HEALTH CYCLES AND HEALTH TRANSITIONS

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We study the dynamics of poverty and health in a model of endogenous growth and rational health behavior. Population health depends on the prevalence of infectious diseases that can be avoided through costly prevention. The incentive to do so comes from the negative effects of ill health on the quality and quantity of life. The model can generate a poverty trap where infectious diseases cycle between high and low prevalence. These cycles originate from the rationality of preventive behavior in contrast to the predator–prey dynamics of epidemiological models. We calibrate the model to reflect sub-Saharan Africa's recent economic recovery and analyze policy alternatives. Unconditional transfers are found to improve welfare relative to conditional health-based transfers: at low income levels, income growth (quality of life) is valued more than improvements to health (quantity of life).

Keywords: Infectious Disease, Cycles, Economic Epidemiology, Morbidity, Mortality, Conditional Transfers, Unconditional Transfers

1. INTRODUCTION

Health and income are each elemental to welfare, but it is perhaps their joint relationship that has most intrigued researchers. Countries that are poor are also likely to have worse population health. Nowhere is this correlation more apparent than in sub-Saharan Africa, with its twin problems of severe poverty and ill health. Much of the ill health stems from infectious diseases, which account for 64% of

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its overall disease burden (DALYs) relative to 30% worldwide [World Health Organization (2002)]. Although the burden falls disproportionately on infants and children, adults suffer too. The probability of death between ages 15 and 60 is $30\% - 60\%$ for African men compared to less than 12% in developed countries (Rajaratnam et al., 2010).

Building on prior work [Chakraborty et al. (2010)], this paper presents a simple dynamic model of poverty and health. In a two-period OLG model of endogenous growth, health is the outcome of exposure to infections early in life. That exposure depends on the prevalence of infectious disease and the extent to which individuals take preventive action to avoid it. Prevention incentives depend on the economic cost of illness and the effect of illness on overall well-beereas, while the ability to undertake costly prevention depends on potential earning.

This paper makes two new contributions to the literature on health and development. First it, shows that the complementarity between poverty and infection risk can lead to a unique poverty trap that consists of periodic health and economic cycles. Epidemiological models are known to admit cycles, usually from some form of predator–prey interaction. Cycles arise here, instead, from a behavioral response to the threat of infection and the effect of that infection on household choices and income.

An economy suffering from poverty and ill health faces a low return from costly prevention because the threat of infection—the disease externality—is high. Despite an underinvestment in prevention and concomitant ill health, as long as aggregate TFP is high enough, the economy grows incrementally. When income crosses a threshold, prevention becomes affordable. This has two effects: it leaves fewer resources for investments that directly augment future income and it lowers the aggregate risk of infection. The first effect depresses the future economy; the second reduces incentives to engage in further prevention. The combined effect is to tip the economy back toward high disease prevalence.

The second contribution of this paper is to inform policies that facilitate economic and health transitions.[1] After decades of stagnation during which African growth hovered at a dismal 1% in real terms and population health problems intensified, there are signs of hope for the continent. GDP per capita grew by 5% during the last three years [International Monetary Fund (2013)] and the HIV crisis has stabilized, and is on the retreat in some areas United Nations (2013)].

Inspired by this incipient growth, we calibrate the model to replicate broad patterns of sub-Saharan Africa's development. The dynamics implied by this exercise shows slow initial growth and health improvement, which then lead to rapid convergence toward the growth frontier. We study several types of policies—unconditional foreign aid, conditional (health-based) foreign aid, and domestic transfers. Much of the debate on the effectiveness of foreign aid and poverty alleviation has occurred in a theoretical vacuum, without close attention to the intertemporal nature of poverty and its incentives. Because ill health is the source of poverty in our theory of African underdevelopment, the model is particularly suited to identifying the dynamic gains and losses from these policy alternatives.

We show that, in principle, foreign assistance can be an effective tool to improve health and economic outcomes.[2] Both conditional and unconditional foreign aid are capable of significantly improving health and income, though, somewhat surprisingly, unconditional aid is better at improving overall welfare. Even though health-based aid is better at effecting a health transition, at low levels of income, those gains to the quantity of life are dominated by the higher value placed on the faster income gains from unconditional aid. We also consider combination policies that ease the economy into transition through foreign aid, later replaced by fully funded domestic transfers: the welfare gains relative to the noninterventionist outcome are sizable, but come at the cost of slow convergence to the growth frontier.

Several bodies of work are related to this paper. We build on Geoffard and Philipson's (1996) insight that ignoring the effect of rational health behavior may convey an imprecise view of disease dynamics and the effectiveness of public health interventions. Two contributions in economic epidemiology most relevant to our work are Goenka and Liu (2010) and Goenka et al. (in press). Both integrate the susceptible–infectious–susceptible (SIS) model from epidemiology into dynamic general equilibrium models. The first paper illustrates the presence of disease and economic cycles due to a variation of the predatory–prey mechanism where there is no health choice. The second paper incorporates public health expenditure that affects people's susceptibility to illness and recovery from it and establishes a rich set of dynamic possibilities for the social planner's problem. In our paper, on the other hand, market failures from disease and production externalities drive the dynamics and open the door for policy interventions.[3]

From the voluminous literature on foreign aid and growth, one paper is particularly relevant to our analysis of conditional health-based aid. Mishra and Newhouse (2009) estimate the effects of aid on infant mortality and find that although overall foreign aid does not have a statistically significant effect on infant mortality, health aid does. On developing country epidemiology, García-Montalvo and Reynal-Querol (2007) find that forced migration and social disruption due to civil wars are significant contributors to malaria incidence. Although we ignore geopolitical factors in the spread of diseases, if the quantitative results presented here are any indication, the cumulative economic cost of this interaction between conflict and health is sizable.

A third literature related to our work studies the effectiveness of in-kind and cash transfers for poverty alleviation. Economists have traditionally preferred cash transfers over in-kind transfers because the latter can lower overall utility by restricting household choices [Currie and Gahvari (2008)]. Yet one of the key aspects of poverty is its dynamic nature. In fact, if poverty were a transitional problem, the need for pro-poor policies would be less pressing. When dynamic incentives matter, in-kind transfers may well be more effective if they are targeted toward investments that improve income in the long run [Mookherjee (2006)]. To the best of our knowledge, this paper is the first to formalize this trade-off in the context of Africa and poverty stemming from infectious disease and ill health.

The remainder of the paper is organized as follows. Section 2 reviews the model and identifies the key forces driving the dynamics of health and development. The model is calibrated to sub-Saharan Africa (henceforth SSA) in Section 3, which then presents convergence dynamics and poverty trap cycles for reasonable parameter values. Section 4 returns to the baseline case of convergence growth and studies various policy packages. Section 5 concludes.

2. THE MODEL

Overlapping generations of families populate a discrete-time, infinite-horizon economy. At every date $t = 1, 2, \ldots$ a unit mass of individuals is born. They are each endowed with a unit of labor time and potentially live for two periods. Survival to the second period depends on whether or not they contract an infectious disease early in life and prematurely die from it. Model particulars closely follow Chakraborty et al. (2010) [henceforth CPP (2010)].

2.1. Disease Transmission

Individuals work in youth and are retired in the second period. During youth, each individual gives birth to one offspring, who is born healthy. An infected adult suffers a productivity loss of θ because of morbidity, supplying $1 - \theta$ units of efficiency labor. He also enjoys a lower quality of life: a consumption bundle c delivers a utility flow $\delta u(c)$ instead of $u(c)$, where $\delta \in (0, 1)$. Finally, an infected young individual faces the risk of dying before reaching old age.

All individuals start their youth healthy. Subsequently some of them contract infectious diseases, susceptibility to which depends on prevention and disease prevalence. Early in youth individuals undertake preventive investment $x_t \geq 0$. This can take the form of expenditures on food and medicine to improve general health, or specific actions such as investing in cleaner environments (e.g., potable water, sanitation), protective goods (e.g., mosquito nets, condoms), and occupational shifts (e.g., more hospitable farmland). Prevention is chosen ex ante, before a susceptible young individual is exposed to infections, funded by zero-interest within-period borrowing against labor income.

