Mortality, Human Capital and Persistent Inequality*

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Final Version

Abstract

Available evidence suggests high intergenerational correlation of economic status and persistent disparities in health status between the rich and the poor. This paper proposes a mechanism linking the two. We introduce health capital into a two-period overlapping generations model. Private health investment improves the probability of surviving from the first period of life to the next and, along with education, enhances an individual's labor productivity. Poorer parents are of poor health, unable to invest much in reducing mortality risk and improving their human capital. Consequently, they leave less for their progeny. Despite convex preferences and technologies, initial differences in economic and health status may perpetuate across generations when annuities markets are imperfect.

KEYWORDS: Life Expectancy, Health, Human Capital, Income Distribution
JEL CLASSIFICATION: I12, I20, O15

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1 Introduction

This paper studies the dynamic interaction between health and wealth in a model of human capital investment. We show that, despite convex technology and preferences, health and wealth inequality may persist across generations interlinked by inheritance or human capital accumulation.

Intergenerational transmission of inequality and its persistence have received considerable attention in development economics in recent years. The current theoretical literature on persistent inequality focuses primarily on credit market imperfections and some form of technological non-convexity.[1] We explore, in contrast, an alternative mechanism operating through endogenous mortality. We postulate a positive relationship between the probability of survival and private health investment and show that, in the absence of perfect annuities markets, the resulting interplay between income and mortality can be instrumental in generating poverty traps by altering the incentives faced by poorer households.

Three types of empirical evidence motivate our work. First is the substantial evidence showing a high intergenerational correlation ($0.5-0.7$) of economic status in developed countries (Solon, 1992; Zimmerman, 1992; Mulligan, 1997). Comparable studies on developing countries are sparse, but the few that exist suggest an even more persistent intergenerational transmission of inequality.[2]

Our second piece of evidence draws from the empirical literature on individual patience rates. Lawrance (1991) and Samwick (1998) argue that individual patience rates are positively correlated with income. For example, Lawrance finds that rates of time preference range from 12% for families in the uppermost quintile of labor earnings to 19% for families in the lowest quintile, while Samwick estimates patience rates ranging from 4% at the topmost decile to 8% at the lowest. Related to this, Fuchs (1986) finds empirical support for

[1] See for example Banerjee and Newman (1993), Galor and Zeira (1993), Freeman (1996), Aghion and Bolton (1997), Picketty (1997), Maoz and Moav (1999), Ghatak and Jiang (2002), Mookherjee and Ray (2002, 2003). A different strand of this literature highlights the role of externalities associated with human capital formation. Examples include Benabou (1996), Durlauf (1996), Galor and Tsiddon (1997).

[2] Estimates reported in the literature typically come from a log-linear regression of son's earnings on father's earnings and other covariates. They are often hard to compare because while some studies look at multiyear measures of earnings, others, especially on developing countries, report estimates from short-run (hourly, monthly, annual) earnings due to data limitations. Some estimates from developing societies are 0.26 (Malaysia), 0.44 (South Africa), 0.54 (Mexico) and $0.58-0.75$ (Brazil) which are typically higher than "corresponding" estimates from developed countries. See Solon (2002) and the references therein for a survey of the international evidence; see also Behrman *et al.* (2001) and Guimaraes and Veloso (2003).

a positive association between individual patience rates and health. In our paper, endogenous mortality implies a positive relationship between discount rates and both health and economic status.

The final evidence we rely on is the well-documented correlation between adult health and socioeconomic status. The age-adjusted relative risk of death in the US, for example, is about two to three times higher for people at the bottom of the income distribution compared to those at the top (Sorlie *et al.*, 1995). Mortality rates in the poorer areas of Porto Alegre, Brazil, are about 75% higher than in the richer areas (World Bank, 1993). Women from the poorest quintile of Bangladesh's population are twice as likely to have low body mass index compared to women from the richest quintile (World Bank, 2003).

In examining the determinants of mortality among whites and non-whites in the US, Menchik (1993) finds that the mortality difference between different ethnic groups reduces dramatically when variables related to economic status are controlled for. Wilkinson (1996) reports that blue-collar workers are more likely to die from eighty percent of the eighty most common causes of death than white-collar workers. Income is found to have strong and significant effects on mortality even after controlling for age, race, urbanization, sex, education and lifestyle factors in Lantz *et al.* (1998).[3]

We argue here that not only does poverty shorten the lifespan of a single generation, but when successive generations are linked through economic variables, mortality risk faced by the current generation has far reaching impact on the welfare of its progeny. Our key innovation is the endogenization of mortality risk, which in turn implies endogenous discounting when annuities markets are imperfect. It is this implied endogeneity of the time preference that lies at the heart of our persistence result. Poorer people, with a lower probability of survival, have a higher rate of time preference and therefore care less for any economic activity that generates utility in the future. When there exists an intergenerational link (in whatever form) that relates children's well-being to their parents' future utility/earnings, endogenous time preference contributes to the transmission of poverty across generations.

Our argument is presented in terms of a two-period overlapping generations model with 'warm glow' altruism (Yaari, 1965, Andreoni, 1989, Galor and Zeira, 1993) where parents leave a part of their earnings to their children as bequest (or alternatively, as *inter vivos* transfers in their adulthood).[4] The probability of survival from the first period of life to the

[3] Deaton (2003) provides an overview of this research. Recent work by Cutler and Glaeser (2005), however, argues that differential health behavior across individuals is due to genetics, not income.

[4] The presence of a (warm glow) bequest motive is by no means necessary for our results. An appendix presents an alternative formulation where 'selfish' parents invest in their future productivity which is partially

next depends on privately incurred health expenditure. We show that a parent's low income status transmits to her descendants through two distinct channels. First, as discussed above, endogenous mortality implies poorer people discount the future more heavily. Since bequests are typically left at the end of one's lifetime (and *inter vivos* transfers generally made in the latter part of a parent's life), a greater discount rate for poorer households implies that not only do they leave less, they are also likely to leave a lower *proportion* of their earnings to their offsprings. Effectively bequests (or *inter vivos* transfers) become a luxury good for poorer households and a convex bequest function gives rise to threshold effects and poverty traps.[5]

Secondly, in the presence of endogenous mortality, income and wealth shocks become correlated with wealth levels. When parents die prematurely, their children inherit their accumulated first period wealth as accidental bequests. When parents live for their entire two periods of life, they leave a part of their lifetime earnings as intended bequests. Since accidental bequests are typically lower than intended ones, a parent's premature death constitutes a wealth shock for the offspring. Premature death of the parent is, of course, a chance event, but its probability is endogenous. Since the poor face a higher probability of premature death, the household distribution of income will exhibit a greater concentration of mass on the tails – children from poorer households are more likely to stay poor (since they mostly receive accidental bequests) and vice versa for children from richer households. Thus endogenous mortality alone can generate strong (short- and medium-run) persistence in our framework.[6]

The paper underscores the crucial role of health in explaining earning differentials and inequality across households over generations. The benchmark model, as described above, focuses on the impact of health on longevity. But apart from enhancing longevity, investment in health capital has other positive effects, notably on labor productivity. The link between productivity and health is multidimensional. Investment in health could directly enhance productivity and work capacity by increasing nutrient intakes. Furthermore, better nutrition and improved health during the early period of one's life enhances cognitive abilities,

inherited by their children. De Nardi's (2003) calibration exercises, however, show parental bequest motives match the actual US and Swedish wealth concentration much better than productivity inheritance alone. Hence in the text we focus on the former.

[5] That a convex bequest function can generate poverty traps has been shown by Moav (2002). Instead of assuming such a bequest function, as Moav does, we show how it arises endogenously. For empirical support of the convexity of bequest functions see Menchik and David (1983).

[6] The two channels of persistence described here are quite independent. The latter mechanism would operate even if bequest functions were to be concave.

indirectly affecting productivity.[7] With this in mind, we augment our benchmark model to allow for a positive link between health and productivity and examine its implications for intergenerational persistence.

The paper contributes to the emerging literature on the developmental implications of health and mortality. Related to our work, Glomm and Palumbo (1993) analyze a life cycle model where the survival probability is determined by health capital via nutritional investment. Their focus is, however, on how consumption patterns respond to various types of income paths when credit markets are absent. Ray and Streufert (1993) consider a model of persistent inequality where the probability of survival is endogenous. But their persistence result derives from the assumed non-convexity in labor supply capacity,[8] while we highlight the crucial role played by endogenous mortality. A different type of non-convexity is at the heart of Galor and Mayer's (2002) analysis of health and education. They posit that individuals require a minimum level of health (basic needs) for successful education and show how this minimal health requirement gives rise to poverty traps in the presence of credit constraints.

Finally, in this paper, we abstract from the role that nutrition and early child development play in the intergenerational transmission, factors especially important in developing societies. The Copenhagen Consensus, for example, notes that a sixth of the billion people suffering from malnourishment around the world are pre-school children (Behrman *et al.*, 2004). Such starvation early in life has lifelong consequences via susceptibility to diseases, low worker productivity and low quality of life. A simple way to think about childhood malnourishment in our framework is to allow for heterogeneity across households in *initial* health capital that is correlated with wealth (presumably as a consequence of parental investment in child nutrition). This will only exacerbate the transmission mechanism we propose: children born of poorer parents will not only receive proportionately lower transfers but will be also less productive in youth, less able to invest in their health as adults and hence, less inclined to invest in their human capital and transfer wealth to their offsprings.

The paper proceeds as follows. The basic intuition is presented in the next section using a static two-period model. The corresponding dynamic analysis is presented in section 3 showing how endogenous mortality gives rise to persistent inequality. Section 4 extends the analysis to incorporate productivity-enhancing effects of health and education. Section 5

[7] Behrman (1993, 1996) explores in detail such health-productivity linkages. Also see Strauss and Thomas (1997, 2000).

[8] The Ray-Streufert result does not change if one replaces the assumption of endogenous survival by exogenous survival.

discusses the extent to which our results generalize in the presence of capital accumulation and externalities in health investment. We conclude the discussion in section 6.

2 A Two-Period Problem

To fix ideas and intuition, we begin with a static two-period model describing the choices of a representative individual. We illustrate how health investment, future consumption and bequests become luxury goods in the presence of endogenous mortality.

An individual potentially lives for two periods, "youth" and "old-age". She lives in youth for sure but may or may not survive into old-age, the probability of which depends on her health capital.

At the end of her youth, the individual gives birth to a single offspring, before she realizes her mortality shock. She is altruistic toward her offspring, deriving a "warm-glow" from the bequests that she leaves in her old-age. We call such bequests *intended*, and distinguish them from *unintended* or *accidental* ones. When the agent does not survive into old-age, her assets are passed on to her offspring as unintended bequests. The agent does not derive any utility from such unintended bequests.

We assume that the individual works for unit time in both periods of life, earning a wage income w. In the first period, she also inherits a certain amount of wealth (W), as either intended or unintended bequest. Her total first period income is then given by $w + W$, out of which she consumes, saves/borrows (in a perfect credit market) and invests in health. At the beginning of her old-age, an individual who invested h in her health, realizes a mortality shock with probability $1 - \phi(h)$. If she survives, she invests her youthful savings in capital, earning an exogenously given gross return R on it. When she does not survive, her savings passes on to her offspring. Since such unintended bequests do not generate any utility for the parent, it does not concern us here (we address its implications for intergenerational dynamics in the next section).

Surviving individuals allocate their second period labor and capital income between old-age consumption and bequests. Thus the representative agent faces the following two budget constraints,

$$c_1 + s + h = w + W, \tag{1}$$

$$c_2 + b = Rs + w, \tag{2}$$

the second one being relevant only when she survives into old age.

