981; Galor and Zeira, 1993).

lated to a regime where both physical and human capital are accumulated during the development process. In the former regime, a decrease in mortality depresses income per capita by the dilution effect on physical capital, especially in the short run. In the latter regime, however, two additional effects improve the average amount of human capital in the labour market. First, a decrease in mortality induces agents to substitute human capital for physical capital by the mechanism mentioned above. This shift in assets not only improves human capital, but also weakens the dilution effect because physical capital is vulnerable to the dilution effect compared to human capital.⁵ Second, a decrease in mortality raises the average age of the working population. Since older workers have more human capital because of human capital accumulation, such a change in the age composition of the workforce increases the average amount of human capital in the workforce. In the determination of per-capita output, the human capital improvement effect acts in the opposite direction to the physical capital dilution effect. Our model predicts that a decrease in mortality is more likely to increase GDP per capita in an economy with more education because the human capital improvement effect dominates the dilution effect, while the opposite is true in an economy with less education.

Figure 1 depicts wage profiles of workers in high- and low-education countries, illustrating the intuition behind our mechanism.⁶ If the wage rate reflects the amount of human capital, the wage profile represents human capital accumulation over a lifetime. The wage profile for high-education countries is steeper and has a higher peak than that for low-education countries, implying that the gap in human capital between young and adult workers is larger in high-

⁵The following example captures the vulnerability of physical capital to the dilution effect. Suppose that parents leave \$10,000 to their two children and there are two measures of asset maintenance, physical and human capital investment, both of which have the same gross rates of return, 100%. First, consider the case where \$10,000 is invested in physical capital. If the two children survive, each child gets physical capital of \$5,000. If one child dies, the other gets physical capital of \$10,000. Next, consider the case where \$10,000 is invested in human capital. If both children survive, each child gets human capital of \$5,000. If one child dies, the other gets human capital of \$5,000. The amount of assets per capita halves by the dilution effect when it is invested in physical capital, whereas it does not change when invested in human capital because human capital is embedded in individuals and disappears with their death.

⁶The classification between high- and low-education countries is based on the empirical result in Section 3 and is the same as the classification in panel (b) of Figure 4. Due to limited data availability, however, the sample in Figure 1 consists of 15 out of 47 countries used in the empirical analysis.



Source: Lagakos et al. (2015)

Figure 1: Wage profile in high- and low-education countries

education countries than that in low-education countries. A decrease in mortality has two effects: a direct effect and a composition effect. First, it lifts the profile from the low-education type to the high-education type by increasing educational investment. This directly improves the average amount of human capital because each agent has more human capital. Second, it shifts the weight on the workforce from young to adult workers, and thus has the effect of improving the average amount of human capital. This composition effect is larger in higher-education countries because they have a larger gap in the amount of human capital between older and younger workers.

A notable implication of our theory is heterogeneity in the effects of mortality on per-capita income among countries. The higher the level of education in a country, the more likely it is that a decrease in mortality will increase income per capita. To test this hypothesis, we use the same data and identification strategy as Acemoglu and Johnson (2007), though our approach differs in that we relax the assumption that all countries are in the same growth regime. Using a finite mixture model (FMM) approach, we investigate whether the effects of mortality on per-capita income differ between countries in high- and low-education regimes.⁷

⁷See Frühwirth-Schnatter (2006) for details about FMM, and Bloom et al. (2003), Owen et al. (2009), and Cervellati and Sunde (2011a) for applications to growth regressions.

Cervellati and Sunde (2011a, 2011b) share our motivation to reinterpret the analysis of Acemoglu and Johnson (2007). Cervellati and Sunde (2011b) split the sample of Acemoglu and Johnson (2007) between pre- and post-demographic-transition countries based on the classification criteria used in demography literature. They show that improved life expectancy has a negative effect on per-capita GDP in pre-transition countries, but a positive effect in post-transition countries. Cervellati and Sunde (2011a) use an FMM approach without imposing any ex-ante classification, obtaining similar results to those of Cervellati and Sunde (2011b). Cervellati and Sunde (2011a, 2011b) base their studies on demographic-transition theory to investigate the role of population growth in determining the effect of mortality on per-capita income. Here, we focus on the role of education in determining the effect of mortality on per-capita income based on human-capital theory. Since the demographic-transition and human capital theories are two pillars in the literature on long-run growth, our study is a natural development of this literature. The empirical part of our study shows that both education and population growth are important in determining the effect of mortality on per-capita income.

The remainder of this paper is organized as follows. Section 2 presents the model and analyses the effect of mortality changes on income per capita. In Section 3, we confront the testable implications of the model with empirical evidence. Section 4 concludes the paper.

2 Model

Consider an overlapping generations model in which agents live for at most two periods, youth and adulthood. Some agents die at the beginning of adulthood and cannot enter the labour market in adulthood.⁸ In both periods, agents inelastically supply one unit of time to the labour market. The efficiency units of labour per unit of time of young agents are normalized to 1 (i.e., the basic skill level), while those of adult agents can increase with an educational investment in their youth. At the end of the youth period, agents receive an inheritance from their ancestors

⁸We here concentrate on the role of adult mortality. See Subsection 2.6 for the effect of introducing child mortality into our model.

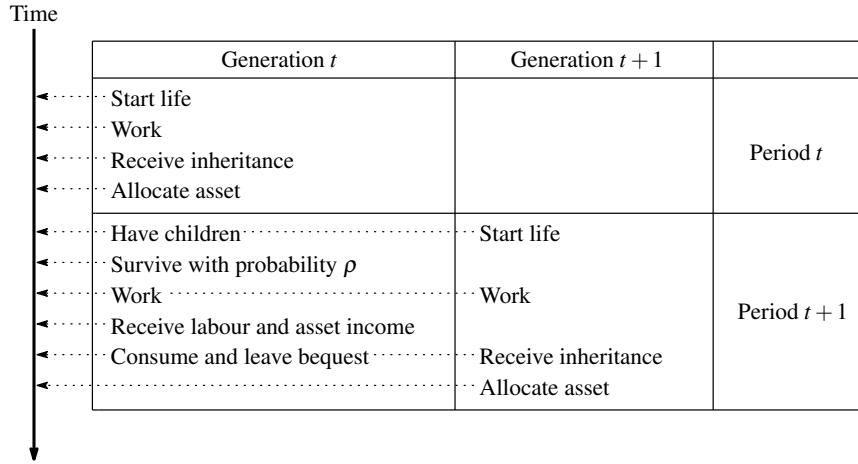


Table 1: Timing of events

and allocate it between savings and an educational investment. Savings and educational investment increase asset income and labour income in adulthood, respectively. In the second period (adulthood), surviving agents supply their efficiency units of labour and allocate the household's labour and asset income between their own consumption and transfers to their offspring. Table 1 depicts the timing of events.