Diseases spread from infected to susceptible individuals either directly via humans or indirectly through vectors such as mosquitoes, tsetse flies, water, and air. Because our objective is to model the transmission of various infectious diseases generally, we take a parsimonious approach, assuming that the microbial load among these disease vectors is simply proportional to the number of infected individuals and that each susceptible individual is exposed to $\mu > 1$ types of disease vectors.[4] Given x_t, the probability that a young individual gets infected by a particular disease vector is

$$\pi(x) = \frac{a}{1 + qx}, \ a \in (0, 1), \ a > 1/\mu, \ q > 0, \tag{1}$$

for which $\pi' < 0$, $\pi(0) = a$, and $\pi(\infty) = 0$.

The assumptions underlying (1) merit further discussion. The parameter a should be thought of as intrinsic resistance, the outcome of virus mutations and genetic evolution of humans in their particular environments. Humans in the Old World acquired immunity and resistance over thousands of years as a consequence of its temperate climate and domestication of animals, endowing those populations with lower values of a that increased the efficacy of preventive behavior. For example, starting from a population with heterogenous disease resistance, if less resistant individuals died without passing on their genes, over time the population would end up with lower average a.

In the case of SSA this evolutionary process may have been confounded by its encounter with the West. Colonization introduced new diseases to nonimmune populations in eastern, central, and southern Africa that were relatively more isolated than western Africa. Previously endemic diseases often took the form of epidemics, so much so that the period 1880–1920 has been described as a time of tumultuous "ecological disaster" [Lyons (1993)]. By the mid-nineteenth century, tropical Africans were afflicted by most of the diseases of the temperate Old World.

Similarly, we interpret q as the effectiveness of medicine and national health institutions. These are taken as exogenous [see Bhattacharya and Qiao (2007) for a model of public health]. The epidemiological literature offers evidence for why q may have been substantially lower in Africa than in Europe. If colonialism brought new diseases to Africa, the public health practices of the colonial powers did not help matters. Often large-scale medical campaigns were launched against single illnesses, which were expensive but made little dent on the overall problem. In some cases, disease-specific knowledge either was absent or had limited transferability to Africa [Dunn (1993)]. These problems have been worsened in the twentieth century by wars and social unrest, and by public health systems that are widely ineffective due tobecause ofcorruption, lack of provision, and, in some cases, scarcity of skilled manpower.

Let p_t denote the probability of being infected for a typical young member of generation t. The probability that this person contracts an infection from exposure to a specific vector is $i_t \pi_t$, where i_t is microbial load, proportional to the fraction of generation $t-1$ who were infected. Hence the probability of contracting infections from exposure to the μ disease vectors is

$$p_t = 1 - [1 - i_t \pi(x_t)]^{\mu}. \qquad (2)$$

Appealing to the law of large numbers, this gives the prevalence rate of the disease at date $t+1$, that is, $i_{t+1} = p_t$.

Our transmission process departs in important ways from the epidemiology literature, where the evolution of diseases is typically exogenous to human decisions. Although the way infection spreads from the infective to the susceptible population in equation (2) is akin to standard epidemiological models, the probability of that transmission depends on a cost–benefit calculation of whether or not prevention
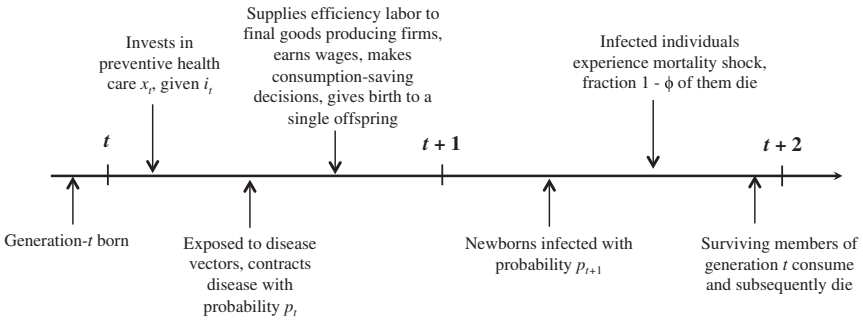
FIGURE 1. Timing of events.

is desired. We show later that the willingness to undertake prevention depends discontinuously on disease prevalence and income. Households are unwilling to spend on prevention in two situations: when the prevalence rate is too low and the threat of infection negligible, or when the prevalence rate is so high that the disease externality trumps private health behavior. Because of this, we streamline the disease dynamics in other respects relative to epidemiological models.

2.2. Preferences and Prevention

As Figure 1 illustrates, household behavior is best studied in two stages: first, economic choices are made contingent on prevention and disease outcomes; then, health choices determine the risk of infections and economic outcomes. Let the superscript U (I) on variables denote decisions and outcomes for uninfected (infected) individuals. An uninfected individual whose preventive behavior has successfully protected him from infectious disease maximizes lifetime utility

$$u\left(c_{1t}^{U}\right) + \beta u\left(c_{2t+1}^{U}\right), \ \beta \in (0, 1),$$

subject to the budget constraints $c_{1t}^{U} = w_t - x_t - z_t^{U}$ and $c_{2t+1}^{U} = R_{t+1}z_t^{U}$. w is the wage per efficiency unit of labor, z denotes savings, and x is given by decisions made early in period t.

An infected individual, facing the constant probability $1 - \phi \in [0, 1]$ of dying from infectious disease in old age, maximizes expected lifetime utility

$$\delta\left[u\left(c_{1t}^{I}\right) + \beta\phi u\left(c_{2t+1}^{I}\right)\right],$$

subject to $c_{1t}^{I} = (1 - \theta)w_t - x_t - z_t^{I}$ and $c_{2t+1}^{I} = \hat{R}_{t+1}z_t^{I}$, where $\hat{R}_{t+1}$ denotes the annuity return that the individual takes as given. Assuming a perfect annuities market, at equilibrium, we have $\hat{R}_{t+1} = R_{t+1}/\phi$. For the utility function, we choose the standard CES, $u(c) = (c^{1-\sigma} - 1)/(1 - \sigma)$, $\sigma \geq 0$.

Conditional on the choice of x, the household's optimization problem yields the saving decisions

$$z_t^{\mathrm{U}} = s_t^{\mathrm{U}}(w_t - x_t), \quad \text{with } s_t^{\mathrm{U}} \equiv \left(\frac{\beta^{1/\sigma} R_{t+1}^{1/\sigma - 1}}{1 + \beta^{1/\sigma} R_{t+1}^{1/\sigma - 1}} \right), \tag{3}$$

and

$$z_t^{\mathrm{I}} = s_t^{\mathrm{I}}[(1 - \theta)w_t - x_t], \quad \text{with } s_t^{\mathrm{I}} \equiv \left[\frac{\phi\beta^{1/\sigma} R_{t+1}^{1/\sigma - 1}}{1 + \phi\beta^{1/\sigma} R_{t+1}^{1/\sigma - 1}} \right], \tag{4}$$

where the equilibrium annuity return has been substituted in. The impact of disease on development partly follows from the result $z_t^{\mathrm{U}} > z_t^{\mathrm{I}}$. The infected save less because they face a shorter lifespan ($\phi < 1$) and are less productive ($\theta > 0$). The third type of cost, a lower utility flow ($\delta < 1$), affects saving indirectly through preventive investment.

Turn to this decision next. Substituting (3) and (4) into lifetime utility gives the two indirect utility functions $V^{\mathrm{U}}(x_t)$ and $V^{\mathrm{I}}(x_t)$ contingent on prices, preventive health choices, and disease realizations. At the beginning of their youth, forward-looking individuals choose x_t to maximize expected lifetime utility,

$$V_t \equiv p_t V^{\mathrm{I}}(x_t) + (1 - p_t) V^{\mathrm{U}}(x_t), \tag{5}$$

subject to $x_t \geq 0$ and equation (2), that is, taking into account how x_t alters their risk of contracting infections. If the marginal cost from health investment exceeds the marginal benefit at $x_t = 0$, no prevention is an optimal choice.