Assuming zero utility from death and zero utility from accidental bequest, the expected lifetime utility of this individual is given by

$$U = u(c_1) + \phi(h)\left[u(c_2) + \theta v(b)\right], \tag{3}$$

which she maximizes subject to (1) and (2).

We assume that u and v are concave and twice differentiable. An individual's chance of surviving beyond the first period depends upon her health investment undertaken during youth, h, according to

$$\phi = \phi(h) \in [0,1],$$

where the probability function satisfies

$$\phi(0) = 0\,, \quad \phi' > 0, \quad \phi'' \leq 0 \quad \text{and} \quad \lim_{h\to\infty} \phi(h) = \bar{\phi} \leq 1. \qquad \text{(Assumption 1)}$$

To economize on notation we ignore any subjective discounting.

For analytical convenience we assume that

$$u(c) = \frac{c^{1-\sigma}}{1-\sigma},\ v(b) = \frac{b^{1-\sigma}}{1-\sigma} \text{ with } \sigma \in (0,1). \tag{4}$$

We restrict σ to be less than 1 for two reasons. In the first place, we require the substitution effect of an interest rate change to dominate the income effect to generate a "well-behaved" savings function. Additionally, since utility from death is zero, having $\sigma > 1$ would perversely yield lower utility from being alive and consuming a positive bundle of (c_1, c_2, b).[9]

For the probability function we choose

$$\phi(h) = \begin{cases} ah^{\varepsilon}, & \text{if } h \in [0, \hat{h}] \\ \overline{\phi}, & \text{otherwise} \end{cases} \tag{5}$$

where $\hat{h} \equiv (\bar{\phi}/a)^{1/\varepsilon}$ and impose the parametric restriction

$$\sigma > \varepsilon. \qquad \text{(Assumption 2)}$$

[9]In general we require $u(c)$ and $v(b)$ to be positive so that being alive is associated with a positive marginal utility from health investment.

But this is not an unusual assumption in the variable time preference literature. With variable time preferences, present and future variables often enter the utility function in a complementary fashion. A cardinal assumption is usually required to ensure that lifetime utility obeys other standard assumptions and the optimization problem remains economically meaningful. Obstfeld (1990), for example, posits lifetime utility to be $u(c_1) + \beta(c_1)u(c_2)$ and requires $u(c) < 0$ so that the marginal lifetime utility of c_1 is positive.

Consider first optimal solutions in the range over which ϕ is increasing in health investment. First order necessary conditions associated with s, h and b are:

$$u'(c_1) = R\phi(h)u'(c_2) \Rightarrow (c_1)^{-\sigma} = R\phi(h)(c_2)^{-\sigma}, \tag{6}$$

$$u'(c_1) = \phi'(h)\left[u(c_2) + \theta v(b)\right] \Rightarrow (1-\sigma)(c_1)^{-\sigma} = \phi'(h)\left[(c_2)^{1-\sigma} + \theta b^{1-\sigma}\right], \tag{7}$$

and

$$u'(c_2) = \theta v'(b) \Rightarrow b = \beta c_2, \tag{8}$$

where $\beta \equiv \theta^{1/\sigma}$. From these it follows that

$$c_2 = \gamma R h \tag{9}$$

where $\gamma \equiv (1-\sigma)/[\varepsilon(1+\beta)]$. Now consider the two-period budget constraint

$$c_1 + h + \frac{c_2}{R} + \frac{b}{R} = y,$$

where $y \equiv (1+1/R)w + W$ is the present discounted value of lifetime income. Substituting (6), (8) and (9) into this, we obtain

$$\psi(h) \equiv h + \gamma h\left[1 + \beta + \frac{R^{1-1/\sigma}}{\phi(h)^{1/\sigma}}\right] = y \tag{10}$$

which implicitly defines health expenditure as a function of income, $h = \eta_0(y)$.

We are interested in how h responds to y to determine whether or not it is a luxury good. Since c_2 and b are linearly related to h via (8) and (9), when health is a luxury good so are future consumption and bequests.

>From (10) above

$$\psi' = 1 + \gamma\left[1 + \beta + \frac{R^{1-1/\sigma}}{\phi^{1/\sigma}}\left(1 - \frac{\varepsilon}{\sigma}\right)\right] \text{ and } \psi'' = -\gamma R^{1-1/\sigma}\phi^{-1/\sigma-1}\left(1 - \frac{\varepsilon}{\sigma}\right)\phi'.$$

A necessary condition for the concavity of ψ is $\sigma > \varepsilon$ which also ensures that $\psi(h)$ is increasing. Under this assumption,

$$\frac{\partial \eta_0}{\partial y} = \frac{1}{\psi'} > 0 \text{ and } \frac{\partial^2 \eta_0}{\partial y^2} = -\left(\psi'\right)^{-3}\psi'' > 0.$$

Since the income-expansion path for health is convex, health is a luxury good.

A little intuition on the relationship between σ and ε will be helpful. There are essentially two ways the individual can increase her future utility: by increasing health which raises the weight attached to future utility and by increasing c_2 (and b). Now consider the effect of an increase in y through the Euler equation $u'(c_1) = \phi(h)u'(c_2)$. This obviously increases present consumption c_1 so that $u'(c_1)$ falls. But when h and c_2 are normal goods, expenditures on them will increase too, which means while $u'(c_2)$ will fall ϕ will rise. Thus in order to satisfy the Euler equation, $u'(c_2)$ must fall proportionately more than the increase in $\phi(h)$. This is precisely what happens when $\sigma > \varepsilon$.

Indeed the parametric condition that $\sigma > \varepsilon$, as specified under Assumption 2, has important implications for the optimization exercise. In Appendix A we show that the second order sufficient conditions are satisfied as long as Assumption 2 holds. Further, Assumption 2 is actually *necessary* to guarantee that first-period consumption is a normal good. To see this refer to equations (6) and (9):

$$c_1 = (R)^{-1/\sigma} \left[\frac{(1-\sigma)R}{(1+\beta)\varepsilon} \right] h \left[\phi(h)\right]^{-1/\sigma} \equiv \tau h \left[\phi(h)\right]^{-1/\sigma}$$

Differentiating with respect to y we obtain

$$\frac{dc_1}{dy} = \tau \left[\phi(h)\right]^{-1/\sigma} \left(1 - \frac{\varepsilon}{\sigma}\right) \frac{dh}{dy}$$

which is positive as long as $\sigma > \varepsilon$. That is, c_1 is a normal good *iff* $\sigma > \varepsilon$.

To fully characterize optimal choices, we also need to consider what happens for lifetime incomes $y > \hat{y}$ where $\hat{y}$ is defined by $\eta_0(\hat{y}) = \hat{h}$. Since the survival probability does not increase with health investment beyond $\hat{h}$, health investment is maintained at $\hat{h}$ for all such income levels and (c_1, c_2, b) determined optimally. The associated first order conditions

$$\begin{aligned} (c_1)^{-\sigma} &= \bar{\phi} R (c_2)^{-\sigma}, \\ (c_2)^{-\sigma} &= \theta b^{-\sigma}, \end{aligned}$$

give the closed form solutions

$$\begin{array}{cc} c_1 = \left[\frac{1}{1+\rho(1+\beta)}\right]\left(y - \hat{h}\right), & c_2 = \left[\frac{\rho}{1+\rho(1+\beta)}\right] R \left(y - \hat{h}\right) \\ b = \left[\frac{\beta\rho}{1+\rho(1+\beta)}\right] R \left(y - \hat{h}\right), & s = \left[\frac{\rho(1+\beta)}{1+\rho(1+\beta)}\right]\left(y - \hat{h}\right) - w/R \end{array} \tag{11}$$

where $\rho \equiv \left(\bar{\phi}\right)^{1/\sigma} R^{1/\sigma - 1}$. Thus in the complete characterization, optimal c_1, c_2 and b will be initially convex (for $y \leq \hat{y}$) and then linear (for $y > \hat{y}$) in lifetime income.

A few clarifications are required here. First, we have implicitly assumed that annuities markets do not exist. As Barro and Friedman (1977) show, if individuals facing uncertain lifetimes have access to actuarially fair annuities markets (Yaari, 1965), the effective discount rate is unaffected by the survival probability. For instance, suppose perfectly competitive annuities markets existed in our model and health choices were publicly observable. Then an individual of health h would face the interest factor $R/\phi(h)$. Maximization of (3) subject to the lifetime budget constraint

$$c_1 + h + \frac{\phi(h)}{R}c_2 + \frac{\phi(h)}{R}b = w + W + \frac{\phi(h)}{R}w$$

leads to the Euler equation $u'(c_1) = Ru'(c_2)$ which is independent of ϕ. It is straightforward to show that although health continues to be a luxury good, future consumption and bequests are necessities in this environment. Since the intergenerational transmission mechanism operates through bequests in our model, this means actuarially fair annuities markets eliminate the persistence result developed in section 3 below. In what follows, we assume that annuities markets are missing. More generally, we need them to be imperfect.

Secondly, following the usual practice in the literature (Fuster, 1999; De Nardi, 2003), we have assumed that parents do not derive utility from unintended bequests. A possible justification for such preferences is that parental altruism develops only when parents are physically present to witness their children's well-being. Alternatively one could interpret the bequests as *inter vivos* transfers that take place only if the parent is alive. However, apart from simplifying the analysis, this particular assumption about parental preferences does not have a significant bearing on our key result that endogenous discounting makes bequests a luxury good. We show in Appendix B that a similar conclusion follows even when we allow parents to derive utility from unintended bequests.

Finally, our persistence result does not depend on the specific transmission mechanism we use in this paper. For instance, we show in Appendix C how a similar persistence result can be obtained if generations were linked through human capital accumulation. In related work, Chakraborty and Das (2005) shows how persistence occurs when selfish parents invest in their children's education under a social arrangement where children support their parents in old age.

We now turn to a dynamic general equilibrium version of our two-period model where the luxury good nature of bequests gives rise to persistent inequality.

3 The Dynamic Economy

Consider a small, open, overlapping-generations economy that operates in a perfectly competitive world and faces a given world rate of interest. Time is discrete and infinite with $t = 0, 1, 2, \ldots \infty$.

3.1 Production

Every period the economy produces a single good that may be consumed or invested. The output is produced using physical capital (K) and efficiency units of labor (H). The technology for this, $F(K, H)$, is neoclassical and satisfies the usual Inada conditions.

In competitive product and factor markets, the economy-wide wage and interest rates are:

$$\begin{aligned} w_t &= f\left(\frac{K_t}{H_t}\right) - \left(\frac{K_t}{H_t}\right) f'\left(\frac{K_t}{H_t}\right) \\ r_t &= f'\left(\frac{K_t}{H_t}\right) - \delta, \end{aligned}$$

where f denotes the intensive-form technology and δ the depreciation rate on capital.

A constant world interest rate, $r_t = \bar{r}$, pins down the domestic physical to human capital ratio at $\bar{k} \equiv f'^{-1}(\bar{r} + \delta)$, which also fixes domestic wages at $\bar{w}$ per efficiency unit of labor. If H_t changes over time due to human capital accumulation, capital (K_t) flows in or out until physical capital per efficiency unit of labor returns to its previous level so that equilibrium wage and interest rates remain constant at $\bar{w}$ and $\bar{r}$ respectively. We denote the gross interest factor by $R \equiv 1 + \bar{r}$.