The basic unit of our analysis is a household consisting of a continuum of individuals. Household members of the same generation jointly choose their behaviour to maximize joint utility. We impose this assumption mainly with couples in mind. In the real world, married couples would make consumption and investment decisions jointly and with concern for the other's living standards after their own death. The assumptions of joint utility and joint decision-making are intended to capture this reality.⁹

⁹The assumption of the continuum is for simplification. If we use a couple consisting of two persons as the basic unit, then heterogeneity arises between households (i.e., both parents survive, one parent survives, or both parents die) and the model becomes too complex to solve analytically. However, the essence of the proposed mechanism does not depend on whether we employ a household consisting of continuous or discrete members as the basic unit.

2.1 Production

Production occurs according to Cobb-Douglas production technology. The output produced in period t is given by $Y_t = K_t^\alpha H_t^{1-\alpha}$, where K_t and H_t are the quantities of physical capital and labour (measured in efficiency units), respectively, employed in production, and $\alpha \in (0, 1)$ is the capital share of income. The factor markets are competitive and physical capital fully depreciates in one period. The wage rate and the gross rate of return to physical capital are:

$$w_t = (1 - \alpha) k_t^\alpha \equiv w(k_t) \quad \text{and} \quad R_t = \alpha k_t^{\alpha-1} \equiv R(k_t), \quad (1)$$

respectively, where $k_t \equiv K_t/H_t$ is the capital-labour ratio.

2.2 Households

There is a continuum of households of measure 1. In every period, a continuum of individuals of measure 1 is born into a household as young members. They survive and enter the labour market in adulthood with probability $\rho \in (0, 1)$. Thus, each household has $1 + \rho$ members (i.e., 1 young members and ρ adult members) in every period. Since the measure of households is 1, the total population size of this economy is $1 + \rho$.

Household members of the same generation jointly choose their behaviour to maximize joint utility. The preferences of generation t (born in t) of household i are defined over consumption in adulthood, ρc_{t+1}^i , and transfers to their offspring, b_{t+1}^i . The variables c_{t+1}^i and b_{t+1}^i are consumption per surviving adult member and transfer per young member, respectively. Since ρ out of 1 adult members survive until the time of consumption, their total consumption is ρc_{t+1}^i . On the other hand, since the size of young members is 1, the total bequest is b_{t+1}^i . The preferences are given by:

$$\gamma \ln \rho c_{t+1}^i + (1 - \gamma) \ln b_{t+1}^i, \quad (2)$$

where $\gamma \in (0, 1)$ denotes the relative weight given to consumption.

In period t , generation t of household i receives an inheritance, b_t^i , from generation $t - 1$ of household i , which is allocated between savings, s_t^i , and education expenditure, e_t^i : $s_t^i + e_t^i = b_t^i$. The budget constraint of generation t of household i is:

$$\rho c_{t+1}^i + b_{t+1}^i = w_{t+1} + \rho w_{t+1} h_{t+1}^i + R_{t+1} (b_t^i - e_t^i), \quad (3)$$

where h_{t+1}^i denotes the efficiency units of labour (i.e., the level of human capital) per adult member. The LHS is the adult-period expenditure, namely their own consumption and transfers to their offspring. The RHS is the adult-period wealth: the first, second, and third terms are the wage income earned by young members, the wage income earned by surviving adults, and the return on savings from the previous period, respectively. Note that we assume that adult members control the young members' wage income. One may argue that this patriarchal assumption does not fit the description of developed countries, though is suitable for some developing nations. However, this assumption is not as restrictive as it may seem. In the real world, people have some degree of control over the wealth of their offspring because the transfer is usually either positive or negative. In this model, the net transfer to offspring, $b_{t+1}^i - w_{t+1}$, can be either positive or negative: the larger the amount of adult members' human capital, the more likely it is that the net transfer will be positive. Therefore, our model can capture the reality that there is an exploitation of child labour in underdeveloped countries, whereas in advanced countries, many people transfer a positive amount of wealth to their children, although they can leave a debt. Furthermore, this patriarchal assumption simplifies the dynamics because we need not consider the decisions of two generations simultaneously.

The level of human capital in adulthood, h_{t+1}^i , is a function of the real expenditure on education in childhood t , e_t^i :

$$h_{t+1}^i = 1 + \mu e_t^i, \quad (4)$$

where $\mu > 0$ represents the efficiency of education. Without capital investment in education,

the level of human capital in adulthood is equal to the basic level during youth.¹⁰ For analytical convenience, we assume a linear human capital production technology.¹¹

Generation t of household i maximizes (2) subject to (3) and (4). The solution to this problem is given by

$$e_t^i = \begin{cases} 0 & \text{if } \rho\mu w_{t+1} < R_{t+1}, \\ [0, \infty) & \text{if } \rho\mu w_{t+1} = R_{t+1}, \\ \infty & \text{if } \rho\mu w_{t+1} > R_{t+1}, \end{cases}$$

and

$$b_{t+1}^i = (1 - \gamma) [w_{t+1} + \rho w_{t+1} (1 + \mu e_t^i) + R_{t+1} (b_t^i - e_t^i)]. \quad (5)$$

There is a threshold level of capital-labour ratio, \tilde{k} , below which households do not invest in human capital, that is, \tilde{k} is defined by $\rho\mu w(\tilde{k}) = R(\tilde{k})$. It follows from (1) that $\tilde{k} = \alpha / [(1 - \alpha)\rho\mu]$. If the capital-labour ratio in the next period is expected to be below \tilde{k} , agents do not acquire non-basic skills.

Before moving to the equilibrium analysis, we should mention two points. First, the optimal level of human capital investment depends on the rate of return and is independent of the level of income. Since there are no credit constraints in this model, the rate of return is the investor's only concern. Second, as mortality declines, households are more likely to invest in human capital than in physical capital (i.e., \tilde{k} decreases with ρ). Since human capital is inherently embodied in people, human capital yields returns in adulthood for surviving members only, whereas physical capital does not have this property. With higher mortality, it is riskier for households to con-

¹⁰We are aware that the opportunity cost as well as the pecuniary cost constitutes a major part of the education costs in the real world (e.g., Bils and Klenow, 2000), and that time devoted to education matters in determining educational attainment. Following the spirit of Galor and Moav (2004), however, we assume that there is no difference in the time agents devote to education, enabling us to concentrate on the portfolio choice between physical and human capital investments.

¹¹If we assume a strictly concave human capital production technology, perpetual growth (Pattern III in Figure 2) does not arise. However, all comparative-static results in Propositions 1 and 2, except for those in the balanced growth path, still hold. Since the emergence of perpetual growth is not essential in this study, we assume linear technology to obtain a closed-form solution.

concentrate their assets in human capital. Thus, human capital investment becomes more profitable compared to physical capital investment as mortality declines.