2.3. Production Technology

Define $L_t = 1 - \theta p_t$ as the aggregate efficiency labor supply at time t, and $k_t = K_t/L_t$ as the capital stock (K) per effective unit of labor. A continuum of firms, indexed by i, operate in perfectly competitive markets to produce the final good using capital and labor. For firm i, the production function is

$$F(K^{\mathrm{I}}, L^{\mathrm{I}}) = A \left(K^{\mathrm{I}} \right)^{\alpha} \left(\bar{k} L^{\mathrm{I}} \right)^{1 - \alpha}, \tag{6}$$

where $\alpha \in (0, 1)$, $A > 0$ is a constant productivity parameter, $\bar{k}$ denotes the average capital intensity across firms, and it augments labor productivity through a learning-by-doing externality. Standard factor pricing relationships under such externalities imply that the wage per effective unit of labor (w_t) and interest factor (R_t) are $w_t = (1 - \alpha)Ak_t$ and $R_t = \alpha A \equiv R$, respectively.

2.4. Equilibrium Dynamics

The solution to the maximization problem (5) defines optimal prevention as a function of capital per effective worker and disease prevalence, $x_t = x(k_t, i_t)$.

Optimal prevention is zero as long as its utility cost dominates. This occurs at relatively low levels of income and very high prevalence rates, or at very low prevalence rates. When people do engage in prevention, the demand for prevention depends on the capital stock and disease prevalence in predictable ways; that is, $\partial x/\partial k > 0$ and $\partial x/\partial i > 0$.

Using optimal health investment $x(k_t, i_t)$, the equilibrium probability of getting infected can be written as $p_t = p[x(k_t, i_t), i_t] \equiv p(k_t, i_t)$. For reasonable numerical values assigned to the parameters, including the ones we use later, $\partial p_t/\partial k_t < 0$ and $\partial p_t/\partial i_t > 0$. The former result is simply an income effect operating through prevention. The latter ($\partial p_t/\partial i_t > 0$) is determined by two opposing effects: disease prevalence directly increases the probability of contracting infections, but also tends to lower it by encouraging prevention. This indirect effect is not sufficiently strong to overturn the externality effect.

Two difference equations fully characterize the global dynamics given the initial conditions (k_1, i_1). To get the first one, observe that aggregate saving is $S_t = p_t z_t^I + (1 - p_t)z_t^U$ and the asset market clears when $K_{t+1} = S_t$. Substituting for equilibrium disease transmission and dynamics leads to

$$k_{t+1} = \frac{p(k_t, i_t)z^I(k_t, i_t) + [1 - p(k_t, i_t)]z^U(k_t, i_t)}{1 - \theta p[p(k_t, i_t)]}. \tag{7}$$

The equilibrium evolution of the prevalence rate follows

$$i_{t+1} = p(k_t, i_t). \tag{8}$$

The nonlinear difference equation system comprising equations (7) and (8) can produce a surprisingly rich set of dynamic behavior. One possibility is two asymptotically stable stationary equilibria. In the better stationary equilibrium, a fully healthy population enjoys rapid improvements in living standards.[5] The worse stationary equilibrium, on the other hand, can be of two types. In one type, the economy is in a poverty trap that exhibits monotonic convergence: in the long run everyone is infected (at some point in their youth), there is no investment in prevention, and growth is low, even zero. Such a poverty trap may be "income-neutral" in the sense that exogenous (higher A) or endogenous increase in income does not take the economy of the trap. This is the case analyzed in CPP (2010).

For future reference we derive these growth paths. Define γ to be the growth rate of the economy's capital stock per effective unit of labor in the steady state. When $i = 0$, no one has to invest in prevention and the economywide saving propensity is s^U. Equation (7) then implies that the high-growth path is

$$1 + \gamma^H \equiv (1 - \alpha)As^U = \left[\frac{\beta^{1/\sigma}(\alpha A)^{1/\sigma-1}}{1 + \beta^{1/\sigma}(\alpha A)^{1/\sigma-1}}\right](1 - \alpha)A. \tag{9}$$

We shall refer to this as a country's potential long-run growth: whether or not it is attained depends on the presence of other local attractors.

Now consider a steady state with full prevalence $i = 1$. If prevention is ineffective in this environment, no one will invest in it and everyone will suffer from ill health.[6] If such a steady state exists and is asymptotically stable, the asymptotic growth factor is

$$1 + \gamma^{\text{L}} \equiv (1-\theta)(1-\alpha)As^{\text{L}} = \left[\frac{\phi\beta^{1/\sigma}(\alpha A)^{1/\sigma - 1}}{1 + \phi\beta^{1/\sigma}(\alpha A)^{1/\sigma - 1}} \right] (1-\theta)(1-\alpha)A. \quad \textbf{(10)}$$

The growth rate is zero if the expression on the right is less than one, positive otherwise.

Interestingly, this is not the only type of asymptotically stable poverty trap that can arise. Section 3 shows that for reasonable values of TFP and the elasticity of intertemporal substitution (EIS), there can be poverty traps featuring periodic cycles in disease and development.

3. QUANTITATIVE ANALYSIS

Among the growth facts development economists agree on is SSA's post–World War II divergence from the rest of the world: SSA's output per worker grew at an average annual rate of 0.6% during 1950–2000 against 1.9% for the United States (Penn World Table 6.2). The quantitative analysis calibrates the model to fit these growth patterns as of 2000–2001. As we will see later, the baseline model will generate a dynamic path consistent with the signs of SSA's economic recovery during the last decade.

3.1. Calibration

Because our objective is to assess the macroeconomic effects of the infectious disease burden, instead of calibrating the model to a specific disease as in CPP (2010), we use aggregate data on mortality and morbidity in SSA whenever possible. That captures more appropriately the complementarities across infectious diseases that simultaneously affect a population. For example, one study estimates that the interaction between malaria and HIV may have been responsible for 8,500 excess HIV infections and 980,000 excess malaria episodes in Kenya [Abu-Raddad et al. (2006)]. Such co-infection may have also made it easier for malaria to spread to areas with high HIV prevalence. Other studies have found that open sores from untreated bacterial STDs in SSA facilitate the transmission of the HIV virus. The result of these complementarities is that the overall economic loss due to mortality and morbidity is higher than the average loss across illnesses.

Table 1 reports the assigned parameter values that we will refer to as the baseline scenario. The model features overlapping generations of agents who potentially live for two periods. To choose the length of one period, we use data on U.S. life expectancy at age 15 (LE15), which was 63 in 2000, according to WHO (2008a).

TABLE 1. Benchmark parameter values

Preference	Production	Health and disease	
$\beta = 0.28$	$\gamma^H = 0.018$	$\theta = 0.15$	$\mu = 6000$
$\sigma = 1$	$\alpha = 0.67$	$\phi = 0.62$	$q = 35$
	$A = 24.18$	$\delta = 0.73$	$a = 0.2$

This implies 31.5 years for each period or generation. Accordingly the discount factor β is calibrated to $0.99^{31.5\times4}$ based on the "standard" value per quarter.

We take preferences to be logarithmic, $\sigma = 1$. Later, we report robustness results for $\sigma = 1.5$.[7] Two parameters need to be calibrated for the aggregate production function: the total factor productivity (TFP) term A and the output elasticity of capital α. We interpret capital broadly (physical, human, organizational) to set $\alpha = 0.67$. The value for A is chosen so that the growth rate is 1.8% in the potential high-growth steady state. This growth rate corresponds to OECD's average growth rate of GDP per capita during 1990–2003 [United Nations Development Program (2005)]. In other words, A is chosen so that $(1-\alpha)s^U A = 1.018^{31.5}$, which implies that $A = 24.18$.