3.2 Preferences

Let us normalize the size of new borns to unity. The life cycle of a representative agent is already described in section 2. Thus, an agent born at t with wealth W_t, inherited either as intended or unintended bequests, maximizes her expected lifetime utility

$$U_t = u(c_t^t) + \phi(h_t)\left[u(c_{t+1}^t) + \theta v(b_{t+1})\right], \tag{12}$$

subject to the budget constraints,

$$c_t^t = \bar{w} + W_t - s_t - h_t, \tag{13}$$

$$c_{t+1}^t = \bar{w} + R s_t - b_{t+1}. \tag{14}$$

As before, u and v are concave and twice differentiable functions while Assumptions 1 and 2 hold.

Individuals differ only with respect to their wealth levels W_t^i. The distribution of wealth in a particular generation t is given by a cumulative distribution function $G_t(W)$ denoting the measure of individuals with wealth below W. The initial distribution G_0 is historically given.

The key idea outlined in the static model is that an individual's health capital h_t at the end of period t is the outcome of her private health investment, h_t, undertaken during that period. The important point is that such health investments, for example, net food intake (that is, nutrients available for cellular growth; see Fogel, 1993), personal hygiene, accessing clinical facilities and related medical expenditure, lower adult mortality risks. Since all individuals are born with identical amounts of health capital (normalized to one), any difference in health status within a particular cohort is entirely due to differences in health investment.[10]

We analyze the case where all individuals save positive amounts in youth. Similar to section 2, a person surviving into old-age leaves some of her second period earnings to her offspring as intended bequest. But when she does not survive, her savings passes on to the offspring. The offspring, however, cannot invest her parent's savings on the market. Thus if the parent dies prematurely, leaving behind savings s_t, the offspring receives an unintended bequest amounting to s_t, instead of Rs_t.[11]

3.3 Optimization

For expositional purposes, once again it will be convenient to work with CES preferences (4) and the probability function (5). The first-order conditions corresponding to (4) and health choices in the range $[0, \hat{h}]$ are now given by

$$\begin{aligned} c_{t+1}^t &= [\phi(h_t)R]^{1/\sigma} c_t^t, & (15) \\ (1-\sigma)(c_t^t)^{-\sigma} &= \phi'(h_t)\left[(c_{t+1}^t)^{1-\sigma} + \theta(b_{t+1})^{1-\sigma}\right], & (16) \\ b_{t+1} &= \beta c_{t+1}^t. & (17) \end{aligned}$$

Solving, we get the optimal consumption, savings and bequest decisions as functions of health investment:

$$c_{t+1}^t = \left[\frac{(1-\sigma)R}{(1+\beta)\varepsilon}\right] h_t \tag{18}$$

[10]More generally $\phi^i = \Phi(h^i, \kappa^i)$, where κ^i is the innate health capital individual-i is born with. Here we have $\kappa^i = 1$ for all i, and define $\phi(h^i) \equiv \Phi(h^i, 1)$. This is obviously a simplifying assumption since offsprings do inherit part of their parent's health status naturally, through birth. Such intergenerational health transmission would only reinforce our persistence results.

[11]This assumption reflects the fact that obtaining access to parental wealth in case of accidental death of parent is a time-consuming and costly affair; it involves the legal costs of establishing one's claim to the property. For convenience we assume that such legal procedure does not involve any direct monetary cost, only the indirect cost of foregone interest income.

$$b_{t+1} = \beta \left[\frac{(1-\sigma)R}{(1+\beta)\varepsilon} \right] h_t \tag{19}$$

$$s_t = \left[\frac{1-\sigma}{\varepsilon} \right] h_t - \frac{\bar{w}}{R}. \tag{20}$$

$$c_t^t = (R)^{-1/\sigma} \left[\frac{(1-\sigma)R}{(1+\beta)\varepsilon} \right] h_t[\phi(h_t)]^{-1/\sigma} \tag{21}$$

Finally, we determine optimal health investment by substituting (20) and (21) into (13):

$$h_t = W_t + \left[\frac{1+R}{R} \right] \bar{w} - \left[\frac{1-\sigma}{\varepsilon} \right] h_t - (R)^{-1/\sigma} \left[\frac{(1-\sigma)R}{(1+\beta)\varepsilon} \right] h_t[\phi(h_t)]^{-1/\sigma}$$

or,

$$\Gamma(h_t) \equiv \mu_0 h_t + \mu_1 h_t[\phi(h_t)]^{-1/\sigma} = W_t + \left[\frac{1+R}{R} \right] \bar{w}, \tag{22}$$

where $\mu_0 \equiv 1 + (1-\sigma)/\varepsilon > 0$ and $\mu_1 \equiv (R)^{-1/\sigma} [(1-\sigma)R/\{(1+\beta)\varepsilon\}] > 0$. Equation (22) implicitly defines health investment as a function of wealth which we denote by $h_t = \eta_0(W_t)$.

Equations (19) and (20) imply that

$$b_{t+1} = \left(\frac{\beta}{1+\beta} \right) \left(\frac{1-\sigma}{\varepsilon} \right) R\eta_0(W_t), \tag{23}$$

and

$$s_t = \left(\frac{1-\sigma}{\varepsilon} \right) \eta_0(W_t) - \frac{\bar{w}}{R}, \tag{24}$$

both of which are increasing in wealth (see below). Longer-lived individuals are more patient decision-makers. Consequently, they are more willing to save and leave larger bequests. Since health investment and wealth are positively related, the implication is that offsprings of wealthier parents also enjoy prosperous and healthier lives.

Note however that the equations (23) and (24) represent the optimal bequest and savings functions of the household only for $\eta_0(W_t) \in [0, \hat{h}]$. In other words, these equations are optimal choices for households with wealth in the range $[0, \hat{W}]$, where $\hat{W} = \eta_0^{-1}(\hat{h})$. As we have seen in the previous section, for any $W > \hat{W}$, health investment is maintained at $\hat{h}$, and the corresponding bequests and savings become linear in wealth.

Using equations (23), (24), and (11) we can now characterize optimal bequest and savings for the entire wealth distribution as follows:

$$b_{t+1} = \Psi_1(W_t) \equiv \begin{cases} \left(\frac{\beta}{1+\beta}\right) \left(\frac{1-\sigma}{\varepsilon}\right) R\eta_0(W_t), & \text{for } W \leq \hat{W} \\ \left(\frac{\beta}{1+\beta}\right) \left(\frac{\rho(1+\beta)}{1+\rho(1+\beta)}\right) R \left(W_t + \bar{w} + \dfrac{\bar{w}}{R} - \hat{h}\right), & \text{for } W > \hat{W} \end{cases} \tag{25}$$

and

$$
s_t = \Psi_2(W_t) \equiv \begin{cases} \left(\frac{1-\sigma}{\varepsilon}\right)\eta_0(W_t) - \dfrac{\bar{w}}{R}, & \text{for } W \leq \hat{W} \\ \left(\frac{\rho(1+\beta)}{1+\rho(1+\beta)}\right)\left(W_t + \bar{w} + \dfrac{\bar{w}}{R} - \hat{h}\right) - \dfrac{\bar{w}}{R}, & \text{for } W > \hat{W} \end{cases} \tag{26}
$$

The slope and the curvature of these two functions depends crucially on the optimal health investment

$$
h_t = \eta(W_t) \equiv \begin{cases} \eta_0(W_t), & \text{for } W_t \leq \hat{W} \\ \hat{h}, & \text{for } W_t > \hat{W} \end{cases} \tag{27}
$$

Proposition 1 *Given Assumption 2, optimal health investment* $h = \eta(W)$ *satisfies the following properties:*

(i) $\eta(0) > 0$ *as long as* $\bar{w} > 0$,

(ii) $\partial\eta(W)/\partial W > 0$ *for* $W \leq \hat{W}$, *while* $\partial\eta(W)/\partial W = 0$ *for* $W > \hat{W}$,

(iii) $\partial^2\eta(W)/\partial W^2 > 0$ *for* $W \leq \hat{W}$, *while* $\partial^2\eta(W)/\partial W^2 = 0$ *for* $W > \hat{W}$.

The technical part of this proposition is proved in Appendix D. Figure 1 illustrates the optimal health investment for different wealth levels. The upward sloping part of the diagram depicts the relationship represented by (22). It shows that for wealth level $W \leq \hat{W}$, $\Gamma(0) = 0$ and $\Gamma(h)$ is monotonically increasing and concave in health. For a wealth level above $\hat{W}$, $\Gamma(h)$ ceases to represent optimal health investment which is now fixed at $\hat{h}$, represented by the vertical section.

Note here that the linear part of intended and unintended bequest lines both have slopes less than one but the intended line is steeper.

3.4 Wealth Dynamics

Given optimal bequest and savings decisions from (25) and (26) above, intergenerational wealth dynamics follows a nonlinear Markov process

$$
W_{t+1}^i = \Psi(W_t^i) \equiv \begin{cases} \Psi_1(W_t^i), & \text{with probability } \phi(h(W_t^i)), \\ \Psi_2(W_t^i), & \text{otherwise,} \end{cases} \tag{28}
$$

where W_0^i is historically given by the initial distribution G_0.

We impose a restriction on the degree of altruism here. The term $\bar{w}/R$ that enters the optimal savings decision in equation (24) is the present value of second-period labor income, and would be zero if individuals were to work only in the first period of their lives. We assume that individuals are altruistic enough in the sense that,

$$\theta > (1/\bar{r})^{\sigma}. \qquad \text{(Assumption 3)}$$

This assumption ensures that even when individuals do not work in the second period, $\Psi_1(0) > \Psi_2(0)$. As long as Assumption 3 is satisfied, individuals at all wealth levels are economically better off with their parents surviving instead of dying prematurely. It also implies that the intended bequest line Ψ_1 is steeper than unintended bequests Ψ_2.[12]

We now turn to an analysis of the wealth dynamics characterized by (28) above. As a by-product of this dynamics, we will learn the extent to which health status and mortality risks persist across generations. Observe first that endogenous mortality introduces a 'stickiness' to intergenerational economic status. Since they do not invest much in health, poorer individuals are more likely to die prematurely. Consequently they leave their offsprings lower assets than they otherwise would, and this generates a tendency for the progeny to remain mired in poverty and ill-health.

Contrast this to an environment where mortality is exogenously specified, independent of health and wealth. Such a stochastic model would have trouble accounting for observed correlations of intergenerational income and consumption. For example, using US data, Mulligan (1997) finds that estimates on these intergenerational correlations are in the range $0.7-0.8$, much higher than we would expect from a model with exogenously driven stochastic shocks to income or wealth (see also Piketty, 2000). Our model of intergenerational wealth transmission effectively endogenizes these wealth and income shocks: the propensity to suffer from adverse shocks is higher, the poorer are one's parents. Endogenous mortality risk, in other words, increases the correlation between parental and child economic and health status.

Exactly how persistent intergenerational wealth inequality is depends on whether or not (28) possesses a globally unique invariant distribution. This, in turn, is decided by the curvature of $\eta(W)$, which through equations (23) and (24), determines the shape of intended and accidental bequests. We illustrate the wealth dynamics in Figure 2.

[12] De Nardi (2003) shows that, for the US and Sweden, intended bequests are quantitatively more important compared to accidental bequests in replicating key features of the wealth distribution. It is important to point out that our persistence result still holds if $\theta < (1/\bar{r})^{\sigma}$. It is easy to show that $\theta < (1/\bar{r})^{\sigma}$ implies that $\Psi_2(W) < \Psi_1(W)$ below a certain wealth level and $\Psi_2(W) > \Psi_1(W)$ above it. As a result, parental death will be associated with a negative income shock for children belonging to poorer households and a positive shock for wealthier households. Since $\Psi_2(W)$ and $\Psi_1(W)$ are both convex-concave, the expected bequest curve will still be convex-concave. But it will be less steep, which means wealth dispersion will be smaller than what we analyze later in this section.