2.3 Aggregate physical and human capital

Assume that all households have identical initial capital ownership. We then focus on a symmetric equilibrium where $s_t^i = s_t$ and $e_t^i = e_t$ for all t and i .¹² Given the inheritance received by households of generation t , the optimization of these households in period t determines the level of physical capital, K_{t+1} , and human capital, H_{t+1} , in period $t + 1$: $K_{t+1} = s_t = b_t - e_t$ and $H_{t+1} = 1 + \rho(1 + \mu e_t)$. It follows that the capital-labour ratio in period $t + 1$ is:

$$k_{t+1} = \frac{b_t - e_t}{1 + \rho(1 + \mu e_t)}. \quad (6)$$

First, suppose that the capital-labour ratio in the next period is expected to be below \tilde{k} . Since $e_t = 0$ in this case, $k_{t+1} = b_t / (1 + \rho)$. The resultant capital-labour ratio is consistent with households' expectation if and only if $b_t / (1 + \rho) \leq \tilde{k}$, that is,

$$b_t \leq \frac{\alpha(1 + \rho)}{(1 - \alpha)\rho\mu} \equiv \tilde{b}. \quad (7)$$

In the case of $b_t > \tilde{b}$, on the other hand, $e_t > 0$. Note that when $e_t > 0$, $k_{t+1} = \tilde{k}$ must hold. If $k_{t+1} < \tilde{k}$, then no one invests in human capital (i.e., $e_t = 0$). If $k_{t+1} > \tilde{k}$, then no one invests in physical capital and, thus, $k_{t+1} = 0$, which is inconsistent with $k_{t+1} > \tilde{k}$. When $e_t > 0$, using (6) and $k_{t+1} = \tilde{k}$, we obtain $e_t = [(1 - \alpha)\rho\mu b_t - \alpha(1 + \rho)] / (\rho\mu)$. Therefore, we can write the

¹²It is also possible that some households specialize in physical capital accumulation and others specialize in human capital accumulation, and that the ratio between the two is determined in equilibrium. However, our results do not change in that situation because the aggregate variables in the asymmetric equilibrium are the same as those in the symmetric equilibrium by households' arbitrage behaviour.

equilibrium educational investment as a function of b_t :

$$e_t = \begin{cases} 0 & \text{if } b_t \leq \tilde{b}, \\ \frac{(1-\alpha)\rho\mu b_t - \alpha(1+\rho)}{\rho\mu} & \text{if } b_t > \tilde{b}. \end{cases}$$

This implies that a higher ρ and larger b_t induce a larger educational investment. Although the level of inheritance does not affect the investment decision at the household level, it matters at the aggregate level because of the general-equilibrium effect: the greater the resources available, the higher the level of human capital investment such that the marginal returns on physical and human capital are equalized.

Then, it follows from (1), (5), and (6) that the evolution of inheritance is:

$$b_{t+1}(b_t) = \begin{cases} (1-\gamma)(1+\rho)^{1-\alpha} b_t^\alpha \equiv \Psi_1(b_t) & \text{if } b_t \leq \tilde{b}, \\ (1-\gamma)\alpha^\alpha(1-\alpha)^{1-\alpha}\rho^{-\alpha}\mu^{-\alpha}(1+\rho+\rho\mu b_t) \equiv \Psi_2(b_t) & \text{if } b_t > \tilde{b}, \end{cases} \quad (8)$$

which governs the dynamics of this economy. The difference equation (8) implies $\Psi_1(\tilde{b}) = \Psi_2(\tilde{b})$, $\Psi'_1(\tilde{b}) = \Psi'_2(\tilde{b})$, $b_{t+1}(0) = \Psi_1(0) = 0$, $b'_{t+1}(b_t) > 0$, $b'_{t+1}(0) = \Psi'_1(0) = \infty$, $b''_{t+1}(b_t) < 0$ if $b_t \leq \tilde{b}$, and $b''_{t+1}(b_t) = 0$ if $b_t > \tilde{b}$. Therefore, depending on the parameter configuration, we can classify the dynamic system into three patterns (see Figure 2). In Pattern I (panel (a)), there exists a steady state where physical capital investment is the only method of capital accumulation. In Pattern II (panel (b)), there exists a steady state with both physical and human capital investments. In Pattern III (panel (c)), the economy shows persistent growth, and both physical and human capital investments exist along the growth path. For Pattern I to arise, $b_1^* = (1-\gamma)^{\frac{1}{1-\alpha}}(1+\rho) \leq \tilde{b}$, where b_1^* is a fixed point of $\Psi_1(\cdot)$. For Pattern II to arise, $b_1^* > \tilde{b}$ and $\Psi'_2(b_t) = (1-\gamma)\alpha^\alpha(1-\alpha)^{1-\alpha}\rho^{1-\alpha}\mu^{1-\alpha} < 1$. For Pattern III to arise, $\Psi'_2(b_t) \geq 1$.

The pattern to arise depends on some parameters. According to the value of the survival rate,

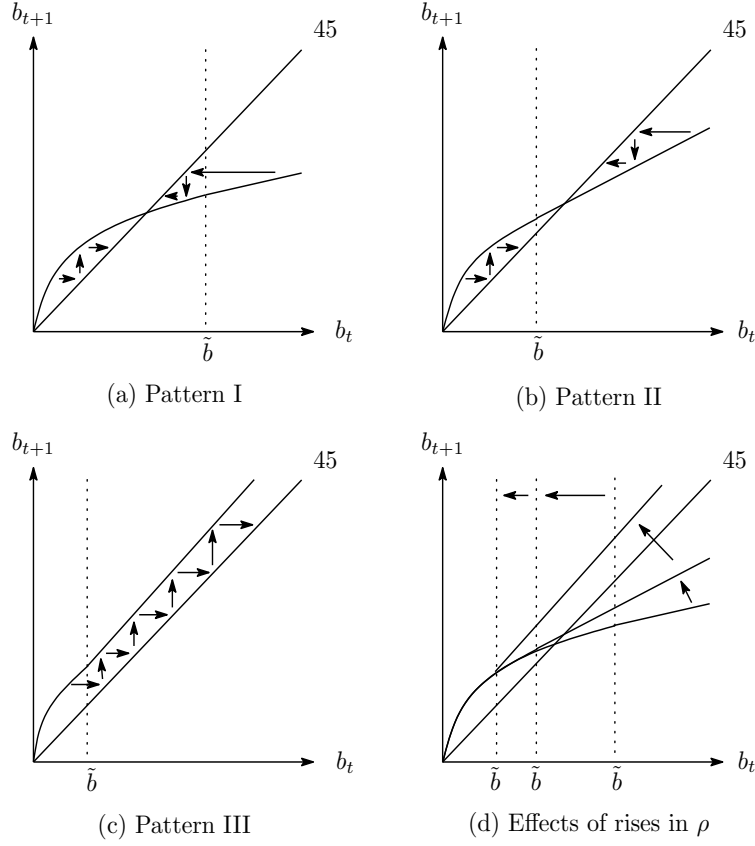


Figure 2: Dynamics

ρ , the main parameter of interest, the patterns of the dynamics of this economy are classified as

$$\begin{cases} \text{Pattern I} & \text{if } \rho \leq \tilde{\rho}_1, \\ \text{Pattern II} & \text{if } \tilde{\rho}_1 < \rho < \tilde{\rho}_2, \\ \text{Pattern III} & \text{if } \tilde{\rho}_2 \leq \rho, \end{cases}$$

$$\text{where } \tilde{\rho}_1 \equiv \frac{\alpha}{(1-\gamma)^{\frac{1}{1-\alpha}}(1-\alpha)\mu} \text{ and } \tilde{\rho}_2 \equiv \frac{\alpha^{-\frac{\alpha}{1-\alpha}}}{(1-\gamma)^{\frac{1}{1-\alpha}}(1-\alpha)\mu}. \quad (9)$$

A rise in ρ shifts $\Psi_1(\cdot)$ and $\Psi_2(\cdot)$ upward and decreases \tilde{b} . A decrease in mortality increases the steady-state value of b_t , if it exists. Otherwise, it raises the growth rate of b_t . Panel (d) in

Figure 2 illustrates this effect. Decreases in mortality induce the economy to move to the pattern with higher education.