This process of calibrating A may not be entirely appropriate for Africa. Besides technologies and institutions, A is affected by the ability of a country's workforce. African soils are typically acidic, nitrogen-deficient, and deprived of minerals such as calcium and phosphorus [Kiple (1993)]. As a result, crops have been lacking in protein and minerals. Because the sub-Saharan African diet was predominantly vegetarian, it was thus nutrition-deficient. Animal protein was relatively scarce: few animals were available because they were quickly hunted down or they fell prey to illnesses from tsetse flies. Although some animals were raised in West Africa, there was a taboo against drinking goats' milk and eating eggs. Given current agricultural technologies and specialization, these factors may plausibly affect Africa's A through a lower labor productivity even when the population is disease-free. We later study the effects of lowering A, and hence γ^H.

Estimates of the quality-of-life effect come from disability weights in the Burden of Disease Project. A disability weight for a specific disease is a scaling factor that ranges from zero (fully healthy) to one (worst possible health state). It is derived from patient surveys on subjective valuations of disease impact and varies according to illness. For example, it equals 0.0 for chagas disease, 0.1 for diarrheal episodes, 0.3 for malaria, and 0.5 for AIDS, according to WHO (2008b). The last three diseases account for much of SSA's disease burden, so we take their average value and set $\delta = 1 - 0.27$.

To obtain an estimate of the income loss due to morbidity, we look at Dasgupta (1993). He finds that workers (in particular, farm workers) who are too ill to work in developing countries lose about 15 to 20 days of work each year, and when they are at work, their productivity may be severely constrained by a combination of malnutrition and parasitic and other infectious diseases. His estimates suggest

that potential income loss due to illness for poor nations is on the order of 15%. This is the value assigned to θ.

We calibrate the survival parameter ϕ using data from the World Health Organization (2001). According to this source, fatalities from infectious diseases represent 38% of all deaths in Africa in 2001 for the adult male population aged 45 to 80—this interval corresponds to the agents' second period of life when they can die because of illness. We require that the model reproduce this number, assuming that SSA has very high prevalence, $i \approx 1$. Because the entire population suffers from ill health under full prevalence, the probability of death from infectious disease has to be 0.38. Hence $\phi = 0.62$ is our benchmark value.

Parameters governing disease transmission (a, μ, and q) are critical to quantifying the effort needed to battle disease prevalence and transmission. Here we have less guidance from aggregates. So we adopt estimates from CPP (2010) that focus on two diseases—malaria and AIDS—that together accounted for 57% of all deaths in SSA from infectious diseases in 2001. They find that transmission probabilities in each encounter with malaria and HIV in the absence of prevention are 25% and 1%, respectively; we choose an intermediate value of 0.2 for a. CPP also report that sexual encounters amount to about 3,402 each model period per male and that a human is bitten by a mosquito at least every 1.4 days. Given that the susceptibility to malaria declines by 66% after 20 exposures, we set $\mu = 3,402 + 31.5(1 - 0.66)365/1.4 \approx 6000$. Finally, CPP calibrate values for q in the HIV and malaria cases of $1/0.0286 = 35$ and $1/0.0015 = 667$, respectively. We adopt the smaller value, assuming that malaria (HIV) prevention does not make HIV (malaria) prevention redundant. This holds for other infectious diseases as well: prevention is necessary for all of them.

3.2. Convergence to the Growth Frontier

Using the baseline values from Table 1, we first present the model's dynamics as it applies to the "average" sub-Saharan African economy. We are also interested in understanding the dynamics more generally, that is, not narrowly restricted to the benchmark values. We do this by departing from Table 1 for key parameters.

Panel (a) of Figure 2 illustrates the phase portrait of the economy for the parameter values in Table 1. It plots the prevalence rate i_t against the economywide stock of capital K_t.[8] The two $x(K_t, i_t) = 0$ lines, in solid gray, represent combinations of (K_t, i_t) for which the optimal decision is not to invest in prevention. For low levels of disease prevalence ($i_t \to 0$), the risk of catching an infection is so low that prevention is not necessary. This is given by the lower part of the $x_t = 0$ locus. At high levels of disease prevalence ($i_t \to 1$), in contrast, the productivity of prevention becomes so small at low levels of capital that prevention is not worthwhile. This is given by the upper part of the $x_t = 0$ locus.
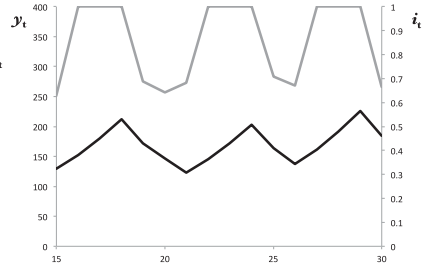
Turn next to the downward-sloping locus, in dashed gray, $i_{t+1} = i_t$. This is defined by $i_t = p(K_t, i_t)$, along which prevalence remains constant. The locus is defined wherever $x_t > 0$. In this area, prevalence is always decreasing above
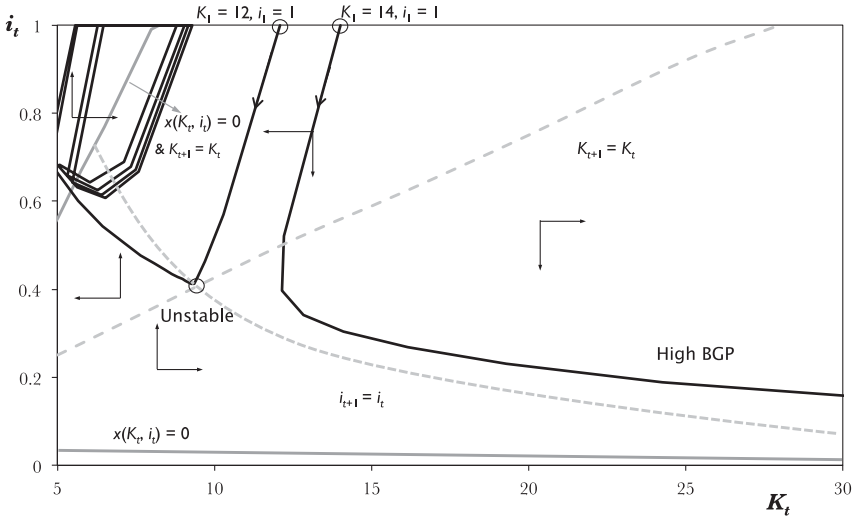
(a) Phase Diagram
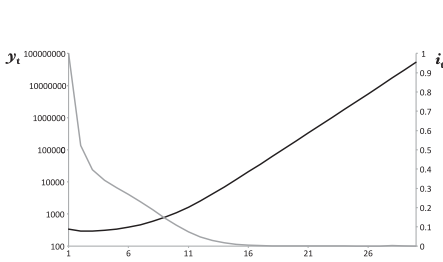
(b) Convergence Growth

(c) Poverty Trap Cycle

FIGURE 2. Dynamics for the baseline model.

the locus, increasing below it. When prevention is zero, on the other hand, the prevalence rate is always increasing because $\mu a > 1$.

Finally, turn to the dynamics of the capital stock. Capital remains constant along the $K_{t+1} = K_t$ locus. As long as $x_t > 0$, the capital stock declines above this locus and increases below. This is because when prevention is positive, the capacity to save diminishes and if K_t is too small and i_t sufficiently large, $\Delta K_t < 0$. When $x = 0$, on the other hand, saving is always high enough to keep capital increasing as long as $K_1 > 0$.