Figure 2 depicts Ψ_1 and Ψ_2 and the corresponding expected bequest line Ψ^E defined by

$$\Psi^E(W_t^i) \equiv \phi(h(W_t^i))\Psi_1(W_t^i) + [1 - \phi(h(W_t^i))]\Psi_2(W_t^i)$$

under the assumption $\bar{\phi} < 1$. In both Figures 2(a) and (b) the expected bequest line is non-concave since both Ψ_1 and Ψ_2 are. But the non-concavity does not matter for long-run dynamics in Figure 2(a) compared to (b). In Fig 2(a), all dynasties converge to the unimodal long-run distribution G_∞ shown in the lower half of the figure. In Fig 2(b), long-run outcomes are history-dependent. Families that start out with sufficient wealth, above $\bar{W}$, converge to a distribution on the support $[\bar{W}_H^2, \bar{W}_H^1]$. Those who do not, converge to $[\bar{W}_L^2, \bar{W}_L^1]$. The wealth level $\bar{W}$ acts as a threshold, determining exactly how persistent intergenerational health and wealth outcomes would be in the long-run.

Long-run persistence in wealth and health status obtains in our model due to the dependence of health and mortality on economic status. It may be argued in this context that medical innovations in the recent past has led to substantial mortality reduction across the world, and especially in developing countries, quite independent of income improvements. Indeed, in so far as medical innovations lead to exogenous reduction in adult mortality, it would undermine the importance of the persistence mechanism highlighted here. It is easy to see that exogenous improvements in the probability of survival would shift up the transition mappings in (28), allowing the poor to accumulate wealth, and reduce mortality, faster than before.

Significant longevity improvements could, for example, alter the wealth mapping from what Figure 2(b) shows to something similar to Figure 2(a). While this gets rid of any non-ergodicity, along the convergence path we would still observe a tendency for poorer households to stay poor relative to wealthier ones. It is equally important to note that the impact of medical innovations (essentially working through public health systems) has been far more visible in instances of sharp declines in infant and child mortality rates, rather than in adult mortality (World Bank, 1993). To the extent that exogenous medical innovations has had limited impact on adult mortality rates, the income-health link emphasized here plays a particularly significant role in generating persistence in developing societies.

4 Health, Education and Labor Productivity

Our analysis of the interdependence between health and economic outcomes in the previous section differs from existing studies in one respect. Much recent work on health has looked at its implication for labor productivity, rather than for the saving and investment decisions we analyzed.

Robert Fogel's work, in particular, has highlighted the historical importance of nutrition and living standard improvements for economic development. Fogel (1997), for instance, estimates that nutritional improvements alone contributed about $20-30\%$ of the growth in British per capita income during 1780-1979 by bringing the impoverished into the labor force and by increasing the energy level available for work.

Microeconometric studies on developing countries, reviewed by Strauss and Thomas (1998), show health status and nutritional intake to be important for an individual's functionality and ability to work productively. A positive relationship between health and earnings holds across education levels, suggesting a health human capital effect that complements the importance of education in this regard. There is also extensive biomedical evidence pointing to the effects of health, particularly child health and nutrition early in life, on educational attainments. For example, Seshadri and Gopaldas (1989), find that iron deficiency significantly affects children's cognitive development and school performance in India. In another study, Kvalsvig *et al.* (1991), present evidence on how parasitic infections combine with malnourishment to impair cognitive processes.[13]

Two papers in particular, Ray and Streufert (1993) and Galor and Mayer (2002), illustrate how health as a form of human capital can be instrumental in generating persistent inequality. Both papers introduce a non-convexity on the technology side. In Ray and Streufert, the non-convexity between food intake and labor productivity arises from biological mechanics: for undernourished individuals food intake only goes towards maintaining necessary bodily functions without improving the capacity to work. This, in turn, gives rise to nutrition-based efficiency wages and equilibrium unemployment among the poor and malnourished. Strauss and Thomas (1998) point out, however, that empirical support for such an efficiency wage hypothesis is weak.

The non-convexity in Galor and Mayer takes the form of a conditional complementarity between health and education. Health and education are complementary inputs but only for individuals of a minimum health status; those who are malnourished and of poor health do not benefit from education since ill-health impairs their cognitive development, attentiveness and mental capacity. In the presence of credit markets imperfections, such threshold effects of health readily give rise to intergenerational persistence of inequality.

In both these papers, the poor face lower returns from investing in health human capital

[13] See Behrman (1996) for a critical review of this literature.

than do the rich. It is clear that such technologies would only amplify the persistence mechanism we highlighted in sections 2 and 3 above. In this section, we incorporate an effect of health investment on labor productivity over and above its consequences for adult mortality. But instead of assuming a technological non-convexity, we show that our persistence result is robust even when poorer individuals face higher returns from investing in health and education human capitals.

To be more specific, suppose that an individual works in both periods of her life, being endowed with time endowments of one unit in each period. Individuals do not differ in their education or health capital in the first period, but their labor income in the subsequent period depends on human capital investment earlier in life. For an individual who invests h_t in health and e_t in her education, second-period income is given by $\bar{w}[1+\zeta(e_t,h_t)]$. The technology ζ is convex and satisfies $\zeta(0,h)=\zeta(e,0)=0$.

We choose the parametric specification

$$\zeta(e_t,h_t)=\bar{A}e_t^{\alpha}h_t^{1-\alpha},$$

where $\bar{A}>0$ and $\alpha\in(0,1)$. Note that this Cobb-Douglas function implies that returns to health (education) are infinite for an individual who invests nothing in it (education). Such a scenario will, in general, give a greater incentive to the poor to invest in health, thereby generating a tendency towards convergence.

Let us once again assume that preferences and probability functions are identical to those given by (4) and (5). Individuals now maximize their expected lifetime utility (12) subject to the two budget constraints

$$\begin{aligned} c_t^t &= \bar{w}+W_t-s_t-h_t-e_t, && (29)\\ c_{t+1}^t &= \bar{w}[1+\zeta(e_t,h_t)]+Rs_t-b_{t+1}. && (30) \end{aligned}$$

Individuals choose the vector (s_t,e_t,h_t,b_{t+1}) to maximize lifetime utility, necessary and sufficient conditions for which are given by

$$\begin{aligned} c_{t+1}^t &= [\phi(h_t)R]^{1/\sigma}c_t^t, && (31)\\ c_{t+1}^t &= \left[\phi(h_t)\alpha A\left(\frac{h_t}{e_t}\right)^{1-\alpha}R\right]^{1/\sigma}c_t^t, && (32)\\ (1-\sigma)(c_t^t)^{-\sigma} &= \left[\phi'(h_t)\left[(c_{t+1}^t)^{1-\sigma}+\theta b_{t+1}^{1-\sigma}\right]+A(1-\sigma)(1-\alpha)\phi(h_t)\left(\frac{e_t}{h_t}\right)^{\alpha}(c_{t+1}^t)^{-\sigma}\right] && (33)\\ b_{t+1} &= \beta c_{t+1}^t, && (34) \end{aligned}$$

where $A \equiv \bar{A}\bar{w}$. We follow our previous strategy in characterizing optimal health investment. Note first that (31) and (32) imply that returns to savings (R) and educational investment ($\bar{w}\zeta_e$) are equalized, so that,

$$e_t = \left[\frac{\alpha A}{R}\right]^{1/(1-\alpha)} h_t, \tag{35}$$

implying that healthier individuals invest more in education.

Now using (34) and (35) in (33), we obtain

$$c_{t+1}^t = \gamma_0 h_t, \tag{36}$$

where

$$\gamma_0 \equiv \frac{1-\sigma}{\varepsilon}\left[\frac{1}{1+\beta}\right]\left[R - A(1-\alpha)\left(\frac{\alpha A}{R}\right)^{\alpha/(1-\alpha)}\right].$$

Evidently c_{t+1}^t is positive as long as

$$R > (\alpha)^{\alpha}(1-\alpha)^{1-\alpha}A, \tag{Assumption 4}$$

which we henceforth maintain. This assumption is necessary to derive any meaningful solution to the optimization problem and can be reduced to an intuitive condition.

For the Cobb-Douglas human capital technology above, the elasticities of labor productivity with respect to education and health are simply α and $1-\alpha$ respectively. Assumption 4 can then be written out as

$$R > \left[\frac{e\zeta_e}{\zeta}\right]^{\alpha}\left[\frac{h\zeta_h}{\zeta}\right]^{1-\alpha} A = (\bar{w}\zeta_e)^{\alpha}(\bar{w}\zeta_h)^{1-\alpha}$$

As noted above, we also have $R = \bar{w}\zeta_e$ at an interior optimum. Hence the inequality above implies that in equilibrium

$$R = \bar{w}\zeta_e > (\bar{w}\zeta_e)^{\alpha}(\bar{w}\zeta_h)^{1-\alpha} \Leftrightarrow R = \bar{w}\zeta_e > \bar{w}\zeta_h.$$

To see why this condition is required, note that each of the first order conditions (31) – (33) equates the marginal cost of foregoing current consumption to the marginal benefit from alternative means of investment. It is clear that if the restriction above is not satisfied, then the marginal benefit from health investment (which comprises of the utility gains from longer lifetimes in addition to $\bar{w}\zeta_h$) will dominate gains from saving and educational investment. Individuals would not invest in education or save in that case.

>From the second-period budget constraint, we obtain using (36),

$$s_t = \gamma_1 h_t - \frac{\bar{w}}{R}, \tag{37}$$

where $\gamma_1 \equiv \left[(1+\beta)\gamma_0 - A(\alpha A/R)^{\alpha/(1-\alpha)}\right]/R$. Likewise, from (31) and (36),

$$c_t^t = \gamma_2 h_t [\phi(h_t)]^{-1/\sigma} \tag{38}$$

where $\gamma_2 \equiv (R)^{-1/\sigma}\gamma_0$. Using these relations in the first-period budget constraint gives us

$$\Pi(h_t) \equiv \gamma_3 h_t + \gamma_2 h_t [\phi(h_t)]^{-1/\sigma} = W_t + \left[\frac{1+R}{R}\right]\bar{w}, \tag{39}$$

where $\gamma_3 \equiv 1 + \gamma_1 + (\alpha A/R)^{1/(1-\alpha)} > 0$ and $\gamma_2 > 0$. Define the wealth level that satisfies the equation above for $h_t = \hat{h}$ as $\tilde{W}$. Then, for all $W \leq \tilde{W}$, (39) defines optimal health investment as a function of wealth, $h_t = \nu_0(W_t)$.

As before, we separately characterize optimal solutions for individuals with $W > \tilde{W}$ since for these individuals health investment only affects labor productivity. Generally when there multiple investment opportunities (here, health, education and savings), an arbitrage condition ensures that marginal returns from these investments are equated. In this case, however, Assumption 4 implies that when survival probability is constant, the marginal return from savings dominates the marginal return from health investment. This means, individuals with wealth above $\tilde{W}$ maintain health investment at $h_t = \hat{h}$ and choose $e_t = (\alpha A/R)^{1/(1-\alpha)}\hat{h}$ to equalize returns from savings and education. For all such individuals labor productivity is $1 + [\alpha A/R]^{\alpha/(1-\alpha)}\hat{h} \equiv z$ in the second period of their lives.

