

Battling Infection, Fighting Stagnation*

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Abstract

Why are some countries mired in poverty and ill health? Can policy facilitate their transition to sustained growth and better living standards? We offer answers using a dynamic model of disease and development. Endogenous transmission of infectious disease generates non-ergodic growth where income alone cannot push a country out of a low-growth development trap. Policy interventions, for example external aid, can successfully accelerate growth only when directed towards improving health and eliminating the burden of infectious disease. Prioritizing improvements to adult mortality over morbidity is better for development.

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1 Introduction

Health and income are each elemental to welfare but it is perhaps their joint relationship that has most intrigued researchers (Bhattacharya and Qiao, 2007, Bhattacharya *et al.*, 2007, Lorentzen and Wacziarg, 2008, Sachs and Malaney, 2002). Countries that are poor in per capita income are also likely to be poor in health. This high positive correlation between income and health is well documented by demographers, economists and epidemiologists (Soares, 2007). Sub-Saharan Africa's prolonged stagnation and overwhelming disease burden is a case in point.

Does better health facilitate economic development? Or is it development that drives health improvements? While there is consensus that the relationship runs both ways, it is unclear if one direction dominates. It also raises important policy questions regarding the type of foreign aid that the donor community should provide to developing countries – income-based or health-specific – a question that is the focus of this study.

We adopt a dynamic general equilibrium model of infectious disease and endogenous growth from Chakraborty, Papageorgiou and Pérez Sebastián (2010) (henceforth CPP). The model yields an empirically relevant development trap; it is especially applicable to sub-Saharan Africa, host to a wide range of infectious diseases. Income deficiency is not the cause of poor health in this equilibrium. Hence marked health improvements can occur only from exogenous improvements in public health or medical innovations.

This property of the development trap opens the door for policy interventions. We show that untied income aid has no growth effect but aid directed towards improving health can propel an economy out of the low-growth equilibrium. The cost of such health aid is substantial unless capital accumulation is simultaneously targeted. We also show that health innovations that improve mortality are preferred to those that lower morbidity because the latter can have perverse effects on incentives to engage in disease prevention.

Current levels of official health aid constitute only about six percent of overall aid. Our findings suggest that the share of health aid should increase drastically to complement recent private initiatives, the Gates Foundation for example, in order to positively impact national income and health.

Our theory sheds light on the “income versus public health” debate. On one side of the debate, McKeown (1976) and notably Fogel (1997) have argued that nutrition played a vital role in Britain's mortality and economic transitions. On the other side, Preston (1996) and more

recently Cutler *et al.* (2006) and Soares (2007), propose that public health initiatives and medical improvements, rather than income gains, caused the worldwide mortality declines of the past century. The possibility of multiple growth trajectories indicates that the interaction between health and income at the aggregate level is more nuanced, and depends on country-specific characteristics.

Our findings also relate to the voluminous literature on aid and growth, from which two papers are worth singling out. After correcting for the bias that aid typically goes to poorer countries and to countries after poor performance, Rajan and Subramanian (2008) find little robust evidence of a positive relationship between aid inflows and growth. Mishra and Newhouse (2009) empirically 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. Our results are consistent with the findings of these two papers.

Finally, our policy analysis informs recent work on political factors influencing health. Specifically, García-Montalvo and Reynal-Querol (2007) find that forced migration and social disruption due to civil wars are significant contributors to malaria incidence across the world. We show that the cumulative economic cost of this interaction between conflict and health is sizable.

The remainder of the paper is organized as follows. Section 2 presents the model and highlights forces driving the dynamics. Since the model closely follows CPP, the presentation is kept relatively brief. Section 3 discusses calibration and introduces different general equilibrium possibilities that inform our policy analysis. Section 4 contains the main contribution of the paper. It presents several policy experiments that culminate in our main recommendation for the appropriate form of international aid. Section 5 concludes.

2 The Model

Overlapping generations of families populate a discrete time, infinite horizon economy. Each individual is born with a unit of labor and potentially lives for two periods: survival to the second period depends on whether or not he contracts infectious disease early in life and prematurely dies from from it.

2.1 Disease Transmission

Individuals work in youth and are retired in the second period. An infected (and infectious) individual suffers a productivity loss of θ due to morbidity, supplying $1 - \theta$ units of efficiency labor. He also enjoys a lower quality of life: a consumption bundle c delivers the 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 being healthy. Subsequently some of them contract infectious diseases, susceptibility to which depends on prevention and disease prevalence. Prevention takes the form of expenditures, x_t , early in youth that are privately costly. This encompasses a variety of health inputs like food nutrients, medicine and medical care. More generally prevention also involves costly behavior such as better hygiene, abstention from risky behavior (Bhattacharya *et al.*, 2007) and occupational choices. Prevention is chosen *ex ante*, before a susceptible young individual meets an infected older person.

Diseases spread from infected older individuals to younger ones through a process of random matching, not all of which result in transmission. Suppose that a susceptible young person meets $\mu > 1$ older individuals. Given his preventive health investment x_t , the probability that a young individual gets infected from an encounter with an infected old is $\pi(x_t)$. We choose

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

for which $\pi' < 0$, $\pi(0) = a$ and $\pi(\infty) = 0$. The parameter q can be interpreted as the quality of national health institutions and medical technology, an improvement in which occurs through a decrease in q (for an explicit treatment of the complementarity between x and q , see Bhattacharya and Qiao, 2007). The parameter a is the probability of getting infected in the absence of prevention. It depends on virus mutations and the genetic evolution of humans.

Let p_t denote the probability of being infected for a typical young member of generation t after the μ encounters. The probability that this person meets an infected old person and contacts the disease is $i_t \pi_t$, where i_t is the fraction of generation $t - 1$ who are infected. The probability of not being infected by any of them is then $[1 - i_t \pi(x_t)]^\mu$. Hence,

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

Appealing to the law of large numbers, this probability also provides the prevalence rate of the

disease at date $t + 1$, that is

$$i_{t+1} = p_t.$$

2.2 Preferences and Prevention

We next discuss how preferences and behavior depend on an individual's health status. The superscript U (I) on variables refers to decisions and outcomes for uninfected (infected) individuals.

An uninfected individual whose preventive behavior has successfully protected him from infectious disease maximizes lifetime utility

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

subject to the budget constraints

$$c_{1t}^U = w_t - x_t - z_t^U, c_{2t+1}^U = R_{t+1}z_t^U,$$

where 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

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

subject to

$$c_{1t}^I = (1 - \theta)w_t - x_t - z_t^I, c_{2t+1}^I = R_{t+1}z_t^I + \tau_{t+1},$$

where τ_{t+1} denotes lump-sum transfers received from the government and utility from death has been normalized to zero. The government collects the assets of the prematurely deceased and distributes them among surviving infected individuals. In equilibrium, these transfers per surviving infected individual will be

$$\tau_{t+1} = \left(\frac{1 - \phi}{\phi} \right) R_{t+1} z_t^I. \quad (3)$$

For the utility function, we choose the standard CES

$$u(c) = \frac{c^{1-\sigma} - 1}{1 - \sigma}, \sigma \geq 0. \quad (4)$$

This takes on negative values for consumption levels less than one. We ensure that consumption exceeds one, and hence survival is always desirable, through appropriate assumptions on the aggregate technology below.

The solution to the optimization problems provide the following saving decisions:

$$z_t^U = s^U(w_t - x_t), \text{ with } s^U \equiv \left(\frac{\beta^{1/\sigma} R^{1/\sigma-1}}{1 + \beta^{1/\sigma} R^{1/\sigma-1}} \right), \text{ and} \quad (5)$$

$$z_t^I = s^I[(1 - \theta)w_t - x_t], \text{ with } s^I \equiv \left[\frac{\phi(\beta\phi)^{1/\sigma} R^{1/\sigma-1}}{1 + \phi(\beta\phi)^{1/\sigma} R^{1/\sigma-1}} \right], \quad (6)$$

conditioned on x . The impact of disease on development partly follows from $z_t^U > z_t^I$. The infected save less since their effective discount rate is lower ($\phi < 1$) and they are less productive ($\theta > 0$). The third type of cost, a lower utility flow ($\delta < 1$), affects saving indirectly through preventive investment.