The vector field generates three stationary equilibria, two of which are stable. The point labeled "unstable" in Figure 2a is a saddlepoint, which, because the initial conditions (K_1, i_1) are predetermined, is asymptotically unstable. One of the asymptotically stable stationary equilibrium is the high-growth path labeled BGP, along which infectious diseases are eradicated and the economy grows

at a healthy rate given by (9). Suppose that inspired by SSA's recent signs of recovery, we choose this initial state to be $(K_1, i_1) = (14, 1)$, which generates growth recovery and eventual convergence to the high-growth path even when the disease burden is high. Figure 2b illustrates the trajectory of this economy: output per worker y_t (equivalently aggregate output) in black and the prevalence rate i_t in gray. Growth is initially negative—the disease burden is too high to maintain a growing capital stock—but the economy keeps investing in prevention. As population health improves, the economy gradually converges to an annual growth rate of 1.8%. By the ninth generation, output growth exceeds 50% of the long-run potential.

The other stationary equilibrium of this economy is a poverty trap, one characterized by irregular 5–6 period cycles. If the economy were slightly poorer, $(K_1, i_1) = (12, 1)$, after an initial period of economic contraction and falling prevalence, it would converge to irregular periodic cycles. The entire trajectory is illustrated in Figure 2a and the associated cycles (after convergence) in Figure 2c. This possibility of health cycles, disease cycles accompanied by changing prevalence behavior, is what distinguishes our poverty trap from the one in CPP (2010).

3.3. Health Cycles in a Poverty Trap

The existence of health cycles is robust to alternative parameter values. Maintaining the disease cost parameters at their benchmark values, for expositional convenience, we change two parameters, the inverse of the EIS σ and total factor productivity A.

Because available estimates of the EIS are 1 or above, consider first the case of $\sigma = 1.5$. As before, we calibrate A to match the OECD growth rate, which now implies a value $A = 43.32$. Starting from the same initial conditions $(K_1, i_1) = (14, 1)$, Figure 3a shows that the economy initially grows at negative rates for reasons similar to those previously stated. By the third generation, health has sufficiently improved so that the economy steadily grows toward the high-growth path. Here too, the higher-growth path is the unique asymptotically stable steady state.

Suppose, however, we delink the calibration of A from OECD growth for reasons alluded to earlier. Instead of an annual growth rate of 1.8%, suppose that γ^H corresponds to an annual growth rate of 1.5%. Over the course of a generation, such an economy would improve by 60% as opposed to 75% before, a sizable welfare gap. For this lower growth rate, the value of A is 37.4 when $\sigma = 1.5$. Figure 3b shows that this economy never converges to the high-growth path. It cycles regularly between periods of high output per worker accompanied by better population health, and low output accompanied by worse health.

Figure 4 illustrates cyclical poverty traps for other values of (A, σ). In the top two panels, the EIS is slightly below 1 and potential long-run growth is quite high for the average sub-Saharan African economy. Panel (a) shows a regular cycle of periodicity 9, panel (b) an irregular cycle of 4–7 periods. Panel (c) returns to the
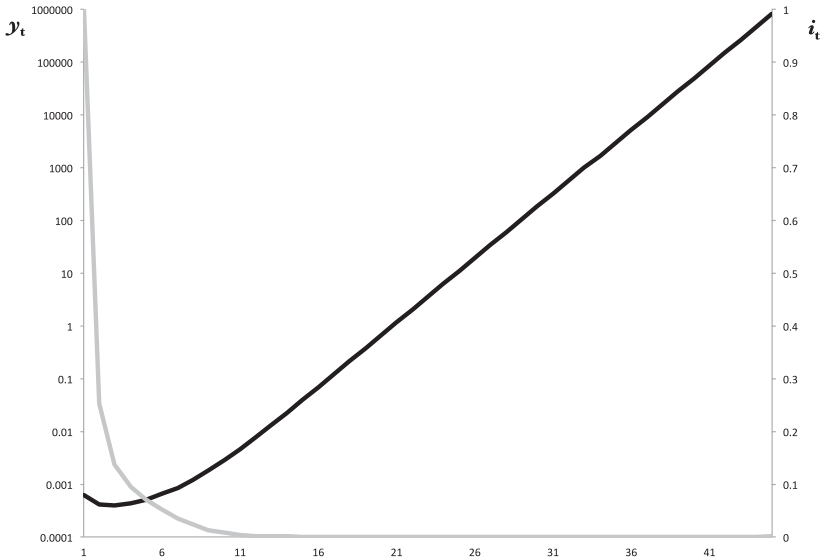
(a) $A = 43.32$, $g = 1.8\%$
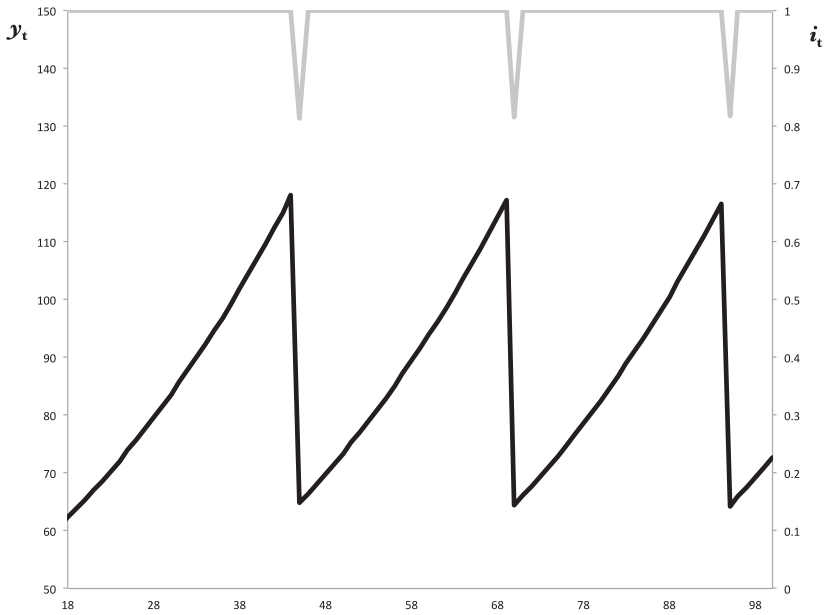
(b) $A = 37.4$, $g = 1.47\%$

FIGURE 3. Sustained growth and poverty trap cycles under $\sigma = 1.5$.

(a) $\sigma = 1.1, A = 24.18, g = 1.45\%$

(b) $\sigma = 1.1, A = 25.5, g = 1.61\%$

(c) $\sigma = 1, A = 23.25, g = 1.67\%$
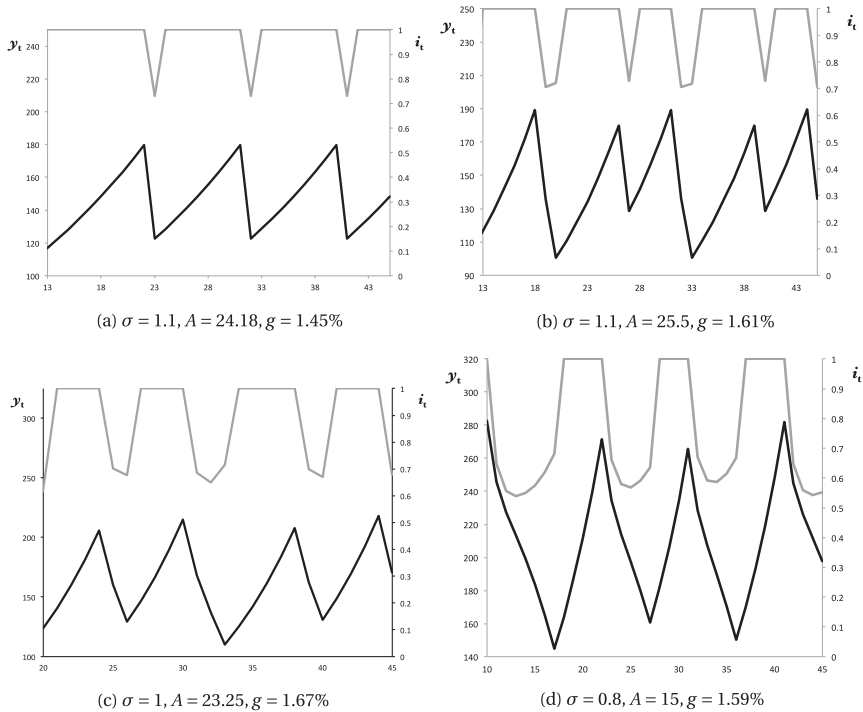
(d) $\sigma = 0.8, A = 15, g = 1.59\%$

FIGURE 4. Regular and irregular cycles.

baseline logarithmic case and shows irregular, 5–7 period, cycles for a slightly lower value of A. Recall that the saving rate is inversely related to the interest factor, hence A, whenever $\sigma > 1$. To show that these cycles are not driven by low EIS, the last panel of Figure 4 shows irregular cycles of 9–10 periods for $\sigma = 0.8$.