Closed-form optimal choices in this case are

$$c_t^t = \left(\tfrac{1}{1+\rho(1+\beta)}\right)\left[W_t + (1+z/R)\bar{w} - \tilde{h}\right], \qquad c_{t+1}^t = \left(\tfrac{\rho}{1+\rho(1+\beta)}\right) R\left[W_t + (1+z/R)\bar{w} - \tilde{h}\right]$$
$$s_t = \left(\tfrac{1}{1+\rho(1+\beta)}\right)\left[W_t + (1+z/R)\bar{w} - \tilde{h}\right] - \bar{w}z/R$$
$$b_{t+1} = \left(\tfrac{\rho\beta}{1+\rho(1+\beta)}\right) R\left[W_t + (1+z/R)\bar{w} - \tilde{h}\right]$$

where $\tilde{h} \equiv [1 + (\alpha A/R)^{1/(1-\alpha)}]\hat{h}$. As before, optimal savings and bequest decisions are linear in wealth for $W > \tilde{W}$. Optimal health investment depends on wealth according to

$$h = \nu(W) \equiv \begin{cases} \nu_0(W), & \text{for } W \leq \tilde{W} \\ \hat{h}, & \text{for } W > \tilde{W} \end{cases} \tag{40}$$

Proposition 2 *Optimal health investment, $h = \nu(W)$ defined by (40) satisfies the following properties:*

(i) $\nu(0) > 0$ whenever $\bar{w} > 0$,

(ii) $\partial\nu(W)/\partial W > 0$ for $W \leq \tilde{W}$, while $\partial v(W)/\partial W = 0$ for $W > \tilde{W}$,

(iii) $\partial^2\nu(W)/\partial W^2 > 0$ *for* $W \leq \tilde{W}$*, while* $\partial^2\nu(W)/\partial W^2 = 0$ *for* $W > \tilde{W}$*.*

Proof. See Appendix E.

Optimal bequests and savings are now given by

$$b_{t+1} = \Phi_1(W_t) \equiv \begin{cases} \left[\frac{\beta}{1+\beta}\right]\left[\frac{1-\sigma}{\varepsilon}\right] R\nu_0(W_t), & \text{for } W \leq \tilde{W} \\ \left[\frac{\beta}{1+\beta}\right]\left[\frac{\rho(1+\beta)}{1+\rho(1+\beta)}\right] R\left[W_t + (1+z/R)\bar{w} - \tilde{h}\right], & \text{for } W > \tilde{W} \end{cases}, \tag{41}$$

and

$$s_t = \Phi_2(W_t) \equiv \begin{cases} \left[\frac{1-\sigma}{\varepsilon}\right]\nu_0(W_t) - \dfrac{\bar{w}}{R}, & \text{for } W \leq \tilde{W} \\ \left[\frac{1}{1+\rho(1+\beta)}\right]\left[W_t + \left(1+\frac{z}{R}\right)\bar{w} - \tilde{h}\right] - \frac{\bar{w}z}{R}, & \text{for } W > \tilde{W} \end{cases}, \tag{42}$$

where Φ_1 and Φ_2 are both increasing functions of wealth.

Once again these individual wealth transition mappings determine the evolution of the wealth distribution according to

$$W_{t+1}^i = \Phi(W_t^i) = \begin{cases} \Phi_1(W_t^i), & \text{with probability } \phi(h(W_t^i)), \\ \Phi_2(W_t^i), & \text{otherwise,} \end{cases} \tag{43}$$

given the initial distribution G_0. Since health investment is a luxury good as before, it is clear how this mapping can give rise to long-run persistence as in the benchmark model of section 3.

5 Extensions

Up to this point health outcomes have depended solely on private investments. Moreover, we shut down standard channels of capital accumulation by assuming the domestic capital per efficiency unit of labor is pinned down by the constant world interest rate. Both these assumptions simplified our analysis by allowing aggregate wealth dynamics to mimic the time-invariant dynastic wealth evolution depicted by equation (28) or (43). We now briefly consider what happens when we allow for meaningful interactions across economic agents. Two scenarios are especially relevant: when there are real externalities in health investment, and when pecuniary externalities arise from the effect of capital accumulation on factor prices.

Human capital formation is typically associated with real externalities (Galor and Tsiddon, 1997). In the context of health, private expenditures in sanitation, personal hygiene, vaccination and nourishment can have significant public health effects through their impact on the incidence of diseases and the population size at risk of contacting these diseases. When such externalities are present, the survival probability for a particular individual depends not only her health expenditure incurred privately, but also on the average level of health investment undertaken in her community.

Suppose for a generation-t individual, the probability of survival is now given by $\hat{\phi}(h_t^i) = \varphi(\bar{h}_t)\phi(h_t^i)$, where $\bar{h}_t = \int_0^\infty \eta(W_t^i)dG_t(W)$ is average health investment at time t.[14] Health externalities are assumed to generate a simple threshold effect

$$\varphi(\bar{h}) = \begin{cases} \varphi_0, & \text{if } \bar{h} < h_c, \\ \varphi_1, & \text{if } \bar{h} \geq h_c, \end{cases} \tag{44}$$

with $\varphi_1 > \varphi_0$, similar to education capital externalities analyzed by Galor and Tsiddon (1997). More generally one would expect these externalities to be increasing, at least over a range, with average health status and/or investment in the economy. Using a general $\phi(h)$ function, the expected bequest line for the i-th dynasty is represented by

$$\hat{\Psi}^E(W_t^i) \equiv \hat{\phi}(\eta(W_t^i))\hat{\Psi}_1(W_t^i) + [1 - \hat{\phi}(\eta(W_t^i))]\hat{\Psi}_2(W_t^i). \tag{45}$$

It is easy to see that this expected bequest line will now comprise of two disjoint segments, one corresponding to $\varphi(\bar{h}_t) = \varphi_0$, and the other to $\varphi(\bar{h}_t) = \varphi_1$, with the former lying below the latter. Starting from an initial wealth level W_0^i, dynasty i moves along the lower expected bequest line as long as $\bar{h}_t < h_c$, and jumps to the upper one when $\bar{h}_t$ exceeds h_c.

Allowing for this kind of threshold externalities in the benchmark model of Section 3 immediately enhances the possibility of a poverty trap. To see how, suppose in the absence of externality the wealth dynamics is represented by Figure 2. Let us now introduce a threshold effect as specified above and assume that for the initial distribution, we have

$$\bar{h}_0 = \int_0^\infty \eta(W_0^i)dG_0(W) < h_c, \tag{Assumption 5}$$

that is, all the dynasties are initially on the lower expected bequest line, corresponding to φ_0. If φ_0 is small enough, this line would have three points of intersection with the 45^{o} line, $(\bar{W}_L^3, \bar{W}_L^2, \bar{W}_L^1)$. On the other hand, if φ_1 is sufficiently large, the upper expected bequest line corresponding to φ_1 intersects the 45^{o} line only once, at $\bar{W}_H$. Figure 3 depicts such a scenario.

[14]The size of each generation has been normalized to unity.

If the average health capital in this economy remains below the threshold level for all t, then of course the economy remains on the lower expected bequest line forever, and wealth dynamics is similar to that illustrated by Figure 2(b). Conversely, if over time the average health capital becomes large enough, all dynasties eventually move to the upper expected bequest line. There is no long-run polarization and everyone enjoys the same healthy state irrespective of their initial wealth. Whether the economy remains stuck at the lower expected bequest line with polarization, or can escape the poverty trap and move to the upper expected bequest line (leaving everybody better-off in the long run) depends crucially on the initial health and wealth distribution.

Note the critical role played by the initial distribution here: it now determines not just the *degree* of polarization, but the very *possibility* of polarization. It is obvious that the economy would escape the poverty trap if a sufficient number of people enjoy health greater than h_c , so that the average health capital exceeds this threshold. Let the steady state health levels associated with the lower expected bequest line be $\eta(\bar{W}_L^3)$ and $\eta(\bar{W}_L^1)$ respectively, and let $\eta(\bar{W}_L^3) < h_c < \eta(\bar{W}_L^1)$. It then follows that the greater is initial inequality, the lower is the proportion of people who eventually attain a health capital greater than h_c, and therefore lower are the chances for this economy to escape the poverty trap. Thus inequality bites in more than one sense.

The mechanism elaborated here assumes special significance in the context of developing countries, particularly those in the tropics, characterized by large-scale incidence of infectious diseases (World Bank, 1993, Murray and Lopez, 1996). Indeed, the mechanism suggests that even controlling for income distribution, exogenous health distributions (working through the externality) could give rise to Pareto inferior outcomes that leave everyone worse-off in the long run (compared to the alternative scenario). This indicates a key role for public health policy in these countries, a point we return to in the concluding section.

A second kind of interaction among economic agents operates through the effect of capital accumulation on equilibrium factor prices. In a closed economy version of the model presented in section 3, wages and interest rates would depend upon the economy's capital-labor ratio, and thus, on the entire wealth distribution at any point in time. Aggregate wealth dynamics would no longer be a simple replica of individual wealth dynamics as we would have to take into account the effects of endogenously determined wage and interest rates on wealth accumulation and investment.

First consider how investment contributes to the accumulation of physical capital. Recall that only the savings of individuals who survive into old-age are invested. The aggregate physical capital stock is hence determined according to:

$$K_{t+1} = \int \phi(W_t^i) s(W_t^i) dG_t.$$

Since both the young and old work, and since corresponding to any wealth level W_t^i a fraction $\phi(W_t^i)$ survive into old-age (by the law of large numbers), capital per worker is given by

$$k_{t+1} = \frac{K_{t+1}}{1 + \int \phi(W_t^i) dG_t}.$$

The wealth distribution G_t determines factor prices (w_{t+1}, r_{t+1}) via this equation. In the process, it impacts the future wealth distribution G_{t+1} through a dynamic system similar to (43) above. The resulting interplay between factor prices and the wealth distribution can give rise to complicated non-linear dynamics, as noted by Banerjee and Newman (1993) and Aghion and Bolton (1997). Without going into the specifics of such problems, we only conjecture to what extent results presented earlier generalize to this case.

Here too the initial wealth distribution turns out to be of significance. Higher are typical wealth levels, the greater is health investment and longer lifespans induce faster accumulation of physical capital. When a greater number of individuals survive into old-age, it increases the size of the labor force. This tends to dilute the effects of capital accumulation by exerting downward pressure on the capital intensity of production. Under the assumption that the investment effect dominates, a more favorable wealth distribution leads to high k, and thus, higher wages, w. Rising wages tend to push up poorer individuals faster by allowing for greater health (and educational) investment.

A second channel works in tandem: capital deepening lowers returns to saving and investment. Relative to physical capital investment, health and education become more attractive investment opportunities (see equation (35)), eliciting even greater human capital investments.

Both effects are, however, predicated on a favorable wealth distribution. If the dynamics corresponding to the initial distribution G_0, for instance, were to look like Figure 2(b), factor price movements might not mitigate tendencies for parental status to persistently affect health investment in the progeny. Specifically, as dynasties move either to the lower or upper stationary distributions, there may be a tendency for the non-ergodic dynamics to be reinforced – with a relatively unequal wealth distribution, fewer people save and invest as much as is required to significantly increase wages and lower interest rates, thereby hindering upward mobility among the poorer households.

6 Conclusion

We have analyzed a model of intergenerational mobility operating through the accumulation of health capital. We show that initial health inequality can be a key factor in explaining the observed persistence in wealth and income inequality across households. We underline the crucial role of health capital in the process of development. Unlike other forms of human capital, the role of health is unique: not only does it generate positive externalities, it also affects individuals' mortality risks, thereby altering their incentives. As we demonstrate here, this latter aspect of health capital has important implications for intergenerational mobility and equality.

The source of persistence we discussed is especially important for developing economies. Though medical innovations in the last half-century have substantially reduced infant and child mortality rates in these countries, adult mortality continues to be high in much of the developing world. Adult mortality risk, the probability of a typical 15-year old dying before age 60, in Sub-Saharan Africa, for instance, is about three times higher than in the established market economies (World Bank, 1993, Table A.5). Persistent poverty in these countries can therefore be attributed to the associated higher mortality risks.