Optimal saving, substituted into lifetime utility, gives the two indirect utility functions $V^U(x_t)$ and $V^I(x_t)$ contingent on prices, preventive health choices and disease realizations. Knowing that, at the beginning of period t , individuals choose x_t to maximize expected lifetime utility

$$p_t V^I(x_t) + (1 - p_t) V^U(x_t). \quad (7)$$

If the marginal cost from health investment exceeds the marginal benefit at $x_t = 0$, then zero prevention is an optimal choice. As shown below, this occurs when the disease externality is high or when the threat of infection is quite low.

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:

$$F(K^i, L^i) = A(K^i)^\alpha (\bar{k}L^i)^{1-\alpha} + bL^i, \quad (8)$$

where A 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. The additive component bL^i , with $b > 0$, can be interpreted as a pre-industrial technology that utilizes only

labor and natural resources. It ensures a positive capital stock in all possible steady-states and, for a sufficiently high value of b , that consumption levels exceed one.

Standard factor pricing relationships under such externalities imply that the wage per effective unit of labor (w_t) and interest factor (R_t) are $w_t = (1 - \alpha)Ak_t + b$, and $R_t = \alpha A \equiv R$, respectively.

2.4 Equilibrium Dynamics

Equation (2) holds the key to this economy's dynamic behavior. Embedded in it is a negative externality that is intrinsic to the transmission of infectious disease. The strength of this externality rises rapidly with prevalence; when prevalence is sufficiently large the marginal benefit from prevention becomes too low to justify its costs and the disease rapidly spreads to the entire population.

Non-ergodicity, that is path dependent growth outcomes, follow from this disease dynamics. When two stable stationary equilibria exist, everybody is infected in the absence of any prevention in the first one and economic growth is low. Along the other growth path, a fully healthy population enjoys rapid improvements in living standards.

More specifically, the solution to the maximization problem (7) defines optimal prevention as a function of the capital stock and disease prevalence, $x_t = x(k_t, i_t)$. The possibility of two local attractors depends on whether or not this prevention level is positive. Optimal health investment is zero as long as its utility cost dominates, which occurs at relatively low levels of income and very high or very low prevalence rates. When people do engage in prevention, the level of that investment 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 sensible numerical values assigned to the parameters, $\partial p_t / \partial k_t < 0$ and $\partial p_t / \partial i_t > 0$. The former result is simply an income effect operating through preventive investment. The latter ($\partial p_t / \partial i_t > 0$) is determined by two opposing effects: disease prevalence directly increases the probability through the matching process but also tends to lower it by encouraging preventive investment. 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_0, i_0) . 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, this asset market clearing condition leads to

$$k_{t+1} = \frac{p(k_t, i_t) z^I(k_t, i_t) + [1 - p(k_t, i_t)] z^U(k_t, i_t)}{1 - \theta p(p(k_t, i_t))}. \quad (9)$$

The equilibrium evolution of the prevalence rate follows

$$i_{t+1} = p(k_t, i_t). \quad (10)$$

The dynamic system comprising equations (9) and (10) can entertain two stationary equilibria whose properties were mentioned above. It is easy to obtain these two growth rates. Define γ as the asymptotic growth rate of the economy's capital stock per effective unit of labor. When $i_t = 0$, the economy-wide saving propensity is s^U and equation (9) implies

$$1 + \gamma^H \equiv (1 - \alpha) A s^U = \frac{\beta}{1 + \beta} (1 - \alpha) A. \quad (11)$$

Under full prevalence, in contrast, everyone suffers from ill health and the economy-wide saving rate is s^I . In this case, long-run growth is

$$1 + \gamma^L \equiv (1 - \alpha) A s^I = \frac{\beta \phi^2}{1 + \beta \phi^2} (1 - \alpha) A. \quad (12)$$

This growth rate is zero if $(1 - \alpha) A s^I \leq 1$ and positive otherwise. Differences in the growth rates in these two stationary equilibria depend on the mortality cost of infectious disease alone (since $\phi < 1$). Morbidity factors, namely productivity loss and the quality-of-life effect, matter only for convergence to these stationary equilibria via their effect on the level of saving.

3 Quantitative Analysis

We rely on quantitative methods to study the global dynamics behind equations (9) and (10). This is necessitated by the nonlinearity of the dynamic system but has the virtue of providing a tight structure to our subsequent policy analysis.

3.1 Calibration

Parameters are calibrated using aggregates so that we can suitably capture complementarities among various infectious diseases that simultaneously afflict a country. For example, people

infected with one disease become more susceptible to other ones, like HIV/AIDS co-infection with tuberculosis and malaria (Abu-Raddad *et al* 2006), or malaria co-infection with anemia. The consequence of these complementarities is that losses for the economy due to mortality and morbidity are higher than the average loss across illnesses (Dow *et al.* 1999). Table 1 reports the assigned benchmark values.

Table 1: Benchmark Parameter Values

β	0.99 ^(31.5×4)	α	0.67	θ	0.15	μ	5
σ	1	γ^H	0.018	ϕ	0.47	q	0.14
b	1	A	24.19	δ	0.9	a	1

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 the World Health Statistics 2008. This implies 31.5 years for each period or generation. Accordingly the discount factor (β) is calibrated to 0.99^{31.5×4}, 0.99 being the “standard” value per quarter.

We take preferences to be logarithmic ($\sigma = 1$). Three parameters need to be calibrated for the aggregate production function: the total factor productivity (TFP) term A , the output elasticity of capital α , and the pre-industrial technology parameter b . We normalize $b = 1$ to ensure that consumption levels are bounded above one for reasons discussed before. We interpret capital broadly (physical, human, organizational) to set $\alpha = 0.67$. The value for A is chosen such that the growth rate is 1.8% in the zero-prevalence steady state. This growth rate corresponds to OECD’s average growth rate of GDP per capita during 1990 – 2003 (UNDP 2005). In other words, A is chosen such that $(1 - \alpha)s^U A = 1.018^{31.5}$, which implies $A = 24.19$.

Parameters governing disease transmission are critical to quantifying the effort needed to battle disease prevalence and transmission. To assign values to a , μ and q we take into account the minimum annual expenditure that the World Health Organization estimates is needed to fight diseases in least-developed countries.

More concretely, we set $a = 1$ as the benchmark and select pairs of μ and q that allow a country to escape the development trap if preventive investment represents at least 7.2% of its GDP. This percentage comes from dividing 34 by 475. The amount 34 (current US\$) is WHO’s (2001a) estimated minimum health expenditure, and 475 (current US\$) is sub-Saharan Africa’s

average GDP per capita in 2001 (UNDP 2003). For each value of μ , the procedure provides a value for q . We perform policy experiments with three pairs of values: $(\mu, q) \in \{(2, 0.55), (5, 0.14), (10, 0.06)\}$. Since we get similar results in each case, we report results only for the intermediate case, $\mu = 5$ and $q = 0.14$.

Estimates of the quality-of-life impact 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. Disability weights vary across infectious illnesses. For example, it equals 0.0 for the chagas disease, 0.1 for diarrheal episodes, and 0.5 for AIDS according to WHO (2008). We choose a conservative value of 0.1 for the per-unit utility decline and assign the value 0.9 to δ .

In order to obtain an estimate of the income loss due to morbidity, we look at Dasgupta (1993). He finds that workers (in particular, farm workers) that are too ill to work in developing countries lose about 15 to 20 days of work each year, and when they are at work, 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 are of the order of 15%. This is the value assigned to θ .

Finally we calibrate the survival parameter ϕ . According to WHO (2001b), fatalities from infectious diseases represent 53% of all deaths in Africa in 2001 for the adult male population aged 15 and over. We require that the model reproduce this number assuming that sub-Saharan Africa is in a growth trap. Since the entire population suffers from ill health in this steady state, the probability of death from infectious disease has to be 0.53. Hence $\phi = 0.47$ is our benchmark value.