Taken together, these results show that it is easy for a poor economy with a high disease burden to remain in a poverty trap. This nonconvergence result is similar to CPP (2010) even though there were no cycles there.[9] These cycles are not always frequent enough to be called epidemics, so we prefer to think of them as long-run Malthusian dynamics in a world aware of the cost and control of infectious disease. That a sharp reduction in prevalence can temporarily depress per capita income mimics Young's (2005) conjecture on the economic effect of AIDS, though the mechanism is different.

It is not uncommon for epidemiology models to exhibit cycles. Cycles typically originate in those models from some form of predatory–prey dynamics, a tension between the population of disease vectors and the population of susceptible humans. Consider specifically Goenka and Liu (2010)'s interesting application of the SIS model to a Ramsey economy. There the disease vectors are infective people, whereas the susceptibles consist of healthy people who may have been infected before and have now fully recovered. There is no health choice: individuals

cannot affect their susceptibility to or recovery from infections. Goenka and Liu (2010) show that cycles depend on the contact and recovery rates. A high rate of contact between infective and susceptible agents implies high mobility from the susceptible to the infective state. At the same time, if the recovery rate from infections is high, mobility from infective to susceptible states is also high. This can generate cycles as a large percentage of the population moves from one state to the other, that is, the relative size of the infectives ("predator") and susceptibles ("prey") fluctuates. When the state of infection lowers labor income (similarly to θ), infections cause the economy to cycle, booms coinciding with an ebbing of the disease.

In contrast, the model here generates cycles through a different channel, closely linked to the *rationality* of disease behavior. To understand the mechanics, it is useful to think of two opposing forces: the positive (negative) effect of income (prevalence) on disease prevention, and the negative effect of prevalence on the growth of output per worker via savings behavior. The former depends on the disease transmission function π, whose parameterization does not change in the experiments presented in the preceding. What does change is the latter, and it changes because of (σ, A), not the disease cost parameters (ϕ, δ, θ).

In every instance of cycles presented in Figures 3 and 4, the downswing of the prevalence rate is preceded by a sharp increase in prevention, from zero to positive levels. As the economy slowly crawls out of poverty, prevalence rates remain high because prevention is too costly and ineffective. Over time, the economy accumulates enough capital to eventually afford some prevention despite the high prevalence rate. With everyone investing in prevention, the cumulative effect is to sharply reduce disease incidence. That prevention, however, extracts a cost, as the economy is still poor (low A): despite the higher average propensity to save, overall investment in capital suffers and the economy is left worse off.[10] This has the effect of discouraging future prevention, an effect amplified by the disease externality: as the prevalence rate drops, the risk of contagion drops too, weakening the need for prevention at the margin. Prevention efforts are scaled back, the economy recovers because more is allocated toward saving, and infectious diseases rebound.

Stated differently, the interplay of the infection rate—the product of the exogenous contact rate and endogenous susceptibility—and the cost of healthy behavior is what underlies the cyclicality of health and output in the poverty trap. Susceptibility can be low or high depending on whether or not prevention is incentive compatible. Rational health behavior based on prevalence alone may not cause cycles [see Philipson (2000, Sect. 3), for an illustration)]. A complementarity between disease prevalence and economic conditions is necessary.

Note also that cycles do not exist when the second effect—how much diseases drag down savings—is too weak or too strong. The effect is too weak for high TFP and low mortality cost (values of ϕ and δ closer to one): even though ill health initially weighs growth down, the economy eventually rebounds and takes off. When the effect is too strong—low TFP and especially high mortality cost as in CPP (2010)—we get a conventional poverty trap with monotonic convergence.

4. AIDING A HEALTH TRANSITION

What is the cost of ill health in terms of lost growth? What is the best strategy for achieving good health and high growth? Can foreign aid help? We turn to these questions next.

Our theory departs from the standard neoclassical model in two ways: there are externalities in the production of goods and of health. Of these, health externalities are critical in depressing economic activity and there may be a case for prioritizing health in order to facilitate overall development. With this in mind, we consider various types of transfers or subsidies. Some of them subsidize health expenditure; others are unconditional cash transfers. We study the effectiveness of each policy in facilitating growth and improving welfare. In conducting these experiments, we ignore problems of implementation or institutional failures. The focus is simply on the *potential* of these policies to make a difference.

4.1. Conditional and Unconditional Transfers

An advantage of our model of poverty and underdevelopment is that it provides a setting for studying the tension between conditional and unconditional transfers. It is not income alone that makes poverty persistent in the model; it is lack of incentives stemming from a high disease burden. The public economics literature has traditionally preferred unconditional transfers to conditional transfers: by giving individuals more choice the former can unequivocally improve welfare relative to the latter. That conclusion rests on static models of poverty. If lack of incentives keeps an economy mired in poverty over time, however, conditional transfers that target those incentives can generate dynamic gains that unconditional transfers do not [Mookherjee (2006)].

We will study two targeted subsidies, or conditional cash transfers, directed toward health: one is financed through foreign aid (x^{f}), and the other is domestically funded (x^{d}) through lump-sum taxation. In addition, we study an unconditional cash transfer policy, a pure subsidy to household income (w^{f}) funded by foreign aid. In other words, x^{f} is tied foreign aid, whereas w^{f} is untied.

Rewrite the decision problem of infected and uninfected individuals as

$$V_t^{\mathrm{U}} \equiv \mathrm{Max}_{z_t^{\mathrm{U}}} \left[\frac{\left(w_t - \tau + w^{\mathrm{f}} - z_t^{\mathrm{U}}\right)^{1-\sigma}}{1-\sigma} + \beta \frac{\left(R_{t+1} z_t^{\mathrm{U}}\right)^{1-\sigma}}{1-\sigma} \right] - \frac{1+\beta}{1-\sigma}$$

and

$$V_t^{\mathrm{I}} \equiv \mathrm{Max}_{z_t^{\mathrm{I}}} \left\{ \frac{\left[(1-\theta)w_t - \tau + w^{\mathrm{f}} - z_t^{\mathrm{I}}\right]^{1-\sigma}}{1-\sigma} + \phi\beta \frac{\left(\hat{R}_{t+1} z_t^{\mathrm{I}}\right)^{1-\sigma}}{1-\sigma} \right\} - \frac{1+\phi\beta}{1-\sigma},$$

respectively, given x_t and where τ is a lump-sum tax. Health investment is chosen ex ante to maximize expected lifetime utility

$$V(x_t) \equiv (1 - p_t)V_t^{\mathrm{U}}(x_t) + p_t V_t^{\mathrm{I}}(x_t),$$

subject to

$$p_t = 1 - \left[1 - \frac{ai_t}{1 + q(x_t + x^{\mathrm{s}})}\right]^{\mu},$$

given i_t and where $x^{\mathrm{s}} \geq 0$, $x^{\mathrm{s}} \in \{x^{\mathrm{f}}, x^{\mathrm{d}}\}$, is a transfer that can be used only toward health. Subsidies crowd out private health expenditure, but not entirely. Hence health outcomes can improve with x^{s}. As long as the household invests an amount in prevention larger than the subsidy, whether this is given as an income transfer or as an equivalent amount of health aid does not matter. The health and financial aid bundles $\{(x^{\mathrm{s}}, 0), (0, w^{\mathrm{f}})\}$ with $x^{\mathrm{s}} = w^{\mathrm{f}}$ both improve health by the same magnitude as long as $x_t > x^{\mathrm{s}}$. However, as we saw earlier, households do not always want to invest in prevention. In that case, an unconditional transfer has no effect on health behavior, whereas conditional aid, by forcing it to be used toward prevention, can make a difference to health outcomes. The issue is whether that health outcome makes a difference to social welfare.