We conclude this subsection by presenting the steady-state inheritances in Patterns I and II (the fixed points of $\Psi_1(\cdot)$ and $\Psi_2(\cdot)$, respectively):

$$\begin{cases} b_1^* = (1 - \gamma)^{\frac{1}{1-\alpha}} (1 + \rho) & \text{in Pattern I,} \\ b_2^* = \frac{(1-\gamma)\alpha^\alpha(1-\alpha)^{1-\alpha}\rho^{-\alpha}\mu^{-\alpha}(1+\rho)}{1-(1-\gamma)\alpha^\alpha(1-\alpha)^{1-\alpha}\rho^{1-\alpha}\mu^{1-\alpha}} & \text{in Pattern II.} \end{cases}$$

2.4 Effect of mortality changes on per-capita output

It follows from the results obtained in the previous subsection that given b_t , b_{t+1} increases with ρ (panel (d) in Figure 2). Then, what about the effect on per-capita output? The answer is not clear, because a higher survival rate increases the population and dilutes physical capital per capita. This subsection attempts to answer this question.

Our analysis distinguishes between the short- and long-run effects of mortality changes on per-capita output: the short- and long-run effects refer to the effects on per-capita output of the current generation and the per-capita output in the steady state (or that for the balanced growth path), respectively. Here we impose an additional assumption:

$$\alpha < \frac{1}{1 + \rho}, \quad (10)$$

which means that human capital has a sufficiently large contribution to production. This assumption seems empirically reasonable. The value of $1/(1 + \rho)$ is in $[1/2, 1]$ because the value of ρ is in $[0, 1]$, whereas it is widely accepted that the capital share, α , is less than $1/2$.

First, we obtain the following proposition regarding the short-run effect of a change in the survival rate on per-capita output.¹³

¹³Although we here analyse the effect of the ‘expected’ mortality change, it is also possible to analyse the effect of the ‘unexpected’ mortality change. The difference between the effects of the expected and unexpected changes is only quantitative. The negative effect on per-capita output is stronger in the case of unexpected change because households do not react optimally in response to a mortality change.

Proposition 1. *In the short run, (i) in the case without human capital investment (i.e., $b_t \leq \tilde{b}$), a rise in ρ decreases per-capita output, and (ii) in the case with positive human capital investment (i.e., $b_t > \tilde{b}$), the effect of a rise in ρ on per-capita output can be either positive or negative: a larger value of b_t is more likely to make the effect positive.*

Proof. Given b_t , output per capita in period $t + 1$, $y_{t+1} \equiv Y_{t+1}/(1 + \rho)$, is

$$y_{t+1} = \begin{cases} [b_t/(1 + \rho)]^\alpha & \text{if } b_t \leq \tilde{b}, \\ \alpha^\alpha (1 - \alpha)^{1-\alpha} \mu^{-\alpha} (1 + \rho + \rho \mu b_t) / [(1 + \rho) \rho^\alpha] & \text{if } b_t > \tilde{b}. \end{cases} \quad (11)$$

(i) It follows that $\partial y_{t+1} / \partial \rho < 0$ when $b_t \leq \tilde{b}$.

(ii) When $b_t > \tilde{b}$, we obtain the following:

$$\begin{aligned} \frac{\partial y_{t+1}}{\partial \rho} &= \frac{\alpha^\alpha (1 - \alpha)^{1-\alpha}}{\mu^\alpha} \\ &\times \frac{(1 + \mu b_t)(1 + \rho) \rho^\alpha - (1 + \rho + \rho \mu b_t) [\rho^\alpha + \alpha (1 + \rho) \rho^{\alpha-1}]}{[(1 + \rho) \rho^\alpha]^2}, \end{aligned} \quad (12)$$

and

$$\frac{\partial^2 y_{t+1}}{\partial b_t \partial \rho} = \frac{\alpha^\alpha (1 - \alpha)^{1-\alpha} \mu^{1-\alpha} [1 - \alpha (1 + \rho)]}{(1 + \rho)^2 \rho^\alpha}.$$

Under the assumption (10), $\partial^2 y_{t+1} / \partial b_t \partial \rho > 0$: a higher value of ρ is more likely to increase per-capita output as b_t rises. We denote the inheritance level such that a change in ρ has no effect on per-capita output by \hat{b} (i.e., $\partial y_{t+1}(\hat{b}) / \partial \rho = 0$). Using (12), we obtain $\hat{b} = \alpha (1 + \rho)^2 / \{\rho \mu [1 - \alpha (1 + \rho)]\}$. We can easily confirm that $\hat{b} > \tilde{b}$. Therefore, when $b_t > \tilde{b}$, $\partial y_{t+1} / \partial \rho > 0$ if and only if $b_t > \hat{b}$. ■

When physical capital investment is the only method of capital accumulation, a decrease in mortality decreases per-capita output in the short run. Since b_t is given, the assets available to the generation with declining mortality do not change. Thus, the quantity of labour input increases, while the quantity of physical capital does not change, as a result of the decrease in mortality.

The average labour productivity decreases by the diminishing marginal productivity of labour, and thus per-capita output decreases.

In the case with positive human capital investment, the short-run effect is positive if b_t is sufficiently large. The intuition behind this result is as follows. As in the case of $b_t \leq \tilde{b}$, a decrease in mortality increases labour inputs and decreases the return per unit of human capital. However, in contrast to the case of $b_t \leq \tilde{b}$, a decrease in mortality increases the average amount of human capital in the labour market, which is the weighted average of young and adult agents' human capital: $1/(1+\rho) \times 1 + \rho/(1+\rho) \times h_{t+1}$. There are two effects of a decrease in mortality on the average amount of human capital. First, a decrease in mortality induces households to invest in human capital (i.e., $\partial e_t / \partial \rho > 0$) and, thus, h_{t+1} increases. Second, a decrease in mortality raises the proportion of adult workers, $\rho/(1+\rho)$, in the labour market. Since adult workers have more human capital than young workers (i.e., $h_{t+1} > 1$), a rise in the proportion of adult workers increases the average amount of human capital. Therefore, the negative effect on physical capital and the positive effect on human capital coexist, and thus the total effect can be either positive or negative. If b_t is large, then the educational investment is large, so the growth rate of human capital from youth to adulthood is high. Then, the higher weight of adult human capital resulting from a decrease in mortality has a greater effect on increasing the average amount of human capital. Therefore, as b_t rises, a higher value of ρ is more likely to make the positive effect dominant.

Next, we obtain the following proposition regarding the long-run effect of a change in the survival rate on per-capita output.