3.2 Phase Portraits

The effect of policies depends on initial conditions and the forces that drive dynamics. The model generates three types of scenarios with different stationary equilibria. Two of them display multiplicity, the third one uniqueness. We discuss each case using a phase portrait.

3.2.1 The benchmark case

Figure 1 displays the phase diagram for the parameter values in Table 1. It plots the prevalence rate i_t against capital per effective unit of labor k_t .

The $x(k_t, i_t) = 0$ line represents combinations of (k_t, i_t) for which the optimal decision is not to invest in prevention. For low levels of disease prevalence ($i_t \rightarrow 0$), the risk of catching an infection is so low that prevention is not necessary. At high levels of disease prevalence ($i_t \rightarrow 1$), in contrast, the productivity of prevention becomes vanishingly small as the disease externality from sequential matching outweighs the benefits from prevention.

The $\Delta k_t = 0$ locus in Figure 1 is obtained by imposing $k_{t+1} = k_t$ on equation (9). Capital per effective unit of labor declines above this locus and increases below. The $\Delta k_t = 0$ line coincides with the $x(k_t, i_t) = 0$ curve to the right of point E . The parabolic shape of the $\Delta k_t = 0$ locus implies that the same prevalence rate can be associated with both high and low levels of capital per effective worker. Two effects underlie this relationship. The prevalence of infectious disease has a negative effect on capital accumulation, the numerator on the right-hand side of equation (9), through premature mortality and labor productivity. An opposing effect comes from the positive effect morbidity has on capital intensity that raises the efficiency wage rate (denominator on the right-hand side of equation 9).

Turn next to the third locus given by the downward sloping line, $\Delta i_t = 0$, defined by

$$i_t = p(k_t, i_t) \tag{13}$$

along which prevalence remains constant. The locus is defined wherever $x_t > 0$ and, in this area, prevalence is always decreasing above the locus, increasing below it. When prevention is zero, on the other hand, the prevalence rate is always increasing since $\mu a > 1$.

There are two poverty traps with zero growth in Figure 1, PT and UPT , and a balanced growth path BGP with positive growth. The PT steady-state is a sink while UPT is a saddle-point. Given both initial conditions i_0 and k_0 , PT is asymptotically stable but UPT is not. Sequences of (k_t, i_t) that do not start exactly on the saddle-arm SS converge either to PT or diverge to the BGP along which infectious diseases are fully eradicated and the economy grows at a healthy rate. Thus the saddle path defines a threshold over the state space.¹

¹More precisely the saddle path is the threshold until it meets the $x = 0$ locus, at which point, the continuation of that locus becomes the effective threshold.

An unusual feature of the development trap dynamics is that if i_t is relatively high (above the $x_t = 0$ locus), the economy always ends up at PT regardless of k_t . The implication is that even a well-off economy can spiral towards the trap if disease prevalence were to sharply increase from an epidemic shock (that subsequently becomes an endemic disease).

3.2.2 No-trap scenario

When the probability of contagion is sufficiently small, the BGP is the unique steady state. To illustrate this, let us entertain values of a that are lower than 1. Recall that a positively affects the probability of being infected after μ matches and, in particular, equals the probability of disease transmission in the absence of prevention. Hence as a falls, preventive investment becomes more efficient. When a falls sufficiently, diseases can be avoided at relatively low cost and enough saving is generated at low incomes to maintain a growing capital stock.

For the benchmark parameterization, a PT exists for $a \in (0.49, 1)$ with a prevalence rate smaller than one. The low-growth trap vanishes when a falls below 0.49. For such low values, the $\Delta k_t = 0$ schedule disappears from the phase plane and optimal preventive investment is positive for all $(k, i) > (0.15, 0.09)$. Hence, no trap exists and all economies converge to the unique BGP irrespective of initial conditions (Figure 2).

3.2.3 Multiple balanced-growth paths

The third interesting possibility shows path dependence like the first, but allows for positive growth in the development trap. This occurs when ϕ is relatively high. When the survival probability exceeds 0.72, the saving rate is always high enough to sustain output growth. Recall, though, that the development trap in this economy is driven fundamentally by the disease externality and that lack of income is not what dooms countries to it. Hence, despite positive income growth, countries do not escape the problem of pervasive ill health. Growth, in this case, does not lead to development.

Assigning a value $\phi = 0.73$ implies that, in the growth trap, the long-run growth rate of output per capita is 0.1%, the average growth for SSA during 1990 – 2003 (UNDP 2005). Figure 3 shows that, as with $a < 0.49$, the $\Delta k_t = 0$ schedule vanishes and positive growth occurs from any point in the (k, i) plane. The figure illustrates dynamics for two economies: both start with the same level of physical capital but differ in their prevalence rates (15% and 20%, respectively).

The economy that starts with a prevalence rate of 15% experiences rising disease prevalence for 2 generations, after which diseases abate as the economy converges to an annual growth rate of 1.8%. The economy with an initial prevalence rate of 20% shows a continuous rise in prevalence until everyone is infected. In the long-run, this economy does not invest in prevention and output per capita grows at 0.1% per year.

4 Policy Experiments

For a stagnant economy afflicted by infectious disease, can external subsidies take it out of the development trap? What is the best strategy to achieve good health and high growth? What is the cost of infection in terms of lost growth?

The calibration strategy we implemented ensures that the minimum amount of health aid, as estimated by the WHO, successfully pushes the economy out of the trap. Having already accounted for the first question, we tackle the other two policy questions in this section.

First we consider subsidies financed through international donations.² Some of these subsidies target health investment (x^{sub}), whereas others come in the form of capital investment (k^{sub}). Then we study the impact of exogenous shocks related to medical technology and politics and examine their policy implications.

4.1 Subsidies

The impact of policy depends on the global dynamics. We conduct policy experiments for each of the three scenarios discussed in section 3.2.

4.1.1 Escaping the poverty trap

Suppose that the country is located at PT . Figure 4 shows the dynamics induced by different policy packages (x^{sub}, k^{sub}). The label next to each line denotes the specific policy package applied and the minimum number of generations for which it needs to be implemented to help the economy escape underdevelopment.

An immediate consequence of the model's dynamics is that no k subsidy alone can take the economy to the BGP . For example, even if international donors were to provide a very large amount of aid for physical capital accumulation $k^{sub} = 0.8$ (26% of GDP at PT) to each

²Qualitatively similar results are obtained if subsidies are financed domestically with lump-sum taxes.

generation, the economy moves to a higher income level at PT' without any impact on disease prevalence and its burden. Figure 4 also illustrates how funds like $x^{sub} = 0.20$ that represent a health investment below 7.2% of GDP, and are therefore insufficient to escape the trap, can reduce the long-run prevalence rate but raise income levels only slightly, from PT to PT'' .

Escaping the trap is possible through health subsidies alone provided that they are large enough. Given the method used to calibrate the disease transmission parameters, $x^{sub} = 0.22$ (7.2% of GDP at PT) is the minimum required to take the economy from PT to BGP . This health subsidy has to be provided for at least 9 generations, which represents a substantial cost. This minimum will be our benchmark to which we compare other policy scenarios.

Important scale economies are associated with x^{sub} in that the number of subsidized generations required to escape the trap falls rapidly with the level of subsidies. For instance, if we double preventive subsidies (i.e., $x^{sub} = 0.44$), it has to be provided for only 3 generations instead of 9. When $x^{sub} = 0.8$ this number falls to 1. This means that the most efficient health investment strategy is a massive attack against infectious diseases, in other words eradication. In present value terms, assuming a real interest rate of 3%, the package $x^{sub} = 0.8$ supplied for one generation costs 0.52, approximately double that the benchmark (whose cost equals 0.24 if provided for nine generations).