The paper highlights a key role for the public health system. Apart from providing cheap health care to the poor, public health expenditure in developing countries can significantly reduce mortality risks by providing a healthier environment in terms of better hygiene, sanitation, availability of clinical facilities etc.

The composition of public health expenditure is especially important in this context. The efficiency of a public health system depends on how broad its reach is. It is here that many developing countries face serious problems. As the World Bank (1993) notes, even though economically backward classes tend to suffer from usually treatable infectious diseases, public money is often spent, instead, on relatively 'more expensive' disease treatments (cancer surgery, for example) that mainly benefits the wealthy. Government spending on health goes disproportionately to the rich in the form of free or cheap health care and insurance subsidies. In Indonesia, for example, "government subsidies to health for the richest 10 percent of households in 1990 were .... almost three times the subsidies going to the poorest 10 percent" (World Bank, 1993, p. 4). In addition, the quality of care available for the poor is typically low.

In the presence of inefficient public health systems, income remains the underlying determinant of adult mortality rates, especially for the poor and malnourished. Apart from quality-of-life considerations, the economic consequences of health inequality we examined provide a strong cause for public health systems to effectively devote more resources towards the poor.

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Appendix

Appendix A: *Second Order Conditions*

We restrict the proof of the second order conditions to our specific functional choices, (4) and (5), in the text. Accordingly we use $u(b)$ instead of $v(b)$. Optimization exercise of the representative household:

$$\text{Maximize } U = u(c_1) + \phi(h)[u(c_2) + \theta u(b)]$$
$$\text{subject to } c_1 + h + \frac{c_2}{R} + \frac{b}{R} \leqslant y;\ c_1, h, c_2, b \geqq 0.$$

Note that the objective function $U(c_1, c_2, h, b)$ is continuous in the budget set. Also note that the constraint set defined by the inequality constraints is non-empty, closed and bounded (compact). Hence by the Weierstrass Theorem, there exist a minima and a maxima for this objective function defined within the constraint set. Moreover, any such maxima must satisfy the Kuhn-Tucker conditions. We find that there exist two possible solutions to the Kuhn-Tucker conditions, an interior solution and a corner solution, both which are candidates for the maxima. However we show that under Assumption 2 ($\sigma > \varepsilon$), the interior solution is associated with higher lifetime utility and is therefore the optimal solution.

The Lagrangian for this optimization problem is:

$$\mathcal{L} = u(c_1) + \phi(h)[u(c_2) + \theta u(b)] + \lambda \left[y - c_1 - h - \frac{c_2}{R} - \frac{b}{R} \right] + \mu_1 c_1 + \mu_2 h + \mu_3 c_2 + \mu_4 b$$

and the associated Kuhn-Tucker first-order necessary conditions are:

(1) $u'(c_1) - \lambda + \mu_1 = 0$

(2) $\phi'(h)[\ u(c_2) + \theta v(b)] - \lambda + \mu_2 = 0$

(3) $\phi(h)u'(c_2) - \dfrac{\lambda}{R} + \mu_3 = 0$

(4) $\phi(h)u'(b) - \dfrac{\lambda}{R} + \mu_4 = 0$

(5) $\lambda \left[y - c_1 - h - \dfrac{c_2}{R} - \dfrac{b}{R} \right] = 0; \lambda \geqslant 0;\ c_1 + h + \dfrac{c_2}{R} + \dfrac{b}{R} \leqslant y$

(6) $\mu_1 c_1 = 0; \mu_1 \geqslant 0; c_1 \geqslant 0$

(7) $\mu_2 h = 0; \mu_2 \geqslant 0; h \geqslant 0$

(8) $\mu_3 c_2 = 0; \mu_3 \geqslant 0; c_2 \geqslant 0$

(9) $\mu_4 b = 0; \mu_4 \geqslant 0; b \geqslant 0$

It follows from the Kuhn-Tucker conditions that (i) the budget constraint is always binding; (ii) in an optimal solution $c_1 \neq 0$. (The latter conclusion follows from $\lim_{c\to 0} u'(c) = \infty$). Thus there are two possible solutions to the above Kuhn-Tucker conditions. One is a corner solution given by: $c_1 = y$, $h = 0$, $c_2 = 0$, $b = 0$. The other one is the interior solution denoted by: $c_1 = c_1^*(y) > 0$, $h = h^*(y) > 0$, $c_2 = c_2^*(y) > 0$ and $b = b^*(y) > 0$. In the first case the total life-time utility is given by $u(y)$. In the latter case it is given by $u(c_1^*) + \phi(h^*)[u(c_2^*) + \theta u(b^*)]$. We show that if $\sigma > \varepsilon$, lifetime utility associated with the latter solution is greater then lifetime utility associated with the former.

The proof follows in two steps. First we choose an arbitrary interior allocation point on the budget line and show that lifetime utility associated with this interior allocation point is higher than the corner solution. Then we argue that therefore the utility associated with the interior optimal point must be greater than that of the corner solution too.

Proof: We know that the utility associated with the corner solution is $u(y)$. Let us now construct an alternative allocation whereby an infinitesimal amount δ is deducted from the first period consumption and is distributed among h, c_2 and b such that $h = c_2/R = b/R = \delta/3$. In this new allocation the lifetime utility of the household will be $u(y-\delta) + (1+\theta)\phi(\delta/3)u(R\delta/3)$. Let $\delta \to 0$. Then the fall in the aggregate utility due to this reduction in the first period consumption is $u'(y)$. On the other hand, the increase in aggregate utility due to the increase in h, c_2 and b is $\lim_{\delta\to 0}(1+\theta)[\phi'(\delta/3)u(R\delta/3) + R\phi(\delta/3)u'(R\delta/3)]$.

We next show that $\lim_{\delta\to 0}(1+\theta)[\phi'(\delta/3)u(R\delta/3)+R\phi(\delta/3)u'(R\delta/3)] = \infty$. For this all we need to show is $\lim_{x\to 0}[\phi'(x)u(Rx)+R\phi(x)u'(Rx)] = \infty$. Recall that given our specification of u and ϕ, $u(Rx) = (Rx)^{1-\sigma}/(1-\sigma)$; $u'(Rx) = (Rx)^{-\sigma}$, while $\phi(x) = ax^{\varepsilon}$ and $\phi'(x) = a\varepsilon x^{\varepsilon-1}$. Hence $\lim_{x\to 0}[\phi'(x)u(Rx) + R\phi(x)u'(Rx)] = aR^{1-\sigma}\left(\frac{\varepsilon}{1-\sigma} + 1\right)\lim_{x\to 0} x^{\varepsilon-\sigma} = \infty$, provided $\sigma > \varepsilon$.

This implies that the marginal gain in aggregate utility due to increase in h, c_2 and b dominates the marginal loss ($u'(y)$, which is finite), and hence the household is better off with such a reallocation on the budget line.

Indeed if the household is better-off at such an interior allocation compared to the corner solution, then it must attain even higher utility at the interior solution (which is the 'best' of all interior points). So the interior solution to the Kuhn-Tucker conditions must be the optimal solution.

Appendix B: *Utility from Accidental Bequests*

Suppose preferences are given by

$$u(c_1) + \phi(h)u(c_2) + \phi(h)\theta v(b) + [1 - \phi(h)]\theta v(s)$$

where $u(c) = c^{1-\sigma}/(1-\sigma)$ and $v(x) = x^{1-\sigma}/(1-\sigma)$ for $x = b,\ s$. The last two terms define expected utility from bequests: bequests are given by parental savings s with probability $1-\phi$ and equal to planned transfers b with probability ϕ. The parent's intensity of altruism (θ) is same for both types of bequests.

For algebraic simplicity we consider individuals who work only in youth and face the budget constraints

$$c_1 + h + s = W + w, \;\; c_2 + b = Rs. \tag{46}$$

Note that s subsumes life-insurance contracts that pay a gross return R to the individual in the event of survival, and a gross return 1 to the offspring in case of death. The first-order conditions for (s, h, b) are

$$\begin{aligned}
(c_1)^{-\sigma} &= \phi R(c_2)^{-\sigma} + (1-\phi)\theta s^{-\sigma} && (47)\\
(1-\sigma)(c_1)^{-\sigma} &= \phi' \left[(c_2)^{1-\sigma} + \theta b^{1-\sigma}\right] - \phi'\theta s^{1-\sigma} && (48)\\
(c_2)^{-\sigma} &= \theta b^{-\sigma} && (49)
\end{aligned}$$

>From (46) and (49)

$$s = \left(\frac{1+\beta}{R}\right) c_2,$$

where $\beta \equiv \theta^{1/\sigma}$. Using this in (47)

$$(c_1)^{-\sigma} = (p + q\phi)(c_2)^{-\sigma}, \tag{50}$$

where $p \equiv \theta[(1+\beta)/R]^{-\sigma}$ and $q \equiv R - p$. Note that $q > 0$ by Assumption 2. Also, from (48),

$$(1-\sigma)(c_1)^{-\sigma} = \left(\frac{1+\beta}{R}\right) q\phi'(c_1)^{1-\sigma} \tag{51}$$

Defining $\lambda(h) \equiv p + q\phi(h)$, from (50) and (51) we obtain

$$c_2 = \frac{(1-\sigma)R}{(1+\beta)} \frac{\lambda(h)}{\lambda'(h)}.$$

Similarly,

$$s = (1-\sigma)\frac{\lambda(h)}{\lambda'(h)} \;\; \text{and} \;\; c_1 = \frac{(1-\sigma)R}{(1+\beta)} \frac{[\lambda(h)]^{1-1/\sigma}}{\lambda(h)}.$$

Optimal health investment is derived by substituting these relationships into the first-period budget constraint:

$$\psi(h) \equiv \frac{(1-\sigma)R}{(1+\beta)} \frac{[\lambda(h)]^{1-1/\sigma}}{\lambda(h)} + (1-\sigma)\frac{\lambda(h)}{\lambda'(h)} + h = W + w.$$

We proceed to show that $\psi(h)$ is increasing and concave for $\sigma > \varepsilon$. First note that the term

$$\frac{\lambda}{\lambda'} = \frac{1}{\varepsilon a q} h^{1-\varepsilon}(p + aqh^{\varepsilon}) \equiv \hat{p} h^{1-\varepsilon} + h/\varepsilon$$

is concave. Hence, it is sufficient to show that the first term for $\psi(h)$,

$$\psi_1(h) \equiv \frac{(1-\sigma)R}{(1+\beta)} \left(\frac{p + aqh^{\varepsilon}}{\varepsilon a q h^{\varepsilon-1}}\right) [\lambda(h)]^{-1/\sigma} \equiv \chi h^{1-\varepsilon} [\lambda(h)]^{1-1/\sigma}$$

is concave. We have,

$$\psi_1' = (1-\varepsilon)\chi h^{-\varepsilon} \lambda^{1-1/\sigma} - \chi \left(\frac{1}{\sigma} - 1\right) h^{1-\varepsilon} \lambda^{-1/\sigma} \lambda'.$$

For this to be positive, we need

$$1 - \varepsilon > \left(\frac{1}{\sigma} - 1\right) \varepsilon_{\lambda}$$

where $\varepsilon_{\lambda} \equiv h\lambda'/\lambda =$ monotonically increases from 0 to $\varepsilon q\bar{\phi}/(p + q\bar{\phi}) < \varepsilon$ as h goes from 0 to $\hat{h}$. Since $\varepsilon_{\lambda} < \varepsilon$, note that $\sigma > \varepsilon$ guarantees that $\psi_1' > 0$. Now,

$$\begin{aligned} \psi_1'' &= -\chi\varepsilon(1-\varepsilon)h^{-\varepsilon-1}\lambda^{1-1/\sigma} - 2\chi(1-\varepsilon)\left(\frac{1}{\sigma} - 1\right) h^{-\varepsilon} \lambda^{-1/\sigma} \lambda' \\ &\quad -\chi \left(\frac{1}{\sigma} - 1\right) h^{1-\varepsilon} \left[-\frac{1}{\sigma} \lambda^{-1/\sigma - 1} (\lambda')^2 + \lambda^{-1/\sigma} \lambda''\right]. \end{aligned}$$