Proposition 2. *In the long run, (i) in Pattern I, a rise in ρ has no effect on per-capita output, (ii) in Pattern II, a rise in ρ increases per-capita output, and (iii) in Pattern III, a rise in ρ raises the growth rate of per-capita output.*

Proof. (i) In Pattern I, output per capita in the steady state is $y^* \equiv Y^*/(1+\rho) = (1-\gamma)^{\frac{\alpha}{1-\alpha}}$. It follows that $\partial y^* / \partial \rho = 0$.

(ii) In Pattern II, the output per capita in the steady state is

$$y^* = \frac{\alpha^\alpha (1-\alpha)^{1-\alpha}}{\rho^\alpha \mu^\alpha - (1-\gamma) \alpha^\alpha (1-\alpha)^{1-\alpha} \rho \mu}.$$

This implies

$$\frac{\partial y^*}{\partial \rho} = - \frac{\alpha^{\alpha+1} (1-\alpha)^{1-\alpha} \rho^{\alpha-1} \mu^\alpha \Omega(\rho)}{\left[\rho^\alpha \mu^\alpha - (1-\gamma) \alpha^\alpha (1-\alpha)^{1-\alpha} \rho \mu \right]^2},$$

$$\text{where } \Omega(\rho) \equiv 1 - (1-\gamma) \alpha^{\alpha-1} (1-\alpha)^{1-\alpha} \rho^{1-\alpha} \mu^{1-\alpha}.$$

Note that $\Omega'(\rho) < 0$ and $\Omega(\tilde{\rho}_1) = 0$. Since we now consider the case of $\rho > \tilde{\rho}_1$, we obtain $\partial y^* / \partial \rho > 0$.

(iii) In Pattern III, since there is no steady state, we analyse the long-run growth rate instead. Using (8) and (11), we obtain the following per-capita output growth rate:

$$\frac{y_{t+1} - y_t}{y_t} = (1-\gamma) \alpha^\alpha (1-\alpha)^{1-\alpha} \rho^{1-\alpha} \mu^{1-\alpha} - \frac{\rho \mu b_{t-1}}{1 + \rho + \rho \mu b_{t-1}}.$$

Since b_t grows persistently, $b_{t-1} \rightarrow \infty$ as $t \rightarrow \infty$. Thus, in the long run, the growth rate converges to $(1-\gamma) \alpha^\alpha (1-\alpha)^{1-\alpha} \rho^{1-\alpha} \mu^{1-\alpha} - 1$, which rises with ρ . ■

In the long run, in contrast to the fixed inheritance in the short run, the future generation's inheritance increases because an increase in the survival rate increases labour inputs and thus output. This effect offsets the diluting effect of an increase in population size. Since physical capital per capita does not decrease, output per capita does not decrease, even if there is no positive effect on human capital investment. In the case with positive human capital investment, a rise in the survival rate necessarily increases output per capita by enhancing human capital investments.

In summary, the above analysis generates the following predictions: (i) the effect of a decrease in mortality on per-capita income is ambiguous; (ii) the effect of a decrease in mortality on per-capita output is more likely to be positive in the long run than in the short run; and (iii) the

higher a country's education level, the more likely it is that a decrease in mortality will increase output per capita.

2.5 Numerical illustration

This subsection presents numerical examples to illustrate our key results. The productivity parameter in education, μ , is set to unity, and the value of physical capital share, α , is set to $1/3$. The parameter related to preferences on own consumption, γ , is specific to the analysis at hand, and little is known about its magnitude. Thus, we specify a value of γ to ensure that the regime switch occurs at a reasonable survival rate and such that $\tilde{\rho}_1 = 0.75$ when $\alpha = 1/3$ and $\mu = 1$. Note that $\tilde{\rho}_1$ represents the threshold survival rate given by (9), below which Pattern I arises. This procedure leads to $\gamma \approx 0.237$.

Heterogeneity in ρ We demonstrate how a once and permanent increase in the survival rate, ρ , affects per-capita output. Suppose that the economies are initially in steady states, with $\rho = 0.6$, $\rho = 0.7$, and $\rho = 0.8$. We now increase the value of ρ from 0.6 to 0.7, from 0.7 to 0.8, and from 0.8 to 0.9. Since $\tilde{\rho}_1$ (the threshold survival rate between Patterns I and II) is 0.75, the economy with an initial survival rate of 0.6 is in Pattern I, both before and after the change, the economy with an initial survival rate of 0.7 switches from Pattern I to Pattern II as a result of the change, and the economy with an initial survival rate of 0.8 is in Pattern II, both before and after the change. Panel (a) in Figure 3 depicts the responses in per-capita output in these simulations. The per-capita output steeply decreases immediately after the change due to the dilution effect on physical capital. Subsequently, per-capita output starts increasing due to physical capital accumulation by the larger working population, returning to the initial steady-state level in Case 1. Per-capita output increases further by increases in the average amount of human capital, converging to the new steady-state levels above the initial levels in Cases 2 and 3.

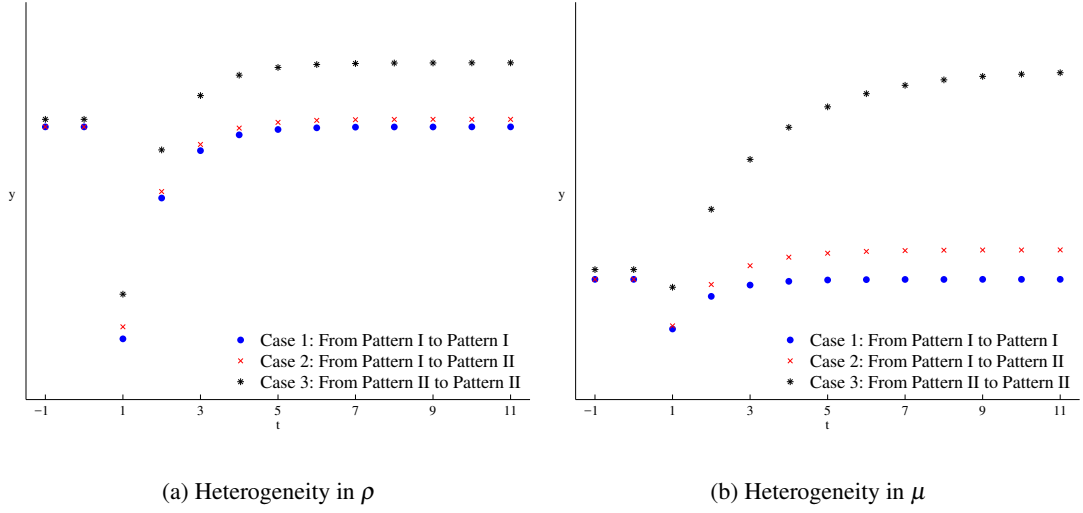


Figure 3: Simulation results

Heterogeneity in μ Next, we examine the differential effects of mortality changes between economies with different education systems. We model the development of an education system as an increase in μ : a higher value of μ implies a more efficient education system. Here, we examine the responses in per-capita output to mortality changes for different values of μ , along with the same values of α and γ used above. Suppose the economies are initially in steady states, with $\mu = 0.5$, $\mu = 1.5$, and $\mu = 2.5$. Then, the respective threshold survival rates between Patterns I and II are 1.5, 0.5, and 0.3. Now, we increase the value of ρ from 0.4 to 0.8. The economy with $\mu = 0.5$ is in Pattern I, both before and after the change; the economy with $\mu = 1.5$ switches from Pattern I to Pattern II as a result of the change; and the economy with $\mu = 2.5$ is in Pattern II, both before and after the change. Panel (b) in Figure 3 displays the responses of per-capita output in these simulations, which decreases steeply immediately after the change due to the dilution effect on physical capital. Subsequently, the per-capita output starts increasing, returning to the initial steady-state level in Case 1 and converging to the new steady-state levels above the initial levels in Cases 2 and 3. The mechanisms behind these dynamics are the same as the simulations with heterogeneity in ρ .