Even though capital subsidies alone cannot take the economy to BGP , they can improve the effectiveness of health subsidies. This is true, again, provided that x^{sub} is sufficiently large. For the benchmark parameterization in particular, x^{sub} needs to be at least 0.11 (3.6% of GDP at PT). In Figure 4, if instead of allocating 0.22 units of international aid only to health prevention, we choose $(x^{sub}, k^{sub}) = (0.15, 0.07)$, the required number of subsidized generations falls to 5. If we double the total subsidy and allocate it equally to capital and health investment so that $(x^{sub}, k^{sub}) = (0.22, 0.22)$, the number of generations declines from 3 to 2. But this type of complementarity between capital accumulation and health aid becomes weaker as x^{sub} becomes smaller. For example, policy packages $(x^{sub}, k^{sub}) = (0.8, 0)$ and $(x^{sub}, k^{sub}) = (0.7, 0.1)$ need to be applied only for one generation, but the package $(x^{sub}, k^{sub}) = (0.6, 0.2)$ has to be provided for at least 2 generations.

4.1.2 Convergence without a trap

Suppose $a = 0.49$. As we saw above, there is only one attractor, the *BGP* with high growth and good health. Suppose also that economic development starts from $K_0 = 0.09$ and $i_0 = 0.96$. Aggregate capital stock of 0.09 corresponds to *PT* in Figure 4, and a prevalence rate of 0.96 is the maximum that the economy can endogenously reach for $a = 0.49$.

Figure 5 presents time paths of the growth rate for different policy packages implemented every period. The comparison line $i_0 = 0$ represents the disease-free scenario. The figure is indicative of the substantial the cost of diseases and ill health. If the economy does not receive international aid, growth-rate convergence takes several centuries. This case is labeled the “no subsidies” time path. Growth rates are close to zero during the first 3 generations and do not reach half of that for the $i_0 = 0$ case until the fifth generation. Indeed, growth even becomes negative when the economy starts investing in prevention with generation 3 when the disease externality is low enough to make this worthwhile. The cumulative cost of infectious disease in terms of lost economic growth can, therefore, be large even though the economy converges to the *BGP* in the long run.

Another result that comes out of Figure 5 is that policy packages that foster capital accumulation are now, not surprisingly, always more effective in raising growth. The package $(x^{sub}, k^{sub}) = (0, 0.22)$ takes the economy’s growth closer to the $i_0 = 0$ path rather than the alternatives $(0.11, 0.11)$ or $(0.22, 0)$. Nevertheless, subsidizing capital alone may not be socially desirable if we take into account life expectancy. A package that includes x^{sub} will decrease the population of infected people and contemporaneously increase life expectancy. In contrast, a package that only subsidizes capital accumulation does not impact the current generation. For example, the policy package $(0, 0.22)$ leaves *LE* of the first generation unchanged at 50 years while the package $(0.22, 0)$ raises their *LE* to 60 years, a substantial improvement (Figure 6). Hence, a social planner who values longevity across generations may prefer an intermediate policy that includes both types of subsidies.

4.1.3 Multiple balanced-growth paths

Compared to the benchmark case, our main results regarding policy effectiveness do not change when there are multiple *BGPs*: capital subsidies alone cannot help an economy overcome the low-growth trap, disease eradication is the most efficient intervention, and a mixed policy pack-

age $(x^{sub}, k^{sub}) > 0$ is preferred when eradication is not feasible. But there are a few differences. The main one is that escaping underdevelopment is now cheaper and faster. Another difference is that capital subsidies are less able to compensate for the lack of health investment.

These differences are illustrated in Figure 7 which plots output growth rates (per generation) over ten generations. The starting point in each simulation is again the capital stock and incidence rate in the benchmark-case PT . Each line is labeled appropriately; some of them correspond to a value of ϕ of 0.47 (the benchmark), others to $\phi = 0.73$ (with two $BGPs$).

Consider first results with $(x^{sub}, k^{sub}) = (0, 0.22)$. For both values of the survival rate, growth falls and later rises towards its long-run value. However, growth rates are always higher and the minimum is reached much earlier when $\phi = 0.73$ than for $\phi = 0.47$. In addition, the package needs to be applied for 4 generations in the former case, compared to 9 generations in the latter. This more rapid escape is due to faster capital accumulation.

The figure also shows that the package $(x^{sub}, k^{sub}) = (0.15, 0.07)$ which is able to take the economy out of the trap when $\phi = 0.47$ cannot do so for $\phi = 0.73$. This is true regardless of the duration of the aid. Put differently, capital accumulation has less of a capacity to compensate for the lack of health investment in this case.

4.2 Shocks

We conclude our analysis by considering the effect of health technology, institutions and political conflict.

Start with medical innovations. Exogenous health improvements can occur through reductions in q and/or a . Any improvement in the health technology that diminishes the transmission of infectious disease raises growth and the effectiveness of policy. This is evident from the *no subsidies* trajectory in Figure 5. Suppose that an economy located at the benchmark-case PT sees a large-scale eradication of disease vectors that cause a to fall from 1 to say 0.49. This effectively makes income less relevant for disease transmission. Our previous analysis has shown that this will trigger a long-run growth take-off. In Figure 5, this take-off is preceded by a slow-down lasting several generations which occurs because the lower value of a initially creates stronger incentives for health investment that dominate other types of (growth augmenting) investment.

Medical innovation can obviously reduce mortality and morbidity. Figure 8 shows the effect

of this for economies that start from the poverty trap PT . The first set of exercises examine how the ability of the economy to escape the trap changes with disease costs. A lower mortality risk (lower ϕ in Figure 8) clearly reduces the costs and makes international aid more effective. In particular, when we increase the survival rate from 47 to 57 percent, GDP growth increases substantially for all generations: compare the trajectory “ $x^{\text{sup}} = 0.22$, benchmark” to the trajectory “ $x^{\text{sup}} = 0.22, \phi = 0.57$ ”. Changes in morbidity parameters have the opposite effect. A higher θ or a lower δ elicits a weaker preventive behavior which diminishes the impact of a given policy package. This is illustrated in Figure 8 with a reduction in θ . When the productivity loss due to infection is only 5%, a health subsidy of 0.22 applied during 9 generations can no longer free the economy from the trap (see trajectory “ $x^{\text{sup}} = 0.22, \theta = 0.05$ ”).

It is also instructive to see what effects changes in institutional factors that raise aggregate productivity, that is, higher A , have. Like the survival probability, A directly affects long-run growth. But changes in A have a modest impact on a developing nation’s long-run growth and health. Suppose that initially, $\phi = 0.72$ and $A = 19$. The latter implies an annual steady-state growth rate of 1 percent in a zero prevalence economy instead of the benchmark 1.8 percent. In this case, the PT coordinates are $(k, i) = (0.6, 1)$. In addition, suppose that improvement in institutions protecting property rights and enforcing contracts raises A to 24.19.³ An economy that was previously located in the poverty trap would now experience a relatively modest increase in long-run growth from zero to 0.2 percent and no change in its population’s health.⁴

Finally, political factors also influence national health beyond their role in molding economic and health institutions. Consider a simple example. García-Montalvo and Reynal-Querol (2007) find that, on average, 13 percent of the cases of malaria reported by the World Health Organization are caused by forced migration due to civil wars. The growth loss of starting development with a prevalence rate of 13 percent versus zero is substantial. Comparing the trajectories “ $i_0 = 0$ ” and “ $i_0 = 0.13$ ” in Figure 8, we see that the latter involves slower growth that, at least for the first 3 generations, is less than half that for the disease-free economy.

³It makes sense to assume that its benchmark value (24.19) is the maximum value that A can take. The reason is that A also affects economies that move along the high-growth BGP and are, therefore, on the technology and institutional frontier.

⁴This exercise, of course, uncouples institutional improvements that increase A from improvements in public health systems that lower q .

5 Conclusions

We have presented an analysis of whether policy can accelerate growth and eliminate the burden of ill health in the developing world. Using the general equilibrium links between infectious disease transmission and economic growth presented in Chakraborty, Papageorgiou and Perez-Sebastian (2010), we have shown that untied income-based aid cannot deliver growth while aid directed specifically towards health improvements can. In other words, when infectious diseases significantly contribute to underdevelopment, successful policy interventions have to be health specific, for instance in the form of vaccination or nutritional supplements.