The last term on the right hand side is positive, the first two are negative. So

$$\begin{aligned} \psi_1'' &< 0 \Rightarrow \varepsilon(1-\varepsilon)h^{-\varepsilon-1}\lambda^{1-1/\sigma} + 2(1-\varepsilon)\left(\frac{1}{\sigma} - 1\right) h^{-\varepsilon} \lambda^{-1/\sigma} \lambda' \\ &> \left(\frac{1}{\sigma} - 1\right) h^{1-\varepsilon} \left[\frac{1}{\sigma} \lambda^{-1/\sigma - 1} (\lambda')^2 - \lambda^{-1/\sigma} \lambda''\right] \end{aligned}$$

Divide both sides by $h^{-\varepsilon}\lambda^{-1/\sigma}\lambda'$ to get

$$\varepsilon(1-\varepsilon)\frac{\lambda}{h\lambda'} + 2(1-\varepsilon)\left(\frac{1}{\sigma} - 1\right) > \left(\frac{1}{\sigma} - 1\right) h \left[\frac{1}{\sigma}\frac{\lambda'}{\lambda} - \frac{\lambda''}{\lambda'}\right]$$

which can be rewritten as:

$$\frac{\varepsilon(1-\varepsilon)}{\varepsilon_{\lambda}} > \left(\frac{1}{\sigma} - 1\right) \left[\frac{1}{\sigma}\frac{h\lambda'}{\lambda} - \frac{h\lambda''}{\lambda'} - 2(1-\varepsilon)\right] = \left(\frac{1}{\sigma} - 1\right) \left[\frac{\varepsilon_{\lambda}}{\sigma} - (1-\varepsilon)\right]$$

Since $\varepsilon_\lambda < \varepsilon$, we have $\varepsilon(1-\varepsilon)/\varepsilon_\lambda > 1-\varepsilon$. So it is sufficient to show that

$$1-\varepsilon > \left(\frac{1}{\sigma}-1\right)\left[\frac{\varepsilon_\lambda}{\sigma}-(1-\varepsilon)\right] \Rightarrow 1-\varepsilon > \left(\frac{1}{\sigma}-1\right)\varepsilon_\lambda$$

Again, since $\varepsilon_\lambda < \varepsilon$, it is sufficient to show that

$$1-\varepsilon > \left(\frac{1}{\sigma}-1\right)\varepsilon \Rightarrow \sigma > \varepsilon$$

which is true by assumption. Since $\psi_1'' < 0$ over $[0, \hat{h}]$, this means, $h(W)$ and hence, c_2 and h, are luxury commodities with convex Engel curves.

Appendix C: *Persistence through Human Capital Inheritance*

We show here the existence of persistence trap when successive generations are linked through human capital inheritance, not bequests. Suppose human capital is the only form of asset. A person is endowed with $\hat{z}$ amount of human capital in the first period of his life, which generates a first period income $y_1 = \bar{w}\hat{z}$, where $\bar{w}$ is the constant return per unit of human capital (skilled labour). First period investment in education (e) generates aggregate skill level $f(e)$, ($f' > 0$; $f'' \leq 0$) and second period income $\bar{w}f(e)$. Without any loss of generality, let us normalize $\bar{w}$ to unity.

Individuals maximize

$$u(c_1) + \phi(h)u(c_2)$$

subject to

$$c_1 + h + e = y_1,\ c_2 = f(e).$$

As in the text, we assume that $u(c) = c^{1-\sigma}/(1-\sigma)$ where $\sigma \in (0,1)$, and $\phi(h) = ah^{\varepsilon}$, which is assumed to be less than unity within the relevant range.

First order conditions associated with e and h are:

$$(c_1)^{-\sigma} = f'(e)\phi(h)(c_2)^{-\sigma} \tag{52}$$

and

$$(1-\sigma)(c_1)^{-\sigma} = \phi'(h)(c_2)^{1-\sigma} \tag{53}$$

>From (52) and (53), it follows that

$$c_2 = \left(\frac{1-\sigma}{\varepsilon}\right) f'(e)h \tag{54}$$

Using (54) in the period 2 budget constraint

$$f(e) = \left(\frac{1-\sigma}{\varepsilon}\right) f'(e)h \Rightarrow h = \left(\frac{\varepsilon}{1-\sigma}\right)\frac{1}{\epsilon_f}e \tag{55}$$

where $\epsilon_f \equiv ef'(e)/f(e)$ is the elasticity of f .

>From period 1 budget constraint, using (53) and (55)

$$[f'(e)\phi(h)]^{-1/\sigma}\left(\frac{1-\sigma}{\varepsilon}\right)f'(e)h+h+e=y_1$$

$$\Rightarrow\left[f'(e)\phi\left(\left(\frac{\varepsilon}{1-\sigma}\right)\frac{1}{\epsilon_f}.e\right)\right]^{-1/\sigma}f(e)+\left(\frac{\varepsilon}{1-\sigma}\right)\frac{1}{\epsilon_f}.e+e=y_1$$

This equation implicitly defines the optimal education as a function of income: $e=\xi(y_1)$. To simplify things, we assume a linear skill-technology, $f(e)=Ae$, $A>1$ so that $\epsilon_f=1$. Hence the equation above reduces to

$$\psi(e)\equiv\left[\phi\left(\frac{\varepsilon}{1-\sigma}e\right)\right]^{-1/\sigma}A^{1-1/\sigma}e+\left(\frac{\varepsilon}{1-\sigma}\right)e+e=y_1$$

Then,

$$\psi'(e)=A^{1-1/\sigma}\phi^{-1/\sigma}\left[1-\frac{\varepsilon}{\sigma}\right]+\left(\frac{\varepsilon}{1-\sigma}\right)+1$$

$$\psi''(e)=-\frac{1}{\sigma}A^{1-1/\sigma}\phi^{-1/\sigma-1}\left(1-\frac{\varepsilon}{\sigma}\right)\phi'\frac{\varepsilon}{1-\sigma}$$

Note that ψ is increasing and concave as long as $\varepsilon<\sigma$. Then we have

$$\xi'(y_1)=\frac{de}{dy_1}=\frac{1}{\psi'}>0$$

and

$$\xi''(y_1)=\frac{d}{dy_1}\left(\frac{de}{dy_1}\right)=-\frac{1}{(\psi')^2}\psi''\frac{de}{dy_1}=-\frac{1}{(\psi')^3}\psi''>0$$

Therefore, $\xi(y_1)$ is convex in y_1, implying that education is a luxury good.

An intergenerational link can be brought into this static framework by positing that offsprings inherit a fraction δ of their parent's accumulated skills. Implicitly we are assuming a three-period life cycle where a representative agent potentially lives for childhood, youth and old-age. She lives for sure for the first two periods but surviving to old-age is uncertain and depends on health investment in youth. Children are born at the beginning of the second period of parent's life. For simplicity let us assume that agents consume nothing during childhood and only learn from their parents. Since a better educated parent can impart knowledge more efficiently, the child's acquired knowledge (skill) is positively related to the parental skill level. Once again for simplicity suppose that this relationship is linear, as represented by the fraction δ. Then the inherited (initial) skill level of a young adult born at $t-1$ would be δz_{t-1}, where z_t denotes the aggregate skill level, accumulated over the lifetime, by an individual born at time $t-1$. The intergenerational dynamics of human capital accumulation will be governed by:

$$z_t = Ae_t = A\xi(y_1) \ = A\xi(\delta z_{t-1}).$$

Since $\xi(\bullet)$ is a convex function, under suitable parameter values, it can intersect the 45° line more than once, generating multiple equilibria and persistence.

Appendix D: *Proof of Proposition 1*

Equation (22) defines optimal health investment as $h_t = \eta_0(W_t)$ for $W_t \in [0, \hat{W}]$. From Figure 1, as long as $\bar{w} > 0$, $\eta_0(0) > 0$. Totally differentiating (22) we get

$$\frac{\partial \eta_0}{\partial W} = \frac{1}{\mu_0 + \mu_1 \phi^{-1/\sigma}(\sigma - \varepsilon)/\sigma} \tag{A.1}$$

and

$$\frac{\partial^2 \eta_0}{\partial W^2} = \left[\frac{1}{\mu_0 + \mu_1 \phi^{-1/\sigma}(\sigma - \varepsilon)/\sigma}\right]^2 \frac{\sigma - \varepsilon}{\sigma^2} \mu_1 \phi^{-1/\sigma - 1} \phi' \frac{\partial \eta_0}{\partial W}.$$

Clearly when $\sigma > \varepsilon$, $\partial\eta_0/\partial W > 0$ and $\partial^2\eta_0/\partial W^2 > 0$, so that $\eta(W)$ is convex for $W \in [0, \hat{W}]$. Finally, since $\eta(W)$ takes a constant value $\hat{h}$ for $W > \hat{W}$, we must have $\partial\eta/\partial W = 0 = \partial^2\eta/\partial W^2$.

Appendix E: *Proof of Proposition 2*

Straightforward differentiation of (39) gives us

$$\frac{\partial \nu_0}{\partial W} = \frac{1}{\gamma_3 + \gamma_2 \phi^{-1/\sigma}(\sigma - \varepsilon)/\sigma} \tag{A.2}$$

and

$$\frac{\partial^2 \nu_0}{\partial W^2} = \left[\frac{1}{\gamma_3 + \gamma_2 \phi^{-1/\sigma}(\sigma - \varepsilon)/\sigma}\right]^2 \frac{\sigma - \varepsilon}{\sigma^2} \gamma_2 \phi^{-1/\sigma - 1} \phi' \frac{\partial \nu_0}{\partial W}.$$

When $\sigma > \varepsilon$, $\partial\nu_0/\partial W > 0$ and $\partial^2\nu_0/\partial W^2 > 0$. Constancy of ϕ (and h) for $W > \tilde{W}$ ensures that both the first and second derivatives of $\nu(W)$ are zero for $W > \tilde{W}$.