Panels (a) and (b) in Figure 3 capture the feature that we want to emphasize: the higher a country's education level, the more likely it is that a decrease in mortality will increase income per capita. Regardless of the cause of lower education levels (i.e., lower ρ or lower μ), a rise in the survival rate is more likely to have a negative effect on per-capita output in an economy with a lower level of education: the size of the initial drop is larger, the pace of the subsequent recovery is slower, and the steady-state level attained finally is lower.¹⁴

2.6 Child Mortality

We have thus far focused on adult mortality and ignored child mortality. However, life expectancy generally reflects both adult and child mortality, and child mortality has a key role in its determination. This subsection introduces child mortality into our model, showing that such an extension has only a minor effect on the results obtained above.

We assume that $\delta \in (0, 1)$ out of 1 young members survive and enter the labour market in youth, that is, new-born agents die at the beginning of their lives with probability $1 - \delta$. Since the sizes of adult and young members change from ρ to $\delta\rho$ and from 1 to δ , respectively, the utility function (2) becomes:

$$\gamma \ln \delta \rho c_{t+1}^i + (1 - \gamma) \ln \delta b_{t+1}^i. \quad (13)$$

The budget constraint remains the same as that in the model without child mortality, (3), because all arguments in equation (3) are multiplied by δ when we introduce child mortality, and it thus has virtually no effect on the equation. Generation t of household i maximizes (13) subject to (3) and (4). The solution to this problem is the same as that in the model without child mortality. That is, introducing child mortality does not change the household's behaviour. The dynamic

¹⁴We here assume a parameter configuration such that per-capita output decreases in the short run in response to a rise in ρ (i.e., the initial steady-state value of b_t is smaller than \hat{b}). For a simulation of Case 3 where an economy is initially in Pattern II, we can choose a parameter configuration that makes the short-run effect positive, as shown in Proposition 1.

equation of the capital-labour ratio also remains the same as that in the model without child mortality, (6), because both the numerator and the denominator in the RHS of equation (6) are multiplied by δ if child mortality is introduced, and thus, it has virtually no effect on the equation. It follows that introducing child mortality does not change the dynamics of the state variable. Therefore, there is no change in the effect of a rise in the adult survival rate, ρ , in Propositions 1 and 2 after introducing child mortality.

In the real world, new drug development and improvements in nutrition and sanitation usually decrease both child and adult mortality. Here, let us consider the effect of a simultaneous change in child and adult mortality. Suppose the child and adult survival rates rise from δ to δ' and ρ to ρ' , respectively. As mentioned above, the effect of a change in ρ does not depend on the presence of child mortality, and thus, the response of per-capita output is similar to the responses depicted in Figure 3. The only difference after adding child mortality changes is in the short-run effect. In the period when mortality changes, the proportion of young members in the population becomes $\delta' / (\delta' + \delta\rho')$, which is larger than the proportion in the case without child mortality change, $1 / (1 + \rho')$. However, the proportion becomes $1 / (1 + \rho')$ in one period and remains the same from then on. This temporary change in the age composition of the population strengthens the dilution effect on physical capital and decreases the average amount of human capital. Therefore, the size of the initial drop is larger in the case associated with a fall in child mortality than in the case without, but there is no qualitative difference in the response of per-capita output with respect to mortality changes between these two cases, and is negligible in the long run.

3 Empirical analysis

A notable implication of our theory is heterogeneity in the effects of mortality on per-capita income among countries: a higher education level increases the likelihood that a decrease in mortality will increase income per capita. In this section, we use data to test this implication.

3.1 Empirical framework and identification

The dependent variable is the change in log GDP per capita, and the main explanatory variable is the change in predicted mortality. Our estimations are based on the following estimation framework:

$$\Delta \ln y_i = C + \pi \Delta m_i + u_i,$$

where $\Delta \ln y_i$ and Δm_i are the changes in log GDP per capita and the (instrumented) predicted mortality of country i between 1940 and 1980, respectively. Following Cervellati and Sunde (2011a), we use the change in predicted mortality rather than life expectancy as the regressor. Note that a negative coefficient π implies that a decrease in mortality promotes growth.

To estimate the causal effect of mortality on per-capita income, we follow the identification strategy proposed by Acemoglu and Johnson (2007). The instrument for the change in mortality is the reduction in mortality from 15 infectious diseases (e.g., tuberculosis, malaria, and pneumonia), following the international epidemiological transition in the 1940s.¹⁵ The basic idea behind this choice of instrument is as follows.¹⁶ There were no appropriate treatments for these diseases in 1940 and the international epidemiological transition enabled the worldwide dissemination of treatment and prevention by 1980. Thus, the change in mortality resulting from these diseases between 1940 and 1980 was exogenous to each country, and we can assume that the exclusion restriction holds.

The departure from Acemoglu and Johnson (2007) is that we relax the assumption that all countries are in the same growth regime to allow for heterogeneous effects of mortality on per-capita income between regimes. For this purpose, we use a finite-mixture approach, jointly estimating the probability of being in a distinct growth regime and the effect of mortality changes in each regime. Compared to the approach that assigns observations into groups a priori, our

¹⁵The international epidemiological transition consists mainly of three factors: a wave of global drug and chemical innovations, the establishment of the WHO and UNICEF, and the change in international values leading to a fast dissemination of medical knowledge. The international epidemiological transition caused a dramatic improvement in life expectancy in much of the world, particularly in less developed areas.

¹⁶See Acemoglu and Johnson (2007) for more detail.

approach has the advantage of being entirely data driven because the country groupings into regimes as well as the growth regression parameters for each regime is estimated.