The implications of this paper echo the recommendations of Rachel Glennerster and Michael Kremer on vaccine research (2000). For example, Kremer (2002), and more recently Glennerster *et al.* (2006) and Berndt *et al.* (2007), propose incentives for “... private sector R&D investments in products for diseases concentrated in poor countries”. In our model, health aid in the form of effective vaccination or drugs that can cure major diseases like malaria and tuberculosis is one way, but not the only one, out of the low-growth poverty trap. Our experiments also reveal that while general institutional improvements have limited impact, institutional changes that improve the quality of the health sector (public and/or private) are instrumental in raising aggregate productivity.

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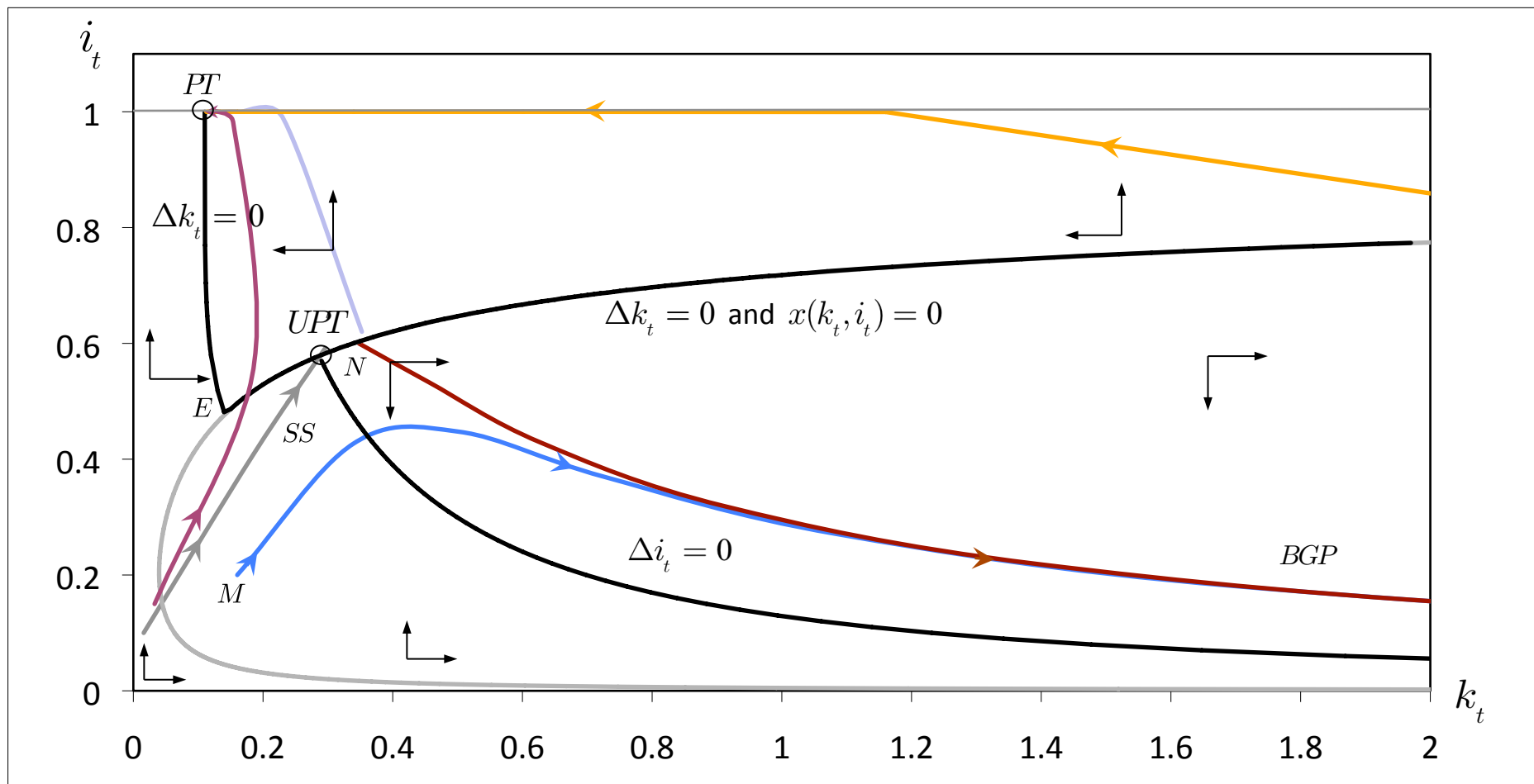