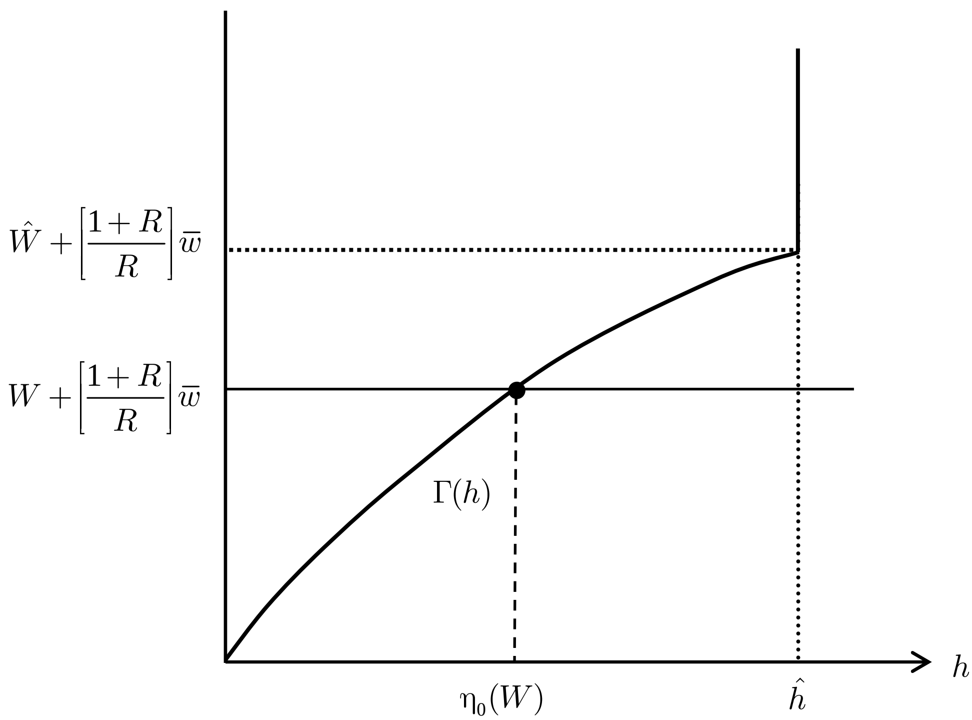

Figure 1: Optimal Health Investment

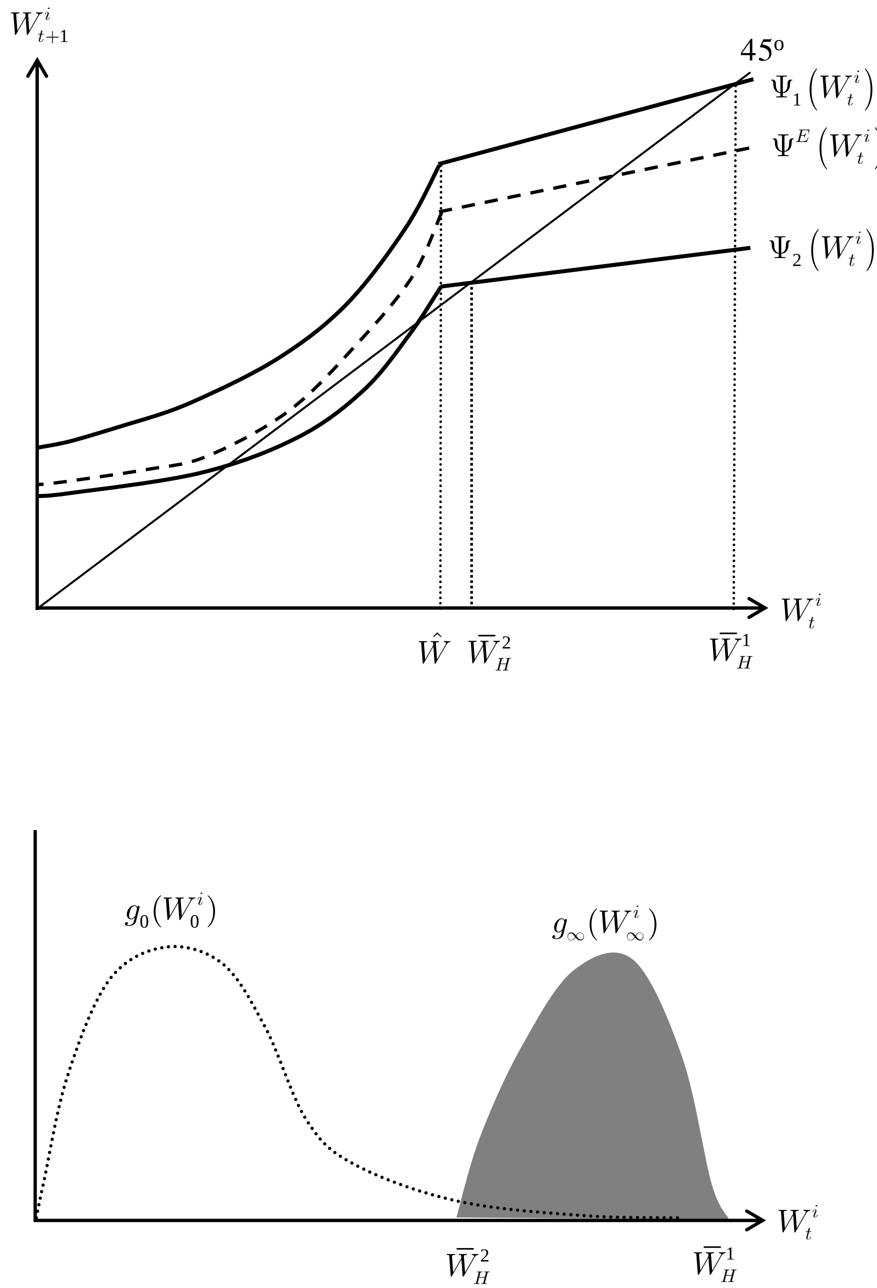

Figure 2(a): Wealth Dynamics: Convergence

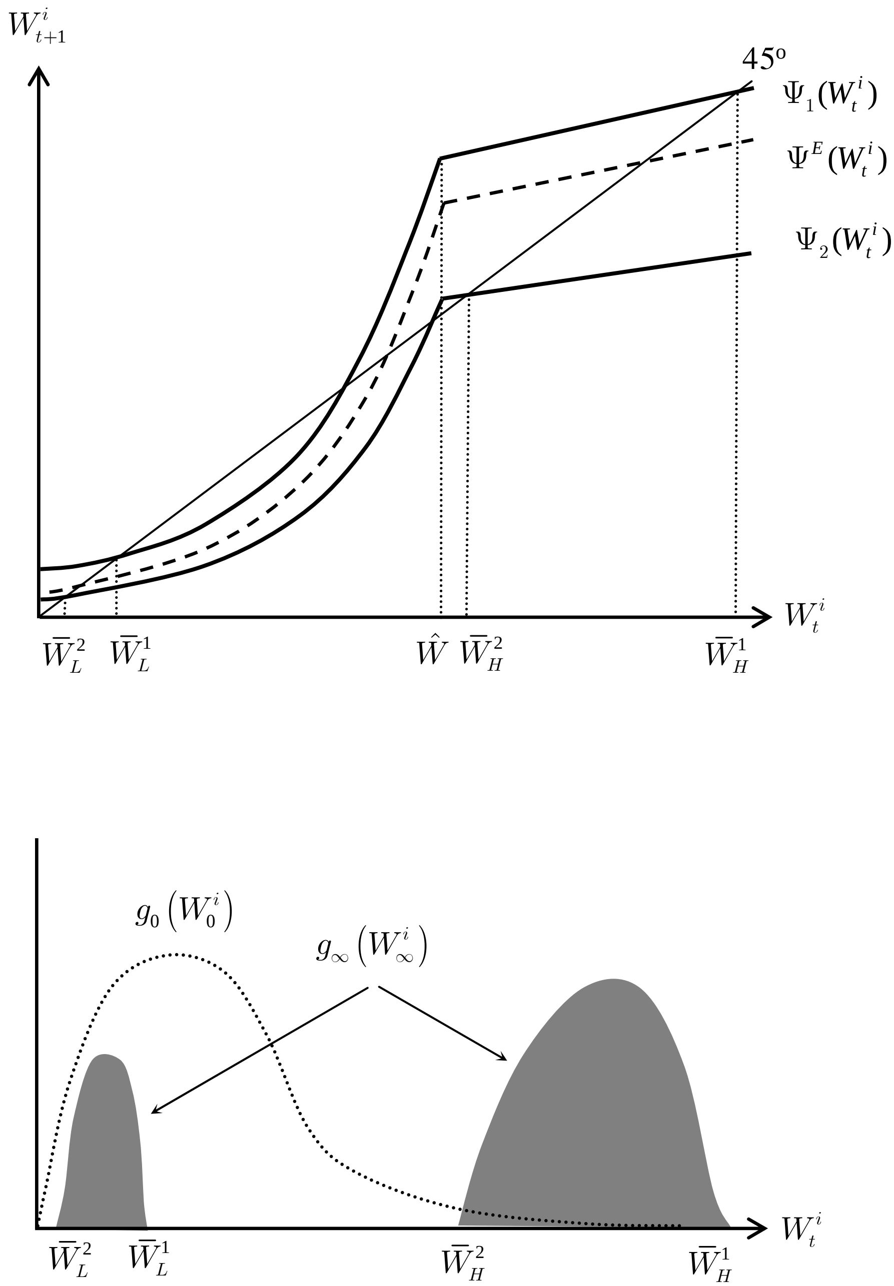

Figure 2(b): Wealth Dynamics: Non-convergence

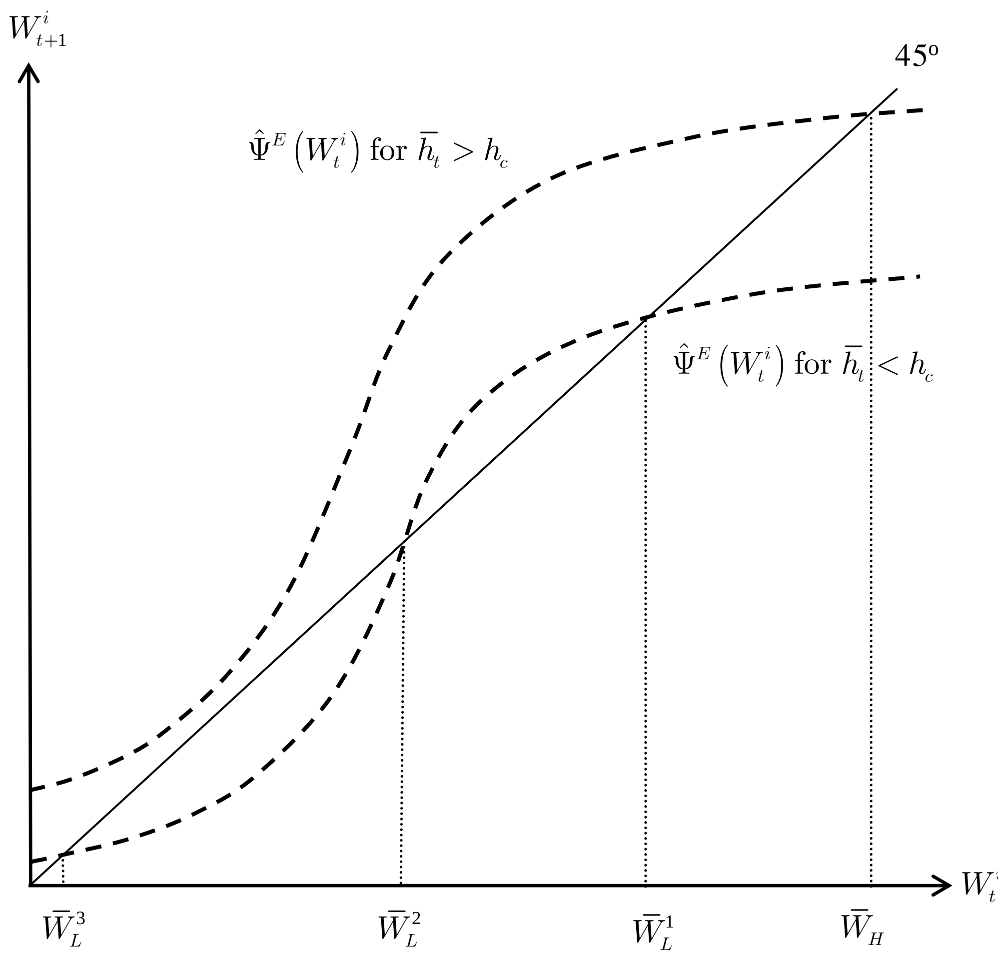

Figure 3: Wealth Dynamics with Health Externalities