For social welfare, we take a finite-horizon social welfare function

$$W_t \equiv \sum_{r=t}^{t+T} V_r, \tag{11}$$

where V_r is given by the expected utility function (under optimal x_r), that is, the weighted average of the welfare of healthy and unhealthy individuals in generation r. We present results for $T = 10$, by which time much of the transition dynamics is over. We report social welfare effects relative to the nonintervention benchmark, noting that preferences are cardinal because of lifetime uncertainty.

4.2. Engineering an Early Escape

Suppose that the economy is moving along the transitional path outlined by Figure 2b. Figures 5–7 illustrate the dynamics of income per worker, life expectancy, and generational welfare under different policy packages $(x^{\mathrm{s}}, w^{\mathrm{f}})$. In each case the amount of the subsidy per generation is always 80 units, applied only for the first three generations. This subsidy amount is chosen so that when it is tied to health investment, it exceeds what individuals would have invested on their own.[11] The five lines on each figure correspond to these scenarios: no subsidies (black solid), domestically funded conditional transfers x^{d} (gray dashed), conditional foreign aid x^{f} (black dash-dotted), unconditional foreign aid w^{f} (gray solid), and a combination policy of x^{f} for one generation followed by x^{d} for two (gray dotted).
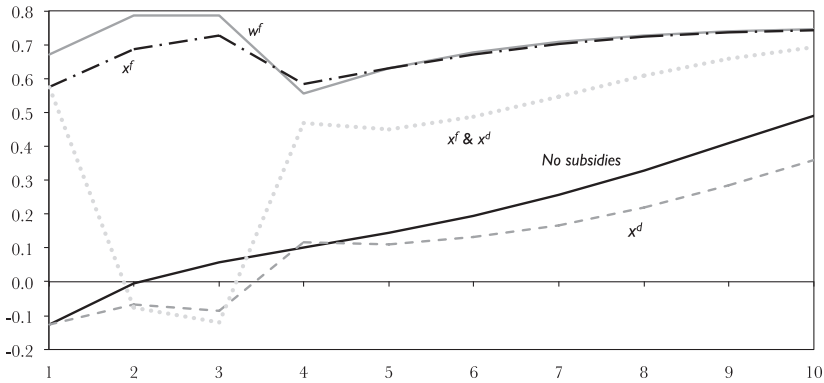
FIGURE 5. Output growth per generation under alternative policies.

In each figure, the horizontal axis measures generational time, starting with the generation that first experiences the policy, and policies are applied only during $t = 1, 2, 3$.

Two things are to be noted. First, at full prevalence, a domestic policy of tax and unconditional transfer would not alter outcomes, because everyone would be unhealthy. Hence we study domestic conditional transfers alone. Second, the combination policy package is meant to capture an emerging view that, given problems of aid dependence, foreign aid ought to be used as a temporary tool. For example, Ranis (2012) argues that engineering economic development may require unpalatable reforms. Rather than the entire cost of that adjustment being borne domestically, foreign aid may be used to ease the economy into the initial phase of adjustment before domestic policies take over. In our combination policy, conditional foreign aid is provided to the initial generation to encourage costly prevention and is then replaced by fully funded domestic conditional aid for two more generations.

As was noted earlier, under no subsidies, the economy initially experiences negative growth and rebounds from the third generation. The "no subsidies" line in Figure 5 reflects the income dynamics of Figure 2b. Domestic conditional aid x^d financed by taxes cannot accelerate the process. This is because both health investment and capital accumulation contribute to take the economy out of slow growth. When the economy allocates more resources to health by taxing income, capital accumulation decelerates and this latter effect dominates the boost that better health provides. Indeed, under this policy the economy experiences three generations of negative growth because of overinvestment in prevention, followed by low growth because of higher prevalence than under "no subsidies." In Figure 7, we see that this package gives the lowest utility of all policies.[12]

Indeed, Figure 5 makes it clear that the best policy to improve growth during the first three generations is unconditional foreign aid w^f followed by conditional foreign aid x^f. By the fourth generation, however, the latter starts to narrowly
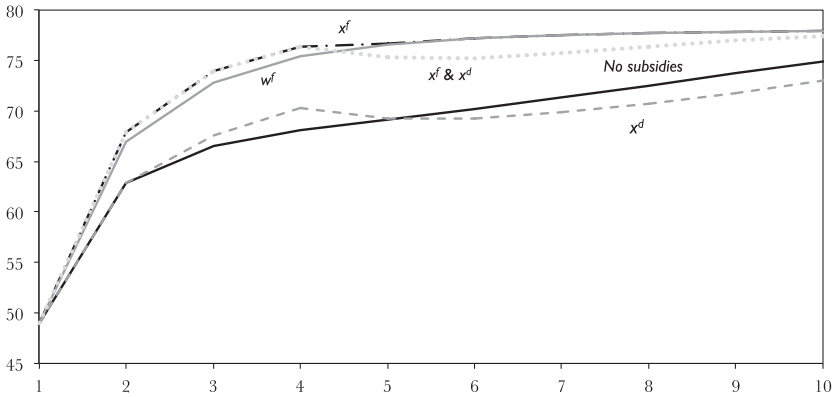
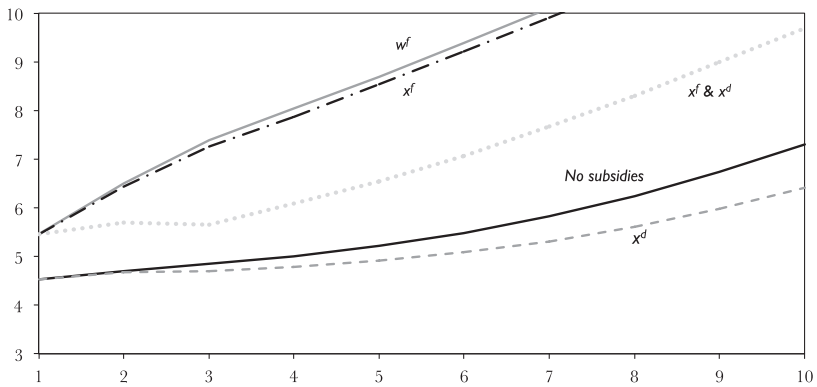**FIGURE 6.** Life expectancy per generation under alternative policies.

FIGURE 7. Welfare per generation under alternative policies.

dominate. The only dimension in which x^f outperforms w^s is in terms of longevity (Figure 6).

Figure 5 also shows that the combination policy of foreign and domestic conditional aid is better than a pure domestic policy, but does come at a cost. Both x^f and x^f together with x^d generate similar initial outcomes because they do not differ in the first period. But the pain of adjustment due to higher taxes from the second generation decelerates capital accumulation. Growth falls drastically (Figure 5) even as life expectancy improves (Figure 6).