Figure 1: Phase Portrait for the Complete Model under Benchmark Parameter Values

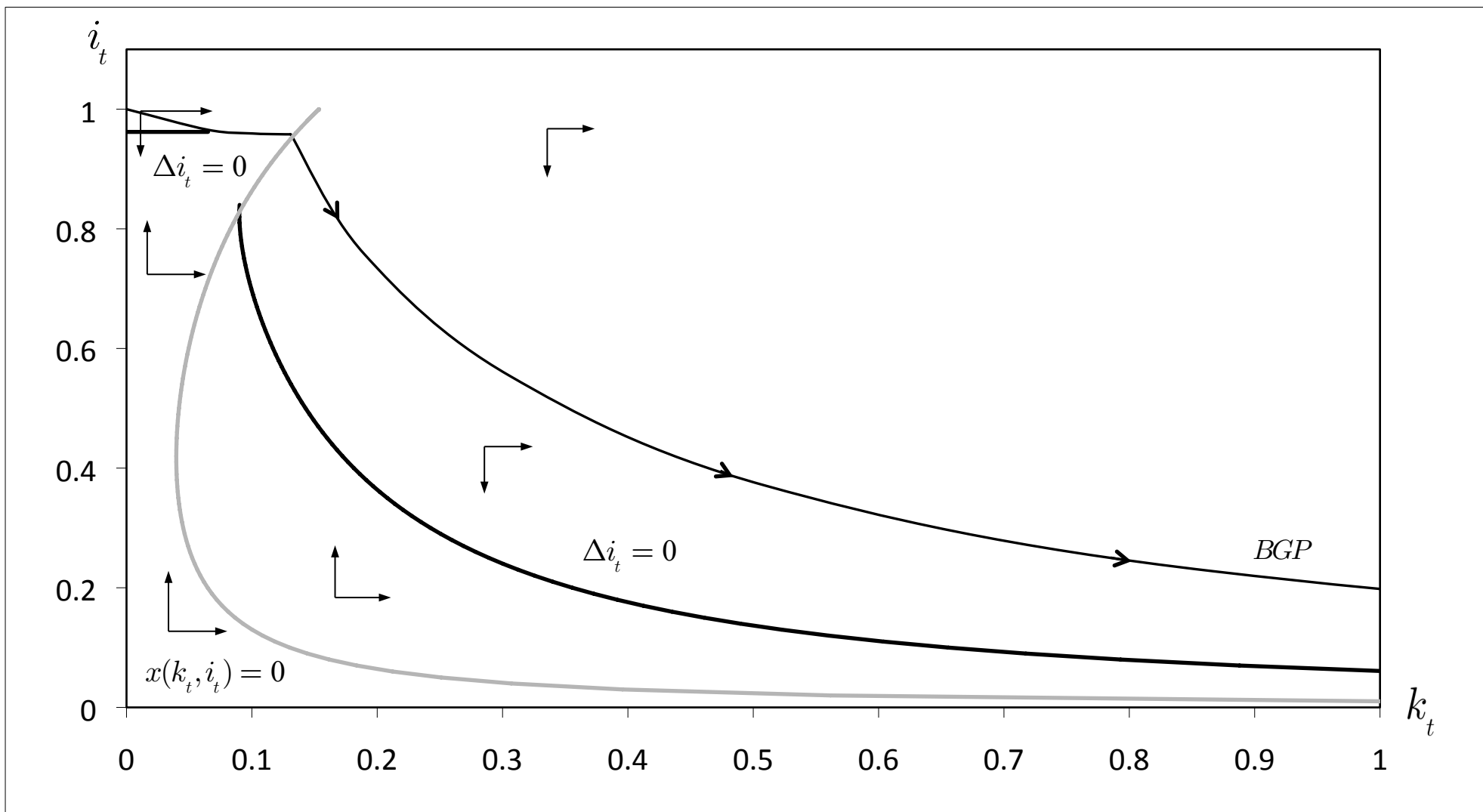


Figure 2: Phase Portrait for $\alpha = 0.49$

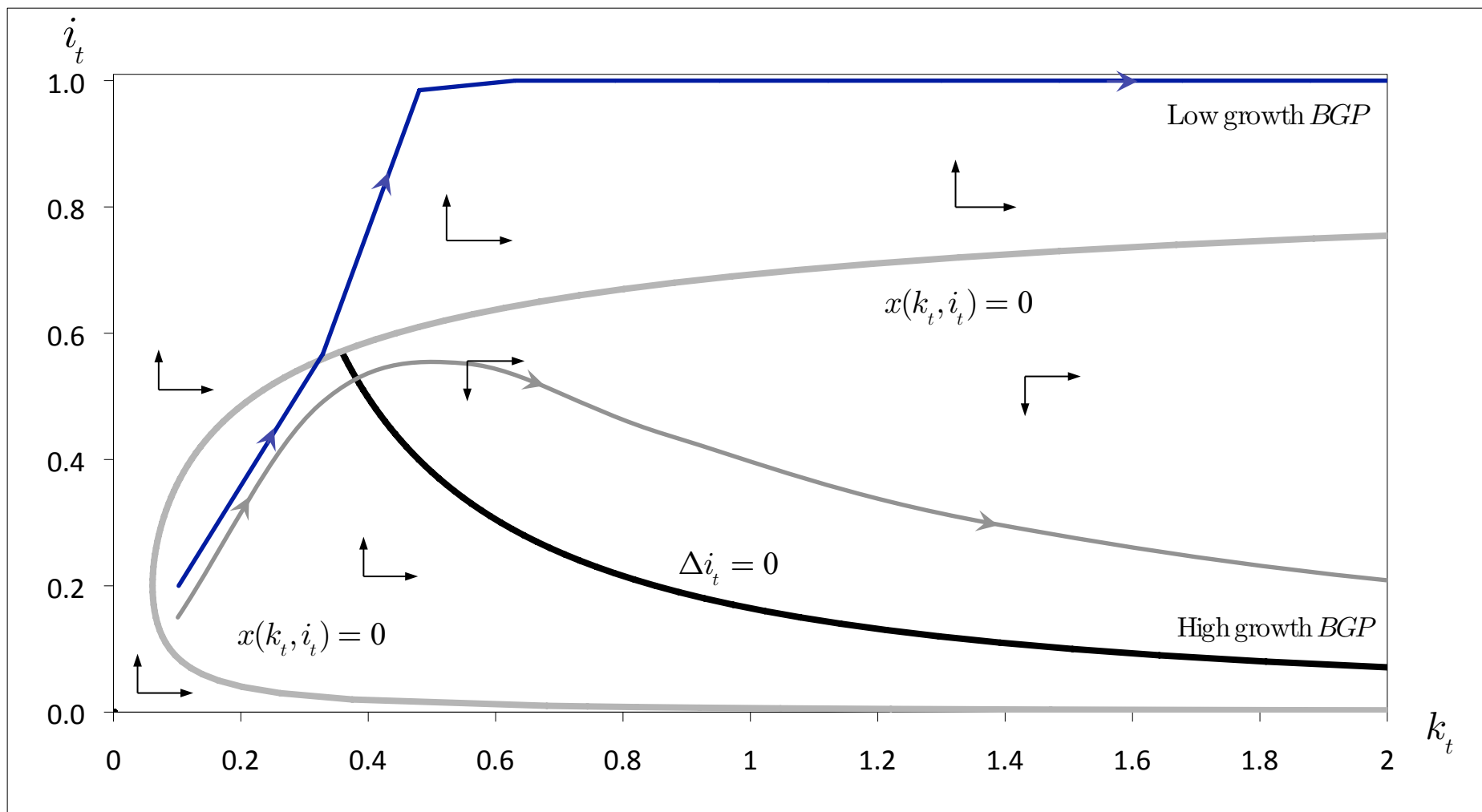