We assume that growth processes can be classified into two discrete regimes.¹⁷ We refer to these as “Low” and “High.” As will become apparent later, education level is a significant predictor of regime membership. The labels “Low” and “High” refer to low- and high-education regimes, respectively. The probability structure for a given country is:

$$f(\Delta \ln y | \Delta m; \pi^{\text{Low}}, \pi^{\text{High}}, \phi^{\text{Low}}, \phi^{\text{High}}) = \phi^{\text{Low}} f(\Delta \ln y | \Delta m; \pi^{\text{Low}}) + \phi^{\text{High}} f(\Delta \ln y | \Delta m; \pi^{\text{High}}),$$

where ϕ^j is the probability of membership in latent regime j , and $f(\Delta \ln y | \Delta m; \pi^j)$ is the distribution of growth rates conditional on membership in latent regime j . The probabilities are parameterized as:

$$\phi^{\text{Low}} = \frac{\exp(\theta X)}{1 + \exp(\theta X)} \quad \text{and} \quad \phi^{\text{High}} = \frac{1}{1 + \exp(\theta X)},$$

where X represents the determinants of regime membership, and θ is the coefficient that captures the effect of X on the probability of being in the Low regime. Based on the prediction derived in the theoretical part, we use variables related to the level of education as covariates X .

We estimate the model via maximum likelihood. Assume that the error term in the growth equation comes from a normal distribution with standard deviation σ^j . The log-likelihood function is:

$$\ln L = \prod_{i=1}^N \left\{ \ln \left[\phi^{\text{Low}} f(\Delta \ln y_i | \Delta m_i; \pi^{\text{Low}}) + \phi^{\text{High}} f(\Delta \ln y_i | \Delta m_i; \pi^{\text{High}}) \right] \right\},$$

where

$$f(\Delta \ln y_i | \Delta m_i; \pi^j) = \frac{1}{\sqrt{2\pi(\sigma^j)^2}} \exp \left[-\frac{(\Delta \ln y_i - C^j - \pi^j \Delta m_i)^2}{2(\sigma^j)^2} \right].$$

¹⁷ Although it is possible to assume more than two regimes, we select the two-regime model based on the results of the likelihood-ratio test.

We estimate this finite mixture model using the Stata package fmm (Deb, 2012).

3.2 Data

The dependent variable is the change in log GDP per capita between 1940 and 1980, and the main explanatory variable is the change in predicted mortality between 1940 and 1980. We use Maddison (2003) and Acemoglu and Johnson (2007) as data sources for these variables. We use the same base sample of 47 countries as in Acemoglu and Johnson (2007), for which all relevant data on these variables are available for 1940 and 1980.

We use education level as a predictor of regime membership. Specifically, we use variables related to tertiary education as regime membership predictors. Recall that our theory addresses agents' decisions about their own education, rather than education provided by parents. It is reasonable to focus on variables related to higher levels of education for consistency with the theory. Further, we check robustness by using the proportion of no-schooling among persons as another measure of education. The data on education comes from Barro and Lee (2013).

This study focuses mainly on the role of education in the heterogeneous effects of mortality on income per capita based on human capital theory, though other studies focus on the role of population growth based on demographic-transition theory (Cervellati and Sunde, 2011a, 2011b). Our baseline estimations only use variables related to education as regime-membership predictors, but we also estimate models that include variables related to both education and population as regime-membership predictors for comparison with previous studies. Our data source for the population variables is Maddison (2003).

Table 2 provides the summary statistics.

3.3 Results and implications

Table 3 presents the main estimation results. The results reported in column [1] are qualitatively the same as the results obtained in Acemoglu and Johnson (2007). With the full sample, the effect of mortality on income per capita is positive (a positive coefficient of predicted mortality

Variables	Obs	Mean	Std. Dev.	Min	Max
Change of log GDP per capita (1940–1980)	47	0.884	0.388	−0.086	1.606
Change of predicted mortality (1940–1980)	47	−0.473	0.280	−1.126	−0.121
Years of tertiary schooling : 30–39 (1950)	47	0.095	0.124	0.002	0.604
Years of tertiary schooling : 30–49 (1950)	47	0.090	0.115	0.001	0.548
Tertiary complete (%) : 30–39 (1950)	47	1.824	2.380	0.038	11.957
No schooling (%) : 30–39 (1950)	47	32.685	29.410	0.2	86.7
Log of GDP per capita (1940)	47	7.739	0.725	6.332	8.855
Change of log population (1930–1940)	47	0.130	0.072	−0.015	0.303

Table 2: Summary statistics

change), that is, the effect of life expectancy on income per capita is negative.

Columns [2]-[9] present estimates of the finite mixture models. In columns [2] and [3], as the baseline estimation, we use the average number of years of tertiary schooling in the population between the ages of 30 and 39 in 1950 as covariate X to predict the latent regime membership. To draw accurate conclusions about country characteristics that sort countries into regimes, we should use membership predictors that are uncorrelated with the error term in the growth model. Since people over 30 years of age in 1950 completed their education by the onset of the international epidemiological transition, it seems reasonable to use a measure of tertiary schooling in the population between the ages of 30 and 39 in 1950 as the covariate. The estimates in columns [2] and [3] indicate the existence of two different regimes with opposite effects of mortality on income growth. Countries with a higher level of education are less likely to be classified into the Low regime (a negative coefficient of covariate). A decrease in mortality tends to increase income per capita in the High regime (a negative coefficient of predicted mortality change) and decrease income per capita in the Low regime (positive coefficient). This result is consistent with our theory.

In the estimations reported in columns [4]-[9], we use different education criteria to those used in columns [2] and [3] as predictors of regime membership to check the robustness of our results. The estimations reported in columns [4] and [5] use the average number of years of tertiary schooling in the population between the ages of 30 and 49, rather than between 30 and 39, as covariates. The estimations reported in columns [6] and [7] use the proportion of the

	OLS	Finite Mixture Model							
Covariate		Years of tertiary				Tertiary complete		No schooling	
Age group		30–39		30–49		30–39		30–39	
Regime		Low	High	Low	High	Low	High	Low	High
	[1]	[2]	[3]	[4]	[5]	[6]	[7]	[8]	[9]
Predicted mortality change	0.585*** [0.147]	0.574*** [0.207]	−0.525* [0.315]	0.590*** [0.224]	−0.532 [0.332]	0.559** [0.242]	−0.497 [0.330]	0.495*** [0.143]	−0.445 [0.419]
Constant	1.160*** [0.069]	1.052*** [0.213]	1.014*** [0.081]	1.071*** [0.253]	1.007*** [0.080]	1.033*** [0.269]	1.022*** [0.087]	0.972*** [0.163]	1.068*** [0.070]
Probability of regime High		0.338		0.324		0.357		0.362	
σ		0.335*** [0.064]	0.163*** [0.034]	0.341*** [0.08]	0.159*** [0.041]	0.332** [0.08]	0.163** [0.038]	0.311*** [0.069]	0.156*** [0.061]
Coefficient of covariate		−17.537** [7.032]		−18.879** [9.329]		−1.077** [0.430]		0.041* [0.022]	
Constant		2.154 [1.332]		2.286 [1.951]		2.274 [1.525]		−0.548 [1.081]	
AIC		42.452		42.365		41.37		40.214	
BIC		57.254		57.166		56.172		55.015	
SABIC		32.163		32.075		31.081		29.924	
Number of countries	47	47		47		47		47	

OLS (Column [1]) and FMM with two components (Columns [2]–[9]). All regressions are long-difference specifications with two observations per country, for 1940 and 1980. The dependent variable is the log of GDP per capita. The independent variable is predicted mortality. Regressions in columns [2]–[5] and [6]–[7] use years of tertiary schooling and tertiary completed in 1950 as covariates, respectively. Regressions in columns [8]–[9] use no schooling in 1950 as covariates. The samples correspond to the base sample of Acemoglu and Johnson (2007). AIC, BIC, and SABIC are Akaike, Bayesian, and Sample Adjusted Bayesian information criteria, respectively. Robust standard errors are in brackets. ***, **, * indicate significance at the 1-, 5-, and 10-% levels, respectively.