How do these policies affect generational welfare? Figure 7 shows that unconditional foreign aid is again the best, whereas domestic unconditional transfers are the worst. To net out the short-run effects from the long run, Table 2 reports social welfare calculations using (11). The first column of results shows that the

TABLE 2. Welfare effects of alternative policies

	$\sigma = 1$, $A = 24.18$	$\sigma = 1.5$, $A = 43.32$	$\sigma = 1$, $i_1 = 0.3$	$\sigma = 1, A = 24.18$, $K_1 = 12$	$\sigma = 1, A = 20.5$, $K_1 = 7$
x^d	↓ 7%	↓ 4.5%		↓ 15.0%	
x^f	↑ 58.7%	↑ 6.5%	↑ 14.2%	↑ 101.7%	↓ 0.44%
w^f	↑ 61.3%	↑ 6.7%	↑ 14.5%	↑ 106.9%	↑ 1.72%
x^f, x^d	↑ 27.3%	↓ 0.9%		↑ 33.9%	

gap in welfare improvements between x^f and w^f is not large, but reflects the dynamics of income more than of health. Both policies, though, clearly dominate the alternatives.

As a robustness check, the second column of Table 2 reports social welfare under a lower EIS but at the same long-run growth rate of 1.8% per year. The relative ranking of unconditional and conditional foreign aid remains unchanged, although welfare changes are now smaller. Under a lower EIS, households are less inclined to postpone consumption either through better health or through savings. Hence policies meant to improve income growth or future population health are valued less.

Does policy effectiveness depend on initial conditions? We examine this by lowering the initial prevalence rate to 30%. Welfare comparisons for the two types of foreign aid appear in the fourth column of Table 2. The forces of convergence at this lower prevalence rate are, of course, much stronger because the population is healthier. Because the prevalence rate is low, prevention is effective and high. Not surprisingly, welfare improvements are smaller than in the baseline scenario, and it remains true that foreign aid yields a higher payoff if it is directed toward augmenting household budgets rather than health alone.

4.3. Policies in a Poverty Trap

Entertain now a scenario where conditional health aid has the potential to be especially important: a poverty trap. We will study two such traps. Start with the poverty trap cycle for the baseline parameter values of Table 1 and initial conditions $(K_1, i_1) = (12, 1)$, for which output fluctuates between 226 and 113. We apply the same policy packages, of 80 units during each of three generations, to this economy. Social welfare effects are reported in the fourth column of Table 2. The relative ranking of these policy alternatives is no different from that in the first column. Surprisingly, unconditional aid still dominates conditional aid, and, as expected, the welfare effects are significantly larger than for an economy already converging to high growth.

To assess the robustness of this result, suppose $A = 20.5$. Now potential long-run growth is 1.27% annually and a poverty trap exists where the economy cycles between output levels of 133 and 196. Suppose the economy starts at

$(K_1, i_1) = (7, 1)$, inside the poverty trap at an income level just above the lower bound of the cycle. If subsidies are sufficiently large to get the economy out of the trap immediately, there is no difference in outcomes between x^{f} and w^{f}. Hence we use a lower subsidy amount than before, 15 units, applied for three generations. Welfare comparisons between conditional and unconditional foreign aid are reported in the last column of Table 2. Here too unconditional aid dominates conditional aid and welfare changes are much smaller than in the baseline scenario. The negative effect of conditional aid is due to the fact that, because income levels are now lower, incentivizing healthy behavior is more costly, as it shifts resources away from capital accumulation.

In all the cases we studied previously, the economy is initially very poor. At such low levels of income, the marginal utility of consumption is high, which means even a little income growth substantially improves welfare. Even though x^{f} improves longevity by more and lowers morbidity, the income growth effect from w^{f} dominates overall welfare calculations. This result bears upon the convergence of living standards around the world. Even though income levels around the world have not converged much since World War II, life expectancy at birth has. By incorporating an income equivalent of these life expectancy improvements, Becker et al. (2005) show that this "full income" has converged much faster across countries than conventionally measured income. The preceding welfare calculations suggest that, despite the remarkable gains in life expectancy, income growth contributes to welfare convergence more strongly. In other words, all else equal, the marginal dollar is better invested in improving income in poorer countries than in improving health. This result is, of course, sensitive to the parameterization. For some African countries the cost of ill health may well be more substantial than in Table 1, in which case health-based assistance would yield larger welfare gains.

In conclusion, we note that our policy experiments suggest that, in principle, foreign assistance can be an effective way to improve income and health in the poorest regions. Despite ill health being the root of poverty here, conditional aid targeted toward healthy behavior does not dominate unconditional aid. The former does improve population health significantly, but the income gains from the latter dominate welfare calculations. Although the welfare gap between conditional and unconditional aid is not large, it is consistently positive across these experiments. Both types of policies dominate purely domestic or combination policies because of the distortionary effect of taxes on capital accumulation.

5. CONCLUSIONS

This paper has presented new theoretical results on the presence of disease cycles in an economic model of epidemiology. Based on the broad patterns of SSA's economic development, it has also quantitatively assessed whether and how policy can accelerate growth, eliminate the burden of ill health, and improve overall welfare in the developing world. For both purposes, we use a modified version of

the general equilibrium model of infectious disease transmission and economic growth presented in Chakraborty et al. (2010). The model is sufficiently simple to clearly point out important policy trade-offs, but at the same time rich enough to capture the main features of SSA's disease ecology and development challenges. In future research we hope to incorporate more precise demographic structure and health behavior to understand the applicability of these results to development policy making and the control of infectious diseases.

NOTES

1. By health transition we mean not just an epidemiological transition out of infectious diseases but also behavioral changes accompanying that transition, specifically the adoption of preventive health measures [Frenk et al. (1991)].

2. This is not to deny the problems of implementation, time inconsistency, and institutional failures that have plagued aid effectiveness. Because most models of poverty are either static or purely income-based, the value added by our exercise lies in identifying policies that are better at improving dynamic incentives and quantifying their human and economic costs.

3. Although Goenka et al. (in press) focus on steady states of their planner's problem, it is possible that their model too admits cycles, because it embeds a similar behavioral response to disease prevalence.

4. For an alternative specification of intra- and intercohort human-to-human transmission, see CPP (2010).

5. The high-growth steady state always exists and is asymptotically stable: indeed it is the conventional steady state in an OLG model with Ak technology. The other stationary equilibrium may not be stable or may not exist.

6. Because we are modeling various types of infections, many of them endemic, the full-prevalence steady state should be viewed as one where the entire population suffers from one or more infectious diseases at some point in youth. We adjust the mortality and morbidity cost of diseases in the calibration later to reflect this average lifetime effect.

7. These values are supported by available estimates. See, for example, Guvenen (2006).

8. This is for ease of exposition. Capital per effective worker, k, can take the same value in two successive periods even when aggregate capital, K, is being accumulated if there are fewer infected people. A phase portrait in (K, i) space better distinguishes the partial equilibrium economic effects from the general equilibrium health effects.

9. There are two reasons for this difference. First, we assume a perfect annuities market here instead of the redistributive scheme in CPP (2010). This lowers the sensitivity of the saving propensity with respect to ϕ. Second, calibrating ϕ to post-middle-age infectious disease mortality alone implies a higher value. The combined effect is to lower the economic cost of infectious disease and raise the saving propensity.

10. Household saving is decreasing in x. In each example in Figures 3 and 4, the dip in the prevalence rate is associated with a dip in output per worker. The cyclicality of output, however, depends on the assumption that prevention is financially costly. It remains to be seen how other types of cost, such as utility loss, alter the nature of these output cycles. Note also that we do not attempt to establish these results formally. This is because, as the economy cycles back and forth across the $x(K_t, i_t) = 0$ locus, the discrete change in prevention introduces a discontinuity into the dynamical system. We leave a more formal analysis for future work.

11. Health-based foreign aid in Africa has often followed a "vertical" versus "horizontal" approach [Easterly (2009)]. In the former, a specific disease has been targeted through information, vaccination, and other preventive campaigns, besides therapy. The latter focuses, instead, on improving the disbursement of health care through public health reforms. Our analysis of health-based interventions avoids the horizontal approach because we do not formalize the public health system or its shortcomings.

At the same time, we do not look at interventions focused on a particular disease such as malaria or HIV/AIDS.

12. We do not study domestic optimal policies for this reason. Any such policy would be dominated by one that wholly or partly relies on foreign assistance.

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