Figure 3: Phase Portrait for $\phi = 0.73$

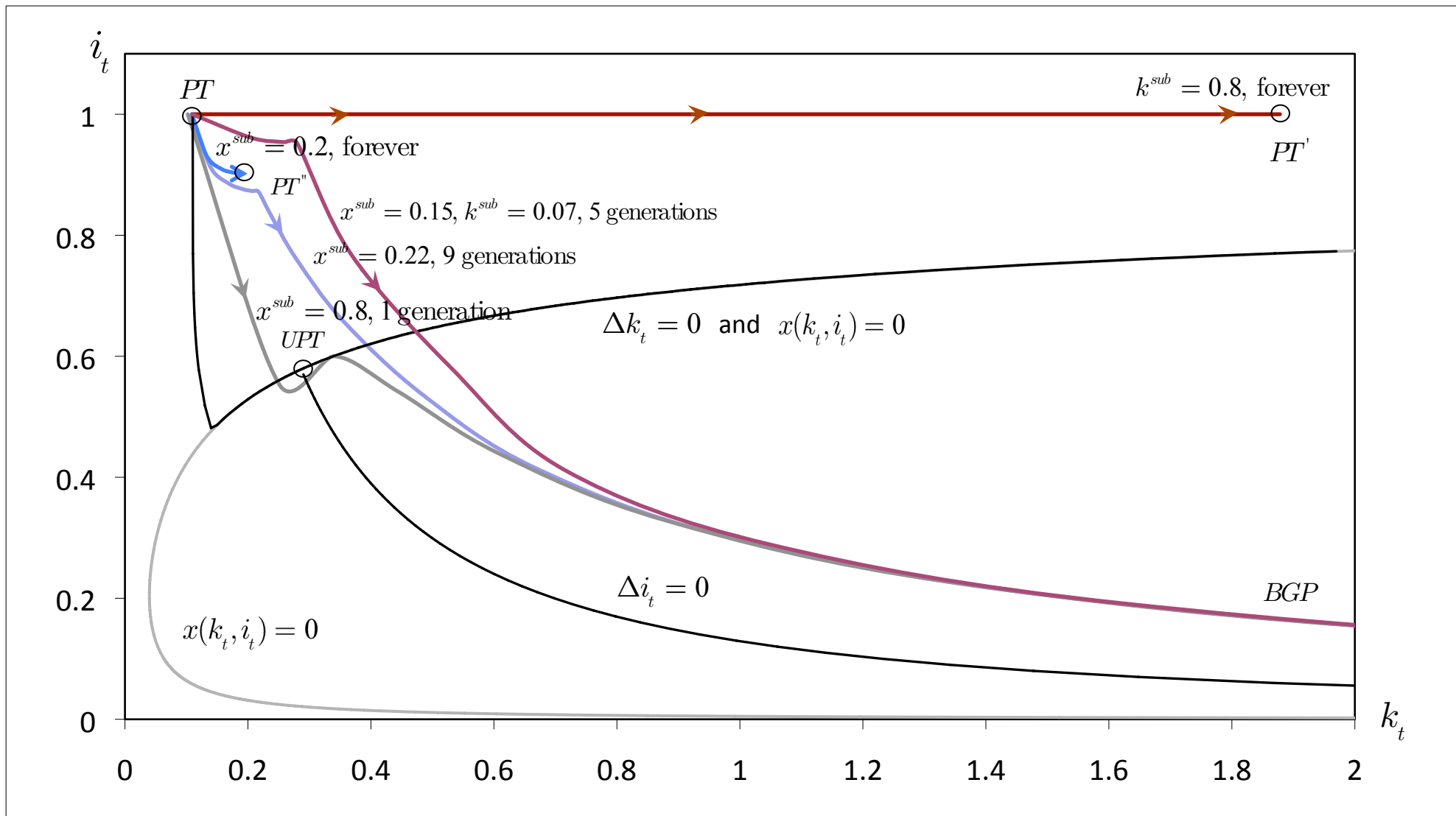


Figure 4: Subsidies to Health and Capital Investment

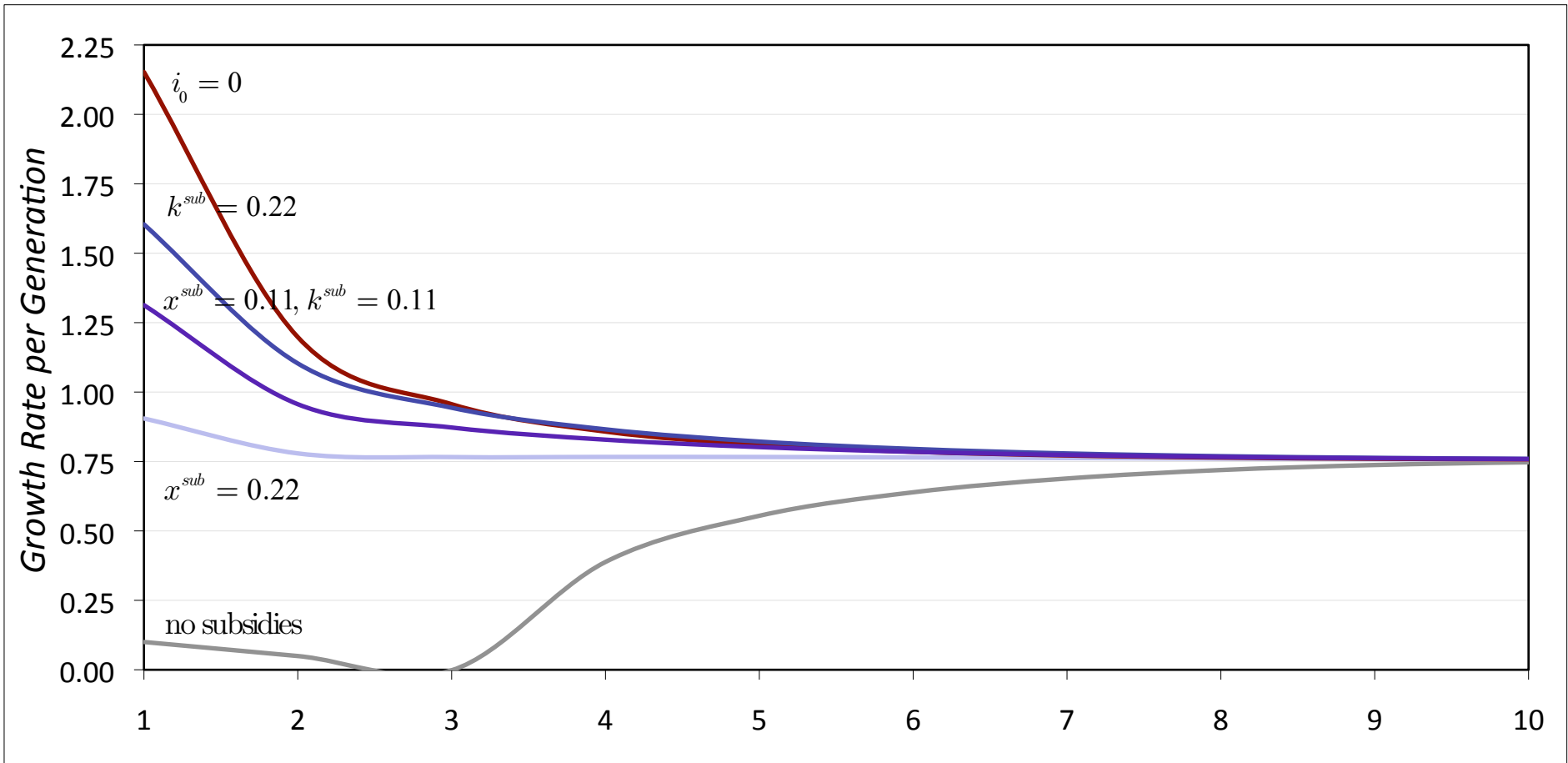


Figure 5: Effect of Subsidies without Poverty Trap ($a = 0.49$)