Table 3: Estimation results I

population that completed tertiary education, rather than the average number of years of tertiary schooling, as covariates. The estimations reported in columns [8] and [9] use the proportion of no-schooling among persons as covariates. The results reported in columns [4]–[9] demonstrate that the choice of measures of education has only minor effects compared to the main finding of heterogeneous effects of mortality on growth.

Figure 4 intuitively captures the difference between our results and those of Acemoglu and Johnson (2007). Panel (a) in Figure 4 shows that in the full sample, as reported by Acemoglu and Johnson (2007), a decrease in mortality depresses income per capita. In panel (b) in Figure 4, we assign countries into two groups based on the estimations in columns [2] and [3] in Table 3. In the first group, the countries have an estimated probability of being in the “High” regime of more than 50% (High group), and the second group consists of all other countries (Low group). It follows from panel (b) in Figure 4 that a decrease in mortality boosts income per capita in the

Finite Mixture Model				
Covariate	Years of tertiary/Log of GDP per capita		Years of tertiary/Population Growth	
Age group	30–39		30–39	
Regime	Low	High	Low	High
	[1]	[2]	[3]	[4]
Predicted mortality change	0.549*** [0.207]	−0.527* [0.301]	0.449*** [0.169]	−0.632** [0.286]
Constant	1.029*** [0.135]	1.017*** [0.070]	0.970*** [0.137]	1.012*** [0.070]
Probability of regime High	0.354		0.362	
σ	0.332*** [0.063]	0.164*** [0.036]	0.335*** [0.051]	0.155*** [0.026]
Coefficient of covariate	−13.323* / −0.568 [7.926]/[0.775]		−15.454* / 22.896** [8.473]/[10.804]	
Constant	6.176 [5.722]		−0.808 [1.219]	
AIC	44.111		38.863	
BIC	60.762		55.514	
SABIC	32.535		27.287	
Number of countries	47		47	

FMM with two components. All regressions are long-difference specifications with two observations per country: 1940 and 1980. The dependent variable is the log of GDP per capita. The independent variable is predicted mortality. The regression reported in columns [1] and [2] uses years of tertiary schooling in 1950 and the log of GDP per capita in 1940 as covariates. The regression reported in columns [3] and [4] uses tertiary education completed in 1950 and the change in the log of population between 1930 and 40 as covariates. Robust standard errors are in brackets. ***, **, * indicate significance at the 1-, 5-, and 10-% levels, respectively.

Table 4: Estimation results II

High group, but depresses income per capita in the Low group.

One may argue that the effect of education only captures the effect of GDP per capita, and that our estimation results suggest that the degree of economic development, not education, influences the effect of mortality on per-capita income. We conduct the estimation reported in columns [1] and [2] in Table 4 to address this problem. The estimation uses not only education criterion but also log GDP per capita in 1940 as covariates. The result shows that education remains a significant predictor of regime membership after controlling for the effect of GDP per capita, whereas log GDP per capita is not a significant predictor after controlling for the effect of education.

The estimation reported in columns [3] and [4] in Table 4 uses not only education criteria but also demographic criteria as predictors of regime membership. The result shows that both criteria are significant predictors of regime membership. Countries with higher levels of education and lower population growth are more likely to be classified into the regime in which a decrease

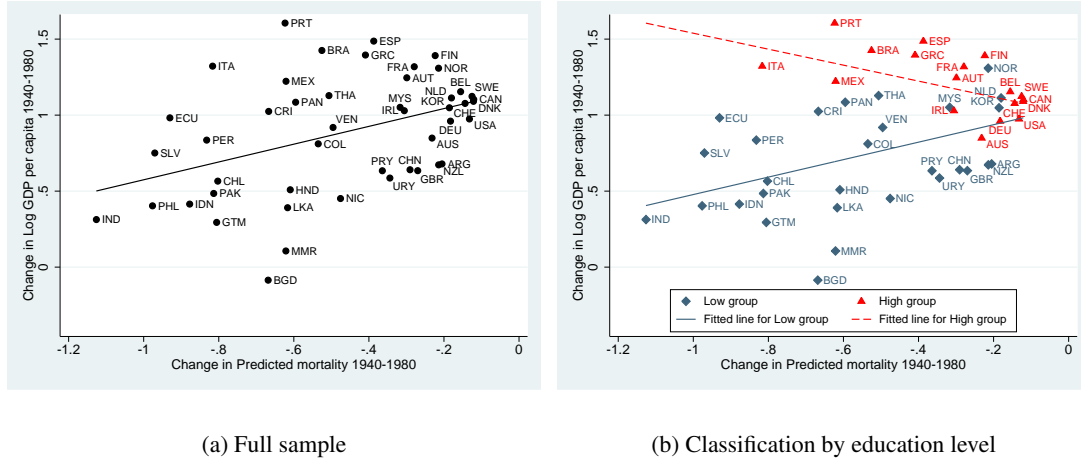


Figure 4: Change in log GDP per capita and change in predicted mortality, 1940-1980.

in mortality increases income per capita. This is consistent with the results of Cervellati and Sunde (2011a, 2011b).

The estimates are quantitatively similar across specifications. Overall, the findings support our hypothesis that education level plays an important role in the heterogeneous effects of mortality on income per capita, even after controlling for the effects of income level and demographic factors.

4 Conclusion

This paper developed a growth theory in which the replacement of physical capital accumulation by human capital accumulation is endogenous and mortality changes affect the replacement process. A testable prediction drawn from the theory is the heterogeneous effects of mortality on per-capita income among countries: the higher a country's education level, the more likely it is that a decrease in mortality will increase income per capita. We applied an FMM approach to the causal estimation of Acemoglu and Johnson (2007), and confirmed that the prediction is supported by the data. Although our analysis is mainly descriptive, our findings have relevant

policy implications. Simply introducing policies to improve health in poor regions where the education system is underdeveloped might decrease per-capita income. Policies that promote education and improve health are needed to put economies stuck in poverty onto a growth path.

The theoretical part of this study assumed mortality as an exogenous parameter and analysed the effect of exogenous changes in mortality. The empirical part of this study used an event where mortality changed exogenously (the international epidemiological transition) to test our theoretical predictions. Whether exogenous mortality has high validity depends on the situation. A decrease in mortality by new drug development is exogenous, especially for developing countries. However, a decrease in mortality via improved nutrition and sanitation is endogenous for many countries to some extent. Human capital accumulation seems to play a key role in life expectancy improvements. Endogenizing mortality in our framework in which the replacement of physical capital accumulation by human capital accumulation is endogenous would be an interesting direction for future research.

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