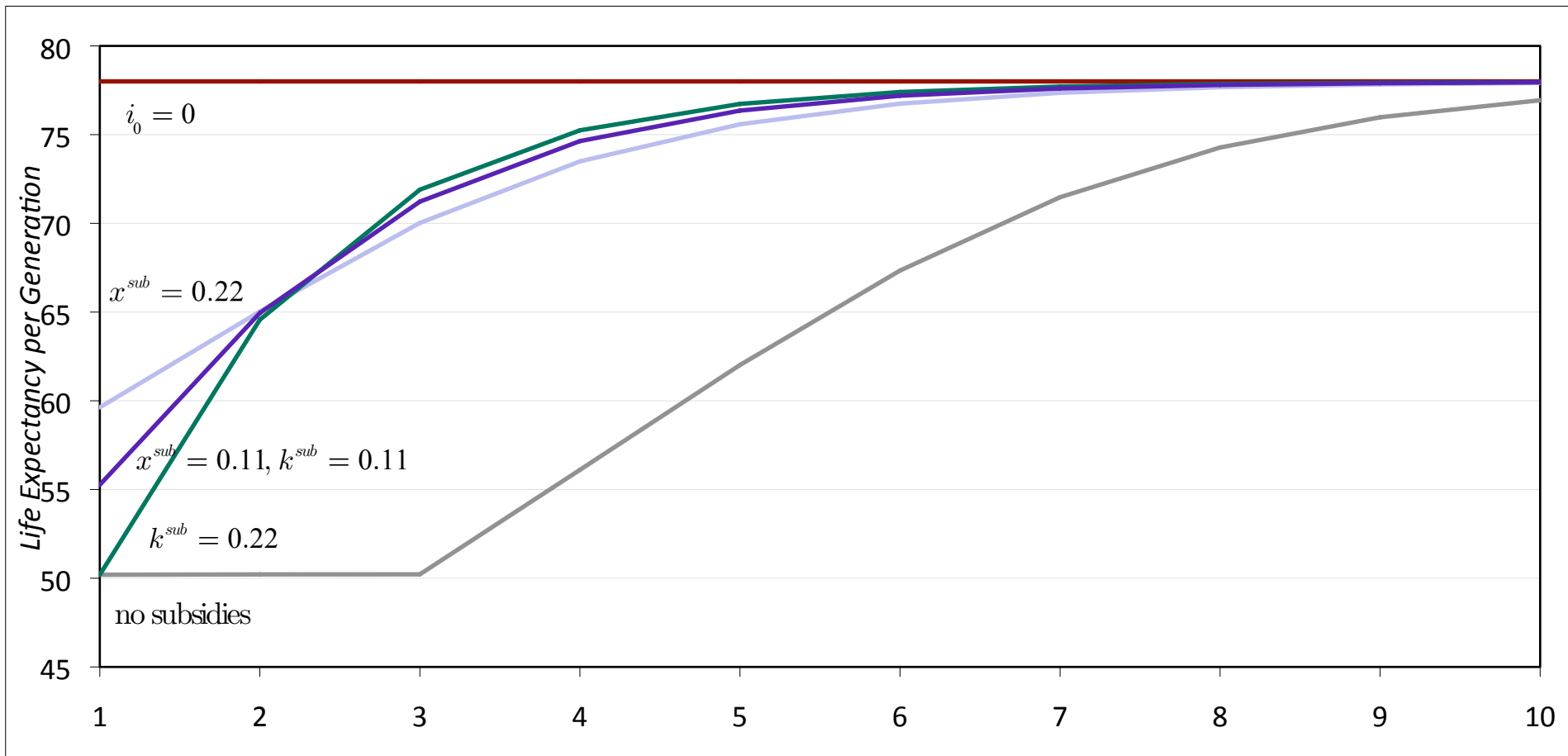


Figure 6: Evolution of LE for $\alpha = 0.49$ under Various Policy Options

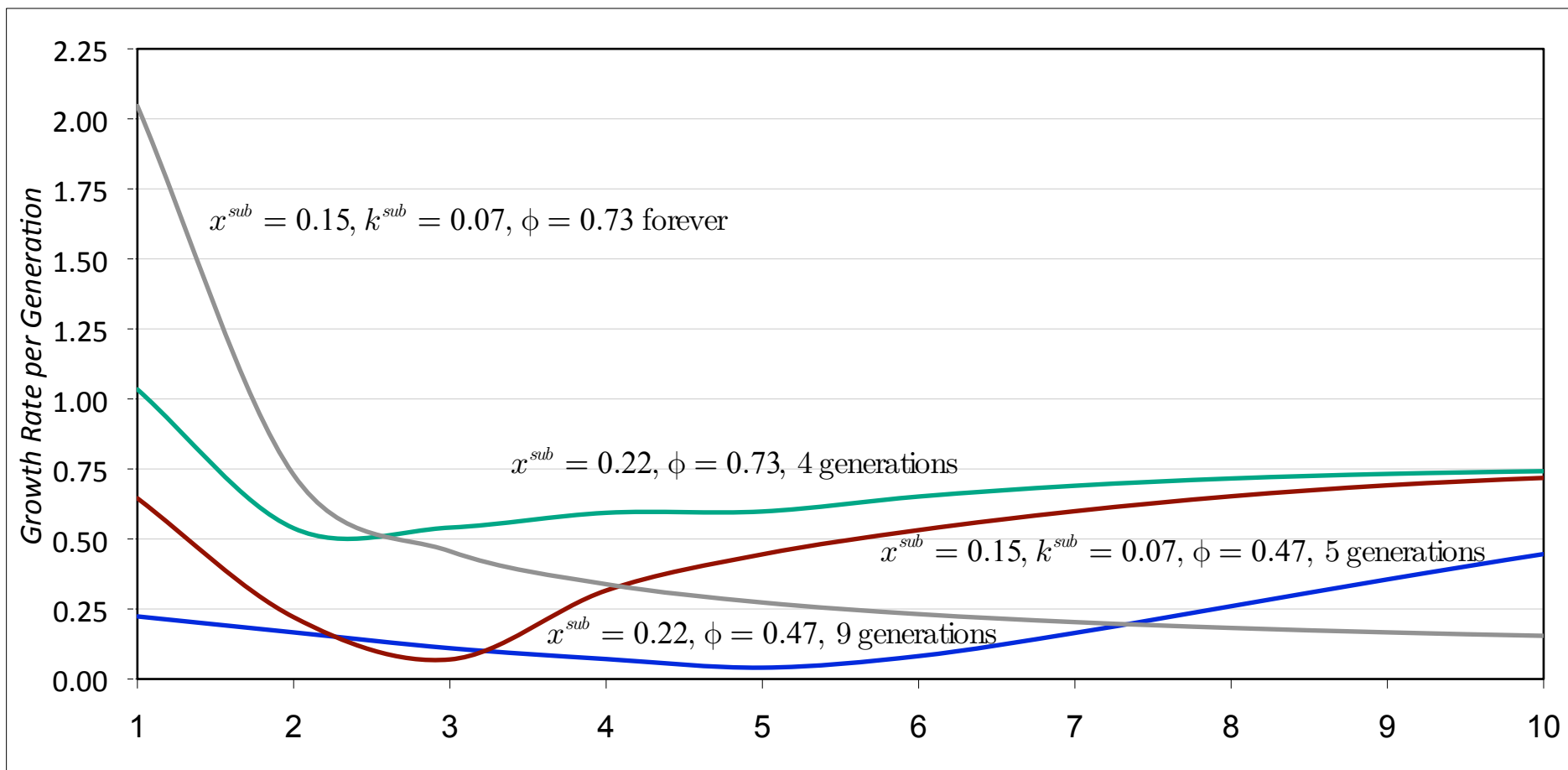


Figure 7: Effects of Subsidies for $\phi = 0.47$ and 0.73

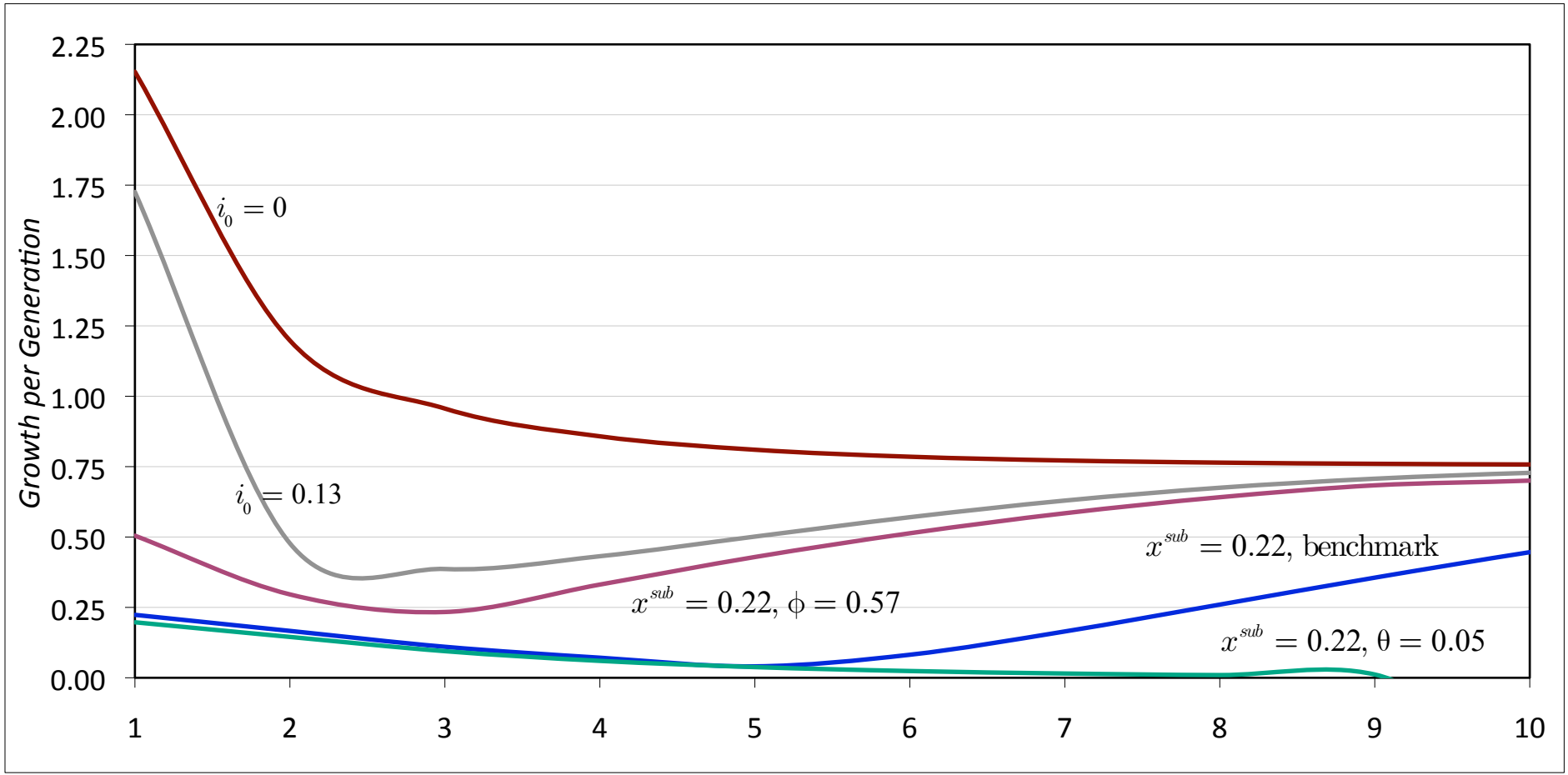


Figure 8: The Impact of Disease Shocks on GDP Growth